# Fusarium Blight of Turfgrasses — An Overview

**by Houston B. Couch**

In 1959 a severe foliar blighting was observed on Merion Kentucky bluegrass in southeastern Pennsylvania. The symptom pattern did not fit that of any of the known foliar diseases of turfgrasses, and isolations from diseased leaves only yielded pathogenic organisms that were known to incite symptoms distinct from those observed for the disease in question.

During 1960 and 1961 this same disease was found on Merion Kentucky bluegrass, bentgrasses, and creeping red fescues in eastern Pennsylvania, eastern Ohio, eastern New York, New Jersey, Delaware, Maryland, and the District of Columbia. Beginning in 1960 and continuing through the following three growing seasons, plant and soil samples were collected from the geographic areas that showed the characteristic symptoms of the disease. Isolations from the diseased leaves were attempted in order to determine if pathogenic fungi were present. The soil samples were also checked for the presence of parasitic nematodes. Certain of the soil samples were found to contain parasitic nematodes of the genera *Hoploaimus, Xiphinema, Paratylenchus,* and *Tylenchorhynchus.* In some samples the populations were high enough to produce foliar stress. However, there was no consistency among the samples — neither in the frequency of occurrence of a given genus nor in populations high enough to cause foliar symptoms. Furthermore, many soil samples obtained from turfgrass that showed symptoms of the disease were free from parasitic nematodes. On the basis of this evidence, it was concluded that the disease was not caused by nematodes.

The isolations from diseased leaves consistently yielded two fungus species — *Fusarium roseum* and *Fusarium tricinctum* f. sp. *poae.* Both of these organisms were known to be turfgrass pathogens, but neither had been identified as foliar parasites. *Fusarium roseum* was known to cause a root and crown rot of turfgrasses, while *tricinctum* had been recognized for several years as the cause of "silver top," a disease of turfgrass floral tissue. Pathogenicity tests with isolates of these two fungus species were made on Merion Kentucky bluegrass, Highland bentgrass, and Pennlawn creeping red fescue. While some of the isolates were weakly pathogenic, a very high percentage of those tested incited 100 percent foliar blighting within two to five days from the time of inoculation.

On the basis of (a) consistency of isolation from diseased turfgrass plants over a broad geographic area for several growing seasons, (b) the general lack of consistency of isolation of other microorganisms, and (c) the high degree of pathogenicity of *Fusarium roseum* and *Fusarium tricinctum,* we concluded that these two organisms were the actual incitants of the disease. With further research it was learned that the total syndrome of the disease consisted of two phases — a blighting of the

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leaves, and a crown and root rot. Because of the predominant leaf-symptom pattern, we named the disease "*Fusarium* blight."

## SYMPTOMS

### Leaf Blighting Stage

In overall view, affected turfgrass stands first show scattered, light-green patches 2 to 6 inches in diameter. Under environmental conditions favorable for disease development, the color of these patches changes in 36 to 48 hours to a dull reddish brown, then to tan, and finally to a light straw color. Initially, the shapes of the patches are elongated streaks, crescents, or circular patches.

The most characteristic feature of the gross symptomatology is seen in the later stages of disease development, when more or less circular patches of blighted turfgrass 1 to 3 feet in diameter are present. Light tan to straw colored, these patches often have reddish-brown margins 1 to 2 inches wide and contain center tufts of green, apparently unaffected, grass. This combination produces a distinctive "frog-eye" effect. When optimum conditions for disease development exist for an extended period of time, these affected areas coalesce. As a result, large areas of turfgrass may be blighted. Leaf lesions originate both at the cut tip and at random over the entire leaf. At first, lesions appear as irregularly shaped, dark-green blotches. These rapidly fade to a light green, then assume a reddish-brown hue, and finally become a dull tan. Individual lesions may involve the enitire width of the leaf blade and may extend up to ½ inch long.

### Root Rot State

Turfgrass plants affected primarily by the root rot phase of the disease are stunted, pale green in color, and

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do not readily recover from mowing or adverse weather conditions. Their roots are characterized by a brown to reddish-brown dry rot. As the disease progresses, these roots become darker in color due to the colonization of soil saprophytes. During periods of relatively high soil moisture, the pinkish growth of *Fusarium roseum* and *F. Tricinctum* can be seen on the root and crown tissue near the soil surface.

## DISEASE CYCLE

### Sources of Inoculum

Both species of *Fusaria* have been reported to be transmitted on turfgrass seed. Also, they are known to be capable of surviving in the soil as saprophytes. These two sources constitute the main reservoirs of primary inoculum for the development of the disease in newly seeded stands of turfgrass. In established turfgrass, the main sources of inoculum are dormant mycelium in plants infected the previous season and thatch that has been colonized by the pathogens.

### How Fusarium Penetrates Leaves

Leaves are infected both by germinating spores and by mycelium from the saprophytic growth of the pathogens on the thatch and other organic matter. Most of the primary infections probably originate from the thatch. Spores germinate 12 hours from the onset of favorable environmental conditions. Penetration of intact leaf surfaces occurs at the junction of epidermal cells. At the points of direct leaf penetration, there is no evidence of degradation of the host cell walls. The most common area of penetration of foliage by the pathogens appears to be cut ends of the leaves. With both direct penetration and entry through cut leaf tips, the fungus grows between the cells over an area of 12 or more cells and then becomes intracellular. This explains the sudden appearance of large blotches on the leaves, instead of small spots that progressively become larger.

### Optimum Conditions for Disease Development

Certain isolates of *F. roseum* and *F. Tricinctum* have been shown to vary in their temperature requirements for optimum pathogenicity. As a general rule, however, the foliar stage of *Fusarium* blight is most severe during prolonged periods of high atmospheric humidity with daytime air temperatures of 80° to 95° and night air temperatures of 70° F. or above. Turfgrass grown under deficient calcium nutrition is more susceptible to *Fusarium* blight than well-nourished turfgrass. Incidence and severity of the disease is also greatest under conditions of high nitrogen fertilization. The development of *Fusarium* blight has been reported to be greater in turfgrass when the soil moisture content has been allowed to be extracted to the permanent wilting percentage.

## CONTROL OF FUSARIUM BLIGHT

### Cultural Practices

While high nitrogen fertilization does increase the susceptibility of turfgrass to *Fusarium* blight, it is unlikely that a significant reduction of the disease can be effected by reducing nitrogen levels. In general, the level of nitrogen fertilization required to significantly reduce the severity of *Fusarium* blight is well outside the range necessary to meet the basic nutritional requirements of the grass. From a field standpoint, then, nitrogen fertilization, and its effects on the disease, should be considered with respect to thatch management.

Since the thatch serves as the major reservoir of inoculum in established stands of turfgrass, a successful program of *Fusarium* blight control requires that the quantity of this material be held to a minimum consistent with the proper management of the grass species in question. For most turfgrasses, this optimum thickness is approximately ½ inch. In order to keep the *Fusarium* blight potential of a stand of turfgrass to a minimum, therefore, increases in the rate of nitrogen fertilization should be balanced with concurrent increases in the intensification of the thatch management program.

**Host Resistance**

Ranked in order of susceptibility to *Fusarium* blight, the bentgrasses are the most prone to the disease. The Kentucky bluegrasses are next in susceptibility. The fescues are most resistant. Among certain varieties of Kentucky bluegrass, the range of susceptibility to *F. roseum* and *F. tricinctum* is determined by a complex interaction of air temperature and pathogen and host genotypes.

**Chemical Control**

A preventive fungicide program, coupled with that control, is essential for effective control of *Fusarium* blight. The fungicide application should be made immediately after the first occurrence of night temperatures that do not drop below 70°F. For most effective control of *Fusarium* blight, spray 1,000 square feet with 6 gallons of water containing 5 to 8 ounces of benomyl 50-percent wettable powder. The total amount of benomyl applied to the turfgrass within one calendar year should not exceed 8 ounces.

# Factors Affecting Fusarium Blight Development

**by Herbert Cole, Jr.**

This symposium provides a unique opportunity to explore in depth a disease that remains an enigma to all who work with turf. From the view of the research scientist, it is a frustrating challenge to gain understanding. From the view of the golf superintendent with bluegrass fairways, it has become an impossible monster. The papers in this symposium will, we hope, present the best knowledge currently available about *Fusarium* blight. There will not be agreement among the participants; in fact, agreement will be out of the question. Each view will be based on the geographic region and experience of the researcher.

The following discussion of factors affecting *Fusarium* blight is based on my personal observations in Pennsylvania and the mideastern United States, complemented by a review of the available research literature. I believe that we do not fully understand *Fusarium* blight development even 10 years after the report of its first occurrence and development (Couch and Bedford, 1966). Our lack of understanding includes all aspects of the disease: symptoms, turf age, water, grass nutrition, thatch, varietal susceptibility, and control practices. Some researchers believe the disease differs in symptoms as well as infection cycle in the various geographic areas of its occurrence. Most, if not all, of the experimental research on the infection cycle of the disease has been done with seedling grass plants in growth chambers of greenhouses. The problem in the field is associated with aging of turf stands (three years and older), yet most of the researcher has been done with seedlings. Our knowledge with other plants diseases has always indicated that it is questionable to use seedlings to study a disease of mature or aged plants. Because of this, we desperately need new disease-cycle research on mature turf.

We are not certain if the predominate problem is a foliar blight phase or a root and crown rot infection phase. On seedling and mature turfgrass in a dew chamber, foliar lesions develop. However, on the golf course or home lawn during dry weather and moisture stress, turf may wilt and die in a period of days with no clear foliar lesion picture — merely badly rotted crowns and portions of roots. Californians feel strongly that in the West only crown and root rot are involved; in the East the battle rages between the foliar blighters and the nematode-root rot complexers. At this time we just don't have an understanding of the Midwest-Eastern problems. I believe the failure of classic protectant fungicides to provide control suggests a major role for the crown and root rot hypothesis in the East also. No one has reproduced the frog eye, ring, or serpentine symptom through artificial inoculation, in either the greenhouse or the field. Classic foliar infection epidemiology cannot explain a ring or a frog-eye tuft in the center of a dead area. No other foliar-infection fungus disease produces similar symptoms on plants, including the grasses. The ring or frog eye seldom or never occurs in the Far West. To my knowledge, no turf pathologist has attempted to explain why rings or frog eyes may occur.

Most researchers would agree that the major factors influencing disease development include the physical and biological environments, especially cultural practices that affect these environments. The major factors that most of us would agree upon in terms of importance in disease development are grass variety, turf age, temperature, moisture and irrigation, thatch, and nitrogen fertilization. The role of plant parasitic nematodes in predisposing turf to *Fusarium* blight remains highly controversial at this date. A serious study of the disease should include review of all the papers listed in the references, among others. In particular, the research and review papers of Cook (1968, 1970), who has worked extensively with a *Fusarium* root and crown rot of moisture-stressed winter wheat, may be among the most pertinent in understanding *Fusarium* blight of turfgrass.

*Fusarium* blight is primarily a disease of bluegrass fairways of golf courses and intensively managed bluegrass home lawns. Although some research would suggest that greenhouse growth chamber studies show bentgrass is most susceptible, the field experience indicates that in practice bentgrass green, tees, or fairways are seldom affected. It would seem this lack of disease is due to the vigorous nature of bentgrass summer growth and stolon production coupled with regular irrigation intervals. In the East we are seeing some problems on fescue and ryegrasses but certainly not any remotely approaching bluegrass disease incidence. Merion is the variety with by far the most problems. The new varieties vary in susceptibility but their ultimate field response is not clear. *Fusarium* is a highly variable fungus genus. Research so far suggests that there will be races and strains of the *Fusarium* organism interacting with different species and strains of grass. A variety may be resistant one place and susceptible in another. In all probability the dense, vigorous, decumbent bluegrass will have problems with the disease if grown widely.

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