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Burping Can be Dangerous if you are a Ruminant: Issues with High Sulfur Diets

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INTRODUCTION

Rumen microorganisms and the host ruminant animal require many macro and micro minerals for normal growth and development. Among these minerals, sulfur is a necessary component of the amino acids cystine and methionine that are building blocks of proteins. In ruminants, many inorganic forms of sulfur (e.g. potassium sulfate and calcium sulfate) can be used because sulfate is reduced in the rumen to sulfide by a group of bacteria referred to as the sulfur reducing bacteria and subsequently incorporated into microbial protein. However, excess production of sulfides in the rumen may be detrimental because high levels can cause polioencephalomalacia (PEM) (Lowe et al., 1996; Gould et al., 1991; McAllister et al., 1992). Polioencephalomalacia is a disease condition characterized by necrosis of the cerebrocortical region of the brain. A thiamine deficiency has been the most common cause of PEM in ruminants. Excess sulfur or sulfate in feed or water has been the second most reported condition associated with PEM. Consumption of excess lead (Christian and Tryphonas, 1971) and water deprivation (Sullivan, 1985) can result in the disease in some instances. High sulfur diets was the topic of a recent symposium at the 2008 ASAS Midwestern Section Meetings (http://ars.sdstate.edu/extbeef/). The objective of this paper is to review the relationships between the intake of high sulfur and PEM, and to discuss potential methods for control.

SULFATE METABOLISM IN THE RUMEN

Sulfur reducing bacteria in the rumen utilize anaerobic respiration pathways for bioenergetic processes. The distinction between aerobic and anaerobic respiration is determined by the nature of the final reduced compound. In the process of aerobic respiration, electrons produced from reduced compounds are coupled to the reduction of oxygen, however, in anaerobic respiration electrons from oxidative reactions are used to reduce a variety of different compounds, e.g. SO_4^-, NO_3^-, or CO_2 (Liamleam and Annachhatre, 2007). Of particular interest is the reduction of SO_4^- (sulfate). The SRB are grouped by the mechanism used to reduce sulfates; either an assimilatory process, or a dissimilatory process. In general, the dissimilatory reduction of sulfur compounds is used for energy production, while the assimilatory process reduces sulfur compounds for the incorporation of the sulfur into other biological compounds necessary for cell survival

(Odom and Singleton, 1993). In the rumen, SRB from both the assimilatory and dissimilatory groups exist, and the latter are responsible for the reduction of sulfur to hydrogen sulfite and hydrogen sulfide. Although many bacteria can produce sulfides, organisms from the Desulfovibrio and Desulfotomaculum genus are most likely the predominant sulfate-reducing bacteria in the rumen (Cumming et al., 1995b).

In the rumen, the extent of dissimilatory sulfate reduction is proportional and limited to the amount of sulfur containing compounds. The sulfide compounds that are predominantly formed in the dissimilatory process are S_2^-, S^o, HS^-, or HSO_3^- (Odom and Singleton, 1993). The pKa values for these compounds are around 7.0 (the pKa for H_2S is 7.2). Because the pH range of a normal rumen is between 5.5 and 7.2, these reduced forms of sulfide are readily protonated. For this reason, most of the sulfide present in the rumen is found in the gas phase as hydrogen sulfide (H_2S), and a small amount is left in the liquid phase in a variety of sulfide containing compounds.

Under normal feeding conditions, Hungate (1966) suggested that if equilibrated with rumen fluid, H_2S concentration in ruminants was 0.1 umol per milliliter. High concentrations of sulfides in ruminal fermentations have been reported in vivo (Gould et al., 1997) and in vitro (Hession et al., 1995). The activity and dynamics of the sulfate-reducing bacteria in the rumen have been studied less than other major groups of bacteria, such as the cellulolytics and methanogens. Cummings et al. (1995a) did not detect a change in numbers of ruminal sulfate-reducing bacteria as percentages of sulfur in the diet increased. However, after being exposed to high levels of sulfur, ruminal organisms did have a greater capacity to produce sulfide after 10 to 12 days. Oliveira et al. (1997) reported that high dietary sulfur resulted in a faster rate of sulfate reduction by ruminal bacteria after several weeks on that diet. Both reports are evidence that adaptive mechanisms for the increased activity by sulfate-reducing bacteria exists.

Added sulfur has improved ruminal fermentations, but only when the diet was deficient in this mineral. For example, Hegarty et al. (1994) reported improved dry matter digestion, increased total volatile fatty acids concentration, and more bacteria in the rumen of sheep fed a high versus a low sulfur diet (< 0.25%, dry matter basis). Patterson and Kung (1988) reported that added sulfur (0.3% of the dry matter) from methionine, methionine hydroxy analog, or sodium sulfate improved cellulose digestion threefold in in vitro fermentations that were void of sulfur. Moderately high percentages of sulfur (0.4 to 0.6%) in the diet have generally had no effects on ruminal volatile fatty acids and ammonia-nitrogen concentrations. However, the effect of extremely high percentages of sulfur (> 1% of the DM) in the diet of ruminants is equivocal. Kahlon et al. (1975) reported that 1.3% sulfur in the diet inhibited microbial protein synthesis in the rumen, but Kennedy et al. (1986) reported that a similar percentage of sulfur was not toxic to ruminal microorganisms. In calves, dietary sulfur as high as 1.72% had no effect on ruminal VFA or ammonia-nitrogen relative to calves consuming a diet with 0.34% sulfur (Slyter et al., 1988). Certainly, the biological availability of the sulfur source, ruminal pH, and interactions with dietary nutrients, such as divalent cations, may explain some of the conflicting results.

SULFUR TOXICITY

In monogastrics, sulfur is relatively inert and can therefore be tolerated at relatively high levels. However, in ruminants, the ingestion of large amounts of sulfur can lead to acute sulfur toxicosis and death. The immediate signs of distress include thrashing, kicking at stomach, staggering, and moaning followed by subsequent death within 48 hours suggesting a fairly high capacity to produce sulfide without the need for adaptation. High concentrations of sulfide in ruminal gas have been reported (McAllister et al., 1992) and have resulted in respiratory distress, reduced feed intake, and reduced ruminal motility (Bird, 1972).

Sulfide is readily absorbed through the rumen wall into the blood stream (Bray, 1969). Once absorbed, sulfide inhibits the functions of carbonic anhydrase, dopa oxidases, catalases, peroxidases, dehydrogenases, and dipeptidases, adversely affecting oxidative metabolism and the production of ATP (Short and Edwards, 1989). Specifically, sulfide is also thought to block the enzyme cytochrome c oxidase. Sulfide also binds to hemoglobin creating sulfhemoglobin, reducing the ability of the blood to carry oxygen to tissues. In addition, sulfide also has a paralyzing effect on the carotid body and therefore may also inhibit normal respiration (Bulgin et al., 1996).

Bulgin et al. (1996) recently reported acute reactions in response to increased levels of ingested elemental sulfur in sheep. These animals had grazed on an alfalfa field that had been sprayed with elemental sulfur (60 kg/ha). Within two hours after being released onto this field, some of the animals began to show signs of distress, and quickly died. Upon necropsy, it was noted that the rumen pH was 6 - 6.5, there was an odor of rotten eggs, lead acetate paper blackened when exposed to rumen contents, and pulmonary edema was observed. Immediate deaths were probably from acute sulfide toxicity.

ROLE OF THIAMINE IN OCCURRENCES OF PEM

Occurrences of PEM have been observed in animals which have access to plants containing high amounts of thiaminase, e.g. bracken fern (Merck, 1993), and in animals exhibiting thiamine deficiency (Gooneratne et al., 1989a; Olkowski et al., 1992). Lesions in affected brain tissue autofluoresce under UV light when prepared for histological observation. Clinical symptoms consist of blindness, head pressing, and circling, followed by recumbency, opisthonos, convulsions, and eventually death (Merck, 1993). Because thiamine is a necessary cofactor in the tri-carboxcylic acid cycle and the pentose shunt, lesions are seen in tissues of which these processes are vital to cell survival, in particular, the tissues of the brain and heart (Merck, 1993). An abrupt change in diet from forage to concentrates has also been suggested to affect thiamine status in ruminants (Merck, 1993). Levels of thiamine decrease because there is a shift in the rumen microflora. Gram positive bacilli, Gram negative cocci, and coccobacilli predominate, producing elevated levels of thiaminase type I activity. Thiaminase type I is deleterious for two reasons. First, it destroys thiamine and secondly, the actual destruction of thiamine produces a thiamine analog that inhibits the thiamine-dependent reactions of

glycolysis and the tri-carboxcylic acid cycle (Brent and Bartley, 1984). In each of the ATP - producing, catabolic pathways, thiamine is a necessary cofactor. This cofactor (thiamine pyrophosphate or TPP) is necessary for the enzymatic actions of the alpha-ketogluturate and pyruvate dehydrogenase complexes in the tri-carboxcylic acid cycle. Olkowski et al. (1993) reported on thiamine destroying activity of ruminal fluid but Oliveira et al. (1997) could not demonstrate a negative effect of a high concentrate diet on thiamine metabolism.

Administering thiamine has been used as a treatment for some cases of PEM and as a prophylactic agent against PEM (Merck, 1993; Low et al., 1996; and Olkowski et al., 1992). With the administration of thiamine other micronutrients should be considered. Gooneratne et al. (1989b) hypothesized that there is an interaction in the rumen between copper, sulfur, and thiamine. They suggested that, in the presence of excess sulfur, the addition of copper forms copper sulfate precipitates in the rumen, keeping the sulfur from maintaining its antagonistic relationship with thiamine. Olkowski et al. (1992) also suggests that elevated levels of thiamine are necessary to protect tissues from the clinical signs of sulfur toxicity, namely brain edema. In this scenario, thiamine would protect cells by decreasing the activity of the ATP-dependent sodium pump, and in this way maintaining osmolar balance.

HIGH SULFATE ASSOCIATED PEM

The National Research Council suggests that ruminants should not be fed more than 0.4% sulfur (DMB) to prevent reductions in intake (NRC, 1987). However, Bouchard and Conrads (1976) suggest that this level should not be higher than 0.26% for lactating cows. If high levels of sulfur inhibit intake, extreme caution should be taken during the close-up and early lactation stages of lactation where DM intake is lower than desired. Some common feeds and minerals that have moderate to high levels of sulfur are shown in Table 1. For example, corn gluten meal, molasses (cane and beet) and Brassicas (e.g., turnips) are high in sulfur. Other feeds worthy of mentioning that contain high concentrations of sulfur include fish, feather, meat and blood meals, that are common sources of rumen undegradable intake protein. Water can also be very high in sulfates with levels in excess of 5,000 ppm (Veenhuizen et al., 1992). Digesti and Weeth (1976) suggested that it was safe for cattle to consume water containing 2,500 ppm of sulfate. Recently, Wagner et al. (1998) reported lower intake and gains in steers fed water with 2,000 ppm sulfate (Table 2). Average daily gain was lower and feed efficiency was worse with increasing amounts of sulfate in water. Moderately high amounts of sulfur or sulfate consumption have resulted in reduced animal performance without acute symptoms of acute sulfur toxicity. For example, many years ago, in Cuba, cattle fed diets rich in molasses (and high in sulfur) developed symptoms of PEM and that were not responsive to thiamine. Feeds that are acidified with sulfuric acid as a preservative (H_2SO_4) and minerals (e.g. those used during balancing for DCAD balance during the dry period of dairy cows) also have high sulfur concentrations. One can easily envision a rather normal diet with alfalfa hay, beet pulp, distiller's grains and other feeds that would approach the upper limit of maximum sulfur intake. Coupled with a source of water with a high level of sulfate, this could easily lead to excessive sulfur consumption.

Symptoms of PEM have been induced in cattle consuming diets with 0.4% (Gould et al., 1991) sulfur but in some studies calves have been fed more than 1.5% sulfur without signs of toxicity (Slyter et al., 1986). In younger animals, development of rumen microflora and size of the rumen may affect responses to high sulfur.

Table 1. A listing of some feeds with moderate to high sulfur content.

Feed	International Feed Number	Sulfur, % dry matter basis
Alfalfa hay, early bloom	1-00-059	0.28
Barley malt sprouts, dehydrates	5-00-545	0.85
Beet pulp, w/molasses, dehydrated	4-00-672	0.42
Brewers grains, wet	5-02-142	0.32
Corn, distillers grains, dehydrated	5-28-237	0.46
Corn gluten meal, 60%	5-28-242	0.72
Molasses, beet	4-00-668	0.60
Molasses, cane	4-04-696	0.47
Rapeseed meal	5-03-871	1.25
Whey, dehydrated	4-01-182	1.12
Turnip, root	4-05-067	0.43
Ammonium sulfate	6-09-339	24.10
Calcium sulfate	6-01-089	18.62
Copper sulfate	6-01-720	12.84
Potassium sulfate	6-06-098	17.35
Sodium sulfate	6-04-292	9.95

Table 2. Effect of sulfate content in water on performance of steers.

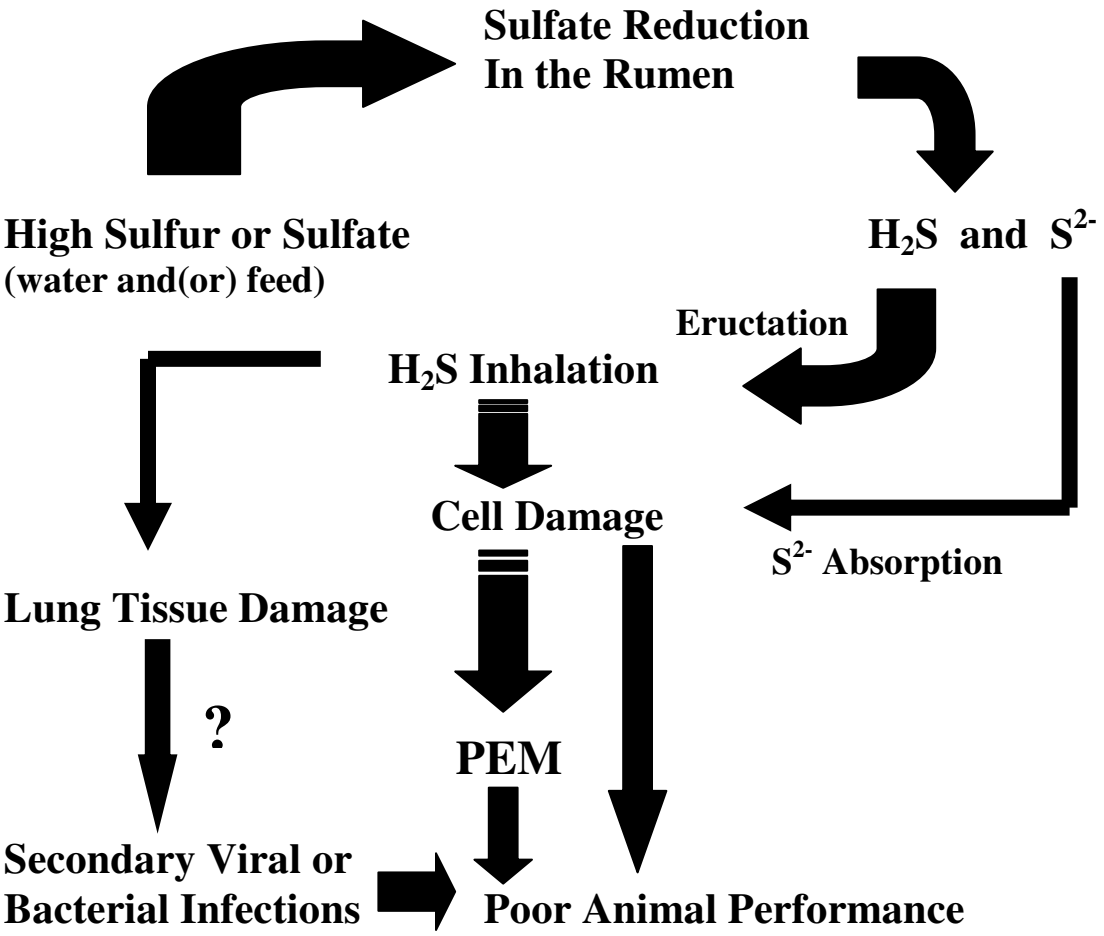
	Sulfate Concentration in Water (ppm)				
Item	125	250	500	1000	2000
DMI, kg/d	9.83	10.78	10.34	9.84	9.93
ADG, kg/d	2.15	2.11	2.14	2.10	2.04
Feed efficiency, DMI/gain	4.58	5.11	4.83	4.69	4.86
Water, liters/d	33.6	34.8	31.6	31.9	29.7

Wagner et al. 1997.

In ruminants, eructation (belching of gasses) is a normal process. However, as much as 60% of eructated gasses are inhaled and enters the respiratory tract (Bulgin et al., 1996). Thus, inhalation of H_2S from diets high in sulfate has been implicated as a potential cause of PEM in ruminants. Exposures to higher concentrations of hydrogen sulfide (200 to 500 ppm) has resulted in the sudden onset of hemorrhagic pulmonary

edema that often ends in death in humans (Green et al., 1991). Although little direct evidence exists, an association between inhaled H_2S and respiratory diseases in ruminants cannot be dismissed. Because H_2S is so toxic (Truong et al. (2006), damage to lung tissue could predispose animals to secondary bacterial or viral infections even if clinical symptoms of PEM do not exist. Some proposed mechanisms for high sulfate induced problems in ruminants are shown in Figure 1.

Figure 1. A proposed mechanism for high sulfate induced polioencephalomalacia.



Some controversy exists as to whether sulfide is the primary cause of sulfur-induce PEM because in some cases high levels of sulfur have been implicated in decreased levels of thiamine (Brent and Bartley, 1984). Animals fed a high sulfur diet were protected from clinical signs of PEM, while still exhibiting the clinical lesions of PEM (Olkowski et al., 1992). Excess sulfur may decrease the levels of thiamine, either through the direct action or through the stimulation of the production of thiaminase, or both. It has also been suggested that the transient sulfite that is produced during the reduction of sulfate to sulfide, could have a direct impact on the brain tissue itself

(Oliveria et al., 1996; Brent and Bartley, 1984; Olkowski et al., 1992). Sulfite-derived radicals have been postulated to cause lipid peroxidation and damage to biological membranes. Because of the high lipid content of the brain, and its inability to be efficiently repaired, it becomes apparent why lesions are first seen in this tissue (Olkowski et al., 1992). Brent and Bartley (1984) suggested that sulfite can cleave thiamine at the methylene bridge. However, Oliveria et al. (1996) proposed that sulfite is not a large contributor to thiamine destruction because sulfite is transitory and therefore it does not accumulate in the rumen. Olkowski et al. (1992) suggested though, that sulfite may be a significant contributor because the sulfite produced is absorbed, oxidized to sulfate, and then recycled back to the rumen, available to be reduced again.

Table 3. Some references to outbreaks of high sulfur associated PEM.

Citation	Symptoms	Diagnosis
Kul et al., 2006	256 cattle died or slaughtered with signs of PEM.	PEM in cattle consuming barley malt sprouts. Total S content in diet was 0.45%.
Loneragan et al., 1998	16 of 150 calves on ranch A and 30 of 4,000 calves on ranch B with clinical signs of PEM.	PEM in calves consuming feeds with high S: grass hay (0.33% S), Canada thistle (0.9% S), turnips (0.63% S) and rape (0.91% S).
McAllister et al., 1997	Steers in a feedlot with visual impairment and ataxia. During hot summer months the incidence of PEM was 0.88%.	PEM in cattle drinking water containing 2,200 to 2,800 ppm sulfate corresponding to about 0.67% sulfur intake.
Hill and Ebbett, 1997	26 of 99 grazing heifers with signs of ataxia, recumbency, and blindness.	PEM in heifers consuming Brassica oleracea that contained 0.85% sulfur.
Bulgin et al., 1996	700 of 2,200 ewes with signs of incoordination and abdominal discomfort, death.	Sulfur toxicity and PEM from field acidified with 35% suspension of elemental sulfur.
Low et al., 1996	21 of 71 lambs with depression, blindness, head pressing, and death	PEM. Lambs consumed a diet with 0.43% sulfur for 15 to 32 days before symptoms appeared.

Some recent cases of high sulfur associated PEM are shown in Table 1. In the majority of studies reported in Table 3, thiamine status was normal and when administered, supplemental thiamine did not always solve the problem. In a group of potential animals, the incidence of PEM is very low even when animals display clinical symptoms. Data in the literature would also suggest that when sulfur levels are moderately high, an adaptive process takes place so that PEM is not manifested until 2 to 4 weeks after the beginning of consumption of high levels of sulfur. Gould et al. (1997) reported on the use of rumenocentesis coupled with the analysis of rumen gas for H_2S and suggested that this may be a useful method for measuring pathological concentrations of H_2S. Under controlled conditions, researchers have smelled the odor of H_2S on the breath of cattle (Gould, personal communication).

INHIBITING SULFIDE PRODUCTION

Production of sulfides has deleterious effects in many biological and non-biological systems. For example, iron and steel structures are prone to corrosion in the presence of sulfides (Odom and Singleton, 1993). In the work place, the presence of hydrogen sulfide gas at low concentrations (50 - 200 ppm) is an irritant to the human respiratory tract, and at higher concentrations (200 - 500 ppm) it can cause hemorrhagic pulmonary edema that is often fatal (Green et al., 1991).

Broad - spectrum biocides such as hypochlorite (Odom and Singleton, 1993), methylenebis thiocyanate (Zhou and King, 1995) and gentamicin (Tanimoto et al., 1989) have been used to control sulfide production from sulfate-reducing bacteria in industrial situations. For ruminants, balancing dietary ingredients to ensure optimal amounts of sulfur in the diet can be easily done. However, there are few options to choose from when faced with a source of water high in sulfate. Treating water by means of reverse osmosis is an expensive proposition. Most, if not all, biocides would be impractical to use in ruminant diets because of their broad antimicrobial spectrums that would have negative impacts on ruminal fermentation. In addition, many of these compounds would be highly toxic to the animal.

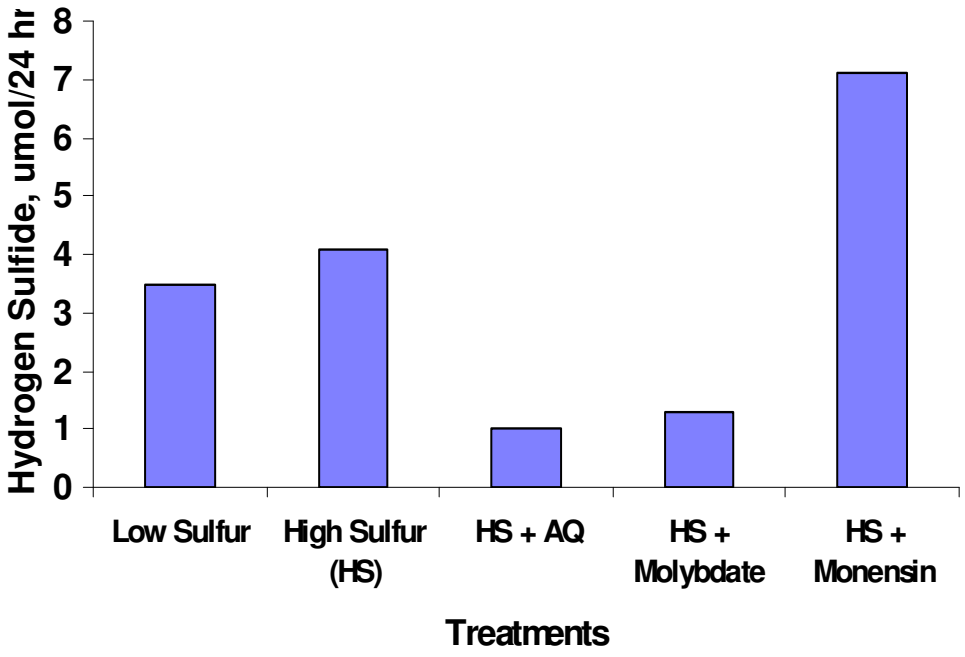
Rumen microbial populations have been manipulated in order to produce more desirable end products (e.g. VFA and microbial protein) and less undesirable end products (e.g. methane and hydrogen). The majority of non-ionophore antibiotics and ionophores administered to ruminants are effective against Gram-positive bacteria (Nagaraja, 1995). Since SRB are Gram-negative, we would not expect direct effects of these compounds on inhibiting sulfide production. However, indirect effects could occur. For example, one proposed mode of action of the ionophore monensin is that it selects against hydrogen and formate producing bacteria such as Ruminococcus albus, Ruminococcus flavefaciens, and Butyrivibrio fibrisolvens (Chen and Wolin, 1979) resulting in an indirect inhibition of methanogens that require hydrogen as a substrate. Selective targeting of sulfate-reducing bacteria may be a method to reduce excess sulfide production in the rumen.

In the literature, we have been able to identify only a few compounds that appear to be relatively specific for inhibiting sulfate-reducing bacteria that may be acceptable for use in ruminants. For example, molybdate (MoO_4) has been proposed as an analog of sulfate that blocks the sulfate activation step that is catalyzed by ATP sulfurylase (Oremland and Capone, 1988). Taylor and Oremland (1979) showed that MoO_4 specifically inhibits sulfate-reducing bacteria in pure culture and other investigations have also shown that MoO_4 inhibits sulfate-reducing bacteria in sediments (Oremland and Silverman, 1979; Sorenson, et al., 1981). However, MoO_4 may not be specific to inhibiting sulfate-reducing bacteria as Jones et al. (1982) demonstrated that MoO_4 inhibited methanogenesis when sulfate was limiting. In contrast, Westerman and Ahring (1987) demonstrated that low levels of MoO_4 (1 mM) slightly stimulated methane production. In light of these conflicting results, some have recommended caution in using MoO_4 in ecological situations (Banat, et al., 1981; Jones, et al., 1982; Jacobson, et al., 1987). Odom and Singleton (1993) also suggested that although MoO_4 was a useful research tool, its use as a commercial biocide was impractical because of the potential negative ecological impact. We have shown that molybdate (> 10 ppm of the fluid) can reduce H_2S production in ruminal fermentations (Bracht and Kung, 1997). At concentrations that we tested (maximum of 25 ppm of the fluid) we observed no effect of molybdate on rumen VFA, methane or hydrogen. This amount of MoO_4, caused a depression in both the liquid and gas sulfide and a slight decrease in total VFA, but no changes in any other culture conditions. Under our conditions, MoO_4 appeared to be a specific inhibitor of sulfate-reducing bacteria because we found no effect on methane or hydrogen production. In ruminants, molybdenum is a trace mineral with a very narrow margin between the amount needed to fulfill the animal's requirements and toxic levels. Underwood (1981) reported that in cattle, molybdenum is toxic in the range of 20-100 ppm on a dry matter basis. However, Huber et al. (1971) reported that lactating dairy cows showed no signs of toxicity when fed a diet containing 100 ppm of molybdenum (1.7 g/d) for 6 months but toxicity occurred when cows were fed 200 ppm molybdenum. Intake of 1.7 g of molybdenum in a 625-kg cow with an 85-liter rumen would equate to a rumen concentration of 20 ppm, which is similar to the amount of MoO_4 used in our study. Recently, Loneragan et al. (1998) reported that sodium molybdate was capable of reducing H_2S concentrations in the gas cap of cattle fed a high sulfur diet, but the effect was not consistent for all cattle, and liver stores of Cu decreased dramatically.

The compound 9,10 anthraquinone (AQ) was first reported by our lab as a methane inhibitor in in vitro ruminal fermentations (Garcia-Lopez et al., 1996). We subsequently have reported the ability of AQ to also inhibit sulfate reduction in the rumen (Hession et al., 1995; Kung et al., 1996; Bracht and Kung, 1997, Kung et al., 1998). In Figure 2, addition of 10 ppm (fluid basis) of AQ reduced sulfide production in a diet containing 1.09% sulfur to levels below that found in a diet with only 0.29% S. Cooling et al. (1996) reported inhibition of sulfide production using 9,10 anthraquinone due to possible uncoupling of the electron transport chain. Decreased levels of ATP results in insufficient energy, which is needed for subsequent activation of sulfate for further sulfate reduction. Cooling et al. (1996) proposed that the uncoupling is due to the redox capabilities of anthraquinones. In our in vitro studies, we surprisingly have found that monensin stimulated sulfide production in in vitro ruminal fermentations (Figure 2). The

reasons for this finding are unknown but indirect inhibition of methanogens by monensin may decrease competition between methanogens and sulfate-reducing bacteria because both classes of organisms can utilize some common substrates such as acetate and H_2. This finding may have far reaching implications because monensin is widely used in the feedlot. In vivo studies are needed to verify our initial findings.

Figure 2. Effect of high sulfur and various compounds on in vitro ruminal hydrogen sulfide production. Low sulfur = 0.29% S; High sulfur = 1.09% S. AQ = 10 ppm (fluid basis) of 9,10 anthraquinone; Molybdate = 25 ppm of molybdate; Monensin = 5 ppm. (Data from Bracht and Kung, 1997.)



In a recent publication, McAllister et al. (1997) reported that the incidence of PEM cases in a feedlot in Colorado was seasonal and related to days in the feedlot. The incidence of PEM peaked between 15-30 d after cattle had entered the feedlot and during summer months. Several factors could have contributed to these findings. First, increased consumption of water, high in sulfate, during hot summer months, coupled with increasing adaptation to high sulfur intake by sulfate-reducing bacteria in the rumen probably increased levels of H_2S in rumen of these cattle. Furthermore, the proportion of concentrate in the diet of incoming cattle was probably increased during the first 4 weeks in the feedlot. As the proportion of concentrate in the diet increased, rumen pH would decrease resulting in a greater proportion of sulfide to be protonated since the pka for H_2S is 7.2. In addition, we hypothesize that another contributing factor to high levels of H_2S production could be the fact that intake of monensin is also gradually increased during

the first several weeks in the study. In our studies (Bracht and Kung, 1997), adding monensin to in vitro ruminal fermentations exacerbated H_2S production.

CONCLUSIONS

Many incidences of high sulfate-associated PEM that were independent of thiamine metabolism in ruminants have been reported in the last several years. Many common feeds and sources of water can contain high levels of sulfur/sulfate. Producers should be aware of all possible sources of sulfur in the diet. Although the incidence of clinical PEM is generally low, subtle decreases in intake and possible associations with respiratory diseases could be decreasing animal performance. More research is needed to study the association between high levels of sulfur in the diet and reduced animal performance in ruminants and to develop strategies to combat high sulfate-induced PEM.

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