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The Nature of Gray Leaf Spot and Its Management

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Gray leaf spot is caused by the fungus *Pyricularia grisea* and is a common disease of St. Augustinegrass (*Stenotaphrum secundatum*) lawns in the southeastern United States. It can be particularly damaging to newly sprigged lawns, but may be a chronic problem in mature stands of St. Augustinegrass grown in subtropical climates. Bermudagrasses (*Cynodon* spp.), centipedegrass (*Eremochloa ophiuroides*), fescues (*Festuca* spp.), bentgrasses (*Agrostis* spp.), Kentucky bluegrass (*Poa pratensis*), and ryegrasses (*Lolium* spp.) are listed as species susceptible to gray leaf spot. Some of the aforementioned turfgrass species only were shown to be blighted by *P. grisea* in growth chamber studies. In the field, however, there is only good documentation that the pathogen inflicts significant damage to St. Augustinegrass, annual (*L. multiflorum*)

and perennial ryegrasses (*L. perenne*), and tall fescue (*F. arundinacea*). The pathogen also causes blast, the most important disease of rice (*Oryza sativa*) worldwide.

History of gray leaf spot on turfgrasses

Gray leaf spot was formally reported causing serious blighting of perennial ryegrass fairways on two golf courses in southeastern Pennsylvania in 1991 (Landschoot and Hoyland, 1992). It appeared in late August/early September, coinciding with unseasonably warm temperatures and high relative humidity. In 1995, gray leaf spot reached epidemic proportions in perennial ryegrass grown on golf courses in the Mid-Atlantic regions. The disease was most severe in southeastern Pennsylvania, Delaware, and non-mountainous areas of Maryland, Virginia, and Kentucky. In Maryland, the disease was first observed in 1986 on a golf course near Annapolis. Between 1986 and 1994, the Maryland disease diagnostic lab received only a few samples of perennial ryegrass affected with the disease. The disease, however, was probably causing low levels of injury in roughs on numerous golf courses for years, resulting in a gradual buildup of inoculum. Evidently, environmental conditions in 1995 were ideal for disease development and the inoculum had by this time reached sufficient levels on many golf courses to initiate the epidemic. The 1995 summer was among the warmest and driest of the century in the Mid-Atlantic region, suggesting that heat and low soil moisture levels were important predisposing conditions. In 1996, the disease recurred, but was not as widespread as in 1995, and most injury occurred in roughs and green surrounds. The 1996 summer, however, was among the coolest and wettest in the prior 50 years. The ability of the pathogen to cause significant levels of injury in two very different summer environments suggested that inoculum levels were sufficient to ensure that the disease would be a recurring problem in the region. By 1998,

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