Tall Fescue Summer Toxicosis in Cattle[1,2]

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Summary

Cattle grazing tall fescue at times display signs of various toxicity syndromes. Among these conditions are fescue foot, fat necrosis, and fescue summer toxicosis. Fescue foot is a condition which ranges from mild lameness to severe gangrenous lesions of the feet and tail and occurs primarily during cold winter weather. Clinical cases of fat necrosis most often leads to digestive disturbances and reproductive problems in clinical cases. Fescue summer toxicosis is characterized by poor performance and a decreased ability to dissipate body heat on hot summer days. With respect to the summer syndrome, research has not led to specific drug therapy. The most effective treatment remains removal of animals from the inciting pasture. Prudent control is best obtained by effective pasture management including the grazing of mixed swards of fescue and legumes and establishing pastures with varieties shown to be associated with few toxicity problems or by the use of fungus-free seed.

Introduction

Tall fescue *(Festuca arundinacea* Schreb.) is one of the most abundant forage crops in the United States and is an important cool season perennial forage for beef cattle. Estimates of U.S. land area in pure or mixed stands of tall fescue range from 7 to over 14 million hectares (1-4). Advantages of tall fescue to animal producers are that it is a widely adapted perennial, is seed-propagated, will withstand trampling, grows well in association with legumes, and can be stockpiled for fall and winter grazing (5-7). Disadvantages are low intake and palatability problems (5) which limit its use for lactating dairy cows and rapidly growing replacement or stocker cattle. In addition, at least three distinct toxicity syndromes have been associated with the feeding of tall fescue. The etiology of these syndromes may or may not be related. The most familiar is that of fescue foot, a lesser known problem manifests itself as fat necrosis or lipomatosis, and perhaps the most insidious of the three is fescue summer toxicosis or summer slump.

Fescue foot occurs primarily during cold weather and is marked by swelling of the hind limbs and dry gangrenous lesions of the extremities (8-12). Initial clinical signs may be no more than mild lameness, reduced gain or loss of weight, rough hair coat, and arching of the back. Severe cases can result in emaciation and sloughing of hooves (8-12).

Bovine fat necrosis has been observed in many countries (13) and was first reported as a herd problem in the United States by Williams *et al.* (14). The occurrence of fat necrosis is associated with grazing of heavily nitrogen fertilized tall fescue (15-16). The most frequent clinical sign is digestive disturbance due to intestinal constriction (12). Dystocia may also occur (17,18) as well as impaired heart and kidney function (14,17). Limited evidence suggests that the lesions will regress following removal of cattle from the inciting pasture (14).

Tall fescue summer toxicosis is a more subtle disease but as such may be the most important in terms of adverse economic impact (19). Current knowledge regarding this syndrome, including our recent work at Beltsville, and the prospects for treatment and control are presented below.

Clinical Signs and Physiological Aspects

Tall fescue summer toxicosis occurs primarily during hot summer weather (20) and is characterized primarily by poor animal performance (21-28). Nutrient composition and digestibility of tall fescue was shown to be comparable to other grasses (22,29-33) and when not overly mature can be highly digestible (29,31-33). However, dry matter intake of tall fescue pasture is often low (22,25,27-29). This leads to reduced rate of gain (22,27) or even loss of weight (28,34-35). In the case of lactating cows, milk production is also decreased (25). Steers grazing experimental toxic varieties of fescue have been observed to spend significantly more time standing and lying in shade and less total time grazing compared to steers on adjacent less toxic tall fescue pasture (24,36).

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Acute phases of tall fescue summer toxicosis are characterized by hypethermia evidenced by increased rectal temperature, increased respiration rate, and excessive salivation (22,24-25,27-28,36-37). Animals suffering the acute disease are often seen standing or wallowing in water or mud (36, figure 1). Recent research at several locations has documented the interaction between high environmental temperature and onset of clinical signs (27-28,37-40). This suggests an impairment of the affected animal's ability to regulate body temperature, i.e., the inability to dissipate body heat under environmentally hot and humid conditions (41).

Figure 1. Calves suffering acute tall fescue summer toxicosis. Note that the affect animals are standing in water, unlike unaffected cattle in the adjacent pasture.

Certain similarities between tall fescue summer toxicosis and ergotism (42-43) as well as decreased milk production in dairy cows associated with tall fescue summer toxicosis (25) led Hurley et al. (40) to suspect that prolactin secretion might be reduced as a result of the disease. These workers showed significantly lower concentrations of circulating prolactin in calves fed freshly cut forage of a known toxic experimental variety of fescue (G1-307) compared to fresh forage of a less toxic experimental variety (G1-306). The toxic variety also inhibited the increase in circulating concentrations of prolactin that usually occurs in animals subjected to increased environmental temperature or injection of thyrotropin-releasing hormone. We have confirmed the effect of G1-307 on reduced prolactin concentrations in both grazing cattle and cattle fed hay (Bolt, D. J., Bond, J. and Hammond, A. C., unpublished data). Our work would also indicate that the reduced prolactin concentrations are relatively immediate (effects seen as early as 24 hours) following ingestion of the toxic forage and that the concentrations rapidly return (within a few days) toward normal following removal of the toxic forage from the diet. Sheep grazing G1-307 at Beltsville have also shown decreased secretion of prolactin. The work with sheep has further shown that conception may be delayed (45) or that there may be an increase in embryonic mortality (46) in ewes grazing this toxic fescue variety.

Reproductive problems in cattle associated with tall fescue summer toxicosis independent of effects associated with fat necrosis (17-18) have not been documented experimentally but casual observations indicate that problems may occur (1,4). In one group of 10 heifers grazing toxic fescue (G1-307), we observed an increase in small or nonfunctional ovaries (60% vs. 25%) compared to 12 heifers grazing nontoxic fescue (Bond, J. and Hammond, A. C., unpublished).

Etiogenic Agents

Both plant alkaloids and fungal infestations of tall fescue have been implicated in the etiology of tall fescue summer toxicosis. Each might be involved in an interrelated manner because fungi can effect the alkaloid content of tall fescue (47). Several attempts have been made to isolate specific etiogenic agents. An ethanolic extract and an anionic fraction of tall fescue have been shown to decrease voluntary intake of calves (48). These fractions were also shown to produce fescue foot (49). In a controlled experiment designed to simulate the elevated environmental temperature and humidity conditions under which tall fescue summer toxicosis occurs, a cationic fraction of tall fescue decreased feed and water intake, caused profuse salivation and resulted in higher morning rectal temperatures in calves. In contrast, calves in this experiment which were administered an anionic preparation increased feed and water intake (38).

Several alkaloids have been identified in tall fescue (12,19,50-52). One of the predominant alkaloids that interested researches in the field was perloline (53). The concentration of perloline was shown to vary among tall fescue varieties and with maturity, amount of nitrogen fertilization and season of the year (50). Perloline was shown by Bush et al. to decrease in vitro cellulose digestion (54-56) and volatile fatty acid production (55-56). This led to their suggestion that perloline may be involved in the poor performance of cattle grazing tall fescue in summer (20,54). University and USDA researchers at Kentucky develped experimental varieties of fescue that varied in perloline content (24-25,53,57). Animal performance data on dairy cows reported by Hemken *et al.* (25) revealed that poorest performance was obtained with fescue that contained the lowest amount of perloline. This reduced performance was further shown not be related to digestibility of the forage. Cows that received variety G1-307 (low perloline) had elevated rectal temperatures, increased respiration rates and a decrease in body weight, which are all typical signs of fescue summer toxicosis. An experiment with yearing steers (24) and work conducted here at Beltsville (36) gave similar results. These experiments suggested that perloline was not the toxic agent. Other analysis showed that the poor animal performance was related to the pyrrolizidine alkaloids, N-

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Table 1. Effect of vasoactive drugs on mean (± SEM) rectal temperature and respiration rate in heifers grazing two varieties of tall fescue.

Tall fescue variety	Treatment	Dose (mg)	n	Pretreatment Rectal Temp. (°F.)	Pretreatment Respir. Rate (resp./min.)	Posttreatment Rectal Temp. (°F.)	Posttreatment Respir. Rate (resp./min.)
G1-307	Dibenzyline	30	2	102.3 ± 0.6	71 ± 1	104.0 ± 0.7	88 ± 1
G1-307	Vasodilan	60	2	103.6 ± 0.4	59 ± 12	106.7 ± 0.7	91 ± 9
G1-307	Control	-	2	102.7 ± 0.1	60 ± 8	104.3 ± 0.2	92 ± 8
Kenhy	Vasoxyl	60	2	102.2 ± 0.2	68 ± 6	102.0 ± 0.4	70 ± 7
Kenhy	Control	-	5	103.7 ± 0.4	82 ± 5	103.2 ± 0.4	84 ± 10

acetyl loline and N-formyl loline (28,53). Jackson *et al.* (28) fed fescue hay or seed that contained a known amount of N-acetyl plus N-formyl loline alkaloids to steers under environmentally controlled and elevated temperature (30,31° C). The higher loline treatments resulted in loss of body weight and significantly lower intake, higher rectal temperature, and increased respiration rate. In addition, they reported that the levels of these alkaloids were found to be related to infestation by an endophytic fungus, *Epichloe typhina.*

Several studies have centered on the possible role of mycotoxins in fescue toxicosis, noteably *Fusarium* toxins (10,58-60). Most recent interest, however, has been with *Epichloe typhina,* which appears to be present in the grass when outbreaks of fescue summer toxicosis occur (26,39,61). Alabama workers have reported that this fungus may actually be a new species of *Acremonium* (37) and not *Epichloe typhina* as reported by Bacon *et al.* (61). However, to resolve the actual relationship between fungal infestations, specific alkaloid content, and the induction of fescue summer toxicosis, further research will be required.

Effect of Vasoactive Drugs On Tall Fescue Summer Toxicosis

It could be hypothesized that the inability of cattle to dissipate excess body heat while grazing toxic tall fescue is due to peripheral vasoconstriction. This would provide some commonality between fescue summer toxicosis and fescue foot. Whittow (41) studied the role of decreased blood flow to the extremities in cattle as a means of thermoregulation which supported the suggestion of vasoconstriction as a possible cause of fescue foot (9,10). In warm summer months peripheral vasoconstriction may reduce the ability to dissipate body heat and cause animals to go off feed, thereby reducing consumption of toxic components and preventing further progress of the disease. In cold winter months where dissipation of body heat is not a problem, toxicosis may progress as animals continue to ingest toxic forage. This would permit the winter condition of fescue foot to develop in absence of most of the signs exhibited during the summer. If this hypothesis were true, treatment via drug therapy to combat the vasoconstriction might be effective against the summer syndrome. Therefore, we determined the effects of three vasoactive drugs on rectal temperature and respiration rate in cattle under grazing conditions previously shown to elicit tall fescue summer toxicosis.

Materials and Methods

Angus heifers (mean ± SEM body weight 262 ± 2 kg) were placed on 0.5 hectare experimental pasture lots seeded to Kenhy (nontoxic) or G1-307 (toxic) tall fescue. Water and trace-mineralized salt were provided *ad libitum.* Animals were placed on pasture mid-May and experiments were conducted mid-July. Forage stands in the lots were over 70% pure with respect to the varieties seeded. Pastures were fertilized with ammonia nitrate in April and June at the rate of 80 kg N/hectare. Stocking rate was approximately 16 animals/hectare throughout the experimental period. A portable corral and squeeze chute were erected near the experimental pastures for handling of animals during administration of treatments and collection of data.

On July 16, 1980 an experiment was conducted where six heifers on G1-307 and seven heifers on Kenhy were used to check dosages and possible treatment effects of two vasodilators, Dibenzyline[5] and Vasodilan[6], and one vasoconstrictor, Vasoxyl[7], on the hyperthermia associated with fescue summer toxicosis (table 1). The purpose of testing vasodilators with animals on toxic fescue was to attempt reduction of elevated body temperature. The

[5] *Phenoxybenzamine hydrochloride, Smith Kline & French Laboratories, Philadelphia, PA 19001.*

[6] *Isoxsuprine hydrochloride, Mead Johnson Pharmaceutical Division, Mead Johnson & Company, Evansville, IN 47721.*

[7] *Methoxamine hydrochloride, Burroughs Wellcome Co., Research Triangle Park, NC 27709.*

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purpose of testing a vasoconstrictor on nontoxic pasture was to attempt induction of elevated body temperature which may reproduce signs of fescue summer toxicosis. The animals were treated by i.m. injection between 1030 and 1130 hours and baseline rectal temperatures and respiration rates recorded. At 1300-1400 hours (ambient temperatures 97.5° F.) rectal temperatures and respiration rates were again recorded.

On July 18, 1980, seven Angus heifers on G1-307 received 60 mg Dibenzyline (administered i.m. in a saline slurry), six heifers on G1-307 received no treatment, and seven heifers on Kenhy received no treatment. Rectal temperatures, heart rates, and respiration rates were recorded at the time of Dibenzyline administration (1030-1200 hours) and approximately two hours later (1300-1400 hours). Ambient temperatures at the morning and afternoon times were 90.0 and 94.0° F., respectively.

Results and Discussion

Results of the first experiment are shown in table 1. All rectal temperatures and respiration rates of heifers on G1-307 were higher in the afternoon than in the morning. These clinical signs are typical of fescue summer toxicosis. No treatment effect of either Dibenzyline or Vasodilan on alleviating the condition was observed. No trend toward change of rectal temperature or respiration rate was observed with heifers or Kenhy. Vasoxyl did not increase rectal temperatures or respiration rates in animals on Kenhy pasture. Results of the second experiment are shown in table 2. All rectal temperatures, respiration rates and heart rates were higher in the afternoon than in the morning. No treatment or pasture effects on these parameters were observed. Although body temperatures and respiration and heart rates were higher in the afternoon, no signs of hyperthermia were observed.

The ability of vasoactive drugs to increase dissipation of body heat by increasing peripheral blood flow was not demonstrated in these experiments. Whether peripheral blood flow was actually altered could not be determined from the present data. Confounding the results was the lack of frank fescue summer toxicosis in animals grazing G1-307 on the day the second experiment was conducted. Nevertheless, slight increases in body temperature, respiration rate and heart rate due to increased ambient temperatures occurred in spite of the drug treatment, indicating that Dibenzyline or Vasodilan treatment might not alleviate the hyperthermia associated with acute fescue summer toxicosis. We have subsequently titrated doses of Vasodilan up to 120 mg (i.m.) in one steer without affecting rectal temperatures or respiration rate nor were any adverse signs of drug reaction observed. A dose of 120 mg (i.m.) of Vasoxyl into the same steer resulted in restlessness, shallow breathing, and frequent urination, presumably due to increased blood pressure, but no change in rectal temperature or respiration rate was observed.

Treatment and Control

In cases of acute fescue summer toxicosis in which hyperthermia is evident, care must be taken not to over exert the animal in the process of administering specific treatment. If possible, cool water should be poured on the body and head, and the animal quietly moved to a cool shaded area. Further therapy may include administration of fluids (normal saline or 5% dextrose in normal saline), rectal enema with cool water, and administration of a tranquilizer (acetylpromazine or xylazine) if necessary. In hot weather it may be difficult to distinguish hyperthermia from acute atypical pneumonia (62), although history which includes grazing of tall fescue along with chronic poor performance should be useful in making the diagnosis. If necessary, treatment for both conditions may be pursued with cortical steroids and antihistamine or aminophyline (62). Continued intake of the toxic fescue should be eliminated or limited to one half of the dry matter intake by supplementing with other feeds.

Management of chronic poor performance may be aided by providing shades for the grazing animals and possibly by the introduction of legumes into the pasture. The latter may be accomplished either by sod seeding or by more extensive pasture renovation. With the prospect that the toxic agents

Table 2. Effect of Dibenzyline on mean (±SEM) rectal temperature, respiration rate, and heart rate of heifers grazing two varieties of tall fescue.

			Pretreatment			Posttreatment		
Tall Fescue variety	Treatment	n	Rectal Temp. (°F.)	Respir. Rate (resp./min.)	Heart Rate (b.p.m.)	Rectal Temp. (°F.)	Respir. Rate (resp./min.)	Heart Rate (b.p.m.)
G1-307	Dibenzyline	7	102.4 ± .4	55 ± 8	79 ± 8	103.3 ± .3	75 ± 5	102 ± 8
G1-307	Control	6	102.6 ± .4	72 ± 7	75 ± 4	103.6 ± .2	86 ± 9	92 ± 7
Kenhy	Control	7	102.6 ± .3	75 ± 5	86 ± 4	103.2 ± .5	82 ± 6	91 ±4

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are limited to only some tall fescue varieties or grasses infested with specific fungi, establishment of any new pastures should take this into account.

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