

The **S**ilent element

16 Sulphur

Sulfur (S) is an essential nutrient for ruminants, required for normal growth and development of both rumen micro-organisms and the host animal. Deficiency of S can lead to problems ranging from poor performance to death, and excess S can equally harm the animals. The toxicity of S-compounds leads to alterations in the rumen environment, neurological disturbances and even death.

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Essentiality of S

S is a constituent of several amino acids such as cysteine, cystine and methionine. It is also present in other S-containing biomolecules such as biotin, taurine, co-enzyme-A, fibrinogen, heparin, thiamine and glutathione, playing an important role in cartilage integrity. S is especially essential for wool-producing animals as the fur is rich in S-containing amino acids, and its deficiency is associated with decreased quantity and quality of the fibres. It is interesting to note that the utilisation of non-protein nitrogen (NPN), such as urea, is dependent on sufficient levels of sulfur in the rumen fluid. The nitrogen:sulphur (N:S) ratio of protein in mixed ruminal bacteria is not constant, but a 20:1 ratio between available

nitrogen and sulphur should be adequate to supply the requirements of the rumen microbes. A marginal deficiency of S has an impact on microbial synthesis and fibre degradation in the rumen leading to reduced dry matter intake (DMI), growth rate or milk production. Severe deficiency symptoms are excessive salivation, weight loss, dullness and death. The maximum tolerable levels of S vary according to concentrate levels in the diet. According to the NRC (2005), 3,000 ppm S is the limit for lactating cows and ruminants fed high concentrate levels, and 4,000 ppm is the limit for ruminants fed high forage diets.

The importance of assessing total S supply

Ruminal bacteria can metabolise elemental, inorganic and organic S; therefore, the total content has to be considered for assessing dietary S intake levels. *Table 1* shows the S content of some feedstuffs used in ruminant nutrition. Water can also be a source of S in some regions and can become problematic if not taken into consideration.

S toxicity and impact on DCAD

In the rumen, S sources serve as a substrate to some bacteria that will convert it into sulfide to be further incorporated into microbial and animal metabolism through S-aminoacids or as sulfide. The excess of sulfur in the diet leads to an accumulation of sulfides in the rumen environment, increasing the risk of acute toxic and polioencephalomalacia (PEM) in those animals. It is worth mentioning that sulfur is an important anion and can reduce the dietary cation-anion difference (DCAD). In lactation rations, DCAD between +25 to +30 meq/100g of dietary DM is required to achieve maximum feed intake and milk yield. Reduced DCAD not only compromises the DMI and milk production but also decreases the neutral detergent fibre (NDF) digestibility.

Formation of toxic gases by rumen bacteria

Sulfates and organic S in the rumen can be reduced by two main bacteria categories: (1) assimilatory bacteria, which will reduce the sulfate for its own metabolic needs and, as an outcome, will incorporate sulfide into sulfur amino acids, thiamine or biotin. The production of sulfide by these bacteria is lower than dissimilatory bacteria as its production is limited by organic sulfur compounds in the rumen. Dissimilatory bacteria (2) are the group of bacteria that also utilise sulfate for their needs, but with a higher proportion of sulfide as an outcome.



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Sulfide is readily absorbed through the rumen wall reaching the bloodstream. Sulfide binds the haemoglobin, creating sulfhaemoglobin that reduces the ability to carry oxygen to tissues. Sulfide also affects energy production at the cell level, causing cell damage. Deaths have been reported from acute sulfide toxicity, as the liver plays an essential role in sulfide detoxication, which impairs liver function. The formation of H₂S in the rumen depends on two main factors: the excess of S, which will serve as a substrate for sulfur-reducing bacteria (SRB) and the ruminal pH. A lower pH in the rumen will result in a great proportion of sulfide as H₂S.

PEM originating from the respiratory system

PEM is a disease characterised by damage to the brain's grey matter leading to neurological dysfunction. Multiple causes can lead to PEM, such as thiamine deficiency, water deprivation, lead toxicity, sodium toxicity and S toxicity. PEM caused by S toxicity is a consequence of the accumulation of H₂S in the rumen produced by rumen bacteria and

then eliminated through eructation, as illustrated in *Figure 1*. It is estimated that 60% of the eructed gas is inhaled by the animal. Once breathed in, H₂S crosses the pulmonary barrier and reaches the brain. H₂S disturbs the oxidative process that generates ATP, the molecule responsible for carrying energy within the cell, causing cellular anoxia and death by apoptosis. The brain is the most affected organ as it has a high energy demand for oxygen, especially in the grey matter due to its many synapses.

Additional concern of S excess

The excess of S can also compromise the absorption of trace minerals in ruminants. In the rumen, free Molybdenum (Mo) and S form complexes called thiomolybdates that bind strongly to Copper (Cu), preventing it from being absorbed. The Cu deficiency caused by the interaction with S and Mo is called a secondary deficiency. Even if Cu is supplied at appropriate levels in the diet, the animals would be deficient as the complexation in the rumen would prevent its uptake by the animal.

Conclusion

Sulfur is an essential nutrient for ruminal bacteria that is often supplemented in ruminant diets. Nevertheless, the increased use of feedstuff containing high S levels raises concerns regarding mineral absorption, reduced feed intake and fibre digestibility affecting animal performance. Sulfur contribution of individual feedstuffs and feed additives has to be taken into account during formulation to manage the exposure of ruminants to high sulfur diets. Additionally, it should not be forgotten to quantify S content. The control of the S content in the animal's drinking water can also give insights about how much it is contributing as a source of S to ruminants.

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Figure 1 - Schematic of polioencephalomalacia caused by excess S in the diet. Sulfur sources, when ingested, are reduced in the rumen by rumen reducing bacteria into H₂S. The inhalation of eructated gases represent 60% in ruminants, and when in high concentrations of H₂S, it can lead to brain damage.

