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Rutgers University scientists provide an overview of the biology, host range, symptomology, and epidemiological factors influencing the pathology of anthracnose basal rot.

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PURPOSE

The purpose of *USGA Turfgrass and Environmental Research Online* is to effectively communicate the results of research projects funded under USGA's Turfgrass and Environmental Research Program to all who can benefit from such knowledge. Since 1921, the USGA has funded more than $40 million for research at universities. The private, non-profit research program provides funding opportunities to university faculty interested in working on environmental and turf management problems affecting golf courses. The outstanding playing conditions of today's golf courses are a direct result of ***using science to benefit golf***.

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Biology and Pathology of Turfgrass Anthracnose

Jo Anne Crouch and Bruce B. Clarke

SUMMARY

Rutgers University scientist provide an overview of the biology, host range, symptomology and epidemiological factors influencing the pathology of anthracnose basal rot. The paper's main points include:

- Anthracnose is a serious disease of annual bluegrass and creeping bentgrass, but knowledge of anthracnose biology in turf is still incomplete.
- Anthracnose is most severe during warm, humid weather, but outbreaks may occur throughout the year.
- Anthracnose is most destructive on weakened or senescent turf; low fertility, low mowing heights, drought and excessive heat enhance the disease.
- Activities causing wounded tissue do not appear to enhance the severity of anthracnose on annual bluegrass putting greens.
- Cultivar susceptibility, environment and variation in pathogen populations may play a role in disease development.

In the mid-1990s, anthracnose basal rot disease emerged as one of the key management issues facing superintendents who maintain annual bluegrass (*Poa annua*) putting greens. In this article, we provide an updated overview of the biology, host range, symptomology and epidemiological factors influencing the pathology of anthracnose basal rot.

Host Susceptibility

Anthracnose is a common disease on grasses and cereal grain crops worldwide (3). Although numerous cool- and warm-season turfgrass hosts are susceptible to anthracnose, the disease is most damaging on annual bluegrass (*Poa annua*) and creeping bentgrass (*Agrostis stolonifera*) maintained at putting green height. For reasons not fully understood, annual bluegrass is particularly susceptible to anthracnose, with stands of the grass severely damaged when conditions are optimal for disease development. Because anthracnose attacks weakened or senescent turf, annual bluegrass is especially vulnerable when maintained under conditions of low mowing, low fertility, or drought stress. The risk of anthracnose infection to annual bluegrass turf is further intensified during hot, humid weather.

Anthracnose disease may also develop on other cool-season turfgrass species such as fine fescues (*Festuca* species), Kentucky bluegrass (*P. pratensis*), ryegrasses (*Lolium* species) and velvet bentgrass (*A. canina*), but is uncommon and rarely destructive on these grasses (7). Anthracnose also occurs on warm-season turfgrasses such as centipedegrass (*Eremochloa ophiuroides*) and bermudagrass (*Cynodon dactylon*) (6, 15).

Anthracnose of Cool-Season Turf

The pathogen causing anthracnose disease in cool-season grasses, the fungus *Colletotrichum cereale*, inhabits temperate climate zones across the United States, Canada, Western Europe, South America, Southeast Asia, New Zealand, and

Anthracnose on centipedegrass turfgrass. (Photo courtesy of M. Tomaso-Peterson)

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Anthracnose on annual bluegrass putting green turf. (Photo courtesy of B. Clarke)

Australia (3). *Colletotrichum cereale*, previously called *C. graminicola*, closely resembles other Colletotrichum fungi that cause anthracnose disease in corn, sorghum, switchgrass, sugarcane, and other members of the grass family. However, while *C. cereale* and other grass anthracnose pathogens are related to each other, DNA fingerprint analysis shows they are unique organisms, each infecting entirely different hosts (4).

Colletotrichum cereale has been documented in the United States for more than a century (7). The fungus was first described in 1908 from Ohio, where *C. cereale* was observed infecting several cool-season grasses, including Kentucky bluegrass, redtop (*Agrostis alba*), orchardgrass (*Dactylis glomerata*), and grain crops such as wheat (*Triticum aestivum*) and rye (*Secale cereale*) (3). Anthracnose of annual bluegrass was first diagnosed from New Jersey in 1928, where *C. cereale* was observed infecting roots and basal leaves (16).

Colletotrichum cereale inhabits many environments outside of golf courses, including ornamental grasses, residential lawns, meadows, forage grasses, prairies, and natural grasslands (7). Sporadic anthracnose disease outbreaks caused by *C. cereale* occurred in wheat, cereal rye, and oats (*Avena sativa*) during the first part of the 20th century, often in association with low fertility (3). Today, with the exception golf course turfgrass, *C. cereale* rarely triggers disease; however, the fungus is able to colonize other hosts without visibly affecting plant health (7).

Even though *C. cereale* infects a wide range of cool-season grasses, DNA fingerprint analysis of the fungus collected from cool-season grasses in the United States, Canada, Japan, Australia and Europe has shown that individuals from different host plants are members of 11 distinct population groups (7). These populations are predominantly divided according to host association. Only one of the 11 *C. cereale* populations includes individuals from both turfgrass and non-turfgrass hosts (for example, agronomic crop plants, native grasses, etc.). Most turfgrass strains of this pathogen possess unique DNA fingerprints that separate them from *C. cereale* collected from other host plants. Additionally, DNA fingerprints from individuals of *C. cereale* infecting annual bluegrass are dissimilar from fingerprints of the fungus infecting creeping bentgrass.

Anthracnose of Warm-Season Turfgrasses

Anthracnose in warm-season turfgrass is less common and less destructive than the disease that affects annual bluegrass and creeping bentgrass. Consequently, anthracnose has been poorly studied in warm-season turfgrasses. Recent DNA research has shown that anthracnose of warm-season turfgrasses is not caused by the same species of *Colletotrichum* that is responsible for disease in cool-season turfgrass (*C. cereale*), although these fungi do share similarities in their appearance (5). Given the many anatomical, structural, metabolic, and biochemical differences between cool-season and warm-season grasses, this finding is not surprising. The dissimilarity between the different anthracnose pathogens may reflect adaptations to the unique hosts that they infect.

Anthracnose of centipedegrass is caused by *C. eremochloae*, a newly discovered fungus that is a closely related sibling of the fungus responsible for sorghum anthracnose, *C. sublineo-*

la (6). Recent DNA fingerprinting of anthracnose fungi from preserved centipedegrass samples intercepted by United States port authorities in 1923 indicates that *C. eremochloae* was likely introduced along with the host when it was first imported from China during the early 20th century (6). Anthracnose on zoysiagrass (*Zoysia* spp.) and bahiagrass (*Paspalum notatum*) is associated with *C. caudatum* and *C. paspali* in Japan, but it is unknown whether these fungi are present on these turfgrass hosts in North America (3). The anthracnose pathogen of bermudagrass is a species of *Colletotrichum* that has not yet been identified.

Symptomology, Diagnosis, and the Disease Cycle: Host Infection by the Anthracnose Fungus

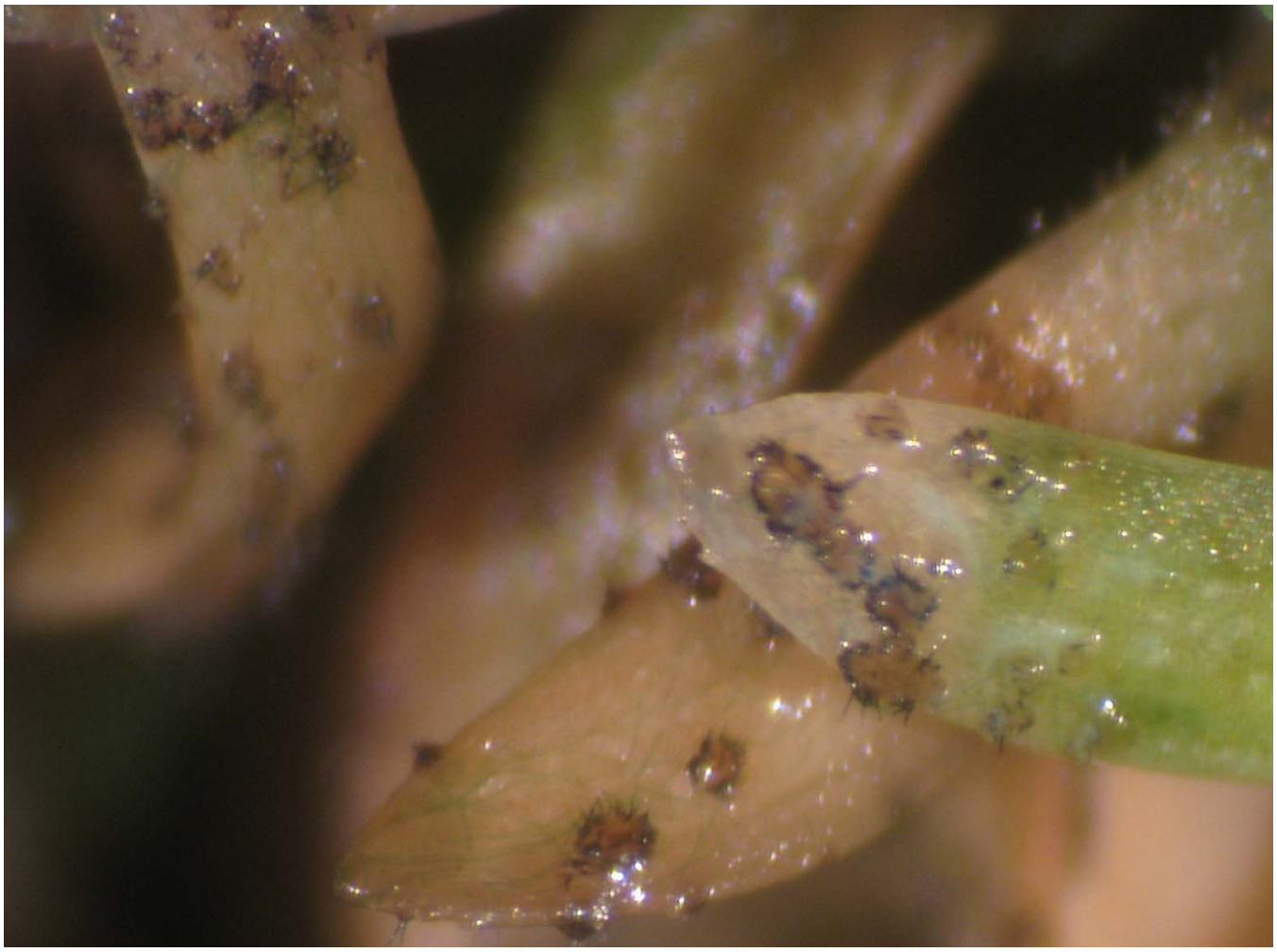
Knowledge of anthracnose biology in turfgrass is still incomplete. Nonetheless, several stages of the life cycle of are known, and research from related *Colletotrichum* grass pathogens provides insight into how the infection process presumably works in annual bluegrass and bentgrass.

Although anthracnose outbreaks sometimes occur on infected turfgrass in winter, the fungus is believed to be largely inactive during cold weather. *Colletotrichum cereale* is thought to survive harsh winter conditions as fungal mycelium in decaying plant residues or living host tissues. On oats and barley (*Hordeum vulgare*) crops, *C. cereale* survives the winter months by forming dormant resting structures called sclerotia on roots close to the soil surface, but this aspect of the fungal lifecycle has not been studied in turf (3).

Colletotrichum cereale spreads from plant to plant through the movement of crescent-shaped, asexual reproductive spores called conidia. Warm weather, high humidity, and high light intensity all serve to promote conidia formation. This is one of the reasons why anthracnose is so common in summer. Laboratory studies have shown that some isolates of *C. cereale* grow best between 70^{o} F and 88^{o} F (21^{o} C and 31^{o} C) and are able to cause foliar infection between 81^{o} F and 91^{o} F (27^{o} C and 33^{o} C) (8).

Colletotrichum cereale conidia are transported by wind and water, or mechanically through foot trafffic, mowers, or other equipment. Upon contact with a susceptible turfgrass plant,

Anthracnose symptoms on annual bluegrass putting green turf. (Photo courtesy of B. Clarke)



Colletotrichum cereale acervuli with setae on leaf sheath of annual bluegrass. (Photo courtesy of L. Stowell)

the conidia germinate within approximately two to six hours, and a structure called a germ tube is produced. Formation of the germ tube leads to the development of a suction cup-like object called an appressorium, a dark-brown, dome-shaped structure measuring less than 0.01 inch (0.25 millimeter) in diameter. Appressoria (plural of appressorium) adhere firmly to the host, allowing the fungus to penetrate the outer plant cuticle into the underlying tissue via a penetration peg that forces its way into the plant using tremendous pressure.

Although this pressure has never been calculated for *C. cereale*, optical wave measurements have shown that a single appressorium from the related corn pathogen, *C. graminicola*, is able to exert forces up to 25 μN on host tissue (1). To put this in perspective, if a force of 17 μN was exerted across the palm of a human hand, that individual would be able to lift a school bus weighing almost 17,000 pounds (7.7 metric tons). Appressorium formation has been observed within six hours after conidia make contact with leaves or stems, and penetration of host tissue may occur within 24 hours (12). High humidity and/or extended periods of leaf wetness are critical during this time, since desiccation of germinating condia will greatly reduce the potential for infection.

Symptoms and Diagnosis of Anthracnose on Cool-Season Turf

It is unknown how long it takes for symptoms to develop once *Colletotrichum cereale* conidia first adhere to the surface of a susceptible host, and symptom expression may vary depending on environmental conditions. For example, in field studies performed over consecutive years in

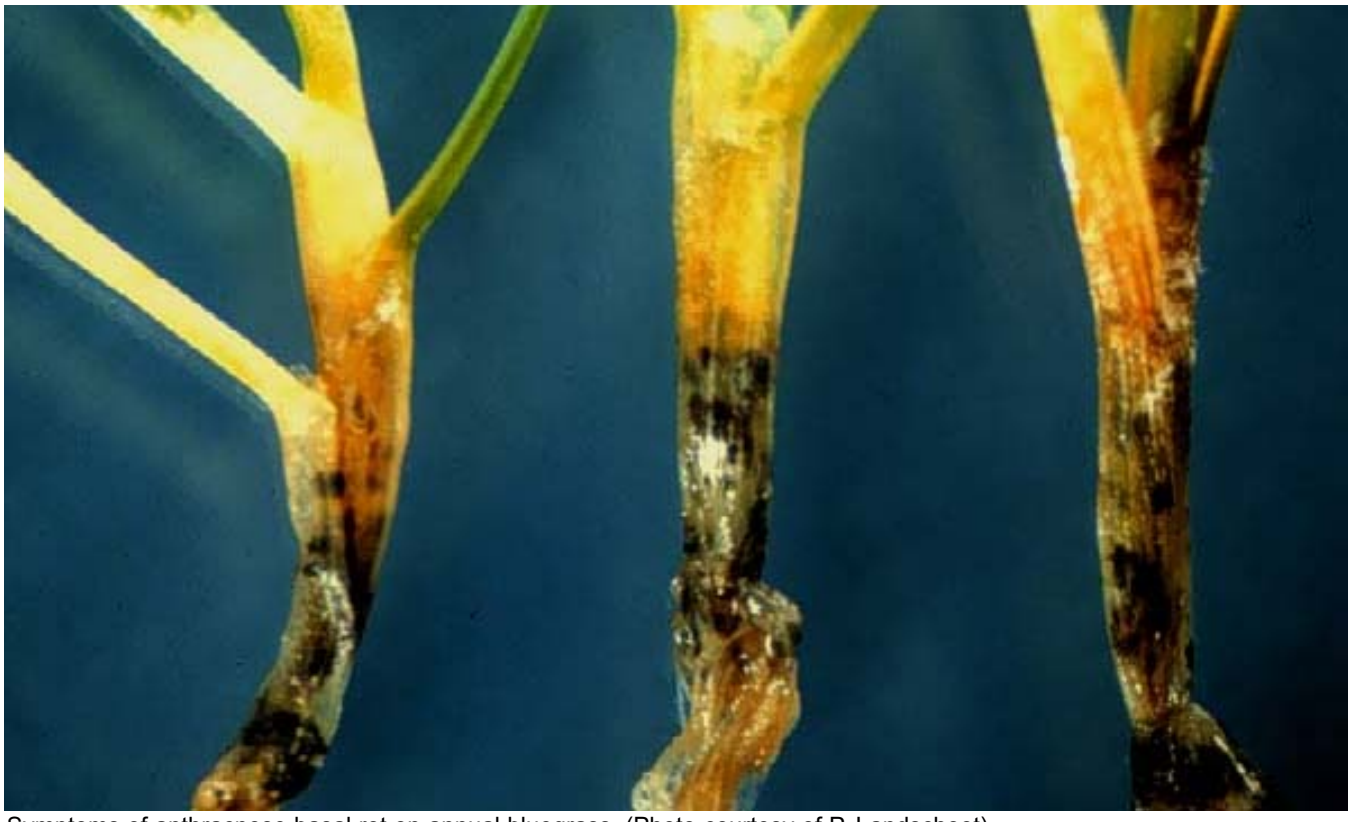
New Jersey, annual bluegrass turf exhibited symptoms of basal rot anthracnose between five or 18 days after inoculation with *C. cereale* conidia (10).

Infection of leaf or stem tissue with *C. cereale* results in the development of acervuli (reproductive bodies containing conidia) on the surface of the host tissue, and these structures may become abundant as the disease progresses. Acervuli are cushion-like fruiting bodies composed of a mat of fungal hyphae. Hyphae within acervuli give rise to two important structures: setae and conidia. Setae are thin, tapering, black hair-like structures that project from the acervuli. These structures are a key diagnostic feature of the anthracnose fungus and can be easily detected using a hand lens. The acervuli are also the primary site of conidia production and provide a source of future infections.

Conidia within the acervuli are suspended in a sticky liquid that serves to protect the conidia from drying, prevents the conidia from premature germination, and may aid the fungus in initiating infection (3). When acervuli are splashed with water, this liquid is diluted, germination inhibitors are broken down, conidia are moved to fresh new host tissue, and the disease cyle begins anew.

Anthracnose is most severe during warm, humid weather. Nonetheless, outbreaks may occur throughout the year, causing either a foliar blight or a basal rot of leaf sheaths, crowns, and stolons (15). Both basal rot and foliar blight symptoms are favored by high temperatures (85-95^{o} F; 29-35^{o} C) and may be present simultaneously on annual bluegrass, especially when turf is under heat stress.

Anthracnose symptoms on cool-season turfgrasses vary based on the host plant. Symptoms on annual bluegrass initially emerge as yellow- to orange-colored spots between 0.25 and 0.5 inch (0.64-1.27 centimeters) in diameter (15). On creeping bentgrass, infected foliage becomes orange or red to reddish brown. If left uncontrolled, spots coalesce, forming large, irregular areas of blighted turf. Older or senescent leaves may be among the first colonized by the fungus. Infection of leaves close to the soil surface in close-cut turf often results in a characteristic "basal rot" symptom, where water-soaked, blackened tissue is easily pulled away from the crown.



Symptoms of anthracnose basal rot on annual bluegrass. (Photo courtesy of P. Landschoot)

Factors Affecting Disease Severity

Scientists have observed that anthracnose is generally most destructive on weakened or senescent turf (15). During the past 10 years, research performed on annual bluegrass greens has provided data in support of this conclusion. Anthracnose may cause extensive injury when turf is maintained at a low height of cut, when fertility is low, or when the turfgrass is stressed by abiotic factors such as drought or excessive heat (10, 11, 13-15). However, contrary to earlier theories, data from recent field studies show that activities causing wounded tissue (for example, foot traffic, rolling, double-cutting and verticutting) do not appear to enhance the severity of anthracnose on turfgrass (10, 11, 13, 14).

Consequently, although plant health and vigor have been demonstrated as major factors influencing disease severity, additional factors such as cultivar susceptibility, environment, and variation in pathogen populations may also play roles in disease development. In particular, variable response to anthracnose has recently been documented on cultivars and experimental selections of creeping and velvet bentgrass (2). Anthracnose response from commercial cultivars of creeping bentgrass evaluated at Rutgers University ranged from very good disease tolerance (‘Shark’, ‘Penneagle II’, ‘Runner’, ‘Penn A-1’, ‘Tyee’, and ‘Authority’) to highly susceptible (‘Viper’, ‘Providence’, ‘Penncross’, ‘Brighton’, ‘Seaside II’, and ‘Pennlinks II’) (2). Similar research is ongoing with annual bluegrass, with certain clones and selections being developed at Penn State University that show improved tolerance to anthracnose disease (9).

Future Research

Much remains to be learned about the anthracnose infection process and how differences across diverse populations of *Colletotrichum cereale* may affect disease severity and control on golf course putting greens. Progress in this area has been hindered by the lack of a reliable greenhouse inoculation procedure. Field inoculations have been sucessfully employed to evaluate the impact of management factors on anthracnose severity, resulting in the development of an evolving set of best management practices that have benefited superintendents throughout the world.

Development of a reliable greenhouse inoculation protocol will facilitate valuable investigations of turfgrass anthracnose disease under controlled environments, including dissections of the infection cycle, pathogenicity testing, screening of turfgrass cultivars for anthracnose resistance, and experimental studies of the factors influencing host-pathogen interactions. Moreover, the use of an increasingly powerful and rapidly expanding set of DNA and genetic tools, including DNA sequencing of several anthracnose pathogen genomes, is continuing to enhance our ability to study and identify the mechanisms involved in the infection process and the suppression of anthracnose through improved management practices. This should also speed up new cultivar development by allowing breeders to identify and screen turfgrass selections for anthracnose-resistance genes before even stepping foot into the field. The knowledge gained from this type of research will greatly enhance our understanding of anthracnose and the ability of superintendents to control this devastating disease in the future.

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