Copper poisoning in sheep

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(A broadcast talk given on the A.B.C. Country Hour.)

ANIMALS must have small amounts of copper for a number of normal bodily functions, including growth and blood formation and, in the sheep, wool quality. The liver is used as a "bank" or store-house for copper which is collected from the diet and in due course issued to the body tissues as required. The sheep—in contrast to other species of animals—is apt to play the miser and hoard unnecessary amounts.

Under certain circumstances the copper content of the sheep's liver builds up to dangerously high levels. A fatal poisoning may then occur following a sudden release of copper from the liver. This results in blood destruction and jaundice or "yellows."

Chronic copper poisoning or toxaemic jaundice has been responsible for serious sheep mortalities in Victoria and New South Wales, where intensive investigations have elucidated the causes and nature of the disease. The problem is not nearly so serious in this State but outbreaks have occurred here from time to time, and for many years.

Let's now consider the factors which may lead to the storage of excessive amounts of copper in the sheep's liver; the conditions determining the sudden release of this copper; the symptoms and post-mortem appearances seen in poisoned sheep; and finally the means of preventing or controlling mortalities.

COPPER STORAGE

The normal copper content of the sheep's liver is 100 to 500 parts per million. Dangerous copper levels of the order of 1,000 to 3,000 parts per million may occur under the following circumstances:—

Firstly, with a high intake of copper, secondly, with a normal copper intake but with the presence of certain components in the diet which promote or "condition" excessive copper storage, or thirdly, as a sequel to liver damage from certain poisonous plants.

An excessive intake and storage of copper may occur in areas where pastures or herbage naturally have a high copper content due to the nature of the soil or to other environmental causes. In this State such areas are very restricted. On a number of occasions, however, poisoning has resulted from excessive supplementation with copper.

Secondly, excessive copper storage may occur even when the diet of the sheep only contains normal amounts of copper. This happens when the diet is low in molybdenum and sulphate. The element molybdenum in association with sulphate has been shown to exert a profound influence on copper storage by the sheep. Heavy losses have at times been experienced in Victoria and New South Wales where sheep were grazing pastures dominated by subterranean clover, and with a very low molybdenum content.

Although clover dominance is much more commonly encountered here, Western Australian pastures, in general, have normal molybdenum and sulphate values. For this reason presumably toxaemic jaundice is not a hazard of clover dominance in this State.

Thirdly, some of the toxic plants which damage the liver have the effect of making this organ particularly avid for copper, so that excessive amounts are stored even when the diet is relatively low in copper content. The most important offender is a species of Heliotrope (H. curoneum). This plant is common in parts of Victoria, New South Wales and South Australia and has been responsible
there for numerous and serious outbreaks of toxaemic jaundice for many years. Fortunately, the plant is rare and of no significance in this State. We have some evidence here, however, which suggests that lupins may on occasions act in a similar way. Under conditions not yet understood the grazing of lupins may lead to liver damage (lupinosis) which in some cases has been associated with high liver copper values and evidence of copper poisoning. The weed Patterson's Curse has also been implicated in the East but is not known to have caused trouble here.

So much for the factors which may lead to excessive copper storage. Let's now consider the results of this. At the outset it must be realised that sheep carrying very high concentrations of copper in their livers may remain in perfect health and continue to show no signs whatever of poisoning. **Provided the copper remains in the liver there is no trouble.**

When, however, it is discharged into the circulation, blood destruction occurs, the animal becomes ill and generally dies. This sudden discharge of copper from the liver occurs particularly during stress, notably following a change of diet, a period of starvation or the effects of mustering and droving. May I illustrate from one case I remember? A flock of sheep on a certain property, maintained on copper topdressed country, had also eaten large quantities of copper lick. A draft was sent to Midland and held for slaughter at the abattoir. In this mob—subjected to stress—there was a heavy mortality from copper poisoning, whereas the mob left quietly on the home pasture, remained perfectly healthy.

In some instances, however, fat sheep undisturbed at pasture may succumb.

**SYMPTOMS**

Quite commonly, sheep are only noticeably ill for a few hours but death may be delayed for up to three or four days. The main signs are dullness, prostration, the occurrence of red or brown urine, and jaundice; the linings of the mouth, nose and eyes and the skin show a yellow discolouration.

The typical changes seen in the carcass are jaundice, or yellow bile staining of the tissues, and the black or gunmetal colour of the kidneys. This characteristic appearance of the kidneys serves to differentiate chronic copper poisoning from other diseases, such as lupinosis, in which jaundice occurs. The dark colour of the kidneys and urine results from the blood destruction caused by the copper and the excretion of blood pigment via the urinary tract.

In the Eastern States mortalities of over 50 per cent. have been reported. These heavy losses are commonly associated with the grazing of heliotrope. In this State occurrences of toxaemic jaundice are relatively uncommon and losses are less severe. A number of outbreaks, however, have been reported and investigated from time to time. These have been associated with excessive copper supplementation, occasionally as a sequel to lupinosis, and in a number of cases apparently as a result of the action of undetermined plant or other factors damaging the liver. On certain pastoral properties in the Goldfields and Murchison region high liver copper values are common and deaths may occur under conditions of stress associated with drought, with shearing, or with transport of sheep to market.

Under Australian conditions occurrences of the disease may be seasonal and deaths may be spread over a period of weeks or months. The Merino is said to be much more resistant than British breeds and their crosses.

**CONTROL**

The control of toxaemic jaundice, must depend upon the control of the various factors responsible for the development of the disease—that is of the causes leading to excessive copper storage, and of the conditions favouring heavy discharge of copper from the liver, which causes illness and death.

Excessive copper supplementation must be avoided. Copper supplements are desirable to control copper deficiency but too high a copper intake is dangerous to sheep (cattle are much more resistant).

Where liver damage due to known plants is responsible, the recommendation is obvious. In locations where high copper storage is a result of the low molybdenum content of soil and pasture topdressing...
with sodium molybdate at the rate of 4 oz. per acre will rectify the position. Alternatively a lick may be used with benefit. The one recommended in the East consists of a mixture of one bag of salt (187 lb.), one bag of ground gypsum (140 lb.), and 1 lb. of sodium molybdate. So much for the prevention and control of high copper storage.

We must remember too that loss of body weight and other stresses precipitate the occurrence of disease. When the disease occurs in a flock, deaths can be minimised by good feeding and by keeping the sheep as quiet as possible. Drovers, mustering and transportation must be avoided. It seems reasonable also to recommend the feeding of the lick not only to prevent excessive copper storage but also to promote a gradual reduction of the dangerous amounts of copper already accumulated by sheep in the affected flock.

**DYSENTERY IN CATTLE**

During the latter part of 1958, widespread outbreaks of infectious dysentery have been reported in dairy cattle throughout much of the south-western portion of the State, and the infection is still spreading. The disease was present amongst cattle exhibited at the Royal Show, and with the return of these cattle to their home properties, further outbreaks occurred over a large area.

The disease is highly infectious and spreads with great rapidity. Almost all of the cattle in a herd become affected in a few days and the interval from the first case until the final disappearance of the disease, is little more than a fortnight.

In the initial stages, there is reduction or loss of appetite and this is soon followed by the development of a profuse watery diarrhoea which sometimes contains flecks or larger quantities of blood and may persist for one to three days. In dairy herds, there is a sharp decline in milk production which may be reduced by one-third to one-half or more, for a period of several days.

Recovery occurs without treatment in three to five days, and except in cows which are nearing the end of their lactation and may dry off, there is a gradual return to full production. Cattle of all ages are susceptible, but dry stock do not appear to suffer any very serious ill-effects.

There is no specific treatment. Drenching with intestinal astringents and sedatives such as copper sulphate and mixtures of chalk and catechu have been recommended, but in view of the duration of the disease, and that it is probably caused by a virus, this would appear to be of doubtful value.

The disease is a new one to Australia and bears a marked similarity to the condition described as winter dysentery in Canada and the United States. It has been transmitted to healthy cattle by bacteria-free filtrates of the excretions from affected cattle which would indicate that it is caused by a virus.

It was first reported in New South Wales early in June, and since that time, very extensive outbreaks have occurred. More recently, a series of outbreaks which stemmed from the Brisbane National Exhibition have been reported from Queensland, and there have been unconfirmed reports of the occurrence of the disease among cattle at the Royal Shows at Melbourne and Adelaide.

How the infection reached Australia has not been determined, but there can be little doubt that it was brought to Western Australia by cattle from the Eastern States which have been imported in much larger numbers than usual during recent months.

Fortunately the infection is a mild one and there is no cause for alarm. Although morbidity is high, few deaths have occurred, and apart from a brief loss of production in dairy herds, there have been no very serious ill effects.
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