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Epidemiology and Management of the Diseases Causal to Asparagus Decline

Asparagus (*Asparagus officinalis* L.), a member of the Lily family, is one of the most important perennial vegetable crops grown in the United States. It has been found growing wild in many places, and its site of origin is not clear. However, a consensus suggests that it evolved somewhere in Asia and/or eastern regions of the Mediterranean seacoast (2). Asparagus was savored by the early Romans, who published methods for cultivating it in 161 B.C. (60). It was grown in England during the time of Christ and was introduced into America in the sixteenth century by French Huguenots (60).

Commercial production in the United States began in the late 1800s, and today 95% of U.S. asparagus is produced by California, Washington, and Michigan. Other states, such as Illinois, Maryland, Massachusetts, New Jersey, Ohio, and Oregon, also support small fresh market industries. In 1992, over 35,000 ha were harvested in the United States, producing in excess of 103,500 t, valued at $162 million. More than half of the produce was for fresh markets (1). The demand for fresh market asparagus has grown steadily since 1980 and should continue to increase.

Asparagus grown in the United States is cut in the spring after 1 to 3 years of plant establishment. Growers usually expect fields to remain productive and profitable for 10 to 20 years. However, asparagus fields can decline in vigor and stand density within 5 to 10 years due to a condition known as asparagus decline, also referred to as asparagus decline syndrome or asparagus stand decline. Asparagus decline was defined by Grogan and Kimble (31) as "a slow decline in the productivity of old asparagus plantings...to the point where the plantings become unprofitable to maintain." Damage from asparagus decline includes a reduction in spear size and number, and eventual death of the crown. Young plantings usually do not exhibit symptoms of asparagus decline in the first or second year of growth, but begin to show decline symptoms once seasonal harvest begins. Additional loss is incurred if abandoned asparagus fields are replanted with asparagus. In these plantings, stunting, chlorosis, wilt, and death appear and prevent stand establishment (31). The replant problem in asparagus has many similarities to asparagus decline. Grogan and Kimble (31) defined the replant problem as "the inability to establish productive plantings...(in fields)...where plantings have declined."

A number of factors contribute to asparagus decline. Abiotic factors, such as alleopathic residues (67), acidic soils (37), soil compaction, winter crown injury (57), and excessive spear cutting pressure (61), can weaken the plant and promote asparagus decline. Biotic factors, such as insects and weeds, also contribute to poor stand density and decline (13,57). However, most researchers are united in the opinion that several diseases, which act individually or in concert, are responsible for asparagus decline. Pathogens associated with asparagus decline in the United States are *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *asparagi* S.I. Cohen & Heald, *Gibberella fujikuroi* (Sawada) Ito G (anamorph *F. proliferatum* (T. Matsushima) Nirenberg), *Puccinia asparagi* De Candolle, *Pleospora herbarum* (Pers.:Fr.) Rabenh., (anamorph *Stemphylium vesicarium* Wallr.), *Cercospora asparagi* Sacc., and three viruses, asparagus virus I, asparagus virus II, and tobacco streak virus. These pathogens have played major roles in influencing the development of asparagus cultivars.

Another destructive root pathogen of asparagus is *Phytophthora megasperma* Drechs var. *sojae* Hildebrand (27). This pathogen, however, more frequently affects the initial establishment of asparagus plantings and not their decline. Hence, it

Fig. 1. Midseason foliar symptoms of Fusarium crown and root rot. (Courtesy J. LaMondia)

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decline, with special reference to their etiology, epidemiology, and strategies available for management.

Fusarium Crown and Root Rot

Fusarium crown and root rot was first described in Massachusetts in 1908 and has since become a limiting factor in all major asparagus producing areas in the United States. Consequently, it is the most frequently cited cause of asparagus decline. It has also been labeled Fusarium wilt and root rot (9), seedling blight (30), crown rot complex (22), and Fusarium stem and crown rot (47).

Typical symptoms of Fusarium crown and root rot are observed in midsummer and include a lemon yellowing of the ferns, which progresses basipetally toward the crown, causing the stalk to senesce prematurely (Fig. 1). Fusarium crown and root rot causes a complete destruction of feeder roots, collapse of the storage roots, and crown discoloration and rot (Fig. 2). Damage from Fusarium crown and root rot is usually more severe when plants are exposed to viral agents (24), high levels of alleopathic residues from the host (33), and insect damage (13).

In the United States, *F. o.* f. sp. *asparagi*, *F. proliferatum*, and *F. subglutinans* (9,13,47) cause Fusarium crown and root rot. *F. proliferatum* and *F. subglutinans* were previously classed as *F. moniliforme*, but have been taxonomically separated (54). The species composition associated with Fusarium crown and root rot can differ in other areas, such as the Netherlands, where *F. culmorum* is associated with the disease and *F. subglutinans* and *F. proliferatum* are conspicuously absent (4).

F. o. f. sp. *asparagi*, *F. proliferatum*, and *F. subglutinans* are isolated from all parts of the plant. However, *F. oxysporum* is more commonly found on young roots and tends to cause more damage in seedling nurseries than in production fields (9,30). *F. proliferatum* overtakes *F. oxysporum* as the dominant colonist in production fields and is commonly isolated from crowns (12,47), stems (50), flowers, seeds (29), and wounds made by feeding insects (13,19). *F. proliferatum* sporulates on the base of symptomatic stalks when humid conditions prevail (Fig. 3). *F. subglutinans* is isolated less frequently than the other *Fusarium* spp. and may be less important in inciting Fusarium crown and root rot.

F. o. f. sp. *asparagi* comprises a fungal group that includes 43 distinct vegetative compatibility groups (VCGs), some members of which are pathogenic on other crops (21). Because this pathogen lacks a known sexual cycle, the members in each VCG are presumably genetically isolated from members of other VCGs. This suggests the ability to cause disease on asparagus may have evolved in many different populations of *F. oxysporum* (21) and may explain why resident populations of *F. o. f.* sp. *asparagi* can be found in soil with no history of asparagus culture (34). Future studies that include DNA-based techniques may lend support to the hypothesis that *F. o. f.* sp. *asparagi* is polyphyletic.

The teleomorph of *F. proliferatum* is *G. fujikuroi*; however, the perithecia have never been observed on asparagus. Conidia are probably more important than ascospores as inoculum. *G. fujikuroi* has at least six distinct mating populations, termed "A" to "F" (52). When 250 isolates of *F. proliferatum* from Connecticut, Massachusetts, and Michigan were assigned to mating populations, 95% fell within the D population (19). Thus, these isolates probably comprise only one biological lineage. Using heterokaryon tests, over 110 isolates collected from these areas were assigned to over 20 VCGs. Three VCGs tended to predominate and were found in most of the plantings sampled (17). These findings suggested that certain VCGs may possess genetically linked traits that allow them to aggressively colonize asparagus in these plantings. Isolates from other locales may have a completely different VCG structure.

All of the *Fusarium* pathogens are soilborne, seedborne, or disseminated on transplants and nursery crowns. These characteristics tend to make them ubiquitous in asparagus plantings. Given these inescapable sources of inoculum, strategies for suppressing Fusarium crown and root rot have focused on management.

Fig. 2. Asparagus crown and roots showing symptoms of Fusarium crown and root rot. The rotted crown has vascular discoloration, collapsed storage roots, and a complete destruction of feeder roots.

Fig. 3. *Fusarium proliferatum* sporulating on the base of an asparagus stalk removed from a crown affected with Fusarium crown and root rot.

Fig. 4. Midseason response of asparagus to NaCl. The five asparagus plants to the left of red bag were treated with NaCl (560 kg/ha). The five asparagus plants to right were left untreated.

Research in the areas of genetic resistance, use of virus-free seed, and cultural practices led to improvements in managing Fusarium crown and root rot. Major advances have been made in cultivar selection with the advent of the all male hybrids. Male plants yield more, live longer, and do not produce volunteer seedlings as do females (14). Howard Ellison of Rutgers University first selected hermaphroditic male plants and produced a series of all male hybrids that have shown remarkable vigor and field resistance to Fusarium crown and root rot (14). Since the release of these hybrids, licensed nurseries have increased the germ plasm for commercial release and have also begun using routine virus-indexing techniques to ensure a virus-free stock. The predisposing effects of viral agents on Fusarium crown and root rot and asparagus decline are discussed below.

Cultural strategies that have been successful in managing Fusarium crown and root rot include using land never planted to asparagus. This practice avoids the high levels of alleopathic residues remaining from the previous declining crop and the high densities of insects and asparagus pathogens. Managing soil pH at neutrality, along with effective weed and insect control in the first couple years, has been shown to promote a vigorous crown and root system and decrease the rate of decline in later years (13,57).

However, suppressing Fusarium crown and root rot in fields planted to the older susceptible cultivars or to new cultivars grown under stressed conditions, has been difficult if not impossible. Recently, however, we documented success with the use of NaCl (common rock salt) to suppress Fusarium crown and root rot in asparagus fields.

The U.S. practice of applying rock salt to asparagus extends back to the Civil War. Nineteenth century agriculturists observed that the natural habitat of asparagus was along seacoasts, and may have erroneously believed that asparagus had a nutritional requirement for NaCl. Several sources in the late 1800s suggested applying rock salt at the exorbitant rate of "two quarts to the square yard" (over 120,000 kg/ha) (5,8,64). Alternatively, growers may have adopted the salting practice to control weeds (5,51). Regardless of the reasons, the practice of salting asparagus beds continued for many years because research showed it increased vigor and yield, especially in older fields (59,63). After synthetic herbicides were developed in the 1940s, the practice of salting asparagus beds was no longer encouraged. Coincident with this cultural change was an increase in the number of reports implicating Fusarium crown and root rot in asparagus decline. In fact, during the 1950s to 1960s, asparagus decline was responsible for the almost complete demise of the asparagus industry in many northeastern states.

In 1987, the hypothesis that salt could suppress disease was tested in Windsor, Connecticut, on replicated field plots containing 5-year-old asparagus crowns of the cultivar Mary Washington. Plots received broadcast treatments of NaCl (1,120 kg/ha) in the spring, and an equal number of plots was left untreated. The next year, marketable spear weights were 170% higher in NaCl-treated plots than in controls. In other experiments, with the cultivar Syn 4-56, the increased yield in NaCl-treated plots was later accompanied by an increase in the vigor and density of the fern canopy compared to untreated plots (Fig. 4). When a 2-year-old field of experimental plots was treated with 1,120 kg of NaCl annually for 5 years, yields became significantly higher than for untreated plots after the third year of harvest (Fig. 5). Although the ameliorating influence of NaCl appears to benefit plantings that are declining from Fusarium crown and root rot, it is not clear whether NaCl applications benefit fields where Fusarium crown and root rot is absent. Despite the long use of NaCl in asparagus culture, its mechanism for limiting Fusarium crown and root rot and asparagus decline is not understood. However, some evidence indicates that host resistance is affected (16,18). A series of greenhouse experiments with asparagus transplants demonstrated that NaCl did not affect the soil densities of the *Fusarium* pathogens but did reduce the number of *Fusarium* colonies that grew from the roots when cultured on selective media (16,18). The use of rock salt has recently gained attention among some commercial growers who are trying to extend the life of their asparagus plantings. However, caution must be stressed since long-term salt use can have deleterious effects on soil structure (7) and may damage other crops once these fields are taken out of asparagus.

Asparagus Rust

Asparagus rust, caused by *P. asparagi*, was first described in 1805 in France and was probably introduced to the eastern United States from Europe shortly before 1896 (55). In 1896, rust was first reported in New Jersey, Massachusetts, Connecticut, and Long Island, New York. Within 6 years, rust had spread to asparagus growing regions of California, Delaware, Iowa, Maryland, New York, North Dakota, Rhode Island, South Carolina, Vermont, and Canada (55). It is now present in all areas of North America and Europe where asparagus is grown.

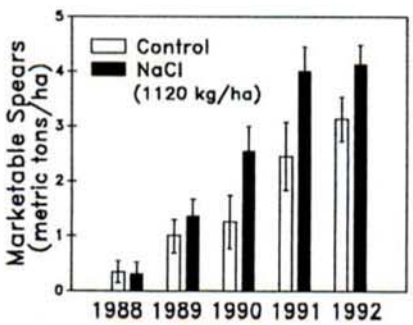


Fig. 5. Effect of NaCl on the yield of asparagus cultivar Mary Washington over a 5-year period. Plots were harvested for 2 weeks in 1988, for 3 weeks in 1989, and for 4 weeks in 1990 to 1992. Bars represent standard errors of the mean.

P. asparagi is an autoecious macrocyclic rust that produces pycnia, aecia, uredinia, and telia in succession. Following infection by basidiospores in early spring, pycnia appear on uncut spears and stems as oval, light green lesions about 6 × 19 mm in size. Aecia, which are light orange in color, develop in the same lesions 1 to 2 weeks later (Fig. 6). Aecia form on volunteer asparagus plants in young, uncut beds and on seedlings grown in commercial beds. Aeciospores are windblown, provide an early source of inoculum for rust epidemics, and give rise to uredinia.

The brownish red uredinia are most commonly observed on asparagus foliage in early to late summer (Fig. 7). Urediniospores are also windblown, and repeated infection of the uredinial stage may occur every 10 to 14 days, depending on moisture and temperature (40). Urediniospores, as well as basidiospores and aeciospores, require moisture for infection, and severe outbreaks of rust are dependent on wet weather from rain, dew, or sprinkler irrigation (48). Telia replace the uredinia in early fall and teliospores overwinter the fungus.

When rust is severe, asparagus foliage senesces prematurely and carbohydrate storage in the crown is reduced, resulting in less yield the following year (48). This

Fig. 6. Aecia of *Puccinia asparagi* on an asparagus stem.

stress factor exacerbates and accelerates asparagus decline. Both spear weight and number are reduced by rust on susceptible cultivars. The effects of rust are cumulative: yield reductions are usually greater after 2 years of infection than after 1 year. In a test in south central Washington, yield reduction for Mary Washington was 19% after 1 year of infection and 50% after the second consecutive year of infection. Similarly, the cultivar WSU-1 had a reduced yield of 23% the first year and 54% the second year. Rust caused little or no yield reduction in the partially resistant cultivars Jersey Giant and UC-157 (45). Severity of rust on the four cultivars during the two growing seasons prior to harvest is shown in Figure 8.

Management of rust requires the integration of resistant cultivars, sanitation practices, and in some locations or years, the timely application of fungicides. Rust resistance in asparagus is an important component of managing rust (36). Since the early 1900s, much effort has been directed toward identifying and developing resistant asparagus cultivars (3,36,46). In 1913, Norton (55) first noted that all asparagus cultivars became infected with rust but that some were less susceptible than others. Since then, rust resistance in asparagus has been recognized as quantitative rather than qualitative, resulting in differences in intensity of infection (36,38,55).

Rust resistance has behaved as a quantitatively inherited trait, in that progeny were continuously distributed in crosses between various combinations of resistant and susceptible parents (36,46). Transgressive segregation for resistance was not observed in populations from highly resistant parents, but it was observed in some populations from moderately resistant parents. An estimation of heritability was 55%, and progress has been made in selecting resistance in the field (46).

Rust resistance in asparagus cultivars currently in the United States has been a stable character for many years (3,43). The pollen parent used for developing Jersey Centennial, Jersey Giant, and other rust-resistant cultivars at Rutgers University was selected from a 15-year-old field of Mary Washington asparagus in 1960 (14,15). Moderately resistant selections have recently been made from Mary Washington (39), which was first released as a rust-resistant cultivar prior to 1919 (55). Although severe rust outbreaks occurred on selections of Mary Washington in Illinois in the 1940s and 1950s, it was not known if more aggressive strains of the pathogen had been selected or if the host had been unintentionally reselected for susceptibility (36).

Asparagus cultivars grown in the United States consist of heterogeneous populations. Cultivars are mostly either open-pollinated or clonal hybrids from heterozygous parents, and considerable heterogeneity for rust resistance exists within asparagus cultivars (39). In addition to the effect of rust resistance, the heterogeneous mixture of rust-resistant plants within resistant cultivars of asparagus may produce an effect similar to cultivar mixtures in restricting the spread of the pathogen in a crop (39,65). The heterogeneity within partially resistant asparagus cultivars may also exert less selection pressure on the pathogen population to overcome various components of partial resistance in the host, such as long latent periods and lower infection frequency, than in cultivars consisting of clones with partial resistance (39).

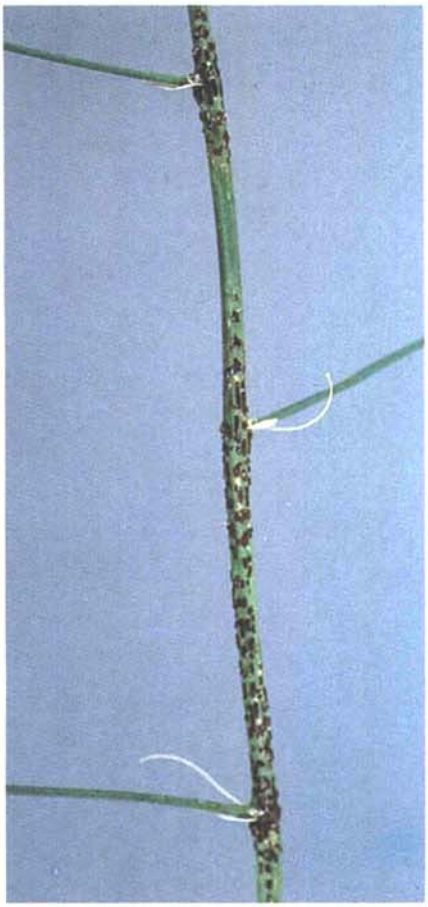


Fig. 7. Uredinia of *Puccinia asparagi* on an asparagus stem.

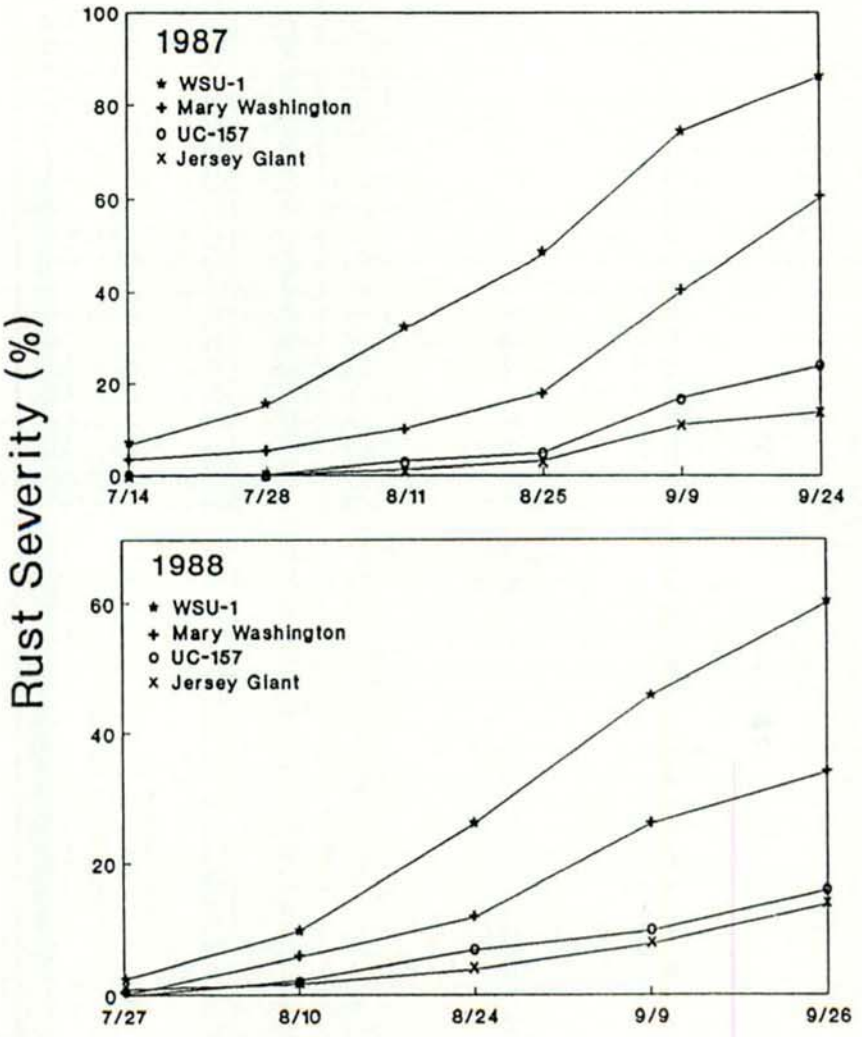


Fig. 8. Disease progress curves of four asparagus cultivars infected with *Puccinia asparagi* in the field in 1987 and 1988. The y-axis indicates percentage of foliage infected. Curves are the means of five replicates.

An important sanitation practice is to prevent the development of aecia. Cutting spears regularly until early summer eliminates aecia in commercial fields. However, aecia will still develop on young, nonharvested plants, such as volunteers, seedlings, and those in new beds. In newly established fields that are not harvested, rust severity can be especially high because of the availability of aeciospores and because young asparagus foliage is more susceptible to infection by basidiospores and urediniospores, and probably by aeciospores, than is older foliage (36,38,40).

An example of the effect of harvesting the spears on rust development was observed in the Columbia Basin of Washington State. Half of a 40-ha field of the cultivar Delmonte 361 contained 2-year-old asparagus crowns, and the other half contained 3-year-old asparagus crowns of the same cultivar. As mentioned before, asparagus is generally not cut until the third year to allow carbohydrate reserves to build up in the crowns. Spears from the 3-year-old asparagus were cut, and the 2-year-old plants were allowed to produce their ferns. Near the end of the harvest season, a rust-conducive rain shower occurred, and 3 weeks later, 60 to 80% of the ferns from the 2-year-old crowns were covered with aecia, whereas no aecia were found on the ferns from the harvested 3-year-old stand (Fig. 9).

Fungicides are typically applied on susceptible cultivars to suppress rust development, but may be required on resistant cultivars when high inoculum levels are present along with wet conditions. Disease thresholds need to be developed to schedule sprays. In Washington, Mancozeb is recommended for rust control if more than 1% of the foliage is infected by 31 July, if more than 5% of the foliage is infected by 10 September, or if 10% of the foliage contained uredinia by 30 September (42). However, to further refine spray decisions, the cultivar, current rust severity as determined by monitoring, length of time remaining in the growing season, and anticipated weather need to be considered.

Purple Spot

Purple spot, caused by *Pleospora herbarum* (anamorph *S. vesicarium*), was first reported in Michigan, California, New Zealand, Washington, and Oklahoma in the early to mid-1980s. However, the disease probably occurred previously, since field notes from the irrigated Agriculture Research and Extension Center in Prosser, Washington, show that J. D. Menzies isolated a *Stemphylium* sp. from asparagus with symptoms similar to those of purple spot near Toppenish, Washington, in 1946.

Purple spot contributes to asparagus decline by damaging the ferns during the growing season, causing defoliation of cladophylls (small needlelike branches). The destruction of photosynthetically active fern tissue reduces the potential photosynthate translocated to the crown and storage roots. Thus, smaller and fewer spears emerge the next year. It also directly reduces spear quality and marketability.

The disease appears on the ferns as tan to brown lesions with dark purple margins. The cladophylls drop off as lesions coalesce or when initial infections are severe. Symptoms on spears consist of small (1 to 2 mm), elliptical, slightly sunken purplish spots, which may result in the spears being rejected when they are graded for the fresh market (Fig. 10). Infections are more numerous and occur after shorter wetting durations on wounded than on nonwounded spears (44). Lesions on spears are frequently numerous on only one side of the spear (49), which may be a result of either wind movement and impaction of ascospores on the windward side of the spears or increased infection following injury from windblown sand (26,44).

Pseudothecia form on senescing ferns in the fall and winter. Ascospores mature by early spring in Washington State, and conidia are also produced on the previous year's fern growth. Infections occur through open stomata and wounds on current-season green asparagus tissue when the temperature is cool and moisture is present from rainfall or overhead irrigation (26). Volunteer asparagus seedlings in commercial fields that become infected during the harvest season are important as a substrate for inoculum increase. They also harbor inoculum from the harvest period, when spears are consistently removed, until the time when plants are allowed to produce their ferns.

Burial or removal of the previous year's fern growth reduces severity of purple spot on spears during harvest (41). Burial in late fall or late winter is equally effective in reducing the disease in Washington State. Burial does not necessarily decompose the pseudothecia before harvest, but it prevents ascospores and conidia from becoming airborne. Less soil erosion from wind and less water loss from soil in rainfed areas would be expected with a late winter or early spring incorporation of debris than with a late fall burial. However, the damage caused to crowns during debris incorporation may provide avenues for

Fig. 9. Field of asparagus cultivar Delmonte 361. Aecial development was prevented on the 3-year-old plants in the left section of field plots when spears were cut and removed for harvest. Aecia developed on 2-year-old plants in right section of field, which was not harvested.

Fig. 10. Purple spot lesions caused by *Pleospora herbarium* on asparagus shoots.

infection by the *Fusarium* pathogen. Cover crop mulches and wind barriers that reduce blowing sand should be investigated in sandy locations as an aid in disease management.

Cercospora Blight

Another important foliar disease that contributes to asparagus decline in warm, humid environments is Cercospora blight, caused by *C. asparagi*. It has been severe in eastern Oklahoma (10) and North Carolina (11), but does not cause appreciable damage in cool or dry climates.

Lesion development usually begins at the base of the fern and progresses upward. Lesions are small, oval, grayish tan in color with purple borders, and can be confused with those caused by *Pleospora herbarum*. Frequently, entire stems become blighted by Cercospora blight (Fig. 11). Blighted ferns turn yellow to brown and eventually die prematurely (11) (Fig. 12). Disease severity on ferns was inversely correlated with yield in Oklahoma (10).

In temperate climates where asparagus is harvested in the spring, lesions first appear on stems and cladophylls in late spring and early summer, usually 6 to 7 weeks after the last cutting (11). This occurs just before row closure and corresponds with increasing humidity within the crop canopy and a more favorable microclimate for sporulation and spore germination (11). The fungus overwinters in infected asparagus stems and debris, and conidia from the debris act as primary inoculum. Numbers of airborne conidia in North Carolina followed a diurnal pattern, with the greatest numbers between 1000 and 1300 h (11).

Integration of residue management, cultural practices, and fungicide applications is needed to adequately manage Cercospora blight, since there are no known sources of resistance. Burning or burial of the previous year's fern residue before the harvest season can delay the onset of disease development. However, fern growth from the previous year is frequently mowed in early spring to prepare for the harvest season. The residue is left on the soil surface to retain moisture and to reduce blowing sand (10). Soil mulches with nonhost material or other practices need to be developed to compensate for the loss of asparagus residues previously left on the soil surface when this material is removed.

Wide row spacing and orienting rows in the direction of prevailing winds will increase air movement in the canopy and facilitate a quicker drying of the foliage. Fungicides are also an important component of an integrated approach. Foliar applications of mancozeb and chlorothalonil reduce disease severity and can increase spear production the following year (10). In Oklahoma, three applications are recommended at 3-week intervals beginning prior to row closure. However, research is still needed on other cultural practices, such as drip and furrow irrigation, and on the timing of irrigation to reduce wet periods.

Fig. 11. Symptoms of Cercospora blight on a stem of asparagus. (Courtesy D. Sanders and C. Averre)

Fig. 12. Defoliation of the lower canopy of asparagus caused by Cercospora blight. (Courtesy D. Sanders and C. Averre)

Viruses Associated with Asparagus Decline

Although asparagus is a perennial plant, relatively few viruses have been found to occur naturally in asparagus plantings worldwide (Table 1). However, in recent years, the interactions of at least three viruses have been shown to exacerbate, if not directly induce, asparagus decline (66). These three viruses, asparagus virus I (AV-I), asparagus virus II (AV-II), and tobacco streak virus (TSV), appear to be widely distributed in asparagus commercial plantings (25,32,53).

Under field conditions, AV-I appears to infect only asparagus. It is transmitted by a wide variety of aphid species in the stylet-borne manner, but it is not transmitted through asparagus seed. Thus, new plantings always begin free of AV-1. In some areas of the United States, AV-1 is rarely found; but in other areas and many other countries, the incidence of AV-1 in established fields has been reported to range from a low of 20 to 30% to over 70% (25,35). However, these values were usually determined in single-year surveys using serological assay methods. Surveys conducted over a period of 15 years in Washington State have clearly demonstrated that none of the asparagus viruses are uniformly distributed throughout plants at any given time during the growing season. Consequently, surveys conducted at different times of the year give differing results. Repeated surveys by both serological and biological testing procedures indicate that most fields planted in the main asparagus-growing areas of central Washington reach approximately 100% infection by AV-1 after 7 to 10 years. Overwintering reservoirs of the virus include adjacent, infected plantings and nearby wild asparagus plants. Applications of insecticides to control aphid infestations in this area have not reduced the spread of AV-1 within or among fields (G. I. Mink, *unpublished data*).

AV-II appears to occur wherever asparagus is grown, primarily because the virus is readily transmitted through seed (62). The virus has a wide experimental host range. However, it has not been found to occur naturally in any plants except asparagus. The virus is transmitted not only through seed collected from infected asparagus plants, but also from seed produced on healthy plants following fertilization by AV-II-contaminated pollen. As a consequence, relatively low levels of AV-II-infected plants in or near fields where seed is harvested can result in significant levels of AV-II-contaminated seed. The

Table 1. Viruses reported to occur naturally in asparagus

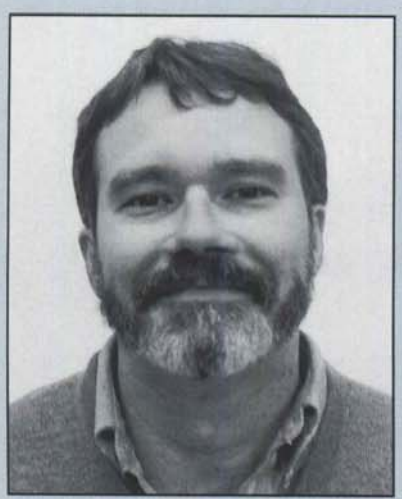
Virus name	Abbr.	Viral group	Occurrence	Ref.
Asparagus virus I	AV- I	Potyvirus	North America, Europe, Asia	35
Asparagus virus II	AV-II	Ilarvirus	North America, Europe, Asia	62
Asparagus virus III	AV-III	Potexvirus	Japan	28
Cucumber mosaic virus	CMV	Cucumovirus	United Kingdom	56
Tobacco streak virus	TSV	Ilarvirus	North America, Europe	6

incidence of AV-II in over 100 commercial seed lots tested in the late 1980s ranged between 5 and 60%, with an industry-wide average of 22% (G. I. Mink, *unpublished data*). In recent years, however, only trace amounts of the virus have been detected in seed lots provided by a few commercial seed producers.

Although AV-II is an ilarvirus and is readily found in association with asparagus pollen on foraging bees, and with flowers containing either one of two common thrips species, evidence is accumulating that plant-to-plant spread in field plots occurs only in the presence of TSV (J. Stark and G. I. Mink, *unpublished data*). If field spread of AV-II occurs alone in Washington State, it must do so only rarely. Spread of AV-II in a 5-year-old field planting in Michigan was believed to occur randomly and to spread very slowly (23). Consequently, use of virus-free seed lots can virtually eliminate the occurrence of AV-II in commercial asparagus fields

TSV, the second most common ilarvirus in asparagus, has not been detected in asparagus seed harvested from TSV-infected plants or in young seedlings grown from such seed. In contrast to AV-II, however, TSV spreads rapidly in the field, presumably through thrips-mediated pollen transmission. In detailed plot studies, 75% of the plants that became infected with TSV over a 4-year period had previously been infected with AV-II from seed. The remaining 25% of the new TSV infections were in plants that were simultaneously infected with AV-II. So far in Washington State, we have not found any asparagus plants infected with TSV alone. In greenhouse studies, we have been unable to infect virus-free asparagus seedlings by mechanical inoculation with infectious plant extracts or highly concentrated virus (G. I. Mink, *unpublished data*). Despite the fact that TSV can be detected in some local weed species, the probable sources for infecting new asparagus appear to be adjacent asparagus plantings. Because field spread of TSV in asparagus seems to require either the prior occurrence of AV-II in plants or its concurrent infection with TSV, control of AV-II through the use of virus-free seed should effectively control spread of TSV.

Although AV-I will eventually infect nearly all asparagus plants, field trials and greenhouse studies indicate that this virus alone has little or no effect on plant growth, yield of spears, or the long-term survival of commonly used asparagus cultivars (G. I. Mink, *unpublished data*). As already mentioned, the incidence of AV-II appears to be determined almost entirely by the level of infection in the seed lots used for planting. Infection by this virus alone appears to reduce plant growth somewhere between 10 and 20%. However, because of the difficulty of maintaining field-grown plants free of AV-I, the effects of AV-II alone on yield and long-term survival have not been determined.



Wade H. Elmer

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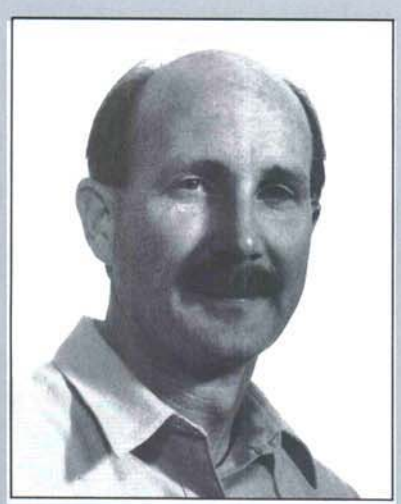

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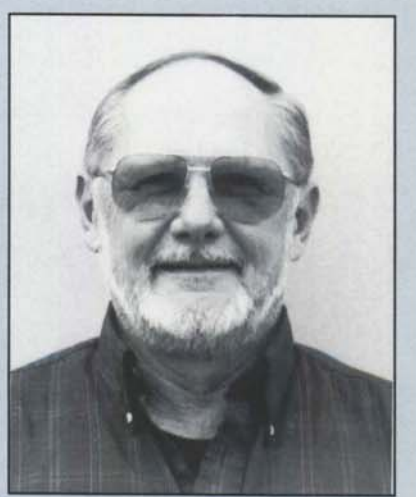

Gaylord I. Mink

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On the other hand, when plants become infected with both viruses, as is usually the case, plant vigor progressively declines over a period of years. Many double-infected plants begin to die in the second or third year of double infection.

Whether or not the double infection is the direct cause of plant death is not yet clear. In many cases, plant death has been attributed to Fusarium crown and root rot, since clear interactions have been observed (24). In other cases, death has been suspected to result from winter injury or other physiological stresses. So far, no definitive studies have defined specific viral agents involved in asparagus decline other than AV-I and AV-II. Again, the use of seed and crowns that are free of AV-II may be the key to the control of this disorder.

Future Outlook and Research Needs

Asparagus will undoubtedly continue to be one of the most important perennial vegetables grown in the United States, provided asparagus decline is effectively retarded and asparagus plantings remain profitable. In order to ensure that the industry is protected against increasing rates of decline, many aspects of the disorder require attention. Research aimed at suppressing asparagus decline falls into three broad areas: disease resistance, improved management, and host–parasite(s) interactions.

The resistance to Fusarium crown and root rot and rust that was found in the all male hybrid has been a tremendous asset to the industry, but commercial production would benefit with further improvements to increase resistance to these diseases. Moreover, genetic resistance to AV-I, AV-II, purple spot, and Cercospora blight has not been found. Since conventional breeding methods in asparagus are slow due to the low regenerative potential (58), the use of cell and protoplast culture with current genetic engineering techniques needs more development to shorten the time required to introduce new germ plasm to the industry (20,58).

The perennial nature of the asparagus plant requires that growers adopt a holistic approach to limit asparagus decline by managing insects, weeds, and diseases. Growers could design better spray programs if more information on damage–yield relationships for asparagus pests were available. Information on the economic thresholds for rust, purple spot, Cercospora blight, and insects, along with identification and registration of effective chemicals, is urgently needed. Additionally, a prescribed use of NaCl for suppressing Fusarium crown and root rot may also find a role in commercial management of asparagus decline once the actual mechanism by which it suppresses decline is deciphered. Information on the optimal rate and the best time of application is not known, and it is probable that soil type, cultivar and/or climate will affect the efficacy of NaCl use in asparagus culture. In addition, the long-term effects of NaCl use on other diseases, along with the soil and environmental impacts, need to be adequately addressed.

Many stimulating hypotheses relating to the pathological and physiological interactions in asparagus are attractive as unique research systems. Asparagus decline offers an economically important problem on a perennial monocot vegetable where soilborne *Fusarium* spp., viral agents, defoliating pathogens, and alleopathic residues can act individually or in concert to affect the ability of the plant to manufacture, store, and later mobilize carbohydrates. A greater understanding of these processes would lead to more effective management of asparagus crops, while at the same time, our understanding of replant and decline problems in other perennial crops may be advanced.

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