SUMMER PATCH AND NECROTIC RING SPOT: NEWLY DESCRIBED COMPONENTS OF THE FUSARIUM BLIGHT SYNDROME[1]

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Fusarium blight is the name for a patch disease which has caused much concern to the managers of fine turfgrasses for many years. The disease was first reported in the Atlantic Coastal States (Bean, 1966; Couch and Bedford, 1966) and is now recognized throughout North America, in nearly all regions where Kentucky bluegrasses are grown (Smiley, 1980).

THE ENIGMA OF FUSARIUM BLIGHT

Many of the environmental conditions and management practices that adversely affect the growth of bluegrass plants (Sanders and Cole, 1981; Smiley, 1980) also predispose this grass to Fusarium blight symptom development. Since there is considerable climatic diversity among the regions where the disease occurs, it is also likely that the stresses on the grass species and cultivars in each region differ considerably. Perhaps it is therefore to be expected that minor differences will occur in the relative efficiencies of disease control practices in the various geographic regions. With Fusarium blight, however, it became well known that very large differences in control measures occurred among regions and also on affected turfs within local areas. Some bluegrass varieties were, for instance, shown to be highly resistant to the disease in some areas and very susceptible in others. Water management practices which provided relief from the disease in some areas differed from those that worked best in other regions. The disease seemed to occur only in fully sunlit sites at some locations, and to also occur in shaded areas at other locations. The precise weather patterns preceding outbreaks of the disease seemed to differ from one site or region to another. With the development of more selective fungicides it became even more apparent that very large differences existed among patches of "Fusarium blight", and that these differences could not be separated on the basis of visual symptoms. Patches of Fusarium blight were easily controlled by a particular fungicide at one site while essentially identical patches at another site were immune to applications of the same fungicide.

Although none of these anomolies was understood, it was thought that they resulted either from differences in the species or isolates of Fusarium that were attacking the plants, or from changes in the interactions among

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plant cultivars and the environments and management systems in which they were being grown. A strong hindrance to research on this disease was that scientists could not experimentally induce the disease to occur at selected field sites or in the greenhouse. They were also unable to transfer the disease from one site to another so that its variants could be critically examined. The inability of scientists to manipulate the disease system hindered progress on disease control strategies that are so badly needed by the turfgrass managers. In view of the strong but indirect experimental and observational evidence that the Fusarium fungi could not cause this patch disease to occur, the original hypothesis was challenged (Sanders and Cole, 1981; Smiley, 1980) and the uncertainties were noted by altering the disease name to Fusarium blight syndrome (Smiley, 1983). Studies on the etiology of Fusarium blight syndrome became re-emphasized, and this work has been especially productive in the states of NY, RI, WI, PA, and WA. Research on spring dead spot bermudagrass in California has also yielded very valuable knowledge.

NEW INSIGHTS

Research during the 1980's has led to the recognition that there are at least two distinct components of Fusarium blight syndrome, and that they are caused by two different pathogenic fungi which cause disease symptoms that are essentially identical. It is likely that additional research will reveal even more complexity within this group of patch disease. Over the years the concept of "Fusarium blight" had become a combined name for these and perhaps even more individual diseases. The reason that the Fusarium fungi were originally implicated is that they are often the most abundant fungi in grass during the summer, and are therefore usually present on plants which are unduly stressed by environmental conditions or by root rots caused by other pathogens. It is also quite true that the Fusarium fungi can cause leaf spots and crown and root rots very similar to those caused by Drechslera, Bipolaris, Curvularia and other fungi; but the affected plants tend to be diffusely spread throughout the canopy or they may occur in irregularly shaped patches. For instance, when Drechslera poae is known to be the pathogen, the disease is named Drechslera leaf spot or melting out. The crown and root rot stage of this disease has been given the descriptive name melting out. For this reason, Fusarium crown and root rot, caused by one or more Fusarium species, is analogous to the disease called melting out. The major difference is that melting out and Fusarium crown and root rot are caused by different pathogens. The preferred term for this irregularly patchy to diffusely distributed disease is Fusarium crown and root rot, to avoid confusion with the distinctive patch disease which has been called Fusarium blight for several decades. These changes in terminology are described more fully by Smiley (1983). Furthermore, I recently proposed to turfgrass pathologists that "the name Fusarium blight is therefore rejected for well-patterned circular to arc-shaped patch disease, where Fusarium spp. are secondary colonists of senescing tissue. Fusarium spp. should be recognized as causal agents only of diffusely distributed or irregularly patchy leaf spots and crown and root rots" (Smiley, 1984).

The preceding information serves as the background for describing the newly named patch diseases. The diseases are not new, they were just

segregated out of a more complex system, and then renamed to provide greater accuracy in our communications about their behavior and control. Likewise, these new findings lead me to question our ability to interpret most of the early research, including much of my own, where the primary pathogen's identity was unkown. We must now determine which control measures are most appropriate for controlling each newly identified disease. This work has just begun.

NEW FUNGI AND NEWLY DESCRIBED DISEASE

A constant association of two new pathogens with patches of the "Fusarium blight syndrome" in New York was recently reported (Smiley and Fowler, 1984a). The fungi are Phialophora graminicola and Leptosphaeria korrae, and they may be present singly or in combination in any one patch. L. korrae was previously known to occur only in Australia, where it causes spring dead spot of bermudagrass. Diagnoses conducted in my lab by Melissa Craven Fowler, and simultaneously by others in other states, have now indicated that L. korrae is associated with "Fusarium blight syndrome" in many states, including New York, New Jersey, Connecticut, Rhode Island, Massachusetts, Pennsylvania, Michigan, Wisconsin, Colorado and Washington. This fungus is now also known to cause spring dead spot of bermudagrass in California but, to my knowledge, has not yet been found associated with this disease in the eastern United States. P. graminicola had not been previously described as having pathogenic capabilities, although it is a well known inhabitant of grasslands and turfs throughout the world. Isolates of P. graminicola have been found in patch-affected bluegrasses that we have examined from many of the eastern and north central states and Nebraska, and from spring dead spot-affected areas in North Carolina. The importance of the latter observation is not yet known. Since the geographical distributions of these pathogens is not known, the areas reported above must be considered as only a rudimentary listing.

Both of the newly described pathogens are ecologically and taxonomically related to the take-all patch fungus, Gaeumannomyces graminis var. avenae. It is therefore of no surprise that the disease caused by these three fungi appear to be rather similar in appearance. In fact, patches on one bentgrass putting green in New York appear to be caused by P. graminicola rather than by G. graminis var. avenae. Perhaps this is only of academic interest, in that it is not likely to influence one's approach toward controlling the disease. These two pathogens appear rather similar in their sensitivities to environmental conditions and control measures. Unfortunately, the same is not true of Leptosphaeria, and the disease it causes must therefore be approached somewhat differently than those caused by Phialophora or Gaeumannomyces. Therein lies the need to assign separate names to each of the components of "Fusarium blight syndrome". The disease caused by P. graminicola is named summer patch and that caused by L. korrae is named necrotic ring spot (Smiley, 1984). These names were accepted by a concensus of plant pathologists during a meeting at Guelph, Ontario in August, 1984. Although specific charcteristics of these diseases are not yet accurately defined, the following sections relate what I consider to be the current state of knowledge.

SUMMER PATCH

This name is derived from the fact that the disease's symptoms appear during warm to hot periods of the year. This presumably is also the period when the pathogen's attack on the root system is most effective. P. graminicola grows most rapidly at 80 to 85 F, provided conditions of moisture, pH and other parameters are not limiting. Rates of root regeneration by the bluegrasses also tend to be at their lowest peak during periods of high temperatures. The net result of this host:pathogen interaction is a root rot which causes the wilting and death of the plant, with or without additional infections by other common fungi, such as Fusarium species.

Symptoms

The affected areas of dead plants are small at first, and grow into larger patches which may or may not have living tillers intermixed with the dead tillers. When the environmental conditions become more favorable for root growth than for pathogen attack, the affected area recovers via encroachment of new tillers into the affected patch area. Additionally, tillers that had not completely succumbed to the root rot often become rejuvenated by the production of new roots, and thereby refill the patch-affected area. The net result is that symptoms of summer patch sometimes completely disappear during the cool weather of fall, winter and spring. When conditions again become more favorable for the pathogen than for the production of replacement roots, the disease symptoms reapper. Since the pathogen tends to keep growing in an outward direction from the original infection site, older patches often appear as rings of dead grass around tufts of apparently healthy grass. This effect has been called several names, including a doughnut shape and a frog-eye. Only the oldest patches attain this degree of development. Younger patches vary considerably in diameter and in the amount of dead tillers which occur in the affected area. Refer to the Compendium of Turfgrass Diseases (Smiley 1983) for additional details and color photographs of the characteristics described for Fusarium blight syndrome.

Time of occurence

Summer patch occurs in New York during hot periods in June through September. Outbreaks which occur early in this period can then remain active throughout the summer, become inactive during the remainder of summer, or go from a period of inactivity to renewed activity. Each of these scenarios depends upon the balance of environmental and management conditions which presumably regulates the net outcome of the pathogen's destruction of roots and the plant's production of replacement roots.

Predisposing Conditions

Summer patch often follows hot, sunny days which occur soon after very wet periods during summer. The pathogen is favored by very moist conditions, and apparently becomes most active during prolonged rainy periods or frequent

irrigations, especially when the temperature is hot. Ironically, sudden droughts can also cause abrupt development of summer patch symptoms, especially on irrigated turfs. In the case of drought, it is unlikely that the symptoms are the result of recent pathogenesis, but rather from the death of plants which had remained symptomless because the level of infection was insufficient to cause death under the conditions which preceded the drought.

Infected grasses are also predisposed to earlier than necessary death when they are mowed very short, as on many golf course fairways. But since it is also true that lawn grasses are affected by this disease, the mowing height is only one of the known stress factors. Other stresses that appear to expedite the development of summer patch include unbalanced fertility (especially low phosphorus and potash, or excessive nitrogen), some annual grass herbicides (such as crabgrass and annual bluegrass control chemicals), very high or very low pH, and excess heat accumulation (fully sunlit sites, especially on south-facing slopes or on areas near roads, sidewalks or buildings).

Disease Control

Cultural control strategies - The most important disease control strategy on existing turfgrasses is to eliminate, in as much as possible, any stresses which tend to amplify the expression of symptoms. A turfgrass manager can accomplish this by favoring root production and by avoiding conditions favorable to growth of the pathogen. This is paricularly applicable to management of water, fertility, soil pH, soil compaction, and herbicides. Light syringes of water during mid-day can also be used to reduce the heat accumulation in turfgrasses.

Management options are broader when new constructions are planned in areas where summer patch is known to occur. Efficient drainage and irrigation systems should be utilized. Soils with favorable textural classes should be used wherever possible, and the soil structure should be protected from compaction wherever it is possible to do so. The soil should be properly tilled for seeding or sodding, and adjustments made in fertility and pH. When attempts are made to construct artificial mounds or other terrain features, it should be recognized that such efforts usually succeed in creating an initially appealing view, but that they are soon converted into eyesores because it is often impossible to manage turf on such features. Patch diseases are but one of the problems experienced on the artistic mounds and ridges placed in some commercial turf areas.

Much controversy often arises over the appearance of patch disease in recently sodded turfs. It is very easy for the consumer to suggest that the sod was the source of the pathogen. This may or may not be true. It is very evident that the disease 1) quickly reappears when new sod is placed over poorly prepared sites that have a previous history of the disease, 2) does not necessarily appear at other sites where the disease had not been known to occur, and which were covered with sod from the same source as that at the disease-affected site, 3) is sometimes first observed on an area only after new sod has been installed, and 4) may become evident sooner on a sodded lawn than on a seeded lawn. This is not evidence that the sod was the source of the pathogen, since it could be equally true that the pathogen was present for

many years on areas where previous grass cover was resistant to the pathogen. However, it is also possible for sod to introduce new organisms into a turfgrass. To safeguard against such instances, deliveries of sod should be rejected if they contain patches of poorly rooted grass. These patches are not likely to be visible in the foliage, but will appear as circular areas in which the soil does not adhere well to the turf mat. Such areas on the underside of a turfgrass sod may be an early signal that development of a patch disease is likely, especially if other stress factors also become present.

Genetic Control Strategies

The most important long-term disease control measure involves the use of disease resistant grass cultivars and species. On lawns and fairways this consists of resistant cultivars of Kentucky bluegrasses, or of mixtures of bluegrasses with perennial ryegrass or tall fescue. The use of susceptible cultivars should not be attempted on areas known to be prone to summer patch. Although a registry of resistant grasses is yet to be developed in systems known to be affected only by P. graminicola, it appears safe to utilize the latest information that has been developed for resistance to "Fusarium blight" in experimental field plots. Turfgrass managers in New York may gain access to an extensive compilation of this data (Smiley and Fowler, 1984b) by contacting the Ornamental Horticultural agent in their County's Cooperative Extension Association. University turfgrass specialists or their agents in other states may request this publication directly from the authors. A tentative list of some bluegrasses thought to be resistant to summer patch is as follows: A-20, A-34, Adelphi, Admiral, America, Baron, Bristol, Challenger, Columbia, Eclipse, Enmundi, Georgetown, Majestic, Monopoly, Mystic, Nassau, Sydsport, Trenton, Victa, and Windsor. This list is considered tentative because screening procedures to identify resistant cultivars are yet to be conducted. We have attempted three screening procedures thus far, and have obtained three different sets of rankings for the relative tolerances or resistances of bluegrass cultivars to attack by P. graminicola. The answer we achieve differs in response to each variation in the testing method. A reliable ranking scheme therefore becomes a high priority challenge for investigations in the next few years. Current evidence also indicates that fine-leaf fescues, bentgrasses, and annual bluegrass are susceptible to attack by this pathogen.

NECROTIC RING SPOT

This name is derived from the description given to a patch disease occuring in Wisconsin. The terms indicate that dead (necrotic) rings occur in well-developed patches. About the same time that the identity and pathogenicity of L. korrae were being described for a component of the Fusarium blight syndrome in New York and in several other states, it also became apparent that the disease in Wisconsin was also being caused by L. korrae. In an attempt to avoid the use of different names in various regions, the name used in Wisconsin would become the accepted name for L. korrae-caused patch diseases of cool-season grasses. The term spot was retained to designate the similarity in cause (e.g., same pathogen) and appearance of this disease to spring dead spot of bermudagrass.

Symptoms

This disease is visually indistinguishable from summer patch. The ecology of the pathogens is similar, and these root and crown rots therefore cause the same patch and ring patterns to occur in affected turfs. Even trained mycologists cannot distinguish the difference between summer patch and necrotic ring spot unless they are lucky enough to observe fruiting bodies of L. korrae in the dead plant tissues. These structures are evidence that L. korrae is present, although it does not exclude the possible co-existence of P. graminicola in the same turf. Since L. korrae is the pathogen which appears to be the more difficult to control, it may not be very important to also determine if P. graminicola is also present. If no fruiting structures are present at the time when a diagnosis is needed, prolonged, expensive and laborious laboratory and/or greenhouse diagnostic procedures must be used to determine the incitant's identity. Guidelines for this diagnostic procedure are being prepared (Smiley et al., 1985). Although summer patch and necrotic ring spot are very difficult to distinguish when their symptoms first appear during summer, it is possible to predict that the patch is necrotic ring spot if it first appears during spring or autumn.

Time of Occurrence

Necrotic ring spot occurs in New York from mid-spring through late-autumn. Patches at some locations appear only during mid-summer, and become inactive and recolonized by grass or weeds during the autumn. At other sites the disease more closely follows the behavioral pattern of a cool-season disease. For example, at some locations on Long Island the disease is most prevalent during July and August, while at other nearby locations the patches occur in May and June, heal over during the hotter months, and reappear when the weather becomes cooler in September and October. This anomoly presumably reflects the relative balance between the overall health and replacement rate of roots and the activity of the pathogen. This pathogen grows most rapidly from 60 to 80 F, which is a range considerably broader than that for P. graminicola. The necrotic ring spot pathogen is therefore capable of infecting grass during mild to warm conditions, and its ability to cause a patch is likely to be dependent upon the overall health of the plant root systems. Our experimental work has shown that this pathogen is capable of killing circular patches of plant roots without causing symptoms in the foliage. If this were to happen in the field, the plants would look perfectly healthy until a period of increased environmental or management stress occurred, and then the patch of rootless plants would suddenly die. The time of death would, therefore, be totally unrelated to the time when the pathogen was most active. It is interesting to note here that this scenario is precisely what is also thought to happen in the spring dead spot disease, the unifying factor being that the warm-season (C4) grass dies during the stress of winter and the cool-season (C3) dies during the stress of summer.

Predisposing Conditions

Very little is known about the conditions which lead to necrotic ring

spot symptom development. The disease occurs over a wide range of temperature and water conditions. The influence of water is not well defined, but it is clear that this disease occurs on both irrigated and nonirrigated turfs. L. korrae is well adapted for growth in relatively dry soils as well as in wet soils.

Disease Control

Cultural Control Strategies--At present it is best to follow the suggestions made for suppressing summer patch.

Genetic Control Strategies--The bluegrass, bentgrass, bermudagrass, and fine-leaf fescue cultivars that have been examined thus far are all highly to very highly susceptible to attack by L. korrae. None can be recommended at this time for suppressing necrotic ring spot, but this is not meant to infer that some resistant cultivars will not be found during additional investigations. The perennial ryegrass and tall fescue cultivars examined thus far have much higher levels of tolerance than any of the previously mentioned grasses. Wherever it is possible to do so, emphasis should currently be placed upon the use of these grasses in new seedings, and on perennial ryegrasses in overseeding programs. The use of large percentages of these grasses in mixtures with Kentucky bluegrasses, for instance, should mask or prevent the development of this patch disease.

Chemical Control Strategies

Fungicides which are highly toxic to the necrotic ring spot pathogen include Banner (yet to be registered), Rubigan, and Tersan 1991, or their equivalents. Bayleton and Chipco 26019 have not been toxic to the pathogen in laboratory test, and have not controlled the disease in the field. This separation has proven invaluable in diagnostic procedures, in that, to my knowledge, L. korrae has now been isolated from all locations where Bayleton has proven ineffective for controlling "Fusarium blight" of Kentucky bluegrass. In New York, on the many sites where Bayleton gives superb control of patches, we feel that the disease is caused by P. graminicola.

The proper timing of fungicide applications for controlling necrotic ring spot is unknown at present. Since the pathogen grows actively over a broad range of temperatures, it is possible that autumn and spring applications could prove to be preferable to summer applications. But additional research is needed to examine this possibility as a control strategy. In any case, it is vital to interrupt the disease process early enough to prevent severe loss of roots at any time during the growth of cool-season species. If this is not accomplished, then any high level of plant stress at any time of the year could cause a sudden appearance of foliar symptoms, even when the actual root damage may have occurred months earlier.

CONCLUSIONS

The recent recognition that there are at least two diseases which have

been masquerading as "Fusarium blight" has placed even more uncertainty into the already ambiguous nature of this disease complex. By separating summer patch and necrotic ring spot into two distinct components, scientists can now initiate studies specifically aimed toward controlling each of these diseases. Our ability to control these diseases will become much more precise as soon as the results of extensive new investigations in many states are released. The experiences of scientists working on take-all patch and spring dead spot now become very relevant to the studies of those working on summer patch and necrotic ring spot. This uniting of efforts will undoubtedly speed the rate of progress in providing more adequate disease control recommendations to the turfgrass industry.

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