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Project Report Summary For:

FV 213

Asparagus: a literature search on viruses

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FV 213

Asparagus: a literature search on viruses

Grower relevant section

Asparagus decline:

Asparagus decline syndrome is a reduction in profitable life of asparagus plantings and has been reported in many areas by commercial growers. Stress factors in asparagus production which are thought to contribute to this decline include tillage, allelopathic compounds (chemicals which are released by plants which inhibit the growth of nearby plants), over-harvesting, defoliation caused by fungal infections, virus infections and *Fusarium* crown and root rots. Asparagus decline is characterised by reducing productivity of asparagus fields, from five to eight years after establishment, resulting in crops that are uneconomical to maintain 10-15 years after establishment. The characteristic symptoms of decline include reduced size and number of spears and fern stalks. This report assesses the importance of the role of viruses in this problem and seeks to identify a control strategy for viruses, which in turn will contribute to the control of asparagus decline in the UK.

Viruses involved in asparagus decline:

Of the seven viruses which have been reported to infect asparagus, asparagus virus 1 (AV-1), asparagus virus 2 (AV-2) and tobacco streak virus (TSV) are the principal viruses associated with asparagus decline.

Detection and diagnosis of viruses:

The three principal viruses associated with asparagus decline can be readily detected using a serological test called ELISA. ELISA can be used to detect viruses in single infections and to differentiate viruses in mixed infections. These viruses can also be detected by host range studies but these are very time-consuming and are not suitable for differentiation of some viruses in mixed infections.

Virus transmissibility:

AV-1 is transmitted by several aphid species in a non-persistent (non-circulative) manner. The virus is also transmitted by mechanical inoculation but not by contact between plants and not in the seed or pollen. AV-1 has a relatively limited host range but there are several susceptible host species other than asparagus, some of which are important weed species (e.g. *Chenopodium* species). AV-1 virus occurs naturally in asparagus plants that often are infected with TSV and AV-2. Few, if any, symptoms are caused by AV-1 alone.

AV-2 is not transmitted by aphids and no other insect vector has been reported. It is transmitted by mechanical inoculation (physically rubbing a healthy plant with sap from an infected plant and by grafting infected plant tissue onto a healthy plant. AV-2 is also transmitted in seed; transmitted by pollen to the seed and transmitted by pollen to the pollinated plant. AV-2 has a relatively wide host range compared with AV-1. The incidence of AV-2 in new plantings is directly correlated with the level of seed infection of AV-2 in seed lots used for planting.

TSV is transmitted by thrips and is also transmitted by mechanical inoculation and grafting but not contact between plants. TSV is reported to be transmitted by seed (in *Chenopodium quinoa, Phaseolus vulgaris, Datura stramonium* to different degrees) but not in asparagus seed. TSV can be transmitted in association with pollen possibly by thrips to the pollinated plant, but not to seedlings. TSV has a wide range host range, compared with AV-1 and AV-2. In the USA, TSV was only found in crops which were already infected with AV-2 from seed so there appears to be a correlation, possibly due to changes in the preference of thrips for plants with AV-2 infections.

Impact of viruses on Asparagus yield:

The effect of infection by AV-1 and/or AV-2 infection has been assessed in Japan, New Zealand and the USA in a number of field experiments. Plants infected singly with AV-1 or AV-2 showed reduced productivity (30% and 10%) and reduced survival (11% and 13%), whereas plants infected with both viruses showed 70% reduced productivity and 43% reduced survival (Japan). In New Zealand AV-2-free plants gave significantly greater (18% and 30%) marketable yields than AV-2-infected plants in both 1992 and 1993 respectively. Other stress factors implicated in asparagus decline were excluded in the experiment.

The role of AV-2 in asparagus decline in New Zealand was further investigated by determining the effects of AV-2 infection on the productivity of asparagus plants in a field trial in New Zealand over a five-year period. Scientists in New Zealand also investigated whether movement of AV-2-infected sap on cutting knives contributed to the spread of AV-2 within an asparagus. Results showed that AV-2 infection caused mean marketable spear yields to be reduced by 14%, 28%, 20%, 48% and 57% and unmarketable yields to be increased by 93%, 105%, 207%, 352% and 167%, during harvest years 1-5 respectively. Total marketable yields from AV-2-free plants increased annually over the life of the trial, but marketable yields from AV-2-infected plants only increased up to the third year of harvest and decreased thereafter. Spread of AV-2 in asparagus crops was shown to be caused by movement of AV-2-infected sap on knives during spear harvest, although there was also the possibility of wind-assisted transmission.

In the United States similarities between asparagus decline and effects of AV-2 infection, in type and time-span of spear and fern symptoms, also suggest that AV-2 is a contributing factor in decline. In addition, spread of AV-2 on cutting knives may result in increasing numbers of AV-2-infected plants, contributing to the overall decline of an asparagus planting.

AV-2 is transmitted not only through seed collected from infected asparagus plants, but also from seed produced on healthy plants following fertilisation by AV-2-contaminated pollen. As a consequence, relatively low levels of AV-2-infected plants in or near fields where seed is harvested can result in significant levels of AV-2 contaminated seed. The incidence of AV-2 in over 100 commercial seed lots tested in the USA in the late 1980s ranged between 5 and 60%, with an industry-wide average of 22% (G I Mink, *unpublished data*). However, virus levels detected in seed lots provided by commercial seed producers there in recent years have been very low. The experience of growers in the USA is that the use of virus-tested seed lots can virtually eliminate the occurrence of AV-2 in commercial asparagus fields and potentially make a significant contribution to the control of asparagus decline.

Control strategy:

Research indicates that control of AV-2 is key to the control of viruses in asparagus, and therefore the virus component of asparagus decline. AV-2 is seed-transmitted and the level of AV-2 in new planting is correlated with levels of virus infection in the seed so it is crucial that seed is virus-tested and only seed lots that are free of virus are grown. Failing that, only seed with very low virus levels should be used. . The virus status of asparagus seed imported into the UK for commercial use is unknown. However, it is known that seed is currently not tested for the presence of virus and neither are plants that are used for seed production. A test for seed-transmitted virus should be developed and implemented as a matter of urgency so that seed can be virus-indexed before purchase, then if AV-2 was found to be a problem growers could work with seed companies to ensure that they produce only virus-tested seed. This strategy has worked well in the USA (Gaylord Mink, personal communication).

AV-2 is present in the embryo of the seed so cannot be removed by surface sterilisation of the seed. AV-2 can be destroyed by heating infected seed to temperatures above 32°C but as asparagus seed is rendered non-viable above 31°C, heat therapy of the seed is not an option for virus eradication. Disinfecting cutting tools between plants may help to control the spread of AV-1 and AV-2 but due to the intensive nature of asparagus harvesting this would not be practical or economic. However, once new virus-free plantations have been established it is important to keep them virus-free by segregating them from older, virus-infected plantings, disinfecting cutting tools between plantations or having a dedicated set of cutting tools for new plantations and disinfecting cultivating machinery between plantations.

The spread of AV-1 could be controlled by chemical control of aphid vectors, but this would be costly, environmentally sensitive and would have minimal benefits, as it would not control the most important virus, AV-2. Similarly, TSV could be controlled by chemical control of the thrips vector but this would have the same problems as control of aphids for minimal benefit. An alternative approach is to develop biological control of AV-2 using a mild strain of the virus to cross-protect against more severe strains.

Other possibilities for long-term control are to produce virus-free clonal material by meristem-tip culture and maintain it in isolated fields away from asparagus production or alternatively, select and breed new varieties with genetic resistance to AV-2.

Recommendations for future research and development work:

i) Survey UK asparagus crops of different ages over three successive seasons to confirm the identity of the three principal viruses involved in asparagus decline in the UK and determine the incidence of virus infection.

ii) Development of an ELISA-based test for sampling and detection of AV-2 in seed before purchase.

Sources of information:

Databases:
Agricola on-line database
BIDS on-line database
CABI database
VIDE database: Plant viruses on-line

Personal communications:
Victor Aveling Esq.
Professor Alan Brunt
Professor Gaylord Mink