Shifting Fungicide Responses and Anthracnose Control

By Dr. Gayle L. Worf
Professor and Extension Plant Pathologist
University of Wisconsin-Madison

Fungicides that are very effective for a period of time do not always remain that way. Today that is a recognized fact of life among turf management professionals, and serious efforts are underway at many levels, both to understand what's happening, and to develop realistic and effective management strategies to cope with it.

One such area of concern has been the diminished control by Bayleton over the past 15 years in its control of Poa decline, of which anthracnose disease (*Colletotrichum graminicola*) is a recognized component of the problem.

First, we should point out that Bayleton remains a very effective tool against many diseases. And the alleged loss of anthracnose control has not occurred on all courses. I frankly have no idea of the extent to which this change has occurred. I know that it is not a problem everywhere, and the conclusion shouldn't be reached when control failures are encountered, that it is a result of an ineffective chemical. We've known from the beginning that control of this disease has depended as much upon a rigorous, properly timed preventive application schedule as well as product. It also depends upon good nutrient and cultural management. Control isn't possible in severe years if nitrogen stress occurs or severe compaction problems are encountered. And when you are working on fairways, have restricted the number of applications because of a limited budget, or perhaps by trying to minimize chemical applications through an IPM strategy, it can add to the confusion when standing on the course trying to analyze what went wrong!

Probably the most confirming evidence in the field of fungicide resistance comes when you are looking at fungicide plots on courses where there is a history of disease development and chemical response. I recall in earlier years marvelling at the beautiful green dense turf following Bayleton treatment, and the agony in subsequent years on the same courses to see the thin yellow diseased turf under similar circumstances. The good news in those instances has been the excellent control still obtained by other chemicals.

Another way to check fungicide "tolerance" is in the greenhouse and laboratory. Two approaches come to mind, and both should be employed where possible. The "in vitro" study involves comparing fungal growth and development of different cultures (or isolates) of the fungus in petri plates to which increasing levels of the fungicide have been introduced into the growing media. Where the fungus is still sensitive (and therefore controllable) it takes only a small amount of chemical in the media to stop growth, but as sensitivity to the fungicide is lost, it takes more and more chemical. It's important to have cultures representing the "before fungicide change" and "after fungicide change" for best comparison and interpretation. Whenever possible, trials involving inoculating and protecting plants with fungicides, e.g., so-called "in vivo" tests, should also be included. This might be particularly important for systemics and sterol inhibitors (SI's), because of potential artifacts in the in vitro study.

Responsible chemical companies are probably more concerned about possible shifts in control with their products than either you or I might be. The Mobay Corporation, manufacturer of Bayleton, has certainly been attentive of this situation, not only because of their product but because of general concern to keep the valuable sterol inhibitor fungicides available and useful into the future. A year ago they provided us with financial support for such a study. I want to share with you what we did and what we found.

We did not have cultures remaining in our fungal collection of *C. graminicola* from courses dating back to the time when Bayleton control was good. So we did what we thought was the next best thing. We used five different isolates, including two (1988 and 1990) from the Nakoma golf course where Randy Smith, Chuck Frazier and the entire membership have patiently put up with our requirements and their interrupted play for so many years, and where we have best history information. Another isolate came from the roots of a Trout Lake diseases Poa specimen. The other two were without specific history. In vitro studies were conducted with 0 ("check"), 0.1, 1, 10 and 100 ppm active ingredient levels of each of the fungicides listed in the tables below. All chemicals except Daconil are registered or experimental sterol inhibitors about which we have some field history of their effectiveness. Daconil was included as a contact fungicide which suppresses the disease, though less effectively than SI's when the latter are working.

In the following figures I've included in vitro results from three isolates, including the two from Nakoma (numbers 1 and 5). Number 1 was isolated in 1988, the other in 1990. Both were after several years of Bayleton use, and after failures were observed. Isolate three was from Trout Lake, where we do not believe Bayleton use pressure had occurred. For the remaining two isolates, generally good suppression occurred with all fungicides although the same trends existed.

We offer two observations. The fungus in every instance was least affected by Bayleton in these tests. The comparisons were virtually the same with all fungicides and isolates, with Daconil not quite as active in vitro (which is what we would have guessed) than the other SI's, which were virtually identical in inhibiting fungus growth at various concentrations. This follows what we saw in the field and tends to support the suspicion that Bayleton activity is diminished.

Secondly, the extreme lack of sensitivity in the Trout Lake isolate strongly intimates that resistance is already present in the environment, and is not "created" by Bayleton use. But Bayleton use may select out and favor such strains.

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What is certainly significant in these observations is the fact that other SI's did *not* show similar evidence of developing resistance. That's encouraging! It leaves us with the hope that these chemicals are not all peas in a pod when it comes to that sort of thing.

What of the in vivo (grass inoculation) studies? No disease developed. Not after one, two or three separate inoculation attempts. Not with varying environments, and not with root inoculation attempts, either.

So we were not able to look at alleged fungicide resistance from that angle. Aside from this study, such results could raise the question of significance of the anthracnose fungus as an important causal agent in the Poa decline syndrome. (Back to that story again!) Interestingly, we had developed some good foliar symptoms in inoculation studies (for another purpose) in some previous winter studies with identical foliar inoculation procedures.

I urge you not to read into this report more than you should. Bayleton remains a good fungicide for many purposes. There's none better for dollar spot, and if you're working primarily with bentgrass with desires to keep out the Poa, allowing anthracnose to work on the Poa (decline doesn't affect bentgrass) makes sense from a biological control approach. Also, Bayleton is one of the more economical fungicides.

And Bayleton literally blazed the trail for the SI's. It was around for years before the next members could gain EPA approval. My most memorable experience with fungicides was the way we could virtually write our name in the grass with the control it provided in its early days.

I can't resist mentioning one more time how intriguing the whole question of Poa decline really is. Over the past twenty years it has become apparent that Poa doesn't "die spontaneously"; that anthracnose can contribute, but it's complete role remains controversial. What I define as Poa decline starts with severe root loss. Anthracnose can cause root rot on some other hosts but I'm unaware of any proof of that on turf. Poa summer patch has become a hot subject the last few years, especially in eastern states. It can really do a number on Wisconsin turf, as many of us have seen. But in my mind it's not the cause of most of our Poa loss, unless it occurs in a much more subtle fashion than anyone has demonstrated to date. Really, until the etiology of this problem is better understood, my view is that the ultimate usefulness of greenhouse and laboratory studies like this remains severely handicapped.

I've always enjoyed mysteries!