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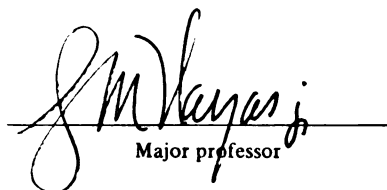
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CHARACTERIZATION OF
DEMETHYLATION INHIBITOR (DMI) RESISTANT
SCLEROTINIA HOMOEOCARPA

presented by

ROBERT CRAIG GOLEMBIEWSKI

has been accepted towards fulfillment
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M.S. degree in BOTANY & PLANT PATHOLOGY


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**CHARACTERIZATION OF
DEMETHYLATION INHIBITOR (DMI) RESISTANT
*SCLEROTINIA HOMOEOCARPA***

By

ROBERT CRAIG GOLEMBIEWSKI

A THESIS

**Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of**

MASTER OF SCIENCE

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ABSTRACT

CHARACTERIZATION OF DEMETHYLATION INHIBITOR (DMI) RESISTANT *SCLEROTINIA HOMOEOCARPA*

By

Robert Craig Golembiewski

Sclerotinia homoeocarpa strains were isolated from several golf courses where demethylation inhibitor (DMI) fungicides had been reported to give little or no dollar spot management. Relative growths for 50 *S. homoeocarpa* isolates, from each of 6 golf courses, were determined for 4 DMI fungicide concentrations based on radial mycelial growth. Isolates collected from golf courses where DMI fungicides were used on a regular basis had higher relative growths than isolates from golf courses where DMI fungicides had never been used.

In field studies on a golf course in southeast Michigan, recommended label rates of the DMI fungicides failed to manage dollar spot. The contact fungicides were the only treatments in the study to provide acceptable disease management. Population analysis of the individual field plots confirmed that DMI resistant strains of *S. homoeocarpa* were environmentally fit and did persist. *S. homoeocarpa* populations continued to shift towards resistance in the presence of the DMI fungicides.

**To my parents
Robert and Mary Golembiewski,
for all their love and support.**

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Dollar spot, caused by *Sclerotinia homoeocarpa* is a common and persistent turfgrass disease that occurs on most turfgrass species throughout the world (53). Golf course superintendents spend more money on fungicides to manage dollar spot than on any other turfgrass disease (55). Superintendents are now confronting one of the most significant problems in the area of dollar spot management, resistance to fungicides. Fungicides are an essential part of dollar spot management and therefore, effective control requires management of fungicide resistance in *S. homoeocarpa* populations. This will help ensure the longest possible useful life of these plant disease control agents.

Resistance is the ability of an organism to exclude or overcome, completely or in some degree, the effect of a fungicide or other damaging factor (2). Dollar spot resistance to fungicides is not uncommon, as cadmium and anilazine-resistant strains were first reported in 1968 (6). This was not considered a major problem for two reasons: 1) resistance was not widespread and 2) the development of the systemic fungicides in the late 1960's were thought to be the new solution in chemical disease management.

Three of the most effective groups of systemic fungicides available for

turfgrass disease control are: the benzimidazoles, the dicarboximides, and the demethylation inhibitors (DMIs). These highly effective fungicides are constantly eliminating sensitive strains from the fungal population and selecting for resistant ones. Pathogen populations shift towards resistance under the selective pressure from repeated fungicide applications (18). Cross-resistance is common within these groups so any pathogen population that is resistant to one fungicide within a group is usually cross-resistant to other members of that group (17).

Turfgrass managers have already experienced dollar spot resistance to the systemic benzimidazole (59) and dicarboximide fungicides (20). There has always been new chemistry to replace obsolete fungicides, but this option is becoming very limiting. The rising costs of research and development and the need to ensure that products are environmentally safe have slowed down the development of new fungicidal chemistry. The demethylation inhibitors are the last new class of fungicides developed for dollar spot control. It was believed there was very little chance of resistance developing to this chemistry. However, our research indicates strains of dollar spot now exist which are resistant to the demethylation inhibitor fungicides.

DOLLAR SPOT

Causal Agent. Dollar spot is caused by the fungus *Sclerotinia homoeocarpa* F. T. Bennett (53). Taxonomists have excluded the fungus which causes dollar spot from the genus *Sclerotinia* because the fungus produces a flat stroma rather

than a sclerotium. Reclassification of the fungus into the correct genus has not been possible, although it is believed that the pathogen belongs in either the genus *Lanzia* Sacc. or *Moellerodiscus* Henn (34, 44). Unfortunately, *S. homoeocarpa* is still widely accepted as the causal agent of dollar spot since reclassification has not been possible.

The dollar spot fungus can be identified in laboratory cultures by a characteristic mat of fast-growing, fluffy white mycelium that becomes feltlike, with variable shades of olive, gray, yellow, or brown as it ages (53).

Symptoms. On closely mowed turf such as golf greens, diseased areas are small and somewhat sunken spots ranging from 1 to 3 inches in diameter. The descriptive name "dollar spot" was given because the familiar spots on golf greens resembled a silver dollar. The grass may be killed and the spot may become depressed if the disease continues to develop. If the disease remains unchecked, the spots may coalesce to form large areas of blighted grass. Affected areas on coarser-textured turf are typically larger and more irregularly shaped, ranging from 4 to 6 inches in diameter (41, 53, 55).

Dollar spot symptoms on individual grass blades appear as lesions which initially become chlorotic, water soaked, and finally a tan or bleached color upon drying (53). Reddish-brown bands are present at the outer edge of the leaf lesions in bentgrass, Kentucky bluegrass, fine-leaf fescue, zoysiagrass, and bermudagrass. The reddish-brown banding does not occur on annual bluegrass (55). On finer-

textured turfgrasses, individual lesions on leaves span the width of the leaf blade with a constricted appearance resembling an hourglass. Lesions on coarser leaves appear as restricted spots on the leaf blade. Leaf symptoms may be confused with those caused by red thread, copper spot, brown patch, and Pythium blight (53).

A white, cottony or cobwebby growth of aerial mycelium often is visible during the early morning hours while the grass is still wet and the pathogen is active. The mycelium disappears as the leaves dry (53, 55).

Disease Cycle. Fungi causing the dollar spot disease survive as dormant mycelium in previously infected tissue. The disease is spread primarily by mowers, maintenance equipment, and people carrying the mycelium and infected plant tissue. The mycelium rapidly invades healthy leaf tissue and extends into moisture-saturated air spaces surrounding infected leaves when the turfgrass environment favors activity of the fungus. Mycelial infection frequently occurs through cut ends of leaves or through stomates on leaf blades (53, 55).

Epidemiology. Dollar spot is active during the late spring to late autumn when environmental conditions favor long periods of high humidity or free moisture within the turfgrass canopy. Outbreaks of the disease typically follow periods of heavy dew deposition on turfgrasses during the night. Dollar spot generally occurs when the temperature ranges from 60° to 90° F (53, 55). The disease incidence tends to be greater on turfgrasses maintained with low soil

moisture and low nitrogen fertility (7, 53).

DOLLAR SPOT MANAGEMENT

Cultural. The incidence and severity of dollar spot can be significantly reduced by maintaining adequate nitrogen levels and soil moisture levels. Therefore, the turf area should be kept near field capacity along with light and frequent applications of nitrogen for the best dollar spot management (53, 55).

Mycelium growth and infection on the surface of the turfgrass plants occurs as long as dew is present. Some commonly used practices to remove this dew include: irrigating lightly, whipping with a bamboo pole, or dragging a hose over the area. Good air movement and reduced humidity should be promoted by clearing barriers such as unwanted vegetation and by relocating desirable plants (41, 53, 55).

Genetic. There are no known creeping bentgrass cultivars that are resistant to dollar spot.

Biological. Satisfactory disease suppression during years of light to moderate dollar spot infection has been shown with composted materials like Ringers Compost Plus and a bacterium called *Enterobacter cloacae* (42, 43). However, during years of heavy infection, fungicide treatments are necessary to obtain good disease management.

Chemical. The most efficient dollar spot management can be obtained when good cultural practices are combined with a sound fungicide program. Both

contact and systemic fungicides can be used for dollar spot control, however, the systemic fungicides are preferred by turfgrass managers because of their efficacy and length of disease suppression. The contact fungicides used for dollar spot management include chlorothalonil and anilazine. Recommended systemic fungicides include: iprodione, vinclozolin, fenarimol, propiconazole, and triadimefon (55).

GENETICS AND POPULATION DYNAMICS

Introduction. Fungi may acquire fungicide resistance through either a disruptive selection (qualitative response) or a directional selection (quantitative response) (35). The difference in resistance development can be explained by different types of selection leading to the buildup of resistant populations. The key in both situations is that the fungus must possess the genetic potential for overcoming the fungicide toxicity (26).

Disruptive Selection (Qualitative Response). The sensitivity distribution of a target population with major-gene control for resistance is discontinuous and the response to selection is qualitative. The resistant members comprise a distinct subpopulation which does not overlap with the sensitive population. The application of a fungicide which is 100% effective against the sensitive population will usually not effect the resistant subpopulation and therefore, the resistant subpopulation increases. The selection proceeds unnoticed in terms of chemical efficacy due to the discontinuity of the sensitivity distribution. The target

population is perceived as sensitive until the resistant subpopulation reaches a detectable level, but by this time the population will be nearly 100% resistant with one or two additional fungicide applications. This results in sudden disease control failure and increasing the rate of application will not improve disease control (26).

Benzimidazole fungicide resistance is the classic example of major-gene resistance and qualitative population responses (25).

Directional Selection (Quantitative Response). The sensitivity distribution of the target population in polygenic resistance is continuous with the overlapping of genotypic classes. The continuity is maintained even after long fungicide exposure of the target population. A gradual, quantitative shifting of the whole distribution towards decreased sensitivity will occur and can be quantified by measuring the ED_{50} at different times after the onset of selection. Disease management may be affected depending on the initial position of the fungal population and the extent of the shift, however, complete loss of fungicide efficacy is infrequent. The quantitative response obtained with polygenic control provides indications of reduced performance (shortened periods of control) before complete failures. Increasing the fungicide rate or shortening the spray interval may improve the level of disease management with quantitative response resistance (26).

A few early examples of quantitative responses have been provided by the work on resistance to dodine in *Venturia inaequalis* (27) and to DMIs in *E.*

graminis f. sp. *hordei* (5, 60) and *Sphaerotheca fuliginea* (50).

BENZIMIDAZOLE FUNGICIDES

History. The benzimidazole fungicides were first introduced for plant disease control in the late 1960's. They were the first practical systemic fungicides that controlled a wide spectrum of pathogens on many plant species (16). Compounds included in this group include benomyl, carbendazim, fuberidazole, thiabendazole, thiophanate-ethyl, and thiophanate-methyl (Figure 1)(54). These compounds represented a class of fungicides with unique and very advantageous properties (11, 16, 19), especially when compared to the protectants which were the dominant, traditional fungicides utilized at that time.

The benzimidazoles first unique attribute was their single-site mode of action. The inhibition of tubulin polymerization resulted in mitotic arrest (9), a reduction in rate of linear growth, and change in cell shape (10, 32). This mode of action permitted differential selectivity between the fungus and host to allow plant safety with a systemic molecule (8, 16).

The attractive fungicidal properties of the benzimidazoles included highly effective, low use rates and postinfection action which extended application intervals. Growers often relied on the benzimidazoles as their sole disease management agent as a result of the fungicidal properties (14, 15).

Resistance was reported to a number of pathogens as a result of the intensive use of the benzimidazole fungicides after only a few years. The primary

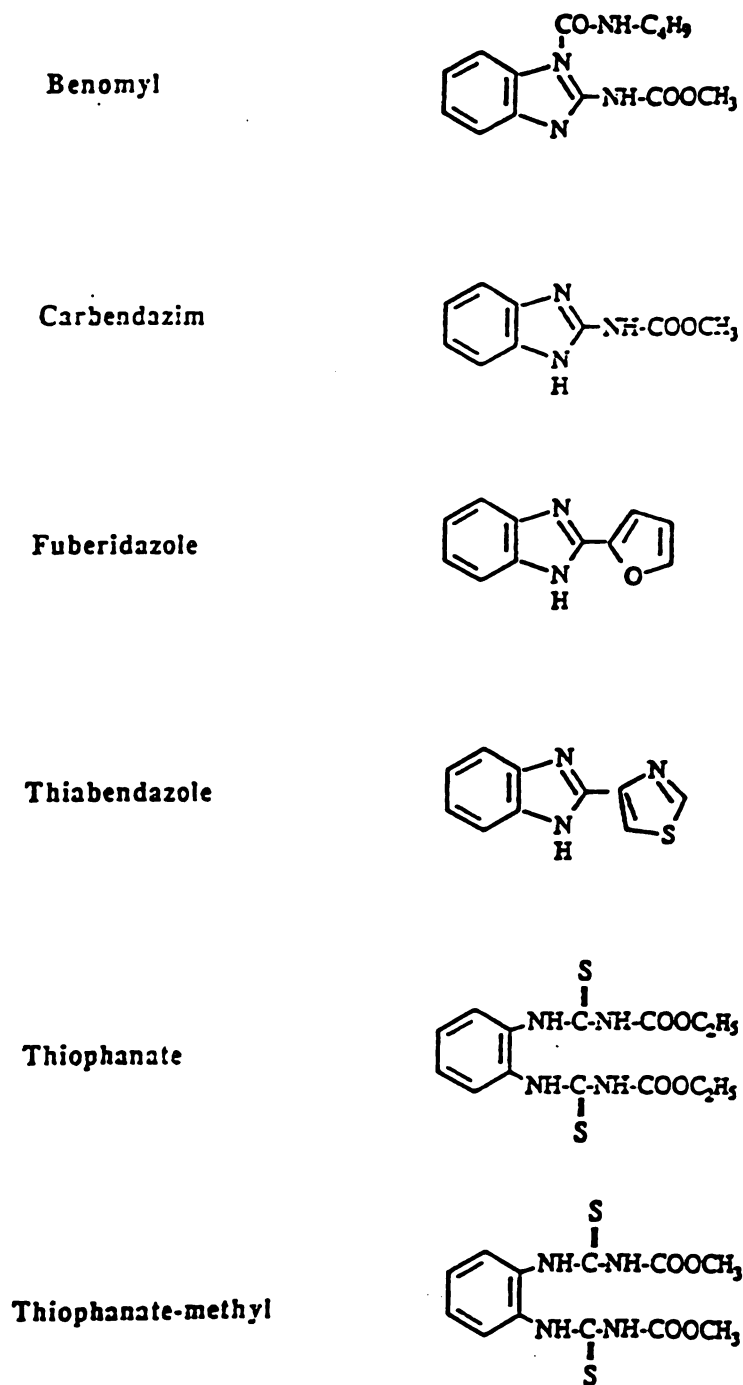


Figure 1. Chemical structures of the benzimidazole fungicides.

mechanism of resistance in pathogens appears to be a mutation in the B-tubulin gene that results in a loss of affinity of tubulin for benzimidazoles. Cross-resistance among the benzimidazoles usually results when a pathogen develops resistance to one of the benzimidazole fungicides (52).

Dollar Spot Resistance. The development of the systemic benzimidazole fungicides provided turfgrass managers with a new option in chemical disease management. They were found to be very effective against many turfgrass diseases, especially dollar spot. The benzimidazoles rapidly began replacing the heavy metal fungicides for dollar spot management. In 1972, isolates of *S. homoeocarpa* were collected from several locations in the eastern and midwestern United States where benomyl had failed to control dollar spot on turfgrass. These isolates were 100 times as tolerant to benomyl in vitro as isolates from areas where no control difficulties had been experienced (28). The protectant fungicides chlorothalonil and anilazine were recommended as replacements for benzimidazoles in areas where management failure had occurred (59). The benzimidazole-resistant strains were determined to have excellent fitness and pathogenic aggressiveness, and consequently persisted.

Benzimidazole resistant strains have persisted for more than 20 years on some golf courses even though those fungicides were no longer used. Resistance of dollar spot to the benzimidazole fungicides is so widespread that it is difficult to find a golf course in the U.S. today where it is not the dominant strain. Many

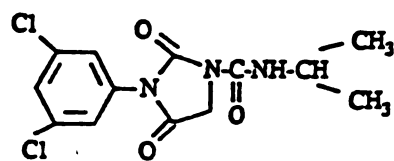
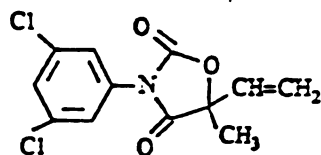
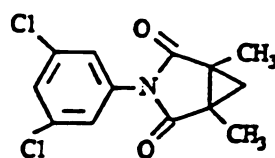
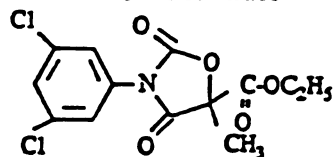
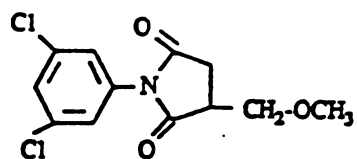
people consider the new, wild-type strain of *S. homoeocarpa* to be the one that has resistance to benomyl, thiophanate-ethyl, and thiophanate-methyl (57). The benzimidazoles represent, most dramatically, the beginning of serious resistance problems to fungicides (14, 16).

Turfgrass managers faced a setback with the development of dollar spot resistance to the benzimidazole fungicides. The resistance to the benzimidazoles pointed out the critical need for better fungicide management to prevent resistance from occurring to a new class of systemic fungicides known as the dicarboximides.

DICARBOXIMIDE FUNGICIDES

History. The fungicidal activity of the dicarboximide fungicides was first reported in 1971 (24), however the first marketable dicarboximides did not appear until 1974. The three new compounds (Figure 2)(39) introduced on the market and used worldwide were: iprodione (37), vinclozolin (46), and procymidone (29). Two other compounds (39) developed into products a few years later included chlozolate (21) and metomeclan (58). The dicarboximides were shown to inhibit spore germination and mycelial growth (30, 37, 46). They were also found at certain concentrations to cause swelling and bursting of the germ tubes and hyphal cells followed by extrusion of the cytoplasm (1, 23).

The primary pathogens against which the dicarboximides are used are *Botrytis*, *Sclerotinia*, and *Monilinia* species (45), however, the degree and spectrum of activity varies among the compounds due to their respective chemical

Iprodione**Vinclozolin****Procymidone****Chlozolate****Metomeclan****Figure 2.** Chemical structures of the dicarboximide fungicides.

structures. The dicarboximide fungicides replaced the benzimidazoles where resistance had become a problem. They were from the outset intensively, if not exclusively, used for disease control because of their excellent activity. Unfortunately, the first case of dicarboximide resistance was reported in a vineyard in West Germany exactly three years after the first registration of the dicarboximides (31). It was also determined that cross-resistance existed among the dicarboximide fungicides regardless of the compound used to induce resistance (45).

Dollar Spot Resistance. The dicarboximide fungicide iprodione was shown to be very efficacious on turfgrass for management of a number of diseases, including dollar spot (47). Turfgrass managers began incorporating iprodione into their fungicide spray program for dollar spot management, especially since the occurrence of benzimidazole resistance was so widespread. Iprodione played an important role in spray programs where benzimidazole resistant strains were not present to forestall benzimidazole resistance in *S. homoeocarpa*.

In 1981, Detweiler et al. (20) isolated an iprodione/benzimidazole-resistant strain of *S. homoeocarpa* from a golf course in southern Michigan where iprodione and the benzimidazoles failed to manage dollar spot. They concluded that the failure of iprodione and the benzimidazoles to manage dollar spot was preceded by the selection of benzimidazole-resistant *S. homoeocarpa* strains through the use of benzimidazole fungicides and then, by further selection of dicarboximide-

resistant strains through the use of iprodione. Turfgrass managers were no longer facing strains of *S. homoeocarpa* resistant to just the benzimidazole fungicides, but multi-resistant strains of *S. homoeocarpa* resistant to both the benzimidazole and dicarboximide chemistry.

Isolations taken from the golf course with the multi-resistant strains one year later, after dicarboximide fungicide use was discontinued, showed that the multi-resistant strain had disappeared and only the benzimidazole-resistant strain remained. Dicarboximide resistance appears more likely to occur in strains already resistant to another fungicide group, but strains of *S. homoeocarpa* that are multiresistant do not tend to persist in the turf once the use of the dicarboximide fungicides has been discontinued (56). This lack of persistence has also been observed with dicarboximide resistant strains of *Botrytis cinera* (38, 40).

Dicarboximide resistance by *S. homoeocarpa* is quite different when compared to the benzimidazole resistance. It has certainly not been as widespread, and when it does occur the resistant strains disappear from the population once the fungicides are no longer used. Turfgrass managers have realized that the successful management of dollar spot resistance will require not only the continued use of contact fungicides, but a much greater need for diversity of action mechanisms among the systemic fungicides.

DEMETHYLATION INHIBITOR (DMI) FUNGICIDES

History. The newest class of systemic fungicides to appear on the market

are the demethylation inhibitors (DMIs), which are classified as a subgroup of the sterol biosynthesis inhibitors (SBIs). They were first patented as compounds with fungicidal properties in the late 1960's, but it was not till a breakthrough in the 1970s when compounds were developed with the capability of managing a broad spectrum of diseases. All DMI fungicides have a common site of action within the fungal sterol biosynthesis pathway. They inhibit demethylation at position 14 of lanosterol or 24-methylene dihydrolanosterol, which are precursors of sterols in fungi (Figure 3)(48). The primary site of action of the DMI fungicides is multigenic (56). The DMIs include fungicides classified as pyrimidines, piperazines, imidazoles, triazoles, or pyridines (Figure 4)(35, 48, 49). The DMI fungicides as a group control a wide spectrum of pathogens represented by all major mycological groups except the Oomycetes. More than 25 different compounds are classified as DMIs and used for control of fungal pathogens of plants and animals. The sterol biosynthetic pathway has proved to be an extremely productive target for the development of effective fungicides and still offers good prospects for the development of new fungicide groups that block at other sites along the pathway (52).

Fungal Resistance to DMIs. Dekker (12) in 1982 gave the prognosis that the risk of resistance to DMIs was relatively low based on laboratory results and information gained from the use of DMI fungicides under field conditions. Despite the prognosis, the first case of DMI resistance was observed in the field

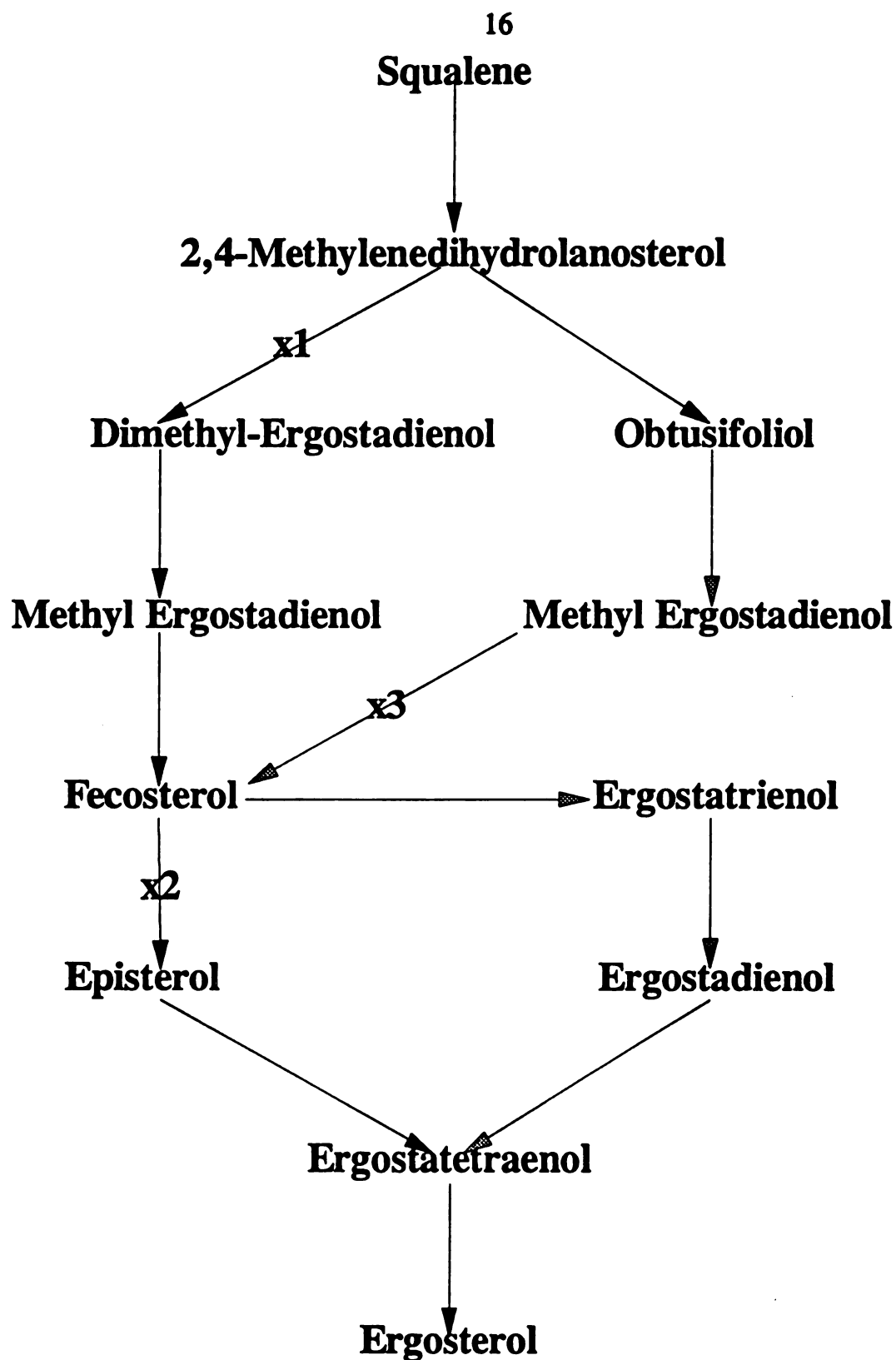


Figure 3. A simplified scheme for ergosterol synthesis and the places where DMI fungicides cause disruption.

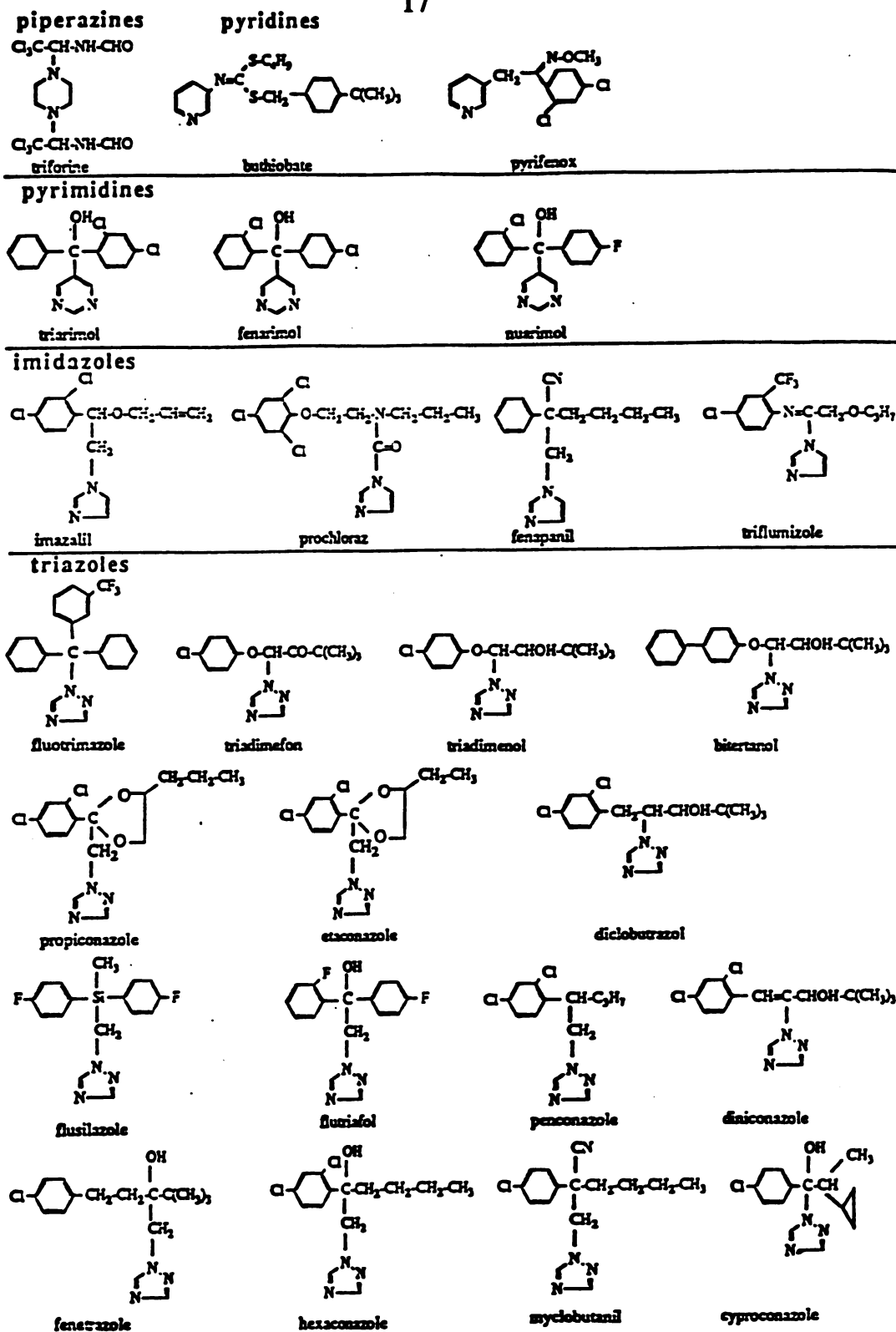


Figure 4. Chemical structures of the DMI fungicides.

and commercial greenhouses to powdery mildew (*Sphaerotheca fuliginea*) on cucumbers in the Mediterranean countries where DMI fungicides were used very intensively for several years (33). Other reported cases of resistance include powdery mildew of cereals in Europe (51, 61) and green mold of citrus in California (22).

DMIs clearly belong to the group of site-specific fungicides, which are in general more prone to resistance than conventional multisite inhibitors (13). It is difficult to determine management failure of the DMI fungicides from other factors like low fertility or heavy disease pressure because DMI resistance develops slowly and in small steps through directional selection (36). This type of selection seems to be related to the genetics, and ultimately, to the mechanism of resistance (36). The biochemistry of resistance has not been studied in great detail; compared to the comprehensive understanding of the mode of action of DMIs.

Field populations of many pathogens probably contain sufficient variation in sensitivity to DMI fungicides to enable resistance problems to develop. Despite early predictions that resistance would not be a problem with the DMI fungicides, instances of resistance to this group of fungicides are indeed increasing (3).

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CHAPTER II

RESISTANCE OF *SCLEROTINIA HOMOEOCARPA* TO THE STEROL DEMETHYLATION INHIBITOR FUNGICIDES

ABSTRACT

Isolates of *Sclerotinia homoeocarpa* were collected from three golf courses where sterol demethylation inhibitor fungicides (DMIs) were never used and from three golf courses where DMIs were being used but with little or no control of dollar spot. The mean relative growth value for the 150 isolates collected from golf courses treated with DMIs was 2-3 times higher than for the 150 isolates collected from golf courses with no DMI treatments. Isolates with high relative growth values for triadimefon also had high relative growth values for fenarimol and propiconazole. The significant difference in the sensitivity of the *S. homoeocarpa* population from courses 1-3 compared to courses 4-6 to DMI fungicides on amended medium indicates that resistance to DMI fungicides has developed in this important fungus on golf courses in Michigan and Ohio. Triadimefon, fenarimol, and propiconazole failed to provide acceptable dollar spot management on a golf course in southeast Michigan with a population of DMI-resistant *S. homoeocarpa*. The contact fungicide chlorothalonil applied at 191 ml/100 m² was the only treatment to provide excellent disease management during

all three (1991-1993) field seasons. An established concentration of 2 $\mu\text{g ml}^{-1}$ triadimefon proved to be an effective dose for the monitoring of *S. homoeocarpa* population shifts. Population analysis of individual field plots demonstrated that when the *S. homoeocarpa* population was exposed to the DMI fungicides applied alone and in combination with a different fungicide chemistry, a population shift towards resistance resulted.

INTRODUCTION

Sclerotinia homoeocarpa F. T. Bennett (9), the causal agent of dollar spot, is a widely distributed and destructive pathogen of turfgrass in the United States (14). The sterol demethylation inhibitor fungicides (DMIs) became available in Michigan beginning in 1979 to control dollar spot. Usage of the DMIs increased rapidly on turfgrass areas where benzimidazole-resistant and/or dicarboximide-resistant strains of *S. homoeocarpa* (3, 15) developed.

The DMI fungicides were used extensively and provided excellent control of dollar spot on golf courses in Michigan through the 1980s, but in 1990, reports of reduced control were received from a few golf course superintendents. In 1991, fairways on two golf courses in southern Michigan and one in northern Ohio treated with DMI fungicides exhibited serious outbreaks of dollar spot disease.

The present study was conducted to determine the sensitivities of populations of *S. homoeocarpa* from six golf courses to three DMI fungicides and to establish the efficacy of these DMI fungicides for the control of dollar spot at

a golf course where triadimefon-resistant strains were detected.

MATERIALS AND METHODS

Baseline sensitivity. The golf courses were selected for study based on the history of DMI fungicides used for disease management. Golf courses 1 through 3, located in Michigan near Warren, Webberville, and Williamston, had never been treated with DMI fungicides. Golf courses 4 through 6, located near Toledo, OH, Grosse Pointe, MI, and Southfield, MI, had been treated with DMI fungicides for several seasons including 1991. Superintendents at courses 4-6 had noted poor control of dollar spot with DMI fungicides beginning in 1990.

Leaf blades of creeping bentgrass (*Agrostis stolonifera*) and/or annual bluegrass (*Poa annua*) with distinct dollar spot lesions were collected with a 2-cm diameter soil probe on 12, 13, and 26 September 1991. Samples were taken from five fairways at each of the six golf courses. Each blade was placed on potato-dextrose agar (PDA; Difco Laboratories, Detroit, MI) amended with 20 $\mu\text{g ml}^{-1}$ of tetracycline and streptomycin sulfate. After the plates were incubated for at least 48 h at 21°C, single 7-mm-diameter agar plugs with mycelium of *S. homoeocarpa* were taken from an area adjacent to each blade of grass and inverted onto PDA. A total of 50 isolates was collected from each golf course.

The sensitivity of the 300 isolates of *S. homoeocarpa* to triadimefon, fenarimol, and propiconazole was determined. Triadimefon (Bayleton 25% DF) was obtained from Miles Inc., Kansas City, MO.; fenarimol (Rubigan 1 AS) from

Dow Elanco Products Co., Indianapolis, IN; and propiconazole (Banner 1.1 EC) from Ciba Co., Greensboro, NC. A 7-mm diameter agar plug was cut and transferred from 1-week-old colonies on PDA to plates containing 20 ml PDA amended with 0, 0.01, 0.05, 0.1, and 0.5 $\mu\text{g ml}^{-1}$ triadimefon or fenarimol, or 0, 0.01, 0.05, 0.1, and 0.2 $\mu\text{g ml}^{-1}$ propiconazole. The fungicides were dissolved in acetone and added to molten PDA (60°C) after sterilization. The final acetone concentration was 0.1% (v/v) in all treatment and control media. The colony diameter (minus the diameter of the inoculation plug) was determined after incubation for 4 days at 21°C. Only one isolate was tested per petri dish. The results were expressed as the relative growth (colony diameter on DMI-amended medium divided by the diameter on unamended medium X 100) for each isolate. ED₅₀ values were calculated by regressing the relative growth against the log of the fungicide concentration. The experiment was replicated three times. Chi-square analysis was used to analyze the frequency distributions based on relative growth for isolates of *S. homoeocarpa* from the six golf courses.

Field trials. The efficacy of triadimefon, fenarimol, and propiconazole for the control of dollar spot was tested over three field seasons (1991-1993) on a fairway at golf course 5 near Grosse Pointe, MI. In addition, iprodione and chlorothalonil were tested alone and in combination with triadimefon. Iprodione (Chipco 26019 2 FL) was obtained from Rhone-Poulenc Ag Co., Research Triangle Park, NC and chlorothalonil (Daconil 2787 4.17 FL) from ISK Biotech

Co., Mentor, OH. The fairway, containing a mixture of annual bluegrass and creeping bentgrass, was irrigated daily, fertilized with 0.12 kg N/100 m² every 14 days, and mowed at a height of 0.1 cm.

The treatments were applied to 1.8 x 1.8 m plots replicated three times in a randomized block design. Each fungicide was applied with a CO₂ sprayer. Treatments were initiated on 28 August 1991, 21 July 1992, and 22 July 1993, with subsequent applications being made at 10 and 21 day intervals. Treatments on a 10-day interval were sprayed 4, 7, and 6 times, those on a 21-day interval 2, 4, and 3 times, in 1991, 1992, and 1993, respectively. Dollar spot ratings were taken on 16 September 1991, 1 October 1992, and 19 September 1993 when disease severity in the control treatment was highest. A 0 to 9 scale, where 0 = 0 to 9% and 9 = 90 to 100% of the plot area with symptoms of dollar spot was used to rate each plot. The ratings were subjected to a square root transformation prior to analysis of variance (12). Treatment means were separated using Tukey's Honestly Significant Difference (HSD) test.

Population sensitivity. The sensitivity of *S. homoeocarpa* to triadimefon was determined prior to the first fungicide application in 1991 and again about 4 wk after the last fungicide application in 1993 as described above. Ten leaf blades infected with dollar spot were collected at random with the soil probe on 21 July 1992 and 15 October 1993 from each fungicide plot. Colony growth of each isolate on PDA amended with 0 and 2 µg ml⁻¹ triadimefon was evaluated after 4

days at 21°C. Differences in mean relative growth values for each treatment were detected using Tukey's Honestly Significant Difference test.

RESULTS

Baseline sensitivity. ED₅₀ values determined for isolates from golf courses 1-3 and 4-6 were 0.12 and >0.5 µg ml⁻¹ for triadimefon, 0.03 and 0.26 µg ml⁻¹ for fenarimol, and 0.002 and 0.103 µg ml⁻¹ for propiconazole, respectively. The relative growth at the highest concentration tested of the isolates of *S. homoeocarpa* from golf courses 1-3 were 2-3 times lower, depending on the DMI fungicide, than the relative growth of the isolates from golf courses 4-6 (Table 1). Isolates that grew well on PDA amended with triadimefon also grew well on PDA amended with fenarimol and propiconazole. Chi-square analysis of the frequency distribution data for each fungicide indicated that isolates from courses 1-3 produced significantly ($P < 0.001$) less growth on fungicide amended media than isolates from courses 4-6 (Figure 1). When representative isolates from golf courses 1-3 and 4-6 were tested on PDA amended with 1 and 2 µg ml⁻¹ triadimefon, growth of isolates from courses 4-6 was 4.7 and 7.8 fold greater, respectively, than the growth of isolates from golf courses 1-3 (Table 2).

Field trials. Disease pressure was higher in 1991 than in 1992 and 1993. The severity of dollar spot in plots treated with triadimefon, fenarimol, and propiconazole was reduced over the control in 1992 but not in 1991 and 1993 (Table 3). The severity of dollar spot in plots treated with iprodione alone and in

Table 1. Sensitivity to three sterol demethylation inhibiting (DMI) fungicides of isolates of *Sclerotinia homoeocarpa* from three golf courses (1-3) where DMIs were never used and three courses (4-6) where DMIs were used extensively.

Fungicide and concentration in the amended medium	Golf course codes	Relative growth ^a	
		Mean ^b	Range
Triadimefon 0.5 $\mu\text{g ml}^{-1}$	1-3	29	17-40
	4-6	61	27-85
Fenarimol 0.5 $\mu\text{g ml}^{-1}$	1-3	17	7-38
	4-6	42	20-57
Propiconazole 0.2 $\mu\text{g ml}^{-1}$	1-3	11	3-36
	4-6	38	10-64

^aColony diameter on amended medium divided by the corresponding diameter on unamended medium x 100.

^bEach value is the average of 50 isolates from each of the three golf courses (150 isolates total).

Figure 1. Frequency distributions of relative growths for isolates of *Sclerotinia homoeocarpa* sampled from six golf courses and tested on media amended with three sterol demethylation inhibitor fungicides.

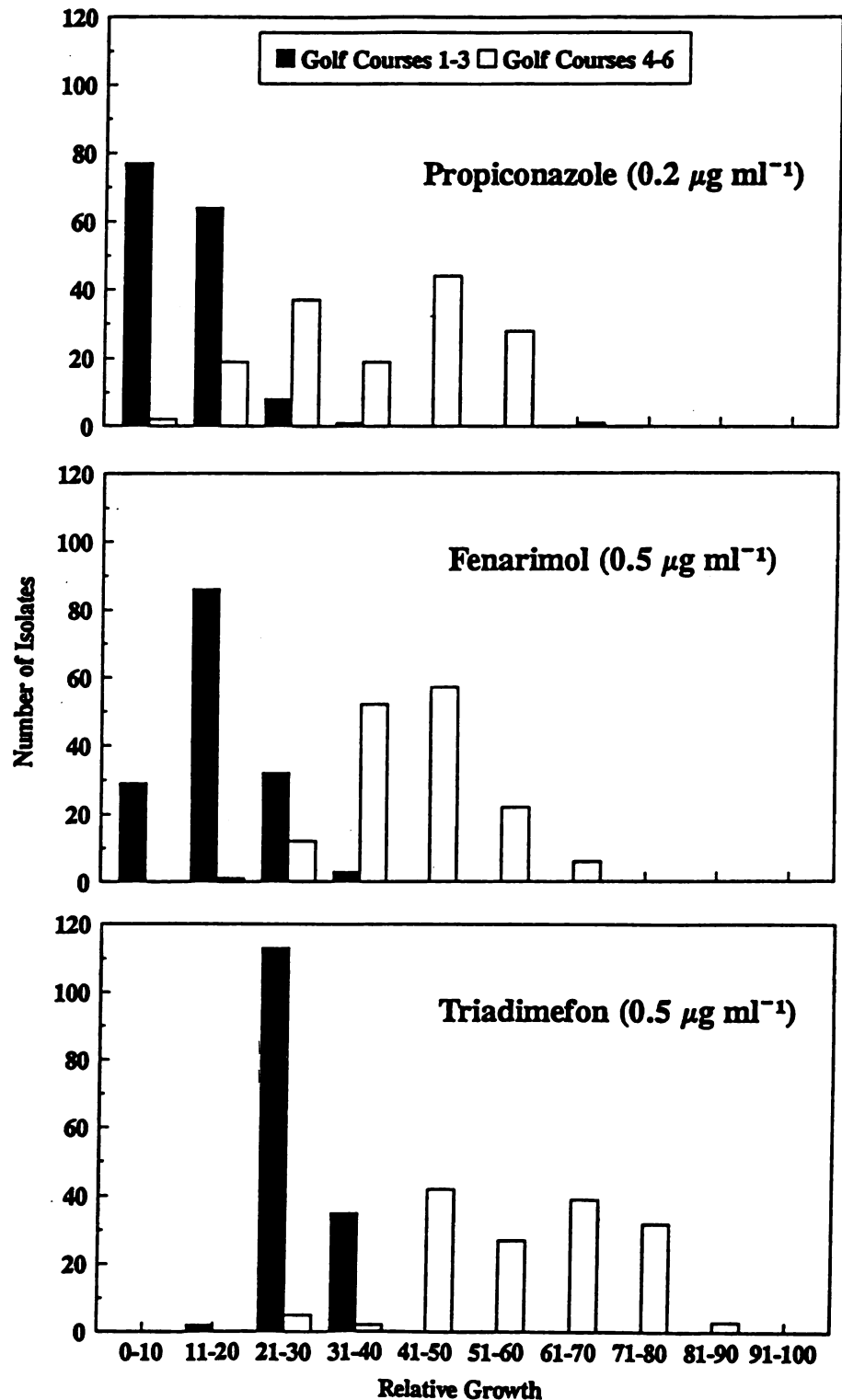


Table 2. Comparison of experimental triadimefon monitoring doses on *Sclerotinia homoeocarpa* isolates.

Golf courses ^a	Relative growth	
	Triadimefon (1 $\mu\text{g ml}^{-1}$)	Triadimefon (2 $\mu\text{g ml}^{-1}$)
1-3	8.0	3.7
4-6	38.0	29.0

^aFive samples from each golf course were tested at 0, 1, and 2 $\mu\text{g ml}^{-1}$ triadimefon. Relative growth was calculated for each isolate tested and a mean for isolates from golf courses 1-3 and 4-6 was reported.

Table 3. Disease ratings for dollar spot and the relative growth of *Sclerotinia homoeocarpa* per treatment on potato dextrose agar amended with 2 µg ml⁻¹ triadimefon.

Fungicide	Product per 100 m ²	Interval (days)	Disease ratings ^{ab}				Relative growth ^{bc}			
			1991	1992	1993	Average	1992	1993	Dif.	
Triadimefon 25% DF	30.5 g	21	9.0 c	3.3 b	4.3 c	5.5 bc	27.2 a	41.2 b	14.0 ab	
Fenarimol 1AS	55.7 ml	21	9.0 c	3.0 b	5.0 c	5.7 bc	26.2 a	41.2 b	15.0 b	
Propiconazole 1.1EC	31.8 ml	21	9.0 c	2.3 ab	6.3 c	5.9 bc	31.3 a	42.9 b	11.6 ab	
Iprodione 2 FL	63.7 ml	21	9.0 c	2.3 ab	3.0 bc	4.8 bc	31.4 a	37.8 ab	6.4 ab	34
Iprodione + triadimefon	63.7 ml + 30.5 g	21	8.7 c	1.7 ab	0.7 ab	3.7 abc	30.5 a	40.9 b	10.4 ab	
Chlorothalonil 4.17 FL	191.0 ml	10	0.0 a	0.0 a	0.0 a	0.0 a	29.5 a	----- ^d	-----	
Triadimefon + chlorothalonil	30.5 g + 191.0 ml	21	6.0 b	0.0 a	0.0 a	2.0 ab	26.7 a	39.8 ab	13.1 ab	
Control	-----	-----	9.0 c	8.0 c	4.0 c	7.0 c	29.4 a	32.9 a	3.5 a	

^a0 = no disease; 9 = severe disease; based on three replications per fungicide treatment.

^bStatistical analysis performed on square root transformation using Tukey's Honestly Significant Difference Test at P = 0.05.

^cExpressed as relative growth calculated from 10 samples per replication per fungicide treatment tested at 0 and 2 µg ml⁻¹ triadimefon.

^dData missing due to lack of disease.

a tank mixture with triadimefon was not statistically ($P=0.05$) different in 1991 and 1992 compared to the incidence in plots treated with triadimefon. The treatment of iprodione applied by itself was not statistically different from the untreated control in 1991 and 1993, but it significantly ($P=0.05$) reduced the severity of dollar spot in 1992. The severity in each of the three years was reduced significantly ($P=0.05$) when triadimefon was combined with chlorothalonil. Chlorothalonil applied alone on a 10-day schedule provided 100% control in each of the three years.

Population sensitivity. A shift in *S. homoeocarpa* populations towards resistance, which was indicated by an increase in relative growth, was observed in plots treated with the DMI fungicides (Table 3). The mean relative growth of isolates of *S. homoeocarpa* from the plots treated with the DMIs were significantly ($P=0.05$) higher compared to isolates from untreated control plots in 1993. There was no reduction in the level of sensitivity in the absence of the DMI fungicides as shown by the mean relative growth of the *S. homoeocarpa* isolates from the untreated control plots. Tank mixtures of triadimefon with iprodione and chlorothalonil also resulted in a shift in sensitivity within the *S. homoeocarpa* populations.

DISCUSSION

The sensitivity to DMI fungicides of *S. homoeocarpa* populations was based on relative growth values derived from the inhibition of mycelial growth on media

amended with triadimefon. This method had been used before (4, 10, 13) and shown to be effective in determining DMI sensitivity distribution. Mycelial growth is the developmental stage most effectively inhibited by DMI fungicides under normal conditions (7).

The significant difference in the sensitivity to three DMI fungicides of isolates from golf courses 1-3 compared to isolates from courses 4-6 indicates that the *S. homoeocarpa* population, presumably in response to the selection pressure of DMI fungicidal sprays, has developed resistance to this group of fungicides. Although there have been previous reports of DMI resistance developing in other pathogens such as *Sphaerotheca fuliginea* (cucumber powdery mildew) (8), *Guignardia bidwellii* (grape black rot) (13), and *Venturia inaequalis* (apple scab) (11), this is the first documented case of DMI resistance to a turfgrass pathogen.

All triadimefon-resistant isolates of *S. homoeocarpa* were also cross-resistant to fenarimol and propiconazole, based on both ED₅₀ and mean relative growth determinations. Baseline sensitivity data has been reported for *V. inaequalis* (4, 10, 13). Cross-resistance was also observed among the DMI fungicides tested, however, baseline sensitivities varied depending on the intrinsic activity of the fungicide (5).

The most disturbing aspect about *S. homoeocarpa* resistance developing to the DMI fungicides is that resistance already exists to the benzimidazole and dicarboximide fungicides, this means that strains of *S. homoeocarpa* now exist that

are resistant to three major classes of fungicides. This would mean the only available treatment for dollar spot management on golf courses where resistance to the DMIs exists is the contact fungicide chlorothalonil.

Fungicide spray programs need to be developed to delay dollar spot from becoming resistant to the DMI fungicides since most of the resistance problems to the systemic fungicides has occurred with this disease. The selection pressure on the *S. homoeocarpa* population can be reduced by limiting the use of DMIs during the time of year when dollar spot is most severe. This should allow the wild-type DMI-sensitive strains of *S. homoeocarpa* to remain dominant for a longer period of time in the population.

The sensitivity of individual isolates should be determined with a range of fungicide concentrations as a prerequisite for resistance monitoring (1). Smith et al. (10) confirmed the need for precise sensitivity data in the monitoring of quantitative fungal population shifts. They also emphasized the need for sample sizes large enough to determine sensitivity shifts of populations. They proposed a simplified procedure by which an established fungicide concentration is used as a monitoring dose to follow shifts in populations towards resistance (10) with the objective of testing isolates with a reduced number of fungicide applications, while maintaining a high level of precision and accuracy. This method has proven to be an effective tool to determine and compare the sensitivity of fungal populations.

Although shifts in the sensitivity of *S. homoeocarpa* populations were observed for plots treated with the DMI fungicides at golf course 5, the shifts were limited due to the fact that the majority of the fungal population was resistant prior to the initiation of the experiment.

We conclude that failure of the DMI fungicides to control dollar spot on a golf course in Michigan during 1991-1993 was preceded by the selection of DMI-resistant *S. homoeocarpa* strains through long term use of DMI fungicides. It would seem wise to anticipate future DMI resistance problems and to adopt defensive usage patterns to avoid the buildup of resistant organisms. Proper fungicide programs need to be implemented, monitored, and constantly adapted if turfgrass managers are to overcome one of the most difficult resistance control problems to date.

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APPENDICES

APPENDIX A

1991 Demethylation Inhibitor (DMI) Resistant
Dollar Spot Fungicide Field Trial

The dollar spot (*Sclerotinia homoeocarpa*) fungicide field trial was conducted at the Lochmoor Country Club near Grosse Pointe, MI. The area was a mixture of annual bluegrass (*Poa annua*) and creeping bentgrass (*Agrostis stolonifera*), irrigated daily, fertilized at $\frac{1}{4}$ lb. N per 1000 ft² every 14 days, and mowed at $\frac{3}{8}$ " height of cut.

Treatments were initiated on 28 August 1991 with subsequent applications being made on a 10 and 21 day interval. Treatments on a 10 day interval were applied four times, 28 August, 5, 19 September, 4 October, while those on a 21 day interval were applied twice, 28 August and 19 September. The treatments were applied to 6' x 6' plots replicated three times in a randomized block design using a CO₂ small-plot sprayer operating at a volume of 48 gal/acre and a pressure of 30 PSI.

Disease ratings were taken 5, 10, 13, 19, 24, September, and 4, 11, October. Due to the heavy disease pressure (90% disease infection) prior to the first fungicide application, turfgrass recovery was slow. However, two applications of the contact fungicide chlorothalonil at 6 oz./1000 ft² every 10 days provided 100% disease control. The incidence of dollar spot in plots treated with the DMI fungicides was not statistically (HSD, $P=0.05$) from the untreated control.

Table 1. 1991 dollar spot fungicide field trial A at the Lochmoor Country Club, Grosse Pte., MI. Disease ratings based on 3 replications per fungicide treatment.

Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{ab}						
			9/5	9/10	9/13	9/19	9/24	10/4	10/11
Triadimefon	1	21	9 A	9 F	9 E	9 F	9 D	9 E	9 F
Fenarimol	1.75	21	9 A	8.3 D-F	9 E	9 F	9 D	9 E	9 F
Propiconazole	1	21	8.7 A	8 B-F	9 E	9 F	9 D	9 E	9 F
Cyproconazole	0.33	21	9 A	6 B-F	6.3 A-E	6 C-F	6.3 CD	5.3 A-E	5.3 A-F
Tebuconazole	0.5	21	9 A	8.7 EF	9 E	9 F	9 D	9 E	9 F
Iprodione	2	21	8.7 A	5.3 A-F	6.3 A-E	9 F	9 D	9 E	9 F
Chlorothalonil	6	10	8.7 A	3 AB	3.3 A-C	0 A	0 A	0 A	0 A
Anilazine	4	10	8.7 A	2.3 AB	2.7 A-C	4 A-D	4.3 A-D	2.7 A-D	2 A-C
Benomyl	1	21	9 A	9 F	9 E	9 F	9 D	9 E	9 F
Triadimefon + Fenarimol + Propiconazole	0.5 + 0.875 + 0.5	21	6.3 A	6 B-F	8 DE	7.7 C-F	7 CD	8 DE	8 EF
Fenarimol + Chlorothalonil	1.5	10	7.7 A	3.7 A-D	3.7 A-D	5.3 B-F	4 A-D	3.3 A-D	2.3 A-D

Triadimefon + Iprodione	1 + 2	21	6.3 A	4.7 A-F	5.3 A-E	8.7 EF	8.7 D	8 DE	7.7 D-F
Fenarimol + Iprodione	1.75 + 2	21	6 A	2.7 AB	5 A-E	8.7 EF	8.7 D	9 E	8.7 F
Propiconazole + Iprodione	1 + 2	21	7.7 A	5.7 A-F	6.3 A-E	7.3 C-F	7 CD	6.7 C-E	6 B-F
Cyproconazole + Iprodione	0.33 + 2	21	8.3 A	6.3 B-F	6.7 B-E	3.7 A-C	3 A-C	2 A-C	1.3 A-C
Tebuconazole + Iprodione	0.5 + 2	21	7.7 A	5.7 A-F	6 A-E	8.7 EF	8.7 D	9 E	9 F
Anilazine + Iprodione	4 + 2	21	6 A	3.3 A-C	4.7 A-E	8 D-F	8.7 D	8 DE	6.7 C-F
Benomyl + Iprodione	1 + 2	21	7 A	5.7 A-F	7 C-E	8.7 EF	9 D	9 E	9 F
Chlorothalonil + Iprodione	6 + 2	21	7.3 A	3.3 A-C	3.3 A-C	5 B-F	4.7 A-D	3 A-D	2.7 A-E
Triadimefon + Chlorothalonil	1 + 6	21	6.3 A	4.3 A-F	5 A-E	6 C-F	5.3 B-D	3.3 A-D	2.7 A-E
Fenarimol + Chlorothalonil	1.75 + 6	21	6.7 A	3.3 A-C	3 A-C	4.7 B-E	4.7 A-D	4 A-E	3 A-E
Propiconazole + Chlorothalonil	1 + 6	21	5.3 A	1 A	2 A	7 C-F	6.3 CD	5.7 B-E	3.7 A-F

Cyproconazole + Chlorothalonil	0.33 + 6	21	6.7 A	4 A-E	3.7 A-D	1.3 AB	1 AB	0.3 AB	0.7 AB
Tebuconazole + Chlorothalonil	0.5 + 6	21	5.3 A	2.7 AB	4.3 A-D	8.7 EF	8.7 D	8 DE	6.3 B-F
Benomyl + Chlorothalonil	1 + 6	21	6.7 A	6 B-F	7 C-E	7.7 C-F	7.3 CD	7 C-E	6.3 B-F
Fenarimol + Anilazine	1.75 + 4	21	6 A	5 A-F	6.7 B-E	8.7 EF	8.7 D	9 E	7.7 D-F
Triadimefon + Anilazine	1 + 4	21	6 A	3.3 A-C	5.7 A-E	8.7 EF	8.7 D	9 E	9 F
Propiconazole + Anilazine	1 + 4	21	4.7 A	1.7 AB	4.7 A-E	9 F	9 D	9 E	8.7 F
Cyproconazole + Anilazine	0.33 + 4	21	5.3 A	3 AB	2.3 AB	7.3 C-F	7.3 CD	7.3 C-E	5 A-F
Tebuconazole + Anilazine	0.5 + 4	21	5.3 A	2.7 AB	4.7 A-E	9 F	8.7 D	9 E	8.7 F
Benomyl + Anilazine	1 + 4	21	6.7 A	6 B-F	8.3 DE	9 F	9 D	9 E	9 F
Fenarimol + Chlorothalonil	4.5	21	7.3 A	5 A-F	5 A-E	8 D-F	8.7 D	9 E	9 F
Control	-----	-----	9 A	9 F	9 E	9 F	9 D	9 E	9 F

^a0 = no disease; 9 = severe disease

^bStatistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05

Treatments were initiated on 8/28/91
Application dates for treatments on a 10 day spray interval: 8/28, 9/5, 9/19, and 10/4
Application dates for treatments on a 21 day spray interval: 8/28 and 9/19

1992 Demethylation Inhibitor (DMI) Resistant
Dollar Spot Fungicide Field Trial

The dollar spot (*Sclerotinia homoeocarpa*) fungicide field trial was conducted at the Lochmoor Country Club near Grosse Pointe, MI. The area was a mixture of annual bluegrass (*Poa annua*) and creeping bentgrass (*Agrostis stolonifera*), irrigated daily, fertilized at $\frac{1}{4}$ lb. N per 1000 ft² every 14 days, and mowed at $\frac{3}{8}$ " height of cut.

Treatments were initiated on 21 July 1992. The total number of applications for treatments with respective time intervals were: 10 day-7 times, 14 day-5 times, 21 day-4 times, 10 alt. 14 day-6 times, 10 alt. 21 day-5 times, and 21 alt. 21 day-4 times. The treatments in study A were applied to 6' x 6' plots replicated three times, while those in study B were applied to 2' x 12' plots replicated four times. Both studies were a randomized block design with applications made using a CO₂ small-plot sprayer operating at a volume of 48 gal/acre and a pressure of 30 PSI.

Disease ratings were taken from both studies on 26 August, 3, 16, 24, September, and 1, 6, October. The disease pressure was more severe in study A, initiated in 1991, compared to study B which was initiated in 1991. The area used for study B had been treated extensively with in contact fungicides in 1991, therefore, resulting in mild disease pressure in 1992. The contact fungicides chlorothalonil and anilazine applied every 10 days provided 100% disease control. Phytotoxicity was observed in plots treated with triadimefon at 2 and 4 oz./1000

ft² every 10 days.

Table 2. 1992 dollar spot fungicide field trial A at the Lochmoor Country Club, Grosse Pte., MI. Disease ratings based on 3 replications per fungicide treatment.

Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{ab}					
			8/26	9/3	9/16	9/24	10/1	10/6
Triadimefon	1	21	0.3 A	1.7 AB	2 A-C	2.3 AB	3.3 A-C	4 A-D
Fenarimol	1.75	21	0.7 A	1.3 AB	1.7 A-C	2 AB	3 A-C	3 A-C
Propiconazole	1	21	0.3 A	0.7 A	1 AB	1.3 A	2.3 A-C	2.3 A-C
Cyproconazole	0.33	21	0 A	0.3 A	0 A	0.7 A	1 A	1 A
Tebuconazole	0.5	21	1.7 AB	2.7 A-C	3.3 B-D	4.7 BC	5.7 B-D	5.7 B-D
Iprodione	2	21	0.7 A	0.7 A	0.3 AB	1.3 A	2.3 A-C	2 A-C
Chlorothalonil	6	10	0 A	0 A	0 A	0 A	0 A	0 A
Anilazine	6	10	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	3	10	0 A	0 A	0 A	0 A	0 A	0 A
Triadimefon	2	10	0 A	0 A	0 A	0.3 A	0 A	0 A
Triadimefon	4	10	0 A	0 A	0 A	0 A	0 A	0 A
Triadimefon + Iprodione	1 + 2	21	0 A	0 A	0 A	0.7 A	1.7 AB	1 A
Fenarimol + Iprodione	1.75 + 2	21	0 A	0.7 A	0 A	1 A	1.7 AB	1.7 AB

Propiconazole + Iprodione	1 + 2	21	0 A	0 A	0 A	0.7 A	1.3 A	1.3 AB
Fenarimol + Iprodione	1 + 2	21	0 A	0.7 A	0.7 AB	1.7 AB	2.3 A-C	2.3 A-C
Tebuconazole + Iprodione	0.5 + 2	21	0 A	0.3 A	0 A	1 A	1.3 A	1 A
Fenarimol	1	21	4 B	4.3 BC	4.7 CD	5.7 C	6.3 CD	6.3 CD
Chlorothalonil + Iprodione	6 + 2	21	0 A	0 A	0 A	0 A	0 A	0 A
Triadimefon + Chlorothalonil	1 + 6	21	0 A	0 A	0 A	0 A	0 A	0 A
Fenarimol + Chlorothalonil	1.75 + 6	21	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole + Chlorothalonil	1 + 6	21	0 A	0 A	0 A	0 A	0 A	0 A
Fenarimol + Chlorothalonil	1 + 6	21	0 A	0 A	0 A	0.3 A	0.7 A	0.3 A
Tebuconazole + Chlorothalonil	0.5 + 6	21	0 A	0.3 A	0 A	0.3 A	1 A	0.7 A
Iprodione alt. Triadimefon	2 alt. 1	21 - 21	1 A	1.7 AB	1.3 AB	2.3 AB	3.3 A-C	3.7 A-D
Iprodione alt. Fenarimol	2 alt. 1	21 - 21	1 A	1 A	1 AB	1.3 A	2.3 A-C	2.3 A-C
Iprodione alt. Propiconazole	2 alt. 1	21 - 21	0.3 A	0.7 A	0.3 AB	1.3 A	1.7 AB	2 A-C
Chlorothalonil alt. Triadimefon	6 alt. 1	10 - 21	1.3 A	0.7 A	1 AB	1.7 AB	2.7 A-C	3 A-C
Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 21	0.7 A	0.3 A	0.3 AB	1 A	1.7 AB	1.7 AB
Chlorothalonil alt. Propiconazole	6 alt. 1	10 - 21	0.7 A	0.3 A	0.3 AB	1.7 AB	2.3 A-C	2.3 A-C
Control	-----	-----	4 B	5 C	6 D	7 C	8 D	8 D

*0 = no disease; 9 = severe disease

^bStatistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05

Treatments were initiated on 7/21/92
Application dates for treatments on a 10 day spray interval: 7/21, 8/4, 8/10, 8/24, 9/3, 9/16, and 9/24
Application dates for treatments on a 10 day alt. 21 day spray interval: 7/21, 8/4, 8/24, 9/3, and 9/24
Application dates for treatments on a 21 day spray interval: 7/21, 8/10, 9/3, 9/24
Application dates for treatments on a 21 day alt. 21 day spray interval: 7/21, 8/10, 9/3, 9/24

Table 3. 1992 dollar spot fungicide trial B at the Lochmoor Country Club, Grosse Pte., MI. Disease ratings based on 4 replications per fungicide treatment.

Fungicide Name Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{ab}					
			8/26	9/3	9/16	9/24	10/1	10/6
Triadimefon	0.5	14	0.3 A	0.8 A	0.5 AB	0.8 AB	1 BC	1 AB
Triadimefon + Chlorothalonil	0.5 + 6	14	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	14	0 A	0 A	0 A	0 A	0 A	0 A
Triadimefon + Vinclozolin	0.5 + 1	14	0 A	0 A	0 A	0 A	0 A	0 A
Vinclozolin alt. Triadimefon	1 alt. 1	21 - 21	0 A	0 A	0 A	0 A	0.3 AB	0.3 A
Vinclozolin	1	14	0 A	0 A	0 A	0 A	0 A	0 A
Vinclozolin	1	21	0 A	0 A	0 A	0 A	0 A	0 A
Tebuconazole	0.5	14	0.3 A	0.3 A	0.3 A	0.8 AB	0.8 A-C	1 AB
Tebuconazole + Chlorothalonil	0.5 + 6	14	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil alt. Tebuconazole	6 alt. 1	10 - 21	0 A	0.3 A	0 A	1 BC	0.8 A-C	0.5 A
Propiconazole	1	14	0 A	0.3 A	0 A	0 A	0 A	0 A
Propiconazole + Iprodione	1 + 2	14	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole + Chlorothalonil	1 + 6	14	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole + Carbonitrile	1 + 7gm ai	14	0 A	0 A	0 A	0 A	0 A	0 A

Carbonitrile	7 gm ai	14	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Fenarimol	1	14	0.5 AB	0.3 A	0.5 AB	1 BC	1 BC	1 BC	1 AB
Fenarimol	1	21	0.5 AB	0.8 A	1 B	1.8 CD	1.5 CD	2 B	2 B
Chlorothalonil	6	10	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	14	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	21	0 A	0.3 A	0 A	0.5 AB	0 A	0 A	0 A
Fenarimol + Chlorothalonil	1 + 6	14	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Fenarimol + Chlorothalonil	1 + 6	21	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 14	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 21	0.5 AB	0 A	0 A	0.8 AB	0.5 AB	0.5 A	0.5 A
Fluazinam	0.3	10	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Fluazinam	0.4	21	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Fluazinam	0.6	21	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Fluazinam	1.2	21	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Control	-----	-----	1 B	0.8 A	1 B	2 D	2 D	2 B	2 B
Iprodione	2	14	0 A	0.3 A	0 A	0 A	0 A	0 A	0 A

*0 = no disease; 9 = severe disease

^bStatistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05

^cTreatments were initiated on 7/21/92

Application dates for treatments on a 10 day spray interval: 7/21, 8/4, 8/10, 8/24, 9/3, 9/16, and 9/24

Application dates for treatments on a 10 day alt. 14 day spray interval: 7/21, 8/4, 8/17, 8/24, 9/16, and 9/24
Application dates for treatments on a 10 day alt. 21 day spray interval: 7/21, 8/4, 8/24, 9/3, and 9/24
Application dates for treatments on a 14 day spray interval: 7/21, 8/4, 8/17, 9/3, and 9/16
Application dates for treatments on a 21 day spray interval: 7/21, 8/10, 9/3, and 9/24
Application dates for treatments on a 21 day alt. 21 day spray interval: 7/21, 8/10, 9/3, and 9/24

1993 Demethylation Inhibitor (DMI) Resistant
Dollar Spot Fungicide Field Trial

The dollar spot (*Sclerotinia homoeocarpa*) fungicide field trial was conducted at the Lochmoor Country Club near Grosse Pointe, MI. The area was a mixture of annual bluegrass (*Poa annua*) and creeping bentgrass (*Agrostis stolonifera*), irrigated daily, fertilized at $\frac{1}{4}$ lb. N per 1000 ft² every 14 days, and mowed at $\frac{3}{8}$ " height of cut.

Treatments were initiated on 22 July 1992. The total number of applications for treatments with respective time intervals were: 10 day-6 times, 14 day-5 times, 21 day-3 times, 28 day-3 times, 10 alt. 14 day-5 times, 10 alt. 21 day-4 times, and 21 alt. 21 day-3 times. The treatments in study A were applied to 6' x 6' plots replicated three times, while those in study B were applied to 2' x 12' plots replicated four times. Both studies were a randomized block design with applications made using a CO₂ small-plot sprayer operating at a volume of 48 gal/acre and a pressure of 30 PSI.

Disease ratings were taken from both studies on 3, 11, 25, 31 August, 9, 16, 22, September, and 7 October. The contact fungicides chlorothalonil and anilazine applied every 10 days provided 100% disease control, while the recommended rates of the DMI fungicides triadimefon, fenarimol, and propiconazole did not provide acceptable disease management. Phytotoxicity was observed in plots treated with triadimefon at 2 and 4 oz./1000 ft² every 10 days.

Table 4. 1993 dollar spot fungicide trial A at the Lochmoor Country Club, Grosse Pte., MI. Disease ratings based on 3 replications per fungicide treatment.

Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{ab}							
			8/3	8/11	8/25	8/31	9/9	9/16	9/22	10/7
Triadimefon	1	21	3.3A-D	4 D-F	3 B-E	3.3 A-F	4.3C-E	4.3C-E	3.3A-D	3.3A-D
Fenarimol	1.75	21	3 A-D	3.3 B-F	3 B-E	3.7 B-F	4.3C-E	5 DE	4.3 CD	3 A-D
Propiconazole	1	21	3.7B-D	4.7 EF	4 DE	4.3 C-F	6.3 E	6.3 E	5 D	3.3A-D
Cyproconazole	0.33	21	1.7A-C	2 A-D	0.3 A	1 A-C	2 A-C	2.3A-D	1.7A-D	2.3A-D
Tebuconazole	0.5	21	5 D	5 F	4.7 E	5.3 EF	5.3 DE	5 DE	3.3A-D	3.7B-D
Iprodione	2	21	1 A	2.7 A-F	1 A-C	2.7 A-F	2.7A-D	3 A-E	4 B-D	4.3 D
Chlorothalonil	6	10	1 A	0.7 A	0 A	0 A	0 A	0 A	0 A	0 A
Anilazine	6	10	0.7 A	1 AB	0.3 A	0.7 AB	0 A	0 A	0 A	0 A
Chlorothalonil	3	10	2 A-C	1.7 A-D	2.7 B-E	1.7 A-D	0 A	0 A	0 A	0 A
Triadimefon	2	10	3 A-D	1.7 A-D	0 A	0 A	0 A	0 A	0.3 AB	0.7A-C
Triadimefon	4	10	1.7A-C	0.3 A	0 A	0 A	0 A	0 A	0 A	0 A
Triadimefon + Iprodione	1 + 2	21	1 A	2.7 A-F	0.3 A	1.7 A-D	0.7 A	0.7AB	2.3A-D	2.3A-D

Fenarimol + Iprodione	1.75 + 2	21	1 A	2.3 A-E	1 A-C	3.3 A-F	1.7A-C	2.7A-D	4 B-D	4 CD
Propiconazole + Iprodione	1 + 2	21	1 A	2 A-D	0.7 AB	2.3 A-E	1.7A-C	1.7A-D	2.7A-D	2.7A-D
Fenarimol + Iprodione	1 + 2	21	1.3AB	3.3 B-F	1 A-C	3 A-F	2 A-C	2 A-D	3 A-D	3.7B-D
Tebuconazole + Iprodione	0.5 + 2	21	1.7A-C	2.3 A-E	0.7 AB	3 A-F	1.7A-C	1.7A-D	3 A-D	4 CD
Triadimefon	1	10	4.3 CD	3.7 C-F	2.3 A-D	0.7 AB	2 A-C	0.7AB	0.7A-C	1.3A-D
Fenarimol	1	21	3.3A-D	3.7 C-F	3.3 C-E	5 D-F	4.7C-E	4.7DE	4.3 CD	3 A-D
Chlorothalonil + Iprodione	6 + 2	21	0.7 A	0.7 A	0 A	0 A	0 A	0 A	0 A	0.3 AB
Triadimefon + Chlorothalonil	1 + 6	21	1 A	1 AB	0 A	1 A-C	0 A	0 A	1 A-C	0.7A-C
Fenarimol + Chlorothalonil	1.75 + 6	21	1 A	1.3 A-C	0 A	1 A-C	0.3 A	0.7AB	0.7A-C	1.7A-D
Propiconazole + Chlorothalonil	1 + 6	21	1 A	2 A-D	0.3 A	1 A-C	0 A	0.3 A	1.3A-D	1.7A-D
Fenarimol + Chlorothalonil	1 + 6	21	0.7 A	1 AB	0.3 A	1.7 A-D	0.7 A	1 A-C	1.7A-D	1.7A-D

Tebuconazole + Chlorothalonil	0.5 + 6	21	1 A	1.7 A-D	0.3 A	2 A-E	0 A	0.7AB	1.3A-D	2 A-D
Iprodione alt. Triadimefon	2 alt. 1	21 - 21	2.7A-D	3.3 B-F	2.3 A-D	3.3 A-F	1 AB	1 A-C	1.7A-D	3.7B-D
Iprodione alt. Fenarimol	2 alt. 1	21 - 21	2 A-C	2.7 A-F	4.3 CD	6 F	4 B-E	3.7B-E	3.3A-D	3.7B-D
Iprodione alt. Propiconazole	2 alt. 1	21 - 21	1.3AB	2.7 A-F	3 B-E	4 B-F	2.7A-D	2 A-D	2.7A-D	2.3A-D
Chlorothalonil alt. Triadimefon	6 alt. 1	10 - 21	1.3AB	1.3 A-C	2.7 B-E	2 A-E	0.3 A	1 A-C	2.3A-D	2.3A-D
Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 21	1.3AB	1 AB	3 B-E	2.3 A-E	0.3 A	1 A-C	1.7A-D	2.3A-D
Chlorothalonil alt. Propiconazole	6 alt. 1	10 - 21	1.7A-C	1.3 A-C	2.3 A-D	2.3 A-E	1 AB	1.3A-C	2.3A-D	3 A-D
Control	-----	-----	4 CD	5 F	4.7 E	3.7 B-F	4 B-E	4 B-E	2.7A-D	2.3A-D
Triadimefon	2	21	3 A-D	3.3 B-F	2 A-D	1.3 A-C	2.7A-D	3.3A-E	3.3A-D	3 A-D
Triadimefon	4	21	2.3A-C	2.7 A-F	0.3 A	0 A	0.7 A	1 A-C	1.7A-D	2.3A-D
Chlorothalonil	6	14	1 A	0.7 A	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	21	1 A	2.7 A-F	1.3 A-C	3.3 A-F	2.7A-D	1 A-C	1.7A-D	1 A-D

Iprodione	4	21	1 A	1 AB	0 A	0.3 A	0 A	0 A	0 A	0 A
Thiophanate-methyl + Iprodione	2.1 fl.	14	1 A	1 AB	0 A	1.3 A-C	1 AB	1 A-C	1.3A-D	0 A

*0 = no disease; 9 = severe disease

^bStatistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05

^cTreatments were initiated on 7/22/93

Application dates for treatments on a 10 day spray interval: 7/22, 8/3, 8/11, 8/25, 8/31, and 9/9

Application dates for treatments on a 10 day alt. 21 day spray interval: 7/22, 8/3, 8/25, and 8/31

Application dates for treatments on a 14 day spray interval: 7/22, 8/3, 8/16, 8/31, and 9/16

Application dates for treatments on a 21 day spray interval: 7/22, 8/11, and 8/31

Application dates for treatments on a 21 day alt. 21 day interval: 7/22, 8/11, and 8/31

Table 5. 1993 dollar spot fungicide trial B at the Lochmoor Country Club, Grosse Pte., MI. Disease ratings based on 4 replications per fungicide treatment.

Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{a,b}							
			8/3	8/11	8/25	8/31	9/9	9/16	9/22	10/7
Triadimefon	0.5	14	3.8 GH	3.8 G-I	4.3 D-F	4 CD	3.8B-D	4.3 DE	4.8 E	4 C-E
Triadimefon + Chlorothalonil	0.5 + 6	14	1.5A-E	0.5 AB	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	14	2.3C-G	0.3 AB	0 A	0 A	0 A	0 A	0.3 A	0 A
Triadimefon + Vinclozolin	0.5 + 1	14	0.5A-C	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Vinclozolin alt. Triadimefon	1 alt. 1	21 - 21	1 A-C	1.8 A-F	1.3 A-C	1.8 A-C	0.5 AB	0.3 AB	0.8 AB	1 A-C
Vinclozolin	1	14	1 A-C	0.3 AB	0 A	0 A	0 A	0 A	0 A	0 A
Vinclozolin	1	21	1.3A-D	1.8 A-F	0.3 A	0.5 A	0 A	0 A	0.8 AB	1 A-C
Tebuconazole	0.5	14	3.5F-H	3.5 F-I	5 EF	5.3 D	5 D	5.3 E	4 DE	3.5B-E
Tebuconazole + Chlorothalonil	0.5 + 6	14	1 A-C	0.3 AB	0 A	0 A	0 A	0 A	0 A	0 A

Chlorothalonil alt. Tebuconazole	6 alt. 1	10 -21	1 A-C	1 A-D	2.8 B-D	1.3 AB	0.5 AB	0.8A-C	1 A-C	1.5A-D
Propiconazole	1	14	3 D-H	2.8 D-I	3.3 C-E	3.5 B-D	3.8B-D	5.5 E	5 E	4.3 DE
Propiconazole + Iprodione	1 + 2	14	1.5A-E	0.8 A-C	0 A	0 A	0 A	0.3 A	0.5 A	0 A
Propiconazole + Chlorothalonil	1 + 6	14	1 A-C	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole + Carbonitrile	1 + 7 gm ai	14	0.3 AB	0 A	0 A	0.5 A	0 A	0.3 AB	0 A	0 A
Carbonitrile	7 gm ai	14	0.8A-C	0.5 AB	0 A	0 A	0 A	0 A	0 A	0 A
Fenarimol	1	14	3 D-H	2.5 C-H	2.8 B-D	4.3 CD	4 CD	5 DE	5 E	4 C-E
Fenarimol	1	21	3.3E-H	4.3 HI	5.3 EF	5.5 D	4.8 D	6 E	4.8 E	5.3 E
Chlorothalonil	6	10	1.3A-D	0.3 AB	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	14	1.3A-D	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	21	1.8A-F	2 B-G	1.3 A-C	2.3 A-C	0.3 A	0.3 AB	1 A-C	1 A-C
Fenarimol + Chlorothalonil	1 + 6	14	1.3A-D	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Fenarimol + Chlorothalonil	1 + 6	21	0.8A-C	1 A-D	0.3 A	1.8 A-C	0 A	0.3 AB	0.8 AB	2.3A-E

Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 14	1 A-C	0.3 AB	0 A	0 A	0.8A-C	0.3 AB	0 A	0 A
Chlorothalonil alt. Fenarimol	6 alt. 1	10 -21	1.3A-D	1.3 A-D	3.8 DE	3.5B-D	2.3A-D	3.3B-E	3.5C-E	3.3B-E
Fluazinam	0.3	10	1.8A-F	0.3 AB	0 A	0 A	0 A	0 A	0 A	0 A
Fluazinam	0.4	21	0 A	1.5 A-E	0.5 A	2.3 A-C	0 A	0.8A-C	0.8 AB	2 A-D
Fluazinam	0.6	21	0 A	0.3 AB	0 A	0.8 A	0 A	0 A	0 A	0 A
Fluazinam	1.2	21	0 A	0 A	0 A	0 A	0 A	0 A	0 A	0 A
Control	-----	-----	3.8 GH	4.5 I	6 F	6 D	5.5 D	6 E	5 E	4.5 DE
Triadimefon + Vinclozolin	0.5 + 0.5	21	1.8A-F	2.5 C-H	1 AB	2.3 A-C	0 A	1 A-C	1.5A-D	2.3A-E
Triadimefon + Vinclozolin	0.75 + 0.75	28	2 B-G	2.5 C-H	0.8 AB	0.8 A	3 A-D	4 C-E	3.3 B-E	0.8 AB
Propiconazole + Anilazine	1 + 6	14	1.8A-F	0.8 A-C	0 A	0.3 A	0 A	0.3 AB	0.5 A	0 A
Propiconazole (gel)	0.31	14	4.3 H	3.3 E-I	4.8 D-F	5 D	4 CD	4.5 DE	3.3 B-E	1 A-C
Myclobutanil	0.6	21	4.8 H	2.5 C-H	5.3 EF	5.5 D	5 D	5.8 E	4.8 E	2.5A-E
Iprodione	2	21	-----	-----	1.5 A-C	1.8 A-C	0.8A-C	1.8A-D	1.3A-C	0.8 AB

*0 = no disease; 9 = severe disease

*Statistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05

*Treatments were initiated on 7/22/93

Application dates for treatments on a 10 day spray interval: 7/22, 8/3, 8/11, 8/25, 8/31, and 9/9
Application dates for treatments on a 10 day alt. 14 day spray interval: 7/22, 8/3, 8/16, 8/25, and 9/9
Application dates for treatments on a 10 day alt. 21 day spray interval: 7/22, 8/3, 8/25, and 8/31
Application dates for treatments on a 14 day spray interval: 7/22, 8/3, 8/16, 8/31, and 9/16
Application dates for treatments on a 21 day spray interval: 7/22, 8/11, and 8/31
Application dates for treatments on a 21 day alt. 21 day spray interval: 7/22, 8/11, and 8/31
Application dates for treatments on a 28 day spray interval: 7/22, 8/16, and 9/16

1993 Dollar Spot Fungicide Field Trial

The dollar spot (*Sclerotinia homoeocarpa*) fungicide field trial was conducted at the Hancock Turfgrass Research Center near East Lansing, MI. The area was a mixture of annual bluegrass (*Poa annua*) and creeping bentgrass (*Agrostis stolonifera*), irrigated daily, fertilized at $\frac{1}{4}$ lb. N per 1000 ft² every 14 days, and mowed at $\frac{1}{2}$ " height of cut.

Treatments were initiated on 5 August 1992. The total number of applications for treatments with respective time intervals were: 10 day-5 times, 14 day-4 times, 21 day-3 times, 28 day-2 times, 10 alt. 14 day-4 times, 10 alt. 21 day-4 times, and 21 alt. 21 day-3 times. DMI treatments were also applied in the early spring to simulate summer patch treatments to determine if the use of the DMIs prior to dollar spot activity had any effect on the development of resistant *S. homoeocarpa*.

The treatments in study A and B were applied to 2' x 12' plots replicated four times in a randomized block design using a CO₂ small-plot sprayer operating at a volume of 48 gal/acre and a pressure of 30 PSI. Disease ratings were taken from both studies on 14, 26 August, 1, 8, 16, September. All treatments provided acceptable dollar spot management.

Table 6. 1993 dollar spot fungicide trial A at the Hancock Turfgrass Research Center, East Lansing, MI. Disease ratings based on 4 replications per fungicide treatment.

Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{ab}				
			8/14	8/26	9/1	9/8	9/16
Triadimefon	0.5	14	1.8 A-D	0.3 A	1.5 AB	0.3 A	0 A
Triadimefon + Chlorothalonil	0.5 + 6	14	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	6	14	1 A-C	0 A	1 AB	0 A	0 A
Triadimefon + Vinclozolin	0.5 + 1	14	0 A	0 A	0 A	0.3 A	0.5 AB
Vinclozolin alt. Triadimefon	1 alt. 1	21 - 21	0.3 AB	0.5 A	0 A	0 A	0 A
Vinclozolin	1	14	0 A	0 A	0 A	0 A	0 A
Vinclozolin	1	21	0 A	1 A	0.3 AB	0 A	0 A
Tebuconazole	0.5	14	2.3 B-D	0.3 A	1.3 AB	0 A	0 A
Tebuconazole + Chlorothalonil	0.5 + 6	14	0 A	0 A	0.3 AB	0 A	0 A
Chlorothalonil alt. Tebuconazole	6 alt. 1	10 - 21	1 A-C	0 A	0.8 AB	1 AB	0 A
Propiconazole	1	14	0 A	0 A	0 A	0 A	0 A
Propiconazole + Iprodione	1 + 2	14	0 A	0.3 A	0.3 AB	0 A	0 A
Propiconazole + Chlorothalonil	1 + 6	14	0 A	0 A	0 A	0 A	0 A
Propiconazole + Carbonitrile	1 + 7 gm ai	14	0 A	0 A	0 A	0 A	0 A

Carbonitrile	7 gm ai	14	1.5 A-D	0 A	1.8 AB	0 A	0 A
Fenarimol	1	14	2.8 C-E	0 A	2.8 AB	0.3 A	0.3 AB
Fenarimol	1	21	3.3 DE	5 BC	2.3 AB	0.3 A	0.3 AB
Chlorothalonil	6	10	1.5 A-D	0 A	0.3 AB	0 A	0 A
Chlorothalonil	6	14	0.8 A-C	0 A	0.5 AB	0 A	0 A
Chlorothalonil	6	21	0.8 A-C	2.5 A-C	2 AB	1.5 AB	3 C
Fenarimol + Chlorothalonil	1 + 6	14	0 A	0 A	0 A	0 A	0 A
Fenarimol + Chlorothalonil	1 + 6	21	0 A	2.3 A-C	1 AB	0 A	0 A
Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 14	1 A-C	0.3 A	3.3 B	0.3 A	0 A
Chlorothalonil alt. Fenarimol	6 alt. 1	10 - 21	1 A-C	0.3 A	3.3 B	4.3 C	2.3 BC
Fluazinam	0.3	10	0 A	0.3 A	0.3 AB	0 A	0 A
Fluazinam	0.4	21	0 A	2.8 A-C	2 AB	0 A	0 A
Fluazinam	0.6	21	0 A	2 AB	1.3 AB	0 A	0 A
Fluazinam	1.2	21	0 A	0 A	0 A	0 A	0 A
Control	-----	-----	4.5 E	5.5 C	7 C	7 D	6.5 D
Triadimefon + Vinclozolin	0.5 + 0.5	21	0.3 AB	1.5 A	1 AB	0 A	0 A
Triadimefon + Vinclozolin	0.75 + 0.75	28	0 A	0.8 A	3.3 B	2.8 BC	3.5 C
Propiconazole + Anilazine	1 + 6	14	0 A	0 A	0 A	0 A	0 A
Propiconazole (gel)	0.31	14	0.3 AB	0 A	0 A	0 A	0 A

Myclobutanil	0.6	21	1.8 A-D	2 AB	1 AB	0.5 A	0.3 AB
Thiophanate-methyl + Iprodione	2.1 fl.	14	1.3 A-D	0.3 A	2.3 AB	0.5 A	0 A
Triadimefon Chlorothalonil	2 3	65°F & 30 10	0 A	0 A	0 A	0.5 A	0 A
Fenarimol Chlorothalonil	2 3	65°F & 30 10	0.8 A-C	1 A	1.3 AB	1.8 AB	0.3 AB
Propiconazole Chlorothalonil	4 3	75°F & 30 10	0 A	0 A	0 A	0 A	0 A

*0 = no disease; 9 = severe disease

^bStatistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05

^cTreatments were initiated on 8/5/93

Application dates for treatments on a 10 day spray interval: 8/5, 8/14, 8/27, 9/8, and 9/17

Application dates for treatments on a 10 day alt. 14 day spray interval: 8/5, 8/14, 9/1, and 9/8

Application dates for treatments on a 10 day alt. 21 day spray interval: 8/5, 8/14, 9/8, and 9/17

Application dates for treatments on a 14 day spray interval: 8/5, 8/18, 9/1, and 9/17

Application dates for treatments on a 21 day spray interval: 8/5, 8/27, and 9/17

Application dates for treatments on a 21 day alt. 21 day spray interval: 8/5, 8/27, and 9/17

Application dates for treatments on a 28 day spray interval: 8/5 and 9/1

Table 7. 1993 dollar spot fungicide trial B at the Hancock Turfgrass Research Center, East Lansing, MI. Disease ratings based on 4 replications per fungicide treatment.

Treatment ^c	Rate oz/1000ft ²	Interval (days)	Disease Ratings ^{ab}				
			8/14	8/26	9/1	9/8	9/16
Cyproconazole	0.16	21	0 A	0 A	0.3 AB	0 A	0 A
Triadimefon	1	21	0 A	0.3 A	0.3 AB	0.3 A	0 A
Triadimefon	2	21	0.3 AB	0.5 AB	0.3 AB	0 A	0 A
Triadimefon	4	21	0 A	0 A	0 A	0 A	0 A
Triadimefon	1	10	0.3 AB	0 A	0 A	0 A	0 A
Triadimefon	2	10	0 A	0 A	0 A	0 A	0 A
Triadimefon	4	10	0 A	0 A	0 A	0 A	0 A
Triadimefon + Chlorothalonil	1 + 6	21	0.3 AB	0.5 AB	0.3 AB	0 A	0 A
Chlorothalonil alt. Triadimefon	6 alt. 1	10 - 21	0 A	0 A	0 A	1 A	0 A
Triadimefon + Iprodione	1 + 2	21	0.3 AB	0 A	0 A	0 A	0 A
Iprodione alt. Triadimefon	2 alt. 1	21 - 21	0.5 AB	1.3 A-C	0.8 AB	0 A	0 A
Propiconazole	1	21	0 A	0.3 A	0.3 AB	0 A	0 A
Propiconazole	2	21	0 A	0.3 A	0 A	0 A	0 A
Propiconazole	4	21	0 A	0 A	0 A	0 A	0 A

Propiconazole	1	10	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole	2	10	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole	4	10	0 A	0 A	0 A	0 A	0 A	0 A
Propiconazole + Chlorothalonil	1 + 6	21	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil alt. Propiconazole	6 alt. 1	10 - 21	0.8 AB	0 A	0 A	0 A	0.8 A	0.3 A
Propiconazole + Iprodione	1 + 2	21	0 A	0.3 A	0 A	0 A	0 A	0 A
Iprodione alt. Propiconazole	2 alt. 1	21 - 21	0.8 AB	1.3 A-C	0.8 AB	1.8 BC	0.5 A	0.3 A
Iprodione	2	21	0.5 AB	2.3 BC	0 A	0 A	0 A	0 A
Chlorothalonil	6	10	0 A	0 A	0 A	0 A	0 A	0 A
Chlorothalonil	3	10	0.8 AB	0 A	0.3 AB	0 A	0 A	0 A
Control	-----	-----	2.8 D	2.5 C	2.8 C	3.5 B	2.5 B	2.5 B
Paclobutrazol	.125 lb ai/A	21	2.3 CD	2.3 BC	2.8 C	3 B	3.5 B	3.5 B
Paclobutrazol + Propiconazole	.125lb ai/A + 1	21	0 A	0.5 AB	0.3 AB	0 A	0 A	0 A
Chlorothalonil	6	21	0 A	0.8 A-C	0.5 AB	0 A	0 A	0 A
Triadimefon Chlorothalonil	2 3	65°F & 30 10	2.3 CD	1.3 A-C	0.8 AB	0.8 A	0 A	0 A
Fenarimol Chlorothalonil	2 3	65°F & 30 10	1.5 BC	0 A	1 AB	0.3 A	0 A	0 A
Propiconazole Chlorothalonil	4 3	75°F & 30 10	0.5 AB	0.3 A	0 A	0 A	0 A	0 A

Chlorothalonil	6	14	0 A	0 A	0.3 AB	0.8 A	1 A
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*0 = no disease; 9 = severe disease

†Statistical analysis performed using Tukey's Honestly Significant Difference Test at $P=0.05$

‡Treatments were initiated on 8/5/93

Application dates for treatments on a 10 day spray interval: 8/5, 8/14, 8/27, 9/8, and 9/17

Application dates for treatments on a 10 day alt. 21 day spray interval: 8/5, 8/14, 9/8, and 9/17

Application dates for treatments on a 21 day spray interval: 8/5, 8/27, and 9/17

Application dates for treatments on a 21 day alt. 21 day spray interval: 8/5, 8/27, and 9/17

APPENDIX B

Table 1. Population analysis of fungicide field trial A at the Lochmoor Country Club, Grosse Pte., MI. Relative growth was calculated from 30 samples per fungicide treatment tested at 0 and 2 $\mu\text{g ml}^{-1}$ triadimefon.

Treatment	Rate oz/1000ft ²	Interval (days)	Sensitivities ^{ab}			
			Summer 1992	Fall 1992	Summer 1993	Fall 1993
Triadimefon	1	21	27.2 A	34.1 AB	40.8 D	41.2 A-D
Triadimefon	2	21	-----	-----	32.2 A-C	39.2 A-D
Triadimefon	4	21	-----	-----	38.4 CD	43.5 CD
Triadimefon	1	10	-----	-----	35.6 A-D	45.9 CD
Triadimefon	2	10	-----	-----	40.0 CD	43.1 CD
Triadimefon	4	10	-----	-----	39.4 CD	-----
Fenarimol	1	21	-----	37.5 AB	33.3 A-D	40.0 A-D
Fenarimol	1.75	21	26.2 A	37.0 AB	39.3 CD	41.2 A-D
Propiconazole	1	21	31.3 A	36.3 AB	38.4 CD	42.9 CD
Cyproconazole	0.33	21	-----	31.6 A	39.5 CD	42.7 CD
Tebuconazole	0.5	21	-----	39.8 B	36.3 A-D	41.5 A-D
Iprodione	2	21	31.4 A	38.8 AB	37.8 B-D	37.8 A-D
Iprodione	4	21	-----	-----	33.5 A-D	-----
Anilazine	6	10	29.0 A	-----	37.7 B-D	-----
Chlorothalonil	3	10	-----	-----	37.3 A-D	-----

Chlorothalonil	6	10	29.5 A	-----	37.3 A-D	32.2 A
Chlorothalonil	6	14	-----	-----	33.5 A-D	-----
Chlorothalonil	6	21	-----	-----	30.4 AB	32.2 A
Triadimefon + Iprodione	1 + 2	21	30.5 A	35.3 AB	32.5 A-C	40.9 A-D
Fenarimol + Iprodione	1 + 2	21	-----	39.1 AB	36.9 A-D	38.0 A-D
Fenarimol + Iprodione	1.75 + 2	21	-----	31.5 A	35.7 A-D	38.0 A-D
Propiconazole + Iprodione	1 + 2	21	-----	38.9 AB	38.9 CD	40.3 A-D
Tebuconazole + Iprodione	0.5 + 2	21	-----	37.5 AB	36.2 A-D	46.7 D
Iprodione alt. Triadimefon	2 alt. 1	21 alt. 21	-----	39.7 B	34.7 A-D	39.4 A-D
Iprodione alt. Fenarimol	2 alt. 1	21 alt. 21	-----	39.0 AB	34.4 A-D	41.4 A-D
Iprodione alt. Propiconazole	2 alt. 1	21 alt. 21	-----	38.9 AB	36.0 A-D	42.1 B-D
Triadimefon + Chlorothalonil	1 + 6	21	26.7 A	-----	36.0 A-D	39.8 A-D
Fenarimol + Chlorothalonil	1 + 6	21	-----	34.9 AB	35.3 A-D	43.3 CD
Fenarimol + Chlorothalonil	1.75 + 6	21	-----	-----	36.3 A-D	44.2 CD
Propiconazole + Chlorothalonil	1 + 6	21	-----	-----	35.7 A-D	42.5 CD
Tebuconazole + Chlorothalonil	0.5 + 6	21	-----	33.9 AB	36.0 A-D	41.2 A-D
Chlorothalonil + Iprodione	6 + 2	21	26.0 A	-----	36.5 A-D	-----
Chlorothalonil alt. Triadimefon	6 alt. 1	10 alt. 21	-----	37.1 AB	35.3 A-D	42.8 CD

Chlorothalonil alt. Fenarimol	6 alt. 1	10 alt. 21	-----	38.0 AB	33.3 A-D	36.6 A-C
Chlorothalonil alt. Propiconazole	6 alt. 1	10 alt. 21	-----	34.2 AB	34.6 A-D	37.8 A-D
Control	-----	-----	29.4 A	32.4 AB	29.5 A	32.9 AB

*Sensitivities expressed as relative growth.

†Statistical analysis performed using Tukey's Honestly Significant Difference Test at $P=0.05$.

Table 2. Population analysis of fungicide field trial B at the Lochmoor Country Club, Grosse Pte., MI. Relative growth was calculated from 40 samples per fungicide treatment tested at 0 and 2 $\mu\text{g ml}^{-1}$ triadimefon.

Treatment	Rate oz/1000ft ²	Interval (days)	Sensitivities ^a		
			Fall 1992	Summer 1993	Fall 1993
Triadimefon	0.5	14	39.0 A	34.4 A-C	37.3 B
Triadimefon + Chlorothalonil	0.5 + 6	14	-----	37.6 A-C	-----
Chlorothalonil	6	14	-----	38.2 A-D	-----
Triadimefon + Vinclozolin	0.5 + 1	14	-----	42.0 B-E	-----
Vinclozolin alt. Triadimefon	1 alt. 1	21 alt. 21	-----	36.8 A-C	37.2 B
Vinclozolin	1	14	-----	35.9 A-C	-----
Vinclozolin	1	21	-----	35.8 A-C	36.3 AB
Tebuconazole	0.5	14	37.3 A	36.9 A-C	33.1 A
Tebuconazole + Chlorothalonil	0.5 + 6	14	-----	35.9 A-C	-----
Chlorothalonil alt. Tebuconazole	6 alt. 1	10 alt. 21	-----	40.1 A-E	34.1 AB
Propiconazole	1	14	-----	38.9 A-D	35.9 AB
Propiconazole + Iprodione	1 + 2	14	-----	37.0 A-C	-----
Propiconazole + Chlorothalonil	1 + 6	14	-----	35.6 A-C	-----

Propiconazole + Carbonitrile	1 + 7 gm ai	14	-----	37.0 A-C	-----
Carbonitrile	7 gm ai	14	-----	48.2 E	-----
Fenarimol	1	14	36.1 A	46.5 DE	-----
Fenarimol	1	21	36.3 A	37.1 A-C	35.8 AB
Chlorothalonil	6	10	-----	36.2 A-C	-----
Chlorothalonil	6	14	-----	31.9 A	-----
Chlorothalonil	6	21	-----	34.8 A-C	-----
Fenarimol + Chlorothalonil	1 + 6	14	-----	34.8 A-C	-----
Fenarimol + Chlorothalonil	1 + 6	21	-----	35.6 A-C	36.7 AB
Chlorothalonil alt. Fenarimol	6 alt. 1	10 alt. 14	-----	38.2 A-D	-----
Chlorothalonil alt. Fenarimol	6 alt. 1	10 alt. 21	-----	38.2 A-D	37.5 B
Fluazinam	0.3	10	-----	42.9 C-E	-----
Fluazinam	0.4	21	-----	33.6 AB	36.4 AB
Fluazinam	0.6	21	-----	38.2 A-D	-----
Fluazinam	1.2	21	-----	37.9 A-D	-----
Control	-----	-----	35.0 A	40.7 B-E	36.0 AB

*Sensitivities expressed as relative growth.

*Statistical analysis performed using Tukey's Honestly Significant Difference Test at P=0.05.

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