MAJOR DISEASES OF ASPARAGUS IN NEW ZEALAND

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ABSTRACT

The main fungal diseases of asparagus in New Zealand are the soil borne diseases Fusarium root and crown rot caused mainly by *Fusarium oxysporum f. sp. asparagi* and *F. moniliforme,* although *F. solani* is isolated occasionally; Fusarium spear spot caused by *F. redolens* and Phytophthora rot caused by *Phytophthora megasperma* var. *sojae.* A foliar disease of asparagus, Stemphylium leaf spot and purple spot caused by *Stemphylium vesicarium*, is mainly restricted to North Island production areas. Asparagus virus II has been isolated throughout New Zealand. Information on symptoms, life cycle, and control is discussed.

Little data exist on the distribution and effect of most asparagus diseases in New Zealand. However, three recent local trials have shown that Stemphylium leaf spot may reduce yield by 85% and Phytophthora rot by 31%. None of the asparagus cultivars presently grown in New Zealand are resistant to any of these diseases although some cultivars e.g. Jersey Giant may have tolerance to Stemphylium leaf spot and Fusarium crown rot.

A reduction in the profitable life of asparagus fields in New Zealand most likely occurs because of an interaction between the various diseases, adverse management and environmental factors.

Additional Key Words: Asparagus decline, Stemphylium leaf spot and purple spot, Phytophthora rot, Fusarium rot, asparagus virus II.

INTRODUCTION

Asparagus officinalis L. is a dioecious, perennial species. The average life of asparagus fields in New Zealand is approximately 12 years (Robb, 1984) although some last considerably longer than this e.g. 55 years (Falloon, 1982a). Over this life span crop production is likely to be influenced by numerous interacting environmental factors e.g. rainfall, soil moisture, temperature, wind, weed competition, pests and diseases.

The 3 156 ha of asparagus currently grown in New Zealand (Douglas, 1986) may be adversely affected by a number of important diseases. These are either foliar fungal diseases (Stemphylium leaf spot, purple spot), soil-borne fungal diseases (Fusarium rot, Phytophthora rot) or viral diseases (asparagus virus II).

There is no adequate level of resistance to these diseases amongst asparagus cultivars currently grown in New Zealand, so a number of different management strategies must be used to minimise the effect of each disease. Recent work e.g. Evans and Stephens (1984; 1986) has shown that some of these diseases may interact and effect production. Hence control of any one disease may reduce the effect of one or more of the other diseases. This has important implications for the control of "asparagus decline" which reduces the profitable life of asparagus fields (Grogan and Kimble, 1959).

FOLIAR DISEASES

Stemphylium leaf spot and purple spot

This disease caused by the fungus *Stemphylium vesicarium* Simmons (anamorph of *Pleospora allii* (Rabenh.) Ces & de Not.) mainly affects production in the North Island where warm, humid conditions prevail. However it has occasionally been identified as far south as South Canterbury in the South Island.

Symptoms

Stemphylium leaf spot produces large elliptical lesions (2 to 6 mm by 6 to 13 mm at maturity) with well defined red-brown to black margins surrounded by a diffuse yellow-green zone and light brown to grey centre on stems and branches (Suzui, 1973; Falloon *et al.,* 1984). Lesions on stems may coalesce, resulting in extensive areas of infected tissue. On cladophylls, lesions first appear as minute, circular, yellowish-tan coloured spots which gradually darken and elongate. The centre of the spots becomes necrotic, greyish or greyish-brown and slightly sunken. When mature the lesions have a reddish-brown margin which is surrounded by a diffuse light yellow zone. Cladophyll infection results in premature defoliation of summer fern growth. Fern branches then start to die back until the whole fern is dead.

Ascospores of *P. allii* infect spears (Falloon *et al.,* 1986c) causing lesions which appear elliptical (0.8 to 1.6 mm across and up to 1 mm long), slightly sunken with a purple margin (hence "purple spot") and brown centre, especially in larger lesions. The internal tissue of the spear is not affected (Lacy, 1982; Falloon *et al.,* 1984).

Infected spears are frequently found close to debris remaining from the previous summer's fern on which the fungal fruiting bodies have formed (Falloon *et al.,* 1984).

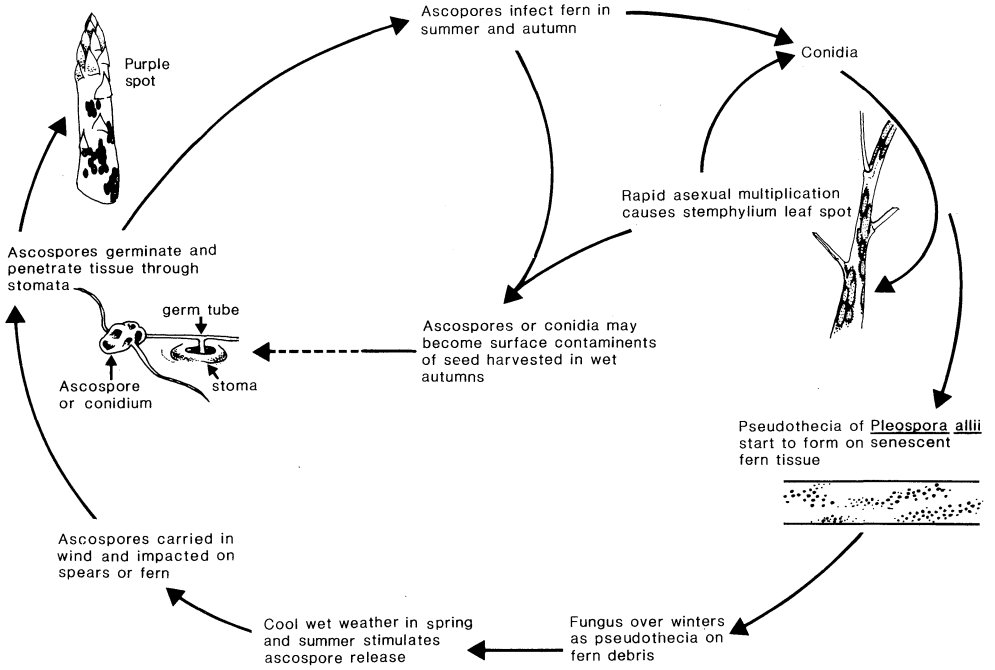

Figure 1: Disease cycle of Stemphylium leaf spot and purple spot of asparagus.

These are small (0.20 to 0.55 mm diam.) black, carbonaceous, globose or depressed globose, erumpent pseudothecia (Falloon *et al.,* 1984). Occasionally, two or more pseudothecia coalesce to form one larger irregular shaped body. Hand sections from pseudothecia reveal bitunicate asci containing eight muriform ascospores which appear yellow-brown when young or golden-brown to dark-brown when mature.

Disease Cycle

Infection of the spears results when ascospores of *P. allii* are released from pseudothecia on dead stalks from the previous summer's fern (Falloon *et al.,* 1986c) (Fig. 1) or conidia are released from fern infections of young seedlings, either volunteers or in nursery blocks. Ascospores are also the most likely source of primary inoculum for foliar infections. Ascospores are forcibly ejected when the pseudothecia are moistened either by rain, dew or irrigation (Falloon *et al.,* 1986c). They may be carried by wind and impacted on the windward side of spears. Hence the symptoms of purple spot may be more severe on one side of the spear than the other (Lacy, 1982; Falloon *et al.,* 1986c).

Spores germinate in conditions of prolonged, relative humidity (95-100% for at least 24 hrs) (Menzies *et al.,* 1986) and penetrate the spears or fern through stomata (Falloon *et al.,* 1986c) although physical damage to the surface of asparagus spears or fern caused by wind-blown sand or soil particles may provide alternative points of entry (Lacy, 1982; Johnson and Lunden, 1986; Falloon *et al.,* 1986c).

It is unusual for conidia to be produced around lesions on spears unless the spear is in an advanced stage of decay. However, conidia are often observed on cladophylls that have dropped off summer fern (Sing, 1977). This is the main source of secondary inoculum for fern infections during the summer and autumn.

As the fern starts to die off in late autumn, black pseudothecia of *P. allii* are frequently observed starting to develop below the epidermis of the senescing fern stalks.

Spores of *Stemphylium* and *Pleospora* have been reported as surface contaminants of asparagus seed (Groves and Skolko, 1944) especially when the seed is harvested in wet autumns (Falloon, 1985). Seed-borne spores of *Stemphylium* and *Pleospora* are therefore a possible method of spread of the pathogen over long distances and could be a source of disease in glasshouse-raised seedling transplants of asparagus.

Effect on Yield

Stemphylium vesicarium causes defoliation, dieback and death of above-ground parts but does not infect the crown or root system (Bansal *et al.,* 1986). Comparison between plots where Stemphylium leaf spot was controlled with regular fungicide treatments applied during summer and those that received no treatment has shown that yield may be reduced the following spring by up to 52% (Menzies, 1983). In a mature field the major effect of Stemphylium leaf spot is from the immediate past season.

Hence control measures started at any year of growth of a mature field should result in yields similar to those if a full spray programme had been used for the life of the stand.

Control of Stemphylium leaf spot in young crops has resulted in an 85% increase in yield of spears in the first harvest (Douglas, 1986). A survey of 14 asparagus growers in the Waikato showed that yield was highly correlated (R^2 = 65%) with control of Stemphylium leaf spot (Douglas, 1986).

Although Purple spot does not affect the internal tissue of asparagus spears (Lacy, 1982; Falloon *et al.*, 1984) severe epidemics may result in rejection rates of 60%-90% of spears for fresh export. Such epidemics, which are associated with periods of spring rainfall (Falloon *et al.*, 1986c) may occur between three and six times each harvest season in Auckland, Waikato and Bay of Plenty but only once every two or three years in areas with drier conditions during spring e.g. Hawkes Bay , Canterbury, Nelson and Marlborough. Seasonal yield losses between five and ten percent of spears for fresh export have been recorded in the Waikato (S.A. Menzies pers comm.).

Control

Almost complete control of Stemphylium leaf spot is possible with regular applications (two weekly) of the fungicide captafol at 1.24 *l* a.i. (Difolatan 5F at 2.5 *l* product) in 200-400 *l* water per ha applied as soon as first symptoms of the disease appear. The optimum frequency of spray application will vary according to the severity of the epidemic; the less severe the epidemic the fewer the number of sprays required. In Europe other fungicides have also given good control of Stemphylium leaf spot e.g. maneb, mancozeb, chlorothalonil and iprodione (Gindrat *et al.*, 1984) and propiconazole has proved effective in New Zealand. (Douglas, 1986).

The most effective way to control spear infections in the spring and to delay the onset of the disease in summer is to eliminate the source of inoculum. Burning dead fern stalks during the winter months effectively controls *P. allii.* Tractor-drawn flame burners travelling at 1.2 km/hr burn most of the fern debris and prevent discharge of ascospores. Alternatively, fine chopping of fern stalks followed by several winter cultivations to bury the trash for minimum periods of two weeks also reduces inoculum (Menzies *et al.*, 1985).

No asparagus cultivars are completely resistant to *Stemphylium* although a number e.g. Jersey Giant and several advanced breeding lines from Crop Research Division are more tolerant than French cultivars e.g. Cito, Minerve, Desto, Brunetto. Whether this is the result of differences in susceptibility of the tissue to the disease or differences in microclimate of the fern is unknown. The French cultivars generally have more compact fern growth than Jersey Giant, and other tolerant cultivars.

Effective weed control and wider spacing between rows (up to 1.8 m) may also reduce humidity within the crop with a resultant reduction in the severity of the disease.

A number of asparagus species e.g. *A. asparagoides, A. densiflorus* cv. Sprengeri, *A. virgatus, A. larcinus, A. verticillatus, A. compactus* and *A. densiflorus* cv. Myers are highly resistant to Stemphylium leaf spot (Bansal *et al.*, 1986).

SOIL-BORNE DISEASES

Fusarium diseases

Fusarium species pathogenic to asparagus have been isolated from asparagus fields throughout New Zealand (Ellison, 1980) (Fig. 2). Four species are involved; *F. oxysporum* (Schlecht.) emend. Snyd. & Hans f. sp. *asparagi* Cohen, *F. moniliforme* (Sheld.) emend. Snyd. & Hans., *F. solani* (Mart.) App. & Wr. emend Snyd. & Hans and *F. redolens* (Wollenw.).

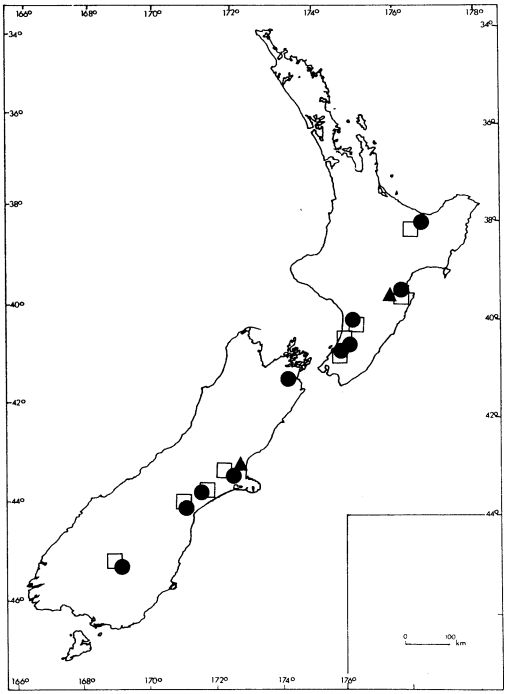

Figure 2: Distribution of *Fusarium* species pathogenic on asparagus in New Zealand. ● = *F. oxysporum* f.sp. *asparagi*, □ = *F. moniliforme*, ▲ = *F. solani.*

Disease cycle of *Fusarium* - Fig. 3

Conidia of *F. oxysporum* f. sp *asparagi* and *F. moniliforme* are seed-borne (Inglis, 1980), the seed becoming contaminated during harvesting and extraction of the seed from the berries. Germinating seedlings become infected by these spores in the nursery.

Fusarium is a soil inhabitant and is able to survive saprophytically on dead organic matter, e.g. asparagus crowns and roots or on other hosts, e.g. corn (Nyvall & Kommedahl 1968, 1970), annual weeds, for many years. In

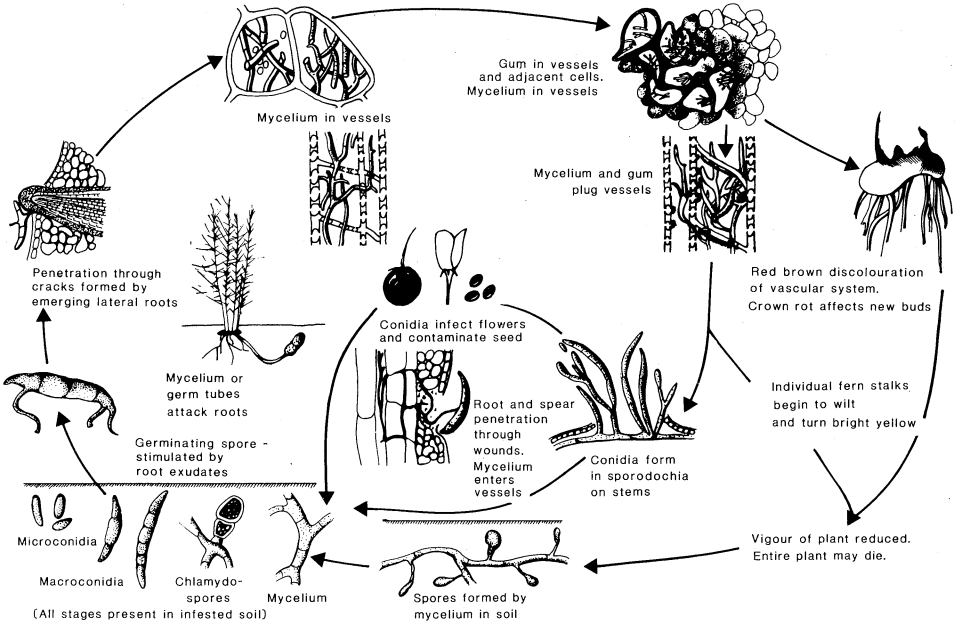

Figure 3: Disease cycle of Fusarium rot of asparagus.

addition *F. oxysporum* f.sp. *asparagi* produces chlamydospores which are capable of surviving for many years in soil. However *F. oxysporum* f. sp. *asparagi* spreads only slowly within a field (Grogan and Kimble, 1959). On the other hand, on infected stems *F. moniliforme* produces airborne spore so it can rapidly disseminate in the field. *F. moniliforme* can also survive on maize stubble and result in severe disease in subsequent asparagus plantings. (Damicone and Manning, 1980).

Spores of pathogenic *Fusarium* spp. can be transported within or between fields in soil which is wind-blown or carried on machinery or in surface water (Hung, 1974). Both *F. oxysporum* f.sp. *asparagi* and *F. moniliforme* can be transmitted by insects (Gilbertson and Manning, 1983).

The optimum soil temperature for development of the disease is 25 °C (Tu, 1986) but disease may occur at temperatures lower than 20 °C. Moisture stress either from waterlogging or drought may favour development of the disease.

Although seedlings can be infected from contaminated seed or soil and produce infected one year old crowns, the most severe disease usually results when old asparagus fields which have a high level of spores in the soil are resown or replanted. As tissues become infected and die back *Fusarium* colonises them and produces spores which ensure dissemination, survival and new infections. The soil above and below the crowns becomes infested with spores (Grogan and Kimble, 1959) which infect new rootlets and spears. Cut spears and crowns damaged by cultivation equipment, wrenching of fern stalks from crown sockets, and other injuries, form easy avenues of infection and rapid colonisation. Weakened asparagus crowns are more susceptible to *Fusarium* than vigorously growing plants.

FUSARIUM CROWN AND ROOT ROT

This disease is caused by *F. oxysporum* f. sp. *asparagi* and in New Zealand usually attacks cortical tissues, although vascular bundles can also be infected. *F. moniliforme* is also involved to a lesser degree, but tends to be more virulent than *F. oxysporum* f.sp *asparagi* in artificial inoculations, growing more rapidly in vascular tissue of stems and crowns.

Symptoms

Seed-borne conidia of *Fusarium* spp. often lodge in deep crevices in the seed coat (Inglis, 1980; Damicone *et al.*, 1981) and may infect young seedlings, causing rot in the developing crown starting at the point of seed attachment. The root system may be stunted with a brown rot and dieback of the feeder roots. Elliptical or "running" longitudinal brown lesions often develop on young storage roots and longitudinal red-brown flecks may appear on stem bases below soil level. The foliage may be stunted, wilted and yellow and needle drop may occur in advanced cases. Such infections sometimes result in death of seedlings and are thought to be the main reason for difficulties that growers have in re-establishing asparagus in soil previously planted with asparagus. However, autoallelopathic compounds may also cause replant failures (Laufer and

Garrison, 1977; Yang, 1982, 1986; Hartung and Stephens, 1983; Hartung and Putnam, 1986).

On mature plants *F. oxysporum* f. sp. *asparagi* may cause rot of fine feeder roots resulting in a reddish-purple discolouration. Most of the feeder roots are often rotted off completely. Sometimes the reddish streaking extends from feeder roots onto the surface of storage roots. Storage roots often show a reddish brown discolouration of the vascular bundle which may extend into the crown. Fern stalks may also have reddish-brown vascular discolouration which often extends many centimeters above the soil surface and in such cases the fern will be yellow and stunted. The stem bases below ground often show rusty coloured flecks and sunken lesions. Stalk rot may also extend into the crown where it causes a dry crown rot (Grogan and Kimble, 1959). In crowns, *F. moniliforme* may produce an aggressive, penetrating, rust coloured vascular infection while *F. oxysporum* f.sp *asparagi* is isolated more frequently from crown tissue immediately below the stem sockets.

Control

Seed should be disinfested prior to sowing. This can be achieved using a seed dust of 5 g thiram and 2.5 g benomyl per kg seed. Higher rates of benomyl may be toxic causing reduced germination and stunting of seedlings (Damicone *et al.*, 1981). Alternatively seeds may be soaked for 24 h in an agitated mixture of 25 g/*l* benomyl in acetone followed by thorough rinsing in water (Damicone *et al.*, 1981). A third method is to soak seed for 10 minutes in a 0.5 to 1% solution of sodium hypochlorite before sowing. It is doubtful that any of these treatments will be completely effective and the effects of seed treatment with non-systemic fungicides on infection of seedlings grown in *Fusarium* infested soil is probably short-lived due to growth of roots away from the fungicides and breakdown of the fungicide in the soil. However all treatments may substantially reduce the level of seed-borne infections and consequently reduce the proportion of *Fusarium*-infected plants that are transplanted to production fields.

When crown transplants are used a fungicide dip in a solution of 2 g/*l* Captan and 0.25 g/*l* benomyl will prevent development of storage rots and may improve crop establishment (R.E. Lill pers. comm.). Care should be taken to ensure that surface moisture on roots and crowns is removed after dipping and prior to cool storage.

Thiabendazole (acid formulation) has proven effective for controlling *Fusarium* in asparagus fields in England (D.R. Ellerton, pers. comm.). Seed treatments, crown dips prior to transplanting and soil drenches on established crops have all resulted in improved establishment and higher yields.

Inoculation of asparagus seedling transplants with an avirulent strain of *F. oxysporum* was shown to protect plants against crown rot development in the field for up to 8 weeks (Damicone & Manning, 1982). The use of similar cross protection techniques may be useful if adequate resistance cannot be developed in *A. officinalis*.

Despite many years of breeding for resistance to *F. oxysporum* f. sp. *asparagi* and *F. moniliforme* (Ellison, 1986) no resistant asparagus cultivars are yet available. Field screening in New Zealand of 75 cultivars and advanced breeding lines, 45 of which were the result of crosses between plants selected for resistance to *F. oxysporum* f. sp. *asparagi* and *F. moniliforme*, showed that progeny from parents selected for *Fusarium* resistance were amongst the most susceptible lines tested (Bussell & Ellison, 1986). The greater tolerance of crown rot associated with several parents that had not been selected for *Fusarium* resistance was probably due to increased vigour rather than any genetic tolerance to *Fusarium* infection. The all-male hybrid Jersey Giant was found to be amongst the least susceptable lines tested. Some other *Asparagus* spp. are immune to *F. oxysporum* f. sp. *asparagi* e.g. *A. densiflorus* cv. Sprengeri (Lewis and Shoemaker, 1964). This species is also highly resistant to *Stemphylium vesicarium* (Bansal *et al.*, 1986) and immune to *Puccinia asparagi DC* (Thompson and Hepler, 1956). However *A. officinalis* ($2n = 20$) and *A. densiflorus* cv. Sprengeri ($2n = 60$) are not closely related, and hybridisation to improve resistance to all three pathogens in *A. officinalis* has proved impossible to date. The use of gene-transfer vectors such as *Agrobacterium tumefaciens* which has been successfully used to transfer foreign genes into *A. officinalis* (Hernalsteens *et al.*, 1984) may enable bridging of this and similar barriers to crossing.

It has recently been shown that asparagus plants infected with asparagus virus II (AV-II) are more susceptable to *F. oxysporum* f. sp. *asparagi* than plants free of this virus (Evans and Stephens, 1986). Hence production of seed free of AV-II is likely to result in cultivars less susceptible to Fusarium root rot (Falloon *et al.*, 1986b) (see section on AV-II).

SPEAR SPOT

F. redolens Wollenweber was first recorded as a pathogen of asparagus in New Zealand in 1960 in Auckland, Gisborne, Hawkes Bay and Canterbury (Dingley, 1965) and has subsequently been found on asparagus in Horowhenua (Cheah, 1986).

Symptoms

Small, brown to rusty brown spots develop on the tips of scales on asparagus spears. Under wet field conditions most of the bracts can be affected and in severe cases the spears may become deformed or stunted. Lesions on the spear tissue between bracts are often larger (2-5mm) and usually occur at ground level (Cheah, 1986).

Dingley (1965) noted that *F. redolens* caused root damage resulting in vascular discolouration and cortical rot of crown tissue. Spears growing from rotten crowns are often distorted.

Effect on yield

F. redolens may reduce the vigour of seedlings (Dingley, 1965) and may cause rejection of harvested spears (Cheah, 1986). However its occurrence is sporadic and epidemics show no clear relationship to rainfall, soil or air temperature or relative humidity (Cheah, 1986).

Control

No field trials have been reported but applications to spears of benomyl or prochloraz both at 50 μg ai.i *l* controlled the development of symptoms on spears in the

laboratory. However, field applications of these fungicides to spears to protect against spear spot seems impractical as it would not be feasible to keep a protective cover on all tissue surfaces because new spears are emerging every day. Soil drenches may be a more practical method of controlling this disease.

PHYTOPHTHORA ROT OF ASPARAGUS

The fungus that causes this disease in New Zealand, *Phytophthora megasperma* Dresh. var. *sojae* Hildebrand has been isolated from asparagus fields throughout the main asparagus growing areas. (Fig. 4) (Boesewinkel, 1974; Falloon, 1982b) and causes rot of spears, crowns, storage and feeder roots. A number of other *Phytophthora* species are known to cause disease on asparagus in other countries e.g. *P. richardiae* Buisman in Australia (Anon, 1961), *P. cactorum* (Lebert & Cohn) Shroeter in France (Molot, 1962; Moreau and Zuang, 1977), *P. megasperma* Drechsler in Switzerland (D. Gindrat, pers. comm.), and *P. megasperma* var. *sojae and P. cryptogea* Pethybr. & Laff. are pathogenic on asparagus in California (Falloon *et al.*, 1983; Falloon unpub.).

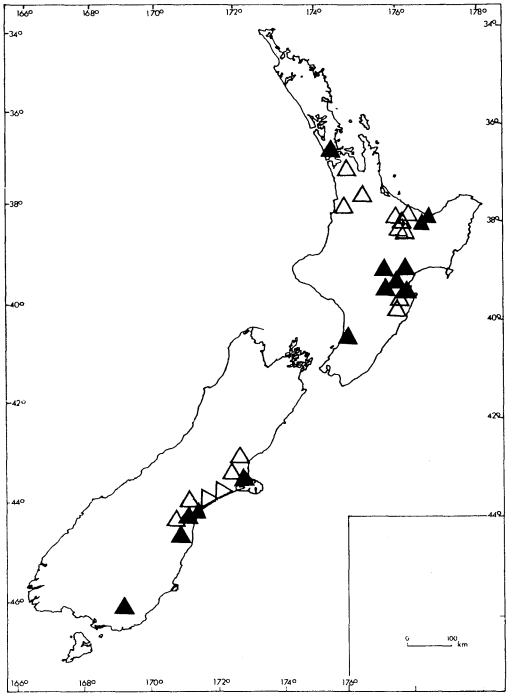

Figure 4.: Distribution of *Phytophthora megasperma* var. *sojae* in soils of asparagus fields in New Zealand. ▲ = *P. megasperma* var. *sojae* detected in soil; △ = not detected.

Symptoms

Phytophthora spear rot is characterised by soft, water-soaked lesions of different sizes on spears. The lesions usually occur at, slightly above, or below soil level but rarely extend into the crown (Falloon *et al.*, 1983). The tissue or bracts around the lesions often appear slightly wilted and the spears are usually crooked with lesions on the inside of the crook. However, crooking of spears may also result from insect or mechanical damage. Abundant white mycelium may be formed on the surface of softened spears under wet conditions (Boesewinkel, 1974). Ark and Barrett (1938) noted that "an extremely vile odour" was often associated with Phytophthora spear rot due to secondary invasion by saprophytic bacteria. Bacterial infection may cause the slimy texture of *Phytophthora*-infected asparagus spears. Under dry field conditions the whole lesion may become light brown and the spear may finally shrivel (Boesewinkel, 1974; Falloon, 1985).

Boesewinkel (1974) noted that mature plants (up to 7-yr-old) when infected with *Phytophthora megasperma* var. *sojae* show yellow to light brown shoots and cladophylls. Under wet field conditions in summer and autumn, lesions may occur at the base of senescing fern stalks (Falloon, 1985). The lesions are grey-beige to brown and the epidermal layers are easily removed by light rubbing revealing slimy tissue beneath.

Phytophthora spp. often can be isolated from newly formed storage roots that appear cream but slightly transparent and water-soaked (Falloon, 1985). Mature storage roots of infected plants often appear brown to reddish brown and occasionally are hollow (Falloon *et al.*, 1983). Feeder roots appear water-soaked when examined soon after infection but may be completely absent from plants infected several weeks previously.

During wet field conditions in winter and spring *Phytophthora* spp. can be isolated from crown tissue where it is associated with a brown or slightly transparent rot (Falloon, 1985). Phytophthora crown rot is usually most extensive in young crown buds and extends from these into the older tissue. It is usually associated with a vile odour similar to that associated with spear infections. This distinguishes it from Fusarium crown rot which usually results in a dry, dark brown rot with no odour.

Disease Cycle (Fig. 5)

Phytophthora rot epidemics in asparagus are always associated with climatic conditions that result in saturated or near saturated soil (Ark and Barrett, 1938; Boesewinkel, 1974; Falloon *et al.*, 1983; 1985; 1986a). Phytophthora rot usually occurs in the early part of the harvest season or shortly after transplanting crowns when soil temperatures are between 9 and 24 °C (Falloon *et al.*, 1986a).

Sporangium formation (Kuan and Erwin, 1982), persistence (MacDonald and Duniway, 1978), zoospore release (Ho and Hickman, 1967a; MacDonald, 1978) and zoospore motility (Duniway, 1979) are all greater in near saturated soil conditions. This was also found for germination of oospores and growth of mycelium (Kuan and Erwin, 1982).

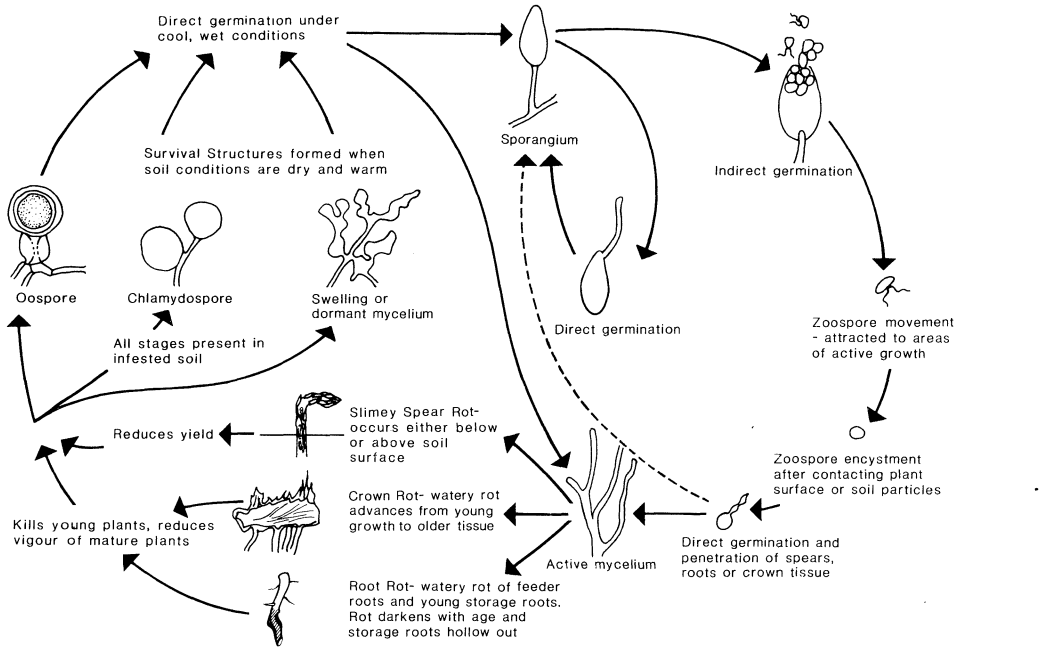

Figure 5: Disease cycle of Phytophthora rot of asparagus.

Although Ho and Hickman (1967b) calculated that zoospores of *P. megasperma* var. *sojae* could travel up to 563 mm in one hour, contact with solid surfaces stimulates zoospore encystment. Hence zoospore movement in water may only be of significance when in close proximity to plant tissue. However movement of *Phytophthora* propagules by rain splashing or in soil drenches, irrigation water and streams probably enables wide dispersal of the pathogen (Hickman, 1958).

Zoospores of *P. megasperma* var. *sojae* are attracted by chemicals e.g. amino acids and sugars released from cut ends of roots or in the region of root elongation (Ho and Hickman, 1967b) and also possibly of spear elongation where maximum exudation of chemotactically active substances is found (Hickman, 1970; Mehrotra, 1970; Carlile, 1983). Following attraction to the root or spear surfaces, zoospores are trapped and encysted. Cyst germination commences within 0.5 to 1 h and is stimulated both by root exudates and root extracts (Ho and Hickman, 1967b).

Following penetration of the asparagus tissue by germ tubes from zoospore cysts, active mycelium develops resulting in rotting of feeder roots, storage roots, crowns, spears or fern stalks. Sporangia may be produced on the surface of plant tissue under wet field conditions or oospores may form within rotten tissue.

Depending on the species of *Phytophthora* involved, zoospores, chlamydospores and/or swollen or dormant mycelium serve as survival structures over the summer months when soil moisture and temperature conditions may not be suitable for disease development. Under wet, cool conditions these germinate and resume growth starting the disease cycle again.

Effect on Yield

Phytophthora rot has been found to cause losses at three stages in the growth of asparagus:

a) During establishment when asparagus crowns are transplanted under cool, wet conditions. Roots are damaged during transplanting and as discussed earlier, zoospores of *Phytophthora* are attracted to broken root surfaces. Transplanting infected crowns into virgin asparagus soil may help spread the disease. At this stage *Phytophthora* spp. may cause emergence to be delayed and increases in root and crown rot which, in combination, reduce vigour of summer fern growth and survival of plants by as much as 83% (Falloon, 1985).

b) During the harvest season *Phytophthora* spp. may attack emerging spears. Reductions in yield of up to 59% have been recorded (Falloon *et al.*, 1985). Two successive years of severe Phytophthora rot was shown to reduce yields in subsequent years (Falloon *et al.*, 1986a). Thus Phytophthora rot was implicated in asparagus decline.

c) *Phytophthora* spp. may cause post harvest losses. After picking, asparagus spears are usually washed to remove soil from the surface of the spear. This is especially important when spears are harvested under

muddy field conditions. Spears are then sorted and packed usually without being dried. Packed asparagus may have a wet pad at the base of the spears to prevent dessication of spears in transit. This helps to maintain high humidity and would delay drying of water droplets that remain on the surface of the spear after washing. Recycled wash water may spread *Phytophthora* zoospores, sporangia or mycelial fragments (Ark and Barrett, 1938) which may germinate and penetrate spears while they are held under conditions of high humidity. In tightly packed boxes hyphae may spread from spear to spear thereby increasing the severity of loss in storage.

Control

a) Establishing asparagus. When crown transplants are used *Phytophthora*-related establishment failures can be avoided if crowns are grown in soil free of *Phytophthora* spp. pathogenic on asparagus and by cool storage of crowns until soil conditions are dry and warm. Production fields without a history of Phytophthora rot should be used. If crowns are planted under wet soil conditions dipping of crowns in solutions of metalaxyl at concentrations as high as 200 μg a.i. *l* or in-furrow spray applications at rates of 0.25 to 0.5 kg a.i./ha immediately after the crowns have been transplanted and covered with soil has provided good control (Falloon, 1985). Fosetyl-Al is an alternative fungicide that has been effective in California (R.J. Mullen pers. comm.) for the control of Phytophthora rot during establishment of asparagus but the best rates to use have not yet been determined.

Alternatively the use of seedling transplants, which are usually transplanted later in the spring when *Phytophthora* is less likely to be a problem, will help avoid the disease during establishment of an asparagus field.

b) Established Asparagus. Metalaxyl applied at rates of 1.0 to 1.3 kg a.i./ha 10 to 14 days before the start of harvest has proved effective for controlling Phytophthora spear rot (Nikoloff, 1984; Falloon *et al.*, 1985). There is no advantage from higher rates or earlier application times. Autumn foliar applications of foestyl-Al have also controlled Phytophthora rot in established asparagus (Mullen pers. comm.).

There is no adequate level of resistance to Phytophthora rot amongst any of the cultivars of *A. officinalis* presently recommended for New Zealand (Falloon, 1986a).

Lupins *(Lupinus angustifolius, L. luteus, L. albus, L. bicoler, L. densiflorus* and *L. succulentus*) are alternate hosts of *P. megasperma* var. *sojae* (Jones & Johnson, 1969; Boesewinkel, 1974). The use of lupins in rotation with asparagus should therefore be avoided.

A number of factors such as drought stress (Blaker and MacDonald, 1981), excessive soil water (Kuan and Erwin, 1980), and high salt concentrations (MacDonald, 1982) are known to predispose a number of crops to Phytophthora root rots. These conditions should be avoided in asparagus culture.

Adequate field drainage will reduce the duration of saturated soil conditions and hence reduce the severity of disease.

Because asparagus roots infected with AV-II leak more electrolytes, glucose, total carbohydrates and amino acids than roots of virus-free plants (Evans and Stephens, 1986) the use of AV-II free seed may reduce the attraction of zoospores to asparagus roots and hence reduce disease severity.

c) Post harvest. Addition of sodium peroxide (0.5%) (Ark and Barrett, 1938) or sodium hypochlorite (0.5%) to water used to wash spears after harvest will help limit spread of post harvest Phytophthora spear rot. Wash water should not be recycled.

The use of circulating air to dry the spears after washing would reduce the amount of surface water on the spears. This would adversely affect the survival of zoospores or sporangia while at the same time, cooling the asparagus through evaporation of water.

ASPARAGUS VIRUS II

This virus is also known as asparagus latent virus and asparagus C-type virus (Uyeda and Mink, 1984) and is the only virus identified in asparagus in New Zealand (Falloon, 1986b). However there has been no systematic survey of asparagus fields throughout New Zealand to determine whether other viruses also are present in the crop. Surveys of asparagus fields in Hawkes Bay in 1977 and Canterbury in 1984 showed that up to 100% of plants were infected with AV-II (J.W. Ashby pers. comm.). Although two strains, P and S are reported in Washington State, USA (Uyeda and Mink, 1981) the identity of the strain(s) that occur in New Zealand has not been confirmed.

Symptoms

Plants infected with AV-II show no distinct symptoms although vigour and productivity are reduced (Weissenfels and Schmelzer, 1976; Yang, 1979).

Disease Cycle (Fig. 6)

AV-II is transmitted through seed and up to 67% of seeds may be infected (Paludin, 1964). AV-II-infected seed is the most likely source of virus in recently planted asparagus fields in New Zealand. As no effort is being made to produce virus-free seed of the main cultivars currently grown in New Zealand most seed of these cultivars imported to New Zealand is likely to be infected with AV-II.

Within asparagus fields AV-II may be spread from plant to plant via mechanical transmission (Fujisawa *et al.*, 1983; Evans and Stephens, 1986) although this occurs at a relatively slow rate (Falloon *et al.*, 1986b). AV-II may also spread via pollen from infected male plants to seed produced by females (Evans and Stephens, 1986). As the incidence of infected plants increases with time the proportion of infected seed harvested from AV-II-infested seed fields will also increase.

Although AV-II has been experimentally transmitted

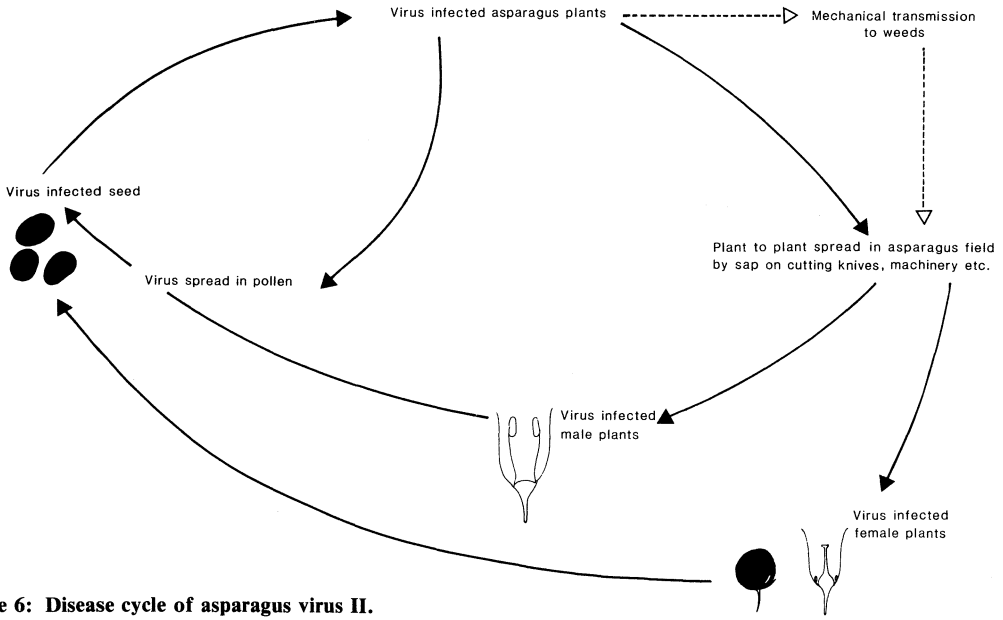

Figure 6: Disease cycle of asparagus virus II.

by mechanical means to 116 species of 27 families of dicotyledonous plants (Uyeda & Mink, 1981) and two species in two families of monocotyledonous plants (Weissenfels *et al.,* 1978) the role of weeds in the life cycle of AV-II is unknown.

Effect on Yield

There has been no work reported on the effect of AV-II on asparagus production in New Zealand. Yang (1979) in Washington State showed that AV-II caused slight reductions in vigour of cloned pistillate plants in the first 16 months of growth. However, when both AV-II and a second insect transmitted virus, asparagus virus I were present in the same plant there was a synergistic effect resulting in a serious decline in vigour and survival of plants.

Recently Evans and Stephens (1986) have shown that roots of asparagus plants infected with AV-II exude seven to eight times more amino acids and two to three times more glucose and carbohydrate than roots of healthy plants. These exudates stimulate germ tube growth of spores of *F. oxysporum* f. sp. *asparagi* and are involved in attraction of *Phytophthora* zoospores to root surfaces. Thus AV-II-infected plants are more susceptible to root rot caused by *F. oxysporum* f. sp. *asparagi* than AV-II-free plants (Evans and Stephens, 1986). This may also apply to Phytophthora rot.

Control

Because plant to plant spread within asparagus fields results in increased incidence of AV-11-infected seed, F2 seed should not be harvested from fields established from cultivars introduced from overseas. The lower yield of crops established from F2 seed may in part be the result of increased incidence of AV-11 in F2 seed in comparison with F1 seed.

It is possible to eliminate AV-II from the parents of asparagus hybrids using tissue culture (Yang and Clore, 1976) which enables production of virus-free asparagus seed (Falloon *et al.,* 1986b). This is at present being done with the parents of Crop Research Division experimental hybrids prior to their commercial release.

CONCLUSION

Although a number of diseases affect asparagus production in New Zealand the relative importance of each is likely to vary from season to season. Several diseases have been implicated in a reduction of the productive life of asparagus fields i.e. Fusarium rot, Phytophthora rot and AV-II. Hence the control of these diseases will likely result in extension of the profitable life of asparagus fields.

Adequate methods of chemical control exist for Stemphylium leaf spot and Phytophthora rot and could be developed for Fusarium rot while AV-II can successfully be controlled and possibly eliminated by the use of virus-free seed. Improved levels of resistance to Stemphylium leaf spot and purple spot are being bred into Crop Research Division advanced breeding lines and approximately 100,000 seedlings have been screened for resistance to Phytophthora rot as the first stage in producing a *Phytophthora*-resistant asparagus cultivar. The use of resistant cultivars will eliminate the current need for annual applications of expensive fungicides and the reliance on often costly and complicated management techniques currently necessary to control the major diseases of asparagus in New Zealand.

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