Ecology and Epidemiology

Effects of Host Density and Number of Disease Foci on Epidemics of Southern Blight of Processing Carrot

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ABSTRACT

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Epidemics of southern blight of processing carrot, caused by *Sclerotium rolfsii*, were influenced positively by high host density and by high initial number of disease foci per row. Disease severity, estimated as area under the disease progress curve (AUDPC), was highest in rows with high host density (52 plants per meter) and the highest number of initial foci (10 foci per row). Host tissue available for infection apparently was limiting in plots with eight to 10 foci per row, as disease progress curves of these plots converged at the end of the season at 32% due to focus extinction. AUDPC for disease severity and the rate of disease increase, derived from a linear model, were directly related to the initial number of disease foci per row. Rate of focus expansion, measured as centimeters per focus per day, was influenced by fluctuations in the number of hours of relative humidity greater than 95% in the plant canopy, near the crown of the plants.

Variations in canopy density, obtained through combinations of thinning the plants and removal of foliage from a portion of the carrot plants, affected disease severity, but the relationship between change in the number of foci per row and change in disease severity was not constant over the different canopy densities. In rows where carrot plants were thinned and foliage removed, disease severity with 10 initial foci per row was almost equal to that at 0 and 4 foci per row. Removing the foliage of the plants enhanced interplant mycelial growth in rows that were not thinned. Disease severity over time was significantly influenced by the number of foci over time, suggesting that quantitative evaluations of epidemics of southern blight of processing carrot may be possible with a rating of disease incidence (number of foci) rather than disease severity.

Southern blight of processing carrot (*Daucus carota* L.), caused by *Sclerotium rolfsii* Sacc., is the limiting factor in commercial production of processing carrot in eastern North Carolina. Many environmental factors affect growth of *S. rolfsii* through the soil and, consequently, the progress of epidemics of southern blight. The disease is most severe in warm, humid regions with light sandy soils (2,12); the optimum temperature for growth of the fungus is 20–27 C (2). High soil moisture appears to favor disease development (4,12). Severe epidemics develop during wet weather following a period of dry weather (2,12). In spite of these observations, no reported studies have quantitatively examined the effects of moisture on development of southern blight of carrot.

In addition to ambient weather conditions, canopy microclimate, i. e., relative humidity and soil moisture in the upper few centimeters of soil, influences epidemics of southern blight. Crop management practices that promoted the development of or maintenance of the foliar canopy were associated with the highest level of incidence of southern blight on peanut (3,24). When defoliating pests and peanut leaf spot pathogens were controlled, the severity of southern blight increased. Conversely, mechanical removal of leaves or defoliation due to infection by *Cercospora arachidicola* Hori reduced damage by *S. rolfsii* (3). The effects of plant spacing and canopy density on epidemiology of southern blight of processing carrot have not been assessed quantitatively, although high plant density and a thick canopy were associated with greater disease incidence in processing carrot in Georgia (14).

Inoculum of *S. rolfsii* is spatially aggregated in naturally infested field soil (17). Peanut plants infected by this pathogen also tend to be clustered within fields (25). Although *S. rolfsii* initially infects

plants in foci within the plant rows, considerable interplant spread by mycelial growth from plant to plant is possible (2,14). Such interplant mycelial growth is important in the epidemiology of southern blight (18) as it is for diseases caused by *Pythium* (5,6), *Sclerotium cepivorum* Berk. (8), and *Sclerotinia* (11). Interplant mycelial growth is facilitated by high host density (short interplant distances). Thus, the number of disease foci within a row and host density are important factors that may influence the rate of epidemic development and, ultimately, disease severity at the end of the season.

To better understand the factors influencing epidemics of southern blight of processing carrots, we monitored epidemics of the disease in the field over three growing seasons. The objectives of this study were to examine the relationships between factors related to host density and canopy density, e. g., air temperature, soil temperature, relative humidity, and incident water (rainfall and irrigation), and disease progress of epidemics of southern blight. Epidemics associated with canopy or host densities considered favorable and unfavorable for disease development were compared, and the effect of initial number of disease foci on final disease severity was evaluated. A preliminary report of these investigations has been published (26).

MATERIALS AND METHODS

Establishment of experimental plots. Fields on the Horticultural Crops Research Station, Clinton, NC, not previously cropped to carrot were selected for use during 1984 and 1985. Soil in the fields was Orangeburg loamy sand (loamy, siliceous, thermic Arenic Paleudult). Before planting, a 300-cm³ sample of soil was taken from the approximate location of each plot with a golf-green cup cutter and assayed for sclerotia of *S. rolfsii* using a wet-sieving

technique (17). Fields were deep-plowed to a depth of 15–20 cm (12) to reduce existing inoculum detected in the fields during sampling in 1984 and 1985.

Carrots (cultivar Danvers 126) were seeded in a 6-cm band on 1-m bedded rows at a rate of 48–60 seeds per linear meter of row on 19 March 1984 and 25 March 1985. Standard fertilization and chemical weed control practices were used. Overhead sprinkler irrigation was applied as needed to maintain good crop growth.

Design of experiments. Experimental plots were established on 7 June 1984 and 14 June 1985. Plots consisted of four rows × 7.6 m (30.4 linear m of row) and two rows × 7.6 m (15.2 linear m of row) in 1984 and 1985, respectively. Plant density in 1984 was about 26 plants per meter. In 1985, half the rows in the plots were thinned and had a plant density of about 26 plants per meter, while half were not thinned and had a plant density of about 52 plants per meter. Treatments consisted of 0, 2, 4, 6, 8, or 10 disease foci per row and were arranged in randomized complete blocks with five replicates. Each disease focus was established within a row by placing colonized oat seed inoculum (15) in a small cavity in the soil about 2 cm deep (total volume approximately 10 cm^3) adjacent to the crown of the carrot plants. A cap of soil was replaced over the cavity to prevent washing out of inoculum. Each placement of inoculum (subsequently a disease focus) was marked with a flag. Placement of inoculum was arranged within the rows to maximize potential distance of mycelial growth from each subsequent disease focus. For example, in rows having two foci, each focus was established such that mycelium growing bidirectionally from each point of inoculation could colonize half the plants sequentially in the row. Plots were irrigated with overhead sprinklers after inoculum placement.

Assessment of disease severity. Disease severity was evaluated at 3- or 4-day intervals by measuring the length of row of plants infected adjacent to each focus. Wilted plants were considered to be infected if mycelium was evident on the crown of the plant and the top of the root was soft when squeezed between thumb and forefinger. Sequential measurements of the length of the row diseased were summed over time, and disease severity expressed as the percentage of the length of row diseased.

Measurement of ambient environmental conditions. Soil temperature 3–4 cm below the soil surface and air temperature and relative humidity within the canopy near the crown of the plants were monitored with thermistors and recorded continuously using a data logger (model 101 temperature probe and model CR-21 Micrologger, Campbell Scientific Inc., Logan, UT). Air temperature and relative humidity were measured with aspirated wet-bulb and dry-bulb thermistors housed in an insulated polyvinylchloride pipe and aspirated at a rate of approximately 2 m/sec with an electrically driven fan (9). Moisture was maintained around the wet-bulb thermistor by covering it with a cotton shoestring connected to a distilled water reservoir. Relative humidity was calculated from the wet-bulb and dry-bulb temperatures. Amounts of natural precipitation and irrigation applied to the plots, collectively called incident water, were recorded by research station personnel.

Effect of plant canopy density on epidemic development. A field previously cropped to carrots was selected for use during 1986. Carrots were seeded as described previously on 25 March 1986 and experimental plots were established on 12 June 1986. Plots consisted of two rows × 7.6 m (15.2 linear meter of row). Initial plant density was approximately 52 plants per meter. Variations in root density and canopy density were achieved by thinning the plants, clipping the petioles of the plants, or both. In thinned rows, plant density was about 26 per meter. During clipping, all petioles were cut as close as possible to the crown of the plants with hand-held pruning shears and the foliage removed from the plot. Foliage was removed, where appropriate, from approximately every other plant. Clipping was repeated as necessary throughout the season. Four canopy environments were established: unthinned plants, unclipped petioles; unthinned plants, clipped petioles; thinned plants, unclipped petioles; and thinned plants, clipped petioles (Fig. 1). The canopy in rows in which plants were not thinned and petioles were unclipped was comparable to that in

rows that were unthinned in 1985, while the canopy in rows in which plants were thinned and petioles unclipped was comparable to that in rows that were thinned in 1985 and all rows in 1984. Treatments, consisting of 0, 4, and 10 initial foci per row, were arranged in randomized complete blocks with four replications and were established as before. Disease severity was evaluated as previously described. Over the course of the experiment, disease foci from naturally occurring inoculum developed within the plots. Location and date of focus occurrence were recorded, and disease severity from each focus was evaluated as previously outlined. Soil temperature, air temperature, and relative humidity were monitored continuously and recorded as previously described in an unthinned, unclipped plot and a thinned, clipped plot. Incident water also was recorded by experiment station personnel. Soil samples were taken concurrently with disease evaluations, and percent soil moisture was determined gravimetrically.

Analysis of data. Data from all experiments were analyzed using the Statistical Analysis System (19,20). Area under the disease progress curve was calculated for all plots from percent disease severity values with the midpoint rule (23):

$$\text{AUDPC} = \sum_{i=1}^{n-1} (Y_{i+1} - Y_i)/2 \times (t_{i+1} - t_i)$$

where t_i = time in days, $i = 0$ to n, and Y_i = disease severity on day i. Standardized AUDPC (SAUDPC) was calculated by dividing AUDPC by the highest AUDPC among plots with that number of foci per row and multiplying by 100. Exponential, linear, and monomolecular models were used to calculate the rate of disease increase. In cases where disease severity was zero, 0.001 was added to the disease severity value before transformation. In the exponential model, the natural logarithm of disease severity (ln Y) was the dependent variable and time in days was the independent variable. In the monomolecular model, transformed disease severity value (ln $[1/(1 - Y)]$) the dependent variable and time in days was the independent variable. The slope of the regression lines thus obtained was the rate parameter for disease. Goodness of fit by a model was evaluated by examination of coefficient of determination (r^2) values and plots of standardized residuals vs. predicted values from regression analysis. In cases where the r^2 values of two models were not numerically different, plots of standardized residuals were given greater importance in determining goodness of fit by a model. The relationships between the dependent variables, AUDPC, and rate parameter derived
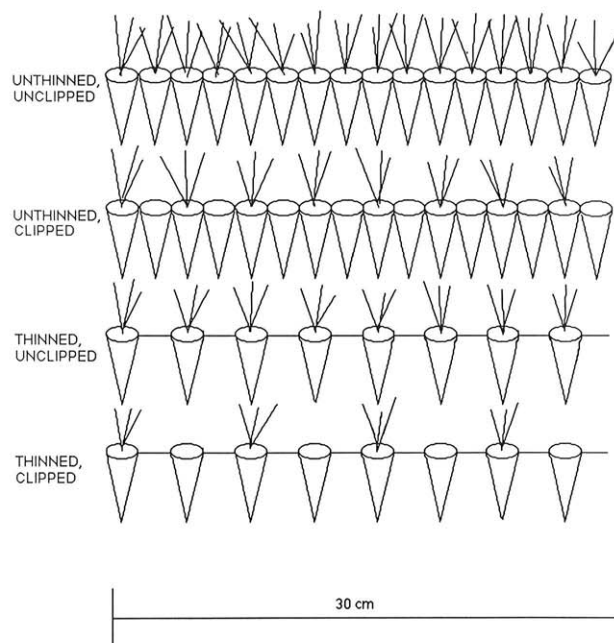

Fig. 1. Plant environments established to investigate the effects of canopy thickness and host density on epidemics of southern blight of processing carrot caused by *Sclerotium rolfsii* in 1986.

from the most appropriate model and the independent variable, number of disease foci per row, were examined using linear and quadratic models in regression analysis.

Correlation coefficients between air temperature, soil temperature, and number of hours of relative humidity greater than 95%, and increase in disease, measured as centimeters per focus per day, were calculated. In the 1986 experiment, focus development from natural inoculum in many of the plots resulted in dissimilar numbers of foci per row in each plot. Thus, for analysis, distinctions between the initial number of foci per row were ignored and each plot, with a unique number of foci, was treated separately. Rate of occurrence of natural foci was calculated as outlined above for rate of disease increase. The spatial arrangement of natural foci among the four contiguous replicate blocks of the experiment was tested for goodness of fit, with a chi-square test, to seven statistical probability distribution models (Poisson, Poisson with zeroes, negative binomial, Thomas double Poisson, Neyman type A, Poisson binomial, and logarithmic with zeroes) using a FORTRAN program developed by Gates and Ethridge (10). The influence of plant microclimate (host density and canopy density) was evaluated using the rate parameter, rate of occurrence of natural foci, and AUDPC as dependent variables in separate linear regression models.

RESULTS

Characteristics of epidemics of southern blight in 1984 and 1985.
The pathogen spread from the initial foci in both directions along the row and diseased plants were evident within 3–7 days. Small amounts of infection from natural inoculum resulted in disease in the plots having zero foci per row. Disease severity was highest in plots with high plant density (52 plants per meter) and highest number of initial foci (10 foci per row); up to 55% of the length of row was diseased in these plots (Fig. 2). In plots with 10 foci per row and 26 plants per meter in 1984 and 1985, SAUDPC was 70.5 and 54.1% of that with 52 plants per meter (Table 1). In the 1984 plots, final disease severity generally increased with an increase in initial focus number; however, plant tissue was limiting, due to
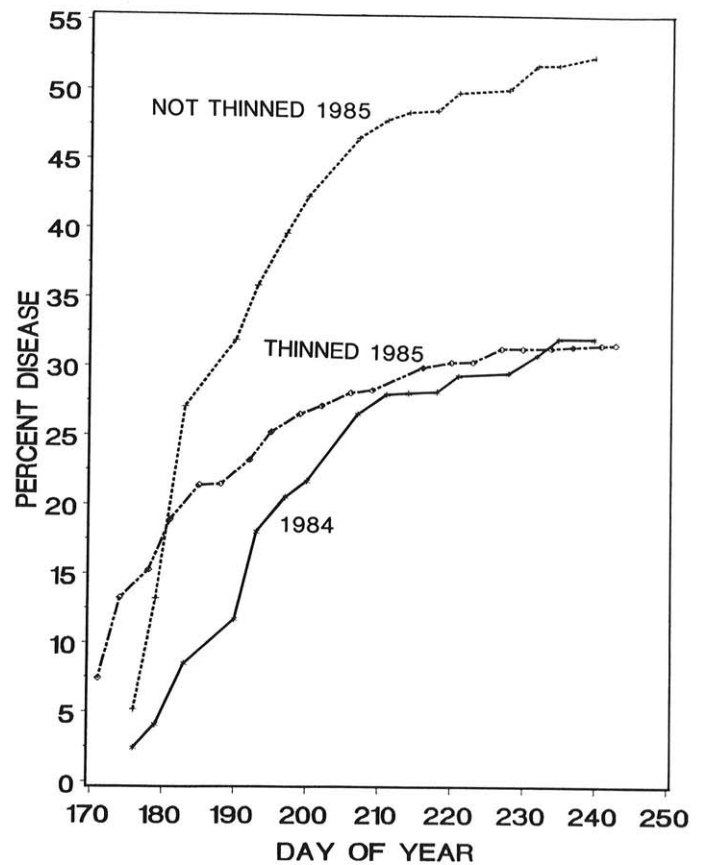

Fig. 2. Disease progress of southern blight of processing carrot caused by *Sclerotium rolfsii* in plots with 10 disease foci per row. Solid line = 1984 plots (26 plants per meter); dashed line = thinned 1985 plots (26 plants per meter); dotted line = not thinned 1985 plots (52 plants per meter). Disease severity values are the mean of five replicate plots.

TABLE 1. Effects of initial number of foci of *Sclerotium rolfsii* established per row and density of carrot plants on AUDPC and the infection rate of southern blight at one location in 2 yr

Foci/row	Year[a]	Density[b]	AUDPC[c]	SAUDPC[d]	Infection rate[e]	Error of estimate	Model[f]	R^{2}[g]
0	1984	26	165.35 ± 2.24	63.8	···			···
	1985	26	71.68 ± 1.92	27.7	···			···
	1985	52	259.01 ± 5.39	100.0	···			···
2	1984	26	523.54 ± 3.36	80.1	0.018	0.0016	E	0.573
	1985	26	424.03 ± 5.15	64.9	0.090	0.012	E	0.438
	1985	52	653.21 ± 7.39	100.0	0.322	0.025	L	0.684
4	1984	26	889.14 ± 5.46	72.7	0.206	0.021	L	0.475
	1985	26	562.91 ± 5.83	46.0	0.211	0.017	L	0.666
	1985	52	1,223.32 ± 13.37	100.0	0.518	0.034	L	0.753
6	1984	26	1,179.80 ± 6.34	73.8	0.014	0.0014	E	0.504
	1985	26	804.39 ± 9.37	50.3	0.361	0.033	L	0.598
	1985	52	1,599.64 ± 15.94	100.0	0.583	0.043	L	0.698
8	1984	26	1,751.78 ± 10.22	95.8	0.004	0.0002	M	0.731
	1985	26	1,069.70 ± 10.12	58.5	0.338	0.038	L	0.502
	1985	52	1,829.20 ± 16.62	100.0	0.560	0.046	L	0.655
10	1984	26	1,866.52 ± 10.62	70.5	0.004	0.0002	M	0.682
	1985	26	1,432.58 ± 13.80	54.1	0.006	0.001	M	0.423
	1985	52	2,246.58 ± 21.85	100.0	0.010	0.001	M	0.604

[a] Disease severity was assessed 21 and 16 times in 1984 and 1985, respectively.
[b] Expressed as number of carrot plants per linear meter of row.
[c] Calculated using percent disease using the midpoint rule (23) and expressed as AUDPC ± standard error.
[d] Standardized AUDPC = (AUDPC/highest AUDPC for rows with the same number of foci) × 100.
[e] Rate = slope of the regression line for the appropriate model.
[f] E = exponential model (ln Y vs. time in days); L = linear model (Y vs. time in days); M = monomolecular model (ln [1/(1 − Y)] vs. time in days). Results presented are for the one model judged to be most appropriate from examining the R^{2}-value and residual plots.
[g] R^{2} = correlation coefficient for the appropriate model.

convergence of some adjacent foci, at eight to 10 foci per row; the disease progress curves for these plots converged at the end of the season (Fig. 3). This was not observed in either thinned or not thinned treatments in 1985 even though final disease severity was higher in not thinned plots in 1985 than in plots of 26 plants per meter in 1984.

The rate parameter, derived from a monomolecular model, was the same for the 1984 plots having eight and 10 foci per row (Table 1). In plots having eight and 10 foci per row, disease severity values reached a maximum at about 32% on day 227 due to convergence of some adjacent foci. Consequently, disease severity values for days after 227 were not included in the calculation of the rate parameter for these plots. Disease increase for plots having two or six foci per row was best described by an exponential model, and disease increase for plots having four foci per row was best described by a linear model.

Rate of disease increase in all plots having 52 plants per meter in 1985 was best described by a linear or a monomolecular model. The rate parameter for plots having six foci per row was higher than that for plots having eight foci per row; however, the rate parameter generally increased with increasing number of foci per row. AUDPC and the rate parameter were significantly influenced by the initial number of disease foci per row. The relationship between AUDPC and the number of initial foci per row was linear (AUDPC = 235.9 + 226.4 [number of initial foci]; $R^2 = 0.82$), and no systematic trend was observed in plots of residuals from regression equations for AUDPC vs. number of foci per row.

In plots having 26 plants per meter and four, six, and eight foci per row in 1985, rate of disease increase was best described by a linear model, while in plots having two foci per row, rate was best described by an exponential model, and rate was best described by a monomolecular model in plots having 10 foci per row. The rate parameter and AUDPC generally increased with increasing number of foci per row, and were significantly influenced by the initial number of foci per row (AUDPC = 79.39 + 129.0 [number of

initial foci]; $R^2 = 0.67$).

Rate of disease increase, measured as centimeters per focus per day, was significantly correlated ($r = 0.5$, $P = 0.05$) with fluctuations in the number of hours of relative humidity greater than 95% in plots having 26 plants per meter and four to 10 initial foci/row. Rate of disease increase and other environmental variables were not correlated.

Effects of plant and canopy density in 1986. Interplant growth of mycelium and frequent occurrence of natural foci contributed to very high disease severity in rows in which plants were not thinned and petioles not clipped. Disease severity and rate of progress were least in rows in which plants were thinned and petioles clipped, where interplant growth of mycelium apparently was restricted by large interplant distances and lower relative humidity in the canopy. Variation in rate of disease increase over time, measured as centimeter per focus per day, was not, however, well correlated with ambient environmental conditions monitored in the plots.

The combination of thinning the plants and clipping the petioles significantly influenced final disease severity. A significant interaction between the combination of thinning the plants and removing the foliage and the initial number of foci per row was observed for the final number of foci ($P = 0.02$) and final disease severity ($P = 0.08$). A significant negative correlation was observed between the rate parameter, derived from a linear model, and the y-axis intercept of a linear regression line.

The effect of plant and canopy density on the rate of occurrence of natural foci was not significant in the linear regression model, but the initial number of foci per row did influence the final number of natural foci in all plant and canopy densities. In rows having zero or four established foci, many more natural foci were detected than in rows with 10 established foci. The frequency of occurrence of natural foci in the four contiguous replicate blocks was best described by the negative binomial probability distribution, and the variance-to-mean ratio consistently exceeded unity, indicating a clustered spatial arrangement of natural foci. Change in the number of foci over time significantly influenced change in disease severity over time. Differences in the slopes of regression lines (maximum disease vs. number of foci) indicated that the relationship between change in disease severity and change in the number of foci per plot differed significantly across plant and canopy densities (Fig. 4).

Rate of disease increase in rows having four initial foci, in which plants were unthinned and petioles were unclipped, was best described by a monomolecular model. Rate of disease increase was best described by an exponential model in rows in which plants were unthinned but petioles were clipped and rows in which plants were thinned and petioles were clipped, each with four initial foci. In the remaining rows, rate of disease increase was best described by a linear model (Table 2).

DISCUSSION

Sclerotia of *S. rolfsii* are aggregated spatially in naturally infested fields (17), and infected plants initially appear as foci (25). Enlargement of disease foci is influenced by many factors, including plant density, canopy density, and conditions in the upper few centimeters of soil.

In the present studies, AUDPC and rate of disease increase were higher in rows having 52 plants per meter than in rows having 26 per meter. Interplant transmission in all rows was primarily via root contact; however, *S. rolfsii* can grow from plant to plant on senescent or dead petioles (14). In rows in which carrot plants were unthinned and petioles clipped from approximately every other plant, rate of disease increase and maximum disease were higher than that in rows in which carrot plants were not thinned. Physiological changes in roots from which petioles were clipped, such as increased exudation, may have contributed to increased disease severity and rapid growth of *S. rolfsii* along the row. Burdon and Chilvers (5,6) found that decreasing host density of garden cress seedlings (*Lepidium sativum* L.) had approximately the same effect on damping-off as a proportionate decrease in inoculum density of *Pythium irregulare* Buis. The rate of advance
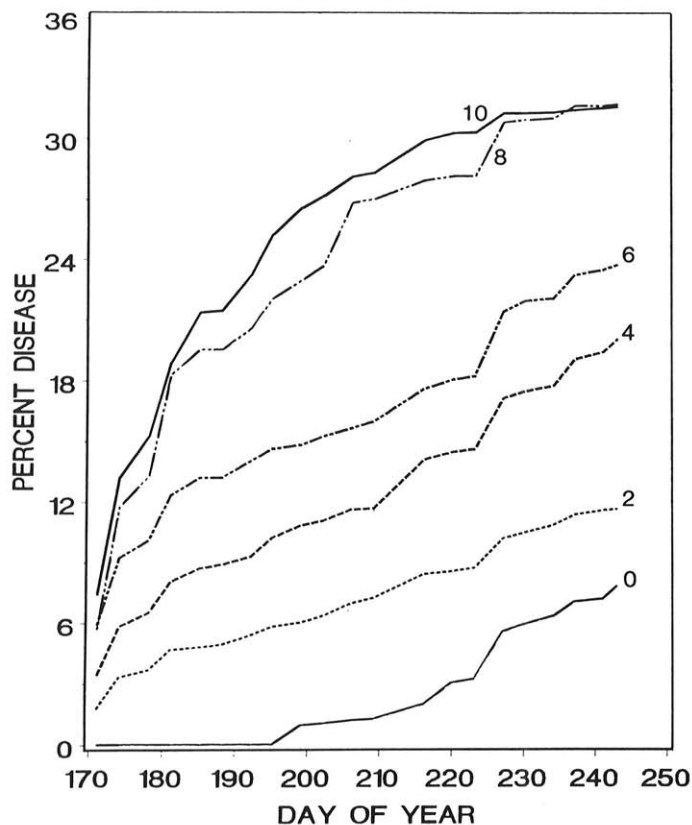

Fig. 3. Disease progress of southern blight of processing carrot caused by *Sclerotium rolfsii* in 1984. Numbers indicate the number of initial foci per row. Plant density was 26 per meter of row. Disease severity values are the mean of five replicate plots.

of the disease front and the rate of multiplication of disease were inversely proportional to plant spacing. Similar results were obtained in studies of infection of onion plants (*Allium cepa* L.) by *Sclerotium cepivorum* (22) and infection of sunflower (*Helianthus annuus* L.) by *Sclerotinia sclerotiorum* (Lib.) de Bary (11). In our studies, disease severity in the 1985 plots having 26 plants per meter was approximately 50% that of rows having 52 plants per meter. Although the spacing of the carrot roots was not specifically measured, the mean distance between plants in rows having 26 plants per meter was greater than in rows having 52 plants per meter.

Although disease increase resulted from mycelial growth of *S. rolfsii* in the rows of processing carrot, we also observed the basidial state, *Athelia rolfsii* (Curzi) Tu and Kimbrough. Hymenia, usually less than 2 cm² in size, were found on senescent or dead plant tissue and were observed more frequently on cool foggy mornings; we never saw them during hot or dry weather or in the hotter hours of the day. Because the basidial state in nature was ephemeral, basidiospores probably had little or no effect on epidemics of southern blight of processing carrot. Basidiospores of an isolate of *S. rolfsii* from turfgrass can reinfect turf, but an exogenous nutrient source may facilitate infection (16). Further investigation on the possible role of basidiospores in epidemics caused by *S. rolfsii* is needed.

In general, the epidemics of southern blight of processing carrot in 1984 and 1985 could be described with either a linear or monomolecular model. Increase in disease generally was not constrained by a lack of host tissue until late in the season, and interplant mycelial growth of the pathogen contributed to increase in disease severity. In rows with 26 plants per meter and eight to 10 foci in 1984; however, disease progress was best described by a monomolecular model. Asymptotic levels of disease of less than 100% in many of the plots were attributed to convergence of foci,

or focus extinction. At the point of convergence of two foci, further disease development was precluded by lack of host tissue available for colonization. Ambient environmental conditions were not limiting, since disease severity continued to increase in adjacent rows with six or fewer foci.

S. rolfsii generally is not considered to be a foliar pathogen, but the mycelium grows on the soil surface and infects plants at the soil-air interface. Canopy microclimate and conditions at the soil surface are, therefore, important in the epidemiology of diseases caused by this fungus. In the present studies, AUDPC was greatest in plots having the thickest, most humid canopy environment. Disease development was favored by increased number of hours of relative humidity >95% per day within the canopy. Disease increase was not, however, influenced significantly by incident water or by percent soil moisture. Diseases caused by *S. rolfsii* are more severe under conditions of fluctuating moisture (2,12), and the sandy nature of the soil in the present study (>75% sand) enhanced rapid drainage and drying of the upper layers of the soil profile. Large fluctuations in soil moisture increased infection of lettuce (*Latuca sativa* L.) by *Sclerotinia sclerotiorum* (1).

In addition to plant density, canopy architecture (the degree of "openness" of the canopy) has been reported to influence infection of plants by soilborne fungi (7,21). In the present studies, disease severity was lowest in plots having the thinnest, most open canopy, those that were thinned and clipped in 1986 and those that were thinned in 1985. Number of hours of relative humidity >95% in plots that were thinned and clipped was consistently one-third to one-half that of the not thinned and not clipped plots, and reduced relative humidity in the canopy may have contributed to decreased disease severity. Increased solar radiation impinging on the soil also may have reduced growth of *S. rolfsii*, since bright sunlight can cause dessication and death of hyphae (2).

The relationship between the number of foci and final disease severity depended on the microclimate in the rows in the plots in 1986. In general, increasing number of foci per row resulted in more disease. Punja et al (18) found that disease incidence as a function of time was linear and resulted from both an increase in the number of disease foci and interplant mycelial growth. In 1986, the rate of disease increase depended on initial disease severity, indicated by a negative correlation between the rate, derived from a linear model, and the *y*-axis intercept of a linear regression line. Such a relationship is characteristic of a polycyclic disease (13). Disease increase as a function of time was linear in all rows in 1985 with 52 plants per meter and in most of the rows in 1986 (Tables 1 and 2). The apparent lack of disease foci from naturally occurring inoculum in rows with 10 initial foci may have been due to an inability to detect natural foci in these rows. The natural foci detected in the four contiguous replicates were aggregated spatially and by chance alone may have occurred more frequently in rows with zero or four initial foci.

Environmental conditions measured in the present studies, air temperature, soil temperature, relative humidity, and incident water, generally were not limiting to epidemics of southern blight of processing carrot. Rate of disease increase in thinned rows having four to 10 foci in 1985 was greater during periods with more hours per day in which relative humidity exceeded 95%, but fluctuations in rate of disease increase in other plots were not significantly correlated with any of the measured environmental parameters. The lack of variation in disease progress attributable to environmental variation in the present studies was surprising. Previous investigators have observed that moist conditions (2,12) or fluctuating wet and dry weather (4,12) favor infection by *S. rolfsii* and *S. sclerotiorum* (1). The lack of a quantitative relationship between environment and disease in the present studies may be due to: insufficient sensitivity of the sensors used to detect slight differences in environmental conditions influencing epidemics, or a uniformly favorable environment for development of epidemics, i. e., the weather was never unfavorable for mycelial growth of the fungus. Further quantification of the effects of environment on disease development will facilitate development of predictive models for diseases caused by *S. rolfsii*.

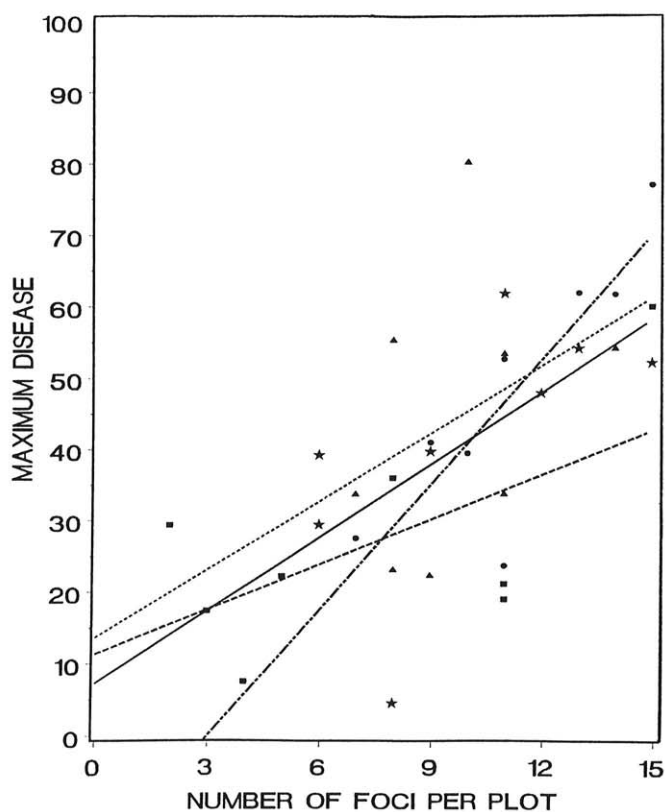Regression analysis of data from plots in 1986 indicated that

Fig. 4. Relationship between final disease severity in the plot and number of foci per row across plant environments. Solid line and stars = not thinned and not clipped rows, slope = 3.43; dotted line and triangles = thinned rows, slope = 3.21; dashed line and squares = thinned and clipped rows, slope = 2.11; and dotted and dashed line and circles = not thinned and clipped rows, slope = 5.89. Data for rows with 10 initial foci are not included on the graph.

TABLE 2. Effects of initial number of foci of *Sclerotium rolfsii* established per row and plant microclimate on the infection rate of southern blight of processing carrot at one location in one year

Plant microclimate[a]	Initial foci/row	YMAX[b]	Infection rate[c]	Error of estimate	Model[d]	R^{2}[e]
Unthinned, unclipped	0	42.88	0.633	0.061	L	0.607
	4	40.09	0.008	0.001	M	0.485
	10	51.84	0.702	0.032	L	0.872
Thinned, unclipped	0	56.08	0.799	0.062	L	0.707
	4	33.63	0.442	0.054	L	0.494
	10	22.63	0.352	0.031	L	0.644
Unthinned, clipped	0	42.87	0.574	0.063	L	0.543
	4	54.28	0.036	0.003	E	0.645
	10	35.54	0.530	0.064	L	0.493
Thinned, clipped	0	19.38	0.275	0.031	L	0.525
	4	34.43	0.119	0.015	E	0.490
	10	24.66	0.381	0.036	L	0.613

[a] Thinned = 26 plants/m: unthinned = 52 plants/m. See text for details on clipping foliage.
[b] YMAX = asymptotic level of disease severity. Values are the mean of four plots.
[c] Rate = slope of the regression line for the appropriate model.
[d] E = exponential model (ln Y vs. time in days); L = linear model (Y vs. time in days); M = monomolecular model (ln $[1/(1 - Y)]$ vs. time in days). Results presented are for the one model judged to be most appropriate from examining the R^{2}-values and residual plots.
[e] R^{2} = correlation coefficient for the appropriate model.

change in number of foci over time significantly influenced change in disease severity over time. Evaluation of epidemic development in processing carrot by disease severity ratings is a tedious and labor intensive procedure, and systematic errors may be incurred during repeated measurements of disease foci. Results of the present studies indicate that disease incidence, i. e., the number of foci, may be useful to quantify epidemics of southern blight of processing carrot, rather than disease severity evaluations. As previously stated, our results also show that the relationship between the number of foci and disease severity differed over plant environment. Further investigation is needed to quantitatively define the relationships between number of foci, expansion of foci, and disease severity.

LITERATURE CITED

1. Adams, P. B., and Tate, C. J. 1975. Factors affecting lettuce drop caused by *Sclerotinia sclerotiorum*. Plant Dis. Rep. 59:140-143.
2. Aycock, R. 1966. Stem rot and other diseases caused by *Sclerotium rolfsii*. N.C. Agric. Exp. Stn. Tech. Bull. 174. 202 pp.
3. Backman, P. A., Rodriguez-Kabana, R., and Williams, J. C. 1975. The effect of peanut leafspot fungicides on the nontarget pathogen, *Sclerotium rolfsii*. Phytopathology 65:773-776.
4. Boyle, L. W. 1961. The ecology of *Sclerotium rolfsii* with emphasis on the role of saprophytic media. Phytopathology 51:117-119.
5. Burdon, J. J., and Chilvers, G. A. 1975. A comparison between host density and inoculum density effects on the frequency of primary infection foci in *Pythium*-induced damping-off disease. Aust. J. Bot. 23:899-904.
6. Burdon, J. J., and Chilvers, G. A. 1975. Epidemiology of damping-off disease (*Pythium irregulare*) in relation to density of *Lepidium sativum* seedlings. Ann. Appl. Biol. 81:135-143.
7. Coyne, D. P., Steadman, J. R., and Anderson, F. N. 1974. Effect of modified plant architecture of great northern dry bean varieties (*Phaseolus vulgaris*) on white mold severity, and components of yield. Plant Dis. Rep. 58:379-382.
8. Crowe, F. J., and Hall, D. H. 1980. Vertical distribution of sclerotia of *Sclerotium cepivorum* and host root systems relative to white rot of onion and garlic. Phytopathology 70:70-73.
9. Davis, J. M., Bruck, R. I., Runion, G. B., and Mowry, F. L. 1983. An aspiration system for meteorological sensors used in epidemiological studies. Phytopathology 73:1246-1249.
10. Gates, C. E., and Ethridge, F. G. 1972. A generalized set of discrete frequency distributions with FORTRAN program. Int. Assoc. Math. Geol. 4:1-24.
11. Huang, H. C., and Hoes, J. A. 1980. Importance of plant spacing and

sclerotial position to development of Sclerotinia wilt of sunflower. Plant Dis. 64:81-84.
12. Jenkins, S. F., and Averre, C. W. 1986. Problems and progress in integrated control of southern blight of vegetables. Plant Dis. 70:614-619.
13. Pfender, W. F. 1982. Monocyclic and polycyclic root diseases: Distinguishing between the nature of the disease cycle and the shape of the disease progress curve. Phytopathology 72:31-32.
14. Punja, Z. K., Carter, J. D., Campbell, G. M., and Rossell, E. L. 1986. Effects of calcium and nitrogen fertilizers, fungicides, and tillage practices on incidence of *Sclerotium rolfsii* on processing carrots. Plant Dis. 70:819-824.
15. Punja, Z. K., and Grogan, R. G. 1981. Eruptive germination of sclerotia of *Sclerotium rolfsii*. Phytopathology 71:1092-1099.
16. Punja, Z. K., and Grogan, R. G. 1983. Germination and infection by basidiospores of *Athelia* (*Sclerotium*) *rolfsii*. Plant Dis. 67:875-878.
17. Punja, Z. K., Smith, V. L., Campbell, C. L., and Jenkins, S. F. 1985. Sampling and extraction procedures to estimate numbers, spatial pattern, and temporal distribution of sclerotia of *Sclerotium rolfsii* in soil. Plant Dis. 69:469-474.
18. Punja, Z. K., Smith, V. L., and Jenkins, S. F. 1984. Relationship of disease incidence to inoculum density in *Sclerotium rolfsii* root rot of processing carrots. (Abstr.) Phytopathology 74:813.
19. SAS Institute, Inc. 1985. SAS User's Guide: Basics. 1985 edition. SAS Institute Inc., Cary, NC. 1290 pp.
20. SAS Institute, Inc. 1985. SAS User's Guide: Statistics. 1985 edition. SAS Institute Inc., Cary, NC. 956 pp.
21. Schwartz, H. F., Steadman, J. R., and Coyne, D. P. 1978. Influence of *Phaseolus vulgaris* blossoming characteristics and canopy structure upon reaction to *Sclerotinia sclerotiorum*. Phytopathology 68:465-470.
22. Scott, M. R. 1956. Studies of the biology of *Sclerotium cepivorum* Berk. II. The spread of white rot from plant to plant. Ann. Appl. Biol. 44:584-589.
23. Shaner, G., and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. Phytopathology 67:1051-1056.
24. Shew, B. B., and Beute, M. K. 1984. Effects of crop management on the epidemiology of southern stem rot of peanut. Phytopathology 74:530-535.
25. Shew, B. B., Beute, M. K., and Campbell, C. L. 1984. Spatial pattern of southern stem rot caused by *Sclerotium rolfsii* in six North Carolina peanut fields. Phytopathology 74:730-735.
26. Smith, V. L., Jenkins, S. F., and Benson, D. M. 1986. Factors affecting epidemics of southern blight of processing carrot in North Carolina. (Abstr.) Phytopathology 76:1083.