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Nontarget effects of fungicide applications

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The development and use of highly effective fungicides have revolutionized turfgrass disease management. Though turfgrass managers are well aware of the selectivity of many fungicides, little attention has been paid to the unintended nontarget effects of such applications on the overall ecology of turfgrass.

It is often assumed that, because a fungicide is selective, it is not capable of causing damage to other turfgrass micro- and macro-organisms or to the grass itself.

Over the years, a number of nontarget effects have been observed following the application of fungicides. Many of the more thoroughly documented nontarget effects are from fungicides no longer used in turfgrass disease management. However, in this article, I am focusing on those turfgrass fungicides currently in commercial use.

Nontarget effects may be direct or indirect

Direct effects of fungicides on pathogen activity can result in the impairment or enhancement of fungal growth and reproduction. Fungicides can alter the abilities of fungal spores to germinate or survive. Indirect effects on pathogen activity are not as obvious and are generally accomplished through more complex mechanisms than those of direct effects. These indirect effects may result from changes in the interactions between multiple turfgrass pathogens, between pathogens and non-pathogenic microorganisms, and between pathogens and their host species.

Among the most poorly understood of the nontarget effects are those that directly affect the host plant and those that result in a basic change in turfgrass physiology. Even though much information is available from research on other agricultural crops, we know little about the nontarget effects of specific fungicide applications on turfgrass growth and physiology, particularly as they affect disease development.

How are fungicides classified?

Some of the major fungicides currently used

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Dealing with the unintended consequences of fungicide applications

by Christopher Sann

At first glance, the unintended, nontarget effects of turfgrass fungicide applications appear to pose little if any problem to the average turfgrass manager. In truth, almost all turfgrass managers who have used fungicides have already had to deal with these nontarget effects.

Enhancement in the severity of a disease or an increase in occurrence of other diseases after a fungicide is applied are the most frequently seen nontarget effects.

Here are some examples:

- Test results from a study to measure the severity of Dreschlera leaf spot in the spring following summer applications of a number of commonly available fungicides found that many of the systemic, sterol-inhibiting fungicides substantially increased the incidence of spring leaf spot. It also found that the previous season's use of benomyl (Tersan 1991) produced the highest levels of next spring's leaf spot damage.

- The repeated use, in the late fall and early spring, of even light rates of fenarimol (Rubigan) to control Necrotic ring spot can lead to unexpected infestations of pythium root rot in treated areas, particularly if the spring weather is cool and wet.

- Dramatic increases in the incidence of Brown patch disease following multiple high rate applications of triadimefon to control Summer patch can sometimes occur.

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for turfgrass disease control and their intended target pathogens are listed in Table 1 (See Table 1 on Page 3.). From among these fungicides, triadimefon has the broadest spectrum of activity. Other sterol-inhibiting and benzimidazole fungicides such as propiconazole, fenarimol, thiophanate methyl, and benomyl are similarly quite broad-spectrum. Of the other fungicides, chlorothalonil, iprodione, and mancozeb are among the more broad spectrum fungicides. Fungicides, such as the "Pythium" fungicides etridiazole, fosetyl Al, metalaxyl, and propamocarb, are highly selective, and are toxic generally only to a few closely related fungi.

Although fungicide selectivity has been known for many decades, few of the traditional contact fungicides exhibit a significant level of selectivity. Many of the older, metal-based fungicides such as mercury- and cadmium-based fungicides have little selectivity and are considered to be general biocides. Other metal-containing fungicides like mancozeb and zineb also have little selectivity. However, many of the newer systemic fungicides, introduced beginning in the 1960's, are so selective (e.g., the Pythium fungicides mentioned above) that only certain taxonomic groups of fungi are affected.

Although the general biocides, such as the metal-containing fungicides, possess the potential for greater nontarget effects, we have learned that even some of the newer, systemic fungicides have greater potential for nontarget effects than earlier believed. In general, all fungicides, regardless of their apparent selectivity against target species, have a wide range of biological activities that go well beyond the intended target function.

How do fungicides work?

Fungicides used for turfgrass disease control inhibit a number of metabolic processes in fungal cells. The cellular location and the biochemical pathway inhibited by the toxic action of the fungicide impart selectivity upon the fungicide being used. The specific modes of action of a number of currently available turfgrass fungicides are listed in Table 2 (see Table 2 on Page 5). Generally, all of the turfgrass fungicides fall into major "mode-of-action" classes. Each of the fungicides within a class affect fungal cells in the exact same way.

Fungicides suppress the activity of fungal pathogens either by killing fungal cells (fungicidal) or by simply suppressing growth and reproduction (fungistatic). Those fungicides that act as multi-site inhibitors or those that affect biochemical pathways (such as nuclear functions or membrane biosynthesis) common to a wide variety of organisms are more likely to exhibit nontarget effects. These would include the broad-spectrum contact fungicides such as chlorothalonil, mancozeb, and thiram as well as the broad-spectrum systemic fungicides such as the benzimidazoles (benomyl, thiophanates) and sterol inhibitors (triadimefon, propiconazole, etc.). Many of the newer fungicides act by enhancing natural plant

Table 1

Turfgrass fungicides and primary intended target pathogens

	Target pathogens														
Fungicide	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
Anilazene	+									+		+		+	+
Benomyl		+		+		+	+		+			+		+	
Chloroneb											+				+
Chlorothalonil	+	+				+			+	+		+		+	
Etridiazole											+		+		
Fenarimol			+		+	+	+	+	+			+		+	+
Fosetyl Al											+		+		
Iprodione	+					+	+	+	+			+		+	+
Mancozeb	+					+			+	+	+	+			+
Metalaxyl											+		+		
Propamocarb											+		+		
Propiconazole		+	+	+		+	+	+		+		+		+	
Quintozene	+								+			+			+
Thiophanate ethyl	+		+			+						+		+	
Thiophanate methyl		+	+			+	+	+	+			+		+	
Thiram									+			+		+	+
Triadimefon	+	+	+	+		+		+	+	+		+		+	+
Vinclozolin	+			+		+			+						

A = *Bipolaris, Dreschlera, and Pyrenophora species* (Leaf spots)
B = *Colletotrichum graminicola* (Anthracnose)
C = *Entyloma, Urocystis, Ustilago* spp. (Smuts)
D = *Erysiphe graminis* (Powdery mildew)
E= *Gaeumannomyces graminis var. avenae* (Take-all patch)
F = *Laetisaria fuciformis* (Red thread)
G = *Leptosphaeria korrae* (Necrotic ring spot)
H = *Magnaporthe poae* (Summer patch)
I = *Microdochium nivale* (Pink snow mold)
J = *Puccinia, Uromyces* spp. (Rusts)
K = *Pythium* spp. (Foliar blight and Root rot)
L = *Rhizoctonia solani, R. cerealis* (Brown patch, Yellow patch)
M = *Sclerophthora macrospora* (Yellow tuft)
N = *Sclerotinia homoeocarpa* (Dollar spot)
O = *Typhula* spp. (Typhula blight)

defenses (e.g., fosetyl Al) and exhibit little or no microbial toxicity. As such, these types of fungicides are less likely to induce nontarget effects.

Even though the fungicide in question may be very specific in its mode of action, fungal turfgrass pathogens are not the only organisms possessing that particular biochemical pathway. Many other non-pathogenic fungi as well as other microorganisms and macroorganisms possess similar pathways, particularly those that are vital to the functioning of all cells. Because of this, nontarget effects on other organisms are inevitable.

When used on turfgrasses, considerable amounts of fungicides are applied on a fairly frequent basis either as foliar sprays or drenches. This provides considerable opportunities for nontarget effects to be seen, particularly those that become more apparent following cumulative applications. Because of the proximity of the turfgrass foliage to the soil surface, the majority of the possible nontarget effects affect soil microorganisms that play important roles in the overall health and vigor of a turfgrass stand.

What are the kinds of nontarget effects?

The influence of fungicides on soil organisms and their processes depends on the physical, chemical, and biochemical conditions in the soil, in addition to the specific type and concentration of the fungicide introduced into the ecosystem. The relationships, therefore, between microorganisms, soils, turfgrasses, and fungicides are quite complex making nontarget effects indi-

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- Red thread and other minor turfgrass diseases can become problems in turf stands, where there has not been a history of such diseases, following applications of the benzimidazoles and other sterol-inhibiting fungicides.

Why do these enhancements occur?

How, why, and when this disease enhancement effect takes place will vary greatly depending on the environment at each application site. Perhaps the greatest reason for these nontarget effects, however, is the often dramatic reduction in competition from other non-pathogenic antagonistic microbes that may result from the application of a broad spectrum fungicide. Once the competitors are reduced by the nontarget effects of a fungicide, other uncontrolled pathogenic species can proliferate and become the dominant disease-causing fungi.

This ability to fill the "microbial void" left by the application of a fungicide has been a particular problem with the various Pythium species. They may be a problem-prone species because it grows rapidly and is not controlled by the majority of available broad-spectrum fungicides. Increases in Brown patch, caused by multinucleate Rhizoctonia species, may be the result of reductions in the populations of the highly competitive and antagonistic bi-nucleate Rhizoctonia species which act as natural disease controls. Although both triadimefon and propiconizole may provide adequate Brown patch control, their high-rate use to control Summer patch may well have a deleterious effect on populations of "good" binucleate Rhizoctonia species.

Should turfgrass managers stop using fungicides?

These examples of nontarget effects are not an encouragement to stop using turfgrass fungicides. Rather they are warnings to turfgrass managers that there can be undesir-

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rect. The nontarget effects of fungicide applications may present themselves in a variety of ways that include general effects:

- on microbial activities and biochemical processes in soil,
- on microbial populations leading to increased intensity of certain diseases and reduced natural biological control,
- on disease tolerance of host plants, and
- on the chemical properties of soils which influence, both directly and indirectly, the activities of turfgrass pathogens.

Fungicides affect soil respiration

Soil respiration is determined by measuring the consumption of oxygen and the liberation of carbon dioxide. This measurement has been used extensively as an indicator of soil microbial activity. Although respiration measurements reveal little about the specific microbial activities in soils, they do provide some indication of the overall health and fertility of soil. In nearly all cases, the greater the soil microbial activity, the greater the overall health and fertility of the soil.

Following the application of most fungicides, soil respiration is inhibited for only a short time. Respiration rates quickly recover and often exceed levels found in untreated soils. Although the respiration rates return to pre-application levels, the composition of the microbial community may be dramatically altered. Most often the increased activity is due to a few microbial species resistant to the applied fungicide. In some cases the increased respiration rate is due to the microbial metabolism of the fungicide itself.

Broad-spectrum fungicides have the most marked inhibitory effect on soil respiration. These include mancozeb, thiram, and triadimefon. However, this inhibitory activity may be extremely rate-specific and soil-specific. For example, in some soils, quintozene (PCNB) applied at rates of 0.2 - 0.4 oz/1000 square feet was inhibitory, whereas in other soils, applications of quintozene did not significantly affect oxygen uptake until application rates exceeded 4 oz/1000 square feet. At high application rates, triadimefon not only inhibits microbial activity, but the inhibition is irreversible. The inhibitory effects of other broad-spectrum fungicides are equally rate dependent and unpredictable. For example, propiconazole is stimulatory to soil respiration in laboratory experiments when applied at rates less than 17 parts per million, but inhibitory at higher rates. However, in the same soil in the field, rates as low as 1.25 parts per million can be inhibitory to soil respiration.

Table 2

Mode of action of turfgrass fungicides

Mode of action	Fungicide	Biochemical pathway affected
A. Broad-spectrum fungicides		
Multi-site inhibitors	Chlorothalonil Mancozeb Thiram	TCA cycle (electron transport)
Nuclear function	Benomyl Thiophanate Methyl Thiophanate Ethyl Chloroneb	Mitosis and Microtubule Formation
Membrane synthesis and function	Fenarimol Propiconazole Triadimefon	Ergosterol biosynthesis
Nuclear function and cell wall synthesis	Iprodione Vinclozolin	Mitotic instability
Unknown mechanism	Quintozene	
B. *Pythium*-selective fungicides		
Respiration	Etridiazole	Mitochondrial oxidation (electron transport)
Nucleic acid synthesis	Metalaxyl	Uredine incorporation into RNA
Cell membrane synthesis	Propamocarb	Unknown
No direct fungal toxicity	Fosetyl Al	

Soil chemical and physical properties can play a significant role in the magnitude of nontarget fungicide effects on microbial activity. For example, the inhibitory properties of benomyl are highly dependent on the pH, texture, and nutrient status of the soil. Applications of benomyl at rates up to 0.5 oz/1000 square feet were not inhibitory to microbial activity as measured by organic matter decomposition in treated clay soils, but rates as low as 0.1 oz/1000 square feet were inhibitory in sandy soil. Similarly, cellulose decomposition (another measure of microbial activity) was strongly inhibited in acidic soils (at a pH less than 6.0) but noninhibitory in alkaline soils (at a pH greater than 7.0). It is believed that benomyl is a particularly effective inhibitor of fast-growing nutrient-dependent fungi, fungi that would be more active in lower pH soils than in more alkaline soils.

It is clear that we currently know little about the means by which applications of many broad-spectrum fungicides affect the microbial activity in soils and this is one area of research in serious need of focused efforts.

Fungicides also affect nitrogen transformations

There has been considerable concern that continuous applications of turfgrass fungicides may detrimentally affect the microorganisms responsible for nitrification (conversion of ammonium to nitrate) and ammonification (conversion of organic sources of nitrogen to ammonia), but also of denitrification (conversion of nitrate to gaseous nitrogen). We can conclude from the studies conducted to date that at least some fungicide applications inhibit these processes.

For example, even though applications of thiram at

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able nontarget effects of their fungicide applications and managers should be prepared for that possibility.

What should turfgrass managers do?

Turfgrass managers should take every opportunity to educate themselves about the nontarget effects of those fungicides that they use. When they make a fungicide application decision, they should make sure they have correctly identified the problem. This includes submitting samples to a diagnostic lab when symptoms are unclear. They should also be sure that all other non-pesticide remedial actions have either been tried or ruled out as impractical. They should have determined whether the disease infestation is transient or recurring and has exceeded their treatment threshold for that site. But, most importantly, managers should select the most narrowly-focused fungicide labeled for that disease and apply it at the minimum rates required to suppress the unwanted symptoms.

If the unintended, nontarget disease symptoms become chronic, have posed an historic problem, or are reoccurring with some regularity, then turfgrass managers should look for an alternative treatment method. This may include alternating fungicides to control the original disease problem and making a major effort to identify and correct those site-specific environmental conditions that favor the nontarget pathogen. Turf sites that have chronic disease problems are most likely to show the adverse nontarget effects of the high levels of fungicide applications.

The most effective disease control is no disease

If managers are able to eliminate or reduce many of the contributing environmental factors, such as shade, poor drainage, vulnerable turfgrass varieties, and poor air circulation, then the primary and secondary chronic disease infestations are very likely to disappear.

Now the goal is achieved: Understand the condition and prevent the disease from occurring. ■

0.02 to 0.2 oz/1000 square feet are not inhibitory or even slightly stimulatory to nitrification, application rates as high as 0.12 and 0.8 oz/1000 square feet can be inhibitory. Applications of quintozene (0.2 - 0.3 oz/1000 square feet), anilazine (0.01 - 2 oz/1000 square feet), and benomyl (0.4 - 1.2 oz/1000 square feet) are also known to be inhibitory. It is believed, at least with anilazine, that the inhibitory effect is primarily on species of *Nitrosomonas* which convert ammonium to nitrite and not on *Nitrobacter* species, which convert nitrite to nitrate.

Metalaxyl applied at rates of 0.01 and 0.02 oz/1000 square feet can significantly reduce nitrification, primarily by inhibiting species of *Nitrobacter* that are responsible for the conversion of nitrite to nitrate. Similarly, triadimefon applied at 10 parts per million is strongly inhibitory to *Nitrobacter* species.

In other studies, however, foliar sprays of anilazine, benomyl, thiophanate methyl, thiophanate ethyl, and mancozeb to Kentucky bluegrass turf for 14 consecutive weeks were not inhibitory to nitrification, even though the same fungicides were inhibitory when incorporated into soil. The low toxicity of surface-applied fungicides is believed to be due, in part, to their retention at the soil surface which results from their low water solubility, low volatility, and sorption to clay minerals and to thatch.

The nontarget effects on denitrification and ammonification processes have been studied less. However, both benomyl and thiram have been shown to be inhibitory to denitrification when applied at high concentrations. At low concentrations, these same fungicides may even be stimulatory. Ammonification processes in soil may be stimulated by applications of thiram and quintozene, but inhibited by applications of anilazene, benomyl, or mancozeb.

What are the effects on soil microorganisms?

Reports vary with respect to the effects of fungicide applications on populations of various groups of microorganisms. Surprisingly, following nearly all fungicide applications, populations of bacteria and actinomycetes actually increase in treated soils. Studies conducted at Cornell University nearly 20 years ago indicate that some combinations of fungicides suppress a wider spectrum of soil fungi than a single fungicide applied alone. However, even those fungicides applied singly may be quite suppressive to certain microbial populations. Fungicides such as benzimidazoles (benomyl and thiophanates) and sterol inhibitors (propiconazole, triadimefon, etc.) generally suppress populations of fungi more than do other turfgrass fungicides. Applications of propiconazole, benomyl, or chlorothalonil may reduce both fungal and bacterial populations, but these generally recover to pre-application levels within one month after the last application. Furthermore, applica-

Table 3

Fungicides that increase the severity of turfgrass diseases

Fungicide	leaf spots	dollar spot	red thread	rusts	stripe smut	yellow tuft	Rhizoctonia diseases	Pythium diseases	summer patch	pink snow mold	Typhula blight
Anilazene		+							+		
Benomyl	+	+	+	+							
Chloroneb							+				
Chlorothalonil	+	+			+				+		+
Etridiazole		+					+				
Iprodione						+					
Mancozeb		+								+	
Metalaxyl			+				+				
Propiconazole								+			+
Quintozene		+			+		+	+			
Thiophanates	+	+	+	+							
Thiram	+	+			+		+				
Triadimefon	+					+		+			

(Modified from Smiley, 1981)

tions of triadimefon, chlorothalonil, or iprodione may reduce populations and spore viability of *Bacillus popillae*, the bacterium causing milky spore disease of white grubs.

Application of fungicides such as anilazene, chlorothalonil, iprodione, mancozeb, and thiram may actually stimulate microbial populations. Furthermore, these fungicides apparently do not inhibit levels of *Acremonium* endophytes in perennial ryegrasses. Regardless of whether fungicides inhibit or stimulate fungal populations, qualitative changes in fungal species composition may be dramatically affected.

More recent studies verify that qualitative changes in bacterial and actinomycete populations are less marked than those for general soil fungal populations. Studies in which fungicides are incorporated into soils suggests similar qualitative and quantitative trends in microbial populations. Even though few long-term studies of microbial responses to fungicide applications to turfgrasses have been conducted, current available information suggests that long-term repeated applications of some fungicides may not necessarily reduce populations of fungi in turfgrass soils, but they may drastically alter the fungal composition of treated turfgrass soils creating a number of undesirable responses. The negative side effects of these applications are often cumulative, occurring over the course of several years. In some instances, these effects may even be seen after as few as one or two applications.

Fungicide can enhance nontarget diseases

It is not uncommon to see increased severity of certain turfgrass diseases following the application of fungicides. In the past, these occurrences have not always been recognizable on uniformly treated turf. However, more of these increases are now being observed as blanket fungicide applications on turfgrass are being replaced with spot applications. The increase in severity of a nontarget disease following fungicide applications has been termed "disease trading". This occurs when the target pathogen is controlled by the fungicide and a minor pathogen is stimulated, and becomes the dominant disease-causing agent. Both systemic and non-systemic fungicides have been shown to exhibit these effects.

A number of mechanisms of increased disease incidence and severity may occur following fungicide applications. They include:

- the appearance of fungicide-resistant pathogen strains that are more virulent than the wild-type population,
- the inhibition of host defense mechanisms, and
- the disruption of microbial antagonism that naturally limits the activity of pathogens.

A number of turfgrass diseases may be intensified following the application of broad-spectrum contact fungicides. For example, applications of chlorothalonil may increase the incidence and severity of *Dreschlera* and *Bipolaris* Leaf spots, Summer patch, and Typhula blight. Increases in Summer patch severity have similarly been observed following applications of anilazene. Other broad-spectrum contact fungicides such as mancozeb and thiram may intensify *Rhizoctonia* diseases (Brown patch and Yellow patch) and *Dreschlera* and *Bipolaris* Leaf spots.

During the past 15 years, nearly 100 examples of fungicide-induced increases in turfgrass diseases have been documented. A number of these examples are summarized in Table 3 (See Table 3 on Page 7.).

Since many of the newer fungicides used for disease control in turfgrasses are relatively broad-spectrum systemics with little or no activity against some of the physiologically unique fungal groups such as *Pythium* species, it is not surprising that *Pythium* diseases are frequently enhanced following the application of many of the systemic fungicides available for turfgrass disease control. Enhanced severity of *Pythium* blight caused by *Pythium aphanidermatum* and other *Pythium* species following the application of benzimidazole and thiophanate fungicides was confirmed nearly 20 years ago. Since then, numerous other examples of increased activity of *Pythium*-incited foliar and root diseases of turfgrasses have been observed. Most recently, applications of propiconazole and triadimefon were shown to increase the severity of *Pythium* crown and root rot of creeping bentgrass.

Even though the exacerbation of *Pythium* diseases has been observed most frequently with systemic fungicides, the potential also exists for disease enhancement from contact fungicides such as quintozene, since similar nontarget effects have been observed consistently in other agricultural and horticultural crops. It is believed that such nontarget effects are not the result of a direct interaction between the fungicide and the nontarget *Pythium* species, but rather on other soil fungi and actinomycetes that may function as antagonists or competitors with the target *Pythium* species. The enhanced development of leaf spot diseases following the application of benzimidazole and thiophanate fungicides has also been attributed to the negative effect of these fungicides on antagonistic microorganisms. These studies indicate the potential for natural biological control processes to limit the activities of turfgrass pathogens and suppress the diseases they cause.

Nontarget effects can reduce natural disease control

The beneficial effects of nonpathogenic antagonistic microorganisms cannot be overestimated in a turfgrass ecosystem. In a perennial plant system such as turfgrasses, these antagonistic microorganisms exist in a delicate balance with the host plant, providing in many cases, a considerable level of natural disease control. The exploitation of these interactions forms the basis of biological disease control. The applications of broad-spectrum fungicides have been clearly shown to affect the activity of antagonistic microorganisms.

In a Netherlands study, it was shown that populations of the leaf spotting pathogen, *Cochliobolus sativus* (=*Bipolaris sorokiniana*), increased on ryegrass receiving benomyl applications. This population increase was highly correlated with reductions in populations of antagonistic bacteria and yeasts on the leaf surfaces. Similar disruptions of natural biological control may be responsible for increases in cool-season diseases such as Typhula blight (*Typhula incarnata*) and Yellow patch (*Rhizoctonia cerealis*) following benomyl applications.

Recently in our laboratory at Cornell University, we have observed that increases in *Pythium* root and crown rot of creeping bentgrass caused by *Pythium graminicola* could be enhanced following applications of propiconazole and triadimefon. Although population levels of bacteria and fungi were not decreased in treated plots, the composition of fungal species was dramatically altered. Populations of the antagonistic fungus, *Trichoderma*, were high in non-treated plots but were undetectable in plots receiving six consecutive monthly applications of either of these two systemic fungicides.

In other intriguing studies, increased populations and activity of antagonistic microorganisms following fungicide applications were shown to be a major factor in the efficacy of the fungicide. Applications of metalaxyl to sterile and non-sterile soils revealed that the fungicide is a better inhibitor of *Pythium* and related fungal species in non-sterile soils than in sterile soils. The applications of metalaxyl apparently increase populations of bacteria capable of destroying the fungal mycelium and largely account for the increased activity of metalaxyl in these soils.

At present, detailed microbiological analyses of fungicide-treated turfgrass soils are lacking. Certainly, these studies will be important in understanding the nature of disease suppression in turfgrass ecosystems. Such studies will also help to clarify pathogen-antagonist interactions that affect turfgrass health and would further reveal those fungicides for which potential harmful side effects might be anticipated.

Fungicides are not the only culprit

Nontarget effects also occur from the application of herbicides, insecticides, and growth regulators. Some of these can be just as damaging as those caused by fungi-

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Common turfgrass fungicides

Listed by active ingredient and trade names

Active ingredient	Trade names
Anilazene	Dyrene, Dyrene 4, Grow Well Lawn Fungicide, Professional Lawn Disease Control, Twin Light Turf Fungicide
Benomyl	American Benomyl, Benomyl Turf Fungicide, Bonide Lawn Fungicide, Hi-Yield Benomyl, Lesco Benomyl, Rockland, Benomyl, Tersan 1991, Twin Light Benomyl
Cyproconazole	Sentinel
Chloroneb	Scotts Proturf Fungicide V, Scotts Proturf Fungicide IX, Terramec SP, Terreneb SP, Twin Light Chloroneb
Chlorothalonil	Daconil 2787, Echo 500, Green Gold Turf Fungicide, Greenview Lawn Fungicide, Hi-Yield Daconil, Lawn Vegetable & Flower Fungicide, Lesco Manicure, Lesco TwoSome, Multi-Purpose Fungicide, Rockland Lawn Fungicide, Tee Time Turf Fungicide
Etridiazole	Koban, Terrazole
Fenarimol	Lesco TwoSome, Rubigan AS
Fosetyl A1	Aliette, Lesco Prodigy
Iprodione	Chipco 26019, Scotts Disease Control + Lawn Fertilizer, Scotts Protruf Fluid Fungicide, Scotts Protruf Fungicide X, Scotts Protruf 23-3-3 + Fungicide VIII
Mancozeb	Bonide Mancozeb Flowable with Zinc, Dithane F-45, Dithane WF, Dragon Lawn & Vegetable Disease Control, Dragon Mancozeb, Duosan, Fore, Lesco 4 Flowable, Pace
Metalaxyl	Apron, Pace, Scotts Proturf Fluid Fungicide II, Scotts Proturf Pythium Fungicide, Subdue, Subdue Granular
Propamocarb	Banol
Propiconazole	Banner
Quintozene	Ferti-Lome containing fungicide, Fluid Fungicide II, Lesco 10-3-23 + PCNB, Lesco PCNB, Lesco Revere, Penstar, Proturf 14-3-3, Terrachlor, Turfcide, Turfgo Engage
Thiophanate ethyl	Clearys 3336-F, Clearys 3336, Dragon Systemic Fungicide, Thimer Plus F
Thiophanate methyl	Disease Control + Lawn Fertilizer, Duosan WSB, Duosan, Fungicide IX, Fungo, Fungo FLO, Fungo 50, Proturf Fluid Fungicide, Proturf 23-3-3 + Fungicide VIII, Proturf Systemic Fungicide
Thiram	Lesco Thiram, Prolawn Thiram 4F, Spotrete F, Spotrete, Twin Light Disease Stopper, Thimer Plus F
Triadimefon	Bayleton, Bayleton (PVA), Hi-Yield Lawn Fungicide, Lawn Disease Control with Bayleton, Lebanon Turf Fungicide, Lesco Granular Turf Fungicide, Lesco 17-0-17 Elite Fertilizer + Lawn Fungicide, Procide G, Proturf Fungicide VII, Proturf 28-012 + Fungicide, Proturf Fluid Fungicide II, Turf Fungicide, Turfgo Accost, 1% Turf Fungicide with Bayleton, Twin Light Disease Stopper
Vinclozolin	Curalan, Touche, Vorlan, Vorlan DF, Vorlan Flo

Turf Grass TRENDS

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Timing is everything for an effective weed management program

by Dr. Joseph C. Neal
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In turfgrass management we are fortunate to have many effective tools (herbicides) for the control of weeds. In general, the herbicides are very effective and have broad windows of application. However, like all other management inputs, to obtain the maximum benefit from a minimum of effort and expenditures, careful attention to product choice, dosage, application uniformity and the timing of these applications are essential. Optimizing when you implement your weed management program will improve scouting results and efficiency, weed control, and turfgrass safety.

Weed scouting

Making informed management decisions requires information. In this case, in order to control weeds you must know what species are present, their relative abundance (has the infestation exceeded your "threshold" for acceptable turf quality?), and where the infestations occur.

Weed scouting need not be a labor intensive or time consuming process. The first step is to divide the area into management units. In lawn care this may be as simple as front, back and side yards. In golf courses obvious management units are tees, fairways, roughs, and greens for each hole. The second step is to determine the intensity of management and what weed or amount of weed cover will be acceptable in that particular site. The third step is to scout the property.

In Cornell University's integrated pest management (IPM) program, we have found that a simple inventory of the species, followed by highlighting the more important or prevalent species, combined with noting when patterns of weed cover are present, i.e. are weeds throughout, spotty, or in a patch somewhere, will provide adequate information for decision-making.

Scouting is best done when all weeds are present and when turf quality concerns can be addressed to improve turf density before weeds germinate. The late summer or early fall has proven to be the best time to scout. Then, summer annual weeds, both monocot and dicot, are mature, perennial weeds are present, and winter annuals are germinating. Also, cool-season turfgrasses are actively growing and can fill gaps left by dying weeds. Also, turf can be successfully overseeded at this time of the year. A follow-up scouting in late spring can identify weeds that escaped fall or spring treatments and seedling summer annual weeds can be treated when they are small and easier to control.

Weed control

Many herbicides are available for controlling turfgrass weeds. Getting the most out of these products requires that they be applied when they can do the most good. Optimum timing of herbicide applications are influenced by many interrelated factors including:

Photo provided by Dr. Joseph C. Neal, Cornell University

large crabgrass

- Weed species and physiology — particularly time of emergence, development and seasonal variation in sugar translocation within the plant;
- Climatic factors — temperature and moisture primarily;
- Turfgrass species and management — warm season versus cool-season species, mowing height, irrigation, fertility, cultivation events, etc.; and
- Herbicide chemical properties and mode of action — each family of herbicides kills plants in different ways and they decompose in the soil at different rates.

To understand how these factors influence herbicides efficacy, different weed control strategies and categories of herbicides must be discussed separately.

Annual grass control

Crabgrass and goosegrass are the most common summer annual grass weeds in turf and are typically controlled with preemergent herbicides. Ideally, preemergent herbicides should be applied about two weeks prior to weed germination. Crabgrass germinates when the soil temperature is between 55 and 60 degrees Fahrenheit. Goosegrass has an absolute requirement for 65 degrees Fahrenheit to germinate, so it emerges later than crabgrass.

Unfortunately, predicting when soil temperatures will reach these critical levels with adequate soil moisture present is an inexact science. Instead we use indicator species to tell when weather is conducive to germination. In warm season turf, preemergent herbicides should be applied by the time dogwoods are in full bloom. In cool-season turf the soil warms more slowly and application may be delayed until shortly after the dogwood blooms fade. In the northeastern U.S. we use forsythia in full bloom as an indicator for the application of preemergent herbicides.

Recent research has shown that with the newer, longer-residual preemergent herbicides there is greater flexibility in the application time. Dormant season (January and February) applications of pendimethalin, Barricade, Dimension, and Ronstar, have controlled crabgrass as well as mid-March treatments. Additionally, Dimension has the added flexibility of controlling crabgrass after it has emerged thereby extending the effective window for application by several weeks. However, once tillers form on crabgrass, Dimension alone does not provide adequate control.

Postemergent control of crabgrass may be accomplished with Acclaim or MSMA. Both products are best applied early in the season to young, about one-tiller, crabgrass. At this time, control is usually superior to later treatments. Lower rates may be used to obtain this control, and more favorable weather conditions will reduce the potential for turfgrass injury. It may also be desirable to tank-mix a low dose of a preemergent herbicide with the postemergent treatments to prevent subsequent crabgrass germination and emergence.

Postemergent control of goosegrass is more difficult. MSMA is ineffective and Acclaim is less active than on crabgrass. Acclaim applications should be made before goosegrass has reached the three-tiller stage, with the earlier the better. Applications to larger mature goosegrass plants will be ineffective.

Nutsedge control

Nutsedge is often mistaken for a grass. With a few notable exceptions, most grass control herbicides do not control nutsedge. In certain warm-season turfgrasses metolachlor (Pennant) may be used preemergently to suppress yellow nutsedge; however, nutsedge is usually controlled postemergently.

Basagran or MSMA are applied to young actively growing nutsedge plants with the optimum timing for these treatments based on uniformity of the emergence and the physiology of nutsedge tuber formation. Tubers sprout over an extended period of time, from late spring to mid summer. Treatments should be delayed until most plants have emerged. However, tubers are formed when days begin to shorten: after June 21st. Delaying treatments much past July 1st will allow the plants to produce tubers which will infest the turf next year. Also, delaying treatments to mid-summer increases the likelihood

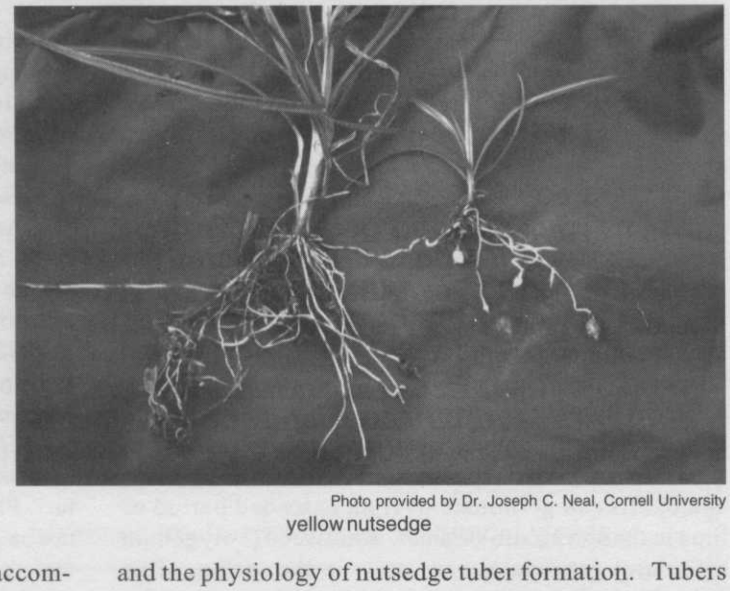

Photo provided by Dr. Joseph C. Neal, Cornell University

yellow nutsedge

and severity of turfgrass injury from the available herbicides. Therefore, the first Basagran or MSMA treatment should be made in mid to late June and followed with a second application in about 14 days.

Broadleaf weed control

Optimum timing for postemergent broadleaf weed control is when weeds are actively growing, there is adequate soil moisture, daytime temperatures are moderate (between 50 and 80 degrees Fahrenheit), and when turfgrass recuperative potential is highest. Additionally, the inherent susceptibility of the weeds must be considered. The weather conditions which favor weed growth and maximum control are usually encountered in the fall or spring. Choosing between these times depends upon the other two factors. Cool-season turfgrasses have a higher recuperative potential in the fall and can fill gaps left by dead annual broadleaf weeds more rapidly and therefore should be treated in the fall. Conversely, annual broadleaf weeds in warm season turf should be treated in the spring, several weeks after greenup, for the same reasons.

The fall is also a preferred time to treat many perennial broadleaf weeds because many are inherently more susceptible to herbicides in the fall; for example, mugwort (or chrysanthemum weed), healall and ground ivy are better controlled in the fall than in the spring. Exceptions to this rule are poison ivy which is best controlled in early summer, and seedling summer annual broadleaves (such as spurge and knotweed) which are easier to control in the late spring when they are young.

Annual broadleaf weeds may also be controlled preemergently but herbicide choice and application timing must be tailored to the species.

Winter annual broadleaves such as chickweed and henbit, germinate in late summer or fall when the soil temperatures begin to cool. Therefore, preemergent herbicide applications for winter annual weeds should be made in late summer.

Summer annual weeds such as knotweed, spurge and oxalis can germinate over an extended period of time in the spring and summer. Knotweed (Polygonum aviculare) germinates very early in the spring, often a month before crabgrass emerges. Consequently, many preemergent treatments applied to control crabgrass or goosegrass miss knotweed. To avoid this mistake, map the affected areas in late summer and consider late fall applications of Gallery or pendimethalin. Oxalis germinates over an extended period of time from early spring to late summer, but not as early as knotweed. Spurge emerges later in the season, late spring through mid-summer. Therefore, early spring crabgrass control treatments may miss these weeds. When spurge or oxalis are the problem, follow-up early summer preemergent treatments may be necessary to supplement spring crabgrass treatments.

Turfgrass safety

To reduce the potential for injury to established turf, avoid herbicide applications when turfgrasses are under stress (heat, drought, disease, etc.).

When turfgrass safety decisions are being made, two aspects of seedling turfgrass safety must be considered: the interval from herbicide application to seeding and the tolerance of seedling turfgrasses to herbicides.

Photo provided by Dr. Joseph C. Neal, Cornell University

Common dandelion

Most preemergent herbicides have treatment-to-seeding intervals of three to four months. However, there are a few exceptions to this standard: Tupersan, Dacthal, Balan and Gallery all have shorter intervals. Similarly, most preemergent herbicides should not be applied to newly seeded turf until that turf is established. Again a few exceptions exist to this rule. Tupersan may be used at the time of seeding in cool-season turf. Dacthal may be applied after the second mowing. Finally, Gallery may be applied after the seedling turf has tillered.

Postemergent herbicides may also injure seedling turf. Phenoxy herbicides, Confront and Dicamba, should not be applied until after the third or fourth mowing and turfgrass seed should not be introduced into treated areas for two to four weeks after an application. Acclaim and MSMA should be applied only to established turf; with a few exceptions which are discussed in detail on the herbicide labels.

As with any pesticide application, the label is the law. For any questions concerning a herbicide's specific use, turfgrass managers should consult the label for instructions for each turf species. ■

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Weed Management Calendar — Warm Season Turf

Late Winter	Preemergent herbicide applications for crabgrass control
Spring	Postemergent herbicide applications for broadleaf weeds
Early Summer	Weed scouting for escaped weeds and seedling summer annuals Postemergent annual grass control and follow-up preemergent applications if needed
Late June	First nutsedge treatment; Repeat 14 days later
Late Summer	Preemergent herbicide treatments for winter annual weeds
Late Summer / Early Fall	Comprehensive weed scouting
Winter	Plan, Evaluate, Repair, and Calibrate

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Weed Management Calendar — Cool Season Turf

Early Spring	Preemergent herbicide applications for crabgrass control
Early Summer	Weed scouting for escaped weeds and seedling summer annuals Postemergent annual grass control and follow-up preemergent applications if needed
Late June	First nutsedge treatment; Repeat 14 days later
Late Summer	Preemergent herbicide treatments for winter annual weeds
Late Summer / Early Fall	Comprehensive weed scouting Mid-Sept. through Mid-Oct. Postemergent broadleaf weed control
Winter	Plan, Evaluate, Repair, and Calibrate

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Are we going to hell, or is it time to disagree?

by Christopher Sann

The fragrance of freshly-cut grass, blooming daffodils, and hyacinths fills the warm air. My eyes are pleasured by the sight of blossoming dogwoods, cherry trees, and crabapples. Yet my ears are subjected to the haranguing of "environmental chicken littles," who, despite significant evidence to the contrary, repeatedly insist that no environmental progress has been made and that civilization is doomed to perish in a sterile hell of its own making. Opportunistic politicians, riding on a temporary wave of anti-governmental sentiment, say that government is wholly incompetent and incapable of successfully completing any activity it undertakes.

With both of these shrilly and emotionally avowed points of view, I loudly but politely disagree.

A substantial body of evidence that the environment is getting better is chronicled in a new book, "*A Moment on Earth: The Coming Age of Environmental Optimism,*" by Gregg Easterbrook (Viking Press). The book shows that these early efforts to stop poisoning ourselves and our planet have borne considerable fruit. The facts are as follows:

- Since 1975, airborne lead levels have been reduced by 96%,
- From 1988 to 1992, the number of people living in U.S. counties that failed federal air pollution standards fell from 100 million to 54 million,
- From 1988 to 1993, smog warnings in East coast cities fell by 64%,
- By the year 2000, the amount of sulfur dioxide emitted by U.S. electrical generating facilities and manufacturing plants will have been reduced from 28 million tons to 9 million tons, or by 68%, even though the number of coal-fired electrical generating facilities has doubled since 1970,
- The total forest acreage in the industrialized portion of the world has actually increased in the past 50 years, with a 30% increase in Western Europe in 57 years, and a 69% increase in the New England states of Vermont, Massachusetts, and Connecticut since the mid-19th century, and
- A ten year government study completed in 1991 found that there was no evidence of general decline in U.S. or Canadian forests due to acid rain, and that, despite estimates of high acidity in 50% of lakes, in fact only 4% of the lakes were found to have become acidified.

If there is evidence that the environment is getting better, then why all the noise?

Why do the hard-core doom sayers continue to discount reports of environmental progress? Most certainly some of the noise coming from environmentalists comes from a genuine sense of concern. But not all.

The rhetoric that served us all so well, to get our attention about the larger dangers of self-poisoning, now seems strangely discordant and out of proportion to the increasingly less infamous examples of self-destructive behavior that still remain. Unfortunately, their continued strident pronouncements about the end of the world are no longer being met with bemusement, befuddlement or tolerance — they are beginning to evoke a rather hostile retort from a sizable portion of the population that appears to be more concerned about individual rights than the collective good.

There have been excesses in the government's zeal to correct past practices that led to such infamous places as the Love Canal and the use of Agent Orange. But even these concerns about the excessive use of government regulation do not fully explain the level and the volume of vitriolic dialog that has taken center stage over the last 12 months.

What does all this have to do with turf?

Like it or not, all turfgrass managers have to abide by some level of government regulations, be they federal, state, or local. As flawed as many of these regulations are, they are designed to protect workers, users and the environment. Turfgrass managers have accepted the fact that regulation is part of the business "landscape," and up until the last 12 months have had some idea where these regulations were heading.

Historically, as new regulations have been implemented, turfgrass managers have ultimately relied on the regulators for information and guidance. If federal funding is slashed, then the money needed to field implement these new regulations and fund extension agents and turfgrass research specialists to develop new strategies in turf management will diminish. The safety net of government support will weaken. Consequently, turfgrass managers will be left to their own devices. Successfully navigating the ever changing regulatory and political shoals will become a major challenge, requiring untold dollars and man hours to comply. As bad as it may currently be, the future holds the possibility that it will get a whole lot worse.

What can the turfgrass industry do?

More now then ever before we in the turfgrass management industry must not only find our collective voice, we must loudly but politely disagree.

The 1990 U.S. census shows that the turfgrass management industry consists of 735,556 men and women. We are a strong force. We can no longer allow our industry's fate to be buffeted by outside forces, be they over zealous environmentalists, uninformed regulators, opportunistic politicians, bottom-line oriented manufacturers and suppliers, or a biased media.

We must find our collective voice and tell the consumers/users that we are as concerned about the environment as they are. We must show them that we are actively looking at and implementing new management strategies that will reduce the amount of pesticides that we use.

We must, loudly but politely, tell everyone involved in our field that we will no longer take a back seat to the agricultural industry. We must let them know that we will no longer just blindly use the manufacturers' products and not ask questions. They must understand that if they continue to take us for granted and continue to ignore our needs, we will buy our "tools of the trade" from manufacturers who value our work and recognize us as partners in the industry.

We must, loudly but politely, tell our local, state, and federal legislators, that we must participate in formulating regulations affecting our industry. We must insist that legislatures and regulators clearly define goals for the future use of pesticides, that we expect legislators to keep the industry well-informed about these goals, and that they must properly disseminate information relevant to our industry.

We must let the media — television, magazines, and newspapers — know that we, loudly but politely, object to their common practice of building readership and ratings with sensational stories about how turfgrass managers are poisoning the world. Let them know that we expect balanced coverage of our industry and that "pesticide horror stories" must be counterbalanced by reports of our advanced environmental work, such as Integrated Pest Management. The media need to understand that we will monitor their stories, and if they do not comply with our request for fair reporting of our work, we will contact their advertisers and let them know that we will boycott products advertised in media that report unfairly about our industry.

We must loudly, but politely, make our voice heard.

We must, loudly but politely, object to environmental doom sayers, government groups, and local media who have made it a practice to pit one side against the other in order to advance their own causes. We must demand to have a place in the decision-making process that charts our future course, and that all involved in regulating our industry provide us with accurate information so that we, as well as our customers, can make informed and sound environmental business decisions. The turfgrass industry of about three quarters of a million individuals must politely demand full and proper consideration and representation. ■

***fungicide** continued from page 8*

cide applications. As a general rule, however, these types of pesticides do not commonly act directly through the soil microbial community (with the exception of insecticides). Furthermore, there are many negative nontarget effects of fungicide applications on other components of the soil biota. For example, fungicides such as anilazene, benomyl, chlorothalonil, and mancozeb can be toxic to earthworms. Other fungicides may be equally harmful to beneficial micro- and macroarthropods.

Conclusions

The specific types of nontarget fungicide effects may be difficult to predict since the effects depend on a variety of soil and application factors such as soil pH, texture, moisture content, and organic matter content, as well as on the application rate and frequency of the fungicide. Even the history of pesticides used on the particular site will determine the nature and magnitude of the nontarget effects.

It is important to understand, however, that the application of fungicides may lead to unpredictable and peculiar effects on turfgrass diseases and general turfgrass health. These peculiar effects are likely to be more common in those sites receiving continuous applications of the same broad-spectrum fungicides. It is important, therefore, that particular attention be paid to the specifics of each application (e.g., chemical class, application rate, etc.) as well as to the intended target pathogens and the observed outcomes of the applications. This will allow one to assess each fungicide used on each particular site for any potential nontarget effects. This, coupled with equally meticulous cultural records, all of which are central to a solid integrated pest management program, will provide a means of more effectively selecting disease control strategies with a minimum of harmful side-effects. ■

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