EPPO Datasheet: *Monilinia fructicola*

IDENTITY

Preferred name: *Monilinia fructicola*
Authority: (G. Winter) Honey
Taxonomic position: Fungi: Ascomycota: Pezizomycotina:
Leotiomycetes: Helotiales: Sclerotiniaceae
Other scientific names: *Monilia fructicola* L.R. Batra, *Sclerotinia fructicola* (G. Winter) Rehm
Common names: American brown rot of stone fruits, brown rot of apple, brown rot of stone fruits, twig canker of apple
view more common names online...
EPPO Categorization: A2 list
view more categorizations online...
EPPO Code: MONIFC


more photos...

HOSTS

The host range of this fungus includes the rosaceous fruit trees: principally peaches and other *Prunus* spp., to a lesser extent apples and pears; the fungus can also be found on *Chaenomeles*, *Crataegus*, *Cydonia* and *Eriobotrya*. There are several reports that grapes (*Vitis vinifera*) are also host plants of *M. fructicola*. A report from Japan (Visarathanonth *et al.*, 1988) states that infected grapes were found in a wholesale market in Tokyo and inoculation tests were successful. In Canada, *M. fructicola* was found in vineyards in British Columbia (Sholberg *et al.,* 2003). There were infected stone fruit trees near one of the infected vineyards but this was not the case for another infected vineyard. Blackberries (subgenus *Rubus,* section *Rubus*) are also susceptible to the fungus (Hinrichs-Berger & Müller, 2010).

In the EPPO region, apples, pears and peaches are the most widely cultivated hosts.

Host list: *Chaenomeles*, *Cornus mas*, *Crataegus*, *Cydonia oblonga*, *Eriobotrya japonica*, *Malus domestica*, *Malus*, *Prunus armeniaca*, *Prunus avium*, *Prunus cerasifera*, *Prunus cerasus*, *Prunus domestica*, *Prunus mume*, *Prunus persica var. nucipersica*, *Prunus persica*, *Prunus salicina*, *Prunus*, *Pyrus communis*, *Pyrus pyrifolia*, *Pyrus*, *Vitis vinifera*

GEOGRAPHICAL DISTRIBUTION

Originally, *M. fructicola* was identified in North and South America, Australia and Japan (EPPO?CABI, 1997). The teleomorph phase of brown rot was first described on mummified peaches from Pennsylvania in 1883 (Batra, 1991). In Asia, *M. fructicola* was first reported in 2005 in Beijing (Zhu *et al.*, 2005). The pathogen was identified as the widespread causal agent of brown rot on nectarine and peach in China (Hu *et al.*, 2011b). *M. fructicola* was detected for the first time in Europe in peach orchards in France (Lichou *et al.*, 2002). Since 2001, this disease spread to many European countries: it was found on peach fruits and trees in Hungary, Spain, Slovenia, the Czech Republic (Duchaslavovà *et al.*, 2007; De Ca*l et al.*, 2009; Munda & Virš?ek Marn, 2010), on stored nectarines in Italy (Pellegrino *et al.,* 2009), on blackberries and plums in Germany (Hinrichs Berger & Müller, 2010), and on apples, pears and plums in Poland (Poniatowska *et al.*, 2013; Martini *et al.*, 2014; Ortega *et al.*, 2019).

Monilinia fructicola (MONIFC)

○ Present ● Transient

2025-04-19
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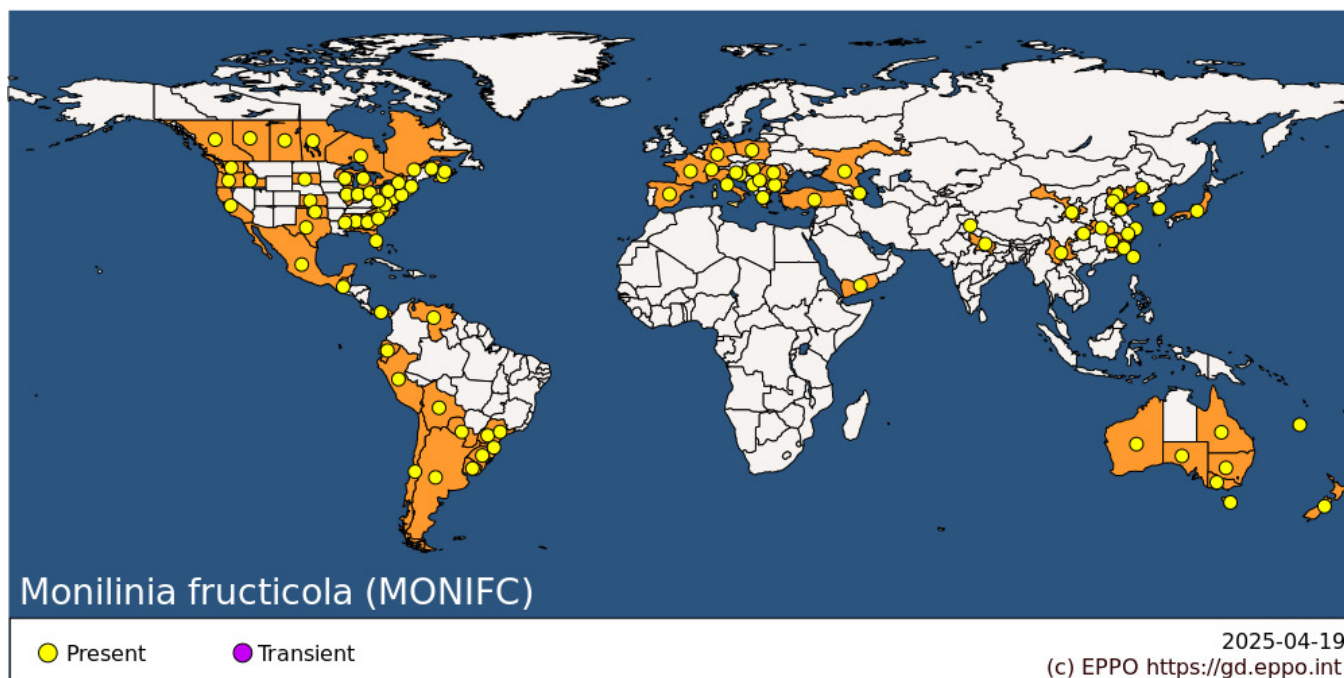
EPPO Region: Azerbaijan, Bulgaria, Croatia, France (mainland), Germany, Greece (mainland), Hungary, Italy (mainland), Montenegro, Poland, Romania, Russian Federation (the) (Southern Russia), Serbia, Slovenia, Spain (mainland), Switzerland, Türkiye
Asia: China (Beijing, Chongqing, Fujian, Gansu, Hebei, Hubei, Jiangxi, Liaoning, Shandong, Shanghai, Yunnan, Zhejiang), India (Himachal Pradesh, Uttar Pradesh), Japan (Honshu), Korea, Republic of, Taiwan, Yemen
North America: Canada (Alberta, British Columbia, Manitoba, New Brunswick, Nova Scotia, Ontario, Prince Edward Island, Québec, Saskatchewan), Mexico, United States of America (Alabama, California, Connecticut, Florida, Georgia, Idaho, Illinois, Indiana, Kansas, Maryland, Massachusetts, Michigan, Mississippi, New Jersey, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, South Carolina, South Dakota, Texas, Virginia, Washington, West Virginia, Wisconsin)
Central America and Caribbean: Guatemala, Panama
South America: Argentina, Bolivia, Brazil (Parana, Rio Grande do Sul, Santa Catarina, Sao Paulo), Chile, Ecuador, Paraguay, Peru, Uruguay, Venezuela
Oceania: Australia (New South Wales, Queensland, South Australia, Tasmania, Victoria, Western Australia), New Caledonia, New Zealand

BIOLOGY

M. fructicola overwinters in or on mummified fruit, or in infected tissues on trees, such as twigs, peduncles and cankers on branches. Conidia produced on these under humid conditions in spring are wind-dispersed and, in the presence of moisture, will infect blossoms, causing blossom blight. This generally leads to infection of the young twigs or leaves (twig and leaf blight) and stem cankers.

Under favorable weather conditions, the disease spreads rapidly. Heavy rains during the flowering period, daytime air temperature from 20 to 25°C, as well as low night temperatures are ideal conditions for the spread of the pathogen (Hrusti? *et al*., 2012).

Moisture plays an important role in the infection pathway of the fungus. Without a wetting period, infection is almost zero even in the presence of high levels of inocula; with only a 3-h wetting period infection remains very low (Wilcox, 1989); with wetness periods of 15 h, over 80% of cherries are infected by the pathogen (Biggs & Northover, 1988).

Further conidia are produced which infect ripening fruits. Fruit damage occurs at all stages of development, but with different intensity. The ripening fruits are most susceptible to infection (Luo & Michailides, 2001). Unripe fruits are less susceptible to infection. The most susceptible phase is 2-3 weeks before harvest (Biggs & Northover, 1988)

Mechanical damage contributes to the penetration of conidia, however, the pathogen can penetrate directly through the epidermis.

Conidial production itself is influenced by temperature. Temperatures around 15°C favour the development of bigger conidia, with a greater nuclear number, higher germination and, most importantly, increased virulence (Phillips, 1984; Phillips *et al.*, 1989). Infected fruits normally mummify, but if infection occurs at or near harvest, post-harvest rot may develop.

Latent infections have considerable epidemiological significance and they are not easily or completely inactivated by host response nor by fungicide treatment (Northover & Cerkauskas, 1994). Affected asymptomatic seedlings and fruits are sources of infection in international trade.

The teleomorph, rarely seen in the related European species *M. fructigena* and *M. laxa*, is significant in the life cycle of *M. fructicola*. Apothecia are erratically formed on fallen mummified fruits in spring. They release ascospores in damp weather which, in the presence of free moisture, will infect blossoms.

DETECTION AND IDENTIFICATION

Symptoms

The pathogen can infect all aerial parts of the plant: flowers, buds, young shoots, branches and fruits. *M. fructicola* causes twig and blossom blight, branch canker and fruit rot. Usually, the disease begins with the appearance of necrotic spots on the petals, later covering all parts of the flower. From flowers the infection passes to young fruiting twigs, which subsequently dry together with leaves. Gradually, the infection spreads to skeletal branches that can dry completely or become covered with cankers. Gum is released from young shoots or branches affected by the disease. In high humidity conditions, grey bunches of spores formed in sporodochia are often found on infected tissues (Michailides *et al*. 2007).

Brown rot on stone-fruits appears in the summer period in the phase of fruit ripening. First, a small brown spot develops on the fruit, which rapidly increases in size, covering the surface of the fruit. The pulp becomes brown, softens and completely loses its taste. On the surface of the fruit spongy, smoky grey pads are formed - these are the sporodochia of the pathogen. Often, they are randomly scattered over surface of the peel. Pustules are grey in *M. fructicola* and *M. laxa*, but distinctly buff-coloured when freshly formed in *M. fructigena*. In low humidity conditions, pustules may not develop; instead, the whole fruit shrivels into a wrinkled mummy.

Morphology

In presence of sporulation on infected flowers, branches, fruits or other parts of plants, *M. fructicola* can be isolated on a growth medium. *M. fructicola* has a high growth rate on PDA medium, averaging 13 mm in 24 hours at 20-22°C. The colony is greyish, light brown to ash-grey. *M. fructicola* produces abundant conidia, the sporulation showing concentric rings. *M. fructicola* has entire colony margins and an even surface (no rosettes with black arcs) (EPPO, 2020).

The macroconidia are one-celled, hyaline (greyish-yellowish/beige), ellipsoid, lemon-shaped, 8–28 ? 5–19 μm (mostly 12–16 ? 8–11 μm) and produced in branched chains. Microconidia are single-celled, 2 ?m in diameter. Microconidia are formed on bottle-shaped phialides borne on microconidiophores. Microconidia are produced both on mummified fruit and in culture media (EFSA, 2011; EPPO, 2020).

Monilinia species responsible for brown rot disease (*M. fructigena*, *M. laxa, M. fructicola)* and *Monilia polystroma* are difficult to distinguish by cultural-morphological characteristics. More reliable identification of species is based on ? combination of morphological and molecular methods.

Detection and inspection methods

Identification of *M. fructicola* is carried out according to the visible symptoms in orchards (during the growing

season) or on harvested fruits. For the detection *M. fructicola,* orchards are inspected during flowering and fruit ripening. Symptoms of infection by *M. fructicola* are similar to brown rot?caused by other *Monilinia* spp., so laboratory methods must be used to reliably identify the species. The wet chamber method, pathogen isolation on media, and molecular tests are used (in various combinations) for the identification of *M. fructicola*.

There are several different PCR tests to identify brown rot pathogens: conventional PCR (Ioos and Frey, 2000) multipex PCR (Cotes *et al.*, 2004,), random amplified polymorphic DNA analysis (Pizzuolo *et al.*, 2006), nested-PCR (Boehm *et al.* 2001), real-time (TaqMan) PCR (Brouwershaven *et al.*,2009, Wang *et al.*,2018). ?ore detailed description of identification methods are provided in the EPPO Standard PM 7/18 (3) EPPO (2020). For rapid identification of the *M. fructicola* LAMP (Ortega *et al.*, 2019) and MALDI-TOF MS-based identification (Freimoser *et al.*, 2016) methods have been developed.

PATHWAYS FOR MOVEMENT

Natural spread of *M. fructicola* can occur by wind and water, with insects and birds. In this way, local distribution occurs: within a tree or garden/orchard. During fruit ripening, insects are one of the main vectors. Damage caused by insects [e.g. *Ceratitis capitata* and *Grapholita molesta*] promotes the penetration of germinating spores into the flesh of the fruit. Insects (honeybees or beetles) may transport the fungal spores to uninfected fruits and other parts of plants.

The most likely means of entry are on plants for planting of susceptible genera, especially rooted plants for planting, and, to a lesser extent, budwood.

M. fructicola overwinters as mycelium in infected plant parts. Conditions of transport and storage of susceptible material do not affect the survival of the pathogen (Sanoamuang, 1992). The fungus can be present in latent state, without visible symptoms, which increases possibility of entry in new areas.

There is also a certain risk of fresh fruit, especially peaches, nectarines and other *Prunus* spp., apples, pears and fruit of *Cydonia* spp.

M. fructicola infects fruits at all stages of development and after harvest during transportation and storage (Agrios, 2005). Harvested fruits may be contaminated with spores and this can result in infection during storage, especially if fruits were damaged. Latent infections (conidia on the surface of fruit or intercellular mycelium) with *M. fructicola* remain quiescent until the fruit ripening when they develop visible rot symptoms.

PEST SIGNIFICANCE

Economic impact

M. fructicola causes severe losses, especially on stone fruits (*Prunus* spp.), both before and after harvest. Postharvest losses in peaches normally occur during transport and storage and may reach 80% (Sestari *et al.*, 2008); even when performing the recommended preventive measures. Heavy losses have been reported in North America on peaches, cherries and plums. Losses of 1 million AUD (Australian Dollars) occurred on peaches in 1969 in the Murrumbidgee area (Australia), and heavy losses have also been reported on apricots in Tasmania. Fungal pathogens (*Monilinia* spp.) causing brown rot and producing severe losses in worldwide stone fruit production, with high economic relevance (1.7 million EUR/year) were reported. These losses are caused by three main fungal species: *Monilinia laxa*, *M. fructigena* and *M. fructicola* (Martini & Mari, 2014).

Control

Integrated control relies on various measures, starting with variety selection, orchard design and crop load management. Reduction of inoculum sources in the orchard at the end of harvest and over winter (primarily mummies) is important, as well as removal of infected material (blossoms, twigs) during the growing season. Protective fungicide applications are recommended from bloom when disease risk is high (prediction via weather based risk models is recommended), taking resistance management into consideration. Postharvest fungicide dips

and appropriate storage and handling can reduce development of rot after harvest.

Many fungicides are approved in the EU for brown rot, including tebuconazole, trifloxystrobin, fenbuconazole, metconazole, pyraclostrobin, boscalid, thiophanate methyl, pyrimethanil, fenhexamid, myclobutanil (EU Pesticides database). The need to spray several times during the growing period in stone fruit orchards has led to a build-up of fungicide resistance in *M. fructicola*. Strains of *M. fructicola* resistant to most of the commonly used fungicides can be found, especially to benzimidazoles (Elmer & Gaunt, 1986) and dicarboximides (Elmer & Gaunt, 1988). Studies in New Zealand showed that, out of 1292 naturally sampled isolates of *M. fructicola*, 19% were tolerant of dicarboximides (Elmer & Gaunt, 1986). In laboratory studies, strains were selected which showed resistance to sterol biosynthesis inhibitors, demethylation inhibitors and morpholine (Nuninger-Ney *et al.*, 1989). Recent studies have shown that some strains of *M. fructicola* are resistant to methyl benzimidazole carbamate, demethylase inhibitor (DMI) and fungicides (propiconazole) (Chen & Liu, 2013). Studies in Italy showed the resistance of strains to thiophanate methyl (Martini *et al.*, 2016).

Biological control of *M. fructicola* is concentrating on the use of *Bacillus subtilis*. This organism has been reported as very effective against the pathogen, and is particularly used in the post-harvest control of *M. fructicola* (Pusey, 1989). Different treatments based on the biocontrol agent (BCA) *Bacillus amyloliquefaciens* CPA-8 to control *M. fructicola* under field conditions were also evaluated as alternative to chemical applications (Gotor-Vila *et al.*, 2017).

Another way to avoid losses due to *M. fructicola* is by using resistant cultivars. There are several cultivars available which are especially resistant to fruit infection (Layne, 1985; Feliciano *et al.*, 1987).

Phytosanitary risk

M. fructicola was first found in France in 2001, in peach orchards in the Rhône valley (EPPO, 2002). Entry of *M. fructicola* with plants for planting, fresh fruits of susceptible genera and by natural means from infested European countries is very likely. Establishment of *M. fructicola* in countries, where the pathogen is absent, is very likely because of the availability of host plants with a long period of susceptibility and suitable environmental conditions. Competition from other *Monilinia* species (*M. laxa* and *M. fructigena*) and currently applied cultural practices and control measures cannot prevent the establishment of the pest. The pest has already been detected in in 12 countries of the European Union (Bulgaria, Croatia, the Czech Republic, France, Germany, Greece, Hungary, Italy, Poland, Romania, Slovenia, Spain). Spread of *M. fructicola* to new areas is very likely because of its multiple means to spread (natural and human assisted), wide distribution of host species and the absence of effective barriers. Potential for yield reduction and negative effects on fruit production in orchards is estimated as moderate, mainly because of the incompleteness of data from the current area of distribution of the pest. In situations where *M. fructigena* and *M. laxa* are already present on flowers and twigs/branches the spread of *Monilinia fructicola* is unlikely to increase incidence and severity of brown rot disease, on flowers and twigs/branches (Pest risk assessment of *Monilinia fructicola*, 2011).

PHYTOSANITARY MEASURES

M. fructicola is an EPPO A2 pest recommended for regulation since 2004 (it was an A1 pest since 1984) and is also an A1 quarantine pest for Inter-African Phytosanitary Council (IAPSC) since 1989. Since symptoms on plants for planting are likely to be not easy to see, it may be most prudent to prohibit import of commodities presenting risk from countries where *M. fructicola* occurs. Alternatively, such plants for planting should come from pest-free areas or issue from a recognized certification scheme.

Fruits of *Prunus* spp., apples and pears from infested countries in the northern hemisphere could arrive in Europe at a period of the year when there is a relatively low risk that fruit trees might be infected. However, fruits from the southern hemisphere may arrive at a high-risk period, and particular care should be given to their inspection. They should come from a pest-free area or the consignment should come from a source found to be free from *M. fructicola* 6 weeks before harvest and treated according to an EPPO-recommended procedure.

The most effective method for preventing the spread of *M. fructicola* into uninfested areas is to impose strict phytosanitary measures on imported host plant material. Consignments should come from an area where *M. fructicola*

is absent, or from an area found to be free from the pest during the last growing season and where official surveys are conducted. To reduce the risk of spread in international trade, other countries are recommended to require area freedom or growing-season inspection.

For latent infections in plants for planting, it may be advisable to use a post-entry quarantine procedure in the country of destination.

In places where *M. fructicola* is officially identified, measures should be taken to contain and to eradicate it. According to the EPPO Standard PM 9/10(1), these could include: investigation to determine the extent and source of outbreak and to assess the risk of spread; delimitation of the infested areas; demarcation of contaminated facilities and equipment; demarcation of infested or and probably infected material; containment measures to prevent further spread such as setting up buffer zone(s); testing of clonally-related or contact-related stocks; methods of disposal of infected or probably infected plants or plant parts, solid waste or liquid waste; cleansing and ? or disinfection of machinery, storage facilities and other equipment; eradication measures for a specified period following an outbreak in the infested area such as cropping restrictions, measures regarding machinery and equipment, additional control measures on movement and additional surveys and use of plant protection products; monitoring of effectiveness of measures (EPPO, 2009).

For infected fruits, the following measures are possible: limiting the end-use of consignments to their processing into jams, juices, etc. when thermal or mechanical conditions prevent survival of the pathogen and prohibition to sell imported fresh fruits in markets. The measures applied by the NPPO may depend on the time of import and severity of infection (e.g. measures could be less stringent in case of import during the cold months of the year).

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Datasheet history

This datasheet was first published in the EPPO Bulletin in 1988 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997. 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 (1st and 2nd edition).* CABI, Wallingford (GB).

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