Leptosphaeria korrae and Phialophora graminicola Associated with Fusarium Blight Syndrome of Poa pratensis in New York

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ARSTRACT

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Two fungi with growth habits similar to that of Gaeumannomyces graminis were found to be associated with a patch disease of Poa pratensis in New York. Both pathogens have been shown capable of causing the disease, which is indistinguishable from the poorly understood Fusarium blight syndrome. The process by which Leptosphaeria korrae and Phialophora graminicola caused the ringlike patterns characterizing Fusarium blight syndrome is being investigated further. This is the first report of L. korrae and P. graminicola in North America and of their occurrence on Poa spp.

Poa pratensis L. (Kentucky bluegrass) is the most important and widely used turfgrass species in temperate regions of North America. Numerous cultivars of P. pratensis have resulted from intensive selection and breeding programs. The diversity in aesthetic and horticultural characteristics provided by these cultivars has enabled this grass to be adapted to many uses and environmental sites. Research on diseases of P. pratensis has grown in proportion to its culture. Our emphasis has been placed on the Fusarium blight syndrome (9), and we report the occurrence of Leptosphaeria korrae Walker & Smith and Phialophora graminicola (Deacon) Walker on P. pratensis in New York.

L. korrae is a graminicolous pathogen reported previously in only Australia (14). L. korrae and L. narmari (Walker & Smith) cause spring dead spot of Cynodon spp. turfgrasses in Australia (11). The infection habit and life cycle of

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these Leptosphaeria spp. closely resemble those of Gaeumannomyces graminis var. tritici (Ggt) and G. graminis var. avenae (Gga), which cause take-all of cereals and take-all patch of Agrostis spp. turfgrasses (4,6,13). Spring dead spot, Fusarium blight syndrome, and take-all patch are each characterized by circular patches of dead grass that tend to increase in size for 3-4 yr (10). Recovery of affected grass, or recolonization of the dead patch, tends to occur first in the center of the patch, often causing the patch to appear as a ring of dead grass. Whereas G. graminis varieties are quite host-specific, existing information indicates that L. korrae is not. Walker and Smith (14) reported L. korrae isolates from grasses in the genera Axonopus, Cynodon, and Eremochloa and showed that isolates from Cynodon are also pathogenic on the cereals Avena sativa L., Oryza sativa L., and Triticum aestivum L. Nilsson and Smith (6) and Scott (8) reviewed reports indicating that Poa spp. are typically quite resistant to attack by Ggt and Gga, although some isolates of Ggt caused moderate levels of disease on some cultivars of *P. pratensis*. Agrostis spp. are more susceptible to infections by Gga than by Ggt.

P. graminicola is a common inhabitant of grasslands in Europe and Australia but has not been formally reported in North America (3). The fungus is thought to be the anamorph of G. cylindrosporus

Hornby, Slope, Gutteridge, & Sivanesan (12). Because previously studied isolates of *P. graminicola* were avirulent (or only weakly so) to cereals and grew sparsely on rhizomes of several grass species (*Poa* was not studied), the fungus has been investigated as a possible biocontrol agent against the take-all disease of wheat (3). Some isolates in Britain have also been shown to enhance shoot growth of *T. aestivum* and *Festuca + Agrostis* turf in nutrient-poor soils in much the same manner as mycorrhizal fungi. The fungus is generally considered a weak parasite (3).

In October 1980, plants affected by Fusarium blight syndrome were collected from the margins of ring-shaped diseased patches (Fig. 1A) in five P. pratensis turfs near Syracuse, Skaneatales, Glen Cove, and Oyster Bay, NY. The affected plants were chopped and used to fill 2-cm lengths of Tygon tubing, which were then set in pots of autoclaved coarse sand. Wheat (T. aestivum) and oat (A. sativa) plants were grown in the debris and became severely affected with a disease visually (Fig. 1B) and histologically identical to take-all, including invasion of stelar tissues by pathogenic fungi. Isolations from 6-wk-old wheat roots with dark ectotrophic hyphae (Fig. 1C) vielded two different fungal morphologies on potato-dextrose agar (PDA) medium. One fungus had a "felted," dark gray, aerial mycelium; the other fungus was characterized by appressed olive brown mycelium with distinctive mycelial strands like that of Gaeumannomyces spp. Numerous attempts to induce fruiting on agar cultures and rotted roots (1) failed from 1981 to 1983.

Direct observation of *P. pratensis* rhizomes and tiller bases in the outer margins of Fusarium blight syndrome patches before symptom development was initiated in 1981. Existing patches in Farmingdale, Oyster Bay, and Syracuse, NY, were marked permanently in 1980

and mapped so they could be readily relocated after regrowth of grass occurred in the patches. Observations of rotting rhizomes and tiller bases of plants before visual symptom expression (Fig. 1F) in 1982 and 1983 showed repeatedly that dark, ectotrophic mycelia were present and active at the margins of the patches, which would develop as the stresses of summer became imposed on the infected grass. Several hundred isolations demonstrated the consistent presence of the same fungi (visual characteristics in culture) isolated from the 1980 wheat and oat traps. Isolates varied in their ability to infect wheat and oats.

Six appressed, stranded isolates and four felted isolates pathogenic on wheat and oats were grown on 0.5× PDA medium and used as inoculum in turfgrass plots in Farmingdale, NY, in April 1982. Additionally, isolates were grown on sterilized, perennial ryegrass (Lolium perenne L.) seed and used as inoculum on adjacent turfs in April 1983. Plugs of turf (2.5 cm diameter × 3 cm deep) were taken at 30-cm intervals in a straight line, and inocula of the fungi (or sterile controls) were placed at the thatchsoil interface (1-2.5 cm deep). In August 1983, patches of dead turf (3-20 cm diameter) appeared in the precise locations (Fig. 1E) where the inocula of five appressed and four felted isolates had been placed in April 1982 and 1983. Diseased roots and rhizomes yielded isolates of the two fungi.

Attempts to produce the teleomorph of the appressed, stranded isolates have been unsuccessful. Many isolates are very pathogenic to *P. pratensis* roots and mildly pathogenic to roots and crowns of *T. aestivum* and *A. sativa*. The fungi rarely colonize green tillers or stems of *P. pratensis* and *A. sativa*, and the mycelial sheath development on lower culms of *T. aestivum* is sparse. With the exception of pathogenic habits, all characteristics of these fungi conform with the descriptions of *P. graminicola* (2,13) and anamorphic

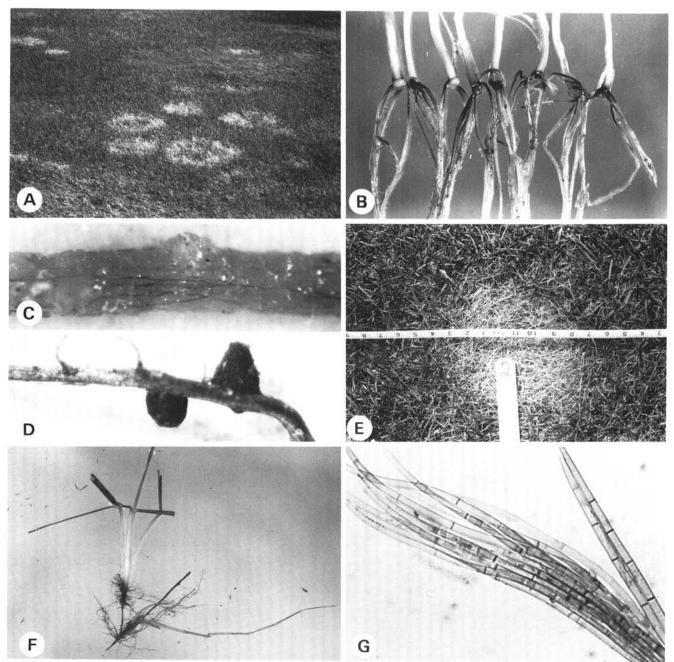


Fig. 1. Fusarium blight syndrome of *Poa pratensis*. (A) Typical ring patterns and distribution of patches. (B) Take-all symptoms caused by *P. pratensis* isolates of *Leptosphaeria korrae* and *Phialophora graminicola* on *Triticum aestivum* and *Avena sativa*. (C) Darkly pigmented ectotrophic mycelia on *T. aestivum* root. (D) Erumpent pseudothecia of *L. korrae* on rotted *P. pratensis* root. (E) First-season patch induced by inoculation of *P. pratensis* with *L. korrae* (identical patches resulted from inoculations of *P. graminicola*). (F) Necrosis of *P. pratensis* rhizome, tiller base, and crown before disease symptom expression in foliage. (G) Ascospores of *L. korrae*.

structures of Ggt "Group VI" (5). In culture, these fungi have very sparse, aerial mycelium and only slight curling back of hyphae at the periphery of colonies. Growth rates at the optimal temperatures (20-25 C) are slow (4.7 mm/day). Colonies are initially colorless to light gray and darken from the colony origin to an olive brown. Distinctive stranding is present on 0.5× PDA medium and on roots and crowns of graminicolous hosts. Germinating phialospores measure 5-13 \times 2-4 μ m, and microspores apparently are not produced. Hyphopodia are subglobose and often occur in groups. Thick black sclerotial rinds are produced on sterile oat grains, but sclerotia have not been observed on agar cultures.

We have succeeded in producing pseudothecia of L. korrae in cultures of the felted isolates. In 1983, diseased roots, crowns, and rhizomes of P. pratensis from naturally occurring Fusarium blight syndrome patches were collected. Affected tissues were placed on continuously wetted cotton mats in trays and rotted for several months in diurnal light in the laboratory or greenhouse. Pseudothecia (Fig. 1D) containing bitunicate asci (155 \times 12 μ m) were formed after 2-3 mo of incubation. Ascospores (Fig. 1G) measured (63) 113-138 (158) \times (3.3) 4–5 (5.8) μ m. The length (but not the width) of these ascospores is comparable to those of Gga [(85) 100-130 $(140) \times 2-3.5$ (4) μ m] and Ggt [(60) $70-105 (110) \times 2-3 (4) \mu m$]. However, Gaeumannomyces spp. have perithecia and unitunicate asci. Pathogenic, cultural, and morphological features of the felted fungus from P. pratensis are nearly identical to those of L. korrae (14); however, the ascospores produced in our laboratory are shorter than those described for L. korrae [(120) 140-170 $(180) \times 4-5 (5.5) \mu m$]. This difference is within the natural range of variation accepted for intensively studied varieties and species of related ascomycetes (14).

More recently, single-ascospore cultures of *L. korrae* have been used to infect graminicolous hosts. The fungus was reisolated and induced to produce pseudothecia either by rotting infected tissue or by incubating it in tubes containing wheat leaf agar medium (irradiated wheat leaf sections in slants of water agar) (R. T. Kane, *unpublished*). Proof-of-pathogenicity tests have there-

fore been conducted in the greenhouse and in the field, with typical field symptoms being produced in the latter. Another pathogen associated with the Fusarium blight syndrome of P. pratensis in New York is therefore L. korrae. This fungus is considerably more virulent than P. graminicola. Specimens of L. korrae have been submitted to the following culture collections: Cornell University Mycological Herbarium (Ithaca, NY), Commonwealth Mycological Institute (Kew, Surrey, England), American Type Culture Collection (Rockville, MD), Centraalbureau voor Schimmelcultures (Baarn, Netherlands), and John Walker (Australia).

The patch diseases with which L. korrae and P. graminicola are associated occur under the same cultural and environmental conditions as the Fusarium blight syndrome (10) of P. pratensis, and the diseases are indistinguishable. The etiology of Fusarium blight syndrome has been questioned repeatedly since it was first attributed to Fusarium species in 1966 (7,9). Relationships between L. korrae, P. graminicola, and Fusarium blight syndrome are being investigated further. At present, we believe these fungi infect circular to irregularly shaped patches of P. pratensis without killing the grass directly. When the infected grass plants are stressed additionally during summer, the predisposed grasses become further weakened and either succumb directly or are invaded by the dominant facultatively parasitic fungi in the turfgrass canopy at that time. Fusarium spp. are generally the dominant population of sporing fungi present in turfs during the summer. This succession describes a pathogenic sequence that is harmonious with nearly all known characteristics of the Fusarium blight syndrome and its control, whereas the ecology of Fusarium spp. indicates that they are incapable of serving as the primary causal agents for this patch disease (9,10). Variations in sensitivities of L. korrae and P. graminicola to fungicides, temperatures, acidity, and osmotic potentials and variations in virulence to turfgrass species and cultivars will be reported later to support further the successions proposed for this disease.

This is the first report of *L. korrae* and *P. graminicola* in North America, and their distribution is therefore not known.

This is also the first report of these fungi on *Poa* spp.

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