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Brief Communication

CONCENTRATION OF SELENIUM IN LIVER IN RELATION TO COPPER LEVEL IN NORMAL AND COPPER-POISONED SHEEP*

Copper absorption, liver accumulation and development of copper toxicosis in sheep are influenced by a variety of other elements, in particular molybdenum, sulphur and zinc (*Underwood* 1977). In a previous study on liver concentrations of copper, molybdenum and zinc in normal and copper-poisoned sheep, no direct correlation was found between the concentrations of the three metals, but molybdenum was significantly lower in the livers from sheep dead from chronic copper poisoning than in normal animals (*Frøslie & Norheim* 1976).

Selenium is known to protect against the toxic effects of several heavy metals, including mercury, cadmium, silver, thallium (*Ganther* 1974) and lead (*Rastogi et al.* 1976). Copper induces lipid peroxidation in hepatocytes (*Lindquist* 1968) and oxidative damage to red cells (*Metz* 1969), both among the kind of lesions selenium is supposed to counteract. Thus, a role for selenium in the defence against manifest copper toxicosis is a tempting hypothesis.

In lambs with suboptimal copper status, *Thomson & Lawson* (1970) observed a significant increase of liver copper concentrations after selenium administration. In copper-supplemented lambs, however, no effect could be seen. *Awad et al.* (1973) and *Lee & Jones* (1976) found small or no effects of selenium on liver copper concentrations in sheep. In both these studies, the levels were within 150—460 μg Cu/g dry matter (about 45—140 μg Cu/g wet weight).

In Norway, copper accumulation in grazing sheep is common (*Frøslie* 1977). The aim of the present investigation was to evaluate a possible relationship between copper accumulation, selenium status and manifest copper poisoning under clinical conditions. The study included 45 livers from cases of diagnosed

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Table 1. Liver concentrations of selenium in 88 normal sheep and 45 fatal cases of chronic copper poisoning, at different class intervals of liver copper. Mean and range.

		Liver copper, μg/g wet weight				
		0—150	151—300	301—450	451—600	> 600
Normal sheep	Numbers	24	20	29	9	6
	Selenium, μg/g w.w.	0.18 (0.02—0.45)	0.20 (0.02—0.68)	0.21 (0.05—0.68)	0.20 (0.13—0.26)	0.42 (0.19—0.64)
Copper poisoned sheep	Numbers	2	9	21	10	3
	Selenium, μg/g w.w.	0.09 (0.04—0.15)	0.21 (0.08—0.44)	0.26 (0.12—0.74)	0.21 (0.04—0.45)	0.13 (0.07—0.21)

chronic copper poisoning during the years 1974—77, with liver copper concentrations ranging from 150 to 910 μg/g wet weight; and 88 livers from normal slaughtered sheep, selected to cover a representative range of liver copper levels, from 2 to 830 μg/g wet weight.

Copper analysis was done by atomic absorption spectroscopy after wet digestion. Selenium was determined by a fluorometric method (*Ihnat* 1974).

In the complete material, liver selenium concentration ranged from 0.02 to 0.74 μg/g wet weight, with an average of 0.22 μg/g; 26 livers contained 0.10 μg Se/g or less. Table 1 shows selenium concentrations in livers from normal and copper-poisoned sheep, at different class intervals of liver copper. There was no difference in liver selenium level between the two animal groups. There was also no significant correlation between selenium and copper levels, neither in the copper-poisoned ($r = -0.02$) nor in the normal animals ($r = 0.29$). In the normal sheep, there seemed to be a slight overrepresentation of high selenium values in samples with copper levels above 600 μg/g wet weight. However, the number of these samples is small.

In conclusion, the present material does not reveal any systematic relationship between copper and selenium concentrations in sheep livers with normal to high copper levels. Furthermore, it lends no support to a hypothesis of selenium influence on the development of manifest copper poiosoning. For such a hypothesis to be ruled out, however, more direct experimental evidence is needed.

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