

Phytophthora alni

System: Terrestrial

Kingdom	Phylum	Class	Order	Family
Chromista	Oomycota	Peronosporae	Peronosporales	Peronosporaceae

Common name

Synonym

Similar species

Summary

Phytophthora alni is an oomycete of unclear origin, which was discovered as the driver of severe Alder stand declines in Britain in 1993. It is thought to be the result of a hybridization event, which may have taken place in a nursery through the introduction of a North American parent species. Since its discovery, it has been reported throughout most of Europe, where it is considered a dangerous invasive due to its severe impact on Alders in riparian ecosystems and the ensuing knock-on effects.



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Species Description

P. alni is an oomycete, a fungus-like organism, which can be identified through the morphology of cultures or genetic means. For a description of identification through symptoms borne on the host please see the general impacts section. Cultures appear appressed-felty or slightly woolly, with few or no aerial mycelium and an irregular growth outline. The oogonial diameter ranges between 27.6 and 50.6µm (Brasier et al., 1995). A high proportion of the oogonia are small, but some are larger and more mature. These oogonia have tapered stalks and are warty. Some of the smaller, partly developed, oogonia are distinctly comma-shaped, some are distorted, and some have beak-like or tube-like protuberances. The antheridia are mostly two-celled and amphigynous, but some can be one-celled or have a difficult to observe basal septum. The length of the antheridia ranges from 20 to 30µm (Brasier et al., 2004). Sporangia, which have a length of around 49.6µm, are borne singly on sporangiophores. They are ellipsoid, non-papillate, noncaducous and have a broad exit pore (Brasier et al., 1995, Brasier et al., 2004). If it's grown on carrot agar, in the dark, for ten days, it exhibits an optimum growth temperature of 22.5 to 25oC, and an upper limit temperature of 29oC (Brasier et al., 1995).

Notes

Taxonomy The precise origin of *P. alni* is unclear. It is considered to be the result of a hybridization event, although the parent species were initially obscure. What was previously thought to be different varieties of the species are now thought to constitute three separate species, two of which are the parent species to *P. alni*. These two parent species were considered to be subspecies of *P. alni* for a number of years, but they were raised to species level in Husson et al., 2015. *P. uniformis* is one of the parental species, and it is believed to be native to North America. Its introduction into Europe, potentially through nurseries, may have been a key factor in the hybridization event. The second parent, *P. multiformis*, is found in Europe but the origin is unclear. The epidemic ensuing from the hybridization event, and the unclear origin of the species, has led to the classification of *P. alni* as an invasive throughout Europe (Aguayo et al., 2012; Aguayo et al., 2016; Husson et al., 2015). **Climate Change** This species is usually referred to as thermophilous. It has low tolerance to suboptimal temperatures, and hence climate change may result in changes in the way the disease spreads and prevails throughout Europe. The disease requires mild temperatures both in summer and winter, being vulnerable to very cold winters and very warm summers. An increase in temperatures throughout Europe may allow spread to northerly regions of Sweden, where cold is thought to limit its establishment. Additionally, the disease may become less severe in southern regions, as warming becomes a limitation to the disease during summers (Aguayo et al., 2014; Redondo et al., 2018; Cerny & Strnadova, 2012).

Lifecycle Stages

P. alni has both a sexual and an asexual phases in its life cycle. However, it does not form cysts under adverse conditions, but instead continues its dispersal in the zoospore stage (Brasier et al., 2004).

Habitat Description

This oomycete is usually found in riparian ecosystems, as it is an aggressive pathogen of the genus *Alnus* (Brasier et al., 1995). However, it can reportedly also produce symptoms in chestnut and walnut (Santini et al., 2003; Santini et al., 2006). The pathogen is insensitive to suboptimal temperatures, and cold winter temperatures are thought to pose a barrier to its spread (Redondo et al., 2018). Increasing water temperature has shown a link to its survival, and it is often denominated a thermophilic pathogen (Thoirain et al., 2007). Its optimal pH is close to neutral; it has been reported to be most stable at a pH of 7 and to be more tolerant to basic than acidic conditions (Aguayo et al., 2014; Kong et al., 2012). Infections by *P. alni* are favoured by slow-moving water (Thoirain et al., 2007). While fine-textured soil such as clay loams favour the disease, it only survives in soil for less than a year in the absence of hosts that can act as reservoirs for the disease (Thoirain et al., 2007; Bjelke et al., 2016).

Reproduction

P. alni is characterized by homothallic (selfing) sexual reproduction. It produces abundant female gametangia after six to ten days in culture (Brasier et al., 1995). However, it also exhibits a high level of zygotic abortion; up to 60% of oospores (Cerny & Strnadova, 2010). These oospores can persist in hosts or soil for a period of time. They can thus spread the disease to new hosts if an infected tree from a nursery is planted in a healthy system, or if oospore-containing soil is carried on boots or machinery to locations with vulnerable hosts. It can also reproduce asexually through sporangia, which produce motile zoospores. The motile zoospores can disperse from infected roots, by entering the water. Hence, once the pathogen is present in a riparian system, it can spread quickly to Alders downstream (Bjelke et al., 2016). It does not appear to form chlamydospores, which may contribute to its lower persistence in the absence of hosts (Brasier et al., 2004).

Nutrition

Parasite of Alder trees (Brasier et al., 1995).

General Impacts

P. alni is a major pathogen of Alder trees, driving large declines in Alder stands in many riparian systems throughout Europe over the last decades. This has made it one of the most important diseases to European ecosystems in that time (Aguayo et al., 2012). It causes a lethal root and collar disease, which can be detected through the display of the following associated symptoms: a thinning of the crown, small and pale yellow leaves, excessive seed production, dieback, tongue-shaped exuding cankers, reddish brown phloem tissue exposed from the root collar, necrotic lesions, tarry exudations on the lower stem, and lesions of the inner bark, which can appear marbled as well as reddish brown (Brasier et al., 1995; Redondo et al., 2018; Gibbs et al., 2003). Additionally, Alder can produce adventitious roots if its root system becomes dysfunctional. These roots can thus indicate the presence of lesions in the phloem down the stem (Gibbs et al., 2003). While the severe decline of Alder stands on its own makes this a highly damaging pathogen, the knock-on effects as a result of Alder loss are very worrisome. Alder trees play very important roles in riparian ecosystems. They are nitrogen fixers, inputting nitrogen-rich litter into streams. Their presence in riparian ecosystems stabilizes river banks and provides habitats for both terrestrial and aquatic organisms. Hence, the loss of these trees results in a multitude of changes at an ecosystem level. The loss of their stabilizing roles changes the structure of the riparian ecosystem, and the reduced nitrogen input into systems alters nutrient cycling and food webs. The importance of these ecosystem functions means that knock-on effects are likely to be even more far-reaching (Bjelke et al., 2016).

Management Info

Because of the difficulty of containing a waterborne pathogen, and the difficult to detect persistence in soil and hosts, prevention is the advocated management method. Research on detection methods has been carried out by multiple authors (Elegbede et al., 2010, loos et al., 2005; Redondo, 2018), and ELISA field test kits are available for detecting the pathogen in suspected infected trees. Other recommendations for preventing spread include the thorough cleaning of vehicles and footwear coming from high-risk areas, as these can spread oospores in soil, the sterilization of soil and equipment in nurseries, and the use of treated water sources for irrigation to prevent spread through zoospores (Taylor, 2013). Monitoring is necessary, as outbreaks can start from nearly undetectable levels if environmental conditions are favourable (Aguayo et al., 2014). Research is also ongoing on the incidence and modes of propagation of resistant Alder strains (Chandelier et al., 2016; Novotna & Stochlova, 2013).

Pathway

The pathogen has been shown to have been introduced due to infected nursery stock. The source of the infection for the stock is unknown. The pathogen then went on to spread downstream through river systems.

Principal source: Aguayo, J., Halkett, F., Husson, C., Nagy, Z. Á., Szigethy, A., Bakonyi, J., ... & Marçais, B. (2016). Genetic diversity and origins of the homoploid allopolyploid hybrid *Phytophthora* × *alni*. *Applied and environmental microbiology*, AEM-02221. Bjelke, U., Boberg, J., Oliva, J., Tattersdill, K., & McKie, B. G. (2016). Dieback of riparian alder caused by the *Phytophthora alni* complex: projected consequences for stream ecosystems. *Freshwater biology*, 61(5), 565-579. Brasier, C. M., Rose, J., & Gibbs, J. N. (1995). An unusual *Phytophthora* associated with widespread alder mortality in Britain. *Plant Pathology*, 44(6), 999-1007. Brasier, C. M., DELCAN, J., COOKE, D. E., Thomas, J. U. N. G., & IN'T VELD, W. A. M. (2004). *Phytophthora alni* sp. nov. and its variants: designation of emerging heteroploid hybrid pathogens spreading on *Alnus* trees. *Mycological Research*, 108(10), 1172-1184. Husson, C., Aguayo, J., Revellin, C., Frey, P., loos, R., & Marçais, B. (2015). Evidence for homoploid speciation in *Phytophthora alni* supports taxonomic reclassification in this species complex. *Fungal Genetics and Biology*, 77, 12-21.

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