CORN CO-PRODUCTS

Managing Sulfur Concentrations in Feed and Water

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INTRODUCTION

Sulfur (S) serves many purposes in the ruminant animal. It is a component of amino acids methionine and cystine, as well as B-vitamins, biotin and thiamine and a number of other organic compounds. Elemental S, sulfates, sulfuric acid, and hydrogen sulfide all may be present in ruminant nutrition. Elemental S, sulfates, and sulfuric acid are relatively non-toxic. Hydrogen sulfide, however, is highly toxic and is responsible for the rotten egg smell that is often attributed associated with S. For most ruminants, dietary S must be between 0.18 and 0.24% of DM to allow microbes to produce sufficient S-containing compounds to support microbial growth and to provide S-containing compounds for the host animal (NRC, 2005). All S-containing compounds, with the exception of thiamine and biotin, can be synthesized from methionine, and all organic S-containing compounds can be synthesized from inorganic S by ruminal microbes (NRC, 1996).

Recent ethanol industry expansion has resulted in a large increase in the amount of corn milling byproducts available for animal feed. Information from the Renewable Fuels Association website (http://www.ethanolrfa.org/industry/resources/coproducts/) indicates that over 13 million ton of distillers grains were produced from United States ethanol plants in 2006, and approximately 85% of this feedstuff was used by beef and dairy cattle. This feedstuff has many desirable characteristics, such as high energy, protein, and fiber. Due to the use of sulfuric acid to maintain fermenter pH levels, distillers grains also contains an appreciable amount of S. The S content of distillers grains can be extremely high and also is quite variable. If not managed properly, high S concentrations in the diet, coupled with S from drinking water, may negatively affect both animal performance and animal health.

SOURCES OF SULFUR AND VARIABILITY IN SULFUR CONCENTRATION

Sulfur consumed by cattle originates almost exclusively from two sources—feed and water. Both sources can be highly variable and need to be taken into account when formulating rations. The 1996 Beef NRC lists the S concentration of distillers grains and corn gluten feed at 0.40 and 0.47%, respectively. The 2001 Dairy NRC lists S concentrations of 0.44% for both distillers grains and corn gluten feed. These concentrations are more than three times those found in corn grain, which consists of 0.14% S (NRC, 1996). The S concentration of these feedstuffs, along with the S concentration of other corn milling byproducts and feedstuffs common in beef cattle rations are listed in Table 1.

Table 1. Sulfur concentrations (% of DM) of corn milling byproducts and common feedstuffs for beef cattle.

Feedstuff	NRC, 1996	Practical Range[1]
Corn	0.14	0.11-0.17
Corn Silage	0.12	0.10-0.20
Alfalfa Hay	0.28	0.21-0.54
Brome Hay	0.21	0.15-0.35
Dried Distillers Grain w/ Solubles	0.40	0.40-1.30
Wet Distillers Grain w/ Solubles	0.40	0.40-1.30
Condensed Corn Distillers Solubles	0.40	0.80-1.50
Dakota Bran[2]	----	0.70-0.80
Wet Corn Gluten Feed	0.47	0.40-0.90
Sweet Bran[3]	----	0.45-0.55
Corn Gluten Meal	0.90	0.80-1.20
Steep	----	0.80-1.10

[1] Based on personal observations as well as data from Galen Erickson (personal communication), and John Wagner (http://www.admani.com/byproducts/Technical_Info/Sulfur%20Toxicity.htm).
[2] Product of Poet Nutrition.
[3] Product of Cargill Corn Milling.

Perhaps of more concern than the high S concentration in corn milling byproducts is the variability. Because the ethanol industry is a relatively new industry with rapidly changing technology, the quality and consistency of byproducts can differ greatly both within and among plants. Data from the University of Minnesota's Distillers Grains By-products website (www.ddgs.umn.edu) reported S content of dried distillers grains with solubles (DDGS) from 34 ethanol plants in 11 states. These data reported a range in S concentration of 0.31 to 1.93%, with an overall mean of 0.68%. From eight Minnesota ethanol plants, the S range was 0.34 to 1.05%, with an overall mean of 0.69%. Spiehs et al. (2002) obtained 118 DDGS samples over two years from 10 ethanol plants in South Dakota and Minnesota, and reported a range in S concentration of 0.33 to 0.74%, with an overall mean of 0.47%. Coefficients of variation (CV) within ethanol plant ranged from 6.4 to 40.8% with a mean of 37.1% in the Spiehs et al. (2002) study, with only two of the ten ethanol plants having a CV of less than 10%. Holt and Pritchard (2004) measured the S concentration of DDGS, wet distillers grains with solubles (WDGS) and distillers solubles from four South Dakota ethanol plants on four consecutive days and reported a range in S concentration from 0.37 to 0.69% for DDGS; 0.36 to 0.39% for WDGS; and 0.25 to 1.5% for distillers solubles. In a Cargill Animal Nutrition analysis of 86 DDGS and 75 WDGS samples from various ethanol plants, S concentrations averaged 0.69% with a CV of 28% for DDGS, and 0.66% with a CV of 46% for WDGS (Pablo Guiroy, personal communication). Table 2 shows the effect of increasing WDGS S concentration on total S intake at different temperatures.

Water sulfate is also a major contributor to total S intake by cattle, especially in summer months. Similar to S from feed sources, water S may be highly variable, and can be extremely site-specific. A USDA APHIS study (USDA, 2000) collected water samples from 263 feedlots in 10 major cattle feeding states to determine water quality. The states included in this study were California, Colorado, Idaho, Iowa, Kansas, Nebraska, New Mexico, South Dakota, Texas, and Washington.

From these samples, a mean water sulfate concentration of 204.9 ppm was reported. Samples from Idaho feedlots had the lowest water sulfate concentrations, at 28.7 ppm, while samples from South Dakota had the highest water sulfate concentrations, averaging 1007.1 ppm. Sulfate is 0.35% S, therefore a 30 ppm water sulfate concentration would equal approximately 10 ppm S. According to Wright (2007), water sulfate concentrations less than 1,000 ppm are generally safe (Table 3), although the NRC (2005) recommends that water for feedlot cattle contain less than 600 ppm sulfate. Water sulfate concentrations between 1,000 and 2,000 ppm will likely have no effect on grazing cattle, but may reduce performance in confined cattle. In addition, these water sulfate concentrations may result in diarrhea and a slight reduction in Cu bioavailability (Wright, 2007). Water sulfate concentrations, and more specifically, water S concentrations, should be assessed in combination with dietary S levels to determine total S intake.

Table 2. Effect of temperature and WDGS S concentration on total S intake by a 1,000 lb. feedlot steer[1,2].

	Temp (F) and Water Intake		
WDGS S, % of DM	40° 9.5 gal	70° 11.9 gal	90° 21.6 gal
0.4	0.24	0.25	0.27
0.5	0.27	0.28	0.30
0.6	0.30	0.31	0.33
0.7	0.33	0.34	0.36
0.8	0.36	0.37	0.39
0.9	0.39	0.40	0.42
1.0	0.42	0.43	0.45

[1]Diet = 57.5% DRC, 30% WDGS, 7.5% alfalfa hay, 5% supplement. Water sulfate = 200 ppm.
[2]Data generated using Colorado State University Total Sulfur Intake Calculator (http://www.dlab.colostate.edu/webdocs/special_cases/sulfurcalc.cfm)

Research from South Dakota (Butler and Wright, 2006) reported mean water sulfate concentrations of 347, 849, and 568 ppm when water samples were collected from runoff-fed dugouts, spring-fed dugouts, and wells, respectively. In Minnesota there are ten primary surface water basins. A 1999 study by the Minnesota Pollution Control Agency (MPCA, 1999) sampled water from multiple locations in each of these basins, and reported mean water S concentrations ranging from 2.7 ppm in the Cedar River basin in south central/southeastern Minnesota to 226 ppm in the Des Moines River basin in south central/southwestern Minnesota. An interesting observation from these data is that these two surface water basins border one another in southern Minnesota, an indication of the extreme variation that can be present in water sulfate concentration.

Table 3. Maximum recommended water sulfate concentrations for cattle[1].

Water sulfate, ppm (mg/L)	Comment
Less than 600	Safe
600-1,000	Generally safe. Slight performance reductions in confined cattle may occur with high water intakes.
1,000-2,000	Grazing cattle not likely to be affected. Performance may be reduced, particularly in confined cattle consuming dry feed. May result in diarrhea. May cause slight reduction in Cu availability.
2,000-3,000	Performance likely to be reduced, particularly in confined cattle consuming dry feed. Grazing cattle may also be affected. Likely to result in diarrhea. May cause substantial reduction in Cu availability. Sporadic cases of S-induced PEM possible.
3,000-4,000	Performance will likely be reduced in all classes of cattle. Likely to result in diarrhea. May cause substantial reduction in Cu availability. Sporadic cases of S-induced PEM are likely.
Greater than 4,000	Potentially toxic. Should be avoided.

[1]Adapted from Wright (2007) with modifications based on NRC (2005) recommendations.

Although water S may not receive as much attention as feed S, they both contribute to total S intake. In instances where water S is extremely high, as in the South Dakota example from the USDA (2000) report, water S may severely limit the inclusion of corn milling byproducts in beef cattle rations. Water S intake will certainly increase during hot temperatures, as cattle may drink more than two gallons of water per 100 pounds of body weight when temperatures exceed 80˚ F (Mader et al., 2000). This equates to greater than 20 gallons of water intake for a 1,000 lb steer, which is more than twice the water intake of a 1,000 lb steer at a temperature of 40˚ F. Table 4 shows the effect that increases in temperature and water sulfate concentration have on total S intake in a 1,000 lb. feedlot steer consuming a diet containing 30% (DM basis) of a 0.6% S WDGS.

Table 4. Effect of temperature and water sulfate concentration on total S intake by a 1,000 lb. feedlot steer[1,2].

	Temp (F)/Water Intake		
Water Sulfate, ppm	40° 9.5 gal	70° 11.9 gal	90° 21.6 gal
50	0.29	0.29	0.29
100	0.29	0.29	0.31
200	0.30	0.31	0.33
500	0.33	0.35	0.40
1,000	0.39	0.41	0.52
1,500	0.44	0.48	0.64
2,000	0.49	0.55	0.76
2,500	0.55	0.61	0.88

[1]Diet = 57.5% DRC, 30% WDGS, 7.5% alfalfa hay, 5% supplement. WDGS = 0.6% S. Dietary S = 0.28%.
[2]Data generated using Colorado State University Total Sulfur Intake Calculator (http://www.dlab.colostate.edu/webdocs/special_cases/sulfurcalc.cfm)

EFFECTS OF HIGH SULFUR INTAKE

The NRC (1996) recommended 0.15% dietary S for both beef finishing cattle and gestating and lactating cows, and the maximum tolerable S concentration was set at 0.40%. More recent guidelines (NRC, 2005) provided two recommendations based on forage concentration in the diet. For beef cattle diets containing less than 15% forage, the maximum tolerable concentration is 0.30% S, and for diets containing greater than 40% forage the maximum tolerable concentration is 0.50% S. It is also noted in the 2005 NRC that drinking water for cattle fed high-concentrate diets should not contain more than 600 ppm sulfate.

Gould et al. (2002) recognized two primary mechanisms by which excess S may affect cattle health and performance. First, ruminal reduction of S produces intermediates that complex with copper (Cu) and possibly others minerals, resulting in decreased mineral bioavailability. Second, sulfate and other non-toxic forms of S are reduced by ruminal microbes to hydrogen sulfide and its ionic forms, which are highly toxic, and these compounds interfere with cellular respiration. Both of these mechanisms may decrease animal performance, with the latter mechanism likely having more critical impacts on animal health. Both mechanisms outlined by Gould et al. (2002) indicate that S is more dangerous in its reduced forms, particularly hydrogen sulfide, than as sulfates or elemental S.

High dietary S can decrease the bioavailability of trace minerals through formation of insoluble complexes within the rumen. One such interaction is that of Cu, S, and molybdenum (Mo), which combine to form Cu tetrathiomolybdate. This complex renders Cu unavailable to the animal (NRC, 2005). Suttle (1991) reported a 50% decrease in Cu absorption when dietary S concentration increased from 0.2 to 0.4%. In two separate experiments, Wright and Patterson (2005) assessed Cu status of steers drinking high sulfate water and reported a 58.5% reduction in liver Cu concentration when water sulfate concentration increased from 404 to 3,947 ppm in the first

experiment, and an 88.6% reduction in liver Cu concentration when water sulfate concentration increased from 441 to 4,654 ppm in the second experiment. For the first and second experiments, total S intake was 0.93 and 1.1% of DM for the high S treatments, respectively, while the control treatments each contained approximately 0.26% total S. Gould (1998) reported the bioavailability of other minerals, particularly iron and zinc, may be limited due to the formation of insoluble salts with sulfide. Availability of selenium (Se) may also be limited due to S, as Ivancic and Weiss (2001) reported decreased true digestibility of Seas dietary S content increased, and Ganther and Bauman (1962) reported increased urinary excretion of Se with excess dietary S concentrations.

A subacute effect of excess dietary S is reduced animal performance. Reduced performance as a result of increased dietary S concentration was reported by Zinn et al. (1997), in which heifers were fed a high-concentrate diet with 0.15, 0.20. and 0.25% dietary S. Average daily gain and feed efficiency both responded quadratically to dietary S concentration, with reductions of 22% in ADG and 18% in feed efficiency when dietary S concentration increased from 0.20 to 0.25%. In growing steers, Zinn et al. (1999) reported a linear decrease in feed efficiency when diets contained 0.17, 0.22, and 0.27% S. Loneragan et al. (2001) reported a linear decrease in ADG and feed efficiency when steers consumed high-concentrate diets containing 0.18, 0.19, 0.22, 0.29, and 0.40% S. Bolsen et al. (1973) reported reductions in ADG of 10, 12, and 43% when dietary S concentration increased from 0.12 to 0.14, 0.19, and 0.41%, respectively. Spears and Lloyd (2005) reported reduced DMI in steers at dietary S concentrations of 0.31 and 0.46% compared with steers consuming a 0.13% S treatment. Average daily gain was also reduced with the 0.41% S treatment compared with the 0.13% S treatment.

More extreme effects of excess S involve hydrogen sulfide toxicity, and may lead to the central nervous system disorder poioencephalomalacia (PEM), which is also known as polio or brainers. Polioencephalomalacia is a softening of the gray matter of the brain. The symptoms of this disorder may initially include separation from group, cattle going off feed, “stargazing” in which cattle hold their head in a high, upward-looking position, head pressing, teeth grinding, and a staggered gait. More advanced symptoms may include blindness, seizures, and coma (Merck, 2006). Because many PEM symptoms may also be present with other common gastrointestinal or respiratory disorders, PEM is often misdiagnosed (Cebra and Cebra, 2004).

Two basic forms of PEM have been described: Thiaminase-induced PEM and S-induced PEM. Thiaminase induced PEM occurs due to the production of thaiminase I in the rumen, which will break down thiamine. The lack of thiamine will inhibit thiamine-dependent reactions of glycolysis and the trans-carboxylic acid cycle (Brent and Bartley, 1984). This activity appears to be due to a shift in the ruminal environment from gram-negative to gram-positive bacteria, which commonly will occur during adaptation to a high-concentrate diet (Brent, 1976). Diets containing bracken fern (Evans et al., 1975) and amprolium (Lilja, 1973) have been associated with decreased thiamine utilization. Lusby and Brent (1972) reported 150 mg/d of thiamine resulted in decreased PEM incidence in sheep, and suggested that a 1 g/d dose could have the same effect in cattle. Ward and Patterson (2004) did not observe a significant decrease in PEM incidence when steers consuming water containing 3,786 ppm sulfate were supplemented with 1 g/hd/d of thiamine, even though PEM incidence dropped from 14.3% without supplementation to 4.8% with thiamine supplementation.

Sulfur-induced PEM has symptoms and outcomes similar to those of thiaminase-induced PEM. Thiaminase- and S-induced PEM were previously thought to be closely related, as the sulfite ion

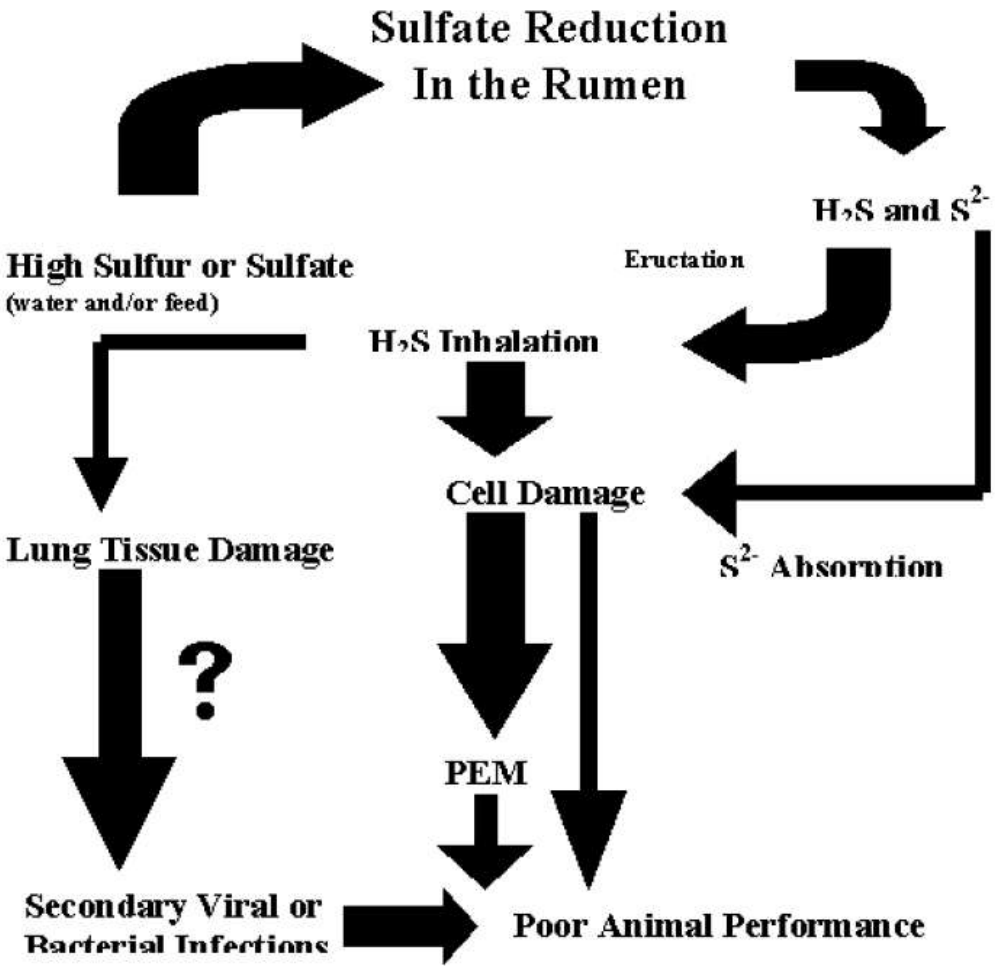
(Brent and Bartley, 1984) and sulfur dioxide (NRC, 2005) may be able to cleave thiamine and therefore limit thiamine availability for cellular metabolism. However, other evidence (Gould et al., 1991; Olkowski et al., 1992; Oliveira et al., 1996; 1997; Loneragan et al., 1997; 1998; McAllister et al., 1997) reported adequate ruminal and blood thiamine levels during cases of S-induced PEM, suggesting that PEM was directly due to S, and not due to S action on thiamine.

Water and feed sources of S have been implicated in cases of S-induced PEM. Patterson and Johnson (2003) reported PEM incidence of 0, 15, and 12.5% when steers consumed water containing 400, 3100, and 3900 ppm sulfate, respectively. When combined with dietary S intake, these sulfate levels corresponded with S intakes of 0.27, 0.74, and 0.93% of DM. Niles et al. (2000) fed 14 heifer calves diets with either 0.39, 0.55, or 0.70% dietary S, and observed clinical PEM symptoms in all ten of the heifers consuming the 0.55 and 0.70% dietary S treatments, while none of the heifers consuming the 0.39% S treatment exhibited signs of PEM.

McAllister et al. (1997) reported the incidence of PEM cases in feedlots is seasonal and related to days on feed. The incidence of PEM peaked in the summer months, and also peaked between 15-30 days on feed. The increased incidence in summer months is likely due to increased water sulfate intake. The relationship to days on feed could be due to the changes in the ruminal environment that are associated with adapting cattle to a high-concentrate diet. Sager et al. (1990) and Low et al. (1996) both observed clinical signs of PEM beginning on day 15 after adaptation to a high-concentrate diet with excess S. During this time, ruminal pH becomes increasingly more acidic. The pKa for hydrogen sulfide is 7.2 (Kung et al., 1998), which indicates that as ruminal pH decreases, more hydrogen sulfide will be in the more toxic protonated form. At a ruminal pH in the 5.0-5.5 range, which may occur with high-concentrate rations, nearly 100% of the hydrogen sulfide would be in the protonated form.

Gould et al. (2002) indicated that the capacity of ruminal microbes to generate hydrogen sulfide increases under conditions of increased dietary S intake. This would limit the amount of sulfide that would be able to be absorbed across the rumen wall, and this proportion is likely detoxified before it reaches the brain (Kandylis, 1984). Protonated hydrogen sulfide is not able to be absorbed across the rumen wall (NRC, 2005); therefore there must be a separate route for hydrogen sulfide to enter the bloodstream and lead to PEM. Dougherty and Cook (1962) reported that during eructation, ruminants normally inhale eructated gases into the lungs. In fact, as much as 60% of eructated gases are inhaled and enter the respiratory tract (Bulgin et al., 1996). Therefore, inspired hydrogen sulfide appears to be a major factor in PEM incidence. Large amounts of hydrogen sulfide can be absorbed across the lungs during eructation and could proceed to cause PEM (NRC, 2005). Upon absorption through the respiratory tract, hydrogen sulfide can bypass hepatic detoxification, which can lead to its toxic effects on the central nervous system (Figure 1).

Figure 1. Proposed mechanism for high-sulfate induced polioencephalomalacia. Adapted from Kung et al. (1998).



Prior to eructation, gases build in the rumen gas cap. Acidic conditions favor a large rumen gas cap (Gould, 1998), which again predisposes high-concentrate fed cattle to potential toxicity due to excess S intake. Gas cap hydrogen sulfide concentrations are much higher than hydrogen sulfide concentrations in ruminal fluid (Gould et al., 1997). It appears that rumen gas cap hydrogen sulfide concentration peaks approximately 1-3 weeks after introduction of a high-S diet. This could indicate that ruminal microbes gradually adapt to increased S intake before increasing hydrogen sulfide production (Cummings et al., 1995; McAllister at al., 1997). The increase in gas cap hydrogen sulfide concentration corresponds with an appearance of clinical PEM symptoms (NRC, 2005).

An in-vitro experiment by Kung et al. (2000) supplemented 5 ppm monensin to in vitro cultures provided with a high-S (1.09% of dietary DM) diet. The authors observed a 54% increase in sulfide gas concentration with the supplemental monensin. In a separate in vitro experiment, the authors observed a 27% increase in sulfide gas concentration when 5 ppm monensin was added to the same high-S diets. The authors hypothesized that monensin may have indirectly inhibited methanogens, therefore reducing competition between methanogens and sulfate-reducing bacteria. Monensin is a common feed additive for United States feedlots, and because of this

further investigations into the effect of monensin on hydrogen sulfide production should be pursued.

TREATMENT OF SULFUR TOXICITY

Although thiamine's role in the occurrence of PEM is not entirely clear, it is the primary method of treatment for afflicted animals. An intravenous injection of thiamine (10 mg/kg of body weight; Cebra and Cebra, 2004) is suggested. Administration of this dose should continue every 6 hours for several days. After the initial dose, injections may be administered intramuscularly, and initial intramuscular injections may also be used for animals with milder symptoms. Repeated administration may be necessary, as cattle may not respond to a single injection (Haydock, 2003). In severely affected animals, residual blindness may continue long after other clinical signs have dissipated (Cebra and Cebra, 2004). In addition to these treatments, high S-containing feedstuffs should be removed or limited in the ration upon PEM occurrence. Addition of roughage to the ration may also be helpful. If possible, high-sulfate water should be replaced or diluted with lower-sulfate water.

MANAGING HIGH SULFUR CONCENTRATIONS

Possible strategies to manage high sulfur concentrations include limiting the amount of high S feedstuffs or water consumed, offering feed additives that may combat high S intakes, or using reported measures of S variability in feedstuffs to maximize use of high S feedstuffs without causing potential S-induced problems. Because high S feedstuffs are increasing in supply and have multiple applications in beef cattle diets, the best strategies are to manage S concentrations through utilizing additives that may offset high S and through accounting for feedstuff variability to maintain animal health and productivity.

In addition to previously discussed supplementation of monensin to in vitro cultures, Kung et al. (2000) analyzed the effects of Mo, avoparcin, bacitracin, bambermycin, lasalocid, chlortetracycline, oxytetracycline, as well as an experimental compound, anthraquinone, on sulfide production in vitro. In vitro sulfide gas production was not affected by 1 ppm molybdenum inclusion, but decreased when 10 and 25 ppm were added to in vitro cultures. The inhibition of ruminal sulfide production by Mo has been reported elsewhere (Loneragan et al., 1998). However, the complex of S and Mo forms molybdate, which binds Cu and results in decreased Cu bioavailability (Loneragan et al., 1998). Anthraquinone, bambermycin, chlortetracycline, oxytetracycline, and lasalocid all reduced in vitro sulfide gas production, with the greatest reductions occurring with anthraquinone, chlortetracycline, and oxytetracycline. Chlortetracycline and oxytetracycline are classified as broad spectrum antibiotics, and have activity against both gram negative and gram positive bacteria (Nagaraja, 1995). Kung et al. (2000) noted that gram negative bacteria are primarily responsible for hydrogen sulfide production in the rumen. Both chlortetracycline and oxytetracycline reduced total in vitro VFA concentration compared with the high S control in the Kung et al. (2000) experiment, and this could indicate both a direct inhibition of sulfate-reducing bacteria and an increase in ruminal pH through decreased VFA production that could decrease the concentration of protonated hydrogen sulfide in the rumen.

As mentioned previously, ruminal thiamine status may not be affected by the occurrence of S-induced PEM. However, dietary thiamine concentrations should be monitored to ensure that adequate thiamine is available to cattle. Although it appears that thiamine may not have a direct

effect on the onset of S-induced PEM, supplemental thiamine should be considered to avoid thaiminase-induced PEM. Supplemental Cu has also been suggested for cattle with high S intakes (Gooneratne et al., 1989). These authors suggested supplementation of Cu up to 50 ppm of diet DM to alleviate deficiencies in both Cu and thiamine.

Another method to manage high S concentrations is to account for variability in feedstuffs. This can be accomplished through formulation that allows for a margin of error in dietary S concentration. Managing variability in feedstuff nutrient concentrations has been described in general terms by St-Pierre and Weiss (2007), and these basics can be applied to S as well. What is required to manage S concentrations in this manner is a measure of mean S concentration in feedstuffs and water and also a measure of variability, such as a standard deviation (SD). A mean value should be available from the ethanol plant where the distillers grains originated, although a SD may be more difficult to obtain. The SD represents the dispersion of values in relation to the mean. Or, as described by St-Pierre and Weiss (2007), a SD is an estimate of how wrong you could be in using a mean value. In a normal distribution, approximately 38% of all observations will be within 0.5 SD units of the mean, approximately 68% of all observations are within 1 SD of the mean, and approximately 95% of all observations are within 2 SD of the mean. For example, the Dairy NRC (2001) lists the S content of DDGS as 0.44% with a SD of 0.15. Using these data, 68% of DDGS samples will have a S concentration between 0.29 and 0.59%. This also indicates that approximately 16% of samples will have S concentrations greater than 0.59%. An analysis of 75 WDGS samples resulted in a mean S concentration of 0.66% with a SD of 0.30 (Pablo Guiroy, personal communication). This indicates that approximately 68% of all WDGS samples will have S concentrations between 0.36 and 0.96%, and 16% will have S concentrations greater than 0.96%.

To apply this to ration formulation, one must first consider what their tolerance is for S concentrations. By simply using the mean S value from the WDGS samples described above, approximately 16%, or 1 out of 6 loads of WDGS sampled will have a S concentration greater than 0.96%. If from previous experience or personal preference this is deemed acceptable, then the mean S value should be used in formulation. If this is not acceptable, one should consider using the mean plus 0.5 or 1 SD to formulate rations. Using 0.5 SD above the mean would result in using a S concentration of 0.81% in ration formulation, and approximately 7%, or 1 out of 14 loads will exceed this formulated value. By using the mean plus 1 SD, rations would be formulated based on a WDGS S concentration of 0.96%, and approximately 2 out of 100 WDGS loads would have S concentrations exceeding 0.96%.

In addition to these management strategies, attention should be given to proper feed mixing and bunk management to ensure that ration ingredients are distributed evenly throughout the bunk. Even if an analysis of distillers grains S content is available from ethanol plants, producers should sample each load of distillers grains as it arrives. Because the turnover time for sample analysis is usually longer than the useful life of the distillers grains, Pritchard (2007) recommended keeping the sample in a freezer until the corresponding load is gone. If problems arise while feeding this particular load of distillers grains, a sample is available for analysis. If no problems arise, the sample can be discarded.

TAKE-HOME MESSAGE

Corn milling byproducts have great application for beef cattle. However, in feeding these products one must consider not only the reported S content but also the variability associated with this

measurement. In addition to accounting for S in feedstuffs, S concentrations in water must also be recognized. Water intakes are not static throughout the year, and increased water intake in the summer will result in greater S intake. For feedlot cattle, total S intakes should not exceed 0.30% of DM. For cattle consuming diets with greater than 40% forage, total S intakes should not exceed 0.50% of DM. Animals will vary considerably in their ability to handle excess S intake. For animals that are affected, reductions in ADG and feed efficiency may occur, with more severe cases potentially resulting in PEM. Cattle fed high-concentrate diets are most susceptible, and susceptibility is also increased when cattle are adapted to a high concentrate diet. During these times, supplementation with oxytetracycline or chlortetracycline may limit the negative effects of excess S. Supplemental Cu may be useful to overcome reduced Cu bioavailability from binding with S and Mo. Through utilization of the mean and SD of S concentration, rations can be formulated to limit the impact of variation in distillers grains S concentration. In addition to management practices specifically employed due to variation in S concentration, normal management practices such as proper feed mixing and bunk management may also assist in preventing negative effects due to excess S intake.

LITERATURE CITED

Bolsen, K. K., W. Woods, and T. Klopfenstein. 1973. Effect of methionine and ammoniumsulfate upon performance of ruminants fed high corn rations. J. Anim. Sci. 36:1186-1190.

Brent, B. E. 1976. Relationship of acidosis to other feedlot ailments. J. Anim. Sci. 43:930-935.

Brent, B. E., and E. E. Bartley. 1984. Thiamin and niacin in the rumen. J. Anim. Sci. 59:813-822.

Bulgin, M. S., S. D. Stuart, and G. Mather. 1996. Elemental sulfur toxicosis in a flock of sheep. JAVMA. 208:1063-1065.

Butler, L., and C. L. Wright. 2006. North Central water quality survey. South Dakota State Univ. Beef Report. BEEF 2006-12: 52-54.

Cebra, C. K., and M. L. Cebra. 2004. Altered mentation caused by polioencephalomalacia, hypernatremia, and lead poisoning. Vet. Clin. Food Anim. 20:287-302.

Cummings, B. A., D. H. Gould, D. R. Caldwell, and D. W. Hamar. 1995. Ruminal microbial alterations associated with sulfide generation in steers with dietary sulfate-induced polioencephalomalacia. Am. J. Vet. Res. 56:1390-1395.

Dougherty, R. W., and H. M. Cook. 1962. Routes of eructated gas expulsion in cattle-a quantitative study. Am. J. Vet. Res. 23:997-1000.

Evans, W. C., A. Evans, and D. J. Humphreys. 1975. Indication of thiamine deficiency in sheep with lesions similar to those of cerebrocortical necrosis. J. Comp. Pathol. 85:253-267.

Ganther, H. E., and C. A. Bauman. 1962. Selenium metabolism. II. Modifying effects of sulfate. J. Nutr. 77:408-412.

Gooneratne, S. R., A. A. Olkowski, R. G. Klemmer, G. A. Kessler, and D. A. Christensen. 1989. High sulfur related thiamine deficiency in cattle: A field study. Can. Vet. J. 30:139-146.

Gould, D. H. 1998. Polioencephalomalacia. J. Anim. Sci. 76:309-314.

Gould, D. H., B. A. Cummings, and D. W. Hamar. 1997. In vivo indicators of pathologic ruminal sulfide production in steers with diet-induced polioencephalomalacia. J. Vet. Diagn. Invest. 9:72-76.

Gould, D. H., D. A. Dargatz, F. B. Garry, and D. W. Hamar. 2002. Potentially hazardous sulfur conditions on beef cattle ranches in the United States. JAVMA. 221:673-677. Gould, D. H., M. M.

McAllister, J. C. Savage, and D. W. Hamar. 1991. High sulfide concentrations in rumen fluid associated with nutritionally induced polioencephalomalacia in calves. Am. J. Vet. Res. 52:1164-1169.

Haydock, D. 2003. Sulfur-induced polioencephalomalacia in a herd of rotationally grazed beef cattle. Can. Vet. J. 44:828-829.

Holt, S. M., and R. H. Pritchard. 2004. Composition and nutritive value of corn co-products from dry milling ethanol plants. South Dakota State Univ. Beef Rep. BEEF 2004-01:1-7.

Ivancic, Jr., J., and W. P. Weiss. 2001. Effect of dietary sulfur and selenium concentrations on selenium balance of lactating Holstein cows. J. Dairy Sci. 84:225-232.

Kandylis, K. 1984. Toxicology of sulfur in ruminants: Review. J. Dairy Sci. 67:2179-2187.

Kung, Jr., L., J. P. Bracht, A. O. Hession, and J. Y. Tavares. 1998. High sulfate induced polioencephalomalacia (PEM) in cattle-burping can be dangerous if you are a ruminant. Proc. Chr. Hansen's Technical Symposium. Pacific Northwest Nutrition Conference.

Vancouver, BC, Canada. http://ag.udel.edu/anfs/faculty/kung/H2S.htm. Accessed July 23, 2007.

Kung, L., J. P. Bracht, and J. Y. Tavares. 2000. Effects of various compounds on in vitro ruminal fermentation and production of sulfide. Anim. Feed Sci. Tech. 84:69-81.

Lilja, C. 1973. Cerebro-cortical necrosis (CCN) in the calf. An experimental reproduction of the disease. Acta. Vet. Scand. 14:464-473.

Loneragan, G. H., D. H. Gould, R. J. Callan, C. J. Sigurdson, and D. W. Hamar. 1998. Association of excess sulfur intake and an increase in hydrogen sulfide concentrations in the ruminal gas cap of recently weaned beef calves with polioencephalomalacia. J. Am. Vet. Med. Assoc. 213:1599-1604.

Loneragan, G. H., D. H. Gould, J. J. Wagner, F. B. Geary, and M. Thoren. 1997. The effect of varying water sulfate content on H2S generation and health of feedlot cattle. J. Anim. Sci. 75(Suppl. 1):540(Abstr.).

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. 79:2941-2948.

Low, J. C., P. R. Scott, F. Howie, M. Lewis, J. FitzSimmons, and J. A. Spence. 1996. Sulphurinduced polioencephalomalacia in lambs. Vet. Rec. 138:327-329.

Lusby, K. S., and B. E. Brent. 1972. An experimental model for polioencephalomalacia. J. Anim. Sci. 35:270(Abstr.).

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