EPPO Datasheet: Plum pox virus

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IDENTITY

Preferred name: Plum pox virus
Other scientific names: PPV, Plum pox potyvirus, Prunus virus 7
Common names: pox of plum, sharka
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EPPO Categorization: A2 list
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EU Categorization: RNQP (Annex IV)

EPPO Code: PPV000

Notes on taxonomy and nomenclature

PPV is so far the only potyvirus known to infect temperate fruit trees. The potential existence of a serologically related virus in some Prunus materials of Asian origin has been reported (Hadidi & Levy, 1994). The existence and identity of this virus, tentatively named prunus latent potyvirus has however not been confirmed in further efforts. In particular, High-Throughput Sequencing of several Prunus sources initially reported to be infected by the prunus latent potyvirus or showing similar PPV-cross reactions to it failed to identify any potyvirus or PPV-like virus (Marais et al., 2016).

HOSTS

The main woody hosts are the species of Prunus grown for fruit production, including apricot (P. armeniaca), peach (P. persica) and plum (P. domestica and P. salicina). Almond trees (P. dulcis) can be infected by PPV but show few symptoms (Dallot et al., 1997, Damsteegt et al., 2007). Natural infection of P. cerasus and P. avium, attributed to the cherry adapted PPV-C strain has been sporadically observed in Europe (Kalashyan et al. 1994; Crescenzi et al., 1997). The recent identification of two other cherry-adapted strains (PPV-CR and CV, Glasa et al., 2013; Chirkov et al., 2018) also shows the epidemiological potential of these PPV strains in the cherry hosts.

Many Prunus species used as rootstock or as ornamentals are natural hosts of PPV, together with a range of wild Prunus species, including interspecific hybrids (James & Thompson, 2006; Damsteegt et al., 2007). PPV infects most wild or ornamental species of Prunus, such as P. besseyi, P. cerasifera, P. insititia, P. spinosa, P. tomentosa, serving as a potential reservoir and source of virus inoculum. Numerous annual cultivated plants or weeds have been shown to be experimental hosts of PPV (Virsek Marn et al., 2004; Llacer, 2006). However, as reports of natural infection of such herbaceous hosts have never been confirmed using two independent diagnostic techniques, and sequence information on the isolate(s) involved has never been provided, their host status is unconfirmed. In any case, natural transmission between such herbaceous plants and Prunus has never been demonstrated in nature, so that the epidemiological contribution of herbaceous hosts, if any, remains questionable.

Host list: Prunus americana, Prunus armeniaca, Prunus avium, Prunus besseyi, Prunus brigantina, Prunus cerasifera, Prunus cerasus, Prunus cundica, Prunus domestica subsp. insititia, Prunus domestica subsp. italica, Prunus domestica, Prunus dulcis, Prunus glandulosa, Prunus holosericea, Prunus incisa, Prunus japonica, Prunus laurocerasus, Prunus mahaleb, Prunus mandshurica, Prunus maritima, Prunus nune, Prunus nigra, Prunus persica, Prunus pumila, Prunus salicina, Prunus serotina, Prunus serrulata, Prunus sibirica, Prunus simonii, Prunus spinosa, Prunus tomentosa, Prunus triloba, Prunus virginiana, Prunus x blireana, Prunus x cistena, Prunus

GEOGRAPHICAL DISTRIBUTION
Typical shakara symptoms, caused by PPV (Atanasoff, 1932) were observed for the first time in plums in Eastern Europe (Bulgaria) around 1914. PPV subsequently spread, over most of the European continent and Mediterranean basin during the 20th century (Garcia & Cambra, 2007). PPV has also been reported from the Americas (Levy et al., 2000; Thompson et al., 2001; Herrera, 2013), from Asia (Maejima et al., 2010) and from Africa (Boulila et al., 2004). It is not yet officially reported from Oceania. In 2019, PPV was reported to be eradicated in the USA (USDA, 2019).

**EPPO Region:** Albania, Austria, Belarus, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Cyprus, Czech Republic, Denmark, France (mainland, Corse), Germany, Greece (mainland), Hungary, Israel, Italy (mainland, Sicilia), Jordan, Kazakhstan, Latvia, Lithuania, Luxembourg, Moldova, Montenegro, Netherlands, North Macedonia, Norway, Poland, Portugal (mainland, Azores), Romania, Russia (Central Russia, Southern Russia), Serbia, Slovakia, Slovenia, Spain (mainland), Switzerland, Tunisia, Turkey, Ukraine, United Kingdom (England), Uzbekistan

**Africa:** Egypt, Tunisia

**Asia:** China (Hunan), India (Himachal Pradesh), Iran, Israel, Japan (Honshu), Jordan, Kazakhstan, Korea, Republic, Pakistan, Syria, Uzbekistan

**North America:** Canada (Ontario)

**South America:** Argentina, Chile

**BIOLOGY**

Infected *Prunus* trees are the major source of inoculum. The virus is transmitted from them either by grafting and other vegetative multiplication techniques or non-persistently by aphid vectors (Ng & Falk, 2006; Moreno et al., 2009). *Aphis spiraecola, Phorodon humuli, Hyalopterus pruni and Myzus persicae* are the main vectors (Cambra & Vidal, 2017). Other aphids have also been shown to transmit the virus: *Aphis craccivora, A. fabae, A. gossypii, A. hederae, Brachycaudus cardui, B. helichrysi, B. persicae, Myzus cerasi, M. varians, Rhopalosiphum padi and Sitobion fragariae* (Labonne et al., 1995; Gildow et al., 2004).

The number of trees becoming infected in an orchard is directly related, in a given season, to the population level of winged aphids. These aphids probe or feed on infected leaves, then fly to other trees where they again probe or feed (Labonne & Quiot, 2006). Aphids can also acquire PPV from infected fruits (Labonne & Quiot, 2001). Analysing the spatial distribution of aphid-borne spread in eastern Spain, Gottwald et al. (1995) concluded that aphids do not spread the disease much to immediately adjacent trees, but to a few trees away. Experiments and modeling show that spread occurs generally within a few hundred meters with about 50% of transmission events occurring within 90 m of the source tree (Pleydell et al., 2018). The capacity for vector transmission can vary between viral isolates even
within the same strain (Dallot et al., 2003; Glasa et al., 2004). After inoculation of a Prunus tree, the incubation period may last several months and systemic spread may take several years. Accordingly, the virus may be distributed very irregularly in trees, possibly explaining the dynamic structure and heterogeneous nature of PPV population(s) in individual hosts (Jridi et al., 2006; Predaj’a et al., 2012). Seed or pollen transmission of PPV in Prunus has not been confirmed, and is unknown in practice (Glasa et al., 1999; Pasquini & Barba, 2006).

Various strains of PPV were originally distinguished (necrotic, intermediate, yellow) on the basis of symptoms obtained by inoculation of herbaceous indicator plants (Sutic et al., 1961). Then two isolates D (Dideron) and M (Markus), the former on apricot in France and the latter originally on peach in Greece, were serologically differentiated (Kerlan & Dunez, 1979). Further efforts led to the identification of these isolates as typifying two strains differing in serological and molecular properties (Candresse et al., 1998). Later sequencing efforts led to the recognition of further strains (Wetzel et al., 1991; Nemchinov et al., 1996; Glasa et al., 2004, Ulubas Serçe et al., 2009; James & Varga A, 2005; Palmisano et al., 2012; Glasa et al., 2013, Chirkov et al., 2018). Currently, a total of ten genetic strains are recognized for PPV (in the order of their discovery: D, M, EA, C, Rec, T, W, An, CR and CV). The three main strains, that have very wide geographical distributions, are PPV-M, D and Rec (Garcia et al., 2014). Some strains have particular biological/epidemiological features (e.g. cherry-adapted strains C, CR and CV) or a restricted geographical distribution (EA in Egypt, T in Turkey). However, due to a high intra-strain variability, most of strains do not show clear-cut epidemiological characteristics that would separate them from others (Sihelská et al., 2017). Several strains, including Rec and T have been shown to result from recombination events involving the D and M strains (Glasa et al., 2004; Glasa & Candresse, 2005; Hajizadeh et al., 2019).

DETECTION AND IDENTIFICATION

Symptoms

Symptoms may appear on leaves or fruits as a consequence of physiological, biochemical, proteomic, and transcriptional or post-transcriptional changes induced by viral infection (Clemente-Moreno et al., 2015). The symptoms are particularly clear on leaves at the beginning of the vegetation period: chlorotic spots, bands or rings, vein clearing, or even leaf deformation in peaches. Infected fruits show chlorotic spots or rings. Diseased plums and apricots may be deformed and show internal browning of the flesh; in apricot, the stones show characteristic pale rings or spots. Premature fruit dropping (up to 100%) can occur in the most susceptible cultivars (Sochor et al., 2012; Garcia et al., 2014). Symptoms of sharka depend very much on PPV isolate, locality, season, Prunus species and cultivar and plant organ (leaf or fruit) (Dosba et al., 1986).

Morphology

PPV has filamentous virus particles 750 nm long and 15 nm in diameter. It has a single-stranded RNA genome of ca 10 000 nucleotides, coding for a large polyprotein with a molecular weight of 3.5 x 10^6 Da. The genome encodes 10 mature proteins processed from the viral polyprotein by the action of three viral proteases. As for other potyviruses, transcriptional slippage allows the extension of an out of frame short open reading frame P3N-PIPO (Rodamilans et al., 2015).

Protein inclusions of the pinwheel type are present in the cytoplasm of infected cells. The full-length nucleotide sequences of a number of virus isolates belonging to all recognized strains have been determined (García et al., 2014). Genome function in PPV is now increasingly understood, and this virus is now a model for studies on the molecular biology of potyviruses (García et al., 2014; Rodamilans et al., 2019).

Detection and inspection methods

In spite of the irregular distribution of the virus in the tree, visual inspection may allow detection of symptoms in susceptible cultivars, especially during the period of active growth. Testing on susceptible indicators (peach GF305 or Prunus tomentosa) by chip-budding can produce symptoms in 6-8 weeks (Damsteegt et al.; 1997; Gentit, 2006). Mechanical inoculation on Chenopodium foetidum or Nicotiana benthamiana produces symptoms in 6-10 days but the inoculation efficiency from Prunus hosts is generally low (Sutic et al., 1961; Glasa & Candresse, 2005; Glasa et al., 2010).
Immunochemical methods, such as ELISA, have still an important role in the diagnostic of PPV (Šubr & Glasa, 2008; Cambra et al., 2011). A range of broad-spectrum or strain-specific antibodies are available (Cambra et al., 1994; Cambra et al., 2006a; Candresse et al., 2011), including monoclonal antibodies. Although all parts of the tree can be sampled for testing, the best detection results rely on the use of composite leaf samples from actively growing shoots taken in different parts of the canopy (Adams, 2008).

Molecular methods based on the amplification of specific parts of the PPV genome show a higher sensitivity than immunochemical methods (Lopez et al., 2003). Various modifications of RT-PCR in single or multiplex format have been developed both for the universal detection of all PPV isolates or for strain-specific detection (Olmos et al., 2002; Šubr et al., 2004).

An effective detection coupled with the possibility to differentiate PPV strains can be achieved using real-time RT-PCR (Varga & James, 2005; Capote et al., 2009; Fotiou et al., 2019). Isothermal amplification methods, such as LAMP (Varga & James, 2006; Hadersdorfer et al., 2011) have also been developed for a simple and direct use in the field. Validated international protocols for detection and characterization of PPV are available (EPPO, 2004, IPPC-FAO, 2012).

PATHWAYS FOR MOVEMENT

The distribution of the disease appears to be at random in orchards. The virus is introduced as a consequence of aphid transmission or of the use of infected planting material. After 2-3 years, infection begins to spread from the first infected trees. Graft transmission can contribute significantly to spread in infected areas if certified virus-free material is not used. Movement of the virus between areas or countries is most often linked to the use of uncertified plants for planting (Rimbaud et al., 2015a, b).

PEST SIGNIFICANCE

Economic impact

The importance of sharka disease on the European stone-fruit production has been reviewed by Cambra et al. (2006b). The disease incidence is particularly high in the fruit-producing areas of central and eastern Europe. Virus infection can lead to considerable yield losses, reaching 100%. European plums may show premature fruit drop, while Japanese plums and peaches show ring-spotting on fruit, and apricots show serious fruit deformation.

Control

There is no anti-virus treatment available to control sharka disease in orchards. There are, however, considerable differences in susceptibility between the cultivars available for use in countries where infection is widespread (Kegler et al., 1998; Martínez-Gómez et al., 2000). However, the frequent plantation of tolerant Prunus cultivars (their fruits remaining generally symptomless in case of infection) has probably contributed to the further spread of PPV in these countries (Glasa et al., 2004). Biological control by inoculation of trees with hypo-aggressive strains has not proved as successful in the field as under controlled conditions (Kerlan et al., 1980) and is not considered a realistic preventative option. Other effective control methods are the production and use of healthy plants for planting within a certification system, and the eradication of diseased trees or orchards to reduce inoculum pressure (Rimbaud et al., 2015a). As for other potyviruses, the control of aphid vectors by regular treatment with aphicides or mineral oils shows only limited effectiveness, with the possible exception of nurseries where some protection has been recorded (Vidal et al., 2013). Such methods are used to contain PPV in several countries (e.g. France, Italy). EPPO recommends a certification scheme for fruit trees, which takes into account PPV (EPPO, 1991/1992). Resistance to PPV shows some promise, whether by traditional breeding or by transgenic methods. The hypersensitive response in plums, resulting in localized cell death, has been found to be an effective resistance mechanism against PPV (Hartmann, 1998). Apricot varieties resistant to the PPV-D strain are now extensively planted in some areas of Spain. While progress has been obtained in plum and apricot, the development of resistant peach varieties has remained a challenge due to the paucity of resistance sources. Biotechnology has also contributed
with the development of the transgenic plum cultivar Honeysweet which shows a high, broad spectrum resistance (Scorza et al., 2016).

**Phytosanitary risk**

PPV is included in the EPPO A2 list of pests recommended for regulation as quarantine pests. It is a quarantine pest for the European Union and many other EPPO member countries. It is also of regulatory interest to other Regional Plant Protection Organizations (e.g. COSAVE, IAPSC and NAPPO).

In the EPPO region, PPV presents a major risk to apricot, plum and peach in many countries where it is still absent or very localized. In addition, its presence in a country creates difficulties for export of certified planting material.

**PHYTOSANITARY MEASURES**

In order to prevent entry or spread of PPV, all imported host material (except seeds) should come from a place of production subject to growing-season inspection (EPPO, 2016). If the virus is present in the exporting country, this inspection should also concern the immediate vicinity of the place of production, and the material should derive from tested mother plants. Material produced following the EPPO certification scheme for virus-free fruit trees would satisfy these requirements (EPPO, 1991/1992).

Measures can effectively be taken to prevent spread of PPV from foci of infection and even to eradicate it. These include planting non-host plants in infected areas, using tolerant or resistant cultivars, controlling the vectors and destroying all diseased trees.

**REFERENCES**


Glasa M, Hříšovský I, Kůdela O (1999) Evidence for non-transmission of *Plum pox virus* by seed in infected plum


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How to cite this datasheet?


Datosheet history

This datasheet was first published in the EPPO Bulletin in 1983 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997, as well as in 2019. It is now maintained in an electronic format in the EPPO Global Database. The sections on 'Identity', 'Hosts', and 'Geographical distribution' are automatically updated from the database. For other sections, the date of last revision is indicated on the right.
