Drought Stress as a Factor Triggering Fungal Diseases of Turfgrass

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Fungal diseases of crop plants, such as potato and wheat, usually worsen with continued monoculture since pathogen populations tend to increase in the crop debris and in the soil. Although turfgrass diseases occur on golf courses each year, their amount and severity vary from year to year and from location to location, bearing little relationship to the age of the planting.

The erratic occurrence of turf diseases is also evidenced by the limited areas of grass that are diseased even under the most favorable conditions. The frequent failure of disease to develop is difficult to explain since even a small lawn consists of millions of ground-hugging plants of similar genetic make-up and disease susceptibility. The crowded plantings and grass debris (mat and thatch) at the soil surface favor the formation and retention of high humidity and even temperatures required for the growth and rapid plant to plant spread of the disease. Guttation and dew formation is almost a daily occurrence and the population of fungal pathogens apparently increase yearly in the soil, in the mat and thatch and on infected plants. Furthermore, turfgrass pathologists have had to rely on natural disease development for fungicide evaluations, because most attempts to create disease artificially in the field have failed. The factors responsible for this failure, and the erratic development of disease are probably biological in nature.

Facultative fungal parasites of turfgrass (e.g. *Rhizoctonia solani, Sclerotinia homeocarpa, Pythium aphanidermatum, Helminthosporium sativum,* etc.) are constantly being exposed in the following ways to antagonism and competition from the flora and fauna, and therefore their development is subject to biological influences throughout their lifetime:

1. The dense planting and the short, prostrate growth habit of the grass plant place it in contact, or in proximity to the microbiologically active surface litter and soil.
2. The plants are constantly being exposed to microorganisms by means of foot traffic, by maintenance practices such as mowing, fertilization, and irrigation, and by the varied activities of the macrofauna such as earthworms, nematodes, birds, and insects.
3. The grass clippings and the death of lower leaves, stolons, rhizomes, roots and tillers form the surface litter which is composed of fresh and decaying grass debris in various stages of decomposition. The constant addition of fresh clippings to the litter during the growing season is unique and

Helminthosporium Vagans *spores.*

constitutes an effective and continuing source of food for the litter-inhabiting microorganisms which actively compete with the fungal parasites for food.

4. Depending upon the depth of the litter, a variable amount of the stems and roots will be covered by the biologically-active litter.
5. Because of the extreme root density and their surface location, the nutrients which leak out from fresh grass clippings may influence the growth of microorganisms living on or near the root surfaces as well as the litter-inhabiting microorganisms.

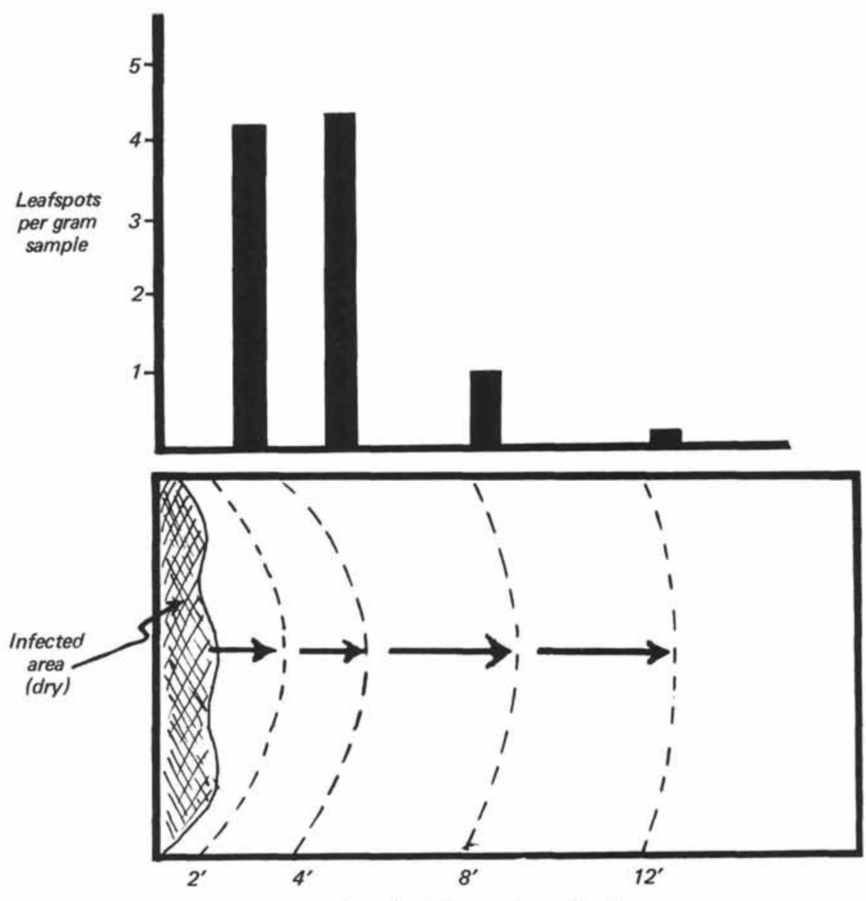
Thus, the total microbiological activity may, at times, be very high in the litter and in the soil, and undoubtedly influences the activity and survival of parasitic fungi.

We suspect that the erratic occurrence of turfgrass diseases caused by these parasites is due to the suppression of them by competitive and antagonistic activities of the flora and fauna. Disease usually occurs when resistance of turfgrass plants has been reduced, or when conditions favor the development of the pathogens more than they favor the development of the competing and antagonistic flora and fauna. Drought stress is an example of a commonly occurring condition or "trigger" which probably frees the fungal parasite from the restraining influence of the competing microorganisms, and allows the parasite to develop. The occurrence of localized dry spots in turf is a commonly occurring problem due to compacted soil, infrequent irrigation, uneven terrain, lack of rainfall, excess mat and thatch, wind disruption of sprinkler patterns and a high degree of water runoff.

The first experimental evidence that low soil moisture may increase certain turfgrass diseases was presented by Couch and associates. They demonstrated this relationship for dollar spot caused by *Sclerotinia homeocarpa* (3) and for greasy spot caused by the water-mold fungus, *Pythium ultimum* (4). Bean (1) has not only noted that the field occurrences of *Fusarium* blight of bluegrass caused by *Fusarium roseum* is correlated with the occurrence of dry spots but also that the disease can be greatly reduced by proper watering.

The mechanisms responsible for this increase in disease in dry soils have not been investigated in turfgrass. It may therefore be instructive to consider the research of Cook and Papendick (2) who found that foot rot of

Figure 1. Disease incidence of Helminthosporium *leaf spot with increased sampling distance from an infected dry area of bluegrass.*



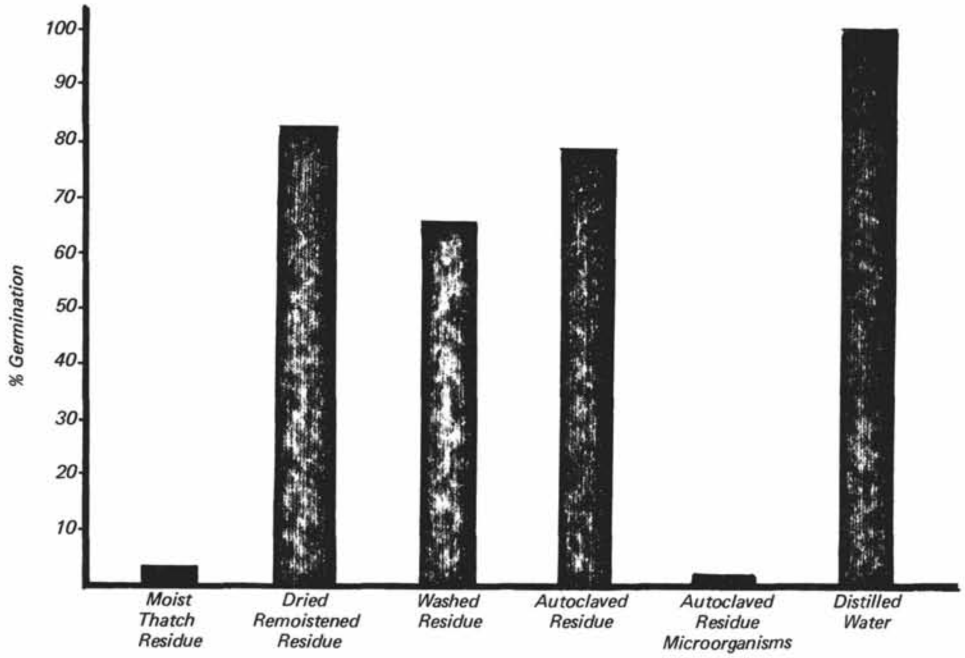


Figure 2. Germinability of Helminthosporium sativum *(T431) conidia on thatch residue of Kentucky bluegrass.*

wheat caused by ***F. roseum,*** the same fungus that causes ***Fusarium*** blight of turfgrass, is favored by dry soils. They found that the number and activities of soil bacteria were reduced greatly at soil moisture levels below -8 bars, that the resistant thick-walled spores of ***F. roseum*** germinated in soil well below the permanent wilting point of plants (-15 bars) and that after germination occurred, the threads of the fungus were able to germinate and infect plants. They also reported that soil bacteria were not only able to inhibit fungal germination but were also able to dissolve the walls of the fungal threads. Cook and Papendick therefore attributed the heightened parasitic activity of the fungus in dry soils to the reduction in populations and activities of soil bacteria.

Following the lead of Cook and Papendick, the effects of drought stress are presently being investigated in turfgrass by P. F. Colbaugh, graduate student at the University of California, Riverside. He has found that disease activity of ***Helminthosporium sativum,*** which causes leafspot and foot rot of Kentucky bluegrass, is increased under conditions of low soil moisture. Field observations on the incidence of the disease indicated that leafspot symptoms decreased with increasing distance of sampling from drought-stressed areas of bluegrass lawns (Figure 1). Severe foot rot and spore production by the fungus on thatch and on infected plants were observed in drought-stressed turf but not in areas receiving adequate water; only occasional leafspots were found in watered areas of the lawn.

The fungus has been recognized by previous workers to be a very weak competitor in the presence of other microorganisms. Evidence which strongly supports the involvement of microbial activity in suppressing the ability of the fungus to develop on the thatch debris is shown in Figure 2. Spores placed on moist thatch residue do not germinate, even though adequate moisture is present, but when washed from the surface of moist debris, they germinate readily. The inhibitory effects of moist thatch residue can be removed by thoroughly washing, sterilizing, or drying debris. The inhibitory property can be restored to the sterilized thatch debris if microorganisms are added to the residue. Immediately after rewetting the dried thatch, it greatly favors germination of ***Helminthosporium*** spores but the inhibitory property of moistened thatch debris returns after a few hours. At the time of rewetting dried thatch debris, large quantities of sugars and proteins are released. Carbohydrate release curves are shown in Figure 3 for both dry and moist thatch residue. Both the level of release and the rate of release were greater from dried debris which was remoistened than from moist debris. Since abundant nutrients are present when the dried debris is remoistened, there is sufficient food to nourish not only the ***Helminthosporium*** fungus, which is a poor com-

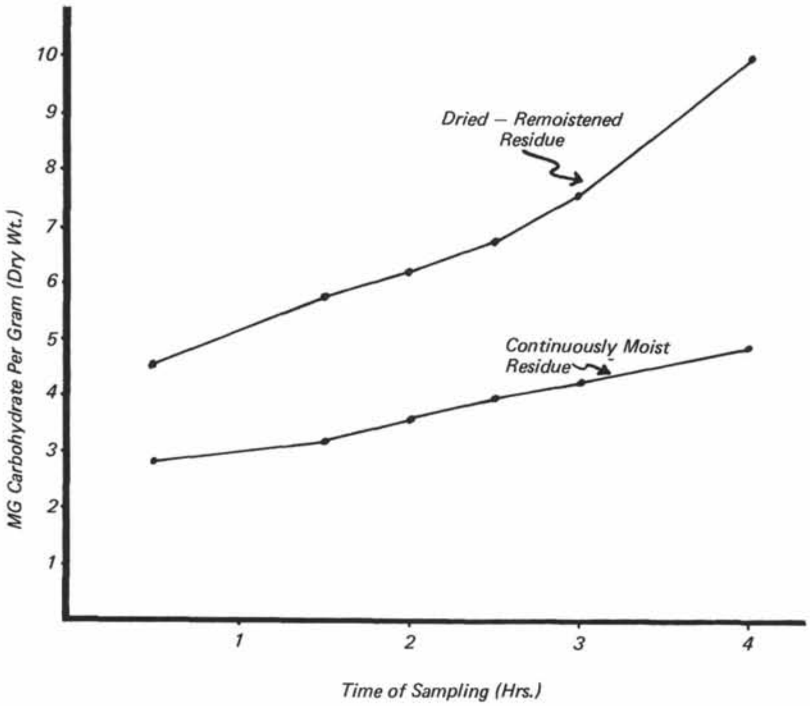


Figure 3. Release of soluble carbohydrates by bluegrass thatch residue.

petitor, but the numerous competing microorganisms as well.

It appears that the inhibitory property of thatch debris is active only when the debris is in a moist state and when microorganisms are present and active. This coincides with the period of greatest microbial activity on the decomposing residue. Drought lowers both microbial numbers and their activities. Upon rewetting, the dried thatch growth and microbial activities are resumed at high levels until equilibrium is once again established with the available food supply.

Another important aspect of drought stress is its effect on stopping plant growth. When growth stops, *Helminthosporium* infections on the lower part of the bluegrass plant tend to develop into the lethal foot rot stage. But when growth is continuous as in the presence of moisture, such infections tend to develop into harmless leaf blade infections.

Effects of drought on reducing microbial activities and increasing the competitive ability of *H. sativum* have been briefly described. Other influences of drought and its effect on turfgrass disease activity await further investigation. Our goal is an understanding of the nature of facultative fungal parasites. Our goal is an understanding of the factors responsible for "triggering" them into activity.

Literature Cited

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