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Asparagus Diseases

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ABSTRACT

Crown and root rot is the most serious disease of asparagus worldwide resulting in plant yellowing, dieback and wilting. As the disease progresses, root parenchyma is completely destroyed whereas crown interior is discolored. The fungi that play major role to this disease are species of the genus *Fusarium*. The two dominant species are *F. oxysporum* f. sp. *asparagi* and *F. proliferatum*. The species *F. solani*, *F. culmorum*, *F. subglutinans* and *Phytophthora* spp. are less frequently isolated from diseased asparagus plants. Besides, *Fusarium* species are the main biotic factors responsible for asparagus decline syndrome. Seeds, crowns, root residues and field soil consist the pathogen inoculum sources. Yield loss results from plant death and from smaller and fewer spears. Other economically important fungal diseases are asparagus rust, caused by *Puccinia asparagi* that infects asparagus green parts and the purple spot, caused by *Stemphylium* spp. which appears as brown lesions with dark purple margins on the main stems, branches and cladophylls. Both diseases cause a severe drop of cladophylls while the plants turn yellow or brown; they also reduce the vitality of the root system resulting in the subsequent year yield reduction. Other fungal diseases of asparagus include stem blight caused by *Phomopsis asparagi*, and Cercospora blight caused by *Cercospora asparagi* causing subsequent yield loss, particularly in humid areas. Furthermore, several viruses have been found to infect asparagus causing latent infections.

Keywords: *Cercospora asparagi*, *Fusarium oxysporum* f. sp. *asparagi*, *Fusarium proliferatum*, *Phomopsis asparagi*, *Puccinia asparagi*, *Stemphylium* spp.

CONTENTS

INTRODUCTION

Asparagus (*Asparagus officinalis* L.) is a perennial dioecious monocot crop which belongs to the Liliaceae family, and is grown for its herbaceous, newly emerged shoots, harvested either green or white and which are referred to as spears. Asparagus cultivation was first practiced by the Greeks around 200 B.C. and then by the Romans (Mills 2000). Most crops are established from one-year old crowns on light, well drained sandy loam soils and they reach profitability after five years. The development of all-male hybrid cultivars has increased significantly yields in recent years. The plantations remain productive, theoretically, for up to twenty years, however the economical life of asparagus plantations is usually shorter owing to several factors, including asparagus diseases, and this condition is known as asparagus decline. Grogan and Kimble (1959) have defined asparagus decline as the gradual decrease of the size and number of spears up to the point where the plantings, after a peak production, become unprofitable to maintain. The crop may suffer from a premature loss in productivity, which can be attributed to a range of factors. Decline can cause problems both in asparagus crops planted on new lands and in replant crops growing on land previously cropped with asparagus. The diseased plants lead to a reduced storage of root carbohydrates resulting in the decline of yields in following years due to a decrease in size, number and saleable spears. In addition wherever plantings have declined it is impossible to reestablish productive ones (replant problem) (Schreuder *et al*. 1995; Elmer *et al*. 1996; Lori *et al*. 1998). In the Netherlands early decline or replant disease is confined only to replant crops while problems with crop establishment rarely occur in new lands (Blok and Bollen 1996a). A number of abiotic and biotic factors contribute to asparagus decline (Elmer *et al*. 1996).

Abiotic factors, such as soil type and physical properties, are extremely important to ensure the long-term cultivation of asparagus. Soils should preferably be light and contain sufficient organic content. It is recommended that the pH should lie between 6.5 and 7.5 at a depth of 30-40 cm. Selecting the optimum harvest duration can help the vigor of the plants. It was recommended that the harvest should begin after the third year of establishment initially lasting for 8 weeks but should progressively be reduced to 6 weeks with the increase in aging of the crowns. Allelopathic compounds, present in asparagus roots, have a direct growth inhibition to new asparagus plants whereas biotic factors, such as several diseases, can play an important role in aspa-

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Invited Review

Fig. 1 Asparagus diseases. (**A**) Internal crown discoloration of asparagus caused by *Fusarium proliferatum*. (**B**) Root rot of asparagus caused by *Fusarium proliferatum*. Feeder roots and parenchyma of storage roots are completely destroyed. (**C**) Stem rot of asparagus caused by *Fusarium proliferatum*. (**D**) Asparagus rust caused by *Puccinia asparagi.* Black telia formed in the uredinia on asparagus fern. (**E**) Lesions caused by *Stemphylium botryosum* on asparagus fern. (**F**) Elongated lesions caused by *Phomopsis asparagi* on the stem of asparagus. (**G**) Lesions caused by *Phomopsis asparagi* on asparagus fern.

ragus decline as well (Elmer 1996).

The diseases that cause serious problems to asparagus cultivations word-wide and their control methods, are included to this review paper.

FUSARIUM CROWN, ROOT AND STEM ROTS

Crown, root and stem rots, wilt and seedling blight caused by *Fusarium* species is the main biotic factor responsible for the asparagus decline syndrome. They are the most serious diseases of asparagus worldwide since by damaging the underground parts, it becomes the limiting factor in all asparagus producing areas. *F. oxysporum* (Schlecht.) Emend. Snyd. & Hans. f.sp. *asparagi* Cohen and *F. proliferatum* (Mats.) Nirenb. were identified as being mainly responsible for attacking asparagus. *F. moniliforme* Sheld. is pathogennic to asparagus but now it has been taxonomically separated into different species (O'Donnell *et al*. 1998) including *F. proliferatum* (presence of polyphialides) that is identified as being mainly responsible for asparagus disease. Isolates from asparagus previously referred to as *F. moniliforme* have recently been reclassified as *F. proliferatum* (personal unpublished data). The species *F. solani* (Martius) Sacc. *F. culmorum* (W.G. Sm.) Sacc. and *F. subglutinans* (Wollenw. and Reinking) Nelson do not appear to be important pathogens of asparagus due to the low frequency of isolation from diseased asparagus plants. *F. redolens* Wollenw. f.sp. *asparagi* Baayen, described as host-specific pathogen involved in root, crown and spear rots of asparagus, was previously reported as *F. oxysporum* var. *redolens* (Wollenw.) Gordon (Graham 1955; Johnson *et al*. 1979; Schreuder *et al*. 1995; Elena and Kranias 1996; Elmer *et al*. 1996; Lori *et al*. 1998; Baayen *et al*. 2000; Wong and Jeffries 2006).

The symptoms of the disease include seedling blight, yellowing, stunting, wilting and death of plants. On the base of the stem and on the fleshy roots, brown oval-shaped le-

sions are observed. The affected plants also reveal a reddish brown internal crown (**Fig. 1A**), stem and root discoloration. As the disease progresses, the fungi cause extensive rot of stem base (**Fig. 1C**), and a complete destruction of feeder roots and parenchyma of storage roots until only the central axis and the epidermis of the root remain (**Fig. 1B**), resulting in the weakness or death of diseased plants. In the asparagus fields large gaps remain with significantly lower crop production until the crop becomes too sparse to harvest economically and the field may be destroyed (van Bakel and Kerstens 1970; Elena and Kranias 1996). Additionally, asparagus spears can be infected both by *F. proliferatum* and *F. oxysporum* f.sp. *asparagi* during the cropping period (Cosme Guerrero *et al*. 1997). Chemical analyses have revealed that fumonosin B1 and moniloformin (both toxins that may pose a potential risk for human health) were present in some of the infected spears sampled in Poland (Weber *et al*. 2006).

Both dominant pathogens enter the plants through their young feeder roots, they spread through the storage roots and crowns, and finally they weaken and kill the plants. *F. oxysporum* f.sp. *asparagi* attacks the feeder and storage roots and occasionally the crowns and stems while *F. proliferatum* attacks the crowns, the stems and rarely the roots. In South Africa, in Greece and also in other countries the two most prevalent *Fusarium* species isolated from crowns, roots and stems of asparagus are *F. proliferatum* and *F. oxysporum* f.sp. *asparagi* and to a lesser extent *F. solani*. *F. proliferatum* isolates are more virulent to asparagus seedlings than *F. oxysporum* and *F. proliferatum on asparagus* was more abundant in Greece and Spain than in the UK while in the Netherlands it was not detected (van Bakel and Kerstens 1970; Blok and Bollen 1995; Schreuder *et al*. 1995; Blok and Bollen 1996a; Elena and Kranias 1996; Wong and Jeffries 2006). A large genetic diversity was present in *F. oxysporum* f.sp. *asparagi* population. In a collection of 79 isolates, derived from the United States, from Europe and from Taiwan, 43 Vegetative Compatibility Groups (VCGs) were identified (Elmer and Stephens 1989) while in the Netherlands 24 isolates were assigned to 18 different VCGs. Using also the VCGs test, over 110 isolates of *F. proliferatum*, collected from the U.S.A., were assigned to over 20 VCGs but three tended to predominate (Elmer *et al*. 1996). The genetic and molecular variation, of 20 isolates of *F. proliferatum,* derived from several geographic areas in Greece, was studied with RAPD and VCGs analyses. It was found that the isolates fell into four different groups but in a different way according to each method. It is deduced that *F. proliferatum* has a wide genetic diversity in Greece (Paplomatas and Elena 2001). The mtDNA RFLP data indicated a significant heterogeneity in *F. proliferatum* isolates obtained from the same or different host species (Laday 2004). There is considerable diversity in *F. solani* populations associated with asparagus in both genomics and pathogenicity terms since some isolates were pathogenic while other populations did not kill asparagus seedlings. However *F. solani*, as just mentioned, does not appear to be an important pathogen for asparagus crops (Schreuder *et al*. 1995; Elena and Kranias 1996; Wong and Jeffries 2006). Recently a PCR-denaturing gradient gel electroforesis (DGGE) method was developed to assess *Fusarium* species diversity in asparagus plant samples. The technique was effective to visually discriminate between the majority of *Fusarium* species, while a further sequencing step permitted to distinguish between the species showing similar migration patterns (Yergeau 2005).

F. oxysporum f.sp. *asparagi* can survive during asparagus-free periods for up to at least 25 years. Persistence of the fungus in asparagus root residues is the major reason for its long-term survival (Blok and Bollen 1996b). *F. proliferatum* is not a strong soil inhabitant but it is also airborne in asparagus and corn fields since it can sporulate on symptomatic stalks when humid conditions prevail (Sharma and Singh 1978; Elmer *et al*. 1996). It should be taken under consideration that corn and asparagus are frequently grown in close proximity and often follow one another at a particular site (Lori *et al*. 1998).

The disease appears to be a stress related syndrome, in part, because some management practices reduced disease development despite crown infections (Damicone *et al*. 1987). The prolongation of the harvest period weakens the plants that become susceptible to infections. There are many soil, environmental and cultural factors (e.g. soil texture, soil moisture, pH, compaction, nutrient stress, drought etc.), virus or other fungal diseases, and damage by insects that affect the growth and subsequently the yield of asparagus plantings. It has been observed in the field that when some of these factors were unfavorable to the plants, they increased the susceptibility of asparagus to *Fusarium* infection and the disease development. Hamel *et al*. (2005) indicated that soil Mn availability was negatively correlated with the percentage of field area affected by Fusarium crown and root rot. In the same study a survey on asparagus plantations in Quebec noted that pathogenic *Fusarium* spp., in particular *F. oxysporum* f.sp. *asparagi* and *F. proliferatum*, were found in all plantations on both asymptomatic and symptomatic plants, suggesting that disease expression requires the combined influence of *Fusarium* strains and other factors, such as reduced Mn availability.

Insects spread the disease operating as vectors of the *Fusarium*. The dipteran insects *Hexomyza* (*Ophiomyia*) *simplex* (Loew), and *Delia* (*Hylemyia*) *platura* (Meigen) which attack the stem of asparagus are vectors of pathogenic strains of *F. proliferatum* to asparagus. The insects had been collected using yellow sticky traps, which were placed in several experimental asparagus plantations in Greece (Elena *et al*. 2006). Asparagus plants mined by the insect *Hexomyza simplex* developed greater stem rot that was apparently related to mine frequency (Damicone 1987).

Furthermore, several viruses have been found to infect asparagus commercial plantings worldwide, causing the asparagus plants to be more susceptible to *Fusarium*.

Now it has become clear from various research studies as well as from field observations that the intensity of damage due to *Fusarium* infection is related to the extent of plant stress. In order to minimize losses in asparagus production due to *Fusarium* infection, all the above factors that can create plant stress should be minimized as much as possible. However, quantitative data on the relationship between specific stress factors and *Fusarium* damage is lacking almost completely.

Asparagus *Fusarium* species can transmit by soil, crop debris, seed or nursery crowns. The disease control is difficult and preventative measures are very important to suppress Fusarium crown and root rot. The selection of land never previously planted with asparagus helps to avoid *Fusarium* propagules and allelopathic compounds that increase the asparagus susceptibility to Fusarium crown and root rot. The land must have light sandy, well-drained soil with pH 6.5-7.5. The use of disease-free propagation material (seeds or crowns) is essential. The seeds or crowns can be treated with recommended fungicide before the planting. For crown production, plants should be produced outside of commercial asparagus plantations. Optimizing crop vigor, using suitable all-male hybrids for each area, maintaining the crops at optimum growing conditions with proper irrigation and fertilization, helped to reduce the financial losses. Cutting pressure should be avoided by shortening the harvest period to minimize the plant stress. Programs that control pests, other diseases and weeds reduce the damage from the disease. When established plantings show signs of decline the diseased plants with the roots and the soil around them must be removed and destroyed out of the field.

Non-pathogenic isolates of *F. oxysporum* were shown to have potential to reduce severity of asparagus root rot caused by *F. oxysporum* f.sp. *asparagi* or effectively induced systemic acquired resistance (Blok *et al*. 1997; He *et al*. 2002). The perennial nature of the asparagus may present obstacles for successful disease control with non-pathogenic strains. An effective biocontrol agent would need to posses

the ability to colonize the young feeder roots every spring (Elmer 2004). In addition, early colonization of asparagus roots by vesicular arbuscular mycorrhizae (VAM) fungi, which provides a natural biological control against root diseases, can suppress infection by *Fusarium* spp. (Elmer 2002).

In experimental conditions, plots receiving NaCl (rock salt) had greater spear numbers and spear weights (Elmer 1989). Field applications of NaCl and post-aplication irrigation did result in significant reduction of rhizosphere populations of *Fusarium* spp. resulting in the suppression of the disease and the increase of marketable yield. Field applications of NaCl were not continual but were applied once or twice per year to avoid damages to other crops if the fields were taken out of asparagus (Elmer 1992). Later, the use of rock salt gained attention among commercial growers who wished to extend the life of asparagus plantings (Elmer 1996). The mechanism of NaCl on Fusarium crown rot is unclear. However there was more suppression when the NaCl was applied directly to the roots, which suggests that multiple mechanisms may be operating in the disease suppression. The systemic activity of chloride may allow growers to band-apply this instead of a broadcast application and minimize sodicity on heavier clay soils (Elmer 2003). Applications of NaCl did not hinder the suppressive ability of the nonpathogenic strains of *Fusarium oxysporum* (Elmer 2004).

To control the soil-borne pathogens, in addition to seed or crown health, soil disinfestation by chemical fumigation or by steaming has been used before the establisment of the plants. The high cost of these methods and the quick re-establishment of the pathogens are the limiting factors for outdoor plantations. The population of *F. proliferatum* was eradicated in experiments by soil solarization (Katan *et al*. 1976), which could be a cultural successful strategy in managing the disease in the Mediterranean region (Elena and Paplomatas 2002). This method can apply alone or in combination with reduced doses of soil fumigants or biological factors. It is relatively cheap, simple and non hazardous and the solarized soils are less receptive to pathogens reinfestation (Katan 1987; Elena and Tjamos 1997).

Biodisinfestation is a technique that involves the incorporation of large quantities of organic amendments in combination with an airtight plastic cover to create anaerobic conditions. Under these conditions, chlamydospores of *Fusarium oxysporum* f. sp. *asparagi* were strongly or completely inactivated after 7 weeks. The method may provide an alternative for soil disinfestations, for high-value crops, where soil solarization is not feasible (Blok 1997; Elena *et al*. 1999; Blok *et al*. 2000).

PHYTOPHTHORA ROT

Several species of *Phytophthora* attack asparagus plantations in Europe, America, Australia and New Zealand (Fallon *et al*. 2002). A soft rot of asparagus spears caused by *Phytophthora* sp. was first reported in California (Ark and Barrett 1938). The disease appeared to be related to a period of heavy and prolonged rainfall and perhaps to the rather common practice of flooding certain areas to produce earlier growth. The main species, pathogenic to asparagus in California, was *P. megasperma* Drechsler var. *sojae* Hildebrand, although highly virulent isolates of *P. cryptogea* Pethybridge and Lafferty were occasionally found (Fallon and Grogan 1988). In spring 2004 *Phytophthora* sp. was isolated from asparagus spears, roots and dormant crowns in Michigan where the diseased spears were often curved, had water-soaked lesions slightly above or below the soil line or they were shriveled at the site of infection or both. Infected storage roots had water-soaked but no soft lesions while infected crowns had fewer roots. The occurrence of excessive rainfall, in Michigan area, the spring 2004 is responsible for the spreading of the disease and yield losses (Saude *et al*. 2005). *P. megasperma* var. *sojae* was recorded for the first time in New Zealand as a new disease of asparagus causing the above same symptoms but the disease was also able to kill young and old asparagus plants (Boesewinkel 1974). *P. megasperma* var. *sojae* caused significant losses in the production, resulting from a slimy rot of white asparagus spears in southwest France (Baudry *et al*. 1995). The same species was isolated from wilted plants of asparagus in Canada (Vujanovic 2003). The species *P. richardiae* Buisman was also reported to cause spear rot of A*sparagus* sp. (Geoffrey 1991). However Phytophthora rot is a problem in some asparagus producing areas with wet, waterlogged soils. Losses by Phytophthora attack was higher during wet seasons and was also higher during the early part of each season when soil conditions were cool and wet (Fallon *et al*. 1986). Several Phytophthora tolerant experimental hybrids were identified in New Zealand for their higher yield and quality (Fallon *et al*. 2002). To avoid *Phytophthora* rots preventative methods such as the use of soils good drainage, without propagules, and disease-free propagation materials are important. If symptoms of the disease are obvious and severe, suitable fungicide application is necessary.

ASPARAGUS RUST

Asparagus rust caused by the macrocyclic and autoecious fungus *Puccinia asparagi* De Candolle is present since 1805 in all parts of Europe, where asparagus is grown, since 1896 in America and from the begin of this century in Australia (Viennot-Bourgin 1949; Sherf and Macnab 1986; Cheah and Davis 2002; Davis 2002).

The first symptoms of the disease appear in spring on spears and main stems as oval light green lesions on which pycnia (spermogonia) are formed. The light orange aeciospores are produced in the same lesions 1-2 weeks later, whereas both pycnia and aecia are formed on young shoots. The air causes new infections carrying the aeciospores when free moisture is present. Uredinial stage covers stems, twigs and leaves with small, rusty-red pustules 2-3 weeks later, and after the spring cutting period. Uredinia are formed in great number below the epidermis that splits, while uredospores that appear as rust colored powder, disseminated by both wind and rain, cause new repeated infections in the presence of moisture and suitable temperature increasing disease incidence. Later in the season (fall) the black telia were formed with large two celled heavy walled teliospores in the same pustules (**Fig. 1D**) or in different bodies. *P. asparagi* overwinters as teliospores on infested asparagus debris. In the spring after the asparagus shoots emerge, teliospores put out a short four-celled germ tube (basidium) producing basidiospores, each from one cell, which germinate causing new infections. Pycnia and aecia occur on volunteer asparagus plants or on non harvested shoots in the spring. The disease stops to develop further if the volunteer plants are destroyed and the shoots are harvested. Temperature is not as important as moisture for the infection and epidemics depend on heavy rain, high humidity and abundant dew which are much more favorable than heavy rains. The disease can cause premature defoliation or death of the ferns reducing the carbohydrates storage in the crown, resulting in fewer yields in the following cutting seasons and in the increase of the incidence of root or crown diseases. Yield reductions are usually greater after two years of rust infection (Viennot-Bourgin 1949; Sherf and Macnab 1986; Johnson 1990b; Johnson and Lunden 1992; Elmer *et al*. 1996).

Rust reduces the total weight and number of spears produced individually by the susceptible cultivars than by resistant ones (Elmer *et al*. 1996). Most of the cultivated varieties of *A. officinalis* were found to be susceptible to asparagus rust (Thompson and Hepler 1956). Resistance in asparagus to urediniospore infection is paramount because of the repeating cycle of infections during a season. However the development of aecia on asparagus is important for the built-up of inoculum during the early phases of the disease (Johnson 1990b). Heterogeneity for rust resistance exists within open pollinated and clonal hybrid asparagus cultivars. To develop more highly resistant cultivars, selected germ-

plasm from commercial asparagus should be used (Johnson 1989).

Control is based on preventative and chemical measures. Asparagus available cultivars resistant to rust should be grown in areas where rust may develop. Sanitation practices apply, such as the destruction of the stubble, wild and volunteer asparagus plants and unused asparagus beds during the winter. The harvest period must keep until early summer to avoid aeciospore infections. The aeration of the crops should be favored by planting wide rows in the direction of prevailing winds to help plants dry off. Asparagus plants during the fern period must have good irrigation while at the end of this period the cut and burial or destruction of the plants reduce the fungus inoculum. The new plantations, which were allowed to produce their ferns, need to be sprayed since they are more susceptible to infection than the older ones and built-up a lot of inoculum. Fungicides application in older crops is useful if disease thresholds are developed, the weather conditions are suitable and the length of time remaining in the growing season is sufficient (Sherf and Macnab 1986; Johnson and Lunden 1992; Elmer *et al*. 1996).

PURPLE SPOT (STEMPHYLIUM LEAF SPOT, SUMMER BURNING)

Purple spot of asparagus incited by *Stemphylium* spp. [teleomorph *Pleospora herbarum* (Pers.) Rabenh. Ex Ces. & de Not.] is a significant problem for asparagus production. The species *S. vesicarium* (Wallr.) Simons and *S. botryosum* Wallr. have usually been isolated from diseased plants (Takahito 1973; Lacy 1982; Johnson 1990a; Sutherland *et al*. 1990; Elena 1996; Meyer *et al*. 2000). The genus *Stemphylium* was founded by Wallroth in 1833 on the single species *S. botryosum*. The host asparagus is the "type host" for the species and the specimen, from which the fungus has been isolated, and consists of four pieces of asparagus stem preserved in Herb. Wallroth at Strazbourg University (Wiltshire 1938).

The fungus affects both plants and spears. Numerous brown small elliptical lesions, 1-2 mm, slightly sunken, with purple margins and brown center were present on harvested spears, which may result in the spears being rejected. The lesions, limited, spread, or flecked, usually appeared only on one side of green asparagus spears, while there were no symptoms on white spears that are always harvested before emerging from the planting beds. Penetration into the asparagus plant appears to be primarily via stomata. No evidence of long-distance attraction to stomata was detected while the internal tissue of the spear is not affected. The major damage from the disease is on the fern growth, where large elliptical lesions, 3 to 5 across by 10 to 13 mm long, with red brown margin appeared on stems, cladoles and branches (**Fig. 1E**). Damage to the fern results in the defoliation of the cladophylls that reduces the carbohydrates of the crowns and roots, leading to lower yields the next years (Falloon *et al*. 1984; Sutherland *et al*. 1990).

The fungus overwinters primary with teleomorph stage on the surface of asparagus senescing ferns or asparagus debris from the previous summer's fern growth. When the debris is laid on the soil surface they serve a primary windborn inoculum for purple spot on spears during the spring (Falloon *et al*. 1987). Removal of this source of inoculum from the soil surface may be a practical method of managing Stemphylium purple spot on spears. Severity of Stemphylium purple spot on spears, during the harvest period, was significantly reduced when debris was incorporated into the soil in late fall or in late winter. Conidia of *S. vesicarium* formed in the spring on the previous year's asparagus ferns and caused infection. Thus conidia, as well as ascospores, served as a source of primary inoculum. Volunteer asparagus seedlings that become infected during the harvest period may be important as a substrate for inoculum increase and as a bridge to carry inoculum from the harvest period, when spears are consistenly removed, to the time when the plants are allowed to produce ferns (Johnson 1990).

The disease is more severe in asparagus fields from the end of August to October. Disease severity was positively correlated with rainfall and negatively with evaporation, but the duration of wetness was more important than the amount of rain. The disease increases after heavy rainfall at temperature between 0 and 20°C (Meyer *et al*. 2000). Wounds are not needed for disease development but infections are more numerous and occur at shorter wetting durations on wounded than on non wounded asparagus plants (Johnson and Lunden 1986; Falloon *et al*. 1987; Elena 1996). Wounds remained entry points when injury occurred up to 24-48 hr before inoculation (Johnson and Lunden 1986). Asparagus fern grown under conditions of low light (particularly a reduced photoperiod), high RH and temperature became severely infected. Disease severity decreased after increasing the age of the fern at the time of the inoculation (Menzies *et al*. 1991).

The disease control sanitation, such as crop debris burial to prevent ascospores and conidia from becoming airborne, is important for disease management. The destruction of volunteer asparagus seedlings help to reduce the fungus inoculum (Johnson 1990a). Since disease severity can vary widely among years it is useful for the growers to prognosticate the effect of the disease on yield in order to develop an economical control. A 14-21 day interval is commonly used for fungicide applications. Weather-based systems for fungicide applications, such as Tom-Cast system, reduce significantly the number to calendar-based sprays, which are necessary to provide economic control. Suitable fungicides application according to weather conditions provides an economic control (Meyer *et al*. 2000).

PHOMOPSIS STEM BLIGHT

Phomopsis stem blight caused by *Phomopsis asparagi* (Sacc.) Bubak is known in most asparagus growing countries of North America, Africa, Europe, Asia and Southern Australia, and also as *Phoma asparagi* Sacc. (Relfschneider and Lopes 1982; Sherf and Macnab 1986; Punithalingam 1990; Uecker and Johnson 1991; Davis 2001). Three species of the genus *Phomopsis* have been described on asparagus stems. According to Uecker and Johnson (1991) the described species *P. asparagicola* Bausa Alcalde is synonym of *P. asparagi* while the species *P. javanica* Uecker *et* D.A. Johnson is distinct and more virulent than *P. asparagi*. The hosts of *P. asparagi* are three species of the genus *Asparagus*: *A. officinalis*, *A. plumosus* and *A. verticillatus* (Punithalingam 1990).

The disease is found on leaves and any part of the stem. Elongated, oval-shaped 0.5 up to 5 cm long lesions are formed on the stems (**Fig. 1F**), starting as light brown lesions that later turn dark reddish brown. As the infection progresses the affected areas become shriveled and turn into well-defined spots with pale in color central tissue surrounded by dark brown margins. The center of the lesions becomes ashy-white with numerous pycnidia, more on old ones. More lesions occur on the stem base than on the upper parts of the plant and all the parts except the berries are susceptible to infection (**Fig. 1G**) (Punithalingam 1990). In severe cases cladophylls turn yellow and later brown, the plants are partially or completely defoliated until complete desiccation and stem death occurs (Punithalingam 1990; Elena 2006). The most devastating symptom of the disease is stem blight, which causes fern death and as seen in subsequent regrowth, debilitates and reduces stands of plants in spring, particularly in moist humid areas after prolonged periods of wet weather, often in early summer. Infected stems senesce rapidly following infection, even if just a single lesion is apparent on the fern (Cheah and Davis 2002). Infection occurs rapidly through wounds. It has been found that conidia are discharged from pycnidia by immersion in water, spraying with water and saturated high humidity (Punithalingam 1990). The fungus survived on infected

stems buried in the soil during ploughing or in the ground for 3-4 months. On diseased stems at soil surface the pathogen survived more than 6 months (Punithalingam 1990).

P. asparagi is likely to disperse over longer distances on infected plant material but its ability to travel short distances during wet, windy conditions is probably quite good (Davis 2001; Cheah and Davis 2002).

For disease control, the burial or destruction of the plant debris and volunteer asparagus seedlings help to reduce the primary fungus inoculum in spring. Additionally, suitable fungicides application according to weather conditions may provide an integrated control.

CERCOSPORA BLIGHT

Cercospora blight of asparagus caused by *Cercospora asparagi* Sacc. occurs worldwide where asparagus is grown but does not cause appreciable damage in cool or dry climates. The disease has appeared in Oklahoma since 1980 and in North Carolina since 1981 and has caused significant damage to the ferns. Diseased ferns become covered with small, oval, grayish tan lesions with purple borders, then turn yellow to brown and eventually die prematurely. Lesions, which can be confused with those of Stemphylium, first appear on stems and needles in late spring and early summer, usually 6-7 weeks after the last harvest, and become progressively severe through the season. Premature death of ferns can reduce photosynthate and subsequent food storage in the crowns, resulting in yield reductions in the following year (Conway and Motes 1984; Cooperman *et al*. 1986). *C. asparagi* germinated readily in free water at a wide range of temperatures and in presence or absence of light (Cooperman and Jenkins 1986). The disease was first identified in Malaysia in 1986 and was considered the most serious disease of asparagus in the country (Saadaoui 1987).

C. asparagi overwinters in infected asparagus stems and asparagus debris. Survival of the fungus was reduced when asparagus debris was buried in the soil while it survived well on intact plants and in debris suspended above the surface (Cooperman *et al*. 1986). Lesions of the disease can be found on asparagus seedlings during harvest period. The ferns grow rapidly and appear disease free because initial infections are small discrete lesions on the secondary branches and chlorophyll's of the lower portions of the ferns. Lesion formation and defoliation are most prevalent at the base of ferns during the initial stages of the epidemic. As the asparagus plants develop, the ferns of adjacent plants overlap and produce a dense canopy that retains the moisture, creating a more favorable microclimate for spore germination and disease development (Cooperman *et al*. 1986). Fern growth in Oklahoma closes between rows during July and coincides with increased aerial conidial densities. Symptoms appear as a general browning of the lower fern by the first of August while ferns will be completely diseased by September (Conway and Motes 1984; Conway *et al* 1987).

In North Carolina the high incidence of the disease may be related to the debris that remains undecomposed or partially decomposed on the ground when the new shoots emerge in spring, and to the ability of the fungus to overwinter in the mild winter climate and to produce inoculum in the spring (Cooperman *et al*. 1986).

A collection of 43 asparagus commercial cultivars was screened for resistance to the disease, and all were found susceptible (Saadaoui 1987).

Management of fern residue before the harvest period in the spring and the protection of the fern with fungicides during the summer can increase the yield of asparagus (Conway *et al*. 1990). Management of the fern residue may also reduce inoculum of other foliar pathogens such as *Puccinia asparagi* and *Pleospora herbarum* (Conway *et al*. 1990).

For the disease control, the destruction of the plant debris and volunteer asparagus seedlings helps to reduce the primary fungus inoculum in spring. Additionally, suitable fungicides application according to weather conditions may provide integrated control (Conway *et al.* 1987).

All the fern diseases can contribute to early decline as secondary factors by weakening the asparagus plants, resulting in more *Fusarium* damages and shorter economic life of asparagus crops.

VIRUS DISEASES

Few viruses have been reported to infect asparagus worldwide, from which the most common are asparagus virus I (AV-I), asparagus II ilarvirus (AV-II), asparagus virus III (AV-III), cucumber mosaic virus (CMV) and tobacco streak virus (TSV), presented in **Table 1** (Elmer *et al*. 1996). Their incidence and severity in a crop are difficult to determine since most viruses are latent because they do not cause distinct symptoms of infection. Damages of these viruses are exhibited as reduction of the vigor, the productivity and quality of asparagus spears and increase of the asparagus susceptibility to other pathogens such as *Fusarium* spp.

AV-I is transmitted by aphids such as *Myzus persicae* Sulz. and *Aphis craccivora* Koch but it is not transmitted through asparagus seed (Elmer *et al*. 1996; Howell and Mink 1985). AV-I concentration in asparagus was different according to the type of sprouts (long or short) the season of testing and the genotype. To isolate virus free tissues for in vitro culture succeeded best from fast growing long sprouts in early spring. This material will be used for a virus free nuclear stock of asparagus cultivars (Kegler *et al*. 1999). The incidence of AV-I in fields has been reported to range from 20 to over 70% (Fallon *et al*. 1986). However repeated surveys indicated that most fields of Central Washington reached 100% infection by AV-I (Elmer 1996).

AV-II is a major viral disease of asparagus detected in all major asparagus growing areas around the world, which is transmitted through seed, sap and pollen (Jaspers and Falloon 1996; Jaspers and Pearson 1997). The AV-II is seed-transmitted and induces visible symptoms in both female and male asparagus plants (Mink and Uyeda 1977; Uyeda and Mink 1981; Fallon *et al*. 1986; Evans 1991). The infected plants produced smaller fern stalks and thinner spears (Jaspers *et al*. 1999). In a field trial at Lincoln University (New Zealand) the marketable yields from AV-II infected plants were reduced by 14-57% while the reject yields increased by 93-167%. In another experiment AV-II free plants gave 18-20% greater marketable yields than the infected plants (Jaspers and Falloon 1996). A sensitive test for AV-II detection was developed using the RT-PCR method to check and produce seeds that are free of the virus (Roose *et al*. 2002).

A potexvirus named AV-III was isolated from asparagus in Japan and was not transmitted by aphids and through seeds of infected asparagus. In Japan it was also reported the viruses AV-I and AV-II, but neither virus produced any distinct symptoms on asparagus (Fujisawa 1986).

CMV was also found later in Britain, occurring together with a virus serologically indistinguishable from AV-II (Phillips and Brunt 1985).

TSV spreads rapidly in the field presumably through thrips-mediated pollen transmission but the plants infected with TSV were also previously or simultaneously infected with AV-II (Elmer 1996).

Table 1 Viruses reported to occur naturally in asparagus. Adapted and updated from Elmer *et al.* 1996.

Virus name	Abbr.	Viral group	Occurence
Asparagus virus I	AV-I	Potyvirus	North America, Europe, Asia
Asparagus virus II	AV-II	Ilarvirus	North America, Europe, Asia, New Zealand
Asparagus virus III	AV-III	Potexvirus	Japan
Cucumber mosaic virus	CMV	Cucumovirus	Europe
Tobacco streak virus	TSV	Ilarvirus	North America, Europe

Asparagus viruses have been spread to commercial fields worldwide. Two viruses, named "asparagus stunt" and "asparagus latent" (probably identical with AV-II), were found in 1962 infecting asparagus in Denmark (Brunt and Paludan 1970). Three distinct viruses designated A, B and C were identified on asparagus in Washington. The A-type identified as TSV differentiated symptomatologically from two other TSV isolates obtained from legume crops (Mink and Uyeda 1977). The type B was considered in Europe as AV-I while C was found later to be very similar to AV-II. AV-I and AV-II were detected in Michigan but AV-II was widespread in asparagus fields, more in the older ones (Hartung *et al*. 1985; Evans and Stephens 1989a). In a survey of California and Delaware asparagus crops both AV-I and AV-II were found while TSV was not found. A virus very similar to AV-I has been isolated from asparagus plants in New Jersey, without obvious symptoms but it may be involved in the general decline of asparagus. Since then both AV-I and AV-II have been detected in asparagus, with AV-I occurring more frequently (Davis and Garrison 1984; Montasser and Davis 1987). AV-II and TSV have been found in asparagus plantings in Ontario, Canada (Wolyn and Stobbs 1991), while the three viruses AV-I, AV-II and TSV were detected in Mexico (Rafael *et al* 1994). In Poland AV-I, AV-II and CMV were isolated from infected asparagus plants where commercial asparagus seeds were infected with AV-II by 2-25%. However the AV-II decreased the quantity and quality of spears and the growth of stem brushes (Fiedorow 2000; Fiedorow *et al*. 2001). In Britain Arabis mosaic, strawberry latent ringspot and tomato black ring virus were isolated from asparagus plants, which were less vigorous than uninfected ones (Posnette 1969).

Asparagus plants infected with AV-I or AV-II alone are not severely affected after two years in the field but plants infected with both show serious decline and mortality in the second year (Yang 1979). Asparagus seedlings infected with AV-I or AV-II alone became more diseased when inoculated with *Fusarium oxysporum* f.sp. *asparagi* than did virus-free seedlings. When the seedlings were infected by both AV-I and AV-II they became more diseased when inoculated with *F. oxysporum* f.sp. *asparagi* than seedlings infected with either virus alone. However, the interactions of viruses exacerbate asparagus decline, probably by increasing plant susceptibility to *Fusarium* infection. Virus infection leads to an increased permeability of cell membranes of the root and an increased leakage of nutrients resulting in an increase in the inoculum level of the rhizosphere pathogens. Additionally, the roots of virus-infected asparagus have a reduced ability to synthesize lignin barriers resulting in an increased susceptibility to infection by *F. oxysporum* f.sp. *asparagi* and *F. proliferatum* (Evans and Stephens 1989). Root exudates, collected from AV-II infected asparagus, increased germination of *F. oxysporum* f.sp. *asparagi* microconidia than exudates of virus free plants (Evans and Stephens 1984). It is difficult to distinguish the effect of viruses to plant health from the effect of other biotic and non-biotic factors.

Evans and Stephens (1989b) believe that virus infection is one of several stress factors that predispose asparagus to *Fusarium* spp. infections. It is very important to use seeds and crowns for new virus-free plantations.

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