Fungus Family Provides Fodder For Several Diseases

By Clint Waltz, Alfredo Martinez, Mila Pearce and Bruce Martin

Gaeumannomyces is a genus of fungus with several species and subspecies associated with plants in the *Poaceae* (formerly known as *Gramineae*) family. The genus *Gaeumannomyces* includes several bona fide plant pathogens, as well as weakly virulent forms that contribute to disease when the host plants are stressed in some manner. Turfgrass diseases with *Gaeumannomyces* causal agents include take-all patch and bermudagrass decline.

Typically, take-all patch (TAP) is considered a disease of bentgrass restricted to cool temperate regions and thought to be relatively uncommon in the Southeastern United States. However, *Gaeumannomyces* species have been associated with bermudagrass decline and have been isolated from other warm-season turfgrasses.

While relatively little research focuses on the relationship between *Gaeumannomyces* and turfgrass, a plethora of information exists on the influence of the pathogen and cereal crops, especially wheat. Some of this information and related control strategies may apply to turfgrass.

Life cycle and epidemiology

Gaeumannomyces graminis var. avenae (Gga) is a soil-borne ascomycete fungus and is the causal agent of TAP on bentgrass. Infection typically begins with the formation of "ectotrophic" (root-surface) hyphae on roots, crowns, stolons and rhizomes of susceptible grasses. Hyphae are dark brown to black and run along the surface of the root with infection hyphae originating from swellings on hyphae (called "hyphopodia") or mycelial mats that have survived in grass tissue or thatch.

At certain times of the year, perithecia may form under and protrude from the lower leaf sheath of infected plants. Perithecia are about the size of a pin head, are black in color and are flask-shaped fruiting bodies that are the sexual stage of the fungus. The presence of perithecia with mature ascospores is diagnostic for this disease without further tests. Within the perithecia, asci and ascospores are produced. Asci are unitunicate containing eight hyaline, multiseptate ascospores. Ascospores can be wind-disseminated and may serve to reinitiate the infection process, but this is uncommon in turfgrass. Instead, the pathogen generally spreads in the soil through mycelial growth and can be transferred on equipment, sod and seed (Smiley et al., 1992).

Conditions favorable for growth and spread of TAP generally include poorly drained alkaline soil and temperatures lower than 86 degrees F (Wetzel et al., 1996). High rhizosphere pH is more commonly associated with root infection than the pH of the bulk soil (Smiley et al., 1992). Also, light textured soils with low organic matter content and unbalanced fertility favor disease.

Thus, the disease is more common on sites recently established to bentgrass than sites with more mature stands. Also, sites from recently cleared forests are more prone to outbreaks of TAP initially. However, these are the conditions associated with epidemics in cool temperate regions.

The epidemiology of TAP in temperate regions is poorly understood, as is the incidence of disease on well-drained soils associated with high-sand content greens.

Factors influencing G. graminis

Soil management practices that reduce soil pH and improve micronutrient availability may suppress TAP. The organism, *G. graminis var. tritici* (*Ggt*), responsible for take-all in wheat is virulent when soil pH exceeds 6.5 (Reis et al., 1983). As soil pH increases, availability of many essential trace elements (e.g. manganese (Mn), iron (Fe), copper (Cu), zinc (Zn) and others) decreases. Therefore, nutrient deficiencies may occur in crop species.

In combination with disease pressure, a conducive environment and imposed nutrient stress, it's possible that a susceptible host may become subject to take-all in wheat.

In studies on the affect of soil nutrients on *Gga* or *Ggt*, it has been difficult to separate the influence of the mineral element on the pathogen and reduced stress resulting from improved nutrition. In a review of soil nutrients on take-all, Hornby (1985) cited studies where the addition of macronutrients (nitrogen (N), phosphorous (P), potassium (K), magnesium (Mg) and sulfur (S) decreased the severity of take-all. Likewise, Reis et al. (1982) reported fewer infected roots of wheat and decreased disease severity with additions of P, K and Mg, while no benefit was realized from the addition of N, calcium (Ca) or S. Improved resistance was attributed to the effect of the applied nutrient on the host with no direct effect on the pathogen. Glenn and Sivasithamparam (1991) reported no effect of Ca on *Ggt*.

While some reports do not show reduced severity of take-all when nitrogen is supplied, other reports demonstrate improved host resistance to take-all when acidifying forms of nitrogen were applied (e.g. ammonium sulfate and ammonium nitrate) (Hornby, 1985; Huber and McCay-Buis, 1993).

It's well-documented that *G. graminis* spp. do not prefer acidic soil conditions (pH less than or equal to 6.5) (Reis et al., 1983; Glenn and Sivasithamparam, 1991; Ownley et al., 1992). Since acidifying fertilizers reduce soil pH, it could be reasoned that by lowering the pH the fertilizers contribute to an unsuitable environment for the pathogen.

Turfgrass diseases with Gaeumannomyces causal agents include take-all patch and bermudagrass decline.

The role of micronutrients on the reduction of disease severity has also been investigated. Reis et al. (1982 and 1983) concluded the deficiency of micronutrients (Cu, Zn, Mn, and Fe) in soil with pH measurements exceeding 6.5 increased take-all in wheat. Proper nutrition can reduce the severity of take-all during favorable environmental conditions. In a review by Huber and McCay-Buis (1993), the importance of plant available manganese (Mn^{+2}) in suppression of take-all in wheat was suggested. Several studies were cited where either Mn^{+2} was added or soil pH was reduced such that Mn^{+2} was made plant available and the effects of take-all were reduced.

Similar results were reported by Hill et al. (1999) when Mn^{+2} was applied to a *Gga*-infested bentgrass turf that was deficient in Mn. An 81 percent and 68 percent reduction, first- and second-year respectively, in disease incidence was reported when 1.82 pounds per acre of Mn^{+2} was applied.

Copper was not observed to have an affect on disease development. These studies indicate maintaining soil pH within acceptable ranges (5.5 to 6.5) may be the best method of reducing host susceptibility to take-all diseases while improving the availability of essential nutrients.

TABLE 1

Pathogen	Hosts	Infection conditions	Chemical control
Gaeumannomyces graminis var. *graminis*	Bermudagrass St. Augustinegrass Zoysiagrass Centipedegrass	Hot, Humid, Soil pH > 6.5	Triadimefon Thiophanate methyl
Gaeumannomyces graminis var. *avenae*	Bentgrass Tall fescue Bluegrass	Cool, Moist, Soil pH > 6.5	Propiconazole Fenarimol Triadimefon Thiophanate methyl Azoxystrobin

Life cycle on warm-season turfgrasses

Gaeumannomyces graminis (Sacc.) Arx & D. Oliver var. graminis (*Ggg*) is considered the most important causal agent of bermudagrass decline, a significant root-rot disease of *Cynodon* species.

The disease is especially damaging during hot, humid and cloudy periods of summer and autumn in the Southeastern United States. This disease is associated closely with turf that is consistently mowed at low cutting heights. Thus, it's much more common on greens than tees or fairways.

The etiology and epidemiology of the disease is not yet completely known, although progress has been made on identification of the causal agents (Elliott, 1991). The disease first appears as irregularly shaped, yellow, chlorotic areas measuring up to 19.7 inches in diameter. Lower leaves are the first to become chlorotic, but the lower leaves die and upper leaves appear yellow and chlorotic as the disease progresses.

By the time symptoms are evident on the foliage, the roots, stolons and rhizomes will be discolored, dark brown and rotted. If severe, the disease appears as large dead patches which

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soon coalesce to form larger affected areas. These symptoms are nonspecific, and other pathogens and environmental stresses (compaction, shade, etc.) may contribute to the development of decline symptoms.

Causal agents

Ggg has been demonstrated to be the most important fungus of this disease complex. But other fungi including *Gaeumannomyces incrustans Landschoot & Jackson*, and several *Phialophora* species (Elliott, 1991) may also be contributing to accentuate the symptoms of the disease. *Curvularia* species and species of *Drechlera* and *Bipolaris* also are commonly associated with declining bermudagrass.

Take-all patch is more common on sites recently established to bentgrass than sites with more mature stands.

G. graminis var. *graminis* forms dark-brown to black runner hyphae on roots, stolons and rhizomes of hosts. Infection hyphae penetrate the host tissue and initiate from lobed hyphopodia that are distinct from those formed by *Gga*. Perithecia are rarely present, but if formed are dark brown and flask-shaped and project from under the lower leaf sheaths of infected plants. The asci formed inside the perithecia are unitunicate and contain eight hyaline, multiseptate ascospores. *G. incrustans Landschoot & Jackson* and *Phialophora spp*. also produce dark ectotrophic runner hyphae and appears to be nonpathogenic or secondary pathogens when invading by themselves but may act synergistically when combined with Ggg (Elliott, 1991; Elliott and Landschoot, 1991; Smiley *et al*, 1992).

G. graminis var. graminis

Gaeumannomyces graminis var. graminis is found on most grasslands and except for temperature relations, pathogenicity, epidemiology and survival are similar to that of *G. graminis var. avenae*. Briefly, conditions that favor growth and spread of *Ggg* include warm to hot temperatures (77 degrees F to 90 degrees F) and poorly drained alkaline soils with unbalanced fertility.

Additionally, soils with light texture and low organic matter content, which are typical conditions in sand-based greens, also favor *Ggg* spread and infection. The fungus survives through the colonization of roots, rhizomes and stolons in the form of mycelium. It spreads by growing along roots or stolons, making plant-to-plant contact. Perithecia are produced during the autumn, winter and spring. The pathogen can also be disseminated by movement of infected roots, rhizomes and stolons on coring and vertical mowers and the transport of turfgrass sod (Elliott and Landschoot, 1991; Smiley et al, 1992).

Control of *Ggg* is achieved through cultural practices that enhance the growth of roots and/or improves the survival and function of healthy foliage. Of course, root and foliage health are interrelated and inseparable. Additionally, mowing height should be raised before severe decline occurs, which relieves stress and aids in the survival of affected plants. Cultural management of this disease requires an acute awareness of environmental factors favoring the pathogen.

Core cultivation should be performed frequently, and cores should be removed rather than spreading them to incorporate the soil as a topdressing. Enusring the presence of adequate soil fertility, with particular attention to nitrogen and potassium and the use acidifying fertilizers such as ammonium sulfate, contributes to plant recovery (Smiley et al, 1992).

Isolated from St. Augustinegrass

Gaeumannomyces graminis var. graminis has been implicated in a variety of root rot disease complexes of zoysiagrass and centipedegrass. The fungus also causes St. Augustinegrass take-all root rot disease, which was first found in Australia on St. Augustinegrass in 1972. Isolation and identification of the fungus was next reported in Florida in 1988. Symptoms of take-all are much like other warm-season turf diseases because the symptoms of take-all root rot of St. Augustinegrass appear in the summer and fall during periods of abundant rainfall.

Like the pathogen in bermudagrass decline, take-all root rot of St. Augustinegrass is an ectotrophic, root-colonizing fungus, and the symptoms appear in the same areas from year to year. Above ground symptoms consist of chlorotic, thinning turf in irregular patches, but the leaves do not separate easily from the plant. Below ground, the roots are short and rotted, making it easy to lift from the ground.

Take-all root rot of St. Augustinegrass has been diagnosed from at least 40 counties within Florida as well as Texas, Alabama, Georgia and California. Studies in Florida and Texas found that seven isolates of *Ggg* were cross pathogenic on St. Augustinegrass, bermudagrass and rice (Elliott et al, 1993; Datnoff et al, 1997). There appears to be no relationship between take-all root rot of St. Augustinegrass and the cultivar, soil type or age of the grass.

Additionally, there are no resistant varieties of St. Augustinegrass as symptomatic selections from the cultivars Delmar, Jade, Mercedes, Bitterblue, Scott's, Raleigh, Sunclipse, Seville, FX33, Floratam and others were isolated in California. The disease is as versatile on soil as it is on cultivars. The disease has been evaluated on soils ranging from fine sandy loams to organic black soils to muck soils. Newly planted lawns are susceptible as well as established lawns, since St. Augustinegrass is vegetatively propagated and the disease is spread in new shoots and roots.

In Georgia, plant disease diagnosis clinic specialists have identified *Ggg* from St. Augustinegrass turf samples from central and south Georgia during the summer and fall 2002 (Martinez, 2002; Pearce, 2002). Since *Ggg* attacks a variety of grasses, management and treatment are similar. Likewise, the concerns of monitoring and containment continue to be a problem (Elliott et al., 1993; Wilkinson and Pederson, 1993).

Chemical controls

Several fungicides have activity and are labeled for control of take-all patch, including the DMI fungicides propiconazole, fenarimol and triadimefon. Also, the benzimidazole fungicide thiophanate methyl has efficacy for TAP control.

More recently, the strobilurin fungicide azoxystrobin has shown excellent control for this disease. Interestingly, all of the fungicides listed above are upwardly mobile penetrant fungicides. They are applied preventively for best control, and thus are applied in the fall and spring prior to or during root infection periods. They should be applied in enough water to move the fungicides into the root zone to protect existing and newly formed roots effectively.

An alternative to a high spray volume would be to use irrigation to move applied fungicide into the root zone. Generally, an irrigation of

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one-half of an inch to 1 inch would be sufficient.

There are few fungicides labeled for control of bermudagrass decline. Labeled fungicides include only triadimefon and thiophanate methyl. However, it has been shown that labeled DMI fungicides (triadimefon) may be phytotoxic to bermudagrass when applied in summer (Elliott, 1995). In addition to thiophanate methyl, and perhaps triadimefon, azoxystrobin has shown effectiveness for alleviation of symptoms of bermudagrass decline.

Mancozeb and fosetyl Al have also shown effectiveness in alleviation of bermudagrass decline symptoms in trials conducted in South Carolina (Camberato and Martin, 2002). The active ingredients of Fore and Chipco Signature will not have efficacy against *Gaeumannomyces* species, but they may improve the health of bermudagrass and alleviate symptoms nevertheless. In the South Carolina trials, these fungicides were applied in 2 gallons of water per 1,000 square feet and not watered in.

It should be noted that azoxystrobin, mancozeb, and fosetyl Al are not labeled for control of Bermudagrass decline at this time.

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