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**

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