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Spring Dead Spot Occurrence in Bermudagrass following Fungicide and Nutrient Applications

Lambert B. McCarty[1], Leon T. Lucas[2], and Joseph M. DiPaola[3]
College of Agriculture and Life Sciences, North Carolina State University, Raleigh, NC 27695-7616

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Abstract. **Spring dead spot (SDS) *[Gaeumannomyces graminis* (Sacc.) von Arx & D. Olivier var. *graminis* Walker] is a serious disease of** bermudagrass ***[Cynodon dactylon* (L.) Pers.] throughout much of the southern United States and is believed to be at least partially influenced by the previous year's turfgrass management practices. Research was performed to: a) determine the efficacy of selected fungicide control measures; and b) determine the influence of N and K nutrient regimes on the expression of SDS symptoms in Tifway bermudagrass (*C. dactylon* x *C. transvaalensis* Burtt-Davy). Averaged over two sites in 2 years, a 72% reduction in SDS followed a fall application of benomyl at 12 kg·ha^{-1}. Fenarimol applied at three rates (1.5, 2.3, and 3.0 kg·ha^{-1}) on three fall dates reduced SDS by a combined average of 66%. A single application of propiconazole (2.5 kg·ha^{-1}) reduced disease by an average of 56%. Application of N (98 kg·ha^{-1}) in late fall increased SDS 128% in one test location. Application of potassium sulfate (269 kg K/ha) in late fall resulted in an average increase in SDS expression of 89% the following spring over all experiments. Turf managers with severe SDS should minimize heavy late-fall K applications and possibly use benomyl, fenarimol, or propiconazole for disease suppression. Chemical names used: α -(2-chlorophenyl)- α -(4-chlorophenyl)-S-pyrimidinemethanol (fenarimol); [methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate] (benomyl); 1-[[2-(2,4-dichlorophenyl)-4-propyl-1,3-dioxolan-2-yl]methyl]--1H-1,2,4-triazole (propiconazole).**

Spring dead spot (SDS) is the most important disease of bermudagrass in the southern United States and Australia (Lucas, 1980). Several ectotrophic root fungi have been shown to cause SDS of bermudagrass. These include *Leptosphaeria narmari* J.C. Walker & A.M. Sm. and L. *korrae* J.C. Walker & A.M. Sm. in Australia (Walker and Smith, 1972), L. *korrae* in California (Endo et al., 1985) and Maryland (Crahay et al., 1988), *Ophiosphaerella herpotricha* (Fr.) Walker in Kansas (Tisserat et al., 1989b), and *Gaeumannomyces graminis* (Sacc.) von Arx & D. Olivier var. *graminis* Walker in the southern United States (McCarty and Lucas, 1989). Researchers have noted that SDS-afflicted bermudagrass has greater fall color retention than nearby healthy bermudagrass (Lucas, 1980). Turf with succulent late-season growth generally becomes more susceptible to injury from low temperatures (Beard, 1973). Whether bermudagrass injury results directly from fungal activity or indirectly from lethal low winter temperatures or a combination of these factors is unclear.

Fungicidal suppression of SDS has been documented. In the early 196Os, a chemical control program for SDS consisted of using four monthly applications of disodiumethylenebisdithiocarbamate (nabam) beginning at least 6 weeks before the first killing frost (Kozelnicky, 1974). Smith (1971), in New South Wales, Australia, reported SDS control with nabam (2.2 kg·ha^{-1}) or bisdimethyl-thiocarbamoyl disulfide (thiram) (10.5 kg·ha^{-1}) applied monthly for nine applications beginning the last month of summer. Because of the need for several applications, and often variable results in some areas, these treatments were used infrequently.

In North Carolina, SDS suppression was achieved in the spring following one application of benomyl(12.2 kg·ha^{-1}) in the previous October or November (Lucas, 1980). This treatment did not always provide complete suppression but was the most consistent of the fungicides tested. When complete suppression was not achieved, SDS patches formed in benomyl-treated areas were smaller and recovered more quickly than those in other treated or nontreated areas.

Management practices that have been associated with increased SDS severity include the use of hybrid bermudagrass cultivars, excessive N fertilizer during the growing season, and excessive thatch development (Lucas, 1980). Reduction of other soil-borne fungi following fertilizer application has been observed. Take-all patch disease, incited by *G. graminis* (Sacc.) von Arx var. *avenue* (E.M. Turner) Dennis & Olivier or G. *graminis* var. *tritici* (E.M. Turner) Dennis, has been lessened by application of ammonium chloride or ammonium sulfate (Dernoeden, 1987; Smith, 1956).

Potassium fertilization also has been shown to lessen dollar spot *(Sclerotinia homoeocarpa* F.T. Bennett) and leaf spot *[Bipolaris cynodontis* (Marig) Shoem.] in bermudagrass (Horn, 1969). Potassium can improve turf winter survival, which could be of importance given the relationship between low-temperature stress tolerance and SDS. Juska and Murray (1974) found that high (336 kg·ha^{-1}·year^{-1}) K levels increased the winter survival of some bermudagrass cultivars 3-fold.

In light of previous findings, it seems that when a root pathogen is present, coupled with low temperatures, SDS symptoms are able to develop. Symptoms appear to be most severe when a hybrid bermudagrass is used and maintained at a high culture level. Questions remain on the effects of specific cultural management practices and chemical treatments on the suppression or enhancement of disease symptoms and/or on the low-temperature tolerance of bermudagrass. The objectives of this investigation were to: a) determine the efficacy of selected fungicides for control of SDS and; b) determine the influence of selected nutrient regimes on the expression of SDS symptoms in bermudagrass.

In 1986-87 (year 1), SDS control experiments were located on two golf courses, one in Birmingham, Ala., where soil was a fine-loamy, siliceous, thermic Typic Hapludult (Nauvoo fine sandy loam), and in Goldsboro, N.C., where soil was a loamy, siliceous, thermic Arenic Hapludult (Kenansville loamy sand) soil. *Gaeumannomyces graminis* var. *graminis* was associated with SDS in both locations. Seven chemical treatments were used (Table 1), but only the 2.3 kg·ha^{-1} rate of fenarimol was applied at the Alabama site. High rates of late-fall N and K treatments also were applied (Table 1), because these two elements have been shown to influence cold-temperature hardiness of turfgrasses (Beard, 1973). At Goldsboro, applications were split (September and October), while single applications were made (October) at the Birmingham site. In 1987-88 (year 2), studies were conducted at golf courses located in Pinehurst, N.C., on a fine-loamy, siliceous, thermic Typic Paleudult (Norfolk sand) soil, and Goldsboro. The year-2 Goldsboro site was adjacent to, but separate from, the year-1 site. All experiments were conducted on Tifway bermudagrass with a previous history of SDS.

Chemical treatments (Table 1) were applied using a CO_2-pressurized backpack sprayer calibrated to deliver 815 liters-ha^{-1} at 276 kPa. Test areas were irrigated within 4 h after application with ≈ 2.5 mm of water. Plots (6.1 × 6.1 m) were arranged in a randomized complete block with four replicates per treatment. Percent of plot area injured by

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[1]Associate Professor, Dept. of Environmental Horticulture, Institute of Food and Agricultural Sciences, Box 110670, Univ. of Florida, Gainesville, FL 32611-0670.
[2]Professor, Dept. of Plant Pathology.
[3]Associate Professor, Dept. of Crop Science.

Table 1. Spring dead spot (SDS) incidence in bermudagrass in 1987 and 1988 following fall 1986 and 1987 treatments, respectively.

Treatment	Rate (kg·ha⁻¹)	Application timing	1987 Goldsboro 17 Mar.	1987 Goldsboro 12 Apr.	1987 Birmingham 19 Apr.	1988 Goldsboro 1 June	1988 Pinehurst 5 May	1988 Pinehurst 19 May
			Disease index[z]					
Benomyl	12	October	60 ab (3)	17 a (3)	356 ab (17)	12 a (5)	69 a (14)	208 a (19)
Fenarimol	1.5	September	15 a (2)	13 a (3)	---	41 ab (7)	73 a (16)	288 a (20)
	2.3	October	181 ab (8)	91 ab (9)	14 a (3)	71 bc (10	181 a (14)	351 a (19)
	3	November	165 ab (9)	156 ab (10)	---	21 a (5)	164 a (18)	384 a (24)
Propiconazole	2.5	October	55 a (4)	11 a (4)	256 ab (18)	173 dc (13)	97 a (14)	246 a (15)
Triadimefon	3	October	96 ab (7)	141 ab (11)	298 ab (19)	248 f (16)	208 a (17)	246 a (16)
Thiophanate	12	October	305 a–d (13)	369 ab (18)	809 bc (11)	88 c (9)	71 a (15)	259 a (17)
SCU[y] (kg N)	49 + 49	September + October	412 bcd (16)	326 ab (17)	955 c (36)	134 d (12)	239 a (18)	383 a (22)
K_2SO_4 (kg K)	147 + 122	September + October	619 d (22)	781 c (27)	1000 c (34)	198 e (18)	743 b (19)	1284 b (23)
Control	---	---	526 cd (20)	465 bc (21)	418 b (21)	149 d (13)	305 a (20)	560 a (24)

[z]Disease index = [(number of SDS patches × percent area of bermudagrass diseased) × 100]. Numbers in parenthesis indicate the average number of SDS patches for that treatment. Mean separation by Waller–Duncan k ratio *t* test at $P = 0.05$.
[y]SCU = sulfur-coated urea.

SDS was estimated visually. Percentages were then multiplied by the number of spots per plot and by 100 to obtain an SDS disease index. This index was used to ascertain the amount of damage in relation to the number of disease spots per plot. We felt that this was a useful way of rating disease seriousness, because disease expression usually ranged from several large dead spots to numerous small ones in a given area, depending on treatment, and the index was an attempt to substantiate these differences. Data were subjected to analyses of variance, and the Wailer-Duncan k ratio *t* test was used to separate treatment effects at $P = 0.05$.

In March of the first year, at Goldsboro, greatest SDS control was obtained when fenarimol (1.5 kg·ha^{-1}) or propiconazole were applied (Table 1). Compared with nontreated turf, these materials provided 97% and 90% control, respectively. Benomyl, 1-(4-chlorophenoxyl)-3,3-dimethyl-1-(1H -1,2,4-triazol-1-yl)-2-butanone (triadimefon), and fenarimol(2.3 and 3 kg·ha^{-1}) provided 89%, 82%, 66%, and 69% control, respectively. Plots treated with dimethyl[1,2-phenylene) bis(iminocarbonothioyl)]bis(carbamate) (thiophanate), potassium sulfate, and sulfur-coated urea had SDS ratings equivalent to the nontreated control, By 12 Apr., SDS control had increased to 98%, 97%, and 96% in plots treated with benomyl, fenarimol(l.5 kg·ha^{-1}), or propiconazole, respectively, when compared with the nontreated control (Table 1).

At Birmingham, only fenarimol (2.3 kg·ha^{-1}) provided SDS suppression. However, sulfur-coated urea and potassium sulfate treatments resulted in SDS index ratings 128% and 139% higher than for the nontreated control, respectively.

In the 2nd year, no significant differences were found during March at Goldsboro (data not shown). By June, SDS incidence for benomyl-, fenarimol- (3, 1.5, and 2.3 kg·ha^{-1}), and thiophanate-treated plots were 92%, 86%, 72%, 52%, and 41% less than the nontreated control, respectively (Table 1). There was, however, a 33% increase in SDS incidence in plots that received potassium sulfate.

At Pinehurst, in early May, all fungicide treatments and sulfur-coated urea provided SDS ratings similar to those in nontreated plots (Table 1), but potassium sulfate-treated plots had a 143% increase in the SDS index compared with the nontreated control. Two weeks later, all treatments had similar SDS indexes as the nontreated control, except again for potassium sulfate, which had a 129% higher SDS index.

Averaged over two sites in 2 years, a 72% reduction in SDS followed a fall application of benomyl at 12 kg·ha^{-1}. Fenarimol applied at three separate rates (1.5, 2.3, and 3.0 kg·ha^{-1}) on three fall dates reduced SDS by a combined average of 66%. A single application of propiconazole (2.5 kg·ha^{-1}) reduced disease by an average of 56%. Application of potassium sulfate (269 kg K/ha) in late fall resulted in an average increase in SDS expression of 89% the following spring over all experiments.

Previous work in North Carolina indicated that benomyl provided SDS suppression (Lucas, 1980), while research in Kansas found no decrease in SDS diseased area or number of spots following propiconazole (0.84 kg·ha^{-1}), fenarimol (3 kg·ha^{-1}), triadimefon (1.5 kg·ha^{-1}), or benomyl (9.2 kg·ha^{-1}) treatments (Tisserat et al., 1988, 1989a). Work in Maryland cited reduced SDS damage following fenarimol treatments either at 6 or 12 kg·ha^{-1} in 2 of 3 years, while propiconazole at 1.7 or 3.4 kg·ha^{-1}, benomyl at 6 kg·ha^{-1}, or triadimefon at 3 kg·ha^{-1} had no influence on SDS damage (Dernoedon, 1989; Dernoeden and McHenry, 1989). Varying control between regions of the United States may be related to different causal agents associated with SDS in each area. From this study, it appears turf managers with severe SDS should minimize heavy late-fall K applications and possibly use benomyl, fenarimol, or propiconazole for disease suppression.

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