European hazelnut or filbert (Corylus avellana L.) is a small to medium-sized tree or shrub that is widely distributed in Europe from Portugal to the southern part of the Urals and from Norway (68° north latitude) to Spain, Italy, and Greece. It also is native to eastern and central Asia, including Turkey, Syria, and Iran, and to Algeria in northern Africa (10).

European hazelnut produces a high-quality nut prized for its flavor. In prehistoric times, hunter-gatherer populations in eastern Asia (13,000 B.C.) and Scandinavia (11,000 B.C.) used the nuts as an important nutrient source (2,17). Remarkably, European hazelnut was one of the dominant trees in northern Europe after the end of the latest glacial period between 8,000 and 6,000 B.C. In some European peat bogs, the amount of hazelnut pollen exceeds that of all other tree species combined by 75% (39).

Currently, European hazelnut is grown commercially for nut production primarily in Turkey, Italy, and Spain. The tree was introduced into Oregon in the United States in the mid-1850s (8,11), where it also is grown commercially (Table 1). Cultivation of European hazelnut is expanding to other countries in the northern and southern hemispheres that have a temperate climate (1,7,16).

The main diseases of European hazelnut are eastern filbert blight, caused by Anisogramma anomala (Peck) E. Muller, in the United States (14), Apple mosaic virus in Spain (23), and bacterial canker and decline, caused by Pseudomonas avellanae, in Italy (25).

### Bacterial Canker and Decline

Bacterial canker and decline on European hazelnut was first observed in northern Greece in 1976 (22). Within a few years, young plantings of the Turkish cultivar Palaz were almost completely destroyed by the disease (20). Subsequently, the same disease, referred to locally as “moria,” was observed in plantations throughout a 20,000-ha area in the Latium region of central Italy (i.e., Viterbo province) (37).

Since it was first discovered, bacterial canker and decline has resulted in the mortality of more than 40,000 trees in central Italy. It continues to damage trees on approximately 1,000 ha in this area. The estimated loss per year is approximately $1.5 million, and the disease is considered a serious problem. There is a national law to partially compensate farmers whose trees have been seriously damaged by this disease (4).

Bacterial canker also has been found in wild European hazelnut trees growing in forests adjacent to commercial orchards in Italy (31). The possibility of the pathogen spreading to the apparently highly susceptible wild European hazelnut population is a serious concern (24).

**Symptoms**

Initial symptoms of bacterial decline may develop in winter during the blossoming of the male inflorescences. Infected catkins release only sparse amounts of viable pollen and often completely wilt. The dead catkins tend to remain firmly attached to the twig during the winter. In February and March, female inflorescences on infected trees may fail to enlarge properly and become necrotic. In spring, diseased trees may exhibit a delayed bud break and leaf emergence. Emerging leaves may wilt and die rapidly on individual branches (Fig. 1). In other cases, trees without any previous symptoms of decline may exhibit pale green foliage in early spring. Such trees often wilt and die during summer. The most striking symptoms oc-

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**Table 1. Production, import-export trade, and domestic consumption of the main European hazelnut producers in 2000**

<table>
<thead>
<tr>
<th>Country</th>
<th>Production (t)</th>
<th>Imports (t)</th>
<th>Exports (t)</th>
<th>Domestic consumption (t)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turkey</td>
<td>610,000</td>
<td>…</td>
<td>400,000</td>
<td>185,000</td>
</tr>
<tr>
<td>Italy</td>
<td>115,000</td>
<td>45,000</td>
<td>48,000</td>
<td>114,000</td>
</tr>
<tr>
<td>United States</td>
<td>34,500</td>
<td>15,000</td>
<td>13,000</td>
<td>32,400</td>
</tr>
<tr>
<td>Spain</td>
<td>25,000</td>
<td>9,700</td>
<td>14,000</td>
<td>18,500</td>
</tr>
</tbody>
</table>

* Source: International TreeNut Council.

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**Fig. 1. Rapid wilting European hazelnut leaves in early spring caused by Pseudomonas avellanae.**
cur in summer when leaves on one to all branches rapidly wilt (Fig. 2A). Wilted foliage and immature fruit remain attached to the twigs for many weeks (Fig. 2B) and sometimes into winter. Necrotic spots do not develop on leaves or nuts. In autumn, cankers develop on branches and the trunk (Fig. 3). 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.

Causal Agent

The host-specific bacterium that causes canker and decline of European hazelnut was originally described as *Pseudomonas syringae* pv. *avellanae* (19,21). Further evidence obtained from fatty acids methyl ester analysis, whole-cell protein profiles, and sequence comparisons of the 16S rRNA gene changed the designation to *P. avellanae* (9). In 1999, based on DNA-relatedness studies (i.e., genomospecies) of *P. syringae* and related species, *P. avellanae* was included in the genomospecies 8, together with *P. syringae* pv. *theae* (5). Subsequently, it has been shown that *P. syringae* pv. *actinidiae* might also be included in this genomospecies (33). *P. avellanae* strains have *hrp* genes encoding for the harpin proteins that are involved in the elicitation of the hypersensitivity reaction in leaf tissues (12).

A less destructive bacterial disease of European hazelnut, causing limited twig and branch wilting, has recently been found in other areas of European hazelnut cultivation in Italy (35). The pseudomonads associated with this disease resemble *P. syringae* pv. *syringae* (35). Unlike *P. avellanae*, these *P. syringae* pv. *syringae*-like isolates do not cause cankers and rarely result in tree mortality. When leaf scars of European hazelnut were artificially inoculated with the two pathogens, *P. avellanae* caused extensive twig dieback even at low inoculum levels, whereas *P. syringae* pv. *syringae*-like strains caused dieback to a lesser extent and only at the highest inoculum doses (Fig. 4).

Pathogen Diversity

Diversity within *P. avellanae* strains was initially examined by biochemical and nutritional tests, fatty acid methyl ester analysis, and whole-cell protein profiles (9,26,37). These analyses indicated a high degree of similarity among strains isolated in Greece and central Italy. However, Italian isolates lose fluorescent pigment production on King’s medium B after subculturing, whereas isolates from Greece do not.

Subculturing of *P. avellanae* on nutrient sucrose agar (NSA) supplemented with 7% instead of 5% sucrose and incubated at 30°C, or subculturing 30-day-old colonies on NSA, resulted in the appearance of

Fig. 2. A, During summer, diseased European hazelnut trees exhibit rapid wilting and branch dieback. B, After the wilting, the desiccated leaves remain firmly attached to the branch throughout the growing season.

Fig. 3. Longitudinal canker on a stem of European hazelnut caused by *Pseudomonas avellanae*.

Fig. 4. Virulence expressed as percentage of wilted twigs after inoculation of European hazelnut with *Pseudomonas avellanae* strains (Group A) and strains resembling *P. syringae* pv. *syringae* (Group B) obtained from different areas of European hazelnut cultivation in Italy (35).
clear, waterdrop-like colony variants. These variants did not induce the hypersensitivity reaction in tobacco leaves. This colony type was occasionally observed on NSA during isolation of P. avellanae from wood and bark. These colonies would sometimes revert to the wild-type colony morphology of P. avellanae upon further subculturing (26).

Genetic variability among P. avellanae strains was determined by plasmid analysis (9) and repetitive polymerase chain reaction (PCR) using ERIC, BOX, and REP primer sets. P. avellanae strains from Greece were clearly differentiated from those of Italy by the selective amplification of approximately 300- and 800-bp fragments using ERIC primers (27). Similarly, monoclonal antibodies raised toward cell wall polysaccharides of strains isolated in Greece and central Italy clearly differentiated two serotypes (18).

P. avellanae is easily differentiated from P. syringae pv. syringae strains obtained from European hazelnut by fatty acid methyl esters and sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) protein analysis (32,35) or molecular techniques (Amplified rDNA Restriction Analysis, repetitive-PCR) (35) (Fig. 5). In addition, all P. avellanae strains lack the syrB gene, whereas most of the P. syringae pv. syringae strains from hazelnut have this gene encoding for the production of phytotoxins (i.e., lipodepsinonapeptides) (38). Genetic variability was also found among many P. syringae pv. syringae strains isolated from diseased trees within one region of European hazelnut cultivation in Italy (Campania, Piedmont, Sicily, and Sardinia). These strains were genetically different from those of the other regions (32,35).

Identification and Detection

The isolation and identification of P. avellanae using traditional techniques is time-consuming. Completion of Koch’s postulates may take up to 7 months. Phenotypic and molecular techniques can greatly facilitate the identification process. SDS-PAGE comparison of protein extracts and fatty acid methyl esters analysis have proven effective (9,26). Repetitive-PCR using ERIC primers also showed good reliability (34). 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.

A sensitive and rapid detection procedure has been developed by using primers targeting part of the 16S rRNA gene. The primers PAV 1 and PAV 2 amplify a 762-bp product that is present in both the Greek and Italian isolates of P. avellanae (30) but is absent in other pseudomonads. The addition of 4% BLOTTO (10% skim milk powder and 0.2% NaNO₃) (3) to the PCR mixture proved essential to avoid interference of C. avellanae extracts during amplification. This technique allows for direct amplification from infected hazelnut tissue (extracts from twigs, branches, or roots) and allows for detection of P. avellanae in asymptomatic trees (30). This aspect is very important for sanitation of diseased plantings or for the screening of propagating material.

Disease Cycle

P. avellanae primarily infects through leaf scars in early autumn (Fig. 6). Leaf scars during this period are not fully suberized and can be infected by rain-splashed or wind-disseminated bacteria. Application of 10 µl of a 1 × 10⁵ CFU ml⁻¹ suspension (i.e., around 1,000 cells) of P. avellanae on a single leaf scar in early autumn resulted in 100% infection (24,28). Once inside the twig, the bacterium overwinters in bark. In spring, the bacterium moves systemically from the infected twig to other branches and even roots (24). An adult tree can be killed in 7 months following inoculation of 25 leaf scars scattered on 1-year-old twigs randomly distributed through the canopy (Fig. 7). Two-year-old trees can be killed by inoculating as few as two or three leaf scars (24).

After spring frosts, P. avellanae may also colonize wounds associated with frost cracks on branches and trunks. If the bacterium colonizes cracks on multiple branches on the same tree, mortality may occur later that spring or early summer. Presently, there is no evidence for the presence of epiphytic populations of P. avellanae on leaf or bark surfaces. The bacterium has also not been isolated from nuts taken from infected trees. Bacterial infection may result in the formation of longitudinal cankers on branches and the trunk during summer and autumn (Fig. 3). The bacterium survives adverse weather in the bark of twigs and roots (24).

Insects may play a role in disseminating P. avellanae from an infected tree to a nearby healthy tree. Several scolytid beetles, including Xyleborus (Anisandrus) dispar (L.) and X. saxesenii (Ratz.) are attracted by the terpenes released by diseased trees. Adult insects may come into contact with the bacterium during oviposition, and larvae may be contaminated during tunneling. P. avellanae has been isolated from both larvae and adult scolytid beetles, but it has not been conclusively demonstrated that these insects can transmit the disease.
Predisposing Factors

Environmental factors can play a significant role in increasing the susceptibility of European hazelnut to *P. avellanae*. Spring frosts often occur in valleys of central Italy where hazelnuts are grown. Surveys conducted in orchards where bacterial canker and decline were prevalent revealed that symptoms were more severe on trees located at the bottom of small valleys where frost was more likely to occur (35). During March and April, temperatures in these valleys dropped to lows of –1 to –5 °C, sometimes on consecutive days, during bud swell. These spring frosts can cause longitudinal cracks to form along the branches and trunk (Fig. 8) that are subsequently colonized by *P. avellanae* dispersed from oozing cankers by rain and wind.

Soils of volcanic origin with pH values <5.0 are present in some areas of central Italy. Acidic soils can increase the susceptibility of fruit tree species to pseudomonads (15,40,41). In addition, the current agro-nomical techniques adopted in central Italy include the use of soil acidifying nitrogen fertilizers. Also for the *P. avellanae*-*C. avellana* pathosystem, a low pH accompanied by a high content of aluminum in the soil (>20%) and in tree roots (>600 mg kg⁻¹), in the absence of adequate calcium, can increase the susceptibility of the tree (36). Acidic soils are also present in northern Greece where *P. avellanae* has eliminated the cultivation of European hazelnut (P. G. Psallidas, personal communication).

Epidemiology

Large-scale dissemination of *P. avellanae* may occur through distribution of latently infected suckers used for propagation. Apparently healthy suckers often develop beneath infected hazelnut trees. However, if the diseased tree is removed and the remaining suckers are left to regenerate a new tree, these suckers often will be diseased within 1 to 3 years (37) (Fig. 9). Long-distance dissemination of *P. avellanae* through shipment of infected suckers has not yet occurred in Italy because historically each region has propagated its own hazelnut germ plasm. Nevertheless, introduction of *P. avellanae* from northern to southern orchards in the Latium region of Italy by means of latently infected propagative material has been demonstrated (37). Interestingly, the sudden appearance of hazelnut bacterial canker and decline in Greece cannot be traced to infected planting stock. The cultivar Palaz, used in the Greek orchards, originated from Turkish hazelnut germ plasm. Apparently there is no record of hazelnut decline in Turkey where *C. avellana* is extensively cultivated.

Local dissemination within and between orchards was studied by following the spread of the disease in orchards soon after the first disease foci were observed (13). Data indicated an aggregated pattern of distribution early in the epidemic followed by a more random pattern afterward. The initial distribution might be explained by introduction of *P. avellanae* on latently infected trees randomly planted in the orchard. These trees served as source of inoculum for secondary spread. New occurrences of infection were significantly (*P < 0.05*) dependent on the presence of a nearby affected tree. In addition, the disease spread preferentially to trees downwind of the infected tree (13).

Control

Effective control of bacterial canker and decline of hazelnut is difficult. The production of disease-free nursery plant material is very important. The best way to avoid the disease is to prevent the introduction of latently infected plants. Monitoring of orchards for early disease symptoms is essential. Inspections should preferably be made during spring and summer with the aim detecting wilting twigs and branches. Infected plant parts must be removed from the orchard and should be burned. In case of completely wilted trees, the roots and suckers also must be removed (Fig. 9). 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.

Taking into consideration that *P. avellanae* mainly lives inside the plant, the efficacy of copper-based compounds, the only chemicals allowed in Italy for controlling bacterial disease, is rather low. How-

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Fig. 6. Bacterial canker and decline of European hazelnut caused by *Pseudomonas avellanae* in central Italy.
ever, a combination of agronomic techniques and applications of copper-based materials at key times can partially manage the disease. As a general rule, farmers apply copper-based chemicals immediately after pruning, spring frost, hail, windy storms in early autumn, and at the beginning and middle of leaf drop, to 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 (36).

Recent progress in the control of bacterial decline has been achieved by artificial induction of systemic acquired resistance (SAR). We used acibenzolar-S-methyl (CGA 245704, by Syngenta Crop Protection), registered in Europe as Bion (Actigard in the United States). In 2001, after 5 years of field trials, Bion was registered in Italy for bacterial canker and decline of hazelnut. The minimum concentration to effect good control without inducing any phytotoxic effect is 5 g a.i. hl⁻¹ (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. In orchards sprayed with Bion in which wilted trees and branches were annually removed, the mean number of dead trees and branches was 25% lower than in the orchards treated only with copper oxychloride at leaf fall. However, the efficacy of the product was lower in orchards where wilted twigs and/or branches were not removed (29). Therefore, an integrated approach including inoculum destruction (wilted twigs, branches, and trees), copper treatments at critical times, insect control, and application of acibenzolar-S-methyl may result in satisfactory management of bacterial decline.

**Conclusions**

Bacterial canker and decline of European hazelnut caused by *P. avellanae* can be devastating in some locations in central Italy. When the disease was first discovered, it was not aggressively controlled and as a consequence was allowed to spread. Realization of the importance of this disease prompted epidemiological and control studies that culminated in the development of an integrated management strategy.

Although progress has been made in controlling bacterial canker and decline, further research is necessary. For example, further population studies might better clarify the relationships among *P. avellanae* isolates and *P. syringae* pv. *syringae* isolates collected from other areas of European hazelnut cultivation in Italy. Use of “classical” methods, including host specificity and virulence assessment, and molecular tools such as the genomic fingerprinting and the sequencing of the “housekeeping genes,” can help elucidate such relationships. There is also a need to improve the knowledge of the life cycle of the pathogens and to understand why sudden and destructive epidemics appeared almost simultaneously in two different areas of Europe. This is especially curious since the *P. avellanae* populations found in Greece and Italy were genetically different and there was no exchange of contaminated propagative material between the areas. One possible link might be the occurrence of acidic soils in both areas, although *P. avellanae* is also virulent to trees in orchards with soils within recommended pH values of 6.5 to 7.5 (28,37).

Knowledge of the diversity of the pathogen is also important for breeding programs aimed to select tolerant and/or resistant hazelnut germ plasm. The inoculation technique has been standardized (24,28), and it is possible to screen many seedlings for susceptibility to bacterial canker and decline within a few months. However,

**Fig. 7.** To test the pathogenicity of *Pseudomonas avellanae*, 25 leaf scars randomly distributed in the canopy were inoculated at the beginning of October by placing approximately 1,000 CFU per leaf scar. The following May, the whole tree wilted.

**Fig. 8.** Longitudinal cracks on the trunk of a European hazelnut tree caused by spring frosts. The wounds subsequently are colonized by *Pseudomonas avellanae*. In this case, pathogen infection of bark cracks in spring resulted in mortality in summer. Note the development of new shoots from roots.

**Fig. 9.** European hazelnut will produce new shoots from roots following removal of cankered or dead trunk. These new shoots become systemically infected with *Pseudomonas avellanae*. Therefore, removal of an infected tree must also include the roots and suckers.
nothing is known about the presence of inherited resistance to bacterial canker and decline in _Corylus avellana_. Another important aspect to consider in future studies is production of host-specific toxins that might explain the strong virulence of _P. avellanae_ toward _Corylus avellana_ germ plasm. Preliminary studies indicate that, in vitro, the pathogen produces a compound(s) with phytotoxic activity to leaf tissue and biocidal activity toward microorganisms (6).

Finally, since _C. avellana_ is, at least in Europe and Asia, a crop still based on traditional cultivars adapted to a particular area, and breeding programs have not yet led to replacement of this ancient germ plasm, it seems to be a plant species suitable for studies investigating the behavior of similar phytopathogens in different environments not heavily modified by human beings.

**Literature Cited**


