



Copper deficiency in sheep and cattle

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Summary

This farmnote looks at the symptoms of copper deficiency in livestock; its causes (soils low in copper, excessive molybdenum and sulphur intake); laboratory tests for copper levels; and preventative measures that can be taken (using fertilisers, injections and other means).

There are two main causes of copper deficiency in sheep and cattle:

- low copper levels in plants due to a lack of copper fertiliser in naturally copper deficient soils;
- an induced deficiency caused by the ingestion of excessive levels of molybdenum and sulphur in pasture or feed supplements.

In Western Australia, naturally occurring copper deficiency is not often seen in black-woolled sheep because most farmers have applied enough copper as fertiliser to ensure an adequate supply for grazing livestock for decades.

In contrast, before 1940, neonatal ataxia in lambs and falling disease in cattle were crippling livestock enterprises on many copper-deficient soils in the Gingin, Dandaragan, Dongara, Vasse, west coastal and Albany areas. There were also scattered incidences on lateritic and sandy soils throughout other agricultural districts.

Recent cases of low copper status

Low copper levels have been found in the blood or liver of animals that have eaten large amounts of soil during a drought or when stocking rates have been excessive, and in cattle with heavy *Ostertagia* infestations. Copper therapy increased liveweight when the deficiency was due to excess molybdenum (that is, responsive or true metabolic copper deficiency), but not where the cause was *Ostertagia*, starvation or the ingestion of soil (marginal copper deficiency or low status).

Deficiency symptoms

Cattle

- A loss of pigment from the coloured hair especially around the eyes giving the animal a bespectacled appearance. This does not show up in cattle with white hair around the eyes;
- falling disease – sudden heart failure.

Sheep

- Swayback or enzootic ataxia of lambs. Lambs with this condition cannot coordinate their legs. They may be severely affected at birth and may be unable to stand; some may be born dead. Other lambs appear normal at birth but between one and six months they develop an uncoordinated gait. This condition is caused by impaired development of the central nervous system in the foetus and cannot be reversed by copper treatment to the lamb once signs appear;
- loss of pigmentation in black-woolled sheep. Because there is usually a wide variation in susceptibility to copper deficiency between individuals within any flock, normal pigmentation in one or two marker black sheep does not guarantee copper sufficiency among the white-woolled individuals. Other conditions can occasionally cause loss of pigmentation;
- loss of crimp, secondary crimping and steeliness of wool are poor guides to copper deficiency because they are not solely caused by lack of copper and experts cannot consistently differentiate between steely and doggy wool. Copper deficiency is rarely the cause of the poorly crimped wool often seen in Western Australia.

Causes of copper deficiency in sheep and cattle

Soils containing little plant-available copper

Soils that were originally copper deficient occur throughout all farming districts – see Farmnote No. 25/88 *Copper, zinc and molybdenum fertilisers on new land*. Where no copper fertiliser has been applied on deficient soils, pastures often contain less than 2.5 ppm copper in spring, which may be not enough for the needs of animals.

Excessive intakes of molybdenum and sulphur

Molybdenum reduces the availability of dietary copper in the rumen by forming a highly insoluble copper–molybdenum–sulphur compound that is excreted.

In Western Australia, unlike many other parts of the world, the naturally occurring levels of molybdenum are rarely high enough to cause copper deficiency.

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However, excessive applications of molybdenum fertilisers have caused copper deficiency in sheep and cattle. Therefore, when considering molybdenum application:

- only apply molybdenum fertiliser to correct deficiencies in plants (see **Further reading**);
- do not apply molybdenum unnecessarily. One application of 75 g/ha molybdenum is enough for 10 to 15 years on slightly acid soils (pH 6.0 to 6.9), particularly those low in free iron and aluminium oxides. On highly acid soils (pH 4.0 to 5.0) high in free iron and aluminium oxides, molybdenum deficiency may recur within one to three years of molybdenum fertiliser application.

Aluminium toxicity also appears to restrict plant production on these soils. Liming may prove to be the better way to keep up an adequate molybdenum supply to plants on the highly acid soils while also correcting aluminium toxicity.

- If molybdenum is applied to pasture *do not allow sheep or cattle to graze the area until after heavy rain*. In many cases animals that have grazed paddocks immediately after the pasture has been top-dressed with molybdenum–superphosphate in autumn have developed severe copper deficiency. It is safer to drill molybdenum fertiliser with a preceding cereal crop than to apply it to a pasture that is to be grazed that year;
- liming soils can increase molybdenum availability but can also reduce copper availability and induce a deficiency if copper levels are marginal.

Indicators of copper status

If the soil provides enough copper for wheat (Farmnote No. 49/85 *Copper deficiency in wheat*), pasture grown on the same area will have sufficient copper for sheep and cattle, unless molybdenum levels in the soil are very high.

Visual indicators

- Swayback in lambs and falling disease in cows can be diagnosed with a high degree of certainty.

While de-pigmentation of the hair around the eyes in cattle and of black wool in sheep indicates copper deficiency is likely, it is not certain as similar symptoms occasionally result from other causes. Use as many other indicators as possible to make sure of your diagnosis.

Laboratory tests

- Liver copper concentration is one of the best, indicators of copper status, although this is still not guaranteed. One cause for uncertainty is that individual sheep or cattle develop copper deficiency with different levels of copper in their livers. Another limitation can be the difficulty and cost of obtaining a liver sample by biopsy from live animals. Even after slaughter it is difficult to retain livers from identifiable animals. Nevertheless, liver analyses are still the most accurate indicators of copper status. (See Table);

- the concentration of copper in the blood can be an unreliable guide to copper status if it is a molybdenum-induced deficiency. Caeruloplasmin activity in plasma or comparing different plasma inorganic copper fractions (TCA-soluble versus TCA insoluble) can indicate if molybdenum is involved;
- pasture analyses for copper and molybdenum concentrations provide only a rough guide to the copper status of sheep and cattle grazing them. Pastures with less than 2.5 ppm copper are sometimes deficient for sheep and cattle. Above 4 ppm copper, they are not deficient, *provided molybdenum levels are less than 1.5 ppm* (normal for Western Australian agricultural areas). The likelihood of copper deficiency can be assessed using levels of copper, molybdenum and sulphur in plants.

Table 1. Interpreting liver analyses

Liver copper level (ppm)	Sheep	Cattle
0 to 2	Deficient	Nearly always deficient.
2 to 4	Deficient	Usually deficient. Normal with heavy <i>Ostertagia</i> burden
4 to 8	Often deficient	Dangerously low, occasionally deficient. Normal with heavy <i>Ostertagia</i> .
8 to 16	Dangerously low, some deficiency likely to develop. Normal for sheep ingesting sand.	Possible deficiency with further decline.
16 to 32	Low/normal. Possible problem if further decline.	Normal
32 to 600	Normal	

Pasture with copper levels between 3 ppm and 4ppm have seldom caused copper deficiency in stock in Western Australia. However, with higher pasture molybdenum and sulphur levels the copper concentration needed to give an adequate supply to sheep and cattle rises. For example, in other parts of the world, animals grazing pastures that have 5 ppm molybdenum and higher have still responded to copper even though the pasture contains as much as 10 ppm copper.

Dosed versus not dosed comparisons

In the absence of clear-cut clinical symptoms, the most convincing evidence of a copper deficiency is a response to treatment with copper, that is; treating some animals then comparing them with the other untreated animals from the same flock or herd.

Both groups must be run together under identical conditions. A minimum of 25, with preferably more animals in each group, is usually needed to detect small but economically important differences in liveweight.

Preventing copper deficiency in sheep and cattle

Fertilisers

The residual effectiveness of fertiliser copper is well established. One application of 0.8 to 2.5 kg/ha copper (3.3. to 10 kg/ha copper sulphate), according to soil type and locality as shown in Farmnote No. 25/88, will supply copper to sheep and cattle for at least 20 years. A second application of 0.5 kg/ ha copper (2 kg/ha copper sulphate) 10 years after the first is probably unnecessary, but may be regarded as good insurance.

Where too much molybdenum has been applied, extra copper fertiliser may not be effective in correcting the induced copper deficiency, since it can occur even when soil and plant copper levels are high.

Injections

Copper compounds injected under the skin (such as Cujec[®], diethylamine cuproxy quinoline sulphonate) can correct a deficiency, but must be repeated every six months. This method is both less convenient and more expensive than supplying copper through fertilisers.

Licks, drenches, copper needles and drinking water

Copper sulphate can be supplied through licks and drinking water, but the dose rates cannot be controlled. Some animals can get too much and risk toxicity while others, especially with licks, may get too little.

Drenches only have a brief effect and are not recommended for the treatment of copper deficiencies. Sheep are highly susceptible to copper toxicity and care should be taken whenever supplementing livestock. Many liver toxins will cause copper to accumulate in the liver and can lead to secondary copper toxicity.

Copper oxide needles are a slow release form of copper administered as a capsule. They are active for 6 to 12 months.

Further reading

Farmnote No. 8/2004 *Trace element deficiency in sheep and cattle*