

Turf Grass TRENDS



Volume 4, Issue 5

May 1995

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Nontarget effects of fungicide applications

by Dr. Eric B. Nelson
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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

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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

Field tips

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

Fungicide	Target pathogens														
	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, particu-

larly 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 propiconazole 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, quinterozone (PCNB) applied at rates of 0.2 - 0.4 oz/1000 square feet was inhibitory, whereas in other soils, applications of quinterozone 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. <i>Pythium</i>-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 A1	

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 non-inhibitory 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 quitozene (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 quitozene, but inhibited by applications of anilazine, 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 popilliae*, 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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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 than 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. ■

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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. ■