25 Greenhouse tomato

BACTERIAL DISEASES

▶ 25.1 Bacterial canker *Figs. 25.1; 18.1a-c*

Clavibacter michiganensis subsp. *michiganensis* (E.F. Smith) Davis *et al.*
(syn. *Corynebacterium michiganense* (E.F. Smith) Jensen)

Bacterial canker is a very contagious and destructive disease of greenhouse tomato. It also can affect field tomato but symptoms differ markedly (see Tomato, bacterial canker, 18.1). The disease is equally prevalent in soil- grown and hydroponic crops. Other hosts of the pathogen include pepper and black nightshade *(Solanum nigrum* L.).

Symptoms Wilting of lower leaflets is normally the first observable symptom. Older leaflets curl upward, progressively die from the margin inward and turn brown *(18.1a).* Leaflets also may have small, cream to gray-white blisters. Frequently, only leaflets on one side of the leaf are affected. Petioles may turn downward but they do not wilt. If leaflet growth is succulent, pale green spots of collapsed tissue may develop between the veins. Younger parts of infected stems and petioles may appear water soaked. Affected plants may wilt and die early *(25.1).* If growth is vigorous, plants may survive in an unthrifty wilted condition and bear some fruit. Severely affected plants display a wilt accompanied by light- colored, longitudinal streaks on the stem and petioles. These streaks may break open to form a canker *(18.1b),* thus giving the disease its name. Infected stems are slightly spongy when squeezed at the nodes. As decay progresses, the pith becomes mealy and cavities form in the soft tissues. Affected plants generally do not display extensive root discoloration.

Fruit infection is common. In young tomato plants, infected fruit is stunted, malformed and occasionally ridged. Fruit infected at later stages may not show symptoms or may have a marbled or mottled surface. The calyx scar tissue may be discolored and the calyx attachment weakened. The vascular tissue of infected fruit is yellowish from the stem scar to well within the pulp. Severely infected fruit have extensive internal breakdown with yellow to brown cavities, especially near the stem. Fruit may also develop small "bird's-eye" cankers *(18.1c)* if overhead irrigation is used. Initially, these appear as snow- white spots that scarcely extend beyond the skin. The margins of the spots are white and flat. The centers of the spots are slightly raised, tan-colored and eventually crack open. These spots do not exceed 3 mm in diameter. Seeds of early infected fruit may be spotted or entirely dark and do not mature.

Causal agent *Clavibacter michiganensis* subsp. *michiganensis* is a motile, rod-shaped, Gram-positive bacterium, measuring 1 by 0.5 µm. It is non-acid fast, non-spore forming and non-lipolytic. It liquifies gelatin slowly and can oxidize carbohydrates. It hydrolyses starch weakly or not at all and requires biotin, nicotinic acid and thiamine for growth. Yellow, white and pink forms occur but on nutrient agar the colonies are characteristically yellow. The yellow and white forms are the most virulent.

Disease cycle The bacteria are carried on the surface and within the coat of seed produced by infected plants. Germinating seedlings are infected through the cotyledons. The pathogen is able to enter the host through wounds, such as broken trichomes, or directly through the stomata. It moves systemically through the xylem and invades the phloem, pith and cortex. Spread to neighboring plants occurs by splashing or running water, insects, implements and workers tending the crop. Infection is favored by high temperatures (24 to 32°C), wet conditions, low light intensity and nutrient imbalances. The spashing caused by forceful spraying of pesticides may also help to spread the disease. The bacteria can survive on or in seed for five years and in soil for shorter periods. They also can persist from season to season on infested plant residues, wooden stakes and perennial hosts, any of which can act as an initial disease focus within a crop.

Management

Cultural practices — For effective prevention, growers should use disease-free seed. If such seed cannot be obtained, seed treatment should be considered. Seed from infected plants can be extracted and substantially freed from bacterial infection by fermenting the undiluted, crushed pulp at room temperature for 96 to 120 hours. Seed can also be soaked in acetic acid (0.6 to 0.8% solution) for 24 hours at 21°C. Other effective seed treatments include a 30-min soak in water at 56°C, a 20- to 40-min soak in 1% sodium hypochlorite, or soaking for 5 to 10 hours in 5% hydrochloric acid. Seed treatments help to reduce inoculum of the pathogen but they are not completely effective. Seed should always be sown in pasteurized growing media, using new or sterile flats, pots or other containers.

Diseased and adjacent plants should be removed as soon as they are noticed by placing them in plastic bags and carrying them out of the greenhouse. Any remaining crop residues should be gathered up or, in the case of soil, buried by rototilling. Spread can be limited by washing hands thoroughly between greenhouse visits and changing clothing when moving from an infected to a healthy crop.

Pruning and pollinating tools should be disinfested at regular intervals, plants should not be handled unnecessarily, and diseased plants should be tended after healthy ones.

Chemical control — Although chemical sprays are sometimes recommended in extension publications, most serve only to spread the disease and have little or no effect on the pathogen.

Selected references

Berry, S.Z., G.C. Madumadu and M. Rafique Uddin. 1988. Effect of calcium and nitrogen nutrition on bacterial canker disease of tomato. *Plant Soil* 112:113-120.

Dhanvantari, B.N. 1989. Effect of seed extraction methods and seed treatments in control of tomato bacterial canker. *Can. J. Plant Pathol.* 11:400-408.

Hayward, A.C., and J.M. Waterston. 1964. *Corynebacterium michiga- nense.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 19. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

McKeen, C.D. 1973. Occurrence, epidemiology, and control of bacterial canker in southwest Ontario.Can. *Plant Dis. Surv.* 53:127-130.

Strider, D.L. 1969. Bacterial canker of tomato caused by *Corynebacterium michiganense. North Carolina Agric. Exp. Stn. Tech. Bull.* 193.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.2 Bacterial speck *Figs. 18.3a,b*

Pseudomonas syringae pv. *tomato* (Okabe) Young *et al.*

This disease is of minor importance in commercial tomato greenhouses but can be a serious problem on field tomato.

It sometimes occurs in greenhouses in which transplants are being grown for the field. (For more information, see Tomato, bacterial speck, 18.3.)

(Original by R.J. Howard)

▶ 25.3 Bacterial stem rot *Fig. 25.3*

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

An unusually high incidence of stem rot, wilt and death of tomato plants occurred in the greenhouse tomato production area of Essex County, Ontario, during the harvest season of 1983. This disease had occurred only sporadically before that time. *Erwinia carotovora* subsp. *carotovora* has a wide host range that includes many vegetables (see Potato, bacterial soft rot, 16.2).

Symptoms Symptoms first appear about the time of first or second harvest *(25.3).* Basal leaf scars may have dark brown lesions, while stem bases become hollow and appear water-soaked. Bark readily sloughs off and stem pith turns brown and disintegrates. At an advanced stage of the disease, plants wilt and die. Bacterial stem rot is favored by high humidity and can be spread by splashing water, workers' hands and tools. See also Tomato, bacterial soft rot, 18.2.

Causal agent (see Potato, 16.2) Several other *Erwinia* and *Pseudomonas* species have been shown to cause bacterial soft rot of greenhouse tomato in Europe.

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

Management

Cultural practices — The sanitation practices outlined for bacterial canker are relevant here. Growers should avoid working with plants when the foliage is wet and direct water dripping from gutters away from tomato plants.

Selected references

Bradbury, J.F. 1977. *Erwinia carotovora* var. *carotovora.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 552. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Dhanvantari, B.N., and V.A. Dirks. 1987. Bacterial stem rot of greenhouse tomato: etiology, spatial distribution, and the effect of high humidity. *Phytopathology* 77:1457-1463.

Malathrakis, N.E., and D.E. Goumas. 1987. Bacterial soft rot of tomato in plastic greenhouses in Crete. *Ann. Appl. Biol.* 111:115-123.

(Original by R.J. Howard)

▶ 25.4 Pith necrosis *Figs. 25.4a-c*

Pseudomonas corrugata Roberts & Scarlett

This disease affects greenhouse tomato, particularly those plants with luxuriant growth. Its occurrence is sporadic and usually limited to a few plants. Tomato is the only crop in which this disease is an economic problem. *Pseudomonas corrugata* has been isolated from symptomless alfalfa roots, and in laboratory tests, some strains can rot carrot tissue. Otherwise, no other hosts are known.

Symptoms Plants affected by pith necrosis are usually vigorous, with thick fleshy stems and a large canopy. Symptoms normally appear just before the first fruit is picked. Initially, affected plants display chlorosis of the upper leaves, sometimes with wilting *(25.4a).* They may be stunted and have elongate, dark brown to black lesions on the stem *(25.4b).* The stem may collapse at lesion sites. The pith of the main stem blackens and there may be large spaces with ladder-like cross strands in the blackened pith in older parts of the stem. Light brown discoloration occurs in younger areas of stems that lack a pith cavity *(25.4c).* In general, pith discoloration extends to the soil level but it does not enter the roots. The peduncle also may have discolored pith but not the fruit. Occasionally, leaf scars will have a creamy-white bacterial ooze. Older plants frequently exhibit prolific development of adventitious roots on the stem, usually coinciding with the infected area. Diseased plants may continue to produce fruit.

Causal agent *Pseudomonas corrugata* is a non-fluorescent rod with a flagellar tuft at the pole and is characterized as aerobic, oxidase positive, gelatin and egg-yolk positive, starch hydrolysis and levan test negative, and HR positive on tobacco. It accumulates poly-ß-hydroxybutyrate (PHB). Erythritol and rhamnose are not utilized. Growth occurs at 37°C but not at 41°C. Colonies on nutrient agar are round, achieving a diameter of 1 mm in two days and 1 to 3 mm in a week. They are raised, cream to buff, later turning beige-yellow. On nutrient agar with 5% glucose, colonies are yellowish, often with green centers at two days and usually with a yellow-green diffusible non-fluorescent pigment, which also develops on King's B medium. The bacterium is readily isolated on King's B medium by streaking exudates from affected tissues. A semi-selective isolation medium (TNR) is available (see Selected references, Scortichini 1989).

Disease cycle The biology of this bacterium is not well understood but it is considered to be soil- and water-borne. The pathogen can infect seedling tomato through the roots. There is no evidence of workers spreading the disease on hands, clothing or tools, and it does not spread easily within the crop. The disease is favored by high humidity and high nitrogen status. Adventitious root formation in affected plants is caused by the accumulation of auxins.

Management

Cultural practices — Growers should try to avoid conditions that lead to wet plants and luxuriant growth. Excessive foliage can be controlled by raising the level of potash in the fertilizer solution to obtain a corresponding decrease in the nitrogen: potassium ratio. Diseased plants should be removed immediately, using the procedures outlined for bacterial canker.

Selected references

Clark, R.G., and D.R.W. Watson. 1986. New plant disease record in New Zealand: tomato pith necrosis caused by *Pseudomonas corrugata. N.Z. J. Agric. Res.* 29:105-109.

Lai, M., D.C. Opgenorth and J.B. White. 1983. Occurrence of *Pseudomonas corrugata* on tomato in California. *Plant Dis.* 67:110- 112.

Scarlett, C.M., J.T. Fletcher, P. Roberts and R.A. Lelliott. 1978. Tomato pith necrosis caused by *Pseudomonas corrugata* n.sp. *Ann. Appl. Biol.* 88:105-114.

Scortichini, M. 1989. Occurrence in soil and primary infections of *Pseudomonas corrugata* Roberts and Scarlett. *J. Phytopathology* 125:33-40.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.5 Stem necrosis *Fig. 25.5*

Pseudomonas sp.

This disease has been reported only from Ontario, where it has caused concern among growers for several years. It usually appears in the spring crop. The pathogen is opportunistic, infecting plants that have been stressed by unbalanced nutrition, excessive humidity or the onset of fruiting. It has been seen in both soil- and rockwool-grown crops.

Symptoms Characteristic symptoms of tomato stem necrosis include dark brown discoloration of leaf bases at the nodes, adjacent leaf rachises and internodes, followed by cortical and pith necrosis and breakdown (25.5). Except for a general pith necrosis, these symptoms differ from those reported for most of the other stem rot diseases of greenhouse tomato. Vascular discoloration is sometimes observed but plants generally do not wilt or collapse. Fruits are free of symptoms.

Causal agent The taxonomy of this *Pseudomonas* sp. is uncertain. It resembles *P. cichorii* except that the stem necrosis pathogen is not pathogenic to chrysanthemum or lettuce, and it has been provisionally assigned to the group of oxidase-positive, arginine dihydrolase-negative, phytopathogenic fluorescent pseudomonads now solely represented by *P. cichorii* (see Lettuce, pseudomonas diseases, 11.3).

Disease cycle The disease resembles the other bacterial diseases in that it is spread by irrigation water and principally by workers carrying out routine operations in the crop.

Management

Cultural practices — Growers should follow the sanitation practices outlined for bacterial canker (see 25.1).

Selected references

Dhanvantari, B.N. 1990. Stem necrosis of greenhouse tomato caused by a novel *Pseudomonas* sp. *Plant Dis.* 74:124-127.

(Original by R.J. Howard)

FUNGAL DISEASES

▶ 25.6 Corky root (brown root rot) *Figs. 25.6a,b*

Pyrenochaeta lycopersici R. Schneider & Gerlach

In greenhouses, corky root is fairly common and serious in early spring crops grown in media that are too cold. While it is most common in soil-grown crops, significant numbers of crops grown in rockwool are also affected. It is unknown how the fungus infests artificial substrates; it has even been reported in the nutrient film (NFT) system. Corky root only rarely affects field tomato crops. The pathogen survives on the surface of roots of lettuce and some weed species.

Symptoms The first symptoms appear as light brown lesions about 5 mm long on the surface of fine roots. This stage is often referred to as brown root rot *(25.6a).* Early symptoms on the upper portions of plants include lack of vigor, foliar chlorosis and stunting. Larger roots develop dry, brown, swollen, corky lesions with splits in the outer sheath *(25.6b).* The cortex of the root can easily be pulled off the central stele at lesion sites. Dark-brown cortical lesions are often present at the base of the stem on severely diseased plants. Massive root failure is the cause of wilt in hot, sunny weather, and is followed eventually by death of the plant. Yields may be reduced.

Causal agent *Pyrenochaeta lycopersici* was long known as a gray sterile fungus, because of the difficulty in obtaining sporulation in culture, although some strains sporulate on roots. In culture, globular to sub-globular, brown to black pycnidia, 150 to 300 pm in diameter, are produced. Pycnidia have 3 to 12 light brown, septate setae, measuring 7 by 120 µm. Conidiophores within the pycnidia are septate and simple. Unicellular, hyaline conidia are produced from the apex and short lateral branches immediately below the septa of the conidiophores within each pycnidium. Conidia are cylindrical to allantoid and measure 4.5 to 8 by 1.5 to 2 pm. Microsclerotia are unspecialized with cell walls of uniform thickness.

Dark brown lesions on roots become swollen and corky in texture. The cortex pulls off the stele readily to leave characteristic "rat-tails." In contrast with black-dot root rot, in which the pathogen produces fruiting bodies late in the season, fruiting bodies on corky root lesions are rare. Microscopic examination of the lesions will reveal mycelia packing diseased host cells to form characteristic rectangular microsclerotia.

Disease cycle The pathogen is soil-borne and can survive as sclerotia for at least two years in soil. It grows at 8 to 32°C and develops very slowly in the soil. Thus, the disease increases slowly over time. The fungus colonizes disinfested soil slowly when introduced into a greenhouse. Infection occurs when host roots make contact with the fungus mycelium, often in soil that is below the level of effective steam pasteurization.

Management

Cultural practices — Crops grown in peat modules or in soilless, NFT or rockwool culture systems are generally not affected. Because the pathogen grows slowly, some recovery may be achieved by mounding soil or soilless media, such as sawdust or peat, around the base of the stem to encourage the growth of adventitious roots. Good ventilation is essential, especially around the stem bases, and splashing should be minimized during watering. Transplanting should be done into warm soil (over 15°C), because corky root is essentially a disease of cool soils (10 to 15°C). For this reason, mulches such as straw, which insulate the soil, should not be put down until the soil is warm.

Resistant cultivars — Resistant rootstocks, such as KNV and KNVF types, are available, so grafting of commercially acceptable scions onto resistant rootstocks is possible.

Chemical control — The pathogen can be eradicated from soil by steam or fumigation, but not at depths below the level reached by these disinfestants. Growers should remove and destroy old roots before disinfesting growing media.

Selected references

Grove, G.G., and R.N. Campbell. 1987. Host range and survival in soil of *Pyrenochaeta lycopersici. Plant Dis.* 71:806-809.
Jarvis, W.R. 1984. A recurrence of tomato corky root in Ontario. *Can. Plant Dis. Surv.* 63:65.
McGrath, D.M., and R.N. Campbell. 1983. Improved methods for inducing sporulation of *Pyrenochaeta lycopersici. Plant Dis.* 67:1245-1248.
Punithalingam, E., and P. Holliday. 1973. *Pyrenochaeta lycopersici.* CMI descriptions of pathogenic fungi and bacteria, No. 398. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.
Richardson, J.K., and G.H. Berkeley. 1944. Basal rot of tomato. Phytopathology 34:615-621.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.7 Damping-off *Fig. 25.7*

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

Damping-off can be severe in newly transplanted crops, especially those grown in soil. The reduction of seedling emergence and the falling over of young seedlings are characteristic symptoms. In greenhouses, the most important causal agents are *Pythium* spp., but other fungi also may be involved, for example, *Phytophthora* spp. and *Rhizoctonia solani.* These fungi are soil-borne. *Pythium* and *Phytophthora* spp. may also contaminate irrigation water. Damping-off occurs frequently in seedlings and transplants raised in rockwool blocks and other soilless substrates. This disease can affect a variety of vegetable crops.

Symptoms Symptoms vary with age and stage of development of the host. If seeds are infected before germination, they fail to germinate, become soft and mushy, turn brown, shrink and finally decompose. Infection of young seedlings results in slightly darkened, water-soaked lesions that expand until the invaded tissues collapse. Infection of seeds or young seedlings is usually apparent by a reduction in seedling emergence.

After emergence, seedling roots can become infected at or below soil level. Lesions are usually pale brown and water-soaked *(25.7).* The basal part of infected seedlings is normally much thinner and softer than the upper part. The invaded stem cannot support the seedling, which falls over, withers and dies.

Causal agents (see Bean, 15B.4; Beet, pythium and rhizoctonia root rots, 5.7, 5.8; and Carrot, cavity spot, 6.8, and pythium root dieback, 6.13)

Disease cycle Damping-off pathogens spread quickly in cool, wet soil. Temperatures of 10 to 15°C are the most conducive to early damping-off by *Pythium* and *Phytophthora* spp. Infection is aided by excess nitrogen and crowding of the plants.

Rhizoctonia solani (see Bean, rhizoctonia root rot, 15B.7) tends to attack more mature transplants, leading to late damping-off. Under drier conditions, *R. solani* is frequently the most troublesome damping-off pathogen.

Management

Cultural practices — To retard spread of these pathogens, greenhouse seedlings should not be crowded or overwatered. Watering should be done only when the soil is dry and preferably in the morning so the soil will be dry by late afternoon. Adequate greenhouse ventilation helps to keep the soil dry. Seed flats should be raised and placed out of the range of splashing water. Bottom heat should be applied to raise the soil temperature above 15°C. Rockwool blocks and other soilless media should never be allowed to come into contact with soil or water splashing from soil and dirty benches or floors.

Chemical control — Seed treatment with hot water, followed by application of a fungicidal seed protectant, and sowing into pasteurized soil, is helpful. Seedling trays can be drenched with fungicide solutions to provide additional protection if conditions warrant.

Selected references

Leach, L.D. 1947. Growth rates of host and pathogen as factors determining the severity of damping-off. *J. Agric. Res.* 75:161-179.

Mordue, J.E.M. 1974. *Thanatephorus cucumeris.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 406. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Sonoda, R.M. 1976. Incorporating fungicides in planting mix to control seedling diseases of plug-mix seeded tomatoes. *Plant Dis. Rep.* 60:27-30.

Van der Plaats-Niterink, A.J. 1981. Monograph of the Genus *Pythium. Stud. Mycol.* 21. Centraalbureau v. Schimmelcultures, Baarn, The Netherlands. 242 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.8 Didymella stem canker *Fig. 25.8*

Didymella lycopersici Kleb.
(anamorph *Diplodina lycopersici* Hollos)

This is an important fungal disease of tomato in Europe, but it is rare in North America. It has been seen occasionally in British Columbia and Nova Scotia. The disease occurs in soil and soilless culture and may spread explosively. Fruit infection occurs in the field in the United States, but only rarely in the greenhouse. Alternative hosts include black nightshade *(Solanum nigrum* L.), eggplant, pepper and potato.

Symptoms Dark brown, sunken lesions appear on the stem at or near soil level *(25.8).* The epidermis and cortex decay and the xylem becomes brown some distance up the stem. The stem above the lesions remains green. Severely affected plants with one or more stem lesions frequently wilt and the lower leaves show varying degrees of chlorosis and necrosis. Under damp conditions, small brown lesions with concentric rings develop on the leaves. The centers eventually become pale brown or tan with a few pycnidia. The lesions may fall out, leaving shot-holes, or they may grow together and kill the whole leaf. The pathogen can attack any part of the fruit; however, it usually infects the calyx end, causing an extensive black rot. Pycnidia form on the infected part and the fruit may drop off the plant. If fruit remain attached, the fungus may grow into the pedicel and ultimately reach the stem. Seed may also become infected.

Causal agent *Didymella lycopersici* is most commonly observed in its anamorphic state. Pycnidia are sub-epidermal, ostiolate, 100 to 270 pm in diameter and scattered or aggregated on raised spots. Conidia are one- to two-celled, sub-cylindrical and measure 4.5 to 17 by 2.5 to 5 µm. Pseudothecia are sub-globose and dark-brown with cylindrical asci 70 to 95 by 9 to 10 µm. Each ascus has eight, spindle-shaped, hyaline, unisep- tate ascospores, each measuring 16 to 18 by 5.5 to 6.5 µm.

Careful examination of stems will reveal the small round pycnidia, which can be confused with the dark brown glandular trichomes present on tomato stems. Accurate diagnosis is difficult without a hand lens because the lesions resemble those of gray mold, which are normally lighter brown, develop aerial conidiophores and cause the stem area above the lesion to turn yellow. Both fungi can occur in the same lesion. On leaves, the small brown lesions with concentric rings resemble those of early blight.

Disease cycle The pathogen survives from season to season in the soil or on alternative hosts. It may survive on infected seed or as spores on contaminated seed boxes, stakes or greenhouse structures. The main mode of introduction to a new crop is by water-splashed conidia produced in pycnidia on plant residue or alternative hosts. The fungus infects host tissues at 11 to 30°C (optimum 20°C), spreading rapidly once established. Numerous, small, black pycnidia appear on the rotting lesions. Rarely, pseudothecia are intermixed with the pycnidia. Later in the season, lesions form higher in the plant canopy and spread to all above-ground parts of the plant. At high humidity, cirrhi of gray to pink conidia extrude in a gelatinous matrix. These spores are spread by splashing water, trimming knives or workers' hands, and occasionally they are air-borne. The conidia are tolerant of desiccation and low temperature, can be transported long distances in the air, and can survive extended periods of unfavorable environmental conditions.

Management

Cultural practices — Resistance to infection increases with plant age and an adequate supply of nitrogen and phosphorus. Strict crop hygiene and disposal of diseased residues, especially at the end of the season, are very important. Stem lesions should not be removed by knife because the blade will become contaminated with spores and infect subsequent plants being trimmed. Fruit should be picked without bruising, allowed to dry and layered singly with the stem-end up to avoid the fruit rot phase of this disease. Disposal of infected plants should be in plastic bags without handling the lesions and all plant residue should be buried or composted as far from the greenhouse as possible.

Chemical control — Soil should be disinfested between crops and the greenhouse should be washed with a disinfestant or fumigated before a new crop is planted.

Selected references

Fagg, J., and J.T. Fletcher. 1987. Studies of the epidemiology and control of tomato stem rot caused by *Didymella lycopersici*. *Plant Pathol.* 36:361-367.

Holliday, P., and E. Punithalingam. 1970. *Didymella lycopersici*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 272. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Knight, D.E. 1960. Studies on *Didymella lycopersici* Kleb., the causal fungus of stem rot disease of tomatoes. *Trans. Br. Mycol. Soc.* 43:519-522.

(Original by J.G. Menzies and W.R. Jarvis)

25.9 Early blight (target spot), alternaria fruit rot *Figs. 25.9a,b; 18.8b,c,f*

Alternaria solani Sorauer
Alternaria alternata (Fr.:Fr.) Keissl.

Early blight is a very common disease of field tomato and occasionally greenhouse tomato. In greenhouses it can occur in crops grown in soil and soilless media. *Alternaria solani* also infects potato (see Potato, early blight, 16.8), eggplant and solanaceous weeds. *Alternaria alternata* occurs widely in nature on organic matter and is generally considered to be a weak, opportunistic plant parasite (see Cucurbits, alternaria leaf blight, 9.8; and Tomato, early blight, 18.8).

Symptoms This disease is most common on older foliage, but it also occurs on the stems and ripening fruits of greenhouse tomato. Leaf spots are circular, about 1 cm in diameter, dark brown to black, and readily recognized by concentric rings or zonations ("target spots") *(25.9a)*. The concentric aspect of the rings may be lost on lesions near the edge of the leaf. Lesions on stalks, branches and pedicels appear black, subsequently enlarging, elongating and sometimes girdling them. Fruit lesions generally start around a pedicel, wound or crack, rapidly enlarging into black, leathery sunken areas *(18.8c)*. If defoliation is severe, unprotected fruit may be damaged by sunscald.

This disease may be confused with septoria leaf spot *(25.9b):* however, septoria leaf spot has pycnidia in relatively small spots, whereas early blight has concentric dark rings in larger spots *(18.8b)*. *Alternaria alternata*, the less pathogenic species, is frequently associated with *A. solani* on lesions *(18.8f)*. Leaf lesions with concentric rings are also associated with didymella stem canker (see didymella stem canker, 25.8).

Causal agent (see Potato, early blight, 16.8) The tapered, muriform conidia of *A. solani* are 150 to 300 pm long and are characterized by a very long beak, about the same length as the body; conidia of *A. alternata* have a very short beak, and are 20 to 63 pm (mean 37 pm) long. Conidia should be mounted in water for spore identification.

Disease cycle (see Potato, early blight, 16.8) The pathogen can survive for long periods in soil and diseased plant residue. It is also seed-borne. Infection of the crop is initiated by conidia produced on diseased hosts or host residues. Disease development is favored by alternating conditions of high humidity at night and dry days. Infection can occur between 10 and 25°C.

Management

Cultural practices — Diseased leaves and stems should be removed and destroyed if practical. Soilbeds should be steamed or fumigated between crops.

Chemical control — Registered fungicides are available.

Selected references

Agrios, G.N. 1988. *Plant Pathology*. 3rd ed. Academic Press, New York. 803 pp.

Ellis, M.B. 1971. *Dematiaceous Hyphomycetes*. Commonw. Mycol. Inst., Kew, Surrey, England. 608 pp.

Ellis, M.B., and I.A.S. Gibson. 1975. *Alternaria solani*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 475. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Pound, G.S. 1951. Effect of air temperature on incidence and development of the early blight disease of tomato. *Phytopathology* 41:127-135.

(Original by J.G. Menzies and W.R. Jarvis)

25.10 Fusarium crown and root rot *Figs. 25.10a-d*

Fusarium oxysporum f. sp. *radicis-lycopersici* W.R. Jarvis & Shoemaker

Fusarium crown and root rot is an important disease of greenhouse tomato in the United States and Canada, and it also has been reported on field tomato. It occurs with equal severity in commercial crops in soil, rockwool, sawdust and the nutrient film techniques (NFT). For practical purposes, the disease is limited to tomato, but many plants have been shown to be susceptible when artificially inoculated with the pathogen.

Symptoms Young tomato seedlings can become severely affected and die, but this disease mainly affects bearing plants. Symptoms usually appear in greenhouse tomato crops just before the first pick. Infected plants can often be distinguished by a marked thinness at the top of the stem. These plants wilt, beginning with the upper leaves, and there may be a chocolate-brown cortical rot at soil level *(25.10a),* with a red-brown vascular discoloration extending upwards in the stem for 5 to 25 cm *(25.10b).* Subsequently, the lower leaves may turn golden-yellow from the tip and eventually die. Wilt symptoms abate on cooler and overcast days and after picking and watering. Adventitious roots may form above stem lesions. Roots of infected plants have dark red-brown lesions, often confluent with hypocotyl lesions. Small, gray- brown lesions form where secondary roots emerge from the main roots. Fruits from affected plants are flaccid and lack the normal bright color *(25.10c,d).* Dead or near-dead plants may have conspicuous external masses of pink-white or salmon-colored mycelium.

Causal agent *Fusarium oxysporum* f. sp. *radicis-lycopersici* is indistinguishable from other forms of *F. oxysporum* in morphology and in characteristics in pure culture. Microconidia are oval-ellipsoid, cylindrical, straight to curved, and measure 5 to 12 by 2.2 to 3.5 pm. They are produced on simple phialides arising laterally on hyphae or from short, sparsely branched conidiophores. Macroconidia are thin-walled, generally three- to five-septate, fusoid-subulate, pointed at both ends with a hooked apex and a pedicellate base, and measure 27 to 66 by 3 to 5 pm. Chlamydospores are generally abundant, solitary and both terminal and intercalary.

Other tomato wilt diseases have similar symptoms, so diagnosis requires the isolation and identification of the pathogen. Special culture media are available for the selective isolation of *F. oxysporum.* A petri- dish test has been developed to distinguish between the fusarium wilt and fusarium crown and root rot pathogens. In this test, tomato seeds are germinated directly on water agar seeded with the suspected pathogen. Fusarium crown and root rot causes soft, chocolate-brown lesions on the hypocotyl, whereas fusarium wilt causes no symptoms or only a faint brown discoloration.

Disease cycle The manner in which the pathogen is introduced into previously unaffected areas is not known. It can survive as chlamydospores in soil below the level of effective sterilization by steam or fumigation, as well as in thick roots and lumps of clay that are difficult to sterilize. In heavily infested seedling and transplant trays, damping-off sometimes occurs; symptoms resemble those of pythium damping-off. There may be a fast wilt of mature infected plants, resulting in early death, or a slow wilt with progressive, acropetal leaf death. Plants suffering from slow wilt may survive until the end of the season and produce a flush of new growth after most of the fruit has been picked.

Field crops do not seem to play a significant role in epidemiology in the greenhouse. Seed transmission or movement of chlamydospores on clothing, shoes, machinery, packing crates and in soil or compost are all possible pathways for spread. Infection of young seedlings occurs in infested soil or from air-borne microconidia from residue piles of tomato vines, soil and straw mulch. The fungal population increases rapidly after introduction into fumigated or steam-sterilized soils, but less so in pasteurized soils. It enters root and hypocotyl cortical tissues through wounds caused by emerging secondary roots, as well as by directly penetrating the epidermis. Microconidia are probably spread by water from mobile irrigation systems or by wind currents. It is also suggested that fungus gnats may spread the pathogen from diseased to healthy plants while feeding. Fungus gnats may further aid the pathogen by creating wounds through which it can invade the roots. The optimum temperature for disease expression is 15 to 18°C.

The significance of other crops in fostering the survival and spread of the pathogen has not been assessed.

Management (For fungus gnats and their control, see Greenhouse cucumber, 22.31.)

Cultural practices — The incorporation of lettuce or dandelion residues into the soil before planting tomato reduces disease severity, as does companion planting with lettuce or dandelion. Soil in beds should be 20°C or warmer at the time of transplanting. Straw mulch should not be laid in spring until soil temperatures reach this level. Late spring plantings are less affected than winter and early spring plantings. In greenhouse crops, removal of the first fruit on heavily infected plants may allow the plant to recover with relatively little loss. Mounding soil or a soil- peat mixture around the base of the stem of infected plants to a height of 10 to 20 cm stimulates the formation of adventitious roots, which generally remain disease-free, allowing the plant to recover.

Resistant cultivars — Resistant cultivars include CR-6, 83W186 and B8-864 (pink-fruited); and Larma, Vicores, Furon, Trend, Farao, XPH2419/88, Cobra and W1601 (red- fruited). Grafting scions of susceptible tomato cultivars with good agronomic characteristics onto resistant tomato rootstocks, such as KNVF-Tm or KVF, has been effective.

Biological control — Cross protection of susceptible cultivars by root inoculation with avirulent *Fusarium oxysporum* f. sp. *radicis-lycopersici* shows promise for control.

Chemical control — Steam sterilization or fumigation of soil beds does not control the disease and often worsens the problem because of the rapid re-entry of the pathogen into the soil. Pasteurization of soil by steam-air mixtures is much more effective, because it preserves a number of competitive and antagonistic microorganisms that can substantially reduce the infective population of *Fusarium oxysporum* f. sp. *radicis-lycopersici* in the soil.

Selected references

Hartman, J.R., and J.T. Fletcher. 1991. Fusarium crown and root rot of tomatoes in the UK. *Plant Pathol.* 40:85-92.

Jarvis, W.R. 1988. Fusarium crown and root rot of tomatoes. *Phytoprotection* 69:49-64.

Jarvis, W.R., and H.J. Thorpe. 1981. Control of fusarium foot and root rot of tomato by soil amendment with lettuce residues. *Can. J. Plant Pathol.* 3:159-162.

Marois, J.J., and D.J. Mitchell. 1981. Effects of fumigation and fungal antagonists on the relationships of inoculum density to infection incidence and disease severity of fusarium crown rot of tomato. *Phytopathology* 71:167-170.

Menzies, J.G., C. Koch and F. Seywerd. 1990. Additions to the host range of *Fusarium oxysporum* f. sp. *radicis-lycopersici. Plant Dis.* 74:569-572.

Sanchez, L.E., R.M. Endo and J.V. Leary. 1975. A rapid technique for identifying the clones of *Fusarium oxysporum* f. sp. *lycopersici* causing crown and root-rot of tomato. *Phytopathology* 65:726-727.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.11 Fusarium wilt *Figs. 25.11a-c*

Fusarium oxysporum f. sp. *lycopersici* (Sacc.) W.C. Snyder & H.N. Hans.

Fusarium wilt of tomato is a common disease and is most destructive in warm greenhouses. The pathogen can survive on other *Lycopersicon* species, on species of *Amaranthus, Digitaria* and *Malva*, and as a saprophyte in association with fibrous roots of other plants.

Symptoms The first symptoms on young plants are the clearing of veins and chlorosis of lower leaves; this is followed by epinasty of the older leaves caused by drooping of the petioles (*25.11a).* Plants infected as seedlings often wilt and soon die. Severely infected older plants may wilt and die suddenly if the weather is favorable for pathogen development. Generally, in older plants, the first symptoms increase in intensity until the entire plant shows symptoms. The plants remain stunted, with occasional formation of adventitious roots, wilting of leaves and stems, defoliation, marginal necrosis of remaining leaves, and they eventually die (*25.11b).* A one-sided discoloration of the stem may occur during late stages of disease development and new, apparently healthy growth is produced from the base, while the top of the stem shows severe symptoms. The woody tissues of affected plants have a brown discoloration (*25.11c).* Fruit may occasionally become infected, rot and drop. Roots can be infected and stunted, with the smaller side roots rotting completely.

Causal agent *Fusarium oxysporum* f. sp. *lycopersici* is indistinguishable from other forms of *F. oxysporum* in pure culture. Abundant microconidia are produced on simple phialides arising laterally on the hyphae or from short, sparsely branched conidiophores. Microconidia are oval-ellipsoid, cylindrical, straight to curved, and measure 5 to 12 by 2.2 to 3.5 pm. Macroconidia are thin-walled, generally three- to five-septate, fusoid-subulate, pointed at both ends, have a hooked apex and a pedicellate base, and measure 27 to 66 by 3 to 5 pm. Chlamydospores are generally abundant, solitary and terminal or intercalary.

Several wilt diseases of tomato have similar symptoms; therefore, isolation and identification are necessary. The pathogen can be isolated from vascular tissue at the top of the plant. There are media for selective culturing of this fungus. A petri-dish test has been developed to distinguish between the fusarium wilt and fusarium crown and root rot pathogens (see fusarium crown and root rot, 25.10).

Disease cycle Long-distance dissemination may occur on seed, in symptomless transplants, and in soil associated with transplants. Once established, the fungus survives as chlamydospores in soil and in root residues. The disease is favored by low soil moisture, short daylength, low light intensity, low pH, plant tissues low in potassium, and soil temperatures around 28°C. Increasing levels of nitrate nitrogen reduce plant susceptibility to wilt. Wounding of the root system through improper handling of transplants favors the disease.

Management

Cultural practices — The use of disease- free seed and transplants helps to prevent the spread of fusarium wilt to uninfested greenhouses. If clean seed is not available, those of questionable status should be given a hot-water treatment (see bacterial canker, 25.1). The disease develops best at high temperatures (28°C), so excessive warming of propagating beds should be avoided. Crop rotation is of limited use because the pathogen survives for long periods in soil. Treatments that adjust the soil close to pH 7 help to control this disease, but a soil pH of 7.5 favors verticillium wilt. The use of peat modules or soilless, rockwool or NFT culture systems aids in control. Growers should supply adequate nitrate nitrogen to plants, but excessive fertilization favors the disease. Greenhouse structures, crates, benches and tools should be cleaned regularly. Precautions should be taken to reduce the spread of the pathogen in infested soil, on implements and by workers during movement between greenhouses, plant beds and production fields. Cultivation may cause root damage and increase the risk of infection.

Resistant cultivars — Fusarium wilt-resistant cultivars and rootstocks, such as KNVF types, provide the best means of control. When grafting susceptible scions to resistant rootstocks, the scion root-system must be severed before transplanting the grafted plants. Resistance may not be expressed when resistant cultivars are grown in soil infested with both *Fusarium* and root-knot nematodes because of physiological changes in the root induced by the nematodes.

Chemical control — Disinfestation of growing media with chemical fumigants or steam is effective and practical in most greenhouses.

Selected references

Brayford, D. 1992. *Fusarium oxysporum* f. sp. *lycopersici.* IMI Descriptions of Fungi and Bacteria, No. 1117. Internat. Mycol. Inst., Kew, Surrey, England. 4 pp.

Clayton, E.E. 1923. The relation of temperature to the fusarium wilt of the tomato. *Am. J. Bot.* 10:71-88.

Sarhan, A.R.T., B. Barna and Z. Kiraly. 1982. Effect of nitrogen nutrition on fusarium wilt of tomato plants. *Ann. Appl. Biol.* 101:245-250.

Sherwood, E.C. 1923. Hydrogen ion concentration as related to the fusarium wilt of tomato seedlings. *Am. J. Bot.* 10:537-553.

Walker, J.C. 1981. *Fusarium Wilt of Tomato.* Am. Phytopathol. Soc. Monogr. 6. 56 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.12 Gray mold (ghost spot) *Figs. 25.12a-d; 18.11 a-c*

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

Gray mold is a common disease of greenhouse tomato that can be minimized if the crop is managed properly. Tomato fruit is susceptible to infection during early stages of development. *Botrytis* survives as mycelium on decaying plant residue or as sclerotia that may persist in dry soil for several months or years. The pathogen has a wide host range that includes many vegetable crops (see Asparagus, botrytis blight, 4.1 ; and Lettuce, 11.10).

Symptoms Tomato leaflets, petioles, whole leaves, stems and fruit can be infected. Older tissues are generally more susceptible to attack than younger ones. Leaf lesions develop as light brown or gray, circular spots and may grow to cover the whole leaflet (*18.11a).* Affected leaves become covered with conidiophores and conidia, and subsequently collapse and wither. The fungus will grow from diseased leaves into the stem and produce dry, light brown lesions a few millimetres to several centimetres in length. Lesions also form at deleafing scars on the stem (*25.12a),* especially on older parts of the stem lying on the greenhouse floor. The stem lesions may also be covered with a gray mold. Severe infection can girdle the stem and kill the plant (*25.12b,c).*

On green tomato fruit, the most common symptom is ghost spot, which is a tiny brown, often raised, necrotic spot surrounded by a pale halo (*25.12d).* Once the fruit reaches 2.5 cm in diameter, the surface becomes smooth and shiny and resists infection; however, fruit can also become infected through flower parts stuck to the surface, especially at the calyx end, resulting in an irregular, brown lesion in the area of the flower parts.

Ghost spotting can also occur on ripe fruit (*18.11b)* and occasionally results in downgrading of shipments. Mature fruit may also be affected by a rot that starts at the calyx end (*18.11c).* Fruit becomes water-soaked and soft at the point of infection. The spots are irregular, up to 3 cm in diameter and light brown to gray. Rotting fruit eventually drops.

Causal agent *Botrytis cinerea* (see Lettuce, gray mold, 11.10) is readily identified by the presence of gray conidiophores, conidia or sclerotia on dead, pale gray to tan tissue. These signs are easily seen with the naked eye or a hand lens. Ghost spot on the fruit is also distinctive. On tomato stems, the mycelium becomes darker, the lesions blacken, and sclerotia may appear in the lesions as the fungus ages. Older lesions bearing sclerotia may be mistaken for those of didymella stem canker (see didymella stem canker, 25.8).

Disease cycle (see Lettuce, gray mold, 11.10)

Management

Cultural practices — Growers should maintain adequate heat and ventilation in the greenhouse, especially during the night. A relative humidity of less than 80% will deter gray mold development. Removal of lower leaves aids in disease prevention by allowing free flow of air through the crop. Leaves should be pruned to clean-cut stubs, 1 to 2 mm long, and kept dry by using drip irrigation or surface watering. Crop residue should be removed promptly because it acts as a source of spores.

Chemical control — Fungicide sprays help to control the disease when properly timed. Strains resistant to benomyl, dicloran, iprodione and captan are known, which is why fungicide rotations and combinations are recommended. Because infection of leaf scars may occur up to 10 to 12 weeks before symptoms appear, protective treatments should be considered at the time of deleafing if humid conditions prevail. Stem cankers can be treated by scraping diseased tissue off the stem and applying a thin paste of fungicide over and slightly beyond the affected area.

Selected references

Coley-Smith, J.R., K. Verhoeff and W.R. Jarvis, eds. 1980. *The Biology of Botrytis.* Academic Press,New York. 318 pp.

Ellis, M.B., and J.M. Waller. 1974. *Sclerotinia fuckeliana.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 431. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Locke, T., and J.T. Fletcher. 1988. Incidence of benomyl and iprodione resistance in isolates of *Botrytis cinerea* in tomato crops in England and Wales in 1986. *Plant Pathol.* 37:381-384.

Morgan, W. 1984. The effect of night temperature and glasshouse ventilation on the incidence of *Botrytis cinerea* in a late-planted tomato crop. *Crop Protection* 3:243-251.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.13 Late blight *Figs. 25.13a,b; 18.12a-d*

Phytophthora infestans (Mont.) de Bary

This disease is far more prevalent in areas with maritime versus mid-continental climates. It is common on potato and outdoor tomato. Under cool, humid conditions, greenhouse tomato can also be infected, whether grown in soil or in hydroponic production. Late blight also can attack eggplant, pepper and some solanaceous weeds.

Symptoms Initially, irregular, water-soaked, green-black spots appear at the tips or edges of the oldest leaves. Under humid conditions, the spots enlarge rapidly to form brown areas with indefinite borders (*25.13a; 18.12a,b).* Spore formation usually occurs at the margin of these lesions. A blue-gray growth of the pathogen may develop on lower leaf surfaces. The fungus may grow through the entire leaflet, affecting all the leaflets on a leaf, which then wilts and dies. Brownish cankers often form on the stems and petioles *(18.12c).*

Infection of the fruit can occur at any stage of development. Green-brown, water-soaked lesions may spread over the entire surface *(25.13b).* Under humid conditions, a blue-gray growth also may develop on affected fruits *(18.12d).*

Causal agent *Phytophthora infestans* (see Potato, late blight, 16.11) is normally identified by microscopic examination of diseased plant tissue. It produces long sporangiophores with thin-walled, oval, colorless sporangia. Water-soaked, green-black leaf lesions are characteristic. Badly affected crops have a fishy odor.

Disease cycle (see Potato, late blight, 16.11) Inoculum to initiate the disease on greenhouse tomato normally originates from nearby infected field potato or tomato crops. Overwintered trash piles are also potential sources of infective spores. Epidemic development is favored at 18 to 21 °C and high humidity.

Management

Cultural practices — Infected leaves should be carefully removed and buried. Any practices that reduce humidity within the crop also help to control this disease.

Chemical control — Fungicides aid in controlling late blight if applied as preventive sprays. If potato crops are grown close to greenhouse tomato crops, a routine spray application should be considered, especially in years when late blight is prevalent on potato. Tomato should be sprayed at the times indicated for potato. Because not all fungicides registered for potato can be used on tomato, growers should consult provincial recommendations.

Selected references

Stamps, D.J. 1985. *Phytophthora infestans.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 838. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Vartanian, V.G., and R.M. Endo. 1985. Overwintering hosts, compatibility types, and races of *Phytophthora infestans* on tomato in southern California. *Plant Dis.* 69:516-519.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.14 Leaf mold *Fig. 25.14*

Fulvia fulva (Cooke) Cif.
(syn. *Cladosporium fulvum* Cooke)

Leaf mold is most important on greenhouse tomato crops, especially in poorly ventilated plastic houses. It affects crops equally in soil or hydroponic production. It also attacks field tomato in cool, humid seasons. Tomato is the only plant affected by this disease.

Symptoms Symptoms usually occur only on the foliage, but they may involve blossoms and fruit. The first symptoms are indefinite, yellow-green areas on the upper surface of leaves, and in some cultivars and environments, pale, nearly white spots on the lower surface. Later, these areas coincide almost exactly with a brown to purplish, velvety fungal growth on the lower surface *(25.14).* Symptoms and signs appear first on older leaves, progressing onto younger ones. Infected leaves eventually become yellow-brown, curl, wither and drop prematurely. Infected blossoms usually die before fruit set. Green and ripe tomato fruits can develop a black, leathery, irregular, stem-end rot that may cover one- third of the fruit surface. Infected fruit may be lopsided with blackened radial furrows, remaining unripe on the affected side. The fungus can be isolated readily from conidia on leaf lesions.

Causal agent No sexual stage is known for *Fulvia fulva.* Conidiophores are unbranched, narrow at the base and broader apically, pale brown to dark at the apex, septate, and measure 57 to 125 by 1.3 to 7 µm. Conidia are brownish, cylindrical to ellipsoid, smooth, straight or slightly curved, and produced in chains. They are one- to two-celled, measure 12 to 47 by 4 to 10 µm, and have a conspicuous thickened hilum. On the plant, a pale sub-stomatal stroma is present.

The pathogen can be identified by its characteristic fruiting structures on diseased leaf tissue. A velvety, purple brown fungal growth beneath indefinite yellowish areas on the leaves is diagnostic. The fungus is readily isolated directly from conidia and it grows on most laboratory media. In culture, the colonies are effuse, velvety, buff to brown or purplish, with a whitish margin.

Disease cycle Disease development is favored by a relative humidity of 85% or more or by liquid water on the leaves. Germination can occur between 5 and 35°C; the optimum temperature is 22°C. The pathogen produces large numbers of conidia on infected tissue. Once the primary infection has occurred, the disease spreads rapidly through the greenhouse. The conidia are readily dispersed by air currents, water, workers moving through the crop, and by insects.

The pathogen survives from crop to crop as sclerotia, conidia or mycelium in soil or crop residues. Conidia are known to survive at least one year under adverse conditions, and new conidia are readily produced in the leaf on substomatal stromata. Contaminated seed may facilitate the widespread dispersal of new races, but spread of the pathogen between greenhouses commonly occurs on workers' clothing.

Management

Cultural practices — Adequate row and plant spacings are necessary to avoid excessive shading and to improve air circulation. As well, growers should avoid excessive nitrogen fertilization. The relative humidity in the greenhouse should not exceed 85%, particularly at night, and water droplets should not be allowed to form and persist on leaves. If the greenhouse is not heated, ventilation should be increased and the lower leaves removed to improve air circulation around the plants. Excessive vegetative growth retards ventilation. Overhead watering and pesticide sprays should be applied early in the day to keep humidity low later in the day. Circulation of unheated air through the greenhouse aids spore dispersal and may keep the relative humidity high. Diseased leaves should be carefully pruned, placed in a plastic bag and destroyed. If leaf mold has been a problem in the crop, all plant residue should be removed and destroyed at the end of the season, and the entire greenhouse should then be disinfested.

Resistant cultivars — Susceptible cultivars can be grown if humidity is kept low but resistant cultivars are preferable. Although a tomato cultivar may carry resistance, leaf mold has a number of races, so the resistance genes must coincide with the local races. Commercial seed is usually labeled as to cultivar resistance to known races. Local authorities should be consulted about cultivars that possess resistance against local populations of the pathogen. Resistant cultivars include Caruso, Capello, Cobra, Vision (from the Netherlands), Buffalo, Trend, Pink KR15 and Pink CR-864 with gene *Cf-5,* Ultra Sweet and Ultra Pink with gene *Cf*-7, and Dombito, Jumbo, Furon and Vetomold with gene *Cf*-2.

Chemical control — Fungicides can be used to control this pathogen, but care must be taken in selection because fungicide-resistant strains of the pathogen exist.

Selected references

Ellis, M.B. 1971. *Dematiaceous Hyphomycetes.* Commonw. Mycol. Inst., Kew, Surrey, England. 608 pp.
Gardner, M.W. 1925. Cladosporium leaf mold of tomato; fruit invasion and seed transmission. *J. Agric. Res.* 30:519-540.
Higgins, V.J., and J. Hollaway. 1987. Prevalent races of *Cladosporium fulvum* in southern Ontario and their benomyl sensitivity. *Can. J. Plant Pathol.* 9:32-35.
Holliday, P., and J.L. Mulder. 1976. *Fulvia fulva.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 487. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.15 Septoria blight (septoria leaf spot) *Figs. 25.15; 18.13a-c*

Septoria lycopersici Speg.

This disease occurs occasionally in poorly ventilated greenhouse tomato crops and overcrowded plug-transplants (25.75). Tomato is the main host of *S. lycopersici*, but the pathogen can also infect black nightshade *(Solanum nigrum* L.) and several other *Solanum* species.

Symptoms Numerous, small, circular, water-soaked spots are produced on petioles, leaves, stems (*18.13c*) and calyces. Septoria leaf spot develops more quickly on the upper leaf surface than on the lower leaf surface *(18.13a).* Normally, older leaves are affected first. The spots have gray centers with black borders, or they may be solid black. In their centers, minute, black pycnidia can be seen under magnification *(18.13b).* Severely affected leaves turn yellow, dry and fall off. Fruit may be exposed to the sun as a result of defoliation, leading to sunscalded, leathery and/or bleached patches on the skin.

Causal agent *Septoria lycopersici* has hyaline, thin-walled hyphae. Hyaline, filiform conidia, measuring 3.2 by 67 pm with up to 10 septa, are produced in pycnidia, which average about 66 pm in width. The fungus is readily isolated directly from cirrhi of conidia extruded from the pycnidia. The best linear growth is obtained on tomato leaf extract agar, potato dextrose agar and carrot agar. The fungus produces mature pycnidiospores after 7 days on tomato leaf extract agar and tomato root extract agar. Optimum temperature for fungal growth on media is 22-25°C and for pycnidial maturation it is 17-28°C. Lesions of 5.*lycopersici* may resemble those of *Alternaria* spp. (see early blight, 25.9), but they have pycnidia and distinctive, long, filiform conidia.

Disease cycle The pathogen can survive from season to season in or on seed, diseased plant residue and infected hosts, and on contaminated greenhouse structures and equipment. Spores can be spread by splashing water, workers, equipment, insects, such as aphids, and wind-blown soil. Wet weather favors spread of the fungus. Growth is greatest at 15 to 25°C, and disease development requires 7 days at 20-26°C and 12 days at 15-20°C. At the higher and lower temperature ranges, infection requires 64 and 88 hours, respectively. In the greenhouse, the disease is favored by warm, dry days and dewy nights. At 100% relative humidity, only 9-10 days are required for the disease process from inoculation to formation of pycnidia. This saturated environment is necessary for abundant spore discharge from pycnidia.

Management

Cultural practices — Good weed control and sanitation in and around the greenhouse enhance disease control. Diseased leaves and stems should be carefully gathered and buried. Field tomato should not be planted close to greenhouses. Seed should be purchased from a reputable source.

Chemical control — Registered fungicides are available.

Selected references

Cook, A.A. 1954. Reaction of *Lycopersicon* species to regional isolates of *Septoria lycopersici. Phytopathology* 44:367-374.

Marcinkowska, J. 1977. Septoria leaf spot of tomato. I. Development of Septoria leaf spot on tomato plants under greenhouse and field conditions. *Acta Agrobotanica* 30:341-358.

Marcinkowska, J. 1977. Septoria leaf spot of tomato. II. Morphology and development of *Septoria lycopersici* Speg. *Acta Agrobotanica* 30:359-372.

Sutton, B.C., and J.M. Waterston. 1966. *Septoria lycopersici.* CMI Descriptions of Pathogenic Fungi and Bacteria. No. 89. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.16 Verticillium wilt *Figs. 25.16a,b*

Verticillium albo-atrum Reinke & Berthier
Verticillium dahliae Kleb.

This is a minor disease of greenhouse tomato in Canada. Both *Verticillium* species have been found on this crop, but *V. dahliae* predominates.

Symptoms (see Tomato, verticillium wilt, 18.14) In the greenhouse, the first visible above-ground symptom is the wilting of one leaflet or more on a single leaf. The oldest leaves usually show symptoms first, and then wilt develops progressively in the younger leaves. Plants that become infected during January, February and early March show sudden wilting of several leaves, which become characteristically patterned with bright yellow and brown *(25.16a).* These plants often die in a few days. During late spring and summer, affected plants usually survive in a considerably stunted, wilted condition *(25.16b).* Heavy watering may induce rapid upward movement of the fungus in infected plants.

Causal agent (see Potato, verticillium wilt, 16.20)

Disease cycle (see Greenhouse cucumber, verticillium wilt, 22.17)

Management (see Greenhouse cucumber, verticillium wilt)

Selected references

Hawksworth, D.L., and P.W. Talboys. 1970. *Verticillium albo-atrum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 255. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

Hawksworth, D.L., and P.W. Talboys. 1970. *Verticillium dahliae.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 256. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by R.J. Howard)

▶ 25.17 White mold *Figs. 18.15a-e*

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

White mold is occasionally serious in individual greenhouses. The pathogen has a wide host range (see Tomato, white mold, 18.15) and can attack most types of greenhouse vegetables (see Greenhouse cucumber, white mold, 22.18; and Greenhouse lettuce, drop, 23.8).

Symptoms (see Tomato, white mold)

Causal agent (For a description of *Sclerotinia sclerotiorum*, see Bean, white mold, 15B.9; and for *S. minor*, see Lettuce, drop, 11.9.)

Disease cycle (see Bean, white mold; and Lettuce, drop.)

Management (see Tomato, white mold, and Greenhouse cucumber, white mold)

Selected references

Mordue, J.E.M., and P. Holliday. 1976. *Sclerotinia sclerotiorum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 513. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.

(Original by R.J. Howard)

VIRAL DISEASES

▶ 25.18 Cucumber mosaic *Figs. 25.18a,b; 18.17*

Cucumber mosaic virus

Cucumber mosaic occurs sporadically in British Columbia, Ontario and Quebec, but it has not been a severe problem. Cucumber mosaic virus has a broad natural host range throughout the temperate regions of the world. It can infect cereals, forages, woody and herbaceous ornamentals, vegetables and fruit crops.

Symptoms Cucumber mosaic virus infection of tomato produces a symptom known as "shoestring," in which the blade of the leaflet is much reduced (tendril-like) or absent, consisting of only the petiole *(25.18a).* Shoestring can be confused with "fernleaf," a symptom caused by tomato mosaic virus or tobacco mosaic virus, in which the leaflets are long and narrow but not as completely suppressed as in shoestring. This first appears about 10 days after infection and consists of a spindling appearance of the young leaves in the terminal bud. These leaves twist in a corkscrew fashion. Another early symptom is yellowing of the older leaves, especially along the veins *(25.18b).*

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

Disease cycle Cucumber mosaic virus is transmitted by numerous species of aphids in a non-persistent or stylet- borne manner. Aphids can acquire the virus from an infected plant and inoculate healthy plants after less than one minute of feeding, and there is no latent or waiting period before the virus can be transmitted. Problems with cucumber mosaic usually occur only when aphids and host plants in which the virus can multiply are present throughout the year. Perennial weed species have been shown to harbor the virus throughout the winter, and several of them, including chickweed *(Stellaria media* (L.) Cyrill and *Cerastium* spp.), *Capsella* spp., corn spurry (*Spergula arvensis* L.), and red dead-nettle (*Lamium purpureum* L.), are known to carry the virus through their seed. The virus commonly infects chrysanthemum and may be spread to tomato by aphids. Once introduced into a greenhouse tomato crop, it can be further spread to healthy plants on tools or the hands of workers.

Management

Cultural practices — No direct control methods, such as viricides, are available, so most control strategies involve measures designed to reduce sources of infection from within or outside of the crop. Since contaminated seed does not appear to be a problem, only a few precautions are required to keep seedlings virus-free. Growers should control weeds within and near the greenhouse. Aphid control in the greenhouse is essential because viruliferous aphids are the most common means of virus introduction and spread. Any suspicious plants should be removed and destroyed. Care should be exercised to minimize mechanical transmission while handling plants. Dipping hands and tools in a solution of skim milk (100 g skim milk powder per litre of water) helps to minimize spread (see tobacco mosaic, 25.20).

Selected references

Francki, R.I.B., D.W. Mossop and T. Hatta. 1979. Cucumber mosaic virus. CMI/AAB Descriptions of Plant Viruses, No 213. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

Kaper, J.M., and H.E. Waterworth. 1981. Cucumoviruses. Pages 257-332 *in* E. Kurstak, ed., Handbook *of Plant Virus Infections and Comparative Diagnosis.* Elsevier/North Holland Biomedical Press, Amsterdam. 944 pp.

(Original by R. Stace-Smith)

▶ 25.19 Double streak *Figs. 25.19a,b*

Tomato mosaic virus and potato virus X

This disease occurs in plants infected by both potato virus X and tomato mosaic virus. Double streak is a minor disease of greenhouse tomato, but its impact on plant health and fruit production is usually greater than that caused by either virus alone (see tomato mosaic, 25.21, 18.18; other viral diseases, 18.20).

Symptoms The onset of symptoms is usually very sudden. Necrotic lesions form on the stem, petioles, leaves and fruit. Stem lesions (*25.19a*) appear as dark, longitudinal streaks extending through the cortex and into the pith beneath. The leaves often show necrotic spots that enlarge and blight the foliage. Fruits may be affected while still green and develop irregular, sunken, necrotic blotches (*25.19b).* Diseased plants are generally stunted and weakened and may die. Symptom expression is suppressed by air temperatures of 27°C or higher.

Causal agent (For tomato mosaic virus, see tomato mosaic, 25.21; for potato virus X, see Potato, mosaic diseases, 16.27).

Disease cycle (see tomato mosaic, 25.21) Double streak is usually found in tomato when potato is grown nearby or when workers handle potato plants before tomatoes. It also has been encountered when unsterilized soil containing potato debris has been used in greenhouses and in greenhouses constructed on land where potato has been grown earlier in the season.

Management

Cultural practices — Diseased tomato plants should be removed from the greenhouse and buried. Potato should not be planted near tomato greenhouses nor should it be handled before working in a tomato crop. Tomatoes should not be grown in soil containing fresh potato crop residues.

Selected references

Asselin, A. 1984. A note on the induction of the streak disease in different tomato cultivars by tomato mosaic virus and potato virus X. *Phytoprotection* 65:81-83.

Caron, M., and R.O. Lachance. 1978. Une forme de bigarrure de la tomate. *Phytoprotection* 59:76-84.

Jarvis, W.R., and C.D. McKeen. 1992. *Tomato Diseases.* Agric. Can. Publ. 1479/E. 70 pp.

MacNeill, B.H., and H. Ismen. 1960. Studies on the virus-streak syndrome in tomatoes. *Can. J. Bot.* 38:9-20.

(Original by J.G. Menzies and R.J. Howard)

▶ 25.20 Tobacco mosaic

Tobacco mosaic virus

Tobacco mosaic is a minor disease of greenhouse tomato. Tobacco mosaic virus is one of the most infectious plant viruses and has a host range of over 150 genera, including greenhouse pepper (see Greenhouse pepper, tobacco mosaic, 24.6) and field tomato and pepper.

Symptoms The symptoms of tobacco mosaic on greenhouse tomato are virtually indistinguishable from those of tomato mosaic (see tomato mosaic, 25.21).

Causal agent Tobacco mosaic virus is in the tobamovirus group. It is rod-shaped, measures 300 by 18 nm, and consists of a single helical strand of RNA with approximately 6400 nucleotides and 2130 protein subunits. Its thermal inactivation point in undiluted plant juice is 93°C. In dried infected leaves, it remains infective even after treatment at 120°C for 30 minutes.

Indicator plants commonly used for confirming tobacco mosaic virus include *Chenopodium. Nicotiana (N. glutinosa*, *N. sylvestris* and *N. tabacum* cvs. Java, Turkish, Turkish Samsun, Samsun (Samsoun), Samsun NN, White Burley, Burley, Xanthi and Xanthi-nc), and *Phaseolus (P. vulgaris* cv. Pinto).

Strains of tobacco mosaic virus are closely related to tomato mosaic virus (see tomato mosaic, 25.21) and their names are often interchanged in the literature. They can be distinguished in the laboratory by serology and, to some extent, by symptoms on indicator plants.

Disease cycle Tobacco mosaic virus is soil-borne and survives in infested plant residues. It also can be seed-borne. The virus is not normally transmissible by insects. Field tomato and tobacco crops may be a source of infection for greenhouse crops, as may infected weeds, though the importance of the latter has not been determined. Spread of tobacco mosaic virus can be rapid because it is readily transmitted by rubbing infected plants against healthy ones, or with hands and tools contaminated with infected sap or residue from tobacco products. Symptom development generally is more severe with short photoperiods and low light intensities. Temperature also affects symptom development and severity in susceptible varieties; lesions develop 16 to 18 days after inoculation at 22 to 28°C, whereas symptoms may not occur at 16 to 20°C.

Management

Cultural practices — Seed should be obtained from virus-free plants. Heat-treated seed (70°C for four days) or seed obtained by acid extraction (see tomato mosaic, 25.21) also can be used safely. One to two days before planting, seed should be soaked for 15 minutes in a 10% trisodium phosphate solution (100 g/L of water), then rinsed thoroughly and spread to dry. Early in the season, diseased plants should be removed when noticed. Later in the season, roguing is ineffective because the virus will have spread and symptomless infected plants will be present within the crop. Plants with symptoms should be handled only after healthy or symptomless plants have been tended.

Sprays of skim milk may slow the spread of tobacco mosaic. The spray solution can be prepared by adding dried skim milk powder to water at the rate of 100 g/L. This mixture should be used each time the plants are pruned or trained, from the time of transplanting to the first fruit harvest. If symptoms are not evident on plants at first harvest, the sprays can be discontinued. If symptoms are noticed on plants after harvesting commences, the affected plants should be picked last, then sprayed with milk powder solution. If virus infection is suspected, workers should dip their hands in skim milk solution between handling individual plants. The mechanism by which skim milk inactivates tobacco mosaic virus is unclear, but it may be a result of binding of the viral particles by protein molecules, thus inhibiting their ability to infect plant cells.

Growers are advised to prohibit the use of tobacco products in the greenhouse and to require workers to wash their hands thoroughly with soap and water after using tobacco products. Clothing should be laundered daily in hot water with a detergent.

Resistant cultivars — Growers should select tomato cultivars with resistance to tomato mosaic virus (see tomato mosaic, 25.21) if tobacco mosaic is a potential problem.

Selected references
Hare, W.W., and G.W. Lucas. 1959. Control of contact transmission of tobacco mosaic virus with milk. *Plant Dis. Rep.* 43:152-154.
Hare, W.W., and G.B. Lucas. 1960. Some effects of pH and milk on tobacco mosaic virus. *Phytopathology* 50:638.
John, C.A., and C. Sova. 1955. Incidence of tobacco mosaic virus on tomato seed. *Phytopathology* 45:636-637.
Zaitlin, M., and H.W. Israel. 1975. Tobacco mosaic virus (type strain). CMI/AAB Descriptions of Plant Viruses, No. 151. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

(Original by W.R. Jarvis, J.G. Menzies and R.J. Howard)

▶ 25.21 Tomato mosaic, single streak *Figs. 25.21a-e; 18.18a-e*

Tomato mosaic virus

This disease occurs wherever tomato is grown and can reduce both yield and quality of fruit (*18.18b-e*). The impact of the disease is even more severe if tomato mosaic and potato virus X occur as a mixed infection (see double streak, 25.19). Tomato mosaic virus can infect plants in the families Solanaceae, Aizoaceae, Amaranthaceae, Chenopodiaceae and Scrophulariaceae. Petunia, snapdragon, pepper and tobacco are frequent hosts.

Symptoms Symptoms of tomato mosaic vary with the strain of the virus, temperature, daylength, light intensity, plant age and tomato cultivar. Initially, affected plants may wilt in sunlight, especially if the crop is growing rapidly. The wilt is temporary, lasting up to two weeks. The leaves most commonly show a light to dark green mottling *(25.21 a,b).* There may also be a reduction in leaflet width, so that individual leaves resemble fern leaves. The lowest distorted leaf generally shows reduced serration; younger leaves show increasing simplification until the individual leaflets are reduced to a narrow strap of tissue *(18.18a).* These “fern- leaves” may have small enations on the underside. Plants recovering from severe leaf distortion produce pinnate, vetch-like leaves (fernleaf, *25.21c).* Eventually, new leaves are produced that are normal in shape but have a conspicuous mosaic. Six to eight leaves can be affected. Involvement of all leaves is not typical. Stem symptoms include pale to dark green or black stripes, often accompanied by yellow-brown older leaves. Plant size is generally reduced.

Brown markings and blotches occasionally occur on green and ripe fruit of greenhouse tomato cultivars that have some resistance (heterozygous for the gene *Tm2)* to tomato mosaic. These blemishes are often circular, up to 3 cm in diameter, and confined to the skin. Usually, the fruit of only one or two trusses is affected, but yield can be severely reduced on young plants. Blotches can appear on both green and ripe fruit, which soon drop off. Sunken areas, sometimes brown or black, and known as pits, may form at the calyx end of the fruit. Fruit pitting is generally restricted *(25.21d).* The virus also causes internal browning of the fruit wall *(18.18d,e)* and abortion of flowers and fruit, usually confined to trusses developing flowers at the time of infection.

Foliar streaking has been reported to be caused by at least one strain of tomato mosaic virus; this condition is referred to as single streak, single virus streak or glasshouse streak. The most characteristic symptoms are longitudinal, necrotic streaks on the stems, leaves and petioles, which sometimes kill the plant. In addition, sunken brown lesions may develop on the fruit *(25.21e).* Streak generally occurs at 26°C or below. Symptoms have been reproduced experimentally by grafting but not by sap inoculation.

Causal agent Tomato mosaic virus is an RNA virus. The particles are straight tubules, measuring 18 by 300 nm. Purified preparations sediment as a major infective component, sometimes also with dimers and trimers.

Indicator plants, such as *Brassica, Chenopodium, Cucumis, Datura, Gomphrena, Nicotiana, Phaseolus, Tetragonia* and *Vigna* spp., distinguish tomato mosaic from the type strain or tobacco forms of the virus, but none is infallible. On *Nicotiana glutinosa,* local lesions differ in size and incubation period from those of tomato spotted wilt and tomato bushy stunt. Strains of tomato mosaic virus are closely related to tobacco mosaic virus (see tobacco mosaic, 25.20).

Disease cycle The most important inoculum sources are seed, plant residues and soil. Alternative hosts in and around greenhouses are another source. Tomato mosaic virus is not normally transmissible by insects. In tomato seed, this virus has been found in the mucilage, testa and endosperm. The virus can survive in plant residue in the soil for up to two years and in moist soil for up to eight months, although its concentration decreases with time. People are the most important vectors of tomato mosaic in the greenhouse. Employees or visitors can spread the virus through contaminated clothing and pruning knives as they tend the crop or walk along and brush the plants. The virus can survive for up to three years on stored unwashed clothing. It is quickly inactivated by sunlight.

Management

Cultural practices — Seed should be obtained from healthy plants. As an extra precaution, one to two days before seeding, seed should be soaked for 15 minutes in trisodium phosphate solution (100 g/L) at room temperature, rinsed thoroughly and spread out to dry. In addition, dried seed can be heated in an oven for four days at 70°C to eliminate surface-borne virus. Perhaps the best method of disinfesting tomato seed is to treat the fruit pulp with one quarter of its volume of concentrated hydrochloric acid and allowing it to sit for 30 minutes at room temperature before straining and washing the seed.

Seedlings and transplants should be grown in soilless mixes or in steam-pasteurized soil in which plant debris has been allowed to thoroughly decompose. Steam-pasteurizing may not kill virus particles in thick roots left in the soil. Seedlings should not be grown in areas of the greenhouse where tomato crops or other hosts are being grown. Infected plants should be removed from the greenhouse and destroyed. Contaminated equipment, tools and machinery should be cleaned by washing, then heat-sterilizing, dipping or rewashing in trisodium phosphate (3 kg/100 L of water) before reuse. Virus transmission between plants can be reduced if tools are frequently dipped in a solution of 10% trisodium phosphate. After working an infected crop, a change to freshly laundered clothing is suggested before going to a mosaic-free crop. Infested clothing should be laundered in hot water with a detergent.

Perennial plants and hanging baskets of ornamentals should not be tolerated in a tomato greenhouse, and greenhouses should be sealed with screens to exclude potential insect vectors.

Resistant cultivars — The most effective means of controlling tomato mosaic is through the use of resistant cultivars. Although most commercial cultivars are resistant to one or more strains of the virus, the resistance of some cultivars can be overcome if young plants are exposed to infection or temperatures exceed 30°C.

Biological control — Mild, almost symptomless strains of tomato mosaic are commercially available to vaccinate susceptible cultivars against more severe strains. While this technique can minimize disease losses, the mild strains may recover virulence and cause appreciable damage later in the season. For this reason, attenuated strains are rarely used in Canada.

Chemical control — Fumigation does not control tomato mosaic virus and may slow the rate of virus inactivation by reducing the populations of soil microflora that break down plant residues.

Selected references

Allen, W.R. 1984. Mode of inactivation of TMV in soil under dehydrating conditions. *Can. J. Plant Pathol.* 6:9-16.

Asselin, A. 1984. A note on the induction of the streak disease in different tomato cultivars by tomato mosaic virus and potato virus X. *Phytoprotection* 65:81-83.

Broadbent, L. 1964. The epidemiology of tomato mosaic. VII. The effect of TMV on tomato fruit yield and quality under glass. *Ann. Appl. Biol.* 54:209-224.

Broadbent, L. 1965. The epidemiology of tomato mosaic. XI. Seed-transmission of TMV. *Ann. Appl. Biol.* 56:177-205.

Broadbent, L., and J.T. Fletcher. 1963. The epidemiology of tomato mosaic. IV. Persistence of virus on clothing and glasshouse structures. *Ann. Appl. Biol.* 52:233-241.

Broadbent, L., W.H. Read and F.T. Last. 1965. The epidemiology of tomato mosaic. X. Persistence of TMV-infected debris in soil, and the effects of soil partial sterilization. *Ann. Appl. Biol.* 55:471-483.

Hollings, M., and H. Huttinga. 1976. Tomato mosaic virus. CMI/AAB Descriptions of Pathogenic Plant Viruses, No. 156. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 6 pp.

(Original by J.G. Menzies and W.R. Jarvis)

▶ 25.22 Tomato spotted wilt *Figs. 25.22a-d*

Tomato spotted wilt virus

This virus is common in temperate regions and can cause significant losses in both field and greenhouse tomato (see also 18.19). The host range of the pathogen includes approximately 300 species in 34 families of plants.

Symptoms The most common symptom of tomato spotted wilt is bronzing of young leaves (*25.22a,b),* followed by one-sided distortion, severe stunting and near-cessation of growth. Bronzing occurs as isolated spots or it may cover most or all of the leaf surface. The intensity of bronzing varies from an inconspicuous green to a brown, distinct dark-brown or almost black, glazed area. Bronzed areas usually roll inward and tissue in affected areas often dies. Necrotic lesions may develop on petioles. Fruits on affected plants develop spots about 1 cm in diameter with concentric, circular markings. Ripe fruits often are distorted and marked with alternate red and yellow bands (*25.22c,d).* Affected fruits may occasionally show internal browning.

Causal agent Tomato spotted wilt virus is an RNA virus with membrane-bound isometric particles, 70 to 90 nm in diameter. The structure of the material inside the membrane consists of a nearly continuous layer of projections about 5 nm thick that stain more densely than the membrane itself. Purified particles may show a tail-like extrusion. Physically and chemically, this is one of the most unstable plant viruses. There are many strains of the virus and the symptoms they produce may differ in severity.

Cucumis, Nicotiana, Petunia, Tropaeolum and *Vinca* spp. are suitable indicator plants for artificial inoculation. It is useful to keep a few petunia plants in a greenhouse as sensitive indicators of the presence of tomato spotted wilt virus.

Disease cycle The primary means of virus spread is by thrips. The virus may also be transmitted through seed. Disease spread is not serious unless thrips are present. Infected cuttings of ornamental plants and weeds can act as sources of infection. Only thrips can acquire the virus. This occurs after feeding periods of at least 15 minutes, after which they transmit it as adults by feeding.

The incubation period is 4 to 10 days; thrips become maximally infective 22 to 30 days after acquisition and may retain the virus for life. The virus is not transmitted from one generation of thrips to another.

Management The basis for control of tomato spotted wilt virus is sanitation, removal of alternative hosts, and thrips control (see thrips, 25.29).

Cultural practices — Infected plants should be removed and buried, and a 3- to 6-m-wide band around the perimeter of greenhouses should be kept free of weeds. Ornamental plants should not be grown in or around the greenhouse because they may act as reservoirs for the virus and its thrips vector.

Selected references

Allen, W.R., and A.B. Broadbent. 1986. Transmission of tomato spotted wilt virus in Ontario greenhouses by *Frankliniella occidentalis. Can. J. Plant Pathol.* 8:33-38.

Hsu, H., and R.H. Lawson, eds. 1991. *Virus-Thrips-Plant Interaction of Tomato Spotted Wilt Virus.* Proc. USDA Workshop, ARS-87. U.S. Dep. Agric. Washington, D.C. 170 pp.

Ie, T.S. 1970. Tomato spotted wilt virus. CMI/AAB Descriptions of Plant Viruses, No. 39. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 4 pp.

Paliwal, Y.C. 1974. Some properties and thrips transmission of tomato spotted wilt virus in Canada. *Can. J. Bot.* 52:1177-1182.

Paliwal, Y.C. 1976. Some characteristics of the thrips vector relationship of tomato spotted wilt virus in Canada. *Can. J. Bot.* 54:402-405.

(Original by J.G. Menzies and W.R. Jarvis)

NON-INFECTIOUS DISEASES

▶ 25.23 Blossom-end rot *Figs. 25.23; 18.21a-d*

Blossom-end rot is usually associated with environmental stresses, such as drought or widely fluctuating moisture conditions and calcium deficiency within the fruit. It appears as a firm, dry, sunken, brown or black area on the blossom-end *(25.23, 18.21c*), although the discoloration may sometimes be totally within the fruit (*18.21d*). This disorder can be prevented by regulating available water and by providing supplemental calcium. (For more information, see Tomato, blossom-end rot, 18.21.)

Selected references

Bradfield, E.G., and C.G. Guttridge. 1984. Effects of night-time humidity and nutrient solution concentration on the calcium content of tomato fruit. *Sei. Hortic.* 22:207-217.

Ward, G.M. 1964. Greenhouse tomato nutrition — a growth analysis study. *Plant Soil* 21:125-133.

Ward, G.M., and M.J. Miller. 1970. Relationship between fruit sizes and nutrient content of greenhouse tomatoes and cucumbers. *Can. J. Plant Sei.* 50:451-455.

(Original by R.J. Howard)

▶ 25.24 Magnesium deficiency *Figs. 25.24a,b*

Magnesium deficiency is a common nutritional disorder of greenhouse tomato but yield losses are rare unless the shortage of this element is acute. The middle leaves usually show symptoms first. They tend to be brittle and may cup upwards *(25.24a).* The veins remain dark green and somewhat bluish, with a thin, dark green leaf margin *(25.24b).* Interveinal areas turn yellow then brown. The greater the distance from the vein, the more intense the discoloration. Stems and fruits have no obvious symptoms. Dead leaf tissues may be colonized by secondary microorganisms.

Management

Cultural practices — Growers should ensure that tomato plants receive an adequate supply of essential nutrients throughout the growing period. Where lime is required in the fertilizer program, a limestone that contains magnesium, such as dolomite, should be used. At the first appearance of symptoms, magnesium sulfate (Epsom salt) should be applied two or three times to the growing medium at 1.0 to 1.5 kg/100 m^2 or to the leaves as a spray at 5 kg/1000 L of water. Where the problem has occurred on previous crops, magnesium should be applied even before symptoms occur. Over-application of potassium may impede magnesium uptake.

Selected references

Sonneveld, C. 1987. Magnesium deficiency in rockwool-grown tomatoes as affected by climatic conditions and plant nutrition. *J. Plant Nutrition* 10:1591-1604.

Ward, G.M., and M.J. Miller. 1969. Magnesium deficiency in greenhouse tomatoes. *Can. J. Plant Sci.* 49:53-59.

Ward, G.M., and M.J. Miller. 1970. Relationship between fruit sizes and nutrient content of greenhouse tomatoes and cucumbers. *Can. J. Plant Sci.* 50:451-455.

Winsor, G., and P. Adams. 1987. *Diagnosis of Mineral Disorders in Plants.* Vol. 3. *Glasshouse Crops.* H.M.S.O., London. 168 pp.

(Original by R.J. Howard)

▶ 25.25 Other disorders *Figs. 25.25a-e; 18.22-18.28*

Blotchy ripening
Catface
Edema
Growth cracks (russeting)
Puffiness

Blotchy ripening

Uneven ripening of the surface of the fruit is characteristic of this physiological disorder *(25.25a; 18.22a,b).* Inadequate nutrition, overcrowding, tomato mosaic and other factors may be responsible. Growers should follow a well-balanced fertilizer program to minimize the occurrence of blotchy ripening. (For more information, see Tomato, blotchy ripening, 18.22.)

(Original by R.J. Howard)

Catface

Catfacing is generally a minor problem on greenhouse tomato but can become serious if environmental control is poor and developing blossoms are injured by low temperatures. Affected fruits are misshapen and the blossom-end may be scarred *(18.23).* Maintaining optimum conditions for growth, especially air temperature, will minimize the incidence of catface. Good growing practices should be followed for crops destined both for fruit production and field transplanting. The cultivars Ohio WR25 and Ohio MR 13 seldom develop catface. (For more information, see Tomato, catface, 18.23.)

(Original by R.J. Howard)

Edema

In poorly ventilated greenhouses, and particularly in plastic greenhouses, a physiological disorder, edema, may occur. It is caused by waterlogging of the leaf tissues, which results in raised, blister-like growths, 2 to 5 mm in diameter, on the upper or lower leaf surface *(25.25b).* Green, callus-like growths result when transpiration is restricted and root pressure continues to pump water to the leaves (see Crucifers, intumescence, 8.21). Edema also occurs on fruits *(25.25c,d),* but it rarely affects yield and is easily counteracted by proper ventilation and watering.

(Original by W.R. Jarvis)

Growth cracks (russeting)

Growth cracking *(25.25e, 18.25*) is occasionally a problem in greenhouse tomato crops. Severe splitting can be avoided by careful watering. Russeting may be a less severe form of growth cracking in which the skin of fruit develops small cracks. Its occurrence is influenced by the ratio of fruit load to leaf area, but environmental factors such as relative humidity also may be involved. Reliable management strategies are not available. (For more information, see Tomato, growth cracks, 18.25.)

(Original by L.M. Tartier and J.G. Menzies)

Puffiness

This disorder *(18.28)* is usually a minor problem on greenhouse tomato. Proper temperature control and adequate humidity in greenhouses help to reduce this problem. (For more information, see Tomato, puffiness, 18.28.)

(Original by L.M. Tartier)

NEMATODE PESTS

▶ 25.26 Root-knot nematodes *Figs. 25.26; 18.30; 22.30a-d*

Northern root-knot nematode

Meloidogyne hapla Chitwood

Southern root-knot nematodes

Meloidogyne arenaria (Neal) Chitwood

Meloidogyne incognita (Kofoid & White) Chitwood

Meloidogyne javanica (Treub) Chitwood

Tomato, pepper and eggplant are very susceptible to damage from root-knot nematodes. Infected transplants from southern latitudes are a source of inoculum for *M. incognita, M. javanica* and *M. arenaria,* which can persist in greenhouses but apparently not outdoors in Canada.

Symptoms include stunting, chlorosis, early senescence, prolific branching of rootlets, and production of small, spherical galls on roots. Tomato plants infected by *M. incognita* may show larger, compound root galls *(25.26)* and purpling of the undersides of the leaves, similar to symptoms of phosphorus deficiency. For a complete description, see Carrot, 6.20, and Greenhouse cucumber, 22.30.

Management Pasteurization of soil, fumigation, and planting of certified, nematode-free transplants are effective. See Carrot, 6.20; see also Management of nematode pests, 3.12.

INSECT PESTS

▶ 25.27 Greenhouse whitefly *Figs. 25.27a-g; 3.10e*

Trialeurodes vaporariorum (Westwood)

The greenhouse whitefly is the both the most common and the most serious pest of greenhouse tomato in Canada and is found in all regions where this crop is grown.

The greenhouse whitefly feeds on over 100 genera of broadleaved plants. It prefers tomato, cucumber and eggplant in greenhouses and is rarely a problem on greenhouse pepper. Contamination of greenhouse-propagated seedlings can result in infestations on various outdoor crops such as cucumber, tomato, eggplant, melon and related plants. The whitefly will feed and develop on most weeds in and around greenhouses, and it is found on a large number of commonly grown ornamentals.

There are no records of the greenhouse whitefly as a vector of virus or other plant diseases to tomato, but it can transmit beet pseudo-yellows virus in cucumber (see Greenhouse cucumber, 22.19).

Damage The greenhouse whitefly damages the plant by sucking sap from the phloem bundles in the leaves. Large infestations can cause yellowing and wilting of the leaves, and a general decline of the plants. When feeding, nymphs and adults eject a sticky, sugar-rich honeydew that coats leaves and fruit. Production costs increase because honeydew deposits must be washed from fruit. If fruit washing is a normal part of the packing process, then moderate honeydew deposits can be tolerated.

Heavy whitefly infestations decrease the vigor of the plant by their feeding. However, greater declines in yield and fruit quality may result from a black sooty mold *(25.27g)* that grows on the honeydew when the humidity is high. The mold is more damaging than the honeydew because it reduces photosynthesis and transpiration.

Identification The greenhouse whitefly (family Aleyrodidae) adult (*25.27b*) is about 2 mm in length and white with four, white-appearing wings. Nymphs (*25.27d*) are translucent, flattened scales, measure 0.5 to 1.0 mm in length, and are usually found on leaf undersides. Pupae are wider and more opaque than the nymphs. The sweetpotato whitefly (*3.10d-g*), which has been found on ornamentals in some greenhouses in Canada (see Foreign diseases and pests, 3.10), is slightly smaller than the greenhouse whitefly, holds its wings more vertically alongside the body (*3.10e*), and has a light yellow thorax. Its nymphs are more variable in size and more irregular in outline than those of the greenhouse whitefly. The pupal stage is used to distinguish between these two whiteflies. The pupa (puparium) of the greenhouse whitefly, viewed laterally, is ovoid with straight sides, 12 large setae and no caudal groove, whereas the sweetpotato whitefly has an irregularly oval, oblique-sided pupa (puparium) with a variable number of short, fine setae and a caudal groove posteriorly *(3.10g).*

Life history Adults are found on the underside of leaves *(25.27a),* mostly near the growing tip of the plant where they feed and lay eggs. Eggs usually are laid individually on the underside of leaves *(25.27c),* but they may be clustered in a circular pattern. Adult longevity varies from 42 days at 18°C to eight days at 27°C. At normal greenhouse temperatures (21 to 24°C), females lay from 150 to 300 eggs. The nymphs also develop on the underside of leaves and feed by sucking plant sap. The first-instar nymph is a mobile, non-feeding crawler. It moves about the leaf for a few hours before becoming a sessile “scale.” After three molts, the scale forms a pupa. The adult emerges from the pupa through a T-shaped slit. The cycle from egg to adult is accomplished in 25 to 30 days at 21°C, or 22 to 25 days at 24°C. Optimal relative humidity for development is 75 to 80%.

The immature stages are often difficult to see. Consequently, they are often brought into greenhouses on seedlings and ornamentals. Adult whiteflies disperse in and between greenhouses by Hying. Because of their small size, they can be carried long distances on wind currents. The adult is attracted to yellow and can easily be spread on yellow clothing and yellow objects, such as buckets and picking containers. Adults and eggs are moderately cold tolerant and may survive the winter on hardy weeds in and around the greenhouse, becoming a source of infestation for the next cropping season.

Management Overlap of the new and old crop is the most frequent cause of early season greenhouse whitefly problems. Effective control measures combine cultural procedures and biological control, and chemical control only near the end of the growing season.

Monitoring — The standard method of monitoring for the greenhouse whitefly is a yellow sticky trap (*22.34d)* available from horticultural supply companies in a variety of shapes, sizes and shades of yellow, either as small, stiff, plastic cards (6 by 15 cm) or as sticky ribbons. Bright yellow is much more effective than orange-yellow. For effective early detection, there should be one trap per 100 plants. This density enables growers to detect the whitefly and time biocontrol releases well before serious infestations develop. The bottom of the trap should be level with the top of the plant canopy. Honeydew deposits and black sooty mold on plants help to indicate the location of developing infestations.

Yellow sticky ribbons (tapes) should be hung so that the middle of the ribbon is level with the top of the plant. The ribbons remove substantial numbers of whiteflies and are particularly useful in “hotspots.” In this situation, they should be used at a rate of one ribbon for every two to five plants. They are generally compatible with the principal biological control agent (see below). After two to three weeks when the whitefly population has been brought under control, the ribbons should be removed because parasites continue to be caught on them.

The action threshold for biological control of the greenhouse whitefly is the first appearance of its adult or immature stages. Growers should release parasites when the population level of whiteflies is low, otherwise biological control will not be successful. Traps also can be used to reduce whitefly populations in small greenhouse areas.

Cultural practices — Sanitation is one of the most important strategies for preventing whitefly infestations in greenhouses. Weeds should be removed and a deadband (3 to 6 m) maintained around the perimeter of the greenhouse. Vents can be screened to prevent the entry of whiteflies from outside the greenhouse, though this may cause ventilation problems unless overall intake area is increased. Non-crop plants should not be brought into the greenhouse, and movement of people and equipment from infested to non-infested areas should be restricted. All crop residue must be removed from the site at the end of the season and immediately destroyed by burying in a landfill to prevent reinfestation of the greenhouse.

Biological control — The principal agent for biological control of the greenhouse whitefly is the parasitic wasp *Encarsia formosa* Gahan (*25.27f*), which lays its eggs in the third and fourth instars of the immature whitefly. Direct feeding on first and second instars also occurs. The parasite larva develops inside the whitefly, eventually causing the whitefly pupa to turn black (*25.27e*). The parasite requires 25 to 35 days to complete development at 21 °C and 16 to 25 days at 24°C.

Introductions of the parasite are made as pupae and should begin as soon as adult or immature stages of the whitefly are detected. Parasites should be introduced weekly at a rate of one parasite pupa per four plants until 80% of whitefly pupae are black from being parasitized. In British Columbia, introductions of *E. formosa* are made routinely over the entire season, regardless of the presence of the whitefly. For optimum effectiveness, the daytime air temperature in the greenhouse should be around 24°C. Temperatures below 18°C severely inhibit the parasite’s searching behavior and development. Tomato plants could be pruned less vigorously and leaves that are pruned should be left in the greenhouse to allow time for the adult *E. formosa* to emerge from any parasitized whitefly pupae on the pruned leaves.

Every year, more growers are using *E. formosa* for control of whiteflies. In 1988, over 65% of the greenhouse tomato industry in Canada used *E. formosa* to control the greenhouse whitefly.

Chemical control — Insecticidal treatments may be used to control whitefly outbreaks, but most are effective only against the adult stage. Applications should be spaced no more than four to five days apart and four to five weeks will be required to control all whitefly stages. If whiteflies are present at the end of the season, the crop residue should be fumigated before removal.

No information is available on treatment thresholds but, as a rule, insecticides should be applied no later than the first appearance of honeydew on the leaves or fruit. Heavy treatment schedules have resulted in greenhouse whitefly populations resistant to all registered insecticides in many regions of Canada.

Most insecticides are harmful to the parasitic wasp *E. formosa.* Synthetic pyrethroids may have a negative, residual effect for 50 days from the time of treatment. Attempts to reduce high whitefly populations with insecticides before introducing *E. formosa* are usually ineffective, making early detection and parasite releases essential.

Selected references

Gerling, D. 1990. Whiteflies: Their Bionomics, Pest Status and Management. Intercept Ltd., Hants. 348 pp.
Gillespie, D.R., and D.M.J. Quiring. 1987. Yellow sticky traps for detecting and monitoring greenhouse whitefly (Homoptera: Aleyrodidae) adults on greenhouse tomato crops. J. Econ. Entomol. 80:675-679.

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

▶ 25.28 Leafminers *Figs. 25.28a-e*

Chrysanthemum leafminer *Liriomyza trifolii* (Burgess)
Vegetable leafminer *Liriomyza sativae* Blanchard

Leafminers occur only sporadically on greenhouse tomato crops. The chrysanthemum leafminer usually is seen only where tomato is grown in close proximity to greenhouse flower crops, particularly chrysanthemum and gerbera. Once in a greenhouse, leafminers should be controlled because populations can increase rapidly and cause substantial losses.

Damage Tomato fruit is not directly affected but damage to leaves (*25.28b,c*) may be extensive. This has the same effect as defoliation, causing reduced yields.

Identification Adults of both flies (family Agromyzidae) *(25.28a,d)* are small, yellow and black (about the same size as a vinegar fly, *18.42g).*

Life history Eggs are laid in leaf tissue (*25.28a*) and hatch into larvae that tunnel between the upper and lower leaf surfaces. The larvae complete development in four to seven days at summer temperatures and drop to the soil to pupate. Adults emerge 5 to 10 days after the larvae have dropped.

Management Leafminers in tomato are most effectively controlled biologically. A strict sanitation program also is helpful in preventing infestations.

Monitoring — Yellow sticky traps (*22.34d*) are excellent for monitoring leafminer flies. They can be used to detect first invasions and to monitor the success of control measures.

Biological control — Parasitic wasps (*25.28e) Diglyphus isaea* (Walker) and *Dacnusa sibirica* Telenga are available commercially and should be used if biological control programs are in progress for other pests.

Chemical control — No effective pesticides are registered for use against leafminers on greenhouse tomato.

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

▶ 25.29 Thrips *Figs. 25.29; 22.34e-g*

Onion thrips *Thrips tabaci* Lindeman
Western flower thrips *Frankliniella occidentalis* (Pergande)

Onion thrips (see Onion, 13.27) and western flower thrips (see Greenhouse cucumber, 22.34) may infest greenhouse tomato crops. The life history is similar for both species. Adult females insert their eggs into leaves, tender plant tissues and sometimes even fruit. The nymphs (or larvae) hatch and feed on the leaves for 5 to 10 days, depending on temperature, then drop to the soil and undergo pupation for about five days before emerging as adults.

Western flower thrips *(25.29, 22.34e-g)* is particularly important because it may be a vector of tomato spotted wilt virus. Onion thrips is rarely a problem on greenhouse tomato and it has not been reported to transmit tomato spotted wilt virus in Canada.

Damage Some strains of western flower thrips have adjusted to feeding on tomato and can cause severe leaf damage. Symptoms of feeding by adults and nymphs of the western flower thrips and the onion thrips are identical (see Greenhouse cucumber, 22.34). In general, thrips confine themselves to the lower leaves on greenhouse tomato and rarely affect new growth or leaves that are actively photo- synthesizing.

Management If tomato spotted wilt is present in the greenhouse, or nearby, every effort should be made to control the virus and its principal vector, the western flower thrips.

Cultural control — Greenhouse vents and doorways can be screened to prevent an influx of thrips. Empty greenhouses can be heated to 35°C for five days or 40°C for two to three days to hasten thrips pupal development; any emerging adults will starve. Soil also can be steam pasteurized to kill pupae and adults.

Chemical control — Thrips may be controlled by using procedures described for western flower thrips (see Greenhouse cucumber, 22.34). Onion thrips alone can be controlled by applying insecticides to the floor of the greenhouse to kill the pupal stage.

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

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

Aphids
Caterpillars

Aphids, such as the green peach aphid and the potato aphid, sometimes colonize the lower stems of greenhouse tomato plants. The life histories of these and other aphids are identical in all respects on other crops where they occur (see Potato, 16.40-16.43). Damage is usually inconsequential unless the stem is colonized near developing fruit. When that happens, honeydew must be washed off the fruit and chemical control should be implemented. Biological control of aphids (see Greenhouse pepper, green peach aphid, 24.12) may help when aphid numbers are low.

Caterpillars of various species of moths, such as the cabbage looper (see Crucifers, 8.40), corn earworm (see Maize, 12.13), also known as the tomato fruitworm, and European corn borer (see Maize, 12.16) are occasional pests on greenhouse tomato. Their development is accelerated in the greenhouse and several generations may occur compared with only one or two generations per year in the field. No specific parasites or predators are available for release in greenhouses, but a non-specific egg parasite *(Trichogramma* sp.) might be tried if available. Where biological control programs are present, growers should use a microbial insecticide containing a formulation of *Bacillus thuringiensis* Berliner.

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

MITE PESTS

▶ 25.31 Tomato russet mite *Fig. 25.31*

Aculops lycopersici (Massee)

The tomato russet mite is found in areas of the United States bordering southern Canada. It is reported to overwinter in unheated greenhouses and on weeds in the United States but its ability to survive the winter in Canada is unknown. Outbreaks have been reported on greenhouse tomato in British Columbia, Ontario and Quebec.

Hosts are, in general, plants in the Solanaceae. Nightshade (*Solanum* spp.) and petunia are mentioned as frequent sources of infestation, but the tomato russet mite may occur on other solanaceous weeds and crops.

Damage The tomato russet mite can cause severe losses, although only a few cases have been noted in greenhouses in Canada. Leaf symptoms *(25.31)* include yellowing, curling and wilting. Flowers may abort and fruit may be deformed. If not controlled, the tomato russet mite will eventually kill tomato plants.

Identification The tomato russet mite (family Eriophyidae) adult is about 0.2 mm in length and 0.05 mm in width. Because of its small size, the mite is not noticed on plants until it reaches damaging levels. Aggregations of the mite on stems, leaves and fruit have a beige or bronze appearance.

Life history Tomato russet mite females lay from 10 to 50 eggs during a lifespan of 20 to 40 days. High rate of reproduction and rapid development are favored by moderate temperature (21°C) and low humidity (30% RH). Under these conditions the life cycle can be completed in six to seven days.

Adult tomato russet mites disperse by wind and may move from the United States into Canada with weather systems. Importations on tomato plants from the southern United States have been suspected but never proven.

Management

Cultural practices — Plants showing wilting or poor growth should be examined for tomato russet mites. Greenhouses should be thoroughly cleaned between crop cycles and old plants, weeds, crop residue and debris should be removed to prevent carry-over from one crop to the next. If an infestation has occurred, the greenhouse structure should be washed and the soil steam-sterilized if possible. A relative humidity of 70 to 80% will help to slow the development of damaging population levels.

Biological control — Various predators will feed on the tomato russet mite, for example, the mites *Amblyseius* (syn. *Neoseiulus*) *cucumeris* Oudemans (*22.34h*) and *Metaseiulus* (syn. *Typhlodromus) occidentalis* (Nesbitt), and the minute pirate bug *Orius tristicolor* (White) *(22.34i)*. These are available commercially but have not been used successfully against the tomato russet mite.

Chemical control — Sulfur, formulated as a liquid or dust, or with other materials in such products as lime-sulfur and nicotine sulfate, provides control and can be integrated with biological control.

Selected references

Perring, T.M., and C.A. Farrar. 1986. Historical perspective and current World status of the tomato russet mite (Acari: Eriophyidae). *Am. Entomol. Soc. Mise. Publ.* 63. 19 pp.

(Original by D.R. Gillespie)

▶ 25.32 Two-spotted spider mite *Figs. 25.32; 22.36a-g*

Tetranychus urticae Koch

This mite (see Greenhouse cucumber, 22.36) is becoming a more common problem on greenhouse tomato in many areas of Canada, particularly in British Columbia. Its biology is similar to that described on greenhouse cucumber but outbreaks tend to spread less rapidly on greenhouse tomato, which is probably related to the presence of sticky glandular hairs (trichomes) on tomato petioles and stems.

Management The two-spotted spider mite *(25.32, 22.36e,f*) can cause severe damage to greenhouse tomato and infestations should be treated promptly. Control may involve applications of miticides, or a program of biological control using sustained releases of a predatory mite.

Biological control — The mite *Phytoseiulus persimilis* Athias-Henriot *(25.32, 22.36g*) is an effective predator of the two-spotted spider mite but it is severely inhibited by the sticky hairs on tomato, which makes its distribution on tomato plants uneven. For this reason, greater numbers of the predator should be used on tomato than on greenhouse cucumber and they should be applied to every leaf that is infested. Growers should consult a biological control supplier for specific rates and timing of introductions.

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

ADDITIONAL REFERENCES

Atherton, J.G., and J. Rudich, eds. 1986. *The Tomato Crop.* Chapman and Hall, London. 661 pp.

Beckman, C.H. 1987. *The Nature of Wilt Diseases of Plants.* APS Press, St. Paul, Minnesota. 175 pp.

Blancard, D. 1992. *A Colour Atlas of Tomato Diseases.* Wolfe Publishing Ltd., London. 212 pp.

Fletcher, J.T. 1984. *Diseases of Greenhouse Plants.* Longman Group Ltd., New York. 351 pp.

Griffen, M.J., and M.J. Savage. 1983. *Control of Pests and Diseases of Protected Crops. Tomatoes.* Ministry of Agriculture, Fisheries and Food (MAFF Publications) Booklet 2243. Alnwick, Northumberland, U.K. 110 pp.

Hussey, N.W., and N.E.A. Scopes, eds. 1985. *Biological Pest Control — The Glasshouse Experience.* Cornell Univ. Press, Ithaca, New York. 240 pp.

Jarvis, W.R. 1992. *Managing Diseases in Greenhouse Crops.* APS Press, St. Paul, Minnesota. 280 pp.

Jarvis, W.R., and C.D. McKeen. 1991. *Tomato Diseases.* Agric. Canada Publ. No. 1479/E. 70 pp.

Jones, J.B., J.P. Jones, R.E. Stall and T.A. Zitter, eds. 1991. *Compendium of Tomato Diseases.* APS Press, St. Paul, Minnesota. 73 pp.

Jones, J.P., and S.S. Woltz. 1981. *Fusarium*-incited diseases of tomato and potato and their control. Pages 157-168 *in* P.E. Nelson, T.A. Toussoun and R.J. Cook, eds., *Fusarium: Diseases, Biology and Taxonomy.* The Pennsylvania State Univ. Press, University Park, Pennsylvania. 457 pp.

Shipp, J.L., G.J. Boland and L.A. Shaw. 1991. Integrated pest management of disease and arthropod pests of greenhouse vegetable crops in Ontario: current status and future possibilities. *Can. J. Plant Sci.* 71:887-914.

Steiner, M.Y., and D.P. Elliott. 1987. *Biological Pest Management for Interior Plantscapes.* Alberta Environmental Centre, Vegreville, Alberta. 30 pp.

Sutton, A., ed. 1991. *Tomatoes: Field and Protected Crops.* Ciba-Geigy, Basel, Switzerland. 64 pp.

Tjamos, E.C., and C.H. Beckman, eds. 1989. *Vascular Wilt Diseases of Plants. Basic Studies and Control.* Springer-Verlag, Heidelberg. 590 pp.