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Eastern Filbert Blight of European Hazelnut

It's Becoming a Manageable Disease

The European hazelnut, *Corylus avellana* L., is produced commercially on 12,000 ha in western Oregon, which represents 98% of production in North America and 5% of production worldwide. Culture of the hazelnut is similar to that of almond in California. Mature hazelnuts fall freely onto a manicured orchard floor in late September and are collected with mechanized harvesters (Fig. 1). The nuts are sold for direct consumption and for use in confections and baked goods.

Historically, the growth of the hazelnut industry within Oregon (and Washington and British Columbia) has been influenced, at least in part, by the disease eastern filbert blight. This canker disease is caused by the pyrenomycete *Anisogramma anomala* (Peck) E. Müller in E. Müller & Arx (*Diaporthales*). *A. anomala* is endemic on American hazel, *Corylus americana* Marsh., a common understory shrub in deciduous forests of northeastern North America, and is specific to *Corylus* spp. On American hazel, *A. anomala* is a nonlethal parasite that causes an insignificant canker (4,7,16,21), which typically ranges from 1 to 10 cm in length. In contrast, on European hazelnut, this pathogen can cause cankers that expand perennially at rates up to 1 m per year (10). Expanding cankers girdle branches and limbs, resulting in canopy dieback (Fig. 2) and death of trees in 5 to 12 years if diseased limbs are not removed. Several of the earliest reports on eastern filbert blight (1,2,7,25) indicate that, in the early part of this century, attempts to establish commercial production of European hazelnut in the northeastern United States failed because the disease could not be controlled. In 1922, the state of Oregon recognized the seriousness of the threat of this disease to the hazelnut industry and established a quarantine prohibiting importation of *Corylus* spp. from east of the Rocky Mountains (2).

The first report of eastern filbert blight west of the Rocky Mountains appeared in 1973 (5), when the disease was found in a European hazelnut orchard located in Lewis County of southwest Washington. By 1976, *A. anomala* had become established in several orchards and in volunteers of *C. avellana* scattered through the woods. Over the next several years, the disease spread to remaining orchards in the area; and by 1994, most orchards in Lewis County had been destroyed as a result of the disease. In 1986, diseased hazelnut trees were observed along the northeastern edge of Oregon's Willamette Valley, the principal hazelnut production area; and subsequently, the incidence of eastern filbert blight has moved southward at an average rate of 2 to 3 km per year. Currently, 30 to 40% of Oregon's hazelnut orchards are diseased or within a few kilometers of a diseased orchard (Fig. 3). Most Oregon orchards in the vicinity of the initial detections also have been destroyed by the disease (23).

Beginning in the 1980s, the Oregon Hazelnut Commission, Oregon State University, and the USDA's Agricultural Research and Cooperative States Research Services funded an intensive research and extension effort designed to reduce the potential impact of eastern filbert blight on Oregon's hazelnut industry. The objectives of the research program were to (i) further our understanding of the biology of *A. anomala* and factors affecting its dispersal, (ii) develop cultural and chemical methods to manage the disease in susceptible orchards, and (iii) develop disease-resistant hazelnut cultivars. The extension effort focused on diagnosis, education, and rapid dissemination of new information. Significant progress has been made on all objectives.

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This is Oregon Agricultural Experiment Station Technical Paper 10,915.

Publication no. D-1996-0729-07F

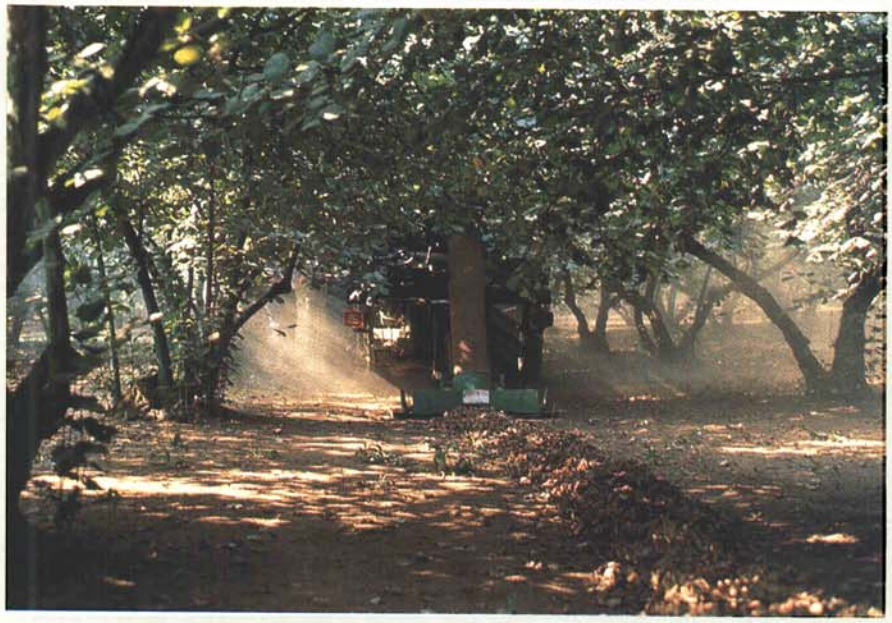

Fig. 1. Harvesting of a European hazelnut orchard in Oregon's Willamette Valley. This crop is grown on 12,000 ha in the valley and has a farm gate value of $15 to 20 million annually.

Infection Biology of *A. anomala*

The most challenging aspect of understanding the biology of eastern filbert blight has been the determination of how and when *A. anomala* gains entry into a hazelnut tree. The first significant information on this process was provided by Gottwald and Cameron (9), who exposed potted hazelnut trees in a diseased orchard for 3-week periods from February to May, and observed that external symptoms of eastern filbert blight developed only after a 12- to 16-month incubation period. These researchers also focused on dormant, vegetative buds as the probable site of infection by *A. anomala*. Based on correlative evidence (3,8,9), they hypothesized that wounding and galling of hazelnut buds by an eriophyid mite (*Phytopus avellanae* Nal.) facilitated infection by the pathogen. The role of eriophyid bud mites in infection by *A. anomala* remained ambiguous, however, until Stone et al. (26) demonstrated that actively growing buds and shoots not infested with bud mites developed a high incidence of infection when inoculated with ascospores of the pathogen. More recently, it was learned that only immature tissues near the apical meristem of growing shoots (Fig. 4) are susceptible to invasion by germinating ascospores (12), which are well adapted to adhere to immature shoot surfaces (24). The fungus penetrates young epidermal cells directly, producing a vesicle-like structure within the initially infected cell (Fig. 5) (24). Thus, hazelnut trees become susceptible to infection by *A. anomala* only after vegetative buds have initiated active growth in spring (budbreak) (12), and are most susceptible during the periods of leaf emergence (mid- to late March) and initial shoot elongation (April) (Table 1). Infection occurs over a relatively broad range of temperatures (8 to 25°C) and is facilitated by a 24- to 72-h period of high humidity (12,26). Neither wounds nor natural openings on hazelnut trees appear to serve as important sites of entry for this pathogen.

Once infections are established within a shoot, *A. anomala* colonizes phloem, cambium, and the outermost layer of xylem. Initial colonization occurs without obvious symptoms of disease. Hyphae of *A. anomala* in symptomless shoots can be detected 1 to 16 months after inoculation by examining hand-cut sections of the outer layer of xylem sampled from near the site of infection (26), or more recently, by performing an enzyme-linked immunosorbent assay (ELISA) procedure on infected shoot tissue (C. J. Coyne, S. A. Mehlenbacher, R. O. Hampton, J. N. Pinkerton, and K. B. Johnson, *unpublished*). The first distinguishing symptoms of disease coincide with the formation of stromata by the pathogen, which occurs in the spring 12 to 14 months after initial infection (26). An extended cold period (either natural or artificial), during which the tree undergoes dormancy, is necessary to initiate formation of stromata (12,26). As the tree subsequently resumes growth, colonized phloem and cambium develop a necrotic, chocolate brown discoloration (12). Under natural conditions, elliptical stromata of the pathogen (each ca. 2 × 5 mm) begin to develop within the discolored (cankered) tissues in April and erupt through the bark in late May to early June. Within the canker, individual stromata develop in parallel rows aligned with the long axis of the branch (Fig. 6). A typical stroma contains 50 to 100 perithecia (Fig. 7), and each perithecium contains approximately 10^3 asci, which reach maturity in autumn (8,22). Cankers expand perennially, with yearly increases in length that range from a few centimeters on small branches to 1 m on large-diameter branches of susceptible cultivars (10).

Fig. 2. Dieback of a mature European hazelnut tree from eastern filbert blight. Mature, nonmanaged trees become unproductive as a result of the disease 4 to 12 years after first infection.

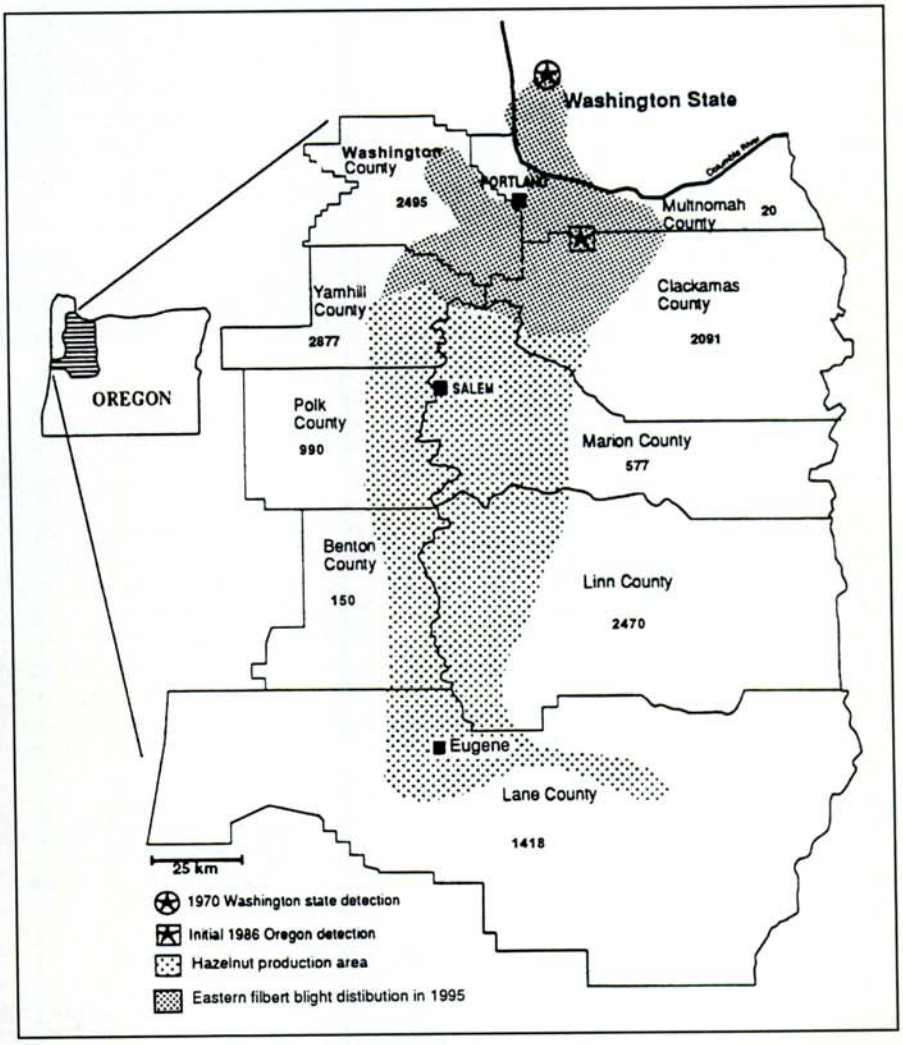

Fig. 3. Map of the commercial hazelnut production area in Oregon's Willamette Valley. The number listed in each county is total hectares in hazelnut production during 1994. Since the first report of eastern filbert blight in southwestern Washington in 1970 (6), the distribution of the disease has expanded southward at an average rate of 2 to 3 km per year (23).

Fig. 4. A vegetative shoot of European hazelnut (*Corylus avellana*) showing the immature, pubescent stem tissue near the apical bud that is highly susceptible to infection by ascospores of *Anisogramma anomala* (12).

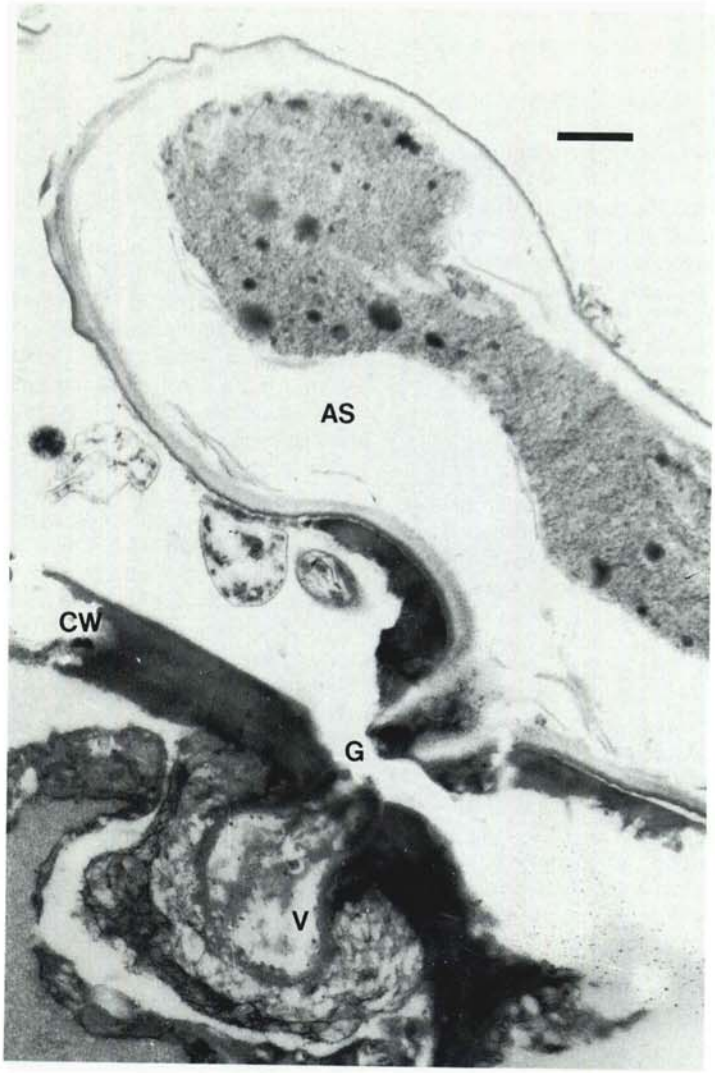

Fig. 5. Transmission electron micrograph showing penetration of a susceptible hazelnut shoot by *Anisogramma anomala*. A germination hypha (G) from an ascospore (AS) has penetrated the wall (CW) of an epidermal cell and formed a vesicle (V) 8 days after inoculation. Bar = 1 µm.

Cankers in large branches also expand laterally, with new rows of stromata being produced each year along the canker margin. Dieback occurs when expanding cankers girdle branches and limbs (3,10). As branches die, the pathogen also dies; there is no interim saprophytic stage or resting structure associated with dead, dried branches or debris.

Biologically, *A. anomala* bears greater similarity to some obligate parasites, such as the rust fungi, than to its closer relative *Cryphonectria parasitica* (Murrill) Barr or to other familiar canker fungi. For example, the inability of *A. anomala* to survive saprophytically in dead hazelnut branches is an indication that this pathogen is both obligate and biotrophic on its host. Unlike most common canker fungi, *A. anomala* does not readily grow on standard mycological media. *A. anomala* has been cultured, however, on a modified Murishige and Skoog medium (MMS) using techniques similar to those used to culture several rust fungi (27,29). As in some rust fungi (28), germ hyphae that emerge from ascospores on solid MMS initially form a vesicle from which hyphae emerge and which eventually produce branches bearing haustorium-like structures (27). Haustorium-like structures also are produced in planta within phloem parenchyma cells (24). Ascospore germination and hyphal growth of *A. anomala* in culture are apparently regulated by a chemical self-inhibitor (27). Both successful establishment of cultures from ascospores and sustained growth require media to be amended with chemical adsorbents (e.g., activated charcoal, bovine serum albumin) to inactivate the inhibitor. Even under optimum conditions, however, colonies of *A. anomala* on solid MMS grow very slowly, reaching a diameter of only a few millimeters after several weeks of incubation. Sporulation in culture has never been observed (27). Learning to culture *A. anomala* on artificial media has contributed significant insight and evidence of the biotrophic nature of this organism. In addition, axenic cultures of the pathogen have been used successfully as a source of antigen for the development of an ELISA-based method for the early detection of incubating infections within inoculated hazelnut genotypes (C. J. Coyne, S. A. Mehlenbacher, R. O. Hampton, J. N. Pinkerton, and K. B. Johnson, *unpublished*).

Ascospores of *A. anomala*

Ascospores are the only known natural means of dissemination of *A. anomala* (8). The two-celled ascospores mature in perithecia during autumn and are released during and after rains (22). Within the asci, ascospores are surrounded by a thick mucilaginous matrix, which may serve to preserve the spores from the time of maturation until release. This mucilage also is important in the initial adhesion of ascospores to host tissue (24). In western Oregon's mild, rainy climate, most ascospores (>70%) are released in late autumn and winter (20), when the host is not susceptible to infection. Ascospore production in perithecia is apparently determinate, and thus the average quantity of ascospores released with rains remains relatively steady through the winter and early spring but then declines rapidly in late April. Ascospore release generally ceases in May and does not begin again until new perithecia mature the following autumn.

Spore-trapping studies in orchards (20) have shown that there are distinct episodes of spore release that correspond with the most intense periods of rain. During these episodes, perithecia hydrate and swell greatly, and ascospores exuded en masse from perithecia can be observed on the surface of stromata. Large numbers of

Table 1. Incidence of eastern filbert blight[x] in seven European hazelnut cultivars after inoculation[y] of vegetative buds and shoots with ascospores of *Anisogramma anomala* at seven different host developmental stages

Cultivar	Disease incidence (%)							
	Dormant	Partial leaf emerged	Full leaf emerged	Multiple leaves emerged	Shoots with 2–4 nodes	Shoots with 8–10 nodes	Shoot growth ceased	LSD[z]
Barcelona	0	81	78	96	80	31	0	16
Casina	0	64	74	85	58	27	0	15
Daviana	0	93	91	97	80	26	0	14
Ennis	0	82	97	90	90	57	0	2
Tonda di Giffoni	0	31	26	72	61	7	0	16
Hall's Giant	0	58	95	79	80	22	0	15
Willamette	0	33	94	96	92	34	0	14
Average	0	63	79	88	77	29	0	

[x] Disease was evaluated in June or July of the year following inoculation (12).
[y] At each developmental stage, 12 buds or shoots on each of four trees of each cultivar were sprayed with a suspension of *A. anomala* ascospores. After inoculation, trees were covered with a plastic bag, incubated in a greenhouse at 20 to 26°C for 7 days, then returned to an outdoor shadehouse.
[z] Fisher's protected least significant differences at $P = 0.05$.

Fig. 6. Eastern filbert blight cankers containing rows of elliptical stromata of *Anisogramma anomala*. The cankers are perennial and expand annually at rates of up to 1 m of branch length per year.

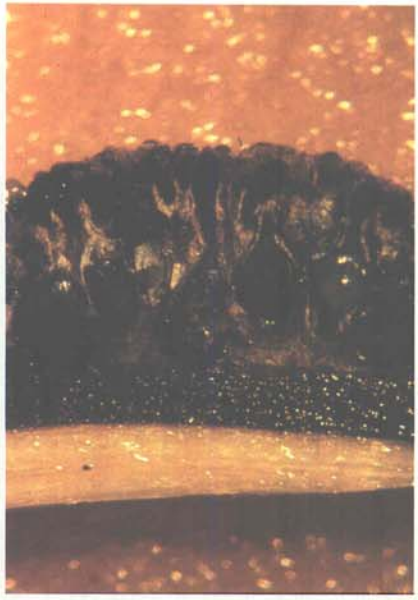

Fig. 7. Cross section of a stroma of *Anisogramma anomala*. Each stroma contains 50 to 100 perithecia. Ascospores in the perithecia mature in autumn and are released during rains from autumn to spring.

released spores are captured in tree canopy throughfall (22), which suggests that rain-splash is an important mechanism of dispersal. Ascospores also have been captured with Burkard spore traps (20), which selectively sample air, as opposed to splash drops. This latter result indicates that some ascospores are discharged forcibly from perithecia into the air currents. Disease gradients, measured by placing potted hazelnut trees at 10-m intervals up to 150 m from the edge of a diseased orchard, also are shallower than would be expected if rain-splash dispersal was the only mechanism of dissemination (20; J. Pinkerton, *unpublished*). Patterns of disease incidence within Oregon orchards show that most local spread of disease occurs to the northeast (20; Fig. 8), which is nearly always the direction of the wind in western Oregon during late-winter and early-spring rains (23). This aspect of spore dispersal has been fortunate for the Oregon hazelnut industry, because the epidemic began on the northeastern edge of the hazelnut growing region, and it is likely that dispersal of ascospores to orchards located south of the diseased area has been slowed greatly by these prevailing wind patterns.

In addition to the release of most ascospores when hazelnut trees are not susceptible to infection, ascospores of *A. anomala* are relatively inefficient at establishing infections, requiring an inoculum concentration on the order of 10^5 spores per ml to infect a shoot reliably (12). The hyaline, thin-walled ascospores of this pathogen also are short-lived. For example, in laboratory experiments conducted at temperatures that ranged from 10 to 20°C, the viability of ascospores placed directly onto filter papers disks and held at fixed relative humidities ≥95% declined by 45 to 60% after 12 h (J. Pinkerton, *unpublished*). In contrast, using similar methods, about 50% of ascospores held at fixed relative humidities ≤92% remained viable for periods of 4 to 5 days. Limited survival of ascospores, low infection frequencies, and spore releases when the host is not susceptible to infection may contribute to the relatively slow spread of eastern filbert blight from infected to noninfected areas. However, once the disease has become established within a susceptible orchard, captures of ascospores in rain traps placed near diseased trees average between 10^5 and 10^7 spores per m^2 of trap surface per day during the critical months of March and April when European hazelnut trees are most susceptible to infection (J. Pinkerton, *unpublished*).

Disease Management

Knowledge of the spore biology of *A. anomala* and the infection process has led to significant improvements in the methods used by growers to reduce the impact of eastern filbert blight. Within susceptible orchards (all Oregon orchards are currently planted with susceptible cultivars), hazelnut growers have implemented the use of regular scouting, fungicides, therapeutic pruning, and other methods of sanitation to slow the eastern filbert blight epidemic. None of the control methods, however, has been 100% effective. Fortunately, about 80% of the orchards in Oregon are planted to the moderately susceptible cultivar Barcelona. With this cultivar, experience suggests that a combination of the above procedures should result in many Barcelona orchards remaining in production as orchards of new, disease-resistant cultivars become established over the next 25 years. In contrast, another 15% of the orchards are planted to the highly susceptible cultivar Ennis, for which even the most diligent growers have concluded that eastern filbert blight, once firmly established in the orchard, is very difficult to slow.

Fungicides, including chlorothalonil, copper hydroxide, fenarimol, and propiconazole, have protected European hazelnut from infection by *A. anomala* when applied during March and April, with the earliest effective applications occurring at budbreak when the host becomes susceptible and the last in late April before ascospore releases cease. Complete protection from infection has been obtained with three to five fungicide applications applied on an 8- to 17-day schedule (Table 2) (13). For fungicide applications made at or shortly after budbreak, the nonsystemic protectants, chlorothalonil and copper hydroxide, have been more effective than the locally systemic materials, fenarimol and propiconazole, probably because there is limited green tissue at this time into which these latter chemicals can be absorbed. Fenarimol and propiconazole are effective later in spring when new shoots expand rapidly; fenarimol also has been shown to have curative activity when applied up to a few days after inoculation with *A. anomala* (13; K. Johnson, *unpublished*). Copper hydroxide is labeled for use on European hazelnut. A section 18 emergency exemption from registration has been granted annually since 1990 for use of chlorothalonil to control eastern filbert blight on hazelnut, and since 1994 for use of fenarimol. Propiconazole can be applied only to nonbearing trees in young orchards and nurseries.

Grower testimonials to the beneficial effects of a chemical control program are only now beginning to be received as they compare their sprayed orchards with those that have not been treated. Some growers, however, have chosen not to apply fungicides in diseased orchards because they consider the cost of treatment to be too high relative to the benefits obtained. Others have chosen an intermediate fungicide-use strategy, applying a fungicide in mid-March and perhaps again in early April, when the majority of new infections occur. Tree size and cultivar, orchard age and

Fig. 8. Aerial, false-color infrared photograph of a European hazelnut orchard with eastern filbert blight located in Oregon's Willamette Valley. The X's represent diseased trees first detected in 1988; diamonds represent diseased trees first detected in 1989; and squares represent diseased trees first detected in 1990. The top of the figure is north. The V-shaped pattern of disease incidence is apparently a consequence of prevailing, southwesterly winds that are associated with rains that release *Anisogramma anomala* ascospores during late winter and early spring.

Table 2. Incidence of eastern filbert blight[w] and mean number of cankers per tree for 2-year-old hazelnut trees of cultivars Ennis and Barcelona planted in rows downwind of a diseased hazelnut planting and treated with fungicides in the spring of 1991

		Ennis		Barcelona	
Fungicide treatment[x]	Timing of application[y]	Trees with cankers (%)	Cankers per tree	Trees with cankers (%)	Cankers per tree
Nontreated control		100 a	4.7 a[z]	75 a	1.0 a
Chlorothalonil	bb	56 b	1.6 b	19 b	0.2 b
Chlorothalonil	bb,pl,fl,ml,se	0 c	0.0 c	13 b	0.2 b
Fenarimol	bb,pl,fl,ml,se	50 b	1.0 b	6 b	0.1 b
Chlorothalonil then fenarimol	bb pl,fl,ml,se	13 c	0.1 c	6 b	0.1 b

[w] Disease was evaluated in June or July of the year following inoculation (13).
[x] Fungicide suspensions were sprayed to runoff; rates of chlorothalonil and fenarimol were 0.180 and 0.038 g a.i./liter, respectively.
[y] Tree phenology on dates of fungicide applications was: bb = budbreak (7 March), pl = partial leaf emergence (28 March), fl = full leaf emergence (11 April), ml = multiple leaf emergence (27 April), and se = shoot elongation (7 May).
[z] Means within a column followed by the same letter are not significantly different according to Fisher's protected least significant difference test at $P = 0.05$.

Table 3. Incidence of eastern filbert blight[v] (%) and disease severity responses measured on selected cultivars of European hazelnut exposed in the field to high doses of ascospores of *Anisogramma anomala* in the springs of 1990 and 1993

	1990[w]			1993[x]		
Cultivar	Disease incidence	Cankers per tree	Diseased wood/tree	Disease incidence	Cankers per tree	Diseased wood/tree
Tonda Gentile delle Langhe	100	2.8	0.34	88	2.9	0.32
Ennis	80	2.4	0.18	90	3.6	0.22
Barcelona	70	1.9	0.13	...	...	...
Casina	63	1.0	0.13	83	3.6	0.23
Willamette	72	1.2	0.10	78	2.0	0.07
Tonda di Giffoni	33	0.7	0.08	42	0.7	0.04
Gem	0	0.0	0.00	30	2.0	0.08
Gasaway[y]	0	0.0	0.00	...	...	...
VR6-28[y]	...	...	...	0	0.0	0.00
LSD ($P = 0.05$)[z]	39	1.1	0.15	24	1.7	0.11

[v] Disease was evaluated in June or July of the year following inoculation.
[w] Data from Pinkerton et al. (21).
[x] Data from Osterbauer et al. (19).
[y] Cultivar possesses a single dominant gene for complete resistance (17,18).
[z] Fisher's protected least significant difference at $P = 0.05$.

productivity, nut price, and disease severity all appear to affect whether or not a grower is willing to implement a fungicide-control strategy. In choosing to apply fungicides, growers also need to come to grips with the perennial nature of the disease. Because eastern filbert blight kills trees slowly, the yield benefits obtained from fungicidal protection are not realized until at least 3 to 4 years after application (11). Thus, fungicide-control programs must be viewed as long-term insurance for which gaps in coverage (e.g., extended periods of rainy weather, skipping spraying for a season because nut prices are low) may jeopardize the benefits gained from effective fungicide and pruning treatments made at other times.

Disease scouting and therapeutic pruning of diseased branches are done primarily during the winter, when leaves do not visually obstruct canker locations. Scouting also is recommended in late summer when cankers can be detected by the presence of dead leaves that are still attached to diseased branches (flagging). Pruning cuts must be made 0.6 to 0.9 m below a visible canker because, during the summer, hyphae of *A. anomala* continue to grow asymptomatically beyond the end of the visible canker that formed the previous spring. Diseased branches pruned from trees must be burned or chipped, as the removed cankers will continue to sporulate until the branches have become completely desiccated. There are two principal reasons why therapeutic pruning has been less than 100% effective in controlling this disease. First, the 12- to 16-month incubation period means that, in addition to the cankers that can be observed visually, there are other symptomless infections still in the incubation phase. Second, small cankers on fine branches near the tops of trees (i.e., last year's new shoots) are difficult to detect, even in leafless trees. Typically, by the time a grower first observes a canker, the disease has been present in the orchard for 3 to 5 years. Nonetheless, pruning of diseased branches effectively eliminates a canker and its future inoculum. Pruning also is done regularly to improve yields by enhancing light penetration into the canopy. Thus, when the incidence of disease is light to moderate, pruning provides effective disease suppression and benefits yields. Therefore, growers do not consider pruning for disease control to be prohibitively expensive. Occasionally, within discrete foci of diseased trees, growers will indiscriminately remove all branches but the major scaffold limbs (e.g., following the first detection of disease). The goal of this extreme pruning is to eradicate all incubating infections from an area. Remarkably, established hazelnut trees with good root systems can regrow rapidly and return to production within 3 years. Shoot regrowth on severely pruned trees, however, is very susceptible to new infection and must be protected with fungicides.

Sanitation also is the basis for several additional management recommendations, including the removal and replacement of susceptible pollenizer cultivars in Barcelona orchards. Because European hazelnut flowers are self-incompatible, pollenizer cultivars comprise 5 to 11% of the trees in an orchard. Within orchards of moderately susceptible Barcelona, it is on the highly susceptible pollenizer cultivars (e.g., Daviana) that eastern filbert blight is usually first observed (21). The disease then spreads to surrounding Barcelona trees. Several partially to completely resistant pollenizer cultivars are available as replacements (17). However, the process of replacing all pollenizers in an orchard must be done over a period of 6 to 10 years to allow time for some of the new, disease-resistant trees to grow to a pollen-bearing age before the last of the older, susceptible trees are removed. In addition, it can be difficult to replace a pollenizer growing in the middle of a mature orchard because there is too much shading for rapid growth of young trees. Consequently, some replacement pollenizers are being planted in rows along the windward edges of orchards. A second recommendation is to eliminate volunteer and other nonmanaged hazelnut trees (e.g., abandoned orchards) from perimeter areas surrounding managed orchards. Both volunteer trees and abandoned orchards are common in some areas, and they serve as important reservoirs for the pathogen. These inoculum sources are best eliminated by pushing or pulling the trees out of the ground to prevent the regrowth of shoots that occurs if the root system remains undisturbed.

Diligent application and integration of these control practices have slowed the rate of disease increase in many Oregon orchards with eastern filbert blight. For successful management, the disease must be detected before the pathogen is distributed throughout an orchard, and the number of cankers in and near an orchard must be maintained at low levels. Regular scouting has proven to be a key to early detection, and continued scouting is essential for determining where to focus management efforts. A recent survey of 141 growers who represent 6,500 ha of orchards that are either diseased or in the neighborhood of the disease revealed that 88% use regular scouting as a means to monitor for new disease outbreaks in their orchards. Ninety-eight percent of growers with diseased orchards (2,500 ha) are employing one to several cultural control methods, including pruning, pollenizer replacement, and removal of volunteer seedlings, and 88% of these growers are applying fungicides at least once per season. Of those growers with healthy orchards endangered by the proximity of eastern filbert blight (4,000 ha), 50% are using cultural controls and 43% are making fungicide applications.

Host Resistance

European hazelnut cultivars vary widely in their susceptibility to eastern filbert blight (Table 3). Mature trees of very susceptible cultivars, such as Daviana, Ennis, and Tonda Romana, are killed by eastern filbert blight in just a few years. In contrast, the cultivars Hall's Giant, Willamette, and Barcelona, the latter of which is the principal cultivar in Oregon, have a moderate but insufficient degree of resistance for sustained commercial production. Quantitative resistance to eastern filbert blight appears to exist in two major forms, which determine how readily a shoot becomes infected and how rapidly cankers expand once the pathogen becomes established. The resistance in Barcelona is mostly the latter type, which means that, relative to the highly susceptible cultivars, Barcelona trees have smaller cankers and are able to sustain their growth and yield for a longer time before the disease becomes severe. In contrast, some cultivars, including Tonda di Giffoni and Gem, appear to possess a degree of quantitative resistance that, in addition to restricting canker size, also greatly reduces the frequency of new infections (4,19,21). The degree of resistance in Tonda di Giffoni and Gem is on par with that of some selections of American hazel (*C. americana*) (21); however, these cultivars also lack one or more of the horticultural characteristics needed for commercial production. Several genotypes with commercial potential and a high degree of quantitative resistance have been selected in a breeding effort at Oregon State University. Currently, some of these genotypes (e.g., OSU 243.002) are being planted in a few orchards in diseased areas. The degree of resistance in these genotypes, when combined with pruning and limited chemical control, may be sufficient to reduce the status of eastern filbert blight from a major to a minor disease.

Research at Oregon State University also has identified a single, dominant gene that confers complete resistance to eastern filbert blight (18). This resistance was first observed in a rarely grown pollenizer cultivar named Gasaway. This cultivar has many undesirable characteristics, including low yield, late maturity, long nut shape, and poor kernel quality. Gasaway is heterozygous for the resistance gene; thus, progeny from crossing it with a susceptible cultivar segregate 1:1 for complete resistance (4,18,19). This resistance gene is being transferred to more acceptable selections with a modified backcross procedure, where the horticulturally superior resistant selections in each generation are crossed with superior susceptible selections. Four selections from the first generation (F_1) were released as pollenizer cultivars to replace the susceptible pollenizers used within Barcelona orchards (17). Roughly 3,700 seedlings from the first backcross

generation BC_1 are now in the field. Based on the rapid progress made to date, it is likely that a completely resistant cultivar with acceptable yield and kernel quality will be released from this or the next generation. The nature and potential instability of the resistance conferred by the Gasaway gene is, of course, a major concern. On one hand, some growth of *A. anomala* can occur in Gasaway if it is inoculated by allowing the pathogen to grow across a graft union from a diseased scion of a susceptible cultivar (24). On the other hand, hazelnut researchers and enthusiasts have grown Gasaway or its resistant progeny for up to 20 years in diseased nurseries located in Washington, Michigan, and Illinois. These trees have not yet become diseased by what would be termed a new race of *A. anomala*.

Within the genus *Corylus*, several species in addition to *C. americana* appear to possess significant resistance to eastern filbert blight (4). These species include the beaked hazel (*C. cornuta* Marsh. var. *cornuta*), Pacific hazel (*C. cornuta* var. *californica* (A.DC.) W. Sharp), Turkish tree hazel (*C. colurna* L.), and two Asian shrub species, *C. heterophylla* Fisch. and *C. seiboldiana* Blume. Little is known about the nature of resistance in these species, although only selections of *C. americana*, *C. colurna*, and *C. jacquemontii* Decne. (the Indian tree hazel) were shown to be hosts of the pathogen via inoculation studies (4). Most of the *Corylus* species listed above have been crossed with European hazelnut to produce interspecific hybrids, and inoculation of such hybrids with *A. anomala* resulted in seedlings that have shown a wide range of disease responses. For example, in one experiment, seedlings that were derived from crosses of *C. avellana* with *C. americana*, *C. c.* var. *californica*, or *C. heterophylla* had resulting disease incidences of 0, 58, and 28%, respectively (4). Further studies are in progress to better understand the genetics of resistance in these three species.

Development of new methods for evaluating hazelnut for resistance to eastern filbert blight has been an important part of the rapid progress made in breeding research (15). Prior to 1988, resistance was evaluated in nurseries of trees exposed to natural inoculum, a process that required 8 to 10 years to detect meaningful differences among genotypes (18). Evaluation of quantitative resistance has been accelerated by exposing 2-year-old trees to artificially high amounts of inoculum (19,21). This is accomplished by placing potted trees at budbreak under wire mesh platforms topped with diseased hazelnut branches (Fig. 9). The branches are collected from orchards during the winter. During periods of rain (or supplemental irrigation), ascospores are released from perithecia in the diseased branches into surface water that drips onto the trees below. To evaluate the type of resistance conferred by the single dominant resistance gene, scions of selections that have survived several horticultural evaluations are grafted onto a rootstock and inoculated in the greenhouse. Greenhouse inoculations are repeated three times to ensure a high probability of infection in susceptible genotypes. Six to 9 months after inoculation, shoot tips are tested for the presence of *A. anomala* with an ELISA procedure (C. J. Coyne, S. A. Mehlenbacher, R. O. Hampton, J. N. Pinkerton, and K. B. Johnson, *unpublished*), the results of which are confirmed by microscopic examination of hand-cut sections of xylem sampled from the shoot (4,26). PCR-based DNA markers and primers for the resistance from Gasaway also have been identified using bulked segregant analysis (6). Routine use of these markers offers the potential to efficiently screen a large number of hazelnut seedlings. Only seedlings with the DNA marker for the resistance gene would be planted in the field for further evaluation, and the more expensive, inoculation-based resistance testing would be limited to only the most promising selections from this group.

Fig. 9. Field inoculation of potted, 2-year-old European hazelnut genotypes with ascospores of *Anisogramma anomala*. Hazelnut branches with eastern filbert blight are collected from diseased orchards in winter, and then in early spring are placed onto wire-mesh platforms suspended over the potted trees. Ascospores of *A. anomala* are released from the diseased branches during rains. Ascospore dose is measured by collecting throughfall in a diagonal gutter that drains into the container shown in the lower right.

Oregon Industry Prospects

Eastern filbert blight is firmly established in western Oregon, and over the long term, it is doubtful that the Oregon hazelnut industry can survive without new, disease-resistant cultivars. The breeding effort now being conducted at Oregon State University has a high likelihood of success, but it also requires time to produce and evaluate new selections. New cultivars must then be propagated in large numbers and grown to bearing age, a process that adds 6 to 10 years to the transition process. European hazelnut is propagated traditionally by layering, but several nurseries are now grafting onto rootstocks (14), a propagation method that should hasten deployment of new cultivars. Fortunately, unlike some horticultural crops, hazelnut kernels are not marketed by cultivar name, and many advanced selections have acceptable yield and kernel quality. Therefore, once the new cultivars are selected and propagated, the transition should proceed smoothly.

Hazelnut grower organizations know that the disease must be managed in existing, susceptible orchards in order to buy time for new cultivar development. These organizations, along with the Oregon State University Extension Service, publish and send to all growers pest management guides, meeting proceedings, and a newsletter on the disease that highlights new control methods, research progress, and the need for management. Riker mounts of cankered branches have been posted in all extension offices and at sales desks of many agricultural supply distributors. The Extension Service holds training courses on how to scout for and control eastern filbert blight in orchards, and with the Oregon Department of Agriculture, it has developed regulatory guidelines for nursery production and shipment of edible and ornamental European hazelnut cultivars within the state. New outbreaks of disease within the Willamette Valley are well-publicized by extension agents and specialists.

Several small outbreaks located to the south of the major disease area have been eradicated as a result of these efforts.

The prognosis for Oregon's hazelnut industry is bright despite the seriousness of the threat posed by eastern filbert blight. The research and extension efforts have addressed and continue to address both the short- and long-term needs that will foster the transition to new high-yielding, disease-resistant cultivars. While the industry's prognosis is good, many individual orchards will be destroyed by the disease in the coming years. Initially, the process of orchard replacement also may cause some unforeseen changes, such as a shift in orchard size that favors relatively larger producers. It is also possible that with the introduction of blight-resistant cultivars, European hazelnut will be grown more widely, including in areas of North America where eastern filbert blight once restricted its cultivation.

Acknowledgments

Research presented was supported by the Oregon State Agricultural Experiment Station, by the Agricultural Research and the Cooperative States Research and Extended Education Services of the United States Department of Agriculture, and by the Oregon Hazelnut Commission.

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