

***Pseudomonas avellanae* (Psallidas) Janse et al.**

Bacterial canker of hazelnut (*Corylus avellana*)

IDENTITY

Name: *Pseudomonas avellanae* Janse, Rossi, Angelucci, Scortichini, Derks, Akkermans, De Vrijer and Psallidas 1997

Synonym(s): *Pseudomonas syringae* pv. *avellanae*

Taxonomic Position: Bacteria: Phylum Proteobacteria (Gram-negative bacteria), Class III - Gammaproteobacteria, order VIII - Pseudomonadales

Common Name(s): Bacterial canker of hazelnut, Haselnusskrebs (German), Chancre du Noisetier (French), Moria del nocciolo (Italian)

MAIN DISEASE CHARACTERISTICS

Rapid wilting of twigs, branches and whole trees in spring and summer due to blockage of the sapstream in the xylem, where the causal bacterium proliferates. In severe infections whole trees and even whole orchards may be destroyed in one season. Less aggressive, but very similar symptoms may be caused by *P. syringae* pv. *syringae* and *P. syringae* pv. *coryli* (Janse, 2006; Scortichini et al., 2001 and 2005). First described from Greece in 1976, where it caused a destructive disease in young trees of the Turkish cultivar Palaz (Psallidas and Panagopoulos, 1979). Substantial losses occurred later in Central Italy (Scortichini and Tropiano, 1994).

HOST RANGE

Hazelnut (*Corylus avellana*), wild and cultivated trees

DISTRIBUTION

- Europe: Greece, Italy

BIOLOGY and DISEASE CYCLE

P. avellanae primarily infects through not yet suberized leaf scars in early autumn via splashing and wind-driven rain containing bacteria (Fig. 1). After leaf scar infection the bacterium may overwinter under the bark in the parenchymal and xylem tissue. In spring, the bacterium can move systemically in the phloem from the infected twig to other parts of the tree, including the roots. Frosty weather, causing the bark of branches and trunk to crack, facilitates colonization by *P. avellanae*. Epiphytic populations of the bacterium could not be demonstrated and it has never been found in fruits or seeds. During summer longitudinal cankers on branches and the trunk may be formed (Fig. 2). The bacterium may survive in branches and roots (Scortichini, 1998). Acidic soils predispose to bacterial infection.

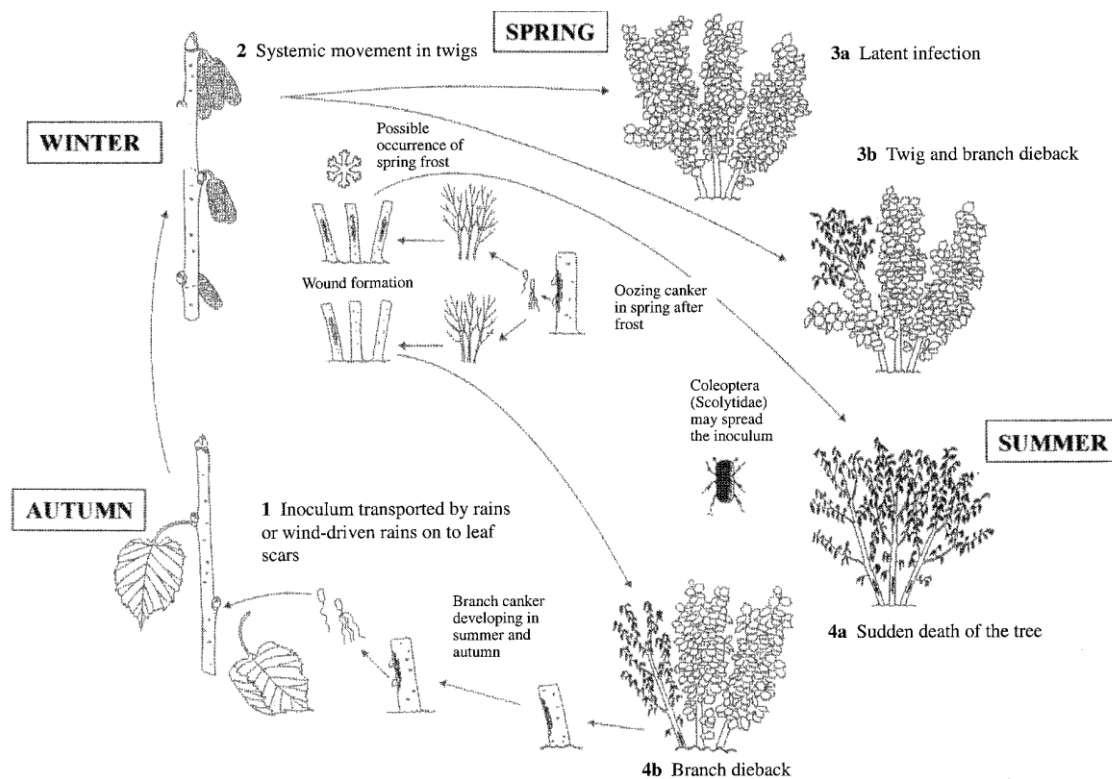


Figure 1: Disease cycle of *Pseudomonas avellanae* in hazelnut (*Corylus avellana*) as it occurs in Central Italy (from Scortichini, 2002)

Vectors

Scolytid beetles, such as *Xyleborus (Anisandrus) dispar* (L.) and *X. saxesenii* (Ratz.) are attracted by terpenes released from diseased trees. Adult insects may come into contact with the bacterium during oviposition, and larvae may be contaminated during tunneling. Although *P. avellanae* has been isolated from larvae and adult scolytid beetles, conclusive evidence for insect transmission is still lacking.

DETECTION & IDENTIFICATION

Symptoms

First symptoms may occur as wilting of the male catkins in wintertime. Dead catkins as well as (later) dead leaves usually remain attached to the twig. Also female catkins may show necrosis in February and March. In spring, early symptoms may be delayed bud break and leaf emergence. (Emerging) leaves wilt and die rapidly (Fig. 3), but other trees sometimes only show some pale green foliage in spring. Such trees often wilt and die during summer. Most obvious symptoms are observed in summer: rapid wilting of leaves on (part of) the tree (Fig. 4). A part from the necrotic leaves, also immature (dead) fruits are still attached to the twigs for many weeks. In autumn, cankers develop on branches and the trunk (Fig. 2). Diseased bark turns reddish brown, and a brown discoloration of the sapwood is apparent if the bark is stripped from the branch. Root necrosis may also occur. Infected trees that survive the winter often die the following summer.



Fig. 2
Longitudinal canker incited by
Pseudomonas avellanae on a hazelnut
tree.



Fig. 3
Wilting of young foliage during
spring incited by *Pseudomonas*
avellanae



Fig. 4
Die-back of hazelnut tree in summer
caused by *Pseudomonas avellanae*

The pathogen and pathogen diversity

The causal bacterium of bacterial canker of hazelnut was originally described as *Pseudomonas syringae* pv. *avellanae*. Further studies, involving fatty acids methyl ester analysis, whole-cell protein profiles, and sequence comparisons of 16S rRNA lead to the reclassification as *P. avellanae*

(Janse et al., 1998). DNA relatedness studies placed *P. avellanae* in the genomospecies 8, together with *P. syringae* pv. *theae* (Gardan et al., 1999). *P. avellanae* strains possess *hrp* genes (12). Less destructive disease of European hazelnut, causing limited twig and branch wilting, without canker formation and death of trees, has recently been found in northern Italy and is caused by either *P. syringae* pv. *syringae* (Scortichini et al., 2002) or by *P. syringae* pv. *coryli* (Janse, 2006; Scortichini et al., 2005).

Repetitive polymerase chain reaction (rep-PCR) using ERIC primers clearly differentiated *P. avellanae* strains from Greece and Italy (Scortichini et al., 1998). *P. avellanae* is easily differentiated from *P. syringae* pv. *syringae* from European hazelnut by fatty acid methyl esters and sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS PAGE) protein analysis or molecular techniques (Amplified rDNA Restriction Analysis, repetitive-PCR). *P. avellanae* lacks the *syrB* gene, whereas most of the *P.s* pv. *syringae* and *P.s* pv. *coryli* strains do possess it (Scortichini et al., 2002).

Detection and identification methods

Isolation of the pathogen may be performed using King's medium B and nutrient sucrose agar (NSA, 5% sucrose). A sensitive and rapid detection procedure uses 16s rRNA targeted primers PAV 1 and PAV 2, amplifying a 762-bp product present in both Greek and Italian isolates of *P. avellanae* (30) but absent in other pseudomonads. The addition of 4% BLOTTO (10% skim milk powder and 0.2% NaN₃) to the PCR mixture proved essential to avoid interference of *C. avellana* tissue compounds in the extracts during amplification. This technique allows or direct amplification from infected and symptomless hazelnut tissue (extracts from twigs, branches, or roots Scortichini and Marchesi, 2001). This method can be used when sanitising diseased plantings or screening propagative material. Other specific primers amplify the *hrpW* gene of the bacterium and can be used for the rapid detection (Loreti and Gallelli, 2002).

Useful discriminative tests have been described in Janse et al., 1998. SDS-PAGE comparison of protein extracts and fatty acid methyl esters analysis have proven effective. Repetitive-PCR using ERIC primers also showed good reliability. Using these techniques, along with some preliminary key biochemical tests (i.e., absence of oxidase, levan production), *P. avellanae* isolates can be identified in 4 to 6 days. Completion of Koch's postulates involves inoculation of leaf scars of young hazelnut trees and may take up to 7 months.

MEANS OF MOVEMENT AND DISPERSAL

Latently infected suckers used for propagation may be a main vehicle for wide-spread dispersal of the pathogen. Regenerated suckers on stumps of removed (diseased) trees have shown re-infection within 1 to 3 years. Introduction with latent infected propagative material from northern to southern orchards in the Latium region of Italy has been demonstrated (Scortichini and Tropiano, 1994). In Greece the infections could not be traced back to infected planting stock. It was mainly found in the Turkish cultivar Palaz, although there is no history of the disease in Turkey. Secondary dissemination in an orchard is mainly by wind-driven rain (Martins and Scortichini, 1998). Bacterial canker also has been found in wild European hazelnut trees growing in forests adjacent to commercial orchards in Italy. The possibility of the pathogen spreading to the apparently highly susceptible wild European hazelnut population is a serious concern (Scortichini et al., 2000a).

PEST SIGNIFICANCE

Economic or Environmental Impact:

Bacterial canker was devastating in young plantations of cultivar Palaz in northern Greece in 1976. Since it was first discovered in the Latium area in central Italy in the late 1990's, bacterial canker and decline has resulted in the mortality of more than 40,000 trees. It continued to damage trees on approximately 1,000 ha in this area. Losses are estimated to be c. \$1.5 million on an annual basis, and the disease is considered a serious problem. Currently, the inoculum pressure of the pathogen would seem reduced.

Control:

There is no direct, curative control of bacterial canker. Production of disease-free plant material and avoidance of introduction of latently infected plants are key factors in prevention of the disease. When the disease is present orchards should be monitored for early disease symptoms during (early) spring and summer. Infected plant parts must be pruned far below the infection and in case of completely infected trees roots and suckers also must be removed. All this material should be burned. Pruning and/or sucker removal should be avoided during humid periods. After a branch is cut, it is advisable to seal the wound with wax or Bordeaux mixture. It may take several years to eradicate the pathogen in severely damaged orchards. Sprays with copper-based compounds are not very effective, given the systemic nature of the pathogen. But when applied immediately after pruning, spring frost, hail, windy storms in early autumn, and at the beginning and middle of leaf drop this treatment may reduce the possibility of wound colonization by the bacterium. Orchards having very acidic soils require lime application to increase the soil pH. It is also very important to control the *Scolytidae* by using chromotropic traps. Recent progress in the control of bacterial decline has been achieved by artificial induction of systemic acquired resistance (SAR). Acibenzolar-S-methyl (CGA 245704, by Syngenta Crop Protection, registered in Europe as Bion) may induce systemic acquired resistance and was found to have a positive effect in disease control when used in concentrations of 25 g a.i ha⁻¹. Five applications of Bion applied once a month from late April to July were required to reduce the number of dead trees and branches. A final application in September after harvest was also important (Scortichini *et al.*, 2000b).

Phytosanitary Measures

Not a quarantine pathogen

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