TURFGRASS TRENDS

DISEASE MANAGEMENT

Research Defines Dead Spot More Clearly

By J.E. Kaminski and P.H. Dernoeden

In 1998, researchers at the University of Maryland discovered a new disease of creeping bentgrass caused by an unidentified species of *Ophiosphaerella*. Through morphological and molecular study, it was shown that the pathogen constituted a new species, *Ophiosphaerella agrostis*, and the disease was named bentgrass dead spot. Subsequently, *O. agrostis* was found in Texas and Florida causing dead spots in hybrid bermudagrass greens. The disease is now referred to as dead spot.

In creeping bentgrass grown on putting greens, dead spot appears initially as small, dime-sized spots that may increase up to 3 inches to 4 inches in diameter. During early stages of disease development, the spots are reddish-brown and often are confused with other turfgrass diseases such as dollar spot, copper spot and microdochium patch. Spots also may be mistaken for damage from black cutworms or ballmarks, which commonly are found on putting greens.

Among commercial creeping bentgrass cultivars, dead spot generally was most severe in L-93, Penn A-1, A-4, G-1, G-6, Imperial and Providence.

In the later stages of dead spot development, grass in the center of the spots becomes tan, while leaves in the outer edge appear reddish-brown. Patches may be distributed throughout the putting green or localized, and generally do not coalesce. Spots often form depressions or pits and may severely reduce the quality and playability of the putting surface.

Active dead spot infection centers generally appear in areas with full sun and good air circulation. Initially, *O. agrostis* infection centers occur predominantly along ridges, on mounds and south-facing slopes of greens. These areas generally are associated with higher soil temperatures and often are the first to exhibit drought symptoms. The aforementioned conditions generally result in higher levels of plant stress and may reduce the defense capabilities of bentgrass plants.

Dead spot only has been found on newly constructed greens or on older greens that were fumigated with methyl bromide. The disease generally develops within one to two years of establishment, but outbreaks have been observed in creeping bentgrass greens less than 1 year old and as old as 6.

With few exceptions, dead spot is most severe during the first year of symptom

Continued on page 60

IN THIS ISSUE

OUR SPONSORS

Continued from page 57

expression. The disease then enters a decline phase which may last from one to three years. All newly constructed greens affected by dead spot had sand as the primary soil medium. Occasionally, dead spot was found on sand-based bentgrass collars and tees, indicating that O. agrostis can attack creeping bentgrass maintained at higher mowing heights. Dead spot, however, has not been found on fairways or other sites where bentgrass turf was grown on native soil.

Biological aspects

On golf courses in the mid-Atlantic region, dead spot symptoms may appear as early as May, but disease activity generally is most severe between July and August.

In a growth-chamber study, winter-dormant creeping bentgrass field samples showing symptoms of dead spot were incubated at temperatures ranging from 59 degrees Fahrenheit to 86 degrees Fahrenheit. After 12 days to 28 days of incubation, disease reactivation occurred at temperatures more than or equal to 68 degrees Fahrenheit, but dead-spot severity was greatest at temperatures between 77 degrees Fahrenheit and 86 degrees Fahrenheit. Similarly, in-vitro studies revealed that the optimum temperatures for mycelia growth of

Continued on page 62

TABLE 1

Bentgrass dead-spot infection centers for 20 field-grown *Agrostis spp.* selections, College Park, Md., between 2000 and 2002.*

Cultivar	Bentgrass species	Infection centers per plot[x]					
		2000		2001		2002[y]	
		6 Sept	29 Nov	15 May	16 Aug	18 July	16 Aug
ABT-CRB-1	creeping	27a-d[z]	11b-e	9bcd	3ab	6abc	22abc
Backspin	creeping	18cde	6fgh	6cde	2bcd	2ef	12c-f
BAR AS 8US3	creeping	21b-e	7d-h	7bcd	2bcd	5a-f	18a-d
BAR CB 8FUS2	creeping	22b-e	10b-e	11ab	2bcd	3c-f	12c-f
Bardot	colonial	32ab	6e-h	4ef	1cd	2def	9def
Bavaria	velvet	8f	4h	2f	0d	2def	4f
Century	creeping	27a-d	14bc	11ab	3ab	6abc	22abc
Crenshaw	creeping	17def	5gh	6de	1bcd	2ef	6ef
Imperial	creeping	33ab	9c-g	8bcd	1cd	4b-f	15b-e
L-93	creeping	37a	11bcd	10abc	2abc	8ab	26a
Penn A-1	creeping	33ab	15bc	11ab	3ab	9a	25ab
Penn A-2	creeping	23b-e	8d-h	8bcd	2bcd	5a-f	17a-d
Penn A-4	creeping	29a-d	15b	12ab	3abc	5a-e	15b-e
Penn G-1	creeping	25a-e	10b-f	8bcd	2abc	3c-f	10def
Penn G-6	creeping	29abc	8d-h	9bcd	1bcd	4b-f	23ab
Penncross	creeping	14ef	6e-h	7bcd	1cd	2ef	7ef
Pennlinks	creeping	17def	7d-h	5de	2bcd	1f	5ef
Providence	creeping	24a-e	11b-e	11ab	3abc	4c-f	9def
SR1119	creeping	22b-e	10b-f	11ab	2bcd	3c-f	18a-d
SR7200	velvet	32ab	24a	14a	5a	6a-d	12c-f

*Numbers that have the same letters next to them are statistically the same.

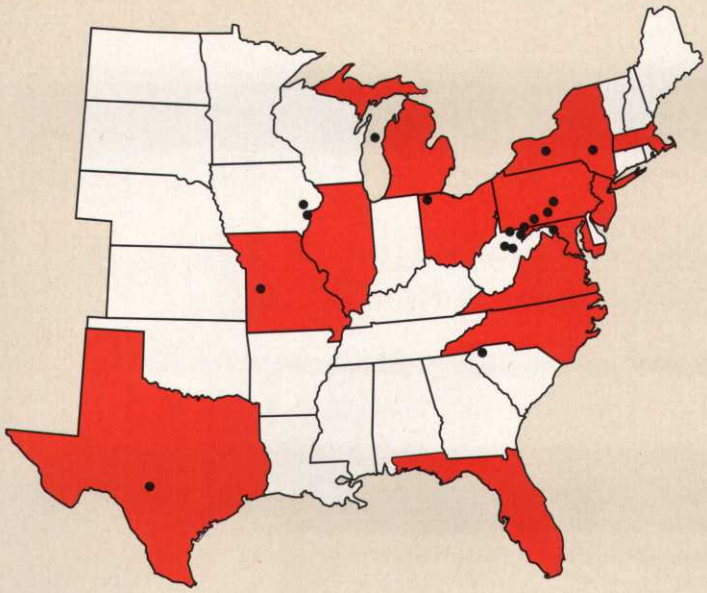
[x] Data were transformed (y+.5), but pre-transformed means are shown.

[y] Bentgrass dead spot fully recovered in the autumn of 2001 and data from 2002 represent new infection centers.

[z] Means in a column followed by the same letter are not significantly different (P £ .05) based on the protected least significant difference multiple mean comparison test.

FIGURE 1

Locations of creeping bentgrass and bermudagrass confirmed to be infected by *Ophiosphaerella agrostis* between 1998 and 2003.



Continued from page 60

O. agrostis ranged from 77 degrees Fahrenheit and 86 degrees Fahrenheit, and growth was suppressed at 95 degrees Fahrenheit.

A major obstacle of dead spot management is due to the rapid development of secondary inoculum. Unlike other *Ophiosphaerella* species associated with disease of turfgrass (i.e., *O. herpotricha*, *O. narmari*, and *O. korrae*), sexual fruiting bodies are commonly found embedded in necrotic tissue in the center of dead spots. Within each fruiting body, hundreds of spores are produced. When mature, ascospores are forcefully discharged and spread through wind currents or ooze out in the presence of water.

In growth-chamber studies, fruiting bodies developed on a tall-fescue seed/wheat bran mix at temperatures ranging from 55 degrees Fahrenheit to 82 degrees Fahrenheit. Under constant light, fruiting bodies development occurs rapidly, and viable spores may be present within one week.

At 77 degrees Fahrenheit, mature ascospores can germinate in as little as two hours. Germination during the first several hours of spore germination was enhanced by both light and the presence of bentgrass leaves or roots. During the early morning hours, naturally developing dew and guttation may create optimum conditions for spore germination and infection. Reducing leaf wetness through whipping or mowing may be important in reducing new infection.

Field studies on dead spot

Bentgrass cultivar susceptibility to *O. agrostis* was assessed on a USGA-specified research green between 2000 and 2002 at the University of Maryland Turfgrass Research Facility in College Park, Md., 20 *Agrostis spp.* (listed in Table 1 and Figure 2), including 17 cultivars and experimental selections of creeping bentgrass, two cultivars of velvet bentgrass and Bardot colonial bentgrass, were seeded in September 1999.

The area was maintained as a putting green and subjected to routine cultural practices (fertilization, vertical mowing, aeration and topdressing). To ensure dead spot infection, the area was inoculated with *O. agrostis* in 2000 and 2002.

In the first year of this cultivar study, disease symptoms were first observed in June, and the disease's severity increased throughout the summer. All common Agrostis species and cultivars grown on golf courses including creeping, colonial and velvet bentgrasses were susceptible to infection by *O. agrostis*.

Among the commercially available creeping bentgrass cultivars, dead spot generally was most severe in L-93, Penn A-1, A-4, G-1, G-6, Imperial and Providence. Creeping bentgrass cultivars Pennlinks, Penncross and Crenshaw as well as Bavaria velvet bentgrass generally were the least susceptible cultivars over the course of the study.

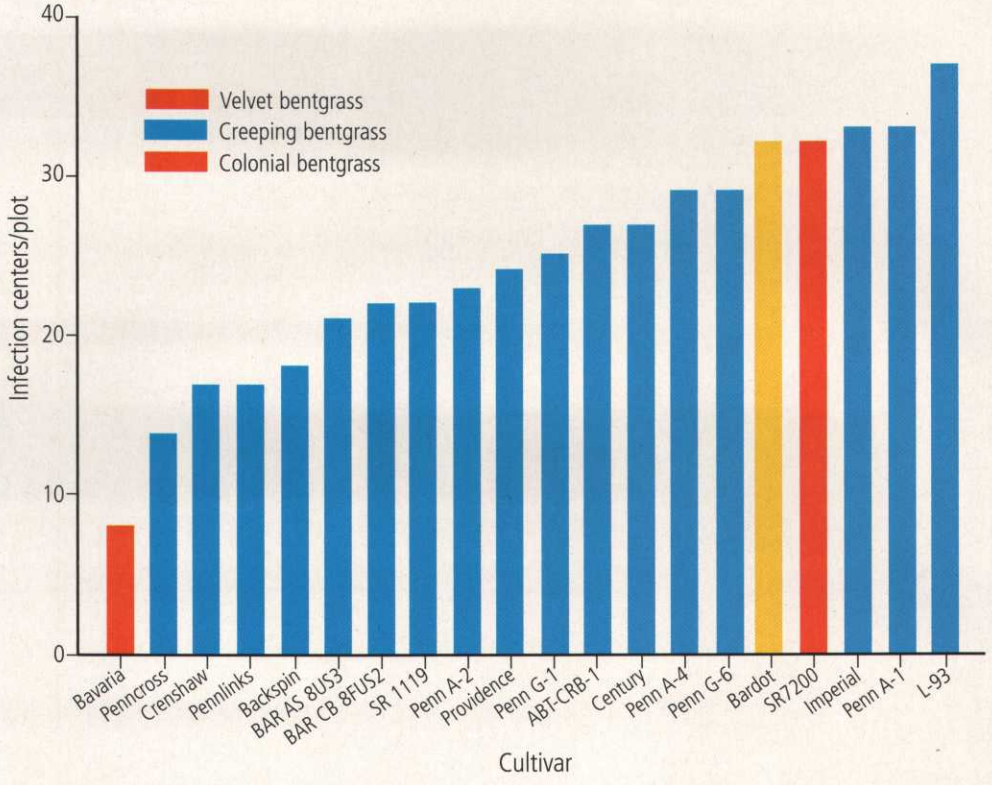
Disease incidence and severity varied between 2000 and 2002. In 2000, the total number of infection centers within each plot was greatest in early September. Recovery of bentgrass into infected patches began by mid-September and likely was enhanced by fertilizer applications in September and November.

The recovery process was slow, however,

Continued on page 64

FIGURE 2

Dead-spot infection centers for 20 *Agrostis spp.* grown on a USGA putting green located in College Park, Md. Ratings were made on Sept. 6, 2000 and represent peak dead-spot infection.



Continued from page 62

and most infection centers remained visible throughout the winter. A majority of the dead-spot patches recovered during late spring and early summer of 2001. Similar to observations on golf courses, dead-spot activity in the second year declined to low levels, and all cultivars fully recovered by November 2001. In 2002, however, disease severity again increased to unacceptable levels. Dead-spot recurrence likely was influenced by an extended period of summer heat stress and reinoculation of the study site. The impact of fertilization and nitrogen (N) source on dead spot severity is unknown.

Preliminary work at the University of Maryland revealed that once symptoms appear, no N source was found to be superior in speeding turf recovery. Applying small amounts of a water-soluble N (.1 to .125 pounds of N per 1,000 square feet) with each fungicide application may help to reduce dead-spot severity and speed bentgrass recovery.

When applied preventively, however, ammonium sulfate may help to reduce the potential for disease recurrence, while urea and nitrate-based N-sources may enhance dead spot.

According to Wetzel (2000), weekly applications of urea in conjunction with an effective fungicide reduced dead-spot severity. On the other hand, when applied alone, urea did not significantly reduce dead-spot severity when compared to the untreated control (Wetzel, 2000).

Field fungicide evaluation trials reported by Wetzel (2000) and Towers et al. (2001) showed

Continued on page 66

Figure 3: *Active dead-spot infections exhibit reddish-brown tissue along the periphery of infected spots and tan, dead tissue in the center.*

Figure 4: *Pseudothecia (sexually produced fruiting bodies) can often be found embedded in necrotic leaves, sheaths and stolons. Spores (arrow) contained within each fruiting body are a source of secondary inoculum.*

Continued from page 64

that propiconazole, chlorothalonil, thiophanate methyl, fludioxonil, iprodione and pyraclostrobin effectively controlled dead spot.

Future work

Dead spot continues to be a problem on newly constructed or methyl bromide-renovated putting greens throughout the eastern United States.

Preventive measures using both cultural and chemical strategies appear to be the best management strategy against the disease. During the first few years of bentgrass putting-green establishment, applying ammonium sulfate may help to prevent dead-spot occurrence.

Soil pH should be routinely monitored when ammonium sulfate is used as the sole nitrogen source because of the potential to reduce turfgrass quality. Preventive applications of the aforementioned chemicals will aid in reducing disease incidence. After disease symptoms appear, however, chemical control is more difficult and fungicides must be applied on a shortened interval (7 days to 10 days).

Curative management of dead spot often requires increased labor and expenses and generally is less effective. Studies currently are being conducted to elucidate environmental conditions that predispose plants to infection. Results from these studies likely will play a major role in the timing and implementation of preventive and curative control strategies aimed at managing dead spot.

Kaminski is a graduate student and Dernoeden is a professor in the Department of Natural Resource Sciences and Landscape Architecture at the University of Maryland in College Park.

REFERENCES

Camara, M.P.S, O'Neill, N.R., van Berkum, P., Dernoeden, P.H. and Palm, M.E. 2000. "Ophiosphaerella agrostis sp. nov. and its relationship to other species of Ophiosphaerella." *Mycologia* 92: 317-325.

Dernoeden, P.H., O'Neill, N.R., Camara, M.P.S. and Feng, Y. 1999. "A new disease of Agrostis palustris incited by an undescribed species of Ophiosphaerella." *Plant Dis.* 83:397.

Kaminski, J. E., Dernoeden, P. H., O'Neill, N. R., and Momen, B. 2002 "Reactivation of dead spot and growth, pseudothecia production and ascospore germination of Ophiosphaerella agrostis." *Plant Dis.* 86:1290-1296.

Kaminski, J. E. and Dernoeden P. H. 2002. "Geographic distribution, cultivar susceptibility, and field observations on dead spot." *Plant Dis.* 86:1236-1259.

Krausz, J.P., White, R.H., Foerster, W., Tisserat, N.A., and Dernoeden, P.H. 2001. "Bermudagrass dead spot: A new disease of bermudagrass caused by Ophiosphaerella agrostis." *Plant Dis.* 85: 1286.

Towers, G.W., Majumdar, P.R., Weibel, E.N., Frasier, C.L., Vaiciunas, J.N., Peacos, M., and Clarke, B. 2000. "Evaluation of chemical and biological fungicides for the control of dead spot in creeping bentgrass." *Rutgers Turfgrass Proceedings* 2000 32: 211-215.

Wetzel, H.C. 2000. "Evaluation of fungicides and urea for the control of dead spot in an 'L-93' putting green in Raleigh, N.C.," 1999. *Fungicide and Nematicide Tests* 55: 510.