FUSARIUM BLIGHT--A CONTINUING PUZZLE

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Introduction

Fusarium blight is a severe mid-summer disease of Kentucky bluegrasses (Poa pratensis) on home lawns, golf course fairways, and other recreational areas. The use of the bluegrass cultivar Merion is widespread on such areas because of its resistance to other diseases, vigor of growth and recovery, and excellent play characteristics. Merion is particularly susceptible to Fusarium blight, and, because of its widespread use, loss of recreational and amenity turf to this disease can be severe in mid-summer. Fusarium blight is also an important concern of the sod production industry, and is causing increasing problems for these enterprises.

The disease was first described by Couch and Bedford (15), who observed it on Merion Kentucky bluegrass in southeastern Pennsylvania. During 1960, 1961, and 1963, the disease became epiphytotic on bluegrass and bentgrass strands in Ohio, New York, New Jersey, Delaware, Maryland, and the District of Columbia (15).

Since Couch and Bedford (15) first described Fusarium blight, a great deal of research effort has been expended in attempts to understand and control this disease. Much of the research to date has led to contradictory conclusions, and control of Fusarium blight is still a difficult problem. In spite of intensive study, many aspects of Fusarium blight remain unexplained, including the role that Fusaria play in the development of the field symptoms. At this time, it is not possible to say with confidence what biotic and environmental factors may be involved in the etiology of Fusarium blight.

Disease description

Couch and Bedford (15) reported that disease symptoms initially appeared in the field as scattered light green circular patches 5-15 cm in diameter. In later stages of disease development the patches were 0.3-1 m in diameter, light tan in color, and often contained center tufts of unaffected grass, producing a characteristic "frog-eye" appearance. In addition to "frog-eyes", affected areas also appeared as crescents, streaks, and circles. Couch (14) postulated that both a leaf-blighting and a root rot phase may occur.

Bean (2,3) reported that Fusarium blight infection centers were small (dollar spot size) at the outset and increased in size as the disease progressed. In his observations, "frog-eyes" were rare, and the disease in the field was confined to the crown area. Endo and Colbaugh (19) described Fusarium blight of Kentucky bluegrass in California as being confined to the stem base and crown. "Frog-eyes" were not observed, and all plants within a blighted area were killed.

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Fulton (22), describing Fusarium blight in Pennsylvania, reported that the first symptoms noted were wilt and darkening of scattered areas of turf in the heat of the day. Within 48 h, these areas took on a permanently-wilted, gray-green appearance, and then collapsed and bleached to a tan color. "Frog-eyes" were common, and the size of the affected area did not increase from the time the darkened patch was observed to the time the plants collapsed and turned tan. In severe infections, these patches coalesced to form large areas of blighted turf.

Leaves, crowns, and roots of Merion Kentucky bluegrass from field infection centers of Fusarium blight were examined histologically (35). Specimens from the "frog eye", dead ring, and periphery of the dead ring exhibited varying degrees of vascular plugging in roots and crowns. A coarse, septate mycelium atypical of Fusarium in morphology and staining reaction was noted in the cortex of the crowns and in the cortex, perivascular fibers, endodermis, and xylem vessels of roots from the "frog eye" and the dead ring. The fungus was most prevalent in specimens from the dead ring, and was never found in crowns from symptomless grass.

Causal organisms

Couch and Bedford (15) concluded that F. roseum and F. tricinctum were the primary incitants of Fusarium blight, based on (i) consistency of isolation over a broad geographic area for several growing seasons, (ii) the high degree of pathogenicity shown by these isolates on greenhouse-grown, seedling grass, and (iii) the lack of pathogenicity or inconsistency of isolation of other microorganisms. Koch's postulates have never been completed for Fusarium blight because attempts to produce the field symptoms on mature bluegrass with consistency by inoculation or environmental manipulation have been largely unsuccessful. Pathogenicity studies with Fusaria have usually been done by foliar inoculation of immature, greenhouse-grown grasses (9,15,18,22,28). Occasionally, greenhouse inoculation of grasses with Fusaria have been attempted by placement of the fungal propagules in the root zone (3,5,34). In all but one instance (22) greenhouse studies have not resulted in disease development that paralleled development in the field. Without a reliable inoculation procedure, it is not possible to determine with certainty whether the Fusaria that are regularly isolated from bluegrass showing symptoms of Fusarium blight are indeed the causal pathogens. A study by Smiley, et al., (41) which attempted to determine the relationship between the numbers of Fusaria isolated from bluegrass areas with the incidence of Fusarium blight, revealed a negative correlation between these two parameters.

Host and cultivar susceptibility

Fusarium blight is not a serious problem on most cool season Kentucky bluegrasses in areas where summer stress conditions are not severe. It is in the long, hot summers of the transition zone that the disease can assume epiphytotic proportions. Funk (23) observed that very few of the best bluegrass cultivars have the qualities which allow production of an attractive, compact, dense, disease-free turf in severe summer environments. Turgeon and Meyer (51) have reported that the incidence of Fusarium blight varied with bluegrass cultivar, mowing height, and fertilization level. In their studies, the order of cultivar susceptibility, from highest to lowest, was: Kenblue, Fylking, Pennstar, Merion, and Nugget. Funk (23) observed that cultivers adapted to higher mowing were severely blighted when clipped closely.

Fulton (22) reported that, in greenhouse studies, Merion was more susceptible

than Fylking or Delta. Couch and Bedford (15) reported that, in growth chamber studies with 6-week-old seedlings of Highland colonial bentgrass, Marion Kentucky bluegrass, and Pennlawn creeping red fescue, Highland was most susceptible, followed by Merion, and then Pennlawn.

Disease development

Funk (23) presented a list of factors which he believed might predispose turfgrass to Fusarium blight. These included high temperature, high humidity, recurring drought stress, reduced air circulation, excessive nitrogen, dense lush growth, thatch, close mowing, nematodes, and other diseases. Partyka (33) stated that, since the Fursarim pathogens were present in most turf areas, infection and disease development were related to stress conditions, primarily drought and high temperature. He noted that anything restricting roots; such as compaction, shortage of nutrients, thatch development, nemotodes, or insects; could predispose turf to drought stress and Fusarium blight development. He stated, further, that anything impeding water penetration; such as thatch, slope, or compacted soil; may induce water stress conditions.

Please note the conflicting conclusions as I discuss the effects on disease development of sunlight, temperature, drought, grass age, thatch, nitrogen, calcium, and nematodes.

Bean (3) concluded that severity of Fusarium blight was directly proportional to intensity and duration of sunlight. The disease was never seen in heavy shade. Pure stands of Merion were most severely infected, especially in sunny, sloping locations.

Most research into the effect of temperature indicates that Fusarium blight incidence and severity increases as summer temperatures increase. Studies employing artificial inoculation of greenhouse-grown, seedling grass reveal that disease was most severe at temperatures between 27 and 33 C, and that severity increased as temperatures increased (18,19,22,48). In the field, Bean (2) observed that the disease was active only during the warmest part of the summer, and Fulton (22) stated that temperatures of 27 C or above are necessary in the 7 to 14 days prior to the development of Fusarium blight symptoms. However, a survey of weather records for 1966 and for 1973-1978 (42) indicated that there was no apparent relationship between disease outbreaks and summer temperatures.

Most workers believe that drought stress is an important factor in the etiology of Fusarium blight (3,15,18,19,23,33,49,52). Bean (3) postulated that moisture stress was essential for the disease to occur. In greenhouse experiments with artificially inoculated seedling grass, employing soils with moisture equivalents from 20-80%, Troll (49) found infection most severe on plants growing on soil with the lowest moisture equivalents. Cutwright and Harrison (18) studied the effect of environmental factors on Fusarium infection of greenhouse-grown grass. Disease severity was greatest under low irrigation at all inoculum levels. Couch and Bedford (15), however, found no significant differences in disease incidence between any of the Fusarium isolate/soil moisture regimes which they investigated in the greenhouse. In a field irrigation plot experiment (19), as the amount of irrigation increased from marginal to abundant, Fusarium blight decreased from 40% of the plot area affected to less than 1%. Troll (49) observed that Fusarium blight was severe in Massachusetts during the moisture stress summer years of 1960-1966, but was far less severe during the wet summer of 1967. In a Merion bluegrass home lawn, no symptoms occurred in 1967, but occurred again in 1968, a year with a dry summer. Fulton (22) observed that outbreaks of Fusarium blight in Pennsylvania are almost always preceded by a period of warm, wet weather followed by dry weather. A survey of weather data in New York (42) indicated, however, that Fusarium blight outbreaks always occurred soon after a major rain.

Fusarium blight appears to be most prevalent on older, established bluegrass areas. Bean (3) observed that a stand must be 3-years-old or more before the disease will occur, and Fulton (22) reported that Fusarium blight in Pennsylvania was common on turf stands older than 5 years and was seldom seen on stands less than 2-years-old. Cutwright and Harrison (27), however, observed Fusarium blight on stands of Merion that were less than 2-years-old. Sanders and Cole (unpublished data) have observed the disease on stands of bluegrass less than 1-year-old.

There is a commonly held belief among those who work with turf that thatch accumulation predisposes turfgrass to Fusarium blight, but there is little research evidence to support this hypothesis. Bean (3), Turgeon (50), and Smiley and Craven (37) found no relationship between thatch depth and Fusarium blight severity. Turgeon (50) observed no relationship between the thatching tendency of a bluegrass cultivar and its susceptibility to Fusarium blight. On a sodded turf area, Fusarium blight was most severe where the thatch layer was thinnest (37). Smiley, et al., (41) suggested that disease incidence may be related to the thatch decomposition rate. Turgeon (50) and Funk (23) have observed coincident increase in thatch and Fusarium blight severity on field plots treated with tricalcium arsenate.

There appears to be a consistent relationship between severity of Fusarium blight and level of nitrogen fertilization. Endo and Colbaugh (19) found that the disease was most severe in the field on plants receiving high levels of nitrogen and low amounts of water. Turgeon (50) reported that certain bluegrass cultivars which had little disease at low levels of nitrogen (1 kg/93 m^2) were severely blighted at high levels (3-4 kg/93 m^2). Bean (3), however, observed no correlation, in the Washington, D.C. area, between soil nitrogen and disease severity. Greenhouse studies (18,15) showed increased susceptibility to Fusarium infection when grass was grown under high levels of nitrogen.

Of the nutrient elements other than nitrogen, the role of calcium has been investigated. Couch and Bedford (15) reported the results of a greenhouse investigation of the influence of normal and 0.1 normal levels of calcium on the susceptibility of foliage of seedling fescue, bluegrass, and bentgrass. In this study, disease incidence was significantly higher in plant groups grown under 0.1 X calcium. Bean (2) was unable to correlate disease severity in the field with low calcium levels. Application of 1 kg hydrated lime/month/93 m^2 were found to significantly increase incidence of Fusarium blight (Cole and Sanders, unpublished data).

Vargas and Laughlin (55) investigated the role of Tylenchorhynchus dubius, a migratory ectoparasitic stunt nematode, in Fusarium blight development of Merion Kentucky bluegrass. A survey by the authors indicated that Merion plants growing in blighted areas had poorly developed root systems and were associated with high soil populations of T. dubius (200-1500/100 cc soil). In two greenhouse experiments, seedling Merion bluegrass was grown in a sterile sand:loam:peat mix artificially infested with either F. roseum, T. dubius, or a combination of F. roseum and T. dubius. Based on the results of these experiments, Vargas and Laughlin (55) and Vargas (52) suggested that Fusarium blight may involve an interaction between F. roseum or F. tricinctum and T. dubius, or that T. dubius alone may be responsible. Couch and Bedford (15), however, were unable to associate high levels of parasitic nematodes with incidence of Fusarium blight. Cole (personal communication) observed that levels of parasitic nematodes were low on two Pennsylvania golf courses with histories of severe Fusarium blight. Vargas, et al., (53) and Smiley (43) have reported field control of Fusarium blight with nematicide application. Couch (16) obtained no disease suppression in field trials employing 4 nematicides.

There is a lack of consistency in results of research done at varying places

and times to determine the effect of specific environmental stresses on the development of Fusarium blight. Recently, Smiley (personal communication) has suggested that, since response to stress at the cellular level is similar regardless of the stress, Fusarium blight may be triggered by stress in general, rather than a particular stress or stresses.

Fusarium blight on newly-sodded areas

The use of sod to establish and repair lawns and recreational areas has become increasingly prevalent. Newly-installed sod can be destroyed rapidly by Fusarium blight, with severe infections occurring in sod that has been established only 1-2 years. Purchasers of such sod believe that they have been sold "diseased grass". The question which is raised in such situations is whether the nature of the sod-growing site or the installation site was responsible for the disease outbreak. Bean (3) has reported that approximately the same number of Fusarium propagules were recovered from a sod-growing site, the blighted areas in the installation site, and the blight-free areas in the installation site. Thus it would appear that potentially pathogenic Fusaria occur with approximately equal frequency in both the sod-growing and the installation sites. There may be stress factors in the transportation of sod, as well as in the environment of the installation site, which can predispose newly-laid sod to Fusarium blight. Partyka (33) suggested that sod which is allowed to dry or to heat in transit may be damaged so that Fusaria can subsequently cause disease. He also observed that sod laid down on dry soil or not watered adequately could be so stressed. He further stated that another source of stress was poor permeation of water or capillary action at the sod/soil (clay) interface, which might result in a poor root system and resultant stress. Bean (2) reported that when turf was removed from Fusarium-blighted areas and these areas resodded with 1-year-old healthy sod, the newly-sodded areas were as heavily blighted as the orginal turf by the following summer. Cole (personal communication) has stated that when Fusarium-blighted sod was transferred to a blight-free site, the Fusarium blight symptoms abated and did not return.

Control

Because of the regular association of Fusarium blight with hot, dry environments, Bean (3) recommended heavy watering for disease suppression. There is research evidence (19) that adequate irrigation suppresses Fusarium blight, however, it encourages invasion of quality bluegrass turf by annual bluegrass (Poa annua) and other weed grasses.

Funk (23) pointed out that Kentucky bluegrass cultivars orginating in areas of cool, moist summers were often severely damaged by the disease, and suggested that Fusarium blight resistance is to be found in "southern turftype" bluegrass cultivars which are heat and drought tolerant. He stated that these cultivars should do well in the southern transition zone of bluegrass adaptation where Fusarium blight is most damaging. He also suggested the overseeding of turftype ryegrasses into blighted bluegrass stands, adding the caution that improved resistance to Pythium and Rhizoctonia is needed for good summer performance of ryegrass in the humid summer heat stress region.

Turgeon (50) suggested, in addition to the "plant-oriented" control approach of resistant Kentucky bluegrass cultivars, an "environmental-oriented" approach of avoiding excessive nitrogen fertilization during spring, providing adequate moisture for turfgrass survival during stress periods through irrigation, performing approprite cultivation practices to control thatch and alleviate soil compaction, and applying effective fungicides.

In the period from 1966 through 1976, many chemical control experiments were conducted. During the early part of this period, most work (2,4,15) involved the contact fungicides, such as mancozeb (Dithane M-45), thiramorganic mercury (Tersan OM), anilazine (Dyrene), Difolatan, chlorothalonil (Daconil), and others. Although a partial level of control was obtained in a few instances, the level of disease suppression was not satisfactory for practical use. For example, Bean, et al., (4) presented the results of a series of fungicide experiments conducted during 1965 and 1966. A single application of hydrated lime (11 kg/93 m^2) reduced Fusarium blight four fold, and Tersan OM applied in weekly sprays provided significant disease suppression. On the other hand, Difolatan, Dyrene, Dithane M-45, Daconil 2787, and Panogen turf fungicide provided no significant benefit. In spite of favorable small plot results, however, neither hydrated lime nor Tersan OM achieved commercial success. Applications of hydrated lime (1 kg/mo/93 m^2) were found by Cole and Sanders (unpublished data) to increase significantly the incidence of Fusarium blight.

Soil fumigation as a method to control Fusarium blight was tested by Cutwright and Harrison (17). In fall 1966, sod was stripped from a test area that had been severely blighted, replicated plots were fumigated with three soil fumigants, and the area was seeded with Merion Kentucky bluegrass two weeks after fumigation. Only a few scattered Fusarium-blighted areas developed in the experimental area during the following summer. The disease ratings for 1968, however, indicated that none of the treatments effectively controlled the disease over even a short 2-year period.

The introduction of systemic fungicides provided the first highly effective chemical treatment. Cutwright and Harrison (17), in 1970, reported excellent control of Fusarium blight with weekly preventive sprays of benomyl. Muse (29) reported control of Fusarium blight with thiophanate methyl and benomyl when they were applied as drenches, and Fulton, et al., (21) obtained suppression of the disease with pre-symptom application of foliar sprays of triarimol and benomyl, followed by a drenching water irrigation. Vargas and Laughlin (54) reported excellent control of the disease with regular preventive applications of benomyl as a drench (foliar sprays were not effective), but no control with Dithane M-45 or thiabendazole, regardless of mode of application. In the ensuing years, various researchers (6,10,11,24,26,31,44,45,54) have obtained successful control of Fusarium blight through preventive and curative applications of benzimidazoles, triarimol (EL 273), and fenarimol (EL 222). However, in 1975 and 1976, control failures were reported with benzimidazole-derivative fungicides due to apparent pathogen resistance to the fungicides (8,46).

During the 1977 and 1978 growing seasons, excellent control of Fusarium blight was obtained with the experimental fungicides, BC 6447 (7,27,80), CGA 64251 (27,30, 38), DPX 4424 (38), and RP 26019 (7,38,43). In depth studies with triadimefon (BC 6447) (34) and iprodione (RP 26019) (39,40) demonstrated no in vitro fungitoxicity to Fusarium spp. Iprodione was found to increase sporulation of Fusarium spp. in vitro and to increase the number of Fusarium propagules in treated field soil (39,40).

Discussion

The literature on Fusarium blight is extensive compared with reported work on other diseases of turfgrass. This attests both to the tenacity of turf researchers in attempting to elucidate the dynamics of symptom development and the elusive nature of the problem. In spite of extensive study, many aspects of Fusarium blight remain unexplained, and information is still lacking in several critical areas. A major shortcoming of inoculation studies to date is that much of this experimental work has been done in moist chambers with seedlings, while in

the field the disease is most damaging on mature bluegrass under stress. Relating growth chamber experiments on seedlings to field situations involving mature stands is precarious. Although potentially pathogenic Fusarium spp. are regularly isolated from turfgrass showing symptoms of Fusarium blight, no one has been able to induce the field "frog-eye" or "dead ring" symptom by inoculating turfgrass with these Fusaria. In addition, the experimental fungicide, triadimefon which provided complete suppression of the field symptoms of Fusarium blight, exhibited lack of in vitro fungitoxicity to Fusarium spp. isolated from diseased turfgrass (34). Neither could fungitoxicity to these pathogens be demonstrated in bioassays of Kentucky bluegrass which had been treated with triadimefon. Fungitoxicity of triadimefon to other turfgrass pathogens was readily demonstrable using both of these techniques. Thus, one can reach the peculiar conclusion that the designated pathogen(s) have not incited the field symptom, and the chemical which suppresses the field symptom does not control the pathogen(s). However, these results more probably indicate that Fusarium spp. are pathogenic within an unexplained biotic or environmental complex which produces the field symptoms of Fusarium blight.

The literature indicates that the majority of the Fusarium blight outbreaks are associated with moisture stress. Research on related diseases may by drawn upon to explain several of the puzzling aspects of moisture stress and disease development. The works of Cook and Papendick (12) and Papendick and Cook (32) regarding plant water stress and soil water potential and the effects of these factors on colonization of soil organic matter and host plants by Fusarium spp. may be particularly relevant to an understanding of Fusarium blight. Foot rot of dryland wheat, incited by F. roseum f. sp. cerealis 'Culmorum', was demonstrated to be most severe under high nitrogen regimes and in seasons of drought stress. Fusarium spp. were able to colonize organic matter under low soil moisture conditions which inhibited the activity of antagonistic microorganisms. In a like manner, the fungi were able to colonize wheat plants rapidly under internal moisture stress. High nitrogen fertilization increased moisture stress within wheat plants, accentuating colonization by Fusarium sp. This increased internal water stress probably was not due to a direct nutritional effect, but rather to indirect effects produced by larger root systems which increased water extraction from the soil, and greater leaf areas which increased transpiration.

Growth chamber studies indicate that bentgrass is most susceptible to Fusarium blight while field observations suggest that the disease is primarily a problem of bluegrass. Turfgrass management practices may explain this discrepancy. Traditionally, bentgrass is grown under adequate, or often excessive irrigation, especially during mid-summer. On the other hand, bluegrass fairways are traditionally kept "dry" to minimize invasion by undesireable grasses. This difference in irrigation practices may explain the absence of Fusarium blight in most bentgrass areas.

At present a satisfactory control system for suppression of Fusarium blight is not available. Practical research aimed at identifying control measures is seriously hampered by the lack of conclusive information about every aspect of disease development. The lack of an effective inoculation technique makes it impossible to determine whether the Fusaria which are regularly isolated from bluegrass showing symptoms of Fusarium blight, are indeed not the causal pathogens or simply that the inoculation procedure employed was not appropriate. We have, in the past, assumed that the latter was the case, when, in fact, the former conclusion is probably equally legitimate. At present, we have no certain information about causal fungi, where or how they colonize the host grass, what environmental conditions are required for disease development, and why some grasses are resistant to the disease. Without such essential basic knowledge, research and control decisions have little basis in biological fact, and often result in unproductive, random effort.

Fusarium blight remains an enigma to all who work with turf and is not fully understood even 13 years after the first report of its occurence. The foliar diseases of turfgrass are relatively well understood, and therefore, may be chemically and/or culturally controlled. This is largely due to the relative simplicity of foliar disease syndromes, compared with the complex environment in which soil-borne diseases, such as Fusarium blight, develop. Soil and multiple pathogen complexes, as they relate to symptom development, have been largely unstudied because of the great difficulties which are encountered in working with the dynamic and interdependent systems of soil environment and microflora. Fusarium blight is only one recognized disease entity in a large number of turfgrass diseases, as yet unstudied, but strikingly similar in symptom presentation. There are, at present, many generally circular symptom patterns on turfgrass which are partially or completely unelucidated. These patterns vary widely in size, shape, character, and environments under which expression occurs, and have been increasingly noted in recent seasons, perhaps coincident with the use of long-acting systemic fungicides. Because of the varying conclusions from research on Fusarium blight, the possibility exists that more than one disease entity has been studied--with the vinculum of consistent isolation of pathogenic Fusaria from field infection centers.

Fusarium blight, and other soil-borne diseases, have been and are increasingly becoming a serious threat to turfgrass, particularly at a time when the soil microflora is being modified by the use of long lasting systemic fungicides. Elucidation of the etiology of Fusarium blight is essential to the development of soundly-based control practices, and may lead to the development of research protocols which can be utilized in the study of other soil-borne diseases of turfgrass.

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