# Distribution and Disease Progress of Phytophthora Root Rot of Fraser Fir Seedlings

C. M. Kenerley and R. I. Bruck

Former graduate research assistant and associate professor, Department of Plant Pathology, North Carolina State University, Raleigh 27650. Present address of senior author: Department of Plant Pathology and Microbiology, Texas Agricultural Experiment Station, College Station 77843.

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## ABSTRACT

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The movement of *Phytophthora cinnamomi* from infested into noninfested portions of five disease focus plots was monitored by quantifying inoculum density of soil samples (nine sampling periods) and recording Fraser fir seedling mortality (18 assessment dates). Comparison of overlapping inoculum density contour maps with regard to changes in inoculum density, new area infested, and shift in area of highest inoculum density revealed four patterns of propagule distribution. Inoculum density maxima were recorded 26 August and 8 September. Greatest increases in total mortality of seedlings also occurred during this period of the growing season. Final cumulative mortality was not correlated with initial inoculum density or seedling density. Seedling mortality was recorded in areas of each plot previously noninfested, but area of propagule detection was more extensive than that of seedling mortality. Plots of calculated relative rates of disease increase vs. the cumulative seedling mortality illustrated fluctuations in disease progression, indicating a lack of fit with widely acceptable models.

Several studies have quantified inoculum densities and dispersal of propagules of aerial pathogens into noninfested areas (5,9,13,16). In contrast, studies that quantify the movement of a soilborne fungal pathogen from a naturally infested area into a noninfested area have been few and the inoculum densities of the pathogens were not determined in these investigations (2,6,20,23). Field experiments designed to quantify disease progression or relationships between inoculum density and severity of disease induced by soilborne fungi have generally been established in soils where the pathogen was known to exist based on previous disease incidence or by uniformly infesting the soil with fungal propagules (1,3,12,15,19). A study designed to quantitate inoculum density of a soilborne pathogen and host mortality of a disease that is continuous over several years without the agronomic interruptions of an annual crop and where the initial presence of the pathogen can clearly be differentiated seems warranted. This information will be useful in further describing the dynamics of epidemics of disease caused by soilborne pathogens.

Movement of the soilborne pathogen *Phytophthora cinnamomi* Rands from infested to noninfested areas of a Fraser fir (*Abies fraseri* (Pursh.) Poir.) nursery bed can be readily studied. Phytophthora root rot generally develops from distinct foci within a nursery bed. Because seedlings remain in the nursery beds for 3 yr, there is an opportunity in time and space for disease development. The objective of this research was to quantify the spatial and temporal development of Phytophthora root rot of Fraser fir seedlings by recording and mapping seedling mortality and determining the inoculum density of *P. cinnamomi* at numerous intervals during a growing season in a production nursery. A portion of these results has been presented previously (10).

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## MATERIALS AND METHODS

**Plot establishment.** Five plots in nursery beds (each bed approximately 1.0 × 75 m) containing Fraser fir seedlings in their third growing season were selected 19 April 1981 at the Linville River Nursery, North Carolina Forest Service, Crossnore. The plots were located in operational nursery beds that receive three applications of ammonium nitrate during the growing season (30 kg/ha during the first weeks of April, May, and June). There were no fungicide or irrigation treatments applied to any of the beds during this study. Seedling densities were not uniform because of establishment of beds by uneven broadcast seeding and the erratic germination of Fraser fir seed. Each plot contained an individual disease focus that had developed during the two previous growing seasons. Within each disease focus there was an area (termed the initial mortality zone) devoid of seedlings. Seedlings originally in these areas died the previous year as a result of infection by *P. cinnamomi* (C. M. Kenerley, *unpublished*) and had been removed (first week April 1981) by nursery personnel before plot establishment. The boundary of the initial mortality zone in each plot was mapped at 5° intervals around a permanent stake centered in each plot. Seedlings adjacent to the initial mortality zones appeared healthy and displayed no symptoms of Phytophthora root rot.

**Determination of extent of disease foci.** Soil and seedling samples were collected on 26 April from each plot to determine the northern and southern (long axis of the nursery bed) extent of the pathogen. A 15 × 10-cm grid pattern was used for collection of the samples beginning 15 cm inside the northernmost and southernmost boundaries of the initial mortality zone and extending 45 cm beyond the boundary. Two seedlings and one soil core (1.9 cm diameter to depth of 15 cm) were removed from each grid intersection (36 samples in each direction per plot). Roots of seedlings and soil samples were assayed by plating onto a Phytophthora selective agar medium (17). The soil assay used has been previously described and has a detection level of 0.01 propagules per gram of dry soil (11).

Once the initial assays were complete, a permanent baseline was

established in the northern and southern portions of each plot perpendicular to the long axis of the nursery bed (Fig. 1). The portion of the plot in which the pathogen was detected (in either soil or seedling samples as of 26 April) was designated the primary infested zone and where assays failed to detect the pathogen, the secondary infested zone (Fig. 1). The baseline served as a reference for future sampling.

**Monitoring spatial development of the pathogen.** The spread of the pathogen from the primary infested zone into the secondary zone was monitored from 10 May to 10 November by quantifying the inoculum density in soil samples (nine sampling dates) and by recording seedling mortality (18 recording dates).

The grid pattern established for determining the extent of the disease focus in each plot was expanded in the long axis of the nursery bed (northern and southern direction) to serve as a guide for the collection of soil samples. The grid pattern for each direction within a plot consisted of seven transect lines (10 cm apart) perpendicular to each baseline and extending 120 cm in the long axis of the bed and eight transect lines (15 cm apart) perpendicular to the short axis of the bed (Fig. 1). Soil samples were collected at each intersection for one center line and the two immediately adjacent transect lines beginning at the baseline (27 samples for each direction, 54 for each plot) for each of nine collection dates. At each intersection three soil cores (1.3 cm diameter × 1.5 cm depth) were removed and bulked for assaying for *P. cinnamomi* propagules. Each of the seven transect lines served once as the center line for collection of soil samples among the three sequential transect lines. Two of the seven transect lines represented a center line as repeats on the last two sampling dates. This sampling pattern was chosen to minimize soil disturbance within the plot, reduce the number of individual samples per plot, allow for samples to be collected over the width of the nursery bed, and still provide repeated samples for some positions.

Inoculum density contour maps were developed for each direction within a plot using computer graphics (4) for each set of three sequential transect lines based on the inoculum density values at every intersect. One to 10 contour intervals can be established using the SYMAP computer mapping program (4). Five intervals were selected for this study as resolution was diminished with less than five contour intervals. When more than five contour intervals were used, the mapping was too detailed for superimposing on mortality data. SYMAP can be programmed for unequal contour intervals. As we had no previous biological data to determine the range of values for each interval, we arbitrarily established that the range of the intervals be equal. The average inoculum density for each contour level within the plot was correlated with that contour's corresponding mortality. These simple linear correlations were performed for each plot (12 observations per plot per date) and across all plots (60 observations per date) for each of the nine sampling dates. On the last sampling date (10 November), soil cores were assayed from intersections in which no propagules had previously been detected.

**Monitoring progress of mortality.** Seedling density within the area of each grid was recorded (26 April) before any seedling mortality. Seedling mortality (flagging of terminal shoot and discoloration of needles) was recorded for each plot on 18 dates between 3 May and 11 November. The distance and azimuth from the permanent center stake to each dead seedling were determined. Computer graphics (4) were used to construct maps (with regard to distance and angle from permanent center stake) of seedling mortality over time. Disease progress data from each plot were examined by plotting relative rates of disease increase ($[1/M]\, dM/dt$) calculated using numerical differentiation (7, equation 2) against the cumulative mortality ($M$, cumulative number of dead trees). In addition, simple linear correlation analysis was performed to determine if there was a relationship between seedling mortality and seedling density within the area of each grid in areas where mortality occurred. This analysis was computed by plot and across all plots.

**Site characteristics.** Residual soil from samples taken for propagule determination (nine dates) was bulked by plot direction (northern and southern) for each plot and analyzed by date for K, Ca, Mg, Zn, Cu, pH, percentage of organic matter, CEC, and percentage of base saturation by the North Carolina Department of Agriculture, Agronomic Division. Soil moisture was determined gravimetrically at nine sampling dates throughout the growing season and compared to a soil drying curve prepared for each plot. Soil texture was also determined for each plot. Soil temperature at depths of 7.5 and 15 cm and rainfall were recorded continuously with Fenwal thermistors and a tipping bucket rain gauge, respectively, attached to a Campbell CR21 micrologger (Campbell Scientific Inc., Logan, UT) from a central location among the plots. Changes in elevation within each plot were determined along eight transects (between 30 and 120 cm in length) at 45° intervals around the permanent center stake.

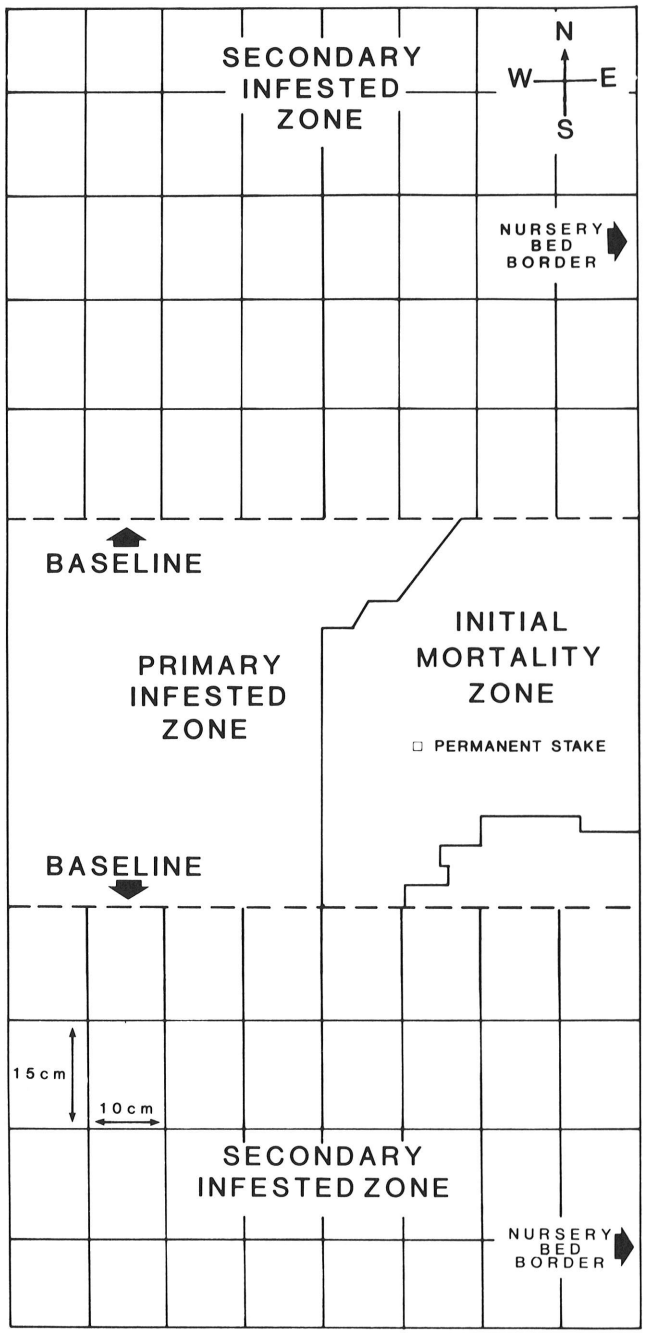


**Fig. 1.** A representative drawing of one of five plots in a Fraser fir nursery at the Linville River Nursery, Crossnore, NC, illustrating the sections of each plot (initial mortality zone, area devoid of seedlings; primary infested zone, area containing infested soil and asymptomatic seedlings; and secondary infested zone, area uninfested at the beginning of sampling period) and the sampling design (transect lines forming grids) for collection of soil and seedling samples to assay for *Phytophthora cinnamomi*.

## RESULTS

**Monitoring distribution of the pathogen.** There were no differences in initial inoculum densities of the primary infested zones among the plots on 26 April (ranging from 0.05 to 0.09 propagules per gram of dry soil). The inoculum density at the baseline and within the secondary infested zone for each plot was 0.00 propagules per gram of dry soil. Initial detection of movement of the pathogen from the primary infested zone into the secondary infested zone varied from 30 to 92 days after the plots were established. The final distance of spread of *P. cinnamomi* (as propagules per gram in the soil) into the secondary infested zone ranged from 30 to 90 cm and from 45 to 105 cm in the northern and southern direction, respectively, among the five plots (Table 1). The total area of the secondary infested zone in which propagules of *P. cinnamomi* were detected ranged from 0.52 to 1.36 $m^2$ (Table 1).

Average inoculum density in the secondary infested zone did not exceed 0.50 propagules per gram of dry soil until 8 July. The greatest average inoculum density values were recorded 26 August and 8 September in the northern and southern portions of the plots, respectively (24.94 and 24.41 propagules per gram of dry soil, respectively). The greatest inoculum density (105.4 propagules per gram of dry soil) for any sampling position was recorded on 26 August. Inoculum density maxima were recorded during the period of greatest increase in total mortality (between 11 and 26 August) and the greatest total area occupied by dead seedlings (26 August). The maximum inoculum density occurred after an extensive period of time (more than 70 days) in which the average daily soil temperature equaled or exceeded 20 C (Fig. 2). Soil moisture was not correlated with the maximum values of inoculum density.

The distribution of propagules of *P. cinnamomi* over time was investigated by comparing three characteristics of the overlapping inoculum density contour maps: changes in magnitude of the inoculum density (value of the contour interval) among five inoculum density intervals, detection of propagules at increasing distances from the baseline into the secondary infested zone, and a directional shift of the area of highest inoculum density away from the baseline. Four distribution patterns were observed in the five plots. In pattern 1, there was an increase in the inoculum density within each contour interval with time, but the area of distribution was relatively the same (Fig. 3A). There was little or no increase in the total area in which propagules of *P. cinnamomi* were detected compared with the same contour map at an earlier sampling date (Fig. 3A). In pattern 2, there was an increase in inoculum density in each contour interval with time and an increase in the total area where the pathogen was detected (Fig. 3B). This increase in total area within the secondary infested zone in which the pathogen was detected generally reflected an increase in area of two or more contour intervals (Fig. 3B). In pattern 3, there was an increase in inoculum density in each contour interval, an increase in total area where propagules were detected, and the movement of the interval of highest inoculum density away from the baseline into the secondary infested zone (Fig. 3C). In pattern 4, there was a decrease in the inoculum density within each contour interval with time, but the area of distribution was relatively the same (Fig. 3D). The percentage in which the four patterns were found among the five plots over the nine sampling dates was 23, 25, 29, and 15% for patterns 1, 2, 3, and 4, respectively. No pattern was established for 8% of the overlapping contour maps. Each pattern was found in every plot and a similar sequence of pattern appearance was noted for all the plots. Generally, pattern 1 was most common during the early sampling dates (16 June–7 July), pattern 2 more common in midseason (11 August), and patterns 3 and 4 most prevalent during the last period (1 October–10 November).

The final mortality within or over all the plots were not correlated with the initial inoculum density determined on 26 April (ranging from 0.05 to 0.09 propagules per gram of dry soil). Inoculum density within the contour intervals was not consistently correlated with final mortality within the contour intervals when analyzed across all plots or separately for each plot. Significant correlations ($P > 0.05$) were found for some plots at some of the sampling periods, but no consistent trend was apparent for plots when considered individually or collectively.

**Monitoring progress of seedling mortality.** Fourteen percent of the seedlings assayed from the primary infested zone were known to be infected, but asymptomatic (determined by plating root samples collected on 26 April). The first mortality was seen on 16 June among seedlings in the primary infested zone. The pattern of seedling mortality was similar for all plots: mortality among seedlings in plots appeared in the primary infested zone first; mortality of seedlings in the secondary infested zone occurred 15–41 days later; and, as the summer progressed, mortality developed further into the secondary infested zone.

Increments of seedling mortality (number of new dead seedlings since the previous assessment period) were compared over all plots at each assessment period between the primary and secondary infested zones. The peak in increments of seedling mortality occurred in the primary infested zone first (Fig. 4). The peaks in incremental mortality in either the primary or secondary infested zone occurred after 10 August.

Mortality of 3-yr-old seedlings in the five plots was recorded at distances of 15–60 cm in the northern direction and 30–75 cm in the southern direction from the baseline into the secondary infested zone (Table 1). The total area in the secondary infested zone occupied by dead seedlings ranged from 0.31 to 0.91 $m^2$ for the five plots (Table 1). The distance from the baseline into the secondary infested zone where mortality was observed or the total area in the secondary infested zone occupied by dead seedlings was equal or less than the distance or area where propagules were detected within each plot (Table 1). The detection of propagules of *P. cinnamomi* always preceded the appearance of mortality in the same area. When the total area where mortality and propagules occurred was compared at the end of the growing season, the area with propagules always exceeded the area of mortality. Samples of asymptomatic seedlings (50 per plot) were removed from the

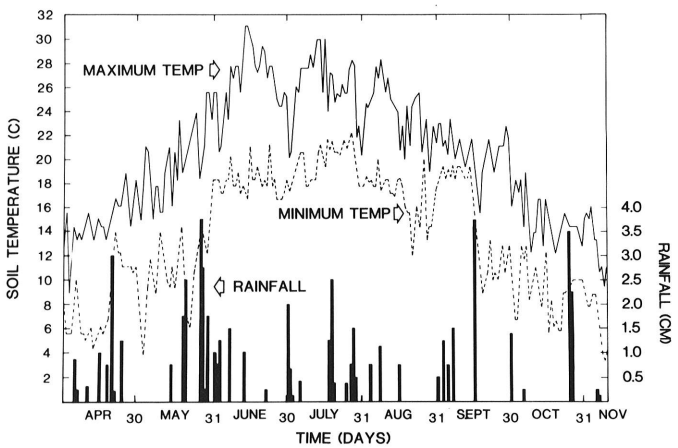


**Fig. 2.** The minimum and maximum soil temperature (C) at a depth of 7.5 cm and daily rainfall (cm) at the Linville River Nursery, Crossnore, NC, between 7 April and 11 November 1981.

TABLE 1. The maximum distance in two directions and total area of spread of propagules of *Phytophthora cinnamomi* and mortality of 3-yr-old Fraser fir seedlings from a disease focus into initially noninfested portions (secondary infested zone) of five plots in nursery beds at the Linville River Nursery, Crossnore, NC

| | Propagules of *P. cinnamomi* | | | Mortality of Fraser fir seedlings | | |
|---|---|---|---|---|---|---|
| | Total | Distance (cm) | | Total | Distance (cm) | |
| Plot | area ($m^2$) | North | South | area ($m^2$) | North | South |
| 1 | 0.52 | 30 | 45 | 0.31 | 15 | 30 |
| 2 | 1.12 | 60 | 75 | 0.91 | 45 | 60 |
| 3 | 0.69 | 45 | 75 | 0.37 | 30 | 45 |
| 4 | 1.22 | 45 | 90 | 0.80 | 45 | 75 |
| 5 | 1.36 | 90 | 105 | 0.84 | 60 | 75 |

intersects where propagules but no mortality had been detected at the end of the season. Thirty-two percent of the asymptomatic seedlings sampled at this time (17 November) were infected with *P. cinnamomi*.

Disease progress curves for the distinct portions (primary and secondary infested zones) and the combined totals of each plot are presented in Figure 5. The total area, initial number of seedlings, and final percent mortality for the primary infested zone of each plot are given in Table 2. There was no correlation among the final mortality in this portion of the plot and the initial propagule density or seedling density. Relative rates plotted against cumulative mortality demonstrated fluctuations in the progression of disease (Fig. 6). This response was evident for either portion of the plots but appeared somewhat more damped as cumulative mortality increased in the secondary infested zone (Fig. 6B). The increments of mortality plotted against the assessment dates (Fig. 4) also illustrate the fluctuations of disease progression particularly with regards to the early assessment dates.

**Site characteristics.** Changes in microelevation were recorded within each plot but no consistent directional pattern (e.g., decreasing elevation always associated with southern direction) was detected. Microelevation maps developed for each plot by computer graphics (4) were overlaid on the mortality maps to discern relationships between elevation changes and mortality: no such relationships (e.g., increased mortality with decreasing elevation) were observed among the plots. There was no difference among the plots in soil fertility or organic matter. The soil texture was a loamy sand (71% sand, 23% silt, and 6% clay) in three plots (plots 1, 2, and 5) and a sandy loam (68% sand, 25% silt, and 7% clay) in the other two plots (plots 3 and 4). Neither seedling mortality nor seedling density was correlated with soil texture. A comparison of the soil matric potential values at the nine sampling dates showed no significant plot differences.

## DISCUSSION

Any attempt to analyze epidemics of perennial crops caused by soilborne pathogens requires consideration of asymptomatic plants that were infected the previous season. The structural components (e.g., rate, infection point) of disease progression

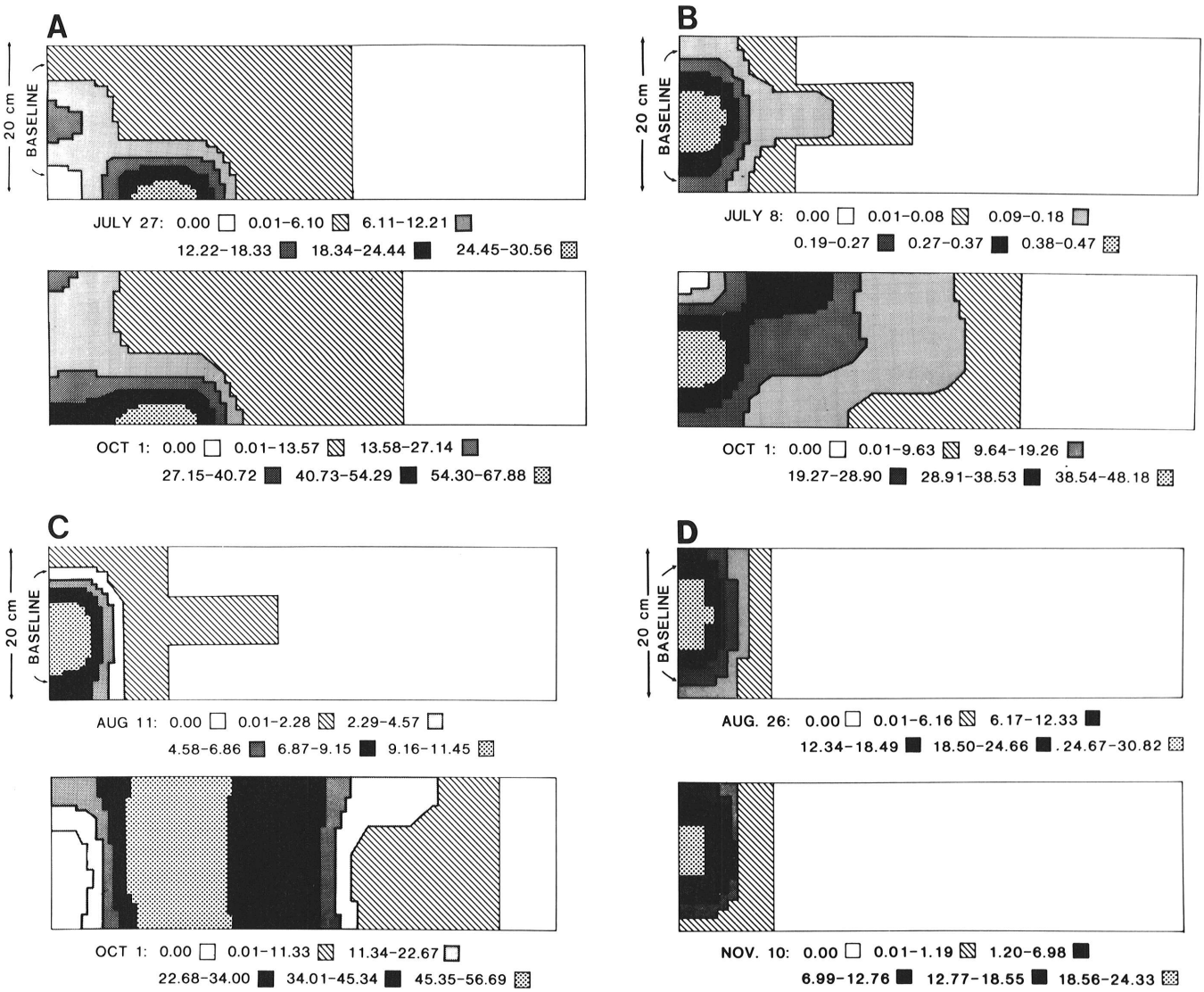


**Fig. 3.** Representative propagule contour maps illustrating the change in distribution and inoculum density of propagules of *Phytophthora cinnamomi* in an initially noninfested portion of nursery bed of 3-yr-old Fraser fir seedlings at the Linville River Nursery, Crossnore, NC. Each contour map represents data from three sequential transect lines with each set of two maps corresponding to the same three lines sampled at different times in the growing season (see date associated with each set of maps). The initial infested portion of the plot (assayed 26 April) is not shown but bordered the left margin of the figure. Shaded areas within each contour map represent different inoculum densities (propagules gram dry soil) of *P. cinnamomi*. **A,** Overlapping maps illustrate an increase in inoculum density within each contour interval with each interval remaining similar in size over time. **B,** Overlapping maps illustrate an increase in inoculum density within each contour interval as well as an increase in the area where propagules were detected over time. **C,** An increase in inoculum density, area infested, and a shift in the location of the contour interval of highest inoculum density over time is illustrated by this set of maps. **D,** A decrease in inoculum density, but contour intervals remaining similar in size over time.

within areas with asymptomatic but infected plants may be different from disease progression that develops in adjacent areas containing initially noninfected plants. This distinction may become more apparent as experiments are conducted to repeat or explain the measured or calculated structural components from data that combine the two areas. Also, if comparative studies of epidemics incited by soilborne fungi are made in which one of the epidemics involves mortality of initially infected but asymptomatic plants that die earlier or at a different rate than subsequently infected plants, the results of these comparisons will be misleading.

This situation is illustrated in the disease progress curves (Fig. 5) where differences in final mortality among plots within the primary or secondary infested zones is found. There was also a change in the ranking of the plots with regard to final mortality when the two areas were treated separately. This difference among the plots in final mortality in the primary infested zone could be explained by the fact that this area was initially limited and contained a specific number of seedlings in each plot. The number of seedlings within this area varied from plot to plot and may account for the low final mortality in plots 3 and 5 (Table 2). However, this does not explain the large difference in cumulative and percent mortality between plot 4 and the other plots. The initial propagule density and number of infected but asymptomatic seedlings determined on 26 April were equal in all the plots. This suggests that unless the initial sampling design used in this study failed to detect small clumps of asymptomatic seedlings or clusters of propagules in plot 4, other factors (e.g., root growth and distribution, pathogen or host variation, propagule efficiency) than initial propagule density may be more important in understanding disease development with this pathosystem.

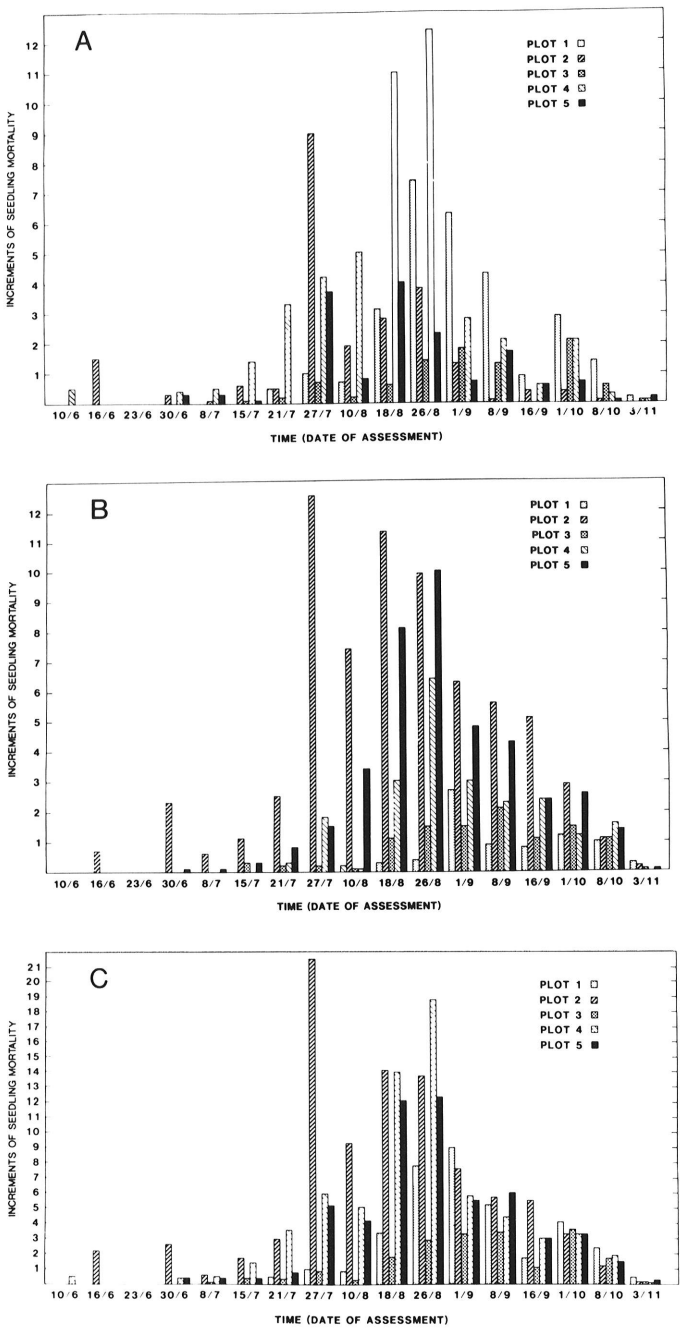


**Fig. 4.** Increments of seedling mortality (number of new dead seedlings since previous assessment period) for 3-yr-old Fraser fir seedlings recorded in five plots at the Linville River Nursery, NC. **A, B, and C** correspond to cumulative mortality in the primary infested zone (area of plots initially containing asymptomatic seedlings), secondary infested zone (initially noninfested portion of each plot), and the combined totals for these areas, respectively.

Final mortality within the secondary infested zones of the plots did not appear to be related to mortality, propagule density, or area of a plot's primary infested zone. Plots 1 and 4, which had the largest areas and greatest amount of mortality in the primary infested zone (Table 2), resulting in larger borders of dead seedlings adjacent to noninfested areas, had relatively low mortality in the secondary infested zone (Fig. 5B). These two plots (1 and 4) had the lowest percentage (23 and 31%, respectively) of their final mortality attributable to mortality in the noninfested portions of the plots. In contrast, plots 2 and 5 had the greatest percentage (77 and 73%, respectively) of their final mortality within the secondary infested zone. These differences in mortality would have been obscured if the areas were combined (Fig. 5C). These data suggest that the pathogen's influence on symptom expression (through infection and root colonization) as influenced by the environment may be very different within the two zones of mortality studied here.

The disease progress curves were further analyzed by plotting their relative rates of disease increase vs. the amount of disease (Fig. 6). This type of analysis was used because it is not dependent on an asymptote of 1 (which was not obtained in either zone and only rarely found in any study) and provides a means of graphically illustrating trends in the data for subsequent model selection. The fluctuations in the plots of the relative rates vs. cumulative mortality for either primary or secondary infested zones clearly demonstrate that this response cannot fit into the Richards family of curves (7,8). The data, in general, suggest that within the secondary infested zones, events in the initial stages of disease development are variable and not suitable for comparison using widely acceptable models (7,14). The relative rate plots for the primary infested zones showed further fluctuations instead of damping as cumulative mortality increased. Whether these responses are an artifact of the analysis, result of the numerous assessment dates, or a biological phenomenon that has not been realized before was not resolved in this study.

The quantification of *P. cinnamomi* propagules during the growing season demonstrated at least four spatial and temporal patterns of distribution of the inoculum that were found in all the plots (Fig. 3). In three of the patterns, there was a continuous increase in the production of secondary inoculum with peaks

TABLE 2. The area and density and mortality of 3-yr-old Fraser fir seedlings in the primary infested zone (area initially containing asymptomatic seedlings) of five plots in nursery beds at the Linville River Nursery, Crossnore, NC

| Plot | Area of plot ($m^2$) | Avg. seedling density (avg. no./ 0.015 $m^2$) | Seedlings in area (no.) | Final mortality | Mortality (%) |
|---|---|---|---|---|---|
| 1 | 0.65 | 19.3 | 836 | 236 | 28 |
| 2 | 0.36 | 23.3 | 560 | 171 | 31 |
| 3 | 0.30 | 18.2 | 364 | 84 | 23 |
| 4 | 0.82 | 11.0 | 601 | 397 | 66 |
| 5 | 0.43 | 12.9 | 370 | 125 | 34 |

occurring in late August or early September. Other studies (12,21,22) have reported increases in the number of chlamydospores with increasing soil temperatures and that high levels do persist in the soil for several months. The dates corresponded to the largest increases in mortality (Figs. 4 and 5), but mortality could not be used as an indication of what pattern may be found when assaying the soil samples. The use of propagule contour maps did demonstrate within the same sampling unit an increase in propagule density and an increase in the area where propagules were detected, both of which can be explained in terms of further root colonization and new infections.

More interesting was the spatial shift in the contour level with the highest propagule density over a period of 4–8 wk (Fig. 3C). One explanation may be that propagules are formed in only certain aged or sized roots or in roots infected under certain environmental conditions or formed only after an extended period of colonization by the pathogen. As these roots deteriorate, these propagules could be released into the soil in large numbers, resulting in a shift in contour levels. Root growth dynamics and the appearance of this third pattern only in late season sampling suggest this is at least biologically feasible. The authors are fully aware that these interpretations may be biased by the arbitrary decisions for programming SYMAP (e.g., number and values of contour intervals) and the sampling design, but feel a mapping program such as SYMAP can be valuable in illustrating large sets of quantitative data for soilborne pathogens.

Another interesting feature of this study was that *P. cinnamomi* propagules (primarily chlamydospores) (C. M. Kenerley, *unpublished*) were detected in advance of symptom development. The pathogen was isolated from 32% of asymptomatic seedlings sampled at the end of the growing season (17 November) from areas where propagules were detected but no mortality had occurred. Examination of these plots, the following spring before seedlings were harvested (April 1982), showed no mortality among seedlings in areas where propagules had been detected. Sampling seedlings in adjacent nursery beds where no soil sample had been removed yielded other asymptomatic seedlings (C. M. Kenerley and R. I. Bruck, *unpublished*). This occurrence necessitates the need to collect soil samples beyond the areas of mortality to determine the extent of spread of the pathogen to optimize disease

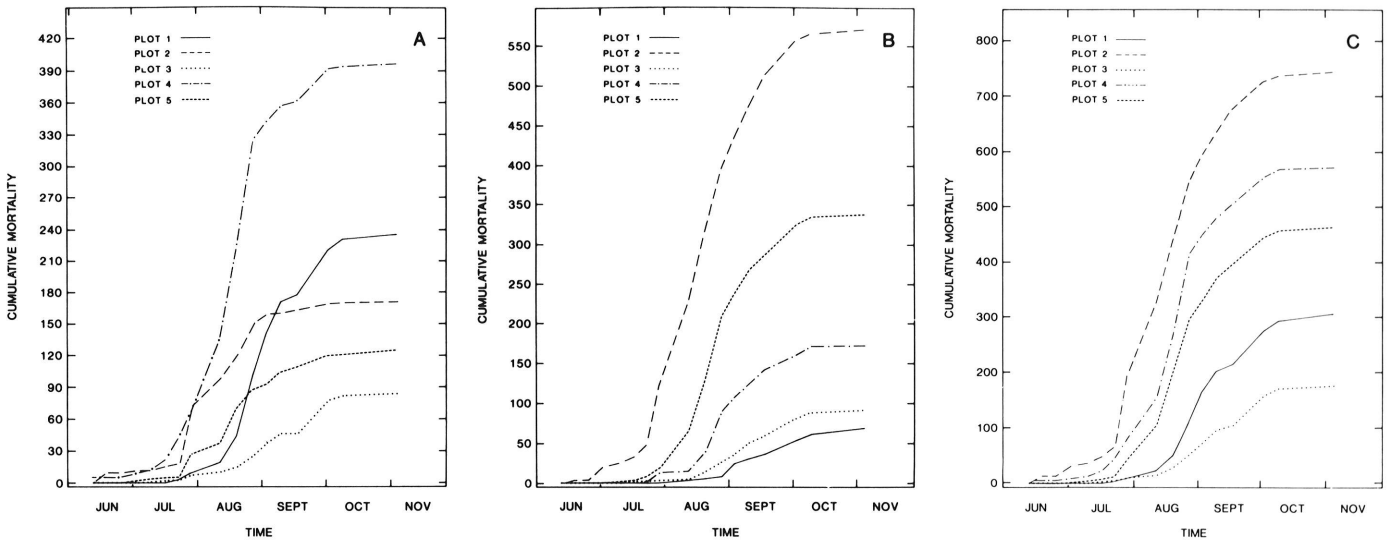


**Fig. 5.** Cumulative mortality of 3-yr-old Fraser fir seedlings resulting from infection by *Phytophthora cinnamomi* in five plots at the Linville River Nursery, Crossnore, NC. **A, B, and C** correspond to cumulative mortality in the primary infested zone (area of plots initially containing asymptomatic seedlings), secondary infested zone (initially noninfested portion of each plot), and the combined totals for these areas, respectively.

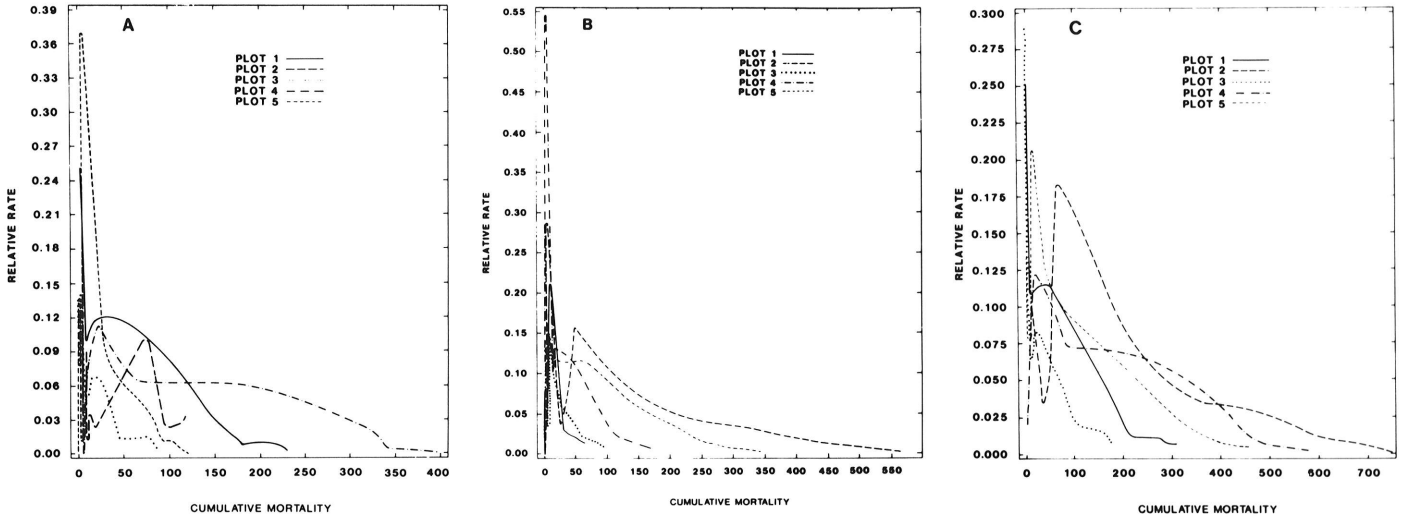


**Fig. 6.** Relative rates of disease plotted against cumulative mortality of 3-yr-old Fraser fir seedlings in five plots at the Linville River Nursery, Crossnore, NC. **A, B, and C** correspond to cumulative mortality in the primary infested zone (area of plots initially containing asymptomatic seedlings), secondary infested zone (initially noninfested portion of each plot), and the combined totals for these areas, respectively.

control procedures. This may also account in part for the lack of correlation between inoculum density and seedling mortality. Some contour levels had high inoculum density values, but no seedling mortality, which would tend to reduce any correlation between inoculum density and mortality. Fraser fir is, however, very susceptible to infection by *P. cinnamomi* and any inoculum density detected by the assay in this study should result in infection. Shew and Benson (18) demonstrated that inoculum densities less than 0.01 propagules per gram of dry soil can result in infection of Fraser fir seedlings. Once seedlings are infected, mortality may depend on the stresses placed on the seedling by the environment. This is supported in part by the large increases in mortality during the months of least rainfall (Figs. 2 and 4) and driest soil moisture recordings.

Research designed to examine the dynamics of infection and secondary inoculum production with respect to root growth and deterioration is necessary to determine if the spatial patterns of propagule distribution illustrated in this study (Fig. 3) are characteristic of soilborne pathogens. The events of infection (e.g., site of infection, number of infections) and root production (e.g., rate and distribution) need to be examined in more detail to substantiate and fully understand the biological significance of the fluctuations in disease progression illustrated by plotting relative rates of disease increase vs. the cumulative seedling mortality. If these fluctuations are biologically based, any modelling attempts for the Fraser fir-*P. cinnamomi* pathosystem and perhaps other soilborne pathogen systems may require that the components of the infection cycle be analyzed separately rather than by growth curve analysis.

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