



Integrated Pest Management

IDENTIFICATION & MANAGEMENT OF TURFGRASS DISEASES

Plant Protection Programs
*College of Agriculture, Food
and Natural Resources*

**Archive version -- See
extension.missouri.edu**

This publication is part of a series of IPM Manuals prepared by the Plant Protection Programs of the University of Missouri. Topics covered in the series include an introduction to scouting, weed identification and management, plant diseases, and insects of field and horticultural crops. These IPM Manuals are available from MU Extension at the following address:

**Extension Publications
2800 Maguire Blvd.
Columbia, MO 65211
1-800-292-0969**

For further information, contact:

Brad Fresenburg
Division of Plant Sciences
University of Missouri
fresenburgb@missouri.edu

Authors

Barb Corwin

Turfgrass Diagnostics L.L.C.
7760 E Mt Zion Ch Rd
Hallsville, MO 65255
corwinb@socket.net
573-219-1156

Ned Tisserat

Bioagricultural Sciences and Pest
Management Department
Colorado State University
Plant Sciences 1177
Ft. Collins, CO 80523
Ned.Tisserat@ColoState.edu
970-491-6527

Brad Fresenburg

Division of Plant Sciences
University of Missouri
1-87 Agriculture Building
Columbia, MO 65211
fresenburgb@missouri.edu
573-442-4893

Unless otherwise identified, photos are by Barb Corwin.

Cover photo: Gateway National Golf Club, Madison, Illinois. Courtesy of Walters Golf Management.

In this publication, no endorsement of named products is intended, nor is criticism implied of similar products that are not mentioned.

On the World Wide Web

For this and other Integrated Pest Management publications on the World Wide Web, visit <http://ipm.missouri.edu>.

Production

MU Extension and Agricultural Information
Dale Langford, editor

© 2007 University of Missouri

Contents

The plant disease triangle 3

Using cultural practices to manage disease . 4
 Modifying the host 4
 Modifying the environment 6

Using fungicides to manage disease 7
 Classifying fungicides 7
 Choosing the right fungicide 9
 Applying fungicides correctly 9
 Preventing or delaying fungicide resistance 10

**Using other approaches to
 manage disease 10**
 Biological control. 10
 Biopesticides 11
 Plant defense activators 11

Tips for diagnosing turfgrass disease 12

Infectious disease

Anthracnose basal rot 14
 Ascochyta leaf blight 16
 Brown patch 18
 Copper spot 20
 Dead spot 22
 Dollar spot 24
 Drechslera leaf spots 26
 Fairy ring 28
 Gray leaf spot 30
 Gray snow mold 32
 Large patch 34
 Leaf and sheath spot 36
 Microdochium patch 38
 Necrotic ring spot 40
 Pythium foliar blight. 42
 Rust. 44
 Spring dead spot. 46
 Summer patch. 48
 Take-all patch 50
 Yellow patch 52
 Yellow tuft 54

Establishment and maintenance of turfgrasses is a multibillion dollar industry in the United States for good reason. Turfgrasses don't just add beauty to one's surroundings. Natural grasses provide safe and attractive playing surfaces for sports and other leisure activities. Vigorous, well-rooted turfgrass stands also protect local watersheds by filtering out harmful substances and reducing soil erosion and runoff. Turfgrasses cool the immediate environment in the heat of the summer and purify the air by removing carbon dioxide and releasing oxygen.

Millions of dollars are spent annually in the United States on fungicides to prevent or arrest turfgrass disease development on golf courses, high-maintenance lawns and other landscapes. Additional money is spent to repair and renovate severely damaged turf. Accurate diagnosis is the key to managing turfgrass disease in an economically and environmentally sound manner.

There are two types of pathogens that cause plant disease: (1) infectious or biotic and (2) noninfectious or abiotic. Most of the common infectious turfgrass diseases are caused by plant pathogenic fungi. The symptoms of these fungal diseases and factors favoring pathogen development have been well described. With some background information, the identification of fungal diseases is a straightforward process.

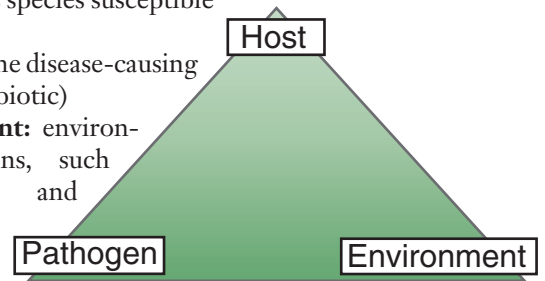
Cultural practices such as mowing, aeration and irrigation or the misuse of herbicides, growth regulators, fungicides and fertilizers can cause turfgrass injury or decline. These abiotic plant diseases are often the most difficult to diagnose. Disease may also result from a complex of organisms in combination with environmental factors that reduce host plant vigor.

This publication is designed to be a useful reference for diagnosticians, turfgrass managers, industry representatives and others who want to learn how to diagnose and manage common turfgrass diseases caused by plant pathogenic fungi. Expert help in turfgrass disease diagnosis can be obtained from Plant Diagnostic Clinics at land grant universities or from the private diagnostic labs specializing in turfgrass disease diagnostics.

The plant disease triangle

The plant disease triangle, which illustrates how disease develops in a plant population, is a useful concept to help understand and manage turfgrass diseases. For turfgrass disease to occur, the following three components must all be present:

- **The host:** a grass species susceptible to the pathogen
- **The pathogen:** the disease-causing agent (biotic or abiotic)
- **The environment:** environmental conditions, such as temperature and moisture that favor pathogen development or reduce host vigor.



Identifying the grass species involved is an important clue for identifying the probable cause. Some diseases occur more commonly on one species than another. After you have identified the turfgrass species showing symptoms, refer to Table 1 (page 4) for a list of the most common turfgrass hosts for the diseases covered in this publication. Cross reference to Table 2 (page 5) to check the prevailing temperature and time of year. The section on Tips for Diagnosing Turfgrass Disease (pages 12–13) can also help with the diagnosis.

Potential turfgrass pathogens are present wherever turfgrass is grown. But manipulating the other two components of the disease triangle can often provide effective disease management. Before applying fungicides, consider modifying the host, modifying the environment, or both. These considerations can be combined in a cultural disease management program that includes decisions related to selection of adapted and resistant turfgrass species and cultivars, fertility, mowing, water management, thatch removal and core cultivation.

Table 1. The most common turfgrass hosts of selected turfgrass diseases.

	anthracnose basal rot	Asochyta leaf blight	brown patch	copper spot	dead spot	dollar spot	Drechslera leaf spot and melting out	Drechslera red leaf spot	fairy ring	gray leaf spot	gray snow mold	large patch	leaf and sheath spot	Microdochium patch	necrotic ring spot	Pythium foliar blight	rust	spring dead spot	summer patch	take-all patch	yellow patch	yellow tuft (downy mildew)
Cool-season grasses																						
Annual bluegrass	●		●			⊙			●		●		⊙	●	⊙				●		⊙	
Creeping bentgrass	●		●	●	●	●		⊙	●		●		⊙	●		●				●	●	●
Fine fescue			⊙				●		●		⊙				⊙		⊙		●			
Kentucky bluegrass		●	⊙			●	●		●		⊙		●	⊙	●	⊙	●		●		⊙	●
Perennial ryegrass		⊙	⊙			●	●		●	●	⊙		●	⊙		●	●					●
Turf-type tall fescue		⊙	●			⊙	●		●	⊙	⊙					⊙	●					
Warm-season grasses																						
Bermudagrass				⊙	⊙	⊙	⊙		●			⊙							●			
Buffalograss						⊙	⊙		●			⊙							⊙			
Zoysiagrass				⊙		⊙	⊙		●		●						●					●

Notes:

● = most likely host ⊙ = occasional host

1. Fairy rings can occur wherever grass is grown, because they are associated with either the thatch or the soil, or both, rather than the grass itself.
2. Pythium blight on creeping bentgrass putting surfaces is rare, but it does occur on bentgrass collars and fairways.
3. Summer patch was recently reported as a disease of creeping bentgrass in North Carolina. Tredway, L. P. 2004. First report of summer patch of creeping bentgrass caused by *Magnaporthe poae* in North Carolina. Plant Disease 89:204.

Using cultural practices to manage disease

Modifying the host

Selecting turfgrass species and cultivars. Choose turfgrass species and cultivars that are adapted to site conditions and local climate. The National Turfgrass Evaluation Program (NTEP) was organized to evaluate cultivar performance of turfgrasses over wide geographic areas of the United States. Information in the NTEP reports is a good source for identifying regional adaptation and relative disease resistance. Visit <http://ntep.org> to review these reports. MU publication G6772, *Cool-Season Grass Cultivars for Athletic Fields*, presents ratings for Kentucky bluegrass, tall fescue and perennial ryegrass. Although the focus of the publication is athletic fields, the listings can be helpful in selecting cultivars for home lawns as well. It is often difficult for individuals to find seed of recommended cultivars. A good approach is to list what local retailers have available and then cross-reference to the cultivars recommended for your location.

Some diseases can be completely avoided by selecting or substituting grass species that are not susceptible to certain pathogens. For example, the severity of summer patch in Kentucky bluegrass lawns or golf green surrounds can be reduced by overseeding with tall fescue.

Fertilizing. Healthy, vigorous turfgrass often withstands infectious disease and adverse environmental conditions. A fertility program that promotes turfgrass vigor is an important part of a sound disease management program. Although the specifics of any program must be left to the turfgrass manager because of the on-site differences in use, expectation and budget, there are several generalizations that can be made. Decisions on amount and type of nutrients to apply should be based on the level of available nutrients in the soil and the plant's nutrient status as determined through soil testing and plant analysis.

For cool-season grasses, most of the nitrogen requirement should be met in the fall. Heavy spring applications of nitrogen result in lush, dense growth that is more disease prone. Growth of foliage at the expense of roots will

Table 2. The most likely temperature ranges for infection or symptom expression of selected turfgrass diseases.

	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec
Anthracnose basal rot												
Ascochyta leaf blight												
brown patch												
copper spot												
dead spot												
dollar spot												
Drechslera leaf spots • Drechslera leaf spot and melting-out • red leaf spot												
fairy ring												
gray leaf spot												
gray snow mold												
large patch												
leaf and sheath spot												
Microdochium patch												
necrotic ring spot												
Pythium foliar blight												
rust												
spring dead spot												
summer patch												
take-all patch												
yellow patch												
yellow tuft												
Note:	This table is based on the authors' experience. Time periods are expected to vary by region.											
Color key:		infection period			symptom expression			infection period and symptom expression				

also make the turfgrass more prone to decline situations during the heat and moisture stress periods frequently encountered in the summer.

For warm-season grasses, nitrogen requirements should be spaced out over the summer months (May-August) when these grasses are actively growing. Late-summer to early-fall applications of nitrogen will make these grasses more susceptible to cold/cool weather diseases.

Mowing. Turfgrass plants mowed shorter than their optimal height of cut are, in general, more susceptible to disease. Optimal cutting heights for cool-season grasses range from 2.5 to 4.0 inches, depending on the species. Height of cut for warm-season grasses can range between 1 and 2 inches.

Frequency of cut should be determined by the “one-third rule” of mowing. Do not remove more than one-third of the leaf growth during a single mowing.

Mowing creates wounds through which fungi can enter the plant and infect it. Leaf cuts made by a sharp mower blade are cleaner and heal faster than the tearing and shredding caused by a dull mower blade. A dull blade inflicts more and bigger wounds that not only increase the potential for infection by turfgrass pathogens but also increase the potential for moisture stress. Observe leaf tips or grass clippings collected on the mower deck immediately after a mowing to determine the quality of cut. Use this as an indicator of when to sharpen blades.

Modifying the environment

Although ambient temperature, relative humidity and amount of precipitation cannot be controlled, there are ways to modify the local environment to reduce the negative impact of turfgrass disease. Development of many turfgrass diseases is favored by extended periods of leaf wetness. Extended periods of leaf wetness in the turfgrass environment are caused by dew, guttation (water droplets that form at the tips of grass leaves) and frequent irrigation or precipitation. Remove dew and guttation from grass leaves by dragging a hose across the surface, using a whipping pole or briefly irrigating it with large droplets, only long enough to remove the dew from the leaf surface. The goal of all three methods is to spread the concentrated dew or guttation droplets over a larger surface area so the turf canopy will dry faster

in the morning. Recent research results suggest that dew removal can improve the performance of certain contact fungicides.

Improper irrigation alone can create a disease problem that normally would not exist. Ideally, irrigation should be applied between midnight and early morning when the turfgrass canopy is normally wet. This avoids extending the duration of leaf wetness. Avoid frequent irrigation that also extends the duration of leaf wetness, especially when environmental conditions are favorable for *Pythium* foliar blight, brown patch and dollar spot. Generally, deep and infrequent irrigation is recommended so that some soil and leaf drying can occur, permitting a gaseous exchange between soil and atmospheric air.

Irrigation water should be tested regularly (every two to three years) for pH, soluble salt concentration, level of bicarbonates and other factors that influence irrigation water quality.

Adequate drainage is necessary for vigorous turfgrass growth. Turf areas that stay constantly wet because of poor drainage are prime targets for decline. Surface contouring and subsurface drainage are costly, but permanent solutions. Core cultivation and slicing are turf management practices that can be repeated during the year to temporarily increase infiltration rates and promote soil drying.

Increase light penetration and air movement by selectively pruning trees and shrubs. This will speed the drying of poorly drained areas and also reduce the humidity in localized turfgrass areas.

The negative impact of turfgrass disease can be reduced by good thatch control. Thatch is a layer of dead and living plant material located between the soil surface and green turf canopy. It is an excellent habitat for both active and dormant stages of many plant pathogenic fungi. When environmental conditions are optimum, fungi can rapidly grow and infect living turfgrass tissue.

Remove excess thatch when it accumulates to ½ inch or more in taller-mowed turf (1.5 to 4 inches) and ¼ inch in lower-mowed turf (below 1.5 inches). Dethatch when the turfgrass is actively growing, so it will recover quickly. Generally dethatching should occur in the spring and fall for cool-season grasses and midsummer for warm-season grasses.

Core cultivation is another tool for thatch control. On sand-based or modified sand-based putting greens, cores are generally removed and the holes are backfilled with sand. On other turf areas, the cores can be broken up and filtered back into the turf canopy to promote the activity of soil microbes that break down organic matter in the thatch layer.

Using fungicides to manage disease

Fungicides are a tool for managing turfgrass disease but are most effective when combined with cultural practices that reduce plant stress. Fungicide applications can be made before the onset of disease (preventive) or after disease development (curative). On high-visibility, high-profile sites such as golf course putting greens and surrounds, a preventive approach is usually desired. For diseases such as brown patch or dollar spot, experienced turfgrass managers can adopt a modified preventive approach by using the concept of sentinel plots to monitor disease potential in a local geographic area. For the professional lawn care operator, the sentinel plot could be a lawn on which brown patch tends to break first. For the golf course superintendent, the sentinel plots could be those greens that historically exhibit the first symptoms of dollar spot or brown patch. Once disease is observed in the sentinel plots, the turfgrass manager can then treat other lawns or other greens on the course preventively. Some diseases have to be managed before the onset of symptoms. Summer patch and large patch are just two examples. The decision to use a preventive or curative approach or a combination of the two ultimately depends on expectations, budget and the diseases in question.

Don't expect immediate improvement to the turfgrass following a curative fungicide application. This is because disease often occurs under conditions that are not favorable for optimum turfgrass recovery. Although suppression of the fungus will occur, some time may pass before the prevailing environmental conditions become favorable for growth and recovery of the turfgrass. Grasses can naturally recover from some diseases once environmental conditions favor growth of the turfgrass. Large patch of zoysiagrass and brown patch of

tall fescue are two examples. Owners or managers of high-visibility turfgrass areas are often unwilling to wait for recovery. Also, the thinned areas may be invaded by undesirable grasses, broadleaf weeds or algae.

Classifying fungicides

Fungicides are classified in several different ways. In Table 3, fungicides are classified in ascending order of FRAC code. The FRAC code was assigned by the Fungicide Resistance Action Committee, an international committee of scientists representing chemical manufacturers. Fungicides with the same FRAC code have the same or very similar biochemical mode of action, i.e., they affect their target fungi in the same way. Most registrants are voluntarily printing the FRAC code on product labels. This will make it easier for turfgrass managers to avoid repeated use of products with the same mode of action, which could result in the selection of resistant fungal populations.

Fungicides can also be classified on the basis of topical mode of action. Topical mode of action describes what happens to a fungicide when it contacts a plant. Fungicides have two basic topical modes of action — contact and systemic. Systemic fungicides are subdivided based on the direction and extent of movement inside the plant. There are three possible fates of systemic fungicides once they have entered a plant: xylem-mobile (translocated upward), phloem-mobile (translocated upward and downward) or limited translocation from the entry site (locally systemic).

Another category of fungicides includes those that have been registered as reduced risk by the Environmental Protection Agency

Applying fungicides correctly

For optimum fungicide performance —

- Know the targeted disease(s).
- Know the location of the pathogen (leaves, crown, roots).
- Know the product(s) and its mode of action.
- Choose a flat fan nozzle to produce either a fine to medium droplet for foliar disease or a medium to a coarse droplet for root and crown rotting disease.
- Know the tolerance for drift.
- Know the weight of the spray solution.
- Know the pressure range of the sprayer.
- Know the boom height and nozzle spacing.
- Know that the sprayer is properly calibrated.

Table 3. Basic fungicides labeled for management of turfgrass diseases.

FRAC code ¹	Group name	Chemical group	Common name	Topical mode of action ²
1	Methyl benzimidazole carbamate (MBC)	thiophanates	thiophanate methyl	XMS
2	Dicarboximide		iprodione	LS
			vinclozolin	LS
3	Demethylation inhibitors (DMI)	pyrimidines	fenarimol	XMS
		triazoles	myclobutanil	XMS
			propiconazole	XMS
			triadimefon	XMS
4	Phenylamide	acylalanines	mefenoxam	XMS
7	Carboxamide		boscalid	XMS
			flutolanil	XMS
11	Quinone outside inhibitor (QoI)	methoxy-acrylates	azoxystrobin	XMS
		methoxy-carbamates	pyraclostrobin	LS
		oximino acetates	trifloxystrobin	LS
12	Phenylpyrrole		fludioxonil	C
14	Aromatic hydrocarbon		chloroneb	C
			PCNB (quintozene)	C
		1,2,4-thiadiazoles	etridiazole	C
19	Polyoxin	peptidyl pyrimidine nucleoside	polyoxin D	LS
28	Carbamate		propamocarb	LS
33	Phosphonate	ethyl phosphonates	phosphorous acid fosetyl-Al	PMS PMS
M	M3	dithio-carbamate	mancozeb thiram	C C
M	M4	phthalimides	captan	C
M	M5	chloronitriles	chlorothalonil	C

Notes:

1. Numbers or letters assigned by the Fungicide Resistance Action Committee (FRAC). Fungicides with the same FRAC code have the same or similar mode of action. For a complete FRAC code list: <http://frac.info>
2. Classification from Paul Vincelli, University of Kentucky
C = contact, LS = locally systemic, XMS = xylem-mobile systemic, PMS = phloem-mobile systemic

(EPA). Several fungicides labeled for use on turfgrass have been registered as reduced risk because of low impact on human health and low toxicity to nontarget organisms, including birds, fish and other plants (Table 4). Other criteria that are considered under the reduced risk registration protocol include low potential for groundwater contamination, low use rates, low pest resistance and compatibility with integrated pest management (IPM) practices.

Table 4. Reduced-risk fungicides labeled for use on turfgrass, as of 2007.

Common name	Trade name	Registrant
azoxystrobin	Heritage	Syngenta
boscalid	Emerald	BASF
fludioxonil	Medallion	Syngenta
mefenoxam	Subdue Maxx	Syngenta
trifloxystrobin	Compass	Bayer

Choosing the right fungicide

In today's turfgrass fungicide market, there is a high level of competition for market share of fungicide dollars. Although there are numerous trade names to choose from, many of these are competing products with the same active ingredient or with the same or similar mode of action. Although active ingredients may be the same, product formulations can be quite different, leading to the possibility of varying levels of efficacy. In addition, the level of company support for products varies widely from one company to the next.

Developing a fungicide plan of action can help with fungicide choice. To help develop the plans, information on the basic fungicides labeled for management of turfgrass diseases is presented in Table 5 as a pullout section. A fungicide plan of action is a season-long program outlining the targeted diseases, application dates and products. The fungicide plan of action is based on the disease history, fungicide budget and expectations of the facility. The environment and the grass species and cultivars you are managing will also influence your plan. Ideally the fungicide plan of action should be developed side-by-side with the plans for other chemical inputs such as fertilizers, herbicides and growth regulators and scheduled core cultivation and other maintenance practices. A well thought out fungicide plan of action makes the most of fungicide dollars.

Why develop a plan?

- To avoid negative interactions with other inputs such as herbicides and growth regulators
- To lower the threat of developing fungicide resistance
- To reduce the amount of fungicide applied
- To achieve better value for your fungicide dollars

Applying fungicides correctly

Once you have developed a fungicide plan of action, there are other factors that need to be considered to ensure optimum product performance. Thorough coverage of the targeted part of the plant (e.g., foliage crowns or roots) must be achieved and the correct amount of product according to label recommendations must be applied. Two of the common causes of poor product performance are spray droplets that are too large to provide the complete coverage needed to control certain diseases and spray volumes that are too low.

Droplet size is highly dependent on nozzle type. Droplet size at a given pressure influences the amount of product applied per unit area, the uniformity of coverage in that area and the off-target drift potential. Reducing droplet size by half will result in about eight times more droplets per unit area. For example, applying a fungicide in a water volume of 44 gallons/acre through a nozzle that delivers 800-micron droplets distributes about 88 droplets per square inch, whereas a nozzle that delivers a 400-micron droplet will result in about 704 droplets per square inch. The smaller the droplet, the more even the distribution of the product in the turf canopy (Figure 1), but the greater the drift potential. Most nozzle manufacturers provide color-coded tables that list droplet sizes for different nozzles at various pressures.



Figure 1. Daconil Ultrex applied at 1.8 oz/1,000 in 41 gal/acre. Left: coarse droplets; right: fine droplets.

D. Shepard, Syngenta Professional Products

There are two basic types of nozzles: flat fan and hollow cone. A flat fan nozzle is the most widely used in the turfgrass industry and under varying ranges of pressure is a good choice for applying both contact and systemic fungicides. Flat fans (specifically designed for low pressures) offer good control of spray drift when operated at low pressures. Nozzles can operate at different pressure ranges, but 30 to 60 psi is typical for turfgrass applications.

There are two types of hollow cone nozzles, one producing very fine droplets and one producing very coarse droplets. The fine-droplet hollow cone distributes droplets finer than flat fan nozzles but has no overlap of the spray pattern. The finer droplets are prone to drift and there is lack of uniform coverage if the nozzle is used on sites other than level ground. The raindrop nozzle is a coarse-droplet hollow cone that is less prone to drift; however, this nozzle results in less uniform coverage.

When fungicides are mixed with water, the solution becomes heavier than water alone (8.34 lb/gallon). Heavy liquids will form narrower spray bands and larger droplets, resulting in poorer coverage. Liquids less dense, or lighter, than water can form wider spray angles and increase flow rates. Nozzle manufacturers have conversion charts for solutions heavier or lighter than water.

Coverage is also determined by spray volume. Nozzle manufacturers provide tables that list spray volumes per unit area for each nozzle at different pressures and speeds. Recent research has shown that a foliar disease such as dollar spot is suppressed effectively by fungicides (both contact and systemic) applied in a spray volume as low as 1 gallon per 1,000 square feet (44 gallons/acre). In contrast, fungicides (contact and systemic) targeting brown patch were most effective when applied at 2 to 4 gallons per 1,000 square feet (87–175 gallons/acre). Fungicides targeting root- and crown-infecting pathogens should be applied at rates of at least 2 gallons per 1,000 square feet; some product labels suggest 2 to 4 gallons per 1,000 square feet. Because most fungicide plans of action are designed to cover more than one disease at a time, turfgrass managers should, as a general rule, use a flat fan nozzle at a pressure sufficient to apply at least 2 gallons per 1,000 square feet.

Finally, make sure that the sprayer is properly calibrated. For step-by-step instructions, refer to MU publication G6751, *Calibrating Sprayers and Spreaders for Athletic fields and Golf Courses*.

Preventing or delaying fungicide resistance

Repeated use of fungicides with similar modes of action can result in the selection of fungal populations with resistance to the fungicide. Strains of the dollar spot fungus resistant to cadmium-based, MBC and DMI fungicides and iprodione have been reported. *Pythium* species resistant to metalaxyl and anthracnose fungi resistant to MBC and QoI fungicides have also been reported. The likelihood of selecting for resistant fungal strains depends on the fungicide mode of action and the biology of the fungus. Table 5 presents the relative risk of resistance for basic fungicides labeled for control of turfgrass diseases.

Plant pathologists generally agree that certain practices can delay the selection of resistant fungal strains. Here are some examples:

- Do not rely on fungicides alone. Combine fungicide use with cultural practices that reduce disease severity.
- Use contact fungicides alone or in combination with systemics as part of the management program.
- Use fungicides on a preventive, rather than curative, basis.
- Rotate or mix systemic fungicides with different modes of action. Repeated use of the same or a similar fungicide selects resistant members of the population.

Using other approaches to manage disease

A number of other strategies may play a role in a turfgrass disease management program, especially when used in combination with other, more traditional methods.

Biological control

Research efforts have looked at two ways to achieve effective biological control practices in a turfgrass disease management program. One approach is to introduce microbial inoculants that have a demonstrated negative effect on target plant pathogenic fungi. The second is to

stimulate resident populations of competitive or antagonistic microorganisms through the application of organic amendments and other microbial food sources.

Research trials with various microbial inoculants, including fungi and bacteria, have been ongoing and well supported by the United States Golf Association, Golf Course Superintendents Association of America, industry, state turfgrass organizations and the federal government for more than 25 years. Although many of the microbes have given promising results in greenhouse or small-scale field trials, few have been successfully developed as commercial products. Of the 33 microbe–disease combinations studied as biological control agents in a turfgrass system (summarized by Eric Nelson, Cornell University, in a 2003 review), only six have been registered as microbial pesticides by the EPA. The six organisms include two fungi (*Trichoderma harzianum* strain 1295-22 and *Gliocladium catenulatum* strain J1446), three bacteria (*Bacillus licheniformis* strain SB3086, *Bacillus subtilis* strain GB03 and *Pseudomonas aureofaciens* strain TX-1) and one actinomycete (*Streptomyces lydicus* strain WYEC 108). Since the first product was registered in 1996, there have been numerous changes in trade names and manufacturers, and the availability of these six products is limited in the United States. Visit <http://epa.gov/pesticides/biopesticides> for the most up-to-date registration information. One of the major limiting factors to successful use of microbial inoculants in a turfgrass system is the difficulty of delivering and sustaining adequate populations of the microbial inoculant in the turfgrass environment.

The second method of biological control — promoting resident microorganisms — gives promise for a more sustainable approach. This approach ranges in scope from composting with different organic amendments to applying food sources such as molasses, with the ultimate goal of “feeding” or promoting a diversity of naturally occurring soil microorganisms that suppress disease. Again, the level of success with these methods has varied considerably and is hampered on a commercial scale by difficulties

in achieving high-quality, consistent product from one batch to the other.

Biopesticides

Biopesticides include the microbial pesticides mentioned above as well as biochemical pesticides and plant-incorporated protectants. Biochemical pesticides are naturally occurring substances in contrast to conventional products that are generally synthetic. One such product registered for use on turfgrass by Arvesta Corporation and licensed to Cleary Chemical is Endorse. Endorse is polyoxin D zinc salt, a naturally occurring product produced by a soilborne bacterium. The bacterium is grown commercially and the polyoxin D zinc salt is extracted and formulated as the fungicide Endorse. Another example is hydrogen dioxide (registered as Zerotol by Biosafe Systems). Essential oils and other plant extracts are included as biochemical pesticides. A more complete listing of registered biopesticides by active ingredient can be found on the EPA Web site cited above. Unfortunately the list is not searchable by crop, so finding all the products labeled for use on turfgrass is difficult.

Plant-incorporated protectants (PIP) are pesticides produced within a plant as the result of the addition of foreign DNA. There are currently no PIP products registered for use on turfgrass.

Plant defense activators

Plant defense activators are naturally occurring or synthetic products that have been shown to induce systemic acquired resistance in various plants. Rather than impact the plant pathogen directly, these products enhance or stimulate a plant’s own natural defense mechanisms. The efficacy of several such products against brown patch and dollar spot has been evaluated in recent years. Although some disease suppression has been shown, the level of disease control with these products alone has not been acceptable. However, some plant defense activators show promise when used in combination with fungicides or cultural practices, such as the application of foliar nitrogen.

TIPS FOR DIAGNOSING TURFGRASS DISEASE

Accurate diagnosis is the key to managing turfgrass disease in an environmentally and economically sound manner. When the cause is not accurately identified and management practices and control measures are not developed accordingly, inputs are wasted and high reestablishment costs may be incurred.

Recognize that there are two types of pathogens that cause plant disease: (1) infectious or biotic and (2) noninfectious or abiotic. Most of the following steps can help you learn to diagnose the cause of turfgrass problems:

The common turfgrass diseases are caused by plant pathogenic fungi. Plant parasitic nematodes can also cause disease in some situations. Keep in mind that insect infestation can also result in damage to turfgrasses.

1. Identify the host plant.

Many infectious diseases of turfgrass are caused by pathogens that are host specific. For example, summer patch can be a severe problem on Kentucky bluegrass, some fine fescues and *Poa annua*, but has little or no effect on perennial ryegrass, tall fescue or bentgrass (Figure 1). Another example is large patch, which affects zoysiagrass, bermudagrass and buffalograss, but not cool-season grasses (Figure 2). Sometimes undesirable grasses such as *Poa trivialis* look diseased to the untrained eye (Figure 3).

2. Note prevailing environmental conditions before and during disease outbreak.

Many turfgrass pathogens develop within a fairly narrow range of temperature and moisture. Learning the environmental conditions that favor infection and disease development will help in disease diagnosis and assist in the timing of cultural management inputs and preventive fungicide applications.

The environment also includes such factors as location in relation to air movement and amount of sunshine, physical characteristics of the root zone and depth of thatch.

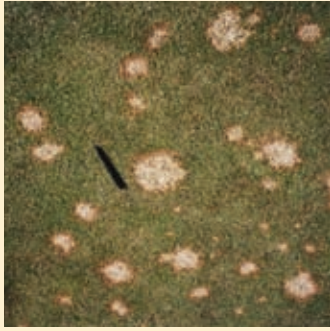
3. Observe the on-site pattern of damage.

The pattern associated with infectious disease often appears random (Figure 4). Infectious disease is often limited to a single species in the stand. In contrast, disease caused by abiotic factors often affects multiple species. The pattern of damage caused by an abiotic pathogen may be uniform or coincide with a recent cultural practice (Figure 5). Disease on putting surfaces often appears patchy because of species or clonal differences in disease susceptibility or response to chemicals (Figure 6).



D. Minner

Figure 1. Summer patch symptoms on Kentucky bluegrass with unaffected perennial ryegrass in the center of some patches.



M. Matthews

Figure 2. Large brown patch of zoysiagrass on two adjacent tees with unaffected Kentucky bluegrass in the middle.



Figure 3. Yellow *Poa trivialis* in a stand of Kentucky bluegrass.



N. Tisserat

Figure 4. Random pattern of *Microdochium* patch on a bentgrass putting surface.

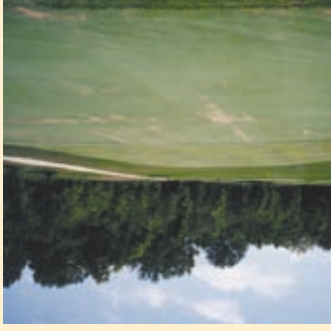


Figure 5. Pattern of damage caused by fertilizer burn on a bentgrass putting surface.

Figure 6. Summer patch of *Poa annua* on a mixed bentgrass/*Poa annua* green. Note yellowing of *Poa* surrounding unaffected bentgrass.

4. Note the symptoms.

A symptom is a plant's visible response to infection or to impact by an abiotic pathogen, causing the turfgrass manager to recognize that there is a problem. Symptoms are first observed as one looks out over the turf area (Figure 7). To identify the problem, it is necessary to take a close

look at individual grass plants at the transition zone between healthy and affected turf. Look for symptoms on individual plant parts such as leaf blades, sheaths, crowns and roots (Figure 8). It is important to take a close look at the affected grass (Figure 9). Always try to compare affected and healthy turfgrass (Figure 10).



Figure 7. Symptoms of brown patch on tall fescue as one looks out over the turf area.



Figure 8. Foliar symptoms of brown patch on tall fescue at the transition zone between healthy and affected turf.



Figure 9. Always take a close look at individual grass plants.

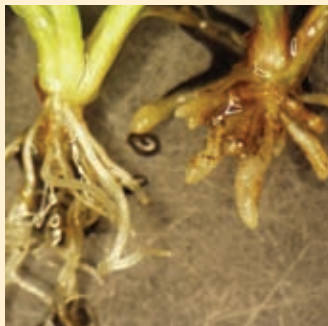


Figure 10. Root system of a healthy bentgrass plant (left) and a root system affected by preemergent herbicide (right).



Figure 11. Mycelium of *Pythium* sp. at the edge of the infection site under heavy morning dew.



Figure 12. Hyphae and lobed sporangium (fruiting body) of a *Pythium* sp.

5. Look for signs of disease.

Signs of disease are visible pathogen structures. Some are visible to the naked eye, while others must be observed with a hand lens or a compound microscope. Examples of signs are:

- **Mycelium** – mass of fungal vegetative growth often visible to the naked eye (Figure 11).
- **Hyphae** – individual strands that are the vegetative growth of the fungus (Figure 12).
- **Bulbils** – hardened masses of fungal tissue that aid in survival during unfavorable conditions (Figure 13).
- **Fruiting bodies** – spore-bearing structures of the fungi. These are extremely variable in size, shape and type of spores produced. They range from the macroscopic mushrooms and toadstools associated with the fairy ring fungi to structures visible only with a compound microscope (Figures 12, 14).
- **Spores** – Reproductive units, which give rise to new individuals, are generally too small to be of value to the turfgrass manager in disease diagnosis. Plant pathologists use the size, shape, color and other characteristics of spores to aid in disease identification (Figure 14 B).

Before you look for signs of disease, ask for information about fungicide application history. Recent fungicide application may have suppressed the pathogen, making it difficult to detect. Other information that can be helpful when making a diagnosis includes history of herbicide use, growth regulator program, insecticide use, fertility program, soil pH, irrigation water source and irrigation water quality.

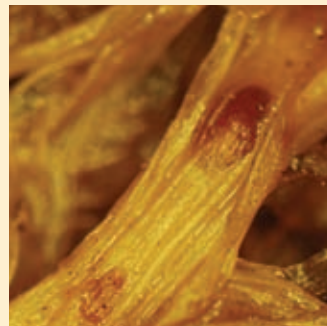


Figure 13. Bulbils of *Rhizoctonia zeae* imbedded in the leaf sheath of perennial ryegrass.

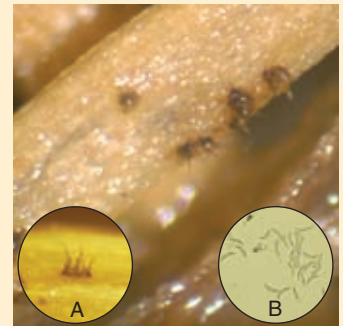


Figure 14. Acervuli (fruiting bodies) of the anthracnose fungus. *Inset A*: Setae (sterile hairs) that surround the acervuli. *Inset B*: Conidia (spores).

Anthracnose basal rot

Pathogen: *Colletotrichum cereale*

Hosts: creeping bentgrass, annual bluegrass

Anthracnose basal rot is a destructive crown rotting disease of creeping bentgrass and annual bluegrass on putting greens. On mixed bentgrass/annual bluegrass putting greens, the causal fungus infects one species or the other but rarely both (Figure 1). In the Midwest, the disease has been observed more often on creeping bentgrass than on annual bluegrass.

Colletotrichum cereale is commonly observed on senescing tissue of many grass species, including those grown as forage crops. When diagnosing anthracnose basal rot on putting greens, it is important to differentiate between an active infection and a secondary infection on damaged, senescing or dead tissue.



Figure 1. Anthracnose basal rot of annual bluegrass on a mixed bentgrass/annual bluegrass putting green.



Figure 2. Anthracnose basal rot of annual bluegrass on a mixed bentgrass/annual bluegrass green.

Symptoms and signs

From a distance, infected annual bluegrass appears unthrifty and has a yellow or bronze cast (Figure 2). Infected bentgrass first appears purple to reddish brown, resembling a dry spot (Figure 3). The symptoms on the green often appear as a patch, because the fungus differentially infects annual bluegrass or susceptible selections (clones) of creeping bentgrass on the putting surface. Bentgrass within the affected patches is thinned.

Colletotrichum cereale produces acervuli (fruiting structures) on leaves, stems and crowns (Figure 4). Acervuli are surrounded by sterile setae (black, hairlike structures) (Figure 4, inset A). Setae are easily visible with a 20X hand lens, so turfgrass managers and other turfgrass professionals often use the observation of setae to diagnose the disease. To determine if the disease is active, however, it is necessary to examine the acervuli for viable conidia (spores), which are produced in a gelatinous matrix on the surface of the acervuli. The conidia are single celled and crescent shaped and can only

be seen under high magnification (Figure 4, inset B). This usually requires the assistance of a turfgrass pathologist. Another indication that the disease is active is the observation of sporulation in green tissue (Figure 5).

Conidia germinate to form darkly pigmented appressoria (infection structures). The presence of appressoria under the leaf sheaths on green tissue is another indication that the disease is active. Appressoria are abundant on lower leaf sheaths and appear through a hand lens as small pepperlike dots (Figure 6). A close-up reveals the germ pore (Figure 7) through which *C. cereale* penetrates host tissue.

Crowns, old stems and roots of bentgrass or annual bluegrass appear black due to the presence of mycelial mats (aggregate hyphae) and other fungal structures in this tissue (Figure 8).

Conditions

Anthracnose basal rot most often develops during the warm, humid weather of midsummer, but it may occasionally occur during cool weather. Disease development is usually asso-



Figure 3. Anthracnose basal rot of creeping bentgrass caused by *Colletotrichum cereale*.

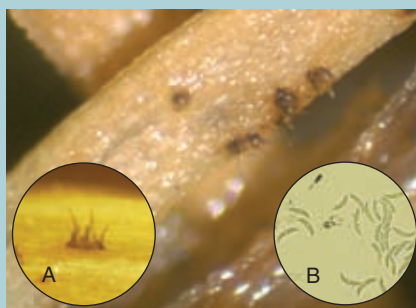


Figure 4. Conidial masses of *Colletotrichum cereale* produced in acervuli. Inset A: Setae surrounding an acervulus. Inset B: Conidia (spores) under a compound microscope.



Figure 5. Acervuli of *Colletotrichum cereale* on green leaf sheaths of creeping bentgrass.

Management

Anthracnose basal rot is one of the more difficult diseases to control, especially after symptoms appear. The best strategy to prevent outbreak of this disease is to combine cultural practices that reduce turfgrass stress with a preventive fungicide program.

Turfgrass grown at optimal nutritional levels is less likely to be damaged. Therefore maintain balanced fertility. Do not starve the turf, especially of nitrogen, during the summer months. Light nitrogen application (0.1 lb/1,000 ft²) during the summer may help the turfgrass withstand stresses and recover quickly from anthracnose basal rot damage.

Low, frequent mowing can enhance disease development. Recent studies indicate that raising the mowing height, even to $\frac{5}{32}$ inch, can help reduce turfgrass stress, which in turn can decrease anthracnose basal rot severity. Avoid double cutting on greens with a history of the disease. Mow the perimeter of the green every other day to prevent compaction. Don't mow when the greens are wet if *C. cereale* is active. Also

suspend topdressing activities, because physical damage can result in more severe infection. Some research results suggest that use of the growth regulator trinexapac-ethyl during the season can reduce disease severity on annual bluegrass. Under heavy disease pressure, however, it is best to suspend growth regulator programs to promote recovery.

Anthracnose basal rot tends to be more severe on putting greens with poor air movement and slow infiltration rate. On greens with a history of the disease, consider a rigorous core cultivation program in fall and spring. Compacted greens may be periodically cultivated by spiking, slicing, or hydro-jecting, but avoid excessive injury to the turf during periods of stress. Don't overwater putting greens either by irrigation or by supplemental hand watering. Excessive soil moisture can damage roots and decrease the rate of photosynthesis. This puts the grass into a decline and predisposes it to infection. Monitor greens regularly for dry spots, especially in the

early spring. Anthracnose basal rot often begins in areas that have undergone moisture stress. Application of a wetting agent can help with moisture management.

On greens with a history of anthracnose basal rot, a preventive fungicide program is necessary. In most regions, it is advisable to begin this program in mid-April. Refer to Table 5 for a list of fungicides labeled for anthracnose control. All products work best when applied on a preventive or early curative schedule. Applications after development of severe damage to putting greens are not very effective. The most rapid improvement from anthracnose damage occurs following significant (cooler) weather changes, which promote turfgrass recovery. Some strains of *C. cereale* resistant to MBC, DMI and QoI fungicides have been reported. Avoid sequential applications of these products and tank mix with a contact fungicide to reduce the selection of resistant populations.

Reports suggest that the preemergent herbicides bensulide and dithiopyr increase disease severity.

ciated with predisposing factors such as high temperature and humidity, excessive soil moisture, low mowing heights and soil compaction, all of which stress the turfgrass. The disease also tends to be more severe in situations where the greens have been allowed to dry out. The

fungus has been observed to overwinter in crowns and roots. Infected but symptomless plants may survive until they are exposed to stress conditions.

Symptoms can be confused with those of

- copper spot
- red leaf spot
- moisture stress
- heat stress

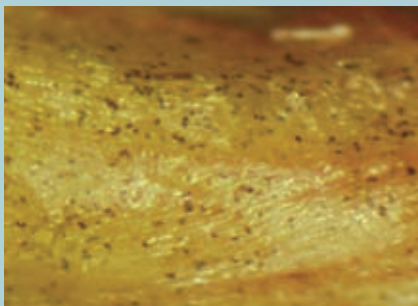


Figure 6. Appressoria of *Colletotrichum cereale* appear as pepperlike dots on green leaf sheaths of creeping bentgrass.

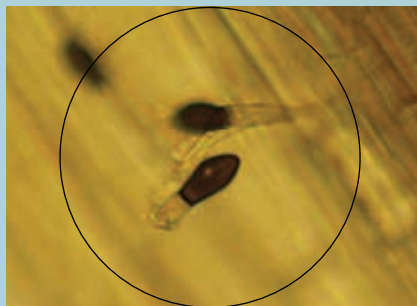


Figure 7. Close-up of appressorium (infection structure) of *Colletotrichum cereale* showing germ pore.



Figure 8. Mycelial mats and other fungal structures form in the basal crown region of infected creeping bentgrass.

Ascochyta leaf blight

Pathogen: *Ascochyta* spp.

Hosts: Kentucky bluegrass, tall fescue, perennial ryegrass

Ascochyta leaf blight results in the rapid development of large irregularly shaped, straw-colored patches on Kentucky bluegrass and occasionally on tall fescue and perennial ryegrass during the summer. Because *Ascochyta* spp. are primarily foliar pathogens, diseased turfgrass usually recovers relatively quickly. Environmental conditions that trigger Ascochyta leaf blight are poorly understood.

Symptoms and signs

Ascochyta leaf blight symptoms can develop throughout the growing season but are more common during hot, droughty periods that were preceded by cool, rainy conditions. Large irregular patches of turf rapidly turn a straw-color and appear dead (Figure 1). The overall appearance of the disease resembles drought stress, except that the symptoms of Ascochyta leaf blight appear quickly (sometimes overnight). Although the blighting within an area appears complete from a distance, healthy leaves are interspersed within the patch (Figure 2). Blighting is usually restricted to the leaves. Bluegrass crowns and roots typically are not killed.

Leaves infected with *Ascochyta* spp. often exhibit a bleached tip dieback that extends about a third to halfway down the leaf blade (Figure 3). The margin between healthy and diseased tissue is abrupt and slightly pinched, with a dark brown to purple banding that is generally wider than that associated with dollar spot. Sometimes spots with distinct borders

occur elsewhere on leaf blades. In other cases leaves may exhibit white banding or be entirely collapsed and shriveled. These foliar symptoms resemble heat or drought stress.

Ascochyta spp. produce minute yellow to dark brown, flask-shaped fruiting bodies (pycnidia) in diseased leaf tissue (Figure 4). These fruiting bodies, which are easy to view with a hand lens, are peppered throughout the dead leaves and can be useful as a diagnostic sign distinguishing Ascochyta leaf blight from dollar spot, abiotic causes and white grub damage. With white grub damage, the turf can be easily peeled back to observe the grubs.

Conidia (spores) produced within a pycnidium are hyaline (colorless) with one to two septa (Figure 5). Septate conidia are often slipper-shaped (Figure 5 inset).



Figure 1. Ascochyta leaf blight of Kentucky bluegrass.



Figure 2. Ascochyta leaf blight of Kentucky bluegrass.



Figure 3. Foliar symptoms of Ascochyta leaf blight.

Management

Ascochyta leaf blight damage can be managed by following good cultural practices that minimize stress to the turfgrass. Reduce thatch and promote water infiltration through the soil by yearly core cultivation. Maintain height of cut between 2½ and 3 inches. Minimize wounding of the leaf blades by maintaining sharp mower blades. Avoid mowing during wet weather, especially when *Ascochyta* spp. are active. Reduce mowing frequency and increase mowing height during *Ascochyta* leaf blight outbreaks. The fungus can be spread from one location to another on grass clippings, although this is unlikely to contribute

significantly to disease development. Similarly, collecting clippings while mowing to reduce the amount of inoculum is unlikely to reduce disease severity. Disease development depends more on environmental conditions than on the initial amount of fungus present in the turfgrass.

Maintain balanced fertility. Avoid excessive applications of nitrogen fertilizer, especially in the spring. Too much nitrogen promotes rapid, succulent leaf growth that requires more frequent mowing and causes more wounding of the turfgrass.

Try to maintain uniform soil moisture. Check the irrigation system to make sure that all irrigation heads are working properly

and that water is being distributed uniformly to avoid drought stress. On the other hand, excessive irrigation and poorly drained soils can also promote disease development.

Ascochyta leaf blight is primarily a leaf disease and not a root or crown disease, so it rarely causes plant mortality. Turfgrass usually recovers completely after a couple of weeks. Although several fungicides will inhibit *Ascochyta* spp., they can be expensive and difficult to apply. Furthermore *Ascochyta* leaf blight development is sporadic and rapid, making timing of preventive and curative fungicide applications difficult.

Conditions

Ascochyta spp. can be found on senescing or dead leaves of several turfgrass species; however, the disease appears to be most serious on Kentucky bluegrass.

Ascochyta spp. survive as conidia in pycnidia on dead leaves or clippings remaining in the thatch. The pycnidia are highly resistant to breakdown by drought or extreme temperatures. Thousands of conidia ooze from a single pycnidium during wet weather and are dispersed by splashing rain, irrigation, mowing or other management activities.

Conditions that favor *Ascochyta* blight development are poorly understood. The disease can occur in late spring or summer on drought-stressed turf resulting from water

restrictions or poor irrigation system coverage. However, the disease also develops during periods of hot weather preceded by unusually wet soil conditions induced by excessive rain or overirrigation. Frequent mowing and dull mower blades contribute to disease severity by creating more wounds that serve as infection sites.

Symptoms can be confused with those of

- dollar spot
- heat stress
- moisture stress
- mower injury
- white grub damage



Figure 4. Pycnidia of an *Ascochyta* sp. in senescing leaf tissue of Kentucky bluegrass.



Figure 5. Conidia of an *Ascochyta* sp. produced in pycnidia. *Inset*: Close-up of a slipper-shaped, septate conidium.

Brown patch

Pathogen: *Rhizoctonia solani*

Hosts: Tall fescue, bentgrass, Kentucky bluegrass, perennial ryegrass, fine fescue

Brown patch is a sheath- and leaf-blighting summer disease that is common on tall fescue and bentgrass. It is particularly severe on tall fescue. With increased use of tall fescue in urban and commercial landscapes, brown patch has become a significant management problem.

Brown patch is caused by the fungus *Rhizoctonia solani*, which is classified into subgroups on the basis of anastomosis groups (AG). Subgroup AG-1 is the most common on tall fescue. Other anastomosis groups are found on bentgrasses, zoysiagrass and perennial ryegrass. The efficacy of some fungicides may vary among anastomosis groups.

Symptoms and signs

Brown patch in tall fescue is first noticed as areas of the turf that turn purple to gray-green, resembling moisture stress. These areas quickly fade to light tan or brown as infected leaves dry out. The symptoms sometimes occur as discrete circular patches ranging from a few inches to several feet in diameter (Figure 1). More commonly, symptoms occur in large, diffuse areas (Figure 2).

Foliar symptoms in tall fescue are easy to see. Straw-colored foliar lesions with dark brown borders are apparent at the transition zone between healthy and diseased turf (Figure 3). Once the lesions have completely girdled the leaf sheath or leaf blade, the entire leaf quickly fades to tan or brown. Foliar symptoms are sometimes confused with those of dollar spot; however, dollar spot is rare on tall fescue.

Symptoms on bentgrass putting surfaces appear as copper-colored circles ranging in size from several inches to several feet in diameter (Figure 4). Foliar symptoms are not commonly observed on creeping bentgrass.

During weather that is favorable to brown patch, mycelium (mass of fungal vegetative growth) can sometimes be seen in the early morning. It is found at the transition zone between healthy and affected turf and can be seen growing over and between the leaf blades (Figure 5). When the disease is active, there is often a dark gray border at the margin of the patch where mycelium can be detected. Hyphae (individual fungal strands) have a predominantly right-angled branching pattern in contrast to that of the dollar spot fungus, which is predominantly Y-shaped (Figure 6).

Conditions

Rhizoctonia solani survives in the thatch and soil as bulbils (resting structures). During favorable weather, hyphae from bulbils begin active growth and infect grass leaves. Brown patch develops rapidly when night temperatures exceed 70 degrees F and duration of leaf wetness exceeds 10 hours. Brown patch can be severe following extended periods of hot (70–90 degrees F), rainy, humid weather.



Figure 1. Brown patch of tall fescue.

P. Sell



Figure 2. Brown patch of tall fescue.

N. Tisserat



Figure 3. Straw-colored lesions caused by *R. solani* on tall fescue leaf. Note brown border surrounding the irregularly shaped lesions.

Management

On tall fescue

Avoid seeding rates greater than 6 to 8 pounds per 1,000 square feet when establishing or renovating lawns. A higher population density makes it physically easier for *R. solani* to move from leaf to leaf and increases humidity and duration of leaf wetness periods.

Most tall fescue cultivars are susceptible to *R. solani*. Some newer cultivars have a higher level of resistance but can still be severely blighted under heavy disease pressure. Recovery, however, may occur more quickly than with older cultivars.

Brown patch occurs less frequently when available nitrogen is adequate to low and phosphorus and potassium levels are adequate. Apply the majority of the nitrogen fertilizer in the fall. Applications of more than 4 pounds of actual nitrogen per 1,000 square feet per year will predispose the turfgrass to increased brown patch activity. Don't apply nitrogen fertilizer when brown patch is active. A light fertilization (less than ¼ pound nitrogen per 1,000 square feet) after a brown patch outbreak may speed turfgrass recovery.

Maintain height of cut at 3 to 3½ inches. Returning clippings does not increase brown patch severity.

Some research results indicate that the use of organic composts (turkey litter, cow manure, brewery waste, etc.) reduces brown patch severity. Other studies resulted in no significant control of the disease with these types of organic amendments.

Do not irrigate turfgrass in late afternoon or evenings if possible. This increases the duration of leaf wetness and increases the likelihood of brown patch development. Irrigation after midnight to early morning is preferable. These are the hours the turf would normally be wet from dew, and irrigation at this time does not extend leaf wetness periods.

R. solani usually infects only the leaves and leaf sheaths, so there is a high potential for recovery with moderating environmental conditions. The turf often fully recovers from blighting in two to four weeks without the use of fungicide. But many homeowners are unwillingly to accept the unsightly appearance of their lawn during this time. Also, with extended periods of weather favorable to brown patch, the crown may also be killed.

The use of fungicide is almost always

needed on high-profile, high-visibility lawns, green surrounds and fairways. Refer to Table 5 for a list of fungicides labeled for control of brown patch. Some of the newer combination products (e.g., Armada or Headway) also fit well in this market. When used in combination with the cultural practices outlined above, these products can provide three to five weeks of disease suppression. Two to three applications may be adequate to provide season-long control. Begin preventive sprays just before the most likely time period for infection in your region.

Another option is to apply a fungicide as soon as symptoms appear. This curative program will suppress growth and development of *R. solani*, but the turf will not recover from brown patch until temperatures are less favorable for the fungus and more favorable for growth of the cool-season grass.

On creeping bentgrass

Brown patch on bentgrass putting greens is best managed with a preventive fungicide program. Many of the cultural practices outlined above for tall fescue can be adapted for use on higher cut creeping bentgrass on tees and fairways.

High levels of available nitrogen increase disease severity on both tall fescue and creeping bentgrass.

Symptoms can be confused with those of

- moisture stress



Figure 4. Brown patch on a creeping bentgrass green.



Figure 5. Mycelium of *Rhizoctonia solani* on and between the leaf blades of tall fescue.



Figure 6. Hyphae of *Rhizoctonia solani* with right-angled branching pattern.

Copper spot

Pathogen: *Gloeocercospora sorghi*

Hosts: Bentgrass, bermudagrass, zoysiagrass

Copper spot is a foliar disease of bentgrass, with severest outbreaks occurring on velvet bentgrass. It also occurs sporadically on creeping bentgrass greens and higher cut creeping bentgrass tees and fairways. *Gloeocercospora sorghi* causes a leaf spot of bermudagrass and zoysiagrass as well.

Symptoms and signs

Although *Gloeocercospora sorghi* initially causes a leaf spot on creeping bentgrass, this phase of the disease is rarely observed in the field, because infected leaves are rapidly blighted. Usually the first indication of disease is the appearance of pinkish orange to copper-colored patches with irregular margins (Figure 1). The patches range from 1 to 3 inches in diameter, but under favorable conditions for disease development, patches often coalesce. Symptoms can also occur in streaks, because the fungus is easily spread by water and equipment.

The color of the patches is more pronounced under moist conditions that favor production of sporodochia (fruiting bodies). The conidia (spores) are produced on the sporodochia in gelatinous masses that can be observed with a hand lens. Look for pink to salmon-colored gelatinous spore masses protruding from leaf surfaces (Figure 2). The long, needle-like conidia within a spore mass are multicelled and usually curved (Figure 3).

Symptoms are easily confused with those of anthracnose basal rot, brown patch, dollar spot, Pythium blight or red leaf spot. Dollar spot occurs under slightly cooler temperatures and the infection sites tend to be tan rather than pink. Temperatures favorable for optimum development of Pythium foliar blight and red leaf spot coincide with those of copper spot. Red leaf spot patches on a putting green are less defined than those of copper spot. Initial infection sites of Pythium foliar blight also have a similar color to those of copper spot, but Pythium foliar blight develops under higher temperatures and is rare on greens-height bentgrass. Copper spot infection sites are generally more discrete than those of anthracnose basal rot, which are more diffuse.



Figure 1. Copper spot on a creeping bentgrass green.

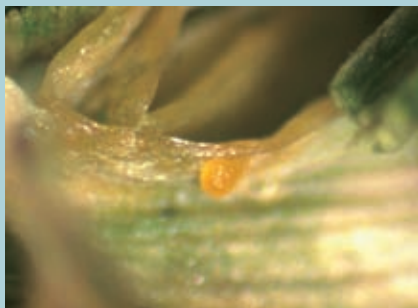


Figure 2. Sporodochium of *Gloeocercospora sorghi* on creeping bentgrass.

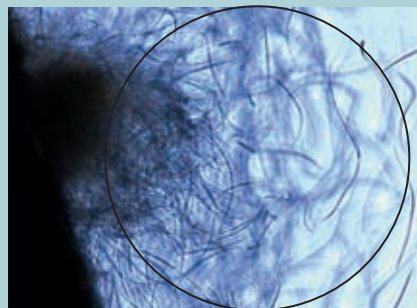


Figure 3. Conidia of *Gloeocercospora sorghi*.

Management

The common fungicide spray programs that are used in creeping bentgrass systems generally suppress copper spot. Refer to Table 5 for a list of fungicides labeled for control of copper spot. If a disease outbreak occurs, remove early morning dew and plant exudates (guttation water) with several turns of the irrigation head. Avoid mowing when the leaves are wet. Also avoid the use of water-soluble nitrogen when copper spot is active.

Conditions

The disease is reported to be more common and severe on velvet bentgrass than on creeping bentgrass. It is also more severe under acidic soil conditions. This may account for the sporadic nature of the disease, because the soil pH of many putting greens is high.

G. sorghi produces huge quantities of conidia in warm, moist weather (68 to 86 degrees F). Disease severity increases with prolonged periods of leaf wetness. Under these favorable conditions, the pathogen is readily spread if the turfgrass is mowed when the leaves are wet. The pathogen is also easily spread by splashing water (rainfall or irrigation) and through surface runoff. Spread also occurs in relation to the activities of people and animals.

Excess available nitrogen enhances disease development.

Symptoms can be confused with those of

- anthracnose basal rot
- brown patch
- dollar spot
- Pythium foliar blight
- red leaf spot

Dead spot

Pathogen: *Ophiosphaerella agrostis*

Hosts: Primarily bentgrass, hybrid bermudagrass

Dead spot is a disease of new sand-based bentgrass greens or renovated greens where methyl bromide was used in the renovation. The disease first occurs one to four years after construction or renovation, gradually disappearing one to three years after its first occurrence. Dead spot has also been reported in Texas and Florida on hybrid bermudagrass greens that were overseeded with rough bluegrass.

Symptoms and signs

The first symptoms of dead spot on bentgrass appear as reddish brown patches about ½ inch in diameter (Figure 1). The patches are most common on mounds, ridges, slopes and other historically dry areas on the putting surface. The diameter of the patches never gets much larger than 3 inches, and the patches do not coalesce. The patches are often sunken, interfering with putting quality. In the early stages of disease development, the small patches may be mistaken for ball marks or copper spot. As the disease progresses, the inner area of the patch turns light tan and at this stage can resemble dollar spot (Figure 2). When the fungus is active, the outer margins of the patch are red-brown to orange.

Disease generally starts in May, continuing throughout the summer until the first hard frost. Disease incidence varies among locations of greens, and the number of infection sites per green can range from a few to several hundred. New growth into the patch is inhibited, so recovery is slow.

All plant parts become infected, including leaves, leaf sheaths, roots and stolons. Unlike the dollar spot fungus, *Ophiosphaerella agrostis* generally does not produce visible mycelium (mass of fungal vegetative growth) in the field. One can sometimes observe a pink to white mycelium when plugs with symptoms are incubated in a moist chamber for several days.

Pseudothecia (fruiting bodies) develop in abundance in leaves, leaf sheaths and stolons (Figure 3). The pseudothecia are darkly pigmented, flask-shaped structures detectable with a hand lens (Figure 4). Ascospores are produced in pseudothecia in saclike structures (asci) (Figure 5). Mature ascospores are forcibly discharged or exude from the fruiting bodies under conditions of high relative humidity. Ascospores are easily spread by rainfall and irrigation water to initiate new infection sites.



J. L. Kaminski

Figure 1. Dead spot on a bentgrass green.



J. L. Kaminski

Figure 2. Close-up of dead spot on creeping bentgrass.



J. L. Kaminski

Figure 3. Black pseudothecia (fruiting bodies) of *Ophiosphaerella agrostis* in senescing leaf tissue.

Management

Dead spot has been reported on nearly all commonly used creeping bentgrass cultivars. Although there is some variability in cultivar susceptibility, dead spot can probably be expected to occur under any new construction, irrespective of the cultivar.

When only a few patches are observed, remove the infection center by plugging out and disposing of the plugs.

When disease incidence is high, the use of fungicides at weekly intervals may be necessary to protect putting quality. Refer to Table 5 for a list of fungicides labeled for control of dead spot. Fungicide application will suppress the activity of *O. agrostis*, but generally will not speed recovery of infection sites. Tank mixing a low rate of nitrogen with the fungicide has been shown to promote recovery.

There is some evidence that dead spot severity is lower when ammonium sulfate is used as the primary nitrogen source, whereas nitrate forms of nitrogen may result in more severe disease. This is probably related to changes in pH at the rhizosphere level. Ammonium sulfate generally lowers rhizosphere pH in contrast to nitrate forms, which generally result in higher pH at the rhizosphere.

Conditions

Dead spot most commonly occurs on new greens or greens treated with methyl bromide during a renovation project. The disease has not been observed on fairways with underlying native soil. The disease gradually disappears, presumably in response to the buildup of competitive microorganisms in the new or fumigated root zone.

Dead spot is a high-temperature (77 to 86 degrees F), low-moisture disease in which symptoms most commonly occur on areas of the green most prone to drying.

Symptoms can be confused with those of

- copper spot
- dollar spot
- ball marks
- cutworms



J. L. Kaminski

Figure 4. Pseudothecia (fruiting bodies) of *Ophiosphaerella agrostis*.



J. L. Kaminski

Figure 5. Multicelled, needle-like ascospores of *Ophiosphaerella agrostis* within an ascus.

Dollar spot

Pathogen: *Sclerotinia homoeocarpa*

Hosts: creeping bentgrass, Kentucky bluegrass, perennial ryegrass, zoysiagrass, bermudagrass, buffalograss

Dollar spot is a common disease on creeping bentgrass, Kentucky bluegrass and perennial ryegrass, but it is rare on tall fescue. It also occurs on zoysiagrass, bermudagrass, buffalograss and annual bluegrass. The disease can occur irrespective of management program or soil fertility status, although damage is usually most severe when the turfgrass is nitrogen-deficient.

Symptoms and signs

In higher cut turfgrass, *Sclerotinia homoeocarpa* infection results in the formation of small, roughly circular tan patches. Patch size varies with mowing height and environmental conditions and can range from a few inches to 6 inches or more in diameter (Figure 1). During extended periods of disease-favorable weather, patches coalesce into large, irregular, dead areas. Infected plants within the diseased spots wilt and eventually turn tan or brown because of girdling lesions on the leaf blades. This can result in substantial damage to the turfgrass.

On putting greens, dollar spot infection sites rarely exceed 2 inches in diameter (Figure 2). During weather that is favorable to dollar spot, hundreds of spots can coalesce to blight large areas of the putting surface. If left untreated, the spots become sunken, leaving a low-quality, pockmarked putting surface.

Foliar symptoms of dollar spot can be seen on plants at the transition zone between healthy and infected turfgrass. On Kentucky bluegrass, the lesions appear as bleached areas with a con-

stricted or hourglass appearance, usually with reddish brown borders (Figure 3). The lesions are often located near the middle of the leaf and extend completely across the leaf blade. On creeping bentgrass, individual infected leaves exhibit light tan lesions that may or may not be surrounded by a dark, reddish-brown border (Figure 4). The lesions are difficult to see on closely mowed bentgrass without a hand lens. On warm-season grasses (bermudagrass, buffalograss and zoysiagrass) the symptoms occur as discrete lesions bordered by brown bands (Figure 5).

In the early morning when dew is still present on the turfgrass, small cottony webs of fungal mycelium can sometimes be seen growing from the diseased leaf blades (Figure 6). This growth can be confused with spider webs, “cotton” from plant seeds, or other fungi, and therefore is not a reliable diagnostic feature.

When viewed under a compound microscope, the fungal hyphae have a characteristic Y-shaped branching, in contrast to hyphae of *Rhizoctonia solani*, which are predominantly right-angled (Figure 6 inset).



Figure 1. Dollar spot on Kentucky bluegrass in a golf course rough.



Figure 2. Dollar spot on creeping bentgrass.



Figure 3. Foliar symptoms of dollar spot on Kentucky bluegrass.

Management

On higher cut turfgrass

The first step to reduce the negative impact of dollar spot on any cool-season turfgrass is to apply enough nitrogen at the right time to meet the needs of the turfgrass species under management.

Dollar spot is rarely serious on home lawns, but irrigation practices that extend the duration of leaf wetness can promote disease development. Irrigation of any turf, whether home lawn, fairway or green is best applied from midnight to early morning when the grass is normally wet.

A preventive fungicide application is generally not needed on home lawns. Nevertheless, dollar spot can reduce the quality of the turf for short periods. On higher cut, cool-season turfgrass on golf courses, the need for fungicides to suppress dollar spot depends on the susceptibility of the turfgrass species being managed and on the facility's expectations and budget.

On bentgrass or bentgrass/annual bluegrass greens

The first line of defense is to use

a bentgrass cultivar with resistance to dollar spot. Check the National Turfgrass Evaluation Program Web site at <http://nstep.org> for the most up-to-date information.

Minimize the duration of leaf wetness by physically removing dew (poling, mowing, syringing) in early morning. Do not allow the grass to go under drought stress. Maintain adequate nitrogen fertility. Biological control of dollar spot by fertilization with composted turkey litter, bovine wastes and other organic amendments has been achieved by some superintendents. However, organic amendments have not consistently reduced dollar spot incidence and severity in university research trials. Similarly, results with the addition of microbial biological control agents (fungi and bacteria) and plant defense activators have not been consistent.

Preventive fungicide applications at 7- to 21-day intervals are needed to suppress dollar spot. The timing and frequency of application depends on weather conditions and on the type of fungicide that is applied. Continuous use of xylem mobile fungicides, including thiophanate methyl and the DMI fungicides (fenarimol, myclobutanil,

propiconazole and triadimefon) may result in the selection and increase of fungicide-resistant strains of *S. homoeocarpa*. Superintendents should consider limiting the number of applications of these fungicides during the growing season and tank mixing or alternating these products with contact fungicides not prone to resistance problems.

Many superintendents have reported that dollar spot on bentgrass or bentgrass/annual bluegrass greens is becoming more difficult to control, raising concerns about fungicide-resistant strains of *S. homoeocarpa*. Careful study has shown that the lack of control is often related to fungicide application errors. One such error is application of lower fungicide rates than recommended on the product label, either intentionally to save money or accidentally because of sprayer calibration issues. Another error is the attempt to extend the interval of protection beyond label recommendations. Although fungicides can suppress *S. homoeocarpa*, bentgrass recovery may not occur quickly. This is especially true when the bentgrass is under growth regulation and generation of new tissue is delayed.

Conditions

Dollar spot is more prevalent in nitrogen-deficient turfgrass. *S. homoeocarpa* survives indefinitely in thatch and soil. In the presence of a thin film of moisture on leaves and moderate temperatures (65 to 85 degrees F), the fungus infects leaves. *S. homoeocarpa* rarely

infects turfgrass roots, although fungal toxins may adversely affect root formation. Dollar spot is most severe in late spring and early summer and again in early fall; however, it can occur throughout the summer months. The patches are more numerous in areas where air circulation or soil drainage is poor.

Symptoms can be confused with those of

- Ascochyta leaf blight
- dead spot
- brown patch
- cutworms



Figure 4. Foliar symptoms of dollar spot on creeping bentgrass.



Figure 5. Foliar symptoms of dollar spot on bermudagrass.

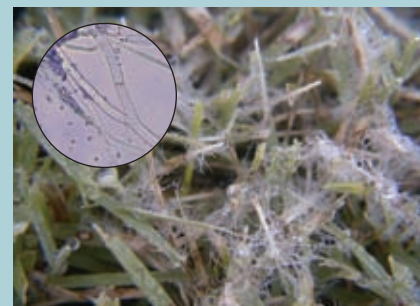


Figure 6. Mycelium of *Sclerotinia homoeocarpa*. Inset: A hyphal strand of *S. homoeocarpa* with Y-shaped branching.

N. Tisserat; Inset: B. Corwin

Drechslera leaf spots

Pathogen: *Drechslera* spp.

Hosts: *D. poae* - Kentucky bluegrass, annual bluegrass, tall fescue and fine-leaved fescues; *D. erythrospila* - creeping bentgrass, colonial bentgrass

Drechslera diseases are part of a group of diseases caused by fungi that were once cataloged in the genus *Helminthosporium*. Over the years, taxonomists have revised the genus *Helminthosporium* to include the genera *Drechslera*, *Marielliotta*, *Bipolaris* and *Exserohilum*. This section covers two diseases: (1) Drechslera leaf spot and melting-out caused by *D. poae* and (2) red leaf spot caused by *D. erythrospila*.

Symptoms and signs

Drechslera leaf spot and melting-out

This disease was once considered the most important spring and fall disease of Kentucky bluegrass, but the widespread use of resistant bluegrass cultivars has lessened the impact of this disease. Still, some leaf spot can be found on most home lawns in the spring, and it can damage newly seeded lawns and certain susceptible cultivars of Kentucky bluegrass. This disease can sometimes be severe on tall fescue.

Drechslera leaf spot and melting-out develops in late March through mid to late May, reoccurring in the fall. Infected plants initially develop small elliptical purple leaf spots (Figure 1). The spots turn light gray or tan but remain bordered by a dark brown to purple margin (Figure 2). Leaf tissue surrounding the spots may turn yellow. The leaf spot phase of the disease usually does not damage the plant significantly. However, the fungus can also infect the crown, rhizomes and roots. As daytime temperatures increase, leaves on crown-infected plants turn light green or yellow, similar to nitrogen-

deficient turf. Eventually these plants die and turn brown or straw colored. This is referred to as the melting-out phase of the disease. Severe melting-out results in irregular patches of dead turf. Damaged lawns are thinned and subject to weed invasion. Symptoms on tall fescue are similar to those on bluegrass.

Drechslera poae, as well as other fungi in this general group, sporulates to produce multicelled, darkly pigmented conidia (spores). As illustrated with a similar fungus, *Bipolaris sorokiniana*, sporulation occurs on the surface of infected grass tissue (Figure 3). The conidia are easily spread by wind, water and mowing, and the dark pigmentation makes them resistant to breakdown by ultraviolet light.

Red leaf spot

Red leaf spot is more common on colonial bentgrass than on creeping bentgrass. In the last several years, however, the disease has occurred with increasing frequency on several newer creeping bentgrass cultivars.

Red leaf spot is a high-temperature disease, occurring in June through August. The



Figure 1. Drechslera leaf spot on Kentucky bluegrass.

N. Tisserat



Figure 2. Drechslera leaf spot on tall fescue.

B. Bockus



Figure 3. Sporulation of *Bipolaris sorokiniana* on Kentucky bluegrass.

N. Tisserat



Figure 4. Red leaf spot on creeping bentgrass.

Management

Drechslera leaf spot and melting-out

The most effective means of controlling leaf spot and melting-out is to plant resistant cultivars. Most of the newer Kentucky bluegrass cultivars have a moderate to high level of resistance to *D. poae*. When seeding, use a blend of three or more of these cultivars. Avoid planting cheaper, “common” types of bluegrass such as ‘Park’, ‘South Dakota’, and ‘Kenblue’.

Avoid excessive nitrogen fertilization in spring that favors lush growth, but do not “starve” the turfgrass of nitrogen during the spring. A well-balanced fertilization program

will reduce the severity of the disease. There is some evidence that frequent watering to keep the thatch moist will help reduce sporulation by the fungus. The best time to irrigate is between midnight and early morning, because this will not extend the duration of leaf wetness. Use higher mowing heights (less than 2 to 2.5 inches) in late spring or during dry weather. Thatch reduction will also help reduce disease severity.

If leaf spot has occurred in previous years but has not caused significant injury, then fungicides are not warranted. Nevertheless, chemical protection may be required on highly susceptible cultivars.

Refer to Table 5 for a list of fungicides labeled for control of leaf spot and melting-out. In this situation, the fungicide should be applied as soon as symptoms are observed. The disease is very difficult to control once the melting-out and crown rot phase has begun.

Red leaf spot

When we have observed red leaf spot on creeping bentgrass, it has usually been confined to only a few greens. Fungicide application may be necessary to suppress red leaf spot. If the disease occurs on only a few greens, mow these greens last to avoid spreading the pathogen in the clippings.

first noticeable symptoms on the putting surface are small brown to red patches, ranging in size from 1 to 8 inches or more (Figure 4). Under favorable conditions for disease development, the infection centers can coalesce to blight large areas of the putting surface (Figure 5). Unlike other leaf spot diseases on the larger-bladed turfgrasses, a true leaf spot symptom is difficult to see on creeping bentgrass. By the time the red patches are observed on the putting surface, individual leaf blades have usually shriveled (Figure 6). Conidia (spores) can often be observed with the aid of a hand lens (Figure 7). Morphologically the conidia are similar to those produced by other genera of fungi in this group (Figure 7, inset).

Conditions

Drechslera leaf spot and melting-out

Leaf spot fungi overwinter in the thatch layer or in small lesions on leaf blades. *Drechslera* leaf spot and melting-out is favored by long periods of leaf wetness and moderate temperatures. *D. poae* is inhibited by hot weather and rarely appears during the summer or early fall.

Red leaf spot

Red leaf spot is favored by hot, dry conditions. Some bentgrass cultivars appear to be more susceptible than others.

Leaf spot and melting-out can be confused with

- white grub damage

Red leaf spot symptoms can be confused with those of

- anthracnose basal rot
- copper spot



Figure 5. Red leaf spot on creeping bentgrass.

D. Minner



Figure 6. Foliar symptoms of red leaf spot on creeping bentgrass.

D. Minner



Figure 7. Sporulation of *Drechslera erythrospila* on creeping bentgrass. Inset: Conidia of *D. erythrospila*.

Fairy ring

Pathogen: Numerous fungi in the Basidiomycetes

Hosts: All turfgrass sites

Fairy ring is caused by more than 60 basidiomycete fungi, including those producing the familiar puffballs and toadstools. The rings result from the activities of these fungi growing radially through the thatch or soil, rather than from a direct parasitic relationship with the turfgrass. Fairy ring occurs wherever grass is grown.

Type 1 rings are those that result in loss of turf at the outer margin of the ring. **Type 2** rings have outer margins that are darker green in color than the surrounding turf. Fruiting bodies may or may not be found in association with Type 1 and 2 rings. **Type 3** rings exhibit a ring of fruiting bodies with no effect on the turf.

Symptoms and signs

Fairy rings are usually first noticed as irregular rings or arcs of darker green grass on the turf surface (Figure 1). The darker color is caused by the release of nitrogen as the fairy ring fungus breaks down organic matter. The margins of the rings are often irregular or wavy. On putting surfaces especially, the entire area defined by the ring may be darker green than the surrounding uncolonized area (Figure 2).

The rings can remain green or progress to the point where dead grass develops at the margin (Figure 3). Death of grass at the margin has been attributed to moisture stress due to hydrophobic soil conditions that develop as the fairy ring fungus grows. The death has also been linked to excess nitrogen release or the formation of hydrogen cyanide or other toxic metabolites. With some fairy rings, death of the grass also occurs in spots where the fruiting bodies have emerged and then collapsed (Figure 4). Fairy ring with resulting loss of turf is one of the most destructive diseases occurring on putting surfaces.

Where two or more rings meet, the margins of the individual rings are incomplete (Figure 4). Rings are also disrupted by changes in soil type (Figure 5) or by impediments such as cart paths or sidewalks. When rings form on a hillside, the bottoms of the rings are not closed.

Fruiting bodies associated with the causal fungus may or may not emerge. Fruiting bodies found on putting surfaces are often puffballs that form at the outer margins of the rings (Figure 6). Other fairy ring fungi, especially those in pastures or other areas of higher cut turf, produce larger and more spectacular mushrooms (Figure 7).

Conditions

Fairy rings are elusive, often visible one year and not the next. Under some environments, fairy rings are visible every year. The factors that favor symptom expression and the growth and development of the fairy ring fungi are not well understood. Until recently, identification of the fungal species causing fairy ring in a given environment has keyed on fruiting body morphology and spore color, size and shape. Recent



Figure 1. Early symptom of fairy ring expressed as an outer green ring (Type 2).



Figure 2. The entire area colonized by this fairy ring fungus is greener than the uncolonized turf. Note incomplete margins where two fairy rings meet.



Figure 3. Firing of the turfgrass at the margin of the ring (Type 1).

Management

Management of fairy ring on putting surfaces has been achieved with varying levels of success by application of one or more of the fungicides labeled for fairy ring control or suppression. Fungicides labeled against fairy ring include Heritage, Insignia and Prostar. Also included in this list are Bayleton and Endorse under a FIFRA Section 2(ee) registration. The level of fairy ring control or suppression with fungicide has varied widely from one location to the next. There are a number of factors that most likely contribute to inconsistent results. The application method is important. To increase the chances for success, the area to be treated should be spiked or quadranted before fungicide application. The use of a wetting agent just before fungicide application or as a tank mix with

the fungicide may increase efficacy. Follow individual label recommendations.

Another factor that no doubt contributes to inconsistent results is that in most cases the targeted fairy ring species is unknown, so there is no effective way to determine how deep the fungus is growing. Also, fungicides labeled for fairy ring management differ in the fairy ring species listed on the labels. Until the biology of these fungi is better understood, finding the right product and application method for your facility may have to be achieved by trial and error.

On greens with a history of fairy ring that expresses as Type 1, fungicide application in combination with cultural practices can be a useful tool. Preventing firing of the fairy rings and subsequent loss of turf can often be achieved by diligent monitoring of moisture status in these areas, combined with wetting agent application,

spiking and hand watering as needed to prevent drying. Removal of excess thatch is also necessary.

Symptom expression can often be masked with application of judicious amounts of either nitrogen or iron, or both. The level of success with this approach will depend on the nutrient status of the turf.

Fairy rings on home lawns seldom cause extensive damage; however, homeowners often consider the mushrooms to be unsightly or express concerns about ingestion by children or pets. Removal of mushrooms by hand or by mowing is recommended where pets or children may be exposed, because many of the fairy ring fungi as well as other mushrooms in lawns are toxic. Masking symptoms with small amounts of nitrogen or iron may be effective in some cases.

advances in genetic techniques have allowed for identification of some species without the presence of fruiting bodies, but this is not a widely available diagnostic tool.

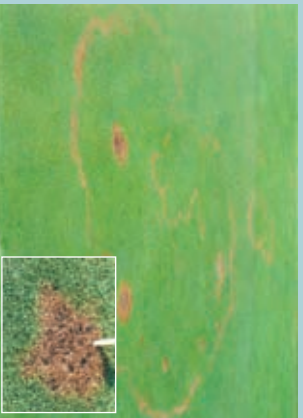
Although the biology of the fairy ring fungi is not well understood, certain generalizations can be made. Fairy ring symptoms are usually more noticeable in nitrogen-deficient turf, simply because more nitrogen is available to the turfgrass plant in areas where the fairy ring fungus is decomposing organic matter.

Fairy ring is more commonly observed under heavy thatch. Fairy ring has been observed on all soil types, but is more common on sand-based putting surfaces. Fairy ring is especially abun-

dant following new construction or renovation,

presumably because of low population levels or lower diversity of competitive or antagonistic microorganisms. Nevertheless, fairy ring can also occur on older putting surfaces, especially under heavy thatch or where severe layering is an issue. Greens toppedressed with fine sand are also more prone to fairy ring.

Moisture is important to fairy ring development and symptom expression. Type 2 rings are more likely to progress to damaging Type 1 rings when moisture is limiting. Ring margins typically fire on windy days with low relative humidity. Once browning has occurred, the soil is difficult to rehydrate. Fruiting bodies are more common under wet, humid conditions.



E. Ervin, Inset: B. Corwin

Figure 4. Loss of turfgrass at margin and around collapsed fruiting bodies. Inset: Turfgrass damage surrounding collapsed fruiting bodies.



E. Ervin

Figure 5. Disruption in continuity of the ring where the root zone changes from sand to soil.

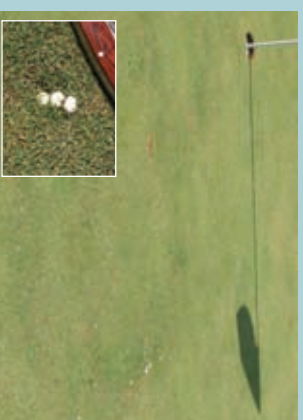


Figure 6. Fruiting bodies (puffballs) emerging at the margin of fairy rings on a putting green. Inset: Close-up of newly emerged puffballs.

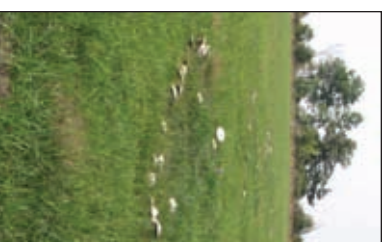


Figure 7. Large, showy mushrooms of a fairy ring in a pasture.

Gray leaf spot

Pathogen: *Pyricularia grisea*

Hosts: Perennial ryegrass, tall fescue

Gray leaf spot, also called blast, was first observed on perennial ryegrass throughout the eastern half of the United States in 1992. The epidemic resulted in extensive damage to golf course fairways and athletic fields. The disease now occurs in most areas of the country where perennial ryegrass is grown. Disease development is sporadic with little or no disease development in some years. Nevertheless, the potential destructiveness of gray leaf spot forces many turfgrass managers to apply preventive fungicide applications every year.

Symptoms and signs

Gray leaf spot often develops first in heat or drought prone areas such as the tops of small mounds or on steep slopes. The diseased turf first appears droughty even though soil moisture is sufficient but soon collapses and turns a dull brown (Figure 1). The fungus may quickly spread from these diseased patches and cause extensive blighting if weather conditions are favorable for infection (Figure 2). In some years the disease never progresses beyond blighting in these “hot spots.” Other turfgrass species that may be mixed in with the ryegrass, including Kentucky bluegrass, annual bluegrass and creeping bentgrass, are not affected by the disease. Blighting can continue through late summer and into early fall and cause significant damage to young ryegrass seedlings emerging from fall overseeding.

Small, chocolate-brown spots about $\frac{1}{16}$ inch in diameter develop on leaves (Figure 3). During early disease development, these leaf spots may not be numerous and can be easily

overlooked. Depending on weather, multiple coalescing leaf spots will girdle and kill both leaf and crown tissue. Dying leaves tend to develop a characteristic twist at the leaf tip, which helps differentiate this disease from brown patch or *Pythium* foliar blight (Figure 4).

Gray leaf spot also occurs infrequently on tall fescue. Symptoms are similar to those on perennial ryegrass, but disease progression is slower and damage is much less severe. Leaves initially develop small, dark brown spots that eventually expand and develop a gray center (Figure 5). The spots rarely coalesce to kill leaf blades or plants.

Pyricularia grisea sporulates prolifically in the center of lesions during periods of high relative humidity (Figure 6). The conidia (spores) are spread by wind, splashing water and equipment to infect new leaves. New foliar symptoms appear within a few days of infection.



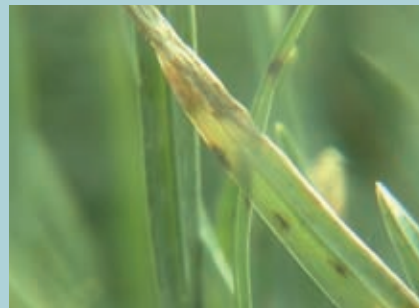
D. Settle

Figure 1. Gray leaf spot of perennial ryegrass.



N. Tisserat

Figure 2. Gray leaf spot of perennial ryegrass.



N. Tisserat

Figure 3. Foliar symptoms of gray leaf spot on perennial ryegrass.

Management

Avoid excessive nitrogen fertilization during mid to late summer. Avoid quick-release forms of nitrogen that result in flushes of growth, beginning just before conditions favorable for gray leaf spot are anticipated.

Time irrigations to minimize leaf wetness periods but avoid letting the turfgrass go under drought stress.

On turf facilities with a history of

gray leaf spot, cultural practices alone are unlikely to control gray leaf spot. Fungicide applications beginning in early to mid-August and continuing through September at regular intervals may be required to prevent damage on golf course fairways and athletic fields. Although thiophanate methyl and the QoI fungicides provide the best control of gray leaf spot, their use should be limited to one or possibly two applications per season to prevent the selection of fungicide-resistant strains. Instead, rotate with

other fungicides listed in Table 5.

Breeding programs to develop perennial ryegrass cultivars with resistance to *P. grisea* are under way. Go to <http://ntep.org> for the most recent information on relative susceptibility of perennial ryegrass cultivars.

On sites where gray leaf spot is a chronic problem, it may be advantageous to replace perennial ryegrass with Kentucky bluegrass or creeping bentgrass.

Conditions

P. grisea probably survives the winter locally in leaf debris or perhaps in living perennial ryegrass plants, although influx of conidia from distant sources occurs in some years. *P. grisea* infects perennial ryegrass plants at non-detectable levels in early summer, then builds in intensity later in the growing season when conditions are more favorable for disease development. Prolific sporulation and relatively short disease cycle are reasons why gray leaf spot progresses rapidly and destructively.

Factors that trigger epidemics include high temperatures or extended periods of leaf wetness interspersed with intermittent dry periods. Symptoms develop under hot (82 to 90 degrees F), humid conditions. At these temperatures, infection occurs with a leaf wetness period as brief as 9 hours. The lower the temperature, the longer leaves must remain wet for infection to occur.

Seedlings are more susceptible than older plants to *P. grisea*, especially under high levels of available nitrogen. Gray leaf spot is often more severe in roughs than on fairways. This is probably related more to microclimate differences in duration of leaf wetness within the turfgrass canopy rather than to height of cut.

Symptoms can be confused with those of

- brown patch
- Pythium foliar blight
- moisture stress

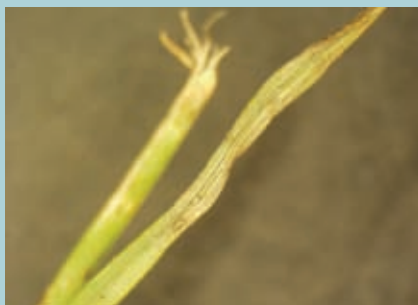


Figure 4. Foliar symptom of gray leaf spot on perennial ryegrass.



Figure 5. Gray leaf spot on tall fescue.



Figure 6. Conidia of *Pyricularia grisea*.

Gray snow mold (speckled snow mold)

Pathogen: *Typhula* spp.

Hosts: Most cool-season turfgrasses

Gray snow mold is a winter turfgrass disease in regions where snow cover persists for long periods. All grasses can be damaged to some extent, but injury is often more severe on annual bluegrass and bentgrass putting greens. Gray snow mold is uncommon in the lower Midwest.

Symptoms and signs

Gray snow mold begins to develop 30 to 45 days after permanent snow cover, but symptoms will not be apparent until after the snow has melted or has been removed.

Yellow to white circular patches of damaged turf are visible immediately following snow melt (Figure 1). These patches vary in size and may overlap to blight almost the entire turf stand (Figure 2). White to gray fungal mycelium may still be evident on diseased leaves, especially on higher cut turf, but will eventually dry out and disappear (Figure 3). As the damaged leaves begin to wither, the patches develop more of a white to gray color (Figure 4).

The observation of clamp connections at high magnification and sclerotia embedded in infected leaf tissue are diagnostic for gray snow mold. Clamp connections are “bridges” from one hyphal cell to another (Figure 5). The sclerotia are initially white to pink or orange, but harden and turn dark brown to black with age

(Figure 6). The mature sclerotia range in size up to 0.2 inch in diameter.

Damage in home lawns and golf course fairways is often cosmetic. The turfgrass quickly recovers and resumes normal growth with the return of warm spring weather. However, damage to annual bluegrass and creeping bentgrass on putting greens and other closely mowed areas may be more extensive and spring recovery slower.



Figure 1. Gray snow mold of creeping bentgrass.



Figure 2. Gray snow mold of bluegrass.



Figure 3. Gray snow mold of Kentucky bluegrass.

Management

The most consistent means of controlling gray snow mold is the use of preventive fungicide applications. Refer to Table 5 for a list of fungicides labeled for control of gray snow mold. Fungicides are rarely needed on home lawns because damage is normally superficial. In regions with a history of gray snow mold associated with extended periods of snow cover, preventive fungicide applications are required to maintain high turfgrass quality on golf courses and athletic fields.

Make a fungicide application in fall as close to permanent snow cover as possible. This helps prevent premature losses of the fungicide and maximizes chemical concentrations during the winter. Some turf managers also apply a gray snow mold preventive in late fall to help suppress

germination of sclerotia and dispersal of spores.

The fungicide quintozene (PCNB) has been the standard snow mold fungicide for many years because of its persistence, efficacy and relatively low cost. However, PCNB may cause phytotoxicity to annual bluegrass and creeping bentgrass, especially on putting greens. Furthermore PCNB is losing its federal registration for use on turfgrasses.

Several other fungicides are used for management of gray snow mold. Generally a mixture of two or more fungicides with different modes of action is used for snow mold suppression. This increases efficacy against a range of snow mold pathogens.

Biological control agents, including isolates of *T. phacorrhiza*, have shown efficacy against gray snow mold; however, these have not yet been successfully

developed into commercial products.

Excessive nitrogen fertilizer applications in the fall may increase gray snow mold severity. Use slow-release formulations of nitrogen if fall applications are needed. Continue mowing until winter dormancy to prevent excessive leaf growth and matting that may enhance fungal colonization.

There is a misconception that snow compaction caused by snowmobiles and skiers increases snow mold severity. In fact, compacting snow lowers temperatures at the soil/snow interface, often to temperatures below freezing, and inhibits snow mold development. Nevertheless there may be other problems associated with snow compaction, including slower spring green-up and freeze damage to subsurface irrigation lines.

Conditions

Gray snow mold fungi are widely distributed in regions with long periods of snow cover. There are several species of *Typhula*, and their abundance in a particular location is governed in part by the length of snow cover. *T. ishikariensis* tends to be more prevalent in areas with long periods of snow cover, whereas *T. incarnata* is more common where snow cover periods are shorter.

These fungi persist through the summer as hardened sclerotia. In late fall, sclerotia germinate and begin to colonize the grass leaves.

In some cases sclerotia may germinate to form fruiting structures (basidiocarps) that in turn produce spores (basidiospores) that help disseminate the fungus and initiate disease.

The growth of *Typhula* spp. is favored by temperatures near freezing (32 to 36 degrees F). Thus, long periods of snow cover that help maintain temperatures at the soil surface just above freezing are ideal for fungal growth. The longer snow cover remains, the more extensive are the fungal colonization of the turfgrass and subsequent damage.



Figure 4. Ash gray appearance of older gray snow mold patches.

T. Blunt



Figure 5. Clamp connections on hyphae of a *Typhula* sp.

N. Tisserat



Figure 6. Mature sclerotia of *Typhula* sp. in infected bluegrass leaves.

N. Tisserat

Large patch

Pathogen: *Rhizoctonia solani*

Hosts: Primarily zoysiagrass, but also buffalograss and bermudagrass

Large patch is a cool-season disease of warm-season grasses. Symptoms are most common when these grasses are either entering or coming out of dormancy. Large patch is a sheath blight disease caused by a *Rhizoctonia solani* that is in a different anastomosing group (AG 2-2 LP) than the *R. solani* causing brown patch in cool-season grasses.

Symptoms and signs

Circular, slightly matted areas of straw-yellow zoysiagrass initially develop in late September to early October during cool, rainy weather (Figure 1). During favorable weather, the fungus progressively blights more shoots, resulting in large, blighted patches of turfgrass with bright orange margins (Figure 2). Examination of individual shoots within the patch reveals reddish brown to black lesions at the base of the leaf sheaths, beyond which the leaf blades exhibit a yellow-orange color (Figure 3). The yellow-orange color is due to an accumulation of anthocyanins caused by girdling of the leaf sheath.

The yellow-orange margin is indicative of active disease, and the large, cinnamon-brown colored hyphae of *Rhizoctonia solani* are most easily observed in the leaf sheaths during this phase (Figure 4). Lesions normally do not develop on the leaves. Depending on disease severity, unaffected, living shoots may be scattered throughout the patch. Patches of blighted turf often exceed 20 feet in diameter.

Sheath infection can continue into November as long as soil temperatures remain above 50 degrees F and soil moisture is high. Patch enlargement after fall dormancy (October) may not be noticed until zoysiagrass resumes spring growth.

Patches of zoysiagrass as well as buffalograss damaged by *R. solani* reappear in spring as light brown, sunken areas that are slower to recover from dormancy than surrounding, healthy turfgrass (Figure 5). Leaf sheath lesions typically are not present in early spring, but sheath rotting may resume in April and May as soil and air temperatures increase. This can result in patch expansion and symptoms similar to those described for fall infection. New patches also may develop in spring. The activity of *R. solani* usually continues through May but is suppressed by high summer temperatures.

The fungus does not kill stolons and roots within the patch, and new shoots will form in the damaged areas with time. Because infected zoysiagrass is slow to recover, weed invasion in the thinned areas of the patch is common (Figure 6).



Figure 1. Early symptoms of large patch of zoysiagrass in the fall.



Figure 2. Symptoms of active large patch on a zoysiagrass fairway.



Figure 3. Lesions resulting from *Rhizoctonia solani* infection and yellow-orange discoloration of leaves beyond this girdled area.

Management

Avoid overwatering the turfgrass, especially in the fall or early spring. Poorly drained areas are especially susceptible to injury from large patch and should be constructed (e.g., draining tiles) to avoid soil saturation. Avoid mowing the turfgrass in early morning when the thatch is spongy or wet. The fungus may be distributed in grass clippings during mowing.

Core cultivation or verti-slicing in June or July helps reduce thatch accumulation and invigorates the turfgrass. A reduction in the thatch layer may also suppress large patch development. Do not cultivate in early spring or at other times when the disease is active. The fungus can be spread on infected turf cores removed during cultivation, and physical damage associated with this cultural practice can weaken the turfgrass and slow the recovery process.

Early spring fertilization increases the possibility of a large patch epidemic. Begin fertilization only after large patch activity has stopped. This is usually sometime in mid- to late May. Research results indicate that routine applications of slow-release forms of nitrogen in the summer (urea formaldehyde, turkey compost litter) may slightly suppress severity of large patch in the fall. Avoid using more than 2 lb/1,000 sq. ft. of actual nitrogen during the growing season. If large patch is severe in the spring, application of a quick-release form of nitrogen, such as urea, in late May or early June may help speed turfgrass recovery during the early summer months. Avoid using quick-release nitrogen forms in late summer.

Several fungicides are effective at suppressing large patch development, but the degree of success depends on timing of the application. Refer to Table 5 for a list of fungicides labeled for management of

large patch. Preventive applications should be made in mid- to late September before large patch symptoms develop. As a general guideline, a single application should be made to areas with a previous history of the disease when thatch temperatures reach 70 to 75 degrees F. A fall fungicide application not only inhibits fall infection, but may also help suppress or delay spring symptoms. A spring (April) preventive fungicide application may be necessary in locations with a history of severe large patch.

Curative fungicide applications are less effective in controlling large patch. Many zoysiagrass shoots are already damaged or killed by the time a curative treatment is applied. Because of the cool temperatures in spring and fall, turfgrass cannot quickly recover from the injury. Nevertheless, curative fungicide treatments may prevent expansion of existing patches.

Conditions

Large patch symptoms can occur anytime during the growing season, but they are most common in spring and fall as zoysiagrass enters or breaks winter dormancy. The disease occurs on both residential lawns and golf courses, but it is more severe when the grass is mowed to heights of less than one inch (e.g., golf course fairways) and where soil drainage is poor.

Large patch development is favored by heavy thatch. *R. solani* overwinters in the thatch as bulbils (resting structures). It does not

produce spores, but can easily be moved from one location to another on contaminated sod.

The *R. solani* that infects zoysiagrass belongs to a different subgroup (AG 2-2 LP) and has a lower temperature range (50 to 86 degrees F) for infection than the brown patch fungus that damages cool-season turfgrasses. Therefore, large patch almost always occurs during relatively cool weather in spring and fall, whereas brown patch is a problem on cool-season turfgrasses in midsummer. Large patch development is favored by frequent rains and excessive soil moisture.

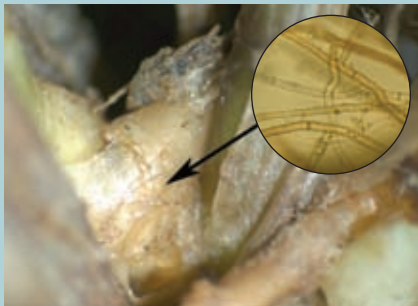


Figure 4. Hyphae of *Rhizoctonia solani* in leaf sheaths of zoysiagrass. Inset: Close-up of hyphae of a *Rhizoctonia* sp. with characteristic right-angled branching.

Inset: J. Thompson



Figure 5. Symptoms of large patch on buffalograss in the spring.



Figure 6. Weed invasion in an area of zoysiagrass recovering from large patch.

N. Tisserat

Leaf and sheath spot

Pathogen: *Rhizoctonia zae*

Hosts: Primarily Kentucky bluegrass, annual bluegrass and creeping bentgrass, but also perennial ryegrass and tall fescue

Rhizoctonia zae, which forms pink to orange bulbils (resting structures), received little attention as a turfgrass pathogen until the early 2000s. Since then the fungus has been observed with increasing frequency. There is ongoing research and debate on the identification of these isolates. In preliminary DNA sequencing studies, Tisserat has identified isolates from annual bluegrass from Kansas and Colorado and an isolate from Kentucky bluegrass from Missouri as *R. zae*. Others have reported that isolates from annual bluegrass are an unknown *Rhizoctonia* sp. with a *Waitea* sp. sexual stage. In this section we are reporting our experiences with this disease.

Symptoms and signs

In Missouri, leaf and sheath spot was first observed on a mixed Kentucky bluegrass/perennial ryegrass athletic field in 2000. Symptoms occurred only on the Kentucky bluegrass in this mixed stand. Since then the disease has been identified on other Kentucky bluegrass athletic fields and sod fields, on perennial ryegrass from a tee and in association with tall fescue. Symptoms in these situations occurred from early to late August under high temperatures.

The symptom on Kentucky bluegrass usually appears as sporadic, dinner-plate-sized rings of yellowed grass (Figure 1). The grass to the inside of the patch is usually darker green in color. Sometimes the disease appears to be more of a general blight (Figure 2). Sheath blight symptoms are commonly visible on individual plants at the margin of a patch (Figure 3).

The hyphae (individual fungal strands) of *Rhizoctonia zae* have the characteristic right-angled branching pattern typical of other *Rhizoctonia* species. Additionally, numerous bulbils have been observed in association with this dis-

ease on Kentucky bluegrass, perennial ryegrass and tall fescue. The pink to orange bulbils are about the size of BBs and range in shape from round to irregular. Bulbils have been observed on the surface of decaying grass tissue and embedded in leaf tissue (Figures 4 and 5).

R. zae has also been reported to cause yellow ring symptoms on creeping bentgrass and annual bluegrass on putting greens. On mixed greens, the disease usually occurs on one or the other species and not both. Bright yellow rings reaching several feet in diameter form in May and June under moderate temperatures (Figure 6). Leaves and leaf sheaths may not exhibit extensive symptoms, but fungal mycelium characteristic of *Rhizoctonia* spp. is usually present on tissue. Bulbils have rarely been observed in association with the yellow ring symptom on bentgrass or annual bluegrass in contrast to the disease on Kentucky bluegrass, perennial ryegrass and tall fescue. Symptoms on putting greens may resemble those of fairy ring or yellow patch. Laboratory examination may be necessary for confirmation.



Figure 1. Leaf and sheath spot of Kentucky bluegrass.



Figure 2. Leaf and sheath spot of Kentucky bluegrass.



Figure 3. Leaf and sheath spot of Kentucky bluegrass, caused by *Rhizoctonia zae*.

Management

Cultural management strategies used to suppress other disease caused by *Rhizoctonia* spp. should be helpful in managing leaf and sheath spot. For example, core cultivation in the fall will help improve infiltration rates and prevent saturated conditions at the surface. Avoid overwatering, especially late in the day.

Managing the thatch layer to avoid excessive buildup should also help to manage leaf and sheath spot. Avoid large amounts of quick-release nitrogen when the disease is active.

On athletic fields or other sites with a history of leaf and sheath spot occurring at high temperatures, a preventive fungicide application should be made just before the historic onset of symptoms. For the most part, fungicides used to suppress

other *Rhizoctonia* diseases should also be effective in controlling leaf and sheath blight, although the MBC group has been reported to be ineffective. Variability in efficacy against *R. zaeae* has also been reported among other groups of fungicides. Check local recommendations for recommended fungicides. To date, no products have *R. zaeae* specifically listed on the label.

Conditions

Little is known about the conditions favoring disease development on Kentucky bluegrass, except that in Missouri it has been observed under high temperatures in August. In contrast, ring symptoms on putting greens tend to occur in late spring or fall during relatively mild temperatures.

As with other diseases caused by *Rhizoctonia* spp., high relative humidity, abundant soil moisture, and heavy rainfall or irrigation tend to make leaf and sheath spot more severe.

Symptoms can be confused with those of

- brown patch
- summer patch
- fairy ring
- yellow patch
- white grub damage



Figure 4. Bulbils of *Rhizoctonia zaeae* in association with Kentucky bluegrass leaves.

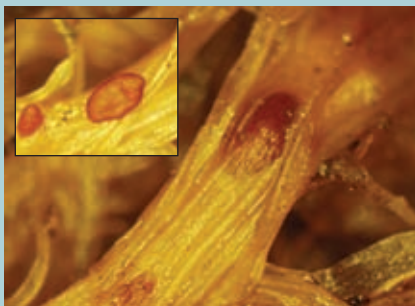


Figure 5. Bulbils of *Rhizoctonia zaeae* in leaf tissue of perennial ryegrass. *Inset*: Close-up of bulbils.



Figure 6. Yellow ringlike symptoms associated with *Rhizoctonia zaeae* on a bentgrass/annual bluegrass putting surface.

N. Tisserat

Microdochium patch (pink snow mold)

Pathogen: *Microdochium nivale*

Hosts: Bentgrass, annual bluegrass, occasionally perennial ryegrass and Kentucky bluegrass

Although Microdochium patch is often referred to as pink snow mold, snow cover is not required for disease development. The disease is most common on new bentgrass greens under cover or in situations where nitrogen fertility was high going into the winter.

Symptoms and signs

Symptoms of Microdochium patch are variable. On creeping bentgrass, the more familiar symptom is a distinct circular to irregular patch ranging in size from 1 to 8 inches in diameter (Figure 1). The patch appears red-brown and water-soaked at first, but rapidly fades to a pinkish white with a red-brown border. Microdochium patch on creeping bentgrass can also occur as a water-soaked, reddish foliar blight instead of a distinct patch. The foliar blight phase is especially common in mid to late spring. In this foliar blight phase, *Microdochium nivale* is readily spread by mowing. Because the symptom pattern coincides with the mowing pattern, the disease has been misdiagnosed as Pythium foliar blight because the latter is also readily spread by mowing. Pythium foliar blight, however, is a high-temperature disease. On higher cut turf such as Kentucky bluegrass, abundant mycelium (aggregate mass of vegetative growth) can often be seen in association with blighted turfgrass (Figure 2).

The pink color is due in part to the development of fruiting bodies known as sporodochia. The white to pink sporodochia are abundant on the leaf surface under cool wet conditions (Figure 3). Conidia produced on the sporodochia are crescent shaped with one or two septa (cross-walls) (Figure 4).

Spring outbreaks of Microdochium patch on putting surfaces are often mild with a few sporadic patches per green. The disease is often observed in association with yellow patch. The symptoms generally disappear with warmer temperatures.



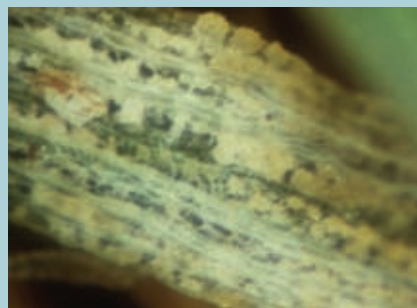
M. Matthews

Figure 1. Microdochium patch on bentgrass putting surface.



N. Tisserat

Figure 2. Microdochium patch of Kentucky bluegrass.



J. Thompson

Figure 3. Sporodochia (fruiting bodies) of *Microdochium nivale* on a bentgrass leaf.

Management

In regions where the snow molds *Microdochium patch* and gray snow mold occur at the same time as a chronic problem, the best disease management tool is a preventive fungicide application. Refer to Table 5 for a list of fungicides labeled for control of *Microdochium patch*. Not all products labeled for control of *Microdochium patch* are efficacious against gray snow mold and vice versa. In this situation the fungicide application should

contain more than one product to be certain that protection is provided against both diseases. In these regions, the preventive application should be made just before the first anticipated snowfall or just before applying winter covers.

In the lower Midwest where gray snow mold is not a problem, not all greens will need a preventive fungicide application specifically for *Microdochium patch*. New greens under cover or greens with dense, lush growth following overseeding to repair summer damage should generally

receive a preventive fungicide application for *Microdochium patch*. Recent research results have also shown a benefit to applying a late fall fungicide as a clean-up, especially on greens with a history of anthracnose basal rot. Curative fungicide applications may be necessary during seasons with prolonged cool, wet weather in early spring.

Avoid late-season application of water-soluble nitrogen. The turfgrass should be mowed into late fall to avoid long leaf blades before the onset of winter.

Conditions

The growth of *M. nivale* is favored by cool temperatures (32 to 46 degrees F) and high relative humidity or frequent precipitation. Even with warmer daytime temperatures in the spring, *Microdochium patch* can develop in response to nighttime temperatures in the high 40s to low 50s and frequent rainfall.

Dense, lush growth resulting from heavy nitrogen application is especially susceptible to *M. nivale*. The disease is more severe on new greens, greens under cover or greens that have been repaired with overseeding and heavy fertilization to promote recovery.

Symptoms can be confused with those of

- Pythium blight
- copper spot



Figure 4. Conidia of *Microdochium nivale*.

Necrotic ring spot

Pathogen: *Ophiosphaerella korrae*

Hosts: Primarily Kentucky bluegrass but also red fescue and annual bluegrass

Necrotic ring spot is a destructive disease of Kentucky bluegrass, but may also occur on red fescue and annual bluegrass. The disease is particularly damaging to bluegrass, because *Ophiosphaerella korrae* infects and kills the roots and crowns, resulting in slow recovery. The disease is also likely to occur in the same location year after year.

Symptoms and signs

The first symptoms usually appear in early fall or late spring. Initially, light green to straw-colored patches from several inches to several feet in diameter develop in the turf (Figure 1). As the disease progresses, patches develop more of a doughnut or frog-eye pattern. The ring becomes matted and craterlike, whereas the grass inside the ring remains healthy and green (Figure 2). The patches may be localized to one part of the turf area (Figure 3) or widely scattered throughout. Rings may eventually coalesce to create larger arcs of dead turf (Figure 4). Symptoms may persist throughout the growing season and increase in size and severity in successive years.

Bluegrass infected by *Ophiosphaerella korrae* exhibits extensive root and crown discoloration and rot (Figure 5). This can be observed by cutting a small sod piece from the lawn and carefully washing off soil and organic matter from roots. Darkly pigmented fungal hyphae may be visible on the root surface with the aid of a hand lens. The presence of large frog-eye patches and

root discoloration are usually diagnostic; however, necrotic ring spot can be confused with summer patch. Summer patch symptoms usually do not appear until late June or July and then symptoms tend to disappear with cooler weather in the fall. Also, frog-eye patches associated with summer patch tend to be smaller than those associated with necrotic ring spot. Plant disease diagnostic labs can verify the presence of necrotic ring spot.

Small pear-shaped pseudothecia (fruiting bodies) may form on decaying roots and crowns, but this rarely occurs in the field (Figure 6). Darkly pigmented multicelled ascospores are produced in asci (sac-like structures) within the pseudothecia (Figure 6, inset).

Conditions

O. korrae survives from year to year on dead, colonized bluegrass roots and crowns or on the surface of living roots. The fungus actively colonizes the outside of roots at soil temperatures between 65 degrees and 80 degrees F. In the Midwest, these temperatures occur in the



N. Tisserat

Figure 1. Necrotic ring spot symptoms initially develop as light yellow to straw-colored rings or frog-eyes in the turf area.



N. Tisserat

Figure 2. Advanced necrotic ring spot symptoms with a frog-eye appearance.



N. Tisserat

Figure 3. Symptoms of necrotic ring spot may be localized to certain areas of the turf.

Management

Necrotic ring spot can be a difficult disease to manage, but there are several genetic, cultural, and chemical options that can help minimize damage. Several varieties of Kentucky bluegrass, including 'Adelphi', 'Apex', 'Eclipse', 'Kelly', 'Midnight', 'NuBlue', 'Nugget', 'NewStar' and 'Wabash' have moderate to good resistance to *O. korrae*. Consider using these when establishing a new area from seed or overseeding extensively damaged areas. Necrotic ring spot is not a problem on perennial ryegrass or tall fescue, so depending on the region, these grasses can also be used to overseed damaged areas. Mixing perennial ryegrass seed with resistant bluegrass seed (20:80 ratio based on weight) can also suppress necrotic ring spot. However, some people

object to the reduced turf quality associated with this grass mixture.

Core cultivate the turf area at least once a year (spring or fall) to help reduce thatch buildup and improve soil drainage. Do not overwater. Water the lawn to a depth of 6 to 8 inches as infrequently as possible without creating moisture stress. Maintain a mowing height of 2.5 to 3 inches.

Avoid excessive amounts of nitrogen fertilizer (more than 4 lb N/ 1,000 square feet), especially when most of this nitrogen is applied in the fall. Consider the use of slow-release formulations of nitrogen. This allows for more uniform release of nitrogen through the growing season and avoids "bursts" of growth following fertilizer application.

If necrotic ring spot develops, water the grass lightly at midday during periods

of high temperatures. This will help cool the turfgrass and may prevent additional turfgrass death.

Preventive fungicide applications may be effective in suppressing necrotic ring spot. Refer to Table 5 for a list of fungicides labeled to control this disease.

Timing of fungicide applications is critical for effective disease management. Products must be applied in the spring before root colonization by the fungus occurs. Apply fungicides in May when soil temperatures reach 65 degrees F at a depth of 2 inches. Make a second application in 30 to 60 days to maximize disease control. Fungicide applications suppress disease development but do not eradicate *O. korrae*. Applications over several years may be necessary to manage necrotic ring spot on severely damaged turf.

spring and fall, but along the Front Range of the Rockies they occur from mid-May through mid-September, with some variation on the west slope and at higher elevations. *O. korrae* eventually penetrates roots and colonizes the root cortex. This either debilitates or kills roots and leads to a decrease in water and nutrient uptake by the plant. Plants with extensively damaged root systems are more prone to injury or death, particularly during periods of high temperature or drought stress. Thus, symptoms often do not show up until late summer or early fall even though root colonization occurs earlier. Symptoms may persist through the winter.

Symptoms generally appear two to three years after turf establishment, although in some cases the disease may not develop for a decade or more. The disease tends to be more severe in areas established from sod and on sites with compacted soils. Excessive irrigation and fertilization may also exacerbate the severity of the disease.

Symptoms can be confused with those of

- leaf and sheath spot
- Microdochium patch
- summer patch
- yellow patch



Figure 4. Advanced necrotic ring spot symptoms with extensive rings and arcs of dead grass.



Figure 5. Root rot of Kentucky bluegrass infected with *Ophiostoma korrae*.

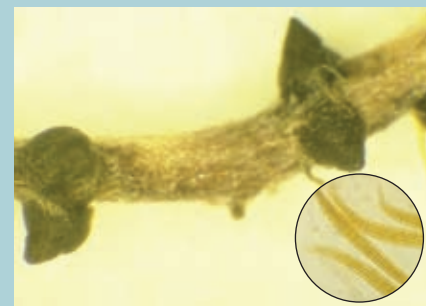


Figure 6. Pseudothecia (fruiting bodies) of *Ophiostoma korrae*. Inset: Darkly pigmented, multicelled ascospores within asci.

Pythium foliar blight

Pathogen: *Pythium* spp., primarily *P. graminicola* and *P. aphanidermatum*

Hosts: Perennial ryegrass, creeping bentgrass, Kentucky bluegrass and tall fescue

Pythium foliar blight is one of the most feared turfgrass diseases, because the disease develops rapidly during periods of high temperature and high relative humidity. If left untreated, extensive loss of turf can occur in a few days. The disease is most destructive on perennial ryegrass and tee- or fairway-height creeping bentgrass. Pythium foliar blight is rare on creeping bentgrass putting greens. Kentucky bluegrass and tall fescue are less commonly affected.

Symptoms and signs

On creeping bentgrass and perennial ryegrass mowed at fairway height, Pythium foliar blight first appears as scattered patches up to 4 inches in diameter (Figure 1). The patches often have a pinkish to red hue and a frogeye appearance, with greener grass in the center (Figures 2 and 3). The patches may merge and form streaks because the causal agent is readily spread by water movement in drainage patterns or by mowing equipment (Figure 4). Diseased plants eventually turn straw color and wither, resulting in craterlike depressions in the turf (Figure 5).

Pythium foliar blight occasionally affects tall fescue and Kentucky bluegrass during hot, humid weather. Softball-sized patches of water-soaked turfgrass form during hot, wet conditions. Diseased grass blades within the patch tend to stick to one another and have a mushy texture. Spots may coalesce and streaks may also form. Affected plants eventually wilt and die to the crown. However, crowns and roots

may not be killed, and plants may recover after several weeks.

When Pythium foliar blight is active, the margins of the patches are gray, and cottony mycelium may be evident, especially early in the morning (Figures 6 and 7). Lobate sporangia (reproductive structures) and hyphae with no cross walls are characteristics of *Pythium* spp. (Figure 8).

Pythium foliar blight is erroneously believed to be common on creeping bentgrass putting greens. In fact this disease rarely occurs on bentgrass mowed less than ¼ inch in height. It is not uncommon to find Pythium foliar blight on the putting green collar but see no damage on the putting surface.

Pythium spp. are also a common cause of damping off and seed rot during turfgrass establishment.

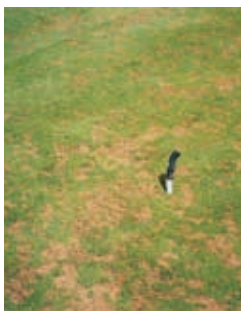


Figure 1. Pythium foliar blight on perennial ryegrass.



Figure 2. Pythium foliar blight on perennial ryegrass.



Figure 3. Pythium foliar blight on creeping bentgrass.



Figure 4. Spread of Pythium foliar blight in direction of water movement across the surface.

Management

Water management is the key to controlling Pythium foliar blight. Improve drainage in areas where water is likely to stand for any length of time. Avoid overwatering, especially during hot, humid periods. Promote rapid drying of turfgrass by proper spacing and pruning of shrubs and trees. Irrigate between midnight and early morning to reduce the number of

hours that grass leaves remain moist.

Excessive nitrogen fertilization stimulates lush growth that is more susceptible to Pythium foliar blight. Maintain a proper balance of nutrients and avoid fertilizing during periods of Pythium foliar blight activity.

Preventive applications of fungicides labeled for Pythium blight should be made during the summer months on perennial ryegrass or creeping bentgrass fairways and

tees. Refer to Table 5 for a list of fungicides labeled for control of Pythium foliar blight. Although Pythium foliar blight is rare on greens-height bentgrass, an application of a fungicide labeled for Pythium can be made at the onset of Pythium-favorable weather as added insurance. Newly sodded lawns, which must be kept constantly wet until rooted down, may benefit from a preventive fungicide application.

Conditions

Pythium foliar blight is most active when air temperatures are high (day temperatures 86 to 95 degrees F and minimum nighttime temperatures of 68 degrees F). Long dew periods, high relative humidity and lush, dense turfgrass growth favor disease development. The disease is severe in low-lying areas where soil is saturated with water following extended periods of rain or overwatering. Compacted and poorly drained sites are also prone to Pythium foliar blight. The disease will commonly develop along water drainage patterns. It also occurs on sites with poor air movement.

Symptoms can be confused with those of

- copper spot
- moisture stress
- grub damage

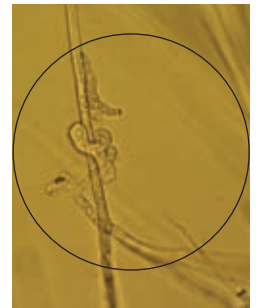


Figure 8. A lobate sporangium (reproductive structure of a *Pythium* sp.) and hypha with no cross walls.



Figure 5. Advanced Pythium foliar blight in Kentucky bluegrass.



Figure 6. Active mycelium of *Pythium* spp. in tee-height creeping bentgrass.



Figure 7. Active mycelium of *Pythium* sp. in tall fescue.

N. Tisserat

Rust

Pathogen: *Puccinia* spp.

Hosts: All turfgrasses

Rust occurs to some extent on all turfgrasses, but the rust fungi are generally host specific. Rust is most severe on susceptible cultivars of Kentucky bluegrass, tall fescue, perennial ryegrass and zoysiagrass. Rust symptoms usually appear in late August to early September and continue through the fall months. Rust can also develop at other times of year, depending on environmental conditions, turfgrass species and rust species. Disease severity can vary dramatically from one year to another.

Symptoms and signs

From a distance, rust-infected turf appears dull yellow or dark brown to purple (Figure 1). The rust fungi that infect turfgrasses produce two types of spores that are easily observable in grass leaf blades. The first to develop are urediniospores. The fruiting bodies containing urediniospores rupture the leaf epidermis, resulting in rust pustules that are visible to the naked eye (Figure 2). Rust pustules range in color from cinnamon brown to orange or yellow, depending on the species of rust fungus (Figure 3). The powdery spore mass rubs off easily on fingers, shoes or clothing. The color, size and shape of urediniospores vary with *Puccinia* sp. (Figure 4). Urediniospores serve as a source of inoculum to reinfest healthy turfgrass in the local area or, when wind-borne, to initiate infection in other areas.

The disease may be more difficult to diagnose later when pustules turn black (Figure 5). The blackened fruiting bodies contain a second type of spore — teliospores (Figure 6). Teliospores germinate to produce basidiospores that

are dispersed by wind to infect alternate non-grass hosts. Although sexual reproduction and recombination occurs on the alternate host, which varies with rust species, this stage of the rust disease cycle is considered of minor importance in turfgrass. Continuous heavy infection causes grass blades to turn yellow, wither and die, resulting in stand thinning (Figure 7). Severely rusted stands are more prone to winterkill.



Figure 1. Brown discoloration of Kentucky bluegrass infected by a *Puccinia* sp.



Figure 2. Rust pustules of a *Puccinia* sp. on Kentucky bluegrass leaves.



Figure 3. Rust pustules of a *Puccinia* sp. on Kentucky bluegrass leaves.

N. Tisserat

Management

Turfgrasses provided with optimal levels of fertilizer and water are less likely to be severely damaged by rust. Avoid night watering which increases the length of time the leaf blades remain wet. Regular mowing that removes infected leaf tips from the plant will help reduce inoculum levels. The best strategy is to mow frequently at a height not less than what is recommended

for the turfgrass. Avoid close mowing or scalping of the turf.

For long-term disease management, choose grass cultivars with a high level of resistance to rust. Check the National Turfgrass Evaluation Web site at <http://ntep.org> for the most up-to-date information on relative resistance of selected turfgrass cultivars to rust.

Refer to Table 5 for a list of fungicides labeled for the control of rust. In general these products work best when applied

early in the development of the disease. The decision to use fungicides is often difficult because applications need to be made relatively early in rust development. Because the rust epidemic is dependent on weather, it is hard to determine if early fungicide sprays are warranted. In most years rust does not reach damaging levels before the turfgrass enters winter dormancy, so fungicides are not routinely used for rust control in home or commercial landscapes.

Conditions

The rust fungi (*Puccinia* spp.) overwinter as urediniospores in infected plants or are reintroduced each summer by wind-blown spores. Infection of leaf blades is favored by moderate temperatures (68 to 85 degrees F) and extended wet periods. The disease tends to be more severe in partially shaded areas, such as under trees or along fence rows. Once infection occurs, slightly higher temperatures favor symptom development. Turfgrasses under stress (nutrient deficiency, drought, shading, close mowing heights and high temperatures) are more likely to be seriously damaged by the disease.



Figure 7. Severe rust infection in Kentucky bluegrass.

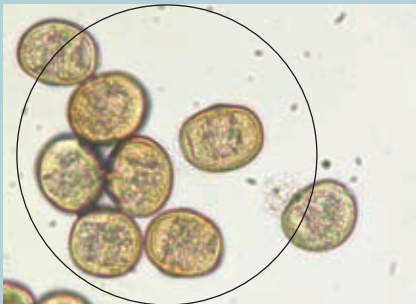


Figure 4. Urediniospores of a *Puccinia* sp.



Figure 5. Blackened fruiting structures of a rust fungus containing teliospores.



Figure 6. A close-up of teliospores of a rust fungus.

Spring dead spot

Pathogen: *Ophiosphaerella* spp.

Hosts: Bermudagrass and buffalograss

Spring dead spot is a destructive disease of common bermudagrass and bermudagrass hybrids throughout the northern range of its adaptation in the United States. Three different root-rotting fungi, known as *Ophiosphaerella herpotricha*, *O. korrae* and *O. narmari*, are responsible for the disease. The relative frequency of the different species varies by region; *O. herpotricha* is found most frequently in the Midwest. Spring dead spot also infrequently occurs on buffalograss. Cool-season grasses are not susceptible to spring dead spot, but *O. korrae* causes necrotic ring spot of Kentucky bluegrass with patch symptoms occurring in late spring and fall.

Symptoms and signs

Spring dead spot appears as circular or arc-shaped patches of dead turf in the early spring as bermudagrass breaks winter dormancy. The dead patches are slightly depressed and straw-colored, ranging in size from several inches to several feet in diameter (Figure 1). The patches normally are randomly distributed throughout the lawn or fairway (Figure 2).

Roots and stolons of infected plants are dark brown to black and are severely rotted (Figure 3). The root rot symptoms are best observed near the margin of the dead area. Roots infected with *Ophiosphaerella* spp. turn dark brown to black as a result of extensive growth of the darkly pigmented fungus in and on the roots (Figure 4).

During the summer, bermudagrass slowly recolonizes the bare areas, but broadleaf weeds and other weedy grasses can also invade and colonize the bare soil, resulting in a clumpy or patchy appearance to the turf (Figure 5). By late summer there may be little or no evidence of the disease. Dead patches reappear the fol-

lowing spring in the same locations. Over a number of years, the patches can become quite large, coalesce, and develop arclike patterns in the turf. After seven to 10 years, disease severity may begin to decrease to the point where the disease no longer occurs.



Figure 1. Spring dead spot of bermudagrass.

B. Fresenburg



Figure 2. Spring dead spot of bermudagrass.

N. Tisserat

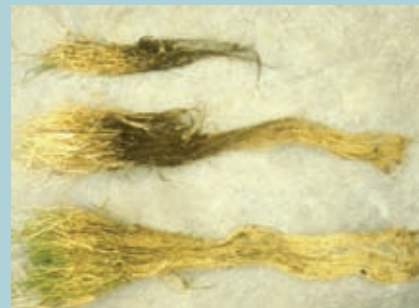


Figure 3. Root symptoms of spring dead spot on bermudagrass compared with a healthy root system (bottom).

N. Tisserat

Management

The best way to manage spring dead spot is through cultural methods that promote vigorous but not excessively lush turf. Dethatch bermudagrass lawns and fairways yearly during active growth to promote good rooting. Also core cultivate to help reduce thatch buildup and improve soil drainage.

A well-balanced and timed fertilizer program will also reduce the incidence of spring dead spot. Applications of excessive amounts of nitrogen (greater than 4 lb nitrogen per 1,000 sq. ft.) will produce excessive amounts of leaf tissue in relation to root tissue, thereby creating stressed tissue more vulnerable to infection. Severity of spring dead spot is lessened by low soil pH, therefore applications of ammonium sulfate (1 lb nitrogen per 1,000 sq. ft.) monthly from June to August will help

reduce the disease incidence.

Application of 0.8 lb of potassium chloride (KCl) per 1,000 sq. ft. at monthly intervals during the summer has also been shown to reduce spring dead spot severity.

Bermudagrass cultivars vary markedly in susceptibility to spring dead spot. None are immune to the disease, but several seeded and vegetatively propagated cultivars exhibit reduced patch sizes and faster summer recovery than susceptible varieties. Resistant vegetative cultivars include 'Midlawn' and 'Patriot'. Another popular variety called 'Quickstand'; is moderately susceptible to spring dead spot. The seeded cultivars 'Guymon', 'Yukon' and 'Reviera' also show moderate resistance. 'Guymon' has a coarse texture and is not recommended for home lawns in the Midwest. Several other experimental seeded selections from Oklahoma State University

also appear promising. Avoid using Arizona common bermudagrass, 'Cheyenne', 'Jackpot', 'Oasis', 'Poco Verde', 'Primavera', 'Sonesta', 'Tifton 10', 'Tifway' (Tifton 419), 'Tropica', 'Vamont' or 'Sunturf' cultivars.

Several fungicides, including several demethylation inhibitors (DMIs) and azoxystrobin (Heritage), are labeled for management of spring dead spot (Table 5). However, disease suppression with these fungicides has been erratic in trials. September applications of these fungicides have, in some years, reduced disease severity and enhanced recovery of the turf in the spring, but they did not completely control spring dead spot development. Therefore, fungicide applications are not currently recommended for control of spring dead spot in the Midwest, and the emphasis for managing this disease should focus on cultural methods.

Conditions

Spring dead spot may occur on bermudagrass lawns of all ages, although it typically appears three to four years after the turf has been established.

Spring dead spot fungi colonize roots, stolons and crowns of bermudagrass plants in late summer or fall. Although most fungal colonization occurs in the fall, foliar symptoms do not develop until late spring. During winter dormancy, infected bermudagrass roots or crowns are either killed directly by the fungus or predisposed to desiccation or cold-temperature injury.

Root colonization by the pathogen decreases the cold hardiness of the bermudagrass.

The disease tends to be more severe on high-maintenance bermudagrass lawns and fairways. Excessive nitrogen fertilization during the summer months enhances symptom development the following spring.

Symptoms can be confused with those of

- Winterkill



N. Tisserat

Figure 4. Close-up of roots infected with one of the fungi causing spring dead spot.



B. Fresenburg

Figure 5. Recolonization of the dead areas by annual bluegrass (upper right) and bermudagrass.

Summer patch

Pathogen: *Magnaporthe poae*

Hosts: Kentucky bluegrass, annual bluegrass and fine fescues

Summer patch, sometimes referred to as frog-eye patch, is a destructive disease of Kentucky bluegrass maintained for golf course fairways, green surrounds, parks and residential landscapes. The disease usually appears two to three years after turf establishment from seed or sod and then reappears in subsequent years in the same location. Summer patch is also a problem on annual bluegrass on putting surfaces. Summer patch was recently reported as a disease of creeping bentgrass in North Carolina.

Symptoms and signs

Aboveground symptoms of summer patch in Kentucky bluegrass initially develop in June through August as small patches of turf, 2 to 6 inches in diameter. Grass blades in the patch change to a dull reddish brown, then tan, and finally a light straw color. In the final stages of the disease, circular patches of dead grass form throughout the turf area. Healthy grass often occurs at the center of the patch, resulting in a characteristic frog-eye pattern (Figure 1). Circular patch symptoms develop because the fungus grows radially from a central infection point.

When weather conditions are ideal for disease development, affected areas may overlap, resulting in large areas of affected turf (Figures 2, 3). In mixed stands of grasses, the pattern of symptoms is much more irregular.

Summer patch can also cause extensive damage to annual bluegrass on golf course putting greens. Infected annual bluegrass surrounding unaffected bentgrass clones turns yellow to bronze (Figure 4). This preferential

damage of annual bluegrass is a useful diagnostic feature of the disease.

Roots and crowns of infected plants are discolored (Figure 5). With a compound microscope, darkly pigmented runner hyphae are visible in and around infected roots (Figure 6).

Symptoms of summer patch can be confused with leaf and sheath spot, insect damage (grubs, billbug), or drought stress. These possible causes should be eliminated before a diagnosis of summer patch is made. The best means of confirming this disease is to submit a turfgrass sample along with images of the symptoms to a plant disease diagnostic lab for confirmation. The turfgrass sample should be cut from the margin of the patch that contains both healthy and diseased turf. Ideally, the sample should be 6 inches square and deep enough to include much of the root system.

Conditions

Although aboveground symptoms of summer patch don't appear until summer, *Magnaporthe poae* colonizes grass roots and crowns in



Figure 1. Summer patch on Kentucky bluegrass lawn. Note frog-eye appearance.



Figure 2. Summer patch of Kentucky bluegrass.



Figure 3. Summer patch of Kentucky bluegrass.

Management

Summer patch is a difficult disease to control. The causal fungus can survive for many years in the soil, and symptoms often develop yearly in the same location. For this reason, on high-profile, high-visibility Kentucky bluegrass stands and annual bluegrass putting surfaces, a preventive fungicide program is recommended. Refer to Table 5 for a list of fungicides labeled for control of summer patch.

Chemical control has not been completely effective in managing the disease but can reduce disease severity if used in conjunction with good turf management. Timing of fungicide applications is critical for effective control. The first preventive application should be made in the spring when soil temperature at a depth of 2 inches remains above 65 degrees F. Timing varies with geographic location, but generally it will be in mid-April in the Midwest. A second application should be made one month after

the first.

Summer patch is more difficult to control with chemicals once symptoms develop. Although fungicide at this time may inhibit the fungus, plants are slow to recover because of high temperatures. A light application of nitrogen may be just as beneficial at this time as a fungicide application.

Any fungicide program should be used in combination with cultural practices to alleviate stress. Turfgrass should be maintained in a vigorous, but not over-stimulated, growing condition. A balanced fertilization program is important. Avoid excessive nitrogen fertilization during the summer months. Seventy-five percent of the nitrogen fertilizer should be applied in the fall. Use of acidifying nitrogen sources such as ammonium sulfate reduces summer patch severity. Slow-release nitrogen fertilizers should be used for spring fertilization.

Mowing heights should be maintained above 2 inches on residential lawns. Thatch

reduction is important for suppressing disease development, and problem lawns should be dethatched or core-aerated yearly.

Diseased turf should be lightly watered (syringed) daily in the early afternoon to cool the plants and provide moisture for the diseased roots.

Certain Kentucky bluegrass cultivars have shown some tolerance to summer patch. The older cultivars Adelphi, Admiral, America, Baron, Bristol, Challenge, Columbia, Eclipse, Majestic, and Monopoly are moderately resistant to the disease. Unfortunately, many of the newer varieties have not been extensively tested for resistance to this disease. The common type Kentucky bluegrasses, including Park, Kenblue, SouthDakota Certified, Ginger, Alene and Greenley are more susceptible to summer patch and should be avoided. In chronic disease situations with Kentucky bluegrass, it may be appropriate to overseed the area with turf type tall fescue, which is not susceptible to summer patch.

the spring. It first grows on the root surface (ectotrophically), but later invades the inner root tissue. Conditions in spring are optimal for turfgrass root growth, and even though fungal colonization is occurring, the turfgrass is able to absorb water and maintain growth.

Infected roots either die or become dysfunctional during the hot, dry summer months. Disease symptoms can be particularly severe

on south-facing slopes. Infected roots are no longer able to supply adequate water to the foliage and plants exhibit aboveground symptoms of moisture stress. Severely damaged plants may die. In others, recovery is slow because generation of new roots is inhibited by high soil temperatures, and plants cannot rapidly replace diseased roots.

Symptoms can be confused with those of

- leaf and sheath spot
- white grub or billbug damage
- moisture stress



Figure 4. Summer patch of annual bluegrass on a mixed bentgrass/annual bluegrass putting surface.

N. Tisserat



Figure 5. Discolored roots and crowns of Kentucky bluegrass infected with *Magnaporthe poae*.

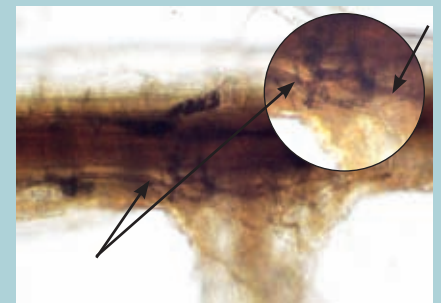


Figure 6. Darkly pigmented runner hyphae of *Magnaporthe poae* in infected root tissue.

Take-all patch

Pathogen: *Gaeumannomyces graminis* var. *avenae*

Host: Creeping bentgrass

Take-all patch of creeping bentgrass is a soilborne disease caused by a darkly pigmented, ectotrophic root-infecting fungus. The disease is most common on new greens. It also occurs on reconstructed greens, especially when methyl bromide had been used in the renovation.

Symptoms and signs

Take-all patch symptoms typically appear in early to mid-summer as dinner-plate-sized or larger patches of wilted, off-color turf (Figure 1). The patches usually have a frogeye appearance, with greener grass in the middle of the patch (Figure 2). The root systems of infected plants are greatly reduced in relation to those of healthy plants (Figure 3). Because of poor root function, symptom expression often follows a period when greens were allowed to dry out. The patches are easy to spot in the early morning, because dew tends to dry on patches first.

The roots of plants infected with *Gaeumannomyces graminis* var. *avenae* are deteriorated, with a black discoloration of the vascular cylinder (Figure 4). The roots have a shrunken appearance, in contrast to healthy white roots taken from outside the patch (Figure 5). Darkly pigmented ectotrophic runner hyphae of *G. graminis* var. *avenae* can be observed on the surface of infected roots (Figure 6). The simple hyphopodium from which the fungus infects

host tissue is one morphological characteristic used to help identify *G. graminis* var. *avenae* (Figure 6 inset).

In the interest of getting a quick answer to the superintendent, diagnosticians usually base their diagnosis on symptom expression in the field and microscopic observation of darkly pigmented runner hyphae and simple hyphopodia associated with the roots. The diagnosis made in this manner is presumptive because there are many nonpathogenic, darkly pigmented ectotrophic fungi associated with turfgrass roots. Confirmation using isolation and molecular techniques may be available at some universities with active research projects on the ectotrophic root-infecting fungi.

Conditions

Greens most at risk for take-all patch are new greens (two to five years old) carved out of woodlands or other areas where grass has not grown for years. Newly constructed greens or renovated greens where methyl bromide was used are also at risk. This risk is thought to be

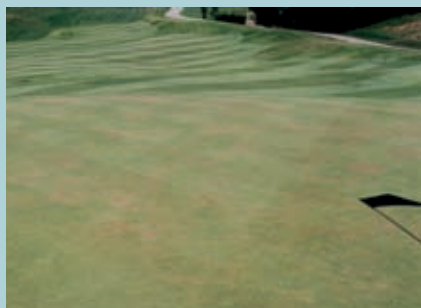


Figure 1. Symptoms of take-all patch on a creeping bentgrass green renovated from sod grown on a nursery green treated with methyl bromide.

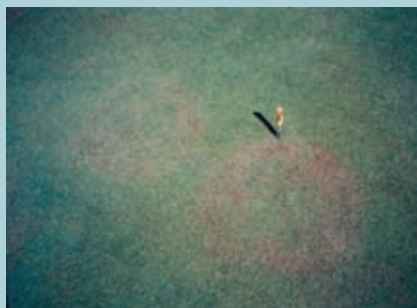


Figure 2. Take-all patch of creeping bentgrass.



Figure 3. A bentgrass plug taken from within the patch (left) has a smaller root system than a plug taken from healthy turf just outside the patch (right).

Management

Take-all patch is more severe under high soil pH. Use of nitrate fertilizer sources, which results in high pH at the rhizosphere, should be avoided when take-all patch is a problem. Ammonium forms of nitrogen such as ammonium sulfate should be used as the primary nitrogen source for one to two years following disease outbreak.

A preventive fungicide program is

recommended for new greens or greens with a history of the disease. Refer to Table 5 for a list of fungicides labeled for control of take-all. Make one fungicide application in late fall, followed by two applications in the spring, beginning in early to mid-April. Fungicide application after symptoms develop will have little effect on the disease, because symptom expression is a moisture stress symptom resulting from the inability of infected roots to meet the water needs

of the turfgrass plant. Frequent syringing in the summer will be necessary to reduce moisture stress symptoms.

Take-all patch is expected to go away on its own over time because of the phenomenon known as take-all decline. However, because the pH of the irrigation water in many regions is high, symptoms may persist, requiring the use of acid injection to lower the pH of the irrigation water.

due to the lack of diversity of soil microorganisms suppressive to *G. graminis* var. *avenae* on such sites.

G. graminis var. *avenae* is a poor competitor when confronted with a diversity of other soil microorganisms. Over time, the natural buildup of soil microorganisms suppressive to *G. graminis* var. *avenae* results in a reduction of take-all disease incidence and severity. This resulting take-all decline is a natural biological control phenomenon. Take-all decline has been extensively studied in the wheat/take-all system. The soils that suppress take-all have been a rich source of biological control organisms such as the fluorescent pseudomonad bacteria that are antagonistic to the take-all fungus. In areas where no grass has grown for years or in cases where the root zone was treated with methyl bromide, the populations of organisms suppressive to *G. graminis* var. *avenae* require time to build up.

Although symptoms typically don't occur until heat and moisture stress periods, infection occurs under cool, wet conditions in the spring and fall. Disease development is favored by high soil pH at the rhizosphere. Nitrate forms of nitrogen, which result in high pH at the rhizosphere, tend to increase the severity of take-all. Liming can also increase disease severity.

Symptoms can be confused with those of

- brown patch
- dry spot

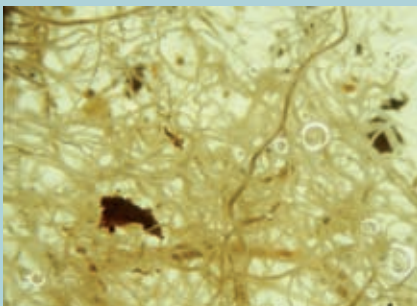


Figure 4. Discoloration of vascular cylinder of roots infected with *Gaeumannomyces graminis* var. *avenae*.



Figure 5. Healthy white roots of plants outside the patch.

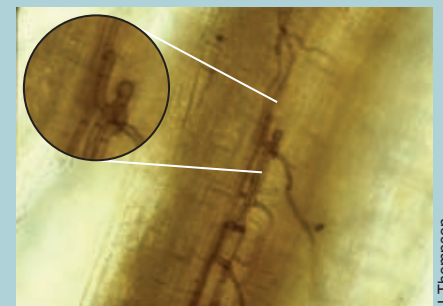


Figure 6. Runner hyphae of *Gaeumannomyces graminis* var. *avenae* in bentgrass roots. *Inset*: Simple hyphopodia characteristic of *G. graminis* var. *avenae*.

J. Thompson

Yellow patch

Pathogen: *Rhizoctonia cerealis*

Hosts: primarily creeping bentgrass, also annual bluegrass, Kentucky bluegrass

Yellow patch is a cool-season disease caused by *Rhizoctonia cerealis*. Yellow patch occurs on bentgrass and annual bluegrass putting greens and sometimes on higher-cut Kentucky bluegrass. Symptoms often disappear with warmer temperatures, but in some regions yellow patch on putting surfaces can be a severe and chronic problem.

Symptoms and signs

Symptoms can occur from October through late April. Yellow to rusty-red rings or arclike patterns ranging in size from a few inches to several feet develop in affected turf. In many cases, only a 1- to 3-inch-wide band of discolored turf is present and the turfgrass inside the ring may show no adverse effects (Figures 1 and 2).

In other cases, turf inside the ring is discolored a light yellow, lending a more patchlike quality to the symptoms on the putting surface (Figure 3). Early stages of yellow patch are most noticeable early in the morning, then difficult to see by midday. Infected plants exhibit a light, water-soaked lesion at the base of the leaf sheaf or in the crown tissue (Figure 4). Lesions on the leaf blade are rare.

A cobwebby growth of mycelium may be visible early in the day when dew is present, but it is sparser than that seen with brown patch. It is usually necessary to use a stereo microscope to detect the mycelium (Figure 5). The hyphae exhibit right-angled branching characteristic of

other *Rhizoctonia* species (Figure 6). *Rhizoctonia cerealis* is somewhat similar to, but distinct from, *Rhizoctonia* species that cause leaf and sheath spot, large patch of zoysiagrass and brown patch of cool-season turfgrasses. *R. cerealis* is binucleate (two nuclei per cell), in contrast to *R. zeae* and *R. solani*, which are multinucleate.

The fungus survives in the soil as bulbils (resting structures). Bulbils are initially light colored, then dark brown when mature. The pinhead-sized bulbils can be numerous in the thatch surrounding damaged turfgrass.

The rings are most conspicuous in the early spring, because they contrast sharply with newly emerging leaves. In most cases the symptoms remain superficial, with rings that are difficult to see. Infected plants recover quickly when temperatures increase. During prolonged cool, wet periods in late winter to early spring, patches can become necrotic and sunken. These damaged areas do not recover quickly.



Figure 1. Yellow patch of creeping bentgrass.



Figure 2. Yellow patch of Kentucky bluegrass.



Figure 3. Yellow patch of creeping bentgrass.

Management

Core cultivation in the fall can help reduce disease severity by improving the infiltration rate. Although there is no direct evidence that high nitrogen rates increase disease severity, avoid excessive nitrogen fertilization in fall. Other cultural practices appear to have little influence on disease development.

Yellow patch symptoms are generally superficial and will not require a targeted fungicide application. However, on golf courses experiencing chronic problems with severe yellow patch, a preventive fungicide program may be necessary to protect putting quality. Refer to Table 5 for a list of fungicides labeled for control of yellow patch. Make the first application before

the historic onset of symptoms or as soon as symptoms begin to develop. In most years, a single fall preventive application is sufficient to suppress disease development throughout the entire winter and spring. During wet, mild winters with heavy disease pressure, make a second fungicide application in late March.

Conditions

Yellow patch development is favored by extended periods of wet, cloudy weather. It is a cool-temperature disease (50 to 65 degrees F). Disease development is greatly suppressed at temperatures lower than 45 degrees F and greater than 75 degrees. Yellow patch tends to be more severe on putting greens with poor subsurface drainage.



Figure 4. Water-soaked lesion caused by *Rhizoctonia cerealis* on creeping bentgrass.



Figure 5. Mycelium of *Rhizoctonia cerealis* near a leaf sheath of creeping bentgrass.



Figure 6. Hyphae of *Rhizoctonia cerealis* with right-angled branching.

Yellow tuft (downy mildew)

Pathogen: *Sclerophthora macrospora*

Hosts: creeping bentgrass, Kentucky bluegrass, annual bluegrass, zoysiagrass, others less common

Yellow tuft is a cool-season disease that is commonly observed on creeping bentgrass and intensively managed Kentucky bluegrass. On creeping bentgrass greens, the disease is generally more cosmetic than damaging, although the disease is chronic in some locations. Yellow tuft can result in serious loss of quality in zoysiagrass sod production and on zoysiagrass green surrounds.

Symptoms and signs

On bentgrass or annual bluegrass putting surfaces, yellow tuft occurs as bright yellow spots ranging in diameter from ½ to 1 inch (Figure 1). The spots are individual plants infected with *Sclerophthora macrospora*, which results in the production of many more tillers than normal (Figure 2). The excess tillering is a condition commonly known as tufting. Tufted plants are shallowly rooted and easily removed from the turf surface. In some regions, infected plants are prone to winterkill, probably as a result of heaving due to the shallow roots (Figure 3).

Yellow spots in Kentucky bluegrass or zoysiagrass are larger than those seen on putting surfaces, depending on turfgrass density and height of cut (Figure 4). Where normal creeping bentgrass and Kentucky bluegrass commonly have fewer than eight tillers, infected plants have 15 or more (Figure 5). In infected zoysiagrass, the excess tillering is often associated with individual nodes rather than individual plants. Infected zoysiagrass can have 30 or

more tillers per node in contrast to the normal three to four (Figure 6).

Under cool moist conditions in spring and fall, *S. macrospora* produces sporangia (fruiting bodies) on turfgrass leaves. Sporangia are best seen early in the morning under heavy dew. They are pearly white and give a downy appearance to leaf surfaces when viewed with a hand lens. The sporangia are ephemeral, quickly collapsing when the leaf surface dries. In the field it is more common to see the collapsed sporangia as a gray to white crystalline residue on the leaf surface (Figure 7). When turgid, the sporangia are lemon shaped (Figure 7 inset) and contain zoospores (motile spores). Zoospores swim in free moisture to infect adjacent healthy meristematic tissue in just a few hours. Oospores (sexual spores) are sometimes produced in infected leaf tissue but are more numerous and easier to detect in crabgrass species.

In zoysiagrass sod fields and on zoysiagrass surrounds, infected stands are often thin and yellow as the grass breaks dormancy in the spring (Figures 8, 9 and 10). Infected zoysiagrass is stunted and extensively tufted (Figure

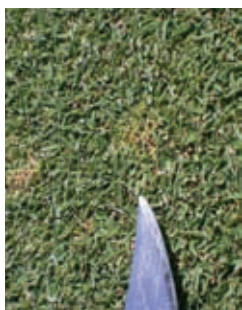


Figure 1. Yellow tuft of creeping bentgrass.

D. Minner



Figure 2. Excess tillering of bentgrass infected with *Sclerophthora macrospora*.

D. Minner



Figure 3. Winterkill of bentgrass infected with *Sclerophthora macrospora*.

D. Minner



Figure 4. Yellow tuft in zoysiagrass.



Figure 5. Excess tillering (tufting) of Kentucky bluegrass caused by *Sclerophthora macrospora*.

Management

Improve drainage as needed to remove water quickly. Where a chronic problem exists, address issues of poor drainage first.

Maximum susceptibility to *S.*

macrospora infection occurs when cool, wet weather coincides with the period when the turfgrass is producing the maximum amount of meristematic tissue for regrowth. For cool-season grasses already established on fairways and greens, this period occurs

in spring and fall. On thinned areas in zoysiagrass surrounds, this period begins in the spring and continues as long as the turf manager is fertilizing to encourage the zoysiagrass to fill in and the temperatures are generally below 70 degrees F. In sod production fields, this period may occur anytime after harvest as the sod producer is managing the zoysiagrass to regenerate. High temperatures and low relative humidity, however, may reduce severity of infection.

Although fosetyl-Al and mefenoxam

are labeled for management of yellow tuft, mefenoxam is usually the more effective. Fungicide should be applied beginning at the period of maximum susceptibility and repeated as needed. On zoysiagrass, core cultivate just before fungicide application to facilitate fungicide placement near meristematic tissue developing on rhizomes. The goal of fungicide application is to protect the new growth. Fungicide application will have no effect on grass that has already produced excess tillers.

10 inset). On zoysiagrass fairways, the disease often appears as a more diffuse yellowing without extensive tufting (Figure 11). Yellow tuft of zoysiagrass is more severe on shaded areas of fairways.

Conditions

Yellow tuft is favored by cool (41 to 68 degrees F), wet conditions. Severe symptoms can occur in low-lying, poorly drained areas of sod production fields or fairways. In turf prone to periodic flooding, especially in the spring or fall, *S. macrospora* infection is likely.

Crabgrass is very susceptible to yellow tuft, and large numbers of oospores, which serve as a source of inoculum, are produced in infected tissues. Sod production fields with heavy crabgrass pressure can have a greater incidence of yellow tuft.

Symptoms can be confused with those of

- Nutrient deficiency



Figure 11. Yellow tuft of zoysiagrass.



Figure 9. Yellow tuft of zoysiagrass.



Figure 10. Yellow tuft of zoysiagrass. Inset: Excess tillering of zoysiagrass caused by *Sclerophthora macrospora*.



Figure 6. Excess tillering (tufting) of zoysiagrass caused by *Sclerophthora macrospora*.

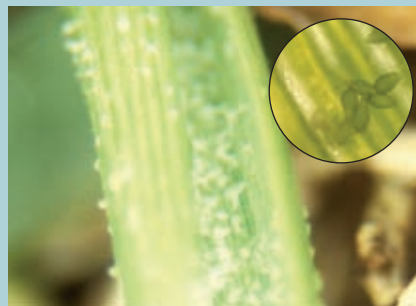


Figure 7. Collapsed sporangia of *Sclerophthora macrospora* on a zoysiagrass leaf. Inset: Sporangia of *S. macrospora* on a zoysiagrass leaf.



Figure 8. Yellow tuft of zoysiagrass.

