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Data Sheets on Quarantine Pests

Pear decline phytoplasma

IDENTITY

Name: Pear decline phytoplasma
Taxonomic position: Bacteria: Tenericutes: Mollicutes: Phytoplasmas
Common names: Pear decline, pear leaf curl, moria disease, Parry's disease (English)
Flétrissement du poirier, dépérissement du poirier (French)
Birnbaumsterben (German)
Decaimiento del peral (Spanish)
EPPO computer code: PRDXXX
EPPO A2 list: No. 95
EU Annex designation: I/A2

HOSTS

The main hosts are pears (*Pyrus* spp.). Pear trees on rootstocks of *P. pyrifolia* and *P. ussuriensis* (and especially scions of Williams, Beurré Hardy and Max Red Bartlett) are prone to tree collapse (quick decline). Mantecosa Precoz Morettini is less affected. Tolerant rootstocks, such as seedlings of *P. communis*, *P. betulifolia* and *P. calleryana*, are more likely to be affected by leaf curl (slow decline). The disease has also been observed on quinces and occasionally on trees grafted onto rootstocks of this species. Pear decline has been transmitted, using the insect vector, to the herbaceous host *Catharanthus roseus*. In the EPPO region, although pears are widely cultivated, the susceptible scion/rootstocks are not commonly grown. For additional information, see Refatti (1964).

GEOGRAPHICAL DISTRIBUTION

EPPO region: Recorded in Moldova. Widespread in Germany, Italy and Switzerland; locally established in Austria, Czech Republic, France, Moldova, Netherlands, Slovakia, Slovenia, Spain and Yugoslavia; unconfirmed records in Belgium and Russia. A decline of pears has been described in Greece but the causal factor has not been identified. Parry's disease, a slightly different disease occurring in the UK, is now accepted to be caused by the same phytoplasma.
Africa: Libya.
North America: Canada (first reported from British Columbia in 1948. Not reported from any other province). In the USA, the disease has been known since 1946 in the Pacific coast states (California, Oregon, Washington), and is now present in parts of the north-eastern states (Connecticut) and probably throughout commercial orchards in California.
EU: Present.

BIOLOGY

The pear psyllid *Cacopsylla pyricola*, which was introduced to the USA from Europe around 1832, not only transmits pear decline, but also causes damage *per se* by injecting phytotoxins in its saliva into leaves as it feeds. This vector is more common at high altitudes, being found between 600 and 1000 m in Switzerland. The psyllid does not migrate from pear, but overwinters in the adult stage in bark crevices. The phytoplasma can be acquired in a few hours and persists in the vector for at least 3 weeks. It has not been clearly demonstrated that the disease is transmitted by *C. pyri* and *C. pyrisuga* (also found on pear trees). Lemoine (1984), however, indicated that *C. pyri* could be a vector of pear decline.

The disease has been transmitted by grafting, but success rates are relatively low, up to 33%. In experiments with the pear psyllid, symptoms appear approximately 2 months after the infective insects have fed. Age of the tree and scion variety do not seem to influence the occurrence of pear decline.

Schneider (1962) recorded that abundant phytoplasmas were found in the narrow sieve tubes of the finer minor veinlets of decline-affected trees, but phytoplasmas were infrequent in sieve tubes of secondary phloem. It was proposed that toxic metabolites produced by either the phytoplasma or the host are translocated from the fine minor veins to the secondary sieve tubes where they cause necrosis.

For more information, see Jensen *et al*. (1964), Refatti (1967), Hibino & Schneider (1970), Bovey *et al*. (1972).

DETECTION AND IDENTIFICATION

Symptoms

Two types of decline symptoms are recognized: quick decline and slow decline or leaf curl. The degree to which decline symptoms are expressed is governed by the sensitivity of the rootstock and cultural practices, especially the level of psyllid control achieved.

Quick decline

Where the phloem at the bud union is sufficiently damaged to starve the roots during the growing season, fruits cease to develop and both fruits and leaves wilt rapidly. This may be followed by some leaf scorching and leaf death. Trees generally die within a few weeks.

Slow decline

There is a progressive weakening of the tree, which may fluctuate in severity. Terminal growth is reduced or may cease completely. Leaves are few, small, leathery and light-green, with slightly up-rolled margins; they become abnormally red in autumn and drop prematurely.

Although blossoming is heavy in the early stages of attack, later on, fewer flowers are produced, fruit set is reduced and fruit does not attain the normal size.

The reduced growth in successive seasons results in shoots appearing as tufts of leaves. Most of the feeder roots are killed, while larger roots may appear normal.

On removing the bark at the graft union, a brown line may be visible on the cambial face in the bark surface at or directly below the union, and vertically fluted ridges may also be seen. The coloration is not consistently recurrent and can fade during the growing season.

Morphology

The causal agent has not yet been cultured *in vitro*. However, the mycoplasma-like organisms found are predominantly filamentous, usually branched bodies bound with a trilaminar unit membrane but lacking a rigid cell wall (Seemüller, 1989).

Detection and inspection methods

It should be noted that symptoms similar to those of pear decline described above can be produced by other factors, such as rootstock-scion incompatibility, girdling, bad drainage, malnutrition, winter injury and drought. Care should be taken to eliminate these possible causes when diagnosing the disease, and observation should be confirmed by microscopy or transmission tests.

Frozen sections of root or stem samples, stained with a DNA-binding fluorochrome (e.g. DAPI) are observed in fluorescence microscopy. In the sieve tubes, small, brightly fluorescent particles appear (singly or in clusters). Best results are usually obtained with root samples, because phytoplasma populations in roots are less subject to seasonal variation.

Transmission tests can also be performed by grafting root or stem scions onto a suitable indicator (e.g. *Pyrus communis* cv. Precocious). On cv. Precocious, the leaves become slightly chlorotic with broadened and swollen veins. Then, first along the adaxial side of the midrib and later on the other framework veins, whitish corky tissue forms which later becomes brown. Simultaneously with development of vein symptoms, the leaves become leathery and brittle. Grafted plants are then kept in a glasshouse and observed until the end of the growing season of the year following grafting (Seemüller, 1989).

In the past the following method has been used successfully on susceptible trees, but is not reliable for tolerant cultivars. Microscopic observation of stained, radial sections of bark strips from the vicinity of the graft union of diseased trees will reveal excess phloem formation with associated small sieve tube elements and marked sieve tube necrosis. This becomes more pronounced as the season progresses (Schneider, 1962). A diagnostic histological feature is the union-specific, pre-senescent callousing of sieve plates. A starch test will show accumulation of starch above the graft union and its depletion or absence below in the roots.

For more information, see Schneider (1962), Refatti (1964; 1967), Bovey *et al*. (1972), Rallo (1973); an EPPO quarantine procedure on the detection of fruit-tree phytoplasmas is in preparation.

MEANS OF MOVEMENT AND DISPERSAL

Natural movement is by migration of the vector but this is generally only over a short distance, e.g., tree-to-tree or orchard-to-orchard movement, or via wild hosts. In international trade, the disease is liable to be carried in infected pear trees, scionwood and rootstock and possibly in the vector.

PEST SIGNIFICANCE

Economic impact

Considerable damage is caused by this pathogen; affected trees may die within a few years after infection or they may live for many years. Fruits, if produced, are small and few. In certain regions of the USA, pear production has been reduced by half. In Italy, during 1945-47, over 50 000 trees were destroyed. Pear decline causes some economic loss in all the EPPO countries in which it is present.

Control

Disease-free, tolerant budwood and rootstocks are of primary importance in control. In the USA, it is reported that transfusion treatment of trees with oxytetracycline hydrochloride solution is feasible in practice to treat infected trees.

Phytosanitary risk

Pear decline phytoplasma is listed as an A2 quarantine pest by EPPO (OEPP/EPPO, 1978) and is also of quarantine significance for COSAVE. This disease is confined primarily to varieties of *Pyrus communis* on the oriental rootstocks *P. pyrifolia* and *P. ussuriensis*, which are common in the USA and are less frequently found in EPPO countries. The psyllid vector is unlikely to cause widespread dissemination of pear decline since it does not migrate, and experience in the USA has shown that heavy infestations of the vector were present for 9 years before the disease appeared. Thus, in the present situation with a limited distribution of susceptible hosts, pear decline is unlikely to spread rapidly in the EPPO region.

PHYTOSANITARY MEASURES

EPPO recommends (OEPP/EPPO, 1990) that consignments of quince plants for planting, and of various named *Pyrus* spp. and their hybrids, should come from a source at which plants suspected of being infected by pear decline phytoplasma have been rooted out during the last three growing seasons; and that the consignment must have been found practically free from viruses and virus-like diseases during the last growing season. The EPPO certification scheme for fruit trees (OEPP/EPPO, 1991/1992) covers pear decline phytoplasma and should give a high security for phytoplasma-free planting material.

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