Bermudagrass Spring Dead Spot

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**Introduction**

Spring dead spot (SDS) is generally considered to be the most significant disease of bermudagrass. Spring dead spot disease can show up each year on bermudagrass home lawns; however, it tends to be more prevalent on intensively managed bermudagrass. Low cutting height, soil compaction, high pH, over-fertilization and thatch accumulation may contribute to the onset of the disease. This disease becomes evident at spring green-up time during March or April in Arkansas. Although several root-infecting fungi have been identified as being responsible for the disease in other regions, *Ophiosphaerella korrae* seems to be the causal fungus in Arkansas.

**Symptoms**

The diseased area appears in the spring as well-defined, dead, circular patches that can range in size from a few inches to more than 3 feet in diameter (Figure 1). Symptoms may sometimes be confused with winterkill and injury from soil insects such as white grubs. Although spring dead spot symptoms may occur on bermudagrass lawns of all ages, they typically appear three to four years after the turf has been established. This disease primarily affects the roots. Death of the plants is believed to occur following normal low winter temperatures. The roots and stolons of diseased plants develop a dark brown to black-colored rot (Figure 2). Leaves become bleached, gray and straw-colored. The dead, sunken patches can often get larger year after year. Diseased areas may not fill in with bermudagrass until July or August. Bermudagrass recolonization is slow, but regrowth may mask disease evidence by the late summer (Figure 3). Weeds will often colonize the affected areas and slow the

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**Figure 1.** Sunken patches of spring dead spot in a lawn.

**Figure 2.** Black roots and stolons of infected bermudagrass.

**Figure 3.** Summer recovery is slow from spring dead spot.
recovery of the turfgrass. Additionally, application of certain preemergence herbicides in the spring may also slow recovery.

**Disease Cycle**

The fungi which cause SDS usually begin to colonize the roots, stolons and crowns of bermudagrass in the late summer or fall and again in the spring when soil temperatures range from 50º-70ºF. Even though root and crown infections occur in the fall, foliar symptoms do not show up until green-up in March and April of the following year. The fungus can overwinter as mycelium in infected roots and crowns of the turf. Excessive nitrogen fertilization during the late summer months tends to enhance symptom development during the following spring season.

**Management**

Proper plant nutrition and thatch management can play a pivotal role in disease management. Homeowners should be sure that there is an adequate potassium level in the soil. A regular soil test should be done to monitor potash and other elemental levels. Acidic soils tend to reduce the severity of the disease. If soil pH is high (> 7.0), ammonium sulfate can be used to lower soil pH in a range of 6.0 to 6.8. Heavy applications of fast-release nitrogen fertilizers should not be made in the summer following an outbreak of the disease. Nitrogen applications should be avoided after August in northern areas of Arkansas and not after September 15 in the southern part of the state if the lawn has a history of SDS. Nitrogen fertilization after mid-September may predispose the turf to many diseases. Fertilizing and irrigating too much can lead to thatch buildup, which favors disease activity. Management of thatch by verticutting and core aerification and soil compaction through core aeration should be integral parts of the disease control/prevention program.

There appears to be a close correlation between cold hardiness of bermudagrass varieties and disease susceptibility. When establishing a lawn, homeowners should consider growing tolerant varieties (Table 1). Bermudagrass varieties show substantial difference in their tolerance to the disease. However, the most tolerant varieties still get the disease but not as severely as the least tolerant varieties.

Removal of affected patches followed by resodding can be useful if there are only a few small diseased areas within the lawn. Fungicides which contain fenarimol, propiconazole, azoxystrobin, thiophanate-methyl or myclobutanil are labeled for disease control; however, control by these materials is often incomplete and inconsistent. Research in the Carolinas found fenarimol to be the most effective of these products. For effective control, emphasis should be placed on the cultural control aspects of fertility, irrigation and thatch management rather than relying exclusively on fungicides. For maximum effectiveness, these materials need to be applied according to label directions at least twice in the fall when the fungus becomes active. Homeowners should consider using a professional service to apply these materials appropriately.

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<tr>
<th>Relative Tolerance</th>
<th>Cultivars</th>
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<tbody>
<tr>
<td>Most</td>
<td>Midfield, Midiron, Midlawn, Patriot, Riviera, Tifsport, Yukon</td>
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<tr>
<td>Moderate</td>
<td>Cheyenne, Mirage, Sundevil II, Tifway (Tifton 419)</td>
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<tr>
<td>Least</td>
<td>Arizona common, Tifton 10, Numex Sahara, Princess 77, Pyramid, Sunbird, Savannah, Transcontinental, Tifgreen (Tifton 328)</td>
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**References**


Acknowledgment is given to Dr. Aaron Patton, former assistant professor - turfgrass specialist, University of Arkansas Division of Agriculture, as one of the original authors of this fact sheet.

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