# 4 Asparagus



## FUNGAL DISEASES

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

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

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

**Management**

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

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

(Original by F.J. Louws)

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

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

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

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

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

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

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

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

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

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

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

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

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

**Management**

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

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

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

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Nelson, P.E., T.A. Toussoun and W.F.O. Marasas. 1983. *Fusarium Species - An Illustrated Manual for Identification.* The Pennsylvania State University Press, University Park, Pennsylvania. 193 pp.

(Original by F.J. Louws)

## ▶ 4.3 Phomopsis blight (stem blight)

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

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

**Management**

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

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

**Selected references**

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(Original by F.J. Louws)

## ▶ 4.4 Phytophthora spear rot

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

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

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

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

**Management**

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

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

**Selected references**

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(Original by F.J. Louws)

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

*Stemphylium vesicarium* (Wahr.) E. Simmons

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

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

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

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

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

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

**Management**

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

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

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(Original by F.J. Louws)

## ▶ 4.6 Rust *Figs. 4.6a,b; 4.6T1*

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

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

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

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

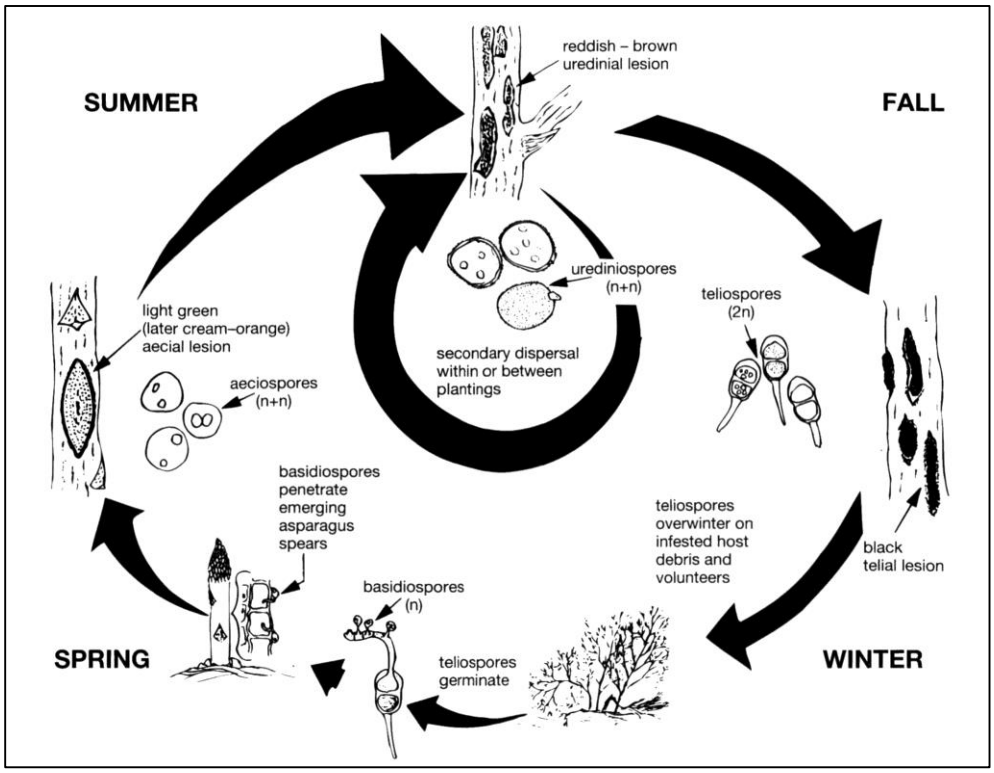
During the summer months, as early as June or as late as September, reddish-brown, blister-like pustules (uredinia) appear on the asparagus shoots *(4.6b).* When the uredinia mature, they release rust-colored urediniospores that cause new infections throughout the summer. Late in the season, the production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

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

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

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

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




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

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

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

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

**Management**

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

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

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

**Selected references**

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(Original by F.J. Louws)

# VIRAL DISEASES

## ▶ 4.7 Miscellaneous viral diseases

Asparagus virus I
Asparagus virus II
Tobacco streak virus

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

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

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

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

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

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

**Management**

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

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(Original by F.J. Louws)

# NON-INFECTIOUS DISEASES

## ▶ 4.8 Miscellaneous disorders *Fig. 4.8*

Autotoxicity
Cold injury

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

(Original by F.J. Louws)

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

(Original by P.R. Ragan)

# INSECT PESTS

## ▶ 4.9 Asparagus aphid *Fig. 4.9*

*Brachycorynella asparagi* (Mordvilko)

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

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

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

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

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

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

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

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

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

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

**Selected references**

Stoetzel, M.B. 1990. Aphids (Homoptera: Aphididae) colonizing leaves of asparagus in the United States. *J. Econ. Entomol*. 83:1994-2002.

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

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

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

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

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

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

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

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

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

**Management**

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

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

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

(Original by J.A. Garland)

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

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

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

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

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

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

## ADDITIONAL REFERENCES


Conway, K.E., J.E. Motes and C.J. Foor. 1990. Comparison of chemical and cultural controls for cercospora blight on asparagus and correlations between disease levels and yield. *Phytopathology* 80:1103-1108.

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


## **Bean** see **15 Pea and bean**