The Effect of Seed Infected with *Phoma betae* on Rot and Sucrose Yield of Stored Sugar Beet

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ABSTRACT

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Sugar beet seeds of cultivar US H20 infected with *Phoma betae* were treated with recommended amounts of fenaminosulf, a mixture of pentachloronitrobenzene and 5-ethoxy-3-(trichloromethyl)-1,2,4-thiadiazole, prochloraz, or thiram. Stand counts for any of the seed treatments were not improved over nontreated seed under field conditions in 1977 and 1978. The thiram-soak treatment in 1977, and all treatments in 1978, reduced *Phoma betae* infection in surviving seedlings. The most effective treatments were thiram soaking, and prochloraz. All seed treatments in 1978 resulted in less rot in roots stored for 150 days at 4–6 C compared with storage rot in roots from nontreated seed. Storage rot was positively correlated with numbers of surviving seedlings infected with *P. betae*. Regression analysis showed a 0.04% increase in storage rot for each 1% increase in infected seedlings. The correlation coefficient was significant ($P = 0.05$) but low ($r = 0.15$), indicating that sources of *P. betae* inoculum other than the seed were contributing toward infection and rot. Therefore, seed treatments to reduce infection by *P. betae* would result only in partial reduction of storage rot, insufficient to increase recoverable white sugar per ton of beets.

In 1915, Edson (9) showed that even though some sugar beet seedlings from seed infected with *Pleospora bjoerlingii* Byford [imperfect state = *Phoma betae* (Oud.) Frank] survived seedling disease and developed into normal, healthy roots, the pathogen still inhabited the crown area of the root and became active and caused rot after the roots were placed in storage. Levels of infection can reach over 90% (5). The amount of seed infected with *P. betae* is variable because infection is affected by wet weather when the seed crop is harvested. Therefore, it is probable that the use of seed lots with a low level of infection by *P. betae* will result in less storage rot and sucrose loss than those with high levels of infection. The routine use of seed treatments effective against *P. betae* should reduce seedling disease and subsequent storage rot. This theory was tested and the results are reported here.

MATERIALS AND METHODS

Sugar beet (*Beta vulgaris* L. seeds 'US H20') were obtained from Monitor Sugar Co., Bay City, MI 48706. The seeds were produced near Salem, OR, by the West Coast Beet Seed Co. One seed lot with over 95% infection by *P. betae* was used in 1977. A second seed lot with 25% infection also was used in 1978.

Seeds were treated with thiram (bis[dimethylthiocarbamoyl] disulfide) two ways in 1977: standard rate of 224 g per 45 kg seed; and soaking seed 24 hr in a 0.2% (w/v) suspension of thiram at 30 C (11). Control treatments were nontreated seed, and a seed soak in water for 24 hr at 30 C. Seeds were planted in two-row plots 9 m long. The four treatments were replicated eight times in a randomized complete block design. Stand counts were taken from the two rows about 3 wk after planting and expressed as an average for 9 m of row for each replicate. When the plots were thinned about 4 wk after planting, 10 seedlings were randomly collected from each plot and the hypocotyl and root portions were plated on an agar medium selective for *P. betae* (2). The plates were examined 8–10 days later for seedlings infected with *P. betae*.

Three additional seed treatments were used in the 1978 test: fenaminosulf was applied as a slurry at 0.25 g per 100 g of seed (6 oz of 70% formulated wettable powder per 100 pounds of seed); a mixture of pentachloronitrobenzene and 5-ethoxy-3-(trichloromethyl)-1,2,4-thiadiazole (PCNB-ETMT) at 0.8 ml per 100 g seed (12 oz formulated emulsifiable concentrate per 100 pounds of seed); and prochloraz at 0.25 ml of a 25% solution per 100 g of seed.

The five seed treatments and nontreated control (total of six treatments) were assigned in a randomized complete block design with the two seed lots comprising a split plot. There were 16 replicates for a total of 192 plots. The four-row plots were 9 m long. Stand counts were made, and seedlings and roots were harvested (as in the 1977 test) from the two middle rows.

The roots from the 192 plots were harvested by hand, washed, and grouped into three lots of about 9–11 roots each. One lot was processed immediately to determine harvest quality, a second lot was dipped in a 1,500 ug/ml suspension of thiabendazole before storage, and a third lot was not treated with thiabendazole before storage. Roots of both stored lots were held in perforated plastic bags at 4–6 C and 100% relative humidity for 150 days, and then assessed for storage rot by cutting out and weighing the rotted tissue. Measurements of rotted tissue were made for the crown and tap portions of the root. The rotted and nonrotted tissue was combined for quality measurements.

Sugar beet quality parameters that were measured at harvest and after storage were: sucrose content, clear juice purity (CJP), raffinose, and invert sugars. The values of these components were used to calculate recoverable white sugar per ton of beets (RWST). The RWST was calculated with an assumed factory loss of 0.03% and a molasses purity of 62.5% (13). Sucrose in juice prepared by the cold digestion method (6) was measured with a polarimeter. After clarification with aluminum sulfate, CJP was determined according to the method described by Dexter et al (7). Raffinose was measured in juice from stored beets by using an enzyme system (galactose oxidase) developed by Yellow Springs Instrument Co., Yellow Springs, OH 45387, USA. The enzyme was immobilized in a polycarbonate membrane fitted on a silver and platinum electrode in a small reaction chamber. The clarified juice (25 μl) was injected into the reaction chamber. The instrument was calibrated with a raffinose standard. Raffinose accumulates in sugar beets during storage, especially below 5 C (13); it is dextrorotatory and contributes to the polariscope reading. Invert sugars were

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determined by the 3, 5-dinitrosalicylic acid method (1); thin-juice polarimeter values were adjusted for accumulations of invert sugar and raffinose. The cold digestion extract was corrected for weight loss, raffinose, and invert sugar.

RESULTS

The thiram seed treatments in the 1977 test did not improve stand counts, but did reduce seedling infection (Table 1). In 1978, five different seed treatments did not improve stand counts over those for nontreated seed (Table 2). The analysis of variance and F test, however, showed a significant difference ($P = 0.05$) in average reduction in stand count between seed with a high and low incidence of *P. betae*. As in 1977, seed treatments in 1978 affected the percentage of surviving seedlings infected with *P. betae*; thiram-soaking, thiram, and prochloraz proved to be the better treatments. Differences in percentage of infected seedlings between the two seed lots for any particular treatment were not statistically significant. Even fenaminosulf, which is not effective against *P. betae*, reduced seedling infection, but only in the most heavily infected seed lots. Thiabendazole used as a root dip did not reduce storage rot in this test. The average rot across all treatments was 10.1 with, and 10.2% without, the fungicide.

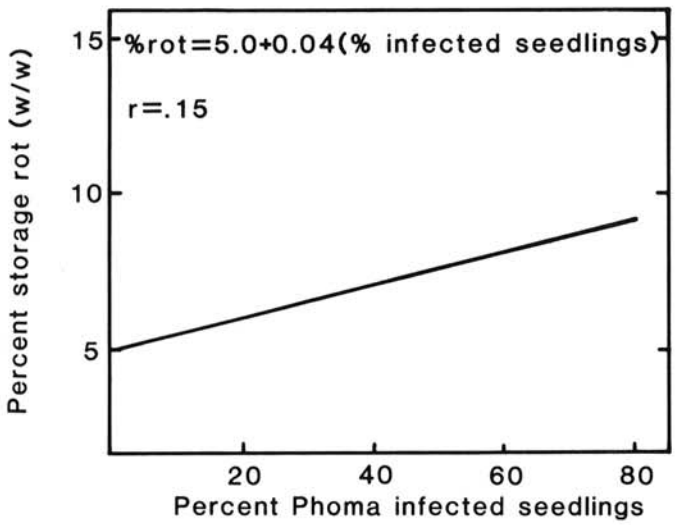

Fig. 1. The effect of the level of infection by *Phoma betae* in surviving sugar beet seedlings on subsequent rot of roots stored 150 days at 5 C and 100% relative humidity. The analysis was based on 192 paired observations.

TABLE 1. Stand counts and amount of seedling infection by *Phoma betae* after thiram treatment of sugar beet seed of cultivar US H20 in 1977. About 95% of the seed was naturally infected with *P. betae*

Seed treatment	Stand count (no. per 9 m of row)	Phoma-infected seedlings (%)
Thiram soak[x]	36 a	16 b
Thiram, 224 g/45 kg of seed	38 a	41 a
Water soak[x]	38 a	36 a
No treatment	40 a	49 a

[x]Seed was soaked 24 hr in 0.2% thiram aqueous suspension or water at 30 C. Means of eight replications; means within a column followed by a common letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

All seed treatments reduced storage rot from the 95% infected seed lot, while only PCNB-ETMT and the thiram-soak significantly reduced storage rot in the other seed lot (Table 2). Most of the storage rot occurred in the crown tissue. Storage rot in crowns of roots from untreated seed averaged 7.2% which was significantly higher ($P = 0.05$) than that in roots from treated seed. The range of crown rot in roots from treated seed was 5.2–6.0%. Storage rot in tap root tissue from untreated seed averaged 6.0% and was significantly higher ($P = 0.05$) than was tap root rot in beets grown from treated seed. The range in tap root rot from treated seed was 3.8–4.4%. The amount of storage rot among the treated seeds did not differ significantly and seed treatments had no effect on RWST.

Analyses of data for storage rot and percentage of infected seedlings from 192 plots showed a significant ($P = 0.05$) positive correlation between infected seedlings and total rot (crown plus tap root) ($r = 0.15$). The correlation for total rot indicated a 0.04% increase in storage rot for each 1% increase in seedlings infected with *P. betae* (Fig. 1).

DISCUSSION

Edson (8) reported that seedborne *P. betae* inhabited crown tissues of the mature sugar beet root. Recent work has shown that treatment of sugar beet roots with thiabendazole 24 hr before inoculation with *P. betae, Botrytis cinerea,* and *Penicillium claviforme* resulted in reduced rot (12) and reduced sucrose loss (4). In this case, the fungicide functioned as a protectant. The roots used in our study probably were field-infected deep in the crown and the pathogen escaped contact with the thiabendazole.

The effects of fungicidal seed treatments confirmed past observations of several tests in the Red River Valley since 1970 (W. M. Bugbee, *unpublished*): seedling disease pressure seldom was great enough to warrant the use of a seed treatment. Even the thiram-soaked treatment, suggested as a replacement for banned mercury-containing compounds (11), did not give stand counts greater than untreated seed. Seed treatments, however, had a dramatic effect on the number of surviving seedlings infected with

TABLE 2. The effect of seed treatments on seedling disease, storage rot, and recoverable white sugar per ton (RWST) of roots of the sugar beet cultivar US H20 with seed naturally infected with *Phoma betae* at two levels[x]

Phoma-infected seed (%)	Seed treatment	Stand count (no.)	Phoma-infected seedlings (%)	Storage rot (w/w) (%)	RWST	
					Harvest (kg)	Stored (kg)
95	None	15 a	44 a	14.0 a	204 a	122 ab
	Fenaminosulf	14 a	27 b	9.4 bc	197 a	136 a
	PCNB-ETMT	14 a	23 bc	9.7 bc	196 a	118 b
	Thiram	14 a	13 cde	9.2 c	199 a	130 ab
	Thiram soak	13 a	7 efg	9.2 c	196 a	132 ab
	Prochloraz	13 a	4 fg	8.1 c	198 a	136 a
	Mean	14*				
25	None	18 a	28 b	12.4 ab	200 a	124 ab
	Fenaminosulf	17 a	20 bcd	10.8 bc	196 a	127 ab
	PCNB-ETMT	19 a	16 cde	9.0 c	198 a	128 ab
	Thiram	17 a	11 defg	10.4 bc	197 a	135 a
	Thiram soak	18 a	5 efg	9.2 c	198 a	131 ab
	Prochloraz	19 a	2 g	10.4 bc	199 a	124 ab
	Mean	18*				

[x]Means of 16 replications; means within a column followed by a common letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test. Asterisk, *, indicates significant difference ($P = 0.05$) according to the F test.

P. betae. A second test in a growth chamber showed fenaminosulf to be ineffective in reducing Phoma seedling infection. There is no explanation for the apparent reduction of Phoma seedling infection by fenaminosulf in the field test.

The reduction in infected seedlings was accompanied by a reduction in storage rot, but the 6- to 10-fold decrease of infected seedlings did not result in a corresponding magnitude of rot reduction. The low, yet statistically significant, positive correlation between infected seedlings and storage rot indicated that sources of inoculum other than seed played a role in initiating storage rot. Koch (10) reported that soil inoculum plays an important role in infection of sugar beet seedlings even though the seeds have been treated with a fungicide. Other inoculum sources could have been root-to-root contact in storage, and windblown inoculum in the field. Infested storage yard soil (3), and roots from infested fields encountered under commercial conditions, but not in this test, probably would lower the correlation further. Therefore, an otherwise efficient seed treatment would be expected to give only a partial and unacceptably low reduction of storage rot.

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