03 Viruses 72-91 final 3/12/08 12:19 PM Page 72

CHAPTER 3

Virus and phytoplasma diseases

There are over 50 viruses to which *Solanum tuberosum* is susceptible. Eight of the most important are covered in this book; the rest are, fortunately, only of academic interest or limited in their distribution and significance. Viruses are sub-microscopic particles comprising a core of nucleic acid within a protein coat. They infect other organisms by invading host cells and using the cells' genetic material to make copies of themselves. Viruses, and the even smaller viroids, require specialized detection methods such as ELISA or molecular techniques. Viruses are spread either by contact or by insects and other vectors. Control relies on controlling vectors, limiting sources of virus, and utilizing host resistance.

Phytoplasmas cause virus-like symptoms in plants, and for many years such problems were attributed to viruses. However, in the 1960s, phytoplasmas were discovered in phloem tissues. They are actually related to bacteria, although they lack cell walls, and are curently placed in the class Mollicutes. Viroids also cause virus-like symptoms and, like viruses, hijack a cell's mechanisms to replicate themselves, but, lacking a protein coat, are a quite different entity.

There is a range of symptoms that viruses produce in potato haulm; some are characteristic of a particular virus, but many are general symptoms that may be ascribed to several different viruses. Thus in certification schemes, viruses are often visually classified in general terms such as 'mild mosaic' (**94**) or 'severe mosaic' (**95**) without knowing with certainty which virus is causing the symptom until further diagnostic tests are applied. To add confusion to diagnosis, different cultivars when infected by the same virus may exhibit different symptoms. In addition, where mixed virus infections occur, symptoms may be very different from those exhibited by either virus singly (**96, 97**). Most viruses associated with potatoes are described in the CAB descriptions of viruses (see reference list) and at various websites.

Although potato viruses rarely kill the plants they infect, they can be devastating in their impact on yield and quality and are considered a major cause of 'degeneration' of the potato crop. Since potatoes are vegetatively multiplied and most viruses can be transmitted to progeny tubers, the proportion of plants infected increases as a seed stock is multiplied. Seed production and certification schemes have been devised to ensure that foundation seed is free from viruses and, as far as possible, seed is kept free during multiplication. It is possible to eliminate virus from a stock by meristem tip culture, with or without heat therapy, and healthy foundation seed is produced in this way. In most seed producing countries, healthy cultures of potato varieties are maintained *in vitro* and from these new seed stocks are generated either as virus-tested stem cuttings or mini-tubers.

03 Viruses 72-91 final 3/12/08 12:19 PM Page 73

94 Mild mosaic symptoms.

95 Severe mosaic symptoms.

96 Mixed infections by *Potato virus X* and *Y*.

97 Mixed infections by *Potato virus X* and *Y*.

General virus references

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03 Viruses 72-91 final 3/12/08 12:19 PM Page 74

Aster yellows phytoplasma

ASTER YELLOWS

Symptoms

HAULM: Symptoms generally appear after mid-season and may resemble Fusarium or Verticillium wilts. Initially, the basal region of upper leaves rolls upward and the leaves often become distinctly purple, red, or yellow in colour (**98**). The rolling gradually spreads to leaves lower down the plant and these may become flaccid and wilted (**99**). New leaves fail to enlarge normally, and apical dominance is lost causing aerial tubers and/or shoots with swollen bases to grow from axillary buds. Lower stems may develop cortical necrosis and vascular discoloration. Some stems on plants may remain uninfected and symptomless.

ROOTS: Roots on infected plants do not produce symptoms.

STOLONS: Aster yellows symptoms have not been described in stolons.

TUBERS: Infected tubers are small, malformed, and sometimes soft and spongy. Infected tubers often fail to sprout but if they do, sprouts are long and spindly. If infected tubers are planted they either fail to establish a new plant or produce weak, spindly plants.

Status of the disease

The causal agent of aster yellows is a phytoplasma. It is not known whether other similar diseases described as stolbur, purple top, or marginal flavescence are in fact caused by the same or related phytoplasmas. Symptomology and epidemiological characteristics of these diseases overlap.

If infection levels are high, disease losses can be considerable. However, the phytoplasmas are transmitted to potato by leafhoppers from nearby weeds, and the efficiency of transmission is low. The disease is not propagated via seed because infected potatoes used for seed generally do not produce plants, or if they do they emerge late, are weak and stunted, and do not produce progeny tubers. Spread from infected tubers is not known to occur. Aster yellows and allied diseases are a significant problem in potato, mainly in areas which have relatively warm climates during the growing season – which favours the leafhopper vectors.

98 Rolling of upper leaflets and discoloration.

99 Symptoms spread to lower leaves, which become flaccid and wilting.

Life cycle and biology

The phytoplasmas are transmitted by several different species of leafhoppers and perhaps some other insects. In North America, the primary vector is *Macrosteles quadrilineatus*, the aster leafhopper. The potato leafhopper reproduces on potato but is not a vector. Weeds and other host crops are important reservoirs of the pathogen. Leafhoppers need to feed for several hours on infected plants in order to acquire the phytoplasma, and an incubation period of about two weeks

03 Viruses 72-91 final 3/12/08 12:19 PM Page 75

is required before the insects become 'phytoplasma-liferous'. Potatoes are not a preferred host for leafhoppers, and although they will feed on potato they do not breed on these plants.

Control

In geographic regions where the disease is a problem, control consists largely of suppressing weeds which may serve as a reservoir for the phytoplasma and control of leafhopper vectors. In arid regions, planting and harvesting may need to coincide with periods of other vegetative growth so leafhoppers are not attracted to potato as the only green vegetation available. Since infected tubers do not sprout adequately to produce progeny, the disease is not tuber borne.

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Potato leaf roll virus

POTATO LEAF ROLL

Symptoms

HAULM: Primary infection, arising from infection in the growing season, appears in the youngest leaves and mostly results in a pale discoloration and in-rolling of leaflets starting at the leaflet base. Some purple discoloration of affected leaflets may occur. Primary symptoms tend to occur only where infection of the plant occurs early in crop development or in hot climates. Secondary infection, where symptoms develop from infected tubers, is always more severe. Inward rolling of lower leaflets (**100**), extending ultimately to the upper leaves, is typical. The leaves become dry and brittle, and if touched the plant makes a characteristic rustling noise. Leaves are chlorotic and often show purple discoloration. Once symptoms appear, a necrosis of phloem tissue in the haulm is characteristic of the virus infection in the field. *Potato leaf roll virus* (PLRV) infected plants are usually stunted and erect (**101**) and produce normal-shaped, but small, tubers.

ROOTS AND STOLONS: No distinct symptoms are normally visible.

100 In-rolling of lower leaflets as a result of *Potato leaf roll virus* infection.

101 Stunted and erect plant infected by *Potato leaf roll virus*.