

# Fescue Toxicosis Update/Bovine Hysteria from Ammoniated Forages Update

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## Fescue Summer Syndrome

Fescue toxicity in cattle includes three distinct entities: fescue foot, fat necrosis and summer syndrome. Summer syndrome has a considerably greater economic impact than either of the other disorders. Summer syndrome denotes poor performance by cattle grazing tall fescue, especially notable during hot, humid weather. Signs include reduced feed consumption, decreased rate of gain and/or milk production, rough hair coat, increased respiratory rate, increased body temperature and general unthriftiness. Only summer syndrome has been positively associated with infection of tall fescue by the fungal endophyte, *Acremonium coenophialum*.

The endophyte of tall fescue was reported in 1941 by J. C. Neil in New Zealand. *Acremonium coenophialum* is a parasite of several grasses, causing a condition known as choke disease in bent grass, prairie wedge grass and orchard-grass. Intercellular, systemic infections without apparent disease occur in tall fescue and perennial ryegrass. In New Zealand, the endophyte in perennial ryegrass has been reported to cause ryegrass staggers in sheep.

The endophyte was discovered in tall fescue in the U.S. when it was looked for in 1973. Surveys revealed a high rate of infection by the endophyte in most existing stands of tall fescue. The field in eastern Kentucky that provided the start of Kentucky 31 tall fescue was tested and found to be highly infected.

The fungus is entirely intercellular within the fescue plant. It resides in the plant crown, parallels tiller growth in the spring, infects the panicle and seed. Hyphae have not been detected in roots and are infrequently found in leaf blades. Hyphae are present in leaf sheaths, more pronounced in stems and most concentrated in seed. The only known method of natural transmission is via seed. Fungal spores of *A. coenophialum* have not been found in nature. The fungus will grow on laboratory media and will produce spores.

A toxin produced by the fungus or by the plant in response to the fungus, that will elicit signs of summer syndrome, has not been demonstrated. *A. coenophialum* has been associated with presence of loline alkaloids in tall fescue and these alkaloids have been related to summer syndrome. Ergo-peptide alkaloids were identified in toxic fescue by Shelly G. Yates, et al. Ergovaline was the most prevalent; ergotamine was not detected. Rats fed infected fescue seed

ate less and grew more slowly than rats fed fungus-free seed. Rats fed a diet containing shredded wheat on which *A. coenophialum* was cultured did as well as rats fed fungus-free fescue seed.

Cattle grazing toxic fescue pastures in hot weather responded with an increased body temperature of about 2° F. Cattle grazing in cool weather were less affected but still had a significantly elevated temperature. Serum prolactin was markedly depressed in cattle fed toxic fescue. Grazing studies have indicated a depressed rate of gain of about ½ to 1 lb per day and a reduction in animal gain per acre of 75 to 150 lb per season due to infection with the endophyte. There is an apparent straight line effect on grazing cattle by infection rate of plants in the field. The reported effect is about 0.1-0.2 lb/day ADG for every 10% change in infection rate. Cattle commingled in a feedlot after having grazed toxic or non-toxic fescue required more than 6 weeks for body temperatures, respiration rate and prolactin levels to equalize. Clinical signs attributed to summer syndrome have been noted in cattle arriving at high plains feedlots from tall fescue pastures to the east.

Limited attempts to measure effects on cattle reproduction have indicated a reduction in pregnancy rates of 20 to 30 percentage points due to grazing infected fescue. Trials with mice fed infected fescue seed resulted in reduced pregnancy rates, pups born per litter, live pups per litter, weight per pup and weight per litter. Differences became more pronounced with each successive litter. Interval between litters was lengthened by feeding infected seed. Male mice were affected only slightly less than female mice. Effects were compounded when infected seed was fed to both male and female mice.

Endophyte-free improved varieties of tall fescue are now available for new seedings. There is AU Triumph from Alabama, Forager from Indiana, Johnstone from Kentucky and MO96 from Missouri. Martin and Mozark from Missouri will be available in the next couple of years. Animal performance has been very encouraging on the endophyte-free varieties.

**Mefluidide, a plant growth regulator, has been successfully sprayed on fescue to suppress flowering and seed head formation. Animal performance was improved but the economic benefits were equivocal.**

When infected fescue is to be replaced with a non-infected variety, seed production should be prevented for at least one year; two is better. On land subject to erosion, where it is not practical to rotate crops, the existing stand can be killed with a herbicide and the new seed drilled directly into the sod. One accepted recommendation is to spray with one quart per acre of Paraquat, followed at least three weeks later with a pint per acre of Paraquat. Fall seeding is usually more successful than spring seeding.

Endophyte-free fescue appears to require better grazing management to stay in stand. It has not been determined if the new varieties are less persistent or if they are just more palatable. Endophyte-infected Kentucky 31 can certainly stand an amazing amount of grazing pressure year after year—all year long. It's not surprising that there are an estimated 35 million acres of Kentucky 31 tall fescue, and increasing, in spite of its shortcomings.

#### References

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#### Bovine Hysteria (Bovine Bonkers)

A wide range of feedstuffs has been treated with ammonia for a variety of reasons. One of the first synthesized protein supplements for cattle and sheep was ammoniated molasses. Shelled corn that could not be safely fed to livestock because of aflatoxin contamination has been detoxified by exposure to anhydrous ammonia. The protein equivalent of corn silage is effectively doubled by ammoniation. The digestibility, palatability and protein equivalent of fibrous roughages is improved by ammoniation. Ammonia has been used to preserve forages above moisture levels at which they would spoil if stored untreated.

**A syndrome of hyperexcitability in cattle and sheep was reported by ruminant nutritionists working with ammoniated molasses in the 1950s. Methods were subsequently developed to produce synthetic proteins that did not cause this "stimulation" and the problem was largely forgotten about. Then, after anhydrous ammonia was produced in large quantities for use as fertilizer, it became readily available for on-the-farm treatment of feedstuffs.**

Rare episodes of "crazy cattle" or "bovine bonkers" were reported from all over. Sometimes the syndrome appeared in cattle eating ammoniated feeds and in other instances it

appeared in the calves nursing cows which were being fed ammoniated feeds. After several cases were investigated and compared, it was determined that the syndrome was related to what had occurred thirty years before when ammoniated molasses was fed to cattle. Ammoniated feedstuffs reported to have caused cattle bonkers include: forage sorghums, sudan hay, wheat hay, wheat straw, barley hay, oat hay, fescue hay, brome grass hay, bermudagrass hay, orchardgrass hay, alfalfa hay and rice straw.

A toxin responsible for bovine bonkers has not been positively identified. The most widely suspected is 4-methylimidazole (4MI), an antimetabolite produced by altering the amino acid histidine. The generally accepted mechanism involves breaking sugar into methyl- or hydroxymethyl-glyoxal and an aldehyde, which then condenses with two moles of ammonia to produce an imidazole. Ammonia, heat, pH, moisture and sugar above some undetermined minimum level, are all apparently required for the chemical reaction that produces the toxic imidazole.

These ingredients are largely uncontrolled in on-farm ammoniation of feedstuffs. The quantity of ammonia is often excessive and its distribution uneven; available sugar, moisture and acidity vary greatly. Rate of the reaction is dependent on ambient temperature, and temperature reached within the product depends on rate of the reaction.

In trials at Kansas State University, calves 1 to 14 days of age, nursing cows fed ammoniated forage sorghum hay, developed signs of toxicity (hyperexcitability, circling, convulsions, death). None of these signs were observed in calves nursing cows fed untreated hay. No signs of toxicity were observed in any cows.

At Oklahoma State University, a cow nursing a 2-month-old calf was given daily oral doses of 4MI in increasing amounts. Signs of bonkers appeared in the cow when the daily dose reached 5 grams. She became acclimated after 3 days and the dose was increased to 10 grams. Signs of bonkers were repeated, followed by acclimation. After a 20-gram dose the cow developed convulsions and died. Her calf nursed regularly and showed no signs of toxicity. Mice fed milk from the treated cow showed no signs of toxicity. Newborn calves were then fed milk to which 200 mg 4MI/kg was added. None showed signs of toxicity.

H. B. Perdok and R. A. Leng, at the University of New England, Armidale, New South Wales, Australia, more recently reported the results of feeding ammoniated rice straw and wheat straw to several hundred cattle. They described a syndrome of hyperexcitability in cattle that ate the straw and also in calves that nursed cows that ate the straw. Signs they reported were: restlessness, rapid blinking, dilation of the pupils, impairment of vision, involuntary ear twitching, trembling, loss of balance, frequent urination and defecation, rapid respiration, salivation, frothing of the mouth, bellowing, sweating and most markedly, sudden stampeding involving galloping in circles and colliding with other animals or fences. Signs lasted for up to five

minutes and were often repeated at 20 to 30 minute intervals. Between attacks affected animals showed no signs, and resumed eating. Prior to appearance of clinical signs, animals consumed straw voraciously and intake was abnormally high. There was no evidence of direct ammonia toxicity. Rumen ammonia and blood ammonia levels of affected animals were normal.

The Australian workers reported problems only with material that exceeded 70° C (158° F) during the chemical reaction. Toxicity was more prevalent from bales of straw that were heated in a commercial oven to hasten the ammoniation reaction.

### Summary

Clinical signs of bovine bonkers have been observed in cows, bulls, yearlings and calves, including newborn calves.

## Questions & Answers:

*Question:*

*Answer:* Yes, that was reported in Oklahoma in some light weight calves. I say light weight, I think something under 500 lbs. Some calves that were on some of the large protein blocks. I might say that this problem was first reported in this country back at the beginning of attempts to make these protein supplements. They were ammoniating molasses back in the early 50s and that's when it was first reported in the literature was in that process. So it's long-standing in the literature that under certain conditions it happens. I think only now we're beginning to understand what those conditions might be.

*Question:* Did introduction of legumes completely remove the problem?

*Answer:* It does not completely remove the problem. It very well goes along with a straight line effect that you dilute out what's there and perhaps there is some thought it is more than just dilution. There actually is some beneficial effect to have the clover there in addition to the dilution factor. But, yes, that's an intermediate step and you still do get some affect.

*Question:* Do nitrates make pasture toxic?

*Answer:* Yes. The comments are that nitrogen, nitrates, applied to the pasture make it more toxic, and I think that's been pretty well demonstrated and it certainly is true in the fat necrosis syndrome and I think it has been pretty well demonstrated in the summer syndrome as well.

*Question:* Regarding preservatives used on hay at the baler to preserve higher moisture hay.

*Answer:* I have very little information on that but there are several. There are organic acids and then there are strictly

Even a calf fed pasteurized milk from a cow that ate toxic straw, was clinically affected. Some concern has been expressed about the potential for toxins in milk from cows fed ammoniated feedstuffs. Ammoniated corn silage, preserved by acid fermentation, has not been reported to have caused bovine bonkers.

### References

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drying agents. Some calcium compounds and so forth. I don't know what has been looked at in relation to the fescue toxicity at all, but there are several approaches to hay preservation.

*Question:* Do you anticipate problems where it is used strictly in a winter program?

*Answer:* One of the things that I didn't go into here is, that is being very much talked about, is pasture management. There are problems at any temperature. We've had some November and December grazing studies in Kentucky which show a definite problem. But it is much less. And so one of the things that is being recommended is to graze in the cool portion of the year. In other words, have some alternate pasture in late June to September and graze in the late winter or early spring and then in the fall. And certainly you're going to have less problems with what is known as summer syndrome by avoiding hot, humid weather grazing.

*Question:* Is there some relationship between this and the fescue foot syndrome or are those two different things?

*Answer:* Probably they are very much related and I probably should have contacted someone from Missouri and seen what more they have learned on that. But they did some work that showed, first of all, a high incidence fescue foot situation, that they had some ergovaline, high levels of ergovaline, but not ergotamine. But also, the pasture that had the high level of ergovaline was highly infected with the endophyte, whereas the pasture that was not causing the fescue foot did not have the ergovaline and also did not have the endophyte. So that would indicate there is some correlation there. But what they've done on that in the past year I really don't know if they've learned anything more about it or not.