A Diagnostic Tool for Fescue Toxicosis

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Fescue toxicosis is the result of grazing endophyte (*Neotyphodium coenophialum*) infected tall fescue (*Festuca arundinacea*). Most tall fescue pastures are endophyte-infected (E+) and this results in the presence of ergot alkaloids in the forage. As a result of these alkaloids in E+, rate of gain and calving rates are decreased (Stuedemann and Hoveland, 1988). Individual animals may express either peripheral necrosis ("fescue foot") or fat necrosis as variants of the toxicosis. Affected animals grazing E+ forage during the summer months particularly, appear stressed with increased respiratory rates, spend excessive time in a shaded or water cooled environment, and have rough hair coats.

Previously documentation of the toxicosis involved examination of the forage for either the presence of the endophyte or analysis of the forage for ergot alkaloids. Characteristically, cattle grazing E+ forage have reduced serum prolactin (Thompson *et al.*, 1987); another entity used diagnostically. However, there are caveats associated with using these variables. First, casual retrieval of pasture samples may provide misleading tiller infection rates and alkaloid concentration. Therefore, in order to appropriately survey a pasture sufficient tiller forage from the entire pasture must be obtained. Of course, this has a significant labor component. Microscopic examination of the tillers for the presence of the endophyte also is time consuming and few laboratories offer analysis of forage for ergot alkaloids. These analyses, however, do not indicate what cattle may have consumed in the pastures and consequently may be misleading.

It is important to recognize that serum prolactin concentrations vary considerably with enviromental daylight length (Peters and Tucker, 1978). Therefore, in order to use serum prolactin then blood samples should be obtained from chronologically matched controls (not grazing E+). Additionally, because gender, age and lactational status affect serum prolactin, there should also be appropriate controls for these variables. Further, serum prolactin is increased by stress so crowding conditions associated with obtaining blood may falsely elevate serum prolactin.

To find a better physiological maker, an experiment was designed to evaluate urinary ergot alkaloids as an indicator of fescue toxicosis. Yearling Angus steers (16) were placed on both E+ and E- pastures. Four steers were assigned to each of the following groups: 1. steers remained on E+ pastures, 2. steers remained on E- pastures, 3. steers grazing E+ pastures were switched to E- pastures and 4. steers grazing E- pastures were switched to E+ pastures.

The steers had grazed their respective pastures since April 9, 1996. After being held off water for 16h, urine was collected via a cup mounted on a pole while the steers were in a holding pen on October 23. Then the appropriate steers were switched from E+ to E- or the reverse. Subsequent urine was collected on days 2, 5, and 7 after switching. This process was again repeated begining on October 30. Total ergot alkaloids were quantitated using a competitive ELISA. The urine was normalized for creatinine and ergot alkaloids are expressed as mg/day in the urine.

Urinary Ergot alkaloids (mg/d) in steers grazing endophyte-infected (E+) and endophyte-free (E-) fescue

Treatment Group	Endophyte	Day	Mean	SD
1	++	0	0.63	0.28
	++	2	0.73	0.34
	++	5	0.63	0.31
	++	7	0.79	0.33
2	—	0	0.06	0.06
	—	2	0.02	0.02
	—	5	0.02	0.03
	—	7	0.02	0.01
3	+-	0	0.75	0.36
	+-	2	0.04	0.04
	+-	5	0.03	0.03
	+-	7	0.04	0.02
4	-+	0	0.03	0.04
	-+	2	0.79	0.83
	-+	5	0.73	0.37
	-+	7	0.68	0.31

n=8 observations/d/trt,
LSD=0.39

The excretion of ergot alkaloids was increased ($P<.05$) in steers grazing E+ compared to steers grazing E-. However, there was no difference in the urinary ergot alkaloid excretion at two or more days after switching in steers that had been switched from E+ to E- compared to those that were maintained on E-. Similarly, there was no difference in this parameter at two or more days after switching from E- to E + compared to those that were maintained on E+. Therefore, in conclusion, urinary excretion of ergot alkaloids was clearly detected in steers grazing E+ forage for two days and has merit in diagnosing fescue toxicosis.

References

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Abstract

Epidemiology of bovine spongiform encephalopathy in Northern Ireland 1988 to 1995

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The epidemiology of bovine spongiform encephalopathy (BSE) in Northern Ireland from 1988, when it was first confirmed, to the end of 1995 is described. All cases of BSE were subjected to a detailed epidemiological investigation, complemented by data from the national animal health records on every bovine animal. Data are presented on 1680 cases. Many of the epidemiological features of the disease were similar to those reported in Great Britain, but the incidence of Northern Ireland was approximately one-tenth that in Great Britain. The epidemic increased to a peak of 56 cases per month in January 1994, and decreased to nine cases in December 1995. Statutory intervention banning the use of meat and bone meal in ruminant feed in January 1989 has produced a marked and continuing reduction in the incidence. The majority of the cases were in Northern Ireland cattle, but 83 cases were imported from Great Britain and five from the Republic of Ireland. Many of the key epidemiological features have remained constant throughout the epidemic: the greater incidence of BSE in dairy herds than in beef suckler herds, the low within-herd incidence, the variation in incidence with herd size, the breed distribution, the distribution of the reported clinical signs and the proportion of purchased cases. Although the source of the BSE epidemic in Northern Ireland has not been established conclusively, the evidence suggests that the importation of meat and bone meal and protein concentrates from Great Britain may have been responsible.