Effects of Plant Density on Progress of Phymatotrichum Root Rot in Cotton

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ABSTRACT

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An experiment was conducted at the Blackland Research Center, Temple, TX, to determine the effects of plant density on the increase of Phymatotrichum root rot, caused by *Phymatotrichum omnivorum*, in cotton. Disease was assessed on 15 occasions beginning 18 June and ending 20 August 1985 in 32 plots (7 × 8 m) of four replicates of eight plot density treatments: two between-row spacings, 69 and 138 cm, and four within-row plant densities, 1, 5, 9, and 13 plants per meter. A logistic model was fitted to disease progress data in each plot. Between-row spacing had no effect on the rate or other parameters of disease progress. A within-row density of five plants per meter reduced initial disease incidence (y_0) and delayed the buildup of Phymatotrichum root rot compared with nine and 13 plants per meter but had no effect on asymptotic disease incidence (M) or area under the disease progress curve (Z). The rate of disease progress (r) was higher in plots with five plants per meter because of the initial delay in Phymatotrichum root rot progress in these plots. A within-row density of one plant per meter reduced y_0, M, and Z. Initial disease incidence and Z increased linearly with plot density (overall plant density within plots) and within-row density. The asymptotic disease value (M) was exponentially related to plot density and indicated a threshold plant density of about one or two plants per meter above which M was unaffected. There were no consistent relationships between r and plot density or within-row density. The between-plant spacing that inhibited plant-to-plant spread of *P. omnivorum* in the experiment was estimated to be between 0.20 and 0.69 cm.

Additional key words: disease progress curves, nonlinear regression, quantitative epidemiology.

Phymatotrichum omnivorum (Shear) Duggar (*Phymatotrichopsis omnivora* (Duggar) Hennebert) is a soilborne pathogen of more than 2,000 dicotyledonous plants in the southwestern United States and Mexico. The fungus survives as sclerotia and strands and is capable of growth through soil as strands (3,18). The spread of Phymatotrichum root rot within a field over several seasons has been studied extensively (7,13,14). McNamara and Hooton (14) kept detailed records of the location and distribution of cotton root rot in several fields over an 8-yr period. They concluded that *P. omnivorum* spreads radially (13), the rate of spread between rows being the same as within rows (14). These conclusions refer to annual rates of spread, however, as they were based on the observed pattern of the disease at one assessment per season over several years. Since these early studies, little has been done to study the spatial increase of Phymatotrichum root rot in relation to row geometry.

There is little doubt that the fungus spreads radially in regularly spaced crops such as alfalfa (6), and in these crops, the rate of spread appears to be similar in all directions. Whether this is the case in row crops such as cotton, where the distance between rows is greater than the distance between plants within rows, has not been tested experimentally. Observations in field plots of cotton and soybean indicated that within-row spread was a major component of disease increase (4,5). The fungus has been observed to move over distances of 3.2 m under experimental conditions (7,8), but there may be a limit on the distance it can move from diseased to healthy plants under field conditions. There is also controversy over whether the fungus grows through soil ahead of diseased plants or whether growth directly from diseased to healthy roots is the primary means of disease increase (7,19).

The purpose of this study was to determine whether the spacing between plants in plots had any effects on disease progress of Phymatotrichum root rot; this was done by varying the within-row plant density and the between-row spacings. The progress of Phymatotrichum root rot was monitored at frequent intervals in the plots throughout the season; analyses of disease progress curves were made as a basis for treatment comparison.

MATERIALS AND METHODS

Field experiment. A 0.4-ha field (83 × 47 m) at the Blackland Research Center, Temple, TX, was planted with cotton (*Gossypium hirsutum* L. cv. GP3774) in rows spaced 69 cm apart on 15 April 1985. Two weeks after planting, the field was divided into four equal blocks, which were each divided into eight treatment plots. Each plot originally contained 10 8-m rows. Adjacent plots were isolated by 3-m alleys, which were kept free of weeds and host plants by cultivating throughout the growing season. The soil type at this site is classified as a Houston black clay (Udic Pellusterts [fine, montmorrillonitic, thermic]). The field was naturally infested with *P. omnivorum* and had been planted with cotton in previous years. Disease incidence (proportion of plants killed by *P. omnivorum*) had exceeded 90% in years with favorable environmental conditions.

Eight treatment combinations consisted of two between-row spacings of 69 cm (10 rows per plot) and 138 cm (five rows per plot) and four within-row plant densities of 1, 5, 9, and 13 plants per meter. The experimental design was a 2 × 4 factorial, completely randomized block design. Row spacings were achieved by roguing alternate rows, postemergence, for the 138-cm between-row spacing treatment and retaining all 10 rows for the 69-cm treatment. Within-row densities were achieved by marking off eight contiguous 1-m lengths in each row with wood stakes and hand thinning each meter to the desired number of plants per meter. Although most analyses were based on the intended within-row densities treatment levels, the actual densities at the time of the first assessment ranged 0.9–1.0, 4.6–4.9, 7.7–8.6, and 10.7–12.4 plants per meter for the 1, 5, 9, and 13 plants per meter treatments, respectively.

Plots were monitored at least twice a week from emergence until the first symptoms of Phymatotrichum root rot were observed, and assessments made on 18 June. Disease assessments were then made on 22, 25, and 28 June; 2, 6, 10, 13, 17, 26, and 29 July; and 2, 9, 12,

and 20 August. Plants were considered diseased if they showed the typical first symptoms of Phymatotrichum root rot: wilting and a slight bronzing of the upper leaves followed by wilting of the whole plant and rapid death. All plants in each plot were assessed, and the location of each diseased plant within-rows was recorded. Disease incidence on each assessment date was calculated as the total number of dead plants in a plot divided by the total number of plants in that plot at the first assessment. Soil cores (about 8 cm diameter × 60 cm) were taken on 5 August at positions within and between rows with a tractor-mounted hydraulic rig, taken to the laboratory, wet-sieved, and the total length of root in the core estimated using a modified line intersect method (20).

Statistical analyses. Analyses were made to test several hypotheses concerning the progress of Phymatotrichum root rot. Ordinary runs analysis was used to test the hypothesis of a random pattern of initially diseased plants in a plot using the Z statistic (12). Repeated-measures analysis of variance was used to test the hypotheses that between-row spacing and within-row spacing treatments had no effect on disease progress. The analysis was implemented with the Statistical Analysis System (SAS) GLM procedure (17). The procedure uses a multivariate approach and analyzes disease incidence at each time as a set of random variables (11) with respect to between-row spacing, within-row density, and their interaction as the treatment factors. The proportion of diseased plants (y) was transformed to logits, where logit $y = \ln(y/(1-y))$ to stabilize the variance and provide equal precision at both extremes of the proportion scale. Orthogonal polynomial contrasts were formed to test for polynomial trends over time for the treatment effects.

A thorough comparison of the epidemics that developed in each plot was made by modeling disease progress in each plot by using the logistic equation

$$y(t) = M/(1 + [(M/y_0) - 1] \exp(-rt)), \quad (1)$$

where $y(t)$ was the proportion of diseased plants at time t, M was the asymptotic disease level, and r was the rate of disease increase for the logistic model. The logistic model was fitted with nonlinear regression by using the SAS NLIN procedure (17). To fit equation 1 to the data from each plot, initial and final disease levels were used as starting values for y_0 and M, respectively; r values estimated from linear regressions of logit y against time were used as starting values for the rate of disease increase in each plot.

Four methods of fitting nonlinear regression models by the method of least squares are available with the NLIN procedure. Each requires good initial parameter estimates. In a comparison of these methods using disease progress data and equation 1, the multivariate secant method consistently provided smaller error variances, was less sensitive to starting parameter values than the other methods, and therefore was used throughout this study.

The disease progress data were fitted to equation 1 in two stages. First, the model was fitted to the data specific to each plot; i.e., each replication of each treatment, giving 32 sets of the logistic parameters y_0, r, and M. From these parameters, the area under the disease progress curve (Z) was calculated by first integrating equation 1 to give

$$Z = M \left\{ t + \ln(1 + [(M/y_0) - 1] \exp(-rt))/r \right\} \quad (2)$$

and substituting the estimated logistic parameters. The set of four random variables, y_0, r, M, and Z, was analyzed for treatment effects using multivariate analysis of variance (MANOVA) as implemented on SAS (17).

To test the overall adequacy of the logistic model, equation 1 was also fitted to the mean data for each treatment. The purpose of fitting the model in this way was to perform a lack-of-fit F test (16) to determine the adequacy of the logistic model with respect to each treatment. The model is rejected for $F(\text{lack-of-fit}) > F_\alpha[a-p, a(n-1)]$, where α is the significance level, a is the levels of time, n is the number of replicates, and p is the number of parameters estimated for the logistic model.

Finally, each of the four parameter estimates was regressed against within-row density and plot density, which is the total number of plants in a plot divided by the area of the plot. The initial disease incidence (y_0) was transformed to $\ln(y_0)$ and regressed against plot density and within-row density. Rate of disease increase (r) and area under the curve (Z) were weighted according to the inverse of the variances of the four replicates. M was regressed against plot density and within-row density by fitting the model $M = M_{max}[1 - \exp(-bd)]$, constraining $M = 0$ when $d = 0$, using the NLIN procedure, where M_{max} is maximum disease asymptote, d is density, and b is a constant coefficient. Except for M, no attempt was made to constrain the parameters to 0, at $d = 0$.

RESULTS

The disease progress data for each treatment are plotted in Figure 1. Each graph contains the data points from four replication plots at each assessment for each treatment; the fitted line is for the mean data and will be discussed below. The first assessment was made on 18 June (day-of-year 169) and the last was made on 20 August (day-of-year 232). In five of the 32 plots, the hypothesis of random pattern was rejected ($P = 0.05$) when symptoms were first observed, indicating an initial clustering of diseased plants in these five plots. These plots were: two plots of 13 plants per meter × 10 rows per plot (33 and 35 diseased plants), one plot of 13 plants per meter × five rows per plot (13 diseased plants), and two plots of nine plants per meter × five rows per plot (eight and 15 diseased plants). In all other plots, it was not possible to reject the hypothesis of random pattern. Analysis of variance of the root density data showed higher density within rows than between rows ($P<0.05$), higher root densities in plots with 10 rows than with five rows per plot ($P<0.01$) but no differences between the 5, 9, and 13 plants per meter treatments as main effects. When sampled within rows, however, there was a direct relationship between root density and within-row density ($P<0.05$). The range in root density was 248 cm/480 cm^3 of soil in the lower density plots to 642 cm/480 cm^3 of soil in the highest, an increase something less than threefold.

Repeated measures analysis of disease progress. The univariate tests for treatment effects on logit-transformed disease incidence showed significant within-row density, time, and within-row density × time effects on disease incidence (Table 1). There was no effect of between-row spacing or of any interaction with between-row spacing. Thus, although most of the variation in disease was accounted for by time, as expected, a significant proportion of variation was also accounted for by within-row density. Also, the effect of time was dependent on within-row density as indicated by the significant interaction. Orthogonal polynomial contrasts for time were constructed and the first four terms tested for significance. Contrasts were analyzed for each source of variation (Table 2). There were significant linear, quadratic, and quartic trends for the mean of all treatments as well as significant quadratic and cubic effects of within-row density. Between-row spacing and

TABLE 1. Repeated measures ANOVA univariate tests for treatment effects on logit-transformed disease incidence for Phymatotrichum root rot of cotton assessed in plot density experiments at Temple, TX

Source of variation	Degrees of freedom	Mean square	P[a]
Between treatments			
BRS[b]	1	5.431	0.4700
WRD[c]	3	68.164	0.0020
BRS × WRD	3	10.338	0.4100
Error	24	10.338	
Within treatments			
Time	13	185.885	0.0001
Time × BRS	13	0.368	0.9400
Time × WRD	39	3.349	0.0001
Time × BRS × WRD	39	0.588	0.8700
Error	312	0.795	

[a] Probability of a greater F value if there was no treatment effect.
[b] Between-row spacing.
[c] Within-row density.

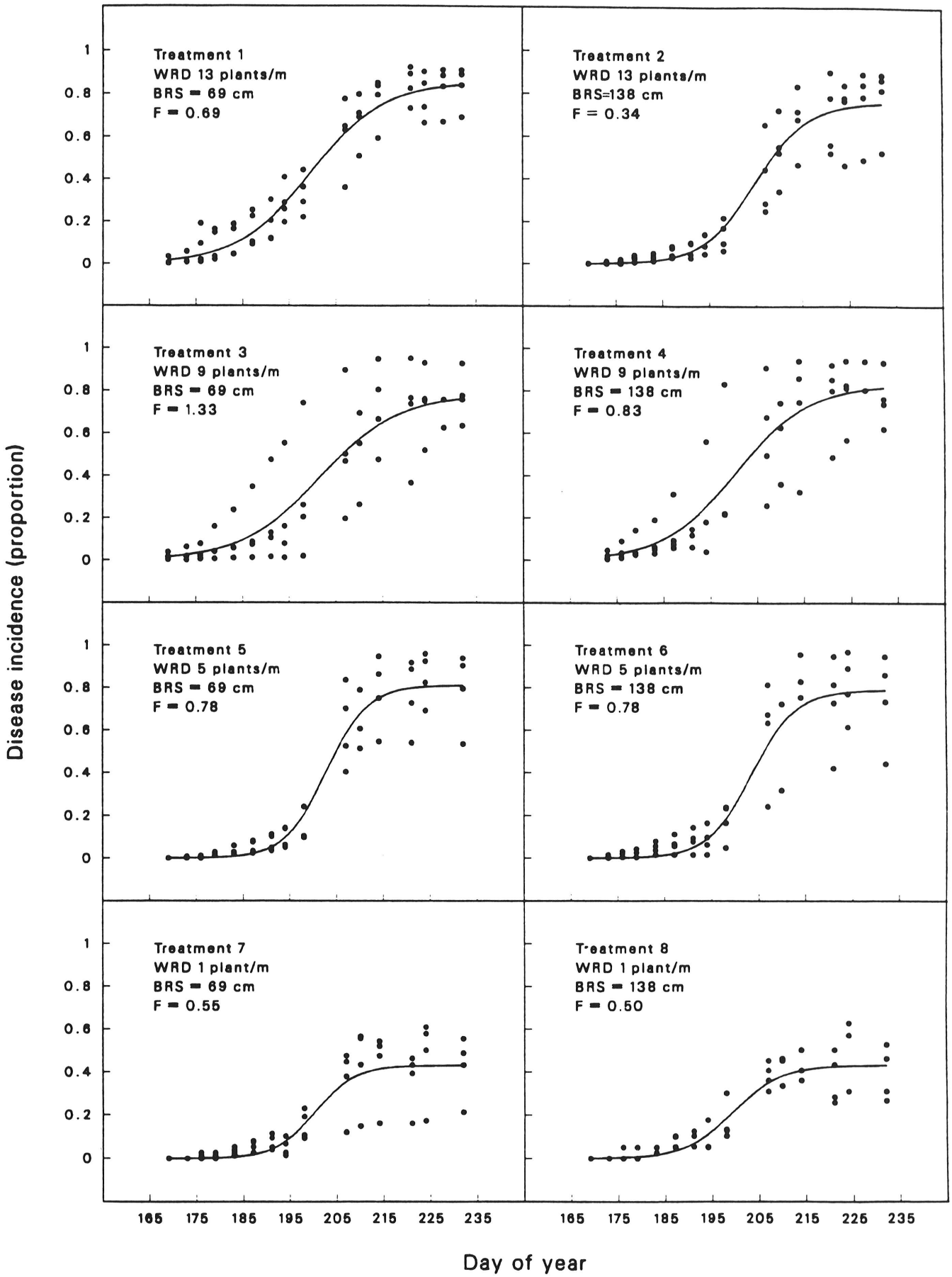

Fig. 1. Effects of within-row plant density (WRD) and between-row spacing (BRS) on progress of Phymatotrichum root rot in cotton. Line was fitted to the replicate means using a logistic model with nonlinear least squares regression. *F* is the lack-of-fit test statistic with 13 numerator and 48 denominator degrees of freedom. Treatment numbers are for text reference.

TABLE 2. Repeated measures ANOVA tests for orthogonal polynomial contrasts over time on logit-transformed disease incidence for Phymatotrichum root rot of cotton assessed in plot density experiments at Temple, TX

Source of variation	Degrees of freedom	Linear		Quadratic		Cubic		Quartic	
		Mean square	P[a]	Mean square	P	Mean square	P	Mean square	P
Mean[b]	1	2,209.460	0.0001	170.969	0.0001	0.023	0.8600	11.664	0.0007
BRS[c]	1	1.024	0.6000	0.408	0.6300	0.009	0.9100	0.252	0.5700
WRD[d]	3	8.417	0.1000	19.511	0.0001	9.262	0.0001	0.528	0.5700
BRS × WRD	3	2.815	0.5200	0.661	0.7700	0.158	0.8800	0.336	0.7300
Error	24	3.706		1.743		0.711		0.769	

[a] Probability of a greater F value if there was no treatment effect.
[b] Mean over all treatments.
[c] Between-row spacing.
[d] Within-row density.

its interaction with within-row density gave no significant polynomial trends. The lack of significance for the linear component for within-row density (with logit-transformed data) indicated no difference in rate of disease increase for the treatments.

Nonlinear regression of disease progress. To reduce the bias created by assuming 100% final disease incidence, the logistic model was fitted to untransformed Phymatotrichum root rot incidence by nonlinear least squares analysis. Analyses were first performed for each treatment using the four replicates and then for the treatment means. These sums of squares were used in the lack-of-fit F test. The model was fitted to the absolute counts of diseased plants (data not shown), but the rate parameter did not differ from those obtained by fitting proportionate data. The fitted lines in Figure 1 are for the treatment means. According to the lack-of-fit test, the logistic model of disease progress could not be rejected for any of the treatments. After the appropriateness of the logistic model was tested, each plot was fitted separately and the set of parameters estimated; i.e., initial disease incidence (y_0), rate of disease increase (r), and maximum disease incidence (M). From these estimates, areas under the disease progress curves were computed for each plot using equation 2 by solving for Z at $t = 63$ days. Table 3 shows the means, MANOVA standard errors, and least significant differences for these parameters. Table 4 shows the Pearson product-moment correlation coefficients calculated for each pair of parameters, y_0, r, M, Z, and plot density. There was a negative correlation between y_0 and r. Asymptotic disease incidence and area under the curve were lower for the low within-row density treatments (Table 3). Final disease levels were independent of y_0 or r, according to linear correlation, but were strongly correlated with plot density. Z was strongly correlated with M and to a lesser extent with y_0. All parameters were correlated with plot density; the rate was apparently negatively correlated.

The apparent negative relationship of the rate to other parameters can be seen from Figure 1. Considering the number of days to about 20% disease, the higher within-row density treatments, 1–4, had shorter times to this amount of disease and high asymptotes. By contrast, plants in treatments 5 and 6 took the longest time to reach 20% disease incidence but still had high asymptotes. The delay early in the season and the high final disease gave higher estimated rates of disease increase. Regardless of rate of disease increase for plants in treatments 7 and 8, the asymptotic level of disease remained low.

Rate of disease increase (r), ln y_0, M, and Z were each regressed against plot density and within-row density. Linear regression models were used for r, ln y_0, and Z. The regressions for r and Z were weighted according to the inverse of their variances. A nonlinear regression model was used for M. The equations for the fitted curves are presented along with the original data and the fitted lines in Figure 2. The transformation of y_0 gave a satisfactory linearization of the relationship with plot density and within-row density. The nonlinear equation describing the relationship between M and plot density in Figure 2 indicates a plot density of one or two plants per square meter as the effective plant spacing that inhibits increase of Phymatotrichum root rot; a value above one or two plants per square meter had no effect on the final level of disease. Although the equation for r was significant, the linear relationship between r and plot density was rejected (and thus the negative slope) based on a marked increase in the variances and nonrandom residual plots. No transformation of r was found to provide a satisfactory linear relationship with plot density. Area under the curve was linearly related to plot density and within-row density for values greater than one or two plants per square meter and one plant per meter, respectively, and increased with increases in these two variables. Although there was a high level of variation, the linear relationship was acceptable and provided the most economical description of the relationship of Phymatotrochum root rot increase with plot density.

TABLE 3. Effects of within-row density and between-row spacing on the nonlinear least squares estimates of initial disease incidence (y_0), rate of disease increase (r), and asymptotic disease incidence (M) and the computed area under the disease progress curve (Z) for incidence of Phymatotrichum root rot of cotton assessed in plot density experiments at Temple, TX

Treatment	WRD[a]	BRS[b]	Estimated parameters[c]			
			y_0	r	M	Z
1	13	69	0.0250	0.14	0.86	27.8
2	13	138	0.0020	0.19	0.76	20.5
3	9	69	0.0100	0.16	0.79	24.4
4	9	138	0.0080	0.17	0.83	25.9
5	5	69	0.0003	0.25	0.81	27.7
6	5	138	0.0003	0.25	0.78	22.9
7	1	69	0.0030	0.22	0.44	13.8
8	1	138	0.0020	0.23	0.42	13.9
SED[d]			0.0080	0.04	0.10	1.8
LSD[e]			0.0170	0.08	0.21	3.7

[a] Number of plants per meter (within-row density).
[b] Distance in centimeters between adjacent rows (between-row spacing).
[c] Each estimate represents the mean of four replicates.
[d] Standard error of the difference between any two means within a column.
[e] Least significant difference ($P = 0.05$, 24 degrees of freedom) between any two means within a column.

TABLE 4. Pearson correlation coefficient matrix for: the logistic parameters rate of disease increase (r), initial disease incidence (y_0), and asymptotic disease incidence (M) and area under the disease progress curve (Z) and plot density (PD)

	r	y_0	M	Z	PD
r	1.00	−0.58**[a]	−0.14	−0.09	−0.48*
y_0	···	1.00	0.33	0.48*	0.56**
M	···	···	1.00	0.89**	0.59**
Z	···	···	···	1.00	0.48*
PD	···	···	···	···	1.00

[a] Probability levels are * = $P = 0.01$ and ** = $P = 0.001$.

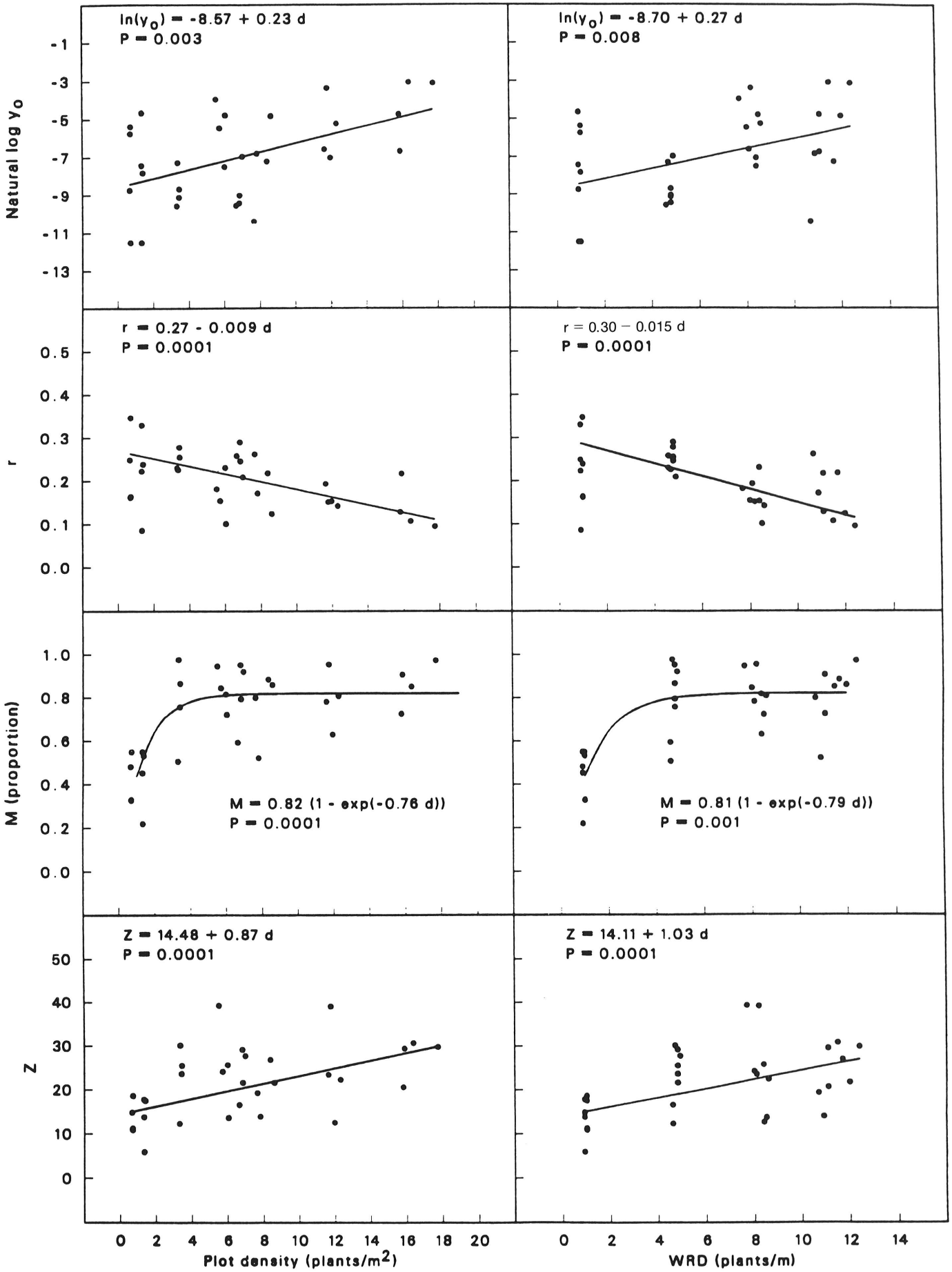

Fig. 2. Effects of plant density and within-row density (WRD) on the logistic parameters y_0 (initial disease incidence), r (rate of disease increase), M (asymptotic disease incidence), and Z (area under disease progress curve).

DISCUSSION

The disease cycle of *P. omnivorum*, as it relates to spread of Phymatotrichum root rot, can be divided into two parts: primary infection, which is the result of root contact with overwintering strands or sclerotia, and plant-to-plant spread from initial foci by root contact with strands or mycelium from diseased plants or by growth of the fungus through soil. In the present study, initial disease incidence decreased logarithmically as plant density decreased. The reduction in initial disease incidence was manifested as lower levels of disease early in the season in the within-row density treatments with one and five plants per meter and was expressed further by the longer time to reach 20% disease in these plots. Regular monitoring of the plots had been undertaken before symptoms first were observed, and it is reasonably certain that no plant-to-plant spread had occurred by 18 June. The small number of plots in which nonrandom patterns of diseased plants were observed indicated that in some plots, at least, there was a clustered pattern of overwintering structures. These plots were at higher plant densities, but there was no discernible difference between disease progress in plots with random or nonrandom patterns of initially diseased plants within a treatment. Although primary infection may occur throughout the season, once foci are established, plant-to-plant spread appears to be responsible for increase of Phymatotrichum root rot and final levels of disease (4,5).

Root contact is not necessary for plant-to-plant spread (9) as was once thought. Strands have been observed to grow up to 3.2 m under sterile conditions in the presence of dead cotton roots in moist sand (8). Also, from crude field observations on excavated cotton plants (15), it was estimated that Phymatotrichum root rot may extend at least 1.1 m beyond the last dead plant of a disease front. We found that the progress of Phymatotrichum root rot was limited in plots with a within-row density of one plant per meter, an average of 100 cm between plants within a row. A within-row density of five plants per meter, an average of 20 cm between plants within a row, did not limit disease progress, even though there was an initial delay early in the season. Because 100-cm spacing between plants limited Phymatotrichum root rot and there was no difference between the between-row spacing treatments of 138 and 69 cm, the minimum distance that did limit Phymatotrichum root rot in this study was 69 cm. We concluded, therefore, that the distance between adjacent plants in a field that inhibited increase of Phymatotrichum root rot by plant-to-plant spread was between 20 and 69 cm. This distance will be dependent on environment, edaphic factors, and cultivar and thus will vary with season and location.

The asymptotic disease incidence estimated from the logistic model was most useful when analyzing the effect of plant density on increase of Phymatotrichum root rot. Asymptotic disease values can be explained in terms of plant-to-plant spread. High plant densities facilitate plant-to-plant spread by increasing the probability of pathogen-host encounter and lead to greater levels of Phymatotrichum root rot. It should be noted, however, that root densities did not vary to the same extent as the plant densities imposed in this study. We found by visual interpolation of Figure 2, that plant densities greater than one or two plants per square meter did little to further increase M. At densities of less than this, values of M were much reduced. Burdon and Chilvers (2) observed a similar leveling-off in incidence of damping-off caused by *Pythium irregulare* with increasing densities of garden cress.

We could find no acceptable linear relationship between r and plant density in this study. Because the rate depends on y_0 and M, and because these two parameters were not correlated in this study, each unique combination of y_0 and M gave a unique r, thus confounding trends in r with respect to plant density. For example, when y_0 was low and M was high, as in treatments 5 and 6, r was high even though disease increase was slow at first. With high y_0 and M, as in treatments 1–4, r values were lower because of high early disease levels. Treatments 7 and 8 had low y_0 and low M with intermediate r values. A more flexible growth model such as the Richards or Weibull models (10) may account for the more pronounced curvature in disease progress for treatments 5 and 6, but as the logistic model could not be rejected as an adequate model, there was little justification for adding additional parameters when considering all eight treatments.

Plant density is a valuable and often overlooked variable when studying plant disease epidemics (1,2). The purpose of this study was to manipulate plant density to study the increase of Phymatotrichum root rot within a field population. In this experiment, we found that a distance of 20–69 cm between plants limited increase of Phymatotrichum root rot. Furthermore, early in the growing season, disease progress (to about 20% incidence) was inhibited when plants were spaced 20 cm apart. Our knowledge of the epidemiology of Phymatotrichum root rot is still incomplete, and density studies can provide some of the necessary information to fill in the gaps.

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