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Effects of Increasing Dietary Sulfur Concentration on the Incidence and Pathology of Polioencephalomalicia in Weaned Beef Calves

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Story in Brief

Fourteen heifer calves weighing 384 ± 39 lb were used in a completely randomized design to evaluate the effects of three levels of dietary sulfur. Corn gluten feed (CGF) was used as the base feed. Sodium sulfate was added to reproduce clinical disease as seen in field cases. The base corn gluten feed contained 4450 ppm sulfur and was diluted with 30% cottonseed hulls (CSH) to make a basal diet to which different amounts of sodium sulfate were added. Treatments were designated as moderate (M), moderately high (MH), and high (H) and contained 3860 ppm sulfur, 5540 ppm sulfur, and 7010 ppm sulfur, respectively. Animals that developed polioencephalomalacia (PEM) were euthanized within 36 h of the first symptoms of disease and all animals that remained at the end of the trial were euthanized. Clinical polioencephalomalacia occurred in all calves assigned to moderately high and high treatments. The calves in this study did not acclimate to dietary sulfur in their diets evidenced by the occurrence of polioencephalomalacia in four animals on d 35 and one on d 37 of the trial. Microscopic lesions confirmed polioencephalomalacia in the calves assigned to moderately high and high diets. Microscopic lesions also were present in the four calves eating the moderate diet although clinical signs were not seen. Breath analysis did not yield evidence of sub-clinical lung damage. High dietary sulfur did not limit feed intake. Diets containing sulfur levels above 4000 ppm produced polioencephalomalacia in 10 calves and sub-clinical brain lesions occurred in four calves consuming a diet with less than 4000 ppm sulfur. Sulfur content should be determined before use.
Key Words: Sulfur, Polioencephalomalacia, Calves, Corn Gluten Feed

Introduction

Polioencephalomalacia is a noninfectious central nervous system disorder of ruminants. In cattle, it is most often seen in stocker calves and feedlot animals. Although it has historically been associated with impaired thiamine metabolism, high levels of dietary sulfur have been shown to initiate the disease (Olkowski et al,. 1997). The term polioencephalomalacia is descriptive of the lesions that occur in the gray matter of the brain. Lead toxicity and water deprivation-sodium ion toxicity also produce similar lesions in the brain. Kerr et al. (1989) reported that lung damage can occur due to inhalation of hydrogen sulfide.

Corn gluten feed (CGF) with a high sulfur content has caused recent cases of PEM diagnosed at Oklahoma Animal Disease Diagnostic Laboratory (OADDL). By-products of the wet milling of corn are reported to induce PEM, but reports of controlled studies using corn by-products to reproduce the disease were not found in the literature.

Corn gluten feed was used in this study to reproduce what has been observed in the field. Although it is reported that PEM affects a small percentage of cattle and that acclimation occurs (Gould et al., 1997), field

cases of PEM diagnosed at OADDL have exceeded 30% morbidity with 10% mortality over several months in grazing stocker calves.

The objective of this study was to determine the occurrence of clinical PEM and the extent of brain and lung damage caused by varying levels of dietary sulfur.

Materials and Methods

Fourteen weaned beef heifers with an average weight of 384 ± 39 lb and 8 mo of age were randomly assigned to one of three treatments M, MH, and H containing 3860, 5540, and 7010 ppm sulfur, respectively. Municipal water containing 56 ppm sulfur was provided to all animals. The CGF used in this research contained 4450 ppm as fed. The base ration (M) consisted of 70% CGF and 30% cottonseed hulls and contained 11.7% moisture. The sulfur content for M was 3860 ppm sulfur. Sodium sulfate was added to increase the sulfur content to 5540 ppm sulfur (MH), and 7010 ppm sulfur (H) for these treatments.

This research was co-sponsored by the OSU College of Veterinary Medicine and the OSU Department of Animal Science, and was conducted at the Animal Science Nutrition and Physiology Research Center. The calves were placed in individual pens and fed the base ration for 3 d before the calves in MH and H were fed their respective rations. Feed intake was monitored twice daily over the 37-d course of this study.

Blood, rumen gas, and breath samples were taken before the calves were exposed to the treatments and repeated weekly or at euthanasia. Scheduled sampling days were d O, 7, 14, 21, 28, and 35.

Blood samples were obtained by jugular veinapuncture.

Rumen gas samples containing hydrogen sulfide were obtained by rumen centesis at the left paralumbar fossa. The procedure for collection and analysis of rumen gas was developed at Colorado State University (Gould et al., 1997). A measured volume of rumen gas was drawn through a hydrogen sulfide detector tube by an airtight sampling pump and the hydrogen sulfide content read from the calibrated tubes.

Filtered breath samples were obtained and analyzed for ethane, nitrous oxide, and H_2S. The presence of significant amounts of these compounds in expired breath is an indicator of oxidative stress. Nitrous oxide is specific in detecting lung damage due to its extremely short half-life. Hydrogen sulfide was measured to investigate whether H_2S was being inhaled. Ethane is less organ specific than nitrous oxide in assessing lung damage. These samples were obtained by allowing the calf to breathe into a mask that filtered the inspired air removing greater than 99.7% of these components from atmospheric air. The breath samples were collected in airtight containers and transported to the respiratory research laboratory at Oklahoma State University College of Veterinary Medicine for analysis. Animals were fed and observed for illness twice daily.

Calves that exhibited signs of central nervous system disease evidenced by blindness, circling, head pressing, or found lying on their side unable to rise were examined and euthanized by administration of intravenous sodium pentobarbital. The same pathologist did the post mortem examinations for all animals. Gross and microscopic examinations of thymus, lung, heart, spleen, liver, kidney, reticulum, rumen, omasum, abomasum, intestine, lymph nodes, and brain was done on all animals. Bacteriology, virology, and immunohistochemistry analysis were done on lung and intestine. Liver copper, selenium, zinc, lead, and brain sodium concentrations were analyzed by the toxicology department of OADDL.

General linear models and analysis by variance (GLM ANOVA) procedures were used for the analysis of these data.

Results and Discussion

Ten calves exhibited clinical PEM (Table 1). This included all of the calves consuming MH and H diets.

The first two cases of PEM occurred on the13th and 15th d in H. The first calf was found down, semi-comatose, and unable to stand on its own. It had to be carried to the trailer. This was the only calf to develop acute symptoms. The second calf was found head pressing and would circle and run into the fence when removed from the corner. It was blind and failed to respond to menacing actions. The other cases of PEM presented with similar clinical signs and developed throughout the trial. Five calves in MH and H were diagnosed with PEM the last 3 d of the study. Acclimation to the increased sulfur did not occur during this 37 d study. Although high dietary sulfur is reported to reduce feed intake, there was no difference ($P>0.1$) in feed consumption between treatments as seen in Table 2.

The copper, selenium, and zinc content in the liver samples were within normal values. All liver samples were negative for lead and the sodium content of the brain tissue was within the normal range for all calves.

The rumen hydrogen sulfide levels of each treatment for each time period are presented in Table 3. The baseline rumen hydrogen sulfide levels ranged from 0 ppm to 60 ppm with a mean of 19.2 ± 19 ppm. Rumen hydrogen sulfide levels of less than 450 have been suggested as normal (Loneragan et al., 1998) The highest recorded value was 24,000 ppm on d 22 of the trial. This heifer developed clinical PEM on d 35. Her rumen hydrogen sulfide was 19,000 ppm on d 28 and 1500 ppm the day she was euthanized. This emphasizes that H_2S concentration of the rumen gas is dynamic and changes readily in response to changes in sulfur intake. This procedure gives valuable information on a group basis but can be misleading on individual animals.

The result of the breath analysis yielded no indication that sub-clinical lung damage occurred throughout this study. Nitrous oxide was not detected in any breath samples during this trial, and traces of H_2S were only present in two samples. Although breath ethane samples did rise in all groups, the difference between values was not significant. This is supported by the fact that no clinical illness other than neurological disease was observed throughout this trial.

Although 10 calves developed clinical PEM, gross brain lesions were seen in 12 of the 14 calves in this study, one calf each in H and M did not exhibit gross lesions. Microscopic lesions were present in all of the calves

with lesions diagnostic for PEM in MH and H treatments and lesions indicating sub-clinical brain damage in the calves assigned to M. The gross lesions included cerebral edema with flattened cerebral gyri, malacia, and focal hemorrhage and cavitation of the cerebellum. Microscopic lesions were severe polioencephalomalacia with multifocal pyogranulomatous vasculitis, meningitis, and marked menigeal edema. Hemorrhages with localized neuronal degeneration of the brainstem occurred. Microscopic lesions present in the calves that did not develop clinical PEM were congestion and edema along with microcavitations of the brainstem.

In addition to sub-clinical brain damage, feed levels of .25% dm have decreased carcass merit (Zinn et al., 1997) and levels of 2500 ppm sulfate in water have decreased feedlot performance (Wagner et al., 1997).

Corn gluten feed was chosen as the base ration for this research because recent cases of PEM diagnosed at OADDL have resulted from eating high sulfur CGF. In one case a rancher had 11 calves die, 25 became blind and 40 calves had residual brain damage out of 150 stocker calves. The CGF used by this rancher contained 6800 ppm sulfate which equates to 2267 ppm sulfur, total sulfur which also includes elemental and organic sulfur was not determined on this feed. Another cattleman had 49 ill with 17 deaths out of 559 calves. Two samples of CGF used by this cattleman contained 6800 and 8200 ppm total sulfur.

The total sulfur content of the CGF samples analyzed at Michigan State University by inductively coupled plasma technique have ranged from less than 3000 ppm to over 8800 ppm. The base CGF in this study contained 4450 ppm sulfur. The NRC (1996) lists 1500 ppm to 2000 ppm sulfur as the requirement for all classes of cattle with 4000 ppm as the maximum tolerated dose.

The presence of lesions consistent with PEM in parts of the brain other than the cerebral cortexes has been reported to be diagnostic for sulfur induced PEM, and useful in distinguishing it from other forms of PEM (Jeffrey et. al, 1994). In this study, the lesions seen in the calves that presented with both acute and classical PEM were found not only in the cerebral cortex, but also in the midbrain and brain stem. These lesions were considered to be the result of brain swelling characteristic of PEM but not diagnostic for sulfur induced PEM specifically.

Further research is needed to evaluate whether dietary sulfur fed to stocker calves at levels less than used in this research have an impact on future feedlot performance and carcass value. It also appears from this and other research that the NRC should consider reevaluating the level it lists as the maximum tolerated dose of sulfur in growing calves. In this study, a CGF based ration containing 3860 ppm sulfur resulted in microscopic brain

lesions without clinical signs PEM occurring but clinical PEM has been reported using feed containing 2600 ppm sulfur (Sagar et al., 1990).

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Table 1. Individual calf data listing PEM status and day on trial.

ID	Treatment	PEM	D/trial
702	H	Yes	13
690	H	Yes	15
529	H	Yes	21
725	H	Yes	21
576	H	Yes	35
253	MH	Yes	15
647	MH	Yes	35
739	MH	Yes	37
881	MH	Yes	35
942	MH	Yes	37
626	M	No	37
689	M	No	37
982	M	No	37

429	M	No	37

Table 2. Days on feed and feed intake of beef heifers consuming diets with three levels of sulfur.

Group	N	Days on trial	Daily DM intake
M	4	21	12.4
MH	5	31	12.1
H	5	37	10.4
LSD			2.2 lb

Table 3. Mean rumen hydrogen sulfide concentrations (ppm) in beef heifers consuming diets with three levels of sulfur.

		Treatment		
Time period		M	MH	H
1		13[a]	27[a]	16[a]
2		812[a]	1840[a]	8000[c]
3		4920[a]	2930[b]	6060[c]
4		3120[a]	13521[c]	12123[c]
5		1700[a]	4096[a]	18642[b]
SE		454	1027	1392

[a,b,c] Means within the same row without common superscripts are different P<.05.

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