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This publication includes lecture notes of papers presented at the 2000 New Jersey Turfgrass Expo. Publication of these lectures provides a readily available source of information covering a wide range of topics and includes technical and popular presentations of importance to the turfgrass industry.

This proceedings also includes research papers that contain original research findings and reviews of selected subjects in turfgrass science. These papers are presented primarily to facilitate the timely dissemination of original turfgrass research for use by the turfgrass industry.

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BENTGRASS DEAD SPOT: A NEW DISEASE OF GOLF COURSE GREENS AND TEES

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INTRODUCTION

First observed in Illinois during the fall of 1997, bentgrass dead spot was not recognized as a disease of creeping bentgrass (*Agrostis palustris* Huds.) until the summer of 1998 when it was identified in Maryland by P.H. Dernoeden. By 1999, the disease had also been reported in New Jersey, Pennsylvania, Virginia, Ohio, North Carolina, South Carolina, Missouri, and Texas. Bentgrass dead spot is caused by the fungus *Ophiosphaerella agrostis* and typically appears from July to October in the Northeast, upper Midwest, and Mid-Atlantic regions, and from April to October in the Southern United States (Dernoeden, 1999).

SYMPTOMS AND SIGNS

Symptoms of bentgrass dead spot first appear as reddish brown spots 0.5 to 1 inch in diameter. Spots quickly fade to a tan color and are often confused with dollar spot, copper spot, cutworm damage, or golf ball marks. Unlike dollar spot, which often produces copious amounts of foliar mycelium in the early morning hours, mycelium is not apparent on turf infested with bentgrass dead spot. When the disease is active, spots may have a bronzed outer margin, rarely coalesce, and are usually distributed randomly over the turf surface. To date, *O. agrostis* has been isolated from several creeping bentgrass cultivars (e.g., L-93, Providence, Penncross, Southshore, Penn G-2, SR 1119, Pennlinks, and Crenshaw) as well as from velvet bentgrass (*Agrostis canina* L.) and bermudagrass (*Cynodon dactylon* (L.) Pers.) (Dernoeden, 1999).

The causal agent infests leaf and crown tissue of susceptible plants. Upon close inspection with a magnifying glass or hand lens, prominent, black, flask-shaped fruiting bodies called pseudothecia can often be seen embedded in dead leaf and stem tissue. Ascospores contained within a pseudothecium may be ejected several feet resulting in new infection centers (Dernoeden, 1999).

CONDITIONS THAT FAVOR DISEASE

Bentgrass dead spot is favored by hot, dry weather. To date, the disease has only been observed on turf maintained at greens and tee height (i.e., less than 0.25 inch). On a recent greens construction study at Rutgers University, the disease was most prevalent on sites with high sand content. In this study, the incidence and severity of the disease decreased as the organic component of the mix (e.g. soil, sphagnum, or South Dakota peat) increased (Murphy et al., 1999). Currently, only turf less than 6 years old has been reported to be affected by bentgrass dead spot.

CULTURAL AND CHEMICAL CONTROL

Since bentgrass dead spot has only recently been identified, little is known about cultural management practices that may affect its develop-

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ment. In a study conducted in North Carolina, light applications of nitrogen (0.13 lb/1000 ft^2 per week) enhanced turf recovery once the disease had been suppressed with fungicides or became inactive in the fall (Wetzel and Butler, 1999). Avoiding stress and mechanical injury may also reduce the spread of this disease.

Few chemical control studies have been conducted for bentgrass dead spot and no fungicides are presently labeled for its control. From the limited work that has been published, it appears that chlorothalonil, iprodione, mancozeb, propiconazole, and thiophanate-methyl may aid in disease suppression (Wetzel and Butler, 1999). Once controlled with fungicides, however, infection centers may persist for 4 to 8 weeks.

During the summer of 2000, a large fungicide evaluation test was conducted by Rutgers faculty on a naturally infested green at the Charleston Springs Golf Course in Millstone, NJ. The test was designed to identify the fungicide classes that are most effective in controlling bentgrass dead spot (Table 1). Fungicides representing ten different chemical classes were applied every 14 days at various rates from 10 July to 11 September in water equivalent to 2 gal/ 1000 ft^2 with a CO_2 powered sprayer. Data were collected from 28 July to 13 September for disease severity. In general, fungicides within the benzimidazole (Clearys 3336 50W at 4.0 and 8.0 oz), dithiocarbamate (Fore Rainshield 80W at 8.0 oz), nitrile (Daconil Ultrex 82.5SDG at 5.0 oz), phenylpyrrole (Medallion 50WG at 0.5 oz) and phosphonate (Chipco Aliette Signature 80WG at 4.0 oz) chemical classes provided the most effective control of bentgrass dead spot (78 to 97% control, compared to untreated turf).

Of the sterol-inhibiting fungicides, only propiconazole (Banner MAXX 1.3MC at 1.0 and 2.0 fl oz) adequately controlled the disease (95% control), whereas myclobutanil (Eagle 40W at 0.6 oz) and triadimefon (Bayleton 50W at 2.0 oz) proved ineffective at the rates tested. Similarly, two experimental strobilurin fungicides (BAS 500 and 505) consistently suppressed the disease (96 to 97% control), while the strobilurins trifloxystrobin (Compass 50WG at 0.15 oz) and azoxystrobin (Heritage 50WG at 0.2 oz) provided poor to fair control (3 and 72% control, respectively) of bentgrass dead spot. Carboximide (ProStar 70WG at 2.2 oz) and phenylamide (Subdue MAXX 2MC at 1.0 fl oz) fungicides and a strain of *Bacillus subtilis* (Companion I at 4.0 and 8.0 oz) did not significantly control bentgrass dead spot, compared to untreated turf. Research is currently underway to evaluate turf recovery and germination after damaged areas are reseeded.

LITERATURE CITED

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Table 1. Fungicides evaluated for the control of bentgrass dead spot on a bentgrass putting green at Charleston Springs Golf Course, Millstone, NJ: 2000.

Chemical Family	Common Name	Trade Name	Comments
Benzimidazoles	thiophanate-methyl	Clearys 3336	Acropetal penetrant. Mode of Action: Fungicide binds tubulin subunits that result in mitotic arrest.
Carboximides	flutolanil	Prostar	Acropetal penetrant. Basidiomycetes control. Mode of Action: Blocks activity of certain respiratory enzymes.
Demethylation/ sterol-inhibitors	myclobutanil propiconazole triadimefon	Eagle Banner Bayleton	Broad-spectrum, acropetal penetrant. Mode of Action: Inhibits sterol (ergosterol) synthesis in fungal cell membrane.
Dicarboximides	iprodione	Chipco 26GT	Localized penetrant. Mode of Action: Affects DNA synthesis and lipid metabolism.
Dithiocarbamates	mancozeb	Fore, Formec, Dithane	Protectant fungicide. Mode of Action: Enzyme inactivation.
Nitriles	chlorothalonil	Daconil Ultrex Daconil Zn, Spectro*	Protectant fungicide. Mode of Action: Cell membrane toxicity.
Phenylamides	mefenoxam	Subdue Maxx	Acropetal penetrant. Mode of Action: Inhibits RNA synthesis.
Phenylpyrrole	fludioxonil	Medallion	Protectant fungicide. Mode of Action: Cell membrane toxicity and inhibits uptake of amino acids.

Table 1 (continued).

Chemical Family	Common Name	Trade Name	Comments
Phosphonates	fosetyl-Al	Aliette	Systemic fungicide. Mode of Action: Direct fungitoxic effect and enhancement of plant's natural defenses.
Strobilurins	azoxystrobin trifloxystrobin experimental	Heritage Compass BAS 500 and 505	Broad-spectrum, acropetal penetrant. Mode of Action: Blocks fungi from generating ATP.
Other:			
Biological fungicide	*Bacillus subtilis*	Companion I	Bacterial biocontrol agent and soil amendment.

*Spectro is a combination product containing thiophanate-methyl and chlorothalonil.