Fight spring dead spot

By Gordon L. Kauffman, Ph.D.

I first encountered spring dead spot (SDS) in 2000 on a Tifway 419 bermudagrass fairway in Virginia Beach, VA while traveling with a USGA agronomist. I remember the agony on the superintendent's face as we discussed potential chemical control options and cultural practices to help reduce the damage from SDS the following spring. Finally the conversation turned to complete renovation and we contemplated a conversion to another species such as creeping bentgrass, or a more resistant cultivar of bermudagrass. At the time, options for this turf manager seemed bleak to say the least.

Researchers understood less back then about environmental and cultural practices impacting the life cycle of SDS, and there was no commercially acceptable chemical control. While a 30-40% reduction in disease severity was attainable, fungicide programs were costly because they typically required two or more applications. In addition, we understood less about the number, method, and timing of chemical applications necessary to provide an effective fungicide program for improved SDS control. Unfortunately, turf managers had very few options other than getting through the season with some turf recovery and hope for the best the following year.

Currently, more insight and information exists on how to fight SDS. Scientists now understand more about the life cycle of the causal pathogens, cultural and environmental factors that influence SDS development and breeding programs designed to select for more resistant bermudagrass cultivars. Also, chemical control options, including the timing and method of application of specific fungicides, are currently being assessed to develop more effective and improved fungicide programs to combat SDS. While my experiences with SDS have been primarily on golf courses, the disease can be problematic for sports turf managers. Athletic field managers often have, or are likely to deal with this disease if they are managing common bermudagrass (Cynodon dactylon) and Bermudagrass hybrids in the transition zone, and primarily in the northern most range of bermudagrass growth in the United States.

Symptoms and causal pathogen

SDS is a particularly devastating perennial disease to Bermudagrass because the pathogen begins its colonization by infecting turfgrass roots, stolons, and rhizomes. After visual diagnosis, control options are limited and likely too late. SDS infects bermudagrass of all ages,

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but most often occurs on established, 2-4 year old, intensely managed turfgrass stands. SDS kills Bermudagrass down to the soil surface while destroying roots, rhizomes, stolons, and shoots, leaving well defined, sunken, circular patches. Patches range in size from an inch up to a few feet in diameter and usually form as the bermudagrass breaks winter dormancy, although the infection likely occurs the previous fall. If left untreated, the patches begin to coalesce and devastate large turf areas.

The causal pathogens of SDS are three different root-infecting fungi called Ophiosphaerella herpotricha, O. korrae, and O. narmari. These pathogens are classified as ectotrophic root-infecting fungi (ERIF) based upon the site of primary infections. Species of these fungi are soil borne and grow over living turfgrass roots, rhizomes, and stolons similar to summer patch (Magnaporthe poae), which infects primarily bluegrasses, and take-all-patch (Gaeumannomyces graminis) of creeping bentgrass (Agrostis stoloniferous). Infected roots and stolons typically become rotted and covered with dark brown or black hyphae. O. korrae also causes necrotic ring spot disease of annual bluegrass (poa annua) and Kentucky bluegrass (poa pratensis).

Environmental, cultural factors

Several key environmental factors increase the chances of SDS infection and subsequent colonization. SDS is most active when temperatures and soil moisture favor fungal growth. Bermudagrass becomes most susceptible when growth slows in the early fall and spring during periods of cool, wet weather. Scientists believe, however, that much of the damage caused by SDS results from winter desiccation, making the initiation of fall dormancy, severity of the winter, and bermudagrass cold hardiness important factors for disease development. It appears that during the winter, SDS kills roots and crowns directly by infection, or indirectly by predisposing the Bermudagrass to winter injury and subsequent desiccation. bermudagrass cultivars with increased cold hardiness tend to exhibit greater resistance to SDS.

Cultural practices that promote increased winter hardiness can minimize SDS damage. For example, excessive fall nitrogen applications are not recommend in SDS prone areas. Avoid more than 4-5 lb. N/M/year, but most importantly, discontinue nitrogen applications at least six weeks before expected bermudagrass dormancy. Additional cultural practices used by turf managers to reduce the damage from SDS include routine core cultivation as an integral part of a thatch management program, improving soil drainage and compaction, maintaining adequate potassium fertility, and the use of acidifying fertilizers. Implementing these cultural solutions can be an effective first step when designing an SDS management program.

Chemical control

Control of SDS with fungicides has been spotty at best in the field and in control studies at turfgrass research plots throughout the United States. In order to suppress disease development, a systemic fungicide should be applied in the fall, when the pathogen begins to infect root tissue. The major factors influencing the control of SDS with fungicides include the timing and dilution rate of the application. When targeting a soil borne pathogen like SDS, chemical applications should be made in sufficient volumes of water so that the fungicide can effectively

move through the turf canopy and positioned for absorption by root and crown tissue. One difficulty in interpreting SDS fungicide trial data is that test plots are often not uniformly infested, resulting in plot rather than treatment effects.

Tredway and Butler (2003) studied both the timing and application method on SDS control for two Tifway bermudagrass athletic fields. In general, they found that Banner Maxx (4 oz/M) and Rubigan 1AS (6 oz/M) provided the best control of SDS, improving control by 41% compared to the control. On one field, the method of application significantly affected SDS suppression. Applications in 5 or 10 gallons/M were more effective than applications in 2.5 gallons/M, or 2.5 gallons/M and watered in with one-quarter inch of water. Therefore, in this study higher dilution rates led to better control of SDS. Syngenta's university sponsored research trials uncovered similar results. Increasing the spray dilution rate led to better control of dollar spot and brown patch on turfgrass maintained as a golf course fairway.

In the same SDS study, trends were noted regarding the most effective timing of fungicide applications on the control of SDS. Multiple applications were most effective and a single application made in August, September, or October was more effective than a one made in November. Fungicide applications were most effective when soil temperatures were between 60-80 degrees, prior to soil temperatures falling below 60 degrees, when bermudagrass root growth declines (Tredway and Butler, 2003). Based on these and other field trial data, a fungicide program should be used as one component to an integrated SDS management approach rather than as a silver bullet.

Biocontrol options are worth mentioning as an alternative approach to fighting SDS in the future. A bacterium was recently discovered that suppressed the growth of O. herpotricha in an Oklahoma State University laboratory. Applying bacterium as a soil drench has the potential for an effective component to an SDS management program. Research designed to test these bacterium on field plots started a couple of years ago, and the results will likely be available in the near future.

Breeding and the future research

According to turfgrass pathologists, bermudagrasses with good winter hardiness will better resist SDS. If feasible, selecting a more cold resistant variety of Bermudagrass like Midlawn, Midfield, Midiron, Mirage and Sundevil is recommended. Some common cold susceptible cultivars include Princess, Sonesta, Tifton 10, Sunturf, Tifway, Tifgreen, Tropica, and Oasis. Researchers are also focusing on specific pathogen-plant interactions in an attempt to breed for cultivars more directly resistant to the causal pathogens of SDS. The greatest limitation for turfgrass breeders remains the lack of a quick screening procedure. Five to six years are generally needed to gather meaningful results. Therefore, controlled studies have been initiated in parallel with breeding to determine specific plant genes that correspond to increased resistance to SDS.

SDS remains a devastating disease on bermudagrass turf in transition zone and specifically in the northernmost range of bermudagrass adaptation because of the variation in over-wintering weather conditions and different levels of bermudagrass resistance to cold. These factors largely dictate the extent of disease severity in the spring. Turf managers currently have more tools to fight the disease now compared to 5 years ago, but we still have a long way to go. Similar to fighting other tough to control diseases that affect turfgrasses, a multifaceted, integrated approach is essential. A program that uses every option including species selection, advantageous cultural practices, careful monitoring of environmental conditions, and both chemical and biocontrol options remains the current best defense used to fight SDS.

References:

Tredway, L.P. and E.L. Butler. 2003. Developing Effective Fungicide Programs for Spring Dead Spot Control. Turfgrass Trends. Dec. 1, 2003.

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