EPPO Datasheet: *Cryphonectria parasitica*

Last updated: 2020-11-10

IDENTITY

Preferred name: *Cryphonectria parasitica*
Authority: (Murrill) Barr
Taxonomic position: Fungi: Ascomycota: Pezizomycotina: Sordariomycetes: Diaporthomycetidae: Diaporthales: Cryphonectriaceae
Other scientific names: *Diaporthe parasitica* Murrill, *Endothia parasitica* (Murrill) P.J.Anderson & H.W.Anderson
Common names: blight of chestnut, blight of oak, canker of chestnut, chestnut blight, sweet chestnut blight
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EPPO Categorization: A2 list
view more categorizations online...
EU Categorization: PZ Quarantine pest ((EU) 2019/2072 Annex III), RNQP ((EU) 2019/2072 Annex IV)
EPPO Code: ENDOPA



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Notes on taxonomy and nomenclature

Although originally described and assigned to the genus *Diaporthe* and species *parasitica* (Murrill, 1906) the species was later placed in the genus *Endothia* and subsequently in the genus *Cryphonectria* due to discrepancies in the shape of ascospores, i.e. ovoid to ellipsoid with a single septum for *Cryphonectria* whereas for *Endothia* these are non-septate (Barr, 1978).

HOSTS

Chestnuts (*Castanea* spp.), particularly *C. dentata* (American chestnut) and to a lesser extent the Eurasian species *C. sativa* (sweet chestnut) are the main hosts of *C. parasitica*. Resistant, but not immune, non-European *Castanea* species include *C. mollissima* (Chinese chestnut), *C. crenata* (Japanese chestnut), *C. davidii* (Père David's chestnut), *C. henryi* (Henry's chestnut) and *C. seguinii* (Seguin's chestnut). Infection in Asian chestnuts results in small cankers which are quickly walled off by callus tissue and only marginal damage to the tree occurs. Within the EPPO region, *Castanea sativa* is the main host.

Rare hosts include oaks (i.e. *Q. petraea*, *Q. virginiana*, *Q. stellata*, *Q. coccinea*), maples (*Acer* spp.), European hornbeam (*Carpinus betulus*) and American chinquapin (*Castanea pumila*).

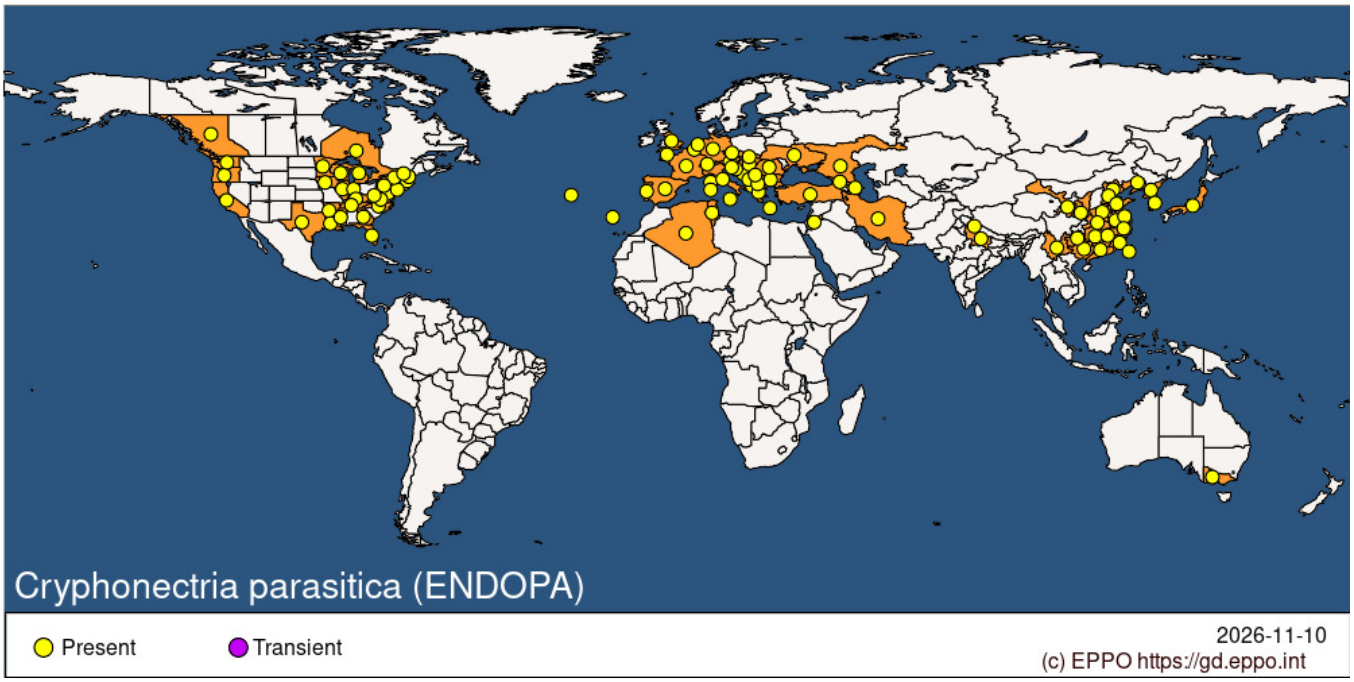
Asymptomatic host plants, from which *C. parasitica* was isolated in nature, or after inoculation, but not causing symptoms of disease, include *Ostrya carpinifolia* and *Alnus cordata*. Other asymptomatic host species include *Carya ovata, Castanopsis chrysophylla, Eucalyptus* spp., *Fagus* spp., *Liriodendron tulipifera, Malus* × *domestica, Quercus rubra,* and *Rhus typhina* (staghorn sumac).

Host list: *Acer, Carpinus betulus, Castanea crenata, Castanea dentata, Castanea henryi, Castanea mollissima, Castanea ozarkensis, Castanea pumila, Castanea sativa, Castanea seguinii, Castanea, Quercus alba, Quercus coccinea, Quercus frainetto, Quercus ilex, Quercus petraea, Quercus pubescens, Quercus stellata, Quercus virginiana*

GEOGRAPHICAL DISTRIBUTION

C. parasitica is native to Asia and it was introduced into North America at the end of the 19th Century when

Japanese chestnuts (*Castanea crenata*) were being imported to continental USA. It spread within the next five decades throughout all the main areas were chestnut was present. The pathogen eliminated the American chestnut (*Castanea dentata*) in Central and Eastern USA, which was previously a main component of hardwood forests (Anagnostakis, 1987). In 1938, the pathogen was first discovered in Europe as an isolated focus near Genova, Italy. Once again, the fungus spread very rapidly and at the end of the 1960s, most parts of Southern Europe where chestnuts are cultivated were affected by the pathogen. In Croatia, Hungary, Italy, Portugal, France and Slovenia, the pathogen is present across the areas where the host plants occur whereas Austria, Belgium, Bulgaria, Germany, Greece, Slovak Republic, and Spain report a restricted distribution. The United Kingdom recently reported re-emergence of the pathogen (in London, West Sussex and Cornwall), after it was thought to be eradicated (Forest Services UK, 2019).



EPPO Region: Albania, Algeria, Austria, Azerbaijan, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Czechia, France (mainland, Corse), Georgia, Germany, Greece (mainland, Kriti), Guernsey, Hungary, Italy (mainland, Sardegna, Sicilia), Jersey, Jordan, Montenegro, Netherlands, North Macedonia, Portugal (mainland, Azores, Madeira), Romania, Russian Federation (Southern Russia), Serbia, Slovakia, Slovenia, Spain (mainland), Switzerland, Tunisia, Türkiye, Ukraine, United Kingdom (England)
Africa: Algeria, Tunisia
Asia: China (Anhui, Beijing, Fujian, Gansu, Guangdong, Guangxi, Guizhou, Hebei, Henan, Hubei, Hunan, Jiangsu, Jiangxi, Liaoning, Shaanxi, Shandong, Yunnan, Zhejiang), India (Uttarakhand, Uttar Pradesh), Iran, Islamic Republic of, Japan (Honshu), Jordan, Korea, Democratic People's Republic of, Korea, Republic of, Taiwan
North America: Canada (British Columbia, Ontario), United States of America (Arkansas, California, Connecticut, Florida, Georgia, Illinois, Indiana, Iowa, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, New Hampshire, New Jersey, New York, North Carolina, Oregon, Pennsylvania, Tennessee, Texas, Vermont, Virginia, Washington, West Virginia, Wisconsin)
Oceania: Australia (Victoria)

BIOLOGY

Infections are triggered when the fungus enters a bark fissure or an open wound in the wood cortex, or in a stem. Wounds produced mechanically, weather related or generated by insects, are common points of infection. It has been recently shown that chestnut galls induced by the chestnut gall wasp *Dryocosmus kuriphilus* can be heavily colonized by virulent strains of *C. parasitica* (Meyer *et al.*, 2015).

Cankers result from the growth of mycelia into the host tissue. A canker can become visible on a tree within three to five weeks. The first visible sign of the disease is a darkened area around the point of infection. The fungus grows as

pale white mycelial fans which destroy the parenchyma and cambium of the plant. The fans will progressively constrain the stem or branch, disrupting sap circulation in the phloem, particularly in the terminal branches of the plant, resulting in wilting and death of tissues. Mycelium can live for up to 10 months in dried bark (Hepting, 1974). On fruit, the fungus is associated only with the nutshell and apparently does not affect seed germination or seedling growth (Jaynes & DePalma, 1984).

Bark overlaying the infection site becomes loose and sheds from the tree. At this stage, the fungal mycelium produces pycnidia followed by production of stromata tissue where the reproductive structures of the fungus (perithecia) are formed in sac-like structures. Perithecia are yellow-orange to reddish-brown and produced in groups. Long, coiled tendrils of conidial spores exude from pycnidia in wet weather. Collateral infections from branch to branch occur, and when infection reaches the main trunk, death of the tree quickly follows.

The fungus can also exist as a saprophyte on broad-leaved trees beyond its parasitic host range. Saprophytic activity of the species has been found in chestnut wood from trees which recently died and the saprophytic phase of the fungus contributed to the dissemination of hypovirulence (Conedera *et al.*, 2006; Prospero *et al.*, 2006). Hypovirulence (i.e. inability to produce severe symptoms) of *C. parasitica* was first observed in the 1950s when heavily infested chestnut stands in Italy showed signs of recovery (Heininger & Rigling, 1994). Hypovirulence of *C. parasitica* results from infection by a double-stranded RNA mycovirus, mainly *Cryphonectria hypovirus 1* (CHV-1), and is transferred between fungal individuals (horizontal transmission) through hyphal fusion (i.e. anastomosis). Vertical transmission can also occur with asexual spores which carry the hypovirus to new hosts (Peever *et al.*, 2000; Cortesi *et al.*, 2001; Rigling *et al.*, 2018). Other mycoviruses have been detected in *C. parasitica* in the genus *Hypovirus* (CHV-2, CHV-3, and CHV-4) as well as in other virus genera, i.e., two Reoviruses, *9B21* and *C-18*; and a mitochondrial virus, *Cryphonectria mitovirus 1/NB631* (Hillman & Suzuki, 2004).

Conidia can become waterborne and spread through water. Wind, rain, as well as mammals and birds that come into contact with the spores can passively disseminate these across large areas. Although insect vectors are not thought to play a very important role in the transmission of the disease, it is noteworthy that chestnut blight cankers have a very large and diverse fauna. In trapping experiments in the USA, 495 insect species were captured on old blight cankers. A considerable number of insects spent parts of their life cycle on cankers and nearly 69 species were found to carry inoculum of *C. parasitica* (Russin *et al.*, 1984). For additional information see Anderson and Rankin (1914), Boyce (1961), Darpoux *et al.* (1975), DePalma (1981) and Rigling & Prospero (2018).

DETECTION AND IDENTIFICATION

Symptoms

Cankers may enlarge so rapidly that the stem becomes girdled without callus formation. Callusing may occur, as a healing phenomenon, temporarily limiting fungal spread. Regions above the point of invasion die; the leaves wilt and turn brown but remain hanging on the tree. At this stage, chestnut blight is easily confused with ink disease (caused by *Phytophthora cambivora*). However, *C. parasitica* causes a definite canker or dead patch on the stem or trunk, below which the branches have healthy foliage; after a short time, adventitious shoot production is stimulated on the stem below this dead patch. With ink disease, on the other hand, the tree will be dead down to ground level and below.

On young, smooth-barked branches, blight-infected patches are bright brown, in contrast to the olive-green colour of normal bark. On older stem infections, the discoloration is less obvious. When the cambium is killed rapidly a sunken area appears, but when disease progress is slowed down by unfavorable weather conditions new layers of bark form under the affected areas and there is a certain amount of swelling and subsequent cracking of the outer bark. Masses of yellow-orange to reddish-brown pustules, the size of a pin-head, develop on infected bark and exude long orange-yellow tendrils of spores in moist weather. Characteristic pale-brown mycelial fans form in the inner bark and may be exposed by cutting away the outer bark. For more information see Anderson & Rankin (1914), Boyce (1961), Darpoux *et al.* (1975).

Morphology

Perithecia in clusters of 10-20. Ascospores hyaline, two-celled, constricted at the septum, 10 x 4 µm. Conidia exuded in yellowish tendrils, straight or slightly curved, hyaline, 2-3 x 1 µm.

Detection and inspection methods

Since all symptoms of infection occur above ground, visual examination and inspection is facilitated. Bark fissures and wounds in the bark should be monitored for the presence of fruit bodies of the fungus, i.e. perithecia. These will look like agglomerates of 1 mm masses of yellow-orange to reddish-brown pustules.

Graft spots are common entry points for spores of the pathogen. Cambium under the infected bark may quickly appear sunken or swollen. Orange discoloration of infected stems or the main trunk can occur. Above these areas leaves wilt. Below the canker leaves may look healthy. When the tissue of the bark is killed quickly, callus will not show, however if the progression of the canker is slow (depending on weather conditions), the tree will produce new layers of bark under the affected area and cracking and new vertical fissures might be visible.

The formation of pale white-brownish mycelial fans in the inner bark is a common sign of the disease. These are easily found in spots where the bark seems to be swollen or peeling off. The bark is easily removed to confirm the presence of mycelia.

Early detection methods of *C. parasitica* from tissue samples such as the inner and outer bark of plants can be carried out using molecular methods, e.g. real-time PCR, which provides a rapid and accurate detection even with low fungal loads (Chandelier *et al*., 2019; Rubio *et al.* 2017). Morphological identification following culturing on PDA medium (Potato Dextrose Agar) allows for visual differentiation of virulent and hypovirulent strains. Virulent, virus free strains, exhibit initial white mycelium growth which quickly pigments to orange-yellow, producing many visible pin-head sized globose red-orange asexual spores (pycnidia) scattered in the culture, when exposed to light. In hypovirulent strains containing the CHV-1 virus the mycelium remains white and the production of pycnidia is very low or absent. Confirmation of CHV-1 in cultures is also done by RT-PCR and sequencing using established protocols for RNA purification and complementary DNA sequencing (Rigling *et al.*, 2018).

Hypovirus transmission can be easily assessed *in vitro* by co-culturing pairs of hypovirus-infected and hypovirus-free strains on PDA. Compatible strains will merge into a single mycelium culture, whereas a barrier will form between incompatible strains. The resulting orange (virus free) or white (virus infected) morphology of an *in vitro* culture following this assay enables the visual assessment of hypovirus transmission success (Cortesi *et al.*, 2001; Rigling and Prospero, 2018).

Recommendations on the morphological and molecular identification of *C. parasitica* are provided in the EPPO Diagnostic protocol (EPPO, 2024).

PATHWAYS FOR MOVEMENT

Natural spread of fungal spores is ensured by wind and rain, as well as by indirect vectors such as birds, mammals and insects. Pruning tools can be a source for contamination and spread if not sterilized. In international trade, the fungus may be carried by host plants for planting traded for forestry or ornamental purposes, or on wood or bark (including wood chips with bark fragments). There is a small risk of transmission by fruits or seeds, but as the colonization rate by the fungus is low and traded nuts do not enter into contact with orchards, this pathway is considered to be of minor importance (EFSA, 2016).

PEST SIGNIFICANCE

Economic impact

Between 1904 and 1950, *C. parasitica* caused almost complete destruction of *Castanea dentata* in the Eastern USA (Hepting, 1974). There has also been extensive spread on *C. sativa* in Europe from Italy throughout most Europe since 1938. For an historical depiction of the spread of *C. parasitica* in Europe consult Robin and Heiniger (2001).

Evidence indicates that the pathogen behaves less virulently in Europe than in the USA; new and healthy coppice shoots arising from stumps originally attacked indicate recovery from the disease. This has been explained by the occurrence in Europe of hypovirulent strains which are vegetatively compatible with virulent strains (Milgroom & Cortesi, 2004; Rigling & Prospero, 2018).

The fungus is indigenous on species of *Castanea* in China and Japan where it does not have significant impact.

Control

The use of naturally occurring or artificially inoculated hypovirulent strains offers good prospects for control (Milgroom & Cortesi, 2004; Rigling & Prospero, 2018). The application of hypovirulent strains around developing lesions may enable these lesions to recover and can convert the virulent strain into a hypovirulent strain (Grente, 1981). Hypovirulence is effective as a biocontrol strategy when a large proportion of the *C. parasitica* population is infected by the virus. Hypovirulence is transmitted by hyphal fusion (anastomosis) to virulent strains of the same vegetative compatibility (vc) type (Anagnostakis & Waggoner, 1981; Robin and Heiniger, 2001). However, strains of *C. parasitica* may show vegetative incompatibility, i.e. inability to achieve hyphal anastomoses and viral transmission (Anagnostakis, 1977; Milgroom & Cortesi, 2004). Virus transmission between *C. parasitica* strains is primarily controlled by vegetative incompatibility (*vic*) genes. Six vegetative incompatible *vic* loci have been identified in *C. parasitica*, each with two alleles (Cortesi & Milgroom, 1998). If isolates of *C. parasitica* share the same alleles at the six *vic* loci, viral transmission can reach 100%. Variable biocontrol results have been achieved in different parts of the globe using hypovirulent strains.

In Europe, the main mycovirus responsible for hypovirulence, CHV-1, occurs naturally. A lower expression of incompatible loci (heteroallelism) within a restricted but widely distributed number of European vc strains of *C. parasitica*, allows for frequent and successful transmission of CHV-1 between infected and non-infected strains (Heiniger and Rigling., 1994; Robin *et al.*, 2009). Although hypovirulence is widespread in most areas of the EPPO region where *C. parasitica* is found (Bryner *et al.*, 2012), the level of incidence varies between populations of the fungus (Heiniger & Rigling, 1994). Over 40 vc types were found in France in 2001, and subsequently expanded to 61 vc types, whereas in some countries only 9 vc types have been identified (Portugal), and in Turkey only one (Robin & Heiniger, 2001; Bragança *et al.*, 2007; Robin *et al.*, 2009). Incidence can be very low such as in North-Western Spain with only 3% of the trees infected by hypovirulent strains (Tizado *et al.*, 2012). As a rule, in Europe, vc type diversity at a local or regional scale is low and comprising one or few dominant vc types which favors compatibility and hence transmission of hypovirulence (Robin & Heiniger, 2001).

In the USA the situation is very different to the one in Europe. Mycovirus CHV?1 does not occur naturally in North America and artificial inoculations with European strains infected by the virus have not been effective at large scale due to the large number of incompatible vc types. Anagnostakis & Kratz (1987) found a total of 48 vc types just within a small forest plot in Connecticut. Similar patterns were found in other regions of the USA. It has been hypothesized that the vegetative incompatible chromosome loci (*vic* loci), controlling compatibility of hyphae during anastomosis, could be polymorphic in North America, or multiple alleles might occur in the *vic* loci of *C. parasitica* in this region of the globe.

Although promising, the use of hypovirulent strains is often limited by reduced competitiveness of hypovirulent mycovirus strains due to their reduced growth and sporulation as well as the lack of transmission into sexual spores of the fungus (Pearson *et al*., 2009). Other strategies have been pursued in an attempt to halt the spread of the disease. Considerable effort was made in the USA towards breeding disease-resistant hybrid chestnuts by making use of the more resistant Asian species. These unfortunately were unsuccessful (Burnham, 1986). More recently, spread of hypovirulent strains of *Cryphonectria parasitica* on grafted American chestnut trees inoculated with hypovirus of European origin were detected in trees exhibiting a high level of blight control (Robbins & Griffin, 2008).

In Switzerland, an extensive selection program was started in the 1950s to select blight-resistant chestnut cultivars. After over 30 years of research, several clones were found which showed some degree of resistance, but the differences between resistant and susceptible chestnuts seem to be subtle (Bazzigher & Miller, 1991). Chestnut hybrid's (*Castanea sativa* × *C. crenata*) resistance to different levels of blight has been recently assessed in Slovakia (Bolvanský *et al.*, 2018) and Romania (Chira *et al.*, 2018) with promising results. However, no trees have been produced which could be planted with confidence to replace present chestnut stands, and it appears unlikely that such a replacement is a practical possibility at the present time.

Since *C. parasitica* may be transmitted by grafting, the use of wax and fungicides to protect grafts has been investigated (Turchetti *et al.*, 1981).

Phytosanitary risk

In the EPPO region, some areas are still free from the disease, in particular in the northern part. Spread of *C. parasitica* from the southern part of the EPPO region into more northern areas could cause considerable losses. Since the occurrence of relatively low strain variability has limited the losses in infected areas, the introduction of new strains might disturb the European balance between virulent and hypovirulent strains and could have a devastating effect on the remaining chestnut areas of Southern Europe. In a risk assessment study, it was considered that the risk of new introductions of *C. parasitica* within the European Union territory was still relatively high (EFSA, 2014, 2016).

PHYTOSANITARY MEASURES

In order to prevent the entry or spread of *C. parasitica*, plants of *Castanea* and *Quercus*, intended for planting, other than seeds, should be free of *C. parasitica* and originate either from a Pest Free Area, or from a place of production where no signs and/or symptoms of the pathogen have been observed in the last growing season. Wood of *Castanea* (and of *Quercus*) imported from areas where the disease occurs, should also come from areas known to be free of the pathogen or should be debarked or heat treated (e.g. kiln-drying). Isolated bark of *Castanea* should also originate in areas known to be free from *C. parasitica* (EPPO, 2017 a,b). The implementation of certification schemes for the production of healthy planting material of *Castanea* could help in reducing the risk of spreading the disease (EFSA, 2016).

The existence of a latent period between the time of infection and the emergence of symptoms is a problem for timely detection and eradication, especially for nursery plants imported from regions outside Pest Free Areas. A latent period of six months between the time of infection and the emergence of symptoms has been previously reported in *Castanea* species (Guerin *et al.*, 2000). Moreover, there is an example of plants imported from France into Australia that started to show symptoms only 16 months after import (Cunnington & Pascoe, 2003). Post-entry quarantine measures for plant material derived from regions or countries outside Pest Free Areas should therefore take this fact into account.

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Datasheet history

This datasheet was first published in the EPPO Bulletin in 1982 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997, as well as in 2020. 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.

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