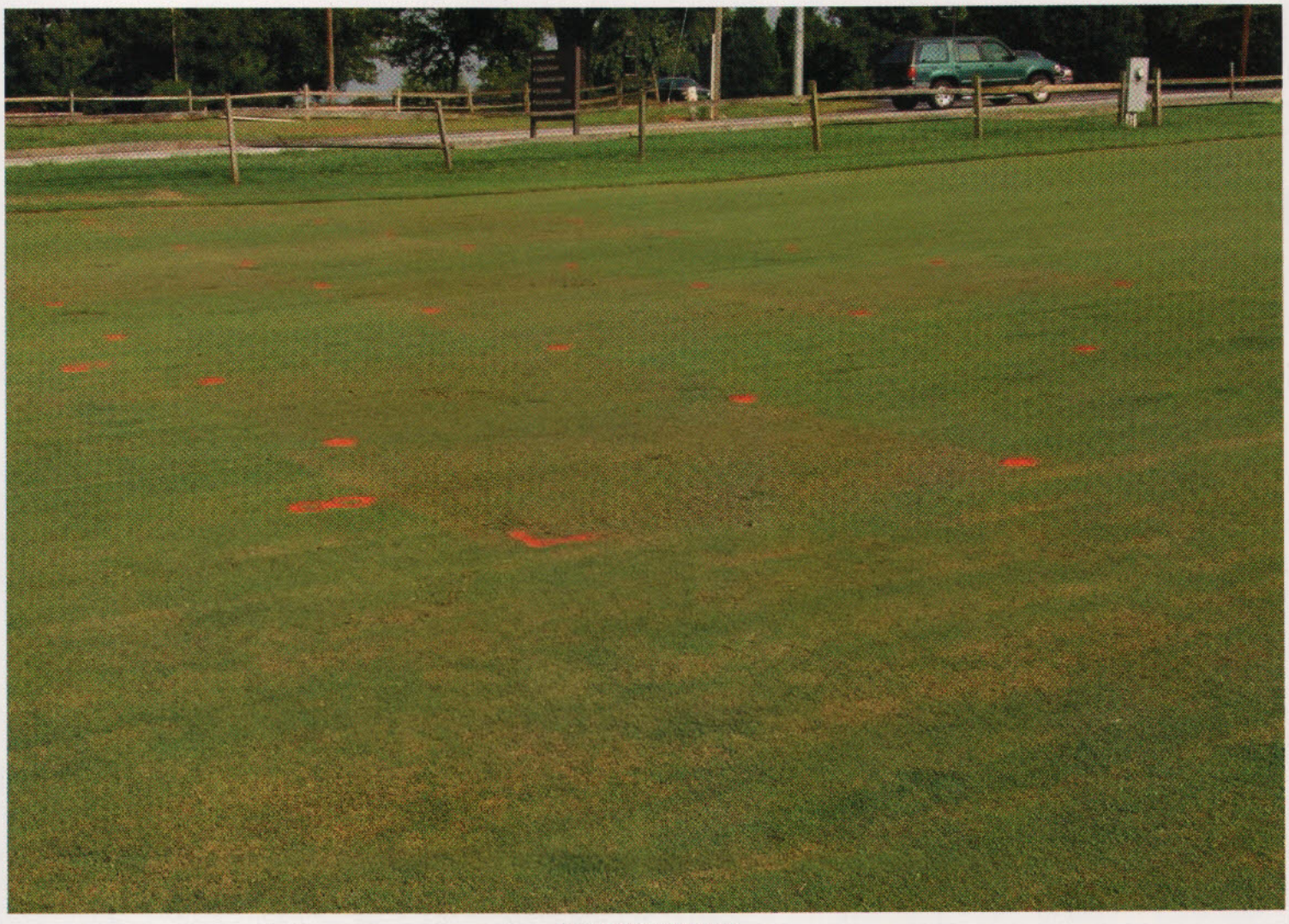
Plant growth regulators' effect on bermudagrass

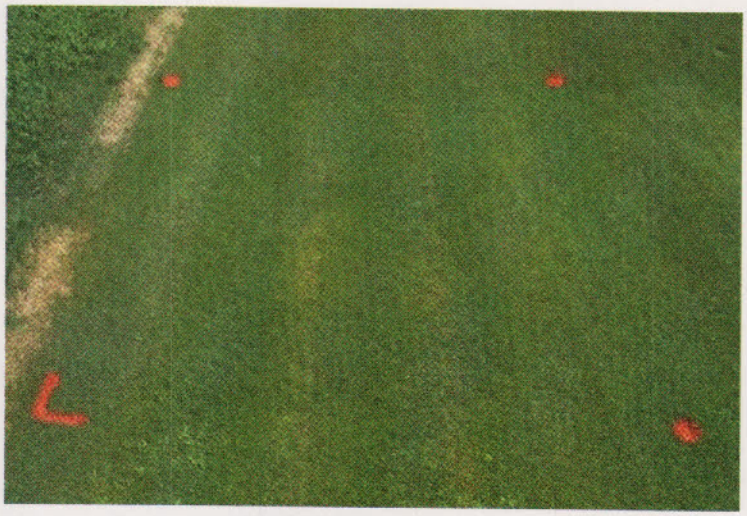
By F.W. Totten and L.B. McCarty

Bermudagrass is a popular turfgrass used on athletic fields due to its aggressive summer growth habit, fine leaf texture, and dark green color. However, if mowed infrequently, excessive scalping and clipping production combine to decrease its aesthetic quality. Plant growth regulators (PGRs) were introduced into the turf industry in the 1950's and since have been used in part to reduce mowing requirements and enhance color of bermudagrass.

PGRs are classified as Type I or Type II based on their mode of action. Type I PGRs, maleic hydrazide, mefluidide, and amidochlor, for example, inhibit cell division. Since being introduced, their use has become limited due to high potential for phytotoxicity and reductions

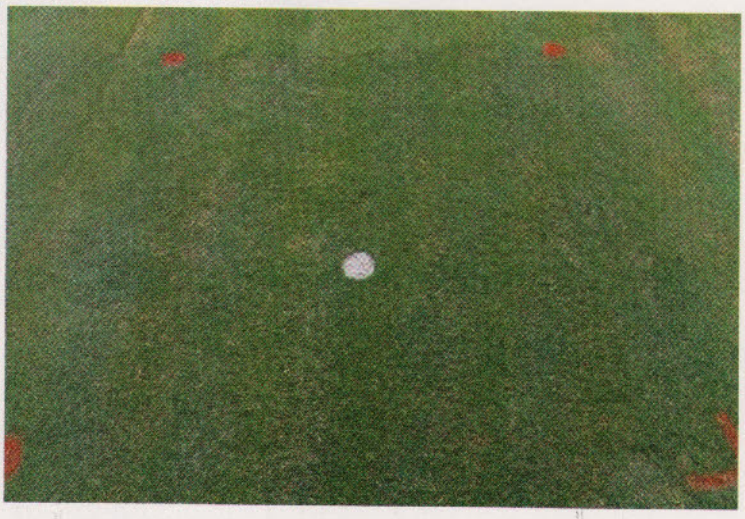


Overview of trial plots.



Untreated areas.

Plot with paclobutrazol at 16 ounces annually.



Plot with TE + paclobutrazol at 12 and 32 ounces annually.

in rooting. Type II PGRs inhibit the biosynthesis of gibberellic acid (GA), the hormone responsible for cell elongation. Alternatively, Type II PGRs can be classified as Class A or Class B based on their route of entry into the plant and the location in which they inhibit the GA biosynthesis pathway.

Flurprimidol interrupts GA biosynthesis early in the pathway via the cytochrome p450 monooxygenase enzyme. The result is shorter internode length, caused by reduced size of plant cells. Flurprimidol is predominantly root absorbed and exhibits excellent soil residual, extending growth regulation over time (www.SePRO.com/documents/cutless-bunnell.pdf). Flurprimidol use on warm season turfgrasses can potentially increase turf color and density, reduce mowing up to 50%, and reduce water use by the plant. The typical rate of flurprimidol on Tifway bermudagrass maintained at 0.5 to 0.75 in. ranges from 4 to 8 oz/A with repeat applications generally every 3 to 6 weeks. Flurprimidol at 8 oz wt/A, based on six applications per season, costs approximately $287/annually.

Paclobutrazol is similar to flurprimidol in that it interrupts GA biosynthesis early pathway via the cytochrome p450 monooxygenase enzyme and is root absorbed. Potential advantages to using paclobutrazol include turfgrass quality enhancement, reduction in mowing frequency, and Poa annua L. suppression. The typical rate of paclobutrazol on Tifway maintained at 0.5 to 0.75 in. ranges from 32 to 48 fl oz/A (depending on soil type), and a repeat application can be no sooner than 8 weeks after the initial application. Paclobutrazol at 32 fl oz/A, based on two applications per season, costs approximately $170/annually.

Trinexapac-ethyl is foliar absorbed and inhibits GA biosynthesis late in the pathway via the 3_-hydroxylase enzyme preventing active GA formation. Trinexapac-ethyl can potentially increase turf color, promote lateral stem and root mass development, and reduce vertical growth (www.syngenta.com/en/products_services/turf_page.aspx). The rate of trinexapac-ethyl typically used on Tifway bermudagrass maintained 0.5 to 0.75 in. is approximately 12 fl oz/A. Trinexapac-ethyl at 12.8 fl oz/A, based on seven applications per season, is approximately $245/annually (www.SePRO.com/documents/cutless_primo.pdf).

Tank-mix more effective?

"Is a flurprimidol + trinexapac-ethyl tank mix more effective in terms of growth regulation and cost?" is a question being asked by many turfgrass managers. Cooper (www.sepro.com/documents/cutlesscooper.pdf) found that a flurprimidol + trinexapac-ethyl tank mix applied at 4 oz wt/A + 6 fl oz/A, respectively, caused less injury and greater turfgrass quality on Tifway, compared to trinexapac-ethyl alone at 12 fl oz/A and flurprimidol alone at 8 oz/A.

Bunnell et al. (2005) reported a flurprimidol + trinexapac-ethyl combination at 4 oz wt/A + 6 fl oz/A provided Tifway bermudagrass turf quality and growth regulation comparable to trinexapac-ethyl used exclusively at 12 fl oz/A. However, our research indicated no advantage in growth regulation by using a flurprimidol + trinexapac-ethyl combination compared to using the products alone.

A flurprimidol or paclobutrazol + trinexapac-ethyl tank mix provides several potential benefits. The combination, or tank mix, provides growth regulation in early and late stages of GA synthesis, and provides both foliar and root absorption of PGRs. This potentially

Treatments included:

Product(s)	Rate
1. Untreated	
2. Trinexapac-ethyl	12 fl oz/A
3. Flurprimidol	8 oz wt/A
4. Trinexapac-ethyl + Flurprimidol	12 fl oz/A + 8 oz wt/A
5. Paclobutrazol	16 fl oz/A
6. Paclobutrazol	32 fl oz/A
7. Paclobutrazol + Trinexapac-ethyl	16 + 12 fl oz/A
8. Paclobutrazol + Trinexapac-ethyl	32 + 12 fl oz/A

provides longer residual growth regulation, and could subsequently reduce the number of annual applications. Also, from a cost-effective standpoint, approximately half the rate of either PGR is needed compared to when either product is used alone. The objective of this research was to evaluate Tifway's phytotoxicity, regrowth, and growth regulation in response to various rates of flurprimidol, trinexapac-ethyl, and paclobutrazol alone and in combination.

Our study was conducted during the summer of 2005 and 2006, at the registered Tifway bermudagrass research site at Clemson University. Experimental design was a randomized complete block with three replications, and treatments were arranged as a 3 by 3 factorial design. Plot size was 36 square feet.

During both summers, turf was mowed six days a week at 0.56 in., and irrigated to maintain a well-watered status. Treatments were applied with a CO2 backpack sprayer calibrated at 20 GPA. After the initial treatment, three sequential applications were made at 21-day intervals.

During both years, turf injury was measured weekly on a scale of 0 to 100% with >30% being unacceptable. Turfgrass quality was rated visually every 14d on a scale from 0 to 9 where 0 = brown turfgrass and 9 = dark green turfgrass. Percent lateral regrowth was measured using methods described by Bunnell (2003). A 4-inch Tifway plug was removed at the initiation of the study from each replicate. The holes were backfilled with an 85:15 sand/peat mix. A wire mesh grid containing 230 uniform squares was constructed in equal dimension to the original hole. A green shoot present in one 0.062 in2 square denoted one point. Percent lateral regrowth was calculated by:

Number of squares green shoot points divided by total points (230) times 100. Clippings were harvested from all treatments for all three replicates at 4, 8, and 12 weeks after the initial (WAIT) application and analyzed for dry weight.

Results

All injury observed during the 2005 and 2006 seasons was acceptable, thus not exceeding the 30% threshold. Injury was greatest, 2 WAIT, for all tank mix combinations containing trinexapac-ethyl and injury ranged from 10 to 25%. No injury was observed 3 WAIT for both years. Turfgrass visual quality was unacceptable, 2 and 4 WAIT, by all trinexapac-ethyl tank mix combinations. However, turfgrass quality was ≥ 7 for all treatments at all other rating dates.

Exceptional turfgrass quality was observed with flurprimidol during both years of the study and consistently provided turfgrass quality ratings around 8. Primo alone and tank mix combinations with paclobutrazol also provided consistent turfgrass quality ratings around 7.5.

Lateral regrowth was reduced 2 WAIT with flurprimidol + trinexapac-ethyl, paclobutrazol (16 fl oz/A/21d) + trinexapac-ethyl, and paclobutrazol (32 fl oz/A/21d) + trinexapac-ethyl combinations. Reductions were approximately 18% from the untreated check. Reductions in lateral regrowth by the tank mix combinations were not surprising and somewhat expected as the rates that comprised these tank mixes were providing a high amount of active ingredient.

Paclobutrazol applied alone at 16 fl oz/A/21 days did not significantly reduce clipping yield, compared to the untreated check, at 4 or

8 WAIT. Paclobutrazol applied alone at 32 fl oz/A/21 days reduced clipping yield 36% 8 WAIT. Flurprimidol applied alone at 8 oz wt/A/21 days reduced clipping yield 21 and 44% at 4 and 8 WAIT, respectively, while trinexapac-ethyl applied alone at 12 fl oz/A/21 days reduced clipping yield 43 and 67% at 4 and 8 WAIT, respectively.

Clipping yield reductions peaked, 8 WAIT, ranging from 67% to 80% with trinexapac-ethyl alone, flurprimidol + trinexapac-ethyl, and paclobutrazol (32 fl oz/A/21 days) + trinexapac-ethyl tank mix combinations. Also of importance, clipping yield was reduced 33%, 12 WAIT, from the untreated check by flurprimidol + trinexapac-ethyl. This tank mix combination showed good residual with respect to reducing clipping yield up to three weeks after the final application.

The tank-mix combination rates in this study, while not necessarily economically feasible, were chosen based of their performance in previous research by the author. Results from this research indicate no apparent advantage to a trinexapac-ethyl + paclobutrazol tank-mix compared to using trinexapac-ethyl alone at 12 fl oz/A/21 days. The flurprimidol + trinexapac-ethyl tank-mix combination produced the greatest reduction in clipping yield while causing acceptable injury to Tifway; however, lateral regrowth was reduced 18% 2 WAIT by this combination.

The flurprimidol + trinexapac-ethyl tank-mix combination exhibited great efficacy in both years with respect to reducing clipping yield. Yield reductions 12 WAIT, or 3 weeks after the final application, exceeded 30% with this treatment. Exceptional turfgrass quality and minimal injury (less than 10%) was observed with flurprimidol applied alone at 8 oz wt/ A/21 days during both years of the study and consistently provided turfgrass quality ratings around 8.

Future research should continue to vary rates and timings of flurprimidol, trinexapac-ethyl, and paclobutrazol tank-mix combinations on hybrid bermudagrass and other cool and warm-season turfgrasses. Residual clipping control of tank mix combinations should continue to be evaluated and better understood. Also, other PGR chemistries should be evaluated for potential use in the area of turfgrass management. ■

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John Mascaro's Photo Quiz

Can you identify this sports turf problem?

Problem: Black spots on turf
Turfgrass Area: Practice field
Location: Blacksburg, Virginia
Grass Variety: Bluegrass

Answer to John Mascaro's Photo Quiz on Page 43

John Mascaro is President of Turf-Tec International

Combating spring DEAD SPOT

By Lane Tredway and Lee Butler

Spring dead spot leaves sunken depressions in the turf stand that may persist for much of the summer.

Hybrid bermudagrasses are excellent performers in athletic fields because of their uniformity, traction, and tolerance to heat and wear. Unfortunately, every turfgrass species has its weaknesses. Although resistant to most disease problems, bermudagrasses are highly susceptible to the fungal disease spring dead spot. This disease annually causes widespread damage to bermudagrass in athletic fields and intensely managed landscapes throughout the transition zone.

As the name implies, symptoms of spring dead spot appear in the spring as the bermudagrass comes out of winter dormancy. Circular patches of turf ranging from 6 inches to several feet in diameter remain dormant as the surrounding turf turns green. The patches of dormant turf eventually disintegrate, leaving depressions in the playing surface. These sunken areas destroy the uniformity of the turf, and may even create a hazardous condition for athletes. Recovery from spring dead spot is slow, and occurs from spread of bermudagrass into the patch

from the outside. In severe cases, recovery can take the entire growing season.

Spring dead spot usually appears within 2 to 3 years after construction of a new athletic field. The disease becomes progressively worse over time, the patches increasing in size and number each year. In some cases, the disease may disappear or decline in its severity. Unfortunately, these are the exception rather than the rule. In most cases, the disease continues to become more severe until steps are taken to control it.

Many turfgrass managers have attempted to manage spring dead spot only to obtain mediocre results. This has led many to conclude that the disease cannot be controlled. It is possible to manage spring dead spot, but effective management requires an integrated approach, as well as a lot of patience and persistence.

Unlike most turf diseases, spring dead spot does its damage below ground. To see this, take a core from the edge of a spring dead spot patch and break it apart. You will notice that the below-ground structure of the turf is very weak. Few rhizomes and roots are present, and the few that are there are black and rotten.

Your goal in spring dead spot management is to re-establish this below-ground structure. This is not something that can be accomplished in a single season. Since it takes several years for spring dead spot symptoms to appear, it will likely take several years to bring it under control as well. No matter what you do, you will not obtain 100% control of spring dead spot in the first year of a management program. Rather, you will notice a gradual reduction in the disease over a period of years.

Another unique feature of spring dead spot is that the pathogens, species of *Ophiosphaerella*, do not appear to kill the bermudagrass directly. Instead, they attack the bermudagrass roots, rhizomes, and stolons during the fall and winter and increase the bermudagrass' susceptibility to winterkill. This is why spring dead spot is always most severe after a cold winter. Maximizing the winter hardiness of bermudagrass turf is one of the most effective ways to manage spring dead spot. Varieties with improved cold tolerance also show some tolerance to the disease.

Symptoms of spring dead spot become evident as the bermudagrass comes out of winter dormancy.

Cultural management

The first step is to evaluate the health of the soil and to alleviate any problems that exist. Any soil condition that reduces bermudagrass root growth will increase the severity of spring dead spot. Soil compaction, poor soil drainage, and excessive thatch accumulations are the most common culprits. Regular aerification of high-traffic areas, at least 3 times annually, is needed to alleviate soil compaction. This will reduce spring dead spot problems and improve the overall quality of the turf throughout the season. In sites that are poorly drained, installation of subsurface drainage will help to reduce the disease. Excessive thatch is a common problem in higher heights of cut and areas that receive little traffic. Vertical mowing or aerification should be conducted as needed to maintain thatch accumulations below 1 inch.

Proper nutrition is also critical. Excessive nitrogen levels reduce the winter hardiness of bermudagrass and enhance damage from the

Recovery from spring dead spot occurs by spread of stolons from outside of the patch.

Spring dead spot pathogens attack the roots, rhizomes, and stolons of bermudagrass and make the turf more susceptible to winter injury.

disease. Generally, bermudagrass should be fertilized with no more than 1 lb N/1000 square feet/month during the growing season, and nitrogen should not be applied within 6 weeks of dormancy. Research has shown that use of ammonium forms of nitrogen, such as ammonium sulfate, can reduce spring dead spot injury. This may be due to reduction of soil pH or other mechanisms (see below).

Potassium applications in the fall have been shown to control spring dead spot, again by increasing bermudagrass winter hardiness. One to two fall applications of potassium chloride or potassium sulfate, totaling 1-2 lb K_2O per 1000 square feet is recommended. The timing of these applications is not critical, but it should be applied early enough so that the bermudagrass can absorb the potassium before it goes dormant.

Take-all patch and summer patch, diseases that are very similar to spring dead spot, are enhanced by high soil pHs. Many have assumed that a similar relationship exists with spring dead spot, but there has been little research to confirm this. We have seen severe cases of spring dead spot in soils with pH ranging from the low 4's to the high 7's. Until the relationship between spring dead spot and soil pH can be clarified, we recommend applications of lime or elemental sulfur only as recommended by routine soil tests.

Speeding recovery

Once the symptoms of spring dead spot appear, very little can be done to control the disease. Steps should be taken, though, to encourage rapid recovery and shorten the length of time when symptoms are evident. It is essential to avoid dinitroaniline herbicides (pendimethalin, prodiamine, oryzalin) or dithiopyr (Dimension) for preemergence grass control in the spring. These also inhibit rooting of bermudagrass stolons into the spring dead spot patches, thereby slowing the recovery process. Where spring dead spot is a persistent problem, oxadiazon (Ronstar) is recommended because it does not inhibit root growth.

Spring dead spot recovery is like a grow-in situation on a small scale. Good stolon-soil contact and light and frequent fertilization and irrigation is needed so that the stolons can root and become established quickly. Regular spiking or aerification is essential to break up the layer of dead turf and provide the roots with access to the soil. Irrigation and fertilization should be light and frequent to 'spoon-feed' the spreading stolons. It is important, though, to avoid saturating the soil or making excessive applications of nitrogen (>1 lb N/1000ft^2/month), as this can make spring dead spot worse in the following year.

What about fungicides?

Many turf managers have attempted to control spring dead spot with fall application of fungicides, only to see the disease return the following spring. This has caused most to abandon fungicide use in frustration. In fact, several extension services specifically do not recommend fungicide applications for spring dead spot control due to erratic results. Remember, though, that spring dead spot control is a long-term venture! Fungicides will never provide complete control in the first year, but rather, certain products will provide a gradual reduction in symptoms over time.

Several fungicides are labeled for spring dead spot control, including azoxystrobin

(Heritage), fenarimol (Rubigan), myclobutanil (Eagle), propiconazole (Banner Maxx and others), and thiophanate-methyl (3336 and others). Over 5 years of research at NC State, we have seen effective and consistent control of spring dead spot from Rubigan 1AS. A single application of 6 fl oz/1000 square feet has been as effective as two applications at 4 fl oz/1000 square feet or 6 fl oz/1000 square feet. We have also seen significant control from Banner Maxx and tebuconazole (Lynx) in some of our experiments.

The timing and method of application has a huge impact on a fungicide's performance. Since spring dead spot is a soilborne disease, fungicides should be applied in large volumes of water (at least 5 gal/1000 square feet) or watered-in with 1/8 to 1/4 inch of irrigation immediately after application. Rubigan applications have been equally effective when made between mid-August and late-October in North Carolina, roughly corresponding to soil temperatures between 60 and 80 degrees. Preventative applications should be made within this window for best results.

Spring dead spot is actually caused by three different species of *Ophiosphaerella*: *O. korrae*, *O. herpotricha*, and *O. narmari*. In the Midwest and Great Plains, *O. herpotricha* is the dominant pathogen, whereas *O. korrae* is most common in the Southeast, Mid-Atlantic, and California. This is important because research in Kansas and North Carolina has shown that *O. herpotricha* is more aggressive than *O. korrae*, and is possibly more difficult to control. Our results may not apply to areas where *O. herpotricha* is the primary cause of spring dead spot.

Certain fungicides claim to speed recovery from spring dead spot rather than reduce the initial amount of symptoms. Some turf managers have also observed that fall applications increased recovery from spring dead spot. Is this wishful thinking or a real phenomenon?

In an attempt to answer this question, we measured the rate of bermudagrass recovery in response to fungicide applications. No fungicides increased bermudagrass recovery, and in fact, Banner Maxx, Heritage, and Rubigan applications actually slowed the recovery rate compared to untreated plots. Regardless, plots treated with Rubigan reached 0% disease more quickly because there were fewer symptoms initially. Recovery rate in the spring should not be considered when selecting a product for spring dead spot control. ■

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Little spring dead spot is evident on this soccer field (left), while severe damage is seen in the surrounding areas (right). What's the difference? The playing surface was aerified three times in the previous year, while the surrounding areas were not.