Evaluation of Chemical and Cultural Methods for the Management of Spring Dead Spot in Bermudagrass Turf

by

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Abstract

Spring dead spot (SDS) is one of the most severe and difficult to control diseases of bermudagrass. Disease symptoms can occur on managed home lawns, athletic fields, golf courses, and sod farms located in the transition zone where winter temperatures are cold enough to induce bermudagrass dormancy. In North America, SDS is reported to be caused by three closely related ectotrophic, root-infecting fungi; *Ophiosphaerella narmari*, *O. korrae*, and *O. herpotricha*. Symptoms are most noticeable in the spring when bermudagrass breaks winter dormancy and can persist well into the summer. The appearance of SDS symptoms on golf courses can interfere with the playability of bermudagrass turf during spring when weather conditions are most desirable for play. The destructive nature of this disease warrants the focus of management practices that emphasize disease prevention and effective levels of control.

Field experiments were conducted during the fall of 2009 and 2010 at Lubbock Country Club and Hillcrest Country Club in Lubbock, TX to examine the efficacy of fungicide applications in conjunction with a spray deposition agent (SDA) on SDS control. Research was located on 'Tifway 419' bermudagrass fairways with a history of severe SDS symptoms. Fungicide treatments included fenarimol (1.52 or 2.29 kg ai ha^{-1}) applied alone or in combination with thiophanate-methyl (7.62 or 9.16 kg ai ha^{-1}). Each fungicide treatment was applied alone (-) or in combination (+) with a SDA at 2.34 L ha^{-1}. Initial fungicide applications were made at Lubbock Country Club on September 25, 2009 and Hillcrest Country Club on September 17, 2010. Sequential

applications were made approximately 30 days later at both locations. A non-treated control (+ or - SDA) was included for comparison. All treatments significantly reduced SDS incidence compared to the + SDA and the - SDA controls when rated in April. Fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) + SDA, fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}), and fenarimol + thiophanate methyl (2.29 + 7.62 kg ai ha^{-1}) + SDA reduced SDS incidence to 4, 7, and 7%, respectively, in April and 1, 3, and 3%, respectively, in June. The addition of a SDA to fenarimol (2.29 kg ai ha^{-1}) and fenarimol (2.29 kg ai ha^{-1}) + thiophanate-methyl (7.62 kg ai ha^{-1}) treatments increased control of SDS in April and June.

Field experiments were conducted during the fall of 2010 and 2011 at Hillcrest Country Club in Lubbock, TX to examine the efficacy of combining cultivation practices and fungicide applications on SDS control. Treatments were arranged in a 2 x 4 factorial split plot design with four replications. Cultivation treatment was the main plot and fungicide treatment was the sub-plot. Cultivation treatments were conducted just prior to initial fungicide applications and consisted of no cultivation, verticutting, hollow-tine aerification, and aerification + verticutting. Fungicide treatments consisted of fenarimol at 2.29 kg ai ha^{-1} or tebuconazole at 0.87 kg ai ha^{-1}. All treatments received a sequential fungicide application 30 days after initial treatment. Cultivation treatments were not conducted prior to sequential fungicide applications. Cultivation treatments that caused the greatest disturbance of the upper rootzone (i.e. aerification) significantly reduced SDS incidence by April and June compared to the non-treated control (no cultivation), regardless of fungicide treatment.

The non-treated control resulted in 12 to 15% SDS incidence by April and 8 to 10% SDS incidence by June, regardless of fungicide treatment. Spring dead spot incidence was 1 to 7% and 2 to 7% by April and 0 to 5% and 0 to 4% by June in response to aerification alone and aerification + verticutting, respectively, regardless of fungicide treatment. Aerification enhanced the efficacy of tebuconazole more than fenarimol for the control of SDS, regardless of rating date. Bermudagrass exhibited 1 to 2% SDS incidence by April and 0% in June in response to tebuconazole (0.87 kg ai ha^{-1}), while fenarimol (2.29 kg ai ha^{-1}) resulted in 7% SDS incidence by April and 4 to 5% SDS incidence in June following aerification or aerification + verticutting.

Field experiments were conducted during the fall of 2010 and 2011 at Hillcrest Country Club in Lubbock, TX to examine the efficacy of fungicide application timings and newer fungicide chemistries on SDS control. Treatments were arranged in a 2 x 8 factorial within a randomized complete block design with four replications. The main factor was application timing [July followed by (fb) Aug. and Aug. fb September] and the sub-factor was fungicide treatment. Fungicide treatments consisted of fenarimol at 2.29 kg ai ha^{-1}, thiophanate-methyl at 7.62 kg ai ha^{-1}, fenarimol + thiophanate-methyl at 2.29 kg ai ha^{-1} + 7.62 kg ai ha^{-1}, fluoxastrobin at 0.43 kg ai ha^{-1}, tebuconazole at 0.87 kg ai ha^{-1}, azoxystrobin at 0.12 kg ai ha^{-1}, and azoxystrobin + propiconazole at 0.99 kg ai ha^{-1}. Initial fungicide applications were made on July 22, 2010 and July 28, 2011 or Aug. 15, 2010 and Aug. 29, 2011. Sequential applications were made approximately 30 days later for all fungicide treatments. A non-treated control (water only) was sprayed at each application timing and was included for comparison. All

fungicide treatments significantly reduced SDS incidence regardless of application timing compared to the non-treated control (60 to 63% SDS incidence) by April. The July fb Aug. application timing exhibited significantly less SDS incidence than the Aug. fb Sept. application timing for all fungicide treatments except fluoxastrobin (April and June) and azoxystrobin + propiconazole (June only). Bermudagrass exhibited the least amount of SDS incidence (8%) by April and (1%) by June in response to tebuconazole (July fb Aug. application timing). Similar levels of SDS were observed in response to July fb Aug. applications of fenarimol, fenarimol + thiophanate-methyl, and azoxystrobin + propiconazole which exhibited 10, 12, and 14% SDS incidence by April and 3, 7, and 6% SDS incidence by June.

Field experiments were conducted during the spring and summer of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX to examine bermudagrass recovery from SDS in response to PRE herbicide applications. Initial PRE herbicide applications were made on March 15, 2011 and March 23, 2012 to a 'Tifway 419' bermudagrass fairway exhibiting severe SDS symptoms. Herbicide treatments consisted of prodiamine at 0.73 kg ai ha^{-1}, pendimethalin at 2.5 kg ai ha^{-1}, oryzalin at 1.68 kg ai ha^{-1}, dithiopyr at 0.56 kg ai ha^{-1}, oxadiazon at 3.4 kg ai ha^{-1}, indaziflam at 0.035 kg ai ha^{-1}, and dimethenamid at 1.68 kg ai ha^{-1}. A non-treated control was included for comparison. In 2011 and 2012 bermudagrass in the non-treated control exhibited similar recovery from SDS (46 and 59%, respectively) as bermudagrass in response to oxadiazon at 3.4 kg ai ha^{-1} (55 and 57%, respectively) 14 weeks after treatment (WAT). Pendimethalin at 2.5 kg ai ha^{-1} resulted in 33 to 38% bermudagrass

recovery from SDS 14 WAT, regardless of year. In 2011, oryzalin, dithiopyr, prodiamine, and dimethenamid applications resulted in 21 to 25% bermudagrass recovery from SDS 14 WAT. In 2012, bermudagrass exhibited similar recovery in response to prodiamine and oryzalin (24 and 26%, respectively), while response to dithiopyr and dimethenamid was 33 and 34%, respectively, 14 WAT. Bermudagrass exhibited the least amount of recovery (13 to 14%) in response to indaziflam at 0.035 kg ai ha^{-1}, regardless of year.

Nomenclature: Bermudagrass, *Cynodon* spp. Rich.; 'Tifway 419,' [*C. dactylon* x *C. trasvaalensis* (L.) Pers.]; fenarimol, a-(2-chlorophenyl)-a-(4(chlorophenyl)-5-pyrimidinemethanol; thiophanate-methyl, (dimethyl 4,4'-o-phenylenebis[3-thioallophanate]); fluoxastrobin, [(1E)-[[6-(2-Chlorophenoxyl)-5-fluoro-4-pyrimidinyl]oxy]phenyl]5,6-dihydro-1,4,2-dioxazin-3-yl)methanon-O-methyloxime]; tebuconazole, a-[2-(4-chlorophenyl)ethyl]-a-(1,1-dimethylethyl)-1H-1,2,4-triazole-1-ethanol; azoxystrobin, methyl (E)-2-{2-[6-(2-cyanophenoxy)pyrimidin-4-yloxy]phenyl}-3-methoxyacrylate; azoxystrobin + propiconazole, methyl (E)-2-{2-[6-(2-cyanophenoxy)pyrimidin-4-yloxy]phenyl}-3-methoxyacrylate + (CAS no. 60207-90-1); Grounded; prodiamine, 2,4-dinitro-N^3,N^3-dipropyl-6-(trifluoromethyl)-1,3-benzenediamine; pendimethalin, N-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitroben-4-(trifluoromethyl)benzenamine; oryzalin, 3,5-dinitro-N^4N^4-dipropylsulfanilaminde; dithiopyr, S,S'-dimethyl 2-(difluoromethyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-3,5-pyridinedicarbothioate; oxadiazon, [2-tert-butyl-4-(2,4-dichloro-5-

isopropoxyphenyl)-Δ-1,3,4-oxadiazolin-5-one]; indaziflam; 1,3, 5-Triazine-2,4-diamine,N-[(1R,2S)-2,3-dihydro-2,6-dimethyl-1H-inden-1-yl]-6-(1-fluoroethyl); dimethenamid, (S)-2-chloro-N-(2,4-dimethyl-3-thienyl)-N-(2-methyloxy-1-methylethyl)-acetamide.

Key Words: Spring dead spot, bermudagrass, cultural practices, soil and spray deposition agent, cultivation practices, hollow-tine aerification, verticutting, thatch layer, fungicide application timing, preemergence herbicides.

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Chapter I

Literature Review

Bermudagrass

Bermudagrass (*Cynodon* spp. Rich.) is a versatile, warm-season perennial turfgrass species that is used for forage, turf, and soil stabilization throughout the world (Christians 2011; McCarty 2005; Zhang et al. 2006). It is primarily utilized for home lawns, athletic fields, recreational areas, and golf courses (Zhang et al. 2006; Turgeon 2011). Bermudagrass is well adapted to the tropical and subtropical regions of the United States, but recent improvements in cold tolerance of several new cultivars have extended its use further north (Anderson et al. 2002; Gatschet et al. 1994). Although less tolerant of cold temperatures than zoysiagrass (*Zoysia* spp. Willd.) and buffalograss (*Bouteloua dactyloides* (Nutt.) J.T. Columbus), bermudagrass may be successfully cultivated in the southern and central regions of the transition zone, which is delineated from east to west at 37 N latitude and separates the temperate and subtropical zones in the U.S. (Anderson et al. 2002; McCarty 2005). However, bermudagrass often experiences winter-kill (desiccation) in the northern transition zone where temperatures may drop below -7°C (Beard 1973; McCarty 2005; Turgeon 2011). Bermudagrass is best adapted to grow in moderately well-drained, fertile soils of relatively fine texture, but may tolerate a wide range of soil types (Beard 1973). It also tolerates a wide range of soil pH (5.5 to 7.5), but optimal growth is achieved at a pH of 6.5 (Beard 1973; Duble 1996).

Bermudagrass requires a medium to high intensity of cultural maintenance when sustained as a golf course fairway. Heavy traffic from golfers and turfgrass maintenance

equipment leads to an increase in soil compaction (McCarty 2005). Compacted soil often results in reduced water/air infiltration and bermudagrass root growth/penetration (Beard 1973; Christians 2011; Turgeon 2011). Hollow-tine aerification is often performed on bermudagrass in spring and late summer to reduce thatch accumulation, alleviate soil compaction, increase water infiltration, and enhance gas exchange within the soil (Carrow et al. 1987; McCarty 2005). Hollow-tine aerification consists of removing 6 to 20 mm cores from the soil profile to a maximum depth of 10 cm (Carrow and Petrovic 1992). Vertical mowing is also conducted to reduce thatch, disperse aerification cores, and correct the appearance of grain (horizontal orientation of turfgrass shoots). Also known as 'verticutting', this process consists of slicing through the turf canopy to a depth of 2.5 to 5 cm with vertically oriented blades mounted on a horizontal shaft (McCarty 2005). Although both practices are necessary components of golf course management and are conducted during periods of vigorous growth and/or low environmental stress, several negative impacts may result from their conduction. Wounding of bermudagrass plants may increase susceptibility to pathogen infections and temporarily increase water loss, while soil cores containing weed seed brought to the surface may lead to increased weed pressure (Brauen et al. 1998; Christians 2011; Turgeon 2011; Younger 1967).

Topdressing practices are often applied in conjunction with hollow-tine aerification to help increase water infiltration, alleviate thatch, reduce winter desiccation, create a smooth playing surface, and encourage a denser, finer-textured turf (Beard 1973; Beard and Beard 2005; Karcher and Rieke 2005; McCarty 2005). Topdressing involves the application of a thin layer of soil, sand, or other finely granulated material over the turf surface (Christians 2011; McCarty 2005; Turgeon 2011). Careful attention is paid to

apply equivalent materials to those already found in the rootzone. Heavy applications (0.130 m^3/100 m^2) are typically made following aerification practices (Christians 2011; Turgeon 2011). Drag mats may be utilized to help smooth the playing surface and further distribute topdressing material through the turf canopy and into aerification holes (Duble 2010b; McCarty 2005). Mechanical damage (wounding) caused from the abrasion of sand materials and drag mats may encourage pathogen infections, increase water loss, and/or reduce bermudagrass photosynthetic capability (Fry and Huang 2004).

Mowing is the most basic and commonly performed cultural practice in turfgrass management (Lowe et al. 2000; Turgeon 2011). In general, the lower the mowing height, the more frequent the mowing schedule, in order to produce a dense, uniform playing surface (Christians 2011; McCarty 2005; Turgeon 2011). For example, hybrid bermudagrass [*C. dactylon* x *C. transvaalensis* (L.) Pers.] fairways are typically maintained with a reel mower at a height of 1.3 cm (Martin et al. 2001; Turgeon 2011). Constant removal of above-ground tissue causes a temporary cessation of root growth, a reduction in carbohydrate production and storage, a port of entry for pathogen infections, a temporary increase in water loss from severed leaf ends, and a reduction in water absorption through plant roots (Fry and Huang 2004; McCarty 2005; Turgeon 2011).

Irrigation is predominantly applied to bermudagrass fairways through an above-ground automated system; however, water may also be applied by hand through the practice of syringing when turfgrass experiences severe environmental stress (Barrett et al. 2003; Christians 2011; Fry and Huang 2004). Irrigation frequency and amount is often affected by soil infiltration rates, evapotranspiration (ET) potential, specific turfgrass watering requirements, and the level of required turfgrass performance (Christians 2011;

Kneebone et al. 1992). Bermudagrass exhibits moderate drought tolerance compared to other warm-season turfgrass species (Fu et al. 2004; Turgeon 2011). For example, a bermudagrass fairway growing in an arid environment may be maintained with 834 to 930 mm of irrigation water annually, depending on bermudagrass variety and ET potential (Garrot and Mancino 1994). However, golf course superintendents often apply irrigation daily to avoid the potential for reduced aesthetic quality in response to environmental stress. Frequent irrigation may saturate the upper few centimeters of the soil profile and discourage the penetration of bermudagrass roots (Duble 2010a; Landry 2010).

Preemergence (PRE) herbicides are often applied to turfgrass stands in early spring for the control of annual grasses and broadleaf weeds (Bhowmik and Bingham 1990; McCullough et al. 2007; Turgeon 2011). However, certain PREs may have a negative impact on the growth of bermudagrass turf (Bingham 1967; Fishel and Coats 1993; Kaminski and Dernoeden 2004). The use of dinitroaniline (DNA) herbicides, such as prodiamine and pendimethalin, may cause abnormal swelling of turfgrass root tips (‘clubbed-roots’), stunting of lateral root growth, and/or severely pruned roots present in the herbicide-treated zone of the soil profile (Fishel and Coats 1993; Mitra and Bhowmik 2005). Soil mobility of DNA herbicides is low compared to other herbicide families, due to low water solubility and high potential for hydrogen bonding (Fishel and Coats 1994). Therefore, DNA herbicides may persist for several weeks to months in the rootzone causing reductions in healthy bermudagrass roots and shoots to occur over a significant period of time (Capo-chichi et al. 2005; Goatley et al. 2003). As a result, utilization of certain PRE herbicides may interfere with recovery of bermudagrass in spring and

summer from damage caused by winter desiccation, heavy traffic, or disease (Kaminski et al. 2004).

The intense nature of the management practices required to maintain bermudagrass as a golf course fairway may also have a negative impact on the overall health and sustainability of this same environment. Aerification, topdressing, verticutting, and mowing cause significant wounding to bermudagrass plants (Christians 2011; McCarty 2005; Turgeon 2011). These management practices, combined with frequent irrigation applications, may increase susceptibility to turfgrass disease infection (Schumann and D'Arcy 2006). In addition, PRE herbicide applications may interfere with the recuperative capacity of bermudagrass from damage caused by freezing temperatures, excessive wear, and disease (Kaminski et al. 2004).

Spring Dead Spot

Spring dead spot (SDS) is one of the most damaging and difficult to control diseases of bermudagrass turf (Crahay et al. 1988; McCarty and Lucas 1989). Although the primary host is bermudagrass, SDS has also been reported on buffalograss and zoysiagrass (Tisserat et al. 1999; Tredway and Butler 2007). Disease symptoms often occur on managed home lawns, athletic fields, golf courses, and sod farms located in the transition zone where winter temperatures are cold enough to induce bermudagrass dormancy (Butler and Tredway 2007; Martin et al. 2001; Perry et al. 2010; Vann and Patton 2012; Wadsworth and Young 1960).

Spring dead spot was first observed in 1936 by a golf course superintendent in Oklahoma (Wadsworth and Young 1960). Even though the causal agent was unknown,

Wadsworth and Young (1960) observed that the disease was most prevalent on intensely managed, well-fertilized, high-quality bermudagrass turf. In North America, SDS is reported to be caused by three closely related ectotrophic, root-infecting pathogens: *Ophiosphaerella narmari*, *O. korrae*, and *O. herpotricha* (Baird et al. 1998; Elliot 1995). Each pathogen is an Ascomycete belonging to the Order *Pleosporales* and Family *Phaeosphaeriaceae* (Tredway et al. 2008b). These species were initially distinguished from each other based on differences in their pseudothesia, asci, and ascospores; however, molecular methods to differentiate DNA (deoxyribonucleic acid) characteristics of each causal agent are required for accurate identification (Tisserat et al. 2004; Tisserat et al. 1989; Wetzel et al. 1999a). Sauer et al. (1993) developed the first species-specific restriction fragment length polymorphism (RFLP) identification probe for *O. herpotricha*. In subsequent years, polymerase chain reaction (PCR) primer sets were also developed for selective amplification of the ITS-rDNA regions from each of the three *Ophiosphaerella* causal agents (Tisserat et al. 1994; Tredway et al. 2008b; Wetzel et al. 1999a).

Although *O. korrae* and *O. narmari* are the primary causal agents of SDS in Australia and New Zealand, all three causal agents have been documented in the U.S. (Tredway et al. 2008b). Tredway et al. (2008b) reported *O. herpotricha* as the primary cause of SDS in the Midwest (N. Texas, Texas Panhandle, Oklahoma, and S. Kansas), while *O. Korrae* is more prevalent in California and the Eastern U.S. (Endo et al. 1985; Wetzel et al. 1999b). Few instances of *O. narmari* have been reported in California, Oklahoma, Kansas, and North Carolina (Tredway et al. 2008b; Wetzel et al. 1999b). The distribution of all three causal agents has been reported to overlap in certain regions of

the U.S. For example, Wetzel et al. (1999b) observed the first occurrence of all three SDS causal agents on bermudagrass in Kansas and Oklahoma. Despite the potential overlap, *O. herpotricha* is still considered to be the most prevalent and aggressive SDS causal agent in the Midwest (Iriarte et al. 2004; Iriarte et al. 2005; Tredway et al. 2008a).

Spring dead spot is caused by soilborne pathogens that colonize the roots, rhizomes, and stolons of bermudagrass plants during spring and/or fall (Tredway et al. 2008b; Walker et al. 2009). Symptoms are first noticeable in the spring when bermudagrass breaks winter dormancy and can persist well into the summer (Dernoeden et al. 1991; Fermanian et al. 1980; McCarty and Lucas 1989; Venkatasubbaiah et al. 1994). In severe cases, bermudagrass may not fully recover before the onset of winter, leaving the turf susceptible to further damage from freezing temperatures (Nus and Shashikumar 1993; Pair et al. 1986).

The development of SDS symptoms on golf courses can interfere with the playability of bermudagrass turf during spring when weather conditions are most desirable for play (Walker et al. 2009). Spring dead spot symptoms appear as well-defined, bleached, circular patches ranging in size from a few centimeters to a meter in diameter (Baird et al. 1998; Dernoeden et al. 1991). Turfgrass plants within the disease patches eventually collapse to the ground leaving sunken necrotic areas (Iriarte et al. 2004; Walker 2009). Infected plant parts such as rhizomes, stolons, and roots appear blackened and necrotic with the presence of dark ectotrophic runner hyphae along their surfaces (McCarty et al. 1991; Tredway et al. 2008b; Vincelli and Williams 1998a). The severity of disease symptoms is directly related to the intensity of cold winter temperatures during bermudagrass dormancy (McCarty et al. 1991). Tisserat et al. (1989)

suggested that soil temperatures ranging from 10 to 25°C may be conducive to the colonization of bermudagrass roots by *O. herpotricha*, while McCarty et al. (1991) observed that bermudagrass injury from SDS increased as low-temperature stress increased. Although the specific mechanisms of this phenomenon are relatively unknown, Tredway et al. (2008b) noted that necrosis of bermudagrass perennial vegetative structures (roots, rhizomes, and stolons) reduced the plant's ability to survive freezing temperatures. Symptoms often reoccur in the same area for several years, but may vary in size, shape, and severity (Perry et al. 2010; Tredway et al. 2008b; Venkatasubbaiah et al. 1994). Weeds often germinate in the center of disease patches causing further decline in turfgrass aesthetic quality (Fermanian et al. 1980). The destructive nature of this disease warrants the focus of turfgrass managers on practices that emphasize disease prevention and effective levels of control (Butler and Tredway 2007; Tredway et al. 2008b).

Previous SDS control in bermudagrass has primarily focused on cultural management techniques to suppress disease colonization/severity by enhancing bermudagrass winter hardiness through the promotion of turfgrass root health (Butler and Tredway 2007; Landry 2010; Lucas 1980). Reductions in bermudagrass root growth and development caused by soil compaction, poor soil drainage, and excessive thatch accumulation increase the occurrence of freezing injury during winter months and may facilitate the development of SDS (Butler and Tredway 2007). Therefore, proper implementation of cultural practices such as irrigation, fertility, and thatch management rather than relying on fungicide applications alone may help reduce SDS severity (Baird et al. 1998; Butler and Tredway 2007; Vann and Patton 2012).

Adjustments to irrigation amounts and frequency may aid in the development of a deeper bermudagrass root system. Irrigation practices that utilize deep and infrequent water applications may increase root penetration into the soil profile and increase tolerance to drought and freezing temperatures (Landry 2010; McCarty 2005). Furthermore, early morning irrigation applications may reduce prolonged leaf wetness and reduce the development of fungal diseases (Duble 1978). Raising bermudagrass mowing heights prior to winter may encourage deeper roots and buffer plant crowns from winter desiccation (Butler and Tredway 2007; Landry 2010; Lucas 1980). In addition, golf course superintendents are discouraged from making nitrogen fertilizer applications during late fall (Butler and Tredway 2007; Lucas 1980). Lucas (1980) suggested that late season growth stimulation resulting from nitrogen applications may increase SDS development in bermudagrass. Prolonged shoot growth could delay bermudagrass dormancy leading into winter months, thereby predisposing the turf to low-temperature damage and SDS (McCarty et al. 1991). McCarty et al. (1992) observed an increase in SDS severity of 128% in response to fall N applications of 98 kg N ha^{-1}. Final nitrogen applications of the year should be made in mid-August to late-September to avoid prolonged shoot growth heading into late fall/winter (Vincelli and Williams 1998b). In contrast, applications of potassium in the fall may improve bermudagrass winter survival and reduce SDS development and/or severity (McCarty et al. 1991). Two fall applications of potassium chloride or potassium sulfate, totaling 48.9 kg K_20 ha^{-1}, on 3 to 4 week intervals may aid in the reduction of SDS symptoms (Butler and Tredway 2007). Although many of these cultural management practices have been effective at reducing SDS development and severity, no single management practice will provide complete

control of the disease and results vary greatly from year to year (Vincelli and Williams 1998b).

Cultural control efforts that focus on nitrogen source or cultivation techniques may also decrease SDS severity in bermudagrass fairways (Butler and Tredway 2007; Tredway et al. 2008b). Spring dead spot may be enhanced by high soil pH (> 7.5) (Butler and Tredway 2007). Maintaining a neutral to slightly acidic soil pH is conducive to the development of beneficial microbial activity, which may result in the reduction of *Ophiosphaerella* spp. (Tomaso-Peterson 2008; Vincelli and Williams 1998a). Previous research has aimed at altering the rhizosphere pH through adjustments in nitrogen fertility source (Dernoeden et al. 1991; McCarty et al. 1992; Tredway et al. 2009). Tredway et al. (2009) observed reductions in *O. herpotricha* disease severity (< 5%) of bermudagrass when applications of ammonium sulfate were applied compared to sulfur coated urea, calcium nitrate, and urea (≥ 45% disease severity) (Tredway 2009). In contrast, applications of calcium nitrate significantly suppressed *O. Korrae* disease severity (3%) in bermudagrass compared to urea, ammonium sulfate, and sulfur coated urea, which exhibited ≥ 21% disease severity (Tredway 2009). Dernoeden et al. (1991) also observed that ammonium-based fertilizers greatly reduced soil pH and decreased the severity of SDS at two locations in Maryland. However, reductions in SDS severity took several years to occur and the disease was not completely controlled.

Soil compaction, poor soil drainage, and excessive thatch accumulation may reduce bermudagrass root depth and growth (Butler and Tredway 2007). Cultural management practices that result in severe disturbances of the upper soil profile may reduce soilborne diseases in turfgrass (Martin and Hudgens 2012; Tisserat and Fry 1997;

Vincelli and Williams 1998b). Liu et al. (1995) observed a significant reduction in the number of dollar spot (*Sclerotinia homeocarpa*) patches (9) when the fungicide benomyl (3 kg ai ha^{-1}) was applied 1 day after hollow-tine aerification compared to benomyl alone (48) and the non-treated control (103). Tisserat and Fry (1997) reported that aerification alone and aerification + verticutting were most effective in reducing SDS symptoms (9 and 10% incidence, respectively) in bermudagrass when conducted twice a year compared to verticutting alone (14%). Disruption of the rootzone may interrupt colonization of *Ophiosphaerella* spp. and/or stimulate new root growth from severed rhizomes and stolons (Tisserat and Fry 1997). However, cultivation practices alone did not completely reduce SDS severity.

Although cultural control practices may provide some disease reduction, fungicide applications have become a necessary component of a SDS control program (Walker 2009). Fungicides such as fenarimol, thiophanate-methyl, propiconazole, myclobutanil, and benomyl have been utilized for SDS control in athletic fields, golf courses, and sod farms (McCarty et al. 1992; Tredway et al. 2008a; 2008b; Vann and Patton 2012). McCarty et al. (1992) observed a 72% reduction in SDS of 'Tifway' bermudagrass in response to benomyl at 12 kg ai ha^{-1}. Fenarimol (1.5, 2.3, and 3.0 kg ai ha^{-1}) applied during the fall resulted in a 66% reduction in SDS, while propiconazole applied at 2.5 kg ai ha^{-1} resulted in 56% disease reduction (McCarty et al. 1992). Butler and Tredway (2006) observed SDS reductions of 66 to 89% in response to fall applications of fenarimol alone (2.3 kg ai ha^{-1}) or two applications at either 1.5 or 2.3 kg ai ha^{-1}, while propiconazole (1.9 kg ai ha^{-1}) provided a 51 to 52% reduction. McCarty et al. (1992) observed a reduction in SDS severity when applications of benomyl (12 kg ai

ha^{-1}), fenarimol (1.5, 2.3, and 3 kg ai ha^{-1}), and thiophanate-methyl (12 kg ai ha^{-1}) exhibited 92, 72, 52, 86, and 41% less disease than the non-treated control, respectively. In addition, Tomaso-Peterson (2011) observed a reduction in SDS severity (3% SDS incidence) with applications of fenarimol (2.3 kg ai ha^{-1}) + thiophanate-methyl (6.2 kg ai ha^{-1}) compared to the non-treated control (18% SDS incidence). Luc et al. (2005) also observed a reduction in SDS severity (4%) compared to the non-treated control (23%) when fenarimol (2.3 kg ai ha^{-1}) was applied in conjunction with thiophanate-methyl (6.2 kg ai ha^{-1}).

While several fungicides have reduced SDS in bermudagrass fairways, few of these chemistries are labeled for use on home lawns and the level of disease control varies greatly from year to year (Tredway et al. 2008b). In addition, Gowan Company recently announced that the production of Rubigan (fenarimol) will cease by December 2012, due to high levels of manufacturing expense and the introduction of less expensive sulfonylurea herbicides for the control of annual bluegrass (*Poa annua* L.) (Tredway 2012). As a result, several new fungicides and tank-mixtures have recently been registered for use in turf (metconazole, tebuconazole, azoxystrobin + propiconazole, and fluoxastrobin + chlorothalonil) that may be efficacious against SDS. For example, Walker (2009) observed SDS severity ratings of 3.63, 3.75, and 3.88 in response to multiple applications (1 spring and 2 fall, 3 fall, and 4 fall, respectively) of tebuconazole (3.1 kg ha^{-1}) compared to the non-treated control (1.00). Disease severity was rated on a scale of 1 (no living plants present within the SDS patch) to 6 (no evidence of SDS symptoms in turfgrass) (Walker 2009).

Investigation into the ecology and epidemiology of SDS may provide clues to enhance fungicide efficacy and ensure long-term control (Butler and Tredway 2006; Walker et al. 2009). The colonization timing of SDS is poorly understood (Tredway et al. 2008a). Butler and Tredway (2007) and Lucas (1980) reported that disease colonization occurs primarily in the fall, while Walker et al. (2009) observed colonization in spring and fall months. Butler and Tredway (2006) evaluated single and sequential applications of fenarimol (1.5 to 2.3 kg ai ha^{-1}) in late summer and/or fall for the control of SDS, but reported no significant differences between application timings. Tomaso-Peterson (2011) compared spring and fall applications of fenarimol, fenarimol + thiophanate-methyl, propiconazole, azoxystrobin, and myclobutanil for the control of SDS in a 'Tifway' bermudagrass fairway. All application timings of fenarimol and propiconazole significantly reduced SDS incidence ($\leq$ 3.0%) compared to the non-treated control (18%); however, treatments were not significantly different from one another (Tomaso-Peterson 2011). Furthermore, spring only applications (March, April, and May) of fenarimol were equally effective in reducing SDS severity as fall only applications of all other fungicide chemistries (Tomaso-Peterson 2011). No adjustments in fungicide application timings have provided complete control of SDS in bermudagrass and results have varied from year to year.

Soil temperature may also play an important role in the development of SDS in bermudagrass (Endo et al. 1985; Tisserat et al. 1989; Walker et al. 2006). Walker et al. (2006) observed infection and colonization of *O. herpotricha* on bermudagrass in the spring with variable occurrences in the fall. Although root lesion length was greatest (13.1 mm) on 'Jackpot' bermudagrass at 17ºC, similar lesion lengths (8.6 to 11.1 mm)

were observed between 12 and 21ºC (Walker et al. 2006). Furthermore, although smaller in size, lesions (2.6 to 3.0 mm) were observed on roots subjected to 25 to 30ºC (Walker et al. 2006). In the field, no differences in SDS colonization were observed in 2003 when disease patches were sampled in July, October, and November, but higher root colonization was observed in October and November in 2004 (Walker et al. 2006). The occurrence of several periods of preferred colonization temperatures between fall and spring may explain the inconsistency of fall fungicide applications for SDS control. Walker (2009) determined that single applications of fungicides in the spring should be followed by at least one fall application; in contrast, the addition of a spring application when making multiple fall applications did not significantly decrease SDS. Therefore, due to the high cost of fungicide applications for SDS control in bermudagrass, two fall or one spring and one fall application provide the most effective control (Walker 2009). The inconsistency of control with fungicide timings and chemistries for the control of SDS warrants further investigation.

The chemical characteristics of certain fungicides, such as K_{oc} values, may also play an important role in the management of soilborne diseases. The K_{oc} (organic carbon distribution coefficient) value of a fungicide is the ability of a pesticide to adsorb to surrounding soil particles and organic matter within the soil profile (Long et al. 2005). A pesticide with a lower K_{oc} (weak adsorption) generally indicates a high level of solubility and is more likely to be relocated from the target zone in the soil profile via irrigation runoff. A pesticide with a higher K_{oc} (strong adsorption) may be strongly bound to soil particles and organic matter, and will more likely be moved by association to eroded soil, organic matter, or sediment particles in the soil profile (Long et al. 2005). As a result, the

higher K_{oc} values of several turfgrass fungicides may limit their distribution in the rootzone. For example, two efficacious fungicides, Torque (tebuconazole) and Rubigan (fenarimol), exhibit high K_{oc} values (803-1251 and 757, respectively), compared to Cleary's 3336 (thiophanate-methyl) (K_{oc}=225); thus, drastically reducing the availability of these compounds to be absorbed and systemically translocated throughout bermudagrass roots (Anonymous 2012a; 2012b; Kegley et al. 2010a; 2010b; Long et al. 2005). The combination of soil disturbance and fungicide application may enhance infiltration of high K_{oc} fungicides into the soil profile (Tisserat and Fry 1997; Vincelli and Williams 2012); however, no research has examined this phenomenon to date.

Soil conditions, such as excessive thatch, soil compaction, salt accumulation, and high soil pH may increase the hydrophobicity of the soil profile, further limiting fungicide distribution throughout the rootzone (Larsbo et al. 2008; Nadeau et al. 1993; Stiles et al. 2007; Vincelli and Williams 2012). Soil surfactants modify the surface properties of liquids by enhancing and facilitating the penetration of fungicides into plants and hydrophobic soils (McCarty 2005). As a result, the combination of soil surfactants and fungicide applications may also enhance infiltration of fungicides that exhibit high K_{oc} values into the soil profile, thus increasing fungicide efficacy against soilborne diseases (Long et al. 2005; Nadeau et al. 1993; Stiles et al. 2007; Wong and Kaminski 2007). Liu et al. (1996) observed that the wetting agent Aqua-Gro (2.54 L ha^{-1}) increased movement, uptake, and biological availability of the fungicide benomyl (1 kg ai ha^{-1}) when applied for the control of dollar spot in creeping bentgrass (*Agrostis stolonifera* L.). Fidanza et al. (2007) observed a reduction in fairy ring (*Lycoperdon* spp.) disease severity ($\leq$ 5% incidence) in response to azoxystrobin (0.62 kg ai ha^{-1}), flutolanil

(9.8 kg ai ha^{-1}), polyoxin-D (0.31 kg ai ha^{-1}), or pyraclostrobin (0.56 kg ai ha^{-1}) plus the soil surfactant Revolution (3.11 L ha^{-1}), compared to the non-treated control (35% incidence). The combination of flutolanil (9.8 kg ai ha^{-1}) plus the wetting agent Primer (19 L ha^{-1}) reduced the number of fairy rings (1) compared to the non-treated control (23) (Fidanza 1999). Similarly, Stiles et al. (2007) observed a reduction in fairy ring severity (< 10% incidence) in response to pyraclostrobin (0.56 kg ai ha^{-1}) plus the soil surfactant Magnus (5.15 L ha^{-1}) compared to the non-treated control (60% incidence). However, no research has examined the effect of soil and spray deposition agents on the efficacy of fungicides for the control of SDS.

Successful SDS management programs should emphasize an integrated pest management strategy of utilizing proper implementation of cultural practices to enhance bermudagrass winter hardiness through the promotion of turfgrass root health. In addition, fungicide applications result in highly erratic levels of SDS control. The combination of cultural practices and fungicide applications may enhance efficacy of SDS control in bermudagrass. As a result, this research evaluated the combination of soil and spray deposition agents, as well as cultivation practices, to increase fungicide penetration and availability in the rootzone. In addition, this research also evaluated earlier fungicide application timings, as well as the availability of newer fungicide chemistries; which may enhance efficacy against SDS by increasing fungicide availability within the rootzone before the pathogen becomes active. As SDS symptoms appear in the spring, focus often changes from disease control to bermudagrass recovery within symptom patches. In a golf course fairway, PRE herbicide applications may coincide with the appearance of SDS symptoms. The presence of chemical barriers may

prevent bermudagrass recovery throughout spring and summer. As a result, this research also evaluated bermudagrass recovery from SDS symptoms in response to PRE herbicide applications.

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Chapter II

Enhancement of Fungicide Efficacy with Spray Deposition Agents for the Control of Spring Dead Spot in Bermudagrass

Abstract

The addition of soil and spray deposition agent (SDA) may increase fungicide penetration into the rootzone and improve fungicide efficacy against soilborne diseases, such as spring dead spot (SDS). Field experiments were conducted during the fall of 2009 and 2010 at Lubbock Country Club and Hillcrest Country Club in Lubbock, TX to examine the efficacy of fungicide applications in conjunction with a SDA agent on SDS control. Research was located on 'Tifway 419' [*C. dactylon* x *C. trasvaalensis* (L.) Pers.] bermudagrass fairways with a history of severe SDS symptoms. Fungicide treatments included fenarimol (1.52 or 2.29 kg ai ha^{-1}) applied alone or in combination with thiophanate-methyl (7.62 or 9.16 kg ai ha^{-1}). Each fungicide treatment was applied alone (-) or in combination (+) with a SDA at 2.34 L ha^{-1}. Initial fungicide applications were made at Lubbock Country Club on September 25, 2009 and Hillcrest Country Club on September 17, 2010. Sequential applications were made approximately 30 days later at both locations. A non-treated control (+ or - SDA) was included for comparison. Disease incidence ratings were conducted the following spring (2010 and 2011) when symptoms appeared following bermudagrass green-up. Percent SDS incidence was visually assessed in April and June of the following year at each location. All treatments significantly reduced SDS incidence compared to the non-treated control (+ or – SDA) when rated in April. Bermudagrass in the non-treated control exhibited 31 to 33% SDS incidence by

April. Fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) + SDA, fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}), and fenarimol + thiophanate methyl (2.29 + 7.62 kg ai ha^{-1}) + SDA reduced SDS incidence to 4, 7, and 7%, respectively, in April. Fenarimol (2.29 kg ai ha^{-1}) alone reduced SDS incidence to 21%, but the addition of a SDA further decreased SDS incidence to 12% by April. Fenarimol at (1.52 kg ai ha^{-1}) + thiophanate-methyl (9.16 kg ai ha^{-1}) (+ or – SDA) reduced SDS incidence 17 to 21% by April. Bermudagrass in the non-treated control (+ or – SDA) exhibited 18 to 20% SDS incidence by June. The lowest SDS incidence in June was observed in response to fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) (3%), fenarimol + thiophanate methyl (2.29 + 7.62 kg ai ha^{-1}) + SDA (3%), and fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) + SDA (1%). Fenarimol (2.29 kg ai ha^{-1}) alone reduced SDS incidence to 11%, but the addition of a SDA further decreased SDS incidence to 5% by June. Fenarimol at 1.52 kg ai ha^{-1} + thiophanate-methyl at 9.16 kg ai ha^{-1} (+ or – SDA) reduced SDS incidence 8 to 9% by June. The addition of a SDA to fenarimol (2.29 kg ai ha^{-1}) and fenarimol (2.29 kg ai ha^{-1}) + thiophanate-methyl (7.62 kg ai ha^{-1}) treatments improved control of SDS in April and June.

Introduction

Spring dead spot (SDS) is one of the most damaging and difficult to control diseases of bermudagrass (*Cynodon* spp. Rich.) turf (Crahay et al. 1988; McCarty and Lucas 1989). It is caused by three ectotrophic, root-infecting fungi; *Ophiosphaerella narmari, O. korrae*, and *O. herpotricha* (Baird et al. 1998; Elliot 1995). Disease symptoms can occur on managed home lawns, athletic fields, golf courses, and sod farms

located in the transition zone where winter temperatures are cold enough to induce bermudagrass dormancy (Butler and Tredway 2007; Martin et al. 2001; Perry et al. 2010; Vann and Patton 2012; Wadsworth and Young 1960). Spring dead spot symptoms appear as well-defined, bleached, circular patches ranging in size from a few centimeters to a meter in diameter (Baird et al. 1998; Dernoeden et al. 1991). Turfgrass plants within the disease patches eventually collapse to the ground leaving sunken necrotic areas (Iriarte et al. 2004; Walker 2009). Infected plant parts such as rhizomes, stolons, and roots appear blackened and necrotic with the presence of dark ectotrophic runner hyphae along their surfaces (McCarty et al. 1991; Tredway et al. 2008b; Vincelli and Williams 1998). Symptoms are first noticeable in the spring when bermudagrass breaks winter dormancy and can persist well into the summer (Dernoeden et al. 1991; Fermanian et al. 1980; McCarty and Lucas 1989; Venkatasubbaiah et al. 1994). In severe cases, bermudagrass may not fully recover before the onset of winter, leaving the turf susceptible to further damage from freezing temperatures (Nus and Shashikumar 1993; Pair et al. 1986).

Cultural practices focused on increasing bermudagrass root growth and cold tolerance have been employed in attempts to reduce SDS severity. Deep and infrequent irrigation applications may increase bermudagrass root penetration and biomass (Landry 2010; McCarty 2005). Furthermore, early morning irrigation applications may reduce prolonged leaf wetness and reduce the occurrence of fungal pathogens (Duble 1978). Raising bermudagrass mowing heights heading into the fall may encourage deeper roots and buffer plant crowns from winter desiccation (Butler and Tredway 2007; Lucas 1980; McCarty 2005). In addition, golf course superintendents are discouraged from making

late fall nitrogen fertilizer applications that prolong shoot growth and delay the onset of bermudagrass dormancy (Butler and Tredway 2007; Lucas 1980; McCarty et al. 1991).

Although cultural control practices may alleviate some disease pressure, fungicide applications have become a necessary component of an efficacious SDS control program (Walker 2009). Fenarimol, thiophanate-methyl, propiconazole, myclobutanil, and benomyl are labeled for SDS control in athletic fields, golf courses, and sod farms (McCarty et al. 1992; Tredway et al. 2008a; 2008b; Vann and Patton 2012). McCarty et al. (1992) observed a 66% reduction in SDS of a 'Tifway' bermudagrass fairway in response to fenarimol (1.5, 2.3, and 3.0 kg ai ha^{-1}) applied during the fall. Butler and Tredway (2006) observed reductions in SDS incidence of 66 to 89% in response to fall applications of fenarimol alone (2.3 kg ai ha^{-1}) or two applications at either 1.5 or 2.3 kg ai ha^{-1}. In addition, applications of fenarimol (2.3 kg ai ha^{-1}) in conjunction with thiophanate-methyl (6.2 kg ai ha^{-1}) reduced SDS incidence to 3% compared to the non-treated control (18% SDS incidence) (Tomaso-Peterson 2011).

Soil conditions, such as excessive thatch, soil compaction, salt accumulation, and high soil pH may increase soil hydrophobicity and limit fungicide penetration into the rootzone (Larsbo et al. 2008; Nadeau et al. 1993; Stiles et al. 2007; Vincelli and Williams 2012). Furthermore, several fungicides have high K_{oc} (organic carbon distribution coefficient) values (ex. fenarimol, K_{oc} =757 compared to thiophanate-methyl, K_{oc}=225), that may limit movement into the rootzone and reduce SDS control (Anonymous 2012b; Kegley et al. 2010a; 2010b; Long et al. 2005). Soil and spray deposition agents modify the surface properties of liquids by enhancing and facilitating the penetration of fungicides into plants and hydrophobic soils (McCarty 2005). As a result, tank-mixing

fungicides with soil and spray deposition agents may enhance fungicide infiltration into hydrophobic soil profiles and increase efficacy against root-infecting diseases (Long et al. 2005; Nadeau et al. 1993; Stiles et al. 2007; Wong and Kaminski 2007). Liu et al. (1996) stated that the wetting agent Aqua-Gro (Aquatrols, Paulsboro, NJ 08066) (2.54 L ha^{-1}) increased movement, uptake, and biological availability of the fungicide benomyl (1 kg ai ha^{-1}) when applied to control dollar spot (*Sclerotinia homeocarpa*) disease in creeping bentgrass (*Agrostis stolonifera* L.). Fidanza et al. (2007) observed a reduction in fairy ring (*Lycoperdon* spp.) disease severity (≤ 5% incidence) in response to azoxystrobin (0.62 kg ai ha^{-1}), flutolanil (9.8 kg ai ha^{-1}), polyoxin-D (0.31 kg ai ha^{-1}), or pyraclostrobin (0.56 kg ai ha^{-1}) plus a soil surfactant (Revolution; Aquatrols, Paulsboro, NJ 08066) at 3.11 L ha^{-1}, compared to the non-treated control (35% incidence). The combination of flutolanil (9.8 kg ai ha^{-1}) plus a wetting agent (Primer; Aquatrols, Paulsboro, NJ 08066) at 19 L ha^{-1} reduced the number of fairy ring patches (1) compared to the non-treated control (23) (Fidanza 1999). Similarly, Stiles et al. (2007) observed a reduction in fairy ring severity (< 10% incidence) in response to pyraclostrobin (0.56 kg ai ha^{-1}) plus a soil surfactant (Magnus Hydration; Precision Laboratories, Waukegan, IL 60085) at 5.15 L ha^{-1} compared to the non-treated control (60% incidence). However, no research has examined the effect of soil and spray deposition agents on the efficacy of fungicides for the control of SDS. Therefore, the objective of this study was to quantify whether the addition of a soil and spray deposition agent could enhance the efficacy of fenarimol and thiophanate-methyl for the control of SDS in a bermudagrass fairway.

Materials and Methods

Field experiments were conducted during the fall of 2009 and 2010 at Lubbock Country Club and Hillcrest Country Club in Lubbock, TX to examine the efficacy of fungicide applications in conjunction with a spray deposition agent on SDS control. Research was located on 'Tifway 419' [*C. dactylon* x *C. trasvaalensis* (L.) Pers.] bermudagrass fairways with a history of severe SDS symptoms and mowed to a height of 0.6 cm. The soil at Lubbock Country Club is an Amarillo fine sandy loam (fine-loamy, mixed, superactive, thermic Aridic Paleustalfs) with a pH of 7.9 and an organic matter content of 1.5%, while the soil at Hillcrest Country Club is an Arch fine sandy loam (fine-loamy, carbonatic, thermic, Aridic Calciustepts) with a pH of 8.2 and an organic matter content of 1.5%. Soils for both locations were collected and measured to a depth of 10 cm below the soil surface. All experimental areas were mowed 24 hrs before fungicide application and twice weekly thereafter. No preemergence herbicides were applied to the research plots in the spring in order to promote recovery from the disease.

Plots (1.5 x 1.5 m) were arranged in a 2 x 5 factorial within a randomized complete block design with four replications. The main factor was soil and spray deposition agent (SDA) (+ or -) and the sub-factor was fungicide treatment. Fungicide treatments included fenarimol (Rubigan; Gowan Company, Yuma, AZ, 85364) (at rates of 1.52 or 2.29 kg ai ha^{-1}) applied alone or in combination with thiophanate-methyl (Cleary's 3336; Cleary Chemical, Dayton, NJ 08810) (at rates of 7.62 or 9.16 kg ai ha^{-1}) (Table 2.1). Fenarimol is a pyrimidine fungicide that controls susceptible fungal pathogens through the inhibition of DeMethylation sterol biosynthesis, while thiophanate-methyl (thiophanate) controls susceptible fungal pathogens through the

inhibition of mitosis and cell division (Anonymous 2012a). Each fungicide treatment was applied alone or in combination with a soil and spray deposition agent (SDA) (Grounded; Helena Chemical Company, Collierville, TN 38017) at 2.34 L ha^{-1}. Fungicide treatments were applied using a CO_2-powered backpack sprayer equipped with XR8008VS nozzle tips (Teejet; Spraying Systems Co., Wheaton, IL 60129) calibrated to deliver 757 L ha^{-1} at 221 kPa. Initial fungicide applications were made at Lubbock Country Club on September 25, 2009 and Hillcrest Country Club on September 17, 2010. Sequential applications were made approximately 30 days later at both locations. A non-treated control (water + or – SDA) was sprayed at each application timing and was included for comparison. Irrigation (0.6 cm) was applied immediately following all fungicide treatments to increase infiltration into the soil profile.

Data collected included bermudagrass phytotoxicity and % SDS incidence. Bermudagrass phytotoxicity was visually assessed 1 and 2 weeks following each fungicide application on a percent scale relative to the non-treated control, where 0% equaled no bermudagrass injury and 100% equaled complete bermudagrass death. Percent SDS incidence was visually assessed on a percent scale relative to the non-treated control, where 0% equaled no SDS incidence and 100% equaled complete SDS incidence. Plots were evaluated in April and June of the following year at each location.

Bermudagrass phytotoxicity and SDS incidence data were arcsine square-root transformed to stabilize variance as described by Ahrens et al. (1990) prior to being subjected to analysis of variance in SAS (version 9.3; SAS Institute, Cary, NC 27513) using error partitioning appropriate to a split plot analysis in the general linear models procedure. Interpretations were not different from non-transformed data; therefore, non-

transformed means are presented for clarity. No significant year-by-treatment interactions were detected in the data; thus, data from each trial were combined. All data were subjected to analysis of variance in SAS using the appropriate expected mean square values described by McIntosh (1983). Treatment means were separated using Fisher's protected least significant difference (LSD) test at $\alpha = 0.05$.

Results and Discussion

Significant SDA ($F = 50.6$, $P < 0.001$) and fungicide treatment ($F = 325.7$, $P < 0.001$) main effects as well as an interaction between SDA and fungicide treatment ($F = 23.2$, $P < 0.001$) were observed for SDS incidence. No bermudagrass phytotoxicity was observed in response to fungicide applications throughout the length of either trial (data not shown). All treatments significantly reduced SDS incidence compared to the non-treated control (+ or – SDA) when rated in April (Table 2.2). Bermudagrass in the non-treated control exhibited 31 to 33% SDS incidence by April. Fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) + SDA, fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}), and fenarimol + thiophanate methyl (2.29 + 7.62 kg ai ha^{-1}) + SDA significantly reduced SDS incidence to 4, 7, and 7%, respectively, in April. Tomaso-Peterson (2011) observed similar SDS incidence (3%) in response to fenarimol (2.3 kg ai ha^{-1}) + thiophanate-methyl (6.2 kg ai ha^{-1}). Fenarimol (2.29 kg ai ha^{-1}) alone reduced SDS incidence to 21%, but the addition of a SDA further decreased SDS incidence to 12% by April. Butler and Tredway (2006) observed reductions in SDS incidence of 66 to 89% in response to sequential fall applications of fenarimol at 1.5 or 2.3 kg ai ha^{-1}. Similarly, McCarty et al. (1992) observed a 66% reduction in SDS of a 'Tifway' bermudagrass

fairway in response to fall applications of fenarimol at 1.5, 2.3, and 3.0 kg ai ha^{-1}. Fenarimol at (1.52 kg ai ha^{-1}) + thiophanate-methyl (9.16 kg ai ha^{-1}) with and without a SDA in our research reduced SDS incidence 17 to 21% by April. The lower rate of fenarimol (1.52 kg ai ha^{-1}) may have contributed to reduced SDS control.

Bermudagrass in the non-treated control exhibited 18 to 20% SDS incidence by June (Table 2.3). The lowest SDS incidence in June was observed in response to fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) (3%), fenarimol + thiophanate methyl (2.29 + 7.62 kg ai ha^{-1}) + SDA (3%), and fenarimol + thiophanate-methyl (2.29 + 9.16 kg ai ha^{-1}) + SDA (1%). Fenarimol (2.29 kg ai ha^{-1}) alone reduced SDS incidence to 11%, but the addition of a SDA further decreased SDS incidence to 5% by June. Fenarimol at 1.52 kg ai ha^{-1} + thiophanate-methyl at 9.16 kg ai ha^{-1} (+ or – SDA) in this research reduced SDS incidence 8 to 9% by June.

The addition of a SDA to fenarimol (2.29 kg ai ha^{-1}) and fenarimol (2.29 kg ai ha^{-1}) + thiophanate-methyl (7.62 kg ai ha^{-1}) treatments increased control of SDS in April and June. Spring dead spot incidence in response to fenarimol (2.29 kg ai ha^{-1}) and fenarimol (2.29 kg ai ha^{-1}) + SDA was 21 and 12%, respectively, by April and 11 and 5%, respectively, by June. Spring dead spot incidence in response to fenarimol (2.29 kg ai ha^{-1}) + thiophanate-methyl (7.62 kg ai ha^{-1}) and fenarimol (2.29 kg ai ha^{-1}) + thiophanate-methyl (7.62 kg ai ha^{-1}) + SDA was 13 and 7%, respectively, by April and 7 and 3%, respectively, by June. Similarly, Stiles (2007) observed < 10% fairy ring (*Lycoperdon* spp.) incidence in response to pyraclostrobin (0.56 kg ai ha^{-1}) + the soil surfactant Magnus Hydration at 5.15 L ha^{-1} compared to the non-treated control (60% fairy ring incidence). Flutolanil (9.8 kg ai ha^{-1}) + the wetting agent Primer at 19 L ha^{-1} reduced the

number of fairy rings from 23 in the non-treated control to 1 (Fidanza 1999), while the addition of the soil surfactant Revolution (3.11 L ha^{-1}) to azoxystrobin (0.62 kg ai ha^{-1}), flutolanil (9.8 kg ai ha^{-1}), polyoxin-D (0.31 kg ai ha^{-1}), or pyraclostrobin (0.56 kg ai ha^{-1}) reduced fairy ring (*Lycoperdon* spp.) incidence to ≤ 5% compared to the non-treated control (35% incidence) (Fidanza et al. 2007).

Thatch layer and organic matter present in the turfgrass microenvironment may have affected the response of fungicide treatments to the addition of the SDA. Tests taken and averaged across both sites revealed that the thatch layer was ≤ 1.3 cm and the organic matter content was 1.5%. Therefore, the presence of physical barriers for the reduction of fungicide infiltration through the soil profile was minimal. Furthermore, utilization of SDA's may have been more beneficial if applied in conjunction with fungicide chemistries that exhibit higher K_{oc} values (ex. tebuconazole, K_{oc} = 803-1251). Although the addition of a SDA did enhance the efficacy of several treatments utilized in this study, complete control of SDS was not achieved. Evaluation of fungicides plus a SDA incorporated into cultural practices that promote soil disturbance (ex. aerification or verticutting) may further increase efficacy for the control of SDS by opening the turfgrass canopy and removing the thatch layer. Despite the fact that complete control was not achieved, this research indicates that the addition of SDA's to applications of fenarimol and fenarimol + thiophanate-methyl, may further increase efficacy for the control of SDS in a bermudagrass fairway.

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Table 2.1. Fungicide treatments (+ or -) SDA[a] and application dates at Lubbock Country Club and Hillcrest Country Club in Lubbock, TX in 2009 and 2010.

Trt #	Treatment	Rate	SDA	Application Dates	
		—kg ai ha^{-1}—		——2009——	——2010——
1	Non-treated Control	——	-	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
2	Fenarimol	2.29	-	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
3	Fenarimol + TM	2.29 + 7.62	-	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
4	Fenarimol + TM	2.29 + 9.16	-	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
5	Fenarimol + TM	1.52 + 9.16	-	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
6	Non-treated Control	——	+[b]	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
7	Fenarimol	2.29	+	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
8	Fenarimol + TM	2.29 + 7.62	+	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
9	Fenarimol + TM	2.29 + 9.16	+	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15
10	Fenarimol + TM	1.52 + 9.16	+	Sept. 25 + Oct. 26	Sept. 17 + Oct. 15

[a] Abbreviations: soil and spray deposition agent, SDA; thiophanate-methyl, TM.
[b] Treatments were applied with a soil and spray deposition agent (Grounded; Helena Chemical Company, Collierville, TN) at 2.34 L ha^{-1}.

Table 2.2. Visual ratings of % SDS[a] incidence in April of 2010 and 2011 in Lubbock, TX in response to fungicide applications (+ or -) SDA.

Treatment	Rate	SDS Incidence (%)[b]	
	—kg ai ha^{-1}—	- SDA	+ SDA[c]
Non-treated Control	——	33 aA[d]	31 aA
Fenarimol	2.29	21 bA	12 deB
Fenarimol + TM	2.29 + 7.62	13 cdA	7 efB
Fenarimol + TM	2.29 + 9.16	7 efA	4 fA
Fenarimol + TM	1.52 + 9.16	21 bA	17 bcA

[a] Abbreviations: spring dead spot, SDS; soil and spray deposition agent, SDA; thiophanate-methyl, TM.
[b] Treatment means are combined from experiments conducted at both locations.
[c] Treatments were applied with a soil and spray deposition agent (Grounded; Helena Chemical Company, Collierville, TN) at 2.34 L ha^{-1}.
[d] Means within both columns followed by the same lowercase letter are not significantly different at $P \leq 0.05$, according to Fisher's Protected LSD test. Means within both rows followed by the same uppercase letter are not significantly different at $P \leq 0.05$ according to Fisher's Protected LSD test.

Table 2.3. Visual ratings of % SDS[a] incidence in June of 2010 and 2011 in Lubbock, TX in response to fungicide applications (+ or -) SDA.

Treatment	Rate	SDS Incidence (%)[b]	
	—kg ai ha^{-1}—	- SDA	+ SDA[c]
Non-treated Control	——	18 aA[d]	20 aA
Fenarimol	2.29	11 bA	5 deB
Fenarimol + TM	2.29 + 7.62	7 cdA	3 efB
Fenarimol + TM	2.29 + 9.16	3 efA	1 fA
Fenarimol + TM	1.52 + 9.16	9 bcA	8 cdA

[a] Abbreviations: spring dead spot, SDS; soil and spray deposition agent, SDA; thiophanate-methyl, TM.

[b] Treatment means are combined from experiments conducted at both locations.

[c] Treatments were applied with a soil and spray deposition agent (Grounded; Helena Chemical Company, Collierville, TN) at 2.34 L ha^{-1}.

[d] Means within both columns followed by the same lowercase letter are not significantly different at $P \leq 0.05$, according to Fisher's Protected LSD test. Means within both rows followed by the same uppercase letter are not significantly different at $P \leq 0.05$ according to Fisher's Protected LSD test.

Chapter III

Combination of Cultivation Techniques and Fungicides for the Control of Spring Dead Spot in Bermudagrass

Abstract

Efficacy of fungicides for the control of spring dead spot (SDS) in bermudagrass turf may be limited by fungicide penetration through the soil profile and into the rootzone. Cultivation practices aimed at reducing the thatch layer and decreasing soil compaction may increase fungicide infiltration. Field experiments were conducted during the fall of 2010 and 2011 at Hillcrest Country Club in Lubbock, TX to examine the efficacy of combining cultivation practices and fungicide applications on SDS control. Plots measured 1.5 x 1.5 m and were arranged in a 2 x 4 factorial split plot design with four replications. Cultivation treatment was the main plot and fungicide treatment was the sub-plot. Cultivation treatments were conducted just prior to initial fungicide applications and consisted of no cultivation, verticutting, hollow-tine aerification, and aerification + verticutting. Fungicide treatments consisted of fenarimol at 2.29 kg ai ha^{-1} or tebuconazole at 0.87 kg ai ha^{-1}. All treatments received a sequential fungicide application 30 days after initial treatment. Cultivation treatments were not conducted prior to sequential fungicide applications. Data collected included % bermudagrass phytotoxicity and % SDS incidence. Cultivation treatments that caused the greatest disturbance of the upper rootzone (i.e. aerification) significantly reduced SDS incidence by April and June compared to the non-treated control (no cultivation), regardless of fungicide treatment. No cultivation treatments resulted in 12 to 15% SDS incidence by April and 8 to 10%

SDS incidence by June, regardless of fungicide treatment. Spring dead spot incidence was 1 to 7% and 2 to 7% by April and 0 to 5% and 0 to 4% by June in response to aerification alone and aerification + verticutting, respectively, regardless of fungicide treatment. Verticutting alone did not significantly reduce SDS incidence compared to the non-treated control in April or June, regardless of fungicide treatment. Tebuconazole at 0.87 kg ai ha^{-1} and fenarimol at 2.29 kg ai ha^{-1} applied alone resulted in 12 and 15% SDS incidence by April and 8 to 10% SDS incidence by June. Aerification enhanced the efficacy of tebuconazole more than fenarimol for the control of SDS, regardless of rating date. Bermudagrass exhibited 1 to 2% SDS incidence by April and 0% in June in response to tebuconazole (0.87 kg ai ha^{-1}), while fenarimol (2.29 kg ai ha^{-1}) resulted in 7% SDS incidence by April and 4 to 5% SDS incidence in June following aerification or aerification + verticutting. Aerification practices open up the turfgrass canopy, thus helping to increase fungicide penetration into the rootzone. As a result, combining aerification practices with fungicide applications may enhance fungicide efficacy for the control of SDS.

Introduction

Spring dead spot (SDS) disease predominantly infects bermudagrass (*Cynodon* spp. Rich.) managed as a home lawn, athletic field, golf course, or a sod farm (Butler and Tredway 2007; Martin et al. 2001; Perry et al. 2010; Vann and Patton 2012; Wadsworth and Young 1960). Symptoms are most severe on bermudagrass turf located in the transition zone where winter temperatures are cold enough to induce dormancy (Crahay 1988; Martin et al. 2001; McCarty and Lucas 1989). Three closely related ectotrophic

root-infecting fungal pathogens: *Ophiosphaerella narmari*, *O. korrae*, and *O. herpotricha* (Baird et al. 1998; Elliot 1995) are known to cause SDS in bermudagrass roots, rhizomes, and stolons during the fall, but symptoms become noticeable in spring when bermudagrass breaks winter dormancy (Dernoeden et al. 1991; Fermanian et al. 1980; McCarty et al. 1991; Tredway et al. 2008; Venkatasubbaiah et al. 1994). In severe cases, diseased areas may not fully recover before winter, further exposing bermudagrass crowns and stolons to freezing temperatures (Dernoeden et al. 1991; Nus and Shashikumar 1993; Pair et al. 1986). These patches often reappear for several seasons, increasing in size and severity (Pair et al. 1986; Tredway et al. 2008). The germination of weeds in the center of necrotic patches may cause further decline in turfgrass aesthetic quality and reduce the playability of turf (Fermanian et al. 1980).

Enhancing bermudagrass root health and cold tolerance has been the primary focus of SDS management. Irrigation applied deeply and infrequently may avoid oversaturation of the upper soil profile and promote root penetration, while early morning irrigation applications help reduce prolonged leaf wetness and the potential for fungal pathogen infection (Duble 1978; Landry 2010; McCarty 2005). Increasing bermudagrass mowing height in late summer/early fall may encourage deeper roots, enhance carbohydrate production, and buffer crowns/stolons from winter desiccation (Butler and Tredway 2007; Landry 2010; Lucas 1980; McCarty 2005). Furthermore, discouraging late fall nitrogen fertilizer applications helps promote a natural transition of bermudagrass dormancy (Butler and Tredway 2007; Lucas 1980; McCarty et al. 1992). However, no single management practice has been observed to completely control SDS in

bermudagrass fairways and often result in erratic levels of control (Vincelli and Williams 1998)

Fungicide applications have become a necessary component of an efficacious SDS control program (Walker 2009). McCarty et al. (1992) reported that fenarimol (1.5, 2.3, and 3.0 kg ai ha^{-1}) applied during the fall reduced SDS on a 'Tifway' bermudagrass fairway 66%. Fall applications of fenarimol alone (2.3 kg ai ha^{-1}) or sequential applications (1.5 or 2.3 kg ai ha^{-1}) reduced SDS 66 to 89% (Butler and Tredway 2006). Walker (2009) observed SDS severity ratings of 3.63, 3.75, and 3.88 in response to multiple applications (1 spring and 2 fall, 3 fall, and 4 fall, respectively) of tebuconazole (3.1 kg ha^{-1}) compared to the non-treated control (1.00). Spring dead spot disease severity was rated on a scale of 1 (no living plants present within the SDS patch) to 6 (no evidence of SDS symptoms) (Walker 2009).

Spring dead spot control may be limited by fungicide infiltration into the soil profile and accumulation in the rootzone (Kegley et al. 2010; Long et al. 2005; Vincelli and Williams 2012). Excessive thatch layers (> 1.3 cm), soil compaction, salt accumulation, and high soil pH may increase soil hydrophobicity and decrease fungicide penetration (Larsbo et al. 2008; Nadeau et al. 1993; Stiles et al. 2007; Vincelli and Williams 2012). In addition, the chemical characteristics of certain fungicides, such as K_{oc} (organic carbon distribution coefficient) values, may also play an important role in fungicide efficacy. High K_{oc} values associated with several turfgrass fungicides may limit their infiltration through the soil profile and distribution into the rootzone (Long et al. 2005). For example, tebuconazole and fenarimol exhibit high K_{oc} values (803-1251 and 757, respectively), thus drastically reducing the absorption and translocation of these

fungicides throughout bermudagrass roots (Anonymous 2012b; 2012c; Kegley et al. 2010; Long et al. 2005).

Hollow-tine aerification is often performed on bermudagrass turf in spring and late summer to reduce thatch accumulation, alleviate soil compaction, increase water infiltration, and enhance gas exchange within the soil (Carrow et al. 1987; McCarty 2005). This is accomplished by removing cores (6 to 20 mm diameter) from the soil profile to a maximum depth of 10 cm (Carrow and Petrovic 1992). Vertical mowing is also conducted to reduce thatch, disperse aerification cores, and correct the appearance of grain (horizontal orientation of turfgrass shoots). Also known as 'verticutting', this process consists of slicing through the turf canopy to a depth of 2.5 to 5 cm with vertically oriented blades (McCarty 2005). Cultural management practices that result in severe disturbances of the upper soil profile may help reduce the presence and infection of soilborne diseases in turfgrass (Martin and Hudgens 2012; Tisserat and Fry 1997; Vincelli and Williams 1998). Liu et al. (1995) observed a reduction in the number of dollar spot (*Sclerotinia homeocarpa*) patches (9) on creeping bentgrass (*Agrostis stolonifera* L.) in response to benomyl (3 kg ai ha^{-1}) following hollow-tine aerification, compared to benomyl alone (48) and the non-treated control (103). Tisserat and Fry (1997) reported that the aerification alone and aerification plus verticutting were most effective in reducing SDS (9 and 10% incidence, respectively) in bermudagrass when conducted twice a year compared to verticutting alone (14% incidence). Disruption of the rootzone may interrupt colonization of *Ophiosphaerella* spp. and/or stimulate new root growth from severed rhizomes and stolons (Tisserat and Fry 1997). The combination of soil disturbance and fungicide application may enhance infiltration of high K_{oc} fungicides

into the soil profile (Tisserat and Fry 1997; Vincelli and Williams 2012). However, no research has examined the efficacy of fungicides in combination with cultivation for the control of SDS. Therefore, the objective of this study was to evaluate the efficacy of cultivation practices in conjunction with fungicide applications for the control of SDS in a bermudagrass fairway.

Material and Methods

Field experiments were conducted during the fall of 2010 and 2011 at Hillcrest Country Club in Lubbock, TX on an Arch fine sandy loam (fine-loamy, carbonatic, thermic Aridic Calciustepts) soil with a pH of 8.2 and an organic matter content of 1.5%. Soils at this location were collected and measured to a depth of 10 cm below the soil surface. Research was located on a 'Tifway 419' [*C. dactylon* x *C. trasvaalensis* (L.) Pers.] bermudagrass fairway with a history of severe SDS symptoms and maintained at a height of 0.6 cm. All experimental areas were mowed 24 hrs before fungicide application and twice weekly thereafter. No preemergence herbicides were applied to the research plots during the spring to encourage recovery from SDS through the summer.

Plots measured 1.5 x 1.5 m and were arranged in a 2 x 4 factorial split plot design with four replications. Cultivation treatment was the main plot and fungicide treatment was the sub-plot. Cultivation treatments were conducted just prior to fungicide applications and consisted of no cultivation, verticutting, hollow-tine aerification, and aerification + verticutting. Verticutting to a depth of 1.9 cm was conducted in two perpendicular directions with a walk-behind vertical mower (Turf Doctor; Allen Seago International, Inc., Hobe Sound, FL 33475) with vertical blades spaced 2.5 cm apart.

Hollow-tine aerification to a depth of 5.1 cm was performed with a ride-on aerator (Stand-AER™ SA-25 aerator; Schiller Grounds Care, Inc., Johnson Creek, WI 53038) equipped with 1.3 cm diameter tines on 6.4 x 6.4 cm spacing.

Fungicide treatments were applied using a CO_2 powered backpack sprayer equipped with XR8008VS nozzle tips (Teejet; Spraying Systems Co., Wheaton, IL 60129) calibrated to deliver 757 L ha^{-1} at 221 kPa. Initial fungicide applications were made on August 19, 2010 and August 29, 2011 and consisted of fenarimol at 2.29 kg ai ha^{-1} or tebuconazole at 0.87 kg ai ha^{-1}. Fenarimol (pyrimidine) and tebuconazole (triazole) control susceptible fungal pathogens through the inhibition of DeMethylation sterol biosynthesis (Anonymous 2012a). All treatments received a sequential fungicide application 30 days after initial treatment. Cultivation treatments were not conducted prior to sequential fungicide applications. Irrigation (0.6 cm) was applied immediately following all fungicide treatments to increase infiltration into the soil profile.

Data collected included % bermudagrass phytotoxicity and % SDS incidence. Bermudagrass phytotoxicity was visually assessed 1 and 2 weeks following each fungicide application on a percent scale relative to the non-treated control, where 0% equaled no bermudagrass injury and 100% equaled complete bermudagrass death. Percent SDS incidence was visually assessed on a percent scale relative to the non-treated control, where 0% equaled no SDS incidence and 100% equaled complete SDS incidence. Plots were evaluated in April and June of the following year for each trial.

Percent bermudagrass phytotoxicity and % SDS incidence data were arcsine square-root transformed to stabilize variance as described by Ahrens et al. (1990) prior to being subjected to analysis of variance in SAS (version 9.3; SAS Institute, Cary, NC

27513) using error partitioning appropriate to a split plot analysis in the general linear models procedure. Interpretations were not different from non-transformed data; therefore, non-transformed means are presented for clarity. No significant year-by-treatment interactions were detected in the data; thus, data from each year were combined. All data were subjected to analysis of variance in SAS using the appropriate expected mean square values described by McIntosh (1983). Treatment means were separated using Fisher's protected least significant difference (LSD) test at $\alpha = 0.05$.

Results and Discussion

Significant cultivation ($F=156.2$, $P < 0.001$) and fungicide treatment ($F = 267.3$, $P < 0.001$) main effects as well as an interaction between cultivation and fungicide treatments ($F = 17.4$, $P < 0.001$) were observed for SDS incidence. No bermudagrass phytotoxicity was observed in response to fungicide applications throughout the length of either trial (data not shown). Cultivation treatments that caused the greatest disturbance to the upper rootzone (i.e. aerification) significantly reduced SDS incidence by April compared to the non-treated control (no cultivation), regardless of fungicide treatment (Table 3.1). No cultivation treatments resulted in 12 to 15% SDS incidence by April, regardless of fungicide treatment. Spring dead spot incidence was 1 to 7% and 2 to 7% by April in response to aerification alone and aerification + verticutting, respectively. Similar reductions in the number of dollar spot patches (9) were observed when benomyl (3 kg ai ha^{-1}) was applied 1 day after hollow-tine aerification compared to benomyl alone (48) and the non-treated control (103) (Liu et al. 1995). Verticutting alone did not significantly reduce SDS incidence in April compared to the non-treated control

regardless of fungicide treatment. Similarly, Tisserat and Fry (1997) reported that aerification alone and aerification + verticutting reduced SDS (9 and 10% incidence, respectively) when conducted twice a year compared to verticutting alone (14% incidence). However, this research only evaluated SDS control in response to cultivation techniques and not the combination of cultivation + fungicides. Verticutting alone may not disrupt the soil profile enough to reduce thick thatch layers (> 1.3 cm). Therefore, fungicide infiltration may have been limited. Tebuconazole at 0.87 kg ai ha^{-1} and fenarimol at 2.29 kg ai ha^{-1} applied alone resulted in 12 and 15% SDS incidence by April. Comparably, Butler and Tredway (2006) observed SDS reductions of 66 to 89% in response to sequential fall applications of fenarimol at 1.5 or 2.3 kg ai ha^{-1}, while McCarty et al. (1992) observed a 66% reduction in SDS incidence in response to fall applications of fenarimol at 1.5, 2.3, and 3.0 kg ai ha^{-1}. In addition, Walker (2009) observed SDS severity ratings of 3.63, 3.75, and 3.88 in response to sequential applications (1 spring and 2 fall, 3 fall, and 4 fall, respectively) of tebuconazole (3.1 kg ha^{-1}) compared to the non-treated control (1.00).

In June, cultivation treatments that included aerification significantly reduced SDS incidence compared to the non-treated control regardless of fungicide treatment (Table 3.2). No cultivation treatments resulted in 8 to 10% SDS incidence by June, regardless of fungicide treatment. Spring dead spot incidence was 0 to 5% and 0 to 4% by June in response to aerification alone and aerification + verticutting, respectively. In June, verticutting alone did not significantly reduce SDS incidence compared to the non-treated control, regardless of fungicide treatment.

Aggressive cultivation treatments that included aerification enhanced the efficacy of tebuconazole more than fenarimol for the control of SDS, regardless of rating date. Bermudagrass exhibited 1 to 2% SDS incidence by April in response to tebuconazole (0.87 kg ai ha^{-1}) following aerification or aerification + verticutting (Table 3.1). Fenarimol (2.29 kg ai ha^{-1}) in combination with aerification or aerification + verticutting resulted in 7% SDS incidence by April. A similar trend in SDS control was observed by June. Tebuconazole (0.87 kg ai ha^{-1}) following aerification or aerification + verticutting completely controlled SDS by June, while fenarimol (2.29 kg ai ha^{-1}) in combination with aerification or aerification + verticutting resulted in 4 to 5% SDS incidence (Table 3.2). Tebuconazole and fenarimol fungicides exhibit high K_{oc} values (803-1251 and 757, respectively), so infiltration into the rootzone may have been limited.

Aerification practices open up the turfgrass canopy helping reduce barriers (ex. thatch layer) that may impede fungicide penetration through the soil profile. Enhanced long-term control of SDS with the combination of fungicides and cultivation practices may reduce pesticide load and overreliance on chemical control alone. Furthermore, the coordination of aerification practices with fungicide applications may reduce labor costs and maintenance hours.

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Table 3.1. Visual ratings of % SDS[a] incidence in April of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX in response to fungicide and cultivation treatments.

Cultivation Treatment[b]	SDS Incidence (%)[c]	
	Fenarimol[d]	Tebuconazole
No Cultivation	15 aA[e]	12 aA
Verticutting	13 aA	10 aA
Aerification	7 bA	1 bB
Aerification + Verticutting	7 bA	2 bB

[a] Abbreviations: spring dead spot, SDS.
[b] Cultivation treatments were applied once prior to initial fungicide applications.
[c] Treatment means are combined from four replication experiments conducted in both years.
[d] Fenarimol was applied at 2.29 kg ai ha^{-1}. Tebuconazole was applied at 0.87 kg ai ha^{-1}. Sequential fungicide applications were made 30 days after initial treatment.
[e] Means within both columns followed by the same lowercase letter are not significantly different at $P \leq 0.05$, according to Fisher's Protected LSD test. Means within both rows followed by the same uppercase letter are not significantly different at $P \leq 0.05$ according to Fisher's Protected LSD test.

Table 3.2. Visual ratings of % SDS[a] incidence in June of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX in response to fungicide and cultivation treatments.

Cultivation Treatment[b]	SDS Incidence (%)[c]	
	Fenarimol[d]	Tebuconazole
No Cultivation	10 aA[e]	8 aA
Verticutting	10 aA	7 aA
Aerification	5 bA	0 bB
Aerification + Verticutting	4 bA	0 bB

[a] Abbreviations: spring dead spot, SDS.
[b] Cultivation treatments were applied once prior to initial fungicide applications.
[c] Treatment means are combined from four replication experiments conducted in both years.
[d] Fenarimol was applied at 2.29 kg ai ha^{-1}. Tebuconazole was applied at 0.87 kg ai ha^{-1}. Sequential fungicide applications were made 30 days after initial treatment.
[e] Means within both columns followed by the same lowercase letter are not significantly different at $P \leq 0.05$, according to Fisher's Protected LSD test. Means within both rows followed by the same uppercase letter are not significantly different at $P \leq 0.05$ according to Fisher's Protected LSD test.

Chapter IV

Efficacy of Fungicide Application Timings and New Fungicide Chemistries for the Control of Spring Dead Spot in Bermudagrass

Abstract

The inability to determine colonization timing has led to the erratic control of spring dead spot (SDS) with labeled fungicides. Furthermore, several new fungicides have recently been introduced to the market that may exhibit efficacy for the control of SDS in bermudagrass turf. Field experiments were conducted during the fall of 2010 and 2011 at Hillcrest Country Club in Lubbock, TX to examine the efficacy of fungicide application timings and newer fungicide chemistries on SDS control. Plots were arranged in a 2 x 8 factorial within a randomized complete block design with four replications. The main factor was application timing [July followed by (fb) Aug. and Aug. fb September] and the sub-factor was fungicide treatment. Fungicide treatments consisted of fenarimol at 2.29 kg ai ha^{-1}, thiophanate-methyl at 7.62 kg ai ha^{-1}, fenarimol + thiophanate-methyl at 2.29 kg ai ha^{-1} + 7.62 kg ai ha^{-1}, fluoxastrobin at 0.43 kg ai ha^{-1}, tebuconazole at 0.87 kg ai ha^{-1}, azoxystrobin at 0.12 kg ai ha^{-1}, and azoxystrobin + propiconazole at 0.99 kg ai ha^{-1}. Initial fungicide applications were made on July 22, 2010 and July 28, 2011 or Aug. 15, 2010 and Aug. 29, 2011. Sequential applications were made approximately 30 days later for all fungicide treatments. A non-treated control (water only) was sprayed at each application timing and was included for comparison. Data collected included % bermudagrass phytotoxicity and % SDS incidence. No bermudagrass phytotoxicity was observed in response to fungicide applications throughout the length of either trial. All

fungicide treatments significantly reduced SDS incidence regardless of application timing compared to the non-treated control (60 to 63% SDS incidence) by April. The July fb Aug. application timing exhibited significantly less SDS incidence than the Aug. fb Sept. application timing by April for all fungicide treatments except fluoxastrobin. Bermudagrass exhibited the least amount of SDS incidence (8%) by April in response to tebuconazole (July fb Aug. application timing). Similar levels of SDS were observed in response to July fb Aug. applications of fenarimol (10%), fenarimol + thiophanate-methyl (12%), and azoxystrobin + propiconazole (14%) by April. Thiophanate-methyl, azoxystrobin, and fluoxastrobin (July fb Aug. applications) resulted in 18 to 27% SDS incidence by April. All treatments applied in Aug. fb Sept. resulted in ≥ 17% SDS incidence by April, regardless of treatment. All fungicide treatments significantly reduced SDS incidence regardless of application timing compared to the non-treated control (28 to 33% SDS incidence) by June. The July fb Aug. application timing exhibited significantly less SDS incidence than the Aug. fb Sept. application timing by June for all fungicide treatments except fluoxastrobin and azoxystrobin + propiconazole. Bermudagrass exhibited the least amount of SDS incidence (1%) by June in response to tebuconazole (July fb Aug. application timing). Similar levels of SDS were observed in response to July fb Aug. applications of fenarimol (3%) and azoxystrobin + propiconazole (6%) by June. Fenarimol + thiophanate-methyl and thiophanate-methyl (July fb Aug. applications) resulted in 7 to 9% SDS incidence by June, while fluoxastrobin and azoxystrobin resulted in 14 to 16% SDS incidence by June. Tebuconazole, fenarimol, azoxystrobin + propiconazole, fluoxastrobin, and fenarimol +

thiophanate-methyl (Aug. fb Sept. application timing) resulted in 8 to 13% SDS incidence by June, while SDS incidence was ≥ 20% for all other treatments.

Introduction

Spring dead spot (SDS) is a soilborne disease that colonizes the roots, rhizomes, and stolons of bermudagrass (*Cynodon* spp. Rich.) managed as a home lawn, athletic field, or as golf course turf (Butler and Tredway 2007; Martin et al. 2001; Perry et al. 2010; Vann and Patton 2012; Wadsworth and Young 1960). Infections from three ectotrophic root-infecting fungal pathogens: *Ophiosphaerella narmari*, *O. korrae*, and *O. herpotricha* appear as well-defined, bleached, circular patches ranging in size from a few centimeters to a meter in diameter (Baird et al. 1998; Dernoeden et al. 1991; Elliot 1995). Spring dead spot symptoms, which are most severe in the transition zone, develop in the spring as bermudagrass breaks winter dormancy and can persist well into the summer (Crahay 1988; Dernoeden et al. 1991; Fermanian et al. 1980; McCarty and Lucas 1989; Venkatasubbaiah et al. 1994). In severe cases, bermudagrass may not fully recover before the onset of winter, leaving the turf susceptible to further damage from cold temperatures (Nus and Shashikumar 1993; Pair et al. 1986). In addition, diseased areas may reappear in infected turf for several growing seasons thereafter, often increasing in size and severity (Pair et al. 1986; Tredway et al. 2008b).

Increasing bermudagrass root health and cold tolerance through cultural management has been employed by turfgrass managers in attempts to reduce SDS severity. Watering deeply and infrequently may increase bermudagrass rooting depth and biomass (Landry 2010; McCarty 2005). Furthermore, early morning irrigation

applications may reduce prolonged leaf wetness and reduce the occurrence of fungal pathogens (Duble 1978). In fall, bermudagrass mowing heights should be raised to encourage deeper root penetration and buffer plant crowns from winter desiccation (Butler and Tredway 2007; Landry 2010; Lucas 1980; McCarty 2005). Late fall nitrogen applications should be discouraged to ensure opportune bermudagrass dormancy, while phosphorus applications may encourage root growth proceeding into winter (Butler and Tredway 2007; Lucas 1980; McCarty 2005; McCarty et al. 1991).

Although cultural control practices may alleviate some disease pressure, fungicide applications have become a necessary component of an efficacious SDS control program (Walker 2009). Fungicides containing fenarimol, thiophanate-methyl, propiconazole, and myclobutanil have been utilized for the control of SDS in athletic fields, golf courses, and sod farms (McCarty et al. 1992; Tredway et al. 2008a; 2008b; Vann and Patton 2012). Butler and Tredway (2006) reported SDS reductions of 66 to 89% in response to single or sequential applications of fenarimol (1.5 or 2.3 kg ai ha^{-1}) and 51 to 52% in response to propiconazole (1.9 kg ai ha^{-1}). McCarty et al. (1992) observed 56 and 66% reductions in SDS in response to propiconazole at 2.5 kg ai ha^{-1} and fenarimol (1.5, 2.3, and 3.0 kg ai ha^{-1}), respectively. Benomyl at 12 kg ai ha^{-1}, fenarimol (1.5, 2.3, and 3 kg ai ha^{-1}), and thiophanate-methyl at 12 kg ai ha^{-1} resulted in SDS reductions of 92, 72, 52, 86, and 41%, respectively, compared to the non-treated control (McCarty et al. 1992). In addition, Tomaso-Peterson (2011) observed a reduction in SDS severity (3% SDS incidence) with applications of fenarimol (2.3 kg ai ha^{-1}) + thiophanate-methyl (6.2 kg ai ha^{-1}) compared to the non-treated control (18% SDS incidence). Luc et al. (2005) also observed a reduction in SDS severity (4%) compared to the non-treated control (23%)

when fenarimol (2.3 kg ai ha^{-1}) was applied in conjunction with thiophanate-methyl (6.2 kg ai ha^{-1}).

While several fungicides have reduced SDS in bermudagrass, few of these chemistries are labeled for use in home lawns and the level of disease control has varied greatly from year to year (Tredway et al. 2008b). Furthermore, Gowan Company recently announced that the production of Rubigan (fenarimol) will cease by December 2012, due to high manufacturing expenses and the introduction of less expensive sulfonylurea herbicides for the control of annual bluegrass (*Poa annua* L.) (Tredway 2012). As a result, several new fungicides and tank-mixtures have recently been registered for use in turf (metconazole, tebuconazole, azoxystrobin + propiconazole, and fluoxastrobin + chlorothalonil) that may be efficacious against SDS. For example, Walker (2009) observed SDS severity ratings of 3.63, 3.75, and 3.88 in response to multiple applications (1 spring and 2 fall, 3 fall, and 4 fall, respectively) of tebuconazole (3.1 kg ha^{-1}) compared to the non-treated control (1.00). Spring dead spot disease severity was rated on a scale of 1 (no living plants present within the SDS patch) to 6 (no evidence of SDS symptoms in turfgrass) (Walker 2009).

The colonization timing of SDS is poorly understood (Tredway et al. 2008a). Butler and Tredway (2007) and Lucas (1980) reported that disease colonization occurs primarily in the fall, while Walker et al. (2009) observed colonization in spring and fall. Butler and Tredway (2006) evaluated single and sequential applications of fenarimol (1.5 to 2.3 kg ai ha^{-1}) in late summer and/or fall for the control of SDS, but reported no significant differences between application timings. Tomaso-Peterson (2011) compared spring and fall applications of fenarimol, fenarimol + thiophanate-methyl, propiconazole,

azoxystrobin, and myclobutanil for the control of SDS in a 'Tifway' bermudagrass fairway. All application timings of fenarimol and propiconazole significantly reduced SDS incidence (≤ 3.0%) compared to the non-treated control (18%); however, treatments were not significantly different from one another (Tomaso-Peterson 2011). Furthermore, spring only applications (March, April, and May) of fenarimol were equally effective in reducing SDS severity as fall only applications of all other fungicide chemistries (Tomaso-Peterson 2011). Minimal research has been conducted on the efficacy of several new fungicides for the control of SDS and no research has evaluated the efficacy of summer fungicide applications. Therefore, the objectives of this study were to evaluate the efficacy of several new fungicides and the efficacy of summer and fall fungicide applications for the control of SDS in a bermudagrass fairway.

Materials and Methods

Field experiments were conducted during the fall of 2010 and 2011 at Hillcrest Country Club in Lubbock, TX on an Arch fine sandy loam (fine-loamy, carbonatic, thermic Aridic Calciustepts) with a pH of 8.2 and an organic matter content of 1.5%. Soils at this location were collected and measured to a depth of 10 cm below the soil surface. Research was located on a 'Tifway 419' [*C. dactylon* x *C. trasvaalensis* (L.) Pers.] bermudagrass fairway with a history of severe SDS symptoms and maintained at a height of 0.6 cm. All experimental areas were mowed 24 hrs before fungicide application and twice weekly thereafter. No preemergence herbicides were applied to the research plots during the spring to encourage recovery from SDS through the summer.

Plots measuring 1.5 x 1.5 m were arranged in a 2 x 8 factorial within a randomized complete block design with four replications. The main factor was application timing [July followed by (fb) Aug. and Aug. fb September] and the sub-factor was fungicide treatment. Fungicide treatments consisted of fenarimol (Rubigan; Gowan Company, Yuma, AZ 85364) at 2.29 kg ai ha^{-1}, thiophanate-methyl (Cleary's 3336; Cleary Chemical, Dayton, NJ 08810) at 7.62 kg ai ha^{-1}, fenarimol at 2.29 kg ai ha^{-1} + thiophanate-methyl at 7.62 kg ai ha^{-1}, fluoxastrobin (Disarm; Arysta LifeScience, Cary, NC 27513) at 0.43 kg ai ha^{-1}, tebuconazole (Torque; Cleary Chemical, Dayton, NJ 08810) at 0.87 kg ai ha^{-1}, azoxystrobin (Heritage; Syngenta Crop Protection, Inc., Greensboro, NC 27419) at 0.12 kg ai ha^{-1}, and azoxystrobin + propiconazole (Headway; Syngenta Crop Protection, Inc., Greensboro, NC 27419) at 0.99 kg ai ha^{-1} (Table 4.1). Fenarimol (pyrimidine), tebuconazole, and propiconazole (triazoles) inhibit DeMethylation biosynthesis, while azoxystrobin (methoxy-acrylates) and fluoxastrobin (dihydro-dioxazines) inhibit respiration, and thiophanate-methyl (thiophanate) inhibits mitosis and cell division for the control of susceptible fungal pathogens (Anonymous 2012). Fungicide treatments were applied using a CO_2 powered backpack sprayer equipped with XR8008VS nozzle tips (Teejet; Spraying Systems Co., Wheaton, IL 60129) calibrated to deliver 757 L ha^{-1} at 221 kPa. Initial fungicide applications were made on July 22, 2010 and July 28, 2011 or Aug. 15, 2010 and Aug. 29, 2011. All treatments received a sequential application approximately 30 days after initial treatment (DAIT). A non-treated control (water only) was sprayed at each application timing and was included for comparison. Irrigation (0.6 cm) was applied immediately following all fungicide treatments to increase infiltration into the soil profile.

Data collected included bermudagrass phytotoxicity and % SDS incidence. Bermudagrass phytotoxicity was visually assessed 1 and 2 weeks following each fungicide application on a percent scale relative to the non-treated control, where 0% equaled no bermudagrass injury and 100% equaled complete bermudagrass death. Percent SDS incidence was visually assessed on a percent scale relative to the non-treated control, where 0% equaled no SDS incidence and 100% equaled complete SDS incidence. Plots were evaluated in April and June of the following year at each location.

Bermudagrass phytotoxicity and % SDS incidence data were arcsine square-root transformed to stabilize variance as described by Ahrens et al. (1990) prior to being subjected to analysis of variance in SAS (version 9.3; SAS Institute, Cary, NC 27513) using error partitioning appropriate to a split plot analysis in the general linear models procedure. Interpretations were not different from non-transformed data; therefore, non-transformed means are presented for clarity. No significant year-by-treatment interactions were detected in the data; thus, data from each year were combined. All data were subjected to analysis of variance in SAS using the appropriate expected mean square values described by McIntosh (1983). Treatment means were separated using Fisher's protected least significant difference (LSD) test at $\alpha = 0.05$.

Results and Discussion

Significant application timing ($F = 235.6$, $P < 0.001$) and fungicide treatment ($F = 190.8$, $P < 0.001$) main effects as well as an interaction between application timing and fungicide treatment ($F = 36.9$, $P < 0.001$) were observed for SDS incidence. No bermudagrass phytotoxicity was observed in response to fungicide applications

throughout the length of either trial (data not shown). All fungicide treatments significantly reduced SDS incidence regardless of application timing compared to the non-treated control (water only) (60 to 63% SDS incidence) by April (Table 4.2). The July fb Aug. application timing exhibited significantly less SDS incidence than the Aug. fb Sept. application timing by April for all fungicide treatments except fluoxastrobin. Bermudagrass exhibited the least amount of SDS incidence (8%) by April in response to tebuconazole (July fb Aug. application timing). Similar SDS incidence was observed in response to July fb Aug. applications of fenarimol (10%), fenarimol + thiophanate-methyl (12%), and azoxystrobin + propiconazole (14%) by April. Comparably, Walker (2009) observed SDS severity ratings of 3.63, 3.75, and 3.88 in response to sequential applications (1 spring and 2 fall, 3 fall, and 4 fall, respectively) of tebuconazole (3.1 kg ha^{-1}) compared to the non-treated control (1.00). In addition, Butler and Tredway (2006) and McCarty et al. (1992) observed 66 to 89% reductions in SDS in response to sequential fall applications of fenarimol at 1.5 to 3 kg ai ha^{-1}. Tomaso-Peterson (2011) observed less SDS incidence (3%) in response to fenarimol (2.3 kg ai ha^{-1}) + thiophanate-methyl (6.2 kg ai ha^{-1}) compared to the non-treated control (18%); however, fungicide applications were made in September with no sequential applications. Thiophanate-methyl, azoxystrobin, and fluoxastrobin (July fb Aug. applications) resulted in 18 to 27% SDS incidence by April. Soika et al. (2008) observed SDS reductions of 47 and 100% in response to applications of thiophanate-methyl (4.5 and 9.1 kg ai ha^{-1}). Dissimilarly, Tomaso-Peterson (2011) observed SDS incidences of 3% when azoxystrobin (0.61 kg ai ha^{-1}) was applied to ‘Tifway’ bermudagrass; however, this rate was 5 times greater than rates examined in this research. Equivalent to the results of this trial, Tredway et al.

(2008a) observed reductions in SDS of 45% when azoxystrobin (0.12 kg ai ha^{-1}) was applied to 'Tifway' bermudagrass. All treatments applied in Aug. fb Sept. resulted in ≥ 17% SDS incidence by April, regardless of treatment (Table 4.2).

All fungicide treatments significantly reduced SDS incidence regardless of application timing compared to the non-treated control (28 to 33% SDS incidence) by June (Table 4.3). The July fb Aug. application timing exhibited significantly less SDS incidence than the Aug. fb Sept. application timing by June for all fungicide treatments except fluoxastrobin and azoxystrobin + propiconazole. Bermudagrass exhibited the least amount of SDS incidence (1%) by June in response to tebuconazole (July fb Aug. application timing). Similar SDS incidence was observed in response to July fb Aug. applications of fenarimol (3%) and azoxystrobin + propiconazole (6%) by June. Fenarimol + thiophanate-methyl and thiophanate-methyl (July fb Aug. applications) resulted in 7 to 9% SDS incidence by June, while fluoxastrobin and azoxystrobin resulted in 14 to 16% SDS incidence. Tebuconazole, fenarimol, azoxystrobin + propiconazole, fluoxastrobin, and fenarimol + thiophanate-methyl (Aug. fb Sept. application timing) resulted in 8 to 13% SDS incidence by June, while SDS incidence was ≥ 20% for all other treatments.

Applications in July fb Aug. resulted in significantly less SDS incidence by April than later applications (Aug. fb Sept.) for all fungicide treatments except fluoxastrobin (0.43 kg ai ha^{-1}) (Table 4.2). Spring dead spot colonization of bermudagrass roots may occur earlier in the fall than previously described. Walker (2009) reported that *Ophiosphaerella* spp. actively infects bermudagrass roots when soil temperatures drop below 20°C. Although root lesion length was greatest (13.1 mm) on 'Jackpot'

bermudagrass at 17ºC, similar lesion lengths (8.6 to 11.1 mm) were observed between 12 and 21ºC (Walker et al. 2006). Furthermore, although smaller in size, lesions (2.6 to 3.0 mm) were observed on roots subjected to 25 to 30ºC (Walker et al. 2006). In the field, no differences in SDS colonization were observed in 2003 when disease patches were sampled in July, October, and November, but higher root colonization was observed in October and November in 2004 (Walker et al. 2006). If colonization occurs under warmer temperatures, then traditional fungicide applications made in September and October would be ineffective, because colonization would occur before fungicides were delivered to the rootzone. Butler and Tredway (2006) evaluated single and sequential applications of fenarimol (1.5 to 2.3 kg ai ha^{-1}) in late summer (Aug.) and/or fall (Oct. and Nov.) for the control of SDS, but reported no significant differences in control between application timings. Similarly, Tomaso-Peterson (2011) compared spring (March, April, and May) and fall (Sept., Oct., and Nov.) applications of fenarimol (2.3 kg ai ha^{-1}), fenarimol + thiophanate-methyl (2.3 and 6.2 kg ai ha^{-1}), propiconazole (1.9 kg ai ha^{-1}) and azoxystrobin (0.61 kg ai ha^{-1}), but observed no significant differences in control between application timings.

The ability of *Ophiosphaerella* spp. to infect bermudagrass during a wider timeframe than previously determined may require further investigation into fungicide application timings and sequential applications. Fungicide applications made in July fb Aug. were most efficacious for the control of SDS in this study compared to applications made in Aug. fb Sept. The onset of a milder fall/winter (prolonging periods of infection) following summer fungicide applications may limit SDS control due to reduced soil residual activity during extended months of SDS colonization. Temperature data (not

shown) indicated that air temperatures in Lubbock, TX averaged 18°C from July 1 to January 1 for both 2010 and 2011. Average air temperature was collected using a Campbell Scientific weather station (Campbell Scientific Inc., Logan, UT 84341) at the Texas Tech University Quaker Avenue Research Farm in Lubbock, TX. The loss of fenarimol may further complicate SDS control; however, results from this study indicate that tebuconazole and azoxystrobin + propiconazole may provide equivalent control.

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Table 4.1. Fungicide treatments and application dates at Hillcrest Country Club in Lubbock, TX in 2010 and 2011.

Treatment	Rate	Application Dates	
	—kg ai ha^{-1}—	——2010——	——2011——
Non-treated Control	——	July 22 + Aug. 15	July 28 + Aug. 29
Fenarimol	2.29	July 22 + Aug. 15	July 28 + Aug. 29
Thiophanate-Methyl	7.62	July 22 + Aug. 15	July 28 + Aug. 29
Fenarimol + TM[a]	2.29 + 7.62	July 22 + Aug. 15	July 28 + Aug. 29
Fluoxastrobin	0.43	July 22 + Aug. 15	July 28 + Aug 29
Tebuconazole	0.87	July 22 + Aug. 15	July 28 + Aug. 29
Azoxystrobin	0.12	July 22 + Aug. 15	July 28 + Aug. 29
Azoxystrobin + Prop[b]	0.99	July 22 + Aug. 15	July 28 + Aug. 29
Non-treated Control	——	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Fenarimol	2.29	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Thiophanate-Methyl	7.62	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Fenarimol + TM	2.29 + 7.62	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Fluoxastrobin	0.43	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Tebuconazole	0.87	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Azoxystrobin	0.12	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30
Azoxystrobin + Prop	0.99	Aug. 15 + Sept. 15	Aug. 29 + Sept. 30

[a] Abbreviations: thiophanate-methyl, TM; propiconazole, Prop.
[b] Azoxystrobin + propiconazole is a prepackaged mixture (Headway; Syngenta Crop Protection, Inc., Greensboro, NC 27419).

Table 4.2. Visual ratings of % SDS[a] incidence in April of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX in response to fungicide application timings and new fungicide chemistries.

Treatment	Rate	SDS Incidence (%)[b]	
		Application Timing	
	—kg ai ha^{-1}—	July fb Aug.	Aug. fb Sept.
Non-treated Control	——	63 aA[c]	60 aA
Fenarimol	2.29	10 cdA	19 cdB
Thiophanate-Methyl	7.62	18 bcA	34 bB
Fenarimol + TM	2.29 + 7.62	12 cdA	21 cdB
Fluoxastrobin	0.43	27 bA	25 cA
Tebuconazole	0.87	8 dA	17 dB
Azoxystrobin	0.12	25 bA	40 bB
Azoxystrobin + Prop[d]	0.99	14 cdA	26 cB

[a] Abbreviations: spring dead spot, SDS; followed by, fb; thiophanate-methyl, TM; propiconazole, Prop.

[b] Treatment means are combined from four replication experiments conducted over two separate years.

[c] Means within both columns followed by the same lowercase letter are not significantly different at $P \leq 0.05$, according to Fisher's Protected LSD test. Means within both rows followed by the same uppercase letter are not significantly different at $P \leq 0.05$ according to Fisher's Protected LSD test.

[d] Azoxystrobin + propiconazole is a prepackaged mixture (Headway; Syngenta Crop Protection, Inc., Greensboro, NC 27419).

Table 4.3. Visual ratings of % SDS[a] incidence in June of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX in response to fungicide application timings and new fungicide chemistries.

Treatment	Rate	SDS Incidence (%)[b]	
		Application Timing	
	—kg ai ha^{-1}—	July fb Aug.	Aug. fb Sept.
Non-treated Control	—	28 aA[c]	33 aA
Fenarimol	2.29	3 efA	10 cB
Thiophanate-Methyl	7.62	9 cdA	20 bB
Fenarimol + TM	2.29 + 7.62	7 deA	13 cB
Fluoxastrobin	0.43	14 bcA	11 cA
Tebuconazole	0.87	1 fA	8 cB
Azoxystrobin	0.12	16 bA	26 bB
Azoxystrobin + Prop[d]	0.99	6 defA	10 cA

[a] Abbreviations: spring dead spot, SDS; followed by, fb; thiophanate-methyl, TM; propiconazole, Prop.

[b] Treatment means are combined from four replication experiments conducted over two separate years.

[c] Means within both columns followed by the same lowercase letter are not significantly different at $P \leq 0.05$, according to Fisher's Protected LSD test. Means within both rows followed by the same uppercase letter are not significantly different at $P \leq 0.05$ according to Fisher's Protected LSD test.

[d] Azoxystrobin + propiconazole is a prepackaged mixture (Headway; Syngenta Crop Protection, Inc., Greensboro, NC 27419).

Chapter V

Effect of Spring Preemergence Herbicide Applications on the Recovery of Bermudagrass from Spring Dead Spot

Abstract

Turfgrass managers often make preemergence (PRE) herbicide applications in early spring for annual grass and broadleaf weed control in bermudagrass fairways. These applications may also coincide with the appearance of spring dead spot (SDS) disease symptoms. Utilization of certain PRE herbicides may delay bermudagrass recovery from SDS and further contribute to a weakened turfgrass system. Field experiments were conducted during spring and summer of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX to examine bermudagrass recovery from SDS in response to PRE herbicide applications. Initial PRE herbicide applications were made on March 15, 2011 and March 23, 2012 to a 'Tifway 419' bermudagrass fairway exhibiting symptoms of SDS. Herbicide treatments consisted of prodiamine at 0.73 kg ai ha^{-1}, pendimethalin at 2.5 kg ai ha^{-1}, oryzalin at 1.68 kg ai ha^{-1}, dithiopyr at 0.56 kg ai ha^{-1}, oxadiazon at 3.4 kg ai ha^{-1}, indaziflam at 0.035 kg ai ha^{-1}, and dimethenamid at 1.68 kg ai ha^{-1}. A non-treated control was included for comparison. Data collected included % bermudagrass phytotoxicity and % SDS incidence. Percent SDS incidence was converted to % bermudagrass recovery by comparing % SDS incidence ratings back to initial ratings recorded in April. No bermudagrass phytotoxicity was observed in response to preemergence herbicide applications throughout the length of either trial. In 2011 and 2012 bermudagrass in the non-treated control plots exhibited similar recovery from SDS (46 and 59%, respectively)

as bermudagrass in response to oxadiazon at 3.4 kg ai ha^{-1} (55 and 57%, respectively) 14 weeks after treatment (WAT). Pendimethalin at 2.5 kg ai ha^{-1} resulted in 33 to 38% bermudagrass recovery from SDS 14 WAT, regardless of year. In 2011, applications of oryzalin, dithiopyr, prodiamine, and dimethenamid resulted in 21 to 25% bermudagrass recovery from SDS 14 WAT. In 2012, bermudagrass exhibited similar recovery in response to prodiamine and oryzalin (24 and 26%, respectively), while response to dithiopyr and dimethenamid was 33 and 34%, respectively, 14 WAT. Bermudagrass exhibited the least amount of recovery (13 to 14%) in response to indaziflam at 0.035 kg ai ha^{-1}, regardless of year. Preemergence herbicide selection is important when a history of SDS pressure has been previously observed on the turfgrass site in question. Use of oxadiazon as a PRE option will have no effect on SDS recovery, while the use of oryzalin, prodiamine, and indaziflam may limit recovery and further contribute to a weakened turfgrass system.

Introduction

Spring Dead Spot (SDS) is a soilborne disease caused by three closely related ectotrophic, root-infecting pathogens: *Ophiosphaerella narmari, O. korrae,* and *O. herpotricha* (Baird et al. 1998; Elliot 1995). Although bermudagrass (*Cynodon* spp. Rich.) is the primary host of SDS, it has also been reported to infect buffalograss (*Bouteloua dactyloides* (Nutt.) J.T. Columbus) and zoysiagrass (*Zoysia* spp. Willd.) (Tisserat et al. 1999; Tredway and Butler 2007). Spring dead spot symptoms may occur on intensely managed bermudagrass home lawns, athletic fields, golf courses, and sod farms located in the transition zone (Butler and Tredway 2007; Martin et al. 2001; Perry

et al. 2010; Vann and Patton 2012; Wadsworth and Young 1960). Disease symptoms become noticeable when bermudagrass breaks winter dormancy in the spring, but disease injury can often persist well into the summer (Dernoeden et al. 1991; Fermanian et al. 1980; McCarty and Lucas 1989; Venkatasubbaiah et al. 1994). Symptoms appear as well-defined, bleached, circular patches that range in size from a few centimeters to a meter in diameter (Baird et al. 1998; Dernoeden et al. 1991). Dark ectotrophic runner hyphae cover the surface of rhizomes, stolons, and roots of infected plants making them appear blackened and necrotic (McCarty et al. 1991; Tredway et al. 2008; Vincelli and Williams 1998a). Turfgrass plants within the disease patches eventually collapse to the ground leaving behind sunken necrotic areas which may allow for the germination of weeds; thus, causing further decline in aesthetic quality and playability of turf (Fermanian et al. 1980; Iriarte et al. 2004; Walker 2009). In addition, infected turfgrass may not fully recover from SDS symptoms before the onset of winter dormancy, leaving the turf susceptible to further damage from freezing temperatures (Nus and Shashikumar 1993; Pair et al. 1986).

Bermudagrass is a versatile, warm-season perennial grass species that is often utilized for forage, turfgrass, and soil stabilization throughout tropical and subtropical regions of the world (Christians 2011; McCarty 2005; Zhang et al. 2006). It is widely used on golf courses in the southern U.S. due to aggressive growth and high recuperative capacity following traffic/wear (Beard 1973; Goatley et al. 2003; McCarty 2005). Recent improvements in the cold tolerance of several new bermudagrass cultivars have extended its use further north (Anderson et al. 2002; Gatschet et al. 1994). Although less tolerant of cold temperatures than zoysiagrass and buffalograss, bermudagrass may be successfully

cultivated in the southern and central regions of the transition zone (Anderson et al. 2002; McCarty 2005). However, bermudagrass often experiences desiccation (winter-kill) in the northern transition zone where temperatures may drop below -7°C (Beard 1973; McCarty 2005; Turgeon 2011).

Preemergence (PRE) herbicides are often applied to turfgrass stands in early spring for the control of annual grasses and broadleaf weeds (Bhowmik and Bingham 1990; McCullough et al. 2007; Turgeon 2011). However, certain PREs may have a negative impact on the growth of bermudagrass turf (Bingham 1967; Fishel and Coats 1993; Kaminski and Dernoeden 2004). The use of dinitroaniline (DNA) herbicides, such as prodiamine and pendimethalin, are capable of causing abnormal swelling of turfgrass root tips ('clubbed-roots'), stunting of lateral root growth, and/or severely pruned roots when healthy tissue comes into contact with the chemical barrier created in the upper soil profile (Fishel and Coats 1993; Mitra and Bhowmik 2005). Fishel and Coats (1994) and McCullough et al. (2007) observed noticeable reductions in growth, as well as abnormal root development, in bermudagrass plants treated with pendimethalin, prodiamine, and dithiopyr. The DNA herbicide family exhibits low water solubility and readily binds to soil particles, so these chemicals tend to remain near the soil surface and do not leach through the soil profile (Fishel and Coats 1994). Therefore, the chemical barrier may remain intact for several weeks to months, until soil microorganisms and other environmental factors degrade these herbicides (Capo-chichi et al. 2005; Goatley et al. 2003). As a result, utilization of certain PRE herbicides may interfere with the recovery of bermudagrass in spring and summer from damage caused by winter desiccation, heavy traffic, or disease (Brosnan et al. 2011a; Kaminski et al. 2004).

Although several cultural and chemical practices exist for the reduction of SDS in bermudagrass turf, no management option has provided complete control (Vincelli and Williams 1998a; 1998b; 2012). In spring, turfgrass managers often change their focus from control of SDS to recovery of bermudagrass from disease symptoms (Vincelli and Williams 2012). Turf in the center of disease patches is necrotic, so recovery requires encroachment of bermudagrass stolons from surrounding, healthy plants (Butler and Tredway 2007; Tredway et al. 2008; Vincelli and Williams 1998a). The presence of PRE herbicides in the soil profile may inhibit the rooting of bermudagrass stolons; thus, prolonging disease symptoms and recovery. As a result, bermudagrass may not recover before the onset of winter, which may lead to further damage from freezing temperatures (Nus and Shashikumar 1993; Pair et al. 1986). Though research has been conducted to quantify the effect of PRE herbicide applications on bermudagrass recovery from divot injury (Cooper and Henry 2010) and wear/traffic (Brosnan et al. 2011a), no research has been conducted to determine the effect on bermudagrass recovery from SDS. Therefore, the objective of this study was to evaluate the effect of spring PRE herbicide applications on the recovery of bermudagrass from SDS symptoms.

Materials and Methods

Field experiments were conducted during spring and summer of 2011 and 2012 at Hillcrest Country Club in Lubbock, TX on an Arch fine sandy loam (fine-loamy, carbonatic, thermic Aridic Calciustepts) soil with a pH of 8.2 and an organic matter content of 1.5%. Soils at this location were collected and measured to a depth of 10 cm below the soil surface. Research was located on a ‘Tifway 419’ [*C. dactylon* x *C.*

trasvaalensis (L.) Pers.] bermudagrass fairway with a history of severe SDS symptoms and maintained at a height of 0.6 cm. Plots measured 1.5 x 1.5 m and were arranged in a randomized complete block design with four replications. All experimental areas were mowed 24 hrs before herbicide application and twice weekly thereafter. No fungicides were applied to the research area the previous spring in order to encourage SDS infestation.

Preemergence herbicide treatments included prodiamine (Barricade; Syngenta Crop Protection, LLC., Greensboro, NC 18300) at 0.73 kg ai ha^{-1}, pendimethalin (Pendulum; BASF Corporation, Research Triangle Park, NC 27709) at 2.5 kg ai ha^{-1}, oryzalin (Surflan, United Phosphorus, Inc., King of Prussia, PA 19406) at 1.68 kg ai ha^{-1}, dithiopyr (Dimension; Dow AgroSciences, LLC., Indianapolis, IN 46268) at 0.56 kg ai ha^{-1}, oxadiazon (Ronstar, Bayer Environmental Science, Research Triangle Park, NC 27709) at 3.4 kg ai ha^{-1}, indaziflam (Specticle; Bayer Environmental Science, Research Triangle Park, NC 27709) at 0.035 kg ai ha^{-1}, and dimethenamid (Tower; BASF Corporation, Research Triangle Park, NC 27709) at 1.68 kg ai ha^{-1} (Table 5.1). PRE herbicide treatment rates were chosen based on label recommendations for each herbicide. Herbicide treatments were applied with a CO_2-powered backpack sprayer equipped with XR8003VS nozzle tips (Teejet; Spraying Systems Co., Wheaton, IL 60129) calibrated to deliver 305 L ha^{-1} at 276 kPa. Herbicide applications were made on March 15, 2011 and March 23, 2012. A non-treated control was included for comparison. Irrigation (0.6 cm) was applied immediately following all preemergence herbicide treatments to increase infiltration into the soil profile and activate the herbicides.

Data collected included % bermudagrass phytotoxicity and % SDS incidence. Percent bermudagrass phytotoxicity was visually assessed 1 and 2 weeks following PRE herbicide application on a percent scale relative to the non-treated control, where 0% equaled no bermudagrass injury and 100% equaled complete bermudagrass death. Visual ratings of % SDS incidence were recorded every two weeks following initial occurrence of SDS symptoms in April on a scale of 0% (no SDS incidence) to 100% (complete SDS incidence) until mid-June [14 weeks after treatment (WAT)]. Spring dead spot incidence was converted to % bermudagrass recovery by comparing SDS incidence ratings recorded in June back to initial SDS incidence ratings recorded in April.

Percent bermudagrass phytotoxicity and % bermudagrass recovery data were arcsine square-root transformed to stabilize variance as described by Ahrens et al. (1990) prior to being subjected to analysis of variance in SAS (version 9.3, SAS Institute, Cary, NC 27513), with main effects and all possible interactions tested using the appropriate expected mean square values described by McIntosh (1983). Interpretations were not different from non-transformed data; therefore, non-transformed means are presented for clarity. A significant year-by-treatment interaction was detected so data were analyzed separately. All data were subjected to analysis of variance in SAS using the appropriate expected mean square values described by McIntosh (1983). Treatment means were separated using Fisher's protected least significant difference (LSD) test at $\alpha = 0.05$.

Results and Discussion

A significant year-by-treatment interaction ($F = 9.12$, $P = 0.005$) was detected so data for each year are presented separately (Table 5.2). No bermudagrass phytotoxicity

was observed in response to preemergence herbicide applications throughout the length of either trial (data not shown). In 2011 and 2012 bermudagrass in the non-treated control plots exhibited similar recovery from SDS (46 and 59%, respectively) as bermudagrass in response to oxadiazon at 3.4 kg ai ha^{-1} (55 and 57%, respectively) 14 WAT (Table 5.2). McCullough et al. (2007) observed similar reductions in growth (2 to 15%) of 'TifEagle' bermudagrass (maintained at 4 mm) roots in response to oxadiazon at 2.2 kg ai ha^{-1} in the greenhouse 25 WAT. In addition, Fishel and Coats (1994) observed that applications of oxadiazon (1.7 and 3.4 kg ai ha^{-1}) did not reduce the number of healthy roots growing from plugs of common bermudagrass [*C. dactylon* (L.) Pers.] sod when compared to the non-treated control. Pendimethalin at 2.5 kg ai ha^{-1} in my research resulted in 33 to 38% bermudagrass recovery from SDS 14 WAT, regardless of year. McCullough et al. (2007) reported a 37% reduction in bermudagrass root mass in response to pendimethalin at 1.7 kg ai ha^{-1} 25 WAT. In TX in 2011, oryzalin, dithiopyr, prodiamine, and dimethenamid applications resulted in 21 to 25% bermudagrass recovery from SDS 14 WAT. In 2012, bermudagrass exhibited similar recovery in response to prodiamine and oryzalin (24 and 26%, respectively), while response to dithiopyr and dimethenamid was 33 and 34%, respectively, 14 WAT. Similar results were observed by Fishel and Coats (1994) when common bermudagrass sod was treated with dithiopyr (0.56 and 1.1 kg ai ha^{-1}), prodiamine (0.56 and 1.1 kg ai ha^{-1}), and pendimethalin (1.7 and 3.4 kg ai ha^{-1}). Both rates of dithiopyr and prodiamine, and pendimethalin at 3.4 kg ha^{-1} reduced roots 36 to 97%, 19 to 19%, and 38 to 70% compared to the non-treated control 4 to 8 WAT. McCullough et al. (2007) also observed that dithiopyr (0.56 kg ai ha^{-1}) significantly reduced root mass 30 to 33% when applied to 'TifEagle' bermudagrass in a greenhouse

setting 2 to 25 WAT. In addition, Fagerness et al. (2002) observed that applications of prodiamine (1.1 kg ai ha^{-1}) and dithiopyr (0.6 kg ai ha^{-1}) suppressed 'Tifway' bermudagrass establishment from sprigs (mowed at 19 mm) up to 25% 4 to 11 weeks after establishment (WAE). Bermudagrass exhibited the least amount of recovery (13 to 14%) in response to indaziflam at 0.035 kg ai ha^{-1}, regardless of year.

Bermudagrass recovery was unaffected by applications of oxadiazon at 3.4 kg ai ha^{-1}. Oxadiazon is primarily absorbed by shoots of susceptible weeds as they germinate and emerge through the herbicide treated zone of the soil profile (Wilen et al. 2011; Yelverton 1995). Recovery from SDS requires the encroachment of bermudagrass stolons from surrounding, healthy plants. Bermudagrass stolons that come into contact with oxadiazon treated soil only absorb minimal amounts of the herbicide; therefore, turfgrass rooting is not adversely affected (Wilen et al. 2011; Yelverton 1995). Dinitroaniline herbicides are absorbed by roots and shoots of susceptible weeds as they germinate and emerge through the herbicide treated zone of the soil profile (Appleby and Valverde 1989; Parka and Soper 1977). Stolons that come into contact with DNA herbicides in the soil may have difficulty rooting due to the abnormal swelling of turfgrass root tips and formation of "clubbed-roots" (Fishel and Coats 1993; Mitra and Bhowmik 2005). Brosnan et al. (2011a) did not observe differences in the recovery of stressed (wear/traffic) 'Tifway' bermudagrass in response to PRE herbicide applications. Bermudagrass recovery was similar in response to oxadiazon (3.4 kg ai ha^{-1}), pendimethalin (3.4 kg ai ha^{-1}), prodiamine (0.56 kg ai ha^{-1}), prodiamine + sulfentrazone (0.8 + 0.4 kg ai ha^{-1}), and dithiopyr (0.4 kg ha^{-1}) 1 to 7 weeks after traffic simulations ceased. However, bermudagrass in this research was allowed to recover from existing turf

(roots, rhizomes, and stolons), while recovery in my research relied on bermudagrass stolon encroachment. Cooper and Henry (2010) observed poor regrowth of 'Tifway' bermudagrass (3 and 0%) in response to applications of prodiamine (1.05 kg ai ha^{-1}) and oryzalin (1.68 kg ai ha^{-1}) compared to the non-treated control (18%) 6 weeks after initial treatment (WAIT). Similar to my research, Cooper and Henry (2010) measured bermudagrass recovery from surrounding stolon encroachment of healthy tissue.

Bermudagrass exhibited the least amount of recovery (13 to 14%) from SDS 14 WAT in response to indaziflam at 0.035 kg ai ha^{-1}. Indaziflam is an alkylazine herbicide that controls susceptible grass and broadleaf weeds through the inhibition of cellulose biosynthesis (Brosnan et al. 2011b; 2012). Although indaziflam offers turfgrass managers an alternate mode of action for the control of key turfgrass weeds (Alonso et al. 2011; Brosnan and Breeden 2012; Brosnan et al. 2011b; 2012; Henry et al. 2012), long soil residual activity (> 150 days) may limit bermudagrass recovery from stress. Henry et al. (2012) reported that perennial ryegrass (*Lolium perenne* L.) cover in plots treated with indaziflam at 0.053 kg ha^{-1} and 0.070 kg ha^{-1} measured 37% to 48% compared to 88% for the non-treated control 257 days after initial treatment. Post and Askew (2011) investigated 'Riviera' common bermudagrass root strength in response to sequential applications of indaziflam (0.035 or 0.052 kg ai ha^{-1}), indaziflam followed by oxadiazon (2.24 kg ai ha^{-1}) or prodiamine (0.84 kg ai ha^{-1}), and prodiamine (0.84 kg ai ha^{-1}). No differences in loose stolon number and root strength were observed between the non-treated control and indaziflam treatments 16 WAIT; however, bermudagrass root strength was significantly lower in response to sequential prodiamine treatments 16 WAIT (Post and Askew 2011). However, this research examined the response of four-year-old rooted

bermudagrass to indaziflam applications, while my research examined the ability of bermudagrass stolons to encroach from surrounding plants and root in indaziflam treated soil.

Differential responses in SDS bermudagrass recovery from year to year may be attributed to the severe drought conditions experienced during 2011. The Palmer drought severity index (PDSI) for the month of April and May in Texas during 2011 was -4.19 and -5.08, respectively, indicating the most severe drought since 1895 (Dawson 2011). Although research plots were irrigated, high temperatures, low humidity, and wind events during spring and summer months may have increased evaporative demand, thus reducing bermudagrass vigor and subsequent recovery from SDS symptoms.

Preemergence herbicide selection is important when a history of SDS pressure has been previously observed on the turfgrass site in question. Use of oxadiazon, as a PRE option, will have no effect on bermudagrass recovery from SDS, while the use of oryzalin, prodiamine, and indaziflam may limit recovery and further contribute to a weakened turfgrass system.

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Table 5.1. PRE[a] herbicide treatments and application dates at Hillcrest Country Club in Lubbock, TX in 2011 and 2012.

Treatment	Rate	Application Dates	
	─kg ai ha^{-1}─	──2011──	──2012──
Non-treated Control	──	March 15	March 23
Prodiamine	0.73[b]	March 15	March 23
Pendimethalin	2.5	March 15	March 23
Oryzalin	1.68	March 15	March 23
Dithiopyr	0.56	March 15	March 23
Oxadiazon	3.4	March 15	March 23
Indaziflam	0.035	March 15	March 23
Dimethenamid	1.68	March 15	March 23

[a] Abbreviations: preemergence, PRE.
[b] PRE herbicide treatment rates were chosen based on label recommendations for each herbicide.

Table 5.2. Visual ratings of % bermudagrass recovery from SDS[a] in June in response to preemergence herbicide applications made in March to a 'Tifway' bermudagrass fairway at Hillcrest Country Club in Lubbock, TX.

Treatment	Rate	Bermudagrass Recovery (%)[b]	
	kg ai ha^{-1}	6/21/2011	6/28/2012
Non-treated Control	——	46 a[c]	59 a
Prodiamine	0.73	23 cd	24 cd
Pendimethalin	2.5	33 b	38 b
Oryzalin	1.68	21 cd	26 bc
Dithiopyr	0.56	22 cd	33 bc
Oxadiazon	3.4	55 a	57 a
Indaziflam	0.035	14 d	13 d
Dimethenamid	1.68	25 bc	34 bc
$LSD_{0.05}$		9	12

[a] Abbreviations: spring dead spot, SDS; least significant difference, LSD.
[b] Visual ratings of % disease incidence was converted to % bermudagrass recovery by comparison to initial % SDS incidence recorded on 4/25/2011.
[c] Means within a column followed by the same letter are not significantly different according to Fishers LSD test (α=0.05).

Chapter VI

Conclusions

Spring dead spot (SDS) is one of the most severe and difficult to control diseases of bermudagrass. The appearance of SDS symptoms on golf courses can interfere with the playability of bermudagrass turf during spring when weather conditions are most desirable for play. The destructive nature of this disease warrants the focus of management practices that emphasize disease prevention and effective levels of control. Successful SDS management programs should emphasize an integrated pest management strategy of utilizing proper implementation of cultural practices, such as irrigation, fertility, and thatch management, to enhance bermudagrass winter hardiness through the promotion of turfgrass root health. Although cultural practices may provide some disease reduction, fungicide applications have become a necessary component of a SDS control program. However, the level of disease control is highly erratic from year to year.

This research indicated that the combination of fungicide applications in conjunction with cultural control practices may enhance fungicide efficacy against SDS. Efficacy of fenarimol and fenarimol + thiophanate-methyl was enhanced by the addition of a soil and spray deposition agent. In addition, cultivation practices (i.e. aerification) that result in severe disturbances of the upper rootzone greatly enhanced the efficacy of fenarimol and tebuconazole against SDS. The utilization of these cultural practices may have served to bypass or remove physical and physiochemical properties within the soil profile to increase infiltration and availability of fungicides into the rootzone.

The results of this research also indicated that several newly available chemistries, such as tebuconazole and azoxystrobin + propiconazole, may provide an effective replacement for SDS control in bermudagrass when fenarimol is no longer available. However, this research also indicates that the timings of initial fungicide applications may be critical to an effective SDS control strategy. The results from this research indicated that summer application timings (July fb Aug.) were more effective against SDS than fall application timings (Aug. fb Sept.).

In addition, this research indicated that PRE herbicide selection is important when a history of SDS pressure has been previously observed on a bermudagrass site. The use of oxadiazon will have no effect on bermudagrass recovery from SDS, while the use of oryzaline, prodiamine, and indaziflam may limit recovery and further contribute to a weakened turfgrass system.