AN ABSTRACT OF THE DISSERTATION OF

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Mary L. Powelson

For the past several years vegetable growers in Oregon's Willamette Valley have experienced reduced yields in their sweet corn plantings. We conducted studies to 1) describe the symptomology and etiology of the disease, 2) test a root rot rating system we developed to evaluate factors in the disease syndrome, and 3) evaluate the effect of soil applied herbicides on severity of root rot. Plant sampling from commercial fields indicated root rot, not stalk rot, is the disease affecting sweet corn plantings. Fumigation with methyl bromide and chloropicrin reduced root rot severity 68-89% at harvest and increased yields by up to 50%, indicating the primary cause of root rot is soilborne and biotic. Pathogenicity trials showed several organisms cause root rot, and a rating system we developed for root rot indicates symptoms differ among pathogens. *Pythium arrhenomanes* reduced dry weight and caused limited necrosis of the radicle and nodal roots, but did not cause rot of the mesocotyl. *Phoma terrestris* and *Drechslera* sp. caused rot of all three components of the root system and reduced plant dry weight. *Fusarium graminearum* primarily caused rot of the mesocotyl and reduced dry weight. *F. oxysporum* did not cause root rot or reduce plant biomass. The rating system also

was used to evaluate the effect of four herbicides, two of which contain safeners, and a safener alone on root rot of sweet corn grown in soil artificially infested with *Pythium arrhenomanes*, *Phoma terrestris*, *Drechslera* sp., or *Fusarium graminearum*. In soil infested with *P. arrhenomanes*, dry weight of sweet corn seedlings was reduced when treated with the herbicide/safener combination EPTC + R-29148. EPTC + R-29148 also increased severity of rot of the nodal roots caused by *Phoma terrestris*. In contrast, in soil infested with *Drechslera* sp., metolachlor increased severity of rot of the radicle and nodal roots and decreased plant dry weight. Metolachlor also reduced biomass of plants grown in soil infested with *F. graminearum*. Atrazine, metolachlor + benoxacor and benoxacor alone generally had no effect on plant dry weight or root rot of sweet corn seedlings grown in soil infested with any of our pathogens. Our results indicate the effect of soil applied herbicides on root rot of sweet corn is pathogen species dependent.

Sweet Corn Decline Syndrome in Oregon's Willamette Valley

by
Elisabeth V. Hoinacki

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I understand that my dissertation will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my dissertation to any reader upon request.

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Elisabeth V. Hoinacki, Author

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CONTRIBUTION OF AUTHORS

Mary L. Powelson contributed ideas for experiments conducted in Chapters 2-4 and provided guidance for those procedures. Robin Ludy helped with the design and implementation of the fumigation trial and assisted with the disease dynamics study in Chapter 2. R. Edward Peachey assisted with the experimental design and herbicide applications in Chapter 4.

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Chapter 1

Introduction

The discipline of plant pathology has its roots in the devastating epidemics of late blight of potato, which contributed to the widespread famine in Ireland in the 1840s. Much of the early work of plant pathologists focused on such dramatic diseases—ergot of rye, rusts and smuts of grains and cereals, and powdery mildew of grapes. Less studied were the not so obvious plant diseases caused by organisms residing below ground—root and crown rots and vascular wilts. Even when diseases caused by soilborne pathogens became more recognized, the complexity of the soil ecosystem with its biological, chemical, and physical components hindered our understanding of them.

It wasn't until nearly 100 years later, with the work of S.D. Garrett in the 1930s that concepts central to the ecology of soilborne pathogens started to develop. For the next few decades Garrett and others devoted themselves to understanding factors, both biotic and abiotic, involved in the development of root diseases and how those factors interact to influence disease severity. At the same time, new techniques and tools were being developed to aid research on the ecology of soilborne pathogens and books on methodology were published periodically (Johnson et al, 1959; Johnson and Curl, 1972; Dhingra and Sinclair, 1985; Singleton et al, 1992).

When growers in Oregon's Willamette Valley wanted to know the cause of a disease that was affecting their sweet corn yields, they turned to researchers at Oregon State University. In many cases an examination of the symptoms of the diseased plant, the isolation of a suspected pathogen, and a review of diagnostic manuals is sufficient in naming the disease. However, root disease diagnosis can be more complicated. Root rots occur on a variety of plants and are reported to be caused by numerous organisms. In many cases, more than one organism is implicated in disease on a particular crop. The Compendium of Corn Diseases (1999) lists a number of species from more than four genera as causing root rot. Many root rot pathogens also have wide host ranges. To establish causality, plant pathologists test Koch's Postulates (Agrios, 1988). Such pathogenicity tests usually are conducted in small pots studies in the greenhouse. When studying root rots, which by nature occur in a complex environment, results obtained from artificially created environments can be misleading. Inoculum dose-disease response relationships are one of the basic principles of plant pathology (Van der Plank, 1975). Testing the density dependent relationship between disease and organisms associated with symptoms can help identify primary pathogens.

One basis for research on plant pathogens, particularly those of agricultural crops, is the desire to reduce their damaging effect. With root rots, abiotic factors often influence severity of disease and the manipulation of such factors can result in an effective disease manangement strategy. In his discussion of the princples of root disease control, Garrett (1970) states that for plant

pathologists to see the results of their work realized in increased crop yields, "a thorough familiarity with the ecology and plant husbandry of the particular crop is absolutely essential; for root-disease control, appropriate modifications or innovations in crop husbandry practice have furnished the foundations for success..." Research on the effect of crop rotation, tillage, and fertilizer and herbicide use on root disease severity has illustrated the success of this approach.

In Chapter 2, I present our study of the symptomology and etiology of sweet corn decline syndrome affecting plantings in the Willamette Valley. Chapter 3 outlines a method we developed for quantifying root rot severity on sweet corn seedlings. Based on our understanding of the development and cause of root rot as outlined in Chapter 2 and using the disease rating system described in Chapter 3, we examined commonly applied herbicides for their effect on the severity of root rot (Chapter 4).

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Chapter 2

Sweet Corn Decline Syndrome in Oregon's Willamette Valley

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ABSTRACT

For the past several years, vegetable growers in Oregon's Willamette Valley have experienced reduced yields in their sweet corn plantings. We conducted studies 1) to describe the development of disease symptoms over time, 2) to estimate the effect of disease on plant growth and yield, 3) to identify organisms associated with disease and, 4) complete Koch's Postulates to determine the causal organism(s). Plant sampling from commercial fields indicated root rot, not stalk rot, was the disease affecting sweet corn plantings. Lesions appear on the radicle by 4-6 wk post-planting and on nodal roots by 7-10 wk post-planting. Root rot developed slowly until silking at which time root necrosis increased rapidly. When root rot was severe, secondary symptoms of leaf chlorosis and necrosis occurred, ears exhibited poor tip fill, and the kernels were dimpled. Fumigation with methyl bromide (67%) and chloropicrin (33%) (450 kg/ha) in two fields with a history of root rot reduced disease severity by 68-89% at harvest and increased yields by up to 50%, indicating the primary cause of root rot was soilborne and biotic. Numerous fungi and oomycetes were isolated from symptomatic root tissue and a Koch's Postulates/inoculum dose-disease response study indicated several organisms cause disease and symptoms differ

among pathogens. *Pythium arrhenomanes* reduced plant dry weight as a result of root pruning and caused limited necrosis of the radicle and nodal roots, but did not cause rot of the mesocotyl. *Phoma terrestris* and *Drechslera* sp. caused rot of all three components of the root system and reduced plant dry weight. *Fusarium graminearum* primarily caused a rot of the mesocotyl and reduced dry weight. Density dependent relationships for root disease caused by these pathogens was established. *F. oxysporum* did not cause root rot or reduce dry weight.

INTRODUCTION

Average yield of sweet corn (*Zea mays* L.) grown for processing in Oregon in 2001 was 19.6 metric tons/ha, the lowest since 1977 (Oregon Agricultural Information Network, OAIN). Over the past several years, many growers in the Willamette Valley have experienced a yield decline in their sweet corn crops. While many factors contribute to the productivity of a crop, anecdotal evidence from growers suggested poor yields were caused by the "firing" disease. "Firing," the chlorosis and necrosis of leaves starting at the base of the plant and progressing upward, typically occurs late in the growing season, from silking until harvest. In severely affected fields, ears are often smaller and exhibit poor tip fill, and kernels may be dimpled, reducing both yield and quality of the crop.

Oregon growers produce approximately 350,000 tons of sweet corn for processing annually, ranking the state 4th nationally (OAIN). Most sweet corn in Oregon is grown in the Willamette Valley where it is one of two crops that

support the vegetable processing industry. The poor yields experienced by many growers makes growing sweet corn unprofitable. The processing industry, however, depends on it and many growers are required to grow sweet corn to get contracts for other, more profitable crops. With the support of the Oregon Processed Vegetable Commission, an investigation into the nature of the "firing" disease became a top priority.

Based on surveys of symptomatic and nonsymptomatic fields in the mid 1990's, root rot was identified as the most prevalent disease symptom associated with leaf firing late in the season. Crown rot was observed occasionally in plants with severely rotted roots but stalk rot was seen rarely (Powelson et al, unpublished). Not documented, however, was when in the phenology of the crop that root rot was initiated and how it developed during the growing season.

Root rot of corn occurs throughout the United States and around the world. Many fungi and oomycetes have been implicated in root rot of corn including species of *Pythium, Fusarium, Phoma, Helminthosporium, Rhizoctonia, Phialophora*, and *Trichoderma* (Compendium of Corn Diseases, 3rd Ed., 1999; Deep and Lipps, 1996; Hellinga et al, 1983; Johann et al, 1928; Mao et al, 1998; Rao et al, 1978; Shepard et al, 1967; Sumner et al, 1982; Sumner et al, 1990; Sutton, 1972; Warren and Kommedahl, 1973; Whitney and Mortimore, 1961). In some cases, individual species are attributed with causing disease but more often pathogen complexes have been implicated. Often one organism will be considered the primary pathogen causing root rot, and others will be labelled with

the role of secondary invaders or opportunistic pathogens. Such is the case with red root rot of corn, caused primarily by *Phoma terrestris* E.M. Hans. but also involving species of *Pythium* and *Fusarium* (Leslie et al, 1990; Mao et al, 1998; Sumner et al, 1990; Warren and Kommedahl, 1973). Disease caused by different microorganisms varies according to the growth stage of the plant. For example, *Pythium ultimum* Trow and *Fusarium graminearum* Schwabe cause damping-off of corn, whereas *P. graminicola* Subr. and *F. oxysporum* Schlecht. emend. Snyd. & Hans. cause root rot (Mao et al, 1997; Rao et al, 1978; Warren and Kommedahl, 1973). In addition, different species of the same genus may cause disease in different geographic areas. *Pythium irregulare* Buisman is associated with root rot of corn in the northeast United States, whereas *P. arrhenomanes* Drechs. causes root rot of corn in the Midwest (Deep and Lipps, 1996; Mao et al, 1998).

The objectives of our work were fourfold: 1) to describe the development of disease symptoms over time, 2) to estimate the effect of disease on plant growth and yield, 3) to identify organisms associated with disease and, 4) complete Koch's Postulates to determine the causal organism(s).

METHODS

Disease dynamics study. In 1997, four fields of sweet corn cv Golden Jubilee, two (1 and 2) near Stayton, Marion County, and two (3 and 4) near Walterville, Lane County, OR and in 1998, two fields of cv Golden Jubilee (5 and

6) near Stayton and one field of cv Supersweet Jubilee (7) near Hubbard, Marion County were sampled. Plants were sampled beginning 4 wk post-planting and every 2 wk (1997) or 3 wk (1998) until harvest. At each sampling date, 10 plants per field were randomly selected, dug and the rootballs washed and assessed for severity of root rot on a scale of 0-100%. Foliar symptoms of chlorosis and necrosis were noted and at harvest ears were examined for tip fill and dimpling of kernels.

Soil fumigation study. Soil fumigation plots (9 x 18 m) were established in 1999 in two commercial sweet corn fields (8 and 9) with a history of root rot. Both fields were located near Stayton, OR and were different than fields in the disease dynamics study. Plots were fumigated with methyl bromide (67%) and chloropicrin (33%) at 450 kg/ha during the first week of June and tarped for 1 wk. Nontreated plots served as the control. Treatments were replicated four times and arranged in a randomized block design. Two weeks after tarps were removed corn seed, cv GH 2684 (field 8), or cv Golden Jubilee (field 9) was planted at 66,500 plants/ha in rows on 1 m centers, 8 rows per plot. All plots were treated with standard grower practices.

At 4 wk, 10 plants and at 8 wk and at harvest three plants were randomly sampled from each plot. Rootballs were washed and assessed for necrosis on a scale of 0-100%. Number of harvestable ears and total ear weight for 3 m from each of two rows in the middle of each plot were recorded. Analysis of variance was conducted to evaluate the effect of fumigation on root rot at the three sample

dates, and on number of ears and yield at harvest. Means were separated using Fischer's protected least significant difference ($P \leq 0.05$) (SAS Version 8.1, SAS Insitute, Cary, NC).

Fungal isolations. Isolations for *Fusarium* and *Pythium* were made from symptomatic roots of sweet corn plants sampled in the disease dynamics study and the fumigation study. Roots were surface disinfested in 10% bleach for 1 min, rinsed in distilled water, and ten 1-cm long root segments per plant were plated onto Nash-Snyder medium (NSM, Nash and Snyder, 1962; Nelson et al, 1983) for the isolation of *Fusarium* and water agar (WA; Difco Laboratories, Detroit, MI) amended with 25 µl/ml rifamicin (Sigma Chemical Co., St. Louis, MO) and 100 µg/ml ampicillin (Sigma Chemical Co., St. Louis, MO) for the isolation of *Pythium*. Plates were incubated at room temperature under fluorescent lights (NSM) or in the dark (WA) for 2 to 4 days. Colonies of Fusaria were subcultured to carnation leaf agar (CLA, Burgess et al, 1988; Fisher et al, 1982) and potato dextrose agar (PDA; Difco Laboratories, Detroit, MI) and identified to species (Nelson et al, 1983). Pythia were subcultured to WA and identified to species (Waterhouse, 1967; Van der Plaats-Niterink, 1981).

In 2000, fungal isolations were made from roots of corn plants grown in the greenhouse in naturally infested field soil. Soil was collected from the two fields included in the fumigation study, seived through a 1 cm screen to remove rocks and clods and placed in 550 ml soil tubes (6.5cm x 25cm). Sweet corn seeds, cv Golden Jubilee, were surface disinfested in 10% bleach for 5 min, rinsed

three times in distilled water, and planted into soil 2.5 cm deep. Five plants per field soil were sampled at each of three dates (95% emergence, 1 wk and 2 wk post-emergence). Plants were removed from soil tubes and roots were washed under running tap water over a 2 mm sieve (No. 10, W.S. Tyler, Inc., OH). Symptomatic root pieces 1 cm in length were surface disinfested for 1 min in 10% bleach and rinsed in distilled water before plating on WA + 100 µg/ml streptomycin (Sigma Chemical Co., St. Louis, MO), cornmeal agar (CMA: Difco Laboratories, Detroit, MI) + 100 µg/ml streptomycin (Sigma Chemical Co., St. Louis, MO), or NSM. Isolates were subcultured to WA, CMA, PDA, and CLA and identified based on colony morphology and reproductive structures. Two isolates of a non-sporulating fungus were identified by analysis of the ITS rDNA. Fungal tissue was grown in 25% potato dextrose broth (PDB; Difco Laboratories, Detroit, MI), collected, lyophilized, and ground with liquid nitrogen. Genomic DNA was extracted by a variation of a standard organic extraction method and quantified using gel electrophoresis. The Polymerase Chain Reaction (PCR) was run using primers ITS4 and ITSF and the PCR products were cleaned using the QIAquick PCR Purification Kit Protocol. Products were submitted to the Oregon State University Center for Gene Research and Biotechnology's Central Sequencing Lab for sequencing. Sequences were compared to others in the NCBI database using a BLAST search.

Koch's Postulates study. Individual isolates of *Pythium arrhenomanes, Phoma terrestris, Fusarium oxysporum, F. graminearum,* and *Drechslera* sp.

were grown on WA at room temperature for 1 wk. Sand (97 g), cornmeal (3 g), and water (28 ml) were placed in 250ml Erlenmyer flasks and autoclaved at 121 C for 1 hr on each of two consecutive days (Tuite, 1969). For each isolate, two 2-mm plugs taken from the colony edge were added to the flasks. Flasks were incubated at room temperature under fluorescent lights for 3-4 wk until the medium was colonized. Inoculum was removed, air dried and stored at 8 C until use.

Field soil was collected and sieved through a 1 cm screen to remove rocks and clods before pasteurizing at 90 C for 1 hr on each of two consecutive days. Inoculum was added to pasteurized soil at four rates (1, 2, 10, or 100x). Amount of inoculum for each pathogen was determined in screening trials (Appendix A). To pasteurized field soil, 13 isolates of *P. arrhenomanes* (97102-2d, 97101-2b, 97102-1c, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9945-1a, 9942-11, 9966-5, 9947-7, 9947-17) were mixed and added at 0.01, 0.02, 0.10, or 1.0 g inoculum/500 ml soil, three isolates of *Phoma terrestris* (0015-9b, 0015-6a, 0015-6c) were mixed and added at 0.05, 0.1, 0.5, or 5.0 g inoculum/500 ml soil, five isolates of *F. oxysporum* (0101-1, 0101-5, 0101-6, 0101-7, 0101-8), two isolates of *F. graminearum* (0016-3a, 0016-8a) and two isolates of *Drechslera* sp. (BPP-2, 0016-7) were each mixed and added at 0.02, 0.04, 0.2, or 2.0 g inoculum/500 ml soil. Inoculum and soil for each treatment were mixed in a V-shaped shell mixer for 20 min and placed in 550 ml soil tubes (6.5cm x 25cm) on a greenhouse bench. Noninfested soil served as the control. Treatments were arranged in a

randomized design and replicated 10 times. Sweet corn seeds, cv Golden Jubilee, were surface disinfested in 10% bleach for 5 min, rinsed three times in distilled water, and planted 2.5 cm deep. Soils were kept moist and after emergence, plants were fertilized once weekly with a water soluble plant food (N-P-K, 20-20-20) at the label rate. At the 6th leaf stage plants were harvested, shoots were cut at the soil surface, and bagged. Roots were collected on a 0.5 cm screen, washed under running tap water, and the severity of root rot was rated on each of the three components of the root system: mesocotyl, 0=healthy, 1=discrete lesion present, 2=100% necrotic; radicle, 0=healthy, 1=at least one lesion present but less than 10% of root necrotic, 2=10-50% necrotic, 3=51-99% necrotic, 4=100% necrotic; nodal roots, 0=healthy, 1=5-10% necrotic, 2=11-25% necrotic, 3=26-50% necrotic, 4= >50% necrotic (Chapter 3). Five 1-cm long symptomatic root pieces per two plants per treatment were surface disinfested in 10% bleach for 1 min, rinsed in distilled water, and plated on WA. Nonsymptomatic root tissue was plated from the control treatment. Shoots and roots were oven dried at 60 C and weighed. To complete Koch's Postulates, isolates recovered from symptomatic root tissue were compared to the original isolates.

The relationship between severity of rot of the mesocotyl, radicle, and nodal roots and plant dry weight and amount of inoculum was evaluated with linear regression using SAS Version 8.1 (SAS Institute, Cary, NC). When relationships were not significant, data for inoculum treatments were combined and compared to the noninfested control with t-tests at the 5% probability level

($P \leq 0.05$). A partial repeat of this experiment was conducted as part of a larger study (Chapter 4).

RESULTS

Disease dynamics. Lesions were first observed on the radicle, or primary root, at 4-7 wk post-planting, and by 7-10 wk scattered lesions were apparent on the nodal roots. Root rot developed slowly until silking (10-12 wk post-planting), at which time root necrosis increased rapidly in some fields (Fig. 2.1A and B). At harvest, severity of root rot was on average four times greater in fields 1 and 2 compared to fields 3 and 4 in 1997 and in fields 5 and 6 compared to field 7 in 1998. When root rot was severe, secondary symptoms of leaf chlorosis and necrosis, also known as firing, occurred. Ears exhibited poor tip fill and the kernels were dimpled (data not shown). When root rot was severe, crowns were often necrotic. Discoloration of nodal tissue close to the crown was occasionally observed. Stalk tissue, however, remained white and healthy.

Soil fumigation study. The severity of root rot was reduced significantly in fumigated versus nonfumigated plots at all three sample dates in both fields (Table 2.1). Fumigation reduced the severity of root rot at harvest by 89 and 68% in fields 8 and 9, respectively. In the nonfumigated plots, numerous lesions were apparent on the radicle at 4 wk and on the nodal roots at 8 wk. By harvest the lesions had expanded and coalesced, resulting in a necrotic rootball. Rootballs of

corn grown in fumigated plots, however, remained white and had numerous fibrous fine roots.

At harvest, the number of ears per plot was 45% greater in the fumigated compared to the nonfumigated plots in field 8 ($P<0.005$), whereas in field 9 the effect of fumigation on number of ears was not significant ($P=0.18$). Yield was increased significantly by 58 and 17% in fumigated plots compared to nonfumigated plots in fields 8 and 9, respectively.

Table 2.1. Effect of soil fumigation[a] on severity of root rot of sweet corn over time and on ear number and yield at harvest.

	Root rot severity[b]			Ear	Yield/plot[c]
	4 wk	8 wk	harvest	number/plot[c]	(Kg)
Field 8					
Fumigated	1.0*	10.0*	10.0*	46*	15.3*
Nonfumigated	3.8	19.5	89.5	32	9.7
Field 9					
Fumigated	0.6*	11.3*	26.3*	40 ns	12.5*
Nonfumigated	1.2	29.2	81.7	36	10.7

[a] Plots were fumigated with methyl bromide (67%) and chloropicrin (33%) at 450 kg/ha.
[b] Scale of 0-100%. Average of 4 replicates, 10 plants per replicate at 4 wk, 3 plants per replicate at 8 wk and harvest.
[c] Average of two 3 m long rows per replicate.
*Significantly different compared to the nonfumigated control according to Fischer's protected least significant difference ($P\leq0.05$).

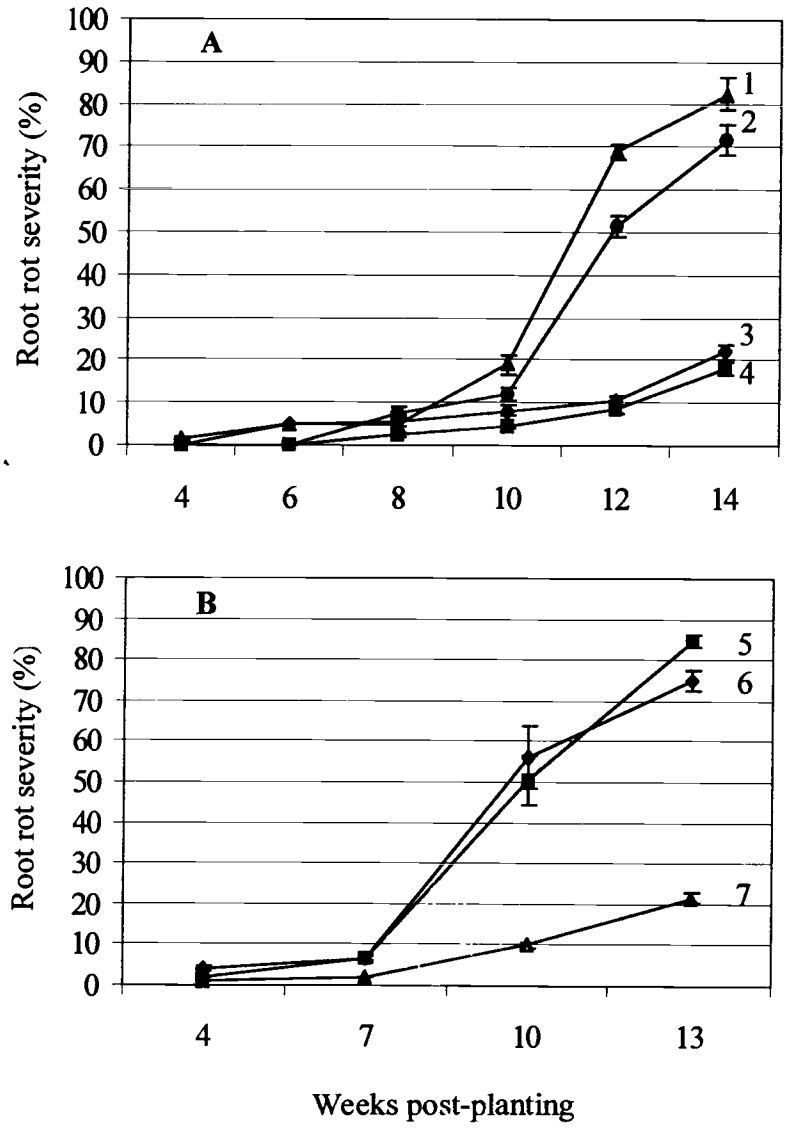

Fig. 2.1. Development of root rot of sweet corn in four fields in Oregon in A) 1997 and three fields in B) 1998. Each point represents an average of 10 plants. Four fields of cv Golden Jubilee were sampled in 1997 and two fields of cv Golden Jubilee (5 and 6) and one field of Supersweet Jubilee (7) were sampled in 1998.

Fungal isolations. The relative number of different species of *Fusarium* and *Pythium* isolated from symptomatic roots in 1997, '98 and '99 were similar. Over the three years, *Fusarium oxysporum* and *F. solani* (Mart.) Appel & Wollenw. emend. Snyd. & Hans. accounted for 70% and 22% of *Fusarium* isolated. *F. graminearum, F. proliferatum* (Matsushima) Nirenberg, *F. moniliforme* Sheldon, *F. semiticum* Berk & Rav., *F. subglutinans* (Wollenw. & Reinking) Nelson, Toussoun & Marasas comb. nov., *F. lateritum* Nees, and *F. sambucinum* Fuckel were isolated infrequently. *Pythium arrhenomanes* and *P. arrhenomanes*-like isolates accounted for 64% of the *Pythium* isolates. *P. sylvaticum* Campbell & Hendrix accounted for 13% of isolates and *P. ultimum* Trow, *P. torulosum* Coker & Patterson, *P. irregulare* Buisman, *P. inflatum* Matthews, and *P. oligandrum* Drechs. were isolated infrequently.

In the greenhouse study at emergence, *Trichoderma* spp., *F. oxysporum* and *Phoma terrestris* accounted for 29, 18 and 14%, respectively of isolates recovered. At 1 wk post-emergence, *Drechslera* sp. and *F. graminearum* each accounted for 25% of fungi isolated. *Phoma terrestris* and *F. oxysporum* each accounted for 17% of fungi and *Pythium* and *Trichoderma* were each isolated only once. At 2 wk post-emergence, *F. oxysporum* and *Pythium* spp. each accounted for 33 and 28% respectively, whereas *F. graminearum*, *F. solani* and *Trichoderma* spp. each accounted for 8%.

Koch's Postulates study. Regression analysis of the relationship between amount of inoculum of *P. arrhenomanes* and severity of rot of the radicle and

nodal roots, and plant dry weight was significant (P=0.01, 0.0002, P<0.0001, respectively). With an increase in amount of inoculum of *P. arrhenomanes*, disease severity increased and plant dry weight decreased (Fig. 2.2). Amount of inoculum had no effect on rot of the mesocotyl (P=0.36) and a t-test comparing the noninfested control to the combined infested treatments was not significant (P=0.12).

Regression analysis of amount of inoculum of *Phoma terrestris* or *Drechslera* sp. was significant for all response variables ($P \leq 0.03$). With an increase in amount of inoculum of *Phoma terrestris* or *Dreschlera* sp., severity of rot of the three components of the root system increased and amount of plant dry weight decreased (Figs. 2.3 and 2.4).

Amount of inoculum of *Fusaruim oxysporum* had no effect on severity of rot of any component of the root system and a t-test for the effect of inoculum alone was not significant (P>0.05).

With increasing amounts of inoculum of *F. graminearum*, the severity of rot of the mesocotyl increased to a maximum of 1.88 at the 10x rate. At the 100x rate, the trend was reversed and mesocotyl rot rating dropped to 1.14. Regression analysis of the relationship between amount of inoculum of *F. graminearum* and severity of rot of the mesocotyl and plant dry weight was significant, regardless of the inclusion or not of disease data at the 100x rate (P=0.01, 0.007, respectively). To fit regression lines, the mesocotyl rot rating at 100x was excluded (Fig 2.5). Amount of inoculum was not related to rot severity of the radicle or nodal roots

(P>0.05). A t-test for the effect of inoculum alone was significant for rot of the radicle (P=0.02) but not nodal roots (P=0.34) (Table 2.2).

P. arrhenomanes, Phoma terrestris, Drechslera sp., and *F. graminearum* were each recovered from root lesions of the two plants plated per ten replicate plants grown in soil infested with the respective pathogen, completing Koch's Postulates.

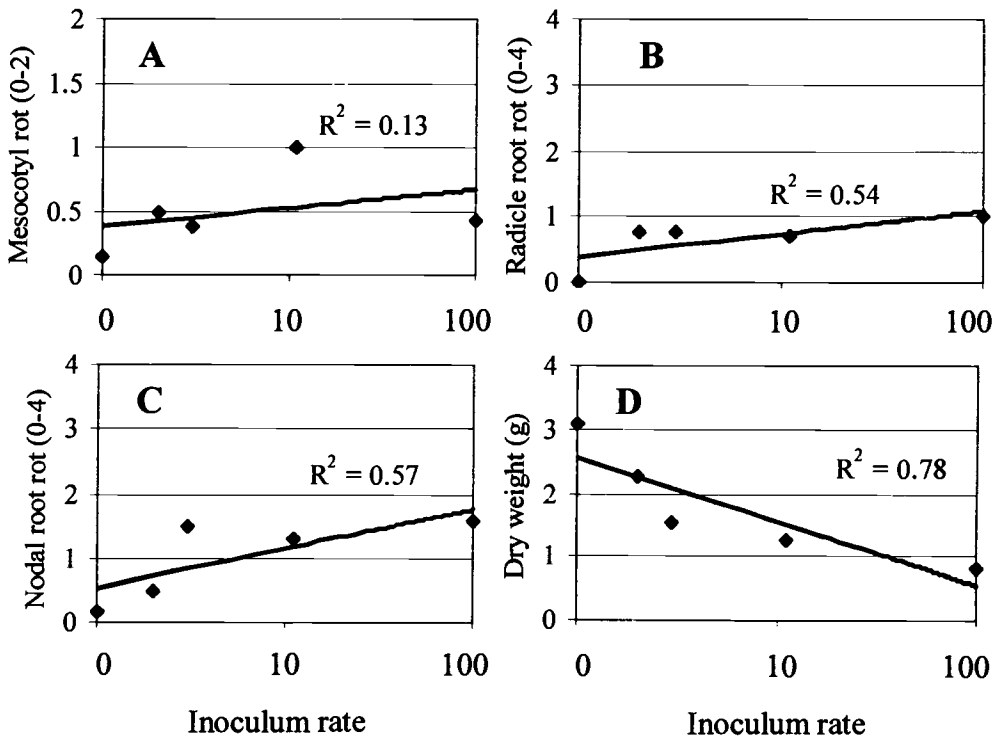

Fig. 2.2. Effect of amount of inoculum of *Pythium arrhenomanes* on rot of the A) mesocotyl, B) radicle, and C) nodal roots, and on D) dry weight of sweet corn. Soil was infested with *P. arrhenomanes* at 0, 1, 2, 10 or 100x. Data points represent the mean of 10 replicate plants. Mesocotyl rot was assessed on a scale of 0-2 where 0=healthy, 1=lesion present, and 2=100% necrotic, radicle rot was assessed on a scale of 0-4 where 0=healthy, 1=lesion present, 2=10-50%, 3=51-99%, and 4=100% necrotic, and nodal root rot was assessed on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, and 4=>50% necrotic.

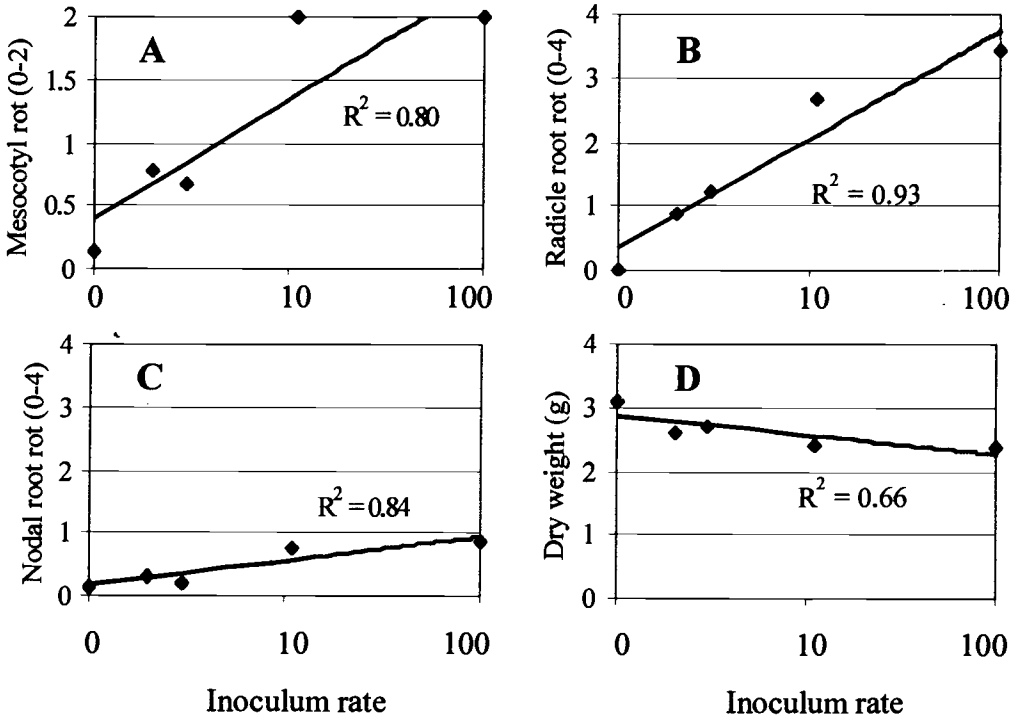

Fig. 2.3. Effect of amount of inoculum of *Phoma terrestris* on rot of the A) mesocotyl, B) radicle, and C) nodal roots, and on D) dry weight of sweet corn. Soil was infested with *P. terrestris* at 0, 1, 2, 10, or 100x. Data points represent the mean of 10 replicate plants. Mesocotyl rot was assessed on a scale of 0-2 where 0=healthy, 1=lesion present, and 2=100% necrotic, radicle rot was assessed on a scale of 0-4 where 0=healthy, 1=lesion present, 2=10-50%, 3=51-99%, and 4=100% necrotic, and nodal root rot was assessed on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, and 4=>50% necrotic.

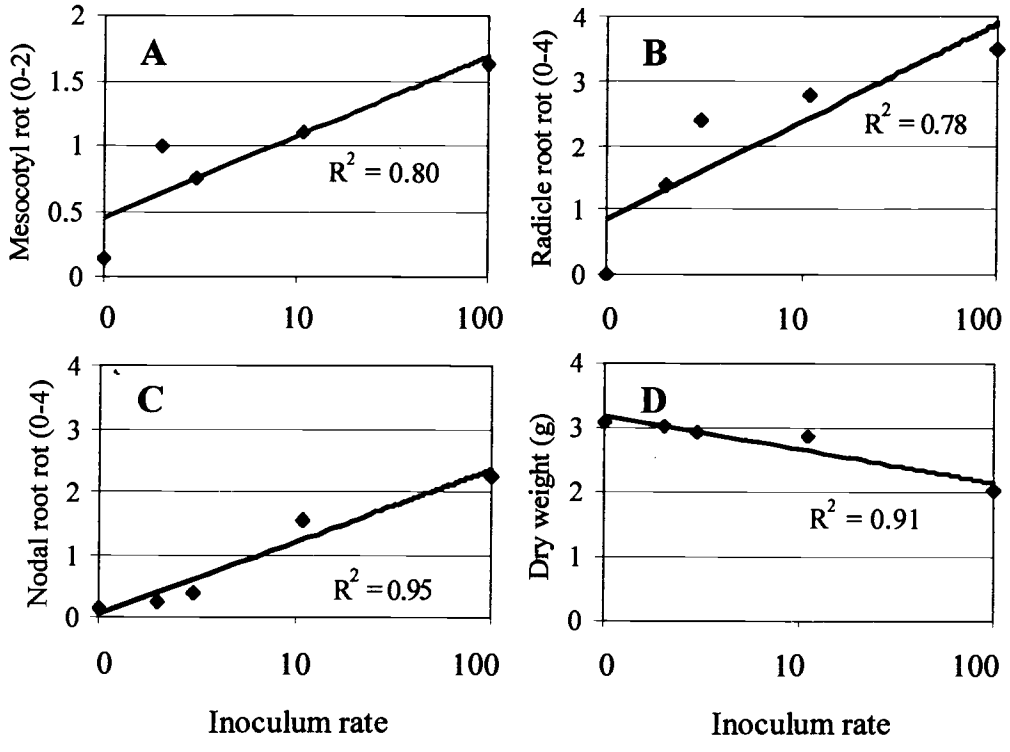

Fig. 2.4. Effect of amount of inoculum of *Drechslera* sp. on rot of the A) mesocotyl, B) radicle, and C) nodal roots, and on D) dry weight of sweet corn. Soil was infested with *Drechslera* sp. at 0, 1, 2, 10, or 100x. Data points represent the mean of 10 replicate plants. Mesocotyl rot was assessed on a scale of 0-2 where 0=healthy, 1=lesion present, and 2=100% necrotic, radicle rot was assessed on a scale of 0-4 where 0=healthy, 1=lesion present, 2=10-50%, 3=51-99%, and 4=100% necrotic, and nodal root rot was assessed on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, and 4=>50% necrotic.

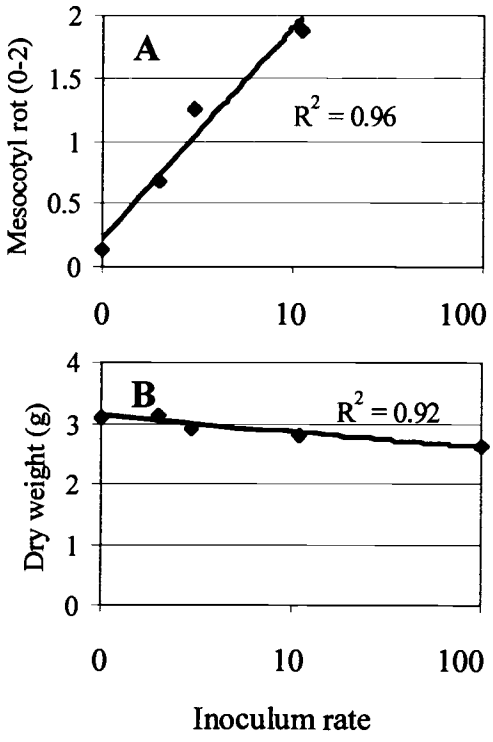

Fig. 2.5. Effect of amount of inoculum of *Fusarium graminearum* on A) rot of the mesocotyl, and B) dry weight of sweet corn. Soil was infested with *F. graminearum* at 0, 1, 2, 10, or 100x. Data points represent the mean of 10 replicate plants. Mesocotyl rot was assessed on a scale of 0-2 where 0=healthy, 1=lesion present, and 2=100% necrotic.

Table 2.2. Mean[a] severity of rot of the radicle and nodal roots of sweet corn seedlings grown in soil infested with *Fusarium graminearum*.

	Radicle[b]	Nodal roots[c]
Noninfested	0	0.14
Infested	0.3*	0.31

[a]Means of 10 replicate plants.
[b]Radicle root rot scale: 0=healthy, 1=lesion present, 2=11-50% necrotic, 3=51-99% necrotic, 4=100% necrotic.
[c]Nodal root rot scale: 0=healthy, 1=5-10% necrotic, 2=11-25% necrotic, 3=26-50% necrotic, 4= >50% necrotic.
*Significantly different than the noninfested control ($P \leq 0.05$).

DISCUSSION

Our disease dynamics study indicates that the disease affecting sweet corn plantings in the Willamette Valley is a root rot, not a stalk rot. The early appearance of root lesions, the relatively slow development of root rot symptoms, and the subsequent rapid increase in severity late in the season is similar to that reported for field corn. Rao et al (1978) and Whitney and Mortimore (1961) reported primary roots of field corn had rotted completely by approximately 4 wk post-planting. Mao et al (1998) reported that root lesions of field corn occurred by 7 wk post-planting and root rot developed slowly until 11 wk post-planting at which time symptoms developed more rapidly. Root growth dynamics may explain this disease pattern (Huisman, 1982). During the first 11 weeks root growth is active and the amount of tissue being evaluated increases as root rot increases. After silking, very little root development occurs (Ritchie et al, 1997) but disease severity continues to increase as lesions expand and coalesce. In our study, the rapid increase in severity of root rot started 7 wk after planting in 1998 compared to 10 wk in 1997. This is likely due to environmental differences between years and kind and density of pathogens present.

Soil fumigation with methyl bromide and chloropicrin decreased the severity of root rot of sweet corn, indicating that the primary cause of the disease is soilborne and biotic. In addition, fumigation increased yields by up to 50%, indicating that root rot can result in severe economic losses. Sumner et al (1982) reported increased plant density and a 42% increase in sweet corn yield with

methyl bromide fumigation at 488 kg/ha. A decrease in severity of root rot and increase in both silage and grain yield of field corn with soil fumigation was reported in the southern United States (Sumner et al, 1985; Sumner et al, 1990).

The fungi and oomycetes we found associated with root rot of sweet corn are common soil inhabitants and can be found in soils of many healthy and diseased crops. Early in our investigation we focused on *Fusarium* and *Pythium* as they are the most prevalent organisms reported to cause root rot of corn. *F. oxysporum* and *F. solani*, the *Fusarium* species we isolated most frequently, have been reported as the most prevalent sprcies of *Fusarium* colonizing roots of field corn (Warren and Kommedahl, 1973; Kommedahl et al, 1979). *P. arrhenomanes* was the most frequently isolated *Pythium* species in this study. This species is the most prevalent species on corn in Georgia (Sumner et al, 1990) and Ohio (Deep and Lipps, 1996) and is closely related to *P. graminicola*, reported earlier as the primary pathogen on corn in Ohio (Rao et al, 1978).

When screening trials failed to identify pathogens able to fully account for symptoms seen in the field, we turned our attention to other fungi infecting roots early in the season. From these isolations we found a number of organisms, all of which have been associated with corn roots (Compendium of Corn Diseases, 1999; Sumner et al, 1990; Whitney and Mortimore, 1961). It is clear that many organisms are associated with symptomatic sweet corn roots.

The results of pathogenicity tests conducted under artificial conditions can be difficult to interpret. The introduction of an organism in an otherwise sterile

environment may lead to disease symptoms whereas in nature the microbe may be non- or weakly- pathogenic. In addition, the amount of inoculum used is very important. While the type and amount of inoculum should mimic that found in nature, these numbers may not be known, or in some cases, biologically relevant. For example, inoculum of Pythiacious fungi may include oospores, mycelium, sporangia and/or zoospores and often the viability of these propagules is unknown. In addition, when working with unfamiliar organisms, the type of inoculum may be unknown and standard methods of inoculum enumeration may be unsuccessful. To address these issues, Koch's Postulates were tested in an inoculum dose-disease response study with fungi and Pythia that caused root lesions and/or reductions in plant dry weight in preliminary screening trials (Appendix A). Our results indicate that several organisms cause disease on sweet corn and symptoms differ among pathogens. Density dependent relationships were established for *Pythium arrhenomanes, Phoma terrestris*, *Drechslera* sp., and *Fusarium graminearum*, indicating that these organisms are primary pathogens. In contrast, no density dependent relationship was evident for *Fusarium oxysporum*, suggesting it is not a primary pathogen. Given its close association with symptomatic roots, it may be a weak or secondary pathogen.

While *Pythium* species, including *P. arrhenomanes*, have been reported to cause damping-off of corn (Compendium of Corn Diseases, 1999; Mao et al, 1997; Johann et al, 1928), this seedling disease was not observed in this study. Our data suggest that *P. arrhenomanes* attacks primarily the nodal, or secondary,

roots of sweet corn. This species also has been reported as causing root rot of field corn (Deep and Lipps, 1996) and other crops, including wheat, rice, sugarcane and grasses (Abad et al, 1994; Dissanayake et al, 1997; Hoy and Schneider, 1988; Singleton, 1981; Lee, 1994; Chun and Schneider, 1998). In addition, rot severity of the nodal roots was relatively mild (1.6 out of 4 points) even at the 100x inoculum rate whereas the reduction in plant dry weight compared to the noninfested control was 74%, indicating that this pathogen, while causing root rot, is primarily a root pruner. Similar effects by *P. arrhenomanes* on plant growth of field corn (Deep and Lipps, 1996; Sumner et al, 1990) and sugarcane (Hoy and Schneider, 1988) have been reported.

Phoma spp. cause root rots of a number of crops, including corn, alfalfa, pea and onion, and *P. terrestris* is considered the primary pathogen in a complex of fungi causing red root rot of field corn (Campbell et al, 1991; Mao et al, 1998; Sumner et al, 1990, Persson et al, 1997; Sumner et al, 1997; Rodriguez et al, 1990). Symptoms produced by this fungus include black or red lesions on roots. The discoloration and wilting of foliage seen late in the season is similar to the firing symptom we have observed with sweet corn.

Studies of *P. terrestris* on field corn suggest it is a late-season pathogen of senescing tissue and usually is not isolated from symptomatic tissue until mid-season (Mao et al, 1998; Rao et al, 1978; Sumner et al, 1990). In contrast, we found *P. terrestris* infecting the radicle at emergence and causing severe disease by the 6th leaf stage.

We found *Drechslera* sp. to be pathogenic to all parts of the seedling root system, suggesting it is an important pathogen of sweet corn. In addition, in screening trials it caused pre- and post-emergent damping-off (Appendix A). The genus *Drechslera* is one of three originally classified as *Helminthosporium*, the other two are *Bipolaris* and *Exersohilum* (telomorphs *Pyrenophora*, *Cochliobolus*, and *Setosphaeria*, respectively) (Alcorn, 1988). These genera were established to differentiate the graminicolous species of *Helminthosporium* from lignicolous ones, most of which are saprophytes. However, this segregation is not universally accepted, making studies of these fungi difficult to interpret. *Helminthosporium pedicellatum* has been described as the primary pathogen causing root rot of both field and sweet corn in California and it also infects sorghum and grasses (Shepard et al, 1967). Similar to our study, it was found to infect corn roots early in the season. This fungus also has been associated with root rot of field corn in Ohio (Rao et al, 1978). *H. pedicellatum* has since been classified as *Exserohilum pedicellatum*, though it also has been included with the genera *Bipolaris* and *Drechslera*. Our pathogen was identified based on the DNA sequence of the ITS and matches sequences studied by Zhang and Berbee (2001) in their phylogenetic analysis of *Pyrenophora/Drechslera* and so the relation of our fungus to those previously described on corn is unclear. *Helminthosporium* spp. cause root rots of other crops as well. The cause of common root rot of wheat and barley has been reported as *Drechslera sorokiniana* and *Bipolaris sorokiniana* as well as *Helminthosporium sativum* (Diehl, 1979; Hampton, 1979; Stack, 1980).

Drechslera spp. also are reported root rot pathogens of turfgrasses (Compendium of Turfgrass Diseases, 1992).

Based on our Koch's Postulates study, bulked isolates of *Fusarium oxysporum* did not cause root rot of sweet corn. While some isolates were mildly pathogenic in a preliminary screening trial (Appendix A), results were not reproducable. *Fusarium* species, particularly *F. oxysporum*, are widespread and have been associated with both diseased and symptomless corn roots (Kommedahl et al, 1979; Leslie et al, 1990; Warren and Kommedahl, 1973). In many root rots, *Fusarium* species are thought to be secondary pathogens. For example, when soils were infested with nonpathogenic isolates of *F. solani*, severity of root rot of pea caused by *Aphanomyces euteiches* was increased (Peters and Grau, 2002). Studies examining the interaction of *F. oxysporum* with *Pythium arrhenomanes*, *Phoma terrestris*, or *Drechslera* sp. on root rot of sweet corn, however, found no such relationship (Hoinacki and Powelson, unpublished; Ludy and Powelson, unpublished). *Fusarium* also have been considered opportunisitc pathogens, able to invade when plants are stressed or wounded (Leslie et al, 1990; Mao et al, 1998; Warren and Kommedahl, 1973). Because we did not test our isolates under conditions stressful to the plant or in conjunction with wounding, it is unclear whether *F. oxysporum* may play such a role in root rot of sweet corn.

Fusarium graminearum caused rot primarily on the mesocotyl, indicating it is a pathogen of the emerging seedling. In a preliminary screening trial

(Appendix A) it also caused pre- and post-emergence damping-off. *F. graminearum* has been reported to cause damping-off of field corn (Mao et al, 1997) and seedling blight of wheat (Jones, 1999). Fungi causing damping-off are generally controlled in commercial sweet corn fields with seed treatments and it is likely that *F. graminearum* contributes little to the development of rot of the radicle or nodal roots.

Although small pot pathogenicity trials with seedlings in the greenhouse allow the rapid evaluation of large numbers of fungi, disease dynamics throughout the growing season are also important. In a large pot study, a complex of *P. arrhenomanes, P. terrestris*, *Drechslera* sp., *F. graminearum* and *F. oxysoprum* caused extensive root rot, secondary symptoms of leaf chlorosis and necrosis, and a significant reduction in yield (Appendix C).

Our research indicates *P. arrhenomanes, P. terrestris* , and *Drechslera* sp. are the primary pathogens causing root rot of sweet corn in the Willamette Valley. Other fungi and soilborne organisms may be involved in the disease syndrome. In addition, it is likely abiotic factors contribute to severity of disease. Because of the complexity of the organisms involved and a wide variety of disease symptoms, including damping-off, root rot, root pruning, foliar chlorosis and necrosis, and reductions in ear yield and quality, we are now calling it Sweet Corn Decline Syndrome.

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Chapter 3

A Rating System for Root Rot of Sweet Corn

E. V. Hoinacki and M. L. Powelson

ABSTRACT

A disease rating system we developed for root rot of sweet corn was tested in an inoculum dose-disease response study. Scales for severity of rot of the mesocotyl, radicle, and nodal roots were based on 1) development of symptoms as described in prior studies, and 2) a review of root rot rating scales from the literature. In our rating system, the mesocotyl is rated on a scale of 0-2 where 0=healthy, 1=lesion present, 2=100% necrotic; the radicle on a scale of 0-4 where 0=healthy, 1=lesion present, 2= 10-50%, 3=51-99%, 4=100% necrotic; and nodal roots on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, 4= >50% necrotic. To test the rating system, naturally infested soil from two fields with a history of root rot was diluted with pasteurized soil to 0, 1, 10, 33, 50, or 100% and sweet corn seeds were planted. Seedlings were harvested at the 6th leaf stage and the three components of the root system were rated for severity of rot. Shoots and roots were dried at 60 C and weighed. Regression analysis of percent naturally infested soil (amount of inoculum) on rot severity of the radicle and nodal roots and on dry weight was significant for both soils and significant for one soil for severity of rot of the mesocotyl. The root rot rating system should be

a useful tool to evaluate the effects of biotic and abiotic factors on severity of disease.

INTRODUCTION

For the past two decades, sweet corn (*Zea mays* L.) growers in the Willamette Valley of Oregon have become increasingly concerned with the emergence of a root rot in their plantings. This root rot is caused by a complex of soilborne organisms including *Pythium arrhenomanes*, *Phoma terrestris*, *Drechslera* sp., and *Fusarium graminearum*. Yield losses in commercial plantings to root rot have been estimated up to 50% (Chapter 2; Hoinacki et al, 2001; Hoinacki and Powelson, 2002).

To quantify effects of biotic and abiotic factors on the severity of root rot, we needed a method to quantify small differences in disease. We developed a root rot rating system based on 1) symptom development on different components of the corn root system, and 2) a review of root rot rating scales from the literature.

The development of root rot of sweet corn has been described previously (Chapter 2) and will be reviewed here briefly. Root rot can occur on three basic components of the sweet corn root system: the mesocotyl, radicle, and nodal roots (Fig. 3.1). The radicle, or primary root, begins to develop before the coleoptile emerges and functions as the primary site of water and nutrient uptake until the development of the nodal roots at approximately the 6th leaf stage

(Ritchie et al, 1997). In the field, lesions appear on the radicle as early as 3 wk post-planting. In severely infested fields the radicle may become completely rotted by 6-10 wk post-planting when lesions begin to appear on the nodal roots. The mesocotyl, or subcrown internode, may or may not become diseased.

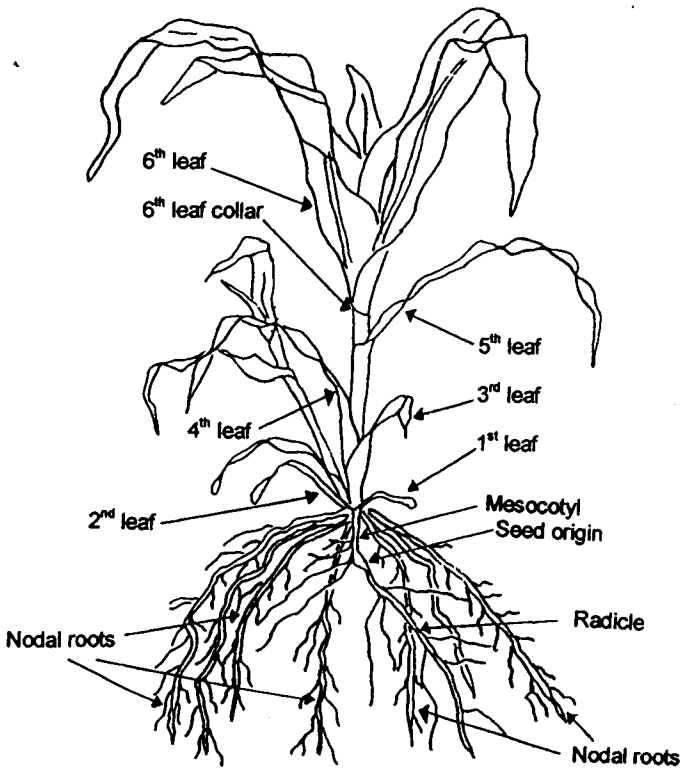

Fig. 3.1. Components of a sweet corn seedling root system.

Root rot rating scales generally fall into two categories and scales for corn can be found in both. The first divides the severity of rot (usually the percentage of necrotic or discolored roots) into more or less equal categories, i.e. a 1 to 5

scale where 1=no disease, 2=3-30%, 3=31-60%, 4=61-90% necrosis, 5=dead plant (Mao et al, 1998; Tan and Tu, 1995). More common, however, is the second category, pioneered by Horsfall and Barratt (1945) which divides the severity of disease unequally and thus weights ratings at the lower and, sometimes, upper ends of the severity scale. For example 1=<2%, 2=2-10%, 3=11-50%, 4>50% necrosis, and 5=dead plants (Dissanayake and Hoy, 1999; Sumner et al, 1990). This method, while incorporating for the difficulty of the eye's ability to differentiate between healthy and diseased tissue near the 50% level, recognizes the significance of small amounts of disease. Other root rot rating scales identify a specific number of lesions (Schreuder et al, 1995), size and/or coalescion of lesions (Rao et al, 1978), or length of root rotted (Rodriguez et al, 1990; Persson et al, 1997). Many other root rot rating scales also incorporate different plant parts but do not distinguish among them. For evaluating disease on sweet corn, cotton, bean and pea, researchers evaluated both the roots and hypocotyl or epicotyl (Kobringer and Hagedorn, 1984; Melero-Vara and Jimenez-Diaz, 1990; Peters and Grau, 2002; Sumner et al, 1982).

For root rot of sweet corn, we chose to assign a rating scale for each component of the root system using unequal categories for disease severity. In our rating system, the mescotyl is rated on a scale of 0-2 where 0=healthy, 1=discrete lesion present, 2=100% necrotic; the radicle on a scale of 0-4 where 0=healthy, 1=at least one lesion present but less than 10% of the root necrotic, 2= 10-50%,

3=51-99%, 4=100% necrotic; and nodal roots on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, 4= >50% necrotic.

The objective of this study was to test the root rot rating system. Our question was: would the root rot rating system reflect changes in inoculum density? To look at amount of disease on each component we conducted an inoculum dose-disease response study using natural inoculum.

METHODS

Soil was collected from two sweet corn fields located near Stayton in Marion County, OR with a history of root rot. Both fields had been frequently cropped to corn in the prior decade and severe root rot had been previously documented (Chapter 2). Both soils were sieved through a 1 cm screen and a portion was pasteurized in an autoclave at 90 C for 1 hr on each of two consecutive days. Naturally infested soil was diluted with the pasteurized soil to 0, 1, 10, 33, 50, or 100% v/v, mixed in a V-shaped shell mixer for 20 min and placed in 550 ml cone tubes (6.5 cm x 25 cm). Treatments were arranged on a greenhouse bench in a complete randomized design and replicated 10 times. Seeds of the susceptible sweet corn cv Golden Jubilee (J. Myers, personal communication), were surface disinfested in 10% bleach for 5 min, rinsed three times in distilled water and planted 2.5 cm deep. After emergence, soils were kept moist and fertilized once weekly with a water soluble plant food (N-P-K, 20-20-20) at the label rate. At the 6th leaf stage, plants were harvested. Shoots were

cut at the soil surface and bagged. Tubes were emptied onto a 0.5 cm screen on which the rootball was collected. Rootballs were carefully washed under running tap water, and the mesocotyl, radicle, and nodal roots were rated as described previously. Shoots and roots were dried at 60 C and weighed.

To evaluate the effect of percent naturally infested soil (amount of inoculum) on severity of rot of the mesocotyl, radicle, and nodal roots, and on plant dry weight, regression analysis was conducted using SAS Version 8.1 (SAS Institute, Cary, NC). To fit regression lines to the data, disease ratings, expressed as proportions (actual root rot rating/maximum allowable rating), were transformed with Gregory's multiple infection transformation, $-\ln(1-y)$ (Gregory, 1948). When disease reached a maximum amount (after the dose-response curve flattens), root rot ratings were excluded from the transformation.

RESULTS

Effect of percent naturally infested soil (amount of inoculum) on severity of rot of the radicle and nodal roots was similar in the two field soils (Fig. 3.2A and B). As amount of inoculum increased to 33%, disease increased. At higher rates of naturally infested soil, amount of disease leveled off. Regression analysis on effect of percent naturally infested soil (amount of inoculum) on severity of rot of the radicle, and nodal roots, and on plant dry weight was significant for both soils ($P<0.0009$) and significant ($P=0.003$) for one soil for severity of rot of the mesocotyl (Table 3). Regression lines fit to transformed root rot ratings while

disease is increasing are shown in Fig. 3.3. The relationship between amount of inoculum and plant dry weight was linear for both soils (Fig. 3.4).

Table 3. Analysis of variance for the regression of percent naturally infested soil (amount of inoculum) on root rot and plant dry weight of sweet corn seedlings grown in two field soils.

	F-value	P-value
Field 1		
Mesocotyl rot[a]	1.17	0.28
Radicle root rot[b]	23.85	<0.0001
Nodal root rot[c]	20.45	<0.0001
Plant dry weight	12.43	0.0009
Field 2		
Mesocotyl rot[a]	9.61	0.003
Radicle root rot[b]	30.67	<0.0001
Nodal root rot[c]	38.36	<0.0001
Plant dry weight	12.92	0.0008

[a] Scale: 0-2 where 0=healthy, 1=lesion, 2=100% necrotic.
[b] Scale: 0-4 where 0=healthy, 1=lesion present, 2=11-50% necrotic, 3=51-99% necrotic, 4=100% necrotic.
[c] Scale: 0-4 where 0=healthy, 1=5-10% necrotic, 2=11-25% necrotic, 3=26-50% necrotic, 4=>50% necrotic.

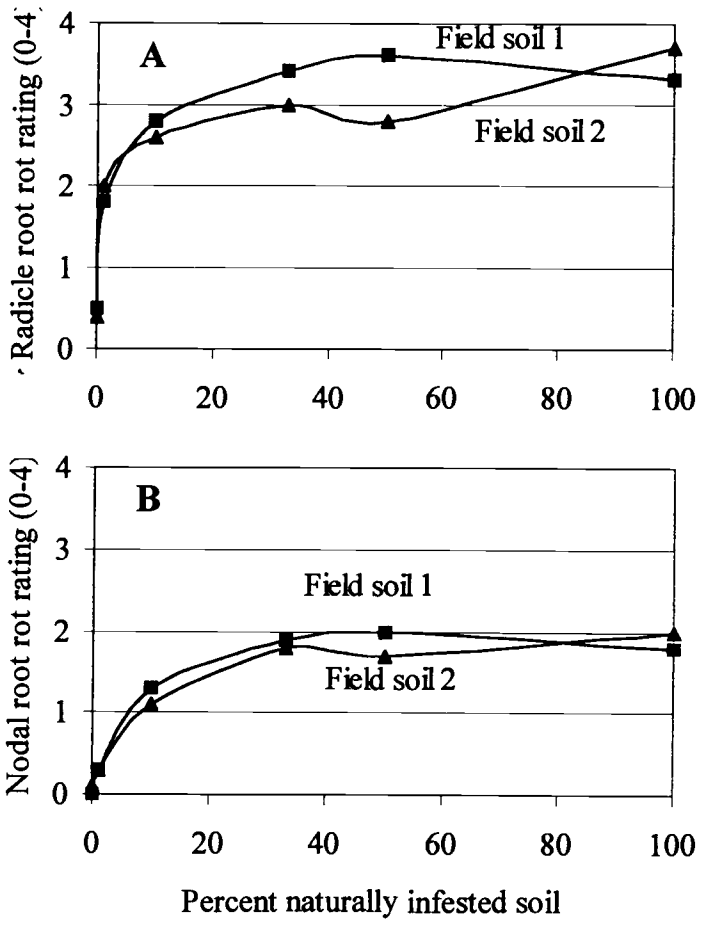

Fig. 3.2. Effect of percent naturally infested soil (amount of inoculum) on rot of the A) radicle, and B) nodal roots of sweet corn seedlings grown in two field soils. Radicle rot was assesed on a scale of 0-4 where 0=healthy, 1=lesion present, 2=10-50%, 3=51-99%, and 4=100% necrotic, and nodal root rot was assessed on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, and 4=>50% necrotic.

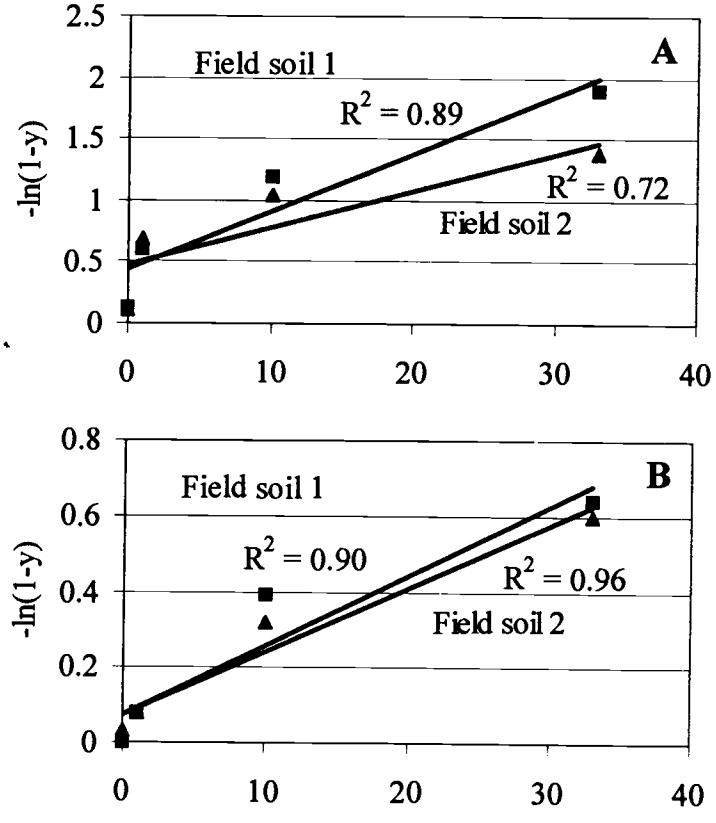

Fig. 3.3. Relationship between percent naturally infested soil and severity of rot of the A) radicle, and B) nodal roots of sweet corn seedlings grown in two field soils. Root rot ratings were transformed by Gregory's multiple infection transformation after conversion to a proportion (actual root rot rating/maximum allowable rating).

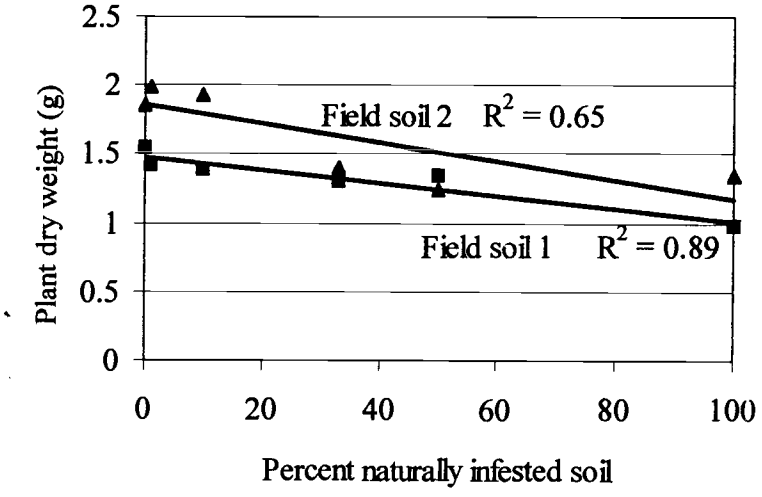

Fig. 3.4. Relationship betweeen percent naturally infested soil (amount of inoculum) and plant dry weight of sweet corn seedlings grown in two field soils.

DISCUSSION

To test the root rot rating scale, a greenhouse bioassay was designed to evaluate the inoculum dose-disease response relationship of naturally occurring inoculum. Of interest was how well root rot rating scale data would fit a dose-response curve as discussed by Van der Plank (1975). While such curves typically plot the proportion of diseased tissue instead of ratings based on unequal amounts of diseased tissue, a disease rating system that reflects a natural system and is biologically relevant should plot in a similar fashion. We found this to be the case for our root rot rating system. Our data reflect what Van der Plank called

the commonest relation in his discussion on pathogen dose-disease response dynamics. In this relation the amount of disease produced per unit of inoculum decreases as the amount of inoculum increases because susceptible host tissue becomes limited and propagules compete for available infection sites.

The fit of regression lines to the transformed root rot ratings for the radicle and nodal roots indicates that the disease rating scales adequately reflect the relation between amount of inoculum and severity of rot when disease is increasing. Lower disease ratings at low amounts of inoculum would probably improve the fit of lines. The disease rating scale could be modified to assign less value to less severe disease. However, there could be an ecologically significant reason for high disease ratings at low inoculum levels. The experiment only takes into consideration the amount of inoculum introduced to the system, or primary inoculum. It is likely that secondary inoculum (i.e. zoospores of *Pythium*) is produced. R-squared values are also likely influenced by the nature of the inoculum. Because inoculum was quantified by volume of naturally infested soil rather than actual colony forming units, some variability in inoculum type (pathogen species present) and amount is likely.

Pathogens of the mesocotyl have been identified (Chapter 2), however severity of rot of the mesocotyl was related to amount of inoculum in soil 2. It is likely that populations of the pathogen(s) that causes rot of the mesocotyl were higher in this soil. Although severity of rot of the mesocotyl in soil 2 was related to amount of inoculum, the fit of a regression line to data transformed with

Gregory's multiple infection transformation was not strong (R^2=0.52, data not shown). Because the disease rating scale on the mesocotyl (0-2) is limited in scope, it may not accurately reflect small changes in inoculum density.

The assigning of disease severity scales to each component of the root system allows us to focus on early season root rot development. The radicle is the primary indicator of rot early in the season. In severely diseased corn fields, the radicle may be completely rotted a few weeks post-planting whereas in healthy plantings the radicle is intact and healthy at harvest (Chapter 2). Focusing on early season disease development, in turn, allows the use of the rating system in both greenhouse and field environments. Moreover, sampling of mature corn plants is laborious and prone to error. Early season sampling is less prone to leaving many roots in the field, especially weakened or necrotic ones.

Root rot ratings when disease is severe may be misleading. Loss of root biomass to rot may lead to lower disease ratings as proportionately more healthy tissue remains. In addition, root pruning pathogens such as *Pythium* may cause little necrosis but significantly reduce root health through the reduction in root biomass. Some root disease scales incorporate the activity of root pruning pathogens as subjective assessments of the reduction in roots as well as root necrosis (Dissanayake and Hoy, 1999). Many studies on Pythium root rots incorporate plant dry weight as a measure of pathogenicity and/or aggressiveness (Deep and Lipps, 1996; Dissanayake et al, 1997; Yanar et al, 1997). A biomass measure, therefore, was included as part of our disease rating system.

The root rot rating system should prove to be an appropriate and useful tool to evaluate the effects of biotic and abiotic factors on severity of disease. Recently it has been used to characterize inoculum dose/disease response relationships for pathogens involved in the root rot complex (Chapter 2). It also has been used to identify effects of cultivar (Appendix C) and herbicides (Chapter 4) on severity of root rot. Darby (2003) used the root rot rating system to evaluate the effect of cover crops and soil amendments on root rot of sweet corn. In addition, the root rot rating system was used to examine the relationship between severity of rot of the radicle at the 6th leaf stage and yield at harvest of field grown plants (Stone et al, unpublished). During the 2002 growing season, sweet corn plants, cv Golden Jubilee, from 24 plantings were sampled at the 6th leaf stage and at harvest, and yield data were obtained from growers. Regression analysis for the effect of severity of rot of the radicle on yield was significant (P=0.004) (A. Stone, personal communication). This suggests that disease severity of the radicle is an indicator of disease potential of a field. Currently, trials are underway to evaluate the rating system as part of a screening bioassay to estimate the risk for disease in field soils. Such a service could be provided for sweet corn growers to aid in deciding whether to plant a particular field to sweet corn or which cultivar to plant.

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Chapter 4

Effect of Herbicides on Root Rot of Sweet Corn is Pathogen Species Dependent

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ABSTRACT

The effect of four herbicides, two of which contain safeners, and a safener alone on root rot of sweet corn grown in soil artifically infested with *Pythium arrhenomanes*, *Phoma terrestris*, *Drechslera* sp., or *Fusarium graminearum* was evaluated in greenhouse experiments. In soil infested with *P. arrhenomanes*, dry weight of sweet corn seedlings was reduced when treated with the herbicide EPTC + R-29148. EPTC + R-29148 also increased severity of rot of the nodal roots caused by *P. terrestris*. In contrast, in soil infested with *Drechslera* sp., metolachlor increased severity of rot of the radicle and nodal roots and decreased plant dry weight. Metolachlor also reduced dry weight of plants grown in soil infested with *F. graminearum*. Addition of the safener benoxacor to metolachlor (metolachlor + benoxacor) and benoxacor alone generally had no effect on plant biomass or root rot of sweet corn seedlings grown in soil infested with any of the pathogens, suggesting benoxacor protects corn seedlings treated with metolachlor from increased root rot caused by specific soilborne pathogens as well as phytotoxic effects of the herbicide. Atrazine had no effect on plant biomass or root rot caused by any of the pathogens. Our results indicate that the effect of soil

applied pre-emergent herbicides on root rot of sweet corn depends on the pathogen species present.

INTRODUCTION

Root rot of sweet corn (*Zea mays* L.) can cause significant losses for growers in western Oregon. It is one of three diseases involved in sweet corn decline syndrome, which also includes seedling rot and crown rot (Chapter 2; Hoinacki and Powelson, 2002a). Primary symptoms of root rot are necrotic lesions on the radicle, or primary root, as early as 3 wk post-emergence and on nodal roots 6-10 wk post-emergence. Lesions expand until entire roots are rotted. In severely affected fields, the entire rootball may be rotted by harvest, decreasing both ear quality and yield. Root rot is caused by a complex of soilborne pathogens including *Pythium arrhenomanes* Drechs., *Phoma terrestris* E.M. Hans., *Drechslera* sp., and *Fusarium graminearum* Schwabe (Chapter 2). *P. arrhenomanes* causes symptoms of root pruning and root tip necrosis. *Phoma terrestris* and *Drechslera* sp. cause extensive necrosis of the entire root system, including the mesocotyl and radicle and nodal roots. *F. graminearum* causes rot of the mesocotyl and pre- and post-emergent damping-off and limited root necrosis.

Root rots have been difficult to control historically, especially those caused by pathogen complexes. Soil fumigation effectively reduces root rot and increases yield of corn and other crops (Chapter 2; Sumner et al, 1985; Yuen et al,

1991), but is not an economically or environmentally acceptable option for many growers. Seed treatments can protect emerging seedlings from attack by soilborne organisms, but their efficacy is short lived and they offer no protection from pathogens attacking older roots. Many root rot management strategies involve the manipulation of cultural practices, such as form of nitrogen fertilizer or type of tillage, to create conditions less favorable for infection and disease development.

Herbicides affect the severity of root diseases of many crops (Altman and Campbell, 1977; Altman and Rovira, 1989; Dissnanyake et al, 1998; Katan and Eshel, 1973; Sanogo et al, 2000), including root rot of field corn (Percich and Lockwood, 1975; Sumner and Dowler, 1983). Mechanisms by which herbicides are thought to increase disease severity include the alteration of structural and biochemical defenses of the plant through effects on plant growth and physiology, and the increase in root exudates due to disruptions in the membrane integrity of plant cells. In addition, herbicides can stimulate pathogen growth and reproduction and inhibit populations of competitive microflora (Altman, 1993; Hess, 1993). Decreases in severity of root disease also have been reported and are thought to occur through increased host defenses and deleterious effects on pathogen growth and reproduction (Altman and Campbell, 1977; Katan and Eshel, 1973).

The use of herbicide safeners or antidotes also may influence disease. The control of weeds in botanically related crops is an ongoing challenge for growers.

Safeners were developed to protect the crop plant from herbicides that target related weeds and are widely used in corn, rice, and sorghum (Hatzios, 1989). For weed control in corn, safeners are usually formulated as a prepackaged tank mixture with the herbicide. While the effect of safeners on plant diseases is largely unstudied, increased severity of both foliar and root diseases has been reported (Craig et al, 1987; Szerszen et al, 1988; Szerszen, 1993). Mechanisms by which safeners affect disease are unclear but their effect on plant growth and their fungicidal activity have been documented (Szerszen, 1993).

Preliminary trials examining the effect of herbicides on root rot of sweet corn in naturally infested soil suggest some herbicides increase disease severity in some soils (Hoinacki et al, 2001; Hoinacki and Powelson, 2002b). This differential effect among soils may be due to differences in the biological composition of these soils. It is likely that the pathogens causing root rot vary in kind and number among field soils. If herbicides that interact with specific pathogens within the root rot complex can be identified, recommendations made to growers concerning herbicide use could be made.

The objective of this study was to determine the effect of four herbicides, two of which contain safeners, and a safener alone on severity of root rot of sweet corn caused by *Pythium arrhenomanes*, *Phoma terrestris*, *Drechslera* sp., and *Fusarium graminearum*. All herbicides are registered for pre-emergent use on sweet corn and are commonly used in western Oregon for weed control. Prior to

this study we determined a 'no-effect' level of each herbicide in a greenhouse trial under similar conditions.

METHODS

Thirteen isolates of *Pythium arrhenomanes* (97102-2d, 97101-2b, 97102-1c, 9888-9, 9893-5, 9878-3, 9887-10, 9945-1a, 9942-11, 9966-5, 9947-7, 9947-17), three isolates of *Phoma terrestris* (0015-9b, 0015-6a, 0015-6c), two isolates of *Fusarium graminearum* (0016-3a, 0016-8a), and two isolates of *Drechslera* sp. (0016-7, BPP-2) all isolated from symptomatic sweet corn roots in 1997-2000, were grown on water agar (WA; Difco Laboratories, Detroit, MI). Two 2-mm agar plugs taken from the colony edge of each actively growing isolate were added to 250 ml Erlenmeyer flasks containing a sterile mix of sand (97 g), cornmeal (3 g), and water (28 ml). Flasks were incubated at room temperature under fluorescent lights for 3-4 wk. Inoculum was removed, air dried and stored at 8 C until use.

A Chehalis sandy loam was collected from the Oregon State University Botany and Plant Pathology Field Lab in Corvallis and pasteurized at 90 C for 1 hr on each of two consecutive days. Inoculum of individual isolates of each pathogen was bulked and added to the soil at four rates (0, 1, 10, or 100x)/500 ml soil as follows: *P. arrhenomanes* at 0, 0.005, 0.05, or 0.5 g inoculum; *Phoma terrestris* and *F. graminearum* at 0, 0.02, 0.2, or 2.0 g inoculum; and *Drechslera* sp. at 0, 0.01, 0.1, or 1.0 g inoculum. Amounts of inoculum for each pathogen

were determined previously in screening trials (Appendix A). Inoculum and soil for each treatment were mixed in a V-shaped shell mixer for 20 min and placed in 550 ml soil tubes (6.5cm x 25cm) on a greenhouse bench. Noninfested soil served as the control. Sweet corn seeds, cv Golden Jubilee, were surface disinfested in 10% bleach for 5 min, rinsed three times in distilled water and planted approximately 2.5 cm deep. The herbicides atrazine, metolachlor, metolachlor + benoxacor, EPTC + R-29148 and the safener benoxacor alone (all chemicals from Syngenta Crop Protection, Greensboro, SC) were applied in water at the following label rates: atrazine at 1.1 kg a.i./ha, metolachlor and metolachlor + benoxacor at 1.5 kg a.i./ha, EPTC + R-29148 at 3.4 kg a.i./ha, and benoxacor at 0.05 kg a.i./ha. A water only treatment served as the control. Treatments were arranged in a randomized design and replicated 10 times. Soils were kept moist and, after emergence, plants were fertilized once weekly with a water soluble plant food (N-P-K, 20-20-20) at the label rate. After approximately 6 wks, at the 6^{th} leaf stage, plants were harvested. Shoots were cut at the soil surface and bagged. Roots were collected on a 0.5 cm screen, washed under running tap water, rated for root rot, and then bagged. Severity of root rot was rated on each of the three components of the root system: mesocotyl: 0 = healthy, 1 = discrete lesion present, 2 = 100% necrotic; radicle root: 0 = healthy, 1 = at least one lesion present but less than 10% of the root necrotic, 2 = 10-50%, 3 = 51-99%, 4 = 100% necrotic; and nodal roots: 0 = healthy, 1 = 5-10%, 2 = 11-25%, 3 = 26-50%, 4 = >50% necrotic (Chapter 3). Shoots and roots were oven dried at

60 C and weighed. Each pathogen was run as a separate experiment and each experiment was repeated once.

Analysis of variance was conducted using the general linear model procedure of SAS Version 8.1 to test for single and interacting effects of inoculum rate and herbicide on severity of rot of the mesocotyl, radicle, and nodal roots, and on plant dry weight. When herbicide by inoculum rate interactions were significant, herbicide main effects were analyzed within inoculum rate levels. Treatment means were generated by the method of least squares and pairwise comparisons were made at the 5% probability level. Regression analysis was used to evaluate the inoculum dose-disease response relationship for each pathogen across herbicide levels when herbicide by inoculum rate interactions were not significant and within the herbicide treatments when they were significant (SAS Institute, Cary, NC).

RESULTS

Pythium arrhenomanes. Inoculum rate by herbicide interaction was significant for plant dry weight in both experiments (Table 4.1). At the 100x rate, dry weight was reduced 24 and 30% with EPTC + R-29148 in experiments 1 and 2, respectively. In experiment 2, dry weight was reduced 21% with metolachlor compared to the no herbicide control. Atrazine, metolachlor + benoxacor, and benoxacor alone had no effect on dry weight (Table 4.2).

Inoculum rate by herbicide interaction was significant for severity of rot on the nodal roots in the first but not the second experiment (P=0.03). At the 100x rate of inoculum disease was less severe on plants treated with EPTC + R-29148 than the no herbicide control (data not shown). Herbicides had no effect on severity of rot of the mesocotyl or radicle in either experiment (Table 4.1).

Regression analysis of amount of inoculum of *P. arrhenomanes* on severity of rot of the radicle and nodal roots and on dry weight was significant (Table 4.3). With an increase in amount of inoculum, severity of root rot increased and dry weight decreased. Amount of inoculum had no effect on severity of rot of the mesocotyl.

Table 4.1. Analysis of variance for severity of rot of the mesocotyl, radicle, and nodal roots and dry weight of sweet corn seedlings grown in soil infested with *Pythium arrhenomanes*.

Sources	df	Mesocotyl rot	Radicle root rot	Nodal root rot	Dry weight
Exp. 1					
Inoculum rate[a]	3	*[b]	*	*	*
Herbicide[c]	5	ns	ns	ns	*
Inoculum rate x herbicide	15	ns	ns	*	*
Exp. 2					
Inoculum rate[a]	3	ns	*	*	*
Herbicide[c]	5	ns	ns	ns	*
Inoculum rate x herbicide	15	ns	ns	ns	*

[a] Inoculum rates were 0, 1, 10, 100x.
[b] * = significant at $P \leq 0.05$, ns = not significant.
[c] Herbicide treatments included atrazine, metolachlor, metolachlor + benoxacor, EPTC + R-29148, the safener benoxacor and a water control.

Table 4.2. Effect of preemergent herbicides on dry weight of sweet corn seedlings grown in soil infested with *Pythium arrhenomanes* at 100x rate of inoculum.

	Dry weight (g)	
	Exp. 1	Exp. 2
No herbicide control	1.74 ab[z]	1.15 a
Atrazine	1.98 a	1.10 ab
Metolachlor	1.36 bc	0.91 bc
Metolachlor + benoxacor	1.74 ab	1.00 abc
EPTC + R-29148	1.22 c	0.87 c
Benoxacor	1.54 ac	1.10 ab

[z] Means with the same letter within columns are not significantly different according to pairwise comparisons at the 5% probability level.

Table 4.3. Analysis of variance for the regression of amount of inoculum on root rot and dry weight of sweet corn seedlings grown in soil infested with *Pythium arrhenomanes*.

	F-value	*P*-value
Exp. 1[a]		
Mesocotyl rot	0.43	0.51
Radicle root rot	245.27	<0.0001
Nodal root rot	21.55-70.74	<0.0001
Dry weight	12.16-39.34	<0.002
Exp. 2[b]		
Mesocotyl rot	0.07	0.79
Radicle root rot	97.79	<0.0001
Nodal root rot	114.57	<0.0001
Dry weight	20.81-49.99	<0.0001

[a] Inoculum rate by herbicide interaction was significant for severity of rot of nodal roots and for dry weight and regression analysis was conducted within herbicide levels. Inoculum rate by herbicide interaction was not significant for severity of rot of the mesocotyl or radicle and regression anaylsis was conducted across herbicide levels.
[b] Inoculum rate by herbicide interaction was significant for dry weight and regression analysis was conducted within herbicide levels. Inoculum rate by herbicide interaction was not significant for severity of root rot and regression anaylsis was conducted across herbicide levels.

Phoma terrestris. Inoculum rate by herbicide interaction was significant in experiment 1 for severity of rot on nodal roots and differed significantly among herbicide treatments in both experiments (Table 4.4). Root rot was more than twice as severe on plants treated with EPTC + R-29148 at the 100x inoculum rate in experiment 1 and 8.5 times more severe across inoculum levels in experiment 2 compared to the no herbicide control (Table 4.5). Metolachlor, metolachlor + benoxacor, and benoxacor had no effect on severity of root rot. The inoculum

rate by herbicide effect was not significant for plant dry weight in either experiment and the effect of herbicide alone on dry weight was significant in experiment 1 only (Table 4.4). In experiment 1, dry weight was significantly reduced for plants treated with metolachlor and atrazine compared to the no herbicide control, metolachlor + benoxacor, and benoxacor ($P \leq 0.05$, data not shown).

Regression of amount of inoculum on severity of rot of the mesocotyl and radicle and on plant dry weight was significant in both experiments and on severity of rot of the nodal roots in the first experiment. (Table 4.6). With an increase in amount of inoculum, severity of root rot increased and dry weight decreased.

Table 4.4. Analysis of variance for severity of rot of the mesocotyl, radicle and nodal roots and on dry weight of sweet corn seedlings grown in soil infested with *Phoma terrestris*.

Sources	df	Mesocotyl rot	Radicle root rot	Nodal root rot	Dry weight
Exp. 1					
Inoculum rate[a]	3	*[b]	*	*	*
Herbicide[c]	5	ns	ns	*	*
Inoculum rate x herbicide	15	ns	ns	*	ns
Exp. 2					
Inoculum rate[a]	3	*	*	ns	*
Herbicide[c]	5	ns	ns	*	ns
Inoculum rate x herbicide	15	ns	ns	ns	ns

[a] Inoculum rates were 0, 1, 10, 100x.
[b] * = significant at $P \leq 0.05$, ns = not significant.
[c] Herbicide treatments included atrazine, metolachlor, metolachlor + benoxacor, EPTC + R-29148, the safener benoxacor and a water control.

Table 4.5. Effect of preemergent herbicides on rot of nodal roots[z] of sweet corn seedlings grown in soil infested with *Phoma terrestris*.

	Exp. 1	Exp. 2
No herbicide control	0.5 a	0.03 a
Atrazine	1.3 b	0.00 a
Metolachlor	0.7 a	0.05 a
Metolachlor + benoxacor	0.8 ab	0.00 a
EPTC + R-29148	1.1 b	0.17 b
Benoxacor	0.6 a	0.03 a

[z] Means from experiment 1 at 100x inoculum rate and exp. 2 across inoculum levels. Nodal root rot rating scale: 0 = healthy, 1 = 5-10, 2 = 11-25, 3 = 26-50, 4 = >50% necrotic. Means followed by the same letter within columns are not significantly different according to pairwise comparisons at the 5% probability level.

Table 4.6. Analysis of variance for the regression of amount of inoculum on root rot and dry weight of sweet corn seedlings grown in soil infested with *Phoma terrestris*.

	F-value	*P*-value
Exp. 1[a]		
Mesocotyl rot	152.60	<0.0001
Radicle root rot	321.10	<0.0001
Nodal root rot	14.56-62.40	≤0.0005
Dry weight	8.65	0.004
Exp. 2[b]		
Mesocotyl rot	74.12	<0.0001
Radicle root rot	106.85	<0.0001
Nodal root rot	1.92	0.17
Dry weight	5.95	0.02

[a] Inoculum rate by herbicide interaction was significant for nodal root rot and regression analysis was conducted within herbicide levels. The interaction was not significant for rot of the mesocotyl or radicle or for plant dry weight and regression analysis was conducted across herbicide levels.
[b] The inoculum rate by herbicide interaction was not significant for any response variables and regression analysis was conducted across herbicide levels.

***Drechslera* sp.** Inoculum rate by herbicide interaction was not significant for any response variables. Effect of herbicide was significant for severity of rot of nodal roots in experiment 1 and the radicle in experiment 2 (Table 4.7). Severity of rot of nodal roots was 67% greater with metolachlor in experiment 1 compared to the no herbicide control. In the second experiment, rot of the radicle was greater with metolachlor compared to EPTC + R-29148 and benoxocor, but not compared to the no herbicide control (Table 4.8). EPTC + R-29148 had no effect on severity of root rot. Plant dry weight was reduced 11-16% with

metolachlor compared to all other herbicide treatments in both experiments (Table 4.9).

Regression of amount of inoculum on severity of rot of the mesocotyl, radicle and nodal roots and on plant dry weight was significant in both experiments (Table 4.10). With an increase in amount of inoculum, root rot increased and dry weight decreased.

Table 4.7. Analysis of variance for severity of rot of the mesocotyl, radicle and nodal roots and dry weight of sweet corn seedlings grown in soil infested with *Drechslera* sp.

Sources	df	Mesocotyl rot	Radicle root rot	Nodal root rot	Dry weight
Exp. 1					
Inoculum rate[a]	3	*[b]	*	*	*
Herbicide[c]	5	ns	ns	*	*
Inoculum rate x herbicide	15	ns	ns	ns	ns
Exp. 2					
Inoculum rate[a]	3	*	*	*	*
Herbicide[c]	5	ns	*	ns	*
Inoculum rate x herbicide	15	ns	ns	ns	ns

[a] Inoculum rates were 0, 1, 10, 100x.
[b] * = significant at $P \leq 0.05$, ns = not significant.
[c] Herbicide treatments included atrazine, metolachlor, metolachlor + benoxacor, EPTC + R-29148, the safener benoxacor and a water control.

Table 4.8. Effect of preemergent herbicides on rot of radicle and nodal roots of sweet corn seedlings grown in soil infested with *Drechslera* sp.

	Radicle rot[y]	Nodal root rot[z]
No herbicide control	1.4 ab	1.3 a
Atrazine	1.7 b	1.4 ab
Metolachlor	1.6 b	1.7 c
Metolachlor + benoxacor	1.5 ab	1.6 bc
EPTC + R-29148	1.3 a	1.5 abc
Benoxacor	1.3 a	1.6 bc

[y] Means from experiment 2. Radicle rot rating scale: 0 = healthy, 1 = lesion present, 2 = 10-50, 3 = 51-99, 4 = 100% necrotic. Means followed by the same letter are not signficantly different according to pairwise comparisons at the 5% probability level.
[z] Means from experiment 1. Nodal root rot rating scale: 0 = healthy, 1 = 5-10, 2 = 11-25, 3 = 26-50, 4 = >50% necrotic. Means followed by the same letter are not significantly different according to pairwise comparisons at the 5% probability level.

Table 4.9. Effect of preemergent herbicides on dry weight of sweet corn seedlings grown in soil infested with *Drechslera* sp.[y]

	Dry weight (g)	
	Exp. 1	Exp. 2
No herbicide control	4.57 a[z]	2.95 a
Atrazine	4.57 a	2.96 a
Metolachlor	4.00 b	2.52 b
Metolachlor + benoxacor	4.52 a	3.00 a
EPTC + R-29148	4.56 a	3.00 a
Benoxacor	4.54 a	2.99 a

[y] Inoculum rate by herbicide interaction was not significant. Herbicide effects reported are across inoculum levels.
[z] Means with the same letter within columns are not significantly different according to pairwise comparisons at the 5% probability level.

Table 4.10. Analysis of variance for the regression of amount of inoculum on root rot and dry weight of sweet corn seedlings grown in soil infested with *Drechslera* sp.

	F-value	*P*-value
Exp. 1[a]		
Mesocotyl rot	138.72	<0.0001
Radicle root rot	107.59	<0.0001
Nodal root rot	74.73	<0.0001
Plant dry weight	103.94	<0.0001
Exp. 2[a]		
Mesocotyl rot	166.51	<0.0001
Radicle root rot	177.10	<0.0001
Nodal root rot	131.73	<0.0001
Plant dry weight	9.12	0.003

[a] Inoculum rate by herbicide interaction was not significant for any root rot response variables or dry weight and regression analysis was conducted across herbicide levels.

Fusarium graminearum. *F. graminearum* caused rot of only the mesocotyl. Inoculum rate by herbicide interaction on severity of rot of the mesocotyl was not significant, and the effect of herbicides was significant in experiment 2 only (data not shown). Inoculum rate by herbicide interaction was not significant for plant dry weight, however herbicides alone were (Table 4.11). Dry weight was 40-42% less for plants treated with metolachlor compared to all herbicide treatments in experiment 1 and 12-13% less with metolachlor compared to EPTC + R-29148 and benoxacor in experiment 2 (Table 4.12).

F. graminearum caused rot of the mesocotyl in both experiments (Table 4.11), however, regression of amount of inoculum on severity of rot of the

mesocotyl and dry weight was significant in experiment 2 only (Table 4.13). With an increase in amount of inoculum, severity of rot of the mesocotyl increased and dry weight decreased.

Table 4.11. Analysis of variance for severity of rot of the mesocotyl and dry weight of sweet corn seedlings grown in soil infested with *Fusarium graminearum.*

Sources	df	Mesocotyl rot	Dry weight
Exp. 1			
Inoculum rate[a]	3	*[b]	ns
Herbicide[c]	5	ns	*
Inoculum rate x herbicide	15	ns	ns
Exp. 2			
Inoculum rate	3	*	*
Herbicide	5	*	*
Inoculum rate x herbicide	15	ns	ns

[a] Inoculum rates were 0, 1, 10, 100x.
[b] * = significant at $P \leq 0.05$, ns = not significant.
[c] Herbicide treatments included atrazine, metolachlor, metolachlor + benoxacor, EPTC + R-29148, the safener benoxacor and a water control.

Table 4.12. Effect of preemergent herbicides on dry weight of sweet corn seedlings grown in soil infested with *Fusarium graminearum*.

	Dry weight (g)	
	Exp. 1	Exp. 2
No herbicide control	3.90 a[z]	2.70 abc
Atrazine	3.89 a	2.66 bc
Metolachlor	2.30 b	2.54 c
Metolachlor + benoxacor	3.84 a	2.77 abc
EPTC + R-29148	3.97 a	2.91 a
Benoxacor	3.94 a	2.89 ab

[z] Means with the same letter within columns are not significantly different according to pairwise comparisons at the 5% probability level.

Table 4.13. Analysis of variance for the regression of amount of inoculum on rot of the mesocotyl and on dry weight of sweet corn seedlings grown in soil infested with *Fusarium graminearum*.

	F-value	*P*-value
Exp. 1		
Mesocotyl rot	0.39	0.53
Dry weight	1.02	0.31
Exp. 2		
Mesocotyl rot	42.75	<0.0001
Dry weight	46.85	<0.0001

DISCUSSION

Effect of soil applied preemergent herbicides on growth and root rot of sweet corn seedlings grown in artificially infested soil was pathogen species dependent. *P. arrhenomanes* is primarily a root pruning pathogen and root rot caused by this organism is usually measured as reduced biomass. In soil infested with *P. arrhenomanes*, dry weight of sweet corn seedlings was reduced when treated with EPTC + R-29148. *P. arrhenomanes* also caused mild rot of the radicle and nodal roots and, in one experiment, severity of rot of nodal roots was reduced with EPTC + R-29148 at 100x rate of inoculum. However, root rot ratings can be misleading when root masses are reduced by root pruning. The reduction in severity of rot of the nodal roots in the EPTC + R-29148 treatment corresponded with a 30% decrease in plant dry weight compared to the noninfested control. The lower root rot rating likely is due to loss of rotted roots. *Phoma terrestris* caused rot on all three components of the root system, however effect of herbicide was seen on the nodal roots only. EPTC + R-29148 increased severity of rot of the nodal roots, but had no effect on severity of rot of the mesocotyl or radicle or on plant dry weight. *Drechslera* sp., like *P. terrestris*, caused rot on all components of the root system. Metolachlor increased severity of rot of the radicle and nodal roots in experiments 1 and 2, respectively and decreased dry weight of plants grown in soil infested with *Drechslera* sp. but had no effect on severity of rot of the mesocotyl. In contrast to our findings with *P. arrhenomanes* and *Phoma terrestris*, EPTC + R-29148 had no effect on root rot

caused by *Drechslera* sp. Metolachlor also reduced dry weight of plants grown in soil infested with *F. graminearum*.

EPTC without a safener can increase (Wyse et al, 1976) or decrease (El-Khadem and Papavizas, 1984) disease caused by root infecting Fusaria. EPTC increased seedling disease of cotton caused by *Thielaviopsis basicola* (Lewis and Papavizas, 1979) but decreased post-emergent damping-off of cotton caused by *Rhizoctonia solani* (El-Khadem and Papavizas, 1984). In contrast, in our study it had no effect on severity of rot of the mesocotyl caused by *Drechslera* sp. or *F. graminearum*, both of which caused pre- and post-emergent damping-off in preliminary screening trials (Appendix A). EPTC is used to control annual and perennial grasses and is required to have a safener for use in corn due to its toxicity to that crop. The herbicide inhibits shoot growth through the disruption of a variety of plant metabolic pathways (Ashton and Crafts, 1993; Vencill, 2002). Because we did not evaluate either EPTC or the safener R-29148 alone on growth or root rot of sweet corn, it is unclear whether the effect of EPTC + R-29148 on root rot and root pruning caused by *Pythium arrhenomanes* and *Phoma terrestris* is because of the safener or in spite of it.

Metolachlor is used primarily to control annual grasses and yellow nutsedge and is known for its toxicity to corn, especially under cool, wet conditions. It is a general growth inhibitor, especially of root elongation (Ashton and Crafts, 1981). In field applications, metolachlor is often applied with its safener benoxacor, which is thought to increase the metabolic degradation of

metolachlor by the crop plant (Ebert and Gerber, 1989; Fuerst et al, 1995). In our trials, the addition of the safener benoxacor to metolachlor (metolachlor + benoxacor) and benoxacor alone generally had no effect on biomass or root rot of sweet corn seedlings grown in soil infested with any of our pathogens. This suggests benoxacor protects corn seedlings treated with metolachlor from increased root rot caused by specific soilborne pathogens as well as phytotoxic effects of the herbicide.

Atrazine generally did not affect severity of disease caused by any of the corn root pathogens. A similar lack of effect of atrazine on root rot of sugarcane caused by *P. arrhenomanes* has been reported (Dissananyake et al, 1998). In contrast to our findings, increased seedling blight of field corn caused by *Fusarium culmorum* was seen with the application of atrazine (Percich and Lockwood, 1975). Atrazine is used for control of broadleaves and suppression of grasses. Mode of action of this herbicide is inhibition of plant growth due to blockage of photosynthesis (Ashton and Crafts, 1981). Corn is tolerant to atrazine, however growers limit their use of it due to potential groundwater contamination.

It has been suggested that crop plants could become more susceptible to pathogens at low inoculum levels when stressed or weakend by herbicides (Altman, 1993). However, when the inoculum rate by herbicide interaction was significant in our experiments, herbicide effects were seen at high inoculum rates. This is similar to what we found in a greenhouse trial examing the effect of

herbicides in field soil with differenct amounts of natural inoculum. As amount of inoculum increased, severity of root rot differed among herbicides (Hoinacki and Powelson, 2002b).

Many studies examining the effect of herbicides on root diseases have reported conflicting results (Altman, 1993). Our study indicates the effect of herbicides on root rot of sweet corn depends on the pathogen species present and its density. Therefore the effect of herbicides on disease may differ among fields. The evaluation of herbicides over a number of fields and the characterization of those fields for pathogens will help elucidate pathogen-herbicide interactions in the sweet corn root rot complex.

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Chapter 5

CONCLUSION

In response to the question "What is it in an individual scientist's relation to nature that facilitates the kind of seeing that eventually leads to productive discourse?" another corn researcher replied, "a feeling for the organism" (Keller, 1983). The six years spent on research on the sweet corn decline syndrome, only a part of which is presented here, has given me a "feeling" for this disease—its characteristics, its causes, and its complexities. It is my hope that the documentation of this feeling, this dissertation, leaves readers with a similar sense.

With respect to "productive discourse," our study of the symptomology and etiology of root rot of sweet corn should be a useful resource for root rot researchers in general and for researchers of sweet corn production in particular. The recognition that root rot is initiated early in the phenology of corn will facilitate the identification of problem fields and plantings at risk. Many organisms are associated with root rot of sweet corn, and our pathogenicity trials indicate several are primary pathogens. Moreover, the root rot rating system we developed was useful in establishing inoculum dose-disease response relationships for these pathogens and for identifying the different components of the root system that the various pathogen target. The rating system should be useful for researchers currently examining the effect of cultural and biological

control tactics on severity of root rot and for identifying resistant germplasm. Our study examining the effect of herbicides on root rot of sweet corn will benefit growers and helps explain the variable effects often seen with herbicides on root disease. The discovery that the effect of herbicides on severity of root rot of sweet corn is pathogen species dependent contributes to our growing understanding of pathogen-herbicide interactions.

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APPENDICES

APPENDIX A

Preliminary pathogenicity screening trials

OBJECTIVE

Fungi and oomycetes consistantly isolated from symptomatic sweet corn roots from 1997-2001 were evaluated in seven different pathogenicity trials for their ability to cause root rot.

METHODS

Inoculum production. Inoculum of isolates was prepared as described previously (Chapter 2).

Screening trial 1. In Trial 1, isolates of the same or similar species were combined and tested as bulked treatments. Treatments included *Fusarium oxysporum* (10 isolates: 9774-4b, 9798-2d, 9773-10c, 9741-6, 9794-3, 9752-9, 9775-6b, 97102-5c, 9746-9b, 97101-3d), *F. solani* (10 isolates: 9798-3c, 9774-5c, 9752-8, 9775-2c, 9793-1b, 97101-1d, 97102-3c, 97111-5d, 9793-9a), 2 sets of *Pythium arrhenomanes* (5 isolates each: 9793-10a, 9798-3a, 97102-2d, 97111-7d, 97101-2b and 9774-6a, 9794-6a, 9793-4c, 9798-5d, 97102-1c), *P. sylvaticum* (5 isolates: 9793-9b, 9798-2c, 9712-W4, 9741-6, 9752-5b), and other Pythia, including *P. torulosum, P. ultimum, P. inflatum*, and *P. irregulare* (10 isolates: 9741-3, 9712-E5c, 9712-W10, 9747-5b, 97101-4d, 9713-4, 9794-4, 97102-2b, 97102-5a, 9794-10). In addition, the two sets of *P. arrhenomanes* was combined

with *F. oxysporum* and *solani* inoculum for twomore treatments. Three grams of *Fusarium* inoculum or 2g of *Pythium* inoculum per 500ml soil were mixed with a sterile greenhouse soil mix (50% pumice, 25% peat, 25% soil) and placed in 550 ml plastic containers (9 cm x 12 cm) on a greenhouse bench. Noninfested soil served as the control. Sweet corn seeds, cv Golden Jubilee, were surface disinfested in 10% bleach for 5 min, rinsed three times in sterile water, and placed in sterile glass petri plates containing moistened filter paper (Whatman No. 1, 90mm). Plates were placed in an incubator at 32C until seeds germinated. Germinated seeds were planted 2.5 cm deep in containers. Treatments were replicated 15 times and arranged randomly. Plants were kept watered and fertilized once weekly with a water soluble plant food (N-P-K, 20-20-20) at the label rate. After 3 wk shoots were cut at the soil surface, measured and bagged. Roots were collected on a 0.5 cm screen, washed under running tap water, and bagged. Shoots and roots were dried at 60C and weighed.

Screening trials 2 and 3. Ten bulked isolates of *P. arrhenomanes* isolated in 1998 (Trial 2, isolates 9864-9, 9885-3, 9890-6, 9879-4, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9891-3) or 1999 (Trial 3, isolates 9962-11, 9966-5, 9960-11, 9958-7, 9947-17, 9947-7, 9947-16, 9945-1a, 9942-11, 9941-2j) were tested. The trials were conducted as outlined in Trial 1.

Screening trial 4. Eight isolates of *F. oxysporum* (9956-26, 9980-8, 9967-46, 9966-4, 9956-19, 9986-7, 9989-8, 9981-13) and four isolates of *F. solani* (9980-17, 9956-1, 9988-17, 9958-11), all isolated in 1999, were tested.

Each isolate was an individual treatment. Field soil was collected and seived through a 1.0 cm screen to remove rocks and clods. The soil was pastuerized at 90C for 1 hr on each of two consecutive days. Two grams of inoculum per 500ml soil was mixed with the soil and placed in 550 ml plastic containers (9 cm x 12 cm) on a greenhouse bench. Noninfested soil served as the control. Treatments were replicated five times and arranged randomly. Plants were treated and harvested as outlined in Trial 1.

Screening trial 5. Trial 5 was a partial repeat of Trial 4 with four of the *F. oxysporum* isolates (9989-8, 9967-46, 9981-13, 9956-26) and one isolate of *F. solani* (9988-17). Five grams of inoculum per 500ml soil and soil tubes (550 ml; 6.5 cm x 25 cm) were used. Sweet corn seeds were not germinated prior to planting. The trial was conducted for 4 wk. Plants were treated and harvested as outlined for Trial 1.

Screening trial 6. Twelve isolates of *F. oxysporum* from 1999 were tested as individual treatments (9962-9, 9956-2, 9988-15, 9948-110, 9960-8, 9966-17, 9973-3, 9960-3, 9967-27, 9962-2, 9962-12, 9958-6). The trial was conducted for 5 wk. All other practices were as outlined for Trial 5.

Screening trial 7. All treatments except *P. arrhenomanes* and the pathogen mix consisted of individual isolates. Eight isolates of *F. oxysporum*, including six of the 1999 isolates from Trial 6 (9956-2, 9966-17, 9960-3, 9958-6, 9948-110, 9967-27) and two isolated in 2000 (0016-4a, 0019-8d), 3 isolates of *F. graminearum* (0016-8a, 0016-3a, 0019-2a), 3 isolates of *Trichoderma* spp. (0015-

9a, BPP-3, 0015-2a), 3 isolates of *Phoma terrestris* (0015-9b, 0015-6a, 0015-6c), 1 isolate of *Drechslera* sp. (BPP-2), 13 bulked isolates of *P. arrhenomanes* (97102-2d, 97101-2b, 97102-1c, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9945-1a, 9942-11, 9966-5, 9947-7, 9947-17), and a bulked isolate mix (a 6:3:6:6:4 by weight combination of the six 1999 isolates of *F. oxysporum*, the three isolates of *F. graminearum*, the three isolates of *Trichoderma* spp., two of the *Phoma terrestris* isolates [0015-9b, 0015-6a] and the 13 isolates of *P. arrhenomanes*) were tested. Field soil was collected and sieved through a 1.0 cm screen to remove rocks and clods before pasteurizing at 90 C for 1 hr on each of two consecutive days. For each treatment 5 g of inoculum per 500 ml soil was mixed with the soil in a v-shaped shell mixer for 20 min and placed in 550 ml soil tubes (6.5 cm x 25 cm) on a greenhouse bench. Noninfested soil served as the control. Treatments were arranged in a randomized design and replicated five times. Sweet corn seeds, cv Golden Jubilee, were surface disinfested in 10% bleach for 5 min, rinsed three times in distilled water, and planted 2.5 cm deep. Tubes were kept moist and fertilized once weekly with a water soluble plant food (N-P-K, 20-20-20) at the label rate. At 6 wk post-planting plants were harvested. Shoots were cut at the soil surface and bagged. Roots were collected on a 0.5 cm screen, washed under running tap water and rated for disease Severity of root rot was rated on each of the three components of the root system for a total of 10 possible points: mesocotyl, 0=healthy, 1=lesion, 2=100% necrotic; radicle root, 0=healthy, 1=lesion present, 2= 10-50% necrotic, 3=51-99% necrotic, 4=100%

necrotic; nodal roots, 0=healthy, 1=5-10% necrotic, 2=11-25% necrotic, 3=26-50% necrotic, 4= >50% necrotic (Chapter 3). Shoots and roots were oven dried at 60 C and weighed.

Analysis. Effect of fungi on plant height and dry weight in trials 1-6 and on severity of rot of the mesocotyl, radicle, nodal roots, and the total root system, and on plant dry weight in trial 7 was examined with analysis of variance using PROC ANOVA or PROC GLM (when missing data points) of SAS Version 6.2 or 8.1. Means were separated with Fischer's Protected least significant difference ($P \leq 0.05$) (PROC ANOVA) or pairwise comparisons were made with least squares means at the 5% probability level (PROC GLM) (SAS Institute, Cary, NC).

RESULTS

Screening trial 1. Seedling height was reduced 15-24% and dry weight was reduced 28-48% in treatments containing *Pythium arrhenomanes* (Table A.1) ($P<0.0001$). Root tip browning and scattered lesions were also observed. The addition of *Fusarium oxyporum* and *solani* had no effect on height or dry weight ($P>0.05$). *P. sylvaticum*, the bulked *Pythium* species and *Fusarium oxysporum* and *solani* alone had no effect on seedling height or dry weight. Rootballs in these treatments appeared white and healthy.

Table A.1. Effect of inoculum on height and dry weight of sweet corn seedlings grown in soil infested with different fungi in Trial 1.

	Height (cm)[a]	Dry weight (g)[a]
Noninfested control	70.3	1.56
Pythium arrhenomanes, group 1[b]	53.4*	0.80*
P. arrhenomanes, group 2[c]	59.6*	1.13*
P. arrhenomanes, group 1 + *Fusarium oxysporum* and *solani*	56.9*	0.94*
P. arrhenomanes, group 2 + *F. oxysporum* and *solani*	58.9*	1.10*
P. sylvaticum[d]	70.4	1.42
Pythium spp.[e]	71.5	1.48
F. oxysporum[f]	69.8	1.47
F. solani[g]	71.1	1.52

[a]Least squares mean of 15 replicate samples.
[b]Isolates 9793-10a, 9798-3a, 97102-2d, 97111-7d, 97101-2b.
[c]Isolates 9774-6a, 9794-6a, 9793-4c, 9798-5d, 97102-1c.
[d]Isolates 9793-9b, 9798-2c, 9712-W4, 9741-6, 9752-5b
[e]Isolates 9741-3, 9712-E5c, 9712-W10, 9747-5b, 97101-4d, 9713-4, 9794-4, 97102-2b, 97102-5a, 9794-10 of species *P. ultimum, P. irregulare, P. torulosum,* and *P. inflatum.*
[f]Isolates 9774-4b, 9798-2d, 9773-10c, 9741-6, 9794-3, 9752-9, 9775-6b, 97102-5c, 9746-9b, 97101-3d.
[g]Isolates 9798-3c, 9774-5c, 9752-8, 9775-2c, 9793-1b, 97101-1d, 97102-6a, 97102-3c, 97111-5d, 9793-9a.
*Significantly different compared to the noninfested control at the 5% probability level ($P \leq 0.05$).

Screening trials 2 and 3. The 1998 (Trial 2) and '99 (Trial 3) isolates of *P. arrhenomanes* reduced seedling height 37-40% and dry weight 41-52% (Table A.2) ($P<0.0001$).

Table A.2. Effect of inoculum on height and dry weight of sweet corn seedlings grown in soil infested with *Pythium arrhenomanes* in Trials 2 and 3.

	Trial 2[a]		Trial 3[b]	
	Height (cm)[c]	Dry weight (g)[c]	Height (cm)[c]	Dry weight (g)[c]
Noninfested	36.2	0.49	45.3	0.62
Infested	21.8*	0.29*	27.3*	0.30*

[a]Isolates from 1998: 9864-9, 9885-3, 9890-6, 9879-4, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9891-3.
[b]Isolates from 1999: 9962-11, 9966-5, 9960-11, 9958-7, 9947-17, 9947-7, 9947-16, 9945-1a, 9942-11, 9941-2j.
[c]Average of 15 replicate samples.
*Significantly different compared to the noninfested control according to Fischer's Protected least significant difference ($P<0.0001$).

Screening trials 4 and 5. *Fusarium oxysporum* and *solani* had no effect on seedling height (P=0.66, 0.40) or dry weight (P=0.93, 0.29) (Table A.3).

Table A.3. Effect of inoculum on height and dry weight of sweet corn seedlings grown in soil infested with different isolates of *Fusarium oxysporum* or *solani* in Trials 4 and 5.

	Trial 4		Trial 5	
	Height (cm)[a]	Dry weight (g)[b]	Height (cm)[c]	Dry weight (g)[d]
Noninfested	74.3	3.06	90.4	4.58
F. oxysporum				
9981-13	74.4	3.09	88.1	3.90
9956-26	72.8	2.86	88.6	3.73
9967-46	69.6	2.82	88.1	3.83
9989-8	69.2	2.65	84.7	3.76
9986-7	71.9	2.98		
9980-8	70.4	2.71		
9966-4	69.9	2.89		
9956-19	69.7	2.73		
F. solani				
9988-17	73.6	2.86	88.5	3.8
9956-1	71.9	2.93		
9958-11	71.1	2.91		
9980-17	70.1	2.94		

[a]Average of 5 replicate samples. Treatment effect on height P=0.66 according to Fischer's Protected least significant difference.
[b]Average of 5 replicate samples. Treatment effect on dry weight P=0.97 according to Fischer's Protected least significant difference.
[c]Average of 5 replicate samples. Treatment effect on height P=0.41 according to Fischer's Protected least significant difference.
[d]Average of 5 replicate samples. Treatment effect on dry weight P=0.29 according to Fischer's Protected least significant difference.

Screening trial 6. Five of the 12 isolates of *F. oxysporum* significantly reduced plant dry weight 35-52% compared to the noninfested control ($P<0.01$) Four of those also reduced height ($P=0.10$) (Table A.4).

Table A.4. Effect of inoculum on height and dry weight of sweet corn seedlings grown in soil infested with different isolates of *Fusarium oxysporum* in Trial 6.

	Height (cm)[a]	Dry weight (g)[a]
Noninfested	66.3	0.46
9956-2	53.5*	0.30**
9966-17	55.8*	0.30**
9960-3	49.7*	0.22**
9958-6	58.0*	0.29**
9967-27	58.1	0.29**
9962-9	64.5	0.46
9988-15	68.1	0.43
9948-110	62.5	0.35
9960-8	65.4	0.38
9973-3	61.0	0.42
9962-2	62.7	0.37
9962-12	64.8	0.40
9988-15	68.1	0.43

[a]Average of 5 replicate samples.
*Significantly different compared to the noninfested control according to Fischer's Protected least significant difference ($P<0.10$).
** Significantly different compared to the noninfested control according to Fischer's Protected least significant difference ($P<0.05$).

Screening trial 7. The *Drechslera* isolate caused post-emergent damping-off in all 5 replicates. One *F. graminearum* isolate (0016-3a) caused pre- or post-emergent damping-off of all 5 replicates and another isolate (0016-8a) caused pre-emergent damping-off in 1 replicate. Two isolates of *Phoma terrestris* (0015-2b, 0015-6c) caused post-emergent damping-off in 1 replicate. One isolate of *Trichoderma* sp. (BPP-3) caused post-emergent damping-off in 1 replicate.

P. arrhenomanes and the pathogen mix significantly reduced seedling height 25 and 43%, respectively ($P<0.0001$) (Table A.5). No other fungi had any effect on height, however plants grown in soil infested with the pathogen mix were 24% shorter than those grown in the *P. arrhenomanes* infested soil ($P=0.0002$). *P. arrhenomanes* and the pathogen mix also reduced plant dry weight 57 and 83% ($P<0.0001$) (Table A.5). One isolate of *F. graminearum* (0016-8a) and 1 isolate of *Phoma terrestris* (0015-9b) also reduced plant dry weight 33 and 28% ($P=0.009$, 0.04).

Table A.5. Effect of inoculum on height and dry weight of sweet corn seedlings grown in soil infested with different fungi in Trial 7.

	Height (cm)[a]	Dry weight (g)[a]
Noninfested	97.4	3.38
Pythium arrhenomanes [b]	73.1*	1.44*
Pathogen mix[c]	55.2*	0.56*
Fusarium oxysporum		
9956-2	98.1	3.58
9966-17	99.9	3.86
9960-3	92.5	3.04
9958-6	99.8	3.94
9948-110	99.2	3.63
9967-27	96.8	3.98
0016-4a	93.7	2.85
0019-8d	97.3	3.52
F. graminearum		
0016-8a	92.8	2.28*
0019-2a	94.8	3.56
Trichoderma sp.		
0015-9a	96.8	2.80
BPP-3	95.1	3.05
0015-2a	94.8	3.05
Phoma terrestris		
0015-9b	98.6	2.42*
0015-6a	96.5	3.67
0015-6c	104.8	3.25

[a]Least square mean of 5 replicate samples.
[b]Isolates tested: 97102-2d, 97101-2b, 97102-1c, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9945-1a, 9942-11, 9966-5, 9947-7, 9947-17.
[c]The pathogen mix included: *P. arrhenomanes* isolates listed above; *F. oxysporum* isolates 9956-2, 9966-17, 9960-3, 9967-27, 9958-6, 0016-4a; *F. graminearum* isolates 0016-3a, 0016-8a, 0019-2a; *Trichoderma* isolates 0015-2a, 0015-9a, BPP-3; *Phoma terrestris* isolates 0015-9b, 0015-6a.
*Significantly different compared to the noninfested control at the 5% probability level ($P \leq 0.05$).

The severity of total root rot (mesocotyl + radicle + nodal roots) of plants grown in soil infested with *P. arrhenomanes*, the pathogen mix, and the three *Phoma terrestris* isolates was greater compared to the noninfested control ($P<0.006$) (Table A.6). When looking at the individual components of the root system, these fungi also caused greater radicle and nodal root rot compared to the noninfested control ($P<0.04$). The severity of mesocotyl rot was not greater on plants grown in any of the infested soils, however mesocotyl and radicle root rot was high in the noninfested control and lesion plating indicated contamination by *Trichoderma* spp. Two isolates of *F. oxysporum* had significantly less mesocotyl rot compared to the control ($P=0.02$) and 1 of those (9967-27) also had less radicle root rot ($P=0.04$). Four additional *F. oxysporum* isolates also had less radicle root rot ($P<0.04$).

The remaining two *F. oxysporum* isolates, one *F. graminearum* isolate, and two *Trichoderma* spp. isolates, had no effect on any of the plant growth or disease parameters compared to the noninfested control ($P>0.05$).

Table A.6. Effect of inoculum on severity of rot of the mesocotyl, radicle, and nodal roots, and total root system of sweet corn seedlings grown in soil infested with different fungi in Trial 7.

	Mesocotyl rot[a]	Radicle root rot[b]	Nodal root rot[c]	Total root rot[d]
Noninfested	1.4	1.6	0.2	3.2
Pythium arrhenomanes [e]	1.4	2.6*	1.6*	5.6*
Pathogen mix[f]	1.6	4.0*	3.2*	8.8*
Fusarium oxysporum				
9956-2	1.4	0.4*	0	1.8
9966-17	1.2	0.4*	0.2	1.8
9960-3	1.4	0.6*	0.6	2.6
9958-6	1.6	2.2	0.6	4.4
9948-110	0.8	0.6*	0.4	1.8
9967-27	0.6*	0.6*	0.4	1.6
0016-4a	0.6*	0.8	0.2	1.6
0019-8d	1.0	1.6	0.4	3.0
F. graminearum				
0016-8a	1.5	1.3	0.8	3.5
0019-2a	1.0	1.0	0.2	2.2
Trichoderma sp.				
0015-9a	1.2	0.4*	0	1.6
BPP-3	0.8	1.3	0.8	2.9
0015-2a	1.2	0.8	0	2.0
Phoma terrestris				
0015-9b	2.0	4.0*	2.0*	8.0*
0015-6a	1.8	4.0*	1.0*	6.8*
0015-6c	1.5	4.0*	1.3*	6.8*

[a] Least square mean of 5 replicate samples rated for mesocotyl rot on a scale of 0-2 where 0=healthy, 1=lesion, 2=100% necrotic.
[b] Least square mean of 5 replicate samples rated for radicle root rot on a scale of 0-4 where 0=healthy, 1=lesion present, 2=11-50%, 3=51-99%, 4=100% necrotic.
[c] Least square mean of 5 replicate samples rated for nodal root rot on a scale of 0-4 where 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, 4=>50% necrotic.
[d] Least square mean of 5 replicate samples on a scale of 0-10.
[e] Isolates tested: 97102-2d, 97101-2b, 97102-1c, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9945-1a, 9942-11, 9966-5, 9947-7, 9947-17.
[f] The pathogen mix included: *P. arrhenomanes* isolates listed above; *F. oxysporum* isolates 9956-2, 9966-17, 9960-3, 9967-27, 9958-6, 0016-4a; *F. graminearum* isolates 0016-3a, 0016-8a, 0019-2a; *Trichoderma* isolates 0015-2a, 0015-9a, BPP-3; *Phoma terrestris* isolates 0015-9b, 0015-6a.

Table A.6 (Continued)

* Significantly different compared to the noninfested control at the 5% probability level ($P \leq 0.05$).

APPENDIX B

Pathogen mixture trial

OBJECTIVE

To evaluate the ability of fungi and oomycetes to cause late season disease symptoms, pathogens shown to cause root rot in screening trials were combined and tested as a complex on sweet corn plants grown to maturity.

METHODS

A total of 600 L of soil from a field with a history of root rot was collected and two-thirds of it steam-pasteurized at 90 C for 1 hr on each of two consecutive days. A pathogen mixture was added to half the pasteurized soil. 4 g of 13 isolates of *P. arrhenomanes* (97102-2d, 97101-2b, 97102-1c, 9888-9, 9893-5, 9866-5, 9878-3, 9887-10, 9945-1a, 9942-11, 9966-5, 9947-7, 9947-17), 18 g of 3 isolates of *Phoma terrestris* (0015-9b, 0015-6a, 0015-6c), 10 g of 5 isolates of *F. oxysporum* (0101-1, 0101-5, 0101-6, 0101-7, 0101-8), 8 g of 2 isolates of *F. graminearum* (0016-3a, 0016-8a), and 9 g of 3 isolates of *Drechslera* sp. (BPP-1, BPP-2, 0016-7) per 20 L of soil was combined and mixed in a concrete mixer for 20 min. Soil for all treatments (naturally infested, artificially infested, uninfested) was placed in 20 L pots and treatments were arranged randomly in the Oregon State University Greenhouse courtyard and replicated 10 times. Sweet corn seeds, cv Golden Jubilee, were surface disinfested in 10% bleach for 5 min, rinsed three

times in distilled water and planted 2.5 cm deep, two per pot. Soils were kept moist and after emergence plants were fertilized once weekly with a water soluble plant food (N-P-K, 20-20-20) at the label rate. Emergence was recorded and at two weeks pots were thinned to one plant. When mature, ears were harvested and weighed. Plants were cut at the soil surface, oven dried at 60 C and weighed. Rootballs were removed from pots over a 0.5 cm screen to catch loose roots, washed and the three components of the root system were rated for rot as previously described (Chapter 3). Five 1-cm symptomatic root pieces per five plants per treatment were surface disinfested in 10% bleach for 1 min, rinsed in distilled water and plated on WA for recovery of pathogens.

Analysis of variance was conducted using the general linear model procedure of SAS Version 8.1 (SAS Institute, Cary, NC) to evaluate the effect of treatments on mesocotyl rot, radicle root rot, nodal root rot, total root rot, number of ears per plant, plant yield, and shoot dry weight. Means were generated by the method of least squares and pairwise comparisons were made at the 5% probability level ($P \leq 0.05$).

RESULTS

At 1 wk post-planting, plants grown in artificially or naturally infested soil were at 15% emergence versus 35 for plants grown in pasteurized soil. At 2 wk, emergence for plants grown in pasteurized soil and artificially infested soil

reached 90 and 100%, respectively. Plants grown in naturally infested soil reached a maximum of 75% emergence (Fig. B).

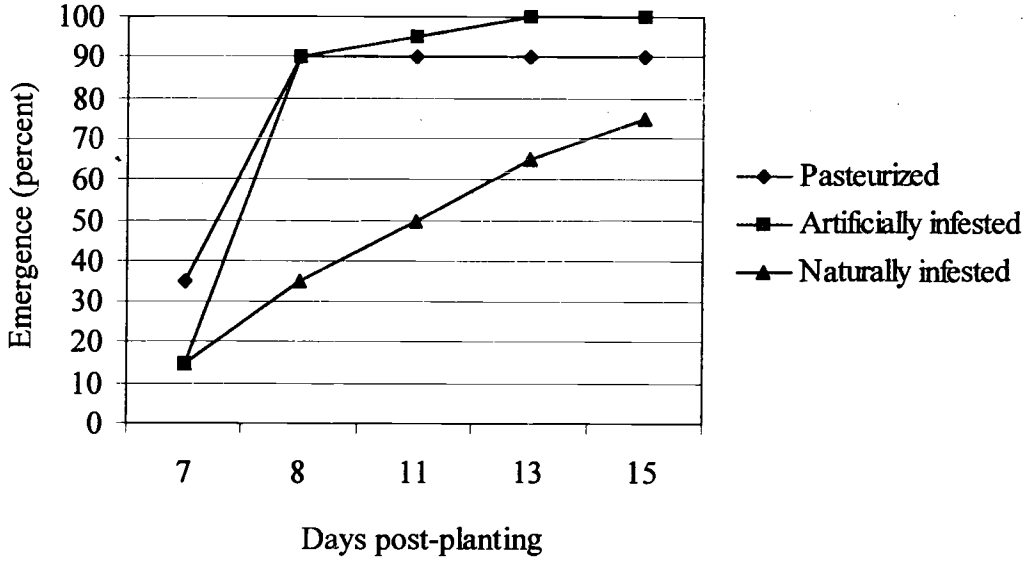

Fig. B. Effect of inoculum on emergence of sweet corn plants grown in naturally and artificially infested soil.

At harvest, mesocotyl root, radicle root rot, nodal root rot and total root rot was greater in the naturally and artificially infested soils compared to the pasteurized soil control ($P<0.0001$). Disease was also greater in the naturally infested soil compared to the artificially infested soil ($P<0.03$) (Table B.1).

Table B.1. Effect of natural and artificial inoculum on root rot of sweet corn plants grown in infested soil.

	Mesocotyl root rot[u]	Radicle root rot[v]	Nodal root rot[w]	Total root rot[x]
Noninfested	0.1 a[z]	0.3 a	0.1 a	0.5 a
Artificially infested[y]	1.2 b	2.9 b	1.6 b	5.8 b
Naturally infested	2.0 c	3.8 c	2.9 c	8.6 c

[u]Mesocotyl rot scale: 0=healthy, 1=lesion, 2=100% necrotic.
[v]Radicle roqt rot scale: 0=healthy, 1=lesion present, 2=11-50% necrotic, 3=51-99% necrotic, 4=100% necrotic.
[w]Nodal root rot scale: 0=healthy, 1=5-10% necrotic, 2=11-25% necrotic, 3=26-50% necrotic, 4= >50% necrotic.
[x]Combined total of mesocotyl + radicle + nodal root rot using the scales described above.
[y] Infested with a 4:18:10:8:9 by weight ratio of *Pythium arrhenomanes*, *Phoma terrestris*, *Fusarium oxysporum* and *F. graminearum*, and *Drechslera* sp.
[z] Means within columns followed by the same letter are not signficantly different at the 5% probability level ($P \leq 0.05$).

Treatment effects were significant for all yield and plant growth parameters ($P<0.0001$). The number of ears per plant was 1 and 1.9 for plants grown in naturally and artificially infested soil compared to 2.2 for plants grown in the pasteurized soil control. Plant yield was 22% greater for plants grown in pasteurized soil compared to those grown in artificially infested soil and more than twice that of plants grown in naturally infested soil. Shoot dry weight was 47-94% greater for plants grown in pasteurized soil compared to plants grown in the artificially and naturally infested soils (Table B.2).

Pathogens were recovered from symptomatic root tissue of plants grown in both the naturally infested and artificially infested soils.

Table B.2. Effect of natural and artificial inoculum on yield and plant growth parameters of sweet corn plants grown in infested soil.

	Number of ears/plant	Plant yield (g)	Shoot dry weight (g)
Noninfested	2.2 a[z]	675.2 a	202.6 a
Artificially infested[y]	1.9 b	553.3 b	137.4 b
Naturally infested	1.0 c	300.9 c	104.5 c

[y] Infested with a 4:18:10:8:9 by weight ratio of *Pythium arrhenomanes*, *Phoma terrestris*, *Fusarium oxysporum* and *F. graminearum*, and *Drechslera* sp.
[z] Means within columns followed by the same letter are not signficantly different at the 5% probability level ($P \leq 0.05$).

APPENDIX C

Cultivar trial

OBJECTIVE

To evaluate the effect of cultivar on severity of root rot of sweet corn in two naturally infested field soils.

METHODS

Soil from two fields with a history of root rot was collected and half of it was pasteurized at 90 C for 1 hr oneach of two consecutive days. Soil was placed in soil tubes (6.5 cm x 25 cm) on a greenhouse bench and sweet corn seeds (cvs Golden Jubilee, Bonus, GH 1861, GH 2298, Eliminator, GH 5702, GH 9595) were surface disinfested and planted 2.5 cm deep. Treatments were arranged randomly and replicated 5 times. Soils were kept watered and after emergence plants were fertilized once weekly with a water soluble plant food at the label rate (N-P-K, 20-20-20). At the 6^{th} leaf stage plants were harvested and rated for rot as described previously (Chapter 3). To evaluate the effect of cultivar on severity of rot of the total root system, analysis of variance was conducted using SAS Version 8.1 and means were separated with Fischer's protected least significant difference ($P \leq 0.05$) (SAS Institute, Cary, NC).

RESULTS

Severity of root rot was 17-55% less with GH 1861 compared to all other cultivars in both field soils, however it was significant in field soil 2 only (Table C). In both soils GH 1861 had the least disease and Bonus the most. Root rot ratings for related cultivars were similar to one another in both soils (GH 9595 and Bonus, GH 2298 and Golden Jubilee).

Table C. Effect of cultivar on severity of root rot[y] of sweet corn seedlings grown in naturally infested soil from two fields.

	Field 1	Field 2
GH 1861	5.0	2.7 a[z]
GH 5702	6.2	4.6 b
Eliminator	6.0	5.0 bc
GH 2298	6.4	5.0 bc
Golden Jubilee	6.8	5.0 bc
GH 9595	6.6	5.8 c
Bonus	6.8	6.0 c

[y] Scale of 0-10 for rot of the mesocotyl, radicle, and nodal roots. Mesocotyl rot scale: 0=healthy, 1=lesion, 2=100% necrotic. Radicle root rot scale: 0=healthy, 1=lesion present, 2=11-50%, 3=51-99%, 4=100% necrotic. Nodal root rot scale: 0=healthy, 1=5-10%, 2=11-25%, 3=26-50%, 4= >50% necrotic.

[z] Means with the same letter are not significantly different according to Fischer's protected least significant difference ($P \leq 0.05$).