Spring dead spot (SDS) is the most destructive disease of bermudagrass in the transition zone. The severity of SDS this spring is very high, probably due to our cool, relatively wet fall of 2003, followed by a long cold winter.

SDS is most severe in areas where the bermudagrass undergoes complete dormancy in winter. Spots generally begin to appear after the turf is at least three to five years old, which would correspond to a build-up of causal fungi and a significant

Spring Dead Spot

A Scourge of Bermudagrass in the Transition Zone

By Dr. Bruce Martin

ABOVE: Spring dead spot on ultradwarf bermudagrass putting green.

INSET: Spring dead spot affected bermudagrass stolon colonized by ectotrophic fungi.

thatch. Infected areas recover slowly in spring and weeds may invade these areas during the summer. The highest maintained turf is generally the most susceptible. Hybrid bermudagrasses which tend to produce excessive thatch are most prone to disease attack while cold hardy cultivars such as Midiron, Midfield, and Vamont are more resistant. Recent success in breeding by Okahoma State University has resulted in cultivars that have improved cold hardiness and improved SDS resistance compared to Tifway. These cultivars include Midlawn and Patriot, both vegetative selections and the seeded cultivars Yukon and Riviera.

The causal agents of SDS are *Ophiosphaerella korrae; Ophiosphaerella narmari;* and *Ophiosphaerella herpotricha*. Previously, two of these fungi were classified as species of *Leptosphaeria*. All of the reported causal agents listed are slow-growing root ectotrophic fungi, similar to organisms causing take-all patch, necrotic ring spot, and summer patch. In fact *Ophiosphaerella korrae* has been reported to cause both necrotic ring spot and SDS, although the strains may differ. Presumably, these fungi infect bermudagrass roots in the late summer and fall, and weaken the turf without visible symptoms. Then, low temperatures and other stresses that affect bermudagrass vigor and spring green up cause death of infected roots, stolons and rhizomes. In spring, roots, stolons and rhizomes are visibly colonized by dark brown to black mycelium from the fungi and vascular tissues are also colonized.

Symptoms include patches of a few inches up to two to three feet in diameter which appear in spring as affected bermudagrass begins to green up. SDS patches are sunken, generally well-defined and circular. Patches may enlarge over three to four years, often developing into rings, and then disappear. Affected spots may also remain greener in late fall going into winter. The patches are usually perennial in nature, recurring in the same location over several years. After a year or two from first occurrence, patches may develop into doughnut or frogeye patterns with relatively non-symptomatic bermudagrass in the centers. ►

Spring dead spot on overseeded bermudagrass putting green. Note Poa trivialis is denser in SDS patch.

Because recovery of patches in summer may depend on stolon re-growth, the use of certain pre-emergence herbicides for summer annual grassy weed control may affect recovery from SDS. Herbicides that work by inhibition of cell division (the dinitroanaline group, as well as dithiopyr) may inhibit new stolons from colonizing patches and slow recovery. Oxadiazon, on the other hand, does not inhibit rooting and might be a better choice of a pre-emergence herbicide if one is needed. Managers may also opt to use post-emergence techniques for weed control in areas prone to severe SDS.

SDS is a disease of bermudagrass that is intensively managed. Use of acidifying fertilizers such as ammonium sulfate at about 1.0 lb of N per growing month has been shown to help speed recovery and reduce disease severity. However, avoid excessive nitrogen fertilization and do not apply nitrogen in the fall. Raise mowing height and ensure adequate potassium levels in the fall. Reduce thatch by aerifying and pulverizing soil cores.

Fungicides labeled for SDS include propiconazole (Banner Maxx), fenarimol (Rubigan), thiophanate methyl (Cleary 3336), myclobutanil (Eagle) and azoxystrobin (Heritage). Complete control with any of these fungicides has been extremely variable, and research is needed to refine timing of application and perhaps application techniques. Research is needed in evaluations of all of these products; nevertheless, at this time, Rubigan is still the most consistent

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in its control of SDS. Because the aim is to control root infection by the causal fungi, these fungicides should be applied as drenches into the root zone. Several years of consecutive use may be required from complete control as the patches are typically reduced in size following each yearly fungicide use.

SDS on Putting Greens

Unfortunately, recent years have seen an increased incidence of SDS on bermudagrass putting greens. Several reasons may explain this. One is that there has been a change in cultivars from older greens of TifDwarf or TifGreen (328) to TifEagle, Champion or more recently MiniVerde. New constructions or renovations to make these changes usually requires fumigation with methyl bromide, which greatly reduces *Poa annua* infestations. The *Poa annua* control program most superintendents in the area utilized was Rubigan AS applied as three applications of four fl. oz. per 1,000 sq. ft. each or two applications of six fl. oz. per 1,000 sq. ft. each. While controlling *Poa annua*, superintendents were also, sometimes unknowingly, getting excellent SDS control. Another, related reason would be the use of alternative methods of *Poa annua* control, such as TranXit or Kerb applications, with no fungicide or a non-affective fungicide applied for SDS control. And possibly a third reason may be that some of our newer ultradwarf bermudagrasses are highly susceptible to the disease. This appears to be the case, but proof is still needed.

Unfortunately, we have seen SDS on ultradwarf greens that are only one year from planting, and before a significant thatch has accumulated. But, we've also seen the disease on older cultivars where an effective fungicide was omitted from the program the previous fall. Interestingly, on a recent trip to Argentina, I identified SDS on ultradwarf bermudagrass greens and on Tifway fairways. Those greens with SDS symptoms had not received Rubigan for *Poa annua* control, but greens that had received that treatment were free of the disease. Although circumstantial, the relationship was significant.

The specific identification of causal agents of SDS in the Carolinas needs re-examination, especially of SDS as it occurs on ultradwarf putting greens. Recently, Dr. Henry Wetzel identified SDS pathogens from Tifway and common bermudagrass in North and South Carolina, and showed that the great majority of causal agents were *O. korrae*. This differs from results obtained in Kansas and Oklahoma, where the primary causal agent has been shown to be *O. herpotricha*. We may find similar results from putting greens, but we need to be sure.

Also needed is research on fungicide and weed control programs to manage *Poa annua* and SDS. For instance, if TranXit is used for Poa annua control, will Rubigan, Eagle, 3336 or Banner Maxx be adequate to control SDS? Can we come up with strategies to mitigate unfavorable growth regulation by some of these fungicides? Are combinations of products in mixes or rotation more successful? Results are slow to accumulate because SDS occurrence and severity is variable and hard to predict. Also, results are unknown until six to eight months after treatments are applied. Nevertheless, research to answer these questions is underway.

In the meantime, it is suggested that superintendents be aware of vulnerability of their greens to SDS, especially if they reside in areas where the bermudagrass undergoes dormancy in winter. Make sure nutrients are not lacking, avoid excessive N in the fall, manage thatch preventively, and consider fall applications of fungicides. ■