Effects of Inoculum Level of *Rhizoctonia solani* on Emergence, Plant Development, and Yield of Dry Beans

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ABSTRACT

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The effect of inoculum density of *Rhizoctonia solani* on hypocotyl infection, plant development, and yield of dry beans was evaluated in two field experiments using microplots. Fumigated soil was mechanically mixed with 0–800 sclerotia per kilogram of soil. The population means of the periods from sowing to emergence, flowering, and podset increased linearly with inoculum level. The proportion of plants infected, numbers of lesions, and lesion areas increased with higher inoculum levels in the form of a saturation curve, the maximum being reached at 250–350 sclerotia per kilogram of soil. Of the yield components, only the numbers of plants per plot decreased significantly with increasing inoculum densities, and the overall yield was not affected by level of infestation (100–800 sclerotia per kilogram of soil). Initial number of lesions per hypocotyl was the only disease measurement that was negatively correlated with yield.

Additional key words: crop loss, inoculum density, microplots, *Phaseolus vulgaris*, yield components.

Rhizoctonia solani Kühn is one of the pathogens of a root rot complex of the common bean (*Phaseolus vulgaris* L.) (5,12). Quantitative relationships between inoculum density and disease incidence or severity have been reported (4,10,25), and disease has generally increased with increasing inoculum densities but at a decreasing rate at the highest levels used (4,10,13,23,28). Most of these studies were performed in greenhouses or growth chambers, and yield data were not obtained.

Prediction of potential yield as a function of disease severity is imperative for integrated management programs for bean production. Beebe et al (3) reported that plot yields of dry beans were only affected by *R. solani* when there was a considerable stand reduction. Sharma and Sohi (19) obtained 13–54% loss in green bean pods at stand reductions of 24–50% in field plots infested with *R. solani*. Abawi and Cobb (1) related inoculum densities in microplots to disease severities and reported significant reductions in snap bean yields in microplots infested with *R. solani*. To aid in the development of an integrated program for root rot control in New York, more quantitative data were deemed necessary concerning the relationship between individual root rot pathogens and yield. In this paper we report on the effects of sclerotial densities of *R. solani* on dry bean growth and development, disease incidence and severity, and bean yield and its components. A preliminary report has been published (22).

MATERIALS AND METHODS

Two field experiments in microplots with eight inoculum levels were conducted in 1982 and 1983 4 km east of Ithaca, NY.

Soil. The soil type was a Darien gravelly silt loam, which is a poorly to moderately drained soil, with a moderately fine texture and distinct horizons. The C-horizon, at about 0.5–1.0 m depth, consists of very firm and slowly permeable silty clay loam (6). Soil chemical analysis indicated that the organic matter content and cation exchange capacity were relatively high (3.5% and 18 meq/100 g, respectively). The pH was low (5.5), the available phosphate and zinc were low (0.74 and 0.13 g/m^2, respectively), and potassium, magnesium, and calcium were high (13.7, 19.5, and 235 g/m^2, respectively). The soil was fumigated with methyl bromide and 2% chloropicrin at a rate of 50 ml/m^2 5 wk before infestation. The soil was limed with agricultural hydrated lime (85% passing 200 mesh) at a rate of 9 MT/ha (17). The resulting pH (in 0.01 M $CaCl_2$) was 7.3. A side-dressing of ammonium nitrate (40 kg of nitrogen per hectare) and triple superphosphate (54 kg of phosphate per hectare) was applied at planting time (5 cm to the side of and 5 cm lower than the bean seeds).

Experimental design. The experiments were arranged in a randomized complete block design with five replications.

Inoculum preparation and soil infestation. One isolate of *R. solani* was used (R-2 from G. S. Abawi's collection, N.Y. Agric. Exp. Stn., Geneva), isolated from beet roots in New York and highly pathogenic to bean hypocotyls and roots. Its anastomosis grouping was unknown, since it failed to anastomose with any of the anastomosis groups tested (AG 1–4) (9). Sclerotia 300–710 μm in diameter were prepared as described by van Bruggen and Arneson (20). The viability of two preparations of sclerotia on acidified water agar (pH 4.8) after 24 hr was 50% in 1982 and 80% in 1983. These percentages were taken into account when the amounts of inoculum to be added to microplots were calculated.

Microplots 1.2 × 1.8 m were constructed, surrounded by fiberglass walls sunk into the soil to a depth of 20 cm (2). Because in a preliminary greenhouse experiment (with 0–700 sclerotia per kilogram of soil) the proportion of plants infected and the number of lesions per hypocotyl leveled off at 350 sclerotia per liter, a similar range of inoculum levels was used in two microplot experiments in 1982 and 1983. Inoculum levels in the microplots included an uninfested check and seven levels with multiples of 120 (1982) and 115 (1983) viable sclerotia per kilogram of air-dried soil. For each plot the required amount of inoculum, together with 1 L of vermiculite (Zonolite, W. R. Grace and Co., Cambridge, MA) as carrier, was mixed with the upper 10–15 cm of soil in a concrete mixer and dumped back into the plot. The actual depth of infestation was 13 (±2) cm in 1982 and 12 (±1) cm in 1983.

Inoculum density. Before sowing, composite soil samples consisting of four subsamples were collected from each microplot with a trowel (13–15 cm deep). The organic debris from subsamples was plated onto Ko and Hora's medium (11) and checked for

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occurrence of *R. solani* as previously described (21). After harvest, population densities of *R. solani* were assessed again both in bulk soil (between rows) and in the rhizosphere (within the rows).

Cultural practices. Cultural practices were performed as recommended to growers (17). Light red kidney beans (cv. Redkloud) were treated with captan 50WP (2.33 g/kg of seed) and diazinon (4% dust, 1.38 g/kg of seed). Seventy-five seeds were sown at a depth of 5 cm in three rows per plot, so that the distance between rows was 60 cm and that between seeds was 5 cm. In 1983, 46 days after planting (just after podset) the plots were irrigated with 57 L of water per plot (equivalent to 25 mm of rain).

Data collection and statistical analysis. Plant development was monitored by counting the numbers of plants emerged, starting to flower (1983 only), and starting to form pods (1983 only) at regular intervals. The percentages of plants in a certain development stage were transformed into probits, which were then regressed on time for each individual plot. From the slopes and intercepts of the regression equations, the mean periods from sowing to a development stage and their standard deviations were calculated and regressed on block and coded inoculum level (0–7).

Senescence could not be monitored on an individual plant basis. However, discrete senescence scores were given per plot on a 0–5 scale, based on percentage of green leaf area. The scores were correlated with the coded inoculum levels (Spearman's rank correlation).

Plant growth and infection were monitored by destructive sampling of random plants in the outer rows of each plot. In 1982 six plants per plot were sampled 1 wk after emergence and just after flowering. In 1983 two or four plants per plot were sampled seven times during the season. Data were collected on: numbers of plants with hypocotyl and root lesions, number of hypocotyl lesions per plant, total lesion area per hypocotyl, and fresh and dry weights of roots, leaf blades, and stems plus petioles (only in 1983). Dry weights were obtained after drying the tissue for 3 days at 60 C. The data were analyzed by regression on block and coded inoculum level, after appropriate transformations. Selected hypocotyls were dipped in 1% sodium hypochlorite for 1 min, rinsed twice in sterile water, and plated onto Ko and Hora's medium (11).

The middle rows were harvested when about 90% of the pods were mature. The yields were divided into their components: number of plants per plot, number of pods per plant, number of seeds per pod, and seed weights. Before being shelled, the pods were dried in an oven at 60 C for 24 hr. The percentage of moisture in the seeds was determined with a Digital Moisture Computer 700 (Burrows Equipment Co., Evanston, IL). The yield per hectare was calculated, based on a moisture content of 18%. The yield components and overall yield were regressed on block and coded inoculum level. The analyses were performed using the SAS or MINITAB statistical packages.

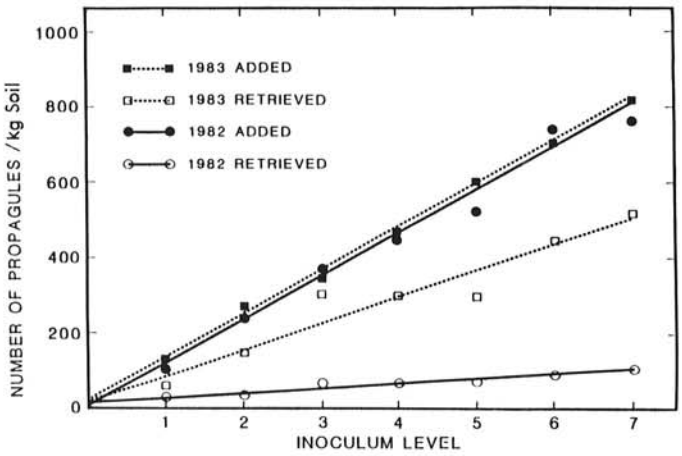

Fig. 1. Numbers of sclerotia of *Rhizoctonia solani* added to a kilogram of soil and numbers of propagules retrieved from soil (using a wet-sieving method) plotted versus intended inoculum level.

RESULTS

There were linear relationships between the numbers of sclerotia added to and the numbers of propagules retrieved from soil and the intended inoculum levels (Fig. 1).

In both years emergence was progressively delayed with increasing inoculum levels. The emergence of seedlings over time formed sigmoid curves resembling cumulative normal distributions. The shape and relative position of the curves for the different inoculum levels were similar in both years (Fig. 2A and B). The mean periods to emergence, calculated from slopes and intercepts of the probit lines (obtained from regression of probits on time), increased linearly with inoculum level (Table 1). Quadratic and higher polynomial terms of inoculum level were not

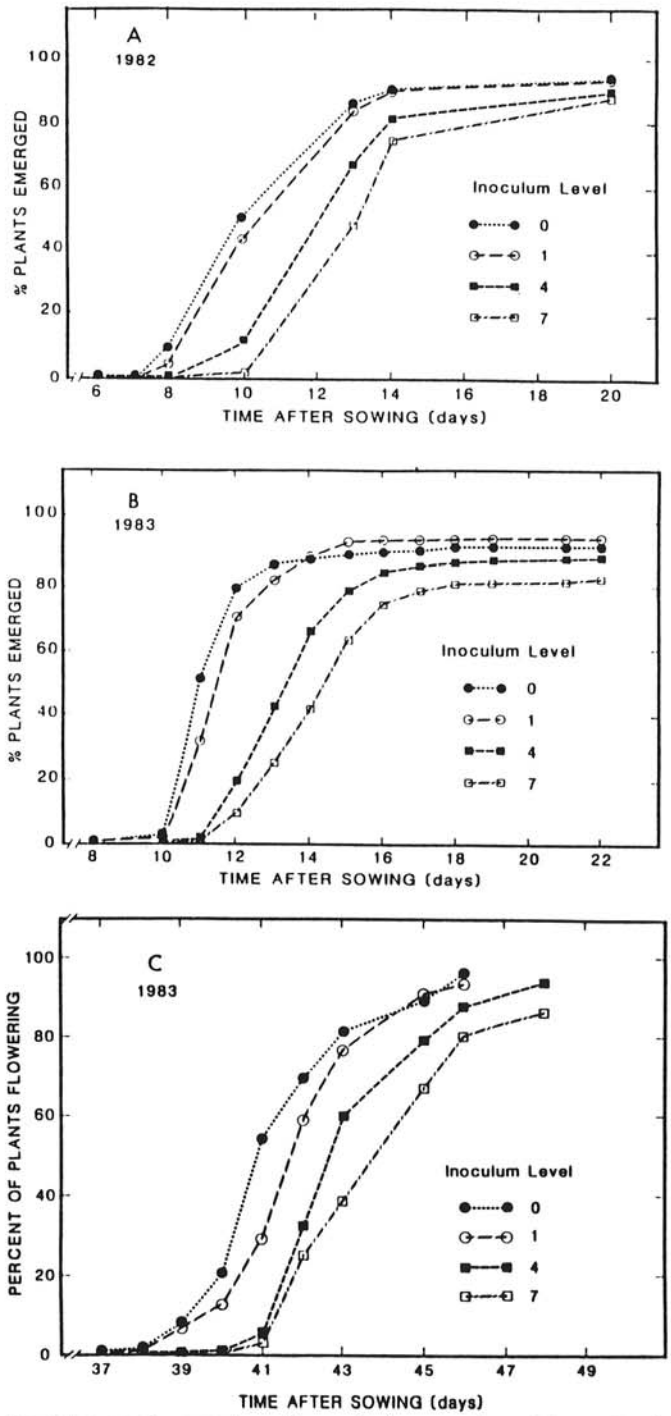

Fig. 2. Increase in percentage of bean plants emerged per plot over time in 1982 **(A)** and 1983 **(B)** and in percentage of plants flowering in 1983 **(C),** at four inoculum levels of *Rhizoctonia solani.*

TABLE 1. Results of regression of mean periods (days) to emergence, flowering, and podset and their standard deviations on inoculum levels of *Rhizoctonia solani*

Year	Dependent variable	Mean	Partial regression coefficient			R^2 (%)	CV (%)
			Intercept	Block	Slope		
1982	Mean period to emergence	12.49 ± 1.14	11.2	−0.05	0.41[a]	70	5
	Standard deviation of emergence	1.91 ± 0.33	1.89	0.04	−0.03	8	17
1983	Mean period to emergence	13.40 ± 1.39	12.5	−0.25[b]	0.48[a]	70	6
	Standard deviation of emergence	1.74 ± 0.64	1.81	−0.08	0.05	6	37
	Mean period to flowering	43.13 ± 1.31	42.2	−0.18	0.40[a]	55	2
	Standard deviation of flowering	1.71 ± 0.36	1.72	−0.05	0.04	9	21
	Mean period to podset	44.79 ± 1.28	43.7	−0.11	0.39[a]	54	2
	Standard deviation of podset	1.40 ± 0.34	1.16	0.01	0.05[b]	15	23

[a]Significantly nonzero at $\alpha = 0.01$.
[b]Significantly nonzero at $\alpha = 0.05$.

TABLE 2. Results of regression of proportion of bean seedlings infected (after natural log and multiple infection transformation) on inoculum level of *Rhizoctonia solani* (after log transformation)

Year	Day	Independent variable	Coefficient	t-Value	R^2 (%)	CV (%)
1982	19	Intercept	0.038	0.41	72	24
		Block	−0.009	−0.40		
		Inoculum[a]	0.980	7.50[b]		
	45	Intercept	0.173	1.54	65	28
		Block	0.003	0.11		
		Inoculum[c]	0.996	5.61[b]		
1983	14	Intercept	0.062	0.68	42	62
		Block	−0.025	−1.22		
		Inoculum	0.521	5.06[b]		
	20	Intercept	0.407	3.52[b]	22	43
		Block	−0.028	−1.05		
		Inoculum	0.400	3.04[b]		
	41	Intercept	0.387	3.31[b]	47	38
		Block	−0.047	−1.70		
		Inoculum[d]	0.682	4.54[b]		

[a]Inoculum levels 0–4 only.
[b]Significantly nonzero at $\alpha = 0.01$.
[c]Inoculum levels 0–3 only.
[d]Inoculum levels 0–5 only.

TABLE 3. Results of weighted regression of number of lesions per bean hypocotyl on inoculum level of *Rhizoctonia solani* (after log transformation)

Year	Day	Independent variable	Coefficient	t-Value	R^2 (%)	CV (%)
1982	19	Intercept	−0.033	−0.21	85.5	31.6
		Block	−0.013	−0.27		
		Inoculum	7.903	14.76[a]		
1983	14	Intercept	−0.032	−0.31	64.6	73.8
		Block	−0.011	−0.36		
		Inoculum	2.117	8.21[a]		
	20	Intercept	2.380	2.48	17.9	42.7
		Block	−0.082	−0.35		
		Inoculum	3.332	2.82[b]		

[a]Significantly nonzero at $\alpha = 0.01$.
[b]Significantly nonzero at $\alpha = 0.05$.

TABLE 4. Results of weighted regression of lesion area per bean hypocotyl on inoculum level of *Rhizoctonia solani* (after log transformation)

Year	Day	Independent variable	Coefficient	t-Value	R^2 (%)	CV (%)
1982	19	Intercept	−0.84	−0.23	64.0	14.4
		Block	0.24	0.22		
		Inoculum	56.64	8.11[a]		
	45	Intercept	16.97	0.40	70.8	3.4
		Block	13.75	1.30		
		Inoculum	423.07	9.38[a]		
1983	14	Intercept	−0.02	−0.06	55.1	29.2
		Block	−0.002	−0.02		
		Inoculum	15.75	6.74[a]		
	20	Intercept	5.11	0.66	60.5	13.0
		Block	−0.34	−0.16		
		Inoculum	64.65	7.53[a]		
	41	Intercept	78.32	1.90	22.1	8.4
		Block	−7.73	−0.71		
		Inoculum	145.48	3.16[b]		

[a]Significantly nonzero at $\alpha = 0.01$.
[b]Significantly nonzero at $\alpha = 0.05$.

significant when included in the regression analysis. The block effect was only significant in 1983. The standard deviation of emergence, calculated from the slopes of the probit lines, was not affected by inoculum level.

The delay in emergence by *R. solani* resulted in corresponding delays in flowering and podset (Table 1), the mean periods to flowering and podset increasing linearly with inoculum level, and there were no significant block effects. At successive development stages the absolute delay by *R. solani* remained the same (95% confidence intervals of the slopes were 0.38–0.58, 0.28–0.52, and 0.27–0.51 for the periods to emergence, flowering, and podset, respectively). The standard deviations of the time to reach these growth stages were not related to inoculum level, except for the standard deviation of time to podset in 1983, which increased with inoculum level.

Senescence ratings 73 and 69 days after planting in 1982 and 1983, respectively, were significantly correlated with inoculum level, with correlation coefficients of −0.66 and −0.44, respectively. Later ratings were no longer related to inoculum level.

The relationship between proportion of plants infected and inoculum level had the form of a saturation curve. In order to linearize this relationship, the proportions of plants infected and inoculum levels were transformed using multiple infection and natural log transformation. Initial analyses indicated that the variances were dependent on the means. Therefore, the final regressions of the transformed data on the natural logarithm of inoculum level were weighted with the inverse of the variances of the transformed data. On all sampling days the proportion of bean plants infected increased significantly with increasing inoculum level (Table 2). In the first year 100% of the plants became infected at about 300 sclerotia per kilogram of air-dried soil; in the second year this happened at 100–200 sclerotia per kilogram of air-dried soil. Some plants of the control plots also became infected, and isolations from infected hypocotyls indicated the presence of *R. solani* in 11% of the control plants with symptoms.

Initial analyses of number of lesions and lesion area per hypocotyl showed that the variances were again dependent on the means, so that weighted regressions were performed as discussed above. The number of lesions per hypocotyl increased linearly with the natural logarithm of inoculum level (Table 3). The total lesion area increased similarly with higher inoculum levels (Table 4). Regression of dry weights of various plant parts on block and inoculum level at three stages of development showed significant inoculum effects, especially earlier in the season (Table 5).

At each stage of development the numbers of seedlings per row were reduced at higher inoculum levels. The differences became less pronounced over time but were still significant at maturity

(Table 6).

The differences in rate of plant development, infection, and increase in dry matter did not result in a difference in overall yield, which averaged 3,118 and 2,327 kg/ha in 1982 and 1983, respectively (Table 6). Of the yield components, only the numbers of plants per row decreased significantly with increasing inoculum level. The relationship was linear in 1982 and nonlinear in 1983. The difference was mainly due to a larger variability at intermediate levels. There were significant block effects of several components in 1982 and of dry weight per seed in 1983. Disease assessment at harvest was not related to any of the yield components or overall yield. Of the earlier disease assessments, only the number of lesions in 1982 was negatively correlated with yield ($r = -0.31$; $P = 0.05$).

At the end of the season the population density of *R. solani* in the bulk soil dropped to one-third (1982) or one-fifth (1983) of the original population, but there was still a linear relation with inoculum level ($Y = 14.0 + 4.3X$ and $Y = 3.6 + 5.8X$, respectively; Y = propagules per kilogram of soil and X = inoculum level). However, because of the large variability, the R^2s were only 26 and 23% in 1982 and 1983, respectively. The populations in the rhizosphere were generally higher than those in the bulk soil, but the variability was even larger. There was no relationship between these populations and the original inoculum levels in the first year and only a weak linear relation in the second ($Y = 21.4 + 6.44X$; $R^2 = 11\%$). In the last year the block effect was significant, with higher populations in the relatively wet plots.

DISCUSSION

R. solani delayed emergence and further development of dry beans in the field, and the delay was proportional to inoculum level. At the highest inoculum level the mean delay was 3 days, both in 1982 and 1983, which is about 24% of the mean period to emergence in the control plots. At the time of podset the differences in development had neither increased nor decreased. The phenomenon of delayed emergence of beans by *R. solani* was also observed by Beebe et al (3) but was not quantified.

McIntyre and Boyer (14) recently pointed out the role of the water balance in seedling development. The cell turgor as regulated by the negative water pressure and osmotic potential was shown to be an important factor in regulating initial plant development. Water uptake by the roots is likely hampered by *R. solani*, and at equal transpiration rates the turgor pressure would be lower in infected plants, resulting in a slower rate of straightening of the hypocotyls and unfolding of primary leaves.

All disease measurements increased with increasing inoculum levels in the form of a saturation curve, and maximum infection was obtained at intermediate inoculum levels (250–350 sclerotia per kilogram of soil). These levels correspond to 5–16 propagules per 100 g of dry soil, as determined with a wet-sieving method, and are similar to those in naturally infested soil (15,24). At these inoculum levels, 100% of the plants became infected, but only about 40% of the hypocotyl areas became infected (45 days after planting).

When organic substrates infested with *R. solani* were used as inoculum, significant reductions in pathogenicity have been reported at higher inoculum levels (8,18). This phenomenon has been ascribed to staling products (18), increase in antagonism due to additional organic matter (8), or increased growth and spread through soil at higher dilutions of the inoculum (10). To avoid these complications we used sclerotia with the least possible additional organic matter as inoculum. And indeed, the effects of *R. solani* did not significantly decrease at higher inoculum levels.

TABLE 5. Relation between dry weights (g/m^2) of plant parts and inoculum levels of *Rhizoctonia solani* at three stages of development in 1983

Days after sowing	Growth stage	Dependent variable	Partial regression coefficient			R^2 (%)	CV (%)
			Intercept	Block	Slope		
14	Unifoliate	Root	2.85	0.01	−0.33[b]	76	26
		Stem	6.50	0.02	−0.27[b]	18	26
		Leaf[a]	2.05	0.07	−0.22[b]	75	19
41	Flowering	Root	25.1	−1.05	−1.49[b]	30	35
		Stem	91.0	−1.49	−8.36[b]	57	31
		Leaf	159.0	−2.61	−12.0[b]	47	28
97	Maturity	Root	19.8	0.34	−0.87	20	31
		Stem	168.0	2.13	−9.78[b]	52	22
		Green leaf	48.8	6.99	−3.16	9	83
		Yellow leaf[a]	9.7	−0.39	−0.40[c]	26	30
		Large pod	270.0	12.1	−6.58	9	34
		Shrivelled pod	6.2	0.51	−0.50	23	55
		Seed	193.0	11.9	−5.23	12	35

[a] Regression after square-root transformation.
[b] Significantly nonzero at $\alpha = 0.01$.
[c] Significantly nonzero at $\alpha = 0.05$.

TABLE 6. Results of regression of yield components (plants per row, pods per plant, seeds per pod, and dry weight per seed) and yield on inoculum level of *Rhizoctonia solani*

Year	Dependent variable	Mean	Partial regression coefficient				R^2 (%)	CV (%)
			Intercept	Block	Slope	Slope^2		
1982	Plants/row	21.3 ± 2.6	24.2	−0.18	−0.68[a]	···	38	10
	Pods/plant	6.6 ± 1.1	6.4	−0.00	0.04	···	1	17
	Seeds/pod	3.0 ± 0.2	3.1	−0.05[b]	0.01	···	15	6
	Dry wt/seed	0.5 ± 0.04	0.5	−0.02[a]	0.00	···	37	6
	Yield (kg/ha)	3,118 ± 678	3,882	−178.0[b]	−65.0	···	19	20
1983	Plants/row	21.0 ± 2.4	20.9	−0.1	1.27[b]	−0.23[a]	30	10
	Pods/plant	6.2 ± 0.8	6.2	−0.03	0.00	···	0.2	14
	Seeds/pod	2.8 ± 0.2	2.8	−0.03	0.03	···	13	7
	Dry wt/seed	0.4 ± 0.03	0.4	0.01[b]	−0.00	···	12	5
	Yield (kg/ha)	2,327 ± 364	2,465	−21.8	−20.7	···	2.5	16

[a] Significantly nonzero at $\alpha = 0.01$.
[b] Significantly nonzero at $\alpha = 0.05$.

Despite the high disease incidence and moderately high disease severity, there was no effect of *R. solani* on yield. Beebe et al (3) used plant survival as a measure of resistance to *R. solani* in beans, because disease severity had little effect on plant yield. They suggested that compensation might have counteracted yield reduction. Wolock (27) suggested the same for the interaction of beans with *Fusarium solani* f. sp. *phaseoli.*

The optimum plant density of snap beans has been estimated at 40 or 47 plants per square meter, when planted equidistantly (7,16). At plant densities lower than 20 or 30 plants per square meter there is a steep decline in the yield per unit area of determinate cultivars (7,16,26). With row planting the decline in yield would probably take place at lower densities (i.e., less than 20–30 plants per square meter) than with equidistant planting, but plants infected by *R. solani* may be less able to compensate than are healthy plants. Threshold densities have not been determined for diseased plants. However, if we assume that the threshold would be 20 plants per square meter, we could possibly have lost 33% of the plants (10 of 30 plants per square meter) before noticing a yield reduction. At the highest level of infestation the stand reduction was about 16% compared with the control, and this stand reduction may have been too small to result in a reduction in yield per plot. Because determinate bean cultivars can compensate to some extent, *R. solani* could have had an effect on yield components other than number of plants per unit area, but this effect remained unnoticed, probably due to compensation. Indeed, the remaining plants in infected plots often were larger and had thicker stem bases than the plants in control plots.

In these experiments the pods were only harvested after 90% or more of them had dried. Therefore, later maturing plots had the chance to make up for the delay in development. In commercial fields uneven maturation may cause yield losses, especially in snap beans which are all harvested at once when the majority of the pods are at the desired stage.

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