Rhizoctonia Root Rot of Sugarbeet Unaffected by Herbicides *

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Received for Publication December 17, 1981

INTRODUCTION

In the past 10 years, there has been a gradual increase in root rot of sugarbeet (*Beta vulgaris L*.) caused by *Rhizoctonia solani* Kuhn in the United States (9), and several research approaches were taken to explain the observed increase. Recent isolates of the fungus were studied, but none was more virulent than those previously reported (14, and unpublished data). Nitrogen fertility levels, nitrogen source, and plant population density also had no effect on disease intensity in the field (10, 11), but soil deposition in sugarbeet crowns (16) and certain systemic insecticides (15) significantly increased disease severity. The latter two cultural practices, however, are not universal and cannot fully explain the overall increase in root rot incidence.

There are several reports in the literature of herbicide-plant disease interactions (3, 13). Such interactions have resulted in either an increase or a decrease in disease, depending on the pathogen-plant-herbicide system employed. Since annual herbicide use in the United States has increased dramatically (3), it is conceivable that some of these chemicals could be involved in the increase of sugarbeet root rot.

Few researchers have reported on the effect of herbicides on rot of mature sugarbeet roots. Altman and Ross (5) reported

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* Joint contribution of the Agricultural Research Service, USDA, the Colorado State University Experiment Station, and the Beet Sugar Development Foundation; supported in part by a grant from the Grower-Great Western Joint Research Committee, Inc. Published with the approval of the Director, Colorado State University Experiment Station, as Scientific Paper Series No. 2706. The authors are Research Plant Pathologist, Research Geneticist, and Research Plant Physiologist, ARS, USDA, Crops Research Laboratory, Colorado State University, Fort Collins, Colorado 80523, respectively. Numbers in parentheses refer to literature cited.

a higher incidence of *Rhizoctonia* in sugarbeets from some fields treated with pebulate, but it was not clear whether they meant seedlings or mature roots. No field experiments were conducted, but they obtained increased rhizoctonia damping-off of sugarbeet seedlings with pebulate and pyrazon in greenhouse tests. Whereas cycloate increased rhizoctonia damping-off of sugarbeet seedlings in the greenhouse (4), we found no effect of the chemical on rot of mature beets in the field.

The purpose of this paper is to report the absence of an effect by several contemporary herbicides on rhizoctonia root rot of mature sugarbeet in the field.

MATERIALS AND METHODS

Three irrigated field experiments were conducted over 3 years at Fort Collins, Colorado, to test the effect of herbicides diclofop methyl and ethofumesate applied preplant, and desmedipham, EPTC, metolacholor, phenmedipham, and trifluralin applied postemergence on the severity of rhizoctonia root rot. All herbicides were applied at recommended rates; only metolachlor is not registered for use on sugarbeet.

Plantings were made in areas heavily infested with *R. solani* to simulate commercial conditions. An additional preplant broadcast application of ground barley-grain inoculum (8) at 56 kg/ha was incorporated 10 cm deep into the experimental sites to assure uniformity.

Randomized complete block designs were used with four-row plots 6 m long and 56 cm between rows. Plants were thinned to 25/row about 35 days after planting in early April. Roots from the center two rows of each plot were harvested in early September of each year and individually rated for amount of rot on a scale of 0 to 7, with 0 = no rot and 7 = dead plant. A disease index (DI) was calculated as a weighted average based on the number of plants in each class. Plants in classes 0 through 3 were combined to calculate % harvestable roots; such roots would be recovered in the harvest. The arcsine transformation of percentage data was used for statistical analyses; however, actual percentages are presented in Table 1.

1978

Desmedipham and phenmedipham were applied as a mixture on an 18-cm band over the row when the plants were in the four-leaf stage before thinning. Both chemicals were used at 0.6 kg active ingredient (a.i.) applied in 373 L water/ha. *Rhizoctonia*-resistant sugarbeet cultivar FC 703 and susceptible commerical cultivar Mono-Hy A1 were used in this test. Treatments were replicated four times.

1980

Broadcast applications of ethofumesate at 2.2 kg a.i. and diclofop methyl at 1.7 kg a.i. in 373 L water/ha, alone and in combination, were incorporated before planting *Rhizoctonia*-susceptible sugarbeet cultivar Mono-Hy D2. Treatments were replicated four times.

1981

Trifluralin (0.6 kg a.i.), EPTC (3.4 kg a.i.), and metolachlor (2.2 kg a.i.), each in 373 L water/ha, were applied broadcast and incorporated 1 wk post thinning. Sugarbeet cultivars used in this test were resistant FC 703, susceptible Mono-Hy D2, and another commerical hybrid HH 32, having intermediate resistance to *Rhizoctonia*. Treatments were replicated three times.

RESULTS AND DISCUSSION

Analyses of variance of DI and % harvestable roots in each test showed significance only between cultivars in 1978, and among cultivars in 1981. In these tests, there also were no significant cultivar X herbicide interactions. Thus, the herbicides used in these studies, and in the 1980 test, had no significant adverse or beneficial effects on rhizoctonia root rot (Table 1).

Except for our previous study with cycloate (10), all experimental studies reporting adverse effects of herbicides on *Rhizoctona*-induced disease in sugarbeet have been performed in the greenhouse or environmental growth chambers with seedlings (1, 2, 4, 5, 6). Further, most reported increases in root

Table 1. Means for disease index (DI) and % harvestable (%H) roots in three tests on the effect of herbicides on rhizoctonia root rot in field-grown sugarbeet.[a]

Herbicide (rate in kg a.i./ha)	Application[b]	Cultivar[c] FC 703 DI	%H	Mono-Hy A1 DI	%H	HH 32 DI	%H	Mono-Hy D2 DI	%H
1978 Test									
Demsmedipham (0.6)+ phenmedipham (0.6)	PE	2.7	75.6	5.9	15.6	–	–	–	–
No herbicide		2.5	76.2	5.8	16.9	–	–	–	–
1980 Test									
Ethofumesate (E; 2.2)	PP	–	–	–	–	–	–	3.4	50.3
Diclofop methyl (D; 1.7)	PP	–	–	–	–	–	–	3.8	44.2
E (2.2) + D(1.7)	PP	–	–	–	–	–	–	3.4	49.6
No herbicide		–	–	–	–	–	–	3.8	46.5
1981 Test									
Trifluralin (0.6)	PE	2.7	72.1	–	–	2.9	64.2	3.8	51.1
EPTC (3.4)	PE	2.2	80.6	–	–	3.4	57.2	3.2	63.8
Metolachlor (2.2)	PE	2.2	81.3	–	–	4.5	34.4	3.3	59.6
No herbicide		1.9	82.6	–	–	4.1	47.2	4.4	42.4

[a] DI based on a scale of 0 to 7, with 0 = no disease and 7 = plant dead; % harvestable calculated by combining DI classes 0 through 3. Means of four replications in 1978 and 1980; three replications in 1981. Means within columns and within years were not significantly different at $P = 0.05$.

[b] PE + postemergence, PP = preplant incorporated. [c] FC 703 = resistant; Mono-Hy A1 and D2 = susceptible; HH 32 = intermediate resistance.

disease due to *Rhizoctonia*-herbicide-plant interactions in other crops also involve seedlings rather than mature plants (3). The negative results with the herbicides used in our experiments suggest that predisposing factors or mechanisms (1, 2) are not effective once the sugarbeets progress beyond the seedling or juvenile stage. That most plants also show "mature plant resistance" to damping-off organisms is well documented in the literature.

We did not observe any rhizoctonia damping-off in our field plots with preplant applications of ethofumesate or diclofop methyl, alone or in combination, or when cycloate was used (10). Sugarbeets were purposefully sown early in April when soil temperatures below 12° C favored growth of the plants over that of *R. solani* (12). Growers following a similar practice should be able to avoid possible increased rhizoctonia damping-off due to predisposition by herbicides; however, later plantings may be affected by such interactions.

Fungi other than *R. solani* also can cause seedling damping-off in sugarbeet. Like *R. solani*, *Aphanomyces cochlioides* and *Pythium aphanidermatum* cause seedling disease when soil temperatures are relatively high; however, *P. ultimum*, *P. debaryanum*, and *Phoma betae* are active in cool soils (7). Studies are needed to determine if these pathogens can induce more sugarbeet damping-off at various soil temperatures when contemporary herbicides are applied preplant. However, there appears to be no evidence that herbicides used in sugarbeet culture play a role in the observed increase of rhizoctonia root rot in mature plants.

SUMMARY

An increased incidence of sugarbeet *(Beta vulgaris)* root rot caused by *Rhizoctonia solani* somewhat paralleled increased herbicide use in the United States. To determine the existence of a possible disease-herbicide interaction, experiments were conducted on field sites heavily infested with the pathogen. In 1978, desmedipham and phenmedipham (each at 0.6 kg a.i./ha) were applied when sugarbeet cultivars FC 703 (resistant to *R. solani*) and Mono-Hy Al (susceptible to *R. solani*) were

in the four-leaf stage. In 1980, preplant broadcast applications of ethofumesate (2.2 kg a.i./ha) and diclofop methyl (1.7 kg a.i./ha), alone and in combination, were incorporated before planting *Rhizoctonia*-susceptible cultivar Mono-Hy D2. Trifluralin (0.6 kg a.i./ha), EPTC (3.4 kg a.i./ha), and metolachlor (2.2 kg a.i./ha) were applied broadcast and incorporated 1 week post thinning to cultivars FC 703, Mono-Hy D2, and HH 32 (intermediate resistance to *R. solani*) in 1981. Individual roots were harvested and rated for rot about 5 mo after planting; a disease index and % harvestable roots were calculated on a plot basis. No significant adverse or beneficial effects of the herbicides on the incidence or severity of root rot were detected in any test. Thus, there appears to be no evidence that herbicides used in this study played a role in the observed increase in rhizoctonia root rot of mature sugarbeet.

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