Anthracnose: prevention is the name of the game

by Wendy Gelernter, Ph.D. and Larry J. Stowell, Ph.D.

Bottom line: During the summer of 2001, *Poa annua* greens across the country were hard-hit by anthracnose, a disease caused by the fungus *Colletotrichum graminicola.* In many areas of the country, anthracnose is now the most difficult to manage disease on *Poa* greens. Anthracnose is sometimes regarded as a secondary disease that is merely a symptom of more serious, underlying stress. However, this is more of a theoretical, rather than a practical viewpoint, since *Poa* greens during the summer are almost always under stress due to compaction, soil salinity, heat, drought, and low mowing heights. For this reason, we view anthracnose as a serious pest of *Poa* greens that warrants equally serious counter measures. Effective management relies on a two-part approach, starting with a preventive cultural program, including practices such as regular aerification, traffic management and leaching programs. A preventive fungicide program that is triggered in late spring/early summer by average air temperatures above 65F further aids in prevention. This preventive approach is by far the best way to avoid the severe and long-lasting damage caused by this disease. In contrast, an attempt to "cure" anthracnose once it has occurred is a difficult, if not impossible task.

Figure 1. Initial symptom of anthracnose on a *Poa annua* putting green. Note the yellowing (chlorotic) turf at the edge of the patch; this indicates that the disease is active, and will continue to move outwards, thus expanding the size of the patch.

Basal rot vs. foliar anthracnose

Anthracnose can be confusing because the same fungus -- *Colletotrichum graminicola* --can produce two somewhat different symptoms on two different parts of the plant. When the disease attacks the **foliage** (this is sometimes called leaf blight anthracnose), the oldest leaves are attacked first (Figure 6). The initial damage shows up on the leaf tips, which turn yellow to reddish brown. Eventually, the whole leaf becomes discolored, and ultimately, even the younger leaves will become infected. As the infection progresses, numerous black fruiting bodies (acervuli) that have black spines (setae) can be seen on the foliage (Figures 6 and 7). When the fungus attacks the stems and crowns, the disease is frequently referred to as **basal rot or crown rot anthracnose.** Under these conditions, the plant tissue on the stems, crowns and at the bases of leaf sheaths becomes watersoaked and eventually dark and discolored. Acervuli are produced here as well, and the plant almost always dies. This is a particularly nasty form of the disease, because it is hard to detect the initial symptoms, which develop beneath the foliage, at the base of the plant. By the time the foliage starts to show more obvious symptoms, the disease is too far gone to control. And because the crown is the area of the plant where new leaves and roots are produced, attack by basal rot anthracnose can actually kill the whole plant. Control of basal rot is also more difficult because the fungus develops underneath the leaf sheaths, or buried within the crown -- areas that are hidden, and therefore almost impossible to target with fungicide applications.

Figure 2. As the disease progresses, irregular patches of chlorotic and dead turf occur throughout the green.

Tracking the spread of infection: a case study

Let's say you have a green that had a few patches of anthracnose last year, but never developed serious damage. You assume (quite reasonably) that the disease shouldn't cause you much trouble the following year. Instead, you experience serious damage that keeps getting worse and worse, despite repeated fungicide applications. What happened?

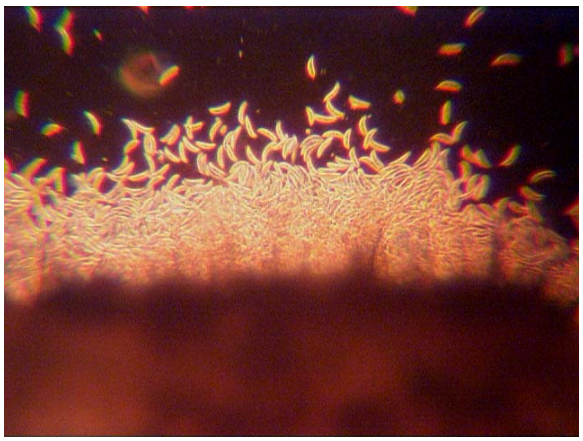
It turns out that once you have anthracnose, it's almost impossible to rid of it. That's because it has the ability to survive adverse conditions (cool weather, low humidity, even fungicide applications) by going into an

inactive stage, and scrounging the minimal nutrients it needs from dead plant tissue in the thatch, or on stems beneath the leaf sheath.

Figure 3. Advanced stages of infection. Note that the areas of anthracnose-killed turf have been colonized by a blue-black layer of algae (cyanobacteria), which use the dead and dying turf as a nutritional source.

When conditions become right for growth of anthracnose (air temperatures in the 70s or higher, muggy weather, and turf that is more susceptible to attack due to the stresses of compaction, heat, salts or other factors), the fungus begins to grow again and starts to produce **conidia** (Figure 4) -- the small, seed-like structures that it uses to propagate itself. The conidia spread easily from plant to plant primarily by splashing water droplets -- from irrigation or rain. They can also be moved by vehicular or human traffic.

Figure 4. Thousands of small, crescent-shaped anthracnose conidia are produced in each acervulus.



When anthracnose conidia land on a new turf plant, they germinate, producing tubular, filamentous hyphae that grow on the plant surface. The formation of appresoria (penetrating structures formed at the ends of certain hyphae), allows the fungus to break through the plant surface, and to grow internally, inside the plant tissue (Figure 5).

Figure 5. Hyphae and appresoria of anthracnose.

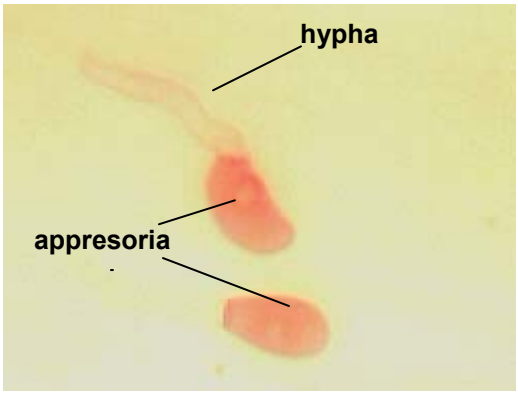
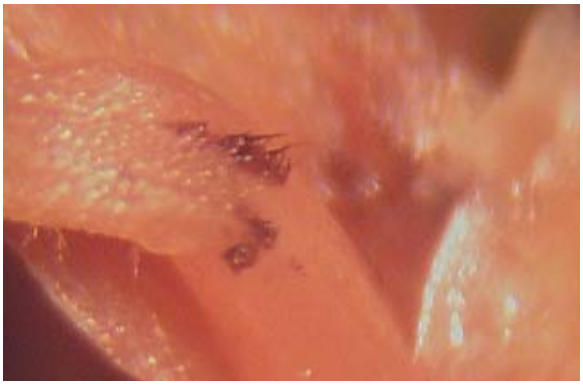


Figure 6. Once the fungus has grown inside the plant tissue for some time, it produces acervuli (see arrow) -- dark colored fruiting bodies with hair-like spines studding their surface. When seen under low magnification (5 X), acervuli may look like small pieces of soil or charcoal.

Figure 7. Higher magnification makes it easy to see the hair-like setae of the acervulus. Each acervulus contains thousands of conidia (see Figure 4) which are capable of infecting new plants with the disease.



Acervuli are produced after infected tissues die and collapse, forming a sunken area in which the acervulus forms. Though the acervuli are formed inside the plant tissues, they eventually break through the surface of the tissue, erupting onto the surface.

The entire life cycle of anthracnose -- from the time the conidium lands on a new and healthy plant, to the time that the plant is infected internally, and has conidium-filled acervuli erupting from it's tissue -- takes only 10 - 12 days. It's easy to see why the disease can spread so rapidly when conditions are right.

Anthracnose vs. Poa annua: an unfair contest

Anthracnose can occur on a wide variety of turfgrass hosts: *Poa annua*, creeping bentgrass, Kentucky bluegrass, fine leaf fescue, perennial ryegrass and bermudagrass. However, we see much more damage on *Poa annua* greens than other turf types used on greens. This is partly due to *Poa*'s genetic susceptibility to the disease. But the architecture of the poa plant also plays an important role (see Figure 8).

Note that the lower, older leaves of the poa plant are the first affected by this disease. See also how the newer leaves form directly above the older leaves (a result of the fact that the leaves are formed folded in the bud, rather than rolled in the bud as bentgrass is), thus completely shielding them from fungicide applications. For this reason, it is almost impossible to target an anthracnose infection on poa with contact fungicides. And because translocation to older or dying foliage is minimal, systemic fungicides are also not effectively delivered to them.

Figure 8. Note how the younger top leaves can shield the lower leaves from fungicide applications.

Factors that contribute to development of anthracnose

CONDITION	PREVENTIVE STRATEGY
Average air temps above 65F, high humidity	**Begin preventive fungicide programs once average air temperatures exceed 65F**
Previous anthracnose infestations	**Consider preventive fungicide programs**
Compaction	**Regular aerification & topdressing, but only when turf is healthy**
High soil salts	**Regular leaching**
Poor drainage/black layer	**Regular aerification & topdressing, but only when turf is healthy**
Tournament preparation (limiting water, double mowing and rolling)	**Apply preventive fungicides during tournament preparation**
Anthracnose resistance to certain fungicides	**Avoid dependence on a single class of fungicides;**

Increasing importance in promoting anthracnose

Weather: Of all of the factors that contribute to the development of anthracnose, none is more important than air temperature and humidity. Research by Danneberger et. al. (1984) show that when temperatures are warm and humidity is high, the forecast for anthracnose is very bad. But high moisture isn't completely necessary for development of serious symptoms. For example, at average air temperatures of 65F, anthracnose requires very little leaf moisture to develop, with symptoms developing 10 - 12 days later. And as temperatures go higher than 65F, the disease severity increases, even at relatively low moisture levels. For this reason, preventive fungicide programs that are triggered by average air temperatures above 65F are a good defense. One common misconception about anthracnose is that it can be "dried up" by decreasing watering times on the green. Unfortunately, the poa is more likely to die under these conditions than the disease. **This strategy is not recommended.** However, changes in irrigation scheduling (see "Management") may help reduce the severity of disease.

Compaction: Soil compaction appears to be strongly linked to the incidence of anthracnose -- especially the basal rot form of the disease. To fight compaction, regular cultivation is needed, as described below:

- Springtime aerification: At least once per year, aerify greens using 5/8" hollow core tines, removing the cores, and topdressing with 1/4" dry USGA specification root zone sand. Follow this with a deep (6-8"), 3/4" solid tine performed

through the sand. The sand can then be dragged, swept and/or blown to fill the holes.

- Monthly **"venting"** through the summer with small (1/4") solid tines can prevent sealing of the thatch surface, enhance water movement into the soil and improve gas exchange -- all without seriously disrupting the surface of the greens This process is essential to maintain healthy turf that is more resistant to traffic and disease.

Resistance: The fungicide azoxystrobin (Heritage), although not the best fungicide for anthracnose, normally does a good job of controlling the disease. However, during the summer of 2001, Heritage failed to control anthracnose in a few locations around the country. Preliminary data from both Cornell and Michigan State University indicates that the fungus has developed the ability to survive applications of Heritage and related products (Compass and Insignia). In other words, resistance is the likely explanation.

How does resistance affect anthracnose management? First of all, anthracnose resistance to Heritage and related products does not appear to be widespread at this point. There's a good chance that these products will still perform as they have in the past at your golf course. However, now that we know that resistance to anthracnose can apparently develop quite rapidly, these products are less reliable for anthracnose control. Other, more effective products for which no resistance has been observed can be substituted, as decribed below.

At the same time, Heritage, Compass and Insignia should continue to serve as important components in programs for other diseases such as summer patch, fairy ring and brown patch. These products are very effective against these summertime diseases, and if they are used carefully (by rotating their use with other types of fungicides), it may be possible to avoid development of resistance by these diseases.

Management: prevention, prevention, prevention

If you have suffered with anthracnose in the past, it is likely to rear its ugly face the following summer, unless the following preventive steps are taken.

Aerification programs (as described above) to improve water and gas exchange, and to decrease thatch, black layer and compaction.

Traffic management to avoid further compaction.

Scheduling irrigation in early morning hours to minimize the number of hours that free water is available for fungal growth.

Increasing mowing heights: even a 1/32" increase in height can result in dramatically healthier turf that can do a better job of staving off fungal infection.

Spoon feeding turf weekly with small amounts (0.1 lb/1000 square feet) of nitrate nitrogen (calcium nitrate or potassium nitrate, for example) will keep Poa healthy, and will avoid over fertilization with nitrogen

Preventive fungicide programs should be initiated once average air temperatures reach 65F for several days in a row. We have seen good results when contact fungicides (**chlorothalonil**: Daconil, Echo, Manicure or Spectro) are rotated every 14 days with systemic fungicides (**myclobutanil**: Eagle; or **propiconazole,** Banner). Please note however that propiconazole should not be used when air temperatures are above 80F. Products should be applied to target the foliage, at 2 gallons of spray solution/1000 sq ft or less, and should not be watered in. The use of flat fan nozzles will improve coverage and control. Avoiding irrigation for at least 4 hours post-application will also improve control.

The curative approach: a vicious cycle

Once anthracnose is diagnosed, it is likely that you will be playing catch-up with the disease for the remainder of the warm weather season. There are unfortunately no real cures, and the turf that is infected will almost certainly die. Any fungicides you apply will only save the healthy turf. When chlorothalonil is mixed with either thiophanate-methyl, myclobutanil or propiconazole, the healthy turf is protected well . Generally, at least 3 weekly applications are necessary before some type of control is regained. This combination of contact and systemic fungicides attacks the disease in two different ways -- from outside the plant, and from within the plant. Though effective, it is an expensive, fungicide-intensive program that is used only as a last resort.

While the turf is diseased, topdressing, which can abrade the turf as well as block sunlight from reaching the plant, should be avoided. Fertility and irrigation practices should be targeted at improving the health of the turf, rather than towards "starving" the pathogen.

References

Couch, H.B. 1995. Diseases of turfgrasses, 3rd edition. Krieger Publishing Co., FL. 421 pp.

Dannenberger, T.K., J.M. Vargas, A.L. Jones. 1984. A model for weather-based forecasting of anthracnose on annual bluegrass. Phytopathology, 74:448-451.

Vargas, J.M. 1994. Management of turfgrass diseases..Lewis Publishers, Boca Raton, FL. 294 pp.

Vargas, J.M., T.K. Danneberger and A.L. Jones. 1993. Effects of temperature, leaf wetness duaration and inoculum concentration on infection of annual bluegrass by *Colletotrichum graminicola*. Int. Turfgrass Soc. Res. Journal. 7:324-328.

Vincelli, P. 1997. Mixing fungicides controls disease on bentgrass greens. Golf Course Management. September, 1997.