Influence of Soil Water Status on the Epidemiology of Tobacco Black Shank

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ABSTRACT

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The relationship of soil water status to the rate of increase in mortality of tobacco in a field infested with *Phytophthora parasitica* var. *nicotianae* was examined. Multiple cycles in the increase in mortality occurred in the susceptible cultivar, Hicks. Generally only a single cycle in the increase in mortality occurred in the resistant cultivar, Speight G-28; this was attributed to the delay of appreciable mortality until after flowering, which limited time for secondary cycles. Increases in the average rates of change in mortality were negatively cross correlated with increases in the average rates of change in soil water status for both cultivars. These increases differed between cultivars in both their magnitudes and the times of the initial and maximum responses. Ordinary runs analysis was conducted over time to ascertain the temporal pattern of the random or nonrandom occurrence of plant mortality. Increases in the percentages of plots with a nonrandom occurrence of plant mortality coincided with periods of increased soil moisture. Evidence for the increase or spread of inoculum was more pronounced in the susceptible than in the resistant cultivar as demonstrated by the eventual near uniformity of mortality throughout plots of Hicks compared with plots of Speight G-28.

Additional key words: *Nicotiana tabacum*, soil water matric potential.

Black shank is a destructive disease of tobacco (*Nicotiana tabacum* L.) caused by *Phytophthora parasitica* Dast. var. *nicotianae* (Breda De Haan) Tucker. The disease cycle generally is initiated with the infection of roots by residual inoculum in the soil. This inoculum is typically in the form of chlamydospores; infection occurs directly by the formation of germ tubes from chlamydospores or after the formation of sporangia and zoospores (13). Inoculum increases within the rhizospheres of infected plants during the course of the epidemic (6,11) by the formation of chlamydospores and sporangia with the subsequent release of zoospores. The final stages of disease development are typified by irreversible wilting of the plants followed by blackening of the lower stems.

The influence of soil water matric potential (ψ_m) on the formation of sporangia, the release and movement of zoospores, and disease development has been studied extensively in *Phytophthora* and related genera (3,4). Sidebottom and Shew (19) observed that production of sporangia on mycelial mats of *P. p.* var. *nicotianae* placed in soil equilibrated at several values of ψ_m varied greatly. The greatest numbers of sporangia were produced at values of ψ_m ranging from -40 to -250 mbar. Shew (18) observed that 60, 27, and 7% of the plants of the susceptible cultivar, Hicks, became infected within 19–21 days at constant values of ψ_m of -10, -20, and -50 mbar, respectively. Saturation of the soil for as little as 0.5 hr induced the release of zoospores and was sufficient to overcome the limiting effects of low ψ_m on disease development.

In greenhouse studies, Wills (20) observed that mortality of tobacco over a 90-day period averaged 71% when the soil alternately was saturated, then allowed to dry to the wilting point of the plants, compared with 32% mortality when the soil was maintained at field capacity. McCarter (14) demonstrated that roots of susceptible and resistant tobacco cultivars were infected by *P. p.* var. *nicotianae* over a range of constant soil moistures from 19–100% water-holding capacity, but disease development was delayed at soil moisture contents of less than 59% water-holding capacity. Root infections resulted in necrotic lesions that exhibited extensive soft rot at high soil moistures and restricted dry rot at low soil moistures.

The influence of soil water status on disease development by root-infecting species of *Phytophthora* under field conditions has received little attention. Wills (20) concluded that soil moisture was the only important variable affecting black shank development under normal cultural practices. Jacobi et al (10) developed multiple regression models for black shank progression based on the environmental parameters, air temperature, rainfall, and number of drought days. Although these models explained 61–81% of the variation in disease progression, these environmental parameters do not influence pathogen activity or disease development directly. Under field conditions, the processes of sporangium production and zoospore release should be repeated as the soil undergoes cycles of wetting and drying. Therefore, disease progression should be reflected more directly by soil water status than by the above parameters.

Another aspect of black shank development that will be influenced by soil water status is the spread of secondary inoculum during the course of the epidemic. In a field known to be initially free of the pathogen, Shew (17) reported that the pathogen had spread from individual infected plants to adjacent plants in 14 of 18 rows of the susceptible cultivar, Hicks, within 45 days. Spread of the pathogen to adjacent plants in resistant cultivars was observed in no more than five of 18 rows, but the pathogen had spread through the soil in all rows. However, Campbell et al (2) concluded from ordinary runs analysis that there was no evidence for the plant-to-plant spread of inoculum during the course of epidemic development in a field previously infested with *P. p.* var. *nicotianae*.

The objectives of this study were to examine the relationship between soil water status and the rate of increase in black shank-induced mortality under field conditions and to examine the temporal pattern of the random or nonrandom occurrence of plant mortality during black shank epidemics in a field infested with *P. p.* var. *nicotianae*.

MATERIALS AND METHODS

Studies on black shank progression were conducted in a field infested with *P. p.* var. *nicotianae*. This field had been planted to

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tobacco annually since 1967 and served as a black shank disease nursery. The soil was predominantly a well-drained, Arredondo loamy sand (to a depth of 1 m) with an area of Kendrick loamy sand (to a depth of 0.5 m). The soil of the Ap horizon had a bulk density of approximately 1.45 g/cm^3.

Six pairs of plots were established for the black shank susceptible cultivar, Hicks, and the resistant cultivar, Speight G-28, in 1982 and 1983; eight pairs of plots were established in 1984. Plots were 3.75 × 12.5 m and contained four rows of 15–22 plants each. Ten-week-old tobacco plants were transplanted on 25 March 1982, 14 April 1983, and 6 and 10 April 1984. Plants were spaced 55–65 cm apart with averages of 90, 82, and 73 plants per plot in 1982, 1983, and 1984, respectively. Plot designs consisted of randomized complete block designs in 1982 and 1983, and a completely randomized design in 1984.

Plant stands were determined once the transplants had become established (after 6–10 days in 1982 and 1984 and after 25 days in 1983). Disease was assessed approximately every 4–7 days as plant mortality indicated by the first signs of irreversible wilting of the plants. Periodically during the season, sections were removed from the advance edges of stem lesions and were plated onto the PARP medium of Kannwischer and Mitchell (12) supplemented with 50 mg of hymexazol (Hymexazol, 99.4% a.i., Sankyo Co. Ltd, Tokyo, Japan) per liter of medium to confirm the presence of the pathogen. Records were kept of the dates and positions within plots that individual plants first exhibited wilt symptoms. Data from the four rows within each plot were combined and treated as a single row for runs analysis (7,8).

Soil water matric potential was monitored using vacuum gauge tensiometers (Irrometer Co., Riverside, CA) inserted to a depth of 15 cm in the soil in either the row of Hicks or the row of Speight G-28 near the center of each pair of plots. Tensiometer readings were taken daily at noon. Soil water status, assessed in terms of ψ_m (−cbar) at the 15-cm depth, was expressed as the average tensiometer reading calculated between successive disease assessment dates (−cbar/day). The relationship of the increase in black shank-induced mortality, expressed in terms of the average rates of change in mortality with respect to both chronological time, $\Delta y/\Delta t$ (% mortality/day), and soil water status, $\Delta y/\Delta\psi_m$ (% mortality/cbar), were examined with respect to the average rates of change in soil water status, $\Delta\psi_m/\Delta t$ (−cbar/day), between successive disease evaluation dates by calculation of the cross correlation coefficients (1) between these variables using the Minitab Statistical Computing System (16).

RESULTS

Different temporal patterns were observed for the increase in black shank-induced mortality when expressed in terms of chronological time compared with soil water status (Figs. 1 and 2). Cycles in the increase in mortality were more pronounced when the increase in mortality was expressed in terms of soil water status than chronological time. Multiple cycles in the increase in mortality were observed for the susceptible cultivar, Hicks. For the resistant cultivar, Speight G-28, only a single cycle in the increase in mortality was observed in 2 of 3 yr and occurred late in the growing season. For both cultivars, negative cross correlation coefficients were obtained between the average rate of increase in mortality with respect to soil water status, $\Delta y/\Delta\psi_m$, and the average rate of increase in soil water status with respect to time,

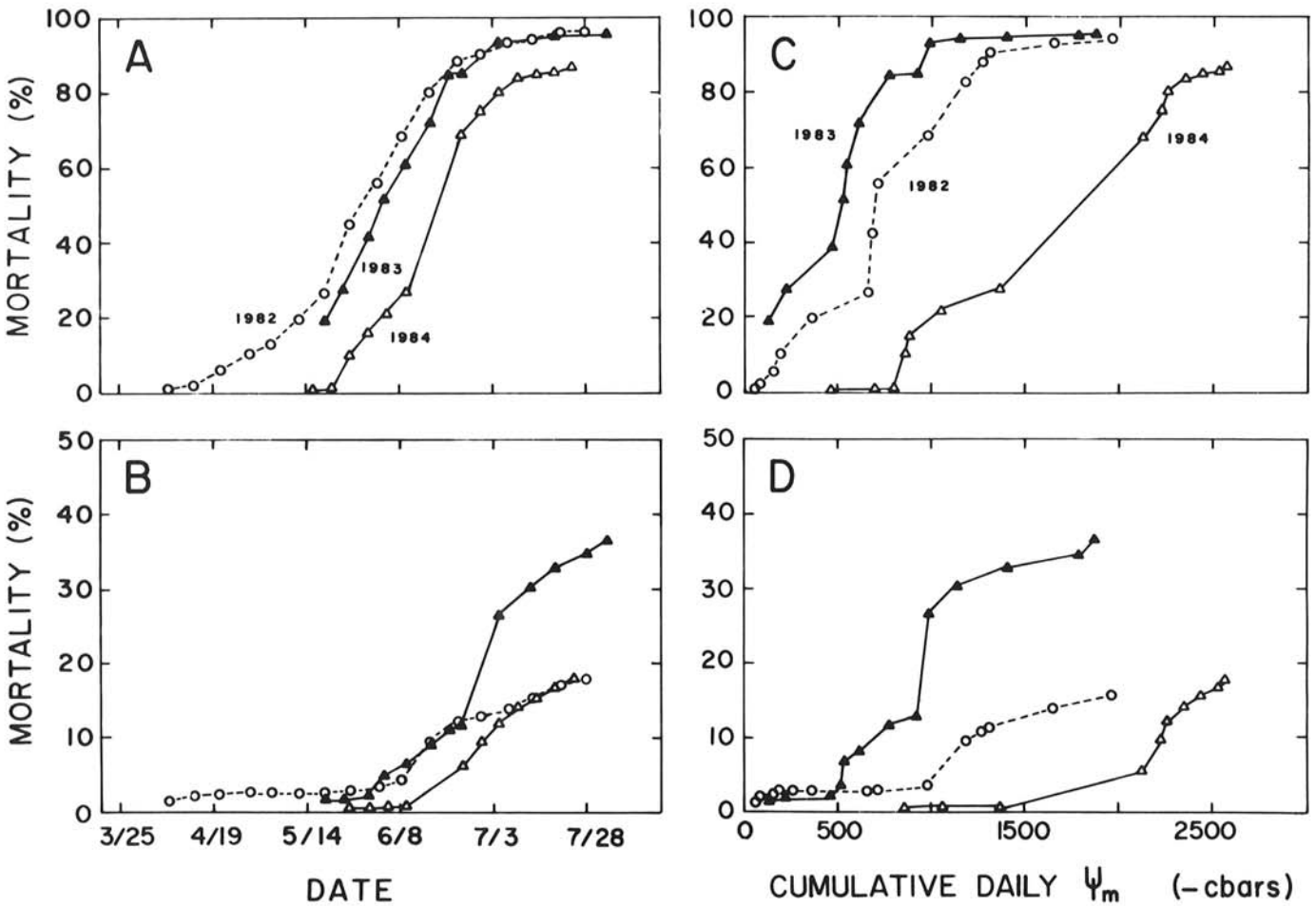

Fig. 1. Black shank-induced mortality of tobacco in 1982, 1983, and 1984: increases with respect to chronological time (days) by date in the susceptible cultivar, Hicks **(A)**, and the resistant cultivar, Speight G-28 **(B)**, and with respect to soil water status expressed as the cumulative daily tensiometer readings (−cbar) in Hicks **(C)**, and Speight G-28 **(D)**.

$\Delta\psi_m/\Delta t$, for each of the 3 yr (Table 1) and coincided with periods of increased soil moisture. The cross correlation coefficients calculated between the average rate of increase in mortality with respect to chronological time, $\Delta y/\Delta t$, and the average rate of increase in soil water status with respect to time, $\Delta\psi_m/\Delta t$, were not consistent with regard to positive or negative associations over the 3 yr.

The random or nonrandom occurrence of plant mortality, as determined by ordinary runs analysis, varied among and within plots over time. The relationship between the z statistic calculated from the runs data averaged over all plots was examined by date (Fig. 3). The value of z generally decreased over time for both cultivars in both years. For Hicks the average occurrence of plant mortality was random initially ($z > -1.64$, $P < 0.05$) but was nonrandom ($z < -1.64$) from late May through the end of the epidemics in both 1982 and 1984. For Speight G-28 the average occurrence of plant mortality was random on each disease assessment date in 1982, but was nonrandom on the final four dates in 1984.

The temporal pattern of the increase in the nonrandom occurrence of plant mortality was evident when the cumulative percentages of plots designated as nonrandom at least once during the epidemic were plotted by date (Fig. 4). Increases in the nonrandom occurrence of plant mortality coincided with periods of high soil moisture and maximum increases in $\Delta y/\Delta\psi_m$. Over the course of the epidemic, the occurrence of mortality had been designated nonrandom at least once for 95 and 75.5% of the plots of Hicks and for 22.5 and 75.5% of the plots of Speight G-28 in 1982 and 1984, respectively. The maximum percentages of plots designated as nonrandom on any given disease assessment date were 50 and 62.5 for Hicks and 22.5 and 50 for Speight G-28 in 1982 and 1984, respectively.

TABLE 1. Cross correlation coefficients calculated between the average rates of change in the increase in mortality of tobacco based on chronological time, $\Delta y/\Delta t$, or soil water status, $\Delta y/\Delta\psi_m$, and the average rates of change in soil water status between successive disease assessment dates, $\Delta\psi_m/\Delta t$, for the black shank susceptible cultivar, Hicks, and the resistant cultivar, Speight G-28

Dependent variable	Year	Cross correlation coefficient with independent variable, $\Delta\psi_m/\Delta t$	
		Hicks	Speight G-28
$\Delta y/\Delta t$	1982	−0.046	0.231
	1983	−0.325	−0.277
	1984	0.291	−0.159
$\Delta y/\Delta\psi_m$	1982	−0.453	−0.374
	1983	−0.592	−0.626
	1984	−0.379	−0.389

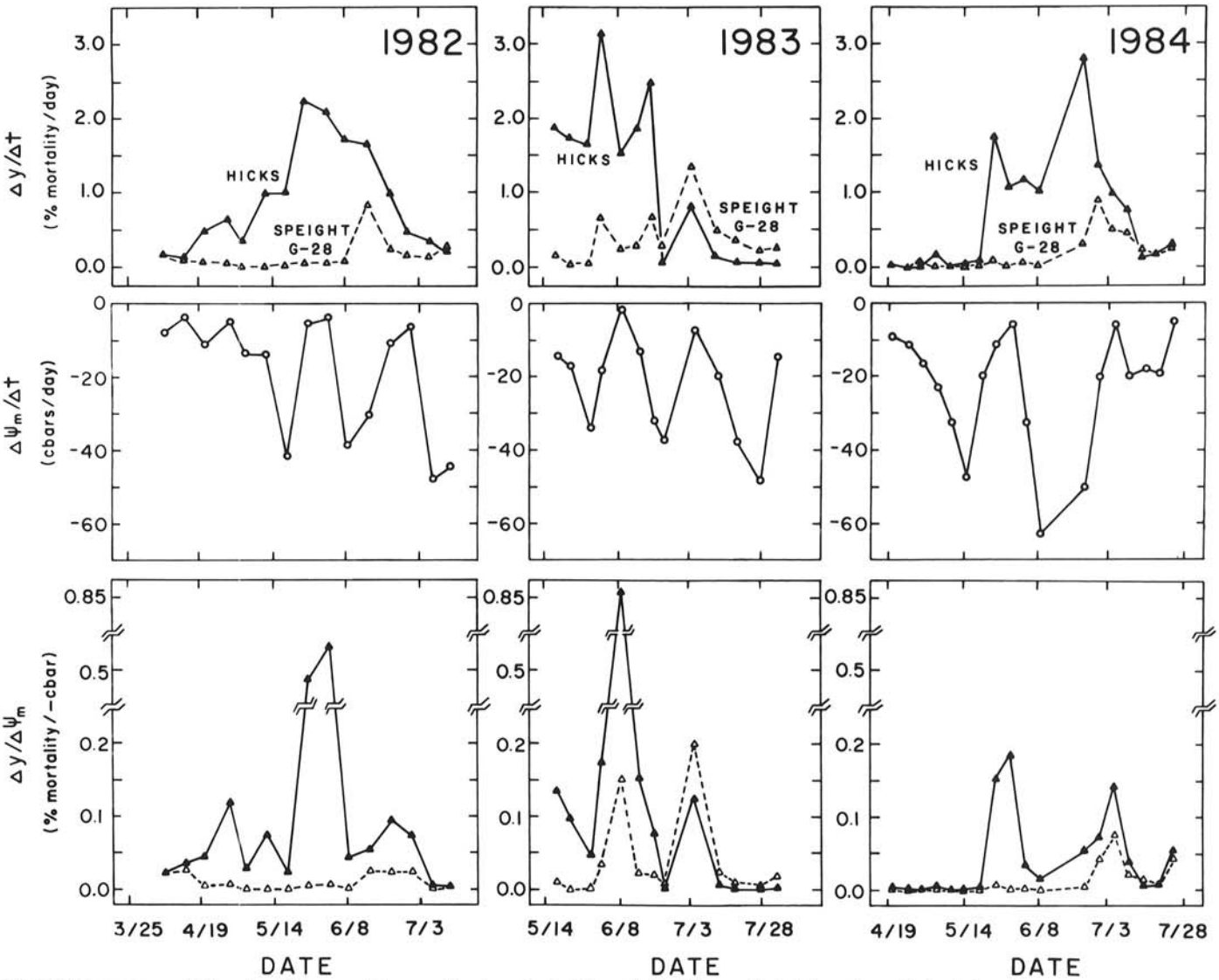

Fig. 2. The average mortality rates of tobacco with respect to chronological time, $\Delta y/\Delta t$ (% mortality/day), and cumulative daily soil water matric potential, $\Delta y/\Delta\psi_m$ (% mortality/cbar), and soil water status, $\Delta\psi_m/\Delta t$ (−cbar/day), calculated between successive black shank disease assessment dates in 1982, 1983, and 1984.

DISCUSSION

Soil water status greatly influenced the epidemiology of tobacco black shank under field conditions. The susceptible and resistant cultivars used in this study exhibited similar responses to soil water status, but they differed both in the magnitudes of these responses and the times of the initial and maximum responses. Mortality increased most rapidly in response to periods of increased soil moisture.

Increase in mortality in the susceptible cultivar, Hicks, occurred in multiple cycles as the result of plant responses to repeated infection periods associated with repeated cycles of wetting and drying of the soil throughout the growing season. In contrast, increase in mortality in the resistant cultivar, Speight G-28, generally occurred in a single cycle. This was most likely the result of the restriction of plant response primarily to infection periods after bloom; generally only a single cycle of drying and wetting occurred during this period. However, multiple cycles in the increase in mortality occurred in the resistant cultivar in 1983 and were associated with a drought period that occurred shortly after the transplants were set. Sufficient stress presumably was placed on the plants by this drought period to reduce their resistance.

The temporal pattern of the nonrandom occurrence of plant mortality led to the conclusion that there was evidence for the increase or spread of secondary inoculum of *P. p.* var. *nicotianae* during the course of the epidemic. Evidence for the increase or spread of secondary inoculum was much more evident in the susceptible cultivar, Hicks, than in the resistant cultivar, Speight G-28. This was most likely because of the differences in the extent of the plant-to-plant spread of the pathogen between susceptible and resistant cultivars, which had been demonstrated previously (17). The different conclusions reached in our study and that of Campbell et al (2) may reflect differences in the spatial pattern of the initial inoculum in the two studies. More randomly dispersed inoculum in the tests of Campbell et al (2) than in our study may have been responsible for their conclusion that there was no evidence for the multiplication and spread of the pathogen. This possibility was indicated by the fact that the final levels of mortality for Speight G-28 were considerably greater in their study than in our study (final mortality > 70 and $< 50\%$, respectively) even though the ranges of initial inoculum densities were similar for the two studies.

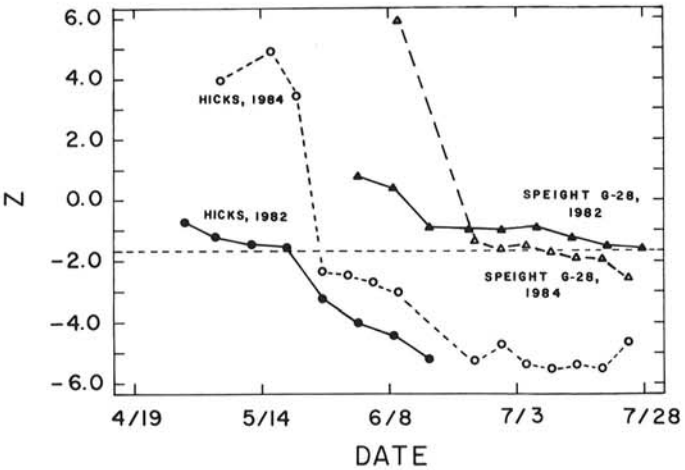

Fig. 3. The values of the *z* statistic used in ordinary runs analysis calculated from the numbers of runs averaged over all plots during the course of tobacco black shank epidemics in 1982 and 1984; the dashed line represents a *z* value of -1.64 and values less than this indicate a nonrandom occurrence of plant mortality ($P < 0.05$).

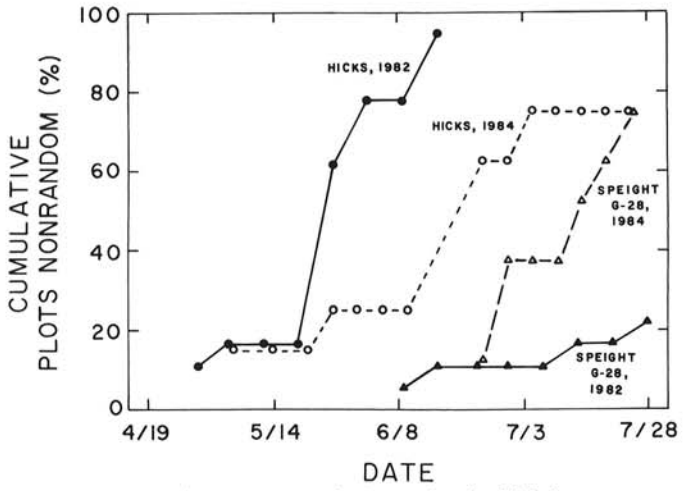

Fig. 4. The cumulative percentages of tobacco plots in which the occurrence of black shank-induced mortality was nonrandom (as determined by ordinary runs analysis) at least once during the course of epidemics in 1982 and 1984.

The nonrandom occurrence of diseased plants, generally taken to indicate that plant-to-plant spread has occurred, may result also from a nonrandom spatial pattern of inoculum in soil. Thus, the nonrandom occurrence of plant mortality observed in our study may have been the result of the observed aggregation of the initial inoculum in soil (5). This indeed may have been the case for Speight G-28. However, the temporal pattern of the increase in the nonrandom occurrence of plant mortality and the subsequent eventual near uniformity of plant mortality throughout the plots of Hicks was concluded to result from the involvement of secondary inoculum. For pathosystems with soilborne inoculum, runs analysis does not allow distinction between the alternative hypotheses of the increase of inoculum within the root systems of individual plants versus the spread of secondary inoculum between plants. Nicot et al (15) supported the use of spatial autocorrelation analysis to provide information regarding the spatial pattern of inoculum or disease on a scale larger than the units sampled. Future use of this method may allow a more definitive conclusion regarding the evidence for the plant-to-plant spread of secondary inoculum in disease development by soilborne plant pathogens in naturally infested fields.

In this study, disease assessments based on the final stages of disease development (mortality) did not allow determination of the exact nature of disease development within root systems of individual plants. Neither did the use of average rates of change in soil water status precisely reflect the daily fluctuations in soil moisture or the duration of periods of soil saturation. Furthermore, root growth dynamics, which will influence disease development considerably (9), were not addressed in this study. However, general trends were still evident. The cyclic increases in soil water status were reflected by cyclic increases in plant mortality. Periods of greatest disease increase coincided with periods of increased soil moisture. The increase or spread of inoculum in the black shank pathosystem was suggested by the temporal pattern of the nonrandom occurrence of plant mortality in the susceptible cultivar followed by the eventual near uniformity of mortality attained.

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