NEW TWISTS TO OLD PROBLEMS

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ANTHRACNOSE BASAL ROT

Under cool, wet conditions, the anthracnose fungus, Colletotrichum graminicola, causes a basal rot on Poa annua. This is the most prevalent form of anthracnose in Pennsylvania. Affected turf is thinned and takes on a mottled yellow appearance. Individual plants appear yellow, with older leaves showing discoloration first. The bases of shoots and leaf sheaths are blackened, and the entire shoot is easily separated from the crown. The black discoloration results from heavy accumulation of mycelium of the causal fungus, and the black-spined spore-bearing bodies (acervuli) that characterize anthracnose may be seen on the blackened bases of central shoots or leaf sheaths. By the time the symptoms are noted, individual plants may be irreparably damaged. Management strategies for anthracnose basal rot include: increased aeration and fertility, and treatment with high label rate combinations of Tersan 1991 or Bayleton with Daconil.

BLACK LAYER IN PENNSYLVANIA

The first time I noticed a 'black layer' was many years ago in a plug submitted by a golf course superintendent. In fact, I didn't really notice the black layer, but rather the pungent stench that emanated from the plug when I unwrapped it. There was no black layer apparent on the outside of the plug, but when I broke it in half, not only did the stench increase, but there IT was. I was working for Herb Cole at the time, and since I had never seen such a thing before, I called him and told him to come and see this peculiar phenomenon. By the time he arrived several hours later, there was neither a stench nor a black layer, and my credibility with my boss was irreparably damaged. Little did I realize that what I had seen was, not only 'black layer', but the cure as well!

Research at Michigan State and elsewhere has shown that black layer develops in the presence of sulfur, sulfur-reducing bacteria, and anaerobic soil conditions. Soil sulfur can occur naturally, or can be introduced as additives (fertilizers, fungicides, etc.), organic matter from plant and animal sources, or from the atmosphere as sulfur dioxide or acid rain. It is likely that one of the chief sources of sulfur in turf soils in humid regions of the US is organic matter of plant origin. This would appear to mean that there is probably sulfur in any soil where grass is growing. We discovered this at PSU, much to our dismay, some time ago in a greenhouse study of Pythium blight. We had infected sand-grown, seedling ryegrass with Pythium aphanidermatum, watered it heavily, and put it in plastic bags at high temperature to await the development of Pythium blight. Within 48 hours, other people working in the greenhouse began

to complain loudly about the foul odor coming from our experiment. The grass was dead, albeit not from Pythium blight. There was no black layer, but the roots of the seedling grass appeared as jet black trails throughout the sand growth medium. The roots had probably been killed by lack of oxygen, but apparently the root material contained enough sulfur and metal ions to allow production of the black sulfides that characterize black layer.

When the turfgrass root zone becomes water-logged or anaerobic, roots are in jeopardy from lack of oxygen and accumulation of carbon dioxide. This condition alone could cause root loss and plant death. However, once anaerobic conditions are present, other detrimental side effects result. Hydrogen sulfide, organic acids, and metal sulfides accumulate, and roots cannot survive long in such a toxic environment.

Spring and summer of 1990 in Pennsylvania were characterized by periods of heavy rain -- and black layer. Problems with black layer were not confined to sand greens, but occurred in both modified and unmodified turf soils -- in poorly drained areas. Irregular patches of stunted, yellow Poa annua appeared in these areas, and in mixed stands, bentgrass was usually not affected. Individual plants were bright yellow, and roots of affected plants showed dark surface blotches and often had blackened steles. Cup-cutter plugs from affected areas were usually characterized by surface algae, a layered soil profile, a blackened area or layer somewhere in the profile, and a foul odor. The two latter characteristics tended to dissipate when the plug was broken apart or allowed to stand.

The common factor in these black layer episodes last year in Pennsylvania seemed to be water-logged soils, therefore anaerobic soil conditions was probably the limiting factor present. The remedy for the problem was therefore difficult, for as Dr. Vargas has pointed out, it is very tough to put tents or umbrellas over golf courses. Strategies for improvement of soil aeration and drainage are essential for management of black layer, however, and vary from reconstruction, through installation of sub-surface drainage, vigorous cultivation, and avoidance of any kind of layering in the soil profile.

SUMMER PATCH, DOLLAR SPOT, AND FUNGICIDE RESISTANCE

There was evidence last summer, in Pennsylvania and elsewhere, that there may be a connection between the above three problems on turfgrasses. Summer patch or Poa patch, caused by Magnaporthe poae, occurs in mid- to late summer, primarily on annual bluegrass. The disease appears during periods of high temperature and high soil moisture as circular patches of yellow, dying plants. In mixed annual bluegrass/bentgrass stands, bentgrass is largely unaffected. Dark strands of the causal fungus may be seen on crown and root surfaces, and roots of affected plants may show blackened steles. Chemical control recommendations usually stress preventive monthly applications of high rates of a sterol-inhibiting systemic fungicide, with applications beginning as early as April and continuing throughout the summer.

During the past summer, there were disturbing reports, from superintendents in Pennsylvania and adjacent states, of shortened intervals of dollar spot control with Bayleton. These reports have not (to my knowledge) been verified as reduced Bayleton sensitivity in isolates of the dollar spot fungus from the suspect sites. I do think, however, we need to consider the possible effect of heavy inputs of sterol-inhibiting fungicides for summer patch control on populations of the dollar spot fungus in the treated areas.

Most systemic fungicides act on fungi at a single location in their growth and development cycles. It is relatively likely that the target fungus will produce some off-spring that can short-circuit the poisoned site. If the site is under the control of only one gene in the target fungus, resistance in the fungal population will occur at a very high level and will develop rapidly and explosively. This is the sort of resistance development we see in the dollar spot fungus to Tersan 1991 and other benzimidazole fungicides, and in the Pythium blight fungus to Subdue. The sterol-inhibiting fungicides (Banner, Bayleton, and Rubigan) also act at single sites in fungi, but the target site is under the control of multiple fungal genes. The development of resistance to these fungicides will, therefore, appear as gradually decreasing sensitivity over time. One indication of such a change is the shortening of control intervals -- such as was reported by superintendents during last growing season. This reported control decrease may or may not be related to decreased sensitivity in the dollar spot fungus to the sterol-inhibitors. One thing is certain, however. The repeated and exclusive use of any group of systemic fungicides is risky from the standpoint of resistance development. Diversity in chemical use is essential for preventing or delaying resistance to fungicides in fungal populations.