



Managing Spring Dead Spot in Bermudagrass

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IMPORTANCE

Spring dead spot is the most destructive disease of bermudagrass in Kentucky. While this disease can occur regardless of how turf is managed, the most serious outbreaks occur under high maintenance conditions (e.g. high nitrogen fertility, low mowing height, and frequent traffic).

SYMPTOMS

Softball- to beach ball-sized patches (approximately 4 to 12 inches) fail to green-up in spring in areas that appeared healthy the previous autumn (FIGURE 1). Turf in these dead patches have a bleached, whitish color, becoming tan to brown as the dead tissue ages. Close inspection of the roots, stolons, and rhizomes of symptomatic turf reveals a brown to black decay (FIGURE 2).

Bermudagrass will often slowly recolonize the dead patches as warmer weather arrives in summer. Regrowth may occur in the center of patches, resulting in tufts of healthy plants (called a “frog-eye” appearance), but more often recolonization is more diffuse. Bermudagrass that has regrown into the patches usually will appear healthy until the following spring. In some cases, however, weeds or cool-season grasses encroach the diseased areas and bermudagrass is unable to compete with them (FIGURE 3). Because patches tend to recur in the same area, expanding patches may appear as rings or arcs of dead turf after 2 to 3 years.

FIGURE 1. SPRING DEAD SPOT PATCHES IN BERMUDAGRASS.



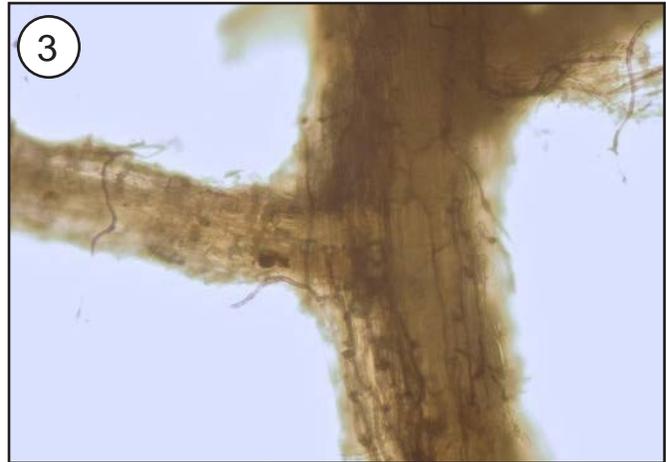


FIGURE 2. ROOTS, STOLONS, AND RHIZOMES OF SPRING DEAD SPOT INFECTED PLANTS DECAY AND TURN BROWN TO BLACK WHEN DAMAGED BY WINTER FREEZES.

FIGURE 3. WEEDS AND COOL-SEASON GRASSES MAY ENCROACH AND DOMINATE SPRING DEAD SPOT PATCHES.

FIGURE 4. BEFORE INFECTING ROOTS, THE PATHOGENS COLONIZE ROOT SURFACES WITH DARK-COLORED ECTOTROPHIC RUNNER HYPHAE.

CAUSES & DISEASE DEVELOPMENT

Spring dead spot is a complex disease involving an interaction between fungi and winter cold; however, it is not fully understood in spite of years of extensive research.

Several soilborne fungi—*Ophiosphaerella korrae*, *O. herpotricha*, and *O. namari*—have been confirmed in spring dead spot-infected bermudagrass in Kentucky. In addition, the soilborne fungus *Gaeumannomyces graminis* var. *graminis*, which can be found in Kentucky soils, has been associated with this disease in the southeastern U.S.; it is possibly one of the causes of spring dead spot in Kentucky under some conditions.

These fungi are thought to be most active in autumn and spring when temperatures are cool and soil is moist. Before infecting roots, the fungi colonize root surfaces with the production of dark-brown, microscopic fungal filaments called ectotrophic hyphae, also referred to as runner hyphae (FIGURE 4). Roots become infected when hyphae penetrate them and grow within the vascular tissue. Colonization and infection of roots by these fungal pathogens are likely

when soil temperatures range from 50°F to 75°F, with the most activity around 60°F. Because bermudagrass roots grow extremely slowly at cooler temperatures, these infectious fungi have a competitive advantage over the turfgrass. Infections in autumn lead to dead patches in spring, while springtime infections may slow the regrowth of bermudagrass into the dead patches, but they do not cause new patches to appear.

Spring dead spot fungi do not directly kill infected turf. In fact, bermudagrass plants with infected roots usually show no symptoms of poor health. However, infected crowns are more sensitive to cold and freeze damage during winter dormancy than those of non-infected plants. Thus, lethal damage is only evident when foliar growth normally resumes in spring, after winter injury has taken its toll. Decay of roots and crowns infected with the spring dead spot fungi occurs after these tissues are first killed by winter injury and later invaded by secondary organisms.

Disease development does not typically occur until several years after turf is established. Occasionally, after 3 or 4 years of severe disease development at a particular site, disease severity can also decline even when management practices have not changed. This poorly understood phenomenon of natural disease decline may relate to the buildup of natural biological control in the soil.

MANAGEMENT

Managing spring dead spot requires an integrated approach, generally over several seasons. Implementing as many of the following practices as possible may not completely eliminate this disease; however, these management tools can help reduce disease severity.

Disease Resistance

High levels of resistance to spring dead spot in bermudagrass varieties have not yet been demonstrated experimentally. However, limited data suggest that those varieties with greater winter hardiness are somewhat less susceptible to the disease. To date, no confirmed cases of spring dead spot have been documented in 'Quickstand' bermudagrass, which may indicate that this cultivar will suffer less from spring dead spot than other available varieties.

Soil pH

In some soil situations, maintaining soil acidity at approximately pH 5.0 to 5.3 can reduce disease pressure substantially. Recognize that this pH range is a guideline; there is no critical pH below which spring dead spot does not develop, at least within the pH range acceptable for bermudagrass growth. Research data from the University of Kentucky indicate that the relationship between soil pH and disease development is very site-specific. Nevertheless, maintaining a low pH may help to reduce spring dead spot damage in some situations.

In soils with high soil pH, reductions should be incremental. This is possible only in bermudagrass swards not receiving frequent applications of high-pH irrigation water. For some soils, exclusive use of ammonium-based fertilizers, such as ammonium sulfate, is the best long-term approach. However, it takes several years of applications to affect disease development, and reductions in disease damage do not occur in all soils.

Ammonium sulfate has a high salt content and can cause leaf injury when in contact with wet foliage during warm temperatures (80°F or greater). Under those conditions, irrigate immediately after application to wash the fertilizer off leaves. Avoid the use of fertilizers containing nitrate as the only

nitrogen source, such as sodium nitrate, as they can increase disease severity.

Another approach to reducing soil pH is to make a light application of flowers of sulfur (one pound per 1,000 square feet) specifically to areas with the disease, and then evaluate the results for 1 or 2 years before deciding to treat again. Combine flowers of sulfur with fertilization using ammonium fertilizers. An incremental approach is recommended and soil pH should be monitored in areas treated. Over-application of sulfur can lead to excessively low pH, which can result in slow spring green-up and turf thinning, particularly in soils with a low organic matter content.

There is some concern that an excessively low pH may predispose the turf to winter injury. In University of Kentucky research, soil pH had absolutely no effect on winter kill over a pH range of 4.6 to 6.5. However, caution is urged to avoid lowering the soil pH too much.

Other Fertility Practices

- Avoid late-season nitrogen applications. Make the last nitrogen application by late July, and avoid slow-release nitrogen fertilizers at that time. The objective is for the turf to run out of available nitrogen around Labor Day.

- Maintain adequate potash fertility. Even slight deficiencies can contribute to more severe spring dead spot. Even when soil tests indicate a high level of potassium, a late autumn application of approximately 80 pounds of K₂O per acre is a good practice.

Other Cultural Practices

- Raise the mowing height before Labor Day. Higher mowing heights result in higher levels of carbohydrate reserves in roots and crowns. This can decrease damage from the disease and provide more insulation from severe winter weather.

- Minimize thatch through a regular aeration program. Studies have shown that spring dead spot is more severe with heavy thatch accumulation. One or two aggressive aerifications (coring) during summer will increase juvenile growth and tiller production.

Fungicides

Several fungicides are labeled for spring dead spot management. While the products tested in the University of Kentucky Turf Research Program have given inconsistent control (sometimes without any measurable disease suppression), some success with fungicide applications has been achieved in various tests in neighboring states. In these tests, one application in mid-October was as effective as two applications—one in mid-September followed by a second application in mid-October.

When using fungicides, irrigate the product into the root zone with 1/8 to 1/4 inch of water immediately after application before the product has a chance to dry on the leaf surface. See the University of Kentucky Extension publication *Chemical Control of Turfgrass Diseases* (PPA-1) for current information on performance of fungicides labeled for spring dead spot.

ADDITIONAL RESOURCE

- Chemical Control of Turfgrass Diseases, PPA-1
<http://www.ca.uky.edu/agc/pubs/ppa/ppa1/ppa1.pdf>

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