Grower Summary

FV 384a

Asparagus – Screening UK crops for virus infection

Final Report, 2012

Disclaimer

AHDB, operating through its HDC division seeks to ensure that the information contained within this document is accurate at the time of printing. No warranty is given in respect thereof and, to the maximum extent permitted by law the Agriculture and Horticulture Development Board accepts no liability for loss, damage or injury howsoever caused (including that caused by negligence) or suffered directly or indirectly in relation to information and opinions contained in or omitted from this document.

The results and conclusions in this report may be based on an investigation conducted over one year. Therefore, care must be taken with the interpretation of the results.

Use of pesticides

Only officially approved pesticides may be used in the UK. Approvals are normally granted only in relation to individual products and for specified uses. It is an offence to use non-approved products or to use approved products in a manner that does not comply with the statutory conditions of use, except where the crop or situation is the subject of an off-label extension of use.

Before using all pesticides check the approval status and conditions of use.

Read the label before use: use pesticides safely.

Further information

If you would like a copy of the full report, please email the HDC office (hdc@hdc.ahdb.org.uk), quoting your HDC number, alternatively contact the HDC at the address below.

HDC
Stoneleigh Park
Kenilworth
Warwickshire
CV8 2TL

Tel – 0247 669 2051

HDC is a division of the Agriculture and Horticulture Development Board.

Project Number:	FV 384a
Project Title:	Asparagus – Screening UK crops for virus infection
Project Leader:	Cathryn Lambourne
Contractor:	Stockbridge Technology Centre Ltd
Industry Representative:	Peter Knight – PK Consultancy
Report:	Final Report 2012
Publication Date:	12/06/2012
Previous report/(s):	None
Start Date:	01 September 2011
End Date:	29 February 2012
Project Cost:	£8,660.00

Headline

Second year trials have confirmed the presence of Asparagus virus 1 (AV-I) and *CMV* as the main viruses in UK asparagus crops. Older crops had more viruses than younger crops but virus presence did not appear to reduce yield nor affect crop performance.

Background

Commercial asparagus crops can remain in production for 10-15 years. However, UK growers have reported a noticeable decline in the yield and quality of their crops in recent years. This project was proposed by the Asparagus Growers' Association to investigate possible causes for this observed decline. A brief review of the literature relating to a phenomenon known as 'asparagus decline' (AD) was carried out in 2010 which identified this asymptomatic disorder e.g. no obvious foliar symptoms, as a possible cause of yield loss. An earlier HDC study (FV 151) investigated the use of salt application over a 4 year period following reports from elsewhere that soil applied salt was effective in reducing asparagus decline and increasing yields. The study reported a consistent trend towards benefits to the asparagus crops from a split dose salt application at the 0.5 to 1.0 t/ha rate. However, the differences observed were not always statistically significant ($p=0.05$). The report concluded that, as a long-term project, treatment differences could be expected to develop over a prolonged number of years. It is understood that whilst salt is still used by a few growers its popularity has declined in recent years due to difficulties associated with the availability of suitably formulated products.

An initial study was carried out in autumn 2010 to investigate the incidence and frequency of a range of 7 viruses in collected fern samples from UK crops (FV 384). The results showed a very high incidence of AV-I and CMV in the crops tested, with a number of other viruses being detected at lower frequencies. Multiple virus infections were common. Growers were asked to rate the performance of each crop as 'poor', 'moderate' or 'good' though the virus incidence results were found not to correlate well with recorded crop performance. Growers were left unsure as to what the consequences or impacts of the virus results were.

A second study was commissioned in 2011 to review the literature pertaining to 'asparagus decline' in greater depth, carry out additional virus testing and to try and determine the possible consequences of the virus infections via analysis of yield data from the crops sampled.

Summary

Literature Review

A thorough search of the scientific literature relating to ‘asparagus decline’ (AD) was carried out. The potential causes of AD were identified by a number of researchers as overuse of herbicides, overharvesting, production of allelopathic compounds by asparagus roots, fungal diseases of roots, crowns and foliage along with infection by viruses (Shelton & Lacy, 1980; Schofield, 1991).

Many researchers have demonstrated that young asparagus crops inoculated with multiple viruses show reduced vigour and yield. Knaflewski (2008) described the situation where viruses and fungi form a ‘disease complex’ and lead to AD. He also reported a correlation between crop age and virus incidence and confirmed that all asparagus viruses could be mechanically transmitted during harvest, stating that this is likely to be to main mechanism of virus infection. Pawlowski & Fiedorow (2002) also confirmed that virus could be spread via natural root grafting in the soil. Asparagus virus 2 (AV-II) alone can be transmitted by seed, work done by Falloon (1986), Bertacchini (1990) and Fiedorow (2002) detected varying levels of seed-borne infection ranging from 0-25%, although very much higher levels had been reported during the 80s.

Much work has been done investigating the role of *Fusarium* and *Phytophthora* infections in AD. It has been shown that virus infected plants are more susceptible to fungal infections by *Rhizoctonia solani*, *Fusarium oxysporum* and *Phytophthora* spp. and that this can be linked to seedling survival as well as crop yield and vigour.

Crop establishment problems following replanting of new crops into land previously affected by AD was also discussed by Grogan & Kimble (1959); Conroy (1975); Young (1984), and Huiskamp & Kanters (1989). This was initially attributed to high levels of *Fusarium* inoculum in the soil, but later work by Young &, Chou (1985) and, Hartung & Putnam (1986) suggested that asparagus tissues and plant residues present in the sites contained toxic substances that inhibited young asparagus plants.

There are no available methods to remove virus from infected crops, therefore prevention or limitation of infection are the only means available to growers. The use of virus-free seed or propagation material is the first step. Careful harvesting, crop monitoring and good geographical spread between plantings can also help.

Virus testing results for 2011

A total of 74 samples, collected from 37 crops were collected and tested for 4 viruses (AV-I, AV-II, CMV and TSV) in 2011. Samples were collected from sites spread between Somerset in the South and Angus in the North. AV-II was found to be present in **all** crops tested and should therefore be considered virtually ubiquitous in UK crops. CMV was found in 63% of samples, whilst AV-II was only detected in 27% and TSV in 7% of samples.

No firm correlations between cultivar susceptibility or location was observed. There was however a strong indication that crop age appeared to be significant, with crops between 5 and 9 years old being found to be most heavily infected with multiple viruses.

Re-testing of 10 crops that were originally tested in 2010, showed a worrying 43% increase in the presence of virus in these crops. However, perhaps surprising given information in the scientific literature, there did not appear to be a correlation between virus incidence and reported crop performance or yield. It is important to note though that yield data was only available on approximately half the tested crops, and generally only as total yield with little data provided regarding the weight of Class 1 and 2 spears. It is possible that more detailed yield data may have shown a significant reduction in crop quality in infected crops, particularly those with multiple infections.

Financial Benefits

No immediate financial benefits will result directly from this work. However, it is hoped that increased knowledge and understanding of the current science and the state of virus infections in UK crops should help growers to make more informed decisions regarding asparagus production in the future and thus mitigate any potential loss of marketable yield or crop quality from such virus infection.

Action Points

- Source new planting material from the most reputable and certified propagators to ensure virus-free healthy material.

- Consider new crop locations e.g. planting in land with a previous history of AD, or close to old or failing crops should be avoided if possible as this can lead to poor establishment and rapid infection with viruses and/or fungal pathogens.

- Keep good yield data records to permit individual monitoring of crops for any signs of failure.

- Consider additional surveying of UK crops on a regular basis to ensure there is a good understanding of the current state of virus infection in asparagus crops.

- Maintain good soil structure and try to reduce the risk of infection with fungal pathogens by the use of suitable fungicides and limit all stress-causing factors in crops as these can increase the risk of AD in crops.

- Insecticides currently in use by growers are used primarily for the control of asparagus beetle. It is worth considering though that as some viruses may be introduced and spread by vectors e.g. aphids or thrips then insecticide application may help slow down virus transmission in crops. They are unlikely to prevent initial infection though as some viruses are transmitted in a non-persistent manner (that is, they acquire and transmit the virus after a very short feeding time) and effective control with chemical application is much less effective in these situations. However, growers may perhaps need to consider seeking SOLAs for insecticides specifically for aphid control.

Literature Review

Asparagus production

Although enjoyed by the Romans and perhaps originally introduced to Britain at this time, asparagus production in the UK is not thought to have begun commercially until around 1540. Production in the UK is almost entirely of green asparagus, with low production levels of white and purple cultivars. Asparagus is also produced across Europe, Asia, Africa and North and South America, Australia and New Zealand. In 2005 China was the world's leading producer of asparagus producing almost 6 million tonnes, whilst Peru produced approximately 200 thousand tonnes and the US 90 thousand tonnes.

DEFRA report that the area of land under asparagus production in the UK in 2009/10 was approximately 1,580 hectares, with a production value in 2009 of approximately £14.5M. The majority of asparagus planting undertaken currently uses crowns produced in the Netherlands, although there is a renewed interest in using UK produced crown material.

Asparagus crowns are produced from seed either in modules or in open field situations. Once planted, crowns generally begin to produce a marketable crop when crowns are 2-3 years old. Some literature suggests that asparagus plantations should remain economically viable for up to 20 years, although a general productive period of 10-20 years is more accepted. In temperate climates, asparagus crops reach peak production after 5-8 years and slowly weaken thereafter so that the commercial life is c. 10-20 years (Johnston *et al.* 1979, Falloon & Tate, 1986; Bussell & Ellison, 1987). However, in crops around the world a disorder termed 'asparagus decline' has been reported which is reducing the economic viability of crops to 5-8 years in many instances.

Asparagus decline

AD was defined by Grogan and Kimble (1959) as "a slow decline in the productivity of old asparagus plantings … to the point where the plantings become unprofitable to maintain". Since this early work was undertaken much research into the causes, interactions, impact and possible control strategies has been undertaken by scientists around the world.

The symptoms of AD are described as

- Reduced number of fern stalks and thinner stems.
- Spears may shrivel or wilt at any stage of growth.
- Ferns are usually yellowed and stunted with reddish brown vascular discolouration and rusty flecks or lesions on the external layers of the lower portion of fern stalks.
- Reddish colouration may extend into crowns from fern sockets or from discoloured fleshy roots.
- Feeder roots may be absent or shrivelled and where present, show discoloured reddish areas at the junction with fleshy storage roots.
- Some of the fleshy roots show vascular discolouration and many collapse completely. Soft rot bacteria or secondary organisms are usually associated with such a collapse.

Factors which are thought to cause stress in asparagus plants and to contribute to decline include tillage, overuse of herbicides, overharvesting, production of allelopathic compounds by asparagus roots, fungal diseases of roots, crowns and foliage, as well as infection by viruses (Shelton & Lacy, 1980; Schofield, 1991).

Impacts of virus infection

Over the last 50 years there has been much research carried out which investigated the presence and impact of virus in asparagus. (*Asparagus officinalis* L.). Much of the work has been carried out in North America and Europe; both areas of high asparagus production. The first report of the presence of virus being linked to a lack of vigour in plants was made by Weissenfels & Schmelzer (1976).

Asparagus is host to several viruses, AV-I, AV-II, Asparagus virus 3 (AV-III), Cucumber mosaic virus (CMV), Tobacco Streak Virus (TSV), Arabis Mosaic virus (AMV) and Strawberry Latent Ring-spot Virus (SLRV). Distribution of the different viruses hosted by asparagus varies geographically. It has been shown that crowns may be infected with more than one virus. Often crops infected with virus show little or no obvious symptoms as noted by Knaflewski *et al* (2008).

In a study carried out by Yang (1979) in Washington USA, young plantlets were cloned from virus-free, AV-I infected, AV-II infected and AV-I + AV-II infected plants. The 4 groups of cloned plants were grown on for 6-8 months in a glasshouse, where no differences in growth were observed, before being planted in separate field sites and monitored for 16 months. Following this time the results shown in Table 2 were observed.

Table 2. Effects of asparagus virus 1 and asparagus virus 2 on asparagus plant survival and vigour after 16 months in the field (Yang, 1979)

Virus infection	No. of plants	No. of survivors	Height (cm)	No. of stems	Fresh weight (g)	Dry weight (g)
None	30	28	119.4	7.6	216.0	103.5
AV-1	35	31	112.5	5.0	157.5	72.0
AV-2	30	26	116.8	6.0	184.5	94.5
AV-1 + AV-2	28	16	94.7	4.6	81.0	31.5

(from Yang, 1979)

Yang noted that AV-I appeared to be slightly more debilitating than AV-II when single infections occurred. Also those plants infected with both viruses showed much greater growth retardation effects than single virus infections.

Studies by Jaspers & Falloon in 1999 showed that the incidence of AV-II in a trial crop in New Zealand reduced marketable yield significantly ($P<0.05$), whilst yield from non-AV-II infected plants increased year-on-year. The number of spears from infected plants which were ≤8mm diameter was significantly greater each year (by 109%, 88%, 220% 499% and 216% in 1992-96 respectively) from AV-II infected plants than from AV-II free plants. They also noted that there was a reduction in fern number and size. Robb (1984) showed that

plants with smaller fern stand accumulate less photosynthate, resulting in reduced carbohydrate reserves and reduced bud development in the following season. Cycles of reducing productivity develop, in which size and number of buds, spears and fern stalks decrease annually and this may be the a mechanism caused by the presence of AV-II.

Knaflewski *et al* (2008) carried out research at the University of Poznan in Poland and reported the results of his research and a summary of work undertaken by other researchers worldwide. He stated that "very often viruses form a complex together with fungi and they are undoubtedly one of the most important biotic factors of asparagus decline". He records that, as virus is stored in the crown material, once infected a plant remains infected for its whole life and becomes the source of infection for other plants. In his research they found a total of 11 viruses (the 8 most frequently detected viruses were investigated in the previous (2010) study) capable of infecting asparagus, with the most common being CMV, AV-I and AV-II in Europe, and AV-I being the most commonly detected virus in the USA. Hartung *et al*, (1985) reported a correlation between crop age and virus incidence, increasing from 35% in 0-2yr old crops to 69% in crops 16-29 years old (AV-II in Michigan USA).

Blaszczak and Weber (1979) found that it was not possible to prove that infection of asparagus by fungi increased the chance of virus infection. They stated that roots infected with fungi often limits virus transmission because most fungi secrete antiviral compounds. Also, necrotic and rotted tissues of plants cannot be infected by viruses.

Knaflewski (2008) states that whilst visible symptoms of virus are often difficult to see, plants are sometimes smaller, produce lower yields and are less vigorous. Yield reductions can range from 8 -30%, but where there is infection with a complex of 2 viruses, reduction by 70% has been observed. However, it is difficult to distinguish the effect of viruses from the effect of fungal infections and abiotic factors except in controlled experiments. The presence of virus in plants can also affect the quality of spears. Jaspers (1996) found, on average, in a two-year experiment, 100% more bent and 78% more thin spears harvested from plants infected with AV-II. The percentage of spears with open heads was also higher from the infected plants. Further work by Fiedorow (2001) and Jaspers (1996) showed that the longer the period that the plants are infected for, the greater the yield reduction. In the 1st year of harvest yield was reduced by 19% and in the 2nd year by 31%.

Virus Transmission

According to Knaflewski (2008), all viruses occurring in asparagus can be transmitted mechanically during harvest by inoculation with the sap of infected plants. This is probably the main mechanism of virus infection.

Studies carried out by Kegler *et al* (1991), Gasiorowska (1998) and Pawlowski (2002) demonstrated that 1/3 of the plants harvested with an infested knife became infected. Pawlowski & Fiedorow (2002) also found that virus can be spread by natural root grafting. Transmission of AV-II through roots in pot experiments did occur in 75% of the cases between 12-22 months.

Only AV-II is known to be transmitted by asparagus seed. This varies from 0-25% infection in some seed batches (Falloon *et al* 1986; Bertacchini *et al* 1990; Fiedorow *et al*, 2000). AV-II is seed-borne because it can be carried in pollen by bees during feeding and pollination. Jaspers & Falloon (1999) carried out studies in New Zealand to investigate other methods of transmission by which AV-II (and other viruses) could be spread in a crop. They planted seedlings of the cultivar UC157 in a field in 1990. Two AV-II infected plants were sown in each row, with 8 virus-free plants on either side (24 rows). Spears were harvested between 1992 and 1995. At the start of each row the cutting knife was disinfected by soaking for 1 minute in a 3% w/v solution of trisodium phosphate. Each row was cut in one direction only over the life of the trial. One block of 12 rows was cut in the direction of the prevailing wind and the other block was cut against the prevailing wind. This created 4 treatments whereby the effect of transmission by knife could be separated from other factors e.g. windblown pollen. They found that the incidence of AV-II infection in plants increased annually, but was significantly greater ($P<0.5$) in areas cut after the initial AV-II infector plants, than in areas cut before the infector plants. By 1995 AV-II incidence was 96.5% in areas cut after source plants compared to 26% in areas cut before. They found that spread caused by wind was less significant.

More recent studies (Anon.) which carried out testing on seed stock plants in the USA for AV-II found that the incidence of infection had reduced dramatically from previously reported levels in the 80s (100%) to only 25% in 2005. This is thought to be due to raising seed plants using tissue culturing of meristems and also because harvesting in Michigan is carried out by snapping rather than cutting.

AV-I and CMV can be transmitted in a non-persistent[1] way by aphids and AV-II and TSV also by pollen. CMV infection can come from other plants, e.g. cucumber, tomato, other vegetable crops and numerous weed species. TSV can occur on clover, bean, soybean and strawberry, among others and is transmitted mostly by thrips. Work by Fiedorow & Pawlowski (2002) has also shown that AV-II can be transmitted by spores of asparagus rust (*Puccinia asparagi*).

AV-III can be transmitted only mechanically with the sap of infected plants (Fujisawa, 1986; Brunt *et al*, 1996) and is still only reported in Japan.

Impacts of Virus & *Fusarium* infections and other fungal pathogens of asparagus

In all situations where AD is reported, isolations have confirmed that *Fusarium* infections are associated with the disorder (Grogan & Kimble 1959, Conroy 1975; Johnston *et al.,* 1979; Fantino, 1990). Work done by a number of researchers indicated that *Fusarium oxysporum* f. sp. *asparagi* and *F. moniliforme* are often considered to be part of the complex. *F. oxysporum* is a wilt pathogen that has been isolated from vascular root tissue, cortical root lesions, crown and stem lesions, and stem vascular tissue. *F. moniliforme* is less commonly isolated from roots but has been frequently isolated from stem and crown tissue where it often causes a more invasive, dry, brown rot than *F. oxysporum* (Schofield, 1991).

Work carried out by Evans & Stephens (1989) showed that AV-II or AV-I + AV-II infected plants were more easily infected *by Fusarium oxysporum* f. sp. *asparagi*. Pawlowski (2002) found the same to be true of *Rhizoctonia solani*. Plants infected with AV-I and AV-II had an 85% higher infection rate than virus-free plants. Joint fungal/viral infections were found to impact greatly on seedling survival rates (Pawlowski 2002 & 2004). Evans & Stephens (1989) and Pawlowski (2002 & 2004) postulated that virus infected roots produce more monosaccharides and other exudates and that this might attract fungi, allow easier germination of fungal spores and also that asparagus roots were less resistant to infection. Other mechanisms proposed for the increased fungal root rot induced by virus infection are that the inherent susceptibility of root tissue is increased by virus infection, and that increased leakage of nutrients from roots of virus-infected plants increases the inoculum potential of the fungi in the rhizosphere (Beute, 1970). In general it is considered that *F. oxysporum* f. sp. *asparagi* plays a more significant role in AD than *F. moniliforme*. Evans & Stevens (1989) conducted a number of studies using seed and seedlings inoculated with

[1] Non-persistent transmission involves the virus particles being carried on the stylet tip of the vector. The virus is then transmitted to the next plant visited by the vector. The virus particles do not enter the gut of the vector and cannot replicate within the vector. The vector only carries the viable virus (viruliferous) for a short period of time if further feeding on a suitable host does not occur.

combinations of AV-I, AV-II, and the two *Fusarium* species. They found that seedlings which were inoculated with AV-II and *F.oxysporum* f. sp. *asparagi* became significantly more diseased than AV-II infected seedlings inoculated with *F. moniliforme*. Seedlings infected with either virus and *F. oxysorum* f. sp. *asparagi* became more diseased than virus-free seedlings and also that seedlings doubly infected with both AV-I & AV-II were more diseased when inoculated with *F.oxysporum* f. sp. *asparagi* than seedlings with a single virus infection. They also carried out work on the exudates produced by virus infected seedlings. They found that seedlings infected with AV-II and grown in a liquid culture released 3 times more electrolytes in a 24-hour period than virus-free seedlings. When tested they found the seedlings released more glucose (3x), total carbohydrates (1.7x) and amino-acids (8x) than virus free plants. When aliquots of the root exudates from AV-II infected and healthy seedlings were added to water agar, significantly greater numbers of microconidia of *F.oxysporum* f. sp. *asparagi* and *F. moniliforme* germinated on the media supplemented with the exudate from the virus infected seedlings. They also found that in virus infected seedlings, the ability of the roots to produce lignin was significantly reduced. Lignin production in plants provides a method of 'walling-off' areas of damage or infection, so reducing the spread of pathogens. Reducing the plant's ability to do this would result in more severe levels of infection of plant tissues.

An additional problem associated with AD is that of replanting problems on land where decline symptoms have been prevalent. Reports from Asia, Australia, North America and Europe all indicate that replanting land with asparagus leads to establishment failure and reduced plant vigour; and where re-establishment is achieved, the economic life of the stand is shorter than with fields established on land that has not previously been in asparagus production (Grogan & Kimble 1959; Conroy, 1975; Young, 1984; Huiskamp & Kanters 1989). Initially researchers attributed the replanting problems to the presence of *Fusarium* inoculum in the soil, as many of the symptoms seen in failing plants were similar to those observed in AD and crown and stem rots caused by *Fusarium* (Grogan & Kimble 1989). Yang (1982) carried out work investigating the effect of soil fumigation on replant disease i.e. reducing *Fusarium* inoculum in the soil. He found that there were some initial benefits, but that the effect was short-lived and the problem persisted. Later work has shown that asparagus tissue and asparagus plant residues contain a toxic substance that is inhibitory to asparagus plants (Young & Chou 1985; Hartung & Putnam 1986; Hartung *et al* 1989).

The root pathogen *Phytophthora megasperma* var. *sojae* has also been noted as a cause of death of asparagus plants, particularly in wet conditions, although it is not often linked

directly to AD. It is, however, linked to problems during replanting when young seedlings can be particularly susceptible to infection by oospores in the soil (Falloon & Fraser, 1991).

Other fungal pathogens of asparagus have been reported as being associated with AD. Cooperman *et al* (1986) identified *Cercospora asparagi* (*Cercospora* blight), which can cause the development of lesions at the base of the fern which progress upward in warm and humid conditions. Blighted ferns turn yellow to brown and eventually die prematurely. Purple spot (caused by *Pleospora herbarum*) also contributed to AD by damaging ferns during the growing season, causing defoliation. The destruction of photosynthetically active fern tissue reduces the potential photosynthate translocated to the crown and storage roots (Elmer *et al*; 1996).

Prevention of virus infections in asparagus

Once infected with one or more viruses no treatments are available to remove the infection. Therefore prevention of infection is the only way that growers can control virus levels in crops. Clearly, the first step is to ensure that virus-free seed or crown material is used for new plantings. Seed producers should take steps to inhibit infection of seed crops, e.g. by using plants raised by meristem tissue culture rather than using already infected seed, and also by protecting seed-raising crops from potential insect vectors, by careful harvesting, and also by good geographical spread between older and new plantings.

Commercial growers should monitor crops for potential virus-carrying aphid and thrips vectors, although this is unlikely to stop insect transmission completely, it may reduce rate of spread through the crop, and a reduction in the incidence of virus reduces rate of spread by insects as there is a lower probability of them collecting virus-infected sap from one plant and transferring it to another. Growers should plant new crops as far as possible from older, possibly already virus-laden crops and should consider adopting less transmissive harvesting techniques, such as snap harvesting or using knife dips between plants, although these option may have impacts on rate of harvest. Work by Fiedorow & Pawlowski (2002) (Table 3) showed that very simple knife cleaning treatments could have a very significant effect on the transmission of AV-II in indicator plants (*Chenopodium murale*).

Table 3. Effect of knife treatment during spear cutting on the infection by Asparagus virus 2

Treatments	Number of local spots on the leaves of *Chenopodium murale* in replicates.				Spot number per leaf	
	I	II	III	IV	Mean	(%)
Not cleaned knife	12	17	14	15	14.5	100
Water cleaned knife	11	14	10	12	11.8	81.4
Milk soaked knife	0	0	0	0	0.0	0.0
Wiped knife	0	0	0	0	0.0	0.0

Source: Fiedorow & Pawlowski (2002)

Reducing AD

Virus infection makes plants more susceptible to attack by *Fusarium.* Reducing *Fusarium* in soils may be a method of reducing AD. Soil fumigation can help, but is not a long-term solution, giving only temporary effects in crops. Reducing or eliminating virus from seed and from cloned propagating material may help by reducing the susceptibility of plants to later *Fusarium* infections (Schofield, 1991). Studies have also shown that the use of various sodium chloride treatments of soils can be effective in reducing the infection rate of *Fusarium* in asparagus (Elmer, 1992), resulting in increased fresh weight of spears and increased fern growth. The practice of salting asparagus beds which was probably used from before 1860 to around the 1940's to control weeds and boost yields (Rudolph, 1927) appears to be a practice unique to the US and was discontinued after herbicides were developed in the 1940s. About this time the number of reports of *Fusarium* crown and root rots in asparagus began to increase (Cohen, 1941; Graham, 1955 & Grogan & Kimble, 1959).

The use of biological control of *Fusarium* spp. by inoculation with a non-pathogenic race of *Fusarium* has been investigated by Damicone & Manning (1982). Work carried out in Taiwan with soil amendments has shown that a mixture consisting of rice husks, bagasse, oyster shell, urea, potassium, nitrate, calcium, superphosphate, and mineral ash suppressed natural populations of *F. oxysporum* and reduced both the incidence and severity of the disease (Sun & Huang, 1985; Tu *et al* 1990). Scher & Baker (1980) also found a species of *Pseudomonas* that suppressed *Fusarium* infections in soils with high inoculum levels.

Further research into the development of *Fusarium* resistant cultivars of asparagus could also be an economically sensible approach to reducing AD.

Fusarium infection can also be reduced by the use of treated seed or fungicide applications to crowns and ferns.