Influence of Inoculum Type and Moisture on Development of *Rhizoctonia solani* on Foliage of Table Beets

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ABSTRACT

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Thanatephorus cucumeris, the teleomorph of *Rhizoctonia solani*, was observed for the first time on table beets under field conditions in New York State during the rainy summers of 1990 and 1992 but not during the dry summer of 1991. Hymenia appeared as a thin, dusty, membranous growth on the crowns and lower part of the petioles. Isolations from small circular lesions (3–6 mm in diameter) on leaves of plants with hymenia present, as well as on adjacent plants lacking hymenia, always yielded *R. solani*. The efficiency of different sources of inocula of *R. solani* (mycelial suspension, colonized beet seeds, and pasteurized soil infested with *R. solani*) in initiating foliar blight (pocket rot) in experimental field plots was tested. The most effective inoculum preparation for consistent and severe disease development was the colonized table beet seeds. With all the inocula sources tested, covering the inoculated beet tissues with field soil to simulate cultivation conditions always increased disease incidence and severity. A leaf wetness period of 72 hr was necessary for infection by *R. solani* on table beets. Incubating inoculated plants in a mist chamber resulted in more severe disease development than placing plants in plastic bags and maintaining them in the greenhouse. Infections by *R. solani* were restricted on inoculated plants that were left on the bench without a cover. We concluded that the primary source of inoculum for development of foliar blight on table beets is infested soil thrown on petioles and crowns of the plants during cultivation. The disease is also exacerbated by high moisture environment and the production and spread of basidiospores.

Additional keywords: *Beta vulgaris*, inoculum sources, root rot

About 931 ha are planted annually to table beets (*Beta vulgaris* L.) in New York State, mostly for processing purposes (12). For many years, root rot has been a major disease of table beets, affecting both the quality and the quantity of fleshy roots and thus the value of marketable yield. *Rhizoctonia solani* Kühn, the anamorph of *Thanatephorus cucumeris* (A.B. Frank) Donk, is one of two major root pathogens involved in this disease of table beets in New York State (1). The other major pathogen is *Pythium ultimum* Trow. Foliar infections caused by *R. solani* that result in the pocket rot disease syndrome also occur on table beets but have not been considered a major problem. However, the incidence and severity of pocket rot have been increasing during the past few years, resulting in significant economic losses (14). Pocket rot foci generally are evident late in the growing season and continue to progress under favorable weather conditions throughout the season. The disease was appropriately referred to as pocket rot to describe the randomly scattered pockets (foci) of infected and dead plants within a field (14,15). The incidence and severity of this disease have been especially high during prolonged periods of excessive moisture, but detailed information on factors influencing pocket rot development on table beets is limited.

R. solani is commonly found in both cultivated and noncultivated soils throughout the world (13) and is known to cause a large number of distinct diseases on a wide variety of plants and under more diverse environmental conditions than any other fungal pathogen (2). It exists in many different groups or strains that differ in morphology, pathology, physiology, and ecology (13,15). The diverse characteristics of the numerous strains provide this pathogen with great potential for adaptability and survival (2). The predominant strain of *R. solani*, causing foliar blight (pocket rot) and root rot diseases on table beets in New York State, has been recently shown to belong to anastomosis group 2-2 (AG-2-2) (15).

R. solani can survive in soil as sclerotia, in association with crop residues by pathogenic growth on hosts, or by saprophytic growth on organic matter. Primary inoculum sources consist of sclerotia and hyphae or, when the teleomorph *T. cucumeris* is present, basidiospores. Infected weeds or rotational crops may also provide sources of inoculum (3). The pathogen can be disseminated by irrigation water, infected or contaminated seeds, transplanted materials, air currents, and rain-splashed soil (3). In the tropics, where the teleomorph of *R. solani* develops regularly, the pathogen spreads rapidly by the production and dissemination of basidiospores (4,7).

Moisture is an important factor impacting on inoculum potential of *R. solani* and the epidemiology of the resultant diseases. The development of diseases incited by *R. solani* on aerial parts of host plants is dependent upon the availability of free moisture or conditions near 100% RH (3). These conditions are required for germination and growth of basidiospores and subsequent infection of host tissues and resulting epidemics (3,10). In soils that remain moist for most of the year, the fungus essentially continues vegetative growth, whereas in soils that become extremely dry for an extended period of time, *R. solani* becomes dormant as thick-walled mycelia or sclerotia (2).

Effective control measures against *R. solani* on table beets need to be developed and implemented, and detailed information on the epidemiology of foliar blight is needed. The objectives of this study were to: 1) identify the inoculum sources responsible for epidemic development of this disease under field conditions, 2) evaluate the efficiency of different inocula sources of *R. solani* in initiating disease in experimental plots, and 3) establish the role of moisture in disease development.

MATERIALS AND METHODS

Source of inoculum for development of pocket rot. During the summer of 1990, several surveys of commercial table beet fields in central New York (Ontario and Yate counties) were conducted to identify sources of inocula as well as to determine the incidence of pocket rot, root rot, and wire stem diseases incited by *R. solani*. Ten 1-m long locations in each field were chosen at random, and the number of infected plants was counted. Disease incidence was determined on three different dates during the summer of 1990 (25 June, 16 July, and 3 August). In late summer, the number and length of individual foci of infected plants were determined in two fields near Geneva to assess the severity of pocket rot. Individual foci were identified and measured in two directions (90 m in length) by walking into and out of the field. Several visits to the fields were made to determine the presence of the teleomorph of *R. solani* and its contribution to the epidemiology of pocket rot.

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Efficiency of inoculum sources for disease development. Three inoculum sources were compared to develop an efficient inoculation procedure for disease development under field conditions: 1) mycelial suspension, 2) colonized beet seeds, and 3) pasteurized soil infested with *R. solani*. Three highly virulent isolates of *R. solani* AG-2-2—513, 521, and 655—were grown in a liquid medium containing 10 g of peptone, 15 g of dextrose, 0.5 g of $KHPO_4$, and 0.25 g of $MgSO_4$ per liter of water (15). After 7 days of incubation at room temperature, the mycelial mats were removed by filtering, washed several times with distilled water, and weighed. The mycelial mats were blended for 2–3 min with water and ice cubes, and the final suspension was adjusted to a concentration of 1 g of mycelium per 100 ml of distilled water. Autoclaved table beet seeds were colonized with the same isolates of *R. solani* by placing the seeds on cultures of the isolates growing on acidified potato-dextrose agar (APDA) and incubating at 28 C for 7 days. Colonized seeds were removed and bulked in equal numbers. The pasteurized soil inoculum of *R. solani* was prepared by growing isolates individually in the soil-potato medium (9). Equal portions of the inocula preparations of the three isolates were first mixed together and then added to potting soil at a rate of 2% (v/v).

Experimental design and inoculation procedure. The table beet cultivar Ruby Queen was used throughout this study. The tests were established in a field that had been previously planted to table beets with naturally occurring diseases over 5 yr. Each plot consisted of two rows 5 m long and 0.9 m apart. Individual plots were separated on each side by a border row and 2-m row areas between the blocks. Inoculum sources were applied to the center 1 m of the plot rows when the plants were 6 wk old. In 1991, mycelial suspension (500 ml), colonized beet seeds (50 ml), and infested pasteurized soil (100 ml) were spread onto the lower petioles and crown tissues of the plants in appropriate treatment plots. An additional treatment was set up by inoculating plants with the pasteurized soil infested with *R. solani* and then covering the inoculated tissues with field soil to simulate current cultivation practices. Control plants were sprayed with 500 ml of water. A randomized complete block design with eight replications was used for this study. In 1992, the three inoculum sources were compared with and without covering the inoculated areas with field soil. The experiment consisted of 2×4 (with and without covering with field soil $\times$ the three inoculum sources of *R. solani* and the uninoculated check) factorial treatments arranged in a randomized complete block design with five replications. In both years, overhead irrigation was applied shortly after inoculation to all the plots in order to create conditions favorable for infection of beets with *R. solani* and for the development of pocket rot.

Disease evaluation. The incidence and severity of pocket rot were determined 2, 4, and 6 wk after inoculation. Incidence of pocket rot was expressed as the number of infected plants in each plot. Disease severity was rated on a scale of 1–9 (15), where 1 = healthy plants with no visible disease symptoms and 9 = 100% of tissues affected. To assess the effect of the different inocula on the spread of pocket rot, infected plants outside the 1-m inoculated area were also counted and rated. Disease incidence and severity data were subjected to analysis of variance (ANOVA) using SAS (SAS Institute, Cary, NC) for each date of evaluation. ANOVA was also performed for the slopes of disease incidence and severity regression curves calculated according to the progression of pocket rot after inoculation. Inocula treatments were also partitioned to give single degree of freedom orthogonal comparisons. Data on spread of pocket rot were analyzed similarly, but ANOVA was not performed for the slopes of disease incidence and severity curves.

Effect of moisture on infection by *R. solani*. The development of *R. solani* on table beet plants incubated in mist chambers or covered/uncovered with plastic bags in the greenhouse was compared. Six-week-old plants of the cultivar Ruby Queen were inoculated with a mycelial suspension (1 g/100 ml) of *R. solani* AG-2-2 isolates 513 and 521 and AG-4 isolate 518 originally obtained from naturally infected table beets. The experiment consisted of 3×3 (three incubation methods $\times$ three isolates of *R. solani*) factorial treatments arranged in a randomized complete block design with five replications. Each replicate consisted of 12–20 plants growing in 10-cm-diameter pots filled with pasteurized soil/sand mixture. Inoculated plants were divided into three groups: 1) plants were maintained on a greenhouse bench (22–28 C) and watered daily as needed; 2) plants were enclosed in plastic bags, pots were placed on clay saucers with water, and plants were maintained in the greenhouse (22–28 C) for 72 hr; and 3) plants were incubated in a continuous misting chamber at 28 C for 72 hr. Severity of infection was recorded on a scale of 1 (no visible symptoms) to 9 (100% of tissue surfaces affected) (15). ANOVA was performed on the data. Incubation method was partitioned to give single degree of freedom orthogonal comparisons.

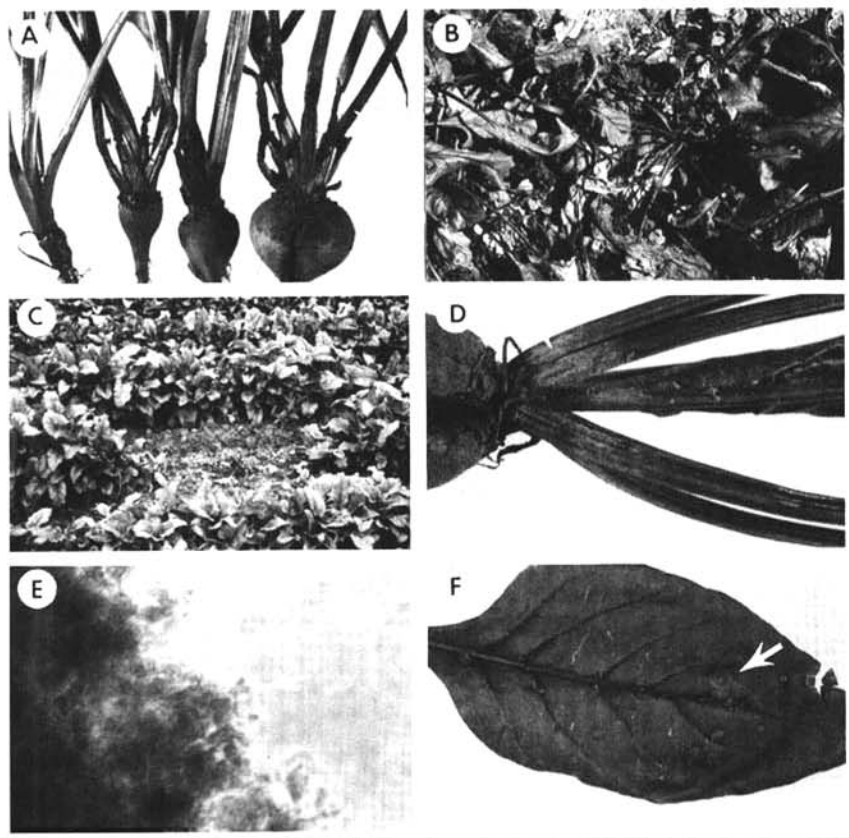

Fig. 1. Symptoms and signs of foliar blight and root rot caused by *Thanatephorus cucumeris* (anamorph: *Rhizoctonia solani*) on table beets: **(A)** Typical cankers on petioles and lesions on upper root tissues (arrow). **(B)** Infected plants forming a pocket. **(C)** Death of infected plants resulting in formation of open areas (pockets or foci) within planting rows. **(D)** Hymenia of *T. cucumeris* on surface of lower petiole tissues (arrow). **(E)** Basidia and basidiospores of *T. cucumeris* (arrow). **(F)** Lesions typical of those caused by basidiospores on leaf tissue (arrow).

RESULTS

Source of inoculum for development of pocket rot. Infection of table beets by *R. solani* in commercial production fields, including the development of pocket rot, increased over the 1990 growing season. For example, the percentages of infected plants in one field showing pocket rot symptoms (foci) were 6.9, 20.4, and 37.1 on 25 June, 16 July, and 3 August, respectively. Root rot incidence was also high during this growing season. The incidence of wire stem at the beginning of the season (25 June) was 5.8% and the incidence of root rot on 16 July and 3 August was 29 and 44%, respectively. Weather conditions during the summer of 1990 in New York State were especially rainy and relatively cooler than normal, which favored a high incidence and severity of pocket rot in most of the commercial table beet fields in central New York. For example, the maximum/minimum air temperature during June, July, August, and September averaged 24.8/13.6, 26.1/15.9, 24.6/15.8, and 20.8/10.8 C, respectively, while total precipitation was 4.3, 12.2, 6.6, and 7.7 cm, respectively.

Initial symptoms of infection of table beets by *R. solani* were brown to black cankers on petioles, especially near the point of attachment to the crown (Fig. 1A). Infections on petioles and crown tissues may progress down into the upper tissues of the fleshy beet roots and also to the side areas of roots. Progression of the disease in the field leads to the death of adjacent plants in each infection locus, resulting in open areas (pockets or foci) within the planting row (Fig. 1B and C). The length of individual foci of infected plants in two different fields in central New York ranged from 70 to 157.1 cm (Table 1), and the number of pockets observed ranged from seven to 37 in 90 m of row.

Hymenia of *T. cucumeris* were observed on infected table beets in different fields during late summer of 1990 and appeared as a thin, dusty growth on the crown and lower part of the petiole tissues (Fig. 1D). The color of the hymenia on beet tissues ranged from white to light brown. Basidia and basidiospores from preparations of the hymenia were observed under the microscope (Fig. 1E). The subcylindrical basidia were 13–15 μm long and 9–10 μm wide. There were four sterigmata per basidium; the sterigmata were straight and 12–14 μm long and 0.5–2 μm wide. There were usually four basidiospores; these were hyaline, ovate, and 7–8 μm long and 4-5 μm wide. All measurements are based on 15 observations. The measurements of basidia, sterigmata, and basidiospores were within those reported for *T. cucumeris* (18). Small circular lesions (3–6 mm in diameter) probably caused by basidiospores of *T. cucumeris* were observed on leaves of plants showing the hymenia (Fig. 1F) as well as on nearby plants lacking hymenia. These lesions were mostly restricted and light brown. Isolations made from lesions on APDA always yielded *R. solani*. Attempts to induce the teleomorph of *R. solani* (*T. cucumeris*) on 2% V8 juice agar media (5) were unsuccessful. It was also difficult to reproduce lesions on leaves directly using basidiospore inoculum collected from naturally infected plants. The teleomorph of *R. solani* was not seen on table beets during the summer of 1991 but it was observed in 1992 in two different commercial beet fields near Geneva.

Efficiency of inoculum sources for disease development. Highly significant differences ($P < 0.01$) in the incidence (number of infected plants per meter of row) and disease severity ratings of pocket rot were evident among the three inoculum sources of *R. solani* at all evaluation

Table 1. Average number and length of individual pockets of infected plants (foci) caused by *Rhizoctonia solani* in two commercial table beet fields in central New York State[a]

	Average number and length of foci			
	Sample 1		Sample 2	
Field	No.	Length (cm)	No.	Length (cm)
A	7	157.1	10	132.5
B	29	98.3	37	70.0

[a] Disease evaluations were made on 3 August 1990. Individual foci in about 90 m of rows were examined and measured in two opposite directions (sample 1 and 2).

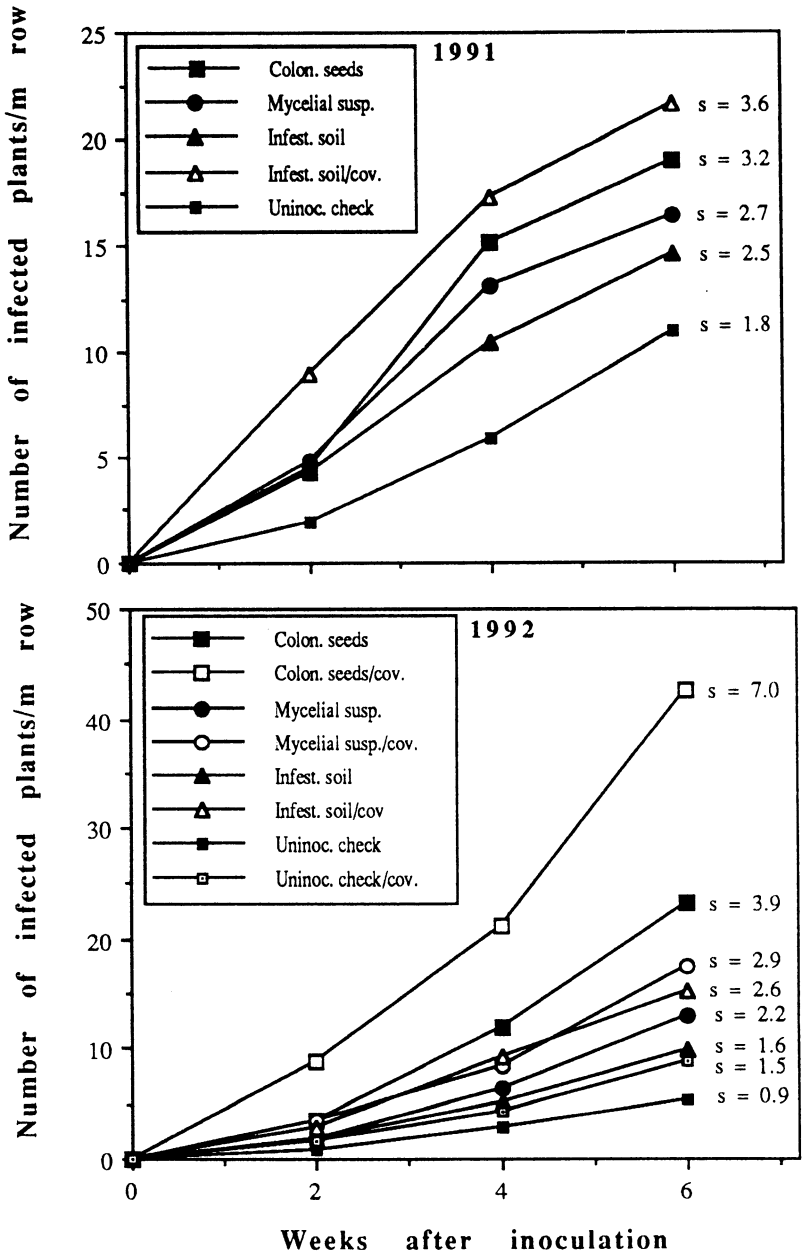

Fig. 2. Pocket rot incidence (number of infected plants per meter) and slope values (s) resulting from different inoculum sources of *Rhizoctonia solani* under field conditions in 1991 and 1992.

dates during 1991 and in 1992 (Figs. 2 and 3, Table 2). Disease also developed on plants in the uninoculated control treatment due to natural infections.

Disease incidence and severity increased over time in all treatments (Fig. 2, Table 2). The colonized table beet seeds resulted in a greater incidence and severity of disease in 1991 than did the infested soil and mycelial suspension, although these were also effective (Figs. 2 and 3, Table 2). However, covering the infested-soil inoculum preparation of *R. solani* with field soil resulted in the highest incidence and severity of pocket rot at each evaluation time (Figs. 2 and 3, Table 2). The ANOVA and orthogonal contrast of the slopes of the disease progress curves showed significant differences among the treatments over all the evaluation dates (Fig. 2, Table 2). The slopes of the regression lines for the infested soil inoculum preparation covered with field soil and those of colonized seed treatments were not significantly different (r = 3.6 and 3.2, respectively) in 1991. Disease also increased at a high rate on plants inoculated with the mycelial suspension (r = 2.7). Significant differences were also seen among the treatments for severity of pocket rot (Fig. 3, Table 2) in 1991. Disease severity increased over time at a higher rate on plants inoculated with the infested soil preparation covered with field soil (r = 1.1) than with the colonized seeds and mycelial suspension inoculum sources, which also exhibited a high rate of pocket rot development (r = 0.9 and 0.9, respectively).

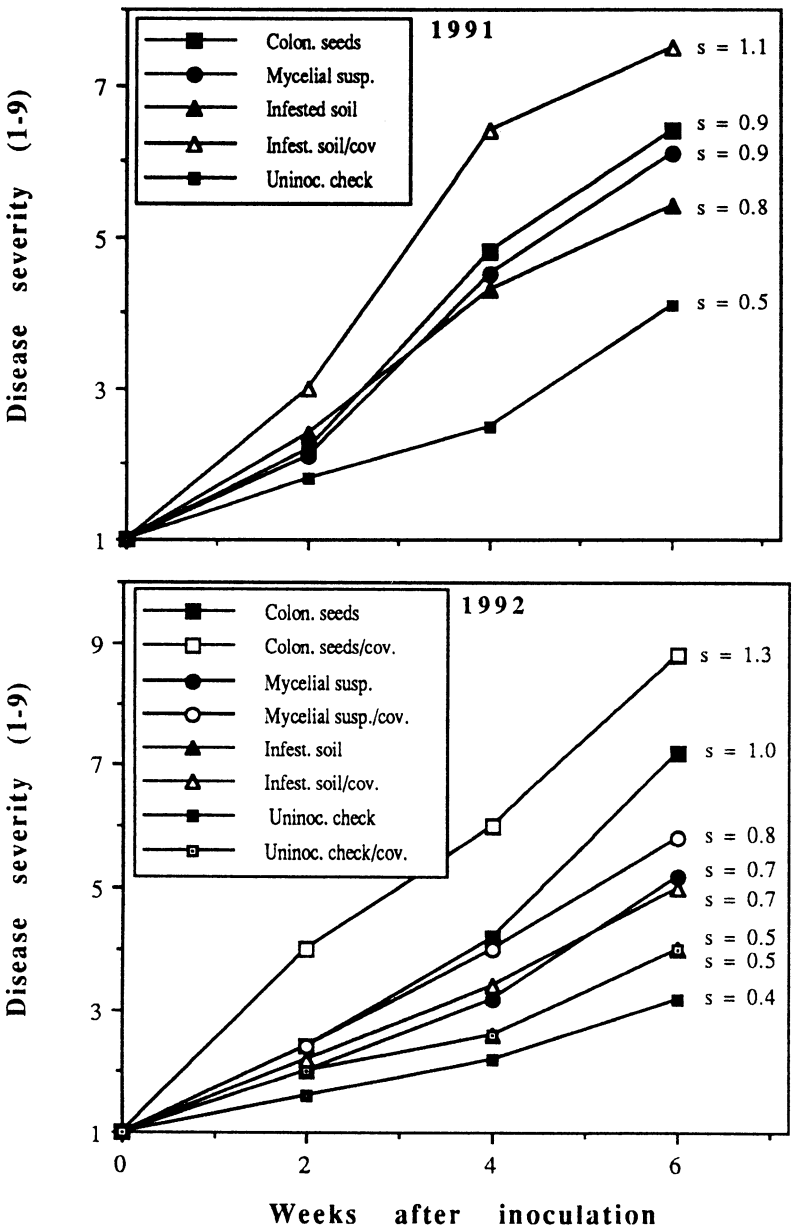

Fig. 3. Pocket rot severity rated on a scale of 1 (no visible symptoms) to 9 (100% of tissue affected) and slope values (s) resulting from different inoculum sources of *Rhizoctonia solani* under field conditions in 1991 and 1992.

In 1992, the most effective inoculum preparation for pocket rot development consisted of *Rhizoctonia*-colonized table beet seeds (Figs. 2 and 3, Table 2). For all inoculum preparations, covering the inoculated tissues with field soil resulted in higher incidence and severity of disease (Figs. 2 and 3, Table 2). Thus, disease development was highest on plants inoculated with the colonized seeds and covered with field soil. The *Rhizoctonia*-infested soil covered with field soil was not as effective as in 1991 (Figs. 2 and 3, Table 2). Single degree of freedom orthogonal contrast of the slopes of pocket rot incidence in 1992 also detected significant differences among the different inoculum sources (Table 2). The colonized seeds covered with soil resulted in higher incidence (r = 7.0) and severity (r = 1.3) than did the other treatments. Pocket rot infections also developed on the uninoculated control plants covered and uncovered with field soil (Figs. 2 and 3).

Effect of inoculum sources on spread of pocket rot. In both years, only a limited number of beet plants outside the 1-m inoculated section showed pocket rot symptoms 2 wk after inoculation, regardless of the inoculum source of *R. solani*. Conversely, there were significant differences in the linear spread of pocket rot (incidence and severity) among the inoculum sources at 4 and 6 wk after inoculation (Table 3). Disease incidence and severity increased over time in all treatments (Table 3). In 1991, the spread of the pathogen from the inoculated loci was similar for the three preparations. However, the greatest spread of the pathogen occurred in plots inoculated with *R. solani*-infested soil preparation and covered with field soil. An average of 16.8 consecutive plants outside the inoculated area were infected after 6 wk. The average pocket rot severity rating was 6.1, the highest among the treatments. In 1992, development of the disease outside the inoculated areas was lower compared to 1991. Spread and severity of pocket rot was greatest on plants inoculated with colonized beet seeds and was further increased with a cover of natural field soil (Table 3).

Effect of moisture on infection of table beets by *R. solani*. Table beet seedlings inoculated with *R. solani* exhibited symptoms of infection after 72 hr of incubation in the mist chamber or when covered with plastic bags in the greenhouse (Table 4). Infection was restricted on inoculated plants that were left on the bench without a cover. Severity of infection was higher on plants incubated in the mist chamber (Table 4). The three isolates of *R. solani* used in this test were able to infect table beets and reacted similarly, especially under the two incubation conditions that increased soil moisture and relative humidity. Across the incubation treatments, *R. solani* AG-

2-2 isolate 513 was the most virulent to table beets and AG-4 isolate 518 was the least virulent (*data not shown*).

DISCUSSION

The observation of hymenia of *T. cucumeris* on table beets under field conditions may implicate the involvement of basidiospores of *T. cucumeris* in the development of pocket rot epidemics in New York, although we have no direct evidence to support this. Basidiospores are an efficient source of inoculum, as they are produced in large numbers and subsequently disseminated by air. Basidiospores of *T. cucumeris* were reported to be disseminated for up to 30 m in sugar beets (10). Foliar blight infections on sugar beet in Japan were shown to be caused by isolates of two AG groups of *R. solani*. Infections caused by AG-1-IB and AG-2-2-IV isolates were initiated principally by mycelium and basidiospores, respectively (13). The incidence and severity of web blight on beans caused by *T. cucumeris* in the tropics is dramatic when the basidial state of the pathogen is present because the progression of the disease becomes polycyclic (4,7).

The small circular lesions believed to be caused by basidiospores of *T. cucumeris* on leaves of table beets resembled the lesions caused by basidiospores of *T. cucumeris* on sugar beet (11) and were somewhat similar to those caused by *Cercospora beticola* Sacc. Thus, it is possible that lesions caused by basidiospores of *T. cucumeris* had occurred before on table beets but may have been confused with those caused by *C. beticola*. In addition, lesions on leaves were also observed when 6-wk-old table beets were inoculated with a suspension of small mycelial fragments (1 g/100 ml) and incubated in a mist chamber for 72 hr (*data not shown*). This suggests that mycelium could also be involved in foliar blight on table beets.

The teleomorph of *R. solani* was not found on table beets in 1991 and occurred at lower frequency in 1992. The summer of 1991 was unusually dry and did not provide favorable conditions for the development of the perfect state. In 1992, rainfall was excessive and of long durations, which did not allow normal

Table 2. Orthogonal comparisons of the incidence and severity of pocket rot and the slope of the disease progress curves resulting from different inocula of *Rhizoctonia solani* under field conditions during 1991 and 1992[a]

Single df orthogonal comparison	Incidence (mean square)				Severity (mean square)[b]			
	2 wk	4 wk	6 wk	Slope	2 wk	4 wk	6 wk	Slope
1991								
Inocula								
Inoculated vs. uninoculated	86.3 **[c]	417.6 ***	302.5 ***	8.9 ***	3.0 *	40.0 ***	33.8 ***	1.2 ***
Mycelium + seeds vs. soil	31.0 *	0.6 ns	2.0 ns	0.1 ns	2.5 +	3.8 +	0.4 ns	0.0 ns
Mycelium vs. seeds	0.3 ns	17.0 ns	25.0 ns	0.8 ns	0.0 ns	0.1 ns	0.3 ns	0.0 ns
Soil covered vs. uncovered	83.3 **	182.3 *	196.0 **	13.3 ***	1.3 ns	17.0 ***	17.0 ***	1.7 ***
1992								
Inocula								
Inoculated vs. uninoculated	45.6 ***	346.8 ***	1283.8 ***	35.6 ***	3.7 ***	16.9 ***	43.2 ***	1.2 ***
Mycelium + soil vs. seeds	92.5 ***	576.6 ***	2406.7 ***	65.3 ***	7.4 ***	21.6 ***	60.0 ***	1.6 ***
Mycelium vs. soil	0.3 ns	0.5 ns	36.5 ns	0.8 ns	0.1 ns	1.8 +	5.0 *	0.2 *
Covering × inocula								
Inoculated vs. uninoculated	5.2 +	23.4 +	74.4 +	2.0 +	0.2 ns	1.0 ns	0.1 ns	0.0 ns
Mycelium + soil vs. seeds	26.0 ***	62.0 **	345.6 ***	8.6 ***	2.8 ***	1.7 ns	1.1 ns	0.0 ns
Mycelium vs. soil	0.6 ns	5.0 ns	1.3 ns	0.1 ns	0.1 ns	0.0 ns	0.2 ns	0.0 ns

[a]Inocula treatments were partitioned to give single degree of freedom orthogonal comparisons. In 1991, mycelial suspension, colonized seeds, and infested soil were tested using a randomized complete block design with eight replications. In 1992, the experiment consisted of 2 × 4 (with and without covering with field soil × three inoculum sources and uninoculated check) factorial treatments arranged in a randomized complete block design with five replications.
[b]Rated on a scale of 1 = healthy plants with no visible symptoms to 9 = 100% of tissues affected.
[c]Statistical significance at *** = $P \leq 0.001$, ** = $P \leq 0.01$, * = $P \leq 0.05$, and + = $P \leq 0.10$; ns = no significant difference.

Table 3. Orthogonal comparisons of the incidence and severity of pocket rot outside the areas inoculated with different preparations of *Rhizoctonia solani* during 1991 and 1992[a]

Single df orthogonal comparison	Incidence (mean square)			Severity (mean square)[b]		
	2 wk	4 wk	6 wk	2 wk	4 wk	6 wk
1991						
Inocula						
Inoculated vs. uninoculated	0.1 ns[c]	54.6 ***	470.9 ***	0.0 ns	1.8 +	34.8 ***
Mycelium + seeds vs. soil	0.1 ns	46.3 **	46.3 +	0.0 ns	3.1 *	5.0 *
Mycelium vs. seeds	1.3 +	0.4 ns	16.0 ns	0.1 ns	0.0 ns	0.3 ns
Soil covered vs. uncovered	0.6 ns	72.3 ***	199.5 ***	0.0 ns	1.9 +	17.2 ***
1992						
Inocula						
Inoculated vs. uninoculated	3.0 **	14.7 ***	75.2 ***	2.1 **	7.5 ***	43.2 ***
Mycelium + soil vs. seeds	4.8 **	7.4 **	88.8 ***	1.1 *	3.8 **	29.4 ***
Mycelium vs. soil	0.1 ns	2.5 +	6.1 ns	0.0 ns	0.5 ns	0.8 ns
Covering × inocula						
Inoculated vs. uninoculated	0.0 ns	0.0 ns	1.4 ns	0.0 ns	0.0 ns	0.1 ns
Mycelium + soil vs. seeds	0.4 ns	1.4 ns	14.0 *	0.1 ns	0.8 ns	6.7 *
Mycelium vs. soil	0.1 ns	0.5 ns	1.3 ns	0.2 ns	0.5 ns	0.2 ns

[a]Inocula treatments were partitioned to give single degree of freedom orthogonal comparisons. In 1991, mycelial suspension, colonized seeds, and infested soil were tested using a randomized complete block design with eight replications. In 1992, the experiment consisted of 2 × 4 (with and without covering with field soil × three inoculum sources and uninoculated check) factorial treatments arranged in a randomized complete block design with five replications.
[b]Rated on a scale of 1 = healthy plants with no visible symptoms to 9 = 100% of tissues affected.
[c]Statistical significance at *** = $P \leq 0.001$, ** = $P \leq 0.01$, * = $P \leq 0.05$, and + = $P \leq 0.10$; ns = no significant difference.

Table 4. Severity of foliar infections incited by *Rhizoctonia solani* that developed on inoculated plants incubated in a mist chamber or on a bench in the greenhouse with or without enclosure in plastic bags[a]

Single df orthogonal comparison	df	Mean square
Bags + mist vs. bench	1	256.71 ***[b]
Bags vs. mist	1	19.20 ***

[a] Incubation methods were partitioned to give single degree of freedom orthogonal comparisons. The experiment consisted of 3 × 3 (three incubation methods × three isolates of *R. solani*) factorial treatments arranged in a randomized complete block design with five replications.

[b] Statistical significance at $P \leq 0.001$.

cultivation of the crop. The lack or limitation of cultivation was associated with the low incidence of pocket rot and infrequent development of *T. cucumeris*. Inoculum applied to the lower petiole and crown areas of beets covered by field soil to simulate current cultivation practices generally resulted in consistent and severe disease and the greatest linear spread. The field soil protected the inoculum and allowed the fungus to spread through the soil and infect adjacent plants. Therefore, current cultivation practices in which large tractors are used at high speeds may be playing an important role in the development of pocket rot, since more infested soil is thrown on the petioles and crowns of the plants. However, it is also possible that seedlings infected by *R. solani* at an early stage produce secondary inoculum in the form of mycelium that infects adjacent plants and produces the sexual state and subsequently basidiospores that function as an alternate source of inoculum for foliar infections. The development of Rhizoctonia root rot on sugar beets was similarly shown to increase by soil depositions in and around the crown area of the plants (17,19). The development of Rhizoctonia crown rot of carrots was not changed significantly by land preparation and cultural practices (8).

The most effective inoculum preparation of *R. solani* for consistent and severe disease development in this study was the colonized table beet seeds. Similar inoculum preparations of *R. solani* (i.e., colonized whole grains of wheat, oat, barley, sorghum, or millet) have been reported in the literature to be highly effective in disease initiation and development (18). It appears that beet seeds and whole small grains are good sources of exogenous nutrients for the fungus (18). Under field conditions, the mycelium of *R. solani* may well be better protected in colonized seeds against drying and antagonistic microorganisms than are mycelial fragments either free or mixed in soil.

A leaf wetness period of 72 hr was sufficient for the establishment of *R. solani* on table beets. Incubation of inoculated plants in plastic bags and in a mist chamber resulted in severe infections. These experiments were conducted because of an initial failure to establish pocket rot in experimental fields with various artificial inoculation procedures. It was previously reported that the development of diseases incited by *Rhizoctonia* spp. requires free moisture or near 100% RH conditions (3). Many fungal plant pathogens are dependent upon the presence of free moisture on host tissues or high relative humidity conditions, especially during the early infection process (6). They may become less dependent upon moisture once they are able to obtain nutrients and water from the host (16). Moisture in the form of rain, irrigation water, or splashing rain plays an important role in the distribution and spread of sclerotia, infected plant debris, or basidiospores of *R. solani* (3). Rain-splashed water droplets are an important factor in the dissemination of *R. solani* causing web blight on beans in Costa Rica (7). According to Kotila (10), high humidity is required for the development of the basidial stage of *R. solani*, and the formation and release of basidiospores depend upon the availability of warm, humid weather conditions. High humidity is also required for germination of sclerotia in soil or in infected plant debris and for basidiospores (3). Pocket rot is always severe when long periods of wet conditions prevail during the growing season. Field inoculation experiments were followed by application of several overhead irrigations in this study for development and spread of pocket rot. The lower petiole and crown areas of table beets retain free water for long periods of time and thus create favorable conditions for development of *R. solani*.

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