Method and Timing of Fungicide Applications for Control of Spring Dead Spot In Hybrid Bermudagrass

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Abstract
The efficacy of five application methods and four fungicides were evaluated for control of spring dead spot (SDS) of bermudagrass from 2002 to 2004. Fenarimol and propiconazole were most effective in reducing SDS, providing from 66% to 89% and 51% to 52% control, respectively. Application water volume (2.5, 5, or 10 gal/1000 ft²), post-application irrigation, and high-pressure injection did not affect SDS control. Further research with fenarimol was conducted from 2003 into 2005 to optimize application rate and timing. In both years, all rates (6, 4 + 4, and 6 + 6 fl oz/1000 ft², with split applications 2 weeks apart) provided equivalent control of SDS when averaged across all application timings. No significant differences were detected among application timings ranging from August 1 to October 1 in 2003 and from August 23 to November 5 in 2004.

Introduction
Spring dead spot (SDS) is the most severe disease of bermudagrass (Cynodon dactylon (L.) Pers.) used on athletic fields and golf courses in temperate and subtropical climates, where bermudagrass is exposed to freezing temperatures for extended periods during the winter. This disease is caused by any of three root-infecting fungi: Ophiosphaerella korrae (J. Walker and A.M. Sm.), O. herpotricha (Fr.:Fr.) J. Walker, and O. narmari (J. Walker and A.M. Sm.) Wetzel, Hulbert, and Tisserat (15). Multiple species have been documented at the same location and within the same patch (3). Based on field inoculation studies, O. herpotricha is the most aggressive of these species, followed by O. korrae (8). Ophiosphaerella herpotricha is the most frequently encountered pathogen causing SDS in the Midwestern United States (8) whereas O. korrae is prevalent in the Mid-Atlantic and Southeastern regions (3,5).

Symptoms of SDS appear in the spring as bermudagrass resumes growth from winter dormancy. Typical field symptoms are circular patches or rings of brown to straw-colored turf, ranging from a few inches to several feet in diameter (Fig. 1). Affected plants remain dormant and eventually die (Fig. 2). Individual patches reappear annually, gradually expand in size, and often coalesce to form larger areas of affected turf. The patches may not be easily detected where bermudagrass has been overseeded in the winter with cool-season grasses or in stands with high populations of weeds. On individual infected plants, extensive necrosis of stolons, rhizomes, and roots is evident during symptom expression (Fig. 3).
Effective SDS management requires an integrated approach including host plant resistance, cultural practices and, when economical, preventative fungicide applications. Bermudagrass cultivars vary widely in their susceptibility to SDS. In general, cultivars with improved cold tolerance are less susceptible to SDS (1). Spring dead spot is typically more damaging on intensively managed turfs compared to low maintenance areas with low fertility regimes (15). Late-season applications of nitrogen have been shown to increase disease severity the following spring (14). In Maryland, monthly applications of NH$_4$Cl + KCl from May through September at 1 lb of N per 1000 ft$^2$ and 0.8 lb of K per 1000 ft$^2$ reduced spring dead spot the following spring by up to 41% and increased spring green-up by 75% when compared to the untreated control (7). Martin et al. (12) showed that SDS patch size was larger for cultivars ‘Mirage’, ‘OKS 91-11’, and ‘Jackpot’ when mown at 1.5 inches versus 0.5 inches.

Despite the severity and widespread distribution of SDS, few specific recommendations related to preventative fungicide programs are available to turfgrass managers. Due to erratic results, several extension services do not recommend preventative fungicide applications for spring dead spot control (13,16,19). Fenarimol, myclobutanil, aoxystrobin, thiophanate-methyl, and propiconazole are currently labeled for SDS control; with fenarimol and myclobutanil being the most commonly applied. While some turfgrass managers have reported excellent results from preventative applications of these products, others have not obtained adequate control. The lack of specific recommendations regarding product effectiveness, timing, or application method may be responsible for some of these inconsistencies. Differences in the aggressiveness of SDS pathogens may also influence fungicide effectiveness and may contribute to inconsistencies in SDS control among locations.

Field evaluations of fungicides for the control of SDS have dated back to the 1960s (9). Most field trials in the past evaluated fungicides that are no longer available, still experimental, or not labeled for SDS. Products such as naramel, carboxin, chloroneb, mane, benomyl, chlorothalonil, PCNB, thiram, tebuconazole, diniconazole, fenarimol, propiconazole, and thiophanate-methyl have all been tested for efficacy against SDS with variable results among researchers and geographic regions. A thorough review of fungicide evaluations conducted prior to 1993 was provided by Dernoeden (6).
In North Carolina, Wetzel (22) tested fungicides for preventative control of SDS in ‘Tifway’ bermudagrass fairways. Only fenarimol provided significant control of SDS in this study, reducing SDS severity by 71 to 85% and increased turfgrass quality. In Oklahoma, Walker et al. (21) reported no significant control of SDS from preventative application of all fungicides examined. In another study by Walker (20), propiconazole, tebuconazole, and fenarimol all significantly reduced SDS severity. Butler and Tredway (4) tested fungicides and reported no significant control of SDS on ‘Tifway’ bermudagrass in North Carolina. In an unrelated field trial, Tredway and Butler (18) reported a significant reduction in SDS severity from two fall applications of Patchwork 0.78G (4 lb/1000 ft$^2$) and Rubigan 1AS (4 fl oz/1000 ft$^2$), which both contain the active ingredient fenarimol.

Several factors have hampered SDS research and the development of effective management programs. The erratic distribution of the disease across a turf stand contributes to experimental error in field trials and reduces the investigator’s ability to detect differences among treatments. The use of inaccurate methods for assessment of SDS severity in field plots has likely contributed to experimental error as well (2). Also, very little is known about the epidemiology of SDS. The majority of pathogen growth and root infection is thought to occur in the fall, predisposing the bermudagrass to winter injury (10,11). However, the soil temperatures that trigger disease development are unknown, making the accurate timing of fungicide applications difficult.

The objectives of this research were to evaluate fungicides for SDS control, determine the impact of application method on fungicide effectiveness, and determine the optimal application timing for fenarimol. Field experiments were conducted from 2002-2005 in order to accomplish these objectives.

**Fungicide/Application Method Experiments**

This research was conducted at the Walnut Creek Softball Complex in Raleigh, NC on ‘Tifway’ bermudagrass that was severely affected with SDS. The fields are managed with monthly application of N (1 lb/1000 ft$^2$) in the form of urea or ammonium nitrate from May until September and an application of K (1 lb/1000 ft$^2$) in the form of potassium chloride in September. The study site was irrigated to prevent drought stress and mowed 3 times per week at a height of 1 inch. Weed control was applied as Simazine (1 qt/acre) in November and as Pendulum 2G (100 lb/acre) in February. The soil in this location is a sandy loam with a pH of 6.0 and a CEC of 6.5.

Fields were scouted in June 2002 when SDS symptoms were evident, and plots were established on Fields 4 and 5 in areas that were most uniformly infested with the pathogen. Based on a survey of the pathogen population, only O. korrae was detected at this location (3). In order to further minimize experimental error, we employed split-plot experimental designs, large sub-plots (5 ft × 10 ft), and 5 or 8 replications. On Field 4, the main plots consisted of fungicides, the sub-plots consisted of application methods, and treatments were replicated 8 times. On Field 5, the opposite design was used (main plots were application methods and sub-plots were fungicides); treatments were replicated 5 times.

Application methods included surface applications of fungicides in 2.5, 5.0, or 10.0 gal H$_2$O per 1000 ft$^2$, a watered-in treatment (fungicide applied in 2.5 gal/1000 ft$^2$ then followed immediately by 0.25 inches of irrigation applied by hand with a 5/8-inch water hose and a shower-type nozzle), or high-pressure injection with a Cushman Enviroject (Textron Inc., Charlotte, NC) at 3600 psi in 18 gal H$_2$O per 1000 ft$^2$ to deliver fungicides to a soil depth of 2.5 inches. The fungicides applied were azoxystrobin (Heritage 50WG, 0.4 oz/1000 ft$^2$), fenarimol (Rubigan 1AS, 6 fl oz/1000 ft$^2$), myclobutanil (Eagle 40WP, 1.2 oz/1000 ft$^2$), and propiconazole (Banner Maxx 1.4ME, 4 fl oz/1000 ft$^2$). All treatments were applied on September 30 and October 31, 2002 (Year One) and on October 2 and October 31, 2003 (Year Two). With the exception of the high-pressure soil injection method, all treatments were applied at 40 psi using a CO$_2$-powered backpack sprayer with TeeJet 8004, 8008, or 8010 flat fan nozzles (Spraying Systems Co., Wheaton, IL) to yield 2.5, 5.0, and 10.0 gal H$_2$O...
per 1000 ft$^2$, respectively. Water-only non-treated controls were also applied using each application method. The same plots were treated in both years of the experiment.

**Fenarimol Rate/Timing Experiments**

In 2003 and 2004, a field experiment was conducted on Field 4 to identify the rate and timing of fenarimol applications that were most effective for SDS control. A split-plot randomized complete block design was employed, with main plots consisting of application timings and sub-plots (5 × 10 ft) of application rates, with 4 replications of each treatment. In 2003, application timings were August 1, August 15, September 1, September 15, and October 1. Applications were scheduled for the same dates in 2004, but due to inclement weather, treatments were applied on August 23, October 4a, October 4b, October 20, and November 5.

Fungicide treatments were (i) no fungicide, (ii) fenarimol applied as Rubigan 1AS at 6 fl oz/1000 ft$^2$, (iii) Rubigan 1AS at 4 fl oz + 4 fl oz/1000 ft$^2$, and (iv) Rubigan 1AS at 6 fl oz + 6 fl oz/1000 ft$^2$; split applications were made 14 days following the initial application. In 2004, weather affected split applications and were applied as follows (initial application/follow up application): August 23/October 4, October 4a/October 20, October 4b/October 20, October 20/November 5, November 5/November 18. All treatments were applied at 40 psi using a CO$_2$-powered backpack sprayer with TeeJet 8008 flat fan nozzles to deliver 5 gal H$_2$O per 1000 ft$^2$. The same plots were treated in both years of the experiment.

**Assessment of SDS Severity and Recovery Rate**

The initial severity of SDS symptoms in the spring and the rate of bermudagrass recovery are the two parameters that are of most interest to turfgrass managers. Both of these parameters may be affected by preventative fungicide applications, consequently, both were used in this study as a measure of fungicide efficacy. Digital image analysis was used to measure SDS severity in field plots (2). With this method, an image of each plot was recorded using a digital camera mounted to a fixed monopod and then analyzed with SigmaScan Pro (SPSS, Inc., Chicago, IL 60611) to determine disease severity, as the percent of plot area affected.

Plots were rated for initial disease severity at 100% green-up, which was on 13 May 2003, 12 May 2004, and 31 May 2005. Recovery rate was determined in the fungicide/application method experiments by assessing disease severity every two weeks until SDS symptoms were no longer evident (13 May - 8 July 2003 and 12 May - 23 June 2004). The recovery rate was estimated for each individual plot using simple linear regression on square root-transformed disease severity values over time.

Initial disease severity and recovery rate values were analyzed using PROC GLM in SAS (SAS Institute Inc., Cary, NC). For factors that exhibited a significant effect on disease severity or recovery rate, mean separations were performed using the Waller-Duncan k-ratio t-test (k = 100).

**Results: Fungicide/Application Method, Field 4**

Symptoms of SDS appeared in mid-April of 2003 and 2004, when the bermudagrass began to resume growth from winter dormancy. In 2003, there were significant fungicide effects, but method and fungicide*application method effects were not significant (Table 1). Azoxystrobin, fenarimol, and propiconazole significantly reduced disease severity by 45, 66, and 51%, respectively, when compared to the untreated control (Fig. 4). No significant differences were detected among application methods (Fig. 5). Only fenarimol treated plots were significantly slower to recover in the spring when compared to the untreated control (Fig. 6). Application method did not significantly influence recovery rate (*data not shown*).
Fig. 4. Spring dead spot severity in response to fungicide applications on Fields 4 and 5 at Walnut Creek Softball Complex in Raleigh, NC, averaged across all application methods.

Fig. 5. Effect of application method on spring dead spot severity on Fields 4 and 5 at Walnut Creek Softball Complex in Raleigh, NC, averaged across all fungicides.
Table 1. ANOVA P-values for initial spring dead spot severity in fungicide and application method studies.

<table>
<thead>
<tr>
<th>Field study</th>
<th>Year</th>
<th>Fungicide</th>
<th>Application method</th>
<th>Fungicide* application method</th>
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</thead>
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<tr>
<td>Field 4</td>
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<td>&lt; 0.0001</td>
<td>0.2134</td>
<td>0.8028</td>
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<tr>
<td></td>
<td>2004</td>
<td>&lt; 0.0001</td>
<td>0.4219</td>
<td>0.3705</td>
</tr>
<tr>
<td>Field 5</td>
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<td>0.0200</td>
<td>0.9844</td>
</tr>
<tr>
<td></td>
<td>2004</td>
<td>&lt; 0.0001</td>
<td>0.5549</td>
<td>0.9510</td>
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In 2004, fungicides had a significant effect on SDS severity, but method and fungicide*application method interactions were not significant (Table 1). Fenarimol provided the best control of SDS, reducing disease severity by 89% (Fig. 4). Plots treated with propiconazole exhibited lower SDS severity compared to azoxystrobin and the non-treated control. No treatments were significantly different in recovery rate from the non-treated control; however, plots treated with azoxystrobin were significantly slower to recover than myclobutanil treated plots (Fig. 6). All application methods provided a similar level of control in 2004 (Fig. 5) and did not influence recovery rate (data not shown).

Results: Fungicide/Application Method, Field 5
In 2003, fungicide and method main effects were significant, but the interaction was not significant (Table 1). Despite the significant fungicide effect, the Waller-Duncan k-ratio t-test did not provide separation among fungicides (Fig. 4). Also, there were no differences among application methods in this study (Fig. 5). Plots treated with fenarimol and propiconazole were significantly slower to recover when compared to the non-treated control (Fig. 6). Recovery rate was not influenced by application methods in 2003 on Field 5 (data not shown).

In 2004, only fungicide main effects were significant (Table 1), and this was due to fenarimol providing significant suppression of SDS on Field 5 (Fig. 4). Plots treated with azoxystrobin, myclobutanil, and propiconazole were not significantly different from the untreated control. No significant differences in recovery rate were detected among fungicides (Fig. 6).

Results: Fenarimol Rate/Timing Experiment
In 2004 and 2005, application rate significantly influenced SDS severity, but application timing effects and rate*timing interactions were not significant (Table 2). In both years, all rates of fenarimol significantly reduced SDS severity when averaged across all timings and compared to the untreated control (Fig. 7). Fenarimol provided between 45 and 52% control in 2004 and between 41 and 48% in 2005.

Table 2. ANOVA p-values for initial spring dead spot severity in the fenarimol rate and timing study.

<table>
<thead>
<tr>
<th>Year</th>
<th>Rate</th>
<th>Timing</th>
<th>Rate*Timing</th>
</tr>
</thead>
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<tr>
<td>2003</td>
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<tr>
<td>2004</td>
<td>&lt; 0.0001</td>
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<td>0.4250</td>
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Conclusions
Spring dead spot can be managed with an integrated approach, implemented over a period of several years. Improving soil conditions, proper nitrogen fertilization, fall potassium applications, and reduction of soil pH are effective options for reducing SDS development (17). Preventative fungicide applications are an option for highly valued turf or where cultural practices alone do not provide adequate control. Of the fungicides that were evaluated in this study, fenarimol and propiconazole were the most effective in suppressing SDS. Fenarimol provided significant SDS suppression in 5 of 6 experiments and reduced disease severity by 41% to 89%. Propiconazole significantly reduced SDS severity in 2 of 4 experiments, providing reductions of 51% to 52%.

Since *O. korrae* infects the below-ground tissues of bermudagrass, application methods that deliver the fungicide closer to the causal organism were expected to be more effective than standard foliar applications. No significant differences were detected among application methods in this study. Fungicides that were watered-in with 0.25 inch or irrigation or that were applied in 5 or 10 gal H\(_2\)O per 1000 ft\(^2\), however, tended to provide better control than foliar applications in 2.5 gal H\(_2\)O per 1000 ft\(^2\). Although not evaluated in this study, many turfgrass managers apply fungicides in water volumes as low as 1.0 gal H\(_2\)O per 1000 ft\(^2\). The impact of applying fungicides to control SDS in
extremely low water volumes remains unknown. Subsurface injection using the Textron Enviroject did not significantly improve SDS control compared to surface application methods. The injection nozzles on this machine deliver the fungicide solution on 4- × 2-inch centers; therefore it is possible that the spacing is too large to provide uniform protection from pathogen infection.

Fenarimol significantly reduced the rate of bermudagrass recovery from spring dead spot in 2 of 4 trials, and propiconazole significantly reduced recovery rate in 1 of 4 trials. These results were unexpected, but there are several possible explanations for this phenomenon. First, these fungicides may have growth regulator effects or other injurious properties on bermudagrass turf when applied closer to dormancy. Alternatively, plots with higher SDS severity levels may exhibit faster recovery due to the exponential relationship between patch diameter and patch area or possibly due to reduced competition among plants. The square-root transformation of the SDS severity data in this study was designed to linearize the disease severity data and account for the exponential diameter-area relationship, but this transformation may not account for potential competition effects. Further research is needed to investigate the influence of preventative fungicide applications on recovery from spring dead spot injury.

Upon further investigation of fenarimol over a two-year period, all rates of fenarimol evaluated provided a similar reduction of SDS severity. However, no differences among application timings were detected. These results suggest that rate and timing do not have a major impact on SDS control as long as an application(s) is made between mid-August and mid-October in North Carolina. This time period generally corresponds to mean daily soil temperatures between 60 to 80°F (measured at 4-inch depth with local weather station).

Based on the results of this project, preventative control of SDS with fungicides is possible. However, complete disease control apparently cannot be obtained within two years following initiation of a fungicide program. In the fungicide/application method experiments, fenarimol provided 66% control in the first year and 89% control in the second year, and propiconazole provided 51% control in the first year and 52% control in the second year. In the fenarimol rate/timing experiment, when averaged across all rates and timings, reduction of SDS was 49% and 45% in the first and second years, respectively. Long-term field research is needed to determine if the level of SDS control may improve after several successive years of preventative fungicide treatments. It should be emphasized that this study was conducted on a site that was infested with O. korrae and that these strategies may not apply to managing SDS caused by other Ophiosphaerella spp.

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**Literature Cited**


