Survival of *Rhizoctonia solani* in Fallow Field Soil and Buried Sugarbeet Roots at Three Depths[1]

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ABSTRACT

Survival of *Rhizoctonia solani* AG-2-2 in infected sugarbeet root tissue and in soil adjacent to the roots at 5-, 10-, and 20-cm depths in fallow field soil was assayed bimonthly in two tests between June 1988-June 1989 and June 1989-June 1990. By August, percent recovery of the fungus from tissue declined 80 and 74% in 1988 and 1989, respectively. Thereafter, recovery was variable but generally continued to decline in both years. In the first test, *R. solani* was not recovered from root tissue buried 5-cm deep after 4 mo, but in the second test, tissue buried at 5 cm yielded the fungus throughout the year. For tissue buried 10 cm deep, the fungus survived without decline for 6 mo in 1988, but only for 4 mo in 1989. In 1988-89, the fungus was not recovered from tissue buried 20-cm deep after 2 and 4 mo, but was recovered at the 6- and 12-mo assays. In 1989-90, *R. solani* was recovered only at the 2- and 4-mo assays from tissue buried 20 cm deep.

[1]*Joint contribution of the Agricultural Research Service, USDA, and the Beet Sugar Development Foundation. This research was partially funded by the joint Western Sugar Company - Grower Research Committee. Mention of a trademark or proprietary product does not constitute a guarantee or warranty by the U.S. Department of Agriculture and does not imply approval to the exclusion of other products that also may be suitable.*

Population densities of *R. solani* were 1.6-2.0 colony-forming units (CFU) per gram of air-dry soil at the 2-mo assay in 1988 and 1989, respectively. Thereafter, population densities tended to decline over time in both years, reaching 0.5-0.7 CFU g^{-1} after 12 mo. All isolates from buried tissue and a 10% random sample of soil isolates were pathogenic in 3-mo-old sugarbeets in the greenhouse, except for one AG-4 isolate from soil. Decline in pathogen survival apparently was not related to precipitation or air and soil temperatures but may have been associated with the degradation of organic food base.

Additional Key Words: *Beta vulgaris*, Rhizoctonia root rot, *Thanatephorus cucumeris*.

Sugarbeets (*Beta vulgaris* L.) affected with root rot, induced by *Rhizoctonia solani* Kühn (teleomorph, *Thanatephorus cucumeris* (Frank) Donk), often are not removed from the field during harvest, because they are too small to be picked up by the harvester or severely rotted roots break off in the ground. Subsequently, the rotted tissue is buried at plow depth ($\leq$ 15 cm) during farming operations. *R. solani* surviving in rotted beet tissue is a potential source of inoculum for future sugarbeet crops or other crop hosts (LeClerg, 1934).

Knowledge on survival of the pathogen in infected sugarbeets over extended time periods and at different depths in the soil is limited. Boosalis and Scharen (1959) detected *R. solani* in nondefined debris particles washed from the upper 2-13 cm of soil 7 mo after harvest of a root rot-affected crop of sugarbeets; however, pathogenicity of their isolates was tested only on sugarbeet seedlings, and anastomosis groups (AG) were not determined. In the United States, at least, root rot of beets >2 mo old is induced mostly by AG-2-2 isolates (Ruppel, 1972; Herr and Roberts, 1980; Windels and Nabben, 1989), whereas seedling damping-off is caused mainly by AG-4 isolates (Ruppel, 1972; Herr and Roberts, 1980; Windels and Nabben, 1989). AG-1, -2-1, -3, and -5 isolates also can induce seedling disease of beets, but these AG types were less virulent to seedlings than AG-4 isolates (Windels and Nabben, 1989). In Ohio, Herr (1976) reported that survival of the pathogen in sugarbeets overwintered on the soil surface essentially was similar to that in roots buried at plow depth. Recovery from buried roots, however, was significantly lower in June compared with April, whereas recovery from roots on the soil surface increased slightly between spring and summer. Reduction in recovery of *R. solani* was attributed to an increased rate of decomposition of buried beet residues compared

to surface beet residues and substantiated the earlier results Townsend (1934) reported on survival of *R. solani* in buried and surface diseased lettuce residues. In Japan, Hyakumachi and Ui (1979) isolated *R. solani* from 80% of plant debris particles obtained at harvest (October) from soil around severely rotted sugarbeet roots; only 20% of debris particles yielded the fungus the following May. Neither Herr (1976) nor Hyakumachi and Ui (1979) reported pathogenicity tests or the AG type of their isolates.

Many studies on survival of *R. solani* in soil or plant debris have been conducted in the laboratory or greenhouse (Papavizas et al., 1975). Field tests mostly were performed in soils naturally high in organic matter and where there was ample moisture (Townsend, 1934; Herr, 1976; Hadar et al., 1982; Bell and Sumner, 1987), or soil and debris samples were obtained from areas where crops were, or recently had been, growing (Townsend, 1934; Papavizas et al., 1975; Herr, 1976; Roberts and Herr, 1979; Herr, 1987; Neate, 1987).

Little information is available on pathogen survival in western calcareous soils having minimal organic matter content, relatively high pH, and limited amounts of natural moisture. The objective of my study in Colorado was to determine bimonthly survival of *R. solani* AG-2-2 in diseased roots buried at three depths in fallow field soil and in soil surrounding the buried roots. Such information may help in determining management strategies for control of the disease.

MATERIALS AND METHODS

A field site, approximately 9 × 6 m, that had not been planted to sugarbeet for at least 7 yr was selected. The soil type was a clay loam with the following characteristics: pH 7.8, 3% organic matter, 36 mg kg^{-1} NO_3-N, 22 mg kg^{-1} P, 326 mg kg^{-1} K, 7.5 mg kg^{-1} Zn, 13 mg kg^{-1} Fe, 6 mg kg^{-1} Mn, 16 mg kg^{-1} Cu, and an electrolytic conductivity of 0.06 S m^{-1}. The site was prepared conventionally for planting, but no fertilizer, herbicides, or other pesticides were applied.

Sugarbeets (susceptible commercial hybrid MonoHy D-2) were grown individually in 15-cm-diameter clay pots of steam pasteurized soil in the greenhouse. When plants were 3-mo old, barley-grain inoculum (Ruppel et al., 1979) of *R. solani* (isolate R-9; AG-2-2) was placed next to the taproot about 2 cm below the soil surface on opposite sides of the root. After 1 mo, roots were harvested, washed, and split longitudinally through the developing extensive rot lesion, which comprised over 60% of the root tissue. Isolations on *Rhizoctonia*-selective medium (Ko & Hora, 1971) from lesions on each root yielded *R. solani* AG-2-2, as determined by AG-typing (Parmeter et al., 1969).

Each root half (approximately 60 cm^3) then was placed in an envelope made from polyethylene net having a mesh size of 950 × 700 μm and a diameter of 150 mm (Kartell Buchner funnel disks, Dynalab Corp., Rochester, NY).

Envelopes containing the diseased root halves were buried at 5-, 10- and 20-cm depths at the field site. There were six envelopes per depth to allow for six bimonthly harvests and assays for the pathogen. Burial positions were spaced equidistant from each other, and the experiment was arranged in a randomized complete block design with five replicates. The experiment was initiated in June 1988 and terminated in June 1989, then repeated in June 1989 through June 1990. The site was fallow and not irrigated, fertilized, or treated in any other way. Soil temperatures at the 5- and 20-cm depths were monitored daily with a two-channel Datapod, Model DP212 (Omnidata International, Logan, UT), and weekly air temperatures and precipitation records during the growing seasons were obtained from Colorado State University, Fort Collins.

Beginning 2 mo after burial and at bimonthly intervals, one randomly selected envelope per replicate at each depth was exhumed and the contents examined and assayed for *R. solani*. Ten pieces of root residue ($\leq$ 2 mm^2 each) from each envelope were placed on *Rhizoctonia*-selective medium (Ko and Hora, 1971) in each of three 9-cm-diameter petri dishes (30 subsamples). Residues were examined for *R. solani* after a 16-hr incubation period at room temperature, and hyphal-tip transfers were made to petri dishes of potato-dextrose agar (PDA). Identifications were based on cultural characteristics on PDA and by anastomosis with tester isolates of known AG types on water agar (Parmeter et al., 1969).

Soil samples taken from the immediate area surrounding the buried envelopes also were assayed for the fungus by means of a pellet soil-sampler (Henis et al., 1978) and the selective medium. There were three subsamples per replicate, with 15 100-mg soil pellets per dish. After 16 hr, hyphal-tip transfers were made from developing colonies to petri dishes of PDA, and identifications and AG typing were done as described previously. Results of the debris assay are expressed as percentage of subsamples yielding *R. solani*; data from the soil assays are expressed in colony-forming units (CFU) per gram of air-dried soil.

All isolates obtained from root debris (47) and 10% from surrounding soil (62) were tested for pathogenicity on 3-mo-old sugarbeets (susceptible breeding line FC 901) in the greenhouse. Barley-grain inocula were prepared (Ruppel et al., 1979) and inoculations made as described previously. Sugarbeet reactions to the isolates were recorded 30 days after inoculation.

RESULTS AND DISCUSSION

In both years, root halves buried at 5 cm still were intact after 2 mo (August), although almost completely blackened with rot. At 10 and 20 cm, root shape no longer was discernible, with only nondescript, blackish organic residue remaining. Root shape still was discernible at the 5-cm depth in October (4 mo), but by December, these root halves were mummified and beginning to disintegrate. The more rapid degradation of roots at the deeper depths possibly was due to greater retention of soil moisture following spring rains (Fig. 1); surface soil quickly dried from the effects of higher soil and air temperatures (Figs. 2A & B, 3A & B) and ample sunshine and wind. Bacteria at the deeper depths also may have contributed to root degradation; however, bacterial isolations were not made.

Recovery of *R. solani* AG-2-2 from root debris was quite variable between years (Fig. 2A & B); however, there was a general trend toward a rapid decrease in pathogen recovery within 2 mo and a slower but steady decline over the 330-day period each year. Assuming that initial root debris was 100% colonized by the pathogen at the onset of each test, percent decrease of subsamples yielding the fungus after 2-mo (August) was 80% at the 5- and 20-cm depths in 1988 and 74% at all depths in 1989 (Fig. 2A & B). The pathogen was not recovered in 10-cm-deep debris samples in August and October 1988, but was recovered in an unexplainably high percentage of subsamples in December and then not recovered again until June 1989. In 1989-90, no *Rhizoctonia* could be recovered after 4 mo (October) from the

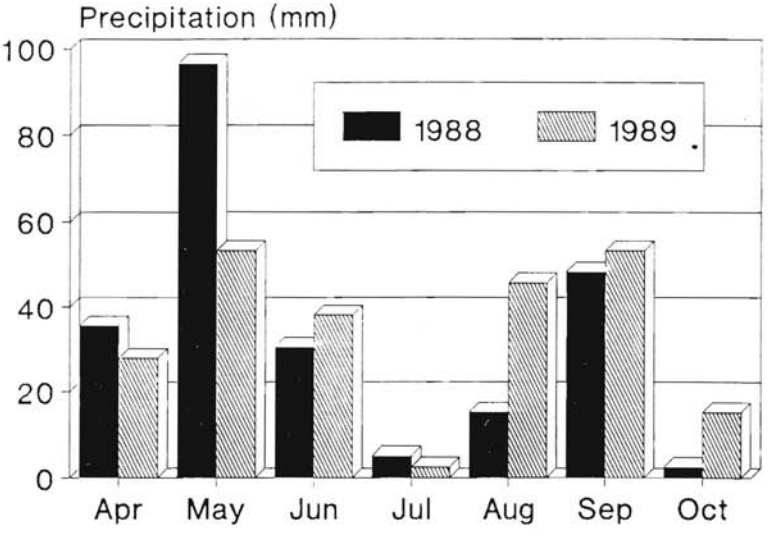

Figure 1. Total monthly rainfall in millimeters at Fort Collins, CO, from April through October 1988 and 1989.

10- and 20-cm samples, but recovery from debris buried 5-cm deep was obtained throughout the duration of the experiment. Variablity in pathogen recovery between years did not seem to be related to differences in precipitation (Fig. 1), air temperature (Fig. 3A & B), or soil temperature (Fig. 2A & B), although soil temperatures and mean maximal air temperatures were somewhat higher during the 1988

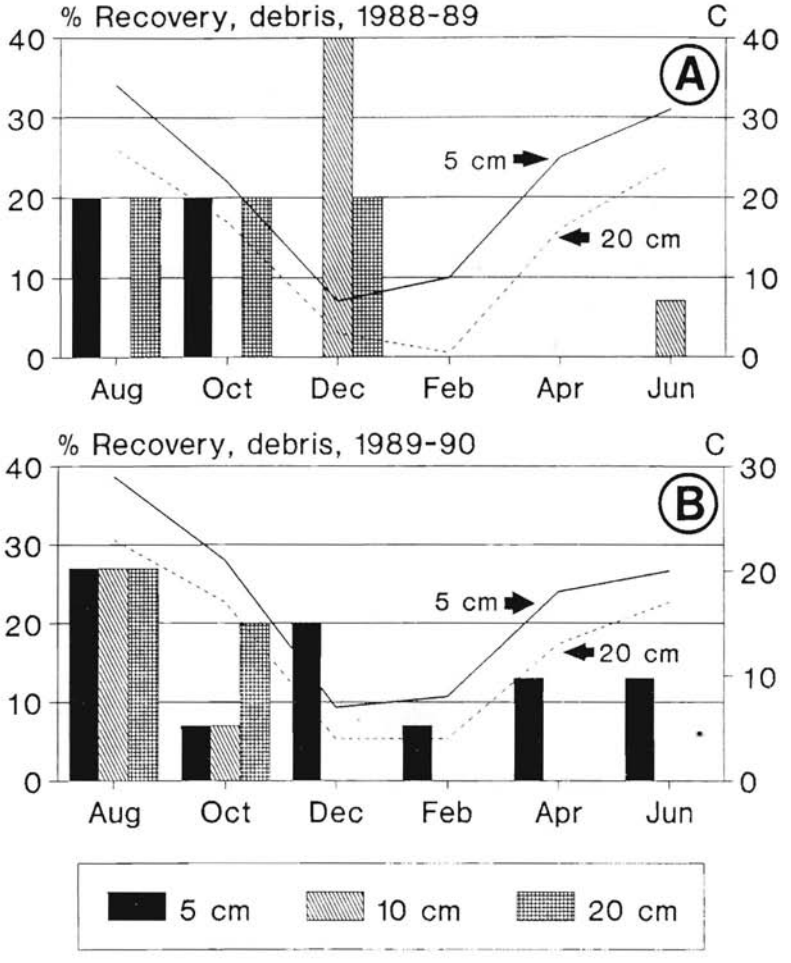

Figure 2. Bimonthly percent recovery of *Rhizoctonia solani* from sugarbeet root debris buried 5, 10, and 20 cm in a fallow field site at Fort Collins, CO; graph lines indicate mean monthly high soil temperatures at 5- and 20-cm depths. A. 1988-1989 samples. B. 1989-1990 samples.

growing season compared with 1989. The decline in recovery of *R. solani* from overwintered debris agrees with results reported by Hyakumachi and Ui (1979) in Japan. Our initiation of the experimental trials in June rather than the normal harvest period (October) precludes direct comparison with survival of *R. solani* in rotted beets buried after harvest (Herr, 1976). However, the initial phase of survival reported here represents the decomposition effect that occurs

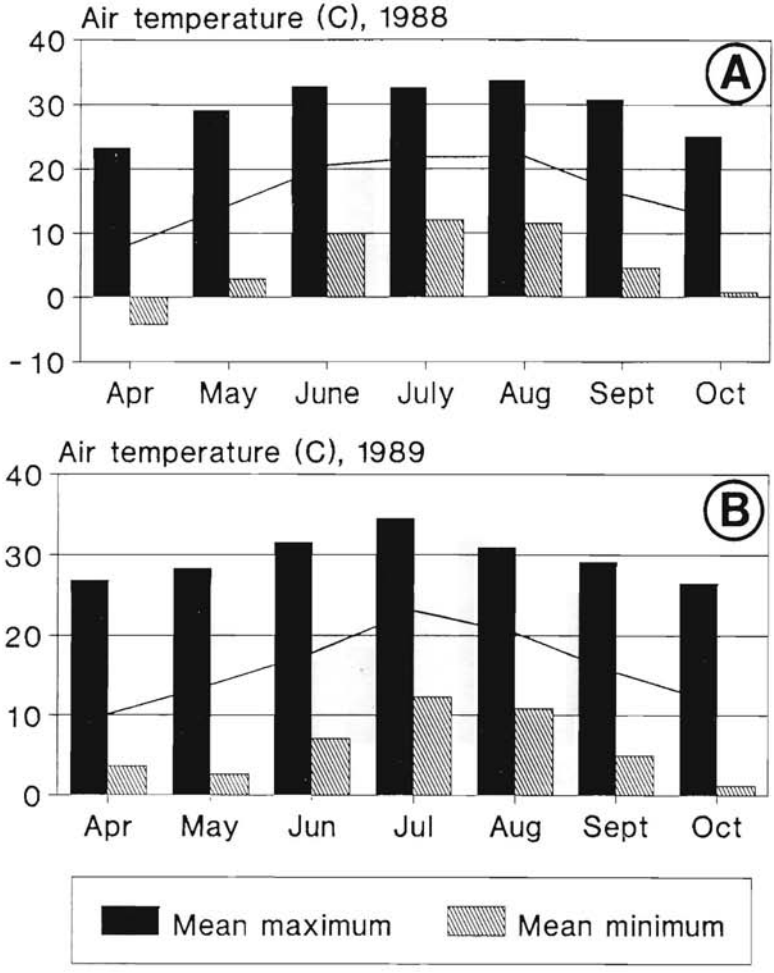

Figure 3. Mean maximal and minimal air temperatures at Fort Collins, CO, from April through October; graph line indicates monthly mean temperature. A. 1988. B. 1989.

during the hottest months of the growing season. In Colorado, severe root rot of sugarbeet occurs in July and August.

Mean population densities of *R. solani* in soil samples were relatively high at the 2-mo sampling date in both years and were inversely proportional to depth of the sample (Fig. 4A & B). In October 1988 (120 days), however, population densities of the pathogen were

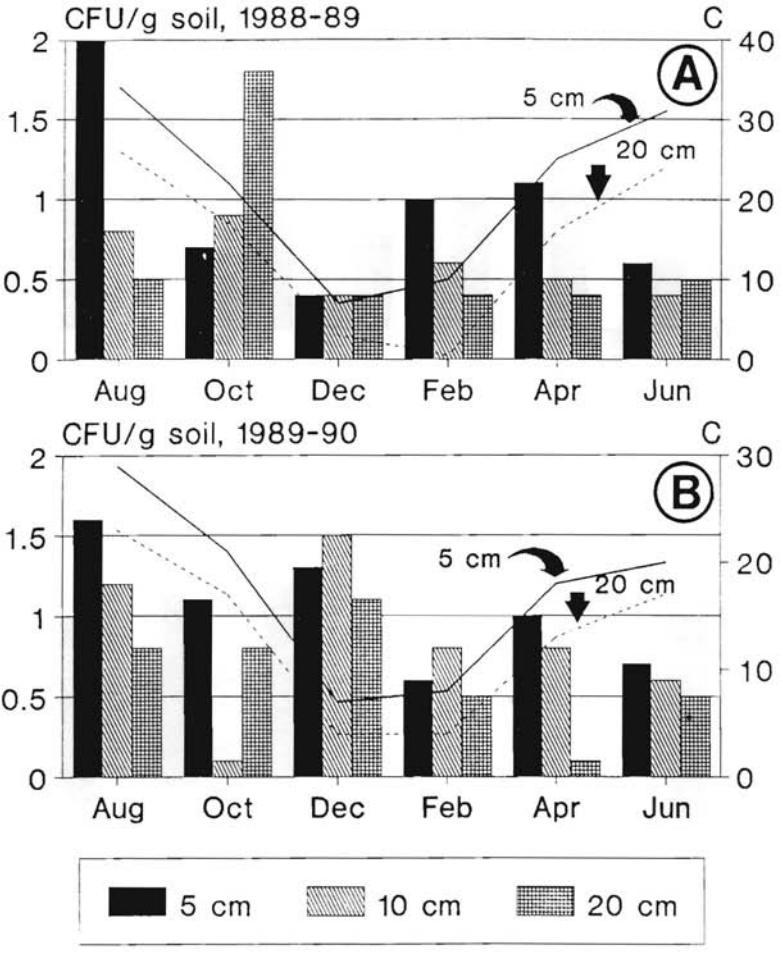

Figure 4. Bimonthly population densities in colony-forming units (CFU) per gram of air-dry soil of Rhizoctonia solani from soil surrounding buried sugarbeet roots infected with *R. solani* (isolate R-9, AG-2-2) and buried 5, 10, and 20 cm in a fallow field site; graph lines indicate mean monthly high soil temperatures at 5- and 20-cm depths. A. 1988-1989. B. 1989-1990.

directly proportional to soil depth. There was a general trend in both years toward a decline in population densities at all depths over time, but the fungus never completely disappeared. These declines over time during the growing season differ from results reported by Herr (1976), who showed that populations of *R. solani* increased slowly over the growing season. Herr's results, however, were obtained from fields in which growth of soybean or sugarbeet provided the fungus with a constant food base to colonize. Similar declines in soil inoculum densities of *R. solani* following a fallow period were demonstrated by Bell and Sumner (1987) and by Maier (1965) working with cotton seedling diseases and bean root rot.

Typically, in Fort Collins, CO, after sugarbeets that had been artificially inoculated in July are harvested (September/October), population densities of *R. solani* in soil range from 1.5-2.0 CFU g^{-1} soil; by the following spring, however, population densities usually decline to 0.5-0.8 CFU g^{-1} soil (Ruppel, *unpublished data*). Similar population densities were detected in summer and spring in both years of the current study. In the Fort Collins area, at least, there is a trend toward decline of *R. solani* populations to a low density in soil, which, apparently, reduces the population density to a point below the economic threshold. In Colorado, a 3- to 4-yr rotation has been effective in significantly reducing *Rhizoctonia* root rot incidence in sugarbeet (*personal observation*). Rotations or fallow periods of the same duration, however, have not been as effective in reducing disease incidence in the Texas Panhandle (C. M. Rush, *personal communication*).

All of the isolates (47) of *R. solani* from sugarbeet debris that were tested for pathogenicity induced severe root rot in sugarbeet, and all were characterized as AG-2-2. Of the 62 isolates tested from soil, only one was an AG-4; all others were AG-2-2. All soil isolates of AG-2-2 were pathogenic in sugarbeet. The AG-4 isolate was nonpathogenic in 3-mo-old sugarbeets. These results contrast with those reported by Kamal and Weinhold (1967) who found that, although *R. solani* in cotton tissue remained viable for at least 3.5 mo, there were changes in pathogenic ability of the fungus. Because the recovered fungus no longer was able to infect cotton seedlings but could attack germinating seed, the authors suggested that nutrients supplied by the germinating seed were necessary for parasitism by the fungus. No changes in parasitic ability of *R. solani* were evident in recovered isolates from sugarbeet debris. Possibly, sugarbeet taproots produce exudates that serve as external nutrients for parasitic activity of *R. solani*; however, because the outer layer of the taproot periderm is composed of cork cells rather than epidermal cells (Artschwager, 1926), such exudates seem unlikely.

It is not clear whether the pathogenic isolates of *R. solani* AG-2-2 from soil adjacent to the buried envelopes represented a resident population of the fungus, or evidence that the fungus had grown out from the buried root tissue and colonized nearby organic matter. When the envelopes were exhumed, soil samples were collected only from the area a few centimeters to the sides of the envelopes; no soil was collected from areas underlying the envelopes, so it is unlikely that soil isolates resulted from colonized debris passing through the fine mesh. Moreover, the envelopes were encased in soil when exhumed, which served to retard if not prevent adjacent soil from becoming contaminated with internal debris. Soil on the envelopes was not used for the assays. Possibly, sclerotia or monilioid cells, formed in hyphae that had grown out from the beet residues in the envelopes, were being sustained by the residues, as reported by Naiki and Ui (1977).

R. solani is reported to persist in soil as sclerotia on the surface of organic debris and as thick-walled monilioid cells within debris particles (Boosalis and Scharen, 1959; Naiki and Ui, 1978; Roberts and Herr, 1979; Neate, 1987). Abundant monilioid cells were observed by microscopic examination of sugarbeet debris particles from the envelopes, but it was difficult to determine whether sclerotia were present on the badly decomposed tissue. Sclerotia are formed infrequently on sugarbeets with Rhizoctonia root rot in Colorado (*personal observation*).

Variability in results between years complicates the development of recommendations for cultural management of Rhizoctonia root rot by methods that increase the degradation of organic debris colonized by the pathogen. Conceivably, even subtle differences in precipitation and air and soil temperatures could affect survival of the fungus in fallow soil. Additionally, the fungus may serve as a weak parasite on weed hosts (Daniels, 1963) or even "nonhost" small grains used in rotation programs with sugarbeet (Ruppel, 1985). A deeper plowdown of infected roots may be the best method to increase the rate of debris degradation and, thereby, effect the decline in pathogen viability. Although the rate of debris degradation was not measured, root shape at the 10- and 20-cm depths was not distinguishable at the 2-mo harvest, compared with root halves buried 5 cm deep. Lack of aeration in deeper soil profiles also can limit the parasitic activity of *R. solani* (Papavizas, 1970).

The constant, albeit low, soil population density of pathogenic *R. solani* prevailing from one year to the next may preclude monoculture of sugarbeet where Rhizoctonia root rot is endemic. In greenhouse experiments and in fields planted to sugarbeet, *R. solani*

at 0.7 CFU g^{-1} can induce serious rot in sugarbeets (Ruppel, *unpublished*). However, factors other than population density may govern disease incidence, which can vary from year-to-year or even within a given field regardless of inoculum potential (Roberts and Herr, 1979; Herr, 1987; Hyakumachi, 1983).

Besides crop rotation, other cultural methods may be necessary to significantly reduce the pathogen's inoculum potential in soil, provided such measures are economically feasible. For example, maintaining wet soil conditions in the absence of host plants reduced soil population densities of *R. solani*, presumably due to microbial activity under moist conditions (Papavizas and Davey, 1961; Sneh et al., 1972; Hadar et al., 1982). If a relatively short duration of flooding would reduce the soil population of the pathogen below the economic threshold level, such a measure might be acceptable to sugarbeet growers. Soil solarization, conversely, although significantly reducing diseases in potato caused by *R. solani* (Elad et al., 1980), would not be economically practical in sugarbeet cultivation.

Attempts to reduce Rhizoctonia root rot in sugarbeet with the antagonistic fungus *Trichoderma harzianum* Rifai have not been too successful (Ruppel et al., 1983, 1988), but additional research is needed on enhancing the activity of this and other potential biological control organisms in soil infested with *R. solani* (Herr, 1988; Schuler et al., 1989; Turhan, 1990). Biological control of Fusarium root rot of bean was achieved with the addition of barley straw to soil (Snyder et al., 1959). Such a measure usually is not effective for organisms like *R. solani* that are efficient colonizers of organic matter (Cook and Baker, 1983); however, Fernandez (1981) reduced disease percentages of Rhizoctonia root rot in sugarbeet by 34-60% with the incorporation of barley straw in the field.

Despite the voluminous amount of research associated with the suppression of diseases caused by the ubiquitous fungus R. solani, numerous questions regarding this serious pathogen of many crops remain unanswered. More epidemiological research is warranted, with the goal of economically and efficiently reducing inoculum potential in soil. Meanwhile, crop rotation and resistant cultivars are the most reliable controls against Rhizoctonia root rot in sugarbeet. Fallow periods and deep plowdown also may serve to reduce inoculum potential in the field.

ACKNOWLEDGEMENT

I thank Patricia A. Mustain for technical assistance.

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