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Copper in ruminants: beware of going from deficiency to toxicity

It has long been recognised that copper (Cu) is an essential trace element in ruminants. As copper is required in many key enzymes, any sub-clinical deficiency will impair animal health, fertility and production performance.

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Copper requirements and the maximum copper level authorised in the European Union are presented in Table 1. For bovine, the requirements are around 10mg/kg DM with higher values (up to 18mg/kg DM) for dairy cows in the close-up period and in the first weeks after parturition.

Higher values are recommended for caprine (up to 25mg/kg DM) but lower for ovine (up to 10mg/kg DM) which are highly sensitive to excess copper.

A genetic variation observed in copper absorption can also influence copper requirements. Literature mentions in particular higher requirements in Scottish Blackface than in Texel sheep and in Jersey than in Holstein Friesian cows.

After the 2016 EFSA Opinion on the revision of maximum contents of dietary copper, the maximum EU copper level authorised in bovine feed recently decreased from 35 to 30ppm, while it increased from 25 to 35ppm for caprine (Regulation (EU) 2018/1039).

In ruminants, copper is not used at growth promoting dosages like it is in monogastric diets. Thus, the re-evaluation of maximum copper levels is not driven by environmental concerns but more by nutritional objectives.

Poultry and pigs have copper requirements well established for decades, in ruminants and especially bovine, more research is still needed. This is due to the high sensitivity of ruminants to copper with a small margin between deficiency and toxicity, but also to the presence of antagonists in the rumen which can decrease the availability of copper for the animal.

Table 1. Cu requirements and maximum Cu dietary level authorised in the EU for ruminants (mg/kg complete feed).

		Bovines before the start of rumination	Other bovine		Small ruminants	
			Beef	Dairy	Ovine	Caprine
Requirements	NRC, USA (2000, 2001, 2007)	10	10	11-18	4-8	15-25
	GfE, DE (1995, 2001, 2003)	–	8-10	10	–	10-15
	CVB, NL (2005)	10	11-13	7-17	6-7	8
	Agroscope, CH (2006, 2009)	6	10	10-15	5	8
	INRA, FR (2018)	–	10		10	15-25
Maximum Cu dietary levels authorised[1]		15	30		15	35

[1]Regulation (EU) 2018/1039

Risk of secondary deficiencies

Copper deficiency is occasionally observed in ruminants. Mineral deprivation occurs due to either a primary or a secondary deficiency. The primary deficiency is the 'classic' form, in which low levels of copper are supplied in the diet and do not meet animal requirements for this mineral.

This scenario is most unlikely to happen, because the main ingredients of the diet meet the animals' need for copper, as shown in Table 2.

On top of the basal diet, mineral feeds are usually added in order to secure a large safety margin.

Secondary deficiency happens when even at the proper level of copper supplementation, the presence of other dietary factors interferes with mineral absorption and metabolism.

This phenomenon is the main cause of copper deficiency in ruminants. Sulphur (S), molybdenum (Mo) and iron (Fe) are the most important dietary factors to negatively impact copper absorption.

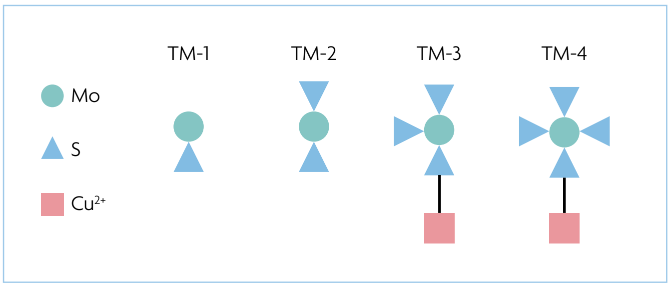
In the rumen, molybdenum and sulphur interact, forming complexes called thiomolybdates (TM). The nomenclature follows the level of sulphur chelation: mono (TM-1), di (TM-2), tri (TM-3) or tetra (TM-4) - thiomolybdate.

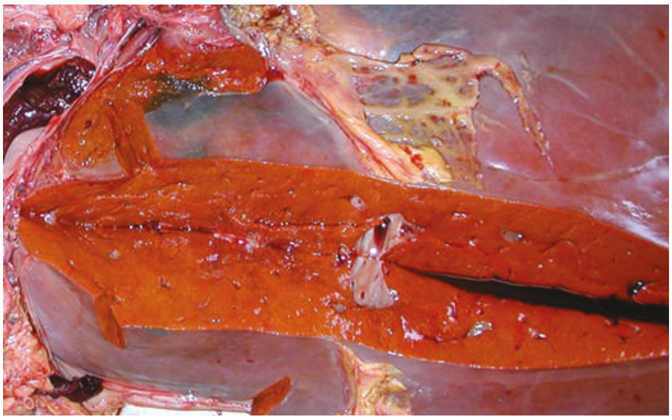
Fig. 1 illustrates how these metals interact in the rumen; TM-1 and TM-2 are less stable bonds and most likely reversible in the acidic environment of the abomasum, as TM-3 and TM-4 are more stable and with a greater affinity for copper.

Ionic form of copper, once released in the rumen environment, will be available for complexation with antagonists.

The attachment of copper molecule to TM-3 and TM-4 form a rather stable complex that precipitates into the solid phase of rumen digesta, remaining stable during the transit in the gastrointestinal tract. This sequestration of copper impedes absorption.

Fig. 1. Schematic representation of interactions between sulphur (S), molybdenum (Mo) and copper (Cu) in the rumen.





Copper toxicity leads to liver damage and jaundice (NADIS).

High levels of iron in water and/or diets also lead to copper deficiency, especially in grazing animals. Animals at pasture consume around 10% DM soil, rich in iron, while grazing.

The mechanism behind Fe/Cu competition is still not clear, but it is believed that iron associates to sulphur, forming FeS in the rumen. This complex dissolves under the low pH of the abomasum releasing sulphur that may bind to copper, compromising its absorption.

The signs of copper deficiency vary from mild symptoms such as loss of coat condition and poor growth, to more severe symptoms like infertility and diarrhoea.

As forage and diet compositions are seasonal and variable from farm to farm, secondary deficiencies are difficult to predict.

Therefore, copper in ruminant diets is usually supplemented well above nutritional requirements, to guarantee copper absorption regardless of the presence of antagonists (Fig. 2).

The slightest alteration in the dietary concentration of one of these minerals (S, Fe and Mo) can modify the host copper status from deficiency to toxicity.

In comparison to deficiency which has been well documented these last decades, excess copper in large ruminants seems to be an underestimated trend affecting animal health and welfare.

Death cases: the tip of the iceberg

While in the past copper deficiencies used to occur in grazing ruminants, researchers have observed in a growing number of countries in Europe, America and Oceania, an increased concentration of copper in the liver of dairy cow herds over the years.

Indeed, surveys in the USA, New Zealand, UK and The Netherlands reported that liver copper was above critical thresholds in a large number of animals and the tendency was increasing when herds were monitored over the years (see Fig. 3).

The main reasons reported for this increase were misinformation of farmers and the fear of deficiency. The concept of 'the more the better', in which recommendation limits are seen as targets to be achieved, is more and more accepted among farmers. As a consequence, there is an oversupply of copper, even without knowing the real mineral concentration of the diet.

This increase in copper supplementation can have a negative impact on animal welfare with cases of chronic toxicity identified more and more. At the opposite of acute toxicity which occurs after the accidental ingestion of a big quantity of copper, Chronic Copper Poisoning (CCP) is the result of the slow accumulation in the liver of smaller quantities of copper during a long period of time. Identifying CCP is not an easy task as in the first stage, there is no change in blood copper and the identification of the problem can only be done by evaluating copper liver concentrations.

The elevation of liver enzymes in the blood, that indicates hepatopathy caused by high levels of copper, can be related to a wide range of other diseases. Additionally, clinical signs are mild and with low morbidity, which can often be overlooked by veterinarians.

An increase in blood copper is only seen as a second stage when the liver is overloaded and copper is released into the bloodstream. This causes acute toxicity with animals often dying within 24-48 hours.

A growing number of lethal cases reported by veterinarians showed that such silent intoxication is spreading in dairy herds, which urges the development of strategies to monitor herd copper status and amplify the awareness of farmers for copper toxicity.

Recent research indicates that copper already accumulates in bovine liver at dietary levels recommended by the industry, and that cattle could be less tolerant to copper than previously thought.

Conclusion

The risk of secondary copper deficiencies in ruminants, and in general the increasing attention now being paid to trace mineral deficiencies, resulted over recent years in an increase in the quantity of copper supplemented in bovine diets.

The usage of theoretically more bioavailable sources, such as chelates, did not reverse this trend.

Because chronic copper toxicity is difficult to diagnose before the final stage where irreversible effects occur up to death, copper supplementation should be driven by the objective to prevent hepatic toxicity and oxidative damage.

Copper bioavailability for ruminants is mainly determined by sulphur, molybdenum and iron levels in the diet. Thus, complete forage analyses are essential to fine tune the quantity of copper needed to meet the animal's requirements, without forgetting drinking water which can be a significant source of iron and sulphur for the animal.

More advanced analytical techniques will be required to understand the effects of the chemical form of these antagonists.

The choice of the source of copper supplemented in the feed is also of importance.

Indeed, copper sources with known physicochemical characteristics and dissolution kinetics can help to select the one which is the less susceptible to form complexes in the rumen.

Copper(I) oxide (CoRouge), recently authorised in the market, combines high bioavailability with a low solubility at rumen pH.

This innovative source of copper will help to restrict the need for higher copper dosages in ruminant diets and to preserve animal productivity, health and welfare. ■

Ingredients	Cu mg/kg
Grass silage	8-10
Corn silage	4
Beet pulp	9
Sorghum	4
Soybean meal	14
Sunflower	20-30
Rapeseed meal	7

Table 2. Cu concentration in some feedstuffs used in dairy cow diets.

Fig. 2. Estimated Cu requirement of beef cattle at several dietary Mo and S concentrations (Jongbloed et al, 2005).

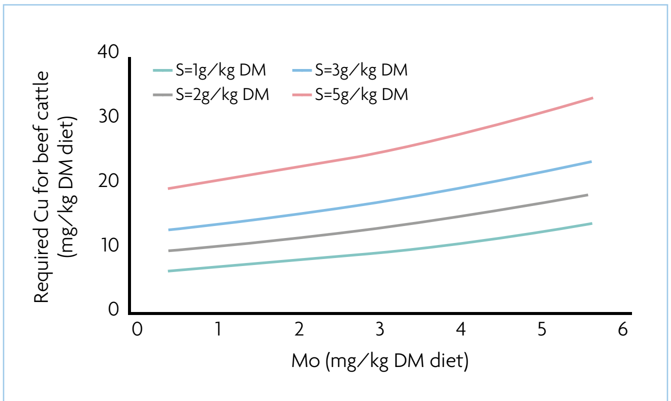


Fig. 3. Evolution of copper concentration in cattle liver over the last two decades (Grace and Knowles, 2015; Counotte et al., 2019).