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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.

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
		Nitriles	Chlorothalonil	Daconil Ultrex	Multi-site	Contact	None
		Phthalimides	Captan	Captan 80WDG	Multi-site	Contact	None
Unknown	33	Phosphonates	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	Benzimides	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
			Vinclozlin	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	Fludioxonil	Medallion 50WP	MAP protein kinases	Contact	Low
	19	Polyoxins	Polyoxin-D	Endorse 2.5WP	Cell wall synthesis	Localized penetrant	Moderate
	21	Qi-(Quinone inside) Inhibitors	Cyazofamid	Segway	Inhibits mitochondrial respiration via electron transport in cytochrome bc_1 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_1 at Qi site	Acropetal penetrant	High
			Fluoxastrobin	Disarm			
			Pyraclostrobin	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).

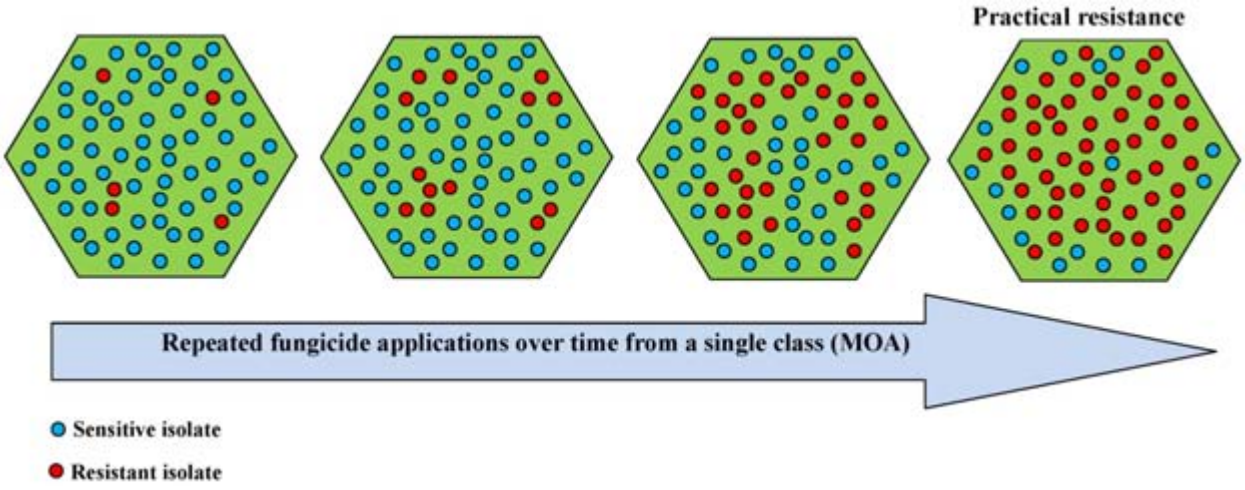

Figure 1. Fungicide resistance development from repeat applications of single-site MOA fungicides from one fungicide class during heavy disease pressure. Figure adapted from Wong (2006), illustrating the effect of selection pressure on a fungal population leading to practical resistance. Practical resistance occurs when the majority of the fungal population is resistant to a fungicide class.

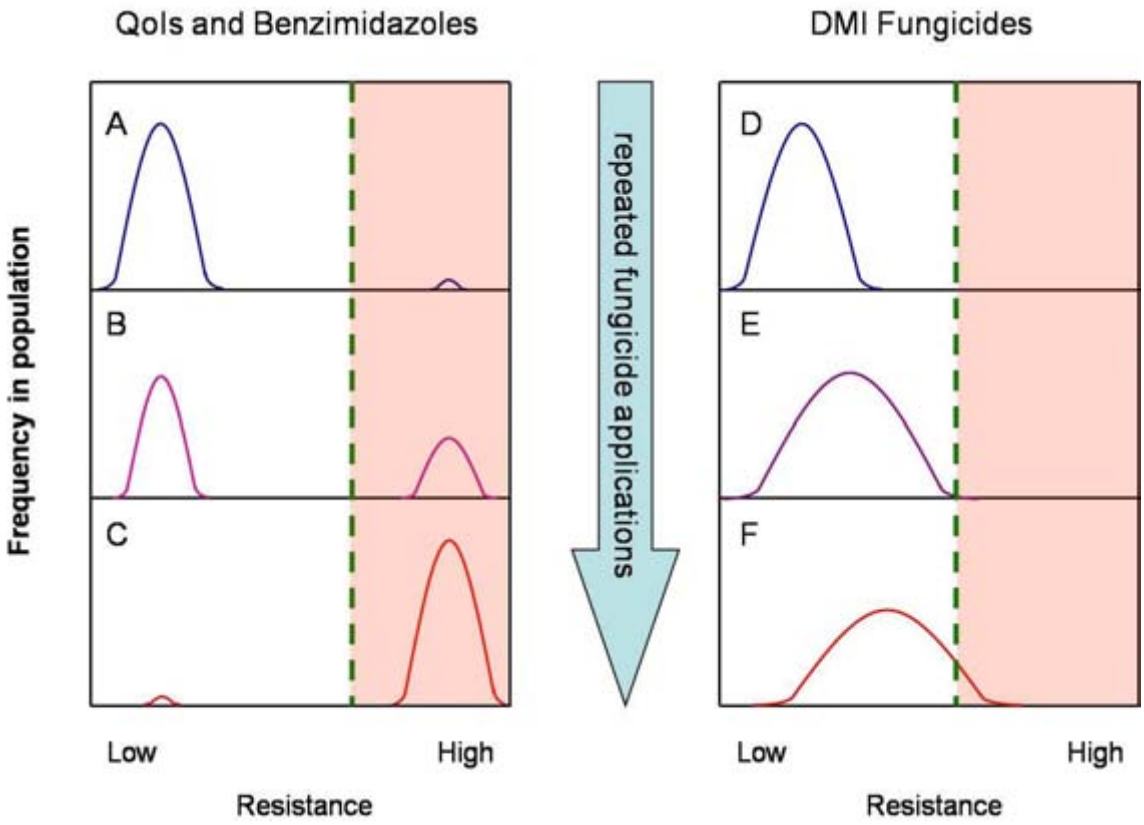

Figure. 2. Two types of resistance (qualitative and quantitative) identified in Qols, benzimidazoles, and DMI fungicides. Figure adapted from Professor Wolfram Köller (Murphy et al., 2008). Qol and benzimidazole fungicides exhibit qualitative resistance (immunity), rapidly reaching a high frequency of 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 premixing 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.uada.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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