Comparison of Tuberborne and Soilborne Inoculum in the Rhizoctonia Disease of Potato

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ABSTRACT

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Field and greenhouse testing confirmed that both tuberborne and soilborne inocula of *Rhizoctonia solani* were important in the development of the Rhizoctonia disease of potato. Five criteria were included in the disease rating: percentage of sprout emergence, stem lesions, stolon lesions, pruned stolons, and percent usable tubers. Tuberborne inoculum primarily affected sprout emergence and soilborne inoculum generally contributed to stolon damage. Lack of visible *R. solani* sclerotia on tubers was not sufficient to indicate absence of the pathogen. Control of tuberborne inoculum is an essential part of an integrated program for control of *R. solani* on potatoes.

The Rhizoctonia disease (caused by *Rhizoctonia solani* Kuhn, AG-3) of potato (*Solanum tuberosum* L.) can be very severe in Maine (7) and eastern Canada (1). Crop rotations for its control have been only moderately successful (6,9). Some research workers have suggested that the ability of *R. solani* to live indefinitely in the soil or on debris and weeds may be responsible for the marginal control provided by rotation (3,6,9,13), but others do not agree (14,15). Several workers suggested that tuberborne inoculum may play the major role in the development of the *R. solani* disease (10,12,14). A recent report (2) suggests that a large percentage of the tuberborne sclerotial strains are pathogenic. This study was conducted to determine the relative importance of soilborne versus tuberborne inoculum on the Rhizoctonia disease of potatoes in Maine.

MATERIALS AND METHODS

Field experiments were conducted in Presque Isle, ME, in 1975 and 1976. The test plot had been planted with potatoes and artificially infested with *R. solani* every summer since 1972. Potato cultivars Katahdin, Kennebec, and Russet Burbank were used in all experiments. The plots were planted 3 June in 1975 and 10 June in 1976. Prior to planting, soil populations of *R. solani* were estimated with a tissue colonization technique (11). All plots received 10-10-10 fertilizer (134.4 kg N/ha), disulfoton (16.8 kg/ha) for insect control, and dinoseb (5.7 L/ha) for weed control. Seed pieces were spaced 30 cm in the rows.

In both years the experimental design of the test was a split-plot with four replications, three cultivars, and four treatments. Each individual plot consisted of 20 hills and there were 48 plots in the entire test. The four treatments were: noninfested soil, clean seed pieces (control); infested soil, clean seed pieces; noninfested soil, infested seed pieces; and infested soil, infested seed pieces. Inoculum was prepared by growing *R. solani* (isolated from potato soil in Presque Isle, ME, in 1972) on autoclaved buckwheat straw pieces 2 cm long for 5 wk prior to planting. In all plots with infested soil, three pieces of the buckwheat straw inoculum were placed adjacent to each seedpiece in the row prior to covering. Potato seed pieces classified as clean had no visible sclerotia on the tuber surface. Infested seed pieces had at least one visible sclerotium within every 2-cm square of tuber surface.

Plants were field evaluated for *R. solani* damage at 4, 6, and 10 wk after planting, and tubers were rated after harvest as described (8). During the growing season, potato plants were treated as necessary with foliar applications of mancozeb (1.6 kg/ha) and methyl demeton (0.95 L/ha) for disease and insect control.

Greenhouse experiments were conducted in December of 1975, 1976, and 1977 in Orono, ME. Field soil without a history of planting to potatoes was selected and placed in 30-cm diameter plastic pots in the greenhouse.

The fungus was grown in shake culture (500-ml flask containing 250 ml of broth, 25 C, in available light) for 3 wk in potato dextrose broth. After this period, 10 cultures were pooled, macerated in a blender and diluted 10× with distilled water. An aliquot (100 ml) of this slurry was placed in each of the pots designated for infested soil. After the initial infestation of soil in 1975 there were no additional infestations in 1976 or 1977. Prior to infestation, the indigenous *R. solani* population was estimated as previously described.

The same three potato cultivars were used in the greenhouse pot experiments as in the field experiments. Four pots of each cultivar were planted for each of six treatments, a total of 72 pots. The six treatments were: clean seed pieces, noninfested soil; infested seed pieces, noninfested soil; clean seed pieces, infested soil; infested seed pieces, infested soil; infested seed pieces with wash treatment, noninfested soil; and infested seed pieces with wash treatment, infested soil.

Infested seed pieces were selected from field grown tubers in 1975 as described in the field tests. In two of the treatments the infested seed pieces were washed in cold running water for 5 min to reduce fungal infestation in adhering soil or hyphal fragments on the tuber surface. For the clean seed piece treatments, tubers without visible sclerotia were dipped for 1 min in 1.05% NaOCl prior to planting. One seed piece was planted in the center of each pot at a depth of 10 cm. Prior to planting 3.5 g of 10-10-10 (N-P-K) fertilizer was incorporated into the soil (~ 134.4 kg N/ha). The emergence data were recorded as the percentage of sprouts pruned by *R. solani*, and the plants were harvested at 120 days after planting. At this time, disease severity on stems, stolons, and tubers was evaluated as previously described and a final disease index was calculated (7).

Within 2 wk after harvest, all pots were sown with Japanese millet (*Echinochloa crusgalli* L.). This crop was maintained until the following November when it was turned under. After 1 mo of fallow, potatoes again were planted in the pots. In 1976 and 1977, the same pots were used for the same cultivars and treatments as in 1975. The following changes were made in the treatments: the infested seed pieces were replaced by tubers that had been harvested from the identical pot in the previous year (required the number of sclerotia per unit of surface area were the same); the infested soil

TABLE 1. Influence of infested soil and/or infested seed pieces on the severity of Rhizoctonia disease of potato in the field

Treatment	Katahdin		Kennebec		Russet Burbank	
	1975	1976	1975	1976	1975	1976
Clean seed pieces						
Noninfested soil	1.8[a]	2.3	2.0	2.5	2.0	2.3
Infested soil	3.3	3.6	2.9	3.7	3.4	3.1
Infested seed pieces						
Noninfested soil	3.2	5.3	3.9	5.9	3.6	6.1
Infested soil	5.6	7.2	6.7	7.6	7.2	9.0
Bayes' LSD ($P=0.05$)	1.3	1.1	1.3	1.1	1.3	1.1

[a]Ratings based on a scale of 1–10 in which 1 indicates no disease and 10 signifies maximum susceptibility. Ratings reflect a composite of sprout pruning, stem lesions, stolon lesions and pruning, and usable yield.

TABLE 2. Influence of infested soil and/or infested seed pieces on the severity of Rhizoctonia disease of potato in the greenhouse

Treatment	Katahdin			Kennebec			Russet Burbank		
	1975	1976	1977	1975	1976	1977	1975	1976	1977
Clean seed pieces									
Noninfested soil	1.0[a]	1.0	1.3	1.5	1.2	1.5	1.0	1.2	1.2
Infested soil	2.6	1.2	2.0	4.7	1.8	2.0	3.2	2.0	3.2
Infested seed pieces									
Noninfested soil	2.3	3.6	3.2	5.6	4.9	3.7	5.1	5.6	4.7
Infested soil	5.1	4.3	6.3	8.1	4.4	7.1	6.7	5.9	7.2
Washed infested seed pieces									
Noninfested soil	2.1	2.2	2.1	3.6	2.7	2.2	1.3	2.6	2.9
Infested soil	2.5	3.1	3.2	5.5	4.7	5.1	2.7	3.4	4.9
Bayes' LSD ($P=0.05$)	1.1	0.7	1.0	1.1	0.7	1.0	1.1	0.7	1.0

[a]Ratings based on a scale of 1–10 in which 1 indicates no disease and 10 signifies maximum susceptibility. Ratings reflect a composite of stem lesions, stolon lesions and pruning, and usable yield.

TABLE 3. Inoculum source effects of sprout emergence and stolon damage in Kennebec potato plants in field plots in 1975 and 1976

Treatment	Nonemergence (%)		Stolons pruned (%)		Stolons with lesions (%)	
	1975	1976	1975	1976	1975	1976
Clean seed pieces						
Noninfested soil	2	3	1	3	5	7
Infested soil	5	6	23	19	17	39
Infested seed pieces						
Noninfested soil	36	56	6	8	8	9
Infested soil	38	62	25	29	22	35
Bayes' LSD ($P=0.05$)	5.12	6.23	7.61	6.42	5.62	4.78

TABLE 4. Comparison between sprout emergence and stolon damage in relation to inoculum source in greenhouse tests of potato (cultivar Kennebec) plants

Treatment	Nonemergence (%)			Stolons pruned (%)			Stolons with lesions (%)		
	1975	1976	1977	1975	1976	1977	1975	1976	1977
Clean seed pieces									
Noninfested soil	0	0	0	2	3	2	2	1	0
Infested soil	2	0	2	26	21	18	39	20	23
Infested seed pieces									
Noninfested soil	14	16	15	4	9	10	7	10	9
Infested soil	22	23	31	29	24	32	36	22	20
Washed infested seed pieces									
Noninfested soil	6	10	10	5	3	2	8	4	5
Infested soil	20	24	21	20	20	13	21	20	12
Bayes' LSD ($P=0.05$)	6.93	6.71	7.03	4.94	4.82	4.60	5.16	4.93	4.87

was not reinfested; and the fertilization rate was lowered to 100 kg N/ha. Disease ratings and *R. solani* soil population estimations were conducted as in 1975.

RESULTS

The indigenous soilborne *R. solani* populations in the 1975 and 1976 field trials provided 2 and 7% beet seed colonizations, respectively. The final disease ratings calculated for this same period are presented in Table 1. The disease was most severe when both soilborne and tuberborne inoculum were present. In 1975, there was no significant difference between the ratings for plots with soilborne or tuberborne inoculum. However, in 1976 the ratings were significantly higher for plots with infested seed pieces than for those with infested soil.

The greenhouse trials conducted in 1975, 1976, and 1977 also indicated that treatments with both sources of inoculum were most severely diseased in all three years (Table 2). The table beet seed colonization from the original soil in 1975 was 0% while both 1976 and 1977 soils provided a 3% tissue colonization following potato culture. With the exception of the 1975 data for cultivars Katahdin and Kennebec, the disease ratings were significantly higher when seed pieces were infested than when inoculum was added to the soil. In general, washing the seed pieces prior to planting significantly reduced the disease rating whether or not inoculum was added to the soil.

Tables 3 and 4 compare emergence data and stolon damage for the cultivar Kennebec in field and greenhouse tests, respectively. The concomitant effect of sprout pruning on emergence was most prevalent when the inoculum source was tuberborne. This same inoculum source produced the lowest percentage of stolon lesions and stolon pruning. In comparison, the addition of inoculum to soil provided a higher incidence of stolon damage and a lower percentage of nonemergence. Results were similar for Katahdin and Russet Burbank. The wash treatments in the greenhouse experiments generally provided an increase in plant emergence, but had little effect on stolon damage.

DISCUSSION

In evaluating the *Rhizoctonia* disease potential on potatoes, considerable emphasis must be placed on soil type and other environmental factors. This disease is often more severe in Maine (7) and eastern Canada (1) than in western potato growing regions (4,5,15). Potato crops in the western USA rarely exhibit the sprout pruning phase of the disease and the resultant reduction in emergence because of the sandy soil, minimal rainfall, and controlled sprinkler irrigation. In Maine, however, the generally wet and cool conditions after planting favor the pathogen more than the plant and many Maine soils also retain mositure which is conducive to early season disease development and damping off. The impact of these conditions required that nonemergence be weighted heavily in our rating scheme (8).

To control *R. solani* on potatoes, the inoculum source must be considered. From our results its appears that both soilborne and tuberborne inoculum are etiologically important. Although either source can contribute to all phases of the disease syndrome, the data suggest that tuberborne inoculum is more important in the early stages of the disease. As the sprouts emerge from the tuber, the inoculum is already present, adjacent to the sprout. The pathogen existing at low density in the soil may have to grow through the soil before reaching the sprout and emergence may occur before the pathogen is able to invade and prune the sprout unless it is on the plane of the infection court. Once the sprout has emerged from the soil, it is generally thought to be resistant to pruning, but aerial infection by the fungus produces stem cankers.

During the invasion of stolons especially those of the cultivar Kennebec, the stolons grow several centimeters from the mother tuber as they develop. The pathogen existing in the outer fringe of the soil volume occupied by stolons and roots may invade stolons more readily than the tuberborne inoculum. In part, this may explain why pentachloronitrobenzene applied to the soil reduces stolon damage on potatoes but has been ineffective in controlling the sprout pruning phase of the disease in Maine (J. A. Frank and S. S. Leach, *unpublished*).

Since both soilborne and tuberborne inoculum are involved in the disease development, it is important to control inocula from both sources in order to minimize disease incidence. A 2-yr potatoes-oats rotation effectively reduces *R. solani* populations in Maine (8). However, growers who follow acceptable rotation practices to reduce the soilborne inoculum often have planted infested seed pieces which negated the beneficial effects of crop rotation. Even if the environmental conditions were not favorable for disease development, the use of seed pieces from infested tubers effectively raised the inoculum level in the soil. Our data also indicate that the pathogen may be present on the tuber surface even though sclerotia are not visible, and that washing may eliminate some of the inoculum. In growing seasons with abundant rainfall late in the season sclerotia do not develop as extensively as in a dry season. The time interval between vine killing and harvest also determines the amount of sclerotium development on tubers. Therefore, seed piece disinfestation treatment should not be confined only to tubers with visible sclerotia. By eliminating tuberborne inoculum, a grower may improve the effectiveness of crop rotations and other controls which reduce the soilborne inoculum.

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