# *Infection and Colonization of Bermudagrass by* Ophiosphaerella herpotricha*, a Causal Agent of Spring Dead Spot*

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**Objectives:**

1. To transform *O. herpotricha* to express fluorescent protein genes.
2. Evaluate infection and colonization of bermudagrass cultivars at different temperatures.
3. Evaluate differences in infection and colonization between bermudagrass cultivars that vary in disease susceptibility.

**Start Date:** 2006
**Project Duration:** three years
**Total Funding**: $59,684

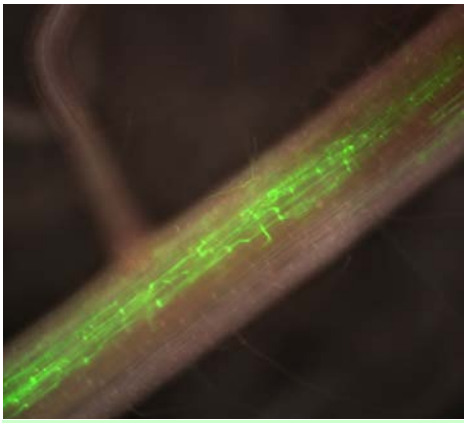
Spring dead spot is the most devastating and important disease of bermudagrass where it undergoes winter dormancy. The disease is caused by one or more of three fungal species in the genus *Ophiosphaerella* (*O. herpotricha, O. korrae, or O. narmari*). The disease results in unsightly dead patches on fairways, tees, and bermudagrass greens, giving way to the encroachment of weeds and costly management efforts to eliminate weeds and re-establish grass in the affected area. The overall goal of this study is to enhance our understanding of the interaction between *O. herpotricha* and its bermudagrass host, and how environmental and host factors influence this interaction.

We have inserted two different fluorescent reporter genes (red and green) into the fungus and examined root and stolon infection of various bermudagrasses. We were able to document differences in infection and colonization for roots and stolons of grasses that vary in their response to the disease. Colonization and necrosis of epidermal and cortical cells was observed for the cultivars 'Midlawn' (resistant) or 'Tifway' (susceptible), while the stele remained uninfected and intact.

For a *Cynodon transvaalensis* accession, epidermal and cortical cells and the stele were colonized, but became much less necrotic than the other cultivars. A confocal scanning laser microscope was also used to produce 3-dimensional images of the fungus in and on bermudagrass roots further supporting observations that the fungus grows intercellularly (between cells) in roots before eventually penetrating and infecting root cells.

These studies have permitted the formation of hypotheses as to the mechanism of plant death when colonized by the fungus and exposed to cool temperatures that induce dormancy. Conventional wisdom would predict that for a resistant plant the fungus would either not be able to gain entry or that fungal growth would be restricted due to the host mounting an aggressive defense response visible as hypersensitive defense necroses.

However, from our data, we hypothesize that a resistance response to spring dead spot could include the fungus living endophytically in root and stolon tissues without causing severe necrosis due to the inability of a grass to recognize and respond to the invading fungus.



*Root cortical cells were rapidly colonized in the interspecific hybrids 'Tifway' and 'Midlawn'.*

Conversely, susceptible plants' recognition of the fungus would elicit an extreme host defense response including a hypersensitive reaction (HR), whereby infected host cells undergo programmed cell death in an attempt to limit or stop pathogen growth. This has been shown to be the case with a few plant fungal disease interactions.

To test this hypothesis, we have inoculated a series of *C. dactylon*, *C. dactylon* x *C. transvaalensis* interspecific hybrids, and *C. transvaalensis* cultivars and collected samples to test for hallmark tags of the hypersensitive response.

*Spring dead spot is the most devastating and important disease of bermudagrass.*

## Summary Points

- Colonization and necrosis of roots were extensive in 'Tifway' and less in 'Midlawn'.
- The *C. transvaalensis* accession was extensively colonized, but with only limited necrosis.
- Disease resistance could be due to the inability of the plant to respond necrotically to the fungus.
- This information will be used to enhance bermudagrass disease resistance through traditional breeding efforts at Oklahoma State University.