# NONTARGET EFFECTS OF PESTICIDES ON TURFGRASSES[1]

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The efficiency of pesticides for controlling specific pests is well known throughout the turfgrass industry. But turfgrass managers and scientists alike have very little information regarding the beneficial and deleterious effects of pesticides on turfgrass processes other than controlling pests. These side effects, or nontarget effects, continue to be one of the least understood aspects of pesticide use. We can be sure that some side effects do result from our use of pesticides and that the beneficial effects are likely to help offset the negative effects. It is also clear that if the beneficial effects can be identified and exploited and the deleterious effects minimized, the art of turfgrass management will become more soundly based and maintenance costs will be reduced to the minimum.

Our knowledge of nontarget effects of pesticides in the specialized turfgrass ecosystem is limited, but even the existing body of information has not been adequately extended to turfgrass managers. This paper highlights some effects of pesticides on turfgrass diseases and the results of my studies on the turfgrass characteristics influenced by fungicides.

## HERBICIDE-INDUCED INCREASES AND DECREASES IN DISEASES

Knowledge about the influences of herbicides on turfgrass diseases is meager. Recent reviews of these effects on other crops (1, 2, 4, 11) indicate that herbicides have the ability to suppress certain diseases and to increase others. Some investigators feel that weed control chemicals can affect diseases by altering (1) the virulence of certain pathogenic fungi, (2) the relationships between the pathogenic fungi and their parasites and/or competitors, or (3) the level of disease resistance in the grass. Papavizas and Lewis (11) concluded that the latter mechanism is the only one with equivocal supportive evidence at this time.

Engel and Callahan (6) demonstrated that growth of Kentucky bluegrass was affected by applications of several herbicides. They concluded that after a herbicde has been used, normal-appearing turfgrass foliage is not sufficient assurance of the chemical's safety. In their study, Betasan (bensulide) generally reduced root growth but Chipcal (calcium arsenate), Xytron (DMPA), Dacthal (DCPA), and some polychlorodicyclopentadiene (PDCP) herbicides did

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not. In contrast, all these herbicides reduced shoot production, some by very little and others by up to 33%. The growth suppressions were not visible but were nevertheless important. Karr et al (10) demonstrated that Betasan and Balan (benefin) slightly enhanced the severity of brown patch and dollar spot on bermudagrass and Pythium blight on perennial ryegrass but had no effect on Pythium blight on bermudagrass. Pythium blight was also unaffected by repeated applications of Zytron to turfgrass (2). Stripe smut and patch diseases of Kentucky bluegrass have been increased by applications of Bandane (PCDP), Chipcal, and linuron (1, 6, 19). Urea-derivative herbicides, such as linuron, have also enhanced powdery mildew and reduced eyespot of wheat (13).

Hodges (9) recently reported the effects of five herbicides on Helminthosporium leaf spot of Kentucky bluegrass. Disease was increased by applications of 2,4-D, 2,4,5-T, MCPP (mecoprop), and Banvel (dicamba) and decreased by 2,4,5-TP (silvex). These hormone like herbicides were considered to increase the leaf senescence rate and, subsequently, the ability of the pathogen to infect the dying leaves. In addition, 2,4-D can increase the severity of wheat and corn foliar diseases caused by Drechslera and Bipolaris species and reduce these diseases on barley (13). Take-all disease of wheat and barley has also been increased by application of MCPP but not of 2,4-D or MCPA (11). MCPP increased the production of perithecia, mycelia, and microconidia by the take-all pathogen. This fungus also causes take-all patch (Ophiobolus patch) of bentgrass, and closely related pathogens cause patch disease of bluegrasses (summer patch and necrotic ring spot) and bermudagrasses (spring dead spot).

Other examples of herbicide-induced increases or decreases in diseases of Gramineae are numerous, but many involve herbicides that are not generally used on turfgrasses. A reasonable conclusion from the few examples discussed here is that generalities regarding the effects of specific herbicide groups on individual diseases are not appropriate at present. Perhaps most important is that managers recognize the potential for nontarget effects and modify their maintenance programs based on their experience.

## THE EFFECTS OF INSECTICIDES AND NEMATICIDES ON DISEASES

If our knowledge about the effects of herbicides on turfgrass disease is meager, then that about the influences of insecticides and nematicides is rare. It is unlikely that this gap in our knowledge exists simply because there are few important examples of such interactions. The trend for scientists to work independently is perhaps a more likely reason for this void.

The work of Gould et al (8) at Puyallup, Washington, probably represents the best known insecticide-disease interaction. Chlordane was much more efficient than any of the fungicides tested for suppressing take-all patch of bentgrass. A more recent study at that location failed to confirm the earlier results but was conducted with longer intervals between applications and over a shorter period of time. Engel and Callahan (7) determined that chlordane increased the rooting capacity of Kentucky bluegrass but, like the herbicides studied, also suppressed leaf production.

Fairways on one New York golf course suffered for several years from

what appeared to be Curvularia blight of Poa annua. The disease control program before the first recognized occurrence of this disease was dominated by benzimidazole fungicides. Curative attempts with Tersan 1991 (benomyl) and several heavy metal fungicides were unsuccessful. By accident, the turf superintendent observed that fairway areas recovered quickly and totally after applications of Dursban (chlorpyrifos). The superintendent repeatedly demonstrated the phenomenon and then began to use the procedure for controlling the disease. The insecticide and its solvent were not toxic to the pathogen in our laboratory tests.

Although the reason for Dursban's success in this instance is not understood, the ability of insecticides and nematicides to enhance or to reduce diseases of other plants is fairly common (2, 11-13). Pesticides may act directly by influencing the capacity of the pathogens for growth or indirectly by altering host resistance or the balance between pathogenic fungi and other microorganisms. Beute and Benson (3) and Powell (2) have also emphasized that interactions between small soil fauna (insects and animals) and pathogenic fungi may be of considerable importance. Vargas (21) demonstrated the importance of nematodes feeding on predisposition of Kentucky bluegrass to Fusarium blight in Michigan. Possibly, predisposition to Curvularia blight on the golf course in New York is caused by the feeding activity of an unsuspected arthropod or nematode, although our tests seemingly ruled out the latter.

The examples here and in review papers (2, 3, 11-13) underscore the necessity for amplified research interest in the nontarget effects of insecticides on turfgrass diseases.

## INFLUENCE OF FUNGICIDES ON DISEASE PREVALENCE

A voluminous data bank is available to anyone wishing to determine the efficacy of specific fungicides for controlling diseases. The positive results from such research are thoroughly extended to turfgrass managers. Moreover, the fungicide package labels pertaining to ornamental turf list nearly all known efficient registered uses, because residue and related problems are few compared to those for food crops. Negative aspects of disease control studies are, however, communicated less frequently. Turfgrass workers can deduce which diseases a fungicide is unable to control efficiently by simply failing to find mention of the diseases on the product's label. But there remains a dearth of available information regarding instances where fungicides have increased the prevalence of diseases. This discussion will concentrate on that void.

Turfgrass managers periodically experience occasions when a fungicide allows a particular disease to become more severe or when a second disease occurs soon after a fungicide has been applied to control the initial, or target disease. These occurrences are not always recognizable on uniformly treated turfgrass areas. If recognized by a turf manager, they are not always brought to the attention of industry or public-sector scientists and extension personnel who could allocate resources to study the phenomena. Replicated and randomized research trials plus demonstration trials frequently reveal such examples. The fact that these results have not been summarized is a basis for concern.

During the past decade, over 90 examples of fungicide-induced increases in turfgrass diseases have been listed in Fungicide and Nematicide Tests, a publication of The American Phytopathological Society. These reports greatly underestimate actual occurrences because 1) most tests are based on single-season studies, 2) most tests are conducted on experiment station research plots where atypical use patterns exist, and 3) the publication presents the results of only a small proportion of the scientists and practitioners who conduct such studies. Detailed papers on this topic have also been published in Phytopathology, Plant Disease Reporter, Journal of the Sports Turf Research Institute, and other periodicals. Additional examples have been reviewed in turfgrass textbooks, and unpublished results of studies conducted in various states and countries are available to turfgrass scientists and pesticide manufacturers.

Benzimidazole-derivative fungicides, such as benomyl (Tersan 1991) and the thiophanates (Fungo, CL 3336), have been given considerable attention during the past decade. Quite early, these fungicides were recognized as not being toxic to oomycetes. The potential thus existed for Pythium blight to become amplified where the benzimidazoles were overly emphasized in a disease control program. This possibility was confirmed in studies by Warren et al (22).

These fungicides were also known to be nontoxic to most basidiomycetes and certain hyphomycetes. Scientists were little surprised, therefore, when benzimidazoles were established as also capable of amplifying diseases caused by fungi in these taxonomic groups. Such documentation is now available for Typhula blight, rusts, red thread, some Rhizoctonia diseases, and some Helminthosporium diseases. The ineffectual control of dollar spot by benzimidazoles in certain areas represents a special circumstance in which strains of the dollar spot fungi have undergone adaptive mutation or selection, and these tolerant strains have thereby gained dominance among this pathogen complex in turfgrass.

Turfgrass variety trials at Cornell University have revealed nontarget effects from fungicides on several occasions. Half of each cultivar plot is treated with fungicides to provide comparative quality observations with the untreated half. Although the common observation is that the fungicide-treated half is superior in quality to the untreated half, the opposite has been noted on several occasions. In 1980, red thread occurred on the perennial ryegrass and red fescue plots about 1 week after Tersan 1991 had been applied. The disease was significantly more severe on the treated half of the plot, and many of the ryegrass cultivars that were free from red thread on the untreated area were quite susceptible on the treated half. Burpee and colleagues (Fungicide and Nematicide Tests, 1978) also found that a benomyl-treated turf became more susceptible to red thread. Another basidiomycete-caused disease was observed in Cornell's Kentucky bluegrass cultivar trials. Again, the disease was greatest in the areas treated with fungicides and less active or absent in the untreated halves. The disease appears identical to that Smith et al (20) found occurring only on benomyl-treated bentgrasses in Australia. Circular patches of fluffy to mealy, white- to cream-colored mycelium caused considerable unsightliness and some premature leaf senescence but did not appear to infect the turf.

Tersan SP (chloroneb) and Actidione TGF (Cycloheximide) predisposed

creeping bentgrass on a New York golf course to a disease caused by Rhizoctonia cerealis. The cool-weather brown patch disease was present where these fungicides had been applied to prevent the anticipated occurrence of a snow mold complex consisting of Fusarium patch and Typhula blight, and was absent where the fungicides had not been used. Experimental confirmation was collected on ole of the untreated putting greens (Fungicide and Nematicide Tests, 1974). The June 1980 issue of Golf Course Management contains an article by A. D. Brede describing a number of similar nontarget effects of fungicides observed on the turfgrass plots at Pennsylvania State University and elsewhere.

## RESULTS OF ONE STUDY ON KENTUCKY BLUEGRASS

Influences of pesticides on the microflora of soil have been extensively studied and reviewed (2, 4, 5, 11, 13). Additional references for effects of fungicides on turfgrass ecosystems may be found among the literature cited in my publications (14-19). One of my studies at Cornell University is briefly described here.

A Kentucky bluegrass sod that had never been sprayed with any pesticide was purchased in 1975 and installed at our field research site. The underlying soil was a moderately well-drained silty clay loam. The new turf stand was marked into 66 (1 x 5 m) plots for the long-term investigation of 22 different pesticide treatments.

Fourteen fungicides were selected as representative of those likely to be used commercially on golf course putting greens (bluegrass was used for these tests because the nontreated controls would have died in our area if bentgrass had been used). These pesticides and no others (except single applications of 2,4-D during 1979 and 1980) were applied to designated plots nine times annually from 1975-82 the fungicides were applied at a 21 day intervals from April to September, except for nine applications of Terraclor 75 (quintozene, PCNB) and Koban (ethazole) made at weekly intervals during July and August. In addition, two drenches of Tersan 1991 and one of the nematicide Nemacur (fenamiphos) were applied annually to designated plots. Five other treatments were more typical of commercial programs than the repetitive applications of only one fungicide: two or three fungicides alternated so that any one material was applied at 42- or 93-day intervals or alternated as described and combined with midsummer treatments of Koban and Terraclor 75.

The first 2 years of study were devoted to establishing a well-documented, long-term fungicide "history" on the plots. The fungicides were grouped according to inhibition of thatch decomposition and acidifying characteristics from 1975 to 1977. Nonacidifying and nonthatching fungicides included Dyrene (anilazine), Captan (captan), Daconil 2787 (chlorothalonil), Actidione TGF (cycloheximide), Koban (ethazole), Terraclor 75 (quintozene, PCNB), and Actidione RZ (a combination of cycloheximide and quintozene). Nonacidifying but thatch-inducing chemicals included Cadminate (cadmium succinate), Nemacur (fenamiphos), and Chipco 26019 (iprodione). Fungicides that induced both thatch and acidity included Tersan 1991 (benomyl), Dithane M-45 (mancozeb), Tersan 75 (thiram), Bromosan (ethyl thiophanate plus thiram), and Duosan (methyl thiophanate plus maneb) and the programs in which these

fungicides were part of the rotation.

Some fungicides caused the soil immediately below the thatch:soil interface to become quite acidic, in spite of undissolved lime granules in the thatch (15). The lowest pH values occurred in plots treated with benzimidazole-containing fungicides, such as Tersan 1991, or with fungicides containing large amounts of sulfur, such as Dithane M-45 and Tersan 75. The largest amount of acidity occurred in plots treated with combinations of these fungicides (Bromosan and Duosan). Acidification was measured to a depth of 20 cm in some plots. The reasons for this acidification are unclear.

Root masses were altered by only a few fungicides, the most notable being a threefold amplification of rooting by Chipco 26019. This fungicide also increased the mass of leaf clippings in the spring but not in the summer. The overall quality of bluegrass was increased by all fungicides and was attributable mostly to increases in shoot density. The nematicide Nemacur greatly improved turfgrass quality without increasing root mass. Populations of pathogenic nematodes were about 2,500/100 $cm^3$ in the control plots, and only 10/100 $cm^3$ in the Nemacur-treated plots.

When the sod was installed in 1975, the depth of its thatch was 2 cm. The thatch depth in the untreated control had decreased to 6 mm by late 1977 but, in some treated areas, the thatch depth had not decreased at all (15). A detailed study of the thatch biology was initiated on this plot. The treatments did not greatly alter the total numbers of micro-organisms (16, 17) but did cause considerable shifts in compositions of species within each microbial genus. To determine if the thatch depth was being influenced by fungicide effects on the decomposer microorganisms, a litter decomposition rate study was performed. Decomposition rates for cellulose and natural thatch, enclosed in thin 10 x 10 cm nylon bags and buried at 2 cm depth in the turf plots, were not significantly reduced by the fungicide treatments. Since it was concluded that the thatch-inducing fungicides did not reduce the litter decomposition rate, it was felt that the only other possibility was that they must be increasing the rate of plant tissue production. This process was evaluated and found to be true. Thatch depths were more related to amounts of root and rhizome production than to leaf growth or microbial decomposition rates. Additional studies with more fungicides and fewer application numbers per year are in progress.

The fungicides also caused considerable variation in the severity of several nontarget diseases. Some interesting comparisons could be made among sets of data. For example, Fusarium blight was controlled by several chemicals (Cadminate, Daconil, Dithane M-45, and Nemacur) that have no suppressive effect on the disease when these chemicals are used in single-season preventative studies. The thatch decomposition rate and plant growth parameters were more associated with the occurrence of Fusarium blight than were any of the known attributes of the fungicides (18).

Much additional research is necessary to determine the undoubtedly different nontarget effects that may or may not occur on various turfgrass general grown in other areas, on various soil types, and under limitless variations in management programs.

## FUTURE NEEDS

That pesticides can exert many effects on nontarget organisms and processes in turfgrasses is readily apparent. In addition to direct effects, each chemical and biological change may cause secondary, tertiary, and other changes until the entire management program becomes improved or hindered by the use of certain pesticides. The effects may be so slight as to be unnoticeable but large enough to increase expenses for certain management procedures. It can be theorized that frequent use of certain pesticides does alter the long-term costs of such management procedures as controlling pests, thatch, and soil acidity. These nontarget effects need greater attention in the original decision-making process. If, for instance, four fungicides were known to be almost equally effective against a target pathogen but three were much more likely to increase thatchiness or weediness, the means of selection could be improved. The long-term costs of thatch and weed control are certainly greater than the immediate cost differences among competitively priced fungicides. Although product costs, application costs, technical services provided, immediate availability of a product, and personal preferences are very important considerations, it is also important for scientists to provide additional facts on which to base pesticide-use decisions.

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