

The Etiologic Dilemma Concerning Patch Diseases of Bluegrass Turfs

The demand for high-quality lawns, sports fields, and commercial landscapes increased rapidly in North America during the late 1940s. Newly released cultivars of Kentucky bluegrass (*Poa pratensis* L.) with improved resistance to foliar diseases became widely accepted. They were planted mostly as monocultures to achieve maximum benefit from their quality and uniformity. These new cultivars were accompanied by an increase in the intensity of turfgrass management practices. More equipment, automatic irrigation systems, pesticides, and high-analysis fertilizers were introduced into turfgrass maintenance programs. New diseases began to occur on the new, intensively managed grasses, and diseases on residential and commercial landscapes became as important as those on golf courses. Turfgrass pathology became accepted as an important and challenging specialty for research and extension and continues to grow in response to demands from our increasingly urban population. This paper illustrates just one of the concepts evolving within the subdiscipline of turfgrass pathology in the United States.

Overview of Fusarium Blight

A disease causing circular to serpentine necrotic patterns in turf (Fig. 1) was the first serious new problem to receive widespread attention on the "improved" Kentucky bluegrasses. The disease was first recognized during the 1950s. Tiller necrosis in distinct patches occurred similarly on turfs along the east and west coasts of North America. In the West, individual plants in the patches died from

a crown and root rot. A foliar blight symptom emphasized in early studies of the patch disease in eastern states did not occur in western ones (7). In the West, all



Fig. 1. Fusarium blight on Kentucky bluegrass lawn, August 1963.

plants in patches were affected, but in the East, patches of apparently healthy tillers often occurred in the centers of disease-affected patches, forming a ring or "doughnut" pattern. The unifying feature for the diseases in the East and the West was the ubiquitous presence of *Fusarium* spp. in and on affected tissues of symptomatic grasses. The primary symptoms, therefore, were described as different manifestations (phases) of a single disease that was named Fusarium blight (5). The concept of Fusarium blight gained rapid and thorough acceptance in the turfgrass trade even though several details leading to this conclusion were not in concert with symptom development in the field.

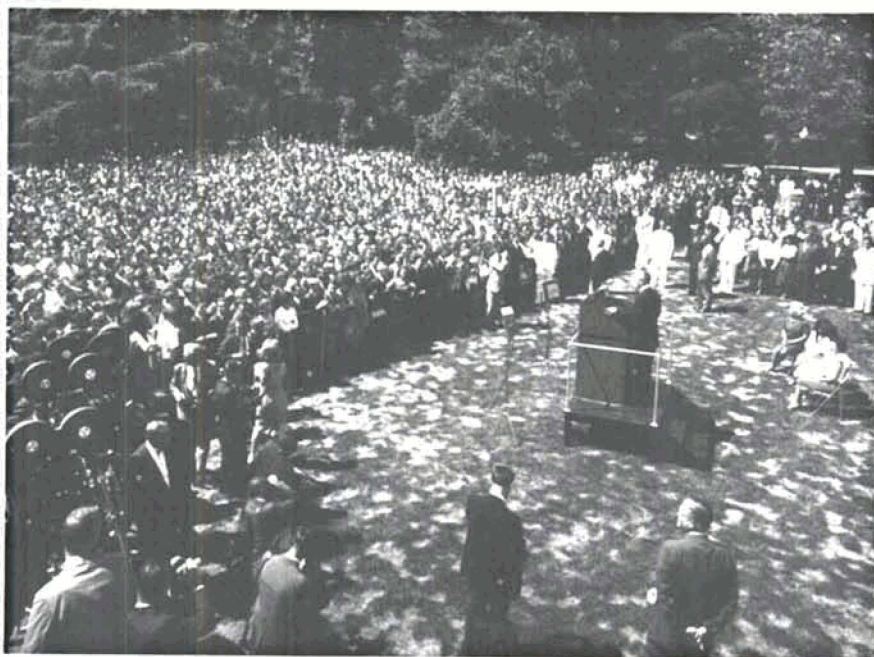


Fig. 2. The devastation of the South Lawn of the White House by Fusarium blight is evident in this photograph of President Lyndon Johnson giving an address in August 1964.

From 1966 to 1975, studies that led to development of moderately effective control strategies and practices made it increasingly doubtful that *Fusarium* spp. were the primary agents for many of the patches diagnosed as Fusarium blight. In all cases, however, the evidence supporting this doubt was entirely circumstantial. Little or no firm evidence was presented to demonstrate that the disease was caused by a primary agent other than *Fusarium*, and the possible involvement or even the identities of other suggested primary agents were speculative. This etiologic dilemma caused increasing disunity among pathologists, who faced increasing pressures from an industry whose technological base was advancing rapidly but whose disease control practices lacked reliability. Pressures were exerted from several directions. Manufacturers of fungicides were puzzled by the success of new compounds in controlling some but not all outbreaks of Fusarium blight, plant breeders were searching for improved inoculation techniques with *Fusarium* spp. to expedite screenings for disease resistance, golf course superintendents were purchasing microscopes to aid in rapid diagnosis of disease, and lawn maintenance supervisors were given and were giving conflicting recommendations regarding water management strategies in different regions where the disease occurred. A new faculty position at Cornell University was established as a direct response to industry concerns over the inability to understand and control Fusarium blight. The issue became even more complex when an association of *Rhizoctonia* spp. and unidentified basidiomycetes was suggested for patches similar to those of Fusarium blight (10,14).

During the early 1980s, the issue of patch disease etiology on bluegrass turfs erupted into a lively controversy. Those who doubted the accuracy of the Fusarium blight concept were faced with two choices: either abandon this line of research or present conclusive evidence to support an alternate hypothesis for the biology of this disease. More intensive examination soon led to the discovery that several newly recognized soilborne pathogens were associated with pre-symptomatic stages of patch development. Evidence indicated that the new fungi were primary agents for at least two different diseases that had been grouped together on the basis of similar patch symptomatology and the nearly ubiquitous presence of *Fusarium* spp. A stronger case was presented that *Fusarium* spp. associated with patch diseases in the eastern and north central United States act as secondary colonists whose importance to the full expression of the disease remains unknown.

Proponents of the original concept of Fusarium blight accept the validity of at

least one new patch disease but consider the other to be a "senescence syndrome" that occurs in precisely shaped patches (3). They also hold that Fusarium blight is a clearly defined disease that has not been investigated during recent studies even though the original and recent studies were conducted in the same region. As we enter the third decade of "Fusarium blight," research activity is intense to determine the merits of recent reports and hypotheses. The evolution of major thoughts on the etiology of Fusarium blight is presented here.

A Concept Is Born

Severe necrotic patches began to occur on Kentucky bluegrass turfs during the 1950s (1,4). The disease gained national prominence in 1964 when it devastated an acre of the South Lawn at the White House (Fig. 2) and much of the Capitol Mall in Washington, D.C. Intense pressure was exerted on pathologists to name and fully describe the disease and

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to develop control strategies. From isolations collected from fully symptomatic plants in several Mid-Atlantic states and using classical foliar epidemiologic methods, Couch and Bedford (5) concluded that the incitants were *Fusarium roseum* f. sp. *culmorum* and *F. tricinctum* f. sp. *poae*. These fungi occurred in high frequency on leaves, crowns, and roots of affected plants and were pathogenic to leaves of inoculated seedlings incubated for 3–7 days in a dew chamber. Couch and Bedford (5) coined the name Fusarium blight, and their report served as the only etiologic basis for control-oriented studies during the next 15 years.

The Concept Is Questioned

Five issues in the original disease description eventually caused concern among the pathologists who later initiated investigations of turf showing "Fusarium blight" symptoms. First, *Fusarium* spp. are ubiquitous on turfs.

Frequent isolation of *Fusarium* spp. from closely mowed grasses is expected, especially from fully symptomatic plants (19). Second, the *Fusarium* spp. tested by Couch and Bedford were more aggressive on bentgrasses than on bluegrasses. This was contrary to observations from the field (1,15,16) but could be conceptualized on the basis of differences in general management of these grasses. The remaining questions regard the appropriateness of the classical foliar epidemiologic tests with seedlings used to study a disease: 1) that rarely occurs on turfgrass stands less than 3 years old, 2) in which foliar necrosis is not always present during incipient stages of patch development in the eastern or western United States, and 3) that is often associated with drought during periods of high temperature (1,7,15,16). Additional questions about the accuracy of the etiologic assignment developed as further research and field observations were conducted. During the two decades following 1966, numerous attempts to reproduce the characteristic patch symptoms on mature turfs in the greenhouse and in the field, by inoculations with species of *Fusarium* on various food bases, have all failed (15,16). A facsimile of a patch disease did occur when *Fusarium* was inoculated into mature Kentucky bluegrass in one greenhouse test, but the patch's shape and overall appearance were not convincing and could not be reproduced.

The role of the foliar phase for Fusarium blight was questioned further when preventive applications of contact protectant fungicides, toxic to *Fusarium* spp., did not reduce the incidence of patches (1,16,20). However, advocates for the concept of this disease countered this argument by emphasizing the variability and uniqueness of certain species and strains of *Fusarium* and suggested that these variants were not properly evaluated in recent chemical control studies. Ecological studies of *Fusarium* spp. in turfgrass also cast doubt on the accuracy of the concept of Fusarium blight. A series of isolations from turf in a chemical control investigation at two locations revealed an inverse relationship between numbers of bluegrass crowns infected by *Fusarium* and subsequent incidence of patches in the treatment areas (20). This relationship was especially dramatic in turfs treated with iprodione, a fungicide that controls "Fusarium blight" but at the same time amplifies the number of *Fusarium* propagules in the soil (19). Excellent disease control is also achieved with applications of triadimefon, even though this fungicide is not highly toxic to the relevant species of *Fusarium*.

Challenges and Controversy

In the late 1970s, several scientists entered a period of open disagreement on



Fig. 3. (A) Necrosis of subterranean tissues and (B) irregularly shaped patches associated with *Fusarium* crown and root rot.

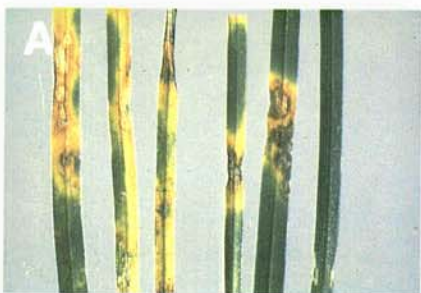


Fig. 4. (A) Leaf symptoms and (B) diffuse distribution of *Fusarium* leaf blight.



Fig. 5. Mapping technique used to relocate boundaries of patches after they fill in and blend with surrounding turf during winter.

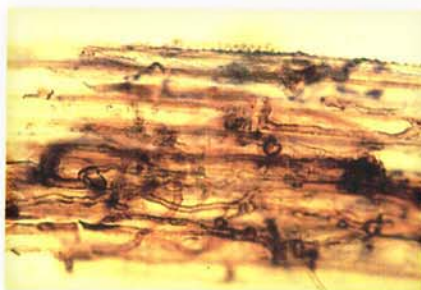


Fig. 6. Ectotrophic mycelium of *Leptosphaeria korrae* on bluegrass rhizome.



Fig. 7. Root rot of wheat grown in sand amended with turfgrass tissue and litter from edges of patch-affected bluegrass. This 34-day trap-crop assay (24–28 C) illustrates effects of (left to right) an unidentified black-mycelial pathogen, *Phialophora* spp., *Leptosphaeria korrae*, and no detectable root pathogen.

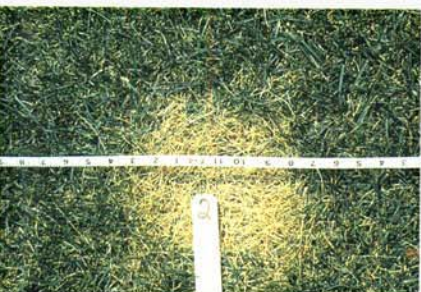


Fig. 8. Summer patch symptom in August 1983 following inoculation in April with *Phialophora* spp. on ryegrass grain in 2.5-cm-diameter hole near center of the patch. In 1984, the patch was larger.



Fig. 9. Summer patch of bluegrass grown in the greenhouse. Full-size patch symptom developed suddenly when environmental stresses occurred 21 weeks after inoculation with *Phialophora* spp.

the etiology of *Fusarium* blight as it related to a disease expressed by concisely shaped patches. The concept of *Fusarium* crown and root rots (6; Fig. 3) and of foliar blights (30; Fig. 4) caused by *Fusarium* spp. was not questioned. These diseases are diffusely distributed across a turf or they intensify into irregularly shaped patches, but they do not fit the precisely patterned disease described as *Fusarium* blight. Several scientists working in the area where the concept for this disease was born began disregarding the obvious presence of *Fusarium* spp. associated with the final stages of *Fusarium* blight and challenged the original concept (15,16). Arguments were based on 1) a preponderance of circumstantial evidence suggesting that *Fusarium* spp. were not the primary agents and 2) the absence of confirming evidence to support the concept that these fungi could cause a precisely patterned patch disease. Unfortunately, neither of the challenges offered a research-based alternate explanation for the etiology of this serious disease. Avenues for new research directions were suggested (16), and new studies were initiated in many states. A second new faculty position, at the University of Illinois, was established in direct response to the continual industry concern over this disease.

Since 1980, a lively controversy has developed over the concept of *Fusarium* blight. The issues came to be tried before popular audiences rather than within the plant pathology profession. Proponents of “*Fusarium* blight” maintained unwavering support from associations of golf course superintendents, but the professional lawn care and sod production industries became aligned with scientists who questioned the original concept. Scientific papers published in refereed journals concentrated on new findings but were always countered by reiterations of findings from the original studies in nonrefereed trade literature (2–4) and in privately published letters. The etiologic uncertainty was again highlighted when I changed the disease name from “*Fusarium* blight” to “*Fusarium* blight syndrome” (17), which quickly polarized proponents and opponents of the *Fusarium* blight concept. The debate over *Fusarium* blight has stimulated much new interest in the ecology of soilborne pathogens in the turfgrass ecosystem. The evolution of concepts regarding this patch disease is still far from complete.

Refinement of Causation

Progress toward unveiling a better understanding of the patch disease

syndrome was made simultaneously in several states during the early 1980s. Notable contributions came from California, Illinois, Michigan, New York, Pennsylvania, Rhode Island, Washington, and Wisconsin. Although much of this work remains to be published, introductions to important findings and concepts are available (8,9,13-16,21,31). The concepts and methodologies presented in the following summary emphasize those in my studies.

The etiologic dilemma of *Fusarium* blight was based on the methods used to coin the concept. Unfortunately, the "proof of pathogenicity" used for *Fusarium* spp. on grasses (5) can be achieved with isolates from roots and crowns of plants in nearly all turfgrass stands. Events early in the development of patch diseases, before foliar symptom expression, had not been investigated because symptoms often disappear between epiphytotic episodes. The patch symptoms are typically expressed during mid to late summer, and affected plants then recover or regrowth from adjacent plants fills the patch during the autumn, winter, and spring. Patches often become visually indistinguishable from healthy turf by midwinter. A method was developed to collect samples before symptoms became visible in the foliage (Fig. 5). Permanent markers (100-cm² metal plates with a 15-cm anchor spike) were installed in numerous patches throughout New York and Connecticut during 1980. A tape measure and compass were laid over the marked patches and they were photographed. The location of each marker was then triangulated to permanent structures, and the patches or their previous locations were observed and photographed annually. Samples were taken from precise positions through the patches and adjoining grass up to 5 months before visual symptoms developed.

We found that roots, crowns, and rhizomes of tillers at the edges of patches supported growth of dark-colored fungal mycelia (Fig. 6) similar to that of *Gaeumannomyces* spp. (21). These fungi were difficult to isolate directly because fungi with more rapid growth rates, such as *Fusarium* spp., typically overran the isolation medium. Indirect techniques that selected for pathogenicity were then adopted to isolate primary root-infecting fungi. Cores of turf from the patch perimeters were buried in coarse sand in greenhouse pots, and wheat, oats, or turfgrass species were grown as trap crops. The pathogenic fungi producing the dark mycelia could then be isolated directly and consistently from young, relatively "clean" roots of the trap crops (Fig. 7). Direct isolations of these fungi from

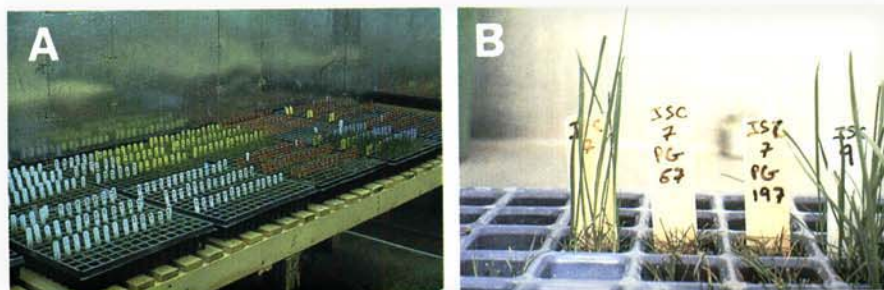


Fig. 10. Screening of the National Turfgrass Evaluation Program's collections for resistance to *Phialophora* spp. (A) Cone-tainers and (B) results.

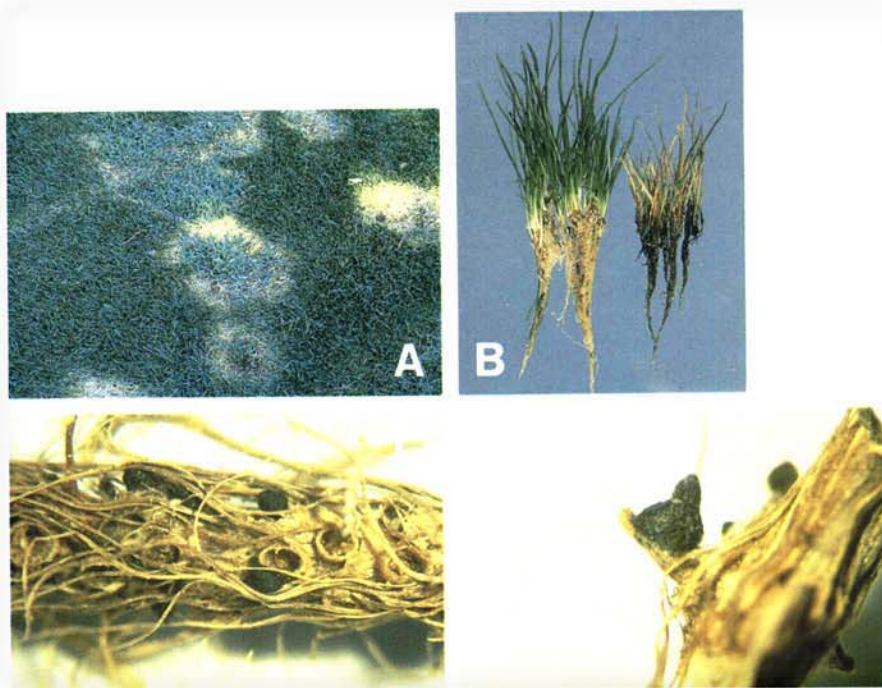


Fig. 11. Necrotic ring spot in (A) turf and (B) growth chamber, and pseudothecia of *Leptosphaeria korrae* on decomposing bluegrass (C) crown and (D) root.

patch-affected turfs became even more reliable and repeatable with a new semi-selective medium for *G. graminis* (11).

Inocula prepared from pure cultures of the fungi were placed into apparently healthy turfs at several locations in New York. Koch's postulates were fulfilled for fungi identified as *Leptosphaeria korrae* and *Phialophora graminicola*. Characteristic patch disease symptoms (Fig. 8) occurred at the loci where these soilborne fungi were placed in the field plots (21,23,24). Symptoms were expressed either in the year of inoculation or 1 or 2 years later, and patches reappeared at the same inoculum loci for 1, 2, 3, or more years. At some locations, only one of the fungi was effective in causing patches, but the fungus that was virulent at one location was not always the same as that causing patches in another inoculated plot. Both pathogens caused patches in some field tests. Effects of environmental conditions on growth of these fungi (25),

a method for demonstrating their patch-producing capability in the greenhouse (27; Fig. 9), and methods for identifying the diseases and pathogens (29) were reported. Inoculation procedures were then refined for investigating the efficiency of inocula placed into field management studies (24), for variety trials (23), and for screening, under controlled environments, the entries of turfgrass species, cultivars, and lines in the National Turfgrass Evaluation Program (22; Fig. 10).

Two New Diseases

Patches caused by *L. korrae* in Kentucky bluegrasses and other cool-season (C3 pathway) grasses have been named necrotic ring spot (31; Fig. 11). This pathogen is also the incitant of spring dead spot of bermudagrass in California (8). Unpublished reports indicated that this or a closely related

Leptosphaeria spp. has also been associated with spring dead spot in the central United States. Refined experimental and diagnostic techniques and the concentrated effort now directed toward the patch diseases open the possibility for identifying additional patch-inducing pathogens in grasses. It is interesting, for instance, that the most prevalent agent (*L. narmari*) of spring dead spot in Australia has not yet been identified in North America.

Summer patch is the name for disease caused by *Phialophora* spp. (18; Figs. 12 and 13). Although taxonomic and ecological characteristics led to the identification of this high-temperature pathogen as *P. graminicola* (21,25,28), further changes in pathogen identity are imminent. *G. cylindrosporus* is recognized as the teleomorph of *P. graminicola*, and Jackson and Landschoot (9) were the first to isolate *G. cylindrosporus* directly from plants grown in the field. Recent evidence indicates that the anamorph derived from *G. cylindrosporus* is not identical to many of the isolates used in my field and greenhouse studies (*P. Landschoot, personal communication, November 1986*). Our original report of these fungi (21) indicated a range in the capacities of isolates to infect wheat and oats in the trap-crop isolation procedure, and our subsequent research publications are based on tests with the most aggressive of the isolates taken from patch-affected grasses. The teleomorphs for most of the isolates used in my studies have now been produced by Landschoot, and the proven incitants of summer patch, such as 57-84 (ATCC 60239), i.e., the most aggressive isolates from a collection of *Phialophora* spp. with uniform appearance, have been identified as the *Phialophora* stage of what appears to be an undescribed species of *Magnaporthe* (13). The name of the agent of summer patch will, therefore, be redescribed by Landschoot and Jackson. Further and more difficult taxonomic separations will become necessary in the diagnosis and study of this poorly defined group of *Phialophora* spp.

At least one additional fungus with an ectotrophic growth habit similar to the previously mentioned organisms is suspected of involvement in this patch complex on Kentucky bluegrasses (Fig. 14; R. Smiley and M. Craven Fowler, *unpublished*). This potential agent, similar to the unidentified "black mycelium" (12), is difficult to study in turfgrass cultures because it is very tolerant of heat and grows very slowly (0.6 mm/day at 25 C, 1.4 mm/day at 32 C). This thermal characteristic contrasts with that of cool-season grasses (25) and causes considerable difficulty in studies on mature sods. Comparative pathogenicity tests in the greenhouse and growth chamber illustrated that the fungus was less pathogenic than the

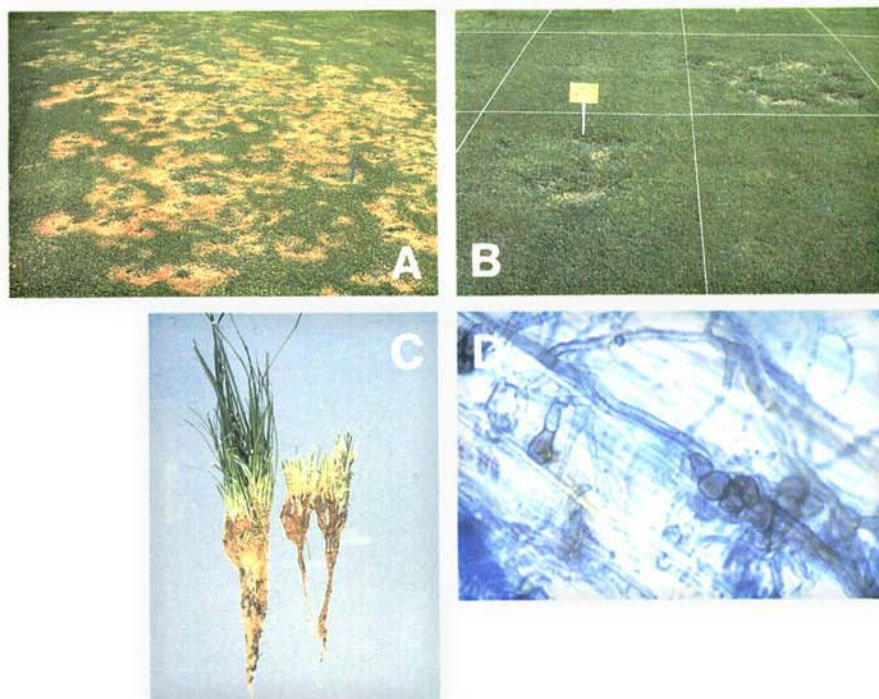


Fig. 12. Summer patch in (A) golf fairway, (B) commercial variety trial, and (C) growth chamber. (D) Hyphopodia of *Phialophora* spp.

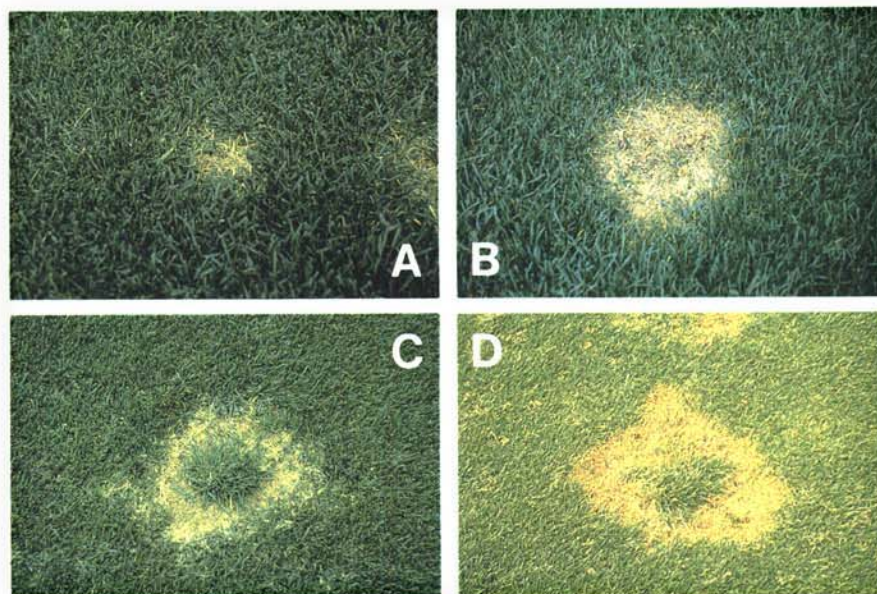


Fig. 13. (A-D) Stages (youngest to oldest) in development of summer patch of Kentucky bluegrass. Symptoms may first appear at any stage, depending on environmental conditions over the months or years during which radial growth of the pathogen has occurred.

Phialophora and *Leptosphaeria* isolates studied at high temperatures and did not cause disease at low to intermediate temperatures. As with the *Phialophora* spp. (25), this fungus has the potential to act as a competitor with *G. graminis* at intermediate temperatures. Field tests have not been attempted with the unidentified "black mycelium."

Future Directions for Research

The etiologic dilemma concerning Fusarium blight continues. Patches conforming to the generic description for this group of diseases have been



Fig. 14. Root and foot rot of wheat caused by an unidentified "black mycelium" isolated from patch-affected bluegrass.

separated into at least two new diseases caused by species of *Leptosphaeria* and *Phialophora*. The ability of *G. graminis* to infect roots in a similar manner and to cause take-all patch of bentgrasses (17) should be ample warning that it too should not be disregarded as a potential agent of patches on bluegrasses. It is clear, however, that studies of "Fusarium blight" prior to about 1982 were conducted on patches of uncertain etiology, and most of that research must now be repeated.

The formation of well-delimited patches, including the "doughnut" or "frog-eye" pattern, by species of *Fusarium* remains uncertain and unproved but must continue to be considered because negative results cannot be considered definitive. Definitive research is still needed to better define the significance and ecological role of *Fusarium* spp. in turfgrass pathology. The information described in this paper and the reclassification of *F. nivale* as *Gerlachia nivalis* and then *Microdochium nivalis* has resulted in the removal of all *Fusarium* spp. as known agents of patch diseases in turfgrasses.

The new agents of patches discussed in this paper have been known only for a relatively few years. In terms of historical mycology or pathology, they have barely entered the time line, and most information about these agents and their diseases has not yet been validated by independent study and the "test of time." Additional research is needed in the areas of mycologic taxonomy, physiology, and ecology.

Monographic treatments are needed for the genera *Leptosphaeria* and *Phialophora*. With the exception of *L. korrae* and *L. narmari*, which are root pathogens, all other pathogenic members of this genus attack foliage. The taxonomic separation of *Phialophora* spp. is quite uncertain. In view of the economic importance of the apparently new species involved in turfgrass diseases, further study becomes of even greater importance. Rapid diagnostic procedures will be required for positive identification of the agents of patch diseases.

Additional research is needed to determine the manner in which these pathogens and diseases are influenced by the environment. Necrotic ring spot and summer patch can both appear on Kentucky bluegrasses during the summer, but necrotic ring spot can also appear during the spring and autumn (25). Reasons for the appearance of a patch at any particular time remain poorly defined. Irrigation and fertilization practices affect each disease (23), but predictive guidelines and precise control recommendations are needed. The influence of herbicides is poorly understood, but it is clear that those that cause physiological stress to Kentucky bluegrass

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can enhance the incidence and severity of patches (26). The rate of anucleation of cortical cells in roots is affected by environmental conditions and affects the ability of *Phialophora* spp. to cause disease (28), but the physiological basis for this morphological effect remains to

be studied. Refinements in the suggested timing and rates of fungicide applications are needed.

Emphasis on development of grass cultivars and species with enhanced resistance to each and on combinations of the primary agents must be continued. Published evidence that cultivar \times isolate interactions are highly variable (22) illustrates the need to use many isolates in breeding and screening studies. Studies of three-way interactions of cultivars, agents, and environmental variables are badly needed in view of the regional responses of cultivars to these patch diseases (15,16).

Finally, in my opinion, two popular concepts used for diseases of grasses in North America must be deemphasized. Names of fungal genera should not be used to describe diseases of subterranean or surface-appressed plant parts. Likewise, the "single pathogen for a single disease" concept does not accurately account for successions or complexes of organisms that occur during the process of patch disease development. Disease names become part of the public domain and are not easily, uniformly, or fairly changed if circumstances indicate that isolates of the originally described genus can no longer be considered the primary agent or if the taxonomic assignment of a well-defined agent is changed. Examples for both of these situations are described in this paper, and further examples can be drawn from diseases caused by fungi that were once classified as species of *Corticium*, *Sclerotinia*, *Helminthosporium*, and *Ophiobolus*. These fundamental changes in philosophy will enable much more rapid progress toward understanding and controlling diseases on turfgrasses and toward much higher uniformity in our ability to communicate information about specific diseases.

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Dr. Smiley is superintendent of Oregon State University's Columbia Basin Agricultural Research Center at Pendleton, where he administers two stations and conducts research on soilborne pathogens of cereals. He is also a professor at Oregon State University and serves on the graduate faculties in the Department of Botany and Plant Pathology and the Department of Crop Science. Rhizoctonia root rot and cereal cyst nematode are currently being emphasized in his research. The work presented here was conducted at Cornell University, where he devoted 12 years to studies on biology and control of Fusarium blight, nontarget effects of fungicides in the turfgrass ecosystem, biology of plant litter (thatch) accumulation and decomposition, and biology and control of soilborne pathogens of cereals.

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