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# Copper metabolism challenges in ruminants

## How to avoid interactions that prevent copper from being effectively absorbed.

ANDREA CLARKSON AND THE ANIMINE TECHNICAL TEAM

Copper supply in ruminants presents a unique challenge. Known in general as being copper-sensitive species, they are prone to the effects of oversupply leading to toxicity. Despite this, ruminants also experience interactions that can prevent copper in their diet from being effectively absorbed and used by the body, which also makes them susceptible to the effects of deficiency.

## Unique ruminants

The rumen creates a unique biological environment. It holds a delicate equilibrium with the millions of microflora that inhabit it and rely on the environment it creates for survival, while in turn, fermenting and catabolizing the components of the ruminant's diet and producing byproducts essential for the animal's survival. Prior to developing full rumen function, young ruminants are able to absorb approximately 75% of their dietary copper, similar to monogastric species. However, once the rumen is fully developed, there is a remarkable drop, down to less than 10% of dietary intake; this significant drop is attributed to interactions that take place in the rumen.

**THE IMBALANCE OF SUPPLY and demand is attributed to the complexity of interactions that influence copper absorption and metabolism.**

## From deficiency to toxicity

The consequences of undersupply can be difficult to detect. In youngstock, lowered immunity, poor growth, loss of body and coat condition, diarrhea or scouring are all incredibly impactful during rearing. Whereas in adult animals, the decline in immune function and impacts on fertility such as depressed or delayed puberty and/or oestrus, infertility, lower conception rates, and a higher neonatal mortality rate, are the most significant effects on production. On a practical level, these effects exist along a spectrum, thus, the problem can go largely unnoticed on a herd-flock basis when the signs are less obvious. As undersupply progresses into serious deficiency, it may be easier to detect as characteristic signs such as spectacling around the eyes, gingering coats, and the incidence of swayback — a neonatal ataxia commonly seen in lambs — begin to emerge.

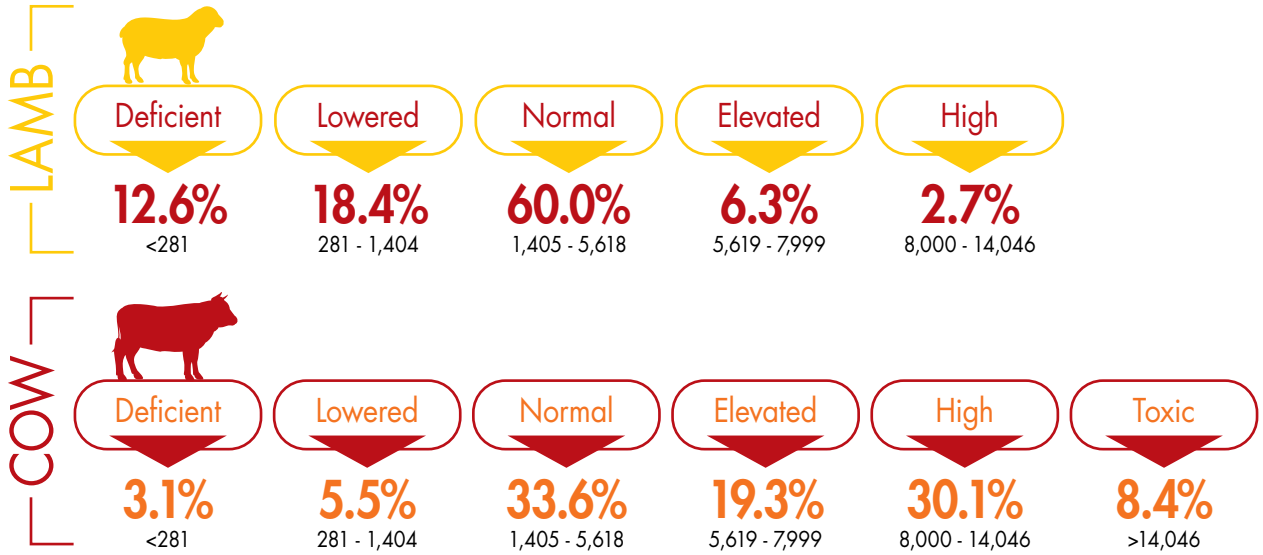
The consequences of oversupply are arguably of more concern, as oversupply has been overlooked for so long, especially in dairy cattle. Copper has the ability to accumulate in the liver and has been observed to accumulate to more than three times the upper end of the "normal" range in some seemingly healthy individuals. Animals which may appear healthy from the outside in this laden state have an increased risk of succumbing to copper toxicity, in the form of a hemolytic crisis. The hemolytic crisis is triggered by a stressor, often as simple as a change in housing, turning out, shearing in sheep or a change in social grouping, as well as more obvious stressors such as illness. The hemolytic crisis occurs as copper surges from the liver throughout the body and destroys red blood cells. Effects such as weak, recumbent "down" animals, jaundice and the heavy presence of blood in the urine are all common signs. Most do not survive, and for those that do, recovery is unlikely leading to euthanasia on welfare grounds.

It is important to remember that insufficient intake of copper will lead to a deficiency of copper. Although a seemingly obvious point, it is often overlooked, especially by sheep farmers who farm breeds considered to be highly copper sensitive, and it is suggested in U.K. sheep enterprises that approximately 30% of sheep may be of suboptimal copper status. This is a significant contrast to dairy cattle, where around 70% were of a copper status in excess of normal.

## Challenges in copper supply

The imbalance of supply and demand is attributed to the complexity of interactions that influence copper absorption and metabolism. Although, there are various interactions that are influential, including competition for intestinal transporters and interactions with other common

## Figure 1: Percentage of liver copper distribution in dairy cows and finishing lambs




**Percentage distribution of liver copper shown in µmol/kg DM in U.K. finishing lambs (Clarkson et al. 2017) and dairy cattle (Kendall et al. 2015).** *Source: Animine*

dietary components. The foremost concern on many farms relates to the presence of molybdenum.

Molybdenum is a natural mineral commonly present in the grazing sward. In the U.K., forages can range greatly in molybdenum content and can vary substantially with geographical area. Plants require molybdenum to assimilate nitrogen and will readily absorb it from the soil. Animals require molybdenum in such minute amounts that natural molybdenum deficiency is never observed. The effects of consuming high molybdenum forage can increase the animal's intake of molybdenum substantially and can lead to interactions that influence their supply of copper. It is important to note that it is not molybdenum alone which causes this effect.

Crucially, it is the combination of molybdenum and sulfur, another common dietary component, that create this problem.

Sulfur is also a normal component of plant matter and is essential for the formation of plant proteins. Like molybdenum, sulfur is required by plants to a greater degree than animals and grazing pasture can be much higher than the animal's requirement, especially when fertilized to optimize forage growth. It is the combination of these two minerals that interact with copper.

To cause their effects on copper utilization, molybdenum and sulfur combine in the rumen to form a compound called thiomolybdate. It is thiomolybdate that interferes with copper.

Thiomolybdate has an incredibly strong attraction to copper and can bind to available copper throughout the digestive tract, preventing it from being absorbed into the animal's bloodstream and leading to it being lost through feces. Perhaps more worryingly, if it does not bind to copper during digestion, thiomolybdate can potentially cross into the animal's bloodstream

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and interfere with copper in use by the body, where it interrupts copper's function in enzymes and other biologically important compounds. Once bound to thiomolybdate, copper becomes inactive. Although it is still present, it will no longer be able to fulfill its role in the body, impacting the animal's health and production. This effect is termed thiomolybdate toxicity and is arguably the underlying cause of many of the clinical signs associated with copper deficiency.

Another challenge lies in assessing the proportion of copper intake that will be biologically available to the animal. In order to be effectively used by the body, any mineral must be in a form that can be absorbed, converted to a physiologically active form, and metabolized. This is dependent on the chemical form of copper, or any other mineral, present. Many farms conduct routine mineral analysis of their forage. However, due to the high sensitivity of the analytical techniques used, these reports show mineral concentration, not bioavailability — an important distinction which makes it difficult to assess how much of the copper present will be available to the animal. It would be naïve to consider all of the copper detected on a forage analysis to be available, and the influence of the various antagonists must also be factored in to give a realistic expectation.

## Conclusion

Our challenge then is to balance the requirements of the animal, with the requirements of the pasture, to mitigate interactions, and provide an available supply, without oversupply. For this, we must ensure we consider the concentration of copper, and its antagonists, in the sward and in the full ration where applicable. We must consider the animal's copper status and look for the effects of interactions on biological parameters, such as the functional activity of copper-based enzymes. In doing this, we can aim to provide any additional copper deemed necessary in a targeted and precise manner, improving the welfare and production of the animals in our care. ■

*Editor's note: This is the third installment in a six-part series of articles by Animine.*

*Andrea Clarkson, Ph.D., is a researcher and lecturer in Animal and Veterinary Science, specializing in trace element nutrition with more than 15 years' experience in the field.*

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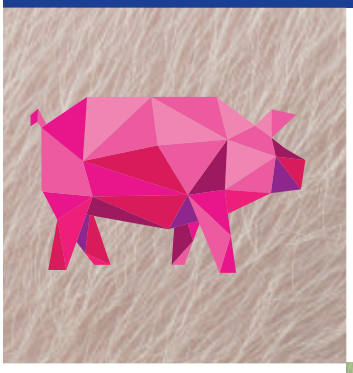

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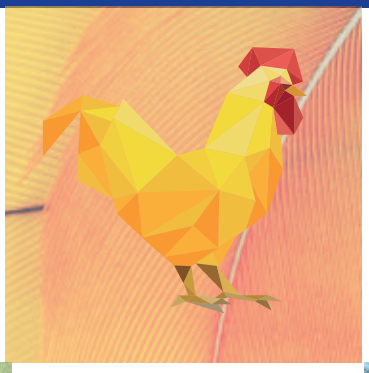

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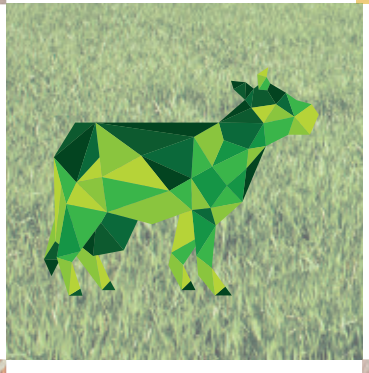

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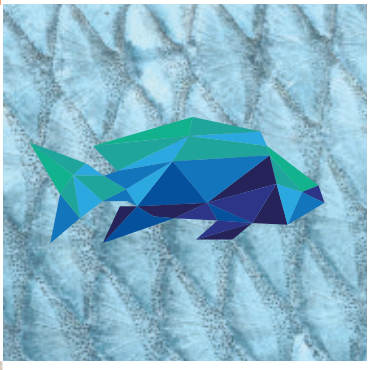

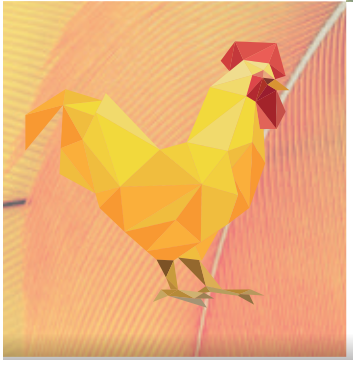

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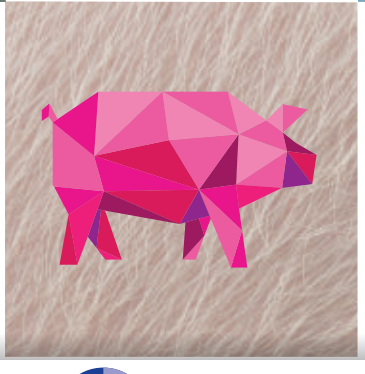

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