AN INTEGRATED APPROACH TO SUMMER PATCH CONTROL IN TURFGRASS

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Summer patch was first recognized as a disease of cool-season turfgrasses in 1984. Prior to that time, it was an unidentified component of Fusarium blight. Summer patch has been reported in North America on fine fescue and Kentucky bluegrass. The causal agent has also been isolated on occasion from bentgrass and perennial ryegrass. The disease generally occurs on turf that has been established for more than two years.

SYMPTOMS

On Kentucky bluegrass, symptoms first appear in early summer as small, circular patches of wilted turf 1.5 to 3.5 inches in diameter. Patches may enlarge to more than 24 inches, but generally remain in the 2 to 12 inch range. Affected leaves rapidly fade from a grayish-green to a light straw color during sustained hot weather (daytime highs 82 to 95°F and nighttime temperatures exceeding 68°F). Irregular patches, rings, frog-eye and crescent patterns may also develop and coalesce into large areas of blighted turf.

In mixed stands of bentgrass and bluegrass maintained under putting green conditions, patches are circular and range from 1 to 12 inches in diameter. As annual bluegrass yellows and declines, bentgrass species frequently recolonize patch centers. On fairways and lawns, rings or frog-eye patches may not develop. In such cases, symptoms may appear as diffuse patterns of yellowed or straw colored turf that are easily confused with heat stress, insect damage, or other diseases. Infected roots, rhizomes, and crowns turn brown as they are killed. Examination of these tissues typically reveals a network of sparse, dark brown to black, ectotrophic hyphae from which hyaline penetration hyphae invade the underlying vascular tissue. In the latter stages of infection, vascular discoloration and cortical rot are extensive. No fruiting structures have been observed under field conditions.

CAUSAL AGENT

Magnaporthe poae Landschoot and Jackson, the causal agent of summer patch, is a newly described fungus whose asexual stage had previously been misidentified as Phialophora graminicola (Deacon) J. Walker. The fungus forms dark brown to black, septate, ectotrophic runner hyphae on roots, crowns, and rhizomes of turfgrass hosts. Sexual fruiting bodies, which have only been observed in culture, are black, spherical and have long cylindrical necks.

DISEASE CYCLE

The pathogen is believed to survive the winter months as mycelia in previously colonized plant debris and in perennial host tissue. Colonization and suppression of root growth has been shown to occur between 70 and 95°F under controlled environmental conditions, with optimum disease development at 82°F. In the field, infection commences in late spring when soil temperatures stabilize between 65 and 68°F. The fungus moves from plant–to–plant by growing along roots and rhizomes. Symptoms develop during hot (86 to 95°F), rainy weather or when high temperatures follow periods of heavy rainfall. Patches may continue to expand through the summer and early autumn and are often still evident the following growing season. The summer patch fungus may be spread by aerification and dethatching equipment as well as by the transport of infected sod.

EPIDEMIOLOGY

Summer patch is most severe during hot, wet years and on poorly drained, compacted sites. Although heat stress plays an important role in disease development, drought stress is usually not a predisposing factor. Under ideal conditions, the causal agent can spread along roots, crowns, and stem tissue at a rate of up to 1.5 inches per week. Symptom expression has been shown to increase with the use of nitrate–based fertilizers, arsenate herbicides, and many commonly used contact fungicides. The disease is frequently stimulated when turfgrass is maintained under conditions of low mowing height, high pH (> 6.0), compaction, and frequent, light irrigation.

CONTROL

Because summer patch is a root disease, cultural practices that alleviate stress and promote root development will reduce disease severity. Since low mowing enhances symptom expression, avoid mowing turf below recommended heights, particularly during periods of heat stress. In the Northeast, symptoms are less apparent when lawns are maintained at a height of 2 to 3 inches and golf greens and fairways are cut at or above 5/32 and 3/8 inches, respectively. Fertilize turf with ammonium sulfate or a slow–release nitrogen source such as sulfur–coated urea. Irrigate deeply and as infrequently as possible without inducing drought stress. Aerification, improving drainage, reducing compaction, and syringing to reduce heat stress are other practices that will aid in the control of this disease.

Overseeding affected areas with perennial ryegrass, tall fescue, or resistant cultivars of Kentucky bluegrass represent one of the most cost–effective means of controlling summer patch. Use mixtures or blends of resistant turf cultivars or species for best results. Conversion of golf areas from bluegrass to bentgrass will also reduce disease incidence.

Fungicides are available that can effectively control summer patch. Applications should commence on a preventative basis in late spring or early summer when soil temperatures stabilize between 64 and 68°F. Systemic fungicides, such as fenarimol (Rubigan), propiconazole (Banner), triadimefon (Bayleton), and the benzimidazoles (i.e., Tersan 1991 and Cleary 3336), have proven to be most effective but must be applied at high label rates. Repeat two to three times at 21–28 day intervals for best results. Efficacy is enhanced when products are applied in at least 4 gallons of water per 1000 square feet. The continued use of contact fungicides at high label rates may stimulate symptom severity.