Influence of Temperature-Soil Water Status Interactions on the Development of Summer Patch in *Poa* spp.

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ABSTRACT

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Kentucky bluegrass (*Poa pratensis*) cultivars Aspen and S-21 and annual bluegrass (*Poa annua*) were treated with live or killed inoculum of *Magnaporthe poae* and incubated in growth chambers at four temperatures (20, 25, 30, 35 C) and four water-stress conditions corresponding to inferred soil matric potential ranges of >−0.05, −0.05 to −0.40, −0.40 to −0.80, or −0.80 to −1.20 MPa. All plants grown at 35 C died of supraoptimal temperature stress before disease symptoms developed. Disease developed only in plants receiving live inoculum. The area under the disease progress curve for disease incidence was greatest in annual bluegrass at all temperatures, and at 30 C for all grasses. At 20 C, disease incidence and symptom expression were greatest at −0.05 to −0.40 MPa. At 25 and 30 C, disease incidence and symptom expression were greatest at >−0.05 MPa. There was a reduction in quality of asymptomatic plants within pots inoculated with live *M. poae* when compared with plants receiving killed inoculum. Summer patch was most severe in plants growing at supraoptimal temperature stress of 25 or 30 C and high soil moisture. Temperature, but not severe drought stress, appears to be a key factor in summer patch development.

Summer patch, caused by *Magnaporthe poae* Landschoot & Jackson (16), is one of two or more diseases that mimic the symptoms of Fusarium blight (8) and Fusarium blight syndrome (21). Turfgrasses affected by summer patch include Kentucky bluegrass (*Poa pratensis* L.), annual bluegrass (*P. annua* L.), and creeping red fescue (*Festuca rubra* L.)(10). Summer patch is one of the most destructive diseases of Kentucky bluegrass in the United States (10).

Summer patch first appears in Maryland in late June to early July, as hot and frequently dry weather conditions become prevalent. Most observations and field studies that assessed the environmental conditions associated with disease development were conducted when the disease was confused with Fusarium blight (3,4,8,9,11,18). A reassessment of the environmental conditions required for the development of patch diseases of Kentucky bluegrass is needed for each of the new organisms associated with symptoms that resemble Fusarium blight.

Smiley and Craven Fowler (21,23) have shown that temperature is an important factor in the development of summer patch. They found that *M. poae* grew through Kentucky bluegrass sod at 21–24 C, but that disease development was slow at these temperatures. Summer patch severity was amplified by raising soil temperatures to 29 or 30 C (23). The role of drought stress, reported as an important factor in Fusarium blight, however, has not yet been clarified.

The objective of this study was to examine the effect of controlled temperature and soil water content interactions on the development of symptoms, disease severity, and overall quality of plants receiving either live or killed inoculum of *M. poae*.

MATERIALS AND METHODS

Soil. Sassafras sandy loam (fine-loamy, siliceous, mesic Typic Hapludult), pH 6.5 and 1.9% organic matter, was collected from

the University of Maryland Turfgrass Research and Education Facility in Silver Spring in November of 1987. Soil was passed through a 1-cm-mesh sieve, pasteurized at 180 C for 30 min, and then spread on paper in the greenhouse to air-dry. After the soil was uniformly dry, subsamples were taken to determine moisture content. Plastic pots (11.5- × 11.5- × 9-cm-deep) were placed on a scale, filled with soil to a uniform weight, and tamped. Aqua-Gro (Marshall Thomas Co., Inc., Lexington, KY) (300 ml L^{-1} H_2O) was applied as a wetting agent to all pots with a Hozon applicator (Metal Seals Co., Willoughbee, OH) at a 1:15 (Aqua-Gro solution/water) ratio to aid in wetting the soil.

Grasses. Two Kentucky bluegrass cultivars, Aspen and S-21, and annual bluegrass were evaluated. Aspen was chosen because it had exhibited good resistance to natural infections of summer patch in University of Maryland turfgrass cultivar evaluations, and S-21 had exhibited high susceptibility. *P. annua* was included because it is also a host for *M. poae*. Kentucky bluegrasses were seeded at the rate 0.50 g per pot, and annual bluegrass at 0.25 g per pot. To aid in the prevention of damping-off by *Pythium* spp., metalaxyl at 2.4 ml L^{-1} was applied as a soil drench when 80–90% of the first leaves had appeared and 3 wk later. Plants were mowed weekly to a height of 6 cm and clippings were removed. Plants were fertilized biweekly with 473 mg L^{-1} N from a balanced, water-soluble 20-20-20 N-P-K fertilizer and were treated with the insecticide abamectin at the rate of 0.66 ml L^{-1} H_2O to control western flower thrips (*Frankliniella occidentalis* (Pergande)) and two-spotted spider mites (*Tetranychus urticae* Koch). Plants received occasional foliar applications of the fungicide chlorothalonil at the rate 4 ml L^{-1} H_2O as needed to control a *Fusarium* spp. that induced foliar blighting.

Inoculum. Inoculum was prepared by soaking seed of tall fescue (*Festuca arundinacea* Schreb.) in water overnight. The seed was rinsed, placed in (100- × 80-mm-deep) Pyrex petri jars, and autoclaved for two 1-hr periods 24 hr apart. Cultures of *M. poae* (ATCC 60239) grown on potato-dextrose agar (Difco Laboratories, Detroit, MI) were ground in a blender in enough sterile distilled water to form a loose slurry and then were poured aseptically onto cooled, autoclaved, tall fescue seed. Jars were incubated in the dark at 25 C for 8 wk, during which time they were periodically shaken to promote uniform colonization of the seed. Heat-killed inoculum was prepared by autoclaving colonized inoculum for two 45-min intervals.

Plants were removed from the greenhouse after 12 wk and inoculated before placement in the growth chamber and the imposition of water-stress treatments. A 2-cm-deep core of soil was removed from the center of each pot with a cork borer, and 0.5 g of the appropriate inoculum was placed into the hole. The core of soil then was replaced and tamped down.

Temperature and water-stress treatments. After inoculation, plants were placed in a growth chamber set at either 20, 25, 30, or 35 C. Growth chambers were lighted for 12 hr per day with approximately 100 $\mu E\ m^{-2}\ sec^{-1}$ of light. Plants were allowed to dry to one of four water-stress treatment levels, corresponding to inferred soil water matric potential ranges of >-0.05, -0.05 to -0.40, -0.40 to -0.80, and -0.80 to -1.20 MPa. These water-stress treatments will be referred to in subsequent discussions by the minimum value of the inferred soil matric potential range (-0.05, -0.40, -0.80, and -1.20 MPa). The relationship between soil water content and soil water matric potential was determined with a soil water characteristic curve for this soil. The soil water characteristic curve was obtained by systematically desorbing saturated samples of the soil at matric potential values from 0 to -1.47 MPa (14). The desired soil water status was maintained by appropriate daily water additions to each container determined by weighing the containers. Because of the coarse texture of this soil, a container depth of 9 cm, and a previously determined infiltration rate of approximately 64 mm hr^{-1} (Hill, *unpublished data*), water redistribution within each container was considered to occur at a rapid rate. To account for plant biomass, plants in four replicate pots of each grass type were washed, and mean fresh weight was calculated. This weight was included in the container weight. To reduce fluctuation in plant weight due to growth, plant heights were maintained at 6 cm.

Disease ratings and assessments. Plants were rated every 48 hr, beginning on day 17 after inoculation and ending on day 39, for disease incidence (percent of the pot area diseased), turf quality, and symptom type. Disease incidence was assessed visually with the aid of a template (circular areas representing a proportion of pot surface area). Turf quality was used as a measure of water stress and other factors affecting the appearance of the turf. The turf area showing distinctive symptoms was not included in this measure. The quality rating was based on a 0–9 scale, with 0 representing totally dead turf and 9 representing lush, green, unstressed turf. A turf quality rating of 7 was considered the lowest acceptable quality for a golf course turf, whereas a rating of 5 was considered the lowest acceptable quality for a home lawn. The symptom rating was used to give a measure of symptom expression or intensity within a symptomatic area. The symptom rating was based on a 1–5 scale, with 1 representing a craterlike patch with rotted, dead grass; 3 representing yellow to brown discolored turf, but with no distinct patch; and 5 representing no apparent disease symptoms.

Experimental design and analyses. Plants were subjected to four water-stress and two inoculum treatments in factorial combination in four experiments differing in the temperature of the growth chamber. A completely randomized design with water-stress inoculum treatments and four replicates was set up within each experiment (temperature). Individual pots represented the experimental units. The experiments were analyzed as 2 × 4 factorials combined over temperatures (17). Experiments were repeated at the same temperatures and inoculum conditions but with a wider range of water-stress conditions. Because results were similar, only results from the narrower range of water-stress conditions are reported here.

Disease incidence data were used to generate disease progress curves. Because there were differences in the shapes of these curves, the area under the disease progress curve (AUDPC) was used to compare the epidemic development among treatments. The AUDPC was calculated for each experimental unit by the equation described by Berger (6). To correct for homogeneity of variance, the AUDPC data were log transformed before an analysis of variance was performed (19). Turf quality and symptom data from days 17, 29, and 39 were selected for analysis since they represented early, mid, and late stages of disease development. Mean separations for significant factors or interactions having two or more degrees of freedom were calculated with a Bayes least significant difference multiple comparison test at $P \leq 0.05$ (19).

RESULTS

Disease development. All plants in the 35 C chamber died of supraoptimal temperature stress within 3 wk; therefore, no data

TABLE 1. Abbreviated analysis of variance table for the area under the disease progress curve for disease incidence (percent disease) combined over all temperatures

Source of variation	df	*F* value	*P*[a]
Temperature (Temp)[b]	2	8.54	0.0578
Rep[c]	3	0.46	0.7083
Grass[d]	2	28.81	0.0001 **
Temp × grass	4	3.20	0.0164 *
Stress[e]	3	16.14	0.0001 **
Temp × stress	6	4.92	0.0002 **
Grass × stress	6	0.72	0.6370
Temp × grass × stress	10	3.42	0.0012 **

[a] * = significance at $P = 0.05$; ** = significance at $P = 0.01$.
[b] Temperature levels were 20, 25, and 30 C.
[c] Replicates nested within temperature.
[d] Grass levels were Aspen and S-21 Kentucky bluegrass and annual bluegrass.
[e] Stress levels were inferred soil water matric potentials based on gravimetrically measured soil water contents that correspond to soil matric potential ranges with minima at -0.05, -0.40, -0.80, and -1.20 MPa.

for this temperature treatment are presented. No disease developed in pots receiving killed inoculum; therefore, these experimental units were not included in the disease development analysis. An examination of the F values (Table 1) revealed that the factor responsible for producing the greatest F-statistic for the variable AUDPC was the type of grass. In all cases, AUDPC was greater for annual bluegrass than the Kentucky bluegrasses, and the AUDPC generally was greater for S-21 than for Aspen (Table 2). The next greatest F value was water-stress level; however, since there was a significant temperature × grass × water-stress interaction, means were compared for each temperature-grass-stress combination. These interactions were attributed to both changes in rank and magnitude (Table 2).

Mean AUDPC at 20 C was greatest for annual bluegrass, with no significant difference between the two Kentucky bluegrass cultivars (Table 2). Disease development for annual bluegrass was greater at the mild stress level (−0.40 MPa) than at the unstressed (−0.05 MPa) and moderate stress (−0.80 MPa) levels. However, only at the severe stress level (−1.20 MPa) did significantly less disease occur than at the unstressed (−0.05 MPa) and the mild stress (−0.40 MPa) levels. Both Aspen and S-21 had significantly greater AUDPCs at the mild stress level (−0.40 MPa) than at the other stress levels. There were no significant differences in AUDPCs among the −0.05, −0.80, and −1.20 MPa stress levels for Aspen, and the −0.05 and −0.80 MPa stress levels for S-21. No disease was detected at the severe stress (−1.20 MPa) level for either Kentucky bluegrass cultivar.

Disease development was greater at 25 C than at 20 C, and disease was greatest at the highest water potential tested (−0.05 MPa) for all grasses (Table 2). Annual bluegrass had greater disease development than S-21 or Aspen. At the severe water stress (−1.20 MPa) level, annual bluegrass plants were unable to survive; therefore, no incidence data were collected for this treatment. S-21 showed a clear decrease in AUDPC with a decrease in soil water matric potential. The cultivar Aspen, however, exhibited an odd pattern of decreasing AUDPC between −0.80 and −1.20 MPa, but there was no injury at −0.40 MPa.

Disease incidence AUDPCs were greatest for all grasses at 30 C (Table 2). Annual bluegrass exhibited the highest disease incidence, with no significant difference in AUDPC among stress levels, except for the −1.20-MPa stress level where all plants died of drought stress. There also was no significant difference in AUDPC among stress levels in S-21. Aspen was not injured at the severe stress (−1.20 MPa) level at 30 C. Aspen exhibited greatest disease development between −0.05 and −0.80 MPa.

Symptom type. No disease was detected in any pots on day 17 at 20 C; however, disease was detected by day 29 (Table 3). There was no significant difference in symptom type among the grasses, with all expressing significantly most severe symptoms at mild water stress (−0.40 MPa). By day 39, symptom expression was most severe, but there were no significant differences in symptom type among the grasses. Symptom expression was significantly most severe in both Kentucky bluegrasses at −0.40 MPa, whereas it was significantly most severe in annual bluegrass at −0.05 MPa.

On day 17 at 25 C (Table 3), disease symptoms were evident in annual bluegrass, whereas none were yet detected in the Kentucky bluegrasses. Annual bluegrass disease expression was significantly most severe at the nonstress (−0.05 MPa) level. By day 29, disease symptoms were evident in all grasses and disease expression was significantly most severe at the highest soil water matric potential (−0.05 MPa). All grasses had no detectable symptoms at the most severe water stress (−1.2 MPa) level and exhibited a trend toward greater disease expression with an increase in soil matric potential. Symptoms on day 29 were most severe in annual bluegrass followed by S-21. Symptom severity of all grasses

TABLE 2. Mean area under the disease progress curve[a] for disease incidence of Aspen and S-21 Kentucky bluegrass and annual bluegrass (PA) grown at 20, 25, and 30 C and exposed to one of four inferred soil water matric potentials

Temperature (C)	Grass	Soil matric potential[b] (MPa) −0.05	−0.40	−0.80	−1.20
20	Aspen	0.98[c]	2.47	0.66	0.00
	S-21	0.57	2.36	1.34	0.00
	PA	2.37	2.55	1.64	1.20
25	Aspen	1.90	0.00	1.02	0.67
	S-21	2.77	2.08	1.96	0.51
	PA[d]	3.21	2.96	2.97	···
30	Aspen	2.83	2.07	2.69	0.00
	S-21	2.88	2.84	2.83	2.72
	PA[d]	3.22	3.26	3.26	···

[a] Log_{10}-transformed area under the disease progression curve means, based on four replicates, are presented.
[b] Soil water matric potentials are based on gravimetrically measured soil water contents that correspond to matric potential ranges with minima at −0.05, −0.40, −0.80, and −1.20 MPa.
[c] $LSD_{0.05}$ = 1.00 for pairwise comparison of means within a row.
[d] $LSD_{0.05}$ = 1.15 for pairwise comparison of means within a row.

TABLE 3. Mean symptom ratings[a] on three dates for Aspen and S-21 Kentucky bluegrass and annual bluegrass (PA) grown at 20, 25, and 30 C and exposed to one of four inferred soil water matric potentials

Time (Day)	Soil water matric potential[b] (MPa)	Symptom rating: Aspen 20 C	Aspen 25 C	Aspen 30 C	S-21 20 C	S-21 25 C	S-21 30 C	PA 20 C	PA 25 C	PA 30 C
17	−0.05	5.0	5.0	5.0	5.0	5.0	4.2	5.0	3.7	4.2
	−0.40	5.0	5.0	4.2	5.0	5.0	4.2	5.0	4.7	3.5
	−0.80	5.0	5.0	4.5	5.0	5.0	4.7	5.0	4.5	3.7
	−1.20	5.0	5.0	4.7	5.0	5.0	4.5	5.0	5.0	5.0
$LSD_{0.05}$[c]		0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.6
29	−0.05	4.7	3.7	2.0	5.0	2.2	2.7	4.0	2.0	1.0
	−0.40	3.0	5.0	3.0	2.7	3.5	2.7	3.0	2.7	1.0
	−0.80	4.7	5.0	3.0	4.0	4.5	2.2	3.7	4.2	1.2
	−1.20	5.0	5.0	5.0	5.0	5.0	3.0	5.0	5.0	5.0
$LSD_{0.05}$[c]		1.4	1.4	1.4	1.4	1.4	1.4	1.4	2.5	1.8
39	−0.05	3.5	2.7	1.0	4.2	1.0	1.0	1.7	1.0	1.0
	−0.40	1.7	5.0	2.0	1.7	2.0	1.0	2.0	1.0	···
	−0.80	4.2	4.5	1.0	3.2	1.0	1.0	3.2	1.0	···
	−1.20	5.0	4.0	5.0	4.0	4.7	1.0	3.7	···	···
$LSD_{0.05}$[c]		1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	3.0

[a] Rating of 1–5 of symptom severity where 1 = dead grass and 5 = no symptoms; means based on four replicates.
[b] Soil water matric potentials are based on gravimetrically measured soil water contents corresponding to matric potential ranges with minima at −0.05, −0.40, −0.80, and −1.20 MPa.
[c] Least significant difference value for pairwise comparison by day within a column.

had greatly increased by day 39. The severely water-stressed (−1.2 MPa) annual bluegrass had died, and symptom ratings among the remaining annual bluegrass soil matric potential treatments were equal. S-21 showed limited symptom expression at −1.2 MPa but exhibited severe symptoms at higher soil matric potentials. Aspen was significantly most severely injured at −0.05 MPa, with little or no symptom expression where plants were stressed.

On day 17 at 30 C, annual bluegrass at −0.40 and −0.80 MPa exhibited the most severe symptoms of all grasses (Table 3). Except at −1.20 MPa, annual bluegrass on day 29 exhibited more severe symptoms than the Kentucky bluegrasses. Annual bluegrass and Aspen exhibited no symptoms at −1.20 MPa, whereas S-21 exhibited no significant difference in symptoms among water-stress levels. By day 39, annual bluegrass plants at all but the lowest stress level (−0.05 MPa) had died. Except for Aspen maintained at −1.20 MPa, all grasses exhibited similar symptom ratings at all stress levels on day 39.

Turfgrass quality. In general, there was a decrease in turfgrass quality for the turf area not showing distinct disease symptoms with a decrease in the soil matric potential (Table 4). At cooler temperatures and high soil matric potentials, annual bluegrass exhibited higher quality; however, at higher temperatures and/or lower soil matric potentials, quality of the Kentucky bluegrasses was higher.

Although symptoms of disease were not yet visible at day 17 at 20 C (Table 2), a significant decrease in quality was exhibited in several of the Kentucky bluegrass treatments receiving live inoculum (Table 4). Aspen receiving live inoculum exhibited reduced quality when compared with treatments receiving killed inoculum at all water-stress levels. S-21 only exhibited reduced quality at the nonstress (−0.05 MPa) level. By day 29, overall quality of the Kentucky bluegrasses had declined, and Aspen treated with live inoculum and maintained at −0.05 and −0.40 MPa and S-21 treated with live inoculum and maintained at −0.05 continued to show a quality reduction. Aspen maintained at −0.80 MPa and S-21 at −1.20 MPa, both treated with live inoculum, also showed quality reduction. At day 39, only Aspen at −0.80 and −1.20 MPa and annual bluegrass at −1.20 MPa did not exhibit a significant inoculum-related reduction in turfgrass quality.

Overall, at 25 C, the inoculum-related decrease in turfgrass quality was more pronounced (Table 4). Only on day 17, all Aspen, S-21 moderately stressed (−0.80 MPa), and the annual bluegrass severely stressed (−1.20 MPa) treatments exhibited no inoculum-related decrease in quality. By day 29, all grass water stress treatment combinations, except S-21 moderate stress (−0.80 MPa) and the annual bluegrass severely stressed (−1.20 MPa) treatments, exhibited an inoculum-related decrease in quality. This same pattern was evident at day 39, where the difference in quality between those plants receiving live or killed inoculum became more pronounced.

The pattern at 30 C was nearly identical to that at 25 C, except that the overall decline in quality occurred earlier and was more dramatic (Table 4). At day 17, only Aspen unstressed and annual bluegrass severely stressed treatments did not exhibit decreased quality with live inoculum. At days 29 and 39, all grasses, except the severely stressed treatments of Aspen and annual bluegrass, exhibited a significant decline in turfgrass quality.

DISCUSSION

Summer patch generally was more severe at higher soil water contents. Comparison of the disease incidence AUDPCs (Table 2) and the symptom ratings (Table 3) at 25 and 30 C showed an increase in disease injury at higher water potentials. Except for annual bluegrass at 30 C, all grasses exhibited greatest disease development and most severe symptoms when unstressed (−0.05 MPa) at 25 and 30 C. Disease incidence AUDPC and symptom ratings indicated that injury was most severe on all grasses at the mild water-stress treatment (−0.40 MPa) at 20 C.

Kentucky and annual bluegrasses are cool season turfgrasses. Maximum sustained root growth for cool season grasses occurs between 10.0 and 18.5 C with optimal growth occurring near 16.0 C (5). As temperatures increase above the optimal range, roots mature more rapidly and become brown, spindly, and inactive (5). This increase in maturation may be followed by death (5). Youngner (24) found that the growth of the roots of Kentucky bluegrass was reduced at 21 C and may cease at 25 C.

Growth of Kentucky bluegrass remains unstressed if it is irrigated when soil water potentials drop to approximately −0.045

TABLE 4. Mean turfgrass quality ratings[a] on three dates for Aspen and S-21 Kentucky bluegrass and annual bluegrass (PA) grown at 20, 25, and 30 C and exposed to one of four inferred soil matric potentials

		Assessment date (day)											
		17				29				39			
Temperature	Grass × inoculum	−0.05	−0.40	−0.80	−1.20[b]	−0.05	−0.40	−0.80	−1.20	−0.05	−0.40	−0.80	−1.20
20 C	Aspen I[c]	8.2	7.5	5.8	5.0	7.8	6.2	5.5	5.0	7.5	5.8	5.0	5.0
	Aspen KI	8.8	8.0	6.0	5.5	8.5	8.2	6.2	5.2	8.2	8.0	5.2	5.2
	S-21 I	8.0	7.5	7.0	5.2	8.0	7.2	7.0	5.5	7.5	6.8	6.5	4.5
	S-21 KI	8.8	7.5	7.0	5.5	8.5	7.5	7.2	6.8	8.5	7.8	7.0	6.2
	PA I	9.0	9.0	8.0	5.8	8.8	8.8	7.8	5.2	8.5	8.5	7.8	5.2
	PA KI	9.0	9.0	7.8	6.0	9.0	9.0	8.0	5.2	9.0	9.0	8.2	5.5
25 C	Aspen I	8.8	7.8	6.5	3.5	7.5	6.8	4.8	3.0	4.8	5.8	4.0	2.0
	Aspen KI	9.0	8.0	6.2	3.5	9.0	8.2	6.5	3.8	9.0	7.8	6.5	3.2
	S-21 I	8.2	7.0	6.0	4.0	4.8	4.8	5.8	3.2	1.5	2.8	4.0	2.8
	S-21 KI	8.8	7.5	5.8	5.2	9.0	7.5	5.2	4.8	9.0	7.5	5.0	4.2
	PA I	7.2	7.2	2.5	0.2	2.0	5.5	1.0	0.0	0.2	1.8	0.2	0.0
	PA KI	7.8	6.2	3.2	0.5	8.2	7.8	1.8	0.2	8.0	7.2	1.2	0.0
30 C	Aspen I	8.8	6.0	4.5	2.5	4.0	4.5	3.8	1.2	1.0	2.2	1.8	1.0
	Aspen KI	8.8	7.0	5.2	2.5	8.8	8.0	4.8	1.8	8.2	8.0	5.2	1.5
	S-21 I	7.5	7.2	5.5	3.2	3.0	4.8	3.8	2.0	1.0	1.8	2.0	1.0
	S-21 KI	8.8	8.5	6.5	4.8	8.8	7.8	6.2	3.0	7.8	7.8	5.8	2.8
	PA I	5.8	3.8	2.8	0.5	0.8	0.5	0.5	0.2	0.0	0.0	0.2	0.0
	PA KI	9.0	6.8	4.2	0.2	7.7	5.0	4.2	0.0	6.7	3.2	3.2	0.0
$LSD_{0.05}$[d]		0.2				0.3				0.4			

[a] Rating of 0–9 of turf quality where 0 = dead turf and 9 = green, nonstressed turf; means were based on four replicates.
[b] Soil water matric potentials are based on gravimetrically measured soil water contents corresponding to matric potential ranges with minima at −0.05, −0.40, −0.80, and −1.20 MPa.
[c] I denotes live inoculum and KI denotes killed inoculum.
[d] Least significant difference value to compare live versus killed inoculum treatments within a grass and assessment date.

MPa and is considered to be under water stress at −0.40 MPa (1). Unstressed plants possess active defense mechanisms that enable them to resist potential pathogens (12). Schoenweiss (20) noted that most pathogens that incite severe disease in water-stressed plants grow poorly or are inhibited in unstressed plants. These pathogens may survive and colonize unstressed host tissue, but only to a limited extent and usually without visible damage. However, when the host is exposed to water stress, plant resistance mechanisms are impaired, and the pathogen moves through the host tissues inciting disease. These same observations may be true for plants exposed to heat stress.

Landschoot (15) determined that *M. poae* colonizes and infects roots in a manner similar to other ectotrophic species of *Phialophora* and *Gaeumannnomyces*. Garrett (12) noted that the ectotrophic habit of certain root pathogens is the result of active defense mechanisms of vigorously growing roots. The essential features of the ectotrophic habit are well illustrated by *Gaeumannomyces graminis* (Sacc.) Arx & Olivier var. *tritici*, the causal agent of wheat take-all (12). This ectotroph grows superficially over roots toward the root tip from the point of inoculation, occasionally penetrating several cells deep in the cortex. This habit is postulated by Garrett (12) to provide a mechanism whereby the parasite can initiate a series of penetrations in rapid succession along the length of the root. These successive penetrations may result in a synergism that can overcome the active defense of roots and allow the pathogen to move into the vascular tissue. Environmental stress enhances the colonization of the vascular system by many ectotrophic pathogens (12). Ayres (2) suggested that drought or any abiotic stress may modify active host responses in two basic ways. First, stress may alter those activities of the plant directed toward resisting the growth or injurious products of the parasite. Second, stresses arising from coincident drought and pathogen-induced stress may be additive. This additive effect may be sufficient to cause injury where none existed before, or to exacerbate existing injury and impair the chances of recovery.

We showed in a companion study that the optimal water potential for the growth of *M. poae* in vitro is a function of temperature (13). Isolates of *M. poae* used in the in vitro study produced maximal growth at the highest osmotic potentials tested (−0.12 to −0.35 MPa) at 25 and 30 C. At 20 C, slight but insignificant shifts in optimal growth were seen between −0.35 and −1.03 MPa. At 35 C, growth was reduced, with a peak between −1.03 and −1.47 MPa.

In this study, the 20 C treatment was below the optimal range for growth of the pathogen but was within an acceptable range for growth of grass roots. Disease development was greatly reduced at this temperature compared with the 25 and 30 C treatments. These results are consistent with the field temperature observations of Smiley et al (23) and with the known in vitro growth response of *M. poae* to temperature (13,15,22). We also observed a peak in disease development at the mild stress (−0.40 MPa) level at 20 C. At the unstressed (>−0.05 MPa) level, active plant defense mechanisms probably were still functional, resulting in less disease. However, because Kentucky bluegrass is considered to be under stress at −0.40 MPa, active defense mechanisms may have been impaired, whereas *M. poae* was capable of maximal growth at this water potential and temperature, resulting in increased disease development. As soil water matric potential became more negative, growth of the pathogen probably was reduced and the pathogen would likely have lost its competitive advantage over the host.

At temperatures of 25 and 30 C, which are within or near the optimum for growth of *M. poae*, disease development increased and was approximately proportional to the in vitro growth of the pathogen (13). At these temperatures, active defense mechanisms of the host plant were probably functioning poorly, if at all. With no barriers to limit root colonization by the pathogen, the development of disease was probably a function of those factors affecting growth of the pathogen. As conditions became drier, growth of the pathogen was restricted and disease development was reduced. Many root diseases are favored by wet soils, with the simplest explanation being that as the water potential of the soil drops, the growth of the pathogen is restricted (7).

Drought does not seem to play a key role in summer patch development. However, there was a marked decrease in overall turfgrass quality in pots receiving live inoculum when compared with pots subjected to the same environmental parameters receiving killed inoculum (Table 4). The decrease in quality appeared as drought stress increased and often was evident before disease symptoms were visually expressed. Both root infection and low soil water potentials result in plant water deficits. The initial pathogen-induced water deficits are not manifested as typical summer patch symptoms. *M. poae* may colonize roots and crowns of turfgrasses at moderate soil temperatures and can grow up to 18 cm over 1.5 yr in mature field-grown sod without causing visible disease symptoms (15). Smiley (22) suggested that plants previously colonized by the pathogen may suffer from levels of vascular dysfunction that are sublethal at higher soil water potentials and which may contribute to a lethal condition when additional stresses are imposed. It also is possible that the decline in quality of infected plants before symptom expression may be mistaken for drought injury in the field, so that when classic symptoms appear they could be erroneously interpreted to be associated with low, soil water potentials. It should also be noted, however, that inoculated plants in our study were maintained at a relatively constant temperature and water-stress level. It, therefore, is conceivable that diurnal fluctuations in temperature and water stress in the field also may affect the symptomotology of summer patch.

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