Synchytrium endobioticum

Introduction

Potato wart disease, caused by the fungus *Synchytrium endobioticum*, was once considered among the most destructive of potato diseases. In more recent times it has been well managed by a combination of strict quarantine measures and the increasing availability of resistant varieties. However, in locations where there are races of the pathogen for which resistant varieties do not exist, the disease can cause serious losses.

Despite the clear successes in the management of potato wart, the disease still retains a potential for significant economic impact arising from the fact that it is a notifiable disease. Potato cultivation in infested fields is prohibited indefinitely and there is a zero tolerance for wart disease in any seed potato scheme.

Identity

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Classification

- Kingdom : Fungi (Alexopoulos, et al., 1996)
- Phylum : Chytridiomycota
- Order : Chytridiales
- Family : Synchytriaceae
- Genus : *Synchytrium*
- Species : *endobioticum*
- Synonyms : *Chrysophlyctis endobiotica*
- Common names : Black wart of potato, galle (noir) verruqueuse de la pomme de terre, kartoffelkrebs, sarna verrugosa de la papa
- Role : Pest

Note on Classification

In view of recent and fundamental changes in the classification of organisms previously grouped in the "fungi", some clarifying notes are in order. The phylum, Chytridiomycota, contains the only fungi that produce motile cells in the course of their life cycles. With few exceptions motile zoospores and gametes have one posteriorly directed, whiplash flagellum.

The designation of orders is no longer based on morphological characteristics but on zoospore ultrastructure. In the future, DNA sequence characteristics are likely to be used. Members of the genus, *Synchytrium*, are endobiotic, holocarpic organisms having inoperculate sporangia. The thallus is colonial, dividing into several sporangia or gametangia that are enclosed within one membrane to form a sorus (Fig.2).

Signs & Symptoms

Potato wart appears on all underground parts other than the roots and it is not uncommon for the disease to escape detection until the tubers are lifted (Fig. 1). Especially under wet conditions, warts may occur on stems and even on foliage and plants may exhibit reduced

growth. Such tumours, or those that become exposed at or just above the soil line, will develop chlorophyll and, therefore, become green. Buds on stems, stolons and tubers are the centres of infection and abnormal growth, which culminate in the characteristic warts or tumours. Initially, the warts may be white to brownish, about the colour of the tuber. They vary from small protuberances (especially from early infections of the "eyes") to large multi-branched swellings that may engulf the whole tuber.

The warts are somewhat pulpy and softer than a tuber since, morphologically, they consist of distorted, proliferated branches and leaves mixed together in a mass of hyperplastic tissue. With age, they darken, eventually becoming black and decayed through the activity of secondary organisms.

Biology , Epidemiology & Morphology

The pathogen, a biotrophic parasite, survives from one season to the next as thick-walled, resting spores (35-80 µm in diameter) that enter the soil in a number of ways:

- During normal decay of warts
- In contaminated soil on farm tools and machinery and the footwear of workers
- In soil attached to plants and tubers grown in contaminated soil
- In manure from animals fed with infected tubers (the spores survive passage through the intestines of farm animals)
- In inconspicuous warts on seed tubers, sometimes even of resistant varieties.

Tubers of highly resistant varieties can be agents of dissemination. Resting spores are capable of surviving in soil for over 30 years and at depths of over 50 cm (Anon., 1980). Each consists of an outer, brittle membrane of disorganised host cells surrounding two inner membranes, the innermost of which is thin and hyaline.

Brief periods of saturated soil are required for the germination of resting spores and sporangia and for the release and motility of zoospores. At temperatures between 8 and 24 °C, about 200-300 motile spores are released from the resting spore (sporangium), move through films of water and locate epidermal cells of the host (Fig. 2, Q-H). The flagellum is shed and the zoospore penetrates the epidermis of the bud tissue of stolons or tubers. Infection is optimal in neutral to slightly acid soil conditions but occurs over a pH range of 3.9 to 8.5 (Weiss, 1925). Soon after infection, a uninucleate, intracellular thallus develops to a large size through hypertrophic growth. The thallus matures to form a prosorus having a thick, orange exospore and a thin endospore. The latter is extruded with the nucleus and cytoplasm to form a vesicle in which the nucleus divides to produce approximately 32 nuclei. Within the vesicle, walls develop to delimit from 4 to 9 sporangia, which comprise the sorus. Nuclear division within the sorus produces 200-300 zoospores.

The sporangia are set free by internal pressure, which ruptures the vesicle membrane and the host cell wall. The zoospores are then released in this quickly reproducing stage that multiplies the inoculum and the disease during the growing season (Fig. 2, B-I). A recently infected bud has many small protuberances that exhibit depressed centres. These are the sori containing sporangia. Zoospore discharges from these results in more centres, stimulation of more dormant buds, with the attendant hyperplasia.

The same type of structure that releases zoospores in the "repeating stage" of the disease may be induced to release motile gametes that resemble zoospores. This dual role of the motile

spore is controlled by the availability of water at a certain stage in the development of the fungus. A lack of water at a critical stage permits the maturation of gametes. When motile cells are released immediately after formation they behave as zoospores. Under conditions that induce gamete formation, there is fusion of pairs to form zygotes each of which infects a host cell, as does the zoospore, and develops within a hypertrophic cell to form the resting spore (Fig. 2, J-P).

Dispersal/vectors

The pathogen is disseminated through the faeces of farm animals, in inconspicuous warts on seed tubers and as zoospores, which swim through films of water in the soil.

Management

Quarantine

Management begins with the enactment of appropriate quarantine legislation. The general approach is to impose strict conditions (sometimes embargoes) on the entry of potatoes from any country where wart occurs. Quarantine restrictions can be applied to locations within countries, as has been done very successfully in the USA.

Scheduling

This involves identifying and officially noting any production area in which the disease has been reported. Further cultivation of the crop is prohibited in the scheduled field(s) and planting of only immune varieties is restricted to a designated safety zone around the scheduled fields. Because of the very long survival period of the pathogen's resting spores, there is hardly any likelihood of a field being descheduled.

Conditions on seed potato production

Potato wart is a notifiable disease. As such, there is a zero tolerance for the pathogen/disease in any seed-producing field. In addition, the infected area becomes scheduled and subject to the prescribed restrictions.

Resistant varieties

The development of resistant varieties is the only feasible control method, since no chemical measure has been found efficacious. The highly resistant sexual spore makes it difficult to exploit any weakness in the disease cycle and the environmental conditions favouring crop growth also favour wart development.

Resistance to wart in potato varieties was recognised quite early in the 20th Century. This led to the establishment of breeding programmes, especially in the UK, Scandinavia, Germany, Holland and Newfoundland, Canada, and the routine testing of varieties for field resistance in agronomic programmes.

Efforts at managing the destructive disease enjoyed early success, e.g., the containment and eradication of wart in limited areas in Pennsylvania, West Virginia and Maryland in the USA. There is now an extensive variety development programme at the International Potato Centre (CIP) situated in the cool, wet Peruvian Andes at over 3000m.

Several races of the pathogen have been detected and more are likely to exist, especially in Peru and Bolivia. However, the wide-ranging success of resistant varieties over many years suggests that the resistance is durable and/or the distribution of aggressive races of the fungus is limited.
It is recognised that host resistance is wide in range; the response of different varieties to single isolates of the fungus varies from resistant to highly susceptible. Moderately resistant varieties, which harbour inconspicuous warts, are important agents of pathogen dissemination. A considerable amount of resistance has been identified in various locations in Europe and South America. Several screening methods have been developed, with the accent on the most rapid detection of resistance. Methods include infecting 1) developing buds in tubers, 2) small sprouts 2-3 mm long and 3) small seedlings treated with growth regulators to induce the formation of stolons and tubers.
Molecular biological techniques are now being applied in the selection of resistant cultivars. Hehl et al., (1999) have reported the selection of resistant potato cultivars being facilitated by the use of markers closely linked with a resistance gene or by transferring a cloned gene for resistance into a susceptible cultivar. The method permits cloning the disease resistance locus based on map position and the establishment of PCR-based marker assays to assist selection of resistant genotypes.
Some of the older varieties with resistance to wart are still in use, especially where late blight can be effectively managed with fungicides. Examples are Kennebec, Katahdin, Russet Burbank and Sequoia. Recently developed varieties such as Asterix, Aurelia, Catriona, Kufri Bahar and Flourball possess resistance to late blight and other diseases. Most of the varieties developed in current programmes have resistance to potato wart, probably in recognition of its destructive potential.

Host Notes

Solanum tuberosum,(Solanum) esculentum, Solanum nigrum, other species of *Solanum, Hyoscyamus niger* (henbane), *Nicandra physaloides* (apple of Peru).

Distribution

Although the first reports of potato wart come from Europe, the disease is considered to be indigenous to South America and was transferred to Europe, probably with breeding material in the aftermath of the late blight epiphytotic of the 1840s (CIP, 1979). The disease was first reported from Hungary in 1895, but it was clearly widespread in Europe at that time. Economically, it was most important in Poland, Rumania and Switzerland as well as in parts of Russia.
It had also been reported from South Africa, China, Nepal, Newfoundland and the states of West Virginia and Maryland, U.S.A., from where it was practically eradicated within twenty years from its discovery. In South America, potato wart occurs in the highlands of Peru and Bolivia. More recently, the disease is reported to be only of local occurrence in Europe, including the UK (Anon, 2001), in kitchen gardens in Newfoundland (Hampson, 1986) and sporadically in the Southland Area, New Zealand (Anon., 2002).

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Web Resources -

http://www.maf.govt.nz/biosecurity/pests-diseases/plants/potato-wart/

http://www.defra.gov.uk/planth/pestnote/pwd.htm

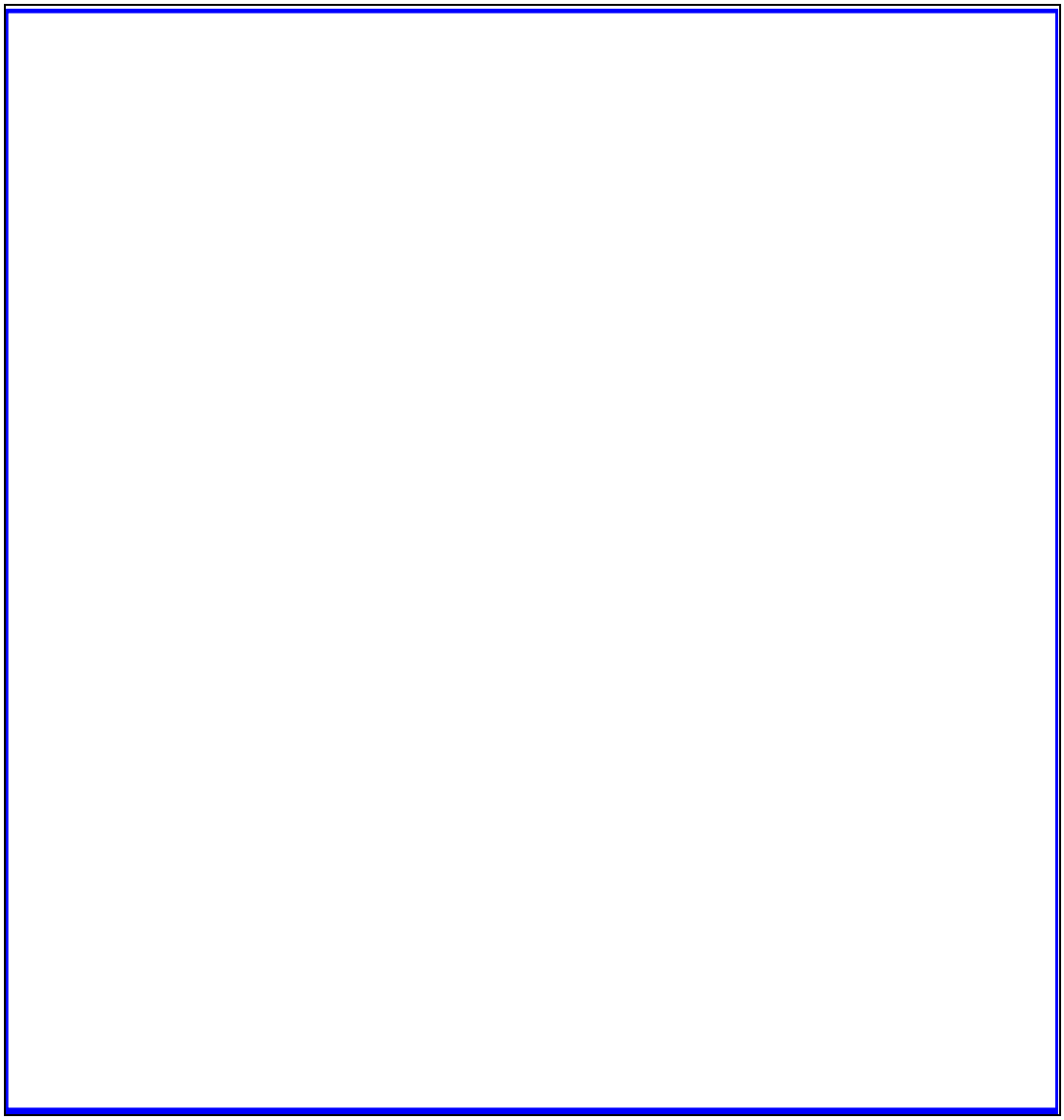

Fig.2 Potato Wart
Life cycle of ***Synchytrium endobioticum***