Role of Drought Stress in the Development of Summer Patch in Field-Inoculated Kentucky Bluegrass

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ABSTRACT

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Field plots of Kentucky bluegrass (*Poa pratensis*) at two sites were treated with either live or killed inoculum of *Magnaporthe poae* (isolate ATCC 60239) and subjected to either a non-drought-stress (>−0.05 MPa) or drought-stress (<−0.05 MPa) treatment. Studies at site I were conducted on 1- and 2- yr-old stands of either the cultivar Aspen (resistant) or S-21 (susceptible). At site II, studies were conducted for one season on a 6-yr-old blend seeded as equal parts of Merion, Vantage, and Sydsport. Disease developed in the first year at both sites only in those plots receiving live inoculum. Disease was more severe in non-drought-stressed plots. There was no significant difference in disease development between cultivars at site I. In the second year at site I, disease developed where both live and killed inoculum had been placed. There was no significant difference in disease severity between stress treatments; however, there was consistently more disease in non-drought-stressed plots. Aspen was injured less than S-21 in the second year. Summer patch was more severe when soil water potentials were high, and drought stress was not a key predisposing factor in the development of this disease.

Summer patch, caused by *Magnaporthe poae* Landschoot & Jackson (16), is one of the most destructive diseases of Kentucky bluegrass (*Poa pratensis* L.) in the United States (10). Symptoms of summer patch in Kentucky bluegrass become evident in Maryland during late June to early July with the appearance of small bleached areas of turf 2.5–5.0 cm in diameter (10). Patches may increase in size to 30 cm in diameter or greater, and are sometimes sunken, forming craterlike depressions. The patches may coalesce, resulting in large areas of blighted turf. A frog-eye patch symptom frequently is reported in more northern regions of the United States, but is only occasionally seen in Maryland (10). There are no distinctive foliar lesions; however, the affected plants show signs of crown and root rot. Dark brown, ectotrophic, runner hyphae and hyphopodia can also be seen on roots.

Summer patch is one of two or more diseases recently segregated from a complex once known as Fusarium blight or Fusarium blight syndrome. In 1984, Smiley and Craven Fowler (21) identified the new disease summer patch and reported that summer patch occurred under the same cultural and environmental conditions as Fusarium blight syndrome (21). Fusarium blight has been reported as a disease associated with the following environmental factors: high light intensity, high soil and air temperatures, and high relative humidity (2,3,6,9,13,17,19). The majority of investigators reporting on the role of water stress in the development of Fusarium blight have suggested that drought stress increases disease severity (2,3,6,9,11,17). However, Smiley and Craven (20) were unable to detect soil water potentials <−0.1 MPa before an outbreak of Fusarium blight in New York. In fact, Smiley (19) found that major occurrences of the disease were associated with periods of abundant moisture or with alternating periods of wetness and drought. Unfortunately, the few studies reporting environmental influences on disease development were conducted on natural infections that are now considered to be of uncertain etiology.

With the establishment of *M. poae* (15,21) as the causal agent of summer patch, an assessment of the influences of environmental conditions on the development of this disease in the field is justified. This study is one of three companion studies to investigate the role of drought stress in the development of summer

patch. In the first of these studies, we (13) assessed in vitro growth of *M. poae* at various water potentials and temperatures. In the second companion study, (14) the effects of various water-potential-temperature treatments on the development of summer patch in Kentucky bluegrass and annual bluegrass (*Poa annua* L.) in growth chambers were investigated. This, the third study, was undertaken to investigate the influence of drought stress under the fluctuating conditions encountered in the field. The objectives of the study reported herein were to study summer patch symptom development in the field with an isolate of *M. poae* and to investigate the role of drought stress on development of summer patch in Kentucky bluegrass.

MATERIALS AND METHODS

Field experiments were conducted on two sites at The University of Maryland Turfgrass Research and Education Center in Silver Spring. Data were collected over two seasons (1987 and 1988) at site I and one season (1988) at site II.

Cultural practices—site I. Plots were established in the fall of 1986 on a Sassafras sandy loam (fine-loamy, siliceous, mesic Typic Hapludult), having a pH 6.5 and 1.9% organic matter. This area was fallow crop land colonized by assorted annual and perennial weeds and had no previous history of turfgrass cultivation. Soil was prepared for seeding by tilling to a 20-cm depth, hand raking to remove rocks and plant debris, and dragging with a heavy chain mat. A 16-8-8 N-P-K fertilizer was applied at 49 kg of N ha^{-1}, and the entire area then was hand-graded and seeded on 22 September 1986. The Kentucky bluegrass cultivars Aspen and S-21 were selected because Aspen had shown good resistance and S-21 had shown high susceptibility to natural infections of summer patch in University of Maryland turfgrass cultivar evaluations. Turfgrasses were maintained at a 6.0- to 7.0-cm height and irrigated as needed to prevent drought stress until water-stress treatments were imposed in May of 1987. These plots received three more applications of 49 kg of N ha^{-1} from urea in October, November, and April. The herbicide siduron was applied preemergence on 14 April and 16 July 1987 to control crabgrass (*Digitaria* sp.) and other annual grasses, and a herbicide containing 2,4-D + mecoprop + dicamba was applied on 18 May to control broadleaf weeds. Fenoxaprop was applied on 10 July 1987 as a spot treatment for crabgrass seedlings that had escaped control.

In September of 1987, renovation of the plots at site I was needed. On 22 September, all plots were sprayed with diclofop for selective control of perennial ryegrass (*Lolium perenne* L.), which was encroaching into the plots. On 5 October 1987, all plots were verticut to a 6- to 7-mm depth in two directions and the debris was removed. Plots then were overseeded with the same cultivar that was originally established and fertilized with 49 kg of N ha^{-1} from a 16-8-8 fertilizer. Plots were firmed with a roller and lightly irrigated until seed germination. This site received two more applications of 49 kg of N ha^{-1} from urea in November of 1987 and May of 1988. In 1988, plots were routinely maintained as described above.

Cultural practices—site II. This study was initiated in May of 1988 on a mature Kentucky bluegrass blend (Merion + Vantage + Sydsport), with no previous history of severe summer patch injury. This area was seeded in September of 1981 on a Chillum silt loam (fine-silty, mixed, mesic Typic Hapludult), having a pH of 7.3 and 1.7% organic matter. Site preparation procedures were the same as those for site I. For the 2 yr before, and during the year of the study, this area received 150 kg of N ha^{-1} yr^{-1} and an annual spring application of pendimethalin for control of annual grass weeds, and an early summer application of 2,4-D + mecoprop + dicamba for control of broadleaf weeds. Turf was maintained at a 6.0- to 7.0-cm mowing height during the test period.

Experimental design—site I. Plots were arranged in a split-plot design with four replicate blocks. The two main plot treatments were either drought stressed or nondrought stressed, and the subplots consisted of two Kentucky bluegrass cultivars and two inoculum levels (live or heat killed) in factorial combination. The subplot size was 3 m^2.

Experimental design—site II. This site was established as a randomized complete block design with four replicate blocks and a factorial set of treatments. The factors and levels were as follows: two water-stress levels (drought stressed or nondrought stressed), and two inoculum levels (live or autoclaved). Drought-stressed and non-drought-stressed treatments and plot size were the same as those at site I.

Water-stress treatments and monitoring. Water-stress treatments were imposed immediately following inoculation and ceased in late September of each year. Drought-stressed plots were irrigated only at the onset of visual symptoms of drought to prevent dormancy. Non-drought-stressed plots were irrigated as needed to maintain soil water matric potentials at levels greater than −0.05 MPa. In both instances, sufficient water was applied during irrigation to saturate the soil to a depth of 30 cm or more. In 1987, soil water potential was determined only in the non-drought-stressed plots with a fast-response type tensiometer (2900 series, Soilmoisture Equipment Corp., Santa Barbara, CA). When the water potential of any subplot in the non-drought-stressed main plot fell to −0.05 MPa, the entire main plot area was irrigated. In 1988, irrigation practices remained the same as in 1987; however, soil water potential was determined by gravimetric methods in all plots. Five soil cores (9-cm length by 1-cm diameter) per plot (approximately 20 g of soil) were placed in a glass vial and weighed. These samples were microwaved on full power for 20 min to dry the sample and then reweighed to determine the soil water content (12). The relationship between soil water content and soil water matric potential from 0.0 to −1.47 MPa was determined for each site with undisturbed soil cores as described previously (14). Site I stressed plots were irrigated five times during 5 May through 3 September 1987 and four times during 6 June through 14 August 1988, whereas nonstressed plots were irrigated 17 times in 1987 and 15 times in 1988. Site II stressed plots were irrigated three times and nonstressed plots 10 times during 7 June through 15 July 1988. In 1988, site I soil temperatures were monitored and recorded periodically from 23 June until 15 September by electronic datapods (Datapod Model 222, Omnidata Inc., Logan, UT).

Fungal isolate and inoculation. *M. poae* (reported as *Phialophora graminicola* (Deacon) Walker; isolate ATCC 60239) was obtained from the American Type Culture Collection in Rockville, MD. This is one of Smiley and Craven Fowler's (21) original summer patch isolates from Kentucky bluegrass roots. Live and heat-killed inoculum was prepared by the method described previously (14).

Turf at site I was inoculated on 22 May 1987, and both sites were inoculated during the second year on 24 May 1988. Grasses were inoculated by removing a plug of turf (60-mm-diameter and 60-mm-depth) and placing 150 ml of the appropriate inoculum in the hole and replacing the plug. Each subplot at site I in 1987 and each plot at site II in 1988 received inoculum at each corner and in the center for a total of five separate points of inoculation. In the second year at site I, each subplot was inoculated at an additional four points located midway between each corner and the center. Immediately following inoculation, plugs were tamped

TABLE 1. Mean area under the disease progress curve (AUDPC) for S-21 and Aspen Kentucky bluegrass at site I, 1987

Irrigation treatment[a]	AUDPC[b]	
	S-21	Aspen
Stressed	1166[c]	1264
Nonstressed	1561	1441

[a] Stressed plots were irrigated only at the onset of visual symptoms of drought; nonstressed plots were irrigated to maintain soil water matric potentials ≥−0.05 MPa.

[b] Mean AUDPC of patch diameters based on 13 assessments of eight replicate plots, 7 July through 9 September.

[c] $LSD_{0.05}$ = 143 used for pairwise comparison both across and within columns.

into place, and the plots were irrigated lightly to help knit the plugs back into the ground. On the night of 24 May 1988, groundhogs (*Marmota monax*) dug up inoculum at the newly inoculated points in site I, eating and spreading some of the inoculum over the plots.

Data collection and analyses. In 1987 at site I and in 1988 at site II, patch diameters were measured in two perpendicular directions every 3 to 5 days. In 1988 at site I, disease had become so severe it was necessary to visually estimate percent of the plot area diseased, since original patches could no longer be discerned. Area under the disease progress curve (AUDPC) was calculated as described by Berger (5) for each plot. All data were subjected to an analysis of variance. Mean separation for significant factors or interactions having two or more degrees of freedom were calculated with a Bayes least significant difference multiple comparison test at $P = 0.05$ (18).

RESULTS

Disease development. During the first year at each site (1987 at site I and 1988 at site II), summer patch developed only in those plots receiving live inoculum and only at the exact points of inoculation. Disease was confirmed by the successful reisolation and identification of *M. poae* by induction of perithecia. Symptoms were noted first on 1 July 1987 and 20 July 1988 at site I and on 12 August 1988 at site II. Disease was present at site I in plots receiving both live and heat-killed inoculum in 1988, and not just at points of inoculation. The presence of the pathogen was again confirmed by reisolation from both live and heat-killed inoculation points and induction of perithecia. The appearance of disease in uninoculated plots may have been due to natural infection, spread of inoculum during renovation the previous autumn, or groundhogs.

Environmental monitoring. At both sites in 1988 the volumetric soil water contents in the non-drought-stressed plots were maintained so that soil conditions would be characterized by matric potentials of ≥ -0.05 MPa at all times. Soil water potentials ≥ -0.05 MPa are considered nonstressed for the growth of Kentucky bluegrass (1). In late May through mid-July, soil matric potentials in the drought-stressed plots often fell to less than -1.5 MPa before irrigation, which is outside the range of plant-available water. However, in the event of rain, drought-stressed plots received water before irrigation would normally have been applied. Rain was particularly frequent from late July to early September 1988. Drought-stressed plots were temporarily covered with plastic during periods of frequent rain, but this often was not effective in preventing the soil from becoming wet from surface runoff and blowing rain. During this rainy period, drought-stressed plots usually were kept drier than the nonstressed plots (-0.05 MPa), but generally dried to no more than -0.40 MPa before they were wet again by rain.

Soil temperatures at the 6-cm depth generally remained between 20–30 C during the period monitored (i.e., 23 June to 15 September 1988). The lowest temperature monitored was 16 C on the night of 1 July, and the highest was 35 C during the day on both 19 and 30 July. There was a malfunction in data recording equipment from 19 to 30 July. Based on temperature trends both before and after 19 and 30 July and climatological data from nearby College Park, however, it appears that the period of highest soil temperatures was from mid-July through the first week of August. During this period, soil temperatures were estimated to be in the 25–35 C range.

Treatment effects—site I. According to an analysis of variance for the area under the disease progress curve for the first season (1987), there was more ($P = 0.031$) disease in the non-drought-stressed plots than in the drought-stressed plots (Table 1). A significant ($P = 0.034$) stress × cultivar interaction also occurred. This interaction was attributed to a change in rank of the cultivars at the two stress levels.

The majority of variation in 1988 was attributed to inoculum and cultivar treatment differences (Table 2). Plots that received live inoculum had a much greater amount of disease than those receiving only killed inoculum ($P = 0.0001$), and S-21 was injured more ($P = 0.0001$) by disease than Aspen. Cultivar × inoculum and stress × inoculum interactions also were significant at $P = 0.011$ and $P = 0.046$, respectively. These interactions were due to a difference in magnitude, not to a change in rank. There was consistently more disease in non-drought-stressed plots.

Treatment effects—site II. There was greater ($P = 0.002$) disease development in non-drought-stressed plots (AUDPC = 385) than in drought-stressed plots (AUDPC = 245) at this site. These results are similar to those for site I during its first year (i.e., 1987) of inoculation.

DISCUSSION

We have demonstrated the feasiblility of inoculating field plots of turfgrass to induce summer patch with a known isolate of *M. poae*. Patch symptoms developed at both sites during the first season after inoculation. Symptoms first appeared as small, circular patches of brown, blighted turf. These patches increased in diameter and most formed sunken craterlike depressions in the turf. Patch diameters at site I reached up to 36 cm in diameter in stressed plots and 40 cm in nonstressed plots in 1987. In the summer of 1988, large blighted areas were formed at site I, and individual patches were no longer distinguishable. At site II (1988), patch diameters reached 12 cm in stressed plots and 19 cm in nonstressed plots. This field-inoculation technique was very useful for studying summer patch in Kentucky bluegrass. Smiley et al (21–23) and Craven Fowler et al (7,8) also have been successful in inducing summer patch and necrotic ring spot (caused by *Leptospharia korrae* Walker & Smith) in the field.

The development of summer patch in our study generally was more severe in situations where soil moisture was not limiting, which agrees with earlier findings from growth chamber studies (14). During the first summer following inoculation at each site, the imposed drought-stress treatment was the single, most important variable and the factor that most influenced disease development. In the second season at site I, there also was more disease in the nonstressed plots, but other factors played a greater

TABLE 2. Mean area under the disease progress curve (AUDPC) for S-21 and Aspen Kentucky bluegrass at site I, 1988

	AUDPC[b]					
	Live inoculum			Killed inoculum		
Irrigation treatment[a]	S-21	Aspen	Mean (IXS)[c]	S-21	Aspen	Mean (IXS)
Stressed	1959	629	1294	629	84	388
Nonstressed	3617	1269	2443	743	328	536
Mean (CVXI)[d]	2788	949		717	206	

[a] Stressed plots were irrigated only at the onset of visual symptoms of drought; nonstressed plots were irrigated to maintain soil water matric potentials ≥ -0.05 MPa.

[b] Mean AUDPC of percent plot area diseased based on 11 assessments of four replicate plots, 21 July through 18 September.

[c] Mean AUDPC for inoculum × stress interaction averaged over cultivar. Bayes $LSD_{0.05} = 726$ for pairwise comparison of inoculum × stress means.

[d] Mean AUDPC for cultivar × inoculum interaction averaged over stress. Bayes $LSD_{0.05} = 678$ for pairwise comparison of cultivar × inoculum means.

role. There was no significant difference between Aspen and S-21 at site I during the first year. By the second year, however, these cultivars did exhibit significantly different disease severity, with Aspen being less severely diseased than S-21. It is likely that the inoculum loads present at the focus of inoculation may have overwhelmed resistance mechanisms in the cultivar Aspen; however, by the second year, as the inoculum spread from the focus of inoculation, it may have become less concentrated and put less pressure on the resistance mechanisms of this cultivar.

Another factor that probably played a key role in the differences observed between the two seasons at site I was the difference in macroclimate. Weather conditions in June of 1987 were more favorable for the growth of *M. poae*. In June of 1987, which was consistently warmer than June of 1988 (mean monthly temperatures of 25 and 23 C, respectively), there was only 1 day when the maximal temperature was less than 25 C and only 1 day when the minimal temperature was less than 10 C. There were 6 days with a temperature maximum less than 25 C and 4 days with a minimum of less than 10 C in June of 1988. The greatly reduced rainfall in June of 1988 (1.5 versus 10.8 cm in June of 1987) should not have played a role, since irrigation controlled soil moisture levels. However, the late summer rainfall in 1988 (8.7 and 14.4 cm for July and August versus 4.8 and 4.1 cm, respectively, in 1987) reduced the difference in soil moisture levels between irrigation treatments. This reduction of the soil moisture differential between the drought- and non-drought-stress treatments may have contributed to the variability in disease levels at site I in 1988.

From these results, and those of the two companion studies (13,14), we conclude that temperature and water potential, but not severe drought stress, play a role in the development of summer patch. Smiley et al (21,24,25) and Landschoot (15) have shown that high temperature (>25 C) is an important predisposing factor for disease development. Temperatures in excess of 25 C severely impair root growth (4,26), yet favor growth of the pathogen (13). Once roots are predisposed to disease by high temperatures, water potential probably becomes important by affecting growth of the pathogen. High water potentials (e.g., moist soil conditions) are more favorable for pathogen growth (13,14,24), and, hence, for colonization and disease development. The warmer conditions present in June of 1987 were more favorable for the growth of *M. poae* and probably were responsible for the earlier development of disease symptoms in 1987. Further, symptoms of summer patch, particularly in plots intended to be drought stressed, did not develop in 1988 until hot, wet weather occurred.

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