

# Managing Spring Dead Spot of Bermudagrass

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## IMPORTANCE

Spring dead spot is the most destructive disease of bermudagrass in Kentucky. The most serious outbreaks occur under high maintenance conditions; e.g., high nitrogen fertility, low mowing height, and frequent traffic. Moderate to severe outbreaks can occur under low-maintenance conditions as well.

High levels of resistance to spring dead spot in bermudagrass varieties have not yet been demonstrated experimentally. Limited data suggest that those varieties with best 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 variety will suffer less from the disease than other available varieties.

## SYMPTOMS

Softball- to beachball-sized patches fail to green up in the spring in swards that appeared healthy the previous autumn. The turf in these dead patches has a bleached, whitish color (FIGURE 1), becoming tan to brown as the dead tissue ages. Close inspection reveals that the roots, stolons, and rhizomes in the infected patches exhibit a brown to black rot (FIGURE 2).



FIGURE 1. SPRING DEAD SPOT PATCHES IN BERMUDAGRASS.



FIGURE 2



FIGURE 3

**FIGURE 2.** ROOTS, STOLONS, AND RHIZOMES OF SPRING DEAD SPOT INFECTED PLANTS DECAY AND TURN BROWN TO BLACK.

**FIGURE 3.** A TUFT OF HEALTHY TURFGRASS IN THE CENTER OF NECROTIC PATCHES GIVES THESE AREAS A “FROG-EYE” APPEARANCE.

Loss of bermudagrass in diseased areas allows weeds to encroach. Bermudagrass will often recolonize the dead patches during the summer, although it may do so slowly. Complete recovery of infected areas can take the entire growing season. In some cases, the bermudagrass will not compete with weeds or cool-season grasses that can dominate the patches. Bermudagrass that has regrown into the patches usually will exhibit no symptoms of the disease until the following spring.

Patches tend to recur in the same spots for several years. After 2 to 3 years, the centers of active patches sometimes develop a tuft of healthy turf in the center (sometimes called a “frog-eye” appearance; FIGURE 3). As these patches continue to expand over a period of years, they can appear as rings or arcs of dead turf in the spring.

In sites where it occurs, spring dead spot typically does not develop until several years after establishment. This may relate to the time required to build up a thatch layer, which may favor the disease. 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 may relate to the buildup of natural biological control in the soil.

## CAUSES & DISEASE DEVELOPMENT

Several soilborne fungi—*Ophiosphaerella herpotricha*, *O. korrae*, and *O. namari*—have been reported to cause this disease in several locations in the United States. All of these organisms have been confirmed in spring dead spot-infected swards in Kentucky. Additionally, the soilborne fungus *Gaeumannomyces graminis* var. *graminis* has been associated with this disease in the southeastern United States.

These fungi are thought to be most active in autumn and spring when temperatures are cool and the soil is moist, although studies are needed on when these pathogens colonize roots. The fungi grow over the surfaces of bermudagrass roots by producing dark-brown, microscopic fungal filaments called ectotrophic hyphae. Roots are infected when hyphae penetrate and grow within the vascular tissues of the root.

Colonization and infection of roots by *O. herpotricha* and *O. korrae* are likely when soil temperatures range from 50°F to 75°F, with the most activity at approximately 60°F. Because bermudagrass roots grow extremely slow at 60°F or lower, the infectious fungi have a competitive advantage over the plant at cool temperatures.

Bermudagrass plants with infected roots usually show no symptoms of poor health. However, crowns of infected bermudagrass plants infected by *O. herpotricha* and *O. korrae* are more sensitive to lethal damage from cold temperatures during winter than are crowns of uninfected plants. Damage from infection is evident only when foliar growth resumes in the spring. Activity of the infectious fungi during the autumn, therefore, leads to dead patches in the spring. Springtime infections can slow the regrowth of bermudagrass into the dead patches, but these do not cause new patches to appear.

## MANAGEMENT

No single management practice will provide complete control of spring dead spot. The best control is to integrate as many of the following practices as possible. Even with all of these practices, some disease can still develop, but it is less severe than if no management program is implemented.

### Soil pH

In some soils, maintaining soil acidity at approximately pH 5.0 to 5.3 (extracted in distilled water) can reduce disease pressure substantially. This is particularly important in the soil immediately around the roots (the rhizosphere). Why this reduces disease is unclear. A soil pH in this range does not prevent growth of the infectious fungi in culture, but low soil pH may enhance plant resistance in some way, or it may create a soil environment more favorable for natural antagonistic microorganisms of the pathogens. Recognize that a pH of 5.0 to 5.3 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. Data from University of Kentucky experiments 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 your bermudagrass.

In soils with high 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 conditions when leaf injury can occur, irrigate immediately after application to wash the fertilizer off the leaves. Avoid the use of fertilizers containing nitrate, such as sodium nitrate, as the only nitrogen source. Studies suggest that the effect of urea and sulfur-coated urea on spring dead spot development is intermediate when compared with ammonium-based fertilizers and nitrate-based fertilizers.

Another approach to reducing the 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 whether to treat again. If flowers of sulfur is applied, take an incremental approach and combine this with fertilization using ammonium fertilizers. An incremental approach is recommended because over-application of sulfur can lead to slow spring green-up and turf thinning, particularly in soils with a low organic matter content. This injury is thought to be due to an excessively low pH in the uppermost soil horizon in areas where sulfur was over-applied.

Monitor the pH of the areas treated. There is some concern that an excessively low pH may predispose the turf to winter injury. In University of Kentucky research, absolutely no effect of soil pH on winter kill over a pH range of 4.6 to 6.5 was found. However, as noted above, there has been clear evidence of slow spring green-up and turf thinning from over-applications of sulfur, so 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, an application of approximately 80 pounds of K<sub>2</sub>O per acre applied in late autumn 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 aerification program. Studies have shown that spring dead spot is more severe with heavy thatch accumulation. One or two aggressive aerifications (coring) during the summer will increase juvenile growth and tiller production.

### **Fungicides**

Several fungicides are labeled for spring dead spot management. The products tested in the University of Kentucky Turf Research Program have given inconsistent control, and sometimes they have provided no measurable disease suppression. However, some success with fungicide application has been achieved in some tests in neighboring states. In such 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.

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## **ADDITIONAL RESOURCE**

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

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**Photos:** Lee Miller, University of Missouri (1, 3) and MaryAnn Hansen, Virginia Tech (2), Bugwood.org

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