Influence of the Antagonist *Laetisaria arvalis* on Infection of Table Beets by *Phoma betae*

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The authors wish to acknowledge the technical assistance of Mary Powers, and thank Rose McMillen for preparation of the figures and Betty Bixler for processing the manuscript.

Research was partially supported by grants or funds from the USDA-SEA (59-2361-1-1-750-0), New York State Table Beet Growers Research Association, and Western Regional Project W-147.

Accepted for publication 14 May 1984.

ABSTRACT

Martin, S. B., Abawi, G. S., and Hoch, H. C. 1984. Influence of the antagonist *Laetisaria arvalis* on infection of table beets by *Phoma betae*. Phytopathology 74:1092-1096.

Interactions of the fungal antagonist, *Laetisaria arvalis*, with the seedborne pathogen, *Phoma betae*, were studied in natural (unpasteurized) and pasteurized beet field soils in greenhouse tests. Portions of natural and pasteurized soils were amended (5%, v/v) with preparations of *L. arvalis*. These soils were planted with seedballs of the table beet cultivar Ruby Queen that were nontreated (~80% infested with *P. betae*), hot water treated to eliminate *P. betae*, or treated with metalaxyl to prevent infection by *Pythium ultimum* in the natural field soils. In pasteurized soils (lacking *P. ultimum* and *Rhizoctonia solani*), disease induced by *P. betae* was greatly reduced by soil amendments with *L. arvalis* or by coating seedballs infested with *P. betae* with sclerotia of the antagonist. In beet field soils naturally infested with *P. ultimum* and *R. solani*, preemergence and postemergence damping-off and wire-stem symptoms were reduced when such soils were amended with *L. arvalis* in comparison to unamended soils.

Hoch and Abawi (8) reported the effectiveness of the antagonist *Laetisaria arvalis* Burdsall (previously *Corticium* sp.) for control of pre- and postemergence damping-off induced in table beet (*Beta vulgaris* L.) seedlings by *Pythium ultimum* Trow. This fungus also has shown potential for control of *Rhizoctonia solani* Kühn on cucumber (*Cucumis sativus* L.), snap bean and dry edible bean (*Phaseolus vulgaris* L.), and sugar beet (10,14). However, there are no reports on the effectiveness of *L. arvalis* for the biological control of fungal plant pathogens other than *Pythium* spp. or *R. solani*.

During recent studies on population dynamics of *L. arvalis* and *Pythium* spp. in soils cropped to table beet (11), we observed that seedlings growing in pasteurized soil lacking *P. ultimum* often died or exhibited black hypocotyls from which *Phoma betae* Frank could be isolated. In similarly treated soil that also contained *L. arvalis*, damping-off or black hypocotyl symptoms on surviving beets was greatly reduced, presumably because *L. arvalis* prevented infection by *P. betae* (11).

The seedborne fungus, *P. betae*, is a well recognized pathogen of sugar beet and table beet. The importance of its role as a foliar and storage rot pathogen of sugar beet is well established (3,4). It has also been implicated as a pathogen of sugar beet seedlings, along with *P. ultimum* (1,6,12,17), *R. solani* (1,6,12,17), and *Aphanomyces cochlioides* Drechs. (5,12,17).

Because *P. betae* is a potentially important pathogen of table beets and because it also frequently complicates screening beet germ plasm for resistance to other soilborne fungi, experiments were designed to define and document the apparent beneficial interaction of *L. arvalis* with *P. betae* on table beets in two New York soils.

MATERIALS AND METHODS

Soil collection and pathogen assays. Soils were collected from two different table beet fields near Geneva, NY, both with histories of severe root rot. Soils were screened through a 1-cm-mesh sieve to remove rocks and debris, and stored at 4 C until needed. For greenhouse experiments the soils were mixed with builder's sand (1:1, v/v) to improve drainage and prevent crusting. A portion of each was steam pasteurized at 60 C for 30 min to eliminate *P. ultimum* and *R. solani*. For clarity, nonpasteurized field soils will be referred to as natural soils, NS1 (Lima silt loam) and NS2 (Cayuga silt loam). Low-temperature *Pythium* spp. (primarily *P. ultimum*), determined by soil dilutions plated on Tsao and Ocana's medium (16), as modified by Pieczarka and Abawi (15), were present at ~250 and ~280 germinable propagules per gram of oven-dry soil for the NS1 and NS2 soil-sand mixtures, respectively. Propagule levels of *R. solani* were determined by wet-sieving organic debris by Weinhold's method (18) and suspending the debris in a modified Ko and Hora medium (9). The population was determined to be approximately four propagules per 100 g of oven-dry-equivalent soil for both the NS1 and NS2 soil-sand mixtures.

Seed treatments. A nonfungicide-treated multigerm (seedball) seedlot of table beet cultivar Ruby Queen that was ~80% infested with *P. betae* was used throughout this study. Seed infestation and infection of seedlings by *P. betae* were determined by plating seedballs or seedlings on Bugbee's diagnostic agar medium (2). Seedball treatments were as follows: nontreated (ie, ~80% *Phoma*-infested); hot water treated (59 C for 8 min, dried for 24 hr, and retreated again) to rid the seedballs of *P. betae* (7); and metalaxyl treated (Ridomil 2E at 12.5 mg a.i./g of seedballs) to inhibit infection of seedballs and seedlings by *P. ultimum*.

The growth rate of an isolate of *P. betae* was studied in Difco potato-dextrose agar (PDA) amended with metalaxyl at 0, 1, 10, and 100 mg a.i./L to test for possible inhibition of *P. betae*. No inhibition was detected; growth at all dilutions was the same as on unamended PDA.

Inoculum preparation. *L. arvalis* isolate NRRL 11375 was used in all experiments. Inoculum of *L. arvalis* was increased on sterile moist wheat bran or was grown in potato-dextrose broth (PDB) for sclerotia production. The wheat bran inoculum was prepared by adding 135 ml of distilled water to 100 g of air-dried wheat bran in 500-ml flasks, mixing thoroughly, and autoclaving for 30 min at 121 C and again for 20 min on the following day. Mycelial disks from cultures of *L. arvalis* grown on PDA were then introduced aseptically into the sterile wheat bran. Broth cultures were prepared

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by aseptically introducing mycelial disks into sterile deep petri dishes containing ~35 ml of PDB. Flasks and dishes containing both inoculum preparations were incubated for 4 wk at 20–25 C, which allowed for thorough colonization of the wheat bran by *L. arvalis* and adequate sclerotia development within the PDB cultures. Sclerotia were harvested from the PDB cultures by macerating hyphal mats in a blender for 20 sec, collecting them on a 425-μm (40-mesh) sieve, and rapidly air-drying. Sclerotia were stored at 4 C before use, and germination tests indicated their complete viability when applied to seedballs.

Greenhouse tests. Ten seedballs of the appropriate seed treatments were planted at a depth of 2 cm in 10-cm-diameter pots containing either pasteurized or natural beet field soils that were unamended or amended with *L. arvalis*-colonized wheat bran (5%, v/v). Each experiment (natural beet field soil or pasteurized soil) constituted a 2 × 3 factorial of soil amended or unamended with *L. arvalis,* each planted with seedballs treated as described. There were five replicate pots for each treatment combination and each experiment was performed three times. Greenhouse temperatures were maintained at 18–25 C to simulate conditions frequently encountered in New York when table beets are planted during May. Soils were watered as often as needed to favor damping-off and root rot development.

An additional experiment was performed to evaluate the effect of seedball treatments with sclerotia of *L. arvalis* for control of seed and seedling disease in pasteurized or natural soil. The natural soils (NS1) and pasteurized, seed, and experimental conditions were the same as employed in the above experiments, except that *L. arvalis* was not incorporated into soils. The seedball treatments were: untreated (infested with *P. betae*); treated with methylcellulose (3%, 2.4-ml of slurry per 6 g of seedballs); treated with methylcellulose and sclerotia of *L. arvalis* (3%, 2.4 ml of methylcellulose and 2 g of sclerotia of *L. arvalis* per 6 g of seedballs); soaked in a 0.2% thiram suspension at 30 C for 24 hr; and treated with thiram formulated as a wettable powder at 5.5 mg a.i. per gram of seedballs.

In all experiments, data were collected weekly (4 wk) for emergence, percentage of postemergence damping-off, and number of healthy seedlings. In the factorial experiments, data were also collected for percentage of wire-stem symptoms. The factorial experiments were analyzed appropriately, with treatment mean squares partitioned into single-degree-of-freedom orthogonal contrasts (comparisons) for main effects and interaction components. The seed treatment experiments were conducted in a randomized complete block design and means were compared by using the Waller-Duncan *k*-ratio *t*-test.

Isolation and identification. As seedlings became diseased in both pasteurized and natural soils, they were carefully removed from the soil for isolation of fungi. Seedlings were washed in running tap water for 30 min, surface-sterilized for no more than 20 sec in 0.525% NaOCl, rinsed immediately in two changes of sterile distilled water, and bisected laterally through the center of the lesions. The halves were placed on water agar and Bugbee's agar medium (2) to detect *Pythium* spp., *Rhizoctonia* spp., and/or *P. betae*. The numbers of seedlings tested and the fungi isolated from them were recorded for each treatment.

RESULTS

Results obtained using the two field soils were very similar; therefore, only those results from NS1 and its pasteurized counterpart are reported in detail.

Results obtained in experiments with pasteurized NS1 soil differed considerably from those in natural soils as all seedling infections in the former were caused by *P. betae*. The most noticeable effect in pasteurized soil was the reduced disease incidence (postemergence damping-off and wirestem symptoms) in soil amended with *L. arvalis* compared to that in unamended soil (Fig. 1C and D, solid bars). However, the general trend of reduced incidence of damping-off and wire-stem in soil amended with *L. arvalis* depended on the particular seed treatment. Seedlings derived from hot water-treated seedballs were essentially disease-

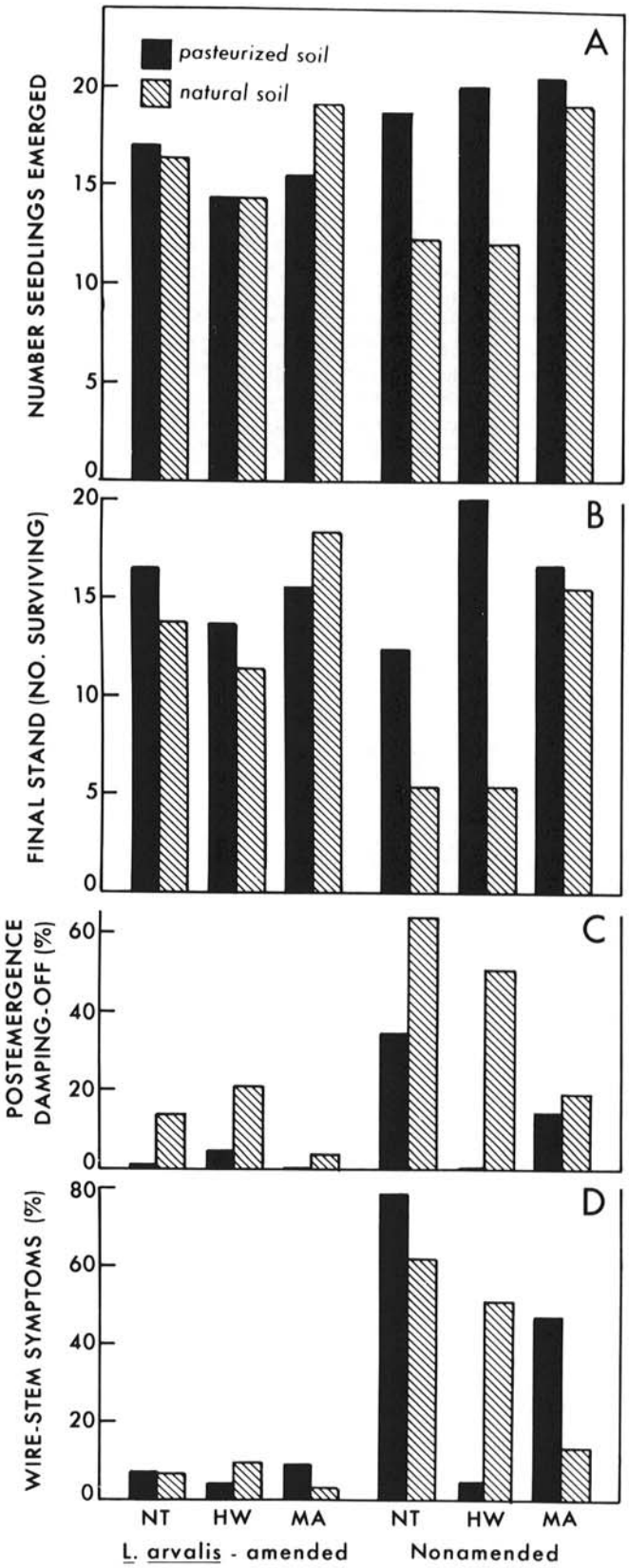

Fig. 1. Mean disease incidence of table beet seedlings planted into pasteurized soil (solid bars) or natural (unpasteurized) beet field soil (cross-hatched bars). Natural soil contained *Pythium ultimum* (~250 germinable propagules per gram of dry soil) and *Rhizoctonia solani* (approximately four propagules per 100 g of dry soil). Both soils were either unamended or amended with *L. arvalis* (5%, v/v) and planted with untreated (NT), hot water-treated (HW), or metalaxyl-treated (MA) seedballs. Data presented were obtained 4 wk after planting.

free, indicating the effectiveness of this treatment for the elimination of *P. betae*. Seedlings from untreated seedballs had the greatest disease incidence in the unamended soil; somewhat less disease incidence was associated with metalaxyl treatments (Fig. 1C and D, solid bars). Emergence was slightly reduced in pasteurized soil amended with *L. arvalis* but this effect was somewhat more pronounced for groups planted to the hot water-treated seedballs (Fig. 1A). The final plant stand was greater in soil amended with *L. arvalis* for these treatments (Fig. 1B).

In natural soil (Fig. 1, cross-hatched bars), seed treatments influenced emergence and subsequent disease incidence of surviving seedlings. Metalaxyl treatment of seedballs resulted in greater emergence, less postemergence damping-off and wire-stem symptoms, and subsequently a greater final stand than did nontreated or hot water-treated seedballs (Fig. 1A–D) in unamended soil. Untreated seedballs and hot water-treated seedballs performed similarly, with slightly greater disease incidence in the untreated than for the hot water-treated seedballs (Fig. 1). These data and the isolation data (Table 1) indicated the effectiveness of metalaxyl in preventing seedling disease due to *P. ultimum*.

In natural soil amended with *L. arvalis*, disease incidence was significantly less than in similar treatments lacking *L. arvalis* (Fig. 1), depending on the seedball treatment. These effects were greatest for seedlings derived from hot water-treated or untreated seedballs, since metalaxyl was very effective in reducing disease incidence in the absence of *L. arvalis*. However, metalaxyl-treated seed planted into natural soil amended with *L. arvalis* resulted in lower disease incidence than any other treatment combination.

The statistical analyses of the factorial experiments are best evaluated with reference to Fig. 1 for the means of the appropriate disease response, and Table 2 (pasteurized soil) or Table 3 (natural soil) for the orthogonal comparisons.

In pasteurized soil (Table 2), significant interactions were associated primarily with the hot water treatment. For instance, emergence from hot water-treated seedballs was depressed more than emergence from other seedball treatments in the presence of *L. arvalis* in comparison to emergence in the absence of *L. arvalis* (Fig. 1, Table 2). On the other hand, the final stand was increased from untreated seedballs in soil amended with *L. arvalis* in comparison to unamended soil, but, for hot water treatments, the opposite was true (hence, a significant interaction).

There were significant interactions of soil treatment (unamended or amended with *L. arvalis*) and seed treatments for all disease evaluations except for emergence and postemergence damping-off in natural soil (Table 3). For natural soil, greater emergence

TABLE 1. Frequency of isolation of *Pythium ultimum*, *Rhizoctonia solani*, and *Phoma betae* from diseased table beet seedlings grown from treated and untreated seedballs in natural (unpasteurized) and pasteurized beet field soils amended and unamended with inoculum of *Laetisaria arvalis*

Soil treatment[a]	Seedball treatment[b]	*P. ultimum*		*R. solani*		*P. betae*	
		Nat.[c]	Past.[d]	Nat.	Past.	Nat.	Past.
(+) *Laetisaria arvalis*							
	Nontreated	9/9	0	0	0	0	0
	Hot water	6/12	0	6/12	0	0	0
	Metalaxyl	0	0	7/7	0	0	0
(−) *L. arvalis*							
	Nontreated	42/47	0	5/47	0	1/47	50/50
	Hot water	28/29	0	1/29	0	2/29	2/2
	Metalaxyl	0	0	12/12	0	0	20/20

[a] *L. arvalis* was added at the rate of 5% (v/v).
[b] Untreated seedballs were ~80% infested with *P. betae*; hot water treatment was 59 C for 8 min, on two consecutive days; metalaxyl was added at 12.5 mg/g of seedballs.
[c] Nat. = natural, unpasteurized beet field soil initially infested with ~250 germinable propagules of *P. ultimum* per gram of soil and approximately four propagules of *R. solani* per 100 g of soil.
[d] Past. = beet field soil pasteurized at 60 C for 30 min.

TABLE 2. Effect of *Laetisaria arvalis* (LA) and beet seedball treatments on seedling diseases of table beet in pasteurized beet field soil; factorial treatments were partitioned to give single-degree-of-freedom orthogonal comparisons[a]

Orthogonal comparison	Disease evaluation; mean squares[b]			
	Emergence	Postemergence damping-off (%)	Final stand	Wire-stem symptoms (%)
1. +LA vs −LA	132.3**[c]	2,046.9**	9.6	6,392.8**
2. Nontreated vs treated	0.3	1,006.1**	29.4*	2,997.8**
3. Hot water vs metalaxyl	3.2	341.1*	3.2	2,791.2**
4. Interaction *a* (comparison 1 × 2)	21.6*	1,023.8**	106.7**	2,127.1**
5. Interaction *b* (comparison 1 × 3)	0.8	993.9**	33.8*	1,774.9**
Error	3.9	75.3	6.0	166.2

[a] Based on a factorial experiment consisting of two soil treatments (nonamended or amended with *L. arvalis* at 5% by volume) and three seedball treatments (nontreated, hot water treated, or metalaxyl treated).
[b] Treatment mean squares for the appropriate orthogonal comparison can be divided by the error mean square to provide an *F* statistic, all with 1 and 24 d.f.
[c] * and ** refer to statistical significance levels at $P = 0.05$ and $P = 0.01$, respectively.

TABLE 3. Effect of *Laetisaria arvalis* (LA) and beet seedball treatments on seedling diseases of table beet in natural (unpasteurized) soil; factorial treatments were partitioned to give single-degree-of-freedom orthogonal comparisons[a]

Orthogonal comparison	Disease evaluation; mean squares[b]			
	Emergence	Postemergence damping-off (%)	Final stand	Wire-stem symptoms (%)
1. +LA vs −LA	14.7[c]	4,496.2**[d]	235.2**	7,220.5**
2. Nontreated vs treated	4.8	817.5*	68.3*	786.0
3. Hot water vs metalaxyl	174.1**	1,833.8**	352.8**	1,923.9*
4. Interaction *a* (comparison 1 × 2)	1.4	392.6	29.4*	1,621.8*
5. Interaction *b* (comparison 1 × 3)	6.1	5.8	9.8	329.5
Error	4.8	126.0	6.9	278.3

[a] Based on a factorial experiment consisting of two soil treatments (unamended or amended with *L. arvalis* at 5% v/v) and three seedball treatments (nontreated, hot water treated, or metalaxyl treated).
[b] Treatment mean squares for the appropriate orthogonal comparison can be divided by the error mean square to provide an *F* statistic, all with 1 and 24 d.f.
[c] This comparison was significant at $P = 0.10$.
[d] * and ** refer to significance levels at $P = 0.05$ and $P = 0.01$, respectively.

(significant at $P = 0.10$) was associated with amendment with *L. arvalis* for all seedball treatments. Among the seed treatments, comparisons indicated that the metalaxyl treatment led to significantly greater emergence in comparison to the hot water treatment (Fig. 1A, Table 3). Similar significant differences were obtained for postemergence damping-off, indicating the effectiveness of *L. arvalis* in reducing this phase of disease and also the effectiveness of metalaxyl as a seedball treatment (Fig. 1C, Table 3).

In all other analyses, significant interactions of soil treatment with seedball treatments were apparent (Fig. 1, Tables 2 and 3). These interactions were due to the differential effect of *L. arvalis* with seedball treatments. For natural soil (Table 3), *L. arvalis* increased final stand more effectively for untreated than for treated seedballs, and was more effective in increasing final stand from the hot water treatment than from the metalaxyl treatment where the stand was increased due to metalaxyl alone.

In pasteurized soil, however, the treatment of seedballs with *L. arvalis* slightly reduced emergence in comparison to the untreated seedballs, similar to the results of the previous experiment in pasteurized soil. Both the fungicide seedball treatments and the treatment with *L. arvalis* increased the final stand of beet seedlings significantly over that of the untreated or methylcellulose checks in pasteurized soil, indicating control of *P. betae*.

Results of the seedball treatment experiments indicated no significant effects of any seedball treatment on emergence of seedlings (Fig. 2) in natural soil. However, increased final stand was obtained with seedballs treated with the *L. arvalis* and the thiram-soak treatments and planted in natural soil. Postemergence damping-off was not significantly decreased by any seedball treatment in comparison to untreated seedballs in natural soil, indicating a lack of control of the postemergence damping-off phase of *P. ultimum*. In pasteurized soil, however, seedlings derived from *L. arvalis*-treated, thiram-soaked, or thiram-dusted seedballs were significantly less affected by postemergence damping-off induced by *P. betae* than were the control treatments.

Fungal isolations from diseased seedlings indicated that *P. ultimum* was the primary pathogen responsible for the disease manifested in natural soil (Table 1). *R. solani* also was isolated, but much less frequently than *P. ultimum*. *P. betae* was detected on Bugbee's medium (2) only three times out of the total number of isolations made, and in all of these cases either *P. ultimum* or *R. solani* was also present. Most isolations were made from seedlings obtained from nontreated seedballs planted in soils that were unamended with *L. arvalis*. *P. ultimum*, *R. solani*, and *P. betae* were detected from 51/56, 5/56, and 1/56 diseased seedlings derived from untreated seedballs, respectively. In contrast, seedlings exhibiting blackened hypocotyls that were obtained from pasteurized soil yielded only *P. betae* (Table 1).

Data from experiments conducted in the second natural soil (NS2) showed that significantly reduced disease was also associated with soil amendments with *L. arvalis* in both the pasteurized and natural portions of the NS2 soil, but again it was dependent on the seed treatment. However, this time there was no observed detrimental effect on emergence induced by the hot water treatment in pasteurized soil amended with *L. arvalis*. Again, final stand was increased in *L. arvalis*-amended pasteurized soil for seedlings derived from untreated seeds. There was no significant interaction of seedball treatment and soil amendment for emergence or final stand for pasteurized soil as in the first soil (NS1), because hot water treatments did not depress emergence to the same degree in pasteurized NS2 as in pasteurized NS1. For the other parameters measured, interactions were significant as before, indicating effectiveness of *L. arvalis* in reduction of disease incidence for seedlings derived from untreated or metalaxyl-treated seeds, but little additional reduction in disease was associated with hot water treatments. These results indicated effectiveness of *L. arvalis* and hot water treatments in reducing disease caused by *Phoma betae*.

DISCUSSION

These experiments confirmed our previous observation (11) that *L. arvalis* was effective in greatly reducing damping-off symptoms

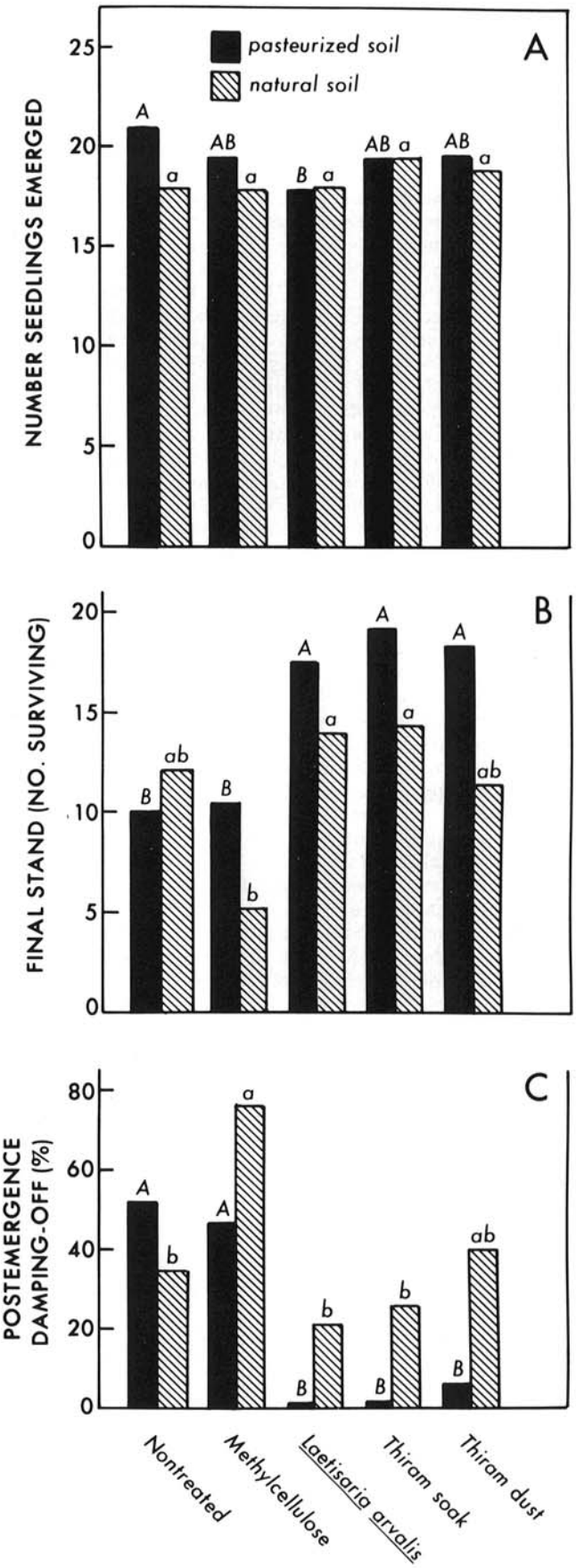

Fig. 2. Mean disease incidence of table beet seedlings planted into pasteurized soil (solid bars) or natural (unpasteurized) beet field soil (cross-hatched bars). Soils were planted to various beet seedball treatments, and data presented were obtained 4 wk after planting. Means with the same letter are not significantly different, $P = 0.05$, according to the Waller-Duncan k-ratio t-test.

induced in table beet seedlings by *P. betae* in pasteurized soil. *L. arvalis* applied as a soil amendment (colonized wheat bran) or seedball treatment with sclerotia significantly reduced the blackened hypocotyl symptom that frequently led to postemergence damping-off and reduced seedling stands, although emergence was slightly reduced by both application methods. It was notable that *P. betae* was detected almost exclusively from diseased seedlings grown in the pasteurized soils. Even though the seedball source we used was heavily infested with the pathogen, beet seedlings grown in the natural field soils rarely became infected with *P. betae*. Instead, *P. ultimum* was implicated as the primary seedling pathogen. Apparently, *P. betae* was less able to compete in these soils under the conditions of our tests. We anticipated that *P. betae* would be detected as a pathogen more frequently than it was in the natural soils. However, these soils are typical of the soils cropped continuously to several vegetable crops in New York State, and they contained *P. ultimum* and *R. solani* at inoculum densities adequate to induce considerable seedling disease, as demonstrated in these experiments. Similar observations were made by Buchholtz (1) with sugar beet in Iowa. He observed that *Pythium debaryanum* (probably = *P. ultimum* [13]) was the species most frequently isolated from diseased seedlings grown in field soils in the greenhouse or in field plantings, and that *P. betae* was rarely isolated from seedlings in the field that were "... not entirely killed by *Pythium debaryanum*." Buchholtz also found, however, that in a particular soil that exhibited low disease pressure from *Pythium*, *Phoma betae* was detected as a pathogen more frequently (1). Likewise, we have planted the *Phoma*-infested seed employed in these experiments into sandy loam soils from Long Island in New York that contained a very low population of low-temperature *Pythium* spp., and in this soil we detected *P. betae* on diseased seedlings more frequently (*unpublished*).

Bugbee and Cole (4) recently questioned the role of seedborne *P. betae* as the primary source of inoculum contributing toward infection and storage rot of sugar beet. They speculated that sources of inoculum other than seed were contributing toward initial plant infection and rot.

Our observations and results would tend to implicate *P. ultimum* as the primary pathogen of beet seedlings in New York vegetable soils. Isolations made from field grown table beet seedlings would support this contention (Abawi et al, *unpublished*). However, *P. betae*, undoubtedly a pathogen of table beet and sugar beet, is potentially capable of infecting beets at virtually any growth stage, thus giving this pathogen the advantage of time to act as a pathogen on older beets that have become resistant to infection by *P. ultimum*.

Our experiments have demonstrated a potential additional benefit of *L. arvalis* antagonism against *P. betae* as well as that previously reported against *P. ultimum* and *R. solani*. Also, it should be noted that diseased seedlings that were derived from metalaxyl-treated seed planted into *L. arvalis*-amended natural soils exhibited lower incidence of postemergence damping-off than the similar treatment lacking *L. arvalis*. This implies control of *R. solani*-induced disease in these experiments in agreement with the results of Odvody et al (14). Therefore, we feel that these experiments present evidence of the potential effectiveness of *L. arvalis* as an antagonist of three very different root rot pathogens of a single host.

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