COOL SEASON DISEASE CONTROL

When confronted with disease in turfgrass, landscape managers must contend with diagnosing, treating and avoiding resistance to fungicides.

by Peter Landschoot, Ph.D., Penn State University

Of all the pests of turf, disease presents perhaps the most challenging problems.

Symptoms are helpful in disease diagnosis but are often unreliable if they are not observed during the early stages of development. Another difficulty in disease management is obtaining adequate control before the problem gets out of hand.

Root and crown disease

The major root and crown diseases that affect cool-season turf in the United States include take-all patch, summer patch, necrotic ring spot, and pythium root rot. Take-all patch, necrotic ring spot and summer patch are generally referred to as the patch diseases, although a distinction should be made between patch diseases caused by root pathogens and those caused by foliar pathogens (i.e. brown patch, fusarium patch, yellow patch and pink patch).

The first indication of patch disease is the presence of dark brown mycelium (sometimes called ectotrophic mycelium or tunner mycelium) on the root surface that cause root discoloration. Unfortunately, this is virtually impossible to detect without a microscope and special preparation of the root tissue. By the time the patches are visible, extensive root damage has occurred and curative treatment is ineffective. This is the main reason why root and crown diseases are so difficult to manage.

Stripe smut appears in Kentucky bluegrass and bentgrass as black streaks along the length of leaf blades.

Golf courses a target

Take-all patch is almost exclusively a disease of bentgrass golf course greens and fairways.

Symptoms of take-all patch typically appear as circular patches of dead or dying turf ranging from a few inches to several feet in diameter. Under conditions favorable for disease development, patches may coalesce and destroy large areas of turf. Since the roots and crowns of affected plants are usually destroyed, recovery of the turf is slow. Undesirable plants such as annual bluegrass or broadleaf weeds often colonize the patches.

Take-all patch occurs more frequently on sandy soils, soils low in phosphorus and potassium, and in soils of high pH (7.0 or greater).

Take-all patch is one of the most difficult diseases to control on golf course turf. Most fungicides that control other turf diseases do not consistently control take-all patch.

Manipulating cultural practices is usually the most effective means of managing this disease.

Previous studies have shown that reducing the soil pH by adding acidifying fertilizers (such as ammonium sulfate and ammonium chloride) or sulfur can reduce the its severity. Correspondingly, applications of lime are not recommended on turf affected with take-all patch.

Making certain that soil phosphorus and potassium levels are not deficient will help to increase turfgrass resistance to this disease.

Bane of bluegrass

Summer patch is one of the most destructive diseases of Kentucky bluegrass and annual bluegrass in the Northeast and Midwest. It has also become a problem on fine fescues in some areas.

Once established, summer patch destroys the roots and crowns of affected plants, causing death of the turf. Summer patch usually occurs during extended periods of high temperatures (83-95°F) and high humidity.

Recent studies at the University of Maryland have shown that the disease is more likely to occur in moist soils than under drought-stress conditions. The peak disease period occurs following heavy rains during warm, hot days in the summer. Summer patch is frequently observed in heavily-trafficked turf and in areas with poor soil drainage and reduced air circulation.

Since summer patch is a root and crown disease, cultural practices that promote good root growth will help reduce disease severity. Increased aeration and improved drainage on compacted and poorly-drained soils will alleviate some root inhibition. It will also enable the turf to better resist infection by the causal fungus.

Because low mowing heights are conducive to plant stress and shallow rooting, raising the height of cut may result in less summer patch injury. Reducing populations of susceptible species and encouraging more resistant species such as perennial ryegrass or creeping bentgrass is another means of reducing summer patch injury.

Summer patch can be controlled with fungicides, provided that: (1) applications are properly timed, (2) the most effective products are used at the correct rates, and (3) the fungicides reach the roots and crowns before the tissues are extensively invaded.

The best response has been achieved by applying the fungicides on a preventive basis, usually three to four weeks before symptoms are likely to appear.

Fungicides must reach roots and crowns to prevent or stop the infection. Since most systemic fungicides do not move efficiently from foliar portions of the plant to the root tissue, the fungicides should be applied so they can reach the roots.

One means of distributing fungicides into the root zone is by applying large amounts of water (5 to 10 gallons/1000 sq. ft.). Similar results can be obtained by irrigating immediately after fungicides are applied. It is important that the fungicide not be allowed to dry on the foliage before watering.

Ring spot hard to stop

Necrotic ring spot is primarily a disease of Kentucky bluegrass lawns in the Northeast, Midwest, and Northwest.

This fungus is also the causal agent of a disease of bermudagrass known as spring dead spot. Necrotic ring spot can affect most cool-season grasses; however, the grasses most often damaged by this disease are Kentucky bluegrass and fine fescues. Sometimes, this disease has been found on annual bluegrass golf course greens in New Jersey and Pennsylvania.

Necrotic ring spot usually occurs in late spring and/or early fall. The disease can also appear in the summer on drought-stressed turf. Research has begun to determine other factors responsible for disease development.

One of the most effective management practices for reducing the severity of necrotic ring spot is overseeding with perennial ryegrass.

Pythium root rot is characterized by thinning of the turf in small, tan-colored patches. It may progress to destroy large areas of grass. This disease is caused by several pythium species that can infect roots under cool (45-60 °F), moist conditions. This disease typically occurs in early spring or late fall.

The severity of pythium root rot can be reduced by using management practices that promote root development and reduce excessive soil moisture. However, fungicide applications may be justified when conditions are favorable for disease development.

Although more research needs to be conducted on the control of pythium root rot with fungicides, trials in Upstate New York have shown that Aliette (fosetyl) and Subdue (metalaxyl) reduce populations of the causal fungi when watered in immediately following application.

New resistance strategies

Fungicide resistance results from the repeated, continuous use of fungicides with the same or similar modes of action. This has been a particular problem of systemic fungicides because they tend to have a narrower mode of action than most contact fungicides.

Dollar spot can infect all cool-season turfgrasses. Lesions are white, often with brown borders. Look for white mycelium in early morning.

One type of resistance occurs when the initial pathogen population consists of members that are either very sensitive to the toxic effects of a particular fungicide or are very resistant. Loss of control is sudden and dramatic.

Another type of resistance occurs when the population consists of members that are very sensitive, slight or intermediate in sensitivity, slight or intermediate in resistance, or very resistant to a particular fungicide. Following continuous repeated use of the fungicide, loss of control is gradual.

Should a fungicide program be necessary, it is important to design a strategy to delay or prevent the onset of resistance. Two conventional approaches to preventing resistance have been to alternate fungicides with dissimilar modes of action.

A combined approach

A recommendation often made by plant pathologists is to mix a contact with a systemic fungicide. Whereas this approach appears logical (since systemics and contacts have distinct modes of action), there is some evidence that suggests that this is not the best resistance prevention strategy available.

A more logical approach is to combine two or more systemic fungicides with different modes of action. This eliminates combining contacts and systemics. Unfortunately, mixtures of systemics at full-label rates are costly and may result in turf injury. Turf managers should take the threat of resistance seriously and avoid continuous and repeated use of fungicides with narrow modes of action. **LM**

Dr. Landschoot is an assistant professor of turfgrass science at Penn State University.

TABLE 1

Diagnostic Features of Common Cool Season Turfgrass Diseases

Disease	Causal Agent(s)	Symptoms/Signs	Susceptible Grasses
Anthracnose	*Colletotrichum graminicola*	Yellowing of leaf blades associated with a black crown rot. Pin cushion-like fruiting bodies with small, spiney projections can be seen with a hand lens.	Annual bluegrass, bentgrasses, and fine fescues.
Brown patch	*Rhizoctonia solani*	Large, circular brown patches or thinning of turf. On low-cut turf, patches often surrounded by dark rings. White, cottony mycelium may be present on high-cut turf in early morning.	Bentgrasses, ryegrass, tall fescue.
Dollar spot	*Lanzia spp. Moellerodiscus spp. (Sclerotinia homeocarpa)*	Small, bleached patches of dead grass appear in turf. Lesions on leaves are white, often with brown borders. White, cottony mycelium may be present on dew-covered turf in early morning.	All cool-season turfgrasses.
Fairy ring	*Basidiomycete* fungi	Dark-green rings become apparent in mature turf. Mushrooms often present around periphery of ring.	All cool-season turfgrasses.
Leaf spot/ melting out	*Drechslera* and *Bipolaris spp.*	Small tan lesions with purple or brown borders on leaf blades. In severe cases, the crowns are rotted and the turf may be significantly thinned.	Primarily Kentucky bluegrass. Other cool-season grasses may be affected.
Necrotic ring spot	*Leptosphaeria korrae*	Large ring-shaped patches, usually creating depressions in turf. Roots and crowns show brown or black rot.	Primarily Kentucky bluegrass. In some cases, fine fescues and annual bluegrass.
Powdery mildew	*Erysiphe graminis*	White, fluffy mycelium on leaf blades, usually present on turf growing in shaded areas.	Kentucky bluegrass.
Pythium blight	*Pythium aphanidermatum* & other *Pythium spp.*	Irregular patches of blighted turf. White, dense, cottony mycelium growing in turf in morning.	Perennial ryegrass, bentgrasses, tall fescue.
Pythium root rot	*Pythium spp.*	Small bleached patches of turf may progress to large blighted areas, crowns and roots rotted.	Bentgrasses, annual bluegrass, Kentucky bluegrass.
Red thread/ Pink patch	*Laetisaria fuciformis/ Limonomyces roseipellis*	Small red to pink patches of blighted turf. Long, slender threads of red mycelium (red thread), or fluffy, pink mycelium (pink patch) growing out of foliage.	Fine fescues, perennial ryegrass, Kentucky bluegrass.
Rust	*Puccinia spp.*	Yellowing of leaves often apparent. Brown pustules occurring on leaves and stems.	Tall fescue, perennial ryegrass, Kentucky bluegrass.
Slime molds	*Myxomycetes*	Blue or tan-colored spore-like structures on leaves.	All cool-season turfgrasses.
Snow mold (grey)	*Typhula incarnata*	Large patches of matted turf appearing at snow melt. Gray mycelium and orange resting structures often present on affected foliage.	All cool-season turfgrasses.
Snow mold (pink)	*Microdochium nivale*	Small patches of matted turf with pink or reddish color on the leaves.	All cool-season turfgrasses.
Stripe smut	*Ustilago striiformis*	Black streaks of spores along length of leaf blades. Shredding of leaf blades.	Kentucky bluegrass and bentgrass.
Summer patch	*Magnaporthe poae*	Large yellow or tan ring-shaped patches. A root and crown rot is usually apparent.	Bluegrass and fine fescues.
Take-all patch	*Gaeumannomyces graminis*	Patches of dead or dying turf ranging from a few inches to several feet in diameter.	Bentgrasses.

Source: Dr. Landschoot

TABLE 2

Generic & Trade Names of Common Turfgrass Fungicides

Generic Names	Contact (C) or Systemic (S)	Common Trade Names[1]
Anilazine	C	Dyrene
Benomyl	S	Tersan 1991, Lesco Benomyl, Lebanon Benomyl
Chloroneb	C	Tersan SP, Teremec SP, Proturf Fungicide II
Chlorothalonil	C	Daconil 2787
Ethazol (etridiazole)	C	Koban, Terrazole
Fenarimol	S	Rubigan
Fosetyl-Al	S	Aliette
Iprodione	S	Chipco 26019, Proturf Fungicide VI
Mancozeb	C	Fore, Formec, Dithane F-45, Lesco Mancozeb, Manzate 200 DF
Maneb	C	Dithane M-22
Maneb + zinc sulfate	C + C	Tersan LSR, Dithane M-22 w/Zinc, Lesco 4 F w/Zinc
Mercury chloride	C	Calo-Clor, Calo-Gran
Metalaxyl	S	Subdue, Proturf Pythium Control
Metalaxyl + mancozeb	S + C	Pace
Pentachloronitrobenzene (quintozene)	C	Terraclor, Turfcide, Proturf FF II, Lesco PCNB
Phenylmercuric acetate	C	PMAS
Phenylmercuric acetate + thiram	C + C	Proturf Broad Spectrum Fungicide
Propamocarb	S	Banol
Propiconazole	S	Banner
Thiophanate-ethyl + thiram	S + C	Bromosan
Thiophanate-methyl	S	Fungo 50, Spot-Kleen, Clearys 3336, Topsin M, Proturf Systemic Flugicide
Thiophanate-methyl + mancozeb	S + C	Duosan
Thiophanate-methyl + iprodione	S + C	Proturf Fluid Fungicide
Thiram	C	Tersan 75, Spotrete, Thiramad, Lesco Thiram
Triadimefon	S	Bayleton, Proturf Fungicide VII, Lebanon Turf Fungicide
Triadimefol + metalaxyl	S + S	Proturf Fluid Fungicide II
Triadimefon + thiram	S + C	Proturf Fluid Fungicide III
Vinclozolin	S	Vorlan

[1] Products may be available only through specialized dealers or only in large quantity. Some products can be purchased and applied only by licensed pesticide applicators. This list is presented for information only. No endorsement is intended for products mentioned, or is criticism meant for products not mentioned.

Source: Dr. Landschoot

Biologicals: the new frontier

Biological control is the reduction of disease-producing activities of a pathogen by another organism.

Biological control is a natural occurrence in turf and is a primary reason why diseases do not destroy all of our lawns, grounds and golf courses.

Organisms that limit the disease-producing activities of a pathogen are referred to as antagonists. Antagonists are usually microorganisms (fungi, bacteria, viruses, nematodes, or actinomycetes) that interfere with the growth and spread of the pathogen. Antagonists may be introduced by artificial means or they may already be present in the turfgrass ecosystem.

Antagonists produce compounds that inhibit the pathogen—antibiotics, for example—or more directly, parasitize the pathogen. The direct application of antagonists is likely to result in failure unless provisions are made for it to successfully compete in turf. Direct application of an antagonist that is not adapted to the turf ecosystem is like sending a soldier into battle without a rifle. The pathogen and the other resident microorganisms are usually well equipped to outcompete and fend-off the introduced antagonist.

Another method of biological control that has yielded success with some turf pathogens is the use of pathogen-suppressive soils. Suppressive soils are those in which the pathogen does not establish or persist in populations great enough to cause severe disease damage. Suppressive soils have been implicated as a factor responsible for the absence or decline of take-all patch of bentgrass turf. Take-all patch usually develops on recently-sterilized soils or on golf courses that were formerly woodland or wetland sites and do not have large populations of resident antagonists.

Over three to five years, the disease begins to disappear from these sites, a phenomenon known as "take-all decline." Studies have shown that the transfer of a small amount of soil from sites where take-all decline has occurred to areas in which the disease is active, resulted in suppression of the disease. Studies in Australia have revealed that suppressive soils can be developed in the laboratory and used as a top dressing to control take-all patch. Suppressive soils have also been reported for other pathogens including various species of fusarium, pythium, and rhizoctonia. To my knowledge, there are no companies

that are marketing pathogen-suppressive soils for use on turf.

continued on page 44

TABLE 3

Methods of Disease Control for Cool-season Turfgrasses

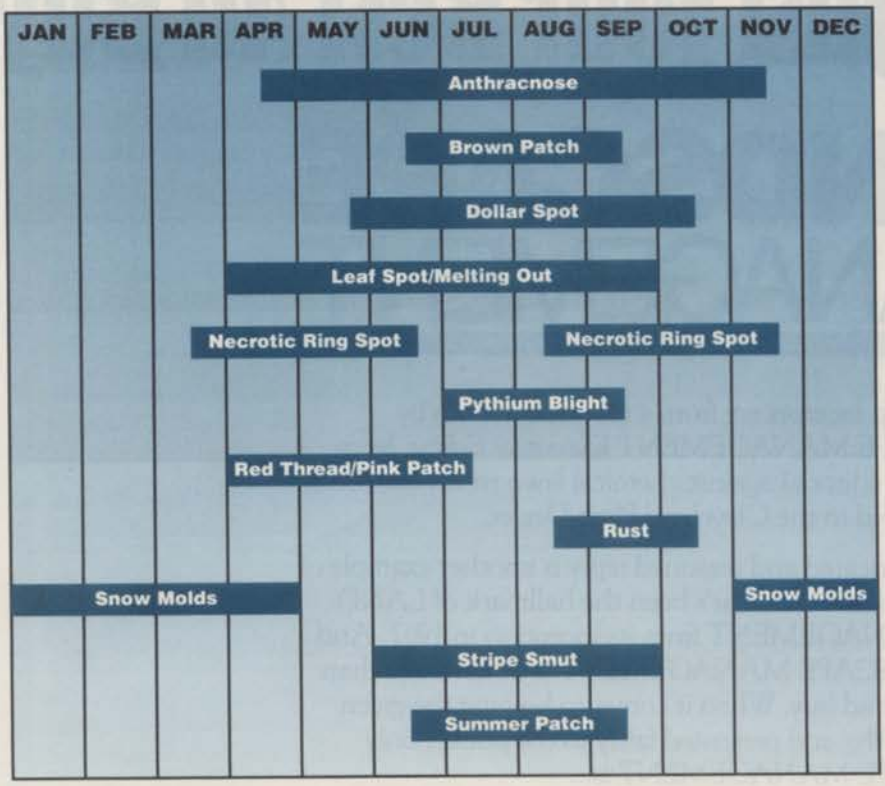
Disease	Cultural control	Chemical control	Resistant species/varieties
Anthracnose	Fertilize to maintain vigor, improve drainage, aeration, and raise mowing height during periods of heat stress.	benomyl, chlorothalonil, mancozeb, propiconazole, triadimefon	Bentgrasses are less susceptible than annual bluegrass on putting greens.
Brown patch	Avoid excess N in summer, increase air circulation, avoid excessive watering, improve drainage.	anilazine, benomyl, chlorothalonil, iprodione, mancozeb, maneb, thiophanates, thiram, vinclozolin	Kentucky bluegrasses are less susceptible to brown patch than other cool-season turfgrasses.
Dollar spot	Avoid N deficiency, maintain good soil moisture, remove guttation and dew from leaf surfaces, avoid night watering.	anilazine, benomyl*, chlorothalonil, fenarimol, iprodione, mancozeb, propiconazole, thiophanates, thiram, vinclozolin	Resistant Kentucky bluegrass varieties include Adelphi, America, Aspen, Challenger, Eclipse, Escort, Nassau, Somerset, & Wabash.**
Fairy ring	Use clean fill during establishment, irrigate, or mask symptoms with N-fertilizer or iron.	None effective, must fumigate with soil sterilant to eradicate the fungus (this will also kill grass).	No resistant species or varieties are available.
Leaf spot/ melting out	Avoid excess N applications in early spring, mow 2" in height, avoid light, frequent irrigation. Do not use benomyl, thiophanates, or triadimefon to control this disease.	anilazine, chlorothalonil, iprodione, maneb, mancozeb, vinclozolin	Resistant Kentucky bluegrasses include: Adelphi, Bristol, Destiny, Eclipse, Enmundi, Glade, Ikone, Leberty, Majestic, Mona, P-104, Rugby, and Somerset.**
Necrotic ring spot	Manage turf for maximum root growth, irrigate to maintain good soil moisture, maintain mowing height at 2" or above.	benomyl, fenarimol, propiconazole	Perennial ryegrasses are resistant.
Powdery mildew	Reduce shade and improve air circulation.	benomyl, fenarimol, propiconazole, triadimefon (fungicides usually not required)	Use grasses adapted to shaded areas such as fine fescues and rough bluegrass. Resistant Kentucky bluegrasses include: A-34, Glade, Touchdown, & Eclipse.**
Pythium blight	Improve drainage, increase air circulation, avoid excess N, reduce irrigation.	chloroneb, etridiazole, metalaxyl*, Fosetyl-Al, propamocarb	Kentucky bluegrass is less likely to be damaged by Pythium blight than other turfgrasses.
Pythium root rot	Increase drainage, aerate	Fosetyl-Al, Subdue as a drench	unknown
Red thread/ pink patch	Maintain adequate fertility of turf (especially N)	alilazine, benomyl***, chlorothalonil, iprodione, propiconazole, thiophanates***, triadimefon, vinclozolin	Resistant perennial ryegrasses include: Allaire, Commander, Delray, Manhatten II, Palmer, Pennant, Prelude, Regal, Regency, SR 4000, SR 4100, and Yorktown II.**
Rust diseases	Avoid N-deficiency and drought-stress (especially in late summer/early fall)	maneb, mancozeb, fenarimol, propiconazole, triadimefon. (fungicides usually not required)	Some resistant Kentucky bluegrasses include: Kenblue, Parade, Rugby, A-34, and Classic**.
Slime molds	Remove spores by spraying water on leaves or brushing turf.	None required.	Not applicable since grasses are not infected.
Snow molds: Gray snow mold	Avoid excess N in fall before grass goes dormant, mow until top growth ceases in fall, prevent accumulation of snow in sensitive areas, rake up mats (patches) in spring to speed recovery.	Fungicides should be applied in late fall before snow cover: chloroneb, fenarimol, iprodione, mercury fungicides,. PCNB, thiophanates, thiram, triadimefon, vinclozolin	Some resistant Kentucky bluegrasses include: Adelphi, Aspen, Enmundi, Plush, and Vantage**.
Pink snow mold	(Same as for gray smow mold)	benomyl, fenarimol, iprodione, mancozeb, mercury fungicides, PCNB, thiophanates, thiram, vinclozolin	Most fine fescues and Kentucky bluegrasses are moderately resistant to this disease.
Stripe smut	Avoid excess N in early spring, avoid drought stress in early summer.	Apply fungicides in early spring or late fall, water-in for good root uptake. Benomyl, fenarimol, propiconazole, thiophanates, triadimefon	Ryegrasses, tall fescues, and the fine fescues are less susceptible to this desease than Kentucky bluegrass.
Summer patch	Avoid low mowing heights, reduce compaction, avoid overwatering in summer, improve drainage.	benomyl, fenarimol, propiconazole, thiophanates, triadimefon	Resistant Kentucky bluegrasses include Adelphi, Enmundi, Sydsport, and Touchdown.
Take-all patch	Use acidifying fertilizers or sulfur to lower pH, avoid P and K deficiency.	Sterol biosynthesis inhibitors may have some benefit if applied prior to root infection.	annual bluegrass

* Resistance has been recorded.
** Based on National Turfgrass Evaluation Program and Penn State data. No endorsement of cultivars is intended for those mentioned, or is criticism meant for cultivars not mentioned.
*** Controls red thread and not pink patch.

Source: Dr. Landschoot

CALENDAR

COMMON DISEASES OF COOL-SEASON TURF



Biologicals:

from page 40

Organics an alternative?

Most of the current interest in biological control of turf pathogens is centered around the use of natural organic fertilizers or organic soil amendments. This practice has been successfully employed with other crops and some success has been achieved in controlling turfgrass diseases. Research at Cornell University has shown that some organic amendments suppressed dollar spot and brown patch diseases when applied as a topdressing to a bentgrass putting green.

Similar results have been obtained by researchers from Michigan State University and The University of Rhode Island for the suppression of necrotic ring spot. Although we do not understand the exact mechanisms involved, there is some evidence to suggest that these products stimulate populations of resident antagonists to levels that will suppress some turf diseases. They may also aid in disease control by providing additional nitrogen to the plant. **LM**

—Dr. Landschoot

Phythium blight is culturally managed by improved drainage, reduced irrigation and a moderate nitrogen diet. Chemical control includes chloroneb, ethazol and metalaxyl.

DISEASE CONTROL IN WARM-SEASON TURF

Integrated disease management first requires the selection of appropriate turfgrass species and cultivars. Proper cultural practices, pesticides, and biological control components follow.

by Monica L. Elliott, Ph.D., University of Florida

Although we can easily divide turfgrasses into cool- and warm-season types, it is people who determine the grass grown in any particular landscape. But people do not necessarily follow the rules. The result is that we find bentgrass grown in Florida and bermudagrass grown in the central U.S.

To confuse the issue even further, we have bermudagrass putting greens over-seeded with ryegrass or bentgrass during the winter months in the southern states. The primary goal is to have attractive turfgrass areas; diseases, however, can quickly blemish this picturesque setting.

Knowing the difference

While turfgrass injuries or disorders may look like diseases, they are not diseases and should not be treated as such.

A disease is primarily an interaction between the plant and a pathogen. It consists of three components: turfgrass host, pathogen and the environment in which the host and pathogen interact. In most turfgrass situations, the environment is the key factor in disease development, since the other two components are virtually always present.

While turfgrasses may be affected by diseases all year long, individual turf diseases are prominent for only a few months each year, based normally on weather patterns/environmental effects. Since it is usually not practical to eliminate the turfgrass host, disease control recommendations are aimed at (1) suppressing the pathogen, and (2) altering the environment so it is less favorable for disease development.

Turfgrass selection

Select turfgrass species and cultivars based on your geographical location

TABLE 1

Diagnostic Features of Common Warm-Season Turfgrass Diseases

Disease	Casual agent(s)	Symptoms/signs	Susceptible grasses
Anthracnose	*Colletotrichum graminicola*	Brown lesion with yellow halo expands to cause yellowing of entire leaf blade. Tiller infection results in stem girdling. Fruiting bodies are dark cushion-like bodies with small black spines and can be seen with hand lens.	bahiagrass, bermudagrass and centipedegrass*
Bermudagrass decline	*Gaeumannomyces* spp. or similiar fungi as yet undetermined.	Begins as small, irregular, yellow patches which expand and thin-out as grass dies. Roots are short, thin and rotted. Usually observed first at edges of putting greens.	bermudagrass*
Brown patch (Rhizoctonia blight); Rhizoctonia leaf and sheath spot	*Rhizoctonia solani, R. zeae, R. oryzae*	Begin as small circular light green patches that turn yellow and then brown or straw-colored. Patches expand to several feet in diameter. Turf at outer margin of patch may appear dark and wilted (smoke ring). Whole leaf facsimiles pull up easily due to basal rot of leaf sheath. Aerial blight common with centipedegrass and St. Augustinegrass. During warm summer months, may also observe distinct light brown foliar lesions.	Bahiagrass, bermudagrass, centipedegrass*, St. Augustinegrass*, and zoysiagrass*
Dollar spot	*Lanzia* spp. and *Moellerodiscus* spp. *(Sclerotinia homoeocarpa)*	Small, bleached patches of dead grass that do not expand but do coalesce with other spots to form large patches. Irregular, light tan lesions with distinct brown borders. White, cottony mycelium may be observed in early morning when dew is present.	bahiagrass*, bermudagrass*, centipedegrass, St. Augustinegrass, zoysiagrass*
Cercospora leaf spot	*Cercospora fusimaculans*	Small, dark brown or purple lesions on leaf blade and sheath that become tan color with age. High disease severity results in leaf death and turf areas that thin-out.	St. Augustinegrass*. Bitter-blue cultivars are less susceptible than yellow-green cultivars.
Fairy ring	*Chlorophyllum, Marasmius, Lepiota* and other basidiomycete fungi	Circular to semi-circular bands of dark green turf with or without mushrooms present in band. Some rings are bands of dead turf. Rings expand each year.	All warm-season turfgrasses.
Gray leaf spot	*Pyricularia grisea*	Lesions begin as small, brown spots that expand into oval areas with tan centers and dark purple or brown margins. Yellow halo may be present. During warm humid weather, lesions covered with gray velvet mat of mycelium. Leaves wither resulting in scorched appearance.	St. Augustinegrass*. Yellow-green cultivars are less susceptible than blue-green/bitter-blue cultivars, St. Augustinegrass treated with the herbicide atrazine is more susceptible.
"Helminthosporium" Leaf Spot/ Melting Out	*Bipolaris, Drechslera* and *Exserohilum* spp. (previously *Helminthosporium* fungi); and *Curvularia* spp.	Leaf spot symptoms vary with specific pathogen and host from small, solid brown to purple lesions to expanded lesions with bleached centers that girdle the leaf blade. Severly infected leaves turn reddish-brown to straw color. "Melting-out" occurs under severe infection as turfareas thin and die. Lesions on stems are dark purple to black. Crown/root rots will also occur.	Bermudagrass*, St. Augustinegrass, zoysiagrass
Pythium Blight	*Pythium* spp.	Small, distinct patches of grass that first appear dark and water-soaked but later turn straw-color. No distinct leaf lesions. Patches spread quickly in "streak" pattern. Cottony mycelium may be observed in early morning when dew present.	All warm-season turfgrasses, primarily Bermudagrass*
Pythium Root Rot	*Pythium* spp.	General turf browning and thinning. Roots appear thin with few root hairs and have a general discoloration. Turf does not repond to N applications.	All warm-season turfgrasses.
Rust	*Puccinia* spp.	Orange to reddish brown pustules on leaves. Severe infections cause yellowing of leaves and thin turf.	Bermudagrass, St. Augustinegrass and Zoysiagrass*
Spring dead spot	*Leptosphaeria korrae, Ophiosphaerella herpotricha* and *Gaeumannomyces graminis* var. *graminis*.	Large, circular patches of bleached, dead grass that appear as dormant turf resumes growth in spring. Root, crown and stolon rot evident.	bermudagrass*
St. Augustinegrass decline	*St. Augustinegrass decline virus (Panicum mosaic virus)*	Initially, observe chlorotic (yellow) mosaic or mottle on leaf blades that gradually becomes more extensive until area appears uniformly yellow and thin. Death may eventually occur.	centipedegrass and St. Augustinegrass* (some cultivars).

*** denotes most susceptible turfgrass species**

Source: Dr. Elliott

and on how the turf will be used and maintained.

Grasses that are not suited for a particular area will be continually stressed, more susceptible to disease, and require increased maintenance costs in terms of labor and pesticides. An area subjected to heavy foot traffic would not be suitable for centipedegrass. Non-irrigated areas would be satisfactory for bahaigrass but not St. Augustinegrass.

Sometimes turfgrass is not even the appropriate plant for a particular landscape. For example, most warm-season turfgrasses do not thrive in heavily shaded areas. Certain cultivars of some turf species are resistant to specific diseases. Selecting disease-resistant cultivars is especially important for controlling viral diseases, in part because there are no chemicals to control these diseases.

The primary viral disease associated with warm-season turfgrasses is caused by the St. Augustinegrass decline virus. It is most frequently observed in centipedegrass and susceptible St. Augustinegrass cultivars. It is normally a mild pathogen of centipedegrass of which there are no resistant cultivars.

However, a number of St. Augustinegrass cultivars are resistant to this virus, including Floratam, the most popular cultivar. Therefore, before you plant a single seed or blade of grass, consult with your local experts to determine the most appropriate turfgrass to plant and then make sure the correct material is installed.

Cutting height matters

Cultural practices should promote an environment that is not conducive for pathogen infection and disease development.

If a disease should affect the turfgrass, these practices should be implemented first or, at the very least, implemented at the same time fungicides are applied.

Mowing is the most common turf maintenance operation. Every time a mower removes leaf tissue, a wound is created through which a pathogen may enter the plant. However, turfgrasses that are cut below their optimum height will be stressed and more susceptible to some diseases.

Always use a sharp mower blade. Turf with active disease areas should be mowed last, as mowers may actually spread the pathogen from one location to another. Likewise, clean the equipment between jobs. A thorough rinse with water is sufficient to remove clippings and debris which may carry plant pathogens.

Not too short on greens

Raise the blade height on golf course putting greens with active disease areas. Over the past few years, the height of cut on greens has been reduced substantially; 3/16 inch or lower is the standard on bermudagrass putting greens. The low height of cut reduces the tissue necessary for photosynthesis, the process by which the plant produces energy.

In addition, diseases eventually reduce the leaf canopy and photosynthesis is reduced even further.

It has often been suggested that leaf clippings should be collected when a leaf disease is active. Clippings disposal is no longer ecologically acceptable and will become illegal in some states in the near future. In general, do not collect leaf clippings unless you have an acceptable method for recycling the material.

A properly constructed compost will kill the pathogen, so you will not infect a turfgrass area by using this composted material in the landscape. In addition, recent studies suggest that using a mulching lawnmower blade with a closed mower deck may help to limit leaf diseases when it is necessary to return clippings to a turf area.

Managing water

Most fungal pathogens require free water or very high humidity to start the infection process. Dew (more importantly, the length of the dew period), which depends on temperature and humidity, is a critical factor. Extending the length of the dew (free water) period by irrigating in the evening before dew forms or in the morning after the dew evaporates extends the dew period. Therefore, irrigate when dew is already present, usually in the pre-dawn hours.

When you do irrigate, apply enough water each time to adequately soak the entire root system. Irrigate to the depth of the roots, but not below them. Shallow irrigations will require you to irrigate more frequently and thus increase the chances for pathogen infection and pathogen movement.

Importance of nitrogen

Many diseases are also influenced by the nutritional status of the grass, especially nitrogen. Both excessively high and low nitrogen fertility contributes to turfgrass diseases. Higher nitrogen applications encourage rhizoctonia diseases, gray leaf spot, helminthosporium leaf spot and pythium blight. Lower nitrogen levels encourage dollar spot, rusts and anthracnose.

Remember: it is easier to add nitrogen to the soil but impossible to remove it. If a foliar disease is active, select a fertilizer blend with a high percentage of the slow-release component and a low percentage of the rapid-release component. This will allow you to "feed" the turf without "feeding" the pathogen.

Note that no single environmental factor influences diease development. One example is centipede decline. Although no specific pathogen has been documented as the causal agent, we do know that excessive fertilization and irrigation contribute to the decline. Dollar spot is another example. Three factors encourage this disease: nitrogen deficiency, dry soils, and high moisture levels surrounding the leaves. Frequent, short irrigation periods in addition to dew periods lead to the dry soils and high leaf humidity. Although fungicides are available for controlling this disease, correcting the three diseases development factors, especially the plant's nitrogen status, will achieve the same goal. Physical and chemical soil properties may not affect disease development directly, but they do affect turfgrass health.

Maintain pH levels

Soil pH is an important growth factor. For example, centipedegrass and

Biological control testing continues

Biological control of turfgrass diseases is a new area of disease management that is still in the experimental research phase for warm-season turfgrasses.

Testing is currently in progress concerning the use of non-pathogenic fungi and bacteria for control of turfgrass diseases. However, the most active area of research involves the use of organic fertilizers for disease suppression. These products are thought to stimulate the development of microorganisms which antagonize turfgrass pathogens. In both cases, further testing is required to substantiate their value in the consistent control of turfgrass diseases and the proper methods for their use. When that is accomplished, biological control will be routinely incorporated into an integrated turfgrass management program.

—*Dr. Elliott*□

TABLE 2

Methods of Disease Control for Warm-season Turfgrasses

Disease	Cultural control	Chemical control	Resistant species/varieties
Anthraconse	Avoid fertility imbalances, improve drainage and remove excessive thatch. Stress due to insects or nematodes should be eliminated.	benomyl, chlorothalonil, fenarimol, mancozeb, propiconazole, thiophanate methyl, triadimefon	No resistant centipedegrass cultivars are available.
Bermudagrass decline	Aerate and topdress greens monthly during late spring, summer and early fall. Apply NH4-N rather than NO3-N. Balance N with K and apply micronutrients. Raise mowing height during disease outbreaks.	benomyl, fenarimol, propiconazole, thiophanate methyl, triadimefon	No resistant cultivars are available.
Brown patch	Avoid excess N, especially readily available forms of N. Avoid excessive irrigation.	anilazine, benomyl, chlorothalonil, iprodione, maneb, mancozeb, PCNB, thiophanate methyl, thiram. Do NOT use benomyl or thiophanate methyl if causal agent is R. oryzae or R. zeae. Use fenarimol, propiconazole and triadimefon as preventative and not curative compounds.	No resistant species are available.
Cercospora leaf spot	Avoid N deficiency. Irrigate deeply and less frequently.	None are currently labeled. Contact fungicides such as chlorothalonil, iprodione or mancozeb may provide disease suppression.	Bitter-blue selections of St. Augustinegrass are less susceptible.
Dollar spot	Avoid N deficiency. Irrigate deeply and less frequently.	anilazine, benomyl, chlorothalonil, fenarimol, iprodione, mancozeb, maneb, propiconazole, thiophanate methyl, thiram, triadimefon, vinclozolin	No resistant species are available.
Fairy ring	Mask symptoms with N fertilizers. Remove mushrooms as some are poisonous. Before planting, eliminate large sources of organic matter such as tree stumps, wood building materials, etc.	None are currently registered. To eliminate fungus, fumigate with soil sterilant and replant.	No resistant species are available.
Gray leaf spot	Avoid excess N. Irrigate deeply and only when necessary.	chlorothalonil, propiconazole, thiophanate methyl + mancozeb	Yellow-green cultivars of St. Augustinegrass are less susceptible. St. Augustinegrass treated with the herbicide atrazine is more susceptible.
"Helminthosporium" leaf spot/ melting out	Avoid excess N. Balance fertility components. Irrigate deeply and less frequently. Avoid thatch accumulation. Raise mowing height during disease outbreaks.	anilazine, chlorothalonil, iprodione, maneb, mancozeb, propiconazole, vinclozolin	No resistant species are available.
Pythium blight	Improve drainage and air circulation. Reduce irrigation. Avoid excess N.	chloroneb, ethazol, metalaxyl, fosetyl-Al, propamocarb	No resistant species are available.
Pythium root rot	Improve drainage, aerate and reduce irrigation.	chloroneb, ethazol, metalaxyl, fosetyl-Al, propamocarb. Except for fosetyl-Al, these fungicides should be watered into the root zone.	No resistant species are available.
Rust	Avoid N deficiency. Irrigate deeply and less frequently.	anilazine, maneb, mancozeb, propiconazole, triadimefon	No resistant species are available.
Spring dead spot	Avoid low mowing heights, thatch, compaction and excess N.	benomyl, fenarimol, propiconazole, thiophanate methyl	No resistant species of bermudagrass are available.
St. Augustinegrass decline	Do not plant susceptible cultivars.	None. Disease is caused by a virus.	Resistant St. Augustinegrass cultivars are Floratam, Floralawn, Raleigh and Seville.

Source: The author

Gray leaf spot as shown on St. Augustinegrass. Yellow-green cultivars are less susceptible. St. Augustinegrass treated with the herbicide atrazine is more susceptible.

bahiagrass have an optimum soil pH between 5 and 6 whereas zoysiagrass and St. Augustinegrass prefer soil with pH between 6 and 7. A soil pH greatly above or below these optimum values results in turfgrass that is constantly stressed and susceptible to turfgrass pathogens. Soils that are compacted and poorly drained result in stressed turf also, especially of the root system.

Install drainage and aerify regularly to help reduce disease development.

Thatch is a natural component of a turfgrass ecosystem. However, excessive thatch accumulation indicates an imbalance has occurred and plant tissue is being produced more quickly than it is being decomposed. Factors that impede microbial decomposition are excessively wet or dry conditions, very high or low thatch pH, inadequate nitrogen levels and repeated use of chemical pesticides. Thatch accumulation is probably most severe with zoysiagrass and does require periodic renovations.

Chemical control

Except for St. Augustinegrass decline, diseases of warm-season turfgrasses are caused by fungi. Chemical control of these diseases is accomplished by using fungicides. However, chemical controls are all too often implemented without considering cultural controls or understanding the reasons behind disease development.

The next time you spray a fungicide, determine what else you can do to prevent or control disease development. We cannot afford to apply fungicides, or any pesticide, without understanding the reasons for the applications. We must explain our actions intelligently to the public—and emphasize the other actions we have taken to reduce a particular pest.

Fungicides *do not* eliminate the pathogens from the turfgrass area. They primarily suppress fungal pathogen growth to prevent them from infecting the plant when the environment is conducive for disease development.

Contacts and systemics

Turfgrass fungicides can be divided into two broad categories based on the location of their activity: (1) contact fungicides and (2) systemic fungicides which include true systemic compounds and local-systemic compounds.

Contact fungicides, generally applied to the leaf and stem surfaces of turfgrasses, act as protective compounds. They should evenly coat the entire leaf surface. These fungicides remain on the plant surface. They remain active only as long as the fungicide remains on the plant in sufficient concentration to inhibit fungi. Leaves which emerge after the fungicide has been applied will not be protected. In addition, fungicide on the plant surface will be gradually lost due to mowing, irrigation, rainfall and decomposition.

Consequently, they are only effective for short durations, usually 7 to 14 days. To obtain optimum protection, contact fungicides should evenly coat the entire leaf surface and be allowed to dry completely before irrigating or mowing. Ideally, the turf area should be mowed and irrigated prior to a fungicide application to allow a maximum time interval between fungicide application and the next turfgrass maintenance operation.

In general, systemic fungicides have curative and protective activities with extended residual activity. Because systemic fungicides are absorbed by the plants, they "work" inside the plant to, (1) control pathogenic fungi which have already entered the plant and initiated a disease (curative action), and, (2) inhibit fungi that enter the plant from initiating a disease (preventive action).

Inside the plant, a systemic fungicide will not be removed by rain or irrigation, and newly emerged leaves may containsufficient concentrations of the fungicide to protect them from fungal infection.

Systemic fungicides do not need to be applied as often as contact fungicides; usually 15- to 30-day intervals are adequate. Systemic fungicides usually have a very specific mode of action and do not have as broad a spectrum of disease control as contact fungicides. However, they will control both foliar and root/crown pathogens. When attempting to control the latter, systemic fungicide should be watered into the rootzone immediately after application, since the majority of systemic fungicides are xylem-limited, i.e., they move in an upward direction in the plant. If these fungicides are only applied to the leaf tissue, the compounds may never reach their root target in the amount needed for control.

Local-systemic fungicides are capable of penetrating the plant surface, but only move very short distances within the plant and usually not within the xylem or phloem tissue. The majority of fungicide remainson or near the plant surface. Included in this group of fungicides are iprodione and vinclozolin. These fungicides are primarily protective in activity wheras the true systemic compounds have both curative and protective activities.

Preventing resistance

Fungicides are grouped according to their chemical properties.To prevent fungicide resistance from developing in a pathogen population, it is important to know which fungicides belong to the same chemical group or have the same mode of action. Fungicides should be periodically alternated or used in mixtures with fungicides belonging to different chemical groups to prevent fungicide resistance.

Trade names are not an indication of the chemical group. For example, alternating between Tersan 1991 (benomyl) and Fungo 50 (thiophanate methyl) does not mean you have alternated between chemical groups, as both fungicides belong to the same chemical group.

If you do not achieve disease control with a fungicide, make sure the disease was properly diagnosed and the fungicide properly applied before assuming that a fungicide resistant strain has developed.

The number of documented cases of fungicide resistance is limited for warm-season turfgrasses. Turfgrass managers can keep it that way by exercising intelligent, prudent use of fungicides. **LM**

Monica Elliott is an assistant professor of turfgrass pathology at the University of Florida in Ft. Lauderdale.