

# EPPO Datasheet: *Trichovirus persicae*

Last updated: 2023-10-26

## IDENTITY

**Preferred name:** *Trichovirus persicae*

**Taxonomic position:** Viruses and viroids: Riboviria: Orthornavirae:

Kitrinoviricota: Alsuviricetes: Tymovirales: Betaflexiviridae

**Other scientific names:** *PcMV*, *Peach American mosaic virus*, *Peach mosaic virus (American)*, *Peach mosaic virus*, *Peach virus 6*, *Prunus virus 5*

**Common names:** American mosaic of peach, mosaic of peach

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**EPPO Categorization:** A1 list

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**EU Categorization:** Quarantine pest ((EU) 2019/2072 Annex II A)

**EPPO Code:** PCMV00

## Notes on taxonomy and nomenclature

Peach mosaic disease was first described in the USA in the early 1930s (Hutchins, 1932) and was later shown to be transmitted by an eriophyid mite (Keifer & Wilson, 1955), but the causal agent remained elusive for a long period.

Much confusion occurred in the 1980s with the observation of some peach cultivars imported to Europe from North America and affected by a mosaic disease. Peach latent mosaic viroid (PLMVd) was isolated, identified and named by European scientists from this material (Desvignes, 1986; Desvignes *et al.*, 1988; Flores & Llácer, 1988; Flores *et al.*, 1990). The disease called peach yellow mosaic in Japan was also shown to be caused by PLMVd, together with the peach blotch and peach calico diseases, and it was realized that different isolates of PLMVd can cause widely different symptoms in peach (Flores *et al.*, 2011). Because of the similarities in symptomatology and host range (but not in transmission) between the mosaic disease caused by PLMVd and the peach mosaic disease described in the USA, it was concluded that PLMVd was the pathogen causing peach mosaic in North America (Flores *et al.*, 2011). However, it has now been clearly demonstrated that the causal agent of eriophyid-transmitted peach mosaic disease is not PLMVd but a Trichovirus which was named peach mosaic virus (PcMV) (James *et al.*, 2006; Larsen & James, 2011). The two agents are therefore clearly distinct and have very different transmission. However, since the symptoms they cause can sometimes be similar and as PLMVd has been shown to occur in North American peaches, it is frequently difficult to interpret some old publications and decide if the agent involved at that time was PcMV or PLMVd.

Further complicating things, a second Trichovirus close to PcMV but distinct from it, peach virus M (PeVM; de la Torre *et al.*, 2019) has recently been described in Mexico and likely has the same transmission by eriophyid mites as PcMV. It is thus possible that some old reports describing the presence of PcMV in Mexico might in fact refer to PeVM and not PcMV.

## HOSTS

Peach mosaic virus has only been identified in natural infection in *Prunus* spp.: peach (*Prunus persica*), almond (*P. dulcis*), apricot (*P. armeniaca*), European plum (*P. domestica*) and a range of other *Prunus* species (Cochran & Pine, 1958; Larsen & James, 2011). The virus has also been experimentally transmitted to *P. besseyi*, *P. japonica* and *P. subcordata* (Cochran & Pine, 1958) and to a few herbaceous plants: *Chenopodium amaranticolor*, *C. quinoa* & *Nicotiana occidentalis* (Larsen & James 2011).

Cherries and some other *Prunus* species have been shown not to host the virus: *P. avium*, *P. cerasus*, *P. emarginata*, *P. fenziiana*, *P. ilicifolia*, *P. mahaleb*, *P. pennsylvanica*, *P. pumila*, *P. serotina* and *P. virginiana* (Cochran & Pine, 1958; Larsen & James, 2011).

**Host list:**

*Prunus angustifolia*, *Prunus armeniaca*, *Prunus bokhariensis*, *Prunus davidiana*, *Prunus domestica*, *Prunus dulcis*, *Prunus hortulana*, *Prunus maritima*, *Prunus mexicana*, *Prunus mume*, *Prunus munsoniana*, *Prunus persica*, *Prunus reverchonii*, *Prunus salicina*, *Prunus tangutica*, *Prunus tomentosa*, *Prunus umbellata*

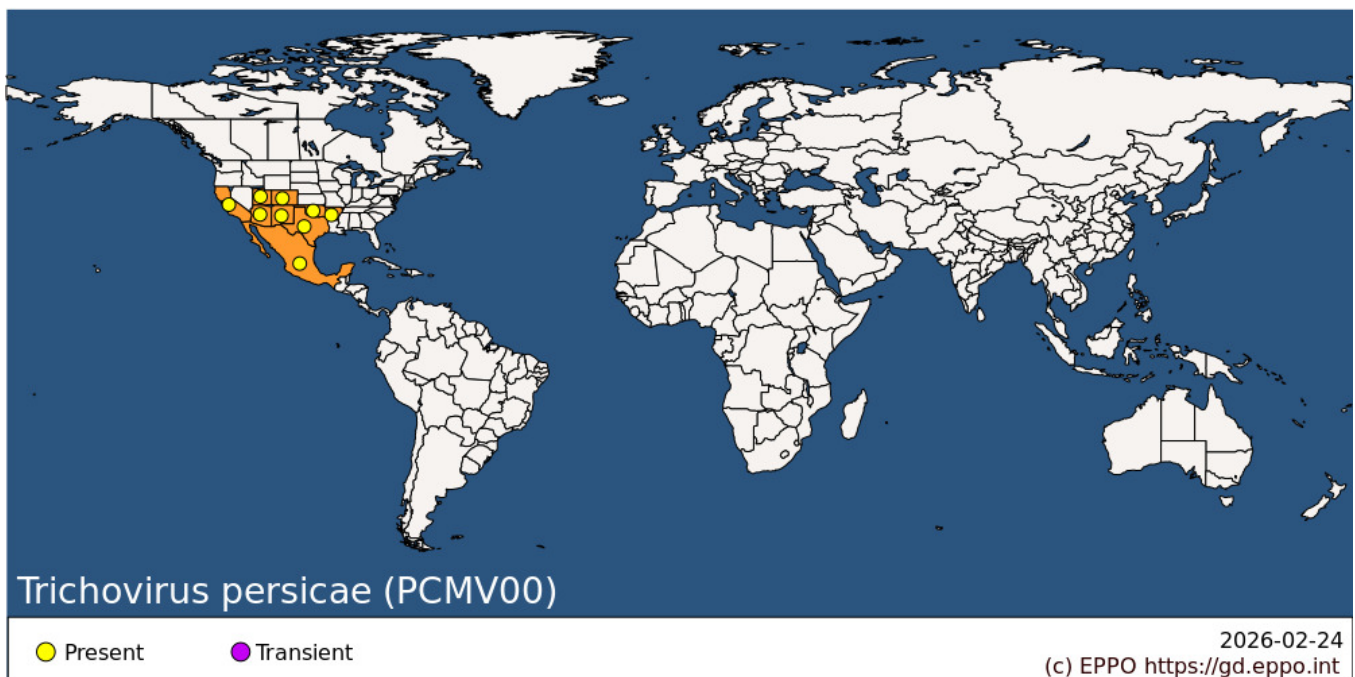
## GEOGRAPHICAL DISTRIBUTION

As indicated in the *Notes on taxonomy and nomenclature*, possible confusion with PLMVd and PeVM may have occurred in older reports and is a source of uncertainty for the geographic distribution of PcMV.

Peach mosaic disease was first observed in 1931 in the United States of America in Texas and in Colorado, and was described in 1932 (Hutchins, 1932). Rapid discovery was subsequently reported in other southwestern States (Arizona, Arkansas, New Mexico, Oklahoma, California and Utah) (Larsen & James, 2011). However, because of the implementation of strict control programs, the disease is today reported to be rare in the United States (Larsen & James, 2011) so it may no longer be present in some of the above States.

The virus has also been reported in Mexico (Larsen & James, 2011). Following initial description in Chihuahua, Baja California and Coahuila, it was also reported from several additional states (Aguascalientes, Guanajuato, Hidalgo, Jalisco, Mexico, Michoacan, Puebla, Queretaro, San Luis Potosi, Zacatecas) (Oldfield *et al.*, 1995).

There are no records of PcMV outside North America.



**North America:** Mexico, United States of America (Arizona, Arkansas, California, Colorado, New Mexico, Oklahoma, Texas, Utah)

## BIOLOGY

Peach mosaic virus is easily graft-transmissible (Larsen & James 2011). As is the case for other systemic viruses, it is also transmitted by other vegetative propagation techniques. It does not appear to be seed or pollen-transmitted (Hutchins *et al.*, 1951 cited in Larsen & James, 2011). The vector of PcMV is the peach bud mite *Eriophyes insidiosus* (Keifer & Wilson, 1955). This mite feeds and reproduces on developing leaf primordia within the bud. In areas of the USA where it infests freestone peaches, it is usually limited to adventitious buds on the trunk or on the lower scaffold branches. *E. insidiosus* has been found on peach and on several American species of *Prunus* in the south-western USA. In Mexico, *E. insidiosus* was observed in buds of wild *Prunus munsoniana* in Chihuahua and in buds of peaches (Oldfield *et al.*, 1995).

A 17% transmission efficiency has been reported using single mites (Gispert *et al.*, 1998a), with transmission parameters suggesting a semi-persistent transmission mechanism. The minimum acquisition access period was 3 days on the infected plant for the vector, there was no latency period before the mite is able to transmit the virus, and the minimum inoculation access period was between 3 and 6 h for the vector to transmit the virus (Gispert *et al.*, 1998a). There are indications that the mite can remain viruliferous for at least two days and possibly longer (Gispert *et al.*, 1998a).

Inoculation experiments indicated that different PcMV isolates may differ in their pathogenicity, suggesting that biological variability exists within the species (Pine & Cochran, 1962).

## DETECTION AND IDENTIFICATION

### Symptoms

As is often the case, symptom expression is affected by host susceptibility, virus isolate and coinfection with other viruses. In peach and nectarine (*Prunus persica* var. *nucipersica*), which are the most commonly affected crops, symptoms include delays in leaf emergence, flowering and fruit maturity by generally 4-6 days but sometimes by up to 10 days. They also show bud necrosis or doubling, leaf mottling and deformation and fruit deformation (Hutchins, 1932; Pine, 1976; Larsen & Oldfield, 1995; Larsen & James 2011).

Leaves of affected trees can show a variety of chlorotic patterns that range from tiny spots and flecks to large blotches. Leaves are smaller and narrower, frequently deformed and asymmetrical. Foliar symptoms become less clear as the season progresses. Fruits can be severely deformed, in particular in the most susceptible peach varieties, and are consequently unmarketable. The fruit surface can be irregular and bumpy, with the suture area particularly affected, and fruits are also smaller than those from healthy trees (Hutchins, 1932; Pine, 1976; Larsen & Oldfield, 1995; Larsen & James 2011).

Shoots of severely affected trees can be dwarfed and show some rosetting with shortened internodes. Flower colour-breaking is also observed, in particular in cultivars with large pink flowers. Besides the discolored streaks and patches, symptomatic petals are smaller, sometimes crinkled.

The *E. insidiosus* vector mites themselves cause some symptoms and infested buds are swollen and reddened, their growth remains retarded, and buds may eventually die (Wilson *et al.*, 1955; Oldfield *et al.*, 1995; Gispert *et al.*, 1998a).

Infected European (*P. domestica*) and Japanese (*P. salicina*) plums show only leaf symptoms, and these are comparable to those seen in peach. Susceptible wild American plum species generally have symptomless infections (Larsen & James, 2011). Infected almonds generally only show mild leaf and fruit symptoms with no impact on yield or quality. Apricot varieties vary in susceptibility with the most susceptible ones showing leaf mottling, shoot growth reduction, reduced vigour and reduced fruit production (Larsen & James, 2011).

### Morphology

PcMV has an undivided single-stranded, positive-sense genome composed of a single RNA molecule. PcMV has flexuous particles typical of Trichoviruses with an estimated size of ca. 750-880 nm in length and 9 nm in width, with striations 3.57 nm in pitch, with some variations between individual reports (James & Howell, 1998; Gispert *et al.*, 1988b).

### Detection and inspection methods

Visual examination may allow the detection of symptoms but is not considered reliable enough since symptoms are not specific and are not always obvious in infected plants. A procedure for inspection of places of production of *Prunus* trees is provided in Standard PM 3/76 (EPPO, 2021).

PcMV can be detected by biological indexing in the glasshouse on indicator peach seedlings such as Elberta, GF305, Rio Oso Gem) (Desvignes, 1976; Gispert *et al.*, 1998a; Larsen & James, 2011).

Polyclonal antibodies have been prepared against PcMV and used for its detection in ELISA tests (James & Howell, 1998; Gispert *et al.*, 1998b). The virus has also been detected using cross-reacting monoclonal antibodies prepared against the related cherry mottle leaf virus (Oldfield *et al.*, 1995; James & Howell, 1998). It is however unclear whether any of these reagents are available today as commercial kits, which limits their usefulness.

Following the identification of PcMV and the determination of its genomic sequence, specific primers have been developed for its detection by RT-PCR tests (James & Upton, 1999; James *et al.*, 2006). These tests represent the most sensitive and straightforward detection approach available today, despite the fact that their inclusiveness has not been extensively validated. PcMV could also be detected by high-throughput sequencing-based approaches.

## **PATHWAYS FOR MOVEMENT**

Natural transmission on a local scale is ensured by the eriophyid vector (see Biology). There is no information on the distance of spread by this vector but the mite is probably dispersed by wind (Wilson *et al.*, 1955). The vector mites are known to remain viruliferous for at least two days (Gispert *et al.*, 1988a). However, the mites are tightly associated with buds (Wilson *et al.*, 1955; Gispert *et al.*, 1988a), so peach fruits or on other commodities not involving buds probably do not represent a significant pathway for long distance movement of the virus. The main means of movement over long distances is the trade or movement of infected propagation material.

## **PEST SIGNIFICANCE**

### **Economic impact**

Fruits from affected trees, especially peaches and nectarines, are generally unmarketable. In the USA, the economic consequences in affected peach orchards have been considerable in the past. It is estimated that more than 390 000 trees were lost in California and Colorado before 1955 (Pine, 1976, Larsen & James, 2011), mostly as a consequence of control efforts. However, the disease is currently of very minor importance in USA, but much more important in Mexico, where control efforts were much less developed (Larsen & James, 2011).

### **Control**

The most efficient control strategy involves the development and use of PcMV-free propagation material as described in EPPO Standard PM 4/30(1) *Certification scheme for almond, apricot, peach and plum* (EPPO, 2001). The destruction of infected plants and the limitation of movement of host plants outside infected areas can also help to reduce the spread of PcMV, together with control of the mite vector (Jones *et al.*, 1970).

In the USA, the rapid spread of the disease during 1932/1935, especially in California and Colorado, led to the establishment of eradication programs that, coupled with the use of healthy planting material, were successful in containing spread and reducing the incidence of the disease to a low level in areas where the virus was present. For example, in Colorado the annual tree losses due to control efforts dropped below 1 000 in 1960, to below 100 in 1969, below 10 in 1982, below one in 1983 and finally to zero in 1987. The only documented incidence in Colorado since 1987 occurred in 1991 (Thresh, 1988; Larsen, 1998). Selection of less susceptible peach cultivars can also provide some level of protection against negative impacts (Larsen & James, 2011). Miticide treatments at the pre-bloom and petal-fall stages can also help by controlling the mite vector population (Larsen & James, 2011).

### **Phytosanitary risk**

PcMV typically infects and has its main impact in peach (*P. persica*). Peach is widely grown in the EPPO region and represents an important fruit crop. Several other *Prunus* species are known to be susceptible. There are no known ecoclimatic constraints for PcMV establishment, except those affecting its hosts; however, the mite vector *E. insidiosus* is not known to occur in the EPPO region, with the exception of Morocco (EFSA, 2019) and there might

be some ecoclimatic limitations to its establishment in some other parts of the EPPO region. It was considered justified by some EPPO countries (e.g. in the EU) to prevent establishment and spread of PcMV.

## PHYTOSANITARY MEASURES

Appropriate phytosanitary measures to import plants for planting (excluding seeds and pollen) of *Prunus* hosts into the EPPO region could require that these plants are produced in a pest free area, in a pest free place/site of production, or shown to be free from PcMV by appropriate diagnostic methods. A number of EPPO countries already ban the import of *Prunus* (other than fruits and seeds) from areas where the pest is present (EU, 2019).

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## How to cite this datasheet?

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## Datasheet history

This datasheet was first published in the EPPO Bulletin in 1978 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997, as well as in 2023. 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.

CABI/EPPO (1992/1997) *Quarantine Pests for Europe (1<sup>st</sup> and 2<sup>nd</sup> edition)*. CABI, Wallingford (GB).

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