SELECTED PROBLEMS ASSOCIATED WITH FUNGICIDE PROGRAMMING FOR DISEASE CONTROL

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Many decisions are needed to formulate pest control programs that are consistent with the overall management needs of every turfgrass stand. The most basic of these decisions is whether a disease needs to be controlled at all; perhaps it is better in the long run to allow certain minor diseases to run their course. This is certainly true on many of the less-intensively managed sites, and maybe also on some highly managed turfs. The short address I am presenting here is not meant to be an all-encompassing treatment of the problems associated with this decision making process. Instead, I will present some of the problems we have seen or have become aware of in New York during recent years. These problems will be identified through a series of questions related to each example.

DID MY FUNGICIDE APPLICATION FAIL BECAUSE THE DISEASE DIAGNOSIS WAS ERRONEOUS?

With the advent of newer, more selective fungicides, the accuracy in disease identification is becoming increasingly important. The scenarios which follow occur with all too great a frequency.

The aerial mycelia of fungi causing dollar spot and Pythium blight can be easily confused. Depending upon the fungal isolates, the weather, and the turf species and its management, the signs of these diseases can appear as a cob-webby mycelium or a cottony type of growth. An erroneous identification of these diseases can set the stage for a complete disaster on fine turfs of bentgrass, ryegrass, or fescue. There is no margin for error in this instance because the spectrums of efficiency for the fungicides used to suppress these diseases have no overlap at all. Of course the situation is far worse if the disease was identified as dollar spot when it was in fact Pythium blight. To complicate matters further, we now know that Nigrospora species also produce similar mycelial growths in the turfgrass canopy, and can serve as pathogens on several turfgrass species.

The disease complex formerly known as Fusarium blight syndrome has been divided into the diseases summer patch and necrotic ring spot. These diseases cannot be separated on the basis of any currently recognized visual characteristics. Fortunately, some of the fungicides recommended for controlling the complex appear to control both of the newly identified components. It is also true, however, that a few of the more popular fungicides only work against one of these diseases. A lack of disease control at one site, and very efficient control at another may simply indicate that different pathogens caused the patches at these sites. More detailed information is presented in another paper written for this conference.

An emerging problem also occurs with our ability to control winter diseases. Mobay Chemical Corporation is currently rewriting its label for Bayleton, and is modifying (or perhaps deleting) its registration for controlling Typhula blight. The problem is that the fungicide controls the disease caused by Typhula incarnata but not that caused by T. ishikariensis. It is not customary in most regions to distinguish among the diseases caused by these closely related pathogens. The possibility, therefore, exists that consumers could innocently make applications which are doomed to inefficiency if T. ishikariensis is the primary species present in their region. We must, therefore, now distinguish among these forms of Typhula blight. The dominant pathogen in most regions appears to be T. incarnata. The loss of this product registration would represent a serious loss to our arsenal of fungicides for preventing the occurrence of this snow mold disease.

The diseases red thread and pink patch have many features in common. They are, however, caused by fungi from two different taxonomic groups. In practicial terms, this means that the diseases are unlikely to be controlled by the same types of fungicides. Proper identification can only be done by clinical diagnostic procedures, using a microscope. Alternatively, there is little substitute for experience in working on a specific turfgrass area. One soon learns which materials work well, and which do not. Differences in fungicide efficiency from one turf to another may again simply indicate that different pathogens are causing this disease complex.

WHY DID MY FUNGICIDE APPLICATION CAUSE A PHYTOTOXIC RESPONSE?

Several instances of phytotoxicity have come to our attention, and need to be shared with others. None of these problems have been adequately studied, and their precise causes are therefore the subject of speculation.

Applications of Rubigan at the upper range of recommended rates have been used at some locations to eliminate annual bluegrass from bentgrass or Kentucky bluegrass turfs. This use for the fungicide has become well known in recent years. It was unexpected, however, when low rates of this fungicide also caused extensive kill of annual bluegrass in some golf course putting greens in New York. The rates used were apparently the same as those that cause no such problems on most other putting greens. The reason for the dramatic low-dosage effect at some locations is unknown, but may relate to the fact that these greens have also been on a soil acidification program to weaken the annual blegrass. It appears possible that a synergism exists among the net effects of these two unrelated processes or chemicals. Such synergistic effects are known to occur among some fungicides; low rate applications of two chemicals are sometimes more effective than a half rate of each, or of a full rate of either one. The converse is also true; applications of some herbicides can reduce the efficiency of fungicides for controlling certain stress-related disease.

It is well known that Actidione should not be applied when the weather will become hot. This water-soluble fungicide can also poison protein synthesis processes in plants, and this causes most concern at high temperature or at high application rates. I have also observed phytotoxic

responses from this chemical applied in late autumn when high temperature was certainly not a complicating factor. The reasons are unclear, but perhaps it was related to overdoses caused by the mist blower being used to apply the fungicide. Mist blowers and any hand held application equipment are impossible to calibrate accurately. This example therefore represents a problem in programming that is caused by an inability to ensure that the correct dosage of a water soluble biocide is applied uniformly to all areas of the targeted area.

Another problem in fungicide programming has emerged at a golf course in New York. The superintendent frequently used Cadminate and Chipco 26019 in his disease control program. When they are applied separately he has no problem, but if they are mixed his turf becomes affected by a severe tip burn, especially if the weather is cool. This situation is not adequately explained, but deserves some speculation. Cadmium-based fungicides are moderately to highly soluble in water and Chipco 26019 is rather insoluble. One would therefore not expect an interaction of these materials in the tank. But the active ingredients of both fungicides have some capacity for being absorbed into the plant. The dual effects of these chemicals inside the plants may be the cause for this problem at this site. More likely, however, the superintendent is seeing the early stages of a heavy metal contamination problem. Cadmium-polluted soil causes an extreme reduction in root and shoot growth in grasses. This occurs as a result of the changes that cadmium causes in the physiology of the plant, and in cadmium's ability to restrict the uptake of potassium from soil. Moreover, cadmium pollution in soil has been shown to cause an increase in the susceptibility of ryegrass to Drechslera leaf spot. These effects occur at reasonably low levels of cadmium in soil. If the persistent use of cadmium leads to an accumulation of this metal in soil, we could therefore expect to go from a situation where the fungicide has provided good control of the diseases for which it was intended, to a situation where any additional applications progressively predispose plants to diseases and increase their sensitivity to other chemical or environmental factors which affect plant growth. The critical concentration of cadmium may be as low as 10 ppm, which could be attained in as little as 5 to 10 years of cadmium fungicide applications on some turfgrasses.

All systemically translocated fungicides have the potential to be absorbed by the plant, and to become active in the plant cytoplasm. Since many of these fungicides also affect plant growth processes at rates higher than those which are used for controlling disease, they should always be used with care. Applications should be controlled so that these fungicides are not applied in excessive amounts; avoid overlaps in boom-mounted application equipment, avoid any use of hand-held equipment, avoid frequent re-applications of the same fungicide, ensure that all applications are calibrated precisely to deliver the intended rate of active ingredient, and related precautions. The onset of phytotoxicity may not be readily apparent. Some fungicides reduce the growth rate of shoots and roots, or alter the leaf growth habit. These minor changes may reduce the competitive ability of the desired grass species, may alter the plants ability to resist infections by some pathogens, or may cause the affected plants to become increasingly sensitive to environmental stresses.

WHY DOES IT TAKE MORE THAN THE RECOMMENDED RATE OF A FUNGICIDE TO CONTROL DISEASE ON MY GRASS?

This problem is often related to the relative nature of the disease interaction between plants, pathogens, and the environment. When one factor is unusually conducive to disease development, the entire balance becomes abnormally shifted toward more disease. A strain of the pathogen at your location may be unusually virulent (strongly pathogenic) or unusually insensitive (resistant) to the fungicide, the grass species or cultivars may be unusually susceptible to attack, or the environment may be unusually favorable for the pathogen or restrictive to growth of the plant. Several such instances have been covered in the foregoing discussion, and additional examples follow.

Control of "Fusarium blight" became nearly impossible at some locations in the early 1970's. Tersan 1991, Cleary's 3336, and Fungo were the only fungicides registered for controlling this disease. They all have a similar mode of action. At some locations they worked as intended for a number of years and then failed. Several years later, the efficiencies of these chemicals increased again on the areas where they had once become ineffective. This sequence has now been shown to be related to the amount of turfgrass stress which accumulated when calcium arsenate herbicide was used to control annual bluegrass. Accumulations of arsenic placed enough stress on the desired grass, Kentucky bluegrass, that the fungicides lost their ability to control "Fusarium blight". After the manufacturing and sale of calcium arsenate was banned the fungicides again became effective for controlling this patch disease. I have conducted experimental work which demonstrated that even the superb ability of Bayleton to control "Fusarium blight" (e.g., the summer patch component of this complex) can be lost if the accumulation of arsenic becomes sufficiently high. This situation occurs at a lower concentration of arsenic than that which is visually phytotoxic to the Kentucky bluegrass. This example is therefore of a herbicide which can cause difficulty in disease control programs. There are undoubtedly many more examples that could be presented. In particular, work in Iowa has shown that some hormonal herbicides, such as MCPP and 2,4-D can alter the susceptiblity of grasses to leaf spots caused by Bipolaris sorokiniana.

Fungicides can also be implicated in causing the potential for certain diseases to become greater, and therefore to demand a higher degree of energy to be expended on disease control measures. The common examples are of the benzimidazole-derivative fungicides (Tersan 1991, Cleary's 3336, Fungo) to sometimes increase the incidence or severity of red thread, rust, some leaf spot diseases, Pythium blight, and possibly the fairy rings. Those are examples of a phenomenon known as disease swapping. When a chemical is applied to control the growth of certain pathogens, there are likely to be present other pathogens which are insensitive to the fungicide. The insensitive pathogens were unapparent previously; perhaps their diseases were less noticeable or perhaps they did not have the right conditions for causing disease before the fungicide had been applied. This possibly could relate the the fungicide's ability to increase the potential for attack by some pathogens, by altering host plant resistance, or by killing saprophytic organisms which in some way limit the ability of pathogenic fungi to penetrate

into the plant. A recent occurrence of winter disease problems on a golf course may also be an example of disease swapping. A disease tentatively identified as Pythium root rot caused extensive damage on a number of golf greens that were treated with a large array of "snow mold" control fungicides during autumn. The fungicides which were used are not toxic to Pythium species. Extensive use of chemicals at this site may have predisposed the grass to problems that are generally not considered important.

The development of fungicide resistance among pathogenic fungi has gained considerable attention during the past decade. It is now well known that excessive reliance on some fungicides can lead to a decreased sensitivity to those chemicals in some components of the pathogen population. An entire branch of pest control science deals with this problem, and I will not go into the details in this paper. It is important to know, however, that fungal populations are very diverse. The application of any chemical selectively suppresses the growth of only those fungal strains that are sensitive to that chemical. Other strains that are less sensitive or insensitive may increase in dominance over time, and may therefore reduce the efficiency of that chemical. The insensitive strains may have been present at low proportions in the original fungal population, or may have resulted from mutations of the sensitive strains. Whatever the reason, the result of their new-found dominance in the population of a pathogen is the same--the disease is no longer controlled by a once-effective fungicide. This problem is reduced by maintaining diversity in your fungicide inventory. Use several materials that each control a given disease; this may be in the form of a tank mix or of alterations in their use during successive applications through the season. Although I cannot prove it, we also suspect that some instances of unconfirmed "dollar spot resistance" to fungicides such as Tersan 1991 or Chipco 26019 are actually expressions of erroneous identification procedures. Diseases which may in some instances mimic dollar spot include Pythium blight, Nigrospora blight, Fusarium leaf spot, Curvularia blight, copper spot, red thread, anthracnose, downy mildew (yellow tuft), and Fusarium patch. There are also several different fungi which cause the complex known as "dollar spot". We have no idea as to the relative differences in sensitivities to fungicides that may exist among these fungal pathogens.

WHY WASN'T THE GRASSES' RECOVERY FROM DISEASE RAPID AFTER I USED THE CORRECT CHEMICAL FOR A DISEASE WHICH WAS ALSO CORRECTLY IDENTIFIED?

The ability of fungicides to control a disease differs for each combination of disease, host grass, and fungicide. In all cases, however, it cannot be strictly assumed that control of a pathogen's activity will automatically cause the turfgrass to regain the state of health that the manager would like to see. The overall interaction of the plant with its environment determines the regrowth phase. If, for instance, a root-infecting pathogen caused a severe root rot during summer, the mere control of the pathogen's activity cannot bring life back to the affected plants, and the heat of summer is not conducive to root regeneration by cool-season grasses. In this instance additional applications of fungicides may serve only to cause the potential for additional problems addressed in other questions posed in this paper. Once the activity of the pathogen has been suppressed, the plants which have already been attacked, but which have not yet expressed visible

symptoms, might continue to die even though the pathogen is no longer active. Further death will be prevented only when all of the severely root-pruned plants have died, or when the weather changes so that conditions are less stressful to the plants.

The scenario described above is particularly true for diseases in which the root system is attacked before symptoms become visible in the foliage. Examples of diseases of this type include those caused by nematodes, and by root-infected species of Pythium, Rhizoctonia, Gaeumannomyces, Phialophora, and Leptosphaeria. The only way to ensure that long lasting damage from these pathogens will not occur is to use preventative control measures against them after they are first identified as being present and causing disease on a particular area. This is so because the symptoms of diseases caused by these pathogens appear whenever the rate of root dysfunction becomes greater than the rate of root regeneration, or whenever the degree of environmental stress increases above a critical limit for the amount of functional root volume on a particular plant. As such, a severely affected plant can remain symptomless as long as the plant is not subjected to high levels of stress from chemicals or the environment. Once stressed, however, the plants may die even though the pathogen did its damage months earlier. Applications of chemicals at the time of symptom development may serve no purpose at all, or may be beneficial in that they might encourage a somewhat earlier regrowth of plants in the autumn or spring. The point to be learned, however, is that a chemical application will not necessarily ensure that the grass will quickly re-attain its previous state of health.

The principles described above also apply to a lesser extent to diseases caused by root-, crown-, and basal stem-infecting species of Drechslera, Bipolaris and Fusarium. Once the melting-out or crown and root-rot stage of these diseases is attained, the application of fungicides will not generate a rapid reversal of the problem. Prevention of the initial infection, perhaps months earlier, is necessary to ensure freedom from plant death during mid-summer. This is not true, however, for pathogens which only infect the leaves. These diseases can be controlled readily after the first symptoms appear. Examples include dollar spot, rusts, red thread, Ascochyta leaf blight, powdery mildew, and related diseases. Although the existing lesions caused by these pathogens cannot be made healthy again, all regrowth can be protected from further infection, and the turfgrass stand can therefore grow out of the problems associated with foliar infections.

SHOULD I SELECT A FUNGICIDE FOR THE IMMEDIATE COST ADVANTAGE OR FOR THE LONG-TERM SAVINGS?

This question may or may not be only of philosophical importance. The turfgrass manager who operates on a very tight budget is obviously going to favor the short-term cost in selecting a chemical. However, when two or more materials have season-long costs (including application costs) that are equal and timing differences are not of great importance, one has to determine whether the long-term cost differences likely to be assoicated with the chemicals are important enough to override the immediate selection forces such as friendship with the sales people, purchasing ease, and others. The long-term costs may involve such factors as the potential for development of

resistance among the pathogen population, for development of abnormally high amounts of thatch, or for development of growth regulatory effects which may lead to increases in certain diseases, weeds, or insects. Many of these long-term effects are not clearly identified, but some are and that knowledge should be used in a turfgrass manager's purchasing process.

CONCLUSION

The problems addressed above are but a few of those involved in the development of fungicide programs. But of far greater importance is that the disease control program fits into the overall scheme of the turfgrass management system. This disease control approach has been of tremendous importance in the development of high quality turfgrasses, and that achievement should not be undermined by abuses of these materials. It is clear from the discussion above that fungicide applications are not always beneficial in the long run. The key to long-term efficiency in grass management is to ensure that all pest control and management procedures are effectively integrated. Thus lies the major thrust for research and implementation of future pest control strategies.

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