ABSTRACT

This review deals with the toxicology of sulfur in ruminants including toxicity, neurotoxic effects, and mechanism of toxic action of hydrogen sulfide, clinical signs, and treatment. It will report effects of excessive intake of sulfur by ruminants on feed intake, animal performance, ruminal digestion and motility, rumination, and other physiological functions. Poisoning of animals with sulfur from industrial emissions (sulfur dioxide) also is discussed. Excessive quantities of dietary sulfur (above .3 to .4%) as sulfate or elemental sulfur may cause toxic effects and in extreme cases can be fatal. The means is discussed whereby consumption of excessive amounts of sulfur leads to toxic effects.

INTRODUCTION

Most of dietary sulfur, whether as amino acid or inorganic sulfur, ingested by the ruminant is reduced to sulfide by ruminal microorganisms and then either incorporated into microbial protein or absorbed and oxidized to sulfate in liver (2). The generation of large quantities of hydrogen sulfide in rumen may depress ruminal motility and cause severe distress of nervous and respiratory systems (20). When sulfur supplements are given to increase the supply of protein and energy to the ruminant tissues, the possibility of toxic effects of excessive intakes of sulfur must be considered.

It has been reported that dietary sulfur, if consumed in large quantities, can be toxic. For example, high contents of sulfur as elemental sulfur (1, 18, 44, 49), calcium sulfate (3, 10, 17, 30), or sodium sulfate (1, 30, 41) have decreased feed intake and reduced performance of ruminants. These compounds have been used to regulate intake of feed supplements, but clinical signs have been reported only with use of elemental sulfur (18, 53). Julian and Harrison (31) reported a case of sulfur poisoning in a group of young cattle given sulfur orally by their owner as a treatment for ringworm.

Single or continuous ruminal infusions of high amounts of sulfur as sulfate or sulfide caused harmful effects (7). Ruminal (21, 45) and abomasal (39, 45) infusions of excessive amounts of methionine may affect adversely feed consumption by ruminants. In addition, abomasal infusions of large quantities of methionine may result in accumulation of large amounts of methionine in plasma and frequently depress wool growth (42).

Little is known of the effect of high sulfur concentrations in the sole drinking water supply of ruminants. Digesti and Weeth (19) found that ingestion of inorganic sulfate in drinking water produced quantitative changes of blood composition and renal function and suggested a safe tolerance limit for inorganic sulfate concentration in drinking water of cattle. However, in practice, it is not feasible to set an exact safe tolerance concentration for sulfate in water as tolerance is dependent on total intake and turnover rate of sulfate in the individual animal.

Results of in vitro studies reported by Hubbert et al. (27) indicated that cellulose digestion by ruminal microorganisms was depressed by high sulfate concentrations of medium. In a similar study (34), no significant toxicity was observed of excess sulfur from sodium sulfate on starch digestion. This suggests that amylolytic microorganisms are more tolerant to variations of concentrations of sulfur than are cellulolytic microorganisms.

Recently, poisoning of animals with sulfur from environmental pollution has been reported (14, 26, 29). Grazing animals exposed to industrial emissions may become poisoned by sulfur dioxide. In addition, sudden exposure to
<table>
<thead>
<tr>
<th>Animal</th>
<th>Method of administration</th>
<th>Form of sulfur</th>
<th>Amount of sulfur</th>
<th>Signs of toxicity</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Elemental S</td>
<td>315 g/day</td>
<td>Muscle twitching, abdominal pain, fast labored breathing, diarrhea, death</td>
<td>(18)</td>
</tr>
<tr>
<td>Sheep</td>
<td>Diet</td>
<td>Elemental S</td>
<td>15 g/day</td>
<td>Strong odor of sulfide on breath, black diarrhea, passage of blood from rectum, death</td>
<td>(53)</td>
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<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Elemental S</td>
<td>270 g/day</td>
<td>Respiratory distress, diarrhea, vasculitis and necrosis of rumen and abomasal wall, twelve of the twenty animals died</td>
<td>(31)</td>
</tr>
<tr>
<td>Sheep</td>
<td>Diet</td>
<td>Elemental S or sodium sulfate</td>
<td>&gt; .4% of DM^a</td>
<td>Reduced animal performance</td>
<td>(1)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Elemental S</td>
<td>up to 1.72% of DM^a</td>
<td>No toxic effects</td>
<td>(16)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Elemental S</td>
<td>N:S = 5:1</td>
<td>Reduced feedlot gain</td>
<td>(49)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Elemental S</td>
<td>9.8 g/kg of diet</td>
<td>Reduced feed intake and weight</td>
<td>(44)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Calcium sulfate</td>
<td>.3% or more of DM^a</td>
<td>Reduced feed intake</td>
<td>(10)</td>
</tr>
<tr>
<td>Sheep</td>
<td>Diet</td>
<td>Calcium or sodium sulfate</td>
<td>.5% of DM^a</td>
<td>Reduced feed intake and growth rate, adversely influenced feed conversion</td>
<td>(30)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Sodium, calcium, potassium, and magnesium sulfate</td>
<td>.35% or more of DM^a</td>
<td>Reduced dry matter intake</td>
<td>(11)</td>
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<td>Cattle</td>
<td>Diet</td>
<td>Sodium sulfate</td>
<td>&gt; 2% of DM^a</td>
<td>Polioencephalomalacia</td>
<td>(41)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Sulfate in drinking water</td>
<td>3000 ppm</td>
<td>Reduced hay intake</td>
<td>(51)</td>
</tr>
<tr>
<td>Cattle</td>
<td>Diet</td>
<td>Sulfate in drinking water</td>
<td>5000 ppm</td>
<td>Lower feed and water consumption, weight loss, diuresis</td>
<td>(50)</td>
</tr>
<tr>
<td>Sheep</td>
<td>Diet</td>
<td>Sodium metabisulfite</td>
<td>2.25 g/kg of body weight</td>
<td>Restlessness, feed refusal, ruminal atony, rapid pulse and respiration, cyanosis, death</td>
<td>(38)</td>
</tr>
<tr>
<td>Sheep</td>
<td>Diet</td>
<td>Methionine hydroxy analog</td>
<td>1.2% of DM^a</td>
<td>Reduced feed consumption</td>
<td>(39)</td>
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<td>Sheep</td>
<td>Single ruminal infusion</td>
<td>Sodium sulfate</td>
<td>7.4 g/day</td>
<td>No toxic effects</td>
<td>(33)</td>
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<tr>
<td>Sheep</td>
<td>Single ruminal infusion</td>
<td>Sodium sulfide</td>
<td>.94 g/day</td>
<td>Respiratory distress and collapse</td>
<td>(7)</td>
</tr>
<tr>
<td>Animal</td>
<td>Infusion Type</td>
<td>Substance</td>
<td>Rate</td>
<td>Effect</td>
<td></td>
</tr>
<tr>
<td>------------</td>
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<td>------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Sheep</td>
<td>Single ruminal infusion</td>
<td>Sodium sulfide</td>
<td>1.01 g/day</td>
<td>Odor of sulfide on the animal's breath, one animal became unsteady on its feet</td>
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<tr>
<td>Sheep</td>
<td>Continuous ruminal infusion</td>
<td>Sodium sulfate</td>
<td>5-6 g/day</td>
<td>Feed and water refusal</td>
<td></td>
</tr>
<tr>
<td>Sheep</td>
<td>Continuous ruminal infusion</td>
<td>Sodium sulfate</td>
<td>6 g/day</td>
<td>Complete inappetence</td>
<td></td>
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<tr>
<td>Sheep</td>
<td>Continuous ruminal infusion</td>
<td>Sodium sulfate</td>
<td>10 g/day</td>
<td>No toxic effects</td>
<td></td>
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<tr>
<td>Sheep</td>
<td>Continuous ruminal infusion</td>
<td>Sodium sulfide</td>
<td>2.93 g/day</td>
<td>Reduced dry matter intake and ruminal motility</td>
<td></td>
</tr>
<tr>
<td>Sheep</td>
<td>Continuous ruminal infusion</td>
<td>DL-methionine</td>
<td>30 g/day</td>
<td>Acute nephrosis, hemolytic anaemia, one animal died</td>
<td></td>
</tr>
<tr>
<td>Cattle</td>
<td>Continuous ruminal infusion</td>
<td>DL-methionine</td>
<td>2.5% or more of DM/1% of DM</td>
<td>Reduced dry matter intake</td>
<td></td>
</tr>
<tr>
<td>Cattle</td>
<td>Continuous ruminal infusion</td>
<td>Methionine hydroxy analog</td>
<td>&gt;.6% of DM</td>
<td>Reduced feed intake</td>
<td></td>
</tr>
<tr>
<td>Cattle</td>
<td>Continuous abomasal infusion</td>
<td>DL-methionine</td>
<td>2.7, 5.4, and 8.1 g/day</td>
<td>Reduced feed intake</td>
<td></td>
</tr>
<tr>
<td>Sheep</td>
<td>Continuous abomasal infusion</td>
<td>DL-methionine</td>
<td>7.4-9.8 g/day</td>
<td>Reduction of wool growth rate</td>
<td></td>
</tr>
<tr>
<td>Sheep</td>
<td>Single intraperitoneal injection</td>
<td>Methionine hydroxy analog</td>
<td>28 g/day</td>
<td>Depressed live weight, three of the five animals died</td>
<td></td>
</tr>
<tr>
<td>Cattle</td>
<td>Environmental pollution</td>
<td>Sulfur dust, sulfur dioxide, and hydrogen sulfide</td>
<td>Animals within 1 km of a sulfur mine</td>
<td>Lesions of respiratory and digestive systems</td>
<td></td>
</tr>
</tbody>
</table>

* Dietary dry matter intake.
hydrogen sulfide emitted from a slurry tank, especially when the tank is agitated, may be quickly fatal (14).

SULFUR INTAKE AND ANIMAL PERFORMANCE

Albert et al. (1) reported that high contents of sulfur (above .4%) as elemental sulfur or sodium sulfate reduced performance of lambs, but some researchers have observed no harmful effects from feeding relatively high sulfur. For example, Chalupa et al. (16) fed elemental sulfur up to 1.72% in a purified diet to Holstein calves with no observable ill effects. In later studies (15), neither sodium sulfate nor elemental sulfur added to the diet of Angus steers to provide sulfur up to .62% and .56% sulfur, respectively, caused deleterious effects in the cattle. Thompson et al. (49) reported reduced feedlot gain for steers supplemented with elemental sulfur to produce a ratio of 5:1 nitrogen:sulfur. Lower intakes of dry matter were recorded for dairy cows when sulfur was .35% or above (11). Rumsey (44) showed that the addition of 9.8 g of sublimed sulfur/kg of diet markedly reduced feed intake and weight of steers fed high concentrate diets.

Quantities of calcium sulfate (gypsum) reduce feed intake of ruminants (17). This principle has been used as an effective means for regulating intake of pasture supplements for livestock. Barrentine and Ruffin (3) reported use of gypsum in this manner, but they found that cattle became sick after consumption of large amounts of sulfate. Johnson et al. (30) found that addition of .5% sulfur (as calcium or sodium sulfate) reduced growth rate of lambs, and both forms of sulfate depressed feed intake and adversely influenced feed conversion. In addition, Bouchard and Conrad (10) reported reduced feed intake by dairy cows when calcium sulfate was used to increase dietary sulfur to .3% or more. They found excessive retention of sulfur by cows for all sources (calcium, sodium, potassium, and magnesium sulfates) or sulfate tested at concentrations above .3% dietary sulfur.

Bird (7) found that ruminal infusion of 6 g sulfur as sodium sulfate per day resulted in complete inappetence; 4 g sulfate sulfur/day infused intraruminally or up to 6 g given duodenally did not affect adversely the sheep. No toxic effects of sulfur were reported by Gawthorne and Nader (24) where 10 g of sodium sulfate/day were infused continuously into rumens of sheep. However, Raisbeck (41) reported an episode of cerebrocortical necrosis (polioencephalomalacia) in cattle fed high-sulfate (>2% sulfate) rations. In a recent experiment (33), we observed no toxic effects after administration of 7.4 g of $[^{35}S]$ sodium sulfate into the rumen of sheep (2.4 g total sulfur in ration plus 5 g carrier). Bird (7) stated that “intake by sheep of not more than 4 g sulphur/day, or the addition to ruminant diets of .2% sulphate sulphur or S-amino acid sulphur, should satisfy microbial and tissue sulphur requirements without adversely affecting feed intake.” (p. 1087). Generally the various sulfur compounds lead to similar toxic effects (see Table 1).

Lower feed and water consumption, weight loss, and diuresis were reported in growing heifers offered water containing 5000 ppm sulfate (50). Water intake was not affected by added 1462 or 2814 ppm sulfate to drinking water, but hay intake was depressed at the higher concentration (51), and rate of gain was decreased by both. In a later study Digesti and Weeth (19) concluded that in heifers 2500 ppm sulfate in drinking water represents a safe tolerance concentration.

Sulfide toxicosis, however, apparently has not been associated with feeding to ruminants of supplemental sulfate (12) or with high sulfates in drinking waters (40). During an investigation into the fate of sulfate infused into the rumen or duodenum, sheep receiving a continuous ruminal infusion of 5 to 6 g/day of sulfate sulfur eventually refused to eat or drink (9). Bird (7) suggested that because duodenal infusions of sulfate do not affect intake, and in view of the presence of dissimilatory sulfate-reducing bacteria in the rumen (28) and their ability to adapt rapidly to sulfate infusions (8, 9, 36), adverse effects of sulfate infusion noted by Bird and Moir (9) were attributed to sulfide toxicity.

Dougherty et al. (20) suggested that generation of large quantities of hydrogen sulfide in the rumen depresses ruminal motility and causes severe nervous and respiratory distress if it is absorbed into the lungs during eructation. Bird (7) showed that single ruminal infusions of sulfide (.94 g sulfur) in solution (Na$_2$S – H$_2$S) resulted in temporary respiratory distress and
collapse of sheep; ruminal motility was abolished temporarily, but smaller doses resulted in a moderate transitory depression of motility. In the same experiment (7), continuous ruminal infusion of sulfide (2.93 g sulfur/day) solution (sodium sulfide in water) resulted in significant decrease of dry matter intake. Rumination was decreased but independently of reduced feed intake; ruminal motility also was decreased, but this was at least partly a result of decreased intake. Bray (13) found that in one experiment where 1.01 g of sulfur as sodium sulfide (supplied as 50 ml of aqueous solution) was infused into the rumen, hydrogen sulfide could be smelled on the animal's breath, and one sheep became unsteady on its feet and fell to its knees 17 min after dosing, but 10 min later it had regained its feet and had recovered fully. In another experiment, where .4 g of sulfur as sodium sulfide was given, there were no observable effects of sulfide on the sheep.

The minimum lethal dose of sodium metabisulfite in sheep was 2.25 g/kg of body weight (38); signs of toxicity were restlessness, feed refusal, ruminal atony, rapid pulse and respiration, cyanosis, and death. Weigand et al. (52) observed no adverse effect on milk yield, milk fat, health of physiological measures in dairy cows fed 2 kg dried beet pellets daily where sodium metabisulfite was added to the pellets to give a sulfite content of 10.2 g/kg. In a similar study, Kaemmerer et al. (32) reported no harmful effects in sheep fed dried beet pulp containing 1% SO₂ as sodium disulfite in a ration of 500 g hay, 500 g dried beet pulp, and 200 g oatmeal.

A detailed review of amino acid toxicities including physiological, metabolic, and biochemical effects associated with the toxicity in nonruminant animals has been made by Harper et al. (25) and Benevenga (5). Benevenga (5) reported that consumption of high methionine (2% or more) resulted in growth depression and tissue damage. However, the means by which excessive consumption of methionine results in the tissue damage are still to be determined.

Excess of methionine or its hydroxy analog can affect adversely feed consumption in ruminants. Dietary supplements, ruminal and abomasal infusions of these compounds, can decrease dry matter intake by ruminants. Thus, in cattle, intraruminal infusion of DL-methionine, at 2.5% or more of dietary dry matter intake, resulted in a toxic effect on feed intake (45). Papas et al. (39) reported that feed consumption by lambs was decreased from 1000 g to 786 g/day when methionine hydroxy analog was included in the diet at 1.2%. Further abomasal infusion of DL-methionine (0, 2.7, 5.4, and 8.1 g/day) resulted in a linear reduction of feed intake from initial intake, whereas methionine hydroxy analog had no effect. Differences between DL-methionine and methionine hydroxy analog may be due, on the one hand, to greater resistance of methionine hydroxy analog to microbial destruction in the rumen (4) and, on the other hand, to a low replacement value of methionine hydroxy analog for methionine in the tissues (39). Emery (22) also reported that DL-methionine, administered as single 50-g doses intraruminally to mature cows, had a mean half-time of disappearance of 2.4 h and concluded that all forms of methionine tested persisted in the rumen for substantial time.

Feed intake of cattle was decreased with abomasal infusion of .6% DL-methionine (45) and also of sheep with .3, .5, and .9% (39). Abomasal infusions of 7.4 to 9.8 g/day of DL-methionine caused a substantial reduction of wool growth rate in sheep as compared to infusions of .6 to 2 g/day (42). Langlands (35) found that a single intraperitoneal injection of 28 g of methionine hydroxy analog depressed live weight and resulted in the death of three of the five sheep injected.

Excessive amounts of methionine fed to monogastric animals (47) or infused into the abomasum of sheep (43) substantially increased plasma methionine concentrations and altered the plasma amino acid pattern. Neither oral supplements of 12.3 g/day methionine hydroxy analog (39) nor 10 g/day DL-methionine (43) given to sheep influenced plasma methionine concentrations or plasma amino acid patterns.

Recently, Doyle and Adams (21) investigated toxic effects of large amounts of DL-methionine infused into the rumen of sheep. The sheep fed for ad libitum intake on a roughage diet of equal parts of chopped lucerne hay and oat chaff, received continuous infusions of DL-methionine into the rumen; the daily dose increased from 0 (control treatment) up to 30 g in 3-g increments at weekly intervals. They found that dry matter intake fell below control when 24 g/day or more of the amino acid was infused, whereas plasma free methionine concentration and plasma methe...
increased substantially when 30 g/day was given; there was no effect of DL-methionine supplementation on body weight of the animals. However, when 30 g/day of DL-methionine was infused, one animal died, and the acute condition of two others necessitated their slaughter. Significant lesions included acute nephrosis and hemolytic anemia with mild changes of liver and pancreas.

Tao et al. (48) found that adult sheep receiving total nutriment by dual infusions (volatile fatty acids infused intraruminally, and glucose, vitamins, and amino acids via jugular cannula) and supplement of L-methionine in excess of 3.6 g/day had elevated plasma methionine concentrations. Valine, isoleucine, leucine, and threonine in blood plasma were decreased by L-methionine supplement, but nonessential amino acids, including glycine and serine, were increased. Toxic amounts of methionine caused lower plasma concentrations of glycine and serine in rats (6) and sheep (46). Tao et al. (48) suggested that their experimental design did not allow adaptation to the particular treatments and, thus, resulted in divergent results.

Animals confined to industrial areas, near smelters, may become poisoned by sulfur dioxide as may animals confined near power plants or factories burning large amounts of coal, particularly if it is a high-sulfur coal and antipollution controls are lax. Exposure of grazing animals to a sulfur dioxide concentration of 500 ppm for 1 h is dangerous (26). According to Buck et al. (14), forage containing 1700 to 2500 ppm sulfuric acid (formed from sulfur dioxide on contact with water) is toxic for grazing livestock. Hatch (26) also reported that in animals, sudden exposure to H₂S concentrations of 400 ppm (such concentrations can occur in barns or stables exposed to the gasses emitted from a slurry tank) may be quickly fatal. Recently, Janowski and Chmielewice (29) reported a case of poisoning of cattle with sulfur from environmental pollution. Cattle within 1 km of a sulfur mine were exposed to sulfur dust, sulfur dioxide, and hydrogen sulfide. Poisoning was associated with lesions of respiratory and digestive systems.

SULFUR INTAKE AND RUMEN DIGESTION

The possibility that diets rich in sulfate might exert toxic effects in the rumen of sheep also was considered by Lewis (36). Thus, when a dose of 150 g Na₂SO₄·10 H₂O was given, no toxic effects were observed even though the concentration of sulfide in the rumen reached 14.7 µmoles/ml ruminal liquor. For normal and maximum concentrations of sulfate in the dry matter of the diet of .3 and 1% sulfate-S, respectively, and a daily intake of 1000 g dry matter, this is equivalent to an intake of 30 and 100 g, respectively, of Na₂SO₄·10 H₂O. Therefore, at this range, Lewis (36) suggested that no gross abnormality or toxicity would be expected.

Hubbert et al. (27) reported that cellulose digestion by ruminal microorganisms in vitro was depressed slightly by 1 mg sulfate sulfur/ml of medium. However, in later studies, Kennedy et al. (34) found no depression of starch digestion by ruminal microorganisms in vitro when sulfur up to 11 mg/ml was added (as sodium sulfate), and no diminution of growth in vitro by ruminal microbes was observed in studies by Bird (7) when sulfide was up to 1.5 mg/ml.

MECHANISM OF TOXIC ACTION

In the ruminant the neurotoxic effects of sulfide apparently are mediated via eructation of hydrogen sulfide, with other gasses, from the rumen and absorption through the lungs (7). Most of the eructated ruminal gasses enter the lungs, and infusion of hydrogen sulfide into the rumen of sheep with blocked trachea produced no clinical signs, whereas sheep with open trachea collapsed after several eructations (20). Sulfide absorbed from the rumen may be detoxified by oxygenated hemoglobin in the blood; in vivo reduction of oxyhemoglobin is reversible (23). The liver itself is also capable of detoxifying sulfide through the action of a sulfide oxidase system (2). Hence, it is unlikely that much free sulfide would reach the brain after being absorbed from the rumen into the portal system (7). Bird (7) stated that “the direct and shorter route to the heart and brain afforded by the inspiration of hydrogen sulphide and transfer into the pulmonary vein effectively by-passes the liver and enables hydrogen sulphide to exert its toxic effect on the respiratory-circulatory systems.” (p. 1095). Evans (23) concluded that spinal vasomotor centers were affected in the same manner by hydrogen sulfide as were medullary centers.
CLINICAL SIGNS OF EXCESS SULFUR

Sulfur toxicity was reported in a group of 20 yearling heifers that had consumed 5.45 kg of sulfur mixed with ground corn (31). Animals showed severe watery diarrhea, respiratory distress, and abdominal pain. Histological examination of one animal revealed vasculitis and necrosis of the rumen and abomasal wall. Twelve animals died over 9 days, making total mortality 60%. Julian and Harrison (31) suggested that toxic effects of sulfur in these animals and those described in (18) were likely due to combined activity of several products that may be produced in the digestive tract: a) hydrogen sulfide gas, b) hydrogen sulfide in solution, c) sulfur dioxide gas, and d) pentathionic acid. Thus, the acute deaths were likely due to action of hydrogen sulfide on respiratory or central nervous systems following inhalation or absorption of hydrogen sulfide gas from the rumen or possibly to absorption of hydrogen sulfide and production of sulfhemoglobinemia. Later mortality may have been due to dehydration from diarrhea. Damage to the digestive tract and severe colic may have been caused by sulfur dioxide (sulfur dioxide gas produces sulfurous acid when dissolved in water, such as on moist mucous membranes) or pentathionic acid, and this may have been involved in the death of animals that survived more than 2 or 3 days.

Clinical signs from sulfur toxicosis have been reported with the use of elemental sulfur in cattle (18) and ewes (53). Coghlin (18) reported that the signs in the early stages of hydrogen sulfide poisoning in cattle included twitching of certain groups of muscles especially those of the jaws, eyelids, and ears. There was evidence of pain as indicated by lying down and getting up, switching, and uneasiness; a staggery gait also was noticed. During the morning there was no evidence of diarrhea, but by evening most of the affected cattle had severe diarrhea. In advanced stages, cattle became unable to rise, acted as if blind, and struggled a great deal, with grunting and fast labored breathing. Finally they became comatose with their heads lying on the side; this was followed by death. During postmortem examination lungs were dark, congested, and edematous; the liver was very light, presenting a boiled appearance; the spleen was normal in color except for some light spots on its surface; stomachs showed little evidence of inflammation, but intestines were acutely inflamed; some of the muscles over the loin and back were almost black. Likewise, in a similar report by White (53) of sulfur poisoning in ewes, those showing clinical signs were depressed, unwilling to stand, breathing heavily, and in some cases had temperature up to 40°C. The breath smelled of H₂S, and ewes excreted black diarrhea with some occult blood, which in some ewes was followed by death. Postmortem examinations revealed intense inflammation of abomasum and small intestine with a large quantity of peritoneal effusion, postmortem fluid containing large fibrinous clots; kidneys were acutely inflamed and appeared nearly black in some cases; petechial hemorrhages were marked throughout carcass, on heart, lungs, pleura, and even in the musculature on the inside of the scapula when the shoulder was removed.

Excessive dietary sulfur may cause acute toxicity resulting in signs of sickness such as abdominal pain, muscle twitching, diarrhea, severe dehydration, strong odor of sulfide on the breath, congested lungs, and acute enteritis (37). Buck et al. (14) reported that acute exposure to 5 ppm sulfur dioxide caused irritation of mucous membranes, whereas pulmonary hemorrhage and emphysema can occur within a day after 8-h exposure to 40 ppm sulfur dioxide.

TREATMENT

Hydrogen sulfide poisoning in ruminants can be treated successfully either with glucose saline, followed with glycerine by mouth and bismuth carbonate, or with bismuth carbonate mixed into the feed in the form of cubes for those animals willing to eat (53). This treatment limits absorption of sulfide from the rumen by reducing the absorption rate constant by increasing rumen pH. In White's report (53) all ailing ewes were drenched daily with saline and 140 liters of glycerine. In addition, hydrogen sulfide poisoning in cattle has been treated with intramuscular injection of camphor in oil or calcium gluconate solution intravenously (18). Coghlin (18) recommended as a chemical antidote for sulfuretted hydrogen the following mixture: 31.1 g of iron sulfate and 15.6 g of liquor ammonia fortis, dissolved in 1.135 liters of warm water. The mixture was prepared extemporaneously and given as one dose.
CONCLUSION

Consumption by ruminants of high percents of sulfur (above .3 to .4%) as sulfate sulfur or elemental sulfur may cause toxic effects. Animals also may become poisoned by sulfur from environmental pollution when exposed to sulfur dust, sulfur dioxide, and hydrogen sulfide. No reports of tissue damage in ruminants associated with large amounts of methionine given orally or infused into the rumen. Accumulants associated with large amounts of methionine hydroxy analog. Can. J. Anim. Sci. 54:587.

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