Diseases and Pests of Vegetable Crops in Canada

Edited by
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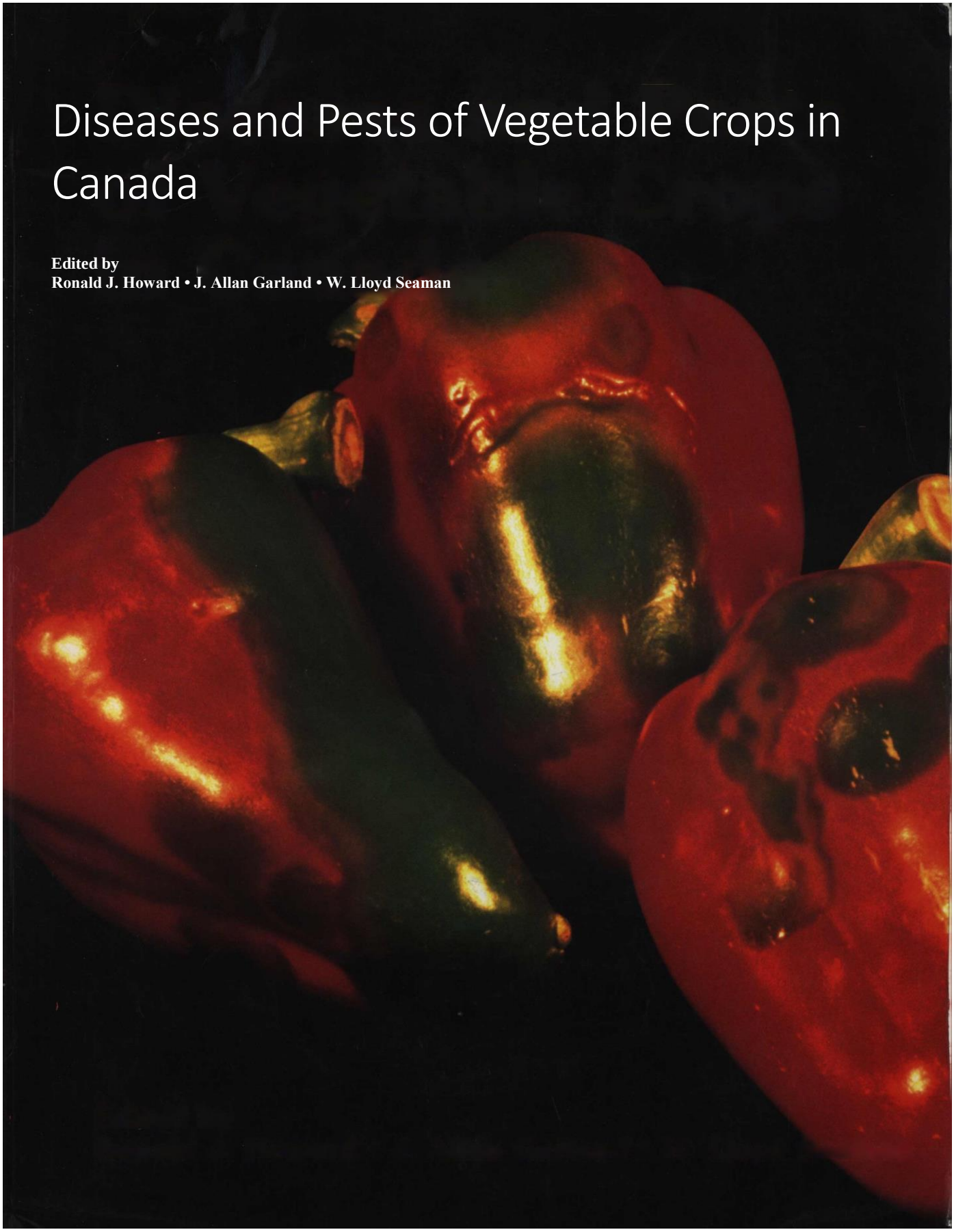

To the amateur biologists and professional plant pathologists and entomologists
whose studies and publications on vegetable diseases and pests have provided the foundation for this book.

In the 19th century, information on identifying and controlling diseases and pests was provided largely by self-taught naturalists, often clergy, through local agricultural societies and colleges and through reports published by government boards of agriculture. The Entomological Society of Canada (founded in 1863, incorporated in 1871 as the Entomological Society of Ontario, and re-established in 1951) frequently included information on economic entomology in its reports. Regional colleges of agriculture (the first founded in 1859 at Sainte-Anne-de-la-Pocatière) provided instruction in pest management, and *Le Naturaliste canadien,* founded in 1868, included articles on diseases and pests. Expert advice was provided by universities, beginning in 1874 with the Ontario Agricultural College, which produced a series of bulletins from 1886. Research and extension on a national scale began in 1886 with the formation of the Experimental Farms of the Dominion Department of Agriculture, with William Saunders as director and James Fletcher as entomologist and botanist. Fletcher and his successors, botanist H.T. Güssow and entomologist C.G. Hewitt, established laboratories of plant pathology and entomology across Canada; the department also introduced several series of bulletins and other publications, including *Canadian Plant Disease Survey* (1920) and the *Canadian Insect Pest Review* (1923). The Quebec Society for the Protection of Plants, founded in 1908 by naturalists and biologists interested in plant diseases and insect and weed pests, published annual reports in English and in French that were widely distributed in Quebec and elsewhere. In 1918 Canadian plant pathologists met to form the Canadian Division of the American Phytopathological Society, which in 1929 became the Canadian Phytopathological Society. The following list of publications is representative of advisory contributions of that period on diseases and pests of vegetable crops.

1868 **Bustin,** W. The potato disease. *J. Agric. Nova Scotia* 1:314-315.

1869 **Provancher, L.** Les pommes de terre et leur maladie; L'anthomye de l'ognon. *Nat. Can. (Que.)* 1:37-44; 155-157.

1872 **Beadle, D.W.** *Canadian Fruit, Flower, and Kitchen Garden.* James Campbell & Son, Toronto. 391 pp.

1891 **Fletcher, J.** Recommendations for the prevention of damage by some common insects of the farm, the orchard and the garden. Dorn. Can. Dep. Agric. Exp. Farms Bull. 11.

1891 **Fyles, T.W.** Kitchen-garden pests and how to deal with them. Pages 44-50 *in* 21st Annu. Rep. Entomol. Soc. Ont., 1891.

1892 **Shaw, T.,** and **C.A. Zavitz.** Weeds and modes of destroying them. Ont. Agric. Coll. Bull. 85.

1893 **Panton, J.H.** Remedies for common plant and insect foes. Ont. Agric. Coll. Bull. 87.

1895 **Craig,** J., and J. **Fletcher.** 1. Spraying for the prevention of fungous diseases; 2. Injurious insects; 3. Potato diseases [potato blights, potato scab]; 4. Black knot of the plum and cherry. Dorn. Can. Dep. Agric. Exp. Farms Bull. 23.

1904 **Harrison,** F.C., and **B. Barlow.** Some bacterial diseases of plants prevalent in Ontario. Ont. Agric. Coll. Bull. 136.

1905 **Fletcher, J.** Insects injurious to grain, fodder crops, root crops and vegetables. Dorn. Can. Dep. Agric. Exp. Farms Bull. 52.

1906 **Lochhead, W.,** and **T.D. Jarvis.** The common fungus and insect pests of growing vegetable crops. Ont. Agric. Coll. Bull. 150.

1907 **Harcourt, R.,** and **H.L. Fulmer.** Insecticides and fungicides. Ont. Agric. Coll. Bull. 154.

1909 **Bethune,** C.J.S. Insects affecting vegetables; and **Eastham,** J.W., and J.E. **Howitt.** Fungus diseases affecting vegetables. Ont. Agric. Coll. Bull. 171.

1909 **Giissow, H.T.** A serious potato disease occurring in Newfoundland. Dorn. Can. Dep. Agric. Exp. Farms, Div. Bot. Bull. 63.

1909 **Swaine,** J.M. Injurious insects of the Montreal area in 1908. Pages 17-23 in 1st Annu Rep. Que. Soc. Prot. Plants.

1912 **Hewitt, C.G.** Legislation in Canada to prevent the introduction and spread of insects, pests and diseases destructive to vegetation, with regulations regarding the importation of vegetation into Canada. Dorn. Can. Dep. Agric. Exp. Farms Div. Entomol. Bull., 2nd Ser. 11.

1913 **Gorham, R.P.** Insecticides and fungicides for orchards and garden crops. N.B. Dep. Agric. Bull. 2.

1914 **Fraser, W.P.** Storage rots of potatoes and other vegetables. Pages **50-51** in 6th Annu Rep. Que. Soc. Prot. Plants.

1914 **Eastham,** J.W. Powdery scab of potatoes. Dorn. Can. Dep. Agric. Exp. Farms Div. Bot. Farmers' Circ. 5.

1916 **Eastham,** J.W., and **M.H. Ruhmann.** Diseases and pests of cultivated plants; and **Hoy, B.** Sprays and spraying. B.C. Dep. Agric. Bull. 68.

1916 **Murphy, P.A.** The black leg disease of potatoes. Dorn. Can. Dep. Agric. Exp. Farms Div. Bot. Cire. 11.

1917 **Gibson, A.** Common garden insects and their control. Can. Dep. Agric. Entomol. Br. Circ. 9.

1917 **Jackson,** V.W. Potato diseases in Manitoba. Man. Agric. Coll. Ext. Bull. 14 (Part II).

1918 **Howitt, J.E.,** and **D.H. Jones.** The more important fungus and bacterial diseases of vegetables in Ontario. Ont. Agric. Coll. Bull. 258.

1918 **Maheux,** G. The protection of plants. Que. Dep. Agric. Bull. 42.

1918 **McCubbin, W.A.** The diseases of tomatoes. Dorn. Can. Dep. Agric. Exp. Farms Div. Bot. Bull., 2nd Ser. 35.

1920 **Dickson, B.T.** Diseases of the potato. Pages 67-103 *in* 14th Annu. Rep. Que. Soc. Prot. Plants.

1921 **Cutler, G.H.,** and **G.B. Sanford.** Potato diseases. Univ. Alberta Coll. Agric. Field Husb. Circ. 7.

1922 **Caesar, L.** The cabbage maggot. Ont. Agric. Coll. Bull. 289.

1922 **Gibson, A.,** and **W.A. Ross.** Insects affecting greenhouse plants. Dorn. Can. Dep. Agric. Bull., N.S. 7.

1923 **Treherne, R.C.** Root maggots and their control. Dorn. Can. Dep. Agric. Pamphlet, N.S. 32.

1924 **Vanterpool, T.C.** The stripe or streak disease of tomatoes in Quebec. Pages 116-123 *in* 16th Annu. Rep. Que. Soc. Prot. Plants.

1926 **Racicot, H.N.** A spotting and shrinking of potatoes in storage. Pages 55-56 *in* 18th Annu. Rep. Que. Soc. Prot. Plants.

1927 **Gingras, P.** La pyrale du maïs. *Rev. Inst. Agric. Oka* 1:228-230.

1928 **Baribeau, B.** Inspection et certification de la pomme de terre. *Rev. Inst. Agric. Oka* 2:2-7.

1929 **Howitt, J.E., D.R. Sands** and **D.H. Jones.** Fungus and bacterial diseases of vegetables. Ont. Agric. Coll. Bull. 345.

1929 **Hurst, R.R.** Studies in potato diseases. 1. Late blight and rot of potatoes caused by the fungus *Phytophthora infestans* (Mont.) de Bary. Dom. Can. Dep. Agric. Bull., N.S. 119.

1932 **Dustan, A.G.** Vegetable insects and their control. Dom. Can. Dep. Agric. Bull., N.S. 161.

Recognition also is extended to **J.C. Walker** for *Diseases of Vegetable Crops* (1952) and to **C. Chupp** and **A.F. Sherf** for *Vegetable Diseases and their Control* (1960), classic textbooks on vegetable diseases in North America; and to **I.L. Conners** for *An Annotated Index of Plant Diseases in Canada* (1967).

Diseases and Pests of Vegetable Crops in Canada

An Illustrated Compendium

Edited by Ronald J. Howard, J. Allan Garland and W. Lloyd Seaman

The Canadian Phytopathological Society

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Contents

Foreword

This compendium is intended for producers, extension personnel, students, diagnosticians and others interested in the diseases and pests of vegetable crops grown in Canada. It is patterned after *Diseases of Field Crops in Canada*, a companion volume published by the Canadian Phytopathological Society in 1984 and revised in 1988. *Diseases and Pests of Vegetable Crops in Canada* represents the combined efforts of plant pathologists, entomologists and vegetable specialists employed in or recently retired from careers in research, extension, teaching and regulation with provincial and federal governments, universities and other agencies.

This book was seven years in preparation and it is the product of an entirely voluntary effort. The presidents of the CPS and ESC, on behalf of the members they represent, extend thanks and congratulations to the editors, to the many contributors of text and illustrations, to the numerous reviewers of the manuscript, and to all others who assisted in making this publication a reality. We hope that it fulfills a need for current information on vegetable crop protection and that it will encourage further collaboration between the two sponsoring societies and foster closer cooperation in research and extension work between their members and the vegetable industry in Canada.

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A joint CPS/ESC Marketing Committee, initially chaired by Ieuan Evans (CPS), was co-chaired by Marilyn Dykstra (CPS) and Lloyd Dosdall (ESC) from 1991 to 1992, and since then by Marilyn Dykstra (CPS) and John Laing and Joe Shorthouse (ESC).

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Shattuck, V.I.: 8.20

Shaw, B.R.: 2.3T1

Sheppard, J.W.: 8.2f

Shipp, J.L.: 3.7j, 3.7q, 3.7t, 3.10f, 18.42i, 22.34c, 22.34e, 22.34f, 22.34g, 22.34h, 22.36a, 22.36e, 24.12d, 24.12e, 24.14a, 24.14b, 24.14d, 25.27b, 25.27c, 25.27d, 25.27e, 25.27f, 25.28e, 25.31, 25.32

Shurtleff, M.C.: 5.2, 5.3, 5.4a, 5.4b, 5.10, 6.7, 6.10, 7.2, 7.4

Skotland, C.B.: 10.9b

Introduction

Diseases and Pests of Vegetable Crops in Canada is a practical guide to the diagnosis and management of the more important diseases and pests of vegetable crops grown in Canada. Pest management recommendations emphasize sustainable approaches to crop protection.

Part 1 Chapter 1 provides an overview of vegetable crops and their commercial importance in Canada. Chapter 2 includes a discussion of the effects of diseases and pests on vegetable production and a description of the major groups of causal agents and physiological factors that limit crop productivity or affect the quality of produce. Chapter 3 provides an outline of management strategies that will prevent crop losses or limit damage from diseases and pests to an acceptable level. This chapter also includes information on recommended cultural control practices, the availability of resistant cultivars, and the current status of monitoring techniques, biological controls, including parasites and predators, and the role of chemicals in management strategies. A special section on management by exclusion deals with pathogens and pests that are subject to import restrictions or quarantine regulations limiting or prohibiting the movement of seeds, plants and soil into or within Canada. Other major sections in Chapter 3 include discussions on the management of weeds and of nematode pests, and a special section on problems likely to be encountered in the home vegetable garden, where pest problems and management strategies often differ from those found in commercial production.

Parts 2 to 5 provide detailed descriptions of the most important diseases and pests that affect specific vegetable crops. For convenience, crops are grouped according to the method of production and are listed alphabetically within each group. The sequence of chapters and illustrations is the same in both the English and French editions of the book. This harmonization was achieved by the use of some unusual crop headings, such as Maize (sweet corn) [Mai's sucré] and Pea and bean [Pois et haricot]. Part 2, chapters 4 to 18, deals with disease and pest problems of the major field-grown vegetable crops. Part 3, chapters 19 to 21, includes plant species that are native to North America. The fiddlehead, or ostrich fern, is not cultivated in the normal sense; rather, it is collected from natural sites in eastern Canada. The Jerusalem artichoke, which is considered a weedy plant in some areas, is grown commercially on a very small scale as a vegetable and for other uses. Ginseng, although field-grown, is protected by lathing. Part 4, chapters 22 to 25, describes the disease and pest problems of the major greenhouse vegetable crops, including cucumber, lettuce, pepper and tomato. Vegetables grown in the greenhouse share many of the same disease and pest problems that are encountered on their counterparts in the field, but there also are important differences in epidemiology and management; in some cases, the reader will be referred to one or another section of the book for further information or for color illustrations. Part 5, chapters 26 and 27, deals with other protected crops, namely, the button mushroom and vegetable sprouts.

Disease and pest descriptions To aid in finding descriptions of diseases and pests and their color illustrations, which are grouped near the end of the book, the major text entries are numbered sequentially by chapter and section; the color illustrations appear in the same order and have the same number as the corresponding text. Halftones and line drawings in the text also are identified by section number and the letter T. For example, 4.6 is the section describing rust of asparagus and is also the figure number of the corresponding illustrations *(4.6a,b)* and text figure *(4.6T1).* Tables also are identified by the corresponding section number. The running head for a left-hand page includes the crop name and section number of the first entry beginning on that page; the running head for a right-hand page contains the section number of the last entry on that page.

The presentation of diseases and pests for each crop follows the same general order, beginning with diseases of bacterial, fungal, viral or physiological origin, followed by problems caused by nematodes, insects, other arthropods, and molluscs. Within these sections, diseases and pests are listed alphabetically by the most widely used common name. Included for some diseases and pests are alternative, often regional, names or names that are associated with distinctive symptoms, such as root rot, seedling blight or damping-off, all of which describe phases of the same disease.

The common name of a disease or pest is followed by the italicized scientific name or names of the causal organism and the name, usually abbreviated, of the taxonomic specialist who described the organism. Many fungi have distinct asexual (anamorph) and sexual (teleomorph) states, and each has a different scientific name. To add to the possible confusion, scientific names change from time to time as the result of research; therefore, synonyms or earlier names of organisms may be included for continuity and reference to earlier literature. Scientific names will be of interest chiefly to specialists but are essential in clearly identifying diseases and pests, which is a prerequisite to prescribing effective management practices.

The description of each disease or pest begins with an introduction to its importance and distribution in Canada, followed by a discussion of symptoms or damage that will assist in the diagnosis of the problem in the field or greenhouse. Sections headed Causal agent or Identification include detailed descriptions of causal organisms, including microscopic features and measurements of reproductive organs, or information on specialized media on which these organisms can be grown in the laboratory for identification. Because this type of information is of interest mainly to diagnosticians and other specialists, these sections are set in smaller type. Other sections describe the disease cycle or the life history of the pest, the epidemiology of the disease, or factors influencing epidemics and fluctuations in pest populations.

The Management section includes currently recommended strategies for limiting economic pest damage to vegetable crops; these include monitoring, cultural practices, resistant cultivars, biological control and chemical control. In this publication,

pesticides and resistant cultivars usually are discussed only in general terms because specific recommendations should be based on local conditions and current information.

Many descriptions include selected references to scientific articles and books containing more detailed information on diagnosis, epidemiology or management that will be of special interest to disease and pest specialists. Guides to further reading are found in additional references at the end of the crop chapters and in the Bibliography.

Each disease or pest description is followed by the name of the contributing author(s). In many cases, the original description has been modified following review and editing.

Color photographs For many users of the book, the color illustrations will provide the initial clues to diagnosing a problem. Illustrations include symptoms of most of the major infectious diseases and disorders described in the text, as well as many of the arthropod (insects and mites), nematode and mollusc pests of each of the crops. To aid in searching for the appropriate descriptive text for a disease or pest, the color illustrations are arranged by crop in the same sequence as in the text chapters, and the figure number for each illustration is the same as the section number of the corresponding text. Multiple illustrations of a disease or pest are identified by letters appended to the number; for example, the figure numbers of the two illustrations of downy mildew of beet are *5.4a* and *5.4b*.

Sources of information In addition to the references specific to pest sections and crop chapters, a list of general references for further reading is included in the Bibliography. Most of the technical terms used in the text are explained in the Glossary.

Advice Vegetable growers are encouraged to consult qualified extension specialists or crop consultants for advice on management strategies appropriate to a given crop and location. In most areas of Canada, expert advice is available at provincial diagnostic clinics and extension offices, at Agriculture Canada research stations, and at some universities and privately operated pest clinics. A guide to the location of such clinics or sources of advice may be found in the Appendix.

Collecting and submitting specimens for diagnosis For many diseases and pests, accurate diagnosis or identification will require microscopic examination of affected tissues for signs of a causal agent. In some cases, the isolation and characterization of a pathogen requires specialized techniques or resources found in diagnostic laboratories.

Plants or pests collected for diagnosis must be as complete and as fresh as possible. It is best to collect several specimens that show the various stages of a problem. Where possible, whole plants should be submitted. To keep root systems intact, plants should be dug rather than pulled from the soil. Soil samples should be submitted along with plants if a soil-borne insect or nematode problem is suspected. To avoid deterioration during shipment, foliage should be free of soil and excessive moisture. It is also important that plants not be mailed in plastic bags as this will hasten spoilage. Intact plants often may be shipped successfully in a cardboard container if a small amount of moist soil is retained around the roots and the root ball is enclosed in plastic wrap or a snuggly fitted plastic bag tied off around the stem to prevent soil from escaping.

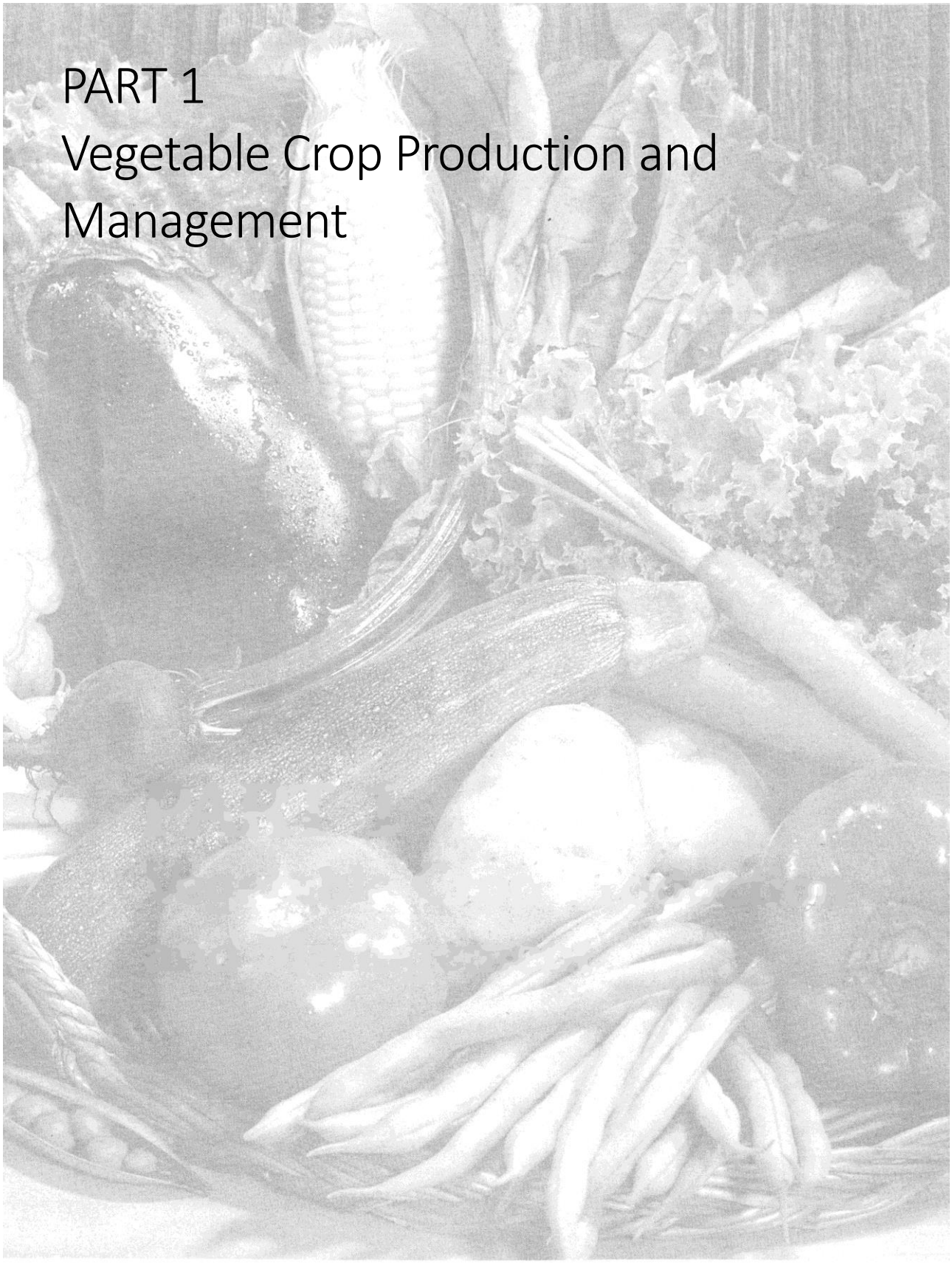
Hard-shelled insects should be placed in cotton wool or another soft material, placed in a sturdy vial, then securely packed in a cardboard mailing container. Dead insects are very fragile and shatter easily, making identification almost impossible. Mites and insects, especially caterpillars and grubs, should be collected live because they often must be reared through to the adult stage before an accurate identification can be made. Such specimens should be provided with adequate food, usually host material, to minimize mortality during shipment.

As much information as possible should be provided with specimens to aid the specialist in making an accurate diagnosis and in recommending appropriate control measures. This should include such details as host and cultivar names, habitat, cropping practices, type and extent of damage, and recent history of weather conditions and crop rotation.

Inquiries about this book General inquiries or comments about *Diseases and Pests of Vegetable Crops in Canada* should be addressed to the editors. We would appreciate receiving constructive criticism of the contents and would welcome new or additional information and photographs that could be used to improve a subsequent edition.

This book is also available in French under the title *Maladies et Ravageurs des Cultures Légumières au Canada.*

Ronald J. Howard J. Allan Garland W. Lloyd Seaman



PART 1
Vegetable Crop Production and Management

Locating Text Sections and Figures

Text sections are numbered consecutively within each chapter. For example, section 16.2 describes bacterial soft rot, the second topic of Chapter 16, Potato. To find a text section, refer to the running heads, which carry the inclusive section numbers for each two-page spread.

Color illustrations, grouped near the back of the book, appear in the same order and have the same number as the corresponding text section; for example, figures *16.2a* and *16.2b* illustrate the symptoms of bacterial soft rot of potato. Line drawings, halftones and tables are numbered similarly, except that a text figure number contains the letter T; for example, Figure *16.2T1* illustrates the disease cycle of bacterial soft rot of potato.

1 Crop production

1.1 Importance of vegetable crops

Vegetable crops are an important component of Canada's agricultural industry, as well as being a staple in many home gardens. In 1992 the farm cash receipts for vegetable crops, including potato, produced commercially in Canada totalled about $1104 million (Table 1.1). About 36% of that was reported in Ontario, 21% in Quebec, 16% in British Columbia, and 7% in Prince Edward Island.

Climate, principally temperature, is the major factor limiting the production of vegetables in Canada. Ontario, Quebec, British Columbia, New Brunswick, Nova Scotia and Prince Edward Island produce the majority of commercial vegetables. Warm-season crops, such as bean, cucurbits, eggplant, onion, pepper, sweet corn and tomato, are produced mainly in the southern portions of Ontario, Quebec and British Columbia. The shorter frost-free period and lower mean temperatures in other areas of Canada generally favor cool-season vegetables, such as asparagus, carrot, celery, cole crops, lettuce, parsnip, pea, potato and rhubarb. Vegetables also are grown in greenhouses across Canada; most of this production is in Ontario, British Columbia, and Quebec.

Table 1.1. Farm value of potato and other vegetable crops in Canada

Province	Potato		Other vegetables		All vegetables	
	Value $ million	% of total	Value $ million	% of total	Value $ million	% of total
Nfld.	1.3	0.4	3.1	0.4	4.4	0.4
P.E.I.	72.9	20.2	6.7	0.9	79.5	7.2
N.S.	7.4	2.0	12.7	1.7	20.2	1.8
N.B.	47.1	13.0	7.8	1.0	54.8	5.0
Que.	59.3	16.4	170.4	22.9	229.7	20.8
Ont.	41.4	11.4	351.3	47.3	392.7	35.6
Man.	53.4	14.8	17.5	2.4	71.0	6.4
Sask.	11.8	3.3	0.0	0.0	11.8	1.1
Alta.	36.5	10.1	32.4	4.4	68.9	6.2
B.C.	30.4	8.4	141.0	19.0	171.4	15.5
Canada	361.5		742.9		1104.4	

Source: Statistics Canada, Catalogue 21-603 (24 November 1993); reported as farm cash receipts, 1992.

Most of Canada's vegetable production is marketed domestically as fresh produce, with a smaller proportion sold as processed products. In most provinces, commercial and market garden vegetable farms are located relatively close to principal population centers or near major transportation corridors. Exports in 1992 totalled $285 million.

Minimizing losses in storage is an important consideration for those involved in processing and marketing vegetable crops in Canada. Root crops, in particular, are often stored for many months, awaiting processing or direct sale. Because stored vegetables are usually sold in competition with fresh produce imported from warmer climates, the stored Canadian product must be of high quality. Therefore, diseases and pests of stored vegetables are a major concern to producers, processors, wholesalers and retailers.

(Original by R.P. Jaques and W.R. Jarvis)

1.2 Field and garden vegetable crops

In Canada, most of the commercially significant vegetable crops are produced in the field, and statistics on production areas and values are reported regularly (see Table 1.2a). There also is a significant commercial production of protected crops, i.e. those grown in greenhouses or under other conditions of modified environment. A wide variety of vegetables also are grown in home gardens from coast to coast and in some areas of the Yukon and Northwest territories. The common and scientific names of the major and minor vegetable crops grown in Canada are listed in Table 1.2b.

Asparagus is an early season crop that is grown mainly in Ontario, Quebec and British Columbia under a wide range of soil and climatic conditions. New shoots are produced annually from a perennial root and harvested as they emerge, starting in early spring. If not cut for food, they eventually become the fern of the asparagus plant. The harvest usually extends three to seven weeks. Afterwards, shoots are grown to maturity to restore carbohydrates for the next year. In Ontario, about 70% of the crop is sold fresh in retail stores or at roadside stands, whereas most of the British Columbia crop is processed into canned or frozen products. There is a limited market for asparagus fern in the commercial floriculture industry.

Table 1.2a. Commercially significant vegetable crops in Canada

Crop	Area planted ('000 hectares)	Production ('000 tonnes)	Farm value ($ million)	Major production by province (%)					
Asparagus	1.7	3.2	7.6	Ont.	72.8	Que.	18.4		
Bean	7.8	43.4	18.0	Que.	43.2	Ont.	38.7		
Beet	0.9	14.5	4.7	Que.	49.5	Ont.	36.0		
Cabbage	4.8	122.2	27.6	Que.	48.6	Ont.	29.1		
Carrot	7.7	297.0	61.0	Que.	43.6	Ont.	40.7	N.S.	6.4
Cauliflower	3.0	40.3	21.0	Ont.	52.6	Que.	29.1	B.C.	6.1
Celery	0.8	38.6	13.5	Que.	51.3	Ont.	40.4	B.C.	5.9
Cucumber, field	2.8	45.3	14.6	Ont.	57.0	Que.	35.9	B.C.	3.2
Cucumber, greenhouse	0.1	7.1*	43.3	Ont.	53.6	B.C.	20.2	Alta.	16.8
Lettuce	2.9	67.4	35.2	Que.	58.2	Ont.	22.9	B.C.	14.2
Maize (sweet corn)	35.7	314.2	60.6	Ont.	61.8	Que.	28.1	B.C.	6.7
Mushroom		53.7	147.9	Ont.	54.4	B.C.	30.6		
Onion (dry)	4.5	129.5	34.4	Ont.	50.6	Que.	41.0;	Man	3.8
Parsnip	0.4	2.1	1.4	Ont.	51.4	Man.	30.1		
Pea	19.5	68.8	23.4	Ont.	48.6	Que.	26.0;	B.C	8.7
Pepper	2.1	19.8	13.8	Ont.	71.7	Que.	25.2		
Potato	125.6	3530.0	361.6	P.E.I.	30.6	N.B.	18.7	Man.	15.3
Radish	0.8	6.1	4.0	Que.	41.6	Ont.	35.9	B.C.	22.5
Rutabaga	2.6	73.2	18.0	Ont.	43.7	Que.	32.3	N.S.	6.2
Spinach	0.5	2.4	1.9	Que.	40.2	B.C.	31.6	Ont.	28.2
Tomato, field	11.4	444.0	83.5	Ont.	96.6	Que.	2.8	B.C.	0.5
Tomato, greenhouse	0.1	29.8	46.2	Ont.	50.2	Que.	22.2	B.C.	22.0

*Greenhouse cucumber production in millions of dozens.

Source: Statistics Canada catalogues 22-003, 1992, 1993; 22-202, 1992; data for 1992.

Bean *(Phaseolus vulgaris*) is grown for consumption as an edible pod for fresh use or processing (snap, green, yellow and wax bean) or as a dry seed (field bean). Modern cultivars are stringless. Beans for fresh pods are grown in most regions of Canada, with production for processing mainly in Ontario and Quebec. Average annual production of green, yellow and wax beans for the period 1980-1987 was 46 000 tonnes from an area of 8300 hectares, most (93%) of which occurred in Quebec, Ontario, Nova Scotia and British Columbia. Broad bean (*Vicia faba*) is a spring-sown crop in Canada and is popular in home gardens for use as a fresh, frozen or dried product.

Beets consist of four distinct agronomic groups: the garden or table beet, cultivated for its edible root and tops; Swiss chard, grown for its edible leaves and petioles (see below, Swiss chard); sugar beet, used for commercial sugar production; and fodder beet, used for livestock. Table beet production occurs in all provinces, with the greatest concentration in Quebec and Ontario.

Cabbage (see Crucifers)

Carrot is consumed primarily as a fresh vegetable and only relatively small quantities are processed. Approximately 7700 hectares of carrot are grown each year, mainly in the muck soil regions of Ontario and Quebec, and to a lesser extent on inorganic soils in Ontario, Nova Scotia and Alberta. Together, Quebec and Ontario are responsible for 80 to 85% of the total Canadian production. About 70% of this country's carrots are grown on organic soils. Canada imports about 15% more carrots than are exported each year.

Celeriac is grown on a small scale in Canada.

Celery is grown in Canada on about 800 hectares. Quebec has the largest production, followed by Ontario, British Columbia and Manitoba. Despite Ontario being a major producer for both fresh and processing domestic needs, imports exceed the value of home-grown product.

Chicory is grown by specialist producers, mainly around major urban centers, for markets as a salad and culinary vegetable. It is grown in the field for one season, then the deleafed roots are lifted, cool-stored and forced in darkness for the tight head of

smoothly folded, blanched leaves. The roots, particularly of the crinkly leaved cultivars, are dried, roasted and ground as a coffee substitute or additive. They also may be cooked and eaten.

Corn (see Maize)

Crucifers are grown on approximately 15 000 hectares distributed quite evenly across the country. Cabbage and cauliflower are the predominant crops. Broccoli, Brussels sprouts, Chinese cabbage, kale, kohlrabi, radish, rutabaga and summer turnip are less common and production is often concentrated in particular regions. The horticulturally important cruciferous crops belong to thirteen species in three genera within the family Cruciferae. Crops belonging to *Brassica oleracea* L. are collectively known as cole crops. Among these, production of cabbage is greatest, followed by cauliflower, broccoli and Brussels sprouts. Kale and kohlrabi are of minor importance. Kale is grown mostly for animal fodder, and some cultivars are grown as ornamentals. Chinese cabbage, though not produced on a large scale, has increased in importance as a specialty vegetable in recent years. Rutabaga is produced chiefly in Ontario, Quebec and the Atlantic provinces. Radishes are produced mainly in Quebec, Ontario and British Columbia.

Table 1.2b. Common and scientific names of vegetable crops

Common name	Scientific name
Alfalfa sprouts	*Medicago sativa* L.
Anise	*Pimpinella anisum* L.
Applemint	*Mentha suaveolens* J.F. Ehrh.
Asparagus	*Asparagus officinalis* L.
Balm, lemon	*Melissa officinalis* L.
Basil	*Ocimum basilicum* L.
Bean, common	*Phaseolus vulgaris* L.
Bean, broad, field	*Vicia faba* L.
Bean, lima	*Phaseolus lunatus* L.
Bean, scarlet runner	*Phaseolus coccineus* L.
Bean sprouts (mung bean)	*Vigna radiata* (L.) Wilczek
Beet, garden	*Beta vulgaris* L.
Beet, sugar	*Beta vulgaris* L.
Borage	*Borago officinalis* L.
Broccoli	*Brassica oleracea* var. *italica* Plenck.
Brussels sprouts	*Brassica oleracea* var. *gemmifera* DC.
Burnet	*Sanguisorba officinalis* L.
Cabbage, Chinese	
(wong-bok)	*Brassica pekinensis* (Lour.) Rupr.
(pak-choi)	*Brassica chinensis* L.
Cabbage, white, red & savoy	*Brassica oleracea* var. *capitata* L.
Calabrese	*Brassica oleracea* var. *italica* Plenck.
Cantaloupe	*Cucumis melo* var. *cantalupensis* Naud.
Caraway	*Carum carvi* L.
Carrot	*Daucus carota* subsp. *sativus* (Hoffm.) Arcang.
Casaba	*Cucumis melo* var. *inodorus*
Cauliflower	*Brassica oleracea* var. *botrytis* L.
Celeriac	*Apium graveolens* var. *rapaceum* (Mill.) Gaud.
Celery	*Apium graveolens* var. *dulce* (Mill.) Pers.
Chard, Swiss	*Beta vulgaris* subsp. *cicla* (L.) Moq.
Chicory	*Cichorium intybus* L.
Chive	*Allium schoenoprasum* L.
Citron	*Citrullus lanatus* (Thunb.) Matsum. & Nakai
Collard	*Brassica oleracea* var. *acephala* DC.
Coriander	*Coriandrum sativum* L.
Corn (see Maize)	
Corn-salad	*Valerianella locusta* (L.) Betcke
Cucumber, Armenian	*Cucumis melo* L.
Cucumber, lemon	*Cucumis sativus* L.
Cucumber, long English	*Cucumis sativus* L.
Cucumber, pickling	*Cucumis sativus* L.
Cucumber, slicing	*Cucumis sativus* L.
Cucumber, snake	*Cucumis melo* var. *flexuosus* Naud.
Dill	*Anethum graveolens* L.

Eggplant	*Solanum melongena* var. *esculentum* Nees
Endive	*Cichorium endivia* L.
Fennel	*Foeniculum vulgare* Mill.
Fenugreek	*Trigonelia foenum-graecum* L.
Fern, ostrich	*Matteuccia struthiopteris* (L.) Todaro
Fiddlehead	*Matteuccia struthiopteris* (L.) Todaro
Garlic	*Allium sativum* L.
Gherkin	*Cucumis sativus* L.
Gherkin, West Indian	*Cucumis anguria* L.
Ginseng	*Panax quinquefolius* L.
Gourd, ornamental	*Cucurbita maxima* Duch.
Gourd, ornamental	*Cucurbita pepo* L.
Hop	*Humulus lupulus* L.
Horsemint	*Mentha longifolia* (L.) Huds.
Horseradish	*Armoracia rusticana* Gaertn., Mey. & Scherb.
Jerusalem artichoke	*Helianthus tuberosus* L.
Kale	*Brassica oleracea* var. *acephala* DC.
Kohlrabi	*Brassica oleracea* var. *gongylodes* L.
Lavender	*Lavandula angustifolia* Mill.
Leek	*Allium porrum* L.
Lettuce	*Lactuca sativa* L.
Maize (sweet corn)	*Zea mays* L.
Marrow, vegetable	*Cucurbita pepo* L.
Melon, honeydew	*Cucumis melo* L.
Melon, serpent	*Cucumis melo* var. *flexuosus* Naud.
Mint	*Mentha* spp.
Mushroom, button	*Agaricus bisporus* (Lange) Imbach
Mushroom, oyster	*Pleurotus* spp.
Mushroom, shiitake	*Lentinus edodes* (Berk.) Pegler
Muskmelon	*Cucumis melo* var. *reticulatus* Naud.
Mustard, brown	*Brassica juncea* (L.) Czern. & Coss.
Mustard, white	*Sinapis alba* L.
Mustard, yellow	*Sinapis alba* L.
Mustard greens (rappini)	*Brassica juncea* (L.) Czern. & Coss.
Onion, bunching	*Allium fistulosum* L.
Onion, common or cooking	*Allium cepa* L.
Onion, Egyptian	*Allium cepa* L.
Onion, multiplier	*Allium cepa* L.
Onion, Spanish	*Allium fistulosum* L.
Onion, Welsh	*Allium fistulosum* L.
Ostrich fern (see fiddlehead)	
Parsley	*Petroselinum crispum* (Mill.) Nym: A.W. Hill
Parsnip	*Pastinaca sativa* L.
Pea	*Pisum sativum* L.
Pennyroyal	*Mentha pulegium* L.
Pepper	*Capsicum annuum* L.
Peppermint	*Mentha* x *piperita* L.
Potato	*Solanum tuberosum* L.
Pumpkin	*Cucurbita maxima* Duch.
Pumpkin	*Cucurbita mixta* Pangalo
Pumpkin	*Cucurbita moschata* Duch.
Pumpkin	*Cucurbita pepo* L.
Radish	*Raphanus sativus* L.
Rappini (see Mustard greens)	
Rhubarb	*Rheum rhabarbarum* L.
Rutabaga (swede)	*Brassica napus* var. *napobrassica* (L.) Reichb.
Sage, garden	*Salvia officinalis* L.
Savory, summer	*Satureja hortensis* L.
Shallot	*Allium cepa* L.

Spearmint, common	*Mentha spicata* L.
Spearmint, Scotch	*Mentha* x *gentilis* L.
Spinach	*Spinacia oleracea* L.
Squash, acorn	*Cucurbita pepo* L.
Squash, butternut	*Cucurbita moschata* Duch.
Squash, crookneck	*Cucurbita moschata* Duch.
Squash, summer	*Cucurbita pepo* L.
Squash, winter (most)	*Cucurbita maxima* Duch.
Squash, zucchini	*Cucurbita pepo* L.
Sugar beet	*Beta vulgaris* L.
Tarragon	*Artemisia dracunculus* L.
Thyme, garden	*Thymus vulgaris L.*
Tomato	*Lycopersicon esculentum* L.
Turnip, summer Turnip (see also rutabaga)	*Brassica rapa* L.
Watermelon	*Citrullus lanatus* (Thunb.) Matsum. & Nakai
Zucchini	*Cucurbita pepo* L.

Cucurbits are grown chiefly in Ontario and Quebec, where the gross farm value of field cucumbers alone is $12 million. In Canada, annual production of field cucumbers is about 45 000 tonnes worth $14 million. Cucurbit crops include seven species in three genera and contain most of the horti- culturally important vine crops, such as pumpkins, summer squash, zucchini, vegetable marrow, acorn, butternut and other winter squashes, ornamental gourds and squashes, gherkins, pickling, slicing, lemon, long English, Armenian and snake cucumbers, musk- and honeydew melons, casabas, watermelon and citrons. In addition, *Benincasa hispida* Cogn., *B. cerifera* Savi and *Cucurbita ficifolia* Bouché are sometimes used as disease- and nematode-resistant rootstocks (see also Protected vegetable crops, 1.3).

Eggplant production in Canada is mainly in southern Ontario. The plants are started indoors and grown in heated frames before being transplanted to the field (see also Protected vegetable crops, 1.3).

Endive, which is botanically related to chicory, is grown commercially mainly in Ontario, Quebec and British Columbia. The leaves are tied in a bunch and blanched in darkness toward the end of the first growing season. Like chicory, the roots can be lifted for winter forcing.

Fiddleheads are derived from the ostrich fern, which occurs across Canada. Fiddleheads, harvested from wild ostrich-fern stands, are the young, vegetative fronds picked in the spring. Originally, they were a traditional part of the diet of the Maliseet Indians of New Brunswick. Fiddleheads are sold in fresh or frozen form throughout Canada and the United States. Their availability, currently estimated at between 250 000 and 500 000 kg annually, has never been sufficient to satisfy market demand.

Herbs and spices include nearly 50 plant species and cultivars grown primarily in home gardens and by specialist producers throughout Canada. Mustard *(Brassica* and *Sinapis* spp.) is grown on a large scale, but most herb and spice production occurs on small farms near large urban centers. Fresh and dried herbs are field-grown during the summer months and greenhouse-grown in the winter. The leaves, stems, flowers and flower buds, roots, fruits and seeds of these plants are used fresh, dried, frozen, or in oil as flavorings and condiments in salads or for cooking.

Hop is a perennial herb that sends up new vines each year. Traditional cultivars require well-constructed, wire trellises up to six metres high. The cones mature in the fall and are removed from the vines, kiln dried and baled. They are used to flavor and condition beer. Although hop is native to various parts of Canada and has been cultivated in Ontario, Quebec and Manitoba, it is now grown commercially only in coastal British Columbia. Production in that province has declined from about 800 hectares in the mid 1940s to 325 hectares in 1989; meanwhile, the monetary value of the crop has increased. Domestic production is sold in Canada, Britain and the United States.

Horseradish is a hardy, perennial cruciferous crop that is grown in home gardens in all regions except the far north in Canada. The tuberous root is used to make a relish condiment and increasingly in Manitoba to supply a peroxidase market.

Jerusalem artichoke, or sunchoke, is a native North American perennial that grows wild in parts of Canada. It has been variously used as a food plant and for fodder. It is closely related to the sunflower and shares its diseases and pests. The swollen, tuber-like rhizomes of the plant can be eaten raw or lightly cooked and were used as a starch vegetable by aboriginal peoples. In recent years, the Jerusalem artichoke has attracted interest as a source of fuel-grade ethanol. The fortunes of the crop have thus been closely linked to world prices for the more traditional fossil fuels. This plant can become a persistent weed if it escapes cultivation.

Lettuce is grown widely for its leaves in Quebec, Ontario and British Columbia. Crisphead or iceberg lettuce is most popular for field growing, and butterhead and leaf lettuce are widely used for greenhouse production. In Ontario, 65% of fresh-market head lettuce is produced on organic soils in York and Simcoe counties, but head lettuce also is grown in Lambton, Niagara and other counties in southern Ontario and to a lesser extent in eastern Ontario. The main production area in British Columbia is the Fraser River delta (see also Protected vegetable crops, 1.3).

Maize, as sweet corn, is grown commercially on about 36 000 hectares, and in home gardens across Canada for fresh consumption. Commercial production of processing corn is very important, particularly in the counties of Ontario that border Lake Erie. Fresh-market sweet corn, although lower in yield per hectare and tonnes produced, gives a greater price per unit-weight than corn for processing. For example, in 1990, the Ontario fresh-market crop had a farm-gate value of $31.6 million, compared to $13.5 million for the processing crop. Sweet-corn types have only been known since the early 1800s, probably arising from a mutation of a dent corn cultivated by the aboriginal peoples around the Great Lakes. Sweet corn differs from dent corn in the possession of the recessive su-gene, which prevents the conversion of sugar to starch within the kernel. In Ontario, sweet corn has not changed substantially for many years, the major cultivar being Jubilee. The supersweet cultivars, distinguished by the "shrunken" or sh2-gene, have received positive market acceptance. However, many of the new lines are poor field performers, and efforts are being made to improve their emergence characteristics through seed treatments, plant breeding, changes in planting date and other cultural practices. Although sweet corn is affected by the same diseases and pests as field corn, the relative importance of different problems varies greatly.

Mint is grown either as a fresh or dried herb or for its oil, which is steam distilled from the foliage. There are about 25 species of mint worldwide. Production in Canada consists of small plantings of peppermint, common spearmint, Scotch spearmint, applemint, pennyroyal and several minor species. Field-scale planting of mint for oil production is centered primarily in southern Alberta.

Onion and other *Allium* **crops** Approximately 4500 hectares of common dry bulb onion are grown in Canada, with about 90% of the production in Ontario and Quebec. Most dry bulb onions are grown on organic muck soils at Bradford, Leamington, Thedford and Grand Bend in Ontario and Saint-Jean-sur-Richelieu, Quebec, and on inorganic soils in Manitoba and southwest British Columbia. Canada imports two to four times as many dry bulb onions as are exported each year. Other *Allium* crops, such as Spanish and bunching onions, leek, garlic and shallots, are grown on approximately 800 hectares. Chive also is a popular home-garden plant that often is potted and grown indoors.

Parsley, as leaf parsley, is an excellent source of vitamins A and C. It is commonly used as a garnish by the food service industry. Cultivars with curled leaves are most popular for this purpose and, in Canada, they are grown in market gardens wherever leafy salad vegetables are produced. Leaf parsley also can be dried and flaked for use as a seasoning. Root parsley is grown less commonly. The taproot is used as a food flavoring and seasoning.

Parsnip is a minor crop in most provinces. Most of Canada's production is centered in Ontario, Quebec and the Atlantic provinces.

Pea is grown in home gardens as a fresh vegetable and commercially for seed and for dry edible and processing uses. In 1992 vegetable pea production was 69 000 tonnes, with 75% in Ontario and Quebec, and with a farm value of $23.4 million. Southern Alberta and interior British Columbia are the best seed-producing areas. Large-scale production of dry edible pea occurs across the Prairie provinces. Processing pea is grown mainly in southwestern Ontario. The green, wrinkle- seeded garden pea, harvested immature for canning and freezing, differs slightly from the yellow, smooth-seeded field pea that is harvested fully mature, but both are affected by the same diseases and pests.

Pepper is grown in the field on a small scale in Canada, mainly in southwestern Ontario and Quebec. Losses from pest damage have reached 50% or more within individual fields. Because of the high standard of pest-free product required by processors, annual losses from product rejection would reach 75% if control measures were not applied. Sweet pepper is an increasingly important greenhouse crop in British Columbia, Alberta and Ontario (see also Protected vegetable crops, 1.3). Various hot peppers are grown locally for specialty markets.

Potato is the most important vegetable crop in Canada. Production is regionally concentrated but distributed across southern Canada, the major producing provinces being Prince Edward Island, New Brunswick, Manitoba, Quebec, Ontario, Alberta and British Columbia, in that order. Home-garden production occurs in every province and territory. More than 125 000 hectares of potato were grown across the country in 1992, with an average yield of 28.5 tonnes per hectare.

In Canada, this crop is subject to over 50 infectious and physiologic diseases and pests, many of which are frequent causes of major losses in both quality and quantity. To avoid the build-up of fungal, bacterial and viral diseases in vegetatively propagated stocks, Canada has rigid seed potato certification regulations. In many provinces, potato cultivars are routinely tissue cultured to provide disease-free seedlings and then are grown for up to seven generations (classed as Nuclear, Pre-Elite, Elite I, Elite II, Elite III, Elite IV, Foundation, and Certified) prior to being sold for seed increase, table stock production or home-garden use. Each generation is inspected for diseases by Agriculture Canada inspectors on farms that have been approved for growing potato seed. The provinces of Alberta, Prince Edward Island, and New Brunswick have laws that compel all commercial potato growers to plant Certified or higher classes of seed.

The certification system provides for monitoring of disease levels in the seed potato crop. There is a zero tolerance for diseases such as bacterial ring rot (see Introduced diseases and pests, 3.11). For some tuber-borne diseases, such as fusarium wilt and blackleg, and for some viruses, there are maximum permissible levels of infection at each certification class. For example, the level of blackleg in an Elite III crop must not exceed certain established guidelines; otherwise the potatoes may be downgraded to a lower class. If disease levels exceed the official level, then certification is denied. Potato losses are considerable

every year in Canada; without this rigid system of seed certification, losses from tuber-borne diseases would make growing the crop uneconomical.

Rhubarb is grown both for fresh use and for processing in plantings ranging in size from a few hectares in British Columbia, central Canada and the Maritime provinces, to backyard gardens in all parts of the country. It also can be forced in heated sheds using crowns brought in from the field. Rhubarb is popular in home gardens because it provides some of the first produce in the spring. The leaf petiole is used as a cooked desert, in pies and to make wine.

Spinach is grown commercially in Ontario, Quebec and British Columbia for use as an ingredient of fresh salads or as a cooked vegetable. It is also an occasional greenhouse crop, where it can be grown in hydroponic culture.

Swiss chard is a minor crop grown throughout Canada, primarily in home gardens, because it is valued for its leafy petioles, which are cooked. A second year's growth can be forced by removing the seed stalks as they emerge.

Tomato ranks second to potato in farm value among field vegetable crops grown in Canada. Ontario produces more than 95% of the Canadian crop, and 83% of that is concentrated in Essex and Kent counties in the southwest. Approximately 80% of all field tomatoes are processed, the remainder being used for fresh-market consumption. Traditionally, transplants have been imported from the southern United States, principally Georgia, to establish the crop, but the use of transplants grown in plugs of soil in plastic trays in Canadian greenhouses is increasing substantially. In Ontario, tomato crops are transplanted from May 1 to mid-June and harvested from mid-August to the end of September. Most tomatoes for processing are harvested by machine, and hand picking is now almost exclusively confined to tomatoes for fresh-market sales. The average yield for processing tomatoes in Ontario is approximately 48 tonnes per hectare. Because of the value of the tomato crop and the need to use lighter, sandier soils for mechanical harvesting, crop rotation is limited, resulting in heavier pest pressures (see also Protected vegetable crops, 1.3).

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▶ 1.3 Protected vegetable crops

Protected crops grown in Canada include alfalfa and bean sprouts, ginseng, some herbs and spices, mushrooms, and greenhouse cucumber, lettuce, pepper and tomato. The techniques and facilities used to grow these crops are often sophisticated and expensive; however, production costs are offset by high returns per unit area of production and the capability of growing successive crops, in many cases year round.

Traditionally, greenhouse lettuce, endive, chicory, cucumber and tomato were grown in soil groundbeds variously amended with peat, manure, straw, peanut hulls or other organic materials. However, since the 1970s, there has been an increasing use of soilless media in both commercial and home greenhouses. In British Columbia and to some extent in Alberta and the Atlantic provinces, tomato and cucumber are grown in bags of sawdust; rockwool and other synthetic substrates are used throughout the country. Crops of high-quality lettuce are grown by the nutrient film technique (NFT) in a few locations.

Soilless production was introduced largely to enable producers to better regulate crop nutrition and environmental factors, such as root-zone temperature and pH, and to reduce the costs of soil disinfestation for root disease control. In the latter case, however, some diseases, such as corky root rot of tomato and black root rot of cucumber, have been more severe in rockwool than in soil. This often results from breaches in hygiene whereby the soilless substrate, with a greatly reduced antagonistic microflora, becomes contaminated with soil-borne or water-borne pathogens.

Control of diseases in hydroponically grown crops presents special problems. Experience has shown that solid substrates, such as rockwool, perlite and other inert materials, as well as sawdust, require pasteurization, especially if recycled. NFT gullies, reservoirs and tubing have to be cleansed thoroughly and disinfested by hypochlorite solutions or other sterilants. The contamination of water supplies, particularly with *Pythium* and *Phytophthora* species, bacteria, and the *Olpidium* fungal vectors of viral diseases, is a constant threat. Growers who use water from wells or creeks, or who store water in outdoor or even indoor reservoirs, should consider installing a water treatment system that uses heat, ozone, ultraviolet light, chlorine or ultrafiltration to remove or inactivate pathogens. Plants for hydroponic production should be raised only in an inert substrate, never in soil or peat. In one survey, all of 52 commercial peats examined contained *Fusarium* spp., and 15 contained *Pythium* spp., many of them potential pathogens.

Alfalfa sprouts are produced on a limited commercial scale in Canada. A crop can be raised in four to five days, giving diseases and pests little time to become established. Marketing in a sealed container (punnet) eliminates many potential pests entirely.

Bean sprouts are seedlings of the mung bean or green gram that are eaten raw as a salad vegetable and used in Chinese cooking. Seeds are germinated in shallow water in a high humidity. One gram of seed yields six to eight grams of sprouts. Mung bean also is grown for seed and for its pods, which are eaten fresh.

Ginseng is native to eastern North America. It has been a minor commercial crop in Canada since about 1900, primarily in Ontario and British Columbia. It is produced from stratified seed in shaded plots known as gardens. The root is the portion of the plant that is processed commercially, but the seeds are of value, and in the Orient the leaves also are used. In North America, the

root is dried before being sold. It is processed into pickles, powders, or extracts. Nearly all Canadian ginseng is exported to the Orient for processing. In 1988, Canada produced about 160 000 kg of root. Ginseng has a number of serious diseases but little research has been done on diseases and pests of this crop.

Greenhouse vegetables are grown in soil and in soil-free culture. In 1991, approximately 312 hectares were devoted to greenhouse vegetable production in Canada. Ontario leads in greenhouse tomato production, but other provinces, especially British Columbia and Quebec, also grow a considerable amount. Soilless culture helps to avoid soil-borne diseases, but high standards of crop hygiene must be maintained to avoid introducing pathogens and pests to the cropping system. In Canada, the most widely grown greenhouse vegetable crops are tomato, cucumber, pepper and lettuce. The annual value of greenhouse vegetable production in 1992 has been estimated at $113 million (Statistics Canada, Cat. 22-202, 1992). The most popular types of greenhouse vegetables include red- and pink-fruited tomatoes, long English cucumber, sweet bell pepper and butterhead lettuce. White-spine cucumber, hot pepper and leaf lettuce also are grown, but to a lesser extent.

Herbs and spices for supermarket retail and restaurant use are grown in greenhouses during the winter months.

Mushrooms are fungi that produce a conspicuous fruiting body. In North America, there are more than 3000 species, of which about one-fifth are edible, but only a very few species are grown commercially. Mushroom cultivation in Canada began about 1912, when they were grown experimentally beneath greenhouse benches in eastern Canada. Canadian fresh market production has developed to the point that mushrooms are the second most valuable vegetable crop, with a farm-gate value of $148 million in 1992. More than 50% of this production occurred in Ontario, and 30% in British Columbia (Statistics Canada, Cat. 22-003, 1993). The commercial button mushroom is the main type cultivated in Canada, accounting for 99% of the total production; other types grown here are the oyster mushroom and the shiitake or black forest mushroom. Canada also imports processed mushrooms.

Vegetable sprouts available in the marketplace in addition to those of alfalfa and bean include onion and radish, but specific disease and pest problems have not been reported.

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▶ 1.4 Insects as pollinators of vegetable crops

Insect pollination is important to vegetable crop production in Canada because some crops require cross-pollination by insects in order for any produce to be harvested. The insects involved are mostly honeybees (*Apis mellifera* L.).

Insect pollination is absolutely required for cucurbit crops, such as cucumber, melon, pumpkin and squash. In *Cucumis* and *Cucurbita*, the vines produce unisexual flowers, although the plants are bisexual. Pollen of these plants is typically large, spiny and oily, showing clear adaptations to insect pollination. Even though melon is self-fertile, insects are needed to effect pollination. Cross-pollination seems to be required in *Cucumis* and *Cucurbita* to obtain seeds and, in most cultivars, fruit. An exception is the long English cucumber, which forms a desirable, seedless fruit if not pollinated; if the flowers are pollinated, the fruits become misshapen (bull-necked), seeded and bitter. Honeybees are used for pollination of most cucurbit crops in North America (see Table 1.4). In Holland, the keeping of honeybees is prohibited in some areas during the summer to avoid the production of malformed cucumbers.

Although honeybees are the most manageable pollinators, they are not the best-suited for some vegetable crops. Pollination of squash and pumpkin is carried out effectively by the solitary squash bee *Peponapis pruinosa* (Say). This native bee is well established in southern Ontario and is probably the main pollinator at some locations. Squash bees emerge from underground nests in synchrony with the blooming of squash and pumpkin. The bees are active on the newly opened flowers from just before dawn, when the flowers open for their short, morning-long life. The female squash bees gather pollen to provision their nests. They have all but finished their pollinating activity by the time honeybees begin to forage. Populations of *P. pruinosa* in the vicinity of squash and pumpkin fields are immediately beneficial to productivity.

Some crops in the pea and bean family (Leguminosae) require pollination, but most are self-fertile and self-pollinating (see Table 1.4). However, scarlet runner bean requires pollination, which is accomplished mostly by insects or sometimes by hummingbirds. Bumblebees are especially adept at pollinating scarlet runner bean and are used in Europe in greenhouse production of this crop. The importance of insect pollinators in other species and cultivars of pea and bean is difficult to judge, the available data indicating no effect or some increase in crop yield (see Table 1.4).

Solanaceous vegetables, such as pepper, tomato and eggplant, usually produce fruit well when grown outdoors. Self-pollination, which is brought about by wind, agitation and gravity, seems to be effective. However, in greenhouses, pollination usually is incomplete unless assisted. Traditionally, growers ensure the transfer of pollen by touching the flower with an "electric-bee" vibrating rod. Recently, culturing of the bumblebee *Bombus terrestris* (L.) has taken on importance in Europe for pollination of these solanaceous crops, especially tomato. The successes there have spurred interest in Quebec, Ontario and British Columbia in North American species of bumblebees. Honeybees work effectively for pepper production in greenhouses.

Some seeds, apart from sprouted beans and alfalfa, also are used as vegetables. One example, sweet corn, requires cross-pollination, which occurs by wind. Newer cultivars of sunflower are self-pollinating, but the older cultivars require cross-pollination by insects. Some native bees, such as *Eumegachile pugnata* (Say), are specialist pollinators of sunflowers and may be useful on Jerusalem artichoke in some areas. Although it is assumed that many umbelliferous flowers require cross-pollination to set seed, the pollination requirements for anise, caraway, celery, coriander, dill, and fennel are incompletely understood or unknown (see Table 1.4). Generally, these plants are visited by a wide variety of insects; it is recognized that insects are necessary to bring about pollination, because the sexual parts of individual florets mature at different times.

Almost all vegetables grown in Canada are propagated from seed, notable exceptions being asparagus, garlic, Jerusalem artichoke, onion, potato, rhubarb and some herbs. Among cruciferous crops, cross-pollination by insects is the rule (see Table 1.4). It is also the generally accepted condition for onion and allied crops, for such umbelliferous crops as carrot, celery, dill, fennel, parsnip and caraway, and for salsify and Jerusalem artichoke. The pollinators involved are varied and include honeybees, other bees, flies, moths and other insects. Beet, chard and spinach require cross-pollination by wind, although insects may visit the flowers of male plants for pollen, which is also the case with corn.

Table 1.4. Pollination requirements of vegetable crops grown in Canada

Crop plant	Pollinating mechanism	Product of pollination	Remarks	Crop plant	Pollinating mechanism	Product of pollination	Remarks
Anise	insects	sf, sp		Gherkin	bees	F, sp, sb	
Applemint	?bees	sp		Ginseng	insects	sp, sb	
Asparagus	insects	sp, sb		Gourd, ornamental	bees	F, sf, sp, sb	
Balm, lemon	?bees	sp		Hop	wind	F, sp, sb	
Basil	?insects	sp		Horsemint	?bees	sp	
Bean, common	mostly selfing	F, sf, sp, sb		Horseradish	insects	sp	
Bean, broad	bees, selfing	sf, sp, sb	see Note 1	Jerusalem artichoke	unknown	sb	
Bean, field	bees, selfing	sf, sp, sb	see Note 2	Kale and collard	insects	sp, sb	
Bean, lima	much selfing	sf, sp, sb		Kohlrabi	insects	sp, sb	
Bean, scarlet runner	bumblebees best	F, sf, sp, sb		Lavender	bees, insects	sp	see Note 4
Beet	wind, insects	sp, sb		Leek	insects	sp, sb	
Borage	bees	sp		Lettuce	selfing, insects	sp, sb	
Broccoli or calabrese	insects	sp, sb		Maize, sweet com	wind	sf, sp, sb	
Brussels sprouts	insects	sp, sb		Marrow, vegetable	bees	F, sf, sp, sb	
Burnet	unknown	sp		Melon, honeydew	bees	F, sf, sp, sb	
Cabbage, Chinese types	?insects	sp, sb		Mint	bees	sp	
Cabbage, white, red	insects	sp, sb		Muskmelon	bees	F, sf, sp, sb	
Cabbage, savoy	insects	sp, sb		Mustard, yellow	insects	sf, sp, sb	
Caraway	insects	sf, sp		Onion, all types	insects	sp, sb	
Carrot	insects	sp, sb		Parsley	insects	sp	
Casaba	bees	F, sf, sp, sb		Parsnip	insects	sp, sb	
Cauliflower	insects, selfing	sp, sb		Pea	mostly selfing	F, sf, sp, sb	
Celeriac	insects	sp		Pennyroyal	?bees	sp	
Celery	insects	sf, sp, sb		Pepper	selfing, bees	F, sf, sp, sb	
Chard, Swiss	wind	sp, sb		Peppermint	?bees	sb	
Chicory	insects	sp, sb		Potato	bees, hand	sb	
Chive	insects	sp, sb		Pumpkin, all types	bees	F, sf, sp, sb	
Citron	bees	F, sf, sp, sb		Radish	insects	sp, sb	
Coriander	insects	sf, sp		Rhubarb	insects, wind	sb	
Corn-salad	?insects	sp		Rutabaga or swede	insects	sp, sb	

Cucumber, Armenian	bees	F, sp, sb		Sage	?bees	sp	
Cucumber, lemon	bees	F, sp, sb		Savory	unknown	sp	
Cucumber, long English	parthenocarpy	F		Shallot	n/a	n/a	
Cucumber, pickling	bees	F, sp,sb		Spearmint	?bees	sp	
Cucumber, slicing	bees	F, sp, sb		Spinach	wind	sp, sb	
Cucumber, snake	bees	F, sp, sb		Squash, all types	bees	F, sf, sp, sb	
Dill	insects	sf, sp		Tarragon	n/a	n/a	see Note 5
Eggplant	bees, selfing	F, sp, sb	see Note 1	Thyme	bees	sp	
Endive	selfing	sp		Tomato	bumblebees, hand	F, sp, sb	
Fennel	insects	sp		Turnip, summer	insects	sp, sb	see Note 6
Fenugreek	unknown	sf, sp		Turnip, winter/table	insects	sp, sb	
Fiddlehead	n/a	n/a		Watermelon	bees	F, sf, sp, sb	
Garlic	none	n/a	see Note 3	Zucchini, see squash			

Symbols:

F	fruit used as human food
sb	seed used for breeding
sf	seed used as human food
sp	seed used for planting
n/a	not applicable
?	suspected

Notes:

1 pollination by bees increases seed set
2 pollination by bees does not seem to increase seed set
3 flowering is rare; if produced, seed is mostly non-viable
4 self-pollination results in seed abortion (but greater oil yield)
5 propagated by cuttings
6 self-pollination results in less seed, and seed of poorer quality, than does cross-pollination

Cross-pollination is important to vegetable breeding, even in plants propagated vegetatively, e.g. asparagus, horseradish, onion, potato and rhubarb. There are special techniques for the management of insect pollinators in vegetable seed-production enclosures. The insects used include honeybees, bumblebees, orchard bees *(Osmia* spp.), leafcutting bees *(Megachile* spp.), flower flies (family Syrphidae), blow flies (family Calliphoridae), and house flies (family Muscidae).

As the technology of vegetable production and improvement advances, pollination needs to be carefully considered. At present, the diversity and quality of insects as pollinators, and the botanical aspects of pollination technology for efficient cropping of vegetables are neglected areas of research in Canada.

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(Original by P.G. Kevan)

2 Crop losses and their causes

Figures *2.3a-q; 2.3T1*

Tables 2.3a-d

Additional references

Tables

2.1 Types of crop losses

Production losses

Diseases, insects, weeds and other pests annually cause substantial losses in the yield and quality of vegetables produced in Canada. Reliable estimates of these losses are not available, but they probably are proportional to losses in the USA. Even with the extensive application of pesticides, the estimated reductions in the farm-gate value of selected vegetable crops in the United States caused by diseases range from 8 to 23%, by insects 4 to 21%, and by weeds 8 to 13%. If it is accepted that the average losses caused by diseases, insects and weeds in Canada are 15.5, 12.5 and 10.5%, respectively, they would have reduced returns to the vegetable industry by $172.7, $138.2 and $115.2 million, respectively, in 1990. If the costs of crop protection practices were factored in, these figures would be even higher. In the United States in 1987, crop losses caused by diseases and insects in specific vegetables were, respectively: cole crops 9 and 13%, lettuce 12 and 7%, potato 20 and 6%, tomato 21 and 7%, sweet corn 8 and 19%, onion 21 and 4%, cucumber 15 and 21%, pea 23 and 4%, and pepper 14 and 7%. Losses in greenhouse lettuce, cucumber and tomato are similar, but pest damage may necessitate replanting the whole crop. Until resistant cultivars of tomato became available, this was regularly the case with fusarium crown and root rot.

Post-harvest losses

Reduced yield and quality from pest damage in the field may be equalled or exceeded by losses in storage. This is especially the case where freshly harvested produce is not rapidly cooled or where it is not transported and stored under controlled conditions. For example, it is not unusual to see truckloads of perishable vegetables parked on farms, at roadside truck-stops and at food terminals rapidly deteriorating in the full summer sun. Similarly, attempts to dry onions in primitive storages with humid air frequently result in wetter, not drier, onions in production areas of the Great Lakes region. Such crops are often destroyed by diseases, such as neck rot and sour skin. Poorly stored carrot, potato and cabbage crops also are subject to substantial losses.

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(Original by R.P. Jaques and W.R. Jarvis)

2.2 Causes of crop losses

Direct crop losses caused by diseases and pests may be measured as the proportion of crop not sold. In addition to losses in yield and quality in the field and later during storage and transport, there are many, less tangible ways in which diseases and pests exact an economic toll. For example, the fungus *Botrytis cinerea* may cause multiple but almost imperceptible ghost spot lesions on tomato fruit, which, depending on the rigor of official or consumer inspection, may result in little or no financial loss to the grower. However, the same fungus causing a single, girdling lesion on the stem of an indeterminate tomato cultivar will result in the total loss in yield from that plant, as often happens in the greenhouse.

Bacterial spot on processing tomatoes makes the skin very difficult to peel by standard factory procedures, so the skins have to be removed by hand, which is very expensive. On the other hand, buyers of fresh-market tomatoes at roadside stands may scarcely notice a few lesions of bacterial spot. Similarly, when cabbage is fermented to produce sauerkraut, or cooked, the lesions caused by thrips are very pronounced and unacceptable, whereas thrips damage may be of little consequence if the cabbage is finely chopped and used fresh in coleslaw.

Nematode damage to roots may be mechanical or chemical, thereby reducing root capacity to absorb and translocate water and nutrients, even when soil moisture is adequate. Some vegetable crops are tolerant of nematode damage, while others are highly sensitive. Seedlings and young transplants usually are especially susceptible. The distribution of nematodes in the soil, whether in the field or in the greenhouse, normally is uneven. Plant-parasitic nematodes may reduce crop yield and quality but other biotic and abiotic stresses on plants make it difficult to predict the impact of nematode damage. Losses may increase significantly if nematodes interact with other pathogens, such as fungi and viruses. In Canada, yield-loss data, where available, are generally restricted to a few crops within a limited geographical area.

Insects and mites may damage vegetable plants directly or indirectly. For example, larvae and adults of the Colorado potato beetle may extensively defoliate a potato plant, substantially reducing its photosynthetic capacity and resulting in significant reduction in yield of tubers or even death of the plant; however, infestations that occur late in the growing season may have little effect on yield. Direct damage also may be caused by wireworms that feed or burrow into tubers, and this damage may be augmented by rot caused by bacteria and fungi. Aphids and leafhoppers suck on the foliage of the plant, reducing its vigor, but these insects also may damage a crop indirectly by transmitting plant viruses. Reduction in the yield of greenhouse-grown tomato, resulting from extensive (piercing and sucking) feeding by adults and nymphs of the greenhouse whitefly and the two-spotted spider mite, is a less direct form of damage to the crop than is the feeding on the fruit of field- grown tomato by hornworms and cutworms. Consumption of foliage of cabbage plants by larvae of the cabbage looper or the imported cabbageworm reduces the vigor of the plant, resulting in a smaller head; these insects also may feed directly in the head, rendering it unmarketable, or on the outer wrapper leaves of fresh-market cabbage and cauliflower, downgrading marketability. Similarly, the presence of insects in a marketed product, such as heads of broccoli, may render the product unmarketable without visible evidence of feeding by the insect.

The economic significance of damage and the action threshold applied in deciding on measures to manage populations of pests depend on the severity of damage, the value of the crop, and the proposed end use of the crop. For example, the threshold approaches zero for species that cause damage directly to the part of the crop to be used by the consumer; these include the carrot rust fly in carrot and the corn borer in pepper and sweet com. On the other hand, low populations of pests that cause foliar damage but do not feed on or damage marketable parts of the plant may be tolerated, and thus the action threshold for implementation of control procedures is higher; examples include the Colorado potato beetle on potato and the cabbage maggot on cabbage. Similarly, relatively high numbers of the two-spotted spider mite and of the greenhouse whitefly can be tolerated on greenhouse-grown cucumber and tomato without affecting the yield of marketed product significantly. Action thresholds also may vary with the stage of development of the vegetable plant when attacked. For example, low populations of the imported cabbageworm and cabbage looper can be tolerated when they feed on the foliage of young plants of cabbage, cauliflower and Brussels sprouts. Later, however, the tolerance for these pests is greatly reduced when they feed on the head or wrapper leaves of cabbage or cauliflower or in the head of broccoli. Likewise, thresholds for pests that cause indirect damage is very low if the pest can disseminate plant pathogens. For example, low populations of aphids do not cause significant loss of yield in potato, but the potential for spread of aphid-vectored viruses is so high that control measures must be considered whenever aphids appear, particularly in seed-potato crops.

Crop losses caused by competition from weeds can be assessed quite readily, but weeds also contribute to overall crop losses by acting as alternative hosts for pathogens and insects. For example, wild cucumber *(Echinocystis lobata* (Michx.) Torr. & Gray) harbors the fungus *Didymella bryoniae,* which causes gummy stem blight in melon and cucumber (see Greenhouse cucumber, 22.11). The universal pathogens *Botrytis cinerea, Sclerotinia sclerotiorum* and *S. minor* are found on many weed species, *B. cinerea* in particular having hundreds of hosts.

Weeds also may act as a reservoir for many vegetable viruses and mycoplasma-like organisms, and of their insect and nematode vectors. The passage of workers and machinery through weed-infested crops can transmit viruses from weeds to crop plants; weed canopies provide the humid and cool microclimate in which fungi and bacteria infect their vegetable hosts; and

finally, weeds provide shelter for pest insects and other types of animals, such as rabbits and rodents. Weed control, therefore, is an important part of a pest management program for vegetable crops.

(Original by R.P. Jaques and W.R. Jarvis)

2.3 Pathogens and other pests *Figs. 2.3a-q; 2.3T1*

Identification

Correctly identifying both the host plant and the causal agent of a disease or pest damage will enable a vegetable grower to choose effective management practices that will prevent further damage to crop plants without affecting harmless or beneficial organisms. In the crop chapters of this book, the scientific or Latin names of pathogenic microorganisms and pests follow their common names; the italicized scientific name usually is followed by the name (often abbreviated) of the scientist(s) who described and named the organism. Using the scientific name for organisms avoids confusion over differences in language and in the selection of common names. For example, in some regions of Canada, the rutabaga is known as a swede or even as a winter turnip, but it is universally recognized by its scientific name *Brassica napus* var. *napobrassica* (L.) Reichb. The recommended common and scientific names of the major and minor vegetable crops grown in Canada are listed in Table 1.3. Classifying and naming plants and animals, including insects and microorganisms, follows a system of binomial nomenclature that is based chiefly on characteristics of vegetative and reproductive structures. Within species, populations also may be described at the functional or molecular level. Characteristics of the chief groups of organisms causing injury and disease in plants are described briefly in this chapter. More detailed descriptions of causal organisms are included in the discussion of specific disease and pest problems in the crops chapters.

(Original by W.L Seaman)

Bacteria and actinomycetes

Bacteria are tiny, one-celled microorganisms (prokaryotes) that, like fungi, require an external food supply for their energy. They, too, are facultative parasites of plants and are capable also of independent existence in plant residues, water or soil. Bacteria differ in certain fundamental ways from fungi in their cell structure, but they have very few morphological features that distinguish them from one another. Thus, a diagnostician has to rely on laboratory tests to identify them. Bacteria gain entry into plants through the stomata or through wounds caused by abrasion, insects or pruning. Bacterial diseases are highly infectious and are particularly difficult to control. Bacteria are spread easily by splashing water, particularly wind-blown rain and overhead irrigation. Some bacteria are carried from plant to plant by insect vectors, and they are all spread by hands, machinery and tools. Many also are carried on or in seed. Some pathogenic bacteria are capable of infecting one or a few host species or cultivars, whereas others, such as *Erwinia carotovora* subsp. *carotovora*, a soft-rotting bacterium, have a very wide host range.

Actinomycetes are classified with bacteria because nuclear fusion does not occur and they have cell wall biochemical characteristics more closely resembling those of bacteria than of fungi. They do resemble fungi in their filamentous morphology, but differ notably in the small diameter (usually about 1 µm) of their vegetative filaments. The most important actinomycete pathogen of vegetables is *Streptomyces scabies*, the cause of scab on such crops as potato, radish, carrot, rutabaga, parsnip and beet.

(Original by W.R. Jarvis and R.J. Howard)

Fungi

Fungi are microscopic plants with a basic, threadlike structure collectively called the mycelium. They have no chlorophyll and thus are unable to utilize carbon dioxide from the air for their nutrition. Instead, they utilize previously formed carbon compounds as a source of energy. They obtain these materials while growing saprophytically on the products or remains of plants and animals, or by parasitizing living plants and animals. In living, green plants, fungi usually degrade the host, producing visible damage, which, in vegetable crops, causes losses in yield and quality. As saprophytes, fungi are responsible for much of the natural breakdown of organic material and hence the recycling of essential elements and compounds in the environment. Mushrooms and toadstools are larger fungi that can be saprophytic, parasitic or, in many cases, symbiotic with green plants (mycorrhiza), living in plant roots to the mutual benefit of both fungus and host.

Parasitic fungi fall into two broad groups: obligate parasites, which depend entirely on a living host for their nutrition and reproduction, and facultative parasites, which can do considerable damage to crop plants as parasites, but can also live indefinitely as saprophytes on plant remains. Obligate plant parasites include the rusts, powdery mildews and downy mildews, whose names broadly describe the symptoms of the diseases they cause. The ubiquitous gray mold fungus *Botrytis cinerea* is a facultative parasite. Virtually all fungi that cause plant diseases form microscopic spores that serve two basic functions: to act as dispersal and infective propagules to spread the disease, and to act as resistant structures permitting the pathogen to survive adverse environmental conditions. In addition, many fungi also form compact, hard structures called sclerotia. These, like spores, are capable of resuming growth under favorable conditions to infect the host plant, sometimes after months or years.

Spores are dispersed in various ways, for example by air, in water through the soil or irrigation systems, by insects, or on hands, clothing and tools. Spores are the principal agents of plant infection. They germinate under suitable conditions, almost invariably in a water droplet or film or on a moist wound, to form a thread-like germ tube that can penetrate through the plant epidermis directly or through a stomatal pore. Once inside the plant tissue, the mycelium permeates the host tissues, sometimes

blocking the waterconducting system, as in the wilt diseases. As the food supply for the fungus diminishes, more spores are formed to spread the pathogen through the crop. By this time the host is either severely damaged or dead.

Spores can be produced by a sexual process, which imparts genetic variability to the fungus and can give rise to pesticide resistance or overcome host resistance, or they can be produced in huge numbers by an asexual, vegetative process. Some fungi form two or more types of spores that often do not much resemble each other in the same fungus. The sexual state is called the teleomorph and gives the fungus its proper, scientific (Latin) name, while asexual states are called anamorphs and frequently have a different Latin name. For example, the gray mold fungus *Botryotinia fuckeliana* is the teleomorph name for a rare, tiny, toadstoollike fungus. However, it is better known as *Botrytis cinerea*, the name that describes its asexual, dispersive and infective spores (conidia), which are arranged in a grapelike cluster. *Botrytis cinerea* is derived from the Greek, meaning an ashy-colored bunch of grapes. The fungus also has anamorphic microconidia, which are not infectious but have a sexual function, and chlamydospores. The latter are durable, long-lived spores in nature.

(Original by W.R. Jarvis)

Viruses and viroids

Viruses are submicroscopic particles consisting of a nucleic acid, either ribose nucleic acid (RNA) or deoxyribose nucleic acid (DNA). They multiply by inducing host cells to form more virus particles at the expense of host metabolism. The nucleic acid can be single- or double-stranded. Virus particles are rod-like, straight or flexuous, bacillus-like (rhabdoviruses), or isometric (polyhedral). Some small viruses are dependent on another virus for multiplication; these are called satellite viruses and require a helper virus for infection. Gemini viruses are paired, isometric particles with single-stranded RNA; an example is maize streak virus. Viroids are small units of single-stranded RNA arranged in a circle, devoid of protein, yet still capable of causing plant diseases; potato spindle tuber is a notable example.

The criteria for identifying and classifying a virus depends on certain physical, chemical and biological properties, including whether the nucleic acid is DNA or RNA, whether it is single- or double-stranded, and whether it has a membrane around the protein coat. In practical terms, indicator plants, often tobacco or *Chenopodium* species, are inoculated with sap from a diseased plant, and they produce symptoms characteristic of a particular virus. Since viruses have a protein coat, specific antibodies can be induced in animal serum, which can be made to react chemically and specifically in various diagnostic tests, such as precipitin or enzyme-linked immunosorbent assay (ELISA) tests.

Most viruses can be transmitted from plant to plant by infected sap introduced by injury, on hands, machinery or clothing, or by grafting. Many viruses are transmitted by insects, especially aphids; others are transmitted by mites, nematodes, fungi, or the parasitic plant dodder (*Cuscuta* spp.). Some viruses are seed- and pollen-borne.

(Original by W.R. Jarvis)

Virus-like pathogens (wall-less prokaryotes)

Lying somewhere between viruses and bacteria in characteristics are a group of microorganisms known as wall-less prokaryotes; in the crops sections of this book they are referred to as virus-like pathogens. They have genetic material but no nucleus or cytoplasmic organelles, in contrast to the more complex eukaryotes that include the fungi. Bacteria are also prokaryotes, but they have a cell wall. Wall-less prokaryotes have been linked with some 200 plant diseases. There are three main groups that cause plant diseases, 1) mycoplasmalike organisms (MLOs) of indefinite form that are more or less restricted to sieve tubes of plant vascular systems; 2) spiroplasma-like organisms, which are helical in form and restricted to sieve tubes; and 3) rickettsia-like organisms that resemble in form the typhus-causing *Rickettsia*, which has a rippled, trilaminate outer membrane. MLOs cause yellows diseases, (e.g. aster yellows) of lettuce, celery, potato, carrot and about 180 other plants, as well as leaf mottling, flower virescence, dwarfing and witches'-brooms. Typically, MLOs are transmitted from plant to plant by leafhoppers and can be controlled by the antibiotic tetracycline. Spiroplasmas cause such vegetable diseases as corn stunt.

(Original by W.R. Jarvis)

Nematodes

Plant parasitic nematodes or eelworms are small (usually less than 1 mm long), worm-like animals that live in soil. They are broadly divided into two groups; ectoparasitic nematodes that attack the plant externally, and endoparasitic nematodes that live, at least for part of their life cycle, inside the host tissues. All parasitic nematodes have mouth spears through which saliva is injected into the host tissues; it is the saliva that induces most of the damage in plants, for example tissue necrosis or the proliferation of giant cells, which can produce galls. Some nematodes, while causing little direct damage to plants, transmit viruses; such nematodes include species of *Xiphinema, Longidorus* and *Trichodorus.*

Worldwide, several hundred nematode species are plant parasites, most of which live in the soil. Many thousands of other species are free-living in the soil, feeding on fungi, bacteria and other microbes. Others are associated with animals, including man; some are naturally occurring biocontrol agents of insects. Most plant-parasitic nematodes feed on a relatively narrow spectrum of hosts, and only a few species are considered agricultural pests. Canada has relatively few nematodes that are of major economic importance in field and greenhouse vegetable crops (see Table 2.3a), mainly because of unfavorable climatic conditions.

Endoparasitic nematodes — These nematodes usually penetrate the roots, and feed and multiply within root tissues; some also invade bulbs, leaves and stems. They include the northern root-knot nematode *Meloidogyne hapla* Chitwood, which attacks almost all types of vegetable crops commonly grown in gardens, fields and greenhouses in Canada (see Carrot, 6.20). The southern root- knot nematodes *Meloidogyne incognita* (Kofoid & White) Chitwood, *M. javanica* (Treub) Chitwood, and *M. arenaria* (Neal) Chitwood do not occur in the field in Canada, but they can persist in greenhouses when imported from warmer climates. The pale cyst nematode *Globodera pallida* (Stone) Behrens and the golden nematode *G. rostochiensis* (Wollenweb.) Behrens have been introduced into Canada (see Introduced diseases and pests, 3.11). Both species occur in Newfoundland, and the golden nematode also occurs on Vancouver Island (see Potato, 16.36). The root-lesion nematode *Pratylenchus penetrans* (Cobb) Filip. & Stek. affects most of the major vegetable crops grown in Canada (see Potato, 16.38). The stem and bulb nematode *Ditylenchus dipsaci* (Kühn) Filip, attacks mainly onion and allied crops. It has been confirmed from Newfoundland, Ontario, Saskatchewan and British Columbia (see Onion, 13.24). The sugarbeet cyst nematode *Heterodera schachtii* Schmidt occurs at scattered locations across Canada. It can affect beet, spinach, rhubarb and cruciferous crops (see Beet, 5.14).

Table 2.3a. Host ranges of economically important nematode pests on vegetable crops in Canada

Crop	RKN*	RLN	PCN	SBN	SRN	SCN
Bean	X	X			X	
Beet, chard and spinach	X	X				X
Carrot	X	X				
Celery and celeriac	X	X				
Crucifers	X	X			X	X
Cucurbits	X	X				
Ginseng	X	X**				
Greenhouse cucumber	X	X				
Greenhouse lettuce	X	X				
Greenhouse pepper	X	X				
Greenhouse tomato	X	X				
Lettuce, chicory and endive	X	X				
Maize (sweet corn)		X			X	
Onion and other allium crops	X	X		X		
Parsnip	X					
Pea	X	X		X		
Potato	X	X	X		X	
Rhubarb	X	X				X
Tomato, eggplant and pepper	X	X	X		X	

*RKN = Root-knot nematode *(Meloidogyne hapla* Chitwood); RLN = Root-lesion nematode (*Pratylenchus penetrans* (Cobb) Filip. & Stek.); PCN = Potato cyst nematodes (*Globodera* spp.); SBN = Stem and bulb nematode (*Ditylenchus dipsaci* (Kühn) Filip.); SRN = Stubby-root nematodes (*Paratrichodorus* and *Trichodorus* spp.); SCN = Sugarbeet cyst nematode (*Heterodera schachtii* Schmidt).

**The root-lesion nematode is regarded as a potentially serious pest of ginseng in British Columbia.

Ectoparasitic nematodes — These nematodes feed on root tissues, such as the epidermis and cortex and, if their stylet is long enough, the vascular tissue. They rarely enter the roots of plants. They include the stubby-root nematodes *Paratrichodorus allii* (Jensen) Siddiqi, *P. pachydermus* (Seinhorst) Siddiqi, other *Paratrichodorus* spp., and *Trichodorus* spp. These nematodes have caused only minor damage to a few gardens in southern Alberta (see Potato). Other ectoparasitic nematodes include the dagger nematodes *Xiphinema* spp., the needle nematodes *Longidorus* spp., the pin nematodes *Paratylenchus* spp., the spiral nematodes *Rotylenchus* spp. and *Helicotylenchus* spp., and the stunt nematodes *Tylenchorhynchus* spp., *Merlinius* spp., *Amplimerlinius* spp., and *Gracilacus* spp. These nematodes are prevalent in some Canadian vegetable fields and often are identified from soil samples, but they are rarely a serious problem. At numbers as high as 5000 or more per kilogram of soil, pin nematodes have reduced yields of rhubarb in Ontario. Dagger and needle nematodes prefer hosts with woody roots and are more frequently associated with strawberry, raspberry, grapes and roses than with vegetable crops, which tend to be more soft-rooted.

Damage caused by plant-parasitic nematodes is often difficult to distinguish from that caused by other pathogens or by abiotic factors. Stunting, chlorosis and early senescence also can indicate a problem with soil nutrition, watering, or a soil-borne pathogen; these conditions need not necessarily be nematode-related. Proliferation of secondary roots, a symptom of attack by some nematodes, also may result from the branching of the tips of young roots of some vegetables in the presence of such unfavorable soil conditions as soil compaction, insufficient decomposition of organic plant residues, extremes in moisture content, poor fertility, and frost heaving. Some nematode problems can be assessed by visual examination of plant tissue. In many cases, however, nematode problems can only be determined after soil sampling and extraction; both procedures are time-consuming and expensive. Nematodes do not spread very rapidly, and a minor infestation may not result in visible symptoms or reduced productivity.

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(Original by T.C. Vrain and B.A. Ebsary)

Insects

Many species of insects, mites, spiders, millipedes, centipedes and like animals, collectively known as arthropods, are present in the plant ecosystem. Only a relatively small proportion of insect species feed or have a detrimental effect on vegetable plants, and only a few of those that feed on vegetable plants are economically important pests; a large proportion occur in small numbers, feed very sporadically, or cause only minor, indirect damage. Nevertheless, the relatively few insect species that are economically important pests, often only one, two or three on a plant species, can destroy a crop or cause sufficient damage to render it unmarketable or unprofitable to grow unless the pest populations are regulated. A list of the major groups of insects associated with vegetable crops in Canada is given in Table 2.3b.

Insects are a diverse group of six-legged invertebrates that undergo complete, gradual or no change of form (metamorphosis) during development. Insects and their close relatives, spiders and mites, are animals that have jointed legs. Adult insects have three body regions (head, thorax and abdomen), three pairs of legs, one pair of antennae, complex mouthparts, and frequently two pairs of wings. The skin of an insect is the external skeleton, which covers the whole body. This exoskeleton must be shed from time to time (molting) as the insect grows.

Life cycle — All insects have an egg and an adult stage. A few, such as springtails, do not undergo metamorphosis; the juveniles resemble adults but are smaller. Juveniles that resemble the adult stage except that they lack wings or have underdeveloped wings are called nymphs; insects that have egg, nymph and adult stages are said to undergo gradual metamorphosis; examples include earwigs, aphids, plant bugs and whiteflies. Juvenile forms that do not resemble the adult stage are called larvae; in some larvae, the thoracic legs are underdeveloped, while others have legs in the abdominal region, and some have no legs at all. Certain types of larvae have distinctive names; larvae of moths and butterflies are called caterpillars, while caterpillars in which some of the abdominal legs are missing are termed loopers; larvae of beetles are known as grubs, and the legless larvae of flies are called maggots. Insects that have egg, larval, pupal and adult stages are said to undergo complete metamorphosis. The juvenile forms (larva or nymph) grow in steps called instars; at the end of each instar they molt, then swell to the new size; the new outer skin hardens and remains unchanged until the next molt. The number of molts varies with the species. The last instar typically spins a cocoon or forms a puparium from which the insect emerges in its adult form. Insects may overwinter in the egg, larval, pupal or adult stage. Juvenile stages usually do the most serious damage to plants, but many adult insects also can inflict damage. It is important in devising appropriate pest management strategies to be able to recognize the different stages of insect development.

The most common, foliage-eating insect pests are larvae of moths and butterflies (Lepidoptera) and larvae and adults of beetles (Coleoptera); nymphs and adults of grasshoppers and other related species (Orthoptera) also may consume foliage. Aphids and leafhoppers (Homoptera), plant bugs (Heteroptera) and thrips (Thysanoptera) have piercing or piercing-and-sucking mouthparts in the nymphal and adult stages and may cause extensive damage to vegetable crops; many of these insects have a major role in transmission of pathogens, especially viruses. Larvae of flies (Diptera) feed and burrow into roots, bulbs and stems of plants and are important pests of root crops, such as carrot, onion and rutabaga, and of several other crops, including cabbage and cauliflower, on which they feed on the roots and stems, and bean and corn, where they feed on the germinating seeds and seedlings. A great many insects also are beneficial, feeding on other pest species of insects as predators or as parasites (see Beneficial insects, mites and pathogens, 3.7). A key to the principal orders of insects associated with vegetable crops in Canada is given in Table 2.3c.

(Original by R.P. Jaques and J.A. Garland)

Mites and spiders

Adult mites and spiders characteristically have four pairs of jointed legs; exceptions include the rust mites and the blister mites, which have only two pairs of legs. Unlike insects, they lack antennae and have only two body regions, the cephalothorax and abdomen. Spiders are predators, chiefly of insects. The spider-like daddy longlegs, or harvestman, which is related to mites and spiders, is commonly seen around home gardens. It also is a beneficial predator of small organisms, including insects.

Table 2.3b. Characteristics of major groups of insects associated with vegetable crops in Canada

Order	Common name	Mouthparts	Metamorphosis
Collembola	springtails	chewing	none
Orthoptera	grasshoppers, crickets	chewing	gradual
Dermaptera	earwigs	chewing	gradual
Thysanoptera	thrips	piercing	gradual
Homoptera	aphids, leafhoppers	piercing-sucking	gradual
Heteroptera	stink bugs, plant bugs	piercing-sucking	gradual
Coleoptera	beetles, weevils	chewing	complete
Lepidoptera	butterflies, moths	chewing (larvae)	complete
Hymenoptera	ants, bees, wasps	chewing	complete
Diptera	flies	rasping (larvae)	complete

Table 2.3c. Key to the principal orders of insects associated with vegetable crops in Canada

This key should be applicable to adults and most nymphs and larvae of the groups indicated.

1. Winged
   Wings entirely or partly membranous with veins 2
1a. Winged or wingless
   If winged, forewing (FW) thickened throughout 7
2. Wings with scales
   Mouthparts coiled ..... butterflies and moths
2a. Wings lacking scales;
   Mouthparts not coiled 3
3. FW only, hindwing (HW) greatly reduced ..... flies
3a. FW and HW same size or HW only slightly smaller 4
4. Wings narrow, fringed around the margin
   Body less than 5 mm in length
   Legs (tarsi) without claws ..... thrips
4a. Wings broad, without fringe around margin
   Body more than 5 mm in length
   Legs (tarsi) with a pair of claws 5
5. Tarsi with 5 sub-segments (tarsomeres)
   Mouthparts for chewing
   HW with tiny hooks (hamuli) along leading edge ..... winged ants, bees and wasps
5a. Tarsi with only 2 or 3 tarsomeres
   Mouthparts for sucking
   HW without hamuli along leading edge 6
6. Mouthparts arising at front of head
   Immatures with wing buds
   Adult FW membranous over outer half, and thickened over inner half ..... plant bugs, stink bugs
6a. Mouthparts arising beneath head
   Immatures with wing buds (as above)
   Adult FW membranous or thickened throughout ..... aphids, leafhoppers
   Adult FW membranous with a waxy coating ..... whiteflies
7. FW hard or leathery, covering HW 8
7a. Entirely wingless 11
8. Abdomen with forceps-like appendages (cerci) ..... earwigs
8a. Abdomen lacking cerci or cerci not forceps-like 9
9. Mouthparts adapted for piercing-sucking ..... true bugs (go back to 6)
9a. Mouthparts adapted for chewing 10
10. FW thickened or leathery with veins
   Wings held roof-like over abdomen or overlapping with HW folded lengthwise fan-like
   Cerci present, straight
   Antennae short ..... grasshoppers
   Antennae long, filamentous ..... crickets and katydids
10a. FW hardened (elytron) and without obvious venation
   Wings held flat over abdomen
   FW's meeting in a straight line
   HW folded crosswise, not fan-like
   Cerci absent ..... beetles

11. Body narrow-waisted
Antennae elbowed ..... wingless ants and wasps
11a. Body broad-waisted
Antennae not elbowed 12
11b Body fleshy and larva-like, lacking division into thorax and abdomen by a waist
Antennae inconspicuous or absent 16
12. Abdomen with a spring (furcula) ..... springtails
12a. Abdomen lacking furcula 13
13. Abdomen with a pair of dorsal projections (cornicles)
Body plump, not narrow
Adults and nymphs may be present ..... wingless aphids (go back to 6)
13a. Abdomen lacking cornicles
Body narrow, not plump and less than 5 mm in length 14
14. Tarsi lacking claws ..... thrips (go back to 4)
14a. Tarsi with claws 15
15. Antennae 4- or 5-segmented
Mouthparts for piercing-sucking (from front of head)
Cerci absent ..... plant bug, young nymph (go back to 6)
15a. Antennae with many more than 5 segments (filamentous)
Mouthparts for chewing
Cerci present ..... cricket, young nymph (go back to 10)
16. Body with a capsule-like head and legs
Head lacking compound eyes
Mouthparts for chewing, moveably attached to head 17
16a. Body lacking a head capsule and legs
Mouthparts in form of mouthhooks, retractable ..... fly larvae (maggots)
17. Head with 1-6 simple eyes (ocelli) on each side
Body with segmented legs (3 pairs) anteriorly
Body with fleshy legs (2-5 pairs) posteriorly ..... butterfly and moth larvae (caterpillars)
Note: sawfly larvae, which have 1 simple eye on each side of the head and may be mistaken for caterpillars, do not have representatives on vegetable crops in Canada.
17a. Head lacking simple eyes
Body with segmented legs (3 pairs) anteriorly
Body lacking fleshy legs
posteriorly ..... beetle larvae (wireworms, white grubs and other beetle grubs)

(Original by J.A. Garland)

Female mites deposit eggs, which hatch into six-legged larvae. The larvae feed and molt to form eight-legged nymphs, of which there are several forms, before the adult stage is reached. In some species the females give birth to live young. Because of their small size, mites often are not noticed. Many are scavengers and some are predators. Many species have rasping and sucking mouthparts and may damage vegetable crops, not only by weakening the plant by sucking out sap, but also by destroying cells and by aiding entry and transmission of pathogenic microorganisms. The two-spotted spider mite is the most important species on vegetable crops in Canada, both in the field and in greenhouses. Some mites are predatory and are being used to control pest mites and thrips in greenhouses (see Beneficial insects, mites and pathogens, 3.7).

(Original by J.A. Garland and W.L Seaman)

Centipedes and millipedes

These animals, sometimes confused with insects, are encountered in the garden or compost. Centipedes are somewhat flattened, have 15 or more pairs of legs, with one pair per body segment, and their antennae (one pair) are long and have 14 or more sub-divisions. Millipedes (*12.21T1*) are cylindrical, have two pairs of legs on each segment except the first several segments following the head, which have only a single pair, and their antennae (one pair) are short, usually with seven sub-divisions. Centipedes are poisonous predators of insects and are usually faster moving than millipedes, which feed primarily on dead plant remains and tend to coil when disturbed.

(Original by J.A. Garland)

Symphylans

Symphylans are small, flattened, white animals that resemble centipedes. They are less than 8 mm long. The adults have 10 to 12 pairs of legs (centipedes have 15 or more pairs), a pair of unbranched antennae, and a pair of hair-like appendages on the last segment. They feed chiefly on microorganisms and plant material in the soil, but they also may feed on the roots of plants, especially in moist soils that are high in organic matter. Damage to root hairs and roots may facilitate the entry of plant pathogens.

(Original by J.A. Garland and W.L. Seaman)

Slugs and snails

All land snails and most slugs have a calcareous shell. In snails the shell is external and twisted in a continually increasing spiral, usually clockwise. The shell takes many forms among different species. The shell aperture (“mouth”) in all land snails in Canada lacks a cover (operculum). In some snail groups, identification is based on characteristics of soft anatomy. In slugs, the shell is

internal, and characteristics of the fleshy body are used in identification; these include the position of the breathing pore, the presence of a groove in the mantle, the ridge (keel) on the back, the caudal mucal pore, the color and pigmentation of the body, and the color of the mucus.

Both slugs and snails may be plant pests in damp situations. They both glide on a slime trail of secreted mucus. Mouthparts are rasping, and most damage is done at night or on cloudy days. Under dry conditions, eggs of slugs may remain unhatched for long periods. Young snails remain close to the area in which they were hatched for several months and may take many months to mature. The brown garden snail, which was introduced into British Columbia, is edible but is regarded as a pest in Canada (see Introduced diseases and pests, 3.11).

(Original by J.A. Garland and W.L. Seaman)

Sowbugs and pillbugs

These are crustaceans adapted to living out of water but in damp environments. They are oval, with a small head, two pairs of antennae, seven pairs of jointed, similar (isopod) legs and a dorsoventrally flattened body that is composed of hard, overlapping plates. They can be distinguished by the ability of the pillbug to curl into a ball, and by the two tail-like appendages on the sowbug. Both are active decomposers of rotting vegetation. They may cause problems in certain situations where vegetables, such as cucurbits, rest on moist ground or where young seedlings are slow to develop in cool, moist weather. These crustaceans have not been implicated in transmission of diseases in vegetable crops.

(Original by J.A. Garland and W.L. Seaman)

Weeds *(Figs. 2.3a-q)*

Weeds generally are considered to be plants growing where they are not desired. They sometimes can be as serious a threat to vegetable crops as diseases and other pests, and they occur wherever vegetable crops are grown in Canada. Successful vegetable production often depends upon the integration of weed management (see 3.13) with other pest management strategies.

Vegetable crops vary widely in their response to weed competition, ranging from non-competitive crops, such as onion, to moderately competitive crops, such as potato and transplanted cabbage. Studies in Canada have shown that direct-seeded onion does not produce marketable bulbs if weeds are not controlled. In southern Alberta, the time from seeding to the two-leaf stage in onion averages 46 days; during that time, wild mustard can emerge, complete its vegetative growth, and flower.

The critical period of weed competition in vegetable crops is the minimum time that weeds must be suppressed to prevent yield losses. In a southern Ontario study, cucumber yields were reduced if the crop was not kept weed-free for up to four weeks after seeding, or if it was weedy for more than three to four weeks. In another trial, the critical period for pickling cucumber ranged from two to five weeks after seeding. In Prince Edward Island, the critical period for rutabaga is two to four weeks after crop emergence when barnyard grass *(2.3a)* and lamb's-quarters *(2.3fq)* are present. Similarly in Quebec, the critical period for carrot crops grown on organic soils was found to be between three and six weeks after crop emergence. Although the concept of a critical weed period has practical limitations because crops and weeds grow at different rates from year to year, early removal of weeds is clearly important in reducing losses from competition. In addition to their direct competition in crop growth, weeds are important reservoirs of most crop viruses and their insect and nematode vectors, and of pathogenic fungi and bacteria. Because of their density and proximity to crop plants, weeds also provide microclimates conducive to infection by fungi and bacteria.

Chemical weed control treatments have been developed mainly for field crops in which the soil becomes shaded early in their development. Many vegetable crops, on the other hand, do not provide rapid shading of the soil around them and weeds continue to germinate over a longer period of time. The continued emergence of weeds necessitates additional tillage, which disturbs the soil and stimulates more weed seeds to germinate, perpetuating the problem.

The weeds most commonly encountered in vegetable fields in Canada are listed in Table 2.3d. Weed problems in vegetable crops are often regional. In the Prairie provinces, for instance, vegetables occasionally follow cereals and, hence, volunteer barley or wheat can be a problem, especially after a dry autumn. In eastern Canada, cereals are sometimes used as cover crops or form part of a stale-seedbed treatment for weed control. Other examples of regional weed problems include: eastern black nightshade, hairy galinsoga, velvetleaf *(Abutilon theophrasti* Medik.) and large crabgrass *(Digitaria sanguinalis* (L.) Scop.) in southern Ontario; kochia *(Kochia scoparia* L.) *(2.3e)* in southern Alberta and hemp nettle *(Galeopsis tetrahit* L.) in central Alberta; barnyard grass *(2.3a)* in New Brunswick; common groundsel *(Senecio vulgaris* L.) *(2.3d)* in Newfoundland; and creeping yellow cress *(Rorippa sylvestris* (L.) Besser) in the Fraser Valley of British Columbia.

Table 2.3d. Weeds commonly occurring in vegetable crops in Canada

Common name	Scientific name
Annual grasses	
Barley, volunteer	*Hordeum vulgare* L.
Barnyard grass	*Echinochloa crusgalli* (L.) Beauv.
Crabgrass	*Digitaria* spp.
Groundsel, common	*Senecio vulgaris* L.
Foxtail, green	*Setaria viridis* (L.) Beauv.

Foxtail, yellow	*Setaria glauca* (L.) Beauv.
Wheat, volunteer	*Triticum aestivum* L.
Broadleaved annual weeds	
Buckwheat, wild	*Polygonum convolvulus* L.
Chickweed	*Stellaria media* (L.) Cyrill
Cudweed, low	*Gnaphalium uliginosum* L.
Flower-of-an-hour	*Hibiscus trionum* L.
Galinsoga, hairy	*Galinsoga ciliata* (Raf.) Blake
Lamb's-quarters	*Chenopodium album* L.
Mallow, round-leaved	*Malva rotundifolia* L.
Mustard, wormseed	*Erysimum cheiranthoides* L.
Mustard, wild	*Brassica kaber* (DC.) Wheeler
Nightshade, black	*Solanum nigrum* L.
Nightshade, eastern black	*Solanum ptycanthum* Dun.
Nightshade, hairy	*Solanum sarrachoides* Sendt.
Pigweed, redroot	*Amaranthus retroflexus* L.
Pigweed, prostrate	*Amaranthus graecizans* L.
Purslane, common	*Portulaca oleracea* L.
Radish, wild	*Raphanus raphanistrum* L.
Ragweed, common	*Ambrosia artemisiifolia* L.
Shepherd's-purse	*Capsella bursa-pastoris* (L.) Medicus
Smartweeds, annual	*Polygonum* spp.
Spurry, corn	*Spergula arvensis* L.
Perennial weeds	
Bindweed, field	*Convolvulus arvensis* L.
Milkweed, common	*Asclepias syriaca* L.
Mint, field	*Mentha arvensis* L.
Quack grass	*Agropyron repens* (L.) Beauv.
Thistle, Canada	*Cirsium arvense* L.
Thistle, sow	*Sonchus arvensis* L.

2.3T1 Parasitic higher plants; yellow strands of dodder, *Cuscuta* sp., entwining a sugar beet plant.

Crop injury from herbicides — See Management of weed pests, 3.13.

(Original by R. Esau)

Parasitic higher plants

More than 2500 species of higher plants are known to live parasitically on other plants. These parasitic plants belong to several different botanical families and vary greatly in their dependence on their host plants. Relatively few of the known higher parasitic plants cause diseases on agricultural crops. In Canada, only dodder *(Cuscuta* sp.) has been observed as a pest in field-grown crops, and only very rarely has it been found in vegetables. In the United States, dodder has been reported to occasionally cause economic losses in carrot, onion, tomato, sugar beet, potato, hops, peppermint and pepper. Dodder forms dense tangles of leafless strands on and through the crowns of host plants (*2.3T1*). It reduces the growth and yield of affected plants.

(Original by R.J. Howard)

▶ 2.4 Climate and environment

Pest distribution

The plant pathogens, insects, mites and other pests that attack and damage vegetable crops in Canada often are the same as those found on the same crops grown in similar climates in other countries. However, the climatic tolerances and other characteristics of pathogens may be altered as they adapt to climate, soil and other factors in Canadian production areas. Similarly, insect and other pests are influenced by climate, often resulting in pest complexes that are substantially different from those in other production areas. Indeed, because Canadian production areas are near the northern limit of distribution of some pest species, their population fluctuations and numbers differ from those nearer the main area of distribution, partly because parasitic and predaceous species, themselves, may not be as functional in Canada. It is apparent, therefore, that some species of pathogens and pests that are common elsewhere may be of minor importance on the crops grown in Canada; they may be secondary or occasional, or they may not be noticed at all. Others, however, may be devastating.

(Original by R.P. Jaques and W.R. Jarvis)

Environment-related disorders

All crop plants have an optimum total environment for productivity; any environmental factors departing markedly from that optimum will decrease yield and, by causing stress to the plants, may make them more susceptible to diseases and pests. Heavy yields also may stress plants by diverting photosynthetic products and other nutrients to the harvested organs (fruit, tubers, roots, leaves) at the expense of other parts of the plant. Many vegetables are raised from transplants, often started in greenhouses or distant geographical areas, and sometimes they are poorly acclimatized, so that they, too, are severely stressed at transplanting, being more susceptible, for example, to late spring frosts or heat damage, particularly on sandy soils.

Other environmental factors that can have minor or disastrous effects on crops include; too much or too little water, poor soil structure and compaction from machinery, poor drainage, heat, cold (including frost), wind, hail, lightning, and industrial pollutants in air, soil and water.

(Original by W.R. Jarvis)

Chemical injury

Experienced extension personnel know that a high proportion of crop damage can be attributed to chemical injury; for example, too much or the wrong fertilizer, or too much or the wrong pesticide. Often, these situations result from simple, arithmetical errors in calculating rates of application, or by applying pesticides on the assumption that twice as much might be twice as good. Indeed, all materials with a '-cide' suffix can damage nontarget organisms and, even at the recommended rates, may reduce yield to an extent that is scarcely compensated for by the control of weeds, insects or pathogens. Not infrequently, herbicide damage occurs simply because various pesticides have been applied from the same sprayer without thoroughly cleaning it after each use.

All fertilizers and pesticides should be applied strictly in accordance with the manufacturer's directions and local expert advice, and with appropriate care to avoid exposure of non-target organisms, including workers and the public. To avoid environmental contamination, precautions about spraying in windy conditions, disposing of pesticide containers, and avoiding contamination of ground water and reservoirs should be observed.

(Original by W.R. Jarvis)

Nutritional disorders

In addition to the gross effects of nitrogen-phosphate-potassium (NPK) fertilizers, there are many disorders of vegetable crops that are caused by excesses or deficiencies of the so-called minor or trace elements. The availability of the essential elements to plants depends largely on soil type and environmental conditions. For example, phosphorus is less available to plants in heavy than in light soils; magnesium becomes deficient in sandy soils leached by heavy rains and irrigation; boron is less available in limed and dry soils; and iron and manganese both become more available in acidic soils. Indeed, manganese toxicity can occur in very acidic soils. Various elements can affect the availability of others; for example, iron availability is depressed by an excess of phosphate. Some of these effects are magnified in greenhouse soils, which are heavily amended with organic materials and may be steam-sterilized. Steaming releases toxic amounts of manganese and ammonia, and steamed soils generally have to be leached before use for those reasons. It is strongly recommended that vegetable soils be sampled regularly for chemical analysis in order to determine exact fertilizer requirements. It is particularly important to provide the proper amounts of calcium and potassium because they enhance the natural resistance of plants to some diseases.

(Original by W.R. Jarvis)

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3 Disease and pest management

Figures *3.7* to *3.13; 3.2T1; 3.2T2; 3.14T1*

▶ 3.1 Integrated pest management

For many years, Canadian vegetable producers relied heavily on protective applications of pesticides to reduce the impact of diseases and pests on their crops. In some cases, excessive amounts of chemicals were applied with little or no regard to effects on non-target organisms or contamination of the produce, soil and water. In the late 1950s, however, scientists, particularly those

dealing with control of arthropod pests, began to develop alternative procedures for crop protection, using a minimum of broad-spectrum chemical insecticides in combination with selective chemicals, biological agents, and modified cultural practices. This work stimulated interest in regulation of plant pathogens by biocontrol agents. In addition, by monitoring population levels of pests and pathogens in relation to crop damage, action thresholds were established for use in crop protection programs as a guide to the timing of control measures. Thus, the present holistic approach to disease and pest control, termed integrated pest management, is evolving and is being widely implemented. Predictive programs, such as BOTCAST for onion botrytis leaf blight, TOM-CAST for tomato early blight, and BLITE- CAST for potato late blight, are resulting in a more precise approach to disease and pest control and are within the spirit of sustainable agriculture. Similarly, action thresholds for application of chemical insecticides to regulate insect pests on such crops as carrot, celery, crucifers, onion, potato, sweet corn and tomato have been or are being developed.

The effective integration of cultural practices and other methods of disease control is illustrated by the development of a system for the control of pea root rot. This system takes into account four pathogenic fungi and their disease cycles, tillage practices, drainage, soil compaction, cultivar susceptibility, green manure crops, and the predisposition of pea to these fungal pathogens by herbicides. A more limited program for the control of potato late blight has been developed, and an integrated system for the control of cucumber powdery mildew in the greenhouse has been described, along with several ways of manipulating the greenhouse crop and its environment to escape diseases. Thus far, none of these systems considers the simultaneous control of insect and other pests, so the term "integrated" is only partially valid, but it is encouraging to note that many greenhouse tomato growers have not used chemical pesticides for several years.

These integrated programs can be improved by the development and use of selective pesticides to minimize the environmental impact of crop protection practices, particularly on indigenous and introduced agents for biological control. Undoubtedly, the most successful selective pesticides developed to date are formulations of the bacterium *Bacillus thuringiensis* Berliner. Applications of these formulations will control most foliage-feeding lepidopterous larvae, including those on cruciferous crops, lettuce and celery, and some coleopterous larvae, notably the Colorado potato beetle on potato and tomato. Such applications have no direct effects on non-target, parasitic or predaceous arthropods and do not contribute to pollution of the soil, water or crop. Furthermore, residues on the crop are not hazardous to humans and other animals. The potential for using viruses, protozoa, fungi and nematodes that kill insects and mites also is promising. Introduced parasitic or predaceous insects and mites are especially successful for control of pests of greenhouse-grown vegetables. Introductions of beneficial insects and mites, in combination with environmental manipulation and hydroponic systems to minimize the occurrence of diseases, have permitted pesticide-free production of greenhouse vegetables, particularly tomato and lettuce, by many growers. Unfortunately, introductions of beneficial insects and mites, and manipulation of those that occur naturally have been exploited very little for control of insect pests of vegetable crops under commercial field conditions.

Other methods to control pest insects and mites, such as introduction of attractant and repellant materials, application of growth-regulating chemicals, or mass release of pheromones to cause mating disruption, which have been tested or applied to other crop systems, have not appeared promising or have not been assessed for controlling pests on vegetable crops. Similarly, the release of sterilized males to reduce development of pest species has not proven economically practical for control of insects on vegetables.

In greenhouse vegetable crops, the high degree of environmental control available to growers enables fully integrated programs to be devised that give good productivity as well as control of insects, mites and diseases. Biological control, using natural and introduced parasites and predators of pathogens and pests, plays a major role in greenhouse crop protection, but it is more difficult to realize in field vegetable crops because of the lack of control over the environment.

Management of viral diseases depends largely on vector control, crop hygiene, resistant cultivars, and regulatory systems of quarantine, inspection and certification. Crop protection strategies for nematode and weed pests are discussed later in this chapter.

(Original by W.R. Jarvis and R.P. Jaques)

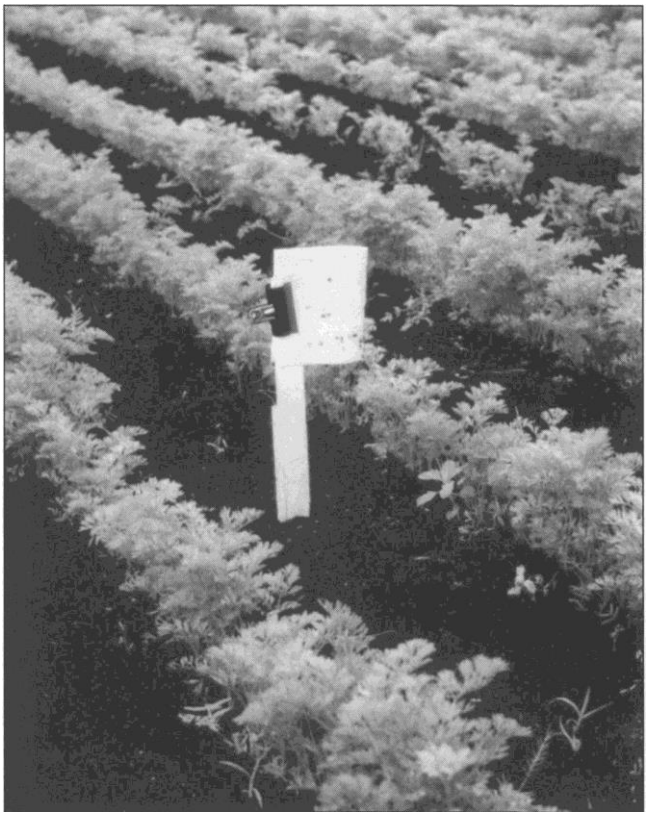
▶ 3.2 Monitoring *Figs. 3.2T1, 3.2T2*

Monitoring involves some form of surveillance to detect the presence of a disease or pest before economic damage has occurred and in time to economically apply eradicative or protective measures. Two such methods involve visual inspection and trapping.

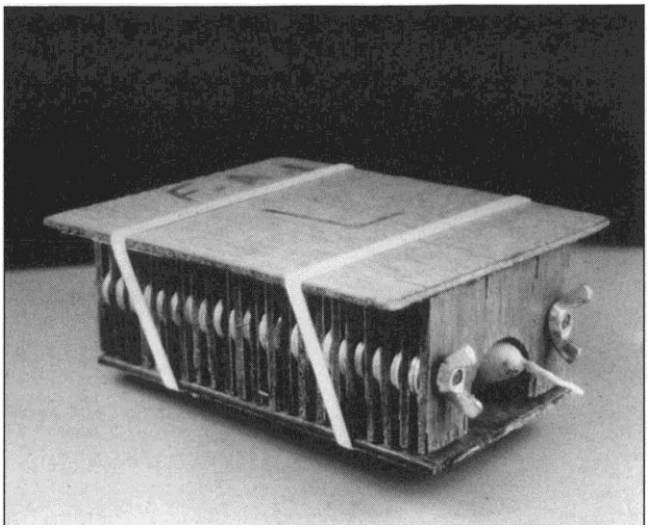
Visual inspection is the simplest method of monitoring and is used for insect and mite pests and for certain diseases, especially in combination with weather data and epidemiological information concerning sporulation, spore dispersal, infection periods and host plant growth stages. Surveillance is done on a regular basis, beginning soon after seeding or planting out, and includes watching for signs of feeding or irregularities of plant growth.

Trapping is necessary for monitoring certain pests; traps may be purchased for certain pests or some may be constructed at home. The type of trap and its location must be suitable for the target species. Sticky strips or glass slides smeared with petrolatum or other adhesive are used for trapping fungal spores, aphids and thrips; colored pans with water in the bottom are used for aphids; colored sticky strips or ribbon-like tapes are available commercially for whiteflies in greenhouses; a variety of lights and light traps are used for moths of many types; and a pheromone in a rubber septum is available for such insects as the European corn

borer. Determining the density in soil of nematode pests before planting is important for some crops. Threshold levels have been established for a number of vegetable crops with regard to the root-lesion and root-knot nematodes.



3.2T1 Monitoring; sticky traps, such as the yellow one shown here in a carrot field, are used to trap insects such as the carrot rust fly, aphids, leafhoppers and thrips; in the greenhouse, blue traps are more attractive than yellow ones to the western flower thrips and may be less attractive, and therefore less harmful, to beneficial insects.



3.2T2 Monitoring; carrot weevil adults may be monitored with traps made of wooden plates spaced 3 mm apart and containing a carrot as bait at the base.

3.3 Cultural practices

Escape and prevention

After genetic resistance, the most important strategy for managing plant diseases and pests is best thought of as escape or prevention rather than cure. Such measures are aimed at avoiding the conditions that predispose the crop to infection or infestation; for example, selection of healthy seed of high germinability from a reputable source; treating seed if necessary to eradicate pathogens; checking the health of transplants at their source; practicing adequate crop rotation; sowing and transplanting only when soil conditions (temperature, moisture, nutrients, tilth) are adequate; maintaining adequate spacing and row orientation parallel to prevailing winds to permit good crop ventilation; avoiding overhead irrigation when the crop is in a vulnerable stage (e.g. at flowering in bean and pea); avoiding working in fields when the foliage is wet with dew or rain; roguing infected plants from seed crops; picking fruit before it is overripe; avoiding mechanical damage and removing field heat as quickly as possible at harvest; controlling weeds that may harbor pathogens and arthropod pests and contribute to humid microclimates in the crop; and controlling insects that may cause infectible wounds and transmit viruses.

Crop hygiene is a major factor in disease escape because it removes many sources of pathogens. Trash piles, contaminated seed trays, pots and stakes, and infected weeds are important sources of wind- and water-borne fungi and bacteria. Personal hygiene and equipment sanitation are also very important. Bacteria and viruses are easily transmitted on hands, clothing, tools and machinery. Tomato mosaic virus, for example, can persist for several months or years on sap-drenched overalls if they are hung dry in a dark closet. In bean fields, bacterial diseases often follow the tracks of workers walking among wet plants.

In nature, populations of insects and mites are regulated primarily by climate-related factors; by predation and parasitism by other arthropods and by other animals, such as birds; by diseases; by preference for food source; by competition with other plant feeders; and by numerous other factors. Because of the significant mortality of insects and mites by predation, parasitism and diseases, the best control practice for many insects and mites is to do nothing that could reduce the impact of these naturally occurring beneficial organisms and other beneficial agents in the environment.

Cultural practices usually influence the impact of pests indirectly. Tillage, crop nutrition, crop rotation, timing of planting and other management practices affect the vigor of the crop plant. A vigorous, healthy plant is more likely to withstand damage from feeding by sap-sucking insects and mites. Attack by a pest sometimes can be circumvented or damage reduced by tillage, by timing of planting and by selection of field conditions. For example, planting cruciferous crops in well-drained soils and delaying planting to reduce exposure of young plants to cold, damp soil conditions will reduce damage by root maggots. The removal of crop residue from the field and other sanitation procedures may reduce infestations of some pests, such as the European corn borer, in the following crop.

(Original by W.R. Jarvis and R.P. Jaques)

3.4 Resistant cultivars

Pathogen resistance

The primary management technique for most plant diseases is to develop cultivars that possess genetic resistance or tolerance to infection; tolerance is the ability of the plant to be productive despite being infected.

Of course, not every cultivar is resistant to every disease, but the prevalence of a disease in a particular locality often will determine the choice of a cultivar. Even when using a resistant cultivar, producers should be vigilant in watching for disease. Microorganisms, particularly those with genetic diversity derived from sexual reproduction, which occurs in many fungi, can quickly overcome resistance with a new form or race. For example, the fungus *Bremia lactucae,* which causes downy mildew of lettuce, exists in a number of pathogenic races, so lettuce growers must be aware of the races that are prevalent in their area and choose a cultivar accordingly. Recently, a new race of the tomato wilt pathogen *Fusarium oxysporum* f. sp. *lycopersici* appeared in Florida, and it is perhaps just a matter of time before it shows up in Canada and elsewhere. It is known as race 3 and it overcomes resistance to the two previously known races 1 and 2. Plant breeders work continually to produce cultivars resistant to new races of pathogens.

Insect and mite resistance

The development and use of resistant or tolerant cultivars has been less successful for arthropod pests than for diseases. Some cultivars of vegetable crops are less attractive to pest insects and mites, and a few cultivars and hybrids are resistant to some insect and mite pests by virtue of chemical or physical characteristics. For example, there is a substantial difference in the frequency of tunneling by the European corn borer among cultivars of sweet com. On the other hand, no apparent difference in feeding of the imported cabbageworm is found among cultivars of cabbage.

Future possibilities

The development of pest-resistant cultivars of vegetable plants will be enhanced by the use of genetic manipulation techniques. These advanced biotechnological procedures permit more precise and faster addition and removal of specific factors or characters than do conventional plant breeding techniques.

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(Original by W.R. Jarvis and R.P. Jaques)

▶ 3.5 Biological control

Concepts and practices

Effective biological control of vegetable diseases is rarely achieved, even on an experimental scale. Unlike predatory and parasitic insects and mites, a disease-controlling organism is almost invariably a fungus or bacterium and has to be registered in the same manner as the microbial insecticide *Bacillus thuringiensis* Berliner or a chemical pesticide. The registration process entails tests to ensure that the biological pesticide is environmentally safe, not harmful to operators or crop handlers, and safe for humans and livestock to consume with food. These tests, combined with the need to demonstrate that the biological pesticide is at least as effective as the best alternatives, as well as being economical and non-phytotoxic, require several years of research and testing, which leads in the end to a very expensive product.

At present, Dygall (*Agrobacterium radiobacter* (Beij. & Van Delden) Conn) is the only biological pesticide available commercially in Canada for control of a bacterial disease, in this case crown gall in nursery stock. However, organisms to control white mold in bean and powdery mildew in greenhouse cucumber offer considerable commercial promise, and a number of patents of biological fungicides are pending. A yeast-like fungus is being developed commercially to control powdery mildew of cucumber in greenhouses, and fungi in the genera *Trichoderma* and *Gliocladium* show promise for control of root-infecting fungi. These agents actively parasitize the pathogen, produce enzymes that degrade the cell wall, or produce antibiotics. Examples are *Coniothyrium minitans* W.A. Campbell, which is a parasite of the white mold *(.Sclerotinia* spp.) and gray mold (*Botrytis* spp.) pathogens, and *Sporidesmium sclerotiovorum* Uecker *et al.*, which parasitizes sclerotia of *Sclerotium cepivorum* (see Onion, white rot, 13.12) and *S. minor* (see Lettuce, drop, 11.9).

A wide variety of indigenous or naturally occurring biological agents affect populations of pest insects and mites, in most cases limiting populations to densities well below economic significance. These indigenous, beneficial organisms include predaceous and parasitic species of invertebrates, principally arthropods, in soil and on plants, and a variety of small animals, such as birds, rodents and toads, that feed on insects and mites, and microorganisms (fungi, bacteria, protozoa and viruses) that infect pest species.

The impact of indigenous beneficial organisms is enhanced by procedures that sustain or promote their activity. It is apparent that the application of a broad-spectrum toxic chemical to the crop will kill not only the target insect or mite but also the beneficial arthropods in a field. In addition, use of the chemical may repel birds but also may discourage their feeding because of the sparse numbers of surviving insects. The use of pesticides that are selective for the target pest species and that have minimum effect on non-target species is desirable in a pest management system. Also, the activity of indigenous organisms may be encouraged by such management practices as provision of hedgerows, cover crops, interplanted crops and field borders that can provide a habitat not only for birds but also for parasitic and predaceous insects and mites.

Beneficial arthropods that occur naturally in small numbers may be augmented by introduction of mass-produced colonies. Releases of ladybird beetles have been found effective in controlling aphids on some field-grown crops. Also, the greenhouse whitefly on cucumber and tomato is controlled in a majority of greenhouses in Canada by release of the parasite *Encarsia formosa* Gahan propagated commercially for this purpose. The release of exotic, predaceous and parasitic arthropods has considerable potential for control of pests, particularly introduced pests, for which indigenous parasitic or predaceous species are not present or are not effective.

The augmentation of naturally occurring pathogenic microorganisms for pest insects has potential for a major role in management of pests of vegetable crops. Applications of the bacterium *Bacillus thuringiensis* Berliner represent the most widespread distribution of a mass-propagated, indigenous biological agent. For example, formulations of *B. thuringiensis* are commonly used as alternatives to chemical insecticides to protect cruciferous crops against the cabbage looper, the imported cabbage- worm and the diamondback moth. More recently, formulations of strains of the bacterium have been developed to control the Colorado potato beetle on potato. Applications of *B. thuringiensis* have little or no direct effect on parasitic and predaceous arthropods and other non-target animals in the field habitat and are not considered to be hazardous to humans, domestic animals or crop plants.

Several species of pest insects are susceptible to viruses. These agents, which are usually specific for one species of insect, occur naturally in the field habitat and cause high mortality of some pests. For example, a granulosis virus is a major factor in natural regulation of the imported cabbageworm, and other naturally occurring viruses cause high mortality in populations of several species of cutworms and armyworms. Some entomoviruses have been found to be very effective bio-insecticides when applied to vegetable crops; for example, applications of viruses are effective alternatives to chemical insecticides to control the cabbage looper and imported cabbageworm on cruciferous crops. These viruses are specific for the target insect, have no direct effect on beneficial species, and are not considered hazardous to higher animals and man.

Several species of naturally occurring entomopathogenic fungi cause substantial mortality of some species of pests, but fungi introduced into the field habitat to augment the natural populations generally have not caused significant predictable mortality of pests on vegetables. An exception is the effectiveness of applications of *Verticillium lecanii* (A. Zimmerm.) Viégas and *Aschersonia aleyrodis* Web. to control the greenhouse whitefly on greenhouse-grown tomato and cucumber.

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(Original by W.R. Jarvis and R.P. Jaques)

▶ 3.6 Beneficial plants (allelopathy)

Some higher plants exhibit biological activity against other plants, a phenomenon known as allelopathy. For example, tomato will not grow under walnut trees or on land that has recently been cleared of walnut because of the phytotoxin juglone, which is secreted by walnut roots. Some higher plants exert a similar influence over soil microorganisms that cause disease. In addition, residues of allelopathic plants act, as do many green manures, to enhance the populations of soil microorganisms, many of which compete with or are antagonistic to pathogens. The use of allelopathic plants for disease control does not require registration. Several examples of allelopathy are associated with lettuce, crucifers, marigold and onion.

Lettuce and allied plants — Fusarium crown and root rot of tomato (see Greenhouse tomato, 25.10) can be controlled by growing lettuce between two successive tomato crops in the greenhouse or by planting dandelion beside tomato. Chemicals in lettuce, dandelion and allied plants, such as endive and chicory, interfere with the growth of the fungus *Fusarium oxysporum* f. sp. *radicis-lycopersici* in the soil. The active chemicals in this case are phenolic materials, which retard the growth of *F. oxysporum* f. sp. *radicis- lycopersici.* Some of these compounds, for example cichor- ic acid, also bind strongly to iron, which is required by the fungus.

Cabbage and other brassicas — Residues of cabbage and other brassicas decompose in the soil, releasing volatile, sulfur-containing isothiocyanates and ammonia. These chemicals are toxic to several pathogens, including: *Aphanomyces euteiches,* the root rot fungus of pea and bean; *Rhizoctonia solani,* a widespread root pathogen; and *Fusarium oxysporum* f. sp. *conglutinans,* the causal agent of cabbage yellows. The toxic effects are enhanced when these metabolites are confined by a plastic tarp, as during soil solarization.

The isothiocyanates released by *Brassica* residues are chemically related to methyl-isothiocyanate, the active ingredient of some commercial soil fumigants. A recent study suggested that the release of isothiocyanates from chopped rapeseed tissue incorporated into root-knot nematode-infested soil also suppressed nematode populations; however, other researchers failed to confirm this and suggested that success may vary with cultivar, nematode species, geographic location and climate.

Marigolds — The African marigold (*Tagetes erecta* L.) and the French marigold (*Tagetes patula* L.) are inhibitory to the nematodes *Pratylenchus, Tylenchorhynchus* and *Rotylenchus* spp., and their use with certain vegetable crops may reduce root lesioning and virus transmission. The toxic compounds secreted by marigolds are sulfur-containing polythienyls.

Onion — White rot caused by the fungus *Sclerotium cepivorum* is one of the most difficult diseases of onion to control. Its sclerotia are very long lived in soil and germinate only in the presence of the host, stimulated by various oils (disulfides and polysulfides) secreted by onion. Synthetic onion oils have been found to stimulate the germination of sclerotia of *S. cepivorum* in the absence of the host. The resulting mycelium is very susceptible to a variety of natural biocontrol mechanisms operated by soil organisms, and the population of *S. cepivorum* is considerably reduced. Unfortunately, synthetic onion oils are too expensive to be used on a field scale.

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(Original by W.R. Jarvis and J.W. Potter)

▶ 3.7 Beneficial insects, mites and pathogens *Figs. 3.7a-z*

Besides allelopathic plants, beneficial organisms include parasitic or predaceous insects, mites, spiders, daddy long- legs, centipedes, nematodes, protozoa, bacteria, viruses, and fungi. Indigenous populations of these organisms are considered to have a major role in the natural regulation of many species of pest insects, mites, slugs and snails, and some nematodes and weeds. Unfortunately, their role in natural regulation of pests in commercial production of vegetables has not been assessed or exploited to a significant extent, and the impact of chemical and other control practices on their effectiveness is seldom considered in present management systems. Although there has been extensive research on the introduction of beneficial organisms for control of insects on vegetables, their use in commercial production has been limited to the control of insects and mites on greenhouse crops, and to the application of formulations of the bacterium *Bacillus thuringiensis* Berliner to control insects in various field-grown crops. Some indigenous and introduced species also are beneficial as pollinators (see 1.4).

The major groups of predatory insects and mites, as well as parasites of pest insects, are discussed here, starting with the predators.

Predators

Beetles (Coleoptera; several families) — The adult and larval stages of many beetles are predatory. For instance, ground beetles (family Carabidae) *(3.7a)* are found in or on the surface of the soil and on low plants and under stones. The larvae of certain ground beetles are beneficial, but the adults may be injurious if they feed upon small fruits. Rove beetles (family Staphylinidae) attack all types of insects and some prey on slugs and snails. Their larvae live in the soil or in damp places.

Lady beetles (family Coccinellidae) — The Vedalia lady beetle, introduced from Australia to California in the late 1800s to control an introduced scale insect on citrus trees, is a classic example of pest management by introduction of a biological control agent. In less than two years, the scale was under control. Most lady beetles *(3.7b,c),* or ladybird beetles, are entirely beneficial because both the adult and larva are predaceous. The two-spotted, seven-spotted, and convergent lady beetles are common in Canada. Aphids are their main food source but the eggs and nymphs of other insects also are accepted. Lady beetles overwinter as adults in any sheltered location, becoming active in spring. The females deposit yellow, cigar-shaped eggs in small groups in the midst of aphid colonies. Lady beetle larvae *(12.16b)* start to feed on the prey as soon as they hatch. Usually one generation and sometimes a partial second generation occur per year. During summer, all stages from egg to adult may be present simultaneously. Imported lady beetles show promise for use in greenhouses and may be available commercially. However, when released outdoors they usually disperse beyond the target crop and become ineffective.

Lacewings (Neuroptera; several families) — Lace wings are fragile-looking insects with four lace-patterned wings and a green or brown body. Both the adult and larva of many species feed chiefly upon aphids, though they also attack other insects and mites. Lacewing eggs are deposited singly or in groups of three or four on leaves and stems, often near a colony of aphids. The green lacewings (family Chrysopidae) *(3.7d,e)* may suspend their eggs on long, thread-like stalks. Lacewing larvae are active and voracious. Their jaws are designed for probing crevices, piercing prey, and sucking body fluids. Some lacewings hibernate as adults. Others overwinter as mature larvae in small cocoons of dense, white silk. Lacewings tend to be habitat-specific, and certain species are more common than others in horticultural crops.

Hover flies (family Syrphidae) — Hover flies, sometimes called flower or syrphid flies, *(3.7f)* have the ability to remain stationary while in flight. The eggs are pale yellow and cylindrical, with small, spine-like projections; they are laid on leaves and shoots where there are colonies of aphids. The legless, slug-like maggot (*3.7g,h*) is pale green or red-green and creeps over the plant. Aphids are its primary food source. Hover flies overwinter among dead leaves either as a maggot or pupa. They can be very abundant in some seasons.

Other predatory flies — The midge *Aphidoletes aphidimyza* (Rondani) (family Cecidomyiidae) *(3.7i; 24.12d)* shows promise as a biocontrol agent for use against the melon aphid in greenhouses (see Greenhouse cucumber, 22.33).

True bugs (Heteroptera; several families) — Many true bugs are predaceous *(3.7k,m),* although some, such as the tarnished plant bug (see Celery, 7.21), are pests. The nymphs and adults of true bugs are very active and have a varied diet. The immature and adult stages occur together throughout the season. Some species hibernate as adults under loose bark, among dead leaves or in other sheltered situations, becoming active again in the spring. Predaceous true bugs eat young caterpillars, aphids and spider mites. The minute pirate bug *Orius tristicolor* (White) (*22.34i)* is an example of a predatory bug (family Anthocoridae) that shows promise for use against the western flower thrips in greenhouses (see Greenhouse cucumber, 22.34).

Mantids (Dictyoptera) — The introduced European praying mantis *Mantis religiosa* L. (3.*7j*) is a non-discriminating predator that now may be found in parts of eastern Canada and in the southern interior of British Columbia, particularly around home gardens where its egg masses are left undisturbed.

Ants (family Formicidae) and social wasps (families Sphecidae and Vespidae) — Most ants are opportunistic predators and scavengers on other arthropods. Ants and social wasps may greatly reduce populations of caterpillars and other insects, and they can be found almost everywhere. Sphecid wasps include species that are solitary and others that occur in nesting aggregations. Adult sphecids consume plant nectar and fluids from their prey, using the bodies of their prey to provision a nest for their larvae. In contrast, all hornets and yellow jacket wasps (Vespidae) (*3.7n*) feed their larvae the masticated remains of their prey, which may be insects or other animals.

Predatory mites — Predaceous mites occur naturally, often in large numbers, and some species are available commercially for biological control of small insects and phytophagous mites in greenhouses. One such mite, *Phytoseiulus persimilis* Athias-Henriot (family Phytoseiidae) (*22.36g*), has been used effectively in greenhouses against the two-spotted spider mite (see Greenhouse cucumber). Other predatory mites mentioned in this book are *Amblyseius* (syn. *Neoseiulus) cucumeris* Oudemans (*22.34h*), *Amblyseius* (syn. *Neoseiulus) barkeri* Schuster & Pritchard, and *Metaseiulus* (syn. *Typhlodromus) occidentalis* (Nesbitt). These predatory, foliage-dwelling mites are being used against thrips in greenhouses (see Greenhouse cucumber, western flower thrips). A soil-dwelling mite, *Hypoaspis* (syn. *Geolaelaps*) sp. *(22.31c),* also is being used in greenhouses (see Greenhouse cucumber, fungus gnats, 22.31).

Parasites

Parasitic wasps (Hymenoptera; several families) — More than three quarters of all Hymenoptera are parasitic. They are important in the natural regulation of many insect populations, and species of several wasp families have been introduced into Canada to control crop pests. The ichneumonids, braconids (3.*7r),* chalcids (*3.7p*), eulophids, pteromalids and scelionids include the major, naturally occurring biocontrol agents. Their bodies are long and slender, and in females often end in an ovipositor. Parasitic wasps attack the immature stages of other insects; some are host specific, whereas others attack a wide range of insects. The female parasite lays an egg in or near the host's body and the parasite larva feeds on the body fluids of the host, slowly killing it. Only one parasite larva is associated with a host in the Ichneumonidae. Braconids lay many eggs on a single host (*3.7u*). The parasitic wasps that are available commercially for use in greenhouses are *Encarsia formosa* Gahan (*3.7q)* against the greenhouse whitefly (see Greenhouse cucumber, 22.32, and Greenhouse tomato, 25.27), and *Aphidius matricariae* Haliday *(3.7s,t)* against aphids (see Greenhouse pepper, 24.12).

Parasitic flies (notably the family Tachinidae) — Tachinid flies parasitize moths, butterflies, sawflies, leaf beetles, and some land slugs and snails. Their host range includes many pests of horticultural crops. The larval stages of tachinid flies are parasitic, usually on other insects (3.7v). Some tachinid females lay their eggs on their hosts, but the majority of them retain their eggs and deposit them fully developed with an active maggot ready to hatch. The females of some tachinids pierce the host and inject their eggs. Others broadcast their eggs, or lay them separately on a plant for the host to swallow. The larval stage passes inside a single host, which is killed in the process. When mature, the larva usually abandons the carcass of its host, pupating and overwintering in the soil. Adult tachinid flies are active, fast-flying insects that feed on nectar or honey- dew and search erratically for suitable hosts. Tachinids figure prominently in importations and releases for biological control programs in Canada.

Pathogens and other parasites of insects — Naturally occurring pathogenic bacteria, viruses, fungi, protozoa and nematodes contribute substantially to regulation of many species of insects on vegetables. For example, several species of naturally occurring bacteria kill insect pests of vegetable crops; however, factors that influence their pathogenicity and procedures required to increase their effectiveness in reducing populations of insect pests are not well understood. The bacterium *Bacillus thuringiensis* Berliner is the only pathogen that is used commercially in Canada to protect vegetable crops from insect pests. Crystals formed in the sporangium of the bacterium contain an endotoxin which, upon ingestion by a susceptible insect, causes paralysis and death. Strains of *B. thuringiensis* are used quite extensively to control the larvae of several foliage-eating butterfly and moth pests of vegetables, particularly on cruciferous crops (*3.7y,z*) and, more recently, to control the Colorado potato beetle on potato and tomato.

Insect pests of vegetables are killed by various species of indigenous fungi, notably species of *Beauveria, Entomophthora* and *Metarhizium.* Under favorable conditions, these and other fungi may decimate insect populations. *Beauveria bassiana* (Bals.) Vuill., for example, kills substantial numbers of adult Colorado potato beetles in some years, and species of *Entomophthora* kill

various species of bugs and flies, such as root maggot adults (see Onion, onion maggot, 13.26). The green muscardine fungus *Metarhizium anisopliae* (Metsch.) Sorokin shows promise for control of white grubs in the soil. Other entomopatho- genic fungi include *Aschersonia aleyrodis* Web. (3.7w) and *Verticillium lecanii* (A. Zimmerm.) Viégas; both infect pupae of the greenhouse whitefly.

Viruses kill several species of insects on vegetable crops, in some cases causing high mortality. The most common are the Baculoviruses, that is, the polyhedrosis viruses and the granulosis viruses, which kill larvae of several species of butterflies and moths (order Lepidoptera). For example, a naturally occurring granulosis virus is considered to be instrumental in regulation of the imported cabbageworm, and polyhedrosis viruses contribute substantially to suppression of the cabbage looper and to the oscillations in populations of the fall armyworm. Applications of these viruses are very effective in controlling the cabbage looper and the imported cabbageworm on cruciferous crops (*3.7x*). However, the viruses are not registered for use at this time.

Several species of protozoa (order Microsporidia) are parasitic on insect pests of vegetables, particularly Lepidoptera. Parasitism by microsporidia may suppress populations of susceptible insects by reducing vigor and fecundity, often without killing the host. Several species have potential for use in pest management; *Nosema locustae* Canning, a parasite of grasshoppers, is of particular interest in Canada.

Many species of pest insects are susceptible to parasitism by nematodes, principally of the families Steinernematidae and Heterorhabditidae. There is particular interest in these insect-parasitic nematodes for control of soil-inhabiting pest insects. One possible disadvantage in using these nematodes is that they are readily killed by desiccation, thus limiting their usefulness.

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(Original by R.P. Jaques and J.A. Garland)

▶ 3.8 Chemical control

Pesticides

Pesticides are regarded by many as controls of last resort because they add considerably to the cost of production and their misuse creates high-profile environmental problems. Nevertheless, in well-planned, integrated pest management programs, pesticides have a valuable role if used judiciously. A major problem with many pesticides is the rapid development of pathogen races or insect and mite populations that are resistant to them. When that happens, their continued use is pointless; not only do they have little or no efficacy, but they can aggravate the situation by killing other organisms that contribute to the natural biological control of pathogens and other pests. Blanket, season-long pesticide programs used as insurance in anticipation of pest outbreaks are seldom justified if other precautions have been taken.

The application of crop protection chemicals to vegetable crops or to the soil is a widely used method of controlling diseases, insects, weeds and many other pests. Because of the environmental impact of the use of chemical pesticides and because of their toxicity to humans, management procedures to minimize their use and their effects on non-target species are being developed and implemented. Accordingly, applications of effective pesticidal chemicals should be made in response to indications that populations of the pest have developed or will develop to economically significant densities. The threshold at which action is taken is dependent on the type of damage inflicted on the crop, especially the marketed portion of the crop. Populations of pests on crop plants are monitored to determine the timing and necessity for applications. This procedure of pest management minimizes the amount of chemical applied throughout the season. Unfortunately, many growers prefer to apply pesticides indiscriminately to crops according to a predetermined schedule as insurance against damage by pests. This practice

increases the detrimental environmental impact of adding pesticide chemicals to the field habitat and increases the cost of crop production. Also, the application of some chemicals causes stress to crop plants and may make them more susceptible to disease and other pests. On the other hand, scheduled applications of pesticides may be necessary to prevent development of economically damaging populations of pests, such as root maggots and other pests within roots or stems, that cannot readily be killed when crop damage is occurring, or pests for which populations cannot be predicted with acceptable accuracy.

Environmental impact

The unnecessary use of pesticides is discouraged because of their direct and indirect effects on the field habitat and environment, on the consumer and the grower, and on the cost and efficiency of production. Most insecticidal and acaricidal chemicals kill or are harmful to several species of non-target arthropods, including parasitic and predaceous species that contribute to regulation of primary or potential pests. There are cases where additional applications of pesticides were required to control pests whose populations increased to a damaging level following application of a pesticide to control a different species. Applications of chemicals may affect populations of birds and other animals that feed on pest species, and residues of some chemicals in soil and on plant debris may affect microbial activity in soil, thus influencing the availability of plant nutrients. In addition, there is concern for the possible harmful effects of residues of chemical pesticides in food and for the effects on growers of excessive exposure to chemicals. Also, the tendency for pest species to become resistant or tolerant to a pesticide is enhanced by repeated use of the chemical, necessitating the use of higher concentrations or the use of other chemicals, often resulting in greater crop loss and increased cost of production.

(Original by W.R. Jarvis and R.P. Jaques)

▶ 3.9 Management by exclusion and regulation

Legislative control measures

Federal legislation administered by Agriculture Canada and complemented by provincial legislation is the means by which certain diseases and pests are regulated in Canada. Such regulations are aimed at preventing the introduction of new diseases and pests and their movement within Canada. The Canada Seeds Act and the Plant Protection Act provide for field surveys; inspection of produce, premises, containers and packages; destruction or treatment; restriction on movement; and other safeguards and requirements as specified in regulations under these Acts. Treatment of infested commodities, short of destruction, consists of fumigation, cleaning in a manner that is satisfactory to an inspector, or diversion to an approved industrial process.

There are substantial reasons for concern about pests that may be associated with vegetable commodities at the time of harvest. For instance, pests may be disseminated to other production regions within the country, a possibility that is greatly enhanced by modem, rapid transportation facilities. Pests also have resting stages that may survive cleaning and packaging operations and storage. The harvested, unprocessed commodity itself may serve as a pathway for the spread of pests. Apart from cold storage, which may affect the survival of some pests, Canada does not yet have the necessary facilities for bulk treatment and disinfestation of commercially produced quantities of vegetable commodities. Research into such alternative pest control technologies has only just begun.

Further information about the current status of federally regulated pests can be obtained from Agriculture Canada.

(Original by W.P. Campbell, P.M.D. Martin and J.A. Garland)

▶ 3.10 Foreign diseases and pests

Exclusion from Canada of the following diseases and pests is of particular importance for vegetable crops.

Potato gangrene

Phoma exigua var.***foveata*** (Foister) Boerema

Potato gangrene is caused by a soil- and tuber-borne fungus not known to occur in North America. Gangrene manifests on the tuber surface as extensive, dark brown or purplish, sharp-edged lesions, with variously shaped hard rots and cavities developing beneath the lesions.

(Original by A.R. McKenzie)

Potato viruses

Andean potato latent virus
Andean potato mottle virus
Arracacha virus B
Tobacco ringspot virus, potato calico strain
Potato deforming mosaic virus
Potato mop top virus
Potato virus T

Potato virus V
Potato virus X, resistance breaking strain

These and other viruses that occur in other countries are of concern to Canada because of their potential effects on crop yield and impact on trade. Their importation into Canada poses a threat to the potato industry in Canada, and thus they are cause for regulatory concern. To protect Canadian producers from potentially harmful viruses, the importation of potatoes from all countries other than the USA is prohibited. However, germplasm for research or commercial evaluation can be imported through Agriculture Canada's post-entry quarantine program.

(Original by I.A. MacLatchy and J.G. McDonald)

Columbia root-knot nematode *Figs. 3.10a,b*

Meloidogyne chitwoodi Golden *et al.*

The Columbia root-knot nematode has not been reported in Canada, but it is widely distributed in the western United States. It was described as a distinct species in 1980 after years of confusion with the northern root-knot nematode, which is widespread in Canada (see Carrot).

The Columbia root-knot nematode is a pathogen of bean, carrot, corn, pea and potato. It also affects canola, sugar beet and cereals, such as wheat, barley and oat. On potato, it induces galls *(3.10a)* on the surface of the tubers, greatly downgrading them. When tubers are sliced, the body of the female nematode can be seen as a tiny, water-soaked dot, and masses of eggs appear as small brown spots *(3.10b).*

(Original by I.R. Evans and T.C. Vrain)

Potato-rot nematode *Fig. 16.37*

Ditylenchus destructor Thorne

The potato-rot nematode is a widespread and serious pest of potato in Europe; it also occurs in many Asian countries and has been reported from New Zealand and Peru. In South Africa it is an important pest of peanut. In the United States, it has been found in Arizona, California, Hawaii, Idaho, Indiana, New Jersey, Oregon, Washington and Wisconsin, and it is subject to quarantine regulations in some of those states. In Canada, the nematode was detected in potato in 1945 in three small areas of Prince Edward Island, and in 1952 in a small potato planting on Vancouver Island, British Columbia. Following eradication procedures and provincial legislation, the infested fields in P.E.I. have been kept out of potato production, and the nematode has not been detected in surveys conducted since the early 1960s. The potato-rot nematode also attacks bulbous iris and over 80 other hosts, including such vegetables as beet, carrot, celery, crucifers, cucurbits, eggplant, onion, pepper, rhubarb and tomato. Information on host range, diagnostic techniques, feeding habits, ecology and persistence of *D. destructor* in the field is incomplete; most infestations are detected only when found in mature potato tubers.

The potato-rot nematode feeds chiefly on fungi and can survive in weed hosts and in fallow soil for several years, but it seldom survives in rotted tubers in storage or in the field. Infestations are spread mainly by seed tubers. The nematode feeds first on the roots and later invades the tubers, in which it feeds just under the skin. Initial symptoms include small (0.3 mm diameter) pits surrounded by snow-white rings that are visible when the tuber is peeled. Continued feeding results in a depressed area in the skin, which becomes necrotic and dries out, resulting in characteristic irregular, triangular cracks. Severely affected tubers have large, sunken areas covered by dried-out skin *(16.37).* The cracks in the skin provide ready access to bacteria and fungi, and affected tubers usually rot in storage or in the soil.

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(Original by W.L. Seaman and R.J. Howard)

Pepper weevil *Figs. 24.13a-e*

***Anthonomus eugenii* Cano**

The pepper weevil was found in Canada for the first time in 1992 in greenhouse pepper. See Greenhouse pepper (24.13) for further information.

Potato tuberworm *Fig. 3.10c*

Phthorimaea operculella (Zeller)
(syn. *Gnorimoschema operculella* (Zeller))

The potato tuberworm, a moth (family Gelechiidae), has a worldwide distribution in tropical and subtropical latitudes. In North America, it occurs in the southern United States and Mexico but not in Canada. Larvae of this moth feed on Solanaceae and may be encountered on potato and tomato imports as a post-harvest pest. The most recent importation into Canada occurred during 1989-90, when larvae were found in or on tomatoes originating from Australia. Interceptions on potatoes imported into Canada have been recorded but not for many years, the last time being 1972- 73 in chipping potatoes originating from the United States.

The only confirmed case of this insect as a field pest in Canada was in 1958 at Duncan on Vancouver Island, British Columbia. That infestation did not survive and it is thought to have been eliminated by exposure to the elements, leaving open to question the potential for survival in warehouses in Canada. (See Mackay, 1972, for a warehouse find.)

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(Original by J.A. Garland)

Sweetpotato whitefly *Figs. 3.10d-g*

Bemisia tabaci (Gennadius)

The sweetpotato whitefly (family Aleyrodidae) is widespread elsewhere in North America, especially at more southern latitudes. It was imported into Canada on cuttings of ornamental plants from the southern United States in 1987, 1988 and 1989. Field infestations were detected in 1988 on tomato plants around an ornamental greenhouse in Leamington, Ontario, but these did not survive the winter. Sweetpotato whitefly occurred at damaging levels in several greenhouse tomato crops in British Columbia in 1988 and 1989; a ripening disorder, similar to blotchy ripening (25.25), was associated with the presence of adults and immatures of this whitefly and resulted in severe losses in those crops.

The sweetpotato whitefly will not survive the winter outdoors in Canada, although infestations could be carried over in ornamental greenhouses and on houseplants. It is considerably more difficult to control both chemically and biologically than the greenhouse whitefly (see Greenhouse cucumber, 22.32, and Greenhouse tomato, 25.27).

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(Original by J.L. Shipp and D.R. Gillespie)

Tomato pinworm *Fig. 3.10h*

Keiferia lycopersicella (Walsingham)

The tomato pinworm (family Gelechiidae) occurs in Mexico, the southern United States, the Caribbean, and Hawaii. Larvae of this moth feed in developing tomato fruits and may be encountered on post-harvest tomatoes as an imported pest. The most recent importation into Canada occurred during 1988-89, when larvae were found in or on tomatoes originating from California. This moth was unknown in Canada prior to 1946, when larvae were confirmed in field tomato crops and in greenhouses in southwestern Ontario; however, these infestations did not survive. There since have been two isolated infestations in Canada: in 1970 in a greenhouse on Vancouver Island, and in 1975 in a greenhouse and surrounding home gardens at Kamloops, British Columbia. Eradication at both locations was accomplished by exposure to the elements and by planting cucumber, which is a non-host crop. These occurrences probably had their origin in post-harvest tomatoes that then were being imported during the off-season from Mexico and the southern and western United States.

These experiences show that the tomato pinworm cannot survive the winter outdoors in Canada. Temporary greenhouse infestations can be avoided by destroying used shipping containers and by screening greenhouse openings, and they can be managed by including cucumber or other non- solanaceous plants in the greenhouse cropping cycle.

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(Original by J.A. Garland)

▶ 3.11 Introduced diseases and pests

The following diseases and pests have been introduced from foreign countries and have become endemic in certain regions of Canada. Regulations have been established to prevent their spread to non-infested areas.

Bacterial ring rot *Figs. 16.1a-e*

Clavibacter michiganensis subsp. *sepedonicus* (Spieckermann & Kotthoff) Davis *et al.*
(syn. *Corynebacterium sepedonicum* (Spieckermann & Kotthoff) Skapston & Burkholder)

In Canada, widespread infections of bacterial ring rot no longer occur in potato fields, although there are still sporadic outbreaks in various regions (see Potato, 16.1). The Canada Seeds Act sets a zero tolerance for this disease, and regulates seed potatoes for domestic and export purposes. There also are provincial restrictions in Alberta, British Columbia, New Brunswick and Prince Edward Island. The combined effect of these programs has been to eradicate the disease in most areas, and there have been relatively few occurrences of ring rot in the last few years in these provinces.

Potato wart *Figs. 16.21a-d*

Synchytrium endobioticum (Schilbersky) Percival

In Canada, this disease occurs only in Newfoundland and parts of Labrador. The French islands of St. Pierre and Miquelon also are considered to be infested. (See the following discussion on cyst nematodes and see also Potato, wart, 16.21.)

Potato spindle tuber *Figs. 16.28a,b*

Potato spindle tuber viroid

Potato spindle tuber viroid has been reported in Canada and the United States, but it has been eradicated from two of the largest seed-producing provinces. It is transmitted mechanically by contact and by chewing insects, and it is dispersed in potato seed and pollen as well as in tubers. In Canada, the effect of a zero tolerance in federal and provincial programs has been to eradicate the disease from potato seed- producing fields. Prince Edward Island and New Brunswick have been declared free of this viroid after official surveys were conducted.

Potato virus Y^N *Figs. 3.1 la-c; 16.27b*

PVY^N (tobacco veinal necrosis strain)

The tobacco veinal necrosis strain of potato virus Y (PVY^N) has been present in Europe for many years, but it seldom has been detected in North America. In 1989, it was diagnosed in tobacco and potato in southern Ontario, and between 1990 and 1992, it was found in potato crops in parts of Prince Edward Island, New Brunswick, Nova Scotia, Quebec, Florida and California.

Most strains of this virus are almost symptomless in potato and cause minimal, if any, yield loss in this host, which at most displays only slight mottling of the leaves. In tobacco, however, the virus causes leaf-yellowing and darkening of the veins, which are conspicuous symptoms, and yield losses may be considerable. In the field, PVY^N is spread non-persistently by aphids. It overwinters principally in potato tubers. (See also Potato, potato virus Y, 16.27.)

Potato cyst nematodes *Fig. 16.36*

Golden nematode *Globodera rostochiensis* (Wollenweb.) Behrens
Pale cyst nematode *Globodera pallida* (Stone) Behrens

These two species of *Globodera* affect potato; both occur in Newfoundland, and the golden nematode also has been found on Vancouver Island. Most countries require freedom from potato wart and from the two potato cyst nematodes. These pests are regulated in Canada from Newfoundland; the golden nematode also is regulated from Central Saanich, British Columbia, and all three pests are regulated from any country in which they occur to all other areas of Canada. Plants or any object that may be contaminated with soil, such as used bags, containers, sacks, covers and vehicles, are regulated. Potato tubers are the most likely vegetable commodity to be affected by and possibly harbor stages of these pests, even after washing. Other commodities, such as vegetable transplants or nursery stock grown in infested soil, are suspect if soil adheres to their roots, fruits, or vegetative parts. For this reason, field- grown tomato and eggplant fruits are regulated from infested areas; they cannot be moved out of an infested area if soil adheres to them. Potatoes in storage must be treated with a sprout inhibitor, and a domestic movement certificate is required.

The use of potato cultivars, such as Cupids, which is resistant to wart and to the potato cyst nematodes, may diminish the risk of dispersing these pests. In the meantime, federal legislation in Canada prevents the spread of these pests by prohibiting or controlling the movement from Newfoundland and Labrador of soil, plants, equipment, and other materials to which resting spores or cysts may be attached. Potato production in the affected area of Central Saanich in British Columbia has been prohibited since 1982.

Colorado potato beetle *Figs. 16.44a-d*

Leptinotarsa decemlineata (Say)

The Colorado potato beetle (see Potato, 16.44) now occurs in most areas of Canada but is regulated on potato plants and plant parts, except true seed, destined for Newfoundland. This regulation is intended primarily for potatoes that are bagged in the field, where the overwintering summer adults may gain access to bags before they are closed. However, deregulation has been proposed because field bagging is no longer practiced commercially.

European corn borer *Figs. 12.16a-h*

Ostrinia nubilalis (Hübner)

The European corn borer (see Maize) is regulated on certain commodities destined for British Columbia. Plants or plant parts of maize (corn) and sorghum are the most likely vegetable commodities to be affected by and possibly harbor the larva, which may tunnel into the shanks and ears of fresh-market sweet corn or be inside pepper fruit. The tops of bunching beets are a potential pathway for larval dispersal.

Japanese beetle *Fig. 3.11d*

Popillia japonica Newman

The Japanese beetle (family Scarabaeidae) occurs in parts of Ontario, Quebec and generally throughout the northeastern United States. It is regulated on plants from areas of the world where it occurs. Proposed regulations between Canada and the United States would ensure a harmonized North American approach to regulating Japanese beetle.

Brown garden snail *Fig. 3.11e*

Helix aspersa Müller

The brown garden snail is present in many localities in southwestern British Columbia, including Vancouver Island. It is regulated on plants destined for areas of Canada where it does not occur.

(Information on introduced diseases and pests courtesy of Agriculture Canada, Food Production & Inspection Branch)

▶ 3.12 Management of nematode pests *Fig. 3.12*

Successful management of plant-parasitic nematodes in vegetable crops requires the integration of cultural practices, resistant cultivars, and chemical nematicides to reduce nematode populations at planting. Pesticide use may be economically feasible when other management strategies prove too costly or inadequate. For information on the major nematode pests of vegetable crops in Canada and their management, see Carrot, root-knot nematode; Onion, stem and bulb nematode; Potato, root-lesion nematode, potato cyst nematode, and stubby-root nematodes; and Beet, sugarbeet cyst nematode.

Monitoring

There is a direct relationship between population density of plant-parasitic nematodes in the soil at planting and crop damage, and theoretically a damage threshold can be established for each parasitic nematode on its respective vegetable host. When nematode density in the soil exceeds this level at planting, losses in yield, quality or both become noticeable. Determining such thresholds is time consuming. Relatively little information of this type is available for Canadian vegetable crops. In practice, nematode numbers in the soil are used to predict nematode damage; this is also the first step in diagnosis. After determining the density of nematodes in a soil sample, the number is often extrapolated to estimate the population in the entire field; however, caution is advised if using this procedure. An average count above a certain threshold could mean only that some areas of the field will show crop injury.

Cultural practices

It is almost impossible to design effective and economical rotation schemes for farms that grow vegetables exclusively. Destruction of infested crop residue, clean fallowing between crops, and rotation with non-host crops are effective measures for nematode species that attack only a few crops. Useful rotations against the sugarbeet cyst nematode are alfalfa, cereals, and bean and potato, which are non-host vegetable crops. The root-lesion and root-knot nematodes have very wide host ranges and are more difficult to manage. Some grasses and cereals, such as wheat, barley, oat and rye, which are non-hosts of the northern root-knot nematode, are used extensively. An ideal practice for reducing numbers of root-knot and root-lesion nematodes in small vegetable plantings is to interplant with French marigold, *Tagetes patula* L., or African marigold, *T. erecta* L. The use of these plants is more effective than fallowing or other cultural practices. The nematodes are attracted to and penetrate *Tagetes* roots but are unable to feed and multiply. Consequently, the density of nematode pests in the soil is lowered. Marigolds are not effective against *Heterodera, Ditylenchus,* or most ectoparasitic species.

Solarization is practical for small gardens. Intense summer sunlight can raise the soil temperature above 40°C beneath a transparent plastic tarp, killing many nematodes to depths of 5 to 10 cm. The soil must be well worked and moistened so that the

heat will penetrate evenly. The soil should be covered for 3 to 6 weeks. Control is complete at the surface but less so at greater depths.

Despite the extra cost, the use of certified, nematode-free seed and transplants is usually worth the expense. Transplants should be vigorous and free of root galls or lesions caused by nematodes. The use of soil-free media, or pasteurized and fumigated soil, is suggested for cucumber, tomato and other susceptible crops. To eliminate plant-parasitic nematodes from small quantities of soil to be used to germinate seeds and grow transplants, growers should moisten the soil for several hours, then heat it to 80°C for one hour. In heavily infested greenhouse soils, annual or even semi-annual pasteurization may be required to avoid severe damage to greenhouse cucumber and tomato crops, as well as to some ornamentals sometimes found in vegetable greenhouses.

Resistant cultivars

A few vegetable cultivars are resistant to species of nematodes. In most cases, there is some degree of resistance to some but not all root-knot species or other plant-parasitic nematodes. There are no vegetable cultivars with specific resistance against the root-lesion nematode or the northern root-knot nematode, but some are more tolerant; for instance, some carrot cultivars have been shown to be more tolerant than others to root-knot nematode infection, and nematode-resistant rootstocks of tomato are available for grafting.

Biological control

Microbial nematicides are not yet commercially available. However, some soil microorganisms are natural enemies of nematodes and there are ways to enhance their activity. Nematode-trapping fungi, for instance, can be stimulated by heavy applications of manure or other types of organic matter.

Chemical control

Several fumigant nematicides were developed in the 1950s. Non-fumigant, non-phytotoxic nematicides that can be applied at planting have been developed, but lack of registration for use on vegetable crops prevents their use. A successful fumigation *(3.12)* under optimal conditions and at the recommended rate should eliminate 80 to 90% of nematodes to a depth of 25 cm. Activity depends on the presence of water and air in the pore spaces of the soil. Fumigant gas dissolves in water, killing the nematodes. Some of the gas adheres to organic matter and plant residues. The gas moves approximately 1000 times further in air than in water, and faster in warm than in cold soil. In wet soil in which pore spaces are filled with water, the gas dissolves but cannot diffuse; consequently, most of the spaces in the medium are not reached and not all nematodes are killed.

All fumigant nematicides are phytotoxic. It is essential to allow enough time for the gas to disperse from the medium being treated before seeding or transplanting. The aeration process usually requires cultivating or turning the medium once a week for two to three weeks between the time of fumigation and planting. A further disadvantage of fumigants is that they kill beneficial organisms, such as mycorrhizal fungi, *Rhizobium* bacteria, predatory nematodes, and fungi and bacteria that compete with or prey upon the plant-pathogenic nematodes.

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(Original by T.C. Vrain)

▶ 3.13 Management of weed pests *Fig. 3.13*

By managing weeds in headlands and other non-productive areas, and by preventing them from setting seed on crop land, growers can gradually decrease the reservoir of weed seeds in vegetable fields (see also Weeds, 2.3).

Monitoring

Scouting vegetable fields, particularly in the early stages of crop emergence, is essential for making decisions on the management of weeds. Although information on the economic threshold levels for specific weeds in vegetable crops grown in Canada is generally not available, some assessment of the weed population is required to decide whether a herbicide application or cultivation is necessary. Similarly, regular monitoring is required to properly time management operations when weeds are at a susceptible stage (*2.3a-q*). In a field where weeds are uniformly distributed, a zig-zag pattern for scouting is recommended.

A map of the field showing dense weed patches or areas requiring special management should be prepared for immediate as well as future use. Because most weeds originate from the soil seed-bank, there usually is some consistency in weed problems from one year to the next.

Cultural practices

Weeds can be managed mechanically by cultivation, mulching and mowing. Cultivation and hand hoeing have been relied upon for centuries to control small weeds in row-cropped vegetables and for improving soil aeration. However, if cultivation is too frequent or if the soil is too moist, compaction occurs and can adversely affect crop growth. Frequent rototilling can destroy soil aggregates and structure.

Mulching with plastic has been a common practice in recent years for heat-responsive crops, such as cucumber, melon, pepper, sweet corn and tomato. Although clear plastic mulch provides the highest soil temperature, it also permits weed growth. Black plastic, which blocks sunlight, will inhibit weed growth. A newly developed plastic permits the transmission of infrared radiation and is being studied for its effectiveness in preventing weed emergence. Recent research also has focused on the use of living mulches or surface crop residues for reducing or suppressing early weed emergence. Other strategies, such as rotation and altering row widths and seeding rates, have an important role in integrated weed management.

Biological control

BioMal, the first commercial mycoherbicide in Canada, was registered in 1992 for the control of round-leaved mallow in certain field crops. The active ingredient is the pathogenic fungus *Colletotrichum gloeosporioides* f. sp. *malvae* Mortensen. In the future, this product also may be registered for use in some vegetable crops. Results from research are expected to provide new mycoherbicides for controlling other species of weeds. In contrast to most chemical herbicides, biocontrol agents (insects or plant pathogens) are very specific for individual weed species. Biological control methods for weeds in vegetable crops will require the application of an organism to the weed to be controlled, a practice called inundative biocontrol. Classical biocontrol of weeds involves the establishment in an area of organisms from another region to provide ongoing control. This latter method is suitable for rangeland and other non- cultivated areas, but not for annual vegetable crops.

Chemical control

Herbicides are commonly used along with cultivation to provide overall weed control. Chemical companies rarely develop herbicides specifically for vegetable crops because of the limited market. Scientists, therefore, have had to adapt existing herbicides for vegetable production. In many instances, federal and provincial researchers in Canada provide the data on efficacy and crop tolerance required for the registration of herbicides through the "User Requested Minor Use Label Expansion" (URMULE) program.

The recent development of sethoxydim and fluazifop-p- butyl has contributed significantly to the control of annual grasses and quack grass in dicotyledonous vegetable crops. These products are applied after emergence. Because they do not have significant soil residual activity, later weed flushes may reinfest vegetable crops and interfere with harvesting. These products are used in vegetable production, on both mineral and organic soils, to destroy cereal windbreaks before they compete with vegetable crops.

The status of chemical weed control for broadleaved weeds in vegetable crops ranges from excellent for carrot, potato, sweet com, and tomato to unsatisfactory in cucurbits and most low-hectarage vegetable crops. Other modes of weed control need to be employed for these crops. In situations where only pre-plant incorporated or pre-emergence herbicides are specified, growers must rely on hand weeding and cultivation to remove weeds that emerge later.

Crop injury from herbicide drift or soil residues

Herbicides, such as 2,4-D and dicamba, which act as plant growth hormones, often injure susceptible vegetable crops by accidental drift or through the use of contaminated sprayers or watering cans. Typical symptoms are stem or petiole bending, leaf curling and cupping, and abnormal leaf- vein development. Picloram residues in manure from cattle fed with a treated crop often have caused problems in home gardens, and occasionally in commercial fields.

Sulphonylurea (chlorsulfuron, metsulfuron methyl) and imidazolinone (imazethapyr, imazamethabenz) herbicides can be injurious to most vegetable crops, even at very low concentrations. Soil residues of these herbicides can produce stunted, chlorotic growth and cause plant mortality. Trifluralin products are widely used for weed control in vegetable crops. However, in the following year, carry-over in soil can seriously damage beet, sweet corn and, to a lesser extent, cucurbit crops. Close monitoring is necessary to ensure that only tolerant crops are grown after triazine and urea herbicides are used.

In greenhouse crops, damage may result from the use of cereal straw or other mulches and amendments that have been contaminated with herbicides. Occasional cases of direct injury to crops from careless spraying of alleys, walkways or areas beneath benches also have been reported.

Future trends in weed management

Alternative methods of weed control will become increasingly important to vegetable growers in the future. Although these techniques will likely reduce the reliance on chemical herbicides, they will be only a partial substitute.

Reducing the use of herbicides — Methods by which herbicide use can be reduced include banding applications; using new, low-rate herbicides; timing of post-emergence herbicides to maximize effectiveness; placing fertilizer below the seed to target the crop instead of weeds; using new herbicide combinations, adjuvants, and application equipment; monitoring and determining weed thresholds as a basis for applying herbicides.

Alternatives to chemical herbicides — Alternative strategies for weed management are becoming increasingly important to vegetable growers. Reliance on chemical herbicides may be reduced but not eliminated. Methods under study and development include: biological control by weed-specific mycoherbicides; weed suppression by cover crops; rotation; the use of allelopathic plants (see Beneficial plants, 3.6) as green-manure crops; and the use of liquid- nitrogen fertilizer to control certain weeds in cruciferous crops. Chemical herbicides will likely remain the major method of weed control for some time, although specific products can be expected to change. The general trend to use less herbicide will continue, with greater reliance on cultural practices, including cover crops for weed suppression and soil conservation.

Genetically engineered crops — Biotechnology may have an impact on weed management through the development of vegetable cultivars with resistance to low rates of "environmentally friendly" herbicides and by the incorporation of allelopathic properties into vegetable crops.

Herbicide-resistant weeds — The first occurrence of a herbicide-resistant weed in North America was documented in 1970 in a commercial nursery in Washington State, where common groundsel, which originally was susceptible to simazine, had become resistant. This herbicide had been used for many years at this location. With prolonged use, in some cases for as few as five years, resistance to most of the herbicide groups with similar modes of action has developed in a number of weed species, although some of these resistant biotypes have only recently been detected. Methods used to delay or prevent the development and establishment of herbicide-resistant weeds include: rotating crops; rotating herbicides (using herbicides with different modes of action); using short-term or non-residual herbicides; using the lowest possible effective rate; using herbicide mixtures; and practicing cultural/mechanical control where possible.

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(Original by R. Esau)

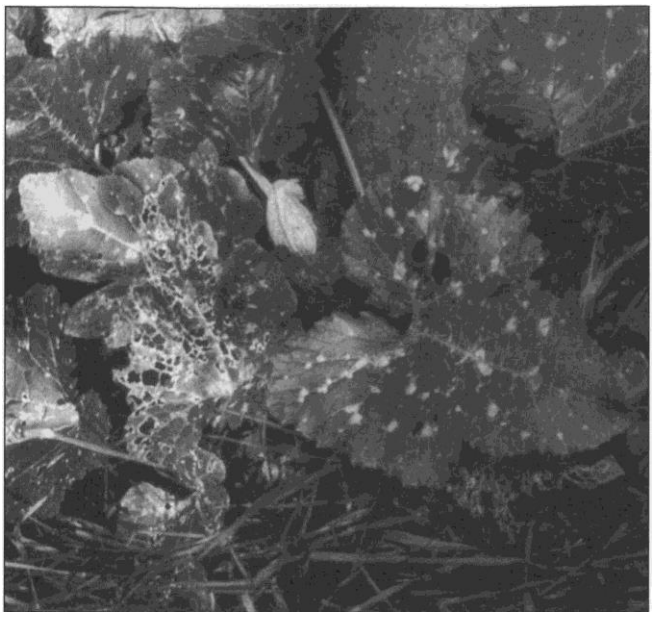
▶ 3.14 Managing diseases and pests in home vegetable gardens *Fig. 3.14T1*

Vegetable gardening is a rewarding and enjoyable hobby that provides a supply of fresh produce in season and that also can help to reduce a family's food costs. Vegetable crops are threatened by various disease, insect and weed problems that may reduce the yield and quality of the produce and even destroy the plants. Diseases and pests can damage vegetables from the time seeds are planted until after the crops are harvested, and gardeners who fail to follow good growing practices that minimize damage from diseases and pests in their gardens will suffer many disappointments.

The impact of plant diseases on vegetables often appears less direct than that of insects and weeds. Insects usually can be seen with the naked eye, and their activities quickly noted, while weeds are conspicuous, competing directly with vegetable plants for space, nutrients and moisture. Diseases, on the other hand, may make their presence known only when plant growth begins to slow down or when productivity is less than expected. No causal agent may be visible, only symptoms indicating that something is wrong.

Based on general cause, there are two kinds of diseases: biotic and abiotic. Biotic diseases are caused by an identifiable microorganism or infectious agent, for example, bacteria, fungi and viruses. Abiotic diseases result from unfavorable growing conditions or environmental stresses, such as extreme temperatures, too much or too little water, and nutrient imbalances. Both kinds of diseases exist widely in vegetable gardens across Canada.

Plant-feeding insects, mites and nematodes become pests if they injure crop plants sufficiently to interfere with the capacity of the plant to produce food. Some species of insects also transmit plant pathogens. Aphids, for example, transmit several viruses, and cucumber beetles can spread bacterial wilt and squash mosaic virus. The presence of some plant-feeding pests can be tolerated, but control may be necessary if the pests begin to cause significant damage. Home gardeners usually tolerate more pest injury, especially cosmetic injury, to their vegetables than do commercial producers. Many pest species are kept at a low level by natural enemies, such as predators and parasites, and additional control measures may not be required. It is important to realize that the population build-up of natural parasites and predators lags behind that of the pests, so some damage usually is evident. Special practices may have to be employed to keep the pest population at a level where damage will not be serious.



3.14T1 European earwig feeding injury to squash; in many urban areas of Canada, the European earwig has become one of the most annoying and difficult to control pests in home gardens. For more information, see Crucifers, 8.43, and color figures ***8.43a-d.***

The most effective pest control strategy for the home gardener utilizes a combination of practices; these include planting resistant cultivars, providing optimum soil conditions, fertility and moisture supply, selecting pest-free seed and transplants, employing crop rotations, monitoring carefully for pests, destroying plant residue, cultivating, mulching, and using pesticides only when necessary. Such practices often keep the populations of garden pests at tolerable levels. The role of these and other techniques in helping to minimize disease, insect and weed problems in home vegetable gardens is discussed here.

Monitoring

Traps and lures — Baits and lures using light, color, sex attractants (pheromones) or food to draw a pest to a trap may be effective for some pests. Wireworms can be trapped by burying pieces of potato or carrot in the soil, then checking the bait every few days for the insect. Infested baits can be collected and destroyed. Slugs can be drawn to reservoir traps of beer or to a mixture of molasses, water and yeast. Boards laid out in the garden can be used to trap slugs because they look for a cool, damp spot to hide during the day. These pests can then be collected and destroyed daily. Earwigs can be trapped in wooden traps or in containers filled with water containing a small amount of detergent. Growing an insect pest's favorite plant as a trap crop to lure it from the garden also works. However, the pests must be controlled on the trap crop or they will migrate back to the garden.

Cultural practices

Healthy seeds and transplants — Many disease-causing organisms are capable of living in and on vegetable seeds; therefore, it generally is unwise for a person to save seed produced in the home garden. Rather, it should be purchased from

dealers who have a reputation for producing or selling high quality, disease-free seed. When growing transplants, gardeners should use high quality seed and a pasteurized growth medium. If transplants or rootstocks are purchased from a supplier, they should be carefully checked for signs of pests and diseases, and any found to be infested should be destroyed.

Crop rotation — Moving the preferred host plant of an insect or disease organism hampers that pest's ability to feed and reproduce. Many disease-causing organisms do not survive long in the soil in which a different crop is planted. Exceptions include fungi that cause such diseases as fusarium wilt on cabbage, potato or tomato, and clubroot of cruciferous crops; once the soil is infested with these pathogens, it can remain so for several years.

Many insects and mites hibernate or lay their overwintering eggs in the soil near their preferred hosts. Moving the garden to a different location, or even switching the host crops from one to another part of the garden, has the effect that, upon emergence in the spring, the pest may find a non-host plant on which it is unable to feed and reproduce. This practice also may help to control weeds, especially if part of the garden is summerfallowed for at least one growing season. Crop rotation has the added advantage of allowing the soil to rejuvenate itself. Cabbage, turnip and potato use large amounts of nitrogen and should be preceded with legumes, for example, pea or bean, followed by fallow and the incorporation of compost or manure.

It is advisable to rotate among vegetable families because different members of a plant family often are susceptible to many of the same pest problems. For example, tomato, potato, eggplant and pepper are in the potato family and share many common diseases and insect pests, as do members of the cucurbit family (cucumber, melon and squash) and the crucifer family (cabbage, broccoli, Brussels sprouts, cauliflower, kohlrabi, turnip and radish). It is desirable to leave as much time as possible between related crops; three to six years is ideal. If rotation is not practical, gardeners should at least alternate the cultivars of vegetables that they are growing, choosing pest-resistant ones whenever possible.

Manual and mechanical methods — Hand-picking large, slow-moving pests, such as caterpillars, Colorado potato beetles and slugs, and dropping them into a container of soapy water or a *5%* isopropyl (rubbing) alcohol solution is effective. Shaking the plants or hosing them off with a spray of water dislodges some insects and mites. Barriers are meant to keep a pest away from the crop that it may harm. Some examples are floating row covers, copper strips, abrasive materials and mulches. Used cans or frozen- juice containers opened at both ends and placed around transplants will protect them from subterranean cutworms. Copper strips repel slugs because their slimy coating interacts chemically with the copper. Applying bands of abrasive materials, such as diatomaceous earth, stone dust or crushed egg shells, around the perimeter or between rows in a garden helps to deter slugs and crawling insects. Diatomaceous earth is an insecticide made up of the ground shells of tiny sea creatures called diatoms. The silica daggers pierce the skin of the insect, causing dehydration. Soap solutions smother the pests to which they are applied. Tar paper collars placed on the soil around individual crucifer seedlings will prevent root maggot flies from depositing their eggs at the base of the plants.

Mulches provide an effective means of weed control in vegetable gardens. Organic materials, such as weed-free straw or grass clippings 7 to 10 cm thick, will inhibit germination of weed seeds brought to the surface by cultivation, retard development of weed seedlings, conserve moisture, and help to maintain a uniform soil temperature. After the crop is removed, the mulch can be incorporated into the soil to enhance its organic matter content. Plastic mulches increase the soil temperature and also conserve moisture. Black plastic mulches, with openings for the crop, will prevent weed growth, except at the opening. Clear and white plastic mulches, however, permit weed growth, as some light can penetrate through the plastic. Plastic mulches are best suited for warm-season vegetables. Mulches also may prevent soil from being splashed onto plants, thereby keeping the produce clean and reducing the risk of spreading some diseases. Weed mats made of woven fabric allow water and air to reach the soil but screen out light and act as a barrier to emerging weeds, thereby providing effective, long-term control.

Removal of infested plant material — Many fungal diseases can build up and spread rapidly through the production of millions of spores. Prompt removal or destruction of diseased plant residues retards the spread of disease-causing organisms. This is an effective practice with such diseases as gray mold, powdery mildew, and various leaf spots and fruit rots. Removing diseased plant material from the garden at the end of the season eliminates an important source of inoculum for the following spring. The remaining residues should be rototilled or spaded into the soil to destroy disease organisms and to expose overwintering pests to the elements and to predators. Composting diseased plant material is not recommended because it may not destroy all of the disease organisms, even if the compost heats properly and is turned frequently. In some cases, leaving the remains of insect-infested plants in the garden may help to increase the population of beneficial parasites.

Good growing practices — When watering vegetable gardens, allow the soil surface to dry before another watering. Avoid frequent, light waterings as they tend to promote disease development and favor the germination of weed seeds. Watering with soaker hoses or in-ground furrows may reduce disease incidence because the foliage stays dry. When using overhead sprinklers, gardeners should be sure that the water is applied during the late morning or early afternoon. This allows for rapid drying of the foliage. Prolonged leaf wetness favors the development of most foliar diseases. Wide row-spacing ensures rapid leaf drying and reduces the spread of certain pests and diseases by direct contact. However, wide spacing also reduces shading of the soil, thus enhancing the loss of soil moisture and favoring the growth of weeds.

Keeping plants as healthy as possible is an important strategy in managing insect and disease problems. Planting into a warm, moist, well-prepared, well-drained seedbed, maintaining a high organic matter content, applying fertilizers correctly, and not cultivating when either the plants or the soil is wet, are recommended practices. Monitoring the crop through the growing

season may help to keep pest problems small by facilitating prompt control. Accurate record keeping is suggested to ensure success from year to year.

Companion planting — Many insects prefer to feed on plants belonging to specific families and reject others. For example, the imported cabbageworm feeds on cabbage, radish, kohlrabi and other cole crops; therefore, interplanting unrelated plants, such as onion, with cole crops will help to deter feeding and limit damage by this pest. Furthermore, garlic, onion and other aromatic plants inter-planted among other garden crops are believed to repel certain insect pests. Alternating vegetable cultivars or species also reduces the chances of disease spread. Where root- knot and root-lesion nematodes are a problem, interplanting with French marigold or African marigold may help to reduce the populations of these pests (see Management of nematode pests, 3.12).

Altered planting times — Seeding dates of vegetables can be altered to allow the plants to grow at times when they are less likely to be injured by certain pests. This requires knowing the life cycles of the common pest species in the area. In some cases, it may be feasible to plant certain vegetables before or after the pests have passed through their most active feeding period. For example, in Ontario, onion maggot eggs are laid near onion plants in May, and maggots soon attack plants and may cause them to die. However, onion sets planted after June 1 will escape most first-generation maggots.

Resistant cultivars

Some vegetable cultivars are resistant or tolerant to certain pests and diseases, and growing resistant plants is an excellent way to decrease the risk of damage. Certain disease-causing fungi can survive for many years in the soil and may not be managed by routine cultural practices; therefore, planting resistant cultivars is the best means of controlling them. Packages of tomato seed marked VFN produce plants that are resistant to the *Verticillium* and *Fusarium* fungi that cause wilt diseases, and to root-knot nematodes that may attack and cause galls on the roots. Other diseases best controlled by planting resistant cultivars include cabbage yellows, potato scab and cucumber mosaic. Certain vegetable cultivars also are less vulnerable to insect damage than others. For example, Red Pontiac potato is more resistant than other potato cultivars to tuber flea beetle, and Champion radish seems to be somewhat resistant to the crucifer flea beetle.

Biological control

Beneficial insects can be attracted to the garden by planting some of their favorite nectar- and pollen-producing plants in the garden or nearby. Members of the carrot (dill, caraway, fennel and parsley), mint (catnip, hyssop and lemon balm), and daisy (yarrow and coneflowers) families are known to attract such insects. Providing a source of water and shelter also helps to keep beneficial insects around. Birds and toads also aid in the control of insect pests. Care must be taken when applying pesticides because beneficial insects can be killed as well as the target pest.

The bacterium *Bacillus thuringiensis,* or Bt, is a pathogen of certain insects and must be ingested by the larva of the insect to be effective. Only butterfly and moth larvae are susceptible to this product, although a strain of Bt has recently been developed that will kill the Colorado potato beetle. Various formulations of this microbial pestcontrol product are available for controlling larvae of certain moths and butterflies on home garden vegetables. This and other treatments must be applied when the pest is most susceptible.

Chemical control

Seed treatment — Planting fungicide- or hot-water-treated seed helps to ensure good stands and may avoid the need to replant. Seed treatments kill disease- causing organisms on the seed and help to protect the vulnerable seed and young seedlings from certain soil-borne disease organisms. Treated seed is available to the home gardener and is marked “treated” on the package. Fungicide-treated seed is usually red or some other easily identifiable color. If untreated seed is used, it should be certified disease-free or be hot-water treated (see Crucifers, black rot, 8.2). Seed can be treated by the gardener, using recommended chemicals according to the manufacturer’s directions. Small packets of seed can be treated by tearing off one comer and putting about twice as much chemical in the packet as can be picked up on the first centimetre of the flat end of a toothpick. The packet should be shaken until the seed is thinly coated.

Foliar treatment — Most foliar diseases can be controlled by spraying or dusting plants with an effective fungicide as a preventative treatment. Protectant fungicides work on the plant surface to protect against infection, but they cannot cure established infections. If a considerable amount of disease is present, it is usually too late to apply a fungicide treatment, except to protect newly emerging leaves. Fungicides should be applied at 7- to 10-day intervals, or as directed by the manufacturer, with reapplication after rain or watering has washed the material away. A thorough covering of the plants is necessary for proper disease prevention. Early detection, timely fungicide applications and removal of diseased leaves are necessary for effective control of many foliar fungal diseases. Gardeners can choose from a wide variety of organic and inorganic fungicides for use in combatting vegetable diseases in home gardens.

The home gardener also has a number of insecticides and miticides to choose from. Most botanical insecticides are derived from plant parts and will break down quickly into harmless substances. Botanical insecticides include pyrethrin and rotenone. Insecticidal soaps are effective against aphids, but they also are non-selective and may upset the natural balance of beneficial insects and their prey.

Chemical control of weeds usually is not practical for small home gardens but can be employed for larger gardens and for market gardens. If the decision is made to use a herbicide, extreme care should be taken to avoid accidental drift onto non-target species. In addition, residual soil herbicides should not be used where residues may carry over and affect succeeding crops.

Growers who apply pesticides in their home gardens should use only products that are registered and recommended for the crops being grown and for the specific diseases or pests that are prevalent in the area. It is very important to carefully follow the manufacturer's directions to ensure maximum effectiveness of a pesticide and to minimize potential adverse effects, such as poor control, crop injury and unacceptable chemical residues on the edible produce. Spot treatment is preferable to a general application because it lessens the risk of harming beneficial organisms, humans and pets.

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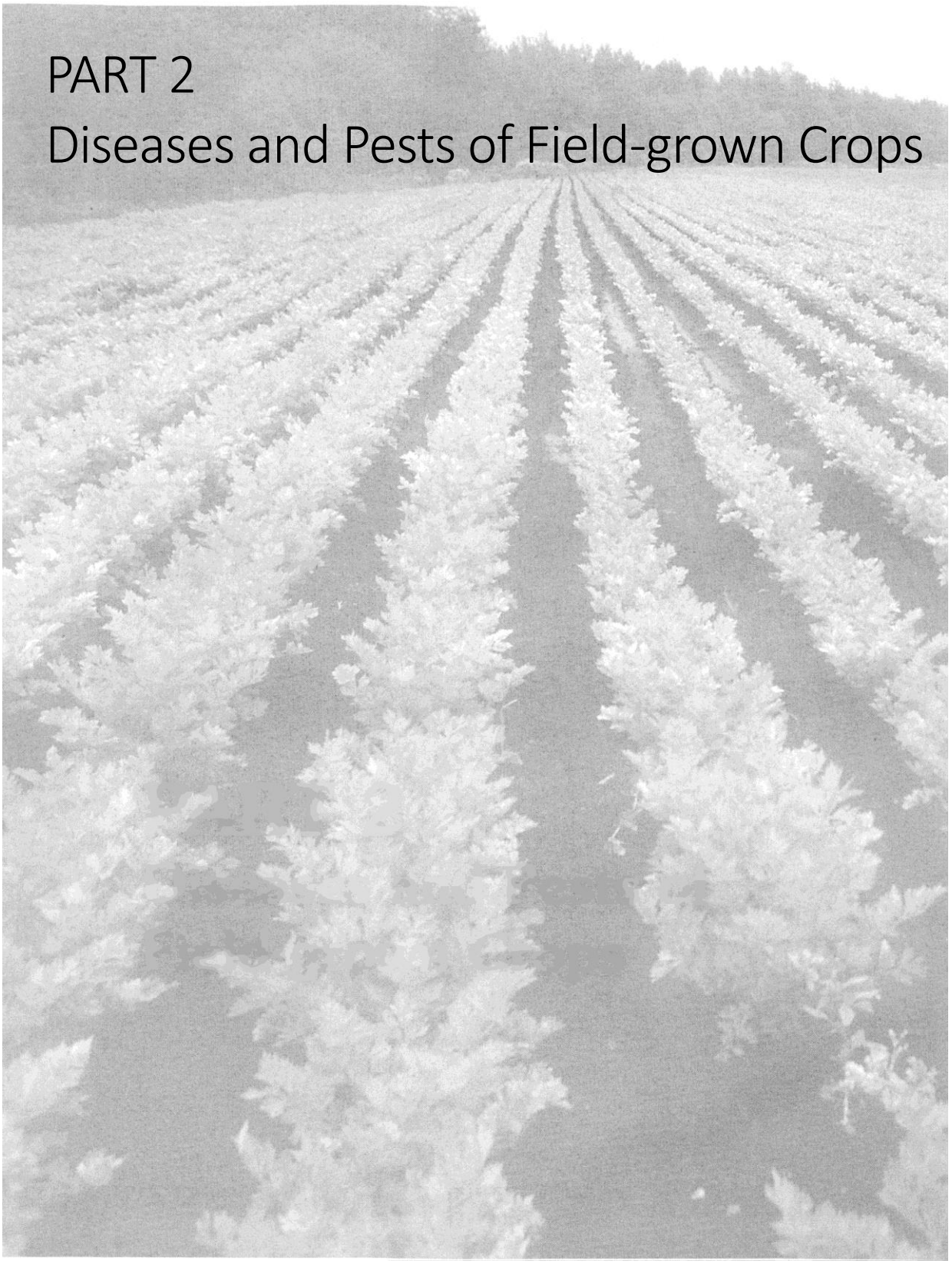
(Original by R.J. Howard, S.J. Barkley and A.M. Pucat)

PART 2
Diseases and Pests of Field-grown Crops



Locating Text Sections and Figures

Text sections are numbered consecutively within each chapter. For example, section 16.2 describes bacterial soft rot, the second topic of Chapter 16, Potato. To find a text section, refer to the running heads, which carry the inclusive section numbers for each two-page spread.

Color illustrations, grouped near the back of the book, appear in the same order and have the same number as the corresponding text section; for example, figures *16.2a* and *16.2b* illustrate the symptoms of bacterial soft rot of potato. Line drawings, halftones and tables are numbered similarly, except that a text figure number contains the letter T; for example, Figure *16.2T1* illustrates the disease cycle of bacterial soft rot of potato.

4 Asparagus

FUNGAL DISEASES

▶ 4.1 Botrytis blight (gray mold) *Figs. 4.1a,b*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Botrytis blight occurs during periods of moderately warm, humid weather. Disease severity is increased by prolonged periods of leaf wetness, especially where plant growth is dense and air movement is limited. The fungus infects senescing flowers or injured ferns and can cause extensive blighting, especially in the lower canopy *(4.1a).* The lesions that develop are tan with dark brown borders *(4.1b)* and have an extended yellow halo. Sporulation of the fungus occurs at high humidity. Newly emerged spears may be completely blighted, turning brown to black. (For a description of the causal agent, see Lettuce, gray mold, 11.10.)

Management

Cultural practices — Destruction of infested crop residues by plowing or other means will reduce inoculum levels. Overhead irrigation may contribute to an increase in botrytis blight.

Chemical control — Recommendations have been developed in most areas where these diseases are a problem.

(Original by F.J. Louws)

▶ 4.2 Fusarium crown and root rot *Figs. 4.2a-e*

Fusarium oxysporum f. sp. *asparagi* S.I. Cohen
Fusarium moniliforme J. Sheld.
(teleomorph *Gibberella fujikuroi* (Sawada) Ito *in* Ito & K. Kimura)

Fusarium crown and root rot may reduce the productivity of asparagus plantations to uneconomic levels within five to six years of planting. Similarly, this disease can reduce plant stands by up to 50% within a single season when infested fields are replanted. The fusarium crown and root rot complex occurs in all major asparagus-growing areas and is the most economically important disease on this crop.

Fusarium moniliforme is non-host-specific and has been associated with diseases in over 32 plant families, including many vegetable crops. However, only in sweet corn does it appear to be a primary pathogen, causing seedling blight, bud, ear, root and stalk rots (see Maize, 12.8). *Fusarium oxysporum* f. sp. *asparagi* is unique to asparagus.

Symptoms Dead and dying plants may be scattered throughout plantations, often occurring in low-lying areas or on steep sandy slopes. One, two or more shoots per crown appear stunted, turn yellow, and may wilt and die *(4.2a,b).* The shoots progressively decrease in size and number with time, eventually making harvest uneconomical. When cut in cross-section, the vascular bundles within the stem sometimes appear discolored *(4.2c).* Reddish- brown elliptical lesions are often seen at the base of the stem, sometimes girdling it and causing a cortical decay. The cortex of diseased roots may be completely destroyed, leaving a persistent, hollow root hypodermis. Brown elliptical lesions are often found at sites of lateral root emergence *(4.2d).* An extensive, dry-brown rot is observed in the crown of diseased plants *(4.2e).*

Pre- and post-emergent blights are associated with seedlings in replanted fields. Emerging seedlings may be stunted and yellow, or wilted. Wilting is associated with the complete collapse of the primary root. Less severely affected roots often possess sunken reddish-brown lesions at emergence sites of the lateral roots and at the internodes.

Causal agent The primary biotic agents associated with decline and replant problems in asparagus are *Fusarium oxysporum* f. sp. *asparagi*, the causal agent of wilt and root rot disease, and *F. moniliforme*, the cause of stem and crown rot.

Fusarium oxysporum f. sp. *asparagi* is highly variable in cultural morphology, pigmentation, conidial septation, and presence or absence of certain spore types. Microconidia, macroconidia and chlamydospores may all be produced. Kidney-shaped microconidia are single-celled and occur on short phialides. Macroconidia, produced on branched conidiophores on sporodochia, are slightly sickle-shaped and have a foot-shaped basal cell and a pointed apical cell. Intercalary or terminal chlamydospores are formed singly or in chains on the hyphae.

Fusarium moniliforme produces microconidia and macroconidia, but not chlamydospores. The microconidia are oval to club-shaped and are formed in long chains or false heads from branched or unbranched monophialides. Macroconidia are three- to four-septate, thin-walled, sickle-shaped to nearly straight, with a sharply curved apical cell and a footshaped basal cell. This pathogen is best diagnosed by culturing singlespore isolates on water agar amended with potassium chloride (8 g/L), which encourages the formation of chains of microconidia.

Fusarium oxysporum f. sp. *asparagi* and *F. moniliforme* are both relatively easy to isolate. The former can be cultured from discolored vascular tissue or from the reddish-brown root lesions. The latter can be isolated from lesions on the lower stem and crown, but rarely from root tissue. To make isolations, plant tissues should be surface-sterilized using standard techniques, placed on potato-dextrose agar, water agar or carnation-leaf agar, and incubated at room temperature. Saprophytic forms of *F. oxysporum* are commonly isolated, so testing is required to confirm pathogenicity toward asparagus.

Disease cycle *Fusarium oxysporum* f. sp. *asparagi* is soil-borne. It persists in soil as chlamydospores and on infected symptomless hosts. This fungus can be a typical vascular parasite, but there is evidence that certain isolates also can produce cortical decay.

Fusarium moniliforme can be isolated from soil and is associated primarily with crop residues. It does not form chlamydospores and is not very persistent in soil. Thickened hyphae may be produced. Conidia are aerially dispersed. The primary location of infection is the lower stem at wound sites created by insects or harvesting. Weak plants are highly susceptible to infection, which proceeds into the crown of the plant. Crown symptoms may not appear until the plants come under stress.

Studies in the United States have shown that *Fusarium* populations in infested fields decline to a base level after five years out of production; however, they may persist for long periods thereafter. In addition, fields with no history of asparagus crops may harbor virulent isolates of both *F. oxysporum* f. sp. *asparagi* and *F. moniliforme.* Both fungi are consistently associated with asparagus seed. Conidia of these fungi may contaminate the seed or they may colonize it directly. Inoculum on the seed may cause damping-off or seedling blight.

Management

Cultural practices — New plantings should not be established where asparagus has been grown within the previous five years. Growers should use vigorous, one-year-old crowns and follow proper transplanting procedures. Environmental factors, such as drought, poor drainage or low pH, and cultural practices, such as harvesting before the crowns are established, over-harvesting, deep disc tillage, poor weed control, improper fertilization, and poor control of insects, rust or blight, may increase the severity of crown and root rot. Integrated control strategies should focus on procedures for reducing plant stress.

Resistant cultivars — No asparagus cultivars have resistance to either *F. oxysporum* f. sp. *asparagi* or *F. moniliforme.* However, new lines, such as Jersey Giant, and European cultivars, such as Limburgia, Lucullus, and Schwetzinger Meisterschus have considerable tolerance to fusarium crown and root rot.

Chemical control — Seed treatment and fumigation of seedling beds have successfully reduced the incidence of seedling blight. Fungicide crown dips offer only limited disease control. The use of fungicides for the long-term control of crown and root rot is of limited value.

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(Original by F.J. Louws)

▶ 4.3 Phomopsis blight (stem blight)

Phomopsis asparagi (Sacc.) Bubâk (syn. *Phoma asparagi* Sacc.)

Phomopsis blight is not an important disease in Canada. It causes elliptical lesions with gray centers surrounded by wide, red-brown margins. The lesions become depressed, and numerous, tiny, black pycnidia may appear within them.

Management

Cultural practices — Destruction of infested crop residues by plowing or other means will reduce inoculum levels. Overhead irrigation may contribute to an increase in phomopsis blight.

Chemical control — Recommendations have been developed in most areas where these diseases are a problem.

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(Original by F.J. Louws)

▶ 4.4 Phytophthora spear rot

Phytophthora megasperma f. sp. *glycinea* T. Kuan & D.C. Erwin
(syn. *Phytophthora megasperma* var. *sojae* A.A. Hildebrand)
Phytophthora cryptogea Pethybr. & Lafferty

Phytophthora spear rot has not been recorded in Canada, but the likelihood of it occurring is high. Asparagus crops in some areas of the United States have been damaged by this disease, especially after heavy and prolonged rainfall. Losses result from the failure of new plantings to become established and from reduced yields in more mature plantings.

Gray-beige to brown lesions occur on the spear slightly above or below soil level. The epidermal layer of the lesion is easily removed by rubbing to reveal the slimy cortical tissue underneath. As the spear rots, it may produce a bad odor as the result of secondary invasion by saprophytic bacteria. Under dry conditions, infected spears shrivel up.

Phytophthora megasperma f. sp. *glycinea* also causes root rot of crucifers and is a pathogen of several other crops, including alfalfa, lupine, soybean and sugarcane. Another species, *Phytophthora cryptogea*, also is pathogenic on asparagus but is not as common.

Management

Cultural practices — Destruction of infested crop residues by plowing or other means will reduce inoculum levels. Overhead irrigation may contribute to an increase in phomopsis and botrytis blights. To minimize the incidence of phytophthora spear rot, asparagus should not be planted in low-lying areas or on poorly drained soils.

Chemical control — Recommendations have been developed in most areas where this disease is a problem.

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(Original by F.J. Louws)

▶ 4.5 Purple spot (stemphylium leaf spot) *Figs. 4.5a-c*

Stemphylium vesicarium (Wahr.) E. Simmons

(teleomorph *Pleospora allii* (Rabenh.) Ces. & De Not.)

Purple spot was first reported in Washington State and Michigan in 1982, and it has since spread into central Canada. It has also been observed in the Fraser Valley of British Columbia, but it is not a problem where drier climatic conditions prevail, such as in the Okanagan Valley. Purple spot has not been observed in the Maritime Provinces. This disease can seriously reduce asparagus yields. During optimum conditions for disease development, up to 100% of fresh market spears in a given harvest may be rendered unmarketable. *Stemphylium vesicarium* has been recorded on a wide variety of herbaceous plants, including onion on which it causes stemphylium leaf blight.

Symptoms Infection occurs on the above-ground plant parts, including stems, branches and cladophylls. Spears may be rendered unmarketable by the presence of numerous elliptical lesions. The lesions are small, 1 to 2 mm in diameter, superficial, slightly sunken and purple *(4.5a).* Larger lesions are brown in the center with purple margins *(4.5b).* The lesions are often more prevalent on one side of the spear. On the fern, the fungus causes stemphylium leaf spot, a disease characterized by light brown lesions with dark purple margins that are 4 to 15 mm long. In cases of severe leaf spot, defoliation and dieback occur. Repeated defoliation leads to reduced yields.

Causal agent *Stemphylium vesicarium* produces golden- to olive-brown conidia that are oblong or broadly oval and have a length-to-width ratio of 1.5 to 2.7. Conidia have a verrucose surface, are 12 to 22 by 25 to 42 pm, multi- septate, with one or more, commonly three, lateral septal constrictions (4.5c).

Pseudothecia, 0.25 to 0.50 mm in diameter, have been observed on overwintering asparagus residue. Ascospores are muriform, darkly pigmented, and produced in bitunicate asci. The fungus can be isolated by surface-sterilizing freshly harvested tissue in 0.5% sodium hypochlorite for 15 minutes and incubating the excised purple lesions on potato-dextrose agar at 20 to 22°C.

Disease cycle *Stemphylium vesicarium* overwinters as pseudothecia on fern residue. Primary infections occur in early spring during cool, wet weather, at which time the ascospores are forcibly discharged and land on the windward side of emerging spears. The germinating spores most frequently penetrate stomatal openings or wounds, but direct penetration of the epidermis may also occur. Wounds on spears caused by blowing sand are important sites of infection. Infection can occur within three hours if wounds are present. Infections on sand-blasted spears are more numerous and occur after a shorter wetting period than those on non-wounded tissue. After penetration, the surrounding epidermal cells collapse to produce a sunken lesion. Once established on the spears, *S. vesicarium* will produce spores throughout the summer. Heavy infection of above-ground growth can cause severe defoliation.

Management

Cultural practices — Sanitation by removing or burying crop residue will help to limit primary infection. Rye or other suitable cover crops may reduce the potential for injury from blowing sand. The cover crop should be seeded in the fall, then killed the following spring with a herbicide.

Resistant cultivars — No sources of resistance have been reported in cultured asparagus varieties. Several ornamental and wild asparagus species have shown resistance, but these are usually genetically incompatible with the cultivated species.

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(Original by F.J. Louws)

▶ 4.6 Rust *Figs. 4.6a,b; 4.6T1*

Puccinia asparagi DC. *in* Lam. & DC.

Asparagus rust was first recorded in Europe in 1805 and first observed in North America in New Jersey in 1896. By 1902, it had spread throughout the asparagus-growing regions of the United States and Canada. Rust may cause economic loss during years with weather conditions favorable for disease development. In Canada, this disease is less prevalent in drier regions, such as the Okanagan Valley of British Columbia. The pathogen also causes infections on *Allium* species, such as onion and chives, but not garlic or leek.

Symptoms Asparagus spears are usually harvested before symptoms appear. Symptoms are first noticeable on the expanding shoots in early summer. Three distinct types of symptoms occur and are related to the stage of the disease. In the aecial stage, which occurs from April to July, slightly raised, light green, oval lesions, 10 to 20 mm in length, are formed *(4.6a).* These lesions

decrease in frequency from the base of the shoots upward and are inconspicuous. They turn cream-orange and become sunken in the center as they mature.

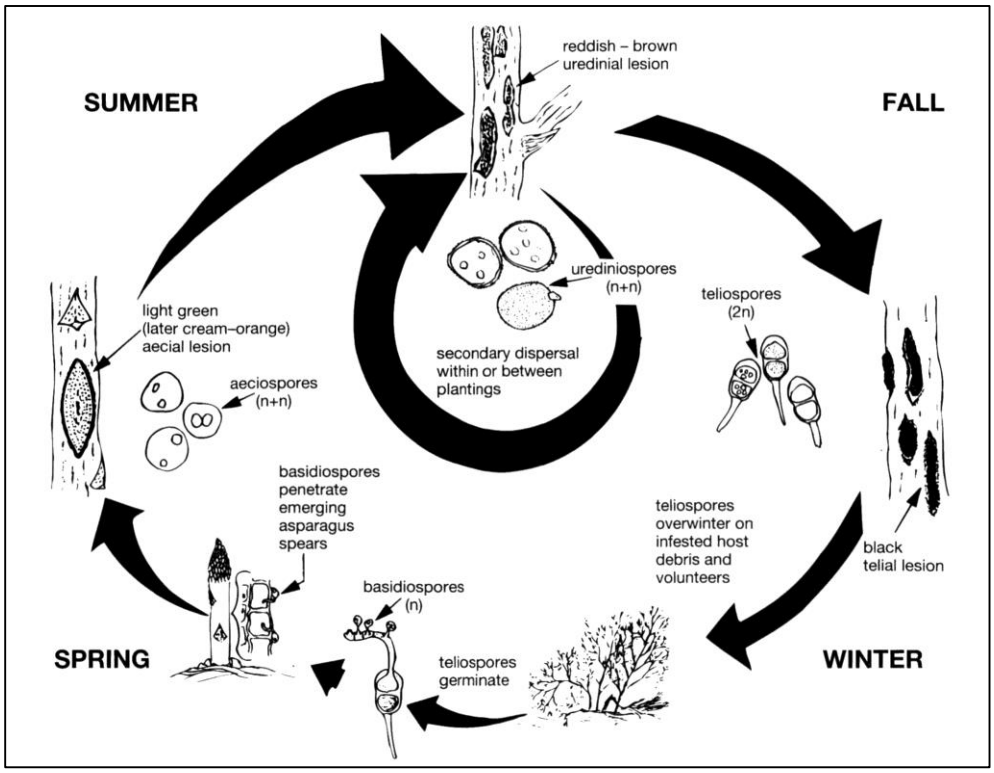
During the summer months, as early as June or as late as September, reddish-brown, blister-like pustules (uredinia) appear on the asparagus shoots *(4.6b).* When the uredinia mature, they release rust-colored urediniospores that cause new infections throughout the summer. Late in the season, the production of urediniospores is replaced by the formation of black teliospores. Teliospores and urediniospores may occur within the same lesion.

Severe rust infections stunt or kill young shoots. Infected foliage dries and falls prematurely, thus impairing the ability of the plant to store food reserves within the crown. The disease eventually reduces plant vigor and yield. Severely rusted plants are thought to be more susceptible to fusarium crown and root rot.

The light green oval lesions, tan blister spots, and black protruding blisters are diagnostic of rust in the early spring, mid summer and late summer to fall, respectively. Spores are easily obtained from mature fruiting bodies. The most useful spore form for diagnosis is the two-celled teliospore, which possesses thick, black cell walls and a pedicel up to twice the length of the spore body. Teliospores are slightly constricted at the septum and possess a thick wall and a rounded apex.

Causal agent *Puccinia asparagi* is an obligate parasite and has not yet been cultured *in vitro*. Pathogenic specialization into races has yet to be documented. Certain previously tolerant cultivars have come to be highly susceptible, suggesting that new virulent strains of the pathogen have developed. *Puccinia asparagi* is pathogenic to most dioecious species of asparagus, and partially or non-pathogenic on the perfect-flowering species.

Aecia of *P. asparagi* are caulicolous, occur in groups or are scattered, and are cupulate or short-cylindric. Aeciospores are light orange-yellow, globose, 15 to 21 by 18 to 27 µm, and have nearly colorless walls that are 1 pm thick and finely verrucose. Uredinia are caulicolous and cinnamon- brown. Urediniospores are single-celled, red-brown, globoid or ellipsoid, 19 to 30 by 18 to 25 mm, have a golden yellow wall that is 1.5 to 2 µm thick and minutely echinulate. These spores possess pores, usually four, which are equatorial. Telia are caulicolous and blackish-brown. Teliospores are 30 to 50 by 19 to 26 µm, rounded above, slightly constricted at the septum, with a chestnut-brown wall, 2 to 3 µm thick at the sides, to 10 µm above, and with pedicels somewhat colored and one half to twice the length of the spores.



4.6T1 Rust; disease cycle of *Puccinia asparagi.*

Disease cycle *Puccinia asparagi* is a macrocylic, autoecious rust (*4.6T1*). The five spore stages (spermatia, aecio-, uredinio-, telio- and basidiospores) all occur on asparagus. There are no alternate hosts. The fungus overwinters on infested host residue in the form of teliospores. These germinate early in the spring to produce four monokaryotic basidiospores, which can cause new infections in emerging asparagus spears. Basidiospore infection results in the production of spermagonia on the host. Haploid spermatia and receptive hyphae form within the spermagonia. Matings between compatible spermatia and receptive hyphae

eventually result in the production of dikaryotic aecia. Spermatia and aeciospores occur together in the light green, oval lesions, with the centrally located spermatia being surrounded by concentric rings of aeciospores. Spermagonia and aecia appear 6 to 29 days after the initial infection, depending on the temperature (optimum 25 to 30°C). The aeciospores mature, disperse by wind and, in the presence of water, cause new infections that result in the production of uredinia and uredin- iospores. If weather conditions are suitable, multiple cycles of urediniospore infection may occur during the growing season. These spores are also wind-borne. Germination of the urediniospores may occur within a wetting period of one hour. Successful penetration may occur within three hours, but a wetting period of nine hours at 15°C is optimal for infection. Uredinia become visible within six or more days. As the primary uredinia mature, secondary and tertiary uredinia originating from the same initial infection focus, often form in a concentric pattern. Aeciospores and uredinospores are responsible for major epidemics.

Teliospores are formed at low temperatures or during periods of dry weather and serve as the source of primary inoculum (basidiospores) for the following season. They are two-celled and possess thick, melanized cell walls. Basidiospores are ephemeral and produce the first localized infections of the year.

Management

Cultural practices — Measures that aid in disease prevention include removal of infested crop residues to minimize the amount of primary inoculum, destruction of wild or volunteer asparagus within 300 m of commercial plantings, and locating new plantings or nurseries away from established plantings. Fields should be clean-cut after harvest and spears should be cut below soil level to avoid infection of the stubs by rust spores. Practices that promote the rapid drying of plant surfaces, such as planting rows in the direction of the prevailing wind, may help to limit infection.

Resistant cultivars — Growers should use asparagus cultivars such as Jersey Centennial that possess rust tolerance. Tolerance is expressed as a low disease incidence called slow rusting. This situation results from a long latent period before symptom development and from fewer uredinia developing on the stems.

Chemical control — Where rust is a recurring problem, growers should spray with a registered fungicide as soon as harvesting has been completed but before the disease normally appears. A 7- to 10-day spray schedule may be required to adequately protect the fern growth. The use of drop nozzles with a high-boy type sprayer is recommended to achieve uniform coverage.

Selected references

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(Original by F.J. Louws)

VIRAL DISEASES

▶ 4.7 Miscellaneous viral diseases

Asparagus virus I
Asparagus virus II
Tobacco streak virus

Several viral diseases of asparagus occur in North America. Asparagus virus I, which has yet to be detected in Quebec or British Columbia, has been recorded infrequently in Washington and Michigan. Asparagus virus II, or asparagus latent virus, has been positively identified and is prevalent in most commercial plantings and seed beds. Generally, the older the planting, the more prevalent is asparagus virus II. A third virus, tobacco streak, occurs naturally on the west coast of Canada, but its importance in asparagus culture is not known.

There are no known alternative hosts in the field that are an important source of inoculum. However, asparagus virus I can produce necrotic lesions on *Chenopodium quinoa* Willd., C. *album* L., C. *capitatum* (L.) Asch. and C. *ama- ranticolor* Coste & Reynier. Asparagus virus II, in the laboratory, can infect *Cucumis sativus* L., *Beta vulgaris* L., *Cucurbita pepo* L., and *Phaseolus vulgaris* L., among a wide range of other hosts.

Symptoms No distinctive disease symptoms occur in plants infected with either asparagus virus I or II under field conditions, but infection by either virus may lead to a reduction in plant height, productivity and vigor. Asparagus virus I is considered to be more virulent than asparagus virus II. When plants are simultaneously infected by both viruses, a severe decline and death can occur. Infection with asparagus virus II has been correlated with an increase in the incidence of fusarium crown and root rot. Tobacco streak virus has been reported to cause stunting and decline of asparagus in Europe, but the symptoms associated with infection have not yet been documented in Canada.

Causal agents Asparagus virus I is a member of the potyvirus group of viruses and has flexuous, rod-shaped particles 700 to 880 nm in length. Asparagus virus II is a member of the ilarvirus group with quasi-isometric particles 26 to 36 nm in diameter. Tobacco streak virus is also a member of the ilarvirus group, with isometric particles 28 nm in diameter.

Verification of virus infection is accomplished with the use of indicator plants and serological techniques. Asparagus virus I causes local necrotic lesions, typically 2 to 3 mm long, on *Chenopodium quinoa*. The reaction is non-systemic. On the same host, asparagus virus II induces local, diffuse chlorotic spots that grow to 5 mm in size, followed by systemic symptoms of mottling, mosaic or necrosis. Tobacco streak virus causes a systemic necrosis similar to asparagus virus II on *C. quinoa*, so these two viruses are best differentiated through serological procedures.

Disease cycle Asparagus virus I is transmitted from plant to plant by aphids, notably the green peach aphid, and is not seed-borne. The asparagus aphid (see 4.9) does not transmit the virus. Asparagus virus II has no known natural vector, but it can be transmitted in seed, with up to 60% transmission reported. The virus may be transmitted from male plants to the seed by pollen. Mechanical transmission by cutting spears may provide a further means of spread of asparagus virus II. Infected seed beds have played an important role in the dissemination of asparagus virus II throughout asparagus-growing regions. Tobacco streak virus may be transmitted by western flower thrips, onion thrips and infested seed.

Management

Cultural practices — Asparagus virus I, asparagus virus II and tobacco streak virus can be eliminated from planting stock through shoot-tip tissue culture techniques. Once virus-free parental stock is established, the spread of asparagus virus II can be limited. In contrast, asparagus virus I spreads rapidly once introduced in a field, making it difficult to control. Nevertheless, losses from infection by asparagus virus I are minimal in the absence of asparagus virus II.

Selected references

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(Original by F.J. Louws)

NON-INFECTIOUS DISEASES

▶ 4.8 Miscellaneous disorders *Fig. 4.8*

Autotoxicity
Cold injury

Autotoxicity Allelopathic chemicals (see Beneficial plants, 3.6) are associated with asparagus residues and can damage subsequent plantings. They cause stunting and may increase susceptibility to *Fusarium* infection. Cultural practices influence the persistence of allelopathic compounds. If crop residues are incorporated into the soil each year, they will dissipate faster than under a minimum tillage regime. An interval of three to four years is recommended before planting asparagus on sites where this crop has been grown previously.

(Original by F.J. Louws)

Cold injury Curvature and purpling of emerging asparagus spears *(4.8)* often results from the effects of cold winds in early spring. Affected spears bend towards the direction of prevailing winds because of reduced cell growth on the exposed side. To minimize this problem, asparagus stands should be sheltered from the wind.

(Original by P.R. Ragan)

INSECT PESTS

▶ 4.9 Asparagus aphid *Fig. 4.9*

Brachycorynella asparagi (Mordvilko)

The asparagus aphid is native to Europe. It was first found in North America in New York in 1969 and is now present in most northwestern and northeastern states. It has been a problem since 1980 in the Okanagan and Similkameen valleys of British Columbia, and it has been reported in southern Ontario.

This aphid is reported to be specific to cultivated asparagus but it has also been reared on ornamental Sprenger asparagus, *Asparagus densiflorus* (Kunth) Jessop.

Damage Feeding causes stunting of the leaf-like, flattened branches (cladophylls) and shortening of the internodes near the feeding site, resulting in rosetted growth at the branch tips and a characteristic blue-green color. Heavily infested plants are weak, show a marked reduction in yield, and may die within two years. Growth abnormalities likely result from a toxin injected into the plant by the aphid during feeding.

Identification The asparagus aphid (this and other aphids discussed in this book belong to the family Aphididae) is long, narrow, green, has very short antennae, and is covered with a mealy gray wax (4.9). The tip of the abdomen (cauda) is moderately long and almost parallel-sided. The paired, abdominal projections (cornicles) are small and mammiform. No other aphid species on asparagus has these characteristics.

Life history The asparagus aphid spends its entire life on asparagus. It overwinters in the egg stage on asparagus foliage. Eggs hatch in late March and early April to become wingless "stem mothers" that produce female nymphs without mating (parthenogenesis). Winged and wingless females are produced throughout the spring and summer. In September, sexual forms are produced and overwintering eggs are laid on asparagus ferns, completing the life cycle. Winged adults disperse during the spring and summer months. The asparagus aphid can also be spread on asparagus spears that are harvested and transported to uninfested areas.

Management Good aphid control in the fall will reduce egg laying and, if accompanied by the prescribed cultural practices, will reduce the following year's aphid population.

Monitoring — Sampling should be done once per week after harvest, when the asparagus has leafed out. At least 150 samples should be taken uniformly throughout the field, using lateral branches from the lower region of the plant stem. Aphids are extracted by placing the branches in a Berlese-Tullgren funnel or other extraction container. Methyl iso-butyl ketone, added during extraction, causes the aphids to withdraw their feeding stylets and drop. Aphid numbers per gram of tissue can then be determined. As few as one asparagus aphid per 10 grams of asparagus fern can cause serious plant damage. Growers should look for aphid damage by walking systematically through immature, pre-harvest fields weekly after seeding or transplanting. Productive fields should be monitored weekly after the harvest period, especially if damage was observed the previous year. Sampling and visual observations should be resumed three to four weeks after spraying with a systemic insecticide, or one week after spraying with a non-systemic insecticide.

Cultural practices — Destruction of asparagus ferns in the fall by mowing will greatly reduce overwintering aphid populations. Spring tillage also is effective and, when combined with fall mowing, gives excellent control.

Chemical control — The timed application of specific insecticides to foliage will adequately control this aphid. Certain systemic insecticides are more effective and longer- lasting than non-systemic materials, and one or two sprays usually provide good seasonal control. Because systemics are applied fewer times than contact insecticides, their potential impact on beneficial insects, such as bees, also is reduced. No resistance to insecticides registered for aphid control on asparagus has been reported in British Columbia.

When aphids exceed the threshold of one aphid per 10 grams of asparagus, or if damage is observed, a systemic insecticide should be applied immediately in the evening or early morning to achieve optimal chemical uptake and to minimize exposure to bees and other beneficial organisms. If problems recur, additional sprays may be required.

Selected references

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(Original by R.S. Vernon and A.R. Forbes)

▶ 4.10 Asparagus beetles *Figs. 4.10a-e*

Asparagus beetle *Crioceris asparagi* (L.)
Spotted asparagus beetle *Crioceris duodecimpunctata* (L.)

These beetles are important pests of asparagus across Canada. Beetle numbers fluctuate from year to year but serious outbreaks are localized. Feeding damage may render spears unmarketable. Asparagus is the only known host.

Damage Adults of both species and larvae of the asparagus beetle reduce the vigor of asparagus by feeding on the fern *(4.10d)* in seedling beds and, after harvest, in established beds. Larvae of the spotted asparagus beetle feed almost entirely inside the berries and affect seed production.

Identification Both beetles (family Chrysomelidae) have adults that are about 6 mm long and similar in shape. Asparagus beetle adults *(4.10a)* are blue-black with four yellow patches with red margins on the forewings (elytra). The larva *(4.10b)* is dark green above and paler laterally with a shiny black head. The eggs *(4.10e)* are laid on end on any above-ground part of the host.

Spotted asparagus beetle adults *(4.10c,d)* are red above with twelve black dots on the elytra, six on each side. The larva varies from orange to yellow-brown. Eggs are laid on their side, mainly on the fern.

Life history Adults of both beetles overwinter in crop residue. They become active in early spring, feed, and lay eggs that hatch in one to two weeks. The larvae feed for three to four weeks, then drop to the ground and pupate at or just below the soil surface.

New adults emerge in late July. Their presence usually overlaps a second generation of larvae in August, which becomes adult in September and overwinters.

Management

Monitoring — During and after spear production is the time to detect the adult beetles, which drop when disturbed or move around the plant stem to conceal themselves.

Cultural practices — Growers should cut spears close to the ground, rogue any volunteer asparagus plants in and around asparagus beds, and remove crop residue and other refuse that may provide shelter for adults in winter.

Chemical control — Granular botanical insecticides may be spread in the row during harvest to control both species of beetle; afterward, recommended insecticidal treatments should be applied as for aphid control.

(Original by J.A. Garland)

▶ 4.11 Other insect pests *Figs.: see text*

Green peach aphid *Myzus persicae* (Sulzer)
Onion thrips *Thrips tabaci* Lindeman
Variegated cutworm *Peridroma saucia* (Hübner)

Green peach aphid The green peach aphid is widespread and polyphagous (see Potato, 16.41) *(16.41).* Though likely to be present, its impact on asparagus is usually negligible. Foliar treatments for the asparagus aphid also control the green peach aphid.

Onion thrips The onion thrips (see Onion, 13.27) *(22.35c)* overwinters in crop residue on the soil surface, moving onto asparagus as soon as the spears emerge. Asparagus is an important host for its populations to increase on, particularly in the spring.

Variegated cutworm The variegated cutworm *(18.35c)* feeds on the succulent tips of emerging asparagus spears. This, and other cutworms (see Tomato, 18.35) *(18.35)* are regular but not serious pests of asparagus.

(Original by R.S. Vernon and J.A. Garland)

ADDITIONAL REFERENCES

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Cooperman, C.J., and S.F. Jenkins. 1986. Conditions influencing growth and sporulation of *Cercospora asparagi* and cercospora blight development in asparagus. *Phytopathology* 76:617-622.

Bean see **15 Pea and bean**

5 Beet, chard, spinach

BACTERIAL DISEASES

▶ 5.1 Scab *Fig. 5.1*

Streptomyces scabies (Thaxt.) Waksman & Henrici
(syn. *Actinomyces scabies* (Thaxt.) Giissow)

Scab is occasionally seen on red beet, where it causes raised, corky growths on the surface of affected roots (5.7). Lesioning usually is greatest just below the soil line. This disease doesn't reduce crop yields, but scabby roots may be unmarketable. For more information on the causal agent, disease cycle and management of scab, see Potato, 16.5.

(Original by R.J. Howard and R.F. Cerkauskas)

FUNGAL DISEASES

▶ 5.2 Aphanomyces root rot (black root rot) *Fig. 5.2*

Aphanomyces cochlioides Drechs.

Aphanomyces root rot is one of the most serious diseases of cultivated beets, especially sugar beet. It is often found in association with other seedling diseases. It has been reported on sugar beet and table beet in Ontario, Quebec and Alberta. The pathogen also attacks Swiss chard, fodder beet and spinach. Many weeds can serve as alternative hosts.

Symptoms There are two phases of the disease, an early seedling blight and a chronic root rot. Symptoms of early seedling blight include poor stands, stunting, yellowing of foliage and falling over of seedlings. It generally starts on emerged seedlings at the soil surface or slightly below, although some pre-emergence damping-off may precede this stage. Initially, hypocotyls have water-soaked areas that are dark brown and later black. This discoloration may extend to the cotyledonary petioles and roots. The stems and hypocotyls dry and shrink to a thread-like condition and the seedlings may fall over. Oospores of the causal agent are present within the collapsed tissues. If dry soil conditions and low temperatures (10 to 12°C) prevail, seedlings may recover from seedling blight by developing new lateral roots, provided the damage is not severe.

The chronic root rot phase, known as taproot tip rot, is expressed in infected plants during the latter half of the growing season when tap roots encounter high moisture deep in the soil. Affected plants may be stunted, yellow and wilted, and have black-tipped tap roots (5.2) with an excess of lateral roots formed in response to destruction of the tap roots. Most of the lateral roots are black, necrotic and shrivelled. The root tips, after desiccation, have a fibrous appearance and will rot if the soil is wet.

Causal agent *Aphanomyces cochlioides* produces two types of zoospores in the asexual stage. Primary zoospores originate from zoosporangia, which consist of 3- to 4-µm long, branched hyphae. After differentiation, the zoospores are extruded from the tips of the zoosporangia. After one to three hours, each zoospore forms a cyst, 6 to 15 µm in diameter, which can then produce secondary zoospores, each measuring 13 µm long and 7 to 8 µm in diameter, with two flagella. These motile zoospores swim to the plants, germinate, and infect through stomata on the hypocotyl.

The sexual stage consists of subspherical terminal oogonia, 20 to 29 µm in diameter, with one to five antheridia wrapped about each oogonium. Hyaline to yellow oospores, 16 to 24 µm in diameter, nearly fill the oogonia.

Isolation of the fungus may be difficult since bacteria and saprophytic fungi are often present, particularly in advanced stages of root rot. Pieces of infected seedlings should be washed thoroughly and placed in petri plates containing 15 to 20 mL of sterile water at room temperature. *Aphanomyces cochlioides* is characterized by extensive formation of zoosporangia and encysted zoospores. An alternative method is to use a selective medium (see Selected references, Pfender et al. 1984) that permits growth of *Aphanomyces* while inhibiting or restricting growth of *Pythium, Rhizoctonia, Fusarium, Verticillium* and *Chalara* species. The appearance of *Aphanomyces* on the selective medium and on cornmeal agar is identical. The fungus forms a sparse, arachnoid, wandering growth on and within the medium, unlike Pythium isolates whose growth is unidirectional. Other differences between Aphanomyces and Pythium are large- diameter hyphae with granular cytoplasm, short side branches with a pointed apex, and main hyphae commonly branched in a Y-shaped junction in Aphanomyces.

On potato-dextrose agar, a whitish, aerial, non-septate mycelium, 3 to 10 pm in diameter, sparingly or moderately branched, is formed. After several weeks, the mycelial mat becomes thick and tough.

Disease cycle Disease development is more severe in warm (22 to 28°C), moist soils than in cool, dry soils. Time of seedling infection is also important. In general, older seedlings have lower levels of disease incidence and severity than young seedlings.

Aphanomyces cochlioides is a soil invader capable of surviving for several years, primarily as oospores in soil or diseased tissue. The mycelium and zoospores are incapable of prolonged survival. Weeds may be important in the persistence of the fungus. Crop residues also may increase the inoculum concentration in the soil, although the fungus has a low competitive saprophytic ability.

Dispersal of inoculum from field to field may be by infected host plants, wind-blown soil or host residues, and movement of surface water from infested to non-infested areas. Dissemination by tools, agricultural machinery and workers is also possible.

Management

Cultural practices — Crop rotations with corn, soybean and small grains should reduce the pathogen population in field soils. Other effective measures include improving soil drainage, subsoiling to promote aeration, maintaining good soil fertility, and controlling weeds.

Resistant cultivars — Sugar beet cultivars tolerant to aphanomyces root rot are available.

Chemical control — Partial control of seedling blight is possible with fungicide seed treatments. Rapidly germinating and vigorously growing seedlings may suffer less damage.

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(Original by R.F. Cerkauskas)

▶ 5.3 Cercospora leaf spot *Fig. 5.3*

Cercospora beticola Sacc.

This disease affects table beet, Swiss chard, sugar beet and spinach in all parts of Canada. Yield losses in sugar beet may be significant due to premature defoliation and difficulty in harvesting. Sugar beet and fodder beet suffer more damage from cercospora leaf spot than table beet, Swiss chard and spinach.

The pathogen has also been reported on species of *Amaranthus, Chenopodium, Malva* and *Polygonum* in the field in the United States but not in Canada. However, since extensive cross-inoculations with other *Cercospora* species and other plants have not been done (see Causal agent), other weed hosts may exist.

Symptoms Large numbers of circular, 2- to 3-mm diameter, light tan to brown lesions with a distinct, dark brown to purplish halo are scattered over the leaf. As lesions mature, the centers become gray and brittle *(5.3),* often falling out and leaving a ragged hole. Lesions may increase in diameter if increasing temperature and moisture conditions follow infection. Lesions may coalesce, leading to chlorosis and death of outer leaves. The dead leaves often remain attached to the plant. Leaves at the center of the plant are often less severely affected. Stromata of the causal agent are often present in the necrotic centers of lesions. They are easily visible with the aid of a hand lens as small black dots. Lesions may also occur on petioles, flower bracts, seed pods and seeds. They are somewhat elongate on petioles.

Causal agent More than 2000 species of *Cercospora* have been described using the host as a criterion. Numerous cross-inoculations have shown that many of these species, including *C. beticola*, actually belong to the *C. apii* group. Variation in the size of the conidiophores and conidia in some of the many species described was shown to be induced by changes in environmental conditions, especially humidity. This is true for *C. beticola*, where a range in conidial length and width of 50 to 400 pm and 2 to 4 pm, respectively, has been reported. Generally, however, conidia are 36 to 107 µm long and 2 to 3 µm wide with 3 to 14 septa, straight to slightly curved, needle-shaped, hyaline, rounded at the point of attachment to the conidiophore, and tapered toward the apex. The conidiophores occur in groups or tufts, usually emerging through stomata, unbranched, olivaceous brown near the base and hyaline near the apex. Conidiophore growth is continuous, and conidiophores have small, thick conidial scars at the apex or where bends occur. The mycelium is dark, septate and grows intracellularly in the host, forming mycelial clusters within the leaf tissue. The fungus has no known perfect state.

Cercospora beticola sporulates on sugar beet-molasses agar when plates are incubated under alternating light and dark at 15 to 22°C. Sporulation is also possible if culture plates containing 1.5% water agar and autoclaved sugar beet leaves are incubated at high humidity.

Disease cycle Optimum conditions for disease development include long periods of 90 to 100% relative humidity with nighttime leaf-wetting and temperatures of 25 to 30°C. Penetration and disease development are higher under nighttime wetting and daytime drying than *vice versa.* Symptoms appear seven to eight days after infection under optimum conditions. Temperatures above or below these values result in slower disease development due, in part, to closure of stomata, through which penetration occurs with or without formation of appressoria. Under field conditions, fungal penetration will occur after a minimum of three to four nights with dew. High relative humidity with temperatures above 16°C are necessary for the formation of large clusters of stromata bearing conidia in the older, gray lesion centers.

High levels of disease may arise from only a few infected plants, since each lesion produces large numbers of conidia. Consequently, several cycles of infection and conidium production may arise under favorable environmental conditions. Dissemination of conidia is primarily by rain-splash, although wind, irrigation water, insects, equipment, and workers may also be responsible. Infected seeds, and weeds of the family Chenopodiaceae may be other sources of inoculum. *Cercospora beticola* can overwinter in residues from infected crops, in weed hosts, and in beet seed.

Management

Cultural practices — Sources of overwintering inoculum should be reduced by deep plowing of infected crop residues and a two- to three-year rotation with non-host crops, such as cereals.

Resistant cultivars — Sugar beet cultivars with resistance to C. *beticola* are available.

Chemical control — Seed of sugar and fodder beets should be treated with a fungicide to prevent seed decay.

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(Original by R.F. Cerkauskas)

▶ 5.4 Downy mildew *Figs. 5.4a,b*

Peronospora farinosa f. sp. *spinaciae* Byford
(syn. *Peronospora effusa* (Grev.) Tul.)
(syn. *Peronospora farinosa* (Fr.:Fr.) Fr.)

This disease has significantly damaged spinach crops in British Columbia in some years. It is a minor problem elsewhere. All cultivated *Beta* spp. are potential hosts of *P. farinosa* f. sp. *spinaciae.* Weeds of the family Chenopodiaceae, such as lamb's-quarters (*Chenopodium album* L.), are also hosts.

Symptoms Typical infection is systemic. The fungus attacks the foliage, causing pale yellow, small to large, irregular leaf spots on the upper surface *(5.4a),* with a corresponding grayish-purple mat on the lower leaf surface *(5.4b).* The lesions are not delimited by the leaf veins. Spores are produced on the lower surface under humid conditions. During wet weather, the diseased leaves become water-soaked, rapidly change color to a yellowish brown, and finally turn black as they rot. Although profuse sporulation generally occurs on the undersurface of infected leaves, the fungus may also sporulate on the upper surface under high humidity. Yellowing, stunting and death of plants are also possible. Infected overwintering plants may have creamy yellow leaves that are stunted and wrinkled or puckered. Leaves that are infected after they have matured turn yellow but are normal in shape.

Causal agent Four races of *Peronospora farinosa* f. sp. *spinaciae* have been reported in North America, with race 4 reported recently in California and Texas. All produce a non-septate mycelium that is intercellular in host tissue. Branched haustoria develop within the cells and resemble elongate, finger-like organs. Conidiophores grow determinately, arise through stomata singly or in fascicles, and are grayish purple when observed in large numbers. Branches arise at acute angles, and secondary branches are dichotomous. The spores are hyaline and generally 17 to 26 by 22 to 37 pm. They are borne at the tips of the branches and germinate by a germ tube.

Oogonia are subhyaline, spherical, 40 to 56 pm, with a thin wall. Antheridia are clavate and 8 to 14 by 20 to 30 pm in size. The oospores are yellowish brown, spherical, 36 to 43 pm in diameter, have thick wrinkled walls, and germinate by germ tubes. Two mating types are required for oospore formation.

Disease cycle *Peronospora farinosa* f. sp. *spinaciae* is an obligate parasite that requires a vigorously growing plant, free water and a temperature of about 9°C for optimum germination and penetration of leaf tissue. Minimum and maximum temperatures for spore germination are 2 and 30°C, respectively. A period of six to seven days at 70 to 90% relative humidity and 16 to 24°C is required from the time of infection until sporulation. The fungus does not require free water on the leaf surface for sporulation and will fruit over a wide temperature range if the relative humidity is 85% or greater. Spores, which are produced in large numbers, may lose viability rapidly when desiccated or exposed to sunlight. Oospores occur in young and old leaf tissues, and are formed in abundance when plants are exposed to stress during the latter half of the latent period.

Peronospora farinosa f. sp. *spinaciae* overwinters as mycelium in seed, as oospores mixed with seed or soil, and on infected spinach plants. Oospores in diseased plants are incorporated into the soil when they are plowed under. The oospores can survive one year in the soil, and at least two years in the seed. Dissemination of fungus spores is by wind, splashing water and infested seed.

Management

Cultural practices — Growers should practice a three-year rotation with non-host crops such as cereals, and plant in well-drained soil. Fall spinach crops should not be grown in or adjacent to fields where infected spring spinach crops have been grown. Infested seed should be hot-water treated for 25 minutes at 50°C.

Resistant cultivars — Resistant cultivars are available from seed suppliers.

Chemical control — Registered fungicides are available for foliar application.

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(Original by R.F. Cerkauskas)

▶ 5.5 Fusarium wilt *Figs. 5.5a-d*

Fusarium oxysporum f. sp. *spinaciae* (Sherb.) W.C. Snyder & H.N. Hans.

Fusarium wilt of spinach was first reported in the United States (Idaho) in 1920 and in Canada (Ontario) in 1932. It has also been reported in Alberta in a hydroponic greenhouse operation. The pathogen also attacks table and fodder beet, Swiss chard, and lychnis *(Silene* spp.).

Symptoms Infected plants may show disease symptoms from seedling through to mature stages (*5.5a-d*). Cotyledons of infected seedlings first turn dull-green, then wilt and shrivel. Seedlings may die within a day. Several stages of wilting may occur in older plants. Initially, only a few outer leaves wilt, but the disease can progress to younger leaves as well. Severely wilted leaves turn yellow and the younger leaves roll inward at the margin (5.5c). Stunted plants easily pull out of the ground. Light brown to almost black lesions of various sizes occur on the lateral and main roots (5.5a). As larger lesions extend into the pith or water-conducting vessels of the tap root, the entire stele turns black. Other fungi enter before the whole root rots and detaches from the

adjoining lateral roots. Several laterals may form above the rotted tissues, although the plant is weak and unthrifty. Clumps of soil may be attached to the rotted root by strands of white fungal mycelium. These symptoms may not appear on spinach until summer. The disease is suppressed in the fall as the temperature falls.

Symptoms of fusarium wilt may be mistaken for those caused by nematodes, nutritional imbalances, poor soil drainage, and adverse weather conditions.

Causal agent *Fusarium oxysporum* f. sp. *spinaciae* is a persistent soil inhabitant. There are two physiologic races: race 1 infects only spinach, whereas race 2 attacks spinach, beet, Swiss chard, and fodder beet. Only race 1 is known to be present in Canada, but both races occur in the United States.

Plating of the discolored vascular tissue on acid- or antibiotic-amended potato-dextrose agar will yield the fusarium wilt pathogen within a few days at 25°C. After a few weeks of growth on potato-dextrose agar at about pH 7 and at 25°C, the mycelium is floccose throughout the colony. Its color varies from white to pale-salmon or seashell-pink. Microconidia and macroconidia are abundant, hyaline, pedicellate and borne on phialides. Microconidia are single-celled, septate, 2 to 4.8 by 6 to 25 pm, and vary from oval to slightly curved. Macroconidia are curved, up to 6 pm in width and 44 pm in length, and generally many-septate. Smooth, thick-walled chlamydospores may be terminal, intercalary, intraconidial, isolated or in chains. They measure 6 to 18 pm in diameter and are mostly round.

Fusarium solani, Rhizoctonia solani and *Pythium ultimum* may also be recovered from diseased plants. These fungi do not produce true wilt symptoms but they cause damping-off, whereas the fusarium wilt pathogen does not.

Disease cycle The pathogen infects through uninjured roots and grows into the vascular tissue. A toxin is produced in advance of its growth, causing vascular discoloration and wilting of the foliage. Conidia are seldom formed in living tissue, but conidia and chlamydospores are produced abundantly as the tissue dies.

The disease is most severe at a soil temperature of 27°C but is completely inhibited below 16°C and above 33°C. The pathogen requires moist soil conditions for root infection. Once infection has occurred, the disease will progress quickly if the soil becomes dry and the plants become stressed. Alkaline soils do not favor disease development.

Once introduced to an area, the pathogen can survive saprophytically in the soil for a number of years. It can spread to other areas by wind-borne soil, surface drainage water, and soil adhering to implements and other agents. Long-distance spread is by infested seed.

Management

Cultural practices — If possible, seed should be obtained from areas where no wilt has been found. Once the disease has appeared in an area, the land should be rotated to non-susceptible crops, such as wheat, rye or barley, for three consecutive seasons before the next spinach crop. Spinach should be harvested as early in the season as possible and diseased residues should be destroyed promptly. Allowing spinach to stand in the field until it has gone to seed is an especially bad practice because it results in a rapid increase of the pathogen population in the soil. Growers should destroy volunteer spinach in the field between spinach crops and seasons. Before planting into wilt-infested soil, the addition of high rates of calcitic lime (ground limestone) at 2 tonnes per hectare may decrease disease severity.

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(Original by A.A. Reyes)

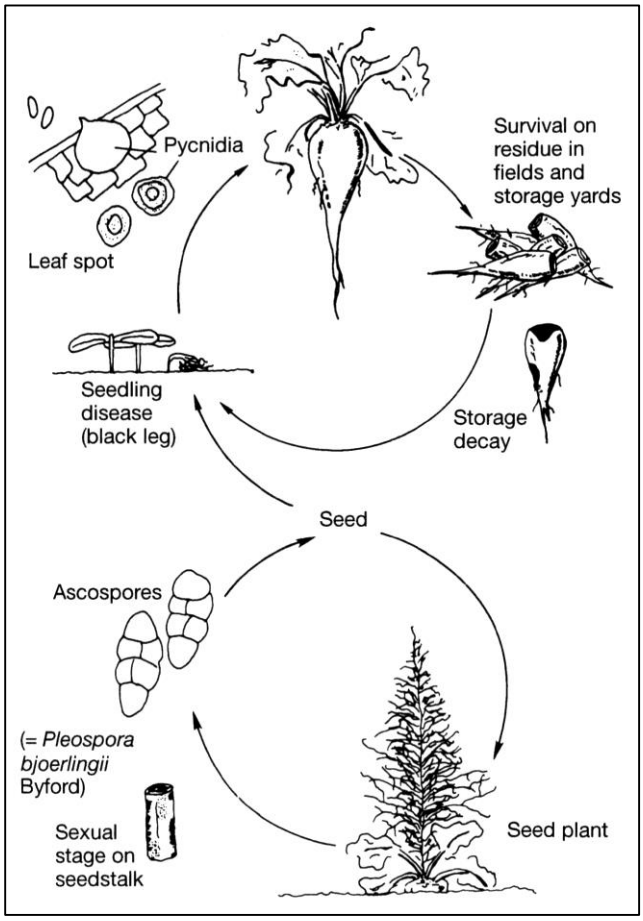
▶ 5.6 Phoma leaf spot and root rot *Fig. 5.6T1*

Phoma betae A.B. Frank
(teleomorph *Pleospora betae* (Berl.) Nevodovsky)
(syn. *Pleospora bjoerlingii* Byford)

Phoma betae has been reported at low levels from most provinces in Canada. It can infect beet and chard, causing seedling blight, leaf spot, root rot or storage rot.

Symptoms *Phoma betae* attacks all plant parts. Pre-emergence damping-off occurs with heavily infested seed. Some emerged seedlings may show brown discoloration in the hypocotyl after infection has occurred. Spots on leaves are up to 2 cm in diameter, brown, round to oval, with dark concentric rings near the perimeter. Small dark pycnidia are found throughout the spots in concentric rings. Older, lower leaves are generally more susceptible than younger leaves, and the leaf spot phase of the disease is generally less destructive than the root rot phase. Pycnidia are also found on seedstalks in dark necrotic streaks with grayish centers. Symptoms on roots begin near the crown as small, dark, sunken spots that become soft and water-soaked and finally turn dark brown to black with prominent black lines separating the diseased and healthy tissues. Older infected tissues become black,

dry, shrunken and somewhat spongy. Cavities lined with grayish-white mycelium may occur within these spongy tissues. Pycnidia are uncommon in root tissue. During storage, other fungi that cause rot of beets may also occur in tissues infected by *P. betae.*



5.6T1 Phoma leaf spot and root rot; disease cycle of *Phoma betae.* Reprinted by permission from E.S. Whitney and J.D. Duffus, eds., *Compendium of Beet Diseases and Insects.* © 1986. The American Phytopathological Society.

Causal agent *Phoma betae* produces elliptic, aseptate, hyaline conidia, usually containing two large guttules. The conidia escape as spore tendrils from dark brown, ostiolate, subglobose pycnidia immersed in infected plant tissues. The teleomorph has been reported in Britain and the United States but not in Canada. It forms pseudothecia containing asci with yellow-green, muriform ascospores.

Conidia and pycnidia vary widely in size due to environmental influences. On potato-dextrose agar, pycnidia range from 210 to 560 µm in diameter and conidia measure from 4.3 to 8.1 (mean 5.7) by 2.9 to 5.8 (mean 3.5) µm. On oatmeal agar, conidia are 6 to 9 by 3.5 to 4.5 µm and colonies are olivaceous-brown with felty aerial mycelium containing gray to white patches. Pycnidia are formed in areas lacking aerial mycelium. The reverse side of culture plates is greenish brown. The pycnidia have an outer wall of two to three layers of pseudoparenchymatous cells and an inner wall of one or two layers of thin hyaline cells.

Under near-ultraviolet light, incubation of surface-disinfested beet tissue or seed on potato-dextrose agar amended with 10 ppm benomyl before autoclaving stimulates pycnidial development of *P. betae* at the expense of mycelial growth, and eliminates other *Phoma* spp.

Disease cycle Infested seed is the primary means of long-distance dissemination, since the fungus may survive in seed for several years (*5.6T1*). Secondary, local spread is by wind- or rain-borne conidia, wind-blown plant residues, irrigation water and, to a lesser degree, insects. Conidium tendrils produced by the fruiting bodies may be washed or splashed to different parts of the plant or to neighboring plants by wind-driven rain. The fungus can survive in soil or in host residue for about two years. *Phoma betae* can inhabit the roots of lamb's-quarters (*Chenopodium album* L.), thus allowing it to survive a longer period of crop rotation. Systemic infection in beet plants that have survived seedling infection has also been reported. Plants under stress, such as from boron deficiency, excessively close topping or unfavorable environmental conditions, are more susceptible to infection by *P. betae.*

Prolonged wet periods during harvest will increase the amount of phoma decay during storage. Roots that have been frost damaged, desiccated, wounded as a consequence of topping, or grown in soils low in phosphate or nitrogen are more susceptible to phoma storage rot.

Disease development on foliage is favored by high humidity and temperatures of 15 to 32°C; on roots it is favored by temperatures of 5 to 20°C. Beet seedlings are readily attacked by the fungus at temperatures below 15°C. As temperature increases above 20°C, the attack on the seedlings decreases.

Management

Cultural practices — The use of clean seed will reduce the incidence of the disease. Other measures include planting after the soil is warm, and promoting vigorous plant growth by supplying sufficient boron and other nutrients. Also, a four-year crop rotation is important. Closetopping and harvest wounds should be avoided in roots that are to be stored. Creating an environment conducive to wound healing during storage is recommended. Corky tissue, which serves as a barrier to microbial invasion, develops in 10 to 14 days at 10°C and a relative humidity of 95% or greater, provided there is adequate air movement.

Chemical control — Seed treatment fungicides may reduce levels of seed-borne inoculum.

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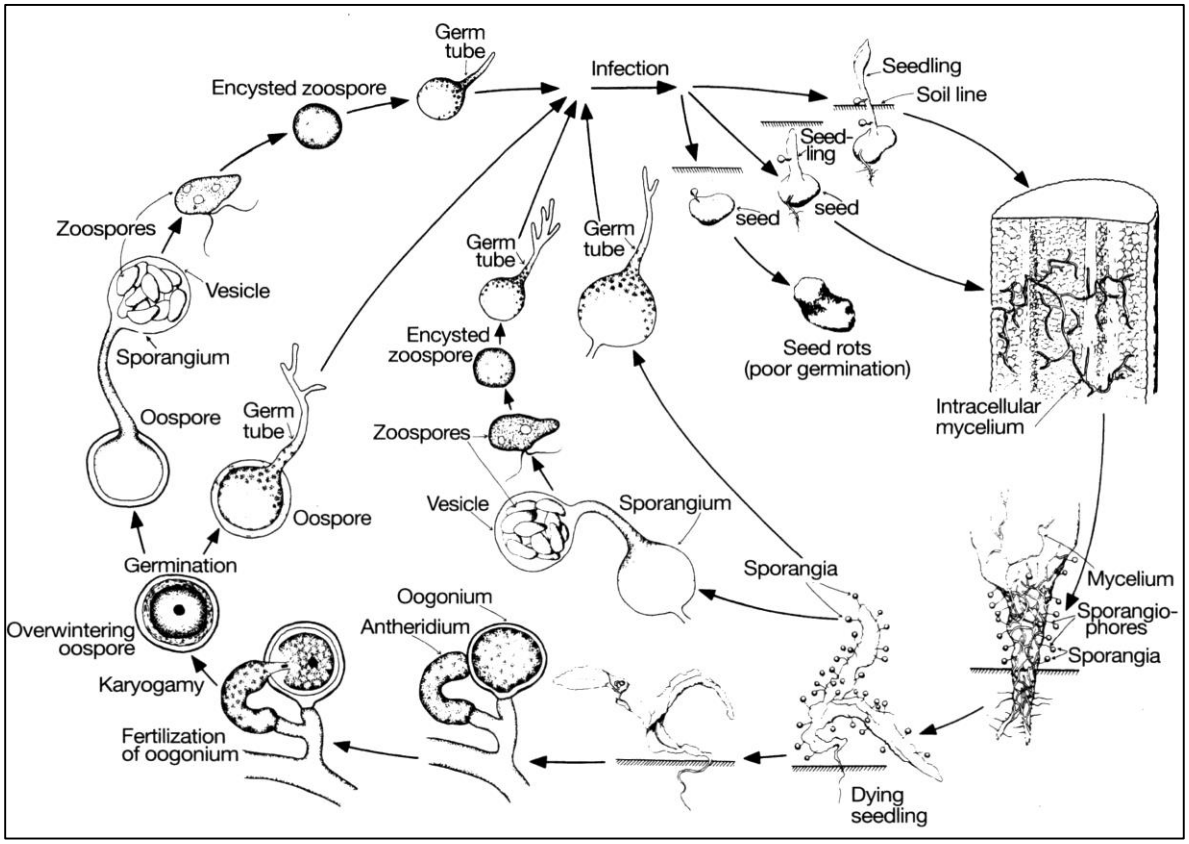
(Original by R.F. Cerkauskas)

▶ 5.7 Pythium root rot *Fig. 5.7T1*

Pythium aphanidermatum (Edson) Fitzp.
Pythium ultimum Trow



5.7T1 Pythium root rot and damping-off; disease cycle of *Pythium* spp. on vegetable crops. Reprinted by permission from G.N. Agrios, *Plant Pathology***.** © 1988 Academic Press.

This disease has been reported in all provinces where major spinach, table beet and sugar beet production occurs. Moderate and occasionally severe losses have been reported on beets. Both pathogens have wide host ranges.

Symptoms *Pythium ultimum* primarily causes a pre-emergence damping-off. If the soil is very moist, post-emergence damping-off may occur. The hypocotyl is most susceptible and may have a slightly dark, water-soaked spot just below the soil surface. If the vascular system is invaded, the affected tissue becomes black and the young seedling dies. If the vascular tissue is not affected, the seedling continues to grow, with a brown cortical lesion that collapses and turns black. Post-emergence damping-off is accompanied by a water-soaked appearance on seedling roots. *Pythium aphanidermatum* causes damage in warmer soils containing excessive moisture. Symptoms on lower leaves consist of wilting, yellowing, and a water-soaked black rot of the petiole bases. Badly infected plants usually die.

Causal agent *Pythium* species produce abundant, branched, non-septate mycelium. *Pythium ultimum* produces sporangia that are mostly terminal and spherical, 13 to 28 µm in diameter, and germinate only by germ tubes. The oogonia are smooth, spherical and mostly terminal, with a diameter of 19 to 23 µm. There are one to three sac-like antheridia per oogonium. The oospores are single, spherical, smooth and thick-walled, with a diameter of 14 to 18 µm. They do not fill the oogonium.

Sporangia of *P. aphanidermatum* are inflated, filamentous and branched or unbranched. Vesicles form from the sporangia and contain kidney-shaped zoospores that measure 7.5 by 12 µm. The oogonia are smooth, spherical and terminal, with a diameter of 22 to 27 µm. The oospores are single with a diameter of 17 to 19 µm, and they do not entirely fill the oogonium.

Colonies of both species produce a cottony aerial mycelium on corn- meal agar. If the infected seedling tissue is washed in running water to remove soil particles then placed in a film of water in a petri plate at 20°C for 24 to 48 hours, a characteristic vegetative mycelium and spores or fruiting bodies are often produced. Other techniques to produce characteristic growth stages for isolation of *Pythium* spp. employ special growth media or different kinds of substrates, such as apple.

Disease cycle *Pythium ultimum* is a common soil inhabitant and survives in cultivated soil as mycelium for one to two weeks, as sporangia for longer periods, or as oospores for many years (*5.7T1*). The fungus attacks the juvenile or succulent tissues of many hosts and can live parasitically on plant roots or saprophytically on organic material, depending upon whether conditions are favorable for the fungus or the host. Seed and root exudates may stimulate oospore germination. Soil temperature and high soil moisture are important factors in disease development. *Pythium ultimum* causes most severe pre-emergence damping-off at soil temperatures between 12 and 20°C, whereas *P. aphanidermatum* is most severe between 30 and 35°C.

Pythium spp. are often implicated in complexes with other fungi, such as *Rhizoctonia solani, Aphanomyces cochlioides, Phoma betae* and *Fusarium* spp.

Management

Cultural practices — Conditions that promote rapid and vigorous seedling growth will minimize damping-off. Effective measures include reducing soil moisture by ensuring adequate field drainage, planting in raised beds, improving soil fertility, and following a three- to four-year crop rotation with cereals.

Chemical control — Seed treatment with protectant fungicides can be effective. However, if different pathogenic fungi are present, a combination of two or more fungicides may be necessary.

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(Original by R.F. Cerkauskas)

▶ 5.8 Rhizoctonia root rot *Figs. 5.8a,b*

Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)
Rhizoctonia cerealis Van der Hoeven
(teleomorph *Ceratobasidium cereale* D. Murray & L.L. Burpee)

Seedling blight, damping-off, and root rot caused by *R. solani* have been reported from most provinces where beet and spinach are grown. The most frequent accounts involve table beet and sugar beet, in which major losses have been reported occasionally. The pathogen has a wide host range. *Rhizoctonia cerealis* may cause damping-off in early seeded sugar beet crops in cold soils.

Symptoms *Rhizoctonia solani* causes pre-emergence and, more frequently, post-emergence damping-off. Later in the season as the crop is maturing, significant root and crown rot is also possible. On seedlings, damage occurs at a slightly later stage than with *Pythium* spp. Infection occurs below the soil-line as dry, brown lesions with definite margins and may extend up to the hypocotyl. Seedlings that are not severely diseased may survive, although root and crown rot may appear later.

Symptoms of crown rot consist of sudden wilting and chlorosis of the foliage *(5.8a)* and the formation of black necrotic tissue on the outer petioles near the crown. This may be followed by extensive rotting of the crown and adjacent root tissue. Large areas of the rotted tissue may be covered with brown, felt-like mycelium.

Another type of rhizoctonia root rot called dry rot may appear on maturing roots *(5.8b).* Symptoms consist of circular brown lesions with concentric rings scattered over the root surface. The lesions develop into cavities filled with mycelium and are separated from healthy tissue by a sharp dark line.

Causal agent (see Bean, rhizoctonia root rot, 15B.7) *Rhizoctonia solani* has several anastomosis groups, of which AG-1, 2-2 and 4 are reported to be aggressive on seedlings in the United States. AG 2-2 is very pathogenic to the roots of older plants. *Rhizoctonia cerealis* also has been shown to cause damping-off in sugar beet at low temperatures and could be a threat to early planted crops in fields where intensive cereal production has occurred previously.

Disease cycle (see Bean, rhizoctonia root rot, 15B.7) Isolates causing dry rot are more active at low soil moisture levels and temperatures of 30 to 35°C, whereas isolates causing crown rot are active in heavy, poorly drained soil and at temperatures of 25 to 33°C.

Management

Cultural practices — Measures that promote good plant growth, including tillage, fertilization and adequate soil drainage, may reduce serious losses. In addition, good weed control, rotation with corn or small grains, and the use of bed planting to avoid hilling-up plants with infested soil are recommended. Rhizoctonia root rot is likely to be more severe following legumes than after corn or cereal crops.

Partial control of the seedling phase of the disease is possible by planting at soil temperatures below 15°C, although other fungi such as *Pythium ultimum* and *Phoma betae* may readily infect beet seedlings at these temperatures. Crown rot and dry rot are not readily controlled.

Chemical control — Growers should treat seed with a protectant fungicide.

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(Original by R.F. Cerkauskas)

▶ 5.9 White rust *Fig. 5.9*

Albugo occidentalis G.W. Wils.

This disease has been observed in most spinach production regions of Canada, although it is generally of minor significance. It has also been reported on weeds belonging to the genus *Chenopodium.*

Symptoms Symptoms are generally local and confined to leaves and petioles, rarely systemic in vegetative plants, and distortions of infected tissues are not present. Chlorotic areas are more pronounced on the upper leaf surface, while many small, 0.5 to 2 mm diameter, generally oval, white, shiny sori or blisters (5.9) appear on the lower surface. The sori contain large numbers of sporangia, which form under the leaf epidermis and rupture it when mature. The sori become more prevalent as the disease progresses and may cover the undersurface of the leaf, as well as the upper surface, petioles, branches, and seed coats. In later stages, the leaves turn brown and die and entire plants may be killed. Large numbers of oospores are produced within the old, infected foliage.

The presence of white sori or blisters containing many sporangia on the leaf undersurface and the ornamentation and diameter of the oospores serve to distinguish this fungus from *Peronospora farinosa,* which causes downy mildew (see 5.4).

Causal agent *Albugo occidentalis* is very closely related to *A. candida* (see Crucifers, white rust, 8.15), except that sporangia are 8 to 16 by 14 to 20 pm, and oospores are yellowish-brown, spherical, 50 to 60 pm in diameter with close, shallow reticulations on the surface.

Disease cycle *Albugo occidentalis* is an obligate parasite that attacks spinach as well as the weed strawberry blite *(Chenopodium capitatum* (L.) Asch.). The fungus overwinters as thick-walled oospores in infected foliage, and oospores on previous spinach crops may serve as primary inoculum for the subsequent crop. Also, the fungus may occur as a surface contaminant of the seed. Dispersal of the sporangia in the leaf sori is mainly by wind, but spread by rain and insects between neighboring plants may also occur.

Disease development is favored by clear and relatively warm, dry days, followed by cool nights with free moisture on the leaves. Water is necessary for sporangial germination and zoospore development. The optimum temperature for germination is 12°C, with sharp decreases occurring above or below this temperature; however, chilling the spores below 12°C for a short period will promote germination. Also, while disease and oospores develop more rapidly and abundantly in plants at 28°C than at 16°C, the production of sporangia is favored by low temperatures.

Management

Cultural practices — The destruction of diseased plant residues by deep plowing and a three-year crop rotation will reduce pathogen inoculum. Spinach crops should be planted some distance from where they were previously grown, and weeds such as strawberry blite should be eliminated.

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(Original by R.F. Cerkauskas)

VIRAL DISEASES

▶ 5.10 Spinach blight *Fig. 5.10*

Cucumber mosaic virus

Major losses resulting from cucumber mosaic virus were reported on spinach in the past; however, with the development of resistant cultivars this disease does not represent a major production problem. Cucumber mosaic virus infects a wider group of plants than any other virus. It is distributed worldwide, and infects vegetable crops such as cucumber, squash, melon, pepper, tomato, tobacco, eggplant, celery, beet, crucifers, sweet potato and parsnip, and ornamental flowers such as delphinium, gladiolus, lily, petunia and zinnia.

Symptoms The first recognizable symptoms on spinach are apparent on the young inner leaves, which exhibit a faint general chlorosis, later extending to one or more of the older leaves. New leaves become narrow, wrinkled, and mottled, often with a characteristic inward rolling of the leaf margin. Plants become stunted, with progressive yellowing and mottling (5.*10)* extending to the older outer leaves, which later become necrotic. Eventually, plants are reduced to a small cluster of yellow, malformed leaves and finally die. Spinach plants infected at the seedling stage are severely stunted. Dwarfing, yellowing, leaf deformation and death are such conspicuous symptoms that the disease can easily be recognized and not confused with other diseases. Symptoms produced by beet mosaic virus in spinach differ from those produced by cucumber mosaic in that fine, discrete, foliar spotting occurs early in the infection and the distortion of younger leaves is not marked. Severe malnutrition may produce some symptoms similar to those of cucumber mosaic virus infection.

Causal agent (see Greenhouse cucumber, cucumber mosaic, 22.20)

Disease cycle Cucumber mosaic virus overwinters in many biennial and perennial weeds and is usually carried to spinach plantings by aphids, such as the green peach aphid and the potato aphid (see Potato, 16.41, 16.42). Within plantings, cucumber mosaic virus is spread in a non-persistent manner by aphids; transmission efficiency varies with the species. The virus can be acquired by all aphid stages after only 10 to 15 seconds of feeding, but the ability to transmit is usually lost within two hours. The virus also may be transmitted, though less efficiently, by the spotted cucumber beetle and the striped cucumber beetle (see Cucurbits, 9.21). Cucumber mosaic virus is readily transmitted by plant sap through contact between healthy and infected plants, and through cultivation and handling.

Symptoms usually appear 4 to 10 days after inoculation, generally developing faster at higher temperatures. Epidemics are usually associated with abnormally high temperatures during the growing season. Warm periods during late fall or early spring result in high populations of feeding aphids and an increase in the amount of field infection.

Management

Cultural practices — The combined use of resistant cultivars and effective weed control provides the most effective approach to managing losses caused by spinach blight. Where practical, spinach should not be grown near other vegetables that are highly susceptible to cucumber mosaic virus, such as cucurbit crops and tomato. Early spring plantings can be grown before viruliferous aphids become prevalent. Perennial weeds should be eradicated, and top growth and roots should be killed the year before the crop is grown. The use of insecticides to control the aphids that spread the virus has generally not been effective.

Resistant cultivars — Spinach cultivars resistant to cucumber mosaic are available from most seed suppliers.

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(Original by L.W. Stobbs)

NON-INFECTIOUS DISEASES

▶ 5.11 Heart rot (boron deficiency)

Heart rot is the most common microelement disorder affecting beet. It has been observed wherever commercial production occurs.

Symptoms Symptoms vary with the location and environment, but generally they occur late in the growing season. Leaf symptoms are correlated with internal root necrosis, although they may precede root symptoms. Leaf symptoms usually appear after mid season on the younger leaves at the crown center, while older leaves remain normal in appearance. Young leaves are malformed, often with onesided development, and their length is greater in proportion to their width. They are also stunted and darker red in the lamina. A slight downward rolling of the leaf margins sometimes occurs. The malformed inner leaves become necrotic and die early, forming a rosette of dead leaves. Late in the season, new leaves develop from the dormant buds at the bases of the dead leaves.

Root symptoms consist of internal necrotic areas that vary in size, shape, and location. They may occur in the central or peripheral areas or be scattered throughout the root. The necrotic tissue is dark, hard-textured and blotchy. If the necrosis occurs well within the periphery, then the root may not show any external symptoms at harvest. Otherwise, a surface canker will occur and secondary organisms may enter. Presence of fungal mycelium on the surface of such cankers and within the underlying peripheral tissue may give the appearance of decay due to microorganisms.

Causal agent Beet is very susceptible to boron deficiency, mostly during dry seasons on sandy, leached soils that are low in organic matter. Boron, although present in the soil, may be unavailable to the plant in soils with a pH of less than 6 or more than 7.5. Seasonal conditions may influence the severity of the symptoms.

Management

Cultural practices — Visual symptoms and plant tissue analysis are useful for determining boron deficiencies. Plants suspected of being nutrient deficient should be sampled as soon as the problem is evident since nutrient levels vary with the age of the plant. Application of boron as a foliar spray is generally faster and more effective than soil application and foliar applications are not adversely affected by soil conditions. The tolerance to boron of other crops grown in rotation with beets must be considered, since the beet plant has a higher requirement for boron than do boron- sensitive crops. Adequate levels are best determined in consultation with local soil and crop advisors.

Selected references

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(Original by R.F. Cerkauskas)

NEMATODE PESTS

▶ 5.12 Northern root-knot nematode *Fig. 6.20*

Meloidogyne hapla Chitwood

Symptoms in spinach include stunting and yellowing of leaves, prolific branching of rootlets, and production of small, spherical galls on roots. For a complete description and management strategies, see Carrot, northern root-knot nematode, 6.20; see also Management of nematode pests, 3.12.

▶ 5.13 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms in beet and spinach include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38; see also Management of nematode pests, 3.12.

▶ 5.14 Sugarbeet cyst nematode *Figs. 5.14a,b*

Heterodera schachtii Schmidt

This nematode has been confirmed from scattered localities in Canada. It attacks beet, spinach and rhubarb, in addition to most cruciferous crops, including broccoli, Brussels sprouts, cabbage, cauliflower, kale, kohlrabi, radish, rutabaga, and turnip.

Symptoms Damage is most noticeable in patches where nematode densities are high. Infected plants are stunted and outer leaves wilt, yellow prematurely and die *(5.14a).* Heart leaves are more numerous than normal but reduced in size. Tap roots are short and stunted, and lateral root development is excessive, giving a whiskered appearance to the tap root. In summer, pin-head sized, white or brown cysts can be seen on washed roots, particularly in the root axils *(5.14b).*

Identification *Heterodera schachtii* (order Tylenchida, family Heteroderidae) is morphologically close to the golden nematode. Males of the sugarbeet cyst nematode are identified by their very short tail; females by the lemon-shaped cyst and characteristics of the cone top or genital (vulval)/anal area. The cysts can be seen with the unaided eye and are diagnostic of this nematode.

Life history *Heterodera schachtii* is a sedentary endoparasite. The second-stage juvenile induces the formation of transfer cells (syncitia) and completes its life cycle at that site. No galls are formed. Exudates produced by roots of the host crop stimulate juveniles to hatch and act as an attractant. The juveniles penetrate young roots and migrate through the cells to the vascular tissue near the root tip. Their feeding causes hypertrophy of several cells, turning them into syncitia that support the nematode as it matures and develops. There are three molts before the adult stage. The mature adult female becomes extremely swollen and eventually breaks out of the root with its head still embedded in the vascular tissue. When the female dies, its body wall hardens to become a protective, egg-filled cyst that may contain 200 to 300 eggs. The cysts are released into the soil when the roots die and may be spread by farm machinery and other physical means.

Management

Cultural practices — Because of the limited host range of this nematode, rotation to non-cruciferous vegetable crops for four to five years will significantly reduce numbers of infective juveniles. Transfer of nematode cysts from field to field in contaminated soil on machinery should be avoided.

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(Original by T.C. Vrain and B.A. Ebsary)

INSECT PESTS

▶ 5.15 Beet leafhopper *Figs. 5.15; 5.15T1*

Neoaliturus tenellus (Baker)
(syn. *Circulifer tenellus* (Baker))

The beet leafhopper is an introduced insect that breeds in the southwestern United States, migrates long distances, and invades southern Canada annually in the dry interior of British Columbia, where it extends as far north as Cache Creek by late July. It feeds on beet and sugar beet and on weeds, such as Russian thistle *(Salsola kali* L.). The beet leafhopper is a potential vector of beet curly top virus.

Identification The beet leafhopper *(5.15)* (family Cicadellidae) can be distinguished from the aster and other leafhoppers by the truncate sub-genital plates in the male and by a dark-margined notch on the pre-genital, seventh abdominal sternite in the female *(5.15T1).*

Selected references

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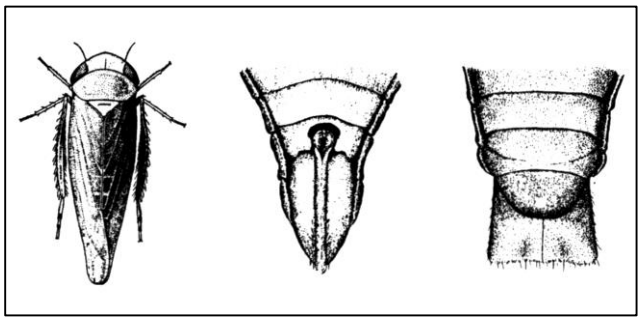
(Original by K.G.A. Hamilton)



5.15T1 Beet leafhopper; adult (left), and genitalia of female (center) and male (right), ventral view. Reprinted from *Utah Agric. Exp. Stn. Bull.* 205 (1928) and *Leaflet* 8 (1934).

▶ 5.16 Flea beetles *Fig. 5.16*

Redheaded flea beetle *Systena frontalis* (Fabricius)
Other flea beetles

Flea beetles (family Chrysomelidae) are common pests of beet. The redheaded flea beetle *(5.16)* is observed in great numbers after the end of July in Quebec. It is larger than other flea beetles and its leaf-feeding damage is much more visible at harvest. Other flea beetles (see Crucifers) are most numerous early in the season and pose a risk to beet, chard and spinach crops when the plants are small and under stress.

(Original by C. Ritchot)

▶ 5.17 Other insect pests *Figs. 5.17a-f; see text*

Aphids
- Black bean aphid *Aphis fabae* Scopoli
- Green peach aphid *Myzus persicae* (Sulzer)
- Sugarbeet root aphid *Pemphigus populivenae* Fitch

Beet webworm *Loxostege sticticalis* (L.)
Leafminers
- Beet leafminer *Pegomya betae* (Curtis)
- Spinach leafminer *Pegomya hyoscyami* (Panzer)

White grubs *Phyllophaga* spp.

Aphids

The black bean aphid (see Potato, 16.43, *16.43T1)* is sometimes a pest of beet and related crops in British Columbia, southwestern Quebec and southern Ontario. It is a known vector of cucumber mosaic virus of spinach.

The green peach aphid (see Potato, 16.41, *16.41a,b*) has been observed on beet in southwestern Quebec. In the past, it has caused economically serious injury to spinach. The green peach aphid is a vector of cucumber mosaic virus.

The sugarbeet root aphid occurs from British Columbia to Quebec. It transmits such viruses as curly top and virus yellows. This aphid feeds below ground on the roots of beet and related crops in the summer and fall, later dispersing to lay eggs on balsam poplar and cottonwood (*Populus* spp.), which are primary hosts. The egg is its main overwintering stage in Canada.

Beet webworm

The beet webworm (*5.17a-c*) (family Pyralidae) occurs across Canada. Its larvae communally web the leaves and petioles of beet and related crops. To some extent, other vegetable crops also are attacked.

Leafminers

(family Anthomyiidae) The beet leafminer and spinach leafminer are considered to be distinct species (see Selected references, Griffiths 1982), the former occurring from Quebec west in Canada, the latter mainly in eastern Canada. These flies (5.*17d)* lay their eggs on the underside of leaves of beet, spinach and related plants, and the newly hatched larvae eat their way into and produce mines in the leaves (*5.17e,f*). The intensity of infestation varies greatly from one field to another in the same area. In southwestern Quebec, populations of the spinach leafminer are low in June and higher in August and September. The damage by these leafminers only affects the appearance of the leaves, not the yield. The primary concern is with beets sold in bundles with their leaves (bunching beets).

White grubs

White grubs are the larvae of June beetles (see Potato, 16.49, *16.49c-e).* They can damage the sides of roots of beet grown in soil previously cultivated to meadows or in weedy fields that are already infested.

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(Original by J.A. Garland and C. Ritchot)

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6 Carrot

BACTERIAL DISEASES

6.1 Bacterial leaf blight *Figs. 6.1a,b*

Xanthomonas campestris pv. *carotae* (Kendrick) Dye

Bacterial leaf blight occurs occasionally throughout Canada but has caused relatively little loss in processing and table- stock carrots. In the United States, leaf blight has sometimes caused serious damage to carrot seed crops. The cultivated carrot is the only host of *X. campestris* pv. *carotae.*

Symptoms Lesions on the leaves are irregular, dark brown, necrotic in the center, and soon become dry and brittle with an irregular yellow halo *(6.1b).* In contrast, lesions on the petioles, peduncles and pedicels appear water-soaked and are linear. Infection often takes place along the leaf margin, inducing lateral curling of the leaflet. If the flower umbel is attacked when young, the entire umbel can be killed or blighted. However, if infection takes place after umbel formation, only part of it may be affected. A gummy bacterial exudate sometimes collects and flows down from the infected plant parts *(6. 1a).* When the disease is severe, most of the leaflets turn yellow and many lower leaves are killed.

Early symptoms can be mistaken for cercospora and alternaria blights, but the flow of bacterial exudate from infected plant parts is characteristic of bacterial leaf blight. Infected plant parts can be cut, placed in water, and examined microscopically to see if bacterial streaming is evident.

Causal agent The cells of *Xanthomonas campestris* pv. *carotae* are cylindrical rods, rounded at the ends, which occur singly or in pairs. The single cells are 1.4 to 2.8 µm long, 0.4 to 0.9 µm wide, and motile by one polar flagellum, 7 µm in length, attached to one end. The bacteria are aerobic, do not form spores, have no pronounced capsulation, and are Gram- negative.

The bacteria grow well on potato-dextrose agar, on which the colonies are straw yellow. The optimum pH for growth in culture is 7 and the optimum temperature is 25 to 30°C. Growth is very sparse below 13°C. The thermal death point is 49°C.

Disease cycle The pathogen survives in or on carrot seed originating from diseased plants and may persist for one winter season in infested crop residues in the soil. The bacteria are dispersed by splashing water and by insects. The presence of liquid water or plant exudates is required for infection. The optimum temperature for multiplication of the pathogen is 25 to 30°C. Symptoms appear 10 to 12 days after inoculation, and epidemics can develop rapidly under warm, wet conditions.

Management

Cultural practices — Growers should plant disease-free seed, follow a two- to three-year crop rotation and plow or disc infested crop residues to reduce inoculum levels. Seed can be disinfested with hot water at 52°C for 25 minutes.

Resistant cultivars — Some tolerance to bacterial leaf blight occurs in the cultivars Waltham Hi-Color and Danvers.

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(Original by A.C. Kushalappa)

▶ 6.2 Bacterial soft rot *Fig. 6.2*

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*

Bacterial soft rot is a common disease of carrot. It can cause major losses in storage, where it occurs as a secondary invader of previously diseased or damaged roots. The pathogen can also cause major losses in the field, although this occurs only sporadically. Field infections are usually associated with extremely wet conditions. Bacterial soft rot also occurs on other vegetable crops, especially those with succulent heads, fruits or tubers.

Symptoms The first symptoms to appear on carrot roots are small water-soaked lesions, which quickly enlarge and coalesce. The affected areas soon become watery and mushy, the surface becomes depressed and the lesions may darken (6.2). The outer surface often remains intact over the liquified interior, but is easily broken if the carrots are moved. The lesions soon become opaque and slimy. Cracks often form on the root surface and the macerated tissue can ooze out. When this tissue is exposed to the air, it turns tan or gray. There is no foul odor associated with bacterial soft rot until the tissues collapse and secondary organisms invade.

Sclerotinia sclerotiorum can also cause a soft rot of carrot (see sclerotinia rot, 6.15). This disease can be distinguished from bacterial soft rot by the presence of white mycelium on the affected roots. Because infection by *Sclerotinia* and *Rhizoctonia* fungi can predispose carrots to attack by soft rot bacteria, it is not unusual for two or more of these pathogens to occur together, thus making diagnosis more difficult.

In the field, soft rot can create pits in the carrot root, with a distinct margin between diseased and healthy tissue. Under severe conditions, for instance where the soil has become waterlogged, rotted portions of the root may remain in the ground when the plant is pulled. If drier soil conditions return, the root will continue to grow, but will be forked or stunted and hence unmarketable.

Causal agent (see Potato, bacterial soft rot, 16.2)

Disease cycle (see Potato, bacterial soft rot, 16.2)

Management

Cultural practices — It is advisable to grow carrots in well-drained soil. Crop rotation with corn, small grains, grasses, alfalfa, clover, beet and bean may help reduce populations of soft rot bacteria in soil. When harvesting, carrot roots should be handled carefully to minimize cuts and bruises. Wherever possible, crops should be graded before storage to remove any damaged or diseased roots. Producers should store carrots close to 0°C and 90 to 95% relative humidity. Care should be taken not to let moisture condense on the roots. As well, ventilation equipment should be set to allow for regular exchanges with fresh outside air.

Chemical control — If carrot roots are washed before storage, transit or packaging, rinsing them in a solution of sodium hypochlorite in clean water may suppress bacteria and inhibit rot. Growers and packers should consult the Health Protection Branch, Health and Welfare Canada, for guidelines on the use of chlorinated water on vegetables. When soft rot becomes a recurring problem, storage rooms, pallet boxes, and washing and handling equipment should be thoroughly disinfested before re-use.

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(Original by M.R. McDonald)

▶ 6.3 Crown gall *Fig. 6.3*

Agrobacterium tumefaciens (E.F. Smith & Townsend) Conn

Crown gall has been reported only rarely on carrot and, when present, disease incidence has been low. Small galls on roots are usually removed during washing, so slightly affected roots may be marketable. Badly affected roots are unsaleable.

The crown gall bacterium is distributed worldwide and occurs across Canada. It affects woody and herbaceous plants belonging to 140 genera in more than 60 families. It is most often found on pome and stone fruits, brambles *(Rubus* spp.), grape and roses.

Symptoms Crown gall in carrot appears as tubular to irregular, yellow to tan galls on the stem near the crown or on the roots *(6.3).* The galls usually develop where lateral roots join the tap root. However, galls can develop wherever the plant has been injured. One or more galls of various sizes may appear on a single plant by about midsummer and continue to increase in number and size until harvest.

Causal agent The ability of *Agrobacterium tumefaciens* to produce galls is resident in a tumor-inducing (Ti) plasmid. Related species, such as *A. rhizogenes* (Riker *et al.*) Conn and A. rubi (Hildebrand) Starr & Weiss, can also cause disease if they contain the Ti plasmid, but with different symptoms.

Bacteria in the genus *Agrobacterium* are Gram-negative, single-celled rods that do not form spores. They are motile, have reduced peritrichous flagella and are oxidative and oxidase positive.

Agrobacterium tumefaciens can be isolated by excising small pieces from the white, actively-growing portion of the gall. The pieces should be macerated in about 0.1 mL of sterile water and let stand for 30 minutes. The liquid should be streaked onto a non-selective medium, such as Difco nutrient agar containing 0.01% yeast extract. Selective media have also been developed. It is not unusual for gall tissue to be devoid of *Agrobacterium*. Not all strains of *Agrobacterium* are pathogenic. Pathogenicity can be confirmed by inoculating the isolated bacteria onto a host plant and reproducing the symptoms.

Disease cycle The pathogen can live as a saprotroph in soil for several years and can also overwinter in galls. Any means that moves infested soil can spread the pathogen. The bacteria enter the plant through fresh wounds where they move intercellularly. In a short time, usually about three days, part of the 77-plasmid is transferred from the bacteria to the plant cell. The infected plant cells are induced to produce hormones, which result in uncontrolled growth and division, and to produce specific chemicals known as opines, which can be utilized only by the crown gall bacteria. Once plasmid transfer has taken place, the bacteria are no longer necessary for the production of a gall. Small galls can be seen 10 to 14 days after inoculation.

Management

Cultural practices — Some reduction in soil populations may be achieved with long crop rotations with onion, corn, oat, grasses and other immune crops.

Biological control — Successful biological control of crown gall on stone fruits and roses has been achieved by inoculating these hosts with a non-pathogenic strain of *Agrobacterium radiobacter* (Beij. & Van Delden) Conn. However, this technique is not yet practical or economical for carrot.

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(Original by M.R. McDonald and R. Crête)

▶ 6.4 Scab *Fig. 6.4*

? *Streptomyces scabies* (Thaxt.) Waksman & Henrici (syn. *Actinomyces scabies* (Thaxt.) Giissow)

This disease occurs throughout the world. In Canada, it has been reported on commercial carrot only rarely. The pathogen can attack several other species of root crop vegetables, including beet, potato, turnip, radish and parsnip.

Symptoms Some forms of *S. scabies* cause damping-off. Surviving plants show typical scab symptoms on the roots *(6.4).* Scab lesions are formed by the abnormal growth of the host cells, resulting in corky tissue that is usually darker than healthy tissue. Lesions are sometimes sunken below or raised above the surface of the healthy skin. Many single lesions may join to form continuous scabby areas.

Causal agent (see Potato, common scab, 16.5) The identification of *Streptomyces scabies* as the cause of carrot scab is provisional. The causal agent, first isolated from scabby carrots in Michigan, was reported to be characteristic of 5. *scabies,* except that the spore surfaces were echinulate rather than smooth. One potato strain of *S. scabies* produced scab symptoms on 40% of the carrots grown in soil infested with this organism. Scab symptoms on carrot have also been attributed to physiological causes; for example, excessive soil moisture before harvest may induce proliferation of lenticels.

Disease cycle (see Potato, common scab, 16.5) The disease tends to be more severe in dry, alkaline soils.

Management Little is known about scab on carrot, so management strategies are based on those recommended for potato.

Cultural practices — Scab is rarely severe enough on carrot to warrant specific control measures. However, because alkaline soils are known to favor the development of scab in other crops, such as potato, some reduction of disease may be achieved by avoiding carrot production on alkaline soils or by applying acid-tending fertilizers, such as ammonium sulfate or sulfur, to lower the soil pH. Growing carrot in soils with good moisture-holding capacity or irrigating to maintain an even water supply may reduce scab. Growers should avoid planting carrot in fields used for potato production. Long rotations with small grains, grasses or corn may also help to reduce scab severity.

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(Original by R. Crête and M.R. McDonald)

FUNGAL DISEASES

▶ 6.5 Alternaria leaf blight *Figs. 6.5a,b*

Alternaria dauci (Kühn) Groves & Skolko

Alternaria blight is the most common foliar disease of carrot. It can lower yields by reducing the leaf area available for photosynthesis and by destroying the carrot tops, which are necessary for mechanical harvesting. *Alternaria dauci* can also moderately affect parsley.

Symptoms Early infection of seedlings can cause damping-off. Foliar lesions on mature plants resemble those caused by *Cercospora carotae* but are more irregularly shaped. The lesions generally first appear along the leaflet margins and are dark brown to black with a yellow border *(6.5a).* When numerous, the spots grow together and the leaflets shrivel and die, giving a blighted appearance to the plant. Under cool, humid conditions, a velvety surface layer of mycelial growth and conidia on the leaves is visible to the naked eye. Merging of the lesions can girdle the petiole and the entire leaf may collapse and die. Blighted carrot tops may break off when gripped by mechanical harvesters, leaving the roots in the ground. Fleshy roots are not attacked by *Alternaria dauci.* In commercial fields, alternaria blight often appears later than cercospora blight because older leaves are more susceptible than younger leaves to *Alternaria.* In the past, damage by *A. dauci* may have been overestimated because of the similarity of symptoms with those caused by *A. alternata,* which is a weak secondary pathogen that produces numerous conidia on blighted leaves.

Causal agent *Alternaria dauci* conidiophores arise singly or in small groups and are straight, flexuous or sometimes geniculate. They are olivaceous brown or brown, about 80 pm long and 6 to 10 pm thick, and bear conidia successively during growth. The conidia are usually solitary, brownish, straight or curved, obclavate and rostrate, with the beak up to three times the length of the body of the spore (*6.5b).* They are 100 to 450 µm long, including the beak, and 16 to 25 µm thick at the broadest part. There are 7 to 11 transverse and one to several longitudinal or oblique septa per conidium. The beaks are often branched and flexuous.

To isolate *A. dauci,* diseased carrot leaves should be collected early in the morning and examined microscopically at 60 x for conidia, which can be picked up by lightly touching them with a sterile inoculating needle. The conidia should be streaked onto potato-dextrose or V-8 agar and the plates incubated at 24°C for 16 hours under cool, white, fluorescent lamps at 50 to 150 µE/sec/m^2. New conidia should form within three days and can be identified by their size and morphology. For long-term storage of cultures, V-8 and carrot-leaf agar have proven effective.

Alternaria alternata is often isolated from the blight lesions, sometimes abundantly and without *A. dauci. Alternaria alternata* can be distinguished from *A. dauci* by its beak, which is shorter than the length of the conidium body and has a swollen tip, and by its habit of forming conidia in long chains.

Disease cycle The pathogen survives in or on seed and can be introduced to the field in this way. It overwinters on diseased crop residues in the soil, on carrot tops discarded in spring after storage, and on weed hosts. *Alternaria dauci* produces conidia at temperatures ranging from 8 to 28°C, with abundant production at 20 to 30°C when there is high humidity. The conidia are spread by wind, running and splashing water, farm machinery and field workers. Most are dispersed during the morning hours when humidity decreases as temperature and wind speed increase. Moisture from dew or rain is essential for germination and

penetration. Symptoms appear in 8 to 16 days, depending on weather conditions. The optimum temperature for fungal growth and infection is 28°C, with some infection occurring as low as 14°C and as high as 35°C. Cool, humid weather favors alternaria blight. This disease normally occurs late in the crop's growth, in contrast to cercospora blight. This may be related to availability of initial inoculum, the growth stage when the plant is most susceptible, and environmental conditions late in the growing season.

Management

Monitoring — The methods recommended for cercospora leaf blight may also be used for alternaria leaf blight.

Cultural practices — Growers should use carrot seed produced in areas where *Alternaria dauci* is absent. A two- to three-year crop rotation and turning under infested crop residues in the fall will reduce the carry-over of inoculum.

Resistant cultivars — Waltham Hi-Color, Orlando Gold and Hi-Color 9 carrrots are tolerant to alternaria blight.

Chemical control — Carrot seed should be treated with a recommended fungicide before planting. Producers should consider initiating fungicide sprays when the disease threshold reaches 25% of plants with the middle leaf diseased and when accompanied by a rain forecast or a minimum temperature greater than 16°C for the next night. When these conditions persist, subsequent sprays should be made at 7- to 10-day intervals. Urea can be applied as a foliar spray toward the end of the season to stimulate the growth of new foliage, which will assist mechanical harvesting.

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(Original by A.C. Kushalappa)

▶ 6.6 **Black root rot (black mold)** *Figs. 6.6a-c*

Chalara elegans Nag Raj & Kendrick
(syn. *Trichocladium basicola* (Berk. & Broome) J.W. Carmichael)
(synanamorph *Thielaviopsis basicola* (Berk. & Broome) Ferraris)

Black root rot has been a severe post-harvest disease on fresh market carrots grown in muck soils in the Fraser Valley of British Columbia. There have also been sporadic occurrences on muck-grown carrots in Ontario and on carrots grown in mineral soils in Alberta.

Chalara elegans has a worldwide distribution and a wide host range, attacking crop plants in the legume, potato and cucurbit families, as well as numerous ornamental and some woody species.

Symptoms Superficial, black, irregular, randomly scattered lesions, varying from 3 to 20 mm, develop on roots after carrots are washed, graded and stored in polyethylene bags at temperatures above 25°C and at high relative humidities *(6.6a).* The root discoloration affects only the epidermis and is due to extensive sporulation of the fungus *(6.6b).* In some cases, the blemishes are less than 1 mm deep.

Chalara elegans can cause a serious root rot of tobacco, bean, pea and many other crops, but field symptoms have not been noted on carrot. Likewise, carrots grown in C. *elegans*-infested soil in the greenhouse show no visible symptoms of root infection. However, if carrots are wounded, the fungus can rapidly invade the root and the damaged area becomes covered with chlamydospores.

Causal agent The fungus may be variable in morphology when grown on V-8 or potato-dextrose agar. Variants may be zoned with dark gray mycelium, albino, brown, aconidial (absence of endoconidia), or miscellaneous mycelial types. Some variants differ not only in cultural characteristics, such as production and shape of chlamydospores, but also in pathogenicity. In general, colonies are slow growing, effuse, gray, olivaceous, brown or black, and often velvety. The mycelium is partly superficial and partly immersed.

Two types of spores are produced *(6.6c).* Chlamydospores are dark brown, thick-walled, subrectangular, and about 6.5 to 14 by 9 to 13 pm. They are produced laterally or terminally from hyaline basal cells in short chains of about five to seven cells that resemble large multiseptate conidia. Only the apical chlamydospore in each chain is rounded at the end; the others are truncate at both ends. The chain of chlamydospores breaks apart at maturity. Endoconidia are subhyaline, produced in long chains from subhyaline phialides that are up to 100 pm long, thicker at the base, and tapering towards the apex. The endoconidia are cylindrical with truncated ends, 7.5 to 19 by 3 to 5 pm, and liberated through the apex of the phialide in succession.

Chalara elegans can be selectively isolated from soil or plant tissues using fresh carrot disks. Semi-selective media have also been developed.

Disease cycle *Chalara elegans* occurs widely as a soil inhabitant and persists as chlamydospores in cultivated and non-cultivated areas. It can survive for long periods in organic matter in the soil; however, survival is greatly reduced by a high moisture content. Chlamydospore germination is optimum at 25°C and is stimulated by carrot residues and alternating dry and moist conditions.

Disease development is associated with freshly harvested carrot roots stored at high temperature and high relative humidity. It is rarely a serious problem if carrots are stored under optimum conditions (0 to 1°C and 98 to 100% relative humidity). Lesions always occur at the sites of wounds incurred during harvesting, grading and sorting. Black root rot has not been reported in late crops harvested under cooler conditions or in carrots dug by hand. Wounding appears to be a prerequisite for infection.

Management

Cultural practices — Growers should avoid bruising or otherwise damaging carrot roots during and after harvest. Efforts should be made to remove as much of the soil adhering to roots as possible before grading. It is advisable to cool freshly harvested carrots as soon as possible and to avoid storing them at high temperatures and high relative humidities. Prompt hydrocooling will rapidly reduce core temperature in the largest carrots below 7°C. Storage temperature should be maintained so that core temperature does not exceed 7°C. This requires a room temperature of 5°C or less.

Chemical control — Washed carrots should be rinsed in chlorinated water before packaging in plastic bags. Growers and packers should consult the Health Protection Branch, Health Canada, for guidelines on the use of chlorinated water on vegetables.

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(Original by R. Cerkauskas and Z.K. Punja)

▶ 6.7 Black rot *Fig. 6.7*

Alternaria radicina Meier, Drechs. & E.D. Eddy
(syn. *Stemphylium radicinum* (Meier, Drechs. & E.D. Eddy) Neergaard)

Black rot has been reported from most provinces where carrot is grown. Losses may be high in the field, although the disease was originally reported causing a serious storage rot. The black rot pathogen can also attack celery, parsley, parsnip and dill.

Symptoms Symptoms occur on seeds, umbels, foliage, petioles and roots. On seeds, *Alternaria radicina* produces a diffuse black weft of mycelium that may envelop seeds and within which black conidia are found. Among the various symptoms that may develop on seedlings are seed decay, damping-off, blackened hypocotyls and deformed roots. The latter symptom occurs when the lower end of the tap root is killed. Pre- and post-emergence damping-off may result from seed-borne infection or planting in infested soil. Affected seedlings may have a continuous, tan-brown to black lesion constricting the stem. This lesion can extend from soil level upward and sometimes reaches the cotyledons.

The greatest damage occurs on the roots. Initially, infection may occur at the petiole base, where shallow, shiny black lesions develop and later spread into the crown and down the sides of the root. When the fungus attacks the crown, the invasion is usually deep and extends along the core. Lesions on the side of the root are generally circular, shallow and slightly depressed. The decayed tissue is greenish to black, with the surface bearing conidiophores and conidia *(6.7).* The secondary lesions that develop below ground are often coincident with cracks and splits that result from nutritional or other stresses. Extensive black rot often develops in roots of carrot seed crops.

A dry, mealy rot without specific odor may develop when carrot roots are held in storage. At high humidity, the root decay is soft and watery with a dark advancing margin.

Under field conditions, foliar symptoms are less severe than those caused by *Alternaria dauci* and generally occur on older outer leaves. Occasionally, irregular black lesions develop on the edges of the leaflets. However, inoculated plants initially show small brown lesions on the leaves and petioles, which later turn black. Lesions on affected petioles extend to the vascular tissue, resulting in foliar chlorosis, wilting and finally death of the leaves. In seed crops, umbels, seedheads and seed stalks are also affected and darken as they approach maturity.

Causal agent Colonies of *Alternaria radicina* are blackish-brown to black and develop rapidly and abundantly on potato-dextrose agar. The hyphae are simple, straight with some branching, brown and septate. The conidiophores are usually simple and unbranched, straight or flexuous, septate, pale to mid-brown or olivaceous brown, smooth, up to 200 by 3 to 9 pm, with one or more conidial scars. The conidia are solitary or in chains of two or rarely three. They are variable in shape, often elliptical to

pear- shaped, with the broadest end attached to the conidiophore. There are usually three to seven transverse and one to several longitudinal or oblique septa. Conidia vary from 27 to 57 pm in length and from 9 to 27 pm in width in the broadest part, with mean values of 38 and 19 pm, respectively. Conidia of *A. radicina* are differentiated from those of *A. dauci* by the absence of a long appendage at the distal end and the smaller size of the conidia, and from *A. alternata* by the absence of long chains of spores.

The fungus is readily isolated from diseased tissue hy surface sterilizing the material and placing it on potato-dextrose agar. Incubation of surface-disinfested seeds in the light in a sterile chamber containing moist filter paper enhances recovery of the fungus from infected seed.

Disease cycle The fungus can infect carrot tissue at all stages of growth. Primary infections may occur on foliage, although this is not essential since root penetration from soil-borne inoculum is also possible through rootlets, wounds or unwounded tissue. Contamination of healthy roots may also occur from infected foliage during harvest, leading to storage decay.

Alternaria radicina is seed- and soil-borne, survives on carrot residues on the soil surface, and persists for at least eight years in mineral soil. However, viability is lost more quickly when the residue is buried than when left on the soil surface.

The fungus tolerates temperatures ranging from –0.5 to 34°C with optimal *in vitro* growth at 28°C, although seed emergence in muck soil infested with *A. radicina* is greatly reduced below 18°C. In storage, the fungus requires a relative humidity of above 92% for rapid root rot development. Extensive leaf and root infections usually reduce the storage life of carrots.

Management

Cultural practices — Where possible, growers should practice a minimum eight-year crop rotation with crops other than carrot, dill, parsley, parsnip and celery, using only seed that has been treated with hot water or a fungicide. For hot-water treatment, carrot seed is placed in a cheesecloth bag, which should be no more than half full with seed, and soaked for 25 minutes in water at 50°C, with continuous stirring to obtain rapid and uniform distribution of heat. Infested crop residues should be turned under to avoid leaving them on the soil surface where the fungus can form conidia and spread. All diseased or damaged roots should be discarded before storage.

In storage, the temperature should be maintained near 0°C and the relative humidity at about 92% to keep storage decay to a minimum. This relative humidity is lower than that required for optimum storage, therefore it is advisable not to store infested crops for more than three or four months. Storages and containers should be cleaned and disinfested before use.

Resistant cultivars — Improved Half Long Chantenay, K-2043 and several other cultivars are relatively resistant to black rot.

Chemical control — Fungicides can be used to control the foliar phase of the disease. They may also reduce the incidence of storage decay.

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(Original by R.F. Cerkauskas)

▶ 6.8 Cavity spot *Fig. 6.8*

Pythium intermedium de Bary
Pythium irregulare Buisman
Pythium sulcatum Pratt & Mitchell
Pythium sylvaticum W.A. Campbell & J.W. Hendrix
Pythium ultimum Trow
Pythium violae Chesters & C.J. Hickman

Cavity spot of carrot is widely distributed and has been reported in North America, Europe and parts of Asia. It affects carrot in all regions of Canada and also has been observed on parsnip. While the disease rarely reduces tonnage, carrot roots with cavities are not acceptable for the fresh market or for processing, and marketable yield can be severely reduced. In extreme cases, fields of carrot with severe cavity spot have been abandoned. This disease occurs on carrot grown in both organic and mineral soils. In some early accounts, cavity spot was referred to as “horizontal lesions.” The *Pythum* species causing cavity spot can also attack a wide range of other vegetable crops.

Symptoms Cavity spot is most easily seen on freshly washed carrots. The cavities appear as elliptical lesions that are sunken a few millimetres below the surface of the root. The lesions are elongated horizontally, arranged randomly, and darken with age (6.8). Vertical cracks are sometimes associated with the cavities.

The first symptoms appear under intact periderm as sunken areas that are either gray or not discolored. As the lesions develop, the periderm ruptures and darkens. Lesions vary in size, and secondary organisms may infect the carrot, causing rapid rotting. The cavities increase in size as the roots grow. There have also been reports that they can increase in size while the carrots are in storage, but it is not clear whether this is due to the initial infection or to secondary invasion.

There are no foliar symptoms of the disease. To determine the severity of cavity spot, carrots must be pulled and the roots washed.

Causal agent Since its description in 1961, cavity spot has been attributed to numerous physiological causes, including calcium deficiency, soil ammonification and anaerobic growing conditions. Biological agents that have been implicated in cavity spot formation include anaerobic pectolytic bacteria (*Clostridium* spp.), fungus gnat larvae (*Bradysia* spp.), and slow-growing *Pythium* species. The issue is further complicated by a disease of carrot roots caused by *Rhizoctonia solani*, which has also been called "cavity spot." In Canada, *Pythium* species appear to be the main cause of cavity spot.

The mycelium of *Pythium* spp. is colorless and the hyphae are aseptate, except for old hyphae. Septa are found at the base of reproductive structures. Cytoplasmic streaming can be seen in young hyphae. Most species do not produce aerial mycelium on cornmeal or potato-carrot agar, but many form cottony aerial mycelium on oatmeal agar. *Pythium sylvaticum* and *P. ultimum* produce thick cottony mycelium on cornmeal agar.

Identification of *Pythium* spp. is based on the morphology of the sporangia, zoospores, oogonia and antheridia. The presence, size, shape and number of these structures varies considerably, depending on culture media, age of the culture, temperature and other environmental conditions. Standard cultural methods are very important for accurate identification.

Pythium violae and *P. ultimum* are similar, but *P. violae* has larger oogonia (mean diameter 29.5 *versus* 21.5 µm) and it has more numerous, sometimes stalked, monoclinous antheridia that originate a short distance from the oogonia. The daily growth rate of *P. violae* and *P. ultimum* on potato-carrot agar at 25°C is 15 and 30 µm per day, respectively. Both species have aplerotic, globose oospores, sac-like antheridia, globose hyphal swellings that can be terminal or intercalary, and no sporangia. Most oogonia are terminal, but *P. violae* may have intercalary oogonia as well.

Pythium sulcatum has filamentous sporangia and zoospores that are produced at 20°C. The oogonia are terminal or intercalary and smooth with oospores that are aplerotic and average 14.5 µm in diameter. The mycelial growth rate on potato-carrot agar is 13 to 14 mm per day. Antheridia are the major distinguishing feature of *P. sulcatum.* There are one to three per oogonium and they are monoclinous and diclinous, but with stalks that are often branched and with antheridial cells that are large, folded or furrowed and often encircle the oogonium. The cardinal growth temperatures are: minimum 2 to 3°C, optimum 20 to 28°C, and maximum 36 to 37°C.

Pythium sylvaticum sometimes produces oogonia in single cultures but is primarily heterothallic and produces oogonia along the line of contact of two compatible cultures. The oogonia are smooth, terminal or intercalary with an average diameter of 19.3 µm. Oospores are aplerotic. The antheridia are two to four per oogonium, diclinous with branched stalks and soon disappear after fertilization. Sporangia are not formed. Daily growth rate on potato-carrot agar is 30 mm or more.

Pythium irregulare is characterized hy its ornamented oogonia, which are irregular in shape and also vary in size. Sporangia are seldom formed. Antheridia are one to two per oogonium and usually monoclinous, originating some distance from the oogonia. Oogonia are usually intercalary, sometimes terminal, 15 to 25 (mean 18.5) µm in diameter, and most oospores are aplerotic. Daily growth rate on cornmeal agar is 25 mm. The cardinal temperatures are: minimum 1°C, optimum 30°C, and maximum 35°C.

Pythium intermedium does not form sporangia. The hyphal swellings are abundant and form regular, dense chains. This species is heterothallic and oogonia which form in dual cultures are globose, mean diameter 21.5 µm, and smooth with a thin wall. The oospores are aplerotic and occasionally there are two per oogonium. Antheridia are diclinous with one to seven antheridial cells. The stalks are often branched. Daily growth rate on potato-carrot agar is about 30 mm.

To isolate *Pythium* spp. from cavities, carrots should be thoroughly washed in running water, but not surface sterilized. Pieces of tissue should be excised from the edge of the cavities and placed on Mircetich medium (see Selected references, Mircetich 1971). This medium should be freshly prepared and kept in the dark. Inoculated plates may be incubated at room temperature in the dark. *Pythium* colonies will begin to grow within 24 to 48 hours. Slow-growing species are more difficult to isolate and may require longer incubation. Transfers can be made to water agar. Placing the mycelial plug on the bottom of the petri dish under a flap of water agar helps to reduce bacterial contamination. *Pythium* spp. will grow upward through the water agar. Hyphal tips can be transferred to rolled-oats agar to stimulate the production of reproductive structures and to allow for identification.

Disease cycle Typical cavity spot symptoms are normally seen on carrots that have been growing for at least 12 weeks and are nearing marketable size and maturity. In Ontario, the first symptoms can be seen on carrots in early to mid-August, and cavity

spot severity increases during September and October. *Pythium* spp. can be isolated from cavities at any time after formation; however, reports from elsewhere in Canada and from Britain indicate that *Pythium* spp. can also be isolated from carrot seedlings. Infection of the carrot root probably occurs at an early stage of carrot growth.

Increases in cavity spot severity have been associated with the application of high rates of chemical fertilizers, and also with increases in soil moisture early in the season or at the time of maturation, while reductions are observed in soils with a pH greater than 8.

Pythium violae and *P. sulcatum* cannot readily be isolated from field soils using standard dilution-plate methods, possibly because faster-growing species overgrow them. Therefore, it is difficult to determine the effects of environmental factors on populations of the fungus in the soil or what effect these populations may have on the incidence or severity of cavity spot. Severe cavity spot may develop on carrot grown in newly cleared land or cultivated fields where umbelliferous crops have never been grown. Conversely, fields where carrot has been cultivated repeatedly may have no history of cavity spot. Fields known to produce carrots infected with cavity spot may not show disease from one year to the next depending on environmental conditions.

Management

Cultural practices — It is advisable to avoid using fields with a history of cavity spot and to grow carrot on raised beds to reduce the likelihood of excessive soil moisture levels. Crop rotation is not recommended because there is no relationship between cropping history and cavity spot severity nor any evidence that rotation will reduce cavity spot. Carrot should not be planted in soils with a high clay content.

While no direct relationship between soil nutrients and cavity spot has been shown, decreasing the level of chemical fertilizers applied to a field has been observed to reduce the severity of cavity spot.

Resistant cultivars — There is a wide range of susceptibility to cavity spot among commercial carrot cultivars. Six Pak, Six Pak II, 24 Karat, Spartan Premium, Dagger 78 and Orlando Gold are relatively resistant. Growers should consult provincial recommendations for a more complete list.

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(Original by M.R. McDonald)

▶ 6.9 Cercospora leaf blight *Figs. 6.9a-d*

Cercospora carotae (Pass.) Solheim

Cercospora blight is frequently serious on carrot. In Quebec, it is more severe than alternaria blight. Generally, cercospora blight occurs earlier than alternaria blight. With both diseases, crop losses are due mainly to the carrots that are left behind by mechanical harvesters. *Cercospora carotae* is reported to attack only carrot.

Symptoms Primary lesions appear on leaflet margins and cause lateral curling. These lesions are elongate, while those that are not along the margin tend to be roughly circular. On the leaf, the lesions first appear as small chlorotic specks that soon enlarge into small, tan, brown or almost black spots with a necrotic center surrounded by a yellowish area having no clear border *(6.9a).* As the lesions increase in number and size, they grow together and the entire leaflet withers and dies *(6.9c).* On the petioles and stems, lesions are elliptical and brownish with a paler center *(6.9b),* while in humid weather the spots are darker and the lower surface of the lesion appears light gray or silvery because of the mass of hyaline conidia, which is characteristic. Lesions may merge and girdle the stem, eventually causing collapse and death of the entire leaf. When mechanical harvesters grip the blighted carrot, the tops break easily, leaving the roots in the ground. When floral parts on carrot grown for seed are infected early, they shrivel before the seed is produced; however, when the infections occur later, the pathogen may enter the seed and serve as seed-borne inoculum. *Cercospora carotae* does not attack the fleshy roots.

Causal agent Host specificity has been used in identifying species of *Cercospora*. Although all cross-inoculations with other genus or species groups have not been made, *C. carotae* infects species of the genus *Daucus*. Colonies of *Cercospora carotae* are gray with small, scattered tufts of conidiophores. On leaf tissue, the conidiophores arise in groups from a pseudostroma in the substomatal cavity, usually emerging through stomata or rupturing the stomatal opening. They are unbranched, straight or flexuous, sometimes geniculate with two to three scars near the tip, olivaceous brown, usually 2 to 3 µm thick, and not enlarged at the base. The conidia are borne successively at the tip as the conidiophore grows. They are filiform, cylindrical, truncate at the base, hyaline to slightly dark, one- to six-septate, and 40 to 110 µm long by 2.2 to 2.5 µm wide (*6.9d*).

The fungus grows and sporulates best in carrot-leaf-infusion agar at a pH from 5 to 6.5 and temperatures from 19 to 28°C; no growth is observed below 7°C and above 37°C. Most of the conidia are produced in 6 to 12 days.

Disease cycle *Cercospora carotae* overwinters on and in seed, in diseased host debris and on wild carrot and other host plants. Conidia are dispersed by wind, splashing rain, farm machinery and workers. Conidia germinate and penetrate through stomata over a wide range of temperature and leaf wetness duration. A significant amount of disease occurs at temperatures from 20 to 30°C (optimum 28°C) and after a leaf wetness period longer than 12 hours. The lesions appear in about 10 days depending on the incubation temperature and the cultivar. In contrast to alternaria blight, the younger leaves are more susceptible to cercospora blight. This may explain why cercospora blight is more severe than alternaria blight in the early stages of plant growth. The initial increase of cercospora blight varies with the seeding date. Epidemics usually develop more rapidly in late-sown carrot because of the influx of inoculum from neighboring fields seeded earlier in the season.

Management

Monitoring — Disease incidence should be determined at biweekly intervals after the five-leaf stage by randomly sampling 50 plants while walking diagonally across the field. Sequential sampling methods can be adopted to reduce the sample size to below 30 plants per field, depending on the disease incidence. Disease incidence levels are used in determining the need for fungicide application.

Cultural practices — Producers should use carrot seed produced in areas where the pathogen is absent. Fall plowing of infected crop residues coupled with a two- to three- year rotation should reduce pathogen populations in the field.

Resistant cultivars — The Spartan cultivars Delite, Delux, Fancy, Bonus, Classic, Winner and Premium are tolerant to cercospora leaf blight.

Chemical control — Carrot seed should be treated with a fungicide before sowing. Growers should consider initiating foliar fungicide sprays 1) after the eight-leaf stage; 2) when degree-days reach 550 above a base of 7°C; 3) 48 days after sowing; or, preferably, 4) when disease incidence in the crop reaches 50% of plants with middle leaves diseased. After the first spray, subsequent applications should be made at 7- to 10-day intervals, provided the temperature is above 16°C and wet periods extend longer than 12 hours. Urea can be mixed with the spray at the end of the season to stimulate foliage production and thereby aid mechanical harvesting.

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(Original by A.C. Kushalappa)

▶ 6.10 Crater rot *Fig. 6.10*

Rhizoctonia carotae Rader (teleomorph *Athelia arachnoidea* (Berk.) Jülich)

Crater rot occurs sporadically as a storage disease of carrot in North America. In Denmark, this disease has caused losses of 50 to 70% in stored carrots, and severe outbreaks have been reported in the United States. Carrot is the only natural host for *R. carotae.*

Symptoms The characteristic symptoms of crater rot are dry, sunken root lesions *(6.10)* lined with white cottony mycelium. Symptoms of the disease are not evident when the carrots are harvested, and in storage symptoms usually take two to three months to develop. The first signs of infection are small, white hyphal knots on the root surface. Small craters develop under the knots and then enlarge rapidly. At advanced stages of disease development, all the carrots in a pallet box may be covered with a weft of white mycelium; at that stage the disease may resemble Sclerotinia rot. Microscopic examination of the mycelium may be necessary to confirm the pathogen involved.

Causal agent The vegetative hyphae of *Rhizoctonia carotae* are 3.5 to 6.0 pm in diameter, hyaline, and have the characteristic branching pattern of *Rhizoctonia spp*. Side branches emerge at acute or right angles to the main hyphae, a septum is present in each side branch near the point of origin, and the hyphae constrict at the point where each side branch originates. Clamp connections are present and hyphal anastomosis is common. Dolipore septa are present and hyphal tips have an average of four to five nuclei per cell.

The pathogen can be isolated from sections of carrot root removed from lesion margins, surface sterilized and plated onto acidified potato-dextrose agar. Colonies form after 10 to 15 days of incubation at 20 to 24°C with a 12-hour photoperiod. Colonies measure 1.5 to 2 cm in diameter after 10 days under these conditions. The optimum temperature range for growth is 16

to 20°C. Colonies are slow-growing, white and interspersed with small hyphal aggregates. The morphological characteristics of different isolates may vary considerably.

In older cultures, crystals of calcium oxalate are formed along the hyphae. *Rhizoctonia carotae* produces oxalic acid in V-8, malt, potato-dextrose and carrot agar, and in liquid salts media with glucose or pectin as a carbon source and ammonium phosphate or asparagine as the nitrogen source.

Disease cycle Crater rot is primarily a disease of stored carrots. The pathogen is a soil-inhabitant and indications are that it can survive indefinitely in soil. Infections that develop in storage probably occur in the field, although symptoms are rarely seen at harvest. Infested pallet boxes may also be a source of inoculum. When carrots are inoculated with R. carotae, the hyphae grow over the root within a few days and appear to penetrate it without forming appressoria or other specialized infection structures. Cells are killed in advance of hyphal penetration.

The fungus can grow at temperatures as low as -1°C. Disease development and spread are accelerated by high relative humidity and especially by the presence of moisture on the root surface. Once the first symptoms become apparent on carrots in storage, the disease can develop rapidly. A carrot can be rendered unmarketable in less than three weeks.

Management

Cultural practices — Clean cultivation, weed control and wide spacing of plants help to reduce the level of field infection by allowing the soil surface to dry more quickly. Delaying harvest to late autumn may accentuate disease development in storage. Carrot roots should be carefully harvested to avoid cuts and bruises. If the disease has developed in storage, containers and handling equipment should be disinfested before reuse.

Proper storage management is the most important means of controlling this disease. Roots should be cooled quickly and stored close to 0°C. The relative humidity should be kept below 95% and moisture should not be allowed to condense on the carrots. It is advisable to regularly circulate outside air through the storage area.

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(Original by M.R. McDonald)

▶ 6.11 Crown rot (rhizoctonia canker) *Figs. 6.11a-c*

Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Crown rot occurs sporadically and occasionally causes economic losses. It is sometimes referred to as “cavity spot,” which creates confusion with the disease caused by *Pythium* species. Crown rot occurs most frequently on organic soils, especially those that have been cropped to carrot for many years. *Rhizoctonia solani* also causes damping-off of carrot seedlings, which can considerably reduce yield. The fungus has a very wide host range that includes many vegetable crops.

Symptoms Although the pathogen can cause damping- off of seedlings, it is usually more damaging on roots of larger carrots. Mid-season infections may continue to cause rot during storage. The earliest symptoms of crown rot are horizontal dark brown lesions that develop at the points where lateral roots emerge from the tap root *(6.11a).* These lesions may penetrate several millimetres into the tap root. This distinguishes them from the cavity spot lesions caused by *Pythium* spp., which are much shallower and develop over the entire root. The crown rot lesions are more numerous on the upper portion of the root. The disease may also appear as a band of dark brown, dry rot around the crown *(6.11b).* The outer leaves of diseased roots wilt and die, leaving the plant with only a few enlarged inner leaves that remain upright *(6.11c).* When infected roots are pulled, they have masses of soil and mycelium adhering to them.

Causal agent (see Bean, rhizoctonia root rot, 15B.7) Studies in the United States have shown that most isolates capable of causing damping- off and crown rot of muck-grown carrots belong to anastomosis group AG-2. The pathogen can be isolated from carrots using the techniques described for *Rhizoctonia carotae* (see crater rot, 6.10).

Disease cycle The pathogen is found in many types of soil and can survive for many years (see Bean, rhizoctonia root rot, 15B.7). Crown rot and damping-off are most severe at 20 to 28°C, with little infection or disease development below 16°C. Soil moisture levels above field capacity (near -0.1 bar) are optimal for disease development. Carrot plants of all ages are uniformly susceptible to *R. solani,* although crown rot is more severe on older plants.

Management

Cultural practices — There are few control measures for this disease. If practical, roots with crown rot symptoms should be culled before storage. The effectiveness of long crop rotations is doubtful.

Chemical control — Carrot seed should be treated with a recommended fungicide to reduce the incidence of damping-off.

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(Original by M.R. McDonald)

▶ 6.12 Fusarium dry rot *Fig. 6.12*

Fusarium acuminatum Ellis & Everh.
Fusarium avenaceum (Fr.:Fr.) Sacc.
Fusarium equiseti (Corda) Sacc.
Fusarium oxysporum Schlechtend.:Fr.
Fusarium redolens Wollenweb.
(syn. *Fusarium oxysporum* var. *redolens* W.L. Gordon)
Fusarium solani (Mart.) Sacc.

Fusarium dry rot is a storage disease of carrots. It has been an infrequent problem in most carrot-producing areas of Canada. The *Fusarium* species that cause dry rot have a wide host range.

Symptoms Root symptoms incited by the various species of *Fusarium* can be differentiated. In general, they all produce a crown rot and cankers of varying size on the sides of roots (6.72). The affected tissues are slightly discolored, dry and crumble easily. Later, after drying, the side cankers become hard and mummified. Often, however, crown decay progresses to affect the entire root. In other cases, sunken lesions varying from a few millimetres to several centimetres in diameter have been observed. These lesions are lined with white to yellow-tan or reddish mycelium, usually with a somewhat granular appearance due to spore production.

Causal agent Various criteria are used in identifying species of *Fusarium.* These include the presence or absence of microconidia and chlamydospores, the shape of the macro- and microconidia, method of conidial production, and cultural characteristics. Taxonomic keys (see Selected references, Booth, Nelson *et al.,* and Toussoun and Nelson) should be consulted for identification to species level.

The fungi are easily isolated from root tissue by surface sterilizing the material and placing it on potato-dextrose agar or other suitable agar media. Spore formation is stimulated by use of natural substrates or various culture media. Macroconidial formation is stimulated and increased in many *Fusarium* cultures by growth under fluorescent lights. Production of disease symptoms may be encouraged by maintaining carrot roots in a humid environment at 16 to 20°C for two to three weeks.

Other organisms are frequently associated with fusarium dry rot. These include *Gliocladium* spp., *Penicillium* spp., *Botrytis cinerea* and *Mucor* spp. The characteristic dry decay of fusarium dry rot may be masked by these secondary organisms. Cankers caused by *Fusarium* spp. that have dried out are nearly indistinguishable from those caused by *Gliocladium* spp. The presence of *Rhizoctonia* hyphae serves to distinguish the crater rot and crown rot diseases from fusarium dry rot.

Disease cycle *Fusarium* spp. occur widely in the soil on below-ground and aerial plant parts, crop residues and other organic matter. They may persist as resistant or dormant hyphae in plant residues colonized parasitically or saprophytically, or as chlamydospores and resistant conidia. *Fusarium avenaceum* and four other species of *Fusarium* have been found on carrot seed in Canada.

In storage, fusarium dry rot may become severe between 15 and 20°C, although it has been observed on carrots stored between 6 and 35°C. Infection occurs from 7 to 20°C with the optimum between 16 and 20°C. In cold storage, fusarium dry rot seldom appears before three months have elapsed. The disease can spread from diseased to healthy roots in storage by mycelial contact or air-borne spores.

Management

Cultural practices — Storage temperatures should be no higher than 1°C. To reduce losses from this disease, the roots should be kept dry and the relative humidity below 95% but above 90% (to prevent shrinkage) by the use of proper ventilation. Pre-storage washing and grading of carrots can markedly reduce losses from storage decay.

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(Original by R.F. Cerkauskas)

▶ 6.13 Pythium root dieback *Figs. 6.13a,b*

Pythium coloratum Vaartaja
Pythium irregulare Buisman
Pythium sulcatum Pratt & Mitchell
Pythium sylvaticum W.A. Campbell & J.W. Hendrix
Pythium ultimum Trow

Pythium root dieback is a disease complex that is incited by one or more pathogenic *Pythium* species. The disease has also been referred to as rusty root, lateral root dieback and forked root. However, pythium root dieback is considered a more descriptive and appropriate name for the disease.

During the 1960s and 1970s, substantial damage to carrots grown on organic soils in North America was attributed to this disease. More recent reports have noted reductions in marketable yield of up to 80% in carrots grown on mineral soils in California. In Canada, the incidence of pythium root dieback in carrot grown on organic soils has been low in recent years. This may result from the wide availability of resistant cultivars and the adoption of precision-seeding methods.

Pythium root dieback, or disorders with similar symptoms, have been reported in Canada, the United States, Norway and the Netherlands. The *Pythium* species that cause root dieback on carrot are also capable of infecting a number of other vegetable crops. *Pythium irregulare, P. sulcatum* and *P. sylvaticum* also cause root dieback of celery, lettuce, parsnip and radish, and stunting of the tops in beet, celery, parsnip, potato, radish and tomato. Oogonia, sporangia and other structures of *Pythium* have been found in corn, cucumber, potato and lettuce. *Pythium* has also been isolated from onion. Root dieback symptoms occur on the roots of lamb's-quarters *(Chenopodium album* L.), pineapple weed *(Matricaria matricarioides* (Less.) Porter) and purslane *(Portulaca oleraceae* L.), and *Pythium* has been observed in the roots of pineapple weed and smartweed *(Polygonum* spp.).

Symptoms Carrots affected by pythium root dieback have numerous, rusty-brown lateral roots. Sometimes, the tap root is stunted and surrounded by many long lateral roots *(6.13a).* In other instances, it is larger, but stunted or forked *(6.13b).* The foliage usually looks healthy but occasionally may appear stunted or wilted. Severely affected seedlings may wilt and die. Older plants may recover by forming an abundance of lateral roots, but such plants usually produce poor quality tap roots.

Causal agent The hyphae of *Pythium* species are hyaline and aseptate, except for old hyphae. Septa are found at the base of reproductive structures. Cytoplasmic streaming can be seen in young hyphae. Most species do not produce aerial mycelium on cornmeal or potato-carrot agar, but many form cottony aerial mycelium on oatmeal agar. *Pythium ultimum* produces thick, cottony mycelium on commeal agar.

Identification of *Pythium* spp. is based on the morphology of the sporangia, conidia, oogonia and antheridia. The presence, size, shape and number of these structures varies considerably, depending on culture media, age of the culture, temperature and other environmental conditions. Standardization of the methods used to culture these fungi is very important for accurate identification (see cavity spot, 6.8, for a detailed description of the *Pythium* species described above; see also Selected references, Van der Plaats-Niterink, 1981).

To isolate *Pythium* spp. from diseased carrot roots, wash the roots in running tap water and plate onto PVPP agar (17 g of Difco cornmeal agar, 5 mg of pimaricin, 250 mg of vancomycin, 50 mg penicillin and 100 mg pentachloronitrobenzene per litre of water) and incubate in the dark at 20 to 26°C for 24 to 28 hours. Transfers from the colony edge can then be made to other media, such as cornmeal agar, for identification. The method outlined for isolating *Pythium* spp. from cavity spot lesions can also be used.

Stunting, forking and proliferation of lateral roots in carrot can also be caused by other factors, including root-knot nematodes, soil compaction and saturation, root-feeding insects, and mechanical injury. *Olpidium* and tobacco necrosis virus, as well as *Alternaria***,** *Cylindrocarpon***,** *Gliocladium* and *Fusarium* species isolated from carrot roots, have been investigated for their possible role in causing root dieback, but pathogenicity tests demonstrate that none is the primary cause of root dieback.

Attempts to correlate root dieback with use of the herbicide linuron and with high soluble salt concentrations in the soil also have had negative results.

Disease cycle In the field, the primary root of the carrot is infected within the first weeks of growth and root tip necrosis can be observed after the two-leaf stage. In controlled- environment studies using naturally or artificially infested soils, root necrosis is evident 21 days after seeding.

The symptomatic root branching and lateral root proliferation occur when injury to the primary root destroys apical dominance. The lateral roots of infected plants are often rusty-brown or have rusty-brown lesions, indicating that infection may take place throughout the growing season.

The *Pythium* species that cause root dieback are common soil inhabitants in North America and may persist indefinitely in fields. In organic soils, the severity of root dieback has been positively correlated with soil moisture levels and total *Pythium* populations. In studies with mineral soils, there has been no correlation between the severity of root dieback and population densities of *P. ultimum* and *P. irregulare.* Many *Pythium* species are active at matric potentials below field capacity (-0.3 bars), making it unlikely that moisture levels in organic soils limit their growth.

The optimum temperature for disease development varies. Carrot plants grown in sand infested with *P. ultimum* and maintained at a soil moisture potential of -2.5 kPa have significantly more forked roots at 23°C than at 27°C. *Pythium ultimum, P. aphanidermatum* and *P. irregulare* are known to kill more carrot seedlings at 35°C than at 25°C.

Management

Cultural practices — Carrot should not be planted in fields that are poorly drained or prone to flooding, and care should be taken not to over-irrigate young crops. Growing carrot on raised beds reduces the incidence of root forking and improves the percentage of marketable carrots. Also, precision seeding has been shown to reduce the incidence of root dieback. Crop rotations with cabbage, corn, mint, onion and potato may reduce the incidence of pythium root dieback in subsequent carrot crops.

Resistant cultivars — Several commercial cultivars are available that have a high degree of tolerance to pythium root dieback. These include Waltham Hi-Color, Hi-Color 9, Paramount, Spartan Fancy, Canada Super X, Gold Pak 28C and Orlando Gold. Growers should consult provincial recommendations for a more complete list.

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(Original by M.R. McDonald)

▶ 6.14 Rubbery brown rot *Fig. 6.14*

Phytophthora porri Foister

Rubbery brown rot is a disease that affects carrot in storage and transit. Losses of up to 20% in stored carrots have been reported. The disease has been observed sporadically in Alberta and British Columbia. *Phytophthora porri* infections of carrot have also been reported in Tasmania and New York State.

Phytophthora porri is known to cause white-tip of leeks. It is also capable of attacking gladiolus, onion, scallion, tulip, stored cabbage and a number of ornamental flowers. Other vegetable species are susceptible when inoculated with this fungus, but beet, parsnip and celery are not susceptible.

Symptoms There are no visible symptoms of rubbery brown rot in the field or when the carrots are first harvested. The disease becomes apparent after the roots have been in storage for some time. Infected carrots develop dark brown, firm, water-soaked areas that may appear anywhere on the root, but most often are found near the middle or crown area. The rot is sometimes present in wide bands. A dense growth of white surface mycelium may also be present *(6.14).* As the decay worsens, the roots turn darker and have a moist glistening surface. The roots tend to collapse easily and the interior portions are brown and rubbery but not wet. Secondary infections are often involved in the later stages of the disease.

Causal agent *Phytophthora porri* has branched, aseptate mycelium when young, which becomes empty and septate in old colonies. On V-8 agar, the colonies form dense aerial mycelium. The margins are smooth to slightly irregular and growth is slow,

about 3.5 mm per day at 25°C. The cardinal temperatures for growth in culture are: minimum 0°C, optimum 15 to 20°C, and maximum 31 to 32°C.

The young hyphae are straight and smooth but soon become irregular, knobby and looped. Hyphal swellings are common and may form singly or in chains. The sporangia are terminal or intercalary, average 50 by 41 pm and are non-caducous. The apex is broad and slightly papillate with a shallow thickening. Many chlamydospores are produced singly or in chains. The oogonia are spherical, colorless and 28 to 37 pm in diameter. The antheridia are predominantly amphigynous, but some are paragynous. Oospores are aplerotic.

The pathogen can be isolated by breaking apart the edges of discolored areas on infected roots and aseptically transferring the underlying tissue to an antibiotic-containing medium such as PVPP agar (see pythium root dieback, 6.13). The fungus can be maintained on cornmeal agar. The production of oospores can be induced by long-term storage on mature barley, wheat or wild oat straw.

Disease cycle The disease has been observed on carrot grown under irrigation and from fields that have received prolonged heavy rainfall during the growing season. Symptoms develop on inoculated carrots from 0 to 20°C. They develop within one week at 20°C, whereas at 0°C, some darkening is observed after seven weeks of incubation and typical symptoms are not seen after 13 weeks. The pathogen can spread by direct contact from diseased to healthy carrots in storage or in transit.

Management

Cultural practices — It is advisable to grow carrot on well-drained soils and to avoid over-irrigation. Carrots should be stored at 0°C and below 95% relative humidity. Infected roots should be culled, if practical. Pallets and storages should be cleaned and disinfested between crops.

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▶ 6.15 Sclerotinia rot (white mold) *Figs. 6.15a,b*

Sclerotinia sclerotiorum (Lib.) de Bary (syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia rot is the most destructive disease of stored carrot and can also affect crops in the field. It can cause damping-off and infection of the petioles that may later spread to the leaves and crown. Foliar infections can reduce yields by weakening the tops so that the carrots cannot be mechanically harvested. *Sclerotinia sclerotiorum* affects many vegetables, including lettuce, celery, bean and cruciferous crops, as well as numerous weeds; the disease is often known as white mold.

Symptoms In the field, foliar infection occurs at the base of the petioles and the fungus spreads rapidly, killing the leaves. Infected foliage is dark brown and often covered with the white, cottony mycelium that is characteristic of *Sclerotinia.* Some time after infection and death of the leaf tissue, black sclerotia may also appear amid the cottony mycelium. Infection of the leaves and petioles is usually accompanied by infection of the crown. Infected roots are often symptomless at harvest, but disease develops in storage.

Sclerotinia causes a soft, watery rot on stored carrots. Infected tissue darkens, turns grayish and is soon covered with white cottony mycelium *(6.15a).* The formation of black sclerotia, 1 to 2 cm long, amid the mycelium *(6.15b)* distinguishes lesions caused by *Sclerotinia* from those caused by *Rhizoctonia* or *Fusarium* spp. Bacterial soft rot also occurs on carrots in storage but is slimy and lacks the surface mycelium.

Causal agent (see Bean, white mold, 15B.9)

Disease cycle (see Bean, white mold, 15B.9) Primary infection probably results from colonization of leaf and stem tissues by mycelium produced from sclerotia in the soil. Root infection may take place after the foliage and crown become infected. Direct infection of roots by mycelium in the field has been postulated but appears unlikely. Ascospores may infect senescent or damaged tissue under conditions of sustained high humidity. Disease development in storage usually starts from infections that have occurred in the field or at harvest. However, infection originating from inoculum on used or dirty pallet boxes has also been reported. The optimum temperature for disease development is 13 to 18°C, but disease will develop provided temperatures are above 0°C. Free moisture and a relative humidity greater than 92% also contribute to disease development. Once infection is established, the infected tissue usually provides enough moisture for further development.

Mycelium from a single infected carrot can spread to adjacent carrots, producing radiating pockets of infection (nesting) on roots stored in pallet boxes, plastic bags or in bulk storage. Secondary bacterial soft rot may follow sclerotinia rot.

Management

Cultural practices — Growers should rotate carrot with non-host crops such as onion, beet, spinach, cereals and corn for three to five years to reduce the level of soil- borne inoculum. This practice must be accompanied by good weed control. A heavy infestation of weeds can contribute to disease development by increasing the relative humidity and duration of leaf wetness within the canopy.

Growing carrot on ridges or raised beds may reduce the incidence of foliar infection by allowing increased air circulation and thereby decreasing the duration of leaf-wetness periods. Flooding of fields between crops can also reduce the numbers of viable sclerotia in the soil. Rapid cooling of harvested carrots and storage at a constant 0°C are critical factors in reducing disease development in storage.

Resistant cultivars — Six Pak II is more susceptible to storage decay than Paramount and Dess Dan.

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(Original by M.R. McDonald)

▶ 6.16 Violet root rot *Fig. 6.16*

Rhizoctonia crocorum (Pers.iFr.) DC.
(syn. *Rhizoctonia violaceae* Tui. & C. Tui.)
(teleomorph *Helicobasidium brebis soni i* (Desmaz.) Donk)

Violet root rot is endemic but economic damage occurs in only a few production areas. The fungus is widely distributed throughout northern Europe and the United States. In Canada, its distribution is not limited to a particular soil type. The disease often occurs in one or two areas of a field. Affected carrots are unmarketable.

The fungus is known to infect other vegetable crops, including asparagus, bean, beet, cabbage, parsley, parsnip, potato, rhubarb, sea kale, sweet potato and turnip. It has also been isolated from alfalfa, clover and rapeseed, and weeds such as yarrow (*Achillea millefolium* L.), quackgrass (.*Agropyron repens* L.), sweet vernal grass (*Anthoxanthum odoratum* L.), thistles (*Cirsium* spp.), silverweed (*Potentilla anserina* L.), creeping buttercup (*Ranunculus repens* L.), sheep's-sorrel (*Rumex acetosella* L.), dock (*Rumex* spp.), and dandelion (*Taraxacum officinale* Weber).

Symptoms Foliar symptoms of the disease may be detected in the field in mid-summer to early fall. The leaves of affected plants become chlorotic, wilt and eventually die. Roots become covered with an external mat of mycelium and spores which initially is pale buff to violet but which gradually turns red-violet and finally purple-brown *(6.16).* The mycelial mat contains numerous papillae that are slightly darker than the rest of the mycelium and resemble sclerotia. Lesions enlarge and grow together as the disease progresses to cause an overall decay. At this stage, the affected areas have a firm, leathery covering, but the underlying tissues are soft and rotted. The disease may develop to this extent in the field. Shallow lesions, which may be present at harvest, enlarge during storage.

When infected carrots are pulled from the ground they usually have a mass of soil clinging to them. *Rhizoctonia crocorum* can also grow from plant to plant as a thick brown mycelial mat on the soil surface. These mats have been reported to be up to 30 cm long and 15 cm wide.

Causal agent *Rhizoctonia crocorum* mycelium is branched, septate and spreads evenly over the surface of the host. The hyphae branch at right angles with a septum not more than 10 pm from each junction. The mycelium aggregates into papillae that vary in size from a few millimetres to several centimetres. The papillae are rounded, flattened, appear to be covered with a thick velvety felt, and function as infection cushions where the fungus penetrates the host tissue.

The basidial stage *Helicobasidium brebissonii* is found only in the spring. Curved basidia are formed directly on the mycelium, which forms a purplish hyménium. The basidia are hyaline, septate, and produce two or three sterigmata, 10 to 35 µm long, which carry hyaline basidiospores. The basidiospores vary in shape from oval to reniform, and measure 10 to 12 by 6 to 7 µm.

The pathogen can be isolated from carrot roots by following the technique recommended for *Rhizoctonia carotae* (see crater rot, 6.10).

Disease cycle The pathogen is soil-borne. It spreads very slowly as the mycelium grows through the soil from plant to plant. The major means of spread within and between fields is by infested soil on farm implements and by infected plants.

Infection and disease development occur slowly. In culture, *R. crocorum* grows between 9 and 39°C, with an optimum of 26°C. Infection of carrot takes place between 5 and 30°C, with an optimum of 20°C. Carrot plants are usually infected in the

spring and it may take several months for foliar symptoms to appear. Experiments in Britain indicate that the number of infected carrots increases the longer they are left in the ground. High soil moisture levels and low pH increase the severity of violet root rot.

Management

Cultural practices — The main control is to avoid planting carrot in infested fields. Crop rotation with grasses and cereals, combined with good weed control, may reduce the inoculum level in the soil. Good soil drainage, proper fertilization, and liming to increase the pH may also help to reduce the level of root infection.

The crop should be harvested as early as possible if the disease is detected in the field. Care must be taken to prevent the spread of infested soil on farm machinery to uninfested fields. Growers should not return diseased plant material to the soil; it should be disposed of away from agricultural land.

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(Original by M.R. McDonald)

VIRAL-LIKE DISEASES

▶ 6.17 Aster yellows *Figs. 6.17a-c*

Aster yellows mycoplasma-like organism

Aster yellows is a common disease of carrot, but in most carrot-producing areas it is of minor concern. In Ontario and Quebec, the prevalence of aster yellows varies from year to year, but it generally affects less than 2% of the carrot hectarage. It occasionally causes economic losses, but the cost of controlling the leafhopper vector may be greater than the value of the crop lost. The pathogen has a wide host range than includes many vegetable crops.

Symptoms The first field symptom is leaf yellowing, with some vein clearing of the younger leaves at the center of the crown *(6.17a).* Later, a mass of sickly new shoots grows from the crown, giving a witches'-broom appearance to the top. Older leaves are whitish at first, then become bronze, reddened or both. The reddened leaves are distinctive and are easily recognizable in the field *(6.17b).*

The petioles are twisted and eventually break off leaving a short bunchy top unsuited for mechanical harvesting or for bunching the carrots for the fresh market. Many malformed fibrous rootlets usually appear in rows along the vertical axis of the main root *(6.17c).* The color, texture and flavor of roots may be altered. The crowns of diseased plants are subject to bacterial soft rot in wet weather. The disease can continue to develop in storage. On seed plants, various degrees of stunting occur, as well as malformation, chlorosis and sterility of the flowering umbels.

The severity of aster yellows and the injury to the carrot crop depend on the age of the crop when infection occurs and the length of time the disease has to develop before harvest. The disease is most severe on late-harvested crops.

Causal agent (see Lettuce, aster yellows, 11.15)

Disease cycle (see Lettuce, aster yellows, 11.15)

Management (see also aster leafhopper, 6.22, 11.23)

Monitoring — In Quebec, symptoms of aster yellows on carrot are rated on a scale of 0 (no symptoms), 1 (symptoms rarely seen), 2 (symptoms every 10 paces), 3 (symptoms every 5 paces), or 4 (symptoms every pace). At a level of 4, it is usually best to obtain a more accurate measure of the incidence of the disease to predict the potential level of crop losses.

Cultural practices — It is important to control weeds on which the aster yellows organism can survive, and to avoid planting carrot near fields of lettuce or other susceptible crops. All residues from susceptible crops must be destroyed immediately after harvest.

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Crête, R. 1980. *Diseases of Carrots in Canada.* Agric. Can. Publ 1615/E. 26 pp.

(Original by R. Crête and G. Boivin)

NON-INFECTIOUS DISEASES

▶ 6.18 Growth cracks *Fig. 6.18*

Growth cracking is the result of fluctuating soil moisture levels throughout the growing season. The growth and expansion of the carrot root are retarded when the soil is dry. If a dry period is followed by a heavy rainfall, growth resumes and the carrot root may expand so rapidly that it splits. Growth cracks occur occasionally on carrot grown under conditions of regular rainfall and moderate soil moisture levels, suggesting that other factors may be involved that have not yet been identified.

Growth cracks occur sporadically in carrot, but rarely is the incidence high enough to affect the marketable yield. In a normal harvest, a 20% grade-out due to crooked, broken or misshappened carrots is not unusual, so a 5% incidence of growth cracks would not be a concern. Occasionally, however, the incidence can be 30 to 50%.

Symptoms Growth cracks on carrot roots appear as vertical cracks, which may vary in length from less than a centimetre to a split down the entire length of the root *(6.18).* Usually there is no apparent lesion, rot or insect damage associated with the crack and the tissues around the crack appear healthy. Some short vertical cracks may be associated with cavity spot lesions, but these are not considered growth cracks. Growth cracks have been observed on all sizes of carrots but usually are more common on larger roots. Growth cracks that have been present on the root for some time may have a layer of rough, suberized tissue over the interior of the crack. The cracks may provide an entry site for soil-borne pathogens such as *Sclerotinia*, *Rhizoctonia* and *Fusarium* spp., and soft rot bacteria.

Management

Cultural practices — To reduce growth cracks, carrot should be grown in soil that is well drained but has a good moisture holding capacity. If carrot is grown under irrigation, regular watering helps to prevent moisture stress and growth cracks.

(Original by M.R. McDonald)

▶ 6.19 Heat canker *Figs. 6.19a-c*

Heat canker results when the carrot root tissue at or near the soil surface is injured and killed by high temperatures. On sunny days, surface temperatures can reach 50 to 65 °C. Heat canker can cause considerable losses in carrot seedlings and may also cause injury at later stages of growth. It can occur on carrots growing on both mineral and organic soils, but dark-colored soils are the most prone to surface heating.

Symptoms In seedling carrot, heat will cause the tissue at or near the soil surface to collapse and die. The top of the plant often falls over or breaks off and the seedling dies *(6.19a).* If the plant is larger when affected, only the cells of the cortex are killed. These shrivel, discolor and form a constriction near the top of the root *(6.19b).* The vascular tissue may remain alive, and consequently the tops continue to grow. Depending on the severity of the injury, the top may eventually break off, or the root may die if nutrient flow in the phloem is interrupted. Other carrot plants may survive, but the injury usually renders them unmarketable *(6.19c).*

Management

Cultural practices — Control of heat canker depends on avoiding or preventing excessive heating of the soil surface. Seeding carrots early in the spring when the soil is moist and cool often avoids the problem. Using overhead irrigation to cool the soil surface and provide moisture to the seedlings has been tried with variable success. Increasing plant density so that the seedlings help to shade the soil can be effective. Broadcast seeding of a cover crop such as barley or spinach to shade the soil and reduce wind erosion has been successful. Leaving weeds to provide shade is another option. Timely removal of the cover crop or weeds with a selective herbicide is essential if either of these approaches is used.

Selected references

Crête, R. 1980. *Diseases of Carrots in Canada.* Agric. Can. Publ. 1615E. 26 pp.

(Original by M.R. McDonald and R. Crête)

NEMATODE PESTS

▶ 6.20 Northern root-knot nematode *Figs. 6.20; see text*

- *Meloidogyne hapla* Chitwood

This nematode attacks almost all types of vegetable crops commonly grown in gardens, fields and greenhouses in Canada. It survives and develops at lower temperatures than do *Meloidogyne incognita* (Kofoid & White) Chitwood, *M. javanica* (Treub) Chitwood, and *M. arenaria* (Neal) Chitwood, the major root-knot nematode pests found at southern latitudes. When introduced, these latter three species can infect and persist in greenhouse crops in Canada. Diseased transplants, infested soil, and culled roots

and tubers are sources of inoculum. Many greenhouses and other areas have been infested with root-knot nematodes by planting infected tomato, celery or pepper transplants.

Symptoms Because of the occurrence of the northern root-knot nematode on almost all types of vegetables grown in Canada, the symptoms on a wide range of crops are presented here.

Root and tuber vegetables (carrot, ginseng, parsnip and potato) — When the density of nematodes in soil is high, there may be areas within fields with missing or stunted plants. Leaves usually appear healthy, although they may be smaller and lighter colored than normal. A reddish tinge may appear on the back of leaves while they are still green. Older leaves often turn yellow and dry prematurely. Infected plants usually senesce early in the season. A few weeks after planting, small swellings and branches may be visible on the lateral roots, even before the tap roots start to size. Tap root development is delayed and mature roots are deformed, short and branched or knobby. Secondary roots are often abnormally branched and hairy *(6.20).* There may be numerous root swellings, from which small rootlets originate. Marketable yields are reduced considerably because of the poor appearance of tap roots or tubers, rather than by a direct weight loss. In potato *(16.35),* root-knot nematodes penetrate the root and tuber lenticels. Scab-like lesions on the skin of tubers may render them unmarketable.

Leaf vegetables (celery, lettuce, rhubarb and spinach) — Symptoms vary depending on the density of nematodes in the soil at planting time. With heavy infestations, affected plants wilt, turn light green and progressively yellow. Roots show numerous small swellings from which adventitious rootlets grow, producing increased branching that which can result in a bushy appearance. The swellings resemble the root nodules formed by root nodulation bacteria *Rhizobium* spp. on legumes, except that nematode galls are spherical and never elongate or colored. The increased branching makes the root systems of diseased plants look more developed than those of healthy plants. In celery and spinach, growth reduction is expressed as a yellowing and stunting of stalks and leaves; in head lettuce, growth reduction is expressed as a delay in maturation or lack of head formation.

Seed vegetables (broad bean, green bean, snap bean, and pea) — Symptoms on the foliage and roots are similar to those on leaf vegetables. In addition, there is often poor flower set, resulting in fewer and smaller fruits and seeds.

Fruit vegetables (cucurbits, eggplant, pepper and tomato) — These vegetables are highly susceptible to the northern root-knot nematode *(18.30).* Infected plants are stunted and show conspicuous symptoms of foliar chlorosis and early senescence. Flowers and sets are affected and fruits are usually fewer and smaller than those of healthy plants. These crops also are grown in greenhouses, where damage can be substantial. In contrast to *Meloidogyne hapla*, the southern root-knot nematode *M. incognita* causes root galls that are usually compound, large and conspicuous *(22.30a-d, 25.26);* tomato plants infested with *M. incognita* sometimes also show purpling of the undersides of leaves, resembling phosphorus deficiency.

Cruciferous crops (broccoli, Brussels sprouts, cabbage, cauliflower, kale, turnip and rutabaga) and Swiss chard — These crops are tolerant or resistant to northern root- knot nematodes and sustain relatively less damage than most other vegetables. Crops with resistance have very small galls that may be hard to recognize. With heavy root-knot nematode infestations, there is a loss of yield and a delay in maturity.

Bulb vegetables (onion, garlic, leek and shallot) — Foliar and root symptoms are similar to those on leaf vegetables. The nematodes infect the roots but not the bulbs. Bulb vegetables are generally quite sensitive to root-knot nematode infestation. Onion is sometimes planted in rotation with carrot because it sustains less damage than carrot from relatively low populations of this nematode.

Identification *Meloidogyne hapla* (order Tylenchida, family Heteroderidae) has a delicate cephalic framework and stylet in both the motile second-stage juvenile and adult female. There is marked sexual dimorphism. Males are migratory, long and robust, with a short round tail. Females are sedentary, globose and stay in the roots. Annulations of the cuticle around the genital opening (vulva) and anus of the mature female form a pattern that is useful in identification.

Life history Nematodes are attracted by root secretions and migrate toward roots soon after seed germination and root elongation. Second-stage juveniles penetrate the root tips. They position themselves with their head in the vascular tissue and induce the formation of giant cells upon which they feed. The juveniles enlarge considerably, undergoing three molts. Migration of these parasites through the cortex and the establishment of feeding sites in the vascular tissue cause changes in root morphology. The root tissue increases in size through hypertrophy and enlargement (hyperplasia) of vascular parenchyma cells, resulting in small swellings, knots or galls. At each gall, and especially at the root tips, nematode development causes the roots to branch, giving them a matted, bushy appearance.

Females become so large that they often protrude from the gall. At soil temperatures around 20°C, several hundred eggs are produced by each female within a few weeks. The eggs are laid at the surface of the gall in dark brown, gelatinous egg masses the size of a small pin head, which can be seen with the naked eye. Infective second-stage juveniles develop in approximately two weeks. They can reinfect newly formed roots and form additional galls.

Management

Monitoring — *Meloidogyne hapla* reproduces quickly and, by mid-season, medium to high densities of juveniles in soil or eggs on roots (500 to several thousand per 100 mL of soil or per gram of roots) usually develop. Low to medium densities of *M.*

hapla before planting generally mean that susceptible vegetable crops will suffer some damage. The damage threshold for carrot and parsnip is one or very few juveniles per 100 mL of soil, which approaches the limit of the detection level.

Cultural practices — Rotation with non-hosts such as cereals help to reduce populations of root-knot nematodes in soil. In small plantings and gardens, interplanting with marigolds *(Tagetes patula* L. and *T. erecta* L.), solarization and fumigation also are effective. See also Management of nematode pests, 3.12.

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(Original by T.C. Vrain)

▶ 6.21 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms on carrot include wilting and stunting in patches in heavy infestations; leaves become yellow. Tap root may be small and branched and slow to mature. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 6.22 Aster leafhopper *Figs. 11.23a,b*

Macrosteles quadrilineatus (Forbes)
(syn. *Macrosteles fascifrons* of authors, not Stål)

Aster leafhopper (see Lettuce, 11.23, for identification and life history) populations begin to develop on carrot when the migrant adults move away from winter cereals and early- seeded vegetables, such as lettuce. Further build-up occurs when the succeeding generation migrates from spring grains. The leafhopper feeds on carrot crops throughout the summer, declining in numbers only in the autumn.

Damage On carrot, the aster leafhopper feeds on the leaves but does not cause economical damage. However, the feeding adult can transmit the aster yellows mycoplasma-like organism (see aster yellows, 6.17).

Management In some years, leafhopper populations or the proportion of leafhoppers infected with aster yellows can increase, necessitating management to minimize economic damage from aster yellows.

Monitoring — Ideally, the need to apply an insecticide should be based on leafhopper numbers and the proportion of adult leafhoppers carrying the aster yellows pathogen. However, a practical method is not available for rapid evaluation of both leafhopper numbers and the proportion of leafhoppers actually carrying the pathogen. For this reason, there is no threshold for carrot crops. Monitoring, which is done with yellow sticky traps similar to those used for the carrot rust fly (3.2T1), helps growers to synchronize insecticidal treatments with increases in population density of the leafhopper. In Quebec, leafhoppers are monitored along with other pests of carrot and are noted on a scale from 0 (no leafhoppers) to 2 (large numbers of leafhoppers). In Ontario, monitoring traps reveal sudden increases in leafhopper numbers, which may indicate a need for control. Growers should monitor early and continue monitoring throughout the season to detect population build-ups.

Chemical control — The adult aster leafhopper on carrot can be controlled by an insecticide. Growers are advised to apply an insecticide if the density of leafhopper adults increases rapidly because of migration from recently harvested lettuce, alfalfa or hay fields, or if symptoms of aster yellows appear. No resistance to insecticides used on carrot has been reported for this leafhopper in Canada.

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(Original by G. Boivin)

▶ 6.23 Carrot rust fly *Figs. 6.23a-e; 3.2T1*

Psila rosae (Fabricius)

The carrot rust fly is a major pest in the principal carrotgrowing areas of eastern Newfoundland, Quebec, Ontario and British Columbia, and recently it has been identified in Alberta. It was introduced into Canada in 1885 but did not become a major pest until the 1940s. In Newfoundland, it was first observed at St. John's in the late 1930s, then spread to communities throughout the Conception Bay area and, by the late 1950s, to carrot-producing areas in the Bonavista Bay area. In Quebec, carrot rust fly was mentioned as early as 1908, but it only became important at the beginning of the 1980s, when it was present at low population levels in all carrot-growing areas. In Ontario, where this insect is a major pest of carrot, infested celery plants may serve as a reservoir for infestation of carrot later in the season. In British Columbia, carrot rust fly is the main insect pest of carrot in southern coastal areas, and a sporadic pest in the southern Okanagan and Kootenay areas.

The carrot rust fly attacks many umbelliferous plants. Carrot is the most important cultivated crop host, but celery, parsley and parsnip also are subject to attack. In British Columbia, carrot rust fly damage on plants other than carrot is of no economic importance. In Ontario, infestations on celery seldom justify treatment (see Celery, carrot rust fly, 7.19).

Damage Damage by the carrot rust fly is caused by the larvae *(6.23a).* They are attracted by carbon dioxide emitted by the carrot plant, and feed on the root radicles. Young carrot plants may die from damage to the radicles. Roots of older carrot plants may become forked, stunted, or fibrous because of these early attacks. Older larvae enter the main root and tunnel in the lower third, root portion *(6.23b,d).* In Quebec and Ontario, the first summer-generation matures before it can damage early carrots. Most damage is caused by the second summer-generation and, in British Columbia, also by the first and third summer-generations. Areas near shelter-plants are more likely to show damage, whereas carrot crops in open areas generally are not affected by this insect. The adult carrot rust fly does not transmit pathogens. However, bacteria and fungi can invade the carrot root through tunnels made by the larvae, and late-maturing larvae can cause important post-harvest damage to carrots in storage.

In eastern Newfoundland, the carrot rust fly is becoming more damaging on small farms and in garden plots. In Quebec, carrot rust fly populations and damage are increasing. In Ontario, this fly is already a major pest of carrot in the Bradford Marsh and Holland Marsh areas. The unreliability of results obtained with chemical insecticides and the lack of a commercially available method of biological control increase the importance of this insect as a pest. However, implementation of a monitoring program, as is being done in British Columbia, can reduce the amount of insecticide used against the carrot rust fly.

Identification Carrot rust fly (family Psilidae) adults (*6.23e*) are black, about 6 mm in length with a small, reddish head and long yellow legs. The larva *(6.23a)* is legless and cream-white with dark mouthhooks. The pupa (puparium) is cylindrical, about 4.5 mm in length, and red-brown *(6.23c).*

Life history The carrot rust fly overwinters as a pupa (puparium) in the soil to depths of 10 cm. Adults emerge in late April or early May in British Columbia, mid-May in Ontario, late May or early June in Quebec, and late June and early July in Newfoundland. These adults leave carrot fields and seek shelter-plants where they feed and mate. In the evening, females leave the shelter-plants to oviposit in carrot fields. The eggs are deposited on the ground. Young larvae feed on carrot root-radicles. Older larvae enter the main root, tunneling generally in the lower third of the root. At maturity, the larvae leave the carrot and pupate in the soil. In Newfoundland, there is only one generation per year, pupae (puparia) of which overwinter. In Quebec and Ontario, adults emerge from mid-August to mid-September; in British Columbia, from late July to mid-August. In Quebec, pupae of the second summer-generation overwinter. In some years in Ontario, a (partial) third summer-generation comprises all or most of the overwintering pupae (puparia). In British Columbia, the second summer-generation matures and breeds in mid-October, and the third summer-generation overwinters (in the pupal stage).

Management

Monitoring — Carrot rust fly adults are monitored with yellow sticky traps in Quebec, Ontario and British Columbia. These traps are clipped vertically to upright stakes, 5 to 10 cm above the carrot canopy and 1 to 2 m inside the field *(3.2T1).* These traps may be placed around the field at 100-m intervals, as is done in Quebec, or at a density of one to two traps per hectare, as in British Columbia. Areas that are protected from wind must be monitored carefully. Traps are serviced twice a week and carrot rust fly adults are counted. The traps are replaced when dirty, or every 7 to 10 days. Monitoring is done from mid-April to harvest in British Columbia, and from mid- August to the end of September in Quebec. In Quebec and Ontario, the economic threshold is 0.2 and 0.1 flies per trap per day, respectively. When captures exceed that threshold, the probability of damage is high. In British Columbia, the threshold is 0.25 flies per trap per day or captures between 0.1 and 0.25 flies per trap per day for more than a week.

Cultural practices — In the Holland Marsh and Bradford Marsh areas of Ontario, damage by first-generation larvae can be avoided by delaying the seeding of carrot until after mid-May. In British Columbia, growers are sometimes advised to mow the carrot tops and leave only enough stem to proceed with mechanical harvesting. This ensures better spray coverage on mature crops, improves ventilation, and also slows the growth and prevents oversizing of the carrots. In general, damage is confined to the edges of fields and near shelters, so field borders should be harvested earlier to remove the most vulnerable carrots before the larvae begin to enter the roots. In Quebec and Ontario, carrots harvested before early October escape most damage.

Growers should avoid planting carrot crops in high risk areas, such as near sheltered or humid areas, in the vicinity of wild or volunteer carrot and parsley, or near fields where there was significant damage the previous year.

Biological control — Two parasites of the carrot rust fly, *Dacnusa gracilis* (Nees) and *Loxotropa tritoma* (Thoms), have been imported into Canada. These were released in Ontario and British Columbia in the 1950s, and *D. gracilis* alone was released in 1986 in Quebec. Neither species has become established. The impact of parasites and naturally occurring microorganisms on populations of carrot rust fly is unknown. No biocontrol agent is commercially available.

Chemical control — A granular insecticide in the seed furrow is no longer used because the insecticide was subject to breakdown by soil microorganisms. Sprays of adulticides are recommended if fly numbers on traps exceed threshold values. In the absence of a monitoring program, treatments must be applied at 7- to 10-day intervals. Such treatments are of limited value because adults are present in carrot fields for only a short time; they migrate from nearby host reservoirs; there is resistance to insecticides; and, in British Columbia, cool temperatures in late summer and autumn reduce the effectiveness of certain insecticides. For best results, sprays should be applied in early evening when flies are present and active in the field. The carrot rust fly developed resistance to organochlorine insecticides in the early 1960s. Presently, one of the recommended organophosphates, diazinon, seems to be less effective in Ontario, but resistance has not been demonstrated definitively.

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(Original by G. Boivin)

▶ 6.24 Carrot weevil *Figs. 6.24a-d; 3.2T2*

Listronotus oregonensis (LeConte)

The carrot weevil is indigenous to North America and occurs in Manitoba, Ontario, Quebec, and Nova Scotia. Like the carrot rust fly, carrot weevil has been one of the major pests of carrot crops grown in organic soil in Quebec and Ontario since the beginning of the 1970s.

The carrot weevil attacks umbelliferous plants. In addition to carrot, celery, dill, parsley and parsnip are subject to attack. There are numerous wild umbelliferous hosts, such as wild carrot, wild parsnip and water parsnip *(Sium suave* Walt.); Polygonaceae, such as broad-leaved dock (*Rumex obtusifolius* L.), and curled dock (*R. crispus* L.); and Plantaginaceae, such as broad-leaved plantain *(Plantago major* L.), and narrow-leaved plantain (*P. lanceolata* L.).

Damage On carrot, the larvae of the carrot weevil cause economic damage by tunneling into the petiole, heart, and root of the plant. The tunnels of young larvae are small. Tunnels of late-instar larvae may be as much as 5 to 8 mm wide. The feeding larva leaves a thin layer of cells, which eventually collapses during the season, leaving visible scars on the roots. Generally, larval tunnels are present in the upper third of the root *(6.24a).* Young carrot plants may wilt or die as a result of attack by carrot weevil larvae, and bacteria and fungi may invade carrot roots through the tunnels made by the larvae *(6.24b).* Damage to poorly treated, commercial fields may reach 12%. In untreated fields, however, the carrot weevil can damage up to 70% of a carrot crop.

Identification Carrot weevil (family Curculionidae) adults are elongate and dark brown to black. A striped pattern on the thorax and forewings (elytra) results from the presence of rows of dark scales *(6.24c).* Adults average 7 mm in length and 2.5 mm in width, males generally being smaller than females. Eggs measure 0.8 by 0.5 mm, are pale yellow when laid, darken with age, and turn black just prior to hatching. There are four larval instars. Larvae are legless and creamy white with an amber-colored head (*6.24d*). Pupae are similar in size and color to the fourth-instar larva.

Life history Carrot weevil adults overwinter in and around carrot fields. They emerge early in the spring and feed on the foliage of young carrot plants. The females oviposit on carrot petioles when the plants reach the four- leaf stage, and on early celery transplants. In Quebec, the oviposition period lasts until the accumulation of 600 degree-days above 7°C. The larvae tunnel into the main root and, after completing their development, leave the root and pupate in the soil. New adults emerge in late August and September. They feed on carrot leaves but cause no economic damage, and search for winter quarters. At that time of year, the conditions of temperature and daylength (photoperiod) are such that the new adults are in a state of reproductive arrest (diapause) and rarely produce a fall brood of eggs.

The carrot weevil usually has only one generation per year on cultivated carrot in Quebec and Ontario. However, there may be a partial second generation if oviposition occurs on other hosts early in the spring, and if the new generation matures in July when conditions are still suitable for reproductive activity and oviposition. In the spring, carrots left unharvested after a rainy fall provide ready oviposition sites for adults when they emerge from their winter quarters.

Management

Monitoring — In Quebec, carrot weevil adults are monitored by traps made of wooden plates spaced 3 mm apart. A carrot as bait is positioned in a depression at the base of the trap *(3.2T2).* Two sets of three traps each are used for every field of four hectares or less. Each set is located near possible infestation sites at 3 to 5 m inside the field. The traps are spaced 2 m apart, pressed slightly into the soil, and held in place with a metal rod.

The traps should be visited twice a week from early May until the carrot plants reach the five-leaf stage. If traps are not available, mature carrots are placed on the soil around carrot fields. Ten carrots at 1-m intervals are placed about 3 m inside the field and renewed twice a week, starting at seeding time. On each carrot, the total number of feeding and oviposition punctures is recorded and the total number of punctures for each seven-day period is calculated.

When wooden-plate traps are used as described, the following thresholds are applicable in Quebec. If the cumulative number of weevil captures from the beginning of the monitoring period is below nine adults per six traps, no treatment is warranted; from 9 to 30 adults per six traps, a treatment notice is given to the grower at the two-leaf stage and treatment should be applied before the four-leaf stage. The traps are then renewed to verify the effectiveness of the treatment. If there are more than 30 adults per six traps, a treatment notice is given at the two-leaf stage and the traps are replaced. In that case, another treatment may be needed at the four-leaf stage to reduce the weevil population below the economic threshold. When carrot roots are used as bait, the threshold in Quebec is 20 feeding or oviposition punctures per 10 root-pieces over a seven-day period.

In Ontario, carrot weevil adults are monitored either by the method used in Quebec or by placing 5- to 10-cm lengths of mature carrots vertically in the soil of carrot fields between the rows. From 5 to 10 groups of five root-pieces each are distributed at both ends of a field. The presence of carrot weevil adults is determined by the oviposition punctures made in the root-pieces. The number of cavities per root- piece per day, the proportion of root-pieces attacked, and the maximum number of attacks observed indicate the likelihood of weevil injury in a field. The threshold is 0.3 oviposition punctures per root-piece per day, or over 25% of the root- pieces with oviposition punctures.

Cultural practices — Crop rotation is often recommended but it is almost impossible to isolate carrot fields from a source of carrot weevil in areas of intensive carrot cultivation. Late sowing can be used to reduce carrot weevil damage because carrot sown after an accumulation of 400 to 450 degree-days above 7°C becomes suitable only after most weevils have laid their eggs. Growers should remove left-over carrot root-pieces which otherwise may serve as overwintering and early oviposition sites, resulting in a second generation. They also compete with the traps and baits in monitored fields. Weeds, if left as windbreaks early in the season, should be removed because later they may act as a barrier between insecticides and the target adult-stage of the weevil. In rotations, non-umbelliferous plants should be used whenever possible.

Biological control — Numerous ground beetles (family Carabidae) attack eggs, larvae and adults of the carrot weevil and, in Quebec, the wasp *Anaphes sordidatus* (Girault) parasitizes over 50% of carrot weevil eggs in untreated plots. Also, the *Listronotus* strain of the entomophagous nematode *Steinernema carpocapsae* (Weiser) (syn. *Neoaplectana car- pocapsae* Weiser and *Steinernema feltiae* (Filipjev) in earlier literature), the fungi *Beauveria bassiana* (Bals.) Vuill. and *Metarhizium anisopliae* (Metsch.) Sorok., and the bacterium *Bacillus thuringiensis* Berliner have potential as biocontrol agents against an insect like the carrot weevil, which spends much of its life in the soil. Despite the natural occurrence of these organisms and a number of studies dealing with them as biocontrol agents of the carrot weevil, biological control is not commercially developed.

Chemical control — The use of a granular insecticide at seeding to control carrot weevil larvae before they enter the root was abandoned in the early 1980s. There are no reports or evidence of resistance to the organophosphate insecticides that are presently in use. At present, adults of the carrot weevil are controlled by applying one or two foliar treatments at 10- to 14-day intervals. Treatments must be applied after most adults have left their overwintering sites but before they have started egg laying. Recommendations are for treatments to coincide with the three-leaf stage of carrot in Quebec, and the one-leaf stage in Ontario.

If damage in the preceding year was higher than 2%, an insecticidal treatment is suggested. When no weeds are present, the insecticide must be applied in 250 to 400 L of water per hectare. If weeds are present, more water should be used for better canopy penetration.

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(Original by G. Boivin)

▶ 6.25 Cutworms *Figs. 6.25a-c; 11.26; 18.35a-g*

For species, see Table 18.35

Cutworms are occasional pests on carrot in all regions of Canada. The larvae are active mainly at night. In daylight they usually are found in the surface soil near the plants and assume a tight curl when disturbed. Many species attack young carrot plants. Coloration and markings differ from one species to another (*6.25a-c*). For more information on cutworms, see Tomato (18.35; *18.35a-g*).

Damage Cutworm larvae (*6.25a-c)* on carrot feed on the petioles, cutting them near the ground. A single larva can destroy numerous plants in the course of a night and the resulting damage is often concentrated in large, circular areas of the carrot field.

Identification (see Tomato, 18.35)

Life history (see Tomato, 18.35)

Management The only management strategy presently available for cutworms is to monitor and use insecticides if necessary.

Monitoring — Growers should check for cutworm damage while monitoring for other carrot pests, particularly in the spring, by watching for cut petioles and then searching for larvae in the nearby soil.

Cultural practices — Because many cutworm moths lay eggs in weedy fields and headlands the previous fall, keeping these areas clean by cultivation may reduce crop damage.

Chemical control — If damage is seen early, the larvae can be controlled with a foliar insecticide applied in the evening. Treatment is best confined to that part of the field where damage is present. No threshold is available but application of an insecticide is advised when cutworm damage is evident in a field. No resistance to insecticides has been reported in cutworms.

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(Original by G. Boivin)

▶ 6.26 Other insect pests *Figs. 6.26; 12.21a,b*

White grubs
Wireworms

White grubs *(6.26)* may be a problem on recently broken land but control measures are rarely necessary. For more information on white grubs, see Potato, 16.49.

Wireworms *(12.21a,b)* may be just as important as cutworms or white grubs in some areas. For more information on wireworms, see Maize, 12.21; Potato, 16.50.

(Original by G. Boivin)

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7 Celery, celeriac

BACTERIAL DISEASES

▶ 7.1 Bacterial leaf spot (northern bacterial blight) *Figs. 7.1a,b*

Pseudomonas syringae pv. *apii* (Jagger) Young, Dye & Wilkie (syn. *Pseudomonas apii* Jagger)

Bacterial leaf spot is sporadic and of relatively minor economic importance on celery, although it has been found repeatedly on transplants imported into British Columbia from California. Celery is the only host on which bacterial leaf spot has been reported to be a problem.

Symptoms The first symptoms appear on the leaves in the form of small, bright yellow, circular spots, 1 to 2 mm in diameter *(7.1a).* These enlarge, turn rusty brown and are usually surrounded by a yellow halo. When the spots are numerous, they merge, kill the leaves and give them a blighted appearance *(7.1b).* Petiole infections are rare.

This disease is distinguished from septoria blight by the absence of tiny black fungal fruiting bodies (pycnidia) scattered across the spots, and from cercospora blight by the absence of spores (conidia) on lesion surfaces.

Causal agent *Pseudomonas syringae* pv. *apii* is a Gram-negative, rodshaped bacterium with one to three polar flagella. Colonies produce a fluorescent green pigment on King's B medium under ultraviolet light. This bacterium can be distinguished from other fluorescent pseudomonads by LOP AT tests.

Newly developing lesions free from adhering soil particles and in which contaminating or secondary pathogenic microorganisms are absent or few in number should be used for isolations. A loopful of a suspension of plant tissue in sterile water may be streaked on the surface of a plate containing King's B medium. Colonies produce a fluorescent green pigment on King's B medium under ultraviolet light. Discrete, well-separated colonies may be transferred to nutrient agar for inoculum increase and further tests.

Disease cycle The pathogen can survive for one year in seeds and can also overwinter in infected plants. In seedbeds and fields, the bacterium is spread by splashing water, tools and workers. Development of bacterial leaf spot is favored by cool, wet conditions. At least 10 hours of leaf wetness are needed for disease establishment. Pathogenicity and growth of the bacterium are optimum at 20 and 25°C, respectively.

Management

Cultural practices — Disease-free seed should be used. If in doubt, seed that is at least two years old should be selected or it should be soaked in hot water at 48°C for 30 minutes. Seed beds should be disinfested through steam pasteurization or fumigation. A two-year rotation with non-susceptible crops is suggested. High plant densities, which promote disease development by increasing relative humidity and leaf wetness, should be avoided. Infected crop residues should be destroyed after harvest.

Resistant cultivars — Selections of Utah 52-70 may be resistant to bacterial leaf spot.

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(Original by L.M. Tartier and R.F. Cerkauskas)

FUNGAL DISEASES

▶ 7.2 Brown spot *Fig. 7.2*

Acremonium apii (M.A. Smith & Ramsey) W. Gams (syn. *Cephalosporium apii* M.A. Smith & Ramsey)

This disease has been observed occasionally in Ontario and Manitoba, especially following warm weather. Moderate infection, sufficient to render the celery unacceptable for soup purposes, has been reported. *Acremonium apii* has been found on celery but not on celeriac.

Symptoms Shallow, dry, rusty-brown lesions, which are circular to oblong and up to 1 cm long by 0.5 cm wide, are found on the inner, concave surface of the petiole (7.2). On the outer, convex surface, the lesions are smaller, more reddish-brown and occur along the ridges. The lesions are more numerous on the inner surface and more conspicuous on the outer petioles than on the leaves. They may grow together and affect the entire stalk, resulting in curling and distortion. Secondary invasion by bacteria into affected areas of older petioles produces wet, sunken, dark-brown lesions. On leaves, lesions are yellowish-brown, circular and 0.5 cm in diameter.

Causal agent *Acremonium apii* produces abundant hyaline conidia, which are variable in form. They measure 4.6 to 11.5 by 1.7 to 2.3 µm on V-8 agar after 16 days of growth. On malt agar, they are cylindrical to ellipsoidal, aseptate, occasionally with a truncate base, 3 to 6 by 1 to 2.5 µm, sometimes occurring in slimy heads. On potato-carrot agar, conidia may be up to 10 µm long and sometimes one-septate. Conidia produced on diseased celery plants may be considerably larger than those in culture. The conidiophores are not distinct from the hyaline hyphae. Conidiogenous cells arising from single hyphae may be aggregated in strands, but are usually solitary and sometimes branched. Each conidiogenous cell has a short, indistinct collarette and produces conidia from the apex in succession without increasing in length. Chlamydospores are 3.6 to 8.8 by 6.2 to 9.3 µm. Colonies on malt agar or potato-carrot agar are white, fluffy, raised, later turning grayish-white, showing restricted growth, and attaining a diameter of 20 mm in 10 days at 22°C. Optimum growth on potato-dextrose agar occurs at pH 6.5 and at 25°C; no growth occurs at pH 4.0.

After surface disinfestation of infected tissue pieces, the fungus can be isolated on potato-dextrose agar (pH 5.0) amended with aureomycin (200 ppm) or on water agar. Alternatively, diseased petioles can be washed and placed in a moist chamber to induce sporulation.

Disease cycle The fungus may be present in seedlings in both the seedbed and field. However, the disease is serious only when unusually high temperatures occur during the growing season. Infections may occur during cool summers, but high temperatures are required for rapid lesion development. The disease develops more quickly at 25 than at 13°C, requiring 6 as opposed to 11 days from time of infection to symptom development. Plants weakened by poor nutrition, excessive salinity or waterlogged soil are more susceptible to infection. The fungus survives in soil or crop residues as chlamydospores, and initial inoculum is likely from these sources since the disease is most common when rotation is not practiced. Dispersal of inoculum is probably by wind, splashing water, workers and implements.

Management

Cultural practices — A four-year rotation with onion, lettuce or potato will reduce initial inoculum. Conditions that favor optimum growth of celery, including adequate nitrogen supply, prevention of waterlogging, and avoidance of excessive salinity, will reduce the susceptibility of celery plants to infection. Growers are advised to reduce the movement of workers and machinery through crops, minimize late cultivation, and use transplants that have been grown in disinfested soil or soilless media.

Resistant cultivars — Selections of Tall Utah 52-70 may be resistant to brown spot.

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(Original by R.F. Cerkauskas)

▶ 7.3 Cercospora blight (early blight) *Figs. 7.3a,b*

Cercospora apii Fresen.

Cercospora blight has been reported from all the major celery-growing areas of Canada. It can cause serious losses in yield and quality, particularly if it attacks early in the season. Defoliation and stunting often occur, necessitating extra labor and time to trim affected leaves and petioles from diseased plants. Celeriac may also be affected.

Symptoms Symptoms appear primarily on the foliage and petioles, starting from the outside and progressing inward and upward. On the leaves, round chlorotic spots are visible on the upper and lower surfaces. The spots enlarge rapidly up to 1 cm or more and turn brownish-gray. They have a dry, papery texture and are generally without a distinct border *(7.3a).* Under humid conditions, the lesions contain large numbers of conidiophores with conidia. Leaves containing a few spots become chlorotic and wither. Under favorable environmental conditions for disease development, affected plants quickly appear blighted *(7.3b).* The lesions on petioles are elongate and parallel to the long axis of the stalk.

Causal agent *Cercospora apii* has a septate, pale brown, torulose mycelium, generally 2 to 4 µm in diameter, and often forms hyphal knots or pseudostromata in the substomatal cavities. Brown, septate conidio- phores in groups of up to 30 emerge through the stomata from these structures. They are unbranched, usually 30 to 70 µm long, and taper from a width of 5 to 9 µm at the base to 3 to 4 µm at the apex. Several conidia are produced separately near the apex of each conidiophore and a scar is left at each site where the conidium has detached. The conidia are straight or curved, slightly obclavate at the base, with a diameter of 3.5 to 5 µm below the middle, and taper toward the apex. They are hyaline, smooth, 9- to 17-septate, usually 60 to 200 µm long, but sometimes longer with a conspicuous hilum.

Cross-inoculations of various hosts with many *Cercospora* species have not been performed, although it is probable that this pathogen occurs on many other plants and has many names. The fungus is easily distinguished from *Septoria apiicola* by the absence of pycnidia within the spots and by the larger lesions evident with cercospora blight.

The fungus is easily isolated from infected tissues; however, sporulation is somewhat sparse on potato-dextrose agar and V-8 agar. Colonies are smooth or with radial folds and measure 16 to 18.5 mm in diameter after seven days at 25°C on V-8 agar.

Disease cycle The fungus is both seed- and soil-borne, and it can survive more than two years in seed. It can also overwinter in infected plant residues in the soil. Primary inoculum arises from seeds and diseased plant material in seedbeds or fields, but the amount of blight on transplants has a major influence on disease development in the field. Cercospora blight generally appears before septoria blight in the field, although the latter is a more frequent problem.

Cercospora blight is favored by a relative humidity near 100% and temperatures from 15 to 30°C. Sporulation is significantly enhanced when these conditions occur for more than 10 hours per day, except when temperatures are below 12°C. Conidium germination and penetration of leaves occur under conditions of high relative humidity, heavy dew or light rain. High temperatures following penetration favor establishment of the fungus and sporulation generally occurs 5 to 14 days later.

Conidia are readily released during periods of decreasing relative humidity, less than 90%, especially during the morning. Daytime cloudiness and rain reduce the number released; however, enhanced leaf wetness periods increase conidium production. Conidia are spread by wind, splashing water, workers and implements. Unlike *Septoria* conidia, which are "sticky" and disseminated only over several metres, conidia of C. *apii* are dispersed over greater distances. Heavy rain will wash them from leaves, thereby reducing their availability for dispersal by wind.

Management The use of healthy transplants is the key to control of this disease.

Cultural practices — (see septoria blight, 7.7)

Resistant cultivars — Most celery cultivars are susceptible. Tolerant cultivars include Emerson Pascal, June-Belle and Earlibelle.

Chemical control — (see septoria blight, 7.7) The time for fungicide application may differ for the two diseases because optimum environmental conditions for development of cercospora blight differ from those for septoria blight. Cercospora blight usually appears first.

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(Original by R.F. Cerkauskas)

▶ 7.4 Damping-off *Fig. 7.4*

Pythium debaryanum of authors, not R. Hesse
Pythium ultimum Trow
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Damping-off can cause serious losses in celery and celeriac seedbeds in greenhouses and in the field. The damping-off pathogens have wide host ranges.

Symptoms Young seedlings are the most susceptible to damping-off, although the problem can occur at any time during the production cycle. There are two types of damping-off, depending on whether the disease occurs before or during germination (pre-emergence) or after emergence (post-emergence).

Pre-emergence damping-off is characterized by seed decay or death of the celery seedling before emergence. The failure of seedlings to emerge from the soil results in well-defined circles or areas without plants *(7.4).* Since there may be no obvious symptoms, this problem is often attributed to poor seed vigor.

Post-emergence damping-off also appears as well- defined areas of seedlings showing visible symptoms of the disease. Plants are attacked at the soil level or on the underground parts. Tissues at the soil level become spongy and water-soaked. As the disease progresses, the stem is girdled and the plants wilt and fall over.

Damping-off progresses quickly. When ambient conditions are favorable, young infected plants may die overnight. When infection occurs after the plants have already begun to harden, they are rarely killed. These plants reveal a girdled, dry, hard, blackish collar. This is the black stem symptom of damping-off. Surviving plants are generally unmarketable.

Causal agents (see Beet, pythium and rhizoctonia root rots, 5.7, 5.8; and Carrot, pythium root dieback, 6.13)

Disease cycle The fungi that cause damping-off are soil- borne. They commonly develop on young seedlings in the seedbed or field during the spring, when ambient conditions are unfavorable to rapid vigorous celery growth. (For further details, see Beet, pythium and rhizoctonia root rots, 5.7, 5.8; and Carrot, pythium root dieback, 6.13.)

Management

Cultural practices — In the greenhouse or seedbed, the soil must be disinfested by pasteurization,

fumigation or by incorporating a fungicide. Where sterilized growing media are used, all precautions are needed to prevent contamination by tools, water and containers. These items should be thoroughly disinfested before use. Good lighting will prevent etiolation, which predisposes seedlings to damping-off.

Promotion of rapid, vigorous seedling growth, reduction of plant density, and reduction of soil moisture through adequate drainage help to minimize damping-off. Plants should be watered before noon, and the greenhouses or seedbeds should be sufficiently ventilated so that the plants are dry by sunset. Healthy seed treated with a protective fungicide should be used for planting.

Chemical control — Registered fungicides are available but only for seed and soil treatment. When the disease appears, a protective fungicide should be applied in a high- volume water drench.

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(Original by L.M. Tarder and R.F. Cerkauskas)

▶ 7.5 Fusarium yellows *Figs. 7.5a-c*

Fusarium oxysporum f. sp. *apii* (R.R. Nelson & Sherb.) W.C. Snyder & H.N. Hans.

Race 1 of the fusarium yellows pathogen caused extensive losses to celery growers from about 1920 until the late 1950s when the resistant celery cultivar Tall Utah 52-70 was introduced. In recent years, significant yield and quality losses due to race 2 of the fungus have occurred on muck soils in British Columbia and Ontario. Only celery is susceptible to this pathogen.

Symptoms Slight stunting, stiffening of the outer petioles, and a brown discoloration of the vascular system of the host plant occur with mild or late infections. The leaves generally become brittle, develop a rough texture and curl upward. In severe infections, the outer leaves become chlorotic first *(7.5a)* and the yellowing spreads to other leaves as the disease progresses throughout the vascular system of the roots and crown. In later stages of disease development, the foliage becomes necrotic *(7.5b).* Also, there is extensive reddish-brown discoloration of the crown and vascular system of the roots and petioles, and plants are severely stunted. The internal crown discoloration may be seen by splitting the plant in half lengthwise *(7.5c).* Bacteria and other microorganisms often invade the affected crown and root tissues and cause secondary rot. Occasionally, dry rot of the crown follows and a cavity may form. Plants may die in the final stages of disease development.

Causal agent On potato-dextrose agar at pH 6.5 to 7, the mycelium of *Fusarium oxysporum* f. sp. *apii* is initially white, becoming pale vinaceous to somewhat purple or mauve. Microconidia are elliptical, generally 5 to 12 by 2.2 to 3.5 µm, produced in abundance, generally non- to one-septate or rarely two-septate. They are borne on simple phialides arising laterally on the hyphae or from short, sparsely branched conidiophores. The macroconidia are sparse, generally three- to five-septate, less than 4 µm wide, usually fusoid-subulate, and pointed at both ends. Chlamydospores are often abundant, both smooth and rough walled, and may be both terminal and intercalary, and generally solitary. They may occasionally be in pairs or in chains.

It is important to use a wild-type culture in identifying and testing this pathogen. Isolations from diseased celery plants yield mycelial cultures that produce abundant microconidia and few macroconidia, in contrast to intermediate, pionnotal and appressed cultural forms that are rare in nature and are less pathogenic to celery. The latter forms often arise from the mycelial isolates after prolonged storage in culture and repeated transfers. Culture propagation by single microconidial transfer, followed by incubation in either complete darkness or diffuse light, will maintain mycelial characteristics true to the wild-type isolates. Isolations from root or crown tissue and determinations of soil populations are possible using Komada's medium or a sorbose-based selective medium.

Race identification based on reactions of yellow and green cultivars in greenhouse virulence tests may be inconclusive and misleading due to variation with environmental conditions, methods of inoculation and host age. Laboratory tests based on colony size of isolates on special sorbose media and heterokaryon (vegetative) compatibility in conjunction with virulence tests may be necessary.

Disease cycle The severity of fusarium yellows in the field depends on the degree of infection and whether the plants are infected early or late in the season. Early infection often leads to severe losses in yield and quality. Other factors, such as the spore populations in the soil and weather during the growing season, are also important. As the population of spores increases, more severe symptoms develop. The disease is most severe during warm seasons and on heavy wet soils.

The distribution of affected plants in fields may appear patchy, probably due to the uneven distribution of spores in the soil. The fungus is spread when infested soil is carried from contaminated to pathogen-free fields by farm equipment or on workers' shoes. It may also be introduced by use of infected transplants or by infested soils used to grow transplants. Fungal spores can be spread in irrigation and flood water and by windblown soil.

The fungus persists in soil for many years in the absence of celery as dormant spores or by colonizing the roots and stems of non-susceptible hosts such as sweet corn, cabbage and especially carrot. The roots of many symptomless host weeds, such as lamb's-quarters, smartweed, barnyard grass and purslane, also are colonized. Crop plants and weeds can allow the fungus to multiply. Thus, population levels increase in the soil in the absence of celery, making shortterm fallowing of infested fields ineffective as a control measure. Populations increase quickly if susceptible celery cultivars or carrots are grown in infested fields. Continuous celery production and incorporation of celery trimmings back into the soil at harvest will greatly increase spore populations and enhance disease development in the subsequent celery crop because the crop residues serve as a food source for the fungus. Sometimes, however, even low spore populations in the soil may cause crop failures.

Management

Cultural practices — Celery trimmings should not be returned to infested fields after harvest.

Growers are advised to practice a two- to three-year rotation with onion or lettuce to reduce *Fusarium* populations in infested fields. Carrot and sweet corn should be avoided in rotations, and weeds should be controlled because they allow the fungus to multiply in the root zone.

Introduction of fusarium yellows on transplants can be prevented by sanitation in the greenhouse: 1) steaming or fumigating soil and flats; 2) disinfesting work benches and surfaces before seeding; 3) using a disease-free commercial potting mix or soil that has been steamed or fumigated; 4) following the recommendations for cleanliness and sanitation, bench and equipment

sterilization, and soil pasteurization for greenhouse crops; and 5) avoiding use of field- grown transplants from areas where fusarium yellows has been reported. To avoid introduction of infested soil, non- infested fields should be worked before entering infested fields, and caution should be exercised in allowing machinery and people into infested fields. Soil washed from machinery should be collected and disposed of. Machinery should not be borrowed from areas where fusarium yellows is present or suspected. Flood and drainage water should be prevented from moving from infested to non-infested fields. Pallet boxes and containers used for packing celery where fusarium yellows has been reported should be washed with hot water or disinfested before being cycled to other growers.

Resistant cultivars — Resistant (Picador, Matador, Starlet) or moderately resistant (Tall Utah 52-70 HK strain, Deacon) cultivars provide the cheapest and easiest method of control in fields infested with race 2 of *Fusarium oxysporum* f. sp. *apii*. Moderately resistant cultivars yield well in fields with light to moderate soil infestations of this pathogen, but yield losses will occur in heavily infested soils. Other cultivars, such as Tendercrisp, are also moderately resistant. Resistant or moderately resistant cultivars should always be grown in infested fields. The use of susceptible cultivars, in addition to resulting in yield and quality losses in infested fields, will exacerbate the disease situation in future years by increasing populations of the pathogen in the soil.

Chemical control — Fungicidal drenches at the time of transplanting or later have been unsuccessful in controlling fusarium yellows. Fumigation of seedbeds may be successful initially, but complete prevention of spread of the pathogen involves frequent cleaning of equipment and control of windblown soil.

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(Original by R.F. Cerkauskas)

▶ 7.6 Pink rot (white mold) *Figs. 7.6a,b*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

This fungal disease is generally of minor economic significance to celery. It can occasionally cause damage in the seedbed, field and in storage. Pink rot also affects celeriac and a variety of other vegetable crops. On the latter, the disease is commonly referred to as white mold (see also Carrot, sclerotinia rot, 6.15).

Symptoms All stages of field celery as well as the stored product can be attacked. The disease causes damping-off in seedbeds, attacking the seedling stems at ground level. A soft rot develops and causes seedlings to collapse. Affected tissues become covered with a fluffy white mold sprinkled with large black resting bodies (sclerotia).

In the field, the fungus also invades the celery plant at the soil level and causes a watery soft rot *(7.6a)*. The diseased tissue takes on a characteristic pinkish color *(7.6b)* and becomes covered with white mold and sclerotia. Eventually, the heart of the celery rots and the entire plant wastes away. Other rot-causing organisms may invade the diseased tissues, leading to rapid destruction of the plant.

Celery with latent pink rot infection may develop disease symptoms in storage. Lesions first appear as water-soaked spots, turning soft as the tissues decay. Lesions are generally light brown with pinkish borders but no odor. Advanced lesions may be covered with a white, more or less appressed mycelial growth of the causal fungus with various stages of sclerotial formation.

Phytophotodermatitis, a blistering disorder of the skin, has sometimes occurred in people working in celery fields, especially in crops affected by pink rot, and in some grocery workers. Celery plants may contain furanocoumarins, which when placed on the skin and exposed to sunlight produce a blistering lesion. Phytophotodermatitis also has been associated with exposure to various fruits, flowers and vegetables, including dill, parsley, lime, parsnip, bergamot and chrysanthemum.

Causal agent (see Bean, white mold, 15B.9)

Disease cycle (see Bean, white mold) Pink rot development is more serious during cool, moist weather. High humidity and poor air movement within the crop canopy will promote the disease. In British Columbia, the prolonged use of floating row covers has been observed to increase the incidence of pink rot.

Management

Cultural practices — Pink rot is difficult to control. The only economical method is to reduce the level of initial inoculum by following a three-year rotation with non-susceptible crops such as onion, corn and cereal grains. Infected crop residues must

also be destroyed in order to avoid more serious soil contamination. Deep plowing to bury the sclerotia may help to control pink rot if other cultivation practices do not bring these sclerotia back up to the soil surface. Celery should be stored at high humidity and between 0 and 1°C. Plants harvested from affected crops should not be stored for long periods.

To protect against phytophotodermatitis, workers should wear protective clothing such as long-sleeved shirts and gloves when harvesting or handling celery or other fura- nocoumarin-containing crops. They also should wash exposed skin with soap and water after handling these crops.

Selected references

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(Original by L.M. Tartier, A.A. Reyes and R.J. Howard)

▶ 7.7 Septoria blight (late blight)*Figs. 7.7a-g*

Septoria apiicola Speg.
(syn. *Septoria apii* F. Chester)
(syn. *Septoria apii-graveolentis* Dorogin)

Septoria blight of celery has been reported from all provinces. It causes yield losses by defoliating and stunting the plants. It also increases harvest costs because of the labor required to trim diseased leaves and petioles. Other losses occur during storage when secondary organisms and other diseases, especially pink rot, occur in association with late blight. The pathogen is restricted to celery and celeriac.

Symptoms Small black pycnidia may be present on the surface of infected seed. Symptoms on the foliage begin on the older outer leaves and later on younger inner leaves. Chlorotic spots and flecks develop, turning reddish brown or brownish black and becoming necrotic *(7.7a).* The necrotic areas usually have a definite margin and are surrounded by chlorotic halos that merge gradually into uninfected tissue. Most lesions are round, but shape may vary near veins or other lesions. Lesions remain less than 3 mm or expand up to 10 mm in diameter with several lesions growing together *(7.7b).* Mycelium is present in the lesions and surrounding tissue. Numerous small, black pycnidia are scattered in the smaller lesions, but in larger lesions they are less numerous and more concentrated near the center. In severe cases, when large numbers of lesions and pycnidia appear, the entire leaf may be destroyed. Older outer petioles have more severe symptoms than inner petioles *(7.7c,d).* Infections arise along the length of the petiole when cirrhi of conidia are extruded from pycnidia in leaves *(7.7j)* and carried down the petioles by rain or dew.

Causal agent The brown to black pycnidia of *Septoria apiicola* are immersed in leaf or petiole tissue *(7.7e).* They have a diameter of 75 to 195 µm with pycnidial walls three cells thick, composed of pale brown pseudoparenchymatous tissue that is darker and thicker near the ostiole. The pycnidia are membraneous when fresh, carbonaceous when dry and are not beaked. The conidia are hyaline, needle-shaped, straight to curved or flexuous, generally three-septate (range one to five septa), with a slight taper toward the apex *(7.7g).* They measure 22 to 56 (mean 35 µm) by 2 to 2.5 µm. The conidia develop as phialospores from hyaline, septate conid- iophores formed from the innermost layer of cells of the pycnidium. The conidiophores measure 8 to 10 by 3 to 3.5 µm. The conidia are extruded in white to tan cirrhi when pycnidia are wet.

On potato-dextrose agar, most strains are initially yeast-like and slow- growing. The sub-surface mycelium is brownish black, sometimes producing a diffusible brown to pink pigment. Aerial mycelium is white. Pycnidia with oozing masses of conidia are often scattered over the colony surface.

Disease cycle Infested seed is the major source of primary inoculum. Pycnidia, mycelium and conidia may occur on seed, but the fungus is absent from embryos and endosperms, even though the mycelium can penetrate pericarps and testas. Seed-borne conidia remain viable for at least 15 months but not beyond two years.

In seedbeds, primary inoculum originates from infected seeds and crop residues, and can be spread by contaminated tools and workers. When the seed germinates, the seed coat containing pycnidia may remain partially attached to the cotyledons. This allows the conidia to ooze onto the cotyledons and infect them. Young leaves are soon infected under favorable conditions, although the spotting on seedling foliage may not always be obvious. The movement of workers and implements, particularly when seedlings are wet, also may introduce the fungus and enhance dissemination within the seedbed.

In fields, primary inoculum originates from the use of diseased transplants, infested soil, contaminated plant containers, implements and workers. Dissemination of primary and secondary inoculum within the field arises by splashing rain, irrigation water and movement of workers and implements, especially when the foliage is wet.

Optimum temperatures for germination of conidia are between 5 and 27°C over a 48-hour period and between 20 and 22°C for germ tube growth. Free water is not essential either for germination or for infection of celery leaves, but relative humidity

below 90% significantly limits infection. The time for infection to symptom appearance is seven to eight days at 21 to 27°C and 12 days at 18°C. The time from inoculation to production of secondary inoculum is 10 to 12 days.

The disease often appears during the latter part of the growing season. The greatest disease development occurs when the fungus develops more rapidly than the growth of the celery plant. Such conditions prevail when dry weather prevents rapid plant growth, and when cool, misty nights or heavy dews are followed by dull days. Under these conditions the leaves and petioles remain wet for considerable periods of time, allowing the pathogen to develop more rapidly than the host.

Management

Cultural practices — The use of disease- free seed will prevent introduction of primary inoculum to the seedbed. Control of the disease in the seedbed is easier and less costly than control in the field. Celery and celeriac seed can be disinfested with hot water (48°C for 30 minutes). If diseased plants are noticed in the greenhouse or seedbed, they should be pulled. Crowding of plants and planting in fields and outdoor seedbeds where the disease has occurred for at least two years should be avoided. If fresh land is not available for seedbeds, the remains of each year's crop should be removed as soon as possible to eliminate the survival of self-set plants that may become infected later in the season. Unless treated with a fungicide, infested seed should not be planted until it is three years old because the fungus cannot survive in seed beyond two years.

Resistant cultivars — No cultivars have a high level of resistance, but Emerson Pascal, Florida Green Pascal, Earligreen, Green Giant and June-Belle are somewhat tolerant.

Chemical control — Celery and celeriac seed should be treated with a protective fungicide before planting. Protective foliar fungicides are important in seedbeds where long periods of leaf-wetness and high relative humidity due to high plant density create conditions for rapid disease development. Timing of protectant fungicide spray applications in production fields is also important. More frequent applications may be necessary under conditions that favor the disease.

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(Original by R.F. Cerkauskas)

VIRAL AND VIRAL-LIKE DISEASES

▶ 7.8 Aster yellows *Figs. 7.8a,b*

Aster yellows mycoplasma-like organism

This disease is common but generally of little economic importance on celery because of its low incidence. Aster yellows affects several other vegetable crops, including carrot, lettuce and potato.

Symptoms Young celery plants affected by aster yellows form more petioles than normal plants (*7.8b).* The outer petioles are rigid, while the petioles at the heart become chlorotic and deformed, remain stunted, grow tendrils and sometimes become tangled. These initial symptoms are followed by an overall yellowish to whitish discoloration of the plant (*7.8a).*

Causal agent (see Lettuce, aster yellows, 11.15)

Disease cycle (see Lettuce, aster yellows)

Management (see aster leafhopper, 7.18, 11.23)

Cultural practices — Growers are advised to eradicate weeds around fields and along ditches bordering celery fields.

Selected references

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(Original by L.M. Tarder)

▶ 7.9 Heart mosaic *Figs.7.9a-c*

Cucumber mosaic virus

Several strains of cucumber mosaic virus can attack celery. It is a disease of little significance, usually affecting only a few scattered plants in a field. The virus also infects other vegetables, such as cucurbits, tomato, spinach, pepper, turnip, carrot, potato and lettuce.

Symptoms Leaves of affected plants are mottled and yellow-veined (*7.9b).* They also are often deformed, appearing stretched and crinkled. The petioles at the heart are curved downward, giving the plant an open appearance. Sometimes, there are no visible symptoms on the foliage, but characteristic elongate, brownish, translucent, slightly sunken spots occur on the petioles (*7.9a).* Marketability is reduced if the spots cover the entire length of the petioles (7.9c).

Causal agent (see Greenhouse cucumber, cucumber mosaic, 22.20)

Disease cycle (see Cucurbits, cucumber mosaic, 9.15) The virus overwinters in weeds along the edges of fields and ditches. It is spread into celery crops by aphids (7.17).

Management Control of aphids helps to reduce the build-up and spread of the disease in celery crops.

Cultural practices — Growers should eradicate weeds along the edges of the fields to eliminate the reservoir of virus available to infect celery crops. After harvest, infected crops should be destroyed by discing or plowing.

Selected references

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(Original by L.M. Tartier)

NON-INFECTIOUS DISEASES

▶ 7.10 Blackheart *Figs. 7.10a,b*

This major disorder can seriously affect the marketability of celery and celeriac crops and has been reported in most provinces. In individual cases, entire fields have been lost.

A similar disease also affects tomato and pepper (blossom- end rot, 18.21) and lettuce (tipburn, 11.19).

Symptoms Blackheart affects the interior of the plant. Initially, the younger leaves appear waterlogged, then wilt and turn black (*7.10a).* If the disorder is temporary, the plants can recover and produce new hearts. When this occurs, affected leaves take the form of blackened tips at the end of lengthened petioles *(7.10b).* If the disorder persists, the growing point dies and the plant is unmarketable. The outer ring of leaves is not affected and appears normal. Diseased leaves are often subject to attack by soft rot bacteria that completely destroy the heart of the plant.

Causal agent Blackheart is a physiological disorder resulting from poor calcium assimilation in the plant. It is also related to fluctuating soil moisture conditions. This disorder appears when environmental conditions stimulate rapid plant growth, such as when heavy rain or irrigation follows a prolonged drought. Available calcium in the soil cannot meet plant requirements and a deficiency develops. High soil nitrogen, a low calcium to nitrogen ratio, and high levels of potassium and sodium, which can reduce calcium absorption, may also be associated with this disorder. Conditions favoring blackheart can occur several times during a season. The disorder becomes more difficult to correct when the plant is in a period of rapid growth or approaching maturity.

Management

Cultural practices — Blackheart can be prevented by encouraging steady plant growth through well-balanced fertilization and a regular supply of water. During periods of drought and stress for the plant, calcium nitrate or calcium chloride sprays should be applied before symptoms appear. Fertilizers can be used to provide a high calcium to potassium ratio.

Resistant cultivars — Selections of Tall Utah 52-70 are more susceptible than Florida 683.

Selected references

Foster, A.C. 1934. Black-heart disease of celery. *Plant Dis. Rep.* 18:177- 185.

Geraldson, C.M. 1954. The control of blackheart of celery. *Proc. Am. Soc. Hortic. Sei.* 63:353-358.

(Original by L.M. Tartier)

▶ 7.11 Chlorosis *Fig. 7.11*

Magnesium deficiency
Manganese deficiency

This disorder is quite often encountered on isolated celery plants in soils that have been cultivated for many years. Nevertheless, it is usually considered a minor problem. In the muck soils of coastal British Columbia, large sections of celery fields are sometimes affected and the overall appearance of these areas may resemble a fusarium yellows infection. The disorder has not been reported on celeriac.

Symptoms Yellowing (chlorosis) symptoms first appear on the oldest leaves. Manganese deficiency produces chlorosis between the leaf veins and eventually affects the entire leaf. In extreme cases, the disease can affect overall growth. Magnesium deficiency takes the form of a much more obvious chlorosis of the older leaves, which then extends to the youngest leaves. Chlorosis begins at the leaf margins *(7.11)* and progresses steadily until the affected leaf turns white. In acute cases, the leaves turn brown, dry out, die and drop off.

Causal agent Chlorosis is caused by a deficiency of manganese or magnesium. Manganese deficiency appears to be more common in alkaline soils, where manganese is often present in a form that cannot be assimilated by the plant. Magnesium is an element that leaches away easily, so a deficiency is often encountered after prolonged heavy rains.

Management

Cultural practices — Celery should be grown in a slightly acid soil where possible. Excessive liming should be avoided. In addition, the soil should be adequately fertilized by incorporating manganese sulfate or magnesium into the fertilizer. Symptoms of these deficiencies on celery crops can be corrected by spraying the leaves with manganese or magnesium sulfate.

Selected references

Johnson, K.E.E., J.F. Davis and E.J. Benne. 1961. Occurrence and control of magnesium deficiency symptoms in some common varieties of celery. *Soil Sei.* 91:203-207.

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(Original by L.M. Tartier)

▶ 7.12 Cracked stem (boron deficiency) *Figs. 7.12a,b*

Boron deficiency

This is a serious disorder that is most often encountered on celery and celeriac crops grown in freshly broken organic soil and in old fields. A similar problem affects other vegetables, especially crucifers (see Crucifers, boron deficiency, 8.23).

Symptoms The symptoms are very characteristic and easily identifiable. Brown, cross-wise cracks appear on the ribs of the petioles. These vary in number according to the intensity of the disease. Their margins are often raised and develop a brown color. The plants remain small and bushy with rigid and brittle petioles when the deficiency is pronounced. Elongate brown translucent spots are sometimes found on the inner surface of stalks. Beginning with the leaves, the petioles bend, become tangled, turn brown and dry out. The growing point is often destroyed and a dry rot gradually destroys the petioles at the heart to the base *(7.12a,b).* Most often, only a crown of outer petioles surrounding a cavity with a brown base remains at the end of the season. Affected plants may be attacked by secondary rot organisms.

Causal agent Cracked stem is caused by a deficiency of available boron in the soil. It is more severe on soils with excess lime, which prevents assimilation of boron by the plant. High levels of potassium and ammonium nitrogen also seem to be associated with this problem. Boron deficiency is often more serious on deeply cultivated soils. Drought promotes this disorder.

Management

Cultural practices — To ensure that there is sufficient boron in the soil, growers should incorporate borax into the fertilizer and maintain a slightly acidic soil pH, where practical. When the first symptoms appear or when drought ensues, the leaves should be sprayed with boron. A well-balanced fertilizer program, especially in potassium, nitrogen and calcium, will help to prevent cracked stem.

Resistant cultivars — Selections of Utah 52-70 differ in their susceptibility to cracked stem.

Selected references

Kendrick, J.B., R.T. Wedding, J.T. Middleton and J.B. Hall. 1954. Some factors affecting development and control of adaxial crack stem of celery. *Phytopathology* 44:145-147.

Yamaguchi, M., F.W. Zink and A.R. Spurr. 1953. Cracked stem of celery. *Calif. Agric.* 7(5): 12.

(Original by L.M. Tartier)

▶ 7.13 Spongy petiole (pithiness) *Fig. 7.13*

This physiological problem is commonly encountered on celery during some seasons, but is of little economic significance.

Symptoms Affected plants have hollow petioles that can be easily crushed by applying pressure with the fingers. These petioles are light in weight and, when cut, the interior appears spongy *(7.13).* All petioles are affected and the disorder may even appear early in the growth of the plant. More frequently, however, only the outer petioles are affected.

Causal agent Spongy petiole is related to unfavorable growing conditions. It is more common in late July and August when plants are growing rapidly and growth is interrupted by cool conditions. The appearance of spongy petiole is more frequent a week or a few days before maturity when available nitrogen and potassium are usually low. Other stress factors, such as an irregular supply of water or unbalanced or poorly timed fertilization, may also encourage this disorder. Spongy petiole can continue to develop in storage.

Management

Cultural practices — A uniform rate of plant growth should be maintained by providing optimal growing conditions. Mature crops should be harvested promptly and stored at temperatures that inhibit senescence.

Resistant cultivars — Selections of Utah 52-70 are more susceptible than Florida 683.

Selected references

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(Original by L.M. Tartier)

▶ 7.14 Stringiness

This is a physiological problem affecting the quality of the celery stalk but rarely resulting in economic loss. It can develop in the newer, "stringless" cultivars of celery.

Symptoms Petioles of affected plants have thickened, fibrous strands around the circumference of the outside edge close to the epidermis. The fibers occur along the length of the petiole and may be found within externally visible ridges. All petioles can be affected, although the disorder more commonly affects the mature, outer petioles.

Causal agent Stringiness is caused by secondary wall thickening of the supporting collenchyma tissue. The condition is related to low nitrogen availability and may appear as the plants reach maturity. Plants subjected to wind stress also may have earlier and greater wall thickening of collenchyma. To some extent, stringiness is an inheritable characteristic.

Management

Cultural practices — Adequate nitrogen availability should reduce the incidence of the disorder. Timely and balanced fertilization and regular irrigation should be followed. Harvest should not be delayed.

Selected references

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Nonnecke, I.L. 1989. *Vegetable Production.* Van Nostrand Reinhold, New York. 657 pp.

(Original by M.M. Gaye)

NEMATODE PESTS

▶ 7.15 Northern root-knot nematode *Figs. 7.15a,b*

Meloidogyne hapla Chitwood

Symptoms on celery and celeriac include yellowing and stunting of stalks, prolific branching of rootlets, and production of small, spherical galls on roots (*7.15a,b*). For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 7.16 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms on celery and celeriac include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38, and Management of nematode pests, 3.12.

Selected references

Townshend, J.L. 1962. The root-lesion nematode, Pratylenchus penetrans (Cobb, 1917) Filip. & Stek. 1941, in celery. Can. J. Plant Sci. 42:314-322.

INSECT PESTS

▶ 7.17 Aphids Figs. 16.41a,b

Green peach aphid *Myzus persicae* (Sulzer)
Other aphids

Aphids, primarily the green peach aphid (see Potato, 16.41), occur on celery but their abundance varies greatly with seasonal conditions, location and control programs. When abundant, aphids cause leaf distortion. The presence of their molted skins (exuviae) and honeydew can make celery heads unmarketable. In Quebec, a reduction of insecticide use, resulting from management programs for the tarnished plant bug, has led to an increase in aphid problems on some farms.

Selected references

Beirne, B.P. 1972. Pest insects of annual crop plants in Canada. VI. Hemiptera - Homoptera. *Entomol. Soc. Can. Mem.* 85. 73 pp.

(Original by A.B. Stevenson)

▶ 7.18 Aster leafhopper *Figs. 11.23a,b*

Macrosteles quadrilineatus (Forbes)
(syn. *Macrosteles fascifrons* of authors, not Stål)

The aster leafhopper infests celery and is economically important on celery as a vector of aster yellows (for further detail, see Lettuce, aster leafhopper, 11.23).

Selected references

Miller, L.A., and A.J. DeLyzer. 1960. A progress report on studies of biology and ecology of the six-spotted leafhopper, *Macrosteles fascifrons* (Stal), in southwestern Ontario. *Proc. Entomol. Soc. Ontario* 90:7-13.

(Original by A.B. Stevenson)

▶ 7.19 Carrot rust fly *Figs. 6.23a,c,e*

Psila rosae (Fabricius)

Celery is very attractive to carrot rust fly (see Carrot, 6.23). The adults are likely to be present on celery grown in locations where carrot also is grown. In Ontario, economic damage to celery by the carrot rust fly is rare and crop losses, in general, are insignificant. However, carrot rust fly populations in celery fields are a threat to nearby carrot and parsnip crops.

Celeriac is also a host of the carrot rust fly, with damage and susceptibility similar to that of celery.

Damage Larvae of the carrot rust fly feed on the roots of celery. The first-generation larvae can develop on early celery with no visible indication of above-ground attack. If larvae are abundant, wilting or stunting can occur on plants that are small and under moisture stress. Such damage is more likely to occur near the periphery of fields. Adult carrot rust flies from early celery crops will supplement the population attacking carrot in late summer and early autumn. By that time, celery is well developed and second- generation larvae are unlikely to cause significant injury to the crop. In general, carrot rust fly is not a major economic pest of celery in Canada and significant injury rarely occurs.

Management

Monitoring — Carrot rust fly adults are monitored with yellow sticky traps (*3.2T1*) (see Carrot, 6.23). No action threshold is available but any threshold on celery would be considerably higher than for carrot.

Cultural practices — Growers should rotate crops, avoid fields with a history of damage the previous year, and remove bolted plants because adults are attracted to the flowers.

Chemical control — Insecticide should be applied sparingly, preferably only to the perimeter of celery plantings early in the growth period.

Selected references

Hanson, A.J., and R.L. Webster. 1941. The carrot rust fly. *Wash. State Agric. Exp. Stn. Bull.* 405. 24 pp.

(Original by A.B. Stevenson)

▶ 7.20 Carrot weevil *Fig. 7.20*

Listronotus oregonensis (LeConte)

Carrot is the major host of the carrot weevil (see Carrot, 6.24) but celery is readily attacked and, under certain circumstances, it can be severely injured. Reports of damage have been mainly from the Holland and Thedford marshes in Ontario and from Quebec.

Celeriac is also a host of the carrot weevil, with damage and susceptibility similar to that of celery.

Damage The adult carrot weevil punctures and lays one to several eggs in the petioles of celery. Upon hatching, larvae either tunnel in the petiole or move to the crown or roots to feed. Celery transplants are susceptible from the time of planting. Injury varies with the number of weevils ovipositing in the plant and the stage of development of the host at the time of attack. Soil moisture also may be a factor; inadequate soil moisture increases the chance of wilting and plant death. In extreme cases, root systems are severely reduced and the plants wilt and die *(7.20).* Instances of severe injury have occurred in smaller plantings or close to overwintering sites. Normally, celery can withstand heavier attack than carrot because much of the weevil's tunneling and feeding occur on petioles that are not part of the marketed plant. Tunneling at the base of the celery stalks can make them unmarketable but serious injury occurs only occasionally.

Management

Monitoring — The carrot weevil in celery is monitored in the same manner as in carrot. Root pieces are placed in the soil near the plants to detect oviposition, or wooden plates are used to trap adults (*3.2T2*). Celery can tolerate a higher level of carrot weevil oviposition than carrot but no action threshold has been determined.

Cultural practices — Location is important; growing celery on or near sites of previously infested carrot or celery crops could increase the level of weevil attack on celery.

Chemical control — An organophosphate insecticide is registered for use against carrot weevil on celery. If celery is planted in late May or early June, the first application should be made immediately after transplanting. Celery planted later than mid-June is not likely to require an insecticidal treatment.

Selected references

Stevenson, A.B. 1977. Seasonal history of the carrot weevil, *Listronotus oregonensis* (Coleoptera: Curculionidae) in the Holland Marsh, Ontario. *Proc. Entomol. Soc. Ontario* 107:71-78.

(Original by A.B. Stevenson)

▶ 7.21 Tarnished plant bug *Figs. 7.21 a-e; 18.42b-e*

Lygus lineolaris (Palisot de Beauvois)

The tarnished plant bug occurs throughout Canada, including the Yukon. Early in the season, oviposition occurs on strawberry, alfalfa and red clover, and on such weeds as chickweed, dandelion and brown knapweed. Later breeding may occur on native hosts, such as lamb's-quarters *(Chenopodium album* L.), ox-eye daisy (*Chrysanthemum leucanthemum* L.), ragweed *(Ambrosia* spp.), fleabane *(Erigeron* spp.), plantain *(Plantago* spp.), and goldenrod *(Solidago* spp.). Besides celery, the adult feeds on other vegetables, resulting in reduced fruit set on bean, pepper and eggplant; cloudy spot blemishes on tomato fruit; necrotic spots on the florets and curd of broccoli, cauliflower and heads of lettuce; dead leaves on potato; stings and gummosis on fruit of zucchini; and foliar injury to cucumber.

Damage Adult tarnished plant bug feeding on the main stalks of celery causes lesions about the feeding sites, ranging from stings to ovoid cavities where sub-epidermal tissues have collapsed *(7.21a).* This type of injury may appear early in the season and most of it, being on petioles that will drop or be trimmed at harvest, may not be economically significant. Nearer harvest, tarnished plant bug damage (commonly called black joint) is sometimes confused with blackheart. Feeding on the leafy parts of the petiole causes necrosis of leaflets and leaf bases *(7.21c),* which in extreme cases can lead to the destruction of the entire petiole by secondary organisms such as bacteria. Just a few petioles damaged to this extent can make the entire plant unmarketable. This type of injury is partly attributable to feeding by nymphs (*7.2lb),* so any control measures that prevent plant bugs from breeding on celery usually prevent the damage from becoming economically important. In general, damage on celery varies, depending on conditions and management strategies. If left untreated, up to 80% of the crop can be unmarketable.

Identification *Lygus* bugs (family Miridae) are elongate-oblong and red-brown. The tarnished plant bug (*7.21d,e*) is distinguished by a submedian, oblique bar on the front of the head, pale or reddish lateral areas on the mid-thorax, and dense, yellow pubescence (setae) on the forewings. Adults are 4.9 to 6 mm in length and 2.3 to 3 mm in width.

Life history The tarnished plant bug overwinters as an adult in sheltered sites, such as hedgerows and under fallen leaves and crop residue. It is present throughout the growing season in Ontario and Quebec, where there are two (and a partial third) generations per year. Overwintered adults become active in mid- to late April and infest and lay eggs on clover and weed hosts. Later in the season, adults breed on various crops, weeds and native plants. Nymphs and adults feed on flowers and shoot tips. First-generation adults appear in Ontario about two months after the overwintered adults become active, which corresponds to about 214 degree-days above 9.3°C. In Quebec, first-generation adults start to appear on celery at the beginning of July regardless of the date of transplanting, nymphs are present from mid-July until the end of September, and second-generation adults appear around mid-August.

Management

Monitoring — A monitoring program in the muck soil area south of Montreal, where the amount of insecticide used on celery has been reduced by nearly 50%, is done by bi-weekly visual inspection of individual plants, beginning when they are less than 10 cm tall; 20 plants per field is sufficient and insecticide is applied if one or more bugs (adult or nymph) per plant are found. When plants exceed 10 cm in height, the threshold is six bugs (adult or nymph) per 30 plants; within three weeks of harvest, the threshold changes to four bugs (adult or nymph) per 40 plants.

In general, growers should monitor from mid-June or early July until harvest. As harvest approaches, the importance of injury increases; therefore, the number of plants examined should be increased and the action threshold should be lowered. The choice of insecticides also becomes more limited because of the requirement for specific intervals between application and harvest. The presence of insects, whether alive or dead, must be avoided in the harvested produce.

Cultural practices — Thorough, year-round weed control in and around celery fields helps to reduce the potential for plant bug infestation.

Biological control — The wasps *Peristenus pallipes* (Curtis) and *P. pseudopallipes* (Loan) are common parasites of the tarnished plant bug in Canada. Other, naturally occurring parasites have been reported but none is available commercially.

Chemical control — The currently recommended insecticide programs for celery insects in Ontario and Quebec prevent serious plant bug injury when carried out properly. Insecticidal treatments in the latter part of crop growth are most important because plant bug injury involves mainly petioles that develop at that time. Several insecticides are registered for control of plant bugs on celery in Canada. Pyrethroids are very effective on celery crops.

Selected references

Boivin, G., J.-P.R. LeBlanc and J.A. Adams. 1991. Spatial dispersion and sequential sampling plan for the tarnished plant bug (Hemiptera: Miridae) on celery. *J. Econ. Entomol.* 84:158-164.

Khoury, H., and R.K. Stewart. 1976. Chemical control of *Lygus lineolaris* (P. de B.) (Hemiptera: Miridae) on growing crops of celery and potato in Quebec. *Ann. Entomol. Soc. Quebec* 21:39-48.

Richardson, J.K. 1938. Studies on blackheart, soft-rot, and tarnished plant bug injury of celery. *Can. J. Res.* 16:182-193.

Schwartz, M.D., and R.G. Foottit. 1992. Lygus bugs on the prairies. Biology, systematics, and distribution. *Agric. Canada Res. Br. Tech. Bull.* 1992-4E. 44 pp, and Table 1 (2 pp).

Stewart, R.K., and H. Khoury. 1976. The biology of *Lygus lineolaris* (Palisot de Beauvois) (Hemiptera: Miridae) in Quebec. *Ann. Entomol. Soc. Quebec* 21:52-63.

(Original by A.B. Stevenson)

▶ 7.22 Other insect pests *Figs. 7.22a,b; 8.40b-f*

Caterpillars
- Cabbage looper *Trichoplusia ni* (Hübner)
- Celery looper *Anagrapha falcifera* (Kirby)
- Celery stalkworm *Nomophila nearctica* Munroe

A number of caterpillars occasionally infest celery. These include the cabbage looper (see Crucifers) (*8.40b-f*), the celery looper (family Noctuidae) (*7.22a)* and the celery stalkworm (family Pyralidae) (*7.22b).* These insects sometimes cause problems and damage has been reported, particularly in Newfoundland and Quebec.

(Original by A.B. Stevenson)

OTHER PESTS

▶ 7.23 Slugs *Figs. 11.27a-c*

Slugs (see Lettuce, 11.27) may be found on celery. Damage has been reported, particularly in Newfoundland and Quebec.

(Original by A.B. Stevenson)

ADDITIONAL REFERENCES

Schneider, R.W. 1985. Common names for plant diseases: celery *(Apium graveolens* L. var. *dulce* (Miller) Pers.). *Plant Dis.* 69:655.

8 Crucifers broccoli, Brussels sprouts, cabbage, cauliflower, radish, rutabaga, turnip

Figures *8.1* to *8.47*

BACTERIAL DISEASES

8.1 Bacterial leaf spot (peppery leaf spot) *Figs. 8.1a,b*

Pseudomonas syringae pv. *maculicola* (McCulloch) Young, Dye & Wilkie

Bacterial leaf spot occurs mainly on cauliflower and, to a lesser extent, on broccoli and Brussels sprouts in most crucifer-producing regions. Losses may be significant in isolated fields, but disease outbreaks occur only occasionally.

Symptoms The disease is first seen as spots or lesions on the underside of the outer leaves, which are always more seriously affected than the inner ones. Each spot is associated with a stomate. The lesions are minute at first, about 1 mm in diameter, and brown to purplish *(8.1a).* Later, a yellow halo develops around each spot as it expands. The spots grow together to form light brown, papery areas that eventually tear, giving the foliage a ragged appearance.

Infection on the veins restricts growth, resulting in puckered foliage. Extensive infection may cause leaves to drop. The pathogen may also cause small, unsightly, gray to brown spots on the curd or head *(8.1b),* especially during cool, wet weather or after a frost. The affected tissues are firm at first but may become soft if invaded by secondary organisms.

Causal agent *Pseudomonas syringae* pv. *maculicola* is a motile, Gram- negative rod with polar flagella. The cells are 0.7 to 1.2 pm in diameter and about 1.5 pm long. Colonies fluoresce under ultraviolet light on King's B medium. The organism is oxidase and arginine dihydrolase negative and produces levan from sucrose. The strains are nonpectolytic, but they cause a hypersensitive response on tobacco upon infiltration. The pathogen can be isolated from diseased tissue using routine procedures. Pathogenicity must be verified by inoculation onto the host to confirm identification.

Disease cycle *Pseudomonas syringae* pv. *maculicola* survives on infested seed and crop residues. It can remain viable in soil for two to three years. A small percentage of infested seed is sufficient to give rise to an epidemic in the field. The pathogen is spread by splashing water, washing soil and possibly by insects. Disease development is favored by cool, wet weather with an optimum temperature of about 24°C. Leaf spot is restricted and may disappear at temperatures above 30°C.

Management

Cultural practices — The use of hot- water-treated seed and the production of seedlings in seedbeds where cruciferous plants have not been grown for at least three years will help to prevent introduction of the pathogen to the field. Cauliflower should be transplanted to land where cruciferous plants have not been grown for at least three years. Incorporation of crop residues into the soil as soon as possible after harvest helps to destroy the pathogen and reduces the possibility of disease appearing in the next cauliflower crop. This is especially important where crop rotation is not practiced. If leaf spot is present during harvest, the harvested heads should be precooled rapidly by cold air or crushed ice to prevent further infection of the curd during transit to market.

Selected references

Palleroni, N.J. 1984. Family I. Pseudomonadaceae Wilson, Broadhurst, Buchanan, Krumwide, Rogers and Smith, 1917. Pages 143-213 *in* N.R. Krieg and J.G. Holt, eds., *Bergey's Manual of Systematic Bacteriology.* Vol. 1. Williams & Wilkins, Baltimore, Maryland. 964 pp.
Sutton, J.C. 1970. Bacterial leaf spot of cauliflower. Ontario Ministry Agric. Food. *Factsheet.* 2 pp.

(Original by P.D. Hildebrand)

▶ 8.2 Black rot *Figs. 8.2a-f*

Xanthomonas campestris pv. *campestris* (Pammel) Dowson

Black rot is one of the most destructive diseases of cruciferous crops worldwide. The disease usually occurs annually in most crucifer-producing regions and yield and quality losses may be high. All *Brassica* vegetables are susceptible to this disease. Several cruciferous weeds are also hosts of the pathogen.

Symptoms Black rot can appear on plants at any growth stage. On young plants, margins of cotyledons turn black and they may drop off. On true leaves, symptoms appear along leaf margins as yellow, V-shaped lesions, with the base of the V usually directed along a vein *(8.2a).* As the lesion expands toward the base of the leaf, the tissue wilts and eventually becomes necrotic *(8.2b).* The infection may move down the vascular tissue of the petiole and spread up or down the stem of the plant and into the roots. Systemic infection can produce scattered, yellow lesions on leaves anywhere on the plant.

The veins of infected leaves, petioles, stems and roots turn black as the pathogen multiplies, thus impairing the normal flow of water and nutrients *(8.2d).* Severely affected plants may lose a few or many leaves. On root crops, such as rutabaga and radish, foliar symptoms may not be visible, but blackened vascular tissues appear in the edible portion of the plant *(8.2e).* On cabbage and Brussels sprouts, infection may spread through the veins into the main stem and leaves of the head rendering the product unmarketable. Black rot infection is often followed by soft rot organisms that further reduce the quality and storage life of the product.

The presence of black veins in yellow lesions along leaf margins is diagnostic of black rot. When affected stems and petioles are cut crosswise, the vascular ring may appear black. The blackened veins also can be seen when stems are cut lengthwise. These symptoms closely resemble those of fusarium yellows, except that vein discoloration in fusarium yellows is brown. Small areas of infection and discoloration may also occur outside the vascular ring with black rot. A yellow, bacterial-laden ooze may exude from cut vascular tissues.

Causal agent *Xanthomonas campestris* pv. *campestris* is a Gram-negative, non-sporing, aerobic rod, 0.4 to 0.7 by 0.7 to 1.8 pm, with a single polar flagellum. The bacterium produces a mucilaginous, extracellular polysaccharide known as xanthan, which is responsible for plugging the xylem tissue and disrupting the flow of water and nutrients. A yellow, membrane- bound, water-insoluble pigment known as xanthomonadin also is produced and imparts a yellow color to bacterial colonies grown on certain media.

The pathogen produces yellow, convex, mucoid colonies *(8.2f)* on yeast-extract, dextrose or calcium-carbonate agar. Several semi-selective media have been developed for isolating the pathogen from seed and soil.

Disease cycle The pathogen survives on seed, which frequently is the most important source of inoculum for infection in seedling beds. Under field conditions, as few as three infected seeds in 10 000 (0.03%) can cause black rot epidemics. The pathogen also can persist in diseased crop residues in the field for up to two years before the residue is completely decayed. The organism may also survive in soil for about 40 to 60 days.

On newly emerged seedlings, black rot bacteria enter stomata along the margin of cotyledons, where the infected seed coat is often attached. The bacteria then migrate intercellularly until they reach the xylem tissue and from there spread thoughout the plant. On true leaves, the bacteria enter through hydathodes located at the ends of veins along leaf margins *(8.2c)* and from there spread throughout the plant.

The optimum temperature for growth of the pathogen is 25 to 30°C. Under these conditions, symptoms may appear on plants 7 to 14 days after infection. At lower temperatures, symptoms develop more slowly.

The pathogen spreads from infected plants and infested crop refuse and soil by splashing water, wind, insects, machinery and field workers. Long-distance spread is by infested seed and transplants.

Management

Cultural practices — Seed that has been certified as disease-free should be selected whenever possible. A hot-water treatment, if done carefully, is an effective method of eradicating black rot bacteria from seed. Seed of cabbage, broccoli and Brussels sprouts should be treated at 50°C for 25 minutes, while seed of cauliflower, kohlrabi, kale, turnip and rutabaga should be treated for 15 minutes. Seed also can be treated with antibiotics, but since these control only bacterial diseases and are usually phytotoxic, they are not generally used.

Seed should be sown in field or greenhouse seedbeds that have been fumigated or sterilized. If this is uneconomical, growers should use seedbeds in which cruciferous crops have not been grown for at least three years. Seed flats should be sterilized with steam, boiling water or a chemical disinfestant. Seedbeds should not receive run-off water from areas where cruciferous crops have previously been grown. Seeding rates should not be too high, because dense plantings can remain wet for long periods, thus favoring spread and infection by the pathogen.

Temperatures are seldom optimal for symptom expression in the seedbed, so infected plants may inadvertently be transplanted to the field where symptoms can subsequently appear. Seedlings with visible symptoms should be rogued and

destroyed, and surrounding seedlings should be observed carefully for possible symptom development. Seedling density should be reduced to allow better drying of the plants. Seedlings should not be trimmed to reduce their growth or to harden them off because the pathogen can be spread by trimming equipment.

Transplanted or direct-seeded crops should be grown where there has not been a cruciferous crop for at least three years. The crop should be kept free of cruciferous

weeds that may also be infected with the pathogen. Plantings should be worked only when the foliage is dry since the pathogen spreads readily under wet conditions. Sprinkler irrigation of diseased crops should be avoided.

If transplants are obtained from a supplier, growers should insist upon certified, disease-free material and documentation that transplants were not trimmed and that only new packaging material was used. Information such as seedlot number and source, dates of pulling and shipping, pest-control schedules, and transit conditions is also useful in judging the health of plant material and helping to identify the source of a disease problem.

Cull piles of rutabaga and other cruciferous crops should not be situated close to production fields or storages. Crop residues should be incorporated into the soil promptly after harvest to speed decomposition and to reduce the risk of pathogen spread. Manure from livestock that have been fed affected culls or land on which affected material has been fed should not be used for the production of cruciferous crops.

Resistant cultivars — Resistant cultivars are available for some types of cruciferous vegetables.

Selected references

Schaad, N.W. 1989. Detection of *Xanthomonas campestris* pv. *campestris* in crucifers. Pages 68-75 *in* A.W. Saettler, N.W. Schaad and D.A. Roth, eds., *Detection of Bacteria in Seed and Other Planting Material.* APS Press, St. Paul, Minnesota. 122 pp.

Williams, P.H. 1980. Black rot: a continuing threat to world crucifers. *Plant Dis.* 64:736-742.

(Original by P.D. Hildebrand)

▶ 8.3 Head rot *Figs. 8.3a-c*

Erwinia carotovora subsp. *atroseptica* (van Hall) Dye
Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*
Pseudomonas fluorescens Migula
(syn. *Pseudomonas marginalis* (Brown) Stevens)
Pseudomonas viridiflava (Burkh.) Dowson

Head rot is frequently a major constraint to successful production of broccoli in Ontario, Quebec and the Atlantic provinces. Losses can exceed 30% and may be as high as 100%. All of the causal bacteria have host ranges that include several other types of vegetable crops, such as carrot, lettuce and potato.

Symptoms (For symptoms and signs of *Erwinia* spp., see Potato, bacterial soft rot and blackleg, 16.2, 16.3; for those of *Pseudomonas viridiflava,* see Lettuce, pseudomonas diseases, 11.3.) Symptoms first appear after periods of rain when heads have remained wet for several days. Areas on heads colonized by pathogenic strains of *P. fluorescens* appear water-soaked where water has formed in a film *(8.3a).* This is in contrast to unaffected areas, where the waxy surface of the florets imparts a gray-green color and causes the water to form in beads. Small, black lesions frequently develop in association with the stomata within the water-soaked areas of the sepals and pedicels of the florets. The lesions are raised and a dark discoloration, initially associated with the guard cells, spreads to the surrounding tissues. Decay develops on these affected florets or on young, tender florets associated with meristematic areas of the head. During long periods of continuous wetness, decay spreads rapidly and extensively, resulting in sunken areas on the head (*8.3b,c*).

When heads are colonized by saprophytic strains of *P. fluorescens,* water-soaking symptoms appear but heads fail to decay. Similarly, decay remains localized with minimal water soaking when heads are attacked by strains of *P. viridiflava* or *Erwinia* spp., but decay and water soaking may be more extensive when these bacteria are present in combination with saprophytic strains of *P. fluorescens.*

On broccoli florets, symptoms of water-soaking followed by soft decay are diagnostic of this disease when pectolytic strains of *P. fluorescens* are present. Occasionally, colonization of heads by saprophytic strains of *P. fluorescens* may be suspected if heads become water-soaked but fail to decay despite prolonged periods of wetness. If only a few florets are decayed and water soaking is minimal, the presence of wound pathogens such as strains of *P. viridiflava* or *Erwinia* spp. may be suspected.

Causal agents Strains of *Pseudomonas fluorescens* hiovars II and IV, also referred to as *P. marginalis,* have been identified as the primary cause of head rot, but occasionally head rot is caused by interactions of saprophytic- strains of *P. fluorescens* with other soft-rotting bacteria, such as *Erwinia carotovora* subsp. *carotovora, E. carotovora* subsp. *atroseptica,* and *P. viridiflava.*

For detailed descriptions of *E. carotovora* subsp. *atroseptica* and *E. carotovora* subsp. *carotovora,* see Potato, bacterial soft rot, 16.2, and blackleg, 16.3; for *P. fluorescens* and *P. viridiflavida,* see Lettuce, pseudomonas diseases, 11.3. Differentiation among these bacteria is relatively easy based on a few presumptive characteristics. Single bacterial colonies on King's B medium

should be obtained from water-soaked or decayed tissue. Fluorescence is determined on King's B medium and presence of pectolytic enzymes is determined on the modified pectate medium of Cuppels and Kelman (see Schaad, Additional references) to which no crystal violet has been added. Biosurfactant activity is detected by mixing colonies on King's B medium in a drop of water with a transfer loop, then transferring the loop to a drop of water on a plastic petri dish. Spreading of the water drop indicates a positive reaction. Pathogenic strains of *P. fluorescens* and *P. viridiflava* fluoresce on King's B medium and cause shallow pits on the pectate medium, but only *P. fluorescens* is biosurfactant positive. Some saprophytic strains of *P. fluorescens* are biosurfactant positive but do not cause pitting. Strains of *Erwinia* spp. are nonfluorescent, do not produce biosurfactant and cause deep pits. Pectolytic activity can also be determined by observing maceration of inoculated potato slices by the various bacterial isolates.

Disease cycle The epidemiology of pathogenic strains of *Pseudomonas fluorescens* is not well understood. The bacterium survives in the soil and may also be present in pond and stream water. During periods of heavy rainfall, the pathogen is splash-dispersed from the soil onto broccoli heads. If the heads remain wet for several days, the bacteria multiply and release a biosurfactant, known as viscosin, and pectolytic enzymes into the drops of water. The biosurfactant reduces the surface tension of water, which then wets the waxy surface of the florets. The biosurfactant enables the bacteria to enter stomata and the surrounding tissues and enhances the action of the pectolytic enzymes. If the heads dry, further ingress of the bacteria and decay do not occur. However, the affected areas on heads are easily rewetted in subsequent wet periods, and decay may develop rapidly if wetness and high temperatures persist for several days.

Head rot develops most rapidly when temperatures are high. The optimum temperature for bacterial growth is 28°C, but growth may occur slowly at temperatures as low as 1 to 2°C. Frost injury in the field and infection by downy mildew may predispose the heads to colonization and decay by the bacteria. Insects such as the tarnished plant bug and flea beetles may cause wounds on the florets that predispose them to infection. Excessive soil nitrogen encourages lush, tender growth that reduces air movement and is very susceptible to bacterial attack. Boron and calcium deficiencies may predispose broccoli heads to infection.

Management

Cultural practices — Production practices that enhance air movement through the crop, such as wider plant spacing and planting rows in the direction of prevailing winds, may be beneficial. Successive plantings should be well away or at least upwind from previous plantings to avoid rain-driven dispersal of inoculum. Excessive application of nitrogen fertilizer should be avoided. Calcium and boron should be maintained at adequate levels.

Heads should be cooled and placed into cold storage soon after harvest. Heads that show signs of severe water soaking in the field are more susceptible to decay in storage.

Resistant cultivars — Cultivars that produce heads above the foliage dry more quickly and appear to be more resistant. Shogun and several breeding lines appear to have some tolerance. No cultivars are immune.

Chemical control — Excessive applications of insecticides or fungicides with surfactants during heading should be avoided since surfactants can enhance bacterial infection.

Selected references

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(Original by P.D. Hildebrand)

▶ 8.4 Scab *Figs. 8.4a-c*

? *Streptomyces scabies* (Thaxt.) Waksman & Henrici (syn. *Actinomyces scabies* (Thaxt.) Güssow)

Scab occurs on the edible fleshy root or enlarged hypocotyl of radish, rutabaga and turnip. Although the disease does not affect yield, it is economically important because scabs on the roots reduce marketability. The disease is not as severe on root crucifers as on potato, red beet and carrot (see Potato, common scab, 16.5; Beet, scab, 5.1; and Carrot, scab, 6.4). It occurs only sporadically in most regions where these crops are grown.

Symptoms On radish *(8.4c)*, small scale-like spots, each about 1 mm in diameter, appear after hypocotyl enlargement begins. Individual spots may reach 1 to 1.5 cm in diameter. The edges of the spots are raised, while the centers are sunken and pitted. The centers of young scabs initially appear white, but infection by secondary organisms may cause the scabs to discolor and decay.

On rutabaga *(8.4a,b)* and turnip, lesions are circular, often reaching 1 to 1.5 cm in diameter, and are scattered over the surface of the root, or frequently grow together to form a corky band around the root just beneath the soil line. Affected tissues may consist of a tan-colored, superficial or raised layer, or tissues may become pitted and dark following secondary decay. The superficial lesions of scab rarely cause reductions in yield, but even a single lesion is cosmetically unacceptable and may render a root unsaleable.

Causal agent (see Potato, common scab, 16.5) The identification of *Streptomyces scabies* as the cause of scab in crucifers is provisional.

Disease cycle (see Potato, common scab, 16.5)

Management

Cultural practices — (see Potato, common scab) Scab on crucifers is often more severe following a potato crop. Although the pathogen is persistent in field soils, rotation is an important means of reducing inoculum levels. A rotation of potato, grain and one year of hay or another green manure crop, followed by a cruciferous crop, is effective. The green manure crop should be worked down early to facilitate rapid decomposition, and the field kept free of weeds during the fall. Green manure crops encourage the growth of microorganisms that are antagonistic to *Streptomyces scabies.* Fields in sod intended for crucifer production should be worked down early in the year before sowing. Adequate soil moisture levels should be maintained, especially during periods of rapid root growth. However, the use of excessive irrigation over many seasons should be avoided as it may lead to increased levels of other pathogens, such as the clubroot organism.

Resistant cultivars — Some European radish cultivars, such as Sar Katra and Large Scharlakenrode, have partial resistance to scab but are not widely grown in North America.

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(Original by P.D. Hildebrand)

FUNGAL DISEASES

▶ 8.5 Alternaria diseases *Figs. 8.5a,b*

Black leaf spot
Gray leaf spot
Alternaria brassicae (Berk.) Sacc.
Alternaria brassicicola (Schwein.) Wiltshire
Alternaria raphani Groves & Skolko

Three species of *Alternaria* can cause serious losses in cruciferous crops in Canada. *Alternaria brassicicola* has been detected in 33% of samples of garden crucifer seed in Saskatchewan and cited as a potential threat to the canola (rapeseed) crop in the region. In another study, *Alternaria brassicae* affected 60 to 100% of cabbage plants in several fields in Ontario.

Alternaria brassicae and *A. brassicicola* infect a wide range of cruciferous vegetables, including Brussels sprouts, cabbage, cauliflower, Chinese cabbage, kohlrabi, kale, turnip and rutabaga. *Alternaria raphani* can infect many of these crops but generally it affects only radish.

Symptoms Leaf spotting is the major symptom associated with *Alternaria* infection. Pre- and post-emergence damping-off and damage to the inflorescence of seed crops and to seed can also occur. Pinpoint spots on leaves enlarge to become circular lesions several centimetres in diameter with target-like concentric rings. Lesions are initially yellow-brown and later turn brown to black *(8.5a,b).* Younger leaf tissue is less susceptible to infection than older tissue.

The pathogens sporulate abundantly on foliar lesions and lesion centers may become thin, papery and fall out to give a "shot-hole" appearance. Lesions often grow together leading to large necrotic areas and early leaf drop as the disease progresses. Lesions caused by *A. brassicae* tend to be small and light brown to brownish-gray and are referred to as gray leaf spot. *Alternaria brassicicola* lesions are large, olive-gray to grayish-black and are referred to as black leaf spot. Elongate lesions may occur on petioles, stems and flowers. Small, circular, brown lesions may appear on flower pedicels and calyces. Small dark spots may form on young pods and eventually cause pod distortion and premature shattering in seed crops. In infected maturing pods, the diseased seeds are shrivelled and germinate poorly.

Infected seed may appear healthy with no obvious lesions, although spores may be present on the seed surface and mycelium within the seed. Infected seedlings have circular, somewhat sunken, dark brown to black spots on the cotyledons and

dark lesions on the hypocotyls, which reduce plant growth and may resemble the symptoms of wirestem caused by *Rhizoctonia solani.* Infected cauliflower curds have sunken, velvety, dark brown spots with large numbers of spores. Affected broccoli heads have a brown discoloration that begins at the margins of individual flowers and flower clusters. Lesions on cabbage heads placed in storage may enlarge and undergo enhanced rot caused by invasion by secondary fungi and bacteria.

Causal agents *Alternaria* species are unspecialized parasites with simple nutritional requirements and a saprophytic stage outside the host. *Alternaria brassicae* and *A. brassicicola* are not host specific, so crossinfection may occur between different crop types. A resting stage consisting of chlamydospores has been reported for *A. raphani,* and microsclero- tia are known for A. *brassicae.* Although all three fungi can survive in susceptible weeds and perennial crops, diseased residue is a major source of inoculum and a means of perennial perpetuation of these pathogens.

These *Alternaria* species lack a sexual state. The mycelium is branched and septate, and conidia are produced on conidiophores that are generally pale brown or brown and arise separately or in clusters. The conidia are mostly in chains and arise through pores in the conidiophore wall. They are typically ovoid or obclavate with transverse and longitudinal septa. The conidiophores of *A. brassicae* are up to 170 pm long and conidia are brown with beaks up to half as long as the entire conidium. The spore body length is 96 to 114 pm and the spore beak length is 45 to 65 pm. The conidia occur in short chains of two to three, with more cross septa than in conidia of *A. brassicicola. Alternaria brassicicola* produces shorter conidiophores, up to 70 pm long, and dark conidia that, in culture, are generally cylindrical to oblong with a beak that is one-sixth the length of the entire conidium. The spores are generally smaller than those of *A. brassicae***,** measure 45 to 55 pm and are produced in chains of up to 20 or more. They often have a central pore that is readily visible in the cross walls. *Alternaria raphani* resembles *A. brassicae* but it forms chlamydospores, the conidia are in chains of up to six, each measuring 60 to 83 pm, and the conidial beak is intermediate in size, generally 10 to 25 pm in length.

Species of *Alternaria* can be readily isolated from diseased tissue using routine procedures. Cultures of *A. brassicicola* on potato-dextrose agar grow faster and develop a black, sooty color compared to cultures of *A. brassicae, which* have a light-colored mycelium. *Alternaria raphani* does not sporulate as abundantly as the other two species and forms a white, cottony mycelium that also develops many irregular forms, varying from thick, dark, heavily septate mycelium to one with characteristic chlamydospores. On malt agar, the aerial mycelium is cottony and white to deep olive-gray; the submerged mycelium is colorless to olivaceous-black.

Disease cycle Spores are produced in large numbers and may be spread throughout fields by wind and splashing rain or on equipment, humans and livestock. Dissemination also occurs as wind-blown, diseased plant tissue, although the chief means of spread to new fields is through use of infested seed. Transfer of the fungus from infested seedling beds to the field in soil adhering to the roots of transplants is also possible.

Alternaria brassicae and *A. brassicicola* require liquid water and temperatures of 15 and 25 °C, respectively, for 16 hours to initiate infection. Subsequent disease development occurs after two to three days, although alternating wet and dry periods will restrict infection by both species. At least 12 hours of continuous high humidity in excess of 90% RH and temperatures above 14°C are necessary for abundant sporulation. At 10°C, *A. brassicae* produces numerous lesions on host tissue after four days, whereas *A. brassicicola* does not infect host tissues under these conditions.

Management

Cultural practices — Hot-water seed treatment (see black rot, 8.2) reduces or eliminates both internal infection and external infestation of seed by *Alternaria* spp.. Long rotations with non-cruciferous crops, incorporation of diseased crop residues into the soil, elimination of cull piles, eradication of cruciferous weeds, and avoidance of overhead irrigation during head development all will reduce inoculum levels. Seedbeds and successive crops of crucifers should be located away or upwind from existing cruciferous crops to avoid wind-borne inoculum. Also, seedbeds should be kept disease-free to prevent the spread of disease. Control of alternaria leaf spot on cabbage heads in the field is essential if the crop is intended for prolonged storage.

Chemical control — Protective fungicide seed treatments control only fungal spores on the seed surface. Regular applications of protective fungicide sprays beginning in midseason, particularly if conditions are warm and wet, will arrest disease development.

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(Original by R.F. Cerkauskas)

▶ 8.6 Blackleg *Figs. 8.6a-d*

Phoma lingam (Tode:Fr.) Desmaz. (teleomorph *Leptosphaeria maculans* (Desmaz.) Ces. & de Not.)

Blackleg is a destructive fungal disease of many cruciferous crops. Although blackleg can spread rapidly in a field, its progress is not as explosive as that of black rot caused by *Xanthomonas campestris* (see black rot, 8.2).

Blackleg is a serious disease of canola, but has become less important in cruciferous vegetables because of successful disease management practices in the seed industry throughout the world.

Symptoms Plants may become infected in the seedbed or at any time in the field. Symptoms in plants in the seedbed occur two or three weeks before transplanting. Seedling infection is first seen on cotyledons or on the first true leaves *(8.6a).* Seedlings may be killed, while other plants may only have dead, withered cotyledons. Inconspicuous bluish lesions may appear on stems of older plants at the cotyledon scar. Later, an elongate, light brown, sunken area with a purplish or black margin forms on the stem near the soil line. As the lesion gradually extends upward and downward, the stem becomes girdled and blackened. Numerous, small, dot-like, black pycnidia form in these lesions. Stem lesions may extend below the soil line *(8.6b),* causing dark cankers and death of the fibrous roots. Severely infected plants wilt and may lodge. On rutabaga and turnip, cankers form on the fleshy root *(8.6c,d)* and a dry rot may occur in storage. Inconspicuous, circular, light brown to gray spots form on leaves. Large numbers of conidia, often in pinkish gelatinous coils, form in these lesions. Infection of seed pods and seed may occur on crucifer seed crops.

Causal agent Pseudothecia of the sexual state are globose, black, 300 to 500 µm in diameter, with protruding ostioles. Each ascus measures 80 to 125 by 15 to 22 µm, has two distinct walls and contains eight ascospores, each measuring 35 to 70 by 5 to 8 µm. Ascospores are biseriate, cylindrical to ellipsoidal, mostly rounded at their ends, yellow-brown, and guttulate. Pseudoparaphyses are filiform, hyaline, and septate.

Pycnidia of *Phoma lingam* on stems and leaves are of two types: Type I pycnidia are sclerotioid, initially immersed in the host tissue, but eventually erupt, and are variable in shape, 200 to 600 µm across, with narrow ostioles; Type II pycnidia are globose, black, and 200 to 600 µm in diameter.

Conidia are hyaline, short, cylindrical, mostly straight, some curved, guttulate with one guttule at each end of the conidium, unicellular, and 3 to 5 by 1.5 to 2 µm.

The pathogen can be isolated from surface disinfested segments of tissue plated on V-8 agar with 40 µg/mL rose bengal and 100 µg/mL streptomycin. To detect the pathogen in crucifer seed, the seed samples should be placed on moist filter paper and incubated at 20°C in darkness for one day. The seeds should then be transferred to a freezer at -20°C for one day, which prevents seed germination but does not affect the development of the pathogen. The seeds should be incubated further at 20°C with a 12-hour photoperiod of fluorescent light. After 7 to 10 days, pycnidia will develop on infected seeds.

Disease cycle *Phoma lingam* may survive for at least four years in seed and for at least three years in crop residues in the field. The pathogen infects seedlings, causing lesions in which pycnidia are formed. Conidia exude from these pycnidia in long, pink to lilac-colored coils. The conidia are splashed to nearby cruciferous plants, where they germinate and cause new infections. Wet, rainy weather aids in the spread of conidia and reinfection.

The teleomorph state has been found in several areas of Canada, the United States and elsewhere. Ascospores become air-borne and may be transported long distances, causing infections that cannot be traced to infested seed or poor rotation practices.

Management

Cultural practices — Seeds should be hot- water treated as for black rot. A four-year crop rotation should be practiced in seedbeds and fields. Seedbeds should be thoroughly inspected and diseased plants should be rogued. After lifting, transplants should not be sprayed or dipped in water before planting. Cruciferous crops should not be planted adjacent to, or downwind from, fields in which crucifers, including rapeseed/canola, were grown in the previous year, because surface water, wind and rain may spread the fungus from one field to another. Growers should choose fields with good water drainage and air circulation, keep fields free of cruciferous weeds, avoid cultivating plants when they are wet and incorporate crop residues promptly after harvest to speed decay. Manure from animals fed infected plants should not be used as a soil amendment.

Resistant cultivars — Horseradish is resistant to blackleg. Cabbage, Chinese cabbage, Brussels sprouts, most canola and some radish and rutabaga cultivars are susceptible. Cauliflower, broccoli and some canola and turnip cultivars are moderately susceptible. Some turnip, rutabaga, radish and mustard cultivars are only slightly susceptible or immune.

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(Original by R.W. Delbridge)

▶ 8.7 Black root *Fig. 8.7*

Aphanomyces raphani Kendrick

Black root of radish is a minor soil-borne fungal disease that is known to occur in Ontario, Quebec and British Columbia, where losses ranging from 10 to 50% and affecting at least one hectare have been reported. The pathogen attacks radish primarily but also may infect broccoli, Brussels sprouts, cabbage, Chinese cabbage, cauliflower, kale, kohlrabi, rutabaga and turnip.

Symptoms Radish plants are susceptible from the seedling to mature stages, although seedling infection is rarely observed in the field. Symptoms on seedlings consist of dark, water-soaked lesions on the lower hypocotyl. Infected roots, stems, petioles and cotyledons turn black and collapse as the disease develops, often resulting in damping-off. Oospores are present in the invaded tissue, especially in the cortical tissues of the secondary roots in seedlings and older plants, and may even occur in plants without symptoms. In older radish plants, the first symptoms consist of bluish-gray to black areas on the skin where secondary roots, growth cracks or wounds occur. The dark lesions may grow together, resulting in constricted rings around the root and reducing growth. This may lead to deformities and stunting of the tops. The blackening extends deep into the root (*8.7*)**.** The rot is dry initially, but as the disease develops, secondary soft-rotting organisms may cause eventual root disintegration. Hypocotyl infection of seedlings of the other crucifers leads to typical damping-off symptoms.

The black or gray discoloration extending deep into the root tissues distinguishes black root from the more superficial, scaly brown lesions associated with rhizoctonia diseases. As well, the mycelium of *Aphanomyces raphani* is non-septate and hyaline.

Causal agent *Aphanomyces raphani* is closely related to *A. cochlioides* (see Beet, aphanomyces root rot, 5.2), and species differentiation is based mainly on oogonial size. The development and life-cycle of the two fungi are similar, although the size and shape of fungal structures differ. The hyphae of *A. raphani* are 8.2 to 11.3 µm in diameter, nonseptate, coarse and profusely branched at right angles, and the oogonia are 32 to 45 µm in diameter. Oospores of *A. raphani* are hyaline and 21.4 to 29 µm in diameter.

Saprophytic bacteria are often associated with diseased host tissue, which therefore should be washed thoroughly and placed in distilled water overnight at room temperature. The identity of the fungus can be verified by zoospore discharge from sporangia produced on pieces of tissue that have been treated in this way. Oospores characteristic of the pathogen are produced in abundance in the secondary roots of radish and other crucifers invaded by the fungus. A partially selective medium containing 150 pg/mL streptomycin sulfate and 10 µg/mL benomyl in radish agar may be used to enhance recovery of *Aphanomyces* spp. from infected plant tissue (see Selected references, Humaydan & Williams). Alternatively, other selective media may be used (see Beet, aphanomyces root rot).

Aphanomyces raphani grows rapidly on artificial media, such as potato- dextrose agar, held at 23°C, often reaching 90 mm in diameter within nine days. Colonies are cream-colored, moist and dense with a tough mycelial mat that is free from aerial hyphae. Cultures are best maintained on radish agar. Abundant oospores and zoospores are produced on this medium. Growth occurs from 12 to 32°C, with the optimum between 18 to 24°C.

Disease cycle The disease is favored by high temperatures, with infection of radish seedlings occurring at 16 to 32°C with a maximum at 27°C. High soil moisture or free water in the soil is necessary for penetration by the motile zoospores. Oospore germination in soil is stimulated by the presence of radish seedlings.

Oospores are produced in large numbers in the secondary roots of many crucifers and in host residues or soil particles that accompany seed. They allow the fungus to persist for long periods and subsequently to infect hosts. Mycelium and zoospores are incapable of prolonged survival in soil but the fungus may subsist as mycelium in volunteer or seed plants. Dissemination is by splashing water or by movement of surface water carrying oospores or zoospores to other plants or neighboring fields. Inoculum dispersal is also possible by infected plants, wind-blown soil or host residue and by tools, agricultural machinery and workers.

Management

Cultural practices — Diseased crop residues should be worked well into the soil and good soil drainage provided. Inoculum levels of *Aphanomyces raphani* in the soil may be reduced by following a four-year rotation with non-cruciferous crops and controlling cruciferous weeds. Some cole crops may escape infection for several weeks after transplanting.

Resistant cultivars — Radish cultivars with resistance to black root include Belle Glade, Fancy Red, French Breakfast and Fuego.

Chemical control — Soil disinfestation with fumigants, although effective, is not economical.

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(Original by R.F. Cerkauskas)

▶ 8.8 Clubroot *Figs. 8.8a-c*

Plasmodiophora brassicae Woronin

Clubroot is a major fungal disease of cruciferous crops worldwide. In Canada, it is a problem primarily in British Columbia and eastern Canada, where entire crops have been destroyed by the disease. Clubroot occurs on broccoli, Brussels sprouts, cabbage, cauliflower, turnip, rutabaga and radish. It can also attack many native and weed species of the mustard family and non-cruciferous genera such as *Agrostis* (bentgrass), *Dactylis* (orchardgrass), *Holcus* (velvetgrass), *Lolium* (ryegrass), *Papaver* (poppy) and *Rumex* (sorrel, dock).

Symptoms The disease can progress considerably before above-ground symptoms are visible. Infected roots enlarge to form galls that vary in size and shape. On crops such as radish, turnip and rutabaga, in which the fleshy root is an enlarged hypocotyl, galls form on the taproot or on secondary roots. These galls are frequently globular or spherical and can be large *(8.8c).* On hosts with fibrous root systems, such as cabbage, cauliflower and broccoli, club-like, spindle- shaped swellings form on individual roots *(8.8b).* These swellings may be isolated and occupy only parts of some roots or they can grow together to occupy the entire root system *(8.8a).* Severely distorted roots are unable to absorb minerals and water. Consequently, top growth is stunted and lower leaves may turn yellow and drop off. Younger foliage may turn bluish and wilt during the day but recover at night. As the disease progresses, the root swellings are often invaded by secondary organisms, causing decay and death of the plant. Seedlings can become infected in seedbeds. Usually, there are no visible symptoms on the top growth, but the tiny, club-like swellings can be seen on the roots. Herbicide injury and hard swellings of unknown origin called hybridization nodules on turnip, rutabaga and rapeseed/canola may be confused with clubroot.

Causal agent *Plasmodiophora brassicae* is a simple fungus that produces a multinucleate mass of protoplasm called a plasmodium, which lacks a cell wall and definite mycelium. Resting spores are uninucleate, hyaline, spherical and measure 2.4 to 3.9 µm. Zoospores are ovoid or variably shaped, biflagellate and 2.5 to 3.5 µm. Zoosporangia average 8 µm. The fungus cannot be cultured *in vitro* but can be grown in callus cultures established from root galls.

Disease cycle In the presence of susceptible roots, resting spores of the fungus germinate to produce motile zoospores that swim in free water and penetrate the surface of the root hairs. The fungus develops into a sporangial plasmodium and subsequently divides into multinucleate portions that develop into zoosporangia. These are released from the host through pores dissolved in the epidermal cell wall, and four to eight secondary zoospores are liberated from each zoosporangium. Some of these zoospores fuse in pairs before germinating and infecting the host.

Plasmodia in the roots cause cells to enlarge abnormally and divide repeatedly, resulting in gall formation. Infected cells are distributed in small groups among healthy cells within the gall. As the plasmodia mature, they transform into masses of resting spores that are released back into the soil when secondary organisms decay the galls. The plasmodium-infected galls use nutrients required by the plant and interfere with the absorption and translocation of nutrients and water in the roots, causing stunting and wilting of the plant. Decay of the galls releases toxic substances that are partly responsible for the wilt symptoms. The fungus invades young root hairs readily, but wounds are necessary for infection of thickened roots and underground stems, although stems also may be invaded through leaf scars.

Plasmodiophora brassicae is disseminated by drainage water, soil that clings to farm equipment, shoes, tools, infected transplants, and contaminated manure and irrigation water. The disease is usually more severe on wet, acidic soils. Repeated crucifer production leads to a rapid build-up of the pathogen. Resting spores may remain viable in the soil for more than 18 years. At least nine pathotypes of the pathogen have been identified. The role of non-cruciferous plants in the epidemiology of the disease is not known.

The optimum mean daily soil temperature for disease development ranges from 19.5 to 23°C. Soil temperatures of at least 16 to 21 °C are required for resting spore germination. Moisture levels above 50 to 70% of the maximum water-holding capacity (about –20 to –15 kPa) of the soil are needed for infection. Clubroot tends to be more severe in soils with a pH of less than 7.0.

Management

Cultural practices — Good soil drainage and the maintenance of a high pH by regular application of lime help to reduce disease incidence. The degree of control may depend on soil pH, which is probably the most important factor influencing disease development. High concentrations of calcium and magnesium may give control below pH 7.2, whereas low calcium and magnesium concentrations may permit disease development above pH 7.2. Calcitic lime is usually more effective than dolomitic lime. If soils are low in magnesium, however, dolomitic lime is preferable. Finely ground lime is more reactive than coarse granules and alters the pH more quickly. Increasing the pH often results in boron deficiency on coarse-textured soils (see boron deficiency, 8.23). Application of boron as a foliar spray or in the transplanting water will alleviate this potential problem.

Long, five- to seven-year crop rotations between cruciferous crops are necessary, and cruciferous weeds must be controlled. The movement of soil or plant material from infested areas into clean fields must be avoided and manure from animals fed infected cull plants or pastured in infected crops should not be used. Transplants should be produced on non-infested soil and the field site should be well- drained and have no history of clubroot. Resting spores of *Plasmodiophora brassicae* may be present in irrigation water and sediment where run-off from infested fields collects in irrigation ponds. Care should be taken to use irrigation water that is not contaminated by the clubroot fungus.

Resistant cultivars — Multi-race resistant cabbage lines and a resistant cabbage cultivar, Richelain, are available. In the Atlantic region, 'Kingston' rutabaga is resistant to all known races and 'York' rutabaga is resistant to several but not all races. The broccoli cultivar Oregon CR1 is resistant to several races but is poor in horticultural quality.

Chemical control — Growers should fumigate seedbeds if disease-free beds are not available. Disease incidence in field plantings can be controlled by using a fungicide in the transplant water.

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(Original by R.W. Delbridge)

▶ 8.9 Damping-off *Fig. 8.9*

Pythium debaryanum of authors, not R. Hesse
Pythium ultimum Trow
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Damping-off can cause serious losses in cruciferous crops in seedling trays in greenhouses *(8.9)* and in direct-seeded plantings in fields. The pathogens causing this disease are common in field soils and can infect many types of vegetable crops.

For information on damping-off, see rhizoctonia diseases in this chapter, 8.13. Also see Celery, damping-off, 7.4.

(Original by P.D. Hildebrand)

▶ 8.10 Downy mildew *Figs. 8.10a-e*

Peronospora parasitica (Pers.:Fr.) Fr.

Downy mildew is a problem on crucifers throughout Canada. Losses in each of broccoli, Brussels sprouts and cauliflower in the lower Fraser Valley, British Columbia, in 1965 and 1966 were estimated at 5 and 2%, respectively. In Ontario, 11 to 30% of cabbage and radish plants in several fields were affected during one survey. In Quebec and Newfoundland, respectively, 10% and 70 to 100% of rutabaga plants in two fields surveyed were affected, while in Nova Scotia 25% of rutabaga plants were diseased.

Downy mildew occurs frequently on broccoli, Brussels sprouts, cabbage, rutabaga, turnip, Chinese cabbage, cauliflower, kale, kohlrabi and radish. Other cruciferous plants such as Arctic draba *(Draba lactea* Adams), dame's-violet *(Hesperis matronalis* L.), false flax *(Camelina microcarpa* Andrz. in western Canada and *Camelina sativa* (L.) Crantz in eastern Canada), garden cress *(Lepidium sativum* L.), gray tansy mustard *(Descurainia richardsonii* (Sweet) O.E. Shulz), pepper grass *(Lepidium densiflorum* Schrad.), scurvy grass *(Cochlearia officinalis* L.), shepherd's-purse *(Capsella bursa-pastoris* (L.) Medic.), tumbling mustard *(Sisymbrium altissimum* L.), wallflower *(Cheiranthus cheiri* L.), and various northern plants also may be hosts for *Peronospora parasitica.*

Symptoms Infection can occur at any stage of growth *(8.10a).* Seedling infection causes the formation of discolored spots on cotyledons. Sporulation of the pathogen may occur on the cotyledon undersurface and hypocotyl. Cotyledons later turn yellow, shrivel and die. At this stage, the fungus is capable of becoming systemic and quiescent.

Initial foliar symptoms consist of discrete, angular, yellow areas on the upper surface of the leaf and fluffy, white, sparse, patchy mycelial growth *(8.10b)* on the undersurface. The affected areas enlarge under moist conditions and turn tan and papery *(8.10c).* In cabbage, systemic invasion of the stem may occur after infection of the lower leaves during the growing season. The fungus may then slowly invade the head leaves and sporulate after the cabbage has been stored. Systemically invaded tissues such as the midrib and blade turn yellowish, then grayish-black and necrotic. Affected cabbage tissue becomes very susceptible to attack by secondary bacteria and fungi. The fungus can cause numerous sunken black spots of varying size on the head.

Cauliflower infection may extend to the head both in the field and in storage. A blackening similar to that observed on cabbage may be evident on curds *(8.10d).* Abundant fungal sporulation and rotting caused by secondary organisms, such as bacteria, often follow the discoloration of cauliflower curds. On broccoli heads, invasion of the fungus cannot be detected by external symptoms. Brown to black streaks may appear in the vascular system of the upper part of the main stalk and branches that lead to the florets. Fungal sporulation followed by rotting occurs on Brussels sprouts as well.

Radish, rutabaga and turnip roots can be invaded systemically, resulting in an irregular, internal brown to black discoloration extending downward from the crown or the soil line. Diseased radish has a brown to black, blotchy epidermal discoloration about half-way down the side of the root *(8.10e).* Often, there is a slight russeting of the epidermal tissue and some cracking. In more advanced stages for all three crops, minute cracks or root splitting may occur. Internal tissue remains firm unless secondary organisms enter and cause decay.

Causal agent *Peronospora parasitica* is an obligate parasite that forms nonseptate mycelium, which occupies the intercellular spaces of the host and forms haustoria within the cells. The hyaline, dichotomously branched sporangiophores emerge through host stomata, bearing hyaline, elliptical, single, terminal sporangia, 16 to 20 by 20 to 22 µm, which germinate by means of a germ tube. Sexual oogonia and antheridia form as the host senesces. The oospore that develops is spherical, 26 to 45 µm in diameter, thick-walled, and yellow-brown. Temperature and relative humidity may influence the morphology of sporangia and sporangiophores, and neither sporangium size and shape nor host range is a reliable character. *Peronospora parasitica* can produce oospores in a range of *Brassica* species and cultivars. However, there is evidence that different isolates consisting of mating types 1 and 2 are required for oospore formation. Evidence suggests that field populations of the fungus are potentially highly variable.

Disease cycle *Peronospora parasitica* exhibits some parasitic specialization at the generic, specific and lower taxonomic levels of the host. For example, turnip isolates may be unable to infect radish or rutabaga. However, the host range of specific isolates of the pathogen from crucifers may be variable and unrelated to the taxonomy of the host family. Also, resistance to the disease increases with the age of the host. Historically, differences in host range were used to delimit distinct species; however, *P. parasitica* is now regarded as a single aggregate species. Although most cruciferous weeds are susceptible to downy mildew, it is not known whether they serve as hosts for the strains of *Peronospora* that occur on cruciferous crops.

Cool, moist conditions favor disease development. Temperatures of 10 to 15°C and abundant moisture on leaves from dew, drizzling rain or heavy fog are optimum for epidemic development. Sporulation, germination and reinfection may occur within four to five days.

The fungus may survive in a latent or quiescent state within systemically infected plants. This may be a source of subsequent lesions on flowering parts like cauliflower curds. The fungus survives between crops as oospores that are formed when the host undergoes senescence. Infection of seedlings in soil contaminated with oospores from decomposed host tissue is possible. Oospores are present in roots and may contaminate seeds also. The survival of sporangia is greatly reduced in dry soil and at low temperatures. Sporangia are disseminated by wind and splashing rain. Mycelium in the seed coats and oospore contamination of seeds are other means of long-distance dispersal.

Management

Cultural practices — Control in the seedbed is very important and includes the use of clean, well-drained soil that has been free from crucifers for the previous two years, avoidance of excessive overhead irrigation to keep seedlings and leaf surfaces as dry as possible, prevention of overcrowding of seedlings and promotion of ventilation within the seedbed by regulation of plant density. Fertilizer can be used to stimulate growth to enable seedlings to outgrow infections. Removal of crop residues from seedbeds should be routine because the oospores can survive in dried foliage.

Resistant cultivars — Several resistant or tolerant hybrid cultivars of broccoli are available. These include Arcadia, Cindy, Citation, Esquire, Eureka, Green Belt, Hi-Caliber, Marathon, Mariner, Pinnacle, Samurai, Sprinter and Zeus.

Chemical control — Preventive spraying in the seedbed with protectant foliar fungicides may be necessary if environmental conditions favor disease development. This may prevent new infections but it will not eradicate established lesions. A regular spray program may be necessary after transplanting or direct seeding in the field if mildew persists.

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(Original by R.F. Cerkauskas)

▶ 8.11 Fusarium wilt (yellows) *Figs. 8.11a,b*

Fusarium oxysporum f. sp. *conglutinans* (Wollenweb.) W.C. Snyder & H.N. Hans.

(syn. *Fusarium conglutinans* Wollenweb.)

Yellows or fusarium wilt was first reported in 1899 on cabbage in New York State. The disease was first observed in Canada in Ontario in 1931. Losses may be significant on a wide range of Cruciferae during warm growing seasons or where resistant cultivars are not commercially available. During extensive surveys of diseases on vegetable crops in Ontario in 1967, the disease was observed in 5 of 24 fields of early heading cabbage, with a range of 10 to 95% of the plants affected.

Cruciferous vegetables that are susceptible to yellows include broccoli, Brussels sprouts, cabbage, Chinese cabbage, cauliflower, collard, kale, kohlrabi and radish. Flowering stock *(Matthiola* spp.) and many native cruciferous plants and weeds are also susceptible.

Symptoms The pathogen can infect plants at any growth stage. The first symptom on cabbage is yellowish-green foliage. Sometimes the yellowing is uniform, but usually it is more intense on one side of the leaf and plant *(8.11a).* The leaf curls or the plant twists when only one side is affected. When the whole plant is affected, the lower leaves first become yellow. This symptom gradually progresses up the plant, which becomes stunted. Affected leaves may drop prematurely. The vascular tissue is yellow at first, then turns brown, dies and becomes brittle. These symptoms may not appear on susceptible plants grown in cool soil in early spring until the soil warms up about the time of crop maturity.

Symptoms of yellows resemble those of black rot and under some conditions may be mistaken for that disease. Both diseases cause curling or dropping of the leaves, drying of the leaf edges, discoloration in the vascular system *(8.11b),* and finally death of the plant. In black rot, the yellowing of the leaf toward the margin is often in a V-shaped pattern. For yellows, vascular discoloration tends to be yellowish-brown rather than black. Plating of yellows-infected vascular tissue on acid- or antibiotic-amended potato-dextrose agar at 25°C will yield the pathogen within a few days. The black rot bacterium will not grow on the amended agar.

Causal agent *Fusarium oxysporum* f. sp. *conglutinans* is related to, but distinct from, other forms of *F. oxysporum* that cause wilts of solanaceous and legume crops. Four physiologic races of *F. oxysporum* f. sp. *conglutinans* exist. Race 1 is most pathogenic to cabbage, Brussels sprouts, kale, cauliflower and radish, but less so to flowering stock, collard and kohlrabi. Race 2 is most pathogenic to radish, but slightly so or nonpathogenic to Brussels sprouts, kale, flowering stock, cabbage, cauliflower, collard and kohlrabi. Race 3 and race 4 are most pathogenic to flowering stock. Race 4 differs from race 3 in that it causes slight to no wilt in the cultivars Apricot, Double Giant Imperial Rose, Double Early Giant Imperial Golden Rose, Double Giant Imperial Shasta, Standard Gold and Tenweeks White flowering stock.

On potato-dextrose agar at pH 6.5 to 7, *F. oxysporum* f. sp. *conglutinans* colonies may be pale white to cream-colored and the mycelium is floccose throughout. Hyaline microconidia and macroconidia are both borne on phialides. Microconidia are generally abundant, variably cylindrical, oval or straight to curved, one-celled or septate, and measure 2.5 to 4 by 6 to 15 µm. Macroconidia, although sparse in some strains, are generally multicellular, sickle shaped, up to 5.5 µm in width, and 33 µm in length. Hyaline, smooth to rough-walled chlamydospores are generally abundant and may be terminal, intercalary, isolated or in chains.

Disease cycle The pathogen usually infects through seedling rootlets and through roots wounded during transplanting. The fungus moves directly to the water-conducting xylem in the stem and leaves. Part or all of the plant may die. The pathogen then produces conidia and chlamydospores, both inside and outside the affected tissue. The browning of vessels and yellowing of the leaves in advance of the fungus is caused by a toxin, which is produced in the xylem and moves upward in the sap stream.

Yellows is favored by warm weather. It develops to its maximum at 27 to 29°C soil temperatures. It is inhibited above 32°C and does not develop well at soil temperatures below 16°C. Soil moisture and pH have little or no influence on disease development.

The pathogen can survive in soil for a number of years in the absence of a host. Once the pathogen has become established, it can spread rapidly to other areas by wind-borne soil, surface drainage water and soil adhering to farm implements. Long-distance spread is by infested seed or diseased seedlings.

Management

Cultural practices — Extreme care must be taken to avoid using infested seedlings or seed. Once the disease has appeared in an area, the best control strategy is to use yellows-resistant cultivars.

Resistant cultivars — Two types of resistance (A and B) exist in cabbage. Type A cultivars are uniformly resistant at all field soil temperatures; Type B cultivars have some degree of resistance below 21°C. Market Prize, Market Topper and King Cole have Type A resistance. Some cultivars with Type B resistance include Red Hollander, Wisconsin All Seasons and Wisconsin Hollander.

Commercial radish cultivars resistant to yellows are Red King and Fancy Red. Most cultivars of broccoli, Brussels sprouts and cauliflower are resistant to yellows, except in hot weather.

Growers should consult local extension crop specialists regarding cultivars best suited to a particular area.

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(Original by A.A. Reyes)

▶ 8.12 Powdery mildew *Fig. 8.12*

Erysiphe polygoni DC. (syn. *Erysiphe cruciferarum* Opiz:Junell)

Powdery mildew is a minor disease of cabbage, cauliflower and the other cruciferous vegetables, except for rutabaga cultivated in southern Ontario in the region bordering Lake Huron, where incidence and severity may be high. *Erysiphe polygoni* has a wide host range.

Symptoms Signs of the disease consist of white, powdery or mealy, superficial, patchy mycelial growth on the upper leaf-surface *(8.12).* The patches grow together as the disease develops, until the entire surface is covered by the fungus. It often spreads to the undersurface of the leaves in the late stages of disease development. Affected leaves successively change color to light green, yellow and tan. The leaves die and abscise in very severe cases, resulting in plant stunting and reduced yield, depending on the stage of growth when infection occurs.

Causal agent The nomenclature of the organism has varied. Previously, the collective name *Erysiphe polygoni* was used for the pathogen of rutabaga, turnip and many other economically important hosts. Recently, some authors have restricted the name to the pathogen affecting the Polygonaceae and used the name *E. cruciferarum* to refer to the pathogen that attacks members of the Cruciferae and Papaveraceae. *Erysiphe polygoni* is an obligate parasite with a wide host range. It forms mycelium that produces numerous haustoria, is white and superficial on the upper leaf- surface, and has a powdery appearance due to the production of numerous, one-celled conidia that are hyaline, narrowly ellipsoid to cylindrical, and measure 24 to 51 by 10 to 17.5 µm. The teleomorph state consists of a dark, completely closed cleistothecium that contains 4 to 10 asci. The cleistothecium also has 10 to 30 characteristically simple, indefinite, mycelioid appendages with lengths one-half to three times the diameter of the cleistothecium.

Disease cycle The fungus occurs in several physiologic races on many plant species. Spores are the chief means of dissemination, being carried long distances by wind to other fields. Survival occurs chiefly in the teleomorph state (cleistothecium), which forms in late summer on the upper leaf-surface of infected plants that remain alive over winter. Limited survival of mycelium in tissue of overwintered plants is possible.

Disease development is favored by low relative humidity, water stress within the host, and the availability of a film of moisture on the leaf surface in which spores can germinate.

Management

Cultural practices — Useful control measures in rutabaga crops include crop rotation, eradication of cruciferous weeds and the destruction of volunteer rutabaga and other crucifers.

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(Original by R.F. Cerkauskas)

▶ 8.13 Rhizoctonia diseases *Figs. 8.9, 8.13a-e*

Damping-off
Wirestem
Root rot (crater rot)
Bottom rot
Head rot
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Diseases caused by *Rhizoctonia solani* occur wherever cruciferous crops are grown. Depending on the time of infection, this fungus can cause different diseases, such as damping-off, wirestem, bottom rot, head rot and root rot. The pathogen exists in numerous strains that are host specific and exhibit a high degree of variability in virulence toward crops.

Symptoms

Damping-off — Pre-emergence damping-off occurs when seeds decay and fail to germinate or they germinate but the young plants fail to emerge. Loss is often attributed to poor seed. Postemergence damping-off occurs when the stem of the seedling is attacked. Infection usually occurs when seedlings are 2 to 5 cm tall. A water-soaked area completely encircles the tender stem near the surface of the soil. Stem tissues collapse, causing the seedling to wilt and eventually topple over *(8.9).* Damping-off usually develops in foci or along rows within the seedbed or seed flat. Invaded seed serves as a food base, enabling the pathogen to reach adjacent seeds or seedlings. Species of *Pythium* also may be associated with damping-off.

Wirestem — Although seedlings of many non-crucifer- ous crops become increasingly resistant to *Rhizoctonia solani* as they mature, crucifers are frequently attacked even after plants are 10 to 15 cm tall. Wirestem may result from an extension of damping-off, but new infections may occur. The stem darkens above and below the soil line and the outer cortex decays and is sloughed off in sharply defined areas encircling the stem *(8.13a,b).* The stem is wiry and slender at the point of the lesion, hence the name wirestem. Because the stiffened stele of the stem provides support, the plant remains erect but may eventually die when transplanted to the field. If the plant survives, it remains unhealthy, stunted and invariably yields poorly.

Root rot (crater rot) — Turnip, radish, rutabaga and horseradish may be attacked in the field or in storage. The pathogen enters through roots, leaf scars or mechanical injuries. Lesions are usually dark brown, slightly sunken and spongy *(8.13c).* Infected horseradish tissue is light yellow to grayish tan and dry. Infected tissues separate easily from the advancing edge of decay. Secondary soft-rotting bacteria may follow the fungal infection *(8.13d).*

Bottom rot — Cabbage plants can be attacked midway through the season when outer leaves of heads touch moist, infested soil. Brown to black, sunken, elliptical, sharply defined lesions initially appear on the undersides and basal parts of leaves. The lesions may become papery during dry weather. Dark-colored, web-like mycelium may grow on the lesions. Eventually, the lower leaves wilt, turn shades of yellow through black, dry up and may drop off. Plants may recover and produce heads or bottom rot may progress into head rot.

Head rot — During damp weather, bottom rot may evolve into head rot on developing cabbage heads *(8.13e).* The fungus attacks the bases of the wrapper leaves, causing them to drop off, exposing the stem. As the hyphae spread up the stem, the bases of the outer head leaves are attacked and their margins on top of the head turn yellow and dry up. A dark-colored, web-like mycelium may be observed between leaves. The disease may spread over the entire surface of the head and several leaf layers deep. The head remains upright and dark and becomes studded with small brown sclerotia. The decay is initially firm but soft-rotting bacteria may invade, turning affected tissues soft and odorous. The sexual state of the fungus may be observed as a gray or chalk-colored membranous growth on the underside of lower leaves or on the surface of the soil, growing from points of attachment to the stems at the soil line. In storage, lesions established in the field may expand as a firm dark decay (see Lettuce, pseudomonas diseases, 11.3).

The damping-off, wirestem, bottom rot and head rot symptoms are diagnostic of the diseases caused by *R. solani.* It is generally possible to differentiate between postemergence damping-off caused by *R. solani* from that caused by *Pythium* spp. *Rhizoctonia solani* produces stem decay near the soil line and may later advance downward into the roots. *Pythium* spp. generally infect root tips and root hairs and advance upward through the plant to the soil surface. Soil particles usually cling to the coarse mycelium of *R. solani* but not to the fine mycelium of *Pythium* spp. when seedlings are pulled from the soil. The coarse, brown, septate mycelium of *R. solani* may be differentiated microscopically from the fine, hyaline, aseptate mycelium of *Pythium* spp.

In the wirestem phase, mycelium is usually not visible. In the bottom and head rot phases, *R. solani* can be differentiated from *Sclerotinia sclerotiorum.* The decay produced by *R. solani* is initially firm, and small, brown-colored sclerotia may be visible over the decayed surface. This is in contrast to the slimy decay and relatively large black sclerotia produced by *S. sclerotiorum.* On root crops, wefts of cream- to brown-colored mycelium and the typical brown- colored sclerotia distinguish *R. solani* from other root rots.

Causal agent (see Bean, rhizoctonia root rot, 15B.7) Strains of *R. solani* that attack crucifers do not normally infect potato and *vice versa.* Identification of strains is possible by determining pathogenicity on specific hosts and by observing anastomosis between standard tester strains and the strain in question. Strains that attack radish and other crucifers typically belong to anastomosis groups AG-4 and AG-2, respectively.

Disease cycle (see Bean, rhizoctonia root rot, 15B.7; and Lettuce, bottom rot, 11.6) The temperature range for growth of the cabbage strain is 9 to 31°C, with an optimum for infection and disease development of 25 to 27°C.

Management

Cultural practices — To prevent damping-off and wirestem in seedbeds, only sterilized soil or soil that has not previously supported crucifers should be used. Seeds should be treated with hot water and a suitable fungicide. Plant density should permit adequate light penetration and air circulation. Growers should avoid overwatering and reduce or eliminate watering on cloudy

days. Water should be applied in the morning so that plants can dry off early in the day. Factors such as deep planting, reduced vigor of seed and excessively cold, hot, moist or saline soils may increase seed decay and pre-emergence damping-off. Deficiencies of calcium, potassium and nitrogen, or excessive nitrogen, may promote disease.

Wirestem on cabbage seedlings may develop into bottom and head rot, so affected seedlings should not be transplanted to the field. A rotation of at least three years with non-cruciferous crops should be practiced. When cultivating, soil should not be mounded or hilled onto lower leaves of plants.

Root crops such as turnip, rutabaga and horseradish with only slight infections may safely be stored at low temperature.

Resistant cultivars — Breeding of cruciferous cultivars resistant to *Rhizoctonia solani* has not been studied extensively because the organism was thought to have a wide host range and differences among isolates were not obvious. With the recognition of host specific strains, the potential for breeding for resistance has improved.

Chemical control — Benches, flats and tools should be sterilized using a suitable disinfestant. Soil may be drenched with fungicides after seeding.

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(Original by P.D. Hildebrand)

▶ 8.14 Sclerotinia rot (cottony soft rot) *Fig. 8.14*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia sclerotiorum is capable of attacking an exceptionally wide range of vegetable crops (see Lettuce, white mold, 11.9). Among crucifers, cabbage and Brussels sprouts are the most commonly affected. *Sclerotinia minor* Jagger also infects cruciferous crops, but it has not been observed on crucifers in Canada.

Symptoms The first symptoms on cabbage are water- soaked areas on stems and lower leaves, especially those in contact with the soil, and also on upper surfaces of the head. As the lesions expand, the leaves wilt and the fungus may spread to the rest of the plant. Affected tissues turn soft and watery and become covered with white cottony fungal mycelium in which numerous, irregularly shaped sclerotia are embedded *(8.14).* The sclerotia are initially white but later turn black. The fungus may spread rapidly from infected to healthy heads within bins during transit or in storage if low temperatures are not maintained.

Stored cabbage can also be attacked by *Botrytis cinerea* (see Lettuce, gray mold, 1 1.10). Gray mold lesions are water-soaked, gray-green and often covered with masses of powdery gray spores, which readily distinguish this disease from sclerotinia rot. Gray mold usually attacks cabbage leaves late in the storage period.

Sclerotinia rot is primarily a storage disease of rutabaga and turnip. After initial infection, a slightly pinkish color may be present on the margin of a lesion, while the inner portion of the lesion is pale brown and water-soaked. The typical white, cottony mycelium and sclerotia appear later on infected tissues.

Causal agent (see Carrot, sclerotinia rot, 6.15)

D**isease cycle** (see Carrot, sclerotinia rot) The pathogen may slowly continue to colonize tissues of field-grown cabbage, rutabaga and turnip placed in cold storage, resulting in pockets of decayed plant material within the bins. The fungus may also survive on infected residue adhering to wooden storage bins, which may act as a source of inoculum.

Management

Cultural practices — Once *Sclerotinia sclerotiorum* has become established in a field, it is difficult to destroy all sclerotia. Soil tillage usually brings enough sclerotia to the soil surface to initiate disease if environmental conditions are favorable. Rotation with non-susceptible crops reduces the number of viable sclerotia. A three- year period with crops such as corn, cereals or grasses is recommended. Susceptible crops should be planted on well-drained soils. Many weed species are susceptible to *S. sclerotiorum,* so fields should be kept weed-free. Pathogens that cause necrotic lesions should be controlled and wounding during harvest should be avoided. Harvested produce should be placed into clean bins for storage. Removal of soil from rutabaga and turnip roots by washing will reduce disease development in storage. Proper temperature and ventilation should be maintained during storage.

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(Original by P.D. Hildebrand)

▶ 8.15 White rust *Fig. 8.15*

Albugo candida (Pers.) Kuntze (syn. *Albugo cruciferarum* (DC.) S.F. Gray)

White rust is of minor importance to radish root crops, but losses to radish seed crops may be severe because of distortion and destruction of flower parts. *Albugo candida* occurs on many cruciferous plants, but yield and quality losses usually are minor, except for turnip rape (*Brassica campestris* L.), in which damage may be significant. The pathogen exhibits a high degree of host specificity, so infection of radish from overwintering inoculum on cruciferous weeds or crops, except canola, is unlikely.

Symptoms Infection may be local or systemic. Local infections, consisting of white, shiny, raised pustules or sori *(8.15)*, develop on upper and lower leaf-surfaces and on stems. The pustules arise from masses of sporangia, which form under the leaf epidermis. When the pustules rupture, the powdery, dry sporangia become wind-borne. Systemic infection of young stems and flowering parts causes enlarged and abnormal development of sepals, petals, pistils and anthers and prevents normal radish seed development. The infected flowers, flower stalks and seed pods become enlarged and develop into distorted, staghead galls, which contain many oospores. The stagheads initially are green, but become brown and brittle at maturity. The fungus often coinfects the same tissues, especially the distorted flower parts, with the downy mildew fungus *Peronospora parasitica.*

Causal agent *Albugo candida* is an obligate parasite that forms mycelium in host tissue that is nonseptate and intercellular with knob-like haus- toria. Club-shaped sporangiophores are produced from the mycelium in a layer beneath the host epidermis. Sporangia are produced in chains from the sporangiophores, with the oldest at the tip of the chain, and become readily detached at maturity. The sporangia are hyaline, nearly spherical, and 14 to 16 by 16 to 20 µm. Germination is primarily by production of zoospores that contain a disk-like vacuole on one side. Oogonia and antheridia are formed from mycelium in the intercellular spaces of the host tissue, especially in systemic infections. The oospores that form have a warty wall, which is a useful trait for distinguishing species of *Albugo*. The oospores are chocolate-colored, 40 to 55 µm in diameter and usually are found in the stems and seed.

Disease cycle The fungus overwinters as thick-walled oospores inside the staghead galls or as mycelium in living tissues. The stagheads may break off the plant and fall to the ground, eventually releasing the oospores. The oospores may germinate and infect the cotyledons and leaves of young plants in the spring. Pustules that develop on leaf surfaces contain many sporangia that are released and dispersed chiefly by wind but also by rain and insects, to neighboring plants. Several generations of sporangia are produced on plants during a growing season. Seed-borne oospores are another important source of inoculum. Under dry conditions in a laboratory, oospores have germinated after 17 years of storage; however, their longevity in natural soils has not been determined.

Disease development is favored by moist conditions and temperatures between 10 and 25°C. Chilling of the sporangia is necessary to initiate zoospore production. Germination occurs over a range of 1 to 20°C and is optimal between 10 and 14°C. Temperatures above 25°C reduce the rate and amount of release of zoospores. The motile zoospores swim about for a short time, then produce germ tubes that invade the host through stomata. Moisture on the host surface is essential for germination and infection.

Management

Cultural practices — The incorporation of infested crop residues into the soil helps to reduce levels of pathogen inoculum. Radish crops should be planted some distance from where they were grown previously. Volunteer rapeseed and wild mustard plants are a source of inoculum and should be eradicated early in the growing season. During the development of the flowering crop, furrow irrigation should be practiced instead of overhead irrigation, because it is less likely to spread the pathogen.

Resistant cultivars — The radish cultivars Chinese Rose Winter, Round Black Spanish, and Burpee White exhibit some resistance to white rust.

Chemical control — Seed treatment with broad-spectrum fungicides is effective in minimizing spread through contaminated seed.

Selected references
Mukerji, K.G. 1975. *Albugo candida.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 460. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.
Petrie, G.A. 1986. *Albugo candida* on *Raphanus sativus* in Saskatchewan. *Can. Plant Dis. Surv.* 66:43-47.
Pound, G.S., and P.H. Williams. 1963. Biological races of *Albugo candida. Phytopathology* 53:1146-1149.
Williams, P.H., and G.S. Pound. 1963. Nature and inheritance of resistance to *Albugo candida* in radish. *Phytopathology* 53:1150-1154.
(Original by R.F. Cerkauskas)

VIRAL DISEASES

▶ 8.16 Turnip mosaic *Figs. 8.16a-c*

Turnip mosaic virus

Turnip mosaic is a widespread, destructive disease of rutabaga in south-central Ontario, where most of the rutabaga production in North America occurs. The host range of turnip mosaic virus is wide and potential sources of inoculum include many cruciferous vegetables and weeds. Losses can be devastating during the growing season in Oriental crucifers, horseradish, lettuce, spinach and mustard crops. Infected rutabaga roots are susceptible to breakdown in storage.

Symptoms The first indication of turnip mosaic infection in rutabaga is premature yellowing of the basal leaves, affecting groups of plants within the field. In severe infections, the entire crop may exhibit yellowing *{8.16a).* The younger leaves become distorted and may assume a wrinkled or blistered appearance with severe light and dark green mosaic-mottling *(8.16b).* Vein banding and veinal flecking may develop on the apical leaves. Pale chlorotic and necrotic lesions are often present on the basal leaves. Older leaves senesce quickly. As new leaves are produced, older ones drop off when mature, resulting in a "goose-necked" appearance. When plants are infected early in their development, roots are severely stunted *(8.16c)* and loss of leaves makes mechanical harvesting difficult. In southern Ontario, disease symptoms are generally evident three weeks after the plants become infected, but symptoms vary depending on the cultivar, the stage of plant development when infection occurs, and the environmental conditions after infection.

Recognition of turnip mosaic in the field is based on foliar distortion and mottling, particularly on younger leaves. Leaf samples should be sent to a diagnostic clinic for positive identification.

Causal agent Turnip mosaic virus is a flexuous filament, approximately 720 nm in length, containing a single linear molecule of RNA. It is sap transmissible to dicotyledonous plants in many families, and it is transmitted by many aphid species in a non-persistent manner.

Turnip mosaic virus occurs worldwide and is frequently reported in the temperate zone of North America. In Ontario, four strains of the virus have been identified, the most common one being identical to a crucifer- infecting strain in New York State. The remaining strains of turnip mosaic virus have narrow host ranges and are limited to a few *Brassica* crops and cruciferous weeds. Only one of the strains is important on rutabaga.

Disease cycle Turnip mosaic virus is not seed-borne. Until recently, the major source of virus has been limited to infected rutabagas, either volunteers or those dumped from storage during early spring. Other reservoir plants are limited to a few weeds in the mustard family.

Natural spread of turnip mosaic virus in the field is only by aphids (see aphids, 8.39). In general, early plantings of rutabaga that become infected provide a source of inoculum for outlying rutabaga fields. Aphid transmission from rutabaga to newly planted winter rapeseed occurs in the fall, allowing for overwintering of the virus.

In 1985, an epidemic of turnip mosaic virus in southern Ontario caused more than 30% loss in the rutabaga crop. The outbreak coincided with an increase in winter rapeseed production near rutabaga fields, which began in the early 1980s. The effect of the virus on rapeseed yield is minimal but losses to rutabaga have increased each year. Infection of rutabaga fields several kilometres distant from the nearest source of inoculum is caused by movement of virulifer- ous aphids from winter rapeseed to rutabaga in early July. This disease has been particularly serious on the major rutabaga cultivar Laurentian.

Management Control of turnip mosaic virus depends on timely control of the aphid vectors (see aphids, 8.39), because symptoms may not appear until long after the aphids have disappeared.

Cultural practices — Volunteer rutabaga plants should be disked in the fall and left on the surface to freeze. In the spring, volunteer plants should be eliminated and culls from storage should be worked into the soil to enhance decomposition. Eradication of mustard and volunteer rape- seed in or around rutabaga plantings helps to reduce infection. If possible, rutabaga should be grown in isolation from other cruciferous crops, in particular winter and spring rapeseed and spring canola. Early season planting allows rutabaga roots to size up before the introduction of virus by aphids, the benefit being that late-infected crops produce a marketable root with better storage success. In southern Ontario, rutabagas should be sown preferably before mid-June. Rutabagas with a high level of virus infection should be marketed early to avoid storage losses.

Resistant cultivars — Sources of resistant germplasm are being developed.

Selected references

Shattuck, V.I., and L.W. Stobbs. 1987. Evaluation of rutabaga cultivars for turnip mosaic virus resistance and the inheritance of resistance. *HortScience* 22:935-937.

Stobbs, L.W., and V.I. Shattuck. 1989. Turnip mosaic virus strains in southern Ontario, Canada. *Plant Dis.* 73:208-212.

Tomlinson, J.A. 1970. Turnip mosaic virus. CM1/AAB Descriptions of Plant Viruses, No. 8. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 4 pp.

(Original by L.W. Stobbs)

NON-INFECTIOUS DISEASES

▶ 8.17 Black speck of cauliflower *Fig. 8.17*

Black speck is a minor physiological disorder of cauliflower, occurring mainly on North American Snowball cultivars and rarely on cultivars originating in Europe, Japan or Australia. The cause of black speck is unknown, but it may be a nutrient deficiency. Symptoms appear as black necrotic specks only on branches or flower stalks in the interior of the curd. Several layers of cells collapse and become discolored, resulting in slightly sunken black lesions *(8.17).* The lesions may range from 0.5 to 4.0 mm in diameter. Specific control measures are not available. Black speck of cabbage is a storage disorder.

Selected references

Loughton, A., and J.W. Riekels. 1988. Black speck in cauliflower. *Can. J. Plant Sei.* 68:291-294.

(Original by P.D. Hildebrand)

▶ 8.18 Brown bead *Fig. 8.18*

Brown bead is a physiological disorder of broccoli that occurs sporadically and is usually associated with rapid growth during periods of high temperature following periods of abundant rainfall. The disorder may cause significant losses. Symptoms appear only on the broccoli heads as they approach maturity. Floral buds turn tan or brown *(8.18)* and become easily detached. Adequate supplies of well-rotted organic matter, particularly on light soils, help to prevent surges in growth by maintaining a constant supply of water and nutrients. Some cultivars appear to be less susceptible than others.

Selected references

Flint, M.L., ed. 1985. *Integrated Pest Management for Cole Crops and Lettuce.* Univ. Calif., Statewide Integrated Pest Management Project, Div. Agric. Nat. Res., Oakland. 112 pp.

(Original by P.D. Hildebrand)

▶ 8.19 Growth cracks *Fig. 8.19*

Growth cracking is a physiological disorder that affects the developing hypocotyls and roots of rutabaga and turnip and heads of cabbage. Marketable yields are usually affected to some degree in most crops.

Symptoms Cracks originate in the neck region and may extend down the side of the root *(8.19).* The exposed tissue may be colonized by soft-rotting bacteria, especially *Envinia* spp., which transform the entire root into a jelly- like mass. The interior of the root may be completely decayed, leaving a shell of outer tissues. Initially there is no odor but the tissue becomes putrid following bacterial decay. Growth cracking also can occur in cabbage heads.

Causal agent Growth cracks appear during periods of rapid growth, especially when heavy rainfall or irrigation follows a dry spell.

Management

Cultural practices — Factors affecting rapid growth must be controlled. Uneven spacing, which allows rapid growth of some roots within the row, can be avoided by precision seeding. Excessive fertilization, especially with nitrogen, and manuring should be avoided. Adequate levels of well-rotted organic matter, particularly on light soils, help to prevent surges in growth by maintaining a constant supply of water and nutrients. Decay caused by secondary organisms is also aggravated by warm, humid weather and dense plant canopies. Maintaining soil moisture at uniform levels reduces the incidence of growth cracking. On a small scale, cabbage plants can be given a half twist to break off some of the roots. This will limit water uptake and slow down the growth until the heads can be harvested. Affected roots and heads should not be stored because they are susceptible to bacterial decay and may act as a source of decay within storage bins.

(Original by R.W. Delbridge)

▶ 8.20 Hollow stem *Fig. 8.20*

Hollow stem is a common physiological disorder of broccoli and occasionally cauliflower.

Symptoms Symptoms occur internally in stems and are not usually visible externally. Small elliptical cracks develop in the stem. As the plant approaches maturity, the cracks may enlarge and grow together, causing the stem to become hollow *(8.20).* In severe cases, the hollow area may extend into the floret region of the head. Tissues within the hollow areas are usually not discolored, but tissue discoloration and breakdown may develop soon after harvest.

Causal agent Although the symptoms are similar to those of boron deficiency in cauliflower, the role of boron in hollow stem is not clear. A balance between boron and nitrogen levels in tissues appears to be important, but this relationship has not been clearly defined. Calcium may also have a role in this disorder. Factors that favor rapid plant growth after initiation of the head tend to promote hollow stem.

Management

Cultural practices — Methods that maintain an even rate of growth should be followed. Growers should avoid excessive nitrogen fertilization, especially after head initiation. A uniform, close plant spacing will help to maintain an even growth rate and greatly reduces the incidence of hollow stem. However, at too high a plant density, head size can become too small. Harvested broccoli should be cooled immediately to inhibit bacterial activity in the stem cavities.

Resistant cultivars — Variable susceptibility to this disorder exists among cultivars, but no cultivar is totally resistant. Many of the recently developed hybrid cultivars of cauliflower are very susceptible to hollow stem.

Selected references

Scaife, A., and D.C.E. Wurr. 1990. Effects of nitrogen and irrigation on hollow stem of cauliflower *(Brassica oleracea* var. *botrytis). J. Horde. Sci.* 65:25-29.

Shattuck, V.I., and B.J. Shelp. 1987. Effect of boron nutrition on hollow stem in broccoli (*Brassica oleracea* var. *Italica). Can. J. Plant Sci.* 67:1221-1225.

Shattuck, V.I., B.J. Shelp, A. Loughton and R. Baker. 1986. Environmental stability of yield and hollow stem in broccoli (*Brassica oleracea* var. *Italica). Can. J. Plant Sci.* 66:683-688.

Tremblay, N. 1989. Effect of nitrogen sources and rates on yield and hollow stem development in broccoli. *Can. J. Plant Sci.* 69:1049-1053.

Vigier, B., and J.A. Cutcliffe. 1984. Effect of boron and nitrogen on the incidence of hollow stem in broccoli. *Acta Horde.* 157:303-308.

(Original by P.D. Hildebrand)

▶ 8.21 Intumescence (edema, enation, neoplasm, thrips pustule) *Figs. 8.21 a-c*

Intumescence is a physiological disorder that can affect the leaves of most crucifers, but it is of most concern when it affects the leaves of cabbage heads.

Symptoms Intumescence is characterized by small, wartlike protuberances that develop on either side of the first 3 to 10 outer leaves of the cabbage head *(8.21a).* The protuberances develop in varying densities and may grow together to form irregularly-shaped, elevated areas. The epidermis may split as the inner cells of the leaf enlarge, subdivide and push outwards *(8.21b).* The exposed cells are initially white and give the protuberances a crystalline appearance. Later, these tissues turn brown and become corky. The margins of severely affected leaves may also dry to a thin, papery texture during storage. Losses due to trimming of affected leaves may be significant.

Causal agent The disorder follows days when the soil is warm and wet and the night air cool and saturated with water. Under these conditions, water uptake by the roots occurs more rapidly than loss by transpiration. This stimulates cell enlargement and division in the hypodermis, resulting in sufficient pressure to rupture the epidermis. The disorder may also be aggravated by injury of the epidermis due to drifting sand, thrips feeding *(8.21c),* herbicide residues and air pollution.

Management

Cultural practices — Growers should avoid excessive irrigation during periods when day-to-night temperatures vary greatly. Using windbreaks to reduce soil erosion and controlling thrips may help prevent the problem.

Selected references

Sherf, A.F., and A.A. MacNab. 1986. *Vegetable Diseases and Their Control.* 2nd ed. J. Wiley & Sons, New York. 728 pp.

(Original by L.S. Bérard)

▶ 8.22 Tipburn, internal browning *Figs. 8.22a,b*

Tipburn and internal browning are physiological disorders of similar origin. Chinese cabbage is particularly prone to this disorder, which also affects Brussels sprouts, cabbage and cauliflower.

Symptoms The inner leaves of heads of cabbage and Brussels sprouts are affected, but there are no external symptoms. Margins of inner leaves turn brown, beginning at the hydathodes, and later desiccate to become papery at the margin or over large portions of the leaf *(8.22a,b).* The affected tissue may turn dark brown to black, occasionally being invaded by secondary bacteria that cause a watery soft rot. When tipburn occurs in stored cabbage, symptoms may not be noticed until heads are spot checked before marketing. Brussels sprouts are usually sampled before processing. If the incidence of internal browning is high, the shipment may be rejected. In cauliflower, internal leaves turn brown and fold over the developing curds. When secondary microorganisms attack these leaves, they become mushy, smear over the curd and render it unmarketable.

Causal agent Tipburn and internal browning are caused by inadequate transport of calcium to rapidly growing tissues. Low levels of calcium at the leaf margin result in tissue collapse. Excess nitrogen results in large outer leaves that accumulate calcium at the expense of young expanding leaves within heads.

Environmental conditions that favor rapid plant growth also favor tipburn. Abundant soil moisture promotes rapid growth, while excess moisture reduces soil oxygen levels, which in turn reduces calcium uptake and movement. A dry spell after a period

of abundant moisture may aggravate the disorder. Cruciferous crops grown on sandy soil are usually more prone to tipburn compared to plants grown on heavier-textured soils.

Management

Cultural practices — Factors that promote rapid plant growth should be avoided. Maintenance of optimum fertility is important. Maintaining a phosphorus to potassium ratio of 1:1 should help to minimize the incidence of tipburn. Irrigation may be necessary to maintain optimum levels of soil moisture. Addition of high levels of calcium to the soil and foliar applications do not seem to alleviate the problem. Close plant spacing and prompt harvesting of crops when mature are beneficial practices.

Resistant cultivars — Cultivars that grow less vigorously are less prone to this disorder. Resistant cultivars of Brussels sprouts and cabbage are available.

Selected references

Maynard, D.N., and A.V. Barker. 1972. Internal browning of Brussels sprouts: a calcium deficiency disorder. *J. Am. Soc. Hortic. Sei.* 97:789-792.

Maynard, D.N., B. Gersten and H.F. Vernell. 1965. The distribution of calcium as related to internal tipburn, variety, and calcium nutrition in cabbage. *Proc. Am. Soc. Hortic. Sei.* 86:392-396.

Palzkill, D.A., T.W. Tibbitts and P.H. Williams. 1976. Enhancement of calcium transport to inner leaves of cabbage for prevention of tipburn. *J. Am. Soc. Hortic. Sei.* 101:645-648.

Rosen, J. 1990. Leaf tipburn in cauliflower as affected by cultivar, calcium sprays, and nitrogen nutrition. *HortScience* 25:660-663.

(Original by P.D. Hildebrand and L.S. Bérard)

NUTRITIONAL DISORDERS

Despite optimum fertilization with nitrogen, phosphorus and potassium, crucifers sometimes show other nutrient deficiency symptoms. Nutrients may become unavailable in soils that are alkaline or excessively acidic. Soil in which crucifers are grown should be maintained at pH 5.8 to 6.5 for optimum growth.

▶ 8.23 Boron deficiency (brown heart, mottled heart, raan, water core) *Figs. 8.23a-d*

Crucifers, especially cauliflower, rutabaga and turnip, are sensitive to boron deficiency. Plants require boron for translocation of carbohydrates and regulation of plant growth hormones. Boron deficiency results in poorly developed cell walls that collapse.

Symptoms The first visible symptom in cauliflower appears on the head as a firm, tan-colored or water-soaked spot *(8.23a).* Water-soaked areas of internal stem tissues may also occur. The discoloration usually darkens and may spread over the entire head, but the curd remains firm

(8.23b). External stem tissues near the base of the midrib of petioles closest to the head may crack, become corky and turn brown. Cavities that eventually form in stem tissues also turn brown, and the tips of the youngest leaves become light brown. The curd acquires a bitter taste.

In rutabaga and turnip, boron deficiency occurs in the edible root and first appears as areas of brown discoloration that are scattered, grouped or arranged in a concentric pattern *(8.23c).* Discoloration is usually more pronounced in the central area of the root. Symptoms are usually restricted to the lower two-thirds of the root, but in severe cases they can extend from the bottom to the crown, where cavities may form *(8.23d).* Affected tissues become fibrous or punky and may develop a bitter flavor. They also may be invaded by secondary soft-rotting organisms. Roots that are mildly affected usually lack external symptoms. In severe cases, the roots are reduced in size and external root tissues may become rough, corky or leathery. Leaf margins of severely affected plants are typically chlorotic, and a purplish tinge may develop on the underside of the leaves.

Causal agent Boron deficiency occurs most commonly on soils that are coarse or sandy and subject to excessive leaching, resulting in soluble boron levels of less than 0.5 ppm, or on soils with a pH greater than 7. Boron also becomes less available during long periods of drought.

Management

Cultural practices — Irrigation may help to prevent boron deficiency by maintaining a uniform soil moisture. On soils deficient in boron, a boronated fertilizer is required. Only foliar sprays of boron should be used on high pH soils. Application of boron after the appearance of deficiency symptoms is usually too late to correct the problem.

Resistant cultivars — Variable susceptibility to this disorder is expressed in some cultivars. No cultivar is totally resistant.

Selected references

Cutcliffe, J.A., and U.C. Gupta. 1987. Effects of foliar sprays of boron applied at different stages of growth on incidence of brown-heart in rutabagas. *Can. J. Soil Sci.* 67:705-708.

Gupta, U.C. 1979. Boron nutrition of crops. *Adv. Agron.* 31:273-303.

Shattuck, V.I., and B.J. Shelp. 1985. Brown heart in rutabaga. Ontario Ministry Agric. Food. *Factsheet.* 2 pp.

(Original by P.D. Hildebrand)

▶ 8.24 Magnesium deficiency *Fig. 8.24*

Broccoli, cabbage, cauliflower and kale are the cruciferous crops that are most sensitive to magnesium deficiency. Brussels sprouts and turnip are much less affected.

Symptoms Symptoms of magnesium deficiency first appear on the older leaves as blotches of interveinal chlorosis *(8.24).* The chlorosis intensifies and may be accompanied by interveinal purple blotches, especially near the leaf margins. As the plant matures, these symptoms progress up the plant. Orange and red tints also may occur, especially on the leaf undersides. Under severe magnesium deficiency, only the youngest leaves remain green.

Causal agent Magnesium is required for the production of chlorophyll in plants. Deficiencies commonly occur on sandy, acidic soils.

Management

Cultural practices — Dolomitic limestone, which consists of calcium and magnesium carbonate, can be applied to correct this problem. This form of limestone simultaneously raises the soil pH. Magnesium may also be applied with fertilizer, or plants may be sprayed with epsom salts (magnesium sulfate).

Selected references

Scaife, A., and M. Turner. 1983. *Diagnosis of Mineral Disorders in Plants.* Vol. 2. *Vegetables.* H.M. Stationery Office, London. 95 pp.

(Original by P.D. Hildebrand)

▶ 8.25 Molybdenum deficiency (whiptail) *Figs. 8.25a,b*

Among cruciferous crops, cauliflower is the most sensitive to molybdenum deficiency, but symptoms may also appear on broccoli, Brussels sprouts and cabbage.

Symptoms In the seedbed, molybdenum deficiency symptoms appear on leaves as small, dark flecks that may have yellow halos. On young plants in the field, molybdenum deficiency symptoms are most common under cool, dry conditions and may appear as areas of interveinal chlorosis, which later become puckered. The chlorotic areas may also develop a purple discoloration. Similar symptoms occur on older plants, particularly along leaf margins, which become thick and brittle *(8.25b).* The chlorotic areas become necrotic with purple borders, and margins of leaves turn upward, resulting in a cup-shaped leaf. Severe molybdenum deficiency prevents development of the leaf blade, leaving only the midrib with fringes of tissue on either side, resulting in a "whiptail" symptom *(8.25a).* This symptom may be confused with feeding damage caused by certain insect larvae.

Causal agent Molybdenum is necessary for chloroplast maintenance and nitrate reduction in plants. Molybdenum availability is greatly reduced under acidic conditions, for example, a pH less than 6.5.

Management

Cultural practices — Soils should be maintained at or slightly above pH 6.5. Molybdenum in the form of sodium molybdonate may be applied as a seed treatment, as a foliar spray to transplants before field setting, in the transplant water, as a foliar application in the field, or mixed with fertilizer applied to the soil. Foliar sprays can dramatically correct a deficiency and cause immediate recovery and regrowth. However, if the "whiptail" symptom is present, the problem may not be correctable during the current growing season.

Resistant cultivars — Sensitivity to molybdenum deficiency is variable among cultivars.

Selected references

Hewitt, E.J., and S.C. Argawala. 1951. Production of "whiptail" in cauliflower grown in sand culture. *Nature (Lond.).* 167:733.

(Original by P.D. Hildebrand)

▶ 8.26 Sulfur deficiency *Fig. 8.26*

Most cruciferous crops are sensitive to sulfur deficiency.

Symptoms Early symptoms of sulfur deficiency appear as diffuse blotches of interveinal chlorosis on the youngest leaves *(8.26).* The leaves may also become reflexed, in contrast to the cupping symptom observed in molybdenum deficiency. As the leaves mature, the chlorotic areas may dry to a tan-colored, paper-thin texture.

Causal agent Sulfur is required for protein synthesis and stabilization of chlorophyll in plants. Sulfur deficiency usually occurs in plants grown on soils low in organic matter, particularly in areas far from industrial sulfur dioxide pollution.

Management

Cultural practices — Low levels of sulfur in the soil may be corrected by applying sulfur-containing fertilizers or gypsum (calcium sulfate).

Selected references
Scaife, A., and M. Turner. 1983. *Diagnosis of Mineral Disorders in Plants.* Vol. 2. *Vegetables.* H.M. Stationery Office, London. 95 pp.
(Original by P.D. Hildebrand)

STORAGE DISORDERS

Physiological disorders of stored cabbage are not easily diagnosed because the causes are not always clear. General senescence is one such disorder. Others may appear early in the storage period or while cabbage is still in the field, and their development may be altered by frost injury. Several disorders characterized by spotting of varying size and intensity may be ascribed incorrectly to plant pathogens.

▶ 8.27 Black midrib *Fig. 8.27*

Black midrib can be found on cabbage at harvest, but usually it develops soon after the heads are placed into storage.

Symptoms Black midrib initially appears as spots with ill-defined borders in the parenchyma tissue at the base of the midrib on the convex surface of the outer head-leaves. A continuous dark discoloration may subsequently extend more than 10 cm up the midrib *(8.27).* When the disorder is severe, the epidermis and underlying parenchyma collapse, creating a large, sunken lesion on the midrib. Black midrib occasionally affects only the middle leaves of the head. Symptoms do not extend past the abscission line between the leaf and the main stem.

Causal agent The exact cause of black midrib is unknown. The variable susceptibilty of cultivars suggests a genetic basis for this disorder, and the sporadic occurrence, from year to year, also suggests involvement of environmental factors.

Black midrib has been associated with phenol deposition on cell walls and a potassium content of less than 1 % of the dry matter in the midrib tissue at harvest. Excessive nitrogen fertilization promotes disease development. Exposure to frost in the field may reduce the severity of symptoms. Controlled atmosphere storage may promote symptoms on the middle leaves.

Management

Cultural practices — Growers should maintain proper nutrient levels in the soil, harvest cabbage after a few light frosts, avoid controlled atmosphere storage if susceptible cultivars are grown, trim the affected outer head-leaves, and spot check for symptoms on middle leaves of heads.

Resistant cultivars — Bartolo, Hidena, Decema Extra, Houston Evergreen, Polinius and Slawdena are tolerant to black midrib.

Selected references
Bérard, L.S., B. Vigier and M.A. Dubuc-Lebreux. 1986. Effects of cultivar and controlled atmosphere storage on the incidence of black midrib and necrotic spot in winter cabbage. *Phytoprotection* 67:63-73.
(Original by L.S. Bérard)

▶ 8.28 Black speck of cabbage (pepper spot, spotted necrosis) *Figs. 8.28a,b*

Black speck is a storage disorder of cabbage that occasionally causes significant losses. It is distinct from peppery leaf spot, a bacterial disease that can affect several cruciferous crops, and black speck of cauliflower, which is a physiological disorder.

Symptoms Two types of black speck are recognized on cabbage. Type I affects the green, outer leaves at harvest or in early storage *(8.28a).* Type II, also known as senescent black speck, affects the pale yellow inner leaves after cabbage has aged in storage *(8.28b).* Black speck is characterized by the collapse and darkening of guard and adjacent epidermal cells of the stomata, causing scattered, black, sharply sunken, pin-point spots less than 1 mm in diameter on both sides of the cabbage head-leaves.

Causal agent The cause of black speck is unknown. Low storage temperatures may promote the symptoms. Black speck has also been associated with high soil salinity promoted by irrigation during periods of high evapo-transpiration. An imbalance of minerals in tissues has been implicated, especially if the cultivar tends to accumulate excess copper, zinc or nickel. The necrosis of cells may also be related to burning by salts from evaporated guttation droplets. High rates of fertilizer and low soil pH may aggravate this problem.

Management

Cultural practices — Proper levels of nutrients and a soil pH of 6.0 to 6.8 should be maintained. Foliar applications of potassium chloride may help reduce this disorder. Cabbage intended for long-term storage should not be film wrapped or coated with a biopolymer. Susceptible cultivars should be stored at 3 to 4°C instead of 0 to 1°C. Controlled atmosphere storage reduces black speck.

Resistant cultivars — Cultivars resistant to black speck are available.

Selected references

Cox, E.F. 1977. Pepper spot in white cabbage - a literature review. *ADAS Q. Rev.* 25:81-86.

(Original by L.S. Bérard)

▶ 8.29 Gray speck *Fig. 8.29*

Gray speck may be present on cabbage at harvest, but the symptoms develop mainly during early storage. This disorder may be easily confused with black speck.

Symptoms The grayish discoloration of epidermal cells near the base and on the convex surface of outer head- leaves is typical of this disorder. The gray discoloration is due to the thickness of the waxy bloom. The cells actually become brown from phenol deposition on the cell wall. Lesions may be 1 to 3 mm in diameter and scattered, or they may grow together to form irregular patches of variable size in the interveinal areas but mostly along the main and larger lateral veins *(8.29).* Lesions may expand to include stomatal cells, but they do not originate with stomata. The lesions are not sunken as in black speck.

Causal agent Heavy soils, soils with a low pH, and high nitrate levels all have been implicated in this disorder. Low levels of manganese and high levels of zinc are associated with affected tissues of susceptible cultivars.

Management

Cultural practices — Growers should avoid excessive levels of soil nitrate during growth and harvest cabbage after a few fall frosts have occurred. Maintaining the soil pH around 6.5, providing adequate levels of soil organic matter, and following a crop rotation program helps to produce heads with good keeping quality. Controlled atmosphere storage reduces gray speck on many cabbage cultivars, but not with the cv. April Green.

Resistant cultivars — The cultivars Polinius, Houston Evergreen, Hidena, Slawdena, and Green Winter have good storage potential.

Selected references

Bérard, L.S., B. Vigier, R. Crête and M. Chiang. 1985. Cultivar susceptibility and storage control of grey speck disease and vein streaking, two disorders of winter cabbage. *Can. J. Plant Pathol.* 7:67-73.

(Original by L.S. Bérard)

▶ 8.30 Necrotic spot *Fig. 8.30*

Necrotic spot is a minor storage disorder of cabbage.

Symptoms Two types of necrotic spot are recognized. Type I appears as uniformly spaced, dark lesions, 1 to 5 mm in diameter, on the leaves or midribs *(8.30).* The lesions are ill-defined initially, but become sharp and sunken as the epidermis and parenchyma cells collapse. Type II appears as spots or cavities of similar size in the pith of the main stem of the head. The lesions of necrotic spot are larger than those of black speck, but smaller than those of black spot and are not confined to the top leaves as in black spot.

Causal agent The cause of necrotic spot is unknown. The disorder is variable among cultivars and seasons.

Management

Cultural practices — Controlled atmosphere storage tends to promote necrotic spot on the cultivars Superslaw, Danish Ballhead, Quick-Green Storage and Hitoma.

Resistant cultivars — Resistant cultivars are available.

Selected references

Bérard, L.S., B. Vigier and M.A. Dubuc-Lebreux. 1986. Effects of cultivar and controlled atmosphere storage on the incidence of black speck and necrotic spot in winter cabbage. *Phytoprotection* 67:63-73.

(Original by L.S. Bérard)

▶ 8.31 Vein streaking *Fig. 8.31*

Vein streaking is a storage disorder of cabbage that develops early in the storage period. Symptoms vary with the season. This disorder causes only minor losses, but trace levels of the disorder can be found in most years.

Symptoms Vein streaking is characterized by superficial brown or black markings on the epidermis of the midrib at the base of the concave surface of the outer leaves *(8.31),* with occasional extension on lateral veins. At present, vein streaking is considered distinct from gray speck because of the differential response among cultivars, storage treatments, and the distinct sites at which symptoms occur.

Causal agent The exact cause of vein streaking is not known. The brown discoloration of cells is due to the deposition of phenols on epidermal cell walls at sites where the wax bloom is thin. High levels of nitrate increase symptom severity.

Management

Cultural practices — Nitrate fertilizer should not be applied in excess. Controlled atmosphere storage usually reduces vein streaking in most cultivars, but not every year.

Resistant cultivars — No cultivar is fully resistant. The least susceptible cultivars do not keep well in storage.

Selected references (see gray speck, 8.29)
Bérard, L.S., M.A. Dubuc-Lebreux and J. Vieth. 1987. Étude histologique de la bigarrure nervale, de la griselure du limbe et de la médiane noire, trois désordres du chou en entrepôt. *Can. J. Plant Sei.* 67:321-329.

(Original by L.S. Bérard)

▶ 8.32 Frost-induced disorders *Figs. 8.32a-e*

Black blotching
Black spot
Epidermal detachment
Frost blemishing
Redheart

Cabbage heads intended for storage are usually harvested in late fall and may be exposed to frost. Although cabbage tissues freeze at –0.8°C, they can tolerate a few cycles of freezing and thawing if not subjected to mechanical shock. However, cooling and thawing rates and severity of freezing temperatures may affect tissue structures and metabolism resulting in disorders that may appear in the field or later in storage.

Black blotching

This is a superficial leaf disorder that develops during storage, possibly due to early fall frosts. Individual spots develop and may grow together to form blotchy areas. The spots are circular, 1 to 3 mm in diameter, with pin-point dark centers and a grayish or brown halo delimited by a dark line *(8.32a).* Spots are usually found on the convex surface of the leaf or on the midrib near or below the equator of the head.

Black spot

This disorder appears as large, black, interveinal necrotic areas, 1 to 5 cm in diameter, located on the top leaf of the cabbage head after several months of storage *(8.32b).*

Epidermal detachment

This disorder may result from repetitive superfreezing and thawing of the exposed outer- head leaves. The epidermis turns white and becomes detached from the parenchyma, creating a blistering effect *(8.32c).* This symptom usually appears on the epidermis of veins on the concave surface of the outermost head leaf. With severe frosts, epidermal detachment may occur on the epidermis of veins on the convex surface, in interveinal areas of both surfaces, and on leaves deeper in the head. Symptoms highly conspicuous in the field may disappear during storage as leaves begin to wilt.

Frost blemishing

This disorder is characterized by large, white, circular to triangular areas (*8.32d)* on the exposed top-head leaves and occurs after severe frosts in the field.

Redheart

This disorder is caused by freezing for more than 24 hours in the field or in storage. The damage is often irreversible. After thawing, leaf tissues several layers deep appear glassy. Outer tissues often retain their healthy appearance. Loose heads may become watery and collapse. A fetid odor may be detected in storage rooms. The internal tissues of affected heads become tan or reddish, and later may dry to a papery texture (*8.32e*). A darkened zone usually delimits affected areas from healthy areas. Heads exposed to severe frost lose their dormancy and senesce more quickly. Symptoms similar to redheart may occur in controlled atmosphere storage, if cabbage is exposed to abnormally low levels of oxygen or high levels of carbon dioxide.

Management *Cultural practices* — Cultivars that mature within the growing season should be selected and late planting should be avoided. If possible, the crop should be harvested before periods of severe frost. If the crop has been exposed to frost, the heads should be allowed to thaw completely before being harvested, and mechanical shock should be avoided. Growers should monitor for glassiness of inner head-leaves at harvest. For long-term storage, especially in controlled atmosphere, growers should select lots that have not been exposed to frost. Cabbage that has superficial frost injury and shows symptoms of epidermal detachment at harvest should not be placed into long-term storage. Outer head- leaves affected by black blotching or black spot can be trimmed after storage. Storage rooms should be ventilated and maintained at constant temperature slightly above 0°C (controlled atmosphere storage is 3°C) and high humidity.

Selected references
Isenberg, F.M.R. 1979. Controlled atmosphere storage of vegetables. *Hortic. Rev.* 1:337-395.

(Original by L.S. Bérard)

▶ 8.33 Other storage disorders

Dormancy
Ethylene
Maturity

Dormancy

At harvest, the apical bud within the head is dormant. During the storage period, dormancy is gradually lost and the lateral buds and apical meristem within the head begin to grow. The outer leaves of the head lose their metabolic reserves and begin to wilt, become yellow or brown, and develop a papery texture. Outer leaves may also abscise from the stem. These are symptoms of general senescence and usually occur late in storage. At that time, the head also becomes highly susceptible to rot.

Ethylene

Severe leaf-yellowing and abscission of leaves deep within the head are symptoms of ethylene exposure in early storage. Ethylene concentrations as low as 1 ppm are sufficient to speed natural senescence of stored cabbage heads. Cabbage should not be stored with fruits, such as apples, that produce ethylene. The atmosphere within a storage must be renewed periodically. In controlled atmosphere storage, levels of oxygen and carbon dioxide should be maintained at 3 and 5%, respectively, to inhibit the effects of ethylene.

Maturity

Winter cabbage intended for long periods of storage must be harvested at the proper stage of maturity. Heads that are immature at harvest usually remain green in storage and often become flaccid because they easily lose water. Overmature heads can be recognized because they are white or yellow-green and their outer leaves may be reddish or bleached from frost injury (see frost-induced disorders, 8.32). Transverse cracking of veins, leaf abscission and splitting of heads, either naturally or by mechanical shock, are also signs of overmaturity.

(Original by L.S. Bérard)

NEMATODE PESTS

▶ 8.34 Northern root-knot nematode *Fig. 6.20*

Meloidogyne hapla Chitwood

Symptoms Crucifers usually show less damage than other vegetables and are considered tolerant or resistant. Very small, spherical galls on roots may be difficult to recognize. With heavy infestations, maturity may be delayed and yield reduced. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 8.35 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38; see also Management of nematode pests, 3.12.

▶ 8.36 Stubby-root nematodes

Paratrichodorus allii (Jensen) Siddiqi
Paratrichodorus pachydermus (Seinhorst) Siddiqi
Paratrichodorus spp.
Trichodorus spp.

This group of nematodes is not well established in Canada and has caused only minor damage to a few gardens in southern Alberta.

Symptoms Affected plants become stunted and chlorotic. Roots proliferate abnormally but appear not to grow in length and their extremities may be somewhat swollen. For a complete description, see Potato, 16.39; see also Management of nematode pests, 3.12.

▶ 8.37 Sugarbeet cyst nematode *Figs. 5.14a,b*

Heterodera schachtii Schmidt

This nematode attacks most cruciferous crops, including broccoli, Brussels sprouts, cabbage, cauliflower, kale, kohlrabi, radish, rutabaga, and turnip.

Symptoms Typically damage is most noticeable in patches where nematode densities are high. Infected plants are stunted and outer leaves wilt, yellow prematurely and die. Heart leaves are more numerous than normal but reduced in size. Tap roots are short and stunted, and lateral root development is excessive, giving a whiskered appearance to the tap root. In summer, pin-head sized, white or brown cysts can be seen on washed roots, particularly in the root axils. For a complete description, see Beet, 5.14; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 8.38 Alfalfa looper *Fig. 8.38*

Autographa californica (Speyer)

The alfalfa looper occurs in western Canada, with sporadic outbreaks in southern Alberta and British Columbia, where this species can be more important than the cabbage looper on cruciferous crops.

The larvae chew ragged holes in the leaves of most vegetable crops, sometimes defoliating them.

Identification This moth (family Noctuidae) lays eggs that are yellow. Like the cabbage looper, the larva *(8.38)* has three pairs of narrow, wavy, white lines on the back and a broad, white lateral line. In contrast, the alfalfa looper's lateral line extends almost to the lower margin of the spiracles, the head is brownish green and has a black line through the eyes; there are no legs on abdominal segments three and four in either species (8.38, *8.40c).*

Life history Two or more generations per year are usual in southern British Columbia. Pupae overwinter among crop residue. Adults emerge early in the spring and are active at night. They fly long distances and local populations may be augmented by migrants from the south. Eggs are laid directly on host plants.

Management

Monitoring — Pheromone traps are used to monitor alfalfa looper moths but action thresholds for chemical control have not been established in Canada.

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(Original by H.S. Gerber and J.A. Garland)

▶ 8.39 Aphids *Figs. 8.39a,b; 16.4la,b*

Cabbage aphid *Brevicoryne brassicae* (L.)
Green peach aphid *Myzus persicae* (Sulzer)
Turnip aphid *Lipaphis erysimi* (Kaltenbach)
Turnip root aphid *Pemphigus populitransversus* (Riley)

The green peach aphid (see Potato, 16.41) and the turnip aphid are widespread in Canada. The cabbage aphid, which is usually the most injurious of the crucifer-infesting aphids, is transcontinental in Canada. The turnip root aphid occurs but is not often recorded as a pest in Canada.

Aphids are most abundant on crucifers during dry weather. Their eggs are laid in the fall and overwinter on woody plants. Summer generations feed on cabbage, rutabaga, other crucifers and other vegetable crops. Aphid populations vary greatly on different cruciferous crops.

Damage On crucifers, all above-ground parts of the plants are attacked, including the flower-head. High populations of these aphids cause leaves to wither and plants to be stunted. Aphids may also transmit turnip mosaic virus. On rutabaga crops in southwestern Ontario, the green peach aphid is the most important vector of turnip mosaic virus but many aphid species can transmit this virus.

Identification The way the aphid colony is aggregated on the host plant is useful for field recognition: the green peach aphid is more uniformly distributed, whereas the cabbage aphid usually has a closely clumped distribution *(8.39a,b).*

The wingless form of the cabbage aphid has a dusky, gray-green abdomen with dark bands. It is covered with a mealy, gray-white wax. Its head lacks well-developed antennal tubercles and is nearly flat. Its antennae and other appendages are dark but paler at the base of each segment.

Life history Depending on the time of year and the nutritional quality of host plants, sexual forms of aphids may develop, mate, and lay eggs. Otherwise, only females are present, giving rise to live young without mating (parthenogenesis). Winged forms develop on the overwintering hosts and move to summer hosts in late spring, becoming most abundant in early summer, especially in dry weather. Aphids generally decline in early to mid-September because of the development of winged forms, a general decline in reproductive activity, an increase in the length of time to reach maturity, a decrease in the nutritional quality of the host plant, and an increase in the abundance of biocontrol agents.

Management strategies

Monitoring — No definite thresholds have been developed for crucifer-feeding aphids in Canada but, after head formation, the threshold is near zero on broccoli, cauliflower, cabbage and particularly Brussels sprouts because aphids are a serious contaminant. Prior to the development of the marketable portion of the crop, relatively high populations can be tolerated. However, because infective aphids inject a toxin, even a few aphids per plant may be serious. On rutabaga, low levels of the green peach aphid, as few as 8 to 10 per leaf, can increase partheno- genetically during warm, dry weather and quickly and completely colonize the upper third of the plant.

Biological control — Naturally occurring predators, parasites and pathogens are often effective later in the growing season. Damaging populations of aphids may appear early in the season before biocontrol agents become abundant.

Chemical control — Foliar sprays may be necessary to prevent serious crop loss and rejection of shipment, particularly when chemical control against other cruciferous pests has destroyed predators and parasites while having only a limited effect upon the aphids.

Control of the aphid vectors of turnip mosaic virus on rutabaga is not easy because they have such a diverse range of hosts and chemical insecticides are ineffective for practical control of migrant, winged aphids. Weekly applications of a light oil are highly effective in delaying and reducing aphid-transmitted turnip mosaic virus infection. The oil interferes with the acquisition and transmission of virus by feeding aphids. For proper application, growers should use 1100 L per hectare of a 1 to *2%* solution. High pressure mist nozzles are needed for effective application; drop nozzles improve coverage of leaf undersurfaces. Oil sprays should be applied weekly during periods of aphid activity until the roots have sized, which is generally late in August. To minimize phytotoxicity, oil should not be applied in bright sunlight or in combination with other spray materials, and chemical insecticides should not be applied within 24 hours of an oil treatment.

Chemical insecticides for aphid control are not needed unless early populations of the cabbage aphid and weather conditions warrant treatment. If aphid populations are sufficiently high to cause wilting, leaf-curl or stunting of plant growth, then it is usually too late to avoid damage by spraying.

(Original by D.T. Lowery, D.G.R. McLeod and L.W. Stobbs)

▶ 8.40 Cabbage looper *Figs. 8.40a-f; 3.7x-z*

Trichoplusia ni (Hübner)

The cabbage looper is an important pest of cruciferous crops in Ontario. It may be less important in other, southern areas of eastern and central Canada and British Columbia. It is not a problem on cruciferous crops in Newfoundland. In British Columbia, it is surpassed in importance by the alfalfa looper (see 8.38).

The cabbage looper does not overwinter in large numbers in Canada. Most infestations start from moth invasions from the south in July and August. In most areas, one generation is usual per season but three generations can develop in warmer areas of southwestern Ontario.

The primary cruciferous hosts are broccoli, Brussels sprouts, cabbage and cauliflower. Other hosts are beet, celery, lettuce, parsley, pea, potato, spinach, tomato and garden flowers, such as carnation, nasturtium and mignonette.

Damage The cabbage looper is generally a minor pest in the more northern regions of Canada. In the southern regions, control of the insect is essential for production of marketable cruciferous vegetables during years of heavy moth invasion. One cabbage looper larva can eat 65 cm^2 of leaf tissue during its development; most damage is done by the last two larval instars. Major concerns are damage to the marketable portion of the crop *(8.40a),* particularly the underside of cabbage and cauliflower heads and the flowers of broccoli, and the presence of larvae in the marketed crop.

The impact of the cabbage looper varies with the region, the crop, and the proposed use of the crop. This insect is not known to disseminate plant pathogens but larval damage to plants may allow entry of secondary organisms.

Identification The egg of this moth (family Noctuidae) is round and pearly white *(8.40b).* The larva is light green with three pairs of wavy white lines on the back and a lateral, white or pale yellow line that is only slightly wider than the dorsal and subdorsal lines *(8.40c).* The head is green without lateral marks. The legs on abdominal segments three and four are vestigial

(8.40d). The cabbage looper first-instar larva has a black head and part of the thorax is black. At rest or when disturbed, the larva raises the middle of its body into a loop. This is a characteristic posture. Mature larvae are 35 to 40 mm in length. The pupa, in a loose cocoon, is light green initially and darkens as it matures *(8.40e).* The adult *(8.40f)* is mottled gray-brown with a wingspan of about 38 mm. It has a silver-white mark on each forewing and, when newly emerged, there is a tuft of raised scales that resembles a collar on the thorax.

Life history Eggs are laid singly or in groups of two or three near the edge of leaf undersides. The larvae usually hatch in three to four days. They feed on the leaf undersides on cabbage and cauliflower heads, and in the flowers of broccoli, developing through five instars in two to three weeks. Pupae are encased in a loose cocoon, which usually is attached to the underside of a leaf. The pupal stage lasts about two weeks. Moths are most active late in the evening.

Management

Monitoring — Populations of adults of the cabbage looper may be monitored by pheromone traps to indicate peak flight-periods. The severity of infestations of larvae on crop plants can be estimated when larvae of the imported cabbageworm and the diamondback moth are monitored. Procedures for monitoring the imported cabbageworm may be used for the cabbage looper. However, because cabbage looper larvae are more difficult to kill with chemical insecticides than larvae of the imported cabbageworm, direct observation to estimate numbers of larvae present on crop plants is superior to indirect monitoring methods, such as estimating the proportion of plants infested or the number of fresh feeding-sites.

The economic threshold varies depending on the crop and the use that is to be made of it. For example, on crops destined for processing, there is a near-zero tolerance for larval contamination, which is the major concern. No head damage to early season cabbage grown for fresh market is tolerated. Some damage to late-season cabbage can be tolerated because the head is trimmed more extensively. The tolerance for damage to the head and inner wrapper leaves of cauliflower and broccoli is minimal, as is the tolerance for damage to crops marketed for their foliage, such as kale.

Biological control — Several parasitic wasps and flies attack larvae of the cabbage looper. The eggs and young larvae are preyed upon by various ants, beetles, bugs and spiders, but viruses are the most valuable biocontrol agents. The cabbage looper larvae are susceptible to infection by several viruses, which cause high mortality of field populations, particularly late in the season. A nuclear polyhedrosis virus is the most common. In some locations, as in southern Ontario, nearly all larvae on plants in late August may be infected by this virus *(3.7x).* It is an effective control agent but has not yet been developed as a commercial insecticide.

The bacterial insecticide *Bacillus thuringiensis* Berliner is the preferred treatment for cabbage looper control (*3.7y,z*), particularly near the time of harvest when pesticide residues are a concern. It is not toxic to or infective in mammals and has no impact on non-target organisms.

Chemical control — The first- to third-instar larvae of the cabbage looper may be controlled by chemical insecticides at concentrations similar to those usually recommended for the imported cabbageworm and the diamondback moth. Higher dosages are required to control later-instar larvae. The first application usually is required in late June on early crops, and in mid-July on late-season crops in areas where the cabbage looper is more of a problem.

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(Original by J.G. Stewart and R.P. Jaques)

▶ 8.41 Cabbage maggot *Figs. 8.41 a-g*

Delia radicum (L.)

The cabbage maggot occurs throughout Canada and is an important pest of all cruciferous crops. It has also been reported on beet, celery, and onion but these are probably erroneous identifications. Other hosts include wild mustard and wild radish.

Damage Cabbage maggot larvae generally feed on the roots of host plants. When numerous, they will destroy or severely stunt the development of young plants. Infestations on larger plants can retard growth, reduce yield, and lower quality. During cool, moist weather, survival of eggs and newly emerged larvae is highest, and root damage is usually most severe.

First-generation maggots, which are the progeny of flies from overwintered pupae, usually cause the most severe damage because the early season weather favors egg and larval survival. Field-seeded stem crucifers, and crops transplanted after mid-June usually escape severe damage because they are well established when the summer genera tions of maggots appear, fewer cabbage maggot eggs survive in areas of summer drought, and egg and larval predators are more abundant and active in July and August.

In late summer or during prolonged dry weather, oviposition and larval development may occur in above-ground stems. Emerging larvae tunnel into the stem tissue at the base of the leaves, which necessitates removal of the affected leaves prior to marketing. Chinese cabbage is especially prone to damage from maggots arising from eggs laid at the base of leaves and often suffers the loss of several leaves because of this type of feeding *(8.41b).*

The cabbage maggot is a problem in production of radish, rutabaga and summer turnip throughout the growing season because the maggot attacks the marketable part of the plant. Wounds caused by first-generation larvae result in scar tissue that persists as unsightly rough areas, reducing the market value of the crop. Larvae of subsequent generations produce furrows *(8.41a,c)* on or near the surface of the roots. These furrows do not heal prior to harvest and, if not removed by trimming, can render the crop unmarketable. Also, larvae in the roots at harvest tunnel inward, resulting in serious storage and marketing problems. The cabbage maggot is particularly important on radish because the presence of even a few maggots may render the crop unsaleable.

Production of marketable rutabaga or summer turnip depends upon adequate control of damage by the cabbage maggot. The effect of the maggot on cabbage, cauliflower, broccoli, Brussels sprouts and kale may be less severe, but infestations may reduce the size of the plants and the quality and quantity of the marketable product, or in extreme cases cause the plants to wilt and die.

Identification Cabbage maggot (family Anthomyiidae) eggs are similar in appearance to those of the seedcorn maggot. However, eggs of the cabbage maggot may be seen with the aid of a hand magnifier to have longitudinal striations and a groove that extends along their ventral aspect. In contrast, the egg of the seedcorn maggot has net-like surface sculpture and the ventral groove extends only about a third of the length of the egg. The legless larvae and pupae (puparia) of the two species can be distinguished at the ventral posterior of the body; the cabbage maggot has a pair of median tubercles that are forked at their apex, whereas the tubercles of the seedcorn maggot are not forked.

Life history In Canada, the cabbage maggot overwinters as pupae *(8.41f)* and may complete two or three generations a year, depending on the weather and soil conditions. The onset of fly emergence in the spring and the period over which flies emerge vary with climate. Therefore, the number of generations of cabbage maggot that attack a specific crop depends on the time of planting and the time of harvest of the crop in relation to the climate. Eggs are laid in the soil near cruciferous plants *(8.41d).* The larvae *(8.41e)* feed on fine root hairs of the plant and eventually burrow into the taproot below ground-level.

In southwestern Quebec, flies *(8.41g)* that emerge from overwintered pupae begin to lay eggs in the middle of May, and oviposition continues until the end of June. Maximum oviposition usually occurs in the first week of June. The larvae may complete development in less than three weeks. Adults of this generation appear in early July and usually lay fewer eggs than the parent flies. Larval survival is also lower during the warmer, drier weather of summer. Subsequent summer-generations overlap, resulting in continuous egg laying until the end of October. The life cycle in southern Ontario and southwestern British Columbia is similar, but the larvae of summer generations do little damage in southern Ontario owing to high temperatures and dry soil conditions during the summer months. The maggot can cause damage throughout most of the growing season in cooler areas.

In eastern Ontario, flies from overwintered pupae begin to lay eggs after the accumulation of 200 degree-days above 4.4°C, measured from March 1, which closely approximates full bloom of serviceberry *(Amelanchier* spp.), and peak egg laying corresponds to full bloom of McIntosh and Cortland apples. Adult emergence is later in the Atlantic provinces and in northern regions of Quebec and Ontario, because the climate is cooler. Fly emergence in the spring in the Prairie provinces coincides with the first blossoms of Saskatoon berry, *Amelanchier alnifolia* Nutt., and pin cherry, *Prunus pensylvanica* L. In many areas of Canada, eggs can be found by the last week of May, and peak activity for adults from overwintered pupae occurs between 8 to 20 June and 7 July (corresponding to the six- to nine-leaf stage of rutabaga), and continues until after mid-July. Flies of this generation begin to emerge in late July. These flies, and those from subsequent summer-generations lay eggs from early August until mid- or late September.

Management

Monitoring — Yellow-pan water-traps can be used to capture adults, thereby monitoring seasonal activity, but they are unreliable as quantitative indicators of infestation potential.

Chemical control — Protection of stem crucifers can be obtained by an application of insecticide in the planting water, as a drench after planting, or both. To protect early crops, whether field-seeded or transplanted, treatment is usually required at the time of planting. If field seeding or transplanting is done after mid-June, treatment may not be necessary because the root system is well developed when eggs are being laid, egg desiccation may occur, and destruction of both eggs and larvae by beneficial

organisms is more likely. Successive and later plantings may need to be protected, and an insecticide is nearly always necessary to protect crops of radish, rutabaga and summer turnip.

Radish: An adequate level of protection can be obtained by treating the seed with an insecticide at the time of seeding, or by the application of a granular insecticide in the furrow along the row.

Rutabaga: Crops planted in May are attacked by the first two generations of the maggot. Crops planted in June are subject to attack by the second and third generations. Although plants at the four-leaf stage or earlier may be less attractive than older plants, growers should not delay treatment until damage has occurred. At least two and sometimes three drench treatments of an insecticide may be required, depending on the insecticide and climatic conditions. The first treatment is applied in the furrow below the seed at the time of seeding. One or two drench applications at five- to six-week intervals may be required, one of which should be timed to give protection during the peak of egg laying. Storage crops should receive an additional treatment applied as a soil drench with a large volume of water. The effectiveness of chemical control in preventing cabbage maggot damage to rutabaga crops in Canada is variable, and some registered insecticides at the recommended rates do not prevent damage. To minimize damage, non-storage crops should be seeded early and harvested before mid-August.

Summer turnip: A single application of an insecticide at the time of seeding is necessary; treatment consists of a granular insecticide in the furrow or a spray applied along the row.

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(Original by C. Ritchot, D.C. Read, M.Y. Steiner and D.G. Harcourt)

▶ 8.42 Diamondback moth *Figs. 8.42a-g*

Plutella xylostella (L.)

The diamondback moth occurs but apparently does not overwinter in Canada. Annual infestations arise from adults carried northward by favorable winds from winter breeding sites in the United States. These migrants arrive during early spring, often prior to the planting of cruciferous crops. The first generation in southern Canada develops largely on cruciferous weeds.

The diamondback moth attacks virtually all cultivated cruciferous crops, wild Cruciferae, and some cruciferous ornamentals. In Canada, the most important vegetable hosts are broccoli, Brussels sprouts, cabbage and cauliflower. The diamondback moth is also a serious pest of canola in western Canada.

Damage The first-instar larva *(8.42c)* mines the leaf tissues. Older larvae feed on the lower leaf-surface, chewing irregular patches in the foliage. Only the upper epidermis may remain intact on severely damaged leaves, giving the leaf a silvery appearance *(8.42a).* Older larvae feed on the florets of broccoli and cauliflower and bore into the edible portions of Brussels sprouts and cabbage. On rutabaga, larvae occasionally damage the crowns.

The diamondback moth is not known to disseminate plant pathogens but larval damage to plants may allow entry of secondary organisms.

Identification The diamondback moth (family Plutellidae; Yponomeutidae also is used) gets its name from three silvery white, diamond-shaped marks that are distinguishable when the adult is at rest with its wings folded *(8.42g).* The egg *(8.42b)* is less than 0.5 mm in length, oval, and yellowish to pale green. Larvae *(8.42c,d)* may reach 12 mm in length. They are relatively hairless, green to gray-green, and subcylindrical. They wriggle when disturbed and suspend themselves on silk threads. When mature, they pupate in a loose, open-mesh cocoon *(8.42e).* The pupa is less than 8 mm long. Initially it is pale green but it darkens as it matures. The adult *(8.42f,g)* is gray-brown with a wingspan of about 13 mm.

Life history Eggs are laid singly or in small groups, usually on the smooth, upper leaf-surfaces of the host plant. They hatch in four to six days, depending on ambient temperatures. The larvae feed on the lower leaf-surface and pass through four instars during a 10- to 14-day period. The mature larva spins a cocoon on the host plant, typically on the lower leaves but not infrequently on the wrapper leaves of cabbage or among the florets of broccoli and cauliflower. Pupation occurs within 24 hours and the adults emerge in about one week. Development from egg to adult averages 25 days in July and August in southern Ontario. The adults become active at dusk and mate within 24 hours of emergence. A female lays an average of 160 eggs during

a lifespan of about two weeks; its fecundity is related to the protein content of the plant on which its larva fed. The thermal requirement for a generation is 283 degree-days above 7.3°C. There may be as many as three to six generations per year.

Management This insect is regarded as an occasional pest of cruciferous crops in most areas of Canada but an infestation may build from endemic to epidemic levels from one generation to the next. Producers must constantly be alert for sudden population eruptions. Control usually is obtained by the same treatments as applied for aphids and other butterfly and moth larvae. However, Brussels sprouts are particularly prone to feeding damage and may require special control measures.

Monitoring — Pheromone and sticky traps can be used to monitor moth flights. The procedures for monitoring the larvae are as described for the imported cabbageworm. Monitoring should begin in early summer.

Cultural practices — After harvesting early season cruciferous crops, such as rape greens and early transplanted cabbage, particularly in warm, dry weather, any remaining foliage should be disked into the soil. Sprinkler irrigation helps to discourage development of this pest while enhancing crop growth.

Biological control — The diamondback moth is attacked by several species of parasitic wasps. In southern Ontario, the most important is *Diadegma insulare* (Cress.), which does not overwinter in association with its host and has limited impact during the host's first two generations. However, during the third and subsequent generations, *D. insulare*, together with *Microplitis plutellae* Muesbeck and *Diadromus subtilicornis* (Grav.), gradually overtake the host. The bacterial insecticide *Bacillus thuringiensis* Berliner provides control in Canada, but resistance has evolved elsewhere in field populations of the diamond back moth.

Chemical control — Foliar applications of short-resid- ual-life chemical insecticides should be applied as necessary. Resistance to carbaryl and permethrin was documented in Nova Scotia and Prince Edward Island in 1990.

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(Original by J.G. Stewart and D.G. Harcourt)

▶ 8.43 European earwig *Figs. 8.43a-d*

Forficula auricularia L.

The European earwig occurs throughout Canada; it is abundant in eastern and central Canada and in southern British Columbia but is not common in the Prairie provinces.

Vegetable hosts include cabbage and other crucifers, celery, lettuce, sweet corn, and Swiss chard. The earwig eats plants and insects, switching readily from one to the other, with a preference for lichens and mosses. The presence of earwigs in fresh produce and contamination from their frass may sometimes be a problem. The impact of earwigs on commercial crop production in Canada appears to be negligible.

Damage Young nymphs may feed on seedlings. Older nymphs and adults are more likely to eat holes in leaves and chew into cabbage heads *(8.43a).*

Identification The European earwig is characterized by a pair of unsegmented, forceps-like appendages (cerci) at the anal end of the abdomen. In the female, the cerci curve inward at the tips; in the male, they are more curved and longer than in the female *(8.43b).* The adult, which usually arches the cerci over its back when disturbed, is dark red-brown. Its wings are short. Nymphs are pale brown, and their wings and cerci are much reduced or absent *(8.43d),* depending on their age.

Life history The European earwig overwinters as an adult, usually in pairs in a nest in the soil. Eggs *(8.43c)* are laid in late winter and the male is driven from the nest, leaving the female to brood the eggs. The eggs hatch during May, although the time of hatching varies with the region. A second clutch of eggs hatches about the end of June, and there may be a third clutch of eggs. Immature earwigs *(8.43d)* molt four times. During the first two instars, the young nymphs generally stay with the female, foraging at night and returning to the nest during the day. At this stage, the nymphs are most subject to mortality from excessive moisture and fungal diseases. Later, they forage more widely and shelter on the soil surface, maturing by late August. The European earwig tends to remain very localized. Adults disperse by crawling or by flight, but their chief means of spread is through man-assisted transport in soil, on equipment, or with plant material. Their habit of sheltering in any available hiding place readily enables them to be transported to new areas.

Management The European earwig seldom causes significant damage to commercial vegetable crops, although sporadic damage to individual fields and home gardens in some areas may warrant control.

Cultural practices — A practical trap can be constructed out of boards with grooves cut in them. Two boards are placed together on the ground with the grooves aligned to form a crawl space. For most effectiveness, the traps should be serviced daily and the earwigs killed. Trapping for young nymphs is done before they disperse from the nest. This same method also is effective against adults.

Biological control — The few known parasites and diseases of earwigs include the fly *Triarthria setipennis* (Fallén) (syn. *Bigonicheto spinipennis* (Meigen)), the nematode *Mermis nigrescens* Dujardin, and a poorly known fungal pathogen described in 1889 as *Entomophthora forficulae* Giard. The fly is established in British Columbia and Newfoundland. No biocontrol agents are commercially available.

Chemical control — Recommendations for chemical control of earwigs on vegetable crops have not been developed in Canada. In most cases, the European earwig is probably controlled by chemicals used against other pests. Insecticidal baits are available commercially for use in gardens and around buildings. Baits commonly consist of fish oil in bran combined with an insecticide.

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(Original by L.M. Crozier)

▶ 8.44 Flea beetles *Figs. 8.44a-e; 10.14a,b*

Cabbage flea beetle *Phyllotreta albionica* (LeConte)
Crucifer flea beetle *Phyllotreta cruciferae* (Goeze)
Garden flea beetle *Phyllotreta robusta* LeConte
Hop flea beetle *Psylliodes punctulata* Melsheimer
Horseradish flea beetle *Phyllotreta armoraciae* (Koch)
Striped flea beetle *Phyllotreta striolata* (Fabricius)

These flea beetles are largely specific to crucifers, feeding in Canada on broccoli, Brussels sprouts, cabbage, Chinese cabbage, horseradish, kale, kohlrabi, radish, rutabaga and summer turnip. They also feed on canola, mustard and cruciferous weeds.

The cabbage flea beetle is native to North America. It occurs from British Columbia to Manitoba.

The crucifer flea beetle (*8.44a; 10.14a*) was introduced on the west coast of North America from Europe in the early 1920s. It was abundant in the Prairie provinces by the late 1930s and early 1940s, and it spread eastward to reach Ontario by 1954 and Quebec and New Brunswick soon afterward. It has become the dominant flea beetle in fields of canola, particularly in the most southerly parts of the canola-growing area, and it is the predominant cruciferfeeding flea beetle across much of southern Canada.

The hop flea beetle is native and present in low numbers across most of Canada. It feeds on a wide variety of crucifers and other crops, such as garden beet, rhubarb and hop (see Herbs and Spices, 10.14).

The striped flea beetle *(8.44b)* was introduced to North America, probably before 1800. By the early 1900s, it was prevalent from the Atlantic provinces to British Columbia. It has long been considered the most common and regularly occurring of the vegetable flea beetles. In Saskatchewan, it is only abundant along the northern fringe of agriculture. Adults of this flea beetle have little feeding preference but show a marked oviposition preference for the plant on which they developed.

Other crucifer-feeding flea beetles, such as the garden flea beetle and the horseradish flea beetle, are incidental and sporadic pests in Canada. The horseradish flea beetle feeds chiefly on horseradish (see Herbs and Spices, 10.14).

Damage During outbreaks, peak numbers of 800 to 1200 flea beetles per m^2 are common. Most damage to crucifers occurs when overwintered adults feed on the cotyledons *(8.44a)* and the first true leaves of young plants early in the spring. By chewing at the stem below ground, they may cause severe, post-emergence seedling losses. Direct-seeded crops are especially vulnerable. Feeding results in small, round holes on the cotyledons and small leaves, giving the plant a "shot-hole" appearance *(8.44d; 10.14b).* A heavy infestation may destroy a young crop and necessitate reseeding, especially in hot, dry weather. Damage may be worse on light, sandy soils.

Cruciferous transplants may suffer less than direct-seeded plants but small, tender transplants that are newly set out can be killed by extensive feeding during warm, dry weather. Extensive feeding by the adult beetles *(8.44c)* also reduces yield because of reduced plant vigor and delayed, uneven maturity. When the plants have reached the six- to eight-leaf stage and are taller than 15 cm, only severe defoliation affects head weight and quality *(8.44e).* By then, plants are well established and have a greater ability to compensate for loss in leaf area.

Feeding by flea beetle larvae on the roots of radish and rutabaga may affect the appearance and marketability of those crops, whereas root damage is not a serious problem in cole crops. On radish and rutabaga crops, injury by flea beetle larvae may be masked by the presence of the cabbage maggot.

Warm, dry conditions accelerate flea beetle development and the appearance of the new generation of adults. There is a potential for severe crop damage by the newly emerged adults, and for severe crop losses the following year. A long, cold spring and/or high rainfall in May or June tend to reduce the severity of damage and economic loss.

Crucifer flea beetles have not been considered serious as pests in British Columbia or Atlantic Canada, but they are a major limitation to successful production of cruciferous vegetables in Ontario and the Prairie provinces, where control of the insects is necessary over large areas in most years. In Alberta, flea beetles have been implicated in stand reduction and yield losses in cabbage and, by inference, other cole crops. In Quebec, flea beetles on cruciferous crops are more sporadic and, although occasional damage occurs, the use of foliar sprays there is not routine.

There is no proof of disease transmission in crucifers by flea beetles in Canada, but research in New York State indicates that the crucifer flea beetle has a limited potential as a vector of the bacterium that causes black rot. Species of *Phyllotreta* and *Psylliodes* are known to spread turnip mosaic virus in Europe.

Identification Flea beetle (family Chrysomelidae) adults (*8.44a; 10.14a)* are small, 2 to 3 mm long, with shiny dark forewings (elytra) and enlarged hindleg segments (femora). Some species have yellow stripes on the elytra *(8.44b).* The adults jump when disturbed. The larvae must be reared to the adult stage for identification to species, which may require consultation with a specialist.

Life history The crucifer-feeding flea beetles are well adapted to the climate in Canada. Winter mortality is usually low, but there are times when their numbers may be severely reduced after several successive severe winters. In Canada, all crucifer-feeding flea beetles are considered to have a similar life cycle with one generation per year. The adults overwinter in leaf litter or occasionally in soil, and they may be found along fencerows, windbreaks and headlands around fields or, less frequently, within cultivated fields. Emergence from overwintering sites begins with the first extended period of warm weather in spring, peaking about mid-May. Adults feed on cruciferous weeds and volunteer canola, moving onto cruciferous crops when those emerge. Eggs are laid in the soil near the roots of host plants, or sometimes on the roots, from the end of May until early July.

The larvae feed on the roots of the host plants. The prepupal and pupal stages develop in the soil in an earthen cell, and the adults emerge from late July onward. The beetles feed on whatever crucifers are present and seek hibernation sites in late September and early October. Development from egg to adult may take as little as seven weeks, making a second generation possible in some years.

Weather plays an important role in flea beetle activity and the extent of crop damage. Adults are very active in hot, windy weather in both spring and autumn. Flight occurs above 20°C, and the adults invade cultivated fields. They feed most actively when conditions are sunny, warm and dry. Cool, damp weather reduces adult activity and the intensity of their feeding, and during inclement weather they shelter in cracks and crevices in the soil. They prefer to attack plants and foliage exposed to bright sunlight, such as seedlings, isolated plants, or plants in widely spaced rows. Shade seems to inhibit their activity.

Management

Monitoring — Infestations may be patchy or sporadic, but most fields are threatened annually in areas with a large acreage of cruciferous vegetable crops. An average of 75 flea beetles per plant can result in severe damage to a mature cabbage crop. However, because of the rapid movements of the beetles and their habit of jumping off plants at the least disturbance, it is difficult to make accurate counts. The best method of damage assessment is to look for the shot-hole injury typical of adult feeding on transplants or on cotyledons as seedlings emerge.

Cultural practices — Flea beetle damage is minimized by late or delayed seeding and by the use of high seeding rates for direct-seeded crops. Cruciferous weeds and volunteer crucifers should be controlled before emergence or transplanting of the crop, and sprinkler irrigation can be applied under warm, dry conditions to drown adult flea beetles when they are most active. Live mulches of clover, vegetable polycultures, and companion planting with marigold generally reduce the number of crucifer-feeding flea beetles, but at the expense of increased competition and lower yields. Spun polyester or other plastic materials as covers on cabbage, radish and rutabaga crops reduce flea beetle damage and promote earlier maturity of the crop, but these covers also favor diseases and weeds.

Resistant cultivars — There are cultivar differences in susceptibility in radish. Also, Chinese cabbage with dark- colored leaves has been damaged less by the striped flea beetle than cultivars with light-colored leaves. In general, crucifer-feeding flea beetles are less damaging on vegetable cultivars with a heavy, waxy bloom.

Biological control — Crucifer-feeding flea beetles are subject to low levels of natural control by native predators, parasites and pathogens, but those that do exist are ineffective for economical control. A European wasp, *Townesilitus bicolor* (Wesmael) (syn. *Microctonus bicolor),* was liberated in Manitoba during 1978-83 but has shown no evidence of establishment.

Chemical control — Chemical control usually is implemented at the first sign of shot-hole damage in cotyledons, and broccoli and other cole heads may need to be protected from cosmetic damage if they are grown near fields of canola. Granular insecticides are used to protect direct- seeded crops, and foliar treatments usually are begun when shot-hole damage appears on the leaves. For greater effectiveness and better coverage, high-volume spraying is suggested early or late in the day when evaporation and wind are at a minimum.

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(Original by JJ. Soroka)

▶ 8.45 Imported cabbageworm *Figs. 8.45a-f*

Pieris rapae (L.)
(syn. *Artogeia rapae* (L.))

The imported cabbageworm, also known as the cabbage butterfly or cabbage white, occurs on crucifers wherever they are grown in Canada. It is a serious pest in all provinces except Newfoundland.

Cruciferous vegetable hosts include broccoli, Brussels sprouts, cabbage, cauliflower, radish, rutabaga and summer turnip. Other cruciferous and non-cruciferous hosts exist.

Damage The larvae chew holes in the leaves of the plants *(8.45a).* Once the heads have started to form, feeding by a single larva can render a cabbage or cauliflower head unmarketable. When crops of broccoli, cabbage, and cauliflower become well established, the plants can tolerate extensive larval feeding. Larval frass contaminates the edible leaves and flower-heads.

The adult of the imported cabbageworm does not transmit plant pathogens but damage by the larvae may allow entry of secondary organisms.

Identification The adult is the white butterfly (*8.45f)* (family Pieridae) familiar to gardeners. The egg *(8.45b)* is elliptical, pointed at the distal end, and flat where it touches the leaf. There are 12 lengthwise ridges *(8.45c)* on its surface. When laid, the egg is creamy white; it changes to light yellow as the embryo matures. The larva is a caterpillar 30 mm in length and pale green when fully grown, with five abdominal legs, a yellow-orange stripe along the length of the dorsal midline *(8.45d),* and faint lateral bands at the level of the spiracles. Short, white hairs give it a velvety appearance *(8.45d).* The chrysalis (pupa) is *(8.45e)* about 18 mm in length, and green to brown, depending on the substrate to which it is attached. The wings of the adult are white and reach 50 mm across, females being slightly larger than males. Males have a single black spot in the middle of the forewing. Females have two such spots *(8.45f).* The forewing in both sexes has a dark patch at the apex and black scales along the leading edge. The hindwing has a small black patch at the outer edge.

Life history In Canada, except possibly in the Prairie provinces, the imported cabbageworm overwinters as a pupa. There are three to four generations of this insect per year in southern Canada. Adults first appear in early April in southern British Columbia, in late April or early May in southwestern Ontario, and in mid- to late May in eastern Ontario, Quebec and the Maritime provinces. In southern Ontario, peak oviposition of the first generation occurs in late May to early June and the generation time varies from 24 to 61 days with an average of 31 days in July and August.

Eggs *(8.45b)* are laid singly near the midrib on the lower surface of the leaf. The young larvae *(8.45c)* hatch in four to eight days after oviposition. There are five larval instars. The first three larval instars feed on the undersurface of leaves on the outside of the plant. The larger, later-instar larvae *(8.45d)* tend to move to the center or head of the plant.

Pupae of summer generations are found on the lower leaves of the crop plant *(8.45e)* or in crop residue. Overwintering pupae occur on crop residue and in debris in fencerows or other protected locations. In summer generations, the adult emerges in 8 to 20 days. After emergence, mating and oviposition commence within 24 hours, and adults are active for most of the daylight hours unless the weather is cloudy, cool or windy. Females obtain nectar from wild flowers near cultivated fields and therefore border rows of the crop tend to accumulate more eggs per plant.

Management

Monitoring — Populations of the imported cabbageworm are usually monitored in conjunction with populations of larvae of the cabbage looper and the diamondback moth. Visual estimation of the number of larvae per plant, expressed as larval units to compensate for different feeding capacity of the larvae, is a reliable basis for action thresholds for application of control measures. In excess of 90% of the crop of cabbage or other cruciferous crop may be marketable when pesticides are applied at most effective times in relation to growth of the plant and development of pest populations. The effects of feeding by pests on plant growth and on marketable yield differ with time of attack relative to plant growth and with species of crop plant (broccoli, cabbage, cauliflower).

A proposed method for estimating the number of larval units is to enumerate new feeding-sites, rather than counting the number of holes per plant, the number of larvae of each species, or the number of infested plants. A new feeding-site is a hole that appears wet and has not yet formed a callus.

Biological control — Parasites, predators and pathogens are the principal biotic factors that determine the abundance of the imported cabbageworm in Canada. For example, the wasp *Cotesia glomerata* (L.) (syn. *Apanteles glomeratus)* is an important

parasite, often infesting more than 30% of larvae. *Cotesia rubecula* (Marshall) (syn. *Apanteles rubeculus),* with a biology similar to that of *C. glomerata,* is the primary larval parasite of the imported cabbageworm in British Columbia and has become established in eastern Ontario. *Pteromalus puparum* (Fabricius), another wasp, kills significant proportions of the pupal population, particularly late in the season. Flies (family Tachinidae), the most common of which is *Phryxe vulgaris* (Fallén), are parasites of pupae of this and other butterflies and moths.

A granulosis virus can cause high mortality in populations of imported cabbageworm larvae, especially late in the season. The virus kills the larvae at all stages of development and kills the pupae by infection from the larval stage. The virus, acknowledged as the key factor regulating the imported cabbageworm in some areas, is not available commercially or registered for use in Canada.

A bacterial insecticide, *Bacillus thuringiensis* Berliner, is very effective. It is the preferred treatment at present, particularly near the time of harvest.

Chemical control — Larvae of the imported cabbageworm can be killed by foliar sprays of chemical insecticides, if coverage is adequate. For early crops, treatment should be about two weeks prior to harvest. Treatment of late crops should start around mid-July and be repeated at two-week intervals as needed. Chemicals with short residual life are preferred, especially near harvest. Alternating insecticides will delay but not limit the development of resistance.

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(Original by J.G. Stewart, R.P. Jaques and D.G. Harcourt)

▶ 8.46 Purple-backed cabbageworm *Figs. 8.46a-g*

Evergeslis pallidata (Hufnagel)

The purple-backed cabbageworm is native to Europe. It occurs in the United States and Canada but is not yet recorded in Labrador or the Yukon. As a pest, this species is more important in Atlantic Canada. The species is variable in occurrence. It is spread by transport of infested produce.

The purple-backed cabbageworm attacks all cruciferous vegetables, particularly broccoli, Brussels sprouts, cabbage, cauliflower, kale, kohlrabi, rutabaga and summer turnip. Horseradish also is susceptible. Eggs of this moth may be found on shepherd's-purse, *Capsella bursa-pastoris* (L.) Medic., and sheep sorrel, *Rumex acetosella* L., but the larvae do not feed on those plants.

Damage When larvae are prevalent on rutabaga, they may completely defoliate the crop and eat holes in the roots *(8.46a).* In other cruciferous crops, they eat large holes in the foliage *(8.46b).* Specific impact studies are not available.

Identification The first evidence of infestation by the purple-backed cabbageworm (family Pyralidae) is the presence of masses of eggs on the plant foliage *(8.46c).* The entire mass is brilliant yellow with a waxy coating. Eggs within each mass are oval and flat with a translucent margin, and average 1.1 mm in length and 0.8 mm in width. Just before hatching, eggs turn brown, then black, because of the formation of the larval head, which is visible through the transparent egg shell.

The newly hatched larva is pale, watery green, and 1.5 to 2.0 mm long with many minute, dark brown tubercles on its body, each bearing one to several long setae. The fully grown larva (*8.46d*) is 20 to 22 mm long, robust and covered with setae. It is purple-brown above and ash-gray beneath, with a conspicuous, narrow, yellow lateral band along the entire length of the body and a narrow white band bordering the lower margin of the yellow band.

The cocoon is oval, 12 to 15 mm long and 5 to 7 mm wide, and lined internally with dark gray silk. Its exterior becomes covered with soil particles attached by a viscid substance, making it resemble a lump of soil. The pupa (*8.46e)* inside the cocoon is light to dark brown.

The moth *(8.46g)* has a wingspan of 22 to 28 mm. It is straw-yellow with irregular, dark brown lines. Males and females are similar in size and color.

Life history The purple-backed cabbageworm has one generation per year in Canada. Eggs *(8.46c)* are laid in compact masses on the undersurface of the lower leaves of susceptible plants. Larvae hatch in four to eight days, feed on the leaf undersurfaces for two to three weeks, hide between leaves during the day, then wander off the host plant and spin cocoons just below the soil surface. Winter is passed as a prepupa (larva) in the cocoon *(8.46f);* pupation occurs the following June. The moth escapes through a loosely constructed end on the cocoon. Local moth dispersal is assisted by wind.

Management

Monitoring — The presence of large numbers of egg masses and/or larvae feeding extensively on leaves indicate a need for action, especially on rutabaga.

Cultural practices — Cultivation or plowing in late fall or early spring helps to bury cocoons, potentially destroying the prepupae and preventing the moths from reaching the surface. A trap crop of summer turnip can be used to attract the moth and usually becomes heavily infested when grown alongside cabbage or rutabaga. Periodic spraying of the trap crop kills the larvae and protects the adjoining crop.

Biological control — The purple-backed cabbageworm is unusually free from natural enemies. No larval diseases have been found. *Bracon montrealensis* Morrison and *Meteorus autographae* Muesebeck are parasitic wasps with potential for biocontrol.

Chemical control — The purple-backed cabbageworm is controlled by the same insecticides that are applied against other crucifer-feeding caterpillars. Foliar sprays can be used to control young larvae.

(Original by R.F. Morris)

▶ 8.47 Red turnip beetle *Figs. 8.47a-c*

Entomoscelis americana Brown

The red turnip beetle is native to the western North American interior plains between latitudes 45 and 68°N. In Canada, it is most abundant in the agricultural parkland zone of the Prairie provinces and occurs in the interior of British Columbia south of 55°N.

The red turnip beetle is an occasional pest of cruciferous crops, mostly in home gardens. The beetle feeds as a larva and adult on cruciferous plants, including canola, mustard, vegetable crops and weeds.

Damage Damage, which usually involves large numbers of beetles completely destroying the crop, is greatest in June when newly emerged adults migrate from fields where they previously infested cruciferous crops and weeds. Newly emerged beetles do not fly but may walk several hundred metres in large numbers in search of food. The arrival of hundreds of beetles can quickly devastate home- garden crucifers.

Identification Adults *(8.47a)* of the red turnip beetle (family Chrysomelidae) are large, about 10 mm long and 5 mm wide, with longitudinal, broad, red and black bands on the forewings (elytra). Eggs are brown, and about 1.5 mm in length *(8.47c).* Larvae *(8.47b)* are wrinkled and dull black, 1 to 2 mm long when just hatched, and 10 to 15 mm long when fully grown. Pupae are orange, and 6 to 10 mm in length *(8.47c).*

Life history The red turnip beetle has one generation per year. The eggs *(8.47c)* occur singly or in small clusters and overwinter on or near the soil surface, or beneath soil lumps or crop residue. Larvae hatch soon after the snow melts in spring, from late March to early May, and larval development normally is completed by the end of May. The larvae enter the soil to pupate and pupal development takes about two weeks. Adults emerge during the first three weeks of June. They briefly feed, then hibernate for about one month, reappearing in late July and August. They fly, feed, mate and oviposit until late October or the onset of cold weather, when they die.

Management

Monitoring — Fields of canola stubble or other sources of infestation close to cruciferous vegetable crops or gardens should be monitored in June when the red turnip beetle adults are easily seen.

Cultural practices — Fall or spring cultivation of infested fields will kill red turnip beetle eggs. Weed control in the spring eliminates volunteer canola and other cruciferous hosts.

Biological control — No parasites of the red turnip beetle are known and the incidence of predation and disease is very low.

Chemical control — Feeding by the red turnip beetle on cruciferous weeds is beneficial, so chemical control usually is discouraged. However, when large numbers of adult red turnip beetles are invading a cruciferous crop, insecticidal applications to the infested area are effective.

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(Original by W.J. Turnock)

▶ 8.48 Other insect pests *Figs. 16.49c-e*

Leatherjackets
White grubs

Leatherjackets are the maggot-like larvae of crane flies (family Tipulidae), of which there are many species in Canada. These larvae are fleshy, grayish black, and about 2.5 cm in length at maturity. They overwinter in the soil and feed on the roots of cruciferous seedlings and transplants. Growers can reduce overwintering leatherjacket populations in areas where they are a problem by practicing clean cultivation, which minimizes the potential for the larvae to damage roots in the spring.

(Original by J.A. Garland)

White grubs (*16.49c-e*) can damage cruciferous root crops, such as rutabaga and turnip, by chewing holes in the sides of the roots. Damage usually occurs when the crop is planted on recently broken land or in weedy fields that already are infested. Chemical control is rarely necessary. (For more information, see Potato, 16.49.)

(Original by K.P. Lim and J.C. Guppy)

OTHER PESTS

▶ 8.49 Gray garden slug *Figs. 11.27c; 18.43*

Deroceras reticulatum (Müller)

The gray garden slug (for other species of slugs, see Lettuce, 11.27) occurs in home gardens in urban areas across Canada. It attacks cruciferous and other vegetable crops, and ornamental lilies *(Convallaria* and *Lilium* spp.).

Damage Cruciferous seedlings are subject to serious injury by infestations of this slug near fences or hedgerows where dense plant growth provides shelter. Later in the season, they may damage Brussels sprouts by climbing the stalks and eating holes in the tender young sprouts; on rutabaga and summer turnip, they make holes that can be mistaken for damage by the purple-backed cabbageworm. During wet weather in the fall, this and other slug species may squeeze between the leaves of Brussels sprouts or cabbage, thereby contaminating the marketable parts.

Identification The gray garden slug (family Limacidae) is 35 to 50 mm long at maturity, and gray-white or creamy flesh-colored with irregular gray markings. The breathing pore is at the rear of center on the right side of the mantle, the body tapers abruptly posteriorly, and the slime is clear.

Life history There is one generation per year. Eggs overwinter. The slugs mature during the growing season, sheltering in tall grass or other plant growth. They breed in the fall, then die (see Lettuce, slugs and snails, 11.27).

Management

Monitoring — Slime trails and excreta are persistent and readily seen signs of slugs. Beer is a strong attractant and has been used to monitor slug populations. Monitoring should begin early, during seedling emergence and after transplanting.

Cultural practices — Vegetable crops should not be planted in low, flat, wet or recently plowed land that has been left idle for several years. Effective ways to keep slug populations low, in the short term, are clean cultivation and removal of sheltering sites along hedgerows and fences.

Biological control — The ground beetle *Calosoma frigidum* Kirby occurs across Canada. It, and birds, snakes, frogs and toads may destroy many slugs, but they are seldom present in sufficient numbers to be effective in vegetable crop fields or home gardens.

Chemical control — Several pesticides have given good to excellent control of slugs in experimental trials but have been of limited value in field operations. Compounds containing tin, aluminum, or a carbamate with sulfur are most effective. The timing of treatments is critical. Compounds for the home garden usually contain the active ingredient metaldehyde, which is also an attractant. Chemicals work best when the slugs are most active, between midnight and early morning when conditions are cool and moist, particularly during periods of dry weather.

(Original by D.C. Read)

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9 Cucurbits (cucumber, melon, pumpkin, squash, zucchini)

BACTERIAL DISEASES

▶ 9.1 Angular leaf spot *Figs. 9.1a,b; 22.1*

Pseudomonas syringae pv. *lachrymans* (E.F. Smith & Bryan) Young *et al.*
(syn. *Pseudomonas lachrymans* (E.F. Smith & Bryan) Carsner)

Angular leaf spot has been reported on field-grown cucurbit crops throughout Canada. It is fairly common and moderately severe on the pickling cucumber crops of southern Ontario, where it affects fruit quality.

The pathogen infects mainly cucumber, but all members of the Cucurbitaceae family are susceptible to some degree.

Symptoms The first symptoms are often noticed soon after crop emergence. Small, round or somewhat irregular water- soaked spots appear on the surface of the cotyledons. Under humid conditions, these spots may ooze droplets of liquid containing bacteria, mostly from the leaf underside. On older leaves, the spots spread until they are confined by the veins, giving them a characteristic angular appearance (*9.1a; 22.1).*

After a few days, the spots dry and turn yellow-brown, and their centers may fall out leaving angular shot-holes *(9.1b)* and tattered leaves. Stems and petioles are also affected by water-soaked areas that later dry to form a whitish crust. Similar spots appear on developing fruit. These are minute and water-soaked at first but later dry and crack, revealing a chalky tissue below. The spots remain mostly superficial. Diseased plants grow poorly and yield losses are possible because of the reduced photosynthetic area. Affected fruit is unmarketable.

Causal agent *Pseudomonas syringae* pv. *lachrymans* is readily isolated from the margin of the lesion. It is an aerobic, non-sporing, motile rod with one to five polar flagella. The cells measure 0.8 by 1 to 2 µm. On beef-peptone agar, colonies are slightly raised, smooth, glistening, and transparent to white with the margin entire. On nutrient agar, levan production results in white mucoid colonies. Acid without gas is formed from glucose, fructose, mannose, arabinose, xylose, sucrose and mannitol. Starch is not hydrolysed and cellulose is not attacked.

Disease cycle Infection is usually initiated in the cotyledons from contaminated seed. It occurs through hydathodes, stomata and wounds when plants are wet. The bacteria multiply rapidly in the intercellular spaces. They also have been recovered from floral parts, suggesting flowers as portals for bacterial invasion of seed. The optimum temperature for disease development is 24 to 27°C. The disease spreads rapidly in rainy weather and when overhead irrigation is used. Bacteria ooze from the leaf spots and are dispersed readily by splashing water, machinery and workers moving through wet crops. Bacteria are washed from diseased leaves into soil by rainfall or overhead irrigation or enter soil when plant parts die or fall at the end of the season. They may survive in association with roots of the host plant and provide inoculum for seedling infection the following season. It is likely that insects also spread the bacteria. They may allow entry of secondary soft-rot organisms. As the fruit ripens, the tissue may rot to the seeds, contaminating them with bacteria. If the bacteria gain access to the vascular system, they can spread rapidly through the plant.

Management

Cultural practices — Pathogen-free seed should be used. Seed from infected crops should never be saved. A two-year rotation from cucurbit crops is recommended. Growers should avoid using fields with cold, wet soils and sites that are poorly ventilated. Infested crop residues should be turned under promptly after harvest to aid the decomposition of plant residue. Furrow irrigation should be used rather than overhead sprinklers. Growers should avoid working in cucurbit crops when the foliage is wet.

Resistant cultivars — Differences in susceptibility among cucumber, squash and melon cultivars have been noted. Seneca Trailblazer and Slice Nice cucumber are resistant to angular leaf spot. Seed catalogs should be consulted for current information on resistant cultivars.

Chemical control — Copper-based bactericides are registered for angular leaf spot control in Canada, but they are largely ineffective and may serve only to spread the bacteria. Chemical seed treatments also are not consistently effective.

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(Original by W.R. Jarvis)

▶ 9.2 Bacterial wilt *Figs. 9.2a,b*

Erwinia tracheiphila (E.F. Smith) Bergey *et al.*

Bacterial wilt is quite common in field-grown cucurbit crops and can be locally severe wherever the beetle vector is prevalent. This disease is best known on cucumbers, but it also affects other cucurbits. Squash, watermelon and cantaloupe are generally less susceptible than cucumber.

Symptoms The disease first appears on leaves as dull green patches that rapidly increase in size. The leaf lobe wilts, soon followed by the rest of the leaf, the branch and then the whole plant. Typically, the centers of rows are worse affected. The vascular system becomes blocked by the build-up of wilt bacteria, causing the plants to wilt and die *(9.2a,b).* Leaves along infected runners wilt, one leaf at a time, until the whole runner is affected. Wilted plants may appear to recover at night but they wilt on successive sunny days, eventually turning yellow and dry. Significant losses in yield may result.

From petiole sections in water on a microscope slide, the bacteria can be seen oozing out as a milky exudate. Another diagnostic character is the stringing out of bacterial gum when a petiole is cut cleanly across and the two surfaces are touched together then slowly drawn apart. In cross sections, bacteria can be seen in the xylem.

Causal agent *Erwinia tracheiphila* is a Gram-negative, motile rod measuring 0.5 to 0.7 by 1.2 to 2.5 µm, with four to eight peritrichous flagellae. It grows poorly on nutrient agar but moderately well on glucose-yeast extract-calcium cabonate agar or

glucose-nutrient agar. Colonies on most media are grayish white to cream, circular, smooth and glistening. They are levan-negative and neither domed nor mucoid on nutrient agar. Some strains utilize formate and citrate, but not tartrate, lactate or galacturonate. Acid but no gas is produced from glucose, fructose, galactose, sucrose and a-methyl D-glucoside.

Disease cycle The pathogen is entirely dependent on spotted and striped cucumber beetles for its transmission. Wilt bacteria overwinter in the gut of adult beetles, which can transmit them to the leaves while feeding. Beetle excreta may also contain the pathogen, which can infect plants through feeding punctures and other wounds made by insects, windblown sand or machinery. The bacteria must swim or otherwise be introduced directly into the vascular system of the plant. Because the beetles are less active in wet weather, the disease is spread from plant to plant mostly on dry days. Seed transmission is not known. Temperatures over 30°C retard disease development.

Management Prevention lies in control of cucumber beetles (see cucumber beetles, 9.21). Once the bacteria have invaded the vascular tissues of cucumber plants, control is impossible.

Cultural practices — Wild or volunteer cucurbits should be eliminated from areas adjacent to cucurbit fields.

Resistant cultivars — There are no highly resistant cultivars, but those that flower later tend to be less affected than early flowering types.

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(Original by W.R. Jarvis)

FUNGAL DISEASES

▶ 9.3 Anthracnose *Figs. 9.3a-c*

Colletotrichum orbiculare (Berk. & Mont.) Arx
(syn. *Colletotrichum lagenarium* (Pass.) Ellis & Halst.)(teleomorph *Glomerella lagenaria* F. Stevens)

Anthracnose in the Cucurbitaceae is caused by a seed- and soil-borne fungus that is widespread and can cause severe losses, particularly in wet summers. The pathogen infects cucumber, watermelon, squash, pumpkin and a few other cucurbits. Marrow appears to be immune.

Symptoms All parts of the plant are attacked, although symptoms vary from host to host. On cucumber leaves, dry lesions first appear on a vein. They become somewhat angular and red-brown, sometimes with a translucent yellowish border *(9.3a).* Dead tissue may drop out of the center of the lesion. Young leaves with multiple lesions become crinkled and distorted *(9.3b).* Lesions on stems and petioles are elongate, slightly shrunken, water-soaked and yellowish. Their surfaces become dry and chalky, and the stem may easily break. Cucumber fruits have lesions that are roughly circular, sunken and water-soaked. Tiny, black, saucer-shaped fruiting bodies (acervuli) form along the veins. The acervuli are numerous and easily seen with a hand lens or the naked eye, particularly when the center of old lesions turns white.

Symptoms on melon and other cucurbits generally resemble those on cucumber. Melon is more severely attacked, with deeper, larger, more sunken lesions. A reddish gummy exudate may form on the lesions. Muskmelon may be completely defoliated because of the severity of lesions on the petiole. Fruit lesions are very conspicuous *(9.3c).* On watermelon leaves, the lesions are black rather than red or brown. Fruit may be infected early, becoming severely misshapen with black lesions. Lesions on older fruits are somewhat raised, flat-topped and circular, and are sometimes called nail-head spots. Under humid conditions, pink spore masses form on the black acervuli of the causal fungus.

Causal agent Acervuli of *Colletotrichum orbiculare* occur on a brown to black stroma on the surface of the host. Setae are not constant but, if present, they are two- to three-septate, brown, stiff, 90 to 120 µm, and taper to a point. Conidia are unicellular, hyaline, oblong or obovate-oblong, slightly pointed, somewhat variable in shape, and measure 13 to 19 by 4.6 µm. The conidia are borne singly on conidiophores. They form a slimy pink mass held by the setae until splash-dispersed. Sclerotia apparently form by the further development of stromatic tissue. Conidia germinate to produce a brown, thick-walled, ovoid to spherical appressorium. Infection occurs from the appressorium on which a round germ pore may be seen.

The pathogen is readily cultured on potato-dextrose agar and other routinely used media. Conidia may form all over the colony rather than on acervuli. Although there is considerable variation in cultural characters between isolates, colonies are usually hyaline at first, then become pink to black. Some isolates form black sclerotia in culture. Seven races have been described on the basis of pathogenicity. Race 1 attacks butternut squash moderately and is virulent on cucumber. Race 2 is moderately virulent on butternut squash and highly virulent to watermelon and cucumber. Race 3 causes flecks on Congo, Charleston Gray and Fairfax watermelon and is highly virulent on cucumber. Race 4 is unable to attack both watermelon and cucumber cultivars,

while race 5 is weakly virulent on cucumbers and highly virulent on watermelons. Race 6 is weakly virulent on cantaloupes and highly virulent on watermelon, and race 7 is weakly virulent on Pixie cucumber, which distinguishes it from race 3.

Disease cycle The disease appears in the field rather late in most seasons, at first in isolated and restricted foci. Spread is largely dependent on splashing water. The disease spreads quickly after heavy, windy rainstorms typical of continental late summers. Spread is also rapid after overhead irrigation, and the disease will follow run-off down slopes. The optimum temperature for epidemic development is about 24°C. Generally, five or six days are required for symptom development. Lesions increase in size more rapidly and conidial production is greater on older versus younger leaves. Increases in the wetting period of leaves at night causes increased conidial production. Disease development that occurs later than 40 to 50 days after planting is unlikely to affect yield. Spores can be spread by workers and tools such as hoes. The fungus is seed-borne. It also overwinters in the field in crop residues, which accounts for about 90% of primary infections the following year. Seed becomes infested during extraction from infected fruit.

Management

Cultural practices — Crop rotation is the primary control for anthracnose, as is the use of pathogen- free seed. Infested crop residues should be plowed under promptly after harvest. Growers should avoid overhead irrigation and avoid working cucurbit crops when they are wet.

Resistant cultivars — Since the fungus exists in a number of specialized races, it is essential to know which races are prevalent in a given area before deciding which cultivars to grow. Resistance to race 2 has been incorporated into commercial cucumber cultivars for many years.

Chemical control — Fungicidal sprays are generally not very effective, because they fail to reach the fungus on the underside of leaves and fruit. Seed treatment fungicides may help to lower the risk of infection from contaminated seed.

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(Original by W.R. Jarvis)

▶ 9.4 Choanephora rot

Choanephora cucurbitarum (Berk. & Ravenel) Thaxt.

The fungus is principally a pathogen of squash, but it also attacks plants both within and outside the Cucurbitaceae family, for example chili peppers *(Capsicum* spp.) and *Amaranthus* sp. It is very uncommon on squash and has not been seen at all on other hosts.

Symptoms The fungus covers the flowers and fruits of squash. It first appears the day after the flowers open and by the second day, especially after a rain, it will have developed fully. Senescent flowers become covered by immature, white conidial heads that rapidly turn brown, then purple-black. Abscission of the male flowers occurs before the fungus reaches the pedicel. Female flowers remain attached, allowing the fungus to pass into the young fruit where it produces a soft, wet rot with profuse conidial development on luxuriant conidiophores that have a distinct metallic sheen.

Causal agent *Choanephora cucurbitarum* produces erect, aseptate conidiophores. The tip broadens into a capitate vesicle bearing several ramuli on which the conidia develop. Conidia are oval to elliptical with conspicuous striations, measure 15 to 25 by 7 to 11 µm, and are light brown to red- brown. The base of each conidium bears a hyaline appendage (the broken sterigma) and the vesicle retains the attachment scar. Sporangia also develop, usually at the center of the plate. They are pendant, white, globular swellings at first. The sporangium becomes separated from the sporan- giophore by a globular columella. At maturity, the sporangia are black, 35 to 160 µm, some being markedly smaller. The sporangia contain numerous light to red-brown, ovoid to elongate spores, which measure 18 to 30 by 10 to 15 µm. The spores lack striations but have two or three hyaline terminal appendages, each consisting of 12 to 20 hair-like hyaline processes in tufts, one to one-and-a-half times the length of the spore. Globose to oblong-ellipsoid chlamydospores form in chains in old cultures. Zygospores, the sexual stage, form between the tips of two hyphae. They also store food for survival. Zygospores are dark brown at maturity and measure 50 to 90 µm in diameter.

The fungus is easily cultured on standard media, such as potato-dextrose agar, but is very variable according to the medium. It forms two fruiting structures in culture: sporangia are formed at high temperatures (25 to 31°C), and conidia are formed in low sugar - high thiamine media in continuous low light or after a bright light - darkness regime.

Disease cycle The fungus produces thick-walled zygospores and chlamydospores that enable it to survive from season to season in crop residues. It is transmitted by bees, cucumber beetles, wind, rain and splashing water.

Management

Cultural practices — Crop rotation is the chief means of control. Adequate spacing of plants to allow for good air circulation within the crop canopy also is important. Growers should avoid the use of overhead irrigation where possible. Cucumber beetles should be controlled when present (see cucumber beetles, 9.21).

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(Original by W.R. Jarvis)

▶ 9.5 Fusarium foot rot *Fig. 9.5*

Fusarium acuminatum Ellis & Everh.
Fusarium equiseti (Corda) Sacc.
Fusarium poae (Peck) Wollenweb.
Fusarium redolens Wollenweb. (syn. *Fusarium oxysporum* var. *redolens* (Wollenweb.) W.L. Gordon)
Fusarium solani (Mart.) Sacc.
Fusarium solani f. sp. *cucurbitae* W.C. Snyder & H.N. Hans.

Fusarium solani may also attack greenhouse cucumber (see Greenhouse cucumber, crown and root rot, 22.7). All cucurbit crops are susceptible to *Fusarium solani* in the seedling stage. A new race of *F. solani* was isolated in 1987 in the Netherlands, first from zucchini then from nine cultivars of six species of Cucurbitaceae. Other *Fusarium* species also may attack cucurbits, but less commonly than *F. solani. Fusarium solani* f. sp. *cucurbitae* race 1 attacks the hypocotyl, causing a cortical stem rot; it also attacks mature fruit. Fusarium foot rots are not common.

Symptoms A cortical rot at the base of the stem and the upper part of the root system may occur at any time after seedling emergence, usually in cold wet soils. In severe cases, flats of seedlings may have a damped-off appearance similar to that caused by *Pythium* spp. Young tissue is water-soaked but older tissue displays little soft rot unless secondary organisms become established. The rot is frequently associated with stem splitting, perhaps a result of uneven growth and stem thickening. The affected area is yellow to pale gold-brown and usually girdles the stem and extends for several centimetres up mature stems. Discoloration extends to the center of the stem. The root system may decay to varying degrees (9.5) and adventitious root initiation may be stimulated.

Causal agent *Fusarium solani* f. sp. *cucurbitae* exists in two races: one attacks hypocotyls causing a cortical stem rot; the other attacks only fruit.

Fusarium solani forms a somewhat sparse, floccose, gray-white mycelium in culture, often with a bluish to blue-brown discoloration in the agar. Microconidia on long lateral phialides are abundant, hyaline, cylindrical to wedge-shaped, and measure 9 to 16 by 2 to 4 µm. Macroconidia form on branched conidiophores. They are cylindrical to falcate, slightly wider near the apex, have a conspicuous foot cell, and measure 40 to 100 by 5 to 7.5 µm. Chlamydospores are globose to ovoid, smooth or rough, intercalary or terminal, and measure 10 to 11 by 8 to 9 µm.

Isolation followed by inoculation tests are necessary to confirm the identity of the pathogen, especially where race 1 of *F. solani* f. sp. *cucurbitae* is suspected.

(For descriptions of other *Fusarium* spp., see Selected references.)

Disease cycle *Fusarium* species are soil inhabitants, surviving for five years or more in the absence of the host. *Fusarium solani* was shown to be seed-borne in zucchini in the Netherlands, where it attacked young plants faster than old ones, and wounded plants more severely than unwounded ones. There were differences in susceptibility among cultivars. Infection also occurred directly from residues in the soil. Generally, this fungus is a facultative parasite of wounds and of plants weakened by poor growing conditions, nematodes or other diseases. It is a cortical pathogen, rather than a xylem inhabitant like *Fusarium oxysporum.* It is spread by splashing rain and in irrigation water. Sand-blast pock marks often become infected in the field. This rot is most severe in dry soils at soil temperatures of 26 to 28°C.

Management

Cultural practices — Growers should maintain good even growth in their crops and follow rotations of at least three years, particularly if the specialized race of *Fusarium solani* is present. Adequate soil moisture is beneficial and irrigation, preferably by drip, should be provided in dry summers. Cucurbit crops should not be planted in fields where foot rot has been severe.

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(Original by W.R. Jarvis and R.J. Howard)

▶ 9.6 Fusarium wilt *Figs. 9.6a,b; 22.9a,b*

Fusarium oxysporum f. sp. *cucurbitacearum* Gerlagh & Blok
(syn. *Fusarium oxysporum* f. sp. *cucumerinum* J.H. Owen)
(syn. *Fusarium oxysporum* f. sp. *melonis* W.C. Snyder & H.N. Hans.)

The fungus may be present in symptomless hosts, including weeds, but it is pathogenic only to Cucurbitaceae. Fusarium wilt is not a common disease.

Symptoms The disease is expressed either as a slow wilt, with progressive yellowing (*9.6a; 22.9b)* of the foliage, or occasionally as a sudden wilt without previous yellowing. In either case, wilting is more severe at times of water or temperature stress on the plant *(9.6b).* Sometimes, very young plants may wilt. Typically, the veins of some leaves turn yellow on one side. This discoloration spreads to the lamina, which thickens and becomes brittle. On stems, longitudinal brown streaks appear *(22.9a),* often exuding gum. In the final stages of the disease, the fungus forms pinkish cushions of spores. The vascular tissue of infected stems is orange-red to brown. This discoloration is sometimes on the side of the stem corresponding to wilted leaves. Fruit does not develop properly and remains flaccid. In situations of high inoculum density and optimum soil temperature, pre- and post-emergence damping-off may occur.

In melon, a faint smell of violets on the leaves of affected plants is considered diagnostic.

Causal agent Earlier accounts of fusarium wilt attributed the disease to different pathogenic forms of *Fusarium oxysporum*, depending upon which cucurbit crops were attacked; for example, *F. oxysporum* f. sp. *melonis* on muskmelon and cantaloupe and f. sp. *cucumerinum* on cucumber, each divided into races depending on the groups of cultivars attacked. In 1988, all forms were merged into f. sp. *cucurbitacearum.*

Differentiation on the basis of pathogenicity of different isolates from cucurbit hosts cannot be done confidently. In seedling tests, isolates are not species specific, often attacking several genera. Mature plants are more exclusively attacked by the corresponding form but notable exceptions occur. For example, some isolates from cucumber cause more wilt on melon than on cucumber. Generally, but not invariably, cucumber and marrow are resistant to isolates from melon.

Fusarium oxysporum f. sp. *cucurbitacearum* is morphologically indistinguishable from many other forms of *F. oxysporum.* It has three- to five- septate, fusoid-falcate macroconidia measuring 27 to 60 by 3 to 5 µm with a somewhat hooked apex and a pedicillate base. The microconidia are borne on short, simple or sparsely branched conidiophores, are oval-ellipsoid, cylindrical, straight or slightly curved, and measure 5 to 12 by 2 to 3.5 µm. Chlamydospores, which are about 10 pm in diameter, are abundant in dead tissues and generally solitary, intercalary or terminal. The mycelium is white to peach-colored, sometimes with a purple tinge. It is sparse to abundant, floccose, and felty in older cultures. Optimum growth in culture occurs at 27 to 30°C. The fungus is readily isolated on general media, but selective media are available.

Disease cycle Soils that are regularly associated with plants having severe symptoms are known as conducive, while those that are not, even in the presence of the pathogen, are classed as suppressive. A much greater population of chlamydospores is required to induce disease in suppressive soils than in conducive soils. The potential of a soil to induce wilt can be assessed by infesting it artificially with *Fusarium* inoculum and growing susceptible plants in it. Suppressiveness is generally associated with soils high in montmorillonite clay and with the antagonistic microorganisms supported in these alluvial soils. The resting chlamydospores of the fungus survive for extremely long periods in soil. They can be killed only by heat or chemical fumigation.

In muskmelon, fusarium wilt is more severe in certain soils than in others. Wilt symptoms are most severe between 18 and 22°C and are rare at 30°C, even in infected plants. Poor light and a decreasing day length increase the severity of fusarium wilt. The disease is more severe in dry soils and when the air is relatively dry, with a relative humidity of 50 to 65%. Wilting is less severe at higher humidities. The pathogen is rarely seed-borne.

Management

Cultural practices — Excessive nitrogen from over-manuring favors this disease. Severity can be decreased by adding potassium, to induce a high K:N ratio, or calcium to the soil. In hot summers, solar heating of soil to 40°C for about 100 hours under polyethylene film mulch is effective, but the land will be out of production for at least four to six weeks. Soils intended for muskmelon production can be assayed for disease suppressiveness, as noted above. This is essential because crop rotation is not very effective.

Resistant cultivars — There are resistant cultivars of muskmelon of the type Delicious 51, e.g. Golden Gopher, Iroquois, Fairfax and Harvest Queen. However, at least three pathovars of the melon form of *Fusarium oxysporum* f. sp. *melonis* exist, so resistance depends on a cultivar having one or more resistance genes, of which two, *Fom1* and *Fom2*, are known. Persian Small Type, Chaca No. 1, Doublon and Orlinabel have *Fom1*. Many Asiatic cultivars have *Fom2*. Certain cultivars, such as Kogane, Nashi, Makuwa and Ogon 9, have a more general resistance, dependent on several genes.

Chemical control — Fumigation is very expensive and is feasible only for very early crops.

Selected references

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Gerlagh, M., and W.J. Blok. 1988. *Fusarium oxysporum* f. sp. *cucurbitacearum* n.f. embracing all *formae speciales* of *F. oxysporum* attacking cucurbitaceous crops. *Neth. J. Plant Pathol.* 94:17-31.

Holliday, P. 1970. *Fusarium oxysporum* f. sp. *cucumerinum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 215. Commonw. Mycol. Inst., Kew, Surrey, England. 1 p.

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(Original by W.R. Jarvis)

▶ 9.7 Gray mold *Figs. 9.7; 22.10a-d*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold can cause considerable damage on cucurbit crops in cool, humid conditions. It is a major disease throughout Canada in cool (12 to 20°C), wet summers. Nesting often occurs in packed produce in which the fungus passes quickly from fruit to fruit, making gray mold one of the most prevalent market diseases of cucurbit crops.

Symptoms On cucurbit leaves and stems, discolored pale gray to tan tissue, copious gray-brown sporulation, and sclerotial development are diagnostic. A water-soaked area is the first sign of infection, usually associated with a piece of dead tissue. This tissue dries and turns pale gray to beige-colored. In humid conditions, a gray-brown mass of fungal conidiophores forms (*22.10a*) and dry masses of conidia disperse in a cloud when touched. Black, hard, flat or somewhat rounded, resistant resting sclerotia, 2 to 5 mm in diameter, form in fleshy tissues (*22.10d*). Sporulation is sparse under very humid conditions, but there is copious development of an off-white cottony mycelium that can be confused with white mold (see white mold, 9.14).

On cucurbit fruit, infection almost always occurs at the flower end or from dead infected petals adhering to the fruit surface (9.7). When senescent flowers are severely affected, the fruit may abort. Infected fruit may deteriorate quickly with a wet rot fostered by secondary fungi and bacteria.

Causal agent (see Lettuce, gray mold, 11.10)

Disease cycle (see Lettuce, gray mold) *Botrytis cinerea* usually infects tissues that are damaged by salt, frost, insects, rough handling, poor pruning and misapplication of fertilizer. Infections are very common at the flower ends of fruits or at torn scars on stems made by careless handling.

Gray mold can become a serious post-harvest disease of cucurbit fruit if it escapes detection at picking and the fruit is stored in damp conditions, especially at sites where water condenses on the fruit. Gray mold is more severe in packing sheds and retail outlets where there are sources of ethylene such as ripening tomatoes or apples.

Management

Cultural practices — Cucurbit crops grown in open fields having well-drained soil with adequate calcium and without excessive nitrogen are seldom seriously affected by gray mold. Because *B. cinerea* has hundreds of hosts, trash piles of almost any plant material are potential sources of inoculum. Accordingly, growers should practice good sanitation. In field sites with cold, wet soils screened from winds, rows are best oriented parallel to the prevailing wind with the rows and plants spaced adequately to give as much ventilation as possible.

Resistant cultivars — Intrinsic genetic resistance is elusive but cultivars with a more open habit, leaving flowers exposed to drying conditions and with flowers that fall as soon as pollination has occurred, tend to be less affected.

Chemical control — Fungicides should be used with caution because the fungus quickly develops fungicide-tolerant strains. Benzimidazole and dicarboximide fungicides may only suppress natural competitors and make gray mold more severe.

Selected references

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Ellis, M.B., and J.M. Walker. 1974. *Sclerotinia fuckeliana.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 431. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by W.R. Jarvis)

▶ 9.8 Leaf blights *Figs. 9.8a,b; 22.12a,b*

Alternaria leaf blight

Alternaria alternata (Fr.:Fr.) Keissl.
Alternaria cucumerina (Ellis & Everh.) J.A. Elliott
Alternaria tenuissima (Kunze:Fr.) Wiltshire
Stemphylium botryosum Wall.
(teleomorph *Pleospora herbarum* (Pers.:Fr.) Rabenh.)

Ulocladium leaf spot

Ulocladium atrum G. Preuss
(syn. *Stemphylium atrum* (G. Preuss) Sacc.)
Ulocladium consortiale (Thiim.) E. Simmons
Ulocladium cucurbitae (Letendre & Roum.) E. Simmons
(syn. *Alternaria cucurbitae* Letendre & Roum.)

Leaf blights of cucurbits are superficially similar. The causal agents have almost certainly been confused in the scientific literature. *Alternaria*, *Stemphylium*, and *Ulocladium* all have dark, more or less muriform spores and are variable in culture. *Alternaria alternata* and *A. tenuissima* are weak parasites, common on necrotic tissue in many plants. A specialized race of *A. alternata* f. sp. *cucurbitae* has been described in Europe; its pathogenicity seems limited to cucumber and melon. *Alternaria cucumerina* attacks watermelon, muskmelon, cantaloupe, cucumber and *Cucurbita* spp. and is a serious market pathogen.

Symptoms Small, yellow-brown flecks, often with a light green halo, appear on the upper surface of the leaves. These areas enlarge, grow together and sometimes develop concentric zonation *(9.8a,b; 22.12a,b).* Severely affected leaves die. Zonate lesions several centimetres in diameter may also occur on the fruit, where they are somewhat sunken and covered with a dark, olive-green, felty mass of fungal conidia.

Causal agents *Alternaria cucumerina* is distinctive because its conidia are long-beaked. In general though, fungi with *Alternaria*-like spores are very easily confused. Short-spored species of *Alternaria* are often mistaken for *Stemphylium* and *Ulocladium* species. For example, *Ulocladium cucurbitae* forms *Alternaria*-like conidia on the plant, but *Ulocladium*-like conidia in fresh axenic culture from which diagnosis is best made.

Identification depends on a study of conidium development (for details, see Selected references).

Disease cycle Most diseases caused by *Alternaria* are more prevalent in periods of hot, dry days and dewy nights. The fungus overwinters in infested plant residues and spreads by means of wind-blown spores. Seed may be contaminated during extraction from diseased fruit.

Management

Cultural practices — Pathogen-free seed should be used and also treated with a fungicide as an added precaution. Infected fruit, especially melon and squash, should not be shipped, because it can rot quickly in storage. Crop rotation is the best means of control. Growers should turn under infested crop residues promptly after harvest and avoid using overhead irrigation. Plants stressed by adverse growing conditions are more susceptible to opportunist pathogens of this type.

Resistant cultivars — Because of the multiplicity of fungi capable of causing very similar symptoms, it is impossible to suggest universally resistant cultivars.

Chemical control — Leaf blight diseases are rarely severe enough to warrant the expense of fungicide application.

Selected references

Booth, C., and K.A. Pirozynski. 1967. *Pleospora herbarum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 150. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.
Butler, D., M.J. Griffin and J.T. Fletcher. 1979. Leaf spot on cucumber caused by *Ulocladium atrum. Plant Pathol.* 28:96-97.
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Simmons, E.G. 1982. *Alternaria* themes and variations (11-13). *Mycotaxon* 14:44-57.
Vakalounakis, D.J. 1990. *Alternaria alternata* f. sp. *cucurbitae*, the cause of a new leaf spot disease of melon (*Cucumis melo). Ann. Appl. Biol.* 117:507-513.
Zitter, T.A., and L.W. Hsu. 1990. A leaf spot of cucumber caused by *Ulocladium cucurbitae* in New York. *Plant Dis.* 74:824-827.

(Original by W.R. Jarvis)

▶ 9.9 Leaf rot (pink mold rot) *Fig. 22.13*

Trichothecium roseum (Pers.:Fr.) Link
(teleomorph *Hypomyces trichothecioides* Tubaki)

The fungus is more common in greenhouses (see Greenhouse cucumber, leaf rot, 22.13) than in the field. It affects all cucurbit crops in cool, wet summers but is usually overlooked as a pathogen because it is a common saprophyte on most decaying vegetation.

Symptoms On young leaves, the lesions remain small, first appearing water-soaked with a broad yellowish margin. The center dries, turns tan-brown and may fall out. On older leaves, the lesions appear large and irregular, and they coalesce *(22.13).* In severe cases, fruit is also attacked.

Large, tan-colored irregular spots with a pink tint are diagnostic as spores develop under continuing humid conditions. Frequently, the fungus is accompanied by *Alternaria* and lesions may be variously pink with spores of *Trichothecium* or black with spores of *Alternaria.* Initiation of the lesion from colonize substrates, such as insect excreta or fallen petals, is sometimes evident.

Causal agent (see Greenhouse cucumber, leaf rot, 22.13)

Disease cycle (see Greenhouse cucumber, leaf rot) Leaf rot is essentially a disease of high humidity environments.

It usually accompanies heavy insect infestations. The fungus is a very common saprophyte and can be found on any decaying vegetation throughout the year.

Management

Cultural practices — Growers can reduce humidity by increasing plant spacing in the field and ensuring good ventilation in the greenhouse. Insect pests should be controlled.

Selected references (see Greenhouse cucumber, leaf rot)

(Original by W.R. Jarvis)

▶ 9.10 Powdery mildew *Fig. 9.10*

Powdery mildew is not an important disease in field-grown cucurbits but is a major problem in greenhouse cucumber crops (see Greenhouse cucumber, 22.15).

▶ 9.11 Pythium fruit rot (leak)

Pythium acanthicum Drechs.
Pythium anandrum Drechs.
Pythium aphanidermatum (Edson) Fitzp.
Pythium debaryanum Auct. non R. Hesse
Pythium helicoides Drechs.
Pythium irregulare Buisman
Pythium mamillatum Meurs
Pythium periplocum Drechs.
Pythium ultimum Trow
Pythium spp.

All species of cucurbits are susceptible to fruit rots caused by various *Pythium* spp. These diseases are more prevalent in cool, rainy seasons.

Symptoms Infection of fruits almost always occurs in mature fruit and from the blossom end, probably through minute cracks left by incomplete closure of the stone cell layer at flower abscission. The rots are dark green and water-soaked, developing into a fast-spreading, watery rot of the whole fruit. Beneath a humid leaf canopy, profuse cottony growth of the fungi may be evident. The fruits become so watery that the disease is often called leak.

Causal agent One or more of several species of *Pythium* may be involved. Their identification requires expert diagnosis in the laboratory. (For more information on *Pythium* spp., see Selected references; also see Bean, root rot, damping-off, seed decay; Beet, pythium and rhizoctonia root rots; and Carrot, cavity spot and pythium root dieback.)

Disease cycle *Pythium* species all produce long-lived oospores that permit their survival through several years of adverse conditions. Infection, however, mostly occurs from rain- or irrigation-splashed propagules. Zoospores are produced in sporangia and swim in water films through cracks in the fruit surface. Once in the fruit, the fungus invades the tissues very quickly, producing oospores again when the fruit is fully rotted.

Management

Cultural practices — *Pythium* spp. are cosmopolitan soil inhabitants and fruit rots may be expected wherever growing conditions are poor. Growers should ensure the soil is well drained and the spacing of the plants is adequate to allow ventilation. In areas with poor natural air drainage, rows should be oriented parallel to the prevailing wind. Fertilization practices that foster luxuriant foliage and overhead irrigation should be avoided where crops are at risk from pythium fruit rot.

Selected references

Drechsler, C. 1925. The cottony leak of cucumbers caused by *Pythium aphanidermatum. J. Agric. Res.* 30:1035-1042.
Tompkins, C.M., P.A. Ark, C.M. Tucker and J.T. Middleton. 1939. Soft rot of pumpkin and watermelon fruits caused by *Pythium ultimum. J. Agric. Res.* 58:401-475.
Van der Plaats-Niterink, A.J. 1981. Monograph of the genus *Pythium. Stud. Mycol.* 21. Centraalbureau v. Schimmelcultures, Baarn, The Netherlands. 242 pp.

(Original by W.R. Jarvis)

▶ 9.12 Pythium root rot *Figs. 22.7a-d*

Pythium aphanidermatum (Edson) Fitzp.
Pythium irregulare Buisman
Pythium ultimum Trow
Pythium spp.

Watermelon, honeydew melon, muskmelon, squash and cucumber may be affected by *Pythium*-induced damping- off in cold, wet soils, especially when premature direct seedings have been made. The roots of more mature and bearing plants may also be attacked.

Symptoms Damping-off of seedlings newly emerged from cold, wet soils is characterized by a softening of the stem at or just above the soil line and by plants toppling over (*22.7a-c*)**.** In wet conditions, profuse, white, cottony mycelial growth of one or more *Pythium* spp. may become evident.

In collapsed mature plants, the roots have light brown, depressed, water-soaked lesions from 3 to 15 mm in diameter. Lesions may grow together and cause rotting of long portions of the roots. Primary and secondary roots are all affected.

In Ontario, a sudden wilt (*22.7d)* has been attributed to "root-nibbling" by *Pythium* spp. In this disease, the plants collapse suddenly without any obvious macroscopic root damage. Tiny, necrotic feeder roots, however, consistently have *Pythium* spp. colonizing them. They are apparent only after culturing of diseased roots in a laboratory.

Causal agent *Pythium* spp. can be identified with certainty only after isolation in the laboratory. All species involved in cucurbit diseases form characteristic oospores and sporangia on agar media or on pieces of colony floating in water (see Selected references and pythium fruit rot, 9.11).

Disease cycle Pythium diseases are common and can be severe in cold, wet soils. *Pythium* species form sporangia in the soil which discharge swimming zoospores. These encyst at the host surface, germinate and colonize the host very quickly. "Root nibblers" seem to be restricted only to the feeder roots, although these species are the same as those forming gross lesions. When the infected tissues rot, they become full of thick-walled resistant oospores. These structures enable the pathogen to survive long periods of adverse conditions, for example, several years in dry soils.

Management

Cultural practices — Effective soil drainage is essential for the control of damping-off and root rotting of mature plants. A two-year rotation with non-cucurbit crops is suggested. Poorly drained and naturally cold soils should not be sown until the soil temperature is at least 15°C.

Chemical control — Seed treatment fungicides may aid in reducing seed decay.

Selected references

Gottlieb, M., and K.D. Butler. 1939. A *Pythium* root rot of cucurbits. *Phytopathology* 29:642-628.
McClure, T.T., and W.R. Robbins. 1942. Resistance of cucumber seedlings to damping-off as related to age, season of year, and level of nitrogen nutrition. *Bot. Gaz.* 103:684-697.
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Younkin, S.G. 1938. *Pythium irregulare* and damping-off of watermelon. *Phytopathology* 28:596.

(Original by W.R. Jarvis)

▶ 9.13 Scab (gummosis) *Figs. 9.13; 22.16*

Cladosporium cucumerinum Ellis & Arth.

Scab is a fungal disease with symptoms that resemble angular leaf spot. It is a widespread and occasionally severe disease, particularly in Ontario. Scab also occurs as a field disease on cucumber, melon, pumpkin, vegetable marrow and summer squash. It is also a serious market disease, especially in melon, pumpkin, squash and marrow.

Symptoms The fungus affects all parts of the leaves, stems and fruits. Numerous, pale, water-soaked leaf spots appear which turn ash-gray to white and are somewhat angular. Affected veinlets may turn brown. Internodes may be short causing a rosette reminiscent of cucumber mosaic disease. Elongate lesions develop on stems and petioles. At temperatures around 17°C, tip dieback may occur. Dead tissue cracks and badly affected leaves become tattered. Symptoms are most conspicuous on fruit at all stages of disease development. Sporulation is more profuse on fruit than on leaves. Young fruit is most susceptible.

On cucumber fruit, water-soaked spots enlarge and deepen rapidly to form irregular cavities about 1 cm across and 2 to 5 mm deep (9*.13).* Often, a golden brown, gummy exudate appears which may dry into brown beads. The fungus sporulates on the fruit and leaves and lines the lesion cavities with an olive-green felt. On older fruit and on more resistant cultivars, there is a corky, tan-colored layer that looks like an irregular scab; it may be slightly raised above the surrounding tissue *(22.16).*

On melon fruit, infection often occurs at the pedicel end in addition to the rest of the fruit surface. The flesh can rot to a depth of about 5 mm in cantaloupe. Rotting is less severe in honeydew melon.

Causal agent *Cladosporium cucumerinum* is a typical *Cladosporium,* resembling several saprophytic species. It has non- to bi-sepate ramoconidia that measure up to 30 by 3 to 5 µm. Aseptate conidia occur in long, branched chains. The conidia are smooth, cylindrical and rounded at the ends, or ellipsoid, fusiform or subspherical, and pale olive-brown. They measure 4 to 25 by 2 to 6 µm.

Colonies on potato-dextrose agar are pale olive-gray and felty with abundant sporulation. Hyphae are sometimes spirally twisted.

Disease cycle The disease is favored by cool, dry days and frequent rainy or dewy nights. The disease develops from mid-summer onward in cool seasons with heavy dews and fogs. The pathogen survives in plant residues. Dry spores may be windblown for considerable distances, although local inoculum is likely to be the most dangerous when cucurbits follow cucurbits. Prolonged high humidity induces sporulation. Spores are liberated and dispersed in dry conditions, but infection occurs in wet plants between 17 and 20°C. At 17°C, resistant plants can be infected. Above 21°C, the corky defence reaction of the host excludes the fungus. In stored melons, the disease develops well at 2 to 8°C.

Management

Cultural practices — Fields with fog pockets should be avoided, as should overhead irrigation in cool seasons. Growers should remove or turn under infested crop residues promptly after harvest. Rotation and the use of resistant cultivars are the primary means of control.

Resistant cultivars — Resistance is controlled by a single dominant gene, expression of which is dependent on temperature and tissue age. At 17°C, only restricted local lesions develop on resistant germplasm lines. On susceptible tissue, large water-soaked lesions and rapid death occur. Scab tolerance, rather than immunity, is usually quoted in seed catalogues.

Chemical control — Fungicides are available for scab control but none is completely effective. Control is very poor when night temperatures fall below 14°C. Dithiocarbamate fungicides may be successful if applied before fruit formation. The fungus is believed not to be seed-borne, but the use of fungicide-treated seed is advisable.

Selected references

Ellis, M.B., and P. Holliday. 1972. *Cladosporium cucumerinum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 348. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Walker, J.C. 1950. Environment and host resistance in relation to cucumber scab. *Phytopathology* 40:1094-1102.

(Original by W.R. Jarvis)

▶ 9.14 White mold (sclerotinia rot) *Figs. 9.14a-c; 22.18a-d*

Sclerotinia minor Jagger
Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

White mold on cucurbits is caused chiefly by *S. sclerotiorum,* but *S. minor* sometimes occurs on cucumber crops. *Sclerotinia sclerotiorum* is widespread throughout Canada, but *S. minor* is confined mostly to southwestern Ontario, principally as a lettuce pathogen (see Lettuce, drop, 11.9).

Symptoms This disease affects the stem (*9.14a)* and fruit of cucurbit plants. Infection usually begins where the tissue is dead or dying, such as wilted cotyledons and especially flowers that remain attached to fruit or adhere to some other part of the plant after dropping off. Affected tissues become water soaked and may show profuse, cottony white growth of the pathogen on rapidly spreading lesions. On fruit, infection occurs mainly at the flower end (*9.14b),* where the symptoms often are mistaken for those of gray mold. However, in white mold, the mycelium is always pure white (*9.14b,c),* never shades of gray or brown. Eventually, the

characteristic sclerotia are formed. Those of *S. sclerotiorum* are flattish, rounded, irregular, black, and measure 3 to 10 mm (*22.18d)*\ those of *S. minor* are smaller, 2 to 5 mm, and coalesced into expansive aggregates.

Causal agent (see Lettuce, drop, 11.9)

Disease cycle (see Lettuce, drop)

Management

Cultural practices — Weeds should be eradicated and trash piles removed and buried deeply. Field crop rows should be oriented parallel to the prevailing wind, with generous spacing in and between rows so that plants dry quickly after rain. Growers should avoid using overhead irrigation where this disease is prevalent.

Resistant cultivars — No resistant cucurbit cultivars are known but those with a more open growth habit are less susceptible than those with dense foliage in which water is slow to evaporate.

Chemical control — Dicarboximide and benzimidazole fungicides may be used, but fungicide tolerance may develop quickly. Efficacy should be closely monitored and spraying stopped at the first sign of tolerance.

Selected references

Mordue, J.E.M., and P. Holliday. 1976. *Sclerotinia sclerotiorum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 513. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by W.R. Jarvis)

VIRAL AND VIRAL-LIKE DISEASES

▶ 9.15 Cucumber mosaic *Figs. 9.15; 22.20a,b*

Cucumber mosaic virus

Cucumber mosaic is a widespread and sometimes severe disease of cucurbit crops when aphid populations are high and viruliferous weeds are present (see Greenhouse cucumber, cucumber mosaic, 22.20).

There are many fresh-market field and pickling cucumber cultivars that are moderately to highly resistant to this virus. Although not immune, these cultivars withstand infection well enough to produce a marketable crop.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 9.16 Zucchini yellow mosaic *Figs. 9.16a-c; 22.24a,b*

Zucchini yellow mosaic virus

Zucchini yellow mosaic virus, previously known as muskmelon yellow stunt virus, occurs in melon, squash, watermelon, field cucumber and greenhouse cucumber, as well as in zucchini and some wild cucurbit hosts. The first report of this virus in Canada was in garden squash and greenhouse cucumber in British Columbia in 1988.

Symptoms In zucchini, symptoms include a prominent yellow mosaic, necrosis and foliar distortion ("shoestring") *(9.16a; 22.24a*). Fruit symptoms depend on the stage of fruit development at the time of infection. Early infection may result in failure to set fruits; later infection results in fruits that are severely distorted (*9.16b,c; 22.24b*), small, and green with glossy yellow protuberances. In muskmelon, the flesh may be mottled and the seeds small and misshapen.

Causal agent Zucchini yellow mosaic virus is a potyvirus which has flexuous, filamentous particles of single-stranded RNA, about 750 nm long, and one coat protein. It is easily transmissible to a fairly wide range of hosts and is transmitted by aphids, particularly the green peach aphid, in a non-persistent manner.

Diagnostic hosts include *Chenopodium amaranticolor* Coste & Reynier and C. *quinoa* Willd. (chlorotic local lesions; no systemic infection); *Cucumis melo* L. (chlorotic local lesions; systemic vein clearing, yellowing, leaf distortion, stunting, and occasional necrosis); *Cucurbita pepo* L. (chlorotic local lesions; systemic vein netting, yellowing mosaic, and leaf distortion, often with necrosis and death of the whole plant); and *Gomphrena globosa* L. (well-defined local lesions; no systemic infection).

The virus is variable and several strains have been reported. A Canadian isolate from cucumber failed to react with antiserum to watermelon mosaic virus 1, reacted weakly with antiserum to watermelon mosaic virus 2, and strongly with antiserum to zucchini yellow mosaic virus.

Disease cycle The severity of symptoms depends on the stage of plant development at the time of infection. Early infections result in great crop losses because of failure to set fruit, while later infections cause substantial loss of fruit quality. Although the disease is spread by aphids, particularly the green peach aphid, spread from a single infected plant may be extensive in the absence of these pests. Such spread occurs as a result of transmission of sap from infected to healthy plants in the course of routine crop maintenance by workers. The virus can be seed transmitted but only at a very low rate.

Management Since aphids are vectors, rigorous aphid control is essential to control zucchini yellow mosaic in cucurbit crops.

Cultural practices — The virus can be spread in sap on fingers and tools during routine greenhouse procedures, so suspect plants should be rogued and buried. Wild cucurbit weeds and volunteer seedlings should be eliminated from fields and from the vicinity of greenhouses.

Chemical control — Although the use of insecticides for aphid control is appropriate, aphids are difficult to control (see Other insect pests, 9.22), and there is no other chemical control.

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(Original by W.R. Jarvis)

▶ 9.17 Other viral and viral-like diseases *Figs. 9.17a,b*

Aster yellows Aster yellows mycoplasma-like organism
Cucumber necrosis Cucumber necrosis virus
Watermelon mosaic Watermelon mosaic virus

Aster yellows is a minor disease of cucurbit crops in Canada (see Lettuce, aster yellows, 11.15).

Cucumber necrosis is an occasional disease of cucurbits in Ontario (see Greenhouse cucumber, cucumber necrosis, 22.21**).**

Watermelon mosaic *(9.17a, b)* is regarded as a minor disease of cucurbits in Canada (see Greenhouse cucumber, watermelon mosaic, 22.23).

(Original by W.R. Jarvis)

NON-INFECTIOUS DISEASES

▶ 9.18 Cold injury *Figs. 9.18a-c*

Field cucumbers require a temperature of between 18 and 24°C for optimum growth, with a minimum of 10°C and a maximum of 32°C. For seed germination, the minimum temperature is 16°C and the optimum is 35°C. Cucumber crops are very susceptible to freezing. When plants are injured by cold, affected leaves wilt *(9.18a),* turn black and die. Frozen vines and fruit may appear water-soaked and feel soft when handled.

On the prairies, direct-seeded cucumbers are especially prone to cold injury in the spring. Chilled cotyledons appear bleached and often become prematurely dry. Plants may recover if the true leaves are not damaged *(9.18b).* However, if the true leaves are also injured, then growth is retarded and some plants may die. Chilled seedlings are usually more susceptible to post-emergence damping-off.

In some parts of Canada, particularly in southern Ontario, growers protect early cucumber seedlings with low plastic tunnels that are removed when the first leaves are too large to be contained. Once the plants are exposed, they are very susceptible to temperatures below 13°C. If chilled, growth is severely retarded and leaves become brittle and susceptible to wind damage. Chilled fruit can become pale green or yellow and mottled *(9.18c).* If cool conditions persist for more than two days, the fruit surface becomes pitted and brown. Fruit quality diminishes rapidly and the fruit becomes susceptible to scab, even in resistant cultivars. Necrotic tissue is susceptible to gray mold and white mold.

Management

Cultural practices — Plastic tunnels or polyester floating row covers will protect young cucurbit plants down to 0°C.

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(Original by W.R. Jarvis and R.J. Howard)

NEMATODE PESTS

▶ 9.19 Northern root-knot nematode *Fig. 22.30d*

Meloidogyne hapla Chitwood

Cucurbits are highly susceptible to damage from this nematode.

Symptoms include conspicuous yellowing, stunting and early senescence. Fruits are fewer and smaller than normal. Prolific branching of rootlets, and production of small, spherical galls on roots are characteristic. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 9.20 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38; see also Management of nematode pests, 3.12

INSECT PESTS

▶ 9.21 Cucumber beetles *Fig. 9.21*

Spotted cucumber beetle *Diabrotica undecimpunctata howardi* Barber
Striped cucumber beetle *Acalymma vittatum* (Fabricius)

The spotted cucumber beetle, also known as the southern corn rootworm, occurs from the Rocky Mountains eastward to Ontario and Quebec in Canada. The striped cucumber beetle occurs in central and eastern Canada. Within their respective ranges, both beetles are found wherever commercial field cucumber is grown. Yield losses caused by these beetles in Ontario are estimated at 15% if control measures are not implemented.

Adults of the striped cucumber beetle prefer such field cucurbits as cucumber, muskmelon, pumpkin, squash and watermelon, and they will feed on bean, corn, pea and the blossoms of other plants. The spotted cucumber beetle is a general feeder on many plants. Its adults feed extensively on all field cucurbits.

Damage Field-grown cucurbit plants are most susceptible to cucumber beetle damage when they are small. Soon after seedlings emerge, adult beetles chew small holes in the leaves and cotyledons, giving the affected plant parts a "shot-hole" appearance. Although many small holes may coalesce to form larger holes, the surrounding tissue remains relatively healthy. At this stage, feeding by adult beetles close to the ground often results in broken stems and eventual plant death. Later, the adult beetles attack flowers, which affects yield, and they may completely skeletonize leaves, leaving only the veins. The larvae of both cucumber beetles tunnel into the base of the plant stems, which may cause wilting. Damage from leaf, stem or root feeding by adults and larvae is generally minimal on older, established plants.

Cucumber beetles transmit bacterial wilt and cucumber mosaic virus, both of which can result in losses far greater than direct feeding by either species of beetle.

Identification Cucumber beetles belong to the family Chrysomelidae. Adults of the striped cucumber beetle (9.27) are 5 to 6 mm long with a black head, a yellow thorax and three longitudinal black stripes on yellow forewings (elytra). Adults of the spotted cucumber beetle *(9.21)* are about 6-7 mm in length and yellow-green with 12 large dark spots on the elytra.

Larvae of the two species of cucumber beetles are indistinguishable from each other, both being slender, legless, white, and less than 1 cm in length with reddish-brown heads. Cucumber beetle larvae can be differentiated from larvae of the squash vine borer because the latter exceed 1 cm in length and have thoracic and abdominal legs.

Adults of the striped cucumber beetle may be confused with western corn rootworm adults (see Maize, 12.15), which also are attracted to the yellow cucurbit flowers later in July. The adults of these two beetles can be distinguished by the elytral stripes, which are straight-sided only in the striped cucumber beetle. Also, the outer leg segments of the striped cucumber beetle are black, giving it the appearance of wearing black socks. In contrast, the western corn rootworm adult has elytra with a wavy central stripe and its legs are entirely black.

Life history Cucumber beetles overwinter as adults that hibernate in dense grass and under leaves and other plant residue. When spring temperatures reach 10°C or higher, the beetles leave their winter quarters and feed on pollen, petals and leaves of various plants. In early to mid-June, when cucumber, zucchini and other cucurbit seedlings emerge or are transplanted to the field, the beetles begin to feed on the leaves and stems, and they mate while feeding on the plants. Eggs are laid in the ground near the host plant. The larvae hatch in about 10 days, feed on the roots for about a month and pupate in the soil. Adults emerge within two weeks and may feed on the rind of cucurbit fruits until frost forces them to seek shelter. There is one generation per year.

Management The main concern is that adult cucumber beetles may transmit bacterial wilt and virus diseases while they feed. Because it is impossible to determine if adult cucumber beetles in the field contain the pathogens of bacterial wilt or virus diseases, control of the initial population of adult beetles is paramount for a successful crop.

Monitoring — Growers should monitor their fields in early spring. Early morning or evening scouting is best and both the foliage and soil-stem areas should be examined. Under windy conditions, the beetles may remain in difficult-to-find places; they often try to conceal themselves by moving around the plant. At each of five different sites within a field, 20 consecutive plants should be examined. Control is warranted if two or more beetles are found at three of the five sites.

Chemical control — If significant beetle populations are present, insecticidal treatments should start at plant emergence and be repeated 10 days later if necessary. Continual monitoring is essential.

(Original by R.E. Pitblado and R.N. Lucy)

▶ 9.22 Other insect pests *Figs. 9.22 a-c; see text*

European earwig *Forficula auticularia* L.
Melon (cotton) aphid *Aphis gossypii*
Glover Potato leafhopper *Empoasca fabae* (Harris)
Seedcorn maggot *Delia platura* (Meigen)
Squash bug *Anasa tristis* (DeGeer)
Squash vine borer *Melittia cucurbitae* (Harris)
Tarnished plant bug *Lygus lineolaris* (Palisot de Beauvois)
Cutworms
Wireworms

A number of insects that are general feeders, such as those listed here, are minor pests of field cucurbit crops in various areas of Canada. General suggestions for control include the use of trap crops and cultural practices, such as incorporating crop residue by cultivating soon after harvest. Applications of chemical insecticides as seed and foliar treatments, if used at all, should be properly timed.

European earwig

(see Crucifers, 8.43; *3.14T1; 8.43a-d)*

Melon aphid

(see Greenhouse cucumber, 22.33) The melon aphid is a pest on field cucurbits and other crops. Its eggs may overwinter on weeds in some parts of Canada, but winged females regularly invade from the United States and winged forms (*22.33a*) also disperse from populations developing locally during the growing season. The melon aphid affects young leaves, shoots, flowers and young fruits but on field cucurbits it is most important as a potential virus vector. In general, it is difficult to control aphids on field cucurbits with insecticides; growers should try to encourage naturally occurring biocontrol agents, such as green lacewings (see Beneficial insects, mites and pathogens, 3.7).

Potato leafhopper

(see Potato, 16.46) The potato leaf- hopper (*16.46b*) is occasionally a minor pest on field cucurbits. It feeds on the underside of cucurbit leaves and causes the leaf margins to turn yellow (“hopperburn”) *(16.46a).* This type of damage is most noticeable under dry conditions. Leafhoppers are readily controlled by foliar insecticides but treatments must be applied before the appearance of extensive yellowing.

Seedcorn maggot

(see Bean, 15B.18) The seedcorn maggot attacks cucumber and other field cucurbits early in the spring *(9.22a,b).* Seed treatments provide good control.

Squash bug

The squash bug (family Coreidae) occurs in southern British Columbia, Ontario and Quebec. It is primarily a pest of pumpkin and squash, although it also will attack other cucurbits. Nymphs and adults suck sap from the leaves, stems and vines, causing light-colored areas that later turn brown and die. Occasionally the nymphs and adults feed on the fruit, particularly in the fall after the leaves have been killed by frost. The squash bug over winters as an adult in sheltered places, under grass and plant residue, along fencerows and in buildings. In June, the adults fly into fields and lay large, yellow eggs in clusters on the foliage of susceptible plants. The eggs turn brown as they mature. Nymphs hatch in 7 to 17 days. The nymphs are pale at first, but soon turn gray. They feed throughout the summer and either become adult or perish before winter. There is one generation per year. Cultural control can be achieved in small field areas and home gardens by removing potential overwintering sites. Because adults tend to congregate under shelter at night, shingle or board traps can be placed near squash and pumpkin plants in June, followed by early morning inspection and hand removal of adults to reduce their numbers. In commercial or large field operations, insecticidal spray applications should be timed to control the nymphs when they are young.

Squash vine borer

is a clear-winged moth (family Sesiidae) *(9.22c).* It occurs in southwestern Ontario chiefly on thickstemmed field cucurbits, such as gourd, pumpkin, squash and vegetable marrow. The larva is a fleshy white stem borer with three pairs of thoracic and five pairs of abdominal legs. At maturity, it exceeds 1 cm in length, thus differing from cucumber beetle larvae, which are less than 1 cm in length. Stems invaded by the squash vine borer eventually become filled with a moist, slimy frass, and affected plants wilt permanently. The larvae may be found inside the stems and in the pulp of maturing fruit. Adults can be monitored, using yellow-pan water traps (see McLeod & Gualtieri, 3.14). Control is best achieved by drenching the base of the plants with an insecticide in late June before larvae enter the stems, and repeating treatment on a weekly basis.

Tarnished plant bug

(see Celery, 7.21) The tarnished plant bug *(7.21b,d,e)* feeds on flowers, leaves and stems of field cucurbits, such as cucumber and squash. Plant bug feeding reduces flower set, although the bug itself is seldom noticed. Growers tend to ignore rather than control plant bugs.

Cutworms

(see Tomato, 18.35; *6.25a-c; 18.35c-g)* and

Wireworms

(see Maize, 12.21; *12.21a,b)* are incidental pests of field cucurbits and seldom require control. Replanting may be required and seed treatments are recommended, particularly against wireworms.

(Original by R.E. Pitblado, R.N. Lucy and J.A. Garland)

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Additional references

FUNGAL DISEASES

▶ 10.1 Canker of hop

Fusarium sambucinum Fuckel
(teleomorph *Gibberella pulicaris* (Fr.:Fr.) Sacc.)

Infection just above the crown can result in girdling and sudden wilting of hop vines. The presence of an obvious canker and the sudden death of the plant differentiates this disease from verticillium wilt, in which the symptoms appear gradually, starting with the lower leaves. Canker has been a minor problem on commercial hop. Prompt removal of infected vines is reported to reduce *Fusarium* inoculum and subsequent infections.

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(Original by D.J. Ormrod)

▶ 10.2 Downy mildew of hop *Fig. 10.2*

Pseudoperonospora humuli (Miyabe & Takah.) G.W. Wils.

Downy mildew is the most important disease of hop worldwide. It was first reported in Japan in 1905, in North America in 1910, and in Britain in 1920. It is well established in the hop fields of coastal British Columbia. Hop is the only host of *P. humuli.*

Symptoms The first sign of infection occurs early in the spring when the new shoots are just emerging from the crowns. Systemically infected shoots, known as primary basal spikes, are stunted, pale and have tightly clustered, curled leaves *(10.2).* Secondary infections of otherwise healthy shoots appear later as discrete spots or brown patches on the growing tips, leaves, flowers and cones. Infected cones may be unsaleable.

Causal agent The mycelium of *Pseudoperonospora humuli* is nonseptate and intercellular. Sporangiophores are 200 to 460 by 7 µm, with wide- spreading, stiff, dichotomous branches. Sporangia are 22 to 30 by 16 µm, ellipsoid with an apical papilla, and germinate by producing zoospores. Oospores are 25 to 40 µm, spherical and have a smooth, light brown wall.

Disease cycle The pathogen overwinters in infected hop crowns and can be carried to new locations as mycelium in diseased rootstocks. Infected dormant buds give rise to primary basal spikes in the spring. When the ambient temperature exceeds 6°C, sporangiophores emerge through the stomates of infected tissue and release sporangia. If free water is available, the sporangia that land on hop leaves or other organs germinate to form motile zoospores. The zoospores encyst and form germ tubes that can penetrate the host tissue, usually through the stomates.

Once inside the host, the mycelium continues to grow, symptoms appear and sporangia are produced when temperatures range from 10 to 25°C. Secondary infection can occur anytime during the growing season when the foliage is wet from rain. Periods of wetness from dew are inadequate for infection.

Management

Cultural practices — Only disease-free crowns should be used for planting and replanting. Volunteer hop plants should be removed from the vicinity of hop yards. Primary basal spikes should be pruned off early in the spring before the pathogen has a chance to sporulate. Regular pruning should be delayed as long as possible to shorten the exposure period of vines selected for training.

Resistant cultivars — The cultivar Fuggles is resistant to downy mildew, while Brewer's Gold, Bullion and Cascade are tolerant.

Chemical control — In Canada, one drench application of a systemic fungicide in early spring reduces the level of primary inoculum. Later, when weather conditions favor mildew, conventional, protectant fungicide sprays can be used to prevent secondary infection. In Europe, disease forecasting systems have been used for several years and, in some cases, have been successful in reducing the number of spray applications by up to 50%.

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(Original by D.J. Ormrod)

▶ 10.3 Leaf scorch of parsley *Figs. 10.3a,b*

Alternaria radicina Meier, Drechs. & E.D. Eddy
(syn. *Stemphylium radicinum* (Meier, Drechs. & E.D. Eddy) Neergaard)

Leaf scorch of parsley has damping-off and petiole spot phases. It has not yet been reported on parsley but does occur on other umbelliferous crops (see Carrot, black rot, 6.7).

Symptoms Seedlings fail to emerge or topple over after emergence *(10.3a).* Later, dark brown to black lesions may develop on petioles and leaves *(10.3b).* These symptoms can be confused with those caused by other *Alternaria* spp.

Causal agent (see Carrot, black rot, 6.7) Damping-off, petiole lesions and dark leaf spots with evidence of black mycelium and/or conidia usually indicate the presence of *Alternaria* spp. Conidia of *A. radicina* are unbeaked, whereas those of other *Alternaria* spp. have long, flexuous or branched beaks, or the beak is shorter than the conidium body.

Disease cycle (see Carrot, black rot, 6.7) *Alternaria radicina* is spread by infested seed and diseased crop residue. It can survive in the soil for up to six years. Conidium viability is lost more quickly if diseased residue is buried rather than being left on the soil surface. Conidium germination and infection requires one to two weeks and is most rapid at relative humidities over 90% and temperatures above 27°C. A fresh crop of conidia can be produced within two to three weeks and, under favorable conditions, secondary spread and infection occur.

Management

Cultural practices — Parsley growers should follow a rotation, avoiding not only parsley but also carrot, parsnip, celery and celeriac. Where the disease has occurred, a four- to five-year rotation is suggested. Pathogen-free seed, if available, should be used, otherwise seed should be hot-water treated at 50°C for 20 minutes. Diseased crop residues should be turned under to reduce the spread of fungal spores.

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(Original by D.J. Ormrod)

▶ 10.4 Leaf spots of parsley *Fig. 10.4*

Alternaria leaf spot *Alternaria dauci* (Kühn) Groves & Skolko
Phoma leaf spot *Phoma anethi* (Pers.:Fr.) Sacc.
(synanamorph *Cercosporidium punctum* (Lacr.) Deighton)
Septoria leaf spot *Septoria petroselini* (Lib.) Desmaz.

Parsley is susceptible to several fungal leaf spot diseases that are closely related to those occurring on carrot and celery.

Symptoms Alternaria leaf spots are irregularly shaped and dark brown to black with a yellow border. When numerous, they may coalesce, giving the leaflets a blighted appearance.

Phoma anethi causes small, sub-circular to irregular, olive to brown spots and can cause blighting of the foliage when numerous. Dill and fennel also are susceptible *(10.10a).*

Septoria leaf spots *(10.4)* are small, somewhat angular, grayish brown, with a definite dark cinnamon-brown margin. They often contain dark pycnidia on the upper side of the foliage. Small, oval, cinnamon-brown lesions may be present on the petioles also. Early lesions may not be readily evident on curled parsley cultivars because of extreme leaf curling and small leaf size.

Causal agent Differentiation of the three genera of fungi that most commonly cause leaf spots on parsley requires microscopic examination of mature lesions with conidia.

Alternaria dauci (see Carrot, alternaria leaf blight, 6.5) conidia have long flexuous or branched beaks.

Phoma anethi is the pycnidial state of *Cercosporidium punctum,* which produces a stroma from which simple, mostly continuous conidiophores with geniculations arise. The conidia are smooth, clavate, cylindrical to obclavate, mostly pale brown, few- to several-celled, and measure 18 to 51 by 4 to 9 µm.

Septoria petroselini forms dark pycnidia that are immersed in the leaf tissue and contain hyaline conidia with three to four erosswalls. Conidial dimensions are 30 to 40 by 1 to 2 pm.

Disease cycle All three pathogens can be carried in or on seed, thereby introducing leaf spot diseases to new areas. These fungi can also survive in undecomposed crop residues. Conidia are produced at temperatures between 10 and 30°C, and they are disseminated by splashing water, by direct contact through handling, and on infested tools. Infection, symptom expression and sporulation can occur in 10 to 15 days at 20°C.

Management

Cultural practices — Parsley growers should obtain seed from dry regions where the diseases do not occur, or use a hot-water treatment at 50°C for 20 minutes to disinfest seed. Sanitation and crop rotation are important steps in reducing the survival of the pathogens in the soil. All umbelliferous crops should be avoided in rotation with parsley. Where no disease has been observed, a two- to three-year rotation should suffice. Where disease problems have occurred, a four- to five-year rotation is suggested. Irrigation should be timed to minimize periods of leaf wetness.

Resistant cultivars — The parsnip-rooted cultivars Early Sugar and Hamburg Thick-Rooted are highly resistant to *Septoria petroselini,* while Plain Dark Green Italian and Improved Market Gardener are resistant, and the curled- leaf cultivars Sherwood Decorator and Banquet are highly susceptible.

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(Original by D.J. Ormrod and R.F. Cerkauskas)

▶ 10.5 Powdery mildew of hop, mint, sage and parsley *Fig. 10.5*

Erysiphe cichoracearum DC.:Mérat

Sphaerotheca macularis (Wallr.:Fr.) Lind
(syn. *Sphaerotheca humuli* (DC.) Burrill)

Powdery mildew, caused by *Erysiphe cichoracearum,* can be a serious disease of scotch spearmint. It is usually a minor problem on peppermint, wild mint (*Mentha arvensis* L.) and sage. *Erysiphe cichoracearum* has a wide host range, mostly on Compositae, and occurs in physiologic strains. Other species of *Erysiphe* and *Sphaerotheca* have been reported on mint in North America. *Erysiphe heraclei* DC. has been recorded in Europe. In the fall of 1990, a 500 m^2 outdoor planting of parsley in coastal British Columbia was 100% infected by an *Erysiphe* species, probably *E. heraclei,* although no cleistothecia were observed.

Powdery mildew caused by *S. macularis* occurs worldwide on hop and on strawberry and several other members of the rose family (Rosaceae). This is the oldest known hop disease, having first been observed early in the 19th Century, but it is no longer a significant problem. Mechanical harvesting, in which the vines are completely removed from the field, has probably been effective in eliminating most of the overwintering inoculum. Powdery mildew resistance has been bred into some of the newer hop cultivars.

Symptoms A powdery, gray-white growth of conidia and conidiophores occurs on the upper surface of leaves and on stems. Infected leaves eventually turn yellow *(10.5)* and drop. Small brown or black fruiting bodies (cleistothecia) may appear on diseased tissues late in the season.

Causal agent (For a description of *Erysiphe cichoracearum,* see Lettuce, powdery mildew, 11.12.)

Sphaerotheca macularis has hyaline, highly branched mycelium that grows on both leaf surfaces but is more persistent on the upper surface. Conidia are in chains, ellipsoidal to barrel shaped, 25 to 38 by 15 to 23 µm. Cleistothecia are globose, dark brown to black, 60 to 125 µm in diameter, with numerous brown, unbranched appendages three to five times the diameter of the cleistothecia. A single ascus, measuring 50 to 90 by 45 to 75 µm and containing eight ascospores that are 18 to 25 by 12 to 18 µm, differentiates it from *E. cichoracearum,* which has multiple asci.

Disease cycle Mildew fungi overwinter as mycelium or cleistothecia on infested stubble. In the spring, conidia or ascospores infect new foliage. Conidial production occurs throughout the summer, but cleistothecia are produced only in the late summer or fall. Little is known about the effect of growing conditions on the incidence of powdery mildew, which is often most severe during cool cloudy weather.

Management

Cultural practices — Fields should be clean cultivated in the fall to bury infested crop residues. Where practical, flaming can also be used to destroy diseased stubble.

Resistant cultivars — Where powdery mildew has been a recurring problem, the use of alternative cultivars should be investigated.

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(Original by D.J. Ormrod)

▶ 10.6 Pythium root rot of parsley *Figs. 10.6a,b*

Pythium spp.

In Canada, root rot of parsley has been attributed to unidentified *Pythium* spp. In Ireland, *Pythium paroecandrum* Drechs. is reported as the cause of root rot on this plant. *Pythium* spp. can attack a wide variety of herbs and spices.

Symptoms Affected plants appear yellow and stunted *(10.6a).* Immature roots are brown and, in severe cases, the entire root system may be rotted *(10.6b).*

Causal agent (see Carrot, pythium root dieback, 6.13) Confirmation of *Pythium* spp. requires isolation on selective culture media.

Disease cycle *Pythium* fungi are soil-borne. High populations can build up if diseased roots are left to rot in the soil. Severe root rot can occur if parsley is seeded into infested beds. High soil moisture favors infection.

Management

Cultural control — For parsley, improved drainage, such as the use of raised beds, and rotation with unrelated crops, such as cereals, corn and allium crops, are recommended. Where feasible, removal of infected plants will reduce the carry-over of inoculum.

Selected references

McCracken, A.R. 1984. *Pythium paroecandrum* associated with a root rot of parsley. *Plant Pathol.* 33:603-604.

(Original by D.J. Ormrod)

▶ 10.7 Rust of mint *Figs. 10.7a,b*

Puccinia menthae Pers.:Pers.

Rust of mint occurs worldwide. It is the most troublesome disease reported by fresh mint growers in British Columbia. There are two races of *P. menthae* on mint; one infects peppermint and the other spearmint. Both races can attack scotch spearmint. The only other rust on mint is *P. angustata* Peck, for which sedges (Cyperaceae) are the primary hosts and mint is the alternate host on which pycnia and aecia occur.

The numerous races of mint rust have varying abilities to infect different hosts. Wild mint (*Mentha arvensis* L.), savory, dittany (*Cunila* spp.) and the herb *Hedeoma* spp. may be attacked by races of *P. menthae* that infect cultivated mints.

Symptoms Rust is usually first noticed in early spring. New shoots are swollen, exhibit yellow, blister-like spots, break off easily, and have malformed, chlorotic leaves *(10.7a).* Aeciospores are produced in reddish-brown blisters on young shoots and infect developing leaves. Later, cinnamon-brown pustules containing urediniospores appear on the stems and undersides of leaves *(10.7b).* Leaf infection results in destruction of the oil glands, yellowing and defoliation. Dark brown teliospores form on the stems and occasionally on the rhizomes in the fall. The gross symptoms and presence of spore forms appropriate for the time of year are sufficient to identify the disease.

Causal agent *Puccinia menthae* has globose pycnia. 90 to 160 µm in diameter, which occur in small groups with the aecia, which are 0.3 to 0.4 µm in diameter. Aeciospores are spheroidal or ellipsoidal, 18 to 24 µm in diameter, and hyaline or pale. Uredinia are scattered or in small concentric groups, buff colored and up to 0.5 µm in diameter. Urediniospores are ellipsoidal or obovoidal, 22 to 26 by 18 to 22 µm, with a finely echinulate wall. Telia are similar to uredinia but dark brown. Teliospores are ellipsoidal, obtuse above, usually with slightly projecting caps, slightly constricted at the septum, and measure 22 to 30 by 17 to 24 µm. The spore wall is yellow-brown and may be verrucose or smooth.

Disease cycle *Puccinia menthae* is an autoecious, macro- cyclic rust fungus. It overwinters as teliospores on mint stubble and on wild or volunteer plants. These spores germinate to form basidia and basidiospores in the late fall to early spring. The basidiospores infect the earliest shoots from late winter to early spring, depending on the geographical area. In spearmint but not in peppermint, the basidiospore infection appears to result in systemic invasion of the shoots. These infections give rise to the pycnial and aecial stages. Aeciospores are produced from early spring to early summer, with the heaviest production in the spring. They disperse over short distances, generally less than one metre, and infect newly emerging leaves, resulting in the uredinial stage. Urediniospores are produced from spring until late summer and infect new leaves under favorable conditions, finally leading to the telial stage in the fall. Urediniospores are responsible for long-distance spread and can result in areawide epidemics. Rust is most severe during cool, damp growing seasons. Urediniospore viability is greatly reduced by bright sunlight and air temperatures greater than 32°C.

Management

Cultural practices **—** Rhizomes can be hot- water treated to rid them of adhering teliospores if it is necessary to start a new planting of a cultivar that cannot be grown from seed. Hot-water treatment should be done under carefully controlled conditions and only where isolation from infected mint makes it worthwhile. Infection can be greatly reduced by turning under crop residues in the fall or early spring, although this is not recommended where there is a danger of spreading verticillium wilt. Flaming the entire crop in the spring to break the cycle between the aecial and uredinial stages has been the most successful control method over the past 25 years. Timing is critical in that all stubble and new growth must be destroyed on an area-wide basis so that the late flush of new growth can emerge without danger of being infected by air-borne urediniospores from adjacent fields or border areas. Flame treatment is highly effective for peppermint and common spearmint; it is not recommended for scotch spearmint because the rust can overwinter on rhizomes.

Resistant cultivars — The peppermint cultivars Murray Mitcham and Todd's Mitcham are resistant to rust under moderate disease pressure.

Selected references

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(Original by D.J. Ormrod)

▶ 10.8 Sooty mold of hop

Fumago vagans Pers.

Sooty mold grows on honeydew produced by the hop aphid and probably by other aphid species during feeding. If unchecked, the entire plant may be covered with the fungus, resulting in contamination and downgrading of the cones. An effective aphid control program will prevent this problem and may also reduce the spread of hop mosaic virus, which is transmitted by the same aphid.

Management When aphid populations are high enough to require the use of control measures, damage from sooty mold may be prevented by the use of registered insecticides.

(Original by D.J. Ormrod)

▶ 10.9 Verticillium wilt of mint and hop *Figs. 10.9a-d*

Verticillium albo-atrum Reinke & Berth.
Verticillium dahliae Kleb.

Verticillium wilt caused by *V. dahliae* is the most serious disease of mint in the major production areas of North America. It has led to the decline of the mint industry in the midwest United States and an accompanying shift in production to Oregon and Washington, where it is now the major disease as well. The distribution of verticillium wilt on mint in Canada is not known. It has been identified in field-grown peppermint in southern Alberta. In Nova Scotia, *V. dahliae* has been reported on savory.

Verticillium wilt caused by *V. albo-atrum* can be a very destructive disease on hop if it is caused by a virulent or "progressive" strain. Both progressive and less virulent, "fluctuating" strains have been identified in England, where considerable research has been done over the past 60 years. Strains of *V. dahliae* can also infect hop, but rarely cause serious symptoms; in Canada, verticillium wilt caused by *V. dahliae* has been found occasionally on hop, but there are no records of it causing significant losses.

Symptoms Infected peppermint and spearmint plants show any or all of the following symptoms: asymmetric leaf growth *(10.9c),* dwarfing, chlorosis (*10.9d*), browning or purpling of the leaves, wilting, stem cankers, death of lower leaves, and finally death of the plant *(10.9a).* The disease first appears in small, well-defined areas that can expand rapidly to cover entire fields within three to five years. The presence of symptomatic plants from which *V. dahliae* can be isolated is indicative of the disease. Other *Verticillium* species may be isolated, but they tend to be non-pathogenic to mint.

On hop *(10.9b),* infected plants exhibit a progressive yellowing and dying of the leaves from the bottom up during the latter part of the growing season. Cutting into the bark shows light brown discoloration of the woody tissue extending well up from the base of the vine. Severely infected vines die completely before harvest, but the crowns survive and in subsequent years send up new vines, which may or may not show symptoms.

Causal agents (see Greenhouse cucumber, verticillium wilt, 22.17)

Disease cycle *Verticillium dahliae* can be introduced on infected plant rhizomes of mint or as microsclerotia in crop residues that are wind-blown or otherwise moved from infested fields. It can persist as microsclerotia in crop residues or in soil for five years or more. When mint is planted in the field, the roots can be infected directly from soil- borne microsclerotia. The fungus then spreads through the roots and into the stems where vascular browning occurs and microsclerotia are produced. Infection is encouraged by high soil moisture and temperature; symptom expression is most pronounced under warm, dry conditions.

In hop, the pathogen overwinters in infected crowns and can be introduced to a new field through infected root cuttings. Propagules can also survive in crop residues in the soil for several years after the removal of an infected crop. Wilt caused by *V. dahliae* has been seen where hop was planted in fields previously cropped to hosts such as potato (see Potato, verticillium wilt, 16.20). Most infection occurs through root contact with mycelial fragments in crop debris, or with microsclerotia in the case of *V. dahliae.*

Management

Cultural practices — Mint growers should plant certified, disease-free rhizomes, especially in fields where mint has not previously been grown. Infested fields should be removed from production before wilt becomes severe. Fields of peppermint with low levels of disease, but which are to be kept in production, should be flamed within one week of harvest to slow the build-up of inoculum. Fields with low to moderate levels of disease can be replanted after rotation for four to five years to non-susceptible crops.

In areas where *V. albo-atrum* is a serious problem on hop, an integrated control program is required. This involves the use of disease-free planting stock, rotation with grasses or cereals for a minimum of two years after removal of a diseased crop, the use of resistant cultivars, and restricting nitrogen fertilization. In areas where *V. dahliae* is of greater concern, the use of disease-free planting stock and the avoidance of fields previously cropped to a susceptible host are generally sufficient.

Resistant cultivars — Todd's Mitcham or Murray Mitcham peppermint cultivars can be used in infested fields, provided inoculum levels are not excessive.

Biological control — Cross-protection by inoculation with the non-pathogenic species *Verticillium nigrescens* Pethybr. has shown promise.

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(Original by D.J. Ormrod)

▶ 10.10 Other fungal diseases of herbs *Figs. 10. 10a-c*

Published records of other fungal diseases of herbs and spices in Canada are scant and incomplete; they refer to anthracnose, blights, downy and powdery mildews, gray mold, leaf scorch and leaf spots, stem and root rots, rusts, and rusty root. In some cases, microorganisms have been identified in association with diseased plants, but no pathogenicity tests have been conducted. In other cases, only tentative identifications of pathogens have been made.

When diagnosing diseases on herbs and spices, lists of pathogens and abiotic disorders on other closely related plants should be consulted. The following list of diseases and causal agents is provided for general information:

Anise
- leaf spot (*Cercospora mcilkoffii* Bubâk)
- rust *{Puccinia pimpinellae* (F. Strauss) Mart.)
- stem rot (*Sclerotinia sclerotiorum* (Lib.) de Bary; see Bean, white mold, 15B.9)

Basil
- powdery mildew (*Sphaerotheca macularis* (Wallr.:Fr.) Lind; see powdery mildew, 10.5)

Borage
- leaf spot (*Ramularia* spp.)

Burnet
- powdery mildew (*Sphaerotheca macularis*; see powdery mildew, 10.5)

Caraway
- stem rot (*Sclerotinia sclerotiorum*; see Bean, white mold, 15B.9)

Chives
- downy mildew (*Peronospora destructor* (Berk.) Casp. in Berk.; see Onion, downy mildew, 13.6)
- rust (*Puccinia allii* F. Rudolphi)

Coriander
- anthracnose (*Gloeosporium* spp.)

Corn-salad
- leaf spot (*Septoria* spp.)

Dill
- phoma blight (*10.10a*) (*Phoma anethi* (Pers.:Fr) Sacc.; see leaf spots of parsley, 10.4)
- rusty root (*Alternaria* spp., *Cylindrocarpon* spp.)
- stem rot (*Sclerotinia sclerotiorum*; see Bean, white mold, 15B.9)

Fenugreek
- leaf spots (*Cercospora traversiana* Sacc.)
- powdery mildew (*Erysiphe polygoni* DC.; see Crucifers, powdery mildew, 8.12)
- thielaviopsis root rot (*10.10b*) (*Chalara elegans* Nag Raj & Kendrick; see Bean, black root rot, 15B.4)

Horseradish
- leaf spot (*Cercospora armoraciae* Sacc., *Entylomella armoraci- ae* (Fuckel) Sif.)
- white rust *{Albugo candida* (Pers.) Kuntze; see Crucifers, white rust, 8.15)

Lavender
- leaf spot *{Septoria lavendulae* Desmaz.)

Lemon balm
- gray mold *{Botrytis cinerea* Pers.:Fr.; see Lettuce, gray mold, 11.10)
- leaf spot *{Phoma exigua* Desmaz.; see Lettuce, phoma rot, 11.11)

Sage

powdery mildew *{Erysiphe cichoracearum* DC.:Mérat; see Lettuce, powdery mildew, 11.12)
stem rot (*Sclerotinia sclerotiorum,* see Bean, white mold, 15B.9)

Savory
root rot (*Pythium oligandrum* Drechs.; see pythium root rot, 10.6)
wilt (*Verticillium dahliae* Kleb.; see verticillium wilt of mint and hop, 10.9)

Tarragon
root rot (*10.10c) {Fusarium* sp.)
rust (*Puccinia tanaceri* var. *dracunculina* (Fahrendorff) Cummins)

Thyme
gray mold (*Botrytis cinerea;* see Lettuce, gray mold, 11.10)

Management In general, fungal diseases of herbs and spices must be controlled by cultural means.

Cultural practices — Good soil drainage, crop rotation, proper fertilization, the use of disease-free seed and the destruction of infested plant residues are important methods.

Chemical control — Fungicides have been registered for use only on dill and horseradish.

Selected references

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(Original by D.J. Ormrod and R.J. Howard)

VIRAL AND VIRAL-LIKE DISEASES

▶ 10.11 Aster yellows *Figs. 10.11 a, b*

Aster yellows mycoplasma-like organism

Parsley, dill *(10.11a,b)* and sage are susceptible to aster yellows (see Lettuce, aster yellows, 11.15).

Symptoms Symptoms on parsley may resemble those of carrot motley dwarf and celery mosaic. Significant damage can occur in areas where the leafhopper vectors are numerous (see Lettuce, aster leafhopper, 11.23).

(Original by D.J. Ormrod and R.J. Howard)

▶ 10.12 Miscellaneous viral diseases *Fig. 10.12*

Broad bean wilt virus
Carrot motley dwarf (carrot mottle virus plus carrot red leaf virus)
Celery mosaic virus
Cucumber mosaic virus
Hop mosaic virus (hop latent virus)
Hop nettle head (arabis mosaic virus plus prunus necrotic ringspot virus)
Tomato spotted wilt virus

Several viruses are capable of infecting hop, parsley, and possibly other herbs and spices. In hop, these viruses can cause a range of symptoms from non-expression (latent) to severe.

Parsley is susceptible to a number of viral diseases, such as carrot motley dwarf (70.72), which is caused by a combination of carrot mottle virus and carrot red leaf virus. Other viral diseases, such as celery mosaic, cucumber mosaic and broad bean wilt, also affect parsley but cause mild or no symptoms. None of these viruses causes significant loss in Canada.

Tomato spotted wilt (see Greenhouse tomato, tomato spotted wilt, 25.22) may affect sage, lemon balm and peppermint.

Symptoms Hop mosaic virus causes severe damage to the cultivar Goldings. Leaves are mottled and curled, and the vines are stiff and cannot climb. Infected plants die within one or two years. Other cultivars, including Fuggles, are symptomless carriers. Hop plants infected with nettle head disease exhibit rigid vines with shortened internodes and do not twist normally. Carrot motley dwarf causes dwarfing, and leaf reddening and yellowing in parsley, much as in carrot; celery mosaic causes a golden-yellow chlorosis with necrotic spotting in parsley; cucumber mosaic and broad bean wilt cause little or no symptoms in parsley.

Causal agent Arabis mosaic virus is a nepovirus with isometric particles about 30 nm in diameter. Broad bean wilt virus is an RNA-containing fabavirus with isometric particles about 25 nm in diameter. Carrot mottle virus has enveloped, isometric, RNA-containing particles that are approximately 52 nm in diameter. Carrot red leaf virus is a luteovirus that has RNA-containing, isometric particles about 25 nm in diameter. Celery mosaic virus, classed as a potyvirus, has flexuous, filamentous particles about 780 nm in length. Cucumber mosaic virus (see Greenhouse cucumber, cucumber mosaic, 22.20) has isometric particles that are 30 nm in diameter. Hop mosaic virus has rod-shaped particles about 656 nm in length. Prunus necrotic ringspot is an ilarvirus with quasi-spherical particles, 25 to 35 nm in diameter.

Carrot motley dwarf can be identified by symptoms and by mechanical and aphid transmission of the viral pathogen to indicator plants. Celery mosaic is identified by serology, electron microscopy and transmission to celery, which becomes malformed as a result of systemic infection. Broad bean wilt and cucumber mosaic can be confirmed serologically or by inoculation onto diagnostic indicator plants.

Disease cycle Arabis mosaic virus is transmitted by the nematode *Xiphinema diversicaudatum* (Micoletzky) Thome, which has a limited distribution in Canada, and by seed and sap. Carrot mottle virus is transmitted in a persistent manner by the carrot-willow aphid. It is usually associated with carrot red leaf virus in umbelliferous plants. Celery mosaic is transmitted in a non-persistent manner by several aphid species, including the green peach aphid, from infected celery, poison hemlock (*Conium maculatum* L.), and other umbelliferous plants. Cucumber mosaic and broad bean wilt both have a wide host range, numbering many plant families, and are transmitted by numerous aphid species, including the green peach aphid. Hop mosaic virus is transmitted by several species of aphids, including the hop aphid and the potato aphid. Prunus necrotic ringspot virus is transmitted by sap and seed but there is no known vector.

Management Aphid control is important. In hop plantings affected by nettle head disease, control of the nematode vector is important.

Cultural practices — Prompt incorporation of the residues from diseased crops and destruction of wild and volunteer plants will reduce the carry-over of viruses and their vectors.

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(Original by D.J. Ormrod and R.J. Howard)

INSECT PESTS

▶ 10.13 Aphids *Figs. 16.41a,b; 16.42a-c*

Carrot-willow aphid *Cavariella aegopodii* (Scopoli)
Green peach aphid *Myzus persicae* (Sulzer)
Hop aphid *Phorodon humuli* (Schrank)
Potato aphid *Macrosiphum euphorbiae* (Thomas)
Other aphids

The carrot-willow aphid occurs in southwestern British Columbia and the Maritime provinces in Canada. It transmits carrot mottle and carrot red leaf viruses in parsley and other umbelliferous plants. Other aphids, including the green peach aphid (see Potato, 16.41), are important in the transmission of celery mosaic, cucumber mosaic and broad bean wilt viruses in parsley.

The hop aphid is the only aphid that has been identified from hop in British Columbia; however, the potato aphid is widespread and common on numerous crops and weeds in that area. Both aphids are vectors of the virus causing hop mosaic, the most important virus disease of hop. Honeydew produced by aphids supports the growth of sooty molds, which can disfigure hop (see sooty mold of hop, 10.8).

Management Aphid control on parsley and hop may be achieved by the use of registered pesticides when necessary. At present, other strategies are not available for managing aphids on these crops.

(Original by D.J. Ormrod)

▶ 10.14 Flea beetles *Figs. 10.14a, b*

Hop flea beetle *Psylliodes punctulata* Melsheimer
Horseradish flea beetle *Phyllotreta armoraciae* (Koch)
Other crucifer-feeding flea beetles *Phyllotreta* spp.

The flea beetles occurring on mustard are the same as those on canola and cole crops (see Crucifers, 8.44). These and other species can attack hop and horseradish. The hop flea beetle is native to North America. It is present in low numbers across most of Canada. It feeds on hop as well as crucifers, garden beet, and rhubarb. The horseradish flea beetle is an incidental and sporadic pest on horseradish in Canada.

Damage After overwintering, adult flea beetles (*10.14a)* feed on the cotyledons and first true leaves of young plants early in the spring, causing small round holes that give the leaf a "shot-hole" appearance *(10.14b).* Extensive flea beetle feeding on mustard causes seedling mortality, delayed plant development and maturity, unevenness in plant height, reduced seed yield and increased chlorophyll content of the seed. Horseradish flea beetle larvae mine the petioles and leaf midribs. Because horseradish roots increase in size in the latter part of the growing season, defoliation by flea beetles moving onto the crop after other cruciferous crops are harvested may result in decreased root size. Overwintered hop flea beetles attack and kill seedlings as these appear just above the soil level, although damage to well-established plants may be light. Flea beetle damage tends to be less in wet years.

Identification (see Crucifers, flea beetles, 8.44)

Life history Flea beetles usually have one generation per year in Canada. Adults overwinter in leaf litter and emerge from late April to early May. They fly to fields, mate, lay eggs and feed on cruciferous weeds and crops when these emerge. Eggs are laid in the soil on or near the roots of host plants, and the larvae feed on the roots; or, in the case of the horseradish flea beetle, on the petioles and midribs of host plants. The prepupal and pupal stages develop in the soil, and adults appear from mid-July onward and seek hibernation sites in late September and early October.

Management *Monitoring* — Growers should look for the shot-hole type of injury that is typical of adult feeding on transplants or cotyledons as seedlings emerge.

Cultural practices — There is higher resistance to flea beetle injury in yellow (white) mustard, *Sinapis alba* L., than in Oriental brown mustard. *Brassica juncea* (L.) Czem. & Coss. Late or delayed seedings and higher seeding rates of direct- seeded crops minimize flea beetle damage. Cruciferous weeds and volunteer crucifers should be controlled.

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(Original by J.J. Soroka)

▶ 10.15 Other insect pests *Figs. 10.15a-c; 8.43b,cK*

Black swallowtails *Papilio* spp.
Carrot rust fly *Psila rosae* (Fabricius)
European earwig *Forficula auricularia* L.

Black swallowtail larvae (see Parsnip, 14.7) (*10.15a)* and carrot rust fly larvae (see Carrot, 6.23) (*10.15b)* are found on parsley and sometimes on dill in home gardens. The European earwig (see Crucifers, 8.43) will eat and defecate on leaves of many plants, including basil and parsley *(10.15c).* The following records on "horsemint" in Manitoba were supplied by A.J. Kolach: leafrollers, *Pyrausta* sp.; sap beetle larvae, *Carpophilus* sp.; and a clear-wing moth borer, possibly *Ramosia rileyana* (H. Edwards).

OTHER PESTS

▶ 10.16 Mites and slugs *Fig. 11.27c*

Mites The mite pests on herbs and spices in Canada are unknown, apart from *Dictyna* and *Tetragnatha* species recorded on "horsemint" in Manitoba by A.J. Kolach, and the two-spotted spider mite *Tetranychus urticae* Koch on hop. (For more information about the two-spotted spider mite, see Greenhouse cucumber, 22.36.)

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Slugs Most vegetable crops in home gardens suffer some degree of slug damage (see Lettuce, 11.27), but dill is particularly prone to attack. The slugs climb the stems of young dill plants and totally devour the leaves. At sites where slugs are numerous, dill tops will be reduced *(11.27c).*

(Original by J.A. Garland)

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11 Lettuce, chicory, endive

Figures *11.1* to *11.27; 11.24T1, T2*
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BACTERIAL DISEASES

▶ 11.1 Bacterial soft rots *Figs. 11.1 a, b*

Bacterial wilt (dry leaf spot)

Xanthomonas campestris pv. *vitians* (Brown) Dye

Head rot (slime rot)

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*

These bacteria also cause soft rot. *Erwinia* is by far the more widely distributed and about 80% of slime rots are caused by *E. carotovora,* mostly in hydroponic greenhouse production, and in marketed heads as a storage disease. Both bacteria are common pathogens on lettuce, endive and chicory. They affect most vegetable crops and some weeds.

Symptoms Bacterial wilt or dry leaf spot caused by *Xanthomonas campestris* pv. *vitians* begins as water- soaked spots in wilted, wedge-shaped areas along the margins of the leaves. As the spots enlarge, they become olive- green around the edge and dry in the center. Infection can extend to the stem, resulting in an olive-green decay and hollowing. The causal bacterium is seed-borne.

Erwinia carotovora subsp. *carotovora* causes wilting of the lower leaves and vascular discoloration in the stem and leaf veins of the growing crop. Stems are generally soft, water- soaked and discolored dark green, brown or black. Dark spots on leaves spread (*11.1a),* leading to a slimy breakdown of one or more leaves and eventually involving the entire head (*11.1b).* The slime phase, in which some leaves or the entire head are infected, is the most common cause of loss in the market place. *Erwinia* infection is easily recognized by soft rot and slime symptoms that are not common to other lettuce pathogens. Species of *Sclerotinia* and *Botrytis* also cause soft rots, but they are accompanied by characteristic mycelium. (See also pseudomonas diseases, 11.3.)

Causal agents To differentiate these bacteria, isolation and comparison of colony color and flagellar characters is a minimum for presumptive diagnosis.

Xanthomonas campestris pv. *vitians* is an aerobic, Gram-negative rod, measuring 0.7 to 3.0 by 0.4 to 0.5 µm. It occurs singly or in pairs and has a single polar flagellum. Colonies on agar are yellow, convex and shiny, and produce a yellow pigment, xanthomonadin.

(For a description of *Erwinia carotovora* subsp. *carotovora,* see Potato, bacterial soft rot, 16.2.)

Disease cycle Both bacteria are relatively weak pathogens and require a wound such as frost injury or mechanical damage to infect a healthy plant. *Erwinia* is most likely to infect injured or harvested lettuce. It commonly follows other pathogens and non-parasitic disorders such as tipburn. Slime development in transit follows infection in the field or during harvest and packing procedures. Breakdown can be rapid and complete if temperatures are allowed to rise in transit and during warehousing.

Management

Cultural practices — Lettuce crops should remain relatively free of bacterial soft rots if grown without excessive nitrogen or overhead irrigation. Because the bacteria are often secondary to other pathogens, a comprehensive disease control program may be required. For dry leaf spot, crop rotation and control of wild lettuce may significantly reduce inoculum. To reduce populations of *Erwinia carotovora* subsp. *carotovora,* most vegetable crops should be avoided in rotations and non-susceptible crops such as cereals, grasses or corn should be grown. To deter both diseases, good air and soil drainage and avoidance of sprinkler irrigation when crops are nearing maturity are suggested. Proper packing and storage procedures also should be observed. Care during harvest and handling to minimize injury and rapid cooling of the harvested heads will reduce bacterial soft rot losses. Heads that are trimmed and wrapped prior to packing suffer less injury from soft rot than untrimmed naked heads that are frequently forced into bulge-packed cartons. Transit and storage temperatures should be as close to 0°C as possible without freezing.

Selected references

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.2 Infectious corky root

Rhizomonas suberifaciens van Brüggen & Jochimsen

Infectious corky root occurs in fields repeatedly cropped to lettuce. As yet, it has not been reported in Canada. The symptoms of this disease can be confused with those of non-infectious corky root (see non-infectious corky root, 11.20). *Rhizomonas suberifaciens* has been reported only on lettuce.

Symptoms Above-ground symptoms range from no apparent effect to yellowing of lower leaves, wilting, stunted growth and poor head formation. The first symptoms on the roots are yellow lesions that enlarge and coalesce until the entire tap root is dark brown, rough and cracked. Lower feeder roots slough off and more roots may be produced near the soil surface. Similar symptoms can be induced by ammonia or nitrite liberated from nitrogenous fertilizers and from chicken manure.

Causal agent *Rhizomonas suberifaciens* is a fastidious, soil-borne, Gram- negative bacterium that has only recently been isolated and characterized (see non-infectious corky root, 11.20). It is rod-shaped (0.3 to 0.6 by 0.6 to 1.4 pm), aerobic and microaerophilic, oxidase positive and catalase negative. It has a single, lateral flagellum. Optimum growth occurs at 29 to 33°C. Colonies are circular, umbonate and translucent, becoming raised at the edge and wrinkled in the centre with age. It produces a low-molecular weight toxin (MW less than 340), which is heat stable and soluble.

On soils repeatedly cropped to lettuce, unthrifty growth and a brown corky root system are suggestive of the disease. Confirmation requires isolation of the causal bacterium. Nutritional imbalances can cause similar symptoms.

Disease cycle The causal organism can persist in the soil for at least three years in the absence of lettuce, and can survive and damage crops equally in sandy and clay soils. The disease tends to build up in incidence and severity with continued lettuce cropping. The number of marketable heads and head weight both decrease with increasing bacterial populations in the soil. Under experimental conditions, disease severity and reduction in plant growth were lowest at 10°C and greatest at 31°C. This finding supports field observations that the disease is generally more severe in warm climates and in summer and fall crops compared to spring crops.

Management

Cultural practices — Poor drainage, excessive irrigation, and soil compaction are thought to aggravate the disease. Regular crop rotation is recommended to prevent corky root. When it occurs, a longer rotation is recommended, including green manure crops. Raised beds and reduced irrigation are recommended to encourage deep rooting.

Resistant cultivars — Tolerant cultivars, including Green Lake, Marquette and Montello, are available, and there is considerable resistance in other species of *Lactuca*.

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(Original by D.J. Ormrod)

▶ 11.3 Pseudomonas diseases *Figs. 11.3a-d*

Slime
Brown rot
Varnish spot
Butt rot
Head rot

Pseudomonas cichorii (Swingle) Stapp
Pseudomonas fluorescens (Trevisan) Migula (syn. *Pseudomonas marginalis* (Brown) Stevens)
Pseudomonas viridiflava (Burkholder) Dowson (syn. *Pseudomonas viridilivida* (Brown) Stevens)

The *Pseudomonas* species listed here all cause soft-rot; they are widely distributed and occur commonly in soil as opportunistic pathogens that can cause significant crop losses from time to time. Pseudomonas diseases often have a very complex etiology, with causal organisms often occurring together. Isolation of the pathogen(s) is necessary to be sure of the cause. This complex etiology has also led to confusion in disease names, so that they have come to be descriptive of symptoms without attribution to a specific pathogen.

Pseudomonas species can infect numerous dicotyledonous hosts. Lettuce, endive and chicory are subject to attack, especially in mechanically damaged and soft-grown plants.

Symptoms More than one bacterium is usually associated with slime rot and brown rot. Slime rots are characterized as water-soaked, translucent wet rots. Brown rots appear as localized oval-shaped, brown to red lesions *(11.3a-c).*

Pseudomonas cichorii causes small, irregular, yellow to brown, circular spots that enlarge and grow together, often along the leaf veins. These spots may be black under wet conditions or pale and papery under dry conditions. This bacterium is also responsible for varnish spot, in which shiny, dark-brown, firm, necrotic spots measuring a few mm in diameter occur on the blades and petioles of the inner leaves. The wrapper leaves show no symptoms, making the disease impossible to detect at harvest without removing the outer leaves.

Pseudomonas fluorescens causes marginal leaf spots. Decayed tissue appears first at the leaf margins and then progresses down the leaves *(11.3b).* The veins become brown and the affected area of the blade turns brown to black under wet conditions or pale and papery under dry conditions. The same bacterium is implicated in butt rot, in which the pith of the stem develops a firm dark-green decay that is apparent when the stems are cut at harvest.

Pseudomonas viridiflava has been reported on lettuce, producing a decay that tends to follow the midrib of older leaves *(11.3d).* Under favorable conditions, the disease will move to younger leaves, but it does not affect the stem or root.

Causal agents *Pseudomonas* species are aerobic, pectolytic, Gram-negative rods with several polar flagella. They form white colonies on nutrient agar and produce a fluorescent pigment on King's B medium. The gross symptoms of infected plants, combined with isolation of characteristic bacterial colonies that fluoresce on King's B medium, are sufficient to identify these pathogens, at least to genus. Nutritional, physiological, protein fingerprinting, and serological techniques differentiate the species.

One survey of chicory roots revealed 61 pathogenic isolates of bacteria in seven taxonomic groups. The rots fell into the slime rot and brown rot groups. *Pseudomonas marginalis* was responsible for about half the rots, together with *P. viridiflava* and three groups of unidentified fluorescent pseudomonad bacteria.

Pseudomonas cichorii is an obligately aerobic, Gram-negative rod, which is about 0.8 by 1.3 µm, motile with several polar flagella, and nonspore-forming. Colonies on nutrient agar are round, white, slightly raised, translucent, and have somewhat irregular margins. On King's B medium, a fluorescent, greenish pigment is formed and may diffuse into the agar.

Pseudomonas fluorescens is an aerobic Gram-negative rod, measuring 0.7 to 0.8 by 2.3 to 2.8 µm. It is normally motile with polar multitrichous flagella. Occasionally it is non-motile. Cultures produce diffusible fluorescent pigments, especially on iron-deficient media, such as King's B medium.

Colonies of *P. fluorescens* are slimy on media containing 2 to 4% sucrose because of levan formation. The bacterium is non-lipolytic. It is able to denitrify and hydrolyse gelatin but is unable to hydrolyse starch. This bacterium produces a highly effective biosurfactant known as vis- cosin (see Crucifers, head rot, 8.3).

Pseudomonas viridiflava is an aerobic. Gram-negative, non-sporing rod with one to two polar flagella. Colonies on nutrient agar vary from cream- colored to yellowish, mucoid and convex to grayish, flatter and matt. A greenish, fluorescent pigment is formed on King's B medium.

In general, pathogenic, fluorescent pseudomonads can be distinguished from non-pathogenic pseudomonads on the basis of LOPAT tests for levan production, oxidase, potato soft rot, arginine dihydrolase, and tobacco hypersensivity. *Pseudomonas fluorescens, P. cichorii* and *P. viridiflava* are distinguished by their ability to utilize nitrate, sorbitol, tartrates and other substrates (see Table 11.3 and Selected references, Schaad 1988).

Table 11.3 Key to differentiate fluorescent Pseudomonas species on lettuce

Test	*P.fluorescens*	*P.cichorii*	*P.viridiflava*	*P.syringae*
Oxidase	+	+	–	–
Arginine dihydrolase	+	–	–	–
Nitrite to nitrogen	+	–	–	–
Growth at 41 °C	–	–	–	–
Utilization of:				
Mannitol	+	+	+	V
Geraniol	–	–	–	–
Benzoate	–	–	–	–
Cellobiose	–	–	–	–
Sorbitol	+	+	+	V
Trehalose	+	–	–	–
Sucrose	+	–	–	V
m-tartrate	V	+	+	V
D-tartrate	V	–	+	V
D-arabinose	–	–	–	–
L-rhamnose	V	–	–	–

Tobacco hypersensitivity	–	+	+	+
Potato rot	+	–	+	–
Symbols: + positive; – negative; V variable.				

Disease cycle Pseudomonas diseases usually follow some type of injury or abnormally wet conditions. Varnish spot can occur in the absence of injury but only on lettuce plants approaching maturity. The latter disease is believed to result from splashing soil and water during sprinkler irrigation.

Management

Cultural practices — In the field, good soil drainage and air movement through the crop canopy deter pseudomonas diseases. Overhead irrigation should be avoided when heads are approaching maturity, particularly in fields with a history of varnish spot.

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(Original by D.J. Ormrod and W.R. Jarvis)

FUNGAL DISEASES

▶ 11.4 Anthracnose (ring spot), fire of endive *Fig. 11.4*

Microdochium pancittonicinum Sutton, Galea & Price in Galea, Price & Sutton
(syn. *Marssonina panattoniana* (Berl.) Magnus.)

This disease is prevalent in overwintered crops and during cool, wet weather in the fall. It commonly infects early crop transplants if inoculum has been allowed to build up in fields previously cropped to lettuce. It is also common in unheated greenhouses with a history of monocrops of lettuce or endive (see Greenhouse lettuce, anthracnose).

Anthracnose affects lettuce, endive and chicory, and also has been reported on related composite weeds, which may serve as a reservoir of inoculum. Isolates from endive can infect chicory and the related *Cichorium pumilum* Jacq., garden lettuce and prickly lettuce *(Lactuca serriola* L.).

Symptoms The fungus causes anthracnose or ring spot on lettuce as well as various leaf spots on weeds in the Compositae. It also causes fire disease in endive. The first symptoms of infection, tiny water-soaked lesions, appear on the undersides of leaves and on veins and petioles. The lesions enlarge to form straw-colored spots, 2 to 4 mm in diameter (7*1.4).* Eventually, the centers of the spots turn white and dry and frequently drop out, giving a characteristic and diagnostic shot-hole appearance. Lesions on the midrib are sunken, more elongate, measure 1 by 4 to 5 mm, and tend to grow together, producing an overall rusty appearance. Outer leaves wilt and, in severe cases, heart leaves may rot entirely. Infected plants tend to be stunted and yellow-brown. Under humid conditions, white to pink spore masses may appear at the edge of the lesions. In endive, the disease is named fire because the lesions on the midribs are red.

Causal agent *Microdochium panattonianum* may or may not produce conidiophores. If present, they are produced in acervuli embedded in infected leaf tissue. The conidiophores are one- to two-septate, hyaline, smooth, discrete, sparsely branched near the point of origin or integrated, with one to four conidiogenous cells. They are 7.5 to 16 µm long, cylindrical to irregular or lageniform, with the conidiogenous region narrower, 1.5 to 2.0 µm in width with a broader base, 2.5 to 4.0 µm. The conidiogenous region proliferates enteroblastically to produce additional conidia at successively higher levels, sometimes combined with sympodial holoblastic proliferation.

Conidia are about 5 to 15 pm, slightly curved and two-celled, with the apical cell larger and slightly beaked. They are holoblastic, hyaline, dry, fusiform, curved, one- to two-septate, obtuse at the apex, with the upper cell wider and the lower cell strongly tapered to a truncate base, measuring 12.5 to 15.5 by 2.5 to 3.5 pm and forming effuse, white to pink, smooth masses that are eguttulate or with several small guttules.

The fungus is readily isolated from conidia on the host. Growth in culture is slow. On malt-extract agar and potato-dextrose agar, colonies have a pale, fleshy pink color with a raised convoluted appearance. On water agar, the fungus forms numerous bulbous cells in a sparse, white, spreading mycelium.

Disease cycle The fungus may be seed-borne but overwinters primarily as conidia, mycelium and microsclerotia in residue from diseased plants and in wild hosts. The primary source of inoculum is infested leaf residue left at or near the soil surface. Germination and infection occur between 15 and 34°C when wind-blown or splashed inoculum contacts lettuce seedlings. The disease can be serious in marketed produce.

Management

Cultural practices — Before plants are started, growers should thoroughly clean and disinfest greenhouse and seedbed areas, and eliminate wild lettuce from the vicinity of propagating areas and fields. Seed should be disease-free, desert-grown, and treated with an approved fungicide. In fields with a history of disease, lettuce and endive should be rotated with non-susceptible crops for at least one year. A long rotation is necessary to decompose inoculum in accumulated trash. Fields should be deep plowed because the fungus can survive for very long periods in dry residue on the soil surface. Weeds should be controlled because they provide a humid microclimate conducive to infection.

Growers should avoid working in infected crops when they are wet, take precautions to avoid moving spores on clothing and equipment, and avoid sprinkler irrigation in fields suspected of having a high population of microsclerotia.

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.5 Black root rot *Fig. 11.5*

Chalara elegans Nag Raj & Kendrick
(synanamorph *Trichocladium basicola* (Berk. & Broome) J.W. Carmichael)
(syn. *Thielaviopsis basicola* (Berk. & Broome) Ferraris)

Black root rot has been seen occasionally on chicory in southwestern Ontario and has the potential to be a problem where the pathogen has built up significant populations, for example on old tobacco soils. It is a major disease of chicory in South Africa. The pathogen has a very wide host range, attacking plants in over 40 genera and 15 families (see Bean, black root rot, 15B.4).

Symptoms Lesions on the taproot vary from superficial, light brown spots to sunken, gray to black areas extending to 3 cm in diameter and 3 to 4 mm deep (77.5). Sometimes the entire internal root tissue has a brown marbled appearance. Secondary roots blacken and die.

Causal agent (see Carrot, black root rot, 6.6)

Disease cycle Little is known of the epidemiology of black root rot on chicory. Chlamydospores enable it to survive for long periods in soil. Research in South Africa has shown that the optimum temperature for infection is about 25°C, with marbling symptoms mostly appearing at 30°C.

Management

Cultural practices — Growers should follow a long rotation, particularly after crops such as tobacco, which is readily infected by the pathogen. The soil in forcing beds should be disinfested if the black root rot pathogen is present.

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(Original by W.R. Jarvis)

▶ 11.6 Bottom rot *Figs. 11.6a,b*

Rhizoctonia solani Kühn

(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

The causal fungus is responsible for bottom rot, a major head rot of lettuce, and it is a principal cause of wirestem or late damping-off. It is a common soil inhabitant in virtually all soils and occurs in several anastomosis groups, some of which are widely distributed. Lettuce, related vegetable crops and some weeds are potential hosts of *Rhizoctonia solani.*

Symptoms Symptoms first appear when head lettuce is approaching maturity and the basal leaves have reflexed and are in direct contact with the soil. Rust-colored sunken lesions appear on the midrib of lower leaves (*11.6a).* Under dry conditions, the lesions enlarge slowly and it may be possible to salvage diseased crops by trimming infected leaves at harvest. Under damp conditions, the lesions expand over the entire midrib and cause the leaf blade to collapse. If conditions are favorable, the fungus will rot the leaves one by one as it progresses upward and inward *(11.6b).*

Bottom rot differs from drop disease in having no conspicuous mycelium, and from gray mold in having no obvious sporulation. Gray mold has gray to brown spore masses. Drop has a fluffy white mycelium associated with the affected tissues; however, mixed infections are not unusual.

Causal agent (see Bean, rhizoctonia root rot, 15B.7)

Disease cycle (see Bean, rhizoctonia root rot, 15B.7) The fungus is active during warm weather, provided moisture is adequate. Under damp conditions, seedlings or more mature plant parts become infected through direct contact with mycelium in the soil. If plant damage is extensive, numerous sclerotia may form, thus increasing the overwintering inoculum.

Management

Cultural practices — It is advisable to seed early during the cooler part of the growing season and to rotate lettuce with grasses, cereals, legumes or other nonhosts in order to increase organic matter and decrease the population of the pathogen. Growers should avoid growing head lettuce in fields with a history of bottom rot and plant in areas with good soil and air drainage. Growing lettuce on raised beds provides improved air circulation near the plant base, where infection is most likely to occur.

Resistant cultivars — Early maturing and more upright types of lettuce such as Romaine are more likely to escape infection.

Chemical control — Soil fumigation helps control bottom rot.

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(Original by D.J. Ormrod)

▶ 11.7 Damping-off, stunt *Figs. 11.7a-c*

Bacteria
Pythium spp.
Other fungi

Damping-off is a frequent problem in early lettuce when cold, wet weather follows seeding. Several soil- and seed- borne fungi and bacteria may cause damping-off, but *Pythium* species are most important and are well known for their role in damping-off, seed decay and root rot. Most seedling vegetable crops are subject to damping-off, but lettuce is particularly vulnerable.

Symptoms Poor emergence, sudden collapse and death of seedlings and unthrifty growth are characteristic. Onset usually coincides with cool, damp conditions following seeding. Under these conditions, soil-borne fungal pathogens may attack and kill seedlings that would otherwise survive under warmer, drier conditions.

In early or pre-emergence damping-off, seedlings fail to emerge either because the seed decays before germination or the seedlings die before reaching the soil surface. In late or post-emergence damping-off, seedlings emerge but they are infected, usually at the soil line, and collapse (*11.7a).* Older seedlings also may be infected in the outer stem or root tissues. They continue to live but with reduced vigor, showing symptoms known as wirestem or root rot. Such plants rarely reach marketable size or quality. Pythium infection may move into the crown tissues, causing "stunt" or collapse of older plants (*1 1.7b,c).*

Causal agent (see Beet, pythium root rot, 5.7; and Carrot, cavity spot, 6.8, and pythium root dieback, 6.13)

Late damping-off is frequently caused by the bottom rot fungus *Rhizoctonia solani.*

Disease cycle Most field soils contain pathogens that can cause damping-off. Some organisms, such as *Pythium* spp., have long-lived resting spores. Others grow saprophytically on decomposing organic matter. (For detailed information, see bottom rot, 11.6; see also Beet, pythium root rot, 5.7; and Carrot, cavity spot, 6.8, and pythium root dieback, 6.13.)

Management

Cultural practices — Raised beds or well- drained soils should be used for early plantings. Growers should avoid seeding or transplanting into cold, wet soils.

Chemical control — It is advisable to use seed that has been freshly treated with an appropriate fungicide.

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(Original by D.J. Ormrod)

▶ 11.8 Downy mildew *Figs. 11.8a, b*

Bremia lactucae Regel

Downy mildew is a common fungal disease wherever lettuce is grown. It is a disease of cool, wet weather. It is most damaging in the field on early spring or late fall crops. It can also be a major disease of greenhouse lettuce. Isolates of *Bremia lactuca* from cultivated lettuce are restricted in host range to species of the same taxonomic sub-section of *Lactuca.*

Symptoms Early infection of seedlings causes a cessation of cotyledon growth that leads to stunting or death of the plant. Sporulation occurs on both sides of the cotyledons, which become chlorotic. Cotyledons become less susceptible as they age and true leaves are less susceptible than cotyledons. Leaves of infected seedlings display slight chlorosis and a rolling of the leaf margins. Severe early infection may delay maturity and result in crops of inferior quality.

On older plants, the first sign may be the appearance of sporangiophores from leaf stomata *(11.8a).* These appear as discrete white projections that are visible to the naked eye. The sporangiophores are usually confined to the undersurfaces of mature leaves but, occasionally, they may occur on the upper leaf surface. On older leaves, lesions appear as light green or yellow areas delimited by large leaf veins on the upper surfaces. These chlorotic lesions turn necrotic or translucent and become brittle, especially near the leaf margin *(11.8b).* The fungus may become systemic in the plant and cause a black-brown discoloration of stem tissues and leaf bases near the shoot tips of mature heads. Diseased leaves often become infected by soft rot bacteria and fungi.

Downy mildew is frequently complicated by the presence of secondary soft-rotting bacteria and trimming waste may be considerable in marketed produce.

Causal agent Sporangiophores of *Bremia lactucae* emerge from stomata in groups of one to three and measure 200 to 1500 by 6 to 19 µm. They are septate with three to seven dichotomous branches, giving the sporangio- phore a tapering, disk-like appearance. The branch tips are swollen into an inverted, cone-like, tapering vesicle, measuring 8 to 15 pm and bearing three to five sterigmata each with a single sporangium. The sporangium is spherical to ovoid, hyaline, and measures 12 to 31 by 11 to 28 µm with a slightly thickened papilla. It has a pedicel up to 2 µm in length. Sporangia resemble those of *Phytophthora infestans,* a closely related fungus that causes late blight of potato and tomato. Oogonia vary in size up to 30 pm. Antheridia are probably diclinous. Oospores are spherical, aplerotic, 20 to 31 µm (mean 25 µm), with smooth walls about 3 pm thick. Remnants of the oogonium may adhere to the oospore wall, giving it a yellow-brown wrinkled appearance.

The disease can be identified by leaf symptoms and microscopic examination of the sporangia and sporangiophores, but race identification requires inoculation of differentially resistant cultivars of lettuce. The white mold on the underside of infected leaves glistens and can be seen with the naked eye. White sporulation from stomata with a very characteristic disk-like mass of sporangiophore branches is diagnostic.

Disease cycle *Bremia lactucae* survives between crops as mycelium and oospores in residue from infected crops and in cultivated and wild lettuce plants that overwinter. Oospores form in infected tissue and are produced in diseased leaves. They are stimulated to germinate near the host. Sporulation occurs at high humidity at night and coni- dia are dispersed by wind or splashing water. Sporangia germinate between 0 and 21°C (optimum around 10°C) to produce hyphae or zoospores, both of which are infective and can penetrate the epidermis within three hours. Infection occurs directly through the stomata. A coenocytic mycelium ramifies through the leaf tissue and produces dendroid sporangiophores that emerge through the stomata of the lower leaf epidermis. Infected tissue eventually turns brown, giving rise to the term "brown margin," and a downy weft of white mold develops on the underside of infected leaves. Sporangia are produced in abundance when night temperatures range from 5 to 10°C and day temperatures are 12 to 20°C, with an overcast sky and relative humidity near 100%. They are readily windblown. Germination and infection can occur if sporangia land on a susceptible host that is covered by a film of moisture for at least five to seven hours. In adjacent successive plantings, the first diseased crop will serve as a source of inoculum for subsequent plantings.

Management

Cultural practices — Crop rotation is a standard method of control for the downy mildews, coupled with deep plowing to bury all crop residues. Fields with a history of downy mildew and impeded soil or air drainage should not be used for early and late plantings.

Resistant cultivars — The use of resistant cultivars has had only limited success because the resistance has been quickly overcome by the pathogen. Analysis of the genetics of host resistance and pathogen virulence has demonstrated that the introduction of resistant cultivars promotes the rapid appearance of the corresponding virulent races of *B. lactucae.* If resistant cultivars are to be introduced into lettuce-growing areas, the racial composition of the *B. lactucae* population should be assessed beforehand.

Chemical control — Protective fungicides are registered in Canada but should be used only in an early preventive program when no other strategy appears to be effective.

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.9 Drop (white mold) *Figs. 11.9a-f*

Sclerotinia minor Jagger
Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia disease of lettuce is usually referred to as drop. On other crops, it is often called white mold or watery soft rot. It also is a major disease of chicory and endive where it is known as white mold or sclerotiniose. *Sclerotinia* spp. often are widespread in the soil. *Sclerotinia sclerotiorum* is much more prevalent than *S. minor. Sclerotinia* spp. have a very wide host range, including many weeds and vegetable crops, such as cabbage, carrot, celery, lettuce, tomato and bean.

Symptoms On lettuce, the name "drop" reflects the first obvious symptom. Plants at various stages of maturity appear wilted and the outer leaves drop to the ground, while remaining attached to the plant (7*1.9a).* The fungus infects the petioles and spreads to the center of the head. Once these symptoms appear, the plant is unharvestable. Pulling up a plant with drop usually reveals a fluffy white mycelium and large, dark, oval or round sclerotia *(S. sclerotiorum) (11.9b)* or aggregates of small irregular sclerotia in various stages of development *(S. minor)* (7*1.9d).*

Any tissues may be affected, and initial infections, like those of gray mold, are often associated with pieces of dead, dying or wounded tissue. Stem infections (77.9c), particularly with *S. minor*, usually occur at the soil level on senescent cotyledons or in leaf axils. Large black sclerotia, often lying in a mass of white mycelium, are characteristic of *S. sclerotiorum* (7*1.9b,f).* Smaller, coalescing masses of always external sclerotia are typical of *S. minor.* Apothecia (77.9c) are inconspicuous but may be found at the soil surface by careful searching in spring and throughout cool moist summers.

White mold is often confused with gray mold, particularly when there is copious development of the cottony mycelium. The mycelium of *S. sclerotiorum* is pure white, not the off-white of gray mold. Large sclerotia lying loosely on the white mycelium are easily visible under leaves and differentiate drop from bottom rot and gray mold.

Causal agent (For a description of *Sclerotinia sclerotiorum*, see Bean, white mold, 15B.9.) Sclerotia of *Sclerotinia minor* are 2 to 3 mm across, always external and embedded in mycelium. They form all over the colony and are often aggregated into crusts or flat masses. One to several flat to slightly concave, disk-like, stalked apothecia may arise from a single sclerotium. The stipe is 3 to 30 mm in length and bears the apothecium, or fruiting body, that becomes convex or funnel shaped with age. It is a fleshy, pale, beige or yellow-brown structure, measuring 3 to 5 mm in diameter. The upper surface, the hyménium, is an area of densely packed cylindrical asci, each containing eight ascospores. The ascospores are unicellular, elliptical-obovoid, hyaline and biguttulate, measuring 8 to 17 by 5 to 7 pm (length:width ratio about 2).

Both fungi grow readily on a variety of agar media and produce typical sclerotia in culture. These usually produce abundant apothecia when placed on damp sand or floated on water in diffuse light at about 25°C.

Disease cycle (see Bean, white mold, 15B.9) Sclerotia form on infected plants and fall to the soil when the host tissue disintegrates. Under prolonged moist conditions, such as beneath a leaf on the soil surface, germination of sclerotia is myceliogenic, particularly in *S. minor.* This mycelium can infect lettuce plants directly.

Management

Monitoring — White mold may occur in chicory stored for forcing (*77.9f*). Before storage, roots should be carefully inspected for any sign of infection; any roots with symptoms should be rejected.

Cultural practices — A four- to five-year rotation with com, cereals or forage grasses is recommended if the disease has been severe. Where it has not been severe, shorter rotations with onion and potato can be used. Because many vegetable crops, weeds and trash piles are sources of inoculum, growers should rogue and remove infected plants to reduce inoculum for succeeding crops. This is not as effective for *S. sclerotiorum* because its ascospores commonly are blown from nearby fields and waste areas. Weeds among the crop may create a canopy conducive to infection. Crop rows should be oriented parallel to the prevailing wind with generous spacing in and between the rows, allowing plants to dry quickly after rain. Weeds and volunteer host plants should be controlled at all times and trash piles should be removed and buried deeply or burned. Control is best achieved by crop rotation. At least three or four years of cereals are needed to reduce appreciably the number of sclerotia by biological attrition. Flooding the soil between crops also helps to control the disease. If the disease appears in chicory forcing beds, the presence of sclerotia makes it necessary to sterilize the soil by steam or fumigation. Overhead irrigation should be avoided where drop is a potential problem.

Resistant cultivars — No resistant cultivars of lettuce, chicory or endive are currently available, but those with a more open growth are less susceptible than those with a dense canopy where water is slow to evaporate.

Biological control — Sclerotia are damaged by fly larvae and parasitized by a number of other fungi. There has been limited success in biological control, but not on a commercial scale.

Chemical control — Soil fumigation reduces the inoculum, but it is probably economic only in light soils where other pathogens or weeds will also be controlled.

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.10 Gray mold *Figs. 11.10a-f*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

The fungus that causes gray mold is a widespread saprophyte on dead and dying plant material. Under cool, humid conditions, it readily invades wounds and soft tissues and can do considerable damage. It usually infects tissues that are damaged by other agents, such as frost, insects, rough handling, guttation (salt damage on leaf points exuding water drops) and improper fertilization. On field-grown lettuce, gray mold is most serious in early spring and late fall. In the greenhouse, gray mold is often said to be a disease of bad management. In retail stores, the fungus can spread from plant to plant, a condition called nesting. Gray mold is one of the most prevalent and damaging market diseases of lettuce, chicory and endive.

This fungus has hundreds of hosts, ranging from forest seedlings to virtually all vegetables and ornamentals as well as weeds. It is a common pathogen of lettuce, chicory and endive (see also Asparagus, botrytis blight, 4.1).

Symptoms Gray mold often infects greenhouse-grown seedlings following attack by other damping-off organisms. Affected seedlings usually fail to produce a marketable head. The fungus also may cause a root rot *(11.10e),* or more commonly a head rot that often destroys the inner leaves (*11.10a,b)* before any external symptoms appear, most frequently in fields planted in early spring under cool, wet conditions. In storage, chicory roots can develop infection anywhere but especially in damaged tissues. Roots can rot quickly. Externally, the rot is barely distinguishable in color from the root tissue, but internally the affected tissue is watery and pale brown, though not appreciably softened.

In the greenhouse, gray mold is the most important head rot disease of lettuce and endive. It thrives under humid conditions and cannot be prevented by using soil fumigation or the nutrient film method. On stems and leaves, especially on leaves in contact with the soil, the first sign of infection is a water-soaked area, usually associated with a piece of dead tissue. The lesion dries out and becomes pale gray to beige-colored *(11.10c).* Under humid conditions, a gray-brown mass of thread-like conidiophores forms and a dry mass of conidia *(11.10f)* disperses in a cloud when disturbed. In fleshy tissues, resistant resting sclerotial bodies are formed *(11.10d).* The sclerotia are black, hard and flat or somewhat rounded, and measure 2 to 5 mm in diameter. Under very humid conditions, sporulation is sparse. Instead, there is copious growth of a dirty white cottony mycelium that is sometimes confused with white mold.

Gray mold is more severe in packing sheds and retail outlets where sources of ethylene, such as from ripening tomatoes or apples, are present. It can cause severe damage in chicory roots stored for forcing, particularly in roots that are wet, dirty and already latently infected from the field. In contrast to phoma rot, there is no sharp line demarcating gray mold rot from the rest of the root.

Causal agent The taxonomy of *Botrytis* and *Botryotinia* is complicated. Not all *Botrytis* fungi of the *cinerea* type necessarily have *Botryotinia fuckeliana* as the teleomorph, or indeed any teleomorph. The type species of *Botrytis* is *B. convoluta* Whetzel & Deighton from iris. It has conidia of the *cinerea* type but the sclerotia are distinctive by being convoluted. Its teleomorph is *Botryotinia convoluta* (Drayton) Whetzel.

Botryotinia fuckeliana is rarely seen in nature. It consists of an apotheci- um, a tiny, fleshy, funnel-shaped, brown structure that is about 1 to 5 mm in diameter and is borne on a slender stalk 2 to 20 mm long arising from a Sclerotium. The top surface of the apothecium bears large numbers of asci, each containing eight hyaline, unicellular ascospores that are ejected violently into the air. The ascospores measure 8.5 to 10 by 3.5 to 4 µm. Apothecia can be obtained in culture by mating isolates of *Botrytis cinerea*.

There are a number of anamorphs of *Botryotinia fuckeliana* by which, collectively, isolates can be identified. The conidial state is referable to the form-genus *Botrytis* Pers., the microconidial apparatus is referable to *Myrioconium* Syd., sclerotia are referred to *Sclerotium* Tode, and the organs of attachment, which are relatively large and complex structures, also are characteristic of *Botrytis.* The generally recognized form-species is *Botrytis cinerea.* Since the teleomorph of *B. cinerea sensu stricto* is rare, most isolates identified as *B. cinerea* are more accurately designated as *Botrytis* of the *cinerea* type.

The conidiophores are tall, stout, dark below and paler near the apex, irregularly branched and 1 to 2 mm or more in length. Near the apex, a number of short, septate, sporogenous branches are produced, each with a terminal ampulla on which the conidia develop synchronously on short, fine denticles. At intervals along the conidiophore, botryose clusters of conidia are formed from short side branches that bear sporogenous cells, giving the appearance of nodal areas of sporulation.

The conidia are hyaline or slightly pigmented, gray to brown in mass, ellipsoid-obovoid with an inconspicuous denticle of attachment. They measure 10 to 13 by 6 to 10 µm. They are produced in abundance on infected tissues under humid conditions.

The sclerotium is a flat, convex or loaf-shaped, hard black body, 2 to 5 mm, usually formed just below the host cuticle, eventually erumpent, and falling onto the soil upon disintegration of the host tissue. It may persist in dry soil for several months or years. Germination is usually conidiogenic, but may also be myceliogenic and cause direct tissue infection. Sclerotia rarely give rise to apothecia.

Microconidia are often formed in sporodochia in culture. They are hyaline, spherical, 2 to 3 µm, and are formed in chains from phialides that are single and inside old hyphae or in penicillate clusters. They are not infective and serve only for spermatization.

Botrytis cinerea grows readily on a wide variety of artificial media. It sporulates best on low-nutrient agar in near-uv light. On rich media It forms abundant sclerotia, randomly scattered over the culture.

Disease cycle The gray mold fungus is common on dead and dying plant parts, especially under moist conditions. Initiation of an epidemic usually occurs from air-borne conidia that can infect soft tissues, wounds and blossoms whenever moisture is available. Sclerotia may also be a significant source of inoculum, especially in lettuce that grows close to the soil and traps moisture under and around the leaves. Sclerotia may persist in dry soil for several months or years. The fungus can sporulate on infected tissue within a few days of infection, so secondary spread can be rapid. The conidia land on a wet surface and germinate, the germ tube penetrating the cuticle in five to eight hours at the optimum temperature of 15 to 20°C. Infection is considerably enhanced if the spores alight on dead or dying tissue. The fungus rapidly invades such tissue, utilizing the nutrients released. Under these conditions, the fungus has considerable infective potential, quickly breaking down any resistance. Thus, infections are very common on senescent cotyledons and freshly damaged leaves.

Once inside the tissue, the fungus advances rapidly, producing a soft rot by dissolving the pectic materials. At this stage, it is also highly infective to healthy tissue in contact with its cottony mycelium. Pockets of infection (nesting) are often seen in packed produce and on chicory roots stored for forcing. When infected tissue has been fully colonized by the fungus, sclerotia are formed which, either immediately or after a period of dormancy, produce copious conidia. Sclerotia are the main source of conidia to maintain the epidemic. Conidia are also formed directly at the surface of affected tissue.

Management

Cultural practices — Crops are seldom affected if grown in well-drained soil and in well-ventilated sites. In field sites with cold, wet soils and protected from drying winds, rows are best oriented parallel to the prevailing wind with the rows and plants spaced adequately to give as much ventilation as possible. Susceptibility may be reduced by decreasing nitrogen and increasing calcium levels in the crop. Trash piles of any plant material should be eliminated because they are potential sources of inoculum and sclerotia. In chicory forcing beds, correct fertilizer balance and adequate air movement should be achieved. On cool clear nights after warm humid days, the moist air should be ventilated from the forcing house and sufficient heat applied at night to prevent dew formation. Watering should be done when the plants will dry quickly. Chicory roots are especially prone to

mechanical damage, but all roots for forcing should be lifted very carefully. Any damaged tissue is vulnerable to phoma rot as well as to gray mold.

Chemical control — Fungicides should be used with caution because the gray mold pathogen is often quick to develop fungicide-tolerant races; fungicides then only serve to suppress natural competitors, often making the disease more severe.

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.11 Phoma rot

Phoma exigua var. *exigua* Desmaz.

This fungal disease is occasionally seen on chicory in storage. The pathogen attacks only damaged roots.

Symptoms The rot may appear anywhere on the root. It is brown and wrinkled in external appearance with a sharp line of demarcation from healthy tissue. The root becomes rubbery.

Causal agent *Phoma exigua* var. *exigua* must be isolated for confirmation. The fungus grows on oatmeal and malt extract agars; on 2% malt agar it is characteristically zonate. This distinguishes it from the closely related *P. exigua* var. *foveata,* which additionally forms anthraquinone pigments that turn red on exposure to ammonia vapor. It forms globose, thin-walled, dark brown to black, immersed pycnidia containing ampulli- form, hyaline phialides with hyaline, cylindrical, aseptate conidia. The pycnidia measure 90 to 200 pm, and the conidia 4 to 5 by 2 to 3 pm. In the rotted tissue, the pathogen forms only septate mycelium with no pycnidia.

In contrast to gray mold, phoma rot does not spread from root to root.

Disease cycle Chicory roots are easily damaged by mechanical handling. The pathogen can attack them at temperatures between 0 and 10°C, and at relative humidities of 95 to 97%.

Management (see gray mold, 11.10)

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(Original by W.R. Jarvis)

▶ 11.12 Powdery mildew *Fig. 23.10*

Erysiphe cichoracearum DC.

Powdery mildew first became a problem on cultivated field lettuce in California in 1951, presumably as a result of mutation from the previously known wild lettuce strain of *Erysiphe cichoracaerum.* The disease rarely occurs in the field in Canada, but it has been observed in hydroponic greenhouses (see Greenhouse lettuce, powdery mildew, 23.10). The cultivated lettuce strain can infect a wide range of hosts in the laboratory, but in the field only cultivated lettuce is a significant source of inoculum.

Symptoms White, powdery accumulations of mycelium and conidia appear in spots on both the upper and lower surfaces of older leaves *(23.10).* As the spots enlarge, leaves lose their bright color and fade to yellow and finally brown. Tiny, black, spherical cleistothecia may appear on mature lesions. Severely infected heads may be stunted and unmarketable. Late-infected heads may be salvaged by removing the outer leaves.

Causal agent *Erysiphe cichoracearum* has a well-developed mycelium. Conidia occur in long chains, are ellipsoid to barrel shaped, and measure 25 to 45 by 14 to 26 pm. Cleistothecia are globose or irregular, 90 to 135 µm in diameter with numerous, rarely branched appendages, one to four times as long as the diameter of the cleistothecium. Asci number 10 to 25 and measure 60 to 90 by 25 to 50 µm. Ascospores number two, rarely three, and measure 20 to 30 by 12 to 18 µm.

Powdery mildew can be distinguished from downy mildew because the pathogen sporulates on both leaf surfaces with conidia in chains, whereas downy mildew sporulates primarily on the leaf undersurface with sporangia attached singly to tree-like sporangiophores. Powdery mildew caused by *Erysiphe* has conidia that germinate by a simple germ tube terminating in an appressorium.

Disease cycle Initial infection is by ascospores released from cleistothecia in residues from previous crops. Subsequent spread during the growing season, both within and between fields, in by conidia. Conidia are released in clumps of two or three under

dry, windy conditions and may be carried up to 200 km by wind. Conidia germinate and infect most rapidly between 18 and 25°C at a relative himidity of 95 to 98%. Some germination occurs at very low humidity, but none occurs at 100% relative humidity. The minor importance of the disease on field lettuce in Canada can probably be attributed to below-optimum temperatures at night even when the relative humidity is optimal for disease development. In the greenhouse, optimal temperature and humidity conditions are more likely to coincide.

Management

Cultural practices — Sanitation and elimination of trash piles in and around the greenhouse, and prompt incorporation of crop residues in the field reduce the likelihood of spread to subsequent crops.

Resistant cultivars — The use of resistant cultivars should be investigated where cultural practices are not sufficient.

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(Original by D.J. Ormrod)

▶ 11.13 Rust *Figs. 11.13a,b*

Puccinia dioicae Magnus (syn. *Puccinia extensicola* Plowr.)
Puccinia hieracii f. sp. *cichoriae* (Bellynck & J. Kickx fil.) Boerema & Verhoeven (syn. *Puccinia patruelis* Arthur)

Puccinia is one of the most economically damaging genera of rust fungi worldwide. It has been recorded frequently in commercial lettuce but is rarely damaging. Rust of chicory and endive has rarely been recorded. *Puccinia dioicae* occurs on lettuce. *Puccinia hieracii* occurs on chicory, endive and on *Hieracium* and other Compositae, but only as host-specific races.

Symptoms On lettuce, groups of 50 to 200 whitish-yellow aecia appear as powdery yellow masses about 1.5 cm across on the lower surface of outer leaves (*11.13a,b*). The corresponding area of the upper leaf surface appears as a large yellow spot.

Causal agent *Puccinia diociae* is macrocyclic and heteroecious, producing spermagonia and aecia on lettuce and uredinia and telia on sedges *(Carex* spp.). Aeciospores are globoid, 12 to 21 µm in diameter, with colorless, finely verrucose walls. Uredinia are light cinnamon-brown. Urediniospores are globoid to obovoid, 12 to 20 by 16 to 26 µm, with cinnamon-brown, echinulate walls containing two pores in the upper part. Tellia are dark chocolate-brown to black. Teliospores are clavate-oblong,

12 to 22 by 32 to 50 µm, rounded, truncate above, narrowed below, slightly constricted, with chesnut-brown walls. Other species of *Puccinia* occur occasionally on lettuce and commonly on sedges.

Puccinia hieracii f. sp. *cichoriae* is autoecious. Spermagonia are yellow. Urediosori are cinnamon, tiny, and form on small pale spots. Urediniospores are globose to ellipsoid, echinulate, yellow-brown, and measure 24 to 29 by 16 to 25 µm, with two germ pores. Telia are similar to the uredinia but somewhat darker brown. Teliospores are ellipsoid-ovate, rounded, scarcely constricted, delicately verrucose, brown and measure 25 to 40 by 16 to 24 µm.

Disease cycle Rust diseases of lettuce generally occur on sedges in the uredinial and telial stages. Basidiospores carry the disease to lettuce wherre spermagonial and aecial stages occur. Aeciospores carry the disease back to sedges. The chicory/endive rust pathogen has no alternate host. Small clusters of spermagonia appear on the leaves, together with uredinia and telia.

Management

Cultural practices — When rust occurs in field lettuce, eradication of sedges within 100 metres is usually sufficient to protect the crop. In trials in Alberta, lettuce seeded in the fall exhibited 100% infection the following spring, compared to less than 1% infection in the same cultivars seeded in the spring.

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.14 Septoria leaf spot

Septoria lactucae Pass.

Leaf spot was first reported on lettuce in Quebec in 1941. It has remained a minor fungal disease and has not been reported west of Manitoba. Lettuce, other vegetable crops and several weeds are affected.

Symptoms Small, irregular, chlorotic spots first appear on the outer leaves. They enlarge and change from yellow to olive-brown. Older lesions may have a chlorotic margin.

This disease is readily distinguished from anthracnose by the larger, more irregular spots that usually have numerous, pin-point, black pycnidia in the center. Microscopic examination should be used to confirm differences in conidial morphology.

Causal agent *Septoria lactucae* pycnidia are chiefly epiphyllous, immersed, becoming erumpent, and measure 100 to 200 µm in diameter. Conidiophores line the inside of the pycnidium. Conidia are hyaline, straight or curved, have one to three septa, and measure 25 to 40 by 1.5 to 2 µm. The fungus can readily be isolated from cirrhi of conidia that extrude from the pycnidia in humid conditions.

Disease cycle Pycnidia are carried on seed, on residues from infected crops, and on weed hosts. Conidia germinate and are infective above 12°C in the presence of free water or at humidity over 90% for 24 hours. Pycnidia and conidia are produced within five days, making rapid disease build-up possible.

Management

Cultural practices — In addition to rotations with non-susceptible crops for at least one year in fields where the disease has occurred, and elimination of wild lettuce from the vicinity of propagation areas and fields, growers should use disease-free, desert-grown seed if possible. Seed can be disinfested by hot-water treatment at 48°C for 30 minutes, but germination may be reduced. Infected crop residues should be disked under promptly to facilitate breakdown and to prevent spores from being transported on clothing and equipment.

Growers should avoid seeding adjacent to infected plantings or in areas with poor air and soil drainage, and avoid working in infected crops when they are wet.

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(Original by D.J. Ormrod)

VIRAL AND VIRAL-LIKE DISEASES

▶ 11.15 Asteryellows *Figs. 11.15a, b*

Aster yellows mycoplasma-like organism

Aster yellows was first reported in Canada in 1930. It now occurs regularly wherever the aster leafhopper is common. Crop losses of up to 100% in lettuce have been reported in Ontario. Though the disease is not seed-borne, it interferes with flower and seed formation. Aster yellows affects more than 300 different plants in 48 families.

Symptoms In lettuce, the center leaves show symptoms first *(11.15a).* They appear chlorotic and fail to develop normally, remaining as short stubs, or they may be twisted and curled, exuding pink to brown latex when straightened. Light brown latex deposits on the underside of the midribs are diagnostic. Plants infected early will develop an overall yellowing of the outer leaves *(11.15b).* If the plant was infected at a very early stage, it will be stunted and have twisted yellow leaves. Plants infected at later stages of growth will be very pale internally and there will be evidence of tipburn. Symptoms usually are most severe in July and August.

The percentage of plants infected with aster yellows in a field can vary from 0 to 100%, depending upon the prevalence of leafhoppers carrying the pathogen. Lettuce is most vulnerable when the plants are in the seedling stage and until they are about three-quarters grown. Excessive heat and drought adversely affect leafhopper development and survival, and may reduce the incidence of aster yellows. Conversely, heavy rainfall may make plants more succulent and attractive to leafhoppers.

Causal agent The pathogen is a prokaryote that is enclosed by a membrane but lacks a true cell wall. Cells contain DNA and ribosomes but no membrane-enclosed inclusions. They measure 0.3 to 0.8 µm, and are irregular in shape, from globose to cylindrical. The pathogen inhabits the phloem tissues of infected plants and is transmitted from plant to plant by phloem-feeding leafhoppers in which it multiplies. Aster yellows symptoms on lettuce differ from mosaics caused by viruses; younger leaves are affected first and the growing point is often malformed. Transmission studies and electron microscopy can be used to confirm visual diagnosis. Advanced techniques such as complementary DNA hybridization are also available, should greater precision be necessary.

Disease cycle The pathogen can overwinter in susceptible grains, perennial weeds and ornamentals. Several leafhopper species can acquire the pathogen and transmit it to lettuce and other susceptible crops.

Management

Cultural practices — Forage legumes grown close to susceptible vegetable crops should be sprayed with a registered insecticide before being cut for hay or seed (see aster leafhopper, 11.23). In areas where leafhoppers are abundant and aster yellows occurs regularly, it is important to control perennial host plants within the crop and around the field margins.

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(Original by D.J. Ormrod and M. Valk)

▶ 11.16 Big vein *Fig. 11.16*

Lettuce big-vein virus

Lettuce big vein is caused by a virus that is vectored by a soil-borne fungus. In Canada, the disease was first reported in Ontario in 1940. It is common in lettuce grown during cool weather in infested soil, but it is not highly destructive to the crop. The fungal vector has a very wide host range, infecting the roots of many plants.

Symptoms The most distinctive symptom is vein clearing of the tissue adjacent to the leaf veins, making them appear wider than normal *(11.16).* During the cooler part of the growing season, infected leaves have enlarged veins that are translucent when held up to the light. Leaves also may appear puffy and ruffled at the margins. Plants infected in the early seedling stage may die or remain stunted.

Causal agent The pathogen has been isolated and characterized as having labile rod-shaped particles, measuring 324 by 18 nm and 152 by 18 nm. Serology suggests a close relationship of the big-vein virus with tobacco stunt virus.

Disease cycle The pathogen can be carried in the soil for at least eight years in resting sporangia of the oomycete fungus *Olpidium brassicae* (Woronin) P.A. Dang. Cool, wet soil conditions favor the fungus vector. In the presence of a susceptible host, the fungal sporangia germinate to produce zoospores that carry the big vein pathogen into the roots. The uninfected fungus acquires the pathogen upon entry into the roots of infected lettuce, and it releases infected zoospores into the soil water or forms resting sporangia in old roots remaining in the soil after harvest.

Management

Cultural practices — Seedbeds and greenhouse soils can be steamed or fumigated to lower the vector and pathogen populations. Growers should avoid early planting in poorly drained fields with a history of big vein.

Resistant cultivars — Lettuce cultivars differ in tolerance to big vein.

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(Original by D.J. Ormrod)

▶ 11.17 Lettuce mosaic *Figs. 11.17a, b; 23.14*

Lettuce mosaic virus

Lettuce mosaic is a widely distributed, seed-borne virus disease. Until recently, up to 5% seed infection was not unusual. Besides lettuce and endive, cultivated hosts such as sweet pea, green pea and a number of flowers are sources of inoculum. In addition, perennial and biennial weeds, such as groundsel (*Senecio* spp.) and sow thistle *(Sonchus* spp.), also can serve as sources of inoculum for subsequent crops.

Symptoms On lettuce, curled-leaf endive and broadleaved endive, lettuce mosaic causes chlorotic or yellow spotting and a reduction in size *(11.17a).* These symptoms may resemble those of turnip mosaic. Some pathotypes are more severe, causing marked yellowing, distortion and stunting. Plants grown from infected seed or those infected in the early stages of growth are

stunted and have pale green to yellow mottled leaves *(11.17b, 23.14).* Leaf margins are ruffled and outer leaves may die. In older plants, mottling may be indistinct, but a uniform, dull gray-green color is characteristic.

Causal agent Lettuce mosaic virus is a flexuous rod in the potyvirus group, measuring 746 by 22 nm. Besides being aphid-borne, it is seed- borne and sap-transmitted. In aphids, it is stylet-mediated and non-persistent. Diagnosis is based on symptoms on indicator plants such as quinoa *(Chenopodium quinoa* Willd.) and globe amaranth (*Gomphrena globosa* L.). On quinoa, lettuce mosaic causes local lesions and mosaic symptoms. Systemic reaction of young leaves may be equivocal. ELISA tests are useful for confirmation. Commercial diagnostic reagents are available. Several other viruses cause similar symptoms on lettuce but they are not as widespread or common.

Disease cycle Seed-borne inoculum is the most important source of lettuce mosaic, but the virus is also transmitted by aphids, particularly the green peach and potato aphids. These aphids acquire the virus by feeding on infected plants. They then infect other plants in the field or in other fields, as well as susceptible weeds in the vicinity. The speed and extent of spread after seedling emergence is proportional to aphid population levels and their movement.

Management

Cultural practices — With the use of virus indexing, which can detect one infected seed in as many as 30 000 seeds, disease incidence has declined markedly. Growers are still advised to plow down crop refuse immediately after harvest, to eliminate host weeds adjacent to lettuce fields, to destroy overwintered broadleaved weeds, and to control aphids (see aphids, 11.24). In climates that permit year-round lettuce production, a lettuce-free period has been used as a control measure. This is only useful if there are no other infected host plants in the vicinity. In Canada, winter serves as the lettuce-free period. Seeding new plantings adjacent to old ones should be avoided.

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(Original by D.J. Ormrod and W.R. Jarvis)

▶ 11.18 Other viral diseases *Fig. 23.15*

Artichoke Italian latent virus
Beet mild yellowing virus
Beet western yellows virus
Chicory yellow mottle virus
Cucumber mosaic virus
Lettuce infectious yellows virus
Tomato spotted wilt virus

Numerous other viruses can infect lettuce. The most damaging are those causing beet western yellows, lettuce infectious yellows, cucumber mosaic, and tomato spotted wilt. Endive is host to beet western yellows, beet mild yellowing, which is probably a closely related strain of beet western yellows, chicory yellow mottle, and artichoke Italian latent viruses. Beet western yellows affects vegetable crops such as sugar beet, rutabaga, turnip, spinach and lettuce, and weeds. Chicory yellow mottle also affects parsley. Cucumber mosaic virus has a very wide host range. Lettuce infectious yellows virus also infects other vegetables, ornamentals and weeds. Tomato spotted wilt virus has a wide host range, including greenhouse tomato, pepper and ornamentals.

Symptoms Beet western yellows, cucumber mosaic, and lettuce infectious yellows cause yellowing, particularly of older leaves, and stunting if plants are infected early. On lettuce, the symptoms resemble those of lettuce mosaic and physiological disorders such as magnesium deficiency. Lettuce infectious yellows virus is semi-persistent in the whitefly vector. In plants affected by beet western yellows, the older leaves tend to yellow, but the veins remain green. Tomato spotted wilt virus causes more severe symptoms *(23.15),* which include numerous tiny necrotic spots, petiole curvature and brown streaks on the underside of the leaf midribs. It can be acquired by thrips larvae and then transmitted when they reach the adult stage.

In endive, artichoke Italian latent virus produces a generalized yellowing and stunting; symptoms of beet western yellows and beet mild yellowing are often very mild, sometimes resembling deficiency symptoms, or they are absent; and, chicory yellow mottle virus causes a bright yellow mottle with a ringspot and line pattern.

Causal agents Many viruses cause yellowing symptoms. Laboratory tests may be needed to identify the virus or combination of viruses present in a particular diseased plant.

Artichoke Italian latent virus is a RNA-containing nepovirus with isometric particles about 30 nm in diameter. In soil, it is transmitted by the nematode *Longidorus apulus* Lamberti & Bleve-Zacheo and is native to southern Italy and Bulgaria. Indexing

is by mechanical inoculation to tobacco or globe amaranth, *Gomphrena globosa* L., both of which develop necrotic, whitish, ring-like local lesions.

Beet mild yellowing virus is a luteovirus serologically related to beet western yellows virus but differs in particle morphology and by its persistence in its aphid vector, usually the green peach aphid or the black bean aphid. Its particles are isometric, about 26 nm in diameter. The virus produces characteristic reddening on Miner's lettuce (*Montia perfoliata* (Donn.:Willd.) Howell) 15 to 25 days after transmission by the green peach aphid. Weed hosts include shepherd's-purse (*Capsella bursa-pas- loris* (L.) Medic.) and groundsel (*Senecio vulgaris* L.).

Beet western yellows virus is an isometric particle in the luteovirus group, measuring about 26 nm in diameter. It is persistent and aphid- borne. Indexing can be done as described for beet mild yellowing virus.

Chicory yellow mottle virus has angular, isometric RNA-containing particles about 30 nm in diameter. It is readily sap-transmitted to the diagnostic species *Phaseolus vulgaris* L., *Cucurbita pepo* L. and other species, but it is found naturally only in chicory and parsley.

Cucumber mosaic virus is an isometric virus in the cucumovirus group, measuring about 30 nm in diameter. It is transmitted mechanically and by numerous aphids in a non-persistent manner.

Lettuce infectious yellows virus, similar to the closteroviruses, has long flexuous rods measuring 13 to 14 by 1800 to 2000 nm.

Tomato spotted wilt virus is an isometric particle measuring 70 to 90 nm in diameter.

Disease cycle Beet western yellows virus is readily transmitted by aphids. The green peach aphid is its most important vector. Lettuce infectious yellows virus is transmitted by the sweetpotato whitefly (see Foreign diseases and pests, 3.10). Infection of greenhouse-grown lettuce crops or transplants may occur if infectious sweetpotato whiteflies are present on ornamentals in the same greenhouse. Tomato spotted wilt virus is transmitted by thrips, particularly the western flower thrips in greenhouses, and the onion thrips outdoors. Cucumber mosaic virus is transmitted by the green peach aphid and other aphids. Artichoke Italian latent virus has two serological variants, both of which are transmitted by nematodes *(Longidorus* spp.). A vector for chicory yellow mottle virus is unknown.

Management

Cultural practices — Growers are advised to keep ditches and roadways free of weed hosts and, in field production, to plow crop residues promptly after harvest. Lettuce or lettuce transplants should not be grown in the same greenhouse as vegetables or ornamentals that harbor viruses, whiteflies, thrips or aphids (see Insect pests, 11.23-11.26, for their control).

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(Original by D.J. Ormrod and W.R. Jarvis)

NON-INFECTIOUS DISEASES

▶ 11.19 Nutrient disorders *Figs. 11.19a-c; 23.16*

Manganese deficiency
Manganese toxicity
Tipburn

Manganese deficiency commonly occurs in lettuce crops grown in muck soils above pH 6.5. Muck soils underlain with calcareous subsoils are also susceptible, as are areas within fields where mineral ridges surface through degrading muck soils. Plants are stunted and have a yellow-gray appearance. Necrotic spots occur on leaf margins (*11.19a).* Midribs may be hollow. Manganese deficiency can be corrected by lowering the soil pH or by applying manganese sulphate to the soil or foliage. Maneb fungicide is a source of manganese when applied to the foliage for downy mildew control.

Manganese toxicity symptoms on lettuce are irregular yellow margins on the older leaves, sharply contrasting with the rest of the leaf that stays green (*11.19b).* Care must be taken when applying manganese sulphate because manganese can be present at toxic levels in soils with a pH below 6.0 and it can damage the leaves when applied in excess.

Tipburn occurs on the inner leaves of head-forming vegetables such as lettuce, cabbage and Brussels sprouts. It is a result of calcium deficiency in the growing tissues of the inner leaves. The first symptoms are necrotic spots near the leaf tips that expand until the entire edge of the leaf is brown *(11.19c, 23.16).* Injured tissues may become affected by bacterial soft rot (see head rot, 11.1). Tipburn is second only to bruise injury as the most commonly reported non-infectious disorder of head lettuce. Many inter-related factors contribute to calcium uptake and tipburn. The condition can be reduced to some extent by soil calcium levels that are high relative to competing elements such as potassium and magnesium, by reducing nitrogen applications to limit growth, especially during warm weather, by harvesting slightly before maturity and, in the case of greenhouse crops, by keeping the nighttime humidity high. Cultivars differ in tolerance to tipburn.

▶ 11.20 Other disorders *Fig. 11.20*

Non-infectious corky root
Pink rib
Russet spot

Non-infectious corky root

is a disease of lettuce characterized by dark brown lesions and corky ridges on the taproot, rotting-off of side roots, and red or yellow-brown discoloration of the stele. These symptoms are induced by ammonia and possibly nitrite released from nitrogenous fertilizers. This condition contrasts with infectious corky root, which is not induced by high nitrogen but by a fastidious bacterium, *Rhizomonas suberifaciens,* and which has different symptoms (see infectious corky root, 11.2).

In experimental studies of non-infectious corky root, external reddish discoloration and corkiness were induced by 160 kg of nitrogen per hectare when both urea and ammonium nitrate were applied, and by 350 kg or more of nitrogen per hectare when only ammonium nitrate was used. Nitrogen toxicity seems to be related more to ammonium nitrogen in the tissue rather than nitrate nitrogen.

The use of ammonium- and nitrite-releasing fertilizers should be avoided. Nitrate fertilizers are relatively safe in the absence of *R. suberifaciens* but infectious corky root increases in severity with increasing nitrogen applied as ammonium sulfate, ammonium nitrate, urea, or calcium nitrate.

Pink rib

first appears as a pink discoloration at the base of the midveins of lettuce leaves *(11.20).* The discoloration extends throughout the veins of the outer leaves, then progresses into the younger leaves. The cause of pink rib is unknown. It is aggravated by bruising, tight packing and high storage temperature.

Russet spot

is a common disorder of head lettuce. It first appears as small tan-colored pits on the outer leaves *(11.20).* The pits may be clustered along the midrib or scattered on the blades. Russet spot occurs on mature and overmature heads in the field and on harvested lettuce in storage and transit. The condition is caused primarily by ethylene injury and can be prevented by reducing ethylene concentrations in the storage atmosphere. It is best to avoid storing or transporting lettuce with ripening fruit, to use battery- powered rather than propane-fueled fork lifts in cold storage areas, and to store lettuce separately from fruit in retail holding rooms and home refrigerators.

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(Original by D.J. Ormrod and W.R. Jarvis)

NEMATODE PESTS

▶ 11.21 Northern root-knot nematode *Fig. 7.15b*

Meloidogyne hapla Chitwood

Symptoms include yellowing, prolific branching of rootlets, and production of small, spherical galls on roots. In head lettuce, there is a delay in maturation or lack of head formation. For a complete description and management strategies, see Carrot, northern root-knot nematode, 6.20; see also Management of nematode pests, 3.12.

▶ 11.22 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 11.23 Aster leafhopper *Figs. 11.23a, b*

Macrosteles quadrilineatus (Forbes)
(syn. *Macrosteles fascifrons* of authors, not Stål)

The aster leafhopper is found throughout Canada wherever cereal grains are grown. It is common in the south-central United States and migrates northward on air currents in the spring to invade the northern states and Canada. Thus, southern Canada is subject to an annual spring migration of adults from the south. They usually arrive before eggs of local populations hatch but nymphs have been found in Alberta before immigrant adults appear.

This insect has been recorded on more than 100 plant species in at least 40 families. Although cereals and grasses appear to be preferred hosts, large populations can develop on such vegetable crops as the head, leaf and Boston types of lettuce. The aster leafhopper is a major pest of certain vegetables because it transmits the causal agent of aster yellows, the severity of which varies widely, depending upon location and seasonal conditions. Losses of up to 100% have been reported in lettuce, celery, and carrot. Potato and onion can be affected to a lesser degree.

Damage Symptoms of aster yellows are described in the viral and viral-like diseases of lettuce section. Direct feeding injury by aster leafhopper normally is of little or no economic importance.

Once leafhoppers have acquired the aster yellows mycoplasma-like organism, they remain infective for life. A feeding period of eight hours on infected plants is required for acquisition. After three weeks, they are able to infect other plants. A feeding period of about eight hours is required to transmit the mycoplasma-like organism, which explains why chemical control of aster yellows is usually more effective than that for aphid-transmitted viruses. The level of infectivity in migrant populations from the southern United States is a major factor determining the severity of aster yellows in the midwestern states and Manitoba. Importance of migrants in relation to disease severity in Ontario is less well documented.

Identification The aster leafhopper (family Cicadellidae) adult is about 3 mm long, slender, pale gray-green with six black spots on the front of its head (*11.23a,b*). If disturbed, it immediately moves by short flights to nearby plants. The nymph resembles the adult in shape but it is smaller, tan colored and lacks wings. A dark-winged strain occurs in western Alberta.

The leafhopper known as *Macrosteles fascifrons* (Stål) has been confused with the aster leafhopper in the past but now is considered to be a separate species, which also is migrant into Canada, feeding strictly on certain types of rushes *(Juncus* sp.) (see Additional references, Hamilton 1983).

Life history Populations infesting crops in Canada arise from two sources. The insect overwinters in the egg stage in winter cereals, wild grasses and weeds, but survival of eggs is contingent upon the severity of winter temperatures and the amount of snow cover. Nymphs emerge from early May in southern Ontario to late May or early June in the Holland Marsh area, depending on geographical location, and they complete their development to adults in two to three weeks. However, southern Ontario and the Prairie provinces also are affected by large numbers of adult leafhoppers that migrate northward and westward on warm air currents from the southern United States. These leafhoppers arrive about mid- to late May or June, before local populations have matured, and lay their eggs on winter and spring cereals, grasses, weeds and such early seeded vegetables as lettuce. Hatching occurs in about eight days and new adults appear in about two to three weeks. As the various crops mature, the new adults, which originate from either overwintered eggs or migrants, disperse to more succulent crops, such as lettuce and other vegetables. There are three to five generations per year, depending on geographic location in Canada. In the fall, as annual crops mature, the leafhoppers move to winter cereals where they lay eggs that may survive the winter and give rise to the next spring generation.

Management The primary purpose in controlling the aster leafhopper is to reduce the potential for the spread of disease. Unfortunately, a method to determine the amount of infection in the leafhopper population has not been developed and there are few alternatives to insecticides, which must be applied without any knowledge of disease potential and may not always be economical.

Monitoring — Aster leafhopper can be monitored by means of yellow sticky traps. Growers should start monitoring for adults in early spring. Spraying is suggested as soon as adults are caught on the traps because action and economic thresholds have not been established in Canada. Although monitoring is useful for establishing the presence of the leafhopper and provides some indication of its abundance, a more important factor in the spread of the disease is the proportion of the population that is infective.

Cultural practices — Lettuce fields should be plowed immediately after harvest to remove sources of disease inoculum and to eliminate leafhopper breeding areas. Weeds in headlands and ditches also should be controlled to prevent susceptible species from serving as a reservoir of disease inoculum. The use of a reflective surface, such as aluminum foil, to repel adult leafhoppers has shown some promise.

Chemical control — In Canada, the aster leafhopper has not shown resistance to insecticide products that are registered for use on lettuce. Granular materials can be placed near the seed at seeding time. Foliar sprays can be applied as soon as leafhoppers appear and repeated at five- to seven-day intervals as long as monitoring indicates a need. With four to five sprays, leafhopper numbers can be sufficiently reduced to avoid significant infection. Attempts should be made to reduce leafhopper populations in adjacent susceptible vegetable crops and in headlands and ditch areas. In Quebec, direct damage from leafhoppers and the incidence of aster yellows infection are low in lettuce and treatment usually is not required. In general, treatments on lettuce for other insects also control aster leafhopper.

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(Original by M. Valk and A.B. Stevenson)

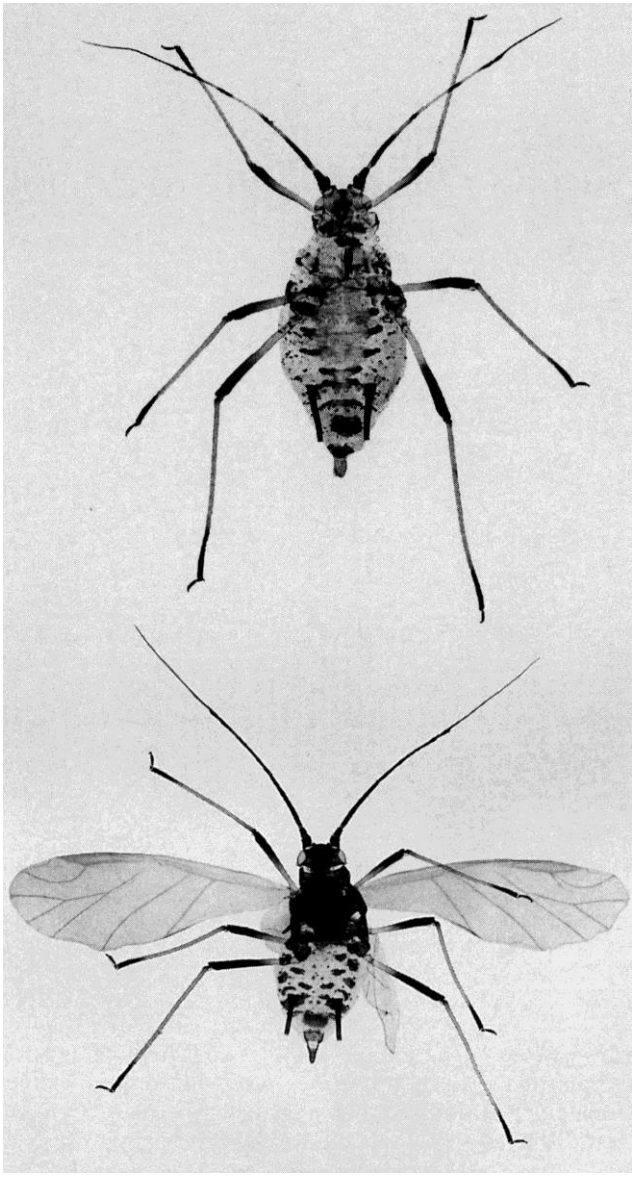
▶ 11.24 Lettuce aphid *Figs. 11.24T1, T2*

Nasonovia ribisnigri (Mosley)

The lettuce aphid is native to Europe. In Canada, it has been reported in British Columbia, Quebec and New Brunswick. It was not a pest in North America until 1981, when it caused significant damage to commercial plantings of head lettuce in southwestern British Columbia. In British Columbia, the head-lettuce market almost collapsed in 1982 because of cosmetic damage caused by this aphid, which remains the major pest of lettuce in that region.

This aphid's primary hosts are *Ribes* spp. Secondary hosts are lettuce and plants in several families.

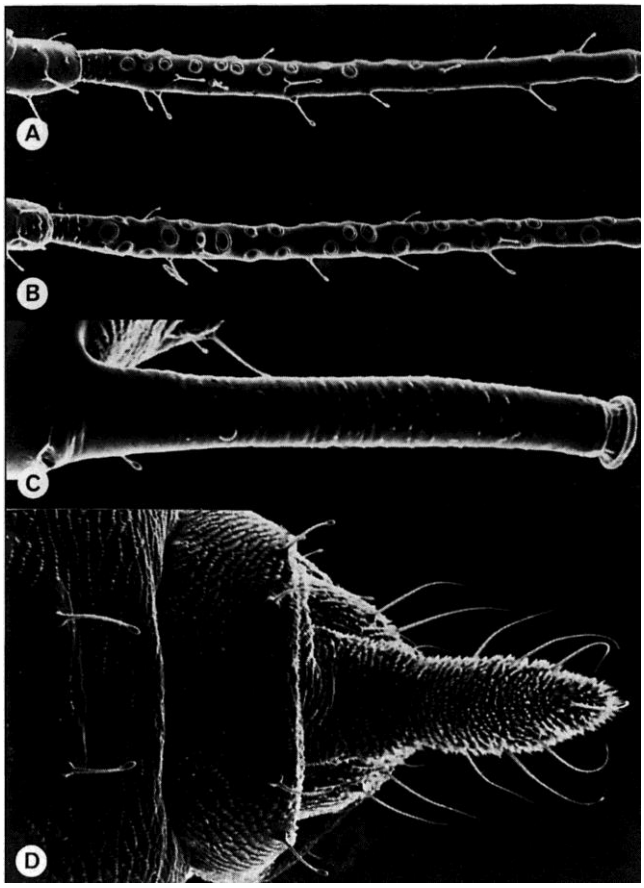
Damage Unlike other lettuce-infesting aphids, this aphid tends to colonize leaves inside the developing heads, causing cosmetic damage and making the head unmarketable. The aphid cannot be killed with foliar contact sprays after it is inside the lettuce head.



11.24T1 Lettuce aphid; photomicrographs of wingless (above) and winged adults, which are usually pale green; length 2-3 mm.

This aphid can transmit cucumber mosaic and possibly beet western yellows. It has not been implicated as a vector of lettuce mosaic.

Identification The adult aphid is 2 to 3 mm in length and olive-green, with a distinctive dorsal pattern, especially in the winged form (*11.24T1*)**.** Antennae are long with sensory organs (secondary sensoria) on the basal part of segment III in wingless forms and along the entire third segment in winged forms. The paired abdominal projections (cornicles) are cylindrical with a distinct, ring-like incision. The tip of the abdomen (cauda) is finger shaped, usually with seven, hair-like setae (*11.24T2).* Various color forms exist, including a pink form.



11.24T2 Lettuce aphid; A,B) scanning electron micrographs of third segment of antenna of wingless (A) and winged (B) adults; C) cornicle; D) tip of abdomen.

Life history The lettuce aphid overwinters as eggs on currant and gooseberry (*Ribes* spp.) and possibly other plants. In British Columbia, eggs hatch in late March and April. Winged aphids migrate into lettuce fields in May and June. There they complete many winged and wingless generations throughout the summer, flying to other lettuce fields and starting new colonies. In October, winged aphids return to primary hosts, mate, and lay eggs.

Management

Monitoring — Commencing about three weeks after seeding, lettuce fields should be checked for aphids twice weekly before heading begins, by walking along the outside beds and examining four heads at 20-pace intervals in each bed. The process of examination entails stripping the plants and inspecting each leaf for aphids. If a single aphid is found, a spray program should begin immediately. After heading, sampling is too time consuming and can be abandoned where growers are on a strict routine of chemical control. Because of a zero tolerance level for this aphid on lettuce in British Columbia, monitoring usually is used to ensure that chemical control programs are efficient.

Cultural practices — To prevent aphids from spreading to other lettuce fields, growers should destroy and bury all crop residue after harvest. The removal of *Ribes* plants over a wide area has reduced the problem.

Resistant cultivars — See Helden *et al.* (1993) in Selected references.

Chemical control — In southwestern British Columbia, this aphid can be controlled on head lettuce only by routine application of insecticides. No other procedure ensures that the field will be free of aphids at harvest. After thinning, sprays should be applied every 7 to 10 days until just before heading. At early heading, a systemic insecticide is required to eliminate any build-up of aphids inside the head. To guard against reinfestation, additional sprays are required on a weekly basis from heading until the prescribed pre-harvest interval.

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(Original by R.S. Vernon and J.R. Mackenzie)

▶ 11.25 Other aphids

Aphids other than the lettuce aphid occasionally infest lettuce. Their presence and their molted skins (exuviae) between the leaves of the head may reduce the attractiveness and marketability of the crop. They are also potential vectors of plant pathogenic viruses. Such infestations may be difficult to control because the aphids are protected from insecticidal applications by the outer leaves. Undetermined species of root aphids have been reported attacking head lettuce and stunting plant growth in the Holland Marsh area of Ontario. They are common in New Brunswick and Nova Scotia, and also are found in Quebec.

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(Original by A.B. Stevenson)

▶ 11.26 Other insect pests *Figs. 11.26; 8.40; 7.21*

Cabbage looper *Trichoplusia ni* (Hübner)
Cutworms
Tarnished plant bug *Lygus lineolaris* (Palisot de Beauvois)

Cabbage looper

(see Crucifers) (*8.40b-f)* Cabbage looper larvae occasionally attack lettuce, chewing holes in the leaves similar to those they chew on cabbage.

Cutworms

(see Carrot, 6.25; and Tomato, 18.35) (*11.26*)

Tarnished plant bug

(see Celery) *(7.21b,d,e)* The tarnished plant bug occasionally attacks the heads of lettuce, causing small holes with brown margins in the leaves. Damage can be sufficiently severe to make the heads unmarketable.

(Original by A.B. Stevenson)

OTHER PESTS

▶ 11.27 Slugs and snails *Figs. 11.27a-c; 3.11e*

Black slug *Arion ater* (L.)
Brown garden snail *Helix aspersa* Müller
Gray garden slug *Deroceras reticulatum* (Müller)
Spotted garden slug *Limax maximus* L.

The pest species of slugs and snails in Canada are of European origin. The gray garden slug *(11.27c)* (see Crucifers, 8.49) occurs locally in most urban areas of Canada. The brown garden snail *(3.11e)* (see Introduced diseases and pests, 3.11) occurs in the lower Fraser Valley and southern Vancouver Island of British Columbia. The black slug (*11.27a)* occurs in the same areas of British Columbia, as well as eastern Quebec, Prince Edward Island, the Avalon Peninsula of Newfoundland and in Sussex, New Brunswick, where it was found recently. The spotted garden slug (*11.27b*) occurs in the eastern Avalon Peninsula of Newfoundland and in the vicinity of Vancouver, British Columbia. Recently it was found at Sillery, Quebec, and there are dated records of its presence in Ontario.

Practically all vegetable crops are suitable hosts for land slugs and snails. Other favored hosts include daisy, gladiolus, iris, narcissus, salvia and strawberry. None of these slugs and snails has been implicated in the transmission of plant pathogens in Canada.

Damage In general, all above-ground plant parts are attacked, including foliage and fruit some distance above the ground, and plants become covered with slime trails and excreta. Below-ground parts of plants, such as bulbs and tubers, also may be attacked (see Potato, 16.53).

Life history Native species tend to be solitary and arouse little concern. The introduced land slugs and snails tend to be colonial. The introduced species also tend to be restricted to urban environments. They are most active at night when it is cool and humid, climbing up vegetation to feed on leaves and fruits. They burrow into the soil or under litter during the day and, in dry conditions, they protect their bodies with mucous secretions. Land slugs and snails tend to become more active as the temperature drops,

feeding at night or on cloudy days and showing a preference for damp situations. All individuals are hermaphroditic, having both male and female reproductive organs. The male organs usually develop first and degenerate later, so these animals mate and then become female.

The brown garden snail can live for several years. It mates and lays its eggs in the spring. Slugs mate in late summer and lay their eggs in the fall. In greenhouses or other protected areas, a slug may lay up to 400 eggs, though clutches of 30 to 150 are usual. Slug eggs hatch early in the spring. The young slugs may mature, breed, and die all in one season, or they may mature the following year, depending upon the species. In greenhouses, slugs may remain active year-round.

Identification Terrestrial slugs and snails in Canada have a shell and a ventral foot for crawling, and they obtain oxygen from either air or water by means of a vascular lung. Their stomach loops through the interior of the foot, and the excretory pore (anus) is positioned near the head on the right side of the body. Land snails do not close the aperture of their shell, thus differing from fresh-water snails, which posses a cover (operculum). The shell in most terrestrial slugs is concealed within a fleshy hood (mantle). Slugs and snails possess eyes at the tip of a second pair of tentacles (*11.27b).* Their eggs are colorless to white.

Management

Monitoring — Slugs and snails can be seen while they are still active in the early morning or late in the evening. Slime trails and excreta are signs of their presence.

Cultural practices — Destruction of hiding places is best done by burying plant residue and by clearing boards or stones away from the sides of buildings. In the past, some heavily infested areas were burned. A less drastic, more permanent, and environmentally more acceptable solution is to eliminate shady, damp areas. Hand-picking can be successful, and trap plants can be used to concentrate slugs and snails prior to hand-picking. Metal barriers are effective but practical only around raised beds.

Biological control — Mammals, birds, reptiles and amphibians are occasional predators on slugs and snails. Some native snails are carnivorous on other snails. Among insects, there are predatory beetles and flies, but none is marketed commercially.

Chemical control — Baits, employing wheat bran or corn meal combined with a pesticide, can be used but they should not be applied directly onto vegetable crops. Feeding stations should be located where pets and birds cannot gain access. Chemical sprays can be effective but timing of applications is critical.

(Original by D.C. Read, R.A. Costello and J.A. Garland)

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12 Maize (sweet corn)

BACTERIAL DISEASES

▶ 12.1 Stewart's wilt *Figs. 12.1 a-c*

Erwinia stewartii (E.F. Smith) Dye
(syn. *Xanthomonas stewartii* (E.F. Smith) Dowson)

This disease was first described in the United States in 1897. It is important on sweet corn worldwide. In Canada, Stewart's wilt occurs mainly in southwestern Ontario in Essex, Kent and Elgin counties where it is usually seen late in the season. Although its impact on yield is limited, many countries regulate against the pathogen, and imported seed corn must be free of the disease. Corn is the main host of the pathogen. Sweet corn and some inbred lines used in seed production are very susceptible.

Symptoms Sweet corn plants infected early in the season wilt and remain stunted. Severely affected plants may die. Late-season infection results in a foliar blight with symptoms that are similar to those of northern leaf blight *(Setosphaeria turcica* (Luttrell) K.J. Leonard & E.G. Suggs). Foliar lesions parallel the leaf veins and are pale green to yellow or brown *(12.1a).* The lesions may extend the entire length of the leaf and have irregular, wavy margins. Older leaves have a scorched appearance (*12.1b,c)* that may

be confused with drought and nutritional deficiency symptoms. If the lower stems of severely infected plants are cut, a bright yellow bacterial slime exudes from the vessels and forms strings when touched. Dark-colored cavities may be present in the pith of the lower stem.

Causal agent *Erwinia stewartii* is a Gram-negative rod, varying from 0.4 to 0.8 by 0.9 to 2.2 pm. It is facultative aerobic and non-motile. On nutrient agar, colonies are pale yellow to orange, slow growing and round to fluidal. Optimum growth occurs at pH 6.0 to 8.0 and 30°C.

Isolation — Necrotic tissue that has exhibited yellow bacterial exudate is the best to select for isolation of the pathogen. Small pieces of this tissue should be placed in a droplet of sterile water, cut and allowed to set for five minutes so the bacteria can ooze out. Using a sterile loop, a droplet of the exudate should be streaked onto nutrient agar or other non-selective media and the plates incubated at approximately 30°C until colonies appear. Representative discrete, yellow colonies should be selected and transferred to fresh media.

Disease cycle The pathogen overwinters in the digestive tract of the corn flea beetle and possibly other insects. Adult beetles spread the bacteria to corn when feeding on seedling leaves. Plants infected early in the season are severely affected. Leaf scorching accompanies late-season infection. Flea beetle feeding-sites, often seen on diseased plants, appear as thin, silvery scars on the leaf blade.

The bacteria may overwinter in infected seed from systemically infected corn plants. Although the bacterium may be found in most tissues of diseased sweet com, it does not overwinter in crop residue. Mild winters favor survival of the insect vector(s) and contribute to disease persistence. Excessive applications of nitrogen and phosphorus increase the severity of the disease.

Management Flea beetles are important in the overwintering and spread of *Erwinia stewartii* and any method to reduce their numbers contributes to wilt control. (See flea beetles, this chapter, 12.18.)

Cultural practices — Plowing of residues and crop rotation reduce pathogen inoculum.

Resistant cultivars — Most sweet corn varieties are susceptible to Stewart's wilt, but some *se*-gene (sugar- enhanced) sweet corn cultivars with the inbred line IL677a in their ancestry show resistance to Stewart's wilt; for example, Merlin, Miracle, Seneca Sentry, Sugar Buns, and Tuxedo. Field corn is generally less susceptible than sweet com.

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(Original by R.E Pitblado and R.A. Brammall)

FUNGAL DISEASES

▶ 12.2 Damping-off *Figs. 12.2a,b*

Stenocarpella maydis (Berk.) Sutton
(syn. *Diplodia maydis* (Berk.) Sacc.)
(syn. *Diplodia zeae* (Schwein.) Lév.)
Fusarium spp.
Penicillium spp.
Pythium spp.
Trichoderma spp.

These fungi are common soil inhabitants. *Fusarium* and *Penicillium* species often are resident upon and within seeds of sweet corn and are particular problems on the "supersweet" cultivars. *Fusarium, Trichoderma* and *Diplodia* species are inhabitants of corn residue and soil.

Pythium damping-off is associated with cold, wet soils and poorly drained sites.

Sweet corn is subject to a number of seed and seedling diseases capable of causing pre- or post-emergence damping-off. Seedling diseases can substantially reduce plant populations and may have the greatest detrimental impact on yield of any disease on this crop.

Symptoms Plant emergence may be slow or uneven in the spring. Plants that emerge may be slow growing, stunted, chlorotic and prone to wilt *(12.2b).* Stem and root tissues rot and may possess lesions that are characteristic of the organism responsible

(12.2a). Pythium causes dark, water-soaked lesions, *Fusarium* causes white, pink or purple lesions, while *Penicillium* and *Trichoderma* produce green to blue sporulation on the lesion surface. Identification requires microscopic examination for the various pathogens.

Causal agents The fungi are similar to those that cause damping-off in other vegetable crops. (For detailed descriptions of the causal agents: *Diplodia maydis*, see stalk rots, 12.8; *Pythium* spp., see Carrot, cavity spot, 6.8, and pythium root dieback, 6.13; *Fusarium* spp., see Bean, root rots, 15B.4; *Penicillium* spp., see three- to five-leaf dieback, 12.9.)

Disease cycle Damping-off may be caused by one or more soil- or seed-borne fungi (see three- to five-leaf dieback, 12.9). Infection and susceptibility are increased by factors that decrease seedling vigor, including planting into cold, compacted or waterlogged soils, planting too deeply and using old or damaged seed.

Management

Cultural practices — Growers should plant high quality seed and follow a crop rotation. Any practices that shorten the interval between sowing and plant emergence, such as selecting well-drained sites for planting, may reduce damping-off.

Chemical control — Chemical seed treatments will limit early infection of seedlings by soil- and seed-borne fungi.

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(Original by R.A. Brammall)

▶ 12.3 Ear and kernel rots *Figs. 12.3a-c*

Fusarium kernel rot
Fusarium moniliforme J. Sheld.
(teleomorph *Gibberela fujikuroi* (Sawada) Ito *in* Ito & K. Kimura) Other *Fusarium* species

Gibberella ear rot
Fusarium graminearum Schwabe
(teleomorph *Gibberella zeae* (Schwein.) Petch)
(syn. *Gibberella roseum* f. sp. *cerealis* (Cooke) W.C. Snyder & H.N. Hans.)

In eastern Canada, *Fusarium graminearum, F. moniliforme, F. subglutinans* and other species of *Fusarium* are associated with stalk rot and seedling blight of sweet corn and field corn. They also cause ear and kernel rot diseases of field corn and wheat and reduce the quality of animal feed and human food by producing mycotoxins in infected grain. The ear and kernel diseases generally are considered to be unimportant in sweet corn production in Canada. However, little is known of the disease in sweet corn, and growers should be aware that sweet corn cultivars are highly susceptible to infection by these pathogens. Supersweet types carrying the *sh2* endosperm mutation, which results in the accumulation of high levels of sugars in kernels, are particularly susceptible, and problems with poor emergence and seedling vigor are common in these hybrids.

Symptoms For symptoms of stalk rot, seed rot and seedling diseases, see stalk rots, 12.8, and three-to-five-leaf dieback, 12.9. Symptoms of ear rot and kernel rot are commonly expressed in infected field corn (*12.3a,b*), in which there is an interval of many weeks between silking and maturity. However, little information is available on the development of these diseases in sweet corn. The normal harvest period of sweet corn for the fresh market, usually two to three weeks after silking, may occur before symptoms are readily apparent; however, this period is well within the time required for the fungus mycelium to reach the developing kernels by growing down the silk channel. The holding ability of *sh2*-hybrids in the field, which permits extension of the harvest period of sweet corn for processing for up to two weeks, also extends the period during which infection and mycotoxin production can occur. In the United States, symptoms of ear rot of sweet corn at the eating stage have been associated with infection by *F. moniliforme* and *Fusarium poae* (Peck) Wollenweb., particularly in virus-infected plants and insect-damaged ears. Kernels infected by *F. moniliforme* may become moldy in an irregular pattern beginning at the tip of the ear; however, kernels free from symptoms also may be heavily infected internally and may be contaminated with the mycotoxin fumonisin.

Causal agents See stalk rots, 12.8.

Disease cycle The development of ear rot and kernel rot of field corn often follows damage to the ears by birds and by insects such as sap beetles *(12.3c).* However, infection also takes place following germination of spores that are deposited on the silks and growth of the fungus mycelium down the silk channel to the developing kernels. Infection and disease development by *F. graminearum* and *F. moniliforme* are favored by warm, showery weather at and following silk emergence. *Fusarium graminearum* infection of developing kernels can take place within 7 to 10 days following colonization of newly emerged silks; disease caused by *Fusarium moniliforme* occurs more readily in warmer climates and under relatively dry conditions. *Fusarium sporotrichioides* Sherb., *Fusarium subglutinans* (Wollenweb. & Reinking) Nelson, Toussoun & Marasas and *F. culmorum* (Wm.

G. Sm.) Sacc. are more common in cooler climates. Spores of *Fusarium* species are commonly found in air samples taken during the growing season and probably originate on debris of corn and other cereal crops, grasses and weeds. Infested debris is considered to be the major source of overwintering inoculum.

Mycotoxins — In small grains and field corn, *Fusarium graminearum* and *F. culmorum* damage the developing kernels and, more importantly, produce in infected kernels mycotoxins such as zearalenone and vomitoxin (deoxyni- valenol). These toxins can produce a variety of toxic effects in animals and humans. In cool climates, *F. sporotrichioides,* which produces several toxins including T2 toxin and diacetoxyscirpenol, is associated with moldy corn toxicosis in farm animals, as well as human toxicoses. In warmer climates, mycotoxins known as fumonisins are produced in corn kernels infected by *Fusarium moniliforme.* In the United States, *F. moniliforme* infection of corn kernels is widespread and fumonisin contamination of field corn is a serious problem in some areas; fumonisins also have been found in edible corn products. *Fusarium poae* is widespread in temperate climates in soil and as a weak pathogen, and it has been associated with the production of several mycotoxins, including diacetoxyscirpenol. No problems have been reported with mycotoxins in sweet corn produced in Canada. Cobs and kernels of sweet corn showing signs of whitish to pink fungal growth (*12.3a,b*), especially if associated with plant stress from drought, disease, or physical damage, as from bird or insect feeding, should not be used for food.

Management

Cultural practices — Corn stalks and other debris should be plowed under to reduce the inoculum load in crop fields; sweet corn should not be grown in rotation with or seeded into stubble of field corn or susceptible small grains, such as wheat and barley, or planted in or adjacent to fields having surface debris on which spores of the pathogens may be produced. Crops should be grown under conditions of well balanced soil fertility and adequate soil moisture. Only undamaged, mold-free ears should be marketed or used in preparing edible corn products.

Resistant cultivars — Genetic resistance offers the best potential for management of these diseases, but relatively little is known of sources and inheritance of resistance.

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(Original by W.L. Seaman and J.D. Miller)

▶ 12.4 Eyespot *Fig. 12.4*

Kabatiella zeae Narita & Hiratsuka
(teleomorph *Aureobasidium zeae* (Narita & Hiratsuka) J.M. Dingley)

Eyespot is found mainly in southern Ontario, especially during warm, humid weather in early spring. It has been a problem in areas where reduced tillage of corn has been practiced. The disease damages leaves and reduces yield. Corn is the only known host of the pathogen.

Symptoms Eyespot is characterized by the presence of small, round lesions (1 to 4 mm) on the leaves. These spots are initially water-soaked and later appear as a brownish ring around a pale central area, surrounded by a narrow yellow halo that gives the lesion the "eyespot" appearance. Older leaves may have numerous infections that grow together, thereby killing large amounts of tissue and reducing photosynthetic area (*12.4).* Examination of lesions for the presence of conidiophores and conidia may be required to differentiate eyespot from bacterial leaf spots.

Causal agent *Kabatiella zeae* produces conidia on short conidiophores that emerge through the stomata of infected leaves. The hyaline, non- septate conidia vary from 3 to 4 by 18 to 33 pm and are curved with pointed ends. The fungus can be cultured by streaking conidia or by placing infected tissue onto media, such as potato-dextrose (PDA), V-8, oatmeal, cornmeal or Czapek agar. On PDA, colonies are at first yellow or pink, but later turn dark blue to black and have a tough, leathery appearance. PDA can be amended with novobiocin at 100 mg/L to aid in isolation. The fungus loses virulence when repeatedly transferred onto artificial substrates.

Disease cycle The fungus overwinters in infested corn residue. It also has been reported to be seed-borne in corn, which may aid long-distance dispersal. Conidia are produced in the spring and are carried to young plants by wind and splashing rain. Foliar lesions are seen from 4 to 10 days after infection and are the source of conidia for secondary spread through the field. The disease is favored by cool, wet weather, but the optimum temperature for spore germination is 24°C.

Management

Cultural practices — Growers should use clean plowing, crop rotation and other tillage procedures that minimize the amount of corn residue on the soil surface.

Resistant cultivars — Eyespot can be controlled through the use of resistant cultivars.

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(Original by R.A. Brammall)

▶ 12.5 Rust, common *Fig. 12.5*

Puccinia sorghi Schwein.

Common rust, although considered a minor problem, has recently increased in prevalence with an expansion in planting of susceptible cultivars. In addition, increased winter corn production in the southern United States has led to increased levels and earlier production of the primary urediniospore inoculum responsible for outbreaks in Canada. The evolution of new pathogenic rust biotypes that can defeat single gene resistance within the host and the extension of the sweet corn growing season in the fall when conditions favor rust development also have contributed to the increased prevalence of this disease. Only corn and the sorrels (*Oxalis* spp.), the alternate hosts of *P. sorghi,* are infected.

Symptoms Common rust becomes noticeable toward the end of the season when reddish-brown pustules (uredinia) appear over the leaf surface (72.5). The pustules vary from nearly circular to elongate and measure 1 to 3 mm in length. They erupt through the leaf epidermis to produce urediniospores. Later in the season, the pustules turn black as the production of teliospores commences. Heavily infected leaves may become chlorotic and die.

Causal agent *Puccinia sorghi* is a macrocyclic, heteroecious rust. The binucleate, reddish-brown urediniospores vary from 21 to 30 by 24 to 33 µm and possess a spiny surface ornamentation. The teliospores are two- celled, dark to golden brown, slightly constricted at the septum, measure 14 to 25 by 28 to 46 µm, and possess a pedicel that may vary from two to four times the length of the spore body. The fungus exists as a number of different specific virulence phenotypes or biotypes which vary in their responses to *Rp* genes in corn. The exact biotype present may vary with geographic location and time.

Disease cycle The fungus overwinters as thick-walled teliospores in corn leaf refuse in or on the soil. In spring, the teliospores germinate to form basidia and basidiospores, which infect only wood sorrel. Sexual reproduction occurs on the wood sorrel, eventually resulting in the production of aeciospores, which are carried by wind to corn leaves where they cause infections that lead to the development of uredinia and urediniospores. Infection also can occcur within the leaf whorl where humidity is high, thus providing favorable conditions for spore germination. Such infections may lead to the development of transverse lesions on the emerging leaves. The urediniospores or "summer" spores cause repeating cycles of infection on corn throughout the season.

In Canada, the role of the alternate host wood sorrel is unimportant. Epidemics arise from air-borne urediniospores that blow in from the American corn belt. This makes it difficult to predict when the disease will appear. Cool temperatures (16 to 23°C) and high relative humidity or prolonged periods of leaf wetness increase the incidence and severity of the disease. Losses in yield and quality and delayed maturity result from severe early-season leaf damage. Older leaf tissue is more resistant to infection.

Management

Cultural practices — Sweet corn planted early often escapes the disease.

Resistant cultivars — Seed companies have developed cultivars with mono- and multigenic resistance for late-sea son plantings. These cultivars should be planted in areas where rust is prevalent.

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(Original by R.E. Pitblado and R.A. Brammall)

▶ 12.6 Smut, common *Figs. 12.6a,b*

Ustilago zeae (Beckm.) Unger (syn. *Ustilago maydis* (DC.) Corda)

Common smut is found wherever sweet corn is grown in Canada. Its incidence and severity are determined by cultivar resistance, presence or absence of inoculum, and weather conditions. In most years, common smut is of little economic importance; however, localized areas of heavy damage are occasionally seen in a few fields. The pathogen has a narrow host range that includes mainly field corn and sweet corn. [In Mexico and elsewhere in the southern hemisphere, corn smut is regarded as an edible delicacy known as cuitlacoche. In the northeastern USA, some farmers produce galls of common smut of sweet corn for restaurants specializing in authentic Mexican cuisine. In discriminating markets, the commercial value of the diseased crop thus may greatly exceed that of the healthy crop. — Eds.]

Symptoms The symptoms are dramatic and easily recognized. The disease results in the production of smooth shiny galls or "boils," often 2 to 10 cm in diameter. The galls form anywhere on the aerial parts of the plant but are most common on the developing ears *(12.6a)* where they transform individual kernels. Externally, young galls are pale green to metallic silver. Internal tissue is rapidly converted into a black powdery mass of spores that are released upon rupture of the gall. Sweet corn ears affected by common smut are unmarketable. If galls form on the tassels, stems or leaf edges, they are usually small, brown and hard. Galls on these tissues contain few spores, usually do not rupture, and cause little damage. Infected tassels *(12.6b)* occasionally form a small unmarketable ear or what appears to be female plant tissue. A less conspicuous symptom is the production of chlorotic spots on the leaves at points of infection. The disease may be differentiated from head smut by the absence of remnants of the host vascular tissue within the gall and by details of teliospore morphology.

Causal agent *Ustilago zeae* produces olive-brown to black, dikaryotic teliospores, also known as chlamydospores or brand spores, which vary from 8 to 11 µm in diameter and possess a spiny surface ornamentation. At maturity, the teliospore nuclei fuse to produce a single, diploid nucleus. A promycelium produced upon germination of the teliospore is the site of meiosis. The promycelium divides by three transverse septa to yield four haploid cells, which divide mitotically until one of the daughter nuclei is contained in a sporidium. The sporidium is functionally a basidiospore formed by budding from promycelial cells.

The sporidia are capable of saprophytic growth by yeast-like budding. Eventually fusion between compatible sporidia occurs. This fungus is generally heterothallic and bipolar, but parasexual recombination is also known to occur. The saprophytic capability of the sporidia has been exploited to allow axenic culture of this spore type, usually in shake culture systems.

Disease cycle The teliospores overwinter in soil and crop residues or in contaminated seed. In the spring, they germinate to produce a promycelium and sporidia. The sporidia are responsible for infection. They germinate on the surface of the host to produce hyphae that either penetrate the corn epidermal cells directly or enter the plant through stomata or wounds. Infection will not proceed unless two compatible sporidia fuse. The resulting dikaryotic mycelium grows intercellularly and stimulates the host tissue to form galls through processes of uncontrolled cell enlargement and division (hypertrophy and hyperplasia). In later stages of infection, intracellular penetration occurs. Eventually, the dikaryotic mycelium converts into diploid teliospores, which release upon rupture of the gall surface. Unlike some smut pathogens, teliospores of this fungus also may cause new infections of meristematic host tissue. Usually the infections are localized and not systemic through the plant.

Wind, hail storms and insects produce wounds that expose plants to infection. Periods of leaf wetness are required for germination of sporidia, but not for germination of teliospores. The development of common smut is favored by dry conditions and temperatures between 26 and 34°C.

Management

Cultural practices — Although crop rotation has been recommended for the control of common smut, aerial spore dispersal often counteracts this strategy. Removal and destruction of the galls before spore release may be of value in small gardens. Growers should avoid the excessive use of nitrogen fertilizers and minimize mechanical injury to plants during field operations.

Resistant cultivars — Genetic resistance is the only practical control measure, but all current cultivars are susceptible to some degree. The high level of genetic diversity in the pathogen has made breeding for monogenic resistance in sweet corn impractical.

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(Original by R.E Pitblado and R.A. Brammall)

▶ 12.7 Smut, head *Figs. 12.7a,b*

Sporisorium holci-sorghi (Rivolta) K. Vanky
(syn. *Sphacelotheca reiliana* (Kühn) G.P. Clinton)
(syn. *Ustilago reiliana* Kühn)

Head smut occasionally causes economic losses in sweet corn. The disease was reported in 1979 from British Columbia, Ontario and Quebec. To date, it has not been commercially significant. Head smut also affects sorghum and sudan grass.

Symptoms Black teliospores occur over the entire surface of the ear and tassel *(12.7a).* Sporulation is infrequent on leaves. Smutted ears usually do not have any kernels or silks. Smutted tassels grow abnormally, developing a brush-like appearance. Leaf-like growth or phyllody is characteristic of the smutted tissues *(12.7b).*

The disease is distinguished from common smut by the absence of galls and the conspicuous production of sori on the tassels. Vascular strands of the host are found in head smut sori.

Causal agent The fungus is dimorphic, producing teliospores and sporidia. The teliospores are globose, reddish-brown to black, possess a spiny surface ornamentation and vary from 9 to 12 pm. On culture media, they produce hyaline subglobose, haploid sporidia that vary from 7 to 15 pm. In soil, they germinate to produce infection hyphae that lack sporidia. The sporidial stage is saprophytic and monokaryotic, and may be maintained on potato-dextrose agar or other simple culture media. Growth occurs by budding. Compatible sporidial lines may combine to yield a parasitic dikaryotic mycelium.

Plants may be inoculated with head smut either by direct injection of mixed sporidial cells of the appropriate mating types, or by germinating seedlings in soil infested with teliospores.

Disease cycle The teliospores overwinter and may persist in the soil or on contaminated seed for more than 10 years. They germinate to produce dikaryotic infection hyphae that directly penetrate the epidermis of emerging seedlings to establish a systemic infection. Sori appear in place of the ears and tassels and new teliospores form. The mature teliospores fall to the soil or contaminate the seed. Teliospores may also germinate to produce a promycelium with haploid sporidia, which are thought to be unimportant as sources of inoculum.

The occurrence of head smut is related to the number of teliospores in the soil. Seedling infection is favored by temperatures of 21 to 28°C and relatively low soil moisture levels.

Management

Cultural practices — Crop rotation and sanitation aid in controlling head smut.

Resistant cultivars — Many commercial corn cultivars possess resistance to head smut.

Chemical control — Chemical seed treatments are available.

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(Original by R.E Pitblado and R.A. Brammall)

▶ 12.8 Stalk rots *Figs. 12.8a-d*

Diplodia stalk rot

Stenocarpella maydis (Berk.) Sutton
(syn. *Diplodia maydis* (Berk.) Sacc.)
(syn. *Diplodia zeae* (Schwein.) Lév.)

Fusarium stalk rot

Fusarium moniliforme J. Sheld.
(teleomorph *Gibberella fujikuroi* (Sawada) Ito *in* Ito & K. Kimura)
Fusarium subglutinans (Wollenweb. & Reinking) P.E. Nelson, T.A. Toussoun & Marasas
(syn. *Fusarium moniliforme* var. *subglutinans* Wollenweb. & Reinking)
(teleomorph *Gibberella subglutinans* (E. Edwards) P.E. Nelson, T.A. Toussoun & Marasas)

Gibberella stalk rot

Fusarium graminearum Schwabe
(teleomorph *Gibberella zeae* (Schwein.) Petch)
(syn. *Gibberella roseum* f. sp. *cerealis* (Cooke) W.C. Snyder & H.N. Hans.)

Pythium stalk rot

Pythium aphanidermatum (Edson) Fitzp.
Pythium spp.

In some areas of Canada, sweet corn is commonly affected by fungal stalk rots. Symptoms usually appear late in the season when plants begin to allocate photosynthate to ear production. The causal fungi overwinter in the soil on infested crop residues.

Fungal stalk rots are not as important in sweet corn as in field corn because sweet corn is usually harvested much earlier. Stalk rots are often associated with senescing or physiologically stressed plant tissues.

Symptoms Diplodia stalk rot causes sudden wilt and plant death after silking *(12.8a).* The leaves turn a dull gray-green color, reminiscent of frost injury. The pathogen produces a dryish, pale brown rot of the pith at the lower stem internodes.

Fusarium stalk rot produces symptoms similar to those of gibberella stalk rot. The crown and root may also be affected. A pink to red discoloration is often observed in the rotted tissues.

Gibberella stalk rot causes pith destruction accompanied by a shredded appearance in the stem interior *(12.8c).* The rotted tissue is often pink to red *(12.8b),* and small, black perithecia may form superficially. Affected plants turn a dull light green and senesce prematurely. Rotting of the lower internode leads to stem breakage and lodging.

Pythium stalk rot causes a sudden, water-soaked stem rot near the soil line *(12.8d)* at the time of tasseling. The plants may not wilt immediately but the rotted area turns brown. Lodged plants are generally twisted at the stalk lesion.

Causal agents

Stenocarpella maydis — Although the taxon *Diplodia maydis* has been reduced to synonymy with *S. maydis,* the former name has been used extensively in the literature. *Stenocarpella maydis* produces globose to elongate, dark-brown pycnidia below the host epidermis. This distinguishes it from *Gibberella* species, which produce perithecia superficially. The elliptical, pale brown conidia are one- to four-celled (usually two), and about 5 by 28 µm. The conidiogenous cells possess a minute collarette, and conidiophores are usually absent. This collarette distinguishes the taxon *Stenocarpella* from *Diplodia.* The conidia are expelled from the pycnidium in cirrhi. The pathogen can be cultured on common agar media and forms a brown mycelium. Pieces of autoclaved corn leaves placed on oatmeal agar encourage the formation of pycnidia.

Fusarium moniliforme — This fungus produces macroconidia with three to seven septa and spindle-shaped to ovoid microconidia in chains on simple phialides. No chlamydospores are produced, further distinguishing this species from *F. oxysporum,* which it closely resembles in appearance. Dark blue, rough-walled perithecia of *Gibberella fujikuroi* form superficially on dead plant tissues. These structures are globose to conical and measure 250 to 350 by 220 to 300 µm. Ascospores are hyaline, elliptical, one- to three- septate, and 14 to 18 by 4.5 to 6 µm. The pathogen is easily cultured on potato-dextrose or V-8 agar. Colonies develop a powdery appearance from the production of chains of microconidia on the colony surface. Cultures vary from colorless to dark violet. Microsclerotia may form in culture, but these structures have not been observed in infected corn tissue.

Fusarium moniliforme var. *subglutinans* — This variety is distinguished from *F. moniliforme* by the production of microconidia on branched conidiophores, which terminate in polyphialides, and by the absence of microconidial chains. Perithecia of *Gibberella subglutinans* resemble those of *G. fujikuroi,* the ascospores are somewhat thinner (12 to 15 by 4.5 to 5 µm). Isolation techniques for this fungus and its appearance in culture are similar to those of *F. moniliforme.*

Fusarium graminearum — This species differs from *F. moniliforme* in that it does not produce microconidia. Macroconidia arise from stubby, doliform phialides. *Fusarium graminearum* has been divided into two groups: Group 1, which is soil-borne, does not or rarely forms perithecia, and usually causes crown rot of cereals; and Group 2, which produces perithecia and air-borne ascospores and causes stalk and ear rot of corn and head blight or scab in small grains. Surveys in northern areas of the mid- western United States have revealed that corn stalk rot isolates are in Group 2, but similar surveys have not been conducted in Canada. Perithecia of *Gibberella zeae* are superficial, blue to black, round to ovoid, and measure 140 to 150 µm in diameter. They usually form in clusters around the lower nodes of affected plants. Asci formed within the perithecia are clavate, 60 to 85 by 8 to 11 µm, and contain four to six (usually eight) hyaline to light brown, non- to three-septate ascospores 19 to 24 by 3 to 4 µm. Cultures on potato-sucrose agar vary from rose to crimson, but pigmentation may differ on other media. Many isolates fail to produce chlamydospores in culture.

Pythium aphanidermatum — Hyphae are broad and aseptate, though septa may occur where sporangiophores are produced on the parent mycelium. Zoosporangia, which develop on the sporangiophores, release biflagellate zoospores. The sexual oospores vary from 17 to 19 pm in diameter and are produced in terminal oogonia.

Stalk rot pathogens can be isolated by placing tissue excised from the lesion margins onto media such as potato-dextrose or V-8 agar amended with 75 ppm streptomycin or other antibiotic to inhibit bacterial growth. When such tissue is obtained from the lower stem, it is often advantageous to wash it under running tap water for several minutes to remove adherent soil. Several media are more or less selective for some of these pathogens; these include Komada's medium for *Fusarium, Trichoderma* selective medium, and *Pythium* selective media (see Additional references, Dinghra & Sinclair 1985).

Disease cycle Stress conditions, such as high plant density, leaf damage, and unfavorable temperature, water and light levels, favor the development of fungal stalk rots. Although corn genotypes vary in susceptibility, all may be susceptible under conditions favorable to the disease. Diplodia, fusarium and gibberella stalk rots are promoted by dry conditions in the spring and by warm, wet weather following silking. Pythium stalk rot is favored by hot, wet conditions and poor soil drainage, and it may occur at any time during the season.

Diplodia stalk rot — *Stenocarpella maydis* can infect a plant from infested seed or through conidia. Conidia may be moved by wind or insects and infect the lower stem through the leaf sheaths or below-ground tissues. Damage to the ear near the time of silking may result in ear rot and seed infection. The fungus overwinters as conidia in pycnidia in crop residues or on seed. *Stenocarpella maydis* has been reported to cause stalk rot in zero-tillage fields.

Fusarium stalk rot — *Fusarium moniliforme* overwinters as sporodochia on corn stubble, as short mycelial fragments, or as thickened hyphae within sclerenchyma and parenchyma cells in crop debris. Seed-borne inoculum is less important in the epidemiology of the disease. Corn roots become infected when they contact fungal inoculum or infested debris. Conidia can directly infect uninjured stalk tissues at the base or leaf sheaths and grow into the nodes. The fungus will also colonize wounds made by insects or hail. Stalk rot symptoms may or may not develop, depending on the vigor of the infected plants, but a purple discoloration is often seen in the node tissues upon colonization. The fungus saprophytically colonizes the tissue and either sporulates on it or forms thick hyphae. Asparagus isolates of the pathogen may be pathogenic on corn.

Gibberella stalk rot — The disease cycle and epidemiology are similar to that of diplodia stalk rot. Ascospores, macroconidia and possibly chlamydospores function as inoculum. *Fusarium graminearum* survives in debris in or on the soil surface as hyphae, chlamydospores or macroconida.

Pythium stalk rot — *Pythium* species may attack seed, seedlings and plants through direct infection of the roots and crown. Seeds with broken pericarps are easily infected by this fungus. Cold soil appears to favor *Pythium* infection of young plants, while stalk rot develops in more mature plants when conditions are hot and wet. The pathogen likely overwinters as oospores and chlamydospores in infested plant debris. These germinate in the vicinity of plant roots to cause new infections. Insects spread *Pythium* from plant to plant.

Management

Cultural practices — Growers should rotate crops to limit exposure of corn to soil-borne pathogens. Stress and mechanical or insect damage to the stem increases susceptibility. By reducing plant density, growers can lessen the risk of plant-to-plant spread of fungal stalk rots. Nitrogen and potassium fertility should be balanced because fungal stalk rots are often more severe in the presence of excessive nitrogen.

Resistant cultivars — Cultivars with resistance to stalk rot fungi are available and should be grown where this disease is a potential problem.

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(Original by R.A. Brammall)

▶ 12.9 Three- to five-leaf dieback *Figs. 12.9a-d*

Penicillium spp**.**

Three- to five-leaf dieback has become a serious problem in the cultivation of the supersweet or shrunken *sh2*-gene corn type. Supersweet corn is displacing the more traditional sweet corn types for processing because it remains fresh longer, which is an asset during harvest and transport. The disease causes pre- and post-emergent plant death, resulting in poor plant stands and low yields.

Penicillium species are common inhabitants of soil and often occur on plant surfaces, seed and crop residues. Several species have been implicated in seed decay and spoilage of stored grain. One of these, *Penicillium oxalicum* Currie & Thom, also causes a stem rot of greenhouse cucumber (see Greenhouse cucumber, penicillium stem rot, 22.14).

Symptoms The disease is characterized by poor emergence and survival *(12.9b,c).* Plants may be killed before emergence by rotting of the seed or the radicle near the level of the seed, or they emerge but are stunted and chlorotic with brown lesions scattered over the subcrown internode and root (*12.9a,d*). The lesions may girdle the plant. Affected plants may wilt and die early in the season from extensive root damage. The disease may be difficult to identify if most plants die before emergence. Poor stands and the presence of stunted, chlorotic plants that die near the three- to five-leaf stage of development are characteristic *(12.9c).* Microscopic examination of seed and affected plants is required to distinguish this from other fungal diseases.

Causal agent Several species of *Penicillium* have been associated with corn seed. These organisms differ in temperature and humidity optima for growth, and the actual seed-borne residents depend on conditions during seed processing and storage. In the United States and Israel, *P. oxalicum* has been associated with seedling blight of com.

All *Penicillium* conidiophores arise from the mycelium, often singly, and branch near the apex in penicillate or brush-like fashion. The conidiophore terminates in a group of phialides that produce one-celled, globose conidia in chains. *Penicillium* spp. are easily grown on standard potato- dextrose or yeast-peptone agar. The cultures are often brightly colored, frequently green or blue, with a dry, dusty appearance.

Disease cycle This disease is associated with the *sh2* gene in supersweet corn cultivars. Seeds appear shrunken and the pericarp is frequently cracked or split, facilitating colonization of the kernels by *Penicillium* or *Fusarium.* Phytotoxins that inhibit germination, such as penicillic and oxalic acids, may be produced in the infested seed.

Colonization by storage molds, including *Pencillium* spp., is enhanced by damage to the seed during harvesting and shelling, and by damp conditions during drying in the field. The disease is most severe under field conditions that delay plant emergence. Losses exceeding 50% of plant stands are not uncommon when temperatures lower than 14°C prevail at planting.

Management

Cultural practices — Growers should use only high quality seed and avoid seeding too deeply. Low temperatures before plant emergence increase disease severity, so seeding should be delayed until soil temperatures favor rapid emergence.

Chemical control — Fungicidal seed treatments may aid in controlling this disease.

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(Original by R.A. Brammall)

VIRAL DISEASES

▶ 12.10 Maize dwarf mosaic *Figs. 12.10a,b*

Maize dwarf mosaic virus

Maize dwarf mosaic virus causes mottling of the foliage, chlorosis and stunting. Commonly grown sweet corn cultivars are susceptible to this disease. In eastern Canada, two strains were thought to occur: strain A, which has Johnson grass *(Sorghum halepense* (L.) Pers.) as a perennial host, and strain B, which does not infect Johnson grass. However, strain B has recently been classified as a strain of sugarcane mosaic virus based on serological studies, amino acid composition and sequence of the viral coat proteins, as well as on pathogenicity and symptom development in selected sorghum cultivars.

Symptoms In very young seedlings and before tasseling, plants may display a dark-green streaking or mosaic stippling over the surface of the lighter colored to chlorotic leaves (*12.10a,b).* Affected seedlings are more susceptible to root and stalk rot pathogens than normal. Stunting may occur through a shortening of the upper intemodes. Infected plants may produce excessive

numbers of tillers and ears, causing a reduction in marketable yield. Later in the season, especially under warm conditions, the mosaic symptom may disappear and be replaced with a general chlorosis.

Causal agent Maize dwarf mosaic virus is a filamentous, flexuous rod that ranges in size from 12 to 15 by 750 nm. It is a member of the potyvirus group. Sugarcane mosaic virus has similar morphology.

The disease is identified by the presence of the dark-green mottling or mosaic pattern on the young leaves. Serological or electron microscopic techniques are required for precise identification of the virus.

Disease cycle The virus overwinters in a wide range of grass hosts (strain B does not overwinter in Johnson grass). Wheat, barley, oats and rye are non-hosts, but sorghum may be infected. The virus is transmitted from these grass hosts to sweet corn by a number of insect vectors, including the corn leaf aphid and the green peach aphid. The virus may be successfully transmitted for up to six hours after aphids have fed on an infected host. The virus is also known to be seed-borne in sweet corn. Disease severity is often greatest on late-planted sweet corn, possibly from the greater numbers of the insect vectors that are present when the crop is developing and as the season advances.

Management Control of maize dwarf mosaic virus may require the application of insecticides to eliminate the insect vectors and on the use of herbicides to eliminate the weedy grass hosts in which the pathogen overwinters.

Resistant cultivars — When possible, cultivars with resistance to both strain A and strain B (sugarcane mosaic virus) of the virus should be grown.

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(Original by R.A. Brammall)

NEMATODE PESTS

▶ 12.11 Stubby-root nematodes

Paratrichodorus allii (Jensen) Siddiqi
Paratrichodorus pachydermus (Seinhorst) Siddiqi
Paratrichodorus spp.
Trichodorus spp.

This group of nematodes is not well established in Canada and has caused only minor damage to a few gardens in southern Alberta.

Damage Affected plants become stunted and chlorotic. Roots proliferate abnormally but appear not to grow in length and their extremities may be somewhat swollen. For a complete description, see Potato, 16.39; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 12.12 Army worm *Figs. 12.12a-c*

Mythimna unipuncta (Haworth)
(syn. *Pseudaletia unipuncta* (Haworth))

The armyworm is native to North America. In Canada, it occurs naturally in areas with warmer winters and invades all corn-growing areas from the Atlantic to the Pacific. These periodic invasions of migrants are associated with storm fronts from south of the Canada-United States border.

Sweet corn is the only vegetable attacked. Other hosts include field corn, oat, wheat and other small grains and grasses.

Damage Corn can suffer severe damage *(12.12a).* The leaf blades are often totally eaten, leaving only the mid-ribs. In the counties bordering Lake Erie, the armyworm often becomes numerous enough to cause serious economic loss.

Identification The armyworm (family Noctuidae) larva (*12.12b)* is a caterpillar about 5 cm in length at maturity, dark green and hairless with five whitish stripes along the length of the body. The head is a pale green- brown with darker mottling. The pupa is about 3 cm long and red-brown. The moth's wingspan is about 4 cm; the forewings (*12.12c*) are pale gray- brown with a white

dot near the centre, which is useful for field recognition; the hindwing has a dark margin. The eggs are whitish, bead-like, and laid in masses.

Life history In the spring, storm fronts carry moths northward from the southern United States, or moths may emerge locally in some areas. However, the moths are seldom seen because they are active mainly at night when they feed on nectar, mate, and lay eggs. Eggs are laid in folded leaves or leaf sheaths in June and hatch in three weeks. Young larvae appear from late June to mid-July and grow rapidly. They feed mostly at night on leaves near the soil surface, hiding in the leaves at the centre of plants during the day. After exhausting a food supply, or when grain is ripe or hay has been cut, they crawl to nearby fields in search of food. In a month, they mature and pupate in the soil. Usually, the pupa does not survive the winter in Canada.

Management

Monitoring — The best time to look for larvae is when they are feeding in the evening or early morning, although they are not usually noticed until they are at least half grown and crop damage is advanced. This may be in early July in southern Ontario, or mid-July in the rest of eastern Canada. Growers should check areas of corn fields that border grain or hay fields. An economic threshold of about 60 larvae per m^2 has been used to determine when control measures are necessary.

Biological control — Outbreaks may occur despite the presence of natural enemies, but usually the armyworm is held in check by flies, wasps, ground beetles, birds, toads, skunks and diseases.

Chemical control — Chemicals work best when the larvae are small and there are few natural enemies present. In a severe infestation, insecticides may be the only effective means of control. The outside rows beside hay, pasture and grain fields are often the only sites affected, in which case only the headlands of adjacent fields and a few outer rows of corn need be treated. Each field should be assessed separately. Before deciding to treat, growers should consult an extension agent because insecticides should be a last resort for control of this insect.

Insecticides should be applied on warm evenings before the larvae become active and when the plants are dry. Only those sections of fields that are infested should be treated, including a 10-m border to catch crawling larvae. For infestations in home gardens adjacent to corn fields, a poison bran bait or cutworm spray can be used.

(Original by M. Hudon)

▶ 12.13 Corn earworm *Figs. 12.13 a-d*

Helicoverpa zea (Boddie)
(syn. *Heliothis zea* (Boddie))

The corn earworm is native to the Americas and the Caribbean. It is not a permanent resident north of 39°N. It migrates from the southern United States, where it normally overwinters, to infest northern states and some parts of Canada. In Ontario, it is the most destructive insect pest of sweet corn after the European corn borer. On account of its inability to overwinter in Canada and the uncertainty of wind patterns from the southern United States, growers never know when the invasions will occur. Normally, the corn earworm is a late-season insect, occurring from late August through September. Problems with corn earworm infestations are caused by using pest control products that are effective for corn borer but ineffective for the corn ear- worm. This, along with poor spray coverage and timing, add to the inconsistency of earworm control.

The larval hosts include corn, pepper and tomato.

Damage The corn earworm can be damaging if detected too late *(12.13a).* The larvae feed initially in the ear silks but move quickly into the ear itself and feed on the kernels *(12.13b,c).* A single larva can consume the whole tip of an ear of corn, eating and fouling the kernels. It also may destroy the silks before pollination has been completed. Molds may invade the larval feeding site. Damage is often overlooked because the husk on the ears rarely displays holes, making the insect difficult to detect or control.

Identification The corn earworm (family Noctuidae) adult is quite a large (a wingspan from about 4.5 to 6.5 cm), yellow-brown moth *(12.13d).* Its eggs are pale green. The mature larva (*12.13b*) is a caterpillar about 4 cm in length. It varies from light green or brown to almost black with a yellow head, black legs, light and dark stripes lengthwise along the body, and a paler underside. Larvae of the European corn borer differ, being dotted with plates (pinacula).

Life history Eggs are laid singly, most often on the corn silks, and the young larvae hatch in three days. They feed on the silks, then enter the ear usually by feeding from the tip of the ear to the kernels, which they eat completely. The larval stage lasts about a month. When mature, larvae drop to the ground and pupate in the soil.

Management

Monitoring — Growers should inspect sweet corn before silking, watching for larval feeding damage at the tips of the ears. Pheromone traps are available to monitor for the adult moths.

Resistant cultivars — Some corn cultivars are more susceptible than others. In general, those with long ears and tight husks extending beyond the ear are more resistant to infestation than short-eared, short-husked cultivars. Growers should consult an extension agent about cultivars for their area.

Biological control — Several parasites attack the eggs and larvae; a number of predaceous insects and birds help to reduce populations.

Chemical control — Treatment is recommended to keep damage to a minimum but the infestation must be detected early. In commercial plantings, growers should use a high- clearance hydraulic boom sprayer, using drop pipes with nozzles directed at the corn ear silks. To ensure good coverage in home gardens, a compressed-air hand sprayer should be used. A foliar insecticidal treatment should be applied when the first symptoms of insect damage appear and repeated at intervals, according to pesticide and crop recommendations.

(Original by M. Hudon and R.E. Pitblado)

▶ 12.14 Corn leaf aphid *Fig. 12.14*

Rhopalosiphum maidis (Fitch)

The corn leaf aphid is an annual immigrant in Canada and is most prevalent in southern Ontario and Quebec, although it is found wherever sweet corn is grown. It overwinters in the United States, moving northward on air currents from areas where crops are more advanced.

Damage The plant may be dwarfed, whorl leaves may become desiccated, tassels and silks may be covered with honeydew, and yields may be greatly reduced. In some cases, the plants will be barren of ears or the ears devoid of kernels. Honeydew produced by the aphids interferes with pollination and the ears of sweet corn may be unsuitable for fresh-market sale because of a sooty mold that grows on the honeydew. Plants affected by moisture stress yield less when this aphid is present. However, rainfall during the weeks just before pollination can reduce the effects of aphid feeding damage. Treatment with chemical insecticides is rarely justified except on commercial seed crops.

The corn leaf aphid is a vector of maize dwarf mosaic virus.

Identification The corn leaf aphid (family Aphididae) is greenish blue and usually occurs on the tassels and upper leaves of sweet corn *(12.14)*. It lacks dorsal markings anterior to the paired, abdominal projections (cornicles) and often has a waxy appearance. A related but less serious species, the birdcherry-oat aphid *Rhopalosiphum padi* (L.), may also infest sweet corn in Canada. It is yellowish green to green-black with rust-colored patches around the base of the cornicles. A specialist should be consulted to confirm which species is present.

Life history All forms of the corn leaf aphid are female. Upon becoming adult, they produce live young without mating. Infestations on corn begin when the plants are in the whorl stage, which provides the aphid with a moist, nutritious, protected area during its reproductive period and accounts for its very rapid build-up. Populations are usually greatest in dry years and die naturally in the fall in Canada.

Management

Monitoring — The corn leaf aphid may persist as an occasional pest on corn foliage in late summer. Populations may be so great by the time the tassels have emerged that the entire upper part of the plant can be covered with aphids. Therefore, before the tassels are exposed, plants in several areas of a field should be examined to determine if there is a pre-pollination build-up of aphids in the whorl. The economic threshold for application of insecticides by the canning industry in Quebec for European corn borer (10% damaged ears for processing sweet corn and 5% for fresh-market sweet corn) can be used for the corn leaf aphid.

Cultural practices — Sweet corn cultivars vary in their susceptibility to the corn leaf aphid. Greatest infestations occur on cultivars that also are susceptible to European corn borer. Early planting of sweet corn can minimize aphid population build-up and reduce the effects of their feeding damage.

Biological control — Lady beetles are important aphid predators in sweet corn.

Chemical control — For plants at the whorl and early tassel stages, a systemic insecticide is generally more effective than a contact insecticide.

(Original by M. Hudon)

▶ 12.15 Corn rootworms *Figs. 12.15a-c*

Northern corn rootworm *Diabrotica barberi* Smith & Lawrence
Southern corn rootworm *Diabrotica undecimpunctata howardi* Barber
Western corn rootworm *Diabrotica virgifera virgifera* LeConte

The northern corn rootworm occurs from Essex County to the Bay of Quinte area of Ontario; also, it is the predominant species east of Toronto to southwestern Quebec. The southern corn rootworm, also known as the spotted cucumber beetle (see Cucurbits,

cucumber beetles, 9.21), occurs from the Rocky Mountains eastward to Ontario and Quebec in Canada. The western corn rootworm, discovered in Ontario in 1975, is found south and west of Ottawa.

Apart from corn, no other economic hosts are known for the northern and western corn rootworms; the southern corn rootworm is a general feeder on many plants, including cucurbit crops.

Damage The corn rootworm adults are pollen feeders. They cause great injury to sweet corn because they clip and destroy the silks while feeding before pollination *(12.15a).* This results in barren ears. In Ontario and Quebec, most sweet corn is pollinated before peak adult emergence. However, high numbers of adults can cause economic damage to later plantings or late-maturing cultivars. If populations are high after silking, adults will feed on the foliage, producing long, silver streaks on the lower epidermis.

Root feeding by the larvae may be severe if sweet corn is grown continuously or after field corn. The larvae feed on and damage small roots during their early instars and tunnel in the larger, brace roots during their later instars. They may make gouges or channels in the roots. Plants with a reduced root system lack vigor and may lean or lodge, especially after a rain or wind storm. With further growth, they bend or elbow upward, becoming "goose-necked." Lodging, if extensive, interferes with harvesting operations and reduces yield.

Corn rootworm adults are known vectors of stalk rot and ear rot fungi, and larvae transmit fusarium root rot fungi.

Identification Corn rootworm (family Chrysomelidae) larvae are white, thread-like, and at maturity about 1 cm long and brownish at both ends. Adults of the northern corn rootworm *(12.15a,b)* are uniformly pale or yellow-green and about 1 cm in length. The adult western corn rootworm *(12.15c)* is similar in size but slightly longer and has black and yellow, slightly wavy stripes that do not extend the entire length of the forewings (elytra). Considerable variability in color exists. The western corn rootworm adult may be confused with the striped cucumber beetle (see Cucurbits, 9.21); however, the striped cucumber beetle's black stripes have straight margins and extend the entire length of the forewings *(9.21).* The striped cucumber beetle can be observed throughout the summer whereas the western corn rootworm begins emerging in July in Ontario. Adults of the southern corn rootworm are about 12 mm in length and yellow-green with 12 large, dark spots on the elytra. (For more information on the southern corn rootworm, see Cucurbits, spotted cucumber beetle, 9.21).

The southwestern corn rootworm *Diabrotica longicornis* (Say) does not occur in Canada.

Life history Corn rootworms have one generation per year. The egg is the overwintering stage. The eggs hatch in late May to mid-June, depending on weather, and larvae migrate through the soil in search of corn roots. They feed for three to four weeks, mature about mid-June to mid-July, and leave the root zone to form pupation cells in the soil. Adult emergence from the soil begins about the first week in July in southwestern Ontario and mid-July in the east. Adult beetles tend to congregate on the corn silks where they feed and mate. From mid-August to October, the females lay eggs in clusters in the soil at depths of 5 to 20 cm, the depth being greater at the base of corn plants and among the brace roots. Adults eventually succumb to frost.

Management

Monitoring — Lodging or goose-necked plants indicate the presence of rootworms, but by then it is too late to apply insecticides. Growers should inspect their fields soon after tasseling to determine the number of root- worm adults per plant. Generally, a threshold of one western or two northern corn rootworms per plant is used to decide whether to apply a granular insecticide for rootworm control the following year or to rotate out of corn for a year.

Cultural practices — Rotation with any other crop for a year effectively prevents damage because the larvae are quite host specific on corn. The use of deeply rooting corn hybrids and adequate fertilizer will minimize losses by ensuring good plant growth.

Chemical control — Insecticides should be used where rotation is impractical or undesirable, and where numbers of rootworm adults the previous year exceeded the threshold. Excessive rainfall or high soil pH may hasten the disappearance of chemicals and limit the effectiveness of control treatments. Granular insecticides should be applied at planting with a spreader attachment that places the insecticide in a 15- cm band in front of the press wheel but not in contact with the seed. The insecticide should not be broadcast.

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(Original by M. Hudon, R.E. Pitblado and G.H. Whitfield)

▶ 12.16 European corn borer *Figs. 12.16a-h*

Ostrinia nubilalis (Hübner)

Since its introduction into southwestern Ontario in 1920, the European corn borer has spread across Canada from the Maritime provinces to the Rocky Mountains. In Alberta, although detected and eradicated in the 1950s, a well-established infestation was discovered in 1981 in the Medicine Hat-Bow Island area and has since expanded throughout the southern part of that Province.

The European corn borer has more than 200 recorded, wild and cultivated herbaceous hosts. Corn is the most important host but other vegetable crops, such as snap bean, pepper and potato, have experienced loss. In the Canadian prairies, corn seems to be the only host.

Damage Different strains of the corn borer cause different types of damage to sweet com. Larval feeding on corn ears is the primary cause of yield loss but all parts of the plant are subject to attack. The larvae eat through the tightly rolled leaves developing in the whorl. This results in the first sign of damage, a row of "pin holes" in the leaves when they unroll from the whorl *(12.16a).* As the leaves enlarge and the holes coalesce, midrib breakage may occur. Some larvae also may bore into the tassel, weakening it and increasing the likelihood of its breaking in the wind *(12.16b).* Eventually, the larvae enter the stalk and developing ears, which may lead to stalk breakage *(12.16c,d),* poor ear development and fallen ears. First-generation larvae cause mainly physiological damage to the growing plant; second-generation larvae are responsible for shank and ear damage.

The European corn borer can cause significant losses in sweet corn as well as other vegetable crops, such as pepper and snap bean. In sweet corn, infestation of the ears is the major concern, regardless of the generation of corn borer involved. Not only are infested ears and damaged shanks unsuitable for fresh-market sale but small larvae *(12.16e)* may reside in kernels of sweet corn destined for processing.

The European corn borer is a vector of shank, stalk and ear rot fungi.

Identification The European corn borer (family Pyralidae) larva is a caterpillar about 3 cm in length at maturity, and gray to tan above with brown, spot-like plates (pinacula) with setae (*12.16f*). The adult moth's wingspan is about 2.5 cm; the wings are light brown with dark wavy bands (*12.16g).* The male is smaller and darker than the female.

Life history At present, there are three strains of corn borer in Canada. A one-generation strain occurs across most of Canada, males of which respond to the Z-type pheromone blend (97:3, Z:E tetradecenyl acetate). A two- generation strain, which often has a partial third generation, occurs south of a line between Simcoe, London and Sarnia in southwestern Ontario. The partial third generation occurs about five out of every six years at Harrow, Ontario (based on the period 1971 to 1988). This strain also responds to the Z-type pheromone blend. A third strain occurs in some locations in southern Quebec. It also has two generations per year but responds to the E-type pheromone blend (96:4, E:Z tetradecenyl acetate).

One-generation strain — Overwintering fifth-instar larvae pupate in corn stalks in the spring. Adults emerge from the third week of June to the end of July and adult flight usually peaks in mid-July. Egg deposition normally peaks one week after peak adult emergence. Eggs are laid in flat masses *(12.16h)* near the midrib on the underside of leaves and hatch in five to seven days. The young borers feed in the leaf axils or the whorl and developing tassel before tunneling into the main stalk. In late summer or early autumn, the mature larvae spin a flimsy cocoon inside the corn stalk and enter a state of arrested development (diapause) that lasts until the following spring.

Two-generation strain — Pupation occurs normally two weeks earlier in the spring than for the one-generation strain, and adult flights occur from the end of May to the first week in June with peak adult emergence in mid- to late June depending on location. Eggs are laid from early June to early July, but they have been found as early as the end of May and occasionally well into July. Larval development is the same as that of the one-generation strain but usually the larvae mature before there is much corn ear development. They pupate rather than enter diapause, giving rise to a second flight of adults that begins about the first week of August and peaks two weeks later. These moths oviposit until early September and lay more eggs than the earlier moths. Their offspring, which enter diapause and mature the following spring, often cause a greater reduction of yield in grain corn. In sweet corn, they are most troublesome if they enter the ears.

Because the corn borer has a facultative response to daylength (photoperiod), both the one- and two-generation strains may have an additional generation. A complete or partial second or third generation may occur if spring temperatures are sufficiently warm to promote early emergence and completion of larval growth during the longer days of early summer. Cool, rainy weather, which restricts moth activity and retards larval development, may offset this situation. Rain also may drown or wash very young larvae off the plants. Very dry summers are unfavorable. Winter cold does not seem to be detrimental.

Moths of the European corn borer extend their range by about 12 km per year by means of flight alone. Rate of expansion was documented during the early years of the insect's presence in Ontario and Quebec, and more recently in Alberta. Dissemination also is aided by transport of larvae in fresh-market sweet corn (see Introduced diseases and pests, 3.11, European corn borer).

Management

Monitoring — The need for insecticides is determined by the stage of susceptibility of the host plant, the value of the crop, the presence of corn borers, and the proportion of plants showing damage. In Quebec and eastern Ontario, egg laying begins during the mid- and late- whorl stages, so corn should be examined twice a week thereafter to determine the need for control. The number of egg masses (*12.16h)* on corn plants is not necessarily related directly to the number of moths present. Temperature, rainfall, parasites, predators and diseases can influence oviposition success. However, the time required to sample for egg masses can be reduced if sampling is restricted to those times when corn borer adults are present and the plants are in a susceptible stage of development. A reliable and quicker method is to evaluate the percentage of plants showing any leaf feeding during the early part of the egg- laying period.

Corn borer adults can be monitored with pheromone lures in sticky traps or with black-light traps. In Quebec, growers are mailed a notice that advises them when to start monitoring. However, as a general guideline across Canada, treatment should be delayed until tassels start to appear in the whorl, unless there are signs of feeding at the late-whorl stage.

Corn for processing or fresh market is very susceptible to corn borer attack between mid-whorl and the commencement of drying of the silks. If corn borer adults are present during this time, there is a need for chemical control. This can be determined by examining the plants for signs of larval feeding on the whorl. It is also possible, although more time consuming, to examine plants throughout the field for egg masses. For the latter procedure, 20 groups of five plants are examined. Four to five egg masses per 100 plants is an indication that corn-ear infestations will exceed 10%. For many years in Quebec, 10% damaged ears has been used as an economic threshold for sweet corn for processing and 5% for fresh-market sweet com.

Cultural practices — Tillage and crop rotation are most often used to control the European corn borer. Fall plowing and spring disking can eliminate 75% of the overwintered larvae in a corn field. Shredding of plant residue after harvest before plowing is an economical and effective way to destroy corn borers in stalks and stubble. Also, larvae in infested corn used for silage are killed in the ensiling process. The practice of burning stalks in the field after harvest is still used by some growers despite its negative environmental impact.

Biological control — Parasites, predators, diseases, and birds can kill large numbers of corn borers but usually they do not reduce populations below economic levels. Commercially produced eggs of a *Trichogramma* species of parasite show potential for biocontrol.

Chemical control — In general, early sweet corn for fresh market requires protection, although the need for insecticides in any area is determined by the value of the crop and the severity of the infestation. Many insecticides are toxic to natural enemies, especially when applied from the air. Ground application is more effective because the spray can be directed into the plant whorl. Granular formulations have a longer residual effect, are less toxic to bees and natural enemies, and their timing is less critical.

Growers should consult their extension agent and the appropriate spray calendar for current recommendations and preharvest intervals. For early sweet corn for fresh market, three to four treatments are customary at five-day intervals, starting when the first larvae hatch or at the first sign of leaf damage. For processing sweet corn, one or two treatments are usually applied as needed. In Alberta, control has been achieved with only one insecticidal application around the third to fourth week of July when the larvae are second instar and abundant. In general, ground sprays should be applied by directing the flow into the plant whorl. After tassels appear, the spray should be directed into the ear zone. For aerial applications, special precautions should be taken to avoid killing bees.

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(Original by M. Hudon and D.G.R. McLeod)

▶ 12.17 Fall army worm *Figs. 12.17a-e*

Spodoptera frugiperda (J.E. Smith)

In North America, this is essentially a southern insect. However, it moves northward in the summer and occasionally reaches Canada. Outbreaks rarely occur but the insect may appear without warning wherever corn is grown in Canada. It normally is associated with cool, wet weather, which favors rapid reproduction along the route of northward migration.

Hosts include sweet corn, other garden vegetables, grasses, alfalfa *(Medicago sativa* L.), clovers and tobacco *(Nicotiana tabacum* L.).

Damage The fall armyworm has only recently become important in eastern Canada. It affects late crops of sweet corn and grain corn. When abundant, defoliation can be severe. Larvae (*12.17a*) feed mainly on the leaves. Early damage is often missed because the young larvae are deep within the whorl, giving the leaves a ragged appearance when they unfold. The tassel also may be damaged. Yield loss on immature corn normally is negligible because the plants can recover from leaf damage even if severe. Yield loss becomes progressively greater as the larvae feed on the ear shanks of more mature plants.

Identification The fall armyworm (family Noctuidae) larva is a caterpillar that resembles the armyworm in habits and appearance. When mature, it is about 4 cm in length. It varies from pale green or tan to nearly black with three pale yellow, lengthwise stripes and a darker line on each side flanked by red and yellow bands (*12.17b,c).* Its head is dark brown with an inverted, white, Y-shaped mark that distinguishes this insect from the corn earworm. The adult moth (*12.17e)* has mottled gray forewings and gray-white hindwings.

Life history The fall armyworm overwinters in the most southern part of the United States and northern Mexico. The moths fly and lay their egg masses at night. The eggs hatch in 2 to 10 days, the larvae mature in about 20 days, and pupation (*12.17d)* occurs in the soil. The moths emerge after about 10 days and often migrate before laying their eggs. In the north of its range and particularly in Canada, there is only one generation per year. When food becomes exhausted, larvae wander in search of another food supply and die with the first fall frosts.

Management

Monitoring — Growers should monitor for larvae inside the leaf whorl during August and early September. In eastern Canada, late-maturing sweet corn is subject to attack in late August or early September, regardless of the weather, and infestations usually are well advanced before they are detected.

Cultural practices — The use of early maturing cultivars is recommended.

Biological control — Parasites and predators, such as flies, wasps, ground beetles, and birds and other vertebrates, attack and kill the larval stages.

Chemical control — Growers should consult an extension agent or provincial production guide when planning a spray application. Larvae in a late stage of development inside the leaf whorl are difficult to control with insecticides. The same action thresholds as for the European corn borer (10% damaged ears for processing sweet corn and 5% for fresh-market sweet corn) also apply to the fall armyworm.

(Original by M. Hudon)

▶ 12.18 Flea beetles

Corn flea beetle *Chaetocnema pulicaria* Melsheimer
Toothed flea beetle *Chaetocnema denticulata* Illiger

These flea beetles range throughout southern Ontario. Their hosts include many grasses and sweet corn.

Damage Adults chew small, circular holes in leaves, skeletonizing and sometimes killing young corn plants. The relationship between the corn flea beetle and Stewart's wilt involves survival of the causal bacteria in the adult beetles' alimentary tract and transmission to corn plants when the beetles feed. Larvae feeding on corn roots may cause stand reduction.

Identification The adult flea beetles (family Chrysomelidae) are small and black, resembling other flea beetles. They are not easily identified to species except by specialists. The larvae are small, whitish grubs that also are difficult to identify.

Life history Adults overwinter in the surface soil of grassy areas. They emerge in early spring and search for grass and corn seedlings. They eat small holes in the tender leaves. Eggs are laid at the base of grasses, including corn. The larvae feed on fibrous grass and corn roots. Adults can be found from May to July, and from mid-August until frost forces them to seek shelter. Their populations are greatly reduced after severely cold winters.

Management

Monitoring — Growers should watch for concentrations of adults near old corn fields after a mild winter by inspecting for signs of adult feeding on young corn plants in early June.

Cultural practices — Fall plowing to bury crop residue eliminates sheltering sites and minimizes survival of the overwintering adults.

Chemical control — Some control early in the season can be expected from granular insecticides applied for corn rootworms. A foliar insecticidal treatment, if applied just after the plants emerge, is effective but seldom necessary.

(Original by R.E. Pitblado and J.A. Garland)

▶ 12.19 Four-spotted sap beetle *Figs. 12.3c, 12.19*

Glischrochilus quadrisignatus (Say)

The four-spotted sap beetle or picnic beetle is common in eastern Canada but it also occurs in British Columbia and Manitoba.

Adults are attracted to a variety of ripe or damaged fruits, feeding on the surface and often entering crevices. They are attracted to overripe fruit, such as raspberries, melons and cracked tomatoes in the field and at roadside stands. The larva develops in plant residue in the field.

Damage Sap beetles can do a great deal of damage if conditions are favorable. The adult beetles enter the tip of the ear and feed on the developing kernels *(12.3c).* Sometimes they attack undamaged ears but generally they infest ears already damaged by the corn earworm, European corn borer, birds or raccoons.

Sap beetles are known to transmit ear rot fungi.

Identification The adult four-spotted sap beetle (family Nitidulidae) is black with two yellow-red spots on each forewing ***(12.19).***

Life history Adults overwinter by hibernating under crop residue, in old tree stumps, in the upper 2.5 cm of soil where there is a cover of grass sod or tall weeds, and possibly in other protected places. There is only one generation per year. Egg laying starts in early May and the larvae develop on decaying plant matter. Newly emerged adults begin to appear from late June to early August, depending on the area.

Management

Monitoring — Inspection of corn ears in various areas of the field from silking onward should reveal the presence of adults if they are there.

Cultural practices — Clean cultivation will reduce sap beetle populations because adults overwinter in crop residue left in the field and the larvae develop on decomposing plant matter from the previous fall. The main source of decomposing plant matter in southwestern Ontario and Quebec is ears of corn that were missed by harvesting machinery, often as a result of shank or stalk damage by the European corn borer.

Resistant cultivars — Cultivars with a short or loose husk are probably more susceptible, so the growing of tight-husked cultivars might help reduce damage by this insect.

Chemical control — Chemical insecticide sprays against the European corn borer and corn earworm in sweet corn will reduce sap beetle infestations.

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(Original by M. Hudon)

▶ 12.20 Seedcorn maggot *Figs. 12.20a-c*

Delia platura (Meigen)

The seedcorn maggot (see Bean, 15B.18) can be a problem in sweet corn in eastern Canada if the seeds are germinating when the fly (*12.20c*) is laying eggs. Infestations in sweet corn are usually worse during cold, wet springs because of prolonged germination.

Damage The larvae (*12.20b*) feed inside germinating corn kernels. They often destroy the germ or allow entry of soil microorganisms and seed rots *(12.20a).*

Management

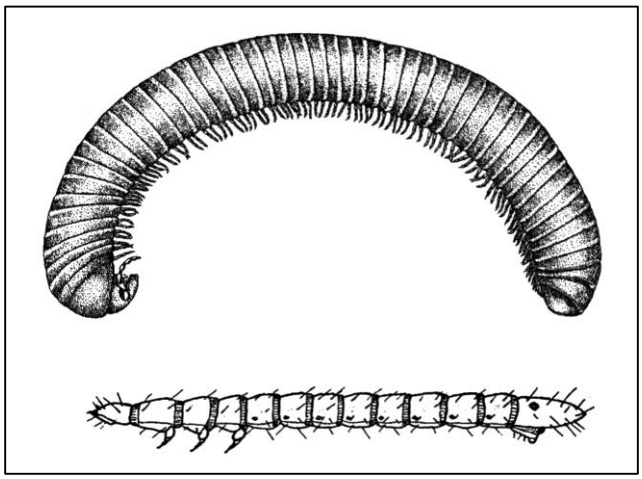
Monitoring — Areas of the field where seedlings have not emerged should be checked for damage to the seed.

Cultural practices — Although infestations are sporadic, growers should use fungicide-insecticide treated seed and follow cultural practices that hasten germination. Growers who use treated seed should have little risk of maggot damage. If untreated seed is used and an infestation occurs, it may be necessary to replant the entire field. Because the fly is attracted to soil humus and moisture, shallow planting will reduce damage, particularly if the soil surface is prepared for rapid germination. If a cover crop is grown on land intended for sweet com, growers should cultivate the field in the fall or spring in order to incorporate all organic matter thoroughly into the soil.

(Original by M. Hudon and C. Ritchot)

▶ 12.21 Wireworms *Figs. 12.21a,b,T1; 16.50*

Corn wireworm *Melanotus communis* (Gyllenhal)
Other wireworms



12.21T1 Wireworm (below): larva of *Agriotes mancus*, note legs on three thoracic segments and eye spot or "muscular impression" on rounded ninth abdominal segment, length 20-30 mm; millipede (above) has two pairs of legs on most segments, length about 25 mm. After Hawkins, J.H. 1936. *Maine Agric. Exp. Stn. Bull.* 381.

Wireworms can be found in all soil types and in all production areas of Canada, affecting the seeds and underground parts of many crops. Infestations of wireworms are widespread but easily overlooked.

Sweet corn is particularly susceptible to attack, but wireworms also can damage roots of other vegetables (see Potato (76.50); and Tomato, 18.41).

Damage Wireworm damage is more severe in crops planted on land that has recently been converted from use as a pasture or grassland. An irregular pattern of plants dying in the field is typical of wireworm damage. The larvae bore into the seeds of sweet corn and consume the germ, or they enter the underground stem and ultimately kill the plant. Symptoms are not always apparent in mature corn but, in general, infested plants do not develop well and seedlings lack vigor or fail to emerge.

Identification Wireworm (family Elateridae) adults are generally dull brown to black, elongate "click" beetles (*12.21b).* They vary from about 0.5 to 2.0 cm in length. The larvae are shiny, yellow-brown, slender, cylindrical, hard-bodied with three pairs of tiny legs near the head-end (12.21T1), and reach 2 to 3 cm in length when mature *(12.21a).* They are particularly noticeable in the spring in almost any kind of soil, often being found on the surface under moist accumulations of leaf litter or unincorporated crop residue See Table 12.21 for key to genera *Agriotes, Melanotus* and *Limonius.*

Table 12.21 Key to some common wireworms damaging vegetable crops

1. Last (ninth) abdominal segment rounded, with two obvious darkened "muscular impressions" (commonly called eye spots) .....*Agriotes* spp.

   Last (ninth) abdominal segment flattened dorsoventrally, without "muscular impressions" 2

2. Hind margin of last abdominal segment without a caudal notch .....*Melanotus* spp.

   Hind margin of last abdominal segment with a small but distinct caudal notch .....*Limonius* spp.

(Original by E.C. Becker)

Life history Wireworms, depending on the species, may take two to five years to mature. They overwinter either as larvae or adults in the soil. The adult beetles are active in the spring and lay eggs in the soil or near grass roots. Eggs are always more abundant in native or cultivated grass or legume pastures. Larvae hatch in two to four weeks and move through the soil in search of food. They move deep into the soil each fall and return to the upper soil to feed on the roots of corn plants soon after seeding in the spring. During their last year of development, the larvae form a cell in the soil in late summer in order to pupate and become adults, and these remain in the ground until the following spring.

Management (see also Potato, 16.50)

Monitoring — Areas of the field where seedlings have not emerged should be checked for damage to the seed.

Cultural practices — Shallow cultivation while the larvae are still young kills the larvae by exposing them to birds and other predators. When bringing new land into production, the ground should be plowed during the summer and corn should not be sown in the next year.

Chemical control — A fungicide-insecticide seed treatment usually is recommended to protect corn from wire- worm injury. For heavily infested fields that must be seeded to corn, granular insecticides may be broadcast and incorporated prior to seeding; however, this increases production costs markedly.

(Original by M. Hudon)

▶ 12.22 Other insect pests *Figs. 12.22a,b; see text*

Cutworms
European earwig *Forficula auricularia* L.
Grasshoppers
Potato stem borer *Hydraecia micacea* (Esper)
White grubs

Cutworms

(see Carrot, 6.25; and Tomato, 18.35) Cutworms *(18.35c-g)* on corn plants feed at their base, and some may climb to feed higher on the stem or on the foliage.

(Original by F. Meloche)

European earwig

The European earwig (*8.43b-d)* will eat the silks of sweet corn, which can result in poor development of the kernels. The presence of the earwig on marketable ears of corn is also a nuisance. (For more information about the European earwig, see Crucifers, 8.43.)

(Original by L.M. Crozier)

Grasshoppers

Grasshoppers (family Acrididae) (*12.22a*) are seldom serious pests on sweet corn in Canada, although they have been a problem in Quebec, particularly in the 1950s. When grasshoppers attack corn, they eat the tassel, tips of ears and portions of the leaves, giving the plants a ragged appearance and reducing seed set. They seldom attack corn before the plants are 50 cm high. Damage is usually confined to the margins of fields.

(Original by M. Hudon)

Potato stem borer

(see Potato, 16.47) The potato stem borer (*12.22b*) is most damaging on sweet corn at the four- to eight-leaf stage. Larvae feed in the stem and on the roots. Collapse, and stand reduction may occur around field borders.

(Original by F. Meloche)

White grubs

White grubs (*16.49c-e*) are the larvae of June beetles (see Potato, 16.49). Damage caused by white grubs on sweet corn is minor and localized, and usually appears during late July or early August; the plants appear reddish and can easily be pulled by hand.

(Original by K.P. Lim and J.C. Guppy)

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BACTERIAL DISEASES

13.1 Slippery skin *Fig. 13.1*

Pseudomonas gladioli pv. *alliicola* (Burkholder) Young, Dye & Wilkie

Slippery skin occurs sporadically in all onion-producing areas of Canada, but it is a greater problem in the southern United States and other areas with warm climates. Affected bulbs may be unmarketable, especially for processing. At harvest, there are often no external symptoms of the disease and the bulbs must be cut to detect symptoms. In Ontario, truckloads of onions have been declared unmarketable and entire fields abandoned because there is no practical method to grade out the affected bulbs. *Pseudomonas gladioli* pv. *alliicola* is not a problem on other *Allium* crops.

Symptoms In the field, onions with severe slippery skin may have one or two wilted leaves in the middle of the leaf cluster. Later, the wilted leaves turn pale yellow to off-white and die back from the tips. Older and younger leaves generally remain green. Lifted bulbs usually are soft and watery; squeezing them at the base will cause the rotted inner portions to slide out through the neck, hence the name slippery skin.

Usually, the disease progresses slowly so that affected bulbs appear sound at harvest. However, the inner scales of horizontally cut bulbs often appear water-soaked. The neck of these bulbs may become softened, after which rot spreads from the neck through the inner scales to the base *(13.1).* The pathogen can then spread throughout the bulb and into the central scales, which become watery and develop a cooked appearance.

Causal agent *Pseudomonas gladioli* pv. *alliicola* forms a diffusible, non-fluorescent, pale yellow to yellow-green pigment on nutrient-dextrose agar and King's B medium. It is a motile, Gram-negative rod that has one to several polar flagella. This bacterium is oxidative, reduces nitrates to nitrites, and will grow at 41°C, giving a weak oxidase reaction. It is able to utilize nicotinate, (+)-tartrate, (-)-tartrate, meso-tartrate, mesaconitase, gamma-aminovalerate, citraconate and laevulate, but not putresceine, glutarate, erythritol or glycollate. In one report, all isolates tested caused considerable degradation of pectate gels at pH 4.9 to 5.1 and pH 6.9 to 7.1, but not at pH 8.3 to 8.5.

Disease cycle Slippery skin occurs most frequently during seasons with high rainfall and in fields that are heavily irrigated. The disease usually appears during the last half of the growing season. The pathogen is soil-borne and is probably transferred to the leaves by splashing water. Infection of the seed stalks and leaves has been reported in Hungary and India. Not all attempts to reproduce the symptoms by re-inoculation have been successful. Plants appear to become more susceptible with age.

Wounds on the leaves may be important points of entry for the pathogen. Accumulation of water in the neck of the onion also may favor infection. After infection, the bacteria spread down the leaf to the corresponding bulb scale, then progress down the scale to the base of the bulb before spreading throughout the bulb. Infection usually takes place while the crop is actively growing, shortly before harvest, or when the onions are topped. High temperatures and slow drying of the bulbs favor infection.

The bacteria grow in the range of 5 to 41 °C, with an optimum of 30°C. In warm weather, a bulb can decay in 10 days. Infected bulbs begin to rot within four weeks when stored at 25°C, but not at 15°C. In storage, the disease may take up to three months to destroy a bulb completely.

Management

Cultural practices — Overhead irrigation should be avoided late in the season and leaf damage kept to a minimum. Onion should be harvested only when the bulbs are fully mature and when the weather is dry. Unnecessary damage at harvest should be avoided and bulbs should be quickly and thoroughly cured (see neck rot, 13.7). Only dry bulbs should be stored and the storage environment should be maintained as close as possible to 0°C and 65 to 75% relative humidity. The high temperatures associated with artificial curing may temporarily encourage rotting, but heat curing is necessary for successful longterm storage. Subsequent cold storage will halt further multiplication of the bacteria.

Resistant cultivars — Spanish onion is more susceptible to slippery skin than common onion.

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(Original by M.R. McDonald)

▶ 13.2 Soft rot (bacterial soft rot) *Figs. 13.2a,b*

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*

Soft rot can affect onion in both storage and transit, especially when proper grading or storage procedures have not been followed. Soft rot bacteria are secondary invaders but nevertheless can cause major crop losses. Soft rot affects many vegetable crops including most cultivated *Allium* species.

Symptoms When the bacteria enter onion necks through injured or dying leaves they may spread directly into one or more of the bulb scales without moving from scale to scale.

Affected scales first become spongy, water-soaked and pale yellow to light gray. As the rot develops, the scales become progressively softer and the whole interior of the onion breaks down to form a sticky mass inside the dry outer scales *(13.2a,b).* When the bulb is squeezed, it is soft and a watery. A foul-smelling liquid usually oozes out at the neck. When infection occurs at the site of an injury, the rot can progress through several scales from the infection site and the bulb will decay.

Causal agent (see Potato, bacterial soft rot, 16.2)

Disease cycle Soft rot may develop in the field, especially after heavy rains and when the leaves are drying near the end of the season. The bacteria require a wound or an infection site of another organism to gain entry to the plant. Once established, soft rot can cause far greater damage than the original injury or disease.

Soil is a major source of bacterial contamination. The pathogen survives on infested crop residues in the soil and may be transferred to neighboring plants by splashing water. Direct contact with infested soil can also result in infection. Onion maggot damage to bulbs is an important entry point for soft rot bacteria. Once the bacteria have entered a bulb, they multiply very rapidly at high temperatures. The temperature range for growth is 6 to 37°C, with the optimum between 24 and 32°C in culture and between 18 and 27°C in plants.

In the field, disease develops quickly during warm, wet weather. In storage and transit, the bacteria continue to multiply at temperatures greater than 3°C. High relative humidity and liquid water also promote the reproduction and spread of soft rot bacteria when temperatures are favorable. One infected bulb can stain several surrounding bulbs in a pallet box or bag, thereby reducing onion marketability.

Management (see onion maggot, 13.26)

Cultural practices — Disease management is based on reducing all types of injury to the bulbs and providing good storage conditions. Growers should avoid using overhead irrigation on onion, especially Spanish types, when the crop is approaching maturity. Onion should be harvested only when the bulbs are fully mature and dry. Efforts should be made to reduce sunscald, bruising and mechanical damage during harvest. Wherever possible, diseased or damaged onions, especially those with onion maggot damage, should not be put into storage. Bulbs should be stored at 0°C and 65 to 75% relative humidity. Adequate ventilation must be provided during storage so moisture is not allowed to condense on the bulbs.

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(Original by M.R. McDonald)

▶ 13.3 Sour skin *Fig. 13.3*

Pseudomonas cepacia Burkholder

Sour skin is not a major problem, but like the related disease slippery skin, it can occasionally cause significant losses. Sour skin was first described in 1950 in New York where it has become prevalent. In Ontario and Quebec, sour skin is usually less common than slippery skin. This disease affects only onion.

Symptoms Affected onion plants usually have one or two leaves that turn light brown. Later, a watery rot develops at the base of the leaf in the bulb neck. Affected leaves can easily be pulled out of the bulb. When disease is advanced, in the field or in storage, the bulb scales become yellow and viscous but not watery *(13.3).* The central scales and scales near the outside of the bulb may be infected in the absence of symptoms on the bulb. Diseased scales may separate from adjacent healthy ones. Squeezing the base of an affected bulb may push the central portion out through the neck; however, it is not watery and remains firm unlike bulbs affected by slippery skin. Secondary organisms such as yeasts are often associated with this disease and may be responsible for the acrid, vinegar-like odor from which the name "sour skin" was derived.

Causal agent *Pseudomonas cepacia* is a motile, Gram-negative rod with rounded ends. It does not form spores. It has one to three polar flagella and averages 0.8 to 1.9 µm in size. These bacteria do not form fluorescent pigments (i.e., no fluorescence at 254 nm), but they do produce a variety of yellowish and greenish pigments. These pigments may diffuse into the culture medium or remain bound to the cells. *Pseudomonas cepacia* is an obligate aerobe.

When transferred to pectate gels, strains of *P. cepacia* cause pitting at pH 4.9 to 5.1 and at 6.9 to 7.1, but not at pH 8.3 to 8.5. This reaction is the same as that of *P. gladioli* pv. *alliicola. Pseudomonas cepacia* macerates onion slices and causes the acidity of the juice to drop from pH 5.5 to about 4.0. When isolations are made from necrotic tissue of onions with sour skin, the pathogen is usually present in low numbers compared to the quantity of saprophytic bacteria present.

Disease cycle *Pseudomonas cepacia* has been isolated from organic soils and irrigation water. Overhead irrigation and splashing rain are probably the most common means by which the bacteria are spread. They do not infect unwounded plants, but enter young leaves through wounds or gain entry to bulbs when the onions are topped. High rainfall and moderately high temperatures may be necessary for infection. In New York State, the disease always occurs in conjunction with rainstorms and warm weather.

When infection of young leaves occurs, the bacteria progress downward, causing a watery rot, and then infect the corresponding bulb scales. The bacteria spread more quickly in water-soaked tissues than in those that are not congested. Infection of the bulbs may also occur at harvest through the cut surfaces created by mechanical toppers. Disease is more severe when the weather is warm and humid and the leaves have not completely dried down.

Management

Cultural practices — Methods used to reduce losses caused by sour skin are similar to those for other bacterial diseases, such as soft rot and slippery skin. Overhead irrigation has been shown to favor sour skin development, especially when applied from bulbing until harvest. Furrow or drip irrigation is less conducive to the development of sour skin. Growers should try to keep damage to the leaves at a minimum during the growing season. To avoid infection at harvest, onions should be lifted when fully mature, cured properly and stored at 0°C and 65 to 70% relative humidity.

Resistant cultivars — Spanish onion is more susceptible to sour skin than common onion.

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(Original by M.R. McDonald)

FUNGAL DISEASES

▶ 13.4 Basal rot *Fig. 13.4*

Fusarium oxysporum f. sp. *cepae* (H.N. Hans.) W.C. Snyder & H.N. Hans.

Basal rot occurs in most areas of the world where onions are grown. It has been found in only a few fields in the major onion-producing areas of Canada. Most yield losses result from disease development in the field, but basal rot can also progress in storage. The pathogen attacks only members of the genus *Allium.* Onion, garlic, shallot, chives and leek are susceptible, but the disease is economically important only on onion.

Symptoms The first symptoms on onion in the field are tip dieback and yellowing of the leaves. The disease may progress until the entire foliage is yellow and withered, which is an indication that decay has already started at the basal plate. Infected onions can be pulled easily out of the ground and often appear lopsided because only one side of the basal plate is infected. The roots decay and a firm, pinkish-brown rot develops at the base of the bulb and later progresses upward *(13.4).* Under moist conditions, white mycelium develops on the rotted area.

When infection occurs late in the crop season, symptoms are not visible until the onions are in storage. However, early signs of infection may be detected by cutting the bulb vertically and looking for a discoloration of the basal plate that begins at the outermost layer and extends upward.

Causal agent *Fusarium oxysporum* f. sp. *cepae* produces chlamy- dospores, microconidia and macroconidia, both on the host and in culture. Isolations from onion bulbs and soil indicate that the pathogen exists in soil primarily as a sporodochial type. The many *formae speciales* of *F. oxysporum* are differentiated by the ability of the specific pathogen to infect a host (see Celery, fusarium yellows, 7.5, for a description of *Fusarium oxysporum***).**

Disease cycle The fungus persists in soil in the form of chlamydospores that can be spread in water, soil or air. It can penetrate roots directly or infect roots and bulbs previously injured by other onion diseases or by onion maggots. Disease development occurs between 15 and 30°C with an optimum of 29°C. The requirement for high soil temperatures is a chief reason that this disease is not a major problem in Canada. Soil moisture levels that will support onion growth are adequate for infection and disease development.

In storage or transit, rot develops rapidly between 20 and 30°C. Below 15°C, the rate of decay is very slow but premature sprouting is still likely to occur. High relative humidity in storage promotes rotting.

Management

Cultural practices — Growers should follow a three-year rotation with non-susceptible crops, such as carrot, lettuce, celery or beet, and grow onion in well-drained soils. Disease-free sets and recommended production practices help to reduce stress on the crop. Bulbs should be cured properly before harvest (see neck rot, 13.7) and damaged or diseased bulbs should be culled before storage. Maintain storage temperature at 0°C and relative humidity at 65 to 70%.

Resistant cultivars — Many commercial onion cultivars have field tolerance to fusarium basal rot, examples being Canada Maple, Granite and Valiant. Growers should check provincial recommendations for up-to-date lists.

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(Original by M.R. McDonald)

▶ 13.5 Botrytis leaf blight *Figs. 13.5a,b*

Botrytis squamosa J.C. Walker
(teleomorph *Botryotinia squamosa* Vien.-Bourg.)

Botrytis leaf blight is one of the major foliar diseases of onion in cool climate areas. The disease has been reported in North America, Britain, Europe, Japan and New Zealand. In Canada, it ocurs annually in most areas where onions are grown. The severity of epidemics depends on local weather conditions. Botrytis leaf blight severely affects only common onion.

Levels of disease affecting less than 11% of leaf area do not decrease yield, but when disease is severe and leaves die back the bulbs may be small and fail to mature properly. Severely affected bulbs may not dry down enough for proper storage. They may also have fleshy leaf tissue at the neck rather than dry papery scales and are therefore more susceptible to storage rots. Rapid senescence of the leaves may also interfere with the application of sprout inhibitors, thus reducing the storage life of bulbs.

Symptoms The first symptom is discrete, circular to elliptical, grayish white leaf spots, about 1 by 3 mm, which later become brownish-white and desiccated. Some lesions may extend through the wall of the leaf and split open with age, exposing the inside (lacuna) of the leaf. Newly formed lesions are often surrounded by an area where the epidermis has separated from the underlying leaf tissue giving the appearance of a silvery-white "halo" with uneven margins (7*3.5a, b).* This is characteristic of *Botrytis squamosa* infection on onion. The gray mold fungus *Botrytis cinerea* may also infect onion leaves, but the resulting lesions are smaller, do not penetrate to the inside of the leaf and do not develop halos. *Botrytis aclada* (see neck rot, 13.7) can cause limited foliar spotting, but it usually remains in a latent state until the bulb is mature or the leaf has senesced. Whitish flecks and spots caused by ozone injury lack the distinct margin and silvery halo characteristic of botrytis leaf blight.

Under favorable conditions, the number of lesions on a leaf increases, the lesions expand and merge, and the leaves begin to die back. Dieback usually begins at the leaf tip and may extend down the entire leaf. The lower, older leaves are usually the first to die. Sporulation occurs on necrotic leaf tips and occasionally on large lesions. Several species of *Botrytis* are associated with neck rot symptoms; that caused by *B. squamosa* is known as small sclerotial neck rot (see neck rot, 13.7).

Causal agent Important diagnostic features of *Botrytis squamosa* include the shape of the conidiophores, which shrink back into accordionlike folds after sporulation, and the size of the obovoid to globose conidia, which are larger (9 to 18 by 14 to 24 µm) than those of the other *Botrytis* spp. found on onion. *Botrytis cinerea* has smaller conidia, 4 to 11 by 6 to 18 µm, and has long conidiophores that are darker than those of *B. squamosa. Botrytis aclada* has the smallest conidia, 4 to 8 by 6 to 16 µm.

Sclerotia of *B. squamosa* are flat, scale-like structures, 0.5 to 4 mm, white at first, but turning black with age. The sclerotia often produce tufts of conidiophores and, in certain locations, apothecia in the spring. The apothecia are stipitate, cupulate and 0.5 to 2.5 mm in diameter. Wild-type isolates are heterothallic. Microconidia are hyaline, unicellular, 2 to 3 µm in diameter, produced in chains from phialides and function as spermatia. Asci are 163 to 200 by 13.8 to 16.5 µm and contain eight ascospores ranging from 15.9 to 17.5 by 10.0 to 12.5 µm. The ascus has an iodine-positive ring.

Botrytis squamosa can be cultured from conidia or sclerotia obtained from onions. The pathogen often sporulates on infected leaves kept in a moist chamber at room temperature for a few days. Conidia will form on the lesions or on necrotic leaf tips. Conidia can then be transferred to and maintained on potato-dextrose agar. To stimulate the production of conidia in culture, transfer mycelium to the basal agar medium described by Bergquist *et al.(see* Selected references), which contains mineral salts, vitamins and dextrin (10 g/L). The plates should be incubated at 22°C and provided with a 12-hour photoperiod using a combination of cool-white fluorescent and near-ultraviolet ("black") lamps. Conidia will form in 7 to 13 days.

Disease cycle *Botrytis squamosa* is a polycyclic pathogen with the potential to cause very rapid disease development. Sclerotia overwinter in the soil, on onion debris, and on bulbs in cull piles. In the spring, these produce conidia that serve as the initial inoculum. Conidia are produced at temperatures ranging from 3 to 27°C, with maximum production at 9°C. Apothecia also have been observed on sclerotia in onion fields.

Sclerotia may survive at least 21 months when buried in organic soil, but conidia usually survive for less than three months. The mycelium of *B. squamosa* does not survive long in plant residues. Infection of seedlings from seed- borne conidia probably occurs rarely in nature.

The conidia of *B. squamosa* are released during the daytime with peaks of spore release between 0900 and 1200 hours promoted by declining relative humidity. A smaller peak may occur in the evening and large releases of spores are associated with rain showers. The conidia are dry and are dispersed in turbulent air. Disease distribution in a field is usually general rather than focal. Germination of conidia and infection require liquid water and temperatures over 6°C. The optimum conditions for infection are 12 hours of leaf wetness at 15 to 18°C. Infection is reduced above 27°C.

Management

Cultural practices — The initial inoculum can be reduced by rotation with carrot, lettuce, celery and other crops unrelated to onion. The removal of onion cull piles and the reduction of overwintering cull onions in the field also reduce inoculum levels.

Chemical control — Disease management depends largely on fungicide applications. Protectant fungicides provide adequate control when applied before infection takes place. The best control has often been achieved by applying mixtures of broad-spectrum (e.g. dithiocarbamate) and single-site-specific (e.g. imide) fungicides.

Three different systems are used in North America to forecast botrytis leaf blight and to time fungicide applications. These are the sporulation index incorporated into the PREDICTOR (also called the PESTCASTER) developed by M.L. Lacy; BOTCAST developed by J.C. Sutton *et al:,* and BLIGHT-ALERT developed by P.C. Vincelli and J.W. Lorbeer. Each of these programs predicts the need for fungicide applications by different means.

The PREDICTOR is based on vapor pressure deficit, which is determined using relative humidity and temperature over the previous 72 hours to arrive at an index that indicates the probability and intensity of spore release. This program utilizes an instrument that measures temperature and relative humidity and can also record leaf wetness, rainfall and soil temperature. It provides a read-out of the sporulation index for that day. A grower or scout checks the sporulation index each day and determines whether the risk is sufficient to warrant chemical control. Fungicides need not be applied more than once every five to seven days. This system is used in Michigan and Quebec. It can be grower-operated and does not depend on field scouting. In Quebec, good control of botrytis leaf blight has been achieved by spraying when the sporulation index is over 50.

The other two programs are based on starting the fungicide spray program when a critical disease level of one lesion per leaf is reached. BOTCAST is designed to use accumulated microclimatic data to predict when this critical level has been reached in the field, using duration of leaf wetness, temperature, relative humidity and rainfall data to provide an inoculum value (0 or 1), and temperature and leaf wetness data to provide an infection value (0, 1 or 2).

These values are multiplied to give a disease severity index for each day. Cumulative disease severity indices (CDSI) indicate two thresholds. Threshold 1 (CDSI = 2) triggers the recommendation to spray before the next rainfall. Threshold 2 (CDSI = 30-40) indicates that a fungicide should be applied as soon as possible. Once the control program has started, fungicides should be applied every 7 to 10 days. Research in Ontario has shown that using microclimatic data to lengthen the spray interval when weather is not favorable for botrytis leaf blight only saves an average of one spray.

The BLIGHT-ALERT program is designed specifically to time fungicide sprays following the first application. Under this system, the threshold of one lesion per leaf is determined by pest management scouts walking the fields and counting lesions on leaves. A sequential sampling method is used and scouts count lesions on the three oldest green (80% green) leaves on 15 to 50 plants per field. Once the one lesion/leaf threshold is reached and growers begin spraying, subsequent sprays are recommended on the eighth day after spraying if the probability of rain is greater than 30% or if the "Inoculum Production Index" is greater than 7. An index of less than 7 indicates that spore release is unlikely. Fungicide protection is considered to last for seven days. If no fungicide application is recommended for day eight, the index and probability of precipitation are checked for day nine and each day thereafter until the next spray is needed.

The Inoculum Production Index, which is based on weather conditions conducive for spore production, is calculated using average temperature, hours of relative humidity above 90% and days after planting. The index ranges from 0 to 25. Significant spore releases usually occur on days where the index is greater than 7. A probability of precipitation greater than 30% indicates that conditions will be favorable for infection if spores are released. In New York State, growers using the spray threshold and BLIGHT-ALERT program have saved two to three sprays per season.

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(Original by M.R. McDonald)

▶ 13.6 Downy mildew *Figs. 13.6a-d*

Peronospora destructor (Berk.) Casp. in Berk.

Onion downy mildew occurs sporadically in the onion-producing areas of British Columbia, Ontario and Quebec. Epidemics in onion fields are potentially explosive and destructive, given favorable weather. In some instances, the disease destroys all of the foliage in just four or five weeks. Severe downy mildew reduces bulb size and can result in bulbs being downgraded for market. Necks of diseased onions often remain succulent and difficult to cure at harvest. Downy mildew can affect dry bulb, pickling, Welsh, Egyptian and multiplier onions, as well as garlic, shallot, leek, chives and several other *Allium* species.

Symptoms The first sign of downy mildew is velvetlike growth of the pathogen on the otherwise green leaves *(13.6a,b).* Early in the morning, the fungal growth appears purplish due to pigment in the fungal spores (sporangia), which form overnight just above the leaf surface. Later, most of the spores are dispersed in the air and a whitish fungal growth remains on the leaf. During the next two to four days, the diseased leaves turn pale green, then yellow, and finally collapse and die *(13.6c).* Blackish growth

of other fungi, especially *Stemphylium botryosum* Wallr., is common on the dead leaves. Small green leaves often emerge from the cluster of dead top growth within a week or two. Necks of affected plants remain succulent. Affected areas on seed stems tend to remain yellow, but may bend over and break when the seed head enlarges. The downy mildew fungus may also invade flowers and seeds.

Causal agent *Peronospora destructor* produces aseptate mycelium, asexual sporangia borne on sporangiophores, and sexual oospores. The mycelium develops only within living host tissues. Sporangia form on sporangiophores that emerge through the stomata of green host leaves and become dichotomous with primary and secondary branches and curved sterigmata *(13.6d).* The sporangia develop in an acropetal sequence on pointed ends of the sterigmata. Mature sporangia are thin-walled, sub-hyaline, pyriform, papillate at the distal end, and measure 18 to 29 by 40 to 72 µm. They are easily detached from the sporangiophores. Oospores form intercellularly within the host tissues, usually late in the growing season, and are released after the tissues die and decompose. The oospores are thick-walled, spherical and 40 to 44 µm in diameter. Oospores survive in the soil for four or five years and germinate by a germ tube.

The pathogen is readily identified from the symptoms and from microscopic examination of sporangia and sporangiophores scraped from the surface of diseased onion leaves. Isolates of *P. destructor* may be stored conveniently for five to six months in inoculated onion sets kept at 1 to 3°C. The sets may be inoculated by injection with a spore suspension of the fungus. To recover the pathogen, the sets are planted in pots, allowed to grow until about 15 to 20 cm high, then placed in a dark, moist chamber to promote sporulation. The pathogen also may be maintained in green onion leaves, but must be transferred every two to three weeks.

Disease cycle The mildew fungus overwinters in infected onion bulbs or sets and on other host plants. In spring, it invades new leaves as they emerge. Invaded leaves usually are paler green than healthy ones. Spores produced on these leaves initiate mildew epidemics in onion and other hosts grown from seed. Infected seeds and oospores in crop residues are possible, but unlikely, sources of inoculum for mildew epidemics. The main sources of inoculum for spring-seeded crops are volunteer onion plants, onion cull piles, onion seed crops, fall-seeded onion plants and perennial onion plants grown in home gardens.

Only a few spores are needed to initiate a destructive epidemic in an onion field. After infection, the fungus grows extensively within the leaf, which remains symptomless until the fungus sporulates. Generations of spores (sporangia) are produced every 10 to 16 days. Disease increase is stepwise, each step coinciding with sporulation and subsequent collapse of the leaves. Successive steps usually increase in size. Given favorable weather, most of the foliage is destroyed after three to four cycles of sporulation and infection, which can occur over a period of about 34 to 45 days.

The relationship of weather and downy mildew is well established. The fungus sporulates on heavily colonized onion leaves between midnight and sunrise. Weather conditions required for spore production at night are: relatively low to moderate temperatures during the preceding day (average of less than 24°C between 0800 and 2000 hours), temperatures between 4° and 24°C at night, continuous high relative humidity (about 95%) between midnight and sunrise, and no rain after 0100 hours.

High daytime temperature and short or interrupted humid periods at night commonly prevent sporulation. Under favorable conditions, as many as 100000 or more spores are produced on each square centimetre of leaf. The spores mature near dawn and are released into the air mainly during the morning. A few spores may be released before the dew dries, but most are dispersed as the relative humidity declines. Some of the spores deposited on onion leaves survive and infect the leaves during the first or second night after dispersal. The spores germinate and infect the leaves within three to six hours when dew is present, except when dew deposition is slow or erratic or temperatures are near freezing or above 26°C.

Management

Cultural practices — Cull onions should be destroyed and not left in piles near onion fields. It is very important not to grow winter onion and other host crops in fields and home gardens near summer production fields. Seed onion should be grown far away from any bulb crops.

Chemical control — Foliar fungicides suppress downy mildew effectively when spray applications are suitably timed. Fungicides should be used when mildew is present in the area and when weather favors disease increase. The downy mildew forecaster DOWNCAST may be used to identify periods when weather favors sporulation and infection by the mildew fungus. DOWNCAST requires weather monitoring in the field and indicates optimal times to look for early signs of mildew and to apply fungicide sprays. Protectant fungicides are effective only when applied before infection.

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(Original by J.C. Sutton)

▶ 13.7 Neck rots *Figs. 13.7a-d*

Neck rot (gray mold neck rot)
Botrytis aclada Fresen.
(syn. *Botrytis allii* Munn)

Mycelial neck rot
Botrytis byssoidea J.C. Walker
(teleomorph *Botryotinia allii* (Sawada) Y. Yamamoto)

Small sclerotial neck rot
Botrytis squamosa J.C. Walker
(teleomorph *Botryotinia squamosa* Vien.-Bourg.)

Neck rot is one of the most common storage diseases of onion and also can occur on shallot, leek, garlic and chives. Disease incidences of up to 50% of the bulbs in storage have been reported. Additional losses can result from secondary infections of bacterial soft rot.

Symptoms Neck rot is most commonly found on bulbs after harvest, although in wet seasons it may appear before harvest. The first symptom is the softening of the affected neck scale tissue, which takes on a sunken, cooked appearance. There is a definite margin between the diseased and healthy tissue, which remains distinct even when the rot is advancing toward the base of the bulb *(13.7a).* As the lesions age, the tissue becomes grayish and later a dense, grayish *(13.7b),* cottony growth of mycelium appears on the surface of the scales. This growth gives rise to a gray- brown powdery mass of spores that can spread the fungus for considerable distances through the air. Still later, small, whitish, kernel-like sclerotia, less than 3 mm in length, appear in the mycelium and soon turn black and hard *(13.7c).* Symptoms usually appear first in the neck *(13.7d)* and spread downward to the base of the bulb, but symptoms may develop first around the basal plate at the base of the bulb and progress toward the neck. Eventually the whole bulb may become dry and mummified, but it can still serve as a source of inoculum.

Causal agent Neck rot is caused chiefly by two closely related species *Botrytis aclada* and *B. byssoidea*. A third species, *Botrytis squamosa*, also infects white onion (see botrytis leaf blight, 13.5) and causes a disease known as small sclerotial neck rot.

Botrytis aclada, the most common species in Ontario, may produce a dense mat of conidiophores near the sclerotia on infected bulbs. Neck rot caused by *B. aclada* is sometimes referred to as gray mold neck rot, but this can be confused with the name "gray mold," which usually refers to diseases caused by *Botrytis cinerea*, which often can be isolated from onion leaves and bulbs.

Botrytis byssoidea infection of onion, sometimes called mycelial neck rot, can be distinguished from gray mold neck rot by the presence of more surface mycelium and sparse sporulation.

Botrytis conidiophores are straight, alternately branched and may proliferate. The conidia are usually one-celled, gray to brown, globose to ovoid, and are botryoblastospores. The teleomorph of *B. aclada* has not been identified.

Both *B. byssoidea* and *B. aclada* can be cultured from mycelium, conidia or sclerotia aseptically transferred from infected bulbs to potato-dextrose agar. In culture, *B. byssoidea* produces few conidia and they are larger, 5 to 11 by 8 to 20 µm, than those produced by *B. aclada,* which measure 4 to 8 by 6 to 16 µm. *Botrytis aclada* conidia sometimes overlap the size range of *B. cinerea* conidia (see Lettuce, gray mold, 11.10).

Disease cycle The sclerotia that form on infected bulbs serve as the primary overwintering structures, and in some soils and in cull piles, they may survive for several years. The sclerotia germinate under suitably moist conditions to produce successive crops of conidia that spread on air currents to neighboring onion fields as well as to those some distance away. In Britain, *B. aclada* was shown to survive on debris in a sandy loam soil for two years, but sclerotia failed to germinate and produce conidia after only six months in soil. In Canada and the northeastern United States, sclerotia in cull piles and on onion debris and unharvested bulbs in the field are the main source of initial inoculum.

Botrytis aclada is also seed-borne and seed lots have been found with as much as 20% infestation. Direct seedling infection has been observed with mycelium from the seed coat penetrating the tip of the cotyledon leaf. However, infected seed lots do not appear to be a major source of neck rot in North America. Virtually all of the onion seed planted in Canada is treated with a systemic fungicide to control onion smut and this chemical also provides some control of seed-borne *B. aclada.*

The air-borne spores of *B. aclada* can infect onion leaves during periods of cool (15 to 20°C), wet weather, but symptoms may not be visible. Either the fungus can grow within the leaf without causing symptoms or it remains quiescent in the epidermis and grows only when the leaf starts to senesce. Conidiophores are produced only on senescent or necrotic leaf tissue. The pathogen can spread rapidly during wet growing seasons and can spread downwind from a diseased to a healthy crop. Cool, wet weather during the ripening and harvest periods also can increase the incidence of neck rot.

Infection can take place during harvest if the bulbs are bruised or damaged. Onions with thick necks or broken outer scales may be infected through these sites, especially if the relative humidity in storage is high. Neck rot rarely spreads from bulb to

bulb. It may become more severe the longer onions are stored because bulbs that were infected, but symptomless when harvested, eventually develop the disease.

Management

Cultural practices — The elimination of onion cull piles and crop rotations of at least two years with carrot, beet, corn and other crops unrelated to onion help to reduce the overwintering inoculum. Crop rotation is most effective where onion fields are widely separated from each other. Proper curing and storage of mature bulbs is the most important means of reducing neck rot. Onions with thick necks, nicks or bruises should be graded out before storage. Onions for long-term storage should have two or three layers of dry outer scales and the neck should be narrow and composed of papery-dry scales.

Onion bulbs should be prepared for storage by maturing or "curing" them. This usually is done by leaving the lifted bulbs in windrows until the necks are thoroughly dry. Onions produced in humid areas should be cured artificially by passing dry outside air through piles of onions in storage for two days. The air should be heated sufficiently to reduce the relative humidity to 60% for 14 days or until the onion skins turn a rich golden brown. Once onions are cured thoroughly, storage conditions should be maintained just above 0°C at a relative humidity of 65 to 75%.

Resistant cultivars — Most commercial onion cultivars grown in Canada are susceptible to neck rot, especially white-skinned common and Spanish onions. Red-skinned onion and cultivars that are strongly pungent are less susceptible to infection.

Chemical control — Foliar fungicides are registered in Canada, but the level of control achieved is usually low.

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(Original by M.R. McDonald and W.R. Jarvis)

▶ 13.8 Pink root *Figs. 13.8a, b*

Phoma terrestris E.M. Hans.
(syn. *Pyrenochaeta terrestris* (E.M. Hans.) Gorenz, J.C. Walker & R.H. Larson)

Pink root is common but usually of minor importance on *Allium* crops in Canada. It also occurs in the United States, Australia, Europe and South Africa. Losses are difficult to document because only bulb size is reduced by this disease. The pathogen is a common soil inhabitant and can infect the roots of common and multiplier onion, shallot, garlic, leek, chives, and many other monocotyledonous and dicotyledonous crops.

Symptoms Symptoms of pink root are frequently seen on onion roots. The disease is recognized most easily by the distinctive, dark pink to maroon color of infected roots *(13.8a).* Infected roots are also partially to totally collapsed and later turn reddish brown as the root dies. In contrast, healthy roots are cylindrical and white. Diseased roots break off easily when the bulb is pulled from the soil. When disease is light and growing conditions are favorable, onion bulbs may have only two or three pink roots and no above-ground symptoms. Under these conditions, no reduction in yield has been observed and bulb sizes are similar to those of uninfected plants. Plants with severe pink root are stunted, have a high proportion of infected roots, are easily pulled from the soil, and fail to produce marketable bulbs. When disease is moderate, the leaves die back from the tip, turning yellow-white or yellow-red. The color is more reddish than when the leaves die from environmental stress. Infected plants rarely die back completely *(13.8b);* they usually remain stunted with only a small area of green leaf tissue remaining near the neck of the bulb.

The disease is not usually observed on seedling onion but can become noticeable by mid- to late season, often showing first when the plants begin to form bulbs. The pathogen is widely distributed within fields but diseased plants often appear in patches, either near the headlands, where the soil is shallow, or on knolls or over drainage tiles where the soil is poorer or drier. Often, the disease appears so late in the season that it has the beneficial effect of reducing moisture uptake by the plant, thereby allowing the bulbs to dry and mature in preparation for harvest.

Causal agent Pycnidia of *Phoma terrestris* form singly within infected roots and later burst through the surface. The pycnidia are globose, dark brown to black, up to 400 pm in diameter, and darkly pigmented around the ostiole. There are usually a number of brown setae, 60 to 180 µm long, around the ostiole. Conidia form inside the pycnidia and are unicellular, hyaline, ovoid to allantoid with rounded ends, and have a guttule at each end.

Isolation of the pathogen is often difficult because fast-growing *Fusarium* species frequently co-inhabit pink root-affected plants. *Fusarium* species do not cause pink root symptoms on their own. To isolate *P. terrestris,* plate symptomatic, surface-sterilized onion roots onto water agar and incubate at 20°C with a 12-hour photoperiod for four to five days. Discard all cultures producing *Fusarium* conidia. Subculture suspected isolates of *P. terrestris* to plates of cornmeal agar amended with 500 ppm of

chloramphenicol and incubate for 10 to 14 days at 24°C. Once again, discard any *Fusarium* cultures. The remaining cultures will be *P. terrestris,* which grows slowly on this medium; the colonies will be pinkish, circular, appressed and have smooth margins.

Disease cycle *Phoma terrestris* is a soil inhabitant that can survive and multiply indefinitely in soil. It is only weakly pathogenic and attacks the roots of plants that are under stress or that have previously been injured. On onion roots inoculated with conidia, symptoms can be seen within 7 to 21 days. The hyphae penetrate the roots and grow through the cortical tissue; pycnidia form in both the epidermal and cortical cells. The pycnidia erupt through the epidermal tissue and release the conidia into the soil.

The fungus can develop at all soil moisture levels that will support the growth of onion. The optimum temperature for infection is 26°C. In organic soils, temperatures this high usually are associated with dry conditions. High levels of soil moisture moderate the soil temperature. Yield losses usually occur when onion is stressed by high temperature and dry soil conditions. This makes it difficult to determine the proportion of yield loss caused by the disease versus that due to environmental stress.

The fungus is spread by infested onion sets and soil. Machinery, dust storms and surface run-off can move soil from one area to another and spread the pathogen. However, the fungus is often so widespread that it is futile to try to prevent its spread from one field to another.

Management

Cultural practices — Long rotations with unrelated crops, such as carrot, lettuce, celery and beet, are suggested. However, cereals, grasses, parsnip, radish and spinach are not recommended for rotations because the pathogen can infect their roots. When onion is grown in infested soil, adequate moisture, fertilizer and pest control encourage vigorous crop growth. Healthy plants are able to tolerate the disease by producing abundant roots.

Resistant cultivars — Leek and chives are resistant to pink root. Several of the early Japanese hybrid onion cultivars suffer from a combination of high temperature and moisture stress at bulbing and may become heavily infected. Many commercial onion cultivars have some tolerance to pink root, examples being Capable, Paragon, Copper King and Granite.

Chemical control — Soil fumigation is not economical, though it will reduce the population of *P. terrestris* and may result in increased onion yields. Fumigation is more effective on sandy soils.

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(Original by M.R. McDonald)

▶ 13.9 Purple blotch *Figs. 13.9a,b; 13.5b*

Alternaria porri (Ellis) Cif.

Purple blotch is a common onion disease that can be very destructive. It is usually secondary, affecting leaves that already have been attacked by other pathogens or by abiotic factors such as hail or ozone. *Alternaria porri* also can infect leek, shallot, Egyptian and Welsh onion, and false shallot.

Symptoms Lesions are initially small, whitish, sunken and elongate *(13.9a);* some may have purplish centers *(13.5b).* Under favorable conditions, the lesions soon expand to become large, oval, purple blotches with concentric rings *(13.9b).* The blotches may merge and become covered with the dark brown spores of the fungus. Diseased leaves eventually die. Older leaves are more susceptible to purple blotch than are younger leaves.

Purple blotch can cause heavy losses in onion seed crops by infecting and destroying the flower stem. The fungus also may infect the onion bulb at harvest through wounds or fleshy neck tissue. In storage, *A. porri* causes a dark yellow or deep-red spongy bulb rot.

Causal agent Conidiophores of *Alternaria porri* form singly or in clusters on diseased leaves. They are pale to medium brown, 10 to 15 pm thick, up to 120 pm long, and may have one to several well-defined conidial scars. Conidia are smooth or minutely verrucose, pale to golden brown, and are borne singly. When they detach, they leave a distinct scar. The conidia are straight or slightly curved, 100 to 300 µm long, 15 to 20 µm thick, and club-shaped with a long beak. The beak is often as long as the main body of the conidium, and is flexible and tapered. The conidia have 8 to 12 transverse septa and none to several longitudinal or oblique septa. Each cell is capable of germination. A teleomorph is not known.

Disease cycle The mycelium and conidia of *A. porri* overwinter on infested onion residues in the field or in cull piles. Conidia are produced in the spring and disperse by wind or splashing water to onion leaves. Liquid water is necessary for the conidia to germinate. Germination can occur in 45 to 60 minutes at 28 to 36°C. Penetration can take place directly through the uninjured epidermis or through wounds or stomata. Lesions may appear one to four days after penetration and conidia are produced shortly thereafter. Long periods of leaf wetness favor infection. Almost no infection occurs at temperatures below 13°C. In culture, the

fungus grows between 6 and 34°C. The optimum for sporulation is 25°C at 90% relative humidity. On calm days, the maximum number of conidia is trapped between 0800 and 1400 hours. Wind, rain, irrigation and spraying may promote spore release. A forecasting system to predict spore release is being developed at Michigan State University.

Management

Cultural practices — To reduce initial inoculum, residues from infected crops should be destroyed and onions should be grown in rotation with non-host crops, such as carrot, celery, lettuce or potato. Culled onions should not be piled near onion fields. To prevent disease development in storage, onions should be harvested during dry weather when the bulbs are fully mature and the tops are dry. Bulbs should be stored at 0°C and 65 to 75% relative humidity.

Resistant cultivars — Yellow cooking onion is not as susceptible as Spanish onion.

Chemical control — Broad-spectrum protective fungicides applied to onion foliage prior to spore deposition provide effective control of purple blotch.

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(Original by M.R. McDonald)

▶ 13.10 Smudge *Fig. 13.10*

Colletotrichum circinans (Berk.) Voglino
(syn. *Colletotrichum dematium* f. sp. *circinans* (Berk.) Arx)

Smudge occurs mainly on white-skinned common and Spanish onion and has been observed sporadically in Canada. It also has been reported on shallot and leek. Infection is mostly superficial, but the formation of black fruiting bodies of the pathogen on the outer scales gives the onions a dirty or "smudged" appearance. Infected bulbs are downgraded or rejected for sale.

Symptoms Smudge can appear on maturing bulbs in the field, usually just before harvest, and on bulbs in storage. The small fruiting bodies of the fungus are dark green at first but turn black when mature. They characteristically occur in groups or in concentric rings near the neck and on the outer scales of the bulb *(13.10).* The black fruiting bodies are covered with stiff black bristles that can be seen with the aid of a hand lens.

When infected onions are stored under moist conditions, small yellow lesions form on the inner layers of the bulb. These enlarge and coalesce and the bulb may shrivel and sprout prematurely. The pathogen can cause damping-off in moist soils and leaf spotting during warm weather.

Causal agent *Colletotrichum circinans* produces septate mycelium that is hyaline at first but becomes darker and thicker with age. Stromata are formed from the thick-walled hyphae, and saucer-shaped acervuli develop from the stromata within the host cuticle. These acervuli contain hyaline conidiophores and numerous dark setae that are triseptate and 80 to 315 µm long. Eventually, the acervulus ruptures the host cuticle and releases the conidia, which bud off the conidiophores individually. Conidia are fusiform, nonseptate, hyaline to pale yellow, and slightly curved with a rounded top. They measure 3 to 4 by 18 to 18 µm. Under moist conditions, they appear as a creamy-colored, gelatinous mass on the dark acervuli. A sexual state is unknown.

Disease cycle *Colletotrichum circinans* can survive in soil for several years, either as a saprophyte or as stromata. The fungus can also be introduced into fields when infected bulbs are planted.

Warm, wet weather is conducive to disease development and spread. High relative humidity is essential for the formation of conidia. They are spread most often by splashing rain, but also can be dispersed by wind and on clothing. The optimum temperature for the germination of conidia is 20°C. No infection occurs below 5°C. The optimum for mycelial growth and symptom development is 26°C. Conidia require free moisture in order to germinate. Germinating conidia produce one to three germ tubes, which develop appressoria on the surface of the plant. An infection hypha pushes through the cuticle by mechanical pressure, and the fungus becomes established between the cuticle and the epidermis. Enzymatic degradation of the host tissue allows further colonization to take place. The fungus can complete an infection cycle within a few days under warm moist conditions.

Management

Cultural practices — It is critical to use disease-free seed and sets. Proper curing of white-skinned onions is essential. Growers should use artificial curing methods if the weather is rainy or very humid. Dry onions should be stored at 0°C and a relative humidity of 65 to 70%. Crop rotation and good drainage also may help to reduce disease incidence.

Resistant cultivars — Yellow- and red-skinned onion cultivars are resistant to smudge because phenolic compounds present in the outer scales inhibit spore germination. White-skinned types are susceptible.

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(Original by M R. McDonald)

▶ 13.11 Smut *Figs. 13.11a-c*

Urocystis magica Pass, in Thüm.
(syn. *Urocystis cepulae* Frost)
(syn. *Urocystis colchici* var. *cepulae* Cooke)

Onion smut has a worldwide distribution and is a major disease of onion in Canada. It occurs in every region where onion is produced, except on newly cleared land. Stand reductions of 50 to 80% have been reported where onion is grown in infested fields and the seed had not been treated with a systemic fungicide.

Urocystis magica infects only *Allium* species. Common and Welsh onion, leek and shallot are very susceptible, whereas garlic and wild leek *(A. tricoccum* Ait.) are only moderately susceptible. Several *Allium* species are resistant to smut, including Winterbeck onion, common and Siberian chives, certain ornamental *Allium* species, and the wild species *A. altaicum* Pall., *A. obliquum* L., *A. nitans* L., and *A. ramosum* L.

Symptoms The first symptoms of smut occur on the cotyledon (flag leaf) and first true leaf of the onion seedling *(13.11a).* A thickening and darkening of these leaves is visible as soon as they emerge from the soil. Long, dark blisters or pustules containing powdery black spores of the smut fungus form within the leaves. The leaves may become bloated and distorted as they grow and often split open, releasing the spores.

Smut infection may kill the seedlings during the first three to four weeks. Sometimes, however, the pathogen remains isolated in the first leaf and, when this leaf dies, the plant continues to grow free of infection. In most instances, however, when the seedling is not killed, the smut fungus continues to grow with the plant, colonizing it systemically. The pathogen progressively invades the leaf bases and bulb scales of successive leaves. Some of these plants die during the growing season, while others survive and produce bulbs with the characteristic elongated dark pustules *(13.11b,c).* Pustules on onions in the field and in storage may be invaded by secondary organisms that can cause soft rot.

Causal agent *Urocystis magica* is heterothallic and produces multi- celled teliospores. The distinctive teliospores are an important taxonomic feature of this pathogen. Each teliospore consists of a central, smooth, spherical to ellipsoidal, thick-walled cell that is dark brown and 12 to 15 µm in diameter. The central cell is surrounded by numerous colorless, thin-walled cells that are 4 to 6 pm in diameter. Only the central cell is capable of germination.

The teliospores develop from the terminal cells of sporogenous hyphal branches. The mature teliospore is diploid and produces a short basidium when it germinates. No basidiospores are formed; instead, the mycelium formed from the basidium fragments to produce new thalli.

The teliospores of *U. magica* will germinate on potato-dextrose agar, but mycelial growth following germination is very slow. The fungus also grows on Dow and Lacy culture medium, which contains malt extract, peptone and inorganic salts. The optimal pH is 5.5 to 6.5. After storage under refrigeration in sterile water for three or four months, the teliospores will germinate readily once transferred to the culture medium.

Disease cycle The teliospores of *Urocystis magica* can persist in soil for 15 years and are the primary overwintering and infective form of this fungus. The mycelium does not play an important role in persistence or infection. The teliospores must undergo a resting phase before germination can occur. Spore germination may be stimulated by root exudates from *Allium* species. However, if germination occurs when no hosts are present, the mycelium can derive nutrients from organic matter in the soil. The seedling is susceptible to infection from about the second day after germination until the first true leaf emerges, a period of about 12 to 15 days, depending on weather conditions. The spore germinates by means of a germ tube, which directly penetrates the epidermis of the cotyledon without forming an appressorium. Infection takes place before the leaves emerge from the soil. Each new onion leaf goes through a growth phase during which it is susceptible to infection. However, developing onion leaves remain enclosed within the preceding leaf until they emerge above ground, so if the preceding leaf has not been infected, the succeeding one will not become infected. Thus, if the cotyledon and first true leaf emerge uninfected, the plant is then resistant to infection. There are two mating types of this pathogen; teliospores of both must be present for infection to take place.

Smut spores germinate at 13 to 22°C. The optimum range for germination on agar is 20 to 24°C. Soil temperatures of 29°C completely inhibit fungal growth. Low soil temperatures result in increased infection levels by slowing seedling emergence,

thereby lengthening the time that the seedling is susceptible to infection. Deep planting of onion seed can also result in increased infection levels by delaying seedling emergence. Soil moisture levels have no direct effect on the germination of teliospores or infection, but high soil moisture can result in lower soil temperatures and delayed seedling emergence which, in turn, can increase the incidence of infection.

The pathogen can be spread in infested soil and water and in infected plant parts. In the field, the smut pustules burst and release the teliospores, recontaminating the surrounding soil. Onion smut was most likely introduced into the oniongrowing regions of Ontario and Quebec through infected onion sets. There are no reports of seed transmission.

Management

Cultural practices — Growers should avoid contaminating smut-free fields with infested soil or crop residues. If onion sets are to be grown, they should be closely inspected and any that are diseased should be discarded. Smut-free sets or seedling onions for transplanting are immune to infection and can safely be grown in smut- infested fields.

Resistant cultivars — Some success in breeding for resistance has been achieved by hybridizing the common onion with the Welsh onion, but the commercial cultivars grown in Canada are very susceptible to smut.

Chemical control — Where onion is grown from seed, seed dressing with a suitable systemic fungicide is effective in protecting seedlings. The fungicide can be applied in the coating process by commercial seed companies or to raw seed by individual growers. When applying fungicide to raw seed, the use of a sticker, such as 1 % methyl cellulose solution, increases the retention of the fungicide on the seed. A drench or granular application of non-systemic fungicide into the open seed furrow at planting is not as effective as a seed dressing.

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(Original by M.R. McDonald)

▶ 13.12 White rot *Figs. 13.12a-e*

Sclerotium cepivorum Berk.

White rot is a very destructive disease of common onion and related species. The pathogen is widespread in Canada and cannot be controlled by quarantine measures. It occurs sporadically in British Columbia, Manitoba, southern Ontario and eastern Quebec. In southern Ontario, several onion fields in the Bradford and Thedford marshes are infested, and surveys between 1962 and 1968 recorded the presence of white rot in the muck soils south of Montreal.

Sclerotium cepivorum attacks edible *Allium* species, including onion, garlic, leek, shallot and chives. Wild and ornamental species are susceptible to varying degrees; *Allium aflatunense* Fedtsch. and *A. stipitatum* Regel, are ornamental species that are highly resistant to the pathogen.

Symptoms The first above-ground symptoms, which usually are yellowing and dieback of leaf tips, can progress down the leaf blades until the affected leaves finally collapse *(13.12a).* However, these symptoms are not diagnostic and can be confused with onion maggot damage.

Identification of white rot in the field is mainly by visual examination of roots and bulbs. The characteristic symptoms are white fluffy mycelium and soft rot around the base of the bulbs *(13.12b).* Masses of tiny black sclerotia form on the mycelium and in infected bulb tissues *(13.12c).* In advanced stages of infection, diseased plants are easily pulled because the bulbs and roots are destroyed *(13.12d).*

When infection occurs late in the crop season, the disease may not be noticed at harvest time and symptoms may appear only in storage. The pathogen may also cause preemergence death of seedlings, a stage of the disease that is not always detected.

Causal agent *Sclerotium cepivorum* has no sexual state. The only known survival structures of this pathogen are sclerotia, which are formed on diseased bulbs. These structures are black, spherical, hard, and 0.2 to 0.5 mm in diameter (*13.12e*). They can remain dormant in the soil for several years in the absence of host plants. Sclerotia are held dormant by the soil microflora, since they germinate readily in sterile soil. Their germination in the field is triggered specifically by organic sulphur compounds exuded by the roots of *Allium* species. In mineral soil, sclerotia have been reported to survive for up to 10 years. In contrast, in organic soil in the Fraser Valley of British Columbia, substantial decay of sclerotia occurs in less than a year. This rapid decay is attributed to environmental factors such as prolonged periods in winter when soils remain saturated. The fungus occasionally forms small secondary sclerotia, either within the parent sclerotium before germination or outside of it from germinating hyphae. The quantity of secondary sclerotia formed in soil and their pathogenicity are unknown.

Sclerotium cepivorum may also produce conidia on sporodochia. These spores seem to be sterile and appear to play no part in the disease cycle. The fungus does not grow in soil as a saprophyte. Mycelial growth in the soil is negligible unless the fungus is sustained from a foodbase, such as a sclerotium or an infected bulb.

Disease cycle Primary infections originate from sclerotia in soil, which germinate any time after onion is seeded. The optimum temperature for germination is between 10 and 18°C. The fungus usually penetrates the host through the roots. It also can infect wounded bulbs, but there is little evidence that it enters unwounded leaves or bulbs. Once the fungus has penetrated the host, the most favorable temperature range for disease development is 10 to 20°C. Above 20°C, there is a marked reduction in infection. The disease is favored by moderate moisture levels (-45 mbars to -3 bars). While mycelial growth of *S. cepivorum* in soil is restricted, secondary spread from plant to plant within rows may occur, but this requires close proximity of roots and stem bases. White rot often develops in patches in the field due to the non-uniform distribution of sclerotia in the soil. In contaminated fields, there usually are areas of high, low or no inoculum. Areas of high concentration are found where infected bulbs were left to decay after harvest. The movement of infested soil by wind, water or equipment can spread the fungus within or between fields. The use of infected onion sets or transplants can introduce the pathogen to non-infested areas. White rot occurs in a variety of soils ranging from light sands to heavy clays with acid to alkaline reactions.

Management

Cultural practices — Good sanitation is essential to avoid an increase in sclerotial populations in infested fields and also to prevent the spread of the fungus to non-infested fields. Recommended practices include the use of clean machinery, tools and pallet boxes, irrigation water free of sclerotia, and healthy onion sets or transplants, together with the removal of infected onions from the field, and the prevention of field-to-field movement of flood water carrying crop debris and soil.

In fields where white rot has developed, a four- to five- year rotation with crops other than *Allium* species is suggested. However, even after four to five years, there may still be viable sclerotia in the soil, and precautions should be taken to prevent an increase in inoculum concentration by using extended rotations and other control measures such as roguing.

In organic soil, flooding for four weeks during the spring promotes sclerotial decay. Flooding is more effective when used in combination with crop rotation because aged sclerotia (two to three years old) are more susceptible than younger ones to decay during flooding.

Biological control — The antagonistic bacterium *Bacillus subtilis* (Ehrenberg) Cohen is a potentially effective biocontrol agent for *Sclerotium cepivorum* but is not yet commercially available in Canada.

Chemical control — Erratic results may occur with the use of fungicides, especially in organic soil. Fungicides registered as pre-planting treatments, when applied according to label recommendations, often do not provide satisfactory control. This may be due to soil adsorption, leaching and microbial degradation.

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(Original by E. Banks)

VIRAL AND VIRAL-LIKE DISEASES

▶ 13.13 Aster yellows *Fig. 13.13*

Aster yellows mycoplasma-like organism

Aster yellows is a widespread disease that affects a large number of cultivated and wild plants, including vegetables such as carrot, lettuce, celery and onion. Onion is less often affected than carrot or lettuce. Onion seed crops can suffer much greater damage than bulb crops.

Symptoms On seeded onion, the symptoms begin with a yellowing at the base of the youngest leaves that spreads toward the top *(13.13).* The leaves then flatten and become marked with yellow and green streaks, although they do not twist. Seed crop plants show yellowing and abnormal elongation of the flower stem and pedicels, causing malformation of the floral cluster and sterility of the umbels.

Causal agent (see Lettuce, aster yellows, 11.15**)**

Disease cycle (see Lettuce, aster yellows)

Management Growers should control leafhopper populations.

Cultural practices — Biennial and perennial weeds on headlands and ditchbanks should be destroyed because the mycoplasma-like organism may overwinter in them.

(Original by R. Crête)

▶ 13.14 Viral diseases *Fig. 13.14*

Garlic mosaic (yellow streak) virus
Leek yellow stripe virus
Onion yellow dwarf virus
Shallot latent virus
Tobacco mosaic virus
Tomato black ring virus

Onion yellow dwarf occurs in several countries, but it is not widespread in Canada because it affects mainly onion seed crops and sets, which are not commonly grown in this country. It also occurs on shallot, garlic, leek, and certain species of narcissus. The disease reduces the bulb yields of onion grown from seed if the infections occur in early spring, and it may lower seed production of onion seed crops by up to 50%. Diseased bulbs keep poorly in storage.

Onion yellow dwarf has been known since the 1920s. The mucilaginous character of *Allium* leaf sap has made characterization of viruses from these crops difficult, so there has been a tendency to associate most viruses of *Allium* spp. with onion yellow dwarf virus. Recent work reveals at least two additional viruses: leek yellow stripe virus, which has been definitely identified only in Europe, and shallot latent virus. In most regions, garlic often harbors more than one virus. The relationship of garlic viruses to other *Allium* viruses remains unclear.

Symptoms Virus infections cause streaking or striping of monocotyledonous leaves; such symptoms are more or less analogous to virus-caused "mosaics" in dicotyledonous species.

Garlic mosaic (yellow streak) —

Symptoms *(13.14)* are generally similar to those of onion yellow dwarf, although leaves may show more curling and distortion. Garlic bulbs often fail to differentiate properly into cloves and produce only small onion-like "rounds."

Leek yellow stripe virus —

This virus causes striping of the entire leaf, most apparent at the leaf base, and general stunting. Infected plants are more sensitive to frost damage.

Onion yellow dwarf —

Leaf symptoms generally appear as pale green, chlorotic streaks or stripes that develop into variable amounts of yellowing, ranging from irregular striping to nearly complete yellowing of leaves. Infected leaves also may curl downward, appear limp or wilted, and show flattening or crinkling. Infected plants are usually stunted. Bulbs from infected plants store poorly and sprout prematurely.

Shallot latent virus —

This virus is widespread but causes no apparent symptoms in shallot.

Tomato black ring virus and tobacco mosaic virus —

These viruses are reported occasionally in *Allium* crops. Both are soil-borne. Symptoms are fairly characteristic when infections by these potyviruses are encountered. They also typically cause fibrous or granular inclusion bodies to form in leaf epidermal cells, often associated with the nucleus, that can be detected with a light microscope. Electron microscopy is required for positive identification of virus particles. Serological procedures are generally not available because relatively few laboratories have produced antisera against *Allium* viruses.

Causal agents Onion yellow dwarf virus is a member of the potyvirus group. The virus particles are flexuous filaments, approximately 15 to 16 nm in diameter and 700 to 800 nm long. Leek yellow stripe virus is also a potyvirus, approximately 820 nm long. Shallot latent virus is a member of the carlavirus group and its particles are straight to slightly curved filaments approximately 650 nm long.

There has been considerable confusion regarding the number and identity of viruses infecting garlic. Garlic "mosaic" (various regions), "yellow stripe" (California), and "yellow streak" (New Zealand) are usually attributed to potyviruses, but other workers have associated symptoms in garlic with a carlavirus. Both a potyvirus and a carlavirus with straighter, shorter particles have been found in several lines of infected garlic in Canada and also in California; most garlic probably contains a "complex" of more than one virus.

Disease cycle Most of the important viruses infecting *Allium* crops have a narrow host range and are restricted to these crops. Reports that onion yellow dwarf virus can be seed-borne have not been verified. The main sources of infection are other *Allium* species grown nearby, overwintering volunteer plants, and vegetatively propagated material of the crop species. In Europe, leek yellow stripe virus becomes prevalent only in areas of year-round leek production.

In addition to transmission through the use of vegetatively propagated material, the *Allium* viruses are transmitted in a non-persistent manner by various aphids. Onion yellow dwarf virus is transmitted by several aphid species that migrate through onion crops but do not colonize them. The shallot aphid can transmit both onion yellow dwarf virus and shallot latent virus among stored bulbs and sprouts. Leek yellow stripe is transmitted in Europe by the black bean aphid and the green peach aphid. Garlic viruses are also aphid-transmitted.

Management Aphid vectors should be controlled with registered insecticides where populations warrant.

Cultural practices — Most control measures against viruses center on prevention or avoidance strategies. Growers should plant onion sets and mother bulbs in disease-free areas and remove any volunteer onion plants. Early removal of infected plants and early harvest of planting stock also should be practiced. In some countries, certified planting stocks are produced some distance from crops grown for consumption. Crops grown from seed are less likely to have virus problems, particularly if isolated from other crops.

Much of the imported garlic sold as planting material is of variable quality. Growers are advised to obtain planting material from an experienced producer. Efforts to obtain "virus-free" garlic by *in vitro* meristem tip culture have been made in various countries. In California, garlic was freed of a symptom-causing virus in this manner, but not of a latent virus. The relationship between garlic virus(es) causing mosaic and onion yellow dwarf virus remains unclear. Some investigators maintain that onion yellow dwarf virus is not very infectious in garlic and that garlic "mosaic" is not very damaging to onion, while others disagree. Viruses have been transmitted from garlic to onion in mechanical inoculation trials. It is likely that aphids can also transmit from garlic to onion, so it would be wise to grow onions some distance from garlic, as the latter is usually infected.

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(Original by J.F. Peterson)

NON-INFECTIOUS DISEASES

▶ 13.15 Herbicide injury *Figs. 13.15a-c*

Herbicides can cause temporary or permanent injury to ***Allium*** crops. Damage can occur when these products are applied at excessive rates, during unfavorable weather conditions, or at the wrong stage of growth. With contact herbicides, burns and necrotic spots are produced (*13.15a*) and leaf tips may wither (*13.15b,c*); with translocated herbicides, the plants may turn yellow. Generally, the plants recover and the symptoms disappear over time. However, if the application rate is too high, the damage will be irreversible. Damage may also be observed if *Allium* crops are grown where the previous year's crop was treated with a residual herbicide to which they are susceptible.

Management

Cultural practices — To avoid injury, growers should follow recommendations concerning rates, volume of water, and growth stage for proper application. Records of weather conditions and stage of the crop when applying herbicides are useful in case of injury.

Chemical control — Growers should tank-mix herbicides only with recommended spreader-stickers and other pesticides specified on the label. Excessive wetting of onion foliage with unsuitable mixtures can lead to severe crop injury. Identifying herbicides that were used on the crop that previously grown in the field may help to pinpoint the cause of a chemical injury.

(Original by R. Crête and M.R. McDonald)

▶ 13.16 Ozone injury *Fig. 13.16*

Ozone injury occurs sporadically in the major onion-producing areas of Ontario and Quebec. Ozone alone causes increased leaf necrosis and reduced bulb size, but the injury is also of concern because it can predispose leaves to botrytis leaf blight and purple blotch.

Symptoms Ozone injury first appears as minute whitish flecks on the onion leaves, especially between the veins. If there are large numbers of flecks, they may coalesce to form whitish spots of varying sizes and shapes with diffuse margins *(13.16).* The flecks do not penetrate to the inside of the leaf but larger spots may. Ozone injury can be distinguished from the symptoms of botrytis leaf blight by the lack of distinct margins and silvery halos. Both ozone injury and botrytis lesions may be present on the leaf at the same time (see also tipburn, 13.19).

When ozone injury is severe, the leaves become pale green to yellowish overall and die back from the tips

(13.16). Damage is usually apparent one to three days after an ozone episode, which occurs in conjunction with warm, hazy, humid weather when air pollution levels are high. Ozone levels of 15 parts per hundred million in the ambient air are considered major episodes and usually cause flecking on onion leaves. Generally, the youngest and the oldest leaves are more resistant to ozone injury.

Causal agent Ozone is a powerful oxidant that exists naturally at very low levels in ambient air. Ozone can be produced by the action of light on air pollutants such as hydrocarbons and nitrous oxide, which are formed when fuel is burned in internal combustion engines. Ozone can also be produced during electrical storms. The levels of ozone generated as a result of air pollution can be high enough to cause injury to onion leaves and to the leaves of other plants.

Management Other than locating onion fields in areas of low air pollution, no control measures are available.

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(Original by M.R. McDonald)

▶ 13.17 Sprout inhibitor injury *Fig. 13.17*

Applying the sprout inhibitor maleic hydrazide too early in the growing season results in spongy bulbs of poor quality. Maleic hydrazide is the only sprout inhibitor registered for use on onion in Canada and it is used widely on crops destined for long-term storage. Without the application of a sprout inhibitor, onions will begin to sprout after three to four months in storage, even under ideal storage conditions. Onion bulbs that are starting to sprout become soft, even before the green leaves emerge at the neck, and are unmarketable.

Symptoms Onions that have received maleic hydrazide too early in the season may be full size at harvest but will feel soft and spongy when squeezed. Sponginess occurs because the rings in the bulb have separated from each other *(13.17)* and are not pressed tightly to the adjacent rings as they are normally. The spaces between the rings are most obvious near the neck. Maleic hydrazide inhibits sprouting by suppressing cell division. If applied before the cells in the bulb have stopped growing and dividing, cell division will be halted but the cells will continue to grow larger, causing the bulb scales to change shape and pull away from one another.

Management

Cultural practices — Correct timing of maleic hydrazide application based on the growth stage of the onion is the key to achieving effective sprout inhibition while avoiding injury. Maleic hydrazide should be applied when the onion has stopped producing new leaves. When this occurs, the neck becomes hollow and weak, and the top lodges as the bulb matures. The ideal time to apply maleic hydrazide is when 50% of the plants in the field have lodged, but the plants still have an average of five to eight green leaves. If the onion plant does not have at least five fairly healthy green leaves, the uptake and translocation of maleic hydrazide will not be sufficient to provide good sprout inhibition.

For maximum effectiveness, maleic hydrazide should be sprayed when temperatures are in the range 10 to 24°C and the relative humidity is high. Low humidity may cause maleic hydrazide to crystallize on the leaves, thereby inhibiting uptake. Rain within 24 hours after application will also reduce uptake.

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(Original by M.R. McDonald)

▶ 13.18 Sunscald

Sunscald is a minor disorder in onion crops. On a hot sunny day during warm, dry springs, particularly on dark organic soils, the temperature at the soil surface may rise to as high as 65°C. The heat damages the sensitive young plants, killing the cells at the neck level. Injured tissues shrivel, strangling the neck, and the plant wilts and withers. The seriousness of the damage depends on the temperature at the soil surface and the tenderness of the plant tissues.

The only way to avoid sunscald is to seed as early as possible so that the plants outgrow the sensitive stage before the soil temperatures become too hot.

(Original by R. Crête)

▶ 13.19 Tipburn (tip dieback) *Fig. 13.19*

Tipburn is a physiological disorder that affects mainly onions. It is a widespread phenomenon in onion-growing areas and has been reported on other monocotyledenous crops. However, it is not related to tipburn of cole crops and lettuce, which is associated with reduced calcium uptake.

Symptoms Tipburn usually starts at the tips of the oldest leaves. These turn yellow and then white because of the loss of chlorophyll. Eventually the affected leaf tissue dies. Tipburn may extend down the entire leaf and affect several leaves on a plant. In most cases, the symptoms appear at the same time over an entire field *(13.19)* or in all the plantings of a particular cultivar growing in the same region. Tipburn most often occurs when the plant is starting to bulb and nutrients are being translocated from leaf to bulb, but it can also occur earlier in the growth of the plant. Onion cul- tivars have been observed to differ in their susceptibility to tipburn. The tipburn symptoms that occur when onions are bulbing often appear quickly in a field and may be mistaken for the symptoms of herbicide injury or botrytis leaf blight. Ozone injury also may be confused with tipburn.

Causal agent The cause of tipburn is not well defined. In general, any factor that puts the plant under stress is likely to result in this disorder. Drought and saturated soil have both been implicated.

Management

Cultural practices — Balanced fertilization and constant moisture levels help to reduce stress.

Resistant cultivars — Some cultivars are commonly afflicted with tipburn and should be avoided if the problem is serious.

Chemical control — Fungicides may help to protect senescent leaf tissues from diseases such as botrytis leaf blight, purple blotch and blackstalk rot (*Stemphylium botryosum* Wahr.).

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Coumin, D. 1957. Onion tip-burn. Pages 3-25 *in Ohio Res. Bull.* **7**98.

(Original by M.R. McDonald)

▶ 13.20 Translucent scale *Fig. 13.20*

This disorder is occasionally seen on onion crops, most often on Spanish onion.

Symptoms This disorder is characterized by a grayish, watery appearance of one or more layers of scales, which makes them look translucent *(13.20).* Sometimes the condition affects all of the layers, but most often only the second and third fleshy scales exhibit symptoms. The affected scales are brownish in cross section.

Causal agent The cause of translucent scale is unknown. The problem seems to be connected with storage, because it appears after harvest and worsens after three or four months of storage. Onions kept at 5 to 10°C for a few weeks before final storage at 0°C are more subject to this disorder. Excessive relative humidity could also predispose onions to this problem.

Management

Cultural practices — Growers should heat-cure onion bulbs well, then store them at 0°C and a relative humidity of 65 to 70%.

(Original by R. Crête)

▶ 13.21 Wind, hail, pelting rain injury *Fig. 13.21*

Allium crops are very prone to wind damage at the seedling stage. Young leaves can be desiccated by hot, dry winds or sandblasted by drifting soil, especially in exposed field locations. The force of raindrops in a heavy storm may also injure leaves, causing whitish spots with irregular margins *(13.21).* These spots can develop quickly, often within 24 hours of the rainfall, and they usually occur on the side of the leaf that was facing the wind. The impact of hailstones may cause similar spots or may cut or shred the leaves, depending on the size of the stones and the intensity of the storm.

Wind and hail can seriously damage *Allium* crops and may necessitate replanting. Pelting rain usually does not cause enough harm to reduce yield, but the spots may be confused with botrytis leaf blight or herbicide injury. The injury may also make the plants more susceptible to infection by fungal pathogens by allowing nutrients to leak out onto the leaf surface.

Management

Cultural practices — To protect seedling onions from wind injury, growers should plant a cereal crop, such as barley, on the same day that the onions are seeded. The barley may be seeded in rows (one row of barley for every four to eight rows of onions) at a rate of 40 barley seeds per metre of row. Even better protection is afforded by broadcast seeding the barley at a rate

of 50 to 80 kg/ha. Once the onion plants are established, the barley can be killed with a selective herbicide. Timing of the herbicide application is critical. If the barley is seeded in rows, it should be sprayed when the plants are approximately 15 cm tall. If broadcast seeded, the barley should be killed when it is 10 to 14 cm high.

Chemical control — The application of a broad-spectrum fungicide may help to protect the injured leaves from subsequent infection by fungal pathogens.

(Original by M.R. McDonald and R.J. Howard)

NEMATODE PESTS

▶ 13.22 Northern root-knot nematode *Fig. 6.20*

Meloidogyne hapla Chitwood

Bulb crops are usually quite sensitive to infestations of this nematode.

Symptoms include conspicuous yellowing, stunting and early senescence. Prolific branching of rootlets, and production of small, spherical galls on roots are characteristic *(6.20).* Bulbs are not invaded. Onion sustains less damage than carrot under light infestations. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 13.23 Root-lesion nematode *Fig. 13.23*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. Bulb size may be reduced *(13.23).* For a complete description, see Potato, 16.38; see also Management of nematode pests, 3.12.

▶ 13.24 Stem and bulb nematode *Fig. 13.24*

Ditylenchus dipsaci (Kühn) Filipjev

This nematode attacks mainly onion and allied crops. The onion strain that is found in Canada has three races; these affect alfalfa, narcissus and onion, respectively. It has been confirmed from Newfoundland, Quebec, Ontario, Saskatchewan and British Columbia. The main crops of concern are chives, garlic, leek, onion and pea. Other strains found in Europe but not in Canada parasitize bean, beet, carrot, lettuce, parsnip, potato and spinach.

Symptoms Plants are infected during or shortly after germination. Seedling bases become swollen and leaves appear twisted and malformed. Leaves yellow and die. Young roots and bulbs may rot. Plants that do not die have badly deformed bulbs and shorter leaves that senesce prematurely *(13.24).* Bulbs are very susceptible to secondary infection by fungi and bacteria. Bulb scales are discolored and bloated. Malformed bulbs may sprout or double. A slight infection in the field may pass unnoticed, but the nematode can multiply in stored bulbs if they are not kept at low temperatures.

Identification *Ditylenchus dipsaci* (order Tylenchida, family Anguinidae) has a delicate labial framework, a thin short stylet with small knobs that are difficult to see under a microscope, and a rounded median bulb with large valves. The adult female has one, very long, anterior ovary and a long slender tail.

This nematode can survive extremely dry conditions for years. Large aggregates of desiccated nematodes can be found in dried, infested plant residue. In the dormant state, they are also able to survive freezing temperatures. Many strains of *D. dipsaci* are recognized and cannot be separated on the basis of morphology. Each strain is more or less specialized with respect to its pathogenicity to a restricted number of hosts.

Nematode extraction from soil, leaf or bulb samples and microscopic examination are needed to diagnose this species.

Life history Nematodes migrate toward the seedling roots and penetrate stems at the soil level, or they may migrate up the stem and infect young leaves. They feed on parenchyma cells and, in the process, release digestive enzymes containing pectinases and cellulases. These chemicals dissolve the middle lamellae between cells and digest the tissues around the nematodes. Small cavities are formed where the mature females lay their eggs. The nematodes reinfest the soil when infested leaves and bulbs are left in the field. In the soil, the fourth-stage juveniles can become quiescent and, as such, are able to survive extreme temperatures and dry conditions for several years. Stem and bulb nematodes are spread with infected plants, seeds and soil.

Management

Cultural practices — Rotation to non- *Allium* vegetables for at least three years, while also avoiding legumes, should reduce numbers of infective juveniles adequately. Where infestation of soil is confirmed, cull bulbs and crop residue should be removed from the plot completely; this may be more practical for home gardens and small field plantings. Growers should avoid buying or

planting onion sets from contaminated sources; infected sets are often discolored (dark brown) and lighter in weight. Questionable sets should be diagnosed or discarded.

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(Original by T.C. Vrain)

INSECT PESTS

▶ 13.25 Onion bulb fly *Fig. 13.25*

Eumerus strigatus (Fallén)

The onion bulb fly was introduced from Europe and is sometimes called the lesser bulb fly or small narcissus fly. It is a minor pest of dry bulb onion grown from sets, bulbs or multipliers and, most importantly, of onion grown from bulbs for seed production throughout western Canada.

The onion bulb fly also commonly infests bulbs of amaryllis, hyacinth, iris, narcissus and shallot. There are anecdotal accounts of onion bulb flies infesting carrot, parsnip and potato. While there is no doubt that healthy daffodil bulbs are attacked, most other plants are subject to attack only when decaying from some other cause.

Damage A single, withered leaf growing from a bulb usually indicates that the bulb is infested with onion bulb fly larvae. Bulbs infested with onion bulb fly may contain as many as 10 to 30 larvae. After harvest, infested bulbs are soft to the touch and soon become a rotting mass in storage. The onion bulb fly has little or no economic impact on commercial production of dry bulb onion crops grown from seed. Damage is restricted to onion plants growing from sets, bulbs or multipliers already damaged by other causes. Seedling onion crops are rarely, if ever, attacked and chemical controls for more important pests maintain populations at low levels. However, the onion bulb fly can be a serious problem for home gardeners who grow onion from bulbs, sets or multipliers, and for commercial growers of onion seed who grow onion from bulbs.

Identification Onion bulb fly (family Syrphidae) has the typical fly life stages: egg, larva, pupa (puparium) and adult. Adults are hover flies, about 8 mm long, and black-green with several white, lunate markings on an almost hairless abdomen (*13.25).* They resemble beneficial species of hover flies (see Beneficial insects, mites and pathogens, 3.7). The eggs are chalk-white, more elongate than oval, and less than 1.5 mm long. The legless larvae are gray-yellow with a markedly wrinkled surface. The maximum larval length is about 1.3 cm. Pupae are similar in color to pupae of the onion maggot, but they are more heavily sclerotized, about 1.5 times larger, and blunter at the ends.

Life history The onion bulb fly overwinters as a larva in soil down to depths of 8 cm but more commonly in infested onion bulbs left in the field. In the spring the mature larva crawls out of the bulb to the soil surface and usually pupates at the base of the developing bulb stem. The adults from overwintered larvae appear in late spring and early summer. They are most active on warm sunny days, feeding for about a week on pollen and nectar from the blossoms of numerous plants and sap from rotten onions before laying eggs. Eggs are laid on leaves near the stem collar, in leaf axils, and on the ground around damaged onion bulbs. Females may lay 40 or more eggs in their lifetime and it is not unusual to find 10 or more eggs on a single bulb. The structural formation of the larval mouthparts prevents them from feeding on healthy, intact bulbs. Thus, only onions damaged from some other cause are infested with onion bulb flies. Larval entry usually occurs at the basal plate. There can be up to two summer-generations of onion bulb fly each year. However, some slow-feeding larvae may require up to two years to develop. The first generation of adults is active during August.

Management

Monitoring — Monitoring procedures and thresholds specific to the onion bulb fly have not been developed. However, the flies are attracted to a number of food baits, particularly decomposing oatmeal, which stimulate oviposition. Such baits may be useful in monitoring adult activity.

Cultural practices — Growers should plant onion in areas where there is air movement (but see wind injury, 13.21); soil-borne diseases, especially basal rot, should be controlled. Also, it is best to discard or otherwise destroy all soft and rotting bulbs, and to lift and destroy all transplanted bulbs that fail to grow. Bulbs that are soft should not be planted. Dormant bulbs can be immersed in hot water for one hour at 40°C before planting. Cultivation too close to the bulbs should be avoided, as injury provides sites for flies to oviposit.

Biological control — There is little if any documentation of the role of natural enemies in the control of onion bulb fly.

Chemical control — No insecticides are registered specifically for control of onion bulb fly in Canada, but many of the insecticides recommended for control of onion maggot and onion thrips will control onion bulb fly, which is not known to be resistant to any of the currently used insecticides.

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(Original by G.J.R. Judd)

▶ 13.26 Onion maggot *Figs. 13.26a-e*

Delia antiqua (Meigen)

The onion maggot is the most serious insect pest of onion in temperate regions. It was introduced into eastern North America from Europe around 1875 and is now present throughout all commercial onion-growing areas of Canada.

Onion is the preferred host of the onion maggot. Related crops, such as bunching onion, chives, garlic, leek and shallot are occasionally infested. Onion maggot flies will oviposit on wild *Allium* species in a laboratory, but none of the wild onions growing in North America or Europe has become important as a host.

Damage The greatest economic damage to commercial onion is caused by first-generation larvae in the spring, when the plants are small. The larvae can destroy 20 to 30 onion seedlings in the loop stage, because they readily move between adjacent plants. Also, because females lay eggs in batches, damage appears clumped within onion beds. Damage from the first-generation larval attack usually can be seen by early June in British Columbia or by mid- to late June in eastern Canada. Above-ground damage symptoms depend on the growth stage of the plants. When damage occurs at the loop stage or earlier, onion may simply wilt and disappear. Plants that are attacked in the two- to three-leaf stage develop a gray cast, wilt, turn pale green to yellow, and usually remain in place within the row (*13.26e*). When these wilted plants are pulled, they often break just below the soil surface, exposing the feeding maggot inside the rotting stem *(13.26a).* Onion plants attacked in late June or early July are not killed and above-ground symptoms are difficult to detect. Fewer plants are damaged at this time because maggots no longer migrate between onion bulbs. However, plants damaged at mid-sea- son will have misshapen bulbs that often are secondarily infected with fungi and bacteria. Damage from later generations of larvae causes little economic loss to growers because most onions will already have been lifted for curing in windrows in the field by the time the females are ovipositing. Eggs are often laid on windrowed onions or in the surrounding soil but very few maggots enter healthy, undamaged bulbs at that time of year. Annual losses to commercial onion crops average about 2 to *5%* across Canada, despite heavy use of costly insecticides. In the absence of insecticidal treatments, average yearly losses to onion maggot would be in the order of 40 to 45% in commercial fields and could reach 100% in small plots or home gardens.

Identification The onion maggot (family Anthomyiidae) has an egg, larval, pupal (puparium) and adult stage. The adult is a pale gray fly (*13.26b*) that resembles the common house fly but is smaller (6 mm long) and has longer legs. Eggs are about 1 to 1.5 mm long and are white, with a striated surface (chorion). Larvae are legless, cream-colored maggots that taper toward the anterior end, with a pair of black mouthhooks (*13.26c).* Fully grown larvae are about 6 to 8 mm long. Pupae (puparia) are 5 to 7 mm long, chestnut-brown (*13.26d),* and resemble grains of wheat.

Life history The onion maggot overwinters as a pupa, usually in the top 15 cm of soil. When spring soil temperatures rise above 4°C, overwintering pupae begin to develop. Precise timing of adult emergence in the spring depends on the distribution of pupae in the soil and the temperatures to which they are exposed. Emergence usually begins when 300 degree-days above 4°C have accumulated after March 1; this occurs from late April to early May in coastal British Columbia. In Ontario and Quebec, adults may not emerge until mid- to late May. The adults disperse randomly until they are reproductively mature, when they fly upwind in response to onion odors. Although adults are capable of dispersing several kilometres, many remain within a few hundred metres of their emergence sites, sometimes aggregating and feeding on flowering weeds surrounding last season's onion fields.

The adults mate after they are five to seven days old. Mating is thought to occur in or near onion fields, because both males and females become responsive to the odor of onion when they are reproductively mature. Once having located onion fields, many adults remain in or near the field borders. Adults of later generations, which emerge in onion fields during the summer, disperse very little.

Egg laying begins about three to four days after mating, or 7 to 10 days after emergence. Each female fly may live about 30 days and lay up to 200 eggs. Eggs are laid in batches just below the soil surface around the stem of onion plants. They hatch in a few days, and the young larvae crawl toward onion roots where they feed. Mature larvae leave the onion plants and pupate in the soil.

There can be up to three generations of onion maggot each year. Seasonal phenology of the generations varies among growing areas, largely because of differences in temperature. In Ontario and Quebec, adults from overwintered pupae are present from mid-May to late June, first- generation adults from early July until early August, and second-generation adults from late August until early October. These periods are at least three to four weeks earlier in British Columbia.

Management (See also control of soil-borne pathogens, especially those that cause onion smut, 13.11, and basal rot, 13.4.)

Monitoring — Onion maggot adults can be monitored with a variety of traps, depending on local preferences and requirements. White sticky traps are used in British Columbia, both yellow sticky traps and interception traps are commonly used

in Ontario, while in Quebec cone traps baited with a volatile onion derivative, dipropyl disulfide, are used. Sticky traps are best used in areas with little wind, otherwise they quickly become covered with dust, which makes them ineffective. For most onion-growing areas, action thresholds based on trap catches are impractical, because of extremely high endemic populations of onion maggot. Instead, trap catches are used to time sprays during peak population activity. In British Columbia, where onion maggot populations are several times lower than in Ontario, an action threshold of one fly per 10 traps per day, using a 300 cm^2 white sticky trap, provides the basis for timing foliar sprays.

Cultural practices — Prevention and good sanitation are extremely important in controlling the onion maggot. Both second- and third-generation female adults prefer to lay their eggs on diseased or damaged onion plants and larval survival is higher on such plants. Ineffective treatments against first-generation maggots result in greater onion maggot injury at harvest, as does mechanical damage from cultivation or damage due to onion smut. Because damaged onion bulbs are the major food source for late-summer larvae, which in turn become the overwintering pupae, every effort should be made to minimize mechanical damage to onions, especially during uprooting for drying and harvest. Onions uprooted by a potato digger, and then windrowed, sustain less damage than those that are uprooted by undercutting. Removal of cull onions from the field in the fall, before onion maggot adults lay eggs, will reduce the next year's populations. Cull onions should not be disced in the fall until after the last summer-generation of flies has had its flight-period. Discing promotes larval survival by increasing the number of sites available for larval entry into onions.

Biological control — Several parasites, predators and diseases of the onion maggot life-stages have been identified but most are relatively ineffective in chemically treated commercial plantings. Ground beetles, primarily *Bembidion* spp., are important predators on eggs of the onion maggot, while the rove beetle *Aleochara bilineata* (Gyllenhal) attacks onion maggot eggs in its adult stage and onion maggot pupae in its larval stage. Also, the fly *Coenosia tigrina* (Fabricius) attacks onion maggot adults. Perhaps the most important agent against larvae of the onion maggot is a parasitic wasp, *Aphaereta pallipes* (Say). The fungus *Entomophthora muscae* (Cohn) is the most important agent against the adult, and it is the only natural agent that has a substantial impact on onion maggot populations during the production season; however, its activity is limited to a great extent by fungicides used to control pathogens. Despite documented evidence that *E. muscae* is a major mortality factor of onion maggot adults, very little is known about this fungus as a biocontrol agent in managed onion fields.

Chemical control — Proper application of a granular insecticide at seeding is the most important feature of onion maggot control with pesticides. Granular insecticides ensure good control of first- and very often second-generation larvae, provided that the dosage is correct and the insecticide is placed in the furrow on top of the seed at planting time. Very often, chemical control of third-generation larvae is unnecessary, especially in British Columbia and Quebec, where populations of this generation tend to be extremely low and where onions are often in the process of being harvested when oviposition begins. Foliar insecticides are effective only if applied on the foliage, not on the soil. Late-season control may be accomplished by applying foliar sprays early in the evening or just before dusk, directed against the adults. Sprays should be applied during population peaks on the basis of trap catches or degree-day accumulations. Because degree-day estimates for emergence of the adults vary among areas, growers should consult extension agents for the proper timing of spray treatments.

In some areas of Canada, particularly southwestern Ontario, the onion maggot has developed resistance to some organophosphorus and carbamate insecticides. Although resistance to these materials has not been documented in other areas of Canada, degradation of carbofuran (a carbamate) by soil microorganisms has reduced the effectiveness of this granular, furrow-incorporated insecticide in British Columbia.

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(Original by G.J.R. Judd)

▶ 13.27 Onion thrips *Fig. 22.35c*

Thrips tabaci Lindeman

The onion thrips is found in diverse habitats and on a variety of plants. It is common in all commercial onion-growing areas of Canada.

The onion thrips is an extremely polyphagous species, infesting forage and vegetable crops as well as numerous weeds. Onion thrips has been collected from most *Allium* crops (see hosts of the onion maggot, 13.26), as well as bean, cole crops, cucumber, pea, pepper, squash and tomato.

Damage Onion thrips feed on onion leaves by piercing the plant tissue with their mouthparts and sucking up the plant juices. This feeding behavior causes silver streaks to appear on the leaves. As the feeding areas coalesce, the silver streaks develop into white patches. If the infestation is severe, the leaves die back from the tips and become distorted. In hot, dry summers, the whole crop may have a "blasted" appearance. Plants may ripen prematurely and produce smaller bulbs, or they may die. Damage is less likely to be serious in cool, moist seasons and infestations are often reduced by a drenching rain. Although thrips rarely destroy a crop, reductions in yield are fairly common. Very often the grower will not recognize the extent or the cause of these yield reductions. In most years and in most areas of Canada, onion thrips is not considered as important a pest of onions as onion maggot. However, onion thrips will likely become a greater threat to onion production under reduced onion maggot spray programs, especially in arid regions and in dry years. While there is a negative relationship between feeding by onion thrips and yield in dry-bulb onion crops, the economic impact of this insect on Canadian onion production has not been established.

Onion thrips has been shown to transmit several plant pathogens, including tomato spotted wilt virus and the causal agent of powdery mildew on other crops, but no diseases of *Allium* crops are known to be transmitted by this thrips. However, onion thrips is one of many thrips suspected of predisposing plants to infection by pathogenic bacteria and fungi as a result of their feeding punctures.

Identification Onion thrips (family Thripidae) adults (*22.35c*) are pale yellow to brown, 1 to 1.2 mm long and slenderly elongate. They have four narrow wings fringed with delicate hairs (setae). The eggs are too small to be seen without magnification, but they can be seen by clearing leaf tissue with alcohol washes, when they appear as translucent, oval structures just beneath the leaf epidermis. The immature, nymphal stages, sometimes referred to as larvae, are pale yellow and resemble wingless adults. Propupae and pupae are rarely seen because they develop in the soil, but they can be distinguished from nymphs by the presence of wing buds. *[Note:* The genus *Thrips* is characterized by laterally paired "combs" on abdominal segments five to eight and antennae with seven or eight divisions (annuli). Identification should be confirmed by a specialist. — Eds.]

Life history The onion thrips overwinters as an adult in a variety of habitats and on various plants. Overwintering females are often found on onion plants or refuse left in the field after harvest, on onion bulbs in storage, or in standing alfalfa and winter wheat crops and weedy vegetation surrounding onion fields. In some areas of Canada a small overwintering reservoir of thrips is maintained on greenhouse crops. As the weather warms in the spring, females lay their eggs in leaf tissue of onion or other host plants. Very often, onion thrips will reproduce on other favored hosts before migrating into onion fields. Infestations usually begin at the field borders and gradually spread in the direction of the prevailing wind through the rest of the crop. Initially, thrips nymphs stay in clusters at the base of the plant, where the leaves are close together. As the nymphs mature, they move over the leaves, feeding like adults by rasping the plant tissue with their mouthparts and sucking up the plant juices. Mature nymphs enter the soil to pupate. The rate of development, and the duration of various stages are temperature dependent. In Michigan, the developmental threshold is 7.4°C for the entire life cycle, from egg-hatch to adult. The same threshold probably is applicable to thrips populations in Canada. The mean development-time from egg-hatch to adult at 20°C is about 14 days. In some areas of Canada, there can be several generations per season.

Thrips are among the weakest of flying insects, yet their finely fringed wings enable them to remain air-borne long enough for the wind to carry them great distances. Airborne dispersal from breeding sites is a regular event in the life cycle of the onion thrips. Many times, onion thrips will invade onion crops when infested adjacent hosts, such as alfalfa or weeds, are removed.

Management

Monitoring — Onion thrips populations are best sampled by removing all leaves from 10 to 20 plants at ground level and washing or shaking them over a suitable trapping surface in the laboratory. Adult onion thrips also can be trapped with yellow, white or blue sticky traps. Color is probably irrelevant because no relationship is known between trap catches and population levels of thrips or their damage on plants. Monitoring programs being developed are based on counts of thrips on plants in the field. There is a negative relationship between the seasonal mean number of thrips per plant and onion yield, yet there is no established spray threshold for onion thrips. However, as few as five thrips per plant per season can have a significant impact on yield. In Quebec, a mean of three thrips, either as nymphs or adults, per onion leaf in a random sample of 20 plants per five hectares of onion is a tentative spray threshold. Sampling is done twice a week in Quebec.

Fields should be sampled at least once per week during hot, dry weather, particularly during mid- to late season and following nearby harvest of hosts.

Cultural practices — Prevention and sanitation will help to reduce damage. Onion should be grown as far as possible from fields of alfalfa, wheat and other crops that may harbor onion thrips because harvesting of those crops before onion will result in invasion of nearby onion crops by migrating thrips populations. Heavy irrigation will often reduce thrips populations, eliminating the need for chemical control. In the fall, overwintering sites, such as tops of onion plants and cull bulbs, should be buried. In the spring, grasslands and headlands bordering onion fields should be cultivated to kill overwintered onion thrips by burying them.

Biological control — The onion thrips has several parasites, predators and pathogens, but there is little documentation of their effect on onion thrips populations in onion fields in Canada. The effectiveness of predaceous mites, which are used to control thrips in greenhouses (see Greenhouse cucumber, western flower thrips, 22.34), has not been evaluated in the field.

Chemical control — In most onion-growing areas, onion thrips have been, or are controlled by foliar sprays directed at onion maggot adults. However, since 1980, the use of granular insecticides and greater dependence on monitoring programs to manage onion maggot have resulted in fewer foliar sprays and greater damage attributable to onion thrips. The onion thrips is not known to be resistant to any of the insecticides currently used in onion production in Canada.

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(Original by G.J.R. Judd)

▶ 13.28 Other insect pests

Shallot aphid *Myzus ascalonicus* Doncaster

This aphid occurs in Canada and can transmit viruses; see onion yellow dwarf and shallot latent viruses, 13.14.

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Figures *14.2* to *14.7*

BACTERIAL DISEASES

▶ 14.1 Scab *Fig. 6.4*

Streptomyces scabies (Thaxt.) Waksman & Henrici (syn. *Actinomyces scabies* (Thaxt.) Güssow)

Scab is a minor disease of parsnip in Canada. Symptoms on parsnip roots are similar to those seen on carrot (see Carrot, 6.4). Overall growth and yield are not adversely affected by this disease, but quality and marketability may be reduced. For more information on the causal agent, disease cycle and management of scab, see Potato, 16.5.

(Original by R.J. Howard and R.F. Cerkauskas)

FUNGAL DISEASES

▶ 14.2 Itersonilia canker *Figs.14.2a-c*

Itersonilia perplexans Derx

Itersonilia canker has been reported on parsnip in Canada and the United States. A similar disease caused by *Itersonilia pastinacae* Channon has been reported in Great Britain. *Itersonilia pastinacae* differs from *I. perplexans* by its more abundant formation of chlamydospores and specificity to parsnip. However, some strains pathogenic to parsnip may not form chlamydospores and some strains of *I. perplexans* may have chlamydospores. The phenetic separation of the two is gradual. *Itersonilia perplexans* is widespread as a leaf surface saprophyte and is pathogenic on parsnip, chrysanthemum, sunflower, several other cultivated plants, and some weeds.

Symptoms Roots, leaves, petioles, inflorescences, and seeds may be affected. In roots, cankers may form at the bases of small lateral roots, although the crown and shoulder are generally the major areas affected. These cankers are reddish-brown with a roughened surface, later often turning a darker color *(14.2a).* Cankers do not extend deeply into roots, except in advanced cases. Secondary invasion by other organisms may follow and cause a decay of the whole root system. On leaves, small, 1 mm diameter, brown or orange-brown necrotic lesions are often surrounded by pale green halos *(14.2b).* Lesions may coalesce to form large necrotic areas. Petiole bases may have extensive gray to black lesions, while the inflorescence may be rotted completely.

Causal agent *Itersonilia* is characterized by dikaryotic mycelium with clamp connections at most septa and the production and discharge of binu- cleate, kidney-shaped ballistospores from upright, narrow sterigmata *(14.2c).* The hyphae are thin to slightly thick-walled, usually straight, septate at 50- to 120-µm intervals, and regularly branched. The hyphal forms develop a yeast phase when growing submerged in water. Ballistospores germinate either to form a mycelium or a secondary ballistospore. Ballistospores are 10 to 16 by 6 to 10.5 µm. Isolates vary in production of chlamydospores, which are 13 to 20 by 10 to 13.5 µm, nearly hyaline to deep golden-brown, thin to thick-walled, and single or in terminal clusters on short lateral branches of the mycelium. Appressoria form from germinating ballistospores and on solid surfaces are stalked and elongate and ovoid or semicircular in outline. Yeast cells occur in strains of *I. perplexans* and *I. pastinacae* and may be ellipsoidal, fusiform, cylindrical, allantoid or lunate. The cells are thin or slightly thick-walled, hyaline and vacuolated. Buds are sessile, borne on short denticles or on distinct sterigmata. Ballistospores are infrequently produced by yeast strains, but are more common in reverted hyphal forms.

To isolate *Itersonilia* from infected parsnip tissue, small pieces should first be dipped in 0.6% sodium hypochlorite to remove surface contaminants, then attached with vaseline to the lid of a petri dish over malt- extract agar. After incubation at 20°C for five to seven days, the ballistospores fall to the medium and initiate colonies. Colonies on most media are slow-growing (about 80 mm diameter in two weeks) and have some hyphal development beneath the surface of the agar. Colonies on malt-extract agar are appressed with thin white mycelium initially and later become gray-white and slimy. They occasionally have weak radial grooves with a sharply delimited margin that may be slightly zonate. The colonies have a weak, somewhat unpleasant odor.

Disease cycle *Itersonilia perplexans* overwinters as mycelium in infected parsnip roots or as chlamydospores in soil. Spread within the field is by wind-borne ballis- tospores, which can infect the foliage. New spores produced on the foliage can fall to the ground, contact the roots, and give rise to root infections, or they can be blown to other plants. The fungus can also be spread through infested seed.

Disease development generally begins late in the growing season but may occur earlier if favorable environmental conditions occur. The fungus requires a cool, wet season with an optimum temperature of 20°C. Disease development is limited by hot, dry conditions.

Management Control of the carrot rust fly (see carrot rust fly, 14.5) is important, because the larvae can predispose parsnip roots to *Itersonilia* infection, which usually occurs at the crown and other points where larvae have penetrated.

Cultural practices — High ridging to cover the shoulder of parsnip roots with soil throughout the season is effective because ballistospores are rapidly lysed by soil microorganisms. Long rotations and good soil drainage are also useful. Measures such as deep plowing, which enhances the decomposition of parsnip crop residues and exposes the fungus to the lytic action of soil microorganisms, are effective in reducing inoculum levels. Removal and destruction of cankered parsnip roots at harvest will eliminate the source of long-term carry-over of the fungus. The eradication of weeds will reduce other possible sources of inoculum.

Resistant cultivars — The cultivar Andover is resistant to itersonilia canker and has good horticultural characteristics.

Chemical control — Seeds suspected of harboring *Itersonilia* should be treated with a registered fungicide.

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(Original by R.F. Cerkauskas)

▶ 14.3 Phoma canker *Figs. 14.3a-d*

Phoma complanata (Tode:Fr.) Desmaz.

Significant losses in field and storage have occurred since the first report of this disease in 1984 from the Bradford region of Ontario. The disease is equally severe on parsnip growing in muck and mineral soils. Phoma canker is specific on parsnip and does not affect other vegetables. The cow-parsnip weed *Heracleum lanatum* Michx., which occurs throughout Ontario in meadows and edges of moist woods, is mildly affected by *P. complanata.*

Symptoms Leaf spotting, blighting and cankers on petioles and roots are the main symptoms and may vary in intensity among cultivars. At first, leaf spots are dark tan or brown, but yellow-green halos develop around the lesions three to four days later. Lesions on leaves vary in size and shape, but generally they are 1 mm or less in diameter. Under severe disease conditions, lesions appear five days after infection has occurred. As these lesions develop, they may coalesce, leading to yellowing, withering and dying of the leaves. These symptoms are known as leaf blighting *(14.3b).* Two weeks after infection, the coalesced lesions are pale brown or purplish-brown with dark, pepper-like fungal fruiting bodies in the center.

On the petioles *(14.3a),* light brown, elliptical lesions form four days after initial infection. Lesions darken to a brown-black color and coalesce, leading to canker formation about two weeks after infection. Cankers may occur anywhere along the petioles, and petioles frequently bend at the canker site, followed by yellowing, withering and dying of the leaf above the site of the canker. When cankers form on the upper part of the petiole, a characteristic "shepherd's crook" is formed *(14.3c).*

Root cankers *(14.3d)* are buff or dark brown to black, often with the small, black, pepper-like fruiting bodies of the fungus on the surface or embedded in the tissue. Cankers are frequently found on the shoulder and crown of the root and occasionally on the shank, and they may penetrate to the inner part of the root. Secondary organisms, such as bacteria and other fungi, are often associated with the root canker and can contribute to further decay. Roots with phoma canker have a distinct, sweet, cinnamon-like odor, and their commercial value is reduced.

White to buff-colored spore masses may be observed on well-developed lesions on diseased leaves, petioles and roots with the aid of a magnifying lens. The presence of fruiting bodies and the production of numerous spore tendrils from these fruiting bodies distinguish phoma canker from itersonilia canker.

Causal agent A scheme based on the behavior of isolates *in vitro* and *in vivo* (host substrate) has been used for the identification of *Phoma* species. The characteristic thick pycnidial wall *in vivo* and *in vitro,* the extent of mycelial growth, cultural characteristics on oatmeal agar, and its occurrence on parsnip distinguish *P. complanata* from other *Phoma* species.

Pycnidia in root tissue are scattered or aggregated, immersed or partly immersed, spherical with a dark brown to black outer wall, and lack setae. Pycnidia in host tissue are unilocular with one ostiole and range in diameter from 165 to 373 µm with an average of 250 µm. On oatmeal agar, pycnidia are superficial, immersed or partly immersed, and initially flesh-colored, but later they turn dark brown to black with a diameter of 228 µm (range 176 to 286 µm) and produce conidia abundantly. The pycnidia have thick walls (35 to 49 µm), which consist of about six layers of pseudo- parenchymatous cells.

Aseptate conidia exude in cirrhi from pycnidia on parsnip petioles, on root tissue, and in oatmeal agar cultures. They are hyaline, ellipsoid, cylindrical, fusiform or globose, often with polar guttulae. Aseptate conidia are 7.4 (5.0 to 10) by 2.4 (2.0 to 3.0) µm. Pycnidia in stored root tissue often contain many swollen, dark, septate conidia. One-septate, hyaline conidia with dimensions of 27.2 (22 to 34) by 8.1 (6 to 10) µm are present in older cankers or cultures.

In culture, *P. complanata* is readily distinguished from *Itersonilia perplexans* by the presence of pycnidia in culture media, the differences between the conidia (*P. complanata)* and ballistospores (*I. perplexans),* and the lack of clamp connections on the mycelium of *P. complanata.* The fungus is readily isolated on potato-dextrose agar or oatmeal agar. Colonies on oatmeal agar are regular, not scalloped, and they lack concentric zonation. Colonies are white to light gray or olivaceous-gray with dense aerial mycelium. The reverse side is light brown. Chlamydospores, sclerotia and an *Epicoccum* state are absent in this *Phoma* species.

Disease cycle In Ontario, phoma canker appears in mid- August. The disease often develops from a few, randomly scattered infected plants. As the season progresses, the affected areas enlarge and may coalesce. Canker development is favored by moderate temperatures and rain or high humidity. Secondary spread in the field is aided by wind- driven rain, heavy dew formation and insects. During rain or heavy dew, the large numbers of spores produced in the petiole cankers are carried down the petiole to the crown, where root infection may occur. Root cankers develop in the field or during cold storage. Cankers developing in cold storage arise from field infections and may be small and visible only after two cuts, perpendicular to each other, are made at the crown. Yield losses can be as high as 100% because of root canker formation and breakage of cankered petioles during harvesting.

The fungus can survive over winter for at least five months in parsnip residue in muck or mineral soil, regardless of the depth of incorporation. The fungus is seed-borne and may affect seed germination and seedling emergence.

Management

Cultural practices — The removal of diseased parsnip residue from the field and a one-year-mini mum rotation with other crops will reduce levels of the fungus in affected fields. Infected seed will show reduced vigor and germination. Workers should avoid direct skin contact with diseased plant tissues when harvesting, because high concentrations of photocarcinogenic furocoumarins are produced in such plants. These compounds, which also occur in carrot, celery and parsley at lower concentrations, produce severe skin rashes and blisters upon exposure of affected skin to sunlight.

Resistant cultivars — Harris Model is highly susceptible to the disease, while Hollow Crown Improved and All America are moderately resistant. The latter two cultivars are not widely grown in Ontario, because the extensive shoulder on the root reduces acceptability to packers and consumers. The control of phoma canker can be improved by harvesting these two cultivars before excessive shoulder development occurs. The cultivar Andover, bred for resistance to itersonilia canker, also has good resistance to phoma canker.

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(Original by R.F. Cerkauskas)

NEMATODE PESTS

▶ 14.4 Northern root-knot nematode *Fig. 6.20*

Meloidogyne hapla Chitwood

Symptoms Like carrot, parsnip is very sensitive to low numbers of this nematode. Mature roots may be deformed, short and branched, and secondary roots abnormally branched and hairy *(6.20).* For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 14.5 Carrot rust fly *Figs. 6.23a-e*

Psila rosae (Fabricius)

Carrot rust fly (see Carrot, 6.23) is one of the principal insect pests of parsnip in Ontario and Quebec. In areas of eastern Newfoundland, where high populations of rust fly exist, parsnip crops have been damaged extensively.

Damage Rust fly damage on parsnip is similar to that on carrot (*6.23 a,b*), but the damage is rarely as severe.

Management Rotation and avoidance of areas with high rust fly populations are the only feasible strategies for most growers, because no insecticides are registered for rust fly control on parsnip in Canada.

Monitoring — Parsnip crops are often grown on mineral soil or shallow muck soil, where rust fly populations are usually low. Nevertheless, adult rust flies (*6.23e)* can be monitored with sticky-board trapping techniques described for carrot. In Ontario and Quebec, areas with high populations of rust fly have been determined, but growers in new areas may have to estimate rust fly populations.

Cultural practices — Growers should practice crop rotation and avoid areas known to have high rust fly populations.

(Original by M.R. McDonald)

▶ 14.6 Carrot weevil *Figs. 6.24a-d*

Listronotus oregonensis (LeConte)

Carrot weevil (see Carrot, 6.24) is one of the main insect pests of parsnip in Ontario and Quebec.

Damage Carrot weevil larvae (*6.24d*) cause economic damage to parsnip in the same manner as on carrot *(6.24a,b).* However, they do not cause as much damage to parsnip, possibly because most parsnip crops are grown on mineral or shallow muck soil and parsnip crops are usually rotated with non-host crops.

Management Crop rotation is the only feasible strategy for most growers, because no insecticides are registered for carrot weevil control on parsnip in Canada.

Monitoring — Where parsnip crops are grown in the same field after celery or carrot, monitoring should be done throughout the field in the spring, because the adult weevils

(6.24c) will have overwintered in the field under crop residue. If a non-host crop was grown the previous year, then carrot weevils may have overwintered in the weeds and grasses along the headlands and ditches. In this case, monitoring traps should be concentrated at the edge of the field (see Carrot, 6.24, for procedures).

Cultural practices — Rotations of up to three years with a non-host crop can be an effective means of reducing carrot weevil populations and consequent crop damage, especially in fields distant from one another.

(Original by M.R. McDonald)

▶ 14.7 Other insect pests *Fig. 14.7; see text*

Black swallowtails *Papilio* spp.
Cutworms
Wireworms

Black swallowtails are butterflies (family Papilionidae), several species of which occur in Canada. Their larvae *(14.7),* which are called ragworms, celery worms or parsley-worms, feed on the foliage of parsnip. In southwestern Ontario, larval populations can become sufficiently large to cause defoliation. Farther north and in other parts of Canada, larval numbers are rarely sufficient to cause economic loss. However, in eastern Newfoundland, larvae of the short-tailed swallowtail *Papilio brevicauda* Saunders have on occasion completely defoliated parsnip grown in home gardens.

Cutworms (see Carrot, 6.25; and Tomato, 18.35) Cutworms *(6.25a-c; 18.35a-g)* may feed on young parsnip plants. Eggs are laid at the base of the plants, and the larvae feed on seedlings at ground level. A significant reduction in plant stand can result when the young petioles are chewed off at or near the soil surface. Close inspection will reveal that leaves and seedlings have been severed. Insecticides applied in the evening, when the cutworm larvae are feeding, can effectively reduce further damage.

Wireworms (see Maize, 12.21) Wireworms *(12.21a,b)* may feed on parsnip roots. They penetrate directly into the root, leaving one or more circular holes on the surface. The root must be cut open to reveal the extent of the interior tunneling. In Ontario, damage is rarely extensive enough to cause economic loss.

(Original by M.R. McDonald)

MITE PESTS

▶ 14.8 Two-spotted spider mite *Figs. 22.36e,f*

Tetranychus urticae Koch

The two-spotted spider mite (see Greenhouse cucumber, 22.36) can often be found on parsnip crops in Ontario.

Damage Under hot, dry conditions, the two-spotted spider mite can cause a stippling of the leaves, which leads to overall chlorosis. When first observed where the plants are dusty, mite damage can be confused with magnesium deficiency, but close examination of the underside of the leaves will reveal high mite populations.

Management Cultural practices that reduce mite populations are the only feasible strategy, because no miticides are registered for use on parsnip in Canada.

Cultural practices — Overhead irrigation helps reduce the mite population, but irrigation is not often used in parsnip production. During hot, dry weather, growers should avoid practices that throw dust onto the leaves.

(Original by M.R. McDonald)

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15 Pea and bean

15A Pea

Figures *15A.1* to *15A.15; 15A.2T1*
Bacterial diseases
15A.1 Bacterial blight
Fungal diseases
15A.2 Ascochyta diseases
- Ascochyta leaf and pod spot
- Foot rot
- Mycosphaerella blight

15A.3 Damping-off, root rot, seed decay, seedling blight, wilt
15A.4 Downy mildew
15A.5 Powdery mildew
15A.6 Rust
15A.7 Sclerotinia stem rot
15A.8 Septoria leaf blotch
Viral and viral-like diseases
15A.9 Miscellaneous viral and viral-like diseases
- Alfalfa mosaic
- Aster yellows
- Bean yellow mosaic
- Pea enation mosaic
- Pea seed-borne mosaic
- Pea streak
- Pea stunt

Non-infectious diseases
15A.10 Nutritionaldisorders
- Boron deficiency

15A.10 Nutritional disorders (cont.)
- Boron toxicity
- Calcium deficiency
- Iron deficiency
- Magnesium deficiency
- Manganese deficiency
- Manganese toxicity
- Nitrogen deficiency
- Phosphorus deficiency
- Potassium deficiency

15 A.11 Other disorders
- Cold injury
- Herbicide injury
- Water congestion

Nematode pests
15A.12 Northern root-knot nematode
15A.13 Stem and bulb nematode
Insect pests
15 A.14 Pea aphid
15 A.15 Other insect pests
- Cutworms
- Other caterpillars
- Pea leaf weevil
- Pea moth
- Pea weevil

Additional references

BACTERIAL DISEASES

▶ 15A.1 Bacterial blight *Figs. 15A.1a,b*

Pseudomonas syringae pv. *pisi* (Sackett) Young, Dye & Wilkie (syn. *Pseudomonas pisi* Sackett)

Bacterial blight is a widespread disease that causes serious losses in some years. However, under dry conditions, pods are seldom severely affected and economic losses are rare. *Pisum sativum* L. (garden pea), *P. sativum* var. *arvense* L. (field pea), *Lathyrus odoratus* L. (sweet pea), *L. latifolius* L. (perennial pea), *Dolichos lablab* L. (hyacinth bean), *Vicia benghalensis* L. (purple vetch),

Vicia villosa Roth, (hairy vetch), *Glycine max* (L.) Merrill (soybean) and *Vigna* spp. (cow pea) have been reported to be susceptible to *Pseudomonas syringae* pv. *pisi.*

Symptoms First symptoms are usually small, water-soaked spots on leaves, pods and stems that grow together as they enlarge (*15A.1a*). Leaves may turn brown and die. Affected areas of leaves are translucent when held up to light. Lesions on pods start out as olive-green, greasy- appearing spots, but brown margins develop as they enlarge (*15A. 1b*). In severely affected plants, seeds may be covered in slime. Lesions may eventually girdle the stems, causing death of the plant above the lesions. Infected flowers either shrivel at an early stage or fail to form pods. When growing tips are killed, new stems are initiated from lower nodes, resulting in irregular maturity. Infected seed may show brownish discoloration or water-soaking.

Causal agent *Pseudomonas syringae* pv. *pisi* has Gram-negative, long, non-sporing rods, 0.7 by 2 to 3 µm, which are motile with one to five polar flagella. This bacterium is a weak fermenter of carbon sources. It produces a green fluorescent pigment on King's B medium. Most species of *Pseudomonas* are white when seen in-mass on solid media. Four races of the pathogen have been described. Races 1 and 2 are the most prevalent in pea-growing areas of Canada and the United States.

Disease cycle *Pseudomonas syringae* survives on seed both internally and externally. It can overwinter in undecomposed crop residue. Initial infection is often from infested seed. Secondary infections occur from bacteria carried by splashing water to leaf stomata or to sites of injury, such as those caused by hail or wind-blown soil. Infection is usually visible four to six days after inoculation. Warm, humid weather favors infection. Early infection may kill seedlings and under optimum conditions, spread may be rapid and considerable yield loss may occur.

Management

Cultural practices — Control of bacterial blight is best achieved by planting disease-free seed and avoiding the use of overhead irrigation. Seed produced in arid areas, such as those of the northwestern United States, is more likely to be free of the pathogen. Crop rotation out of peas for at least one year to allow pea residues to break down reduces inoculum levels, as does plowing infested residues.

Resistant cultivars — Cultivars resistant to each race and with combined resistance to races 1 and 2 are available.

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(Original by H.S. Pepin)

FUNGAL DISEASES

▶ 15A.2 Ascochyta diseases *Figs. 15A.2a-g; 15A.2T1*

Ascochyta leaf and pod spot

Ascochyta pisi Lib.

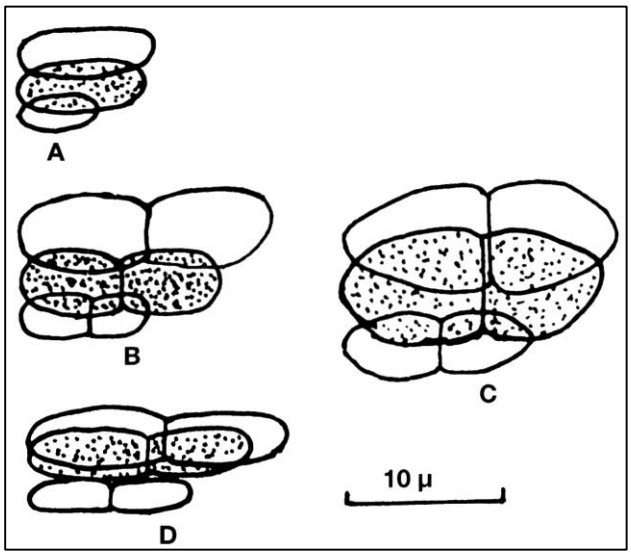
Foot rot

Phoma medicaginis var. *pinodella* (L.K. Jones) Boerema (syn. *Ascochyta pinodella* L.K. Jones)

Mycosphaerella blight

Mycosphaerella pinodes (Berk. & Bloxam) Vestergr. (anamorph *Ascochyta pinodes* L.K. Jones)

The complex of diseases known as leaf and pod spot, foot rot and mycosphaerella blight are caused by three closely related fungi. These diseases are present worldwide wherever pea is grown, particularly in temperate zones. Losses may be as high as 50% in processing peas, especially if the disease is caused by *Mycosphaerella pinodes. Ascochyta pisi* has been reported on *Pisum, Lathy rus* and *Vicia* spp.; *M. pinodes* occurs on *Pisum* (pea), *Lathyrus* (wild pea), *Vicia* (vetch) and *Phaseolus* (bean) spp.; *P. medicaginis* var. *pinodella* is known to infect *Pisum sativum, Trifolium pratense* L. (red clover) and other Leguminosae. The disease on *Trifolium* is known as black stem and leaf spot.



15A.2T1 Ascochyta diseases; shape and relative size of *Phoma medicaginis* var. *pinodella* conidia (A); *Mycosphaerella pinodes* conidia (B) and ascospores (C); and *Ascochyta pisi* conidia (D). Reprinted by permission from DJ. Hagedorn, ed., *Compendium of Pea Diseases.* © 1984. The American Phytopathological Society.

Symptoms Symptoms vary depending upon the causal agent. All three organisms can infect pods and, if they penetrate the pod wall, the seed may become infected and serve to spread the pathogen(s) over great distances. Seedlings from infected seeds show a blackening of the stem tissue extending from the soil line upward for 5 to 15 cm. Seedlings may be killed, but more commonly they survive in a weakened state. In the field, it is not possible to differentiate symptoms caused by *M. pinodes* and *P. medicaginis* infections of seedlings. In dry weather, lesions remain small and there is a general yellowing of the foliage, particularly the lower foliage, in both diseases.

Ascochyta leaf and pod spot is characterized by lesions on the leaves, stems and pods; rarely are the cotyledons or roots involved. Lesions of A. *pisi* are slightly sunken, tan to brown, with a distinct dark border. They tend to be circular on leaves and pods and elongate on stems, with numerous pycnidia (*.15A.2a,b).*

Phoma medicaginis lesions tend to develop near or just above the soil line, causing foot rot (*15A.2c*). The fungus penetrates the cortical region of the tap root and hypocotyl. Lateral roots may be invaded and destroyed. Very susceptible cultivars can be killed. Pods and seeds may become infected (*15A.2d*).

Mycosphaerella blight symptoms are irregular, usually consisting of dark flecks on leaves, pods and stems (*15A.2e-g*). Under favorable weather conditions they may enlarge to produce characteristic ring patterns in alternating shades of brown and tan. The blue-black or purplish stem lesions become longer and wider and may girdle the stem. Petal infection causes the blossoms to fall off.

Causal agents The pseudothecia of *Mycosphaerella pinodes* are dark brown, globose with papillate ostioles, and measure 90 to 180 µm in diameter. The asci are cylindric-clavate, ascus wall bitunicate, sessile, and contain eight, hyaline, two-celled ascospores that are constricted at the septum and rounded at the ends. Ascospores measure 12 to 18 by 4 to 8 µm.

Pycnidia of the three species vary in size and color. They form within the tissue of stems, leaves, pods and seeds. Initially, they are totally immersed but become erumpent as they mature. Pycnidia of *M. pinodes* are a darker brown and have thicker walls than those of *A. pisi* or *P. medicaginis.*

Comparison of conidial size (*15A.2T1*) shows *M. pinodes* to measure 8 to 16 by 3 to 5 µm, *A. pisi* to be 10 to 16 by 3 to 4.5 µm, and *Phoma medicaginis* var. *pinodella* to be 5 to 8 by 2 to 4 µm. Conidia of *P. medicaginis* can be distinguished from those of the other two species by their smaller size and also by being generally non-septate. Conidia of *M. pinodes* and *A. pisi* are generally one-septate, although in *M. pinodes* some conidia are two- or three-septate. Those of *M. pinodes* are usually ellipsoid, guttulate and slightly constricted at the septa. On oatmeal agar, *A. pisi* produces an exudate of carrot-red spore masses after 8 to 12 days at 18°C, whereas the exudate of *M. pinodes* is light buff to flesh-colored. *Phoma medicaginis* cultures are felty, grayish-brown turning black, with occasional sectoring with light buff conidial exudate.

Disease cycle All three pathogens can be seed-borne. Infected seed is the most important means of transmission for *Ascochyta pisi,* which is a weak saprophyte and does not produce a soil-borne resting stage. Conversely, *M. pinodes* and *P. medicaginis* are vigorous saprophytes, colonizing pea residues both on and below the soil surface. They produce sclerotia, chlamydospores, pycnidia and, in the case of *M. pinodes,* pseudothecia on straw fragments, and these structures survive as infectious agents for disease establishment. Ascospores are forcibly ejected from the pseudothecia and can be borne by the wind for a kilometre or

more and thus are able to disseminate over large areas. Ascospores require dry conditions for release, but high humidity, such as that found under a dense canopy, for spore germination. New crops of ascospores can be produced on the current year's diseased foliage at intervals of 13 days or more.

Conidia are extruded in a gelatinous matrix and depend on splashing rain and wind-borne droplets for dispersal. Germ tubes are produced, which penetrate directly through the cuticle and the cell walls. Symptoms appear in two to four days for both *M. pinodes* and *P. medicaginis*, and in six to eight days for *A. pisi*. Development of pycnidia in new lesions results in the release of more conidia, which enhances spread of the disease under moist conditions.

Management

Cultural practices — Growers should remove or plow down diseased pea vines after harvest. Crop rotations of four to five years should be practiced in areas where pea is a major crop, and susceptible crops such as *Lathyrus, Phaseolus, Trifolium* and *Vicia* spp. should be avoided in the rotation. Seed grown in dry areas reduces the likelihood of seed-borne infection.

Resistant cultivars — No commercial cultivars of processing peas are resistant to any of the ascochyta diseases, although resistance to *A. pisi* and to the foot rot phase of *P. medicaginis* has been reported in some cultivars of field pea.

Chemical control — There are no fungicides registered for the control of ascochyta diseases on pea in Canada. However, there is a fungicide approved for treatment of harvested pea seed destined for export to the United Kingdom.

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(Original by H.S. Pepin)

▶ 15A.3 Damping-off, root rot, seed decay, seedling blight, wilt *Figs. 15A.3a-f*

Aphanomyces euteiches Drechs.
Fusarium oxysporum f. sp. *pisi* (van Hall) W.C. Snyder & H.N. Hans.
Fusarium solani f. sp. *pisi* (F.R. Jones) W.C. Snyder & H.N. Hans.
Fusarium spp.
Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Pea seeds, stems and roots may be infected by an array of soil-borne pathogenic fungi from planting to harvest. Symptoms such as wilt, root rot, foot rot, damping-off, seedling blight and seed decay are common to all and, as a result, it is difficult to differentiate among them. Losses, at times, may be high. Both *Pythium* and *Rhizoctonia* have wide host ranges. *Aphanomyces euteiches* affects a wide range of Leguminosae. *Fusarium oxysporum* f. sp. *pisi* and *F. solani* f. sp. *pisi* are pathogenic only to pea.

Symptoms *Pythium* spp. have been described as the cause of seed decay (*15A.3a*), pre-emergence and post-emergence damping-off (*15A.3b,c),* and root-tip necrosis of pea. Symptoms vary from rotted seeds covered with a gray-white mold, to seedlings that fail to emerge, are stunted, or collapse as the roots and hypocotyl decay. Plants affected by root rot develop brownish to black root decay (*15A.3d,f)* and may show stunting, leaf yellowing, premature ripening, and wilt (*15A.3e*)**.**

Fusarium spp. develop pink masses of fungal spores at the base of the stem under humid conditions. *Fusarium oxysporum* infections do not develop brownish or blackened roots. Externally, the roots appear normal, but examination of longitudinal sections will reveal vascular tissue that has yellow to orange or dark red discoloration, which may extend well into the stem. Leaves wilt and turn yellow from the base upward.

Aphanomyces euteiches typically causes a watery soft rot near the soil line. Root cortex tissues collapse and die leaving the central woody tissue.

Rhizoctonia root rot is similar to fusarium root rot but usually occurs earlier in the season when soil temperatures are still low.

Causal agents For a general description of *Pythium* species, see Beet, pythium root rot, 5.7; and Carrot, pythium root dieback, 6.13; for *Rhizoctonia solani,* see Bean, rhizoctonia root rot, 15B.7. *Thanatephorus cucumeris,* the sexual state of *R. solani,* is rarely observed in pea fields.

Seed rots, damping-off and seedling blights are caused mainly by *Pythium* spp. Root rots are caused by *Aphanomyces euteiches, Fusarium solani* f. sp. *pisi, Fusarium* spp. and *Rhizoctonia solani,* and may develop any time after the seedling stage. Wilt is caused by *Fusarium oxysporum* f. sp. *pisi.*

Aphanomyces euteiches reproduces sexually by means of oospores produced by the fusion of antheridia and oogonia, which arise from aseptate mycelia. Sporangia, which are produced asexually, are indistinguishable from the hyphae.

The *Fusarium* spp. are mostly known in their asexual states. Both *F. solani* and *F. oxysporum* produce one- to five-septate, fusoid macroconi- dia, cylindrical to oval microconidia, and globose chlamydospores that are intercalary or borne on short lateral branches. *Fusarium solani* produces microconidia on long conidiophores, terminating in a single cylindrical to barely subulate phialide measuring 45 to 80 µm long, while those of *F. oxysporum* are produced on short conidiophores with numerous short, simple phialides.

Disease cycle *Aphanomyces* and *Pythium* survive in the soil as thick-walled oospores and can persist in the dormant state for years. Upon germination, hyphae or sporangia are produced from which asexual swimming zoospores are extruded. Zoospores are attracted by root exudates and, following encystment, penetrate feeder root-tips. *Aphanomyces* requires low soil temperatures (14 to 20°C) and high soil moisture for infection, but higher temperatures favor symptom development. *Pythium* infections are favored by high soil temperatures and high soil moisture.

Fusarium species survive in the soil as chlamydospores, which can remain viable for as long as 10 years. *Fusarium oxysporum* hyphae penetrate directly through the cortex into the vascular system, with little or no development of cortical lesions. *Fusarium solani* hyphae generally penetrate through the stomata of the epicotyl and hypocotyl, although direct penetration through the cuticular surface of pea epicotyls also occurs.

Rhizoctonia solani survives in the soil as thick-walled mycelium and thrives at temperatures between 21 and 25°C. It infects pea plants at the soil line. This pathogen is especially serious when soil moisture and soil organic matter are high, particularly when no tillage is practiced. Penetration is direct, with lesion development in the cortex.

Management Seed decay, seedling blight, root rot and wilt are difficult to control.

Cultural practices — A legume-free rotation of at least five years will reduce inoculum levels but will not eliminate them. Some of these fungi can survive saprophytically. Soils planted to peas should be well-drained. Compaction and over-irrigation should be avoided. Pea crop residues should be buried by deep plowing. Overcrowding of plants encourages the spread of root rots. Phosphate and nitrogen fertilizers help to reduce damage, particularly when the weather is cold and wet.

Resistant cultivars — Resistant cultivars are available for all described races of *F. oxysporum* f. sp. *pisi* and should be used wherever possible. Cultivars with resistance to *Pythium* spp. and *F. solani* f. sp. *pisi* are available, but they may not be acceptable for processing.

Chemical control — Seed treatments help to control damping-off and seed decay, but they are of little value for controlling root rots or wilt.

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(Original by H.S. Pepin)

▶ 15A.4 Downy mildew *Figs. 15A.4a-c*

Peronospora viciae (Berk.) Casp.
(syn. *Peronospora pisi* Syd.)

Downy mildew is favored by cool, moist growing conditions and is a problem only in coastal regions or in cool, wet years. Within the legume family, downy mildew affects *Pisum* (pea), *Vicia* (vetch) and *Lathyrus* (wild pea) species.

Symptoms Infection starts on the lower leaves and progresses upward with continuous cool, moist weather. Greenish yellow to brown lesions appear on the upper leaf surface (*15A.4a*), while directly below, on the lower surface, angular areas of downy

growth composed of sporangia and sporangiophores can be seen (*15A.4b*). This growth is white at first but later changes to violet, then mouse-gray, and at an advanced stage may turn black. Severely infected leaves often wither and die. Systemic infection from oospores in the soil or on the seed may result in severe stunting, distortion and even death of the seedlings. Affected pod tissues turn brown. Severely infected pods are distorted and a mycelial weft may form inside (*15A.4c*).

Causal agent *Peronospora viciae* sporangiophores emerge in clusters of five to seven through stomata from the underlying intercellular mycelium. Branching of the sporangiophore is right- or obtuse-angled. Tips of the branchlets are short, pointed and bear a single sporangium. Sporangia are oval to elliptic, hyaline, measure 11 to 22 by 13 to 29 µm, and germinate directly. Oogonia and antheridia are produced by the mycelium within the host tissue. Spherical, light-brown to deep yellowish-pink oospores are readily produced, particularly in senescent tissue; they measure 25 to 37 µm in diameter. Germination of the oospores is by a single germ tube emerging at any point on the surface.

Disease cycle Oospores of *P. viciae* are produced abundantly in diseased tissue and are the main means of overwintering in the seed, soil and on crop residue. Some may survive for up to 15 years in the soil. Oospores are the primary source of systemic infections in the spring. Sporangia produced from these infections supply the inoculum for secondary infections on the leaves, stems and pods. Sporangial production requires 12 hours at 90% relative humidity; more abundant spore production occurs at relative humidity above 95%. Optimum germination of the sporangia occurs at 4 to 8°C, but some germination may take place from 1 to 20°C. Air temperature greatly affects viability of the sporangia, with the period of viability increasing as the temperature decreases.

Management

Cultural practices — A three-year rotation out of legumes usually will reduce inoculum to negligible levels. Burial of infested crop residues by deep tillage is also effective. Disease-free seed from an area of low rainfall should be used.

Resistant cultivars — Some tolerant cultivars are available.

Selected references

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(Original by H.S. Pepin)

▶ 15A.5 Powdery mildew *Figs. 15A.5a,b*

Erysiphe polygoni DC.
(syn. *Erysiphe pisi* Syd.)

Powdery mildew is a widespread but rarely destructive disease on processing pea because the crop usually is harvested before the disease becomes severe. It is more prevalent on late-maturing garden pea and on field pea grown for seed. The powdery mildew fungus has a very wide host range, occurring on more than 350 species of plants. The pathogen occurs in many physiologic races, each of which affects only the most closely related crops. The pea race apparently infects only pea, although it is thought that it can also attack sweet pea.

Symptoms Small, diffuse, off-colored spots appear on the upper and lower surfaces of older leaves. The lesions later appear powdery white (*15A.5a*). The host tissue turns purplish and as it ages, tiny, golden-brown to black, pinheadsized cleistothecia develop (*15A.5b*). If leaves, stems and pods become infected, the foliage withers and the plants may die. Severe pod infection causes a gray-brown discoloration of the seed.

Causal agent *Erysiphe polygoni* has hyaline hyphae and produces colorless, one-celled ascospores in asci enclosed in black cleistothecia on the surface of living plants. Appendages on the cleistothecia of *Erysiphe* are hypha-like or absent. The cleistothecia are dark and measure up to 180 µm in diameter, each containing 3 to 10 asci with four to eight ascospores per ascus. The ascospores are hyaline and 9 to 14 by 10 to 25 µm. Large, one- celled conidia are terminal in chains on isolated, unbranched aerial conidiophores. The hyphae penetrate epidermal cells in which they feed by means of haustoria.

Disease cycle Cleistothecia are produced abundantly on diseased tissue and are the usual overwintering state of the pathogen. The disease can also be seed-borne. Primary infections in temperate regions can occur from ascospores produced in cleistothecia or from conidia blown in from southern areas. Infection follows spore germination, a process that takes about one hour and occurs during dry weather when nights are cool. Secondary infections result from conidia spread by winds and air currents both within and between fields. *Erysiphe polygoni* can grow from 15 to 28°C, but conidial germination is optimal at 20 to 24°C. Conidia that fall to the ground die. The presence of moisture on the leaf surface tends to inhibit germination. The spores contain a relatively high water content and are able to germinate in a dry atmosphere.

Management

Cultural practices — In processing pea, significant levels of powdery mildew seldom develop before harvest; therefore control measures are seldom necessary. Crop rotation, early seeding, and plowing down of infested crop residue are effective in reducing inoculum levels.

Resistant cultivars — Some mildew-resistant cultivars are available.

Selected references

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(Original by H.S. Pepin)

▶ 15A.6 Rust *Fig. 15A. 6*

Uromyces fabae (Grev.) Fuckel
(syn. *Uromyces viciae-fabae* (Pers.) J. Schröt.)

Rust is widespread on processing pea in eastern Canada but is seldom destructive. It affects many species of *Vicia,* including spring vetch, broad bean and faba bean, as well as species of *Pisum* and *Lathyrus* (wild pea). Rust-colored, blister-like pustules develop on leaves (*15A.6)* and stems. As the pustules age, they may be surrounded by a chlorotic halo. Later, the pustules turn black as overwintering teliospores are produced. The life cycle of this fungus is complex, with five reproductive stages. There is no alternate host.

Management A crop rotation of two years prevents build-up of this disease.

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(Original by H.S. Pepin)

▶ 15A.7 Sclerotinia stem rot *Fig. 15A.7*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia stem rot is a minor disease of processing pea. Infection rarely occurs before full bloom and the disease does not usually develop rapidly enough to cause damage before harvest. However, under conditions of heavy vine growth and wet weather, sporadic outbreaks may cause severe rotting of the leaves, stems and pods (*15A.7*). White mycelial mats appear on the rotted plant tissue. Dark scle- rotia, the overwintering stage of the fungus, develop in the mycelial mats (see Carrot, sclerotinia rot, 6.15).

Management Control measures are usually not necessary for processing pea, but susceptible crops such as bean should not follow pea crops. Deep plowing and a five-year rotation to non-susceptible crops may be necessary where the disease has been severe.

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(Original by H. S. Pepin)

▶ 15A.8 Septoria leaf blotch *Fig. 15A.8*

Septoria pisi Westend.

Leaf blotch is common on pea. It affects mainly aging foliage and thus has little effect on yield of processing pea. Yellow to straw-colored blotches with ill-defined margins surrounded by chlorotic halos appear on older leaves. At maturity, the blotches become speckled with dark brown, pin-point pycnidia (*15A.8*). In humid weather, conidia are spread by splashing rain. The fungus overwinters on plant residues.

Management Crop rotations of two to three years reduce disease incidence.

Selected references
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(Original by H.S. Pepin)

VIRAL AND VIRAL-LIKE DISEASES

▶ 15A.9 Miscellaneous viral and viral-like diseases *Figs. 15A.9a-g*

Alfalfa mosaic virus
Aster yellows mycoplasma-like organism
Bean yellow mosaic virus
Pea enation mosaic virus
Pea seed-borne mosaic virus
Pea streak virus
Pea stunt (red clover vein mosaic virus)

A number of viral diseases of pea have been reported. None of these is known to cause much economic loss except pea seed-borne mosaic, which on occasion has caused serious problems in some pea breeding-lines. Most pea viruses are aphid transmitted.

Symptoms Symptoms vary but mottled yellowing of leaves, vein clearing, stunting, shortened intemodes, malformation of the growing point, and brown streaking of the stems without death of the tissues are suggestive of viral disease (*15A.9a-g).*

Causal agents Laboratory tests may be needed to identify the virus or combination of viruses present.

Alfalfa mosaic virus is a bacilliform particle that measures 18 nm in diameter and is variable in length. It is transmitted by aphids in a persistent manner. Seed transmission rates range from 1 to 5%. It is not serologically related to any other well-characterized virus.

Aster yellows is caused by a mycoplasma-like organism that is transmitted by leafhoppers (see Lettuce, aster leafhopper, 11.23); its symptoms include proliferation of leaves (*15A.9a).*

Bean yellow mosaic virus is a flexuous rod, about 750 by 15 nm, belonging to the potyvirus group. It is transmitted by sap, aphids and seed.

Pea enation mosaic virus is an isometric particle, about 30 nm in diameter and is one of the ungrouped viruses. Transmission is by aphids.

Pea seed-borne mosaic virus is a flexuous rod about 750 nm long and is in the potyvirus group. Seed transmission may be 30% or higher. Field spread is primarily by aphids.

Pea streak virus is a slightly flexuous rod about 12 by 620 to 630 nm. It is in the carlavirus group and is closely related to red clover vein mosaic virus.

Pea stunt is caused by red clover vein mosaic virus, which is a fairly rigid rod measuring 12 by 650 to 670 nm and is in the carlavirus group. It is transmitted readily in sap. Aphids inefficiently transmit the virus in a non-persistent manner. It is seed transmitted in *Trifolium pratense* L. (red clover) and *Vicia faba* L. (broad bean), but not in pea.

Disease cycle Initial infection in the field may be from overwintering virus in leguminous weeds or crops, such as alfalfa, or from seed transmission. Subsequent spread is largely by aphid transmission.

Management

Cultural practices — Virus-free seed should be used. Pea crops should not be planted near other leguminous crops, and weeds should be controlled to reduce aphid movement.

Resistant cultivars — Cultivars resistant to pea enation, bean yellow and pea seed-borne mosaic are available.

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(Original by H.S. Pepin)

NON-INFECTIOUS DISEASES

▶ 15A.10 Nutritional disorders *Fig. 15A. 10*

Boron deficiency
Boron toxicity
Calcium deficiency
Iron deficiency
Magnesium deficiency
Manganese deficiency
Manganese toxicity
Nitrogen deficiency
Phosphorus deficiency
Potassium deficiency

Pea is only moderately sensitive to nutritional disorders. Visible symptoms may be useful for preliminary determination, but plant tissue and soil analyses in conjunction with pH determinations may be necessary for positive diagnosis. The following disorders can be of significance in pea production.

Boron deficiency

Pea plants are occasionally damaged by a lack of boron. Affected plants appear stunted, with thickened and stiffened stems caused by the death of growing points, which results in a bushy growth-habit. Young leaves may be small and chlorotic with scorched tips.

Boron toxicity

Pea is sensitive to excess boron. This disorder usually occurs when pea follows other crops that require relatively high boron levels. Symptoms may resemble potassium deficiency. Stunted growth, yellowing and chlorosis of leaf margins, the development of irregular, drab olive, water-soaked spots along the margins followed by necrosis of the spots are characteristic (*15A. 10).* Yield may be substantially reduced. Growers should avoid planting pea after crops that have high boron requirements.

Calcium deficiency

Small, red, slightly sunken spots near the midribs, which progress outward until they involve the whole leaf, are indicative of calcium deficiency. On younger leaves, the interveinal tissue changes to pale green, yellow, then almost white as the disease progresses. Plants are stunted, with severe wilting of stem, pedicels and leaves of young tissue.

Iron deficiency

Symptoms of iron deficiency include the yellowing of upper leaves and chlorosis of the terminal foliage, which is greatly reduced in size. A partial remedy may be obtained by foliar application of iron sulfate or chelate.

Magnesium deficiency

Interveinal chlorosis and green leaf margins and veins on older leaves are symptomatic of magnesium deficiency in pea plants. This disorder is most likely to occur in acid soils or in soils low in magnesium.

Manganese deficiency

in pea is sometimes called marsh spot. Crops growing in alkaline soils may be unable to take up sufficient manganese. While affected plants may appear normal, individual seeds have brown, necrotic lesions on one or both cotyledons. Foliar-applied manganese sulfate prevents the disorder, but care must be taken to avoid toxicity from over-application.

Manganese toxicity

Pea plants affected by this disorder, also known as purple blight, are stunted with purple, shrivelled foliage at the base, purplish foliage along the lower to middle stem blending into rust-colored foliage above, and chlorotic and green foliage at the top. Only one or two pods may develop, and these usually do not have seeds. Low pH tends to increase manganese uptake and toxicity. Application of lime raises the pH above 6.0 and alleviates symptoms. High levels of iron and aluminum result in similar disorders.

Nitrogen deficiency

This is not usually a problem with pea because of its ability to utilize nitrogen fixed by nitrogen-fixing *Rhizobium* bacteria. However, nitrogen deficiency can appear where cold soils or low pH inactivate the bacteria. Symptoms include retarded growth of both shoots and roots, and aerial portions of the plant are shortened, spindly with small yellow leaves, and tend to remain upright. Blossom production is severely reduced, and lower leaves may prematurely defoliate. Application of nitrogen fertilizer at a low rate will remedy this problem.

Phosphorus deficiency

The symptoms of phosphorus deficiency are very similar to those of nitrogen deficiency. The development of dull, dark, bluish green foliage, which tends to wither prematurely, distinguishes this disorder. A soil test determines the amount of phosphorus needed to correct the situation.

Potassium deficiency

Internodes, particularly those in the upper portion of the plant, are shortened, resulting in a marked stunting of the pea plants. Leaves display marginal chlorosis, then necrosis of small spots near the leaf edges. The small spots coalesce, resulting in major necrosis of the entire leaf, which may curl upward and inward. An early spring application of potassium (K_20) at the rate of 18 to 20 kg/ha should prevent this disorder.

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(Original by H.S. Pepin)

▶ 15A.11 Other disorders *Figs. 15A. 11a-d*

Cold injury
Herbicide injury
Water congestion

Cold injury can occur when temperatures drop below 0°C, particularly if pea plants have been growing at high temperatures. Cold-hardened plants are less susceptible to injury. Plants growing in low areas in the field and in areas of poor air movement are more prone to injury. Symptoms include killing of the growing tips on very young growth; roughened, jagged edges or leathery, bilobed young leaves; and long, water-soaked, translucent lesions between the main veins. These lesions may turn necrotic on the underside of older leaves. If frost occurs at the green-harvest stage, lacy white lesions form on pods, resulting in uneven quality. Tall cultivars are more susceptible to frost injury than shorter ones. Although there is no preventive control of frost injury, some cold-resistant cultivars are available. They generally have colored seeds.

Herbicide injury may occur through improper use of herbicide on a pea crop itself (*15A.11a*), as a result of herbicide drift *(15A.11c)* from nearby cereal crops, or from herbicide residues in the soil resulting from applications to a previous crop *(15A.11b).* Symptoms vary with the herbicide and may include yellowing and stunting of the plant, mild to severe distortion and epinasty of the stems and leaves, abortion of flowers, pod abscission, reduced seed set, foliage chlorosis, and necrosis of the lower leaves. Proper use of herbicides reduces this problem, but special precautions may be needed to prevent drift between fields.

Water congestion may result from adverse environmental conditions, such as high humidity, high temperature and high soil moisture, particularly on muck or clay soil. If the conditions that foster this disorder persist, considerable yield loss may occur, because of the reduction in vigor that accompanies the loss of photosynthetic surfaces. Small, water-soaked spots first appear near the outer edge and on the underside of pea foliage. The terminal portions of leaves and stipules become completely water-soaked as the spots increase in size. The affected tissue first appears darker green, but as gradual death occurs, which progresses from the outermost tip and edge of the foliage, the necrotic tissue takes on a shrunken, dry, straw-colored appearance *(15A. 11d).* Most of the foliage at one to several nodes may be affected, reducing vigor and yield. Improved weather conditions generally arrest the development of this disorder.

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(Original by H.S. Pepin)

NEMATODE PESTS

▶ 15A.12 Northern root-knot nematode *Fig. 7.15b*

Meloidogyne hapla Chitwood

Symptoms include yellowing and stunting of foliage, prolific branching of rootlets, and production of small, spherical galls on roots *(7.15b).* Seed set may be reduced. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 15A.13 Stem and bulb nematode

Ditylenchus dipsaci (Kühn) Filipjev

For a complete description of this nematode, see Onion, 13.24; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 15A.14 Pea aphid *Fig. 15A. 14*

Acyrthosiphon pisum (Harris)

The pea aphid, which is worldwide in distribution, occurs across Canada. It is the only important aphid pest of pea in Canada and can cause significant yield loss both in peas harvested for the fresh or frozen markets and in peas harvested dry for the soup or feed industries.

Damage Aphids feed by sucking plant sap, usually from the new growth at the tips of plants. During vegetative plant development, aphid infestations usually do not cause economic damage because their density is low and the plants are growing vigorously. At flowering, early pod-set and pod-filling, aphids can be found feeding on flowers and particularly on the young, developing seed pods. Feeding on the flowers and pods can reduce the number of seeds produced, particularly if aphid numbers are very high. The usual result, however, is a reduction in seed size, which also lowers yield. The protein content of seeds is not affected.

The pea aphid is capable of transmitting many plant viruses, but virus transmission by aphids has not been important in Canadian pea production.

Identification The pea aphid (family Aphididae) is light green, soft bodied, and measures up to 5 mm in length (*15A.14).* Winged and wingless forms occur. Both forms have long legs, antennae, paired abdominal projections (cornicles), and a well-developed tip of the abdomen (cauda). Identification is not usually a problem because no other aphids commonly occur with the pea aphid on field pea in Canada.

Life history The pea aphid overwinters as eggs on perennial cultivated legumes, such as alfalfa, and on wild legumes. It also migrates into Canada from the United States. Pea plants are infested soon after the seedlings emerge, which is usually by the beginning of June. In areas where the season is short, aphid populations build slowly through June and early July, but they can expand rapidly in mid-July to a single density-peak in late July or early August. In areas with a longer season and pea crops that are at a suitable growth stage, there may be two population peaks, one in early summer and a second in late summer. Adult aphids either emigrate from the pea plants or die as the crop ripens. In August or September, sexual forms appear on perennial legume hosts and lay the overwintering eggs on the plant stems.

Management

Monitoring — Economic thresholds and monitoring procedures have been developed in Canada for chemical control of the pea aphid in field pea, which should be monitored when about 50% of the plants have begun to flower. At least 20 stem tips, 20 cm in length, are cut from plants in various parts of the field, beaten into a white tray, and the aphids counted. If the aphids reach a density of two to three per stem tip at the flowering stage, an insecticidal treatment is warranted (based on 1985 market conditions).

Resistant cultivars — The effect of the pea aphid varies from cultivar to cultivar, but cultivar-specific thresholds have not been developed.

Biological control — Many natural enemies contribute to the control of the pea aphid. Often, however, natural enemies are insufficient to protect the crop from significant yield loss.

Chemical control — Once the threshold is exceeded, a recommended insecticide should be applied within a few days.

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(Original by R.J. Lamb)

▶ 15A.15 Other insect pests *Figs. 15A.15; see text*

Cutworms

Other caterpillars

Pea leaf weevil *Sitona lineatus* (L.)

Pea moth *Cydia nigricana* (Fabricius)
(syn. *Cydia rusticella* (Clerck))
(syn. *Laspeyresia nigricana* (Fabricius))

Pea weevil *Bruchus pisorum* (L.)

Cutworms

(see Tomato, 18.35) affect mainly the seedlings of pea crops, sometimes making some reseeding necessary, especially in home gardens *(6.25; 18.35).*

Other caterpillars,

such as the alfalfa looper *(8.38)* and cabbage looper *(8.40b-f)* (for more information about either of these, see Crucifers, 8.38, 8.40), feed on pea leaves and can become a serious contaminant in harvested processing peas. Insecticidal sprays are used when outbreaks are detected.

Pea leaf weevil

(family Curculionidae) The pea leaf weevil occurs in southern British Columbia and the adjacent northwest United States. The related clover root curculio, *Sitona hispidulus* (Fabricius), which also may affect pea crops, occurs from British Columbia to Nova Scotia. Adults of both species *(15A.15)* overwinter in plant residue, invade emerging pea seedlings and chew notches in the leaf margins. The larvae infest the roots. Both insects are minor pests of pea in home gardens. They usually are not a problem on pea crops grown in rotation.

Pea moth

(family Tortricidae) The pea moth is a sporadic and usually inconspicuous pest. The larva feeds inside the seed pod and overwinters in the soil. There is one generation per year across Canada. In the pea-growing areas of the lower Fraser Valley in British Columbia, releases of two parasites have provided partially effective biological control. In general, processing and fresh-market pea crops should not be grown in areas with dry (seed) pea or seed vetch crops. After harvest, all remaining pods and vines should be destroyed by ensiling, feeding or deep cultivating. Insecticides, if required, should be applied when eggs or larval entry holes in the pea pods are first observed, which may occur any time between late June and early August.

Pea weevil

(family Bruchidae) The pea weevil is a sporadic pest in pea-growing areas in the southern interior of British Columbia, where it sometimes requires control measures. Larvae hollow out the pea seeds, there being but one larva per seed. Adults develop within the seeds by late summer and overwinter either in harvested peas in storage or in peas left in the field. Infested seed should be fumigated before planting and plant residue should be removed by ensiling or buried by deep cultivating. Insecticides, if required, should be applied just prior to blossom and again two weeks later to kill adults before they lay eggs.

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(Original by C. Ritchot, M.E. Sweeney and R.S. Vernon)

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Figures *15B.1* to *15B.19; 15B.7T1; 15B.9T1*

BACTERIAL DISEASES

15B.1 Bacterial blights *Figs. 15B.1a-i; 8.2f*

Bacterial brown spot

Pseudomonas syringae pv. *syringae* van Hall

Common blight and fuscous blight

Xanthomonas campestris pv. *phaseoli* (E.F. Smith) Dye
(syn. *Xanthomonas phaseoli* (E.F. Smith) Dowson)
(syn. *Xanthomonas phaseoli* var. *fuscans* (Burkholder) Starr & Burkholder)

Halo blight

Pseudomonas syringae pv. *phaseolicola* (Burkholder) Young, Dye & Wilkie

Bacterial blights are important diseases of edible pod bean. In 1987, for instance, commercial crops of green bean and yellow bean in Manitoba sustained losses of about 70%. Dry edible bean is also occasionally damaged by these diseases. Common,

fuscous, and halo blight bacteria cause disease in bean and a few other legumes, whereas the brown spot pathogen has a very wide host range that includes many plant families.

Symptoms These diseases are difficult to distinguish from one another in the field. The causal organisms must be isolated and identified by laboratory procedures.

Bacterial brown spot is characterized by leaf lesions that are circular, brown and necrotic, and often surrounded by a bright yellow zone (*15B.1a*). Lesions may fall out, giving the leaf a shot-hole appearance. Water-soaking of the leaf is rare. Similar lesions may occur on the stem and pods. Lesions on pods are initially water-soaked, then turn brown. Infected pods may be twisted or bent where lesions develop.

Symptoms of common blight on leaves (*15B.1b,c*) initially appear as water-soaked spots that gradually enlarge and become flaccid. The lesions turn brown with irregular margins and are often surrounded by a narrow yellow border. As lesions enlarge and coalesce, the leaves become necrotic and appear burnt. On pods (*15B.1e*), the lesions are circular and initially appear greasy and gray. Later, they become slightly sunken and dark red-brown. Infected seed (*15B.1f)* sometimes may be shrivelled and show poor germination and vigor. A shiny, yellow bacterial deposit may be seen on pods and seeds. The symptoms of fuscous blight are indistinguishable from those of common blight.

Halo blight symptoms on leaves (*15B.1g,h*) appear first on the lower surface as small, water-soaked spots that become necrotic and are surrounded by a zone of yellow- green tissue. In severe infections, plants develop a generalized systemic chlorosis. Bacteria ooze from substomatal cavities and give the lesions a greasy, water-soaked appearance. Lesions on pods (*15B.1i*) are generally red or brown or sometimes green, and may appear water-soaked and show a crusty white bacterial deposit on the surface. Infected seeds show no symptoms or may be shrivelled and have poor germination and vigor. Root nodulation may be reduced.

Causal agent *Pseudomonas syringae* pv. *syringae* is similar to *P. syringae* pv. *phaseolicola.* However, the former has a wider host range and produces a bacteriocin syringacin W-1, whereas the latter is unable to use mannitol, inositol, sorbitol and erythritol, and produces the toxin phaseolotoxin.

Xanthomonas campestris pv. *phaseoli* is a Gram-negative, aerobic, straight rod, 0.4 to 0.7 by 0.7 to 1.8 µm, and motile by a single polar flagellum. Colonies on agar media are mucoid, convex, yellow and shiny *(8.2f).* The yellow color derives from membrane-bound xanthomonadin pigments that are insoluble in water. The fuscous strains produce a brown, diffusible, water-soluble pigment on media containing beef or yeast extract. Races have not generally been recognized in *X. campestris* pv. *phaseoli.*

Pseudomonas syringae pv. *phaseolicola* is a Gram-negative, aerobic rod, 0.5 to 1.0 by 1.5 to 4.0 µm and motile by polar flagella. It produces a diffusible fluorescent green pigment on iron-deficient media. The pathovar *phaseolicola* is identified by isolation on semi-selective media, by physiologic tests, and by pathogenicity to bean. Three races of *P. syringae* pv. *phaseolicola* have been described. Races 1 and 2 occur in North America and worldwide. Race 3 is confined to Africa.

Disease cycle Bacterial blights occur throughout the world on common bean. In Canada, the epidemiology of the four diseases described above is very similar. Seed is the major source of primary inoculum. It may become infected or contaminated while developing on the plant, or during harvesting, threshing or cleaning operations. The bacteria can survive in or on seed for many years. Other minor sources of primary inoculum include crop and weed plants, plant residues, soil and contaminated farm implements. Overwintering survival in soil is limited in Canada. Seed-borne inoculum infects the seedling systemically or may develop on the surface of the plant and penetrate through wounds or natural openings such as stomata and hydathodes. The bacteria are spread from infected plants to other plants by splashing water, people, insects and other animals, or machinery. Epidemics often follow wet, windy weather. Warm weather (25 to 30°C) favors common blight, while halo blight, particularly chlorosis, develops more rapidly under cooler conditions (16 to 20°C). Distinct foci of infection spreading from infected seedlings may be seen in fields (*15B.1d*).

Management

Cultural practices — Seed free from the pathogens can be obtained through a seed health and certification program, such as is administered for white bean in Ontario and for several kinds of bean in the United States. Crop rotation, cultivation to bury crop residues, and thorough washing of equipment are important because the bacteria may overwinter in crop residues, in soil or on machinery. Movement of people, other animals and machinery through fields should be restricted to reduce transmission of bacteria.

Chemical control — Seed treatments and foliar sprays with copper-based bactericides or antibiotics may help to reduce disease but do not provide a consistently high level of control.

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(Original by R. Hall)

FUNGAL DISEASES

▶ 15B.2 Anthracnose *Figs. 15B.2a-d*

Colletotrichum lindemuthianum (Sacc. & Magnus) Lams.-Scrib.
(teleomorph *Glomerella lindemuthiana* Shear)

This disease occurs widely on common bean and can cause complete loss of the crop when contaminated seed of a susceptible cultivar is planted and conditions favorable to disease development occur during the growing season. Anthracnose has been important on occasion, especially when new races of the fungus have appeared that are able to overcome the resistance available in currently used bean cultivars. The host range of the fungus includes tepary bean, cowpea, kudzu bean, mung bean, lima bean, scarlet runner bean, and faba bean.

Symptoms Lesions may occur on any part of the shoot. Infected cotyledons show small, dark brown to black lesions. Dark brown elliptical lesions may develop on the hypocotyl or stem and cause young stems to break. On older stems, these "eyespot" lesions rarely exceed 7 mm in length. Lesions occur most commonly on petioles and leaves. Initially, leaf lesions (*15B.2a*) occur on the lower surface along the veins. They are elongate, angular, brick red to purple, becoming dark brown to black. Later, similar lesions may occur on the upper leaf surface. Brown lesions of various sizes may also develop on the leaves, usually around veins. On pods, lesions first appear as brown specks and develop into sunken brown spots, 5 to 8 mm in diameter, with a dark brown or purplish border (*15B.2c,d*). Fungal fruiting bodies appear as small black specks over the lesion, producing a viscous, pinkish droplet of spores under humid conditions. On infected seeds (*15B.2b*), brown to black spots may be restricted to the seed coat or extend into the cotyledon.

Causal agent *Colletotrichum lindemuthianum* produces hyaline to gray hyphae that become dark brown to black with compact aerial mycelium at maturity. Growth in culture is most rapid at 22 to 24°C. Conidia are hyaline, uninucleate, single and cylindrical, with a clear vacuole-like body near the center. The ends of each conidium are obtuse or have a narrow, truncate base. They range in length from 9.5 to 22 µm, and in width from 2.5 to 5.5 µm. Conidia are produced on the surface of an acervulus that develops within and beneath the epidermis and may reach 300 µm in diameter. Brown, septate setae form at the margin of the acervulus and measure 4 to 9 by 100 µm. The mass of conidia in the acervulus may be pale flesh- to salmon-colored. The perfect state rarely forms on diseased beans.

Disease cycle *Colletotrichum lindemuthianum* can survive in bean seed as long as the seed remains viable, and it may overwinter in dry residues of an infected bean crop. Under favorable conditions, the fungus can germinate within six to nine hours and infect the plant. Several days later, acervuli are formed and break through the plant cuticle. Conidia form on the surface of the acervuli within a water- soluble, gelatinous matrix. They are carried to other parts of the plant or to other plants in splashing water or by machines, people or other animals. Production and germination of conidia occur from 13 to 26°C, with maximum development at 17°C. Relative humidity greater that 92% or free water is required for germination, infection and sporulation. Moderate, frequent rainfalls and windy weather are essential for the development of severe epidemics.

Management

Cultural practices — Seed should be pathogen-free, infected crop residues should be buried, and rotation with non-susceptible crops is suggested.

Resistant cultivars — The main approach used to control anthracnose is genetic resistance. At least six major races of the fungus exist: alpha, beta, gamma, delta, kappa and lambda. Bean cultivars may have resistance to one or more of these races but few are resistant to all the major races. Before 1976, only races alpha, beta and gamma were known in North America, and resistance to one or more of these races had been used in commercial bean cultivars. In 1976, races delta and lambda were identified in Ontario. These can be controlled by the Are-gene.

Chemical control — Seed can be treated with recommended fungicides. Applications of foliar fungicides may also provide some control.

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(Original by R. Hall)

▶ 15B.3 Gray mold *Figs. 15B.3a,b*

Botrytis cinerea Pers.:Fr.
(telemorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold is a widespread and important disease of bean, especially of green types, and can be very destructive during flowering and pod maturation in the field and during storage and transit of harvested pods. The pathogen has a very wide host range (see Lettuce, gray mold, 11.10).

Symptoms *Botrytis cinerea* causes disease in the field and in harvested beans. In the field, the disease can affect all parts of the shoot but is most commonly found on pods (*15B.3a)* and leaves. Infection usually starts on senescent tissues, such as cotyledons and flowers, or on parts damaged by frost, hail, wind, insects, sand or machinery. Affected tissues develop an extensive rot that is soft but not mushy or slimy. Lesions on leaves and pods, at first dark green and water-soaked, become gray to beige and often develop concentric zones. Longitudinal brown streaks may form on stems and petioles. As lesions dry out, the tissue turns brown. Fungal mycelium, conidiophores and conidia form a gray-brown powdery mass, and small black, flattened sclerotia may develop in stems and pods. The post-harvest condition known as nesting, which is a generalized rot accompanied by profuse, dirty white mycelium, occurs in humid transit and market packs.

Of the bean diseases discussed in this chapter, gray mold is similar only to white mold (*15B.3b*), which produces a white cottony mycelium without conidia and large, black, irregular sclerotia in infected tissue.

Causal agent (see Lettuce, gray mold, 11.10)

Disease cycle Gray mold is a common disease of green bean and many other vegetables (see Lettuce, gray mold, 11.10) under moist, cool conditions. The primary inoculum usually consists of conidia. Ascospores have been reported in New York State. Conidia originate from infected crop residues or plants or from sclerotia, and are probably always present as air-borne inoculum.

The mycelium infects injured or uninjured tissue and produces symptoms. Senescent and damaged cotyledons are usually the first tissues affected. Flowers are also infected by conidia. Leaves and pods are usually infected by mycelium growing from infected flowers. Conidia are produced on the infected plant two to three days after infection under a wide range of temperature and moisture conditions and serve as secondary inoculum to spread the disease. Conidia are produced on infected stems continuously into the bloom period. Conidia can also be produced from sclerotia on the surface of the soil, in plant residues and on fallen flowers. Sclerotia produced on stems and pods remain in the field or are disseminated with seed. Sclerotia survive longer in the soil than at the soil surface. Pods inoculated or infected in the field may develop symptoms in transit or in storage, and mycelium may spread to adjacent pods.

Management

Cultural practices — Growers should try to avoid conditions that lead to high humidity and prolonged wetness in the crop, such as dense canopies, narrow row widths, excess nitrogen fertilizer, rows perpendicular to the prevailing wind, and excess irrigation. Weeds provide a source of inoculum and contribute to a microclimate suitable for disease development. In transit after harvest, pod rots can be controlled by cooling the pods to 7 to 10°C and storing them with adequate ventilation. Plowing buries crop residues and sclerotia and thus may reduce inoculum, but it also may bring old sclerotia to the surface, where they can sporulate. Beans should be preceded by crops that are not important hosts for *B. cinerea*, such as cereal grains and com.

Resistant cultivars — Cultivars are available that tend to resist lodging, have an upright and open canopy, produce small non-persistent flowers, and bear pods that do not touch the ground.

Chemical control — Fungicide sprays applied while the crop is flowering may reduce disease, but are uneconomic in most regions. In coastal British Columbia, where disease pressure is often intense, fungicide applications are necessary.

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(Original by R. Hall)

▶ 15B.4 Root rots, damping-off, seed decay

Root rot, damping-off and seed decay of bean are caused by several fungi that occur singly or together. The pathogens are soil-borne and all are major problems on bean throughout the world and can be controlled in similar ways. Each fungus can cause decay of seeds and collapse and death of seedlings by damping-off. Distinctive symptoms are produced on hypocotyls and roots. Effects on the crop may include delayed and uneven emergence and stands, reduced growth, delayed or accelerated maturation, and reduced yield. The symptoms, epidemiology, and technical details for each disease are considered separately below. Management strategies are discussed under rhizoctonia root rot, 15B.7, and selected references are given at the end of that section.

▶ 15B.4 Black root rot *Fig. 15B.4*

Chalara elegans Nag Raj & Kendrick
(syn. *Trichocladium basicola* (Berk. & Broome) J.W. Carmichael)
(synanamorph *Thielaviopsis basicola* (Berk. & Broome) Ferraris)

Black root rot is widely distributed and causes disease on alfalfa, bean, beet, carrot, celery, corn, cotton, pea, peanut, tomato, squash, sweetpotato and tobacco.

Symptoms Symptoms first appear as elongate reddish- purple lesions on the hypocotyl and roots. The lesions enlarge, coalesce and become dark brown to black (*15B.4).* They may remain superficial and cause little damage to the plant, or they may penetrate deeply into the cortex and vascular tissue and cause stunting, early senescence, defoliation and plant death.

Causal agent (see Carrot, black root rot, 6.6)

Disease cycle Chlamydospores of the pathogen survive in soil and crop residues. Under favorable conditions, they germinate to produce mycelium that grows onto hypocotyl and root surfaces. The hyphae penetrate the intact plant surface or enter through wounds, lesions and natural openings to grow through and between the plant cells. Chlamydospores are produced on the hyphae throughout the infected tissue. Under moist conditions, hyphae emerge through the epidermis and produce chlamydospores and endoconidia. As the infected plant tissue decays, chlamydospores are liberated into the soil and may germinate to infect plants or colonize organic residues.

Growth and sporulation of the fungus are favored by high temperatures, but damage to bean is favored at 15 to 20°C, and by high soil moisture, neutral to alkaline soils, and high levels of nitrogen fertilizer.

Management See rhizoctonia root rot, 15B.7.

(Original by R. Hall)

▶ 15B.5 Fusarium root rot *Figs. 15B.5a-c*

Fusarium solani f. sp. *phaseoli* (Burkholder) W.C.Snyder & H.N. Hans.

Fusarium root rot can be a severe disease on bean. The pathogen also attacks peanut, cowpea and adzuki bean.

Symptoms Longitudinal, narrow, brick-red lesions or streaks develop on the hypocotyl and tap root (*15B.5a*). As these become more numerous, they coalesce and large portions of the below-ground stem and root system may become covered with superficial reddish-brown lesions (*15B.5b*). Necrosis is largely confined to the cortical cells but may extend into the stele. In coarse-textured (light) soils where growth of the roots is relatively unrestricted, rotted stem and upper root portions regenerate cortical tissue and plant productivity appears unaffected by the disease. In fine-textured (heavy) soils or where root growth is restricted by compaction, root rot tends to be more severe and yields are reduced. Severely diseased plants respond by producing numerous adventitious roots that develop from the hypocotyl in vertical rows near the soil surface. Damage to the stem and roots results in stunting and premature senescence of the shoot (*15B.5c*).

Causal agent *Fusarium solani* f. sp. *phaseoli* produces septate, hyaline mycelium and mostly three-septate (44.5 by 5.1 µm) and four-septate (50.9 by 5.3 µm), rarely five-septate, macroconidia that are nearly uniform in diameter along their length, and curved and rounded or slightly pointed at the apex. Microconidia are rare. Conidia are borne in sporodochia. Chlamydospores are globose, 11.6 µm in diameter, terminal or intercalary, single or in short chains, in conidia or hyphae. Cultures vary from blue to green, depending on the medium, when viewed from the underside. The surface mycelium is usually grayish-white. No perfect state is known for *F. solani* f. sp. *phaseoli.*

Disease cycle The pathogen survives in soil as chlamy- dospores, which germinate in response to nutrients released by root tips and germinating seeds. The hyphae penetrate the bean root directly, or through wounds or natural openings, to colonize the cortex but rarely extend into the endo- dermis or stele. Conidia may be produced on the stem near the soil surface. Conidia and hyphae in the soil or in degenerating plant tissue convert to chlamydospores, which also germinate and reproduce near seeds and roots of non-sus- ceptible plants and other types of organic matter. The fungus can perpetuate itself in soil in the absence of bean plants.

The pathogen is disseminated in dust on seeds or in seed bags, and in wind- and water-borne soil. By the third successive bean crop in land previously uncropped to bean, the level of inoculum may be sufficiently high to cause serious disease. Development of root rot is favored by cool, moist soil conditions, but reduction in yield is more evident when infected plants are subjected to drought.

Management See rhizoctonia root rot, 15B.7.

(Original by R. Hall)

▶ 15B.6 Pythium diseases *Fig. 15B.6*

Pythium aphanidermatum (Edson) Fitzp.
Pythium irregulare Buisman
Pythium myriotylum Drechs.
Pythium paroecandrum Drechs.
Pythium ultimum Trow

Several species of *Pythium* can cause seed rot, damping- off, root rot, stem rot and blight. These fungi are widely distributed in cultivated soils and affect bean wherever it is grown.

Symptoms *Pythium* species typically infect young tissues and cause a soft decay that is initially water-soaked and becomes necrotic. Seeds, roots and seedlings become soft and mushy and rapidly decay (*15B.6*). Infected seedlings that emerge may wilt and die within the first few weeks of growth. These plants may develop a lesion that extends from the roots up the hypocotyl and sometimes reaches the growing point of the shoot. Seed decay and damping-off reduce the density of the stand. A soft, brown decay may occur on stems and is common on feeder roots. These symptoms may be accompanied by stunting, premature senescence and reduced productivity of the affected plants.

Causal agent Numerous species of *Pythium* are pathogenic to bean. Those of major importance can be divided into two groups based on morphology and temperature relations (see Disease cycle). *Pythium ultimum, P. irregulare* and *P. paroecandrum*, and isolates resembling these species, lack oospores and have spherical sporangia. *Pythium myriotylum* and *P. aphanidermatum* are more common. These species have filamentous, lobate sporangia. Several other *Pythium* species also have been associated with bean diseases (see Beet, pythium root rot, 5.7; and Carrot, pythium root dieback, 6.13).

Disease cycle *Pythium* species are common in soil, have a wide host range, can colonize living plants and fresh organic debris, and can persist for many years as oospores. Their populations can increase on a wide range of crops. Disease severity is favored by high soil moisture. Low temperatures (20°C or lower) favor *P. ultimum, P. irregulare* and *P. paroecandrum*, while high temperatures (25 to 30°C) are required for the development of severe blight caused by *P. myriotylum* and *P. aphanidermatum* (see Beet, pythium root rot, 5.7; and Carrot, pythium root dieback, 6.13).

Management See rhizoctonia root rot, 15B.7.

(Original by R. Hall)

▶ 15B.7 Rhizoctonia root rot *Figs. 15B.7; 15B.7T1*

Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

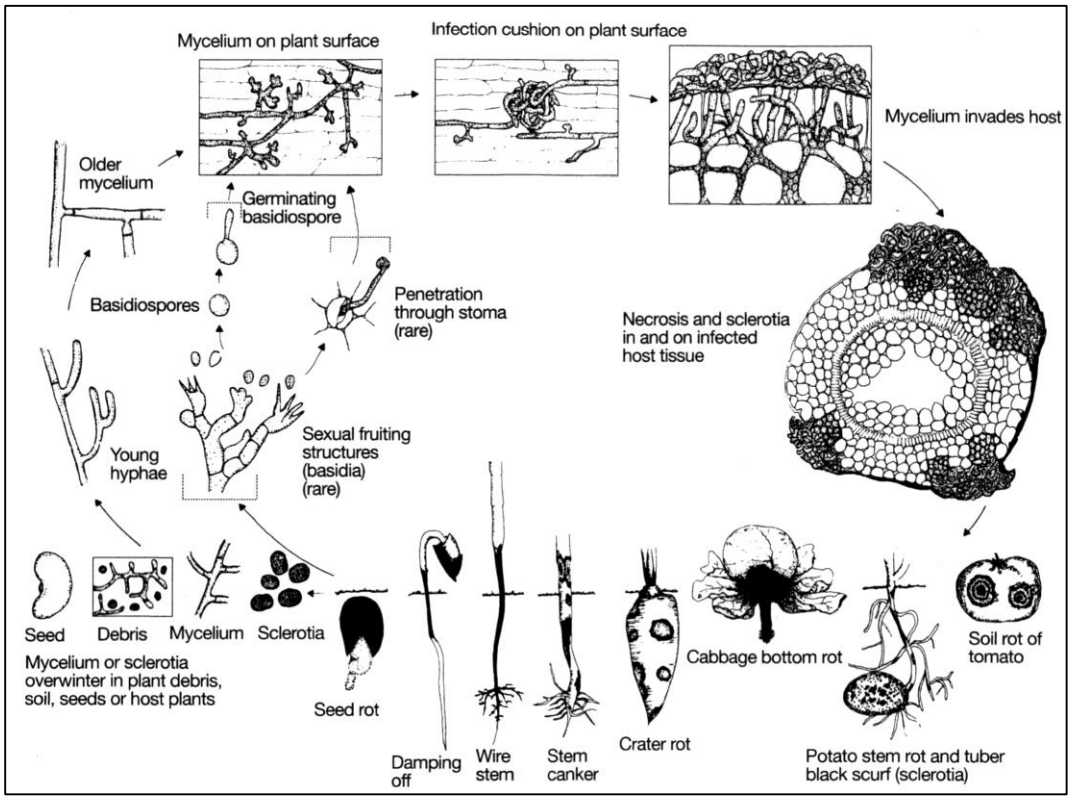
This disease can reduce stands and delay maturity. Yield losses in green bean pods of 13 to 54% have been associated with stand reductions of 24 to 50%. The pathogen, *R. solani*, has anastomosis groups, of which AG-4 and AG-2 type 2 are pathogenic to bean. AG-4 causes disease in many crops and AG-2 type 2 causes crown and brace root rot in corn.

Symptoms Elongate, sunken, reddish-brown lesions may occur on hypocotyls and roots (*15B.7*). Hypocotyls may be girdled as the lesions enlarge and grow together. Severe infections lead to damping-off of seedlings, or stunting or death of older plants. Older lesions may be rough and dry and may extend as a brown discoloration into the pith. Small, brown or black sclerotia may form inside the stem and occasionally on the surface of older lesions.

Causal agent *Rhizoctonia solani* commonly occurs as sterile, septate mycelium that is colorless when young and light brown when older. Hyphal cells are 5 to 12 µm wide and up to 250 µm long. Hyphal branches are typically constricted at the base, develop a septum near the base, and grow at right angles to the main hypha. Groups of broad, short, oval to triangular cells may form and act as chlamydospores or develop into black sclerotia. Black, barrel-shaped basidia of the perfect state develop under conditions of high humidity on a thin membranous layer of mycelium on soil or on moist plant surfaces. The basidia measure 10 to 25 µm in length and 6 to 19 mm in width. Sterigmata develop on a basidium, each producing a hyaline, oblong, unilaterally flattened basidiospore. The sterigmata number two to seven (mean = four) and measure 5.5 to 36.5 µm in length, while the basidiospores are 6 to 14 by 4 to 8 µm. The fungus is variable in appearance in culture, in host preference, and in response to environmental conditions.

Disease cycle The fungus overwinters as mycelium and as sclerotia in soil and plant residues (*15B.7T1*). It may be carried on or in bean seed. It is disseminated in infested soil or infected debris by wind, rain, irrigation water and machinery. Soil remains

infested indefinitely. *Rhizoctonia solani* can infect plants directly through the cuticle or through wounds or natural openings. Soil temperatures optimal for disease development depend on the strain of the fungus and may range from 15 to 27°C. Disease severity tends to be greater the drier the soil, but disease incidence is less affected by soil moisture content. Seedlings and young plants are more susceptible to infection than older plants. Low temperatures favor disease by reducing the growth rate of seedlings.



15B.7T1 Rhizoctonia damping-off and canker; disease cycle of *Rhizoctonia solani* on vegetable crops. Reprinted by permission from G.N. Agrios, *Plant Pathology.* © 1988 Academic Press.

Management

Cultural practices — Seed decay and damping-off can be controlled by using high quality seed that has high percentage germination and vigor, by treating seed with recommended fungicides, and by adopting practices that encourage rapid germination and emergence.

Control of the root rot complex in bean requires the integration of a number of production practices. The disease may be reduced by sowing seed as shallowly as possible into a well-prepared seed bed in warm, moist but not wet soil. Land preparation method that minimize soil compaction and structural damage will generally lessen disease severity. Bean crops should be rotated with a cereal or pasture crop. Cover crops and other practices to increase organic matter and improve soil structure are useful; for example, improving drainage to reduce excessive water content in soil. Practices that reduce soil compaction should be adopted, and fields can be indexed for their root rot potential. Growers are strongly advised to avoid incorporating a green manure crop into the soil immediately before planting bean, damaging roots by shallow cultivation, and planting in fields that are heavily infested with root rot organisms.

Resistant cultivars — Some cultivars have partial resistance to root rot.

Chemical control — Seed treatment with recommended fungicides can reduce seed decay and damping-off, but usually has little effect on root rot.

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(Original by R. Hall)

▶ 15B.8 Rust *Figs. 15B.8a,b*

Uromyces appendiculatus (Pers.:Pers.) Unger
(syn. *Uromyces phaseoli* (Pers.) G. Wint.)

Bean rust is most prevalent on common bean but has been reported on other species of *Phaseolus* and a few species of *Vigna.* It occurs wherever bean is grown, but is not important. Rust was once a major disease of pole bean in British Columbia, but production has since shifted to bush bean types on which the disease is a minor problem.

Symptoms The most obvious symptoms of rust are reddish-brown circular pustules (uredinia) (*15B.8a*), ranging up to 2 mm in diameter, that occur mostly on leaves, occasionally on petioles and pods, and rarely on stems. These lesions are first seen as minute, white, slightly raised spots that are often surrounded by a yellow halo. They rupture seven to nine days after infection and produce reddish- brown urediniospores. Larger uredinia are often surrounded by a yellow halo and may be surrounded by a ring of smaller, secondary uredinia. Severely infected leaves shrivel and drop prematurely. In the fall, the uredinia turn dark brown or black as a result of the production of black teliospores (*15B.8b*).

Causal agent *Uromyces appendiculatus* is an obligate parasite with an autoecious, macrocyclic life cycle. The fungus is extremely variable and more than 250 races have been identified. Infection by basidiospores leads to the production of spermagonia on the upper surface of the leaf. The spermagonia appear as chlorotic flecks, 2 mm in diameter, and produce a white nectar containing spermatia. After movement of spermatia to a sper- magonium of the opposite mating type, circular clusters of white aecia, 1 to 2 mm in diameter, form on the lower leaf surface. The aecia produce colorless, ellipsoid or oblong aeciospores, 20 to 28 by 18 to 20 µm, which infect the host to produce brown uredinia containing obovoid or broadly ellipsoid, cinnamon or golden brown, spiny urediniospores, 24 to 30 by 20 to 27 µm. The latter infect the host to produce new uredinia and urediniospores, several generations of which may occur in a season. Black or chestnut-brown, ovoid, ellipsoid or globoid, thick-walled teliospores, 28 to 33 by 22 to 27 µm, form within aged uredia. After a period of dormancy, the teliospores germinate to produce a metabasidium and four reniform to ovate-elliptical, smooth, hyaline basidiospores, 10.7 to 20.7 by 5.8 to 11.4 µm. Germination of teliospores is rarely reported and spermagonia and aecia are seldom observed.

Disease cycle Rust is most common in tropical and temperate areas where dew periods of 10 to 18 hours occur frequently. It is less common in arid regions. The teliospores can overwinter and produce basidiospores in the spring. Infection of bean plants by basidiospores leads to the development of spermagonia containing spermatia, and aecia containing aeciospores; infection by aeciospores leads to the production of urediniospores in the rust pustules. Spermagonia and aecia are rarely seen. Most bean rust infections originate from urediniospores.

Infection by urediniospores is favored when moisture films persist on the plant surface for 10 to 18 hours at moderate temperatures. Germination occurs in six to eight hours at 16 to 25°C, and not above 28°C. New urediniospores are produced in seven to nine days at 16 to 24°C and are favored by high humidity, long daylength and vigorous plant tissue. Urediniospores are spread by wind, tools, insects and animals to produce secondary infections throughout the summer. Teliospores form in the rust pustules in the fall.

Management

Cultural practices — Damage from rust can be reduced by rotating bean with non-host crops, discing under infested crop residues, and eradicating volunteer bean plants. Selecting planting dates and scheduling irrigation to minimize exposure of crops to long dew periods when temperatures are favorable for infection also are recommended. In pole bean production, poles should be disinfested before re-use if they have been used in an infected crop.

Resistant cultivars — Pole bean is generally more susceptible than bush bean to rust. Most bush bean cultivars are resistant to several races. Where the disease is a problem, the race involved must be identified in order to select an appropriately resistant cultivar.

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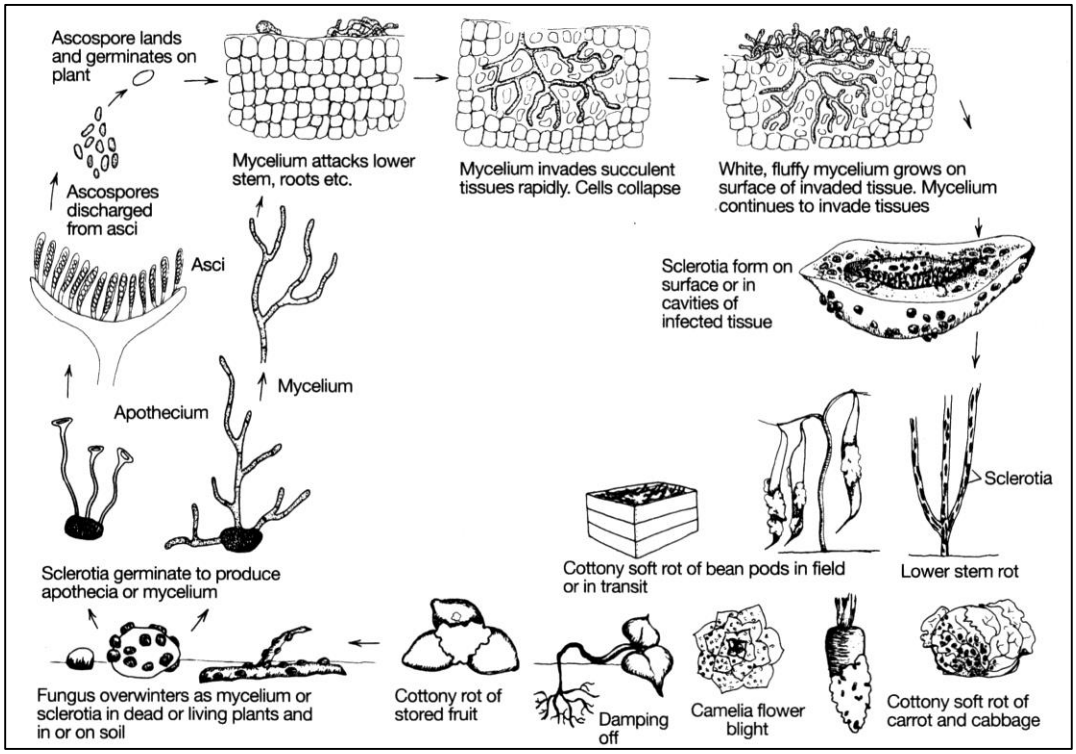
(Original by R. Hall)

▶ 15B.9 White mold *Figs. 15B.9a,b; 15B.9T1*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

White mold can attack over 360 species of plants, mostly herbaceous dicotyledons. It is a major disease of bean worldwide, especially in cool, moist regions or seasons and in irrigated crops, and it can cause complete crop loss.

Symptoms The fungus attacks all aerial parts of bean plants, as well as pods in storage and in transit. Symptoms develop during or after flowering of the crop. Lesions on pods, leaves, branches and stems are initially small, circular, dark green and water-soaked but rapidly increase in size and may eventually encompass and kill the entire organ and adjacent tissue. Affected tissues dry out and turn pale brown (*15B.9a*). Water-soaked spots can develop on any aerial part of the plant in contact with attached or detached flowers but usually first appear low on the stem at the points of attachment of branches or leaf petioles. Infected tissues may develop a white, cottony appearance as the fungus grows on the surface (*15B.9b*). Black, globose to elongate or irregular sclerotia form in infected tissue. Entire branches or plants may be killed and yields may be reduced by more than 50%. Apothecial cups of the fungus occur at or near the soil surface beneath the canopy.



15B.9T1 White mold (sclerotinia rot); disease cycle of *Sclerotinia sclerotiorum* on vegetable crops. Reprinted by permission from G.N. Agrios, *Plant Pathology*. © 1988 Academic Press.

Causal agent The mycelium of *Sclerotinia sclerotiorum* is hyaline, septate and branched. The fungus grows rapidly in standard culture media, producing white aerial mycelium and black sclerotia. Sclerotia produced in culture tend to be globose and usually develop at the margin of the colony. Those produced on infected plants may be rounded, elongate or irregular in shape, and are sometimes formed inside hollow stems. Sclerotia are 2 to 30 by 2 to 15 mm and have a black outer rind and a white inner cortex and medulla. One or more beige to salmon-colored apothecia may arise from a sclerotium under appropriate conditions. The stipe

is 3 to 30 mm long and 1 to 2 mm wide. The cup is 2 to 10 mm across, flat to concave when young, and flat to convex at maturity. Asci are cylindrical, have a thickened apex possessing a pore channel, and contain eight ascospores. The ascospores are 9 to 14 by 4 to 6 µm and are uniseriate, hyaline, ellipsoid and biguttulate, and contain two to four nuclei. Microconidia are globose, hyaline, 2 to 4 µm in diameter, and are produced from phialides in sporodochia, or on hyphae, or superficially on the hymenium surface or culture. The fungus is homothallic. Microconidia are thought to have a sexual function and no role in pathogenesis. Conidia are not produced.

Disease cycle The fungus can survive as sclerotia for five or more years in soil, in crop residues and in seed (*15B.9T1*). Sclerotia are preconditioned to produce apothecia by exposure to moist, cool (about 4°C) conditions for several weeks. To produce apothecia, preconditioned sclerotia require light, soil water potentials greater than -5 bars for one to several weeks, and temperatures of 11 to 20°C. Apothecia are produced from sclerotia lying within 2 to 5 cm of the soil surface. Apothecia generally do not develop until a dense crop canopy has formed, producing a cool, moist microclimate. The cup of the apothecium, formed at or above the soil surface, releases ascospores. Large numbers are often discharged simultaneously by "puffing" in response to physical disturbance or changes in relative humidity. Ascospores that initiate infections may have originated within the field or may have been carried by the wind for several kilometres. The ascospores need an exogenous source of energy in order to invade healthy bean tissue. Senescing bean flowers are the most common source of this energy. Bean plants become infected usually after flowering has started. Ascospores infect flowers and, in turn, mycelium from infected flowers invades adjacent tissue. Petals may be infected while attached or detached. Lesions typically develop where plant parts touch attached flowers or where fallen flowers become lodged, such as in axils of branches. Plant surfaces must remain continuously wet for 48 to 72 hours for infection to occur. Secondary infection by mycelial spread occurs between plant parts that are in contact. Disease develops most rapidly at 20 to 25°C, and not at all at 5°C or 30°C. Sclerotia are produced in infected pods, seeds, stems and branches. They may fall to the soil, remain with crop residues, or persist in the harvested pods and seeds. Infection of pods in storage or transit occurs by mycelial growth from lesions initiated in the field.

Management

Cultural practices — Crop rotation and deep plowing are of only limited value in destroying sclero- tia, which can survive for many years in soil. The wide host range of the fungus also restricts the value of rotation. Losses of fresh pods can be reduced by timely harvest, rapid cooling, and storage and transport under refrigeration. The disease is more common where canopies are dense, cool and moist. Canopy density can be reduced by increasing row width and by careful use of nitrogen fertilizer. Planting in the direction of the prevailing wind will facilitate drying of the rows. Conversely, irrigation increases soil moisture content and humidity of the canopy microclimate and thereby favors production and survival of apothecia and infection of the plant by ascospores and mycelium. The sclerotia can also be transported in irrigation water.

Resistant cultivars — Some cultivars have limited resistance to the disease.

Chemical control — Fungicide sprays can be effective if applied to cover the canopy thoroughly during the flowering period.

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(Original by R. Hall)

VIRAL DISEASES

▶ 15B.10 Bean common mosaic *Fig. 15B.10*

Bean common mosaic virus

Many strains of bean common mosaic virus occur, but strain 1 and New York strain 15 are prevalent in Canada. Bean common mosaic occurs worldwide and can cause losses of up to 100%. The natural host range of the virus includes common bean, some other species of *Phaseolus*, and *Rhynchosia minima* (L.) DC. Several other plant species, such as cowpea, lupin, and pea, have been infected with the virus experimentally.

Symptoms In plants susceptible to systemic invasion by the virus, leaves develop mosaic patterns in which irregular light and dark green patches are intermixed. The dark green areas grow more rapidly, causing puckering of the leaves (*15B.10*), which may be cupped downwards, curled and stunted. Plants may be dwarfed and pod and seed yields reduced. Systemic necrosis, in which the roots and shoot become blackened (black root symptoms), develops in cultivars with hypersensitive resistance conferred by the *I-* gene. These plants can become infected by necrosis-inducing strains at temperatures around 20°C or by other strains at

temperatures of 26 to 32°C. Reddish-brown necrotic lesions or spots (local lesions) may appear on cultivars resistant to systemic mosaic infection.

Causal agent Bean common mosaic virus is a member of the potyvirus group. Its particles are flexuous, filamentous rods, 12 to 15 nm wide and 730 to 750 nm long, and contain a single strand of RNA. Cytoplasmic inclusions in plant cells are common and occur as filaments, lamellates and pinwheels.

Disease cycle The primary source of the virus for infection of bean plants is infested bean seed. The virus multiplies and spreads systemically throughout the plant. Aphids feeding on bean plants can spread the virus in the crop in a nonpersistent manner. In addition, the virus can be transmitted mechanically by machinery and tools, as well as in pollen. Thus, several sources of the virus and mechanisms for its dissemination and inoculation are commonly available during the growing season.

Management

Cultural practices — If resistant cultivars are not available, seed should be certified free of bean common mosaic virus and perennial weedy legumes should be eliminated in the vicinity of the field. Also, bean should not be planted adjacent to crops harboring large aphid populations. Seeding should be timed to minimize the period during which the crop will be exposed to aphids migrating from other crops.

Resistant cultivars — The disease is controlled most effectively through the use of resistant cultivars. At least seven genes for resistance have been identified, including the dominant *I*-gene that confers hypersensitive resistance to many strains of the virus, five strain-specific recessive resistance genes, and a strain-nonspecific recessive gene.

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(Original by R. Hall)

▶ 15B.11 Bean yellow mosaic *Fig. 15B.1I*

Bean yellow mosaic virus

Bean yellow mosaic virus is worldwide in distribution and losses are reported to range from devastating to minor. Several disease resistance genes are available in bean and are widely used to protect the crop. The virus infects a wide range of legume plants, including bean, pea, alfalfa, clover, and vetch.

Symptoms A mosaic pattern of dark green and yellow areas develops on leaves *(15B.11),* often accompanied by bright yellow spots. Initial symptoms are small, chlorotic spots 1 to 3 mm in diameter, often surrounded by a halo. Enlargement of the spots produces general mottling symptoms on the leaf. However, symptoms are unreliable for a positive diagnosis of the disease. Many strains of the virus occur and symptoms range from mild, chlorotic mottle of leaves to severe mosaic and curling of leaves and stunting of the plant. Leaf symptoms may also include necrotic spots, veinal and apical necrosis, wilting, and premature death. Older leaves may become tough and leathery, and may drop prematurely. Pods may also become mottled and misshapen. Plants are usually stunted and bushy as a result of reduced internode length and development of lateral branches. Tips of shoots may rapidly wilt and die. The disease delays plant maturity and reduces the quality of seed and pods. Conclusive diagnosis requires laboratory testing of the sap by serological, microscopic, physical, and other techniques designed specifically to identify particles of the virus.

Causal agent Bean yellow mosaic virus is a potyvirus. Its particles are long, flexuous rods, 750 nm long and 15 nm wide, containing a single strand of RNA. The virus can infect most legumes and some non-legumes, such as gladiolus and pigweed (*Chenopodium* spp.). Cytoplasmic inclusions in cells of infected plants include crystals, spirals, rings and lamellate pinwheels.

Disease cycle Bean yellow mosaic virus is not carried in bean seed. The major source for infection of bean is perennial legumes. The virus is transmitted by more than 20 species of aphid, which can acquire it after feeding for a few minutes on an infected plant and are able to transmit it for several hours after acquisition. Plants become infected after a few minutes of feeding by an aphid carrying the virus. Secondary dissemination of the virus occurs by aphids and possibly by contaminated machines or tools. The virus has been easily transmitted mechanically in experimental studies.

Management Eradication of overwintering hosts is impractical, and application of insecticides to control aphids has not provided effective control of the disease.

Cultural control — If possible, avoid growing bean near perennial legumes, such as sweetclover (*Melilotus* spp.), or other host plants, such as gladiolus.

Resistant cultivars — Resistant cultivars are available for some types of beans and provide the most effective means of controlling the disease. A single, dominant gene *(By*-2) is able to provide resistance to many strains of the virus. Other resistance genes are known. Control of bean yellow mosaic in cultivars that lack resistance is difficult.

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(Original by R. Hall)

NON-INFECTIOUS DISEASES

▶ 15B.12 Nutritional disorders *Figs. 15B.12a-c*

Aluminum toxicity	Nitrogen deficiency
Boron toxicity	Phosphorus deficiency
Iron deficiency	Zinc deficiency
Manganese deficiency	

Nutritional disorders are rarely encountered in bean grown on fertile or fertilized soils. The major nutritional problems include deficiencies in phosphorus, nitrogen, manganese, iron and zinc, and toxicity caused by high levels of aluminum, manganese and boron.

Aluminum toxicity causes plants to be stunted and to have poorly developed root systems, numerous adventitious roots near the soil surface, and chlorotic lower leaves with necrotic margins. Aluminum toxicity results from high levels of aluminum in the soil, is associated with acidic soils, and is strongly correlated to phosphorus and calcium deficiencies. Aluminum toxicity is usually corrected by soil amendment with lime, at the rate of one to five tonnes per hectare to raise the soil pH to the range 6.0 to 7.2. Some cultivars of dry bean tolerate moderate levels of aluminum.

Boron toxicity may occur where bean is planted following a crop that was heavily fertilized with boron. Bean has a very low requirement for boron and toxicity symptoms may appear when the boron content in the soil exceeds 5 ppm. Boron toxicity causes yellowing and necrosis of the margins of the margins of older leaves and of primary leaves shortly after emergence.

Iron deficiency can occur on calcareous soils containing free calcium carbonate, in alkaline soils, or in acidic soils that have received excess lime. Excessive phosphate may precipitate iron as iron phosphate. A temporary deficiency may occur in less than 48 hours on younger leaves of bean plants grown in alkaline soils that have been saturated with moisture from rainfall or irrigation, especially if the days are cool. Symptoms of iron deficiency appear in young leaves, which become pale yellow almost white, while the veins remain green (*15B.12a*). Profuse, irregular necrosis may develop on severely chlorotic leaves. Fully expanded leaves curve downward and leaf tips may wilt. Young, unexpanded leaves may senesce. Iron deficiency can be corrected by soil application of iron chelates or foliar application of iron salts, such as 0.5% iron sulphate. Some cultivars are less sensitive than others to low levels of iron.

Manganese deficiency can occur in alkaline, organic, poorly drained, or over-limed soils. Symptoms (*15B.12b*) include interveinal chlorosis and fine speckling on younger leaves. These leaves may also appear pimply when examined closely. Older leaves are smoother and generally chlorotic. Pods may be yellow and unfilled. Plants are stunted. Foliar sprays of manganese salts, with 0.5% manganese sulphate, usually correct the problem. Manganese toxicity occurs in acidic volcanic soils. Poor drainage aggravates the problem. Symptoms may appear as purple-black spots on the stem, petiole, midrib and veins of leaves, especially on the lower surface. The pulvinus region does not discolor. Chlorosis may develop between major veins, especially on younger leaves. Affected leaves may cup downward and have necrotic margins. Some cultivars are less sensitive to manganese toxicity than others. Improved drainage, addition of organic matter, and soil amendment with lime may alleviate the problem.

Nitrogen deficiency can occur on all kinds of soils but is especially severe in sandy soils with low organic matter. Bean plants can meet some of their nitrogen needs by fixing atmospheric nitrogen in the presence of appropriate strains of *Rhizobium* bacteria, but they may require additional nitrogen. Deficiency symptoms appear as a uniformly pale green to yellow discoloration of all except the younger leaves. Growth is reduced, few flowers develop, and pods may fill poorly. Nitrogen deficiency can be corrected by the application of nitrogen fertilizer and organic matter.

Phosphorus deficiency can occur on many soils, especially those with low pH. Symptoms appear initially on upper leaves, which are small and dark green. Older leaves may turn brown and senesce prematurely. Plants are often stunted and have thin stems and shortened intemodes. The vegetative period may be prolonged, whereas the flowering phase may be delayed and shortened. Often, many flowers abort and the number of pods and seeds may be reduced. Phosphorus deficiency can be

controlled chemically by band application of various rock phosphates or superphosphate fertilizers. Cultivars differ in their sensitivity to phosphorus deficiency.

Zinc deficiency can occur in soils with a high pH or in acid soils that have received too much lime or phosphorus. The problem can also be aggravated by soil compaction, low organic matter and excessive applications of manure or crop residues. Elevated absorption of other nutrients, such as iron, can also induce zinc deficiency. Soon after emergence, younger leaves develop an interveinal chlorosis (*15B.12c*) and become deformed, dwarfed and crumpled. Older leaves may develop necrotic areas in and between veins. Terminal blossoms and pods may abort. If the deficiency is severe, new leaves are white and plants may die. Deficiencies may appear in spots within a field or throughout an entire field. Zinc deficiency can be corrected by soil (6 to 12 kg/ha) or foliar (0.5% solution) application of zinc sulphate. Some cultivars are less sensitive to low levels of zinc than others.

▶ 15B.13 Other disorders *Figs. 15B.13a-e*

Baldhead
Herbicide injury
Ozone injury
Sunscald
Wind injury

Baldhead

Baldheads (*15B.13a*) are plants with broken or dead growing points caused by mechanical damage to the seed. These plants may develop vegetative tissue from the axillary buds at the cotyledonary nodes but they seldom yield well. A similar injury called snakehead can be caused by the seedcorn maggot, which leaves a ragged edge around affected plant parts. Internal contamination by bacterial pathogens can also damage or kill the growing point of a germinating seedling.

Herbicide injury

If herbicides are not applied according to the manufacturer's recommendations, bean plants may be damaged during the growing season, especially during germination and seedling development. Herbicide drift from nearby sprayed areas can also damage bean. The symptoms depend on the kind of herbicide and may include color changes, such as increased greening, yellowing, or browning of leaves, and distorted growth of shoots (*15B.13b*). Leaves may show a wide range of distortions in shape, such as twisting, puckering and string-like margins.

Ozone injury

Ozone (0_3) is a common air constituent formed by electrical discharge during thunderstorms. However, photochemical production from gases liberated by combustion engines is the most important source of phytotoxic ozone. Symptoms of ozone injury, or bronzing (*15B.13c*), appear on the upper leaf surface first as small, water-soaked or necrotic lesions that may coalesce and become bronze or reddish-brown, resembling sunscald. The upper surface of the leaf may have a glazed appearance. Premature senescence and defoliation may then occur. The severity of damage is affected by ozone concentration, cultivar sensitivity, leaf age, light intensity or cloud cover, temperature, humidity, soil moisture and texture, and plant nutrition. There is no effective way to control ozone injury.

Sunscald

Sunscald of bean leaves, stems, branches and pods may occur during periods of intense sunlight, especially after conditions of high humidity and cloud cover. High temperatures also may induce sunscald damage. Symptoms appear as small, water-soaked spots on the exposed side of the plant. The spots become reddish or brown and may grow together to form large necrotic or discolored lesions (russetting) (*15B.13d*) on affected plant structures. These symptoms may resemble those caused by spider mites or ozone. Flowers and pods may abort.

Wind injury

Wind speed and direction can affect bean plant development. Water loss from the plant may be increased by wind and thereby aggravate moisture stress caused by low soil moisture. Violent wind movement may damage roots and predispose them to root rot problems, break stems and branches, and cause plant lodging, especially if soil moisture is high. Leaves may have whitish necrotic areas (*15B.13e*) or be abraded, tom or shredded. The plant also can be damaged by the abrasive action of wind-blown soil. Pathogens such as blight bacteria may enter the wounded areas.

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(Original by R. Hall)

NEMATODE PESTS

▶ 15B.14 Northern root-knot nematode *Fig. 7.15b*

Meloidogyne hapla Chitwood

Symptoms include wilting and yellowing of foliage, prolific branching of rootlets, and production of small, spherical galls on roots. For a complete description and management strategies, see Carrot, northern root-knot nematode, 6.20; see also Management of nematode pests, 3.12.

▶ 15B.15 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms include wilting and stunting in patches in heavy infestations; leaves become yellow. Secondary roots become necrotic, with dried areas. For a complete description, see Potato, 16.38, and Management of nematode pests, 3.12.

▶ 15B.16 Stubby-root nematodes

Paratrichodorus allii (Jensen) Siddiqi
Paratrichodorus pachydermus (Seinhorst) Siddiqi
Paratrichodorus spp.
Trichodorus spp.

Symptoms include proliferation of short roots; damage is rare in Canada. See Potato, 16.39.

INSECT PESTS

▶ 15B.17 European corn borer *Figs. 12.16fg*

Ostrinia nubilalis (Hübner)

European corn borer (for identification and life history, see Maize, 12.16) can be a problem in snap bean grown for processing because larvae may occasionally be found in the finished product. Snap bean plants in flower during the corn borer's oviposition period appear to be most susceptible and damage is most likely to occur when the bean crop is grown near fields that were in corn the previous year. Only intensive pest management will allow production of snap bean in close proximity to corn.

Damage In snap bean, corn borer larvae begin feeding in the stems, then they transfer to the developing pods. In bean crops destined for canning in Quebec, the one-generation corn borer, which is also most damaging in field corn, causes most concern. In Essex County in southwestern Ontario, snap bean for freezing is most often threatened by the first generation of the two-generation corn borer. Infestations of corn borer are never high enough to reduce yield but the presence of one corn borer larva per 1000-pod sample is sufficient reason to reject the whole field for either freezing or canning.

Identification (see Maize, 12.16)

Life history (see Maize, 12.16)

Management It is nearly impossible to find corn borer larvae in snap bean fields because the crop is very dense, with numerous pods on each plant, and the larvae are present in very low numbers.

Monitoring — Corn borer adults can be monitored with pheromone or light traps in corn fields adjacent to snap bean fields. In Quebec, this is done in collaboration with a provincial agency, Le Réseau d'Avertissements Phytosanitaires du Québec (RAPQ), located at the Service de Phytotechnie de St-Hyacinthe, where the seasonal progress of corn borer oviposition is followed. This information is passed on to growers and processors who then decide when to treat, the number of treatments, and the fields to be treated.

Cultural practices — The most practical and effective way to avoid the risk of corn borer infestation is to avoid planting snap bean near corn or near fields that were in corn the previous year.

Chemical control — If chemical control is necessary, it should be applied when the bean crop begins to flower and corn borer adults are present, and timed to coincide with the earliest appearance of corn borer egg masses if the seasonal progress of oviposition is being followed.

(Original by C. Ritchot and D.G.R. McLeod)

▶ 15B.18 Seedcorn maggot *Fig. 15B.18*

Delia platura (Meigen)

The seedcorn maggot occurs across southern Canada and extends into the Yukon and Northwest territories. It usually is not a threat to vegetable crops because it normally feeds on decaying vegetable matter. On occasion, however, it may devastate a field.

Vegetable crops, in general, and bean, pea, cucumber and corn, in particular, are vulnerable to attack while the seeds are germinating. Seedling stems, seeds and seed pieces in the ground, and roots and tubers of a wide range of vegetable crops may be affected. The seedcorn maggot is most injurious in cold, wet springs when germination is slow.

Damage In pea and bean crops, the seedcorn maggot larva develops in the cotyledons (causing snakehead; see bald- head, 15B.13) and root collar of young seedlings (*15B.18*). The plants may wilt and die by the time they emerge through the soil surface. Damage is not usually widespread in a region. However, severely damaged crops may require reseeding. Often, in adjoining bean fields sown on slightly different dates, one field will be almost totally ruined by the seedcorn maggot; other fields may be quite unaffected.

Identification The seedcorn maggot (family Anthomyiidae) adult is a small, 5 mm long, gray fly. The egg has net-like surface sculpture and a ventral groove that extends only about one third the length of the egg. The legless larva is white, plump, and tapered at the anterior end with small black mouthhooks. At maturity, the larva is about 5 mm long. The pupa (puparium) has a pair of unforked, median posterior tubercles.

The seedcorn maggot is the only maggot that attacks the seeds of bean and pea crops at germination and infests the cotyledons and stems of the seedlings. (For comparison with the cabbage maggot, see Crucifers, 8.41.)

Life history *Delia platura* overwinters as a pupa in the soil. In southern Ontario and Quebec, there may be four generations per year. Adults begin to appear in the spring when the temperature is favorable, which may be late April in southern Ontario or during May in Quebec. Eggs are commonly laid in moist soil with an abundance of decaying plant residue. They may hatch at temperatures as low as 10°C. First-generation eggs appear in May or June, depending on the region, and second-generation eggs can be found from the beginning of June in southern Ontario and from the end of June in southern Quebec. Subsequent generations overlap and egg laying continues at low levels until the end of the growing season. First- and second- generation larvae cause most damage in Ontario and Quebec.

Management

Cultural practices — Clean cultivation is important because rotting vegetation is particularly attractive to the female flies. Treated seed should be used wherever seedcorn maggot is a problem.

Chemical control — Insecticidal seed treatments are recommended for control of the seedcorn maggot in Canada.

(Original by C. Ritchot)

▶ 15B.19 Other insect pests *Figs. 15B. 19a, b; see text*

Cutworms
European earwig *Forficula auricularia* L.
Mexican bean beetle *Epilachna varivestis* Mulsant

Cutworms

Various species of cutworm (*6.25a-c; 18.35a-g)* affect mainly the seedlings of bean crops, sometimes making reseeding necessary, especially in home gardens. (For more on cutworms, see Carrot, 6.25; and Tomato, 18.35.)

European earwig

(see Crucifers, 8.43) Earwigs (*8.43b-d)* may cause ragged holes in bean leaves (*15B.19a*).

Mexican bean beetle

The Mexican bean beetle (family Coccinellidae) is an occasional pest of bean. The spiny larva and spotted adult (*15B.19b*) feed together on the underside of leaves, skeletonizing them. Stems and pods can be damaged, but infestations in Canada are rarely cause for concern.

(Original by C. Ritchot and J.N. McNeil)

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16 Potato

Figures *16.1* to *16.50; 16.2T1; 16.5T1; 16.11T1; 16.38T1; 16.43T1, T2; 16.44T1; 16.49T1, T2*
Table 16.43

Bacterial diseases
16.1 Bacterial ring rot (ring rot)
16.2 Bacterial soft rot (soft rot)
16.3 Blackleg
16.4 Pink eye (brown eye)
16.5 Scab (see also 16.16)
- Common scab
- Acid scab
- Russet scab

Fungal diseases
16.6 Black dot
16.7 Dry rot
16.8 Early blight
16.9 Fusarium wilt
16.10 Gray mold
16.11 Late blight
16.12 Leak
16.13 Phoma rot (button-hole rot, pocket rot)
16.14 Pink rot
16.15 Rhizoctonia canker (black scurf)
16.16 Scab, powdery (see also 16.5)
16.17 Seed-piece decay
16.18 Silver scurf
16.19 Skin spot
16.20 Verticillium wilt
16.21 Wart (canker)
16.22 White mold

Viral and viral-like diseases
16.23 Aster yellows (haywire, purple dwarf, purple-top wilt)
16.24 Calico
16.25 Corky ring spot (spraing, stem mottle)
16.26 Leafroll
16.27 Mosaic diseases
- Potato virus A
- Potato virus M
- Potato virus S
- Potato virus X
- Potato virus Y

16.28 Spindle tuber
16.29 Witches'-broom

Non-infectious diseases
16.30 Blackheart
16.31 Growth cracks
16.32 Hollow heart
16.33 Jelly end rot
16.34 Other physiological disorders
- Genetic abnormalities
- Herbicide injury
- Internal black spot
- Internal sprouting
- Nutritional disorders
- Secondary tubers
- Stem-end browning
- Tuber greening
- Miscellaneous disorders

Nematode pests
16.35 Northern root-knot nematode
16.36 Potato cyst nematodes
- Golden nematode
- Pale cyst nematode

16.37 Potato-rot nematode
16.38 Root-lesion nematode
16.39 Stubby-root nematodes

Insect pests
16.40 Buckthorn aphid

16.41 Green peach aphid
16.42 Potato aphid
16.43 Other aphids
- Black bean aphid
- Bulb and potato aphid
- Crescent-marked lily aphid
- Foxglove aphid
- Melon (cotton) aphid

16.44 Colorado potato beetle
16.45 Potato flea beetle
16.46 Potato leafhopper
16.47 Potato stem borer
16.48 Tuber flea beetle
16.49 White grubs (June beetles)
- Common June beetle
- Other June beetles

16.50 Wireworms
- Dusky (European) wireworm
- Eastern field wireworm
- Wheat wireworm

16.51 Other insect pests
- Aster leafhopper
- Blister beetles
- Cutworms
- European corn borer
- Grasshoppers
- Potato psyllid
- Potato scab gnat
- Seedcorn maggot
- Stalk borer
- Tarnished plant bug

Other pests
16.52 Millipedes
16.53 Slugs
Additional references
Table 16.43
Key to wingless female aphids colonizing potato

BACTERIAL DISEASES

▶ 16.1 Bacterial ring rot (ring rot) *Figs. 16.1a-e*

Clavibacter michiganensis subsp. *sepedonicus* (Spieckermann & Kotthoff) Davis *et al.*
(syn. *Corynebacterium sepedonicum* (Spieckermann & Kotthoff) Skapston & Burkholder)

Bacterial ring rot is a very serious potato disease because of its destructiveness and highly infectious nature. It was originally described in Germany in 1906 and was first reported in North America in 1931. It often has caused major crop losses in Canada, but since the early 1970s rigid seed inspection procedures and disease control programs have kept outbreaks to a minimum. The objective in Canada is to systematically eradicate bacterial ring rot (see Introduced diseases and pests, 3.11).

In nature, the ring rot organism infects only potato, but under laboratory conditions it is also able to infect tomato, eggplant and a number of other *Solanum* species. The pathogen has been isolated from sugarbeet seed.

Symptoms Symptoms in the field vary with cultivar and overall crop condition and can range from nothing visible to distinctive symptoms. Foliar symptoms usually do not appear until after flowering, but certain cultivars, such as Russet Burbank, may develop an early season dwarf- rosette. More conspicuous foliar symptoms generally appear at mid-season or later, and usually begin on middle and lower leaves *(16.1a).* Wilted leaflets are slightly rolled at the margins, and light green to pale yellow areas develop in the interveinal spaces. As wilt progresses, the affected foliage becomes necrotic and symptoms move upward until the entire stem wilts and dies *(16.1b).* The most important diagnostic symptoms in the field are one or more wilted stems in a hill and a milky exudate that can be squeezed from the stem near the point of attachment to the mother tuber piece. Vascular tissues in these stems may appear brown.

The characteristic tuber symptom of ring rot is a decay of the vascular ring tissues *(16.1c).* Vascular discoloration is usually most apparent at the stem end of the tuber, which may exude a milky or cheezy ooze when squeezed. This decay usually occurs after the appearance of foliar symptoms, but it does not always develop in the tubers of infected plants.

Badly affected tubers *(16. 1d)* exhibit ragged skin cracks, reddish-brown discoloration near the eyes, and may collapse into a semi-liquid soup when handled *(16.1e).* Secondary invaders, notably soft rot bacteria, can cause a foul-smelling decay that masks the symptoms of ring rot.

Under cool growing conditions or in fields with high levels of soil nitrogen, plants may not develop symptoms but still produce infected tubers. Infected tubers may be symptomless at harvest and can take two or three months to develop ring rot symptoms in storage, if at all.

Causal agent Cells of *Clavibacter michiganensis* subsp. *sepedonicus* are Gram-positive, club-shaped, non-motile rods, measuring 0.4 to 0.6 by 0.8 to 1.2 pm. Colonies on nutrient agar are white, thin, translucent and glistening. Cells are non-acid-fast. Gelatin is not liquefied and nitrates are not reduced. Acid is produced from arabinose, xylose, dextrose, galactose, fructose, sucrose, maltose, cellobiose, mannitol and salicin, but not rhamnose. The optimum temperature for growth in culture is 18 to 21°C, with a maximum of 30°C.

Laboratory diagnostic techniques for confirming the presence of this pathogen from typical ring rot or symptomless tubers are varied. The Gram-staining technique, while reliable, has been superceded largely by the immunofluorescent antibody staining (IFAS) and latex agglutination tests. These highly specific antibody systems can detect extremely low levels of ring rot bacteria and are particularly useful for checking symptomless potatoes. The enzyme-linked immunosorbent assay (ELISA) test also offers sensitivity and specificity. It is used routinely in many provinces to detect both bacteria and viruses in potatoes. DNA probes are under development and should soon be available for general use.

Disease cycle The bacteria normally overwinter in infected tubers in storage, or in mild climates in the field. Infected tubers may not exhibit disease symptoms. The organism can survive many months on dry surfaces such as bags, crates and walls at temperatures below freezing. The viability of the organism is rapidly lost in warm, moist soil.

Tuber infection generally occurs through wounds from seed-cutting knives, picker planters and harvesters. Conditions for the spread of ring rot are most favorable at planting when tubers are cut into seed pieces. Bacteria from infected tubers may be smeared by cutting knives onto freshly cut seed surfaces. Extremely high levels of infection can occur from very few infected tubers in a multi-tonne seed lot. If infection levels or diseased tubers exceed 5% in the field, there is a high risk of the entire crop rotting in storage. Reports from the United States suggest that the Colorado potato beetle and green peach aphid can vector the pathogen.

No potato cultivars are immune, but some, such as Désirée, Rose Gold, BelRus, Urgenta and Teton, rarely exhibit symptoms yet may harbor the bacterium. There is also a range of susceptibility among cultivars; for example, Norchip, Red Pontiac and Shepody nearly always exhibit symptoms, whereas Russet Burbank and Red La Soda do not always show typical symptoms. More importantly, even among susceptible cultivars, individual plants may remain symptomless. This so-called “latency” is the major reason bacterial ring rot cannot be eradicated by visual inspection alone.

Management

Cultural practices — Planting Certified or higher class disease-free seed tubers and following strict sanitation procedures are the most practical control measures. If ring rot is confirmed on a farm, the grower should take the following measures to eradicate the disease:

- Dispose of all potato stocks by using them for food or feed;
- Thoroughly wash with a high pressure spray the storage area, pallets and all machinery involved in the handling of potatoes during planting, harvesting, storage and grading, and then disinfest them with products such as formaldehyde, commercial bleach, and quaternary ammonium solutions specifically formulated for this purpose are effective; washing and disinfesting should be done daily, and always between seed lots, when planting;
- Dispose of all used potato bags;
- Plant only Certified or a higher class of seed;
- Plant small, whole (uncut) seed tubers;
- Follow a three-year crop rotation, particularly where the climate is mild enough to allow volunteer potato plants to overwinter in the field;
- Avoid growing potato for at least two to three years after sugar beet has been grown in the same field;
- Control potential insect vectors such as the Colorado potato beetle and green peach aphid.

The Canadian certification system has a zero tolerance for this disease. All potatoes grown for seed are visually inspected for ring rot, and a major proportion of the seed hectarage is also checked by post-harvest laboratory tests. Seed potato growers who have had crops affected by bacterial ring rot must follow rigorous sanitation procedures before they will be permitted to produce seed potatoes again. Any fields in which ring rot was found cannot be replanted to potato for at least two years.

Resistant cultivars — No cultivars are known to be immune. The growing of resistant cultivars is discouraged in most areas of Canada, because they can be symptomless carriers of ring rot bacteria and a source of infection for susceptible, main crop cultivars. This risk can be reduced by requiring all growers to plant certified seed.

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(Original by I.R. Evans and B. Otrysko)

▶ 16.2 Bacterial soft rot (soft rot) *Figs. 16.2a,b; 16.2T1*

Bacillus spp.
Clostridium spp.
Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*
Erwinia carotovora subsp. *atroseptica* (van Hall) Dye
Flavobacterium spp.
Pseudomonas spp.

Soft rot can damage potato in the field and storage. Losses may be severe, especially in storage. Soft rot bacteria can attack a wide range of root, fruit and leafy vegetables, such as carrot, cabbage, onion and tomato.

Symptoms Bacterial soft rots generally occur with other diseases such as ring rot, late blight, leak and blackleg. Symptoms usually are confined to the tubers, which may be infected through lenticels, causing the surrounding tissue to collapse and form brown sunken lesions up to 1 cm in diameter *(16.2a).* If a tuber becomes badly bruised during handling, the whole tuber may become infected. Infected areas are cream-colored and later become brown, slimy and foul smelling *(16.2b).* A sharp line in the tuber separates diseased from healthy tissue. Wet growing conditions can result in infected tubers rotting in the ground. Stored potatoes can be seriously damaged if frozen or harvested under wet conditions.

Causal agents *Erwinia carotovora* subsp. *carotovora* is a rod-shaped, Gram-negative bacterium, 0.5 to 0.8 by 1.0 to 3.0 µm, with peritrichous flagella. It occurs singly, in pairs or as short chains, does not form spores, and is facultatively anaerobic. (For a description of *Erwinia carotovora* subsp. *atroseptica,* see blackleg, 16.3.)

Erwinia bacteria can be readily isolated from plant tissue taken from the edge of a rotted area. When plated onto selective media containing polypectate, such as Stewart-MacConkey or Cuppels-Kelman crystal violet pectate medium, these bacteria form deep pits or craters in the agar. Other aerobic, pectolytic bacteria that may be associated with soft rot, such as *Bacillus, Pseudomonas* and *Flavobacterium* spp., generally grow poorly or only form shallow pits on pectate media.

Most strains of *E. carotovora* subsp. *carotovora* do not form acid from α-methyl glucoside or reducing substances from sucrose. They do not grow on nutrient agar or in nutrient broth at temperatures greater than 36°C. Most strains do not produce acid from maltose. Serological techniques can be used to distinguish soft rot from blackleg bacteria.

Disease cycle Soft rot bacteria can survive for several months in the soil and can be dispersed by irrigation water *(16.2T1).* They usually invade tubers following mechanical, freezing or insect injury and following damage by other disease organisms. Soft rot is favored by high temperatures and abundant soil moisture. Immature tubers are more susceptible to rot than mature ones.

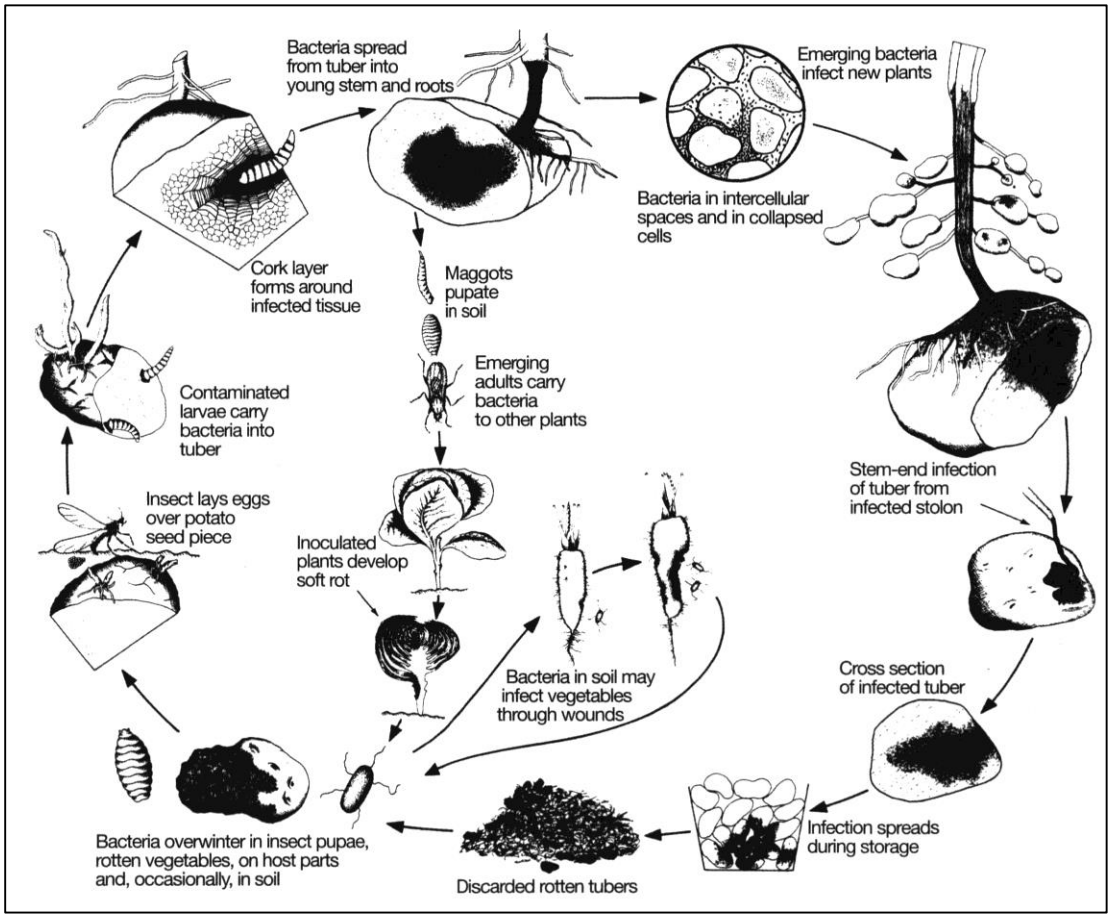
The disease can spread from infected to healthy tubers during storage or transit under conditions of high humidity and poor ventilation. Washing potatoes in dirty water and packaging warm, wet tubers in poorly ventilated plastic bags promote soft rot development.

Management

Cultural practices — Potato should be grown in well-drained, fertile soils, and only mature tubers should be harvested. Vine killing before harvest hastens tuber maturation. Potatoes that are to be packaged soon after harvest should be dug when soil

and air temperatures are low to cool the tubers. Controlling other tuber diseases and the careful handling of tubers to minimize wounds and bruises minimizes soft rot. Growers should follow the disease management strategies described for blackleg.

When washing table-stock tubers, the water should be changed frequently to avoid the build-up of high bacterial populations. Prolonged soaking should be avoided. Clean water should be used for the final rinse to cleanse the tubers and they should be dried before packaging. Seed potatoes should not be washed, thus avoiding damage and moisture build up on the tubers that could favor soft rot infection and increase seed piece decay.



16.2T1 Bacterial soft rot; disease cycle of *Erwinia* spp. on vegetable crops. Reprinted by permission from G.H. Agrios, *Plant Pathology.* © 1988 Academic Press.

Chemical control — Chlorination of rinse water reduces the residual populations of soft rot bacteria on tubers before packaging for market. Growers and packers should consult the Health Protection Branch, Health Canada, for guidelines on using chlorinated water on vegetables.

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(Original by I.R. Evans and R.J Howard)

▶ 16.3 Blackleg *Figs. 16.3a—c*

Erwinia carotovora subsp. *atroseptica* (van Hall) Dye
Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*

Blackleg occurs in all potato-growing areas and is often found in conjunction with soft rot. This disease can cause heavy losses in both seed and table stock crops. Blackleg severity depends on seed-handling techniques, amount of inoculum on seed tubers, soil moisture and temperature at planting, growing conditions, the cultivar used, and on external sources of bacteria. Potato is the only

agricultural crop affected by *Erwinia carotovora* subsp. *atroseptica*, whereas *E. carotovora* subsp. *carotovora* has a wide host range that includes many vegetable crops.

Symptoms Seed piece decay can significantly reduce germination and plant stands. The disease is generally first noticed at flowering, when one or more stems on a plant suddenly wilt. Wilting is usually most apparent during hot weather and may be accompanied by leaf yellowing *(16.3a).* The lower part of the stem often appears grayish brown or black. The stem may later turn an inky black, generally from just above the soil line to around 15 cm up the stem *(16.3c).* Blackleg occurs with varying degrees of intensity in a crop and is most frequently seen in low areas of fields. Affected stems become soft above and below the soil line.

Blackleg can stunt the growth of young potato plants. On older plants, interveinal yellowing and browning and the upward cupping of leaves are characteristic symptoms. Under some conditions, affected plants appear stiff and erect. In certain cultivars, wilting and leaf symptoms caused by blackleg resemble those caused by ring rot, except that the tubers do not show the vascular decay typical of ring rot.

Blackleg bacteria also can infect and cause rotting of tubers, but such decay is not always associated with plants that have typical foliar symptoms. Tuber symptoms can range from a slight vascular discoloration to complete soft rot *(16.3b).* Infection usually begins at the stem end and appears as a creamy, odorless, soft decay that is sharply separated from the healthy tissue by a dark brown to black line. Infected lenticels are usually surrounded by sunken, water-soaked, circular lesions.

Infection by *Erwinia carotovora* subsp. *carotovora* can also produce blackleg symptoms under warm growing conditions.

Causal agent *Erwinia carotovora* subsp. *atroseptica* is a Gram-negative rod that occurs as single or paired cells or occasionally in chains of a few cells. It is 0.5 to 0.8 by 1 to 2.5 µm, motile by peritrichous flagella and facultatively anaerobic. It can be distinguished from *E. carotovora* subsp. *carotovora* (see bacterial soft rot, 16.2) by its failure to grow at 37°C and by the production of reducing substances from sucrose and acid from maltose and a-methyl glucoside.

Disease cycle Blackleg bacteria are borne in or on seed tubers, on the roots of some crops, such as cereals and sugar beet, and on the roots of weeds such as nightshade, lamb's-quarters, pigweed, Russian thistle, kochia, purslane and mallow (see Weeds, 2.3). Blackleg bacteria can also be dispersed in irrigation water. They probably do not survive in the soil for more than a year in the absence of a host. The bacteria can readily spread from diseased to healthy tubers during seed cutting, and infection is favored if wound healing is delayed. Bacteria can cause decay of infected seed pieces before sprouts emerge, especially if the soil is wet. In some cases, infected seed pieces decay slowly and the bacteria move into vascular tissues of stems after the sprouts emerge. *Erwinia* species can multiply in the stems, degrade the vascular and pith tissues and cause foliar symptoms. During the growing season, bacteria from decaying seed pieces may move as far as 60 cm in soil water and contaminate new tubers. In addition, bacteria from decaying stems and tubers may contaminate non-infested tubers during harvest. Infested tubers may rot in storage, further spreading this disease, or the bacteria may survive the winter in tuber lenticels. These infested tubers, if planted the following spring, can decay and allow the disease cycle to repeat.

Culled tubers are an important source of the blackleg bacteria. As they decay, large numbers of bacteria are produced. Insects such as flies may move these bacteria to non-infested potato crops.

Wet soil and low temperatures (<18°C) favor infection of tubers and spread of blackleg bacteria. Immature tubers are more susceptible to infection than those with a mature skin. Proper curing after harvest followed by consistent low storage temperatures (see dry rot, 16.7) prevent or restrict tuber soft rot. Moisture from soft-rotted tubers favors disease development and the spread of infection in storage.

Management

Cultural practices — Use of blackleg-free seed and good growing practices are the best means of controlling this disease. Seed from stem cutting or micropropagation programs is usually the most suitable. Seed tubers should be warmed before cutting by storing them at 10 to 13°C in 95% relative humidity for 10 to 14 days. Ideally, small whole seed should be planted, If seed is cut, good sanitation practices such as disinfesting cutting and handling equipment daily and between seed lots, should be followed. Cut seed should be treated with a recommended fungicide and planted immediately. Pre-cut potato seed pieces should be suberized at 10°C and 95% relative humidity for five to seven days before planting to reduce infection by *Fusarium* spp. and other pathogens that may predispose plants to bacterial soft rot. Potatoes should be planted in moist, well-drained soil when soil temperatures are 10°C or higher. Excessive irrigation must be avoided. At least two or three years should be allowed between successive potato crops in the same field. Cereals, grasses and forages are preferred rotation crops.

Culled potatoes and vegetables should be properly composted, buried or fed to livestock to prevent the spread of *Erwinia* spp. to potatoes by insects. Planters, harvesters and conveyers should be cleaned frequently and disinfested to lessen the risk of contaminating healthy tubers. Seed potatoes should not be washed and they should be handled carefully to minimize damage. If practicable, seed growers should remove infected plants as soon as they appear in the field.

Tubers should be allowed to mature before harvest. Freshly harvested crops should be cured and stored as outlined under the management strategies for dry rot.

Resistant cultivars — Russet Burbank has intermediate resistance to blackleg. All other cultivars commonly grown in Canada are susceptible.

Chemical control — Fungicidal seed treatments that are effective against fusarium seed-piece decay may reduce the severity of blackleg and bacterial soft rot.

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(Original by I.R. Evans, J.R. Letal and R.J. Howard)

▶ 16.4 Pink eye (brown eye) *Figs. 16.4a,b*

? *Pseudomonas fluorescens* (Trevisan) Migula

Pink eye occurs chiefly in eastern Canada. It is usually a minor disease of potato. *Pseudomonas fluorescens* occurs widely in nature in soil and water, and it is often associated with food spoilage. It is epiphytic and is often isolated from diseased plants. Some strains are ice-nucleation-active and may predispose plants to frost injury.

Symptoms Pink to brown, skin-deep patches are found around eyes or at the apex of tubers during harvest *(16.4a).* The skin of affected tubers dries and cracks easily. Internal reddish-brown to black discoloration, cavities and soft rot are often associated with severe pink eye *(16.4b).* Diseased tubers usually fluoresce under ultraviolet light.

Causal agent (see Lettuce, pseudomonas diseases, 11.3)

Disease cycle A consistent association with a pathogen has not been established for this disease. Pink eye is sometimes associated with verticillium wilt, rhizoctonia canker and late blight, but it can occur on tubers free from these diseases. *Pseudomonas fluorescens* survives on organic matter in the soil and is generally a saprophyte rather than a pathogen. It requires high soil moisture for entry through lenticels, or damage such as wounds or other diseases for direct invasion. Under warm (greater than 7°C), moist storage conditions, the bacterium can spread to other tubers through wounds and lenticels.

Management

Cultural practices — Avoiding large fluctuations in soil moisture, especially late in the season, may reduce pink eye incidence. It may be possible to arrest pink eye development in storage by lowering the temperature, reducing the humidity and increasing air flow through the pile. Tubers with severe pink eye should be processed immediately.

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(Original by I.R. Evans and R.J Howard)

▶ 16.5 Scab *Figs. 16.5a-c; 16.5T1*

Common scab

Streptomyces scabies (Thaxt.) Waksman & Henrici
(syn. *Actinomyces scabies* (Thaxt.) Güssow)
Streptomyces spp.

Acid scab

Streptomyces acidiscabies Lambert & Loria

Russet scab

? *Streptomyces aureofaciens* Duggar

Common scab, acid scab and russet scab are unsightly diseases of potato tubers that can adversely affect grade and cooking quality but not yield or storability. *Streptomyces scabies* also can cause scab on the fleshy roots of beet, carrot, parsnip, rutabaga, turnip and radish, but damage is seldom severe on these crops (see Beet, Carrot and Crucifers, scab). *Streptomyces acidiscabies*

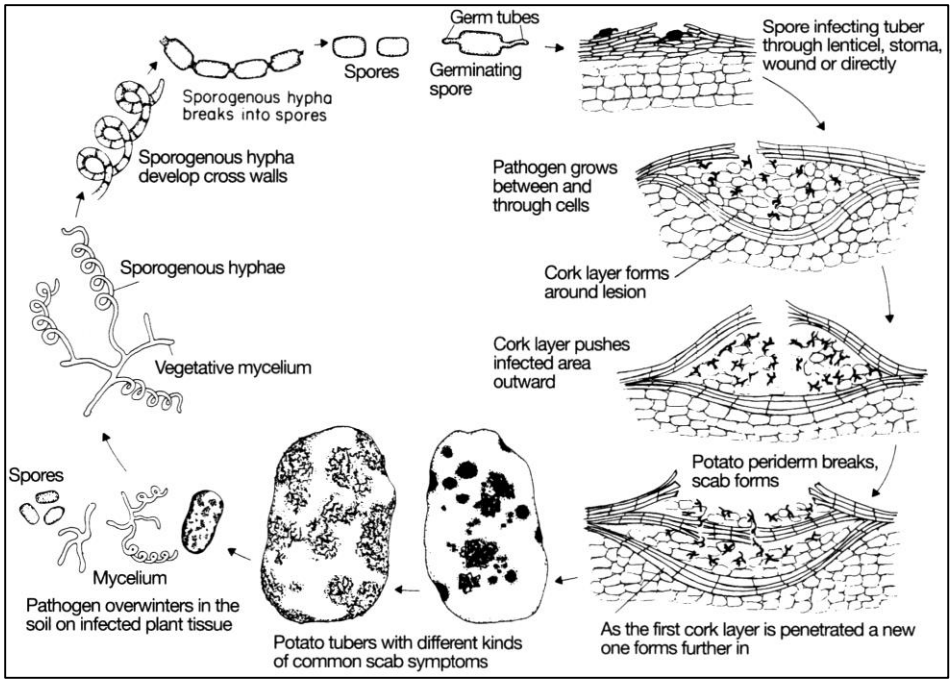
has been reported to cause scab on potato in Quebec and some parts of the United States. Russet scab has been known in the United States and Europe since 1902, and more recently in Canada. Powdery scab, which may be confused with scab caused by *Streptomyces* species, is a fungal disease; see scab, powdery, 16.16.

Symptoms The symptoms of acid scab and common scab are indistinguishable on potato. Round, irregular, brown lesions, generally less than 1 cm across, occur on the tuber surface. On potato tubers, symptoms of scab can be of three types: shallow, with lesions that are superficial and corky (76.5c); raised, with erupting lesions (76.5a); and deep-pitted, with dark brown lesions up to 6 mm deep *(16.5b).* In contrast, russet scab is characterized by corky reticulations on the tuber surface *(16.5c).* Underground stems and stolons also may be attacked. Infection can range from a few spots to total coverage of the tuber surface. The severity and type of scab depends on such factors as the strain of *Streptomyces* present in the soil, the potato cultivar, the soil organic matter content, crop rotation practices, weather conditions, and moisture availability. Scabby, warty lesions associated with powdery scab (76.76) are smaller and rounder than those of common scab and acid scab, and the lesions are filled with dark brown, powdery masses of spore balls. See Scab (powdery), 16.16.

Causal agent *Streptomyces* species are actinomycètes that can survive in the soil either in a vegetative, mycelioid form or as spores. These organisms are classified with the bacteria because nuclear fusion does not occur and cell wall biochemical characteristics more closely resemble those of bacteria than fungi. *Streptomyces scabies* resembles a fungus in its mycelioid morphology, but differs in its very thin mycelial filaments, which are about 1 µm in diameter. The mycelium has few or no septa. The spores or conidia may be cylindrical or barrel shaped, averaging 0.5 by 0.9 to 1.0 µm. They are produced on branched hyphae or conidiophores that develop successive septa from their tip toward the base. As the septa constrict, the conidia are pinched off and eventually separate from the hyphae. The conidia may develop in spiral chains and germinate by means of one or two germ tubes that develop the mycelioid form. The morphology of isolates obtained from potato, red beet or carrot and root crucifers may differ markedly.

Streptomyces scabies is a weak pathogen and its cells may exist in low numbers or disappear during symptom development, which limits its successful recovery from affected tissues. This organism is more easily isolated from young lesions than from old ones, which may become overgrown by secondary organisms. In culture, *S. scabies* produces colorless vegetative filaments and pale, mouse-gray, aerial mycelium, often with melanin pigmentation of the medium surrounding the colony. Several selected media have been developed for isolation of this pathogen. Recovery from potato is usually easier than from radish and rutabaga. Not all isolates obtained from potato will infect other vegetable hosts. The nature of this specificity is not clearly understood. There is no reliable way to differentiate between pathogenic and saprophytic strains.

The primary characteristics of S. *acidiscabies* are the production of white spores borne in flexous chains of 20 or more, the inability to synthesize melanoid pigments, and the ability to utilize all ISP (International Streptomyces Project) sugars except raffinose. Its spores are smooth, cylindrical and 0.4 to 0.5 by 0.6 to 1.1 µm. It has a growth-medium-dependent spore mass color ranging from white to orange-red, with a pH-sensitive diffusable pigment that is red above pH 8.3 and golden yellow below pH 8.3. This species also is acid tolerant and will grow on agar media at pH 4.0 versus 5.0 for *S. scabies. Streptomyces acidiscabies* survives in the soil either in a vegetative, mycelioid form or as spores.



16.5T1 Common scab; disease cycle of *Streptomyces scabies* on potato. Reprinted by permission from G.H. Agrios, *Plant Pathology*. © 1988 Academic Press.

Actinomycetes causing russet scab are characterized by a bright yellow mycelium on yeast-malt extract agar, which turns brown after about two weeks of growth. The aerial mycelium forms flexuous spore chains, which appear as a gray mass on colonies in culture. Spores are cylindrical, smooth, and 0.5 to 0.6 pm in diameter by 0.7 to 0.9 pm in length. Canadian isolates of *S. aureofaciens* do not produce melanin but degrade xanthine and xylan. Most strains utilize D-fructose, D-glucose, D-mannitol, raffinose, sucrose and D-xylose. *Streptomyces aureofaciens* differs from *S. scabies* in that it produces pigmented mycelium and flexuous spore chains but no melanin. It differs from *S. acidiscabies* in mass spore color and in its inability to grow at pH 4.5.

Disease cycle *Streptomyces* species can persist indefinitely as saprophytes on decaying plant residue in the soil, and possibly on the roots of living plants and in animal manure (*16.5T1*). These pathogens can be spread by rain and windblown soil and by infected tubers. In potato, *Streptomyces* infects through lenticels, usually during the first five weeks of tuber development. If tubers are dry during the period, bacteria antagonistic to *Streptomyces* that are normally present in the lenticels disappear, allowing scab organisms to infect more easily. Scab lesions do not continue to develop in storage.

In other vegetable hosts, infection occurs through immature lenticels of young tissues and wounds caused by insects. After penetration, the scab pathogen colonizes a few layers of cells, which die, and it survives saprophytically on the necrotic tissue. Lesions may be invaded by secondary organisms, which can decay the host tissues.

Scab is generally more severe in light, sandy or gravelly soils that dry quickly. The disease develops most rapidly at soil temperatures of 20 to 22°C and may develop slowly at 11 to 13 and 30°C. Scab caused by *S. scabies* and *S. aureofaciens* is not a problem in acidic soils, but it increases in severity as soil pH increases from 5.2 to 8.0. *Streptomyces acidiscabies* occurs in soils with pH values as low as 4.5. It is not known whether this organism and *S. aureofaciens* can infect vegetable crops other than potato under field conditions in Canada.

Management

Cultural practices — Maintaining adequate soil moisture is an important method of controlling scab. This disease is most likely to occur in soil with moisture potentials of -0.4 to -0.6 bars or drier; it is less common under wetter conditions. Growers should maintain adequate soil moisture during and after tuber set, which is about 4 to 6 weeks after planting. Reduced oxygen concentrations in moist soils are thought to inhibit scab organisms. Moist soils also encourage the growth of antagonistic microorganisms.

Overliming may result in a high soil pH and should be avoided. Soil acidification with sulfur and acid-forming fertilizers may be effective in reducing pH and the incidence of common scab, but tends to be impractical in highly buffered organic soils and may increase the severity of acid scab. Manure from animals fed on scab-infected tubers and roots should not be used on land to be sown to susceptible crops. Well-rotted organic matter helps to retain moisture in the soil and reduces the onset of the dry conditions that favor scab. Organic matter should be allowed to decompose thoroughly before the land is cropped with potato or other susceptible vegetables. Potato growers are advised to plant scab-free tubers and to avoid short rotations between potato and other susceptible crops, including sugar beet, carrot and crucifers.

Resistant cultivars — Norking, Superior, Cherokee and Huron have good resistance to common scab. Chieftain, Russet Burbank, Monona, Norchip, Norgold Russet, Norland, Viking, Avon, Jemseg, Sable, Mirton Pearl and Sebago are intermediate in resistance.

Chemical control — Seed treatment fungicides may provide some control of tuber-borne scab.

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(Original by RJ. Howard, I.R. Evans and P.D. Hildebrand)

FUNGAL DISEASES

▶ 16.6 Black dot *Figs. 16.6a,b*

Colletotrichum coccodes (Wahr.) S J. Hughes
(syn. *Colletotrichum atramentarium* (Berk. & Broome) Taubenhaus)

Black dot causes tubers *(16.6b)*, stolons, roots and stems to rot. The pathogen produces dot-like, black microsclerotia *(16.6a)* (see Tomato, anthracnose, 18.6). Foliar symptoms may be confused with those of fusarium and verticillium wilts and rhizoctonia canker. Disease-free seed, crop rotation and optimal soil fertility are the only known control procedures.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.7 Dry rot *Figs. 16.7a,b*

Fusarium avenaceum (Fr.:Fr.) Sacc.
Fusarium sambucinum Fuckel
(syn. *Fusarium sulphureum* Schlectend.)
Fusarium solani var. *coeruleum* (Lib.:Sacc.) C. Booth

Dry rot is a common disease of stored potatoes and may cause heavy losses. Under humid conditions, secondary soft rot organisms may follow dry rot and result in even greater damage. Dry rot is often a problem on lower classes of seed potatoes. This disease occurs in all potato-growing areas. The fungi that cause dry rot can also attack a variety of other crops including cereals, grasses, fruits, ornamentals and some vegetables.

Symptoms Dry rot symptoms usually first appear around wounds about one month after tubers are put into storage. Diseased tissue may appear light brown to black and be conspicuously dry. Large, sunken, concentric rings, which collapse under light pressure, may form on any part of the tuber *(16.7a).* Fully rotted tubers become shrivelled and mummified. Cavities underneath the rotted area *(16.7b)* are usually lined with the white, pink-tinted or bluish mycelial growth of the *Fusarium* fungus. Tubers also may be soft and wet if soft rot is present as well.

Causal agent Dry rot fusaria can be readily isolated and will grow rapidly on acidified potato-dextrose agar. *Fusarium solani* produces a dense, white mycelial mat that may develop a blue, blue-green or purple pigmentation with age. It forms stout, thick-walled, cylindrical macroconi- dia, oval to kidney-shaped microconidia, and chlamydospores in culture. *Fusarium sambucinum* grows with or without a dense aerial mycelium on potato-dextrose agar. When aerial mycelium is present, it may be white, tan, pink or reddish-brown. Macroconidia are short, stout, thick-walled and strongly curved. Microconidia are generally absent in culture, while chlamydospores are formed abundantly and quickly. *Fusarium avenaceum* forms a dense, aerial mycelium that may vary in color from tan to reddish- brown. Macroconidia are very long, slender and thin-walled with an elongate apical cell and a foot-shaped or notched basal cell. Microconidia are rare and chlamydospores are absent in this species.

Disease cycle *Fusarium* spp. can survive for many years in potato fields, but most infections originate from infested seed tubers. Diseased seed pieces decay and the fungus may infect young potato plants or end up in the soil particles and lumps that are harvested with the crop, thereby contaminating harvested tubers and the equipment used in their handling or storage. New infections can occur when tubers are wounded during harvesting, grading or seedcutting. Seed pieces infected during cutting turn brown under moist soil conditions. Soft rot bacteria can invade these rotted areas as secondary pathogens. *Fusarium* spp. alone or in combination with soft rot bacteria may damage or completely destroy potato seed pieces. This can result in stunted or missing plants and reduced yields. Dry rot fusaria cannot penetrate intact tuber skin, lenticels or suberized seed pieces. Tubers with well-developed skins and those that are harvested without wounding are resistant to dry rot. Immature tubers that are skinned or bruised are highly susceptible to the disease. Damp storage conditions favor dry rot.

Management

Cultural practices — Potatoes should be harvested during dry, cool weather and care should be taken to avoid bruising and wounding. Freshly dug tubers should be stored for 7 to 10 days at 12°C to favor wound healing, then the temperature should be lowered to 2 to 5°C (10°C for processing tubers). Relative humidity should be maintained at 90% with adequate air circulation. Seed tubers from cold storage should be warmed to 15°C for a few days before cutting. Planting cut seed immediately into warm,

moist soil will promote growth and wound healing. Alternatively, cut seed can be stored under well-ventilated conditions (15°C at 95% relative humidity) for five to seven days to hasten suberization before planting.

Resistant cultivars — Irish Cobbler and Kennebec are moderately resistant to dry rot.

Chemical control — Registered fungicides to prevent dry rot are available for treating cut seed and tubers going into storage.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.8 Early blight *Figs. 16.8a-c*

Alternaria solani Sorauer

Early blight occurs wherever potato is grown. Crop losses can be heavy if severe foliar infection occurs before or soon after flowering. This disease can also infect tomato, pepper, eggplant and many solanaceous weeds.

Symptoms Early blight first appears as small, brown, pinhead-like dots on older leaves. These lesions are circular, 3 to 10 mm across and consist of concentric rings of dead tissue that give them a target board appearance *(16.8a).* Lesions become angular in shape when expansion is limited by leaf veins. Diffuse, yellow margins often border the lesions. Heavy infection under prolonged wet or heavy dew conditions can cause loss of leaf area extensive enough to reduce yields. Infected leaves usually do not fall off *(16.8b).* As the disease spreads, lesions appear on the upper leaves and on the stems. Early blight is more prevalent on aging vines or those under stress from nitrogen deficiency and other diseases.

Tuber symptoms can appear during storage. Infections are visible as dark, sunken, generally circular areas surrounded by raised borders that may increase in size during storage *(16.8c).* Where they penetrate into the tuber flesh, the lesions are brown, leathery and remain superficial.

Causal agent *Alternaria solani* forms grayish-brown to black mycelium. Conidia are brown, beaked, 150 to 300 by 15 to 19 µm, with up to 11 transverse septa. The fungus grows well in culture but sporulates sparingly.

Disease cycle The fungus persists in infested crop residues, soil, infected tubers and on other solanaceous plants. Spores can contact leaves touching the soil and may be carried to leaf surfaces by wind or splashing water. These spores germinate in the presence of water and penetrate directly through leaf surfaces. Lesions are usually first seen around flowering. Young leaves and crops growing with high nitrogen regimes do not generally show symptoms because they are somewhat resistant to infection. Secondary infections occur when conidia produced on primary lesions are spread to healthy leaves. Conditions for conidial production are optimal at or near 20°C, and development stops at temperatures above 27°C. Cool nights (less than 15°C) and moist or dew-forming conditions favor spore production and germination. In many cases, early blight is principally a disease of senescing plants. Premature senescence may occur as the result of nitrogen deficiency, drought and other environmental stresses, or because of other diseases. Dead or dying potato foliage also may be colonized by other *Alternaria* spp. that are weakly parasitic or saprophytic.

Tubers can become infected as they are lifted through soil infested with *Alternaria* spores, which are concentrated at ground level. Conditions that increase the severity of foliar blight increase the quantity of spores on the soil surface. Tuber infections occur mostly through wounds. Harvesting immature tubers increases the risk of infection.

Management

Cultural practices — Growers should turn under diseased vines after harvest, maintain good soil fertility and avoid growing potato, tomato or eggplant in a crop rotation for at least two or three years. Tubers should be allowed to mature fully before harvest and should not be dug when the soil is wet. Mechanical injury during harvesting and handling should be avoided.

Chemical control — Several foliar fungicides are registered in Canada for controlling early blight. They should be applied as soon as most of the lower leaflets contain one or more spots. Several weekly applications may be necessary if weather conditions are warm and humid. For severe disease late in the season, sprays at or after vine killing may help to reduce the

production of spores that cause tuber infection. Computer models are available to help growers predict disease outbreaks and schedule fungicide applications.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.9 Fusarium wilt *Fig. 16.9*

Fusarium avenaceum (Fr.:Fr.) Sacc.
Fusarium oxysporum Schlechtend.:Fr.
Fusarium solani (Mart.) Sacc.
Fusarium solani f. sp. *eumartii* (Carpenter) W.C. Snyder & H.N. Hans.

Several species of *Fusarium* cause wilt disease in potatoes. Yield losses are most pronounced during hot, dry seasons. Potato is the only known host for *F. solani* f. sp. *eumartii***;** the other species can attack a wide variety of plants.

Symptoms Symptoms caused by the four wilt-causing *Fusarium* species are similar. Tubers exhibit surface blemishes and decay, including browning and decay at the stem end and vascular discoloration. Vascular browning may not be visible during grading, but it can substantially reduce market quality. Infection generally results in wilting and premature death of leaves and stems, often one stem at a time *(16.9).* Vascular discoloration and cortical rot usually are present in lower stems and roots. Additional symptoms may include chlorosis, yellowing or bronzing of the foliage, rosetting and purpling of aerial shoots, and formation of tubers in leaf axils.

Fusarium wilt can be confused with verticillium wilt, which causes similar foliar symptoms. However, symptoms of internal stem infections are generally extensive with fusarium wilt, while those of verticillium wilt are restricted to the vascular system.

Causal agent Differentiating the various fusarium wilt fungi may not be easy, even when the causal organisms are grown in pure culture. (For descriptions of *Fusarium solani* and *F. avenaceum,* see dry rot, 16.7; *F. oxysporum* is described under Celery, fusarium yellows, 7.5). *Fusarium solani* f. sp. *eumartii* produces sparse mycelium, light brown sporodochia, three- to four-septate macroconidia that are nearly straight in the lower half and slightly curved in the upper half, and occasional microconidia.

Wilt-causing *Fusarium* species are best isolated from infected roots and lower stems. *Fusarium solani* f. sp. *eumartii* and *F. oxysporum* are rarely isolated from discolored tuber tissue.

Disease cycle Fusarium wilt pathogens persist for many years in the soil and are spread by infected seed pieces. Standard three- or four-year crop rotations may not be effective in reducing the level of soil-borne inoculum in heavily infested fields. Wilts are most commonly observed when hot, dry growing conditions place potato plants under stress. Root infection can take place at temperatures below 20°C, but wilt fungi are most active at higher soil temperatures. Continual growing of potato on infested land leads to a build-up of wilt pathogens and may result in the land becoming unsuitable for potato production.

Management

Cultural practices — Growers should use seed potatoes free of wilt disease and grow the crop on land that has been properly rotated with cereals, grasses or forages. It is advisable not to contaminate clean fields through the transfer of infested soil, diseased tubers or infested plant residues on machinery. Destruction of infected potato vines by tillage encourages rapid decomposition of residues and lessens the carry-over of *Fusarium* spp.

Chemical control — Fungicidal seed treatments may help to reduce disease levels on infested tubers and protect seed pieces from soil-borne infection, especially if freshly cut seed is to be planted.

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(Original by I.R. Evans and R.J Howard)

▶ 16.10 Gray mold *Fig. 16.10*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)

(syn *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold generally attacks plants weakened by other diseases or environmental stresses. The pathogen requires moist conditions in order to infect. It can attack many species of vegetables (see Lettuce, gray mold, 11.10). Airborne spores carried by wind and rain hasten the decay of aging potato floral parts and leaves, particularly late in the growing season. The pathogen may also cause tuber rot.

Gray mold is sometimes mistaken for late blight, but the gray-black to brown botrytis lesions on leaves *(16.10)* and occasionally on stems will produce a characteristic grayish growth of mycelium and spores during wet weather. The fungus overwinters in crop residue.

Fungicide sprays applied for early or late blight control often hold gray mold in check as well. Tuber rot can be reduced by allowing the tubers to mature before harvesting, eliminating excess soil and stems from the potatoes during digging, and storing the crop at low temperatures.

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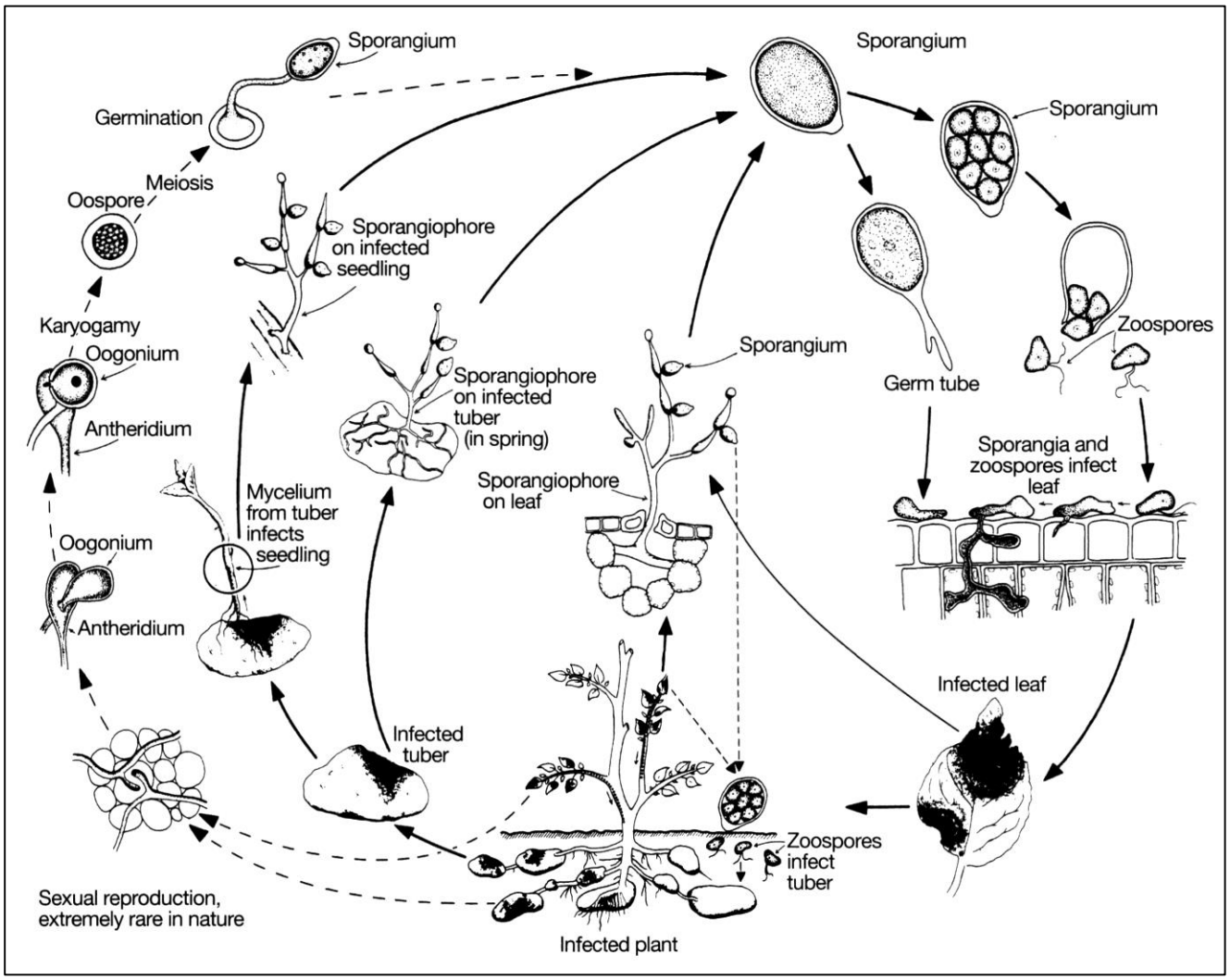
(Original by I.R. Evans and R.J. Howard)

▶ 16.11 Late blight *Figs. 16.11a-d; 16.11T1*

Phytophthora infestans (Mont.) de Bary

Before the general use of foliar fungicides, late blight was the most destructive fungal disease of potatoes. In the 19th and 20th centuries, dramatic crop losses caused by this disease resulted in major famines in Ireland and Germany. Late blight is most destructive in the Maritime provinces, less so in central Canada and British Columbia, and is usually negligible on the Prairies and in Newfoundland. Late blight can also affect tomato, pepper, eggplant and various solanaceous weeds.



16.11T1 Late blight; disease cycle of *Phytophthora infestans* on potato and tomato. Reprinted by permission from G.H. Agrios, *Plant Pathology*. © 1988 Academic Press

Symptoms The first symptoms of late blight usually appear on older leaves soon after flowering, following warm and wet or humid weather *(16.11a,b).* Dark green, water-soaked areas at the leaf tips spread inward and become dark brown and brittle in one or two days. On the underside of infected leaves, lesion edges may exhibit a fluffy white fungal growth that is visible on dewy mornings and during periods of high humidity. This fluffy mildew produces sporangia that are spread by rain and wind to other plants. Under suitably wet or humid conditions, the disease can spread rapidly within the crop, resulting in defoliation, plant death (*16.11c*) and yield loss.

Late blight lesions can resemble those of early blight in the early stages of development. However, late blight will obliterate the pattern of veination on leaves, whereas early blight does not.

Tubers at or near the soil surface can be infected. Lesions on the surface of tubers are irregular, sunken and usually appear in and around the eyes. Affected tissue is granular and reddish in appearance and it may penetrate up to 2 cm into the tuber *(16.11d).* Storage of diseased tubers can result in infection of other tubers and cause extensive crop loss.

Causal agent *Phytophthora infestans* typically reproduces asexually by sporangia borne on branched sporangiophores. Sporangia are lemonshaped, single-celled, hyaline, and measure 21 to 38 by 12 to 23 µm. Sexual reproduction occurs only when mating types A1 and A2 occur together. A1 has been known to occur world wide since the late 1800s, while A2 has long been found in Mexico. Since the early 1980s, A2 has appeared in European and Mediterranean countries and in some potatogrowing areas of the United States and western Canada. The thick-walled oospores that are produced probably play a role in the survival of the pathogen between growing seasons.

Disease cycle In Canada, *P. infestans (16.11T1)* can overwinter only in living potatoes. The fungus persists as mycelium in seed tubers, in cull piles or in volunteer potatoes that overwinter in the field. The fungus spreads primarily through sporangia, which

can travel considerable distances. They germinate at 2 to 24°C on potato tissue either directly by formation of infection hyphae, or indirectly by zoospores that form in each sporangium. The optimal temperature for indirect germination via zoospores is 21°C, whereas direct germination of sporangia occurs best at 24°C. Zoospores, released by the rupture of the sporangial wall, swim in thin films of water. Zoospores encyst on solid surfaces, such as leaves, and produce germ tubes that can penetrate and infect potato tissue. Once penetration has occurred, infection and subsequent disease development are most rapid at 21°C. The fungus exists as 20 or more races that can attack individual potato cultivars with differing degrees of resistance.

Management

Cultural practices — Growers should destroy cull piles by composting, freezing or burying, and eliminate volunteer potato plants in nearby fields with herbicides and effective cultural practices. Sprinkler irrigation should be carefully scheduled, especially late in the season when the canopy is closed in and conditions for late blight development are favorable. Infected potato tops should be killed two weeks before harvest to reduce tuber infection and avoid serious disease problems in storage. Potatoes from infested fields should not be used for seed.

Resistant cultivars — Fundy, Kennebec, Brador, Nooksack and Sebago are resistant to some late blight races. No potato cultivars are immune to this disease.

Chemical control — Foliar fungicides provide control of late blight when applied at 7- to 10-day intervals from early July until the foliage begins to senesce. Where disease forecasting systems are available, the frequency of application can be reduced during periods when dry conditions reduce disease development. To reduce tuber infections late in the season, fungicides should be applied at or following topkilling. Most commercial fungicides prevent infection, but a few are curative in their activity. Continued use of curative fungicides can lead to resistance in the late blight pathogen.

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(Original by H.W. Platt)

▶ 16.12 Leak *Figs. 16.12a, b*

Pythium ultimum Trow
Pythium spp.

Leak occurs in all potato-growing areas. It is particularly troublesome in immature potatoes harvested under warm, moist soil conditions. *Pythium* spp. cause seed decay, damping-off and root rot in many kinds of vegetable crops.

Symptoms Leak is a disease of cut or bruised tubers. Infection is seen as extensive, light to dark brown lesions on the tuber surface, particularly at the stem end. Water may drip or run from diseased tubers and is most obvious in plastic retail packages stored at high temperatures. Diseased tuber tissue is granular, watery and cream-colored to black *(16.12a,b).* Infected seed pieces can decay in the soil before emergence.

Causal agent Leak is caused by *Pythium ultimum* or occasionally by other *Pythium* species. The fungus produces spherical sporangia that are 12 to 30 µm when produced terminally, or are barrel-shaped and 17 to 27 by 14 to 24 µm when they are intercalary. Oospores are smooth, thick- walled spheres, 14 to 20 µm, that are produced terminally on the hyphae. The pathogen is sometimes difficult to isolate from diseased potato tissue.

Disease cycle *Pythium* species can live indefinitely as saprophytes in the soil, where they may attack the underground parts of many plant species. Potatoes are infected through cut surfaces or bruises that occur during harvesting. Significant losses can occur when harvesting takes place during hot weather if tubers are handled roughly, and when crops are stored at high temperatures with poor ventilation. Leak can also be a problem on cut seed in the field.

Management

Cultural practices — It is advisable to allow the crop to mature in the field before harvest and to minimize mechanical injury to the tubers during digging, grading and storage operations. As well, growers should harvest during cool weather or during the coolest part of the day. If tubers are dug during warm weather, they should be immediately cooled before storage and air movement increased to hasten drying. Freshly harvested potatoes should not be kept in the sun for extended periods as this increases susceptibility leak. Potatoes should not be planted in low, poorly drained fields.

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(Original by I.R. Evans)

▶ 16.13 Phoma rot (button-hole rot, pocket rot) *Fig. 16.13*

Phoma exigua var. *exigua* Desmaz.

Phoma rot is a less serious problem than that caused by the closely related pathogen *Phoma exigua* var. *foveata* (Foister) Boerema, which is not present in Canada but causes gangrene in potato crops in Europe and Australia (see Foreign diseases and pests, 3.10). *Phoma exiqua* infects tubers through wounds incurred during harvest and grading operations, particularly when soils are wet and cool. Small, dark depressions appear on the tuber, and the covering skin may crack. Lesions are easily removed, leaving a cavity bordered by healthy tissue *(16.13).*

Control measures include the use of clean seed, crop rotation of three to four years, minimizing tuber wounds, applying of post-harvest fungicides, and providing proper wound-healing conditions in storage immediately after harvest.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.14 Pink rot *Fig. 16.14*

Phytophthora erythroseptica Pethybr.

Pink rot causes a soft, spongy rot in harvested tubers *(16.14).* On exposure to air, the infected flesh develops a salmon pink color. *Phytophthora erythroseptica* infects underground parts of potato plants, particularly when soil moisture levels are high. Wilt symptoms can occur on the foliage. Tubers become infected through stolons, eyes and lenticels. Infected tubers are dull brown with dark skin tones around eyes and lenticels. A dark line on the skin delineates the extent of infection. Internal rot usually begins at the stem end and is creamy or light brown. Cut tubers ooze a clear odorless liquid when squeezed. Infected tissues change to pink, brown and black shortly after exposure to air.

Disease management involves use of healthy seed tubers, planting in well-drained soils, roguing diseased plants, and removing diseased tubers before crop storage.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.15 Rhizoctonia canker (black scurf) *Figs. 16.15a-g*

Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

This disease is common wherever potato is grown. It is most readily recognized by the "black scurf" on the potato skin that resists washing off. The disease has assumed more importance in recent years because of the increased trend toward consumption of potatoes with intact skins. Most of the strains of *Rhizoctonia solani* that attack potato are specific to this crop.

Symptoms *Rhizoctonia* causes the most severe losses when soil conditions are cold and wet, and when potato crops are grown too frequently in the rotation. The main damage is a loss of tuber quality. The fungus is responsible for a considerable range of symptoms on potato *(16.15a-f),* including emergence failure, reddish-brown discoloration of roots, stem and stolon cankers, swollen stems, aerial tubers, leaf rolling, wilting and purpling, premature death of vines, and tuber malformations. A growth-regulating toxin produced by the fungus causes root necrosis, stolon pruning, leaf curling, stunting and leaf margin chlorosis *(16.15e).* Signs of the fungus include sclerotia (black scurf) on tuber surfaces *(16.15c)* and a grayish white, felt-like mycelium on stems at the soil surface. This covering is the hymenial stage of the teleomorph *(16.15g)* and it forms only under humid

conditions. Basidiospores are produced on basidia within this growth but they do not appear to play an important role in disease development on potato.

Causal agent (see Bean, rhizoctonia root rot, 15B.7) *Rhizoctonia solani* strains pathogenic to potato generally belong to the anastomosis group AG-3, while those affecting crucifers belong to anastomosis groups AG-2 or AG-4. The mycelium is usually dark brown and coarse, 8 to 10 pm in diameter, with characteristic right-angled branching. Dark sclerotia of various sizes and shapes are produced in agar culture.

Disease cycle The fungus persists between seasons as sclerotia (black scurf) on tubers and in the soil or as mycelium on crop residues. Sclerotia germinate and the mycelium infects emerging potato sprouts, roots, stolons and tubers throughout the growing season. Sclerotial formation on young tubers is affected by tuber maturation and senescence in mother plants. *Rhizoctonia* may survive for long periods in potato fields by saprophytically colonizing plant residues other than those of potato.

Management

Cultural practices — Growers should plant seed potato tubers free from sclerotia in fields that have not grown potato or any other solanaceous crop for at least three years. Practices that favor rapid emergence, such as warming the seed, planting in warm soil, seeding shallow, hilling after emergence, and delaying irrigation until sprouts have emerged, may reduce the incidence of stem cankers and sprout damage. Potato should be rotated with cereals or grasses. In small plantings, early harvesting and hand-pulling the tops to remove stems, stolons and roots will help to reduce scurf.

Chemical control — Fungicidal seed treatments may help to control seed-borne inoculum, but they are not effective in protecting young plants in heavily infested soils. To ensure adequate coverage, fungicides should be applied to tubers that are free of soil.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.16 Scab, powdery *Fig. 16.16*

Spongospora subterranea (Wallr.) Lagerh.

This disease causes scabby, warty lesions on tuber surfaces. The lesions fill with dark brown, powdery masses of spore balls. Each spore ball contains numerous, individual, uninucleate resting spores, which germinate by releasing a zoospore. The zoospore is motile and can infect root hairs of potato plants. Tuber lesions usually remain superficial unless the soil is wet. Tuber symptoms may be confused with those of common scab *(16.5),* but powdery scab is characterized by lesions that are smaller and rounder *(16.16)* than those of common scab. Disease outbreaks are usually sporadic. *Spongospora subterranea* is a vector of potato mop-top virus, which occurs in Europe and South America. See also Bacterial diseases: scab, 16.5.

Powdery scab can be managed by planting Certified or a higher class of seed, following crop rotations of three to four years, not planting potato in infested fields, and not over-irrigating. Russet cultivars are tolerant to powdery scab.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.17 Seed-piece decay *Figs. 16.17a-c*

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*
Erwinia carotovora subsp. *atroseptica* (van Hall) Dye
Fusarium spp.
Pythium spp.

Failure of seed pieces to produce vigorous seedlings (plant misses) in potato crops may be caused by disease or environmental factors. Disease organisms associated with seed-piece decay may be fungal, bacterial or a combination of the two. Other tuber- and soil-borne diseases and pests also may play a role in the failure of plants to become established.

Symptoms Gaps, missing hills or delayed emergence *(16.17a)* are the first indications of a seed-piece decay problem. Occasional blanks may have little effect on the crop, but misses that exceed 10% of the planted seed may have economic consequences on yield and quality of the harvested crop. The extent of decay *(16.17b,c)* can be determined by digging to recover the seed piece. If the tubers are sound, then physiological causes such as chilling injury or broken sprouts may be the problem.

Causal agents *Fusarium, Pythium, Erwinia* and other genera of pathogens may be isolated from decaying tuber pieces. (For descriptions of these pathogens, refer to dry rot, leak, blackleg and bacterial soft rot in this chapter.)

Disease cycle (Refer to dry rot, 16.7; leak, 16.12; blackleg, 16.3; bacterial soft rot, 16.2.)

Management

Cultural practices — Using high quality seed that has been properly stored and handled should be the primary consideration in managing seed-piece decay. Growers should plant Certified or a better grade of seed potatoes and avoid using old tubers. The cutting of seed pieces can spread pathogens from diseased to healthy tubers, and it also exposes the seed pieces to soil-borne disease organisms. In Europe, virtually all potato planting is done with small, whole tubers to minimize the risk of mechanical spread of pathogens. Seed should be planted into warm soil immediately after it has been cut. If this is not feasible, the seed should be stored in open containers and held at 10 to 15°C for three to four days under high humidity, with good ventilation to promote suberization. If further storage is necessary, temperatures should be dropped to 5°C, then raised again before planting. Cut seed should never be stored under closed, moist conditions. Seed-cutting equipment should be disinfested frequently, using steam or disinfectants, especially between different lots of seed potato to prevent the spread of tuber-borne pathogens.

Chemical control — Chemical treatment of tuber pieces is not a substitute for the use of Certified seed and proper handling practices. Seed treatments will protect against pathogens in the soil and on the cut surface of the tuber, but not from infections already established within the tuber. Post-harvest treatment of seed tubers with fungicides, followed by dusting of seed pieces immediately after cutting, offers the best chemical protection against seed-piece decay.

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(Original by I.R. Evans)

▶ 16.18 Silver scurf *Figs. 16.18a-c*

Helminthosporium solani Durieu & Mont.
(syn. *Spondylocladium atrovirens* (C. Harz.) C. Harz.:Sacc.)

Silver scurf is a skin disease which is usually of minor importance on unwashed potatoes intended for peeling. However, with the marketing of washed potatoes in clear plastic bags, the increased consumption of potato skins, and the use of unpeeled tubers for french fries and chips, concern for this problem is increasing. Potato is the only vegetable crop affected by silver scurf.

Symptoms Symptoms can develop prior to harvest or during storage. Round, light brown or grayish, leathery spots form on the tuber *(16.18a).* These lesions enlarge to cover most of the surface *(16.18b).* Moist or wet infected tubers have a distinctive silvery sheen. Spore formation, especially at the margins of young lesions, gives the tubers a sooty appearance. Silver scurf is most noticeable on red potatoes, where it may obscure the color. Infection reduces quality and causes tubers to lose excessive amounts of moisture and shrivel in storage. Lesions penetrate a few millimetres into the flesh and are difficult to remove during commercial peeling operations *(16.18c).*

Causal agent *Helminthosporium solani* produces brownish mycelium and septate conidiophores with conidia borne in whorls that are visible to the naked eye. Spores are 7 to 11 by 24 to 85 µm, dark brown, two- to eight-septate, tapered at the apex, and possess a distinct dark scar at the base.

Disease cycle The fungus can be carried on seed pieces and on tuber residues in the soil. Clean seed can be infected when planted in infested soil. Mature tubers left in the field late in the season in warm, moist soil are also susceptible to infection. The pathogen can infect tubers through lenticels or directly through the skin. Disease levels increase in storage, and further tuber infections can occur at humidities over 93% and temperatures above 3°C.

Management

Cultural practices — Growers should plant disease-free tubers and practice crop rotation. Tubers should be harvested when mature. Storages should be ventilated with dry, warm air to remove moisture from tuber surfaces, and then tubers should be stored at temperatures as low as possible.

Chemical control — When symptoms of silver scurf are present, or where the potential for disease development in storage is high, potatoes going into storage, particularly seed potatoes, should be treated with a fungicide. Seed pieces should be treated with a recommended fungicide before planting. Resistance to benzimidazole fungicides in the silver scurf fungus has been recorded.

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(Original by I.R. Evans and J.D. Holley)

▶ 16.19 Skin spot*Fig. 16.19*

Polyscytalum pustulans (M.N. Owen & Wakef.) M.B. Ellis (syn. *Oospora pustulans* M.N. Owen & Wakef.)

Skin spot primarily damages tubers, but roots, stolons and underground stems can also exhibit symptoms. Symptoms on tubers *(16.19)* appear as small sunken depressions with raised centers that are darker than healthy tuber skin when dry. These lesions can grow together to form larger diseased areas. The fungus overwinters on potatoes and in soil. New infections are caused by the pathogen entering lenticels, wounds and tuber eyes. Planting healthy tubers and avoiding tuber damage before storage will control this disease.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.20 Verticillium wilt *Figs. 16.20a-c*

Verticillium albo-atrum Reinke & Berthier
Verticillium dahliae Kleb.

Verticillium wilt is a common disease of potato that is often confused with other wilt and early maturity diseases. This disease is often implicated with early dying, a syndrome caused by a combination of several pathogens and environmental conditions such as ozone injury and drought. The most important pathogens associated with early dying are *Verticillium* spp., *Erwinia carotovora* (see bacterial soft rot, 16.2) and the root-lesion nematode (see Nematode pests, 16.38). The distribution of early dying in Canada has not been determined. *Verticillium* pathogens have a wide host range and are capable of attacking tomato, pepper, eggplant, cucurbits, and other broadleaf crops and weeds (see Greenhouse cucumber, verticillium wilt, 22.17).

Symptoms Characteristic symptoms of verticillium wilt are the early dying of leaves and stems on plants *(16.20a)* in irregular patches in the field, particularly on well-drained sandy soils. Lower leaves are generally affected first. Typically only one stem or leaves on one side of a stem shows wilting, especially during hot, windy days or when soil conditions are dry. Areas between leaf veins turn yellow and later brown. Wilted plants may recover at night or during cool, moist weather. Wilted stems cut at ground level show a brown discoloration of the vascular tissues *(16.20b).* Tubers from infected plants may have discolored vascular rings, particularly at the stem end *(16.20c).*

Causal agent *Verticillium dahliae* produces ovate, single-celled conidia, 2.5 to 8.0 by 1.4 to 3.2 µm, at the tips of phialides. The phialides are arranged in whorls (verticils) on septate conidiophores and are often produced within the xylem vessels of the host, which accounts for the rapid systemic spread of the pathogen. It forms small, thick-walled, variably shaped, brown to black microsclerotia, 15 to 50 µm in diameter. They occur within infested plant residues and eventually are released into the soil.

Verticillium albo-atrum is similar in appearance to *V. dahliae,* except that instead of microsclerotia it develops a septate, dark, resting mycelium on potato stems and in culture. Its conidia are slightly larger, 3.5 to 10.5 by 2.0 to 4.0 µm.

Both *Verticillium* species are relatively slow-growing in culture. They can be readily cultured on potato-dextrose or V-8 agar. The mycelium is floccose, white to grayish-white, compact and occasionally sectored. The production of microsclerotia and resting mycelium in culture is variable, but often occurs as cultures age.

Disease cycle *Verticillium albo-atrum* and *V. dahliae* are soil-inhabiting fungi. Both may be present in the same field or on the same plant. *Verticillium albo-atrum* is generally more pathogenic than *V. dahliae,* but disease responses vary depending on climatic conditions, potato cultivar and pathogen. These fungi are spread by infected or contaminated seed pieces and by infested soil, farm machinery and irrigation water. They can persist and build up in the soil if potato is grown several years in succession. Other crops and weeds can harbor these pathogens without symptoms. Infection of the potato plant occurs through the root, particularly root hairs. Hyphae of the fungi grow within the xylem vessels and block the movement of water. Spores may be air-borne and the fungi can also spread to other plants by root contact. High soil temperatures (22 to 27°C) favor the growth of *V. dahliae,* while *V. albo-atrum* has a broader temperature range (16 to 27°C). Some soil-borne plant parasitic nematodes have been shown to increase the incidence and severity of verticillium wilt.

Management

Cultural practices — A high level of plant vigor should be maintained by adequate fertilization and irrigation practices. It is advisable to follow a three- or four-year crop rotation. Both fungi can survive for many years in the soil, in the absence of potato, on related solana- ceous crops or weeds. *Verticillium dahliae* has a longer survival potential than *V. albo-atrum* in soil. Cereals, oilseeds, grasses and legumes grown in rotation with potato should be kept weed-free. Highly susceptible potato cultivars should not be grown. Certified or better grades of seed potato should be used.

Resistant cultivars — No resistant cultivars are available but considerable variation exists among cultivars. Russet Burbank is somewhat less susceptible to *V. albo-atrum* than to *V. dahliae.*

Chemical control — Seed pieces can be treated with a recommended fungicide immediately before planting. Soil fumigants (nematicides, fungicides) are effective in reducing disease losses, but may not be economical.

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(Original by I.R. Evans and R.J. Howard)

▶ 16.21 Wart (canker) *Figs. 16.21a-d*

Synchytrium endobioticum (Schilberszky) Percival

Potato wart disease is prevalent in most potato-growing regions of the world and is one of the most damaging potato diseases. The causal agent is a soil-borne fungus that attacks growing points on the potato plant, such as eyes, buds and stolon tips.

The considerable economic importance of wart is due to the resting spores that can live for 40 years in soil and are spread easily by contaminated tubers and soil. Once in the soil, the fungus cannot be eradicated without fumigation. In severe infestations, no tubers develop. The presence of this pathogen in Newfoundland warrants strict quarantine legislation (see Introduced diseases and pests, 3.11).

The fungus is believed to be indigenous to Peru, where the potato originated. It spread rapidly throughout western Europe at the turn of this century and was discovered in Newfoundland in 1909 and in Maryland, Pennsylvania and West Virginia 10 years later. These infestations probably originated with the import of European table potatoes, some of which likely were used as seed. In Newfoundland, most infested soils are found in backyard gardens. Infestations in the United States were believed to have been eradicated, but recent examination of old quarantine areas in Maryland has shown that the fungus is still present.

Potato is the only host plant of economic importance. However, other *Solanum* hosts are susceptible, including tomato and henbane *(Hyoscyamus niger* L.). The pathogen is spread by infected potato tubers and infested soil. The fungus can serve as a vector for potato virus X.

Symptoms *Synchytrium endobioticum* causes galls and warty outgrowths that may be pea- to fist-sized. Aerial galls (*16.21a*) are greenish and turn brown and then black at maturity. They can decay and slough-off into the soil. On occasion, galls may form on the upper parts of the plant and even on the flower parts, but generally galls are found on stem bases, stolon tips and in tuber eyes *(16.21b-d).* Eye infections are whitish and resemble small cauliflower heads. Potato roots are not attacked by the wart fungus.

The tuber surface may be over-grown with warty tissue, as the fungus continually germinates and repenetrates the original infected area. The entire tuber may rot and disintegrate. Infected tubers that appear clean at harvest may develop warty outgrowths in storage.

Causal agent *Synchytrium endobioticum* is an obligate parasite and belongs to the Chytridiomycetes, a group of fungi generally found in fresh water habitats. Up to 20 races or pathotypes have been identified, four of which (1,2, 6/7, and 8) occur in Newfoundland.

The fungus has a complex life cycle. It does not produce hyphae but enters a host epidermal cell through an infection peg formed from an encysted zoospore. Zoospores measure 2 to 4 µm and possess a tail-like flagellum about seven times the length of the spore. After penetration, potato cells surrounding the infected cell enlarge, the fungus multiplies and the zoospores, which are produced asexually, are released. Some zoospores will penetrate to continue the cycle, others will conjugate and then penetrate. Conjugation produces a sexual entity that re-infects several cells deep, giving rise to a resting spore.

Resting spores are golden brown and spherical, have prominent ridges or flanges, and measure 35 to 80 µm. Resting spores are known to live for 40 years or more in undisturbed soil. When they germinate, the contents flow into a sac which forms a wall around itself. The resulting sporangium is expelled from the sac. The wall of the sporangium splits and releases 200 to 300 motile zoospores. Zoospores live for one to two hours in soil water before encysting. If the zoospore encysts on susceptible potato tissue, the cyst forms a penetration peg and the contents flow into the host cell to renew the cycle.

Disease cycle The disease occurs in soils of pH 3.5 to 9.0, and is favored by low summer temperatures and abundant water. Water is required for germination of the resting spores, dispersal of zoospores, and rotting and dissolution of infected plant tissue. In areas where the disease occurs, the annual precipitation is equal to or greater than 700 mm, summers are cool, with an average temperature of 18°C or less, and the winters have approximately 160 days that are 5°C or colder. The fungus is most active when susceptible tissues, such as sprouts, tuber eyes and stolon buds, are being formed.

Management

Monitoring — Soil tests are available to determine the number and viability of resting spores per unit of soil. Tests for viability of resting spores include plasmolysis, vital stains, fluorescent stains, and dark-field microscopy but none gives absolute values. The simplest method is to plant a susceptible tuber in soil suspected of harboring the fungus. However, this test also may be unreliable because of low numbers of spores or other inhibiting factors. In addition, this test only gives estimates of population viability and not of single spores. Recent bioassay work indicates that nodal cuttings may provide a more sensitive measure of population viability than tubers.

Cultural practices — Studies in Newfoundland have shown that crushed crabshell, which contains chitin, reduces the level of the fungus in the soil when used as a soil amendment. Absolute control has been recorded in greenhouses trials, and clean potato crops have also been raised using this material in the field. Plant quarantine regulations prohibit the planting of potato on land infested with the wart pathogen. In addition, the importation of potato tubers and soil from Newfoundland into other Canadian provinces, or from infected areas of other countries into Canada, is prohibited (see Introduced diseases and pests, 3.11).

Resistant cultivars — Several resistant cultivars have been developed in Newfoundland. These include Anson, Blue Mac, Brigus, Cupids, Mirton Pearl and Pink Pearl.

Chemical control — Copper sulfate and methyl bromide are known to destroy *S. endobioticum*, but their use is not practical or economical in commercial fields.

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(Original by M.C. Hampson, S. Wood and I.A. MacLatchy)

▶ 16.22 White mold *Fig. 16.22*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia sclerotiorum white mold has a very wide host range that includes many solanaceous, cruciferous and leguminous plants (see Bean, white mold, 15B.9). In potato, damage may occur to the growing vines *(16.22)*, particularly to intensively managed crops, and occasionally on tubers in storage. The presence of white mycelium and large, black, irregularly shaped sclerotia in or on infected tissue is a characteristic sign of this disease.

White mold can be controlled by rotating potato with non-host crops, such as cereals and grasses, and by planting on well-drained soils. Growers should apply balanced levels of nitrogen fertilizer and irrigate as required. If the disease appears, irrigation should be stopped to allow the plant canopy to dry to halt disease development.

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(Original by I.R. Evans and R.J. Howard)

VIRAL AND VIRAL-LIKE DISEASES

▶ 16.23 Aster yellows (haywire, purple dwarf, purple-top wilt) *Fig. 16.23*

Aster yellows mycoplasma-like organism

Aster yellows is an uncommon but potentially destructive disease of potato. Many vegetables (see Lettuce, aster yellows, 11.15), ornamentals, field crops and weeds are affected by aster yellows.

Symptoms Upper leaflets roll and develop purple or yellow pigmentation *(16.23).* Affected stems usually die prematurely, preventing some tubers from reaching maturity. Aerial tubers occasionally form on affected plants, while below-ground tubers may feel spongy. The causal agent rarely survives in stored tubers, and the usual evidence of aster yellows infection in the second year is a failure of seed to produce plants of normal size and vigor.

Causal agent The aster yellows mycoplasma-like organism is characterized by pleomorphic particles bound by a unit membrane and measuring 50 to 1000 nm in diameter. Absolute proof of the host-parasite relationship has not been established.

Disease cycle The aster yellows pathogen does not normally overwinter in potato tubers and it can be transmitted only by leafhoppers. The extent of its spread to potato depends on the access of the aster leafhopper vector to other infected hosts. Weather conditions that favor the increase and mobility of the vector promote the spread of the disease. The pathogen propagates within the leafhopper vector which remains infectious for life.

Management

Cultural practices — The use of carefully selected, Certified or a higher class of seed grown in areas where aster yellows is rarely found is important. Growers located in areas where aster yellows is widespread should use insecticides to control leafhoppers that migrate into potato fields, particularly along field margins.

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(Original by N.S. Wright)

▶ 16.24 Calico *Fig. 16.24*

Alfalfa mosaic virus

Calico is a minor disease of potato. It occurs in most potato-growing areas of Canada. The virus has a wide host range, including Leguminosae, Solanaceae and 10 other plant families.

Symptoms Alfalfa mosaic virus causes pale to bright yellow mottling or blotching of potato leaflets *(16.24)* and usually some necrosis in leaflets, stems and tubers.

Causal agent Alfalfa mosaic virus has bacilliform particles of different lengths, the largest being about 60 nm. The three largest RNA species comprise the genome; the fourth is a sub-genomic messenger for the coat protein.

Disease cycle Alfalfa mosaic virus overwinters in potato tubers and perennial hosts. It is transmitted by seed in some alfalfa cultivars and in chili pepper. While rub transmission is possible, most natural spread probably occurs through the feeding of several aphid species, including the green peach aphid. Alfalfa mosaic virus is transmitted in a stylet- borne or non-persistent manner.

Management

Cultural practices — Producers should avoid planting seed potatoes adjacent to alfalfa and clover crops or in fields where volunteer plants of these two crops are present. Only Certified or a higher class of seed should be planted and plants visibly affected with calico symptoms in seed fields should be rogued.

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(Original by R. Stace-Smith, N.S. Wright and P.J. Ellis)

▶ 16.25 Corky ring spot (spraing, stem mottle) *Figs. 16.25a,b*

Tobacco rattle virus

Corky ring spot is rare in Canada. It has been found only in a few isolated home gardens in Alberta and Saskatchewan where it is of no economic significance. The pathogen has a host range that includes plant species in over 50 families.

Symptoms Stem mottle, a symptom rarely seen in North America, is caused by the feeding of numerous viruliferous nematodes on emerging stems. Secondary symptoms vary from a slight mottle to severe distortion. Ring spot symptoms on tubers also vary considerably from small necrotic flecks to prominent concentric rings *(16.25a,b).*

Causal agent Tobacco rattle virus is a variable, rod-shaped virus, with particles 17 to 25 nm in diameter by 180 to 210 nm long. It is usually accompanied by short, non-infectious rods (about 45 to 115 nm), which are involved in synthesis of coat protein.

Disease cycle Tobacco rattle virus is spread by nematodes of the genera *Paratrichodorus* and *Trichodorus* (see stubby-root nematodes, 16.39). The virus can persist in these vectors indefinitely. Primary tuber infection follows nematodes feeding directly on tubers. Transmission through seed tubers is infrequent.

Management

Cultural practices — Only Certified or a higher class of seed should be used if this virus occurs in the area.

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(Original by R. Stace-Smith, N.S. Wright and P.J. Ellis)

▶ 16.26 Leafroll *Figs. 16.26a,b*

Potato leafroll virus

Potato leafroll occurs worldwide and causes significant losses in yield and quality. It is widespread in Canada and is generally considered to be the most serious virus disease of potato. Processing potatoes from fields severely affected by leafroll usually are rejected. Several crop and weed species, mostly in the potato family (Solanaceae), are known hosts for this virus.

Symptoms Aphids may transmit potato leafroll virus to potato plants at any time during the growing season. Plants infected late in the growing season may not show any symptoms, while symptoms that develop following aphid feeding early in the season differ from those in plants grown from infected tubers.

Primary (current season) leafroll — If infection occurs early, the upper leaves roll, turn pale green (sometimes pink-tinged), and are stiffer than normal. Primary infection in some cultivars, for instance Russet Burbank, Norgold Russet, Green Mountain and Irish Cobbler, causes internal net necrosis in the tubers either before or during storage (*16.26b).* Similar symptoms can be caused by aster yellows, verticillium and fusarium wilts, top killing and heat stress.

Secondary (chronic) leafroll — Plants that develop from tubers infected with potato leafroll virus develop characteristic symptoms *(16.26a).* The lower leaflets are rolled, stiff, dry and leathery, and may rattle when shaken. The older leaves of some cultivars turn pink or yellow and become severely necrotic; the plants often appear stunted and rigid. Symptoms are less pronounced on the upper leaves, which may be pale or chlorotic. Symptom severity is determined by the virus isolate, the potato cultivar and the growing conditions.

Causal agent Potato leafroll virus is classified as a member of the luteovirus group. The virus particles are isometric and about 24 nm in diameter.

Disease cycle Potato leafroll virus overwinters in infected tubers. In Canada, no other hosts have been implicated as important reservoirs. Weeds, such as shepherd's-purse *(Capsella bursa-pastoris* (L.) Medic.) and black nightshade *(Solanum nigrum* L.), can be infected by potato leafroll virus, but they are not significant reservoirs. Infected seedstocks, sprouted cull potatoes and volunteer potato plants are the most important sources of the pathogen. In some areas, leafroll-infected potato plants in backyard gardens are significant reservoirs of the virus. Several aphid species transmit the virus, the green peach aphid being the most efficient and important vector. Aphids acquire the virus while feeding on infected plants and remain viruliferous for life. Potato leafroll virus is spread over long distances by winged aphids, and to adjacent plants by wingless aphids. Plants become more resistant to infection as they mature. Some tubers from plants that are infected late in the season may escape infection. Potato leafroll virus is not transmitted by rubbing healthy plants with sap from an infected plant.

Management (See individual aphid species for their control.)

Cultural practices — Seed certification is the foundation of any management program for potato virus diseases. Growers should select seedstocks from those inspected and found to be free of leafroll and other virus diseases. Recommendations to control aphid vectors should be followed. Volunteer potato plants that appear in or near potato fields should be destroyed.

Potatoes that are free of potato leafroll virus traditionally are grown by specialized seed potato growers in areas relatively free of aphids. Seed plots are usually harvested early to avoid infection late in the season, when aphid numbers are highest. Often, the potato vines are cut or chemically killed to reduce chances of potato leafroll virus infection. In seed-growing operations, clonal selection and roguing of infected plants are practiced to minimize or eliminate spread of the virus.

Resistant cultivars — The traditional approach to plant breeding has not successfully controlled potato leafroll. The difficulty of combining desirable agronomic characteristics with resistance to potato leafroll virus makes it unlikely that present cultivars will be replaced by leafroll-resistant cultivars in the near future. However, genotypes with low concentrations of potato leafroll virions may be useful for delaying virus acquisition by vectors and in slowing plant- to-plant spread.

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(Original by P.J. Ellis and I.R. Evans)

▶ 16.27 Mosaic diseases *Figs. 16.27a,b; 3.11a-c*

Potato virus A
Potato virus M
Potato virus S
Potato virus X
Potato virus Y

Mosaics are the most common viral diseases of potato in Canada. Their effects on tuber yield and quality can range from no discernible damage to severe damage. The impact of these viruses is greater in seed than in table-stock potato production.

Potato virus A

Potato virus A is relatively rare, especially in pedigreed seed. Its host range is limited to solanaceous plants.

Symptoms Potato virus A causes varying degrees of leaf mottling or mosaic, depending on virus strain, potato cultivar and weather conditions. Symptoms are intensified by cloudy, cool weather. Symptoms caused by a combined infection of potato virus A and potato virus X are more severe than those resulting from infection by either pathogen alone. There are no tuber symptoms.

Causal agent Potato virus A is a potyvirus with filamentous particles, 750 by 15 nm. Potato virus A infections can be identified by specific monoclonal antibodies utilized in enzyme-linked immunosorbent assay (ELISA) procedures involving extracts from infected leaf tissue.

Disease cycle Potato virus A overwinters in infected tubers. It is transmitted in a non-persistent manner by aphids, mostly by the potato aphid and the green peach aphid. The pathogen is not transmitted in sap because of virus instability. For this reason, potato virus A is not likely to be spread by seed tuber cutting or by foliar contact. Aphids acquire the virus on their mouthparts (stylets) during feeding. Virus retention on the stylets usually is limited to the time between its acquisition and the first feeding on a mosaic-free plant.

Management (See aphids, 16.40-16.43, for control of individual species.)

Cultural practices — Growers should plant Certified or a higher class of seed.

Selected references

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De Bokx, J.A. 1981. Potato virus A. Pages 71-72 *in* W.J. Hooker, ed., *Compendium of Potato Diseases.* APS Press, St. Paul, Minnesota. 125 pp.

(Original by N.S. Wright, R. Stace-Smith and P.J. Ellis)

Potato virus M

Potato virus M, although widespread and important in eastern Europe, is very rare in North America. In addition to potato, some other solanaceous plants, including tomato, and a few species in the families Chenopodiaceae and Leguminosae are suceptible.

Symptoms Foliar symptoms are a slight to severe mottle, mosaic, crinkling or rolling of leaves, the stunting of shoots, and leaflet deformation. Severity of symptoms is influenced by virus strain, potato cultivar and weather. Temperatures of 24°C or higher tend to mask symptoms.

Causal agent Potato virus M is a carlavirus with filamentous rods, 650 by 12 nm. The pathogen can be transmitted by rubbing leaves of healthy plants with sap from infected plants. The virus can readily be detected by serological methods.

Disease cycle Potato virus M overwinters in infected tubers. It is transmitted in a non-persistent manner by several aphid species, including the potato aphid and the green peach aphid. As with most viruses, some strains are more readily transmitted by aphids than others.

Management (See individual aphid species, 16.40-16.43, for their control.)

Cultural practices — Growers should plant Certified or a higher class of seed.

Selected references

Bagnall, R.H., R.H. Larson and J.C. Walker. 1956. Potato viruses M, S and X in relation to interveinal mosaic of the Irish Cobbler variety. *Wisconsin Agric. Exp. Stn. Res. Bull.* 198. 45 pp.

Hiruki, C. 1981. Potato virus M. Pages 74-75 *in* W.J. Hooker, ed., *Compendium of Potato Diseases.* APS Press, St. Paul, Minnesota. 125 pp.

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(Original by R. Stace-Smith, N.S. Wright and P.J. Ellis)

Potato virus S

Potato virus S is the most widespread potato virus and occurs even in pedigreed seed in many areas. It appears to have been eradicated from the Pemberton Seed Potato Control Area in British Columbia as a result of a control program that began in 1967. Yield losses of up to 20% caused by potato virus S have been reported.

Symptoms Potato virus S infection is virtually symptomless in most potato cultivars. However, some strains cause a deepening of veins, and mottling, bronzing or rugosity of the foliage. There are no tuber symptoms.

Causal agent Potato virus S is a carlavirus with slightly curved, filamentous particles, 650 by 12 nm. The pathogen is strongly antigenic, so infections may be detected by most serological methods, especially in mid-season when virus concentration in plants is highest.

Disease cycle Potato virus S overwinters in infected tubers. It is readily transmitted in sap and extensive spread may occur if seed cutting knives are contaminated, if sprout contact occurs, or if the foliage of growing plants rubs together. Some strains are transmitted by the green peach aphid in a non-persistent manner.

Management (See individual aphid species for their control.)

Cultural practices — Growers should plant Certified or a higher class of seed. Cutting knives should be thoroughly disinfested between seed lots.

Selected references

Bagnall, R.H. 1981. Potato virus S. Pages 75-77 *in* W.J. Hooker, ed., *Compendium of Potato Diseases.* APS Press, St. Paul, Minnesota. 125 pp.

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Wright, N.S. 1988. Assembly, quality control and use of a potato cultivar collection rendered virus-free by heat therapy and tissue culture. *Am. Potato J.* 65:181-198.

(Original by N.S. Wright, R. Stace-Smith and P.J. Ellis)

Potato virus X *Fig. 16.27a*

Since 1967, when clones of virus-free potato cultivars first became available in Canada, potato virus X has declined from high to low levels in commercial seed stocks. The yield reduction caused by this pathogen in many cultivars can be 15% or more.

Symptoms Depending on strain of virus, cultivar and weather conditions, potato virus X can cause a very mild to severe mottle of the foliage *(16.27a).* Symptoms are most visible under cool, cloudy growing conditions. There are no tuber symptoms.

Causal agent Potato virus X is the type member of the potexvirus group. It has filamentous particles that measure 515 by 13 nm. The pathogen is strongly antigenic, and infections may be detected by serological methods.

Disease cycle Potato virus X overwinters in infected tubers. It is readily spread from infected to healthy potato plants by cutting knives, sprout contact before planting, and by contact of foliage or roots during the growing season. Biting or chewing insects may spread the virus but not piercing and sucking insects, such as aphids.

Management

Cultural practices — Growers should plant pedigreed seed. Cutting knives and machinery should be thoroughly disinfested between seed lots.

Selected references

Koenig, R., and D.-E. Leseman. 1989. Potato virus X. AAB Descriptions of Plant Viruses, No. 354. Assoc. Appl. Biol., Inst. Hort. Res., Wellesbourne, Warwick, U.K. 5 pp.

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(Original by N.S. Wright, R. Stace-Smith and P.J. Ellis)

Potato virus Y *Figs. 16.27b; 3.11a-c*

Potato virus Y occurs commonly on solanaceous plants (see also Tomato, 18.20) worldwide. In North America, most potato virus Y infections involve common strains of the virus (designated PVY°), but occasional infections by necrotic strains (designated PVY^N) have been found (see Introduced diseases and pests, 3.11). The necrotic strains cause weak mosaic symptoms, if any, on potato foliage but induce a bronzing and necrotic reaction on tobacco *(3.11a,b).* These strains occur in Europe and their possible importation to Canada is a concern. In 1990, a strain of PVY^N that causes systemic veinal necrosis in tobacco was discovered in seed potatoes in eastern Canada. Potato virus Y° is one of the most damaging potato viruses in terms of yield loss. In combination with potato virus X, it causes an even more destructive disease known as rugose mosaic *(16.27b).*

Symptoms Symptoms in potato vary with virus strain and potato cultivar and range from a mild mottle to severe foliar necrosis. Some European strains of PVY^N can cause brown rings on the skin of tubers.

Causal agent Potato virus Y is the type member of the potyvirus group; it has long filamentous rods, 730 by 11 nm. The pathogen is antigenic, and serological detection, especially with enzyme-linked immunosorbent assay (ELISA) procedures, is quite effective.

Disease cycle Potato virus Y overwinters in infected tubers and is readily sap-transmissible. Ground cherry *(Physalis heterophylla* L.) *(3.11c)* has been found to be infected with a strain of PVY^N in Ontario. The spread of potato virus Y depends mainly on the presence of winged aphids. Many species of aphids are vectors, but the green peach aphid is probably the most efficient. Potato virus Y is transmitted in a non-persistent manner.

Management

Cultural practices — Growers should plant only carefully selected seed of Certified or a higher class. Aphids should be controlled (see buckthorn aphid, 16.40, and green peach aphid, 16.41).

Selected references

Boiteau, G., R.P. Singh, R.H. Parry and Y. Pelletier. 1988. The spread of PVY in New Brunswick potato fields: timing and vectors. *Am. Potato J.* 65:639-649.

De Bokx, J.A. 1981. Potato virus Y. CMI/AAB Descriptions of Plant Viruses, No. 242. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

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(Original by N.S. Wright, R. Stace-Smith and P.J. Ellis)

▶ 16.28 Spindle tuber *Figs. 16.28a,b*

Potato spindle tuber viroid

Before 1980, spindle tuber had been reported from several provinces and was of concern to seed growers and breeders. However, as a consequence of rigorous control measures, spindle tuber has not been found in Canadian seed potatoes since 1980. The spindle tuber viroid can infect potato, tomato, eggplant, tobacco and several other species of broadleaved plants.

Symptoms The characteristic symptoms of spindle tuber are long spindly tubers *(16.28b)* that often are deformed by cracking and generally are smaller than those from healthy plants. Russet skins may become smooth and colored skins may lighten. Eyes may be more numerous and heavily indented with pronounced brows. Necrotic spots often appear around lenticels and in the flesh of infected tubers. Not all tubers from an infected plant may display symptoms.

In the field, infected plants are more upright and have fewer stems than do healthy plants. Leaves are folded upward with a ruffled margin and are smaller in size *(16.28a).* The leaves on affected plants often are attached to the stem at sharp angles, and the main petiole does not drop or curve as in viroid-free plants. Symptoms may vary with cultivar and environmental conditions. High temperatures early in the growing season favor the development of symptoms.

Causal agent Potato spindle tuber viroid is a circular ribonucleic acid molecule. It is smaller than a virus and devoid of the protein coat commonly associated with viruses. Conventional serological techniques will not detect this viroid. Biochemical techniques, particularly the return-polyacrylamide gel electrophoresis and nucleic acid hybridization procedures, are used for large-scale detection. For individual tests, inoculation to indicator plants can be employed. Potato spindle tuber viroid occurs in many strains.

Disease cycle Potato spindle tuber viroid can be transmitted by sap, direct contact, grafting and through true seed. It spreads in the field largely by mechanical means, such as seed cutting and handling, and cultivation practices. The viroid is important in breeding programs because it is transmitted in pollen and true potato seed. Insects capable of spreading the viroid include the Colorado potato beetle, grasshoppers, green peach and potato aphids, potato and other flea beetles and leaf beetles, and *Lygus* bugs.

Management

Cultural practices — Growers should plant high-quality, disease-free seed. Maintaining seed stock of new cultivars viroid-free will prevent the introduction of the pathogen into the seed-production system. Since 1980, the Canada Seeds Act has had a zero tolerance for potato spindle tuber viroid in all classes of seed potatoes.

Selected references

De Bokx, J.A., and P.G.M. Piron. 1981. Transmission of potato spindle tuber viroid by aphids. *Neth. J. Plant Pathol.* 87:31-34.

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(Original by R.P. Singh and I.R. Evans)

▶ 16.29 Witches'-broom *Figs. 16.29a,b*

Potato witches'-broom mycoplasma-like organism

Witches'-broom is a minor disease on potato. It occasionally is transmitted to potato crops that are grown near alfalfa, clover or other legumes that have the disease, or which are growing among grasses and other hosts that sustain the leafhopper vectors.

Symptoms Natural infections in potato usually occur too late in the season for foliage symptoms to develop or to influence tuber size. When tubers from infected plants germinate, however, they produce an unusually large number of stems *(16.29a,b)* and are somewhat chlorotic. These symptoms may appear at emergence or later during the growing season. Such plants produce numerous small tubers. In successive years, the degeneration usually continues until no plants or tubers are formed. However, complete recovery from witches'-broom has been observed three years after initial infection in the cultivar White Rose. Apparently the pathogen failed to remain fully systemic in all tubers and some eyes produced normal stems and tubers.

Causal agent Witches'-broom is associated with an organism that resembles the one associated with aster yellows. As with aster yellows, there is no proof that the mycoplasma-like organism is the cause. Three strains, all of which are associated with the same symptoms in potato, have been distinguished by symptoms induced by graft transmission to tomato and tree tomato (*Cyphomandra betacea* (Cav.) Sendtn.).

Disease cycle The witches'-broom pathogen can be transmitted to potato from other hosts by leafhoppers *(Scleroracus* spp.) but not from potato to potato because it is an unsuitable host for these leafhoppers. In nature, inoculum probably is acquired from infected legumes and is transmitted to potato when the leafhoppers migrate into potato fields.

Management

Cultural practices — Growers should use Certified or a higher class of seed grown in areas where witches'-broom has not been found. In affected areas, leafhopper migration into potato fields should be controlled by modifying cultural practices and by using insecticides, particularly on field margins.

Selected references

Deng, S.J., and C. Hiruki. 1990. Molecular cloning and detection of DNA of the mycoplasmalike organism associated with clover proliferation. *Can. J. Plant Pathol.* 12:383-388.

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(Original by N.S. Wright)

NON-INFECTIOUS DISEASES

A large number of non-infectious disorders can affect potatoes. These result from stresses such as mechanical damage, nutritional imbalances, temperature and moisture effects, poor soil aeration, chemical injury and air pollution. The common problems are described in the following sections.

▶ 16.30 Blackheart *Fig. 16.30*

Blackheart is primarily a disorder of stored potatoes. It is often seen in tubers stored in closed bins or in deep piles that lack adequate aeration. Blackheart develops when oxygen is limited or excluded from stored potatoes, particularly at temperatures above 15°C. It is seen occasionally in potato fields that were excessively wet before harvest, resulting in the internal tuber tissues having insufficient oxygen for respiration.

Symptoms Affected tubers develop an intense, blackish, irregular discoloration in the center *(16.30).* The blackened tissue is initially firm, but later becomes soft and watery.

Management

Cultural practices — Growers should avoid storing potatoes in closed containers or bins, or in deep piles that lack adequate ventilation. Storage temperatures should be reduced as soon as possible after harvest. Potato should not be grown on land that is subject to flooding or has poor drainage.

Selected references

Bennett, J.P., and E.T. Bartholomew. 1924. The respiration of potato tubers in relation to the occurrence of blackheart. *Calif. Agric. Exp. Stn. Tech. Paper.* 14. 41 pp.

Stewart, F.C., and A.J. Mix. 1917. Blackheart and the aeration of potatoes in storage. *New York Agric. Exp. Stn.* (*Geneva*) *Tech. Bull.* 436:321-362.

(Original by I.R. Evans and R.J. Howard)

▶ 16.31 Growth cracks *Fig. 16.31*

Several types of growth cracks can occur in potato tubers and can reduce quality. Thumbnail cracks result from rough handling and drying of the skin during or after digging. This disorder is aggravated by dry storage conditions. Cracks may also result from mechanical pressure or impact during harvest. Factors such as cultivar type, frost damage, top-killing and harvesting during cold soil conditions can induce growth cracking.

Symptoms Thumbnail cracking appears as if a thumbnail has been pressed into the skin of the potato *(16.31).* The cracks are usually shallow but tubers may be rendered unmarketable. Tubers may also split from internal pressure following excessively rapid growth, which usually occurs along the longitudinal axis of tubers. They may heal during the growing season and be of little consequence. Cracked tubers in the field and in storage are much more liable to be infected by bacteria and fungi that may cause them to rot.

Management

Cultural practices — Producers should follow good soil and crop management practices, especially when fertilizing, irrigating and spacing plants. Tubers should be handled as little and as gently as possible from harvesting to storage. Following storage, the tubers should be warmed prior to handling. Tubers should be stored at high humidity to minimize thumbnail cracking.

Selected references

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(Original by I.R. Evans and R.J. Howard)

▶ 16.32 Hollow heart *Fig. 16.32*

Hollow heart is a common disorder in oversized or rapidly growing, early maturing tubers. Up to half of the tubers of some cultivars may be affected. It is most severe under conditions that favor rapid tuber enlargement, such as unbalanced fertilization, dry soil conditions followed by high moisture levels, and soils with a low organic matter content, especially those that are well-drained and in areas where the annual rainfall may exceed 750 mm. Wide plant spacing or loss of hills can increase incidence of hollow heart. Potassium deficiency also has been implicated in this disorder.

Symptoms Tubers with hollow heart lack external symptoms. They usually are detected only after the potatoes have been cut in half. Initially, affected tubers exhibit a brown area at or near the center. Later, tan- to brown-walled angular cavities, up to 5 cm in diameter, develop at these sites *(16.32).*

Management

Cultural practices — Specific gravity tests or X-rays can be used to detect affected tubers. Close and regular spacing of hills, maintenance of uniform soil moisture levels, adequate potassium fertilization, and the planting of highly susceptible cultivars, such as Kennebec, closer together in the row will reduce hollow heart incidence.

Selected references

Finney, E.E., Jr., and K.H. Norris. 1978. X-ray scans for detecting hollow heart in potatoes. *Am. Potato J.* 55:85-105.

Crumbly, I.J., D.C. Nelson, and M.E. Duysen. 1973. Relationships of hollow heart in Irish potatoes to carbohydrate reabsorption and growth rate of tubers. *Am. Potato J.* 50:266-274.

(Original by I.R. Evans)

▶ 16.33 Jelly end rot

This disorder is most common in western Canada, particularly in the cultivar Russet Burbank. Affected tubers are unmarketable. Conditions that interfere with starch deposition, such as high soil temperatures and drought, followed by abundant mositure, are usually responsible for this problem. Jelly end rot is prevalent in misshapen tubers, particularly those with secondary growth. Because this disorder is strongly influenced by growing conditions, it tends to be seasonal in occurrence.

Symptoms The flesh at the stem end of the potato becomes glassy and jelly-like and then shrivels and dries up.

Management

Cultural practices — Consistent and adequate moisture conditions must be maintained during the growing season, particularly during tuber formation.

Selected references

Iritani, W.M., and L. Weller. 1973. The development of translucent end tubers. *Am. Potato J.* 50:223-233.

(Original by I.R. Evans)

▶ 16.34 Other physiological disorders *Figs. 16.34a-n*

Genetic abnormalities
Herbicide injury
Internal black spot
Internal sprouting
Nutritional disorders
Secondary tubers
Stem-end browning
Tuber greening
Miscellaneous disorders

Genetic abnormalities

Wildings are potato plants that are characterized by the production of foliage that is darker green than normal, numerous stems, and many small tubers. Affected plants rarely flower and yield is reduced. Feathery wildings are another variation of this problem and can result in multiple stems and reduced yields. The causes are unknown and control is by roguing these plants during the seed certification process.

A condition known as “giant hill” causes potato plants to mature later than normal, grow to be extremely large and vigorous, and produce numerous flowers. Tubers are usually few and rough. The cause is unknown and control is by roguing during the inspection of seed fields. The cause of pink discoloration in the flesh of white-fleshed tubers *(16.34a)* is also unknown.

Selected references

Dearborn, C.H. 1963. “Stitched end,” “giant hill,” and fasciated stem of potatoes in Alaska. *Am. Potato J.* 40:357-360.

Herbicide injury

The drift onto growing crops of specific herbicides may cause severe damage and significant yield and quality losses. Potatoes are sensitive to soil-borne residues of herbicides such as picloram, clopyralid, chlorsul- furon and dicamba. If potato is grown on land that has been recently treated with these chemicals, particularly picloram *(16.34b)*, severe yield losses, tuber malformation (*16.34c*) and stem-end discoloration may result. These chemicals can be taken up by seed tubers and subsequent crops may show typical herbicide injury symptoms. Potato foliage is sensitive to picloram at levels of less than one part per billion. Drift of picloram onto potato crops or soil residues of this herbicide may adversely affect potato yield and quality.

Internal black spot

Grayish spots develop just under the skin, particularly at the stem-end of tubers. Bruising and potassium deficiency are thought to cause this condition.

Internal sprouting

Tubers can sprout internally to the extent that they split and small tubers form inside *(16.34d).* Storage temperatures above 16°C, which physiologically ages the tuber, and insufficient concentrations of CIPC (isopropyl-m-chlorocarbanilate), a sprout inhibitor, may induce this disorder.

Nutritional disorders

Nitrogen, phosphorus, potassium, sulphur, calcium and magnesium are the major nutrients essential for normal potato growth. An absence or shortage of any of these can cause severe yield reduction. Magnesium deficiency is common on coarse-textured, acidic soils in eastern Canada.

Micronutrients generally considered essential for potato are boron, chlorine, copper, iron manganese, molybdenum and zinc. Some or all of these elements may be in short or limiting supply on coarse-textured (sandy) soils that are otherwise favorable for growing potato. Deficiency symptoms may resemble specific infectious or non-infectious diseases *(16.34e,f).* Excesses of micronutrients such as boron, aluminum and manganese also may cause considerable yield loss, particularly on acidic soils with a pH of around 5.0.

Nutritional disorders can generally be corrected by applications of fertilizers containing the missing elements. Most fertilizers are applied at or before planting, but applications can also be made in irrigation water (fertigation) or by spraying directly onto the foliage. It is advisable to use the results of soil and/or tissue analyses to properly gauge the amount of fertilizer necessary to correct the problem.

Selected references

Collier, G.F., D.C.E. Wurr and V.C. Huntington. 1980. The susceptibility of potato varieties to internal rust spot. *J. Agric. Sci.* **(***Cambridge***)** 94:407-410.

Dyson, P.W., and J. Digby. 1975. Effects of calcium on sprout growth and sub-apical necrosis in Majestic potatoes. *Potato Res.* 18:290-305.

Secondary tubers

In storage or following planting, tubers may form secondary, bead-like tubers but no shoots *(16.34g,j).* Storing tubers above 16°C or planting tubers into cold, dry soil will induce this disorder. The result may be hills with missing plants. Physiologically old seed tends to form more secondary tubers than fresh seed brought out of cold storage.

Selected references

Van Schreven, D.A. 1956. On the physiology of tuber formation in potatoes. 1. Premature tuber formation. *Plant Soil* 8:49-55.

Stem-end browning

Brownish streaks at the stem end of the tuber (*16.34k*) are often associated with rapid killing of potato tops by chemicals or frost. To minimize the risk of this disorder, growers should apply slow-acting top killers at least two weeks before harvesting the crop. Alternatively, reduced rates of chemical, higher rates of water to enhance coverage of vines, and two applications spaced one week apart should be considered. This will kill the vines more gradually. While these practices may prevent or reduce stem-end browning, they can increase the incidence and severity of infectious diseases that affect the tubers. Late blight, leafroll and rhizoctonia scurf are examples of diseases that may have more time to affect the maturing potato crop.

Selected references
Halderson, J.L., D.L. Corsini and L.C. Haderlie. 1985. Potato vine kill: stem-end discoloration effects on Russet Burbank. *Am. Potato J.* 62:273-279.

Tuber greening

Greening (formation of chlorophyll in the leucoplasts of tubers) results from exposure of tubers to sunlight or to artificial light in storage *(16.34h).* This disorder is serious in potatoes intended for consumption, since exposure to light also simultaneously increases levels of glycoalkaloids. Low levels of steroid glycoalkaloids (solanine and chaconine) are present in all potato tubers, but potatoes are considered unfit for consumption when glycoalkaloid levels exceed 20 mg/100 g fresh weight. Glycoalkaloids may impart a bitter taste, are toxic at concentrations over 40 mg/100 g and may be teratogenic. They are not decomposed or removed by normal cooking or food processing methods. Other factors that may induce high levels of glycoalkaloids in potatoes in the absence of greening are rough handling, Colorado potato beetle attack, and harvesting under very cold conditions.

Greening can be controlled by proper hilling, by avoiding cultivars that set tubers near the soil surface, by applying sufficient moisture to avoid soil cracking, and by storing tubers in darkness.

Selected references
Poapst, P.A., I. Price and F.R. Forsyth. 1978. Controlling post storage greening in table stock potatoes with ethoxylated mono- and diglyceride surfactants and an adjuvant. *Am. Potato J.* 55:35-42.
Rizk, A.F.M. 1991. *Poisonous Plant Contamination of Edible Plants.* CRC Press, Boca Raton, Florida. 183 pp.

Miscellaneous disorders

There are many other non-parasitic disorders of potato that are caused by such factors as air pollutants (sulphur dioxide and ozone), toxins from feeding by various insects, wet soil (enlarged lenticels, *16.34m),* low or high air and soil temperatures (sprouting, *16.34i;* tuber necrosis, *16.34n),* and wind, hail and lightning injury. In Ontario, atmospheric ozone levels of 70 ppb for one or two days have caused significant injury to potato foliage. Research there has shown that ozone, an indirect by-product of automobile exhaust, can defoliate susceptible cultivars, such as Norland, while tolerant cultivars, such as Kennebec, may hardly be affected. (For more information on these disorders, see Additional references.)

(Original by I.R. Evans and B. Otrysko)

NEMATODE PESTS

▶ 16.35 Northern root-knot nematode *Fig. 16.35*

Meloidogyne hapla Chitwood

Symptoms In potato, root-knot nematodes penetrate root and tuber lenticels. Scab-like lesions on tubers may make them unmarketable. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 16.36 Potato cyst nematodes *Fig. 16.36*

Golden nematode *Globodera rostochiensis* (Wollenweb.) Behrens
Pale cyst nematode *Globodera pallida* (Stone) Behrens

These two species of potato cyst nematodes have been introduced into Canada (see Introduced diseases and pests, 3.11). Both species occur in Newfoundland, and the golden nematode also occurs on Vancouver Island. Hosts of both species are potato, tomato, eggplant and other solanaceous plants.

Symptoms Foliage of affected plants appears pale and may wilt under dry conditions. Nematodes in large numbers can cause stunting, early senescence and root proliferation. In heavy infestations, plants look as though they are under water stress or suffering from a nutrient deficiency. Yield loss varies with nematode density and resistance level of the potato cultivar.

Identification *Globodera* spp. (order Tylenchida, family Heteroderidae) are morphologically similar and related to the sugarbeet cyst nematode *Heterodera schachtii.* In *Globodera*, the female body is subspherical to round before turning into a brown cyst; the genital opening (vulva) is a transverse slit between finely papillated, crescentic areas; and the cuticle surface between the anus and vulva is arranged in parallel ridges.

These two potato cyst nematodes differ in color before encystment. In *G. rostochiensis,* the female's cuticle is golden yellow, whereas in *G. pallida* it is white. Identification must be confirmed by a specialist. In the field, the non-specialist needs to know that a spherical white or golden- yellow cyst, about 1 mm in diameter, can be seen on potato roots if potato cyst nematodes are present *(16.36).*

Life history Eggs, stimulated by root exudates, hatch while encysted in the soil. Second-stage juveniles leave the cyst, migrate through the soil, and invade roots to feed. They molt three times before becoming adult. Juveniles penetrate the roots to feed near

the vascular bundles, inducing large multinucleate cells. While the rest of the female's body, which is white or yellow at this stage, enlarges and erupts through the root epidermis, the head remains inserted. Males feed and mature, then leave the root tissues, migrate through the soil, and mate with protruding females. Fertilized females fill with as many as 500 eggs. The body becomes a sub-spherical, hard, dark cyst that retains the eggs, which may remain viable for twenty years or more. The cyst may adhere to the roots or eventually become free in the surrounding soil.

Management

Cultural practices — Movement of potatoes and other solanaceous crops, soil, contaminated machinery, tools, and storage containers from quarantined areas is prohibited in Canada (see Introduced diseases and pests, 3.11).

Resistant cultivars — Races of both *Globodera* species vary in their ability to multiply on potato clones and hybrids, which has encouraged breeding for resistant potato cultivars to use in Newfoundland.

Selected references

Evans, K., and A.R. Stone. 1977. A review of the distribution and biology of the potato cyst-nematodes *Globodera rostochiensis* and *G. pallida*. *Pans* 23:178-189.

Mulvey, R.H., and A.M. Golden. 1983. An illustrated key to the cystforming genera and species of Heteroderidae in the Western Hemisphere with species morphometries and distribution. *J. Nematol.* 15:1-59.

Stone, A.R. 1973. *Heterodera rostochiensis.* CIH Descriptions of Plant Parasitic Nematodes, Set 2, No. 16. Commonw. Agric. Bureaux, England. 4 pp.

Stone, A.R. 1973. *Heterodera pallida.* CIH Descriptions of Plant Parasitic Nematodes, Set 2, No. 17. Commonw. Agric. Bureaux, England. 2 pp.

(Original by B.A. Ebsary and I.A. MacLatchy)

▶ 16.37 Potato-rot nematode *Fig. 16.37*

Ditylenchus destructor G. Thorne

This nematode is a serious pest of potato in many countries, but it has not been reported in Canada since the 1960s. For a description, see Foreign diseases and pests, 3.10.

▶ 16.38 Root-lesion nematode *Figs. 16.38; 16.38T1*

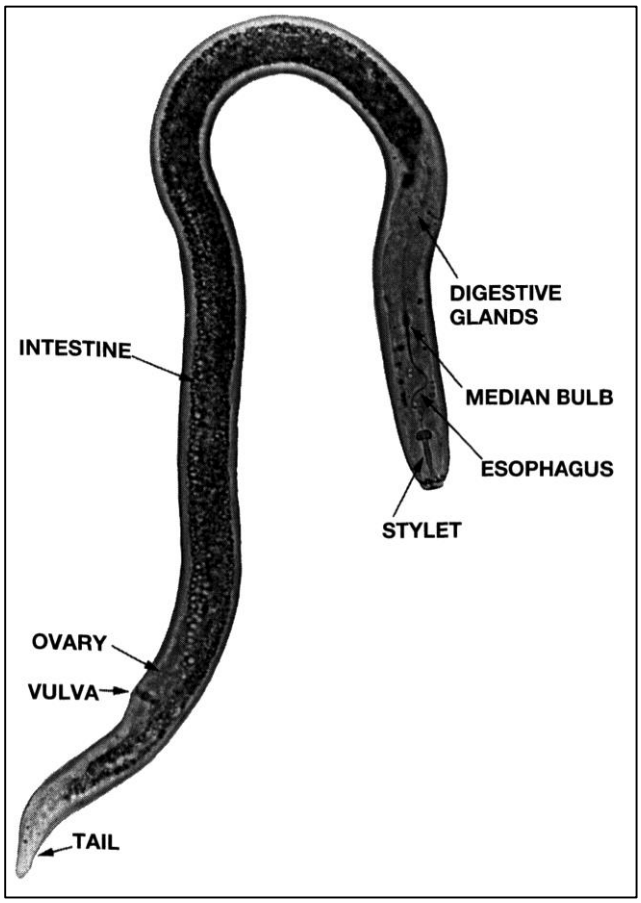
Pratylenchus penetrans (Cobb) Filip. & Stek.

The root-lesion nematode affects most of the major vegetable crops grown in Canada. Yield reductions of 10 to 40% have been observed in commercial potato fields and experimental plots in Ontario and Prince Edward Island.

Symptoms Plant growth is stunted in heavy infestations. Affected plants occur in patches, usually extending along the rows, or elongated in the direction of cultivation. Plants wilt readily on hot days and leaves become progressively yellow. Older leaves may die prematurely. Secondary roots are necrotic with dried areas. Yields of leaf vegetables are lowered. Tap root maturation is delayed and they may be smaller and branched. Tubers and bulbs are smaller and may be commercially down-graded *(16.38).* However, stunting, chlorosis and early senescence also are caused by other infectious and non-infectious diseases. Small lesions on feeder roots and black rotting are indicative of root- lesion nematode damage, but may not be sufficient evidence to conclude that nematodes are the sole problem.

Identification *Pratylenchus* spp. (order Tylenchida, family Pratylenchidae) are difficult to separate because of the small number of diagnostic characters at the species level and intra-specific variability.

Adults of *P. penetrans* are about 0.8 mm long. The head is low and broad. Stylet and labial sclerotization are well developed. The oesophageal glands overlap the intestine ventrally. In females (*16.38T1*), the genital opening (vulva) is posteriorly situated and the posterior branch of the ovary is reduced to a post-uterine sac. In males, the tail is pointed with a well-developed genital pouch (bursa).



16.38T1 Root-lesion nematode; female.

Life history *Pratylenchus penetrans* migrates through the soil and infects developing roots, preferentially in the root- hair zone. Second-stage juveniles through to the adults are migratory endoparasites, penetrating roots and migrating through the cortical tissue while feeding. To feed, they push their stylet through a cell wall, inject digestive enzymes secreted from the oesophageal gland, and ingest the cell contents. By repeatedly thrusting the stylet through the cell wall, they make a slit-like opening and move forward to feed on the next cell. The females lay eggs as they move through the tissues. A second-stage juvenile hatches from each egg and starts feeding on adjacent cells, progressively enlarging the zone of necrosis. Lesions enlarge and coalesce as the nematodes feed. The nematodes leave a decaying root when they become over-crowded. Soil bacteria and fungi can grow rapidly in the lesions and accelerate the decomposition of cortical root tissues. In soil, the nematodes are attracted to newly formed roots and to uninfested parts of the same root.

The life cycle on most crops varies with the type of host and soil temperature, ranging from 40 to 90 days at 25 to 15°C, respectively. When soil conditions become unfavorable for nematode migration or root growth, many juveniles and adults become quiescent for several months. Approximately 50% of the nematodes can survive two years in soil at 4°C. This nematode is also a moderate anhydrobiote, being able to enter a coiled, dehydrated state and survive in moderately dry soil for several months.

Management

Monitoring — Low to medium densities of nematodes in soil or roots (50 to several hundred per 100 mL of soil or per gram of roots) at mid-season do not generally constitute a serious threat to susceptible crops. However, when soil sampling reveals high populations (100 or more per 100 mL of soil) of root-lesion nematodes before planting, significant damage may result.

Cultural practices — In gardens, interplanting with certain marigolds *(Tagetes patula* L. and *Tagetes erecta* L.) and the use of solarization are effective. See also Management of nematode pests, 3.12.

Selected references

Ferris, J.M. 1962. Some observations on the number of root lesion nematodes necessary to cause injury to seedling onions. *Plant Dis. Rep.* 46:484-485.

Kimpinski, J., and K.B. McRae. 1988. Relationship of yield and *Pratylenchus* spp. population densities in Superior and Russet Burbank potato. *Ann. Appl. Nematol.* 2:34-37.
Olthof, T.H.A., and J.W. Potter. 1972. The relationship between population densities of *Pratylenchus penetrans* and crop losses in summer maturing vegetables in Ontario. *Phytopathology* 63:577-582.
Potter, J.W., and T.H.A. Olthof. 1977. Analysis of crop losses in tomato due to *Pratylenchus penetrans. J. Nematol.* 9:290-295.
Townshend, J.L. 1962. The root-lesion nematode, *Pratylenchus penetrans* (Cobb, 1917) Filip. & Stek. 1941, in celery. *Can. J. Plant Sei.* 42:314-322.

(Original by T.C. Vrain and J. Kimpinski)

▶ 16.39 Stubby-root nematodes

Paratrichodorus allii (Jensen) Siddiqi
Paratrichodorus pachydermus (Seinhorst) Siddiqi
Paratrichodorus spp.
Trichodorus spp.

This group of nematodes is not well established in Canada and has caused only minor damage to a few gardens in southern Alberta. Reports of damage to onion by *P. allii*, and of the transmission of corky ringspot disease of potato by *P. pachydermus* and possibly other species, are open to question. Several species of *Paratrichodorus* and *Trichodorus* induce the same symptoms and have the same common name. Stubby-root nematodes attack many types of vegetables, including bean, cabbage, corn and tomato. These nematodes are most prevalent and damaging in sandy loam or sandy soils; they are heat and drought tolerant.

Symptoms When stubby-root nematode density is high, which is rare in Canada, affected plants become stunted and chlorotic. Roots proliferate abnormally but appear not to grow in length and their extremities may be somewhat swollen. In potato, there is very little direct damage; however, large necrotic lesions form in tubers affected by these nematodes, which vector tobacco rattle virus, the cause of corky ringspot disease of potato (see corky ringspot, 16.25).

Identification Stubby-root nematodes (order Dorylaimida, family Trichodoridae) are short, thick nematodes with a non-axial, dorsal curved stylet (onchiostyle). Extraction and identification from representative soil and/or root samples are necessary to confirm that stubby-root nematodes are the cause of disease.

Life history Stubby-root nematodes are strictly ectoparasitic, feeding aggressively on epidermal cells and devitalizing root tips. They develop and multiply in the root zone without ever entering the plant roots. When new roots emerge, the nematodes immediately feed upon them. The roots stop growing and remain damaged, swollen and short.

Management *Cultural practices* — Solarization is effective in small areas to reduce numbers of stubby-root nematodes, which tend to be shallow feeders.

(Original by T.C. Vrain)

INSECT PESTS

▶ 16.40 Buckthorn aphid *Figs. 16.40a,b*

Aphis nasturtii Kaltenbach
(syn. *Aphis abbreviata* Patch)

The buckthorn aphid occurs from Manitoba eastward in Canada. The overwintering host is any of several species of buckthorn, such as the alder-leaved buckthorn (*Rhamnus alnifolia* L'Hér.), the European buckthorn (*R. cathartica* L.) and alder buckthorn (*R. frangula* L.). Secondary hosts include species of Solanaceae and some species of Cruciferae, Labiatae and Polygonaceae.

Damage The buckthorn aphid infests the lower leaves of potato. It rarely increases in numbers sufficiently to weaken a crop or reduce potato yields. Summer populations usually are localized within a field, where nevertheless this may be the most abundant species of aphid; in a dry year, it can spread throughout the field.

The buckthorn aphid is an effective vector of potato virus Y.

Identification The buckthorn aphid is the smallest of the potato-colonizing aphids. Adults range from 1.2 to 2.0 mm in length, the body is flattened, egg-shaped, lemon-yellow or green, the tip of the abdomen (cauda) has fewer than 10 hairs (setae), and the antennae do not have prominent tubercles. Winged adults have a conspicuous, dark brown to black head and thorax (*16.40b).*

Life history The buckthorn aphid overwinters as an egg on buckthorn, although in warm regions adult females also may survive the winter on weeds. In spring, the buckthorn aphid migrates from the overwintering host, at first colonizing weeds and by mid-July potato crops, *(16.40a).* Late in the summer, winged males and females appear and migrate to buckthorn. The winged females give birth to wingless sexual forms, which mate and lay the overwintering eggs.

Management

Monitoring — Abundance of the buckthorn aphid is estimated by counting the number of aphids on leaves. A sequential sampling plan, using 100 plants and a count of 0.6 or more aphids per plant, provides a satisfactory estimate of the distribution of aggregations of the buckthorn aphid. In central and eastern Canada, it is rarely economical to apply control measures to prevent direct damage to the plants. For that reason, the empirical threshold is set very high. In New Brunswick, for example, the action threshold is 150 aphids per plant and 80% of the plants infested.

Cultural practices — To reduce the spread of potato virus Y, mineral oil should be applied to seed-potato fields at weekly intervals at the first appearance of the buckthorn aphid (or other aphid vectors). Eradicating buckthorn shrubs from areas where potato is grown has been suggested, but the impact of this practice has yet to be evaluated.

Chemical control — Any recommended insecticide should be applied whenever sudden, rapid and significant increases in buckthorn aphid occur. The buckthorn aphid is susceptible to a wider range of insecticides than are other potato-colonizing aphids. However, because of its preference for the undersides of lower leaves, it may be more difficult to control. Nevertheless, chemical insecticides are the only means available for control of the buckthorn aphid when its populations are sufficiently high to affect yield or to increase the spread of virus diseases. No resistance to insecticides has been recorded in Canada.

(Original by G. Boiteau)

▶ 16.41 Green peach aphid *Figs. 16.41a,b*

Myzus persicae (Sulzer)

The green peach aphid occurs worldwide and is transcontinental in Canada, being present in all vegetable-producing areas. It is thought to be Asian in origin. In British Columbia and most of southern Ontario, the green peach aphid is the most abundant and potentially damaging potato-colonizing aphid. In the rest of Canada, it is secondary to other aphids, such as the potato aphid and the buckthorn aphid, because it colonizes potato late in the season and its populations rarely increase sufficiently to weaken the crop and reduce yields.

The overwintering hosts are peach *(Prunus persica* (L.) Batsch.), Canada plum *(P. nigra* Ait.) and black cherry *(P. serotina* Ehrh.). Although Canada plum is endemic along the southern border of Canada from Manitoba to New Brunswick, the green peach aphid rarely overwinters successfully on this host in New Brunswick. In some areas of the Prairie provinces, no overwintering host has been found.

Summer hosts number in the hundreds, belong to many plant families and include vegetable crops in the Solanaceae, Chenopodiaceae, Compositae, Cruciferae and Leguminosae. Among the vegetable crops, potato is a preferred summer host.

Damage The green peach aphid affects mainly the lower leaves of potato, but flowers and shoot tips also are subject to attack. The green peach aphid is the most important vector of potato leafroll virus, and it is an effective vector of potato virus A, potato virus S, and potato virus Y (including the tobacco necrotic strain).

Identification The adult green peach aphid is 1.2 to 2.5 mm in length and egg-shaped. The body has paired abdominal projections (cornicles) that are slightly swollen toward the tips, and the bases of the antennae have prominent, inwardly directed tubercles. Wingless adults *(16.41a)* are light green and almost translucent. Winged adults (*16.41b*) have a black or dark brown head and thorax, and a dark dorsal patch in the center of the abdomen.

Life history The green peach aphid gives birth to live young without mating (parthenogenesis) during the growing season. Later, in response to changing daylength and low fall temperatures, sexual forms appear, mate and lay eggs. The green peach aphid can overwinter in the warmer areas of Canada, such as southern Ontario and British Columbia, either as adult females on weeds and woody shrubs or as eggs on peach trees. This is why early planted potato in British Columbia and southern Ontario can be colonized by the green peach aphid soon after plant emergence in late May or early June. Almost everywhere else in Canada, early colonization of potato occurs less frequently because the aphids move from bedding plants or overwintering sites in greenhouses; in that case, colonization usually occurs later in the season and the aphids are thought to originate from the United States or from local weed hosts around greenhouses or storage areas.

In late summer, winged males and females are produced, but their numbers on potato dwindle as they seek overwintering hosts. Adults may survive in storage areas.

Management It is important to manage populations of the green peach aphid to prevent or reduce the spread of virus diseases in the potato crop.

Monitoring — The green peach aphid is usually located on the lower leaves of potato. The technique for monitoring the abundance of this and other potato-colonizing aphids is to count the number of aphids (winged and wingless) on 100 plants chosen at random while walking in a pattern (“X” or “W”) through the field. Counts are based on three compound leaves per plant, which should be taken from the upper, middle and lower parts of the plant because the different species of aphids do not distribute themselves evenly over the entire plant. This procedure often must be modified to suit the requirements of a given region. It may be necessary to adjust the counts for plant size in order to compare aphid populations on different potato cultivars or on potato crops grown under different conditions. If the plants are very small, they can be beaten over a tray, in which case all aphids are counted.

In general, the need for control is based on the occurrence of sudden and significant increases in the number of green peach aphids on potato plants or in monitoring traps. Traps provide information that can be related to the dispersal and redistribution of aphids. Because yellow traps attract aphids, they are used to monitor the time of aphid flights. Yellow water-pan traps are used in Prince Edward Island, New Brunswick and Ontario. Yellow sticky traps have been used in British Columbia. In Quebec and New Brunswick, estimates of the aerial population are obtained by using suction traps at heights of 1.5 and 12 m. Landing rates in the crop can be estimated by using leaf-green water-pan traps.

The empirical threshold for the green peach aphid on potato in New Brunswick is 25 aphids per plant, using a sampling unit of three compound leaves per plant, and 10% of all plants being infested; monitoring is continued until mid- to late August, after which control usually is not required. In British Columbia, the threshold is 30 aphids per compound leaf per plant and 20% of all plants infested.

In areas where seed-potato crops are grown, top-killing is recommended as soon as possible after the onset of flights of the green peach aphid. To indicate when leafroll may begin to spread within the crop, a threshold of five cumulative catches in any yellow water-pan trap is used in New Brunswick; in British Columbia, a significant increase in aphid catches serves as an indicator.

Cultural practices — To prevent or reduce the spread of virus diseases, growers should plant only Certified seed, and rogue and top-kill to eliminate infected plants as soon as possible after aphid flights begin. Mineral oil may be applied to seed-potato crops at weekly intervals to reduce the spread of mosaic. To limit the spread of virus diseases, some provinces establish mandatory dates each year for top-killing, which should be done as soon as tubers are suffficiently mature to escape injury.

Chemical control — To keep field populations of the green peach aphid low, thereby preventing the formation of winged forms that might spread virus diseases, growers usually have to apply chemical insecticides. In areas where early colonization from overwintering hosts or greenhouse colonies is likely, a systemic insecticide at planting will be effective for seven weeks or longer. This sometimes eliminates the need for further control measures. Resistance to pesticides has been reported for the green peach aphid in coastal British Columbia.

(Original by G. Boiteau)

▶ 16.42 Potato aphid *Figs. 16.42a-c*

Macrosiphum euphorbiae (Thomas)

The potato aphid is worldwide in distribution and probably Asian in origin. In Canada, it is transcontinental and the most abundant aphid on potato in eastern Canada and possibly across Canada. In Atlantic Canada, this is the first aphid to appear on potato. It usually can be found in June in most fields. It shows a preference for the middle and upper part of the plant, which is where the winged forms usually settle. High numbers can weaken the crop and reduce yields.

Overwintering hosts are Rosaceae, such as shining rose *(Rosa nitida* Willd.), swamp rose (*R. palustris* Marsh.) and rough rose *(R. rugosa* Thunb.). Summer hosts include a variety of vegetable crops and various other plants; potato is one of the preferred summer hosts.

Damage General characteristics of high numbers of the potato aphid include wilting of the plants and honeydew on the leaves. Mainly flowers and shoots are attacked.

The potato aphid transmits potato virus Y in tobacco, but almost never in potato. It is considered to be a poor vector of potato virus A and potato leafroll.

Identification Wingless potato aphid adults are 1.7 to 3.6 mm in length, which makes them the largest of the potato-colonizing aphids in Canada. The body is elongate, wedge-shaped, yellowish-green to pink and darker along the middle of the back *(16.42a,b).* The head has prominent antennal tubercles that are directed outwards. Winged adults have a pale yellow- brown, green-brown or dark brown head and thorax *(16.42c).* The potato aphid is quite restless and will drop when disturbed.

Life history The potato aphid overwinters as nymph-producing females in sheltered sites, such as greenhouses, and as eggs on rosaceous plants. The eggs hatch in the spring as the buds begin to swell. Two to six generations of females are produced on the overwintering host, during which time winged females mature and migrate to various weeds and to potato plants. Population build-up occurs between 5 and 25°C. Aphid numbers continue to increase in July and winged forms are produced. By early or mid-August, the population of *M. euphorbiae* on potato declines, mainly because winged males and females start to move to their overwintering hosts.

Management

Monitoring — Abundance of the potato aphid is estimated by counting aphid numbers on leaves (see green peach aphid). A sequential sampling plan, using 100 plants and a level of one or more aphids per plant, gives a satisfactory estimate of the distribution of the aphid. In eastern Canada, management is not necessary unless the potato aphid becomes sufficiently abundant to affect yields, which is why the empirical threshold is set fairly high. For example, in New Brunswick, the threshold is 50 aphids per compound leaf per plant and 40 to 60% of plants infested. In western Canada, management of the potato aphid rarely is required.

Biological control — Of the potato-colonizing aphids, the potato aphid is most subject to control by naturally occurring parasites, predators and fungi, but these biocontrol agents generally are not being used. In some integrated pest management programs in British Columbia, beneficial insects are a major component of aphid management strategies, especially in areas where the use of broad spectrum insecticides against other insect pests has been reduced or eliminated.

Chemical control — Any recommended insecticide can be applied whenever sudden, rapid and significant increases in abundance of the potato aphid occur. However, chemical control of the potato aphid is probably not economical in western Canada and may only be needed occasionally in central and eastern Canada.

(Original by G. Boiteau)

▶ 16.43 Other aphids *Figs. 16.43; 16.43T1, T2*

Black bean aphid *Aphis fabae* Scopoli
Bulb and potato aphid *Rhopalosiphoninus latysiphon* (Davidson)
Crescent-marked lily aphid *Aulacorthum circumflexum* (Buckton)
Foxglove aphid *Aulacorthum solani* (Kaltenbach)
Melon (cotton) aphid *Aphis gossypii* Glover

Black bean aphid —

The black bean aphid occurs worldwide and throughout Canada. It visits potato fields in eastern Canada and occasionally colonizes potato in western Canada. It has a very large host range.

Bulb and potato aphid —

The bulb and potato aphid occurs in British Columbia but has only rarely been reported on potato. It affects mainly tulip and gladiolus bulbs, and it infests the roots of many plants in the field. It may attack potato tubers in storage.

Crescent-marked lily aphid —

The crescent-marked lily aphid, also called the mottled arum aphid, occurs in British Columbia, where it is common on potato crops. It has a wide host range, but no overwintering hosts are known in Canada.

Foxglove aphid —

The foxglove aphid *(16.43)* is virtually worldwide in distribution. It occurs across Canada, extending into the Northwest Territories. It is only occasionally present on potato crops in eastern Canada and the Prairie provinces, but it can be as abundant as the potato aphid in British Columbia. Where the climate permits, it overwinters as eggs on a variety of hosts, such as common foxglove *(Digitalis purpurea* L.), plantain *(Plantago maritima* L.), red clover *(Trifolium pratense* L.), hawkweed *(Hieracium* spp.) and strawberry *(Fragaria* spp.). During the summer, it is found on numerous mono- and dicotyledonous plants, including potato.

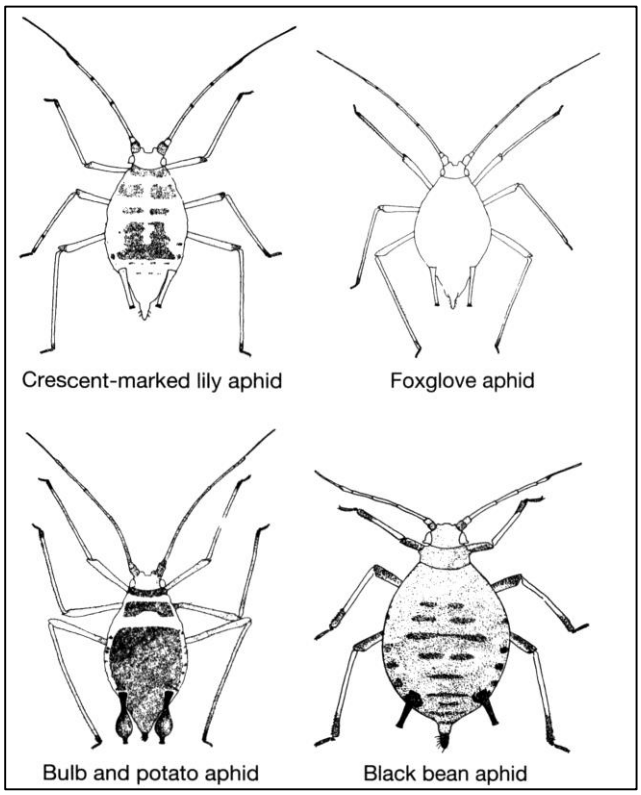
Melon aphid —

The melon (or cotton) aphid attacks many crops (see Greenhouse cucumber, 22.33). Recently, it has been a problem in commercial potato crops in British Columbia.

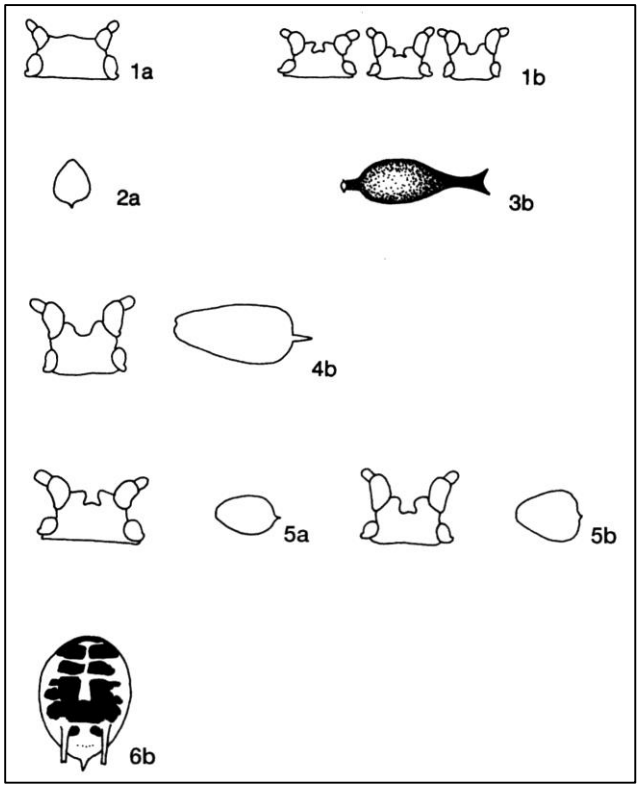
Damage The foxglove aphid can cause discoloration and irregular curling of the leaves, especially unfolding leaflets.

Table 16.43 Key to wingless female aphids colonizing potato in Canada

1a	Antennae shorter than body, antennal tubercles absent or weak	2
lb	Antennae as long as, or longer than, body, antennal tubercles prominent	3
2a	Body yellow to green, without dark marks on dorsum, cauda of abdomen with fewer than 10 setae	Buckthorn aphid *Aphis nasturtii*
2b	Body color variable with dark marks on dorsum (easily confused with *Aphis nasturtii*)	Melon aphid *Aphis gossypii*
2c	Body dull black, without dark marks on dorsum, cauda of abdomen with more than 10 setae	Black bean aphid *Aphis fabae*
3a	Cornicles brown to yellow-green, slightly swollen or not at all	4
3b	Cornicles shiny black, extremely swollen	Bulb and potato aphid *Rhopalosiphoninus latysiphon*
4a	Body ovoid or pear-shaped, antennal tubercles convergent inward or parallel sided	5
4b	Body elongate or wedge-shaped, antennal tubercles sloping outward	Potato aphid *Macrosiphum euphorbiae*
5a	Body pear-shaped, abdomen pigmented	6
5b	Body ovoid, abdomen unmarked	Green peach aphid *Myzus persicae*
6a	Thorax unmarked, abdomen pigmented at base of cornicles only	Foxglove aphid *Aulacorthum solani*
6b	Thorax with transverse bands or patches, abdomen with large black patch	Crescent-marked lily aphid *Aulacorthum circumflexum*



16.43T1 Aphids; four species that occasionally infest potato.



16.43T2 Aphids; body parts of wingless females described in Table 16.43.

Other aphids usually are not sufficiently abundant to be damaging, but they may cause loss of vigor in plants or sprouts. In general, none of these aphids has an economic impact on potato as a standing crop.

The foxglove aphid and the crescent-marked lily aphid transmit potato leafroll. Their scarcity excludes them as vectors of any importance, except in British Columbia. The black bean aphid transmits both leafroll and mosaic, but it is not a major factor in the spread of these diseases. Were it not for its rare occurrence, the bulb and potato aphid could be important as a vector of leafroll to potato in storage.

Life history The black bean aphid prefers young leaves and shoots. It overwinters as eggs on weed hosts. The bulb and potato aphid will breed continuously in storage areas. The foxglove aphid is seldom abundant on potato except in British Columbia, where it is most abundant during mid- to late August. It overwinters as eggs on weeds or as nymph- producing females inside greenhouses and other protected places. It prefers potato leaves close to the ground and occasionally it is found on sprouting tubers. The crescent- marked lily aphid overwinters either as an egg on weeds or as nymph-producing females.

Identification (see key to wingless female aphids, Table 16.43, and Figs. *16.43T1, T2*)**)**

Black bean aphid — The wingless adult is dull black. Winged forms are variably striped. The tip of the abdomen (cauda) characteristically has more than 10 hairs (setae).

Bulb and potato aphid — The wingless adult is shiny, dark or olive- green, and its paired abdominal projections (cornicles) are shiny, black and swollen in appearance. The nymph is paler green but it also has shiny, black, swollen cornicles. The winged forms have shiny, olive-green to black, dorsal abdominal marks.

Crescent-marked lily aphid — No winged adults are produced. The adult is shiny, white to bright-green, and has a horseshoe- or crescentshaped patch on the back (dorsum) of the abdomen.

Foxglove aphid — The wingless adult is shiny, light yellow-green to dark green, and darker at the base of the cornicles. It is pear-shaped, globular, and widest just in front of the cornicles. The foxglove aphid is larger than the green peach aphid and smaller than the potato aphid.

Melon aphid — The adult varies in color but has black marks on the back of the abdomen, which helps distinguish it from other potato-colonizing aphids. The melon aphid may be confused with the buckthorn aphid. (For more on the melon aphid, see Greenhouse cucumber, 22.33.)

Management

Monitoring — These aphids rarely require control and there are no empirical thresholds.

Chemical control — No resistance to insecticides has been reported in Canada for any of the above species. Any recommended insecticides can be applied if needed.

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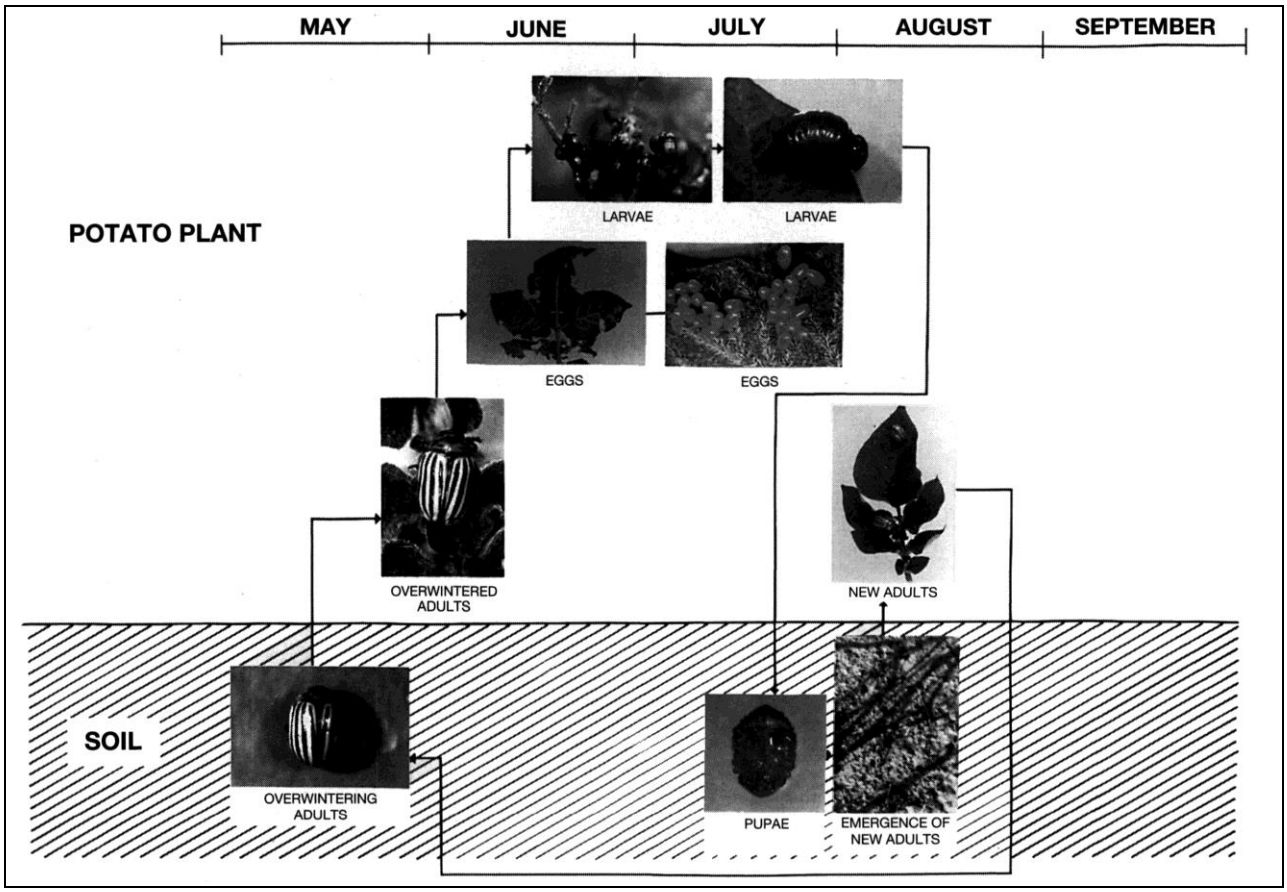
(Original by G. Boiteau)

▶ 16.44 Colorado potato beetle ***Figs.****16.44a-d; 16.44T1*

Leptinotarsa decemlineata (Say)

THE COLORADO POTATO BEETLE

LEPTINOTARSA DECEMLINEATA (SAY)



16.44T1 Colorado potato beetle; life cycle in regions with one generation.

The Colorado potato beetle probably entered Canada first in Ontario in 1870, reaching Prince Edward Island by 1883 and British Columbia by 1919. To date, it is absent from insular Newfoundland, and from coastal British Columbia and Vancouver Island. It is the most important insect pest of potato from New Brunswick and Prince Edward Island to Manitoba and in Alberta. In Nova Scotia, Saskatchewan and interior British Columbia, it is a secondary or rare pest.

Hosts of the Colorado potato beetle are limited to the Solanaceae. The beetle prefers potato but it also attacks eggplant, tomato and weeds, such as ground cherry *(Physalis* spp.) *(3.11c),* wild tomato *(Solanum triflorum* Nutt.), and bittersweet or climbing nightshade *(S. dulcamara* L.).

Damage The adult and all larval stages feed mostly on foliage, chewing irregular holes in and along leaf margins, but they also may attack stems. High populations can completely defoliate plants (*16.44c*) throughout large portions of a field. Extensive feeding at any time during the season, especially when the crop is in bloom, can reduce yield. Generally, a reduction in leaf surface decreases the ability of potato plants to produce nutrients for storage in the tubers.

The suggestion that the Colorado potato beetle might be a vector of certain bacterial and viral diseases of potato has not been confirmed, but mechanical transmission is likely.

Identification The Colorado potato beetle (family Chrysomelidae) adult is about 10 mm long and 7 mm wide, and somewhat rounded. Its head and anterior thorax (pronotum) are brown-orange to yellow and covered with variously shaped black markings. Ten black lines run the length of the forewings (elytra), which otherwise are pale yellow *(16.44a).* Females can be recognized by their greatly distended abdomen and the absence of a depression in the last abdominal segment when viewed from below. The eggs are elongate and yellow to orange, and usually they are laid on the underside of leaves of the host plant in clusters of about 30 (*16.44b).* The larva is humpbacked, and red-orange with two rows of black spots along the sides of the body (*16.44c,d).*

The Colorado potato beetle cannot be confused with any other beetle in Canada. It most resembles *Leptinotarsa juncta* (Germar), a similar species limited to an area southeast of Pennsylvania in the United States.

Life history The adult overwinters in the soil of the previous year's potato fields (*16.44T1*). As the temperature increases in the spring, the beetles move upward in the soil. They first appear in the last week of May or early June, and immediately seek host plants. They feed for a few days, after which mating (*16.44a*) and egg laying occur. Individual females lay 300 to 500 eggs from

June to late July. During this period, they may move from older to younger plants. The extended oviposition period means that larvae may be present in the field for three to five weeks, although larval development from hatch to pupation requires only two to three weeks. Pupation occurs in the soil and new adults appear after a further one to two weeks. In most regions of Canada the poor quality of the host food, the short daylength (photoperiod), and relatively low temperatures during the emergence period prevent the new adults from mating. Instead, they feed and prepare for overwintering (reproductive diapause). Thus, there is only one generation of the beetle throughout most of Canada except in southern Ontario, where there are two generations. Occasionally in New Brunswick, Nova Scotia and Prince Edward Island, some of the new adults emerge early enough to mate and lay some eggs, creating a partial second generation. However, these mated adults soon stop producing eggs and start preparing for overwintering. The partial second generation usually develops too late in the season to have a significant impact on potato yield in those provinces. In southern Ontario, however, the second generation is complete and in some years there is even a partial third generation.

Management

Monitoring — The density of egg, larval, and adult stages can be estimated by visual counts on a fixed number of whole plants or plant stems chosen randomly from different parts of the field. For medium-maturing potato cultivars, a sequential sampling plan for larvae in their third and fourth instar assumes an economic threshold of 20 large larvae per plant. As many as 40 plants must be examined to obtain a valid estimate of beetle density. Sampling by sweep net is not suitable for estimating numbers. Currently, there are no specific economic thresholds. Recommendations to commence control are based solely on empirical observations: leaf damage exceeding 10% in Manitoba; an average of two larvae per plant in 12 m of row in Atlantic Canada; and, in Quebec, 3.5 overwintering adults per plant, 10 larvae per plant before 90% bloom, or 20 adults per plant toward the end of the season. In Manitoba, the economic injury level ranges from 0.14 to 0.82 larvae per plant on the potato cultivar Norland. There is on-going research in New Brunswick, Manitoba and Quebec to establish economic thresholds. Present information suggests that only very low levels of Colorado potato beetle do not justify control, and that the economic threshold is affected by the intensity of other factors stressing the crop.

Cultural practices — Apart from the use of insecticides, crop rotation is one of the few control techniques currently available to potato growers. Rotation can significantly reduce beetle numbers, and it also results in the initial localization of incoming beetles at the periphery of the potato field, where insecticides can then be applied as a spot treatment. Not all growers can use this strategy, because they may not have the land needed for rotation and alternative crops tend to be less profitable than potato. In these cases, it is best to avoid planting in fields where high numbers of adult beetles were present at the end of the last growing season.

Resistant cultivars — None of the present commercial cultivars of potato is resistant to the Colorado potato beetle. Research is in progress to develop cultivars with low to moderate levels of resistance, which might then be used along with chemical and other control techniques.

Biological control — Populations of native predators, which include the ground beetles *Lebia* and *Pterostichus* spp., the two-spotted stink bug *Perillus bioculatus* (Fabricius) and the spotted lady beetle *Coleomegilla maculata* (DeGeer), and the parasite *Myiopharus doryphorae* (Riley) (syn. *Doryphorophaga doryphorae* (Riley)) rarely have a significant impact on the abundance of the Colorado potato beetle. Strains of the bacterium *Bacillus thuringien- sis* Berliner have been registered and have good potential for commercial use because of their specificity to Colorado potato beetle.

Chemical control — An array of systemic and foliar insecticides is available. Growers are encouraged to spray only when necessary and to alternate their choice of insecticides from among the different chemicals available.

Resistance to organochlorine insecticides was reported as early as the 1960s in Alberta, Ontario and Quebec. More recently, Colorado potato beetles in one part of Quebec were found to be resistant to most of the currently recommended organophosphate, carbamate and pyrethroid insecticides. In New Brunswick, the genetic potential for resistance is present in many locations, although to date it has expressed itself only in a few populations. The spread of resistance has been limited, considering that populations of this insect have developed insecticide resistance on occasion in different parts of Canada. Spot treatments with foliar insecticides should be applied against adults early in the season where numbers warrant. Insecticides for control of the larval stages should be applied during bloom where numbers exceed the economic threshold, and at the end of the season for control when unusually high numbers of adults are present.

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(Original by G. Boiteau)

▶ 16.45 Potato flea beetle *Figs. 16.45a-c*

Epitrix cucumeris (Harris)

The potato flea beetle is found in all Canadian provinces except British Columbia. In the eastern United States, it extends as far south as Florida.

The potato flea beetle is primarily a pest of potato but it also attacks cucumber, eggplant, pepper, tomato and other plants, such as jimsonweed (*Datum stramonium* L.), tobacco *(Nicotiana tabacum* L.), large white petunia (*Petunia axillaris* (Lam.) BSP.), ground cherry (.*Physalis pubescens* L.), horse nettle (*Solanum carolinense* L.), bittersweet or climbing nightshade (*S. dulcamara* L.), black nightshade (*S. nigrum* L.), and Jerusalem cherry *(S. pseudocapsicum* L.).

Damage Feeding by adults on leaves in spring or late summer results in rounded feeding scars, 0.1 to 5 mm in diameter *(16.45a),* which frequently penetrate through the leaf to form a hole. When the potato flea beetle is abundant, potato leaflets sustain numerous perforations, eventually exhibiting a "shot-hole" appearance *(16.45b).* Direct damage to tubers caused by larvae generally is minor and can be removed by peeling.

The potato flea beetle is not considered a major pest in commercial potato production in Ontario and Quebec. It is most damaging in the Maritime provinces, where yield losses of 10 to 25% have been shown. Early season feeding by adults normally is not considered significant and insecticides used against the Colorado potato beetle will kill adult flea beetles. The most significant direct damage by the potato flea beetle in Canada is from late-summer feeding by adults in crops that do not receive insecticide late in the season. In Manitoba, Norland potato can withstand a peak adult density in August of up to 100 beetles per plant, although yield declines sharply when beetle numbers exceed that density.

The incidence of the potato flea beetle and the severity of common scab have been correlated positively in Manitoba. By their feeding, larvae may directly transmit pathogens to tubers and roots and increase secondary infection of damaged tubers. Also, adults may spread pathogens when they emerge from pupation sites in the ground. Fungal diseases associated with the potato flea beetle include common scab, fusarium dry rot, rhizoctonia, and verticillium wilt. In addition, bacterial diseases and potato spindle tuber viroid may be transmitted mechanically.

Identification The potato flea beetle (family Chrysomelidae) adult is 1.7 mm long and 1 mm wide. It is black with brown legs and brown antennae *(16.45c).* The femur of the hind legs is thicker and darker than the other leg segments. The larva is small, slender and white with a dark brown head and minute legs. When fully grown, it is about 5 mm in length. As a rule, larvae inhabit the soil around potato roots. On occasion, they may enter the tubers, forming small hollows at the point of entry and tunnels, which usually are straight, about 0.8 mm in diameter and less than 6 mm long, and filled with corky tissue. This type of tuber injury contrasts with the deeper, more penetrating tunnels made by the tuber flea beetle.

Life history The potato flea beetle has one generation per year. Adults overwinter at the soil surface among litter or undergrowth either in or near the potato fields where they fed the previous summer. In spring, they move to potato fields and feed on the foliage of newly emerged potato plants. They fly and jump actively, particularly when disturbed, and they may be found on all above-ground parts of the potato plant and on the soil surface. They feed on both the upper and lower leaf surfaces, but more frequently on the upper surface. If potato plants have not yet emerged, the beetles feed on weed hosts. Females lay eggs in the soil around potato plants and then die. Usually all overwintered adults die by first bloom of potato plants.

Larvae hatch about one week after the eggs are laid. They feed primarily on the fine roots of potato plants, completing development in about four to five weeks. They pupate in the soil and become adults in about one week. These new adults emerge from the soil, usually in late July or August, and feed on potato leaves. The number of adults increases rapidly and may exceed 100 per plant in August. Adult feeding continues on potato leaves until the weather becomes too cold or the foliage becomes unsuitable. Adults that leave potato plants may feed on other favored hosts before entering overwintering sites.

Management

Monitoring — The enumeration of feeding punctures is a much better method of assessment than counting the mobile adults. A preliminary estimate of the economic threshold for the cultivar Norland in Manitoba is 65 to 75 feeding punctures per terminal leaflet from the lower third of the plant taken two weeks after the first appearance of bloom. This threshold may need to be lowered for plants that are under stress from weather or from attack by other insects or pathogens. Later in the season, however, the number of feeding punctures may exceed this threshold without signifying a need for control.

The threshold also may differ for other cultivars and in other regions of Canada. In Atlantic Canada, an economic threshold of 15 feeding punctures on the fourth terminal leaflet (counting down from the apex of the plant) has been advocated, but this number now appears to be too low.

Cultural practices — The potato flea beetle tends to be more abundant in parts of potato fields that are adjacent to uncultivated areas. Such areas often have suitable food hosts for the adult beetles and retain snow, which may enhance their survival over winter. Because the adult potato flea beetle does not readily fly, the separating of uncultivated areas from potato fields may reduce populations. Eliminating volunteer potato and other hosts also may be used to starve overwintered adults in the spring before potato crops emerge.

Resistant cultivars — None of the commercial cultivars of potato is known to be resistant to the potato flea beetle.

Biological control — Parasites do not appear to have a significant impact on the population density of the potato flea beetle. At present, no biocontrol agent is commercially available.

Chemical control — Insecticides used to control other insect pests of potato also control flea beetles. For instance, granular systemic insecticides applied at planting to kill adults of the Colorado potato beetle may be effective against the potato flea beetle for the whole season. Where systemic insecticides are not used at planting, the need for control of the potato flea beetle depends on the timing of the appearance of its immature stages relative to other insecticidal applications. For example, in Manitoba, foliar applications against the Colorado potato beetle usually are applied when the potato flea beetle is a larva or pupa in the soil, which protects it. Potato flea beetle adults emerge from the soil in large numbers over several weeks, so a foliar insecticide that is relatively persistent but which does not cause residue problems at harvest, is desirable. Insecticides recommended against emerging adults should be applied about two weeks after the first adults appear.

Resistance to currently used insecticides has not been reported. However, the potato flea beetle seems able to detect and thus avoid insecticides, such as malathion, on potato foliage sufficiently to reduce both mortality and the effectiveness of the treatments.

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(Original by N.J. Holliday and J.G. Stewart)

▶ 16.46 Potato leafhopper *Figs. 16.46a,b*

Empoasca fabae (Harris)

The potato leafhopper is found throughout the Western Hemisphere, but it does not overwinter at more temperate latitudes. Each year it migrates into Canada, dispersing from the Gulf Coast of the United States on southerly winds. From early to mid-June until fall, it usually can be found in Manitoba, southern Ontario and Quebec. It is found only occasionally in Saskatchewan, New Brunswick, Nova Scotia, Prince Edward Island and Newfoundland.

The potato leafhopper is a pest of alfalfa and occasionally clover *(Trifolium* spp.). Vegetable crops affected include bean, celery, corn, cucumber and potato. The host range numbers over 100 broad-leaved plants, including apple and other trees.

Damage Damage by potato leafhopper depends on the density of the insects, the duration of their feeding, and the growth stage of the plants. Both nymphs and adults feed on stems and leaves, and they seriously affect seedlings. Damage to potato is characterized by yellowing and eventual death of the affected foliage. Initially, the damage consists of yellowing at the tips and margins of the leaflets. Gradually, the leaf margins die and roll inward, resulting in the typical "hopperburn" symptom *(16.46a).*

Although the potato leafhopper is present every year in Manitoba, only once in six or more years are its numbers high enough to cause hopperburn. However, in southern Ontario, the potato leafhopper has been shown to reduce potato yields by approximately 40% when it exceeded the nominal threshold (see Monitoring) and was left to feed uncontrolled for the duration of the season. If modifications to current control procedures for Colorado potato beetle were to incorporate the use of more pest-specific insecticides, natural enemies and cultural practices, the potato leafhopper likely would become a more important pest.

No disease is known to be transmitted by the potato leafhopper.

Identification Leafhoppers (family Cicadellidae) are wedge shaped, which helps distinguish them from other small insects. The adult potato leafhopper is 3-4 mm long, bright yellow-green and otherwise unmarked *(16.46b).* It is very active and flies readily when approached. Nymphs are also yellow-green. They usually remain on the foliage, moving to the opposite side of the leaf when disturbed.

Life history Potato leafhopper adults generally arrive in southern Ontario in early June and are often found on alfalfa, which is their preferred host for laying eggs. The nymphs hatch in about 10 days and feed on alfalfa foliage until they mature. The adults from this generation then invade potato and initiate one or more generations on that crop. There are two generations on potato in southern Ontario, but usually only one in the rest of Canada.

Females may lay three to five eggs per day and up to 35 eggs in a lifetime. The eggs are inserted into the plant stem. There are five nymphal instars, of which the last two cause more damage than the earlier stages or the adult. Nymphal development requires 10 to 25 days. Maximum development occurs at 30°C.

Management

Monitoring — A nominal threshold of 10 nymphs per 100 mid-plant leaves has been established. Leafhopper populations above this threshold for two or more growth stages of the plant, such as vegetative through flowering or flowering through

senescence, or feeding for more than three or four weeks can cause a significant yield loss. On a commercial scale, no monitoring programs are in effect in Canada.

Cultural practices — At present, alternative approaches to control the potato leafhopper are not available.

Biological control — There are no important natural enemies of the potato leafhopper in Canada, at least none that appreciably affects the growth of its populations.

Chemical control — In Canada, control of the potato leafhopper has not progressed beyond the use of chemical insecticides intended for control of the Colorado potato beetle. Several foliar organophosphate and pyrethroid insecticides are effective against the potato leafhopper. The choice usually is dictated by materials that are most effective against the Colorado potato beetle but which also control other pests, including the potato leafhopper. Suggestions include better timing of foliar insecticide applications during the season and the use of systemic granular insecticides at planting. No information is available on resistance to insecticides in potato leafhopper populations.

(Original by M.K. Sears)

▶ 16.47 Potato stem borer *Figs. 16.47a,b; 12.22b*

Hydraecia micacea (Esper)

The potato stem borer, popularly known as the rosy rustic moth in Europe, was introduced into North America in Nova Scotia and New Brunswick in the early 1900s. It now occurs in eastern Canada, including Newfoundland, and in the mid-western United States. To date, there are no confirmed records from western Canada.

The potato stem borer is an occasional pest of a variety of cultivated vegetables, including corn *(12.22b),* onion, pea, potato, tomato and rhubarb. In Canada, the main vegetable crop hosts are potato in Newfoundland, corn and other vegetables in New Brunswick and Nova Scotia, corn in Prince Edward Island, corn and potato in Quebec, and corn and rhubarb in Ontario. Other hosts include strawberry, sugar beet, barley, wheat, *Gladiolus,* wild plants such as dock *(Rumex* spp.) and plantain *(Plantago* spp.), and grasses.

Damage Problems occur most frequently in newly established fields with weedy grasses. Plants are attacked when young. In late spring, the larva enters and feeds within the stem, causing the stem to wilt and die, and eventually killing the plant. Generally, larval feeding kills or severely damages the plant or, in the case of rhubarb petioles, makes the crop unmarketable. Although a variety of crops may be severely affected in individual fields, widespread reductions in crop yield seldom occur. This insect makes production costs marginally higher because of increased seeding to compensate for damage near fencerows and the need for additional plowing.

In New Brunswick, the potato stem borer has been associated with blackleg of potato, of which it may be a vector.

Identification The potato stem borer is a moth (family Noctuidae). It lays eggs that are finely ribbed and shiny. The larva has a pale brown head and pink body (*16.47b; 12.22b).* The pupa has two slender spines at the anal end. The moth itself measures about 42 mm across the wings *(16.47a).* It has dark, olive-brown shading on an otherwise pale brown forewing, and a dark median band that curves inward at the leading edge. The hindwing is yellow-brown with a diffuse, central, gray spot and a darker transverse line.

Life history Eggs are laid in August in parallel rows on leaf blades, usually on grasses, on which they overwinter. The larvae hatch in early May and feed on grasses in and around cultivated fields. As the larvae mature, they move to larger plants and feed inside the stem or root crown. They become fully grown by mid-July and pupate in the soil. The moths are active from late July to late September. Migration is suspected but the patterns are not known. Hay that is cut in the fall is a potential medium for the spread of eggs of the potato stem borer and may be how it was introduced to North America.

Management

Monitoring — A sex pheromone has been identified and is very effective in trapping male moths. The pheromone could be used as a monitoring tool, although present procedures are limited to scouting for damage during July.

Cultural practices — Populations of the potato stem borer are best controlled by clean cultivation, either by plowing or by the use of herbicides to remove weedy sites and reduce egg numbers on wild plants. New ground for crops should be plowed immediately after haying, and seeding should be at a higher rate in the border rows to offset seedling mortality.

Biological control — In North America, the tachinid fly *Lydella radicis* Townsend parasitizes potato stem borer larvae. Several wasps are known to parasitize the eggs, larvae *(3.7u)* and pupae.

Chemical control — Contact insecticides are unsuitable for use against the potato stem borer, because its larvae generally feed inside the plant. Systemic insecticides are not cost-effective.

(Original by R.J. West)

▶ 16.48 Tuber flea beetle *Figs. 16.48a-c*

Epitrix tuberis Gentner

The tuber flea beetle is the most serious insect pest of domestic and commercial potato crops in British Columbia. It has been found recently in home gardens in Alberta, and it also occurs in the United States.

The preferred host of the tuber flea beetle is potato, although feeding and reproduction occur on tomato and other solanaceous plants.

Damage Feeding by adults is seldom of economic concern. However, large numbers of adults can defoliate and kill young potato plants, especially in small potato fields and home gardens. Commercial potato crops are invaded in a number of ways. For instance, in second-year potato fields, overwintered beetles may emerge directly from the soil and infest volunteer potato plants or the emerging crop. In first- and second-year potato fields, tuber flea beetles from surrounding headlands tend to migrate mostly into the outer rows. Thus, damage to potato crops is usually greatest in the border rows. Even small populations early in the year are considered potentially dangerous to late-harvested potato cultivars.

Larval feeding on the developing tubers is a major economic concern to commercial growers. Although potato yields usually are not affected, the larvae cause cosmetic tuber damage in the form of pimpling, surface channels and shallow networks of fine tunnels *(16.48a,b).* Early potato crops generally escape serious injury because they are harvested before the populations have increased to economic levels. Mid- and late-season potato crops are subject to severe damage from second- and third-generation larvae in August and September, when the tubers are maturing. Should significant tuber damage occur, the crop might be considered unmarketable, or the grade may drop from Canada No. 1 to Canada No. 2.

The relationship between feeding by the tuber flea beetle and disease incidence in potato crops has not been established as it has for the potato flea beetle.

Identification The tuber flea beetle (family Chrysomelidae) adult is small, 1.5 to 2.0 mm in length, black and shiny *(16.48c).* Feeding by adults gives potato leaves a characteristic "shot-hole" appearance. Other insects, such as caterpillars and springtails (Collembola), also cause holes in potato leaves, making observation necessary. Larval feeding is restricted to the roots or developing tubers. Their presence is not conspicuous above ground.

Life history The tuber flea beetle overwinters as an adult in the soil in and around potato fields. Winter survival is highest in elevated, grassy headlands that are free from flooding. There are two to three generations per year, starting between mid-May and early June when the overwintered adults emerge. Adults feed and mate on the upper surfaces of the leaves of potato plants from morning until dusk. They rapidly jump from the plant if disturbed, and they can fly. Mating and egg laying may continue for a month. First-generation larvae feed from early June to mid- July, second-generation larvae from mid-July to mid- September, and third-generation larvae after mid-August. The larval stage is completed within three weeks and is followed by a pupation period of about two weeks. One complete life cycle normally takes about six weeks.

The potential for population increase is high; theoretically, a single female that emerges in May can give rise to thousands of progeny by fall. Soils that have high moisture levels and relatively high temperatures, such as organic soils, favor population growth. Thus, potato crops in muck soils are usually at greater risk than those in mineral soils.

Management

Monitoring — Monitoring the adult tuber flea beetle throughout the growing season ensures efficient and cost-effective control. Because beetle populations are higher in outer rows than in inside rows, these two areas usually are sampled separately. Plants must be inspected on a weekly basis from the time of early emergence until the crop is 30 cm tall. The procedure requires that 35 samples of 10 consecutive plants be thoroughly inspected, each sample being taken at intervals of 40 paces along the perimeter rows of the field. An equivalent number of samples should be taken along randomly selected rows inside the field. If an average of one beetle per 60 inspected plants is observed, a spray should be applied to the inner rows or to the perimeter rows, as required.

Thorough application of control measures early in the season will eliminate the need to spray later for tuber flea beetle. Moreover, late-season spraying may damage plants and may result in aphid outbreaks. When plants are taller than 30 cm, samples must be taken on a weekly basis with a sweep net. Sweeping is done by striking the tops of the plants vigorously with the net while walking, taking 20 consecutive 180° sweeps at 30-pace intervals along the outside rows. An equivalent number of samples is required along arbitrarily selected inner rows of the field. A spray is required if an average of more than one beetle per 10 sweeps is observed in either the outer or the inner rows, using a 30 cm diameter sweep net.

In new fields of potato, an inexpensive and effective strategy for routine sampling is to plant about 20 perimeter rows earlier than the main crop. These perimeter rows can be treated with a systemic granular insecticide or can be sprayed if the number of beetles reaches the threshold determined by the monitoring program. Sampling for flea beetles and other pests inside the field must still be performed, but sprays need only be applied when necessary. This procedure should not be attempted without a monitoring program.

Cultural practices — In British Columbia, yearly crop rotation is the key cultural practice, because beetle populations build up in fields planted repeatedly to potato. Furthermore, volunteer potato plants are host to the beetle early in the season, providing a source of infestation for later spread to the main crop. New fields of potato also may be invaded, but the beetles will be fewer and usually will be confined to the outer rows.

Chemical control — Normally, only mid- and late-season potato cultivars require chemical control of the tuber flea beetle. The most effective procedure for commercial plantings is to apply systemic insecticides to the seed furrow in May and June. Uptake of the insecticide into the leaves, upon which the overwintered adults feed, can provide excellent control before the adults lay eggs. The systemic insecticides work best in soils low in organic matter. Foliar sprays can be applied routinely or timed according to the results of population sampling, which must be done on a weekly basis from crop emergence until harvest.

In the interior of British Columbia, the tuber flea beetle has shown some degree of resistance to chlorinated cyclodiene insecticides.

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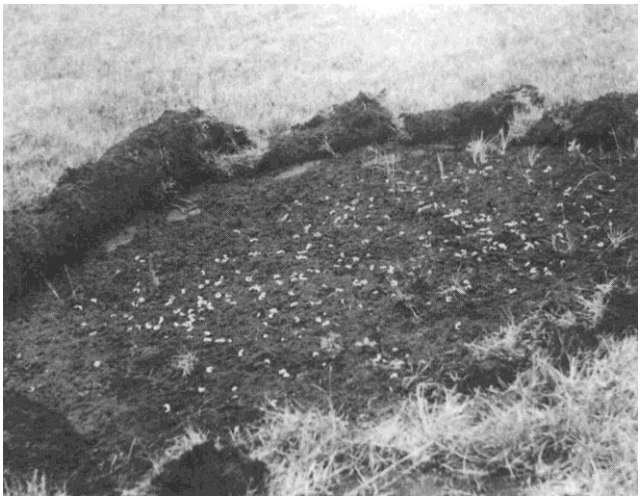
(Original by R.S. Vernon)

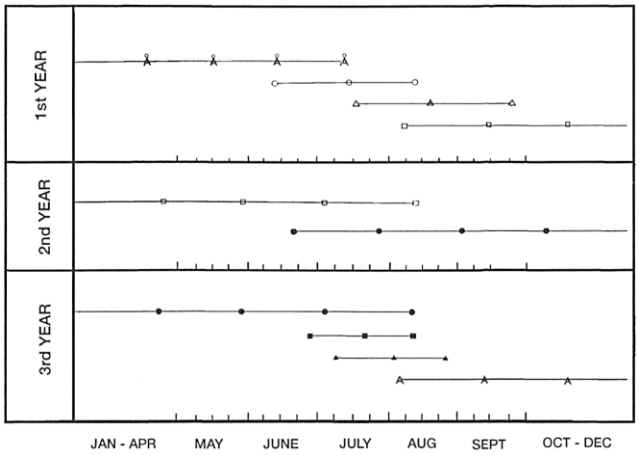
▶ 16.49 White grubs (June beetles) *Figs. 16.49a-e; 16.49T1, T2*

Common June beetle *Phyllophaga anxia* (LeConte)
Other June beetles *Phyllophaga* spp.

White grubs, the larvae of June beetles, spend up to three years in the soil while developing from egg to adult. The common June beetle is one of many *Phyllophaga* species that are native to North America. Adults are referred to as May or June beetles, and the larvae as white grubs. The common June beetle occurs throughout Canada and almost everywhere in the United States. It and several other June beetles may cause economic damage to horticultural crops, but *Phyllophaga anxia* is the species most likely to cause damage in Canada.



16.49T1 White grubs; larvae under sod.



16.49T2 White grubs; life cycle of the common June beetle *Phyllophaga anxia;* Å adult; O egg; A larva, 1st instar; □ larva, 2nd instar; # larva, 3rd instar; ■ prepupa; A pupa; A adult, ten- eral. Reprinted from Lim, Yule and Stewart (1981).

The larvae are best known for their damage to the fibrous roots of lawn grasses (*16.49T1*), but they have a broad host range that includes cereal crops, young evergreens and root crops. Adults feed mainly on the foliage of deciduous trees. Adult feeding also has been observed on leaves of weeds, vegetable crops and flowers.

Damage The grubs may damage potato crops in fields that recently have been in sod. If white grub feeding on potato is severe, damage is indicated by dwarfing or wilting of the plants. White grubs chew deep cavities in potato tubers, making them unmarketable *(16.49a,b).*

Identification Adults of these beetles (family Scarabaeidae) are light to dark brown and measure about 20 mm in length and 11 mm in width *(16.49c).* Eggs are pearly white, about 2.5 mm long and 2.0 mm wide *(16.49d).* The larva is a C-shaped white grub with three pairs of anteriorly located legs and a red-brown head (*16.49d,e*). When mature, larvae are about 30 mm long. Pupae are yellow-white.

Life history All June beetles have similar life histories and habits (*16.49T2*). In most parts of Canada, the insects have a three-year life cycle. The adults are nocturnal. They remain in the soil during the day and on warm evenings fly to nearby trees, where they feed on the foliage. Flight activity begins in May after the accumulation of about 176 degree-days above 5°C, measured from April 1, coinciding with bud break on such trees as trembling aspen *(Populus tremuloides* Michx.). Adult activity may continue for a month on evenings when the temperature is 10°C or higher, during which time mating occurs and egg laying begins. Eggs are laid in the soil at a depth of about 17 cm, usually in grassy areas. The larvae hatch within 30 days and first-instar larvae are commonly found in July. They feed on decaying vegetation, fungi and plant rootlets, usually to a depth of 5 cm or more on plants other than sod; they move from plant to plant as they consume the roots. They molt in mid-August, and the second-instar grubs feed until late fall, then move downward in the soil to overwinter. The second year of the life cycle is known as the "white grub" year. During that time, the second-instar larvae feed and molt into the third (final) instar around the third week of June. Third-instar grubs are most injurious, feeding at a depth of 5 to 25 cm, depending on the soil and the type of food plants. Again, the larvae move downward in the soil to overwinter and, in the third growing season, those that were not fully grown continue to feed before pupating. Most of the grubs form cells in the soil 20 to 25 cm below the surface during July, and pupate. Adults emerge the same year but remain in their earthen cells until the following spring, which is the beetle "flight" year.

Management Information is not available specifically for potato, but the following remarks apply.

Monitoring — Black-light traps fitted with a collection funnel are used to monitor June beetle flights. The traps are set with the rim of the funnel 1.2 m above the ground. Estimates of the density of the soil-inhabiting stages are done by taking sample units of 0.09 m^2 (one square foot) of soil to a depth of 30 cm. The soil samples are examined and larvae counted. No economic threshold is available for vegetable crops. However, tolerance for white grubs in root crops would probably be much lower than in sod or in new forest plantations, such as those in Quebec, where an average of 0.5 grubs per 0.09 m^2 is suggested as a threshold, based on 50 samples at 5 m intervals.

Cultural practices — Planting vegetable crops in recently cultivated sod should be avoided. Summer tillage usually is beneficial, because the grubs are killed by physical injury or exposed to natural enemies and the elements. Tillage should be timed between early May and late June to kill second- and third-year grubs, and from late July to early September to kill first-year grubs. If vegetables are rotated with other crops, then legumes or corn are best in a beetle "flight" year. In other years, oat or barley crops may be used.

Biological control — Natural control agents include insect parasites and predators, nematodes, protozoa, bacteria, fungi, viruses, birds, small mammals and toads. The nematode *Steinemema carpocapsae* (Weiser) (syn. *Neoaplectana car- pocapsae*

Weiser and *Steinemema feltiae* (Filipjev) in earlier literature) is effective against white grubs in pastures. The fungus *Metarhizium anisopliae* (Metsch.) Sorokin has proven effective against white grubs in field studies.

Chemical control — Resistance to organochlorine insecticides was indicated in 1971, when white grubs completely destroyed a crop near Nicolet, Quebec; the field had been treated with chlordane specifically for white grubs.

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(Original by K.P. Lim and J.C. Guppy)

▶ 16.50 Wireworms *Figs. 16.50; 12.21a,b, T1*

Dusky (or European) wireworm *Agriotes obscurus* (L.)
Eastern field wireworm *Limonius agonus* (Say)
Wheat wireworm *Agriotes mancus* (Say)

Wireworms attack potato planted in newly broken land following permanent sod. Several indigenous species are major or minor pests of commercial or domestic potato across Canada. In eastern Canada, the most troublesome species is the eastern field wireworm. The dusky wireworm and the wheat wireworm are the primary pests in central and western Canada. The dusky wireworm, which was accidentally introduced into British Columbia about 1900, has caused major economic damage to potato crops in recent years. It is advisable to identify the species of concern before taking control action, because the host preference and biology of each species varies.

Damage Wireworms tunnel into potato seed-pieces and the developing roots and shoots in the spring. With heavy infestations, a potato crop may be weakened and spotty. Wireworms also feed on the developing tubers *(16.50)* later in the growing season, producing tunnel-like holes 3 mm in diameter and up to 4 cm deep. These holes become lined with periderm, and subsequent tuber growth may be severely distorted.

Identification (see Maize, wireworms, 12.21)

Life history Wireworm larvae in potato fields spend much of the growing season in the upper 10 cm of soil. If mid-summer soil temperatures exceed 27°C, the larvae move downward into cooler zones. Wireworm larvae generally overwinter deep in the soil to avoid freezing.

Management

Monitoring — Annual monitoring is recommended for potato to determine the degree of infestation and the probable success of control methods. If a wireworm problem is suspected, the field should be monitored, using any of the following procedures. The simplest way to determine the presence or absence of wireworms is to follow the cultivator in early spring or fall when soil temperatures are above 10°C and wireworms are near the surface. After cultivating and before planting, baits of whole wheat flour can be used to determine the level of infestation more accurately. This is done by placing about 30 g of flour at a depth of 10 cm in the soil, using an ordinary corn planter or a shovel, and marking each bait station with a stake. For reliable results, 30 to 50 bait stations per hectare should be used. After three or four days, the baits can be uncovered and the wireworms counted. If an average of one or more wire- worms is encountered per station, damage to potato can be severe. Baiting is effective in the warmer spring and summer months, but not if the soil contains a lot of plant residue or if the weather is cold, wet, or very dry. Carrot pieces, buried in the same manner as flour baits, also can be used to sample wireworms (see Carrot, carrot weevil, 6.24).

Cultural practices — Where wireworm population levels are high, careful consideration must be given to proper crop rotation. Wireworms thrive in sod, in red clover and sweet clover, in small grains such as barley and wheat, and in truck crops, all of which should be avoided in rotations with potato. Alfalfa in rotations has the potential to reduce wireworm populations because it is a soil-drying crop and wireworms do poorly under these conditions. Alfalfa can be cropped in infested fields for three or four years and is suitable for rotation with potato as long as appropriate weed control measures are performed. Corn is another option in rotations, because some effective chemicals are registered for use against wireworms on corn. Where wire- worm populations exceed economic thresholds, growers should grow alfalfa or corn in the affected fields for at least three years before again planting potato.

Chemical control — A limited number of chemicals are directed against wireworm larvae. For best control, if potato must be planted in an infested area, granular insecticides may be broadcast and worked into the soil to a depth of 12 to 15 cm, or applied as a band treatment in the furrow with the potato seed-pieces. When soil temperatures are below 10°C, such as in the early spring, efficacy is usually reduced where organic matter is high and chemicals with short residual activity should not be used.

(Original by R.S. Vernon and A.T.S. Wilkinson)

▶ 16.51 Other insect pests *Figs.: see text*

Aster leafhopper *Macrosteles quadrilineatus* (Forbes) (syn. *Macrosteles fascifrons* of authors, not Stål)
Blister beetles
Cutworms
European corn borer *Ostrinia nubilalis* (Hübner)
Grasshoppers
Potato psyllid *Paratrioza cockerelli* (Sulc)
Potato scab gnat *Pnyxia scabiei* (Hopkins)
Seedcorn maggot *Delia platura* (Meigen)
Stalk borer *Papaipema nebris* (Guenée)
Tarnished plant bug *Lygus lineolaris* (Palisot de Beauvois)

Aster leafhopper (see Lettuce, 11.23) The aster leafhopper *(11.23a,b)* can be important on potato because it spreads aster yellows, or purple-top wilt disease. Growers should keep potato and nearby crops free from weeds. If purple-top wilt has been found in the area, application of insecticides may be required.

Blister beetles (family Meloidae) Adults of several species of blister beetles may feed on potato. They vary in size from 0.8 to 2.5 cm and may be black, gray, brown, blue, spotted or striped, often with metallic iridescence. They usually feed in swarms and move about a great deal. Control is rarely necessary.

Cutworms Many species of cutworms may attack potato crops. Some species cut the stems at soil level, others feed on the roots and underground stems. The larvae (*6.25a-c; 18.35a-g*) are gray or brownish, hairless caterpillars, measuring 2 to 5 cm when fully grown. Most live in the top layer of soil during the day and feed at night. (For more information on cutworms, see Tomato, 18.35.)

European corn borer (see Maize, 12.16) The European corn borer (*12.16f-h*) can reduce yields in potato crops by feeding in the stems, but the likelihood of infestation is slight and control measures are rarely necessary. In Prince Edward Island in 1989, a density of 1.16 corn borer larvae per stem in untreated plots reduced total yields of Russet Burbank by 7.5% compared to plots treated with sprays of the microbial insecticide *Bacillus thuringiensis* Berliner.

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Grasshoppers may be a problem, particularly in western Canada. Several species (*12.22a*) may be found in any one locality. They damage mainly the foliage of potato, and they may spread spindle tuber viroid and possibly other pathogens. Disease transmission is purely mechanical and the pathogens can be spread by any species of grasshopper. If the grasshopper forecast indicates that their eggs are plentiful, growers should summerfallow stubble fields. Surface tillage should begin in the fall and be repeated in the spring.

Potato psyllid (family Psyllidae) The potato psyllid has been reported from British Columbia, Alberta, Saskatchewan and Quebec. Feeding by nymphs causes a disorder known as psyllid or potato yellows, which causes the outer leaves to curl and turn light green or yellow; tubers grow slowly and aerial tubers may form in the leaf axils. Growers should apply a recommended insecticide if psyllids become numerous.

Potato scab gnat (family Sciaridae) The potato scab gnat has been found in southern Ontario (confirmed from Barrie on the basis of specimens in the Canadian National Collection), and in eastern Quebec. The larvae of this small fly may attack potato seed-pieces, tubers, and sometimes stems. Eggs are laid in soft spots in tubers, on cut potato seed-pieces and in loose soil. Heavy infestations of seed- pieces may result in weak sprouts and lower yields due to larval feeding on root hairs; superficial pitting reduces the grade of the tubers. This insect is rarely a problem where clean cultivation is practiced in conjunction with good drainage and crop rotation. As a post-harvest pest, potato scab gnat can be important, because its larvae enlarge old wounds and facilitate the entry of other organisms.

Seedcorn maggot (see Bean, 15B.18) The seedcorn maggot is occasionally a pest of potato crops, chiefly in the Maritime provinces, Quebec and Ontario. Damage is greatest in cool, wet seasons. There may be two or more generations a year, depending on the season. The maggot *(12.20b,c)* attacks the seed-piece through unhealed injuries or diseased surfaces. It may spread the bacterium that causes blackleg. Seed treatment may be necessary.

Stalk borer (family Noctuidae) The stalk borer has been found in all provinces from Manitoba eastward, except Newfoundland, and occasionally in Alberta. The larvae tunnel in the stalks, causing the plant to wilt and die. The young larva has a dark brown

or purple band around a cream-colored body. The fully grown larva is grayish or light purple. Clean cultivation, as recommended for the potato stem borer, also controls the stalk borer.

Tarnished plant bug (see Celery, 7.21) The tarnished plant bug *(7.21b,d,e)* feeds by piercing the plant tissues and sucking the sap. On potato, its feeding destroys flowers and may cause the leaves to curl and the new growth to wilt. This insect also can spread spindle tuber viroid. Post-harvest weed sanitation helps reduce levels of the tarnished plant bug by destroying overwintering habitats. When insecticides are required, growers should consult local spray guides.

(Original by L.S. Thompson)

OTHER PESTS

▶ 16.52 Millipedes *Fig. 12.21T1*

Sometimes mistaken for wireworms, millipedes are hard, slender, gray to purple-brown, and worm-like. Their bodies are divided into many segments, most of which have two pairs of legs (*12.21T1*). They enter potato through injuries caused by insects or disease and are especially destructive in cold, wet seasons. They tunnel into the tubers and also may feed on the planted seed. They thrive in land that is heavily manured. Growers should avoid planting potato crops too soon after manuring.

(Original by L.S. Thompson)

▶ 16.53 Slugs *Figs. 11.27a-c*

Several species of slugs (see Lettuce, 11.27) occasionally damage potato tubers. They also injure potato plants by eating the stalks and foliage. To prevent damage, it may be enough to avoid long grass and remove plant residue, old sacks and boxes on the ground, and other locations where slugs may hide in the daytime.

(Original by L.S. Thompson)

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17 Rhubarb

BACTERIAL DISEASES

▶ 17.1 Crown gall *Fig. 17.1*

Agrobacterium tumefaciens (E.F. Smith & Towns.) Conn

Crown gall is a minor disease of rhubarb in Canada. The pathogen has a wide host range that includes several vegetable crops (see Carrot, crown gall, 6.3).

Symptoms Galls appear on the new growth of roots and crowns as firm white masses that become enveloped in a heavily mottled covering as the season progresses (*17.1).* By late summer, the galls usually disintegrate. There appears to be little or no detrimental effect upon productivity of the plants.

Causal agent (see Carrot, crown gall, 6.3)

Disease cycle (see Carrot, crown gall, 6.3)

Management *Cultural practices* — Diseased plants should be removed as soon as they are noticed. (For other control measures, see Carrot, crown gall, 6.3.)

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(Original by R.J. Howard and D.J. Ormrod)

▶ 17.2 Red leaf (bacterial soft rot) *Figs. 17.2a,b*

Erwinia rhapontici (Millard) Burkholder
(syn. *Bacterium rhapontici* Millard)

Red leaf is one of the most destructive diseases of rhubarb in Canada and has destroyed up to 50% of the plants in some fields. This disease was often referred to as crown rot until the mid 1950s, but since then it has been called red leaf in Canada and bacterial soft rot in other countries. The pathogen also has been reported on wheat and on pea seed in some areas of Canada.

Symptoms *Erwinia rhapontici* causes a crown rot disease on rhubarb. The main symptoms are decay of the terminal bud, a soft, chocolate-brown rotting of the pith, and the formation of a cavity within the crown *(17.2a).* Spindly side shoots may grow out but usually rot off. During wet weather, the bases of older leaves may also be affected. Dull red leaves are usually present on diseased plants *(17.2b).* Turnip mosaic virus (see Viral diseases, 17.9) and various fungi (see crown rot, 17.3) can cause symptoms that closely resemble those of red leaf.

Causal agent *Erwinia rhapontici* is a Gram-negative, non-spore-forming, non-capsulate rod, 0.5 to 0.8 by 1.2 to 1.5 µm. The cells are motile by several, usually about five, peritrichous flagella. This bacterium is a facultative anaerobe.

The pathogen can be isolated by plating pieces of diseased rhubarb tissue onto general-purpose bacteriological media. On nutrient agar, colonies are round, entire, smooth, glistening, butyrous and translucent white. Some isolates may produce a pink pigment that diffuses into the medium.

Disease cycle Relatively little is known about the dispersal and survival of the red leaf pathogen. Transplanting of infected crowns and foliage- and root-feeding insects moving from diseased to healthy plants are the most likely methods by which the disease spreads.

Management

Cultural practices — Only disease-free crowns should be used for planting and replacement. Plants with symptoms of red leaf or crown rot should be dug up and destroyed. New crowns should not be replanted in the same area from which diseased ones were removed. Aphids and other foliage-feeding insects should not be allowed to build up in rhubarb plantings.

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(Original by R.J. Howard and D.J. Ormrod)

FUNGAL DISEASES

▶ 17.3 Crown rot

Phytophthora spp.
Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Crown rot is occcasionally a problem in rhubarb, especially under wet soil conditions. *Phytophthora*, *Pythium* and *Rhizoctonia* species occur in many field and garden soils and are pathogens of several vegetable crops.

Symptoms Infection usually occurs at the base of the stalks or a little below the soil surface. Stalk lesions are brown and sunken. Rotted crown tissue is usually firm and brown, but may turn mushy if bacterial soft rot ensues. Leaves wilt and die, usually a few at a time, and badly infected plants eventually die. Symptoms of crown rot can resemble those of red leaf disease.

Causal agents (For detailed descriptions of *Phytophthora* spp., *Pythium* spp. and *Rhizoctonia solani*, see the Bean, Beet, Carrot, Cucurbits, Crucifers and Potato chapters.)

Disease cycle (see Bean, root rots, 15B.4; Carrot, crown rot, 6.11.)

Management

Cultural practices — Growers should establish rhubarb plantings in well-drained, fertile, weed- free fields. Rhubarb should not be planted immediately after other vegetable crops in the rotation. If possible, allow at least two to three years between these crops and rhubarb. The practices for managing red leaf can also be applied to crown rot.

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(Original by R.J. Howard)

▶ 17.4 Downy mildew

Peronospora rumicis Corda

Downy mildew is most destructive on seedlings grown in cold frames, but it can affect rhubarb plants at any stage of growth. Rhubarb is the only reported host of *Peronospora rumicis.*

Symptoms Large brown lesions are formed on the leaves, and the lower surfaces often are covered with a violet to white fungal growth. When the invasion is extensive, the leaf dies. Small spots often tear away from the healthy tissue, leaving a ragged-appearing leaf.

Causal agent The sporangia of *Peronospora rumicis* are borne at the tips of dichotomously branched sporangiophores. Sporangia are ovoid, subhyaline, 16 to 18 by 25 to 34 µm, and germinate by germ tubes.

Disease cycle The pathogen requires cool, wet weather for rapid reproduction. The sporangia remain alive only for a day or so when the temperatures are moderate and the relative humidity low.

Management

Cultural practices — Downy mildew can be managed by using disease-free propagation stock and by not planting into fields where rhubarb has been grown within the previous three years.

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(Original by R.J. Howard and D.J. Ormrod)

▶ 17.5 Gray mold

Botrytis cinerea Pers.:Fr.
(telemorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold is occasionally damaging in rhubarb fields in high rainfall areas, but is more likely to be a problem in crops forced indoors. It is the most destructive post-harvest disease of rhubarb, especially when stalks are packed and shipped with the leaves still attached. The disease occurs in all of the major rhubarb-producing areas in Canada. The gray mold pathogen has a wide host range that includes many vegetable crops (see Lettuce, gray mold, 11.10; Asparagus, botrytis blight, 4.1).

Symptoms Gray mold may appear on old leaves and injured stalks under humid conditions in the field. Other stalks can become contaminated during harvest and packing. Red spots or water-soaked brown areas appear on the stalks and masses of dusty gray spores can form on these lesions. A semi-watery decay often follows, especially under non-refrigerated storage conditions.

Causal agent (see Lettuce, gray mold, 11.10)

Disease cycle (see Lettuce, gray mold, 11.10)

Management

Cultural practices — Growers should remove the leaves from rhubarb stalks before they are packed and shipped. Harvested stalks should be kept refrigerated. (For other management strategies, see Lettuce, gray mold, 11.10.)

Chemical control — Registered fungicides are available for use on rhubarb crops forced indoors.

(Original by D.J. Ormrod and R.J. Howard)

▶ 17.6 Leaf spots *Figs. 17.6a,b*

Ascochyta leaf spot

Ascochyta rhei (Ellis & Everh.) Ellis & Everh.
(syn. *Phyllosticta rhei* Ellis & Everh.)

Ramularia leaf spot

Ramularia rhei Allesch.

Ascochyta and ramularia leaf spots are common on field rhubarb. Losses are usually minor, except under prolonged wet growing conditions. Rhubarb is the main host of these pathogens.

Symptoms The first indications of ascochyta leaf spot are numerous, small, yellow-green spots in the upper leaf surfaces. When these lesions unite, as they often do, the leaf has the appearance of mosaic mottling. In less than a week, the invaded tissue

usually turns brown and dies, resulting in circular to angular spots that vary in size from 1 to 15 by 1 to 3 mm. These spots have white centers surrounded by a wide red margin that is bordered by a gray-green zone. In some of the smaller spots, only the red color may be present. Fruiting bodies of the pathogen are rarely visible in the spots, being sunk so deeply in the rhubarb tissue that only the openings are flush with the leaf surface. When the affected tissue dies, it may drop out, leaving large ragged holes in the leaves.

Leaf infection by *Ramularia rhei* first appears as small red dots. These gradually enlarge to form more or less circular lesions 1 cm or more in diameter *(17.6a).* Large spots are white to tan with purplish halos. Stalk infections, which occur later, first appear as small spots that elongate as the stalks grow. The larger ones become tan-colored, sunken lesions up to 1 cm long *(17.6b).* A white accumulation of conidia may be present in the center of spots on both leaves and stalks.

Causal agent *Ascochyta rhei* has globular, black pycnidia filled with short, hyaline, cylindrical conidia that are slightly constricted near the center. A small percentage of the spores have cross walls near the center. *Ramularia rhei* has hyaline conidia that are non- to three-septate, and 2 to 3 by 7 to 35 µm.

Disease cycle Both pathogens produce spores that are dispersed by splashing water and wind. New infections can result in visible lesions within 10 to 14 days. When old infected leaves or stalks drop to the ground, mycelial masses or fruiting bodies are formed and can remain alive over winter. Leaf spot fungi also can be spread in infected root stocks that are used for propagation.

Management

Cultural practices — As little crop residue as possible should be left on the soil surface after harvest. In gardens, the leaves should be gathered and composted or destroyed as soon as the first frost has killed them. In commercial fields, growers should thoroughly incorporate the crop residues between the rows after harvest. During the harvest of stalks in the spring, those with spotted leaves should be taken first, as much of the diseased material can be removed by this method. Rhubarb plants should be fertilized as soon as growth starts in the spring and another application should be made as soon as the harvest is completed to encourage strong, rapid regrowth.

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(Original by R.J. Howard and D.J. Ormrod)

▶ 17.7 Powdery mildew

Erysiphe polygoni DC.

Powdery mildew rarely affects rhubarb seriously. The pathogen can attack wild and cultivated members of the buckwheat family (Polygonaceae) (see Crucifers, powdery mildew, 8.12).

Symptoms The characteristic symptom of this disease is diffuse, dusty white lesions on the surfaces of leaves and stalks.

(For a description of the causal agent and a discussion of the disease cycle, epidemiology and control of powdery mildew, see Crucifers, powdery mildew, 8.12.)

(Original by R.J. Howard and D.J. Ormrod)

▶ 17.8 Rust

Puccinia phragmitis (Schumach.) Körn.

Rust is a minor disease of rhubarb in Canada. Rhubarb is the only vegetable crop attacked by *P. phragmitis.*

Symptoms Rust produces large crimson spots on rhubarb leaves. The central part of the spot is crowded with the cluster-cups (aecia) of the fungus. The edges of the cups become torn, producing a fringed border.

Disease cycle The pathogen is a long-cycle, heteroecious rust. The aecia and pycnia appear on rhubarb and other species of *Rheum* and *Rumex,* while the uredinia and telia occur on reed grass, *Phragmites australis* (Cav.) Trin. The aecia appear on the lower side of the leaf surrounding the pycnia. The fungus overwinters as teliospores, which upon germination produce basidiospores that can infect the leaves of rhubarb.

Management Control measures are usually not necessary.

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(Original by R.J. Howard and D.J. Ormrod)

VIRAL DISEASES

▶ 17.9 Miscellaneous viral diseases *Fig. 17.9*

Arabis mosaic virus
Cherry leaf roll virus
Cucumber mosaic virus
Strawberry latent ringspot virus
Turnip mosaic virus

Viral diseases occasionally have caused serious damage to rhubarb plantings in British Columbia. All of the causal viruses have wide host ranges that include several species of vegetable crops.

Symptoms Mottling (17.9) and ring spotting of leaves, with or without stunting, is frequently observed in individual rhubarb plants or in patches within a crop. These symptoms may be due to infection by one or more viruses. In British Columbia, turnip mosaic virus is the one most commonly found. This virus can cause symptoms that mimic those of red leaf.

Causal agents (For a description of arabis mosaic virus, see Herbs and spices, 10.12; for cucumber mosaic virus, see Greenhouse cucumber, 22.20; for turnip mosaic virus, see Crucifers, 8.16.)

Cherry leaf roll virus has isometric particles, which measure about 28 nm in diameter. It is readily transmitted by sap inoculation and seed, and some strains are pollen-transmitted.

Strawberry latent ringspot virus is an RNA virus with isometric particles about 30 nm in diameter. It is sap- and nematode-transmissible.

Disease cycle Viruses can be introduced in infected crowns or seed and may subsequently be spread by aphid or nematode vectors. Several genera of plant parasitic nematodes have been found in rhubarb fields in Canada; some nematodes, such as *Xiphinema* spp., are known vectors of several of the viruses infecting rhubarb. These pests may be present in soil attached to crowns and farm equipment and thus may be responsible for virus spread, both within a field and from field to field.

Management

Cultural practices — Growers should plant rhubarb in fields that have not grown this crop within the previous two to three years and that do not have a detectable population of plant parasitic nematodes, particularly potential virus vectors. Only disease- and nematode- free rhubarb should be used for planting.

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(Original by R.J. Howard and D.J. Ormrod)

NEMATODE PESTS

▶ 17.10 Northern root-knot nematode *Fig. 7.15b*

Meloidogyne hapla Chitwood

Symptoms With heavy infestations, affected plants wilt, turn light green and progressively yellow. Roots show numerous small spherical swellings from which adventitious rootlets grow, producing increased branching that which can result in a bushy appearance. For a complete description and management strategies, see Carrot. 6.20; see also Management of nematode pests, 3.12.

▶ 17.11 Pin nematodes

Paratylenchus spp.

Symptoms These ectoparasitic nematodes feed on root tissues, such as the epidermis and cortex or, if their stylet is long enough, the vascular tissue. They never enter the roots of plants. At numbers as high as 5000 or more per kilogram of soil, pin nematodes have reduced yields of rhubarb in Ontario. See Nematodes, 2.3; see also Management of nematode pests, 3.12.

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▶ 17.12 Sugarbeet cyst nematode *Fig. 5.14b*

Heterodera schachtii Schmidt

Symptoms are most noticeable in patches where nematode densities are high. Infected plants are stunted and outer leaves wilt, yellow prematurely and die. Lateral root development is excessive, giving a whiskered appearance to the tap root. In summer, pin-head sized, white or brown cysts can be seen on washed roots, particularly in the root axils. See Beet, 5.14; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 17.13 Miscellaneous insect pests *Figs.: see text*

Black bean aphid *Aphis fabae* Scopoli
European earwig *Forficula auricularia* L.
Potato stem borer *Hydraecia micacea* (Esper)
Rhubarb curculio *Lixus concavus* Say

The black bean aphid (see Potato, 16.43) (*16.43T1*) has been reported on rhubarb plantings in British Columbia, where it has been suspected but not proven to be a vector of turnip mosaic virus. The potato stem borer (see Potato, 16.47) *(16.47a,b)* and the rhubarb curculio affect rhubarb petioles and may be minor pests in Ontario and Quebec. Earwigs (see Crucifers, 8.43) *(8.43b,d)* may eat holes in rhubarb leaves, which can be important during establishment of young plants.

OTHER PESTS

▶ 17.14 Slugs *Figs. 11.27a-c*

Slugs have caused significant direct injury to rhubarb in southern coastal British Columbia, according to D.J. Ormrod. For information about slugs, see Crucifers, 8.49, and Lettuce, 11.27.

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18 Tomato, eggplant, pepper

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BACTERIAL DISEASES

18.1 Bacterial canker *Figs. 18.1 a-c*

Clavibacter michiganensis subsp. *michiganensis* (E.F. Smith) Davis *et al.*
(syn. *Corynebacterium michiganense* (E.F. Smith) Jensen)

Bacterial canker is encountered in the field and greenhouse (see Greenhouse tomato, bacterial canker, 25.1), occasionally causing considerable damage. Tomato is the major crop affected by this disease.

Symptoms Bacterial canker symptoms in field tomato vary dramatically from those observed in the greenhouse (see Greenhouse tomato, bacterial canker, 25.1). Primary or systemic infections from seed or clipped tomato seedlings cause the greatest level of plant loss, whereas secondary infection causes fruit lesions and a foliar "firing" or blight phase observed later in the season. Transplants may not express symptoms until six to eight weeks after infection. Initial symptom expression is accelerated by environmental stress. Diseased plants are stunted, vascular tissues discolor, open stem cankers become visible, and considerable plant mortality can occur. Transplants brought into Canada from the southern United States may show dark brown necrotic tissue at the site of clipping wounds. These wounds often extend deep into the stem tissue and may act as infection sites. A characteristic symptom of canker in field tomato is brown to black leaf margins with a thin, yellow, chlorotic band *(18.1a).* A necrotic peppering can be observed under the calyx scar by detaching the young tomato fruit. Stem nodes often appear puffy white at spots where the bacteria and its toxin accumulate. When stems are split lengthwise, a thin, light reddish-brown area usually can be seen within the vascular tissue *(18.1b).* This discoloration is most noticeable just above the soil line. Movement of bacteria into the fruit produces internal breakdown, with yellow to brown cavities extending into the seed cavity and eventually to the seeds. Positive identification of canker can be made in the field when "bird's-eye" spots are observed on the fruit *(18.1c).* These are relatively small, 2 mm diameter spots with minute light brown centers generally surrounded by a characteristic snow-white halo.

Early symptoms of bacterial spot on the fruit include a whitish, mottled lesion *(18.1c),* which then turns a characteristic corky black. However, bacterial canker does not produce the black spot phase on the leaves that is characteristic of the bacterial spot disease.

Bacterial canker can be misdiagnosed as bacterial wilt, which can have similar foliar wilt symptoms. The vascular browning, however, is more intense in bacterial wilt, moving into the pith and progressing further down into the stem below ground.

Causal agent (see Greenhouse tomato, bacterial canker, 25.1)

Disease cycle (see Greenhouse tomato, bacterial canker) The pathogen may survive in non-decomposed tomato crop residues in the field and on seed for up to five years. Infested seed is the main means of long-distance dispersal. The bacterium is easily spread within fields by splashing water, handling of infected plants and other cultural operations. It infects the host through wounds on leaves, broken trichomes or directly through leaf edge hydathodes and stomata. When tomato transplants are grown under field conditions in the southern United States, the mechanical clipping operation used to produce a shorter, sturdier plant often spreads the bacteria throughout the production beds. This results in many symptomless infected plants being shipped north. Within four to five weeks in Canadian fields, these plants begin to show canker symptoms. Increasing levels of nitrate nitrogen and decreasing levels of calcium have been shown to increase disease severity. Warm (24 to 32°C), wet weather favors pathogen spread and disease development.

Management

Cultural practices — The use of disease- free seed is the most effective management strategy. Proper fermentation during seed extraction is important because this process will control seed-borne bacterial pathogens. Acids, bleach or hot water can be used to disinfest seed of questionable status (see Greenhouse tomato, bacterial canker). The increasing use of locally grown greenhouse transplants has led to a reduction in canker incidence compared to the use of imported material.

Resistant cultivars — Sources of genetic resistance are being used in several breeding programs.

Chemical control — Copper-based bactericides have not been useful in controlling canker in field tomato.

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(Original by R.E. Pitblado and L.M. Tarder)

▶ 18.2 Bacterial soft rot *Fig. 18.2*

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*

Bacterial soft rot can cause the complete collapse of fruit, but more commonly it reduces fruit marketability by causing a slimy rot. This disease occurs more frequently on pepper and eggplant than on tomato. The pathogen has a wide host range that includes many vegetable crops (see Potato, bacterial soft rot, 16.2).

Symptoms Stem and fruit wounds are initial sites of infection. Rot progresses into the stem and, in time, invades the fruit. The entire fruit fills with a watery, soft, slimy mass that is kept intact by the thin outer skin *(18.2).* When the skin breaks, the fruit collapses and dries into a shrivelled wrinkled mass. In pepper, the fruit stem (peduncle) initially discolors and several small dark lesions develop that later turn slimy See also Greenhouse tomato, bacterial stem rot, 25.3.

Causal agent (see Potato, bacterial soft rot, 16.2)

Disease cycle Soft rot bacteria are native inhabitants of field soils. Splashing rain carries them from the soil to the foliage where, under moist conditions, a rapid build-up of bacterial populations can occur. The bacteria can enter fruit through cuts, breaks, insect damage and abrasion. Frequently, a high incidence of soft rot is associated with harvesting during rainy periods and with washing the fruit after harvest. Moisture increases the susceptibility of fruit to the bacteria, which can enter the newly broken fruit stems at harvest as well as through other wounds.

Management

Cultural practices — Bacterial populations in soil often are high after crops of potato or cabbage; therefore, growers should avoid planting eggplant and pepper after those crops, rotating instead with crops such as bean, corn and soybean. It is best to harvest during dry weather and to minimize fruit injury at harvest. Fruit should be kept cool and dry during packing and storage. Prevention of insect wounds, such as those caused by the European corn borer, also is important.

Chemical control — Chlorination may help to eliminate soft rot bacteria from wash water and it reduces the risk of infection during washing. However, it does not arrest soft rot development in fruit infected before washing. Growers and packers should consult the Health Protection Branch, Health Canada, for guidelines on chlorinating vegetable wash water.

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(Original by R.E. Pitblado)

▶ 18.3 Bacterial speck *Figs. 18.3a,b*

Pseudomonas syringae pv. *tomato* (Okabe) Young *et al.*

This disease affects both greenhouse tomato and field tomato and can be particularly serious in wholepack and fresh-market crops. Tomato is the only known horticultural crop affected by bacterial speck.

Symptoms Symptoms may occur on the leaves, stems and fruit. On the leaves, tiny black specks, generally no more than 2 mm in diameter, appear first and are soon surrounded by a yellowish halo *(18.3a).* When numerous, the chlorotic areas can merge, giving the appearance of early blight. Affected leaflets become distorted, shrivel and fall off, thereby exposing the fruit to sunscald. Small black specks can also be seen on the fruit *(18.3b).* On green fruit, they are sometimes slightly raised, rough to the touch and surrounded by a narrow, green to yellow halo. Only green fruit is infected; the pH of skin tissue on green fruit is about 6.3, whereas on ripe fruit it is 5.2, which is too low to support bacterial growth. Once green fruit is infected, black lesions remain and are observed on red fruit late in the season. The tissue around the specks is slow to ripen and remains green longer. The same symptoms can also develop on the stems, but this is less characteristic. Fruit affected by bacterial speck is not acceptable for fresh market because of its appearance, nor for wholepack processing because it peels poorly, resulting in visible remains of specks and skin pieces (“tags”) on canned tomatoes as well as in the juice. This extraneous material lowers product quality.

Causal agent *Pseudomonas syringae* pv. *tomato* is an aerobic. Gram-negative, rod-shaped bacterium, measuring approximately 0.83 by 2.2 pm, with one to three flagella. Colonies produce a fluorescent green pigment on King's B medium in culture. This bacterium can be distinguished from other fluorescent pseudomonads by biochemical and physiological tests. It is negative for oxidase reaction and arginine dihydrolase. It is positive for levan production and ß-glucosidase activity. It utilizes D(-)tartrate but not erythritol or DL-lactate as sole carbon sources. It produces a hypersensitive reaction when infiltrated into tobacco at high concentrations (10^8 cfu/mL).

Disease cycle The pathogen is disseminated on seed, transplants and through infested crop residues. In cool (18 to 24°C), rainy weather, the bacteria multiply rapidly in infected plants. Through splashing water and cultural operations, such as picking, weeding, spraying and pruning, the bacteria are spread from infected to healthy plants. They can persist in the soil on infected plant material until it completely decomposes. The pathogen can also survive on seed. The main source of contamination in Canada, however, is from transplants imported from the United States. Transplants often appear healthy upon arrival, but harbor *Pseudomonas* bacteria that can develop later under cool, wet conditions.

Management

Cultural practices — The most important management strategy is to use disease-free seed. Proper fermentation and hot water, bleach or acid treatments all serve to disinfest contaminated seed (see Greenhouse tomato, bacterial canker, 25.1). In the case of transplants, it is important to purchase disease-free seedlings of known origin and to avoid clipping the seedlings to minimize secondary spread of the bacteria. Strict water management practices in local greenhouse-grown transplants significantly reduce the spread of the disease, delaying its appearance in the field. Growers should avoid working in fields when foliage is wet. If overhead irrigation is necessary, it should begin early in the day so that foliage can dry before evening. A two-year crop rotation is suggested to allow infested residues to decompose before tomato is planted again.

Resistant cultivars — Along with the discovery of the single, dominant Pto-gene, found in the breeding line ONT 7710, other sources of genetic resistance have been incorporated into many Canadian processing tomato cultivars.

Chemical control — Protective sprays of bactericides, such as copper compounds, are recommended only at the transplant seedling stage. Once tomato plants are transplanted in the field, effective control of bacterial speck is difficult to achieve using chemicals.

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(Original by R.E. Pitblado and L.M. Tartier)

▶ 18.4 Bacterial spot *Figs. 18.4a-f*

Xanthomonas campestris pv. *vesicatoria* (Doidge) Dye

Bacterial spot is a special problem for processors of wholepack tomatoes because infected fruit is difficult to peel. In field pepper, it is usually the most common and damaging disease. Tomato and pepper are the main crops affected by this disease.

Symptoms Symptoms can appear on the leaves, stems and fruit. On the leaves and stems of tomato, the first symptoms are black, circular spots, about 1 mm in diameter, surrounded by a yellow halo. These spots may be indistinguishable from those caused by bacterial speck. When the spots are numerous, they cause the foliage to dry up and are often misidentified as early blight. On the fruit, the first symptoms are small, dark brown to black raised spots *(18.4a)*, which are sometimes surrounded by a greasy-looking white halo that is often mistaken for bacterial canker bird's-eye spotting. The spots increase to 4 to 5 mm in diameter, which is two to three times larger than those of bacterial speck, and become scabby. Spots eventually develop a corky appearance and their centers turn gray or brown (*18.4b,c*).

In pepper, foliar symptoms of bacterial spot are slightly different from those in tomato. Lesions turn dark brown to black with a pale tan central area (*18.4d,e*), giving the foliage a shot-hole appearance. Advanced symptoms show an irregular blighting throughout the leaf canopy. When spots are numerous, entire leaves may drop off. Fruit spots (*18.4f)* have a brown to black, raised, wart-like appearance similar to that observed on tomato. Secondary fruit rots may develop around the spots during damp weather.

Causal agent *Xanthomonas campestris* pv. *vesicatoria* is an aerobic, Gram-negative, rod-shaped bacterium measuring approximately 0.85 by 2.2 µm with one polar flagellum. It exhibits slow, viscous growth on nutrient or yeast extract-dextrose-calcium carbonate agar and has wet, shining yellow colonies. The bacterium produces acid but no gas from arabinose, glucose,

sucrose, galactate, trehalose, cellobiose and fructose. Starch hydrolysis is variable. Pepper and tomato pathotypes of the pathogen have been described.

Disease cycle The bacterial spot pathogen has a higher optimum temperature (24 to 30°C) than does the bacterium that causes speck. Both organisms are spread rapidly from plant to plant by splashing water and by mechanical means. *Xanthomonas* bacteria survive in the soil on non-decom- posed plant residues and on seed from infected plants. The main source of infestation in Canada, however, is transplants imported from the United States.

Management

Cultural practices — Prevention of bacterial spot requires the use of the same methods of seed disinfestation as those recommended for bacterial canker (see Greenhouse tomato, bacterial canker, 25.1). Lowering the relative humidity, coupled with strict water management in locally grown plug-transplant greenhouses, retards disease development. Growers should avoid following pepper with tomato crops and *vice versa*. In addition to reducing contamination from one crop to another, tomato and pepper should not be grown in the same greenhouse.

Resistant cultivars — A low degree of field resistance has been identified, but high levels of resistance have not been incorporated into commercial cultivars.

Chemical control — Copper-based bactericides are available, but their effectiveness is limited because they kill only those bacteria on leaf surfaces. These products have given some control in fresh-market tomato crops, but spraying is not recommended for processing crops production. Spray intervals beyond four days are ineffective. Copper-resistant strains of *Xanthomonas campestris* have been detected in some areas.

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(Original by L.M. Tartier and R.E. Pitblado)

▶ 18.5 Bacterial wilt (brown rot, southern bacterial wilt)

Pseudomonas solanacearum (E.F. Smith) E.F. Smith

Bacterial wilt affects chiefly tomato crops in the southern United States, but some Canadian growers have experienced losses after using infected imported transplants. This disease can also affect pepper and eggplant. The pathogen is capable of attacking more than 200 species of plants in 33 families.

Symptoms Canadian growers usually notice leaf wilting within five weeks after transplanting plants from the southern United States, with the eventual collapse and death of infected plants. Field symptoms can be confused with those of bacterial canker, but a distinguishing symptom is the extensive vascular and internal discoloration in the lower stem associated with bacterial wilt. Infected plants exhibit dark vascular browning that extends into the cortical or pith regions and sometimes deep into the below-ground part of the stem. Most infected transplants die within two weeks, beyond which no further loss occurs. When infected stems or roots are cut crosswise and squeezed firmly, a gray to yellowish ooze appears. This disease can be distinguished from other wilts by placing an infected stem section in a glass of water. If bacterial wilt is present, a milky stream flows from the cut surface within five minutes.

Causal agent *Pseudomonas solanacearum* is an aerobic, Gram-negative, rod-shaped bacterium measuring approximately 0.6 by 1.7 pm with one to four polar flagella. All strains except those from banana and other musaceous hosts produce a dark brown diffusable pigment on a variety of agar media containing tyrosine. The pathogen is catalase- and oxidasepositive and forms nitrites from nitrates. It is a nonfluorescent pseudomonad. The pathogen is negative for levan production, starch hydrolysis, indole production, hydrogen sulfide and aesculin hydrolysis.

Disease cycle *Pseudomonas solanacearum* occurs throughout the world in areas with warm climates. Infection and disease development are favored by high temperatures (optimum 30 to 35°C) and high moisture. It survives in infested soil and crop residues, is seed-borne and can be found in numerous weed hosts. The bacteria do not survive in the field in Canada, but may persist in greenhouses. The disease quickly spreads within the cortex and pith of an infected plant, eventually causing death. In Canada, bacterial wilt has not been observed to spread in the field beyond initially infected plants.

Management

Cultural practices — Since the disease does not appear to spread in the field, it is self-eliminating and no control measures are warranted. Growers should use disease-free transplants and preferably those grown in local greenhouses.

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(Original by R.E. Pitblado)

FUNGAL DISEASES

▶ 18.6 Anthracnose *Figs. 18.6a-c*

Colletotrichum coccodes (Wahr.) Hughes
(syn. *Colletotrichum atramentarium* (Berk. & Broome) Traub.)

Anthracnose is a major disease of ripe tomato fruit and can be especially serious for processors because it affects the taste of wholepack tomatoes, tomato juice and concentrate. Machine-harvested tomatoes are at the greatest risk because ripe fruit is held in the field longer for once-over mechanical harvesting. This disease also affects eggplant, pepper and potato (see Potato, black dot, 16.6).

Symptoms The most noticeable symptoms occur on ripe fruit. Small, circular, sunken spots appear at first, gradually expanding to about 20 mm in diameter (*18.6a,b*). Lesions formed by *Colletotrichum coccodes* are usually characterized by numerous, submerged, black microsclerotia which often form in concentric rings. When spots are numerous, they can merge and affect large areas of the fruit. Under humid conditions, the centers of these spots darken due to the development of hairs (setae) on the fruiting bodies of the pathogen *(18.6c).* Pink gelatinous masses of conidia ooze from these sunken lesions, which often crack, allowing secondary organisms to invade and cause soft rot. Green fruit also can be infected but the symptoms may not appear until just before or more usually after harvest. Thus, this latent infection can be more serious than its appearance at harvest would indicate. Since infection results from spores present on plant residues, the side of the fruit touching the soil is more commonly infected and develops the greatest number of lesions. Tiny spots also may appear on the stems and leaves. Such spots are usually overlooked, but they often act as the initial source of inoculum that will infect the fruit once it has ripened.

Causal agent *Colletotrichum coccodes* has cylindrical-allantoid, one- celled, biguttulate hyaline conidia with rounded ends that measure 4 by 16 to 24 µm. In mass the conidia within the fruiting bodies (acervuli) appear pinkish. Black, straight or curved, septate setae, 65 to 112 µm long, are usually, but not invariably, present on the acervuli. Numerous, small, black microsclerotia about 0.5 mm in diameter form both on fruit and in culture. The fungus is readily isolated from diseased fruit on potato-dextrose agar.

Disease cycle Many common weeds and crop plants serve as symptomless hosts for *C. coccodes.* Weed tissue containing sclerotia could act as a source of primary inoculum for subsequent seasons or as a source of secondary inoculum during the current growing season. The fungus survives as microsclerotia from season to season on seed and in plant debris from infected crops. Microsclerotia can produce both hyphae and conidia. Soil- borne microsclerotia and conidia can be splashed onto foliage and fruit where appressoria are formed, with the infection peg penetrating the fruit cuticle. The pathogen penetrates green fruit and stems and remains latent until these tissues begin to mature. Leaf infection and some fruit infection takes place by at least mid-July in southern Ontario, depending on the weather. Conditions that favor plant infection are temperatures from 10 to 30°C (with an optimum of 20 to 24°C), together with free moisture. Splashing water and extended periods of leaf and fruit wetness encourage the spread and development of anthracnose. The longer the period of free moisture on tomato fruit the greater the disease severity. The fungus can also enter through wounds caused by sand blasting or other diseases, such as early blight.

Management

Cultural practices — Growers should use disease-free, fungicide-treated seed or seed that has been disinfested (see bacterial canker, 18.1). Infested crop residues may take three years to decompose completely. Keep fields free of weeds. Uncontrolled weed populations can support increased inoculum levels between rotations. Tomato crops should be rotated with non-solanaceous crops. Field sorting will lower the percentage of defects delivered to processors.

Resistant cultivars — Sources of resistance exist and promising new tomato cultivars are being evaluated but many are small-fruited and late maturing.

Chemical control — Registered fungicides are available. A weather-timed fungicide spray program called TOM- CAST is available to assist growers in scheduling sprays (see early blight, 18.8).

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(Original by L.M. Tarder and R.E. Pitblado)

▶ 18.7 Damping-off *Fig. 25.7*

Phytophthora spp.
Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Damping-off (see Greenhouse tomato, damping-off, 25.7) can be a serious problem in newly seeded or transplanted tomato, pepper and eggplant crops. The effect of this disease has been significantly reduced in commercial production with the introduction of plug transplants. With proper water management and the use of disease-free, soilless mixtures, damping-off has almost been eliminated as a production concern. However, the collar rot phase of damping- off remains a problem in hydroponically grown transplants.

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(Original by R.E. Pitblado)

▶ 18.8 Early blight (target spot) alternaria fruit rot *Figs. 18.8a-g; 25.9a,b*

Alternaria solani Sorauer
Alternaria alternata (Fr.:Fr.) Keissl.

Early blight can cause serious defoliation of tomato crops. It is often associated with septoria leaf spot, and these two fungal diseases, either separately or together, are responsible for most of the defoliation caused by diseases in field tomato crops in Canada. The pathogen also infects potato (see Potato, early blight, 16.8) and solanaceous weeds. Pepper and eggplant are rarely affected. See also Greenhouse tomato, early blight, 25.9.

Symptoms Early blight can affect all above-ground plant parts throughout the growing season. In unusual cases, a collar rot can girdle the base of the stem at the time of emergence. In greenhouse plug production of seedlings, a stem blight phase with black, elongate lesions on the stems is often noted under hot, moist growing conditions. Early blight is more commonly known as a leaf spotting or foliar blight disease *(18.8a).* Initially, the small circular spots have characteristic dark concentric rings (target spots) *(18.8b, 25.9a).* The spots later become irregular in shape, and affect both the central portion and the edges of the tomato leaf. Early blight lesions produce a characteristic yellow blighted area around the dead tissue similar to the spring symptoms of bacterial speck and bacterial spot under especially dry soil conditions. Spots first appear on the older leaves, progressing upward to the new growth. This disease may be confused with septoria leaf spot *(25.9b),* the lesions of which also produce a black wavy appearance somewhat similar to the concentric rings of early blight. However, septoria lesions have a lighter tan center with pycnidia therein, giving a black-dot appearance under magnification. Under conditions of extended leaf wetness and high temperatures, tomato plants can be completely defoliated by early blight, thereby exposing fruit to sunscald and anthracnose while reducing yield. Brown concentric circles are also found on stems and flower parts. On fruit, symptoms are common when extended wet periods occur at harvest. A blackened area, similar in appearance to blossom- end rot, can develop at the stem end of the fruit *(18.8c).* Dark, leathery sunken areas *(18.8d-g)* occasionally form around wounds or fruit cracks.

Causal agent (see Potato, early blight, 16.8) In addition to *Alternaria solani, A. alternata* is often isolated from typical early blight lesions, especially on fruit. Conidia of the two species differ in morphology and length (see Greenhouse tomato, early blight, 25.9).

Disease cycle (see Potato, early blight) The pathogen survives between crops mainly in diseased plant residues. Spores can be carried for several kilometres by the wind. The early blight fungus also can be seed-borne in tomato. Its relatively short disease cycle allows for numerous, repeated infections, resulting in rapid defoliation under favorable conditions. Plant susceptibility increases with age, heavy fruit load and inadequate nutrition. *Alternaria alternata* is a weakly virulent pathogen that typically infects wounded or senescent tissues. It is sometimes found in early blight lesions on tomato leaves and fruit. In addition, it is known to colonize tissues damaged by frost, growth cracks, sunscalded areas, and wounds caused by mechanical injury, chemical phytotoxicity or other diseases on both green and ripe fruit. The fungus is able to grow through exposed epidermal layers, resulting in small black lesions. In time, even during cold storage, these lesions may coalesce and cover large areas of the fruit, especially on the shoulders. Diseases caused by *Alternaria alternata* are often referred to as black shoulder, black mold rot or alternaria fruit rot *(18.8f,g).* The general environmental conditions favoring infection and disease development on tomato are the same as for potato.

Management

Cultural practices — Control can be achieved by extending crop rotations to three or four years, using disease-free transplants, minimizing plant injury, and maintaining plant vigor. When irrigation is required, morning watering will allow the leaves to dry before a new dew period begins in the evening.

Resistant cultivars — Varying levels of genetic resistance exist among currently grown field cultivars. HY 9478, Malinta and Medalist are tolerant to early blights.

Chemical control — Properly timed foliar fungicides are effective in reducing losses caused by early blight. TOM- CAST, a weather-timed fungicide spray program, is available to commercial tomato growers to help determine when applications are warranted. Daily disease severity values (DSV) are calculated from surface wetness and temperature data. Fungicide sprays are recommended only when specified accumulated DSVs have been reached.

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(Original by R.E. Pitblado and R.J. Howard)

▶ 18.9 Fusarium crown and root rot *Figs. 25. 10a d*

Fusarium oxysporum f. sp. *radicis-lycopersici* W.R. Jarvis & Shoemaker

Fusarium crown and root rot is primarily a disease of greenhouse tomato (see Greenhouse tomato, fusarium crown and root rot, 25.10), but it also has been reported in field tomato in Ontario. Fusarium crown and root rot contamination of seedling tomato has occurred where tomato and pepper seedlings have been grown in close proximity to or within the same greenhouse complex as full-season greenhouse tomato production. Growers should avoid this practice. The use of soilless mixtures in seedling trays placed on racks above the soil surface lessens the opportunities for fusarium crown and root rot infection. Piles of crop residues are an important source of spores of the pathogen and should be eliminated.

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(Original by R.E. Pitblado)

▶ 18.10 Fusarium wilt *Figs. 25.11a-c*

Fusarium oxysporum f. sp. *lycopersici* (Sacc.) W.C. Snyd. & H.N. Hans.

Fusarium wilt (see Greenhouse tomato, fusarium wilt, 25.11) is a minor disease of field tomato. Since the introduction of the *I-1* gene, which provides resistance to race 1 in most tomato hybrids grown in Canada, losses from this disease have been minimal. Race 2 of *Fusarium oxysporum* f. sp. *lycopersici* has been identified throughout the southern United States but has not yet become established in Canada. Excellent monogenic resistance is available in germplasm having the *I-2* gene, which is now routinely being used in tomato breeding programs throughout the world.

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(Original by R.E. Pitblado)

▶ 18.11 Gray mold (ghost spot) *Figs. 18.11a-c; 25.12a-d*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold (see gray mold of Greenhouse tomato, 25.12; Greenhouse pepper, 24.3; and Lettuce, 11.10) occurs in the field under conditions of prolonged high humidity. The extent of damage is usually minor but it can be a problem in newly- planted soft-grown transplants in cool, wet springs. Roughly handled transplants are particularly susceptible. Affected tissues are tan-colored *(18.11a),* soft and often occur on plant parts close to the ground. Field symptoms are mainly ghost spots on fruit (*18.11b*) and fruit rotting *(18.11c).* Ghost spots appear on the fruit as a superficial pale halo or ring with a brown to black pinpoint spot in the center. On green fruit *(25.12d),* the halo may be pale green or silvery and the tissue inside the halo is generally paler green. On ripe fruit, the halo is usually pale yellow. Ghost spots rarely develop further, but they can reduce market quality. (For management strategies, see Lettuce, gray mold, 11.10.)

(Original by R.E. Pitblado)

▶ 18.12 Late blight *Figs. 18.12a-d; 25.13a,b; 16.11T1*

Phytophthora infestans (Mont.) de Bary

Late blight (see Potato, late blight, 16.11) can infect tomato, especially when plantings are close to blighted potato crops. The disease causes severe defoliation and fruit rot *(18.12a-d).* From 1946 to 1948 and in 1957 and 1976, late blight was epidemic in the tomato-growing areas of southern Ontario. However, with dry weather patterns and effective fungicide spray programs, late blight is no longer considered to be a serious disease in southern Ontario.

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(Original by R.E. Pitblado)

▶ 18.13 Septoria leaf spot *Figs. 18.13a-c*

Septoria lycopersici Speg.

Septoria leaf spot is a common disease of field tomato in central Canada and often is found in association with early blight. The disease usually does not become prevalent until late in the season. Rapid defoliation and heavy crop losses can occur. Tomato is the only crop attacked by *Septoria lycopersici.*

Symptoms Under conditions favorable for infection, temperatures between 20 and 25°C and extended periods of leaf wetness, lower leaves are peppered with small dark circular spots. As the spots enlarge, the centers of the lesions turn light tan with dark margins *(18.13a).* Within the lesions, black, pinhead-sized pycnidia can be seen (*18.13b*) that help distinguish septoria lesions from those of early blight. Septoria leaf spots often have several wavy black lines at the edge of each lesion, which may result in the disease being misidentified as early blight. The disease spreads from lower leaves and stems (*18.13c)* to upper leaves on affected plants. There is seldom direct fruit damage; however, yield loss can occur as a result of reduced fruit size and an increased susceptibility to anthracnose and sunscald. Septoria leaf spot does not produce the degree of foliage yellowing that early blight does. Defoliation can occur rapidly under favorable conditions for disease development.

Causal agent (see Greenhouse tomato, septoria blight, 25.15)

Disease cycle *Septoria lycopersici* is seed-borne and also can overwinter in decomposing tomato residues in fields. Spores can be spread by water, workers, equipment, insects, wind-blown soil, and infested plant debris. Extended periods of leaf wetness and temperatures above 18°C favor disease development. Epidemics are usually delayed under normal spring conditions and the disease is seldom observed in the field until late July.

Management

Cultural practices — Control measures for septoria leaf spot are similar to those for early blight and anthracnose. Growers should use disease-free seed and transplants and provide balanced nutrition to promote healthy, vigorous growth.

Resistant cultivars — Resistance to septoria leaf spot is available in several breeding lines and is being incorporated into commercial tomato cultivars.

Chemical control — Registered fungicides are available. The TOM-CAST forecasting system is available to help growers time their foliar fungicide applications (see early blight, 18.8).

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(Original by R.E. Pitblado)

▶ 18.14 Verticillium wilt *Figs. 18.14a-c; 25.16a*

Verticillium albo-atrum Reinke & Berth.
Verticillium dahliae Kleb.

Verticillium wilt can affect tomato, pepper and eggplant. It is caused by two species of soil-inhabiting fungi. In general, *Verticillium dahliae* prefers warmer soils and is commonly encountered in southern Ontario and British Columbia. It is also the main cause of verticillium wilt in greenhouse tomato. *Verticillium albo-atrum* is mostly present in cooler areas; in particular, it is found in Quebec and the Maritime provinces. These two fungi are often found together in the same field. They can attack potato and other solanaceous plants (see Potato, verticillium wilt, 16.20), strawberry, raspberry and certain stone fruits. They are also found on many weeds, which thus aid in their carry-over from crop to crop (see also Greenhouse cucumber, 22.17).

Symptoms Symptom expression is similar for tomato, eggplant and pepper. The first symptom on leaves is yellowing followed by wilting *(18.14a).* Lesions on leaves of *Verticillium*-infected plants have a characteristic V-shaped yellowing pattern *(18.14b),* which is widest at the leaf margins, narrowing to a small V toward and sometimes including the leaf midrib. The deep brown tissue within these lesions is always surrounded by a large, yellow, irregular area *(18.14c; 25.16a).* In tomato, this leaf symptom is often confused with those of early blight. A further diagnostic feature is that several of the surrounding leaves also may show the distinctive yellow coloration without any dark or necrotic tissue, the initial sign of the systemic leaf toxin. The disease affects lower leaves first, then moves upward. The fungus affects the vessels, so symptoms often appear only on one side of the plant and sometimes only on one side of the leaf. For the same reason, symptoms are more pronounced during drought periods. Leaf wilting is followed by necrosis and stunting. When the stem of an infected plant is cut lengthwise, the vascular tissue is brown, another characteristic symptom of the disease. Infected pepper and eggplant usually collapse rapidly and eventually die.

Verticillium wilt can be confused with fusarium wilt. Both diseases affect vessels within the vascular system of plants. In seedlings, verticillium wilt causes tan necrosis, while fusarium wilt produces a mahogany discoloration of the vascular tissues. In questionable situations, the two diseases can be distinguished only by isolating the causal organism.

Causal agent (see Potato, verticillium wilt, 16.20)

Disease cycle (see Potato, verticillium wilt) In tomato fields, *Verticillium* species survive on infected crop residues in the form of microsclerotia in *V. dahliae* and as resistant mycelium in *V. albo-atrum.* Weeds often serve as symptomless hosts. Infection takes place in the roots; the fungus invades the vessels and interferes with water transport either by obstructing the vessels or by producing a toxin that causes wilt. Hastened entry occurs when accompanied by plant parasitic nematodes. Combinations of nematodes and *Verticillium* cause a decline and loss in yield similar to that found in potato.

Management

Cultural practices — Growers should follow a four- to five-year crop rotation to allow infested plant residues to decompose in the soil. Long rotations help reduce the level of fungal inoculum in fields; however, *Verticillium* spp., especially *V. dahliae,* can survive for many months in the absence of susceptible plants. Grain crops should be included in the rotation. Whenever possible, infested plant material should be gathered and destroyed after harvest.

Resistant cultivars — Most commercial tomato cultivars have the *Ve*-gene that confers a level of resistance. However, a second race of *V. dahliae,* first reported from Ohio in 1962 and since observed in Ontario, has resulted in the decline of several popular tomato cultivars. Resistance to *Verticillium* spp. in pepper and eggplant is poor. As with tomato, genetic resistance is available, but it is specific only for certain races of *Verticillium.* One control measure is the grafting of eggplant onto *Verticillium*-resistant tomato rootstocks. The grafting is done in a greenhouse and the plants are then transplanted to the field.

Biological control — Toxin-producing or antagonistic organisms are being evaluated, but none is commercially available.

Chemical control — Growers should fumigate fields before transplanting if soil tests indicate high levels of *V. dahliae* or plant parasitic nematodes.

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(Original by L.M. Tarder and R.E. Pitblado)

▶ 18.15 White mold *Figs. 18.15a-e*

Sclerotinia minor Jagger
Sclerotinia sclerotiorum (Lib.) de Bary

(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

White mold, caused mainly by *Sclerotinia sclerotiorum,* and rarely in Canada by *S. minor,* can be a destructive disease of field tomato in Ontario. Either species can cause a collar rot of transplants and a stem and fruit rot of mature plants. Both species of *Sclerotinia* have a wide host range, particularly in vegetable crops such as bean, carrot, cauliflower, cabbage, celery, cucurbits, pea and rutabaga. Weeds and refuse piles are potential sources of inoculum.

Symptoms In young transplants, the hypocotyl can be infected by mycelial growth from senescent cotyledons. Lesions are water-soaked at first, though the rotted area usually remains fairly firm. The affected tissue appears bleached, and there is almost invariably a prolific, pure white fungal growth on the stem (*18.15a,c,e*). The fungus may also infect stems at the soil line, especially if senescent tissue is present. This can result in collar rot *(18.15b),* a condition in which the stem rots and causes the affected plant to wilt and die. In the case of *S. minor,* small, flat, black sclerotia about 1 to 2 mm in size appear on the outside of the stem, often coalescing into masses *(18.15b).* In *S. sclerotiorum,* the sclerotia are black, larger (5 to 8 mm) and irregular in shape *(18.15d).* They are formed mostly inside the stem.

On older plants, lesions may be initiated anywhere on the shoot, usually at the site of a leaf scar or where fallen flower has lodged. Lesions may attain several centimetres in length and girdle the stem *(18.15b,c)*\ all of the tissue above a large lesion dies. Fruit becomes infected from colonized, senesc- ing tissue adhering to it and sometimes from latent infections in the senescent flower parts. Infected fruits rot completely *(18.15a).*

Black sclerotia lying in a mass of white mycelium inside hollow stems are diagnostic of *S. sclerotiorum,* while smaller sclerotia, aggregated in masses and always external, are typical of *S. minor.* Pale, beige-colored apothecia, 1 to 3 mm in diameter, are produced at the soil surface in spring and throughout cool, moist summers; careful searching may reveal an abundance of small, inconspicuous apothecia on the soil surface.

Causal agent (For a description of *Sclerotinia sclerotiorum,* see Bean, white mold, 15B.9, and for *S. minor,* see Lettuce, drop, 11.9.)

Both fungi grow readily on a variety of agar media. The mycelium is always pure white and abundant, and both species produce typical sclerotia in culture. The sclerotia usually produce abundant apothecia when placed on damp sand or floated on water in diffuse light at about 25°C.

Disease cycle (see Bean, 15B.9, and Lettuce, 11.9)

Management

Cultural practices — The sclerotia are long-lived and germinate whenever they are brought to within 2 to 3 cm of the soil surface by cultivation. Control by a short rotation usually is not very successful. An interval of at least three or four years of cereal cropping is needed to reduce their numbers appreciably in the field. Weeds should be eradicated because many of them also are hosts, and they serve to maintain a humid microenvironment in the crop. Refuse piles should be removed and buried deeply or composted properly to ensure the destruction of sclerotia. Field crop rows should run parallel to the prevailing wind so that plants dry out quickly after rain. Excessive overhead irrigation should be avoided where white mold is a potential problem.

Resistant cultivars — No resistant cultivars of any vegetable are known, but those with a more open habit are less susceptible than those with a dense habit in which water is slow to evaporate.

Biological control — Sclerotia are damaged by sciarid flies and are parasitized by a number of other fungi. There has been limited success in biological control with one or two of these fungi but not on a commercial scale.

Chemical control — Dicarboximide and benzimidazole fungicides may be used, but fungicide tolerance may develop quickly. Efficacy should be monitored closely and spraying stopped at the first sign of resistance to the fungicides.

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(Original by W.R. Jarvis)

VIRAL AND VIRAL-LIKE DISEASES

Viral diseases can adversely affect tomato and pepper crops in Canada. Symptoms vary depending on the virus type, virus strain, host plant, time of year and environmental conditions, and often go unrecognized or are misdiagnosed. Seven viruses have been identified as attacking field tomato and pepper in Canada, but those affecting eggplant have not been studied. Several other viruses are known to attack these crops worldwide.

▶ 18.16 Aster yellows

Aster yellows mycoplasma-like organism

Aster yellows is a minor disease of tomato, eggplant and pepper. Affected plants are stunted, chlorotic and may have a stiff growth habit. Fruits from yellows-infected tomato plants may be catfaced (see catface, 18.23). Aster yellows is a more important disease in crops such as lettuce (see Lettuce, aster yellows, 11.15) and celery (see Celery, aster yellows, 7.8). Management of this disease centers on controlling the aster leafhopper (see Lettuce, 11.23), which vectors the aster yellows pathogen.

(Original by R.J. Howard)

▶ 18.17 Cucumber mosaic *Figs. 18.17; 25.18a,b*

Cucumber mosaic virus

Cucumber mosaic is often misdiagnosed as tomato mosaic, and they both occur commonly in tomato crops. Cucumber mosaic virus can attack a large number of plant species (see Greenhouse cucumber, cucumber mosaic, 22.20).

Symptoms Cucumber mosaic causes a distinct narrowing ("shoestring") of young tomato leaves (*25.18a)* and conspicuous mosaic symptoms (*25.18b*) similar to those produced by tomato mosaic virus. The shoestring leaf symptoms sometimes resemble injury caused by growth-regulating herbicides, such as 2,4-D. Shoestring is often confused with "fern- leaf," a symptom caused by tomato mosaic virus (*18.18a, 25.21c)* (see Greenhouse tomato, 25.21), but shoestring often can be distinguished by its narrower, tendril-like leaflets. Plants with severe shoestring often are stunted and produce little or no marketable fruit.

In pepper, severe foliar mosaic may occur and older leaves sometimes exhibit large, necrotic rings *(18.17).* Fruit may be malformed, with conspicuous, yellow, concentric rings and/or spots on immature fruit.

Causal agent (see Greenhouse cucumber, cucumber mosaic, 22.20)

Disease cycle (see Greenhouse cucumber, cucumber mosaic) Limited spread of cucumber mosaic virus may occur in the field through handling plants. The green peach aphid is the most widespread and efficient vector, but potato and melon aphids also may spread the pathogen.

Management (see Cucurbits, cucumber mosaic, 9.15)

Selected references

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(Original by R.E. Pitblado and R.J. Howard)

▶ 18.18 Tomato mosaic, single streak, double streak *Figs. 18.18a-e; 25.19a,b; 25.21 a-e*

Tomato mosaic virus

Tomato mosaic is widespread on field tomato in Canada but often goes unnoticed or is not diagnosed accurately. The names "tomato mosaic" and "tobacco mosaic" are often used interchangeably (see Greenhouse tomato, 25.20 and 25.21). The extent of crop losses from this virus in field tomato, pepper and eggplant in Canada is not well documented. The virus occurs in many strains and its effect on susceptible cultivars can range from none to severe. Tomato mosaic virus has a wide host range (see Greenhouse tomato, tomato mosaic, 25.21).

Symptoms Light and dark green mottling (mosaic) of the leaves *(25.21b),* distortion, and a reduction in the size of the leaflets *(18.18a)* are the most characteristic symptoms of tomato mosaic in tomato. Plants attacked early in the season may be slightly stunted *(25.21a),* while later infections have little or no noticeable effect on plant growth (see also symptoms of "femleaf," *25.21c,* and "shoestring," *25.18a).* Fruit set may be severely reduced. Internal browning of the fruit wall, yellow blotches and necrotic spots may occur on both green and ripe tomato fruit *(18.18b-e; 25.21d,e).*

At least one strain of tomato mosaic virus is also involved in another distinct disease of tomato called "streak" or "single streak." This disease is characterized by longitudinal brown streaks on the leaves and petioles, and dark blemishes on the fruit. A more severe disease, "double streak" *(25.19a,b),* is the result of a combined infection of tomato mosaic virus and potato virus X (see Greenhouse tomato, double streak, 25.19).

On pepper, very prominent mosaic symptoms appear on the foliage accompanied by leaf puckering and reduction in leaf size. Vein clearing of the young leaves becomes extremely pronounced. Older leaves fall prematurely. Yield is reduced because fewer fruit are set and those that do are small and misshapen.

Causal agent (see Greenhouse tomato, 25.21) Tomato mosaic virus differs only slightly in host, serological and cross-protection reactions from tobacco mosaic virus. Strains of tomato mosaic virus have been classified by their ability to induce symptoms in plants of certain *Lycopersicon* spp. or in isogenic tomato lines.

Disease cycle (see Greenhouse tomato, 25.21) Tomato mosaic virus can be spread by anyone who handles or brushes against diseased plants then handles healthy ones in operations such as tying, pruning, cultivating and harvesting. The virus also can be spread on tools and machinery.

Management (see Greenhouse tomato, 25.21)

Cultural practices — Growers should keep seedling production areas free of weeds and ornamentals. Young seedlings should not be clipped. Diseased plants should be pulled and left to die in the field as soon as virus symptoms are noticed; this practice minimizes the spread of the virus by direct contact between plants. Cultivators, tools and other equipment should be disinfested before moving from diseased to healthy crops.

Resistant cultivars — Tomato and pepper cultivars with some resistance to tomato mosaic virus are commercially available in Canada.

Chemical control — Suitable herbicides should be used to eliminate weeds that may harbor viruses within tomato, pepper and eggplant crops and in borders and fence rows that surround production fields. Insect pests should be controlled with insecticides where populations warrant.

Selected references

Broadbent, L. 1976. Epidemiology and control of tomato mosaic virus. *Annu. Rev. Phytopathol.* 14:76-96.
Fletcher, J.T., and D. Butler. 1975. Strain changes in populations of tobacco mosaic virus from tomato crops. *Ann. Appl. Biol.* 81:409-412.
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Zaitlin, M., and H.W. Israel. 1975. Tobacco mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 151. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

(Original by R.E. Pitblado and R.J. Howard)

▶ 18.19 Tomato spotted wilt *Figs. 24.8b,c; 25.22a-d*

Tomato spotted wilt virus

Tomato spotted wilt (see Greenhouse tomato, 25.22, and Greenhouse pepper, 24.8) is a viral disease that was relatively uncommon in field tomato and pepper crops in Canada before 1989. However, greenhouse tomato and pepper crops have been affected by this disease since 1984, when tomato spotted wilt became an important problem in the greenhouse floriculture industry in Canada. In the spring of 1989, tomato spotted wilt was observed extensively throughout Ontario wherever tomato and pepper crops were grown. The virus and its thrips vector (see western flower thrips, 18.42) were imported into Ontario with vegetable transplants from the southern United States. A protocol to produce virus-free transplants has been developed for use by Canadian growers. The virus has a very wide host range.

Symptoms Moderate to heavily infected transplants, once planted in the field, appear stunted but seldom die. They remain unproductive, never growing beyond the seedling size. Transplants only slightly infected at planting time initiate growth, but later show characteristic leaf symptoms. Pepper leaves become mottled *(24.8b)* and often have circular, raised, yellow zones. Tomato foliage turns purplish- brown (bronzing) *(25.22a,b).* Fruits are irregular in shape and color with circular markings of alternate red and yellow bands *(25.22c,d; 24.8c).*

Causal agent (see Greenhouse tomato, tomato spotted wilt, 25.22)

Disease cycle (see Greenhouse tomato, tomato spotted wilt)

Management

Cultural practices — Management of tomato spotted wilt should emphasize exclusion of the insect-disease complex. Field-grown transplants imported from the USA often require repeated applications of insecticides to control the thrips vector. Locally grown transplants require stringent sanitation strategies to produce disease-free plants. The practice of growing vegetable transplants with bedding plants often leads to transplants becoming infected with the virus.

Using an effective protocol for managing thrips and tomato spotted wilt virus in vegetable seedling greenhouses will provide healthy, disease-free transplants. Recommended practices include:

- providing a break in cropping for at least one month before vegetable seedlings emerge; temperatures should be set at 22°C or warmer to accelerate the hatching of any thrips eggs that are present;
- monitoring for thrips and disease using blue sticky traps and growing petunias as indicator plants;
- not growing vegetable transplants in close proximity to houses used for flower production;
- maintaining weed-free greenhouses;
- restricting visitors;

- using appropriate insecticides when necessary.

Selected references

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(Original by R.E. Pitblado)

▶ 18.20 Other viral diseases *Figs. 18.17; 25.19a,b; 25.21b*

Alfalfa mosaic virus
Potato virus X
Potato virus Y
Tobacco etch virus

Alfalfa mosaic has been observed in field pepper in Ontario. The symptoms depend on the virus strain and the environmental conditions under which the crop is growing. Yellow blotches or sometimes mosaic mottling, chlorotic rings, spots and other patterns appear in affected leaves. Severe leaf necrosis also may occur. Alfalfa mosaic virus overwinters in alfalfa crops and it is commonly transmitted by green peach aphids. (For information on the virus, see Potato, calico, 16.24.)

Potato virus X On tomato foliage, potato virus X causes a distinct light and dark green mottle similar to that produced by tomato mosaic *(25.21b).* Small dead spots sometimes appear on the affected leaves. Plants infected by both potato virus X and tomato mosaic virus exhibit symptoms of the disease known as double streak (*25.19a,b*) (see tomato mosaic, 18.18, 25.19). Pepper shows a mild mosaic symptom with leaf puckering. Leaf size may be slightly reduced. The virus is spread by contact between diseased and healthy plants and during handling. Grasshoppers reportedly are vectors. Potato virus X may carry over in potato tubers (see Potato, potato virus X, 16.27), and it also can infect a large number of other solanaceous plants.

Potato virus Y (see Potato, 16.27) is found more frequently in pepper than in tomato. The virus causes mild to severe mottling depending on the strain involved. The virus is not seed- transmitted but is spread by several aphid species, of which the green peach aphid is probably the most efficient vector.

Tobacco etch Tomato plants infected with tobacco etch virus appear somewhat stunted with mildly mottled, slightly distorted foliage. In pepper, the virus causes a very mild chlorotic mottle with some foliar distortion. Large, concentric rings and line patterns may be produced on both leaves *(18.17)* and fruit. Fruit often becomes misshapen. Root necrosis occurs, causing some wilting. Wilted plants recover but they are usually stunted and bushy. Stems of older plants sometimes show reddish-brown spots and streaks. Bud drop may occur. Tobacco etch virus overwinters in weeds of the family Solanaceae and is spread mostly by the green peach aphid and occasionally the potato aphid.

Management

Cultural practices — Most of the minor viral diseases of tomato, pepper and eggplant can be kept at low levels by using virus-free seed and transplants, by controlling insect vectors and weed hosts, and by employing strict sanitation programs in propagation greenhouses and with field equipment.

Selected references

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(Original by R.E. Pitblado and R.J. Howard)

NON-INFECTIOUS DISEASES

▶ 18.21 Blossom-end rot (bottom rot) *Figs. 18.21 a-d; 24.9; 25.23*

Blossom-end rot affects tomato, pepper and eggplant. It generally occurs on the first fruit clusters and sometimes causes significant economic losses. It is caused by a localized deficiency of calcium in the fruit and is induced by unfavorable growing conditions, especially drought. Blossom-end rot develops when there are fluctuations in water supply such as result from long

periods of hot, dry weather followed by heavy showers. The cause is a failure of the plant to absorb calcium quickly enough, even though it may be abundant in the soil. This disorder affects the first fruit clusters on tomato most severely. Clusters developing later may not be affected. Rapid plant growth, low potassium and calcium levels in plant tissue, large quantities of magnesium and nitrogen in the soil, high soil salinity, damage to the roots, and high relative humidity all serve to predispose plants to blossom-end rot.

Symptoms The first symptoms usually appear on young fruits that are a third or more developed but the disorder can occur at any stage. Initially, light brown patches appear at the blossom end of the fruit, although they can occasionally appear on the sides as well *(18.21c,d).* These patches darken and an area of sunken black tissue forms, sometimes affecting up to half the fruit *(18.21a; 24.9; 25.23).* These dead tissues may be invaded by secondary organisms that can cause the fruit to rot. In tomato, an internal rot symptom can also occur. Black areas throughout the fruit often go unnoticed until it is cut open *(18.21b).*

Management

Cultural practices — The best way to prevent blossom-end rot is to ensure steady plant growth through careful irrigation. This promotes the uptake and assimilation of calcium by the plant. In addition, providing balanced fertilization and avoiding root damage while cultivating will encourage deep root development and enable the plant to obtain adequate water during drought periods. Mulches that aid in conserving soil moisture may help to prevent blossom-end rot. Applications of lime to the soil well before planting and the use of calcium chloride or calcium nitrate sprays on the foliage during the growing season, prior to the onset of symptoms, also may be helpful.

Resistant cultivars — Cultivars differ in their susceptibility to blossom-end rot, but this has not been well documented.

Selected references

Banuelos, G.S., G.P. Offermann and E.C. Seim. 1985. High relative humidity promotes blossom-end rot on growing tomato fruit. *HortScience* 20:894-895.

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(Original by L.M. Tartier)

▶ 18.22 Blotchy ripening *Figs. 18.22a,b*

Blotchy ripening is most often encountered in the greenhouse and damage can be significant. It is also found in the field in fresh-market, processing and staked tomato crops. The cause of this disorder and its relationship to "graywall" are uncertain. It has been linked to potassium or boron deficiency and to high nitrogen levels, which favor excessive growth. Blotchy ripening also has been attributed to infection by tomato mosaic virus, but this does not appear to be the definitive cause. Weather conditions also seem to play a role in the development of blotchy ripening; the disease is more frequent when temperatures are very high and is less prevalent during sunny, mild conditions.

Symptoms Affected fruit ripens at an uneven rate. Large patches of hard, grayish or yellowish tissue are immediately visible on the green fruit. The fruit does not turn a uniform red; the patches remain gray or turn yellow *(18.22a).* This gives the characteristic appearance of poorly ripened fruit. When affected fruits are cut in half, the vascular tissues may appear brown *(18.22b).*

Management

Cultural practices — Growers should ensure that a well-balanced fertilizer program is followed.

Resistant cultivars — Some cultivars appear to be less susceptible to this problem than others.

Selected references

Dangler, J.M., and S.J. Locascio. 1990. External and internal blotchy ripening and fruit elemental content of trickle-irrigated tomatoes as affected by N and K application time. *J. Am. Soc. Hortic. Sci.* 115:547-549.

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Matsumoto, T., and C.A. Hornsby. 1974. Influence of weekly changes in temperature and light regimes on the incidence of blotchy ripening of tomatoes. *Can. J. Plant Sci.* 54:129-133.

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(Original by L.M. Tarder)

▶ 18.23 Catface (stylar cork) *Fig. 18.23*

Catface originates during the early stages of flower bud development, approximately two or three weeks before blossoming. It results from abnormal development of tissues at the junction of the style and ovary, and the resulting fruit is misshapen. Unfavorable growing conditions, such as several days of temperatures below 15°C when the plants are young, seems to be the main cause of this disorder, although other impediments to flower bud development also can result in catfacing. High levels of soil nitrogen and excessive pruning aggravate the problem. Aster yellows and injury from hormonal herbicides, such as 2,4- D, also can lead to the production of malformed fruit, but foliar symptoms can be used to distinguish these problems from true catfacing. Catface is generally most prevalent on large-fruited, fresh-market tomatoes.

Symptoms Affected fruits are generally flattened on the blossom end. Large bands of cork-like, malformed scar tissue cover the whole end of the fruit *(18.23).* The scars often criss-cross and the fruit seems to consist of lobes. Cavities sometimes form in healthy tissue. This problem is sometimes confused with injury caused by hormonal herbicides.

Management

Cultural practices — Good growing practices, especially temperature control, should be followed in greenhouse production of field transplants. High levels of soil nitrogen, excessive pruning and accidental exposure to phenoxy herbicides should be avoided in tomato crops.

Resistant cultivars — Large-fruited tomato cultivars are more susceptible to catface and should be avoided if this disorder is a persistent problem.

(Original by L.M. Tartier)

▶ 18.24 Cold injury

Exposure to low temperatures can reduce seed germination, retard growth, and damage tomato foliage and fruit. Chilling injury to tomato fruit can occur when temperatures of 0 to 10°C persist for prolonged periods, either before or after harvest. Chilling injury is likely to occur if there is an accumulation of 120 hours below 15°C during the week before harvest.

Freezing damage to tomato begins when temperatures drop to -1 or -2°C. Injury is caused by ice crystals that form in the plant tissue, thereby rupturing the cells. Plants are generally more susceptible to frost when the soil is dry, rather than moist. One factor that can affect the temperature at which injury occurs is the presence of ice-nucleation bacteria on the plant surface. When these bacteria are not present, ice does not form until the temperature reaches about -5°C.

Chilling injury to fruit frequently occurs in the home when it is stored in the refrigerator, in stores where it is put in cold storage over the weekend, and in trucks when being transported over long distances.

Symptoms Fruits not fully ripened at the time of exposure to chilling temperatures fail to ripen normally. Affected tissues may become soft and water soaked. As the tissue breaks down, it becomes increasingly susceptible to decay. When fruit is exposed to low temperature in the field, injury may not be apparent at harvest but may appear after five to seven days during storage or shipping. Frozen leaves, fruit surfaces and other tissues quickly become water soaked, then turn black. Tomato plants can usually recover from a brief exposure to frost but damaged fruit is unmarketable.

Management

Cultural practices — Growers should plant early-maturing cultivars, and avoid excessive use of nitrogen fertilizers that may delay maturity. Hotcaps or tunnels help to protect newly transplanted seedlings.

Selected references

Patterson, B.D., and L.A. Payne. 1983. Screening for chilling resistance in tomato seedlings. *HortScience* 18:340-341.

(Original by R.J. Howard)

▶ 18.25 Growth cracks *Fig. 18.25*

Growth cracking is a physiological disorder that occurs as the fruit is sizing and is the result of variations in soil moisture and temperature. Growth cracks may occur during periods of rapid fruit growth when relative humidity and air temperatures are

high or after a drought period when water becomes abundantly available after a rain storm or irrigation. Growth cracks are easily invaded by secondary organisms such as *Alternaria* spp. and soft rot bacteria that promote fruit rot.

Symptoms There are several types of growth cracks *(18.25).* Radial cracks cover the fruit, beginning at the stem end; concentric cracks encircle the stem end. As the fruit grows, these cracks deepen and expose the flesh but seldom the locules. Severe cracking or "bursting" also can occur, exposing the locules.

Management

Cultural practices — The problem can be minimized by irrigating tomato plants regularly. Since ripe fruit is more susceptible to growth cracking, irrigation should be avoided once the fruits ripen. Excessive application of fertilizers should be avoided.

Resistant cultivars — Cultivars differ in their susceptibility to growth cracking, but this has not been well documented.

(Original by L.M. Tartier)

▶ 18.26 Leafroll, herbicide injury *Figs. 18.26a-d*

Leafroll is a common physiological disorder of tomato. It also can occur on pepper, eggplant and potato. The edges of the leaves roll upward and inward to the extent that they may appear tubular *(18.26a).* This rolling appears to be a moisture conservation measure by the plant and is often permanent. Affected leaves also may have a leathery texture. The lower leaves are generally the first to exhibit leafroll symptoms, but the entire plant eventually may be affected. Overall growth and fruit production usually are not impaired. The onset of symptoms is greatest following hot, dry growing conditions. Some cultivars appear to have a genetic predisposition to this disorder. Leaf rolling and other abnormalities *(18.26b-d)* can also occur as a result of exposure to hormonal herbicides, such as 2,4-D.

Management

Cultural practices — Growers should ensure that tomato plants receive adequate moisture, especially during drought periods.

(Original by R.J. Howard)

▶ 18.27 Nutritional disorders *Figs. 25.24a,b*

Field tomato, pepper and eggplant crops are subject to a number of nutritional disorders, principally deficiencies of nitrogen, phosphorus, potassium, calcium and magnesium. Yield and quality of fruit are reduced when serious toxicides and deficiencies occur (see Greenhouse tomato, 25.23, 25.24, for descriptions of calcium and magnesium deficiencies; also see blossom-end rot, 18.21).

Selected references

Besford, R.T., and G.A. Maw. 1975. Effect of potassium nutrition on tomato plant growth and fruit development. *Plant Soil* 42:395-412.

(Original by R.J. Howard)

▶ 18.28 Puffiness *Fig. 18.28*

Puffiness affects both greenhouse and field tomato. Affected fruit loses its commercial value. Puffiness results when unfa

vorable weather conditions adversely affect pollination or fertilization of the ovule. Temperatures above 35°C or below 13°C may result in poor pollination. Extreme variations in soil moisture, genetic factors, over-fertilizing with nitrogen, and the use of fruit-setting hormones also may play a role in the development of this disorder.

Symptoms Fruit affected with puffiness is bloated, light in weight and soft. In some cases, the locules are only partially filled with gel. When the fruits are cut in half, the locules may be empty, sunken and have few seeds *(18.28).*

Management

Cultural practices — Fertilizer programs should be balanced to avoid excessive levels of nitrogen. Fruit-setting hormones should be used with caution.

(Original by L.M. Tartier)

▶ 18.29 Sunscald *Figs. 18.29a,b*

Sunscald primarily affects fruit tissue; however, leaves and stems also can be scalded, resulting in collapse of the mesophyll. Sunscald results when fruit and young foliage are exposed to the direct sunlight. The disorder is aggravated by high humidities and temperatures. This problem appears on fruit that is suddenly exposed to the sun because the foliage no longer offers protection. This can result from defoliation through diseases, such as early blight, septoria leaf spot, bacterial and wilt diseases, excessive heat, excessive leaf loss caused by fruit-ripening chemicals or insect feeding, or when vines are trained, alleys are made through fields or fruit is picked for the fresh market. Sunscalded fruit is unmarketable.

Symptoms Sunscald symptoms appear on the part of the fruit that is exposed to the sun. Affected areas are sunken and light brown to white *(18.29a,b).* These areas are often invaded by secondary organisms that can cause the fruit to rot. Early in the season, tender leaf and stem tissues can also be sunscalded and may turn a light gray to brown. The underside of the upper foliage has irregular bands across the leaf, while the stems turn whitish on the exposed side.

Management

Cultural practices — This problem can be partially avoided by keeping the foliage healthy so that the fruit is sheltered from the sun.

Resistant cultivars — None is currently available, but growers might use varieties that have sufficient foliage to protect the fruit.

(Original by L.M. Tartier and R.E. Pitblado)

NEMATODE PESTS

▶ 18.30 Northern root-knot nematode *Fig. 18.30*

Meloidogyne hapla Chitwood

Tomato, eggplant and pepper are highly susceptible to damage from the northern root-knot nematode *(18.30).* Infected plants become stunted and chlorotic and senesce early. Fruit set and size are reduced. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

▶ 18.31 Root-lesion nematode *Fig. 16.38T1*

Pratylenchus penetrans (Cobb) Filip. & Stek.

Symptoms Plant growth is stunted in heavy infestations. Affected plants occur in patches, usually extending along the rows, or elongated in the direction of cultivation. Plants wilt readily on hot days and leaves become progressively yellow. Older leaves may die prematurely. Secondary roots are necrotic with dried areas. See Potato, 16.38; see also Management of nematode pests, 3.12.

Selected references

Potter, J.W., and T.H.A. Olthof. 1977. Analysis of crop losses in tomato due to *Pratylenchus penetrans. J. Nematol.* 9:290-295.

▶ 18.32 Stubby-root nematodes

Paratrichodorus allii (Jensen) Siddiqi
Paratrichodorus pachydermus (Seinhorst) Siddiqi
Paratrichodorus spp.
Trichodorus spp.

See Potato, 16.39, and Management of nematode pests, 3.12.

INSECT PESTS

▶ 18.33 Aphids *Figs. 16.40-16.43*

Green peach aphid *Myzus persicae* (Sulzer)
Potato aphid *Macrosiphum euphorbiae* (Thomas)
Other aphids

The same species of aphids that attack potato (see Potato, 16.40-16.43) also attack field tomato, eggplant and pepper. These aphids form colonies on the undersides of leaves, and often in or around flowers. In hot, dry weather, aphid populations increase rapidly. High populations sometimes follow the early-season use of insecticides to control the Colorado potato beetle, resulting in reduced numbers of aphid predators and parasites. Aphids and their effects on crop yields and fruit quality are often overlooked. However, the ability of aphids to transmit viruses is the greatest concern.

Damage Aphids transmit several viruses that can be devastating to solanaceous vegetable crops; these include cucumber mosaic virus, alfalfa mosaic virus, potato virus Y and tobacco etch virus. Moderate to heavy aphid infestations can cause the foliage to turn yellow, resembling symptoms that can be mistaken for fungal blights. However, the foliage is covered with whitish, molted skins (aphid exuviae) and the bodies of parasitized aphids, which appear swollen and bronzed.

Aphids do not cause significant losses in production except under dry conditions, when they can reduce plant growth, thereby lowering yields. Leaves become twisted and cupped as a result of feeding by clusters of aphids on the underside of the foliage. Additional damage results from honeydew, which supports the growth of sooty mold that reduces the marketability of the fruit.

Identification (see Potato, 16.40-16.43)

Life history (see Potato, 16.40-16.43)

Management

Resistant cultivars — A number of tomato, eggplant and pepper cultivars carry a level of resistance to some virus diseases transmitted by aphids, but there is no apparent resistance to aphids.

Biological control — Growers often rely on lady beetles and other predators (see Beneficial insects, mites and pathogens, 3.7) to keep aphid populations low.

Chemical control — In most years, chemical control of aphids is not warranted and is difficult to achieve because of poor under-foliage coverage when insecticides are applied with conventional sprayers. Other factors mitigating the efficacy of chemical control are the development of resistance to chemical insecticides by aphids and rapid increases in aphid populations once initial control has been achieved. Products with activity against aphids and the European corn borer or Colorado potato beetle are preferred. Chemical insecticides should only be applied when they are required, and control can be improved by using a systemic insecticide.

(Original by R.E. Pitblado)

▶ 18.34 Colorado potato beetle *Figs. 16.44a-d; 16.44T1*

Leptinotarsa decemlineata (Say)

The Colorado potato beetle occurs in all tomato-growing regions of Canada. In Essex and Kent counties in southwestern Ontario, where 80% of the Canadian field-tomato crop is grown, the Colorado potato beetle is of particular concern to growers, especially early in the growing season at the time of transplant establishment. High beetle populations are present because of the numerous potato fields in that area and the high proportion of insecticide-resistant beetles found in southwestern Ontario. The tendency for both tomato and eggplant crops to be grown on lighter, sandy soil-types, where high overwintering populations of adult beetles occur, coupled with the lack of crop rotation, also have played a role in the Colorado potato beetle populations with which tomato growers now have to contend.

Eggplant and pepper are not seriously affected by this insect.

Damage Adults and larvae of the Colorado potato beetle are capable of defoliating young tomato seedlings. Damage is scattered throughout a field wherever the in-field overwintering adults emerge and begin to feed, or it is concentrated along field borders when adults from neighboring fields move in and later where mated females have laid their eggs. Severely defoliated plants may have only the stem and larger leaf mid-ribs remaining, causing a 50% loss in yield. If 50% of the foliage remains after a beetle attack, a tomato seedling may often outgrow this early season damage with no subsequent loss in yield. Colorado potato beetles also cause feeding damage to tomato fruit.

Identification (see Potato, 16.44)

Life history (see Potato, 16.44) Overwintered adults of the Colorado potato beetle emerge from the soil in early spring. They immediately seek and begin to feed on host plants. Both the adults, popularly called "hard shells," and the larvae or "soft shells" feed on tomato foliage early in the season. Adults that emerge after mid-July often fly to nearby potato crops.

Management

Monitoring — Emerging adults within a tomato field begin to feed, mate and lay eggs immediately after transplants are planted. Therefore, early detection is important. There are no established action thresholds, but the presence of 10 larvae or adults per 100 plants is a useful guideline for timing application of an insecticide to young transplants. Populations of Colorado potato beetle in tomato fields are clumped and spotty, and extensive sampling is required to monitor populations accurately. Monitoring field edges gives an indication of beetle populations moving into vegetable fields. Growers while cultivating should take note of areas that are moderately or heavily damaged by beetles.

Cultural practices — Crop rotation is only moderately effective as a management strategy because beetles that overwinter in nearby potato fields readily fly into tomato fields in the spring. Eggplant transplants or, preferably, potato rows planted strategically in a tomato field act as trap crops, reducing Colorado potato beetle damage to the tomato crop.

Resistant cultivars — Tomato cultivars with substantial anti-feeding resistance, which results from an increase in glyco-alkaloid content, are being developed but few of these are available commercially. There is concern about the safety to humans of these high glyco-alkaloid cultivars. Other resistance mechanisms are being evaluated.

Biological control — Foliar application of the bacterium *Bacillus thuringiensis* Berl., San Diego strain, which is effective against larvae of the Colorado potato beetle, is an alternative to the use of chemical insecticides.

Chemical control — Insecticidal applications are effective in reducing damage caused by Colorado potato beetle. However, in contrast to the range of insecticides available for potato, only a few foliar insecticides are recommended for tomato. These may be "spot-sprayed" onto heavily infested areas of fields where beetle numbers warrant. If possible, growers should not apply insecticides to entire fields and should avoid over-treating, because most of the insecticides used against the Colorado potato beetle kill beneficial insects, such as aphid predators and parasites, resulting in high populations of aphids in late July and

August. If adult Colorado potato beetle populations are low, microbial insecticides can be used to control the larvae after eggs have hatched, rather than applying chemical insecticides to kill the adults. Overwintered adults, if abundant, must be treated to prevent defoliation of young transplants. The use of insecticides in transplant water helps in early season control.

Colorado potato beetle was reported in 1971 to have developed resistance to endosulfan and other organochlorine insecticides but organophosphate and carbamate insecticides remained effective. Since then, widespread resistance to insecticides has been documented, limiting the choice of chemical insecticides available to tomato growers in Canada. Carbamates are not very effective against adult potato beetles in comparison to the synthetic pyrethroids and organophosphates, which still have fast knock-down capabilities. Resistance to azinphos-methyl, an organophosphate, has been demonstrated in the major tomato-growing areas of Canada. Endosulfan, an organochlorine that was once ineffective because of resistance and was thus not used for many years, now appears to be effective against the Colorado potato beetle population in some areas.

Selected references

Jaques, R.P., and D.R. Laing. 1989. Effectiveness of microbial and chemical insecticides in control of the Colorado potato beetle (Coleoptera: Chrysomelidae) on potatoes and tomatoes. *Can. Entomol.* 121:1123-1131.

(Original by R.E. Pitblado)

▶ 18.35 Cutworms *Figs. 18.35a-g; 6.25a-c; 11.26*

Variegated cutworm *Peridroma saucia* (Hübner)
Other cutworms (see Table 18.35)

Cutworms cause problems to tomato, eggplant and pepper transplants by feeding on the plant stems at or near ground level (see also Carrot, cutworms, 6.25).

Cutworms are solitary feeders and many are subterranean. They have been known to cause serious damage to vegetable crops, especially in the second year after sod or pasture. However, cropping after sod is no longer a common practice. Early cutworm damage now is observed only at field edges along ditch banks or hedgerows, or within fields where weeds are present.

The variegated cutworm is a sporadic pest of tomato in southern Ontario and across Canada. High moth catches are frequent, beginning in the second week of July. Whether the moths are transitory, flying into Canada from more southern overwintering sites, or local, is open to question.

In years when natural biocontrol agents are ineffective, variegated cutworm larvae may cause substantial losses in fruit quality and yield. The variegated cutworm has an extremely wide host range and does not confine itself to tomato.

Damage Damage to foliage and fruit of tomato is common during late July and throughout August. Foliar damage consists of scattered leaf feeding, particularly along the leaf edges, but sometimes the entire leaf is eaten, leaving only the midrib. Fruit damage may consist of light surface feeding or feeding that produces holes deep into the fruit *(18.35a,b).* Injured fruit is often invaded by soft rot organisms.

Table 18.35 Cutworms commonly found in Canada

Common name	Scientific name
Army cutworm	*Euxoa auxiliaris* (Grote)
Black army cutworm	*Actebia fennica* (Tauscher)
Black cutworm	*Agrotis ipsilon* (Hufnagel)
Climbing (spotted) cutworm	*Xestia adela* Franclemont
Dark-sided cutworm	*Euxoa messoria* (Harris)
Dingy cutworm	*Feltia jaculifera* (Guenée)
Glassy cutworm	*Crymodes devastator* (Brace)
Granulate cutworm	*Agrotis subterranea* (Fabricius)
Pale western cutworm	*Agrotis orthogonia* Morrison
Redbacked cutworm	*Euxoa ochrogaster* (Guenée)
Sandhill cutworm	*Euxoa detersa* (Walker)
Striped cutworm	*Euxoa tessellata* (Harris)
Variegated cutworm	*Peridroma saucia* (Hübner)
White cutworm	*Euxoa scandens* (Riley)

On tomato transplants and other vegetable crops, cutworm larvae may chew partly or completely through the stem or petioles at ground level or, in the case of climbing cutworms, somewhat higher on the plant.

Identification Larvae of the variegated cutworm (family Noctuidae) are brown with longitudinal stripes *(18.35b,c).* They have mottled, diamondshaped markings along the back and sides of the body. Other cutworm larvae (*18.35d; 6.25a-c; 11.26)* are similarly marked and difficult to distinguish from the variegated cutworm without rearing to the adult stage. Adult identification should be confirmed by a specialist.

Feeding damage is accompanied by large, brown or black droppings (frass) on the soil surface.

Life history Moths of the variegated cutworm appear in mid-July and their numbers peak during the first to second week of August. Eggs are laid on the foliage and hatch within 5 to 10 days. The young larvae begin eating the tomato foliage and later attack the fruit. They often chew a feeding hole into the side of the developing tomato fruit. The larvae of the variegated cutworm often remain curled inside the feeding holes in the fruit, feeding mostly at night. The larvae complete their development and pupate in the soil. The variegated cutworm pupae overwinter in British Columbia, especially in the coastal areas, but whether they overwinter in Ontario is uncertain.

Management

Monitoring — Moths of the variegated and other cutworms can be monitored by black-light or pheromone traps, starting early in the season. Once observed, routine field inspection is necessary.

Biological control — Naturally occurring predators, parasites and pathogens usually exert significant influence because in most years cutworm populations cause little damage. Wherever feasible, growers should try to preserve these natural biocontrol agents.

Chemical control — Growers are advised to apply foliar insecticide treatments in the evening when larval activity is greatest, particularly early in the season when natural biocontrol agents are insufficient to suppress cutworm populations. Good spray coverage with penetration into the lower canopy is essential for adequate control.

Selected references

Rockburne, E.W., and J.D. Lafontaine. 1976. *The Cutworm Moths of Ontario and Quebec.* Can. Dep. Agric. Publ. 1593. 164 pp.

(Original by R.E. Pitblado and J.A. Garland)

▶ 18.36 European corn borer *Figs. 18.36a,b*

Ostrinia nubilalis (Hübner)

The European corn borer overwinters in pepper- and eggplant-growing areas throughout Canada except British Columbia, where this insect does not occur. Crops also may become infested by adults that fly in or are blown in during the growing season.

Field pepper is an important host of the European corn borer, more so than eggplant, potato and snap bean.

However, pepper is not as attractive to the corn borer as is sweet corn, its preferred host. Tomato is not affected by this insect.

Damage In areas having single-generation corn borer populations, damage to pepper occurs from mid-July to early August. Where two generations are produced, damage occurs during late July, then again throughout August and September. Corn borer populations are heaviest in southwestern Ontario and Quebec, which are also the areas of highest pepper production in Canada.

Larvae (*18.36a*) usually enter the fruit under the calyx stem-cap. A yellowish-brown, sawdust-like residue of droppings (frass) (*18.36b*) may be noticeable around the entry hole. If the larva enters directly through the side of the fruit during early pepper development, the fruit becomes dimpled in that area.

Severe loss in field pepper crops can be caused directly by fruit damage by the feeding corn borer larvae, or indirectly by rotting caused by pathogens introduced by the larvae. Fungi or bacteria often gain entry through the entrance holes created by the larvae, causing the fruit to collapse or rot. Losses are greatest for farmers who grow processing peppers because of a contractual load-rejection clause controlled by the processors, by which they can reject an entire load with more than 1 to 5% borer-damaged fruit.

Identification (see Maize, 12.16)

Life history (see Maize, 12.16) Overwintering corn borer larvae occur mainly in the stalks of field corn where they pupate and emerge as adults in the spring. Adults invade pepper fields in the spring, congregating in “hot spots” in response to the stage of pepper growth and the proximity of grassy areas, which they use as daytime refuges. Corn borer eggs are laid in masses on the leaves of pepper plants or on the pepper fruit-cap. Soon after the eggs hatch, larvae feed on the foliage, causing visual damage, and soon move toward the fruit, boring into the side or under the calyx stem-cap.

Management ***Monitoring*** — Field pepper crops are susceptible to corn borer attack as soon as the fruit is 3 cm in diameter. From then until harvest, the crop must be monitored and protected if corn borers are present. Adult corn borers can be monitored with black-light traps or commercial pheromone traps. Difficulties with sampling preclude the determination of a threshold value for corn borer egg masses on pepper.

Resistant cultivars — All bell-type peppers are susceptible to corn borer. Only the pepper cultivar known as Sweet Hungarian and the hot-type cultivars, such as Hungarian Wax and Long Thick Red, have high levels of resistance to corn borer attack. Pepper cultivars grown in Ontario show corn borer resistance in descending order, as follows: Hungarian Wax (Hot), Long Thick Red (Hot), Sweet Hungarian (Yellow Banana), Super Set 19, Staddon’s Select, Super Shepherd, MA 79252,

Greenboy, Early Niagara Giant, Golden Bell, Lady Bell, Midway, Romanian Wax (Hot), Gedeon, Vinedale, Jupiter, Bell Boy, Emerald Giant 38, California Wonder, Keystone Resistant Giant and Yolo Wonder 43.

Chemical control — When corn borer populations are present, pepper growers should follow a preventive foliar spray program once the fruit has attained 3 cm in diameter (walnut size). Insecticidal applications then should begin when adults are trapped for three consecutive days and should be repeated every 7 to 10 days, depending on temperature and subsequent moth catches. Once eggs hatch, larvae quickly move under the fruit cap and bore into the fruit. This limits exposure, reducing the likelihood of an insecticide contacting the larvae, and makes early spray coverage essential for good control of European corn borer.

(Original by R.E. Pitblado)

▶ 18.37 Other caterpillars *Figs. 18.37a-c; 8.40; 12.13*

Cabbage looper
Trichoplusia ni (Hübner)
Corn earworm
Helicoverpa zea (Boddie)
Hornworms
Manduca spp.

Other caterpillars are observed occasionally in commercial tomato fields. In southern Canada, these include the cabbage looper (see Crucifers, 8.40), the corn earworm (see Maize, 12.13), and two *Manduca* species of hornworms (*18.37a-c*).

Tomato hornworms occur in the warmer, southern areas of Canada. They are particularly prevalent in southwestern Ontario and southern British Columbia. The large, green larvae are easily diagnosed by the seven or eight oblique white lines along the sides of the body and a prominent caudal horn *(18.37b).* Hornworm larvae feed on the foliage and may strip an entire leaf, leaving only the midrib. Although larvae feed on green fruit *(18.37c),* damage is rarely extensive.

In some years, hornworms and other caterpillars require control measures, but naturally occurring predators, parasites and pathogens usually keep them under control. Late- season damage to foliage in August and September by the cabbage looper and hornworms is usually of little concern. Fruit damage by the corn earworm is minor.

(Original by R.E. Pitblado)

▶ 18.38 Pepper maggot *Figs. 18.38a-g*

Zonosemata electa (Say)

The pepper maggot is native to and occurs throughout the eastern United States and southwestern Ontario. In Essex County, Ontario, it is an important but sporadic pest of field pepper. Horse nettle-infesting populations have been found as far north as London, Ontario.

The pepper maggot confines itself to solanaceous plants. The primary crop-host is pepper but larvae have been collected from eggplant and tomato. Several solanaceous weeds, including ground cherry *(Physalis* spp.), serve as hosts. Horse nettle, *Solanum carolinense* L., *(18.38a)* a particularly persistent, perennial weed in soybean and corn fields in southwestern Ontario, is thought to have been the original wild host of the pepper maggot.

Damage The first sign of damage on pepper is the small egg puncture formed by the female's ovipositor *(18.38b).* Egg punctures usually occur when fruit is about 1 to 3 cm in diameter. When pepper fruit increases in size, the area around the egg puncture becomes depressed, forming a shallow dimple. Most larval activity occurs in the soft placental tissue, or core, of the pepper fruit *(18.38c).* Damage appears as a brown, mined area and can be distinguished from corn borer damage by the absence of droppings (frass) *(18.36b).* Sometimes larval mines are visible beneath the epidermis of the pepper fruit, particularly after harvest. Pepper usually has only one larva per fruit, but eggplant commonly contains several larvae that mine the fruit extensively.

Pepper maggot has little impact on production of field pepper in Canada, because it is restricted to southern Ontario. However, in the absence of controls, growers of fresh-market crops in southern Ontario experience losses as high as 90% in some years. The zero tolerance for pepper maggot in processing peppers means that this insect can be devastating.

Identification Pepper maggot (family Tephritidae) adults are brightly colored, yellow-striped flies with banded wings and green eyes (*18.38d*). The head, thorax and abdomen are pale yellow, with two black dots on the dorsal side of the last abdominal segment. The legs are yellow with short black bristles ventrally. Males are about 6.5 mm and females about 7.5 mm long. The egg is 2.0 to 2.2 mm long and opaque white with a striated shell and a distinctive, narrow stalk *(18.38e).* Larvae reach 11 to 12 mm in length at maturity. They are legless, white maggots that turn yellow as they mature *(18.38f).* Pupae (puparia) are about 8 mm in length, 4 mm at the widest point, and medium buff-brown (*18.38g*). The above measurements are based on individuals reared on pepper. When reared on horse nettle, all life stages are about 10% smaller.

Egg punctures often go undetected early in the season. Frequently, the stalk of the egg protrudes from the skin of the fruit and may be seen with the unaided eye, but detection often requires at least 10X magnification. Pepper fruit suspected of

containing pepper maggot should be cut open to expose the internally feeding larva. Infested eggplant fruits usually feel very spongy.

Previously, populations of pepper maggot infesting horse nettle and pepper were thought to be distinct host races with different biologies. However, recent work has shown that the life history and behavior of the pepper maggot on each of these hosts are similar. Thermal requirements, phenologies and activity periods also are similar. In the laboratory, females collected from horse nettle, pepper and eggplant will oviposit in fruits of pepper, eggplant, tomato and ground cherry, regardless of the plant from which they originated.

Life history The pepper maggot has the typical fly life stages of egg, larva, pupa (puparium) and adult. The pupa overwinters in a state of arrested development (diapause) in the top 15 cm of soil, the majority at depths of 5 to 10 cm. Diapause development requires about 150 days below 5°C; adults begin to emerge when soil at the 10-cm depth has accumulated 475 degree-days (range 409 to 541) above 9.5°C, measured after January 1. Peak emergence occurs near the end of June, and 50% of adults will have emerged when the soil at the 10-cm depth has accumulated about 600 degree-days above 9.5°C. Emergence extends over a period of 10 to 12 days, depending on the weather. Males and females emerge at the same time. They fly to host plants where they mate. The dispersal range is unknown; however, new infestations have occurred at least 1 km from known populations. The first mating may occur within 24 hours of emergence. Both sexes may mate several times in their lifetime. The pre-oviposition period lasts about six to seven days or until accumulating 180 degree- days above 9.5°C (air temperature).

Females live about 23 days and lay an average of 54 eggs, but some females live as long as 45 days and are capable of laying up to 200 eggs. Females seem to prefer small fruit (1 to 3 cm diameter) for oviposition, but this may reflect the size of fruit available during egg laying. Small fruits usually harbor single eggs while large fruits may contain several eggs. Larvae hatch within 10 days and young larvae may feed in the wall of the fruit, but most move directly to the core of soft placental tissue and remain there. When mature, the larvae move to the lower third of the fruit, bore through the fruit wall, drop to the ground and enter the soil to pupate. There is one generation per year in Canada.

Management

Monitoring — Detecting pepper maggot damage on pepper before the larva leaves the fruit is often difficult, although larvae are present until harvest when their exit holes become visible. Sampling for adults must be done visually or with a sweep net. Adults are most commonly observed resting on small fruits (1 to 3 cm diameter) in the early morning. Despite a zero tolerance for pepper maggot in pepper fruit destined for processing, there are no reliable traps for monitoring adult populations and no threshold is available.

Cultural practices — Horse nettle near plantings of pepper or eggplant should be removed, because this plant is a potential reservoir and source of pepper maggot infestations. Destruction of horse nettle in the first year of a rotation may not decrease damage to crop plants because pepper maggot flies emerging from horse nettle sites will continue to infest pepper and eggplant. Weed control must be practiced for several years. Fruit known to be infested with pepper maggot should be harvested early and buried deeply to reduce the next year's population.

Resistant cultivars — In fields and regions with a history of damage from pepper maggot, infestations can be reduced by planting the less preferred or more resistant pepper cultivars. Females prefer to oviposit on dark green, fleshy peppers, particularly the bell and cherry types. Thin- walled Yellow or Red Banana, Cayenne, Jalapeno, Tabasco, and Serrano pepper cultivars are the least preferred for oviposition by pepper maggot females, and these cultivars are highly resistant to larval feeding. Late-maturing cultivars also sustain less damage, because few flies are present after early August; any fruit that develops at that time is not likely to be infested.

Biological control — A wasp, *Opius sanguineus* (Ashmead), has been recorded in low numbers in pepper maggot larvae infesting horse nettle in Ontario, but never from larvae from pepper or eggplant. Predatory beetles likely cause some pupal mortality.

Chemical control — The pepper maggot is very susceptible to most chemical insecticides, and resistance has not been documented to any of the insecticides currently used in production of field pepper in Ontario. Because the eggs and larvae are protected within the fruit, foliar sprays for control of pepper maggot are effective only against adults. Insecticides used against European corn borer provide good control of pepper maggot and preclude the need for additional sprays. For optimum control of pepper maggot, the first application should be when 180 degree-days above an air temperature of 9.5°C have accumulated after the actual or predicted 50% emergence date for pepper maggot adults. An additional spray should be applied seven days later.

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(Original by G.J.R. Judd)

▶ 18.39 Sap beetles *Fig. 18.39*

Various species of sap beetles (family Nitidulidae) are attracted to cracked or squashed tomato fruits in the field or on wagons, hoppers or trucks standing in the field. Eggplant and pepper fruit also attract sap beetles but to a lesser extent than tomato. Although the transport system used in machine harvesting of processing tomato greatly reduces the need for concern of sap beetles, these insects remain a problem when tomatoes are hand-picked.

Life history (see Maize, four-spotted sap beetle, 12.19)

Management Growers must rely on cultural practices to reduce the occurrence of sap beetles in the field, because practical methods for the use of insecticides to control these beetles are not available.

Cultural practices — Roadways should be provided in the field at suitable intervals to allow movement of farm vehicles without squashing fruit. Growers should harvest as close as possible to the time of delivery, avoid leaving picked tomatoes in the field for long periods, and expose loaded wagons to air circulation if the load is not to be delivered promptly.

(Original by R.E. Pitblado)

▶ 18.40 Stink bugs *Figs. 18.40a,b; 3.7k,m*

Various species of stink bugs occur throughout the tomatogrowing areas of Canada. Until recently, they have not been considered seriously as pests. However, recent changes in cultivar selection and cultural practices have enhanced stink bug presence and their damage to tomato fruits.

Stink bugs have a wide host range, which includes alfalfa, cereals, soybean, bean, pea, tomato, and many weeds.

Damage As weedy areas dry out or mature during the summer, stink bugs move into tomato fields, presumably in search of their liquid diet. Consequently, damage to tomato fruit is often limited to the edge of the field nearest weedy areas. The piercing-sucking mouthparts of the stink bug adults and nymphs inflict damage to the surface of the tomato fruit, causing development of cloudy yellow blotches just below the skin of the fruit as a result of enzymes injected by the feeding insect (*18.40a,b).* Surface depressions also can form at the feeding sites. Stink bug feeding causes fruit distortion and defects, such as peel "tags" remaining on the fruit and a yellow blemish in the tomato flesh. As fruits enlarge, sites of early feeding expand and may rupture the thin epidermis over the wound, permitting entry of secondary organisms. Increase in sorting costs or rejection of the entire load at the factory may result from stink bug injury to tomato fruit. Losses can be significant for the wholepack and fresh- market industries.

Identification Stink bugs (family Pentatomidae) are 10 to 15 mm long and vary in color from green to brown. Their wings are folded flat over the abdomen with the membranous outer halves of the wings directed toward the rear of the body. The adults have pointed "shoulders" on the front part (pronotum) of the thorax (*18.40a; 3.7m).* Nymphs are similar in appearance but lack fully developed wings and the pronotum is not as pointed *(3.7k).*

Life history Stink bugs overwinter as adults in protected areas, such as fencerows, ditches, windbreaks or other areas where plant litter is abundant. In early spring, when temperatures reach 21°C or above, the adults become active. They feed initially on weeds. A single female may lay an average of 30 egg clusters during a month or more. Each egg cluster may contain 300 to 500 eggs. The nymphs hatch within a week and develop through five instars. The adult stage is attained after about six weeks. Repeat generations occur at five- to six-week intervals during the summer. Adults and nymphs spend much of their time deep within the plant canopy and, at times, slightly below ground. Adults move out of weedy areas in search of moisture in tomato fruits, especially during dry summers.

Management

Cultural practices — Stink bug damage has increased with the introduction of programs that include conservation tillage, extensive use of cover crops, and preventive practices to control wind erosion. These practices inadvertently favor stink bugs by increasing the availability of hosts and hiding places. Tomato cultivars that have an extensive foliage cover can be damaged severely. Increased damage during relatively dry years suggests a greater and earlier dispersal from the weed hosts to secondary, crop hosts. Growers are advised to eliminate weedy patches near field edges.

Chemical control — Treatment thresholds and reliable sampling methods have not been developed for stink bugs on tomato, so growers are wise to adopt a conservative approach in countering stink bug damage. Moreover, a proportion of some stink bug populations usually remains either on or in the soil where spray coverage is poor, resulting in inadequate control. Chemical insecticides normally would be applied in the latter part of July but may not be economical for an entire acreage. Spray applications, if necessary, should be directed around the field borders.

(Original by R.E. Pitblado)

▶ 18.41 Wireworms *Figs. 12.21a,b,T1; 16.50*

Wireworms (see Maize, 12.21) are found in all soil types and in all crop-production areas of Canada. Many different species cause damage to vegetable and field crops. Among vegetables, all root crops are susceptible, as well as potato, sweet corn and transplanted crops, such as cole crops, tomato, eggplant and pepper.

Damage Wireworms cause damage to plant roots and stems just below the soil surface. Plants attacked by wire- worms wilt, and the top 1 to 2 cm of plant tissue "flags" and eventually dies. Wireworms can be observed burrowing into the underground stems and roots of transplants, which eventually results in above-ground death of the plant. Damage is more severe in years when soils are cool and wet, conditions which retard plant growth but not insect feeding. Under good growing conditions, transplants often outgrow minor wire- worm damage. Pepper plants, however, do not compensate as well as tomato and eggplant, and each pepper plant lost represents an important loss in total yield.

Identification (see Maize, 12.21; Potato 16.50)

Life history (see Maize, 12.21)

Management (see also Potato, 16.50)

Monitoring — Wireworms remain in a field for several years, so problem areas must be treated or managed for consecutive seasons.

Cultural practices — In the past, growers were advised to protect vegetable crops, especially after sod. However, wireworms have caused significant damage whether sod preceded the crop or not. Weed control to eliminate grasses reduces the availability of egg-laying sites within fields. Rotation with alfalfa is beneficial but not always practical.

Chemical control — Growers will become aware of areas of fields that must be treated, because wireworms remain in the soil for several years. Insecticides applied in the transplant water effectively reduce the damage caused by wireworms. Some insecticides may be phytotoxic under cool, wet spring conditions.

(Original by R.E. Pitblado)

▶ 18.42 Other insect pests *Figs. 18.42a-m*

Crickets and grasshoppers
Flea beetles
Greenhouse whitefly *Trialeurodes vaporariorum* (Westwood)
Tarnished plant bug *Lygus lineolaris* (Palisot de Beauvois)
Vinegar flies *Drosophila* spp.
Western flower thrips *Frankliniella occidentalis* (Pergande)

Crickets and grasshoppers

Crickets (family Gryllidae) and grasshoppers (family Acrididae) migrate into tomato fields in August and September from nearby weedy or grassy areas. These insects may damage foliage, and crickets also often eat the skin of tomato fruits. If crickets begin to move into commercial tomato fields, one or two insecticidal applications directed around the border of the field usually will protect the crop.

Flea beetles

Flea beetles (not the same species as those on cabbage and other crucifers) are not serious pests of tomato, eggplant or pepper, although they may act as vectors of fungi, such as the early blight pathogen *Alternaria solani.* The existing schedules for fungicidal applications, using the weather-timed spray program TOM-CAST, have all but eliminated concern about flea beetle transmission; the small, circular leaf damage caused by the adult flea beetles is easily compensated for by the newer, rapidly growing tomato cultivars. Flea beetle damage to emerging, direct-seeded tomato seedlings has led to the development of control recommendations. However, direct seeding of tomato is no longer practiced commercially in Canada.

Greenhouse whitefly

The greenhouse whitefly (see Greenhouse tomato, 25.27) can be found on field tomato in significant numbers, mainly in southern Ontario and southwestern British Columbia. Their sucking method of feeding and honeydew deposits result in stickiness to foliage and fruit. This is not a problem in tomato crops that are machine harvested for processing, but the stickiness resulting from high whitefly populations can be a nuisance for pickers in hand-harvested operations and for fresh-market crops.

Tarnished plant bug

The tarnished plant bug (see Celery, 7.21) (*18.42b-e*) feeds on the flowers and stems of tomato, eggplant and pepper, causing flower drop which reduces yield in some years. Fruit also may be attacked *(18.42a),* leading to indentations and yellowing of the flesh where the fruit is "stung" by the piercing-sucking mouth- parts of the plant bug nymphs and adults. Early damage often is not noticed by growers and control measures are seldom used.

Injury by tarnished plant bug is likely to increase as cultural practices within the vegetable-growing areas of Canada shift toward reduced tillage, which increases plant residue on soil surfaces and thereby improves the available habitat for these insects (see stink bugs, 18.40).

Vinegar flies

are also known as vinegar fruit flies (family Drosophilidae) (*18.42f,g*). They are only a minor problem in fruits wounded in the field by equipment, birds or insects, such as crickets and variegated cutworms. These so-called "fruit flies" hardly merit consideration except when fruit is damaged or bruised at harvest, or when it is kept or stored for an extended period, in which case their impact is greatest on fresh-market tomatoes.

Western flower thrips

The western flower thrips (see Greenhouse cucumber, 22.34) (*18.42h-m*) has recently expanded its range as a field pest to eastern Canada from western Canada and the southern United States. It can be a serious problem in the production of tomato and pepper because it transmits tomato spotted wilt virus. In 1989, both the western flower thrips and tomato spotted wilt virus were recorded for the first time in Canada on field tomato and pepper in southern Ontario. The western flower thrips and the virus are thought to have been imported on transplants from the southern United States and may continue to be imported because both pests are endemic in the southern United States, which is the source of a large portion of the transplants used to establish the tomato and pepper crop each year in Canada. As the local plug-transplant industry expands in Canada, fewer transplants will be obtained from the southern United States, reducing the importation of the virus and the thrips. The western flower thrips is difficult to control because the immature and adult thrips prefer to feed in flower blossoms, which shelter them from predators and where they often escape the lethal effects of chemical insecticides. (For more information about the western flower thrips, see Greenhouse cucumber, 22.34.)

(Original by R.E. Pitblado)

OTHER PESTS

▶ 18.43 Slugs *Figs. 18.43; 11.27a-c*

During wet conditions, slugs may shred leaves of pepper plants and attack young pepper and tomato fruit. Feeding holes made in the fruit can serve as entry sites for bacteria. (For more information on slugs, see Crucifers, 8.49; Lettuce, 11.27.)

(Original by R.E. Pitblado)

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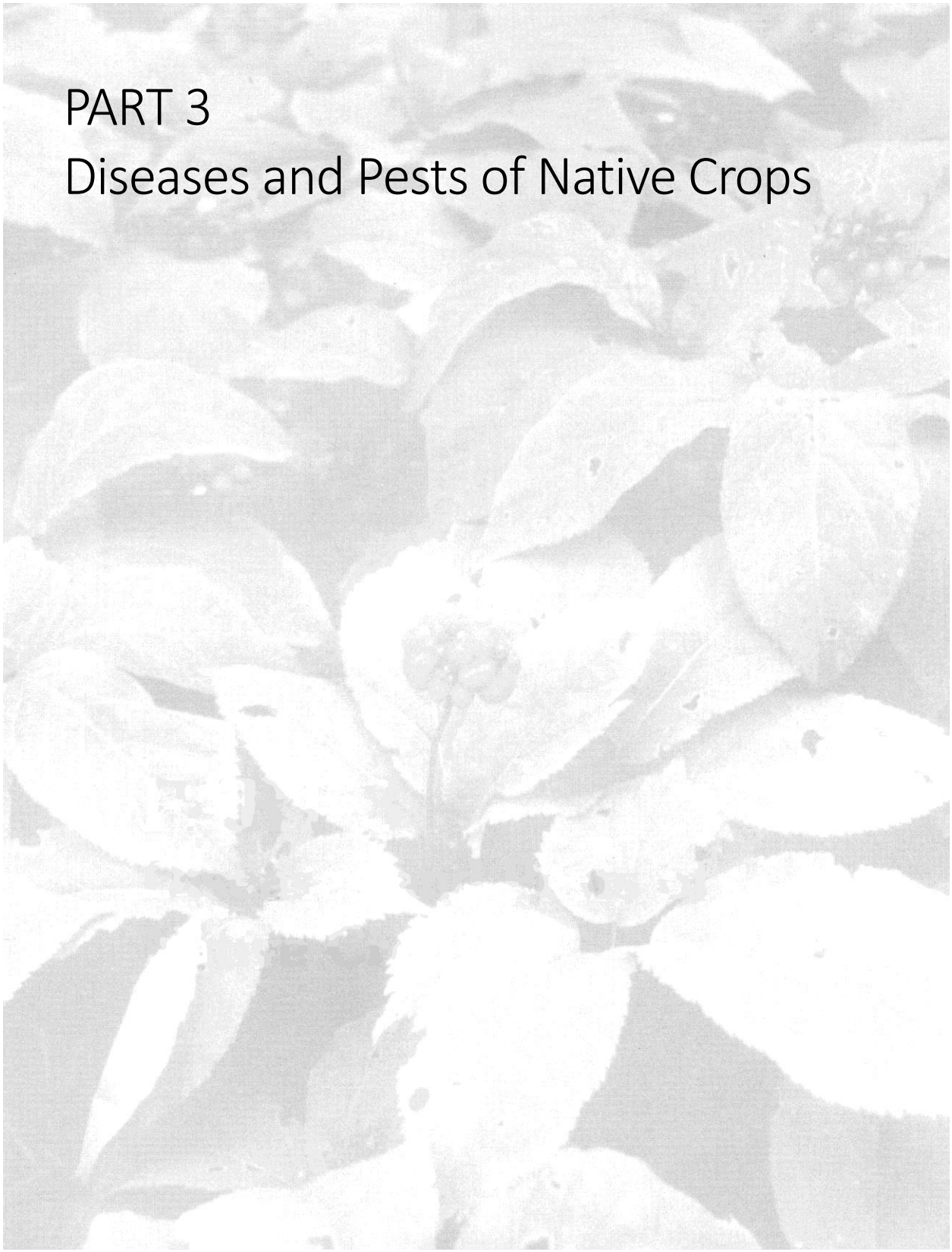

PART 3
Diseases and Pests of Native Crops

Locating Text Sections and Figures

Text sections are numbered consecutively within each chapter. For example, section 16.2 describes bacterial soft rot, the second topic of Chapter 16, Potato. To find a text section, refer to the running heads, which carry the inclusive section numbers for each two-page spread.

Color illustrations, grouped near the back of the book, appear in the same order and have the same number as the corresponding text section; for example, figures *16.2a* and *16.2b* illustrate the symptoms of bacterial soft rot of potato. Line drawings, halftones and tables are numbered similarly, except that a text figure number contains the letter T; for example, Figure *16.2T1* illustrates the disease cycle of bacterial soft rot of potato.

19 Fiddlehead (ostrich fern)

FUNGAL DISEASES

19.1 Gangrene *Fig. 19.1*

Phoma matteuccicola von Aderkas et al.

Gangrene is the most important disease of ostrich fern. It is frequently observed in the spring when wet soil, night frosts and low temperatures favor disease development. Infected fiddleheads are unsaleable.

Symptoms Small black patches appear on the frond rachis as it emerges in the fiddlehead stage. These patches are usually most apparent on the first flush of fiddleheads in the spring. The rachis can be completely blackened and weakened *(19.1).* The frond rachillae and pinnules are not affected. The black lesions caused by *Phoma* infection can be present on both fresh and frozen fiddleheads.

Causal agent *Phoma matteuccicola* produces noticeable pycnidia with extruding masses of conidia. The thin-walled, spherical pycnidia usually produce two-celled conidia, but in culture a majority of the spores are one- celled. The two-celled conidia are up to 16 pm long by 1.4 to 3 pm wide. The conidia are guttulate and arise from doliform or ampulliform, phialidic, conidiogenous cells in the inner layer of the pycnidium.

The fungus can be isolated from diseased tissue using routine techniques. Cultures on agar media are regular in outline and never scalloped. The mycelium is whitish-gray and the medium turns yellowish brown below the mycelial mat. The culture margins react positively but weakly to Boerema's sodium hydroxide test for Antibiotic E. These characteristics are similar to those of the potato pathogen *Phoma exigua* var.*foveata,* which is not known to occur in North America. *Phoma matteuccicola* is not pathogenic on potato, and it has a different pigment chemistry, grows more slowly in culture, and forms less aerial mycelium than *P. exigua var. foveata.*

Disease cycle The pathogen overwinters in the stems, roots and sub-meristematic tissue in the crown of the fern sporophyte. It can also survive in the soil and on decomposing leaves. The mycelium grows through the xylem to newly emerging fiddleheads and through rhizomes to other crowns in the same clone in the spring. The infected fronds become weak and break near the base. Severely infected crowns may die. The pathogen does not completely destroy fern stands.

Management

Cultural practices — All plant residues, including leaf litter and infected plants should be removed and buried or composted. Straw mulch or sawdust plus wood shavings or chips should be added in late autumn to protect exposed crowns during the winter and early spring.

Selected references

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(Original by R.K. Prange)

19.2 Other fungal diseases

Leaf blotch *Taphrina struthiopteris* Nishida
Rust *Uredinopsis struthiopteridis* StörmenDietel

These diseases are of minor importance on ostrich fern in Canada.

ADDITIONAL REFERENCES

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FUNGAL DISEASES

▶ 20.1 Alternaria blight *Figs. 20.1 a-c; 20.3a*

Alternaria panax Whetzel in Whetzel & Rosenb.

Alternaria stem and leaf blight was first described from New York State in 1904 and has been known in Ontario since that time. This disease now affects ginseng production throughout North America and Asia. It can produce severe epidemics when not controlled with fungicidal sprays. Alternaria blight may kill young plants directly or limit the yield of harvested roots by causing premature defoliation. *Alternaria panax* has been reported to cause disease in several members of the Araliaceae, most of which are tropical foliage plants.

Symptoms Stem infection results in the production of elongate, reddish to dark brown lesions *(20.1b).* Any portion of the stem may be affected as well as the petioles and peduncles. The lesions often appear to originate where the stem is in contact with straw mulch. Leaves collapse downwards and turn yellow to reddish-brown. These symptoms are similar to those of phytophthora root rot. Leaves are killed when the lesions girdle the stem. In seedlings, the entire plant collapses, resulting in a damped-off appearance *(20.3a).* The fungus sporulates on the surface of the lesions, producing conidia in chains or as solitary spores, giving the lesions a velvety-brown appearance.

The foliar symptoms of alternaria blight are distinguished from botrytis blight by the presence of a yellowish border *(20.1a)* and the absence of a gray surface mold. However, microscopic examination of the lesions may be required to differentiate between these two diseases. Stem symptoms are distinguished from *Rhizoctonia* damage by the velvety brown surface sporulation and the occurrence of lesions on the upper portions of the stem. Characteristic, club-shaped *Alternaria* conidia may be seen on the lesion surface at low magnifications.

When foliage is infected, the pathogen causes rapidly enlarging, circular, water-soaked spots, which may later dry, turn brown and have a target-board appearance with a dark yellow-brown margin. Lesions may appear anywhere on the surface of the leaves. The interior portions of the lesions may eventually fall out to produce a shot-hole effect. Plants may be defoliated by the rapidly spreading foliar infection.

Causal agent *Alternaria panax* produces conidia on the surface of infected plant tissues. It can overwinter on residues from the previous crop. Conidia *(20.1c)* are large, beaked, 150 to 160 by 12 to 20 µm, with 9 to 11 transverse septa in the main body of the spore. The central four to five segments may possess one to two longitudinal septa. Conidiophores are geniculate. Much smaller (less than 30% of the described size), often swollen-appearing conidia are frequently encountered on older lesions or in artificial culture. Production of conidia in culture is erratic and only small spores in chains are formed. The fungus grows well on standard media, such as potato-dextrose or V-8 juice agar, and can be isolated from lesion margins or directly from conidia that have been dislodged from the lesion surface.

Disease cycle Alternaria blight is first seen on the leaves in early to mid summer, especially in warm (20 to 25°C), rainy or humid weather. Stem cankers appear earlier in the spring before the foliar lesions are formed. All ages of plants are affected, but stem lesions predominate on the youngest plants. *Alternaria panax* is also suspected of causing dark brown to black root lesions and the abortion of developing berries, but critical work remains to be done on these aspects of the disease. Often, plants affected by alternaria blight occur in patches in the garden, suggesting spread from an initial focus. Plants defoliated one year may re-emerge the following year, but the yield of roots is reduced.

The pathogen is thought to overwinter as conidia or mycelia on the straw mulch and infested crop residue from the previous year. Plant-to-plant spread is by air-borne conidia. Relatively little is known about the infection process on ginseng.

Management

Cultural practices — Growers should remove affected plants from the garden, if practical. The movement of machinery or workers from infested to non- infested gardens should be restricted. Excessive rates of nitrogen should be avoided to limit canopy growth and improve air circulation and the penetration of sprays into the plots.

Chemical control — Registered fungicides are available.

Selected references

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(Original by R.A. Brammall)

▶ 20.2 Botrytis blight *Fig. 20.2*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Botrytis blight is common in southwestern Ontario and is one of the most important foliar diseases currently affecting the crop. Botrytis blight also occurs in British Columbia, but the arid growing conditions of central British Columbia are less favorable for disease development than those in southern Ontario. The pathogen affects the leaves, flowers and fruit, leading to defoliation of the plants and poor seed set. Although the disease is easy to diagnose by signs, growers often mistake the foliar lesions for those of alternaria blight. *Botrytis cinerea* has a wide host range that includes many vegetable crops (see Lettuce, gray mold, 11.10).

Symptoms Botrytis blight of the foliage is characterized by rapidly enlarging, water-soaked lesions. Infection often starts at the leaf tip and spreads back along the midrib, giving the lesion a V-shape. Lesions may be found anywhere on the surface of the leaf (20.2). They show a target-board appearance, similar to those of alternaria blight. Affected leaves may turn reddish to brown prematurely. The fungus often sporulates on the rotted tissues, producing a characteristic fuzzy gray mold.

Infection of the developing flowers causes them to abort. Aborted flowers desiccate and the persistent pedicels turn reddish to brown. Fruit development over the umbellate inflorescence is uneven. When immature green berries become infected, they turn brown and often show signs of *Botrytis* sporulating over the surface. Mature red fruit may become covered by a dense gray fungal growth.

Causal agent *Botrytis cinerea* (see Lettuce, gray mold, 11.10) can be isolated from margins of lesions or from conidia removed from the surface of rotting tissue. It grows well on common fungal growth media, such as potato-dextrose or V-8 agar.

Disease cycle Plants may be infected at any stage of their development and at any time throughout the growing season. The disease is favored by temperatures lower than 20°C. Symptomatic plants first appear near garden edges, especially if these plants have been subjected to sand-blasting injury, a common occurrence in southwestern Ontario. Abundant conidia are formed on the surface of rotted tissues and are aerially dispersed to produce new infections. Spread through the garden is rapid, especially when a dense canopy is present, usually in gardens three years of age and older. Lesions are usually first seen on the uppermost leaves, but a careful examination often reveals the disease to be well developed on the shaded understorey leaves. Infected leaves collapse and drape over those lower in the canopy, thus initiating new infections at points of contact. The fungus overwinters as sclerotia within infested plant residue, primarily stems, from the previous year. These structures often produce conidiophores and conidia early in the spring at the time when leaves first emerge from the soil.

Management

Cultural practices — Diseased crop residues should be removed if practical. In the past, growers would often burn off the straw mulch on the beds each autumn, but this is no longer a common practice. Cultural methods that prevent sand-blasting may limit infection.

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(Original by R.A. Brammall)

▶ 20.3 Damping-off, root rot, seed decay *Figs. 20.3a,b*

Alternaria panax Whetzel in Whetzel & Rosenb.
Fusarium spp.
Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk) Root-knot nematode *Meloidogyne* sp.
Root-lesion nematode *Pratylenchus penetrans* (Cobb) Filip. & Stek.

Seed decay, damping-off and seedling root rots are caused by a range of organisms that are common in soils or in plant residue as facultative saprophytes. Root damage is often extensive in first-year ginseng gardens, even where soil fumigants have been applied. Damping-off is an important and widespread disease, often leading to a significant reduction in plant stand. These diseases are often poorly diagnosed and have not been well studied. Damping-off fungi and nematodes can attack a wide range of vegetable crops.

Symptoms Seed decay may occur in the seed stratification boxes or in the gardens after planting. The seed coat often remains intact, with interior tissues first developing a cheesy texture, then rotting away. Post-emergent damping-off may appear on individual plants or clusters of plants. Initially, the stems remain erect with the leaves drooping downward *(20.3a),* followed by stem collapse and death of plants. Circular bare patches may develop within the garden *(20.3b).*

Damping-off caused by *R. solani, Pythium* or nematodes *(Pratylenchus* and *Meloidogyne* spp.) results in plants with a water-soaked or brown rot of the roots or below-ground portion of the stem. *Pratylenchus penetrans* may also be found in root tissue with these symptoms (see Potato, 16.38). In contrast, *Alternaria panax* produces girdling and brown stem lesions above the soil line *(20. 1b)* (see alternaria blight, 20.1).

Although root damage often leads to plant death, other plants may exhibit lesser amounts of root or stem damage. In such cases, most of the primary root may be rotted. The production of secondary roots may permit the plant to survive, in which case the damage results in roots with bizarre shapes. The humanoid appearance of some ginseng roots may be a result of these infections.

Causal agents Accurate identification of the cause of damping-off requires isolation and confirmation of pathogenicity of the organisms in inoculation trials. *Pythium, Rhizoctonia, Fusarium, Alternaria panax* and *Pratylenchus penetrans* may be recovered from plants with root-rotting or damping-off symptoms. Bacteria also can cause certain types of damping- off or seed decay. In Korea and Japan, for example, the bacterium *Erwinia carotovora* (Jones) Holland has been implicated in seed decay and red root rot of ginseng. In Ontario, fluorescent pseudomonads and *Fusarium* spp. are commonly recovered from decayed seeds. All of these organisms are common in soils or as saprophytes in plant residue. Because many of them often attack diseased tissues as secondary invaders, much critical work needs to be done before damping-off and root rot diseases are better defined and more easily diagnosed.

Disease cycle Infection may occur before or during germination of seed. Fungal pathogens, such as *Pythium* or *Fusarium,* can produce spores within or upon decaying host tissue. Certain pathogens, especially *Alternaria* species, may be seed-borne. Others can use the straw mulch that covers the raised garden as a pathway for plant-to-plant spread or may spread through the soil from saprophytically colonized bits of plant residue.

Typically, harvested berries are fermented to remove the pulp from the seed, then buried in seed boxes, often filled with sand, for a period of about one year before being recovered and planted in a new garden. This stratification is apparently required to break seed dormancy, although the percentage germination is often poor. Rotting of seeds and premature sprouting of seeds in the box are common. Growers often treat seed with formaldehyde before placing it in a seed box. Fluorescent pseudomonad bacteria and *Fusarium* spp. can be recovered from rotted and partially rotted seeds. It is likely that a percentage of planted seed is infected with such seed-rot organisms, which may kill the seed before germination or cause pre- or post-emergence damping-off. The presence of infected seeds probably causes seed death before germination and spread of seed-rotting organisms causes pre- or post-emergent damping-off. Seed infected by *A. panax* may introduce the pathogen into new gardens, although this has not been documented.

Management

Cultural practices — Seed boxes should be isolated from ginseng gardens to prevent contamination of the seed by infested crop residue or run-off. Losses caused by seed rots, premature sprouting and poor germination could be avoided if stratification procedures were carried out above ground in refrigerated facilities. Unfortunately, the environmental conditions required to break seed dormancy are not well established.

Gardens should be situated on well-drained soils. Growers generally use raised beds to promote drainage but it is not clear whether these are required on sandy soils.

Chemical control — Soil fumigation is often carried out before seeding new gardens. Such procedures destroy beneficial soil microflora and may facilitate establishment and spread of pathogens in the soil. In Ontario, growers generally fumigate soil in late summer before making the raised beds into which seed is planted. Despite fumigation, root-lesion nematodes often can be recovered from damped-off seedlings in the year after planting. Growers in British Columbia may or may not fumigate, depending on local nematode populations or previous experience. Registered fungicides are available for control of damping-off caused by *R. solani.*

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Mordue, J.E.M. 1974. *Thanatephorus cucumeris.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 406. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by R.D. Reeleder)

▶ 20.4 Disappearing root rot *Figs. 20.4a,b*

Cylindrocarpon destructans (Zinssmeister) Scholten
(teleomorph *Nectria radicicola* Gerlach & L. Nilsson)

Disappearing root rot affects plants of all ages and has been a major problem in south-central Ontario for over 50 years. The nearly total destruction of the root gives the disease its common name. The disease occurs in the northern United States and in Korea and China. In Ontario, it may occur in a broad range of different soil types and may cause severe plant losses in individual gardens. *Cylindrocarpon destructans* is common in soils of coniferous woodlands and is an important pathogen in tree nurseries. Whether it persists on organic residues in garden sites is unknown.

Symptoms The disease affects all of the subterranean portions of the plant. Diseased plants may fail to emerge in the spring or they repeatedly wilt and recover, but the aerial portions are usually killed. Small, discolored, gold to brown areas appear on the root surface in the early stages of infection *(20.4a).* The lesions are shallow at first, then enlarge rapidly and deepen, producing a reddish-brown, spongy rot below the level of the periderm. The exterior of the root develops a dark brown discoloration at infection sites during the more advanced stages of the disease *(20.4b).* Lateral rootlets may be affected, producing a distorted taproot. The rot may progress into the crown and the stem. Attempting to pull such plants from the soil causes the stem to separate from the root. In later stages, only fragments of root periderm and the vascular tissues remain. Damage to the root system produces wilting of the aerial portions of the plant that is often one-sided. The foliage may turn red to brown after repeated wilting. In gardens that are harvested after three to four years, most affected plants have only partially rotted roots.

Disappearing root rot is distinguished from phytophthora root rot by dark-colored lesions and the absence of a persistent soft rot in the cortex. Conidia of *Cylindrocarpon destructans* occasionally may form on the surface of rotted roots. These conidia resemble and may be confused with the macroconidia of *Fusarium* species. (See also rusted root, 20.6.)

Causal agent In North America, disappearing root rot was originally ascribed to *Ramularia panicicola* Zinssmeister, *R. mors-panicus* Hildebrand, and *R. robusta* Hildebrand. The latter species was considered to be less pathogenic than the other two. Now, however, *Cylindrocarpon* is preferred over *Ramularia* based on criteria such as conidial and cultural characteristics. In China, this root rot is reported to be caused by *Cylindrocarpon destructans.* It is likely that C. *destructans* is also the correct name for the pathogen in Canada.

The septate hyphae vary from less than 1 to 4 µm in width and are mostly hyaline in culture. Melanized hyphae eventually arise and may produce brown chlamydospores upon aging. The thick-walled chlamydospores are mostly intercalary, solitary or in chains of two to four, and have a diameter of 12.5 to 20.0 µm. Conidia form on and within the rotted root tissues. Eventually, thick-walled, brown-pigmented, globose chlamydospores form and are presumed to be the overwintering stage. Microconidia are oval to elliptical, 6 to 10 by 3.5 to 4 µm, while the one- to five-septate macroconidia are cylindrical with rounded ends, 20 to 40 by 5 to 6.5 µm.

The fungus can be isolated from the margins of young lesions by washing off any adhering soil from the roots, then surface sterilizing them in 0.6% sodium hypochlorite for five minutes. Tissue segments may be plated onto potato-dextrose agar amended with 75 ppm streptomycin sulphate to minimize bacterial contamination. Macro- and microconidia and chlamydospores are readily produced in pure culture on potato-dextrose agar, on which colonies grow relatively slowly, attaining 10 to 12 mm in seven days, with the mycelium changing from grayish-white to brown to deep red-brown.

Disease cycle Disappearing root rot is characterized by concentrically expanding patches of wilting or dead plants in the garden. The pathogen can spread by conidia that form on the surface of rotted roots and are carried on clothing or machinery throughout the garden when machinery or workers move infested soil. Dense plant populations may allow direct plant-to-plant spread of the disease at points of root contact.

The fungus is believed to overwinter as thick-walled chlamydospores in soil or on infested plant residue. The disease occurs in a wide range of soil types. It has been reported to be less severe in soil with pH below 6.5. *Cylindrocarpon* is common in coniferous woodland soils.

Management

Cultural practices — High plant densities favor rapid plant-to-plant spread of the disease. A soil pH of less than 6.5 may limit the incidence of disappearing root rot.

Chemical control — Soil fumigation before planting may reduce levels of *Cylindrocarpon* inoculum.

Selected references

Bei, R.L., and Z.Q. Wang. 1986. Studies on the latent infection of the pathogen causing ginseng rootrot and its control. *Acta Phytopathol. Sinica* 16:41-46.

Booth, C. 1967. *Nectria radicicola.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 148. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Hildebrand, A.A. 1935. Root rot of ginseng in Ontario caused by members of the genus *Ramularia. Can. J. Res.* 12:82-114.

Matsuo, T., and Y. Miyazawa. 1984. Scientific name of *Cylindrocarpon* sp. causing root rot of ginseng. *Ann. Phytopathol. Soc. Jpn.* 50:649-652.

(Original by R.A. Brammall)

▶ 20.5 Phytophthora mildew and root rot *Fig. 20.5*

Phytophthora cactorum (Lebert & Cohn) J. Schrot.

Phytophthora mildew and root rot has occasionally caused epidemics in Ontario, British Columbia and the United States. All parts of the plant are affected and spread through a garden may be rapid. The disease tends to be more serious in older gardens.

Phytophthora cactorum is found on a number of different plants, most commonly in southern Ontario and British Columbia on fruit trees. However, it has not been found infecting any other plants or weeds in the vicinity of ginseng gardens.

Symptoms Phytophthora mildew produces a leaf spotting that is initially similar in appearance to that caused by *Alternaria* and *Botrytis.* Lesions are water-soaked and dark green, and the center later becomes whitish. Leaflets of diseased plants collapse downward from the base of the petiole and the stems become hollow and brown. Phytophthora lesions lack the yellow-brown margins associated with *Alternaria* infections or the gray surface mold associated with *Botrytis.*

Infection of the roots by *Phytophthora* causes a light brown discoloration of the surface. Symptoms often appear first on the crown and extend down the tap root or they may develop further down on the root system. The interior of rotted roots is creamy white with a soft texture *(20.5).* Squeezing infected roots by hand often causes the soft inner tissue to extrude like toothpaste from a tube. Broad, aseptate hyphae ramify throughout the affected tissues. Thick-walled oospores are common. The foul odor sometimes associated with *Phytophthora*-rotted roots is usually due to secondary colonizers.

Causal agent In northeastern North America, the disease is attributed to infection by *Phytophthora cactorum*, which produces broad, mostly aseptate hyphae. Septa may delimit sporangiophores from the mycelium. Airborne dispersal of zoosporangia has caused severe epidemics in the mid- western United States. In Korean ginseng, *Panax pseudoginseng* Wallich, oospores form in all infected tissues except the roots. Other fungi, including *Pythium* sp., are frequently isolated from affected roots.

In culture, hyphae vary from 2.5 to 5 µm in width but they appear to be wider in the host. The thick-walled oospores produced in culture are roughly spherical to elliptical and vary between 10 and 20 pm in diameter or axial length.

Disease cycle In the foliar mildew phase, zoosporangia produced on leaf lesions are spread by rain or wind to new plants where they establish new infections. The disease is favored by cool, wet or humid weather conditions. Phytophthora crown and root rot often is found where soils are excessively heavy and drainage is poor, or along the lower edges of the raised beds where water often pools. At such sites, diseased plants occur in groups. Zoospores are the probable means of plant-to-plant spread in the soil. Oospores are produced within the rotted tissues and are the likely overwintering stage.

Management

Cultural practices — Removal of affected plants has been recommended, but the practice is rarely practical. Growers should avoid planting in excessively heavy or poorly drained soils.

Selected references

Ohh, S.H., and C.S. Park. 1980. Studies on *Phytophthora* disease of *Panax ginseng* C.A. Meyer: its causal agent and possible control measure. *Korean J. Ginseng Sei.* 4:186-193.

Waterhouse, G.M., and J.M. Waterston. 1966. *Phytophthora cactorum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 111. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by R.A. Brammall)

▶ 20.6 Rusted root (rusty root) *Fig. 20.6*

Cylindrocarpon sp.

Rusted root of ginseng was first reported in New York State in 1909 and in southwestern Ontario in 1927. The disease causes a superficial, reddish scabbing of the root, downgrading its quality or causing it to be culled. Rusted root is a common problem and may be locally severe in some gardens.

Symptoms In early stages of infection, rusted roots display slightly raised, reddish-brown lesions ranging in size from minute flecks to enlarged areas that girdle the root *(20.6).* All ages of plant and regions of the root may be affected. The lesions give the root a rough appearance. In later stages of infection, the lesions grow together and may cover most of the root surface. The infection remains superficial. The periderm ruptures and sloughs in a manner similar to that seen in common scab of potato. The rusted tissue is easily scraped off the root to reveal healthy white tissue of the underlying cortex. Affected plants may be slightly stunted and mature earlier in the season than normal. They are not killed.

Causal agent Early work in New York suggested that *Chalara elegans* Nag Raj & Kendrick was the causal agent. Subsequent studies showed that *Ramularia* species *(Ramularia destructans* Zinnsmeister and *R. panicicola* Zinnsmeister) caused the disease in New York and Wisconsin. In Ontario, the cause of the disease is also thought to be a *Ramularia* species. A species of *Cylindrocarpon* has been associated with rusty root in British Columbia, and perithecia of *Nectria galligena* Bres. *in* Strass, have been observed on ginseng seed. The correct designation for the pathogen is likely *Cylindrocarpon destructans* (Zinnsmeister) Scholten (see disappearing root rot, 20.4). Rusted root and disappearing root rot are probably related disorders. Whether differences between these two diseases reflect soil, environmental, host or pathogen factors is unknown.

Disease cycle (see disappearing root rot, 20.4)

Management (see disappearing root rot)

Selected references

Hildebrand, A.A. 1935. Root rot of ginseng in Ontario caused by members of the genus *Ramularia. Can. J. Res.* 12:82-114.

(Original by R.A. Brammall)

▶ 20.7 Sclerotinia white rot *Fig. 15B.9T1*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia sclerotiorum (see Carrot, sclerotinia rot, 6.15) occasionally causes a stem and root rot of ginseng. Infected foliage wilts, discolors and dries up. Diseased roots show no discoloration but become soft and watery. Black sclero- tia often form on infected plant parts.

Management

Cultural practices — To manage this disease, growers should remove and destroy infected plants to reduce inoculum build-up, and avoid cultural practices that promote dense plant canopies, which favor white rot development.

Selected references

Mordue, J.E.M., and P. Holliday. 1976. *Sclerotinia sclerotiorum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 513. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by L.S. MacDonald and R.J. Howard)

▶ 20.8 Verticillium wilt

Verticillium dahliae Kleb.

Verticillium wilt generally affects ginseng plants in older gardens. The disease has been reported from the northern United States but not in Canada. Early publications on diseases of ginseng refer to this disease as acrostalagmus wilt, a name that has been retained in error in some popular publications. Verticillium wilt may have declined in importance with the adoption of relatively short, three to four year cropping cycles. *Verticillium dahliae* has a wide host range and may persist on symptomless weed hosts.

Symptoms Affected plants display wilting of the foliage.

In diseased plants, the leaves wilt and droop parallel to the stem. This wilting eventually kills the plant. Roots are firm but the vascular tissue is conspicuously discolored yellow. Microscopic examination of root cross-sections reveals the vessels to be colonized by fungal hyphae.

Causal agent (see Potato, verticillium wilt, 16.20)

Disease cycle (see Potato, verticillium wilt, 16.20) *Verticillium dahliae* overwinters as microsclerotia in infected plant tissues. The fungus infects ginseng by penetrating into the vascular tissue at the sites of leaf scars. The fungus likely can also penetrate

roots directly. It grows within and spreads through the xylem vessels. Microsclerotia form in tissues that have been killed by the fungus. The optimum temperature for verticillium wilt is generally below 20°C. Symptoms may not appear until later in the season when the plants become senescent.

Management

Cultural practices — Removal of affected plants may reduce inoculum in the garden. Infested soil may be moved throughout a garden on machinery, so steps should be taken to disinfest it between plots.

Chemical control — Soil fumigation before planting may reduce levels of *Verticillium* inoculum.

Selected references

Hawkesworth, D.L., and P.W. Talboys. 1970. *Verticillium dahliae.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 111. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by R.A. Brammall)

NON-INFECTIOUS DISEASES

▶ 20.9 Nutritional and other disorders *Figs. 20.9a-d*

Zinc deficiency
Phytotoxicity
Sunscald

Zinc deficiency causes interveinal yellowing *(20.9a).* In severe cases, the interveinal areas become white while the large veins remain dark green.

Foliar applications of zinc are effective but can be toxic except in very low amounts. Growers are advised to have the soil tested for adequate levels of plant nutrients before planting.

Phytotoxicity Mancozeb fungicide applied at the full label rate during hot weather has caused red ring spots and necrotic blotches *(20.9b)* on the leaves of ginseng seedlings in British Columbia. Mancozeb toxicity has not been observed in Ontario, but anilazine has sometimes caused damage when applied at greater than label rates during hot weather.

Growers in British Columbia routinely use half the label rate or less of mancozeb on one-year plants to reduce phytotoxicity. To minimize fungicide toxicity *(20.9c),* growers should be cautious about applying these fungicides when air temperatures exceed 30°C.

Sunscald Ginseng is a shade-loving herb that grows naturally only where there is a minimum of 70% shade. The leaves are very easily damaged by high intensity light, so cultivated ginseng must be grown under shaded conditions that match its native habitat in the forest understory. Direct sunlight burns leaf tissues, causing them to wilt, turn brown and die *(20.9d).* The proper use of shading materials will prevent this type of damage from happening.

(Original by L.S. MacDonald and R.J. Howard)

NEMATODE PESTS

▶ 20.10 Northern root-knot nematode

Meloidogyne hapla Chitwood

Symptoms Mature roots may be deformed, short and branched, and secondary roots abnormally branched and hairy. For a complete description and management strategies, see Carrot, 6.20; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 20.11 Miscellaneous insect pests

Cutworms
Thrips
Weevils
Wireworms

Cutworms were first documented in ginseng plantations in Canada in May and June, 1989, in Norfolk County in southern Ontario.

Thrips damage to flower heads was first documented on ginseng in Canada in 1989, also in Norfolk County, southern Ontario.

Weevils have been a problem at a garden on Vancouver Island, British Columbia.

Wireworms have caused stem and root damage in British Columbia.

Management

Chemical control — Insecticides have not been registered in Canada for control of these pests on ginseng.

Selected references

Cheng, H.H. 1990. Insects and related pests of miscellaneous crops/Les ravageurs de cultures diverses. Ginseng. Page 48 *in The Canadian Agricultural Insect Pest Review/La revue canadienne des insectes nuisibles aux cultures.* Vol. 67 (1989). 90 pp.

(Original by H.H. Cheng, B.F. Zilkey and L.S. MacDonald)

OTHER PESTS

▶ 20.12 Slugs *Figs. 11.27a-c*

Many growers attempt to control slugs in Ontario gardens, according to R.A. Brammall. For information about slugs, see Crucifers, 8.49; Lettuce, 11.27.

ADDITIONAL REFERENCES

Duke, J.A. 1989. *Ginseng: A Concise Handbook.* Reference Publ. Inc., Algonac, Michigan. 273 pp.

Ohh, S.H. 1981. Diseases of ginseng: environmental and host effect on disease outbreak and growth of pathogens. *Korean J. Ginseng Sci.* 5:73-84.

Savage, J. 1991. American ginseng culture in the arid climates of British Columbia. *Korean J. Ginseng Sci.* 15:41-73.

Van Hook, J.M. 1904. Diseases of ginseng. *Cornell Univ. Agric. Exp. Stn. Bull.* 219:167-186.

Whetzel, H.H., and J. Rosenbaum. 1912. *Diseases of Ginseng and Their Control.* U.S. Bureau Plant Industry Bull. 250. 44 pp.

21 Jerusalem artichoke

BACTERIAL DISEASES

▶ 21.1 Apical chlorosis *Figs. 21.1a,b*

Pseudomonas syringae pv. *tagetis* (Hellmers) Young, Dye & Wilkie
(syn. *Pseudomonas tagetis* Hellmers)

Apical chlorosis has been a severe problem in Jerusalem artichoke in parts of the United States. In Canada, it has been reported from Quebec, Ontario and Manitoba. The disease can reduce plant stands by as much as 50%. Sunflower (*Helianthus annuus* L.), marigold (*Tagetes* spp.) and zinnia are other hosts for the pathogen.

Symptoms Newly emerged shoots exhibit a yellowing of the growing tips (apical chlorosis) which can spread downward over most of the shoot *(21.1a).* Affected leaves are yellow to white and, in time, turn brown. Diseased shoots are stunted and usually fail to survive *(21.1b).* Other foliar symptoms include brown leaf spots, varying from 1 to 2 mm in diameter, surrounded by a faint yellow halo, or large, yellow-green spots with small patches of gray-colored tissue in the center. Droplets of bacteria may ooze from these spots.

Causal agent *Pseudomonas syringae* pv. *tagetis* is a Gram-negative, oxidase-negative, arginine dihydrolase-negative, aerobic rod. Two strains of the bacterium have been reported, based on differences in symptoms that develop on spray-inoculated Jerusalem artichoke. "CN" strains produce chlorosis and necrotic spots on the host, while "N" strains cause only small, 1 to 3 mm, necrotic spots.

The pathogen may be isolated from necrotic foliage or from infested tubers by surface sterilizing the tissues in sodium hypochlorite, transferring treated pieces to sterile distilled water to allow the bacteria to ooze out, then streaking the water onto either nutrient agar or King's B medium. On these media, the pathogen produces colonies with slightly irregular margins. Flat colonies with highly irregular margins may be produced by some isolates upon repeated subculturing. The pathogen produces a fluorescent diffusate when grown on King's B medium.

Pathogenicity testing may be done by spraying the isolated bacteria, at a concentration of 5×10^6 cells/mL, onto the foliage of test plants, followed by an additional misting of the foliage for two days. Symptoms of foliar chlorosis and/or necrosis develop on Jerusalem artichoke, sunflower and marigold within two weeks.

Disease cycle The pathogen is tuber-borne in Jerusalem artichoke. The shoots that emerge from infested tubers usually become diseased. When necrotic tissue becomes wet, the bacterial cells are liberated and, under favorable conditions for infection, they promote secondary spread of the disease in the field. Apical chlorosis is less severe when infection occurs in older plants and they may eventually outgrow it. The disease also occurs in ragweed *(Ambrosia artemisiifolia* L.). Infection of ragweed and other weedy hosts within the family Asteraceae may provide natural reservoirs of inoculum. In sunflower, *P. syringae* pv. *tagetis* is seed-borne, but it is not known if this type of transmission occurs in Jerusalem artichoke.

Management

Cultural practices — Seed stock tubers should be obtained only from fields free from apical chlorosis. It is important to check such fields for the disease before flowering because the symptoms may become less conspicuous as the plants mature.

Selected references

Gulya, T.J., R. Urs and E.E. Banttari. 1982. Apical chlorosis of sunflower caused by *Pseudomonas syringae* pv. *tagetis. Plant Dis.* 66:598-600.
Laberge, C., and W.E. Sackston. 1986. Apical chlorosis of Jerusalem artichoke (*Helianthus tuberosus). Phytoprotection* 67: 117-122.
Shane, W.W., and J.S. Baumer. 1984. Apical chlorosis and leaf spot of Jerusalem artichoke incited by *Pseudomonas syringae* pv. *tagetis. Plant Dis.* 68:257-260.
Styer, D.J., and R.D. Durbin. 1982. Common ragweed: a new host of *Pseudomonas syringae* pv. *tagetis. Plant Dis.* 66:71.

(Original by R.A. Brammall)

FUNGAL DISEASES

▶ 21.2 Downy mildew *Fig. 21.2*

Plasmopara halstedii (Farl.) Berl. & De Toni in Sacc.

This disease *(21.2)* is a minor problem in Jerusalem artichoke; it has been noted by W.E. Sackston (unpublished) in the field in Manitoba and in a disease nursery at Sainte- Anne-de-Bellevue, Quebec, that was infested with soil from a sunflower field at La Pocatière, Quebec. *Plasmopara halstedii* is a soil-borne fungus that also affects other members of the Asteraceae, including sunflower.

(Original by W.L. Seaman and W.E. Sackston)

▶ 21.3 Powdery mildew *Fig. 21.3*

Erysiphe cichoracearum DC.:Mérat

Powdery mildew affects the mature foliage of Jerusalem artichoke. The economic consequences of the disease are unknown; heavy infestations likely cause a reduction in yield and plant vigor. The disease has been reported from Quebec, Ontario and Manitoba. *Erysiphe cichoracearum* is capable of infecting a variety of cultivated and wild Asteraceae, as well as lettuce, cucurbits and several herbs and spices.

Symptoms Powdery mildew appears as a powdery white growth on the surface of the stems and leaves *(21.3).* This growth consists of the spores and mycelium of the causal fungus. Initially, the mildew lesions are discrete, but later enlarge, merge and may cover much of the foliage. Affected tissues turn yellow and eventually die.

Causal agent (see Lettuce, powdery mildew, 11.12)

Disease cycle The pathogen is an obligate parasite. Conidia, which are formed on the infected tissue, are dispersed by wind to cause new infections throughout the season. With the approach of autumn, the fungus produces cleistothecia on the older mycelium, generally on the upper surface of infected leaves. In spring, the cleistothecia split open when wetted and the ascospores are forcibly discharged. Those that land on leaves and stems of Jerusalem artichoke and other hosts may germinate and infect these plants.

Management

Cultural practices — Mildew-susceptible weeds should be controlled in the vicinity of Jerusalem artichoke plantings. Destruction of residues from infected crops may help to reduce the amount of disease the following year.

Resistant cultivars — Certain cultivars of Jerusalem artichoke differ in susceptibility to powdery mildew. Late- maturing cultivars may be less severely affected than early ones.

Selected references

Kapoor, J.N. 1967. *Erysiphe cichoracearum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 152. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Laberge, C., and W.E. Sackston. 1987. Adaptability and diseases of Jerusalem artichoke (*Helianthus tuberosus)* in Quebec. *Can. J. Plant Sei.* 67:349-352.

Lorenzini, G., and E. Triolo. 1980. Sunflower and Jerusalem artichoke, new hosts of *Erysiphe cichoracearum* DC. in Italy. *Informatore Fitopatologico* 30:9-11.

McCarter, S.M., and S.J. Kays. 1984. Diseases limiting production of Jerusalem artichokes in Georgia. *Plant Dis.* 68:299-302.

(Original by R.A. Brammall)

▶ 21.4 Rust *Fig. 21.4*

Puccinia helianthi Schwein.

Rust has been a serious disease of Jerusalem artichoke in the southeastern United States. In Canada, it is a minor problem on Jerusalem artichoke in Ontario and Quebec; however, *Puccinia helianthi* is a serious pathogen of cultivated sunflower *(Helianthus annuus* L.) in Manitoba. Rust also affects other *Helianthus* spp. and occurs throughout the range of these species. Rust is one of the most important diseases affecting cultivated *Helianthus* spp. throughout the world.

Symptoms The first symptom of rust usually noted is the production of uredinial pustules on the foliage and, occasionally, on the stem. Reddish-brown urediniospores form within the uredinia during mid- to late summer. Young foliage may be affected and fail to grow because of heavy infections. The uredinia are most frequently found on the undersurface of the leaves. At the end of the growing season, the uredinia turn black as teliospores are produced *(21.4).*

Causal agent *Puccinia helianthi* uredia are mostly hypophyllous, irregularly scattered, cinnamon, and 0.5 to 1.0 µm in diameter. Urediniospores are ellipsoidal, obovoidal or cylindrical, 25 to 32 by 19 to 25 µm, very finely echinulate, and reddish brown. The

telia resemble the uredinia, but are darker colored. The teliospores are cylindrical to clavate, slightly constricted at the septum, 40 to 60 by 18 to 30 µm, and reddish to chestnut brown.

Disease cycle *Puccinia helianthi* is a macrocyclic, autoe- cious rust. Teliospores are formed and overwinter on host residues. They germinate in the spring to produce sporidia, which can infect the young plants. Sporidial fusions result in the production of pycnia and aecia (see Asparagus, rust, 4.6). These spore stages are often inconspicuous. Infection by the aeciospores results in the production of urediniospores in the uredinia. The urediniospores are dispersed during the summer to cause new infections. In autumn, the uredinia cease to be formed and overwintering teliospores are produced. Volunteer Jerusalem artichoke plants and wild sunflowers may serve as the initial source of infection during the growing season.

Management

Cultural practices — Destruction of infested crop residues will reduce the amount of overwintering inoculum. Crop rotation or the selection of new sites for each successive planting may also help to limit disease development.

Resistant cultivars — No rust-resistant cultivars of Jerusalem artichoke have been developed, but resistance genes for specific races of *P. helianthi* have been identified in this crop.

Selected references

Laundon, G.F., and J.M. Waterston. 1965. *Puccinia helianthi.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 55. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Putt, E.D., and W.E. Sackston. 1957. Studies on sunflower rust. I. Some sources of rust resistance. *Can. J. Plant Sci.* 37:43-54.

Zimmer, D.E., and D. Rehder. 1976. Rust resistance of wild *Helianthus* species of the north central United States. *Phytopathology* 66:208-211.

(Original by R.A. Brammall)

▶ 21.5 Sclerotinia wilt, stalk and tuber rot *Figs. 21.5a, b*

Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Sclerotinia wilt and stalk and tuber rot have been reported on Jerusalem artichoke in Quebec, Ontario, Manitoba and British Columbia. The pathogen causes similar diseases on cultivated sunflower (*Helianthus annuus* L.). *Sclerotinia sclerotiorum* has a wide host range, which includes many vegetable crops and broadleaved weeds. The ubiquitous nature of the pathogen means that wilt and stalk and tuber rot will likely occur, to some extent, wherever Jerusalem artichoke is grown.

Symptoms *Sclerotinia* infection produces basal cankers, root rot, tuber rot, and wilt symptoms *(21.5a).* The root system and tubers may be destroyed by the disease. Rotting may extend up the stem above the soil line *(21.5b).* Dense white mycelium and black sclerotia often form upon and within the affected tissues (see Carrot, sclerotinia rot).

Causal agent (see Carrot, sclerotinia rot, 6.15)

Disease cycle The pathogen persists in soil as sclerotia (see Carrot, sclerotinia rot). Wilt and root and basal stem rot of sunflower are caused by penetration of the roots and stems by hyphae from germinating sclerotia. Head rot can be caused by air-borne ascospores released from apothecia produced by sclerotia in the soil. The infection process in Jerusalem artichoke is probably similar to that in sunflower, but this remains to be determined.

Management

Cultural practices — Growers should select fields that do not have a history of sclerotinia diseases. Tubers used for planting should be free of the pathogen. Cereals, corn or grasses should be used in crop rotations and at least four years should be allowed between successive crops of Jerusalem artichoke and other susceptible species. Volunteer Jerusalem artichoke plants and susceptible weeds should be controlled in these rotational crops.

Selected references

Huang, H.C., and J. Dueck. 1980. Wilt of sunflower from infection by mycelial-germinating sclerotia of *Sclerotinia sclerotiorum. Can. J. Plant Pathol.* 2:47-52.

Mordue, J.E.M., and P. Holliday. 1976. *Sclerotinia sclerotiorum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 513. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by R.A. Brammall)

INSECT PESTS

▶ 21.6 Stem borers *Fig. 21.6*

In western Quebec some years ago, W.E. Sackston found a stem borer identified as the sunflower maggot *Strauzia longipennis* (Wiedemann) (see Additional references, Westdal and Barrett 1960), which burrows in the stem, overwinters in the crown as a

pupa, and occurs in Canada from Manitoba east; and A.J. Kolach found a stem borer in Manitoba that tentatively was assigned to *Eucosma* sp.

ADDITIONAL REFERENCES

Gulya, T.J., and S. Masirevic. 1991. Common names for plant diseases: sunflower (*Helianthus annuus* L.) and Jerusalem artichoke *(H. tuberosus* L.). *Plant Dis.* 75:230.

Kiehn, F.A., and M. Reimer. 1992. Alternative crops for the prairies. Agric. Can. Publ. 1887/E. 46 pp.

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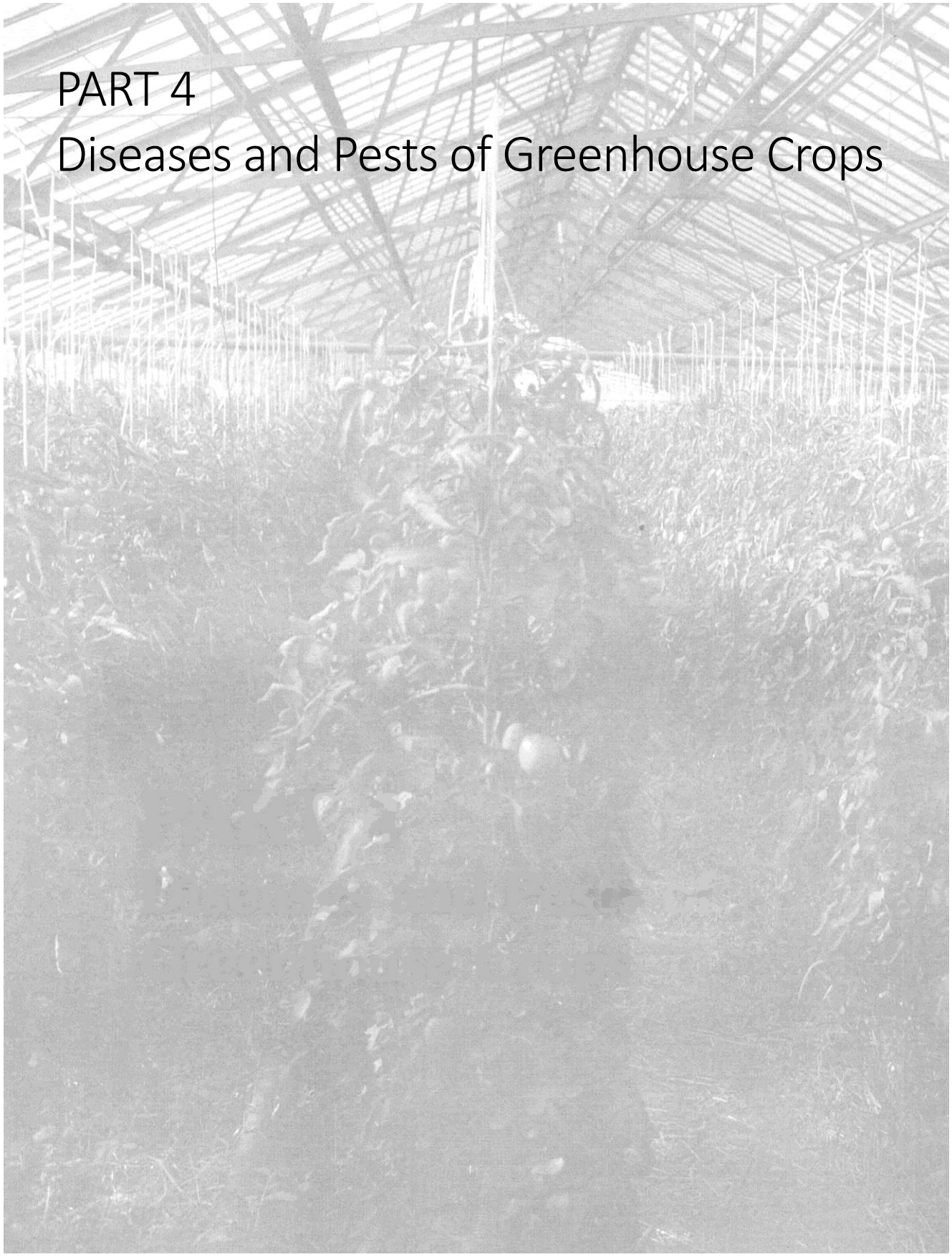
Westdal, P.H., and C.F. Barrett. 1960. Life-history and habits of the sunflower maggot, *Strauzia longipennis* (Wied.) (Diptera: Trypetidae), in Manitoba. *Can. Entomol.* 92:481-488.



PART 4
Diseases and Pests of Greenhouse Crops

Locating Text Sections and Figures

Text sections are numbered consecutively within each chapter. For example, section 16.2 describes bacterial soft rot, the second topic of Chapter 16, Potato. To find a text section, refer to the running heads, which carry the inclusive section numbers for each two-page spread.

Color illustrations, grouped near the back of the book, appear in the same order and have the same number as the corresponding text section; for example, figures *16.2a* and *16.2b* illustrate the symptoms of bacterial soft rot of potato. Line drawings, halftones and tables are numbered similarly, except that a text figure number contains the letter T; for example, Figure *16.2T1* illustrates the disease cycle of bacterial soft rot of potato.

22 Greenhouse cucumber

Figures *22.1* to *22.36; 22.15T1; 22.31T1; 22.36T1*

Bacterial diseases

22.1 Angular leaf spot
22.2 Bacterial wilt

Fungal diseases

22.3 Anthracnose
22.4 Black root rot
22.5 Black rot
22.6 Choanephora rot
22.7 Crown and root rot damping-off
22.8 Downy mildew
22.9 Fusarium wilt
22.10 Gray mold
22.11 Gummy stem blight
22.12 Leaf blights
- Alternaria leaf blight
- Ulocladium leaf spot

22.13 Leaf rot (pink mold rot)
22.14 Penicillium stem rot
22.15 Powdery mildew
22.16 Scab (gummosis)
22.17 Verticillium wilt
22.18 White mold (sclerotinia stem rot)

Viral diseases

22.19 Beet pseudo-yellows
22.20 Cucumber mosaic
22.21 Cucumber necrosis
22.22 Cucumber pale fruit
22.23 Watermelon mosaic
22.24 Zucchini yellow mosaic

Non-infectious diseases

22.25 Chilling injury, cold injury
22.26 Nutritional disorders
- Boron
- Calcium
- Copper

22.26 Nutritional disorders (cont.)
- Iron
- Magnesium
- Manganese
- Molybdenum
- Nitrogen
- Phosphorus
- Potassium

22.27 Premature fruit yellowing
22.28 Root death
22.29 Sudden wilting

Nematode pests

22.30 Root-knot nematodes
- Northern root-knot nematode
- Southern root-knot nematodes

Insect pests

22.31 Fungus gnats
22.32 Greenhouse whitefly
22.33 Melon (cotton) aphid
22.34 Western flower thrips
22.35 Other insect pests
- Caterpillars
- Cucumber beetles
- Spotted cucumber beetle
- Striped cucumber beetle
- Leafminers
- Chrysanthemum leafminer
- Vegetable leafminer
- Onion thrips
- Plant bugs

Mite pests

22.36 Two-spotted spider mite
Additional references

BACTERIAL DISEASES

▶ 22.1 Angular leaf spot *Figs. 22.1; 9.1a,b*

Pseudomonas syringae pv. *lachrymans* (Smith & Bryan) Young *et al.*

Angular leaf spot (see Cucurbits, angular leaf spot, 9.1) is rare in greenhouse cucumber and has been seen only where affected field cucumber crops have been handled by green house workers. It is likely to be a problem only in poorly ventilated greenhouses with overhead irrigation or excessive condensation.

Management

Cultural practices — Pathogen-free seed should be used for planting. Overhead irrigation should be avoided and relative humidity kept low. Leaf injury should be minimized and crops should not be worked in if the foliage or fruit is wet.

Selected references

Kritzman, G., and D. Zutra. 1983. Systemic movement of *Pseudomonas syringae* pv. *lachrymans* in the stem, leaves, fruits and seeds of cucumber. *Can. J. Plant Pathol.* 5: 273-278.

(Original by W.R. Jarvis and J.G. Menzies)

▶ 22.2 Bacterial wilt *Figs. 9.2a,b*

Erwinia tracheiphila (Smith) Bergey *et al.*

Bacterial wilt (see Cucurbits, bacterial wilt, 9.2) is occasionally a problem on greenhouse cucumber.

Management

Cultural practices — The disease can be controlled in greenhouses by placing screens on ventilators and doors to prevent entry of cucumber beetles, which vector the pathogen, and by roguing diseased plants as soon as possible. Raising the temperature briefly above 30°C activates the defense mechanisms of the host and helps to control the disease.

(Original by J.G. Menzies and W.R. Jarvis)

FUNGAL DISEASES

▶ 22.3 Anthracnose *Figs. 9.3a-c*

Colletotrichum orbiculare (Berk. & Mont.) Arx
(syn. *Colletotrichum lagenarium* (Pass.) Ellis & Halst.)
(teleomorph *Glomerella lagenaria* F. Stevens)

Anthracnose is a minor disease of greenhouse cucumber (see Cucurbits, anthracnose, 9.3).

Management

Cultural practices — Anthracnose can be controlled by reducing high relative humidity through ventilation and heating, and by avoiding overhead irrigation. Crops should not be worked if the foliage is wet. Greenhouses should be thoroughly cleaned after a diseased crop has been removed.

(Original by J.G. Menzies)

▶ 22.4 Black root rot *Figs. 22.4a,b*

Phomopsis sclerotioides van Kesteren

Black root rot is generally a minor disease of greenhouse cucumber, but it has caused yield losses of up to 50% in a few greenhouses in British Columbia. This disease can affect plants grown in soil, rockwool and other soilless media. It is more prevalent in operations where crop hygiene is poor The host range of the pathogen is restricted to members of the Cucurbitaceae family.

Symptoms Roots develop pale brown areas which darken and eventually turn black as the disease progresses *(22.4a).* These areas may be sunken and bordered by darker areas. Coalescence eventually occurs, with diseased tissue demarcated from healthy tissue by a fine wavy black line. Close examination of diseased roots with a 10X hand lens will also reveal a second symptom, a definite mosaic, almost like a chess-board pattern, of small black sclerotia *(22.4b)* on dark mycelium. The roots may be girdled and killed.

The cortical tissue of the roots eventually sloughs off, exposing the vascular strands. The expression of symptoms in the aerial parts of the plant is determined by the degree of root infection and by environmental conditions. Stems may become infected at the base as the fungus grows from diseased roots. Stem lesions are elongate and brown or black, with amber gummy exudations. Infected plants are stunted with few lateral shoots and small, downward- cupped leaves. The leaves, initially darker green than usual, may also exhibit chlorosis and necrosis. Partially developed fruit will not mature. An irreversible wilt often occurs when fruiting begins. The disease is more severe in cool, overwet soils and substrates where root growth is suboptimal. It is often seen in soil where straw mulch has been put down prematurely, thereby insulating cold soil.

Causal agent *Phomopsis sclerotioides* is readily isolated from surface- sterilized incipient lesions on roots. Pycnidia, though very rare on roots, are subglobose to variable in shape, stromatic, and up to 300 pm wide. Conidiophores are hyaline and simple or rarely branched. The conidio- genous cells are enteroblastic, phialidic, simple, and cylindrical to subob- clavate. They produce A-conidia, which are hyaline, unicellular, fusiform to ellipsoid, usually guttulate with a guttule at each end, and measure 7 to 10 by 2.5 to 3.5 pm. On sterile bean pods, B-conidia are also produced, but rarely. In culture, the fungus produces abundant sclerotial plates.

Confirmation of the disease requires culturing and identification of conidia. *Phomopsis sclerotioides* produces pseudosclerotia, whereas *Phomopsis cucurbitae,* which causes black rot, produces A- and B-conidia but no sclerotia. Media suitable for sporulation include cherry agar and sterile green bean pods. The fungus is fast-growing on malt agar, at first sparse and off-white, then darkening with age.

Disease cycle The pathogen survives and is transmitted in soil, usually in infected plant residues. It grows quickly through soil and rapidly colonizes plant roots. Rockwool and other soilless substrates can become infested through soil contamination, such as by placing transplant blocks on the ground or by allowing soil-contaminated water to splash or flood onto them.

Management

Cultural practices — Annual steam pasteurization of infested soil and the use of non-infested or soilless rooting media can help in disease prevention. It is important not to allow infested soil to contaminate soilless media. Seedlings and transplants should be raised on clean benches, well out of reach of splashing soil and flooding. If the disease is detected early enough by wilt symptoms that go into remission at night and on cloudy days, the stem base may be mounded up with clean, peaty soil. This promotes the growth of adventitious roots that permit the plant to survive and yield perhaps 80% or more of its potential. Grafting onto gourd rootstocks *(Cucurbita ficifolia* Bouché) helps maintain crop yields, but in heavily infested soil gourd rootstocks can become infected.

Chemical control — Soil fumigation may provide some control if done to effective depths.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.5 Black rot

Phomopsis cucurbitae McKeen

Black rot is an uncommon fungal disease of greenhouse cucumber and has not been observed in the field in Canada. It has been reported only from Ontario and British Columbia. This disease has been found only on cucurbits.

Symptoms Black rot occurs on the stems, leaves, petioles, peduncles and fruit of cucurbits. Infections first appear as water-soaked, oily green areas on senescent and moribund tendrils, fruit stems, petioles and suckers that arise at the stem nodes. Amber-colored gummy exudations accompany nodal infections. Nodal lesions can spread both up and down the stem. They are initially superficial but eventually penetrate the vascular tissue and girdle the stem, resulting in the death of plant tissues above the lesion. Fruit may be infected via the flowers, and the flesh quickly becomes soft, rotted and water- soaked. Finally, the fruit shrinks, becomes mummified and emits a lemon-like odor. On all affected tissues, tiny, black, spore-bearing pycnidia of the fungus break through the outer epidermis, often in long, parallel rows on blanched, tattered, dry outer tissues. If seed is sown in artificially infested soil, lesions develop on the cotyledons and hypocotyl. The lesions are initially pale beige but become blackened with pycnidia. Roots are pale brown and are soft and spongy.

Symptoms of black rot can resemble those of gummy stem blight and distinguishing between them may require microscopic observations or culturing of the pathogens.

Causal agent *Phomopsis cucurbitae* produces dark brown to black pycnidia that are variable in shape, stromatic, and as much as 1 mm wide. Conidiophores are hyaline, and simple or branched. Conidiogenous cells are enteroblastic, phialidic and simple cylindrical to subobclavate. Conidia are of two types: A-conidia are hyaline, unicellular, fusiform to ellipsoid, usually two-guttulate with a guttule at each end, sometimes three-gut- tulate, and measure 8 to 12 by 2.5 to 3 µm; B-conidia are hyaline, unicellular, filiform, curved, and measure 18 to 26 by 1 µm.

Phomopsis sclerotioides, which causes black root rot, superficially resembles *P. cucurbitae* but produces mostly A-conidia and sclerotia (see black root rot, 22.4).

Disease cycle Infections often occur first on dead and dying tendrils, peduncles, petioles and suckers arising from stem nodes. These infections move to the stem, girdle it and cause death of tissues above the lesion. Fruit can be infected via attached flowers. The method of survival between crops is unknown, but the fungus is unlikely to be seed- borne. The disease progresses quickly on succulent plants and is favored by high humidity. Because the fungus produces sticky, hydrophilic spores in long tendrils from the pycnidia, spread occurs mainly by splashing water and on tools and fingers.

Management

Cultural practices — Spread of this disease may be prevented by good ventilation, which promotes rapid drying of senescing plant parts and prevents establishment of the fungus. Removal of infested crop residues and a thorough cleaning of the greenhouse after crop removal helps prevent spread and survival of the pathogen.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.6 Choanephora rot

Choanephora cucurbitarum (Berk. & Ravenel) Thaxt.

Choanephora rot (see Cucurbits, choanephora rot, 9.4) is a minor disease of greenhouse cucumber.

Management

Cultural practices — Choanephora rot can be controlled by avoiding the use of overhead irrigation and by not working in the crop when the foliage is wet. Greenhouses should be thoroughly cleansed after a diseased crop has been removed.

(Original by J.G. Menzies)

▶ 22.7 Crown and root rot, damping-off *Figs. 22.7a-d*

Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)
Fusarium spp.
Bacteria

Pythium species appear to be the primary cause of damping-off and crown and root rot, but other fungi and soft-rotting bacteria are occasionally associated with plants affected by these diseases. The pathogens that cause these diseases have wide host ranges that include many types of vegetable crops.

Symptoms Plants affected by crown and root rot usually are noticed when they suddenly wilt (22.*7d),* particularly during warm, sunny weather. *Pythium* causes an orange-brown rot of the crown, which may extend 8 to 10 cm up the stem *(22.7a,c).* Stem lesions appear chlorotic and yellow-white initially. As the disease progresses, the lesions turn brown- orange. Affected tissues usually appear dry rather than water- soaked, and infected plants produce few lateral roots from the crown region. Severely affected plants are weakly anchored and can be easily lifted out of the growing medium. Plants may recover if not severely diseased, but prolonged wilting leads to plant death. Mature crops can also wilt suddenly because of destruction of tiny feeder roots by *Pythium* spp. There may be no other symptom. See also 22.29.

Symptoms of damping-off include a reduction in seedling emergence and toppling over of young seedlings

(22.7b) (see Cucurbits, pythium root rot, 9.12). Root and stem lesions on young seedlings are pale brown and appear water-soaked.

Causal agents Several species of *Pythium* (see Cucurbits, 9.11, 9.12) may be involved in damping-off and crown and root rot. Sporangia are various sizes and shapes and intercalary or terminal on the mycelium. They may germinate via a germ tube or via a short germ tube and terminal vesicle within which zoospores are produced. The oogonia have a single oospore at maturity and

germinate directly or produce zoospores. These spores are released through a pore or evacuation tube into a vesicle where they mature before being liberated.

Fusarium solani and *F. oxysporum* (see Cucurbits, fusarium foot rot, 9.5, and fusarium wilt, 9.6) are frequently isolated from the rotting tissues at the base of the stem but their etiologic role is uncertain. Bacteria are also profusely evident and undoubtedly contribute to soft rotting (see Tomato, fusarium crown and root rot, 18.9).

Rhizoctonia solani (see Bean, 15B.7) tends to attack older seedlings, causing late damping-off.

Disease cycle The fungi that cause crown and root rot and damping-off are common in propagation mixes, soil and untreated water. Symptoms of crown and root rot typically appear about 8 to 12 weeks after seeding, at early fruit set, or during the late season on older plants. Plants rarely show symptoms before being transplanted. Severe infections are often associated with stresses such as high temperatures and excess moisture. Damping-off pathogens can spread quickly in cool, wet growing media, especially soil. Infection and pathogen spread are aided by excess nitrogen and overcrowding of the plants. Spread of the pathogens in the greenhouse can occur through irrigation water. *Pythium* spp. also can be vectored by fungus gnat larvae and shore flies. Both water and flies can explain the damaging appearance of pythium root rots in hydroponic greenhouses. In addition, soilless media are often contaminated by soil splash, flooding, or simply by putting planting blocks on the floor. Hanging baskets of ornamentals are also sources of inoculum.

Management (see fungus gnats, 22.31, for their control)

Cultural practices — For damping-off (see Cucurbits, pythium root rot, 9.12), seedlings should not be overcrowded. Adequate ventilation helps to keep the growing media and foliage dry. Seed flats should be raised and placed out of the range of splashing water. Pathogen-free growing media should be used to produce transplants and for the main crop. NFT troughs, tanks and supply lines should be disinfested. Good drainage and avoidance of overwatering help to control damping-off and crown and root rot. Plants severely infected early in the season can be rogued and the area replanted with disease-free transplants. Sawdust mounded around the base of infected plants encourages adventitious root growth and extends plant survival a few weeks. The amendment of hydroponic nutrient solution with 100 ppm of soluble silica has been reported to reduce crown and root rot.

Chemical control — Hot water seed treatment followed by the application of a fungicidal seed protectant is recommended for controlling seed- and soil-borne damping-off fungi. Seedling trays can be drenched with fungicidal solutions to provide additional protection. Fungicide seed treatments are ineffective against crown and root rot.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.8 Downy mildew *Figs. 22.8a,b*

Pseudoperonospora cubensis (Berk. & M.A. Curtis) Rostovzer

This disease is common and often very destructive in end- of-season crops in poorly ventilated plastic greenhouses. It occurs throughout Canada but has been a problem mostly in Ontario. The pathogen attacks only Cucurbitaceae, mostly species that are cultivated, but it can occur on a few wild hosts, including wild cucumber.

Symptoms Downy mildew infections occur only on the leaves. Initial symptoms include angular, pale green areas separated by dark green islands on the upper surfaces of the leaves which may resemble mosaic mottle *(22.8a).* The pale-green areas eventually turn into the characteristic yellow, angular spots of downy mildew, bordered by the leaf veins. The lesions are brown on the lower surface of the leaves *(22.8b).* Under humid conditions, the lesions become covered with purple-brown sporangia as sporangiophores emerge in groups of one to five from stomata on the underside of the leaf. The leaf may wither and die as the lesions increase in size.The fruit is seldom infected by the pathogen but may be small and of poor quality as the result of leaf destruction.

Causal agent *Pseudoperonospora cubensis* is an obligate parasite. Sporangiophores are 180 to 400 µm long by 5 to 7 µm wide. They are basally inflated and dichotomously branched in the upper third. The sporiferous tips are subacute and bear single sporangia that are pale gray to olive-purple, ovoid to ellipsoid, thin walled, and have a papilla at the distal end. The sporangia measure 20 to 40 by 14 to 25 µm. Flagellate zoospores, 10 to 13 µm in diameter, are produced by germination of the sporangia. Oospores are rare, but if produced are light yellow or hyaline, globose, and measure 22 to 42 µm in diameter.

Disease cycle The fungus does not live in soil, but may overwinter in some areas as thick-walled oospores that can withstand low temperatures and humidities in the field or greenhouse. Oospores are unlikely to overwinter in the field in northern areas. Rather,

the fungus probably arrives as wind-blown sporangia from the south. Sporangia are produced four to five days after infection and may be spread by wind, insects, clothing and tools to neighboring plants. Surface water on the foliage is essential for infection to occur. Once wet, the sporangia must remain so until they germinate, otherwise they die. Germinating sporangia give rise to motile zoospores that produce infective germ tubes to achieve infection. Sporangia germinate from 8 to 30°C, with the optimum being 15 to 20°C. Infection occurs over a temperature range of 16 to 22°C.

Management

Cultural practices — Infected leaves should be removed as soon as symptoms are noticed, and growers should destroy trash piles by composting or burial. Measures to promote air movement and reduce relative humidity, such as wider plant spacing, help to control the spread of the pathogen. Control of cucurbit weeds and volunteer hosts that harbor the pathogen also will reduce its spread. Growers should adjust ventilation and heating so that dew does not form on the plants and avoid the use of overhead irrigation.

Resistant cultivars — Some resistant cultivars are known, but they may be of poor quality and should be evaluated on a trial basis by growers.

Chemical control — Protectant fungicides will control the disease if applied on a preventive basis.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.9 Fusarium wilt *Figs. 22.9a,b*

Fusarium oxysporum f. sp. *cucurbitacearum* Gerlagh & Blok (syn. *Fusarium oxysporum* f. sp. *cucumerinum* Owen)

Fusarium wilt (see Cucurbits, fusarium wilt, 9.6) is uncommon on greenhouse cucumber. The pathogen is both soil- and seed-borne.

Management

Cultural practices — Greenhouse growers should destroy diseased plants, prunings and other plant residues. Diseased crop residues should be handled carefully to minimize spore dispersal. If the disease is severe, the greenhouse area, containers and growing media should be disinfested and care should be taken to prevent reinfestation of growing media. Ideally, the greenhouse structure and benches also should be disinfested. Seed can be disinfested by heating it to 75°C for three days or to 80°C for two days.

Resistant cultivars — Some commercial cultivars are more resistant than others. The rootstock *Cucurbita ficifolia* is resistant.

Selected references

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.10 Gray mold *Figs. 22.10a-d*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

In greenhouses, gray mold (see Lettuce, gray mold, 11.10) is often considered to be a disease of poor management, although environmental conditions also play an important role. Outbreaks occasionally are serious and may reduce fruit yields.

Management

Cultural practices — Heat and ventilation should be adequate to keep the humidity low enough to prevent dew from forming. Pruning the lower leaves opens the canopy to air circulation and helps keep humidity low in the crop. Infested crop residues should be buried.

Chemical control — Fungicides can provide effective control, but they should be used in alternation with one another to avoid the buildup of resistant strains.

Selected references

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.11 Gummy stem blight *Figs. 22.11a-e*

Didymella bryoniae (Auersw.) Rehm
(syn. *Mycosphaerella melonis* (Pass.) Chiu & J.C. Walker)
(syn. *Mycosphaerella citrullina* (C.O. Smith) Gross.)
(anamorph *Ascochyta cucumis* Fautr. & Roum.)

This disease is common on greenhouse cucumber and can be a major problem. In Europe, it is still called mycosphaerella rot after the former name of the fungus. Gummy stem blight also can occur on field cucumber and melon, but it is generally a minor disease in the field.

Symptoms The first symptom on greenhouse cucumber is lesions on stubs left after the removal of fruit, leaves, tendrils or lateral shoots. These lesions may elongate and crack *(22.11a).* Sometimes, but not always, they exude a gold- brown gum *(22.11b).* Occasionally, they may girdle the stem *(22.11c),* causing wilt and eventual death of the plant *(22.1 11d).* Leaf symptoms usually appear at the edge of the leaf, initially as water-soaked lesions surrounded by a yellow halo. Lesions may also appear as circular spots on the middle of the lamina. These spots enlarge and become light brown and papery, and the leaves turn yellow and die. Fruit lesions appear as soft, wet, gray-green rots that are circular or irregular in shape *(22.1 le).* Sometimes, a gummy exudate develops at the center of the lesion and dries to a firm, golden brown deposit. Fruit may not show external signs of infection except for a constriction of the distal end, but it will be firm with a black internal discoloration. Pale-colored pycnidia and dark, globular pseudothecia are eventually produced on all leaf, stem and fruit lesions. Seedlings also may become infected, resulting in circular, tan or black spots on the cotyledons and stems. Deep lesions kill the seedlings; less severe lesions may not. Infected seedlings act as a source of inoculum for other plants.

Causal agent The pseudothecia of *Didymella bryoniae* are black, globose and immersed, becoming erumpent. They are 140 to 200 µm in diameter. The asci are bitunicate, cylindrical to subclavate, short stipitate or sessile, and measure 60 to 90 by 10 to 15 µm. Each ascus produces eight hyaline, biseriate ascospores, which are ellipsoid with their ends mostly rounded, slightly constricted at the septum, guttulate, and 14 to 18 by 4 to 7 µm. Associated pseudoparaphyses are hyaline, septate and branched. The pycnidia are dark brown, solitary or gregarious, immersed, eventually becoming erumpent. The pycnidia measure 120 to 180 µm in diameter. The conidia are hyaline, short, cylindrical with rounded ends, guttulate, mostly one-septate, though some are unicellular, and 6 to 10 by 3 to 4 µm.

Disease cycle The fungus can survive for up to two years as chlamydospores or dormant mycelium on undecomposed plant residue in the greenhouse or field. Primary infection of the new crop results from ascospores or conidia originating from pseudothecia or pycnidia, respectively, on plant residue. Primary infection occurs on cotyledons, leaves, stems, blossoms and fruit. Symptoms appear 3 to 10 days after infection. As the lesions age, pycnidia are produced and, under humid conditions, conidia ooze out in long gelatinous cirrhi that are dispersed by splashing water. Soon afterwards, ascospores are produced in pseudothecia on lesions and are spread by air currents. Spores can also be dispersed on pruning knives, wet hands and clothing. A water film on the plant surface is necessary for release of conidia, infection and further spread of the disease. Disease severity increases in high humidity. Plants may also be predisposed to infection by cucumber beetles, aphids and powdery mildew. Wounding is essential for the infection of older leaves and for fruit rot. Leaf infections usually occur at points of guttation under high humidity and where repeated guttation and evaporation have left toxic salt deposits. Flowers can be infected in as little as two hours when wet.

Management

Cultural practices — Environmental control is the most important means of managing this disease. To prevent spread of conidia, growers should try to enhance the evaporation of water from plant surfaces. Dew should never be allowed to form on the plants and the humidity should be kept low at night to avoid guttation. Overhead irrigation should be avoided. The ability of the fungus to survive for long periods in plant residue means that strict sanitation must be followed. Plants should be removed far from the greenhouse after harvest and soil beds should be disinfested. Prunings should also be removed and buried or composted. As soon as symptoms are observed on the new crop, measures should be taken to check the spread of the disease. Fruit should be handled with care to prevent wounding and stored at 12°C to reduce rot without affecting shelf-life.

Chemical control — Adequate control is hard to achieve with fungicides because cucumber plants grow fast, have dense foliage, and are continuously being wounded by picking and trimming. Fungicide sprays, if needed, must be applied weekly, which increases the risk of resistant strains of the pathogen evolving.

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(Original by J.G. Menzies, W.R. Jarvis and I.A. MacLatchy)

▶ 22.12 Leaf blights *Figs. 22.12a,b; 9.8a,b*

Alternaria leaf blight *Alternaria* spp.
Ulocladium leaf spot *Ulocladium* spp.

These two diseases occur infrequently in greenhouse cucumber and control measures normally are not necessary. Melon and other cucurbits are alternative hosts for the pathogens. Symptoms and causal agents are described in Cucurbits, leaf blights, 9.8.

Management

Cultural practices — Growers can avoid introducing the pathogens by not growing melon and other cucurbits in or adjacent to cucumber greenhouses. Proper sanitation and humidity control help to control these diseases.

Chemical control — Foliar fungicides can be applied if leaf blight becomes a problem.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.13 Leaf rot (pink mold rot) *Fig. 22.13*

Trichothecium roseum (Pers.:Fr.) Link (teleomorph *Hypomyces trichothecioides* Tubaki)

Leaf rot is a minor disease of greenhouse cucumber that has been reported only on leaves. Elsewhere, it has been reported to cause stem and fruit rots (see Cucurbits, leaf rot, 9.9. It may occur anywhere in poorly managed greenhouses. *Trichothecium roseum* is a widely distributed fungus and grows on many types of organic substrates including several species of woody and herbaceous plants.

Symptoms On greenhouse cucumber, small lesions usually occur on the upper leaves and larger, more numerous lesions on lower leaves. Initially, the lesions appear water- soaked with a fairly broad yellowish margin. Later, their centers dry and turn brownish-tan *(22.13).* The centers may eventually fall out. On thin leaves, the lesions spread fast and may grow together to cover one-third or more of the leaf.

Causal agent *Trichothecium roseum* is easy to isolate. The fungus is a prolific producer of conidia in culture and is a "weed" mold in the laboratory. Growth is rapid, white at first, then pale rosy pink as the conidia develop. Conidiophores are long, slender, simple and septate. Conidia are borne apically and singly. They are attached in groups or chains, but not end to end. The conidia are hyaline or brightly colored, two-celled, ovoid to ellipsoid, measure 12 to 18 by 8 to 10 µm, appear in basipetal succession from the tip of the conidiophore, and bear a detachment scar and a thickened zone of contact with the neighboring conidium.

Disease cycle The fungus is air-borne but it may also enter the greenhouse on mulch materials or manure. Infection is almost always initiated from insect frass, petals or dead plant parts that fall onto leaves in a humid environment. The fungus is frequently seen on fruit rotted by other organisms.

Management

Cultural practices — Strict attention to greenhouse ventilation and humidity control should prevent this disease.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.14 Penicillium stem rot *Figs. 22.14a,b*

Penicillium oxalicum Currie & Thom

This disease was first reported from Ontario and subsequently from England, the Netherlands and Scandinavia. It is a major disease in southwestern Ontario, particularly in rockwool-grown crops in the Niagara area. *Penicillium oxalicum* has a cosmopolitan distribution and is commonly found in soil and on decaying organic matter. It has been reported on *Cucumis, Sorghum* and *Zea* species.

Symptoms Symptoms are sometimes confused with those of gummy stem blight and gray mold. Symptoms appear as water-soaked, olive-green areas at one or more of the nodes, 1 to 1.5 m from the ground in plants about 2 m tall, and usually only at nodes that have been pruned. Within a day or so, a bluish-gray fungal growth appears *(22.14a),* giving off a cloud of conidia when touched. The stem splits open easily to reveal more fungal growth and a mass of conidia inside the rotted tissue. Unsupported stems collapse at the infected nodes and the top dies. The lesion expands rapidly, extending for several centimetres above and below the node and has a dry, pale brown margin, not unlike that caused by gummy stem blight and gray mold, if the external fungal growth is restricted. Senescent flowers bear conidiophores and a soft brown rot extends back into the fruit, which appears rather pointed *(22.14b).* Wounds on the fruit, including fingernail marks, can become infected. Infection may escape notice at harvest and develop further in storage, often followed by bacterial soft-rot.

Causal agent *Penicillium oxalicum* produces lesions that are readily distinguished from those of gummy stem blight and gray mold by profuse bluish-green sporulation. Initially tentatively identified as *P. crustosum* Thom, the pathogen is now recognized as *P. oxalicum.*

The fungus is readily isolated from conidia on lesions. It grows rapidly on a wide variety of agar media. Colonies on Czapek-Dox agar are blue- green, becoming jade-green with age, with a white margin. The texture is flat and velvety with a deep layer of conidia that shifts when the culture dish is tapped. The conidiophore is asymmetrical, biverticillate and smooth-walled. Conidia are smooth, elliptical and delicately verruculose. They measure 3.8 to 5.5 by 2.8 to 4.4 pm. Oxalic acid, which is produced in culture by *P. oxalicum,* may be of etiologic significance.

Disease cycle The fungus sporulates profusely on the lesions and inside the stem. It infects pruning wounds on the stem and wounds on the fruit made by pulling instead of cutting fruit cleanly. Precise environmental conditions for epidemic development are not known, but the disease is more prevalent in "soft" crops and it tends to be more severe in outside rows. Conidia are mostly air-borne but they can also be carried on knives and fingers. Infection is likely to occur where sap is exuded from pruning and other wounds in conditions of high humidity. The disease seems to be associated with excessive nitrogen and the stress of too many stem fruits. It is significantly more severe in crops grown in rockwool versus soil. No differences between cultivars have been detected. The pathogen is a very common soil fungus but is known as a pathogen only of cucumber and of corn seedlings and ears. It is seed-borne in corn. It probably survives between crops in crop and weed debris and in the soil.

Management

Cultural practices — The environment should be adjusted so that dew does not form on the plants at night and the difference between day and night temperatures is minimized. Warm, humid daytime air should be purged at dusk, particularly if the night promises to be cold and clear, in which case heat should be supplied at night and the ventilators left slightly open. Air circulation should be good at all times. To prevent plants from growing too luxuriantly, the potassium:nitrogen ratio of the fertilizer solution should be adjusted by increasing potassium and reducing nitrogen.

All stem fruits should be removed to a height of about 1 m. Side shoots should be cut cleanly with a sharp knife and knives should be disinfested at frequent intervals while working an affected crop. Badly infested areas should be worked last. Collapsed plants should be cut below the lesions and carefully removed in plastic bags to avoid spreading spores.

Fruit should be harvested with a knife, leaving 5 mm of stem, and should be handled carefully to avoid wounds. The fruit should be cooled as soon as possible. The storage area should be well ventilated. Overhead irrigation should be avoided.

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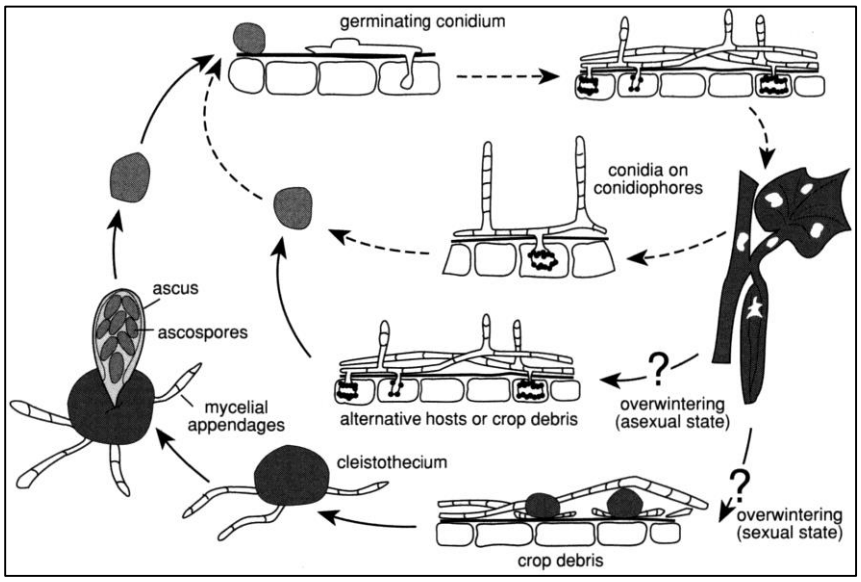
(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.15 Powdery mildew *Figs. 22.15a-d; 22.15T1*

Erysiphe cichoracearum DC.
Sphaerotheca fuliginea (Schlechtend.:Fr.) Pollacci



22.15T1 Powdery mildew; disease cycle of *Sphaerotheca fuliginea.*

This is a major disease of greenhouse cucumber and can be caused by two species of fungi. In North America, *S. fuliginea* is more common than *E. cichoracearum.* In Canada, *E. cichoracearum* is known only in Alberta; elsewhere *S. fuliginea* is the only pathogen. In Europe, both fungi are common and may co-exist in the same crop. The two fungi, and especially *E. cichoracearum,* have wide host ranges that include many species of cultivated and native plants.

Symptoms Small, round, whitish, talcum-like spots on the stems and leaves are the first sign of disease *(22.15a).* These powdery colonies increase in number and grow together, eventually covering most of the upper and lower leaf surfaces (22.*15b).* Older colonies of *S. fuliginea* turn a dirty white with age; those of *E. cichoracearum* remain pure white. Severely affected leaves turn yellow, then brown and shrivelled. Fruit is usually, but not always *(22.15c),* free of visible infection, even in the presence of heavily infected foliage, but yield and fruit quality can be reduced by loss of leaves and water stress. Vines and fruit exposed when the foliage dies may wither and become whitish from sunburn. The teleomorph state of these fungi, imbedded in the colonies as small, brown or black, globose cleistothecia *(22.15d),* is rare in Canada. Cleistothecia of *S. fuliginea* have been reported only twice in Ontario.

Causal agent Most powdery mildew fungi are identified by characteristics of the sexual state. The brown to black, globose cleistothecia of *Sphaerotheca fuliginea* have branched appendages and contain one ascus, while the black globose cleistothecia of *Erysiphe cichoracearum* have unbranched appendages and contain 10 to 15 asci. Cleistothecia, however, are rarely present, so these two fungi are normally identified by the conidial state. Both *Erysiphe* and *Sphaerotheca* have *Oidium*-type conidiophores with long conidial chains and external mycelium. Conidia are ovoid-cylindrical, more cylindrical in *E. cichoracearum,* and measure 27.5 to 40 by 15 to 18 µm.

Conidia of *S. fuliginea* tend to produce forked germ tubes, whereas those of *E. cichoracearum* are unbranched. In addition, conidia of *S. fuliginea* contain refractive fibrosin bodies that are best seen in 3% potassium hydroxide solution.

Disease cycle These fungi survive from season to season in either the sexual or asexual state *(22.15T1).* The sexual cleistothecia are rare and probably are not of any significance to the disease cycle, except as a source of genetic variability. The fungi may survive as conidia or mycelium on a variety of host plants. Because of host specialization, weeds are not a usual source of powdery mildew for cucumber. The pathogens more likely survive from season to season in the asexual state on living cucurbits, spreading by wind-blown spores. Conidia may also survive in the greenhouse for short periods, infecting new cucumber crops, particularly when the new crop overlaps or follows too soon after removal of the old crop.

On a new crop, the disease cycle is initiated when conidia or ascospores contact susceptible host tissue. Conidia germinate at temperatures between 22 and 31°C, with the optimum 28°C. They survive for only a few hours at 27°C or higher, much longer at 5°C, but not at 1°C or lower. Conidia can germinate at relative humidities of 20% or lower, but the incidence of infection increases with the relative humidity. Conidial germination of *S. fuliginea* requires a deposit of dew but it does not occur in the presence of waterlogging. Paradoxically, mildew develops best in diurnally fluctuating conditions of temperature and humidity.

Cucurbit powdery mildew is more severe in shade than in full light, in close plant spacings, and where luxuriant growth occurs because of high nitrogen levels. Colonies from single conidia are rarely over 2 cm in diameter and slower growth occurs on senescent host tissue. Under ideal conditions, coni- dia are produced on the new colony five to seven days after infection but this may take longer if conditions are unfavorable. Conidial dissemination is almost exclusively by air currents but thrips and other insects have been reported to promote local spread. If sexuality and nutritional conditions are favorable, cleistothecia form,

imbedded in the mycelia of the colonies, but this is rare. Mature ascospores are forcibly discharged as the cleistothecium imbibes water and ruptures. The ascospores are disseminated by air currents.

Management

Cultural practices — Spraying affected cucumber plants with water every two to three days will control powdery mildew. Plants should be sprayed in the morning, so they can dry in two to three hours. This prevents infection by other disease-causing organisms. Fogging considerably reduces disease severity.

Other cultural control techniques involve environmental management and sanitation procedures. Greenhouse temperatures should be maintained at about 21°C by heating and ventilation. Growers should avoid conditions that promote excessive succulent growth, such as excessive fertilization. Overcrowding, shading and overwatering also should be avoided. Allowing a period of two to three weeks when the greenhouse is thoroughly cleaned and empty between successive crops helps to prevent a carry-over of powdery mildew from an infected crop to a new one. Greenhouses and the surrounding area should be kept clear of susceptible crops, weeds and trash piles.

The amendment of hydroponic nutrient solutions with 100 ppm of soluble silica helps to control powdery mildew. The silica can be added in the form of potassium or sodium silicate. For effective control, silica must be constantly supplied to the plants. Silica sprays (1000 ppm) applied to the foliage may also reduce powdery mildew, but the solution must be adjusted to a pH of 5.5 using an acid such as phosphoric acid.

Resistant cultivars — Aramon, Bella, Cordoba, Fidelio, K8200, Marillo, Miland, Profito and TW242 are resistant to powdery mildew. Unfortunately most of these have not proven suitable for main crop (long-season) production in Canada.

Biological control — The fungi *Ampelomyces* and *Tilletiopsis* spp. are effective biocontrol agents of powdery mildew on greenhouse cucumber but are not commercially available.

Chemical control — Fungicides can be useful in controlling powdery mildew, especially systemics that offer protection to upper and lower leaf surfaces. However, some fungicides can damage cucumber plants, especially at high temperatures. Powdery mildew fungi may develop resistance to certain fungicides, for example, benzimidazoles, so an alternating spray program using two or more different fungicides is necessary to deter fungicide-resistant strains of the pathogen from evolving. Fungicides may also be harmful to certain biocontrol agents used in greenhouse pest management programs.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.16 Scab (gummosis)*Figs. 22.16; 9.13*

Cladosporium cucumerinum Ellis & Arth**.**

Scab or gummosis (for symptoms and causal agent, see Cucurbits, scab, 9.13) is not common in well-managed greenhouses, but it is seen occasionally throughout Canada.

Management *Cultural practices* — In the greenhouse, growers should minimize dew formation on the plants through ventilation and temperature control. Overhead irrigation should be avoided.

Resistant cultivars — Virtually all modern cultivars of long English cucumber are resistant. Growers should consult current seed catalogs.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.17 Verticillium wilt *Figs. 22.17a,b*

Verticillium albo-atrum Reinke & Berthier *Verticillium dahliae* Kleb.

This is an uncommon disease of greenhouse cucumber and is of minor economic importance. Both pathogens can attack several types of vegetable crops (see Potato, verticillium wilt, 16.20) and exist in specialized physiologic strains.

Symptoms The symptoms are very similar to those of fusarium wilt. Initial symptoms include wilting of the lower leaves, with recovery of the plant at night. As the disease progresses, some marginal and interveinal chlorosis develops on lower leaves (*22.17a*) and they may also show characteristic V-shaped lesions in which yellowing occurs in a fan pattern, narrowing proximally from the leaf margins *(22.17b).* The vascular tissues in the stem may become prominent. Cutting the stems of affected plants longitudinally reveals a brown discoloration of the vascular tissues. Wilt-infected plants usually die prematurely.

Causal agent (see Potato, verticillium wilt)

Disease cycle (see Potato, verticillium wilt, 16.20) *Verticillium albo-atrum* and *V. dahliae* survive primarily as dark, resting mycelium or microsclerotia, respectively, in plant debris in the soil. Because of this, verticillium wilt is more common in greenhouse cucumber grown in soil, but it also occurs in soilless media that become infested with the pathogens. *Verticillium* spp. infect the roots, where they invade the vessels and interfere with water transport.

Management

Cultural practices — If greenhouses become infested with *Verticillium* species, growers should fumigate the soil before transplanting, or spread plastic over the soil and use a soilless growing medium. Diseased plants should be removed from the greenhouse and destroyed. A thorough clean-up of the premises should be done after a diseased crop has been removed.

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(Original by J.G. Menzies)

▶ 22.18 White mold (sclerotinia stem rot) *Figs. 22.18a-d*

Sclerotinia minor Jagger
Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

White mold (see Cucurbits, white mold, 9.14; see also Lettuce, drop, 11.9) is a fungal disease that is aggravated by poor management. It is not common in greenhouses and should not occur if plants are kept free from persistent water droplets and film, especially at flowering. In individual greenhouses it can occasionally destroy plants (*22.18a,b*) and rot fruit after picking (22.*18c,d).*

Management

Cultural practices — Plants should not be crowded unnecessarily and vigorous soft growth should be avoided. Good weed control around the greenhouse will remove or reduce alternative hosts of these pathogens. If the fungi become established in the greenhouse, steam pasteurization will kill the sclerotia in the soil. Chemical fumigation may not always be effective. Polyethylene sheets on the floor prevent ascospores dispersing from apothecia.

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(Original by J.G. Menzies and W.R. Jarvis)

VIRAL DISEASES

▶ 22.19 Beet pseudo-yellows *Fig. 22.19*

Beet pseudo-yellows virus

This disease is not usually a problem, but it can be destuctive in greenhouses where the greenhouse whitefly is also a problem. Hosts of beet pseudo-yellows virus include carrot, cucumber, flax, lettuce, muskmelon, spinach, squash and sugar beet. Some ornamentals and weeds also are attacked and may be a reservoir of the virus.

Symptoms This virus causes a chlorotic yellowing between the veins of older leaves *(22.19)* and a downward curling of the margins of affected leaves. The younger leaves may not be affected in the early stages of disease development. Symptoms have not been observed on fruit but affected plants age prematurely and are less productive.

Causal agent Attempts to observe beet pseudo-yellows virus using electron microscopy have not been successful. The nature of this virus is therefore uncertain.

Disease cycle The virus is transmitted by the greenhouse whitefly. Conditions that favor this pest also favor epidemic development of the virus. The whitefly becomes infective after only one hour of feeding on an infected host and it need feed for only one hour on a susceptible host to transmit the virus. The latent period of this virus in the vector is less than six hours, and the whitefly can retain the virus for four days. This virus has not been shown to be transmitted mechanically or by seed.

Management Control of the greenhouse whitefly is necessary to management of this disease (see greenhouse whitefly, 22.32).

Cultural practices — Ornamentals and other non-crop plants should not be introduced into the greenhouse as they may carry the virus.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.20 Cucumber mosaic *Figs. 22.20a,b; 9.15*

Cucumber mosaic virus

Cucumber mosaic is a worldwide problem on cucurbits. It is particularly important in temperate regions. Cucumber mosaic virus can infect plants in more than 40 angiosperm families. Crop hosts in Canada include clover, corn, cucumber, French bean, lettuce, melon, pepper, tomato, safflower, spinach, squash and sugar beet. Ornamental hosts include narcissus, gladiolus, impatiens, petunia, phlox and rudbeckia.

Symptoms This virus may infect plants at any stage of growth, but normally cucumber plants are infected at the six- to eight-leaf stage when they are growing rapidly. If seedlings are infected, the cotyledons wilt or turn yellow, and the plants are dwarfed. New leaves will be slightly mottled, wrinkled and distorted *(9.15)* with a slight downward curling of the edges. Infection of older, vigorously growing cucumber plants results in young leaves developing small, about 1 to 2 mm, greenish-yellow translucent lesions, which are normally confined by the leaf veins. The leaf edges curl downward and the leaf surface becomes finely wrinkled and distorted with slightly raised tissue between the small veins. Eventually, a yellow-green mottling develops *(22.20a).* Occasionally, only the leaf tip turns yellowish without a sharply defined mottle. Older leaves may become severely affected and die, resulting in a slow decline of wilted plants. After infection, plant growth is stunted with shortened internodes. In lush-growing crops, infected vines may wither before showing signs of distortion and may die within seven days. Few fruits are set once infection occurs and fruit that does set has a yellow-green mottle on the stem, gradually extending over the entire fruit surface *(22.20b),* interspersed with dark green areas that are usually raised and wart-like. Occasionally, fruit becomes smooth, green-white and blunt at the end. This symptom, known as white pickle, is characteristic.

Causal agent Cucumber mosaic virus particles are isometric, 30 nm in diameter and are composed of 180 subunits in pentamer-hexamer clusters. The particle center is hollow. Molecular weight is 5.8 to 6.7 by 10^6. Identification can be confirmed by inoculation to indicator plants. Cucumber develops a green or yellow-green systemic mosaic. Leaves of tobacco (*Nicotiana tabacum* L., N. *clevelandii* A. Gray, and *N. glutinosa* L.) leaves remain symptomless or develop chlorotic or necrotic lesions followed by a green or yellow-green systemic mosaic or ringspots, usually without necrosis. Tomato develops a systemic mosaic and fern leaf, with the leaf laminae much narrowed. French bean (*Phaseolus vulgaris* L.) develops local, pin-point, non-systemic, necrotic lesions in winter but not in summer. *Chenopodium amaranticolor* Coste & Reynier, and *C. quinoa* Willd. develop chlorotic or necrotic local lesions. Some cultivars of cow- pea (*Vigna unguiculata* (L.) Walp.) develop local lesions, and most isolates of cucumber mosaic virus are non-systemic in this plant.

Disease cycle This virus survives from season to season in alternative hosts. Transmission is facilitated by more than 60 species of aphids and at least two species of beetles (the striped and spotted cucumber beetles). All aphid stages can transmit the virus, which is acquired and inoculated in about one minute. There is no latent period and the virus is retained by the aphid for less than four hours. The virus is not transmitted transovarially to aphid offspring. The virus can also be transmitted by at least 10 species of dodder. The virus is systemic and can be spread in plant sap carried on pruning knives or pickers' hands. This virus is only rarely transmitted by seed. Symptoms usually appear within four to five days of infection in young plants and within 14 days in older plants. Symptoms develop faster at temperatures of 26 to 32°C than at 16 to 24°C. Symptoms are more severe on plants exposed to short days or reduced light.

Management

Cultural practices — Elimination of reservoir hosts in the area of cucumber greenhouses generally will reduce the amount of initial inoculum unless many different hosts are native to the area. If possible, growers should avoid double cropping and should not plant a new crop near infested crops or their residues. Elimination of alternative hosts in a 100-m-wide area around the perimeter of greenhouses will give satisfactory control of the virus in most years. Aphid control may also be useful in preventing spread of the virus, but it must be done promptly to prevent virus transfer. In greenhouses, ventilators can be fitted with screens to exclude aphids, aluminum mulch can be used to repel incoming aphids, and a barrier crop of wheat may delay or reduce viral spread to susceptible cucumber plants. Reduced handling of plants may also delay spread of this virus, though in general it is not readily transmitted by contact. Hands, clothing and tools should be washed frequently, and dipping hands and tools in skim milk minimizes spread.

Resistant cultivars — European seedless cultivars have little or no resistance to cucumber mosaic virus.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.21 Cucumber necrosis

Cucumber necrosis virus

Cucumber necrosis virus is soil-borne, affecting mainly greenhouse cucumber, but it can also infect field cucumber. It causes a minor disease of greenhouse cucumber.

Symptoms Young leaves turn upright and yellowish. The youngest leaves develop a purple tinge as their tissues desiccate and die. Yellow-green to tan-colored areas with pinpoint necrotic areas, 1 to 8 mm in diameter, may form on the leaf blades. These may fall out, leaving "shot-holes" of various sizes. The virus moves systemically through the plant, and leaves may show symptoms on only one side of the midrib. Severely affected leaves are malformed with dark green, flap-like enations on the lower surface. Enations often occur around a shot-hole or along a vein approximately two to three weeks after the first appearance of systemic symptoms; enations are often the only symptom during summer.

Causal agent Cucumber necrosis virus is an RNA-containing virus with isometric particles about 31 nm in diameter. The properties and host range of this virus and the cucumber strains of tobacco necrosis are similar, making serological tests the only reliable way to distinguish between them.

On cucumber, necrotic lesions develop in inoculated cotyledons in three to four days, enlarging to 3 to 5 mm. The cotyledons desiccate and die. Systemic symptoms may include: chlorotic or tan-colored areas with pinpoint necrotic centers that usually fall out leaving shot-holes of various sizes; severely deformed leaves, sometimes with dark green enations; and small fruits on stunted plants, occasionally with a green mottle.

Virus identity can be confirmed by inoculating indicator plants. *Gomphrena globosa* L. develops irregular, grayish necrotic lesions with reddish margins in three to five days. *Chenopodium amaranticolor* Coste & Reynier develops local necrotic lesions, 0.5 to 1 mm, in two to five days. The margins redden as the leaves age. Heavily infected leaves desiccate and fall.

Disease cycle This virus is transmitted by a soil-inhabiting fungus, *Olpidium radicale* Schwartz & Cook, which appears to infect only members of the family Cucurbitaceae. The virus can infect roots in soil contaminated with infested crop residues or sap. It also can be transmitted mechanically from plant to plant by rubbing abraded leaves with infective sap, a pathway that may apply in commercial greenhouses. Amaranthaceae, Chenopodiaceae, Compositae, Cucurbitaceae, Leguminosae and Solanaceae can be infected by mechanical inoculation with plant sap. In greenhouses, the virus is most severe in autumn and winter. As the days lengthen, symptoms become milder and indistinct, and plants that exhibited severe symptoms in the spring may display almost complete recovery by summer.

Management

Cultural practices — Steam sterilization of soil is necessary to control this soil-borne virus. Chemical fumigation is ineffective. Diseased plants should be rogued and removed without touching other plants, and they should be burned, not buried. Sap-contaminated tools should be heat sterilized, hands should be washed, and clothes changed after handling diseased plants and before entering a disease-free area.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.22 Cucumber pale fruit *Fig. 22.22*

Cucumber pale fruit viroid

This fruit disease of greenhouse cucumber was first observed in the Netherlands and has been reported since 1963 in other countries. If pale fruit becomes established in a cucumber crop, large reductions in fruit yield and quality can occur. In Canada, cucumber pale fruit has been a problem only in British Columbia. The pathogen is similar in its host range and reactions to potato spindle tuber viroid (see Potato, spindle tuber, 16.28) and chrysanthemum stunt viroid.

Symptoms The disease is more severe at higher temperatures. The most distinctive symptoms are pale-green, small and often slightly pear-shaped fruits (22.22). However, in plants grown at 30°C, leaf symptoms may appear before flower and fruit development. Developing leaves are small, blue-green and rugose. Leaf blades are undulating, with the edges turned downward and the tips bent downward or turned backwards. Upon aging, leaf symptoms fade, chlorosis appears and the plant may be somewhat stunted. Flowers may be stunted and crumpled with the edge of the petals slightly notched.

Causal agent This is a typical plant viroid: low molecular weight RNA, 1.1 to 1.3 x 10^5, having a unique structure. It is not found in healthy plants. It replicates autonomously despite its small size. Unlike viral nucleic acids, viroids are not encapsulated, so no virion-like particles can be isolated from infected tissues.

Several cucurbits are susceptible and exhibit discernible symptoms. *Benincasa hispida* (Thunb.) Cogn. (Chinese watermelon) may be most useful as an indicator plant because its incubation period is short and symptoms are pronounced. A local-lesion host has not been found. In cucumber, symptoms are identical to those of hop stunt viroid from which this viroid differs at 16 nucleotide positions with about 95% sequence homology. Cucumber plants can be individually tested by polyacrylamide gel electrophoresis. For direct detection in seed samples, DNA complementary to potato spindle tuber RNA can be used by the spot hybridization technique.

Disease cycle The pathogen is mechanically transmissible with crude sap expressed from infected cucumber plants. Slashing of the stems with a contaminated knife also infects plants. Seed transmission has been proven experimentally in tomato. The viroid normally occurs only in greenhouses. It does not overwinter in crop residue, and it does not appear to be transmitted by insects. Infected seed is the most likely initial source of inoculum.

Management

Cultural practices — Good sanitation during pruning and harvesting operations will minimize spread of the disease from infected to healthy plants.

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(Original by R. Stace-Smith, J.G. Menzies and W.R. Jarvis)

▶ 22.23 Watermelon mosaic *Figs. 22.23; 9.17a,b*

Watermelon mosaic virus

This virus was first reported on cucumber in 1975 in southern Ontario and has not been found elsewhere in Canada. Watermelon mosaic is generally of minor importance on greenhouse cucumber. Various hosts of watermelon mosaic virus occur in the families Cucurbitaceae and Leguminosae.

Symptoms Initially, leaves may show a yellow veinclearing and flecking, followed by a uniform green to dark green mosaic *(9.17a).* The leaf margins turn upward. There is subsequent leaf distortion with downward hooking of the edges, irregular venation, dark green vein-banding *(9.17b),* and dark green blisters between the veins *(22.23).* Leaf symptoms are usually uniform, resembling injury due to herbicides or other abiotic disorders. The fruits are severely shortened, curled and gnarled.

Causal agent Watermelon mosaic virus particles are flexuous filaments, about 750 nm long. The particles precipitate in plant sap at pH 4.9. The electrophoretic R_0 value is 0.25. Two distinct strains have been described in North America. Strain 1 is restricted to cucurbits; strain 2 is not. On cucurbits, these strains cannot be differentiated by their symptoms.

This virus can be distinguished from squash mosaic and cucumber mosaic because it infects watermelon systemically, and from cucumber green mottle mosaic and tobacco ringspot because it is transmitted by aphids. Watermelon develops systemic vein-banding, mosaic and leaf-dis-tortion. Pumpkin develops interveinal chlorosis, mosaic, raised green blisters and leaf distortion.

Disease cycle Limited evidence suggests that the virus is seed-borne. Sources of primary infection have not been fully identified. The virus is transmitted from plant to plant in a non-persistent manner by the green peach, melon and black bean aphids. The green peach aphid can acquire the virus from an infected plant after only 10 to 30 seconds of feeding. Secondary spread is also by aphid vectors. Disease symptoms appear one to two weeks after inoculation.

Management Effective control of this virus requires the elimination of reservoir hosts and aphid vectors.

Cultural practices — Greenhouses should be located in areas with limited populations of reservoir hosts, if possible, or upwind from them. Herbicide applications or other practices to control reservoir hosts around the greenhouse, in ditch banks or along hedge rows are helpful. The use of aphid-repellant aluminum foil mulches, oil sprays and protective crops around greenhouses can also reduce virus incidence in cucumber crops. Wheat grown as a protective crop around greenhouses provides feeding sites for aphids. As they feed, virus on their stylets is diluted by the wheat sap and loses its infectivity. Seed should be bought only from reputable sources.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 22.24 Zucchini yellow mosaic *Figs. 22.24a,b; 9.16a-c*

Zucchini yellow mosaic virus

In greenhouse cucumber, severe mosaic, yellowing and distortion of the leaves and fruit may occur *(22.24a,b).* Up to 80% of a cucumber crop was infected in one Ontario greenhouse. The disease has a limited distribution in Canada, having been reported only from Ontario, Alberta and British Columbia. (For more detail on this disease, see Cucurbits, zucchini yellow mosaic, 9.16.)

(Original by W.R. Jarvis and J.G. Menzies)

NON-INFECTIOUS DISEASES

▶ 22.25 Chilling injury, cold injury *Figs. 22.25a,b*

Cucumbers are sensitive to temperatures lower than the recommended minimum day or night temperatures for particular cultivars, and the effects are exaggerated under conditions of poor light. (See also field injury, *9.18a-c.*)

In the early part of the season, plants develop slowly with excessively large leaves. In a greenhouse with poorly insulated walls, there may be a marked gradient in heat distribution and plant height across the house. Similarly, turbulator fans cause patches of poor growth when they bring cold air down from the roof space. Flowering is delayed by low temperatures, and fruits may abort. Applying cold water may cause leaf puckering *(22.25a).*

Greenhouse cucumber fruit can be injured on the vine (cold injury) or in post-harvest storage (chilling injury) by low temperatures. Maturing fruit that is subject to cold drafts and sudden drops in temperature develops characteristic scars. These consist of white or light brown longitudinal marks, often on one side, but occasionally all around the fruit *(22.25b).* The scars are corky in appearance and, although superficial, they detract from market quality. Similar symptoms can result from thrips feeding on the developing fruit. In severe cases, the fruit may split. Slow- growing fruit may be bitter.

Chilling injury results in pitting of the fruit because of a collapse of epidermal tissues. These pits turn yellow and eventually may cause the entire fruit to become chlorotic. Chilling injury may increase the susceptibility of fruit to microbial decay.

Management

Cultural practices — The risk of cold injury can be lessened by reducing cold drafts in the greenhouse and by avoiding spraying plants with cold water. Air temperatures in the greenhouse should not be allowed to fall below 16°C. Chilling injury can be prevented by storing cucumber fruit between 10 and 13°C.

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(Original by W.R. Jarvis and J.G. Menzies)

▶ 22.26 Nutritional disorders *Figs. 22.26a-g*

Boron
Calcium
Copper
Iron
Magnesium
Manganese
Molybdenum
Nitrogen
Phosphorus
Potassium

Boron

(22.26a) Leaves of boron-deficient plants are leathery, acquire a dark green color and may exhibit death of the apical bud. Older leaves usually develop a brownish- yellow, interveinal chlorosis and scorched margins, while younger leaves may be malformed and cupped with prominent veins. Affected plants are brittle. Young fruits sometimes die. Developing fruits are malformed with longitudinal white stripes, as though exposed to cold.

Calcium

Calcium deficiency results in growing points turning dark and twisted, with young leaves stunted in size. Leaf edges curl downward and turn a pale brown color. Young fruit may shrivel, discolor and show signs of blossom-end rot. This problem can be quite common in rapidly growing crops. Affected plants are unable to regulate the internal distribution of calcium adequately owing to insufficient transpiration. High levels of potassium may restrict the uptake of calcium.

Copper

Copper deficiency results in short internodes, small leaves and yellow blotches between the veins on older leaves. This chlorosis usually moves steadily up the plant. The leaves will become dull green or bronze colored and wither.

Iron

Plants suffering from iron deficiency display interveinal yellowing of the youngest leaves (*22.26d*), with the veins gradually becoming yellow. Eventually, the whole leaf may turn yellow or yellowish-white. Older leaves generally remain green. Affected plants may cease to grow and fruit may become pale-colored.

Magnesium

Leaves on plants with magnesium deficiency exhibit interveinal yellowing, but the veins remain green *(22.26b).* In many cases, a green margin will remain around the leaf even under conditions of severe deficiency. Leaves may curl downward, become hard or brittle, and overall growth may be stunted. Affected fruits usually are pale green. These symptoms may be difficult to distinguish from those of manganese deficiency.

Manganese

Symptoms of manganese deficiency occur first on the oldest leaves in the form of pale green areas. Soon afterward, a net-like pattern appears with only green tissues occurring along the main veins. The leaves will become thin and die around the edges. This symptom may be difficult to distinguish from magnesium deficiency.

Molybdenum

Molybdenum-deficient plants are stunted and have scorched leaves (*22.26c*) with up-rolled margins. The symptoms generally start on the lower leaves and move upward on the plant.

Nitrogen

Nitrogen deficiency results in restricted plant growth and reduction in fruit numbers. Young leaves are small and pale yellow-green *(22.26e),* while older leaves turn yellow uniformly and may die prematurely. Affected fruits are short and pale green and the blossom end may be pointed (*22.26f*).

An excess of nitrogen results in stunted plants with small, dark green leaves. Affected leaves tend to curl downward and the petioles droop slightly. Chlorosis of leaf margins also may occur.

Phosphorus

Slight phosphorus deficiency may result in stunted plants without leaf symptoms. By contrast, a severe deficiency stunts growth, and younger leaves become small, stiff, turn dark grayish-green and expand slowly. Leaf veins and petioles turn reddish-purple and large brown areas may develop on the lower leaves and spread up the plant. Affected leaves eventually shrivel up.

Potassium

Cucumber plants suffering from potassium deficiency have small leaves showing chlorosis, bronzing and marginal scorching (*22.26g*). Deficient plants are usually severely stunted, while affected fruits have enlarged tips but remain undeveloped at the stem end. Such fruit may develop brown marks and have a bitter taste.

Management

Cultural practices — Low light and poor weather may result in a lack of ventilation, creating high humidity in the greenhouse, which restricts transpiration. Increasing the ventilation may improve the transpiration activity of the plants, and symptoms may disappear on new growth.

For the majority of these nutrient disorders, adjusting the mineral content of the nutrient solution will correct them. For calcium deficiency, altering the composition of the nutrient solutions may not always alleviate the problem. Foliar applications of calcium nitrate (0.5 kg/500 L water plus a wetting agent), or 150 g potassium nitrate plus 100 g calcium nitrate per litre of stock solution diluted 1:200, will alleviate calcium stress.

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(Original by J.G. Menzies)

▶ 22.27 Premature fruit yellowing

This is a common post-harvest fruit disorder of greenhouse cucumber. The cause is unknown, but it appears to be related to growing conditions during fruiting.

Management

Cultural practices — Increasing the concentration of fertilizer in the nutrient solution and decreasing the fruit number per plant may lessen the incidence of premature fruit yellowing. This disorder also can be reduced by increasing the amount of light reaching the fruit through practices such as leaf pruning.

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(Original by J.G. Menzies)

▶ 22.28 Root death *Fig. 22.28*

In healthy plants, roots die and are replaced throughout the cropping season, but sometimes roots die suddenly and faster than they can be replaced. Collapse of the plant is then spectacular, particularly in systems using the nutrient film technique (NFT).

Symptoms Plants wilt and collapse within five to eight hours, and there is no recovery. In NFT systems, the roots turn brown *(22.28)* and slimy and, when disturbed, disintegrate completely into fragments. Usually, most of a crop is affected simultaneously.

Causal agent Several factors may cause root death but the condition is usually associated with stress. Conditions such as low or high temperature, high electrical conductivity, poor oxygenation of the nutrient solution, or too heavy a fruit load on small, weak or diseased plants may occur singly or together, exacerbating root death. Extensive root growth in NFT systems can lead to inefficient flow rates of nutrient solution around the roots, which can result in high electrical conductivity and poor aeration of the solution. In hydroponic systems, the sudden collapse of the crop is diagnostic, together with the complete disintegration of the root system.

Disease cycle Root death is more a physiological disorder than a disease caused by a specific microorganism. However, it is not unusual to isolate pathogenic fungi and bacteria from dead roots. *Clostridium* bacteria are often present. They are anaerobic and their presence suggests a local deficiency of oxygen at the root surface, which could cause the roots to autolyse or self-destruct. Usually, most of a crop is affected simultaneously, probably reflecting a nutritional or osmotic imbalance in the fertilizer system, or marked shifts in source- sink relationships in the plant. Generally, cucumber soil media temperatures must be above 15°C. Lower temperatures result in decreased root activity and reduced plant growth. If the air temperature is around 25°C and root temperature is 15°C, the root system is unable to sustain the top growth. This results in root dieback and eventual death of the

plant. A high electrical conductivity around the root reduces or prevents water uptake into the root from the growing medium. Eventually, excess water accumulates around the roots, thus decreasing aeration and causing root death by anoxia. The electrical conductivity of the feed solution and growing medium is determined by, and adjusted for plant vigor, age of plants, time of season, environmental conditions in the greenhouse and outdoors, and type of growing medium. In a rockwool system, the electrical conductivity is usually high at the start of the season when the plant is small and light intensity is low. As the plant develops and light levels increase, the conductivity should be lowered. However, during these periods, the conductivity may be raised or lowered by a few points (0.2 mS/cm) when an extended period of cloudy or sunny warm weather is experienced. In an artificial growing medium, such as rockwool, the conductivity can be as high as 3.0 mS/cm compared to 1.5 to 1.8 mS/cm in soil.

Management

Cultural practices — Root death can be corrected by avoiding stress conditions; however, once wilting has begun, it is irreversible and rapid. NFT and rockwool systems lack the buffering capacity of soil to mask and retard symptoms, so constant attention is needed to avoid the sudden stress imposed by any of a number of production accidents.

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(Original by W.R. Jarvis and J.G. Menzies)

▶ 22.29 Sudden wilting

Sudden wilting of cucumber plants can be caused by temperature and moisture extremes. If cucumber is exposed to rapid decreases in temperature, sudden wilting may occur. Affected plants usually recover but, if cool conditions persist, such as near the insulated wall in a greenhouse, permanent stunting may result. High temperatures also will cause temporary wilting of cucumber plants, and persistent high temperatures may cause the margins of lower leaves to die. This type of wilt often expresses itself in the early part of the season on warm, sunny days. If the young plants have not had time to develop adequate root systems, they wilt, but most plants recover.

Insufficient water can result in wilting or stunting of cucumber plants and, if uncorrected, they will eventually die. Overwatering can result in wilting, yellowing, root injury and reduced growth rates. See also 22.7.

Management

Cultural practices — Greenhouse temperatures should be maintained between 18 and 27°C. Irrigation schedules of cucumber crops should be carefully monitored, especially during hot weather.

(Original by J.G. Menzies)

NEMATODE PESTS

▶ 22.30 Root-knot nematodes *Figs. 22.30a-d*

Northern root-knot nematode

Meloidogyne hapla Chitwood

Southern root-knot nematodes

Meloidogyne arenaria (Neal) Chitwood
Meloidogyne incognita (Kofoid & White) Chitwood
Meloidogyne javanica (Treub) Chitwood

Greenhouse cucumber and solanaceous vegetables are very susceptible to damage from root-knot nematodes. Infected transplants may be a source of inoculum in greenhouses. Southern root-knot nematodes occasionally are imported with transplants from the United States.

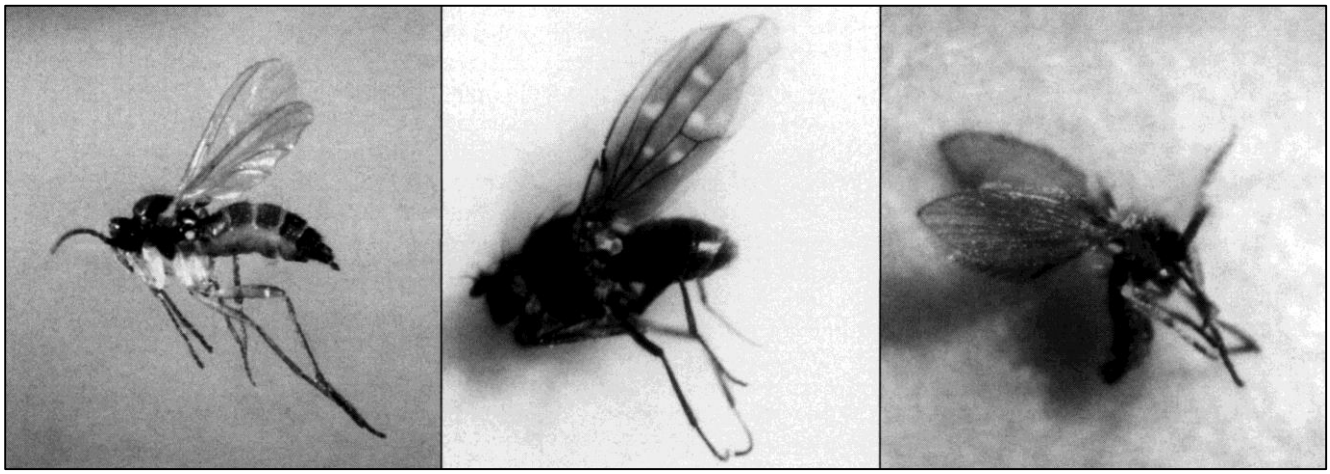
Symptoms include yellowing and stunting of stalks, early senescence, prolific branching of rootlets, and production of small, spherical galls on roots (*22.30a-d*). For a complete description, see Carrot, 6.20; see also Greenhouse tomato, 25.26, and Management of nematode pests, 3.12.

INSECT PESTS

▶ 22.31 Fungus gnats *Figs. 22.31a-c; 22.31T1*

Bradysia spp.
Corynoptera spp.

Fungus gnats are found wherever greenhouse crops are grown in Canada. They are rarely a problem on greenhouse tomato or pepper, but they may damage greenhouse cucumber crops and bedding plants. Fungus gnats are more of a problem when cucumber roots are first damaged by a nutrient imbalance or waterlogging, as in sawdust media. Adults can annoy workers through sheer numbers, but generally they are a minor and easily controlled nuisance.



22.31T1 Fungus gnat (left), shore fly (center), and moth fly (right); compare wings, body shape and leg length; adults 2-3 mm long.

Fungus gnats form a normal part of the decomposer chain in greenhouse soils, regardless of the type of crop being grown.

Damage The larva is the damaging stage, feeding on roots and root hairs. Only rarely are plants killed, but growth reduction may result and feeding opens wounds through which pathogens may invade. Plants grown free of soil, such as in hydroponic and sterile potting media, are more susceptible to damage than plants grown in soil.

There is evidence that fungus gnat adults may transmit or be involved in the movement of soil-borne diseases of greenhouse crops, notably root rots caused by *Pythium* species in cucumber and crown and root rot caused by *Fusarium oxysporum* f. sp. *radicis-lycopersici* in tomato.

Identification Fungus gnats are small dark flies (*22.31b*) (family Sciaridae), often referred to the genera *Bradysia* and *Corynoptera.* The adults are 2 to 3 mm in length. They may be mistaken for shore flies (Ephydridae) and moth flies (Psychodidae) (*22.31T1*), both of which are common but not damaging in greenhouses, or for adult *Aphidoletes,* which are biocontrol agents for aphids (see melon aphid, 22.33). Larvae of fungus gnats grow to 4 to 5 mm in length, are legless, characteristically white or almost transparent and have a prominent, black head *(22.31b).* The eggs are ovoid and cream-colored.

Life history Fungus gnat females lay eggs singly or in clusters of 2 to 10 in crevices in moist soils, in potting mix and in hydroponic media. Females can lay 100 to 200 eggs over a two- to four-day period. The eggs hatch in two to four days, and larvae require 14 to 16 days to complete development at 20°C. They feed on fungal mycelia, detritus, roots and root hairs. Pupation (*22.31a*) occurs at or just below the surface of the medium; after three to five days, the pupa wriggles to the surface before the adult emerges. The gnats are found outdoors and probably enter greenhouses continually through doorways and vents, becoming established where suitable oviposition sites are available.

Management

Monitoring — There is no action threshold for fungus gnats. Tolerable populations vary from grower to grower. If signs of damage are observed in the greenhouse, such as afternoon wilting, in conjunction with large numbers of fungus gnats around the base of the plants and larvae in the culture medium, then control measures should be initiated immediately. Also, if yellow sticky traps (see greenhouse whitefly, 22.32) become covered with fungus gnat adults within seven days, then treatment is warranted.

Cultural practices — Growers should avoid overwatering because wet conditions favor outbreaks. Waste plant material should be removed and good sanitation should be followed at all times.

Biological control — If an outbreak occurs in greenhouses where biological control is being used for other greenhouse pests, then every attempt should be made to use a biocontrol agent for fungus gnats. Various biocontrol agents are available for

this purpose. A nematode *(Heterorhabditis* sp.) is effective in controlling fungus gnat larvae, but it does not reproduce in the body of the gnat larva and must be reapplied whenever gnat populations resurge. A predatory mite, *Hypoaspis* (syn. *Geolaelaps)* sp. *(22.31c),* also is available commercially in Canada. If applied early in the season, it can effectively limit the increase in fungus gnat populations. The mite should be applied at a rate of 50 individuals per plant at planting-out to achieve satisfactory control.

Chemical control — Fungus gnat larvae and adults may be controlled by spray or drench application of insecticides to the surface of the culture medium. There are no documented cases of resistance to insecticides but resistance has been suspected in several instances.

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(Original by D.R. Gillespie)

▶ 22.32 Greenhouse whitefly *Figs. 22.32a-d; 22.34d*

Trialeurodes vaporariorum (Westwood)

The greenhouse whitefly (see Greenhouse tomato, 25.27) can be found on greenhouse cucumber crops wherever they are grown in Canada. On greenhouse cucumber, its biology is similar to that described for greenhouse tomato, the major difference being that whitefly egg production and adult longevity are 10 to 15% greater on cucumber than on tomato.

Damage The greenhouse whitefly is a major pest of greenhouse cucumber. Whitefly outbreaks have the potential to decrease yield and fruit quality through feeding damage (*22.32a*) and honeydew deposits, which encourage the subsequent growth of mold fungi *(22.32b).*

On greenhouse cucumber, the greenhouse whitefly is important as a vector of beet pseudo-yellows and contributes to the rapid spread of the disease throughout the crop.

Identification Whiteflies should be seen by an expert to confirm their identity. In general, the greenhouse whitefly is snowy white, and the wings are held horizontally over the body at rest *{22.32c).* It can usually be found on the undersides of upper leaves, flying weakly when disturbed. (For more detail, see Greenhouse tomato, 25.27.)

Life history (see Greenhouse tomato, 25.27)

Management The greenhouse whitefly can be controlled effectively using cultural procedures and biological control. At present, whiteflies are being controlled biologically on the majority of greenhouse cucumber crops in British Columbia, Alberta and the Maritime provinces.

Monitoring — The standard monitoring method for the greenhouse whitefly is the yellow sticky trap (*22.34d*). Traps should be hung as soon as plants are set out, and spaced uniformly throughout the greenhouse at a density of one trap per 20 to 50 plants. The traps should be hung with the middle of the trap level with the top of the crop, and inspected weekly. Monitoring should begin when the crop is planted, and biocontrol implemented when the first whitefly is detected.

Cultural control — Yellow sticky traps also can be used at a rate of one trap per two to five plants in whitefly hotspots to remove excessive numbers of whitefly adults from the tops of plants.

Biological control — The parasitic wasp *Encarsia formosa* Gahan is an effective biocontrol agent for the greenhouse whitefly on cucumber crops, and it should be introduced into the greenhouse as soon as the first whitefly is detected on a trap. The wasp is not attracted to the yellow sticky traps used for monitoring. The number of parasites that are released on each introduction date is greater for cucumber than for tomato because cucumber has more hairs on the leaf underside, causing the parasite to move more slowly and search for hosts less efficiently on cucumber than on tomato. Releases at a rate of one parasite pupa per two cucumber plants is recommended each week for 8 to 10 weeks or until 60 to 80% parasitism is achieved, which is estimated by counting the proportion of whitefly pupae (scales) that have turned black (*22.32d*). Whitefly scales turn black 10 to 14 days after parasitization.

Chemical control — As with greenhouse tomato, whitefly resistance to registered insecticides limits the value of chemical control. Insecticidal treatments should only be necessary toward the end of the growing season. If whiteflies are present on the crop at the end of the season, the crop should be fumigated prior to removal.

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(Original by J.L. Shipp and D.R. Gillespie)

▶ 22.33 Melon (cotton) aphid *Figs. 22.33a,b*

Aphis gossypii Glover

The melon aphid can be a problem on greenhouse cucumber throughout Canada, especially in the fall. It has only recently become a problem in greenhouse cucumber in British Columbia.

The melon aphid feeds on a variety of plants, including such vegetable crops as bean, celery, cucumber, melon, pepper and potato.

Damage Concentrations of as many as 2000 aphids per leaf develop quickly on cucumber, causing infested leaves to wilt and collapse. Younger leaves become dark green and stunted, usually with rolled or distorted edges. Large amounts of honeydew are deposited onto the leaf surfaces below colonies of aphids, encouraging a dense growth of sooty mold. Even after damaging levels of melon aphids have been controlled by pesticides, yields may take five to seven weeks to return to normal levels.

This aphid can act as a carrier of plant viruses into the greenhouse, and is an efficient vector of cucumber mosaic and watermelon mosaic viruses.

Identification The melon aphid (family Aphididae) is 1 to 3 mm in length, and globular in shape with a pair of short, black projections (cornicles) and several dark marks on the abdomen. The tubercles between the antennae are not prominent. When populations are sparse, adults are nearly black or green, nymphs somewhat lighter. As crowding increases, nymphs range from olive-green to light yellow in the densest colonies *(22.33b).* Winged adults are dark green to black *(22.33a).*

Life history Melon aphids are adapted to high temperatures. At 27°C, the aphids mature in seven days. Adults can produce an average of 40 nymphs in as few as seven days. Populations increase as much as 10- to 12-fold per week on cucumber (but only 4-fold on greenhouse eggplant crops). On greenhouse cucumber, infestations usually occur first on the lowest leaves, then spread throughout the plant. When colonies become crowded, winged forms are produced and these migrate to neighboring plants. Winged aphids also move from outdoor crops, ornamentals or weeds into greenhouses during the growing season or in early fall.

Management Action to control melon aphids must be taken as soon as the first aphid is detected because they reproduce so quickly.

Monitoring — Growers should check their crop routinely for the first sign of aphids. A threshold of seven melon aphids per square centimetre or 1000 aphids per plant has been determined (in England) as an action threshold for the application of pesticides; this threshold is unrealistically high if cucumber mosaic or watermelon mosaic are present.

Cultural practices — Growers should screen greenhouse vents, maintain a weed- and garden-free area around the greenhouse, and keep ornamentals and vegetable crops out of the same greenhouse range.

Biological control — The most promising biocontrol agent is the predatory midge *Aphidoletes aphidimyza* (Rondani). Preliminary studies in Canada suggest that it should be applied at a rate of 15 to 20 midges per plant for several weeks to achieve control. The midge must be introduced when the aphid population is extremely low, with releases starting when the first aphid is detected.

Chemical control — Aphids frequently develop resistance to pesticides, so periodical product rotation is suggested. Insecticides, where effective, must be used frequently to achieve satisfactory control. Insecticidal soap and pyrethroid sprays are not effective against the melon aphid.

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(Original by L.A. Gilkeson)

▶ 22.34 Western flower thrips *Figs. 22.34a-i; 25.29*

Frankliniella occidentalis (Pergande)

The western flower thrips is a major pest of greenhouse cucumber in Canada. It is widely distributed throughout the greenhouse industry and can be found wherever greenhouse cucumber crops are grown from British Columbia to Nova Scotia.

The host plant range for the western flower thrips is very broad and includes many field and greenhouse vegetables, ornamentals, weeds, berries and tree fruits.

Damage Both immature and adult western flower thrips feed by piercing the leaf surface with their mouthparts and sucking the contents of the plant cells. This causes the formation of silvery white streaks or specks on the leaves and blossoms, accompanied

by dark specks of fecal deposits (frass) *(22.34a).* Extensive thrips damage to the leaves reduces the photosynthetic ability of the plant, resulting in lower yield. Feeding damage also may be present on the fruit in the form of flecks and striations similar to those on the leaves *(22.34b).* This thrips also causes severe distortion or curling of cucumber fruit *(22.34c),* which lowers the grade of the fruit. Thrips damage can occur at any time in the growing season. In Alberta and British Columbia, the most severe problems occur in May and June. In Ontario, thrips impact is greatest in July and August, at which time feeding damage on the leaves and fruit, as well as damage from too frequent application of pesticides, can seriously decrease the yield and fruit quality.

The western flower thrips is a vector of tomato spotted wilt virus in pepper and tomato.

Identification In Canada, the thrips genus *Frankliniella* can be distinguished from other thrips genera by its eight-segmented antennae, well-developed setae on the anterior part of the thorax (prothorax) (a long pair anteriorly and shorter ones mid-laterally), and two longitudinal veins bearing two rows of setae on the forewings.

The western flower thrips (family Thripidae) adult (*22.34g; 25.29)* is winged, 1 to 2 mm in length, and females have a straw-brown head and thorax and a darker brown abdomen. Because of its small size, variation in color, and the existence of similar species, this thrips is impossible to identify with certainty at the species level without high-power magnification.

In greenhouses, the western flower thrips might be confused with the onion thrips, which is usually uniformly brown. However, the adult western flower thrips has two, very small divisions (annuli) at the tip of the antennae and a pair of dorso-anterior setae on the prothorax, whereas the adult onion thrips has only one terminal antennal annulus and lacks dorso- anterior prothoracic setae. Immatures of these and other thrips are impossible to identify to species.

Life history Female adults of the western flower thrips live up to 30 days and lay 2 to 10 eggs per day. Eggs are inserted individually into the plant's leaves, stems, and flowers and hatch in three to six days. There are two nymphal (or larval) instars that feed and mature on leaves and flowers. After six to nine days, the mature, second- instar nymph drops to the soil and enters the propupal and pupal stages *(22.34e,f).* The pupa is a non-feeding stage during which the wings and other adult structures form. Adults emerge after five to seven days and fly to host plants where they feed, mate and lay eggs. Development from egg to adult takes approximately 15 days at 25°C. Temperatures above 25°C and low humidities are optimal for development and longevity of the western flower thrips and the induction of its outbreaks. The adult is a weak flier and, under most conditions, it tends to hop and run rather than fly, but adult thrips can disperse throughout the greenhouse by flying. Adult thrips are transported on wind currents and enter greenhouses through vents and doorways. All thrips stages may spread between greenhouses by the movement of infested plants, soil, farm implements and picking tools, and on workers' clothing.

Management The western flower thrips can be effectively controlled using cultural procedures and biological control. Repeated introduction of the biocontrol agents usually is necessary, and both the pest and predators must be carefully monitored. Over-reliance on chemical control is unwise because thrips rapidly acquire resistance to insecticides.

Monitoring — Population monitoring for the western flower thrips is critical to the success of biological control and consists of several distinct steps. An early detection program should be implemented as soon as the crop is planted or earlier. For this, blue or yellow sticky traps *(22.34d)* are positioned evenly throughout the crop. The western flower thrips is more attracted to blue than to yellow, so blue sticky traps are preferred for monitoring the adult thrips population. Early detection provides adequate early warning of thrips.

Once biological control has started, predators and thrips both should be monitored by checking a minimum of 25 leaves per 2000 m^2 weekly. Commercial pest management monitoring services may be available for this. There should be no more than 10 immature thrips per leaf. The predators should be evenly distributed throughout the greenhouse. Once the predators are established, thrips populations can be expected to decline or remain stable. Lack of establishment of predators may indicate poor quality of predators or that pesticide residues are present on the crop or the greenhouse structures.

Blue or yellow sticky traps should be maintained to monitor population trends and detect immigration of adults, which may occur in June and August, particularly after hay harvesting in nearby fields.

Cultural practices — Commercially available blue or yellow sticky ribbons can be used for mass trapping as well as for monitoring, but other cultural practices also are effective in preventing outbreaks. For instance, at the beginning of the cropping season, the soil might be steam- sterilized to kill soil stages of the thrips. Greenhouse vents can be covered with very fine screening to prevent thrips infiltration from outdoors, weeds should be removed from around the perimeter of the greenhouse, and ornamentals should not be planted in close proximity to the greenhouse. Also, non-crop material, such as ornamental plants, should not be brought into the greenhouse.

If thrips are present at the end of the growing season, an infested crop should be treated with an appropriate insecticide, preferably a fumigant, prior to removing the plants. Then, all plant material should be removed and destroyed to prevent reinfestation. The greenhouse then should be heated for at least two days to control any remaining thrips. This is particularly important between spring and fall crops. During the winter, below-freezing temperatures in the greenhouse will not control the western flower thrips, but keeping the greenhouse heated for two or more days without any plants is an effective control practice.

Biological control — Several biocontrol agents are commercially available to control western flower thrips. They should be released immediately after the first thrips or thrips damage is seen in the greenhouse. Commercially available biocontrol agents

include the predatory mites *Amblyseius* (syn. *Neoseiulus*) *cucumeris* Oudemans and *Amblyseius* (syn. *Neoseiulus) barkeri* Schuster & Pritchard.

Amblyseius cucumeris (22.34h) is the mite most commonly used to keep the western flower thrips below economically damaging levels on cucumber. The mites are sometimes introduced when the crop is planted. This mite normally is sensitive to daylength and does not lay eggs during short-day conditions, such as occur between late September and late February, unless the greenhouse temperature remains at or above 21 °C. A non-diapausing strain now is commercially available and it has replaced the short-day sensitive strain. Releases should be at a rate of 50 to 100 mites per plant every one to two weeks until approximately 75% of the leaves have mites or until every leaf with a thrips also has a predatory mite. If more than 10% of the leaves have adult thrips, or more than 25% have immature thrips, the initial thrips population is too high, in which case growers should apply a non-residual insecticide and wait at least two weeks, depending on the insecticide, before releasing predatory mites.

A soil-dwelling predatory mite, *Hypoaspis* (syn. *Geolaelaps)* species *(22.31c)*, marketed as a biocontrol agent for fungus gnats and applied to the floor or growing media, also preys on propupae and pupae of the western flower thrips, reducing adult emergence by about 40 to 60%. This rate of predation is not sufficient to provide control, but this species of mite can contribute to the overall success of a biological control program for the western flower thrips.

The minute pirate bugs (*22.34i*) *Orius tristicolor* (White) and *O. insidiosus* (Say) also are effective biocontrol agents for western flower thrips and are available commercially. Greenhouse releases at the rate of one bug per one or two cucumber plants over a period of several weeks will provide control within four to six weeks after application. These bugs are effective from March throughout the growing season.

Chemical control — The western flower thrips is difficult to control with chemicals because adults and immatures feed in the crevices of blossoms and fruit and on leaf undersides, making chemical contact difficult. Moreover, the western flower thrips is becoming resistant to all insecticides registered for use on greenhouse cucumber crops in Canada, and it seems to acquire resistance very rapidly to new products. Nevertheless, chemical control may be necessary toward the end of the growing season.

Synthetic pyrethroid products are not compatible with predatory mites or any biocontrol agents. Also, elemental sulfur for control of powdery mildew may interfere with the success of the predatory mites. In Alberta, spray applications of sulphur at label rates have had minimal effect.

When using pesticides, several applications are needed. They should be spaced at approximately four-day intervals to provide adequate control of both adult and immature thrips.

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(Original by D.R. Gillespie and J.L.Shipp)

▶ 22.35 Other insect pests *Figs. 22.35a-d; 9.21*

Caterpillars
Cucumber beetles
- Spotted cucumber beetle *Diabrotica undecimpunctata howardi* Barber
- Striped cucumber beetle *Acalymma vittatum* (Fabricius)

Leafminers
- Chrysanthemum leafminer *Liriomyza trifolii* (Burgess)
- Vegetable leafminer *Liriomyza sativae* Blanchard

Onion thrips *Thrips tabaci* Lindeman
Plant bugs *Lygus* spp.

The above-mentioned insect pests occur sporadically on greenhouse cucumber crops in various parts of Canada.

Caterpillars

of various species can cause some degree of defoliation on greenhouse cucumber crops. They or the adult moths invade the greenhouse from outside and are usually only casual pests. Occasionally they may be a severe problem in individual greenhouses (see Greenhouse tomato, 25.30). Besides screening vents, application of the microbial insecticide *Bacillus thuringiensis* Berliner is the recommended control procedure for these pests.

Cucumber beetles

The spotted cucumber beetle and striped cucumber beetle (9.27) occasionally can be important to greenhouse cucumber crops because the adult beetles are efficient vectors of bacterial wilt. The striped cucumber beetle is the more important vector of the two species (see Cucurbits, 9.21). The adult beetles overwinter in weeds and trash, and become active in early spring. The disease can be transmitted to the crop at any time, but the beetles usually do not move into greenhouses until early to mid-summer. The best management strategy for cucumber beetles in greenhouse cucumber is cultural control by screening vents and maintaining a weed- and trash-free barrier around the greenhouse. Pesticides can be used but require application at least once per week, and they may cause plant injury and yield loss. No biocontrol agents are available for cucumber beetles.

Leafminers

The chrysanthemum leafminer (22.*35a,b)* and vegetable leafminer occur sporadically on greenhouse cucumber crops (see Greenhouse tomato, 25.28). The chrysanthemum leafminer is usually a problem only where greenhouse cucumber is grown in proximity to greenhouse flowers, particularly chrysanthemum. The vegetable leafminer is not yet a problem in greenhouse cucumber crops in British Columbia or Alberta, but it is more important than the chrysanthemum leafminer in Ontario. Control measures should be implemented as soon as the first leafminer is detected in the crop because leafminer populations can increase rapidly. Infestations can be controlled by pruning the mined leaves from the infested plants. Parasitic wasps are commercially available for biological control of leafminers (see Greenhouse tomato). Adult leafminers also can be controlled using insecticides, but leafminer populations are usually resistant.

Onion thrips

(see Onion, 13.27; see also Greenhouse tomato, 25.29) Before about 1985, the onion thrips (*22.35c*) was the only thrips pest on greenhouse cucumber. Since then, although still relatively common on greenhouse cucumber in all parts of Canada, it has been eclipsed by the western flower thrips. The onion thrips tends to be restricted to the lower strata of cucumber crops. It is less common in cucumber flowers and does not appear to be involved in fruit curling or to cause direct feeding-damage to the fruit. It is relatively easy to control with a variety of insecticides. Biocontrol agents for western flower thrips and fungus gnats also feed on onion thrips.

Plant bugs,

particulary various *Lygus* species (family Miridae), have recently become a problem on greenhouse cucumber crops. Information on their biology and control is limited. Adults and nymphs are relatively large, brown to green bugs (*22.35d*). They are very active at greenhouse temperatures. Adults and nymphs are sucking insects and feed on plant sap from stems, particularly near the tender growing tip of the plant. Feeding damage eventually may kill the growing tip, but the plant can generate a new growing tip from lateral shoots. This slows growth and can cause substantial yield loss. Flowers and developing fruit also may abort or be deformed, causing loss of quality and yield. Plant bugs migrate into greenhouses in late summer, being more of a problem to the fall crop. There are reports of plant bugs overwintering in greenhouses and being found on transplants in early spring. Treatment with insecticides is the current control recommendation for plant bugs.

(Original by D.R. Gillespie and J.L. Shipp)

MITE PESTS

▶ 22.36 Two-spotted spider mite *Figs. 22.36a-g; 22.36T1*

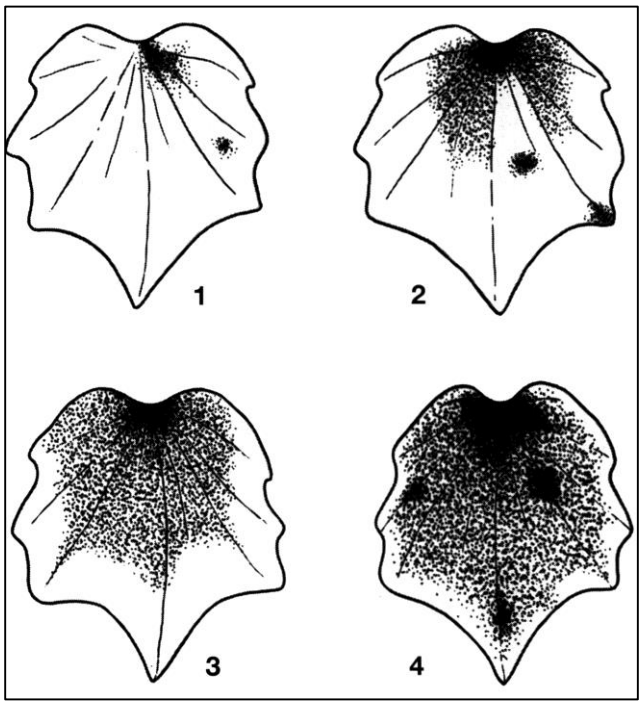
Tetranychus urticae Koch

The two-spotted spider mite can be found across southern Canada on a variety of crops, including fruit trees, vegetables, ornamentals and field, vine and berry crops. In greenhouses, hot, dry conditions favor its multiplication in the upper reaches of the crop in mid- to late summer.

The mite has a very broad host range, but greenhouse cucumber is a preferred host. Chickweed (*Stellaria* spp.) is an important weed host in the greenhouse.

Damage The two-spotted spider mite is a major pest of greenhouse cucumber. Outbreaks will cause moderate to severe losses and, in extreme cases, can destroy a crop. Spider mites feed by puncturing plant cells. This produces small, yellow or white, speckled feeding lesions that can coalesce and cause leaf death when extensive. Initial infestations may occur anywhere on the plant but are usual on leaf undersides. As populations increase, the greatest damage is generally on the canopy leaves because the adult females tend to move upward and the life cycle is faster at higher temperatures.

Spider mite damage (*22.36a-c*) is distinguished from thrips damage by the presence of fine webbing on leaf undersides (*22.36c*) and/or a silver sheen on damaged surfaces (22.*36b,c),* and by the absence of fecal deposits.



22.36T1 Two-spotted spider mite; indices (1-4) for leaf damage on greenhouse cucumber. Reprinted from *Biological Pest Control — The Glasshouse Experience*, N.W. Hussey & N.E.A. Scopes, eds. Copyright © 1985 by N.W. Hussey and N.E.A. Scopes. Used by permission of the publisher, Cornell Univ. Press.

Identification The two-spotted spider mite (family Tetranychidae) adult is 0.5 mm in length and is visible without magnification. During the growing season, it is pale yellowish-green with two distinct, bilaterally symmetrical black spots, one on either side of the body (*22.36e*). Overwintering adults are bright orange *(22.36f)* with a pair of dark spots. The eggs are small, 0.14 mm in diameter, spherical and white. The larval stage is small and white, and has three pairs of legs. Later stages have four pairs of legs (*22.36e,f*).

Life history Spider mites have five developmental stages: egg, larva, protonymph, deutonymph and adult. Adult females lay approximately 100 eggs on the lower leaf surface. The rate of oviposition is affected by relative humidity. At 20°C and a relative humidity of 36%, females lay an average of seven to eight eggs per day. At a relative humidity of 95%, this reduces to about five eggs per day. Development time from egg to adult is inversely dependent on temperature; 3.5 days at 32°C, 14.5 days at 21°C, and 21 days at 18°C. The two-spotted spider mite is very mobile when it is dispersing into a crop. Development usually takes place on the same leaf where the egg was deposited, and the mite confines itself to the infested plant as long as there is adequate food. Once the plant dies, or when the mite population is very high, the mites aggregate at the top of the plant or leaf tips and hang from the plant by dense “ropes” or silken strands. These silken strands and any mites on them are easily spread by attachment to people and equipment.

The adult females have a non-feeding stage (diapause) that is induced by short daylength and influenced by low temperature and reduction in host plant quality. Under short-day conditions, the normally pale yellowish-green females turn bright orange (*22.36d*), cease feeding and egg laying, and leave the plants to seek dark crevices in which to overwinter. Daylengths of less than 13 hours induce this behavior. Low temperatures, such as when greenhouse heating is shut down, and the presence of only senescing plant material for food facilitate diapause induction. High temperatures and succulent, growing plant material discourage diapause induction. A period of chilling is required to terminate diapause, and warm conditions induce the females to move from the wintering sites and seek new host plants. In general, hibernating females begin to emerge when the heat is turned on in the greenhouse, and these mites are the usual source of early spring infestations.

Management The two-spotted spider mite can be controlled effectively, using a combination of cultural practices and biological control.

Monitoring — Spider mite infestations in a crop are detected by routine examination of the leaves. As soon as the mites or their damage are found, predatory mites should be introduced at the appropriate rate. A damage-rating system has been devised in Britain. For successful control, only a few leaves in the greenhouse should be in damage indices 1 and 2 (*22.36T1*), and the average damage index should be much less than 1. Significant crop loss will occur at a mean damage index of 1.9, and a 40% loss can be expected five weeks after a damage index of 2.5. Growers who use predatory mites to control spider mites frequently

tolerate localized areas of high damage, particularly if the predators are numerous, because the infestation usually collapses quickly.

Cultural practices — Sanitation is one of the most important cultural practices to prevent spider mite infestations in greenhouses. Weeds should be removed from the vicinity of the greenhouse and a 3-m-wide, weed-free zone should be maintained around the perimeter. Movement of equipment, people and plant material from infested to non- infested areas should be restricted. If spider mites are present on the crop at the end of the season, the crop should be treated with a fumigant miticide before removal. All crop residue should be removed and destroyed to prevent reinfestation of the greenhouse. Particular attention should be given to spider mite infestations late in the season. Control at that time will reduce the number of overwintered mites available to infest the greenhouse in the spring.

Biological control — The predatory mite *Phytoseiulus persimilis* Athias-Henriot (*22.36g*) is an effective biological control agent for the two-spotted spider mite, being widely used in all areas of Canada where greenhouse cucumber is grown. To ensure success, the predatory mite must be introduced when the prey population is low. Mites should be introduced on young plants at the rate of one predator per plant, plus weekly additions of one predator per infested leaf until predators are present on every infested leaf. On large plants, introductions should be on a weekly or biweekly basis at a rate of 10 predators per plant, plus 10 per infested leaf. Uniform distribution of the predator is important for control, which can be expected within five weeks after the initial introduction. The miticide fenbutatin-oxide does not harm the predatory mite.

Survival and reproduction of the predatory mite decline under hot, dry conditions. Otherwise, it has a short, one- week life cycle compared to the spider mite, which takes two weeks to mature. Thus, the number of predatory mites can increase to the point that they overtake and eliminate the spider mite from the greenhouse. Recurrence of spider mites should be monitored carefully, and predatory mites should be re-introduced if necessary.

Chemical control — Spider mite outbreaks can be controlled using miticides, but it is important to treat populations when their numbers are low. For effective chemical control, all leaf surfaces must be covered with the pesticide. If moderate to severe, localized outbreaks of spider mites occur after periods of hot, dry weather, then a miticide can be used prior to the re-introduction of predators.

The two-spotted spider mite has acquired extensive resistance to most miticides. Resistance varies from region to region and between greenhouses. The selection of miticides must be determined on a case-by-case basis.

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BACTERIAL DISEASES

▶ 23.1 Butt rot (head rot) *Figs. 11.3a-d*

Pseudomonas fluorescens Migula
(syn. *Pseudomonas marginalis* (Brown) Stevens)

Butt rot is most often seen in lettuce plants growing under conditions of low light and excessive leaf wetness. It occurs both in soil and in soilless production by the nutrient film technique (NFT).

Symptoms Infected stems develop a black to green, firm rot that may spread along the veins of the lower leaves and progress down to the roots. Secondary organisms cause wilt or collapse of the plant and storage rots of marketed heads.

Causal agent (see Lettuce, pseudomonas diseases)

Disease cycle (see Lettuce, pseudomonas diseases, 11.3)

Management

Cultural practices — Most greenhouse lettuce cultivars are susceptible; however, severity varies among cultivars. Growers should adjust fertilizer programs to ensure that the plants are not unduly soft, and avoid frequent wetting of the foliage. Careful regulation of ventilation and heating helps to reduce humidity and prevent condensation, which favors bacterial diseases. In NFT production the disease is more severe when the system is contaminated by soil or when soil starter blocks are used for transplants.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.2 Stem rot

Pseudomonas cichorii (Swingle) Stapp
Pseudomonas cichorii has been implicated in a stem rot of hydroponically grown lettuce in Ontario.

Symptoms Near-mature plants in NFT production systems have firm, dark brown rot, and petioles of the inner leaves are streaked. This disease may be similar to varnish spot reported from California. Ontario isolates of *P. cichorii* are also pathogenic to chrysanthemum, celery and tomato.

Causal agent (see Lettuce, pseudomonas diseases)

Disease cycle (see Lettuce, pseudomonas diseases, 11.3)

Management (see butt rot, 23.1)

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.3 Other bacterial diseases *Figs. 11.1a,b*

There are a number of other diseases of greenhouse lettuce caused by pathogenic bacteria (see Lettuce, bacterial soft rots, 11.1). Control of these diseases in the greenhouse can be enhanced by proper heating, ventilation and sanitation.

(Original by J.G. Menzies and W.R. Jarvis)

FUNGAL DISEASES

▶ 23.4 Anthracnose (ring spot), fire of endive *Fig. 11.4*

Microdochium panattonianum Sutton, Galea & Price in Galea, Price & Sutton
(syn. *Marssonina panattoniana* (Berl.) Magnus)

This fungal disease, which is common in field lettuce, also affects greenhouse lettuce. It tends to occur in plants located under gutters and in other areas of the greenhouse where water drips onto the plants. It also is common in unheated greenhouses that have a history of monocropping to lettuce or endive. In endive the disease is called fire.

Symptoms (see Lettuce, anthracnose, 11.4)

Causal agent (see Lettuce, anthracnose)

Disease cycle (see Lettuce, anthracnose)

Management

Cultural practices — Control measures usually are not necessary, because of the infrequent occurrence of the disease. However, trimmings and trash piles should be properly composted or destroyed, because the fungus can survive for long periods in dry residues. Headerhouse floors, machinery, flats and other surfaces should be cleaned of soil and plant residues. Growing media should be disinfested before use.

Selected references

Galea, V.J., and T.V. Price. 1988. Survival of the lettuce anthracnose fungus *(Microdochium panattonianum)* in Victoria. *Plant Pathol.* 37:54-63.
Galea, V.J., T.V. Price and B.C. Sutton. 1986. Taxonomy and biology of the lettuce anthracnose fungus. *Trans. Br. Mycol. Soc.* 86:619-628.
Sutton, B.C., and M. Holderness. 1991. *Microdochium panattonianum.* IMI Desccriptions of Fungi and Bacteria, No. 1034. Internat. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.5 Bottom rot *Figs. 11.6a,b*

Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

In greenhouses, the fungus rapidly colonizes sterilized soil if hygienic practices are not strictly observed. It can be introduced in peat and loam composts and from contaminated planting trays and tools. Bottom rot is one of the major head rot diseases of field lettuce (see Lettuce, bottom rot, 11.6). Generally, it is less common in hydroponic crops than in those grown in soil.

Management

Cultural practices — Seedling flats should be raised on benches out of the range of splashing water or soil. Once the fungus has infested growing media, thorough disinfestation is necessary after the crop has been cleared. ***Rhizoctonia solani*** and most soil-borne pathogenic fungi can be eliminated by steam heat (60°C for 30 min). Above 80°C, microorganisms antagonistic to pathogens are destroyed and severe outbreaks can ensue.

Chemical control — Growers should disinfest growing media with registered fumigant chemicals, but methyl bromide should be avoided because lettuce tissues may accumulate excess bromine from treated soil.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.6 Damping-off, stunt *Figs. 23.6a,b*

Pythium spp.
Other fungi

In greenhouses *Pythium* species are the most important causal agents of this disease (see Lettuce, damping-off, 11.7). Once the disease is established *(23.6a,b),* fungicides may not give satisfactory control. Although pythium damping-off in soil is usually associated with chilled plants, lettuce produced by the nutrient film technique (NFT) is attacked by *P. aphanidermatum* at temperatures above 23°C, and *P. dissotocum* Drechs. is a dominant pathogen at 17 to 22°C. *Pythium* species can attack the tiny feeder roots and cause substantial losses in yield without obvious damage to the root system.

Management

Cultural practices — Damping-off can be controlled by treating seed with a protectant fungicide and by sowing into a pasteurized growing medium. Growers should avoid overwatering and overcrowding seedlings. Adequate ventilation helps keep the growing medium dry. Water for irrigation should be warm. (See bottom rot, 23.5, for methods of disinfesting growing media.) In soilless cultivation, soil starting blocks should never be used and strict hygiene should be maintained to avoid contamination by soil. Species of ***Pythium*** are often present in water from creeks, wells and outdoor reservoirs, so growers using these sources for irrigation should consider installing filters and an ultraviolet water sterilizer or some other type of disinfestation equipment.

Chemical control — Post-emergence fungicide treatment may be effective in controlling the spread of damping-off fungi.

Selected references

Bates, M.L., and M.E. Stanghellini. 1984. Root rot of hydroponically grown spinach caused by *Pythium aphanidermatum* and *P. dissotocum. Plant Dis.* 68:989-991.

Van der Plaats-Niterink, A.J. 1981. Monograph of the Genus *Pythium. Stud. Mycol.* No. 21. Centraalbureau v. Schimmelcultures, Baarn, The Netherlands. 242 pp.

Stanghellini, M.E., and W.C. Kronland. 1986. Yield loss in hydroponically grown lettuce attributed to subclinical infection of feeder rootlets by *Pythium dissotocum. Plant Dis.* 70:1053-1056.

Zinnen, T.M. 1988. Assessment of plant diseases in hydroponic culture. *Plant Dis.* 72:96-99.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.7 Downy mildew *Figs. 11.8a,b*

Bremia lactucae Regel

This disease (see Lettuce, downy mildew, 11.8) can be a major problem on greenhouse lettuce.

Management

Cultural practices — Greenhouse climate control is important to prevent the formation and retention of free water on plant surfaces and to avoid periods of high humidity. Dew should be prevented from forming on the foliage by keeping the night temperature above 16°C and by expelling humid air through ventilation. Growers should remove crop residues completely from the greenhouse, use steam for disinfesting growing media, and avoid planting new lettuce crops near those showing symptoms of the disease. Overhead irrigation should be curtailed where downy mildew is a problem.

Resistant cultivars — These are available and should be evaluated on a local basis to determine their suitability and resistance to the locally prevalent race(s) of *B. lactucae.*

Chemical control — Chemical disinfestation of growing media can be useful, but methyl bromide should be avoided because lettuce tissues accumulate excess bromine from the soil after fumigation. The daily dietary intake of bromine is regulated for health reasons, and heavy consumption of leafy vegetables from bromine-fumigated soil is not generally recommended. Foliar fungicide sprays should be applied as soon as symptoms appear.

Selected references

Crute, I.R. 1988. Lettuce downy mildew: A case study in integrated control. Pages 30-53 *in* K.J. Leonard and W.E. Fry, eds., *Plant Disease Epidemiology.* Vol. 2. McGraw-Hill Publ. Co., New York, New York. 300 pp.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.8 Drop *Figs. 11.9a-e*

Sclerotinia minor Jagger
Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

Drop (see Lettuce, drop, 1 1.9) is common in greenhouse lettuce grown in soil under conditions of high humidity and temperatures above 22°C. The disease can be common during the summer and it is not easy to control, particularly if sclerotia infest the soil. The disease is not common in hydroponic production but can occur if ascospores are carried into greenhouses by air currents from trash piles outside.

Management

Cultural practices — Diseased plants and trimmings should be removed from greenhouses, taking care to ensure that sclerotia of the fungi do not remain. Chemical and steam disinfestation of soil can be used to kill the sclerotia (see bottom rot, 23.5).

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.9 Gray mold *Figs. 23.9; 11.10a-f*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold (see Lettuce, gray mold, 11.10) is the most common disease of greenhouse lettuce and is often said to be a disease of poor management. Upright-growing types of lettuce are generally less susceptible than cabbage types in which the cotyledons and outer leaves often lie on the soil.

Symptoms In greenhouses, the pathogen can infect seedlings that have been weakened by damping-off organisms. A soft brown rot (25.9) may appear on the stems and at the bases of older leaves on older plants and it may extend through the stems. When this occurs, the leaves turn gray-green and the plant eventually wilts and dies. Under humid conditions, the lesions become covered by a gray mold (*11.10c,f).* Black sclerotia form on decaying tissue (11.10*d).*

Management

Cultural practices — Cultural practices should promote optimum growth of lettuce while avoiding damage to plant tissues. To reduce mechanical damage, lettuce seedlings should be transplanted before they get too large. Prompt removal of crop residue and disinfestation of growth media help limit infection and spread. Proper heating and ventilation of greenhouses will reduce periods of high humidity, particularly at night, thereby minimizing sporulation and infection by the fungus. Growers should avoid overcrowding and excessive use of nitrogen fertilizer. Overhead irrigation should not be used, particularly in poorly ventilated greenhouses.

Chemical control — Growers should apply a registered fungicide before the onset of disease when cool (15 to 20°C) and moist conditions prevail.

Selected references
Jarvis, W.R. 1977. Botryotinia and Botrytis Species: Taxonomy, Physiology and Pathogenicity. Can. Dep. Agric. Res. Br. Monogr. 15. 195 pp.
Morgan, W.M. 1954. The effect of night temperature and greenhouse ventilation on the incidence of Botrytis cinerea in a late planted tomato crop. Crop Prot. 3:243-251.
(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.10 Powdery mildew *Fig. 23.10*

Erysiphe cichoracearum DC.

Powdery mildew (see Lettuce, powdery mildew, 11.12) has occasionally been a serious disease of lettuce grown in nutrient film hydroponic greenhouses in Ontario. Abundant conidia form on the white colonies on leaves *(23. JO)* and are dispersed on air currents to plants all over the greenhouse. Cleistothecia and thick-walled mycelium in dry crop residues are suspected to be the main survival stages between successive crops.

Management

Cultural practices — Crop residues should be removed and disposed of regularly.

Selected references

Crute, I.R., and I.G. Burns. 1983. Powdery mildew of lettuce *(Lactuca sativa). Plant Pathol.* 32:455-457.
Dhanavantari, B.N., and W.R. Jarvis. 1985. Powdery mildew *(Erysiphe cichoracearum)* of greenhouse lettuce in Ontario. *Plant Dis.* 68:177.
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(Original by J.G. Menzies and W.R. Jarvis)

VIRAL AND VIRAL-LIKE DISEASES

▶ 23.11 Aster yellows *Figs. 11.15a,b*

Aster yellows mycoplasma-like organism

This disease (see Lettuce, aster yellows, 11.15) is a minor problem in greenhouse lettuce and only in poorly managed crops where weeds are prevalent in and around the greenhouse, and where leafhoppers are permitted to enter.

(Original by W.R. Jarvis)

▶ 23.12 Big vein *Fig. 11.16*

Big-vein virus

This virus (see Lettuce, big vein, 11.16) is not common on greenhouse lettuce, but if plants become infected while being grown in a nutrient film system, the pathogen and its fungal vector *(Olpidium brassicae* (Woron.) Dangeard) can quickly spread through a crop.

Management

Cultural practices — Partial control can be obtained by disinfestation of growing media, watering systems and all equipment, but this will not completely eradicate the vector or the virus. Growers of hydroponic lettuce should consider the installation of a water sterilizer if water is drawn from a creek or outside reservoirs.

Selected references

Campbell, R.N., A.S. Greathead and F.V. Westerlund. 1980. Big vein of lettuce: Infection and methods of control. *Phytopathology* 70:741-746.
Tomlinson, J.A., and E.M. Faithfull. 1980. Studies on the control of lettuce big-vein in recirculated nutrient solutions. *Acta Hortic.* 98:325-332.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.13 Cucumber mosaic

Cucumber mosaic virus

This disease (see Cucurbits, cucumber mosaic, 11.18) fluctates from season to season. On greenhouse lettuce, symptoms vary with the stage of growth at the time of infection, time of year, cultivar and strain of the virus. Transmission occurs mechanically or by aphid vectors. The virus is not known to be seed transmitted in lettuce.

Symptoms Infected lettuce plants are stunted, with a yellow mottle or necrotic spotting on the leaves. These symptoms are indistinguishable from those of lettuce mosaic. Occasionally, the two viruses infect the same plant, resulting in severe stunting, yellowing and necrosis.

Management

Cultural practices — Growers should rogue infected plants and control aphids (see aphids, 23.17).

Selected references

Francki, R.I.B., D.W. Mossop and T. Hatte. 1979. Cucumber mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 213. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.14 Lettuce mosaic *Figs. 23.14; 11.17a,b*

Lettuce mosaic virus

Lettuce mosaic (see Lettuce, lettuce mosaic, 11.17) is the most important viral disease of greenhouse lettuce. It can infect plants at any stage, affecting both size and quality *(23.14).* It is most severe where lettuce is grown successively in blocks, the virus being transmitted from older to younger crops by aphids. As aphid populations increase, virus spread becomes more rapid. Commercial diagnostic kits are available for lettuce mosaic virus.

Management

Cultural practices — Control of this virus in the greenhouse involves the use of mosaic-indexed seed combined with a scheme of isolating blocks of lettuce to minimize the spread of the virus from crop to crop. Diseased plants should be removed and destroyed and aphids should be controlled (see aphids, 23.17).

Selected references
Grogan, R.G. 1980. Control of lettuce mosaic with virus-free seed. *Plant Dis.* 64:446-449.
Tomlinson, J.A. 1970. Lettuce mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 9. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 4 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 23.15 Other viral diseases *Fig. 23.15*

Beet western yellows virus
Lettuce infectious yellows virus
Tomato spotted wilt virus

These viruses (see Lettuce, other viral diseases, 11.18) are not common in greenhouses, but they can cause damage if they become established in reservoir plant hosts of the viruses and their vectors. Commercial diagnostic kits are available for the lettuce strain of tomato spotted wilt virus *(23.15)* and for beet western yellows virus.

Management

Cultural practices — Growers should remove crop residues from the vicinity of the greenhouse, avoid growing ornamental plants in lettuce greenhouses, and control insect vectors and weeds.

Selected references
Duffus, J.E. 1972. Beet western yellows virus. CMI/AAB Descriptions of Plant Viruses, No. 89. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 4 pp.
Ie, T.S. 1970. Tomato spotted wilt virus. CMI/AAB Descriptions of Plant Viruses, No. 39. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 4 pp.
Walkey, D.G.A., and D.A.C. Pink. 1990. Studies on resistance to beet western yellows virus in lettuce (*Lactuca sativa*) and the occurrence of field sources of the virus. *Plant Pathol.* 39:141-155.
Van Doist, N. Huijberts and L. Bos. 1983. Yellows of glasshouse vegetables, transmitted by *Trialeurodes vaporariorum. Neth. J. Plant Pathol.* 89:171-184.

(Original by J.G. Menzies and W.R. Jarvis)

NON-INFECTIOUS DISEASES

▶ 23.16 Tipburn *Figs. 23.16; 11.19c*

This is a major disease of greenhouse lettuce (see also Lettuce, tipburn, 11.19).

Symptoms Tipburn affects the inner leaves of head lettuce and results from calcium deficiency in the growing tissues of the inner leaves. The first symptoms are necrotic spots near the leaf tips that expand until the entire edge of the leaf is brown *(23.16, 11.19c).* Many inter-related factors contribute to calcium uptake and tipburn.

Management The condition can be reduced to some extent by assuring that calcium levels in the soil are high relative to competing elements, such as potassium and magnesium, by reducing nitrogen applications to limit growth, especially during warm weather, by harvesting slightly before maturity and by keeping the nighttime humidity high in the greenhouse. Cultivars differ in tolerance to tipburn.

(Original by J.G. Menzies and W.R. Jarvis)

INSECT PESTS

▶ 23.17 Aphids *Figs. 8.39; 11.24T1,T2;15A.14; 16.41; 16.43T1*

Black bean aphid *Aphis fabae* Scopoli
Cabbage aphid *Brevicoryne brassicae* (L.)
Green peach aphid *Myzus persicae* (Sulzer)
Lettuce aphid *Nasonovia ribisnigri* (Mordvilko)
Pea aphid *Acyrthosiphon pisum* (Harris)

These aphids are widely distributed in Canada. Winged females develop and disperse to new host plants during early summer. Once air-borne, they may be blown long distances, eventually entering greenhouses wherever lettuce is grown. They may be black, yellow, or pink but most are a shade of green. They often develop large colonies on the undersurface of leaves. A colony can consist of winged and wingless adults and nymphs in various sizes and stages of growth.

All of these aphids attack lettuce and other vegetable crops. They overwinter as eggs on woody plants.

Damage Aphid populations can increase very quickly, particularly under warm, moist conditions in greenhouses. Their feeding can discolor the foliage, curl the leaves and damage developing buds. The plants may be covered by their honeydew and molted skins (exuviae), and large numbers of aphids can cause severe yield reductions or even crop failure. By their presence, even small numbers may make the crop unmarketable.

Some of the above aphids may transmit viruses to greenhouse lettuce.

Life history (see Crucifers, 8.39; Lettuce, 11.24; Pea, 15A.14; Potato, 16.41, 16.43)

Management The short time from seeding to harvest makes biological control impractical and, because the entire above-ground part of the plant is sold, high levels of chemical control are necessary for aphids infesting greenhouse lettuce. Early infestations must be controlled to avoid later crop damage, and infestations close to harvest must be controlled to avoid contamination of the marketable produce.

Cultural practices — Cultural practices are very important in reducing or eliminating aphid infestations. Growers should screen greenhouse vents, maintain a weed- and garden-free area around the greenhouse, and refrain from growing woody and other host plants in the same greenhouse.

Chemical control — The usual procedure is to apply chemical insecticides as high volume sprays. A number of applications may be needed for satisfactory control.

(Original by R.A. Costello)

▶ 23.18 Caterpillars *Figs. 8.40b-f*

Cabbage looper *Trichoplusia ni* (Hübner)
Other caterpillars

Various species of caterpillars, especially the cabbage looper (see Crucifers, 8.40), can be pests of greenhouse lettuce. Their life histories in the greenhouse are similar to those on field vegetables but shorter and the number of seasonal generations is greater.

Caterpillars feed on a wide range of plants including many vegetable crops.

Damage Caterpillars eat large holes in lettuce foliage. Once the inner leaves are damaged, the plant is unmarketable.

Management

Cultural practices — Vents should be screened, and doorways and other openings should be kept closed during the late evening and nighttime to exclude the adult, egg-laying moths.

Biological control — Caterpillars in greenhouses can be effectively controlled by use of the bacterium *Bacillus thuringiensis* Berliner. Additional feeding damage can be expected after application of the bacterium before the pests are killed. Growers should apply the microbial biocontrol agent as soon as caterpillars or their damage are detected on lettuce leaves.

(Original by R.A. Costello)

▶ 23.19 Other insect pests *Figs. 22.31 a-c; 26.29T1*

Fungus gnats

Fungus gnats Several species of fungus gnats (see Greenhouse cucumber *(22.31a,b)* and Mushroom (*26.29T1*)) occur in greenhouse lettuce wherever it is grown. Fungus gnats are not usually a problem in the production of this crop. The main problem is that they annoy people working the crop.

Fungus gnats can be controlled during preparation of the growth medium, whether by steam sterilization or by chemical fumigation.

(Original by R.A. Costello)

OTHER PESTS

▶ 23.20 Slugs *Figs. 11.27a-c*

Various species of slugs (see Lettuce, 11.27) are widely distributed throughout Canada, thriving in areas of moderate temperature and high humidity. These conditions occur in greenhouses that receive liberal amounts of overhead irrigation. Slug eggs, immatures and adults usually are brought into the greenhouse on material formerly stored outdoors. They are usually a minor and easily controlled pest of greenhouse lettuce. Slugs feed on a wide range of plants, including many vegetables and weeds. Lettuce is highly favored.

Damage Silvery slime trails, which can be found on damaged plants as well on the soil surface, distinguish slug damage from that caused by cutworms and other caterpillars. Roots fed on by slugs will have smooth-sided pits, 3 to 12 mm deep and usually less than 12 mm in diameter. Foliage damage typically involves removal of tissue between the veins. On greenhouse lettuce, leaf skeletonization can be extensive.

Management Routine steam-sterilization of greenhouse beds should kill all life stages of slugs. Although some organisms prey on slugs, none is available commercially, so poison bait is the only other control option in greenhouse lettuce.

Chemical control — Commercial metaldehyde-based preparations are available for use at the base of plants but not on the foliage. These products paralyze slugs for about 48 hours. Under moist greenhouse conditions, slugs may recover and escape. However, the chemical induces excessive production of slime, revealing the presence of poisoned slugs that can then be hand-picked and destroyed in soapy water. There is no reported resistance of slugs in Canada.

(Original by R.A. Costello)

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Hussey, N.W., and N.E.A. Scopes, eds. 1985. *Biological Pest Control — The Glasshouse Experience.* Cornell Univ. Press, Ithaca, New York. 240 pp.

Jarvis, W.R. 1992. *Managing Diseases in Greenhouse Crops.* APS Press, St. Paul, Minnesota. 280 pp.

Patterson, C.L., R.G. Grogan and R.N. Campbell. 1986. Economically important diseases of lettuce. *Plant Dis.* 70:982-987.

Steiner, M.Y., and D.P. Elliott. 1987. *Biological Pest Management for Interior Plantscapes.* Alberta Environmental Centre, Vegreville, Alberta. 30 pp.

FUNGAL DISEASES

▶ 24.1 Damping-off *Fig. 24.1*

Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Damping-off is common and can be severe where greenhouse hygiene is poor. It is more prevalent in soil-based media than in soilless mixes or rockwool. *Pythium* spp. and *R. solani* can attack many species of vegetable crops.

Symptoms Symptoms of damping-off of pepper are the same as those of greenhouse tomato. Pre-emergence damping-off has no above-ground symptoms,, as plants are killed before emerging. This problem usually is seen as well- defined areas in seedbeds and flats that are devoid of plants. Post-emergence damping-off is characterized by falling over of young seedlings *(24.1).* Occasionally, large groups of seedlings in beds or flats are killed. Older plants can also be attacked and may die if the disease is severe.

When attacked by *Pythium* spp., stems develop a wet, brown decay at the base; stems attacked by *R. solani* remain drier and are constricted and brown (wirestem symptom). Infected plants subsequently die.

Causal agents (see Bean, root rots, damping-off, seed decay, 15B.4; Beet, pythium and rhizoctonia root rots, 5.7, 5.8; and Carrot, cavity spot, 6.8, and pythium root dieback, 6.13)

Disease cycle (see Greenhouse tomato, damping-off, 25.7) *Pythium* species, the chief causal agents, are favored by prolonged cool, damp growing conditions, excess nitrogen in growing media, and overcrowding of seedlings.

Rhizoctonia solani can cause severe damping-off, wirestem and root rot of mature plants, especially in warm, moist, acidic growing media.

Management

Cultural practices — Growers should follow practices that favor rapid emergence and vigorous growth. Seedlings should be watered as lightly as possible in the morning so they can dry before the end of the day. They should be raised on benches with adequate ventilation and drainage. Seedlings in rockwool plugs or other soilless substrates should be kept on clean, disinfested benches, well away from the ground and water-splashed soil. They should never be below hanging baskets of ornamentals.

Chemical control — Growers should use pathogen-free or fungicide-treated seed and sow into growing media that have been disinfested by steam, chemical fumigation or incorporation of fungicides. Alternatively, inert growing media such as rockwool can be used. Further fungicide treatment of seedlings in the form of drenches may be useful.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 24.2 Fusarium stem and fruit rot *Figs. 24.2a-c*

Fusarium solani (Mart.) Sacc.
(teleomorph *Nectria haematococca* Berk. & Broome)

In Canada, this disease was reported from pepper in commercial greenhouses in Ontario and British Columbia in 1991. Losses in fruit yield and plants were approximately 5%. *Fusarium solani* can attack a wide variety of plants including most greenhouse vegetables. Many physiologic races adapted to specific hosts have been recognized.

Symptoms Soft, dark brown or black lesions are formed on the stem, usually at nodes or wound sites *(24.2a).* These lesions may eventually develop orange to red spots, which are the fruiting bodies of the fungus *(24.2b).* Stem lesions can kill the plant. Pepper fruits *(24.2c)* may also develop black, water-soaked lesions beginning around the calyx. The lesions grow, coalesce and spread down the sides of the fruit. Copious mycelial growth of the pathogen occurs under humid conditions.

Causal agent *Fusarium solani* generally has single-celled, oval to kidney-shaped microconidia which vary from sparse to abundant. Macroconidia are abundant, stout, thick-walled and generally cylindrical. The dorsal and ventral surfaces are parallel for most of their length. The apical cell is blunt and rounded, or it is distinctly foot-shaped or notched. Conidiophores are unbranched but possess branched monophialides. Chlamydospores are formed singly or in pairs and are numerous.

On potato-dextrose agar, *F. solani* grows rapidly with abundant aerial mycelium. The agar surface quickly becomes covered with confluent sporodochia that give the appearance of pionnotes and color the surface cream, blue-green or blue but never orange. The undersurface is generally colorless but may be dark violet.

The pathogen is distinguished from *Fusarium oxysporum* by the morphology of the macroconidia, the elongate monophialides bearing microconidia, and the distinctive cream, blue-green or blue color of colonies on potato-dextrose agar. Some isolates form abundant perithecia in culture on potato-carrot agar, mostly on the sides of the petri dish.

Disease cycle *Fusarium solani* is an extremely common inhabitant of soils in Canada and is frequently saprophytic. It can invade pepper stems at the nodes or at the soil line, taking advantage of wounds created by pruning or salt damage. Rapidly growing, succulent crops are the most susceptible, as are ripening fruit compared to green fruit. Fruit that is damaged, especially around the calyx, is very susceptible to infection. The rot can continue in storage. Healthy, undamaged fruit is not usually attacked. Fallen or aborted fruit and senescent flowers may be colonized by the fungus.

Management

Cultural practices — Good crop hygiene and pruning by clean-cutting will help to control this disease. Diseased plants and fruit should be removed from the greenhouse and buried. If the disease is severe, the fruit should be picked at the green stage. Rockwool blocks should not be allowed to dry out at the top because damaging levels of evaporated fertilizer salts may accumulate around the stem base and thus favor infection. At the end of the growing season, greenhouses should be thoroughly cleaned and disinfested. If the crop was grown in soil, the beds should be disinfested. Soilless growing media should be discarded far away from the greenhouse or buried.

Selected references

Booth, C., and J.M. Waterston. 1964. *Fusarium solani.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 29. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Nelson, P.E., T.A. Toussoun and W.F.O. Marasas. 1983. *Fusarium Species: An Illustrated Manual for Identification.* The Pennsylvania State University Press, University Park, Pennsylvania. 193 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 24.3 Gray mold *Figs. 25.12a-c*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold can infect leaves, stems and fruits of greenhouse pepper. It is widespread and can be severe in poorly managed greenhouses. It is not a problem where thorough hygiene and good cultural practices are observed. *Botrytis cinerea* can attack many plant species (see Lettuce, gray mold, 11.10).

Symptoms On greenhouse pepper, the first symptoms are often noticed when a lesion girdles the stem, which then collapses. The lesions are olive-green, sunken and soft with distinct margins. Leaf and stem lesions resemble those of gray mold on tomato

(25.12a-c). Infection can cause the entire fruit to rot, usually from the calyx end. The characteristic gray mold of the pathogen can develop on affected fruit, especially if the skin within a lesion is broken.

Causal agent (see Lettuce, gray mold, 11.10)

Disease cycle (see Lettuce, gray mold, 11.10). The fungus is favored by high humidity, invading the host through wounds in healthy tissue and through senescent tissue such as old flower parts.

Management (see Greenhouse tomato, gray mold, 25.12)

Cultural practices — Balancing heating and ventilation to maintain a relative humidity of 70 to 80% helps to prevent epidemics. It is essential to prevent dew forming on the plants, which should never be watered overhead. Gray mold is a disease of cool conditions (12 to 16°C), and thus adequate heat should be maintained without large fluctuations that cause dew to form. Crop residue should be removed and destroyed unless required for the survival of biocontrol agents, such as *Encarsia formosa* Gahan, a parasite of the greenhouse whitefly, in which case it should be kept dry to prevent colonization and sporulation of the fungus.

(Original by J.G. Menzies and W.R. Jarvis)

VIRAL DISEASES

▶ 24.4 Cucumber mosaic *Fig. 18.17*

Cucumber mosaic virus

Cucumber mosaic is a minor disease of greenhouse pepper, but has the potential to become a serious problem. The virus has a wide host range (see Greenhouse cucumber, cucumber mosaic, 22.20).

Symptoms Cucumber mosaic causes a severe mottling on pepper foliage, and older leaves may exhibit large, necrotic rings *(18.17).* Fruit may be malformed, and conspicuous yellow, concentric rings or spots may occur on green fruit.

Causal agent (see Greenhouse cucumber, cucumber mosaic, 22.20)

Disease cycle (see Greenhouse tomato, cucumber mosaic, 25.18)

Management (see Greenhouse tomato, cucumber mosaic)

Selected references

Francki, R.I.B., D.W. Mossop and T. Hatta. 1979. Cucumber mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 213. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

(Original By J.G. Menzies)

▶ 24.5 Pepper mild mottle *Figs. 24.5a,b*

Pepper mild mottle virus

This disease occurs in virtually every country in the world where peppers are grown as a greenhouse crop. It was first observed in Canada on field-grown pepper in Richmond, British Columbia, in 1985. At the time, it was a minor problem. However, in 1990 it was detected in greenhouse pepper in British Columbia's Fraser Valley and, in the course of the growing season, became a serious problem in three greenhouses. The estimated loss in the 1990 growing season was $1.2 million. The virus reappeared in a number of greenhouses in 1991, but the extent of losses is not known. British Columbia is the only province in which pepper mild mottle has been reported to date.

The virus systemically infects all *Capsicum* spp., including sweet peppers and hot peppers. Other species in the Solanaceae are susceptible, but not tomato or *Nicotiana glauca.*

Symptoms It is difficult to detect the disease in pepper foliage, because it causes a mild mottle that can be mistaken for other disorders. New growth shows more distinctive symptoms than older leaves *(24.5a).* Interveinal yellowing, especially at the basal half of the leaf, mottling and growth reduction may be evident. The most conspicuous symptoms are on the fruits *(24.5b)*, which may have distinct bumps, pointed tips and chlorotic or necrotic depressed areas. Necrotic, depressed areas frequently occur in the crease at the calyx end.

Causal agent Pepper mild mottle virus is a member of the tobamovirus group. The virus particles appear identical to those of tobacco mosaic virus, the type member of the group. Pepper mild mottle virus is serologically related to several members of the tobamovirus group. It can be distinguished from other tobamoviruses that infect pepper on the basis of its serological reactions and its response when mechanically inoculated to a range of diagnostic indicator hosts. The virus multiplies in inoculated leaves of *Nicotiana tabacum* cv. Samsun and most tomato cultivars, but it does not become systemic. This distinguishes it from both tobacco mosaic virus and tomato mosaic virus.

Disease cycle The epidemiology depends to a large extent on the initial source of infection. Preliminary evidence in British Columbia indicates that the initial infection source may be contaminated or infected seed. This virus, like tomato mosaic virus, is seed-borne, either on the seed coat (contaminated) or in the endosperm (infected). Pepper mild mottle virus can be eradicated from the seed coat by treatment with acid, and virtually all sources of commercial pepper seed are treated with acid before distribution. However, a very small percentage of the seed harvested from an infected mother plant may carry the virus in a dormant state in the endosperm. This infection is not eradicated by seed treatment. The virus becomes systemic in seedlings arising from infected seed and such seedlings constitute a primary source of inoculum. Unless extreme care is used, one infected seedling in a flat could infect several seedlings at the time of transplanting. These, in turn, could be sources of secondary infection during the course of tending the crop from seedling to fruit production.

Once a disease outbreak has occurred in a greenhouse complex, the virus may survive for several months on plant debris and on the surface of equipment. In such instances, the primary infection source for a new crop may be surviving virus from the previous crop. It is speculated that pepper mild mottle virus could spread in water, which would be a concern where recirculating systems are used.

Management

Monitoring — Growers should inspect their plants at frequent intervals during the growing season and immediately rogue out any that are suspected of carrying the virus.

Cultural practices — Seed should be obtained from a reliable source. Seed producers are becoming aware of the problem and it is expected that virus-free seed will become available. Growers should not save their own seed unless the growing crop has been certified as free of the disease by an experienced advisor.

The fact that the virus may be seed-borne means that particular care should be taken at the transplanting stage. Seedlings should be sprayed with a skim milk solution a day before transplanting and workers should dip their hands in skim milk at frequent intervals during transplanting (see Greenhouse tomato, tobacco mosaic, 25.20). Care should be taken to minimize spread of the virus during growth of the crop. Any suspicious plants should be carefully removed from the greenhouse and employees should dip their hands in skim milk when working in those areas. All crop debris, growth bags and rooting media should be removed from the greenhouse at the end of the growing season. The entire greenhouse interior, including ceilings, walls, supports, wires and walkways should be pressure washed and scrubbed with a disinfestant such as quaternary ammonium.

Resistant cultivars — Sources of resistance have been identified, and it is anticipated that commercial cultivars that are resistant or immune to pepper mild mottle virus will be available within a few years. Cultivars with TM2 resistace, e.g. Samathan, Cubico and others, can become infected, but do not express symptoms unless infected when young or with high levels of the pathogen.

Selected references

Wetter, C., and C. Conti. 1988. Pepper mild mottle virus. AAB Descriptions of Plant Viruses, No. 330. Assoc. Appl. Biol., Wellesbourne, Warwick, U.K. 4 pp.

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(Original by R. Stace-Smith and L.S. MacDonald)

▶ 24.6 Tobacco mosaic

Tobacco mosaic virus

This disease occurs on greenhouse pepper worldwide but is not common in Canada. Tobacco mosaic virus affects more than 150 plant genera, primarily herbaceous dicotyledons, including greenhouse tomato (see Greenhouse tomato, tobacco mosaic, 25.20; and Tomato, eggplant, pepper, tomato mosaic, 18.18).

Symptoms Symptoms vary with plant cultivar, virus strain and environmental conditions. The first symptoms on green-house pepper are necrosis along the main veins, with wilting and defoliation. Subsequent growth from lateral buds shows a mosaic and leaf distortion, but plants are rarely killed. Affected fruit is mottled and rough in appearance and, in severe cases, necrotic areas may occur on the surface.

Causal agent (see Greenhouse tomato, tobacco mosaic, 25.20)

Disease cycle (see Greenhouse tomato, tobacco mosaic)

Management

Cultural practices — Most of the control recommendations for greenhouse tomato (see Greenhouse tomato, tobacco mosaic) also can be applied to greenhouse pepper.

Resistant cultivars — Most cultivars of greenhouse pepper are susceptible to tobacco mosaic. However, a few resistant cultivars, including Cubico and Samantha, are available.

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(Original by J.G. Menzies, W.R. Jarvis and R.J. Howard)

▶ 24.7 Tomato mosaic

Tomato mosaic virus

Tomato mosaic is generally of minor importance in greenhouse pepper. This virus causes symptoms that resemble those caused by tobacco mosaic. Tomato mosaic virus also attacks greenhouse tomato (see Greenhouse tomato, tomato mosaic, 25.21). Laboratory tests are necessary to distinguish clearly between strains of tobacco mosaic and tomato mosaic viruses. Inoculations to indicator plants also help in the diagnosis.

Symptoms Symptoms vary with temperature, daylength, light intensity, plant age and cultivar. In general, pepper plants with tomato mosaic may show severe leaf necrosis and abscission, chronic mosaic and stunting.

Causal agent (see Greenhouse tomato, tomato mosaic, 25.21)

Disease cycle (see Greenhouse tomato, tomato mosaic)

Management Control of tomato mosaic in greenhouse pepper is similar to that in greenhouse tomato.

Cultural practices — Growers should use pathogen-free seed and apply seed treatments to kill the virus. Infected plants and crop residues should be removed, and strict sanitation practices observed.

Selected references
Hollings, M., and H. Huttinga. 1976. Tomato mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 56. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.
Pategas, K.G., A.C. Schuerer and C. Wetter. 1989. Management of tomato mosaic virus in hydroponically grown pepper *(Capsicum annuum). Plant Dis.* 73:570-573.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 24.8 Tomato spotted wilt *Figs. 24.8a-c*

Tomato spotted wilt virus

This virus infects greenhouse pepper but it is not a widespread problem. It could be a serious threat to pepper crops if thrips were present, particularly the western flower thrips, which is the principal vector. Alternative hosts, including dahlias, impatiens and other ornamentals, are important sources of infection. Infected crops should be isolated and destroyed. This virus has a wide host range that includes many vegetables, ornamentals and weeds (see Greenhouse tomato, 25.22; and Tomato, eggplant, pepper, 18.19).

Symptoms Lesions surrounded by a black margin may form on stems of infected plants *(24.8a).* This may lead to branch dieback and a loss of leaders. Leaves *(24.8b)* may have blackish-brown, circular lesions or, more commonly, tan lesions surrounded by a black margin that resemble scorching damage from heating pipes. If plants are infected before fruit set, fruits develop unevenly and become misshapen. Orange, yellow or red spots surrounded by a dark green margin may develop on the fruit and may occur in ring patterns *(24.8c).* If fruits are infected after setting, ripening is uneven.

Causal agent (see Greenhouse tomato, 25.22)

Disease cycle (see Greenhouse tomato, 25.22)

Management The disease can be managed by monitoring for thrips and infected plants as early as possible. Vector control is the only way to limit disease spread, short of destroying the crop, and it is the only way to ensure that the disease is confined to the infested greenhouse.

Cultural practices — An infested greenhouse should not be used for pepper, tomato or any other susceptible crop until it has been thoroughly disinfested. Growers should remove and bury infected plants, including ornamentals, in and around the greenhouse. To eliminate reservoir hosts of both virus and thrips, a 3- to 6-metre-wide band around the perimeter of the greenhouse should be kept free of weeds. Weeds within greenhouses also should be controlled. Ornamental plants, particularly perennials, such as grapevines or oleander, and hanging baskets of annuals must not be grown near pepper crops. Sticky straps should be used to monitor thrips and control measures should be started at the first signs of this pest (see western flower thrips, 24.14).

Selected references
Ie, T.S. 1970. Tomato spotted wilt virus. CMI/AAB Descriptions of Plant Viruses, No. 39. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 4 pp.

(Original by J.G. Menzies, J.L. Shipp and D.R. Gillespie)

NON-INFECTIOUS DISEASES

▶ 24.9 Blossom-end rot *Figs. 24.9; 18.21c,d*

Blossom-end rot is a very common physiological disorder in greenhouse pepper and tomato (see Greenhouse tomato, blossom-end rot, 25.23). It is associated with environmental stress, especially drought, widely fluctuating moisture conditions, and calcium deficiency. Water-soaked spots, which later turn brown or black and become dry, firm or sunken, appear at or near the blossom-end of the fruit (*24.9; 18.21c,d).* Occasionally, discoloration may extend well into, or occur totally within, the fruit. Growers should avoid unnecessary stress on the plants by regular watering, ensuring that calcium uptake is adequate during fruiting. A soil test should be done before planting to determine whether adequate calcium will be available to the crop.

Selected references

Bradfield, E.G., and C.G. Guttridge. 1984. Effects of night-time humidity and nutrient solution concentration on the calcium content of tomato fruit. *Sei. Hortic.* 22:207-217.

(Original by R.J. Howard)

▶ 24.10 Sunscald *Fig. 18.29b*

Sunscald affects pepper fruits, especially those near maturity. Soft, bleached areas, which later become slightly sunken, develop on the exposed sides of the fruit *(18.29b).* Growers should maintain adequate foliage to shade the fruit and provide supplementary shading or mist the plants with water during hot, sunny weather.

(Original by R.J. Howard)

NEMATODE PESTS

▶ 24.11 Northern root-knot nematode *Figs. 18.30; 25.26*

Meloidogyne hapla Chitwood

Symptoms Pepper, eggplant and tomato are very susceptible to damage from root-knot nematodes; damage includes stunting, chlorosis, early senescence, prolific branching of rootlets, and production of small, spherical galls on roots. Infected transplants may be a source of inoculum in greenhouses *(25.26).* For a complete description, see Carrot, 6.20; see also Greenhouse tomato, 25.26; and Management of nematode pests, 3.12.

INSECT PESTS

▶ 24.12 Green peach aphid *Figs. 24.12a-e*

Myzus persicae (Sulzer)

This aphid (see Potato, 16.41) is an important pest of greenhouse pepper. It and other aphid species are difficult and expensive to control on greenhouse pepper, and relatively low numbers can cause economically important damage from the deposition of honeydew on fruit.

Outdoor hosts provide a source of wind-borne, winged forms (alates) of the green peach aphid, which may enter greenhouses through vents or other openings. Growers who overlap transplant production with the end of the previous crop often find that this aphid is well established on the new plants by the time they are set out. Clones of the aphid often continue to reproduce year-round.

Damage In greenhouse pepper, damage is worst under high intensity lights, and in the spring and fall when temperatures are moderate. High summer temperatures suppress aphid reproduction. High levels of soluble nitrogen in leaves may predispose the plants to aphid infestation, as does drought stress. Symptoms of aphid attack include honeydew accumulation, loss of blossoms and, in extreme infestations, defoliation of the plants. All aphid stages *(24.12a)* suck phloem sap, which weakens the plant. Severe infestations of more than 300 aphids per leaf *(24.12b)* result in leaf drop, high populations in flowers cause flowers to drop, and infestations on plant tips distort the growing leaves.

Honeydew becomes a nutrient source for sooty-mold fungi *(24.12e),* which block light penetration, interrupt photosynthesis and lower fruit quality. On pepper fruit, economically important damage occurs from honeydew long before aphids directly damage the plants. Removing honeydew from the fruit involves extra handling by growers or packing houses, and fruit with sooty mold in the calyx may be rejected. If growers have to bear the cost of washing, virtually no honeydew is tolerated. If washing is part of the handling at packing houses, then honeydew is acceptable provided it can be washed off easily.

The green peach aphid is known to vector over 50 plant viruses but these have not been a problem in greenhouse pepper.

Identification Greenhouse populations of the green peach aphid (*24.12b*) sometimes appear pink or yellowish in the fall, although usually they are light green (see Potato, 16.41). Winged forms have a black or dark brown head and thorax, dark tips on the pair of abdominal projections (cornicles), and a dark central patch on the back of the abdomen. *Myzus nicotianae* Blackman, long regarded as a pink form of the green peach aphid in greenhouses, also attacks pepper.

Life history Green peach aphids mature 7 to 10 days after birth. They usually reach maximum reproductive rates five days later in the greenhouse, depending on the temperature, humidity and host plant. Average reproduction rate is three to four nymphs per day for approximately 20 days, yielding 50 to 100 offspring per female. Under springtime conditions of long days with warm but not excessively high temperatures, green peach aphid populations may increase 10- to 12- fold per week on greenhouse pepper crops. The populations usually occur as colonies of nymphs around a founding female on the underside of the oldest leaves, and on growing tips where movement of nutrients within the plant is greatest.

Management

Monitoring — Growers should start monitoring when the plants are small. Development of treatment thresholds in greenhouse pepper has been hampered by the generally patchy distribution of aphids in greenhouses. However, when aphid numbers exceed five per leaf *(24.12a)*, honeydew accumulations begin to be economically important. A random weekly sample of one upper, one middle and one lower leaf from each of 60 plants per hectare, spaced evenly throughout the greenhouse, is suggested. The sample leaves should be individually bagged and aphids, predator eggs and larvae *(24.12d)*, and unemerged parasite mummies (*24.12c*) counted later under magnification.

Biological control — Biological control of this aphid has been practiced in Canada since 1986 and is popular on greenhouse pepper crops. For best results, growers should combine low level, early releases of the parasitic wasp *Aphidius matricariae* Haliday *(3.7s; 24.12c*) with larger introductions of the predatory midge *Aphidoletes aphidimyza* (Rondani) *(24.12d)* when aphid populations start to increase quickly in March. The wasp should be released at a rate of one wasp per 20 plants as soon as the first aphid is seen, then weekly for two to three weeks. The midge should be released at a rate of one midge per small pepper plant, continued weekly for two to three weeks, when aphid populations exceed a mean of one aphid per lower leaf. On larger plants, or when aphids exceed three per leaf, releases should be increased to two midges per plant.

Chemical control — Effective control can be obtained with fumigants and sprays. Usually, treatments must be applied at three- to four-week intervals throughout the season.

Selected references

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(Original by L.A. Gilkeson)

▶ 24.13 Pepper weevil *Figs. 24.13a-e*

Anthonomus eugenii Cano

The pepper weevil occurs in the southern United States, Mexico, Central America, and the West Indies. In Canada, it has been found on pepper fruit imported from Florida as recently as 1989-90. In 1992, and again in 1993, it was detected on a greenhouse pepper crop at Langley, British Columbia; however, its status as an established introduction in Canada is still open to question (see Foreign diseases and pests, 3.10).

Pepper cultivars with a thick mesocarp seem to be preferred, although all pepper species and cultivars are susceptible. Besides pepper, host plants include eggplant and other *Solanum* spp. In its native range, the nightshades and burs *Solanum americanum* Mill., *S. pseudogracile* Heiser, *S. carolinense* L., *S. nigrum* L., and *S. rostratum* Dunal are particularly important as overwintering hosts. The last two species also occur in western Canada and could be important as alternative hosts. Potato and tomato are fed upon by adults, but eggs apparently are not laid in the flowers or fruit of these plants.

Damage Adult pepper weevils feed on leaves *(24.13a)* and blossoms, and adults and larvae both bore into young fruit pods and feed among the seeds *(24.13c)*. Fruit pods *(24.13b)* become discolored and usually abort after withering at the stem and/or calyx. Seeds in young pods fail to mature, are brown in color, and become withered. Although the adult weevil prefers to lay its eggs in young fruit pods, near-mature fruits also may be attacked. These will reach full ripeness but will contain weevil droppings (frass) and areas of decaying tissue.

Life history Eggs are laid in punctures made by the adults in flower buds or young fruit pods. After hatching, which occurs in three to five days, larvae bore into and feed on tissue within the developing fruit pod. They mature in 13 to 17 days, and pupate in the fruit pod in chambers lined with silk *(24.13d)*. Adults *(24.13e)* emerge after a further three to six days. Larvae, pupae and adults may overwinter inside the infested fruit pod. The time to complete one generation ranges from 2 weeks under hot conditions to 6 weeks under cold conditions. There may be many generations a year. Adults are attracted to yellow, and adult males emit a pheromone that is attractive to the adult females.

Identification The pepper weevil (family Curculionidae) adult is 2.5 to 3.1 mm long and pale reddish brown to black, with shiny, gray or yellowish, scale-like hairs (setae) that impart a brassy pubescence. The snout (rostrum) is longer than the head and thorax combined, or about half as long as the body. Larvae are legless, white with a pale brown head, and about 4 mm long at maturity. The adult is winged and can fly.

Management ***Monitoring*** — Yellow traps alone are useful for monitoring populations in greenhouses.

Cultural practices — Sanitation is an effective control method for this pest because larvae and pupae are mostly inside the aborted flower buds and fruit. Growers should remove all aborted buds and fallen or infected fruit from the greenhouse every day and destroy them. The male pheromone may be available commercially; otherwise, yellow traps can be baited with adult male weevils to catch females, which then can be destroyed. Another practical strategy for use in some greenhouses and in some parts of Canada is to remove all plant residue from the greenhouse at the end of the cropping cycle and allow the temperature to drop below 0°C for several days during the winter. Where temperatures do not drop below freezing, or where mechanical or cultural factors preclude allowing the interior of the greenhouse to freeze, the greenhouse should be maintained at 25°C and as dry as possible for 5 to 7 days. Yellow sticky traps should be used to monitor adult weevils to ensure they are no longer present in the affected greenhouse, and cucumber or other non-host greenhouse crop should be grown in the next cropping cycle instead of pepper. All solanaceous weeds inside and on the outside perimeter of greenhouses must be removed.

Chemical control — No pesticides are registered against the pepper weevil in Canada.

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(Original by J.A. Garland, D.R. Gillespie and R.A. Costello)

▶ 24.14 Western flower thrips *Figs. 24.14a-d; 18.42i*

Frankliniella occidentalis (Pergande)

The western flower thrips (see Greenhouse cucumber, 22.34) is a major pest of greenhouse pepper wherever it is grown. This thrips can directly damage the fruit or indirectly decrease yield by reducing the photosynthetic capacity of the plant. A predatory mite has been used successfully to control this thrips on the majority of the greenhouse pepper acreage in British Columbia.

Damage The western flower thrips may occur on pepper crops any time after transplanting. In general, symptoms are similar to those on greenhouse cucumber. However, because the adult and immature thrips feed on pollen, which is a food source that is available on pepper but not on cucumber, they are found in large numbers on pepper flowers and fruit. Feeding scars often are apparent at the calyx end of pepper fruit and on the fruit itself (*18.421*). On developing fruit *(24.14b),* adult and immature thrips feed under the calyx, causing the ends of the calyx to turn up and exposing the pepper fruit to bacterial infection. Egg-laying scars occur on leaves (*24.14d)* and on fruit, on which "ghost" spotting *(24.14c)* also may occur where thrips eggs have hatched. Feeding by the thrips upon the growing tip of plants also deforms the leaves *(24.14a).*

The western flower thrips is an important vector of tomato spotted wilt virus in greenhouse pepper.

Identification (see Greenhouse cucumber)

Life history (see Greenhouse cucumber, 22.34)

Management

Cultural practices — (see Greenhouse cucumber, 22.34) The application of effective cultural practices is very important in preventing outbreaks of the western flower thrips on greenhouse pepper.

Monitoring — Growers should start monitoring for thrips when pepper is transplanted into the greenhouse. The western flower thrips can be monitored on greenhouse pepper with the same blue or yellow sticky traps *(3.7t)* discussed under greenhouse cucumber. Blue sticky traps are preferred in pepper crops because yellow traps catch high numbers of the parasitic wasp *Aphidius matricariae* Haliday, thereby interfering with the biological control of aphids (see green peach aphid, 24.12). The first detection of thrips is an appropriate time to implement biological control.

Biological control — A predatory mite *Amblyseius* (syn. *Neoseiulus) cucumeris* Oudemans is available commercially for control of western flower thrips. On greenhouse pepper, the mite should be introduced at the first detection of the thrips. Usually, only two or three releases are necessary.

To achieve thrips control on greenhouse pepper, about 10 mites per plant are necessary (fewer than for greenhouse cucumber). The mite is able to survive and increase in the absence of thrips because it also feeds on pepper pollen. Pollen feeding by the mite does not interfere with pepper pollination. If thrips control is desired during September to March, more frequent introductions of A. *cucumeris* may be necessary because subsequent generations of mites will enter into a state of arrested development (diapause).

The minute pirate bugs *(22.34i) Orius tristicolor* (White) and *O. insidiosus* (Say) also control western flower thrips on greenhouse pepper. Both species are available commercially and will provide adequate control when introduced at the rate of one bug per plant.

Chemical control — The value of chemical control is questionable because of extensive insecticide resistance in the western flower thrips. Most populations of the western flower thrips seem to have varying degrees of resistance to all chemicals used for thrips control on greenhouse vegetables. However, effective control may be obtained by spacing insecticidal treatments at approximately four-day intervals, repeated two or three times if and when thrips become too abundant. Fogging and other fumigant methods work best for adult and immature thrips in blossoms and on growing tips. Ground applications are the only way to control the pre- pupal and pupal stages.

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(Original by D.R. Gillespie and J.L. Shipp)

▶ 24.15 Other insect pests

Caterpillars (loopers and other caterpillars)
Leafminers
- Chrysanthemum leafminer *Liriomyza trifolii* (Burgess)
- Vegetable leafminer *Liriomyza sativae* Blanchard

Melon (cotton) aphid *Aphis gossypii* Glover
Onion thrips *Thrips tabaci* Lindeman
Plant bugs *Lygus* spp.

For information on these pests, which occur sporadically on greenhouse pepper in Canada, see Greenhouse cucumber, 22.33; 22.35, and Greenhouse tomato, 25.28, 25.29.

MITE PESTS

▶ 24.16 Two-spotted spider mite *Figs. 22.36a-g*

Tetranychus urticae Koch

The two-spotted spider mite (see Greenhouse cucumber, 22.36) occurs in Canada wherever greenhouse pepper is grown.

Damage The two-spotted spider mite is a common pest of greenhouse pepper and, if uncontrolled, can cause serious damage to the crop. Symptoms are much the same as on greenhouse cucumber (*22.36a-f)* but early infestations and leaf damage on pepper are hard to detect. Unlike in cucumber, severe outbreaks do not kill pepper plants but may result in a significant decline in yield.

Management Control in greenhouses involves a combination of cultural practices and biological control because no pesticides are registered for use on greenhouse pepper in Canada.

Monitoring — The system described for greenhouse cucumber can be used, but comprehensive action thresholds have not been developed for greenhouse pepper.

Biological control — The two-spotted spider mite can be controlled effectively by the predatory mite *Phytoseiulus persimilis* Athias-Henriot (*22.36g*). (For the rate of introduction and timing of releases, of the predatory mite, see Greenhouse cucumber, two-spotted spider mite, 22.36).

(Original by J.L. Shipp and D.R. Gillespie)

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BACTERIAL DISEASES

▶ 25.1 Bacterial canker *Figs. 25.1; 18.1a-c*

Clavibacter michiganensis subsp. *michiganensis* (E.F. Smith) Davis *et al.*
(syn. *Corynebacterium michiganense* (E.F. Smith) Jensen)

Bacterial canker is a very contagious and destructive disease of greenhouse tomato. It also can affect field tomato but symptoms differ markedly (see Tomato, bacterial canker, 18.1). The disease is equally prevalent in soil- grown and hydroponic crops. Other hosts of the pathogen include pepper and black nightshade *(Solanum nigrum* L.).

Symptoms Wilting of lower leaflets is normally the first observable symptom. Older leaflets curl upward, progressively die from the margin inward and turn brown *(18.1a).* Leaflets also may have small, cream to gray-white blisters. Frequently, only leaflets on one side of the leaf are affected. Petioles may turn downward but they do not wilt. If leaflet growth is succulent, pale green spots of collapsed tissue may develop between the veins. Younger parts of infected stems and petioles may appear water soaked. Affected plants may wilt and die early *(25.1).* If growth is vigorous, plants may survive in an unthrifty wilted condition and bear some fruit. Severely affected plants display a wilt accompanied by light- colored, longitudinal streaks on the stem and petioles. These streaks may break open to form a canker *(18.1b),* thus giving the disease its name. Infected stems are slightly spongy when squeezed at the nodes. As decay progresses, the pith becomes mealy and cavities form in the soft tissues. Affected plants generally do not display extensive root discoloration.

Fruit infection is common. In young tomato plants, infected fruit is stunted, malformed and occasionally ridged. Fruit infected at later stages may not show symptoms or may have a marbled or mottled surface. The calyx scar tissue may be discolored and the calyx attachment weakened. The vascular tissue of infected fruit is yellowish from the stem scar to well within the pulp. Severely infected fruit have extensive internal breakdown with yellow to brown cavities, especially near the stem. Fruit may also develop small "bird's-eye" cankers *(18.1c)* if overhead irrigation is used. Initially, these appear as snow- white spots that scarcely extend beyond the skin. The margins of the spots are white and flat. The centers of the spots are slightly raised, tan-colored and eventually crack open. These spots do not exceed 3 mm in diameter. Seeds of early infected fruit may be spotted or entirely dark and do not mature.

Causal agent *Clavibacter michiganensis* subsp. *michiganensis* is a motile, rod-shaped, Gram-positive bacterium, measuring 1 by 0.5 µm. It is non-acid fast, non-spore forming and non-lipolytic. It liquifies gelatin slowly and can oxidize carbohydrates. It hydrolyses starch weakly or not at all and requires biotin, nicotinic acid and thiamine for growth. Yellow, white and pink forms occur but on nutrient agar the colonies are characteristically yellow. The yellow and white forms are the most virulent.

Disease cycle The bacteria are carried on the surface and within the coat of seed produced by infected plants. Germinating seedlings are infected through the cotyledons. The pathogen is able to enter the host through wounds, such as broken trichomes, or directly through the stomata. It moves systemically through the xylem and invades the phloem, pith and cortex. Spread to neighboring plants occurs by splashing or running water, insects, implements and workers tending the crop. Infection is favored by high temperatures (24 to 32°C), wet conditions, low light intensity and nutrient imbalances. The spashing caused by forceful spraying of pesticides may also help to spread the disease. The bacteria can survive on or in seed for five years and in soil for shorter periods. They also can persist from season to season on infested plant residues, wooden stakes and perennial hosts, any of which can act as an initial disease focus within a crop.

Management

Cultural practices — For effective prevention, growers should use disease-free seed. If such seed cannot be obtained, seed treatment should be considered. Seed from infected plants can be extracted and substantially freed from bacterial infection by fermenting the undiluted, crushed pulp at room temperature for 96 to 120 hours. Seed can also be soaked in acetic acid (0.6 to 0.8% solution) for 24 hours at 21°C. Other effective seed treatments include a 30-min soak in water at 56°C, a 20- to 40-min soak in 1% sodium hypochlorite, or soaking for 5 to 10 hours in 5% hydrochloric acid. Seed treatments help to reduce inoculum of the pathogen but they are not completely effective. Seed should always be sown in pasteurized growing media, using new or sterile flats, pots or other containers.

Diseased and adjacent plants should be removed as soon as they are noticed by placing them in plastic bags and carrying them out of the greenhouse. Any remaining crop residues should be gathered up or, in the case of soil, buried by rototilling. Spread can be limited by washing hands thoroughly between greenhouse visits and changing clothing when moving from an infected to a healthy crop.

Pruning and pollinating tools should be disinfested at regular intervals, plants should not be handled unnecessarily, and diseased plants should be tended after healthy ones.

Chemical control — Although chemical sprays are sometimes recommended in extension publications, most serve only to spread the disease and have little or no effect on the pathogen.

Selected references

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.2 Bacterial speck *Figs. 18.3a,b*

Pseudomonas syringae pv. *tomato* (Okabe) Young *et al.*

This disease is of minor importance in commercial tomato greenhouses but can be a serious problem on field tomato.

It sometimes occurs in greenhouses in which transplants are being grown for the field. (For more information, see Tomato, bacterial speck, 18.3.)

(Original by R.J. Howard)

▶ 25.3 Bacterial stem rot *Fig. 25.3*

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*

An unusually high incidence of stem rot, wilt and death of tomato plants occurred in the greenhouse tomato production area of Essex County, Ontario, during the harvest season of 1983. This disease had occurred only sporadically before that time. *Erwinia carotovora* subsp. *carotovora* has a wide host range that includes many vegetables (see Potato, bacterial soft rot, 16.2).

Symptoms Symptoms first appear about the time of first or second harvest *(25.3).* Basal leaf scars may have dark brown lesions, while stem bases become hollow and appear water-soaked. Bark readily sloughs off and stem pith turns brown and disintegrates. At an advanced stage of the disease, plants wilt and die. Bacterial stem rot is favored by high humidity and can be spread by splashing water, workers' hands and tools. See also Tomato, bacterial soft rot, 18.2.

Causal agent (see Potato, 16.2) Several other *Erwinia* and *Pseudomonas* species have been shown to cause bacterial soft rot of greenhouse tomato in Europe.

Disease cycle (see Potato, bacterial soft rot, 16.2)

Management

Cultural practices — The sanitation practices outlined for bacterial canker are relevant here. Growers should avoid working with plants when the foliage is wet and direct water dripping from gutters away from tomato plants.

Selected references

Bradbury, J.F. 1977. *Erwinia carotovora* var. *carotovora*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 552. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

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(Original by R.J. Howard)

▶ 25.4 Pith necrosis *Figs. 25.4a-c*

Pseudomonas corrugata Roberts & Scarlett

This disease affects greenhouse tomato, particularly those plants with luxuriant growth. Its occurrence is sporadic and usually limited to a few plants. Tomato is the only crop in which this disease is an economic problem. *Pseudomonas corrugata* has been isolated from symptomless alfalfa roots, and in laboratory tests, some strains can rot carrot tissue. Otherwise, no other hosts are known.

Symptoms Plants affected by pith necrosis are usually vigorous, with thick fleshy stems and a large canopy. Symptoms normally appear just before the first fruit is picked. Initially, affected plants display chlorosis of the upper leaves, sometimes with wilting *(25.4a).* They may be stunted and have elongate, dark brown to black lesions on the stem *(25.4b).* The stem may collapse at lesion sites. The pith of the main stem blackens and there may be large spaces with ladder-like cross strands in the blackened pith in older parts of the stem. Light brown discoloration occurs in younger areas of stems that lack a pith cavity *(25.4c).* In general, pith discoloration extends to the soil level but it does not enter the roots. The peduncle also may have discolored pith but not the fruit. Occasionally, leaf scars will have a creamy-white bacterial ooze. Older plants frequently exhibit prolific development of adventitious roots on the stem, usually coinciding with the infected area. Diseased plants may continue to produce fruit.

Causal agent *Pseudomonas corrugata* is a non-fluorescent rod with a flagellar tuft at the pole and is characterized as aerobic, oxidase positive, gelatin and egg-yolk positive, starch hydrolysis and levan test negative, and HR positive on tobacco. It accumulates poly-ß-hydroxybutyrate (PHB). Erythritol and rhamnose are not utilized. Growth occurs at 37°C but not at 41°C. Colonies on nutrient agar are round, achieving a diameter of 1 mm in two days and 1 to 3 mm in a week. They are raised, cream to buff, later turning beige-yellow. On nutrient agar with 5% glucose, colonies are yellowish, often with green centers at two days and usually with a yellow-green diffusible non-fluorescent pigment, which also develops on King's B medium. The bacterium is readily isolated on King's B medium by streaking exudates from affected tissues. A semi-selective isolation medium (TNR) is available (see Selected references, Scortichini 1989).

Disease cycle The biology of this bacterium is not well understood but it is considered to be soil- and water-borne. The pathogen can infect seedling tomato through the roots. There is no evidence of workers spreading the disease on hands, clothing or tools, and it does not spread easily within the crop. The disease is favored by high humidity and high nitrogen status. Adventitious root formation in affected plants is caused by the accumulation of auxins.

Management

Cultural practices — Growers should try to avoid conditions that lead to wet plants and luxuriant growth. Excessive foliage can be controlled by raising the level of potash in the fertilizer solution to obtain a corresponding decrease in the nitrogen: potassium ratio. Diseased plants should be removed immediately, using the procedures outlined for bacterial canker.

Selected references

Clark, R.G., and D.R.W. Watson. 1986. New plant disease record in New Zealand: tomato pith necrosis caused by *Pseudomonas corrugata. N.Z. J. Agric. Res.* 29:105-109.

Lai, M., D.C. Opgenorth and J.B. White. 1983. Occurrence of *Pseudomonas corrugata* on tomato in California. *Plant Dis.* 67:110- 112.

Scarlett, C.M., J.T. Fletcher, P. Roberts and R.A. Lelliott. 1978. Tomato pith necrosis caused by *Pseudomonas corrugata* n.sp. *Ann. Appl. Biol.* 88:105-114.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.5 Stem necrosis *Fig. 25.5*

Pseudomonas sp.

This disease has been reported only from Ontario, where it has caused concern among growers for several years. It usually appears in the spring crop. The pathogen is opportunistic, infecting plants that have been stressed by unbalanced nutrition, excessive humidity or the onset of fruiting. It has been seen in both soil- and rockwool-grown crops.

Symptoms Characteristic symptoms of tomato stem necrosis include dark brown discoloration of leaf bases at the nodes, adjacent leaf rachises and internodes, followed by cortical and pith necrosis and breakdown (25.5). Except for a general pith necrosis, these symptoms differ from those reported for most of the other stem rot diseases of greenhouse tomato. Vascular discoloration is sometimes observed but plants generally do not wilt or collapse. Fruits are free of symptoms.

Causal agent The taxonomy of this *Pseudomonas* sp. is uncertain. It resembles *P. cichorii* except that the stem necrosis pathogen is not pathogenic to chrysanthemum or lettuce, and it has been provisionally assigned to the group of oxidase-positive, arginine dihydrolase-negative, phytopathogenic fluorescent pseudomonads now solely represented by *P. cichorii* (see Lettuce, pseudomonas diseases, 11.3).

Disease cycle The disease resembles the other bacterial diseases in that it is spread by irrigation water and principally by workers carrying out routine operations in the crop.

Management

Cultural practices — Growers should follow the sanitation practices outlined for bacterial canker (see 25.1).

Selected references

Dhanvantari, B.N. 1990. Stem necrosis of greenhouse tomato caused by a novel *Pseudomonas* sp. *Plant Dis.* 74:124-127.

(Original by R.J. Howard)

FUNGAL DISEASES

▶ 25.6 Corky root (brown root rot) *Figs. 25.6a,b*

Pyrenochaeta lycopersici R. Schneider & Gerlach

In greenhouses, corky root is fairly common and serious in early spring crops grown in media that are too cold. While it is most common in soil-grown crops, significant numbers of crops grown in rockwool are also affected. It is unknown how the fungus infests artificial substrates; it has even been reported in the nutrient film (NFT) system. Corky root only rarely affects field tomato crops. The pathogen survives on the surface of roots of lettuce and some weed species.

Symptoms The first symptoms appear as light brown lesions about 5 mm long on the surface of fine roots. This stage is often referred to as brown root rot *(25.6a)*. Early symptoms on the upper portions of plants include lack of vigor, foliar chlorosis and stunting. Larger roots develop dry, brown, swollen, corky lesions with splits in the outer sheath *(25.6b)*. The cortex of the root can easily be pulled off the central stele at lesion sites. Dark-brown cortical lesions are often present at the base of the stem on severely diseased plants. Massive root failure is the cause of wilt in hot, sunny weather, and is followed eventually by death of the plant. Yields may be reduced.

Causal agent *Pyrenochaeta lycopersici* was long known as a gray sterile fungus, because of the difficulty in obtaining sporulation in culture, although some strains sporulate on roots. In culture, globular to sub-globular, brown to black pycnidia, 150 to 300 pm in diameter, are produced. Pycnidia have 3 to 12 light brown, septate setae, measuring 7 by 120 µm. Conidiophores within the pycnidia are septate and simple. Unicellular, hyaline conidia are produced from the apex and short lateral branches immediately below the septa of the conidiophores within each pycnidium. Conidia are cylindrical to allantoid and measure 4.5 to 8 by 1.5 to 2 pm. Microsclerotia are unspecialized with cell walls of uniform thickness.

Dark brown lesions on roots become swollen and corky in texture. The cortex pulls off the stele readily to leave characteristic "rat-tails." In contrast with black-dot root rot, in which the pathogen produces fruiting bodies late in the season, fruiting bodies on corky root lesions are rare. Microscopic examination of the lesions will reveal mycelia packing diseased host cells to form characteristic rectangular microsclerotia.

Disease cycle The pathogen is soil-borne and can survive as sclerotia for at least two years in soil. It grows at 8 to 32°C and develops very slowly in the soil. Thus, the disease increases slowly over time. The fungus colonizes disinfested soil slowly when introduced into a greenhouse. Infection occurs when host roots make contact with the fungus mycelium, often in soil that is below the level of effective steam pasteurization.

Management

Cultural practices — Crops grown in peat modules or in soilless, NFT or rockwool culture systems are generally not affected. Because the pathogen grows slowly, some recovery may be achieved by mounding soil or soilless media, such as sawdust or peat, around the base of the stem to encourage the growth of adventitious roots. Good ventilation is essential, especially around the stem bases, and splashing should be minimized during watering. Transplanting should be done into warm soil (over 15°C), because corky root is essentially a disease of cool soils (10 to 15°C). For this reason, mulches such as straw, which insulate the soil, should not be put down until the soil is warm.

Resistant cultivars — Resistant rootstocks, such as KNV and KNVF types, are available, so grafting of commercially acceptable scions onto resistant rootstocks is possible.

Chemical control — The pathogen can be eradicated from soil by steam or fumigation, but not at depths below the level reached by these disinfestants. Growers should remove and destroy old roots before disinfesting growing media.

Selected references

Grove, G.G., and R.N. Campbell. 1987. Host range and survival in soil of *Pyrenochaeta lycopersici. Plant Dis.* 71:806-809.
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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.7 Damping-off *Fig. 25.7*

Phytophthora spp.
Pythium spp.
Rhizoctonia solani Kühn
(teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk)

Damping-off can be severe in newly transplanted crops, especially those grown in soil. The reduction of seedling emergence and the falling over of young seedlings are characteristic symptoms. In greenhouses, the most important causal agents are *Pythium* spp., but other fungi also may be involved, for example, *Phytophthora* spp. and *Rhizoctonia solani.* These fungi are soil-borne. *Pythium* and *Phytophthora* spp. may also contaminate irrigation water. Damping-off occurs frequently in seedlings and transplants raised in rockwool blocks and other soilless substrates. This disease can affect a variety of vegetable crops.

Symptoms Symptoms vary with age and stage of development of the host. If seeds are infected before germination, they fail to germinate, become soft and mushy, turn brown, shrink and finally decompose. Infection of young seedlings results in slightly darkened, water-soaked lesions that expand until the invaded tissues collapse. Infection of seeds or young seedlings is usually apparent by a reduction in seedling emergence.

After emergence, seedling roots can become infected at or below soil level. Lesions are usually pale brown and water-soaked *(25.7).* The basal part of infected seedlings is normally much thinner and softer than the upper part. The invaded stem cannot support the seedling, which falls over, withers and dies.

Causal agents (see Bean, 15B.4; Beet, pythium and rhizoctonia root rots, 5.7, 5.8; and Carrot, cavity spot, 6.8, and pythium root dieback, 6.13)

Disease cycle Damping-off pathogens spread quickly in cool, wet soil. Temperatures of 10 to 15°C are the most conducive to early damping-off by *Pythium* and *Phytophthora* spp. Infection is aided by excess nitrogen and crowding of the plants.

Rhizoctonia solani (see Bean, rhizoctonia root rot, 15B.7) tends to attack more mature transplants, leading to late damping-off. Under drier conditions, *R. solani* is frequently the most troublesome damping-off pathogen.

Management

Cultural practices — To retard spread of these pathogens, greenhouse seedlings should not be crowded or overwatered. Watering should be done only when the soil is dry and preferably in the morning so the soil will be dry by late afternoon. Adequate greenhouse ventilation helps to keep the soil dry. Seed flats should be raised and placed out of the range of splashing water. Bottom heat should be applied to raise the soil temperature above 15°C. Rockwool blocks and other soilless media should never be allowed to come into contact with soil or water splashing from soil and dirty benches or floors.

Chemical control — Seed treatment with hot water, followed by application of a fungicidal seed protectant, and sowing into pasteurized soil, is helpful. Seedling trays can be drenched with fungicide solutions to provide additional protection if conditions warrant.

Selected references

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.8 Didymella stem canker *Fig. 25.8*

Didymella lycopersici Kleb.
(anamorph *Diplodina lycopersici* Hollos)

This is an important fungal disease of tomato in Europe, but it is rare in North America. It has been seen occasionally in British Columbia and Nova Scotia. The disease occurs in soil and soilless culture and may spread explosively. Fruit infection occurs in the field in the United States, but only rarely in the greenhouse. Alternative hosts include black nightshade *(Solanum nigrum* L.), eggplant, pepper and potato.

Symptoms Dark brown, sunken lesions appear on the stem at or near soil level *(25.8).* The epidermis and cortex decay and the xylem becomes brown some distance up the stem. The stem above the lesions remains green. Severely affected plants with one or more stem lesions frequently wilt and the lower leaves show varying degrees of chlorosis and necrosis. Under damp conditions, small brown lesions with concentric rings develop on the leaves. The centers eventually become pale brown or tan with a few pycnidia. The lesions may fall out, leaving shot-holes, or they may grow together and kill the whole leaf. The pathogen can attack any part of the fruit; however, it usually infects the calyx end, causing an extensive black rot. Pycnidia form on the infected part and the fruit may drop off the plant. If fruit remain attached, the fungus may grow into the pedicel and ultimately reach the stem. Seed may also become infected.

Causal agent *Didymella lycopersici* is most commonly observed in its anamorphic state. Pycnidia are sub-epidermal, ostiolate, 100 to 270 pm in diameter and scattered or aggregated on raised spots. Conidia are one- to two-celled, sub-cylindrical and measure 4.5 to 17 by 2.5 to 5 µm. Pseudothecia are sub-globose and dark-brown with cylindrical asci 70 to 95 by 9 to 10 µm. Each ascus has eight, spindle-shaped, hyaline, unisep- tate ascospores, each measuring 16 to 18 by 5.5 to 6.5 µm.

Careful examination of stems will reveal the small round pycnidia, which can be confused with the dark brown glandular trichomes present on tomato stems. Accurate diagnosis is difficult without a hand lens because the lesions resemble those of gray mold, which are normally lighter brown, develop aerial conidiophores and cause the stem area above the lesion to turn yellow. Both fungi can occur in the same lesion. On leaves, the small brown lesions with concentric rings resemble those of early blight.

Disease cycle The pathogen survives from season to season in the soil or on alternative hosts. It may survive on infected seed or as spores on contaminated seed boxes, stakes or greenhouse structures. The main mode of introduction to a new crop is by water-splashed conidia produced in pycnidia on plant residue or alternative hosts. The fungus infects host tissues at 11 to 30°C (optimum 20°C), spreading rapidly once established. Numerous, small, black pycnidia appear on the rotting lesions. Rarely, pseudothecia are intermixed with the pycnidia. Later in the season, lesions form higher in the plant canopy and spread to all above-ground parts of the plant. At high humidity, cirrhi of gray to pink conidia extrude in a gelatinous matrix. These spores are spread by splashing water, trimming knives or workers' hands, and occasionally they are air-borne. The conidia are tolerant of desiccation and low temperature, can be transported long distances in the air, and can survive extended periods of unfavorable environmental conditions.

Management

Cultural practices — Resistance to infection increases with plant age and an adequate supply of nitrogen and phosphorus. Strict crop hygiene and disposal of diseased residues, especially at the end of the season, are very important. Stem lesions should not be removed by knife because the blade will become contaminated with spores and infect subsequent plants being trimmed. Fruit should be picked without bruising, allowed to dry and layered singly with the stem-end up to avoid the fruit rot phase of this disease. Disposal of infected plants should be in plastic bags without handling the lesions and all plant residue should be buried or composted as far from the greenhouse as possible.

Chemical control — Soil should be disinfested between crops and the greenhouse should be washed with a disinfestant or fumigated before a new crop is planted.

Selected references

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.9 Early blight (target spot), alternaria fruit rot *Figs. 25.9a,b; 18.8b,c,f*

Alternaria solani Sorauer
Alternaria alternata (Fr.:Fr.) Keissl.

Early blight is a very common disease of field tomato and occasionally greenhouse tomato. In greenhouses it can occur in crops grown in soil and soilless media. *Alternaria solani* also infects potato (see Potato, early blight, 16.8), eggplant and solanaceous weeds. *Alternaria alternata* occurs widely in nature on organic matter and is generally considered to be a weak, opportunistic plant parasite (see Cucurbits, alternaria leaf blight, 9.8; and Tomato, early blight, 18.8).

Symptoms This disease is most common on older foliage, but it also occurs on the stems and ripening fruits of greenhouse tomato. Leaf spots are circular, about 1 cm in diameter, dark brown to black, and readily recognized by concentric rings or zonations ("target spots") *(25.9a).* The concentric aspect of the rings may be lost on lesions near the edge of the leaf. Lesions on stalks, branches and pedicels appear black, subsequently enlarging, elongating and sometimes girdling them. Fruit lesions generally start around a pedicel, wound or crack, rapidly enlarging into black, leathery sunken areas *(18.8c).* If defoliation is severe, unprotected fruit may be damaged by sunscald.

This disease may be confused with septoria leaf spot *(25.9b):* however, septoria leaf spot has pycnidia in relatively small spots, whereas early blight has concentric dark rings in larger spots *(18.8b). Alternaria alternata,* the less pathogenic species, is frequently associated with *A. solani* on lesions *(18.8f).* Leaf lesions with concentric rings are also associated with didymella stem canker (see didymella stem canker, 25.8).

Causal agent (see Potato, early blight, 16.8) The tapered, muriform conidia of *A. solani* are 150 to 300 pm long and are characterized by a very long beak, about the same length as the body; conidia of *A. alternata* have a very short beak, and are 20 to 63 pm (mean 37 pm) long. Conidia should be mounted in water for spore identification.

Disease cycle (see Potato, early blight, 16.8) The pathogen can survive for long periods in soil and diseased plant residue. It is also seed-borne. Infection of the crop is initiated by conidia produced on diseased hosts or host residues. Disease development is favored by alternating conditions of high humidity at night and dry days. Infection can occur between 10 and 25°C.

Management

Cultural practices — Diseased leaves and stems should be removed and destroyed if practical. Soilbeds should be steamed or fumigated between crops.

Chemical control — Registered fungicides are available.

Selected references

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.10 Fusarium crown and root rot *Figs. 25.10a-d*

Fusarium oxysporum f. sp. *radicis-lycopersici* W.R. Jarvis & Shoemaker

Fusarium crown and root rot is an important disease of greenhouse tomato in the United States and Canada, and it also has been reported on field tomato. It occurs with equal severity in commercial crops in soil, rockwool, sawdust and the nutrient film techniques (NFT). For practical purposes, the disease is limited to tomato, but many plants have been shown to be susceptible when artificially inoculated with the pathogen.

Symptoms Young tomato seedlings can become severely affected and die, but this disease mainly affects bearing plants. Symptoms usually appear in greenhouse tomato crops just before the first pick. Infected plants can often be distinguished by a marked thinness at the top of the stem. These plants wilt, beginning with the upper leaves, and there may be a chocolate-brown cortical rot at soil level *(25.10a),* with a red-brown vascular discoloration extending upwards in the stem for 5 to 25 cm *(25.10b).* Subsequently, the lower leaves may turn golden-yellow from the tip and eventually die. Wilt symptoms abate on cooler and overcast days and after picking and watering. Adventitious roots may form above stem lesions. Roots of infected plants have dark red-brown lesions, often confluent with hypocotyl lesions. Small, gray- brown lesions form where secondary roots emerge from the main roots. Fruits from affected plants are flaccid and lack the normal bright color *(25.10c,d).* Dead or near-dead plants may have conspicuous external masses of pink-white or salmon-colored mycelium.

Causal agent *Fusarium oxysporum* f. sp. *radicis-lycopersici* is indistinguishable from other forms of *F. oxysporum* in morphology and in characteristics in pure culture. Microconidia are oval-ellipsoid, cylindrical, straight to curved, and measure 5 to 12 by 2.2 to 3.5 pm. They are produced on simple phialides arising laterally on hyphae or from short, sparsely branched conidiophores. Macroconidia are thin-walled, generally three- to five-septate, fusoid-subulate, pointed at both ends with a hooked apex and a pedicellate base, and measure 27 to 66 by 3 to 5 pm. Chlamydospores are generally abundant, solitary and both terminal and intercalary.

Other tomato wilt diseases have similar symptoms, so diagnosis requires the isolation and identification of the pathogen. Special culture media are available for the selective isolation of *F. oxysporum.* A petri- dish test has been developed to distinguish between the fusarium wilt and fusarium crown and root rot pathogens. In this test, tomato seeds are germinated directly on water agar seeded with the suspected pathogen. Fusarium crown and root rot causes soft, chocolate-brown lesions on the hypocotyl, whereas fusarium wilt causes no symptoms or only a faint brown discoloration.

Disease cycle The manner in which the pathogen is introduced into previously unaffected areas is not known. It can survive as chlamydospores in soil below the level of effective sterilization by steam or fumigation, as well as in thick roots and lumps of clay that are difficult to sterilize. In heavily infested seedling and transplant trays, damping-off sometimes occurs; symptoms resemble those of pythium damping-off. There may be a fast wilt of mature infected plants, resulting in early death, or a slow wilt with progressive, acropetal leaf death. Plants suffering from slow wilt may survive until the end of the season and produce a flush of new growth after most of the fruit has been picked.

Field crops do not seem to play a significant role in epidemiology in the greenhouse. Seed transmission or movement of chlamydospores on clothing, shoes, machinery, packing crates and in soil or compost are all possible pathways for spread. Infection of young seedlings occurs in infested soil or from air-borne microconidia from residue piles of tomato vines, soil and straw mulch. The fungal population increases rapidly after introduction into fumigated or steam-sterilized soils, but less so in pasteurized soils. It enters root and hypocotyl cortical tissues through wounds caused by emerging secondary roots, as well as by directly penetrating the epidermis. Microconidia are probably spread by water from mobile irrigation systems or by wind currents. It is also suggested that fungus gnats may spread the pathogen from diseased to healthy plants while feeding. Fungus gnats may further aid the pathogen by creating wounds through which it can invade the roots. The optimum temperature for disease expression is 15 to 18°C.

The significance of other crops in fostering the survival and spread of the pathogen has not been assessed.

Management (For fungus gnats and their control, see Greenhouse cucumber, 22.31.)

Cultural practices — The incorporation of lettuce or dandelion residues into the soil before planting tomato reduces disease severity, as does companion planting with lettuce or dandelion. Soil in beds should be 20°C or warmer at the time of transplanting. Straw mulch should not be laid in spring until soil temperatures reach this level. Late spring plantings are less affected than winter and early spring plantings. In greenhouse crops, removal of the first fruit on heavily infected plants may allow the plant to recover with relatively little loss. Mounding soil or a soil- peat mixture around the base of the stem of infected plants to a height of 10 to 20 cm stimulates the formation of adventitious roots, which generally remain disease-free, allowing the plant to recover.

Resistant cultivars — Resistant cultivars include CR-6, 83W186 and B8-864 (pink-fruited); and Larma, Vicores, Furon, Trend, Farao, XPH2419/88, Cobra and W1601 (red- fruited). Grafting scions of susceptible tomato cultivars with good agronomic characteristics onto resistant tomato rootstocks, such as KNVF-Tm or KVF, has been effective.

Biological control — Cross protection of susceptible cultivars by root inoculation with avirulent *Fusarium oxysporum* f. sp. *radicis-lycopersici* shows promise for control.

Chemical control — Steam sterilization or fumigation of soil beds does not control the disease and often worsens the problem because of the rapid re-entry of the pathogen into the soil. Pasteurization of soil by steam-air mixtures is much more

effective, because it preserves a number of competitive and antagonistic microorganisms that can substantially reduce the infective population of *Fusarium oxysporum* f. sp. *radicis-lycopersici* in the soil.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.11 Fusarium wilt *Figs. 25.11a-c*

Fusarium oxysporum f. sp. *lycopersici* (Sacc.) W.C. Snyder & H.N. Hans.

Fusarium wilt of tomato is a common disease and is most destructive in warm greenhouses. The pathogen can survive on other *Lycopersicon* species, on species of *Amaranthus, Digitaria* and *Malva*, and as a saprophyte in association with fibrous roots of other plants.

Symptoms The first symptoms on young plants are the clearing of veins and chlorosis of lower leaves; this is followed by epinasty of the older leaves caused by drooping of the petioles (*25.11a).* Plants infected as seedlings often wilt and soon die. Severely infected older plants may wilt and die suddenly if the weather is favorable for pathogen development. Generally, in older plants, the first symptoms increase in intensity until the entire plant shows symptoms. The plants remain stunted, with occasional formation of adventitious roots, wilting of leaves and stems, defoliation, marginal necrosis of remaining leaves, and they eventually die (*25.11b).* A one-sided discoloration of the stem may occur during late stages of disease development and new, apparently healthy growth is produced from the base, while the top of the stem shows severe symptoms. The woody tissues of affected plants have a brown discoloration (*25.11c).* Fruit may occasionally become infected, rot and drop. Roots can be infected and stunted, with the smaller side roots rotting completely.

Causal agent *Fusarium oxysporum* f. sp. *lycopersici* is indistinguishable from other forms of *F. oxysporum* in pure culture. Abundant microconidia are produced on simple phialides arising laterally on the hyphae or from short, sparsely branched conidiophores. Microconidia are oval-ellipsoid, cylindrical, straight to curved, and measure 5 to 12 by 2.2 to 3.5 pm. Macroconidia are thin-walled, generally three- to five-septate, fusoid-subulate, pointed at both ends, have a hooked apex and a pedicellate base, and measure 27 to 66 by 3 to 5 pm. Chlamydospores are generally abundant, solitary and terminal or intercalary.

Several wilt diseases of tomato have similar symptoms; therefore, isolation and identification are necessary. The pathogen can be isolated from vascular tissue at the top of the plant. There are media for selective culturing of this fungus. A petri-dish test has been developed to distinguish between the fusarium wilt and fusarium crown and root rot pathogens (see fusarium crown and root rot, 25.10).

Disease cycle Long-distance dissemination may occur on seed, in symptomless transplants, and in soil associated with transplants. Once established, the fungus survives as chlamydospores in soil and in root residues. The disease is favored by low soil moisture, short daylength, low light intensity, low pH, plant tissues low in potassium, and soil temperatures around 28°C. Increasing levels of nitrate nitrogen reduce plant susceptibility to wilt. Wounding of the root system through improper handling of transplants favors the disease.

Management

Cultural practices — The use of disease- free seed and transplants helps to prevent the spread of fusarium wilt to uninfested greenhouses. If clean seed is not available, those of questionable status should be given a hot-water treatment (see bacterial canker, 25.1). The disease develops best at high temperatures (28°C), so excessive warming of propagating beds should be avoided. Crop rotation is of limited use because the pathogen survives for long periods in soil. Treatments that adjust the soil close to pH 7 help to control this disease, but a soil pH of 7.5 favors verticillium wilt. The use of peat modules or soilless, rockwool or NFT culture systems aids in control. Growers should supply adequate nitrate nitrogen to plants, but excessive fertilization favors the disease. Greenhouse structures, crates, benches and tools should be cleaned regularly. Precautions should be taken to reduce the spread of the pathogen in infested soil, on implements and by workers during movement between greenhouses, plant beds and production fields. Cultivation may cause root damage and increase the risk of infection.

Resistant cultivars — Fusarium wilt-resistant cultivars and rootstocks, such as KNVF types, provide the best means of control. When grafting susceptible scions to resistant rootstocks, the scion root-system must be severed before transplanting the grafted plants. Resistance may not be expressed when resistant cultivars are grown in soil infested with both *Fusarium* and root-knot nematodes because of physiological changes in the root induced by the nematodes.

Chemical control — Disinfestation of growing media with chemical fumigants or steam is effective and practical in most greenhouses.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.12 Gray mold (ghost spot) *Figs. 25.12a-d; 18.11 a-c*

Botrytis cinerea Pers.:Fr.
(teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)
(syn. *Sclerotinia fuckeliana* (de Bary) Fuckel)

Gray mold is a common disease of greenhouse tomato that can be minimized if the crop is managed properly. Tomato fruit is susceptible to infection during early stages of development. *Botrytis* survives as mycelium on decaying plant residue or as sclerotia that may persist in dry soil for several months or years. The pathogen has a wide host range that includes many vegetable crops (see Asparagus, botrytis blight, 4.1 ; and Lettuce, 11.10).

Symptoms Tomato leaflets, petioles, whole leaves, stems and fruit can be infected. Older tissues are generally more susceptible to attack than younger ones. Leaf lesions develop as light brown or gray, circular spots and may grow to cover the whole leaflet (*18.11a).* Affected leaves become covered with conidiophores and conidia, and subsequently collapse and wither. The fungus will grow from diseased leaves into the stem and produce dry, light brown lesions a few millimetres to several centimetres in length. Lesions also form at deleafing scars on the stem (*25.12a),* especially on older parts of the stem lying on the greenhouse floor. The stem lesions may also be covered with a gray mold. Severe infection can girdle the stem and kill the plant (*25.12b,c).*

On green tomato fruit, the most common symptom is ghost spot, which is a tiny brown, often raised, necrotic spot surrounded by a pale halo (*25.12d).* Once the fruit reaches 2.5 cm in diameter, the surface becomes smooth and shiny and resists infection; however, fruit can also become infected through flower parts stuck to the surface, especially at the calyx end, resulting in an irregular, brown lesion in the area of the flower parts.

Ghost spotting can also occur on ripe fruit (*18.11b)* and occasionally results in downgrading of shipments. Mature fruit may also be affected by a rot that starts at the calyx end (*18.11c).* Fruit becomes water-soaked and soft at the point of infection. The spots are irregular, up to 3 cm in diameter and light brown to gray. Rotting fruit eventually drops.

Causal agent *Botrytis cinerea* (see Lettuce, gray mold, 11.10) is readily identified by the presence of gray conidiophores, conidia or sclerotia on dead, pale gray to tan tissue. These signs are easily seen with the naked eye or a hand lens. Ghost spot on the fruit is also distinctive. On tomato stems, the mycelium becomes darker, the lesions blacken, and sclerotia may appear in the lesions as the fungus ages. Older lesions bearing sclerotia may be mistaken for those of didymella stem canker (see didymella stem canker, 25.8).

Disease cycle (see Lettuce, gray mold, 11.10)

Management

Cultural practices — Growers should maintain adequate heat and ventilation in the greenhouse, especially during the night. A relative humidity of less than 80% will deter gray mold development. Removal of lower leaves aids in disease prevention by allowing free flow of air through the crop. Leaves should be pruned to clean-cut stubs, 1 to 2 mm long, and kept dry by using drip irrigation or surface watering. Crop residue should be removed promptly because it acts as a source of spores.

Chemical control — Fungicide sprays help to control the disease when properly timed. Strains resistant to benomyl, dicloran, iprodione and captan are known, which is why fungicide rotations and combinations are recommended. Because infection of leaf scars may occur up to 10 to 12 weeks before symptoms appear, protective treatments should be considered at the time of deleafing if humid conditions prevail. Stem cankers can be treated by scraping diseased tissue off the stem and applying a thin paste of fungicide over and slightly beyond the affected area.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.13 Late blight *Figs. 25.13a,b; 18.12a-d*

Phytophthora infestans (Mont.) de Bary

This disease is far more prevalent in areas with maritime versus mid-continental climates. It is common on potato and outdoor tomato. Under cool, humid conditions, greenhouse tomato can also be infected, whether grown in soil or in hydroponic production. Late blight also can attack eggplant, pepper and some solanaceous weeds.

Symptoms Initially, irregular, water-soaked, green-black spots appear at the tips or edges of the oldest leaves. Under humid conditions, the spots enlarge rapidly to form brown areas with indefinite borders (*25.13a; 18.12a,b).* Spore formation usually occurs at the margin of these lesions. A blue-gray growth of the pathogen may develop on lower leaf surfaces. The fungus may grow through the entire leaflet, affecting all the leaflets on a leaf, which then wilts and dies. Brownish cankers often form on the stems and petioles *(18.12c).*

Infection of the fruit can occur at any stage of development. Green-brown, water-soaked lesions may spread over the entire surface *(25.13b).* Under humid conditions, a blue-gray growth also may develop on affected fruits *(18.12d).*

Causal agent *Phytophthora infestans* (see Potato, late blight, 16.11) is normally identified by microscopic examination of diseased plant tissue. It produces long sporangiophores with thin-walled, oval, colorless sporangia. Water-soaked, green-black leaf lesions are characteristic. Badly affected crops have a fishy odor.

Disease cycle (see Potato, late blight, 16.11) Inoculum to initiate the disease on greenhouse tomato normally originates from nearby infected field potato or tomato crops. Overwintered trash piles are also potential sources of infective spores. Epidemic development is favored at 18 to 21 °C and high humidity.

Management

Cultural practices — Infected leaves should be carefully removed and buried. Any practices that reduce humidity within the crop also help to control this disease.

Chemical control — Fungicides aid in controlling late blight if applied as preventive sprays. If potato crops are grown close to greenhouse tomato crops, a routine spray application should be considered, especially in years when late blight is prevalent on potato. Tomato should be sprayed at the times indicated for potato. Because not all fungicides registered for potato can be used on tomato, growers should consult provincial recommendations.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.14 Leaf mold *Fig. 25.14*

Fulvia fulva (Cooke) Cif.
(syn. *Cladosporium fulvum* Cooke)

Leaf mold is most important on greenhouse tomato crops, especially in poorly ventilated plastic houses. It affects crops equally in soil or hydroponic production. It also attacks field tomato in cool, humid seasons. Tomato is the only plant affected by this disease.

Symptoms Symptoms usually occur only on the foliage, but they may involve blossoms and fruit. The first symptoms are indefinite, yellow-green areas on the upper surface of leaves, and in some cultivars and environments, pale, nearly white spots on the lower surface. Later, these areas coincide almost exactly with a brown to purplish, velvety fungal growth on the lower surface *(25.14).* Symptoms and signs appear first on older leaves, progressing onto younger ones. Infected leaves eventually become yellow-brown, curl, wither and drop prematurely. Infected blossoms usually die before fruit set. Green and ripe tomato fruits can develop a black, leathery, irregular, stem-end rot that may cover one- third of the fruit surface. Infected fruit may be lopsided with blackened radial furrows, remaining unripe on the affected side. The fungus can be isolated readily from conidia on leaf lesions.

Causal agent No sexual stage is known for *Fulvia fulva.* Conidiophores are unbranched, narrow at the base and broader apically, pale brown to dark at the apex, septate, and measure 57 to 125 by 1.3 to 7 µm. Conidia are brownish, cylindrical to ellipsoid, smooth, straight or slightly curved, and produced in chains. They are one- to two-celled, measure 12 to 47 by 4 to 10 µm, and have a conspicuous thickened hilum. On the plant, a pale sub-stomatal stroma is present.

The pathogen can be identified by its characteristic fruiting structures on diseased leaf tissue. A velvety, purple brown fungal growth beneath indefinite yellowish areas on the leaves is diagnostic. The fungus is readily isolated directly from conidia and it grows on most laboratory media. In culture, the colonies are effuse, velvety, buff to brown or purplish, with a whitish margin.

Disease cycle Disease development is favored by a relative humidity of 85% or more or by liquid water on the leaves. Germination can occur between 5 and 35°C; the optimum temperature is 22°C. The pathogen produces large numbers of conidia on infected tissue. Once the primary infection has occurred, the disease spreads rapidly through the greenhouse. The conidia are readily dispersed by air currents, water, workers moving through the crop, and by insects.

The pathogen survives from crop to crop as sclerotia, conidia or mycelium in soil or crop residues. Conidia are known to survive at least one year under adverse conditions, and new conidia are readily produced in the leaf on substomatal stromata. Contaminated seed may facilitate the widespread dispersal of new races, but spread of the pathogen between greenhouses commonly occurs on workers' clothing.

Management

Cultural practices — Adequate row and plant spacings are necessary to avoid excessive shading and to improve air circulation. As well, growers should avoid excessive nitrogen fertilization. The relative humidity in the greenhouse should not exceed 85%, particularly at night, and water droplets should not be allowed to form and persist on leaves. If the greenhouse is not heated, ventilation should be increased and the lower leaves removed to improve air circulation around the plants. Excessive vegetative growth retards ventilation. Overhead watering and pesticide sprays should be applied early in the day to keep humidity low later in the day. Circulation of unheated air through the greenhouse aids spore dispersal and may keep the relative humidity high. Diseased leaves should be carefully pruned, placed in a plastic bag and destroyed. If leaf mold has been a problem in the crop, all plant residue should be removed and destroyed at the end of the season, and the entire greenhouse should then be disinfested.

Resistant cultivars — Susceptible cultivars can be grown if humidity is kept low but resistant cultivars are preferable. Although a tomato cultivar may carry resistance, leaf mold has a number of races, so the resistance genes must coincide with the local races. Commercial seed is usually labeled as to cultivar resistance to known races. Local authorities should be consulted about cultivars that possess resistance against local populations of the pathogen. Resistant cultivars include Caruso, Capello, Cobra, Vision (from the Netherlands), Buffalo, Trend, Pink KR15 and Pink CR-864 with gene *Cf-5*, Ultra Sweet and Ultra Pink with gene *Cf*-7, and Dombito, Jumbo, Furon and Vetomold with gene *Cf*-2.

Chemical control — Fungicides can be used to control this pathogen, but care must be taken in selection because fungicide-resistant strains of the pathogen exist.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.15 Septoria blight (septoria leaf spot) *Figs. 25.15; 18.13a-c*

Septoria lycopersici Speg.

This disease occurs occasionally in poorly ventilated greenhouse tomato crops and overcrowded plug-transplants (25.75). Tomato is the main host of *S. lycopersici*, but the pathogen can also infect black nightshade *(Solanum nigrum* L.) and several other *Solanum* species.

Symptoms Numerous, small, circular, water-soaked spots are produced on petioles, leaves, stems (*18.13c*) and calyces. Septoria leaf spot develops more quickly on the upper leaf surface than on the lower leaf surface *(18.13a).* Normally, older leaves are affected first. The spots have gray centers with black borders, or they may be solid black. In their centers, minute, black pycnidia can be seen under magnification *(18.13b).* Severely affected leaves turn yellow, dry and fall off. Fruit may be exposed to the sun as a result of defoliation, leading to sunscalded, leathery and/or bleached patches on the skin.

Causal agent *Septoria lycopersici* has hyaline, thin-walled hyphae. Hyaline, filiform conidia, measuring 3.2 by 67 pm with up to 10 septa, are produced in pycnidia, which average about 66 pm in width. The fungus is readily isolated directly from cirrhi of conidia extruded from the pycnidia. The best linear growth is obtained on tomato leaf extract agar, potato dextrose agar and carrot agar. The fungus produces mature pycnidiospores after 7 days on tomato leaf extract agar and tomato root extract agar. Optimum temperature for fungal growth on media is 22-25°C and for pycnidial maturation it is 17-28°C. Lesions of 5.*lycopersici* may resemble those of *Alternaria* spp. (see early blight, 25.9), but they have pycnidia and distinctive, long, filiform conidia.

Disease cycle The pathogen can survive from season to season in or on seed, diseased plant residue and infected hosts, and on contaminated greenhouse structures and equipment. Spores can be spread by splashing water, workers, equipment, insects, such as aphids, and wind-blown soil. Wet weather favors spread of the fungus. Growth is greatest at 15 to 25°C, and disease development requires 7 days at 20-26°C and 12 days at 15-20°C. At the higher and lower temperature ranges, infection requires 64 and 88 hours, respectively. In the greenhouse, the disease is favored by warm, dry days and dewy nights. At 100% relative humidity, only 9-10 days are required for the disease process from inoculation to formation of pycnidia. This saturated environment is necessary for abundant spore discharge from pycnidia.

Management

Cultural practices — Good weed control and sanitation in and around the greenhouse enhance disease control. Diseased leaves and stems should be carefully gathered and buried. Field tomato should not be planted close to greenhouses. Seed should be purchased from a reputable source.

Chemical control — Registered fungicides are available.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.16 Verticillium wilt *Figs. 25.16a,b*

Verticillium albo-atrum Reinke & Berthier
Verticillium dahliae Kleb.

This is a minor disease of greenhouse tomato in Canada. Both *Verticillium* species have been found on this crop, but *V. dahliae* predominates.

Symptoms (see Tomato, verticillium wilt, 18.14) In the greenhouse, the first visible above-ground symptom is the wilting of one leaflet or more on a single leaf. The oldest leaves usually show symptoms first, and then wilt develops progressively in the younger leaves. Plants that become infected during January, February and early March show sudden wilting of several leaves, which become characteristically patterned with bright yellow and brown *(25.16a).* These plants often die in a few days. During late spring and summer, affected plants usually survive in a considerably stunted, wilted condition *(25.16b).* Heavy watering may induce rapid upward movement of the fungus in infected plants.

Causal agent (see Potato, verticillium wilt, 16.20)

Disease cycle (see Greenhouse cucumber, verticillium wilt, 22.17)

Management (see Greenhouse cucumber, verticillium wilt)

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(Original by R.J. Howard)

▶ 25.17 White mold *Figs. 18.15a-e*

Sclerotinia minor Jagger
Sclerotinia sclerotiorum (Lib.) de Bary
(syn. *Whetzelinia sclerotiorum* (Lib.) Korf & Dumont)

White mold is occasionally serious in individual greenhouses. The pathogen has a wide host range (see Tomato, white mold, 18.15) and can attack most types of greenhouse vegetables (see Greenhouse cucumber, white mold, 22.18; and Greenhouse lettuce, drop, 23.8).

Symptoms (see Tomato, white mold)

Causal agent (For a description of *Sclerotinia sclerotiorum*, see Bean, white mold, 15B.9; and for *S. minor*, see Lettuce, drop, 11.9.)

Disease cycle (see Bean, white mold; and Lettuce, drop.)

Management (see Tomato, white mold, and Greenhouse cucumber, white mold)

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(Original by R.J. Howard)

VIRAL DISEASES

▶ 25.18 Cucumber mosaic *Figs. 25.18a,b; 18.17*

Cucumber mosaic virus

Cucumber mosaic occurs sporadically in British Columbia, Ontario and Quebec, but it has not been a severe problem. Cucumber mosaic virus has a broad natural host range throughout the temperate regions of the world. It can infect cereals, forages, woody and herbaceous ornamentals, vegetables and fruit crops.

Symptoms Cucumber mosaic virus infection of tomato produces a symptom known as "shoestring," in which the blade of the leaflet is much reduced (tendril-like) or absent, consisting of only the petiole *(25.18a).* Shoestring can be confused with "fernleaf," a symptom caused by tomato mosaic virus or tobacco mosaic virus, in which the leaflets are long and narrow but not as completely suppressed as in shoestring. This first appears about 10 days after infection and consists of a spindling appearance of the young leaves in the terminal bud. These leaves twist in a corkscrew fashion. Another early symptom is yellowing of the older leaves, especially along the veins *(25.18b).*

Causal agent (see Greenhouse cucumber, cucumber mosaic, 22.20)

Disease cycle Cucumber mosaic virus is transmitted by numerous species of aphids in a non-persistent or stylet- borne manner. Aphids can acquire the virus from an infected plant and inoculate healthy plants after less than one minute of feeding, and there is no latent or waiting period before the virus can be transmitted. Problems with cucumber mosaic usually occur only when aphids and host plants in which the virus can multiply are present throughout the year. Perennial weed species have been shown to harbor the virus throughout the winter, and several of them, including chickweed *(Stellaria media* (L.) Cyrill and *Cerastium* spp.), *Capsella* spp., corn spurry (*Spergula arvensis* L.), and red dead-nettle (*Lamium purpureum* L.), are known to carry the virus through their seed. The virus commonly infects chrysanthemum and may be spread to tomato by aphids. Once introduced into a greenhouse tomato crop, it can be further spread to healthy plants on tools or the hands of workers.

Management

Cultural practices — No direct control methods, such as viricides, are available, so most control strategies involve measures designed to reduce sources of infection from within or outside of the crop. Since contaminated seed does not appear to be a problem, only a few precautions are required to keep seedlings virus-free. Growers should control weeds within and near the greenhouse. Aphid control in the greenhouse is essential because viruliferous aphids are the most common means of virus introduction and spread. Any suspicious plants should be removed and destroyed. Care should be exercised to minimize mechanical transmission while handling plants. Dipping hands and tools in a solution of skim milk (100 g skim milk powder per litre of water) helps to minimize spread (see tobacco mosaic, 25.20).

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(Original by R. Stace-Smith)

▶ 25.19 Double streak *Figs. 25.19a,b*

Tomato mosaic virus and potato virus X

This disease occurs in plants infected by both potato virus X and tomato mosaic virus. Double streak is a minor disease of greenhouse tomato, but its impact on plant health and fruit production is usually greater than that caused by either virus alone (see tomato mosaic, 25.21, 18.18; other viral diseases, 18.20).

Symptoms The onset of symptoms is usually very sudden. Necrotic lesions form on the stem, petioles, leaves and fruit. Stem lesions (*25.19a*) appear as dark, longitudinal streaks extending through the cortex and into the pith beneath. The leaves often show necrotic spots that enlarge and blight the foliage. Fruits may be affected while still green and develop irregular, sunken, necrotic blotches (*25.19b).* Diseased plants are generally stunted and weakened and may die. Symptom expression is suppressed by air temperatures of 27°C or higher.

Causal agent (For tomato mosaic virus, see tomato mosaic, 25.21; for potato virus X, see Potato, mosaic diseases, 16.27).

Disease cycle (see tomato mosaic, 25.21) Double streak is usually found in tomato when potato is grown nearby or when workers handle potato plants before tomatoes. It also has been encountered when unsterilized soil containing potato debris has been used in greenhouses and in greenhouses constructed on land where potato has been grown earlier in the season.

Management

Cultural practices — Diseased tomato plants should be removed from the greenhouse and buried. Potato should not be planted near tomato greenhouses nor should it be handled before working in a tomato crop. Tomatoes should not be grown in soil containing fresh potato crop residues.

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(Original by J.G. Menzies and R.J. Howard)

▶ 25.20 Tobacco mosaic

Tobacco mosaic virus

Tobacco mosaic is a minor disease of greenhouse tomato. Tobacco mosaic virus is one of the most infectious plant viruses and has a host range of over 150 genera, including greenhouse pepper (see Greenhouse pepper, tobacco mosaic, 24.6) and field tomato and pepper.

Symptoms The symptoms of tobacco mosaic on greenhouse tomato are virtually indistinguishable from those of tomato mosaic (see tomato mosaic, 25.21).

Causal agent Tobacco mosaic virus is in the tobamovirus group. It is rod-shaped, measures 300 by 18 nm, and consists of a single helical strand of RNA with approximately 6400 nucleotides and 2130 protein subunits. Its thermal inactivation point in undiluted plant juice is 93°C. In dried infected leaves, it remains infective even after treatment at 120°C for 30 minutes.

Indicator plants commonly used for confirming tobacco mosaic virus include *Chenopodium. Nicotiana (N. glutinosa***,** *N. sylvestris* and *N. tabacum* cvs. Java, Turkish, Turkish Samsun, Samsun (Samsoun), Samsun NN, White Burley, Burley, Xanthi and Xanthi-nc), and *Phaseolus (P. vulgaris* cv. Pinto).

Strains of tobacco mosaic virus are closely related to tomato mosaic virus (see tomato mosaic, 25.21) and their names are often interchanged in the literature. They can be distinguished in the laboratory by serology and, to some extent, by symptoms on indicator plants.

Disease cycle Tobacco mosaic virus is soil-borne and survives in infested plant residues. It also can be seed-borne. The virus is not normally transmissible by insects. Field tomato and tobacco crops may be a source of infection for greenhouse crops, as may infected weeds, though the importance of the latter has not been determined. Spread of tobacco mosaic virus can be rapid because it is readily transmitted by rubbing infected plants against healthy ones, or with hands and tools contaminated with infected sap or residue from tobacco products. Symptom development generally is more severe with short photoperiods and low light intensities. Temperature also affects symptom development and severity in susceptible varieties; lesions develop 16 to 18 days after inoculation at 22 to 28°C, whereas symptoms may not occur at 16 to 20°C.

Management

Cultural practices — Seed should be obtained from virus-free plants. Heat-treated seed (70°C for four days) or seed obtained by acid extraction (see tomato mosaic, 25.21) also can be used safely. One to two days before planting, seed should be soaked for 15 minutes in a 10% trisodium phosphate solution (100 g/L of water), then rinsed thoroughly and spread to dry. Early in the season, diseased plants should be removed when noticed. Later in the season, roguing is ineffective because the virus will have spread and symptomless infected plants will be present within the crop. Plants with symptoms should be handled only after healthy or symptomless plants have been tended.

Sprays of skim milk may slow the spread of tobacco mosaic. The spray solution can be prepared by adding dried skim milk powder to water at the rate of 100 g/L. This mixture should be used each time the plants are pruned or trained, from the time of transplanting to the first fruit harvest. If symptoms are not evident on plants at first harvest, the sprays can be discontinued. If symptoms are noticed on plants after harvesting commences, the affected plants should be picked last, then sprayed with milk powder solution. If virus infection is suspected, workers should dip their hands in skim milk solution between handling individual plants. The mechanism by which skim milk inactivates tobacco mosaic virus is unclear, but it may be a result of binding of the viral particles by protein molecules, thus inhibiting their ability to infect plant cells.

Growers are advised to prohibit the use of tobacco products in the greenhouse and to require workers to wash their hands thoroughly with soap and water after using tobacco products. Clothing should be laundered daily in hot water with a detergent.

Resistant cultivars — Growers should select tomato cultivars with resistance to tomato mosaic virus (see tomato mosaic, 25.21) if tobacco mosaic is a potential problem.

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(Original by W.R. Jarvis, J.G. Menzies and R.J. Howard)

▶ 25.21 Tomato mosaic, single streak *Figs. 25.21a-e; 18.18a-e*

Tomato mosaic virus

This disease occurs wherever tomato is grown and can reduce both yield and quality of fruit (*18.18b-e*). The impact of the disease is even more severe if tomato mosaic and potato virus X occur as a mixed infection (see double streak, 25.19). Tomato mosaic virus can infect plants in the families Solanaceae, Aizoaceae, Amaranthaceae, Chenopodiaceae and Scrophulariaceae. Petunia, snapdragon, pepper and tobacco are frequent hosts.

Symptoms Symptoms of tomato mosaic vary with the strain of the virus, temperature, daylength, light intensity, plant age and tomato cultivar. Initially, affected plants may wilt in sunlight, especially if the crop is growing rapidly. The wilt is temporary, lasting up to two weeks. The leaves most commonly show a light to dark green mottling *(25.21 a,b).* There may also be a reduction in leaflet width, so that individual leaves resemble fern leaves. The lowest distorted leaf generally shows reduced serration; younger leaves show increasing simplification until the individual leaflets are reduced to a narrow strap of tissue *(18.18a).* These "fern- leaves" may have small enations on the underside. Plants recovering from severe leaf distortion produce pinnate, vetch-like leaves (fernleaf, *25.21c).* Eventually, new leaves are produced that are normal in shape but have a conspicuous mosaic. Six to eight leaves can be affected. Involvement of all leaves is not typical. Stem symptoms include pale to dark green or black stripes, often accompanied by yellow-brown older leaves. Plant size is generally reduced.

Brown markings and blotches occasionally occur on green and ripe fruit of greenhouse tomato cultivars that have some resistance (heterozygous for the gene *Tm2)* to tomato mosaic. These blemishes are often circular, up to 3 cm in diameter, and confined to the skin. Usually, the fruit of only one or two trusses is affected, but yield can be severely reduced on young plants. Blotches can appear on both green and ripe fruit, which soon drop off. Sunken areas, sometimes brown or black, and known as pits, may form at the calyx end of the fruit. Fruit pitting is generally restricted *(25.21d).* The virus also causes internal browning of the fruit wall *(18.18d,e)* and abortion of flowers and fruit, usually confined to trusses developing flowers at the time of infection.

Foliar streaking has been reported to be caused by at least one strain of tomato mosaic virus; this condition is referred to as single streak, single virus streak or glasshouse streak. The most characteristic symptoms are longitudinal, necrotic streaks on the stems, leaves and petioles, which sometimes kill the plant. In addition, sunken brown lesions may develop on the fruit *(25.21e).* Streak generally occurs at 26°C or below. Symptoms have been reproduced experimentally by grafting but not by sap inoculation.

Causal agent Tomato mosaic virus is an RNA virus. The particles are straight tubules, measuring 18 by 300 nm. Purified preparations sediment as a major infective component, sometimes also with dimers and trimers.

Indicator plants, such as *Brassica, Chenopodium, Cucumis, Datura, Gomphrena, Nicotiana, Phaseolus, Tetragonia* and *Vigna* spp., distinguish tomato mosaic from the type strain or tobacco forms of the virus, but none is infallible. On *Nicotiana glutinosa,* local lesions differ in size and incubation period from those of tomato spotted wilt and tomato bushy stunt. Strains of tomato mosaic virus are closely related to tobacco mosaic virus (see tobacco mosaic, 25.20).

Disease cycle The most important inoculum sources are seed, plant residues and soil. Alternative hosts in and around greenhouses are another source. Tomato mosaic virus is not normally transmissible by insects. In tomato seed, this virus has been found in the mucilage, testa and endosperm. The virus can survive in plant residue in the soil for up to two years and in moist soil for up to eight months, although its concentration decreases with time. People are the most important vectors of tomato mosaic in the greenhouse. Employees or visitors can spread the virus through contaminated clothing and pruning knives as they tend the crop or walk along and brush the plants. The virus can survive for up to three years on stored unwashed clothing. It is quickly inactivated by sunlight.

Management

Cultural practices — Seed should be obtained from healthy plants. As an extra precaution, one to two days before seeding, seed should be soaked for 15 minutes in trisodium phosphate solution (100 g/L) at room temperature, rinsed thoroughly and

spread out to dry. In addition, dried seed can be heated in an oven for four days at 70°C to eliminate surface-borne virus. Perhaps the best method of disinfesting tomato seed is to treat the fruit pulp with one quarter of its volume of concentrated hydrochloric acid and allowing it to sit for 30 minutes at room temperature before straining and washing the seed.

Seedlings and transplants should be grown in soilless mixes or in steam-pasteurized soil in which plant debris has been allowed to thoroughly decompose. Steam-pasteurizing may not kill virus particles in thick roots left in the soil. Seedlings should not be grown in areas of the greenhouse where tomato crops or other hosts are being grown. Infected plants should be removed from the greenhouse and destroyed. Contaminated equipment, tools and machinery should be cleaned by washing, then heat-sterilizing, dipping or rewashing in trisodium phosphate (3 kg/100 L of water) before reuse. Virus transmission between plants can be reduced if tools are frequently dipped in a solution of 10% trisodium phosphate. After working an infected crop, a change to freshly laundered clothing is suggested before going to a mosaic-free crop. Infested clothing should be laundered in hot water with a detergent.

Perennial plants and hanging baskets of ornamentals should not be tolerated in a tomato greenhouse, and greenhouses should be sealed with screens to exclude potential insect vectors.

Resistant cultivars — The most effective means of controlling tomato mosaic is through the use of resistant cultivars. Although most commercial cultivars are resistant to one or more strains of the virus, the resistance of some cultivars can be overcome if young plants are exposed to infection or temperatures exceed 30°C.

Biological control — Mild, almost symptomless strains of tomato mosaic are commercially available to vaccinate susceptible cultivars against more severe strains. While this technique can minimize disease losses, the mild strains may recover virulence and cause appreciable damage later in the season. For this reason, attenuated strains are rarely used in Canada.

Chemical control — Fumigation does not control tomato mosaic virus and may slow the rate of virus inactivation by reducing the populations of soil microflora that break down plant residues.

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(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.22 Tomato spotted wilt *Figs. 25.22a-d*

Tomato spotted wilt virus

This virus is common in temperate regions and can cause significant losses in both field and greenhouse tomato (see also 18.19). The host range of the pathogen includes approximately 300 species in 34 families of plants.

Symptoms The most common symptom of tomato spotted wilt is bronzing of young leaves (*25.22a,b),* followed by one-sided distortion, severe stunting and near-cessation of growth. Bronzing occurs as isolated spots or it may cover most or all of the leaf surface. The intensity of bronzing varies from an inconspicuous green to a brown, distinct dark-brown or almost black, glazed area. Bronzed areas usually roll inward and tissue in affected areas often dies. Necrotic lesions may develop on petioles. Fruits on affected plants develop spots about 1 cm in diameter with concentric, circular markings. Ripe fruits often are distorted and marked with alternate red and yellow bands (*25.22c,d).* Affected fruits may occasionally show internal browning.

Causal agent Tomato spotted wilt virus is an RNA virus with membrane-bound isometric particles, 70 to 90 nm in diameter. The structure of the material inside the membrane consists of a nearly continuous layer of projections about 5 nm thick that stain more densely than the membrane itself. Purified particles may show a tail-like extrusion. Physically and chemically, this is one of the most unstable plant viruses. There are many strains of the virus and the symptoms they produce may differ in severity.

Cucumis, Nicotiana, Petunia, Tropaeolum and *Vinca* spp. are suitable indicator plants for artificial inoculation. It is useful to keep a few petunia plants in a greenhouse as sensitive indicators of the presence of tomato spotted wilt virus.

Disease cycle The primary means of virus spread is by thrips. The virus may also be transmitted through seed. Disease spread is not serious unless thrips are present. Infected cuttings of ornamental plants and weeds can act as sources of infection. Only thrips can acquire the virus. This occurs after feeding periods of at least 15 minutes, after which they transmit it as adults by feeding.

The incubation period is 4 to 10 days; thrips become maximally infective 22 to 30 days after acquisition and may retain the virus for life. The virus is not transmitted from one generation of thrips to another.

Management The basis for control of tomato spotted wilt virus is sanitation, removal of alternative hosts, and thrips control (see thrips, 25.29).

Cultural practices — Infected plants should be removed and buried, and a 3- to 6-m-wide band around the perimeter of greenhouses should be kept free of weeds. Ornamental plants should not be grown in or around the greenhouse because they may act as reservoirs for the virus and its thrips vector.

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(Original by J.G. Menzies and W.R. Jarvis)

NON-INFECTIOUS DISEASES

▶ 25.23 Blossom-end rot *Figs. 25.23; 18.21a-d*

Blossom-end rot is usually associated with environmental stresses, such as drought or widely fluctuating moisture conditions and calcium deficiency within the fruit. It appears as a firm, dry, sunken, brown or black area on the blossom-end *(25.23, 18.21c*), although the discoloration may sometimes be totally within the fruit (*18.21d*). This disorder can be prevented by regulating available water and by providing supplemental calcium. (For more information, see Tomato, blossom-end rot, 18.21.)

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(Original by R.J. Howard)

▶ 25.24 Magnesium deficiency *Figs. 25.24a,b*

Magnesium deficiency is a common nutritional disorder of greenhouse tomato but yield losses are rare unless the shortage of this element is acute. The middle leaves usually show symptoms first. They tend to be brittle and may cup upwards *(25.24a).* The veins remain dark green and somewhat bluish, with a thin, dark green leaf margin *(25.24b).* Interveinal areas turn yellow then brown. The greater the distance from the vein, the more intense the discoloration. Stems and fruits have no obvious symptoms. Dead leaf tissues may be colonized by secondary microorganisms.

Management

Cultural practices — Growers should ensure that tomato plants receive an adequate supply of essential nutrients throughout the growing period. Where lime is required in the fertilizer program, a limestone that contains magnesium, such as dolomite, should be used. At the first appearance of symptoms, magnesium sulfate (Epsom salt) should be applied two or three times to the growing medium at 1.0 to 1.5 kg/100 m^2 or to the leaves as a spray at 5 kg/1000 L of water. Where the problem has occurred on previous crops, magnesium should be applied even before symptoms occur. Over-application of potassium may impede magnesium uptake.

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(Original by R.J. Howard)

▶ 25.25 Other disorders *Figs. 25.25a-e; 18.22-18.28*

Blotchy ripening
Catface
Edema
Growth cracks (russeting)
Puffiness

Blotchy ripening

Uneven ripening of the surface of the fruit is characteristic of this physiological disorder *(25.25a; 18.22a,b).* Inadequate nutrition, overcrowding, tomato mosaic and other factors may be responsible. Growers should follow a well-balanced fertilizer program to minimize the occurrence of blotchy ripening. (For more information, see Tomato, blotchy ripening, 18.22.)

(Original by R.J. Howard)

Catface

Catfacing is generally a minor problem on greenhouse tomato but can become serious if environmental control is poor and developing blossoms are injured by low temperatures. Affected fruits are misshapen and the blossom-end may be scarred *(18.23).* Maintaining optimum conditions for growth, especially air temperature, will minimize the incidence of catface. Good growing practices should be followed for crops destined both for fruit production and field transplanting. The cultivars Ohio WR25 and Ohio MR 13 seldom develop catface. (For more information, see Tomato, catface, 18.23.)

(Original by R.J. Howard)

Edema

In poorly ventilated greenhouses, and particularly in plastic greenhouses, a physiological disorder, edema, may occur. It is caused by waterlogging of the leaf tissues, which results in raised, blister-like growths, 2 to 5 mm in diameter, on the upper or lower leaf surface *(25.25b).* Green, callus-like growths result when transpiration is restricted and root pressure continues to pump water to the leaves (see Crucifers, intumescence, 8.21). Edema also occurs on fruits *(25.25c,d),* but it rarely affects yield and is easily counteracted by proper ventilation and watering.

(Original by W.R. Jarvis)

Growth cracks (russeting)

Growth cracking *(25.25e, 18.25*) is occasionally a problem in greenhouse tomato crops. Severe splitting can be avoided by careful watering. Russeting may be a less severe form of growth cracking in which the skin of fruit develops small cracks. Its occurrence is influenced by the ratio of fruit load to leaf area, but environmental factors such as relative humidity also may be involved. Reliable management strategies are not available. (For more information, see Tomato, growth cracks, 18.25.)

(Original by L.M. Tartier and J.G. Menzies)

Puffiness

This disorder *(18.28)* is usually a minor problem on greenhouse tomato. Proper temperature control and adequate humidity in greenhouses help to reduce this problem. (For more information, see Tomato, puffiness, 18.28.)

(Original by L.M. Tartier)

NEMATODE PESTS

▶ 25.26 Root-knot nematodes *Figs. 25.26; 18.30; 22.30a-d*

Northern root-knot nematode

Meloidogyne hapla Chitwood

Southern root-knot nematodes

Meloidogyne arenaria (Neal) Chitwood

Meloidogyne incognita (Kofoid & White) Chitwood

Meloidogyne javanica (Treub) Chitwood

Tomato, pepper and eggplant are very susceptible to damage from root-knot nematodes. Infected transplants from southern latitudes are a source of inoculum for *M. incognita, M. javanica* and *M. arenaria,* which can persist in greenhouses but apparently not outdoors in Canada.

Symptoms include stunting, chlorosis, early senescence, prolific branching of rootlets, and production of small, spherical galls on roots. Tomato plants infected by *M. incognita* may show larger, compound root galls *(25.26)* and purpling of the undersides of

the leaves, similar to symptoms of phosphorus deficiency. For a complete description, see Carrot, 6.20, and Greenhouse cucumber, 22.30.

Management Pasteurization of soil, fumigation, and planting of certified, nematode-free transplants are effective. See Carrot, 6.20; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 25.27 Greenhouse whitefly *Figs. 25.27a-g; 3.10e*

Trialeurodes vaporariorum (Westwood)

The greenhouse whitefly is the both the most common and the most serious pest of greenhouse tomato in Canada and is found in all regions where this crop is grown.

The greenhouse whitefly feeds on over 100 genera of broadleaved plants. It prefers tomato, cucumber and eggplant in greenhouses and is rarely a problem on greenhouse pepper. Contamination of greenhouse-propagated seedlings can result in infestations on various outdoor crops such as cucumber, tomato, eggplant, melon and related plants. The whitefly will feed and develop on most weeds in and around greenhouses, and it is found on a large number of commonly grown ornamentals.

There are no records of the greenhouse whitefly as a vector of virus or other plant diseases to tomato, but it can transmit beet pseudo-yellows virus in cucumber (see Greenhouse cucumber, 22.19).

Damage The greenhouse whitefly damages the plant by sucking sap from the phloem bundles in the leaves. Large infestations can cause yellowing and wilting of the leaves, and a general decline of the plants. When feeding, nymphs and adults eject a sticky, sugar-rich honeydew that coats leaves and fruit. Production costs increase because honeydew deposits must be washed from fruit. If fruit washing is a normal part of the packing process, then moderate honeydew deposits can be tolerated.

Heavy whitefly infestations decrease the vigor of the plant by their feeding. However, greater declines in yield and fruit quality may result from a black sooty mold *(25.27g)* that grows on the honeydew when the humidity is high. The mold is more damaging than the honeydew because it reduces photosynthesis and transpiration.

Identification The greenhouse whitefly (family Aleyrodidae) adult (*25.27b*) is about 2 mm in length and white with four, white-appearing wings. Nymphs (*25.27d*) are translucent, flattened scales, measure 0.5 to 1.0 mm in length, and are usually found on leaf undersides. Pupae are wider and more opaque than the nymphs. The sweetpotato whitefly (*3.10d-g*), which has been found on ornamentals in some greenhouses in Canada (see Foreign diseases and pests, 3.10), is slightly smaller than the greenhouse whitefly, holds its wings more vertically alongside the body (*3.10e*), and has a light yellow thorax. Its nymphs are more variable in size and more irregular in outline than those of the greenhouse whitefly. The pupal stage is used to distinguish between these two whiteflies. The pupa (puparium) of the greenhouse whitefly, viewed laterally, is ovoid with straight sides, 12 large setae and no caudal groove, whereas the sweetpotato whitefly has an irregularly oval, oblique-sided pupa (puparium) with a variable number of short, fine setae and a caudal groove posteriorly *(3.10g).*

Life history Adults are found on the underside of leaves *(25.27a),* mostly near the growing tip of the plant where they feed and lay eggs. Eggs usually are laid individually on the underside of leaves *(25.27c),* but they may be clustered in a circular pattern. Adult longevity varies from 42 days at 18°C to eight days at 27°C. At normal greenhouse temperatures (21 to 24°C), females lay from 150 to 300 eggs. The nymphs also develop on the underside of leaves and feed by sucking plant sap. The first-instar nymph is a mobile, non-feeding crawler. It moves about the leaf for a few hours before becoming a sessile "scale." After three molts, the scale forms a pupa. The adult emerges from the pupa through a T-shaped slit. The cycle from egg to adult is accomplished in 25 to 30 days at 21°C, or 22 to 25 days at 24°C. Optimal relative humidity for development is 75 to 80%.

The immature stages are often difficult to see. Consequently, they are often brought into greenhouses on seedlings and ornamentals. Adult whiteflies disperse in and between greenhouses by Hying. Because of their small size, they can be carried long distances on wind currents. The adult is attracted to yellow and can easily be spread on yellow clothing and yellow objects, such as buckets and picking containers. Adults and eggs are moderately cold tolerant and may survive the winter on hardy weeds in and around the greenhouse, becoming a source of infestation for the next cropping season.

Management Overlap of the new and old crop is the most frequent cause of early season greenhouse whitefly problems. Effective control measures combine cultural procedures and biological control, and chemical control only near the end of the growing season.

Monitoring — The standard method of monitoring for the greenhouse whitefly is a yellow sticky trap (*22.34d)* available from horticultural supply companies in a variety of shapes, sizes and shades of yellow, either as small, stiff, plastic cards (6 by 15 cm) or as sticky ribbons. Bright yellow is much more effective than orange-yellow. For effective early detection, there should be one trap per 100 plants. This density enables growers to detect the whitefly and time biocontrol releases well before serious infestations develop. The bottom of the trap should be level with the top of the plant canopy. Honeydew deposits and black sooty mold on plants help to indicate the location of developing infestations.

Yellow sticky ribbons (tapes) should be hung so that the middle of the ribbon is level with the top of the plant. The ribbons remove substantial numbers of whiteflies and are particularly useful in "hotspots." In this situation, they should be used at a rate of one ribbon for every two to five plants. They are generally compatible with the principal biological control agent (see below). After two to three weeks when the whitefly population has been brought under control, the ribbons should be removed because parasites continue to be caught on them.

The action threshold for biological control of the greenhouse whitefly is the first appearance of its adult or immature stages. Growers should release parasites when the population level of whiteflies is low, otherwise biological control will not be successful. Traps also can be used to reduce whitefly populations in small greenhouse areas.

Cultural practices — Sanitation is one of the most important strategies for preventing whitefly infestations in greenhouses. Weeds should be removed and a deadband (3 to 6 m) maintained around the perimeter of the greenhouse. Vents can be screened to prevent the entry of whiteflies from outside the greenhouse, though this may cause ventilation problems unless overall intake area is increased. Non-crop plants should not be brought into the greenhouse, and movement of people and equipment from infested to non-infested areas should be restricted. All crop residue must be removed from the site at the end of the season and immediately destroyed by burying in a landfill to prevent reinfestation of the greenhouse.

Biological control — The principal agent for biological control of the greenhouse whitefly is the parasitic wasp *Encarsia formosa* Gahan (*25.27f*), which lays its eggs in the third and fourth instars of the immature whitefly. Direct feeding on first and second instars also occurs. The parasite larva develops inside the whitefly, eventually causing the whitefly pupa to turn black (*25.27e*). The parasite requires 25 to 35 days to complete development at 21 °C and 16 to 25 days at 24°C.

Introductions of the parasite are made as pupae and should begin as soon as adult or immature stages of the whitefly are detected. Parasites should be introduced weekly at a rate of one parasite pupa per four plants until 80% of whitefly pupae are black from being parasitized. In British Columbia, introductions of *E. formosa* are made routinely over the entire season, regardless of the presence of the whitefly. For optimum effectiveness, the daytime air temperature in the greenhouse should be around 24°C. Temperatures below 18°C severely inhibit the parasite's searching behavior and development. Tomato plants could be pruned less vigorously and leaves that are pruned should be left in the greenhouse to allow time for the adult *E. formosa* to emerge from any parasitized whitefly pupae on the pruned leaves.

Every year, more growers are using *E. formosa* for control of whiteflies. In 1988, over 65% of the greenhouse tomato industry in Canada used *E. formosa* to control the greenhouse whitefly.

Chemical control — Insecticidal treatments may be used to control whitefly outbreaks, but most are effective only against the adult stage. Applications should be spaced no more than four to five days apart and four to five weeks will be required to control all whitefly stages. If whiteflies are present at the end of the season, the crop residue should be fumigated before removal.

No information is available on treatment thresholds but, as a rule, insecticides should be applied no later than the first appearance of honeydew on the leaves or fruit. Heavy treatment schedules have resulted in greenhouse whitefly populations resistant to all registered insecticides in many regions of Canada.

Most insecticides are harmful to the parasitic wasp *E. formosa*. Synthetic pyrethroids may have a negative, residual effect for 50 days from the time of treatment. Attempts to reduce high whitefly populations with insecticides before introducing *E. formosa* are usually ineffective, making early detection and parasite releases essential.

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(Original by J.L. Shipp and D.R. Gillespie)

▶ 25.28 Leafminers *Figs. 25.28a-e*

Chrysanthemum leafminer *Liriomyza trifolii* (Burgess)
Vegetable leafminer *Liriomyza sativae* Blanchard

Leafminers occur only sporadically on greenhouse tomato crops. The chrysanthemum leafminer usually is seen only where tomato is grown in close proximity to greenhouse flower crops, particularly chrysanthemum and gerbera. Once in a greenhouse, leafminers should be controlled because populations can increase rapidly and cause substantial losses.

Damage Tomato fruit is not directly affected but damage to leaves (*25.28b,c*) may be extensive. This has the same effect as defoliation, causing reduced yields.

Identification Adults of both flies (family Agromyzidae) *(25.28a,d)* are small, yellow and black (about the same size as a vinegar fly, *18.42g).*

Life history Eggs are laid in leaf tissue (*25.28a*) and hatch into larvae that tunnel between the upper and lower leaf surfaces. The larvae complete development in four to seven days at summer temperatures and drop to the soil to pupate. Adults emerge 5 to 10 days after the larvae have dropped.

Management Leafminers in tomato are most effectively controlled biologically. A strict sanitation program also is helpful in preventing infestations.

Monitoring — Yellow sticky traps (*22.34d*) are excellent for monitoring leafminer flies. They can be used to detect first invasions and to monitor the success of control measures.

Biological control — Parasitic wasps (*25.28e) Diglyphus isaea* (Walker) and *Dacnusa sibirica* Telenga are available commercially and should be used if biological control programs are in progress for other pests.

Chemical control — No effective pesticides are registered for use against leafminers on greenhouse tomato.

(Original by D.R. Gillespie and J.L. Shipp)

▶ 25.29 Thrips *Figs. 25.29; 22.34e-g*

Onion thrips *Thrips tabaci* Lindeman
Western flower thrips *Frankliniella occidentalis* (Pergande)

Onion thrips (see Onion, 13.27) and western flower thrips (see Greenhouse cucumber, 22.34) may infest greenhouse tomato crops. The life history is similar for both species. Adult females insert their eggs into leaves, tender plant tissues and sometimes even fruit. The nymphs (or larvae) hatch and feed on the leaves for 5 to 10 days, depending on temperature, then drop to the soil and undergo pupation for about five days before emerging as adults.

Western flower thrips *(25.29, 22.34e-g)* is particularly important because it may be a vector of tomato spotted wilt virus. Onion thrips is rarely a problem on greenhouse tomato and it has not been reported to transmit tomato spotted wilt virus in Canada.

Damage Some strains of western flower thrips have adjusted to feeding on tomato and can cause severe leaf damage. Symptoms of feeding by adults and nymphs of the western flower thrips and the onion thrips are identical (see Greenhouse cucumber, 22.34). In general, thrips confine themselves to the lower leaves on greenhouse tomato and rarely affect new growth or leaves that are actively photo- synthesizing.

Management If tomato spotted wilt is present in the greenhouse, or nearby, every effort should be made to control the virus and its principal vector, the western flower thrips.

Cultural control — Greenhouse vents and doorways can be screened to prevent an influx of thrips. Empty greenhouses can be heated to 35°C for five days or 40°C for two to three days to hasten thrips pupal development; any emerging adults will starve. Soil also can be steam pasteurized to kill pupae and adults.

Chemical control — Thrips may be controlled by using procedures described for western flower thrips (see Greenhouse cucumber, 22.34). Onion thrips alone can be controlled by applying insecticides to the floor of the greenhouse to kill the pupal stage.

(Original by D.R. Gillespie and J.L. Shipp)

▶ 25.30 Other insect pests *Figs.: see text*

Aphids
Caterpillars

Aphids, such as the green peach aphid and the potato aphid, sometimes colonize the lower stems of greenhouse tomato plants. The life histories of these and other aphids are identical in all respects on other crops where they occur (see Potato, 16.40-16.43). Damage is usually inconsequential unless the stem is colonized near developing fruit. When that happens, honeydew must be washed off the fruit and chemical control should be implemented. Biological control of aphids (see Greenhouse pepper, green peach aphid, 24.12) may help when aphid numbers are low.

Caterpillars of various species of moths, such as the cabbage looper (see Crucifers, 8.40), corn earworm (see Maize, 12.13), also known as the tomato fruitworm, and European corn borer (see Maize, 12.16) are occasional pests on greenhouse tomato. Their development is accelerated in the greenhouse and several generations may occur compared with only one or two generations per year in the field. No specific parasites or predators are available for release in greenhouses, but a non-specific egg parasite *(Trichogramma* sp.) might be tried if available. Where biological control programs are present, growers should use a microbial insecticide containing a formulation of *Bacillus thuringiensis* Berliner.

(Original by D.R. Gillespie and J.L. Shipp)

MITE PESTS

▶ 25.31 Tomato russet mite *Fig. 25.31*

Aculops lycopersici (Massee)

The tomato russet mite is found in areas of the United States bordering southern Canada. It is reported to overwinter in unheated greenhouses and on weeds in the United States but its ability to survive the winter in Canada is unknown. Outbreaks have been reported on greenhouse tomato in British Columbia, Ontario and Quebec.

Hosts are, in general, plants in the Solanaceae. Nightshade (*Solanum* spp.) and petunia are mentioned as frequent sources of infestation, but the tomato russet mite may occur on other solanaceous weeds and crops.

Damage The tomato russet mite can cause severe losses, although only a few cases have been noted in greenhouses in Canada. Leaf symptoms *(25.31)* include yellowing, curling and wilting. Flowers may abort and fruit may be deformed. If not controlled, the tomato russet mite will eventually kill tomato plants.

Identification The tomato russet mite (family Eriophyidae) adult is about 0.2 mm in length and 0.05 mm in width. Because of its small size, the mite is not noticed on plants until it reaches damaging levels. Aggregations of the mite on stems, leaves and fruit have a beige or bronze appearance.

Life history Tomato russet mite females lay from 10 to 50 eggs during a lifespan of 20 to 40 days. High rate of reproduction and rapid development are favored by moderate temperature (21°C) and low humidity (30% RH). Under these conditions the life cycle can be completed in six to seven days.

Adult tomato russet mites disperse by wind and may move from the United States into Canada with weather systems. Importations on tomato plants from the southern United States have been suspected but never proven.

Management

Cultural practices — Plants showing wilting or poor growth should be examined for tomato russet mites. Greenhouses should be thoroughly cleaned between crop cycles and old plants, weeds, crop residue and debris should be removed to prevent carry-over from one crop to the next. If an infestation has occurred, the greenhouse structure should be washed and the soil steam-sterilized if possible. A relative humidity of 70 to 80% will help to slow the development of damaging population levels.

Biological control — Various predators will feed on the tomato russet mite, for example, the mites *Amblyseius* (syn. *Neoseiulus*) *cucumeris* Oudemans (*22.34h*) and *Metaseiulus* (syn. *Typhlodromus) occidentalis* (Nesbitt), and the minute pirate bug *Orius tristicolor* (White) *(22.34i).* These are available commercially but have not been used successfully against the tomato russet mite.

Chemical control — Sulfur, formulated as a liquid or dust, or with other materials in such products as lime-sulfur and nicotine sulfate, provides control and can be integrated with biological control.

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(Original by D.R. Gillespie)

▶ 25.32 Two-spotted spider mite *Figs. 25.32; 22.36a-g*

Tetranychus urticae Koch

This mite (see Greenhouse cucumber, 22.36) is becoming a more common problem on greenhouse tomato in many areas of Canada, particularly in British Columbia. Its biology is similar to that described on greenhouse cucumber but outbreaks tend to spread less rapidly on greenhouse tomato, which is probably related to the presence of sticky glandular hairs (trichomes) on tomato petioles and stems.

Management The two-spotted spider mite *(25.32, 22.36e,f*) can cause severe damage to greenhouse tomato and infestations should be treated promptly. Control may involve applications of miticides, or a program of biological control using sustained releases of a predatory mite.

Biological control — The mite *Phytoseiulus persimilis* Athias-Henriot *(25.32, 22.36g*) is an effective predator of the two-spotted spider mite but it is severely inhibited by the sticky hairs on tomato, which makes its distribution on tomato plants uneven. For this reason, greater numbers of the predator should be used on tomato than on greenhouse cucumber and they should be applied to every leaf that is infested. Growers should consult a biological control supplier for specific rates and timing of introductions.

(Original by D.R. Gillespie and J.L. Shipp)

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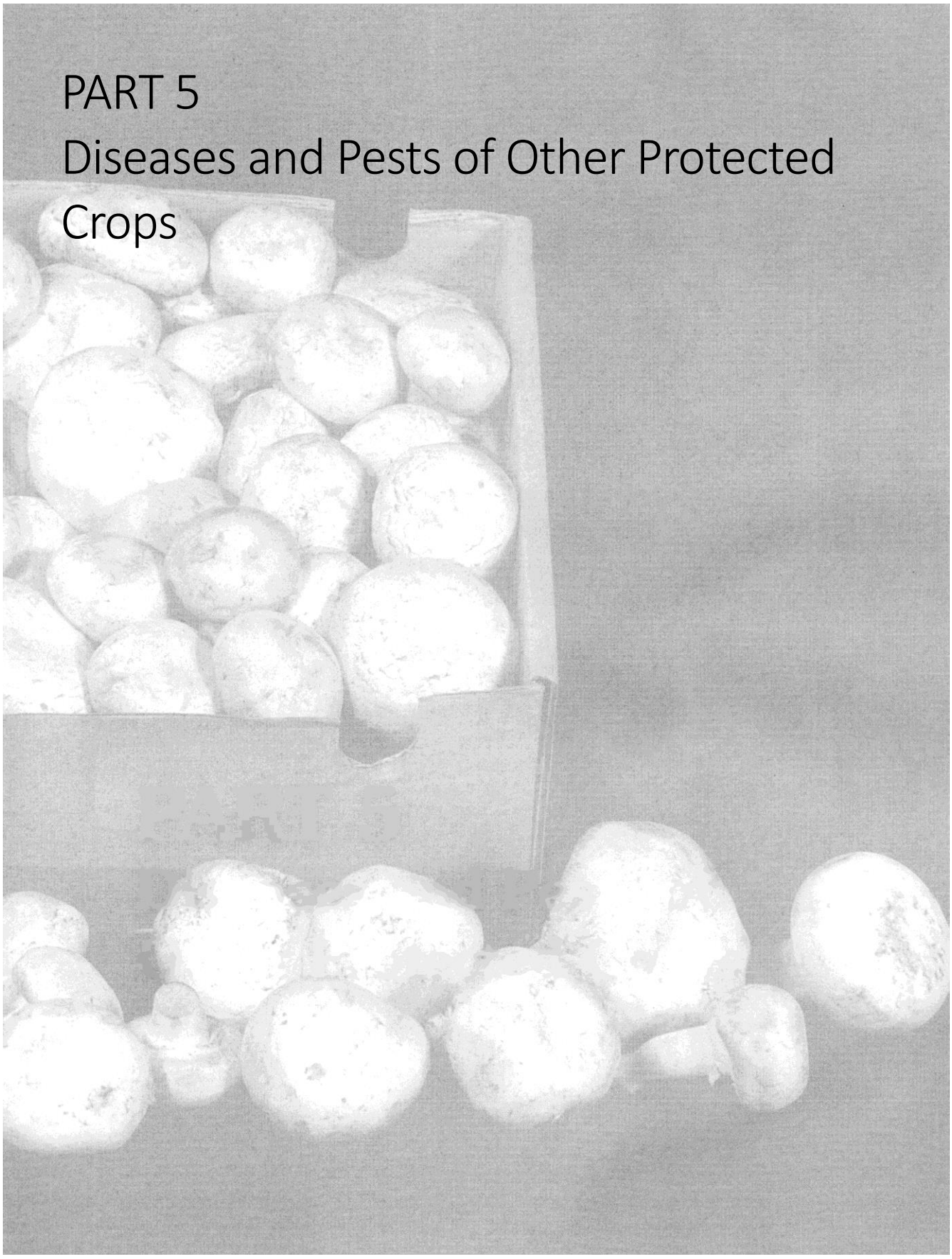
PART 5
Diseases and Pests of Other Protected Crops



Locating Text Sections and Figures

Text sections are numbered consecutively within each chapter. For example, section 16.2 describes bacterial soft rot, the second topic of Chapter 16, Potato. To find a text section, refer to the running heads, which carry the inclusive section numbers for each two-page spread.

Color illustrations, grouped near the back of the book, appear in the same order and have the same number as the corresponding text section; for example, figures *16.2a* and *16.2b* illustrate the symptoms of bacterial soft rot of potato. Line drawings, halftones and tables are numbered similarly, except that a text figure number contains the letter T; for example, Figure *16.2T1* illustrates the disease cycle of bacterial soft rot of potato.

BACTERIAL DISEASES

▶ 26.1 Brown blotch (bacterial blotch) *Fig. 26.1*

Pseudomonas tolaasii Paine

Brown blotch is the most common bacterial disease of commercial mushrooms, and it causes considerable economic losses each year through quality reduction.

Symptoms The symptoms most often observed include pale yellow areas or blotches on the cap that later turn golden yellow, yellow-brown or chocolate brown *(26.1)*. The stem (stipe) also may be affected. Occasionally, the caps will have an overall dingy

off-color with rapid deterioration and discoloration after harvest. Symptoms occur more frequently on mushrooms that remain wet for a long time and in places where they touch one another. Brown blotch symptoms can be confused with those of other diseases, such as verticillium spot.

Causal agent *Pseudomonas tolaasii* is an aerobic, Gram-negative, non- sporing rod, about 0.5 by 1 to 2 µm. It is fluorescent, oxidase-negative and arginine dihydrolase-negative. It can utilize L-arginine and L-arabinose, and does not grow at <4°C. It is white-line positive when challenged with ATCC 14340 (American Type Culture Collection, Rockville, MD). Drops of broth culture can induce browning on fresh slices of internal pileus tissue within 24 hours. Dissociation is common. The reference culture is ATCC 33618.

Disease cycle The pathogen is a natural inhabitant of the peat and lime used for casing material and can be isolated easily from compost after pasteurization. It probably survives between crops on structural surfaces, in debris, and on tools and other equipment. The bacterium can be moved readily from one crop to another on hands of pickers and on materials or equipment used in harvesting, by insects, mites and water droplets, and on mushroom spores. Once the disease is established, watering the crop will readily disperse the pathogen. Generally, disease incidence is highest in the first break. As the crop matures, there are fewer mushrooms and air circulates more freely around the crop resulting in better drying after watering and less disease.

Management

Cultural practices — Brown blotch is best managed by manipulating the growing environment. High relative humidity and surface wetness encourage the expression of symptoms. When the mushrooms stay wet longer than two to three hours, blotch can easily develop. Additional ventilation after watering will assist in drying. Maintaining a stable difference of 1 to 1.5°C between wet and dry bulb readings will lessen the chance of condensation and significantly reduce the incidence of blotch. When blotch is a problem, the crop should not be watered on consecutive days and growers should avoid watering mushrooms that are within one or two days of being harvested.

Chemical control — Chlorinated water may lower the bacterial population on the mushroom surface and thereby reduce the amount of blotch; however, chlorine alone is not a cure for this disease. Growers should consult the Health Protection Branch, Health Canada, for guidelines on the use of chlorinated water on mushrooms. The use of chlorine may negate the effectiveness of fungicides previously applied to the casing surface through a process of chemical inactivation. Proper management humidity and recommended watering practices also must be followed.

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.2 Mummy *Fig. 26.2*

Pseudomonas sp.

Mummy disease is frequently encountered in mushroom crops; however, an assessment of annual economic loss is not available. When present, the loss is typically only in a portion of any mushroom production chamber.

Symptoms There is no known effect of this disease on spawn or case run, but once fruiting has begun the symptoms appear. The first symptom may be a delayed first break. Mushrooms affected by mummy disease are characterized by curved stems with tilted caps *(26.2).* At the base of the stem, the rhizomorphs are stringy and cling to the casing. The base is frequently swollen and covered with a fluffy growth of mycelium. When harvested, a large amount of casing adheres to the base. Affected mushrooms die, become dry and are tough and leathery. Harvesters can often detect the disease by the tough feel of the stems when cut. Internal stem tissue will often have longitudinal brown streaks or be discolored, and when cut across, minute brown spots can be seen.

Causal agent The pathogen is suspected to be a fluorescent pseudomonad, proximate to *Pseudomonas tolaasii.* Goor *et al.* (see Selected references) provided discriminating characteristics for the mummy pathogen, but the pathogenicity of these cultures from the National Collection of Plant Pathogenic Bacteria, Harpenden, England, was not verified. Other than the original report on causation by Schisler *et al.* (see Selected references), no one has satisfied Koch's postulates with any isolate from symptomatic mushrooms and it has not been possible to induce disease with the reference culture ATCC 25415 from the American Type Culture Collection, Rockville, MD. Disease symptoms are distinctive and reliable for diagnosis. However, confirming the identity of the pathogen requires further study.

Disease cycle The pathogen spreads intracellularly through infected mycelium and not by spores. In a shelf system, the rate of spread is quite rapid, 10 to 30 cm per day. In the tray and bag systems, where mushrooms are cultivated in more discrete units, this symptom is less apparent. However, once the disease develops, the area will not produce harvestable mushrooms. Mummy is often observed in crops where the compost is exceptionally wet after pasteurization and especially when casing is applied over compost in which excess water has accumulated during the spawn-run period.

Management

Monitoring — Compost should be examined for wetness after the pasteurization of phase II compost. Wet areas on the compost surface at casing time or areas where the casing dries rapidly should be carefully examined for the disease.

Cultural practices — Once mummy has been identified, diseased areas should be isolated from uninfested ones by completely removing the compost at least 1.5 m on either side of the affected area to a width of 20 cm and covering them with plastic. At the end of the crop, the compost should be thoroughly pasteurized. Netting and shelving should be carefully cleaned and disinfested before re-use.

Chemical control — Infested netting and shelving should be treated with a formalin solution or hydrated lime.

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(Original by D.L. Rinker and P.J. Wuest)

FUNGAL DISEASES

▶ 26.3 Cobweb (soft mildew) *Fig. 26.3*

Cladobotryum dendroides (Bull.:Fr.) W. Gams & Hoozemans
(syn. *Dactylium dendroides* (Bull.:Mérat) Fr.)
(teleomorph *Hypomyces rosellus* (Albertini & Schwein.) Tui.)

Cobweb occurs infrequently. However, it occasionally can be widespread and destructive on individual mushroom farms.

Symptoms Cobweb disease occurs only on the casing material and may appear at any stage from pinhead onward. Circular patches of white mycelium attack the mushrooms and cover them with a coarse, white growth *(26.3).* Affected mushrooms turn brown and rot. Cobweb mycelium turns pink or red as it ages.

Causal agent *Cladobotryum dendroides* grows rapidly on malt, oatmeal, and potato-dextrose agar. Growth is aerial, cottony and grayish, and a red pigment develops in the medium after five to seven days. Conidia (26 to 32 by 10 to 13 µpm) are two- or three-celled, hyaline, and originate on erect, simple or branched conidiophores (annellophores).

Disease cycle The pathogen is a soil-inhabiting fungus and can be introduced into the casing with soil, spores or mycelium. If the casing is contaminated with spores, symptoms will not develop until the fourth or fifth break. However, when the casing is infested with mycelium, symptoms may occur on the first break. Humidity greater than 90%, air temperatures greater than 18°C, and water condensation encourage the growth of cobweb.

The pathogen is spread via air-borne spores, workers and infested casing material. Wild mushrooms can serve as a host and reservoir for the pathogen. Inadequately pasteurized post-crop compost can serve as a medium for pathogen growth and reproduction.

Management (see verticillium disease, 26.8.)

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.4 Green mold *Figs. 26.4a,b*

Trichoderma harzianum Rifai (teleomorph *Hypocrea vinosa* Cook)
Trichoderma koningii Oudem.
(teleomorph *Hypocrea ceramica* Ellis & Everh.)
Trichoderma viride Pers.:Fr.

(teleomorph *Hypocrea rufa* (Pers.:Fr.) Fr.)

Green mold is frequently grouped with the non-infectious molds as an indicator of compost quality. However, some species can reduce crop yield and quality.

Symptoms The white, fluffy mycelial growth will turn green as spores are produced *(26.4a,b).* Sporulation may be observed on compost before casing is applied, as well as on the casing material where it can produce symptoms that resemble cobweb mildew. *Trichoderma koningii* tends to sporulate late in the crop towards third break, whereas *T. viride* will sporulate any time during the cropping period. The pathogen may attack the mushroom cap, resulting in a reddish- or purple-brown coloration that can be confused with symptoms of verticillium spot.

Red pepper mites (see Mite pests, 26.32) are a good indicator species of green mold growing in the compost. They feed on the spores and mycelium of *Trichoderma* and large populations can build up in an infected crop. When mushrooms are fruiting, these red-colored mites will usually aggregate on the cap surface.

The use of protein supplements at spawning or casing can stimulate the growth of *Trichoderma,* especially if the distribution of the supplement in the compost is not uniform or is clumped.

Causal agent *Trichoderma* spp. grow very rapidly on general isolation media. The growth is appressed, sometimes tufted and usually covers a petri dish in a few days. The vegetative mycelium is septate, hyaline and initially gray to white. The colony appearance changes to gray-green when conidia develop. Phialides arise alternately, in pairs or in verticils, and are usually at right angles to the parent branch. Phialides are narrow, 25 to 70 by 2 to 5 pm, with a distinctive taper at the apex where the conidia are borne in masses, 10 pm in diameter. Conidia are hyaline or green, non-septate, smooth, small, and either globoid to ovate or oblong to elliptical, depending on the species. The relative importance and pathogenicity of *Trichoderma* spp. associated with this disease require further study.

Disease cycle The optimum temperature for growth of *Trichoderma* spp. is 22 to 26°C. Sporulation can be observed within 10 days of infestation. The fungus grows particularly well in substrates where the pH is below 6. Compost with a carbon to nitrogen ratio (C:N) of 22:1 will favor development of the pathogen. Normal compost at spawning should have a C:N ratio of 15:1 to 18:1.

Trichoderma species are readily found in soil and on organic matter. The spores are easily dispersed by air currents, water, mites and mechanical means. They can infest mushroom crops at any stage of production.

Management

Cultural practices — Careful preparation of compost will reduce green mold in mushroom crops. A carbon: nitrogen ratio of 15:1 to 18:1 should be achieved. Attention to quality control throughout the compost-making process will produce a properly digested, balanced and selective compost. Supplements should be thoroughly mixed to avoid clumping.

Spawning in a clean-air environment reduces the likelihood of infestation by *Trichoderma* spp. Direct infection of mushrooms can be reduced by lowering the humidity in the growing room.

Standard post-crop pasteurization (65°C) for eight hours is also recommended. Production rooms where crops were severely affected by green mold should be re-steamed before re-filling for the next cropping cycle.

Chemical control — Registered fungicides are available in Canada.

Selected references

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.5 Mat and confetti *Fig. 26.5*

Mat (vert de gris)

Chrysosporiwn luteum (Cost.) Carm.

Confetti

Chrysosporium merdarium (Link:Grev.) Carm.
(teleomorph *Gymnoascus uncinatus* Eidam)

Mat and confetti diseases occur infrequently, and losses to the mushroom industry as a whole are not known. However, losses on individual farms may be quite severe. Mat disease also is called vert de gris.

Symptoms Primordia (pins) do not form in the casing and, consequently, mushrooms do not develop. The cottony, white mycelium of C. *luteum* may aggregate in a distinctive matted layer located between the compost and the casing. Confetti-like

mycelial mats interspersed throughout the compost are caused by C. *merdarium.* These small mats yellow as they age *(26.5)* and may be difficult to see.

Causal agent *Chrysosporium luteum* has irregularly branched mycelium. Small oval conidia, 3.0 to 4.5 µm, are borne irregularly on swollen cells, and short chains of two or three conidia may be produced on a pedicel. Cultures are white when young, but yellow after a few weeks incubation.

Conidia of *C. merdarium* are typically formed on lateral branches as alternate arthroconidia; they are subglobose or pyriform with a broadly flattened base or cuboid when intercalary, smooth-walled to conspicuously roughened, and mostly 5 to 6 by 4 to 5 µm. Colonies on potato-yeast-extract agar develop intensely yellow centers that turn green or reddish brown after three to four weeks.

Disease cycle The two species of *Chrysosporium* infest compost at spawning, ramify concurrently with mushroom spawn, and infect after the casing is applied. Both species tend to be more serious when mineral soil is used as casing rather than peat moss. Formation of mats, within the compost for confetti disease, and on the surface of the compost for mat disease, reflects the growth stage of the pathogen and its interaction with the mushroom mycelium. Little evidence exists on the nature of pathogenesis, but it is thought that secondary metabolites interfere with the formation of the mushroom primordia.

The spores of these pathogens are very small and readily air-borne. Initial infestation of compost likely occurs from sources outside of mushroom operations, for example, when surrounding fields are plowed or eroded by wind. Infested compost can also be a source of large amounts of inoculum. Compost exposed to inoculum of *Chrysosporium* when it is spawned will be thoroughly infested and crop losses will result.

Management

Cultural practices — Mat and confetti diseases, when present in compost, can only be managed through pasteurization (12 hours at 70°C) before the compost is removed from the growing rooms. *Chrysosporium* species are more difficult to control than other molds, hence the need to use a higher-than-normal temperature and a longer time for pasteurization. Otherwise, disease management is based on reducing the number of spores released into the air in the vicinity of the spawning operation. High efficiency particle (HEPA) filters and positive air pressure should be used in the spawning area to minimize infestation of the compost.

Selected references

Allard, C. 1961. Sur les myceliophthora du champignon de couche (*Psalliota hortensis* Cooke). *Ann. Epiphyties* 12:263-291.
Carmichael, J.W. 1962. *Chrysosporium* and some other aleuriosporic hyphomycetes. *Can.J. Bot.* 40:1137-1173.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.6 Sepedonium yellow mold *Fig. 26.6*

Sepedonium niveum Massee & Salmon (teleomorph *Hypomyces* sp.)

Heavy infestations of yellow mold have been associated with yield reductions.

Symptoms The white mold produced by *Sepedonium* turns dull yellow to tan with age *(26.6).* It competes with the mushroom mycelium. In bulk pasteurization and conditioning methods, it has been found at the bottom layers of the tunnels.

Causal agent *Sepedonium* conidia (aleuriospores) are large (13 to 17 µm), globose, thick- and rough-walled, light yellow and borne singly at the apex of short conidiophore branches. In culture, the thallus is white when young, then turns golden yellow both in and on the substrate.

Disease cycle The spores of *Sepedonium* are resistant to high temperature and may easily survive peak heat. They can spread to the compost by air currents during the filling and spawning operations, or during spawn-run. Unpasteurized or spent compost sticking to beds or trays can spread this mold to the crop.

Management

Cultural practices — Yellow mold can be prevented through careful attention to hygiene and by proper air filtration. Careful monitoring of phase II and postcrop pasteurization temperatures are also necessary.

Selected references

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.7 Truffle *Fig. 26.7*

Diehliomyces microsporus (Diehl & Lambert) Gilkey

Truffle disease is encountered infrequently in commercial mushroom operations. However, when present, it can cause significant yield losses.

Symptoms Affected areas are circular, about 0.8 to 2.0 m in diameter, and generally exhibit poor growth. Mushrooms on the periphery of such areas experience premature opening of their veils and the stems are thicker than normal; mushrooms may not develop until later breaks. Compost beneath the affected areas becomes soggy, sunken and brown, and the spawn sometimes disappears. Ascocarps (truffles) of the causal fungus develop in infested compost as early as the time of spawn colonization or as late as third break, depending on spawn strain.

Causal agent *Diehliomyces microsporus* has creamy-white mycelium (26.7), in which convoluted ascocarps resembling a calf's brain develop. Oval asci contain eight golden brown, smooth-walled ascospores, and each ascospore contains an oil droplet. Ascocarps rarely form in pure culture and usually require the presence of *Agaricus bisporus* spawn to develop.

Disease cycle The pathogen enters production houses either in mud in hay, straw bales or on unpaved composting wharves, or as air-borne inoculum from previously infested compost. This fungus grows concurrently with the vegetative mycelium of *Agaricus bisporus,* forming ascocarps throughout the compost when its thallus reaches a certain level of maturity. Ascocarps will not form in the presence of all mushroom strains, which can make it difficult to confirm its presence in the compost. It is assumed that this pathogen produces metabolites that inhibit the continued development of primordia into mushrooms.

Not all soils harbor *D. microsporus,* but it is impossible to discriminate between soils that are and are not infested. Once the disease has become established, the pathogen is able to infest wooden shelves or trays and survive in the wood.

Management

Cultural practices — Baled straw should not be allowed to become muddy, and compost should be made on a concrete surface to reduce the likelihood of introducing the pathogen. Wood can be disinfested only by subjecting it to a thorough pasteurization with steam; a temperature of 75°C for 6 to 12 hours is recommended for most woodwork. Infested compost should be pasteurized at 60 to 65°C for 18 hours before it is removed from a growing room. When the pathogen is not killed, propagules can readily contaminate compost during the filling or spawning stages, and infestation at either of these times poses a serious threat to the crop.

Selected references

Kligman, A.M. 1944. Control of the truffle in beds of the cultivated mushroom. *Phytopathology* 34:376-384.
Sinden, J.W. 1971. Ecological control of pathogens and weed-molds in mushroom culture. *Annu. Rev. Phytopathol.* 9:411-432.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.8 Verticillium disease (dry bubble, split stipe, verticillium spot) *Fig. 26.8*

Verticillium fungicola (G. Preuss) Hassebring
(syn. *Verticillium malthousei* Ware)

Verticillium disease is the most significant fungal disease of commercial mushrooms, resulting in losses of approximately $7 million annually in Canada.

Symptoms The various names for the disease are descriptive of symptoms resulting from infection at different stages of mushroom development. Early infection of the developing mushroom primordium disrupts its growth, causing it to form a ball-like mass (dry bubble), 0.5 to 1.0 cm in diameter. Infection of older mushrooms (button stage) causes the stem to shatter and the cap may tilt slightly (split stipe). If the pathogen infects the cap tissue, the area turns brown *(26.8)* and the diseased tissue may have a grayish hue (verticillium spot). Lesions are generally less shiny than those caused by bacterial blotch. These brown spots will eventually produce a grayish-white "bloom" of *Verticillium* spores.

Causal agent A selective medium has recently been developed for isolated *V. fungicola* from casing soil, debris in mushroom production rooms, on- farm premises, flies and mushroom sporophores (see Selected references, Rinker *et al.* 1993). It has been successfully used to monitor the efficacy of sanitation programs in commercial mushroom operations in Ontario. *Verticillium fungicola* grows 23 to 28 mm in 10 days on potato-dextrose agar at room temperature and is velvety white in appearance. Undersides of cultures are colorless to yellow. Some isolates grow into the medium and the surface morphology becomes crenate in about 10 days. Mycelium is septate, hyaline, branched and narrow (1 to 3 µm). Numerous phialides are borne in whorls on erect conidiophores, 8 to 10 by 1 to 3 µm, tapering to 0.5 to 1.0 µm at the apex where conidia develop. Conidia form acropetally, but push into a gelatinous matrix rather than being catenate. Conidial masses contain 6 to 20 ovoid to short cylindric to slightly crescent shaped, hyaline, non-sep- tate, smooth-walled conidia, 6.6 by 2.5 µm. Another type of conidium (large aleuriospore) may form in the occasional culture incubated for many weeks. No sclerotia are formed by this species.

Two varieties of *V. fungicola* have been described, but the legitimacy of this differentiation is conjectural. *Verticillium psalliotae* Treschow, another nonsclerotial *Verticillium* species associated with mushrooms, is isolated only from crops cased with topsoil rather than peat moss, when the soil is not adequately pasteurized. A red pigment forms in the medium and the conidia are crescent shaped, but other physical characteristics are very similar to *V. fungicola.*

Disease cycle The optimum temperature for disease development is about 20°C. At this temperature, dry bubble and split stem symptoms will develop in 10 to 14 days from infection. Thus, if bubbles are present at first break, the pathogen was likely introduced with the casing material or at anytime through pin-set initiation. Verticillium spot develops within 48 hours of inoculation. Contaminated casing material and dust are probably the most common sources of *Verticillium.* Contamination may occur through air-borne spores or by spores carried by insects, mites or farm workers. The spores of *Verticillium* are produced in sticky clusters, which enables them to attach to dust, flies, mites, debris, clothing, tools and workers. The sticky spores cannot be removed by washing hands with hot, soapy water. Distribution on the hands and clothing of workers can be the most significant means of transporting the pathogen within a crop or between crops. Watering the crop can distribute the spores through splash and runoff to lower shelves and the floor. Insects, especially phorid flies (see 26.31), and mites (*Tyrophagus* spp.) that feed on *Verticillium* spores can easily move the pathogen within and between crops. Disturbance of contaminated dust on floors increases the concentration of airborne spores and is thought to be the cause of primary outbreaks.

Management

Cultural practices — The strategy for controlling verticillium disease is principally one of good sanitation and hygiene. The basic principles are 1) start with clean facilities and growing media; 2) prevent infection of healthy crops; 3) prevent disease spread within and between crops; 4) keep disease levels as low as possible; and 5) pasteurize to eliminate the pathogen in diseased crops when picking stops.

Casing material should be stored in dust-free areas, and storage and mixing areas should be disinfested before casing preparation. Casing material should not be exposed to dust or flies during mixing. Workers involved in casing application should not have worked in infested rooms or be wearing contaminated clothing. All casing equipment should be sanitized before use.

Insects should be prevented from entering production rooms by physical and chemical means. Controlling fly populations during the cropping period significantly reduces disease spread (see dark-winged fungus gnat, 26.29). Spore filters should be installed on all supply and vent air ducts.

To minimize spread of the *Verticillium* fungus from old (fourth break) to new (first break) rooms, new rooms should be harvested first each day. Harvesters should avoid picking diseased mushrooms. If diseased mushrooms are to be removed, this should be done separately from the regular harvest and workers should wear gloves that are disinfested periodically.

If the disease is severe, the crop should be terminated before the fourth break. The use of a three-break crop can significantly reduce the incidence of dry bubble disease on commercial farms. Crops that have been terminated, either early or on schedule, should have the compost temperature raised to 70°C for 12 hours. Spent compost and debris should be removed from the farm premises. Walls, floors and tray or shelf surfaces should be washed thoroughly after compost is removed.

Chemical control — Registered fungicides are available in Canada; however, benzimidazole-resistant *Verticillium* strains have been reported. Dithiocarbamate products can only be applied as dusts to the casing. Formalin can be mixed into casing material before it is placed onto the spawn-run compost. Local infections can be controlled by covering with salt and/or by cups, or by spraying the infected bubble and surrounding area with formalin before removal.

Selected references

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.9 Wet bubble *Fig. 26.9*

Mycogone perniciosa (Magnus) Delacr.
(teleomorph *Hypomyces* sp.)

Although this disease has been found from time to time, it is not a serious problem on commercial mushrooms.

Symptoms The disease is best recognized by the large cauliflower-like distortion of the mushroom *(26.9).* This coral-like mass can measure up to 10 cm across. Under conditions of high humidity, amber to dark brown drops of liquid form on the fluffy white surface. Dry bubbles, caused by *Verticillium fungicola,* do not get as large as wet bubbles and do not turn brown. Under dry conditions, wet bubbles can desiccate and may resemble dry bubbles.

Causal agent Two different types of conidia routinely develop, a phialo- conidium and an aleurioconidium. The phialoconidia are hyaline and are borne on verticillately branched conidiophores. Early research with the phialoconidial stage of *Mycogone perniciosa* resulted in confusion because of its similarity in appearance to *Verticillium fungicola. Mycogone perniciosa* is easily

identified by the presence of aleurioconidia produced on short branches that develop at the base of conidiophores and, more routinely, are intercalary along nonspecialized, narrow (3 to 4 µm) hyphae. Aleurioconidia are borne on a thin-walled, bulbous basal cell, 10 to 14 by 9 to 12 µm. The numerous aleurioconidia, 18 to 20 by 14 by 17 µm, are light amber brown, multinucleate, thick-walled with some warts, and the cytoplasm is dense and granular. Optimum growth occurs at 23 to 25°C on most general-purpose media. There is no light requirement for sporulation and aleurioconidia develop in 7 to 10 days. Phialoconidia are associated with young (3- to 7-day) cultures. Mycelial growth is white. A transformation occurs from white to buff as the culture ages. Aleurioconidia are numerous, while phialoconidia are rarely seen in older cultures.

Disease cycle Contaminated casing material is the primary source of *Mycogone perniciosa*, which is a ubiquitous soil-borne fungus. In young infected mushrooms, the pathogen will take 10 to 14 days to form its distinctive mass. Diseased first-break mushrooms may indicate contaminated casing. Spores may survive on structural surfaces and in crop residue. Once established, the primary means of spread is through water splash and runoff to lower beds. Insects and mites are suspected to be carriers of the pathogen, but there is no firm evidence of this. Harvesters, tools and equipment can spread the pathogen. The spores are light and may be air-borne. Spores in dust on floors or in soil may be another source of contamination.

Management (see verticillium disease, 26.8.)

Selected references

Smith, F.E.V. 1924. Three diseases of cultivated mushrooms. *Trans. Br. Mycol. Soc.* 10:81-97.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.10 Other fungal diseases

Aphanocladium cap spot *Aphanocladium album* (G. Preuss) W. Gams
Gill mildew *Cephalosporium* spp.
Hormiactis cap spot *Hormiactis alba* G. Preuss
Shaggy stipe *Mortierella bainieri* Cost.

These less common fungal diseases occasionally have been reported to cause significant yield loss to commercial mushroom production.

Selected references

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(Original by D.L. Rinker and P.J. Wuest)

VIRAL DISEASES

▶ 26.11 Miscellaneous viral diseases *Fig. 26.11*

La France
Other viral diseases

To date, five or more types of virus particles have been reported to infect commercial mushrooms. Singly and in combination, they produce a variety of symptoms. "Virus disease" frequently occurred in the commercial mushroom industry during the 1960s before its etiology was understood. La France continues to be a serious threat. In many cases, viral diseases are so devastating that mushroom growers have to cease production temporarily in order to eradicate the problem.

Symptoms The symptoms of viral diseases vary from reduced yields to distorted mushrooms. During the spawn- run period, there is no visible indication of disease; however, once the casing has been applied, distinctive symptoms may be expressed. The mycelium may have difficulty growing into the casing in some areas or will grow and then die back, leaving patches with no mushrooms *(26.11).* Mushrooms that do form may 1) be normal, 2) have small caps on normal-sized stems, 3) have elongated stems that are slightly bent, 4) die rapidly followed by a bacterial soft rot, 5) open prematurely, 6) turn off-white, ashen, or tan in color, 7) pin later than normal and frequently below the surface, 8) turn brown rapidly when harvested, or 9) be loosely attached to the casing. In other cases, the crop may appear completely normal, the only effect being an unexplained drop in production.

Causal agents There is a strong correlation between the presence of a specific double-stranded RNA pattern and the symptoms of La France disease. This pattern consists of dsRNA with molecular weights of 2.50, 2.05, 1.95, 1.85, 1.70, 1.10, 0.89, 0.53, and 0.50 x 10^6. Molecular weights of the dsRNAs are estimated from their electrophoretic mobilities relative to the Bst E II restriction endonuclease fragments of lambda DNA. Healthy mushrooms, including the newer hybrid strains, contain another dsRNA, molecular weight 1.6 x 10^6, that is not associated with the presence of disease.

Mushroom viruses (MV) also have been classified or recognized according to the size and shape of the particles. According to Fletcher *et al.* (1989; see Additional references), these include MV1, spherical particles, diameter 25 nm; MV2, spherical

particles, diameter 29 nm; MV3, bacilliform particles, 50 by 19 nm; MV4, spherical particles, diameter 35 nm; MV5, spherical particles, diameter 50 nm.

Positive identification of mushroom viruses is available through comparative growth rates of mycelium on agar, direct electron microscopy (EM), immunosorbent electron microscopy (IEM or ISEM), polyacrylamide gel electrophoresis (PAGE), and enzyme-linked immunosorbent assay (ELISA).

Disease cycle Viral diseases are transmitted through hyphal fusion (anastomosis) of healthy and diseased mycelium; the latter may produce mushrooms that release virus- infected spores. There are no known vectors (insects, mites or nematodes) of mushroom viruses other than the indirect distribution of spores by insects or mites. Mushroom spores can easily become air-borne and thus carried about the room or farm. Wild mushrooms have not been shown to be hosts of the viruses that attack *Agaricus bisporus.*

The compost can be infested any time after peak heat. Once established in the crop, the virus can spread through the mycelium. One mushroom with an 8 cm cap can discharge approximately 1.3 billion spores. It has been reported that as few as 10 to 100 virus-infected spores over approximately 3 m^2 of compost surface will induce recognizable symptoms.

Virus-infected mushroom spores in a dry state and stored at room temperature are capable of transferring virus to healthy mycelium after six years. Mushroom spores stored at 4°C are still viable after 10 years. Research has shown that mushroom spores survive 16 hours at 60°C but not at 65°C. Other reports suggest that 54°C for 10 minutes is lethal to mushroom spores.

Mycelial fragments greater than several cells remaining on woodwork, netting, or equipment can anastomose with healthy mycelium and transfer the virus particles.

Management

Cultural practices — The successful control of mushroom viral diseases can be accomplished through a strict hygiene program (see verticillium disease, 26.8). Since spores are a major vector of viruses, diseased mushrooms must not be allowed to open and release their spores. Filters must be fine enough to capture the 5 by 7 pm spores. Ventilation systems must be tight and not create a negative pressure, thereby sucking spores in beyond the filter.

The technique of ruffling the casing, which consists of breaking up the mycelium and relocating it within the casing layer, emphasizes the need for disinfesting equipment between each use. Also, the new technique of adding fully colonized compost to the casing material ("cacing") increases the risk of disease spread and highlights the need for a comprehensive program of preventive hygiene on mushroom farms.

Changing spawn strains can be helpful in restoring yield. Selecting a strain that does not anastomose as readily with the infected strain can help reduce the inoculum on the farm. Thus, common commercial practice has been to grow a strain of a different color or texture, such as cream or off-white. A related species, *A. bitorquis,* is reported to be tolerant to viral diseases. If hybrid strains of *A. bisporus* are used, switching from white to off-white hybrids or the reverse may not be effective because the hybrid strains are reported to anastomose with their parent lines.

Storage and shipment of spawn and mushrooms in the same cooler should be avoided. If equipment or picking/shipping containers are shared between farms, they should be disinfested before use.

Selected references

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(Original by D.L. Rinker and P.J. Wuest)

WEED MOLDS

There are several non-infectious fungal diseases of commercial mushrooms, all of which are usually referred to as indicator or weed molds. Production can be significantly affected by their presence, but the mushroom itself is not generally infected.

A. MOLDS CHIEFLY IN COMPOST

▶ 26.12 Ink caps *Fig. 26.12*

Coprinus comatus (Müller in Fl. Dan.:Fr.) S.F. Gray
Coprinus niveus (Pers.:Fr.) Fr.

Symptoms The mycelia of these *Coprinus* spp. are fine, gray to white, and not easily distinguished from mycelium of the mushroom fungus. Fruiting bodies of *Coprinus* spp. *(26.12)* generally develop after casing and before mushrooms are produced. Occasionally, they are observed at the end of phase II composting. The fruiting body quickly degenerates into a black, inky slime.

Causal agent *Coprinus* spp. are periodically seen in mushroom compost, first appearing during spawn-run or after the casing (peat moss) has been applied (top-dressed) to the compost after it has been colonized by *Agaricus bisporus.* These black-spored mushrooms usually have conical pilei and are most easily recognized when deliquescence transforms the mushrooms into a black liquid, which is the basis for the common name "ink caps." Morphological traits, besides deliquescence, that distinguish *Coprinus* spp. from other mushrooms include widely spaced narrow gills, pileus tissue quite thin, very thin lamellae with parallel sides, basidia separated by paraphyses that are shorter and broader than the basidia, and smooth basidiospores with a pore at the apex. At least two species, *Coprinus niveus* and C. *comatus*, have been observed on North American *Agaricus* mushroom farms.

Coprinus comatus originates within the compost and pushes its way to the surface, either before or after casing is applied. Its distinctive, musty, somewhat sweet odor permeates the growing room before it is seen, and the unique odor of the deliquesced mushrooms remains for a few days after deliquescence has occurred. The pileus is 2 to 8 cm high, the stipe is 8 to 20 cm long by 10 to 15 mm thick, and the elliptical spores are 13 to 16 by 7 to 8 µm. The caps are narrowly conical, expand to a bell shape, and are covered with scales that turn brown-red with age and become recurvate.

Coprinus niveus grows on the compost surface in small lumps of black compost, or on the surface of the casing at about the time *Agaricus* primordia are developing. Its primordia develop in 24 to 36 hours and mature after a similar amount of time. Only one bloom occurs, always before the mushroom harvest begins. After deliquescence, no trace remains of C. *niveus.* The caps are 10 to 30 mm high by 10 to 20 mm wide at the base and they are narrowly conical. The stipe is 3 to 6 cm high by 2 to 9 mm thick. Basidiospores are 15 to 17 by 8 to 11 µm and elliptical.

Ink caps bloom within a few days of each other, always before the mushrooms, and should be removed to eliminate the odor and the spore load and to prevent discoloring the mushrooms.

Disease cycle The presence of ink cap fungi indicates insufficiently converted nitrogen-containing compounds in the compost. This may result from an imbalance of the carbon: nitrogen ratio (C:N), overly composted material, too wet or too compacted compost at filling, too dry compost, or in an increase in the compost temperature of as little as 1 to 2°C during phase II composting. *Coprinus* spp. can readily use free ammonia and have an optimum pH for growth near 8. Greater than 700 ppm of ammonia at spawning can stimulate ink cap production. Once the free ammonia is released and the pH declines, mushroom mycelium will colonize the area. Spore masses released by mature ink caps can infect freshly prepared compost.

The presence of a few ink cap fungi has no effect on mushroom yield. It indicates that the compost contained a desirable nitrogen content, but that the ammonia had not completely dissipated before spawning.

Management

Cultural practices — Ink cap fungi can be controlled through proper composting. Attention to compost formula and the composting process is critical to achieving a well-balanced C:N ratio, optimum moisture level, and proper breakdown. Uniformity in the filling process and control over the environment during phase II composting will minimize ink cap infestation. The odor of ammonia should not be present in compost that is ready for spawning.

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.13 Olive-green mold *Fig. 26.13*

Chaetomium globosum Kunze:Fr.
Chaetomium olivaceum Cooke & Ellis

Symptoms The mycelium in the compost is grayish- white and fine. If plastic is not used on the compost surface after spawning, a fine aerial growth on the surface may be visible after 10 days; it has a distinctive moldy odor. Within 14 days of spawning, olive-green burs (perithecia) visible to the naked eye will appear on the straws *(26.13).* The mold will frequently occur in black areas of the compost that are uncolonized by the mushroom fungus.

Causal agent *Chaetomium globosum* is homothallic. Its perithecia are dark brown to black (olive-green on mushroom compost) and 225 to 350 µm. The asci are clavate and contain dark, olive-brown spores when mature. The ascospores are lemon-shaped, and flattened, with dimensions 8.5 to 11.5 by 7.0 to 8.5 by 6.5 to 7.5 µm. *Chaetomium olivaceum* has larger ascospores, 8.7 to 12.0 by 8.7 to 10.0 µm; it also is homothallic.

Disease cycle Spores of the olive-green mold fungus are quite common and can be found in straw, soil and spent compost. The ascospores can be carried along in air currents and on clothes and materials. They are heat tolerant and can survive 60°C for six hours. The development of olive-green mold is favored by a lack of oxygen (less than 16% oxygen) during phase II of the composting process. This may occur when the compost is too wet, too compacted at filling, overcomposted, or over-heated

during peak-heat (temperatures greater than 62°C with insufficient aeration). Olive-green mold is able to tolerate higher levels of ammonia than the mushroom mycelium. Thus, it can survive and thrive in conditions adverse for spawn growth.

Mushroom yield may be affected proportionally to the amount of compost that is affected, although different mushroom strains respond distinctively to olive-green mold.

Management

Cultural practices — Once the compost is infested, it is not possible to control olive-green mold. Reheating or re-spawning the compost is not effective. The best means to manage the problem is to avoid conditions that favor its establishment. Preparation of good compost during phase I, attention to filling trays, shelves or tunnels, and providing adequate aeration during phase II composting will prevent the occurrence of olive-green mold.

(Original by L. Rinker and P.J. Wuest)

▶ 26.14 Penicillium mold

Penicillium janczewskii Zaleski
(syn. *Penicillium nigricans* Bainier in Thom.)

Symptoms *Penicillium janczewskii* is another of the green molds that can occur in the compost and on the casing soil. Colonies are usually green but also can be blue- green, white, yellow or brown. *Penicillium* species are opportunistic fungi. They prefer simple carbohydrates but will grow on cellulose, fats and lignin. Poorly mixed or clumps of supplements, dead pins and cut stumps are common sites for the growth of this mold.

Causal agent *Penicillium janczewskii* is frequently identified from collections at mushroom farms. *Penicillium* species appear innocuous to crop health and mushroom quality, but no one has investigated either aspect in detail. The mycelium grows well on general fungal growth media. Colonies reach about 3.0 cm in diameter in 12 days at 25°C. The colonies are feltlike, light to dark olive-gray above and yellow to orange beneath. The conidia are globose, coarsely warted and measure 3.0 to 3.5 µm in diameter. When observed microscopically, the phialides have a tapering apex and conidia are brown pigmented. Several *Penicillium* spp. are associated with mushrooms, wooden boxes and shelves used to hold compost, and with nutrient supplements added to mushroom compost.

Disease cycle Compost can be infested by air-borne spores at spawning and colonization occurs thereafter. From time to time, *Penicillium* spores may contaminate the grain substrate used in spawn, but this is considered to be a secondary source of infestation.

Management

Cultural practices — Follow the same sanitation and hygiene methods used to manage green mold (*Trichoderma* spp.), La France and mat diseases.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.15 Other molds in compost

During composting, many mesophilic and thermophilic actinomycètes and fungi are part of the conversion process. The actinomycetes include species of *Streptomyces, Thermoactinomyces* and *Thermomonospora.* The fungi include *Humicola* (or *Thermomyces*) spp., *Stilbella thermophila,* and species of *Mucor, Thermoascus, Torula, Myriococcum, Malbranchea* and *Talaromyces.* The visible presence of these microorganisms at the time of spawning is a positive indication of the composting process.

(Original by D.L. Rinker and P.J. Wuest)

B. MOLDS ON COMPOST AND CASING

▶ 26.16 Black whisker mold *Fig. 26.16*

Doratomyces microsporus (Sacc.) F.J. Morton & G. Smith

Symptoms This mold can easily be recognized by gray- black, spore-bearing bristles (2 mm) on the surface of straws or casing material *(26.16).* Heavily infested compost will appear gray to black because of the high density of spores. When disturbed, the spores are released, resembling smoke. *Aspergillus, Penicillium* and *Chaetomium* mold species also may be present.

Causal agent *Doratomyces microsporus* conidiophores (annellophores) are aggregated into ereet, black synnemata, up to 600 pm, each having a feathery spore-bearing head. Each head consists of anastomosing hyphae that branch toward the outside and bear numerous conidiophores. Conidiophores are short, 4 to 9 by 3 to 4 pm, and produce long chains of conidia. Conidia are globose to ovoid, smooth-walled, and 3 to 5 by 2 to 3 pm.

Disease cycle Whisker mold is a cellulolytic fungus. The mold will develop when compost has been under-composted, where the carbon to nitrogen ratio (C:N) at spawning is above 18:1, or where the compost has overheated during the spawn-run period. Secondary infection by whisker mold in a crop is unlikely. Human allergic responses to the spores have been reported.

Management

Cultural practices — Attention to proper compost preparation during both phase I and phase II will prevent the occurrence of black whisker mold.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.17 Brown mold

Oedocephalum glomerulosum (BulLChev.) Sacc.
(teleomorph *lodophanus testaceus* (Moug.:Fr.) Korf)

Symptoms Brown mold is silvery gray initially, but as the spores mature the color changes to a dark tan, beige, or light brown. The growth on straws may be sparse to dense. It grows slowly through the casing, appearing near the time of pinning. The spores of this mold feel gritty compared to the smooth, flour-like feel of a plaster mold.

Causal agent *Oedocephalum glomerulosum* has determinate conidiophores that are erect, simple, hyaline, septate and enlarged at the apex into ampullae. Globose, non-septate conidia are produced synchronously on denticles over the surface of the terminal ampullae.

Disease cycle The spores are common in some composts. Development of this fungus is encouraged if ammonia and amines are not eliminated during phase II composting.

Management

Cultural practices — Techniques favoring the production of good compost will reduce the occurrence of this fungus.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.18 Lipstick mold *Fig. 26.18*

Sporendonema purpurascens (Bon.) Mason & Hughes (syn. *Geotrichum candidum* of authors, not Link)

Symptoms Lipstick mold appears on the compost during spawn-run or on the casing during production. This white fungus is not easily distinguished from mushroom mycelium. Cottony white balls, resembling mushroom pins, may develop on the straw or casing surface. As the spores mature, however, a distinctive pink to cherry-red color develops *(26.18).* In the Netherlands, where serious viral infections have occurred, this fungus has also been observed as a secondary mold. In Australia, it has been suspected as a vector of the La France pathogen.

Causal agent Some authors have regarded *Geotrichum candidum* and *Sporendonema purpurascens* as synonyms. However, according to van Greuning and Eicker (see Selected references), the two genera are distinct and the correct identification for lipstick mold is *S. purpurascens.* Lipstick mold can develop on both casing and compost, producing its distinctive red conidia (arthroconidia). Conidiophores are lacking. Vegetative hyphae are hyaline when young but turn brown with age. Conidia are produced by basipetal septation and fragmentation of vegetative hyphae. Small vestiges of cell walls remain on the four corners of the uniformly sized conidia. Germination of conidia has never been observed in culture. This fungus is very difficult to culture and grows very slowly. Growth is diffuse and an irregular, red-black pigment is formed in agar. Another type of conidium (aleuriospore) occasionally forms in cultures more than two months of age.

Disease cycle Lipstick mold is often associated with old, decomposed chicken manure in the compost formula and with wet compost. The fungus tends to spread slowly but it can colonize well-conditioned compost. Dissemination is through spores and any means that transmits them can spread this organism. Its appearance during spawn-run may result in lowered yields, but its occurrence during the harvest period does not affect yields.

Management

Cultural practices — Some authorities recommend raising the compost temperature to 65°C during peak heat for four hours to control lipstick mold. However, this practice can increase the chances of other molds developing. The best methods to reduce the risk of this fungus are to monitor the compost quality and to attend carefully to hygiene and phase II composting (pasteurization) temperatures.

Chemical control — Formalin can be used to spot-treat small areas of lipstick mold infestation.

Selected references

Van Greuning, M., and A. Eicker. 1991. The identity of the lipstick mold of cultivated mushrooms, *Agaricus bisporus. Bot. Bull. Acad. Sin.* 32:57-62.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.19 Plaster molds *Fig. 26.19*

Botryotrichum piluliferum Sacc. & March.
(teleomorph *Chaetomium piluliferum* J. Daniels)
Papulaspora byssina Hotson
Scopulariopsis brevicaulis (Sacc.) Bainier
Scopulariopsis fimicola (Cost. & Matr.) Vuill.
Trichothecium roseum (Pers.:Fr.) Link
(teleomorph *Hypomyces trichothecioides* Tubaki)

Symptoms White plaster mold, *Scopulariopsis fimicola*, may appear on the compost surface near the end of phase II composting as irregular patches of white, filamentous, aerial growth. After spawning, the aerial growth disappears and the white mold becomes appressed to the compost surface, appearing like flour or plaster of Paris. The mold may grow up through the casing material. Other fungi with similar gross morphology that may occur on or in mushroom compost are *Botryotrichum piluliferum* and *Trichothecium roseum.* There are color differences as these fungi mature. *Scopulariopsis fimicola* remains white, while *B. piluliforum* takes on a tan-buff appearance and *T. roseum* develops a rose-pink tint.

The brown plaster molds *Papulaspora byssina* and *Scopulariopsis brevicaulis* appear during the spawn-run period as 15- to 40-cm patches of a dense, plaster-like white mold. When mature, the center of the colony turns brown or orange-brown *(26.19).* The fungus grows through the casing and develops the characteristic brown center with white fringe. Both fungi grow well in compost with a pH of 8.0 or more.

Causal agents *Scopulariopsis fimicola* is the most commonly encountered white plaster mold in mushroom compost. Its mycelium is colorless, septate, sparsely branched, and 2 to 5 µm in diameter. Conidiophores occur in groups of four or five, separated from each other by long portions of sterile mycelium. They are 50 to 100 µm, cylindrical, taper to the apex, and have irregular, U-shaped, dichotomous branching. Conidiophores initially bear a terminal conidium (phialospore) 6.5 to 8.0 by 4.5 to 5.3 µm.

Conidia (aleuriospores) of *Botryotrichum piluliforum* are globose, 13 to 15 µm, and are produced on repeatedly racemosely branched, hyaline conidiophores.

Colonies of *Trichothecium roseum* are dusty and rose-colored. For a description of this fungus, see Greenhouse cucumber, leaf rot, 22.13.

Scopulariopsis brevicaulis colonies are initially white, but the center develops brown or golden coloration extending to a white edge. Conidiophores are short annellophores, 9 to 25 by 2.5 to 3.5 µm, with the base swollen to 5 µm. Non-septate conidia (amerospores), 5 to 8 by 5 to 7 µm, are globose to ovoid and frequently truncate at the point of attachment. They usually occur in long chains.

The genus *Papulaspora* is characterized by the presence of papulospores or "bulbils" which are sclerotium-like reproductive units consisting of irregular clusters of cells. The bulbils are pale brown to orange pigmented, 100 to 250 µm across, and arise from lateral branches.

Disease cycle These molds are associated with composts where there has been insufficient conversion of the nitrogen sources during phases I and II. They often appear to be associated with compost ingredients.

Management

Cultural practices — Modification of composting procedures for both phases to improve the quality of mushroom compost will significantly reduce the occurrence of the plaster molds.

(Original by D.L. Rinker and P.J. Wuest)

C. MOLDS CHIEFLY IN AND ON CASING

▶ 26.20 Cinnamon brown mold (peat mold) *Fig. 26.20*

Chromelosporium fulvum (Link:Fr.) McGinty, Korf & Hennebert in Hennebert & Korf
(syn. *Botrytis fulva* Link:Fr.)
(teleomorph *Peziza ostracoderma* Korf)

Symptoms Cinnamon brown mold grows mainly on the casing within the first two weeks. On occasion, it has been observed on the surface of the compost during spawn run. It is frequently seen on the surfaces of wooden shelves and trays. The fungus first appears as fine, white, aerial mycelium. The spores form in a few days, which changes the color to light yellow or golden brown. The thick, white, fluffy mycelial edges remain. It has been reported that a dense growth of this mold will retard first break and may cause a slight reduction in yield.

The fungus is opportunistic, not readily tolerating other organisms. It tends to grow on casing that has been overly pasteurized, where a strong formalin solution has been used, or where a virus has killed the mycelium in the casing. The mold will disappear by first break, and 10 to 14 days later the small, dark brown, disk or cup-shaped apothecia of the teleomorph state will appear *(26.20).*

Causal agent *Chromelosporium fulvum* has erect, septate conidiophores with an unbranched main axis bearing 7 to 12 sporogenous ampullae, the spore-bearing heads. Conidia develop simultaneously on denticles located on the surface of each ampulla. The globose conidia are lightly pigmented and tan-brown and cover each ampulla at maturity.

Disease cycle The mold is easily air-borne, which facilitates the contamination of casing material. Since the fungus is not a strong competitor with the mushroom mycelium, secondary infections in a crop are highly unlikely when the mushroom mycelium is healthy. Serious outbreaks on mushroom farms are usually indicative of poor hygiene or improper cultural practices. Cinnamon brown mold is favored by high humidity and high temperature after casing.

Management

Cultural practices — When pasteurizing casing material, careful monitoring of the temperature is necessary to reduce the possibility of over-heating the material. The casing should be heated to 70 to 75°C and maintained at this temperature for 30 min, with a thermometer placed in the coolest area of the pile. Careful attention to hygiene will reduce the risk of spread.

Chemical control — When treating casing material with formalin, no greater than a 2% solution of commercial formalin (37% formaldehyde) should be used.

Selected references

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(Original by D.L. Rinker and P.J. Wuest)

▶ 26.21 Nematode-trapping fungi

Arthrobotrys spp.

Symptoms When nematode populations are high, a superficial, sparse, white growth of the fungus is frequently observed on the casing surface. The affected area can be greater than 1 m in diameter. Some species of *Arthrobotrys* can produce brown colonies on the casing layer. This fungus traps and feeds on free-living, saprophytic nematodes *(Rhabditis* spp.).

Causal agent *Arthrobotrys* colonies are spreading, thin and hyaline or pink. Conidiophores are erect, arising from the substrate or from fasciculate aerial hyphae, and are simple or branched. They produce apical clusters of two-celled, hyaline conidia successively on broad denticles on sympodial branches. Conidial heads often become intercalary by renewed growth of the conidiophore.

Disease cycle Since the fungus needs nematodes to survive, it will appear only in association with heavy infestations and usually near the end of the mushroom crop.

Management

Cultural practices — Sanitation and other practices that deter the build-up of saprophytic nematodes in casing (see 26.28) will help to prevent the growth of *Arthrobotrys* spp.

(Original by D.L. Rinker and P.J. Wuest)

NON-INFECTIOUS DISORDERS

▶ 26.22 Hardcap (hardgill)

The primary cause of this disorder is believed to be the degeneration of spawn cultures. Occasionally, dramatic changes in the temperature of spawn cultures or compost have also been implicated.

Symptoms Affected mushrooms appear normal when viewed from above. From below, however, they are open and lack a veil. The gills are pink or frequently white. Occasionally, the gills are distorted, resembling those of a polypore. The cap is hard and brittle. The mycelium grows well through the compost and casing. First break is delayed and the time between breaks lengthened. The condition occurs throughout the crop. Mushroom production can be reduced to as little as 20% of a normal harvest.

Management

Cultural practices — Growers should follow practices recommended for the maintenance of spawn cultures and the growth of mushroom crops.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.23 Open veil

In some strains of *Agaricus* spp., watering too close to harvest can cause the mushrooms to open prematurely. This often occurs when the mushrooms have been under a water stress and a generous watering follows. Changes in temperature also can trigger opening of the veil, as can excessive carbon dioxide levels during cropping.

Symptoms The cap opens prematurely and the gills are fully developed and brown pigmented. On occasion, the cap is disproportionally smaller than the stem. Open veil sometimes can be a symptom of a viral disease.

Management

Cultural practices — Generally, open veil can be avoided by maintaining suitable growing conditions and by not putting the crop under stress.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.24 Rose comb

Rose comb is associated with hydrocarbons, phenols and other compounds contaminating the casing or contacting the mushroom surface. Diesel oil, exhaust from engines, and petroleum-based pesticides are thought to be the principal source of these chemicals.

Symptoms Pink, gill-like tissue develops on the surface of the mushroom cap. The mushrooms are grotesque and unsaleable.

Management

Cultural practices — Growers should avoid exposing mushroom crops to the harmful chemicals that have been associated with this disorder. To assess their possible toxicity, paints, caulking compounds and other products that are to be used in the growing rooms should be applied to a board and placed next to developing mushrooms. If no symptomatic mushrooms develop, the material is likely safe to use.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.25 Stroma

Stroma is related to the genetic characteristics of the mushroom strain. Some strains will characteristically produce more stroma than others. At other times, however, stroma may be accentuated through mishandling of the spawn in transit, storage or during preparation. Non-uniform casing moisture, especially wet areas, often is associated with the occurrence of stroma.

Symptoms The mycelium on the compost or casing surface aggregates into discrete, white patches, which later develop into a dense layer that can be peeled from the surface of the substrate. The formation of stroma occurs in advance of pinning.

Management

Cultural practices — Spawn should be carefully handled and stored to minimize the risk of this disorder.

(Original by D.L. Rinker and P.J. Wuest)

▶ 26.26 Other abnormalities

There are a number of other abiotic conditions that result in the formation of abnormal fruiting bodies, such as weepers, hollow cores, shaggy stipe, purple stem and saggy socks. Although these conditions are rare, they often concern growers. (For more information, see Additional references.)

(Original by D.L. Rinker and P.J. Wuest)

NEMATODE PESTS

Parasitic and saprophytic nematodes are rarely a problem in mushroom crops grown in modern facilities. Historically, the overall economic impact of nematode pests on Canadian mushroom production has been minimal; however, significant yield losses have been reported occasionally.

▶ 26.27 Parasitic nematodes

Aphelenchoides spp.
Ditylenchus spp.

Extensive sampling of commercial mushroom houses in Canada has not revealed the presence of *Aphelenchoides* and *Ditylenchus* species. For a detailed discussion of these pests, see Hussey *et al.* and Goodey, 26.28.

▶ 26.28 Saprophytic nematodes

Acrobeloides spp.
Caenorhabditis spp.
Choriorhabditis spp.
Rhabditis spp.

Saprophytic nematodes are common in mushroom houses, but there is inconsistency in the scientific literature concerning the correlation of their populations with yield reductions. Nevertheless, most commercial mushroom operations attempt to minimize the incidence of these nematodes.

Damage Black necrotic areas may be visible on the surface of spawned compost prior to casing. These spots may show some colonization by the mushroom fungus, but the mycelium will be fragmented and the compost appears wet. These areas will not be recolonized by mushroom mycelium. The surrounding, colonized compost degenerates and the nematodes subsequently migrate into the casing layer. With careful observation under bright light, the nematodes can be seen with the naked eye as they move or "flicker" on the compost straws.

Often, the casing is well colonized by the mycelium, but after ruffling or scratching it does not re-knit well in spots or in the whole shelf. Sometimes, however, the casing is colonized more slowly than normal after inoculation because of nematode activity. Similar effects have been observed with high populations of entomopathogenic nematodes. In either case, the mycelium is fragmented and the casing does not hold together well. Growers sometimes confuse the dieback symptoms of viral diseases with those caused by nematodes. As in compost, nematode flickering is the key diagnostic feature and may be observed on the casing surface with the aid of a bright light.

The whiteness of the mushrooms may also be reduced. The bacteria on which the saprophytic nematodes feed reproduce well in moist environments. Both the nematodes and their associated bacteria can reduce the quality of the fresh mushrooms.

Identification The majority of saprophytic nematodes in mushroom casing belong to the genera *Acrobeloides, Caenorhabditis, Choriorhabditis* and *Rhabditis.* They are all bacterial feeders and are characterized by having three or six lips fused or replaced by other structures. Their mouth openings (stoma) lack a stylet and their cuticle is annulated or smooth. Amphids are inconspicuous and the oesophagus has a terminal bulb. The tail of the male usually possesses a genital cavity (bursa) supported by rays. There are no caudal glands.

Life history Saprophytic nematodes are common inhabitants of compost and casing mixtures. Under optimum conditions, a 50- to 100-fold increase each week is possible. Under slow drying conditions, especially during the prepasteurization phase of compost preparation, some nematodes can form resistant stages that enable them to survive pasteurization. Insects, equipment, workers and irrigation of the casing can disperse the nematodes. In older mushroom houses with wooden ceilings, nematodes can reproduce in the wet insulation and drop onto the compost in condensation from the ceiling.

Management

Cultural practices — Good sanitation reduces the spread of nematodes in mushroom houses. Thorough, post-harvest pasteurization and cleaning of production rooms, netting and equipment will reduce carryover from one crop to another. Houses with wooden shelving require an especially thorough, post-harvest clean-up because nematodes may be located in the crevices of boards.

Maintenance of proper temperatures during pasteurization of compost is critical. If the surface of the compost becomes dry during the pre-pasteurization phase, resistant stages of the nematodes form and may survive the pasteurization process. Changing the length of the pre-pasteurization period, adjusting ventilation and controlling humidity are also necessary.

Insects are excellent vectors of nematodes. A good integrated pest management program is required to reduce the impact of flies on mushroom crops (see Insect pests, 26.29-26.31).

The casing material can be a source of nematodes. Although peat may be infested with nematodes, packaged peat is generally not a problem if it is handled properly. However, if the bags are broken and the peat becomes wet, the nematodes will multiply. Once opened, the peat should be mixed in a clean area with disinfested equipment and used within 24 hours.

Once nematodes are noticed on compost or casing, steps should be taken to reduce their spread on the farm and to determine the source of the infestation. Tools and equipment should be disinfested between shelves during the spawning operation. Ruffling or scratching the shelves should be avoided where nematodes are visible on the casing.

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(Original by D.L. Rinker and T.H.A. Olthof)

INSECT PESTS

▶ 26.29 Dark-winged fungus gnat *Fig. 26.29T1*

Lycoriella mail (Fitch)

The dark-winged fungus gnat, also known as the sciarid fly, big fly or mushroom fly, occurs across Canada wherever commercial mushroom crops are grown. It is the most important fly pest of commercial mushroom production. Of the three species of mushroom cultivated commercially in Canada, only the button mushroom is subject to severe attack by this gnat. Fly reproduction is continuous because mushrooms are cultivated year-round. In the winter, gnat populations are lowest. Although the threat is minimal in winter, gnats can move from one production room to another through corridors, lofts or outside the building, and they have been seen on the surface of snow 7 to 10 m away from a functional production room. During warmer months, the gnats will move from building to building, and to farms several kilometres distant.

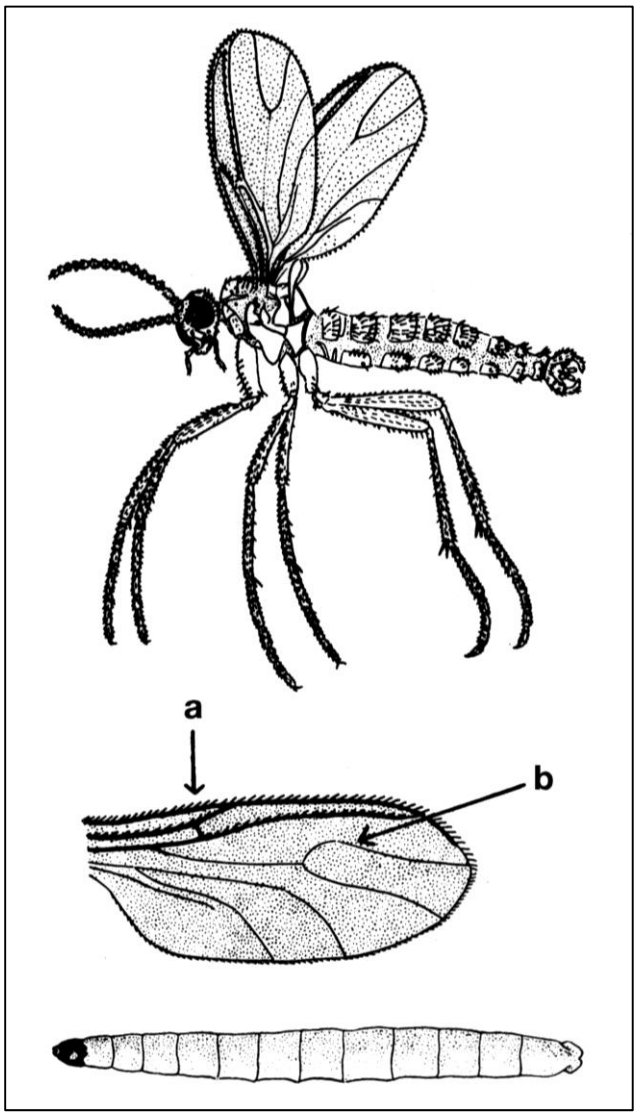
In general, the dark-winged fungus gnat can be found in greenhouses in both soil-free and soil mixtures, in composting debris such as leaves, and outdoors on wild mushrooms. The commercial button mushroom appears to be its preferred host, but it also breeds on oyster and shiitake mushrooms.

Damage The dark-winged fungus gnat can be found on practically any mushroom farm, but direct yield losses occur only when the gnats go unchecked. Larvae of this fly are general feeders, consuming mushroom compost, mycelia, spawn grains, mushroom primordia, and mushroom sporophores. When mushroom primordia are small, up to a cap diameter of about 1.5 cm, the larvae eat the internal contents. The mushrooms will appear glossy and light brown, but the pileus may be completely perforated and, when picked, the tissues crumble. Mushrooms that are larger when attacked show black necrotic areas in the stipe where the larvae made feeding galleries. Some larvae do not tunnel into the stipe but consume the mycelium at the base of the stem, in which case the mushroom does not develop normally. Little direct damage from this gnat will be evident on first-break mushrooms because they are harvested before larval development is extensive. However, second-break mushrooms may show damage from larval feeding. If fly populations are sufficiently high, then larval damage will be visible on the third and subsequent breaks.

The primary disease agent carried by this gnat is that of verticillium disease, which often increases between the second and third breaks. The presence of adult flies during the third break facilitates spread of the disease inside the room. In most cases though, the crop is terminated before a new generation of flies emerges.

This gnat often appears to be an indicator of poor management; as a pest, it is usually only of secondary or minor importance. However, on farms where the gnat is not controlled, individual crop loss can be as much as 50%. Perhaps its greatest impact is as a vector of mushroom pathogens from diseased to clean areas in the same production room or to clean crops in adjacent production rooms.

Identification Eggs of this fly (family Sciaridae) measure 0.25 by 0.15 mm and are smooth, oval, white and translucent. Larvae are legless, about 7 mm long at maturity, and have a white, translucent body and a black head. Pupae (puparia) are 2.0 to 2.5 mm long; they are white at first but turn black prior to hatching. Adult males and females measure 2 to 3 mm in length; most often they can be found near a light source. The wing has a forked vein and a crossvein (*26.29T1*; compare with phorid fly, *26.31T1).*



26.29T1 Dark-winged fungus gnat; adult, 2-3 mm long (top); note crossvein (a) and forked vein (b) in wing (center) and black head capsule of larva (bottom). Adapted from Snetsinger (1972).

Life history This fly usually invades production rooms at or near the time of spawning. After invasion, adults may oviposit on mushrooms, on compost, or in the casing soil, laying eggs singly or in small groups. The larvae have four instars and may feed on the spawn grains, mushroom compost, mycelium and mushrooms. There may be two complete generations during each cropping cycle on commercial mushroom farms; progeny of the second generation tend to be most damaging to the crop. Farms that harvest five flushes commonly have a third generation of fungus gnats.

The optimum temperature for development and maximum survival is 18.3°C. At higher or lower temperatures, mortality increases and female fecundity decreases.

Within four hours of emergence, female gnats are sexually receptive. Egg laying begins soon afterward. Female flies are strongly attracted to odors emanating from the mushroom house, particularly as the compost cools after the pasteurization and conditioning phases. Peak fly-invasion generally occurs within four days of spawning. Flies always seek the nearest site for oviposition; in commercial mushroom operations, this is usually nearest to doors of the production facility. Larvae developing in the surface layer of compost, which is later to be cased, will move through and pupate at or near the surface of the casing layer.

Management The movement of these flies from one production room to another or from one mushroom farm to another is accomplished mainly by unassisted adult dispersal and the persistence of the insects, which will crawl through any crack or crevice into the mushroom-production facility. Under conditions of poor sanitation and hygiene, these flies can enter the room on equipment that was not cleaned from previous spawning or casing procedures. Consequently, by leaving spawn overnight in a fly-infested corridor, an infestation can be introduced into the production room through the spawn. Growers who obtain

mushrooms from other growers run the risk of introducing fungus gnats. The dark-winged fungus gnat is the major insect pest, so management strategies have been developed particularly with this insect in mind.

Monitoring — When an invasion occurs, the size of the initial population and predicting its future size are important considerations. Monitoring gnat populations provides this information, but such factors as climate, disease, immature and adult insect populations, and growing practices are also important. The adult stage of the dark-winged fungus gnat is the main concern when monitoring on a commercial mushroom farm. Monitoring for adults is done by means of a fluorescent or "black" light as an attractant and a sticky surface or pan of water as a trap for the flies. Generally, the wharf end of a room at the upper level has considerably more flies than the breezeway end. In cool weather, the breezeway may serve as a bridge for fly movement between rooms. On standard mushroom farms, the trap should be placed on the wharf wall at the upper level. In the conventional shelf or tray system, the monitor should be placed in the room prior to the compost cool-down period. Monitors positioned outside, where flies normally roost, and in the breezeway, provide an indication of the background or endemic population level and measure the effectiveness of an outside or breezeway control program. Fly catches should be recorded daily to evaluate present control programs and to design future strategies. Threshold levels at different stages of a crop vary. For this reason, growers are encouraged to determine their own economic threshold levels. A suggested action level for the dark-winged fungus gnat is one to two flies each day from before spawning to four days after spawning when the growth room is cooler than 43°C; 15 flies per day for the remainder of the spawn- run; 30 to 40 flies per day after casing; and if a monitor light is used, one fungus gnat at spawning.

Cultural practices — Cropping for only three breaks is a viable, economical management practice because fly and disease problems are reduced. Rapid cool-down at the end of phase II reduces the time available for fly invasion. Higher temperatures occur in the compost during spawn- run and after casing, compared to the harvest period. Shortening the spawn- and case-run prolongs fly emergence in the cycle of the crop's phenology. In general, less damage will occur to a crop if the spawn-run is short.

Fly control at the end of a crop is just as important as control during spawn-run. Growers should treat mushroom houses by steaming-off, or by holding the compost at 60 to 65°C for four hours to kill flies at all stages of development; these conditions also kill most disease-causing fungi and bacteria. A crop may have to be terminated earlier than the schedule dictates to ensure that the population of emerging flies can be controlled prior to its spread to other locations.

Prevention is the most effective way to control fungus gnats. The problem is avoided if adults can be prevented from entering the growth rooms. Cracks in the walls, doors and around air conditioners and pipes are the usual routes of initial fly invasion. The installation of netting over doors, and limiting the amount of traffic into the room at critical times can help reduce the likelihood of infestation. Traps can be used to determine the tightness of a room and the need for doorway management. In general, if flies can be excluded until casing, they will have little or no impact.

Good sanitation is also important for fly control. Flies can breed in the butt and fragments of discarded mushrooms, and spent compost may serve as breeding material. Spent compost and mushroom stumpage should be removed from the premises. Growers also should remove and dispose of trash promptly.

The farm community must also be considered. Each mushroom room, block and farm probably has an endemic or background population of fungus gnats. These populations are specific for each farm and will vary from crop to crop and season to season. Pest populations throughout the farm community can only be brought under control if each grower understands the benefits of a consistent and total fly control program. Cooperation among growers also promotes better gnat management, based on sharing of knowledge about fly biology and behavior, and about the essential conditions that favor colonization and spread of pathogens.

Biological control — *Bacillus thuringiensis* var. *israelensis* and predatory nematodes have provided effective fungus gnat control in research trials. At present, the bacterium is not registered for use on mushrooms in Canada; the use of predatory nematodes does not require registration.

Chemical control — Studies in Pennsylvania and Delaware have demonstrated resistance within the darkwinged fungus gnat population to such insecticides as permethrin and dichlorvos, and there is some suggestion of resistance to the insect growth regulator diflubenzuron. Dichlorvos has been used in Canada for a number of years, and both diflubenzuron and permethrin are in the process of being registered for mushroom fly control in Canada. Many insecticides commonly used in the mushroom industry are metabolized by the enzyme system implicated in permethrin and dichlorvos resistance. Fungus gnats in Canada have not been examined for resistance, but the extensive use of insecticides means that resistance may eventually be present in Canada.

Premise sprays should include resting, swarming and roosting areas during the peak of the fly season. Growers also should treat walls, door jambs and the plastic cover that is placed over the compost after spawning. Adulticides in the form of aerosols or dusts should be applied when the action threshold is reached. For maximum effectiveness, larvicides must be applied when larvae are susceptible; this is especially true for formulations containing the insect growth regulator methoprene, which is a juvenile hormone mimic. For this chemical to be effective, the fourth-instar larva must ingest it; therefore, monitoring populations and maintaining records are essential for accurately timing its application. The use of chemical insecticides can be an important part of fungus gnat control on a farm, but growers should try to integrate this with other practices.

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(Original by D.L. Rinker)

▶ 26.30 Gall midges

Mycophila spp.

Gall midges (cecid flies) are minor pests of mushroom in Canada. The larvae apparently do not become pests on shiitake mushrooms cultivated on a sawdust medium.

Damage Midge larvae feed on the outside of the stipe or at the junction of the stipe and gills of both the commercial button mushroom and the oyster mushroom. Their presence can result in a loss of volume of fresh or processed, marketable product. They are also a factor in the spread of bacteria that induce browning.

Identification Gall midges (family Cecidomyiidae) are small, rarely seen flies, about 1.5 mm in length. However, when populations are high, their larvae are readily noticed because they wander off the beds and accumulate in heaps on the floor. Midge larvae are white or orange, depending on the species; mature larvae are about 2 mm in length.

Life history Gall midges in mushroom cultivation sometimes reproduce by the mature larva giving birth to 12 to 20 daughter larvae without becoming an adult fly and mating. Larvae usually feed for about 14 days, pupate and produce adults in 18 to 21 days. At the initiation of primordia, larvae must feed on mycelium in the casing and on the forming mushrooms. At maturity, the larvae construct pupation chambers in mushroom compost and enter a one-day prepupal stage. After pupation, adults emerge and become active.

Gall midge development is strongly influenced by temperature. During the spawn- and case-run production periods, first-generation flies can emerge within 18 days. During later stages of production, when the substrate temperature is allowed to drop to about 19 to 21°C, the developmental time per generation lengthens to about 21 days.

Management Gall midges are associated with infested casing material, especially peat, and they disperse on inadequately sterilized growing surfaces and especially on tools, equipment, and workers' shoes and clothing. Any practice that minimizes fly dispersal contributes to gall midge control.

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(Original by D.L. Rinker)

▶ 26.31 Phorid flies *Fig. 26.31T1*

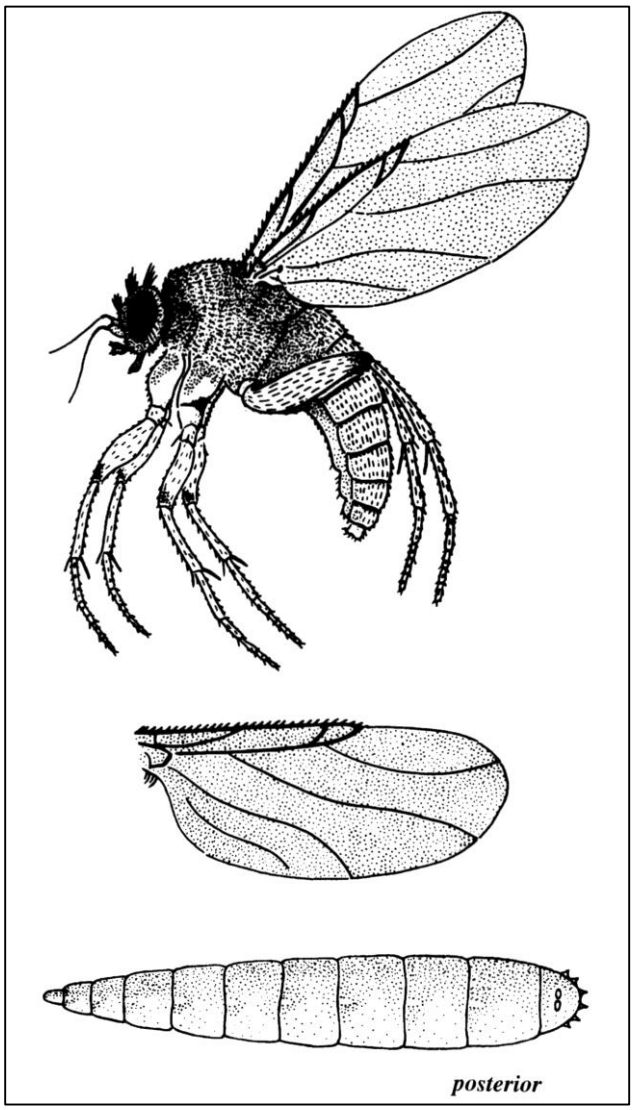
Megaselia halterata (Wood)

Phorid flies have only been observed on one mushroom farm in Ontario. However, they have been a significant problem in the United States and Europe.

Damage *Megaselia halterata* larvae feed at the growing hyphal tips of the mushroom mycelium. This species, unlike other *Megaselia* spp. that were pests in the 1940s in the United States, does not consume the sporophores. Thus, direct yield loss correlates with the number of larvae grazing on the mushroom mycelium. More than 12 000 females per m^2 of production surface are necessary before significant yield loss occurs; this is 12 times greater than the damage threshold for the dark-winged fungus gnat. Although direct yield loss can be a problem, the greater threat is the transmission of *Verticillium fungicola* (see verticillium disease, 26.8).

Identification The eggs of the phorid fly (family Phoridae) are about 0.2 by 0.5 mm in diameter and lack surface sculpture. Fertile eggs are translucent; infertile ones are cloudy and opaque. The legless larvae lack an apparent head and possess posterior respiratory structures (horns), thus differing from sciarid larvae, and the first-, second-, and third-instar larvae have mouthhooks (cephalo-pharyngeal skeletons) measuring 50, 84, 114 µm, respectively. The pupae (puparia) are approximately 2 mm in length. Young pupae are whitish with respiratory horns barely visible. Older pupae are yellow-brown with fully developed respiratory horns. The outline of the adult fly is visible through the puparium near the time of emergence. The adults of both sexes are small, measure 2 to 3 mm, lack forked veins and crossveins in the wings (*26.31T1;* compare with fungus gnat, *26.29T1)*, and are easily recognized by their "humpbacked" appearance, laterally flattened hind femora and quick, jerky movements.

Life history Adult females are attracted to actively growing mycelium and oviposit near the hyphal tips. In commercial operations, the mycelium is actively growing about four days after spawning and after casing. Unspawned compost does not support reproduction. Adult females mate in 24 to 48 hours after emerging (eclosion), and have a two- to three-day pre-ovipositional period before laying about 50 eggs. The average developmental time from egg to adult at 16 and 24°C is 51 and 37 days, respectively, with adults surviving four to eight days.



26.31T1 Phorid fly; adult, 2-3 mm long (top); wings lack cross- and forked veins; note posterior respiratory horns and lack of head capsule on larva (bottom). Adapted from Snetsinger (1972).

Newly emerged and older adult phorid flies readily fly to a suddenly exposed light, especially the shorter wavelengths of black light, black-light blue or cool white. Outside, flight activity is restricted to the daylight hours.

Management The integrated pest management strategies for control of sciarid flies, 26.29, also are effective in managing phorid flies.

Monitoring — Shorter wavelength lights than those used for sciarids are more effective for phorid flies. The action threshold can be at least five times higher than for sciarid flies.

Cultural practices — Since the phorid is smaller than the sciarid fly, the size of screening must be smaller to prevent passage of the fly.

Biological control — The larvae, pupae and adults of *M. halterata* are frequently parasitized by the endoparasitic nematode *Howardula husseyi* Richardson, Hesling & Riding (Tylenchida: Allantonematidae). Parasitism by this nematode does not obviously change the external appearance of the fly or appreciably affect the length of its life cycle; the most significant effect is a marked reduction in fecundity. Laboratory fly populations can be virtually annihilated within five generations by this parasite. Parasitic nematode populations can be favored by preventing compost temperature from exceeding 27°C.

Chemical control — The juvenile hormone mimics and insect growth regulators do not control *M. halterata* populations very well.

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(Original By D.L. Rinker)

MITE PESTS

▶ 26.32 Red pepper mites

Pygmephorus spp.

Red pepper mites, also known as pyemotid or pigmy mites, actually feed on molds (*Trichoderma, Monilia,* and *Humicola* spp.). They seem to be found only in production of the commercial button mushroom.

Damage Red pepper mites do not cause direct damage to cultivated mushrooms, but their presence often contributes to a loss in marketable yield. Additionally, they are a nuisance to mushroom harvesters.

Identification These mites (family Pyemotidae) are tiny, 0.25 mm long, and brown. Because they tend to congregate on top of the mushroom caps, they can be seen by shining a light across the pre-harvest mushrooms.

Life history The mites have a sexual, adult stage and a generation time of four to five days. Adult females may lay up to 160 eggs over a five-day period.

Management These mites disperse on inadequately sterilized production surfaces, on workers' clothing and by clinging (phoresy) on fungus gnat flies. Proper preparation and pasteurization of compost to minimize weed molds will reduce or eliminate red pepper mite populations.

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(Original by D.L. Rinker)

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Figures *27.1* to *27.2; 27.2T1*
Bacterial diseases
27.1 Bacterial soft rot of alfalfa sprouts
27.2 Bacterial soft rot of bean sprouts, physiological collapse

BACTERIAL DISEASES

27.1 Bacterial soft rot of alfalfa sprouts *Figs. 27.1a,b*

Erwinia chrysanthemi Burkholder, McFadden & Dimock

Soft rot usually occurs in seed germination trays. It can be particularly severe when temperatures exceed 28°C. The pathogen is capable of attacking a variety of horticultural plants.

Symptoms The first symptom is a yellowish, translucent appearance of the alfalfa root *(27.1b),* which soon stops growing. A smelly rot develops, which can spread rapidly and destroy whole trays of seedlings *(27.1a).*

Causal agent *Erwinia chrysanthemi* is a Gram-negative, non-spore-forming rod, 0.5 to 0.7 by 1 to 2.5 µm, with a physiology characteristic of the genus, except that it shows some growth in 5% sodium chloride. It liquefies gelatin and rots potato tuber tissue. On King's B medium, colonies are light-colored, translucent, round, and slightly umbonate with undulate margins and a strong odor of banana. On Miller-Schroth medium, colonies have a fried-egg appearance, being orange in the center and lighter toward the margin. The colonies and surrounding medium turn orange within one to three days, then green after four days.

Erwinia carotovora subsp. *carotovora,* another species causing soft rot of vegetables, apparently has not been reported on alfalfa sprouts, but it too may cause bacterial soft rot.

Erwinia chrysanthemi can be distinguished from *E. carotovora* subsp. *carotovora* by the fried-egg-type colonies on potato dextrose agar and Miller-Schroth medium, by the blue pigment when produced, by its failure to produce acid from lactose, maltose, trehalose or p-methyl glucoside in seven days, failure to grow in 5% sodium chloride, positive indole, lecithinase and phosphatase, and sensitivity to erythromycin.

Disease cycle The bacterium is probably introduced in water, because it does not survive on dry seeds longer than two weeks. The disease is very contagious and develops rapidly between 16 and 34°C.

Management

Cultural practices — Scrupulous hygiene is important to eliminate or reduce inoculum in water and air-borne dust. Temperatures of 21°C or less, although slowing germination, can reduce disease progression.

Chemical control — Before germination, seeds may be soaked in 0.5% calcium hypochlorite solution for two hours to disinfest them. Sodium hypochlorite and hydrogen peroxide solutions are less effective. As with bacterial soft rot of mung bean sprouts (see bacterial soft rot of bean sprouts, this chapter), calcium chloride or calcium nitrate added to the germination water at about 0.005 molar (0.5 to 0.6 g/L) is an effective control measure.

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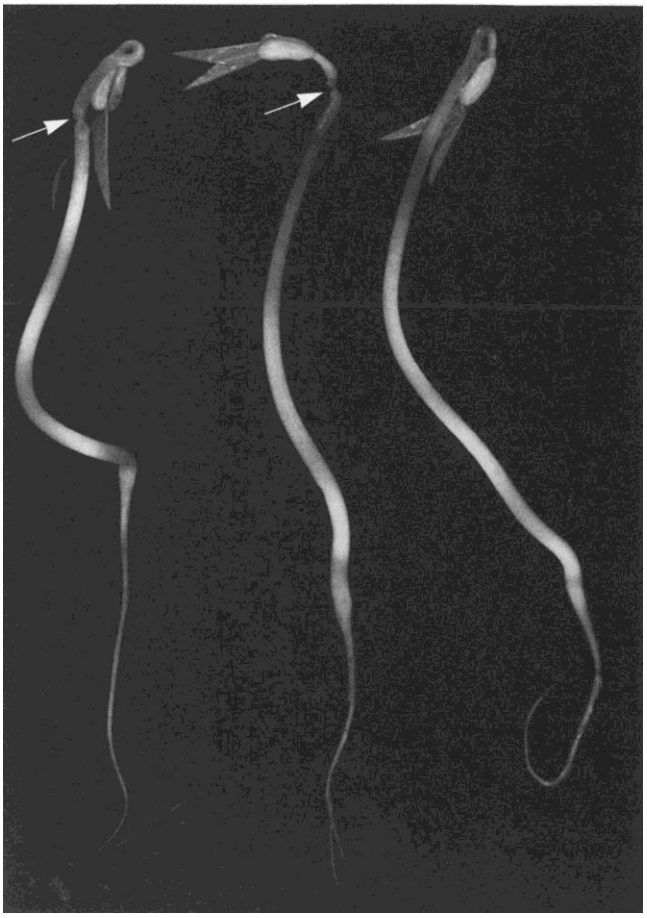
(Original by W.R. Jarvis)



27.2T1 Bacterial soft rot of bean sprouts; water-soaked constriction (arrow) of the hypocotyl at the crook just below cotyledons is associated with calcium deficiency; the tissue becomes necrotic and is invaded by soft-rot bacteria.

▶ 27.2 Bacterial soft rot of bean sprouts, physiological collapse *Figs. 27.2; 27.2T1*

Erwinia carotovora subsp. *carotovora* (Jones) Bergey *et al.*
Erwinia chrysanthemi Burkholder, McFadden & Dimock

Physiological collapse can occur when there is inadequate calcium in the water used to germinate seeds. Affected seedlings are susceptible to bacterial soft rot. The bacteria that cause soft rot usually belong to the genus *Erwinia*, either *E. carotovora* subsp. *carotovora* or *E. chrysanthemi*. Both species are ubiquitous and may readily contaminate unhygienic sprout production systems.

Symptoms A water-soaked lesion appears just below the cotyledonary hook (27.2) and the hypocotyl becomes constricted (*27.2T1*). Bacteria invade the damaged tissue and cause a smelly soft rot that very quickly spreads through the bean sprouts.

Causal agent The initial damage is usually from a lack of calcium in the cell walls of the bean sprouts. Calcium links parallel pectin chains, which provide structural strength in the cell wall. It also renders pectin more resistant to microbial degradation. (For descriptions of *Erwinia chrysanthemi* and *E. carotovora* subsp. *carotovora*, see bacterial soft rot of alfalfa sprouts, 27.1, and Potato, bacterial soft rot, 16.2.)

Disease cycle (see bacterial soft rot of alfalfa sprouts, 27.1; and Potato, bacterial soft rot, 16.2)

Management

Cultural practices — Calcium can be added to the germination water as calcium chloride or calcium nitrate, creating about a 0.005 molar (0.5 to 0.6 g/L) solution. Because the invading bacteria may be waterborne, care should be taken to avoid contamination of the water supply by soil or plant residue. Creek and well water should never be used without prior filtration and sterilization. Growers should maintain hygienic conditions in the production system. An essential part of this strategy is the surface sterilization of seed in 0.5% calcium hypochlorite solution for two hours immediately before germination.

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(Original by W.R. Jarvis)

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Appendix Plant diagnostic laboratories in Canada

Plant Diagnostic Laboratory
British Columbia Ministry of Agriculture, Fisheries and Food
17720 - 57th Avenue
Surrey, British Columbia
V3S 4P9
Tel: 604-576-5600
Fax: 604-576-5652

Brooks Diagnostics Limited
Box 1701
Brooks, Alberta
T1R 1C5
Tel: 403-362-5555
Fax: 403-362-5556

Plant Diagnostic Laboratory
Alberta Environment
Alberta Environmental Centre
Bag 4000
Vegreville, Alberta
T9C 1T4
Tel: 403-632-8211
Fax: 403-632-8379

Crop Protection Laboratory
Saskatchewan Agriculture and Food
3211 Albert Street
Regina, Saskatchewan
S4S 5W6
Tel: 306-787-8130
Fax: 306-787-0428

Crop Diagnostic Centre
Manitoba Agriculture
Agricultural Services Complex
201 - 545 University Crescent
Winnipeg, Manitoba
R3T 5S6
Tel: 204-945-7707
Fax: 204-945-4327

Pest Diagnostic and Advisory Clinic
Ontario Ministry of Agriculture and Food
Agriculture and Food Laboratory Service Centre
Box 3650, 95 Stone Road West, Zone 2
Guelph, Ontario
N1H 8J7
Tel: 519-767-6256
Fax: 519-676-6240

Laboratoire de diagnostic en protection des cultures
Le Service de Phytotechnie de Québec
Ministère de l'Agriculture, des Pêcheries et de l'Alimentation
2700, rue Einstein, D. 1.110
Sainte-Foy (Québec)
G1P 3W8
Tel: 418-643-5027
Fax:418-646-0832

Plant Diagnostic Laboratory
New Brunswick Department of Agriculture
Plant Industry Branch
Box 6000
Fredericton, New Brunswick
E3B 5H1
Tel: 506-453-2172
Fax: 506-453-7978

Plant Pathology Laboratory
Nova Scotia Department of Agriculture and Marketing
Plant Industry Branch
Kentville Agricultural Centre
Kentville, Nova Scotia
B4N 1J5
Tel: 902-679-6040
Fax: 902-679-6062

Department of Biology
Nova Scotia Agricultural College
Box 550
Truro, Nova Scotia
B2N 5E3
Tel: 902-893-6600
Fax: 902-895-4547

Provincial Plant Health Laboratory
Prince Edward Island Department of Agriculture
Box 1600
Charlottetown, Prince Edward Island
C1A 7N3
Tel: 902-368-5487
Fax: 902-368-5629

Potato Services Division
Prince Edward Island Department of Agriculture
Box 306
Kensington, Prince Edward Island
C0B 1M0
Tel: 902-836-5450
Fax: 902-836-3161

Newfoundland Department of Forestry and Agriculture
Provincial Agricultural Building
Box 8700
St. John's, Newfoundland
A1B 4J6
Tel: 709-729-0022
Fax: 709-729-6046

Glossary

Abbreviations for units of measure

°C degree Celsius (°C = (°F - 32) X 5/9)

cm centimetre (1 cm = 0.01 m; 2.54 cm =1 inch)

g gram (1 g = 0.001 kg = 0.035 ounce; 454 g = 1 lb)

ha hectare (1 ha = 10 000 m^2 = 2.47 acres)

h hour

kg kilogram (1 kg = 1000 g = 2.2 lb)

km kilometre (1 km = 1000 m = 0.62 mile)

kPa kilopascal (1 kPa = 0.145 psi; 1 psi = 6.89 kPa)

L litre (1 L = 1000 mL = 0.88quarts)

m metre (1 m =100 cm = 39.4inches)

min minute

mL millilitre (1 mL = 0.001 L)

mm millimetre (1 mm = 0.001 m)

pm micrometre (1 pm = 10^{-6}m)

mS millisiemens (unit of electrical conductivity; 1 mS = 1 mmho)

nm nanometre (1nm = 10^{-9} m)

ppm parts per million

t tonne (1 t = 1000 kg = 2205 lb)

A

abaxial the leaf surface facing away from the axis of a stem; the lower surface of a leaf.

abdomen the posterior division of an insect's body.

acaricide a chemical or other agent used to control mites.

acervulus (-i) a subepidermal, saucer-shaped fruiting body of a fungus that produces conidia on a hymenium of conidiogenous cells lining the cavity and which also may produce setae, e.g. in some *Colletotrichum* species.

acid-fast bacteria bacteria, especially mycobacteria, that stain with basic dyes and fluorochromes, and resist decolorization by acid solutions.

acidic having a pH of less than 7.0.

acropetal development of organs or cells in succession towards the apex, with the oldest at the base and youngest at the apex.

actinomycete bacteria that form branching filaments.

active ingredient the portion of a formulated pesticide that is the actual toxicant; abbreviated **a.i.**

adaxial the leaf surface facing toward the axis of a stem; the upper surface of a leaf.

adult a mature stage in animals and plants; non-molting in most arthropods.

adventitious arising in an abnormal position, e.g. roots developing from a plant part other than a root, such as a stem or leaf cutting.

aeciospore a dikaryotic, unicellular fungal spore of the rust fungi (Uredinales) produced in an aecium after fertilization by hyphal fusion or the union of uninucleate pycniospores.

aecium (-ia) an asexual fruiting body of the rust fungi containing dikaryotic conidia (aeciospores); usually cup-like.

aerobic pertains to the need for oxygen, as in a physiological process or the capacity of an organism to live in the presence of oxygen.

agar a gel-like carbohydrate derived from certain red algae; used to solidify culture media on which some microorganisms can be grown; a term also applied to the medium itself.

aleurioconidium (syn. aleuriospore) a thick-walled, pigmented, or sometimes thin-walled and hyaline conidium developed from the blown-out end of a conidiogenous cell or hyphal branch from which it separates with difficulty.

alkaline having a pH above 7.0; basic.

allantoid slightly curved with rounded ends; sausage-like in form; especially in reference to fungal spores.

allele one of two or more alternative forms of a gene at a given site on a chromosome.

allelopathy the harmful influence on a plant by another living plant that secretes a toxic substance.

alluvial pertaining to minerals or soils deposited by running water.

alternate host a plant species required to complete a parasite's life cycle, e.g. macrocyclic rusts.

alternative host a plant species that serves as a host for a parasite, but which is not specifically required for the parasite to complete its life cycle; see **alternate host.**

ameroconidium (syn. amerospore) one-celled (non-septate) conidium with a length:width ratio of less than 15:1.

amphigynous fungi (Pythiaceae) having an antheridium throughout which the oogonial intercept grows.

ampulla (-ae) the swollen tip of a conidiophore that is either conidiogenous or develops a number of short branches or discrete conidiogenous cells.

ampulliform flask-like in shape.

anaerobic pertains to the lack of a need for oxygen, as in a physiological process, or the capacity of an organism to live where there is no oxygen.

anal pertains to the region of the anus in animals and to the last abdominal segment in insects.

anamorph that part of the life cycle of a fungus characterized by the production of asexual spores borne on conidiomata.

anastomosis group a grouping of similar strains of a fungal species in which all members can anastomose (fuse) with each other.

anastomosis (-es) the fusion between branches of the same or different hyphae or other structures to make a bridge or network; leads to the combination of hyphal contents.

anhydrobiote a nematode that is able to enter a coiled dehydrated state and survive in moderately dry substrates for several months.

annelloconidium (-ia) (syn. annellospore) a conidium formed from an annellophore.

annellophore a conidiophore or conidiogenous cell that proliferates per-currently and bears a succession of ring-like scars (annellations) of previous conidial succession around the apex; a conidiophore that produces annelloconidia in basipetal sequence.

annual a plant that completes its life cycle within one year.

antheridium (-ia) the male sex organ or sex cell (gametangium).

anthraquinone a quinone comprised of three linked benzene rings; produced by the oxidation of anthracene.

antibiotic a synthetic chemical or microbial product that is toxic to microorganisms, killing them or inhibiting their growth.

antibody a specific protein formed in the blood of warm-blooded animals in response to the injection of an antigen.

antigen any foreign chemical, normally a protein, that induces antibody formation in animals.

antiserum (-a) blood serum containing antibodies.

anus the opening of the alimentary tract in animals through which excrement is voided.

apical pertains to the end, tip or outermost part.

apical dominance inhibition of lateral bud growth by the apical bud of a shoot; a response to auxins produced by the apical bud.

aplerotic oospores not filling the oogonium; pertains to Pythiaceae.

apothecium (-ia) a type of ascocarp; cup-shaped and becoming open at maturity; with or without a stalk.

appressorium (-ia) the swollen tip of a hypha or germ tube that facilitates attachment to and penetration of the host by a fungus.

apterous wingless.

arthroconidium (-ia) (syn. arthrospore) a conidium formed by segmentation and fragmentation of vegetative hyphae, usually but not always in a chain.

arthropod animals with segmented appendages, including aquatic and terrestrial forms; in this publication, refers mainly to insects and mites.

artifact the remains, workings or damage left by an organism that may no longer be present.

ascocarp the sexual fungal fruiting body of an ascomycete; contains asci and ascospores.

ascospore a haploid fungal spore produced in an ascus.

ascus (-i) a component of an ascocarp; a sac-like cell; functions meiotically to produce ascospores.

asexual reproduction without fertilization.

asexual state in a fungus the part of the life cycle in which cells are produced only mitotically.

asymmetric flattened, concave or irregular on one side.

autoecious a parasitic fungus that can complete its entire life cycle on one host species, e.g. certain members of the rusts (Uredinales).

autotoxicity the capacity of a component to cause injury to the organism producing it.

avirulent a lack of pathogenicity by known pathogens, especially on particular plant cultivars.

axenic culture the growth of organisms of a single species in the absence of cells or living organisms of any other species.

axillary placed or growing in the axil of a branch or leaf.

axis the center line of an organism, organ or other plant part.

B

bactericide a chemical or other agent used to control bacteria.

bacteriocin bactericidal substances produced by certain strains of bacteria and active against some other strains of the same or closely related species.

bacterium (-ia) a one-celled microorganism without a true nucleus in the cell.

ballistospore a forceably ejected basidiospore.

bar a unit of pressure equal to 10^5 pascals.

basal pertains to the base or point of attachment.

basidiospore a haploid fungal spore produced on a basidium after meiosis.

basidium (-ia) a fungal structure that produces basidiospores; produced by basidiomycetes.

basipetal development of organs or cells in succession toward the base, with the oldest at the apex and youngest at the base.

biennial a plant that completes its life cycle in two years.

biflagellate possessing two flagella.

biguttulate spores or cells containing two oil-like drops inside.

binucleate containing two nuclei.

biocontrol agent see **biological control.**

biological control agent see **biological control.**

biological control the use of living organisms (biological, biotic or biocontrol agents) for the inhibition or destruction of a pest population.

biotic agent see **biological control.**

bipolar at both ends or poles of a bacterial cell or spore; also, sexual compatibility in some basidiomycetes in which two of the basidiospores on a basidium are of one strain and two are of another.

biseriate in two rows.

biverticillate having parts in two rings or whorls.

blastoconidium (syn. blastospore) a condium produced by the marked enlargement of a recognizable conidial initial before the initial is delimited by a septum.

blight sudden, severe and extensive spotting, discoloration, wilting, or destruction of leaves, flowers, stems, or entire plants, often affecting young, growing tissues; in disease names, may be coupled with the name of the affected part of the plant (e.g. leaf blight), the kind of causal organism (e.g. bacterial blight), or a distinctive symptom (e.g. halo blight).

blotch a symptom characterized by large, irregularly shaped spots on above-ground plant parts.

botryoblastoconidium (syn. botryoblastospore) one of a cluster of coni- dia borne on the swollen apex (ampulla) of a conidiogenous cell, either singly or in chains.

botryose clustered like grapes.

breeding line plant strain used in a breeding program and usually containing one or more desirable characteristics.

budding a method of vegetative propagation of plants by implantation of buds from the mother plant onto a rootstock.

bug the group name given to insects with piercing - sucking mouthparts and wings; a popular term for any insect.

butyrous resembling the texture or color of butter.

C

caducous spores falling off readily; deciduous.

calcareous containing lime.

calyx (-ces) outermost part of a flower, consisting usually of green, leaflike structures known as sepals, which in the bud stage enclose and protect the other flower parts.

canker a dead portion of a stem, root or fruit that is sunken or shrivelled; a sharply delimited necrosis.

canopy a mass of leaf-bearing shoots characterized by height, width or density; the formation of upper branches (shoots), providing a cover of foliage.

capitate having a well-formed head.

carbohydrate one of numerous chemical compounds comprised of carbon, hydrogen and oxygen, e.g. sugars, starches and cellulose.

cardinal growth temperatures the minimum, maximum and optimum temperatures at which an organism can grow.

carlavirus a group of viruses of which the type member is carnation latent virus.

casing a layer of material (usually peat moss or soil) used to cover spawned compost in edible mushroom production; mushrooms are produced on the casing.

catenate (syn. catenulate) in chains or end-to-end series.

caterpillar a popular term for the larva of an insect, usually reserved for larvae of moths and butterflies.

cauda the posterior region of the abdomen in aphids.

caulicolous living on herbaceous stems.

causal agent an organism or agent that produces a given disease.

cellulolytic having the ability to decompose cellulose, e.g. certain species of bacteria and fungi.

chlamydospore a fungal resting spore; thick walled, asexual and frequently intercalary.

chlorophyll green pigment of plants that absorbs light energy during photosynthesis.

chlorosis the yellowing of normally green tissues.

chromosome string- or bead-like structure(s) in the nucleus that contains genes.

chrysalis (-ises, -ides) a butterfly pupa that is not enclosed in a case of silk or other matter.

cladophyll a branch arising from the axil of a true leaf and resembling a foliage leaf.

clamp connection a hyphal outgrowth in some basidiomycete fungi which, at cell division, makes a connection between the two resulting cells by fusion with the lower cell; buckle; nodose septum; by-pass hypha.

clavate club-shaped.

cleistothecium (-ia) an ascocarp lacking a specialized opening or ostiole and containing asci and ascospores.

coalesce to run together, as in small lesions merging into larger blotches.

cocoon a covering made by an insect larva before pupation; composed wholly or partly of silk.

coenocytic a multinucleate mycelium, not divided by cell walls.

collarette a cup-shaped structure at the apex of a phialide.

collenchyma tissue providing mechanical support to young, actively growing, plant structures; consists of living cells with walls strengthened by cellulose thickening; commonly found in cortex of herbaceous stems.

colony growth of a microorganism in mass, especially as a pure culture in the laboratory.

columella a sterile central axis within a mature fruit body which may be uni- or multicellular, unbranched or branched; of fungal or host origin.

conidiogenesis the production of conidia.

conidioma (-ata) a specialized, multi-hyphal, conidia-bearing structure.

conidiophore a modified, fertile fungal hypha bearing conidiogenous cells from which conidia are produced.

conidium (-ia) an asexual, non-motile fungal spore borne on a conidiogenous cell, usually decidious; not formed by cleavage or free cell formation.

cornicle a tube-like, abdominal structure in aphids; dorsal and bilaterally paired.

cortex a more or less thick outer covering in plants; parenchyma tissue surrounding the vascular cylinder in stems and roots, and bounded on the outside by the epidermis.

cortical pertaining to the cortex.

cotyledon (-s) the first leaf or pair of leaves in a germinating seed; frequently remain below ground.

crenate having the edge with rounded teeth.

cultivar an artifically bred, cultivated plant variety, including hybrids, inbreds, open-pollinated and a sexually propagated lines; abbreviated **cv.**

culture a colony of organisms on a medium; also refers to the growing of an organism on a medium.

culture medium see **medium.**

cupulate cup-like in form.

cuticle the water-repellent, waxy covering (cutin) of epidermal cells of plant parts, such as leaves, stems and fruits; the outer sheath or membrane of arthropods and nematodes.

cyst a nematode artifact, consisting of the dead remains of the female with or without viable eggs in some groups of nematodes; rounded and sac- like; also the resting spore of some fungi.

D

damping-off the collapse or death of a seedling; occurs suddenly before or after emergence.

defoliation loss of leaves from a plant.

degree-day a unit of accumulated temperature above a certain threshold over a period of days; abbreviated DD.

deliquescence becoming liquid after maturing.

dendroid tree-like in form.

denitrification the reduction of nitrate or nitrite to gaseous products such as nitrogen, nitrous oxide and nitric oxide; brought about by denitrifying bacteria.

denticle a small, tooth-like projection on which spores are borne.

desiccated dry.

diapause a genetically determined, physiological condition of arrested development in arthropods.

dichotomous dividing into two more or less equal branches.

diclinous having the oogonium and the antheridium that fuses with it on different hyphae.

dicotyledon a plant with two cotyledons.

dieback a progressive dying from the tip of shoots or roots.

dikaryon a cell with two genetically compatible haploid nuclei that divide synchronously; usually in fungi.

dikaryotic pertaining to a dikaryon.

dimorphic having two forms, e.g. fungi with yeast and mycelial forms.

diploid having paired chromosomes.

disease a dysfunctional, abnormal condition caused by one or more pathogens, toxic chemicals, nutritional deficiencies, environmental stress, and genetic abnormalities; often restricted to conditions caused by pathogens and accompanied by definite symptoms.

disease cycle the chain of events involved in the development of a disease; includes the stages of development of the pathogen and the effect of the disease on the host.

disinfectant a physical or chemical agent that is used to free a plant or plant part from infection.

disinfestant an agent that is used to kill or inactivate a pathogen in the environment or on the surface of a plant or plant part before infection occurs.

disorder a dysfunctional condition caused by factors other than pathogens, such as toxic chemicals, nutritional deficiencies, environmental stress during growth or in storage, and genetic abnormalities; usually accompanied by visible symptoms.

dissemination spread of infectious material (inoculum), often from diseased to healthy plants.

dissociation the appearance of a novel colony type on solid media after one or more subcultures of the microorganism in liquid media.

distal pertains to an area furthest from the body or point of attachment.

diurnal related to the 24-hour cycle of day and night.

DNA deoxyribosenucleic acid; an organic compound formed from nucleotides, each of which contains the sugar deoxyribose, a low-energy phosphate group, and usually one of four different bases: adenine, thymine, cytosine or guanine; DNA forms the basic material in the chromosomes of the nucleus; it contains the genetic code and transmits hereditary patterns.

doliiform barrel-like in form.

dolipore septum (-a) a septum of a dikaryotic basidiomycete hypha which flares out in the middle portion, forming a barrel-shaped structure with open ends.

dorsal pertains to the top, back or upperside.

E

echinulate having small pointed processes or spines.

economic injury level the lowest population density of a pest that will cause damage equal in value to the cost of control measures.

economic threshold the population density of a pest at which control measures should be applied to prevent it from reaching the economic injury level.

ectoparasite a parasite that lives outside the body of its host.

edema (syn. oedema) a swelling of tissue, mainly through increases of intercellular fluid content; results in a general over-development of plant cells.

egg an early developmental stage in animals.

eguttulate refers to cells or spores that lack oil-like drops inside.

ELISA enzyme-linked immunosorbent assay; a serological test in which the antibody-antigen reaction is detected by having the antibody attached to an enzyme which catalyzes the reaction of a substrate and causes a color change.

elytron (-a) the stiffened, thickened or leathery forewing of beetles.

enation a small swelling or gall.

encyst to become surrounded by a cyst or shell.

endemic being native or permanently established in a particular area; usually existing at low, stable population levels.

endoconidium (-ia) (syn. endospore) a conidium formed inside of a hypha, e.g. a phialoconidium produced within a phialide, as in *Chalara***.**

endoparasite a parasite that enters and feeds within its host.

endosperm the nutritive protein material within the embryo sac of seed plants; storage tissue in the seeds of gymnosperms.

endodermis the innermost layer of cortex surrounding the stele; characteristic of all roots and the stems of a few plants.

enteroblastic a situation where the inner wall (tretic conidium formation) or neither wall (phialidic conidium formation) of the blastic conidio- genous cell contributes to the formation of a blastoconidium.

enzyme a protein produced by a living organism that catalyzes organic reactions.

epidemic an increase of disease with time; in popular terms describes a disease or pest that has rapidly increased to a high level.

epidemiology the study of the initiation, development and spread of infectious disease.

epidermis the outermost layer of tissue on the surface of an organism.

epinasty a conditon resulting from the more rapid growth of the upper side of an organ, e.g. in a leaf, resulting in downward curling of the leaf blade.

epiphyllous on the upper side of a leaf.

eradication the elimination of an organism from a specific area.

erumpent bursting through the surface of the substrate.

etiology the study of the cause of disease.

exudate a substance that is exuded or discharged; ooze.

exuviae an arthropod artifact consisting of the molted body wall or fragments thereof.

F

facultative parasite/saprophyte an organism capable of changing its lifestyle, e.g. from saprophytic to parasitic or the reverse.

facultatively aerobic relating to a microorganism that sometimes lives, or a process that sometimes occurs, in the presence of molecular oxygen.

facultatively anaerobic relating to a microorganism that sometimes lives, or a process that sometimes occurs, in the absence of molecular oxygen.

falcate curved like the blade of a scythe or sickle.

fallow refers to fields or soils that are cultivated and kept free from a crop or weeds during the normal growing season.

family a category of classification in plants and animals; above genus and below order.

fascicle a small group or bundle of conidiophores.

fasciculate growing in fascicles.

fastidious having special growth and nutritional requirements; usually in reference to bacteria.

femur (-ora) a segment in the insect leg.

fibrosin bodies straight or slightly curved, dark structures occasionally found in fungal spores, e.g. *Erysiphe cichoracearum.*

field capacity water content of soil after it has been flooded and allowed to drain.

filiform thread-like.

flaccid not stiff; limp.

flagellum (-a) a whip-like appendage of a motile cell.

fleck a small spot.

flexuous flexible; bending in a zigzag manner; wavy.

fluorescence emission of light that is caused by the flow of some form of energy into the emitting body and which ceases abruptly when the flow of energy ceases.

foot cell the basal cell of the macroconidium of *Fusarium* species; also, the basal cell supporting the conidiophore of *Aspergillus* species.

foot rot necrosis of the basal stem, crown, and often the roots.

forma specialis (formae speciales) a category in classification within a species; distinguishable mainly by pathogenicity on specific host plants; abbreviated **f. sp.**

frass an animal artifact; consists of solid excrement or a mixture of excrement and destroyed plant tissues.

fruit body a fungal structure that produces or contains spores.

fumigant a toxic chemical in the form of a gas or a volatile liquid that becomes gaseous upon release; used to disinfest an area or space from pest organisms.

fumigation the practice of injecting a gas or a volatile liquid chemical into soil or other type of growing media; the use of chemicals in gaseous form for the purpose of disinfestation, whether performed in storage areas, in the field under tents, or by direct application to the soil under cover of a tarpaulin or plastic sheet.

fungicide a chemical used to inhibit or kill fungi.

fungistasis the nonlethal inhibition of fungal growth or spore germination.

fungus (-i) a non-photosynthetic (heterotrophic) lower organism composed of hyphae, usually reproducing by spores, and deriving nutrients from other organisms or dead organic matter.

funiculus an ovule stalk in the ovary or fruit or a flowering plant.

fusiform spindle-shaped; tapering toward the ends.

fusoid somewhat fusiform.

G

gall an abnormal growth or swelling of plant tissue caused by certain bacteria, fungi, viruses, insects, mites and nematodes.

gametangium (-ia) cells or structures that fuse to produce sexual cells or spores, e.g. zygospores in Mucorales.

gene a unit within a chromosome organism controlling one or several heritable characteristics.

generation the life of an organism from any given stage in its life cycle to the same stage in its progeny.

genetic relating to heredity; referring to heritable characteristics.

geniculate bent abrubtly at an angle, as in a bent knee.

genome the genetic endowment of an organism; the haploid set of chromosomes.

genus (-era) a category of classification in plants and animals; above species and below family.

germ tube a fungal hypha produced by a germinating spore.

globose spherical or almost so.

Gram-negative bacteria that decolorize and stain with the pink counterstain when treated with Gram's stain.

Gram-positive bacteria that retain the violet stain and do not decolorize when treated with Gram's stain.

granulosis virus (-es) a baculovirus with a single virion embedded in each granular inclusion body.

grub a popular term for the larva of insects such as beetles and moths; thick-bodied, whitish and usually slow moving.

guttation pertains to the exudation of water droplets from plants; occurs particularly along leaf margins through hydathodes.
guttulate containing one or more oil-like drops, e.g. in spores or cells.

H

habitat the natural place of occurrence of an organism.

haploid the state of having unpaired chromosomes.

haustorium (-ia) a modified fungal hypha inside a host cell used for the absorption of nutrients.

head the anterior division of an animal's body.

hectare a unit of measure for an area of land 10 000 m^2 in size; abbreviated **ha.**

herbs the leaves and stems of soft-stemmed plants of which the main stem dies down to the ground at the end of the growing season; occasionally, woody- or semi-woody-stemmed plants, e.g. rosemary, sage and thyme, are considered herbs, used for food flavoring or medicinally; distinguished from spices by their lower content of essential oils and their use to produce delicate or subtle flavors, in contrast to the aromatic flavors imparted by spices; see spices.

heteroecious undergoing different parasitic stages on two unlike hosts, as in the rust fungi.

heterokaryon a cell with two or more genetically different haploid nuclei.

heterothallic existing in two or more self-incompatible mating types, as in many fungi and some algae.

heterozygous having two dissimilar alleles in a diploid cell.

hilum (-s) in plants, a scar on a seed marking the point of detachment from the funiculus; in fungi, a scar or mark on a spore at the point of attachment to a conidiophore or sterigma.

holohlastic pertaining to eggs that undergo total cleavage due to the absence of a yolk mass; a process by which fungal spores are formed from a conidiogenous cell and where both the inner and outer walls of the conidiogenous cell contribute to the formation of a blastoconidium.

homothallic having genetically compatible hyphae; the absence of self- incompatible mating types in a fungal species.

homozygous having two identical alleles in a diploid cell.

honeydew a sugary fluid excreted from the anus of insects such as aphids.

host a living plant or animal from which a parasite obtains its food.

host range all the host species that are subject to attack by a parasite.

hyaline colorless or transparent.

hybrid the offspring of sexual reproduction by two individuals differing in one or more heritable character traits.

hydathode a structure on a leaf with one or more openings that exudes water from the interior onto the surface.

hydrolysis decomposition or alteration of a chemical substance by water; reactions of cations with water to produce a weak base or of anions to produce a weak acid.

hyménium (-ia) the spore-bearing layer of a fungal fruiting body, especially of ascomycetes and basidiomycetes.

hyperplasia increase in cell number causing an increase in the size of a tissue or organ.

hypersensitive extremely or excessively sensitive; having a type of resistance resulting from extreme sensitivity to a pathogen.

hypertrophy excessive growth or abnormal cell enlargement.

hypha (-ae) a vegetative filament of a fungus.

hypocotyl the portion of a stem below the cotyledon.

hypodermis the outermost cell layer of the cortex of plants, also known as the exodermis; the layer of cells that underlies and secretes the cuticle in anthropods and some other invertebrates.

hypophyllous located or growing on the lower surface of a leaf.

I

ilarvirus a group of viruses of which the type member is tobacco streak virus.

imago (-s) see **adult.**

imperfect state or **stage** see **anamorph.**

imperfect fungus (-i) see **imperfect state.**

incipient an unnoticeable or hidden stage early in the progression of a disease.

infect to enter and colonize a host.

infection the establishment of a pathogen within a host.

infectious disease a pathogen-induced disease that can spread to a healthy plant.

infective capable of infecting a host.

infest to be present in or on a substrate or area; usually implies large numbers of a pest organism.

inoculate to bring a pathogen into contact with a host plant or plant part.

inoculation the transfer of a pathogen to a host.

inoculum (-a) any infectious material; a pathogen or its parts that can cause infection.

inoculum potential the energy or potential of inoculum to cause disease.

insect an arthropod with three main body divisions and three pairs of legs in the adult stage.

insecticide a chemical or other agent used to control insects.

instar a developmental period between molts of the larval or nymphal stage in immature insects; numbered from beginning to end as first instar, second instar, etc.

integrated control a management strategy that makes use of any or all available methods to control a disease or pest, or to control all the diseases and pests of a crop, at the lowest cost and with the least damage to the environment.

intercalary being along and within a fungal hypha rather than at the ends of the hypha.

intercellular between cells.

internode a portion of a stem between the nodes.

intracellular within a cell or cells.

intumescence production of blisters on leaves, usually under conditions of high moisture and restricted transpiration.

invasion the spread of a pathogen or pest into a host or crop.

invertebrate animals without an internal bony skeleton.

in vitro in artificial culture

in vivo in real life; in nature.

isolate a culture of a microorganism, and usually the subcultures derived from it; also, a collection of a pathogen made at a specific time.

isolation pertains to the separation of an organism from its substrate or habitat, and the culture of same on a nutrient medium.

isometric the variation of pressure with temperature when the volume of the substance is held constant.

isometric particle a virus particle with all sides of equal length; appears spherical when viewed with an electron microscope.

K

kingdom the highest category in the classification of living organisms.

L

lacuna a small space or depression.

lageniform flask-shaped.

lamellae thin scales, plates or membranes, e.g. the hymenium-covered vertical plates on the underside of the cap of a mushroom.

lamina a thin sheet or layer of tissue; a scale-like structure; the blade of a leaf.

larva (-ae) a developmental, immature stage in invertebrate animals; worm-like, with or without legs; occurs between the egg and pupa in certain insects.

latent infection the state in which a host is infected with a pathogen but symptoms are not apparent.

lenticel a loose-structured opening in the periderm beneath the stomata in the stem of many woody plants and in potato tubers that facilitates gas transport.

lesion localized area of diseased tissue.

lethal dose a statistic about a toxicant; usually given as the amount of active ingredient needed to kill a given proportion of an animal or pathogen population; for example, the **LD 50** value refers to the amount required to kill 50% of individuals exposed to it.

leucoplast a nonpigmented plastid, capable of developing into a chromoplast.

life cycle the series of changes undergone by an organism from fertilization or spore formation to death.

lipolytic capable of dissolving fat; fat-reducing.

locule a cavity, especially in the stroma of an ascomycete.

looper a moth larva that lacks some of the abdominal prolegs, causing the body to arch when moving.

LOPAT test a series of tests used to distinguish between pathogenic and non-pathogenic fluorescent pseudomonad bacteria; L- levan production, 0 - oxidase positive, P - potato rot, A - arginine dihydrolase positive, and T - hypersensitivity on tobacco.

lunate like a new moon; crescent-shaped.

luteovirus a group of viruses of which the type member is barley yellow dwarf virus.

M

macroconidium (-ia) the larger, septate and generally more diagnostic conidium of a fungus, which also produces microconidia; a long, large conidium.

macrocyclic a rust fungus having binucleate urediniospores as well as teliospores and sporidia; having a life cycle that is long or complex.

macroscopic visible to the naked eye without the aid of a magnifying lens or microscope.

maggot a fly larva that lacks legs and has no apparent head.

mating types morphologically identical forms of a fungus that are sexually self-incompatible but can interact to produce sexual spores.

matric potential a measure of the water content of soil; measured in bars or atmospheres.

matrix intercellular substances in which cells are embedded.

medium (-ia) an artificial food or substrate on which an organism can be grown.

medulla pith; central core of usually parenchymatous tissue in those stems in which the vascular tissue is in the form of a cylinder; functioning in food storage; may occur in some roots where central tissue develops into parenchyma instead of xylem.

meiosis sexual or reduction division of cell nucleus whereby chromosome number is halved.

meiotic pertaining to meiosis.

melanin dark brown pigment in which different concentrations give brown and yellow coloration.

meristem localized region of active cell-division in plants from which permanent tissues are derived.

metabasidium the developmental stage of a basidium in which meiosis occurs.

microconidium (-ia) a smaller, usually nonseptate conidium of a fungus also having macroconidia.

microorganism any organism of microscopic size.

microsclerotium (-ia) a very small sclerotium.

microscopic visible only with the aid of a magnifying lens or microscope.

midrib large central vein of a leaf.

millipede arthropods with a head and an undifferentiated body consisting usually of more than 20 segments, many of which have two pairs of legs; cylindrical, coiling when disturbed.

mite arthropods with two main body divisions and usually four pairs of legs in the adult stage, related to spiders and ticks; microscopic.

miticide see **acaricide.**

mitosis asexual or duplication division of chromosomes.

mitotic pertaining to mitosis.

molar solution a solution that contains one mole (gram-molecular weight) of solute in one liter of the solution; abbreviated M.

mold (syn. mould) any fungus that produces conspicuous spores or profuse or woolly mycelium on the outer surface of a host or substrate.

mollusc invertebrate animals that may or may not have an external shell; includes terrestrial slugs and snails.

molluscicide a chemical or other agent used to control molluscs.

molt a process in an animal's life cycle when old epidermis is shed, usually but not always before entering another stage of growth and development.

moniliform having swellings at regular intervals like a string of beads.

monoclinous having the antheridium on the oogonial stalk.

monocotyledon a plant with one cotyledon, e.g. grasses, small grain cereals and corn.

monoculture exclusive production of a single crop.

monogenic resistance resistance determined by a single gene.

monokaryon a cell containing one haploid nucleus.

monokaryotic pertaining to a monokaryon.

monophialide a conidiogenous cell from which a basipetal succession of conidia develop without an increase in length of the phialide itself.

montmorillonite a group name for all clay minerals with an expanding structure, except vermiculite.

mosaic pattern of light and dark green or yellow areas, as in a leaf.

multitrichous having many hairs.

muriform resembling the arrangement of courses in a brick wall, e.g. fungal spores having both horizontal and vertical septa.

myceliogenic forming or growing as mycelium.

mycelium (-ia) a collective term for fungal hyphae.

mycoplasma (-s) a popular term for an organism that is Gram negative, generally non-sporing, non-motile, lacking a cell wall, and resembling a bacterium; usually occurs in association with vertebrates, often as a pathogen; belongs to the class Mollicutes.

mycoplasma-like organism similar to a mycoplasma but not fully understood; a term applied to certain fastidious prokaryotic plant pathogens capable of causing "yellows" type diseases; formerly thought to be viruses with unusual characteritics but now thought to be unique; occurring in phloem sieve cells; transmitted by and propagated in insects, particularly leafhoppers; abbreviated MLO.

mycorrhiza (-e) a symbiotic association of a fungus with the roots of a plant that is beneficial to both organisms.

mycotoxin a toxin produced by certain fungi.

N

necrosis death of cells; in plants, often associated with loss of color or with darkening or discoloration of tissues.

necrotic pertaining to (causing or undergoing) necrosis.

necrotroph a parasite that derives its energy from dead cells of the host.

nematicide a chemical or other agent used to control nematodes.

nematode invertebrate animals that live in water or soil as saprophytes or as endo- and ectoparasites of plants; generally worm-like and microscopic.

neoplasm (-s) a tumor; abnormal localized multiplication of a cell.

non-acid-fast bacteria see **acid-fast bacteria.**

non-infectious disease a disease that is caused by an environmental factor, not by a pathogen.

non-persistent transmission a form of virus transmission in which the virus remains transmissible for a short period, e.g. hours or days, while in association with its vector.

nucleic acid an acidic substance in the cell nucleus having to do with heredity by being duplicated and passed from one generation to the next.

nucleotide a compound composed of an organic phosphate, a pentose sugar, and a nitrogen base; the structural units of DNA and RNA.

nutrient film technique a form of crop culture that requires no soil and recycles a balanced supply of nutrients and oxygen in water; abbreviated **NFT.**

nymph a developmental, immature stage in insects that do not have a larva; occurs between the egg and adult; adult-like, but lacking fully developed wings and sex organs.

O

obclavate inversely clavate; widest at the base.

obligate parasite a parasite that, in nature, can grow and multiply only on or in a living host.

obovate inversely egg-shaped; the narrow end is usually attached to a stalk, e.g. some leaves or spores.

obovoid inversely ovoid; roughly egg-shaped with the narrow end downward.

obpyriform inversely pear-shaped; attached at the base or with the wider end downward.

obtuse rounded or blunt.

oedema see **edema.**

oogonium (-ia) the female gametangium of oomycete fungi; contains one or more oospheres.

oomycete a category of fungi, the Mastigomycotina; produces oospores and biflagellate zoospores.

oospore a diploid fungal resting spore; develops in the oomycetes as a thick-walled resting stage arising from the fertilized oosphere.

order a category of classification in plants and animals, above the family and below the phylum.

osmosis a diffusion that takes place between two miscible liquids through a permeable membrane.

ostiole a pore-like opening in perithecia, pycnidia and pseudothecia through which spores escape.

oviposition the act of laying eggs.

ovipositor egg-laying structure(s) in certain insects.

ovule a structure in the ovary of a seed plant that develops into a seed after fertilization.

oxidation the combining of oxygen with or the removal of hydrogen from an element or compound.

ozone a highly reactive form of free oxygen that consists of three oxygen atoms; may cause plant injury, even at very low concentrations.

P

papilla (-ae) a small, rounded process.

papillate having or covered with papillae.

papulaspore a large multicellular asexual spore, e.g. in *Papulaspora sepe- doniodes.*

paragynous having the antheridium at the side of the oogonium.

paraphysis (-es) a sterile, upward growing, basally attached hyphal element in a hymenium.

parasexual recombination genetic recombination other than by means of the alternation of karyogamy and meiosis that is characteristic of sexual reproduction.

parasite an organism that lives on or in a plant or animal host, harming and sometimes killing the host.

parenchyma a tissue of higher plants consisting of living cells with thin walls that are agents of photosynthesis and storage; abundant in leaves, roots, and the pulp of fruit, and found also in stems.

parthenogenesis refers to eggs that develop without fertilization in some aphids, beetles, wasps and other insects, and in some nematodes.

pathogen an infective agent; any living organism that incites disease.

pathogenicity the capacity of a pathogen to cause disease.

pathovar a category in bacterial classification below the level of species that is characterized by pathogenic reaction in one or more hosts; pathotype.

pectin a carbohydrate in cell walls often extracted from the inner portion of the rind of citrus fruits, or from apple pomace; consists chiefly of partially methoxylated polygalacturonic acids.

pectolytic capable of degrading pectin through enzyme activity, e.g. pectinase and pectase.

pedicel the stem of a fruiting or spore-bearing organ.

pedicellate having a pedicel.

peduncle a flower-bearing stalk; also a stalk supporting the fruiting body of certain thallophytes.

penicillate having a tuft of fine hairs; brush-like.

percurrent growing through in the direction of the long axis, as in a conidial germ tube emerging through the hilum or as a proliferation through the tip of a conidiogenous cell.

perennial a plant that can survive continuously for three or more years.

perfect state see **teleomorph.**

periderm a group of secondary tissues forming a protective layer that replaces the epidermis of many plant stems, roots, and other parts; composed of cork cambium, phelloderm, and cork; in animals, the superficial transient layer of epithelial cells of the embryonic epidermis.

perithecium (-ia) a fungal ascocarp; flask-like with an ostiole.

peritrichous having hairs or flagella all over the surface.

persistent transmission a form of virus transmission in which the virus remains transmissible for a prolonged period, i.e. several weeks, while in association with its vector.

pest any living organism that is undesirable and of economic concern.

pesticide any substance or mixture of substances that is used to kill or manage pests.

petiole the stalk of a leaf.

pH an expression of the degree of acidity or alkalinity of a solution, where the hydrogen ion concentration changes by a factor of ten for each unit of change on a scale of 0 to 14; see **acidic** and **alkaline.**

phenology study of periodic and developmental biotic events, e.g. flowering, migration, etc., in relation to climate and other factors.

pheromone a volatile substance that insects secrete; usually active at very low concentrations; affects the behavior of other members of the same species.

phialide a conidiogenous cell that develops one or more open ends from which a basipetal succession of conidia develops without an increase in length of the phialide itself.

phialidic the sort of enteroblastic conidiogenesis in which each conidium is delimited by a new wall that is not derived from existing walls or layers of the wall of the conidiogenous cell.

phialoconidium (syn. phialospore) a conidium produced on a phialide.

phloem the food-conducting tissue of a vascular plant, consisting of sieve tubes, companion cells, parenchyma cells and fibers.

photosynthesis formation of sugars in plants from carbon dioxide and water under the influence of light and dependent upon chlorophyll.

phyllody change of floral organs to leaflike structures.

phylum (-a) a category of classification in plants and animals, above order and below kingdom.

phytophagous plant-feeding animals, such as certain insects, mites, millipedes, molluscs, and nematodes.

phytotoxic the capacity to harm or kill plants.

pileus (-ei) the umbrella-shaped upper cap of mushrooms and other basid- iomycete fungi.

pinnule the secondary branch of a plume-like or pinnate organ.

pionnote a spore mass having a fatty or grease-like appearance, e.g. in some *Fusarium* species.

plasmid an extrachromosomal genetic element found in various strains of bacteria.

plasmodium (-ia) a multinucleate, motile mass of protoplasm, generally reticulate and lacking a firm wall; characteristic of the growth phase of myxomycete fungi.

plastid a small, specialized body in the cytoplasm of a plant cell that is the site of activities such as food manufacture and storage.

pleomorphic variable in shape; in fungi, having more than one spore state.

polyhedrosis virus a baculovirus with several virions embedded in a multi-sided inclusion body.

polypore members of the family Polyporaceae, a group of wood-decaying fungi.

polysaccharide a carbohydrate composed of many monosaccharides.

potexvirus a group of viruses of which the type member is potato virus X.

potyvirus a group of viruses of which the type member is potato virus Y.

predator an animal that kills and feeds on other animals, consuming many of its prey in its lifetime.

predisposed prone to infection because of some specific environmental factor.

prepupa (-ae) a developmental stage in insects; a fully fed larva, usually quiescent, before molting to the pupal stage.

primary root the first root that develops from a seed.

primary inoculum (-a) the inoculum of a pathogen that causes primary infection of a plant or crop.

primary infection the first infection of a plant by the overwintering or oversummering inoculum of a pathogen.

primordium (-ia) the earliest stage of development of an organ.

proboscis (-es) a set of protruding mouthparts in sucking insects.

prokaryote an organism lacking membrane-limited nuclei and not exhibiting mitosis, e.g. in bacteria.

proleg a fleshy leg on the abdominal body segments of an insect larva; bilaterally paired.

promycelium the germ tube of the teliospore (Uredinales) or ustilospore (Ustilaginales) from which promycelial spores (sporidia) are produced.

propagule a reproductive structure, e.g. a spore or vegetative part capable of dissemination and able to germinate or propagate.

propupa (-ae) a developmental stage in thrips before becoming a pupa; non-feeding, usually quiescent but capable of movement.

protectant a substance that inhibits or prevents infection.

proximal nearer the main body or point of attachment.

pseudoparaphysis (-es) a fungal structure resembling a paraphysis.

pseudoparenchyma fungal tissue resembling parenchyma.

pseudosclerotium (-ia) a fungal structure resembling a sclerotium, often containing remnants of decayed host tissue and substratum such as soil or stones.

pseudostroma (-ata) a stroma of fungal tissue and remnants of host tissue.

pseudothecium (-ia) a fungal ascocarp resembling a sexually derived perithecium; an ascostromatic ascocarp having asci in numerous, unwalled locules, as in the Loculoascomycetes.

pulvinus a cushion-like enlargement of the base of a petiole which functions in turgor movements of leaves.

pupa (-ae) a developmental, immature, non-feeding stage in insects with a larval stage; occurs between the larva and adult stages; often capable of limited movement; may be in a covering or cocoon.

puparium (-ia) a pupa that forms inside the last larval body without it molting, such as in flies and whiteflies.

pustule an eruption on the exterior of a host usually containing spore masses of the pathogen.

pycnidiospore a conidium formed in a pycnidium.

pycnidium (-ia) a flask-like asexual (anamorphic or conidial) fungal fruiting body.

pycniospore a haploid spermatium formed in a pycnium, i.e. in the Uredinales.

pycnium (-ia) a fungal fruiting body containing pycniospores and arising from basidiospore infection, e.g. in the Uredinales.

pyriform pear-like in form; attached at the narrowest end or with the narrow end down.

Q

quarantine human intervention to prevent the spread of diseases and pests by exclusion or enforced isolation.

quiescent inactive, latent or dormant; often referring to disease or a pathological process.

R

race a category of classification in plants and animals below the species and subspecies level; a genetically distinct group of a plant pathogen specific to one or several plant cultivars.

racemose bearing flowers or structures in clusters (racemes).

rachilla (-e) the axis of a grass spikelet.

radicle the main root(s) of a germinating seedling.

ramoconidium (-ia) an apical branch of a conidiophore that secedes and functions as a conidium, as in *Cladosporium***.**

ramulus (-i) a small branch.

recessive an allele that is not expressed phenotypically when present in the heterozygous condition.

recurvate bent backwards.

repellent a chemical that drives pests away from a treated object or area.

resistance the ability of an organism to withstand attack.

resting spore a thick-walled spore of a fungus which is resistant to extreme conditions of temperature and moisture, and which often germinates only after a period of dormancy.

rhizomes underground horizontal stems, often thickened and tubershaped, and possessing buds, nodes, and scale-like leaves.

rhizosphere that part of the soil occupied by living roots and in which the activity of microorganisms is increased.

RNA ribonucleic acid; a long chain, usually single-stranded nucleic acid consisting of repeating nucleotide units containing four kinds of hetero

cyclic organic bases: adenine, cytosine, guanine, and uracil; involved in intracellular protein synthesis.

rogue removing diseased or atypical plants from a crop.

rostrate beaked.

rot softening, discoloration and often disintegration of plant tissue as a result of fungal or bacterial infection.

rotation growth of different kinds of crops in succession in the same field.

russet an area on a plant surface that is brownish and roughened due to cork formation.

rust a type of fungus (Uredinales); the disease caused by one of the rust fungi.

S

sanitation the removal of infected plant residue; the decontamination of tools, equipment, hands and clothing.

saprophyte an organism that utilizes dead or decaying plant material as its food.

saprotroph an organism that utilizes dead or decaying organic matter for its food.

scab a roughened crust-like area on a plant surface; a disease that causes such areas to form.

scion a section of a plant, usually a stem or bud, which is attached to the stock in grafting.

sclerotioid resembling a sclerotium.

sclerotium (-ia) a mass of fungal mycelium that serves as a resting stage; often hard and compact.

scorch the burned appearance of leaf margins caused by infection or unfavorable environmental conditions.

secondary root a root that develops from a crown or node.

secondary infection infection subsequent to primary infection which is caused by secondary inoculum and usually involving spatial dissemination of the pathogen.

secondary inoculum (-a) the inoculum that spreads disease within a crop, arising from a primary infection during the same season.

secondary organism an organism that multiplies in already diseased tissue but is not the primary pathogen.

sedentary stationary; staying in one place.

seed leaf see **cotyledon.**

seed piece a portion of a potato tuber that is planted to produce a new plant; contains at least one bud eye.

seed treatment application of a biological agent, chemical substance or physical treatment to seed, to disinfect; to disinfest or protect seed or plant from pathogens or to stimulate germination of the seed or growth of the plant.

selective medium a culture medium suitable for the isolation of one or a few kinds of microorganisms.

senesce to age.

sepal one of the structures composing the calyx.

septate having cross walls.

sessile attached directly to a branch or stem without an intervening stalk; the condition of a plant or fungal structure lacking a stalk or stem.

seta (-ae) a hair-like structure.

sexual state see **teleomorph.**

siemens a unit of conductance; equal to the conductance between two points of a conductor such that a potential difference of one volt between these points produces a current of one ampere; abbreviated **S**.

skeletonize feeding that removes most of the interveinal tissue of a leaf.

slug terrestrial mollusc with no apparent or a much reduced external shell.

smut a type of fungus with dark powdery spores; the disease produced by smut fungi (Ustilaginales).

snail mollusc with an external shell; may be aquatic or terrestrial.

solarization the practice of covering the soil with a transparent plastic sheet to trap heat from the sun, thus warming the soil; used to reduce the population of certain pathogens, such as nematodes and fungi.

sorus (-i) a fungal spore mass, as in rusts and smuts.

spawn-run the colonization of compost by mushroom spawn.

species a category of classification in plants and animals, theoretically referring to a group of similar individuals capable of interbreeding; below genus and above subspecies; abbreviated **sp.** (singular) or **spp.** (plural).

spermagonium (-ia) a walled structure in which spermatia are produced, as in ascomycetes and rust fungi (= pycnium).

spermatium (-ia) a haploid fungal cell usually produced in a spermagonium; functions sexually.

spices dried plant products used primarily to season food, including "true spices" (e.g. pepper, cinnamon, nutmeg), potent herbs (e.g. basil, marjoram), aromatic seeds (e.g. sesame, cardamon), blends (e.g. pumpkin pie spice) and dehydrated vegetables (e.g. onion, garlic); see herbs.

spiracle a breathing pore on the surface of the body in insects; usually lateral and bilaterally paired.

sporangiophore a specialized hypha, bearing one or more sporangia.

sporangium (-ia) a fungal fruiting body that is usually microscopic; contains one or more endogenous, asexual spores, e.g. in the Myxomycètes and Mastigomycotina.

spore a fungal reproductive structure, one- or many-celled.

sporidium (-ia) a basidiospore of rust and smut fungi.

sporodochium (-ia) a superficial, cushion-like fruiting body or conidioma bearing conidiophores on its surface.

sporogenous producing spores.

sporophyte the diploid or asexual phase in the life cycle of a higher plant.

sporulate to produce spores.

stadium (-ia) see **stage.**

stage a developmental period in the life of an arthropod, e.g. the egg stage, larval stage, pupal stage, and adult stage.

stele the part of a plant stem including all tissues and regions from the cortex inward, including the pericycle, phloem, cambium, xylem and pith.

sterigma (-ta) a slender stalk arising from the basidium of some fungi, on the top of which basidiospores are formed.

sterilization the elimination of pathogens or other living organisms from a substrate, such as soil or containers, by means of heat, chemicals or irradiation.

stipe a stalk.

stipitate stalked.

stolon a creeping stem or runner capable of forming roots and stems, and ultimately a new individual; hyphae produced above the surface and connecting groups of sporangiophores.

stoma (-ata) a specialized opening in the plant epidermis.

stomate see **stoma.**

strain a distinct form of an organism or virus within a species, differing from other forms of the species biologically, physically or chemically.

stroma (-ata) a compact mycelial structure on or in which fungal reproductive structures usually are formed.

stylet a slender feeding structure in some nematodes and some insects, e.g. aphids and whiteflies.

suberization infiltration of plant cell walls by suberin resulting in the formation of corky tissue that is impervious to water.

suberin a complex mixture of oxidation and condensation products of fatty acids present in walls of cork cells, rendering them impervious to water.

subglobose approximately spherical.

subobclavate approximately obclavate.

subspecies a category of classification in plants and animals; below the level of species; abbreviated **subsp.** (singular) or **subspp.** (plural).

substomatal situated beneath the stomata of plant epidemis; often in association with a chamber or cavity.

substrate the substance on which an organism lives or from which it obtains nutrients; chemical substance acted upon, often by an enzyme.

subulate awl-shaped; narrow and tapering from a base to a fine point.

susceptible lacking the ability to resist disease or attack by a given pathogen.

sympodial a continuation of growth of a conidiogenous cell, after the main axis has produced a terminal spore, by developing a succession of new apices, each of which originates below and to one side of the previous apex.

symptom any external or internal sign of disease in a plant.

synanamorph applied to any one of the two or more anamorphs which have the same teleomorph.

synnema (-ata) (syn. coremium) a group of conidiophores, usually united, that bear conidia.

synonym a term in biological nomenclature referring to another, usually previously used name for the same organism; abbreviated **syn.**

systemic spreading from within, throughout the entire body; applies to a pathogen or chemical in a host.

T

tassel the male inflorescence of corn and certain other plants.

teleomorph that part of the life cycle of a fungus in which sexual spores, i.e. ascospores or basidiospores, are produced.

teliospore a spore produced by some basidiomycete fungi, primarily the Uredinales, often with a thick, dark wall; sometimes a resting or overwintering stage that gives rise to a basidium; dikaryotic at first, with the nuclei fusing and undergoing meiosis before spore germination; the “black” stage of rust fungi.

telium (-ia) a sorus producing teliospores.

teneral the state of being incompletely hardened or darkened, as in insects after molting.

teratogenic causing morphological malformations or monstrosities.

testa a seed coat.

thallophyte a thallus plant, e.g an alga, bacterium or fungus; a member of the Subkingdom Thallophyta.

thallus a plant body that is not differentiated into special tissue systems or organs and may vary from a single cell to a complex, branching, multicellular structure.

thorax (-ces) the middle division of an insect body.

threshold a level beyond which an activity begins or ceases.

Ti-plasmid a tumor-inducing plasmid found naturally in the gall-producing bacterium *Agrobacterium tumefaciens.*

tiller side shoot arising at ground level.

tissue a group of cells of similar structure and function.

tobamovirus a group of viruses of which the type member is tobacco mosaic virus.

tolerance according to law the allowable amount of toxic residue in, or on, an edible plant or plant part on the allowable percentage infection of pedigree seed on certified potato tubers with a pathogen; the ability of a plant to sustain attack by a pathogen or other organism without suffering heavy yield loss.

torulose cylindrical but having swellings at intervals; moniliform.

toxic poisonous; producing injury.

toxicant the poisonous chemical or agent in a formulated product.

toxicity the capacity of a compound to produce injury.

toxin a poisonous substance.

transmission the spread of an infective agent from one host individual to another.

transovarial through the ovary.

transpiration water loss by evaporation from leaf surfaces and through stomata.

trichome an appendage derived from the protoderm in plants, including hairs and scales.

true leaf any leaf produced after the cotyledons.

truncate abbreviated at an end, as if cut off.

turgid a state in which the cell wall is rigid, stretched by an increase in volume of cellular contents due to the absorption of water.

U

umbel an indeterminate inflorescence with the pedicels all arising at the top of the peduncle and radiating like umbrella ribs; there are two types, simple and compound.

umbonate having or forming a rounded or conical protuberance.

undulate to move in waves; wave-like in outline.

unilocular having a single cavity.

uniseriate in one row.

urediniospore (syn. urediospore) an asexual spore produced by rust fungi; binucleate, summer or "red" stage of rust fungi.

uredinium (-ia) (syn. uredium) a sorus producing urediniospores.

V

vacuole a membrane-bound cavity within a cell; may function in digestion, storage, secretion or excretion.

variety the taxon below subspecies; a group that distinctly differs from other varieties within the same subspecies.

vascular pertains to the conductive tissue or region of conductive tissue of a plant.

vascular pathogen a pathogen that grows primarily in the conducting tissues of a plant.

vector an organism that carries and transmits a pathogen from one host to another.

vegetative pertains to the somatic, non-reproductive growth stage of plants.

vein a vascular bundle in a plant leaf or the rib-like vessels in the wing of an insect.

ventral pertains to the lower or underside.

verrucose having the surface covered with wartlike protuberances.

verruculose delicately verrucose.

verticil a whorl, an arrangement resembling spokes of a wheel.

vessel a xylem element or series of such elements; functions to conduct water and mineral nutrients.

virescence the abnormal greening of floral parts.

virion a complete virus particle.

viroid (-s) the smallest known infective agent of plants, consisting of a short strand of ribonucleic acid with no protein coating; unable to multiply outside of living plants and unable to persist in the soil or in any kind of resting stage outside of a living host.

virulence the ability of a particular race or strain of a given pathogen to attack a host cultivar; the severity of attack on the plant.

virulent causing a disease; being pathogenic.

viruliferous carrying virus particles, as in an aphid.

virus (-es) an infective agent consisting of genetic material within a protein coat; unable to multiply outside of living plants or animals and unable to persist in the soil or in any kind of resting stage outside of host tissue.

W

water potential a measure of water content of soil and tissues based on the energy level of the water relative to pure water.

water-soaked a disease symptom in which the host tissue appears wet and dark or somewhat translucent.

wilt drooping or loss of turgidity of plants or plant parts due to insufficient water.

wing pad the undeveloped, encased wing of a nymphal insect; bilaterally paired.

witches'-broom an abnormal cluster of small branches, twigs or roots that grow as a result of attack by fungi, viruses, dwarf mistletoes or insects.

X

xylem the plant tissue involved in water conduction and which consists of tracheids, vessels, parenchyma cells and fibers.

Y

yellows a plant disease characterized by yellowing and stunting of the host; often associated with mycoplasma-like organisms.

Z

zonate having concentric lines, often forming alternating pale and darker zones near the margins.

zoosporangium (-ia) a sporangium producing zoospores.

zoospore a motile asexual fungal spore bearing flagella and capable of movement in water.

zygospore the resting spore of zygomycetes resulting from the conjugation of similar sex cells (isogametes) or by the fusion of like gametangia, as in the Zygomycetes.

Color plates

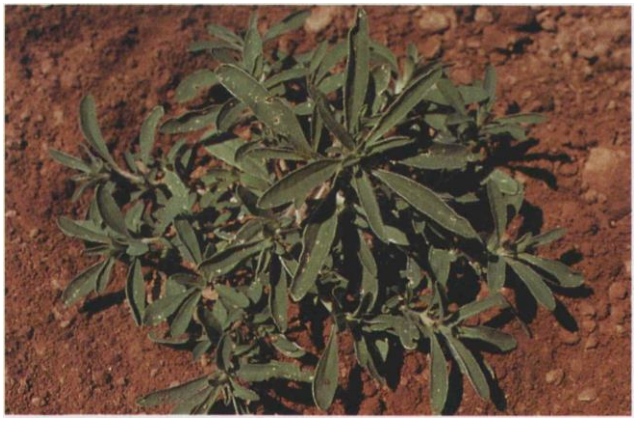
Locating Text Sections and Figures

Text sections are numbered consecutively within each chapter. For example, section 16.2 describes bacterial soft rot, the second topic of Chapter 16, Potato. To find a text section, refer to the running heads, which carry the inclusive section numbers for each two-page spread.

Color illustrations, pages 397-533, appear in the same order and have the same number as the corresponding text section; for example, figures *16.2a* and *16.2b* illustrate the symptoms of bacterial soft rot of potato. Line drawings, halftones and tables are numbered similarly, except that a text figure number contains the letter T; for example, Figure *16.2T1* illustrates the disease cycle of bacterial soft rot of potato.

2.3a Barnyard grass; seedling.

2.3c Green foxtail; seedling.



2.3e Kochia; seedling.

2.3g Redroot pigweed; seedling.

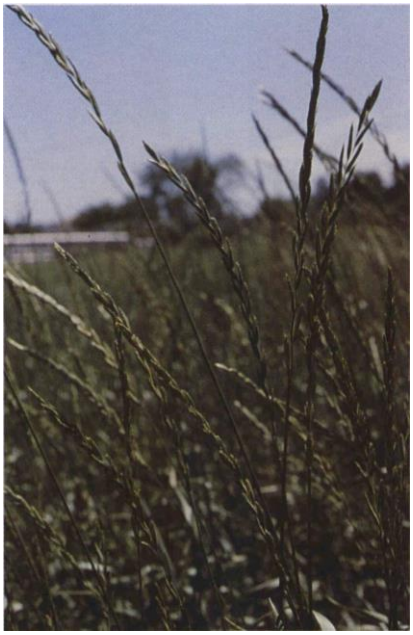
2.3b Wild buckwheat; seedling.

2.3d Common groundsel; flower bud stage.

2.3f Lamb's-quarters; seedling.

2.3h Shepherd's-purse; seedling.

2.3i Quack grass; seedling.



2.3j Quack grass; in head.

2.3k Canada thistle; in bloom.

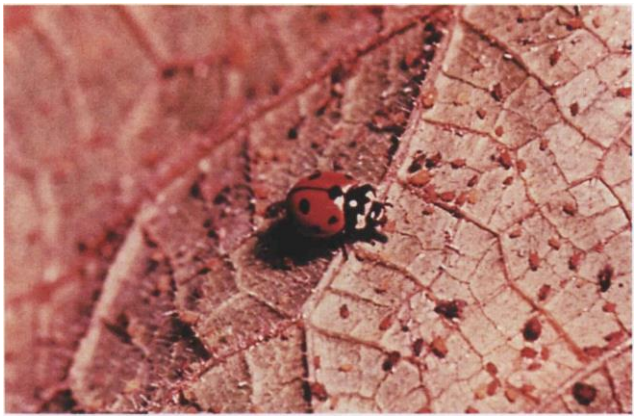
2.3m Quack grass; rhizomes.

2.3n Common ragweed; seedling.

2.3p Annual smartweed; seedling.

2.3q Clockwise from top: lamb's-quarters, shepherd's-purse, red- root pigweed, purslane.

3.7a Ground beetle (carabid); larvae beneficial, adults may damage small fruits; 4-12 mm.



3.7b Lady beetle; adult; both adults and larvae are predators on aphids.

3.7c Lady beetle; pupa on asparagus fern.

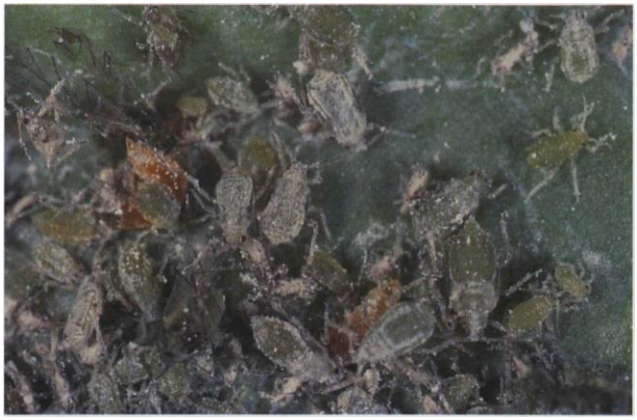
3.7d Green lacewing; larva feeding on an aphid (at top).

3.7e Green lacewing; adult and larva among aphids.

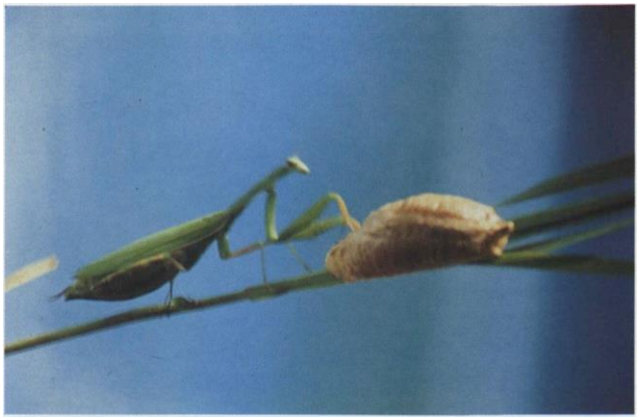
3.7f Hover fly; adult on flower.

3.7g Hover fly; larva on corn leaf.

3.7h Hover fly; larva.



3.7i Predatory midge (cecidomyiid); larvae (brownish) feeding on aphids on cabbage.



3.7j Praying mantis; a non-selective predator, adult and its egg mass.

3.7k Predatory stink bug; nymph attacking Colorado potato beetle larva.

3.7m Predatory stink bug; adult attacking Colorado potato beetle larva.

3.7n Predatory wasp (vespid); adult attacking a bee.

3.7p Parasitic wasp (chalcid); adults emerging from pupa of an imported cabbageworm.

3.7q *Encarsia formosa;* wasp parasite of greenhouse whitefly.

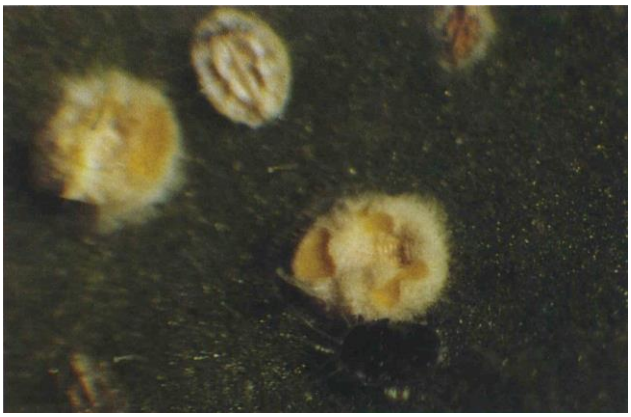
3.7r Parasitic wasp (braconid); cocoons on back of hornworm larva.

3.7s *Aphidius* sp.; a braconid wasp parasite of aphids.

3.7t Sticky trap for thrips; blue is preferred because it is less attractive than yellow to parasitic wasps.

3.7u Parasitic wasp (braconid); larvae of *Macrocentrus* sp. emerging from a potato stem borer larva.

3.7v Parasitic fly (tachinid); adult emerged from pupa, and host European corn borer larva.



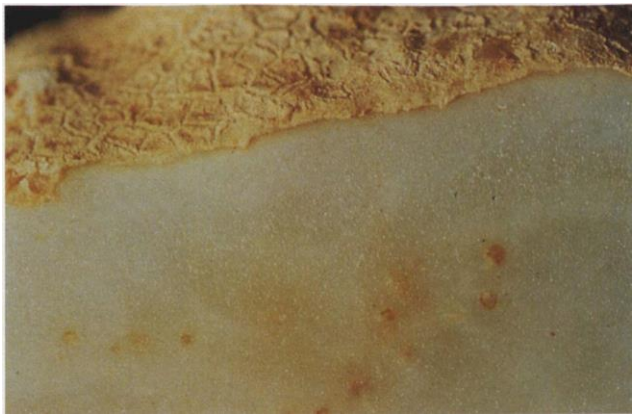
3.7w Greenhouse whitefly; pupae infected with the pathogenic fungus *Aschersonia aleyrodis.*

3.7x Cabbage looper; larva killed by nuclear polyhedrosis virus.

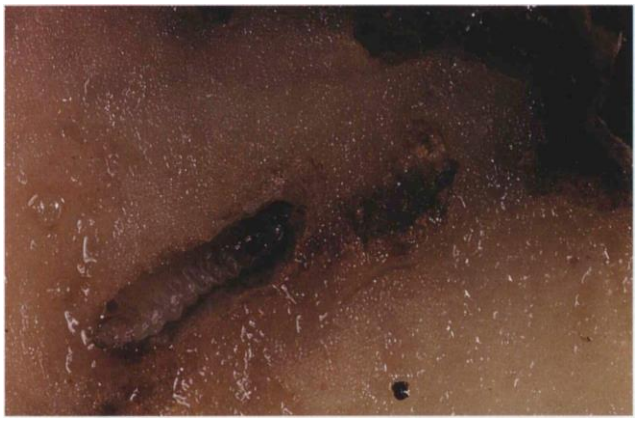
3.7y Cabbage looper; larval damage to untreated cabbage plants.

3.7z Cabbage looper; controlled with *Bacillus thuringiensis,* five applications.

3.10a Columbia root-knot nematode; galls on Russet Burbank potato tuber.



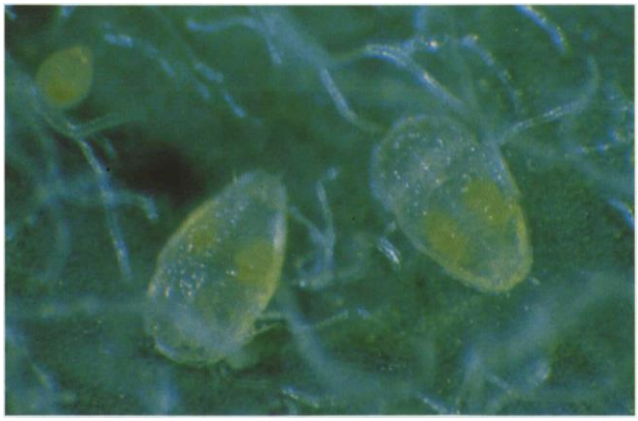
3.10b Columbia root-knot nematode; female nematodes and egg masses inside a potato tuber.



3.10c Potato tuberworm; larva tunneling in potato tuber.

3.10d Sweetpotato whitefly; adult and pupa.

3.10e Sweetpotato whitefly, adult (lower left); greenhouse whitefly, adult (right); note difference in angle of wings.



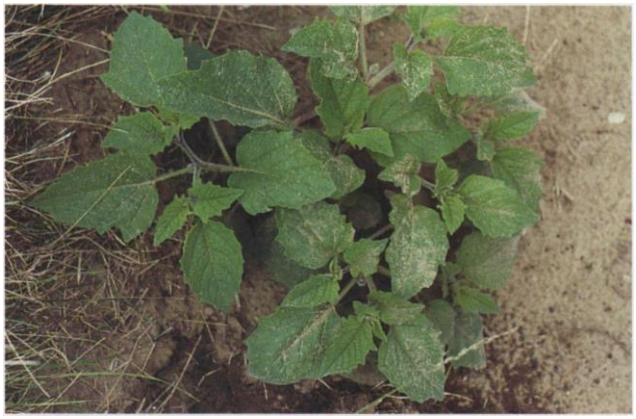
3.10f Sweetpotato whitefly; immature stages.

3.10g Sweetpotato whitefly; pupa parasitized by *Encarsia formosa*.

3.10h Tomato pinworm; larva on surface of a damaged tomato fruit.

3.11a Potato virus Y^N; mosaic symptoms in tobacco.

3.11b Potato virus Y^N; veinal necrosis symptom in tobacco.



3.11c Potato virus Y^N; ground cherry *(Physalis* sp.), a reservoir host of PVY^N.

3.11d Japanese beetle; adult resting on cinquefoil flower.

3.11e Brown garden snail; shell; width 32-38 mm. height 29-33 mm, number of whorls 4.0-4.5.

3.12 Nematode management; cucumber affected by southern root- knot nematodes (left); plants in fumigated soil (right) are taller.

3.13 Weed management; herbicide injury (leaf burn) to pea seedlings from atrazine residue in soil.

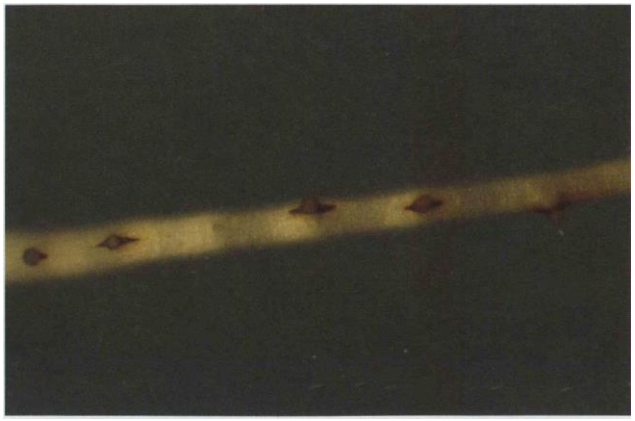
4.1a Botrytis blight; infection of lower canopy of asparagus ferns.

4.1b Botrytis blight; sporulation in the center of lesions on a fern stem.

4.2c Fusarium crown and root rot; decayed cortex and brown vascular bundles.

4.2a Fusarium crown and root rot; yellowing of infected ferns.

4.2b Fusarium crown and root rot; wilting of infected ferns.

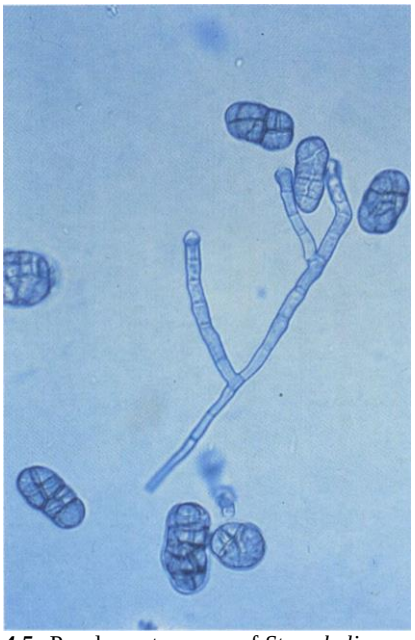


4.2d Fusarium crown and root rot; seedling root showing lesions at sites of emergence of lateral roots.

4.2e Fusarium crown and root rot; cross section of symptomless (left) and infected (brownish decay) crowns.

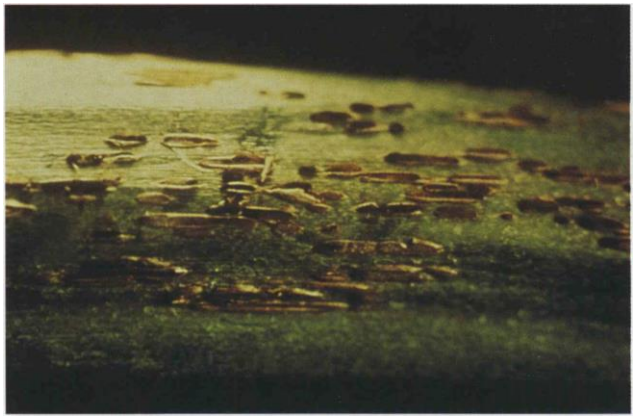
4.5a Purple spot; lesions at base of spear.

4.5b Purple spot; stem lesions.



4.5c Purple spot; spores of *Stemphylium vesicarium.*

4.6a Rust; aecial lesion on a fern stem.



4.6b Rust; lesions with urediniospores (reddish-brown) and teliospores (black).

4.8 Cold injury; stunting, curvature and purpling of spears early in the growing season.

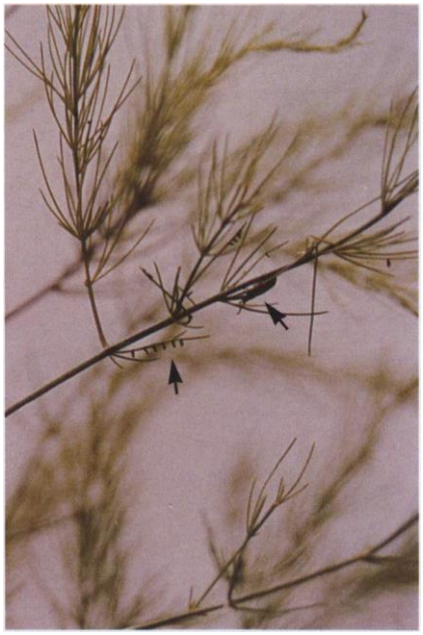
4.9 Asparagus aphid; nymphs.

4.10a Asparagus beetle; adult; length 6-7 mm.

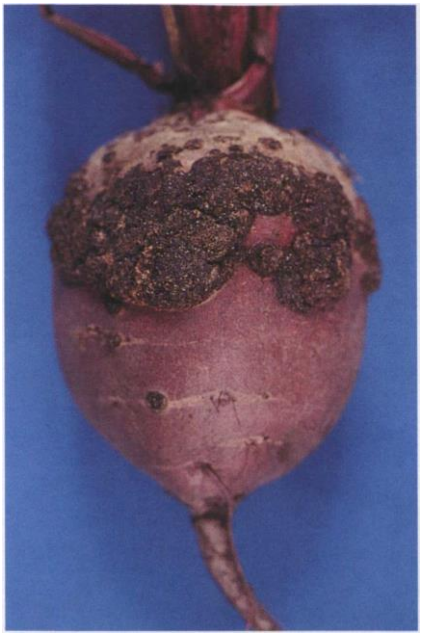
4.10b Asparagus beetle; larva.

4.10c Spotted asparagus beetle; adult; length 6-7 mm.

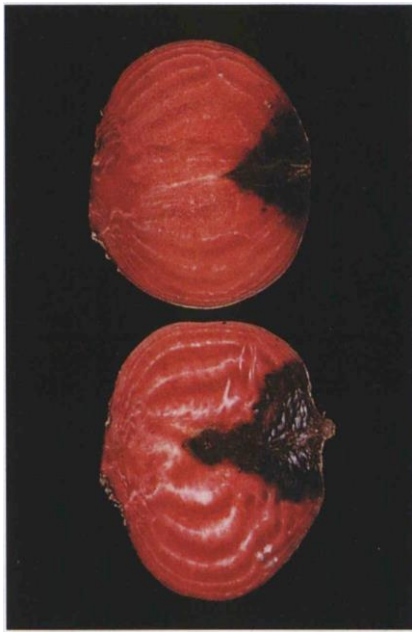
4.10d Spotted asparagus beetle; adults feeding on fern.



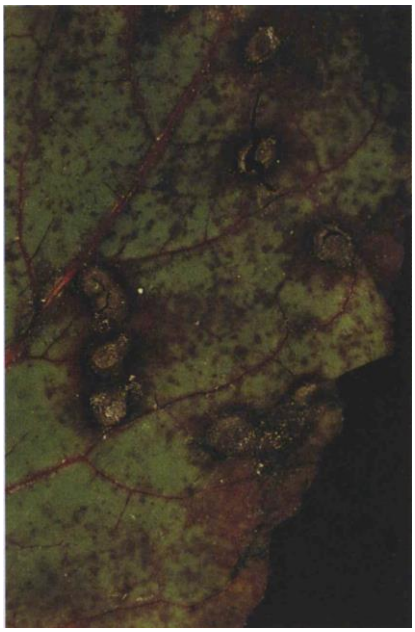
4.10e Asparagus beetle; adult and eggs on fern.



5.1 Scab; raised, corky lesions on a red beet root.



5.2 Aphanomyces root rot; red beet root with black decay progressing inward from the tip.



5.3 Cercospora leaf spot; lesions with purplish halo and brittle, gray centers.

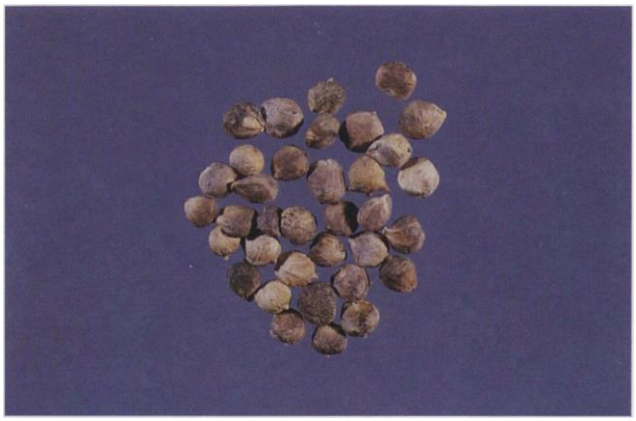
5.4a Downy mildew; severely affected spinach.

5.4b Downy mildew; chlorotic lesion (below), purple sporulation on lower surface of leaf.

5.5a Fusarium wilt; discoloration of spinach root.

5.5b Fusarium wilt; range of symptoms on spinach seedlings.

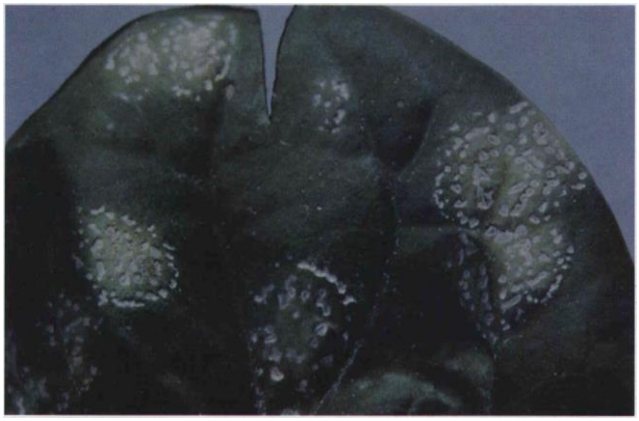
5.5c Fusarium wilt; diseased (left) and healthy spinach plants.



5.5d Fusarium wilt; discolored spinach seed infested with *Fusarium oxysporum* f. sp. *spinaciae.*

5.8a Rhizoctonia root rot; wilting and necrosis (browning) of foliage of red beet.

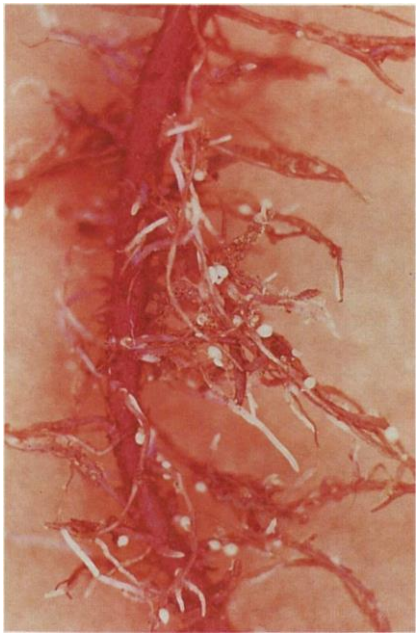
5.8b Rhizoctonia root rot; dry rot lesions on red beet root.



5.9 White rust; sori (white pustules) on a spinach leaf.

5.10 Spinach blight; yellowing, mottling and stunting of red beet seedlings.

5.14a Sugarbeet cyst nematode; infested (foreground) and healthy sugar beet crop.



5.14b Sugarbeet cyst nematode; red table beet roots with white immature females.

5.15 Beet leafhopper; adult; length ±3 mm.

5.16 Redheaded fleabeetle; adult; length 4-5 mm.

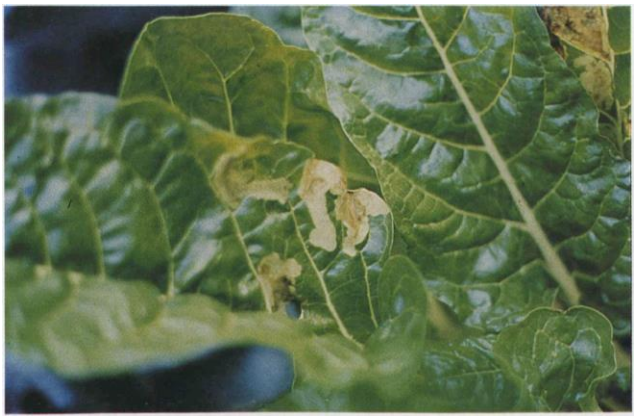
5.17a Beet webworm; larvae.

5.17b Beet webworm; left to right: spin-up, opened spin-up, pupa.

5.17c Beet webworm; adult moth; wingspan ± 25 mm.

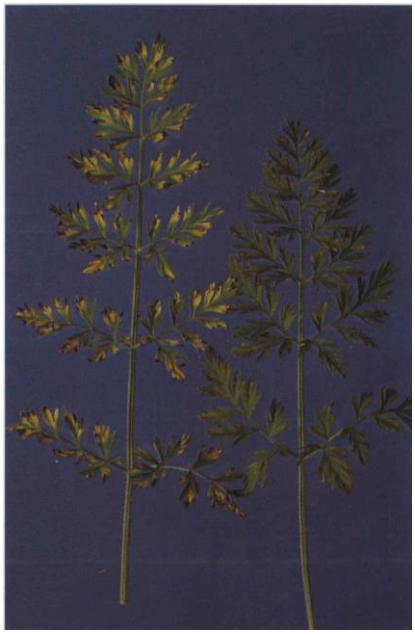
5.17d Beet leafminer; adult fly; length 6.0-6.5 mm.

5.17e Leafminer; damaged (mined) beet leaves.



5.17f Leafminer; damage to Swiss chard leaf.

6.1a Bacterial leaf blight; droplets of bacterial exudate on carrot umbel.

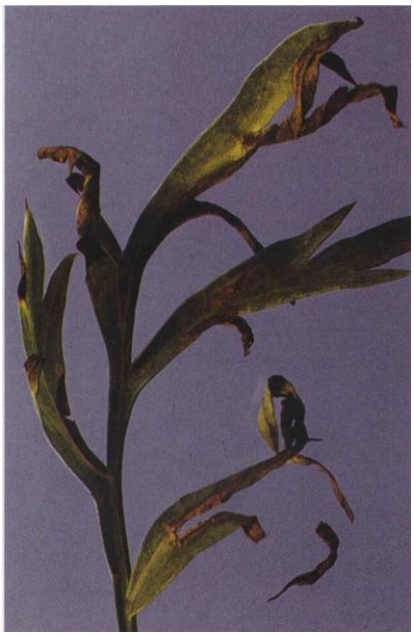


6.1b Bacterial leaf blight; diseased (left) and healthy carrot leaves.

6.2 Bacterial soft rot; internal decay of carrot roots.

6.3 Crown gall; roots with prominent galls.

6.4 Scab; lesions on root.



6.5a Alternaria leaf blight; leaf symptoms.

6.5b Alternaria leaf blight; conidium of *Alternaria dauci.*

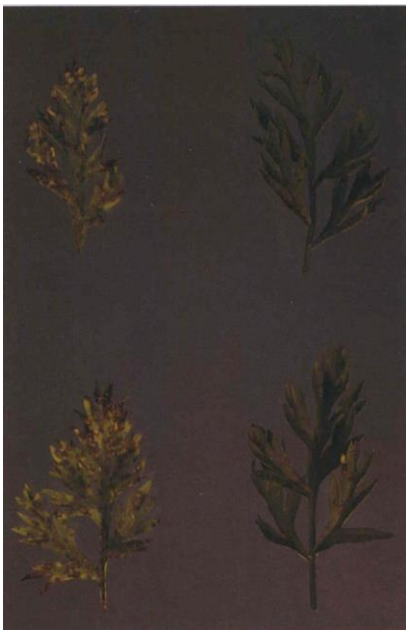
6.6a Black root rot; early symptoms.

6.6b Black root rot; advanced symptoms.

6.6c Black root rot; chlamydospores (large) and endoconidia (small) of *Chalara elegans.*

6.7 Black rot; greenish-gray mat of *Alternaria radicina.*

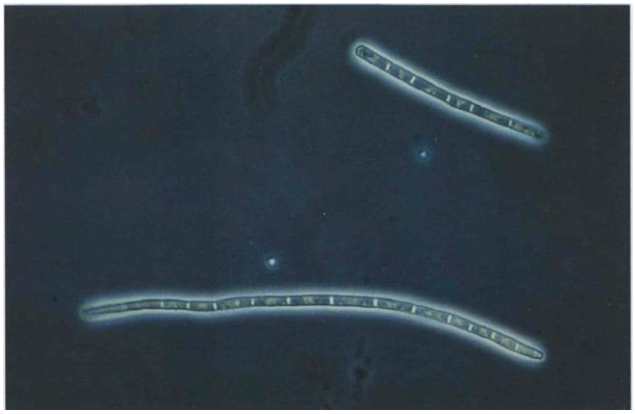
6.8 Cavity spot; root symptoms.



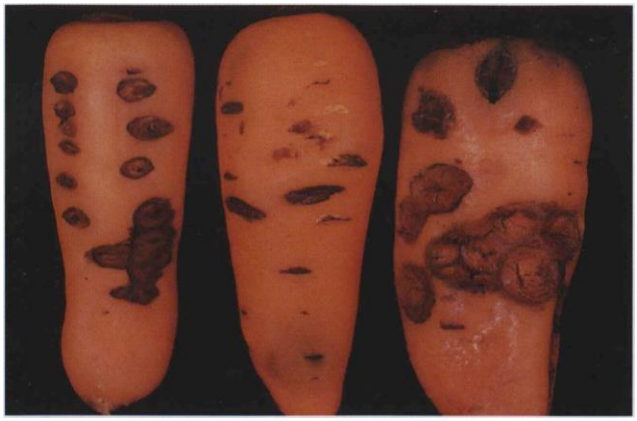
6.9a Cercospora leaf blight; lesions on leaflets.

6.9b Cercospora leaf blight; petiole lesions.

6.9c Cercospora leaf blight; coalesced lesions give the foliage a blighted appearance.



6.9d Cercospora leaf blight; conidia of *Cercospora carotae.*

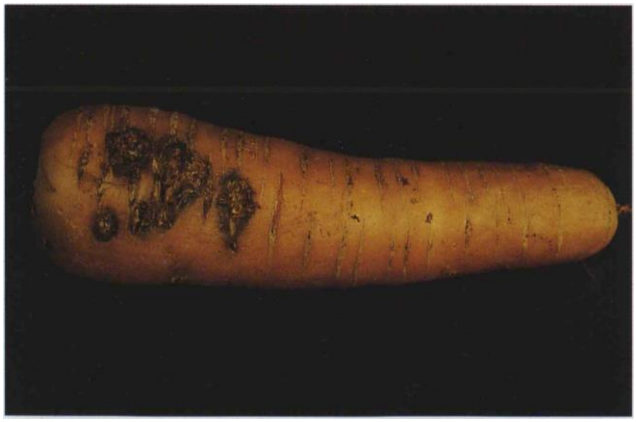


6.10 Crater rot; sunken lesions on roots.

6.11a Crown rot; healthy (bottom) and diseased roots.

6.11b Crown rot; note severed crown (at left), soil adhering to affected root, and cankers at points of lateral root emergence.

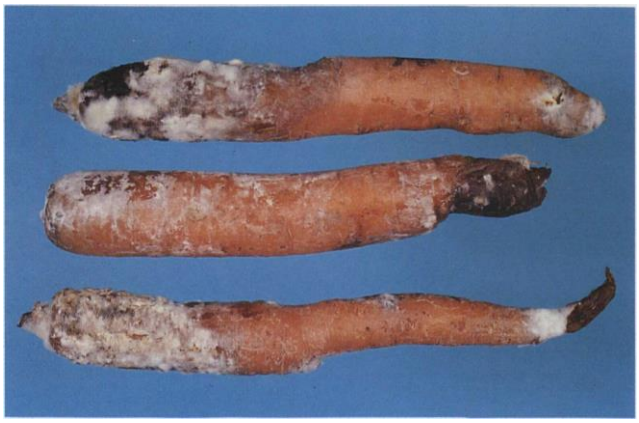
6.11c Crown rot; severe infection causes tops to wilt and die.



6.12 Fusarium dry rot; lesions on a carrot root.

6.13a Pythium root dieback; note discoloration and reduced size of the affected root (right).

6.13b Pythium root dieback; taproot forking symptom.



6.14 Rubbery brown rot; diseased roots from storage.

6.15a Sclerotinia rot; mycelial growth on stored roots.

6.15b Sclerotinia rot; white mycelium and black sclerotia of *Sclerotinia sclerotiorum* on roots.

6.16 Violet root rot; purple-brown net of mycelium and spores on affected roots.

6.17a Aster yellows; bronzing and yellowing of affected leaves.

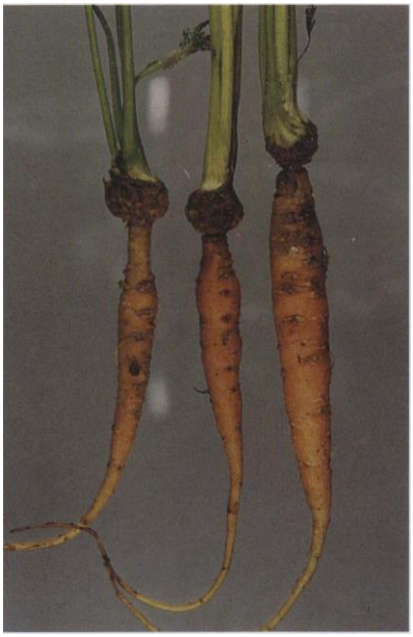
6.17b Aster yellows; affected plants (bronzed foliage) in a field.

6.17c Aster yellows; proliferation of lateral roots on infected taproots; deformed foliage (above).

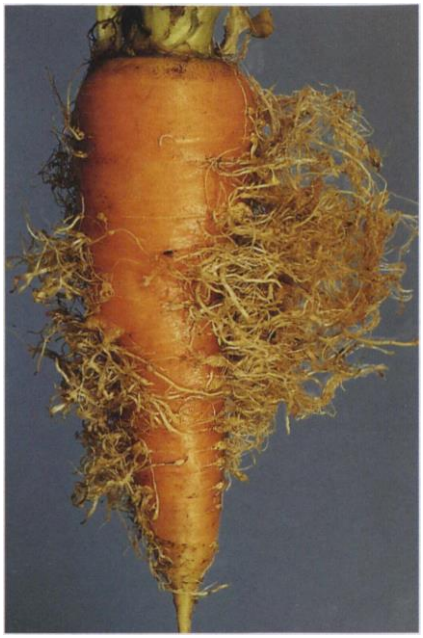
6.18 Growth cracks; longitudinal cracks on roots; healthy (left).

6.19a Heat canker; collapsed and dying seedlings.

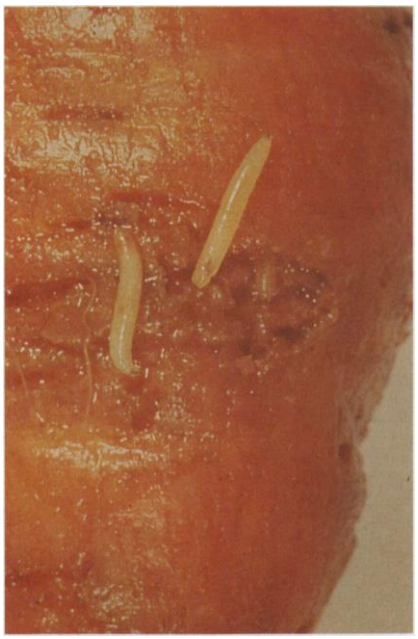
6.19b Heat canker; note constriction and severing of the tap root at the crown.



6.19c Heat canker; note constriction of the tap root just below the crown.



6.20 Northern root-knot nematode; galls and extensive lateral root proliferation.



6.23a Carrot rust fly; larvae and feeding injury on a carrot root.

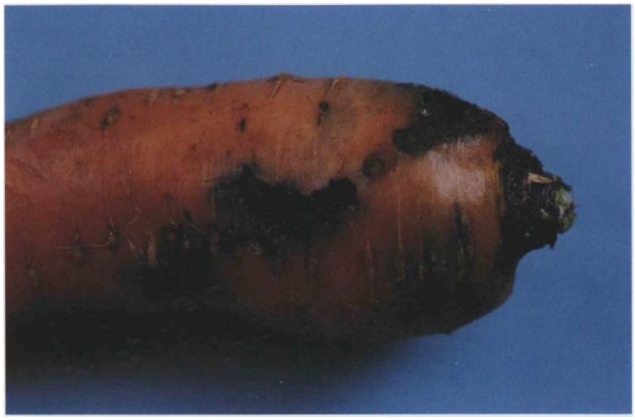
6.23b Carrot rust fly; larval feeding damage to carrot roots.

6.23c Carrot rust fly; pupae; length ±4.5 mm.

6.23d Carrot rust fly; older larvae affect the lower third of carrot roots.

6.23e Carrot rust fly; adult; length ±6 mm.

6.24a Carrot weevil; feeding damage to carrot roots.



6.24b Carrot weevil; feeding injury to a carrot root.

6.24c Carrot weevil; adult; length ±7 mm.

6.24d Carrot weevil; larva (left) and pupa.

6.25a Black cutworm; larva.

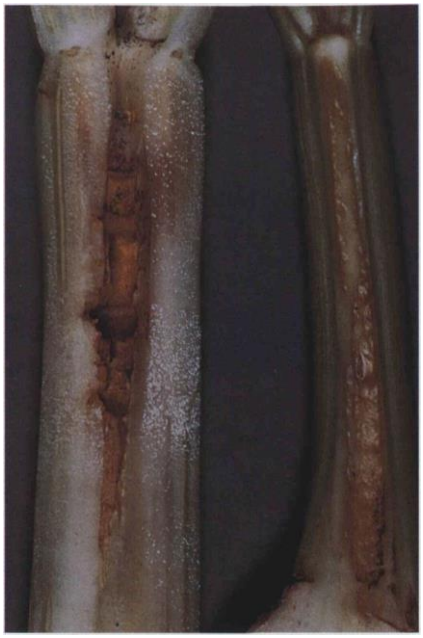
6.25b Dark-sided cutworm; larva.

6.25c redbacked cutworm; larva.

6.26 White grubs; larvae.

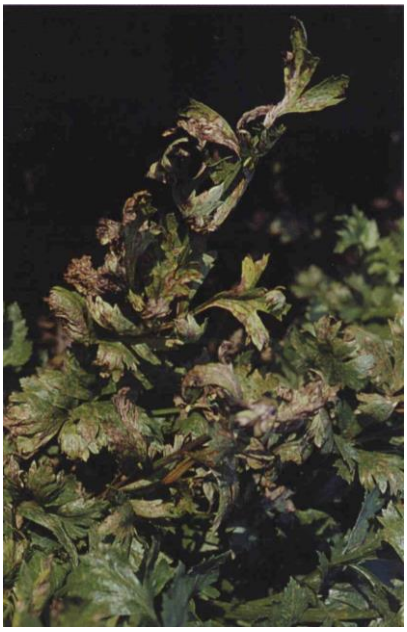
7.1a Bacterial leaf spot; rusty brown lesions with halos.

7.1b Bacterial leaf spot; leaf lesions and chlorosis.

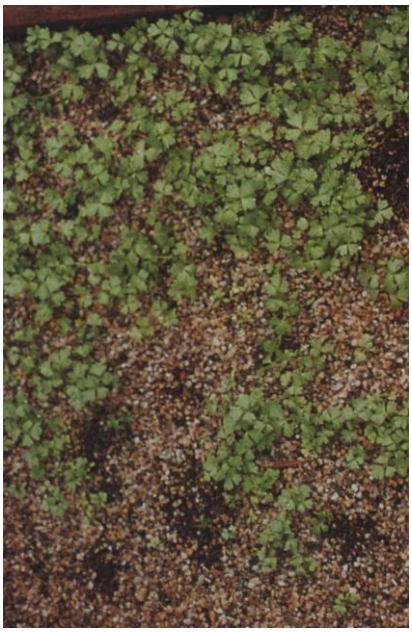


7.2 Brown spot; lesions on the inner surface of petioles.

7.3a Cercospora blight; lesions on celery leaves.



7.3b Cercospora blight; moderately severe symptoms on celery.



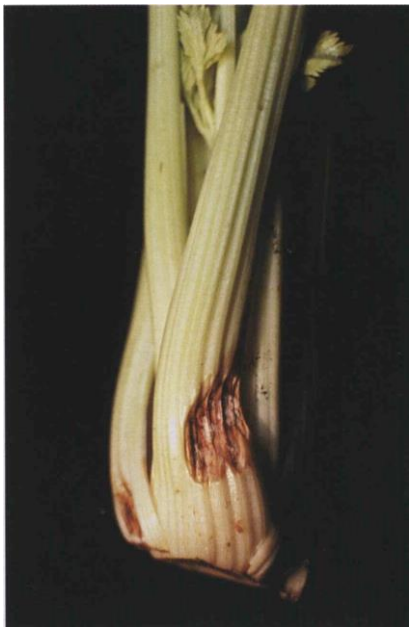
7.4 Damping-off; note missing plants at front of flat.

7.5a Fusarium yellows; chlorosis of outer leaves of celery.

7.5b Fusarium yellows; leaf dieback on celery plants.

7.5c Fusarium yellows; reddish-brown discoloration of internal crown tissue.

7.6a Pink rot; lesions on celery petioles.



7.6b Pink rot; lesion showing characteristic pinkish color.

7.7a Septoria blight; lesions on celery foliage.

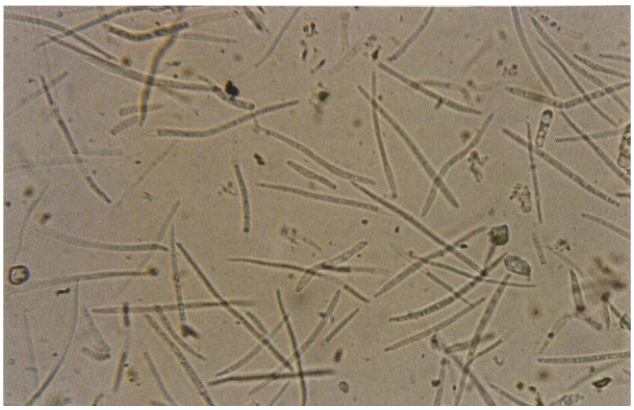
7.7b Septoria blight; coalescing lesions on underside of leaflet.

7.7c Septoria blight; lesions on celery petioles.

7.7d Septoria blight; pycnidia in lesions on celery petioles.

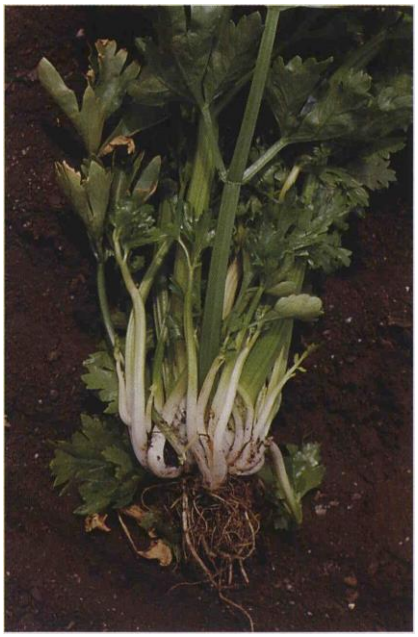
7.7e Septoria blight; pycnidia in lesion (enlarged).

7.7f Septoria blight; pycnidia with cirrhi of spores.



7.7g Septoria blight; conidia of *Septoria apiicola.*

7.8a Aster yellows; foliar symptoms on celery.



7.8b Aster yellows; proliferation of shoots at the crown.

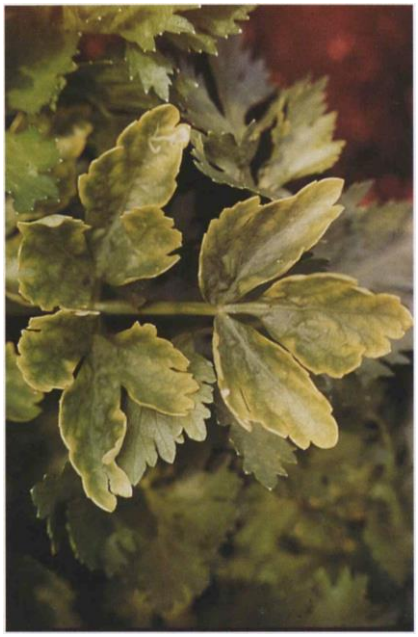
7.9a Heart mosaic; characteristic elongate, brownish spots on celery petioles.

7.9b Heart mosaic; mosaic and crinkling symptoms on a celery leaf.

7.9c Heart mosaic; necrosis of celery petioles.

7.10a Blackheart; affected celery plant.

7.10b Blackheart; leaf symptoms on celery.



7.11 Chlorosis; magnesium deficiency symptoms on a celery leaf.

7.12a Cracked stem; symptoms on celery.

7.12b Cracked stem; internal breakdown in celeriac.

7.13 Spongy petiole; affected celery petioles.

7.15a Northern root-knot nematode; stunted celery plant with galls on roots.

7.15b Northern root-knot nematode; galls on roots.

7.20 Carrot weevil; severe root and crown injury to a celery plant.

7.21a Tarnished plant bug; feeding injury on a celery petiole.

7.21b Tarnished plant bug; a feeding nymph.

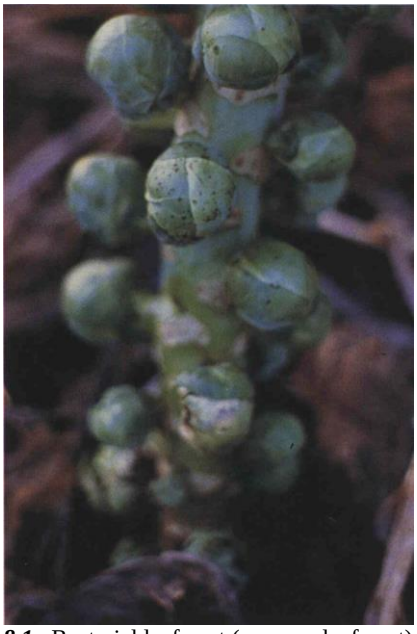
7.21c Tarnished plant bug; sequence of inner petioles showing breakdown from feeding injury.

7.21d Tarnished plant bug; adult (right), length 5-6 mm; and young nymph.

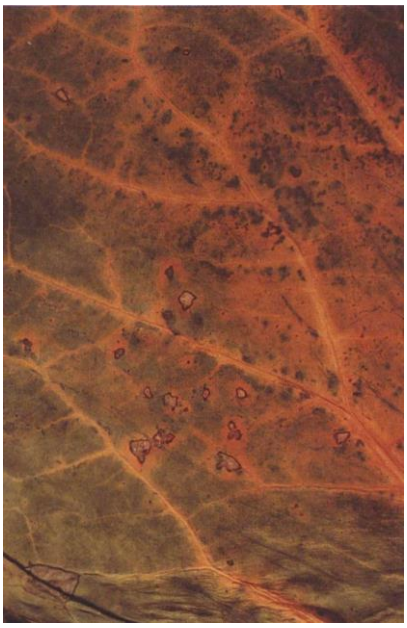
7.21e Tarnished plant bug; adult, pale specimen; length 5-6 mm.

7.22a Celery looper; lateral line narrower than in cabbage looper; actual size 28 mm.

7.22b Celery stalkworm; larva; actual size 25 mm.



8.1a Bacterial leaf spot (peppery leaf spot); lesions on Brussels sprouts.



8.1b Bacterial leaf spot (peppery leaf spot); lesions on a cauliflower leaf.

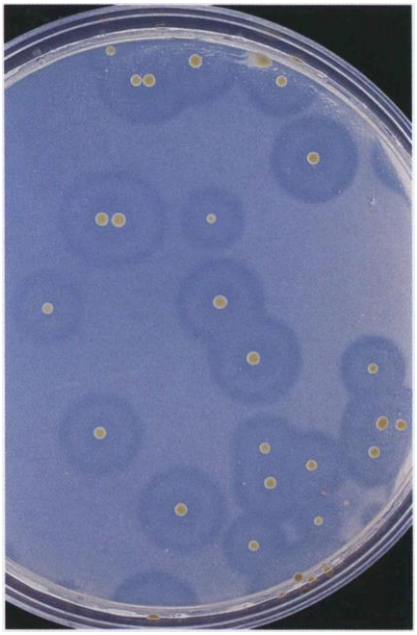
8.2a Black rot; leaf symptoms (V-shaped, yellow lesions) on cabbage.

8.2b Black rot; advanced (dry) lesions along leaf margins of cabbage.

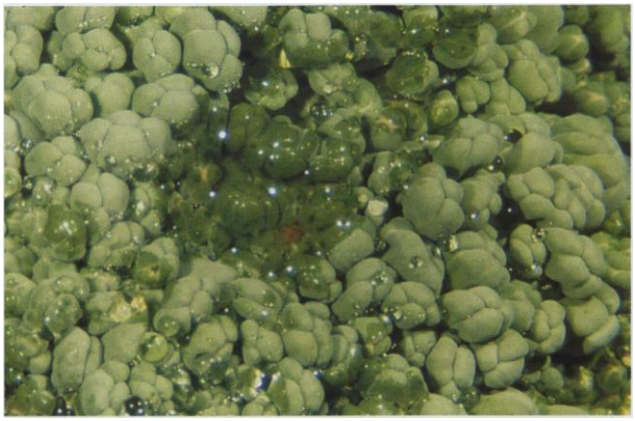
8.2c Black rot; bacteria enter leaf in guttation droplets from hydathodes.

8.2d Black rot; blackened veins on a broccoli leaf.

8.2e Black rot; discoloration of vascular tissues in rutabaga stem.



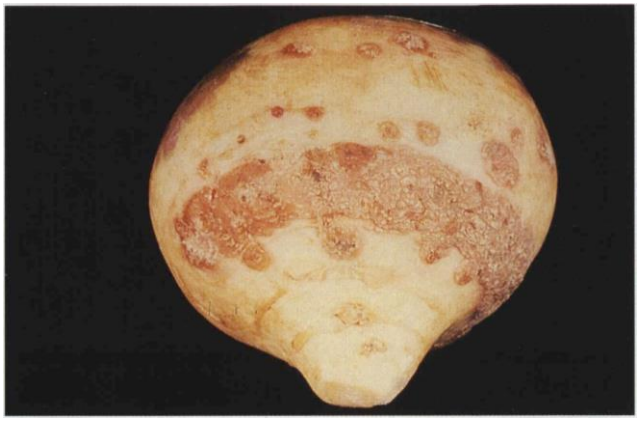
8.2f Black rot: yellow colonies of *Xanthomonas campestris* from seed wash.



8.3a Head rot; early symptoms on broccoli; note water film on waxy florets from surfactant activity of bacteria.

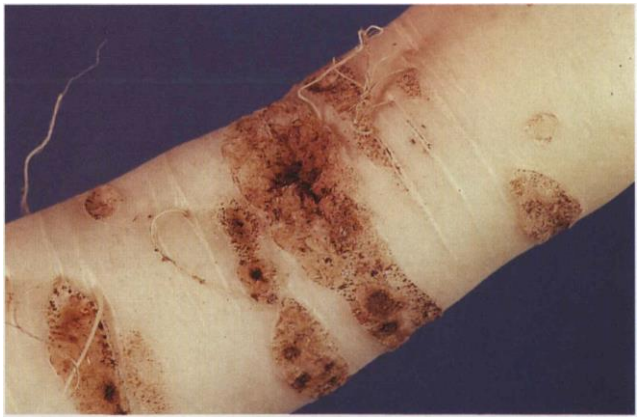
8.3b Head rot; advanced decay on broccoli head.

8.3c Head rot; severe symptoms on cabbage.



8.4a Scab; corky lesions on rutabaga root.

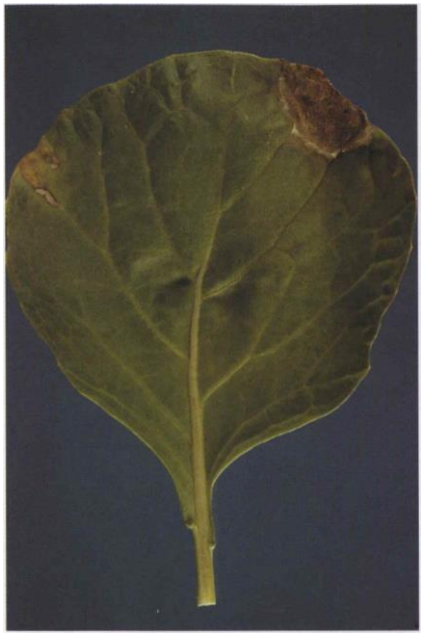
8.4b Scab; raised corky lesions on rutabaga roots.



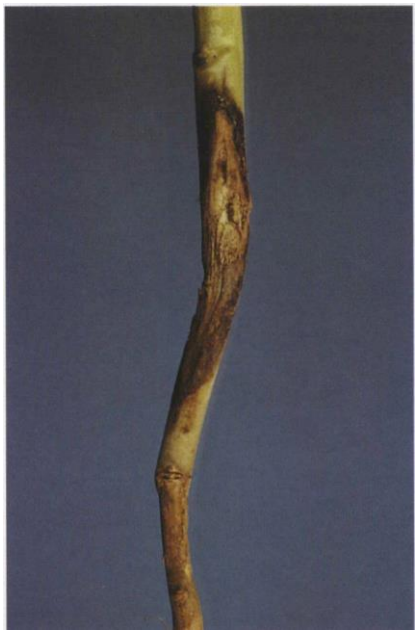
8.4c Scab; lesions on a white radish root.

8.5a Alternaria leaf spot; black spot symptoms on cauliflower foliage and head.

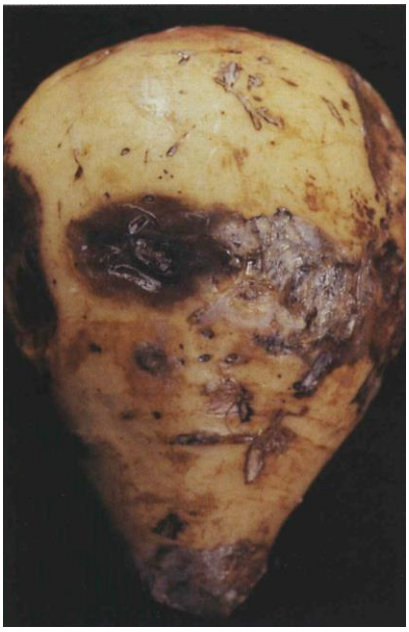
8.5b Alternaria leaf spot; gray spot symptoms on Brussels sprouts.



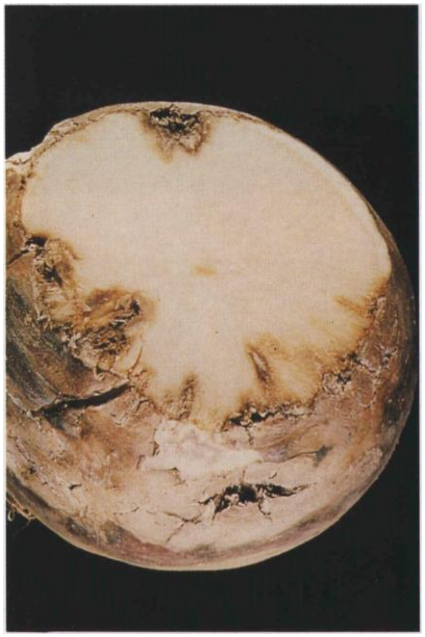
8.6a Blackleg; leaf lesions on cabbage.



8.6b Blackleg; stem lesion on cabbage.



8.6c Blackleg; brown lesions on rutabaga root.



8.6d Blackleg; cutaway rutabaga root showing penetrating lesions.

8.8a Clubroot; galled cabbage roots.

8.8b Clubroot; severely galled roots of broccoli.

8.7 Black root; decayed areas on radish root.

8.8c Clubroot; galls on tap and lateral roots of rutabaga.

8.9 Damping-off (rhizoctonia); wilted cabbage seedlings (foreground) in a propagation tray.

8.10a Downy mildew; diseased broccoli plants showing dying leaves.

8.10b Downy mildew; sporulation of *Peronospora parasitica* on a broccoli leaf.

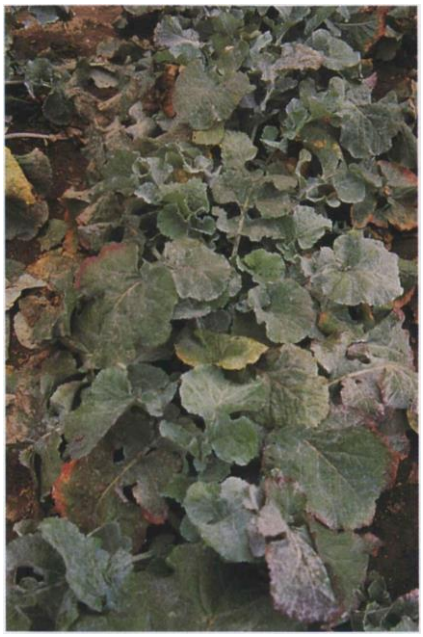
8.10c Downy mildew; lesions on a rutabaga leaf.

8.10d Downy mildew; brown lesions on cauliflower curd.

8.10e Downy mildew; black lesions on radish roots.

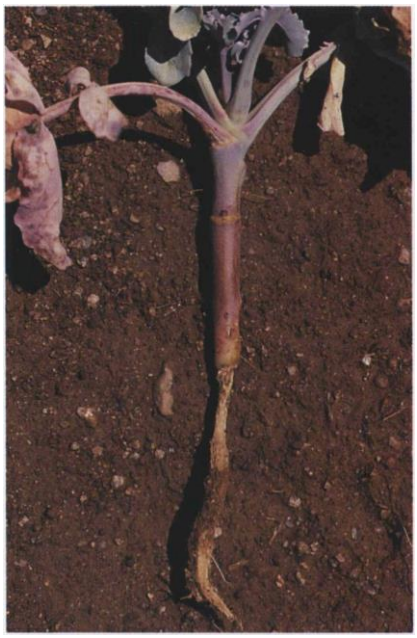
8.11a Fusarium wilt (yellows); diseased cabbage plant.

8.11b Fusarium wilt (yellows); discoloration of xylem in infected stem (cutaway) of a cabbage plant.

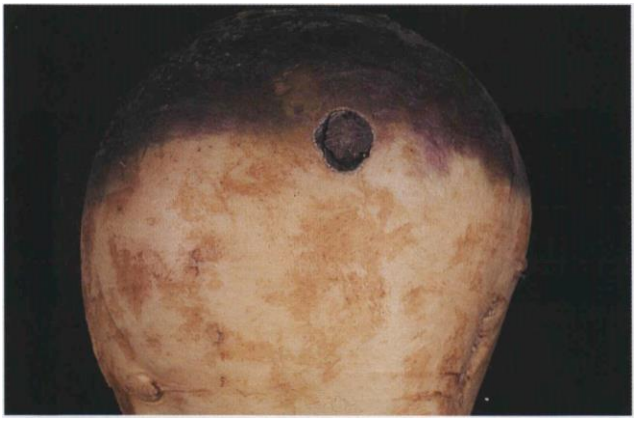


8.12 Powdery mildew; affected rutabaga plants.

8.13a Rhizoctonia wirestem; constriction of cauliflower stem.



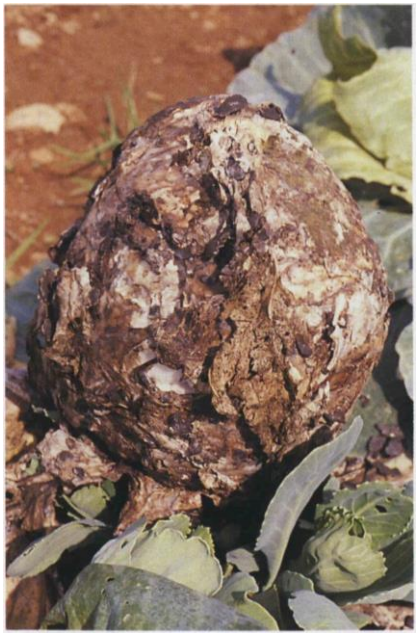
8.13b Rhizoctonia wirestem; soil line canker on stem of a young broccoli plant.



8.13c Rhizoctonia root rot (crater rot); lesion on a rutabaga root.

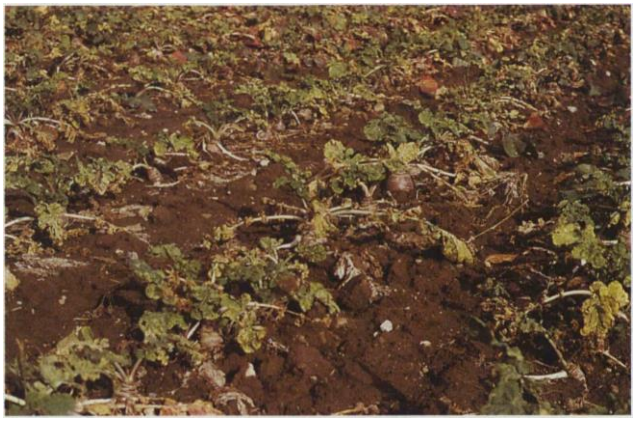
8.13d Rhizoctonia root rot; dark lesion with secondary bacterial decay (upper lesion) on rutabaga root.

8.13e Rhizoctonia head rot; dark brown, firm decay, with small sclerotia (bottom).



8.14 Sclerotinia rot; white, cottony mycelium and black sclerotia.

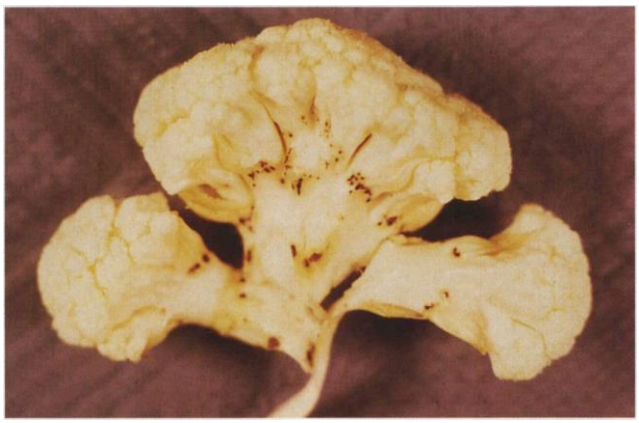
8.15 White rust; sori (white lesions) on a horseradish leaf.



8.16a Turnip mosaic; severely affected rutabaga plants.

8.16b Turnip mosaic; characteristic foliar mottling and wrinkling in rutabaga.

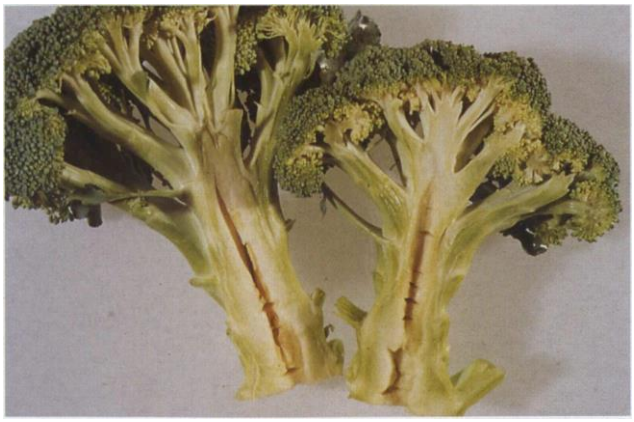
8.16c Turnip mosaic; healthy (left) and diseased rutabaga roots.



8.17 Black speck; symptoms on a cauliflower curd.

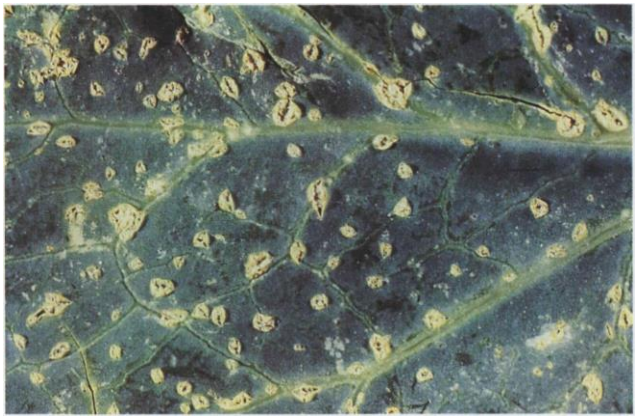
8.18 Brown bead; discolored florets on a broccoli head.

8.19 Growth cracks; cracks in rutabaga roots are frequently invaded by soft rot bacteria.



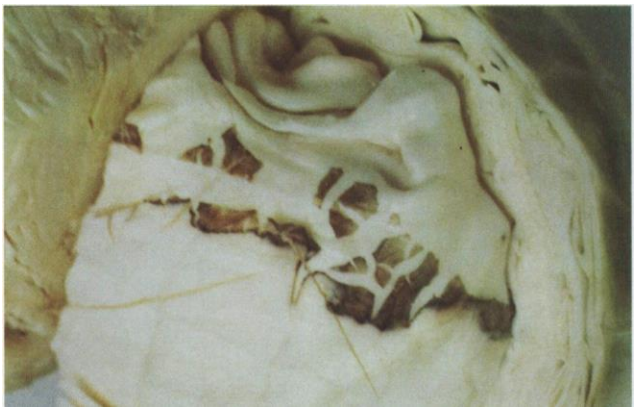
8.20 Hollow stem; elliptical cavities and discoloration in broccoli stems.

8.21a Intumescence; small, wart-like protuberances on cabbage leaves.



8.21b Intumescence (edema); lesions on a cabbage leaf.

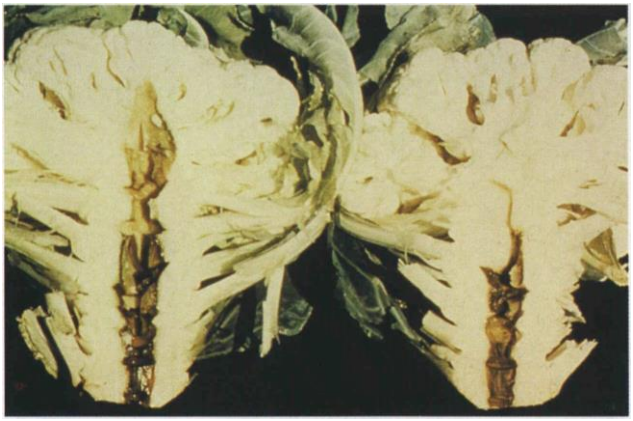
8.21c Intumescence (thrips pustule); damage from thrips feeding on cabbage head.



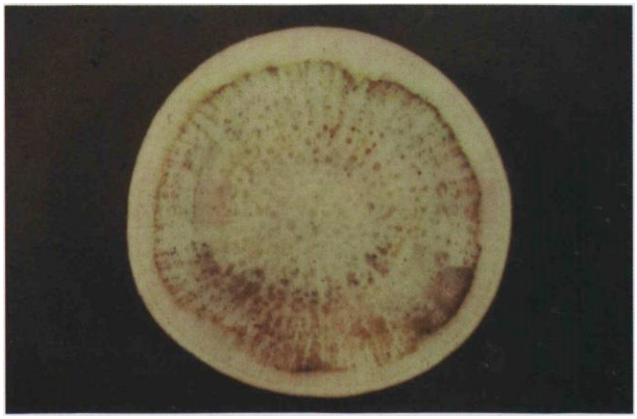
8.22a Tipburn; internal leaves of cabbage desiccate to a thin, papery consistency.

8.22b Tipburn; internal browning of Brussels sprouts.

8.23a Boron deficiency; stem cavities and curd discoloration on cauliflower.



8.23b Boron deficiency; brown discoloration of stem cavity on cauliflower.



8.23c Boron deficiency; water core symptom in a rutabaga root.

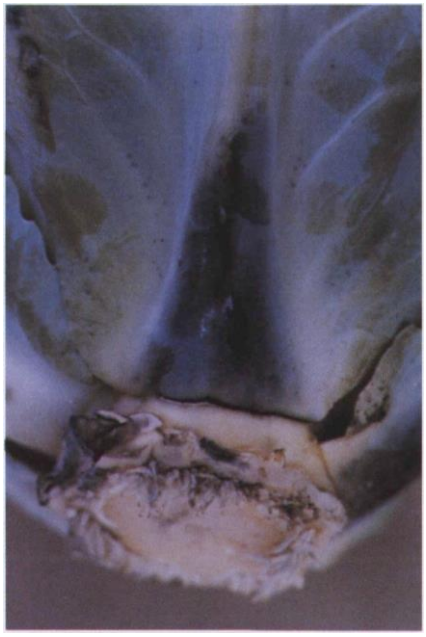
8.23d Boron deficiency; severe brown heart symptom in a rutabaga root.

8.24 Magnesium deficiency; leaf chlorosis of broccoli.

8.25a Molybdenum deficiency; whiptail symptom on cauliflower leaf.

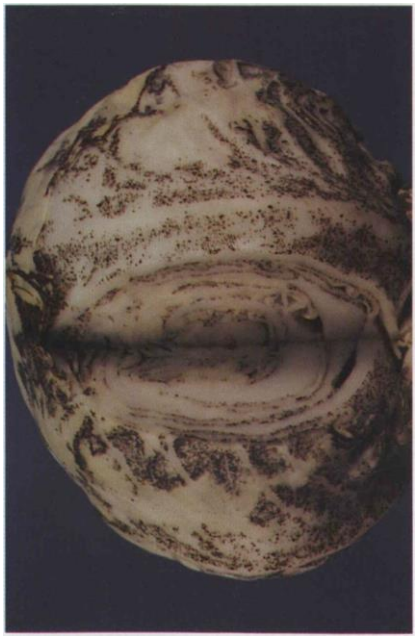
8.25b Molybdenum deficiency; cupped leaves and blotchy chlorosis and necrosis on cabbage.

8.26 Sulfur deficiency; blotchy interveinal chlorosis and reflexed leaves of cauliflower.

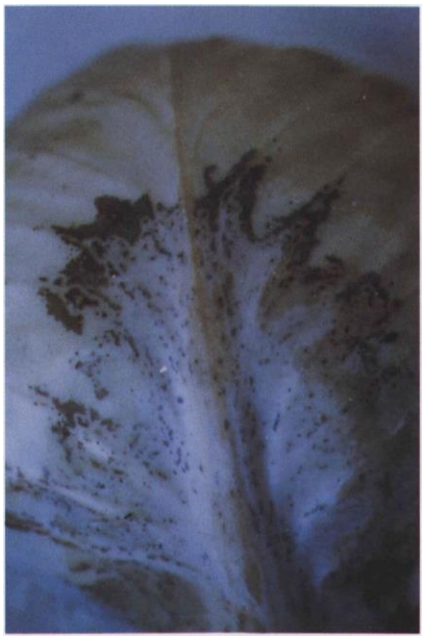


8.27 Black midrib; large, ill-defined spots at base of midrib on outer leaves of cabbage.

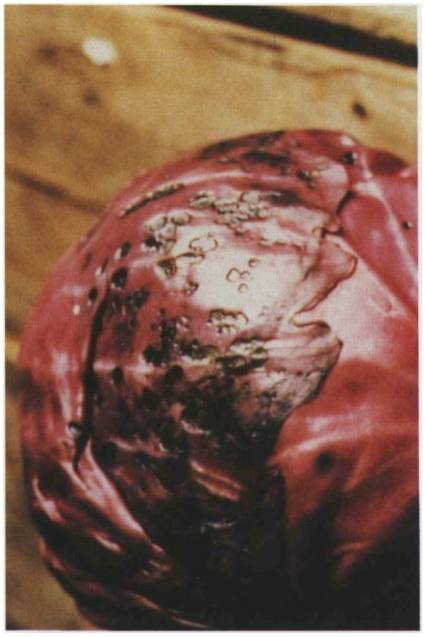
8.28a Black speck (type I); sharply sunken, pin-point spots on outer leaves of cabbage .



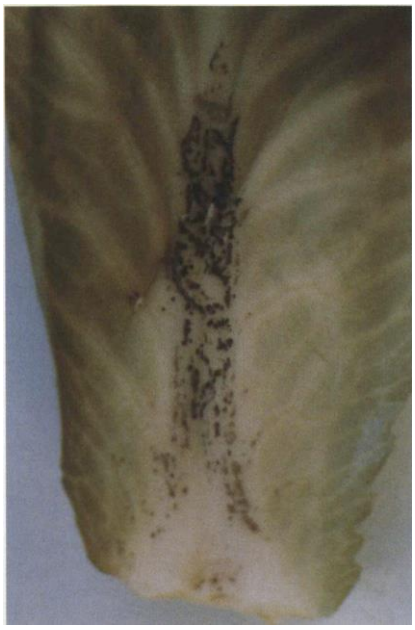
8.28b Black speck (type II, senescent black speck); a severely affected cabbage head .



8.29 Gray speck; grayish discoloration at the base of a cabbage leaf.

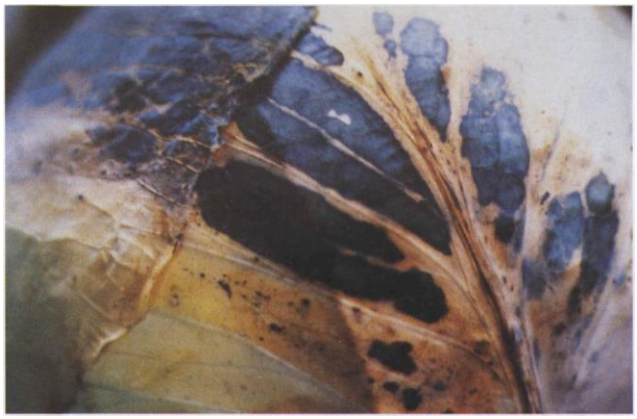


8.30 Necrotic spot (type I); dark, sharply defined, sunken lesions on cabbage.



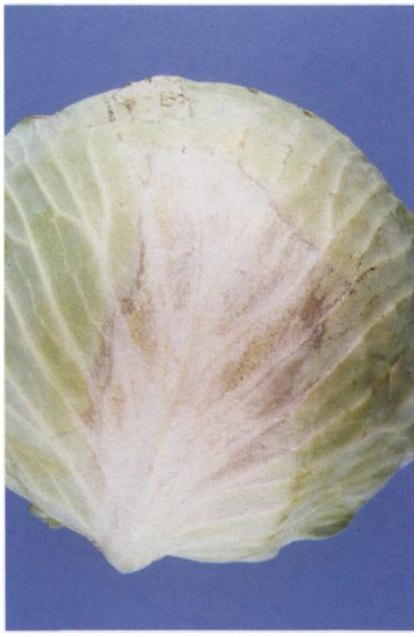
8.31 Vein streaking; superficial brown-black lesions on midrib of cabbage leaf.

8.32a Black blotching; frost induced, superficial spots with pin-point dark centers.

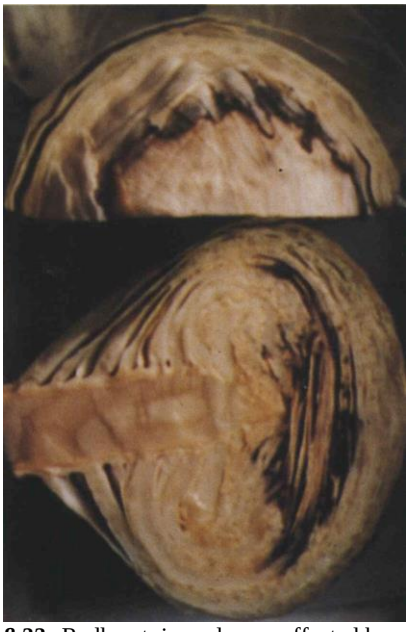


8.32b Black spot; frost-induced areas of interveinal necrosis on cabbage; appears in storage.

8.32c Epidermal detachment; loosened epidermis on top leaves of cabbage from repeated freezing in the field.



8.32d Frost blemishing; large, white blemishes on exposed head leaves of cabbage.



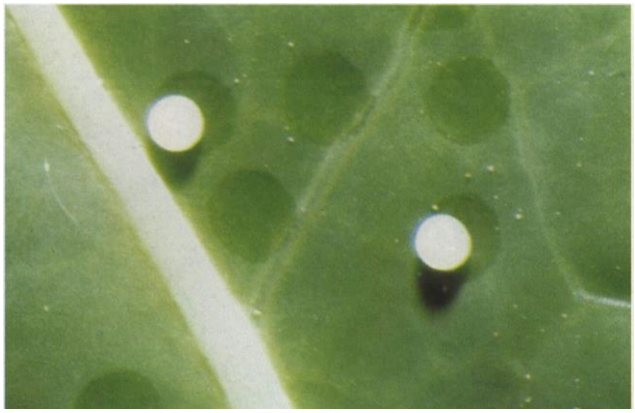
8.32e Redheart; inner leaves affected by extended period of freezing in field or storage.

8.38 Alfalfa looper; larva; note wide lateral line.

8.39a Cabbage aphid; infested Brussels sprouts.

8.39b Aphids; nymphs on cabbage.

8.40a Cabbage looper; injury to cabbage leaves.



8.40b Cabbage looper; eggs.

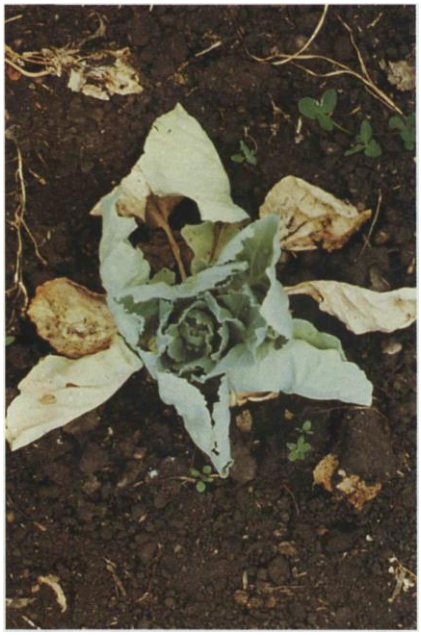
8.40c Cabbage looper; larva (top view).

8.40d Cabbage looper; larva (side view); note narrow lateral line.

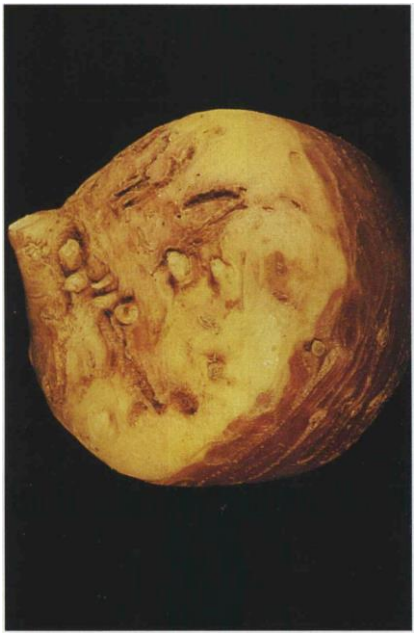
8.40e Cabbage looper; pupae.

8.40f Cabbage looper; adult moth; wingspan ± 38 mm.

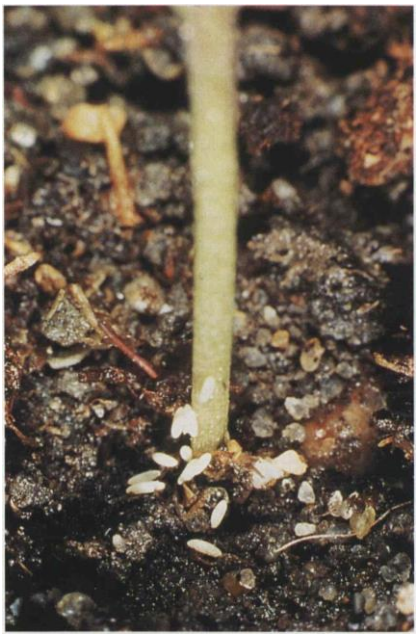
8.41a Cabbage maggot; larval feeding damage to radish root.



8.41b Cabbage maggot; above-ground symptoms on cabbage plant.



8.41c Cabbage maggot; larval feeding damage to rutabaga root.



8.41d Cabbage maggot; eggs at base of stem.

8.41e Cabbage maggot; larvae.

8.41f Cabbage maggot; pupae.

8.41g Cabbage maggot; adult flies.

8.42a Diamondback moth; larval feeding injury to cabbage leaves.

8.42b Diamondback moth; eggs and larval silk strands on leaf.

8.42c Diamondback moth; larva at leafmining stage.

8.42d Diamondback moth; intermediate and mature larvae.

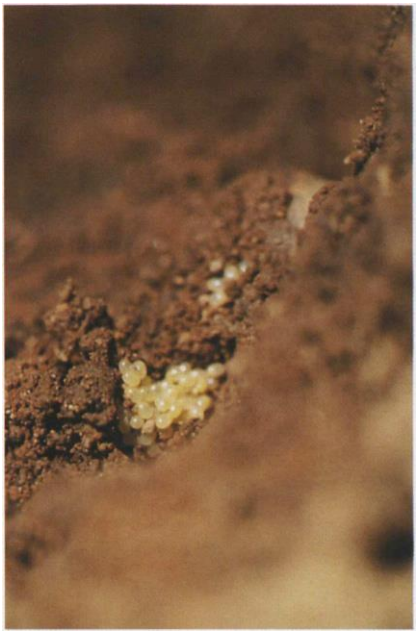
8.42e Diamondback moth; cocoon and pupa.

8.42f Diamondback moth; adult moth; wingspan ± 13 mm.

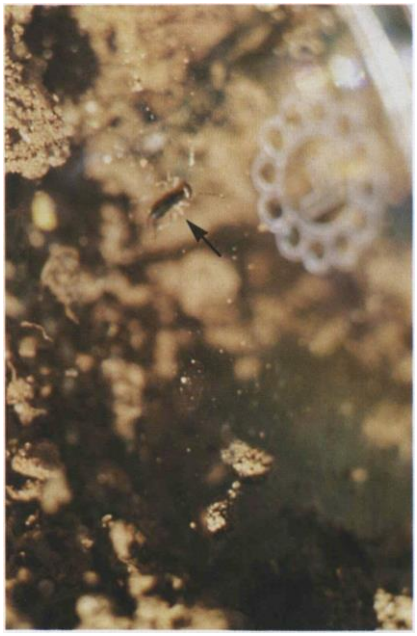
8.42g Diamondback moth; adult moth; note the pattern on its back.

8.43a European earwig; damage to cabbage head; note adult earwig and frass (black dots) on midrib.

8.43b European earwig; adults, male (left) and female; length 11-18 mm.



8.43c European earwig; eggs in soil.



8.43d European earwig; first instar nymph.

8.44a Crucifer flea beetle; adults on a broccoli cotyledon.

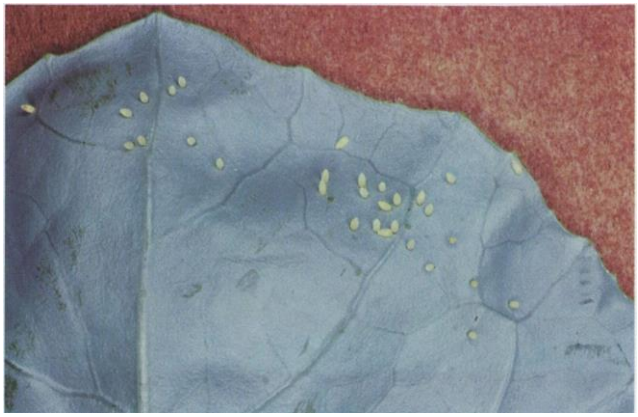
8.44b Striped flea beetle; adult; length ± 2 mm.

8.44c Crucifer flea beetle; adults on rutabaga leaf.

8.44d Crucifer flea beetle; adult feeding damage on cabbage leaves.

8.44e Crucifer flea beetle; late-season adult feeding causes cosmetic damage to broccoli head.

8.45a Imported cabbageworm; larval feeding damage to cabbage leaves.

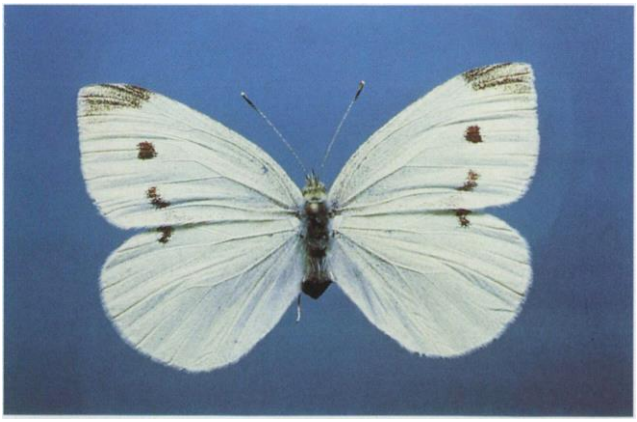


8.45b Imported cabbageworm; eggs on cabbage leaf.

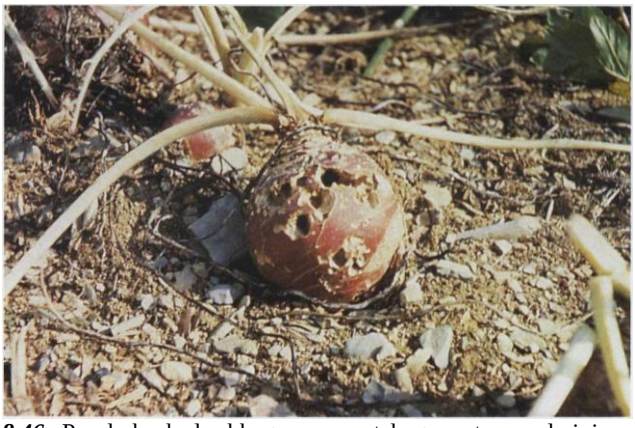
8.45c Imported cabbageworm; egg and newly hatched larva.

8.45d Imported cabbageworm; near-mature larva.

8.45e Imported cabbageworm; pupa, characteristically on underside of leaf.

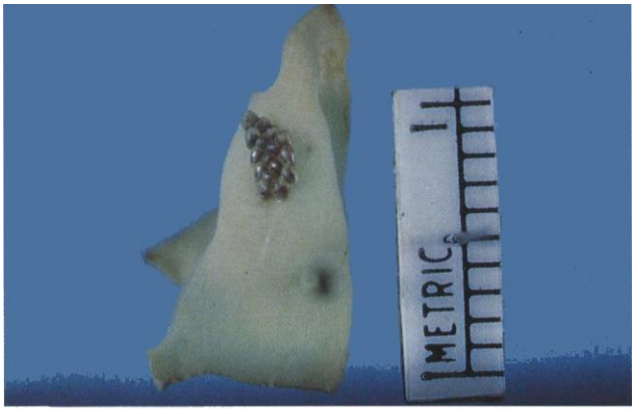


8.45f Imported cabbageworm; adult female butterfly; wingspan ±50 mm.



8.46a Purple-backed cabbageworm; rutabaga root severely injured by larval feeding.

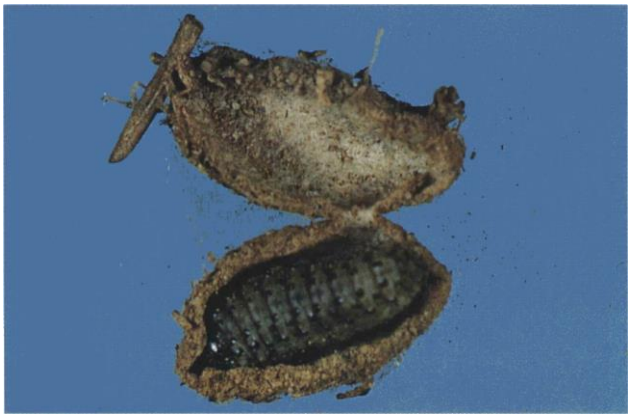
8.46b Purple-backed cabbageworm; cabbage leaves and head severely injured by larval feeding.



8.46c Purple-backed cabbage worm; egg mass on cabbage leaf.

8.46d Purple-backed cabbageworm; full-grown larva.

8.46e Purple-backed cabbageworm; pupa within cocoon.



8.46f Purple-backed cabbageworm; open cocoon showing the overwintering larva (contracted).

8.46g Purple-backed cabbageworm; adult moth; wingspan 22-28 mm.

8.47a Red turnip beetle; adult; length ± 10 mm.

8.47b Red turnip beetle; larva feeding on a leaf.

8.47c Red turnip beetle; eggs (left) and pupae (right) on soil.

9.1a Angular leaf spot; moderately severe infection of cucumber.

9.1b Angular leaf spot; lesions and shot-holes on cucumber leaf.

9.2a Bacterial wilt; wilted cucumber plants.

9.2b Bacterial wilt; wilted cucumber vine.

9.3a Anthracnose; lesions on a muskmelon vine and leaves.

9.3b Anthracnose; extensive chlorosis and necrosis of cucumber foliage.

9.3c Anthracnose; lesions on muskmelon fruit.

9.5 Fusarium foot rot; severely affected muskmelon plant.

9.6a Fusarium wilt; affected (chlorotic) muskmelon plant.

9.6b Fusarium wilt; healthy (left and foreground) and diseased muskmelon plants.

9.7 Gray mold; sporulation of *Botrytis cinerea* on infected cucumber fruit.

9.8a Alternaria leaf blight; lesions on cucumber leaf.

9.8b Ulocladium leaf spot; an affected cucumber leaf.

9.10 Powdery mildew; whitish, talc-like lesions on squash foliage.

9.13 Scab; lesions on cucumber fruit; see also *22.16.*

9.14a White mold; stem rot of pumpkin.

9.14b White mold; cucumber fruit with mycelium and sclerotia of *Sclerotinia sclerotiorum.*

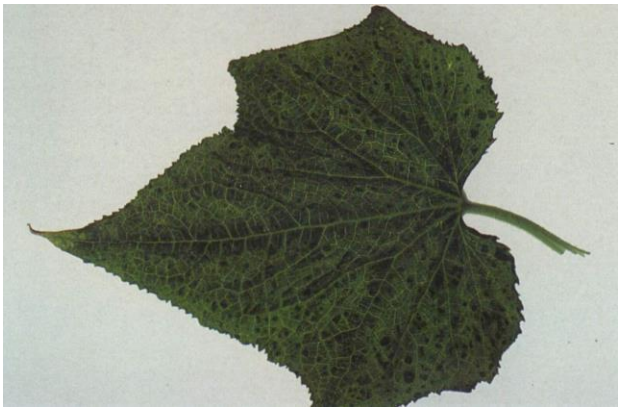
9.14c White mold; affected pumpkin fruit.

9.15 Cucumber mosaic; severe distortion and mosaic of zucchini leaves.

9.16a Zucchini yellow mosaic; distorted cucumber leaf.

9.16b Zucchini yellow mosaic; distorted cucumber fruit.

9.16c Zucchini yellow mosaic; slight malformation of squash infected by ZYMV.

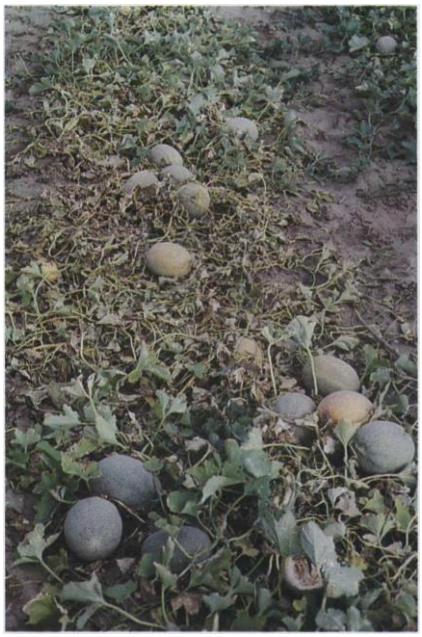


9.17a Watermelon mosaic; mosaic symptoms of strain 2 virus infection on cucumber.

9.17b Watermelon mosaic; prominent etching and upward cupping of cucumber leaves.

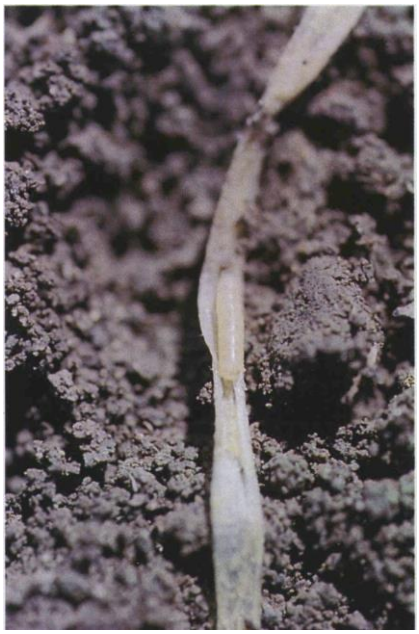
9.18a Cold injury; frost damage to a young cucumber plant.

9.18b Cold injury; necrosis on the cotyledons and leaves of a cucumber seedling.



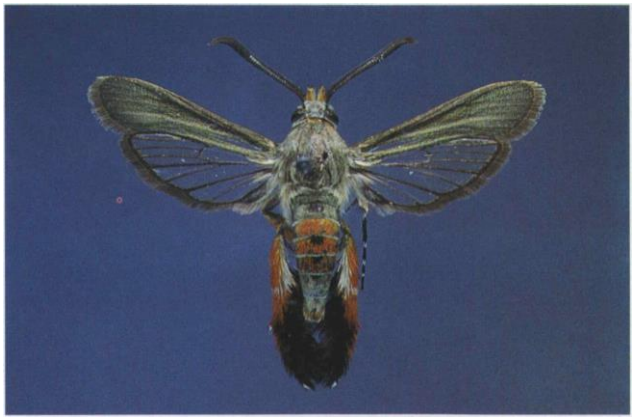
9.18c Cold injury; frost damage on melon plants.

9.21 Cucumber beetles; adults, spotted (top) and striped; length 6-7 mm.



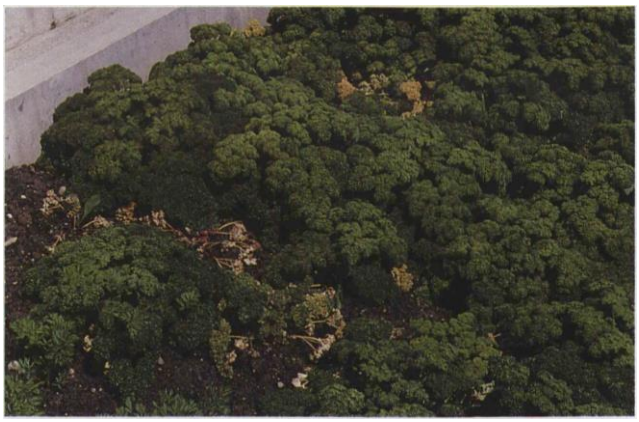
9.22a Seedcorn maggot; larva inside cucumber stem.

9.22b Seedcorn maggot; wilted cucumber seedling.



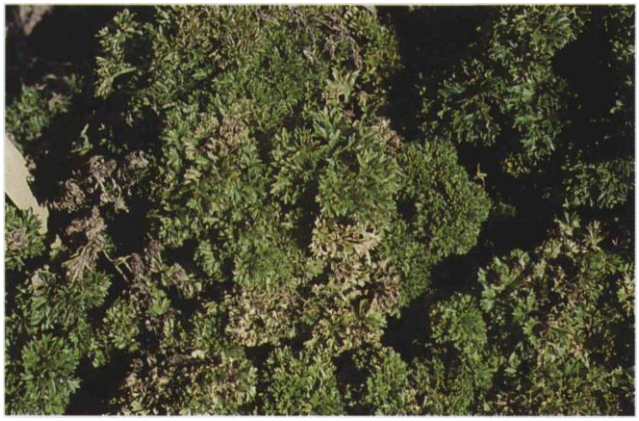
9.22c Squash vine borer; adult; wingspan 30-34 mm.

10.2 Downy mildew of hop; infected (chlorotic) shoots.



10.3a Leaf scorch of parsley; damping-off of scattered plants.

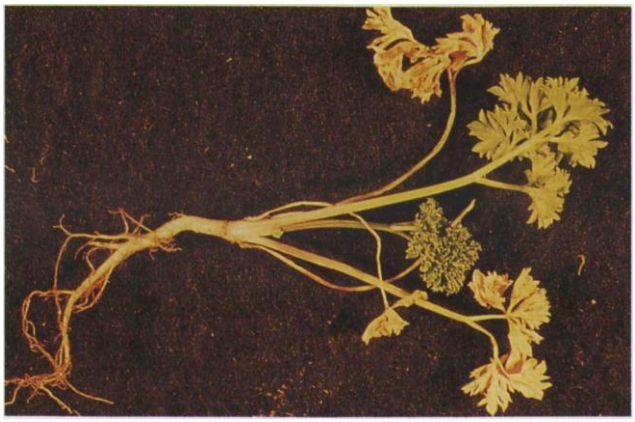
10.3b Leaf scorch of parsley; moderately affected plant.



10.4 Septoria leaf spot; parsley plants showing foliar chlorosis and necrosis.

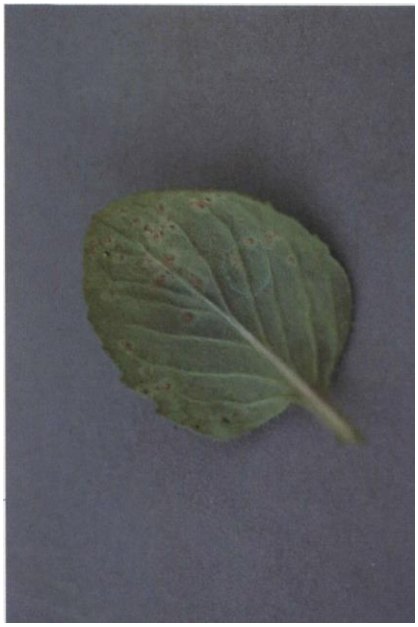
10.5 Powdery mildew; lesions on parsley leaf.

10.6a Pythium root rot; affected parsley plants showing yellowing of leaves.

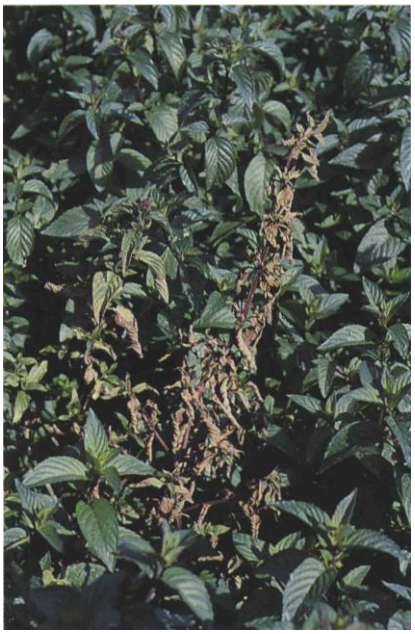


10.6b Pythium root rot; root rot of parsley.

10.7a Rust of mint; lesions on leaves of native spearmint.



10.7b Rust of mint; pustules on leaf of scotch spearmint.



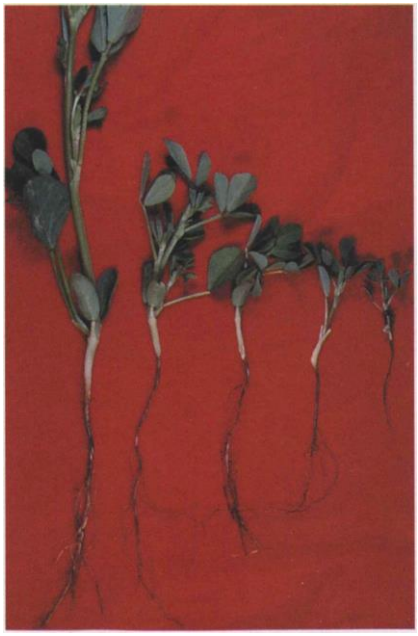
10.9a Verticillium wilt; severe wilt and necrosis of peppermint.

10.9b Verticillium wilt; wilted shoots of hop.

10.9c Verticillium wilt; asymmetrical leaf growth of Scotch spearmint.

10.9d Verticillium wilt; chlorosis of young leaves of Scotch spearmint.

10.10a Phoma blight; lesions on dill umbel.



10.10b Thielaviopsis root rot; symptoms on fenugreek seedlings.

10.10c Root rot; dead tarragon plant.

10.11a Aster yellows; an affected (chlorotic) dill plant.

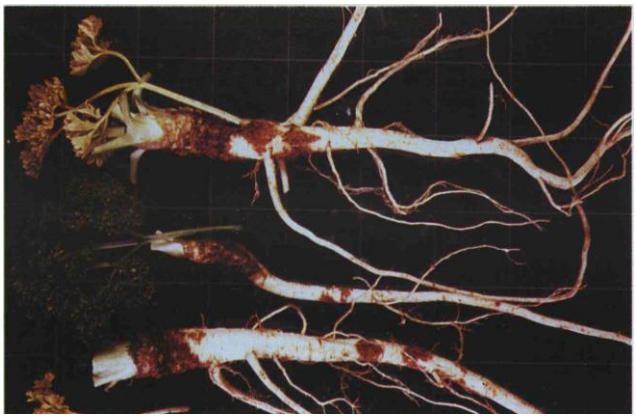
10.11b Aster yellows; dill umbel showing chlorotic, bunchy growth.

10.12 Carrot motley dwarf; severely affected parsley field.

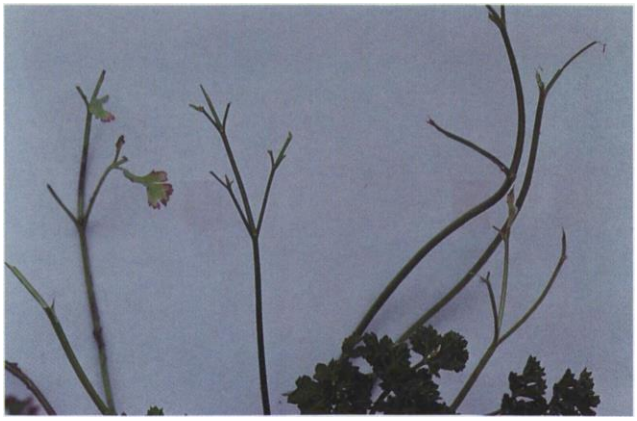
10.14a Crucifer flea beetle; adult.

10.14b Crucifer flea beetle; severe injury from adult feeding.

10.15a Black swallowtail; intermediate larvae on dill.

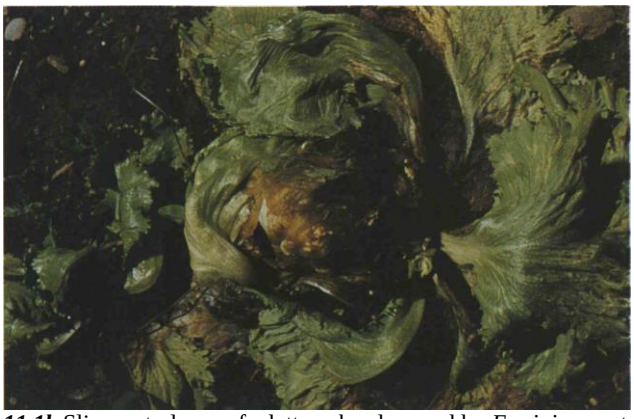


10.15b Carrot rust fly; larval feeding damage on parsley roots.



10.15c European earwig; severe defoliation of young parsley.

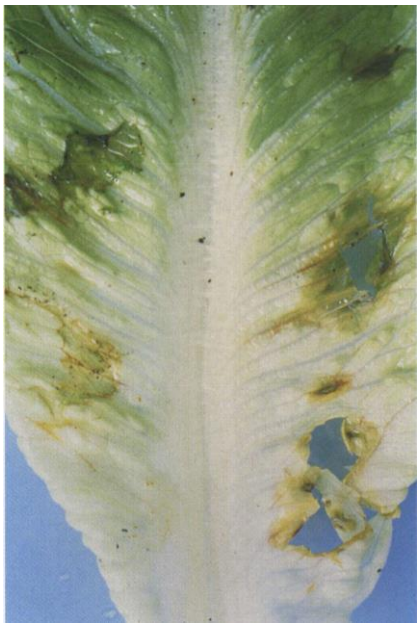
11.1a Head rot; breakdown of the youngest leaves in lettuce head.



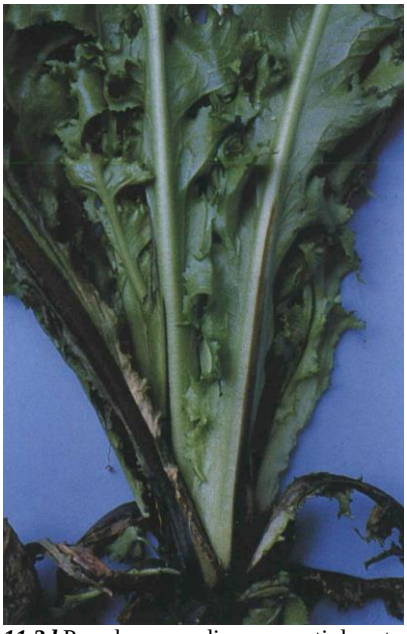
11.1b Slime rot; decay of a lettuce head caused by *Erwinia carotovora.*

11.3a Pseudomonas diseases; brown rot lesions (individual spots) on lettuce.

11.3b Pseudomonas diseases; marginal leaf spot of lettuce caused by *Pseudomonas fluorescens.*



11.3c Pseudomonas diseases; brown rot lesions on lettuce.



11.3d Pseudomonas diseases; petiole rot of lettuce.

11.4 Anthracnose; lesions on lettuce leaves.

11.5 Black root rot; lesions on chicory roots.

11.6a Bottom rot; basal decay on lettuce.

11.6b Bottom rot; severe decay of lettuce petiole.

11.7a Damping-off; wilted lettuce seedlings.

11.7b Stunt; lettuce plants killed by *Pythium* sp.

11.7c Stunt; discoloration of crown tissue of lettuce caused by *Pythium* sp.

11.8a Downy mildew; sporulation of Bremia lactucae on lettuce leaf.

11.8b Downy mildew; a severely affected lettuce leaf.

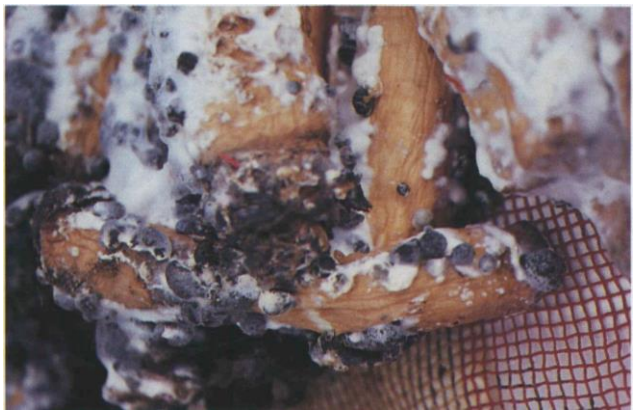
11.9a Drop; lettuce plant infected by *Sclerotinia minor*.

11.9b Drop; lettuce infected by *Sclerotinia sclerotiorum*; note white mycelium and large black sclerotia.

11.9c Drop; stem infection on lettuce.

11.9d Drop; lettuce infected by *Sclerotinia minor;* note small black sclerotia at base of plant.

11.9e Drop; aggregated sclerotia and apothecia of *Sclerotinia minor*.



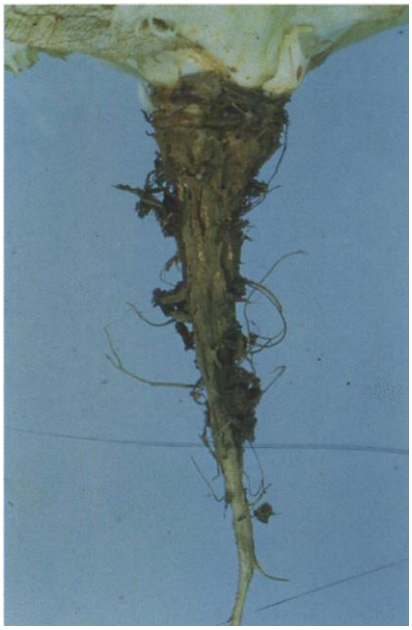
11.9f Drop (white mold); severely decayed chicory roots from storage; note white mycelium and black sclerotia.

11.10a Gray mold; crown decay of iceberg lettuce head.

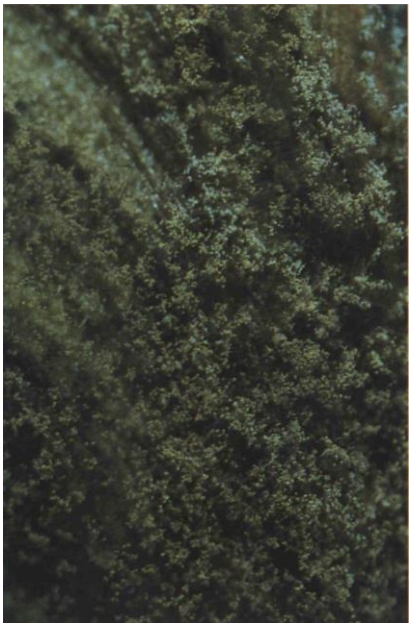
11.10b Gray mold; head infection of butterhead (Boston) lettuce.

11.10c Gray mold; basal stem infection of lettuce; note gray growth of *Botrytis cinerea* on petioles.

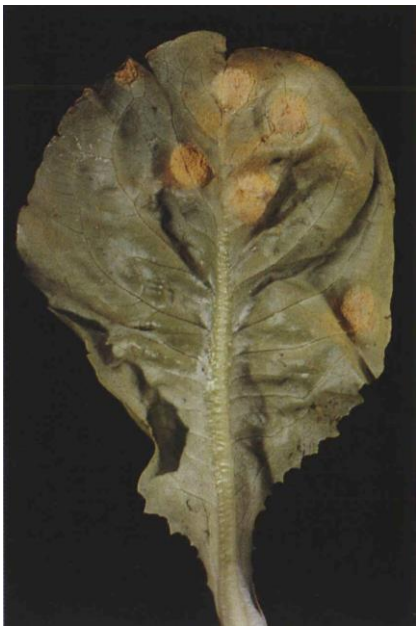
11.10d Gray mold; black sclerotia of *Botrytis cinerea* on dead lettuce tissue.



11.10e Gray mold; root rot of lettuce.



11.10f Gray mold; conidia of *Botrytis cinerea* on lettuce leaf.



11.13a Rust; lesions on lettuce leaf.

11.13b Rust; close-up of aecia of *Puccinia dioicae* on lettuce.

11.15a Aster yellows; chlorosis and bronzing of lettuce leaves.

11.15b Aster yellows; affected (left) and healthy lettuce plants.

11.16 Big vein; an affected lettuce leaf.

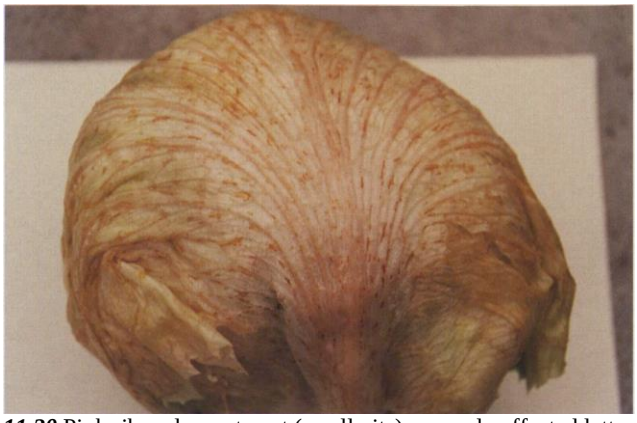
11.17a Lettuce mosaic; an affected lettuce plant.

11.17b Lettuce mosaic; mosaic pattern on an affected lettuce leaf.

11.19a Manganese deficiency; lettuce appears stunted, yellow-gray, with necrotic spots on leaf margins.

11.19b Manganese toxicity; chlorosis on Romaine lettuce leaves.

11.19c Tipburn; symptoms on the inner leaves of lettuce head.



11.20 Pink rib and russet spot (small pits); severely affected lettuce head.

11.23a Aster leafhopper; adult; length ±3 mm.

11.23b Aster leafhopper; adult, showing diagnostic head spotting.

11.26 Redbacked cutworm; curled larva and its feeding injury on lettuce.

11.27a Black slug; side view (contracted), scale in cm; length up to 150 mm.

11.27b Spotted garden slug; scale in inches; length ≥100 mm.

11.27c Gray garden slug; juvenile with severe feeding damage on dill; length 35-50 mm.

12.1a Stewart's wilt; pale green streaks on leaves later turn brown.

12.1b Stewart's wilt; affected plant.

12.1c Stewart's wilt; severely affected plants in a field.

12.2a Damping-off; root and seed decay symptoms on a seedling.

12.2b Damping-off and root rot; circular area (center) of plants with leaf yellowing.

12.3a Gibberella ear rot; characteristic pinkish mycelium of *Fusarium graminearum.*

12.3b Fusarium kernel rot; white mycelium (*Fusarium*) and black perithecia (*Gibberella*)

12.3c Ear and kernel rot; sap beetles may be vectors of *Fusarium* spp.

12.4 Eyespot; moderately (right) and severely affected leaves.

12.5 Common rust; uredinial pustules on affected leaf.

12.6a Common smut; galls (boils) on an affected ear.

12.6b Common smut; affected tassel with galls.

12.7a Head smut; affected (brush-like) tassel.

12.7b Head smut; phyllody (left) and teliospore formation on ear.

12.8a Diplodia stalk rot; dry, pale brown rot of pith causes sudden wilt.

12.8b Gibberella stalk rot; pink to red discoloration of stem tissues.

12.8c Gibberella stalk rot; healthy (left) and severely affected stalks.

12.8d Pythium stalk rot; dark brown, water- soaked rot causes lodging.

12.9a Three- to five-leaf dieback; dieback, root rot and stunting (healthy, left).

12.9b Three- to five-leaf dieback; dead and weakened seedlings.

12.9c Three- to five-leaf dieback; affected older seedlings (healthy, right).

12.9d Three- to five-leaf dieback; note hypocotyl and *Penicillium*-colonized seeds.

12.10a Maize dwarf mosaic; mosaic stippling and dark green streaking on young plant.

12.10b Maize dwarf mosaic; symptoms of strain B (= sugarcane mosaic virus).

12.12a Armyworm; injury to corn foliage.

12.12b Armyworm; larva.

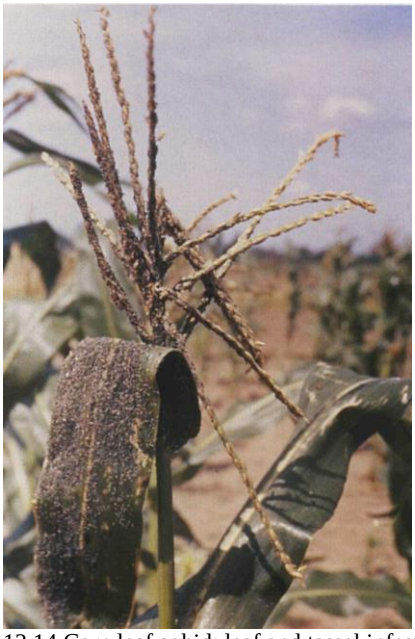
12.12c Armyworm; adult moth; wingspan ± 40 mm.

12.13a Corn earworm; damage to ears.

12.13b Corn earworm; larva and damage on ear.

12.13c Corn earworm; larva and damage on ear.

12.13d Corn earworm; adult moth; wingspan 45-65 mm.



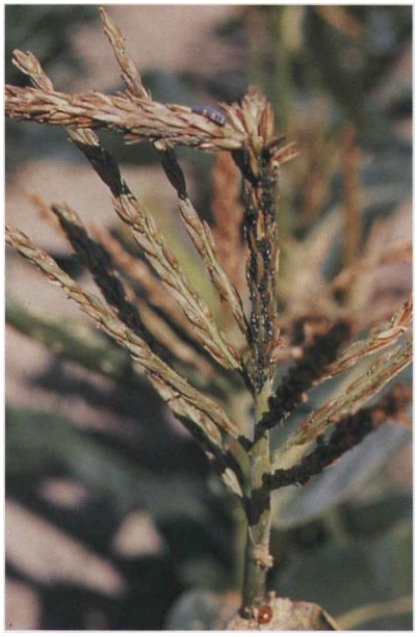
12.14 Corn leaf aphid; leaf and tassel infestation.

12.15a Northern corn rootworm; adult beetle on corn silks.

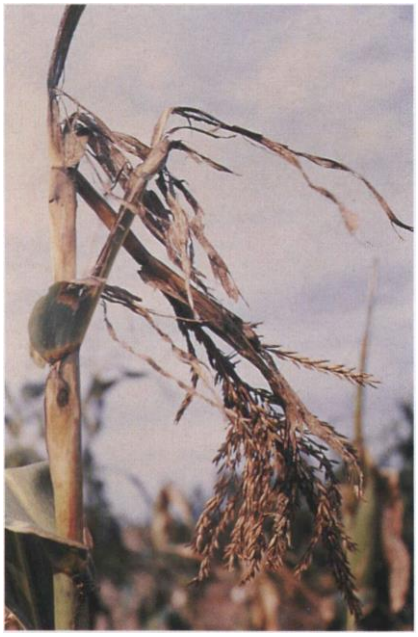
12.15b Northern corn rootworm; adult; length ± 10 mm.

12.15c Western corn rootworm; adult; length ± 10 mm.

12.16a European corn borer; leaf injury from larval feeding.



12.16b European corn borer; broken tassel; note aphids and lady beetle larva.



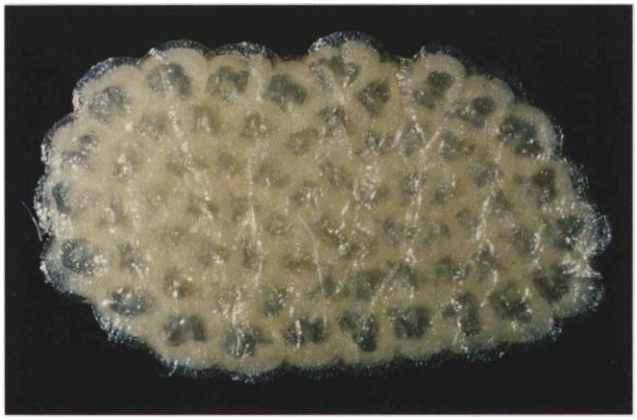
12.16c European corn borer; broken stalk from larval feeding.

12.16d European corn borer; lodging from larval feeding at the base of stalks.

12.16e European corn borer; larva feeding on ear.

12.16f European corn borer; larva and feeding damage on kernels.

12.16g European corn borer; adult moth; wingspan ±25 mm.



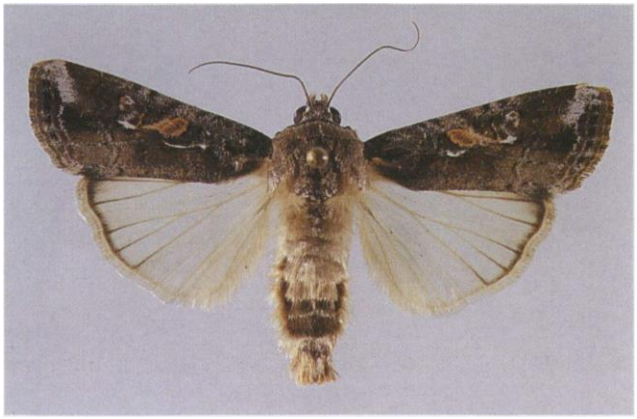
12.16h European corn borer; egg mass.

12.17a Fall armyworm; young larva and injury to corn silks.

12.17b Fall armyworm; intermediate larva.

12.17c Fall armyworm; mature larva, about 40 mm long.

12.17d Fall armyworm; pupa (normally found in the soil).



12.17e Fall armyworm; adult moth (dark form); wingspan 30-38 mm.

12.19 Four-spotted sap beetle; adult; length 4-7 mm.

12.20a Seedcorn maggot; larvae and damage to a germinating seed.

12.20b Seedcorn maggot; larva.

12.20c Seedcorn maggot; adult fly; length 7-9 mm.

12.21a Wireworm; larvae.

12.21b Wireworm; adult "click" beetle; length 3-30 mm.

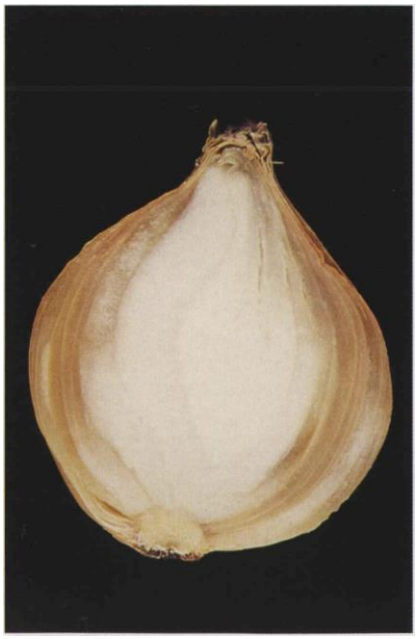
12.22a Two-striped grasshopper; adult; length 25-40 mm.

12.22b Potato stem borer; larva on a corn seedling.

13.1 Slippery skin; deterioration of inner scales on an onion bulb.

13.2a Soft rot (bacterial); external symptoms (neck decay).

13.2b Soft rot (bacterial); internal symptoms (scale rot).



13.3 Sour skin; scale discoloration on an onion bulb.

13.4 Basal rot; onion bulb with surface growth of *Fusarium oxysporum* f. sp. *cepae.*

13.5a Botrytis leaf blight; grayish white leaf spots usually have a characteristic silver halo.

13.5b Botrytis leaf blight (left); with downy mildew (center); with downy mildew and purple blotch (right).

13.6a Downy mildew; purplish, velvety mat on leaves in early morning.

13.6b Downy mildew; close-up of sporulating lesions on onion leaf.

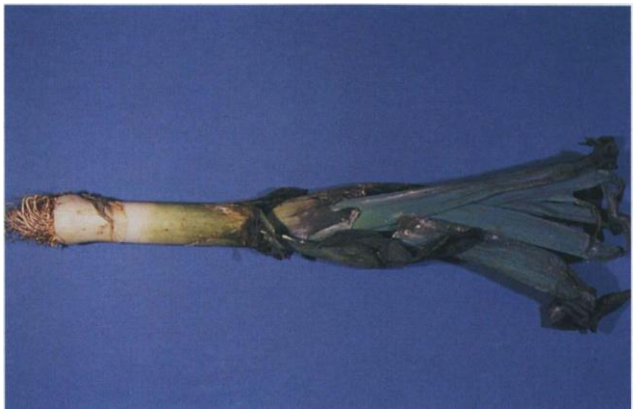
13.6c Downy mildew; an affected (collapsed, yellowed) onion crop.

13.6d Downy mildew; microscopic view of sporangiophores of *Peronospora destructor*.

13.7a Neck rot; internal symptoms (decay) on onion bulb.

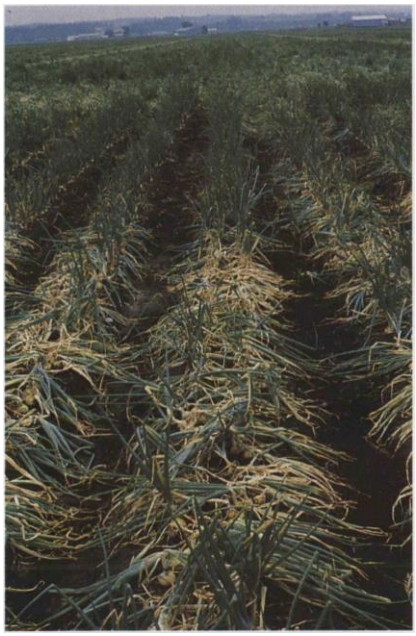
13.7b Neck rot; external symptoms (decay, sclerotia) on onion bulb.

13.7c Neck rot; sclerotia of *Botrytis* sp. on onion bulb.



13.7d Neck rot; external symptoms (dieback) on leek.

13.8a Pink root; discoloration of onion roots.



13.8b Pink root; foliar dieback of onion.

13.9a Purple blotch; early symptoms on onion leaves.

13.9b Purple blotch; advanced symptoms (large lesions) on onion leaves.

13.10 Smudge; dark lesions on onion bulbs.

13.11a Smut; leaf dieback on young onion plants.

13.11b Smut; elongate pustules on young onion plant.

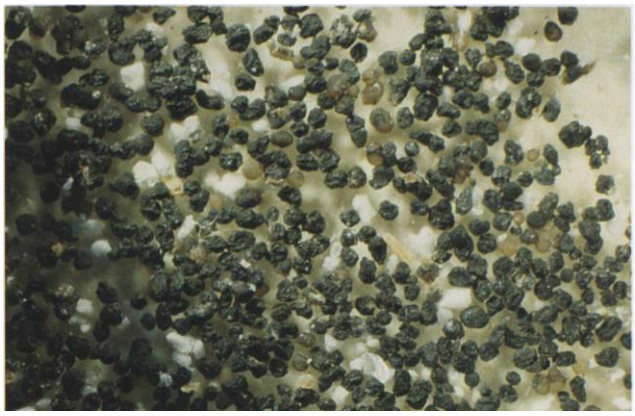
13.11c Smut; dark pustules on garlic bulbs.

13.12a White rot; yellowing and dieback of garlic plants (foreground).

13.12b White rot; grayish white mycelium and black sclerotia at base of onion bulbs.

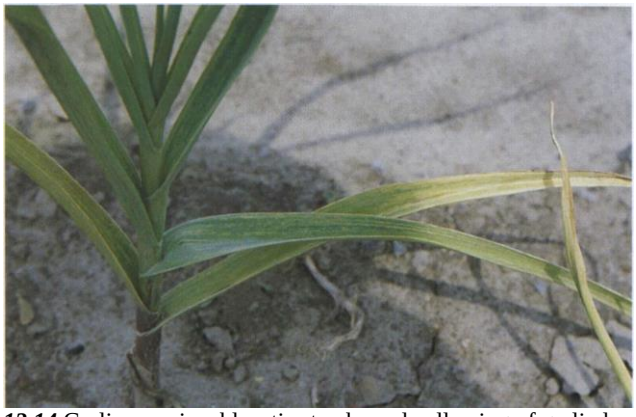
13.12c White rot; mycelium and sclerotia on garlic bulb.

13.12d White rot; garlic bulb with small black sclerotia on the surface.



13.12e White rot; sclerotia of *Sclerotium cepivorum.*

13.13 Aster yellows; affected onion plants are stunted and yellow.

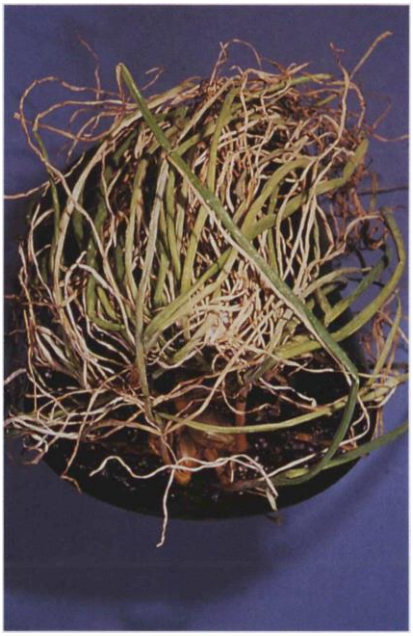


13.14 Garlic mosaic; chlorotic streaks and yellowing of garlic leaves.

13.15a Herbicide injury; bleaching of an onion leaf caused by oxy- fluorfen.

13.15b Herbicide injury; leaf curling on onion.

13.15c Herbicide injury; leaf yellowing and dieback of onion.



13.16 Ozone injury; severely affected onion plants.

13.17 Sprout inhibitor injury; scale separation of onion bulb from maleic hydrazide.

13.19 Tipburn; an affected onion crop.

13.20 Translucent scale; symptoms on Spanish onion.

13.21 Pelting rain injury; lesions on an onion leaf.

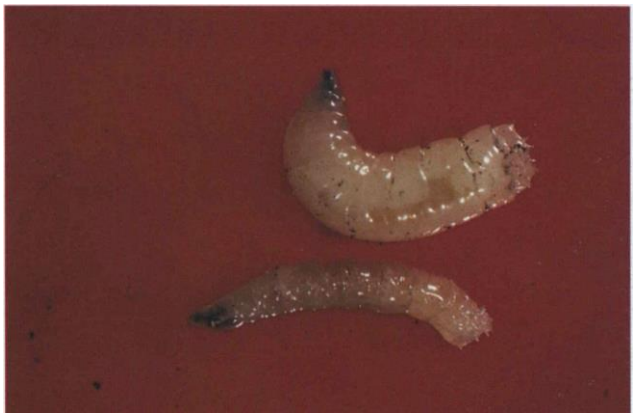
13.23 Root-lesion nematode; yield and size of onion bulbs decrease with increasing numbers of nematodes in soil.

13.24 Stem and bulb nematode; affected onion bulbs are deformed, and leaves are short and senesce early.

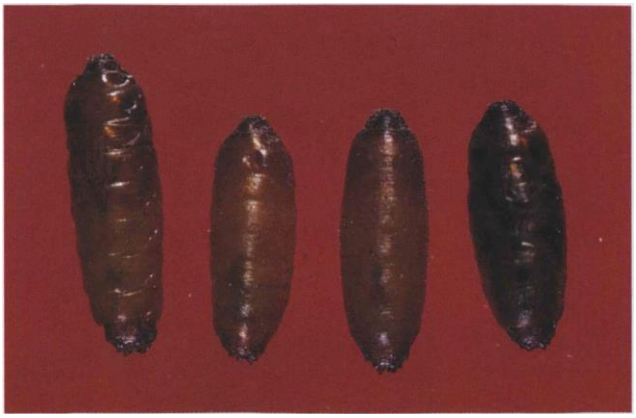
13.25 Onion bulb fly; adult; length ± 8 mm.

13.26a Onion maggot; larvae on an onion bulb.

13.26b Onion maggot; adult fly, male (left), and female; length ± 6 mm.



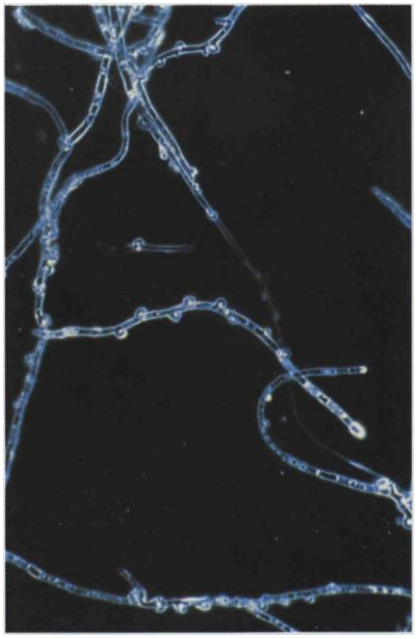
13.26c Onion maggot; larvae; length 6-8 mm at maturity.



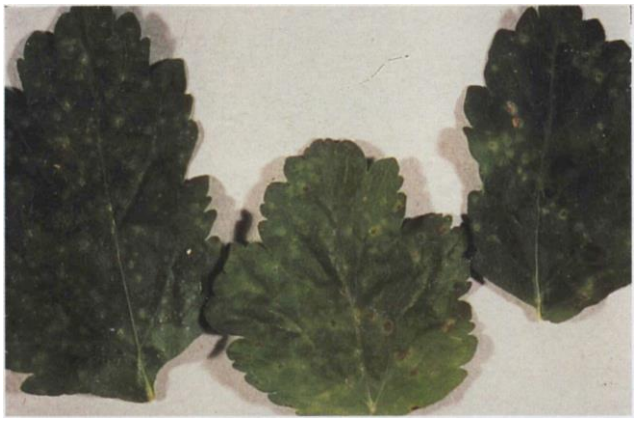
13.26d Onion maggot; pupae; length 5-7 mm.

13.26e Onion maggot; infested (left) and healthy onion plants.

14.2a Itersonilia canker; lesion on the shoulder of a parsnip root.



14.2b Itersonilia canker; mycelium of *Itersonilia perplexans* with clamp connections.

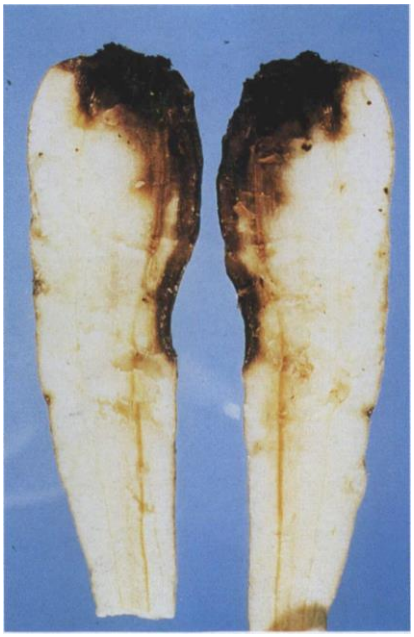


14.2c Itersonilia canker; brown necrotic foliar lesions surrounded by pale green halos.

14.3a Phoma canker; light brown lesions become dark and form cankers on petioles.

14.3b Phoma canker; leaf blight symptoms.

14.3c Phoma canker; on upper petiole, forming "shepherd's crook."



14.3d Phoma canker; lesion on shoulder and crown of a parsnip root.

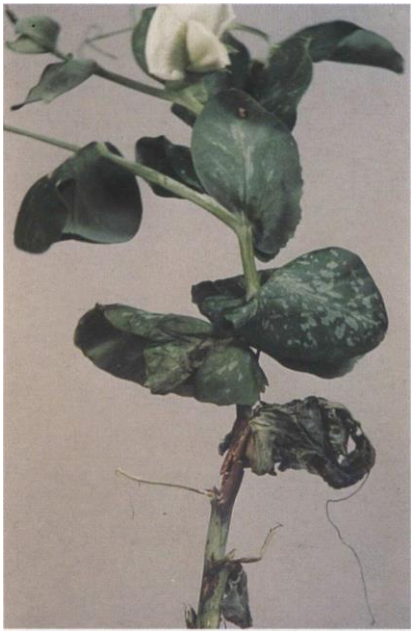
14.7 Black swallowtail; mature larva with scent glands extended.

15A.1a Bacterial blight; leaf lesions.

15A.1b Bacterial blight; pod lesions.

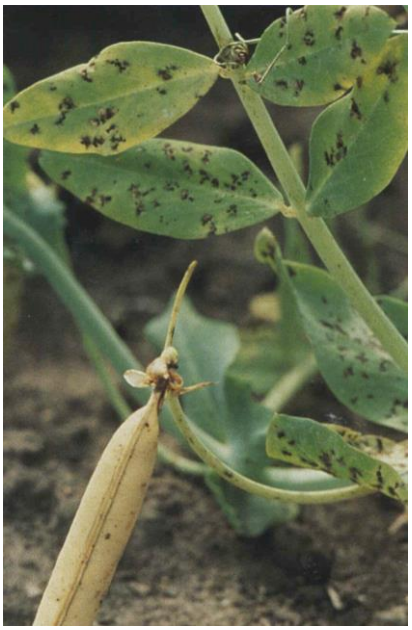
15A.2a Ascochyta leaf and pod spot; lesions on leaves and stem.

15A.2b Ascochyta leaf and pod spot; lesions on pods.



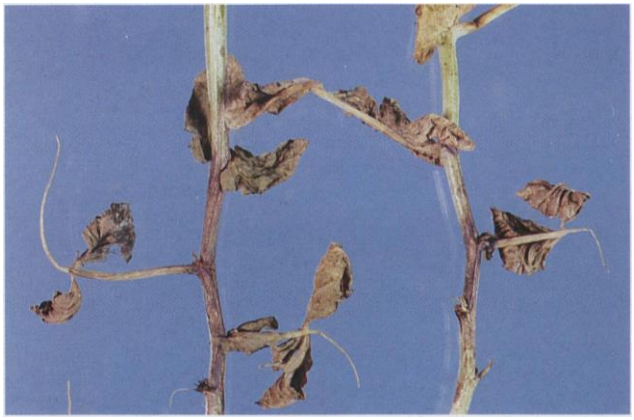
15A.2c Ascochyta foot rot; stem lesion.

15A.2d Ascochyta foot rot; pod lesions.



15A.2e Mycosphaerella blight; lesions on leaves and pod.

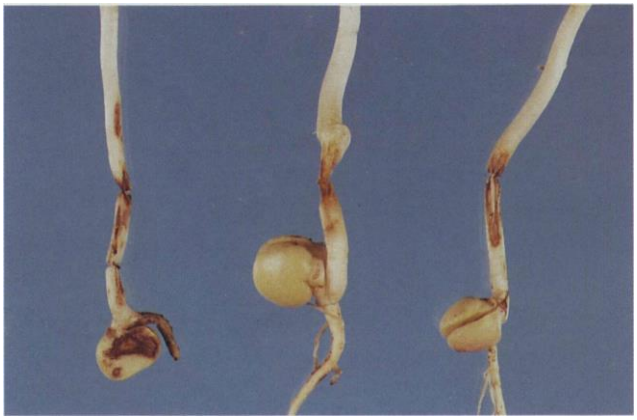
15A.2f Mycosphaerella blight; pod lesions.



15A.2g Mycosphaerella blight; stem lesions.

15A.3a Seed decay; badly rotted seeds.

15A.3b Seedling blight; uneven stand caused by seed decay and damping-off.



15A.3c Seedling blight; stem lesions.

15A.3d Root rot; extensive damage in a pea field.

15A.3e Root rot; above-ground symptoms (dieback).

15A.3f Root rot; degrees of severity from healthy (left) to severe.

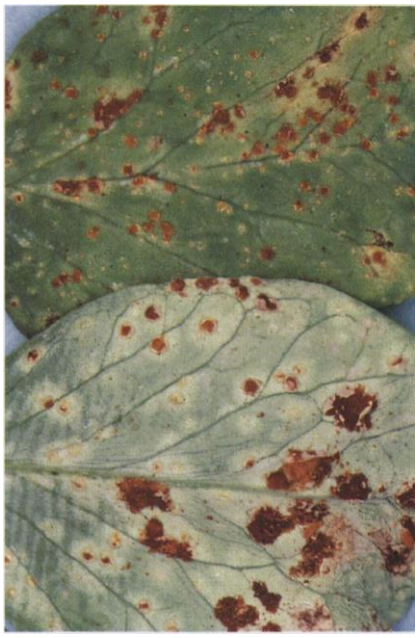
15A.4a Downy mildew; lesions on leaves.

15A.4b Downy mildew; sporulation of *Peronospora viciae* on undersides of leaves.

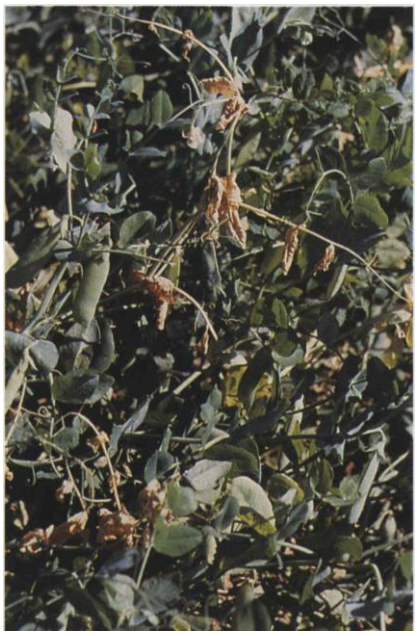
15A.4c Downy mildew; growth of *Peronospora viciae* mycelium inside a pod.

15A.5a Powdery mildew; foliar symptoms.

15A.5b Powdery mildew; leaves with small, black cleistothecia of *Erysiphe polygoni*.



15A.6 Rust; pustules on leaves.



15A.7 Sclerotinia stem rot; a wilted plant.

15A.8 Septoria leaf blotch; leaf lesions with small, dark pycnidia.

15A.9a Aster yellows; leaf proliferation.

15A.9b Bean yellow mosaic; foliar symptoms (mosaic).

15A.9c Pea enation mosaic; blisters (enations) on leaves.

15A.9d Pea streak; leaf and pod symptoms.

15A.9e Pea stunt; foliar symptoms.

15A.9f Pea seed-borne mosaic; diseased (left), note downward cupping of leaflet margins, short internodes.

15A.9g Pea stunt; chlorotic foliage (center).

15A.10 Boron toxicity; chlorotic foliage.

15A.11a Herbicide injury; leaf chlorosis from foliar application of bentazon plus adjuvant.

15A.11b Herbicide injury; leaf chlorosis from preplant-incorporated metribuzin.

15A.11c Herbicide injury; leaf distortion from a growth regulator herbicide.

15A.11d Water congestion; healthy (left) and affected foliage.

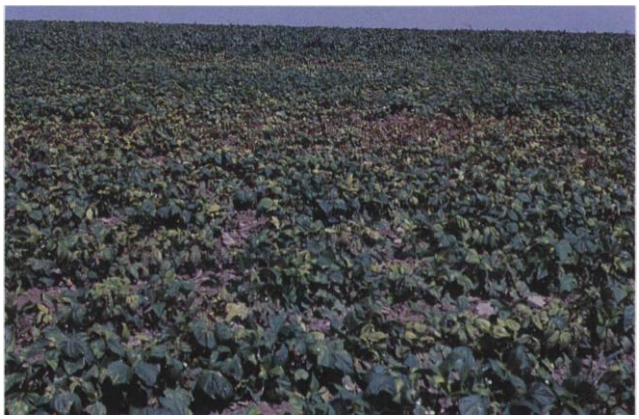
15A.14 Pea aphid; colonies on pea leaf and pod.

15A.15 Pea leaf weevil; adult (below), length 4.5 mm; clover root curculio, adult (above), length 5 mm.

15B.1a Bacterial brown spot; leaf lesions.

15B.1b Common blight; leaf symptoms (brown besions).

15B.1c Common blight; leaf infection.

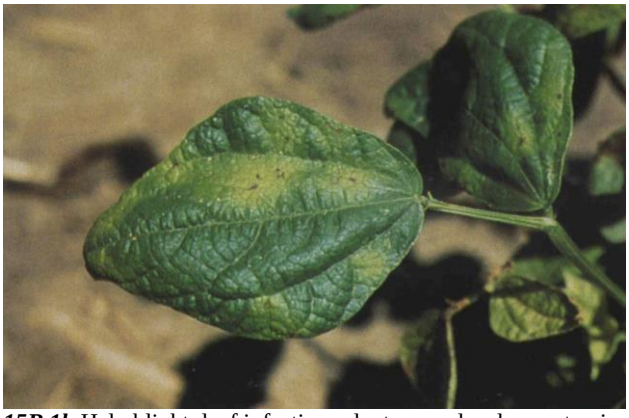


15B.1d Common blight; area of severe infection in a field (center).

15B.1e Common blight; pod infection.

15B.1f Common blight; symptoms on infected seeds.

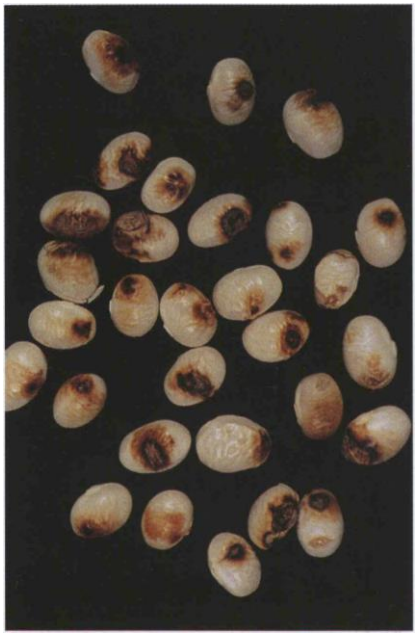
15B.1g Halo blight; leaf symptoms include small, necrotic spots with yellow-green haloes.



15B.1h Halo blight; leaf infection; plants may develop systemic chlorosis.

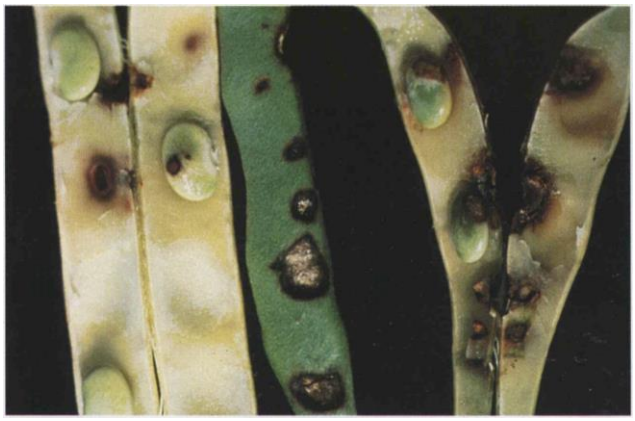
15B.1i Halo blight; pod lesions appear water-soaked, then become red or brown.

15B.2a Anthracnose; leaf infection usually involves necrosis of veins.



15B.2b Anthracnose; severe seed infection.

15B.2c Anthracnose; lesions on an immature pod become sunken and brown with a dark border.

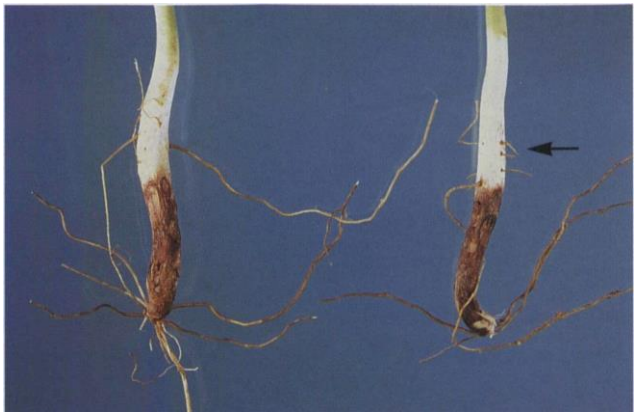


15B.2d Anthracnose; severe pod and seed infection.

15B.3a Gray mold; infected pod showing sporulation of ***Botrytis cinerea.***

15B.3b Gray mold (right) and white mold (left); pod infections.

15B.4 Black root rot; root and hypocotyl lesions become dark brown to black; healthy (left) to severely affected.

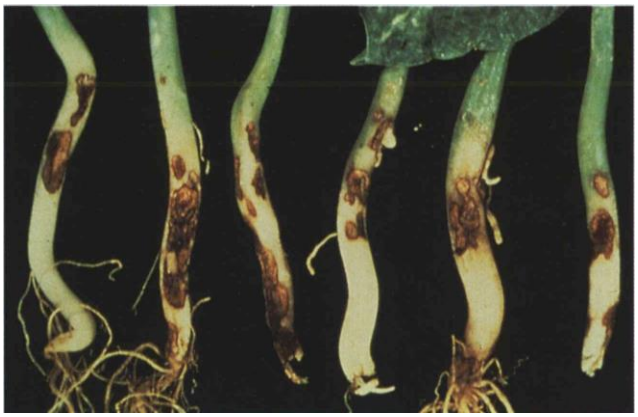


15B.5a Fusarium root rot; dry, reddish-brown root rot symptoms; note adventitious roots on hypocotyl.

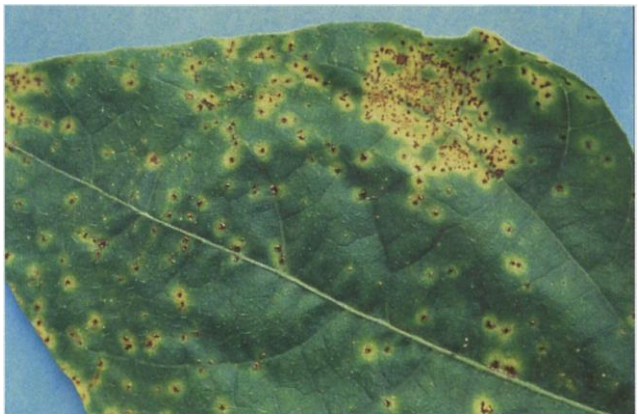
15B.5b Fusarium root rot; root symptoms.

15B.5c Fusarium root rot; note leaf yellowing on affected plants.

15B.6 Pythium diseases; seed decay and seedling root rot.



15B.7 Rhizoctonia root rot; elongate, sunken, reddish-brown lesions on hypocotyl and roots.



15B.8a Rust; leaf infection; reddish-brown uredinial pustules with yellow haloes.

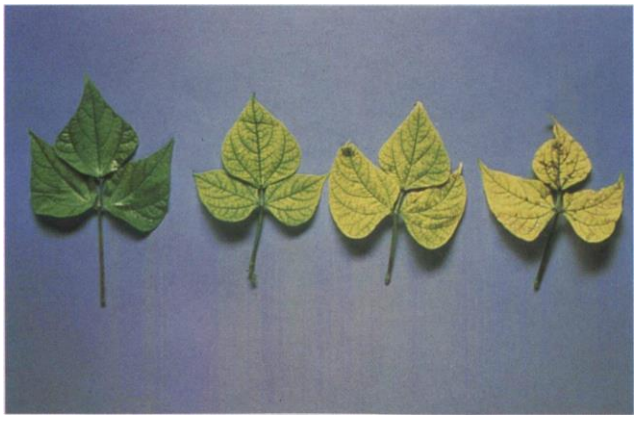
15B.8b Rust; pustules become dark brown to black as teliospores develop.

15B.9a White mold; affected bean plant (center).

15B.9b White mold; pod symptoms, white cottony mycelium with sclerotia; healthy (bottom).

15B.10 Bean common mosaic; irregular light and dark green patches and puckering of leaves.

15B.11 Bean yellow mosaic; mosaic symptoms; some strains produce chlorotic or necrotic spots.



15B.12a Iron deficiency; healthy (left) to severely affected (right) leaves; veins remain green.

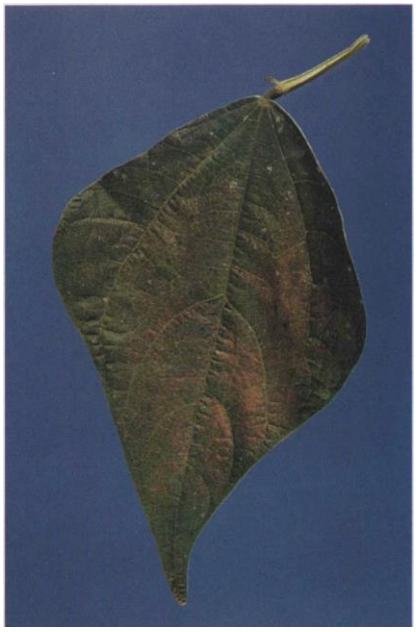
15B.12b Manganese deficiency; severe interveinal leaf chlorosis.

15B.12c Zinc deficiency; foliar symptoms (chlorosis).

15B.13a Baldhead; growing point killed by mechanical damage to seed.

15B.13b Herbicide injury; damage to growing point from dicamba.

15B.13c Ozone injury; bronzing symptoms on leaves.



15B.13d Sunscald; a damaged leaf.

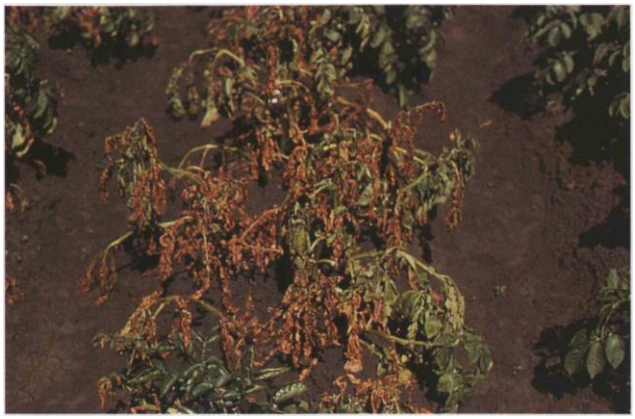
15B.13e Wind injury; note white areas on affected leaves.

15B.18 Seedcorn maggot; larval damage to roots of seedlings.

15B.19a European earwig; damage to bean leaves.

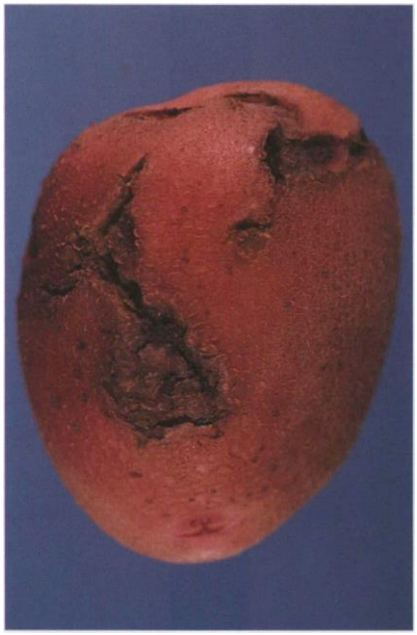
15B.19b Mexican bean beetle; adults; length 6-8 mm.

16.1a Bacterial ring rot; wilted stem, a characteristic susceptible reaction.

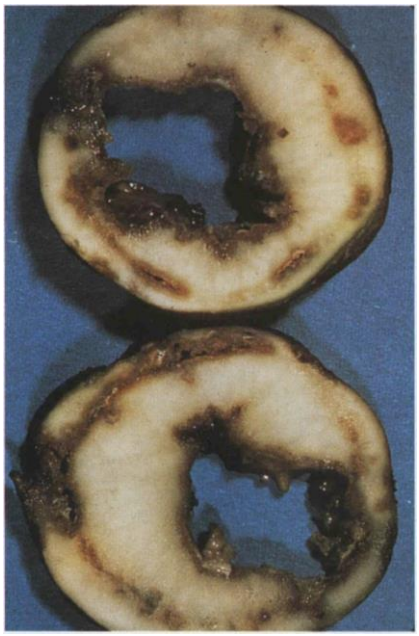


16.1b Bacterial ring rot; wilted stems and premature dieback of foliage.

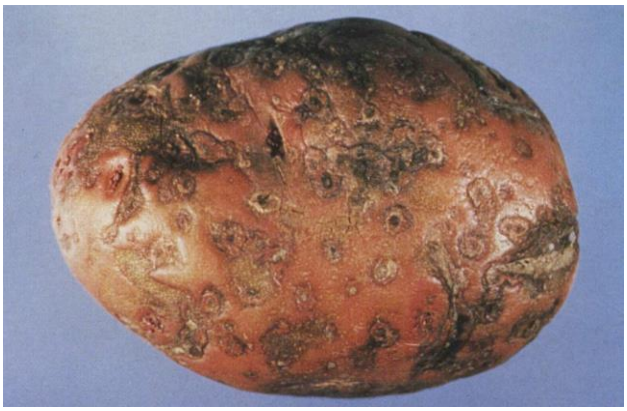
16.1c Bacterial ring rot; moderately severe symptoms in tubers.



16.1d Bacterial ring rot; skin cracking on tuber.

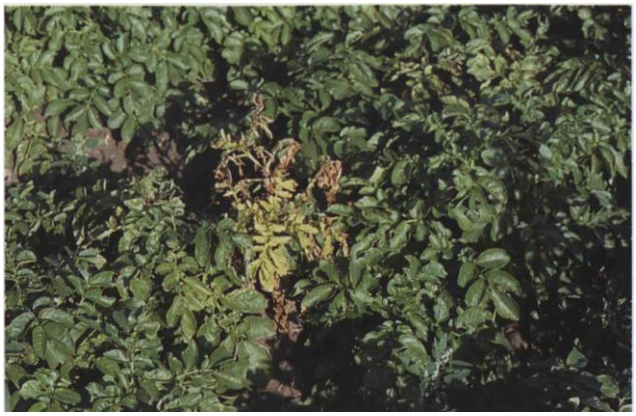


16.1e Bacterial ring rot; very severe symptoms in tubers.

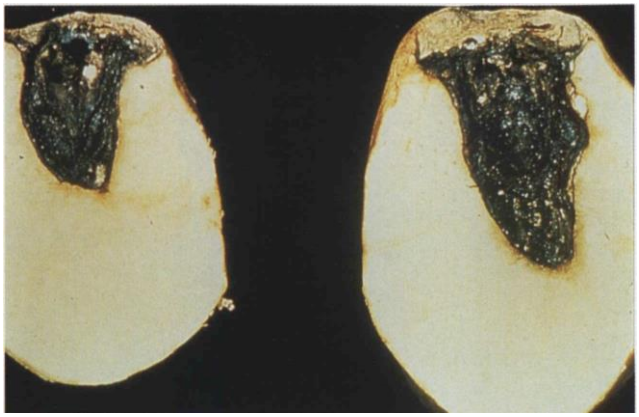


16.2a Bacterial soft rot; lenticel infections of tuber.

16.2b Bacterial soft rot; severe decay of tubers in a plastic bag.

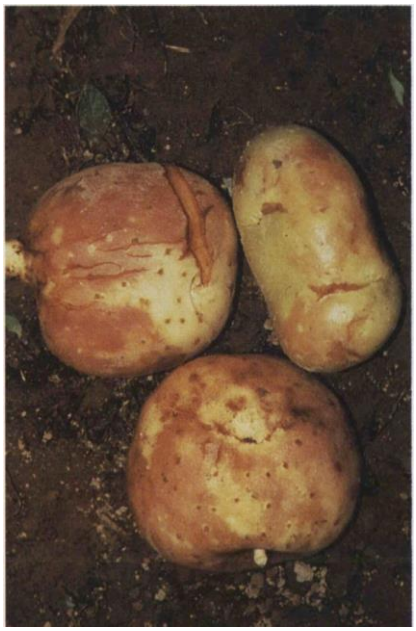


16.3a Blackleg; affected plant with wilted, chlorotic foliage.

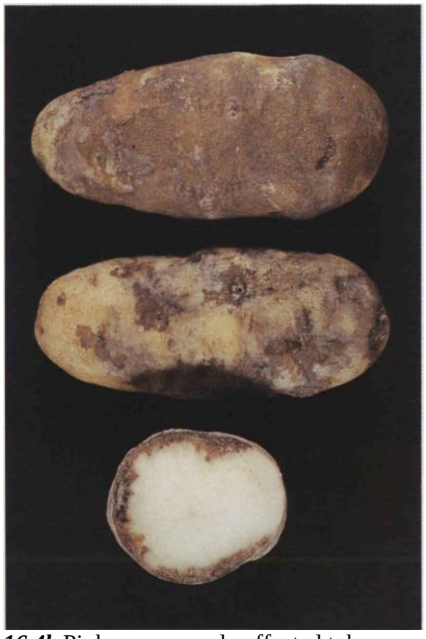


16.3b Blackleg; rotted tuber; note infection started at stolon end.

16.3c Blackleg; soot-black discoloration of stem and bacterial ooze.



16.4a Pink eye; cracked skin with pink- brown discoloration.



16.4b Pink eye; severely affected tubers.

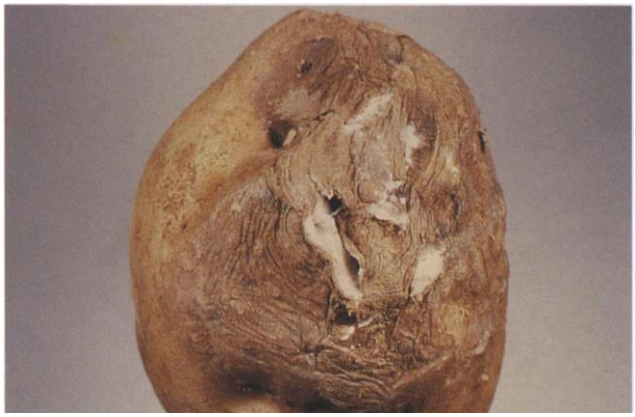
16.5a Common scab; raised symptom, erupting lesions.

16.5b Common scab; deep-pitted symptom, lesions up to 6 mm deep.

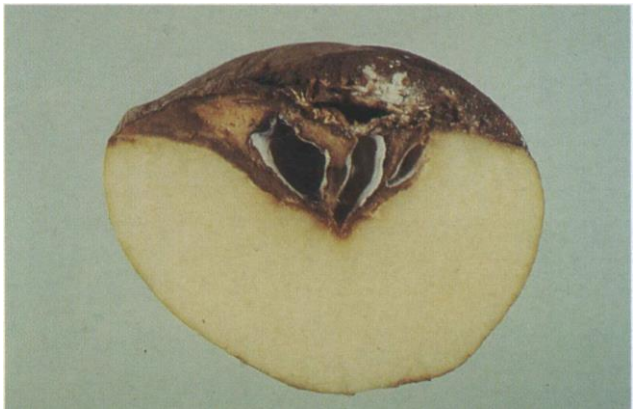
16.5c Common scab (above); shallow symptom. Russet scab (below); corky reticulations.

16.6a Black dot; microsclerotia of *Colletotrichum* on dead vine.

16.6b Black dot; an affected tuber with microsclerotia of *Colletotrichum* (tiny black dots).



16.7a Dry rot; sunken, shrivelled lesion and concentric rings on affected tuber.

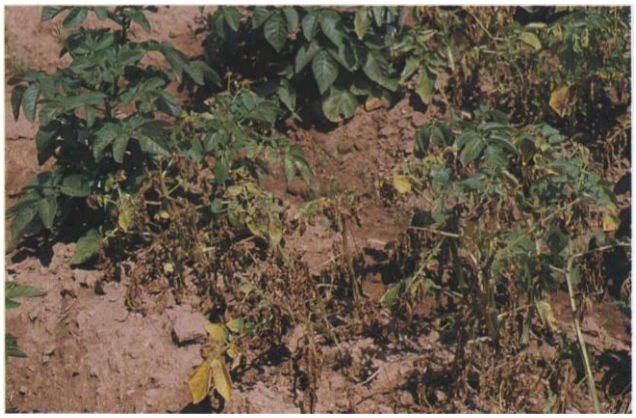


16.7b Dry rot; tuber cavity lined with white mycelium of *Fusarium.*

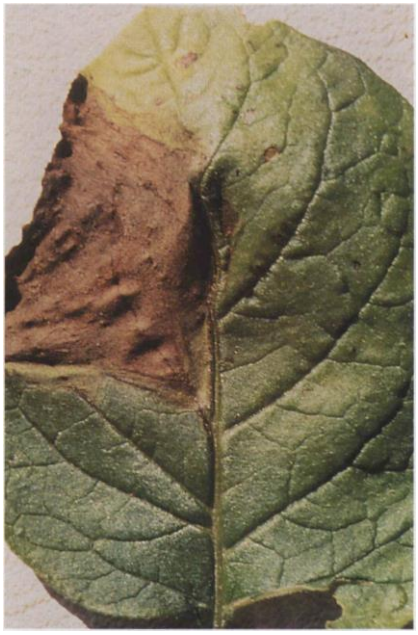
16.8a Early blight; leaf lesions are often delimited by veins and have concentric rings. See late blight, *16.11a,b.*

16.8b Early blight; severe top-killing in potato field.

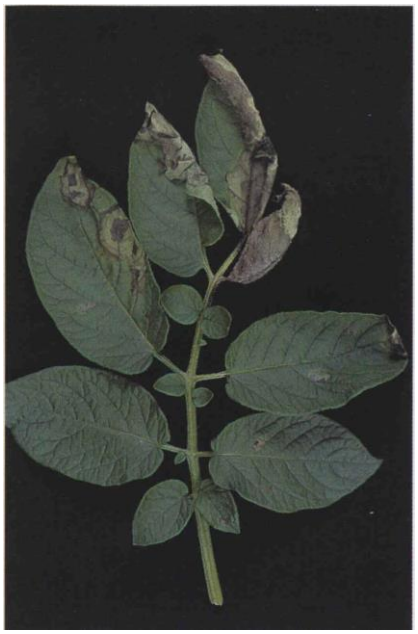
16.8c Early blight; sunken area on tuber caused by *Alternaria* infection.



16.9 Fusarium wilt; severely wilted plants.



16.10 Gray mold; gray-black lesions turn brown during dry weather.

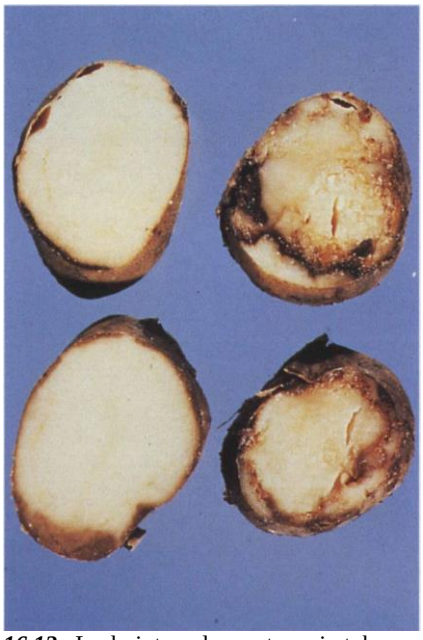


16.11a Late blight; leaf lesions, at first water- soaked, quickly become dark brown, brittle.

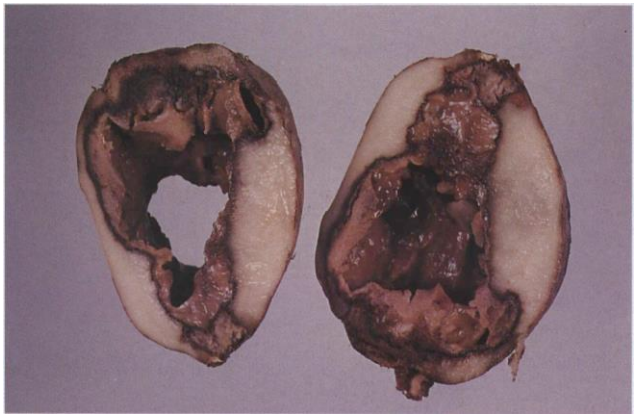
16.11b Late blight; diffuse leaf lesions; compare with early blight, *16.8a*.

16.11c Late blight; stem infection.

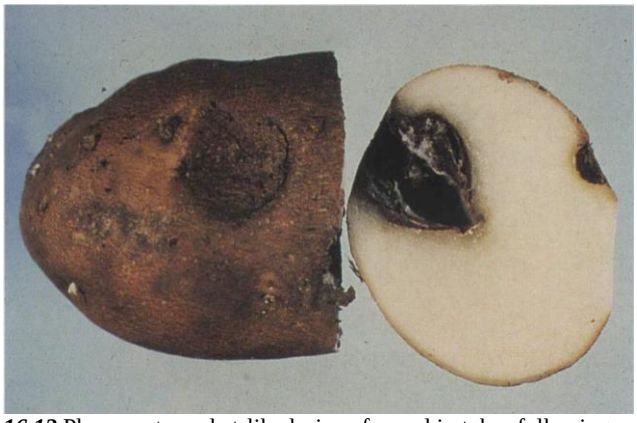
16.11d Late blight; reddish, granular, sunken lesions in tuber.



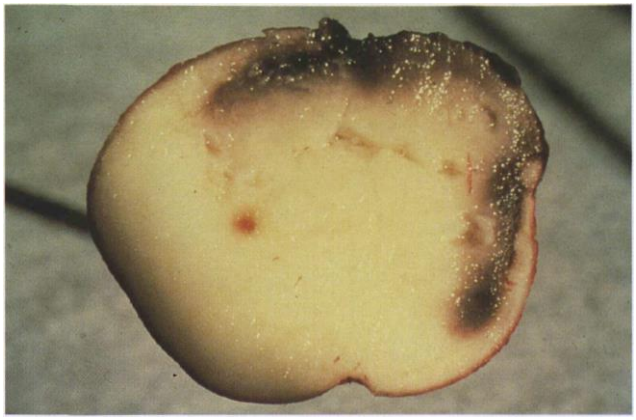
16.12a Leak; internal symptoms in tubers.



16.12b Leak; severely rotted tubers.



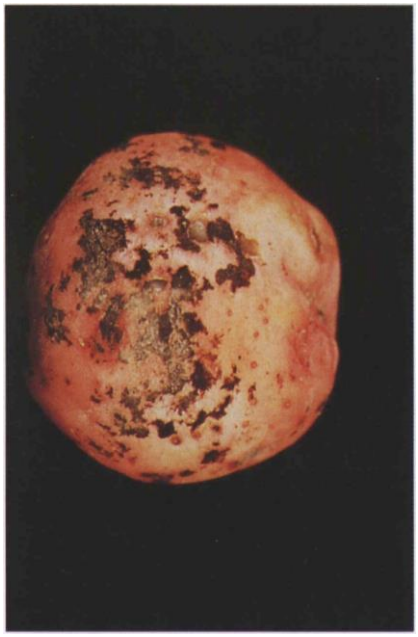
16.13 Phoma rot; pocket-like lesions formed in tuber following wounding.



16.14 Pink rot; tissues surrounding the decay have a pinkish tinge.

16.15a Rhizoctonia canker; lesions on newly emerged sprouts on seed piece.

16.15b Rhizoctonia canker; characteristic stem cankers.



16.15c Rhizoctonia canker; black scurf stage (sclerotia) on tuber.

16.15d Rhizoctonia canker; knobby tubers from infected plants.

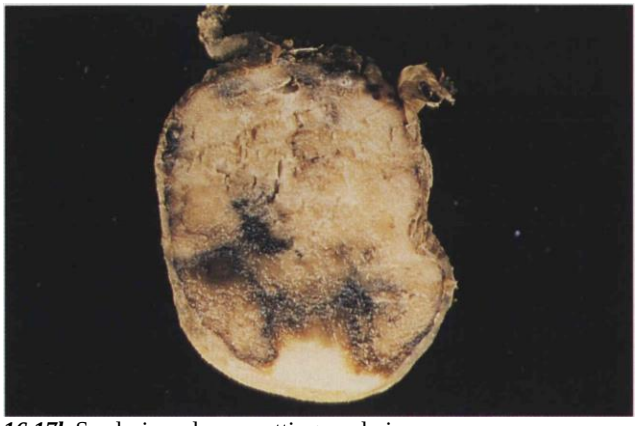
16.15e Rhizoctonia canker; cupped, pinkish upper leaves, often mistaken for purple-top wilt.

16.15f Rhizoctonia canker; aerial tubers formed on infected vines.

16.15g Rhizoctonia canker; perfect state *(Thanatephorus cucumeris)*, gray-white mycelium (center) just above soil line.

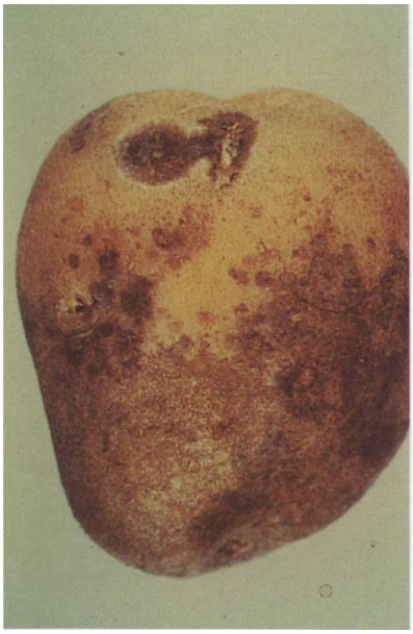
16.16 Powdery scab; numerous deep pits, usually smaller and rounder than in common scab (see *16.5).*

16.17a Seed-piece decay; very poor emergence in affected crop.

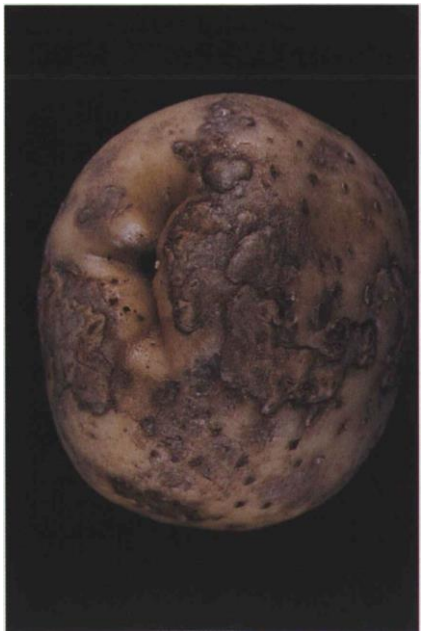


16.17b Seed-piece decay; rotting seed piece.

16.17c Seed-piece decay; decayed seed piece.



16.18a Silver scurf; moderately affected tuber.

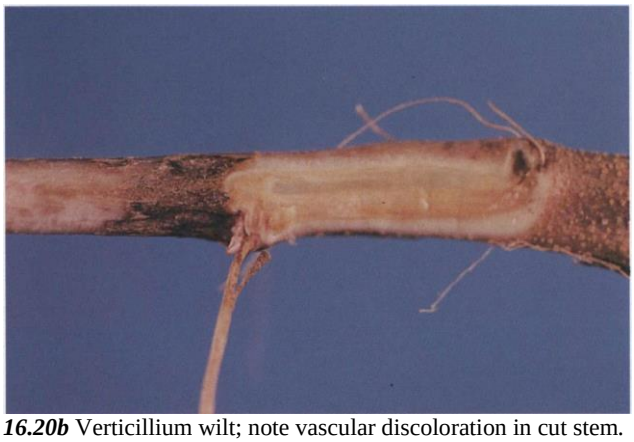


16.18b Silver scurf; severely affected tuber.

16.18c Silver scurf; unpeeled and abrasive-peeled tubers; affected tubers are difficult to peel, skin tissue may remain.

16.19 Skin spot; small, sunken spots with raised centers on tuber surface.

16.20a Verticillium wilt; wilted plants.



16.20b Verticillium wilt; note vascular discoloration in cut stem.

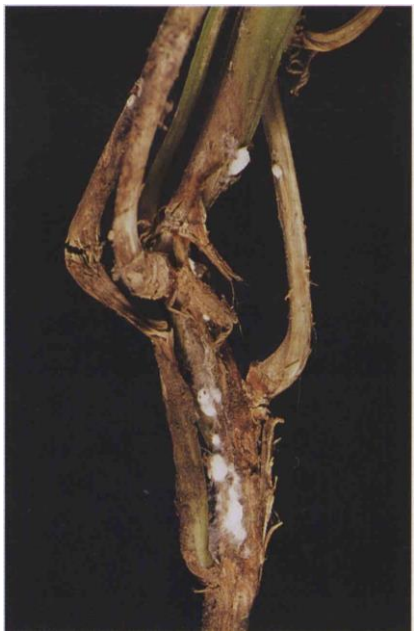
16.20c Verticillium wilt; vascular discoloration at stem end of infected tuber.

16.21a Wart; galls on above-ground stem tissue.

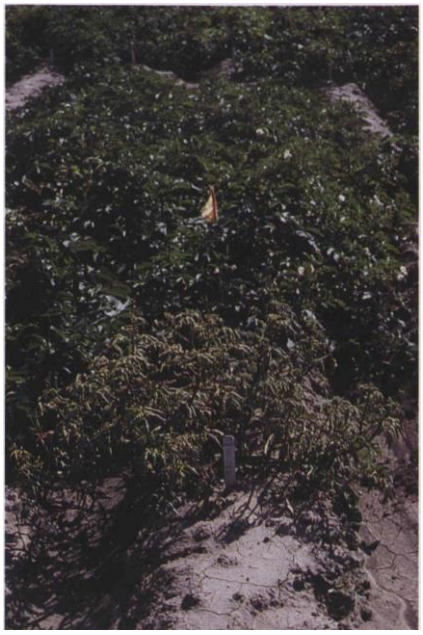
16.21b Wart; infection at base of potato stem.

16.21c Wart; tuber infection (large gall).

16.21d Wart; black-wart stage and stolon tip infection.

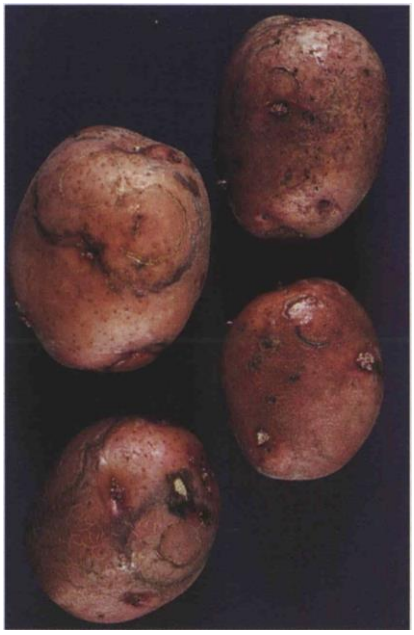


16.22 White mold; potato vine with mycelium of *Sclerotinia sclerotiorum.*



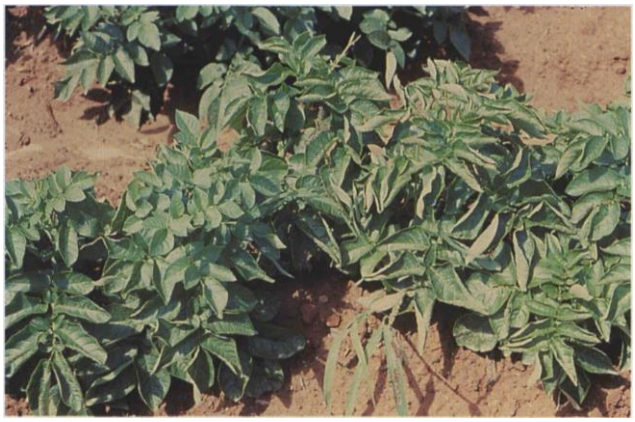
16.23 Aster yellows; plant showing purple- top wilt (foreground).

16.24 Calico; leaf yellowing symptoms.

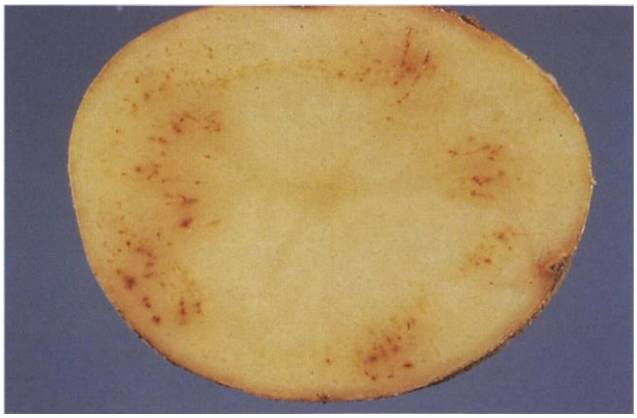


16.25a Corky ring spot; external ring spotting symptoms on tubers.

16.25b Corky ring spot; brown lines and areas in tubers.



16.26a Leafroll; marked upward rolling of leaves on affected plant (center).



16.26b Leafroll; net necrosis may form in tubers of some cultivars during storage.

16.27a Mosaic; mild mottling and distortion of leaves (potato virus X).

16.27b Rugose mosaic; severely wrinkled leaves (potato virus X plus Y).

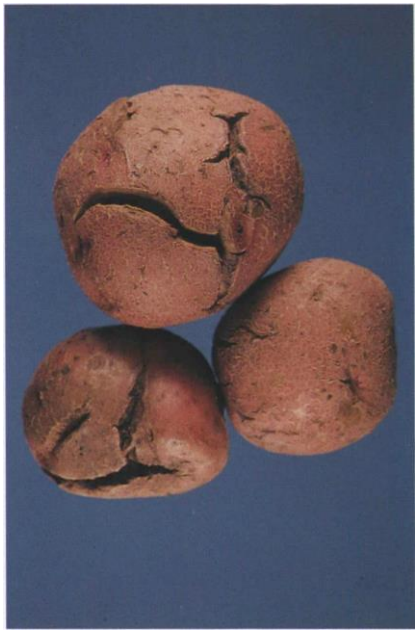
16.28a Spindle tuber; affected plant displaying upright growth habit.

16.28b Spindle tuber; spindle-shaped tubers from affected plants.

16.29a Witches'-broom; numerous shoots arise from infected tuber.

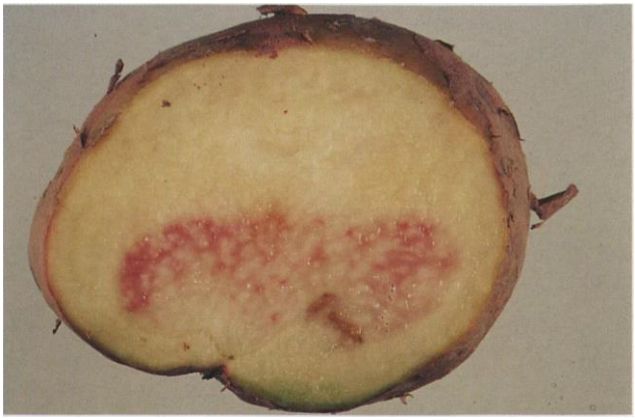
16.29b Witches'-broom; upright, multi- branched stems, small aerial tubers.

16.30 Blackheart; discoloration usually from lack of oxygen.



16.31 Growth cracks; note callus in the cracked areas.

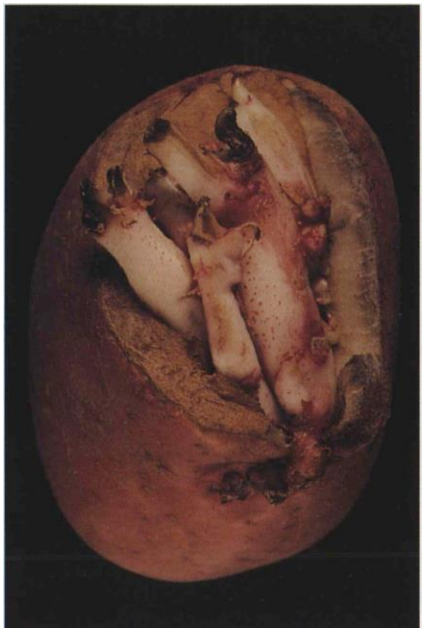
16.32 Hollow heart; large, angular, tan to brown cavities inside affected tuber.



16.34a Genetic abnormality; pink color in center of tuber of white- fleshed cultivar.

16.34b Herbicide injury; fiddlehead distortion caused by picloram.

16.34c Herbicide injury; tuber malformation from amitrole residue in soil.

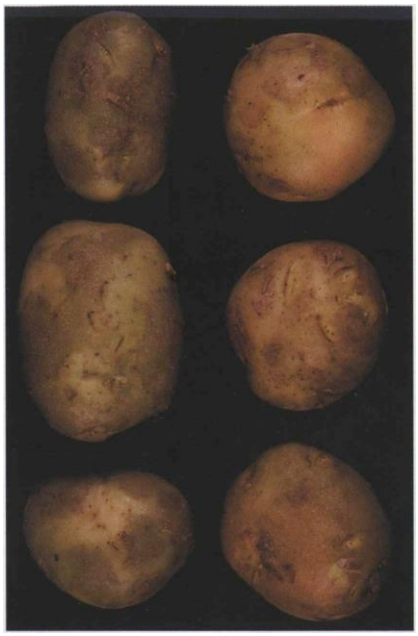


16.34d Internal sprouting; from storage above 16°C or from sprout inhibitor.

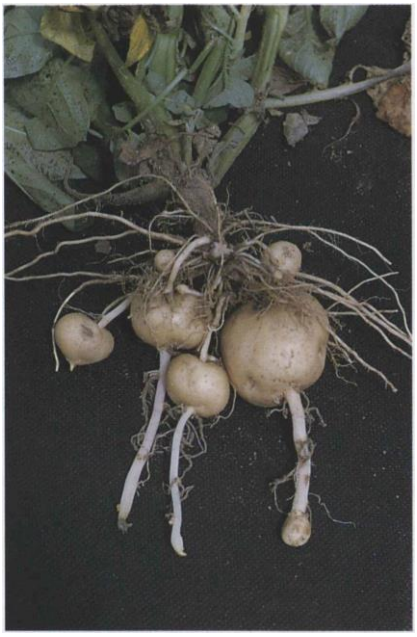
16.34e Leaf flecking; induced by highly acidic soil (left), healthy leaf (right).

16.34f Manganese deficiency; chlorotic foliar symptoms.

16.34g Secondary tubers; new tubers on seed-piece sprouts.



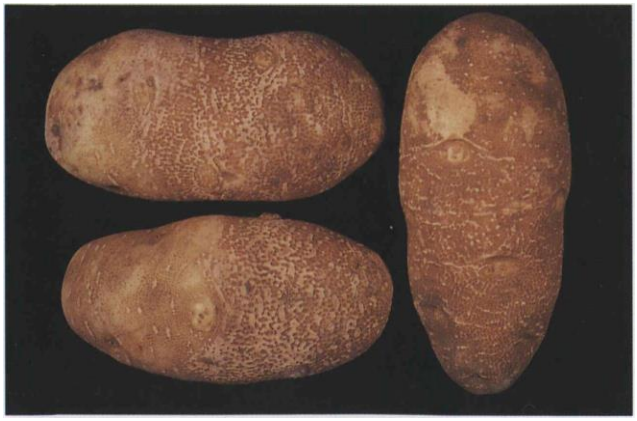
16.34h Tuber greening; chlorophyll and toxic alkaloids from exposure to light (left).



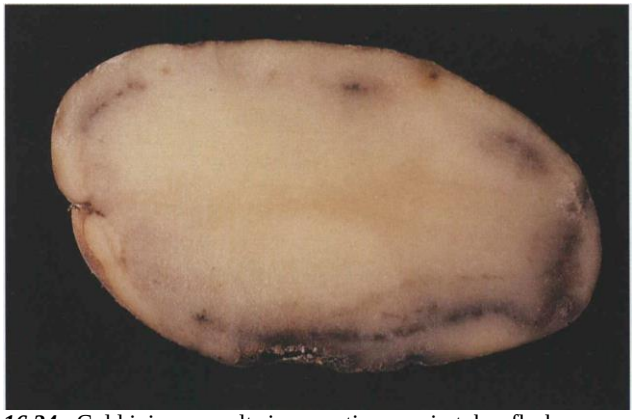
16.34i Sprouting; stolons from young tubers; induced by high soil temperatures.

16.34j Secondary tubers; new tubers on sprouts from mother tubers.

16.34k Stem-end browning; brownish streaks at stem end of affected tubers.



16.34m Enlarged lenticels; tuber symptoms, caused by wet soil conditions.



16.34n Cold injury; results in necrotic areas in tuber flesh.

16.35 Northern root-knot nematode; penetration of lenticels results in scab-like lesions.

16.36 Golden nematode; white or golden-yellow cysts on potato roots.

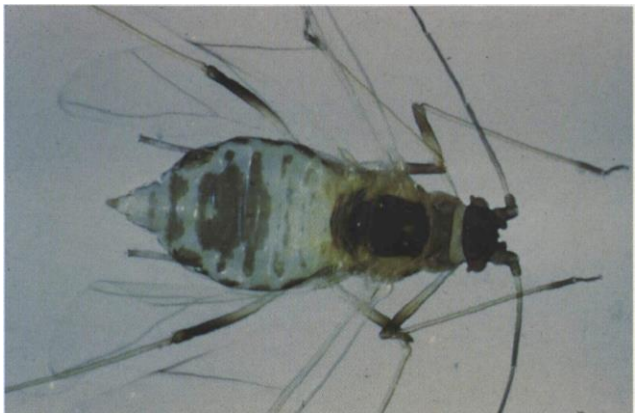
16.37 Potato-rot nematode; symptoms on tubers inoculated with U.S. isolates; not found in Canada since the 1960s.

16.38 Root-lesion nematode; yield in plots having 0 (left) to about 18000 nematodes per kg (8100/lb) of soil.

16.40a Buckthorn aphid; wingless adult and nymphs.

16.40b Buckthorn aphid; winged adult; length 1.2-2.0 mm.

16.41a Green peach aphid; wingless adults and a nymph.



16.41b Green peach aphid; winged adult; length 1.2-2.5 mm.

16.42a Potato aphid; aphids on potato leaf; color varies from yellow-green to pink.

16.42b Potato aphid; wingless adult; length 1.7-3.6 mm.

16.42c Potato aphid; winged adult; largest of the potato-colonizing aphids in Canada.

16.43 Foxglove aphid; nymph.

16.44a Colorado potato beetle; adults mating on foliage; length ± 10 mm.

16.44b Colorado potato beetle; eggs on underside of leaf.

16.44c Colorado potato beetle; larvae, and severe defoliation.

16.44d Colorado potato beetle; larva showing distinctive markings.

16.45a Potato flea beetle; leaflets scarred by adult feeding.

16.45b Potato flea beetle; leaf damage from adult feeding, resulting in shot-hole appearance.

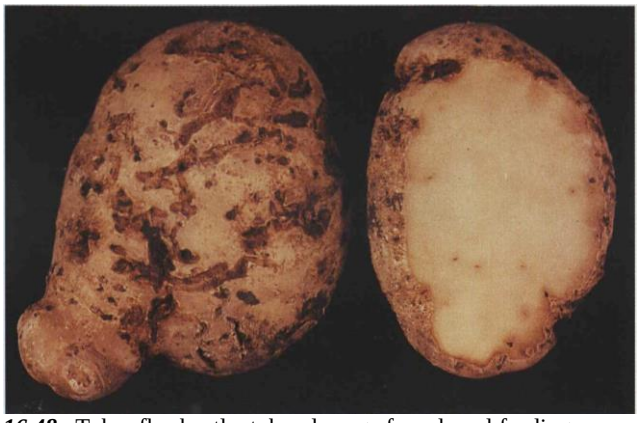
16.45c Potato flea beetle; adult; length ±1.7 mm.

16.46a Potato leafhopper; hopperbum symptom, caused by feeding.

16.46b Potato leafhopper; adult; length 3-4 mm.

16.47a Potato stem borer; female moths laying eggs; wingspan ± 42 mm.

16.47b Potato stem borer; eggs and newly hatched larva.

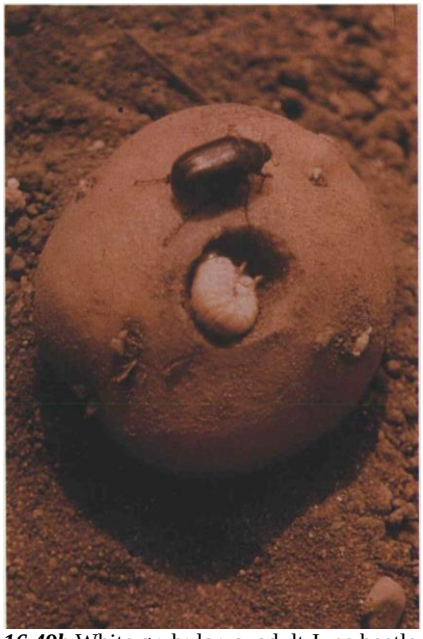


16.48a Tuber flea beetle; tuber damage from larval feeding.

16.48b Tuber flea beetle; partially peeled tuber showing damage to flesh.

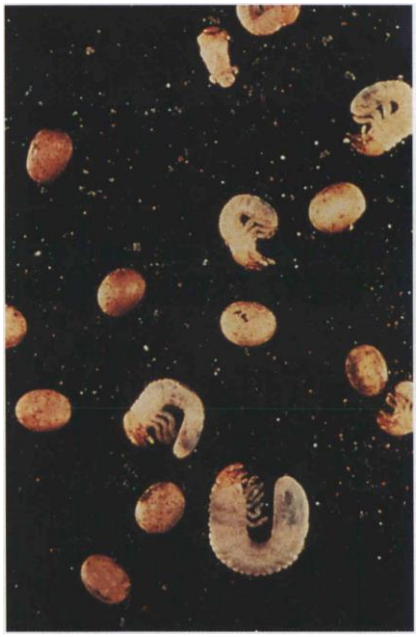
16.48c Tuber flea beetle; adults; length 1.5-2.0 mm.

16.49a White grub; damage to potato.



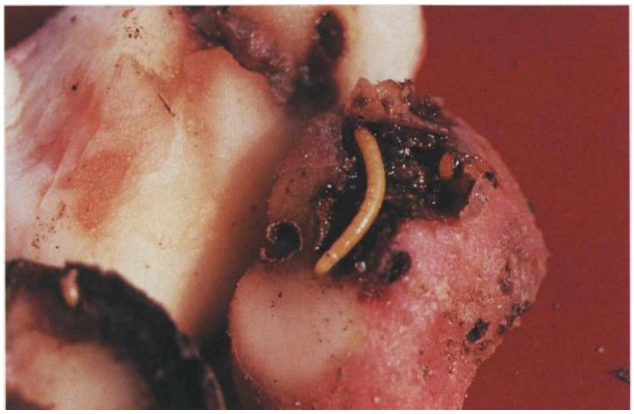
16.49b White grub; larva, adult June beetle, and damage from larval feeding.

16.49c White grub; adult June beetles; length ± 20 mm.



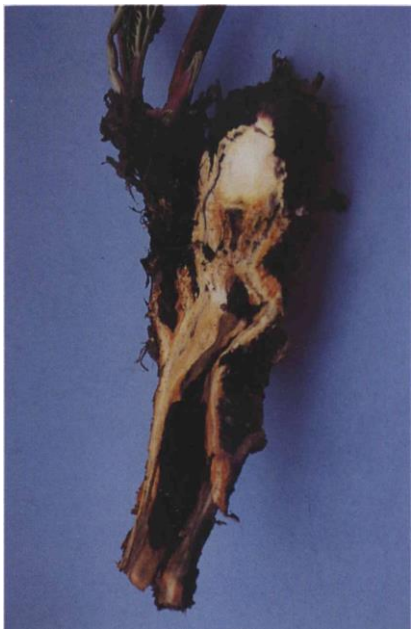
16.49d White grub; eggs and newly hatched larvae.

16.49e White grub; mature larva; length ±30 mm.



16.50 Wireworm; larvae and severe injury to a tuber.

17.1 Crown gall; note deformed crown with large yellow galls.



17.2a Red leaf; crown and root rot.

17.2b Red leaf; severe leaf symptoms.

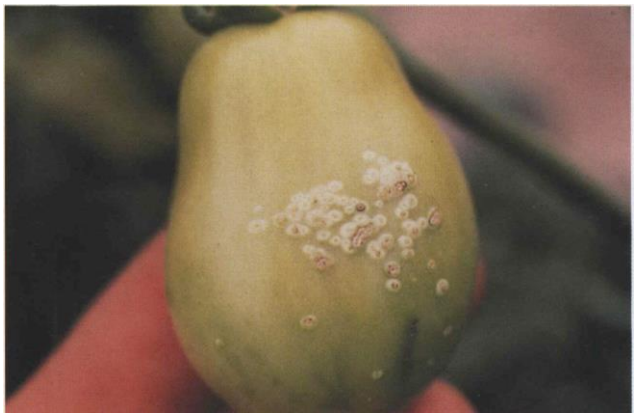
17.6a Ramularia leaf spot; leaf lesions.

17.6b Ramularia leaf spot; petiole lesions.

17.9 Viral disease; color mottling on a rhubarb leaf.

18.1a Bacterial canker; lesions on tomato leaves; see also *25.1*.

18.1b Bacterial canker; discoloration of stem tissues in tomato.

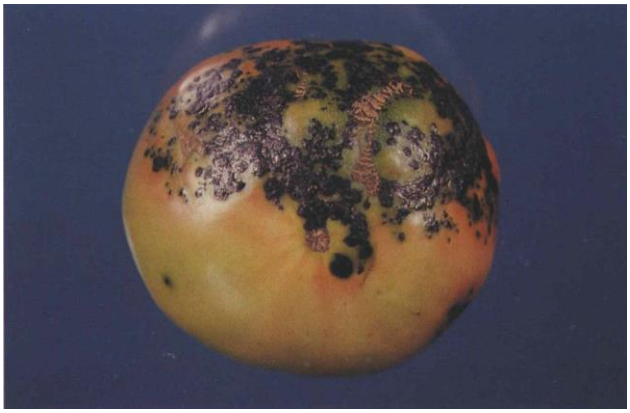


18.1c Bacterial canker; raised "bird's eye" lesions on a tomato fruit.

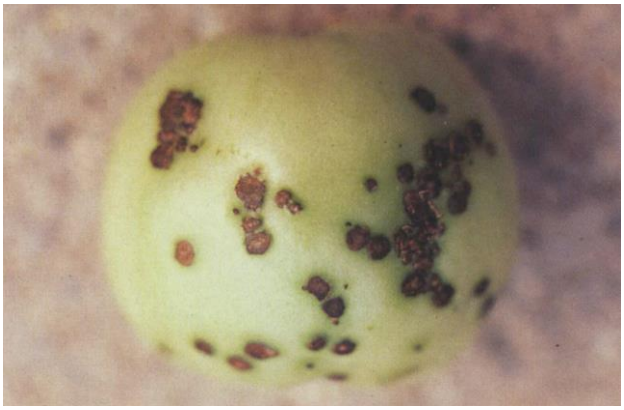
18.2 Bacterial soft rot; decay on ripe tomato fruits.

18.3a Bacterial speck; lesions on tomato leaves.

18.3b Bacterial speck; lesions on tomato fruits.



18.4a Bacterial spot; black lesions on tomato fruit.

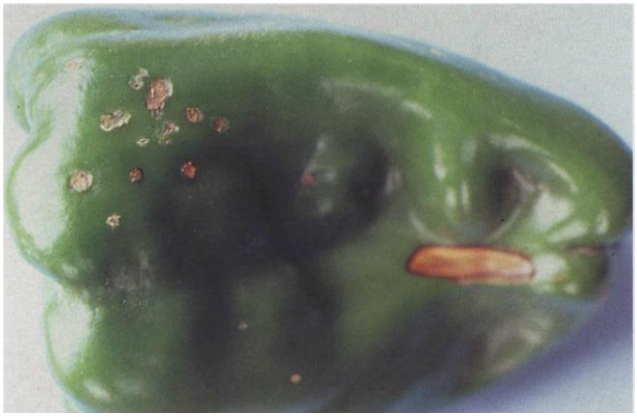


18.4b Bacterial spot; corky lesions on tomato fruit.

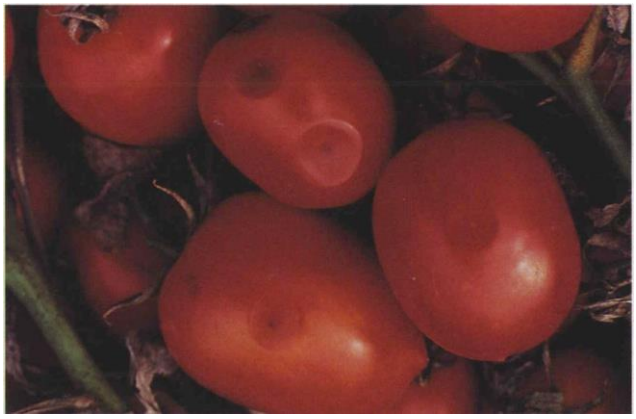
18.4c Bacterial spot; lesions on ripe tomato fruit.

18.4d Bacterial spot; lesions on pepper leaves.

18.4e Bacterial spot; lesions on pepper leaves.



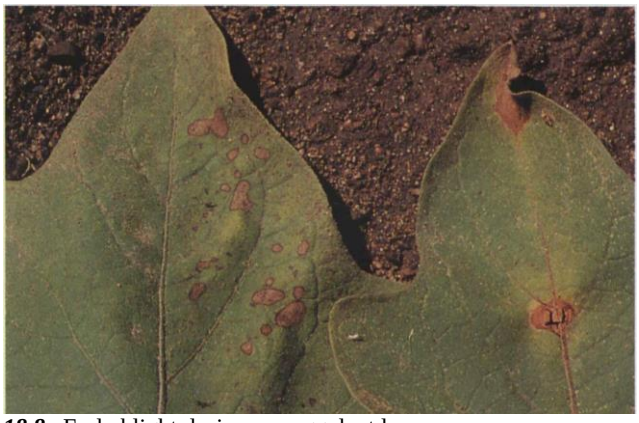
18.4f Bacterial spot; lesions on pepper fruit.



18.6a Anthracnose; young lesions on ripe tomato fruit.

18.6b Anthracnose; sunken lesion on pepper fruit.

18.6c Anthracnose; mature lesions with dark centers on ripe tomato fruit.

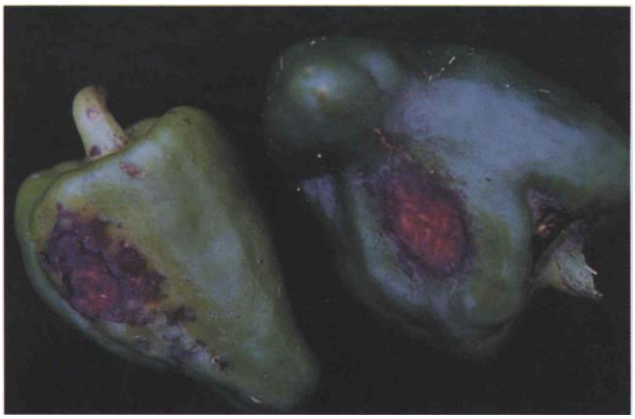


18.8a Early blight; lesions on eggplant leaves.

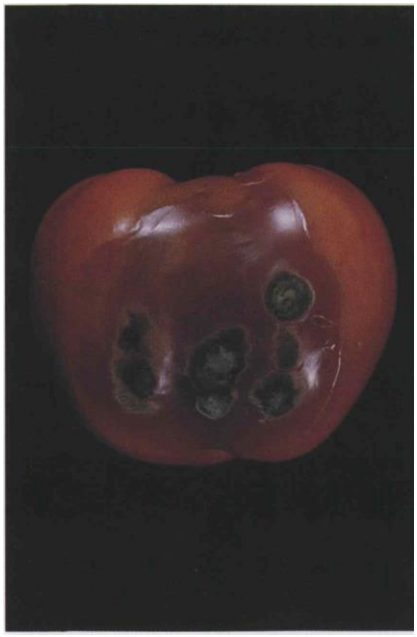
18.8b Early blight; characteristic target-spot lesions on tomato leaves.

18.8c Early blight; tomato fruit rot caused by *Alternaria solani*; characteristic blackened area at stem end.

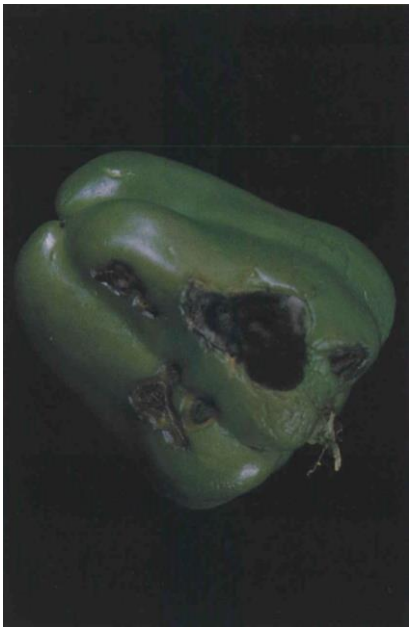
18.8d Early blight; lesions on eggplant fruit.



18.8e Early blight; lesions on pepper fruit.



18.8f Alternaria fruit rot; lesions on tomato fruit caused by *Alternaria alternata*.



18.8g Alternaria fruit rot; lesions on pepper fruit caused by *Alternaria alternata*.

18.11a Gray mold; leaf infection on tomato.

18.11b Gray mold; ghost spot lesions on tomato fruit; see also *25.12d.*

18.11c Gray mold; advanced decay of tomato fruit.

18.12a Late blight; characteristic lesion on tomato leaf.

18.12b Late blight; mature lesions on tomato leaf.

18.12c Late blight; stem necrosis on tomato.

18.12d Late blight; advanced decay of tomato fruit; note white mycelium of *Phytophthora infestans.*

18.13a Septoria leaf spot; lesions on tomato leaf.

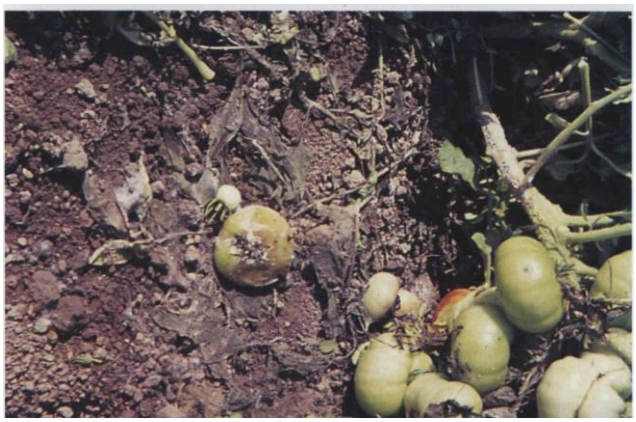
18.13b Septoria leaf spot; lesions with black pycnidia in tan centers.

18.13c Septoria leaf spot; lesions on tomato stem.

18.14a Verticillium wilt; general wilting, chlorosis and necrosis of eggplant foliage.

18.14b Verticillium wilt; early yellowing symptoms on eggplant leaves.

18.14c Verticillium wilt; wilted eggplant leaves with necrotic lesions.

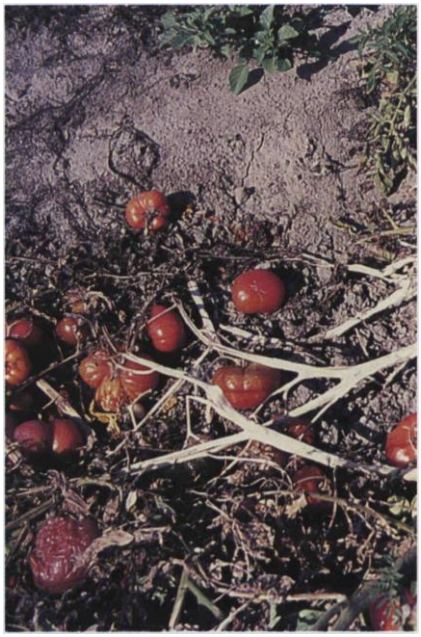


18.15a White mold; tomato stem and fruit infected by *Sclerotinia minor*.

18.15b White mold; collar rot on tomato plants caused by *Sclerotinia minor*.

18.15c White mold; lesions on tomato stem and petiole near ground level.

18.15d White mold; tomato stems with gray mycelium and black sclerotia of *Sclerotinia sclerotiorum.*



18.15e White mold; infected tomato stems often turn white.

18.17 Cucumber mosaic; dual infection with tobacco etch virus in pepper.

18.18a Tomato mosaic; distortion and narrowing of leaflets; see also *25.18a, 25.21a-c.*

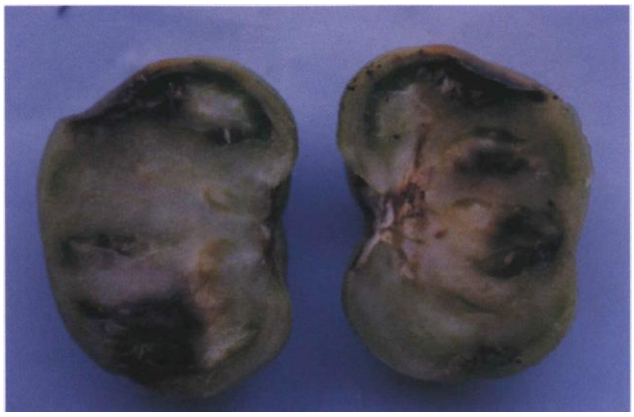
18.18b Tomato mosaic; lesions on tomato fruit.

18.18c Tomato mosaic; fruit lesions following early infection of cotyledons.

18.18d Tomato mosaic; grayish discoloration of green fruit caused by internal browning of the fruit wall.

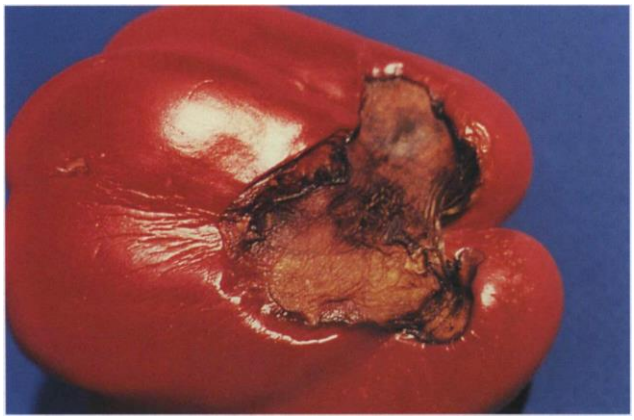
18.18e Tomato mosaic; internal browning of fruit wall near stem end.

18.21a Blossom-end rot; symptoms on tomato fruit.

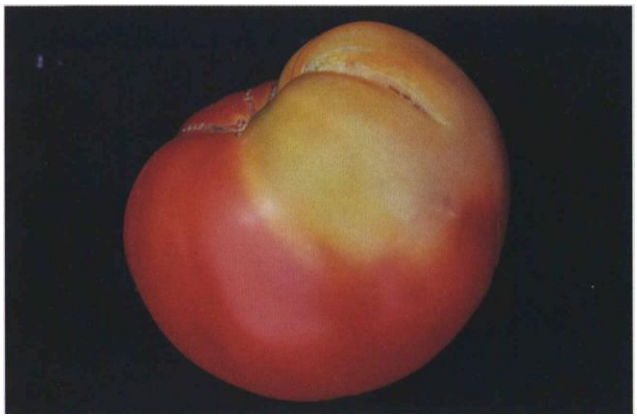


18.21b Blossom-end rot; internal breakdown of tomato fruit.

18.21c Blossom-end rot; early symptoms on pepper fruit.

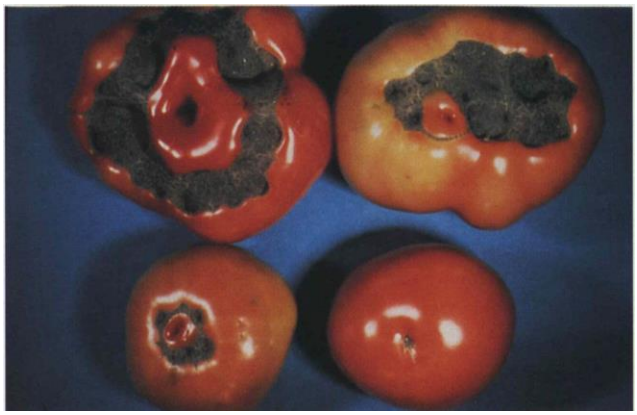


18.21d Blossom-end rot; sunken, brown lesion (advanced symptom) on pepper fruit.



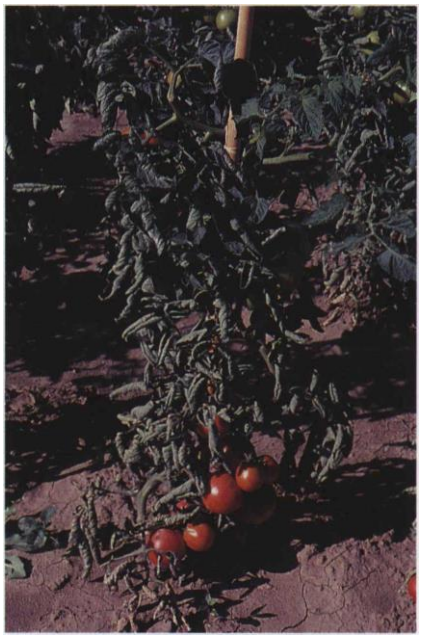
18.22a Blotchy ripening; external symptoms on a ripening tomato fruit.

18.22b Blotchy ripening; uneven coloration of internal tissues of a tomato fruit.



18.23 Catface; symptoms on ripe tomato fruit.

18.25 Growth cracks; radial and concentric cracks on tomato fruit.

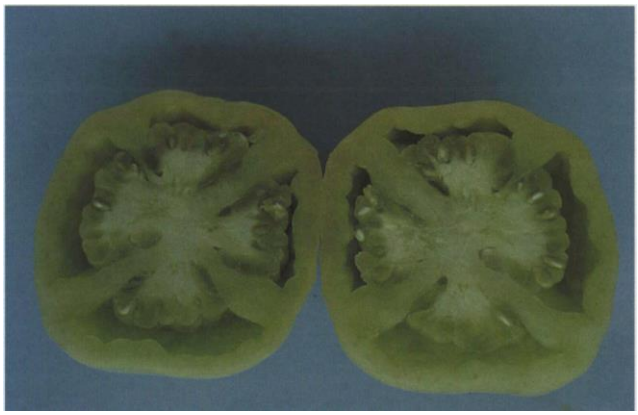


18.26a Leafroll; a response to moisture stress, cultivar related.

18.26b Herbicide injury; leaf cupping in tomato from 2,4-D drift.

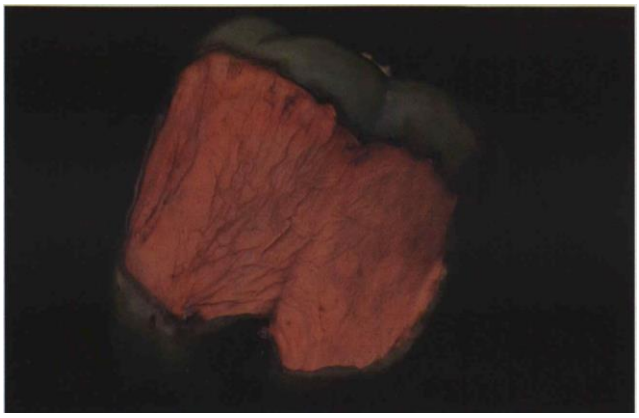
18.26c Herbicide injury; pepper fruit deformed by 2,4-D.

18.26d Herbicide injury (2,4-D); fruit (right) has over-developed placental tissues and lacks seeds and jelly.



18.28 Puffiness; symptoms in a green tomato fruit.

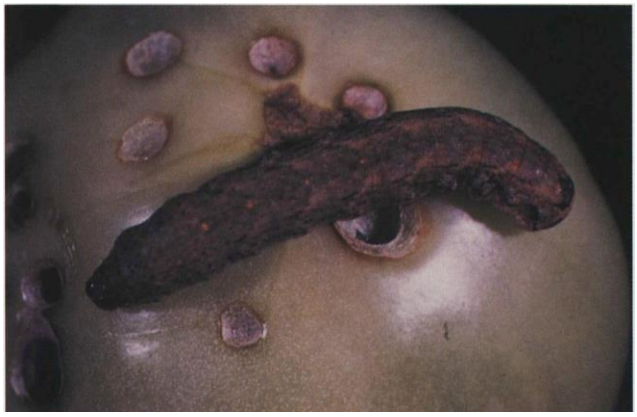
18.29a Sunscald; symptoms on tomato fruit.



18.29b Sunscald; severely affected pepper fruit.

18.30 Northern root-knot nematode; small galls and root branching symptoms on tomato.

18.35a Variegated cutworm; damage to tomato fruit.



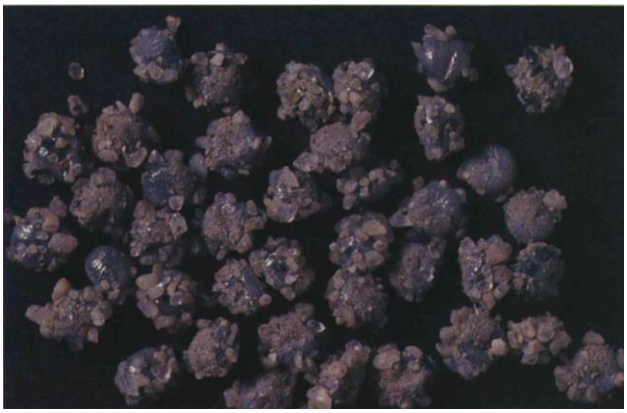
18.35b Variegated cutworm; larva feeding on tomato fruit.

18.35c Variegated cutworm; larvae, showing distinctive back and side markings.

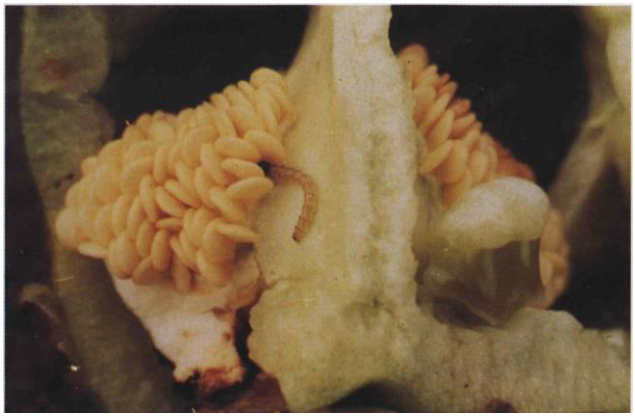
18.35d Dark-sided cutworm; larvae, showing range of color variation.

18.35e Dark-sided cutworm; pupa.

18.35f Dark-sided cutworm; adult; wingspan 35-40 mm.



18.35g Dark-sided cutworm; eggs with adhering soil.

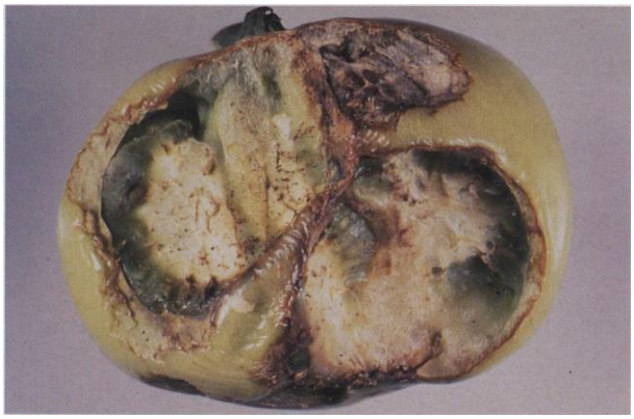


18.36a European corn borer; larva feeding in a pepper fruit.

18.36b European corn borer; larval entry under pepper stem cap; note sawdust-like frass around entry hole.

18.37a Hornworm; adult moth, tomato hornworm; wingspan ± 130 mm

18.37b Hornworm; larva (head at left).



18.37c Hornworm; extensive feeding damage on tomato fruit.

18.38a Pepper maggot; horse nettle (shown here) is an alternative host.

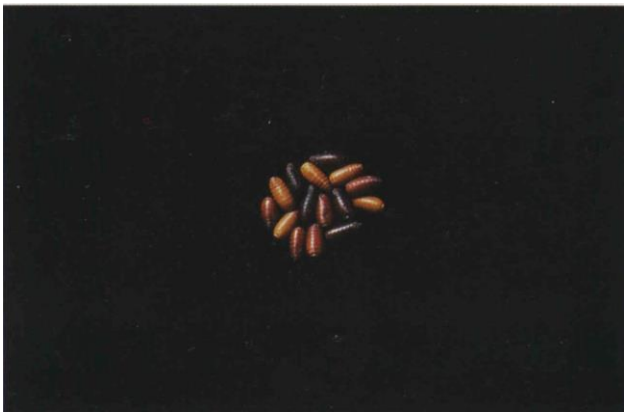
18.38b Pepper maggot; female laying an egg in pepper fruit.

18.38c Pepper maggot; damage to inside of pepper fruit.

18.38d Pepper maggot; adult female; length ±7.5 mm.

18.38e Pepper maggot; egg in pepper fruit (cutaway view).

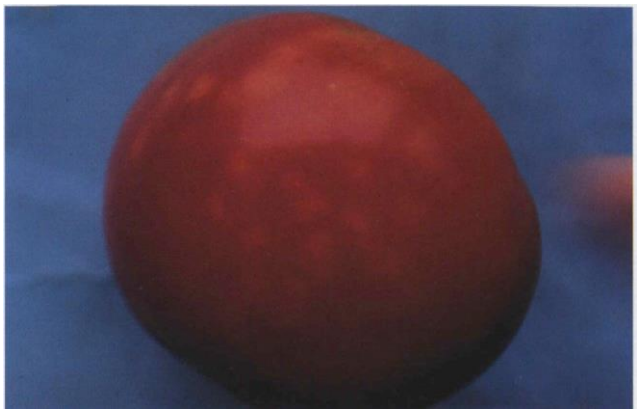
18.38f Pepper maggot; larva tunneling toward core of fruit after emerging from egg (above).



18.38g Pepper maggot; pupae, showing different shades of color; length ± 8 mm.

18.39 Sap beetle; adult, four-spotted sap beetle; length 4-7 mm.

18.40a Stink bug; adult (length 10-15 mm) and cloudy spot feeding damage on tomato fruit.



18.40b Stink bug; cloudy spot damage on ripe tomato fruit.

18.42a Tarnished plant bug; feeding injury to tomato fruit.

18.42b Tarnished plant bug; nymph, 1st instar.

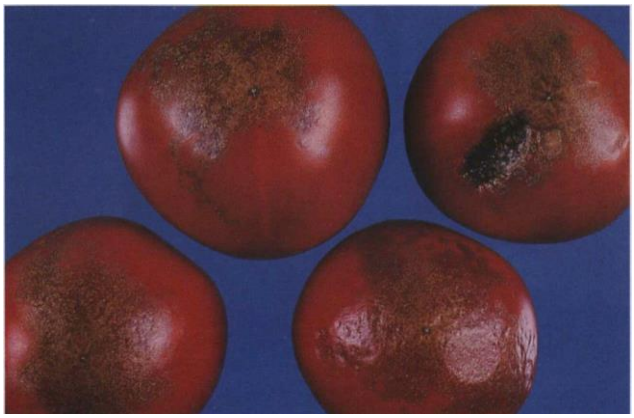
18.42c Tarnished plant bug; nymph, 5th instar.

18.42d Tarnished plant bug; adult; length 5-6 mm.

18.42e Tarnished plant bug; eggs.

18.42f Vinegar fly; larva ***(****Drosophila* sp.).

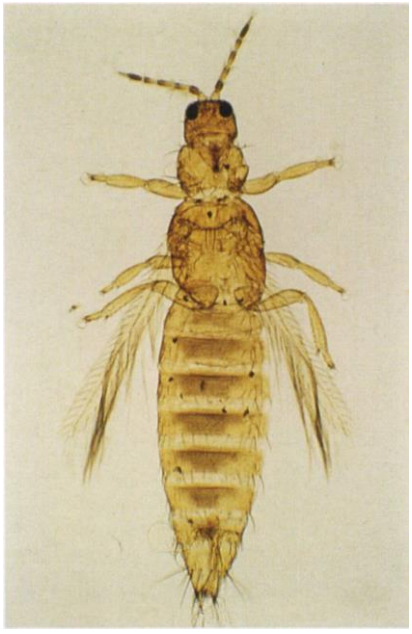
18.42g Vinegar fly; adult female ***(****Drosophila* sp.); length ±2.5 mm.



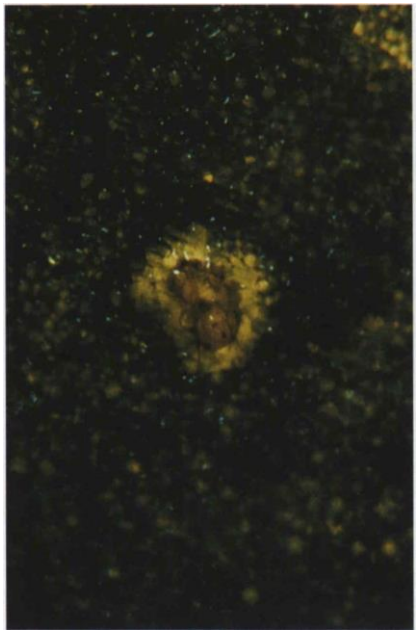
18.42h Western flower thrips; feeding damage to tomato fruit.

18.42i Western flower thrips; feeding damage to pepper fruit.

18.42j Western flower thrips; propupa; note wing buds; see also *22.34e*.



18.42k Western flower thrips; adult; length 1-2 mm.



18.42m Western flower thrips; eggs inserted in leaf; note egg-laying scar.

18.43 Gray garden slug; adults, slime trails and feeding damage on tomato fruit.

19.1 Gangrene; blackened fronds of fiddlehead (ostrich fern).

20.1a Alternaria blight; leaf lesions on a 1-year-old ginseng plant.

20.1b Alternaria blight; lesions near the soil line on a ginseng stem.

20.1c Alternaria blight; conidia of *Alternaria panax.*

20.2 Botrytis blight; leaf lesion on a 3-year- old ginseng plant.

20.3a Damping-off; ginseng seedling (left) killed by *Alternaria panax* stem infection.

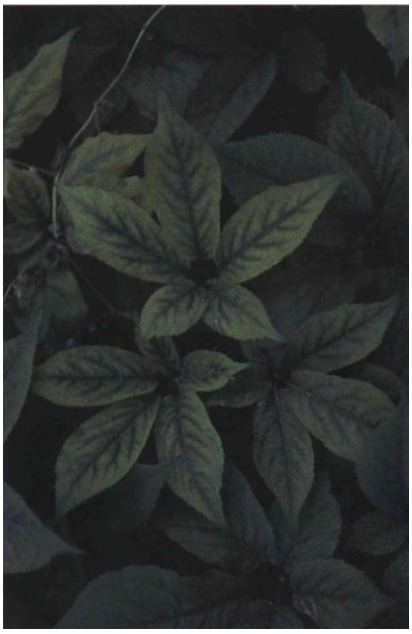
20.3b Damping-off; circular patches of ginseng plants lost to *Rhizoctonia solani* infection.

20.4a Disappearing root rot; superficial root rot lesions.

20.4b Disappearing root rot; advanced root decay.

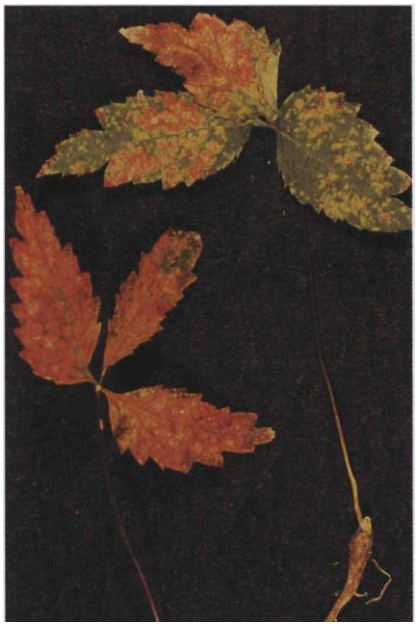
20.5 Phytophthora mildew and root rot; healthy and affected (right) roots.

20.6 Rusted root; raised, reddish brown lesions may girdle root.



20.9a Zinc deficiency; interveinal yellowing of leaves.

20.9b Phytotoxicity; mancozeb injury and sunscald.



20.9c Phytotoxicity; chemical injury on leaves.

20.9d Sunscald; injured foliage.

21.1a Apical chlorosis; severely affected plant.

21.1b Apical chlorosis; stunted, chlorotic plants in a field.

21.2 Downy mildew; leaf symptoms.

21.3 Powdery mildew; severe foliar symptoms.

21.4 Rust; overwintering telial stage on leaves.

21.5a Sclerotinia wilt; dead and dying plants.

21.5b Sclerotinia wilt; stem infection, light brown basal cankers.

21.6 Stem borer; foliar symptoms (chlorosis, necrosis) from feeding by sunflower maggot in stem.

22.1 Angular leaf spot; lesions delimited by veins, centers may fall out.

22.4a Black root rot; pale brown to black lesions.

22.4b Black root rot; roots with small, black sclerotia of *Phomopsis sclerotioides.*

22.7a Crown and root rot; orange-brown stem rot caused by *Pythium* sp. on cucumber grown in sawdust.

22.7b Damping-off; affected plant, showing constriction of stem near soil line.

22.7c Crown and root rot; orange-brown pythium rot may extend 10 cm up the stem.

22.7d Crown and root rot; sudden wilting may be first symptom noted.

22.8a Downy mildew; angular, chlorotic mottle on upper leaf surface.

22.8b Downy mildew; purple-brown sporangiophores of *Pseudoperonospora cubensis* on lower leaf surface.

22.9a Fusarium wilt; basal stem lesion.

22.9b Fusarium wilt; advanced wilt symptoms.

22.10a Gray mold; affected fruit with sporulation of *Botrytis cinerea.*

22.10b Gray mold; stem lesion with dark gray spores of *Botrytis cinerea*.

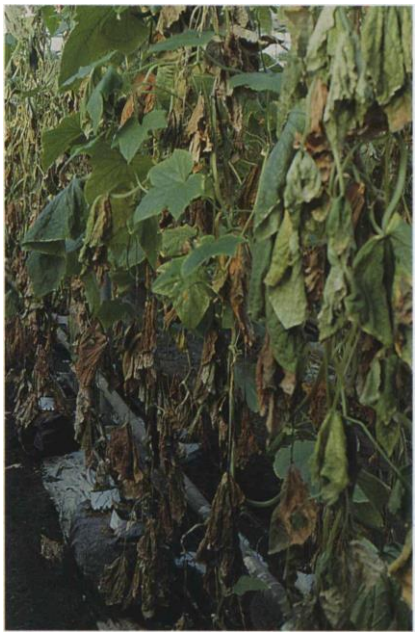
22.10c Gray mold; an infected leaf petiole.

22.10d Gray mold; sclerotia of *Botrytis cinerea* on an infected cucumber stem.

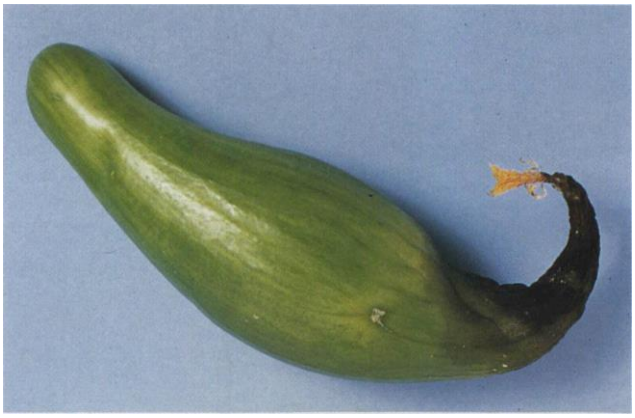
22.11a Gummy stem blight; stem canker with dark pycnidia of *Ascochyta cucumis.*

22.11b Gummy stem blight; a stem canker with droplets of amber- colored ooze on the surface.

22.11c Gummy stem blight; stem canker with dark pseudothecia of *Didymella bryoniae.*



22.11d Gummy stem blight; wilted plants.



22.11e Gummy stem blight; fruit with black *Ascochyta cucumis* pycnidia at blossom end.

22.12a Alternaria leaf blight; severely affected leaves.

22.12b Alternaria leaf blight; lesions on honeydew melon leaf.

22.13 Leaf rot (pink mold rot); lesions on affected leaves.

22.14a Penicillium stem rot; stem lesion, usually only at pruned nodes.

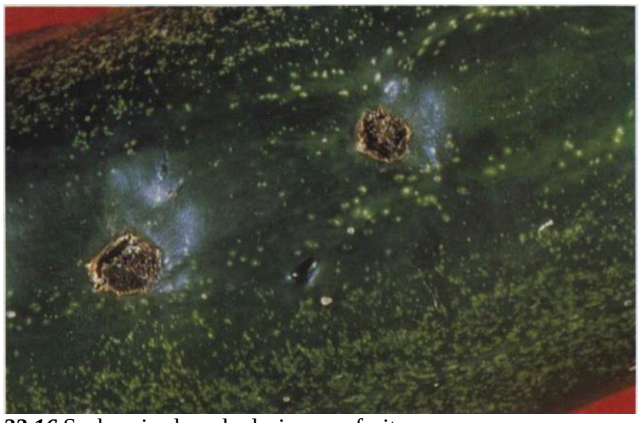
22.14b Penicillium stem rot; lesions on fruit, note blue-gray sporulation.

22.15a Powdery mildew; leaf symptoms with whitish fungal growth.

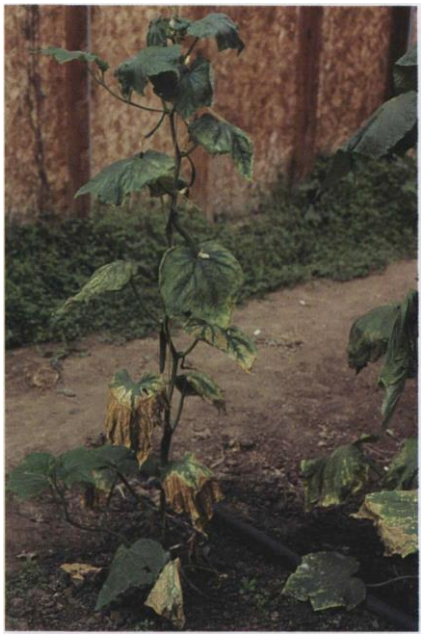
22.15b Powdery mildew; severely affected plant.

22.15c Powdery mildew; fruit infection of long English cucumber.

22.15d Powdery mildew; cleistothecia of *Erysiphe cichoracearum* on cucumber petiole.



22.16 Scab; raised, corky lesions on fruit.

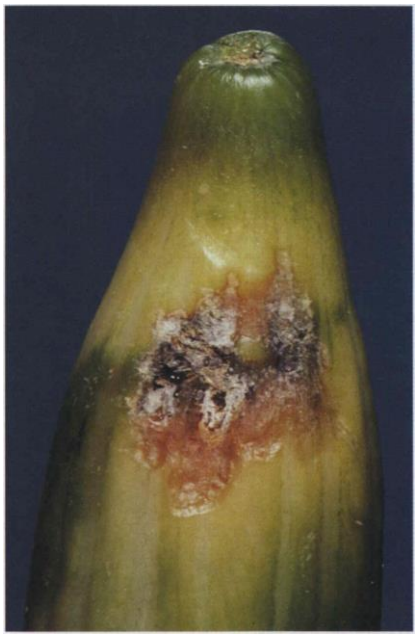


22.17a Verticillium wilt; wilting and chlorosis begin on lower leaves.

22.17b Verticillium wilt; characteristic V- shaped lesions on leaf.

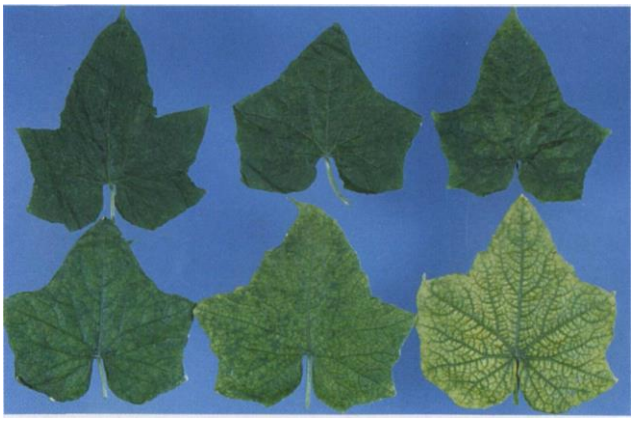
22.18a White mold; stem infection; note bleached appearance.

22.18b White mold; stem lesions with cottony white mycelium of *Sclerotinia*.



22.18c White mold; fruit infection.

22.18d White mold; advanced decay with white mycelium and black sclerotia.



22.19 Beet pseudo-yellows; interveinal chlorosis and yellowing of leaves.

22.20a Cucumber mosaic; severe mottling on leaves.

22.20b Cucumber mosaic; fruit mottling.

22.22 Cucumber pale fruit; pear-shaped distortion of fruit.

22.23 Watermelon mosaic; mosaic and distortion of a young leaf.

22.24a Zucchini yellow mosaic; leaf puckering.

22.24b Zucchini yellow mosaic; mosaic and distortion of leaves and fruit.

22.25a Cold injury; leaf puckering of cucumber seedlings from applying cold water.

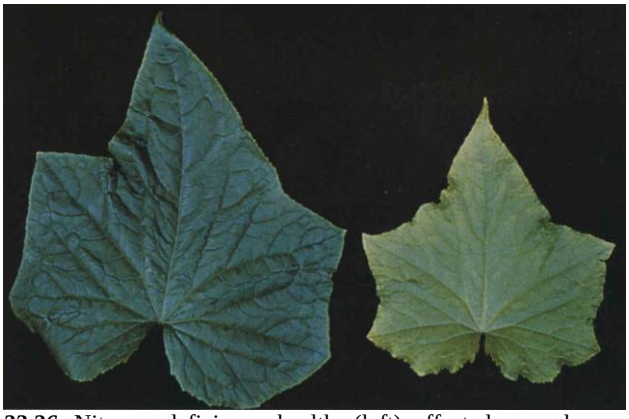
22.25b Chilling injury; scarring and curvature of fruit caused by cold storage.

22.26a Boron deficiency; healthy (left); note skin cracking on affected fruit.

22.26b Magnesium deficiency; chlorotic flecking on leaves.

22.26c Molybdenum deficiency; leaf scorch symptoms.

22.26d Iron deficiency; healthy (left); young affected leaves show severe interveinal yellowing.



22.26e Nitrogen deficiency; healthy (left); affected young leaves are small and pale yellow-green.

22.26f Nitrogen deficiency; affected small fruit (top) has a pointed blossom end.

22.26g Potassium deficiency; chlorosis of leaf margins may be followed by bronzing and scorching.

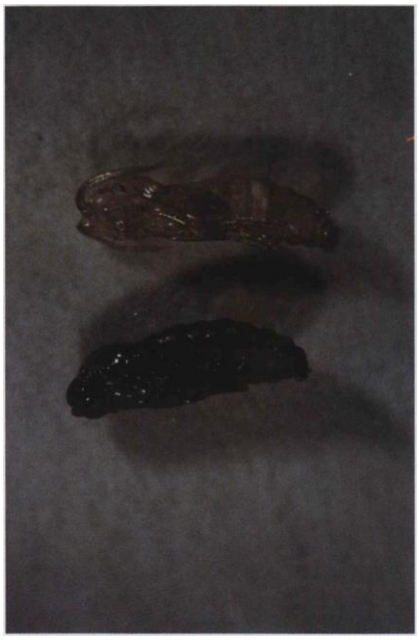
22.28 Root death; browning of cucumber roots growing in an NFT system.

22.30a Southern root-knot nematode; stunted (center) and healthy plants.

22.30b Southern root-knot nematode; severely wilted cucumber plants.

22.30c Southern root-knot nematode; galls on roots of greenhouse cucumber.

22.30d Northern root-knot nematode; healthy (left) and infested roots.

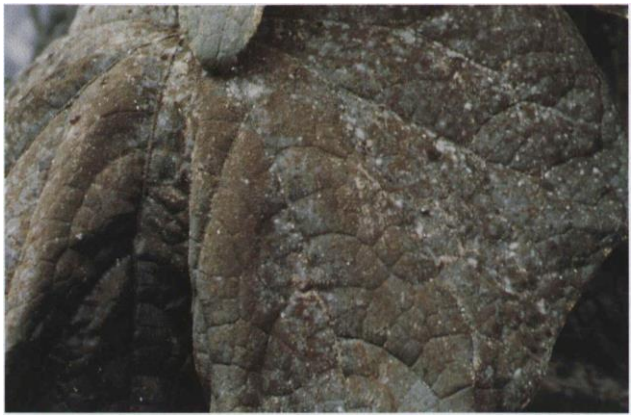


22.31a Fungus gnat; pupae.

22.31b Fungus gnat; adults and larvae; adult 2-3 mm long.

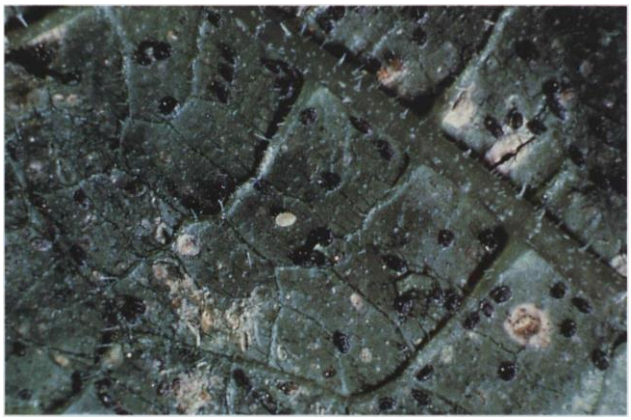
22.31c *Hypoaspis* sp.; a mite predator of fungus gnats and western flower thrips.

22.32a Greenhouse whitefly; adult-infested leaf.



22.32b Black sooty mold; on honeydew produced by greenhouse whitefly.

22.32c Greenhouse whitefly; adult; length ± 1.5 mm.



22.32d Greenhouse whitefly; black scales are pupae parasitized by *Encarsia formosa'*, note a healthy (white) pupa in center.

22.33a Melon aphid; winged adult.

22.33b Melon aphid; wingless adult and nymphs.

22.34a Western flower thrips; leaf damage and frass (black dots).

22.34b Western flower thrips; fruit scarring resulting from feeding injury.

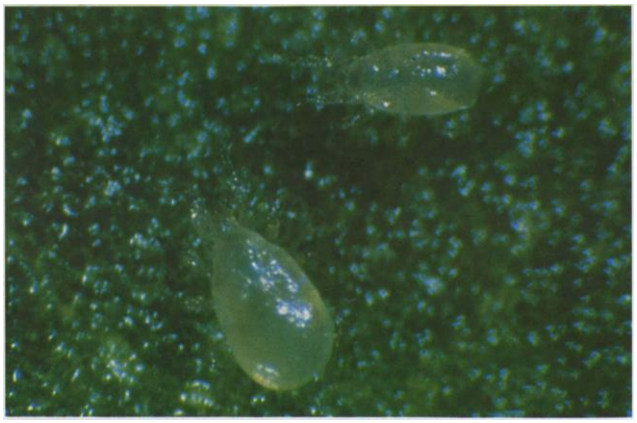
22.34c Western flower thrips; fruit curvature resulting from feeding injury.

22.34d Western flower thrips; yellow sticky traps for monitoring thrips and greenhouse whitefly in a greenhouse cucumber crop.

22.34e Western flower thrips; early propupa; note wing buds and eye pigmentation (see also *18.42j).*

22.34f Western flower thrips; pupa.

22.34g Western flower thrips; adult; length 1-2 mm.



22.34h *Amblyseius cucumeris*; a mite predator of western flower thrips.

22.34i Minute pirate bug; predator of western flower thrips; length 3-5 mm.

22.35a Chrysanthemum leafminer; leaf damage.

22.35b Chrysanthemum leafminer; adult flies on cotyledons of cucumber.

22.35c Onion thrips; adult on cucumber leaf; length 1.0-1.2 mm.

22.35d Plant bug; adult *Lygus* sp.; length 2-10 mm.

22.36a Two-spotted spider mite; damage to leaves.

22.36b Two-spotted spider mite; leaf yellowing from feeding injury.

22.36c Two-spotted spider mite; infested leaf, showing silvering and mites in webbing.

22.36d Two-spotted spider mite; winter phase (red-orange) on cucumber leaves.

22.36e Two-spotted spider mite; adult, summer phase (pale green).

22.36f Two-spotted spider mite; adult, winter phase (red-orange).

22.36g *Phytoseiulus persimilis;* a mite predator of two-spotted spider mite.

23.6a Damping-off; a severely affected seedling.

23.6b Damping-off; root rot symptoms.

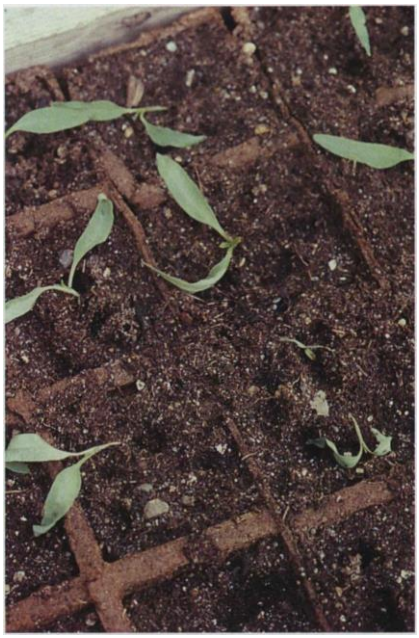
23.9 Gray mold; decay of leaves in the center of a head.

23.10 Powdery mildew; a chlorotic lesion on a lettuce leaf.

23.14 Lettuce mosaic; leaf puckering and mottling.

23.15 Tomato spotted wilt; systemic necrosis leading to complete breakdown of the heads.

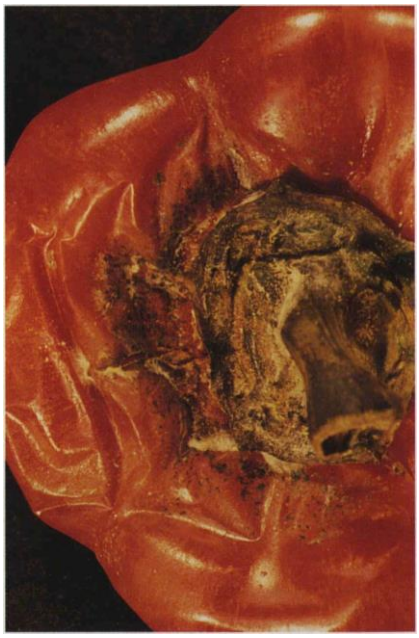
23.16 Tipburn; necrosis of the leaf margins, a result of calcium deficiency.



24.1 Damping-off; wilted seedlings.

24.2a Fusarium stem and fruit rot; early stem infection at node.

24.2b Fusarium stem and fruit rot; canker with mycelium and perithecia *(Nectria).*



24.2c Fusarium stem and fruit rot; fruit rot with perithecia of *Nectria haematococca*.

24.5a Pepper mild mottle; mottling of young leaves.

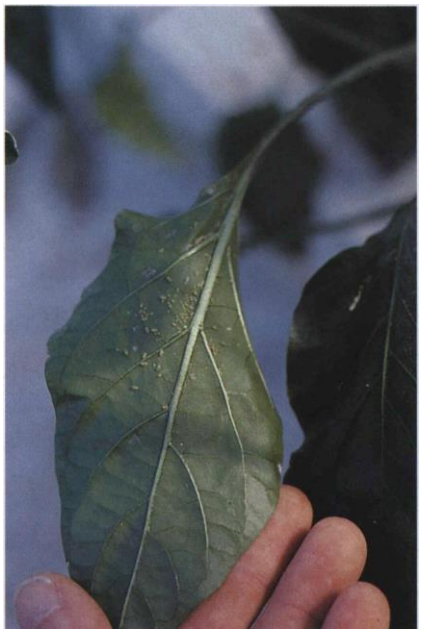
24.5b Pepper mild mottle; color mottling on pepper fruit.

24.8a Tomato spotted wilt; necrotic stem lesions.

24.8b Tomato spotted wilt; leaf mottling, distortion and rosetting.

24.8c Tomato spotted wilt; ring patterns and uneven ripening of fruit.

24.9 Blossom-end rot; sunken lesions on fruits; see also *18.21c,d.*



24.12a Green peach aphid; infested pepper leaf.

24.12b Green peach aphid; wingless adults, nymphs, and a molted skin (center).

24.12c Green peach aphid; mummified adult parasitized by a wasp.

24.12d Predatory midge, *Aphidoletes* sp.; larva attacking aphid.

24.12e Black sooty mold growing on aphid honeydew.

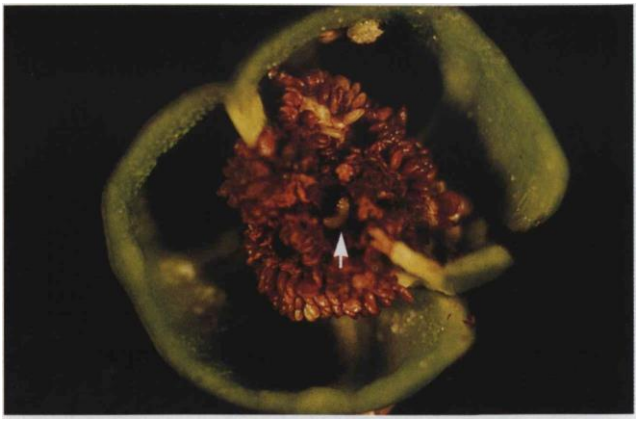
24.13a Pepper weevil; feeding damage to leaf by adult.

24.13b Pepper weevil; exit hole left by adult in young fruit.

24.13d Pepper weevil; pupa in pupal cell in fruit.

24.14a Western flower thrips; damage to the growing tip causes leaf deformity.

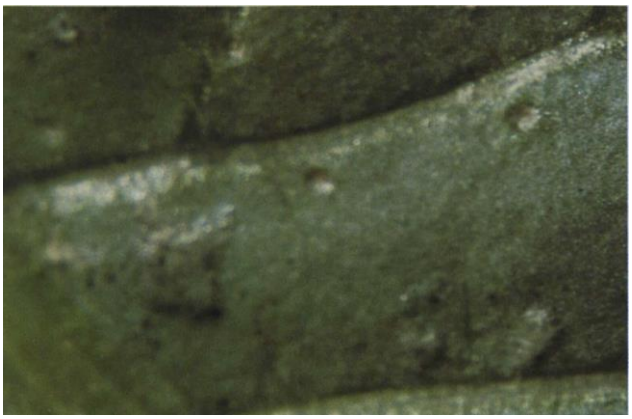
24.14c Western flower thrips; egg-laying scar and "ghost spot" on a fruit.



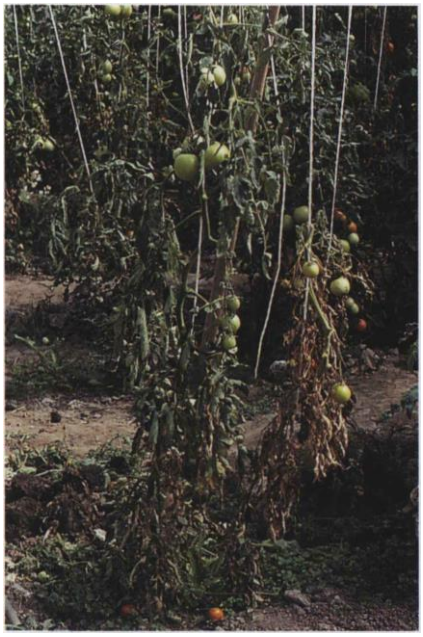
24.13c Pepper weevil; damage to fruit; note brown seeds and larva in feeding cavity (center).

24.13e Pepper weevil; adult; length 2.5-3.1 mm.

24.14b Western flower thrips; damage to fruit under and around the calyx exposes the fruit to bacterial infection.



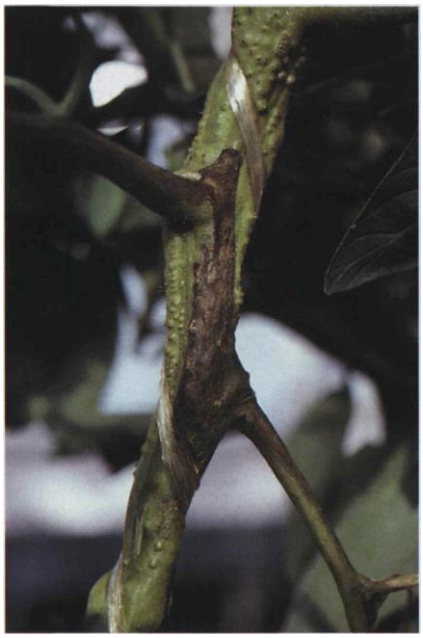
24.14d Western flower thrips; egg-laying scars on leaf.



25.1 Bacterial canker; wilted plants; see also *18.1a-c.*

25.3 Bacterial stem rot; petiole and leaf symptoms.

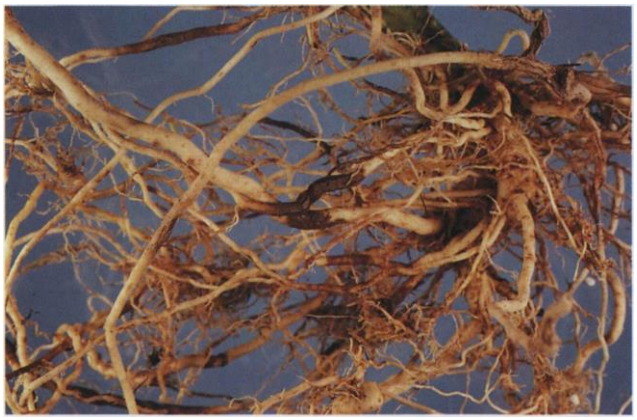
25.4a Pith necrosis; plant, showing chlorosis and wilt.



25.4b Pith necrosis; brown lesion on stem, often with many adventitious roots.

25.4c Pith necrosis; discoloration and collapse of pith tissues.

25.5 Stem necrosis; stem lesion, internal discoloration.



25.6a Corky root; lesions on roots.

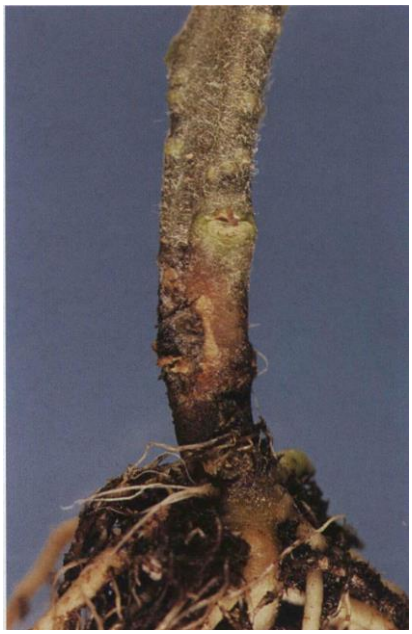
25.6b Corky root; corky lesion showing splits in surface tissue.

25.7 Damping-off; affected seedlings.

25.8 Didymella stem canker; lesion on stem near soil line.

25.9a Early blight; "target spot" leaf lesions; see also *18.8.*

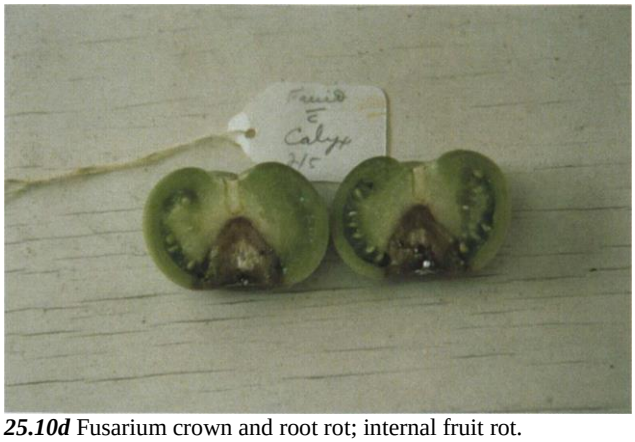
25.9b Early blight (left) and septoria leaf spot (right); typical leaf symptoms.



25.10a Fusarium crown and root rot; basal stem canker.

25.10b Fusarium crown and root rot; internal browning of lower stem.

25.10c Fusarium crown and root rot; fruit rot caused by *Fusarium oxysporum* f. sp. *radicis-lycopersici.*



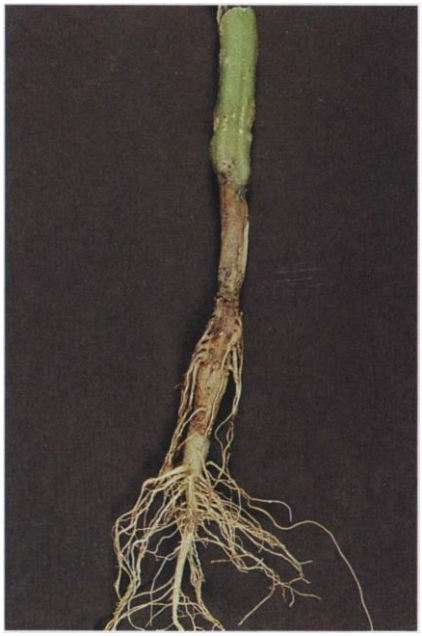
25.10d Fusarium crown and root rot; internal fruit rot.

25.11a Fusarium wilt; wilted plant, showing chlorotic leaves.

25.11b Fusarium wilt; wilted plant.

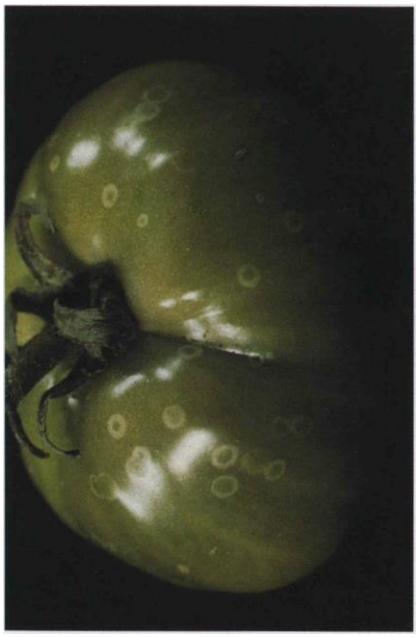
25.11c Fusarium wilt; brown vascular discoloration in stem.

25.12a Gray mold; stem canker with sporulating *Botrytis cinerea.*



25.12b Gray mold; basal stem canker.

25.12c Gray mold; severely affected plants.

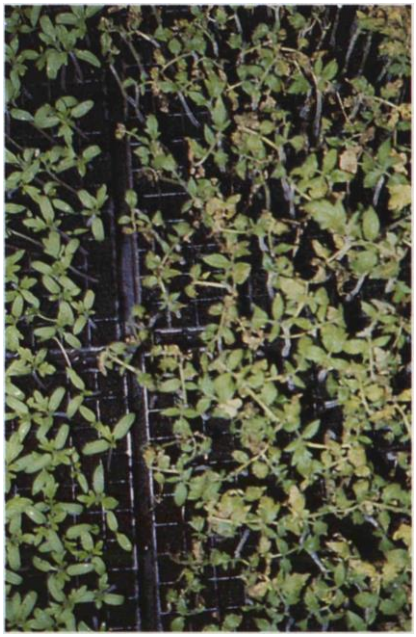


25.12d Gray mold; ghost spots on a green fruit; see also *18.11b*.

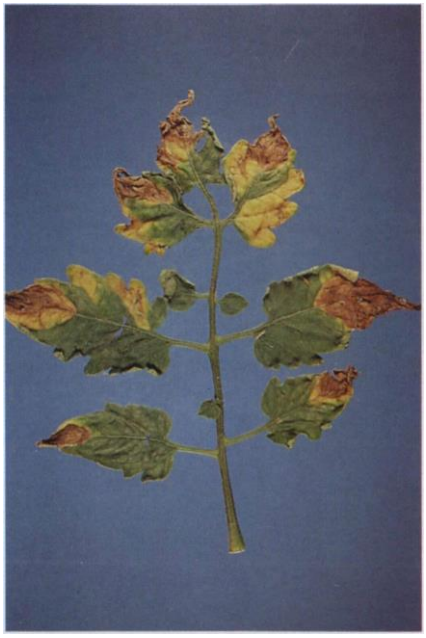
25.13a Late blight; leaf and fruit lesions.

25.13b Late blight; fruit rot.

25.14 Leaf mold; lesions on underside of leaf.



25.15 Septoria blight; blighted tomato seedlings; see also *18.13.*



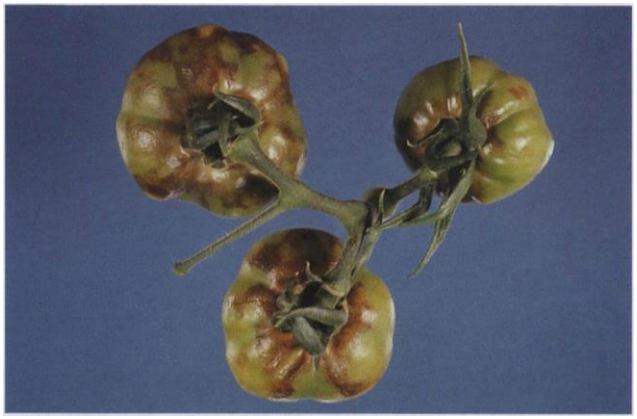
25.16a Verticillium wilt; leaf chlorosis and dieback.

25.16b Verticillium wilt; wilted tomato plant.

25.18a Cucumber mosaic; severe "shoestring" symptom; leaf blade is reduced or absent; see also *18.18a.*

25.18b Cucumber mosaic; mosaic symptoms on leaf.

25.19a Double streak; brown streaks on stem infected by potato virus X and tomato mosaic virus.



25.19b Double streak; dark lesions on fruit.

25.21a Tomato mosaic; stunted growth and pale-colored foliage.

25.21b Tomato mosaic; mosaic symptoms on leaves; see also *18.18a*.

25.21c Tomato mosaic; infected plants (between tall, healthy ones) are stunted and have small, fern-like leaves.

25.21d Tomato mosaic; internal and external symptoms on fruit.

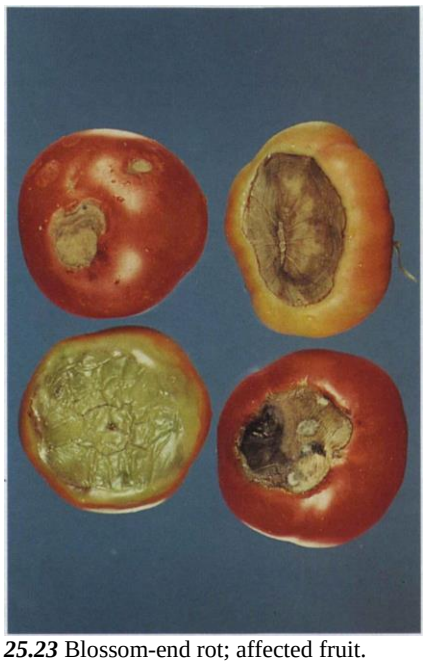
25.21e Tomato mosaic; severely affected (right) and healthy fruit.

25.22a Tomato spotted wilt; foliar chlorosis and necrosis.

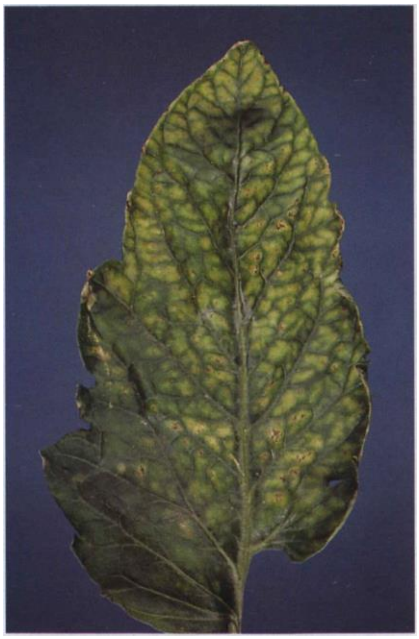
25.22b Tomato spotted wilt; severe foliar symptoms (leaf bronzing).

25.22c Tomato spotted wilt; discoloration of ripening fruit.

25.22d Tomato spotted wilt; blemished fruit of cultivar Jumbo.

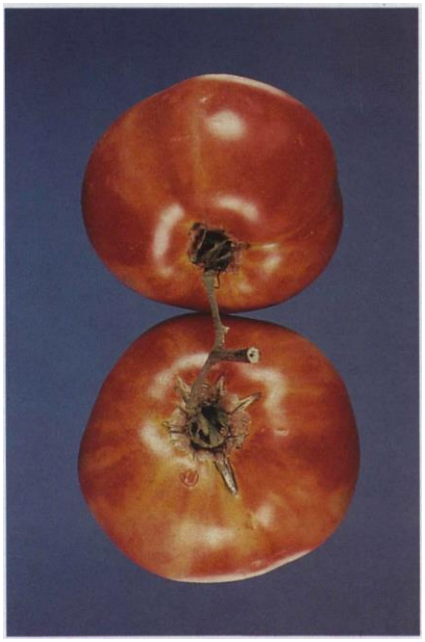


25.23 Blossom-end rot; affected fruit.

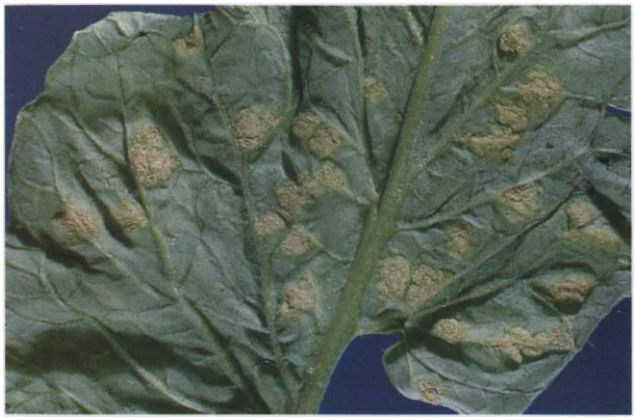


25.24a Magnesium deficiency; leaf symptoms; note upward cupping of margin.

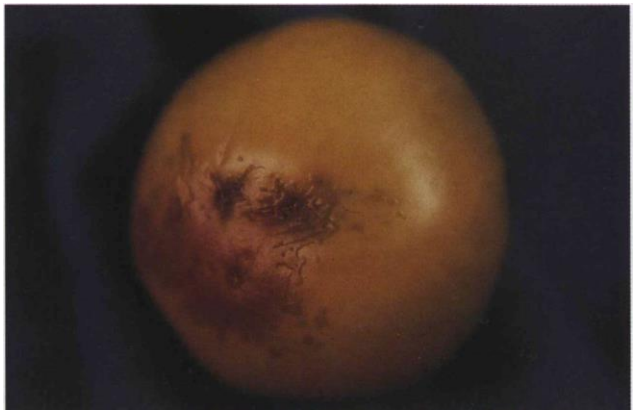
25.24b Magnesium deficiency; severe symptoms; veins, margins remain green.



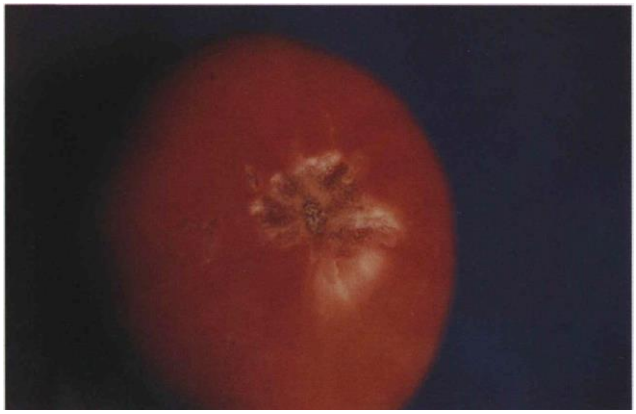
25.25a Blotchy ripening; moderately severe symptoms; see also *18.22.*



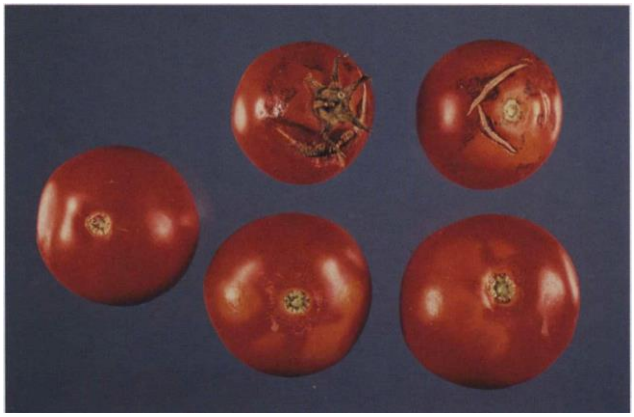
25.25b Edema; blistered areas on underside of leaf.



25.25c Edema; blisters on blossom-end of fruit.



25.25d Edema; on ripe fruit.

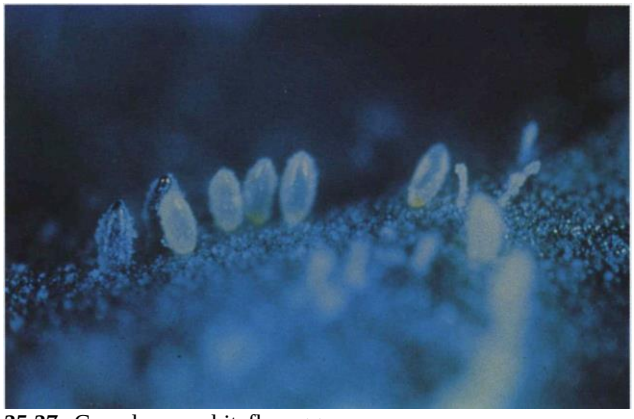


25.25e Growth cracks; healthy and affected fruit.

25.26 Root-knot nematodes; southern (left) and northern (right); note larger galls on plant at left.

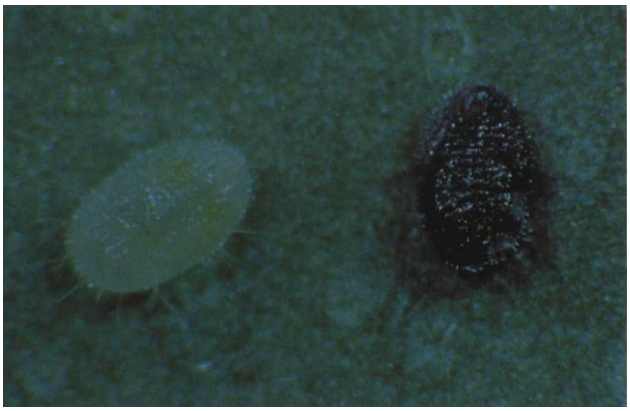
25.27a Greenhouse whitefly; infested leaf.

25.27b Greenhouse whitefly; adult; length ± 1.5 mm.

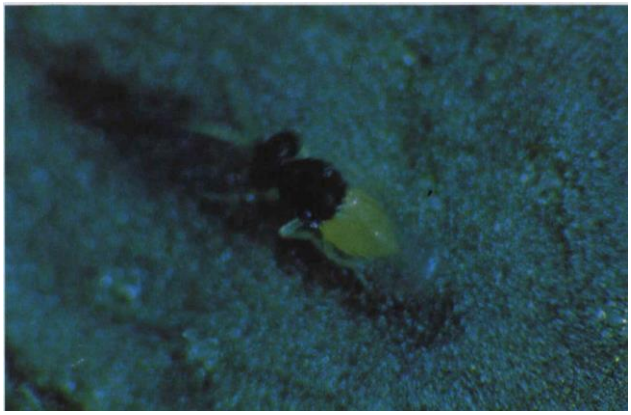


25.27c Greenhouse whitefly; eggs.

25.27d Greenhouse whitefly; immature (nymphal and pupal) stages.



25.27e Greenhouse whitefly; pupae, parasitized by *Encarsia formosa* (black), non-parasitized (white).

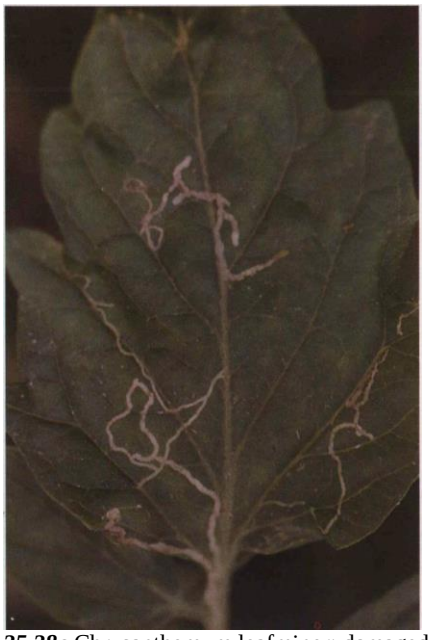


25.27f *Encarsia formosa*, adult; a parasite of the greenhouse whitefly.

25.27g Black sooty mold growing on honeydew produced by greenhouse whitefly.

25.28a Chrysanthemum leafminer; adults feeding, and egg-laying scars.

25.28b Chrysanthemum leafminer; infested (mined) leaves.



25.28c Chrysanthemum leafminer; damaged tomato leaf.

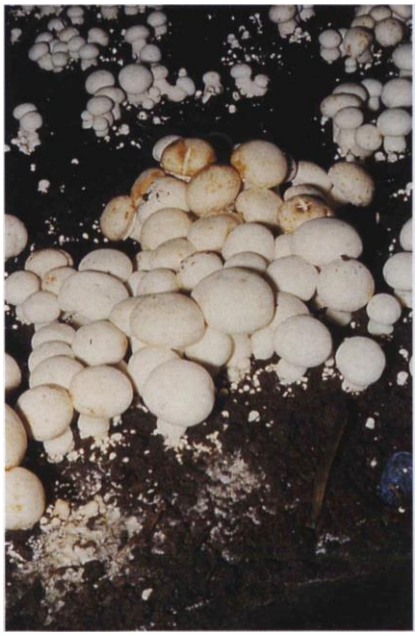
25.28d Chrysanthemum leafminer; adult fly; length ±2.5 mm.

25.28e Braconid wasp; a leafminer parasite resting on a mined leaf; note leaf mine with frass (dark line).

25.29 Western flower thrips; adult; length 1-2 mm.

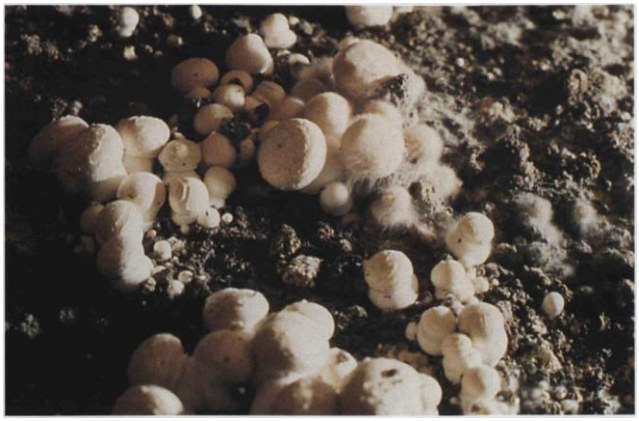
25.32 Two-spotted spider mite; summer phase (below), and its predator *P. persimilis.*

25.31 Tomato russet mite; leaf damage.



26.1 Brown blotch; yellow to brown discoloration of caps.

26.2 Mummy; mushrooms dwarfed with slightly curved stem, tilted cap, and mycelial overgrowth of swollen base.



26.3 Cobweb; cottony growth habit of the pathogen *Cladobotryum dendroides*.

26.4a Green mold; distinctive green color of sporulating *Trichoderma* sp.

26.4b Green mold; white, fluffy mycelium of *Trichoderma* sp. grows on compost that has not been properly prepared.

26.5 Mat; white to yellow mycelial mats of *Chrysosporium* sp. on wooden sideboards of a mushroom production tray.

26.6 Sepedonium yellow mold; characteristic yellow color develops in compost after three weeks of harvest.

26.7 Truffle; *Diehliomyces microsporus* mycelium forms dense knots in compost.

26.8 Dry bubble; sunken lesions on caps turn brown, mushrooms may be distorted.

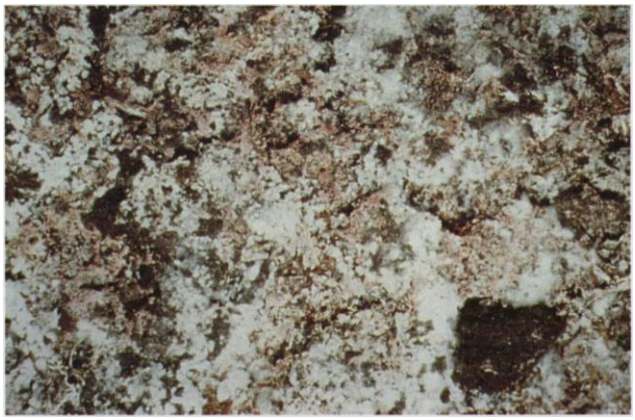
26.9 Wet bubble; mushrooms fail to develop normally and form a ball- or coral-like mass.

26.11 Viral diseases; infected mycelium may have difficulty growing into the casing, leaving patches with no mushrooms.

26.12 Ink caps; *Coprinus* spp. grow quickly, eventually becoming black and liquefying.

26.13 Olive-green mold; fruiting bodies (top) resemble gray-green cockleburs.

26.16 Black whisker mold; spore-bearing bristles on incompletely composted straw.



26.18 Lipstick mold; Sporendonema purpurascens is red to buff and grows on casing or compost.

26.19 Brown plaster mold; white colony with brown center; spores feel smooth; spores of brown mold (26.17) feel gritty.

26.20 Cinnamon brown mold; small, saucer-shaped fruiting bodies of the perfect state Peziza ostracoderma.

27.1a Bacterial soft rot of alfalfa sprouts; fast-spreading, smelly rot destroys seedlings.

27.1b Bacterial soft rot of alfalfa sprouts; rot begins with yellowish, translucent appearance of roots.

27.2 Bacterial soft rot of bean sprouts; healthy (left) and decayed sprouts.

Index

Indexed items are referenced by section number, which consists of the chapter number followed by the number of a major topic, such as a disease or pest description. **The number, *in bold italic*, of each illustration is the same as that of the corresponding section in the text;** for sections having more than one illustration, letters follow the section number; for example, the color illustrations for Colorado potato beetle (text section 16.44) are figures *16.44a* to *16.44d.* For text figures (line drawings and halftones), the figure number includes the letter T (e.g. *16.44T1).* To aid in finding items in the text, the running heads for each two-page spread identify the inclusive section numbers beginning on those pages; the number in the running head for a left-hand page is that of the first section beginning on that page, while the running head for the facing (right-hand) page carries the number of the last section beginning on that page.

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About the book

An illustrated guide to identifying destructive diseases and pests affecting vegetable crops in fields, gardens, greenhouses and other environments.

An indispensable manual for commercial growers, crop advisors, market gardeners, diagnosticians, teachers, master gardeners and students.

A unique collection of more than 1000 full-color photographs of infectious diseases, environmental disorders, nematode injury, and damage from insects, mites, slugs and snails.

A valuable source of plant health management strategies for all major vegetable crops, from asparagus to zucchini; also including herbs and spices, mushrooms, vegetable sprouts, and such native crops as ginseng. Jerusalem artichoke and fiddlehead.

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