Disease Increase and the Dynamics of Spread of Canker Caused by *Anisogramma anomala* in European Filbert in the Pacific Northwest

T. R. Gottwald and H. R. Cameron

Research plant pathologist, USDA-SEA-AR, Southeastern Fruit and Tree Nut Research Laboratory, Byron, GA 31008, and professor of plant pathology, respectively, Department of Botany and Plant Pathology, Oregon State University, Corvallis 97331.
This research was supported in part by the Nut Growers Society of Oregon, Washington, and British Columbia.
The authors wish to thank Bob and Mary Powelson for their helpful comments and John Lee Andersen and Dan Sisemore for skillful field assistance.
Portions of a Ph.D. thesis submitted to Oregon State University by the first author.
Oregon State University Agricultural Experiment Station Technical Paper 5320.
Accepted for publication 15 May 1980.

ABSTRACT

GOTTWALD, T. R., and H. R. CAMERON. 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.

In southwestern Washington, eastern filbert blight, which is caused by *Anisogramma anomala* (Diaporthaceae), was first discovered in 1973. The rate of disease increase and the geographical pattern of spread from the original focus of infection were studied. Increase in length of individual cankers averaged 31.72 cm/yr. Regression analyses of disease progress curves gave disease increase values of r = 1.085 and r = 1.236 unit per year within single trees (treated as independent epidemics) and disease increase within orchards, respectively. Within the area presently affected the original focus was determined to be a group of five orchards in the northeast quadrant from which the disease spread south and west to 44 additional orchards. Inoculum dispersal over long distances is infrequent; therefore, the southernmost diseased plantings pose the greatest threat of disease spread into orchards farther south in the main filbert-growing areas of Oregon.

Additional key words: disease survey, *Corylus avellana.*

In western Washington, eastern filbert blight, which is caused by *Anisogramma anomala,* was first discovered in 1973 (5). Since that time 49 infected orchards have been found. Incidence of this disease within an individual orchard may be as high as 100%, and in a few cases, entire orchards have been killed by the disease. The fungus enters the tree primarily through galled buds infested with the eriophyid mite *Phytocoptella avellanae* Nal (3,7). Wounds which occur during the dormant season, when callus formation is slow, may remain receptive to infection by *A. anomala* for long periods

0031-949X/80/11108706/$03.00/0

of time (3). This mode of entry also has been reported for other canker-causing fungi (1,6). Early spring infections through galled axillary buds is normally followed by a 10– to 14–mo incubation period prior to symptom expression. Infection takes place from January through April which overlaps the period of ascospore discharge. The vast majority of observations indicate symptoms are not expressed until May or June of the second growing season (8).

Ascospores are discharged the following winter (November–April). Release is closely related to the presence of free moisture. Dispersal over short distances is mainly by splash dispersal of ascospores. This was evidenced by field observations of spore discharge, analysis of spore trap slides, and spore dispersal patterns within individual trees (8). Dispersal over longer distances, often several kilometers, is still in question. Rankin (11) suggested that birds and rodents may vector ascospores of *Endothia parasitica* (Murr.) Anderson, a closely related fungus. It is possible that similar vectors also may play a role in dispersal of *A. anomala* ascospores. No conidial or spermatial stages are known (7).

Susceptibility of *Corylus avellana* cultivars varies greatly. Barcellona, the main commercial cultivar, is moderately susceptible while its commercial pollenizers, Daviana and DuChilly are severely attacked (2). Cultivar susceptibility is correlated with the preference of eriophyid mite infestation (2).

The present study was conducted to describe the dynamics and potential for spread of Anisogramma canker in epidemiological terms and to elucidate the danger of this disease to the filbert industry of the Pacific Northwest. Disease increase was studied as the colonization of filbert branches by individual cankers (infections), the disease increase within individual trees, and disease progress within individual orchards, and a hypothesis was developed for spread of the disease between orchards and over long distances.

MATERIALS AND METHODS

Disease increase of individual cankers, within individual trees, and within the orchard. Disease increase, measured as the number of detectable new individual infections (cankers) per year, was measured for three successive years in a block of 23 Daviana trees in La Center, WA. The Daviana trees were planted every third tree in every third row of an orchard planting 6.1 m between trees in rows 6.1 m apart. The experimental trees were approximately 10 yr old at the onset of the study and were located on the east edge of a severely infected older block. Prevailing winds were west to east. The older infected block served as a line source of inoculum (Fig. 1). Only 14 infections were recorded in the experimental block prior to 1975. New cankers were flagged with surveyor's tape each year as they were discovered. The increase in canker length was measured for both pre-existing and new infections.

Two parameters, the percent total surface area of plant tissue attacked and percentage of the total number of limbs attacked, were estimated visually to determine disease severity. Comparison of "r = values" obtained from both estimates shows that the two correlated very well. The first method gave an extremely low percentage of attack and did not show the true danger of spread from perennial cankers. The second is considered to be more precise and is used as the basis for computations because it better estimates the portion of the tree under attack and in danger from further spread of existing cankers.

Disease spread between orchards. Disease surveys were undertaken in 1977 and 1979 to estimate disease severity in all filbert orchards and minor plantings in Washington and northwestern Oregon. All pollenizer trees in each orchard and planting were examined. The orchards were then assigned a disease severity rating of 1–10 with 1 = 10% of the trees infected, 2 = 20%, and so on. Discussion with the orchard owners and determination of the age of recurring cankers permitted an estimate of the number of years the disease had been present within an orchard and aided in the determination of the original focus of the disease.

RESULTS

Disease increase. *Growth of individual cankers.* Canker growth varied greatly depending upon its location within the tree. Infections that took place through galled axillary buds on small

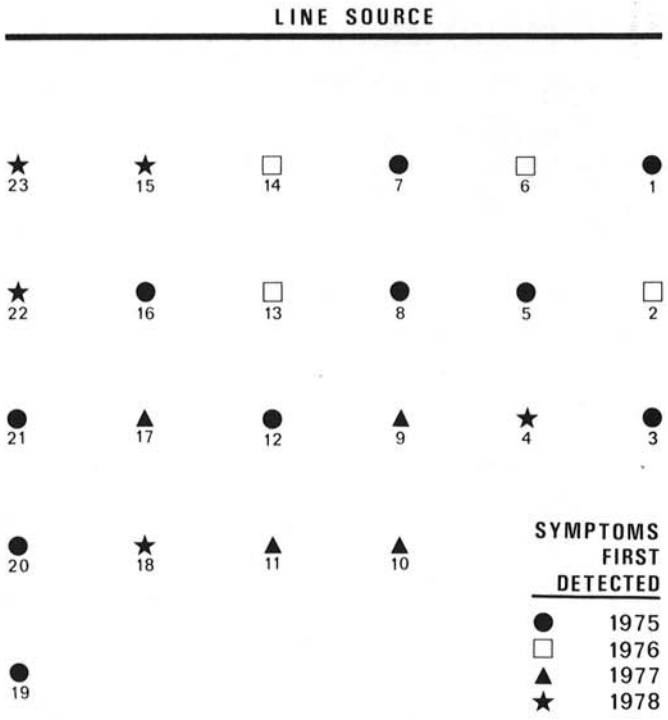

Fig. 1. Map of *Anisogramma anomala* canker increase within an experimental block of European filbert trees showing the location of 23 pollenizers (cultivar Daviana) and their relative distances from the line source of inoculum. • Pollenizers originally diseased in 1975. □ Trees that first developed symptoms in 1976, ▲ Trees that developed symptoms in 1977, ★ Trees that first developed symptoms in 1978.

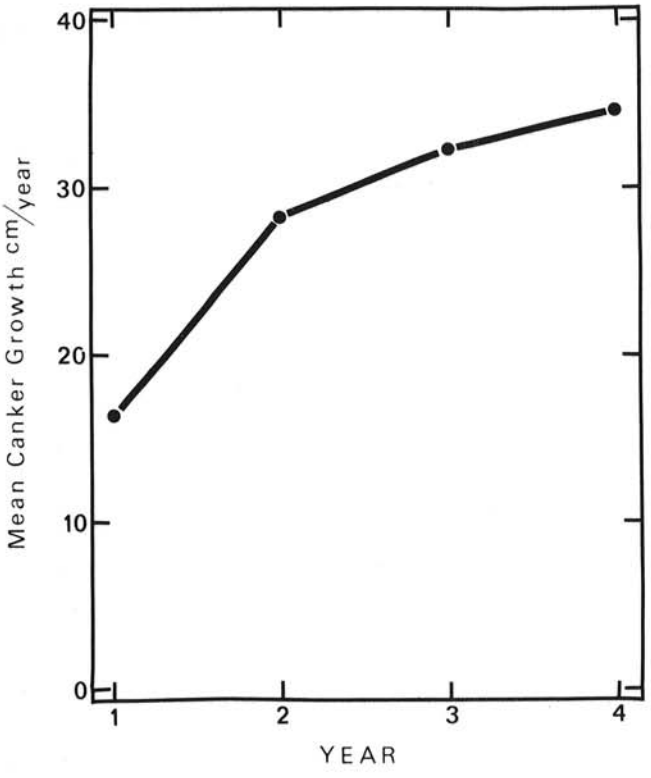

Fig. 2. Mean yearly increase in length of cankers caused by *Anisogramma anomala* on European filbert trees. Measurements represent average growth measured from the edge of the previous year's margin. Differences in growth rates among years were significant at $P = 0.01$.

twigs off a main scaffold limb, usually spread into the main scaffold and expanded for several years before they girdled the limb. During this time the mycelium traveled both up and down the limb and into side branches by colonized phloem tissue. However, infections that took place on small terminal branches, often girdled the branch above within the first or second season. In this case all subsequent spread of mycelium was down and into lower side branches. *A. anomala* grows up or down limbs or into side branches at the same rate. The rate of growth increased significantly with canker age (Fig. 2). However, this increase tended to level off with time, due to girdling and death of terminal tissues which resulted in decreased availability of susceptible tissue for colonization. In many cases areas within individual trees became so heavily infested that cankers began to coalesce.

During the 4 yr of this study 495 individual cankers were measured and recorded. The maximum growth ever recorded for an individual 4-yr-old infection was 189 cm. This was also the largest canker of any age ever recorded in any diseased orchard. This canker extended from the soil line, up the trunk and out a main scaffold limb. It continued into a secondary and tertiary branch. Canker growth ceased when the main scaffold was girdled due to lateral spread from this infection. In the case of individual cankers, spread was from a single locus. There is no production of secondary inoculum during a single season. Therefore, even though the expanding canker is perennial, we still must consider an individual infection to be a simple interest type disease. Yearly mean canker length for the 495 cankers is represented in Fig. 3. By simple regression analysis of average canker length vs canker age in years, the apparent infection rate was found to be $r = 31.72$ cm/yr with a coefficient of determination of 99.8, and 95% confidence limits for disease increase of $r = 37.91$ and $r = 25.53$ cm/yr.

Epidemics within a single tree. Disease progress curves were plotted for 14 of the 23 individual trees for which there were at least 3 years of data. The disease increase within each tree was treated as an independent epidemic. The percent disease for each tree was determined in 1978, and the average portion of each tree occupied by cankers was calculated. The percent infection for the previous years was estimated from this value multiplied by the number of lesions in each year. Figure 4 shows the disease progress curve for seven of these trees. Using Van der Plank's (12) logarithmic transformation for percent disease of a compound interest type, regression lines for the individual epidemics occurring in the 14 independent trees were obtained (Table 1). The individual r-values were combined and a mean slope of $r = 1.085$ units per year was obtained for the 14 epidemics. All of the individual epidemics were adjusted to a common starting point to deal with disease increase alone and to eliminate the necessity of dealing with height differences of the individual regression lines. The height differences correspond to the differences in time of the onset of the individual epidemics and not to the rate of disease increase itself. The variance of each of the individual r-values from the mean r-value was

TABLE 1. Regression analysis[a] of the increase of canker caused by *Anisogramma anomala* within 14 individual European filbert trees in orchards in southwestern Washington

Tree	y-intercept[b]	Slope[c]	Coefficient of Determination
1	−7.512	0.983	0.772
2	−4.589	1.216	0.953
3	−4.238	1.035	0.897
5	−5.664	1.375	0.963
6	−9.128	2.152	0.873
7	−5.069	0.495	0.890
8	−6.017	1.182	0.923
12	−5.887	1.171	0.969
13	−3.044	0.584	0.750
14	−6.484	1.616	0.750
16	−4.100	0.527	0.909
19	−5.522	1.470	0.949
20	−4.643	0.823	0.888
21	−3.493	0.554	0.884

[a] Regression based on calculations of rates of increase for a compound interest disease, $r = [\log_e x_2/(1-x_2) - \log_e x_1/(1-x_1)]/(t_2-t_1)$, in which r is the rate of disease increase and x_2 and x_1 are proportions of diseased tissue at times t_2 and t_1 (in years), respectively.

[b] y-intercept is expressed in terms of $\log_e x_1/(1-x_1)$.

[c] Slope is expressed in terms of r.

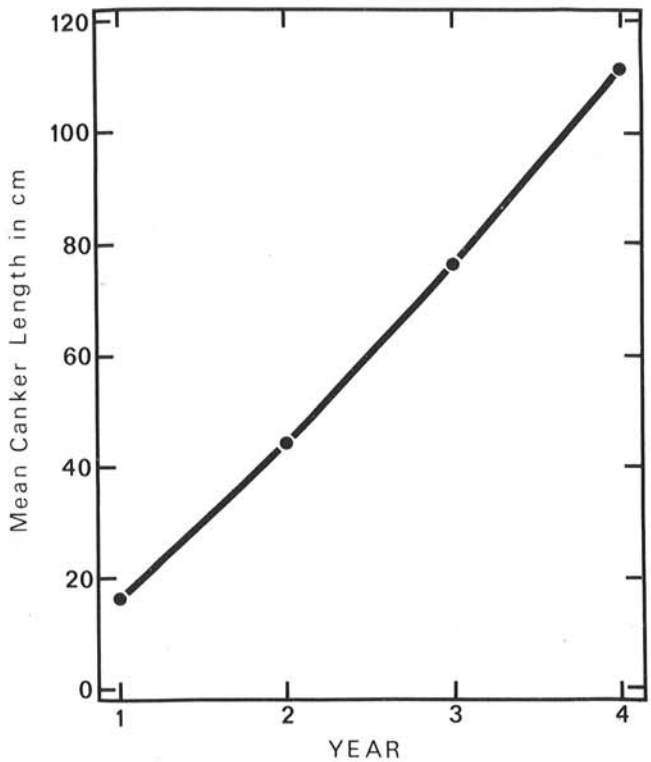

Fig. 3. Cummulative increase in mean length of cankers caused by *Anisogramma anomala* on European filbert trees.

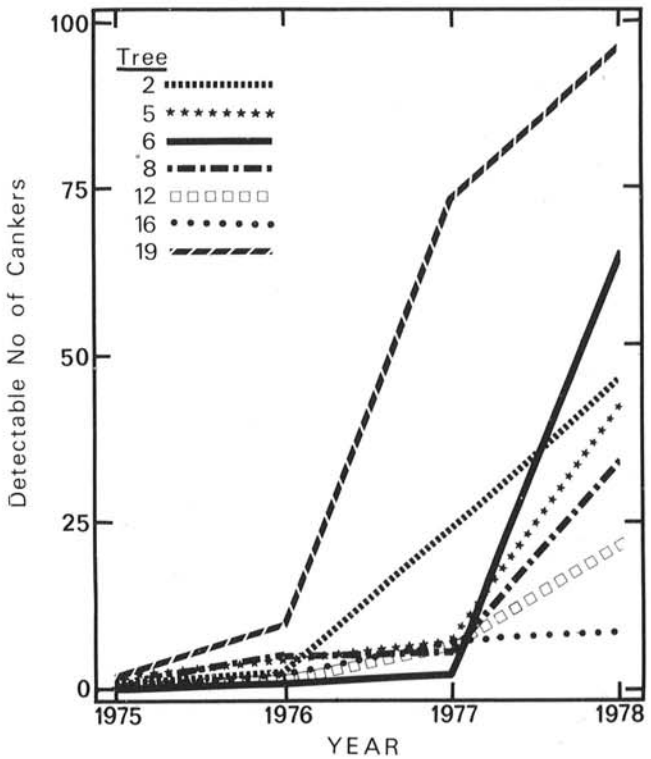

Fig. 4. Disease progress curves for seven of the 23 individual pollenizer trees in the experimental block for *Anisogramma anomala* cankers on European filbert.

calculated and the average variance was computed. This average variance was used to calculate 95 and 99% confidence limits of ± 0.275 and ± 0.384 units per year, respectively.

Rate of spread within an orchard. In 1976, 58 cankers were identified in the 23-tree experimental block. Of these, only 44 cankers were determined to be the result of 1975 infections. The remaining 14 cankers were ⩾ 2 yr old. These 14 cankers were located in 10 different trees. The increase from 14 to 58 cankers encompassed only four more trees in the block. These four trees, in turn, developed 14 new cankers or 31.8% of the total disease increase for 1976. These four trees were located within the first two pollenizer rows from the line source of the older, more severely infected planting. This same pattern was repeated in the next two years; four more trees became infected in 1977 and five in 1978 (Fig. 1).

When the level of infection was low, the major portion of inoculum came from the older established line source. As the disease increased within the new block, secondary foci were established and played an increasingly important role as inoculum sources within the block under study.

Disease increase within the experimental block as a whole, measured as the number of detectable new infections per year, followed the classic curve for disease progress (12) (Fig. 5). At the end of the 4-yr study, disease severity within the block was calculated to be 18.13% of the limbs attacked. Although perennial cankers can eventually girdle limbs, and infect whole trunks, this process may take several years. Therefore, we believe that the disease severity will continue to increase in the experimental block for several years and will eventually reach a level much higher than its present level.

Ascospores are released only during winter and early spring months in one fairly continuous infection period (8). No conidial or spermatial stages are known. Therefore, within a single season the disease is considered to be a simple interest type of disease, but over a period of several years it must be considered to be a compound interest type. Thus, by transforming X, the percent disease, to $\log_e (X/1\text{-}X)$ (12) the apparent infection rate "r" was found to be highly consistent from year to year, averaging 1.24 units per year (Fig. 6). This indicates that the disease is relatively insensitive to year to year variations in weather such as the drought of the 1975–1976 season and the ice storm of the 1977–1978 season.

Disease spread among orchards. Long distance spread is a complicated problem. Several different cultivars and, indeed, different *Corylus* species are used for commercial filbert production. These different genotypes differ in susceptibility, although in general cultivars used as pollenizers are usually more susceptible to Anisogramma canker than are the genotypes composing the bulk of commercial production. In southwestern Washington there are numerous backyard plantings of just a few trees, as well as numerous escapes in the surrounding brush from nuts dropped by birds and rodents. These potentially susceptible trees may act as "stepping stones" between major commercial orchards.

In Oregon and Washington there are 49 orchards located within a 77 km^2 area that are either presently or were previously diseased with eastern filbert blight (Fig. 7). Data collected from the 1977 and 1979 surveys of Washington and Oregon orchards are shown in Figs. 8 and 9, respectively. The numbers correspond to the disease severity rating for each orchard. We believe that the initial outbreak of the disease occurred within the area indicated by the dotted circle (Figs. 8 and 9). This conclusion is based on discussions with individual growers and on estimates of age of perennial cankers. This also correlates with the high severity ratings of the orchard within this area. We believe that the disease has existed in this area for 15–20 yr. Thirteen newly infected orchards were discovered in the 1979 survey. Six of these had not been surveyed in 1977, but due to the relatively low disease severity in these orchards in 1979, we concluded that probably they were free of the disease in 1977. The secondary foci have increased in size even though some of the more severely attacked orchards within the secondary foci were removed.

DISCUSSION

Disease increase measured as growth of individual cankers was highly consistent with only small variations due to spread into side branches or girdling of terminals. At the peak of canker growth, spread from the canker edge of the previous season is 15–20 cm in one direction. After 4–6 yr, the main scaffolds often are girdled and the infection may have spread into the main trunk. A mature tree

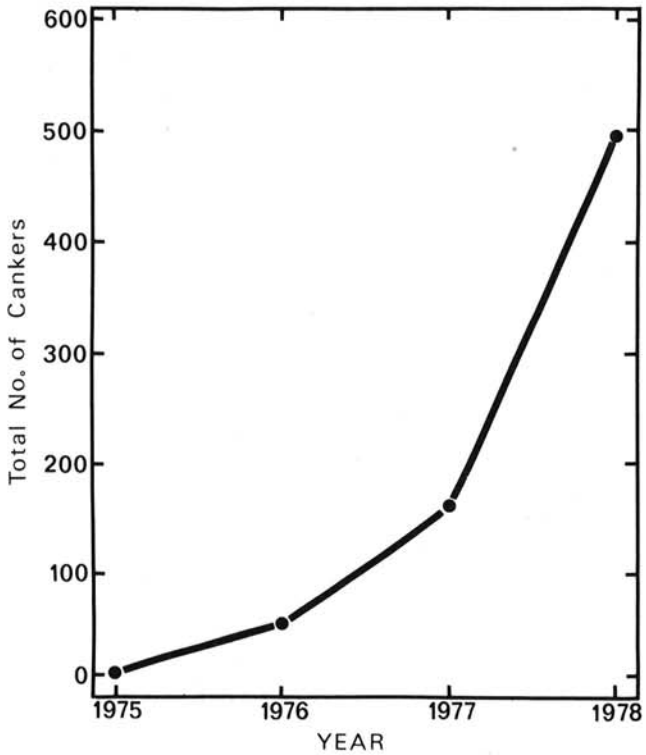

Fig. 5. Increase in numbers of *Anisogramma anomala* cankers within a 23-tree experimental block of European filbert trees from 1975 through 1978.

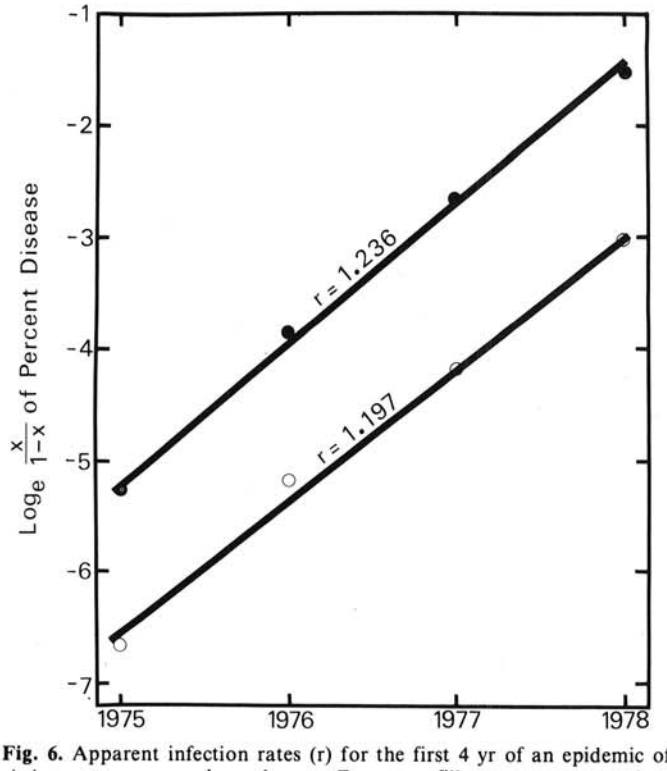

Fig. 6. Apparent infection rates (r) for the first 4 yr of an epidemic of *Anisogramma anomala* canker on European filbert trees measured as percent of the total number of main scaffold limbs attacked (•), or percent of the total surface area of limbs attacked (o).

may be killed within 5–15 yr after cankers first appear. Younger, more vigorous trees succumb to the disease more quickly and trees often are commercially worthless 4–7 yr after becoming infected.

Of added importance is the occurrence of the filbert bud mite *Phytocoptella avellanae* Nal (4,9,10). The more heavily infested a tree is with bud mites, the more prevalent are galled buds which act as the primary infection court. The presence of numerous favorable infection courts (mite-galled-buds) greatly increases the incidence of the disease both within a single tree and within an orchard as a whole.

The "r-values" of r = 1.085 and r = 1.236 unit per year, for increased incidence of disease within a single tree and disease increase within an orchard, respectively, are both near unity. In effect, the disease buildup within individual trees and whole orchards is a doubling of disease each year.

The original focus of the eastern filbert blight epidemic in the Pacific Northwest probably was initiated by importation of infected *Corylus avellana* nursery stock or wild *Corylus americana* seedlings from the eastern United States. The original focus of disease is thought to lie in the northeast quadrant of the affected area. This has been implicated by the higher disease severity ratings of these northern orchards. Because the severity rating scale is logarithmic in design and reflects the amount of tissue colonized by the organism, we believe that it relates to the elapsed time that *A. anomala* has been present in the orchard. It would require considerable time for the disease to build up sufficiently to be classed in the next highest rating. Hence, high disease ratings of 7–10 represent substantial tissue colonization and thus a considerable time factor.

The disease has spread mainly south and west from the original focus. Secondary foci usually include one or two orchards also with relatively high disease severity. The disease severity ratings of these orchards are, however, less than those in the original foci. We hypothesize that long distance dispersal of inoculum from the original focus occurred approximately 5–10 yr ago and that subsequent spread has been more localized. The environmental conditions necessary for long distance dispersal of inoculum are unknown. Dispersal over shorter distances appears to be more frequent and the environmental parameters necessary for short distance spread have been recently met as evidence by the 13 newly diseased orchards discovered during the 1979 survey. It follows that

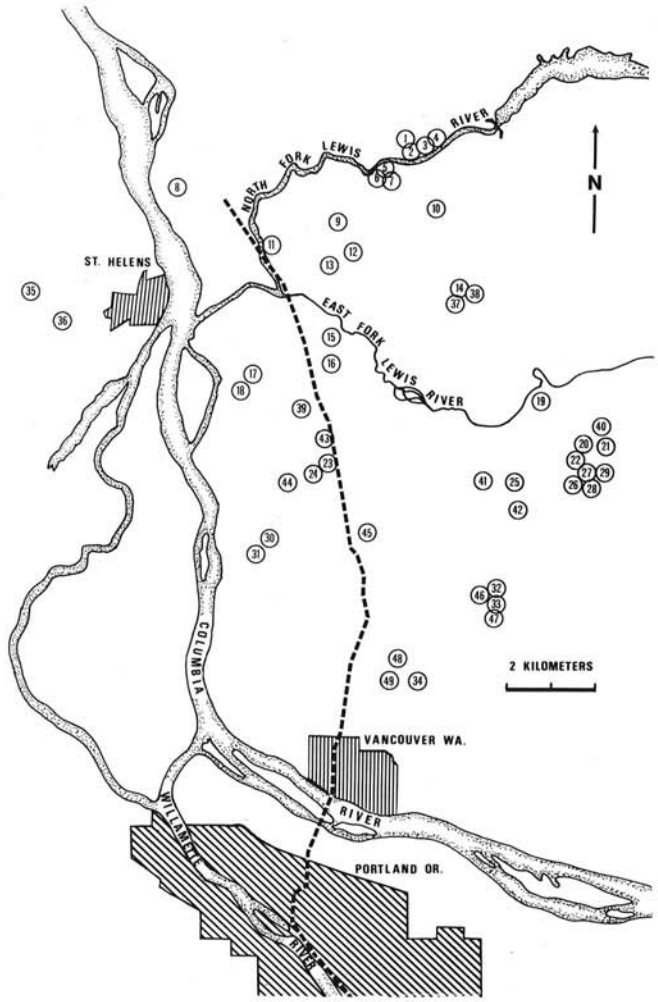

Fig. 7. Map of the lower Columbia River and surrounding vicinity of Clark County, WA and Cowlitz County, OR. Circled numbers show location of diseased filbert orchards.

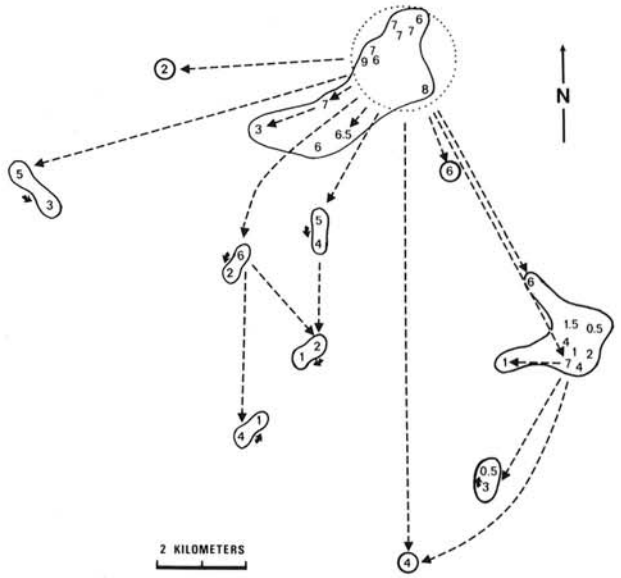

Fig. 8. Proposed pattern of spread of canker caused in filbert trees by *Anisogramma anomala* from the original focus of disease (dotted circle) to secondary and tertiary foci (areas surrounded by solid lines) detected in the 1977 disease survey. Numbers correspond to disease ratings for individual orchards: 1 = 10% of trees infected, 2 = 20%, etc. Dashed lines represent proposed avenues of spread of inoculum for longer distances (> 1.24 km). Small solid curved arrows represent proposed avenues of spread over shorter distances (< 1.24 km).

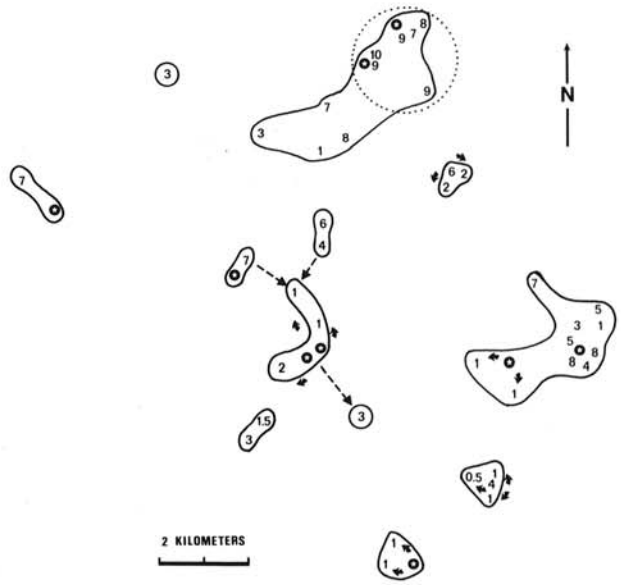

Fig. 9. Extent of disease and severity of infection found during the 1979 survey of canker caused by *Anisogramma anomala* in filbert orchards. Stars represent diseased orchards that have been removed. Compare increase in areas of secondary epidemics and corresponding increase in severity ratings of individual orchards over those of 1977 (Fig. 8).

local secondary foci are the more probable source of inoculum to adjacent orchards than even the more heavily diseased, inoculum-dense plantings a few miles to the north. If the present pattern of spread repeats itself, the southern-most secondary foci will pose the greatest threat to nearby disease-free orchards and to those further south across the Columbia River in the main filbert growing areas of Oregon. Therefore, the best approach to protect the areas, is the removal of these southernmost infections.

No fungicide or other chemical control measure for Anisogramma canker has yet been discovered (4,5). Due to the 10- to 14-mo latent period for symptom expression, sanitation methods such as severe pruning or diseased tree removal have been of little value in controlling the disease. However, control of mite populations with a miticide to reduce the prevalence of infection courts, followed by severe pruning and tree removal to decrease the amount of inoculum, should delay the spread of the disease in orchards presently under attack.

LITERATURE CITED

1. BAGGA, D. K., and E. B. SMALLEY. 1974. The development of Hypoxylon canker of *Populus tremuloides*: Role of interacting environmental factors. Phytopathology 64:658-662.
2. CAMERON, H. R. 1976. Eastern filbert blight established in the Pacific Northwest. Plant Dis. Rep. 60:737-740.
3. CAMERON, H. R., and T. R. GOTTWALD. 1977. Eastern filbert blight progress report. Proc. Nut Growers Soc. Oregon, Washington and British Columbia 62:41-46.
4. CAMERON, H. R., and T. R. GOTTWALD. 1978. Progress report on Eastern filbert blight. Proc. Nut Growers Soc. Oregon, Washington and British Columbia 63:522-523.
5. DAVIDSON, A. D., and R. M. DAVIDSON, Jr. 1973. Apioporthe and Monochaetia cankers reported in western Washington. Plant Dis. Rep. 57:522-523.
6. DUBIN, H. J., and H. ENGLISH. 1975. Epidemiology of European apple canker in California. Phytopathology 65:542-550.
7. GOTTWALD, T. R., and H. R. CAMERON. 1979. Morphology and life history of *Anisogramma anomala* Mycologia 71:1107-1126.
8. GOTTWALD, T. R., and H. R. CAMERON. 1980. Infection site, infection period, and latent period of canker caused by *Anisogramma anomala* in European filbert. Phytopathology 70:1083-1087.
9. KRANTZ, G. 1980. The role of *Phytocoptella avellanae* (Nal.) and *Cecidiophyopsis vermiformis* (Nal.) (Eriophyidae) in big bud of filbert. Proc. Fourth Int. Congr. Acarology, Sallfelda, Austria (In press).
10. LAREW, H. G. 1977. "Big Bud" Disease of Filbert: A Descriptive Cytological Study of the Affected Plant Tissue. M.S. thesis. Dept. Botany and Plant Pathology, Oregon State University, Corvallis, Oregon.
11. RANKIN, W. H. 1914. Field studies on the Endothia canker of chestnut in New York. Phytopathology 4:233-259.
12. VANDERPLANK, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York and London. 349 pp.