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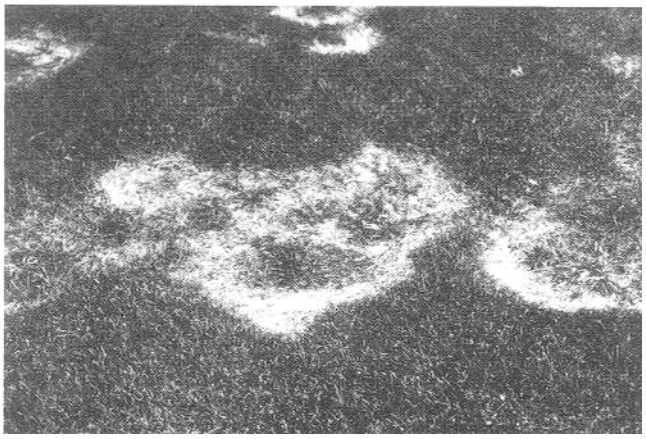
NECROTIC RINGSPOT ON BLUEGRASS TURF

For the past several years bluegrass lawns in both eastern and western Washington have been plagued by a disease which looks very similar to take-all patch, a disease of bentgrass. However, since bluegrass is generally considered to be resistant to the take-all patch pathogen, *Gaeumannomyces graminis* var. *avenae (Ophiobolus*), there was some doubt as to what actually was causing the problem on bluegrass.

Research at Washington State University's Western Washington Research and Extension Center has been directed toward identifying the cause of this problem and its control.

The fungus *Leptosphaeria korrae* has been shown to be the causal agent of this bluegrass disease. Recent studies in other states, including Wisconsin and New York, have implicated this same fungus as one of several pathogens involved with the disease complex historically known as Fusarium blight (not to be confused with Fusarium patch, common to northwest lawns). This fungus also has been described in Australia and California as the cause of spring dead spot on Bermuda grass. During the annual meeting of the American Phytopathological Society in August, 1983, a consensus of turfgrass pathologists present agreed on the name "necrotic ringspot" for this disease.

FIGURE 1: "Necrotic ringspot" on bluegrass is caused by the fungus *Leptosphaeria korrae*. Affected areas first appear as small spots or patches of dead turf during late spring-early summer or late summer-early fall. Patches can expand to form rings,arcs,and larger patches up to several feet in diameter.



The disease is most commonly seen in our area on two- to five-year-old turf established from sod, but it has also been observed on seeded turf. Highly maintained turf, especially turf being overfertilized and overwatered, seems to be more subject to attack. The disease has also been observed on bluegrass turf in Idaho and Oregon.

Affected areas first appear as small spots or patches of dead turf during late spring-early summer or late summer-early fall. These patches can expand to form rings, arcs, and larger patches up to several feet in diameter (Fig. 1). Weeds and sometimes unaffected grasses invade the center of the rings. During the spring and fall when the fungus is active, the margins of the necrotic areas can have a maroon-brown coloration. Diseased plants can be easily lifted from the soil. Microscopic examination of the surface of lower stems and roots reveals the presence of dark hyphae which are similar in appearance to runner hyphae of *Gaeumannomyces (Ophiobolus)* (Fig. 2). Short-necked, black pseudothecia are sometimes found on infected crowns and roots. The ascospores produced in these pseudothecia appear similar to those of *Gaeumannomyces*, and Washington isolates measure 135(80-188) x 4-5 u with 7(5-11) septa.

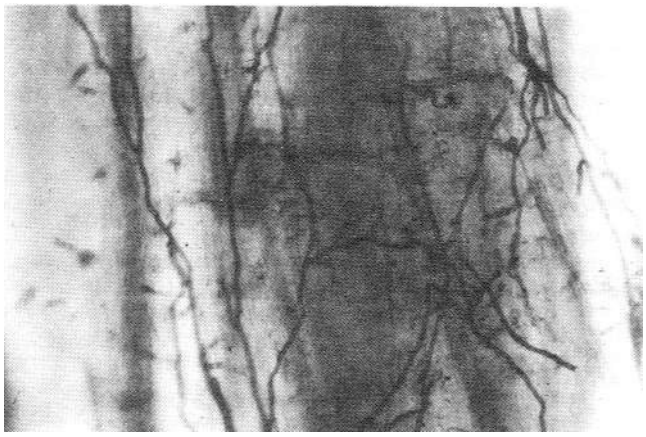


FIGURE 2: Microscopic examination of the surface of diseased lower stems and roots reveals the presence of dark hyphae which are similar in appearance to runner hypae *of Gaeumannomyces (Ophiobolus)* in this photograph.

Little is known at this time concerning the life cycle of this organism. Pseudothecia of the pathogen are seen infrequently but have been observed with mature ascospores in Washington during the fall. Their importance in disease development is unknown. In both eastern and western Washington, the disease is most active in the spring and early fall. Frequently, the disease is inactive during mid-summer and the turf appears to recover, but the same ringspots usually reappear in the fall.

Although applications of sulfur are helpful in controlling take-all patch on bentgrass, **applications of sulfur have been ineffective in controlling necrotic ringspot on bluegrass.** However, a balanced fertilizer program including sulfur should not be ignored, since it is beneficial in helping the turf to "recover" when conditions for the disease become unfavorable during the summer. In addition to maintaining good nutrition to help in "summer recovery," it is important not to overfertilize or overirrigate affected turf. Infrequent deep watering is suggested as opposed to frequent light irrigations.

Initial laboratory screening of fungicides indicated that Rubigan, Banner and Tersan 1991 had better activity against this fungus than Chipco 26019 and Bayleton. In 1983, Banner, Bayleton, and Rubigan were field tested by making monthly applications during May, June, and July. These spring and early summer applications of Rubigan and Banner provided good disease control during the late summer and early fall.

In 1984 trials, one application of Rubigan 50W applied at 2 ounces per 1000 square feet on May 24, gave excellent control of the disease during the late summer and early fall. Three monthly applications of Banner also gave similar control, but it is not presently registered for use. When these spring and early summer applications have been made to obviously diseased turf, there are no immediate observable benefits. However, the benefits are noticed in the fall following the "summer recovery" period. In the untreated turf the rings reappeared, whereas little or no disease was evident in the Rubigan and Banner treated plots. Bayleton, Chipco 26019, and Tersan 1991 failed to give adequate protection even with three monthly applications.

Warning: Rubigan can cause damage to bluegrass turf. Damage was observed when 4-6 ounces of Rubigan 50W was applied to 1000 square feet of turf. Rubigan is also detrimental to *Poa annua*. Be sure to carefully calibrate your sprayer and read and follow all label directions and precautions.

Research to determine the timing of fungicide applications for best disease control are continuing. In addition, a graduate student is studying some of the basic aspects of the biology of this disease.
