

Arsenate Herbicide Stress and Incidence of Summer Patch on Kentucky Bluegrass Turfs

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ABSTRACT

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Investigations were conducted on the relationships between arsenical weed control programs on Kentucky bluegrass and the incidence of summer patch (formerly known as Fusarium blight syndrome), caused by *Phialophora graminicola*. At locations where the disease is likely to occur, applications of calcium arsenate caused extreme amplifications of disease, enough to render it uncontrollable with fungicides. Where the disease is not known to occur, large excesses of arsenate failed to induce new occurrences of summer patch. These results are discussed in relation to soil moisture extremes known to predispose bluegrasses to the disease and to stresses caused by arsenicals in the root zones of plants.

A group of distinctively shaped patch diseases previously known as Fusarium blight or Fusarium blight syndrome (20) are now known to be caused by *Phialophora graminicola* (Deacon) Walker, *Leptosphaeria korrae* Walker & Smith, and similar fungi (25). The diseases are known to occur and to become most severe during periods of environmental stress (17,19). Because the causal agents were only recently identified and techniques for inducing field symptoms under controlled conditions were unavailable before 1983 (17,19), most investigations have placed emphasis on cultural and chemical control procedures in the field.

The disease caused by *P. graminicola*, now named summer patch (21), has been investigated annually (as "Fusarium blight") over a 10-yr period at the Mill River Club (Oyster Bay, Long Island, NY). Countless observations of the disease's occurrence on specific fairways and over many seasons and management

conditions were made at this golf course. Management records at the Mill River Club revealed that large amounts of calcium arsenate had been used on their Kentucky bluegrass (*Poa pratensis* L. cv. Merion) fairways to suppress encroachment of annual bluegrass (*P. annua*). This weed control program was used extensively by turfgrass managers in the late 1950s (5,15). It is estimated that about 3 million pounds of calcium arsenate was used on turfgrasses during 1972 (W. H. Daniel, *personal communication*). Applications of the herbicide at the Mill River Club were made from 1969 to 1973.

At a time when summer patch was especially difficult to control on this Long Island golf course (23,29), Dickson (6) observed that the disease was much more prevalent on Merion Kentucky bluegrass areas treated with calcium arsenate (27% affected area) than on adjacent, untreated areas (3%). The disease appeared in Dickson's study of cultivar \times herbicide interactions in New Jersey during a drought period 2 yr after the herbicide had been applied. Other cultivars varied widely in their disease responses. For example, Fylking was very susceptible (17 and 50% affected area, respectively, in the untreated and treated areas) and Enmundi and Adelphi had almost no

disease (0-2%). Dickson's (6) data indicated that the arsenate may have caused the increase in "Fusarium blight" by effecting an increase in the depth of thatch on the treated plots. This proposal conflicts with our observation (19) that site-to-site variations in disease incidence on the Merion fairways at the Mill River Club could not be associated with variations in thatchiness.

The first objective of this study was to determine the influence of additional applications of calcium arsenate on the severity of summer patch on a Mill River Club fairway that contained residual arsenic from previous applications. A second objective was to determine whether the arsenate herbicide could be used experimentally to induce summer patch on susceptible bluegrass cultivars at other sites where the disease had never appeared. Additionally, we also sought to determine if difficulties in controlling this disease with benzimidazole fungicides at the Mill River Club and three other golf courses in Pennsylvania and Maryland could be associated with the history of arsenate herbicide use. To accomplish this, herbicide application records supplied by golf course superintendents were compared with published reports of disease control trials at these courses.

MATERIALS AND METHODS

Disease-affected site. A plot was established during 1978 on a 12-yr-old Merion Kentucky bluegrass fairway at the Mill River Club. This location was selected to represent an area where summer patch occurred frequently on soils containing residues of arsenic (135-528 μg total As/kg soil). A fairway with the lowest concentration of residual arsenic was selected for this study. The soil was a well-drained Riverhead sandy loam. Thatch depths averaged 1.9 cm (range 1.5-2.8 cm). Chipco tricalcium

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arsenate herbicide (48% a.i.) was applied at the rate of 24.4 kg of calcium orthoarsenate per hectare to three replicated strips (1.8 × 9.2 m) across the recently core-cultivated fairway on 17 May and 15 June. Half of each treatment area was sprayed twice (17 May and 15 June) with triadimefon at a rate of 6.05 kg a.i./ha per application. Summer patch began to appear in mid-August, and in late September, the areas of turf affected by the disease were evaluated. Cores (2.5 cm in diameter) of thatch and soil were collected from depths of 0–2, 2–4, and 4–6 cm for analyses of total arsenic concentration and pH. Total arsenic analyses were conducted by the Cornell University Soil Testing Laboratory; soil samples were digested with aqua regia and extracts were analyzed by atomic absorption.

Disease-free sites. Plots were established on Long Island at two locations where summer patch was not known to occur:

Pine Hills Country Club, Manorville, NY. A 5-yr-old Fylking Kentucky bluegrass fairway at this golf course was used as one disease-free site. The soil was a very well-drained Dukes loamy sand with thatch depths of about 1.5 cm. Chipco tricalcium arsenate was applied at six treatment rates and three application timings to replicated plots during 1980 and 1981. Treatments with cumulative totals of 140–525 kg a.i./ha were made, including single application rates up to 210 kg/ha. During the summers of 1980, 1981, and 1983, cores of thatch plus soil (from a depth of 3 cm) were taken for analysis of arsenic concentrations. Observations of turfgrass quality and summer patch development were made at near-monthly intervals during the summer each year.

Pinelawn Memorial Park, Farmingdale, NY. An experimental Kentucky bluegrass area at this cemetery was used as the other disease-free site. The turf consisted of Merion Kentucky bluegrass established from 1-yr-old sod in 1979. The plot area was divided so that half was watered frequently (usually daily during summer) and the other half infrequently (once a week or only as required to prevent severe drought) by an automatic sprinkler system. The soil was a well-drained Sassafras loam with thatch depths averaging about 2 cm. Tricalcium arsenate was applied to replicated plots in both irrigation schemes at the treatment

rates and schedules described for plots on the Pine Hills Country Club fairway. Assessments of arsenic concentrations, turf quality, and disease were also made as described.

Sods with differing arsenic treatment histories were also established at the Pinelawn Memorial Park experimental area for observation of possible occurrences of summer patch. A 1-yr-old blended Kentucky bluegrass (1:1:1, Fylking-Merion-Adelphi) was moved from Imperial Sod Farms, Wading River, NY, where arsenite defoliant had been used previously for production of potatoes. A 5-yr-old Fylking Kentucky bluegrass from the Cornell University Turfgrass Field Laboratory, where arsenates had never been used, was also used for this study. Background arsenic concentrations in these soils were 119 and less than 5 µg/g, respectively. Each sod was treated with tricalcium arsenate at application rates of 118 and 236 kg a.i./ha in April 1980 before being cut. Treated sods and adjacent untreated controls were cut in June 1980 and reestablished at Pinelawn Memorial Park under an infrequently irrigated regime. Observations of turfgrass quality and disease were made six or more times annually through 1983.

Arsenates and benzimidazole control failures. Comparisons were made of arsenic applications and benzimidazole control failures at four golf courses with common histories of arsenate applications and dramatic declines in the efficiency of benzimidazole-derivative fungicides for controlling "Fusarium blight." At these three sites, the inefficiency of the benzimidazoles persisted for only a few years before levels of control from these fungicides began to rise again. The golf courses included the Mill River Club, Oyster Bay, NY, the Waynesboro Country Club, Paoli, PA, the Moselem Springs Golf Club, Fleetwood, PA, and the Hunt Valley Golf Club, Baltimore, MD. The golf course superintendents at each location provided their records of tricalcium arsenate applications and of their ability or inability to control the disease with the benzimidazoles. Published reports from experimental fungicide trials conducted at these sites were also evaluated. The disease control trials were conducted by pathologists and agronomists at Cornell University, Pennsylvania State University, and the University of Maryland.

RESULTS

Disease-affected site. Symptoms of summer patch at the Mill River Club on Long Island developed rapidly during 16 and 17 August after 13.5 cm of rain fell between 4 and 12 August. Daily maximum air temperatures between 14 and 17 August were 30–31 C. The disease did not become amplified during a relatively dry period in late August to early September. Another sequence of heavy rainfall followed by a hot day occurred on 19–21 September, and the severity of summer patch increased. The extent of disease-affected areas was assessed on 27 September.

Applications of calcium arsenate greatly accentuated the incidence of summer patch (Table 1). The triadimefon fungicide was not capable of overcoming the increase in disease caused by the herbicide, although nearly total control of the disease was achieved with this fungicide at the other experimental sites on this golf course during 1978 and other years (26,27). The 3-mo period between initial application of arsenate and occurrence of the disease was much too short to have been associated with accumulations of thatch. Other mechanisms of disease induction appear to have been responsible for the increase in summer patch on the arsenate-treated areas.

Arsenic concentrations were nearly three times higher in the recently treated plot area than in areas with residues from past weed control programs. The high concentrations of arsenic in the depth zones of 2–4 and 4–6 cm in treated plots presumably resulted from the core cultivation procedure that had been conducted soon before calcium arsenate was applied. A slight increase in pH on the arsenate-treated plots presumably occurred because the manufacturing process for calcium arsenate results in an excess of calcium hydroxide in the commercial product. A background level of about 25 µg of arsenic per gram of soil was present in nearby areas of the golf course roughs, which were never directly treated with the herbicide but may have been contaminated by herbicide drift or leaf clippings from the treated fairways.

Disease-free sites. Applications of arsenate consistently failed to incite new instances of summer patch even though the tests were conducted on grasses considered highly susceptible to the disease. Detailed results of the arsenic

Table 1. Incidence of summer patch and the pH and arsenic concentrations in soil profiles of a calcium arsenate-treated golf course fairway and an untreated rough (Mill River Club, Long Island, NY)

| Treatment | Patch-affected area (%) | | Arsenic concentration (µg/g) | | | pH | | |
|-------------------|-------------------------|-------------|------------------------------|--------|--------|--------|--------|--------|
| | No fungicide | Triadimefon | 0–2 cm ^y | 2–4 cm | 4–6 cm | 0–2 cm | 2–4 cm | 4–6 cm |
| Untreated fairway | 9 c ^z | 0 d | 135 b | 76 c | 64 c | 5.8 a | 5.8 a | 5.7 a |
| Calcium arsenate | 52 a | 30 b | 380 a | 131 b | 141 b | 6.1 a | 6.0 a | 5.8 a |
| Untreated rough | 0 d | 0 d | 23 d | 26 d | 25 d | 4.2 b | 4.4 b | 3.2 c |

^y Cores (2.5 cm in diameter) of thatch and soil were collected from indicated depths for analyses of total arsenic concentrations and pH.

^z Within each main heading, means followed by the same letter do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.

concentrations in soil, turfgrass quality, and lack of summer patch are therefore not presented. Unfortunately, these tests were conducted when *Fusarium* spp. were thought to cause the patch disease, and the grasses were therefore not assayed for the presence of currently recognized primary pathogens. Concentrations of arsenic in treated sods from Imperial Sod Farms and the Cornell Turfgrass Field Laboratory, which were cut and reestablished as turfs at Pinelawn Memorial Park, were not determined after treatments.

Concentrations of arsenic were raised from a background of less than 5 $\mu\text{g/g}$ at Pine Hills Country Club to as high as 193 $\mu\text{g/g}$ in areas treated with the highest rates. In these treatment areas, the arsenic concentrations were maintained higher than 175 $\mu\text{g/g}$ from July 1980 to the end of the monitoring period in August 1983. These concentrations are not as high as those found on some disease-susceptible areas of fairways at the Mill River Club. In the latter instance, concentrations of arsenic varied from 135 $\mu\text{g/g}$ on sandy sites to 526 $\mu\text{g/g}$ on heavier, more poorly drained areas. The previously reported study at the Mill River Club (Table 1) was conducted on a sandy area of a disease-

affected fairway. Nevertheless, phytotoxic concentrations of arsenic accumulated at Pine Hills that was illustrated by wilting and deterioration of turfgrass quality on the plots treated with the higher rates of arsenate.

Arsenic accumulated to much higher concentrations in the tests at Pinelawn Memorial Park than in those at Pine Hills Country Club. The maximum concentrations at each location were 833 (Table 2) and 193 $\mu\text{g/g}$, respectively. The surface horizon of soil on the golf fairway is much coarser than that at the cemetery, and the thatchiness of the fairway turf is less than for the newly sodded plot at the cemetery. Because it was not the purpose of this study to evaluate factors responsible for retention or loss of arsenic from soils, the reasons for such large differences in recoverable arsenic were not determined. It is of interest, however, that the retention of arsenic in the frequently watered half of the cemetery test was much less than that in the infrequently watered half (Table 2). A gradual increase in background concentrations of arsenic in the untreated cemetery turfs, from 5–10 to 25–30 $\mu\text{g/g}$ over a 3-yr period, may indicate a transfer of arsenic from treated to untreated areas in the grass

clippings, but this hypothesis was not examined. Clippings fell randomly over the plot area and were not removed. Thatch accumulated much more rapidly in treated areas than in untreated areas, especially where water was applied infrequently. The pH values in the surface zone did not differ greatly and were unrelated to thatch accumulation.

Arsenates and benzimidazole control failures. Cumulative amounts of calcium arsenate applied to fairways at three golf courses are presented in Figure 1. Also illustrated are the years in which the fairways were subject to occurrences of "Fusarium blight" and in which the disease was considered uncontrollable with benzimidazole fungicides.

Arsenates were applied at the Mill River Club from 1969 to 1973. The disease first appeared in 1970 and was very severe through 1976. Experimental chemical control plots conducted annually on fairways at this golf course illustrate a period in which disease control was extremely difficult. Compared with untreated control areas, the reduction in disease severity by applications of benzimidazole fungicides in replicated plots decreased from 41–59% in 1974 (28) to a low of 20% in 1975 (22), whereupon the efficiency increased to 45–60% control in 1977 (27) and 64% in 1979 (26). In these study areas, fungicides with stronger abilities to control the disease, such as triadimefon and other sterol-biosynthesis inhibitors, generally provided total or near-total control of the disease. Exceptions were illustrated earlier (Table 1) where triadimefon also became ineffective on an experimental area treated with very high rates of arsenate.

Similar trends occurred on experimental plots on the Pennsylvania golf courses. Large amounts of arsenate were applied at Moselem Springs from 1969 to 1972, and "Fusarium blight" became worse from 1969 to 1971 and very severe in 1972 and 1973. Accentuation of disease control difficulties at Waynesboro in 1974 to 1976 followed arsenate applications in 1973 and 1974. The disease at both golf courses was of minimal importance by 1977. Applications of benzimidazole fungicides apparently

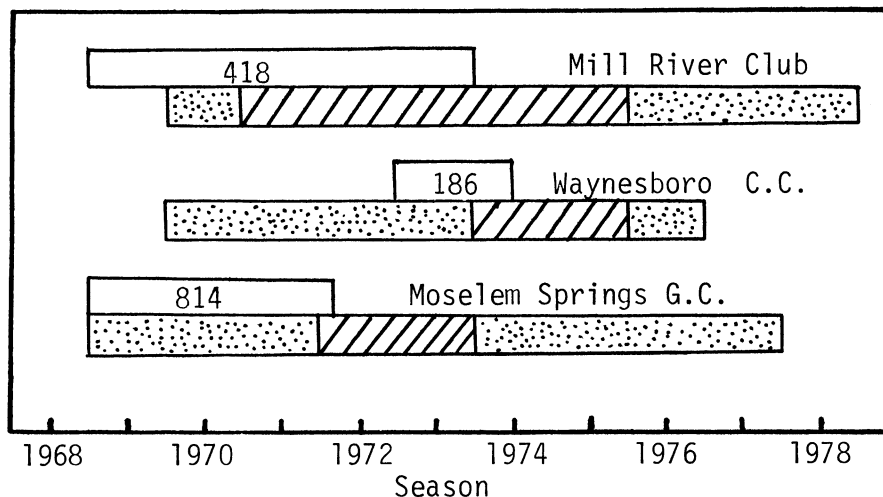


Fig. 1. Chronological sequence of "Fusarium blight" occurrences in forms controllable (dotted bar) or uncontrollable (crossed bar) with benzimidazole-derivative fungicides in relation to applications of calcium arsenate (open bar) at three golf courses (total calcium orthophosphate [in kg/ha] applied as indicated by open bars).

Table 2. Arsenic concentration, thatch depth, and pH in surface 2 cm of a calcium arsenate-treated Kentucky bluegrass turf where summer patch had never occurred (Pinelawn Memorial Park, Manorville, NY)

| Irrigation schedule | Application rate ^x | Arsenic concentration ($\mu\text{g/g}$) ^y | | | | | Thatch depth | pH |
|---------------------|-------------------------------|--|-----------|-----------|------------|-----------|--------------------|-----------|
| | | Dec. 1979 | July 1980 | Apr. 1981 | Sept. 1981 | July 1983 | July 1981 | July 1981 |
| Daily | None | <5 | <5 | 14 | 22 | 34 | 1.4 d ^z | 6.1 a |
| | Moderate | ... | 70 | 439 | 356 | 321 | 5.8 bc | 5.5 b |
| | High | ... | 180 | 641 | 500 | 433 | 5.9 c | 5.9 ab |
| Infrequently | None | <5 | <5 | 27 | 25 | 40 | 2.4 d | 5.7 ab |
| | Moderate | ... | 156 | 664 | 543 | 306 | 9.1 a | 5.6 b |
| | High | ... | 372 | 833 | 827 | 483 | 8.3 ab | 5.7 ab |

^x Calcium arsenate was applied at a standard (1X) rate in April and September 1980 and at a 0.5X rate in April 1980; the standard 1X rates were 140 and 210 kg/ha for the moderate and high application rates, respectively.

^y Arsenic concentrations were determined in nonreplicated composite samples.

^z Means followed by the same letter do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.

provided efficient disease control in 1970 but not in subsequent years. This fungicide continued to provide unacceptable levels of control at Waynesboro and Moselem Springs in 1976 (2). Disease control with applications of benzimidazoles at Moselem Springs increased to 71% in 1977 (3), but in some years, it continued to provide inconsistent protection from the disease (18) at this heavily treated site. Experimental applications of hydrated lime and additional arsenate accentuated the disease on plots at these golf courses in 1976 (H. Cole, Jr., and P. Sanders, unpublished).

Total amounts of arsenate applied at the Hunt Valley Golf Club in Maryland were uncertain. It is clear, however, that applications made in 1972 preceded the first occurrence of "Fusarium blight" in 1974 and the inability of benzimidazoles to control the disease in 1975. Subsequent experimental work at this location in 1980 (7) indicated that the benzimidazoles provided 85% reduction in disease incidence, which was superior to the efficiency of all other fungicides evaluated. Applications of lime in experimental areas accentuated the disease.

Although causal relationships cannot be determined from the observations and reports reviewed in this paper, it is obvious that periods of highly inefficient disease control with benzimidazoles closely followed periods in which arsenate herbicides were applied.

DISCUSSION

Arsenate herbicides applied to Kentucky bluegrass turfs can lead to explosive and uncontrollable occurrences of a summer patch disease previously known as Fusarium blight syndrome. It is also clear, however, that these disease outbreaks were apparently experienced on only a few of the many golf courses where arsenate herbicides have been used. This would indicate that for most golf courses, the primary causal agents were absent or inactive, the grass species or cultivars were tolerant of the pathogens and/or the arsenic, or the atmospheric and soil chemical and physical conditions that influence arsenic availability and phytotoxicity were not present during seasons in which the summer patch would have been likely to occur.

Because bioassays to detect the presence of primary causal agents were not reported until after our studies were concluded (25), it was not possible to determine the presence or activity of these pathogens. At the experimental sites, pathogenic *Fusarium* spp. were known to be ubiquitous and to remain so throughout the periods reported (14,24). Earlier work in our laboratory indicated the presence of benzimidazole-tolerant *Fusarium* spp. associated with crowns

and leaves of "Fusarium blight"-affected bluegrass (29). We also reported that disease control could be achieved with fungicides of the sterol-biosynthesis inhibitory type (23). Summer patch at the Mill River Club was not controllable with benzimidazoles at that time, thus apparently supporting the original hypothesis that *Fusarium* spp. incite the disease. When the benzimidazoles again began to provide control, it was also clear that the benzimidazole-tolerant *Fusarium* spp. remained in the population of facultative parasites associated with necrotic foliage in or around the disease patches. Evidence presented in this report indicates further that even such highly efficient fungicides as triadimefon can also be caused to fail when herbicide-induced stress is increased to very high levels. It is now known (R. W. Smiley and M. Craven Fowler, unpublished) that the benzimidazole and sterol-biosynthesis inhibiting fungicides are also toxic to *P. graminicola*. There appears to be an inverse relationship between the level of herbicide stress and the ability of fungicides to suppress the summer patch disease, and this relationship apparently has no bearing upon the presence or absence of fungicide-tolerant members of the *Fusarium* population. Our interpretation (29) of the importance of benzimidazole tolerance among the *Fusarium* population at the Mill River Club coincided with the existing state of knowledge relating to the summer patch disease, but that interpretation is now known to have been incorrect.

Rainfall and turfgrass watering programs each affect the incidence, severity, and timing of symptom development for patch diseases on Kentucky bluegrass (17,19). Periods of drought or of excess moisture, which may or may not be followed by short drought periods, appear to predispose bluegrasses to patch disease symptom development. Induction of disease symptoms by excess moisture has been recognized less frequently and is understood less than induction by drought. It is now recognized that reports of the summer patch disease occurring after overly wet periods during summer were often (but not always) derived from golf fairways and residential lawns with a history of arsenate herbicide applications. Soil chemical studies at the Mill River Club and elsewhere (30) illustrated that poorly oxidized conditions, including accumulations of toxic gases, reached growth-limiting levels in warm, wet turfgrasses and that the presence of arsenic in soil amplified this physiological stress. Conditions conducive to reduction of arsenate to arsenite (8,12) were measured by Thompson et al (30). Compared with the oxidized form of arsenic, reduced forms are much more soluble in water and are magnitudes higher in phytotoxicity (1). When extreme arsenic toxicity is

expressed, plant root and leaf growth is retarded and the plants may wilt (4,16).

In fully aerated soils, arsenate is the predominant form of arsenic. Because arsenic toxicity involves competition for uptake of phosphorus as well as poisoning of the sulfhydryl enzymes, high concentrations of this element in the soil solution can be expected to interfere with phosphorus nutrition. Hall and Miller (11) and Carrow et al (4) have illustrated the deleterious effects of arsenic competition and of low phosphorus availability on bluegrass growth. Phosphorus applications made to soils with low to adequate amounts of this element have been reported to reduce the numbers of wheat plants infected by *Gaeumannomyces graminis* var. *tritici* as much as 75% (13). This pathogen is ecologically similar and taxonomically close to *P. graminicola*, which causes summer patch of bluegrasses. Therefore, management practices that interfere with phosphorus nutrition may be expected to cause an increase in the occurrences of these diseases.

Our study was conducted by measuring the total arsenic concentrations of the tested soils. Because the soluble fraction is likely to be in a continual state of flux, we chose to only measure the total amounts that contribute to the soluble pool. Although this measurement has been correlated significantly with arsenic phytotoxicity to corn, it is not as accurate as individual determinations of arsenic forms present or of extractable arsenic (32). From our data, we therefore cannot judge what proportion of the reported arsenic concentration was in the solution phase. It was apparent from our field observations that total arsenic concentrations of less than 100 $\mu\text{g/g}$ at one golf course were more toxic than concentrations four times higher at another site.

We must conclude that statements of potential hazards associated with arsenate herbicide applications on turfgrasses (9,10) should be extended to include the potential for causing irreparable damage to turfgrass quality through the deterioration caused by the summer patch disease. Because the atmospheric and soil environments that determine arsenic phytotoxicity (31) cannot be controlled, use of this herbicide is to be discouraged on high-quality Kentucky bluegrasses.

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