The silent element

Sulfur (S) is an essential nutrient for ruminants, required for normal growth and development of both ruminan 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.

Essentiality of S

S is a constituent of several amino acids such as cysteine, cysteine and methionine. It is also present in other S-containing biomolecules such as biotin, taurine, co-enzyme A, ribo-
gen, 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 sulfur (N:S) ratio of protein in mixed ruminal bacteria is not constant, but a 2:1 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 saliva, weight loss, dullness and death. The maximum tolerable levels of S vary according to con-
centrate 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 organo-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 accumula-
tion of sulfides in the rumen environment, increasing the risk of acute toxic and polioencephalomalacia (PEM) in those ani-
mals. 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 out-
come, 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 or-
ganic 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.

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 fac-
tors: the excess of S, which will serve as a substrate for sul-
fur-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 depriva-
tion, lead toxicity, sodium toxicity and S toxicity. PEM caused by S toxicity is a consequence of the accumula-
tion 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 anaoxia and death by apopto-
sis. The brain is the most affected organ as it has a high ener-
gy 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 lev-
els in the diet, the animals would be deficient as the complexa-
tion 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 feedstuffs containing high S levels raises concerns regard-
ing mineral absorption, reduced feed intake and fibre di-
gestibility affecting animal performance. Sulfur contribution of individual feedstuffs and feed addi-
tives has to be taken into account during formulation to manage the exposure of ruminants to high sulfur diets. Addi-
tionally, 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.