Identification of Parasitic Bacteria as Biological Control Agents Against Summer Patch Disease

Dr. Donald Kobayashi

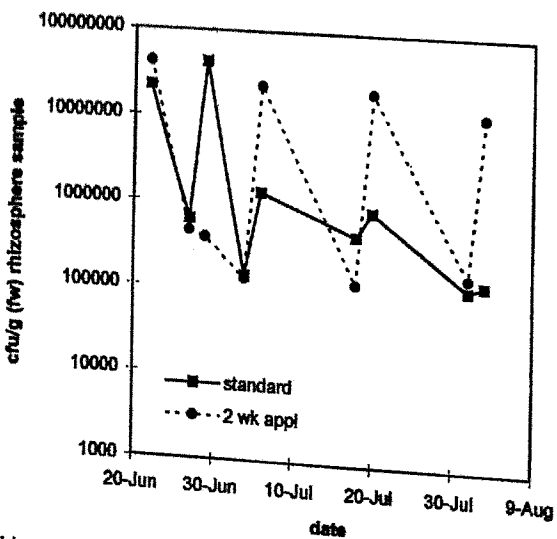
Rutgers University

Goals:

- *Isolate and identify bacteria which can colonize and parasitize the "mycelia" of Magnaporthe poae, the causal agent of summer patch disease.*
- *Screen isolated bacteria for disease control potential using controlled growth chamber and field studies.*

Cooperator:

Dr. Bruce Clarke



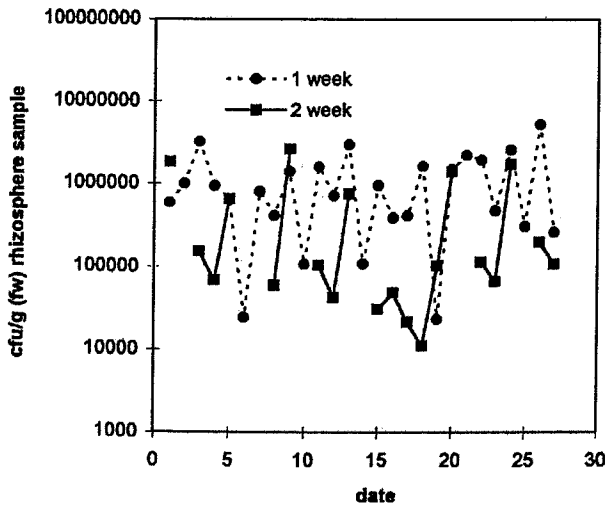
Rhizosphere populations of *Xanthomonas maltophilia* 34S1 repeatedly applied to Kentucky bluegrass var. Baron. *X. Maltophilia* 34S1 (Xm34S1) populations applied on weeks 2 and 3 after seeding were compared to populations of Xm34S1 in the rhizosphere of Kentucky bluegrass treated every two weeks.

A fungal trapping method and an enrichment culture method were used to isolate several hundred bacteria from turf and soil sources. These isolates were screened in a greenhouse/growth chamber assay for the suppression of summer patch disease, caused by *Magnaporthe poae,* on Kentucky bluegrass. At least eight bacterial isolates were identified that were capable of consistently suppressing summer patch symptom development by greater than 50% compared to fungal-inoculated control plants. All eight isolates expressed activity of one or more of the extracellular enzymes chitinase, glucanase, protease and lipase. In addition, all eight isolates were capable of colonizing and persisting in the rhizosphere of Kentucky bluegrass at significant populations.

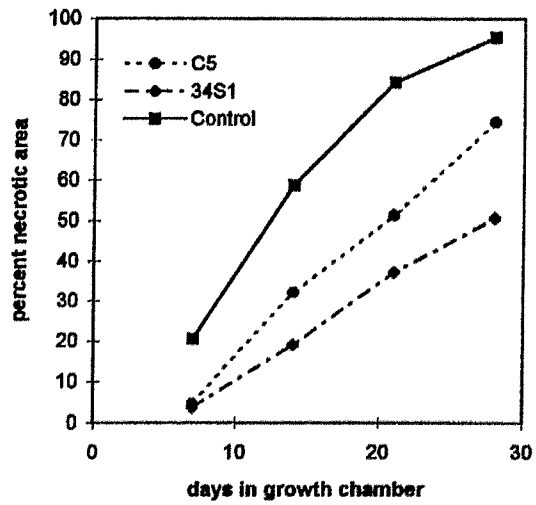
Two bacterial strains, *Xanthomonas maltophilia* 34S 1 (Xm34S 1) *and Serratia marcescens* 9M5, were capable of suppressing summer patch by greater than 70% and 50%, respectively, compared to disease in untreated control plants over a 3 week period. The rates at which disease progressed in plants treated with bacteria were not different compared to untreated plants; however, disease onset was significantly delayed in bacteria-treated plants. These results were interpreted to reflect the activity of antagonistic bacteria in reducing pathogen colonization of turfgrass roots. Disease onset was delayed for an extended period when Xm34S1 was applied to plants on a repeated schedule. Rhizosphere populations of Xm34Sl indicated that disease suppression was associated with populations between $> 10^5$

and > 10^7cfu/g rhizosphere sample.

Xm34S1 was applied to pathogen-inoculated field plots in 1994 and 1995. Summer patch suppression was not observed in field trials in either year. High disease pressures and insufficient population establishment of Xm34S1 were attributed to the lack of performance in the field. Extensive field population studies in 1995 indicated that populations of Xm34S I were maintained above 10^5 cfu/g rhizosphere sample throughout the entire summer season, but did not reach populations above values of 10^7 cfu/g rhizosphere sample. These populations were clearly 10-fold lower than the critical population values established in growth chamber studies.



Rhizosphere populations of *Xanthomonas maltophilia* 34S1 in field plots of Kentucky bluegrass in 1995. Populations of weekly applications (1 week) were compared to applications every two weeks (2 week).



Summer patch suppression by *Xanthomonas maltophilia* 34S1 and the mutant C5 deficient in chitinase production on Kentucky bluegrass var. Baron. Plants were treated with the wildtype *X. maltophilia* 34S1 in standard growth chamber assays, and compared with the *chi* mutant C5 and untreated disease control plants in growth chamber assays.