Components of Resistance to *Rhizoctonia solani* Associated with Two Tall Fescue Cultivars

D. E. Green II, Environmental Horticultural Science Department, California Polytechnic State University, San Luis Obispo 93407; L. L. Burpee, Department of Plant Pathology, Georgia Station, University of Georgia, Griffin 30223-1797; and K. L. Stevenson, Department of Plant Pathology, University of Georgia, Athens 30602-7274

ABSTRACT

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Components of resistance to *Rhizoctonia solani* in the tall fescue cultivars Kentucky 31 (moderately resistant) and Mojave (susceptible) were evaluated under controlled environmental conditions. Size and expansion rate of foliar lesions were recorded on 100 individual plants of each cultivar. Lesions on the first fully expanded leaves of 6- to 10-month-old inoculated plants covered a significantly greater proportion of the leaf width on cv. Mojave compared to Kentucky 31. Rate of lesion expansion was also greater on cv. Mojave than on Kentucky 31. Lesion size and rate of expansion were greater on the second compared to the first fully expanded leaf of both cultivars. Wider leaves and slower lesion expansion are two components responsible for the greater level of resistance to *R. solani* in cv. Kentucky 31 compared to Mojave tall fescue.

Additional keywords: brown patch, *Festuca arundinacea*, host resistance, *Rhizoctonia* blight

Rhizoctonia blight, caused by *Rhizoctonia solani* (teleomorph: *Thanatephorus cucumeris*), is a serious disease of tall fescue (*Festuca arundinacea*) in the southeastern United States (5). When night temperatures >15°C coincide with leaf wetness durations >10 h, infection by *R. solani* results in light-brown foliar lesions with dark-brown margins (5,6,30,31). As symptoms progress, circular patches of necrotic foliage form and often coalesce to produce large areas of blighted turf.

Results of numerous studies have shown that tall fescue cultivars differ in susceptibility to *R. solani* (4,9,13,16,24,34-36,38). Although breeding programs have improved resistance to *Rhizoctonia* spp. in tall fescue, current levels of resistance are not adequate to limit disease to levels that are acceptable (<10% disease severity) to owners or managers of tall fescue turf (14). Linear reductions in the severity of *Rhizoctonia* blight were detected in blends of tall fescue cultivars as the proportion of cv. Kentucky 31 (moderately resistant to *R. solani*) was increased in a blend (15). Limited disease severity in cv. Kentucky 31 (K-31) was associated in part with a foliar canopy of low density that resulted from a lower shoot reproductive rate when

compared to the *Rhizoctonia* spp.-susceptible cv. Mojave. These observations confirmed findings which showed that shoot density influenced severity of foliar blights caused by *R. solani* in tall fescue and rice cultivars (13,28). However, reduction in disease severity even when the low shoot density of cv. K-31 was compensated for by an increase in seeding rate (15) suggests that additional factors function in resistance of tall fescue to *R. solani*.

Several constitutive factors (formed prior to infection) including epicuticular wax thickness (21-23,37), cuticle thickness (26), cell wall calcium content (2,3), and tolerance of cuticle and epicuticular wax to pathogen enzymes or toxins may function in *Rhizoctonia* spp. resistance (18). In addition, induced resistance factors (formed after infection) such as hypersensitive responses (21), and increased production of pathogenesis-related (PR) proteins, may also be involved (1). In conjunction with these factors, age and maturity of the foliage or plant have been reported to influence expression of components of resistance in several host-pathogen relationships (10,27,29,37).

The assessment of host resistance is based on the quantification of symptoms, or growth and development of a pathogen on a host. Incidence and severity of symptom expression is influenced by several factors or components, such as infection efficiency (infection frequency), time from inoculation to symptom expression (incubation period), and rate of lesion expansion (25). Analysis of differences in these components of resistance between

cultivars that exhibit differential susceptibility to *R. solani* should provide insight into potential mechanisms of *Rhizoctonia* spp. resistance in tall fescue. Such knowledge could also lead to more precise breeding programs for the development of *Rhizoctonia* spp. resistance, as has been demonstrated for resistance to *Bipolaris sorokiniana* in tall fescue (19). The objectives of our study were to identify possible components of *Rhizoctonia* spp. resistance in the tall fescue cvs. K-31 and Mojave, and to determine the influence of leaf age on these components, by comparing differences in size, area, and expansion rate of foliar lesions caused by *R. solani*.

MATERIALS AND METHODS

Plant culture. Controlled environment experiments were conducted 10 March through 5 May and 6 June through 14 November 1997 at the Georgia Experiment Station in Griffin, Georgia. Approximately 20 seeds of cvs. K-31 or Mojave were evenly distributed over the surface of calcined clay (Turface, Applied Industrial Materials Corp., Deerfield, IL) in each of 100 Styrofoam cups (9 cm in diameter by 12 cm deep) per cultivar. The seeds were lightly watered twice daily and, at 7 days post-germination, the seedlings were thinned to three per cup. At 3 weeks post-germination, the plants were thinned to one per cup, so that each cultivar was represented by 100 plants. The plants were grown in a greenhouse at 23 to 30°C for 9 months. Beginning 1 week after seeding, plants were watered daily and fertilized weekly with a solution containing 474, 99, and 373 ppm of N (4% NH₃-, 6% NO₃-, 10% CO(NH₂)₂-forms of N), P (P₂O₅), and K (K₂O), respectively. At 1 month prior to inoculation, the plants were trimmed to a 10-cm height and transferred to 20-cm-diameter pots containing calcined clay. Throughout the study, diazinon (O,O-diethyl O-[2-isopropyl-6-methyl-4-pyrimidinyl] phosphorothioate), isotox (acephate [O,S-dimethyl acetylphosphoramidothioate]) and lambda-cyhalothrin ([1 α (S), 3 α (Z)]-($\pm$)-cyano (3-phenoxyphenyl) methyl-1-3-(2-chloro-3,3,3-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylate) were applied as needed to control fungus gnats (*Bradysia coprophila*) and greenbugs (*Schizaphis graminum*).

Inoculum production and inoculation. Inoculum consisted of portions of foliage

Corresponding author: D. E. Green II
E-mail address: dgreen2@calpoly.edu

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excised from a tall fescue plant and infested in vitro by *R. solani*. Sections were excised from the first fully expanded leaf of a 9- to 12-month-old cv. K-31 plant. The 2-mm² leaf sections were placed on dry filter paper in glass petri dishes and autoclaved for 20 min. After the petri dishes had cooled, the filter paper was moistened with 1.5 ml of sterile distilled water. At 48 h prior to preparation of the leaf pieces, *R. solani* AG 1-Ia (isolate Rh136) was grown on acidified potato dextrose agar. In the first experiment, three or four 2-cm² agar discs were removed from the leading edge of the *R. solani* colony and placed on each filter-paper disc approximately 20 mm apart and approximately 35 mm inside the periphery of the autoclaved dishes containing the leaf sections. The cultures were incubated at 26°C for 4 days to allow colonization of the leaf sections. In an effort to improve inoculum uniformity in the second experiment, only one 2-cm² agar disc was removed from the leading edge of the *R. solani* colony and placed at the center of each filter-paper disc inside the autoclaved petri dishes containing equally spaced leaf sections. Cultures were then incubated at 26°C for 5 days to allow colonization of the leaf sections.

In the first experiment, prior to inoculation, the first fully expanded leaf on each of 10 shoots per plant was selected and marked, for ease of identification, with a 5-mm-diameter hole made with a paper punch approximately 3 cm from the leaf tip. In the second experiment, the first and second fully expanded leaves on each of the 10 selected shoots was marked as described for the first experiment. The remainder of the unmarked foliage was trimmed to a 20-cm height to expedite assessments. Plants were then lightly misted with tap water and the marked leaves were inoculated by placing one colonized leaf section at the center of the adaxial surface approximately 15 cm distal to the leaf collar. After inoculation, plants were placed at 100% relative humidity in a mist chamber situated inside a growth chamber set to maintain a diurnal

cycle of 14 h light at 30°C and 10 h dark at 25°C.

In the first experiment, 75 plants of each cultivar were screened in a randomized complete block design with nine blocks over time. Due to space limitations in the mist chamber, nine plants of each cultivar were arbitrarily selected and tested in the first eight blocks, and the final three plants per cultivar were assessed in the ninth block. In the second experiment, the remaining 25 plants of each cultivar were screened along with 29 arbitrarily selected plants per cultivar previously assessed 3 months earlier in the first experiment. Inoculation of the latter group of plants served as a test for reproducible results from the first experiment. The second experiment consisted of a randomized complete block design with six blocks over time and nine plants of each cultivar per block.

Data collection and statistical analysis. The width and length of lesions were measured to the nearest millimeter on inoculated leaves of eight shoots per plant at 18, 30, and 48 h post-inoculation. At 48 h post-inoculation, 3 of the 10 inoculated leaves were arbitrarily selected from each plant for determination of mean lesion area. These selected leaves were removed from the plant, placed between two glass cover slips in 75% glycerol, and pressed between two layers of corrugated cardboard at 26°C for 3 to 7 days. Gray-scale images of the mounted leaves were obtained through a computer-aided image scanner (SprintScan, Polaroid Corp., Cambridge, MA) adapted with a microscope-slide mount (PathScan Enabler, Meyer Instruments, Inc., Houston). Lesion area was measured with the aid of data image analysis software (SigmaScan, Jandel Scientific, San Rafael, CA).

Values of lesion width divided by leaf width and lesion area divided by (leaf width × 30 mm) were calculated, where 30 mm was the maximum length of field that could be scanned using the microscope-slide mount. Lesion expansion rate was estimated for lesion width and length between 18 and 30 h, and 30 and 48 h post-

inoculation using the formula $\Delta y/\Delta t = (y_{(i+1)} - y_i)/(t_{(i+1)} - t_i)$, where y_i was either lesion width or length and t_i was the time at the i th rating (7). A proportional expansion rate expressed as the percentage of lesion expansion across the leaf width was also calculated between 18 and 30 h, and 30 and 48 h, post-inoculation from values of lesion width divided by leaf width using the same formula.

The SAS UNIVARIATE procedure (Statistical Analysis Systems Institute, Inc., Cary, NC) was used to test for normality of each measured response variable within a treatment. Analysis of variance procedures were used to detect differences in response variables between cultivars. To simplify analysis and avoid inflation of the error term due to the unbalanced design, treatment means of each component measured were calculated for each block. These means were derived from data collected on all plants of each cultivar within a block, so that blocks became replicates. This procedure was equivalent to using the block by treatment interaction as the error term in a model containing all plants.

RESULTS

In each experiment, lesions initially appeared between 15 and 24 h post-inoculation on both cultivars as several small (<1 mm diameter) spots with a water-soaked appearance surrounding the inoculum. The spots coalesced into a necrotic lesion typical of Rhizoctonia blight symptoms within 1 to 2 h after observation of initial symptoms. Frequently, a dark-brown band formed around the margin of a lesion between 18 and 30 h post-inoculation. An increase in lesion size was associated with the development of water-soaked spots, followed by necrosis, on the outside of the brown margin. On some plants, no brown margin was observed, and evidence of lesion expansion was seen as water-soaked tissues preceding necrosis.

In the first experiment, lesion width at 18 h post-inoculation on the first fully expanded leaf was significantly greater ($P \leq 0.03$) on cv. K-31 than on Mojave (Table 1 and Fig.

Table 1. Probability values from analysis of variance of the size of foliar lesions on tall fescue cultivars Kentucky 31 and Mojave inoculated with *Rhizoctonia solani*

Experiment, source	df	Component of resistance										
		Lesion width (mm)			Lesion length (mm)			Lesion area (cm ²)	Lesion width: leaf width			Lesion area: leaf area
		18 h ^a	30 h	48 h	18 h	30 h	48 h	48 h	18 h	30 h	48 h	48 h
1												
Block	8	<0.01	<0.01	0.02	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.02	0.04
Cultivar	1	0.03	0.07	0.08	0.15	0.85	0.76	0.81	0.30	0.04	0.05	0.15
2												
Block	5	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.01	<0.01	<0.01	<0.01	0.03
Cultivar	1	<0.01	0.33	0.06	<0.01	0.07	0.12	0.91	<0.01	<0.01	<0.01	0.50
Leaf ^b	1	<0.01	0.04	0.28	<0.01	0.06	0.15	...	<0.01	0.01	0.09	...
Cultivar × leaf	1	0.40	0.71	0.48	0.39	0.87	0.65	...	0.39	0.67	0.37	...

^a Hours post-inoculation.

^b Comparison between the first and second fully expanded leaves.

1A). Although leaves of cv. K-31 were significantly wider ($P \leq 0.03$) than those of Mojave (*data not shown*), the proportion of lesion width to leaf width was significantly greater ($P \leq 0.05$) at 30 and 48 h post-inoculation on cv. Mojave than on K-31 (Table 1 and Fig. 1B). Lengthwise rate of lesion expansion and proportional rate of lesion expansion (percent expansion across leaf width per hour) between 18 and 30 h post-inoculation were significantly greater ($P = 0.05$) on cv. Mojave than on K-31 (Table 2 and Fig. 1C and D). No significant differences between cultivars were detected for any of the other components measured in the first experiment.

In the second experiment, leaf width was again significantly greater ($P < 0.05$) for cv. K-31 plants than for Mojave (*data not shown*). In contrast, the proportion of lesion width to leaf width was significantly greater ($P < 0.01$) at 48 h post-inoculation on the first fully expanded leaf of cv. Mojave when compared to K-31 (Table 2 and Fig. 2A). Widthwise rates of lesion expansion between 30 and 48 h post-inoculation were significantly greater ($P \leq 0.04$) on cv. Mojave than on K-31 (Table 2 and Fig. 2B). In addition, proportional rate of lesion expansion was significantly greater between 18 and 30 h ($P \leq 0.03$), and between 30 and 48 h post-inoculation ($P <$

0.01) on cv. Mojave compared to K-31 (Fig. 2C).

Analysis of differences in resistance components between the first and second fully expanded leaves, in the second experiment, revealed that lesion length at 18 h post-inoculation and lesion width and the proportion of lesion width to leaf width at 18 and 30 h were significantly greater ($P \leq 0.04$) on the second fully expanded leaf compared to the first fully expanded leaf (Table 1 and Fig. 3A-C). However, leaf age did not significantly influence lesion expansion rate (Table 2). Lesion width and length at 18 h post-inoculation were greater on cv. Mojave than on K-31 on the second fully expanded leaf (Fig. 3D and E) but not on the first fully expanded leaf. The proportion of lesion width to leaf width was also significantly greater ($P \leq 0.05$) on the second fully expanded leaf of cv. Mojave compared to K-31 at all three sampling periods (Fig. 3F). No significant differences in lesion expansion rate or proportional expansion rate were detected between cultivars on the second fully expanded leaf in the second experiment.

DISCUSSION

In addition to previously reported effects of shoot density (13,15), results of this study indicate that the difference in susceptibility of the tall fescue cvs. Mojave and Kentucky 31 to *R. solani* is a function of variance in leaf width and lesion expansion rate between the cultivars. The narrower leaves of cv. Mojave resulted in lesions that covered a greater proportion of leaf width when compared to K-31 at 48 h post-inoculation, when leaf width became a limiting factor for further lateral lesion expansion. No apparent differences in lesion width or lateral spread were observed between tall fescue plants screened in a controlled environment and natural infection of tall fescue turf mown at 7.5 cm height in the field. It is possible, however, that lesion length and longitudinal spread may occasionally be limited by the proximity of a lesion to the leaf base on closely mown tall fescue turf. In the field where

Fig. 1. Development of foliar lesions on the tall fescue cultivars Mojave and Kentucky 31 inoculated with *Rhizoctonia solani* in the first experiment. (A-B) Lesion size on the first fully expanded leaf. (C-D) Lesion expansion rate on the first fully expanded leaf. *, ** indicates a significant difference at $P \leq 0.05$ and $P \leq 0.01$, respectively.

Table 2. Probability values from analysis of variance of the expansion rate of foliar lesions on tall fescue cultivars Kentucky 31 and Mojave inoculated with *Rhizoctonia solani*

Experiment, source	df	Component of resistance					
		Lesion width expansion rate (mm/hr)		Lesion length expansion rate (mm/hr)		Proportional expansion rate (% lesion width expansion/hr)	
		18-30 ^a	30-48	18-30	30-48	18-30	30-48
1							
Block	8	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Cultivar	1	0.19	0.24	0.05	0.63	<0.01	0.73
2							
Block	5	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Cultivar	1	0.30	0.04	0.13	0.33	0.03	<0.01
Leaf ^b	1	0.71	0.26	0.88	0.47	0.96	0.80
Cultivar × leaf	1	0.86	0.44	0.84	0.60	0.97	0.34

^a Interval (hours post-inoculation) between sampling periods.

^b Comparison between the first and second fully expanded leaves.

tall fescue is typically mowed at 5 to 7.5 cm, *R. solani* frequently infects within 2 to 3 cm of the leaf base where dew and guttation fluids collect. A greater proportion of leaf width exhibiting necrosis near the leaf base in cv. Mojave may explain, in part, observations of severe Rhizoctonia blight on Mojave under controlled environmental and field conditions (15).

Variations in lesion expansion rates between the cultivars suggest that cvs. K-31 and Mojave differ quantitatively in induced resistance to *R. solani*. In bean (*Phaseolus vulgaris*), infection by *R. solani* was immediately followed by cytoplasmic aggre-

gation, a common result of host injury, in cells 2 to 5 layers away from the infection site (18). Bateman and co-workers (2,3) found that an increased mobilization of calcium ions into the cells surrounding lesions of *R. solani*-infected bean hypocotyls was associated with limitation of lesion expansion. In the cells surrounding lesions, calcium was observed to bind with pectic substances in the cell walls to form calcium pectate that was resistant to degradation by polygalacturonase produced by the pathogen. Anuratha et al. (1) observed a delayed lesion spread in moderately resistant rice cultivars infected with *R. solani* compared to susceptible cultivars. They suggested that differences in the induction of two chitinases and two β -1,3 glucanases, between the moderately resistant cultivars and susceptible cultivars, were responsible for the slower spread of the lesions on the resistant hosts.

A lack of consistent cultivar differences in lesion size early in pathogenesis (18 h post-inoculation) suggest that constitutive

forms of resistance, such as cuticle thickness, had little influence on the differences in disease severity observed between cvs. K-31 and Mojave. These findings differ from results of previous research with other crops which showed that resistance to *R. solani* was primarily due to constitutive factors (e.g., cuticle thickness, epidermal resistance to pathogenic enzymes; 11,12,17,20,22,32,37). For example, Stockwell and Hanchey (32) reported that increased cuticle thickness of bean plants was associated with the inability of *R. solani* to form infection structures. It may be possible that constitutive forms of resistance differ in tall fescue cultivars, but the heterogenic nature of tall fescue cultivars may have prevented the detection of these types of resistance expressed in individual plants.

Cuticle thickness of plants also has been shown to vary with plant age and environmental conditions (8,32,33). Younger plants and those grown in the shade or under controlled environmental conditions

Fig. 2. Development of foliar lesions on tall fescue cultivars Mojave and Kentucky 31 inoculated with *Rhizoctonia solani* in the second experiment. (A) Lesion size on the first fully expanded leaf. (B-C) Lesion expansion rate on the first fully expanded leaf. *, ** indicates a significant difference at $P \leq 0.05$ and $P \leq 0.01$, respectively.

Fig. 3. Development of foliar lesions on tall fescue cultivars Kentucky 31 and Mojave inoculated with *Rhizoctonia solani* in the second experiment. (A-C) Lesion size between the first and second fully expanded leaves. (D-F) Lesion size on the second fully expanded leaf between cvs. Kentucky 31 and Mojave. *, ** indicates a significant difference at $P \leq 0.05$ and $P \leq 0.01$, respectively.

were found to have a thinner cuticle compared to older plants or those grown under field or greenhouse conditions (8). Removal or abrasion of the cuticle, simulating weathering effects in the field, resulted in increased formation of infection structures of *R. solani* and increased disease severity in other crops (22,32,37). Cuticle thickness remains a possible component of *Rhizoctonia* resistance in tall fescue grown under field conditions.

We found that leaf age influenced susceptibility to *R. solani*. However, unlike results from previous studies (28,37), which showed that less mature foliage was more susceptible to *R. solani*, we found that the older second, fully expanded leaf of tall fescue exhibited greater susceptibility to *R. solani* prior to 30 h post-inoculation. Initiation of senescence, observed as chlorosis and necrosis of the leaf tip, in the second fully expanded leaf may have been associated with the observed differences in susceptibility. Similar results were observed in wheat (*Triticum aestivum*) to *Leptosphaeria nodorum*, where longer incubation and latent periods were associated with the more mature F-2 leaf compared to the flag leaf (10).

Results of this study revealed components of *Rhizoctonia* spp. resistance in tall fescue that have not been reported previously. The greater leaf width of cv. K-31 compared to Mojave that resulted in a lower proportion of lesion width to leaf width is unlikely to be used in selective breeding programs because wide leaf blades are an undesirable aesthetic characteristic of turfgrass. However, the reduced lesion expansion rate observed in cv. K-31 may be significant in breeding programs designed to increase *Rhizoctonia* spp. resistance in tall fescue cultivars. Numerous tall fescue cultivars with improved *Rhizoctonia* spp. resistance are currently being developed and marketed for use in the turfgrass industry. However, the observed delayed lesion expansion rate in cv. K-31 could offer advanced tools for further selection of tall fescue breeding lines and cultivars with greater *Rhizoctonia* spp. resistance through identification and development of genetic markers of biochemical factors associated with this resistance component.

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