Biological Control of Rhizoctonia Stem Canker and Black Scurf of Potato

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ABSTRACT

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The efficacy of several fungal antagonists for control of *Rhizoctonia solani* on potato was evaluated in greenhouse and field tests. Fermentor biomass (FB) preparations of *Trichoderma viride* (T-1-R9) and *Gliocladium virens* (Gl-21) applied as dusts to seed potatoes infested with sclerotia of *R. solani* before planting, reduced disease incidence in the field by 50 and 55%, respectively. Viability of sclerotia from seed pieces retrieved from the field was reduced 54–89% by specific antagonists. In the greenhouse, up to 88% reduction in germination of sclerotia was obtained by treating sclerotia-infested tubers with FB of T-1-R9 before planting. FB preparations of *T. viride* (T-1-R9), *T. harzianum* (WT-6), *T. hamatum* (Tri-4), and *G. virens* (Gl-21) added to the soil in a mixture of pulverized pyrophyllite (anhydrous aluminum silicate), significantly reduced numbers of propagules of *R. solani*. After 4 wk, populations of *R. solani* were eliminated in soils infested with FB of Tri-4. Population levels of the antagonists increased 1,000-fold within 4 wk. Sclerotia of *R. solani* retrieved from antagonist-amended soils were heavily colonized by the antagonists. The viability of colonized sclerotia was reduced 13–100%. Results suggest that both soilborne and tuberborne propagules of *R. solani* can be effectively reduced by biological means.

Black scurf, which is caused by *Rhizoctonia solani* Kühn (AG 3), is a serious and widespread disease of potatoes. Both soilborne and tuberborne inoculum is important in disease development (8). Soilborne inoculum contributes to stolon damage while tuberborne inoculum may affect sprout emergence, stolon damage, and the stem canker phase of the disease. Sclerotia that develop on infected tubers are present on certified seed potatoes in many parts of the world and are considered a primary inoculum source (5,9,11,27). Elimination of tuberborne inoculum results in better emergence, cleaner progeny tubers, and larger tuber size (15).

Present control practices include the use of chemical and cultural methods to reduce the soilborne and tuberborne sources of inoculum, although research has been directed toward the use of antagonists for the biocontrol of *R. solani* on potatoes (3,6,7,24). Jager et al (12–14) reported the suppression of *R. solani* in potato fields and attributed it to the presence of antagonistic fungi in the soil and on sclerotium-infested seed pieces. *Gliocladium roseum, G. virens, G. nigrovirens, Trichoderma hamatum*, and *Verticillium biguttatum* were common hyperparasites of sclerotia and reduced their viability (12–14,26). *V. biguttatum* introduced on seed pieces did not reduce disease severity on stems in field tests, but it did decrease numbers of sclerotia on progeny tubers (26). Aluko and Hering (3) reported complete eradication of pathogen sclerotia from tubers treated with *G. virens* prior to storage; however, biocontrol in the field was unsuccessful when pathogen-infested tubers were treated immediately before planting.

Tuberborne inoculum of *R. solani* is capable of causing disease on young stems after planting but before shoots emerge from the soil. Shoots may become resistant to infection after emergence (25). For a biocontrol agent to be effective it should be active during this time period and after emergence. Application of antagonist inoculum directly to the pathogen-infested seed piece provides an efficient means of placing the antagonist in close contact with the primary inoculum on the seed piece.

The objective of the present study was to evaluate the efficacy of fungal antagonists for the biological control of both tuberborne and soilborne inoculum of *R. solani* on potato in the greenhouse and in the field. Specific objectives were to determine the effect of fungal antagonists on: viability of tuberborne sclerotia of *R. solani*; disease incidence and severity; tuberborne sclerotium viability in the field; and survival of mycelium and sclerotia of *R. solani* in soil. A portion of this work has been presented (4).

MATERIALS AND METHODS

Fungal antagonists used were: *Trichoderma viride* Pers. ex. S. F. Gray biotypes T-1-R9, T-1-R4, and T-1(B-3M); *T. hamatum* (Bon.) Bain isolates Tm-7, Tm-17, Tm-19, Tm-23, and Tri-4; *T. harzianum* Rifai isolates Th-1, Th-5, Th-7, Th-25, WT-6, and biotypes Th-1(DEM-3M), Th-1(BAS-2M), and Th-23-R9; *Gliocladium virens* (Miller et al) isolates Gl-3, Gl-9, Gl-17, and Gl-21; *Talaromyces flavus* (Klöcker) Stolks and Samson (*Penicillium vermiculatum* Dangeard) isolates Tf-1 (18), Tf-4, Tf-5, Tf-8, Tf-16, Tf-73, and biotype Tf1-1. Biotypes T-1-R9, T-1-R4, Th-23-R9, and Tf1-1 are resistant to benomyl; Th-1(DEM-3M) is resistant to chloroneb; Th-1(BAS-2M) is resistant to vinclozolin; and T-1(B-3M) is resistant to chlorothalonil (1,20,21). Stock cultures of *T. flavus* were maintained on potato-dextrose agar (PDA) slants and all other isolates were maintained on V-8® juice agar slants. All isolates were obtained from the culture collection of the Soilborne Diseases Laboratory, Beltsville, MD.

Isolates of *Trichoderma* and *Gliocladium* were grown on V-8 juice agar under continuous fluorescent light (700 μEin/m^2/sec) at room temperature for 7 days for conidia production. Isolates of *T. flavus* were grown for 3 wk on PDA in the dark at 30 C for ascospore production or on molasses corn steep liquor agar (16) under continuous fluorescent light at room temperature for production of conidia.

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All data were subjected to analysis of variance. Significant treatment means were separated by using the Waller-Duncan *K*-ratio *T*-test.

Effect of fungal antagonists on viability of tuberborne sclerotia. Strains of *Trichoderma* spp., *Gliocladium* spp., and *T. flavus* were evaluated in greenhouse tests to determine their effect on viability of tuberborne sclerotia of *R. solani*. Spore suspensions of the antagonists were prepared by adding distilled water to the culture plates and rubbing the agar surface gently with a rubber policeman. Suspensions were adjusted with distilled water and added to Pyrax® ABB (pulverized pyrophyllite, anhydrous aluminum silicate, pH 7.0, R. T. Vanderbilt Company, Inc., Norwalk, CT) to provide 10^6 spores per gram. Fermentation biomass (FB) was prepared as described recently (19). Fermentor biomass consisted of fungal mycelium, very few conidia, and an abundance of chlamydospores of *Trichoderma* spp. and *Gliocladium* spp. or mycelium of *T. flavus*. Five-day-old FB was filtered, dried, and ground to pass a 0.38-μm (40-mesh) screen. Antagonists were applied either as spores or FB (5×10^5 propagules per seed piece) in Pyrax® to seed pieces cut from seed potatoes (*Solanum tuberosum* L. 'Superior') infested with sclerotia of *R. solani*. These infested potato tubers were obtained from fields in Idaho and Maryland. Treated potato tubers were planted individually in 10-cm-diameter pots in natural Rumsford sandy loam soil (500 g per pot) in the greenhouse at temperatures of 25 ± 2 C and watered twice daily. Control treatments consisted of Pyrax®-treated or untreated seed potatoes. The potatoes were recovered after 3 wk, and sclerotia removed individually from seed pieces (10 per tuber) were placed on antibiotic-amended water agar (AWA, 1.5% water agar; streptomycin sulfate and chlorotetracycline HCl at 100 and 50 μg/ml, respectively). Percent germination of sclerotia was determined after 48 hr. Data were transformed by using the arcsin transformation. Each treatment was replicated five times and the experiment was repeated once.

Effect of fungal antagonists on disease incidence and severity and on viability of tuberborne sclerotia in the field. Eight fungal antagonist preparations listed in Table 1, selected from greenhouse tests, were evaluated in the field for their effect on viability of tuberborne sclerotia, and on disease incidence and severity. Antagonist preparations consisting of either conidia or FB in Pyrax® (10^6 propagules per gram) were prepared as described previously and applied to sclerotium-infested seed potatoes by shaking the tubers and FB in a plastic bag. Approximately 0.5 g of FB per tuber was added in the bag before shaking. Twenty potatoes infested with *R. solani* were planted per row with 23-cm row spacing in an Elsinboro sandy loam in a field with negligible amounts of natural soilborne *R. solani* as determined by the method of Henis et al (10). Whole potato tubers of cultivar Superior were used in the test planted in the fall of 1983 and cut seed pieces of cultivar Russett Burbank were used in the test planted in the spring of 1984. Each treatment was replicated four times and arranged in a randomized complete block design. Pyrax®-treated, sclerotium-infested tubers served as controls. For a chemical control, one treatment consisted of pentachloronitrobenzene (PCNB, 75% wettable powder) sprayed in the furrow prior to hilling at the rate of 7.0 g/100 ml of water, in a 30- to 36-cm-wide band per 4.5 m of row. A total of 800 potatoes were planted in the test. Potato plants were harvested after 5 wk and disease incidence and severity were recorded. Disease severity was expressed on a scale of 0–4 as follows: 0 = no apparent stem lesions, 1 = up to 25% of stem covered with lesions, 2 = 26–50% of stem covered with lesions, 3 = 51–75% of stem covered with lesions, 4 = >75% of stem covered with lesions. Ten sclerotia were individually removed from each seed piece with forceps and placed on AWA. Percent germination of sclerotia was determined after 48 hr. The field test was done in the fall of 1983 and repeated in the spring of 1984.

Effect of fungal antagonists on the survival of mycelium and sclerotia of *Rhizoctonia* in natural soil. Sand-cornmeal inoculum of *R. solani* isolate R-108 was added to 4 kg of moist (14% moisture, w/w) nonsterile Rumsford sandy loam at a rate of 1.5% (w/w). The soil was divided into five 800-g batches and FB in Pyrax® of isolates T-1-R9, Tri-4, Gl-21, and WT-6 was added at 10^4 propagules per gram of soil. The control consisted of soil amended with *Rhizoctonia*. Four 200-g batches of infested soil was placed in 400-ml beakers, covered with polyethylene film, and kept at 24 ± 2 C. To determine antagonist population levels, 1-g soil samples were removed immediately and at 2- and 4-wk intervals and serially diluted in 99 ml of distilled water. One-milliliter aliquants were spread over the surface of Trichoderma medium E (TME) (22). Plates were incubated under continuous fluorescent light and colonies were counted after 5 days. To determine populations of *Rhizoctonia*, soil was removed from beakers at 0, 2, and 4 wk and sampled with the multiple pellet soil sampler (MPSS) (10). A total of 75 pellets were sampled per replication and placed on AWA. The percentage of soil pellets that produced a colony of *R. solani* after 24 hr was recorded. Data were transformed to the number of propagules per gram of dry soil ($\log_e 1/(1-y)$ in which y = the proportion of the pellets colonized by *R. solani*). Each treatment was replicated four times and the test was done twice.

Sclerotia of isolate R-110 of *R. solani*, which was isolated from a sclerotium on a naturally diseased tuber, were produced in potato-dextrose broth (PDB). We used isolate R-110 because it produced sclerotia readily in liquid culture. Isolate R-108 did not. Sclerotia were sieved from PDB after 3 wk and placed on Whatman No. 1 filter paper. Nonsterile soil infested with 10^4 propagules per gram of FB of isolates T-1-R9, Tri-4, Gl-21, or WT-6 was placed in 250-ml beakers. Soil without the antagonists was used as control. Filter paper with sclerotia of *R. solani* was placed on top of the soil and the sclerotia were covered with additional soil (100 g total). In the second experiment, nylon mesh (10-μm pore size) was used instead of filter paper as a carrier for the sclerotia. Beakers were covered with polyethylene film and incubated at 24 ± 2 C. Sclerotia were removed from the soil after 0, 3, 6, and 9 wk, washed in water, surface-disinfested in 0.05% sodium hypochlorite solution, and placed on AWA and TME. Percent germination of sclerotia and colonization of sclerotia by antagonists was determined after 48 hr. Each treatment was replicated four times and the experiment was repeated once.

RESULTS

Effect of antagonists on viability of tuberborne sclerotia. Several isolates of *Trichoderma* and *Gliocladium* effectively reduced the

TABLE 1. Biological control of tuberborne *Rhizoctonia solani* on potatoes in the field in 1983 by selected isolates of *Trichoderma* and *Gliocladium* species

Antagonist, isolate, and form of inoculum	Disease incidence[v] (%)	Disease severity[w]	Germination of sclerotia[x] (%)
Pyrax® (control)	99 a[y]	2.11 a	27 a
G. virens (Gl-21 conidia)	83 ab	1.88 ab	5 cd
T. viride (T-1-R9 conidia)	82 ab	1.88 ab	8 bcd
T. hamatum (Tri-4 FB)[z]	80 ab	1.51 bc	11 bc
T. hamatum (Tm-23 conidia)	75 ab	1.17 cd	7 bcd
T. hamatum (Tm-19 conidia)	71 ab	1.49 bc	9 bcd
T. harzianum (WT-6 FB)	56 ab	1.41 bc	10 bcd
T. viride (T-1-R9 FB)	49 b	1.35 c	4 d
G. virens (Gl-21 FB)	45 b	0.75 de	3 d
Pentachloronitrobenzene	35 b	0.52 e	13 b

[v] Data are mean percent disease incidence on stems. Data were transformed using the arcsin transformation.

[w] Mean severity of disease of individual stems expressed on a scale of 0 to 4.0; 0 = no symptoms, 1 = <25% of the stem covered with lesions, 2 = 26–50% of the stem covered with lesions, 3 = 51–75% of the stem covered with lesions, and 4 = >75% of the stem covered with lesions.

[x] Mean germination (%) of sclerotia removed from sclerotium-infested potatoes (cultivar Superior) and placed on antibiotic water agar. Data were transformed using the arcsin transformation.

[y] Means in a column followed by the same letter are not significantly different ($P \leq 0.05$) according to the Waller-Duncan *K*-ratio *T*-test.

[z] Fermentor biomass (FB) consisted of conidia, chlamydospores, and mycelium of *Trichoderma* and *Gliocladium* spp. added as a mixture with Pyrax® at 5×10^5 colony-forming units per seed piece.

viability of sclerotia of *Rhizoctonia* in greenhouse tests (Table 2). T-1-R9, applied as either conidia or FB in Pyrax®, reduced sclerotial germination by 76 and 88%, respectively. Tm-19 and Gl-21, applied as conidia in Pyrax® to sclerotium-infested tubers, reduced sclerotial germination by 80 and 86%, respectively. FB of Tri-4 and WT-6 reduced sclerotial germination by 83 and 74%, respectively. None of the isolates of *T. flavus* that were evaluated (not shown in Table 2) reduced germination more than 49%. All fungal antagonists shown in Table 2 except Tm-23 significantly reduced sclerotial germination. These isolates were tested in the field to determine their biocontrol efficacy.

Effect of antagonists on disease incidence and severity and on viability of tuberborne sclerotia in the field. Two isolates, T-1-R9 and Gl-21, significantly ($P < 0.05$) reduced disease incidence in 1983 in the field by 50 and 55%, respectively, but only when applied as FB (Table 1). This degree of control was comparable to that achieved with PCNB. Conidial preparations in Pyrax® of these same isolates reduced disease incidence by only 16 and 17%, respectively, but the differences between these figures and that for the control are not statistically significant. Disease severity was significantly ($P \leq 0.05$) reduced in the field by most of the fungal antagonists that were evaluated. Treatment of tubers with FB of isolate Gl-21 resulted in the greatest reduction in disease severity.

All of the isolates tested in the field reduced sclerotial germination by 59% or more. Even though sclerotial germination was significantly reduced, disease incidence was still high in some treatments. Treatment of tubers with FB of isolates T-1-R9 and Gl-21 resulted in the greatest reduction in sclerotial germination concomitant with the greatest reductions in disease incidence. The fungicide PCNB was less effective in reducing sclerotial germination than the biological agents that were evaluated. Linear regression analysis of the disease severity versus sclerotial germination data did not reveal a significant relationship between these variables.

A significant reduction in disease incidence was not obtained with any of the antagonists evaluated in 1984. Treatment of potatoes with all the biocontrol agents tested except conidia of T-1-R9 and Tm-23 reduced disease severity in the field. A high percentage of sclerotia (68–86%) were viable at harvest on tubers planted in the 1984 field test. Treatment of sclerotium-infested seed potatoes with FB of T-1-R9 numerically reduced sclerotial germination by 21% in 1984, but the treatment differences were not statistically significant.

Effect of fungal antagonists on the survival of mycelium and sclerotia of *R. solani* in soil. FB preparations of T-1-R9, WT-6, Gl-21, and Tri-4 significantly reduced the survival of propagules of *R. solani* in soil (Fig. 1). Isolates Gl-21 and Tri-4 were the most effective in reducing soilborne inoculum levels of *R. solani*. By 4 wk after soil infestation, *R. solani* was not recovered from soils treated with FB of Tri-4. Population levels of all the antagonists increased in soil over time (Fig. 2). Up to 1,000-fold increases in colony-forming units were measured by dilution plating techniques over the course of the experiment. Greatest increases in antagonist population levels were obtained with isolates WT-6 and Gl-21.

FB preparations of *Trichoderma* and *Gliocladium* species significantly reduced the viability of soilborne sclerotia of *R. solani*

TABLE 2. Biological control of tuberborne *Rhizoctonia solani* on potatoes in greenhouse experiments by selected isolates of *Gliocladium* and *Trichoderma* species

Antagonist, isolate and form of inoculum	Germination of sclerotia[x] (%)
None (control)	42 a[y]
Pyrax® alone (control)	19 ab
T. hamatum (Tm-23 conidia)	19 ab
T. harzianum (WT-6 FB)[z]	11 b
T. viride (T-1-R9 conidia)	10 b
T. hamatum (Tm-19 conidia)	8 b
T. hamatum (Tri-4 FB)	7 b
G. virens (Gl-21 conidia)	6 b
T. viride (T-1-R9 FB)	5 b

[x] Data are mean percent germination of sclerotia removed from sclerotium-infested potatoes and placed on antibiotic water agar 3 wk after fungal antagonist treatment. Data were transformed using the arcsin transformation.

[y] Means in a column followed by the same letter are not significantly different according to the Waller-Duncan *K*-ratio *T*-test.

[z] Fermentor biomass (FB) consisted of mycelium, conidia, and chlamydospores of *Trichoderma* spp. added as a mixture with Pyrax® at 5 $\times 10^5$ colony-forming units per seed piece.

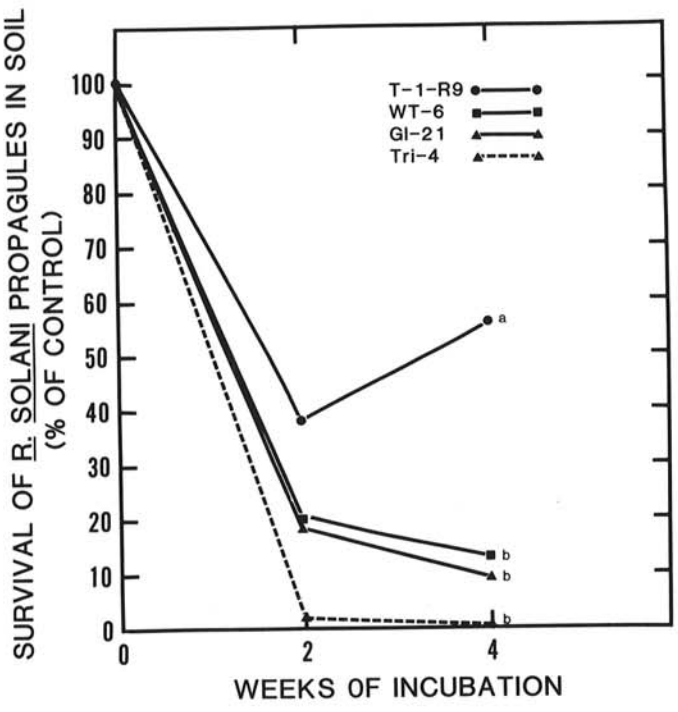

Fig. 1. Survival of propagules of *Rhizoctonia solani* in soil (calculated as a percentage of the control at each time period) over a 4-wk period as affected by the addition of fermentor biomass of *Trichoderma viride* (T-1-R9), *T. harzianum* (WT-6), *Gliocladium virens* (Gl-21), and *T. hamatum* (Tri-4) at 10^4 propagules per gram of soil.

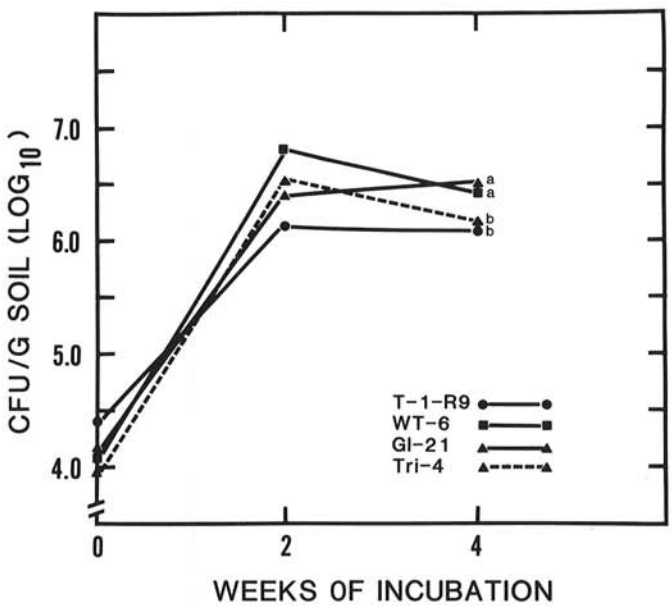

Fig. 2. Colony-forming units of *Trichoderma viride* (T-1-R9), *T. harzianum* (WT-6), *Gliocladium virens* (Gl-21), and *T. hamatum* (Tri-4) over a 4-wk period in a sandy loam amended with fermentor biomass of the isolates at 10^4 propagules per gram of soil.

by 9 wk after soil infestation (Table 3). Isolates Tri-4 and WT-6 were most effective in reducing the viability of soilborne sclerotia. A significant number of sclerotia retrieved from antagonist-amended soils were colonized by *Trichoderma* and *Gliocladium* species.

DISCUSSION

FB of T-1-R9 and Gl-21 applied as Pyrax® to sclerotium-infested potato tubers significantly reduced disease incidence and severity and the viability of tuberborne sclerotia of *R. solani* in the field in 1983. FB of *Trichoderma* and *Gliocladium* species also significantly reduced soilborne levels of *R. solani* in laboratory tests. Viability of soilborne sclerotia was reduced and sclerotia were colonized by the antagonists.

The greenhouse screening procedure used in these tests with natural soil provided an effective means of determining which isolates were most suitable for biocontrol of the pathogen sclerotia in a particular ecological niche, namely the tuber surface in nonsterile soil. Since tuberborne inoculum is primarily responsible for the stem canker phase of the disease, isolates that reduce tuberborne sclerotium viability in the field might be expected to reduce disease incidence and severity as well. Disease incidence and severity were reduced in most treatments with biological agents. However, reduced sclerotium viability was not positively correlated with either low disease incidence or severity. Other workers (2) have shown that disease incidence on stems is closely related to the frequency of sclerotia on seed tubers and the origin of seed tubers (14). In our first field test, only those antagonists that caused greater than an 85% reduction in sclerotium viability (T-1-R9 and Gl-21) significantly reduced disease incidence. In other treatments, the remaining viable sclerotia may have been sufficient to cause high incidence of disease. Sclerotial isolates of *R. solani* have been reported to differ in pathogenicity to potato (2). This may also explain why in some treatments sclerotial viability was not correlated with disease incidence.

Significant reductions in disease incidence and sclerotial germination were not obtained in the second field test with FB of T-1-R9 and Gl-21. Environmental conditions were substantially different between the two field tests. Different potato cultivars and whole and cut tubers were planted in the two tests. Different cultivars were used in the second test because tubers of cultivar Superior infested with *R. solani* were unavailable. Wet, cold soil conditions during the test in the spring of 1984 were not conducive to disease development.

T-1-R9 and Gl-21, when applied as FB to tubers, may possess advantageous biocontrol attributes that make them better antagonists than the others that were evaluated. T-1-R9 is a mycoparasite of hyphae of *R. solani* in vitro (J. E. Beagle-Ristaino, *unpublished*). The production of one or more toxic substances by these fungi may have been involved in the reduction of sclerotial viability. Aluko and Hering (3) reported that concentrations of gliotoxin and viridin lethal to *R. solani* were present on sclerotia from potatoes treated with *G. virens* and placed in storage. Jager et al (12) speculated that the destruction of sclerotia probably depends on the production of a toxic substance rather than direct parasitism. No attempts were made in our study to isolate or identify inhibitory compounds produced by *Trichoderma* or *Gliocladium* species on sclerotial surfaces.

Even if a large percentage of the sclerotia on infested tubers are heavily colonized or killed by antagonists, the remaining viable sclerotia which germinate, enter the soil, and form mycelium or sclerotia may be sufficient to cause disease. In regions where soilborne inoculum is present, control of the soilborne phase of the pathogen is important. T-1-R9, Tri-4, WT-6, and Gl-21 significantly reduced propagules of *R. solani* in soil when applied as FB. Tri-4 was more effective in reducing soilborne populations of *R. solani* than the other isolates tested.

It is generally accepted that an antagonist must be metabolically active and grow in soil to be an effective biocontrol agent. The antagonist must become established in the soil and interact with the pathogen to reduce disease. The isolates of *Trichoderma* and *Gliocladium* evaluated in this study increased up to 1,000-fold in natural soil over a 4-wk period when applied as FB. This increase probably represented germination of chlamydospores, hyphal elongation, and production of conidia. The obvious green color of the soil infested with antagonist FB is indicative of this. The increase of the antagonists in soil occurred even when propagules of *R. solani* were not present. Residual nutrients in the FB may have provided a food base for the increase (19).

TABLE 3. Germination and colonization of soilborne sclerotia of *Rhizoctonia solani* following treatment with fermentor biomass preparations of *Trichoderma* and *Gliocladium* species

Treatment	Germination[x] (%)			Colonization[y] (%)		
	3 wk	6 wk	9 wk	3 wk	6 wk	9 wk
None	64 a[z]	56 a	56 a	5 b	0	0
T. viride (T-1-R-9)	32 ab	27 b	49 a	100 a	97 a	95 ab
G. virens (Gl-21)	14 bc	16 bc	13 b	100 a	62 b	79 b
T. harzianum (WT-6)	28 bc	0	5 b	100 a	97 a	100 a
T. hamatum (Tri-4)	0	0	0	100 a	89 a	93 ab

[x] Sclerotia were placed on filter paper and buried in soil infested with 10^4 colony-forming units of fermentor biomass preparations of *Trichoderma* and *Gliocladium* species per gram of soil. Percent germination was determined by retrieving sclerotia from soil, surface disinfesting them in 0.05% sodium hypochlorite, and placing them on antibiotic water agar.

[y] Percent colonization was determined by retrieving sclerotia from soil, surface disinfesting them in 0.05% sodium hypochlorite, and placing them on Trichoderma medium E.

[z] Means in a column are not significantly different according to the Waller-Duncan *K*-ratio *T*-test.

Benomyl-resistant T-1-R9 reduced Fusarium wilt of vegetative and flowering greenhouse chrysanthemums (17). Conidia of T-1-R9 colonized pasteurized greenhouse soil mix rapidly and reduced disease to levels comparable to a chemical control. Conidia of *T. viride* were unable to colonize unpasteurized soil (17). Conidia of the isolates of *Trichoderma* and *Gliocladium* evaluated in this study were less effective than FB in controlling disease. Conidia of *Trichoderma* species are highly sensitive to soil fungistasis (23). FB of *Trichoderma* and *Gliocladium* species consists mostly of chlamydospores. Chlamydospores of *Trichoderma* and *Gliocladium* species produced in liquid fermentation systems germinate readily in soil (J. E. Beagle-Ristaino and J. A. Lewis, *unpublished*). It is believed that chlamydospores are the active propagules in biocontrol in this system.

Production of FB from relatively inexpensive ingredients could easily be adapted by industry for commercial manufacture of biocontrol agents. Dusting of potato seed pieces with antagonists either alone or in conjunction with fungicides is possible and would be a practical means of introducing the antagonist into agricultural production systems.

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