

# Fairy Ring

[*Basidiomycetes*]

## SYMPTOMS

The symptoms of fairy ring appear in patches, rings, or arcs that are initially 1 foot or less in diameter, but expand in size year after year, reaching up to several hundred feet in diameter in old turf stands. Most fairy ring fungi do not infect or parasitize the turf. Instead, growth of these fungi in the soil can indirectly affect, or even kill, the turfgrass above. Three types of fairy ring symptoms are observed in turfgrasses: Type I, Type II, and Type III. A Type I fairy ring causes the soil and thatch to become hydrophobic, killing the turf in patches, rings, or arcs. In areas affected by a Type I fairy ring, the thatch and soil are extremely dry and repel water. Type II fairy rings appear as rings or arcs of turf that are dark green and growing more quickly than the surrounding turf. In a Type III fairy ring, mushrooms or puffballs are produced in a ring or arc. The type of symptom expressed by a particular fairy ring may change during the year according to weather conditions. Type III fairy ring symptoms are more prevalent during extended periods of wet weather. Type I and Type II fairy ring symptoms are most common during hot, dry weather in the summer.



fairy ring puffballs

Characteristic	Description
Host Grass Species	all
Month(s) with symptoms	all
Stand Symptoms	patches (1 to 3 feet), rings
Foliar Symptoms - Location/Shape	dieback from leaf tip, no distinct leaf symptoms
Foliar Symptoms - Color	tan, yellow, orange
Root/Crown Symptoms	none
Fungal Signs	mushrooms, puffballs, or none

**Note:** Still not sure if this is the right disease? The [Turfgrass Disease Identification](#) program may be helpful. Or consult the experts at the [Turf Diagnostics Lab](#). Check the TurfFiles [glossary](#) for definitions of unfamiliar terms.

## FACTORS AFFECTING DISEASE DEVELOPMENT

Some of the fungi that cause fairy rings are wood-rotting fungi that grow on stumps, dead tree roots, waste lumber or other woody materials. Once established, the turf produces thatch and organic matter, which provides a source of food for continued expansion of the fairy ring. In golf course putting greens and other sandy soils, most fairy rings are caused by puffball fungi, which do not grow on woody materials but instead thrive in the thatch that is produced by turf. On golf course putting greens, fairy rings are often observed spreading into the root zone mix from the surrounding native soil. Mushroom fungi are also prolific spore producers and may be spread into putting greens by wind, water, or other means..

Type I fairy rings are most damaging to turf. Most cases of Type I fairy ring are caused by hydrophobic (water-repellent) residues that are produced as the fairy ring fungus grows through the soil. In other cases, ammonium nitrogen that is released into the soil by fairy ring fungi may accumulate in the soil to

toxic levels. Either way, the expression of Type I symptoms can be further encouraged by drought stress, inadequate irrigation, and infrequent aerification.

Type II fairy ring symptoms are a result of the release of nitrogen and other nutrients into the soil as organic matter is degraded by the fairy ring fungi. These symptoms are most evident in turf that is deficient of nutrients, especially nitrogen and iron.

Type III fairy ring symptoms are most common after periods of heavy or frequent rainfall. They may occur more frequently in areas that are poorly drained or over-irrigated.

## **CULTURAL CONTROL**

In landscape turfgrasses, the most effective means for fairy ring control is to prevent the causal fungi from becoming established in the turf. Remove large pieces of woody material (stumps, waste lumber, and dead tree roots) before turf is planted to prevent the establishment of fairy rings. Landscape contractors should remove this debris around new construction sites before seeding or sodding the turf.

The source of fairy ring infestations on golf course putting greens is unclear. Sterilization or fumigation of the root zone mix has not been effective in preventing or delaying fairy ring establishment. Installation of a plastic barrier between the root zone mix and surrounding native soil may help to limit the spread of fairy ring into golf course putting greens.

Power raking or vertical mowing to remove excessive thatch will help to minimize fairy ring problems. Golf course superintendents should regularly aerify and topdress putting greens to prevent thatch buildup and maintain soil aeration. Avoid extremes in soil moisture (too wet, too dry), apply nitrogen based on local University recommendations, and ensure balanced fertility through regular soil testing.

Once a fairy ring appears, the best management practices depend on the type of symptom that is observed. To control a Type I fairy ring, the water-repellent thatch and soil beneath the affected turf must be re-wet. Hollow-tine aerification, spiking, water-injection, application of soil surfactants, and heavy irrigation are effective strategies for re-wetting this hydrophobic layer. Affected areas should be hand-watered to prevent over-watering of the surrounding, unaffected turf.

Symptoms of a Type II fairy ring can be masked with an application of nitrogen or iron. This will cause the surrounding turf to green-up, making the affected turf less evident. Collect soil or tissue samples for nutrient analysis from the turf immediately surrounding the Type II fairy rings, and correct any nutrient imbalances as recommended. Use caution when applying nitrogen to mask Type II fairy ring symptoms on cool-season grasses during the summer. Too much nitrogen may over-stimulate the grass and lead to the development of more serious diseases. In this case, iron should be used to increase turf color without causing excessive foliar growth.

Drastic methods for control of fairy rings, such as soil fumigation, removal of infested soil, or turf renovation by tilling and mixing the soil may be effective in the short-term, but the fairy rings usually become re-established over a period of years.

## **CHEMICAL CONTROL**

Over 60 species of fungi have been associated with fairy ring symptoms in turfgrasses, and these species likely vary in their sensitivity to fungicides. Control of fairy rings with fungicides is a site-specific venture for this reason. Turfgrass managers should experiment with different products to identify those that will control the disease in their location.

Fungicides are most effective for fairy ring control when used on a preventative basis. Curative applications have little effect because the symptoms are caused by a change in the soil environment, and fungicides do nothing to change the soil. A preventative fungicide program should be initiated in the

spring when mean daily soil temperatures are consistently above 55°F. Regular use of soil surfactants will help to maintain uniform soil moisture and may reduce the appearance of Type I fairy ring symptoms.

Because fairy ring fungi are in the thatch and soil, fungicides must be watered-in or applied in large volumes of water for best results. Applications in 2 gallons H<sub>2</sub>O per 1000 ft<sup>2</sup> followed by 0.25" of irrigation have provided excellent results in research trials. Irrigation must be applied immediately before the spray begins to dry on the turfgrass foliage. Tank-mixing some fungicides with a soil penetrant may also enhance movement into the soil and improve fairy ring control.

The DMI fungicides provide excellent preventative control of puffball fungi, such as *Lycoperdon*, *Vascellum*, *Bovista*, or *Arachnion*, in golf course putting greens. Two applications on a 30 day interval, beginning in spring when 5-day average soil temperatures reach 55°F have provided season-long control in many cases. The DMI fungicides, however, should not be tank-mixed with soil surfactants as this has been found to reduce their efficacy and increase the potential for phytotoxicity.

Fungicide	Efficacy <sup>(1)</sup>	Resistance Risk <sup>(2)</sup>	Class <sup>(3)</sup>	Products <sup>(4)</sup>
tebuconazole**	++++	2	DMI	Torque
triadimefon	++++	2	DMI	Bayleton, Granular Turf Fungicide, Systemic Fungicide
fluoxastrobin + myclobutanil	++++	3	DMI + Qol	Disarm M
triadimefon + trifloxystrobin	++++	2	DMI + Qol	Armada, Tartan
pyraclostrobin + boscalid**	+++	3	carboxamide + Qol	Honor
metconazole	+++	2	DMI	Tourney
propiconazole	+++	2	DMI	Banner MAXX, Kestrel, Kestrel MEX, ProPensity, Propiconazole, Propiconazole G-Pro, Propiconazole Pro, Savvi, Spectator, Strider
triticonazole	+++	2	DMI	Trinity, Triton
azoxystrobin + propiconazole	+++	3	DMI + Qol	Headway
chlorothalonil + fluoxastrobin**	+++	3	nitrile + Qol	Disarm C
azoxystrobin	+++	3	Qol	Heritage
pyraclostrobin	+++	3	Qol	Insignia
flutolanil + thiophanate-methyl	++	2	benzimidazole + carboxamide	SysStar
flutolanil	++	2	carboxamide	ProStar
polyoxin D	++	2	polyoxins	Endorse, Affirm

\*\* Not for application to residential lawns.

**Footnotes:**

(1) **Efficacy Codes:**

- ++++ excellent control when conditions are highly favorable for disease development
- +++ good control when disease pressure is high, or excellent control when disease pressure is moderate
- ++ good control when disease pressure is moderate, excellent control when disease pressure is low

- + good control when disease pressure is low
- 0 does not provide adequate control under any conditions
- ? cannot be rated due to insufficient data

(2) **Resistance Risk:**

- 1 Rotating and tank-mixing not necessary, but recommended to avoid potential side effects from continuous use of same chemical class.
  - 2 Rotate to different chemical class after 3-4 applications; tank-mixing not necessary.
  - 3 Rotate to different chemical class after 2-3 applications; tank-mixing not necessary.
  - 4 Rotate to different chemical class after 1-2 applications; tank-mixing not necessary.
  - 6 Rotate to different chemical class after 1-2 applications; tank-mixing with low or moderate risk product recommended.
  - 9 Rotate to different chemical class after EVERY application; tank-mix with low or moderate risk product for EVERY application.
- (3) Continual use of fungicides with similar control mechanisms (modes of action) can result in fungi that are resistant to some chemicals. Poor or ineffective disease control can be expected when this occurs. Managers can reduce the chances of this happening by mixing or alternating fungicides belonging to different chemical classes.
- (4) Recommendations of specific chemicals are based upon information on the manufacturer's label and performance in a limited number of trials. Because environmental conditions and methods of application may vary widely, performance of the chemical will not always conform to the safety and pest control standards indicated by experimental data. When more than one brand name exists for an agricultural chemical, the name of brand that first came onto the market is listed first. Otherwise, brand names are listed in alphabetical order. The order in which brand names are given is not an indication of a recommendation or criticism.

Recommendations for the use of agricultural chemicals are included in this publication as a convenience to the reader. The use of brand names and any mention or listing of commercial products or services does not imply endorsement by North Carolina State University or discrimination against similar products or services not mentioned. Other brand names may be labeled for use on turfgrasses. Individuals who use agricultural chemicals are responsible for ensuring that the intended use complies with current regulations and conforms to the product label. Be sure to obtain current information about usage regulations and examine a current product label before applying any chemical. For assistance, contact your county's Cooperative Extension agent.

**Useful links:**

Glossary: <http://www.turffiles.ncsu.edu/Glossary.aspx>

Turf Diagnostics Lab: <http://ncstateturfdiagnostics.com/TDL/Home.html>

Turfgrass Disease Identification Program: <http://www.turffiles.ncsu.edu/diseaseID/>

Turfgrass Disease Management Program: <http://www.turffiles.ncsu.edu/diseasemgmt/>

Turf Irrigation Management System: <http://www.turffiles.ncsu.edu/tims/>

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## Florida Region

# Bermudagrass Decline: Disease or Simply Declining Bermudagrass

By John Foy  
August 5, 2005

Contrary to the common assumption, the mid to late summer in Florida is not ideal for bermudagrass turf growth and maintaining premium quality putting green conditioning. Yes, hot weather is needed for sustained active bermudagrass growth, yet frequent and at times extremely heavy thunderstorm activity, typical of the summer rainy season, brings on a variety of additional problems. With the occurrence of almost daily thunderstorms, a moisture-saturated rootzone exists for extended periods of time and oxygen content is depleted. This results in the development of a weak and shallow root system, and the turf's tolerance to other environmental and mechanical stresses is greatly reduced.

Significantly reduced sunlight intensity, as a result of dense cloud cover, is another concern because photosynthesis and storage of carbohydrates is limited. Inevitably, during this time of the year we receive SOS calls and visit courses where problems with declining turf health and quality are being experienced. Problems can be especially pronounced at facilities with older Tifdwarf greens that contain a moderate to high percentage of off-type surface contamination.

Constant high humidity, warm temperatures and free surface moisture are also the ideal environmental conditions for outbreaks and rapid development of many fungal disease pathogens. While the bermudagrasses have better disease tolerance compared to most other species, when it is in a weakened condition due to intense environmental stress, its susceptibility is increased. A number of years ago, Dr. Monica Elliot, with the University of Florida, determined that *Gaeumannomyces graminis* var. *graminis* was the primary causal organism for Bermudagrass Decline (BGD) disease. Other fungi were also found and the complex is oftentimes lumped together and described as ectotrophic root infecting fungi (ETRI). These fungi are always present, but it can be debated as to whether they are a primary problem or a secondary concern, the consequence of a weak and more susceptible host. The fact that no fungicide treatments provide curative control of Bermudagrass Decline just adds fuel to the fire with regard to this debate.

Research conducted by Dr. Bruce Martin, at Clemson University, and others has found that preventative fungicide treatments in the mid to late spring can help minimize overseeding transition difficulties as well as maintain improved bermudagrass health and quality in the summer. However, it has been my experience over the years that adjustments in other management practices are needed to minimize the occurrence of and recovery from declining bermudagrass or BGD.

Raising the height of cut of bermudagrass putting greens prior to the onset of environmental stress conditions is one of the key adjustments. Increasing leaf surface area is needed to compensate for reduced sunlight intensity. With Tifdwarf putting greens, it is recommended to maintain a height of cut in the range of 0.160 to 0.180-inch. While the ultradwarf bermudagrass cultivars such as Champion, Mini-Verde and TifEagle can tolerate extremely low heights of cut, it is still advisable to come up to at least 0.125-inch or slightly higher during the summer stress period.

Naturally, with maintaining a higher height of cut, complaints about slow putting speeds

naturally, with maintaining a higher height of cut, complaints about slow putting speeds will arise. Regular double cutting and/or rolling in combination with growth regulator treatments are measures that can be employed to maintain acceptable conditioning of a consistent, smooth true ball roll and a medium fast putting speed when hosting general play. For tournament play or special events, lower heights of cut can be practiced for a short period of time, but they should be raised back up as soon as the event has concluded.

Furthermore, although verticutting is an important basic management practice for managing thatch accumulation and grain, it also exerts significant mechanical stress on the turf. Aggressive verticutting during the mid to late summer can be especially damaging to areas of off-type surface contamination, and thus care needs to be exercised with regard to both the depth and frequency of verticutting. Brush or groomer attachments on putting green mowers are less stressful alternatives, but not a total substitute for verticutting. Also, increasing the frequency of light topdressing to an every 7- to 10-day interval is very helpful in maintaining an acceptable playing quality as well as providing a degree of protection to the base bermudagrass.

# Environment and Culture Affect Bermudagrass Growth and Decline

The roles temperature and shade play in ultradwarf bermudagrass health are not totally understood.

BY RICHARD H. WHITE

**B**ermudagrass decline is a devastating root disease of highly managed bermudagrass turf, especially on golf greens in the southern United States (Elliott, 1991). It is caused by an interaction of host-predisposing abiotic stresses and the soil-borne, ectotrophic, root-infecting fungus *Gaeumannomyces*

*graminis* var. *graminis* (*Ggg*). Bermudagrass decline results in large areas of turf with weakened, short, brown-to-black root systems and an absence of feeder roots and root hairs. Symptoms include foliar chlorosis, a thinning stand, poor response to fertilizer and irrigation, and premature plant death. The pathogen causes root, rhizome, and stolon rotting.

Nutrient and irrigation management is difficult because of diminished root systems. Above-ground symptoms of infection often become evident anywhere from spring green-up through the spring and summer months, when heat and moisture stress challenge the weakened root systems of affected plants. Symptoms also are common

during cloudy, wet periods of late summer and early fall.

Drs. Joseph Krausz, Philip Colbaugh, Roy Stanford, and Richard White were part of the turfgrass research team at Texas A&M University that explored the effects of cultural, environmental, and plant growth factors on the re-

covery of bermudagrass from damage caused by *Ggg*. A strong component of the research was to gain a better understanding of how temperature and light levels influence dwarf bermudagrass growth and development. The influence of light and temperature on dwarf bermudagrass growth was of interest

because bermudagrass decline often becomes more severe during persistent cloudy and overcast conditions (Waltz, 2003). Texas A&M University research also focused on cultural practices to enhance recovery of turf exhibiting bermudagrass decline symptoms. Although bermudagrass decline is not inevitable, when the disease does occur, strategies are needed to hasten turf recovery.

## CULTURAL STRATEGIES TO ENHANCE RECOVERY FROM BERMUDAGRASS DECLINE

Recommendations for hastening turf recovery from bermudagrass decline often include raising the mowing height and applying acidifying fertilizers and/or foliar feeding (Fermanian et al.,

2003). A four-year-old Tifeagle bermudagrass putting green with severe symptoms of bermudagrass decline was used to explore cultural approaches for alleviating the disease symptoms. The green was maintained at 0.125 inch, had a soil pH of 9.1, and was previously fertilized with a coated urea nitrogen fertilizer.



Tifeagle bermudagrass green at the time of treatment initiation in July.

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**Table 1**

Turf quality in August and October for Tifeagle bermudagrass as influenced by monthly aerification with hollow or solid tines and biweekly fertilizer applications to provide 6, 12, and 24 pounds of N per 1,000 sq. ft. annually<sup>1</sup>

Nitrogen	Hollow Tine		Solid Tine	
	August	October	August	October
6	1.5a <sup>2</sup>	2.5a	2.8a	4.0c
12	2.0a	2.9a	3.2a	5.3b
24	2.2a	3.2a	3.0a	6.4a

<sup>1</sup>Plots were rated on a 1-to-9 scale, with 9 as the highest quality. A 5 was considered the minimal accepted quality level for putting greens.

<sup>2</sup>Means within months followed by the same lower-case letter are statistically similar.

Previous applications of several fungicides in a replicated trial conducted on the green the previous year were not effective in controlling the disease. A series of treatments was established in early July, including nitrogen (N) regimes, aerification, and topdressing arranged in a split-plot design. Nitrogen regimes included bimonthly applications of ammonium sulfate at 0.25, 0.5, and 1.0 lb. N per 1,000 sq. ft. to supply total annual N of 6, 12, and 24 lb. per 1,000 sq. ft. Ammonium sulfate was used in this study because of its acidifying effect on soils. Nitrogen regimes were supplemented with potassium, phosphorus, and micronutrients based on soil tests. Aerification treatments included monthly application of 0.5-inch solid tines and 0.5-inch hollow tines with cores removed. Topdressing treatments included none, 0.125-inch monthly, and 0.02-inch bimonthly. Treatments were

initiated in early July and ended in early September. Visual evaluations of turfgrass quality were taken every two weeks. Microscopy was used to assess presence of *Ggg*.

Aerification is an important management tool that is used to remove and control thatch, reduce compaction, and improve root development. In this study, hollow-tine aerification disrupted the surface more than solid-tine aerification (Table 1). Aerification of any type had not been applied to the green for several years, resulting in heavy thatch, increased disease severity, and poor rooting. The poor rooting prevented the initial hollow-tine aerification from producing quality cores and uniform coring holes, resulting in greater surface disruption and a longer healing time than when solid tines were used.

Turfgrass quality improved and evidence of bermudagrass decline

symptoms decreased with increasing N, especially for the solid-tine aerification treatments. Although 24 lb. N per 1,000 sq. ft. is considered excessive, this treatment, in conjunction with solid-tine aerification, resulted in rapid turf quality recovery and diminished bermudagrass decline symptoms. Also, turf quality increased and bermudagrass decline symptoms decreased to a greater extent for increasing N in conjunction with heavy topdressing than when compared to no and light, frequent topdressing (Table 2).

The most advantageous treatment combination for recovery from bermudagrass decline symptoms and improvement of turfgrass quality was solid-tine aerification, heavy topdressing, and 24 lb. N per 1,000 sq. ft. This improvement in quality was accomplished without raising the mowing height above 0.125 inch. Excessive N can cause rapid thatch accumulation, and the combination of the greatest amount of N and no topdressing became excessively soft by early October. However, plots that received the greatest amount of N in conjunction with heavy or light topdressing remained firm. Surprisingly, the large amounts of N used did not contribute to an excessive vertical growth rate.

The marked recovery of Tifeagle from bermudagrass decline symptoms for specific treatment combinations occurred even though *Ggg* was still present. Large amounts of N, as used in this study, should not be applied to bermudagrass for long periods of time due to the potential to negatively impact the environment and because of the potential to cause excessive thatch accumulation and contribute to reduced stress tolerance. Increasing N for short periods to enhance recovery followed by lower amounts of N to sustain turf density is, however, a more reasonable approach. Careful rootzone pH management combined with a sound nutrient management plan may reduce severity of bermudagrass decline (Waltz, 2003).

**Table 2**

Turf quality of Tifeagle bermudagrass in October as influenced by none, biweekly dusting, and monthly heavy topdressing<sup>1</sup>

Nitrogen	Topdressing Treatments		
	None	Dusting	Heavy
6	3.1b <sup>2</sup>	3.0b	3.2c
12	3.5b	3.6b	5.0b
24	4.1a	4.4a	6.1a

<sup>1</sup>Plots were rated on a 1-to-9 scale, with 9 as the highest quality. A 5 was considered the minimal accepted quality level for putting greens.

<sup>2</sup>Means within months followed by the same lower-case letter are statistically similar.

## DWARF BERMUDAGRASS GROWTH AND DEVELOPMENT IN RESPONSE TO ENVIRONMENT

The optimum temperature for growth of bermudagrass is 80 to 95°F (Beard, 1973) and for *Ggg* the optimum growth temperature in culture is 86°F (Fermanian et al., 2003). Thus, both the bermudagrass and pathogen should grow and develop well at a range of 80 to 90°F. Controlled environment chambers were used to explore the effect of temperature on bermudagrass and *Ggg* and on subsequent disease development.

A single sprig of Tifdwarf bermudagrass was surface sterilized and then grown in a greenhouse for several months to create planting stock for use in the experiment. Sprigs were obtained from the single stock plant and established in individual containers to receive the following treatment combinations. Treatment combinations included inoculated and uninoculated plants, nitrogen treatments of 4, 8, and 12 pounds of N per 1,000 sq. ft., and temperature regimes of 95/80, 80/66, and 66/51°F day/night temperatures. Artificial lighting provided about one-third of full sunlight.

Nitrogen nutrition influenced growth but was not as influential as the temperature regime. The effects of temperature regimes were robust. Internode length was 0.23, 0.51, and 0.83 inch, and leaf length was 0.29, 0.45, and 0.49 inch for the 95/80, 80/66, 66/51°F (day/night)

bermudagrass. Although Tifeagle was not a main focus of the growth chamber study, Tifeagle bermudagrass was exposed to the same temperature regimes to determine if growth responses were similar in Tifdwarf and Tifeagle. Similar responses to tempera-

ture regimes were observed for Tifdwarf and Tifeagle.

### SUMMARY

The results of this study explain why raising the mowing height is often recommended as a cultural practice to reduce bermudagrass decline symptoms. In coastal and other areas affected by long periods of overcast, rainy weather, growth habit of dwarf bermudagrasses may change dramatically



Tifdwarf bermudagrass growth response to temperature regimes of 95/80°F (left) and 80/66°F day/night (right). Plants were grown in growth chambers with 14 hours artificial light at about one-third of full sunlight.

temperature regimes, respectively. While the growth responses exhibited by Tifdwarf in these growth chamber studies were consistent with growth under low light (shade), temperature was a controlling factor in the degree of response. Additional studies were conducted with light levels of about 10, 25, and 50% of full sun within temperature regimes of 95/80 and 80/66°F (day/night). Decreasing light caused increases in leaf and internode length, but the degree of increase was regulated by temperature. The results of this study indicate that temperature as well as light levels regulate expression of dwarfism in Tifdwarf

in response to low light and lower temperatures. The altered growth form of Tifdwarf that may occur during overcast and rainy weather would not likely tolerate close mowing heights. Thus, mowing stress would result in increased sensitivity to pathogenic organisms such as *Ggg* and may result in more substantial expression of disease symptoms. Weather data from Texas A&M University indicate that the high and intermediate temperature regimes and light levels used in this study can occur in southern climates in late summer and early fall during periods of heavy rain and week-long

periods of overcast skies. Evidence of the altered growth form was observed within three to four days of exposure to the moderate temperature regime and light levels used in this study.

In addition to the potential implications that the changes in growth habit in response to environment may have for disease tolerance in dwarf bermudagrass, the effect of temperature on dwarf bermudagrass growth habit has tremendous implications for dwarf bermudagrass golf green management. The effects of temperature on growth habit of dwarf bermudagrass may explain why excessively large amounts of N applied during summer did not cause more robust

vertical growth of Tifeagle in the field study described earlier in this text. Temperature should also be a major consideration in the timing and severity of cultivation practices such as core aerification. Healing of surface disruption caused by core aerification and vertical mowing may occur extremely slowly during July and August, periods previously perceived to support maximum bermudagrass growth. During exposure to the high temperatures of July and August, for example, the growth habit of many dwarf bermudagrasses may be extremely compact and not conducive to recovery

from injury caused by aerification and vertical mowing. Tolerance to pests and wear also may be less during high-temperature periods. Establishment rates of dwarf bermudagrasses may be dramatically affected by seasonal changes in temperature, with slow

- Low light caused increased leaf and internode length in dwarf bermudagrasses, but temperature regulated expression of the dwarf growth habit.
- The alterations in growth form in Tifdwarf bermudagrass caused by low light and cooler temperatures that often

occur during overcast rainy periods justifies raising the mowing height to reduce mowing stress that may contribute to bermudagrass decline severity.

- High temperatures cause a compact growth habit in dwarf bermudagrasses and may slow healing of surface damage caused by cultivation or pests.



Effect of monthly solid (front three rows) and hollow-tine (back three rows) aerification, nitrogen at 6, 12, 24 and 6, 12, 24 lb. N per 1,000 sq. ft. (front to back), and heavy, light, and no topdressing (left to right) on the appearance of Tifeagle bermudagrass.

establishment occurring at temperatures greater than 90°F. This notable discovery about the effects of temperature on dwarf bermudagrass growth and development provides strong rationale for additional research on numerous aspects of bermudagrass culture, establishment, and pest and abiotic stress tolerance.

### RESEARCH SUMMARY POINTS

- Tifeagle bermudagrass recovery from bermudagrass decline symptoms was enhanced by aerification, heavy topdressing, and aggressive fertilization with ammonium sulfate.

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# A Guide to Fungicide Resistance in Turf Systems

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This fact sheet is written as a guide to better understand fungicides and how they can be better utilized to control turfgrass diseases. Words in **bold** type are defined in the glossary at the end of this fact sheet.

## Introduction

Fungicides are applied to turf to prevent the growth or penetration of disease-causing fungal organisms. Fungal organisms cause more diseases on turf than other microorganisms (Wong, 2006). Fungicides are the class of pesticides used to control fungal organisms, and they can be categorized into many different groups based on their **biochemical mode of action** (MOA) and chemical structure. Additionally, fungicides are classified based on their mobility in the plant after application.

**Topical MOA** characterizes fungicides based on their mobility in the plant. The four topical MOA categories are **contact**, **localized penetrant**, **acropetal penetrant** and **true systemic** (Martinez et al., 2006) (Table 1). Contact fungicides do not enter the plant but instead coat the leaf surface to inhibit fungal germination or penetration of a broad range of active fungi. Since the fungicide remains outside the plant and is exposed to environmental factors, contact fungicides remain active for only 7-10 days. They may be lost

due to rain and irrigation or by mechanically removing the fungicide by mowing. Proper spray coverage is critical with contact fungicides because the fungicide only protects the portion of the plant it contacts.

The remaining three topical MOA categories enter the plant but differ in the distance **translocated** (moved) once inside. Localized penetrants enter the leaf where the fungicide rests and move to the opposite side of the leaf, only protecting the small area covered due to limited mobility of the fungicide within the plant. Acropetal (upward moving) penetrants enter the plant and move through the **xylem**, protecting the initial leaf entered and younger plant material above the entrance point. The only fungicide that is translocated as a true systemic is fosetyl-Al (Chipco Signature). This fungicide enters the plant and moves in the xylem and **phloem**, distributing the chemical throughout the entire plant. The three penetrant types result in a longer protection (14-28 days), since they are not affected by external environmental conditions. Since these fungicides are inside the plant, they can be applied as curative fungicides when active infection has taken place. These fungicides have a tendency to be more selective than contact fungicides; therefore, it is important to identify the fungus you are targeting before choosing a penetrant fungicide.

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**Table 1. FRAC groupings, biochemical mode of action and mobility of fungicides applied to control turfgrass diseases. Table compiled from Jung et al. (2007), FRAC Code List (2010) and Wong (2006).**

	FRAC Group	Chemical Group	Active Ingredient	Trade Names*	Target Site	Mobility	Resistance Risk?
Multi-site	M	Dithiocarbamates and Ethylene-bis-dithiocarbamates (EBDCs)	Mancozeb	Fore 80WP	Multi-site	Contact	None
			Thiram	Thiram 75DG	Multi-site	Contact	None
			Chlorothalonil	Daconil Ultrex	Multi-site	Contact	None
Unknown	33	Phthalimides	Captan	Captan 80WDG	Multi-site	Contact	None
			Fosetyl-Al	Chipco Signature	Unknown	True systemic	Unknown
			Phosphoric acid	Alude	Unknown	True systemic	Unknown
Single-site	14	Aromatic Hydrocarbons	Chloroneb	Teremec SP	Lipid peroxidation	Contact	Low
			Etridiazole	Terrazole 35WP			
			Quintozene (PCNB)	Terraclor 75W			
	1	Benzimidazoles	Thiophanate methyl	Cleary's 3336	Beta-tubulin assembly for cell division	Acropetal penetrant	High
	43	Benzimidazoles	Fluopicolide	Stellar (fluopicolide + propamocarb hydrochloride)	Delocalization of spectrin-like proteins	Acropetal penetrant	Unknown
	28	Carbamates	Propamocarb	Banol	Fatty acid synthesis	Localized penetrant	Low
	2	Dicarboximides	Iprodione	Chipco 26GT	Lipid peroxidation via NADH cytochrome c reductase	Localized penetrant	Moderate
			Vinclozolin	Curalan		Localized penetrant	
	7	Oxathiins	Boscalid	Emerald	Inhibition of mitochondrial respiration via succinate-dehydrogenase	Acropetal penetrant	Moderate
	Flutalonil	Prostar 70WP	Acropetal penetrant				
4	Phenylamides	Metalaxyl	Subdue	RNA polymerase I (oomycetes only)	Acropetal penetrant	High	
		Mefanoxam	Subdue MAXX		Acropetal penetrant		

FRAC Group	Chemical Group	Active Ingredient	Trade Names*	Target Site	Mobility	Resistance Risk?	
Single-site	12	Phenylpyrroles	Medallion 50WP	MAP protein kinases	Contact	Low	
	19	Polyoxins	Endorse 2.5WP	Cell wall synthesis	Localized penetrant	Moderate	
	21	Qi-(Quinone inside) Inhibitors	Cyazofamid	Inhibits mitochondrial respiration via electron transport in cytochrome bc <sub>1</sub> at Qi site	Acropetal penetrant	Moderate to High	
	11	Qo-inhibitors (QoI's ) or Strobilurins	Azoxystrobin	Heritage 50WG	Inhibits mitochondrial respiration via electron transport in cytochrome bc <sub>1</sub> at Qi site	Acropetal penetrant	High
			Fluoxastrobin	Disarm			
			Pyraclastrobin	Insignia 20WG			
			Trifloxystrobin	Compass 50WG			
	3	Sterol Biosynthesis-inhibitors (DMIs)	Fenarimol	Rubigan AS	Ergosterol biosynthesis needed for cell membrane functions	Acropetal penetrant	Moderate
			Metconazole	Tourney			
			Myclobutanil	Eagle 20EW			
			Propiconazole	Banner MAXX			
			Triadimefon	Bayleton			
Triticonazole			Trinity/Triton				

\*Other products (trade names) not listed may be available with the same active ingredient.

Many factors and environmental conditions play a role in selecting which topical MOA fungicide should be included in specific applications.

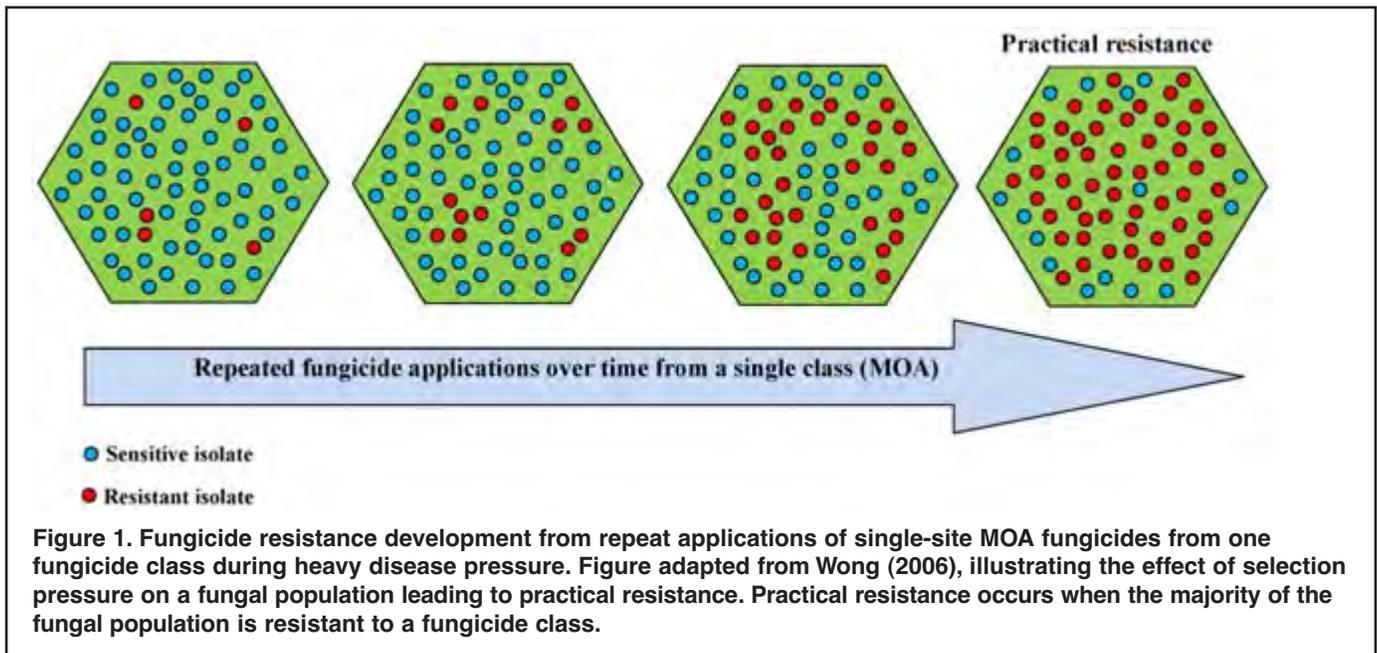
Biochemical MOA indicates the physiological portion (vegetative hyphae or spores) or metabolic process (growth or respiration) of the organism that is affected by the fungicide (Martinez et al., 2006). Fungicides are grouped into classes based on their target site within the fungal organism. Biochemical MOA can be divided further into **multi-site** and **single-site** MOA fungicides (Table 1). These names are synonymous with the fungicides' activity on the fungus. Multi-site fungicides target many locations and metabolic processes. Multi-site fungicides mostly consist of the contact fungicides discussed previously. The majority of these fungicides are older chemistries that were developed prior to the 1960s. The more recently developed fungicides target a single site and enter the plant, resulting in greater curative potential at low rates to selective fungi. The single-site fungicides target one specific location in fungal organisms. This specificity is a beneficial characteristic of these fungicides, but it also creates the potential for fungicide resistance.

## Fungicide Resistance

Fungicide resistance first became problematic with the introduction of the single-site MOA fungicides. Since these fungicides target specific locations in **genes** or **metabolic processes**, single changes in fungal DNA sequences or structural changes of **binding sites** may cause these fungicides to lose their

effectiveness. Resistance in cropping systems developed rapidly for some **fungicide classes**. The first turf pathogen to exhibit resistance was the dollar spot fungus, *Sclerotinia homoeocarpa*, to the benzimidazole class of fungicides in the 1970s (Warren et al., 1974). To date, *S. homoeocarpa* isolates from throughout the U.S. have been identified as resistant to benzimidazole, dicarboximide and demethylation inhibitor (DMI) fungicides. Further research identified fungicide resistance in *Blumeria graminis* (powdery mildew), *Pythium* spp. (pythium blight), *Pyricularia grisea* (gray leaf spot), *Microdochium nivale* (pink snow mold) and *Colletotrichum cereale* (anthracnose). The biochemical MOA plays a significant role in the rapidity and type of fungicide resistance formation.

Fungicide resistance occurs due to selection pressure (Avila-Adame and Köller, 2003). A small portion of the population may not be controlled by fungicide applications due to a genetic change in a target site. As fungicide applications are made using chemicals within a single fungicide class, selection pressure for resistant isolates is increased. The application will control all the isolates without the genetic change, but the resistant isolates persist and reproduce more fungi exhibiting the genetic change, resulting in resistance. If multiple applications are made under heavy disease pressure, resistant isolates may outnumber sensitive isolates in a short period of time. If this situation arises, continual fungicide applications from a single class may lead to chemical control failure, which is known as **practical resistance** (Martinez et al., 2006) (Figure 1).



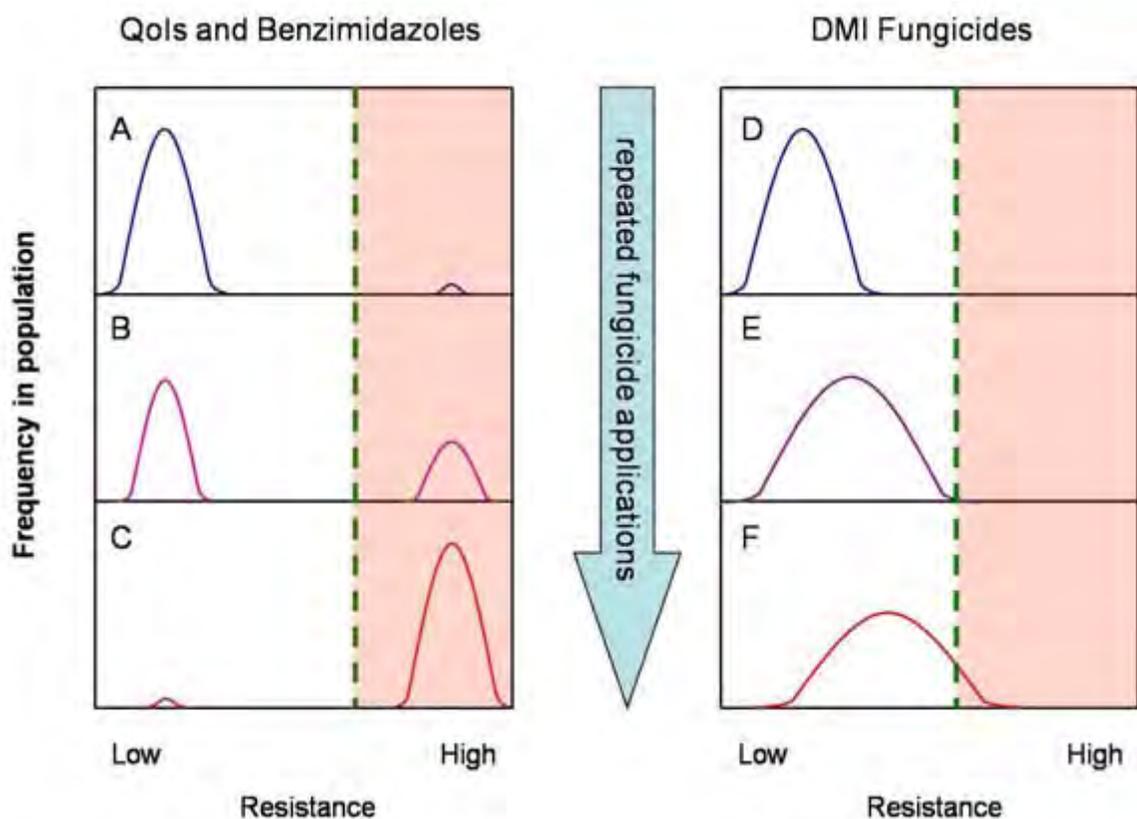


Figure 2. Two types of resistance (qualitative and quantitative) identified in QoIs, benzimidazoles, and DMI fungicides. Figure adapted from Professor Wolfram Köller (Murphy et al., 2008). QoI and benzimidazole fungicides exhibit qualitative resistance (immunity), rapidly reaching a high frequency of resistance with repeated fungicide applications. DMI fungicides exhibit quantitative resistance where increased rates or decreased spray intervals can still achieve acceptable control of the fungal population.

Two types of fungicide resistance have been described for fungal populations – **qualitative** and **quantitative resistance**. The more common of the two is qualitative resistance, which is simply immunity. When fungal isolates express qualitative resistance, increasing the rate of fungicide or decreasing spray interval will not affect the resistant isolates (Figure 2). However, populations with quantitative resistance toward a fungicide can be controlled by higher rates or decreased spray intervals between applications (Figure 2). This type of resistance has only been observed in DMI (sterol biosynthesis inhibitor) fungicides. These two types differ in that qualitative resistance occurs when a single location in a gene is targeted, whereas quantitative resistance occurs when a few metabolic processes must be altered. The continual development and use of single-site MOA fungicides led to increased reports of fungicide resistance in turf and many agricultural crops. These issues spawned the formation of the Fungicide Resistance Action Committee (FRAC) in 1987 to investigate the dynamics of fungicide resistance including management and delayed resistance for future chemistries.

Fungicide resistance has been identified within many turfgrass pathogens infecting various hosts. Resistance has also been identified in all the regions of the United States including Arkansas. Many of these studies have also identified **cross resistance** and **multiple resistance** of pathogens to fungicides. Cross resistance occurs when chemicals within the same fungicide class, sharing similar MOA, exhibit reduced sensitivity toward a fungal population. On the other hand, multiple resistance exists when a fungal population has reduced sensitivity to two or more chemical classes with completely different MOA.

This information indicates the magnitude of the problem of fungicide resistance to chemical companies producing and selling the fungicides and to golf course superintendents managing golf courses. Due to the expense of creating new chemistries and increased regulations from the Environmental Protection Agency (EPA), chemical companies are producing few new fungicides. Instead, chemical companies are focusing efforts on creating new formulations or pre-mixing products with different MOA to diminish the

potential of fungicide resistance. Golf course superintendents are under pressure to produce high quality turf. This means that fungicides are necessary for superintendents to give golfers the product they expect. There are five steps that can be used to reduce the risk of practical resistance and increase the effectiveness of fungicides (Brent, 2007).

## Managing Fungicide Resistance

### 1. **Do not use any one product or MOA exclusively.**

It is important to rotate various MOA into weekly or bi-weekly fungicide applications. Continually applying fungicides from a single fungicide class may increase selection pressure and decrease the time it takes for the resistant population to outnumber the sensitive population. In this situation, tank-mixing or buying pre-mixed packages of fungicides with different MOA can be advantageous. For example, applying a fungicide that is potentially exhibiting resistance to the target pathogen along with a contact fungicide (multi-site MOA) or a fungicide with a separate MOA not thought to be resistant can help manage resistance. The fungicide appearing to exhibit resistance will control all isolates sensitive to that fungicide, and the second fungicide will manage isolates in the population exhibiting resistance since it has a separate MOA. Therefore, rotating MOA and tank-mixing different MOA keeps the resistant population stable, reducing the potential for mass reproduction of the resistant form of the pathogen.

### 2. **Restrict the number of applications applied per season.**

This idea sounds good but may not be practical for golf course superintendents in all situations. As a reminder from general plant pathology, there are three factors that must be present at the same time for disease to occur – **susceptible host plant, virulent pathogen** and suitable environmental conditions. Collectively, these three factors are referred to as the **disease triangle**. The main factor that adjusts from season to season is environment. If environmental conditions were not conducive for disease to occur, the number of fungicide applications could be reduced; however, if conditions are similar to previous seasons when disease occurred, it would be better to manage the disease preventively with a fungicide rotation.

### 3. **Maintain manufacturer's recommended dose.**

There is some debate on whether or not lower or

split rates of fungicides can increase the frequency of resistant isolates. Keep in mind, the research and development departments at various chemical companies create label recommendations based on multiple research studies. If the chemicals are applied below label rates, the fungicides may not manage the fungal population in the desired manner.

4. **Avoid curative rates.** As mentioned previously, the fungicides capable of entering the plant are beneficial because of their effectiveness at low concentrations. For some pests, especially insects and nematodes, pesticides are not applied until a threshold is reached. This is not a practice that should be implemented for disease control because it would greatly increase selection pressure for fungicide resistance. Generally, curative rates of fungicides are higher, exposing resistant isolates to higher concentrations of the fungicide. To avoid the use of curative rates, preventive fungicide applications should be made on a regular basis to maintain problematic fungal populations based on correlating the history of disease pressure and the factors forming the disease triangle. If environmental conditions persist and the infected area is expanding rapidly, curative applications should be made, but this should be a final option.

5. **Use integrated disease management.** One of the factors in the disease triangle is a susceptible host. **Integrated disease management** is the process of utilizing management practices and chemical applications together to manage turf and reduce disease. For most turf diseases, host plants lack vigorous growth when infected. The best way to achieve vigorous turf growth consistently is by growing turf adapted for the environmental conditions present. However, maintenance practices required to prepare a golf course often reduce the ability of these grasses to withstand certain environmental stresses. Proper irrigation, fertilization, soil pH and management practices (mowing, aerification, topdressing, etc.) should be used to improve the health of turf when grown under these adverse environmental conditions in order to minimize disease.

**Chemical diversity.** Unfortunately, the chemical diversity for turfgrass fungicides seems to be headed in a less diverse direction. Many fungicides are being taken off the market or having label rates reduced due to government regulations. There have been few

completely new biochemical MOAs released to the market over the last several years. Additionally, most of the newly released products are improved formulations or pre-packaged combinations of older fungicides. These products can be used successfully to manage and prolong the sensitivity of fungal populations, but they are still the same biochemical MOA used over the years. Some of the new chemicals released over the last ten years may have been new chemistries but similar enough to older chemistries to be included in the same class (i.e., the QoIs; azoxystrobin and pyraclostrobin). Pyraclostrobin (Insignia) has shown increased control to certain pathogens although the population may have exhibited resistance to azoxystrobin (Heritage). Care should be taken with the use of any chemicals sharing closely related biochemical MOA.

## Summary

- Fungicide resistance has been identified in various turfgrass pathogens to many single site MOA fungicides throughout the U.S. and Arkansas.
- Preventive fungicide applications rotating topical and biochemical MOA may decrease the potential of practical resistance.
- Applying fungicides preventively and rotating biochemical and topical MOA fungicides can increase the effectiveness of fungicide applications.
- Use an integrated pest management strategy that includes a combination of cultural practices and fungicide applications to manage turfgrass diseases.

## Additional Information

Additional publications available at <http://www.uaex.edu/>.

Additional information about managing the turf on golf courses is available at <http://turf.uark.edu/>.

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## Glossary

Glossary adapted from Beard and Beard (2005) and D'Arcy et al. (2001).

- Acropetal penetrant** = A fungicide that is translocated only in the xylem of plant; thus, after entering a plant, this type of fungicide can only move upward.
- Binding sites** = specific sites within a gene where the chemical structure of a fungicide forms a chemical bond.
- Biochemical mode of action** = the impact of the chemical on key biochemical process(es) responsible for its effect on fungal growth.
- Contact (syn. Protectant)** = a fungicide that remains on the surface when applied; no after-infection activity.
- Cross resistance** = the condition when resistance to one chemical confers resistance to another via the same biochemical or physiological mechanisms and/or genetic factors.
- Disease triangle** = a memory aid that diagrams the three important components for disease: susceptible host, virulent pathogen and favorable environment.
- Fungicide class** = a classification of fungicides into groups based on the gene or metabolic process targeted by fungicidal chemistries.
- Gene** = the physical and functional unit of heredity that encodes a functional protein or RNA molecule.
- Integrated disease management** = a combination of strategies to reduce losses due to pathogens based on environmental and economical considerations.
- Localized penetrant** = fungicide enters the plant but remains in this location protecting a small area of plant material.
- Metabolic processes** = processes occurring within the cell in which there is the transformation of nutrients into energy, new cellular material and by-products.
- Multiple resistance** = fungi that exhibit resistance to two or more separate fungicide classes.
- Multi-site mode of action** = fungicide that affects multiple metabolic pathways within the fungus.
- Phloem** = the complex living tissue of the vascular system in higher plants that functions primarily to transport metabolic compounds from the site of synthesis or storage to the site of utilization.
- Practical resistance** = majority of fungi causing disease symptoms are resistant to a specific fungicide or fungicide class.
- Qualitative resistance** = resistance reactions that can be placed in distinct categories, usually conferred by one or a few genes.
- Quantitative resistance** = resistance reactions that have no distinct classes but vary continuously from resistant to susceptible, the result of few to many genes the individual effects of which may be small and difficult to detect.
- Single-site mode of action** = fungicide only affects a single gene or metabolic pathway within target fungi.
- Susceptible host plant** = not immune; lacking resistance; plant prone to infection.
- Topical mode of action** = identifies the location in or on the plant where a fungicide's activity will take place.
- Translocate** = to move or transfer from one place to another within the plant.
- True systemic (syn. Amphimobile)** = a fungicide that is absorbed into the plant and moves in the phloem in both the upward and downward direction; may offer some curative or after infection activity.
- Virulent pathogen** = the degree of pathogenicity or the capacity to cause disease.
- Xylem** = the complex, nonliving tissue in the vascular system of higher plants that functions primarily in the conduction of water and mineral nutrients from the roots to the shoots.

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