Fusarium Blight and Merion Bluegrass

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Diseases of turfgrass are caused by fungi, and controlling them is often perplexing and complex.

During the years of low rainfall, in 1960-1966 in Massachusetts, a species of Fusarium appeared to be causing a severe disease of Merion Kentucky bluegrass and, to a lesser degree, it also infected annual bluegrass and creeping bentgrass.

In 1959, Houston B. Couch, of Virginia Polytechnic Institute, first described the symptoms of this turf disease, known as Fusarium blight, incited by *Fusarium roseum*. In an overall view, infected turfgrass first shows scattered light green to grayish green patches 2-6 inches in diameter; the color of these patches changes in a 24-48 hour period to a dull reddish brown, then to tan, and finally to a light straw color.

The shapes of the infected areas, as described by Couch, are either elongated streaks, crescents, or circular patches. Leaf lesions which may occur at the cut ends, or randomly over the grass blade, are typically white-centered, surrounded by light to dark brown colored tissue.

Once the organism has successfully entered the plant, it moves inter- and intracellularly upward into the leaf tips and downward into the crown bud region. If environmental conditions necessary for the organism change, and the crown bud region has not been infected, the plant may live; if not, *Fusarium roseum* will actually cause death of the grass plants via crown rot and foliar cortical decay.

Fusarium species have been described by a number of investigators to be ubiquitous in nature, but the presence of these organisms does not always cause disease. Investigations by plant pathologists have shown that soil-inhabiting organisms have specific requirements for growth, such as, carbohydrate:nitrogen ratios, oxygen, micronutrients, etc. However, even when all nutritive demands have been met, a pathogen may still not pass over into a disease state, but instead remain saprophytic. *Fusarium roseum* is a fungus that changes from non-infectious to an infectious organism, but what causes it to change is not yet known.

A fungus growing and multiplying in a pure culture, i.e., on agar, is quite unlike a fungus co-existing in the soil with other organisms. Once within a living host, perhaps, an organism may not have competition other than outside environmental stresses. In the soil, the fungus may experience severe competition with its co-inhabitants, which it may or may not overcome in order to meet nutritive requirements. Whatever the condition is, it is most likely not an isolated one, but perhaps part of a chain in the ecological system.

It has been shown by investigators of certain other plants that it might be possible to control a pathogenic organism by influencing populations of microflora lytic or competitive to the pathogen responsible for a disease (1,4,5). Their investigations evolved from a hypothesis that if a certain material in a

relatively pure form was added to a soil, then organisms that possessed an enzyme capability of utilizing this material might build up their population either to the exclusion or the detriment of the hostile pathogenic organism.

For example, ground lobster shell was selected by these investigators as a soil amendment. The lobster shell is rich in chitin and Fusaria species contain chitin as a component of their cell wall. Therefore, with the addition of lobster shell it was postulated that chitinovorous organisms would be stimulated, and once large populations were built up, these organisms would utilize any chitinaceous material in the soil, and perhaps lyse those organisms that possessed it.

Based on the above, Joseph Keohane, as part of his master's degree program at the University of Massachusetts, investigated Fusarium blight. He studied the influence of lobster shells, sucrose, fungicides and an organophosphate insecticide on the soil microflora, which included *Fusarium roseum* infecting Merion Kentucky bluegrass. He also investigated the influence of soil moisture systems which appeared to be the most interesting phase of the study.

It was established that *Fusarium roseum* was causing Fusarium blight on Merion bluegrass in Massachusetts. This was proven by isolating the organism, growing it in pure culture, and comparing the cultures taxonomically and physiologically with known cultures. The pathogenicity of these isolated cultures was also determined by inoculating pots of Merion.

A higher percentage of inoculum take was found in pots of grass which were cut by a razor and then inoculated. Infection signs also appeared only 30 hours after inoculation, whereas it required 50 hours to appear in plants cut with shears, or uncut plants. Possibly the razor-cut grass blades exuded more sustenance for a longer time than the duller tearing action of shears.

The investigations of the influence of soil amendments on the soil microflora were carried out on newly established turf grown in soil contained in boxes and inoculated with *Fusarium roseum*. Lobster shell, sucrose, insecticide and fungicides were also applied to plots of Merion bluegrass growing on a football field and not infected with Fusarium, and to Merion in a home lawn that was infected.

The addition of even small amounts of some of these above-mentioned amendments did induce broad changes in the spectrum of the soil microflora. In general, the insecticide did not stimulate fungal response; it did depress actinomycetes and enhanced the appearance of a few dominant types of bacteria. The addition of lobster shell stimulated actinomycete growth, but none of the materials appeared to bring about a decrease in Fusarium infection.

The infection by *Fusarium roseum* of the Merion bluegrass lawn appeared to be most prevalent close to a tree trunk, along the edge of a driveway, and in other areas that were well drained. The lawn had received ample applications of lime and fertilizer. The lawn apparently was healthy, but it had a layer of thatch. The turf was growing in a sandy loam soil which was on the dry side but was irrigated periodically until a local water ordinance forbade its use.

Fungicides, as well as the afore-mentioned amendments, were applied to plots, but none stopped the spread of the disease. As the drought persisted, the soil became increasingly dry although not to the permanent wilting point. The incidence of disease increased during this period. However, after a thunderstorm producing 1.75 inches of rain it was observed that fungal activity slowed down, but increased again as the soil dried.

Investigations of the presence of other organisms in the plots showed that an organism known as *Trichoderma viride* was present wherever Fusarium was active. However, it was not found in any foliar samples. Tests in the laboratory showed that Fusarium and Trichoderma exercised little influence on each other.

Because of the effect soil moisture appeared to have on the incidence of Fusarium blight and the presence of *Trichoderma viride*, a greenhouse study was undertaken to determine their relationships. Composite samples of sand, silt and clay were mixed in varying amounts to obtain a relative degree of moisture equivalence. Soils having a moisture equivalence of 20, 40, 60 and 80 per cent were placed in pots and then seeded to Merion. Soil from diseased lawn turf area had a moisture equivalence (m.e.) of 34 per cent. Four pots within each moisture regime, were inoculated with Trichoderma alone, four with Fusarium alone, and four received a combination of both organisms.

Within 72 hours Trichoderma plus Fusarium pots at 20 and 40 per cent moisture showed

infection; lesions were observed on the leaves. Twenty-four hours later all plants were cut to 1 ½ inches; eight hours following this, some lesions were on the leaves at all m.e. level treatments where Fusarium was included. Lesions and signs of fungal infection or proliferation did not persist at the higher moisture levels of 60-80 percent. The disease did not increase in intensity in those pots in which only Fusarium had been added; and most of these plants so inoculated appeared to recover after clipping. The clipping evidently aided removal of active Fusarium stages.

In the combined treated pots, *Fusarium roseum* was identified as the pathogen, and apparently was the sole pathogen. Disease symptoms found in Trichoderma-inoculated plants appeared to be more the result of a distress condition engendered by chlorosis in the leaves. There was no general plant debilitation even in the 20 per cent m.w.-treated pots after 40 days.

The incidence of disease in pots inoculated with *Fusarium roseum* showed that the immediate effect was not as rapid as that which occurred when Trichoderma was present. There was no recovery where lesions, mycelia, and spores of Fusarium were observed throughout the plants. Once a plant had been killed, close-growing grass plants did not spread or grow into the void left by the dead plant.

The results showed a highly significant effect of both the soil moisture and the interaction of the dual inoculation. From their investigation of Fusarium blight, Couch and Ellis R. Bedford stated that under three soil moisture regimes, no significant difference in the incidence of the disease occurred. Their study might have been carried out with a single fungal parasite and without regard to the possible role of other organisms. It is quite possible, as shown by Keohane's work, that moisture is only one of a number of stress situations and that microbial interrelations may be the second group of determinants.

It should be added that in 1967 the drought in Massachusetts was broken, and the home lawn mentioned above did not show any symptoms of Fusarium blight. However, in 1968 the early spring was dry, and this was followed by a considerable amount of rain in the late spring, followed by a dry summer. During the dry period this same lawn again exhibited symptoms of the disease, but the incidence was less than in 1966. Did the lawn soil also contain a large population of *Trichoderma viride* in 1968? Diseases of turfgrass and their control certainly appear to involve the sum total of the ecological system.

References

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Correction and Additional Information

In the article "Reorganization Moves Green Section Ahead" (November, 1968, USGA Green Section Record), the annual fee for the USGA Green Section Visiting Service program was stated to be $225 for an 18-hole course. This is incorrect. The following rates apply to the various classes of courses:

Less than 18 holes	$175
18 to 27 holes	$200
36 holes	$225
Per regulation course in addition to 36 holes	$ 60

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