

Antracnose, Summer Patch and Poa annua Decline: A 1990 Perspective

By Dr. Gayle L. Worf Department of Plant Pathology University of Wisconsin-Madison

In spite of efforts over the past several years to eliminate it, *Poa annua* remains the primary fairway turf on most of our more intensely managed golf courses in Wisconsin. It's not the subject of this article to discuss whether that is good or bad. But because it's there, most superintendents have to face the job of keeping *Poa* alive during critical summer months.

That is the subject for today.

Research from a number of locations has demonstrated that *Poa* on northern golf courses can be kept alive and functional during the summer by careful attention to compaction problems, irrigation and nutritional management practices — if these are also combined with protective fungicide treatments. Diseases are important, and their control is critical.

In addition to the diseases that attack most grasses, e.g., dollar spot, brown patch and Pythium, we have to deal with some that are peculiar to *Poa*. Anthracnose is one of those, and summer patch it another.

If you are not really sure of what's attacking your Poa, you may have good reason to be confused. Their symptoms can overlap, but these two diseases are usually easy to tell apart. Anthracnose is usually recognized as a leaf blight problem that causes some general yellowing and thinning of the turf and, in its extreme, some serious kill in erratic patterns. Summer patch causes a sudden appearance of dead and dying Poa in distinct circular patches that range from 4 to perhaps 14 inches in diameter. Anthracnose often appears in early July, while patch rarely occurs in Wisconsin before August. Anthracnose is more subtle. Patch is very distinct, unless there's enough unaffected bentgrass in the mix to mask its effect.

Controlling anthracnose and summer patch Best treatments for anthracnose include two sterol inhibitors — Banner

and Rubigan — and also Daconil. (The benzimidazoles also perform well against anthracnose, but there's too much dollar spot disease resistance on most courses to consider this alternative.) These need to be applied before the ''anthracnose season'', e.g., **about the first of June** during a warm year. Two bi-weekly applications of the sterol inhibitors, followed by Daconil treatments seem to work well in controlling anthracnose, dollar spot, and suppressing brown patch.

Banner, Bayleton and Rubigan appear to perform well against summer patch. However, the timing is different. There's a consensus emerging among turf pathologists that says the fungus starts invading the crowns and roots of the turf in the patches, beginning when the soil temperatures are reaching 65°F. Several pathologists from other states have reported success with early applications, when applied at the highest label rates. Probably, the earlier in the season that a consistently warm soil temperature occurs, the greater will be the damage from summer patch (and anthracnose, too).

Our results in 1988, at Nakoma, and 1989, at Pine Hills, would support those observations. Control was good with our better treatments at Nakoma — we applied in late April and May. We didn't take soil temperatures in 1988, but we all remember what a warm year it was. But last year the soil temperatures at Pine Hills stayed cool way into June, according to our thermometer readings, and we didn't encounter any disease. This is in spite of a bad summer patch history there for several previous seasons.

Most believe that irrigating right after treatment improves chances for controlling summer patch.

Problems with dovetailing anthracnose and summer patch control

You no doubt noted that summer patch treatments are earlier than what

we've been suggesting for anthracnose control needs. And making four applications before any disease shows up is way too much — for lots of reasons — too much chemical, too expensive, potential fungicide resistance, potential phytotoxicity are some of them.

For some of you this shouldn't be a problem. My impression is that both problems don't occur on every course or every fairway. Disease history should serve as a starting point in deciding what you need to protect. Anthracnose appears to be much more common than summer patch in Wisconsin. If I were losing *Poa* in the summer without knowing why (and I had eliminated traditional diseases, Atenius, drouth, hungry turf and excessive compaction!), I'd be suspicious of anthracnose.

Do we get useful anthracnose control from the early spring (summer patch) treatments? Maybe. Perhaps a single early application, followed by a mid-June treatment and subsequent summer contact-type fungicide applications would be a satisfactory compromise where both diseases are a problem. We haven't had opportunity to evaluate this in our trials to date nor have I seen such information from research reports elsewhere. But we hope to gain some insight on that from this year's work.

Additional comments on anthracnose

There appears to be a considerable variation among Wisconsin isolates. We've been working this winter in the growth chambers with six Wisconsin isolates. Several have not shown any inclination to cause turf damage, regardless of different temperature and inoculation approaches we've tried.

But a couple are quite pathogenic. At about 75° to 82°F, there is severe yellowing and killing of the foliage, and after two or three recycles, the inoculated turf begins to die. If we had any doubt about *Colletotrichum*'s ability to cause serious Poa damage, working with this isolate would erase any doubt.

The variability is probably real, and may account for differences from one golf course to another in their experiences with anthracnose.

The literature on turf anthracnose damage is argumentative. Some says it is not important, and is only a secondary problem. At worst, according to this thesis, it causes older, senescing leaves to turn yellow and to guit prematurely. But loss of Poa has been attributed to anthracnose for many years, beginning with work done by Drew Smith in Canada, and supported by Vargas' work in Michigan. Publishing in 1954, Smith described anthracnose as starting as small patches of yellowish dying grass which increase in size up to 6 inches or so in diameter, assuming an irregular outline. Smith emphasized root and collar effects - he scarcely mentions direct damage to foliage. He described a deterioration of the base of the individual shoots, root systems that were poor, and with brown or black discoloration. Indeed, his description of Poa loss is very close to what we usually encounter in our most severe situations.

Smith didn't close the loop entirely in confirming these symptoms to be caused by *Colletotrichum* (anthracnose). He only worked with seedlings through the seminole root phase. Nor have we done so, through the inoculations described above, or via root inoculation trials which we've also tried.

We lean more closely today to the idea that "Wisconsin *Poa* decline" is in fact caused primarily by a root and crown-rotting phase of anthracnose. But the proof is still lacking. There are some other possibilities. We'll continue to struggle with it, maybe add another chapter this summer. We need more isolations and samples from diseased *Poa* this summer — maybe from your course!





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