

# Dealing With Bacterial Wilt on *Poa*

By Nathaniel Mitkowski

**W**ithin the past decade, bacterial wilt of annual bluegrass has become a pernicious problem in the Northeast. While it is not observed as frequently as anthracnose or dollar spot, the disease can usually be found at low levels on most golf courses with a significant level of annual bluegrass infestation.

The University of Rhode Island Turfgrass Disease Diagnostic Clinic has isolated the pathogen from golf courses in Connecticut, Massachusetts, Montana, New Hampshire, New York, Pennsylvania, Rhode Island and Canada. When the disease does become aggressive, it is extremely difficult to manage. Under optimal conditions, the disease will move through a stand of annual bluegrass, killing existing plants and infecting newly emerged seedlings throughout the course of the growing season.

Bacterial wilt of annual bluegrass was originally identified in the United States in 1985 in the lab of Dr. Joseph Vargas (Roberts *et al.* 1995). At the time, it was considered a strain of *Xanthomonas campestris*. The same lab also identified *Xanthomonas campestris* pv. *graminis* affecting only the creeping bentgrass cv. Toron- to in the early 1980s.

In 1995 a major revision of the genus *Xanthomonas* was published and reclassified those Xanthomonads that attack grasses as *Xanthomonas translucens*, a group of organisms that had been most previously revised under that name in the 1940s (Hagborg 1942).

The causal agent of bacterial wilt of annual bluegrass is *Xanthomonas translucens* pv. *poae* (Mitkowski *et al.*, 2005). Researchers in Japan have been studying the pathogen since the early 1990s and have made significant progress in understanding its biology and pathogenicity. As the name implies, its host range is restricted to the genus *Poa*. It will not attack any other grass genus and only goes to a few *Poa* species. While it is most aggressive on the annual form of *Poa annua*, it can also kill *Poa trivialis*. It has been observed multiplying in the leaf tips of *Poa compressa* and *Poa pratensis* but has not been observed to cause disease on either of these species.

Its specificity for *Poa annua* led some researchers to investigate its use as a *Poa annua* bio-

control. Unfortunately the pathogen is extremely difficult to artificially inoculate, requiring extremely large numbers of bacteria to initiate a successful infection, thus limiting its potential as a biological control.

*Xanthomonas translucens* pv. *poae* grows optimally at about 30 degrees Celsius, but it is observed from early April until October in the Northeast. It can be active anytime infected plants are not dormant. It is often seen in the spring following severe winter damage. Although the reason for this is unknown, it is likely that large amounts of decaying tissue, caused by winterkill, provide an excellent nutrient source for the pathogen.

Eventually populations of the bacteria living epiphytically or saprobially will reach the minimum threshold necessary to incite disease. Infection occurs and plants begin to wilt and die. It is also likely that the stress of severe winter damage predisposes many plants to the pathogen. Bacterial wilt of annual bluegrass has also often been seen in conjunction with cool season *Pythium* in the spring. It is unclear what relationship may exist between these two organ-

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*The two containers to the left feature greenhouse-grown *Poa annua* plants inoculated with bacterial wilt of annual bluegrass. The pair of containers on the right are uninoculated control plants for comparison.*



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isms or if the association is simply coincidental.

After the spring flush of disease, plants tend to rebound until July or August. Temperatures are approaching the bacterial optimum in these months and disease predictably resurges. Bacterial wilt resurgence usually coincides with anthracnose resurgence, devastating affected *Poa annua* greens. As temperatures decline in the fall, disease usually recedes. However, warm fall days can cause additional outbreaks.

Symptoms of the disease begin as small yellow spots. Often etiolation (bleaching caused by failure to photosynthesize) will occur, although this symptom is much more pronounced if plants are not mown for two or three days. In the diagnostic lab, etiolated plants are commonly observed after a day of incubation and produce significant amounts of bacterial streaming.

Following etiolation, plants wilt, collapse and ultimately die. Sometimes, leaf-tip necrosis can be observed. Although the time course of disease is variable based on temperature, strain virulence and presumably genotype resistance, infected plants usually begin to show symptoms within two weeks of inoculation in the greenhouse. Greenhouse experiments using very virulent isolates at extremely high inoculation levels have produced symptoms in as little as four days

(Mitkowski, unpublished data). Once symptoms are observed, plants begin to die rapidly.

Bacterial wilt has been observed on fairways sporadically. Fairway symptoms, however, tend to be far milder than symptoms on putting greens. The most common fairway symptom is profuse etiolation. Frequently superintendents will notice that a sizable portion of plants on an

## The only truly effective management strategy is to eliminate annual bluegrass.

affected fairway produce etiolated shoots, growing as fast as an inch per day. Plants appear to be otherwise healthy but require frequent mowing to keep the stand from appearing ragged. We have isolated both *X. t. pv. poae* and *X. t. pv. graminis* from affected fairways. *X. t. pv. graminis* has very low virulence on *Poa annua* and we have never isolated it from putting greens.

Virulence within *X. t. pv. poae* appears to be variable. Research from both American and Japanese isolates suggests that some strains of the pathogen are not very aggressive and produce minimal damage. Other isolates or strains are extremely aggressive and can quickly kill entire stands of *Poa annua*. This provides a likely explanation as to why some superintendents have such difficulty managing the disease while others experience little noticeable damage.

Similar to anthracnose, bacterial wilt favors plants that are severely stressed by low heights of cut, compaction, under-fertilization and excessive wear. Shaded plants and those found in low-lying areas are also more prone to the disease.

## Control options

Remediating cultural problems can mitigate bacterial wilt but once it has taken hold on a putting green, it is very difficult to eliminate. Chemical control options are virtually nonexistent. Because the pathogen is bacterial in nature, fungicides have no effect. Antibiotics that target bacteria are not available for use on turfgrass.

There are anecdotal reports of superintendents experimenting with an oxytetracycline antibiotic, but the chemical can be very phytotoxic and most of these reports indicate that more harm was done than good. Additionally, the use of such products on turf is contrary to its labeling and ille-

**TABLE 1**

### Host range of the M-1 isolate of *Xanthomonas translucens* pv. *poae*, isolated from Torrington Country Club in Torrington, Conn., 2001.

Host Species	Host Reaction*
<i>Poa annua</i> var. <i>annua</i>	+
<i>Poa annua</i> var. <i>reptans</i>	- (+)
<i>Agrostis palustris</i> cv. Providence	-
<i>Agrostis capillaris</i> cv. Exeter	-
<i>Poa pratensis</i> cv. Lofts 1757	-
<i>Festuca rubra</i>	-
<i>Lolium perenne</i> cv. Night Hawk	-
<i>Poa nemoralis</i>	-
<i>Poa attenuata</i>	+
<i>Poa compressa</i> cv. 95-29	-
<i>Poa compressa</i> cv. Harmony	-
<i>Poa trivialis</i> cv. Sabre	- (+)

\* "+" = positive host, "-" = negative host, "- (+)" indicates no disease observed but limited bacterial reproduction occurred in leaf tips at inoculum level of ~108 cfu/ml.

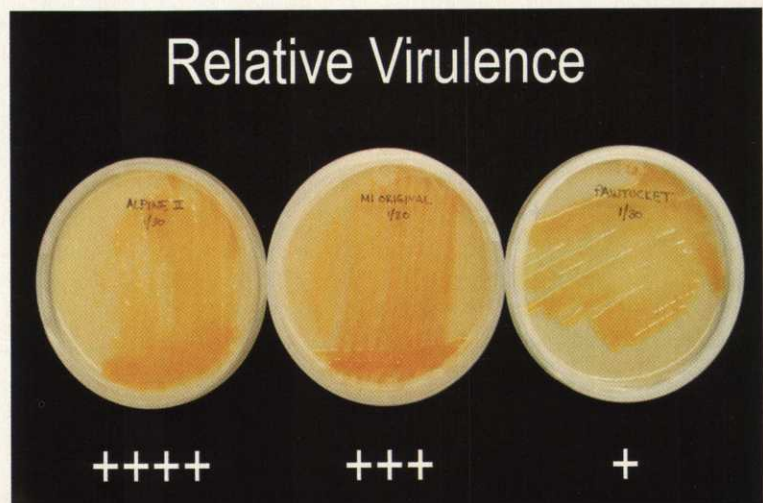


To the left, growth of chamber-grown *Poa annua* plants inoculated with bacterial wilt of annual bluegrass. In the container to the right are uninoculated control plants for comparison.

gal. The product is only labeled for peaches, pears, nectarines and with a number of state-specific Section 18 registrations for use on apple. General biocides can be used against bacterial wilt. Kocide and Junction (copper hydroxide and mancozeb, respectively) are often mentioned for controlling the pathogen and can reduce bacterial population on leaf surfaces. However, these chemicals do not penetrate leaves (and can be very phytotoxic), so only bacteria on the surface of the leaf or in the soil will be affected.

Bacterial wilt enters plants through wounds, most commonly through mower wounds. Although an application of a general biocide may have some effect, new wounds will be produced as soon as the stand is next mowed. New wounds will be unprotected by applications of Kocide or Junction made previous to the most recent mowing.

While additional applications of these chemicals might seem prudent following every mowing, severe phytotoxicity would surely result long before any meaningful bacterial control was achieved. The frequency with which these chemicals must theoretically be applied to produce reliable control makes them a less than optimal solution to disease. Zeritol has been considered as a potential management option for bacterial wilt. As a contact biocide, however, it is unlikely to provide any more significant control than either Kocide or Junction. Its lack of residual activity may even provide less con-



Three isolates of *Xanthomonas translucens* pv. *poae*, all with a different level of disease virulence, as indicated by the number of plus signs.

trol when applied on a 7-10 day schedule.

During the 2004 field season, URI fungicide trials demonstrated that Zeritol provided no control of dollar spot at the 12 fluid ounces per 1,000 square feet rate and minimal control at the 6 fluid ounces per 1,000 square feet rate every 14 days. However, at low rates it is less likely to cause phytotoxicity than Kocide or Junction and might therefore be applied following every mowing.

This strategy has yet to be tested but has some merit. The time and effort required to

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*Streaming of Xanthomonas translucens*  
*pv. poae* from the cut leaf of an infected  
*Poa annua* plant.



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spray all effected putting greens following every mowing, however, may be prohibitive.

Bacterial wilt of annual bluegrass is certainly not a new pathogen. It has a worldwide distribution and is likely to be found anywhere that annual bluegrass exists. The most important question regarding this pathogen is not even how to control it.

The only truly effective management strategy is to eliminate annual bluegrass.

While guaranteed to eliminate the pathogen, this technique is far easier said than done. What is most intriguing about bacterial wilt of annual bluegrass is why has it become so prevalent in the past decade. It is possible that it may have always been prevalent but simply overlooked or misdiagnosed as some other fungal pathogen?

The more likely reason for its prevalence, however, is an almost universal shift towards management techniques that favor annual bluegrass while consequently discouraging creeping bentgrass. Until this issue is addressed (and other high intensity management practices related to increasing ball rolling speed), it is probable that bacterial wilt

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of annual bluegrass will continue to pose an intractable threat to golf course putting greens.

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