Plant Pathology Seminar Series

"Annual Ryegrass Toxicity: Corynetoxin Poisoning of Livestock Due to Toxin Production by *Rathayibacter toxicus* "

Kathleen McKeever

Abstract

Annual Ryegrass *(Lolium rigidum)* is an important pasture species in the high rainfall zones of Australia (6). This grass is also a significant weed in valuable, high rotation crops such as wheat and oats (6). Seed gall-forming nematodes in the genus *Anguina* vector a soil-borne coryneform bacterium, *Rathayibacter toxicus*, into the galled seedheads of annual ryegrass (3,6). As the plant senesces in early summer, the bacteria begin production of a toxic yellow gummosis that fills the galls and coats the seedheads (11). Bacterial toxin production by *R. toxicus* in grasses has become known as Annual Ryegrass Toxicity (ARGT). The toxins produced in the gummosis are glycolipid compounds belonging to a group of tunicamycin antibiotics (3). These toxins are collectively referred to as corynetoxins and have structural similarity to the tunicamycin toxins produced by members of the *Streptomyces* (3). Toxin production by *R. toxicus* is dependant on the maintenance of a phage-carrier state with a lytic bacteriophage that encodes for proteins integral to the toxin synthesis pathway (8). The potential hazard posed to animal and human health by production of this toxin has lead to the inclusion of *R. toxicus* on the United States Select Agent List as outlined by the Bioterrorism and Protection Act of 2002.

Livestock that ingest toxin-contaminated ryegrass suffer neurological poisoning and subsequent mortality. Breakdown of neurological and cellular processes is attributed to the malfunction of the N-acetylglucosamine-1-phosphotransferase enzyme leading to a decline in protein glycosylation (4). Glycoproteins have many important roles in the cell such as proper protein folding, secretion of proteins from the endoplasmic reticulum, and localization of proteins to other cellular organelles (3). Breakdown of protein glycosylation in the brain results in neuron death and loss of proper brain function (2). Symptoms observed in animals suffering from corynetoxin poisoning include loss of coordination, seizures, involuntary muscle tremors, and death (3).

There are many options for mitigation of toxic ryegrass poisoning. Control options targeting ryegrass include removal of seedheads, field burning, and herbicide use. After years of continued herbicide use in Australia, several glyphosate resistant populations of ryegrass have occurred, leading to a realization for the need of integrated weed management (9). Studies done by McKay et al. have demonstrated the use of nematicides and nematode-parasitizing fungi in the genus *Verticillium* to be largely unsuccessful as a control option for reduction of *Anguina* spp. (7). Toxin-binding agents such as cyclodextrins have been tested as a prophylactic control for protection of livestock, and have proven successful (5). Finally, control options for reduction of the bacterium have included the use of antagonistic non-toxigenic *Clavibacter* species and biocontrol using the "Twist" fungus, *Dilophospora alopecuri* (1,10). Many field studies using the Twist fungus have proven successful and this biocontrol option has led to a decline in the incidence of ARGT in current years (1).

4:10 PM • Monday, November 2, 2009, • Johnson Hall 343

Plant Pathology 515, Fall, 2009

References:

1.) Barbetti, M.J. and I. T. Riley. (2006). Field application of *Dilophospora alopecuri* to manage annual ryegrass toxicity caused by *Rathayibacter toxicus*. *Plant Disease* 90: 229-232.

2.) Chang, J.Y. and V.V. Korolev. (1996). Specific toxicity of tunicamycin in induction of programmed cell death of sympathetic neurons. *Experimental Neurology* 137: 201-211.

3.) Finnie, J.W. (2006). Review of corynetoxins poisoning of livestock, a neurological disorder produced by a nematode-bacterium complex. *Australian Veterinary Journal* 84(8): 271-277.

4.) Jago, M.V., A.L. Payne, J.E. Peterson, T.J. Bagust. (1983). Inhibition of glycosylation by Corynetoxin, the causative agent of annual ryegrass toxicity: a comparison with tunicamycin. *Chemical-Biological Interactions* 45: 223-234.

5.) May, C. and P.L. Stewart. (1998). Development of a toxin-binding agent as treatment for tunicaminyluracil toxicity: protection against tunicamycin poisoning of sheep. *Australian Veterinary Journal* 76: 752-756.

6.) McKay, A.C. and K.M. Ophel. (1993). Toxigenic *Clavibacter/Anguina* associations infecting grass seedheads. *Annual Review Phytopathology* 31: 151-167.

7.) McKay, A.C., J.M. Fisher, A.J. Dube. (1982). Control of the nematode associated with annual ryegrass toxicity. *Australian Journal of Agricultural Research* 34: 403-413.

8.) Ophel, K.M., A.F. Bird, A. Kerr. (1993). Association of bacteriophage particles with toxin production by *Clavibacter toxicus*, the causal agent of annual ryegrass toxicity. *Phytopathology* 83: 676-681.

9.) Riley, I.T. and G.S. Gill. (1994). Relationship between herbicide resistance in *Lolium rigidum* and populations of *Anguina funesta*, the nematode vector in annual ryegrass toxicity. *Australian Journal of Experimental Agriculture* 34: 633-35.

10.) Riley, I.T. and G.Yan. (1999). Evaluation of non-toxigenic, nematode-vectored *Clavibacter* spp. for potential biocontrol of *Clavibacter toxicus*, the bacterium responsible for annual ryegrass toxicity. *Australian Association of Nematologists Newsletter* 10(2).

<nematologists.org.au/July99/clavibacter.html>

11.) Stynes, B.A. and A.F. Bird. (1983). Development of annual ryegrass toxicity. *Australian Journal of Agricultural Research* 34: 653-660.