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 in asparagus causing latent infections.

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

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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; Elmet 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-
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 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 fungus causes 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 yield. The crop becomes less marketable economically and the field may be destroyed (van Bakel and Kerstens 1970; Elena and Kranias 1996). Additionally, asparagus 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 fumonisin B1 and moniliformin (both toxins that may pose a potential risk for human health) were present in some of the infected samples presented 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, 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 et al. 2001). The mtDNA RFLP analysis demonstrated 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 proliferation of the harvest period weakens the plants that become susceptible to infections. There are many soil, environmental and cultural factors (e.g. soil texture, water availability, soil pH, climate, disease pressure, drought, 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.

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Fusarium Pearls. The dipteran insects Hexomyza (Ophiomyia) simplex (Loew), and Delia (Hylyemia) 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 Hexasmyza 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 the disease. The FKFPLD data show that fungicide application on seedlings and 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 propagation, plants can 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). Applications of NaCl in the field and application in solution 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 disinfection by chemical fumigation or by steaming has been used before the establishment 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 Plapomatas 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 reinfection (Katan 1987; Elena and Tjamos 1997).

Biodisinfection 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 disinfections, 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 Batson 1938). The disease appeared after the harvest period and destroyed the bulbs. The disease 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 Asparagus sp. in Europe. 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 aciospores are produced in the same lesions 1-2 weeks later, whereas both pycnia and acia are formed on young shoots. The air causes new infections carrying the aciospores 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 acia 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 on which it is growing. The disease is usually more severe in the second and following years than in the first year following the infection. Reduced 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 varietes 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 acia 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 ascospore infections. The aeriation of the harvest period 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 (STEMPHYLUM LEAF SPOT, SUMMER BURNING)**

Purple spot of asparagus incited by *Stemphyllum* spp. (teleomorph *Pleospora herbarum* (Pers.) Rabenh. Ex Ces. & de Not.) is a significant problem for asparagus production. The species *S. vescarum* (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 *Stemphyllum* 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 before emerging from the planting beds. Penetration was significantly reduced when debris was incorporated while the internal tissue of the spear is not affected. No evidence of long-distance attraction to stomata was detected before emerging from the planting beds. Penetration was significantly reduced when debris was incorporated while the internal tissue of the spear is not affected.

The fungus overwinters primarily with teleomorph stage in 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 consistently 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 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 economic 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. (Reifsneider 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. asparagicolica* 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 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 ilarivirus (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 planting 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 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 (Roese 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. CMV was also found later in Britain, occurring together with AV-I and AV-II, and CMV also infected spears and asparagus leaves (Fujisawa 1986).

CMV was also found later in Britain, occurring together with the 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>
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<tr>
<th>Virus name</th>
<th>Abbr.</th>
<th>Viral group</th>
<th>Occurrence</th>
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<tr>
<td>Asparagus virus I</td>
<td>AV-I</td>
<td>Potyvirus</td>
<td>North America, Europe, Asia</td>
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<td>Asparagus virus II</td>
<td>AV-II</td>
<td>Ilarivirus</td>
<td>North America, Europe, Asia, New Zealand</td>
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<td>AV-III</td>
<td>Potexvirus</td>
<td>Japan</td>
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<td>Cucumber mosaic</td>
<td>CMV</td>
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<td>Europe</td>
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<tr>
<td>Tobacco streak virus</td>
<td>TSV</td>
<td>Ilarivirus</td>
<td>North America, Europe</td>
</tr>
</tbody>
</table>

Table 1 Viruses reported to occur naturally in asparagus. Adapted and updated from Elmer et al. 1996.
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 symptomologically from two other TSV isolates obtained from legume crops (Mink and Davis 1987). The type B was confused 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 (Yang 1979). Asparagus spears 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 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 involved.

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 seed and crowns for new virus-free plantations.

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