Natural Infection Period and Susceptibility of Vegetative Seedlings of European Hazelnut to *Anisogramma anomala*

J. K. STONE, Research Associate, and K. B. JOHNSON, Assistant Professor, Department of Botany and Plant Pathology, Oregon State University, Corvallis 97331-2902; J. N. PINKERTON, Research Plant Pathologist, Agricultural Research Service, U.S. Department of Agriculture, Horticultural Crops Research Laboratory; and J. W. PSCHEIDT, Assistant Professor, Department of Botany and Plant Pathology, Oregon State University, Corvallis 97331-2902

ABSTRACT

Stone, J. K., Johnson, K. B., Pinkerton, J. N., and Pscheidt, J. W. 1992. Natural infection period and susceptibility of vegetative seedlings of European hazelnut to *Anisogramma anomala*. Plant Dis. 76:348-352.

Infection of European hazelnut (*Corylus avellana*) by *Anisogramma anomala*, the cause of eastern filbert blight, was investigated in artificial inoculation experiments and by sequentially exposing potted trees for 1 wk to natural inoculum in an orchard severely infected with the disease. In the orchard exposure experiment, European hazelnut was found to be susceptible to infection in the spring during leaf emergence and new shoot elongation but not before vegetative budbreak, as previously reported. Artificial inoculation experiments confirmed the susceptibility of vegetative buds and shoots over a range of phenological stages of development. In other experiments, 3-wk-old hazelnut seedlings were found to be highly susceptible to infection by *A. anomala* after ascospores of the fungus were placed on vegetative tissues. Established infections were detected in freehand sections of the cambium and outer xylem tissues 1-3 mo after inoculation.

Additional keywords: *Corylus americana*

Anisogramma anomala (Peck) E. Müller in E. Müller & Arx is an endemic parasite of the American hazel, *Corylus americana* Marsh., a common understory shrub in forests of the eastern United States. This fungus also causes the severe disease eastern filbert blight on cultivated European hazelnut, *C. avellana* L. (1). Failure to establish commercial production of *C. avellana* within the natural range of *C. americana* was attributed primarily to this disease (1). Outside the natural range of *C. americana* and *A. anomala*, however, a hazelnut industry was established along the Pacific coast in Oregon, Washington, and British Columbia. Within this region, a quarantine against the importation of *Corylus* spp. was established to protect the industry from the introduction of eastern filbert blight (2). In 1973, *A. anomala* was discovered in a commercial orchard in southwestern Washington (3). Subsequently, the disease has spread into about 800 ha of cultivated European hazelnut in northwestern Oregon and western Washington (6,7).

Morphology, development, and aspects of the infection cycle of *A. anomala* on *C. avellana* were described in a series of papers by Gottwald and Cameron (4-6). Perithecia mature in late summer or early autumn, erupting through the periderm within 2- to 4-mm-diameter stromata (4). Ascospores are the only known spore type and are disseminated in rain from November through May (5,8). Trees exposed to naturally disseminated ascospores require 12-16 mo of incubation to develop symptomatic cankers (5). The pathogen initially attacks young branches and progresses downward into the main scaffold limbs (6). The progressive annual expansion of cankers girdles limbs, usually killing the aboveground portion of mature trees in 6-10 yr (6). New, susceptible sprouts may continue to emerge from the roots, however, for longer periods of time.

Several aspects of the infection cycle and life history of *A. anomala* on *C. avellana* remain unresolved. These include the relationship between host phenology and susceptibility to infection and identification of the host tissues initially invaded by *A. anomala*. Gottwald and Cameron (5) examined the former by sequentially exposing healthy hazelnut trees in a diseased orchard monthly for 1 yr. Six of 120 trees developed symptoms 12-16 mo after exposure during the period from 12 February to 21 May 1978. To investigate the tissues initially invaded by *A. anomala*, Gottwald and Cameron (5) introduced ascospores with a hypodermic syringe into stem wounds, leaf petioles, male catkins, dormant healthy buds, and dormant galled buds infested with the eriophyid mite *Phytoptus avellanae* Nal. Disease symptoms resulted only from inoculation of healthy and mite-galled buds. They concluded that quiescent or dormant buds were the primary site of invasion by *A. anomala* and that the bud mite, *P. avellanae*, facilitated entry of *A. anomala* by creating a loose, open arrangement of galled bud scales and by wounding bud tissue during feeding (5). The latter conclusion was supported by the observation that some cultivars of *C. avellana* susceptible to bud mites also are highly susceptible to eastern filbert blight (2). The low incidence of infection, however, in both the orchard exposure experiment (5%) and under greenhouse conditions in the inoculation experiments (5%) warranted a reappraisal of these conclusions.

The objective of this study was to examine the relationship between host phenology and susceptibility of European hazelnut to infection by *A. anomala*. Results of phenological investigations led to studies of infection of *C. avellana* by ascospores of *A. anomala* applied to nonwounded, nonmite-infested vegetative tissues.

MATERIALS AND METHODS

Orchard exposure experiment. At weekly intervals from 2 February through 2 May 1988 and from 2 December 1988 through 2 May 1989, groups of 30 potted 2-yr-old *C. avellana* trees were sequentially exposed to natural inoculum of *A. anomala* in a severely diseased orchard located in northeastern Multnomah County, OR. Dormant, bare root trees were obtained from a commercial nursery located outside the known geographic distribution of eastern filbert blight. Before exposure, experimental trees were potted in 7.6-L containers and maintained in Corvallis, OR, which also was located outside of the known infected area (J. N. Pinkerton, *unpublished*). Thirteen groups of trees were exposed in 1988 and 22 groups were exposed in 1989. Each weekly exposure group contained 20 trees of cv. Butler and 10 trees of cv. Daviana, which were

Address correspondence to the second author.

Research supported by USDA Agricultural Research Service Specific Cooperative Agreement 58-401-7-00169 and the Oregon Agricultural Experiment Station. Published as technical paper 9655 of the Oregon Agricultural Experiment Station.

Accepted for publication 18 November 1991 (submitted for electronic processing).

© 1992 The American Phytopathological Society

evenly distributed over five sites within the orchard. After exposure, potted trees were moved to an outdoor holding site located in southern Washington County at which the trees were maintained for 12–27 mo. This site was located in an area relatively free of eastern filbert blight (2 km from the closest known infected orchard) and complied with Oregon Department of Agriculture regulations on movement of *Corylus* plant material from areas infested with the disease. Trees were inspected for stromata of *A. anomala* in June 1989 and June 1990. Symptomatic trees from 1988 were destroyed in June 1989; asymptomatic trees from 1989 were held for an additional year and reexamined in June 1990.

In both years, precipitation and tree phenology were monitored in the diseased orchard during the exposure periods. In addition, during the second season of the study, the temporal distribution of discharge and removal of *A. anomala* ascospores from cankers by rain was monitored at five sites within the disease orchard. At each site, ascospores in rain were collected by fixing a 12-cm-diameter funnel to a metal post placed directly under the canopy of one of the 40-yr-old diseased trees. Funnels were positioned 1 m above the ground; each funnel drained into a 2-L plastic bottle into which 15 ml of a 5% (w/w) $CuSO_4$ solution had been added to inhibit ascospore germination and prevent other microbial growth in the trapped rain water. Plastic bottles were changed weekly, corresponding with placement and removal of potted trees. In the laboratory, a subsample of the rain water in each bottle was filtered through a 0.80-μm cellulose nitrate membrane (Whatman Ltd., Maidstone, England). Membranes with trapped spores were stained for 6–8 hr in a solution of 0.05% (w/w) trypan blue in lactoglycerine. Stained membranes were mounted on a glass slide, and the number of *A. anomala* spores was determined by examining each membrane microscopically (×250). Spore samples for each week were averaged over site and converted to the number of ascospores released per square meter per day.

Inoculation experiments. A series of four inoculation experiments were conducted from April 1988 through August 1989. In each experiment, ascospore suspensions were prepared for inoculation by dissecting whole perithecia from mature stromata of *A. anomala* on *C. avellana* twigs collected the previous fall and stored frozen at −10 C in polyethylene bags. Just before inoculation, perithecia were hydrated and crushed in sterile distilled water. Ascospores were collected with a drawn pasteur pipet and diluted to 1×10^5 spores per milliliter. A similar pipet was used to apply 0.5–1 ml of the spore suspension onto the surfaces of vegetative buds and/or shoots of each experimental tree. Ascospore germination was verified on a growth medium developed for cultivation of *A. anomala* (9).

Two inoculation experiments were concerned with the phenological stage of development of vegetative buds of *C. avellana* and susceptibility to infection by ascospores of *A. anomala*. In the first experiment, five trees each of 2-yr-old potted cvs. Butler and Daviana were inoculated on 6, 11, and 26 April 1988. The timing of the inoculations coincided with leaf budbreak and initial emergence of vegetative shoots. Five to seven buds were inoculated on each tree; the trees then were incubated for 14–26 mo in an unheated, rain-sheltered shadehouse. Symptoms of eastern filbert blight were recorded after 14 and 26 mo of incubation. In the second phenological inoculation experiment, 12 groups of 20 1-yr-old potted hazelnut trees grown from seed produced on open-pollinated *C. avellana* 'Royal' were sequentially inoculated with a suspension of *A. anomala* ascospores at weekly intervals from 2 February to 28 April 1989. The phenological condition of buds was noted for each group of trees at the time of inoculation. Trees were inoculated and maintained in an unheated, rain-sheltered shadehouse for 14–18 mo until symptoms developed. An additional 60 trees were maintained in the shadehouse as noninoculated controls.

Remaining inoculation experiments were done with 2- to 6-wk-old hazelnut seedlings grown from seed obtained from open-pollinated cv. Royal. In the first experiment, 80 seedlings were inoculated in February 1989 with a suspension of *A. anomala* ascospores in a greenhouse maintained at 20–25 C. Inoculated seedlings were covered with polyethylene bags for 24 hr and then grown on greenhouse benches and periodically examined for symptoms of infection. A group of 20 trees from this seed lot were kept as noninoculated controls. Inoculated trees were examined histologically for the presence of *A. anomala* hyphae within stems beginning 10 wk after inoculation. Fresh, freehand sections one to two cells thick were dissected from subperidermal stem tissue, stained in a drop of 0.05% trypan blue in lactophenol on a glass slide, and covered with a cover glass. The slide was heated over a flame to remove air trapped in the plant tissue and then examined microscopically (×100–×400) for the presence of *A. anomala* hyphae. Nine months after inoculation, 20 of the inoculated seedlings with infection confirmed histologically were transferred to the unheated shadehouse to induce a dormancy over the winter season. The following May, these trees were examined for stromata of *A. anomala*.

In the second seedling inoculation experiment, the effect of moisture on infection by *A. anomala* was investigated by inoculating groups of 25 3-wk-old hazelnut seedlings with a suspension of *A. anomala* ascospores. Immediately after inoculation, groups of seedlings were placed in an intermittent mist chamber (fine mist for 30 sec every 10 min during daylight hours, 20–25 C) for 0, 3, 7, or 14 days. One replication was initiated on 14 June 1989 and a second replication with new seedlings began on 10 August 1989. After the mist periods, seedlings were grown on greenhouse benches and examined periodically for symptom development. Care was taken to avoid wetting the foliage for 3–4 wk after removal from the mist chamber. Histological techniques described above were used to determine the presence of *A. anomala* hyphae in stems 100 days (14 June replication) or 40 days (10 August replication) after inoculation. Incidence of seedling mortality (death) was recorded 280 and 223 days after inoculation for the 14 June and 10 August replications, respectively. Data from each replication were combined and statistically analyzed by regressing incidence of *A. anomala* hyphae in stems and seedling mortality on duration of postinoculation mist treatment. In addition, a correlation coefficient was computed for incidence of *A. anomala* and seedling mortality.

RESULTS

Orchard exposure experiment. In the 1988 and 1989 field studies, infection did not occur in 235 trees exposed in the diseased orchard before early March (Table 1). Fifty-two percent of the trees (132 of 253) exposed for 7-day intervals beginning in early March through early May developed symptoms after 13–15 mo. For both seasons, the earliest infections corresponded to the time of initial leaf budbreak (Table 1), and subsequent infections corresponded to periods of leaf emergence and new shoot development. Discharge of *A. anomala* ascospores, monitored during the second season of the study, began in early December 1988 and continued through late April 1989 (Table 1). Unusually cold winters during both years of the orchard experiments contributed to high rates of mortality for the potted trees. Accordingly, infection percentages shown in Table 1 are based on the number of trees surviving in each exposure group.

For trees exposed in 1988, 17 of 156 (11%) trees that were alive and symptomless after 13–15 mo developed stromata of *A. anomala* after 25–27 mo (Table 1). Furthermore, 16 of 17 trees that developed symptoms after 27 mo were in weekly exposure groups that had symptomatic trees the previous year (Table 1).

Overall, for exposure groups that contained at least one infected tree, the incidence of infection among surviving

trees of cultivars Daviana and Butler was similar over both years: 57 and 40%, respectively, in 1988 and 52 and 53%, respectively, in 1989.

Phenological inoculations. Of the 30 trees inoculated in April 1988, 26 remained alive 14 mo after inoculation, of which 21 (80%) developed stromata of *A. anomala*. Incidence of infection was 90, 100, and 60% for April 6, 11, and 26, respectively. Most trees had two to three cankers, indicating successful infection of more than one bud per tree. The remaining five trees without symptoms remained healthy after 26 mo.

Results of weekly inoculations between 2 February and 28 April 1989 showed a pattern of susceptibility similar to the orchard exposure experiments. Infection did not result from inoculation on 2 and 9 February 1989 before leaf budbreak. One tree became infected from inoculation on 16 February 1989; seven to 13 trees were infected in each of the nine remaining 20-tree groups inoculated weekly between 23 February and 28 April 1989. Average disease incidence for this time period increased from 35 and 40% on 23 February and 8 March, respectively, to between 55 and 65% for the four inoculation dates in April. Phenologically, vegetative buds were tightly closed on 2, 9, and 16 February but swollen with loose bud scales on 23 February and 8 March. Leaf tips were visible on 8 March, and full leaf emergence occurred between 24 and 31 March. Multiple leaves on new shoots had emerged on most trees by 7 April. The group of 60 noninoculated trees from the same seed source remained healthy during the experimental period.

Seedling inoculations. In the first inoculation experiment with 2- to 6-wk-old *C. avellana* seedlings, most inoculated plants were stunted, and many had abnormal stems and symptoms of interveinal chlorosis or necrosis on leaves by 8–10 wk after inoculation. Over half of the trees with these symptoms died within 3 mo of inoculation. Symptomatic seedlings kept in a heated greenhouse, and thus prevented from becoming dormant, did not develop stromata of *A. anomala* the following spring. In contrast, stromata were produced after 14–15 mo of incubation on infected seedlings placed in an unheated shadehouse and allowed to become dormant. Noninoculated controls were vigorous and had normal growth. Histological examination of cambial tissue and outer xylem of diseased trees revealed the presence of intracellular hyphae characteristic of *A. anomala* in xylem vessels (Fig. 1) (4).

Three-week-old seedlings inoculated with *A. anomala* and held for varying periods in intermittent mist became infected and displayed stunting and leaf chlorosis or necrosis 10–12 wk after inoculation. For the 14 June replication, disease incidence was 38, 100, 100, and 100% among groups of inoculated seedlings misted for 0, 3, 7, and 14 days, respectively. Disease incidence for the second replication averaged 42% in seedlings that received a postinoculation mist treatment compared with 4% for nonmisted plants. When data from both replications were combined, the regression relationships for incidence of infection and seedling mortality both increased with the length of the postinoculation mist treatment (Fig. 2A and B), but only the latter relationship was statistically significant ($P = 0.045$). Among mist treatments, incidence of mortality was significantly correlated ($r = 0.89$, $P < 0.01$, df = 6) with incidence of infection.

DISCUSSION

In both orchard exposure experiments and the 1989 weekly inoculation experiment, infection of *C. avellana* by *A. anomala* first occurred near the time of leaf budbreak in spring. Inoculations and exposure periods subsequent to leaf budbreak also resulted in successful infections. These observations, combined with data from the other inoculation experiments, suggest that actively growing vegetative tissues (opening buds,

Table 1. Incidence of eastern filbert blight in 3- to 4-yr-old hazelnut trees 14 and 26 mo after exposure to natural inoculum[a] of *Anisogramma anomala* during the winter and spring of 1988 and 1989

Exposure period	Vegetative bud phenology[b]	Rainfall during period (mm)	Percentage of plants infected after period		Number of plants surviving[d]	Ascospore discharge (spores/m²/day)
			14 mo	26 mo[c]		
1988						
2 February–7 March[e]	Quiescent, scales tight	57[f]	0	0	81	ND[g]
7 February–14 March	Swollen, scales loose	37	0	8	13	ND
14 March–21 March	Swollen, scales loose	13	67	95	21	ND
21 March–28 March	Leaf tips emerged	114	68	72	22	ND
28 March–4 April	Partial leaf exposed	74	25	25	20	ND
4 April–11 April	Full leaf emerged	60	24	45	20	ND
11 April–18 April	Multiple leaves emerged	13	45	45	11	ND
18 April–25 April	Shoot elongation	155	56	88	16	ND
25 April–2 May	Shoot elongation	33	29	29	21	ND
1989						
2 December 1988–10 March 1989[h]	Quiescent, scales tight	513	0	ND	154	32,670[i]
10 March–17 March	Swollen, scales loose	61	7	ND	14	89,181
17 March–24 March	Leaf tips emerged	20	30	ND	10	75,749
24 March–31 March	Partial leaf exposed	41	50	ND	8	88,061
31 March–7 April	Full leaf emerged	28	77	ND	9	669,223
7 April–20 April[j]	Multiple leaves emerged	4	75	ND	20	39,020
20 April–26 April	Shoot elongation	46	70	ND	24	326,139
26 April–2 May	Shoot elongation	13	66	ND	24	ND

[a] Groups of 30 2-yr-old potted trees were sequentially exposed under severely diseased trees for 1 wk. Ascospores of *A. anomala* are dispersed in winter and spring during periods of rain.
[b] Average phenology of vegetative buds of potted hazelnut trees at beginning of exposure period.
[c] Cumulative disease incidence 26 mo after exposure.
[d] Number of trees of original 30 in each group surviving after 14 (1989) or 26 (1988) mo.
[e] Because no infected trees were obtained, five weekly exposure groups were combined.
[f] Rain gauge was installed 22 February.
[g] Data were not determined.
[h] Because no infected trees were obtained, 14 weekly exposure groups were combined.
[i] Average discharge of *A. anomala* ascospores for 13 weekly exposure periods from 2 December 1988 to 10 March 1989. No precipitation occurred during two of these exposure periods: 9–15 December 1988 and 26 January to 1 February 1989. Ascospore discharge in remaining exposure periods ranged from 1,515 to 89,181 spores per square meter per day.
[j] Precipitation did not occur between 7 and 13 April 1989; thus, the trees were exposed for an additional week.

elongating shoots) are likely sites of infection for this fungus. Assuming that *A. anomala* penetrates its host via young or newly emerging vegetative tissues, the difference between the earliest observed infection in the 1989 weekly inoculation experiment (16 February) and the 1989 orchard exposure experiment (10 March) were attributable to differences in date of budbreak. Plants used in the weekly inoculation experiment were stored in a sheltered location and were advanced phenologically relative to the trees used in the orchard exposure experiment at similar dates of inoculation and exposure. Also, hazelnut trees that originate from seed produced on open-pollinated flowers were more variable with respect to time of budbreak than were the clonal cultivars used in the orchard exposure study. Furthermore, the high concentration of ascospores used in the inoculation experiment may have increased the likelihood of successful infection in swollen bud tissue.

During the second season of the orchard exposure experiment, ascospores of *A. anomala* were found in rain samples collected in 17 of the 19 weekly exposure periods (Table 1). Although these data were not obtained for the first year of the study, it is likely a similar pattern of ascospore discharge occurred for that season. The temporal distribution of ascospore discharge measured by Gottwald and Cameron (5) was contiguous with rain events from November 1978 through April 1979. In addition, we have recorded similar distributions of ascospore discharge for the winter and spring seasons of 1990 and 1991 (8) (J. N. Pinkerton, *unpublished*).

In all experiments with woody trees, stromata of *A. anomala* were not produced until at least the second growing season after inoculation. This observation agrees with the reported incubation period required for production of stromata by this fungus (5). Observations from seedling inoculations also indicate that a cold period or dormant period in the host is required to initiate stromata formation. In addition, some infections may not produce stromata until more than 24 mo after natural exposure to *A. anomala* ascospores. This conclusion is based on the facts that all exposure groups in the orchard exposure experiment were incubated at the same disease-free holding site, trees at this site that developed stromata after 13–15 mo were destroyed before they became infectious, and 16 of 17 trees that required 25–27 mo to develop stromata were from exposure groups that had trees with symptoms in the previous year.

Stem tissue of 3-wk-old hazelnut seedlings is physiologically similar to the elongating shoots that emerge after leaf budbreak. This similarity, together with small size and susceptibility to infection, makes young seedlings convenient for experimentation. The intracellular hyphae of *A. anomala* within young hazelnut stems were distinctive (Fig. 1), and the use of this characteristic allowed for confirmation of infection 6–12 wk after inoculation. Histological detection of the fungus greatly shortened the time required to assess experimental results compared to the 12- to 16-mo minimum incubation period the fungus requires to produce stromata. Chlorosis and stunting symptoms observed after 2 mo in severely infected seedlings were good indicators for when samples should be taken for histological observation.

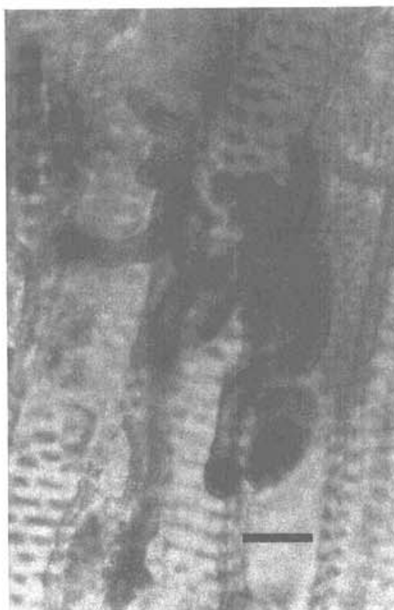

Fig. 1. Hyphae of *Anisogramma anomala* in xylem vessels of a 3-mo-old seedling of *Corylus avellana* 10 wk after inoculation. Scale bar = 20 μm.

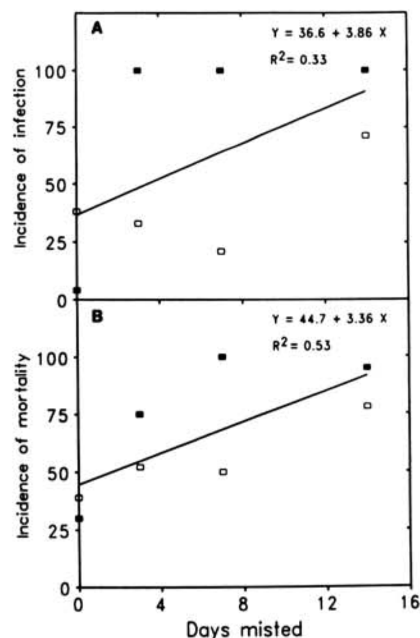

Fig. 2. Incidence of **(A)** eastern filbert blight and **(B)** mortality in young seedlings of *Corylus avellana* inoculated with ascospores of *Anisogramma anomala* and misted intermittently for 0, 3, 7, or 14 days. Twenty-five seedlings were inoculated per treatment on 14 June (■) and 10 August 1989 (□). Incidence of infection was determined histologically 100 and 40 days after inoculation for the 14 June and 10 August replications, respectively. Seedling mortality was determined 280 and 223 days after inoculation for the 14 June and 10 August replications, respectively.

Wetness periods appeared to enhance infection of *C. avellana* by *A. anomala*, but infection of some plants that did not receive a mist treatment after inoculation indicated that long periods of free moisture apparently are not a requisite for infection (Fig. 2). In addition, only short periods of rainfall may be necessary to release spores and inoculate trees. For healthy trees exposed in a diseased orchard from 14 to 21 March 1988, a high incidence of disease was obtained (95% after 27 mo), but only one light rainfall occurred on the morning of 21 March (Table 1).

Results of this study conflict with conclusions of Cameron (2) and Gottwald and Cameron (4–6) regarding the role of the eriophyid mite, *P. avellanae*, in infection of *C. avellana* by *A. anomala*. These researchers established infections of *A. anomala* in *C. avellana* by injecting ascospores into quiescent, vegetative buds with a hypodermic syringe on three dates: 12 December 1977 and 10 January and 10 February 1978. A total of 413 2-yr-old, greenhouse-grown trees were inoculated by this method, but only 21 trees (5%) showed symptoms after incubation for 24 mo. Although a similar number of healthy and mite-galled trees became infected, they concluded that mite-galled buds facilitated entry of *A. anomala* into the plant by creating a loose arrangement of bud scales and mite-induced feeding wounds. Successful inoculation of healthy buds was attributed to mechanical injury caused by the syringe. Our study found the period of natural susceptibility to infection by *A. anomala* did not begin until close to leaf budbreak and extended over the period when actively growing, vegetative tissue was present on trees in spring. Moreover, few of our clonal trees and none of our seedlings were infested by *P. avellanae* at the time of exposure or inoculation. Our experiments do not exclude the possibility that buds galled by infestations of *P. avellanae* lengthen the period of natural susceptibility to earlier in the winter; however, mite-galled buds are relatively infrequent in many diseased orchards. Thus, the role of these mites in the infection cycle of *A. anomala* may be minor.

LITERATURE CITED

1. Barss, H. P. 1930. Eastern filbert blight. Calif. Dep. Agric. Bull. 19:89-490.
2. Cameron, H. R. 1976. Eastern filbert blight

established in the Pacific Northwest. Plant Dis. Rep. 60:737-740.
3. Davidson, A. D., and Davidson, R. M. 1973. Apioporthe and Monochaetia cankers reported in western Washington. Plant Dis. Rep. 57:522-523.
4. Gottwald, T. R., and Cameron, H. R. 1979. Studies on morphology and life history on *Anisogramma anomala*. Mycologia 71:1107-1126.
5. Gottwald, T. R., and Cameron, H. R. 1980. Infection site, infection period, and latent period of canker caused by *Anisogramma anomala* in European filbert. Phytopathology 70:1083-1087.
6. Gottwald, T. R., and Cameron, H. R. 1980. Disease increase and the dynamics of spread of canker caused by *Anisogramma anomala* in European filbert in the Pacific Northwest. Phytopathology 70:1087-1092.
7. Pinkerton, J. N., Griesbach, J., Theiling, K. M., and Johnson, K. B. 1990. Incidence of eastern filbert blight in Oregon hazelnut orchards. (Abstr.) Phytopathology 80:970.
8. Pinkerton, J. N., Johnson, K. B., Stone, J. K., and Pscheidt, J. W. 1990. Ascospore discharge of *Anisogramma anomala* under field and controlled conditions. (Abstr.) Phytopathology 80:1031.
9. Stone, J.K. 1991. Axenic culture of *Anisogramma anomala* from ascospores. (Abstr.) Mycol. Soc. Am. Newsl. 42:35.