Path Coefficient Analysis of Effects of *Rhizoctonia solani* on Growth and Development of Dry Beans

Ariena H. C. van Bruggen and P. A. Arneson

Former graduate research assistant and associate professor, Department of Plant Pathology, Cornell University, Ithaca, NY 14853. Present address of the first author: Department of Plant Pathology, University of California, Davis 95616.
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ABSTRACT

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A path coefficient analysis is presented for the data of two field experiments in which the effects of eight inoculum levels of *Rhizoctonia solani* on growth, development, and yield of dry beans were assessed. The analysis indicated that all three measures of disease (number of plants infected, number of lesions, and lesion area) were equally important in reducing and delaying emergence in the first year, whereas only number of plants infected determined these effects in the second year. Of the yield components, pods per plant exerted the largest influence on yield per unit area. Plants per row was the second most important yield component, but its correlation with yield was not significant or only weakly so, because of a negative correlation between plants per row and pods per plant. The most important path of influence of infection on yield was via numbers of plants infected, numbers of plants established (at flowering) and final numbers of plants per row (at harvest) in both years, and via numbers of lesions, shoot dry weight (at flowering), and number of pods per plant in the first year. Of the yield components only the number of plants per row was significantly reduced by inoculum level, but because of compensation by the other yield components, *R. solani* did not affect overall yield.

Additional key words: correlation coefficients, *Phaseolus vulgaris*, yield components.

Quantitative relationships between inoculum levels or disease incidence or severity and crop yield have been established only for few soilborne pathogens and their hosts (7,9,13,14), and effects on yield have been divided over effects on individual yield components in a few instances only (13,14).

Relationships among inoculum levels, disease, and plant growth and yield variables can be given by correlation coefficients. However, these are "merely the resultant of all connecting paths of influence," and "in many cases a small actual correlation between variables will be found on analysis to be the resultant of a balancing of very much more important but opposed paths of influence leading from common causes" (17). In other words, if there are significant effects along one path in a system, but opposing effects along another path, the end result is likely a nonsignificant correlation coefficient between the end variables connected by those different paths. The nonsignificant correlation does not mean, however, that the individual paths would not be important. Wright (17) developed a technique in which the correlations between variables are divided over direct and indirect influences along different paths in a system, the so-called path coefficient analysis. The direct influences of a set of variables (causes) upon a certain variable (effect) indicate the degree to which variation of that effect is determined by each particular cause. These direct influences are called path coefficients. The assumptions of path analysis are that relationships between variables can be causally structured and that they are causally closed and linear. If variables are not completely explained by other variables in the system, an extra variable can be added that contains residual influences and experimental error.

Path coefficient analysis has often been used in population genetics (10) and agronomy (4–6,16) but rarely in plant pathology (2,8). There are several reasons why we might be interested in path coefficient analysis: 1) to indicate the relative importance of certain factors contributing to yield reduction by a pathogen (e.g., bean yellow and bean common mosaic viruses reduced yield of bean plants primarily by a decreased number of pods per plant [8]); 2) to unravel opposing effects between variables along different paths of influence, which may obscure the importance of certain factors along those paths; and 3) to determine which variables need to be measured to enable yield prediction.

The effects of inoculum levels of *Rhizoctonia solani* Kühn on infection of dry bean seedlings, plant development, and yield were given in a previous report (15), but the interrelationships among the measured variables were not analyzed. The objective of this paper is to determine the relative importance of intermediate variables that determine the influence of *R. solani* on plant development and yield of dry beans.

MATERIALS AND METHODS

In two field experiments the effects of eight inoculum levels of *R. solani* on growth and development of red kidney beans (cv. Redkloud) were determined. Detailed experimental procedures have been described previously (15). Certain variables were selected for path coefficient analysis, namely 1) inoculum level: 0–7, with equal increments of about 120 sclerotia per kilogram of soil; 2) plants infected: numbers of plants with hypocotyl lesions per sample 19 and 14 days after planting in 1982 and 1983, respectively; 3) lesions: numbers of lesions per hypocotyl on the same days as above; 4) lesion area: lesion area per hypocotyl (square millimeter) on the same days as above; 5) emergence: numbers of seedlings emerged 10 and 12 days after planting in 1982 and 1983, respectively; 6) flowering: numbers of plants with at least one flower 43 days after planting in 1983 (no flowering data for 1982); 7) senescence: categorical rating of yellowing of foliage 73 and 69 days after planting in 1982 and 1983, respectively; 8) plant stand: maximal number of plants emerged in midseason (flowering); 9) shoot weight: dry weight of shoot per sample in midseason (flowering), 45 and 41 days after planting in 1982 and 1983, respectively; 10) plants per row, pods per plant, seeds per pod, weight per seed: yield components at harvesttime; and 11) yield: weight of seeds per row.

Numbers of plants emerged or flowering reflected both stand reduction and a delay in development by *R. solani*, since the observations were made in the logarithmic phase of the respective development stages (15).

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The variables selected were grouped in relational diagrams, incorporating possible pathways of influence of inoculum density on plant development and yield. Implied causal relations among variables are commonly represented by unidirectional arrows, and noncausal correlations are represented by two-headed arrows in such diagrams (5,6,8,16,17). Simple linear correlation coefficients were calculated between pairs of all measured variables. All variables were plotted against each other, and if the relationship between two variables seemed curvilinear, the data were transformed to obtain linearity. The specific transformations used are indicated in footnotes to Tables 1 and 3. The correlation coefficient between each pair of variables was decomposed into a direct effect (= path coefficient) and indirect effects (= path coefficient × correlation coefficient) (17). The method may become clear with the following example of the determination of wheat yield by its components (heads per square meter, kernels per head, and kernel weight). A relational diagram is given in Figure 1. The corresponding equations according to Wright (17) are as follows:

$$r_{14} = P_{14} + r_{12} \times P_{24} + r_{13} \times P_{34} \quad (1)$$

$$r_{24} = P_{24} + r_{12} \times P_{14} + r_{23} \times P_{34} \quad (2)$$

$$r_{34} = P_{34} + r_{13} \times P_{14} + r_{23} \times P_{24}, \quad (3)$$

in which r_{ij} are correlation coefficients and P_{ij} path coefficients. P_{ij} reflect direct effects, and $r_{ij} \times P_{ij}$ indirect effects. Thus, correlation coefficients are equal to the summation of direct and indirect effects. After calculation of the correlation coefficients, we have three equations with three unknowns (the path coefficients), which can be solved for the three path coefficients. Path coefficients can also be calculated as standardized partial regression coefficients (5). The first approach, simultaneous solution of a set of equations, was followed for the path analyses we report. Because some of the sets of equations contained as many as 15 unknowns, the equations were solved by calculating generalized inverses of the matrices derived from the equations (12), with the computer package Matlab (11).

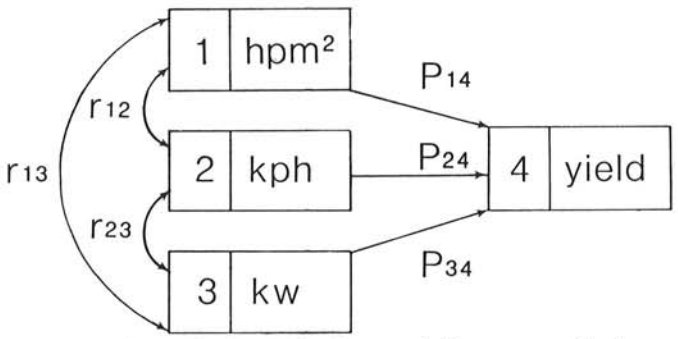

Fig. 1. Path diagram for the relations between yield components (heads per square meter, kernels per head, and kernel weight) and yield per square meter of a wheat crop, to illustrate causal (single-headed arrows) and noncausal (double-headed arrows) relationships in a system.

RESULTS

A relational diagram of inoculum, infection, emergence, flowering, and senescence is given in Figure 2, and the corresponding correlation coefficients are presented in Table 1. Most correlation coefficients were significantly different from 0, but none was close to 1, indicating that a large part of the variability was due to other factors. Yet higher inoculum densities generally resulted in higher levels of infection, and more infection was associated with fewer plants emerged both in 1982 and in 1983. A reduction and delay in emergence resulted in delays in flowering and senescence, which were all positively correlated. Path coefficient analysis indicated that the direct effects of percentage of plants infected, numbers of lesions and lesion area were about equally important in reducing emergence in 1982 but that the direct and indirect effects of percent plants infected were the main determinants in 1983 (Table 2).

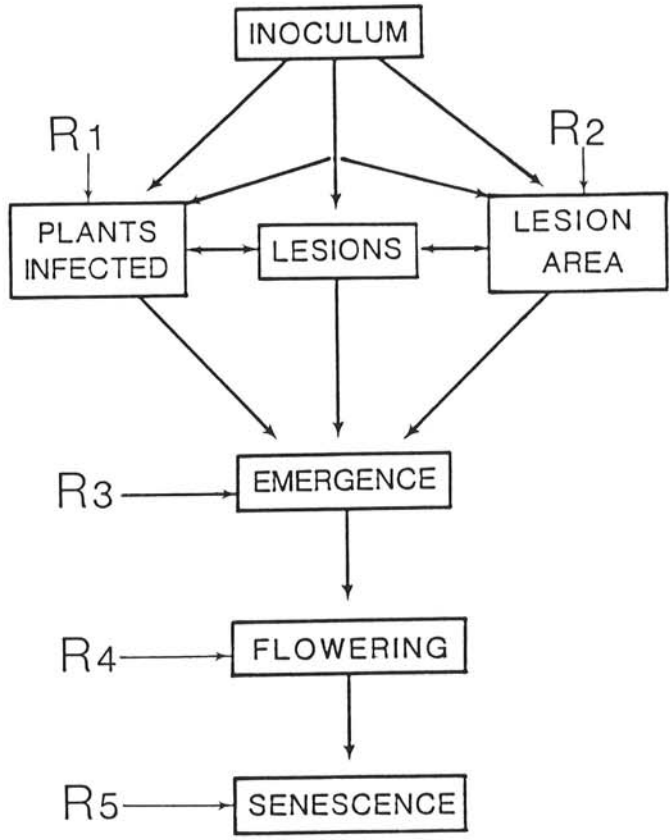

Fig. 2. Path diagram with possible paths of influence of inoculum level of *Rhizoctonia solani* on development of dry beans; the variables measuring infection (plants infected, number of lesions, and lesion area); and emergence were subjected to a path coefficient analysis. R_{1-5} represent unidentified residual factors.

TABLE 1. Linear correlation coefficients[a] between inoculum densities of *Rhizoctonia solani*, percentage of plants infected, number of lesions per hypocotyl, lesion area per hypocotyl, number of plants emerged and flowering, and senescence rating in 1982 (upper right of diagonal) and 1983 (lower left of diagonal)

	Inoculum density	Plants infected (%)	Lesions (no.)	Lesion area	Plants emerged (no.)	Plants flowering (no.)	Senescence rating
Inoculum density		0.84[b]	0.74[b]	0.69[b]	−0.86[b,c]	...[d]	−0.61[b]
Plants infected (%)	0.69		0.78	0.58	−0.61[c]	...	−0.38
Lesions (no.)	0.55	0.76		0.59	−0.61[c]	...	−0.43
Lesion area	0.54	0.67	0.67		−0.59[c]	...	−0.64
Plants emerged (no.)	−0.89[c]	−0.63[c]	−0.46[c]	−0.39[c]		...	0.61
Plants flowering (no.)	−0.69	−0.50	−0.43	−0.44	0.73		...
Senescence rating	−0.43	−0.44	−0.33	−0.16	0.63	0.62	

[a] Significant correlations $r > 0.31$ ($P = 0.05$, df = 38).
[b] After square root transformation of inoculum density.
[c] After square root transformation of numbers of plants emerged.
[d] Not quantified.

The second diagram illustrates the relations between percentage of plants infected, number of lesions, lesion area, plant stand, shoot weight, yield components, and final yield (Fig. 3). The corresponding coefficients are given in Table 3. There were significant positive correlations between pods per plant and yield, and seeds per pod and yield in both years but not between final plant stand and yield in 1982 and weight per seed and yield in 1983. The correlation coefficients seemed to indicate that final yield was determined more by pods per plant and seeds per pod than by numbers of plants or weight per seed.

Path coefficient analysis of the contribution of the yield components to overall yield showed that in both years, pods per plant was indeed the yield component with the largest direct effect on yield (Table 4). Contrary to the expectations from correlation coefficients, the direct effect on yield of plants per row was larger than that of seeds per pod, but the effect of plants per row was largely negated by the negative indirect effect of pods per plant. The negative indirect effects indicate compensation among yield components. In both years an increase in pods per plant and a slight increase in seeds per pod compensated for a reduction in plants per row. In 1983 even the weight per seed compensated slightly for a reduction in plant stand.

TABLE 2. Path coefficient analyses of the relations between percent plants infected by *Rhizoctonia solani*, numbers of hypocotyl lesions, lesion areas, and numbers of bean seedlings emerged

Pathways of association	1982	1983
Infected plants vs. emergence		
Direct effect	−0.248	−0.676
Indirect effect via no. of lesions	−0.185	+0.005
Indirect effect via lesion area	−0.181	+0.052
Total correlation	−0.614[a]	−0.619[a]
Lesions vs. emergence		
Direct effect	−0.231	+0.006
Indirect effect via plants infected	−0.198	−0.511
Indirect effect via lesion area	−0.184	+0.043
Total correlation	−0.613[a]	−0.462[a]
Lesion area vs. emergence		
Direct effect	−0.311	+0.065
Indirect effect via plants infected	−0.145	−0.456
Indirect effect via no. of lesions	−0.136	+0.004
Total correlation	−0.592[a]	−0.387[b]
Residual factors	0.721	0.777

[a] Significant at $P = 0.01$.
[b] Significant at $P = 0.05$.

Path coefficients were calculated for all paired relations between three measures of infection, shoot weight in midseason (flowering), the yield components, and the yield (Fig. 3). Because the numbers of possible paths between the measures of infection and yield were very large (24 possible paths for four to five variables per path), the complete path analysis is not presented here. Although the overall correlation coefficients between the disease measures and yield were generally not significant, path analysis indicated that the lack of significance was due to counteracting effects between variables within the system.

In 1982 the negative correlation between percentage of plants infected and yield ($r = -0.20$, Table 3) was for a large part determined by the negative correlation between numbers of lesions and shoot weight, especially via lesions, shoot weight, and pods per plant. The paths from percentage of plants infected to yield via number of lesions or lesion area and plant stand in midseason and at harvest were also important determinants, because of the negative correlations between number of lesions or lesion area and plant stand in midseason. In 1983 a similar negative correlation between percentage of plants infected and yield (Table 3) was mainly determined by the paths via plant stand in midseason and at harvest directly and indirectly via number of lesions and plant stand in midseason and at harvest. Pathways via shoot weight were relatively unimportant.

Number of lesions were significantly correlated with yield in 1982 ($r = -0.38$, Table 3). This correlation was mainly determined by the path from number of lesions via shoot dry weight at flowering and pods per plant to yield. Moreover, the paths via plant stand in midseason and at harvest (both direct and via lesion

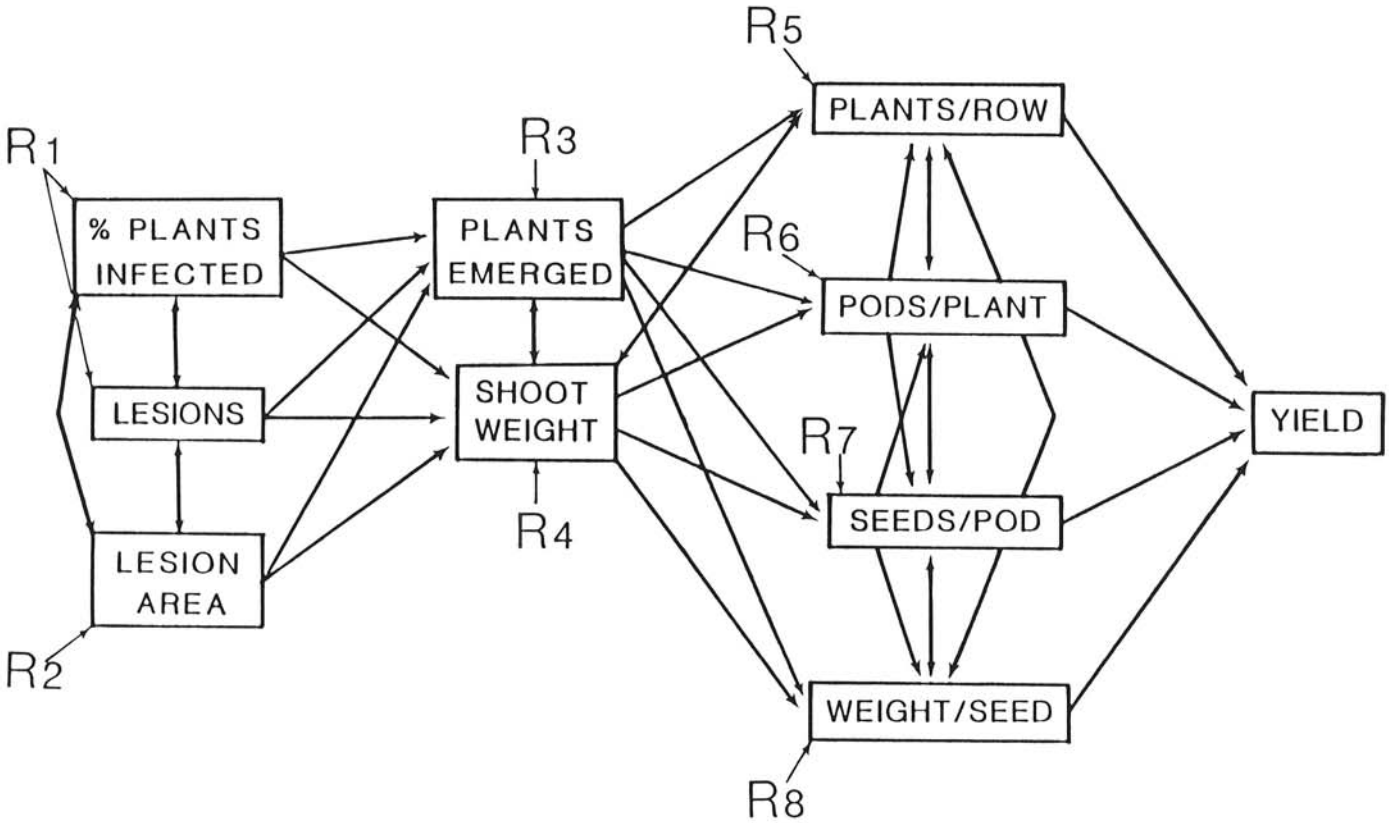

Fig. 3. Path diagram with possible paths of influence of measures of infection by *Rhizoctonia solani* on yield of dry beans; all variables measured at three developmental stages (emergence, flowering, and maturity) were subjected to a path coefficient analysis. R_{1-8} represent unidentified residual factors.

TABLE 3. Linear correlation coefficients[a] between percentage of plants infected by *Rhizoctonia solani*, numbers of lesions and lesion area per hypocotyl, plant stand and shoot dry weight, and yield and its components in 1982 (upper right of diagonal) and 1983 (lower left of diagonal)

	Plants infected (%)	Lesions (no.)	Lesion area	Plants emerged (no.)	Shoot dry weight	Plants per row	Pods per plant	Seeds per pod	Weight per seed	Yield
Plants infected (%)		0.65[b]	0.33[b]	−0.14[b]	−0.26[b]	−0.43[b]	−0.08[b]	0.19[b]	0.13[b]	−0.20[b]
Lesions (no.)	0.76		0.25[b]	−0.22[b]	−0.49[b]	−0.41[b]	−0.22[b]	0.05[b]	0.02[b]	−0.38[b]
Lesion area	0.67	0.67		−0.68[b]	−0.07[b]	−0.61[b]	−0.37[b]	0.12[b]	−0.02[b]	−0.11[b]
Plants emerged (no.)	−0.44	−0.39	−0.31		0.03	0.66	−0.42	−0.07	0.04	0.09
Shoot dry weight	−0.38[c]	−0.30[c]	−0.41[c]	0.34[c]		0.02	0.50	0.39	0.20	0.61
Plants per row	−0.04	0.00	−0.17	0.48	0.30[c]		−0.52	−0.30	0.00	0.13
Pods per plant	−0.18	−0.17	−0.10	−0.02	0.06[c]	−0.35		0.38	0.28	0.67
Seeds per pod	0.15	0.04	0.02	−0.18	−0.39[c]	−0.15	0.00		0.23	0.50
Weight per seed	−0.24	−0.26	−0.15	−0.17	−0.14[c]	−0.13	−0.17	0.02		0.65
Yield	−0.20	−0.21	−0.26	0.21	0.04[c]	0.37	0.51	0.40	0.12	

[a] Significant correlations: $r > 0.31$ ($P = 0.05$, df = 38).
[b] Quadratic transformation of percentage of plants infected, number of lesions, and lesion area per hypocotyl in 1982.
[c] Negative inverse square root transformation ($-1/\sqrt{y}$) of shoot dry weight in 1983.

area) were important determinants too. In 1983 the negative correlation between number of lesions and yield was not significant ($r = -0.21$, Table 3). Paths via shoot dry weight were not important or had positive path coefficients (via shoot weight and seeds per pod). Paths via plant stand in midseason and at harvest (both direct and via percent plants infected) were the main determinants for the negative correlation between number of lesions and yield.

For the correlations between lesion area and yield ($r = 0.11$ and -0.26 in 1982 and 1983, respectively), the direct path via plant stand in midseason and at harvest was of overriding influence in 1982, and the indirect path via percent plants infected and plant stand in midseason and at harvest was most important in 1983. In 1982 the indirect path via numbers of lesions, shoot dry weight, and pods per plant was also important.

In summary, for all relationships between measures of infection and yield, some paths via plant stand at flowering and at harvest were important in both years, and some paths via shoot dry weight at flowering and pods per plant were important only in 1982.

DISCUSSION

Path coefficient analysis of the effect of *R. solani* on development of dry beans indicated that percentage of plants infected was an important measure of disease, especially in 1983. In 1982, numbers of lesions and lesion area were equally important. The overwhelming effect of percentage of plants infected in 1983 (and not in 1982) may be explained by the fact that in 1983 the plants were sampled at an earlier stage than in 1982, so that the differences in percentage of plants diseased between inoculum levels were more pronounced in 1983 than in 1982. Weather conditions were about equally favorable for infection in both years. Beebe et al (3) mentioned that plants severely infected by *R. solani* developed more slowly, but they did not quantify this effect.

The greater importance of numbers of lesions per hypocotyl in 1982 than in 1983 was also expressed in the significant negative correlation between this variable and overall yield in 1982. Path coefficient analysis showed that the number of lesions exerted its influence mainly via a reduction in shoot dry weight in midseason and in numbers of pods per plant at harvesttime. The individual correlations between number of lesions and shoot weight and between shoot weight and pods per plant were significant in 1982. However, the relationships were not strong enough to result in a significant effect of inoculum level on number of pods per plant. In 1983 there was no significant correlation between shoot weight and pods per plant, and the pathways via these variables were unimportant. In 1983, there was a dry spell during flowering and podset. Although the plants were irrigated at early podset (15), this was probably too late for differences in podding potential to be expressed. The occurrence of compensation at later stages in development, notably by increased seed weight in 1983, substantiates this hypothesis. During the stage of pod-fill, rain was more abundant that year, so that seed weight could compensate for reductions in plants per row and pods per plant. The influence of environmental stress on yield component compensation in beans was pointed out by Adams (1).

TABLE 4. Path coefficient analyses of the relations between yield components (plants per row, pods per plant, seeds per pod, weight per seed), and yield

Pathways of association	1982	1983
Plants per row vs. yield		
Direct effect	+0.619	+0.795
Indirect effect via pods/plant	−0.401	−0.301
Indirect effect via seeds/pod	−0.091	−0.074
Indirect effect via weight/seed	+0.000	−0.048
Total correlation	+0.128	+0.372[a]
Pods per plant vs. yield		
Direct effect	+0.777	+0.852
Indirect effect via plants/row	−0.320	−0.281
Indirect effect via seeds/pod	+0.115	+0.001
Indirect effect via weight/seed	+0.100	−0.060
Total correlation	+0.673[b]	+0.513[b]
Seeds per pod vs. yield		
Direct effect	+0.307	+0.508
Indirect effect via plants/row	−0.183	−0.116
Indirect effect via pods/plant	+0.292	+0.002
Indirect effect via weight/seed	+0.081	+0.008
Total correlation	+0.497[b]	+0.401[b]
Weight per seed vs. yield		
Direct effect	+0.357	+0.358
Indirect effect via plants/row	+0.001	−0.107
Indirect effect via pods/plant	+0.218	−0.142
Indirect effect via seeds/pod	+0.070	+0.011
Total correlation	+0.646[b]	+0.120

[a] Significant at $P = 0.05$.
[b] Significant at $P = 0.01$.

In 1983, the influence of *R. solani* on yield was largely exerted via numbers of plants infected and plant stand at flowering and harvest. This pathway was of secondary importance in 1982. Final plant stand was the only yield component that was significantly reduced by inoculum level in both years (15). However, the reduction at the highest inoculum level was only 16% compared with the control (15), and this was apparently not enough to influence final yield. Beebe et al (3) used plant survival as an indicator for resistance to *R. solani*, because hypocotyl lesions and disease severity had little effect on plant yield, and only after severe stand reduction was plot yield reduced. The lack of effect on plant yield might have been caused by counteracting effects of the pathogen and yield component compensation. The same mechanism appeared to be operational in our experiments. The direct effects of plants per row on yield were mainly negated by indirect effects of pods per plant on yield. The importance of

number of pods per plant at different plant densities has been reported (4,16).

The stages of development at which environmental stresses are exerted, including those by pathogens, determine which yield components are affected (13). If the stress is exerted at an early stage, as is the case for *R. solani*, compensation by later yield components can negate effects on an earlier component.

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