Rhizoctonia Root Rot of Small Grains Favored by Reduced Tillage in the Pacific Northwest

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ABSTRACT

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Rhizoctonia root rot of wheat and barley caused by *Rhizoctonia solani* and responsible for bare patch in the field was identified in the United States for the first time. The disease was recognized at six sites: a research winter wheat plot and a commercial field of spring barley in Oregon, a commercial field of spring barley and research spring wheat plots in Washington, and a commercial field of durum wheat and one of winter wheat in Idaho. Diseased plants were severely stunted and occurred in distinct patches of various sizes. Seminal and crown roots of diseased plants had distinct brown sunken lesions and "pinched-off" pointed tips, symptoms previously reported as characteristic of Rhizoctonia root rot. Isolates of *R. solani* recovered from diseased tissue were multinucleate and produced identical root symptoms in greenhouse tests. At all sites where the disease occurred, the wheat or barley was either direct-drilled (no-tillage) into stubble, sown with minimal prior tillage, or sown the same day the soil was tilled. In experimental plots with winter wheat, there were 9.9, 2.8, and 1.4 patches per treatment (130 m²) in which no-tillage, reduced tillage, and conventional tillage, respectively, were practiced.

Rhizoctonia solani Kühn (teleomorph = Thanatephorus cucumeris (Frank) Donk) was reported as causing a severe root rot of wheat and oats in Australia by Samuel and Garrett in 1932 (32). Several names since have been used to describe this disease on cereals, including Rhizoctonia root rot (15), Rhizoctonia patch or bare patch (17,19,20), purple patch (14), and barley stunt disorder (23,24). Plants affected by this disease are severely stunted and occur in distinct patches, which presumably reflects the distribution and growth of the pathogen mycelium in soil. Infected roots have brown sunken lesions in which the cortex of the root is collapsed, leaving only the stele. Lesions may girdle and sever roots, leaving "pinched-off" pointed brown tips ("spear tip" or "needle point"); the roots appear severely pruned. Strains of R.

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solani that cause root rot are different from R. cerealis Van der Hoeven (6,16,28) and other strains of R. solani (35) that are responsible for sharp eyespot on wheat. Besides Australia (2,14,15, 17,19,20,30-32), the same or a similar root disease of cereals occurs in England (9,11), Canada (3), Scotland (23,24), and possibly South Africa (33,34). Dana (8), Bessey (4), and Richards (29) all have mentioned the occurrence of Rhizoctonia root rot of wheat in the United States, but in no case was the disease fully described and there are no previous reports in the United States of the bare patch symptom in the field.

In Australia, the severity of disease caused by R. solani is greater when the crop is sown by direct-drilling than when sown after cultivation (18,20,22,25,30). Various forms of conservation tillage, including "no-till" seeding, are being rapidly adopted for all cereals in the United States. In 1984, patches of severely stunted common wheat, barley, and durum wheat caused by R. solani were identified in all three Pacific Northwest states of Idaho, Oregon, and Washington. This paper reports results that verify the occurrence of Rhizoctonia root rot of small grains in the Pacific Northwest and the relationship of disease severity to conservation tillage.

MATERIALS AND METHODS

Fields with patches of severely stunted plants were recognized independently by six crop consultants, county agents, or agronomists working in their respective, widely separated areas in the Pacific Northwest in the spring of 1984. Each site was visited to obtain plant samples as well as information on cropping history in the fields and tillage practices.

Isolation and identification of *Rhizoctonia* spp. Wheat roots and stems were washed for 1 hr under running tap water. Tissue samples from roots, subcrown internodes, coleoptiles, and leaf sheaths near the soil were placed on 2% water agar or water agar supplemented with $100~\mu g$ of rifampin per milliliter. After 3-5 days of incubation at 22 C, *Rhizoctonia*-like fungi were transferred to homemade potato-dextrose agar (PDA) (200 g of potatoes, 20 g of dextrose, and 15 g of Difco agar in 1 L of water) supplemented with $100~\mu g$ of rifampin per milliliter, then transferred to PDA.

Isolates were identified as R. solani on the basis of accepted morphological characteristics (27). The nuclear condition was determined using a Giemsa staining procedure (10), a rapid-staining procedure with 0.5% aniline blue in lactophenol (13), and a modification of an acridine orange staining procedure used to stain nuclei of wheat and barley roots (12). For acridine orange staining, 5-mm-diameter plugs from the edges of 3-day-old cultures were trimmed of excess agar and placed in a solution of 0.001% acridine orange for 5 min. A single plug was placed on a slide, squashed with a coverslip, and viewed with an Olympus BH-2 microscope equipped with reflected light fluorescence.

The nuclear condition of candidate isolates was compared with that of R. solani AG-3 previously isolated from wheat (not causing root rot) and with that of four binucleate isolates of R. cerealis (1.80, 5.80, 14.80, and 23.80) from wheat culms with sharp eyespots from Ohio (16). Anastomosis between candidate isolates and tester strains from five anastomosis groups was tested on 2% distilled water agar by the methods of Parmeter et al (26).

Pathogenicity of isolates. Inoculum was prepared by growing individual isolates on sterile oat kernels in 1,000-ml widemouth flasks. Oats were inoculated with one plate of a I-wk-old PDA culture per flask, incubated at 22 C for 10 days, shaken, incubated for 10 more days, and then air-dried before use.

The pathogenicity of isolates was tested using plastic tapered tubes (2.5 cm in diameter × 16.5 cm long), each with a hole at the bottom (Ray Leach Conetainer Co., Canby, OR), supported in a hanging position on plastic racks. Each cone was filled about half full with sterile vermiculite followed by a 5-g layer of a fumigated (methyl bromide) Shano silt loam (pH 6.3). One colonized oat kernel or two 8-mm-diameter plugs from a 5day-old PDA culture of the candidate isolate were placed on the top of the soil and covered with a 2-cm-thick layer of vermiculite. Each cone received 10 ml of water and then was incubated for I wk at 15 C before sowing. McDonald and Rovira (21) previously demonstrated that preincubation significantly increases the severity of root rot. Two wheat seeds (cultivar Stephens) were sown in each cone and the seed covered with a 2-cmthick layer of vermiculite. Ten milliliters of deionized water was added to each cone and the plants were grown for 3 wk.

The severity of root rot was determined by a rating scale of 0-5, where 0= no root disease and 5= all roots destroyed (21). Seedlings were rated also for necrosis of the coleoptile.

Location and cropping systems of fields with Rhizoctonia root rot. Six areas (or fields) with evidence of Rhizoctonia root rot were identified. The first site was planted to winter wheat and was located in a series of reduced-tillage research plots on the Oregon State University Experiment Station at Hermiston. The site had been in alfalfa for 3 yr before receiving various tillage treatments followed by sowing wheat in the fall of 1983. A second site, near Clyde, WA, in Walla Walla County comprised several fields of spring barley sown in early 1984 into stubble of the 1983 wheat crop. No-tillage (chemical fallow) had been used in some fields, but a portion of one field received a single cultivation

followed immediately (same day) by sowing. The frequency and size of patches were similar whether no-tillage or one tillage followed immediately by sowing was used. The third site with Rhizoctonia root rot was in Nez Pierce County near Lewiston, ID, in a field of winter wheat sown without prior tillage into winter wheat residue in the fall of 1983. The fourth site was on the Washington State University Dryland Research Unit at Lind in an experimental plot where winter wheat was removed with a single cultivation with a Lely Rotera because of severe winter injury and the site sown that same day to spring wheat. The fifth site was near Tetonia, ID, in a field of durum wheat under pivot irrigation and sown after only one superficial tillage and in which potatoes had been grown the previous year. The sixth site was in Umatilla County, OR, in a field of spring barley direct-drilled into stubble.

Effects of tillage on disease severity. The severity of Rhizoctonia root rot was assessed in an existing experiment at Hermiston, OR, that was designed to study the effects of conservation tillage on wheat production after 3 yr of alfalfa. In this experiment, Rhizoctonia root rot developed unexpectedly. Treatments (6.1 ×21.3 m) consisting of wheat grown after conventional tillage (CT), reduced tillage (RT), or no-tillage (NT) were established in a randomized block design and replicated six times. In the CT treatment, the soil was plowed and packed, then seed was sown with a Melroe disc drill; in the RT treatment, the soil was worked with a sweep (paraplow) and packed, then sown with a Melroe disc drill; and in the NT treatment, a Crust Buster hoe drill was used for sowing.

The severity of disease was determined by counting the patches in each treatment. Because of variability in patch size, greater weight was given to larger patches. A patch up to about 20 cm in diameter was considered a single patch, a patch up to 1 m in diameter was considered two patches, and a patch greater than 1 m was considered three patches.

RESULTS

Disease symptoms. Generally, patches of plants with Rhizoctonia root rot were irregularly circular with distinct borders and ranged from about 20 cm to 3 m in diameter (Fig. 1). Plants inside the patch were stunted and chlorotic; the barley plants in the patches near Clyde, WA, showed symptoms of sulfur deficiency, but those outside the patches were normal green. Virtually every plant of durum wheat in the 100-ha field at Tetonia, ID, was stunted.

Lesions on diseased roots were brown and often girdled the root. The cortices of infected roots collapsed and the roots had "pinched-off" pointed tips (Fig. 2). Lesions were detected on the primary and secondary roots as well as on the subcrown internodes. The basal leaf sheaths near the soil surface often were discolored brown.

Isolation, identification, and pathogenicity. Rhizoctonia-like fungi were isolated from the roots, subcrown internodes, coleoptiles, or discolored leaf sheaths of samples of wheat or barley from all but the Lind site. Isolates from roots were not obtained from this site despite repeated attempts, probably because the samples were too old. Recovery of isolates from plants at all sites was easiest from the leaf sheaths and most difficult from roots. The difficulty in isolating the pathogen from old, diseased roots has been reported (15,23,32). Pythium spp. and Fusarium spp. were also abundant in infected roots.

All Rhizoctonia-like isolates, except the isolate from spring barley in Umatilla County, were identified as R. solani by mycelial and cytological characteristics previously described (27). The Umatilla isolate was lost before being identified. Characteristics of the isolates included multinucleate cells in young vegetative hyphae, branching near the distal septum

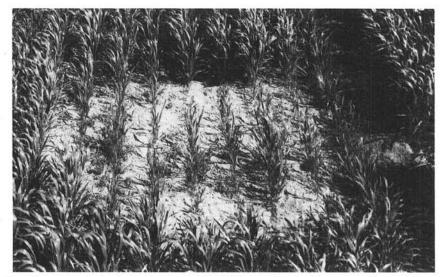


Fig. 1. Bare patch of winter wheat caused by *Rhizoctonia solani* in an experimental plot at Hermiston, OR.

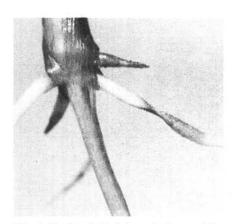


Fig. 2. Root rot of winter wheat caused by *Rhizoctonia solani*. The pointed, rotted tip (spear tip) is a typical symptom on roots.

of cells in young vegetative hyphae, constriction of the branch and formation of a septum in the branch near the point of origin, mycelium with a shade of brown, and dolipore septa (27). Colonies on PDA varied from brown to tan. Some isolates produced abundant sclerotia, whereas others produced few sclerotia. All Rhizoctonia isolates from plants with root rot failed to anastomose with AG-1 (isolates 43, 245, and 465), AG-2 (isolate 229), AG-2-1 (isolate 457), AG-2-2 (isolates 460 and 481), AG-3 (isolate 141), AG-4 (isolate 283), and AG-5 (isolate 441), tester isolates previously described by Adams and Butler (1).

Two pathogenic types of R. solani were isolated from wheat in bare patches at Hermiston. Two isolates from roots, represented by isolate H-1, caused mainly a root necrosis and stunting of the seedlings but little discolorization of the coleoptile. Seven isolates from leaf sheaths, represented by isolate H-18, caused a discolorization of the coleoptile and a twisting and deformity or preemergence blight of the plumule and only slight root rot (Table 1). The isolate from a root of barley from Clyde produced a severe root rot and was similar in its effects to isolate H-1 from Hermiston, whereas all eight isolates from roots and leaf sheaths of durum wheat produced symptoms similar to isolate H-18. Six isolates from wheat roots from Lewiston produced only a root tip necrosis of wheat in the tube test.

Effects of tillage. At Hermiston, 9.9 patches were present in the wheat in the NT treatments compared with 2.8 and 1.4 patches in the RT and CT treatments, respectively (LSD 5% = 6.6).

DISCUSSION

As far as we know, this report is the first fully documented case of Rhizoctonia root rot (bare patch) of small grains in the United States. The pathogen has probably been present in soil on the Hermiston field station and probably in many other fields of the Pacific Northwest for many years. The root rot and patches of diseased plants (bare

patch) closely resemble symptoms originally described for the disease in South Australia (32). Particularly diagnostic is the "spear tip" or "needle point" symptom on roots that results from lesions girdling and eventual severing the roots. R. solani was isolated from diseased tissue and caused a root rot of wheat when tested in the greenhouse. No other fungi isolated from the roots caused a root rot typical of that caused by R. solani.

Our identification of the candidate isolates as R. solani is based on accepted morphological and cytological characteristics; however, the identity of the isolates could not be confirmed because of their failure to anastomose with isolates of AG-1 to AG-5 available to us. Murray (23) reported that the R. solani responsible for barley stunt disorder was AG-3. In contrast, Neate (25) was unable to place R. solani from wheat roots in South Australia into any anastomosis group and suggested that a new group be erected for the wheat isolates. Further studies are needed on the Pacific Northwest isolates to determine if they too are unique. We recognize that only a small number of tester isolates was used in this study, and as further testers become available the Pacific Northwest, isolates may be placed in one or more known anastomosis groups.

Of particular interest is the difference of isolates from the roots and lower leaf sheaths of diseased plants. When tested in the greenhouse, some isolates infected mainly the roots and others infected mainly the shoot with little root infection. Whether the shoot infection would produce sharp eyespot lesions on the culms of adult plants is not known, because only seedlings were used in this study. However, it is possible, since some eyespots were present on coleoptiles of some seedlings. Although isolates of Rhizoctonia usually attack either roots or shoots of wheat (5-7,16,35), Elnur and Chesters (11) reported an isolate of R. solani that infected both roots and shoots. Our isolates were pathogenic mainly on roots or the shoots but nonetheless attacked both.

Table 1. Effects of isolates of Rhizoctonia solani on the growth of wheat

Isolates ^x	Source of isolate	Root rating ^y	Emergence (%)	Plants with necrotic coleoptiles (%)
H-1	Root	2.5 a ^z	98 a	20 b
H-18	Leaf sheath	0.3 b	40 b	100 a
Check	****	0.0 с	100 a	0 с

[&]quot;Isolates were tested in plastic tubes containing (from bottom to top) vermiculite, fumigated soil, two 8-mm-diameter plugs from a PDA culture of the candidate isolate, vermiculite, two wheat seeds, and vermiculite.

The future importance of this disease in the Pacific Northwest is unknown. The disease occurred mainly in fields where minimum or NT was used before sowing. In the experimental plots at Hermiston, the frequency of patches was greater in NT than in CT plots and intermediate in RT plots. The disease also occurred in fields that were tilled and then sown immediately. It is possible that some time must elapse between tillage and sowing to control this disease. Our observations on the effects of tillage on the disease correspond to those previously reported in Australia (18,20,22,25,30); namely, the disease is more common in cereals grown under conservation tillage. Rhizoctonia root rot could become more important in the Pacific Northwest with the increasing practice of growing small grains with conservation tillage.

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^{*}Isolates H-I and H-18 were from Hermiston. The symtpoms produced by H-I and H-18 were similar to the symptoms produced by the isolates from barley at Clyde and durum wheat at Tetonia, respectively.

Severity of root disease was rated on a scale of 0-5, where 0 = no root disease and 5 = all roots destroyed.

Means in the same column followed by the same letter are not significantly different (P = 0.05). Each value is the mean of five replicates with 10 plants per replicate.

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