5 Beet, chard, spinach

BACTERIAL DISEASES

▶ 5.1 Scab *Fig. 5.1*

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

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

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

FUNGAL DISEASES

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

Aphanomyces cochlioides Drechs.

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

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

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

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

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

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

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

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

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

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

Management

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

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

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

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(Original by R.F. Cerkauskas)

▶ 5.3 Cercospora leaf spot *Fig. 5.3*

Cercospora beticola Sacc.

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

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

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

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

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

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

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

Management

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

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

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

Selected references

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(Original by R.F. Cerkauskas)

▶ 5.4 Downy mildew *Figs. 5.4a,b*

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

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

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

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

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

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

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

Management

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

Resistant cultivars — Resistant cultivars are available from seed suppliers.

Chemical control — Registered fungicides are available for foliar application.

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(Original by R.F. Cerkauskas)

▶ 5.5 Fusarium wilt *Figs. 5.5a-d*

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

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

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

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

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

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

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

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

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

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

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

Management

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

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(Original by A.A. Reyes)

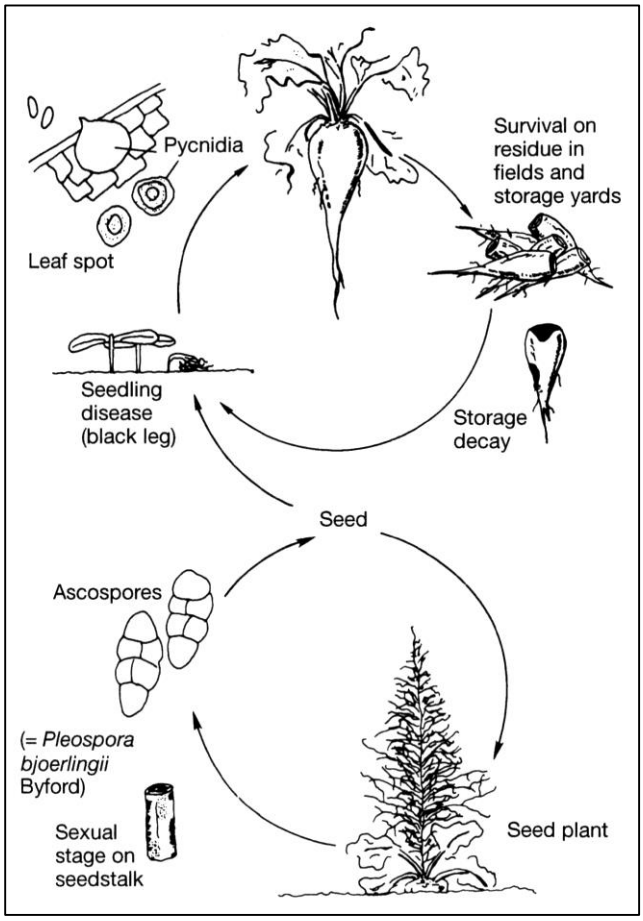
▶ 5.6 Phoma leaf spot and root rot *Fig. 5.6T1*

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

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

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

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



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

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

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

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

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

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

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

Management

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

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

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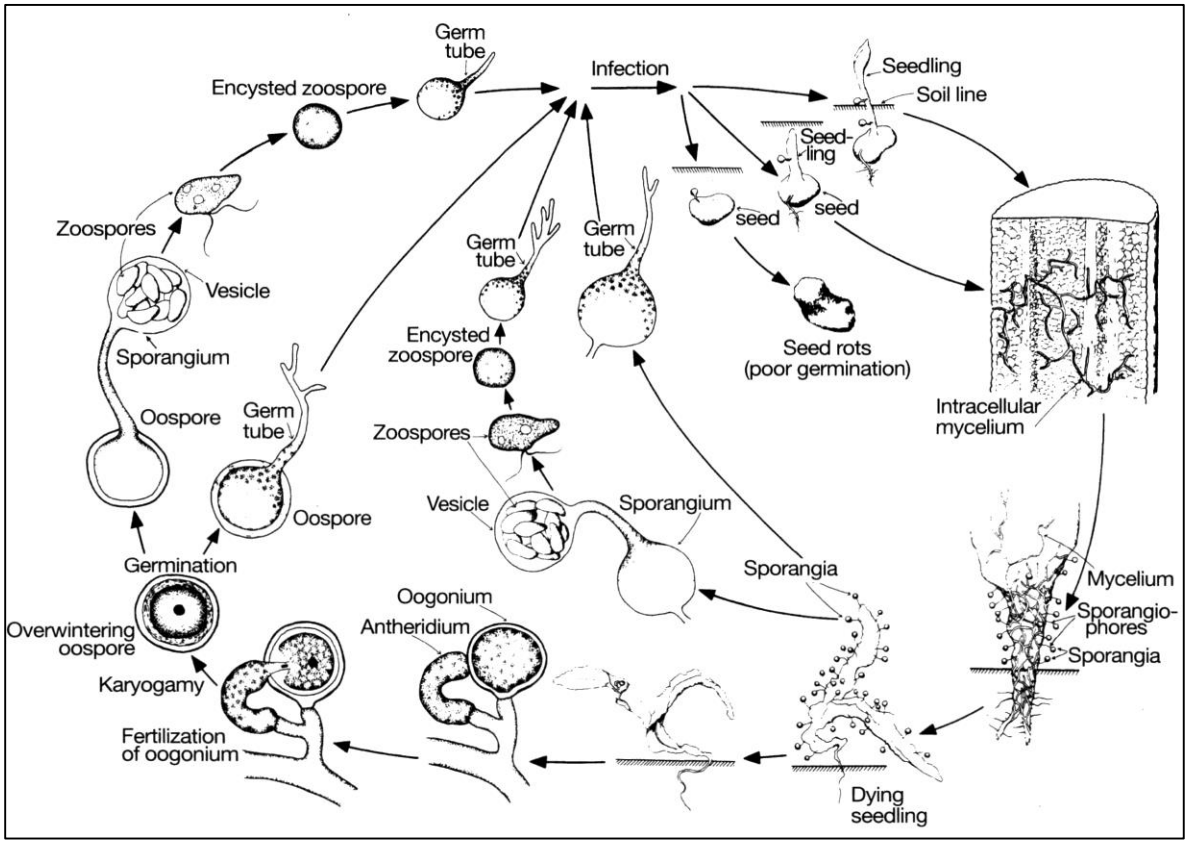
(Original by R.F. Cerkauskas)

▶ 5.7 Pythium root rot *Fig. 5.7T1*

Pythium aphanidermatum (Edson) Fitzp.
Pythium ultimum Trow



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

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

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

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

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

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

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

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

Management

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

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

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(Original by R.F. Cerkauskas)

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

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

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

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

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

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

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

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

Management

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

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

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

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(Original by R.F. Cerkauskas)

▶ 5.9 White rust *Fig. 5.9*

Albugo occidentalis G.W. Wils.

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

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

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

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

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

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

Management

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

Selected references

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(Original by R.F. Cerkauskas)

VIRAL DISEASES

▶ 5.10 Spinach blight *Fig. 5.10*

Cucumber mosaic virus

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

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

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

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

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

Management

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

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

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(Original by L.W. Stobbs)

NON-INFECTIOUS DISEASES

▶ 5.11 Heart rot (boron deficiency)

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

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

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

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

Management

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

Selected references

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(Original by R.F. Cerkauskas)

NEMATODE PESTS

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

Meloidogyne hapla Chitwood

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

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

Pratylenchus penetrans (Cobb) Filip. & Stek.

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

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

Heterodera schachtii Schmidt

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

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

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

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

Management

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

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

INSECT PESTS

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

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

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

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

Selected references

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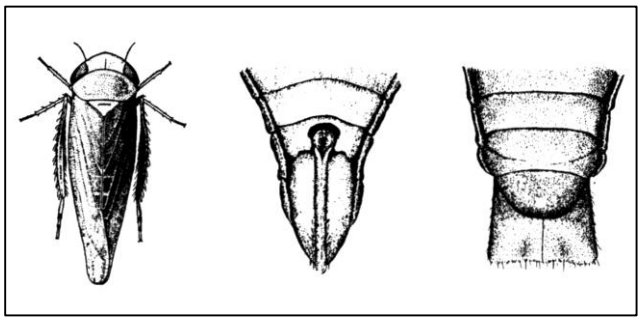
(Original by K.G.A. Hamilton)



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

▶ 5.16 Flea beetles *Fig. 5.16*

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

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

(Original by C. Ritchot)

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

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

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

White grubs *Phyllophaga* spp.

Aphids

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

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

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

Beet webworm

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

Leafminers

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

White grubs

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

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

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