Copper is essential for life and sheep have a dietary requirement for copper. Copper is required for normal chemical and physical processes that occur in the body. Copper is necessary for normal iron metabolism, red blood cell formation and production of skin and hair pigments, or melanin. It is essential for the formation of myelin, a substance that supports and protects the central nervous system. Copper is also necessary for cross-linking collagen and elastin which is required for normal bone formation. It is essential for wool production and is involved in the animal’s immune response to disease challenges.

The amount of copper required, however, is dependent on dietary and genetic factors, making it difficult to specify an actual requirement without describing the conditions for which it applies. The amount of molybdenum and sulfur in the diet are major factors influencing the requirement for copper. These minerals form complexes with copper, reducing its absorption or its biochemical availability in the body, and may increase the amount of copper required in the diet.

Breed differences also affect the minimum copper requirements of sheep. These differences appear to be due to differences in copper absorption and relate to differences in blood and liver copper concentrations among the various breeds. Different breeds are more susceptible to copper deficiency and toxicity. High retaining breeds such as the Texel are particularly vulnerable to toxicity while low retaining breeds such as the Scottish Blackface are more prone to deficiency. Mature ewes of British breeds seem to be quite susceptible to toxicity while Finnsheep retain less copper and are less susceptible to toxicity. Fine wool sheep, such as Merino are intermediate.

**Copper Deficiency**

Copper deficient areas of the Unites States have been reported in Florida and the coastal plains of the Southeast. Several western states have areas with high molybdenum levels capable of inducing copper deficiency.

*Lambs.* Ewes that undergo copper deficiency during pregnancy can give birth to offspring with congenital nervous system disease. Lambs may appear normal at birth and may not express neurological disease until they are between 1 week and several months of age, even though the condition results from the copper-deficient status of the dam. Affected lambs may show signs of “swayback” or may have difficulty standing or walking, know as ataxia. Lambs may be born weak and may die because of their inability to stand and nurse. Copper deficiency can also lead to defective production of red blood cells, causing anemia. In addition, lambs may develop bone fragility, known as osteoporosis, and can result in bowing of long bones and spontaneous fractures.

*Older and mature* sheep may exhibit the bone effects described above. Sheep suffering from copper deficiency have “steely” or “stringy” wool that is lacking in crimp, tensile strength, affinity for dyes and elasticity. Wool may also lose its pigmentation, resulting from deficient melanization.
Copper Toxicity

The most common type of copper toxicity in sheep is called chronic copper poisoning and is actually the result of a two-phase process. The first is the steady accumulation of copper in the liver over time. The second is the sudden release of copper from the liver into the bloodstream. Copper can build up in the liver over a matter of weeks, months, or more than a year depending on a variety of factors. Toxicity results when stress such as severe weather, disease or transportation causes the liver to release copper directly into the bloodstream with rapid and severe consequences. The breakpoint at which the liver can no longer retain its copper load is hard to predict.

Under normal circumstances, copper is absorbed from the diet and transported in the bloodstream to the liver for storage. Excess copper from the diet is stored in the liver and is released into the blood as needed for regular body functions. The circulating copper level tends to remain constant regardless of the amount of excess copper accumulating in the liver. This makes it difficult to ascertain a sheep’s copper status. The objective is to maintain a balance between the amount of copper required by the sheep, the amount provided in the diet and the amount accumulating in the liver.

When the breakpoint is reached and the liver releases stored copper, blood copper levels rise tenfold or more. This rush of copper into the sheep’s bloodstream causes a widespread breakdown of red blood cells. The released hemoglobin, which normally carries oxygen throughout the body, is converted to methemoglobin. This form of hemoglobin is unable to carry oxygen to tissues. The kidneys attempt to filter the large volume of hemoglobin and red blood cell fragments and become clogged, suffering severe tubular damage. Related symptoms are anemia, jaundice (icterus) which can be seen as pale, yellow membranes. Urine is typically a dark brown-red coloration. Affected sheep are anorexic, lethargic and extremely thirsty. This deteriorating situation results in shock, prostration and frequently death. Death usually occurs with 1 to 2 days of clinical signs, but massive copper release may result in dead sheep without any clinical signs. Surviving sheep should be treated to inactivate the copper, usually with a drench of ammonium molybdate and sodium sulfate.

The dietary copper required is best expressed as a range, rather than a specific value. Even so, these values are conditional and adjustments may be required for individual situations. With adequate molybdenum, a dietary copper range of 12-36 mg/kg of ration dry matter, or parts per million, is specified for sheep (Underwood and Suttle, 2001). Values below the lower limit rule out chronic copper poisoning while values above the upper limit strongly suggest chronic copper poisoning. The authors also recommend dietary molybdenum levels. Diets below a 0.5:1 copper:molybdenum ratio have a high probability of impairing health and production. Diets above a 3:1 copper:molybdenum ratio indicate minimal benefit from additional copper supplementation.

To minimize the risks of copper deficiency and toxicity it is important to become familiar with copper and molybdenum levels of purchased and home grown feeds, including pasture. Sheep are much more susceptible to copper toxicity than other livestock, so be cautious when feeding products and ingredients that are not specifically formulated for sheep.

Merrick’s Super Lamb is designed specifically for lambs. The typical Super Lamb copper level is 4 ppm, which is well within the safety range that rules out chronic copper poisoning. Molybdenum is provided in a 2:1 copper:molybdenum ratio, providing an optimum balance between the two minerals.