Rhizoctonia solani on White Lupine

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ABSTRACT

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Rhizoctonia solani anastomosis group 5 (AG-5) was identified as a major pathogen of white lupine (*Lupinus albus*). The symptoms observed included reduced nodulation, seed rot, stem nipping, stem lesions, reduced root growth, and apical bud mortality. Terminal bud mortality has not been previously described on lupine. Strains of AG-5 hypovirulent on potato are nearly nonpathogenic on lupine. Strains of AG-1 and AG-4 also infected lupine but produced only very small stem lesions. Strains of AG-2 and AG-3 did not infect lupine.

White lupine (*Lupinus albus* L.) recently was introduced into the northeastern United States. In Maine, lupine currently is being tested as a rotation crop with potato for use as a green manure/nitrogen source and/or grain. In these tests, a high degree of damping-off and other root disorders were observed. Also, mortality of terminal shoot tips was observed and thought to be a genetic defect because of its high frequency. Isolations from affected plants showed the major causes of diseases were *Rhizoctonia solani* Kühn, *Pythium* spp., and *Fusarium* spp.

Weimer (10) reported that Rhizoctonia root rot was the most widely distributed and destructive disease of lupine. *R. solani* attacks seedlings before emergence and often kills large numbers of seedlings due to infection at the soil surface after they emerge. Diseased plants usually have yellowish green foliage and may not set pods. Also, he reported that both *Pythium* spp. and *Fusarium* spp. caused root rots to a lesser degree than *R. solani*. Roemer (9) reported that under humid, spring conditions, root and stem lesions caused by *Fusarium* spp., *Rhizoctonia* spp., and *Pythium* spp. often were found. He stated that these diseases seem to have little economic importance, but severity may increase if lupines are too often grown on the same site.

All of the above pathogens are reported as attacking lupines in the United States (5,10) but not in Maine. Baker (2) did not include the terminal bud killing we have observed as a symptom on lupines caused by *R. solani*.

In this paper, we report that isolates of *R. solani* AG-5 primarily attack lupine and that terminal bud mortality caused by *R. solani* is a major symptom on infected plants in Maine.

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MATERIALS AND METHODS

Identification of pathogens affecting lupine. Diseased lupine plants from a planting date study were selected and isolations were made on acidified water agar (AWA) and potato-dextrose agar (PDA). Single spore or hyphal tip isolations were made of each fungus isolated and cultures were identified to genus. Multinucleate *R. solani* isolates were identified by nuclear staining young vegetative cells with 0.5% aniline blue in lactophenol combined with a wetting agent (3,6). Isolates determined to be *R. solani* were tested to determine their anastomosis groupings by pairing each isolate with tester isolates from each of AG-1, AG-2, AG-3, AG-4, and AG-5 (7,8).

Pathogenicity tests. Two tests were conducted. The first identified the pathogenicity of isolates within anastomosis groups of *R. solani* on lupine, and the second determined the pathogenicity of AG-5 strains isolated from soil and potatoes on lupine and if strains hypovirulent on potato also were hypovirulent on lupine. In the first test, virulent isolates of *R. solani* AG-1 through AG-5 were selected from our culture collection and designated as AG-1 through AG-5 throughout the test. In the second test, virulent isolates of AG-5 (Rs9, Rs10, Rs18, Rs19, Rs53, and Rs59) were obtained from S. M. Tavantzis, University of Maine, Orono, ME, and RO1 was obtained from our culture collection. Rs1 and Rs5 were hypovirulent on potato (S. M. Tavantzis, *personal communication*), RO2 (AG-3) and all of the above strains were isolated from potato or soil, and RO16 was isolated from lupine. All isolates were grown at 25 C on PDA. Inoculum was prepared by blending the contents of two 7-day-old cultures of *R. solani* in 800 ml of sterile distilled water for 10 s in a Waring blender. Inoculum density was determined by spreading 1 ml of suspension on the surface of five PDA plates. After 3 days at room temperature,

the number of colonies appearing on the plates were counted and the mean of the five plates was determined. This produced a viable inoculum suspension with three to five propagules per milliliter. Four hundred milliliters of inoculum was mixed thoroughly with enough premoistened Redi-Earth (W. R. Grace & Company, Cambridge, MA), a sterile, soilless mix, to fill eight 20.5-cm-diameter pots. Mixing was accomplished by placing moist Redi-Earth into a large plastic bag, adding the inoculum, and rotating the bag until mixing was complete. This procedure provides repeatable results (S. S. Leach, *unpublished*). All inoculum was prepared in this manner. Eight pots filled with moist Redi-Earth that had blended suspensions of only agar added represented the control.

In the first test, 10 seeds of the lupine cultivar Ultra were planted in each pot. A randomized complete block design was used with eight replications in a greenhouse maintained at 21-C days and 13-C nights. Supplemental light to maintain 12-hr days was provided with cool-white fluorescent lamps. A liquid fertilizer (Peters 20-18-19 Peat-Lite mix for soilless mixes, W. R. Grace & Company) was applied once a week to provide 473 mg/L of nitrogen. Plants were watered as needed. Emergence, final stands and disease incidence, and type of symptom and severity were recorded when plants in the control pots flowered, approximately 35 days after planting.

With the identification of AG-5 as the anastomosis group that caused the most severe disease on lupine, a second pathogenicity test was conducted. Ten AG-5 isolates and one AG-3 isolate identified above were used. Twelve 11.5-cm-diameter square plastic pots were filled with moist Redi-Earth mix previously infested with 600 ml of a suspension of *R. solani*. Five seeds of the lupine cultivar Ultra were planted 3 cm deep in each pot. The pots were placed in an environmental chamber in a randomized complete block design and maintained at 13.5 C and 95% RH with 12-hr days supplied by cool-white fluorescent lamps. Water was added to individual pots as needed. Emergence, stand, and disease data were recorded. The disease data recorded were divided into type of symptoms observed including stems nipped, stems with lesions, and terminal buds killed. Random seedlings were selected and isolations were made to verify the presence and identification of the pathogen.

RESULTS

Isolates obtained from diseased lupines were in three major genera: *Pythium*, *Rhizoctonia*, and *Fusarium*. *Rhizoctonia* spp. and *Fusarium* spp. were most often found together in root and stem lesions. *Fusarium* spp. also were found on diseased pods and beans. *Pythium* spp. were found only on stems and not associated with other fungi. *Pythium* spp. caused damping-off and *Fusarium* spp. caused some root necrosis. All isolates (107) of *R. solani* were identified for anastomosis group and 84 were AG-5. The remaining isolates were AG-1 (three), AG-4 (two), or unidentifiable (16). Because our major interest was to determine which anastomosis groups of *R. solani* infect lupine and if they also attack potato, *Pythium* spp. and *Fusarium* spp. were not further identified.

Anastomosis groups attacking lupine. Only isolates of *R. solani* AG-5 reduced the rate of emergence of lupine (Table 1). Isolates of anastomosis groups 1, 4, and 5 all produced stem lesions, with those in AG-5 causing the greatest incidence and severity. Isolates of AG-1 and AG-4 produced only small (2–4 mm) lesions, whereas AG-5 produced lesions 1–4 cm long that often girdled the stem. Isolates from all groups reduced root growth compared with check plants. The number of plants with nodules was reduced significantly by isolates AG-4 and AG-5 (Table 1). From these data, we concluded that AG-5 is the major anastomosis group in which isolates attack lupine and that the AG-3 potato strain is of minor importance in the lupine disease complex.

Table 1. Disease in lupine (*Lupinus albus*) caused by different anastomosis groups of *Rhizoctonia solani*[v]

Anastomosis group	Emergence[w]	Final stand[x]	Plants without nodules	Percent diseased plants[y]
1	7.1 a[z]	8.0	0.5 bc	7.8 a
2	7.6 a	8.0	0.0 c	0.0 a
3	7.5 a	8.5	0.0 c	0.0 a
4	6.9 a	8.6	1.2 b	11.5 a
5	2.4 b	8.1	4.3 a	70.6 b
Control	7.5 a	8.1	0.0 c	0.0 a

[v] Data present the mean of eight replications of 10 plants planted per pot per replication.
[w] Mean number of plants emerged per pot 21 days after planting.
[x] Mean number of plants per pot 35 days after planting.
[y] Percent diseased plants = plants showing any symptom of disease caused by *R. solani* based on final stand counts.
[z] Numbers within a column followed by the same letter do not differ significantly ($P = 0.05$) according to Duncan's new multiple range test.

Symptoms produced on lupine when planted in soil infested with *R. solani* AG-5 are illustrated in Figure 1. All symptoms except the killing of terminal buds (Fig. 2) also were found with strains AG-1 and AG-4.

Pathogenicity of AG-5 isolates. Eight AG-5 isolates from potato produced disease symptoms on lupine similar to those produced by an AG-5 isolate obtained from lupine. The AG-3 isolate did not infect lupine. Two of the AG-5 isolates (Rs1 and Rs5) produced very minor symptoms on lupine plants. These isolates were known to be hypovirulent on potato (S. M. Tavantzis, *personal communication*), and they did not significantly reduce stand nor produce disease symptoms on lupine (Table 2).

All of the eight isolates that produced disease on lupine reduced emergence and stand, produced stem lesions, rotted stems, and produced terminal bud mortality (Table 2). The percentage of plants with terminal buds killed was high (45–85%) for the eight AG-5 isolates. The two hypovirulent isolates (Rs1 and Rs5) produced very little terminal bud killing of 0 and 2.2%, respectively, compared with 85.7% for isolate Rs53. The number of stems with lesions or number of stems nipped was not correlated with the number of terminal buds killed.

DISCUSSION

R. solani AG-5 is a major pathogen of white lupine. The symptoms expressed were the same as those described by Weimer (10). However, ours is the first report of terminal bud killing by *R. solani* on lupine. Lupine plants affected by the terminal bud killing do not produce any pods, which results in a direct yield loss. The high incidence of this symptom observed in our study (up to 85%) shows that it might become a major problem where inoculum densities are high. Roemer (9) reported that root and stem lesions were of little importance where rotations were used but were responsible for yield losses when lupines were monocropped. In contrast, our field observations indicated that terminal bud killing was independent of rotation; it was observed in equal amounts in both rotated and monocropped plots.

The large number of plants found without nodules when grown in AG-5 infested soil also is of interest. One of the reasons for using lupine in a rotation is because it efficiently fixes nitrogen. If this ability is restricted by *R. solani*, the importance of lupine as a rotation crop and supplier of organic nitrogen could be greatly reduced in the presence of this pathogen.

The lack of pathogenicity of AG-3 isolates on lupine indicates that lupine in a potato rotation would not be greatly affected by isolates of *R. solani* that usually attack potato. In comparison, *R.*

Fig. 1. Symptoms caused by *Rhizoctonia solani* AG-5 on lupine. Left to right, rotted seed, stem nipped and poor roots, terminal bud killed, lesions on stem and few secondary roots, and healthy plant.

Fig. 2. Terminal bud of lupine killed by *Rhizoctonia solani* AG-5.

Table 2. Disease symptoms on lupine caused by isolates of *Rhizoctonia solani* anastomosis groups AG-3 and AG-5

Isolate	Emergence[u]	Final stand[v]	Stems with lesions	Stems nipped	Percent terminal buds killed
RO1	1.8 ef[w]	3.3 b–d	1.2 ab	1.0 a–c	67 a
RO2[x]	2.2 e	3.4 b–d	0.0 c	0.0 c	0 c
Rs53	0.4 g	2.8 d	1.7 a	1.8 a	85 a
Rs59	2.6 de	3.8 ab	1.2 ab	0.7 bc	47 ab
Rs1[y]	4.1 a	4.4 a	0.0 c	0.7 bc	0 c
Rs19	1.0 fg	2.9 cd	1.9 a	1.6 ab	79 a
Rs5[y]	3.9 ab	4.4 a	0.1 c	0.5 c	2 c
Rs9	3.2 b–d	3.4 b–d	0.4 bc	0.7 bc	76 a
Rs18	2.6 de	3.4 b–d	0.6 bc	1.1 a–c	53 ab
Rs10	2.6 de	3.7 a–c	0.6 bc	0.5 c	72 a
RO16[z]	2.4 de	3.2 b–d	1.6 a	1.2 a–c	41 b
Control	3.5 a–c	4.0 ab	0.0 c	0.0 c	0 c

[u] Mean number of plant per pot emerged 21 days after planting.
[v] Mean number of plants per pot 35 days after planting.
[w] Data reported are the means of 12 replications with a maximum of five plants per replication. Numbers within a column followed by the same letter do not differ significantly ($P = 0.05$) according to Duncan's new mutiple range test.
[x] AG-3 isolated from potato.
[y] Hypovirulent strains from potato; all remaining virulent isolates from potato.
[z] AG-5 isolated from lupine.

solani AG-5 is a pathogen of both potato and lupine, with lupine the more sensitive host. A potato-lupine rotation could increase the soil density of AG-5 such that it could become a serious pest to potato as well as lupine. In a related study, we found that potatoes grown after lupine had 30% more viable AG-5 sclerotia than potatoes after an oat-clover rotation (S. S. Leach, *unpublished*). Abe and Tsuboki (1) also have observed sclerotia of AG-5 on potato tubers. Because *R. solani* AG-5 has been isolated from both soil and potato in Maine (4), a lupine-potato rotation could result in soil populations increasing to a density sufficient to cause severe losses in both lupine and potato.

The data reported show that *R. solani* AG-5 can cause serious losses in lupine by reducing nodule formation, attacking roots and stems, and by killing the apical bud but does not cause the characteristic Rhizoctonia disease of potato.

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