## Sulfur Toxicity in Feedlot Cattle<sup>1</sup>

John J. Wagner, Ph.D.
Professor and General Manager
Southeast Colorado Research Center
Colorado State University
Lamar, Colorado

### **Introduction:**

Sulfur is an important component of many functions in the body and is an essential nutrient for beef cattle. It is an important part of the amino acids methionine, cysteine, and cystine. The B-vitamins thiamine and biotin also contain sulfur. Rumen microbes require sulfur for their normal growth and metabolism. A large portion of the sulfur found in typical feedlot diets is a component of the natural protein and most practical diets are adequate in sulfur (NRC, 1996). However, feeding diets high in non-protein nitrogen or high in rumen undegradable intake protein may reduce the amount of sulfur available for rumen microorganisms thus increasing the need for supplemental sulfur. The requirement for sulfur as stated by the National Research Council is 0.15% of diet dry matter and maximum tolerable level is listed as 0.40% of diet dry matter (NRC, 1996).

## **Sources of Sulfur:**

Total sulfur intake from all feed and water sources must be considered when evaluating nutritional programs for sulfur adequacy or excess. Table 1 lists the sulfur concentration found in several common feed ingredients. Typical diet components for feedlot cattle, including corn, alfalfa hay, and corn silage contain relatively low to moderate concentrations of sulfur. Under most circumstances, typical combinations of these feeds generally used for cattle pose little or no danger for sulfur toxicity. Several feeds, especially the co-products from the grain wet or dry milling industries may be high in sulfur. As these products are included in the diet, sulfur concentration generally increases and the risk of experiencing sulfur toxicity rises.

Sulfur concentrations in water can vary tremendously. In 1999 the National Animal Health Monitoring System conducted a study of feedlots with greater than 1000 head capacity (NAHMS, 2000). Two hundred and sixty three feedlots from 10 states supplied a water sample for analysis. Approximately 77% of the samples contained less than 300 ppm sulfate, 15% of the samples contained 300 to 999 ppm sulfate, and 8% of the samples registered greater than 1000 ppm sulfate. If a feedlot steer consumes 40 L (approximately 10 gallons) of water daily, sulfate intake from water is 4, 40, and 120 g per day if the water contained 100, 1000, or 3000 ppm sulfate. Sulfate is approximately one-third sulfur. Therefore, sulfur intake from water by the steer would be 1.3, 13.0, 40 g per head daily, respectively. If the steer was consuming 9 kg (19.8 LB) of dry matter daily that was 0.12 % sulfur, total sulfur intake expressed as a percent of dietary dry matter intake would be 0.13, 0.26, or 0.56%, respectively. It is highly likely that the steer consuming 3000 ppm sulfate would experience some degree of sulfur toxicity. At 100 or 1000 ppm the likelihood of sulfur toxicity is reduced considering the base diet was assumed to

\_

<sup>&</sup>lt;sup>1</sup> Submitted for the "Use of Ethanol By-Products in Beef Cattle Operations" meeting. Oklahoma Panhandle Research and Extension Center, Goodwell, OK. October 28, 2008.

contain 0.12% sulfur. However, if the base diet contained 30% wet distiller's grains on a dry matter basis, and if the distiller's grains contained 0.60% sulfur, an additional 0.14% ((0.60 – 0.13)\*0.30) sulfur would be added to the diet. In this instance, the steer consuming 1000 ppm water is now at risk of developing sulfur toxicity. Early in the growth of the ethanol industry, several feedlots that had successfully used marginal quality water ( $\approx$ 1000 ppm sulfate) for many years started to experience sulfur problems only after the addition of distiller's grains in the diet.

## **Manifestation of Sulfur Toxicity:**

Elemental sulfur is considered one of the least toxic minerals, however, hydrogen sulfide, a product of sulfate metabolism in the rumen, is as toxic as cyanide (NRC, 2000). The manifestation of sulfur toxicity in feedlot cattle is often a condition called polioencephalomalacia (PEM) which is characterized by necrosis of the cerebral cortex. Symptoms of the condition include blindness, poor coordination, lethargy, and seizures. Very often affected cattle are observed standing in the corner of the pen like a saw horse with all four feet spread to the extreme corners of their body (Photograph 1). Pen riders, doctors, and other feedlot personnel often refer to cattle exhibiting these signs as "brainers". This colorful name is appropriate when one considers that PEM literally means, as described by Gould (1998), softening (malacia) of the gray matter (polio) of the brain (encephalo).

Gould (1998) listed a number of research findings linking PEM outbreaks to thiamin status including a reduction in the activity of a thiamin diphosphate dependent enzyme (transketolase) in blood and an increase in the levels of thiaminases in the gastrointestinal tract. PEM has been induced by feeding thiamin antagonists (Spicer and Horton, 1981). Davies (1965) demonstrated that calves recovered from the early symptoms of PEM if high doses of thiamin are administered. The large body of evidence that associates PEM with thiamin status has led to the often erroneous assumption that outbreaks of PEM are the result of altered thiamin status and intraveneous thiamin administration is often automatically used to treat cattle with PEM. The addition of 100 to 200 mg of thiamin per head daily is often added to diets of cattle perceived to be at risk of developing PEM.

The results from efforts to treat or prevent PEM with thiamin are mixed. Much of the confusion surrounding thiamin therapy may be attributed to the fact that high sulfate intake may induce PEM through either one of, or a combination of, two distinct mechanisms. High sulfate intake has been shown to reduce duodenal thiamin flow (Goetsch and Owens, 1987) and sulfite, a product of sulfate reduction, can destroy thiamin in the rumen resulting in thiamin deficiency. This form of sulfate induced PEM may respond to thiamin therapy or may be prevented by thiamin supplementation. However, an alternative mechanism through which sulfate causes PEM may be involved particularly if sulfate intake is extremely high.

Sulfides inhibit cytochrome C, an enzyme of the electron transport chain (Evans, 1967 as cited by Loneragan, 1998). Loneragan (1998) proposed that rumen generated sulfides escaped detoxification in the liver and were responsible for sulfate induced PEM. High sulfate intake results in extreme concentrations of hydrogen sulfide in the rumen gas cap. These sulfides are inhaled during eructation, absorbed into the blood stream in the lung, and transported to the brain, thus by-passing the liver. In addition, Loneragan (1998) also suggested that the high amounts of sulfides absorbed through the rumen wall and transported to the liver may overwhelm the capacity of the liver to detoxify sulfide. Thus a portion of these sulfides may also

reach the brain. Cattle experiencing PEM caused by the inhibition of cytochrome C will not respond to thiamin therapy.

Cattle consuming high sulfur do not necessarily need to show symptoms of PEM to experience reduced feedyard performance (Wagner and Loneragan, 1996; Loneragan et al., 2001). Feedlot steers were provided with water of various sulfate concentrations ranging from 136 to 2360 ppm resulting in dietary sulfur concentrations ranging from 0.21 to 0.88% of DM. No clinically apparent symptoms of PEM were reported and performance by all steers in the study was outstanding. However, increasing sulfur concentration resulted in linear decreases in daily gain, gain to feed ratio, final weight, hot carcass weight, and dressing percentage (Table 2). Sulfur concentration by period interactions were evident for dry matter intake, average daily gain, and feed efficiency. Water sulfate concentration also influenced water intake. The effect of water sulfate on performance was greatest during the early periods of the trial and less evident toward trial completion. Water intake differences were greatest during the periods of the greatest performance reduction and not evident during the last period (Figure 1). The trial was started during the early summer (July 16) and ambient temperatures were greatest during this time. It appears that extreme water sulfate concentrations inhibit water intake by nearly 18%. It is possible that performance reductions observed for cattle consuming high sulfate water in summer may actually be a function of reduced ability of the cattle to effectively combat heat stress.

## **Respiratory Distress and Heart Failure:**

Reports from the field are surfacing that link sulfur intake with respiratory distress, pulmonary edema, and heart failure. Bulgin et al. (1996) and Coghlin (1944) have noted pulmonary edema as a feature of sulfide poisoning. Loneragan (1998) observed elevated pulmonary arterial pressure with increasing sulfur intake. Mean pulmonary arterial pressures were 29.6, 33.7, and 38.1 mmHg for steers consuming water that contained 125, 500, and 2000 ppm sulfate. Furthermore, cattle from the 2000 ppm group experienced short periods of shallow breathing immediately following eructation. It is possible that chronic inhalation of H<sub>2</sub>S is low-grade pulmonary damage.

#### **Nutritional Interventions:**

In addition to supplemental thiamin, several other nutritional manipulations have been proposed to help control sulfur induced PEM. Colorado State University scientists demonstrated up to a 37% reduction in the rate of hydrogen sulfide production from an *in vitro* fermentation system with the addition of nitrate (Gould<sup>2</sup>, personal communication). Bracht and Kung (1996) demonstrated a 77% reduction in hydrogen sulfide production when an *in vitro* system was treated with molybdenum and a 71% reduction in hydrogen sulfide production when the system was treated with 9,10-anthraquinone. Hydrogen sulfide production rate was reduced by over 75% when an *in vitro* system was exposed to clinoptilolite, a form of zeolite (Dalke<sup>3</sup>, personal communication). Feeding high levels of ammonium nitrate, molybdenum, or zeolite often reduced the hydrogen sulfide concentration in the rumen gas cap but did not improve feedlot

-

<sup>&</sup>lt;sup>2</sup> Dan Gould, Ph.D. Colorado State University. Fort Collins, CO.

<sup>&</sup>lt;sup>3</sup> Brad Dalke, Ph.D. Formerly with Grant County Feeders, ContiBeef LLC. Currently with ADM Alliance Nutrition, Inc. Ouiney, IL.

performance by steers consuming high sulfate water (≥ 2000 ppm) in experiments conducted by the author at the Southeast Colorado Research Center in the late 1990's.

Bracht and Kung (1997) presented the information shown in Figure 2 at the 1997 Rumen Function Conference. In an *In vitro* batch culture system, it appears that the addition of Chlortetracycline or Oxytetracycline to the system containing high levels of sulfur inhibited H<sub>2</sub>S production. However, the addition of Lasalocid appeared to not affect H<sub>2</sub>S production but the addition of Monensin to the system increased the concentration of H<sub>2</sub>S. These results may have implications concerning the use of various feed additives when distiller's co-products are fed.

The industry has accepted that using distiller's co-products in starter and step-up diets reduces the risk of sub-clinical acidosis due to their low starch content as compared with feed grains. Many step-up programs simultaneously introduce cattle to increased grain, distiller's co-products (including sulfur), and monensin. As grain is introduced to the diet, the rumen becomes more acidic. The hydrogen ions generated by increased acidity are generally dealt with through the production of methane. Monensin is a very effective methane inhibitor. The hydrogen ions need to go somewhere and in the presence of sulfate, H<sub>2</sub>S is produced.

Figure 3 shows the incidence rate for PEM from a commercial feedyard superimposed upon the concentration of  $H_2S$  in the rumen gas cap. Peak PEM incidence rate and peak  $H_2S$  concentration occur at approximately 21 days post feedlot arrival or about 6 days after the introduction of the finishing diet (28-30 g monensin per ton) to the cattle. These charts were originally interpreted to suggest that cattle adapt to high sulfur exposure and with time the incidence of PEM is diminished. Two peaks in H2S concentration, the first occurring almost immediately with the introduction of sulfur and the second occurring shortly after day 20 was thought to be a result of shifts in the microbial population to effectively deal with high sulfur. However, in light of the apparent effect of monensin on  $H_2S$ , maybe the initial peak in  $H_2S$  occurs as a result of exposure to Sulfur while the second peak occurs in response to the introduction of monensin. Perhaps the increase in  $H_2S$  and PEM can be alleviated through alternative strategies to introduce monensin and/or the source of sulfur into the diet.

### **Management Recommendations:**

- 1. Sample all sources of water and evaluate for sulfate concentration. Blending water from various sources to reduce the sulfate concentration to less than 1000 ppm may reduce the risk of sulfur induced PEM and lost performance.
- 2. Sample all co-product feed ingredients and analyze for sulfur.
- 3. Make certain total (water plus feed) dietary sulfur intake expressed as a percentage of dry matter intake is less than 0.40%.
- 4. Avoid stacking sulfur risk factors. Feedyards forced to use marginal or poor quality water may simply not be able to successfully utilize grain milling co-products. Likewise, simultaneous use of several high sulfur grain milling co-products should be avoided.
- 5. Logic may suggest the elimination of high sulfur trace mineral sources such as copper or zinc sulfate from the diet. However, the amount of sulfur contributed to the diet by trace mineral source is minimal compared with the sulfur contribution from grain milling coproducts or marginal to poor quality water.
- 6. Thiamin supplementation or intravenous thiamin administration may provide some measure of success in managing PEM if thiamin metabolism is compromised in the

- rumen. However, thiamin therapy or supplementation will likely be of limited value if exposure to hydrogen sulfide is excessive.
- 7. Avoid simultaneous adaptation of cattle to grain, distiller's co-products (read sulfur), and monensin. It may be more desirable to introduce cattle to sulfur only after they have been fully adapted to monensin.
- 8. To date, despite modest successes in laboratory *in vitro* systems and non-research based testimonials to the contrary, no dietary modifications have been shown to effectively control PEM or improve performance in feedlot cattle exposed to high sulfur intake.

#### **Literature Cited:**

- Bracht, J.P. and L. Kung, Jr. 1996. Inhibition of sulfide production in vitro ruminal fermentations. J. Anim. Sci. 74:2276.
- Davies, E.T. 1965. Cerebrocortical necrosis in calves. Vet. Rec. 77:290.
- Evans, C.L. 1967. The toxicity of hydrogen sulfide and other sulfides. Quart. J. Exp. Physiol. 52:231.
- Goetsch, A.L. and F.N. Owens. 1987. Effect of supplement sulfate (Dynamate) and thiamine HCl on passage thiamine to duodenum and site of digestion is steers. Arch. Anim. Nutr. 37:1075.
- Gould, D.H. 1998. Polioencephalomalacia. J. Anim. Sci. 76:309.
- Loneragan, G.H. 1998. Evaluation of the effects of sulfur intake on cattle. Master's Thesis. Department of Clinical Sciences. Colorado State University.
- Loneragan, G.H., J.J. Wagner, D.H. Gould, F.B. Garry, and M.A. Thoren. 2001. Effects of water sulfate concentration on performance, water intake, and carcass characteristics of feedlot steers. J. Anim. Sci. 79:2941.
- NAHMS. 2000. Water quality in U.S. feedlots. United States Department of Agriculture. Animal and Plant Inspection Service.
- NRC. 1996. Nutrient requirements of beef cattle. Seventh revised edition. National Academy Press, Washington, DC.
- NRC. 2000. Mineral tolerance of domestic animals. National Academy Press. Washington, DC.
- Spicer, E.M. and B.J. Horton. 1981. Biochemistry of natural and amprolium-induced polioencephalomalacia in sheep. Aust. Vet. J. 57:230.
- Wagner, J.J. and G.H. Loneragan. 1996. The effects of water sulfate content on feedyard performance, water intake, ruminal hydrogen sulfide production, and carcass merit in feedlot cattle. Continental Beef Research Res. Rep. 9608.



Photograph 1. Steer exhibiting classic symptoms of PEM.

Table 1. Sulfur concentration in feeds typically fed to feedlot cattle.

Feed commodity	NRC, 1996	Practical Range <sup>a</sup>	
Alfalfa hay	0.28	0.21 - 0.54	
Corn silage	0.12	0.10 - 0.20	
Corn grain	0.13	0.11 - 0.17	
Corn gluten feed	0.47	0.40 - 0.75	
Corn gluten meal	0.90	0.80 - 1.20	
Condensed Corn Distiller's Solubles	0.40	1.00 - 2.23	
Wet Corn Distiller's Grains plus solubles	0.44	0.35 - 0.90	
Soybean meal	0.46	0.35 - 0.60	

<sup>&</sup>lt;sup>a</sup>Based on the author's experience.

Table 2. Effect of sulfur concentration (Feed + Water) on feedyard performance and carcass merit.

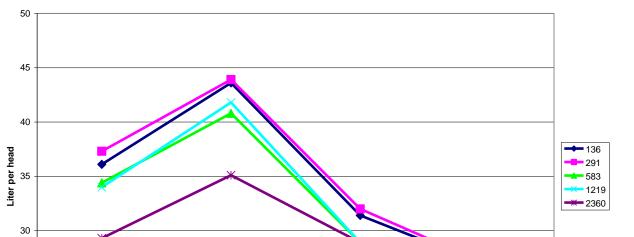
	S Concentration, % of DM					
Period	0.21	0.25	0.34	0.56	0.88	$SEM^a$
Initial weight, lb	666	665	666	663	667	
Final weight, lb	1203	1196	1204	1191	1180	9.30
ADG, lb/hd/d	4.76	4.69	4.76	4.67	4.54	0.07
DMI, lb/hd/d	21.60	23.80	22.70	21.69	21.86	0.33
F/G	4.35	5.00	4.76	4.55	4.76	0.13
$HCW^b$	767	768	768	758	751	6.47
Dressing %	63.78	64.23	63.75	63.65	63.61	0.16
Fat depth, in.	0.55	0.56	0.61	0.62	0.55	0.03
Yield grade <sup>c</sup>	3.55	3.54	3.59	3.47	3.32	0.13
Marbling <sup>d</sup>	5.07	5.08	5.15	5.08	5.02	0.07
Ch & Pr <sup>e</sup>	50.0	48.9	61.7	53.2	38.3	

<sup>&</sup>lt;sup>a</sup>Standard Error of the Mean.

bHot carcass weight, LB.
cCalculated from carcass measurements.

<sup>&</sup>lt;sup>d</sup>Marbling score units,  $5.00 = \text{Small}^{00}$ .

<sup>e</sup>Percentage of individual carcasses grading low choice or higher.



Period

Figure 1. Effect of water sulfate concentration on daily water intake by period.

Figure 2. Effect of various compounds on H2S Production.

# Effect of Various Compounds on H<sub>2</sub>S Production

