

15 Pea and bean

15A Pea

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Pea weevil

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BACTERIAL DISEASES

► 15A.1 Bacterial blight *Figs. 15A.1a,b*

Pseudomonas syringae pv. *psii* (Sackett) Young, Dye & Wilkie (syn. *Pseudomonas psii* 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.

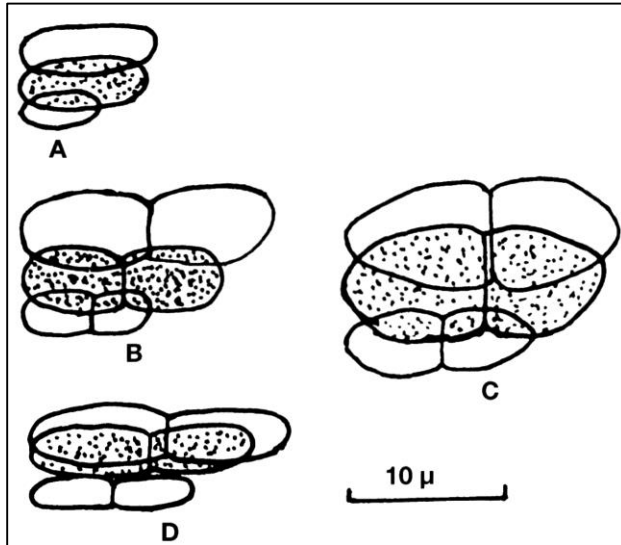
Foot rot

Phoma medicaginis var. *pinodella* (L.K. Jones) Boerema (syn. *Ascochyta pinodella* L.K. Jones)

Mycosphaerella blight

Mycosphaerella pinodes (Berk. & Bloxam) Vesterg. (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*, *Lathyrus* 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 cylindrical-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. *pisii* (van Hall) W.C. Snyder & H.N. Hans.
Fusarium solani f. sp. *pisii* (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. *pisii* and *F. solani* f. sp. *pisii* 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 macroconidia, 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 web 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.

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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.

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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 sclerotia, 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.

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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 internodes, 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 (K₂O) 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*

Acyrtosiphon 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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► **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 reseedling 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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15B Bean

Figures 15B.1 to 15B.19; 15B.7T1; 15B.9T1

Bacterial diseases

- 15B.1 Bacterial blights
 - Bacterial brown spot
 - Common blight and fuscous blight
 - Halo blight

Fungal diseases

- 15B.2 Anthracnose
- 15B.3 Gray mold
- 15B.4 Root rots, damping-off, seed decay
- 15B.4 Black root rot
- 15B.5 Fusarium root rot
- 15B.6 Pythium diseases
- 15B.7 Rhizoctonia root rot
- 15B.8 Rust
- 15B.9 White mold

Viral diseases

- 15B.10 Bean common mosaic
- 15B.11 Bean yellow mosaic

Non-infectious diseases

- 15B.12 Nutritional disorders
 - Aluminum toxicity
 - Boron toxicity
 - Iron deficiency
 - Manganese deficiency
 - Nitrogen deficiency
 - Phosphorus deficiency
 - Zinc deficiency
- 15B.13 Other disorders
 - Baldhead
 - Herbicide injury
 - Ozone injury
 - Sunscald
 - Wind injury

Nematode pests

- 15B.14 Northern root-knot nematode
- 15B.15 Root-lesion nematode
- 15B.16 Stubby-root nematodes

Insect pests

- 15B.17 European corn borer
- 15B.18 Seedcorn maggot
- 15B.19 Other insect pests
 - Cutworms
 - European earwig
 - Mexican bean beetle

Additional references

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 than 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 corn.

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 chlamydospores, 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 endodermis 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-susceptible 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)

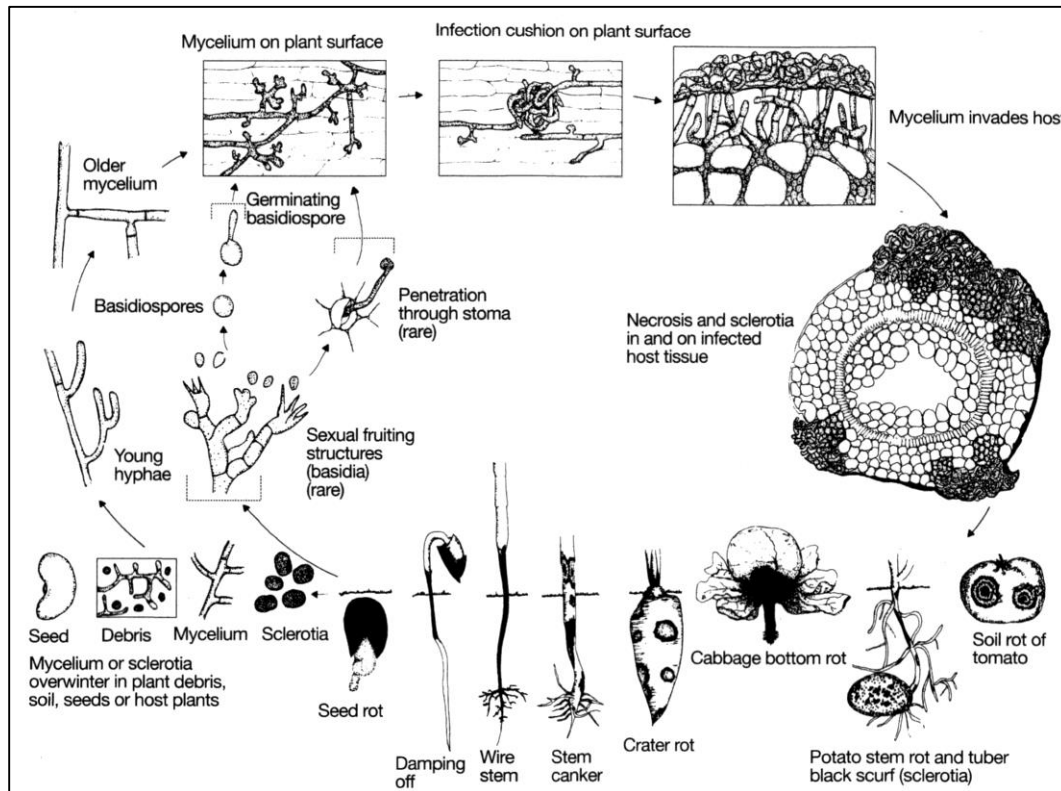
This disease can reduce stands and delay maturity. Yield losses in green bean pods of 13 to 54% have been associated with stand reductions of 24 to 50%. The pathogen, *R. solani*, has anastomosis groups, of which AG-4 and AG-2 type 2 are pathogenic to bean. AG-4 causes disease in many crops and AG-2 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 µm 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.7TI *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 spermatogonia on the upper surface of the leaf. The spermatogonia appear as chlorotic flecks, 2 mm in diameter, and produce a white nectar containing spermatia. After movement of spermatia to a spermatogonium 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 spermatogonia 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 spermatogonia containing spermatia, and aecia containing aeciospores; infection by aeciospores leads to the production of urediniospores in the rust pustules. Spermatogonia 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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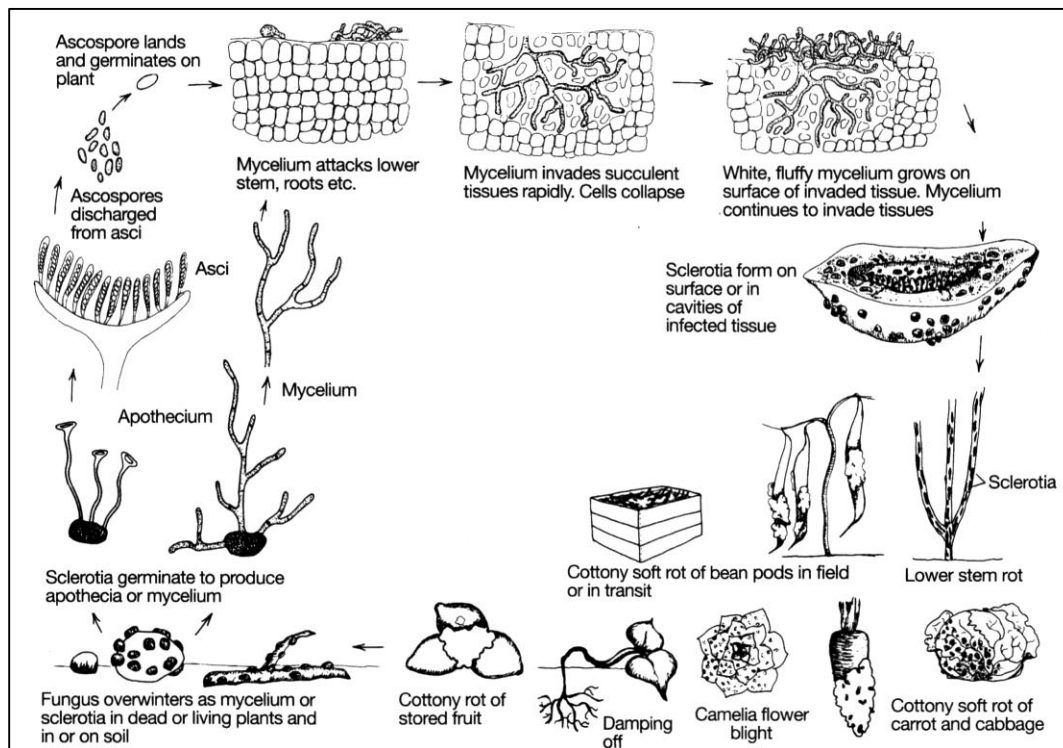
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 (Original by R. Hall)

► **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.9TI*). 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 sclerotia, 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.11*

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 internodes. 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
- Sunscauld
- 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 (O₃) 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 sunscauld. 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.

Sunscauld

Sunscauld 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 sunscauld 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, torn 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 reseeded. 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 reseeded 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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