

I own livestock and have heard that copper can be harmful to them, especially sheep. Is this true?

Yes, that is true. Primary copper toxicity can be due to sudden or long-term ingestion of excessive amounts of copper. Secondary copper poisoning occurs when animals ingest certain plants, which damage the liver and then make it easier for copper to be deposited in the liver.

Sources of copper that may cause sudden toxicity include chemicals used to kill algae or fungus, footbaths with copper salts, treated wood, and injectable copper formulations.

Chronic toxicity occurs when animals are given feed rations or salt/mineral supplements with excessive copper levels, or levels that exceed toxic amounts when all feedstuffs are taken into account.

This may also occur when goats are given cattle copper boluses. Ingestion of excess copper relative to other minerals can result in toxicity as more copper is absorbed rather than being excreted by the animal.

Once copper is ingested, it is absorbed through the intestines and travels in the blood to the liver. Over weeks to months, copper builds up in the liver. Eventually massive liver cell death occurs either spontaneously or following a stressful event.

At this point there is a sudden release of stored copper from the liver into the circulation system, which results in membrane damage to red blood cells. This ultimately results in death of these cells and diminishes the ability of red blood cells to transport oxygen to tissues where it is needed. This can finally result in difficulty breathing, kidney failure, and death.

A major risk factor for development of copper toxicity is species susceptibility; swine and cattle are relatively resistant, while sheep are very susceptible. Goats, llamas, and alpacas may develop this as well.

A significant risk factor for sheep is feeding of rations intended for other species such as cattle and horses. In addition, feeding diets with a copper to molybdenum ratio greater than 10:1 predisposes sheep to accumulation of toxic levels of copper in the liver.

Signs of acute copper toxicity include difficulty breathing, head-pressing, staggering, and circling. Signs of chronic toxicity are depression, going off feed, weakness, watery/dark/blood-tinged stool, yellow or brown mucous membranes, and "port-wine" colored urine.

A diagnosis of copper toxicity should be made by a veterinarian since other diseases may appear similar. All feedstuffs and minerals can be assayed for copper content to determine total amount of dietary intake.

Treatment includes drugs that can bind copper, called copper chelators (ammonium molybdate, sodium thiosulfate, D-penicillamine, ammonium tetrathiomolybdate), intravenous fluids to

minimize kidney damage, occasionally blood transfusions, and always supportive care.

The prognosis is usually poor once blood cells start to be destroyed. The prevention of copper toxicity is possible by avoiding high copper to molybdenum ratios (keep them between 6:1 and 10:1) and sheep should never be fed cattle or horse rations.

This column is provided by the faculty of the OSU Boren Veterinary Medical Teaching Hospital. The large volume of questions does not allow us to directly respond to specific email questions so please watch for your answer in the column. Email your questions for the column to dvmonc.all@postoffice.cvhs.okstate.edu and watch for your answer.

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