Macrophomina phaseolina Infection and Vine Decline in Cantaloupe in Relation to Planting Date, Soil Environment, and Plant Maturation

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ABSTRACT

Bruton, B. D., Jeger, M. J., and Reuveni, R. 1987. *Macrophomina phaseolina* infection and vine decline in cantaloupe in relation to planting date, soil environment, and plant maturation. Plant Disease 71: 259-263.

The percentage of root systems of cantaloupe infected by *Macrophomina phaseolina* increased sigmoidly from the time of planting in eight experiments over three calendar years. There were no significant relationships between derived soil temperature and infection variates, although high temperatures consistently inhibited infection. At about flowering, the percentage of root systems infected was directly related to soil moisture content, whereas this effect was reversed after fruit set. Vine decline was directly related to soil matric potential before flowering. More than 80% of root systems were infected with *M. phaseolina* 49 days after planting, but symptoms in the crown area did not develop until after 85–90 days, indicating the importance of latent, especially early, root infection in vine decline.

Additional key words: *Cucumis melo*, epidemiology

Macrophomina phaseolina (Tassi) Goid. incites disease in a wide range of hosts, especially under conditions of high temperature and drought stress. In many cases, disease develops in plants with poor vigor or at the flowering and seed development stages (4). There is some ambiguity in the literature concerning the responses of various host plants to *M. phaseolina* in different environmental conditions of temperature and soil moisture.

Reuveni et al (20) reported infection to be more prevalent in summer- than in winter-grown cantaloupe in Israel. Cook (3) found disease severity in castor beans to be greatest where warm and moist springs were followed by extended hot and dry weather. Meyer et al (15) reported seedling disease of soybean to be greatest at 30 and 35 C, although some infection occurred at 20 and 25 C. Maximum damage in soybean occurred at temperatures between 35 and 40 C. Charcoal rot of soybean in India occurred at air temperatures between 35 and 40 C (1).

Soil moisture conditions often have been found to mitigate the effects of high temperature. Edmunds (5) demonstrated that charcoal rot in sorghum was severe in plants inoculated near maturity at temperatures of 35–40 C and 25% available soil moisture. At 80% available soil moisture, no infection occurred. Norton and Frank (16) found that charcoal rot in guayule was readily controlled by irrigation despite high temperatures. Cotton plants stressed by drought developed severe charcoal rot, whereas those receiving adequate water were less affected at soil temperatures ranging from 20 to 40 C (7). Conditions of low soil moisture leading to drought stress have been reported as more important than high temperatures in predisposing plants to charcoal rot (7,8,12,20,23). Some investigators, however, have reported a higher incidence of charcoal rot in various hosts grown under conditions of high soil moisture (13,14,19,22).

Plant juvenility may be a factor in the epidemiology of *M. phaseolina*; susceptibility of field beans to infection was greatest at preemergence and during the seedling stage and declined with age (11). Other workers, however, have found no relationship between plant maturation and susceptibility (1,25).

The purpose of this study was to quantify the infection of root systems by *M. phaseolina* under different environmental conditions and to evaluate the relationship with subsequent symptom expression, as vine decline, in cantaloupe (*Cucumis melo* L.). Throughout the study, we were concerned to distinguish adequately between infection and symptom development. Planting date, soil temperature and moisture, and plant maturation were evaluated as factors influencing infection by *M. phaseolina* and the subsequent development of vine decline.

MATERIALS AND METHODS

Field husbandry and soil environment. The cantaloupe cultivar Perlita was planted at Weslaco, TX, on each of eight dates covering 3 yr: 28 September 1982; 3 March, 11 March, 21 April, and 3 May 1983; and 17 February, 2 March, and 16 March 1984. At least five soil samples per replicate were taken in each experiment (before pesticide application) to estimate initial inoculum density of *M. phaseolina* and determine soil type. The soil samples were processed by the method of Papavizas and Klag (17) to determine inoculum density of *M. phaseolina* and by the method of Estiri (6) to determine soil type. The different plots (four replicates of four rows, each 20 m long) were pretreated with nematicide and herbicide before planting and furrow-irrigated after planting. Soil temperature

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Accepted for publication 19 July 1986.

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was recorded continuously in all eight experiments at depths of 7.5, 15, and 30 cm with a three-point thermograph (Qualimetrics, Inc., Sacramento, CA). Soil samples at a depth of 0–20 cm were taken at 7-day intervals and oven-dried to determine soil moisture content in five of the experiments. Moisture content (%) was converted to matric potential using release curves developed for each experimental plot.

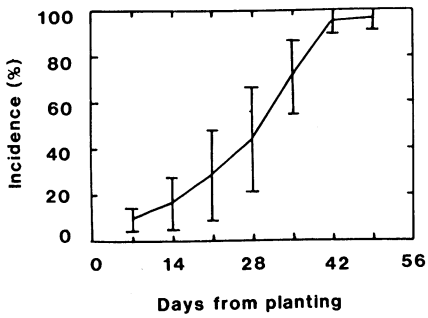

Fig. 1. Percentage of root systems of cantaloupe infected with *Macrophomina phaseolina* with time. Values plotted correspond to the mean of eight planting dates. Vertical bars represent 95% confidence limits.

Assessment of root infection and vine decline. The percentage of root systems infected with *M. phaseolina* (incidence) was determined at regular intervals after planting. At least 10 plants were excavated at random from each of the four replicate plots at 7-day intervals from planting to 49 days; each plot contained about 525 plants. On each occasion, a 20-cm-diameter area of soil was excavated to a depth of about 20 cm. Every effort was made to avoid disturbing neighboring plants and to retain root systems as intact as possible. After the 17 February 1984 experiment, assessments were not made until day 35. After the 21 April 1983 experiment, assessments were not made after day 35. In each experiment, the tops of the plants were removed above the crown and discarded. Each root system was rinsed in tap water to remove adhering soil, surface-disinfested in 0.5% sodium hypochlorite for 1 min, placed on potato-dextrose agar (Difco) plus streptomycin sulfate (0.1 g/L), and incubated in darkness for 3–4 days at 30 C. Several dishes were used, as required, to accommodate the larger root systems and observed for developing colonies of *M. phaseolina*. The number of root systems that produced at least one *M. phaseolina* colony subsequently was recorded. No attempt was made to record the number of infection sites on roots.

Ultimate development of vine decline in the field was recorded at harvest on the basis of the percentage of plants showing symptom development in the crown area. Plants did not mature to harvest after the 28 September 1982 planting date and were not rated for vine decline.

Data analysis. The following disease variates were derived from the weekly data on incidence (%) of cantaloupe roots infected by *M. phaseolina* and development of vine decline. A minimum of 10 plants were excavated from each of four replicate plots at each sampling occasion (y = proportion of root systems infected):

1. Rate parameter: linear regression of $\ln[y/(1-y)]$ against time (all data) (not assessed for 17 February 1984 planting date).
2. Rate parameter: linear regression of $\ln[1/(1-y)]$ against time (excluding first assessment).
3. Number of days for incidence (percentage of infected root systems) to reach 5% (not assessed for 17 February 1984 planting date).
4. Number of days for incidence (percentage of infected root systems) to reach 50% (not assessed for 17 February 1984 planting date).
5. Number of days for incidence (percentage of infected root systems) to reach 80%.
6. Incidence of infected root systems at 21 days (flowering) (not assessed for 17 February 1984 planting date).
7. Incidence of infected root systems at 35 days (fruit set).
8. Incidence of infected root systems at 49 days (last assessment) (not assessed for 21 April 1983 planting date).
9. Area under the curve representing the percentage of infected root systems with time (0–49 days) (not assessed for 17 February 1983 planting date).
10. Percentage of plants showing symptoms as vine decline at harvest (plants did not mature to harvest for 28 September 1982 planting date).

The rate parameters were calculated by linear regression of transformed incidence against time. The number of days for incidence (percentage of root systems infected) to reach 5, 50, and 80% in each experiment was estimated by linear interpolation of the mean observed values for incidence. The assessments for days 21 and 35 corresponded approximately to the average lengths of time to flowering and fruit set, respectively. The area under the curve of percentage of infected root systems was calculated numerically from days 0–49 using the trapezoidal rule.

Table 1. Initial microsclerotial population of *Macrophomina phaseolina*, percentage of plants with infected root systems at 49 days, and percentage of plants developing decline at harvest in eight experiments over 3 yr

Planting date	Initial microsclerotial population/g dry soil	Plants with infected root systems (%)	Plants developing decline (%)
28 September 1982	2.8 (0.3)[a]	94	–[b]
3 March 1983	7.4 (1.2)	93	45
11 March 1983	3.4 (0.2)	83	68
21 April 1983	18.2 (1.0)	80[c]	43
3 May 1983	3.5 (0.3)	79	57
17 February 1984	11.5 (1.6)	95	69
2 March 1984	12.9 (2.2)	95	79
16 March 1984	8.8 (1.5)	95	71

[a] Standard errors of mean values are shown in parentheses.
[b] Plants did not mature to harvest.
[c] Assessment at 35 days.

Table 2. Correlation matrix for disease variates[a,b]

	1	2	3	4	5	6	7	8	9	10
1	1.00	···	···	···	···	···	···	···	···	···
2	*0.89*	1.00	···	···	···	···	···	···	···	···
3	−0.55	*−0.75*	1.00	···	···	···	···	···	···	···
4	*−0.90*	*−0.67*	0.52	1.00	···	···	···	···	···	···
5	*−0.94*	*−0.92*	*0.71*	*−0.86*	1.00	···	···	···	···	···
6	*0.76*	0.57	−0.60	*−0.92*	*0.83*	1.00	···	···	···	···
7	*0.93*	*0.87*	*−0.77*	*−0.91*	*−0.91*	*0.83*	1.00	···	···	···
8	*0.85*	*0.93*	−0.61	−0.64	−0.52	0.58	0.60	1.00	···	···
9	*0.93*	*0.85*	*−0.74*	*−0.94*	*−0.95*	*0.89*	*0.98*	*0.74*	1.00	···
10	0.25	0.49	*−0.81*	−0.20	−0.47	0.32	0.34	0.12	0.40	1.00

[a] |Values| for significance of correlation coefficients are: 0.67 ($P = 0.05$), 0.80 ($P = 0.01$), and 0.90 ($P = 0.001$); those significant at $P < 0.05$ are shown in italics.
[b] Disease variates: 1 = rate parameter: linear regression of $\ln[y/(1-y)]$ against time and 2 = rate parameter: linear regression of $\ln[1/(1-y)]$ against time (y = proportion of root systems infected by *Macrophomina phaseolina*); 3–5 = number of days for incidence to reach 5, 50, and 80%, respectively (estimated by linear interpolation between observed mean values for incidence); 6–8 = incidence of infected root systems at 21 days (flowering), 35 days (fruit set), and 49 days (last assessment), respectively; 9 = area under infection dynamics curve (0–49 days); and 10 = percentage of plants developing vine decline (at harvest). (Plants did not mature to harvest for the 28 September 1982 planting date.) Variates 1–4, 6, and 9 not assessed for 17 February 1984 planting date, and variate 8 not assessed for 21 April 1983 planting date.

Soil environment variates were derived from the records of soil temperature and moisture. Variates were constructed to give disjoint and overlapping time periods; apart from the first 7 days, these were based on 14-day intervals. Variates analyzed were mean, maximum, minimum, and range (maximum minus minimum) for 1) soil temperature at the 7.5-cm depth, 2) moisture content, and 3) matric potential. These were calculated for each period and, in the case of temperature, as the mean of daily values. The correlation coefficient between pairs of infection and environmental variates was calculated subject to the environmental variate preceding or referring, approximately, to the same time period as the infection variate. Selection of variates for analysis by regression was made on the basis of significant correlation ($P < 0.05$). Because of the small number of degrees of freedom, only linear regressions were attempted except where stated.

RESULTS

Root infection. Curves representing the incidence of infected root systems (percentage of root systems in which *M. phaseolina* was isolated) were sigmoid for each of the eight experiments. The incidence of root infection on the last assessment date, 49 days after planting, was near 80% or higher in each experiment. The mean percentage of plants with infected roots, and 95% confidence limits, over the eight planting dates are plotted in Figure 1. Table 1 gives, by planting date, the initial microsclerotial population per gram of dry soil, the incidence (%) of infected root systems after 49 days, and the percentage of plants ultimately developing vine decline. The correlation matrix (Table 2) between the infection variates demonstrated a high level of intercorrelation. The percentage of plants that ultimately developed decline, however, was significantly related ($P < 0.01$), negatively, only to the time for incidence to reach 5% (Fig. 2). There was no significant correlation ($P = 0.05$) between an infection variate and planting date (counted as Julian days) or initial microsclerotial population, although at $P = 0.10$, the percentage of infected root systems at 21 days was positively correlated with microsclerotial density.

Soil temperature. There was no significant correlation (at $P = 0.05$) between an infection and soil temperature variate. There were many correlations significant at $P = 0.10$, however, and these consistently indicated a negative relationship between infection and temperature during most of the 49-day period. There was no significant correlation between an infection variate and the number of hours soil temperature was in the range 25–35 C. The number of days for the percentage of infected root systems to reach 5% increased with the slope of the regression line giving daily maximum temperature as a function of time (Fig. 3). Rapidly increasing soil temperatures inhibited infection by *M. phaseolina*, although the linear relationship was highly dependent on the negative coefficient for the September planting. At higher rates of temperature increase, there did appear to be a reduction in the time to reach 5% incidence, but including a quadratic term in the regression did not significantly account for more of the variance.

Soil water status. *Soil moisture content.* There were direct relationships between the percentage of infected root systems at day 21 and soil moisture content during the first few weeks after planting, although this was only significant ($P < 0.05$) for the moisture range during the period 0–35 days (Fig. 4). Given the variation in flowering date, this relationship suggests that the greater the difference between maximum and minimum moisture content during the early period of root growth, the more root infection at about flowering. The influence of soil moisture content was reversed, however, when considering periods after fruit set. The number of days for the incidence of infected root systems to reach 80% was directly related to maximum moisture ($P < 0.01$) and moisture range ($P < 0.05$) during the period 36–49 days after planting (Fig. 5). At high soil moisture contents, and for greater differences between maximum and minimum moisture content during this period, a longer time was taken to reach 80% infection. The area under the incidence curve was negatively related to maximum soil moisture ($P < 0.05$) and moisture range ($P < 0.01$) during the same period (Fig. 6), again indicating the inhibitory effect of higher moisture content on late infection of roots. Similarly, the rate of root infection, calculated using the $\ln[y/(1-y)]$ transformation, was negatively related to the moisture range ($P < 0.01$) in the period 35–49 days (Fig. 6). Thus, although higher soil moisture content favored root infection before flowering, this influence was reversed after fruit set.

Matric potential. The percentage of plants that developed vine decline was directly related to the mean and minimum matric potential (both $P < 0.05$) during the period 0–21 days (Fig. 7). Higher matric potentials (i.e., wetter conditions) during the early period of root growth increased the percentage of plants developing decline 60–85 days later. The number of days for the percentage of infected root systems to

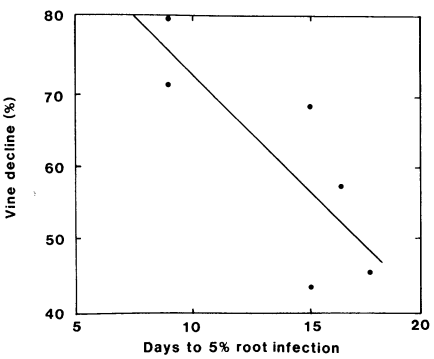

Fig. 2. Incidence of vine decline (%) in relation to the number of days for incidence of infected roots to reach 5%. Line represents the regression equation: $y = 104.7 - 3.2x$ ($R^2 = 0.64$).

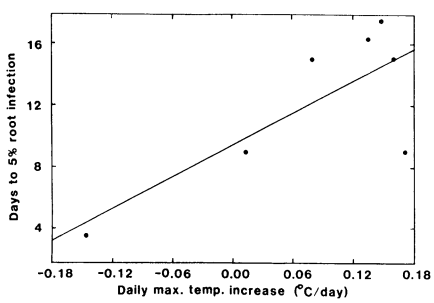

Fig. 3. Days for incidence of infected roots to reach 5% in relation to the rate of maximum temperature increase (degrees [C] per day). Line represents the regression equation: $y = 10.5 + 29.5x$ ($R^2 = 0.62$).

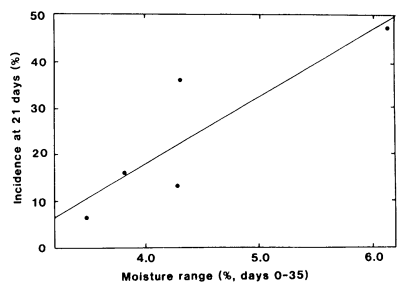

Fig. 4. Incidence of infected roots (%) at 21 days in relation to soil moisture range in the period 0–35 days after planting. Line represents the regression equation: $y = -39.2 + 14.3x$ ($R^2 = 0.76$).

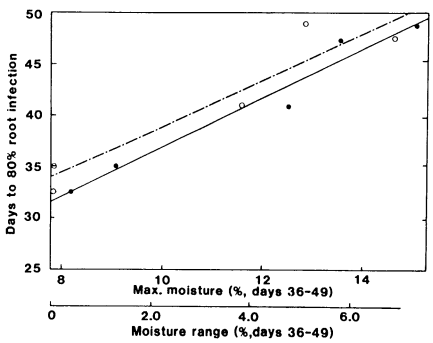

Fig. 5. Days for incidence of infected roots to reach 80% in relation to 1) maximum soil moisture (•) and 2) moisture range (o) during the period 36–49 days after planting. Lines represent the regression equations: 1) $y = 12.6 + 2.4x$ ($R^2 = 0.96$) (———) and 2) $y = 33.9 + 2.3x$ ($R^2 = 0.90$) (—·—).

reach 5% (the one infection variate significantly correlated with vine decline) was negatively correlated with the mean matric potential during the first 21 days but only at $P = 0.10$. These data again support the view that infection and subsequent symptom expression, as vine decline, are favored by early wet conditions.

DISCUSSION

Although microsclerotial populations of *M. phaseolina* may increase in soil (26) and in roots (18) during the growing season, it is not known whether current root infections lead to additional infections within the same season and thus to a polycyclic pattern of root infection. Although the curves representing the percentage of infected root systems were sigmoid, this cannot be used as evidence to support a polycyclic pattern of infection. Many factors in the epidemiology of soilborne pathogens believed to be monocyclic may lead to sigmoid curves (2,9,10); these include cultural practices before planting, changes in host susceptibility during maturation, a variable environment, increases in root density, and artifacts introduced by procedures for retrieving, incubating, and examining root systems.

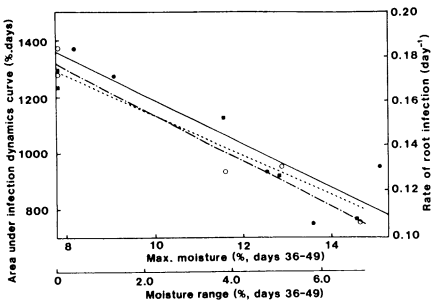

Fig. 6. Area under incidence (percentage of infected root systems) curve (% days) in relation to 1) maximum soil moisture (•), 2) moisture range (o) during the period 36–49 days after planting, and 3) rate of root infection (day^{-1}) (■) in relation to moisture range during the period 36–49 days after planting. Lines represent the regression equations: 1) $y = 1{,}966.0 - 77.6x$ ($R^2 = 0.79$) (———), 2) $y = 1{,}317.6 - 81.8x$ ($R^2 = 0.96$) (—·—), and 3) $y = 0.173 - 0.009x$ ($R^2 = 0.92$) (----)

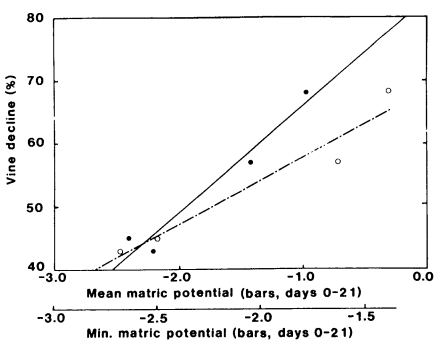

Fig. 7. Incidence of vine decline (%) in relation to 1) mean (•) and 2) minimum (o) soil matric potential bars in the period 0–21 days after planting. Lines represent the fitted regression equations: 1) $y = 82.8 + 16.9x$ ($R^2 = 0.95$) (———) and 2) $y = 89.0 + 17.5x$ ($R^2 = 0.94$) (—·—).

The response to soil moisture content found in this study, in terms of early infection by *M. phaseolina*, casts some doubt on the view that drought conditions are required for extensive infection—at least in cantaloupe. Although the analysis undertaken only suggests association between infection and soil environment variates rather than indicating cause and effect, the consistent reversal in response to soil moisture before flowering and after fruit set does indicate a real effect. At present, we prefer not to speculate as to why higher soil moistures inhibit root infection after fruit set until the effect has been studied under controlled conditions.

The cultivar Perlita begins vining and flowering about 21 days after planting, pollination at about 27 days, fruit set at 27–42 days, and full slip at about 97 days (E. Cox, *personal communication*). Apart from the reversed effects of soil moisture before flowering and after fruit set, no information is available on whether increases in resistance or susceptibility to *M. phaseolina* occur as the cantaloupe plant matures. Agarwal et al (1) found no correlation between soybean plant maturity and susceptibility to *M. phaseolina*. Wyllie and Calvert (25) concluded that maturity, as well as temperature and soil moisture, did not affect infection of soybean.

Infection by *M. phaseolina* occurs almost exclusively on the secondary and tertiary roots of the cantaloupe plant; the fungus then moves into the primary root and on into the crown area. Symptoms of vine decline in the crowns of cantaloupe plants were not expressed until about 85–90 days after planting, despite substantial infection of root systems after 49 days, suggesting the possibility of a latent or quiescent infection similar to those found in postharvest decays (21,24). Young and Alcorn (26) described a similar latent infection by *M. phaseolina* in *Euphorbia lathyris*. Edmunds (5) states that the fungus is probably present in the crowns of most sorghum plants but that stress conditions are necessary for symptom development. Meyer et al (15) reported early but asymptomatic infection of soybean and suggested that such infections were a latent source of inoculum for the mature-plant phase of the disease.

In our view, latent infection is involved with the development of vine decline, caused by *M. phaseolina*, in cantaloupe. Observations made during the past 4 yr indicate that plants that are stunted or showing poor vigor have a low incidence of vine decline; conversely, plants showing good growth and vigor have a very high incidence of vine decline. Edmunds (5) noted a more complete disintegration of tissue in the lower stalk portion of sorghum, especially near the nodal plates, and suggested that stress conditions may cause rapid formation or concentration at these sites of an unidentified plant constituent that favors parasitic growth. It was further hypothesized that greater amounts of such a constituent may be formed in vigorous plants, which are then more susceptible after stress conditions. A similar situation may occur in cantaloupe, with vigorously growing plants being more susceptible in terms of eventual vine decline.

In contrast to reports with other hosts of *M. phaseolina*, drought stress and high temperature were not requirements for early infection of cantaloupe roots. Indeed, the early incidence of infection was favored by soil moisture contents that did not correspond to drought conditions. Similarly, high temperatures consistently (if not significantly) inhibited root infection. The importance of early infection in the development of vine decline and the subsequent inhibition of late root infection by high soil moisture content suggest a more subtle control of disease expression in cantaloupe than has been reported in other crops.

LITERATURE CITED

1. Agarwal, D. K., Gangopodhyay, S., and Sarbhoy, A. K. 1973. Effect of temperature on the charcoal rot disease of soybean. Indian Phytopathol. 26:587-589.
2. Campbell, C. L., Jacobi, W. R., Powell, N. T., and Main, C. E. 1984. Analysis of disease progression and the randomness of occurrence of infected plants during tobacco black shank epidemics. Phytopathology 74:230-235.
3. Cook, A. A. 1955. Charcoal rot of castor bean in the United States. Plant Dis. Rep. 39:233-235.
4. Dhingra, O. D., and Sinclair, J. B. 1978. Biology and Pathology of *Macrophomina phaseolina*. Universidade Federal de Vicosa, Brasil. 166 pp.
5. Edmunds, L. K. 1964. Combined relation of plant maturity, temperature, and soil moisture to charcoal stalk rot development in grain sorghum. Phytopathology 54:514-527.
6. Estiri, M. 1980. A comparison of modified hydrometer and pipette methods for soil particle size analysis. M.S. thesis. Texas A&I University, Kingsville. 80 pp.
7. Ghaffar, A., and Erwin, D. C. 1969. Effect of soil water stress on root rot of cotton caused by *Macrophomina phaseoli*. Phytopathology 59:795-797.
8. Ghaffar, A., Mallik, M. A. B., and Kahn, T. H. 1971. Some factors affecting infection of cotton seedlings by *Macrophomina phaseoli*. Pak. J. Bot. 3:83-87.
9. Gilligan, C. A. 1983. Modeling of soilborne pathogens. Annu. Rev. Phytopathol. 21:45-64.
10. Huisman, O. C. 1982. Interrelations of root growth dynamics to epidemiology of root-invading fungi. Annu. Rev. Phytopathol. 20:303-327.
11. Kendrick, J. B. 1933. Seedling stem blight of field beans caused by *Rhizoctonia bataticola* at high temperatures. Phytopathology 23:949-963.
12. Livingston, J. E. 1945. Charcoal rot of corn and sorghum. Univ. Nebr. Res. Bull. 136. 32 pp.
13. Ludwig, C. A. 1925. A new stem rot of bean in South Carolina. Plant Dis. Rep. 9:60.
14. Menon, K. P. V., Nair, U. K., and Pandalai, K.

M. 1952. Influence of water-logged soil conditions on some fungi parasitic on the roots of the coconut palm. Indian Coconut J. 5:71-79.
15. Meyer, W. A., Sinclair, J. B., and Khare, M. N. 1974. Factors affecting charcoal rot of soybean seedlings. Phytopathology 64:845-849.
16. Norton, D. C., and Frank, F. A. 1963. Charcoal rot (caused by *Sclerotium bataticola* Taub.) on guayule in southwest Texas in 1951 and 1952. Plant Dis. Rep. 37:41-43.
17. Papavizas, G. C., and Klag, N. G. 1975. Isolation and quantitative determination of *Macrophomina phaseolina* from soil. Phytopathology 65:182-187.
18. Pearson, C. A. S., Schwenk, F. W., Crowe, F. J., and Kelly, K. 1984. Colonization of soybean roots by *Macrophomina phaseolina*. Plant Dis. 68:1086-1088.
19. Philip, C. T., Kartha, K. K., Joshi, R. K., and Nema, K. G. 1969. A Rhizoctonia disease of 'Mung' (*Phaseolus aureus* Roxb.) in Madhya Pradesh. JNKVV Res. 3:40-43.
20. Reuveni, R., Krikun, J., Nachmias, A., and Shlevin, E. 1982. The role of *Macrophomina phaseolina* in a collapse of melon plants in Israel. Phytoparasitica 10:51-56.
21. Swinburne, T. R. 1983. Quiescent infection in post-harvest diseases. Pages 1-21 in: Post-Harvest Pathology of Fruits and Vegetables. C. Dennis, ed. Academic Press, New York. 264 pp.
22. Uppal, B. N., Kolhatkar, K. G., and Patel, M. K. 1936. Blight and hollow-stem of sorghum. Indian J. Agric. Sci. 6:1323-1334.
23. Vasudeva, R. S. 1937. Studies on the root-rot disease of cotton in the Punjab. III. The effect of some physical and chemical factors on sclerotia formation. Indian J. Agric. Sci. 7:259-270.
24. Verhoeff, K. 1974. Latent infection by fungi. Annu. Rev. Phytopathol. 12:87-98.
25. Wyllie, T. D., and Calvert, O. H. 1969. Effect of flower removal and pod set on formation of sclerotia and infection of *Glycine max* by *Macrophomina phaseolina*. Phytopathology 59:1243-1245.
26. Young, D. J., and Alcorn, S. M. 1984. Latent infection of *Euphorbia lathyris* and weeds by *Macrophomina phaseolina* and propagule populations in Arizona field soil. Plant Dis. 68:587-589.