Evaluation of Chlorothalonil, Fenarimol, and Flusilazole for Control of Eastern Filbert Blight

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ABSTRACT

Johnson, K. B., Pscheidt, J. W., and Pinkerton, J. N. 1993. Evaluation of chlorothalonil, fenarimol, and flusilazole for control of eastern filbert blight. Plant Dis. 77:831-837.

In a greenhouse experiment, treatment of 3-wk-old hazelnut (*Corylus avellana*) seedlings with suspensions of chlorothalonil (1.8 g a.i./L), fenarimol (0.038 g a.i./L), or flusilazole (0.030 g a.i./L) prior to inoculation with ascospores of *Anisogramma anomala* resulted in 4, 5, and 7% incidence of eastern filbert blight, respectively, compared to 53% in the inoculated control. In field experiments, exposure of 1- or 2-yr-old potted trees to natural releases of *A. anomala* ascospores demonstrated that hazelnut is susceptible to infection after vegetative budbreak, and that one to five applications of chlorothalonil, fenarimol, or flusilazole onto vegetative tissues that develop after budbreak significantly ($P \leq 0.05$) reduced disease development. Eastern filbert blight was controlled completely when four or five fungicide applications were made beginning at vegetative budbreak in March and extended on a 2–3-wk schedule into May. The relative efficacy of chlorothalonil, a protectant fungicide, appeared to be independent of hazelnut phenology. In contrast, the efficacy of fenarimol, a locally systemic fungicide, may depend on the amount of vegetative tissue into which it can be absorbed.

Eastern filbert blight (EFB), caused by *Anisogramma anomala* (Peck) E. Müller in E. Müller & Arx, is a devastating disease of European hazelnut (*Corylus avellana* L.) that currently threatens Oregon's hazelnut industry (14). Since the early 1980s, when EFB was first introduced into the Willamette Valley of Oregon, nearly all orchards within a 10-km radius of the initial site of disease detection have been destroyed as a result of this disease (14). More recently, EFB spread southwestward into the major hazelnut production areas of the Willamette Valley (14). All cultivars of hazelnut grown commercially in Oregon are susceptible to the disease (12). In the future, resistant cultivars will likely reduce the threat of EFB (8,9,12); however, until these cultivars represent a significant proportion of production, there is a need for management practices that will limit the impact of the disease in susceptible plantings.

The development of chemical methods for control of EFB has been hindered by an incomplete understanding of the infection biology of *A. anomala*. Recently, Stone et al (16) demonstrated that European hazelnut is susceptible to infection by ascospores of *A. anomala* after vegetative buds break dormancy in March through leaf emergence and shoot elongation, the latter of which extends into May. Consequently, an effective chemical control program for EFB must protect trees from new infections over a period of susceptibility that may extend up to 2 mo. Once established, infections of *A. anomala* in European hazelnut systemically colonize the cambium, causing progressive dieback of branches and limbs at an average rate of 0.3 m/yr (3).

In preliminary studies (4–6,15), we identified several fungicides that show potential for a control program for EFB. The growth of *A. anomala* in culture was inhibited by low concentrations (<0.01 μg a.i./ml) of the locally systemic fungicides fenarimol and flusilazole, and by the protectant fungicide chlorothalonil (5,6). In a field study conducted prior to the results of Stone et al (16), chlorothalonil applied in mid-February and again in mid-March reduced the number of new EFB cankers by 63% (15). In contrast, another field experiment, also conducted without a full understanding of when infection by *A. anomala* occurs, resulted in a similar level of disease control when fenarimol was applied once in early May (4).

The purpose of this study was to evaluate the potential for the fungicides chlorothalonil, fenarimol, and flusilazole to control EFB in susceptible hazelnut cultivars and to learn the number and timing of fungicide applications needed for effective disease control.

MATERIALS AND METHODS

Greenhouse study. Three-week-old hazelnut seedlings grown from open-pollinated seed of the cultivar Royal were inoculated with ascospores of *A. anomala* and incubated at 17–25 C for either 4, 7, or 14 days in an intermittent mist chamber constructed on a greenhouse bench. A fine mist was applied intermittently for 30 sec every 10 min during daylight (16). Ascospore suspensions were prepared by crushing perithecia of *A. anomala* in sterile distilled water. Perithecia were excised from diseased hazelnut twigs that had been collected the previous fall and stored at −10 C. The resulting suspension was filtered through three layers of cheesecloth, and the spore concentration was adjusted to 1×10^5 spores per milliliter with a hemacytometer. Approximately 0.5 ml of the spore suspension was sprayed onto the shoot tip near the apical meristem of each experimental seedling immediately before placing it in the mist chamber. Suspensions of chlorothalonil at 1.8 g a.i./L (Bravo 720), fenarimol at 0.038 g a.i./L (Rubigan 1EC), or flusilazole at 0.030 g a.i./L (Nustar 20DF) were applied to the seedlings to runoff with a hand-held pump sprayer either 4 hr before inoculation or after the period of postinoculation mist.

Individual replications of the experiment were initiated on 5 and 19 April and 31 May 1990. Each replication had 10 seedlings per fungicide × mist treatment and 10 seedlings in a nontreated control. After incubation in the mist chamber, the seedlings were grown and overwintered in an unheated greenhouse for 12–13 mo (temperature range from −10 to 35 C). We evaluated the incidence of disease (percent trees infected) in May 1991 after stromata of *A. anomala* became obvious on the trunks of infected trees. Data obtained from preinoculation and postmist fungicide treatments were analyzed separately. Each data set was analyzed as a split plot design with fungicide as the whole plot and length of the mist treatment as the subplot.

Field studies. In 1990 and 1991, the efficacy of chlorothalonil, fenarimol, and flusilazole for control of EFB was evaluated in field experiments (described below) at three study sites. Each site used a different method to expose hazelnut trees to ascospores of *A. anomala*: 1) artificial exposure, 2) exposure within a diseased orchard, and 3) exposure downwind of a line source of diseased trees. In all experiments, fungicides were applied to initially healthy, 1- or 2-yr-old

Oregon Agricultural Experiment Station Technical Paper 10,146.

Accepted for publication 20 April 1993.

trees that were propagated from seed or cloned at a commercial nursery. Fungicide suspensions were sprayed to runoff with a backpack sprayer equipped with a hand-held wand and adjustable hollow-cone nozzle. In early summer of the year following the fungicide applications, incidence of disease, tree mortality, and number and total length of cankers per tree were recorded.

Artificial exposure experiment. In a field on Washington State University's Southwest Washington Research and Extension Center (WSU SWREC) in Vancouver, potted hazelnut trees were placed under three 3 × 4 m wire mesh platforms that were elevated 1.8 m above the ground and topped with diseased hazelnut branches. A total of 240 branches 1 m long and 2–4 cm in diameter were positioned uniformly on the wire mesh. Methods for collection and storage of diseased branches were described previously (12). An automated sprinkling system installed on each inoculum platform (six nozzles per platform) applied water at 0.8 mm/min over three 5-min periods between 5:00 and 6:00 each morning. One set of trees was placed under the inoculum platforms in December 1989 and removed on 30 April 1990, and the other set was under the platform from 19 March to 30 April 1991. After these exposures, the trees were planted and grown in an irrigated field until external symptoms of EFB developed in June the following year.

Fungicides were applied to the trees under the inoculum platforms in spring 1990 and 1991. In 1990, the 1-yr-old seedlings grown from open-pollinated seed of cultivar Royal were arranged in a randomized block design with 13 treatments (including nontreated control), six replications (two per inoculum platform), and five trees per plot. Four treatments received chlorothalonil at 1.8 g a.i./L on one or more of the following dates: 27 February (budbreak), 12 March (partial leaf emergence), 27 March (full leaf emergence), and 13 April 1990 (shoot elongation). Fenarimol at 0.038 g a.i./L and flusilazole at 0.030 g a.i./L were each applied to three treatments on one or more of the following dates: 12 and 27 March and 13 April. One treatment received chlorothalonil on 27 February followed by fenarimol on 12 and 27 March; another was sprayed with chlorothalonil on 27 February and 12 March, followed by fenarimol on 27 March and 13 April.

In 1991, 2-yr-old hazelnut trees of cultivar Barcelona were arranged in a design similar to that used in 1990, except that flusilazole treatments were not included and the number of replications was reduced to five because of tree loss due to freeze injury. Treatments were applied on 19 March (partial leaf emergence, chlorothalonil only), 27 March (full leaf emergence), 11 April (multiple leaf emergence), and 27 April (shoot elongation).

Orchard exposure experiments. Two separate experiments were conducted within a 12 ha, 50-yr-old hazelnut orchard near Troutdale, Oregon, which had been abandoned 4 yr prior to the study. In the first experiment, 2-yr-old trees of cultivar Ennis were interplanted in January 1990 and again in January 1991 between mature orchard trees that were severely diseased with EFB. The experiment was arranged in a randomized block design with six replications. An individual plot consisted of two (1990) or three (1991) trees planted 0.5 m apart. In spring 1990, two-tree plots were treated with fenarimol at 0.038 g a.i./L or flusilazole at 0.030 g a.i./L on 28 March and 13 and 30 April, which corresponded to partial leaf emergence, full leaf emergence, and shoot elongation, respectively. In 1991, the same rates of fenarimol and flusilazole were applied to three-tree plots on 2, 12, and 23 April and 7 May, which corresponded to partial leaf emergence, full leaf emergence, multiple leaf emergence, and shoot elongation, respectively. Each year, two plots in each replication served as nontreated controls.

In the second experiment, healthy 2-yr-old trees of hazelnut cultivars Barcelona and Butler were interplanted in another part of the diseased orchard in January 1990 and again in 1991. A split plot design was used with eight fungicide treatments (including nontreated control) and eight replications. Whole plots were cultivars, and the subplots were fungicide treatments. Each subplot was comprised of two trees of the same cultivar planted 1 m apart. In 1990, five treatments received chlorothalonil at 1.8 g a.i./L on one or more of the following dates: 8 March (budbreak), 26 March (partial leaf emergence), and 9 April (full leaf emergence). Two treatments received fenarimol at 0.028 g a.i./L or flusilazole at 0.030 g a.i./L at partial leaf emergence (28 March) and again at full leaf emergence (9 April). In 1991, fungicides were applied only to trees planted in January of that year. As in 1990, chlorothalonil at 1.8 g a.i./L was applied to five treatments on one or more of the following dates: 28 February (budbreak), 12 March (partial leaf emergence), and 12 April (full leaf emergence). Two treatments received fenarimol at 0.028 g a.i./L or flusilazole at 0.030 g a.i./L on 12 April and again on 27 April (shoot elongation).

Downwind exposure experiment. In January of 1991, healthy 2-yr-old hazelnut trees of cultivars Ennis and Barcelona were planted on the WSU SWREC in three rows on the downwind (north) side of a planting of hazelnuts heavily diseased with EFB. Spacing between the rows was 2.5 m, and individual trees were spaced 0.3–1 m apart. The experiment was arranged as a split plot design with seven fungicide treatments (including a nontreated control), two trees of each cultivar per subplot, and six replications. Three fungicide treatments were chlorothalonil at 1.8 g a.i./L, fenarimol at 0.038 g a.i./L, or flusilazole at 0.030 g a.i./L, each applied on 7 March (budbreak), 28 March (partial leaf emergence), 11 April (full leaf emergence), 27 April (multiple leaf emergence), and 7 May (shoot elongation). Two fungicide treatments received chlorothalonil at budbreak (7 March) followed by fenarimol or flusilazole on the other four treatment dates. Chlorothalonil applied only at budbreak was the sixth fungicide treatment.

Measurement of ascospore release. In both the artificial exposure experiment and the orchard exposure experiment, the release of *A. anomala* ascospores from diseased branches into rain water (2,13) was verified by placing spore traps under the branches. The spore trap design used under the inoculum platforms consisted of a semicircular gutter made by bisecting a 4.4-m length of a 2-cm-diameter polyvinyl chloride pipe (12). One gutter-type trap was positioned under each of the three inoculation platforms. Within the diseased orchard, the release of ascospores of *A. anomala* into rain was monitored by fixing a 12-cm-diameter plastic funnel directly under a sporulating canker on a branch 1–3 cm in diameter. Five funnel-type traps

Table 1. Incidence of eastern filbert blight in hazelnut seedlings[w] inoculated with an ascospore suspension of *Anisogramma anomala* and treated with a fungicide either before inoculation or after a postinoculation mist treatment

Fungicide	Rate[x] (g a.i./L)	Seedlings infected (%)	
		Preinoculation treatment[y]	Postinoculation treatment[y]
Inoculated control	...	53.3 a[z]	53.3 a
Chlorothalonil	1.800	4.4 b	45.6 ab
Fenarimol	0.038	5.5 b	32.2 bc
Flusilazole	0.030	6.7 b	21.0 c

[w] Inoculated 3 wk after emergence.
[x] Fungicide suspensions were sprayed to runoff.
[y] Treatment means were averaged over postinoculation mist durations of 4, 7, and 14 days. Analysis of variance is based on arcsine $\sqrt{x}$; values presented are nontransformed means.
[z] Means within a column followed by the same letter are not significantly different according to Fisher's protected least significant difference test at $P = 0.05$.

were positioned at different locations within the orchard. Both types of spore traps drained into plastic reservoirs to which 50 ml of 5% copper sulfate (w/w) had been added as a preservative. Reservoirs containing trapped rain or irrigation water were collected every other week in the artificial exposure experiment and weekly in the orchard exposure experiment. The number of ascospores released during each collection period was estimated by microscopically examining a 0.80-μm cellulose nitrate membrane through which a subsample of the captured rain water had been filtered (16).

Data analyses. For all experiments, the SAS (Statistical Analysis Systems, Cary, NC) analysis of variance (ANOVA) procedure was used to test whether the fungicide treatments significantly affected the incidence of EFB, and number and total length of cankers per tree. To reduce nonhomogeneity of within-treatment variances, disease-incidence data were transformed with the arcsine $\sqrt{x}$ transformation. Similarly, the number of cankers per tree and total canker length per tree were transformed by taking the $\log_{10}$ of raw data values. Fisher's protected least significant difference test ($P = 0.05$) was used to compare the statistical similarity of the obtained means.

The mortality of trees in the first year of the artificial exposure experiment influenced the statistical analysis procedure. Data were analyzed by first performing an ANOVA on the proportion of trees in each treatment that died prior to symptom development. Subsequent ANOVAs to evaluate the effect of fungicide treatment on disease incidence, number of cankers, and total canker length were performed with data from which the dead trees had been subtracted. Significant cultivar × fungicide interactions occurred in the first year of the orchard exposure experiment and in the downwind exposure experiment. Consequently, mean separation procedures for the effect of fungicide on the disease variables were applied separately to each cultivar.

Spore trap data were summarized by converting the number of ascospores captured by each rain trap during a collection period to $\log_{10}$ (ascospores collected per square meter of trap surface area per day). This value was averaged among rain traps for each collection period.

RESULTS

Greenhouse study. Application of chlorothalonil, fenarimol, or flusilazole prior to inoculation with *A. anomala* significantly ($P \leq 0.05$) reduced the incidence of EFB by an average of 90% compared to the inoculated control. However, the degree of disease control did not differ among fungicide treatments (Table 1). The duration of the mist treatment did not affect the proportion of plants that became infected.

The application of fungicides 4, 7, or 14 days after inoculation with *A. anomala* resulted in significant ($P \leq 0.05$) main effects for fungicide and mist duration. The incidence of disease in trees treated with fenarimol and flusilazole averaged over mist duration was 32.2 and 21.0%, respectively, compared to 45.6% in trees treated with chlorothalonil and 53.3% in the inoculated control (Table 1). Twenty-five percent of trees misted for 4 days became diseased compared to 44% of trees misted for 7 or 14 days. The fungicide × mist duration interaction was not significant ($P > 0.05$); however, for trees misted for 4 days and treated with chlorothalonil, fenarimol, or flusilazole, disease incidence was 25, 23, and 5%, respectively, compared to 47% for the inoculated control. Mist for 7 days followed by treatment with fenarimol or flusilazole resulted in 33 and 16% infected trees, respectively, compared to 60 and 68%, respectively, in the inoculated control and the chlorothalonil treatments.

Artificial exposure experiment. Ascospores of *A. anomala* were released from diseased branches during every spore collection period from late February to early May of each year (Fig. 1). In 1990, the average rate of ascospore release during a collection period ranged from 1×10^5 to 7×10^6 spores/m^2/day; in 1991, the rate was between 8×10^4 and 2×10^6 spores/m^2/day (Fig. 1).

In the experiment conducted in 1990, many of the experimental trees obtained from open-pollinated seed of cultivar Royal died before cankers developed. Similar proportions of dead trees occurred in the nontreated control and in trees treated with fenarimol, flusilazole, or once or twice with chlorothalonil (Table 2). However, three or four applications of chlorothalonil significantly reduced ($P \leq 0.05$) tree mortality (Table 2). The number and total length of EFB cankers per tree also were significantly reduced ($P \leq 0.05$) by three or four applications of chlorothalonil (Table 2).

In the 1991 experiment, less than 12% of trees of cultivar Barcelona that received chlorothalonil at partial leaf emergence (19 March) were infected compared to 80% of nontreated control trees (Table 2). Delaying the first application of chlorothalonil until full leaf emergence (27 March) resulted in increased incidence of disease to an average of 48% regardless of the number of additional fungicide applications (Table 2). No disease developed in trees that received applications of chlorothalonil at partial and full leaf emergence followed by applications of fenarimol at multiple leaf emergence (11 April) and shoot elongation (27 April).

Orchard exposure experiments. With-

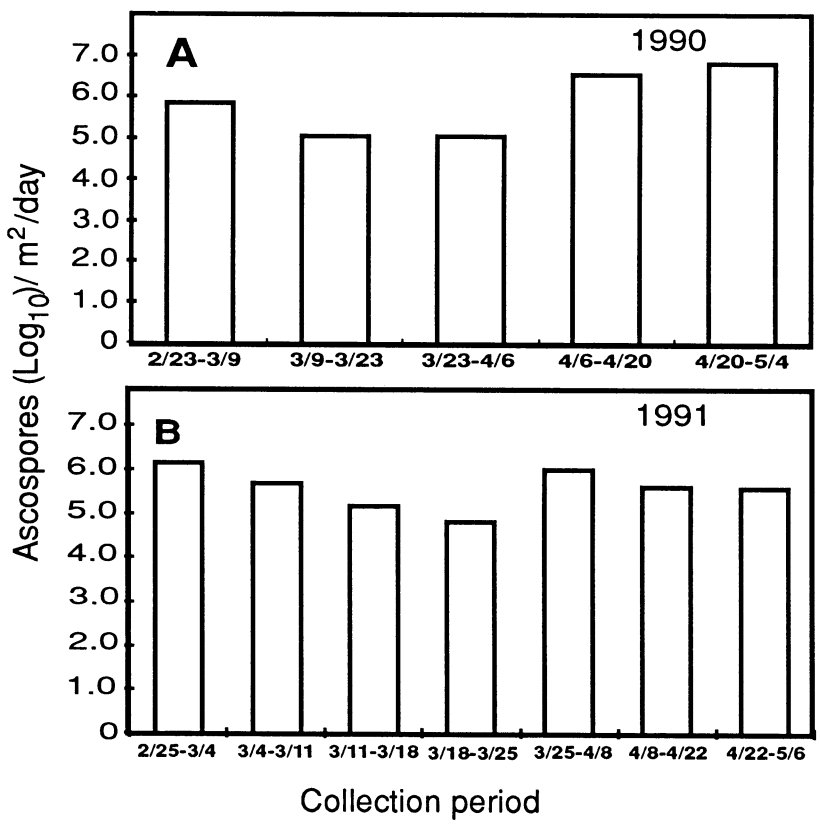

Fig. 1. Rate of recovery of ascospores of *Anisogramma anomala* in rain traps placed under hazelnut branches containing perithecia of the fungus elevated on wire mesh platforms 1.8 m above the ground.

Table 2. Incidence of eastern filbert blight and tree mortality[t], number of cankers, and total canker length per tree for 1-yr-old hazelnut seedlings (1990) and 2-yr-old hazelnut trees cultivar Barcelona (1991) treated with fungicides and exposed to ascospores of *Anisogramma anomala*

	1990					1991			
Fungicide treatment[u]	Timing of fungicide application[v]	Trees dead[w] (%)	Trees with cankers[w] (%)	Cankers per tree[x] (no.)	Total canker length[x] (cm)	Timing of fungicide application[v]	Trees with cankers[w] (%)	Cankers per tree[x] (no.)	Total canker length[x] (cm)
Nontreated		47 a[y]	86 ab	11 b	125 ab		80 ab	3.4 a	42.7 a
Chlorothalonil	bb	46 ab	83 ab	11 b	108 ab	pl	8 d	0.2 d	0.1 e
	bb pl	36 ab	93 a	13 ab	98 ab	fl ml	47 c	0.8 c	6.1 c
	bb pl fl	14 cd	75 bc	4 c	31 c	fl ml se	50 c	1.1 bc	13.9 bc
	bb pl fl ml	6 d	60 c	3 c	44 c	pl fl ml se	12 d	0.2 d	2.4 d
Fenarimol	pl	54 a	88 ab	15 ab	98 ab	fl	92 a	3.9 a	46.3 a
	pl fl	43 ab	100 a	15 ab	112 ab	fl ml	75 ab	1.6 b	14.5 bc
	pl fl ml	36 ab	100 a	12 b	96 ab	fl ml se	59 bc	1.4 bc	19.1 b
Flusilazole	pl	48 a	93 a	20 a	132 a		...[z]	...	...
	pl fl ml	40 ab	88 ab	13 ab	123 ab		...	...	...
	pl fl ml	24 bc	100 a	11 b	75 b		...	...	...
Chlorothalonil then fenarimol	bb pl fl	30 abc	89 ab	10 b	108 ab	pl fl ml	4 d	0.1 d	0.5 de
Chlorothalonil then fenarimol	bb pl fl ml	33 a	93 a	11 b	107 ab	pl fl ml se	0 d	0.0 d	0.0 e

[t] Significant mortality of trees attributable to eastern filbert blight was only observed in the first year of the experiment.
[u] Fungicide suspensions were sprayed to runoff; rates of chlorothalonil, fenarimol, and flusilazole were 1.8, 0.038, and 0.030 g a.i./L, respectively.
[v] Tree phenology on fungicide application dates was: bb = budbreak (27 February 1990), pl = partial leaf emergence (12 March 1990 and 19 March 1991), fl = full leaf emergence (27 March 1990 and 1991), ml = multiple leaf emergence (13 April 1990 and 11 April 1991), and se = shoot elongation (27 April 1991).
[w] Analysis of variance based on arcsine $\sqrt{x}$; values presented are nontransformed means.
[x] Analysis of variance based on $\log_{10}(x+1)$; values presented are nontransformed means.
[y] Means within a column followed by the same letter are not significantly different according to Fisher's protected least significant difference test at $P = 0.05$.
[z] Treatment was not included in experiment.

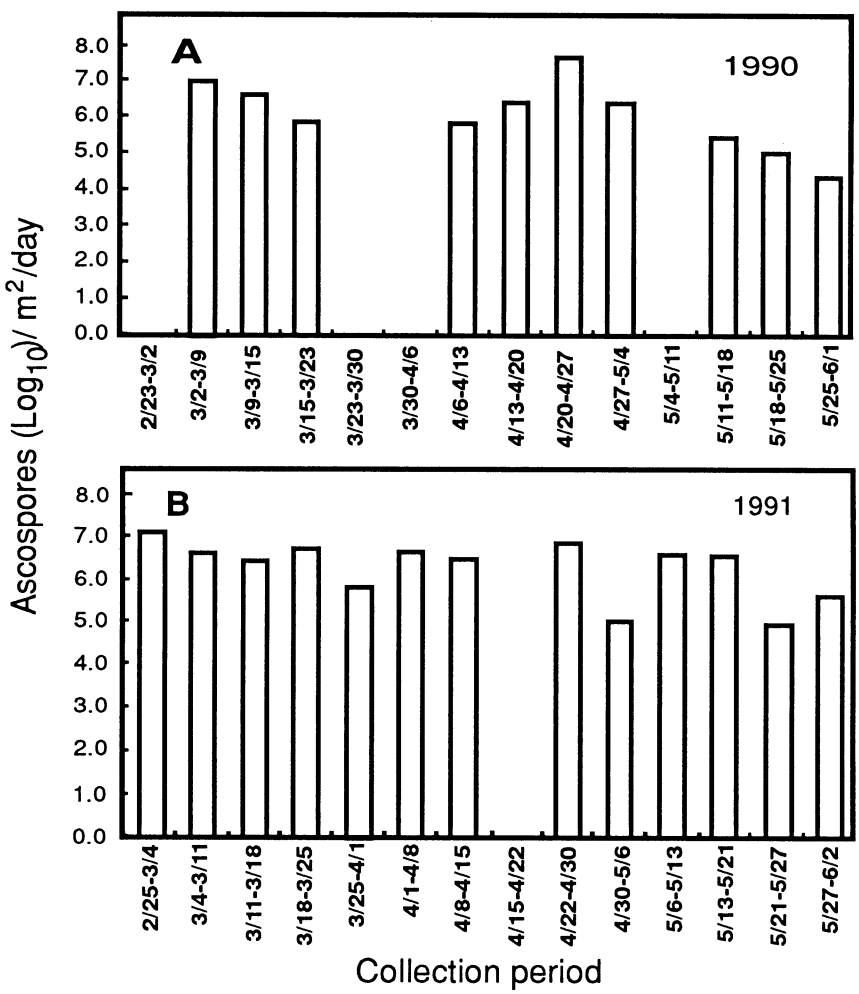

Fig. 2. Rate of recovery of ascospores of *Anisogramma anomala* in rain traps placed under individual eastern filbert cankers containing perithecia of the fungus in 50-yr-old hazelnut trees.

in the abandoned orchard, ascospores of *A. anomala* were recovered in rain traps in 10 of 14 collection periods in late winter and spring of 1990, and in 13 of 14 collection periods over the same period in 1991 (Fig. 2). Measurable rain did not occur during collection periods for which ascospores were not recovered. In 1990, the largest release of ascospores (5.6×10^8 spores/m^2/day) occurred from 20 to 27 April (multiple leaf emergence to shoot elongation). For 1991, the largest weekly releases of ascospores occurred between 25 February and 4 March (1.8×10^7 spores/m^2/day, budbreak) and between 22 and 30 April (8.1×10^6 spores/m^2/day, multiple leaf emergence) (Fig. 2).

In the 1990 experiment with cultivar Ennis, trees treated with fenarimol or flusilazole at partial leaf emergence, full leaf emergence, and shoot elongation had a significantly ($P \leq 0.05$) lower incidence of disease, fewer cankers per tree, and less total canker length per tree than nontreated controls (Table 3). In 1991, disease incidence in nontreated trees averaged 89% with 2.8 cankers per tree, but no disease developed when flusilazole was applied four times beginning at partial leaf emergence (Table 3). Four applications of fenarimol resulted in 17% incidence of disease and an average of 0.2 cankers per tree (Table 3).

In the second experiment, more infections occurred on cultivar Barcelona than on Butler during the spring of 1990 (Table 4). On Barcelona, an application of flusilazole at partial leaf emergence (28 March) and again at full leaf emer-

gence (9 April) resulted in 38% infected trees with 0.8 cankers per tree compared to 88% of nontreated trees with 3.4 cankers per tree (Table 4). Trees of cultivar Butler that received chlorothalonil at budbreak (8 March) and at partial and full leaf emergence (26 March and 9 April, respectively) had 6% infected trees and 0.1 canker per tree; the nontreated control trees averaged 81% infected trees and 2.8 cankers per tree (Table 4).

In 1991, similar amounts of disease developed on both cultivars. Application of the first chlorothalonil treatment at budbreak (28 February) resulted in 17% infected trees (Table 4). When the first chlorothalonil application was delayed until partial leaf emergence (12 March), 63% of the trees were infected (Table 4). In contrast, two applications of fenarimol or flusilazole, the first of which was delayed until full leaf emergence (12 April), resulted in 29 and 19% infected trees, respectively (Table 4).

Downwind exposure experiment. Significantly ($P \leq 0.05$) less disease developed on cultivar Barcelona than on Ennis. All fungicide treatments significantly ($P \leq 0.05$) reduced disease development on both cultivars (Table 5). On Barcelona, there was no significant difference among the fungicide treatments; but on Ennis, the most effective treatments were five applications of chlorothalonil or flusilazole (Table 5). Applying chlorothalonil on Ennis only at budbreak (7 March) resulted in partial disease control. Application of chlorothalonil at budbreak followed by applications of fenarimol at partial, full, and multiple leaf emergence and at shoot elongation (28 March, 11 and 27 April, and 7 May, respectively) resulted in significantly better ($P \leq 0.05$) disease control than application of only fenarimol over the same five dates (Table 5).

Table 3. Incidence of eastern filbert blight, number of cankers, and total length of cankers per tree on 2-yr-old hazelnut trees of cultivar Ennis interplanted among 50-yr-old diseased hazelnut trees in an abandoned orchard and treated with fungicides on three or four dates[u] in spring 1990 and 1991[v]

	1990			1991		
Fungicide treatment[w]	Trees with cankers[x] (%)	Cankers per tree[y] (no.)	Total canker length[y] (cm)	Trees with cankers[x] (%)	Cankers per tree[y] (no.)	Total canker length[y] (cm)
Nontreated control I	92 a[z]	4.0 a	17.2 a	89 a	2.8 a	31.8 a
Nontreated control II	83 a	4.0 a	24.1 a	89 a	2.7 a	31.7 a
Fenarimol	8 b	0.2 b	1.7 b	17 b	0.2 b	2.6 b
Flusilazole	17 b	0.3 b	0.5 b	0 c	0.0 b	0.0 c

[u] In 1990, 28 March and 13 and 30 April, which corresponded to partial leaf emergence, full leaf emergence, and shoot elongation, respectively. In 1991, 2, 12, and 23 April and 7 May, which corresponded to partial leaf emergence, full leaf emergence, multiple leaf emergence, and shoot elongation, respectively.
[v] Different sets of initially healthy trees were sprayed each year.
[w] Fungicide suspensions were spray applied to runoff; rates of fenarimol and flusilazole were 0.038, and 0.030 g a.i./L, respectively.
[x] Analysis of variance based on arcsine $\sqrt{x}$; values presented are nontransformed means.
[y] Analysis of variance based on $\log_{10}(x + 1)$; values presented are nontransformed means.
[z] Means within a column followed by the same letter are not significantly different according to Fisher's protected least significant difference test at $P = 0.05$.

DISCUSSION

This is the first report of complete control of EFB under natural conditions by fungicide applications. All treatments that attained complete control required four or five fungicide applications beginning near vegetative budbreak and continuing on a 2–3 wk schedule into early May. The need for multiple fungicide applications was not unexpected given the length of the period that ascospores are naturally released from cankers (13, Fig. 2). In addition, Stone et al (16), in two seasons, showed that *A. anomala* infected potted hazelnut trees which were sequentially exposed within a diseased orchard for 2-wk periods beginning at budbreak and extending through leaf emergence and shoot elongation. In Virginia, Kosztarab et al

Table 4. Incidence of eastern filbert blight, number of cankers, and total length of cankers per tree for 2-yr-old hazelnut trees of cultivars Barcelona and Butler interplanted among 50-yr-old diseased hazelnut trees and treated with fungicides in the springs of 1990 and 1991[s]

		1990						1991[x]		
		Barcelona			Butler					
Fungicide treatment[t]	Timing of fungicide application[u]	Trees with cankers[v] (%)	Cankers per tree[w] (no.)	Total canker length[w] (cm)	Trees with cankers[v] (%)	Cankers per tree[w] (no.)	Total canker length[w] (cm)	Trees with cankers[v] (%)	Cankers per tree[w] (no.)	Total canker length[w] (cm)
Nontreated control		88 ab[y]	3.4 a	25 a	81 a	2.8 a	19 a	69 a	1.4 a	16 a
Chlorothalonil	bb	75 abc	2.5 ab	16 ab	88 a	2.4 a	16 a	13 c	0.1 c	2 c
	bb pl	63 abcd	2.5 ab	18 a	44 bc	1.2 b	11 ab	19 bc	0.3 bc	5 bc
	bb pl fl	50 cd	1.3 bcd	10 bc	6 d	0.1 c	1 c	19 bc	0.2 c	2 c
	pl fl	56 bcd	1.3 bcd	8 bc	31 cd	1.1 bc	9 bc	50 ab	0.6 ab	8 b
	fl	69 abcd	1.5 bc	13 ab	25 cd	0.6 c	6 bc	75 a	1.4 a	16 a
Fenarimol	pl fl	94 a	2.5 ab	17 ab	44 bc	1.3 b	9 bc	...	...	...
	fl se	...[z]	...	...	...	...	...	29 bc	0.3 bc	2 c
Flusilazole	pl fl	38 d	0.8 d	6 c	67 ab	1.1 bc	9 bc	...	...	...
	fl se	...	...	...	...	...	...	19 bc	0.2 c	2 c

[s] Different sets of initially healthy trees were sprayed in each year of the experiment.
[t] Fungicide suspensions were sprayed to runoff; rates of chlorothalonil, fenarimol, and flusilazole were 1.8, 0.028, and 0.030 g a.i./L, respectively.
[u] Tree phenology on fungicide application dates in 1990 was: bb = budbreak (8 March), pl = partial leaf emergence (26 March [chlorothalonil] or 28 March [fenarimol and flusilazole]), and fl = full leaf emergence (9 April). Phenology on dates in 1991 was: bb = budbreak (28 February), pl = partial leaf emergence (12 March), fl = full leaf emergence (12 April), and se = shoot elongation (27 April).
[v] Analysis of variance was based on arcsine $\sqrt{x}$; values presented are nontransformed means.
[w] Analysis of variance was based on $\log_{10}(x + 1)$; values presented are nontransformed means.
[x] The main effect for cultivar was not significant in this year; thus means presented were averaged over both cultivars.
[y] Treatment means within a column followed by the same letter are not significantly different according to Fisher's protected least significant difference test at $P = 0.05$.
[z] Treatment was not included in experiment.

(7) reported that an annual program of five applications of mancozeb at 3.6 kg a.i./ha (Manzate 200) reduced EFB in a planting of *C. avellana* seedlings over a period of several years. However, neither the degree of disease control attained nor the phenology of the trees with respect to the timing of the mancozeb applications was provided in their report.

The methods developed in this study to evaluate fungicides for control of EFB required hazelnut trees that had not been infected by *A. anomala* in a previous year. The systemic, perennial cankers produced by this disease make it difficult to maintain healthy, uniform trees for experimental purposes. Furthermore, the incubation time from infection to development of external symptoms of EFB is usually 13–15 mo but occasionally 25–27 mo (16). This long, variable incubation period could potentially confound the interpretation of results if the same trees were used for experimentation in successive years.

Chlorothalonil, fenarimol, and flusilazole were chosen for evaluation based on their superior ability to inhibit the growth of *A. anomala* in culture (5,6) and on results obtained in preliminary field trials (4,15). The greenhouse study demonstrated that each of these chemicals effectively controls EFB when applied shortly before inoculation. Results obtained in the greenhouse also indicate that fenarimol and flusilazole can partially control the disease when applied up to 7 days after inoculation. The temperature of the greenhouse during inoculation and misting was comparatively warmer (17–25 C) than usual spring conditions in the Willamette Valley (mean daily temperature 6–18 C). Thus, the effect of temperature on the postinoculation efficacy of locally systemic fungicides for control of EFB needs further investigation.

The 1990 artificial exposure experiment revealed disease-control activity in the protectant fungicide, chlorothalonil, that was not observed in the locally systemic materials. In this study, three and four chlorothalonil applications were the only treatments to have a significant effect on disease development (Table 2). We attribute the superior results obtained with chlorothalonil to the residual and redistributive properties of this fungicide (1,10), especially over the 17-day interval between last fungicide application (13 April) and the end of the experiment (30 April), when the trees were exposed to high ascospore doses (Fig. 1). Hazelnut seedlings obtained from open-pollinated trees of cultivar Royal are extremely susceptible to EFB, which is likely the reason that disease-induced mortality and canker length were higher in 1990 than in the following year when trees of the moderately resistant cultivar Barcelona were exposed under the inoculum platforms (Table 2).

All three fungicides, chlorothalonil, fenarimol, and flusilazole, are potentially valuable components of an EFB control program. Chlorothalonil, because of its protectant activity, may provide superior disease control over the critical period from budbreak to full leaf emergence when temperatures are relatively cool and there is little green, vegetative tissue present on the tree into which locally systemic materials can be absorbed. Evidence for this conclusion was obtained in the 1991 artificial exposure experiment (Table 2) and the 1991 downwind exposure experiment (Table 5), where applications of fenarimol were more effective if preceded by an application of chlorothalonil. In contrast, fenarimol and flusilazole may be better suited for preventing infection later in spring after multiple leaves emerge and vegetative shoots begin to expand. On apple, young vegetative tissues absorb higher amounts of fenarimol and flusilazole than do older tissues, and the rate of absorption of these chemicals is enhanced with warmer temperatures (11). Inoculation studies we have conducted (K. B. Johnson, *unpublished*) show that germ tubes rising from ascospores of *A. anomala* penetrate the tips of vegetative shoots via tissue that is less than a week old. Thus, upward translocation of fenarimol and flusilazole to shoot tips may efficiently protect the principal site of entry (11,17).

Economic considerations may influence the rate of adoption of chemical control programs for EFB by Oregon hazelnut growers. Yields, premiums paid for nut quality, tree size, orchard size, and the level of host resistance to EFB vary greatly among orchards. Consequently, the expense associated with multiple fungicide applications will be viewed differently by individual producers.

Table 5. Incidence of eastern filbert blight, number of cankers, and total canker length per tree for 2-yr-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[v]	Timing of fungicide application[w]	Trees with cankers[x] (%)	Cankers per tree[y] (no.)	Total canker length[y] (cm)	Trees with cankers[x] (%)	Cankers per tree[y] (no.)	Total canker length[y] (cm)
Nontreated control		100 a	4.7 a[z]	67.8 a	75 a	1.0 a	9.2 a
Chlorothalonil	bb	56 b	1.6 b	19.2 b	19 b	0.2 b	2.5 b
Chlorothalonil	bb pl fl ml se	0 c	0.0 c	0.0 c	13 b	0.2 b	3.4 b
Fenarimol	bb pl fl ml se	50 b	1.0 b	12.1 b	6 b	0.1 b	1.3 b
Flusilazole	bb pl fl ml se	19 c	0.3 c	3.8 c	6 b	0.1 b	0.6 b
Chlorothalonil then fenarimol	bb pl fl ml se	13 c	0.1 c	0.8 c	6 b	0.1 b	0.9 b
Chlorothalonil then flusilazole	bb pl fl ml se	0 c	0.0 c	0.0 c	0 b	0.0 b	0.0 b

[v] Fungicide suspensions were sprayed to runoff; rates of chlorothalonil, fenarimol, and flusilazole were 1.8, 0.038, and 0.030 g a.i./L, respectively.
[w] Tree phenology on fungicide application dates 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).
[x] Analysis of variance was based on arcsine $\sqrt{x}$; values presented are nontransformed means.
[y] Analysis of variance was based on $\log_{10}(x + 1)$; values presented are nontransformed means.
[z] Treatment means within a column followed by the same letter are not significantly different according to Fisher's protected least significant difference test at $P = 0.05$.

ACKNOWLEDGMENTS

The research was funded by the Oregon Filbert Commission, USDA Cooperative States Research Service Special Grant 91-34242-5922, and by the Oregon Agricultural Experiment Station. We thank Steve Cluskey, Karen Theiling, Julie Jenkins, and Teresa Sawyer for technical assistance. We also thank Carl Shanks and the support staff at Washington State University's Southwest Washington Research and Extension Center in Vancouver.

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