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Brief Communication

Ovine White-Liver Disease (OWLD). Treatment trials

Ovine white-liver disease (OWLD) occurs in cobalt/-vitamin B_{12} deficient lambs in Norway, and can be prevented by Co supplementation or regular dosing with B_{12} (*Ulvund & Pestalozzi* 1990a, *Ulvund* 1990a). Lambs which developed OWLD showed early signs of liver damage (*Ulvund* 1990b). Serum copper was higher in OWLD lambs than in Co/B_{12} supplemented controls and although the Cu content of OWLD pasture grass was scarce (*Ulvund & Pestalozzi* 1990b), Cu supplementation alone worsened the condition (*Ulvund* 1990c). There were some indications that OWLD lambs recovered very slowly on indoor feeding. It therefore became of interest to evaluate various treatments of lambs with clinical OWLD, to explore whether the hepatic damage was persistent and irreversible, and to examine the length of convalescence when affected lambs were put on indoor feeding only. There are few earlier reports on these aspects. Some authors report clinical improvement of OWLD after B_{12} treatment (*Richards & Harrison* 1981, *Mitchell et al.* 1982).

Experiment A: Three lambs with typical OWLD were treated on pasture (September 6) every second week with either B_{12} (hydroxocobolamin, 2 mg, NYCO, Norway), copper oxide needles (Copporal, 2 g, Beecham Animal Health, England, CuO), or both B_{12} and CuO.

Experiment B: Three OWLD affected lambs were treated on pasture fortnightly with B_{12} (from August 9).

Experiment C: Six affected lambs were put indoors (September 4) and fed hay and silage ad lib., and an individual daily ration of about 300 g concentrates containing 12.5 % digestible crude protein, with 0.2 mg cobalt (CoO) and 10 mg copper (CuO) added per kg feed. Four of these were treated fortnightly with B_{12}, while 2 were left untreated.

Dosing of lambs on pasture with B_{12} (Exp. A and B), or B_{12} and CuO, gave a quick clinical improvement, with 2–3 kg in live weight (lw) gain per week. Only the lamb dosed with CuO showed no improvement. The four B_{12} treated lambs fed indoors (Exp. C) regained normal appetite during the first week after treatment. The eye discharge and the crusts on ears disappeared after 2–4 weeks, and weekly lw gain was 1–2 kg. The 2 untreated lambs regained their appetite after 6–8 weeks, and then started to gain weight. During 3 months, the 4 B_{12} treated lambs gained on average 8 kg more than the untreated ones.

B_{12} treatment (Exp. A) resulted in lowered serum Cu 1 month later, while this effect was not seen in the lamb given both B_{12} and CuO (Table 1). Serum Cu was only slightly higher in the CuO treated lambs than in the untreated ones. B_{12} treatment in Exp. B also depressed serum Cu, viz. from pretreatment value 14 µmol/l to 9 µmol/l about 4 weeks after first treatment. In Exp. C, mean serum Cu was 13 µmol/l 2 months after treatment, as compared with 20 µmol/l for the undosed.

Serum iron (Fe), glutamate dehydrogenase (GLDH), and plasma vitamin B_{12} and methylmalonic acid (MMA) in lambs of Exp. A are given in Table 2. Dosing with CuO, with or without B_{12}, kept Fe and GLDH elevated,

Table 1. Experiment A. Mean serum Cu (μmol/l) in unsupplemented lambs grazing OWLD pastures, and values in 3 individuals which were treated on September 6 with either copper oxide needles (CuO), CuO *and* vitamin B_{12}, or B_{12} only.

	Dates of sampling			
Treatment	July 12	Aug. 9	Sept. 13	Oct. 7
Untreated (mean)	16	17	18	11
n	6	6	3	3
CuO	12	13	18	15
CuO & B_{12}	13	15	22	15
B_{12}	–	18	16	4

n = number of lambs sampled.

while B_{12} treatment alone reduced the values. Cu seemed to inhibit the restoring effect of B_{12} on damaged liver cells. Plasma B_{12}/MMA were normalized after B_{12} treatment, with ot without CuO.

CuO treatment resulted in reduced packed cell volume (PCV) 1 month later (28 %) as compared with CuO & B_{12}, or B_{12} treatment (> 33 %). In Exp. B, B_{12} treatment yielded increased plasma glucose, total cholesterol and urea 2 weeks later. GLDH was reduced from pretreatment range 26-72 U/l to 2-10 U/l, but γ GT remained unaltered. Seven weeks after treatment, mean serum Fe was lowered (from 39 to 28 μmol/l). In Exp. C, B_{12} treatment resulted in elevation of plasma glucose 3 days later, and GLDH was lowered (< 10 U/l) after 10 days. Similar experiments have not been performed earlier in OWLD, but the quick glucose elevating effect of B_{12} corresponds to findings by *MacPherson et al.* (1976) in simple Co deficiency. The 2 untreated lambs fed indoors had low plasma glucose (< 2.8 mmol/l), low tot. cholesterol (< 1.0 mmol/l) and elevated GLDH (> 30 U/l) for 8 weeks, while γ GT was > 40 U/l and PCV < 30 % for 2–3 months.

Table 2. Experiment A. Serum iron (Fe, μmol/l), serum glutamate dehydrogenase (GLDH, U/L), plasma vitamin B_{12} (pmol/l) and methylmalonic acid (MMA μmol/l) in 3 lambs with OWLD, treated on September 6 with either CuO, CuO *and* B_{12}, or B_{12} only. Mean values for 6 (August) and 3 (September/October) untreated lambs are included.

	Dates of sampling											
	Aug. 9				Sept. 13				Oct. 7			
Treatment	SI	GLDH	B_{12}	MMA	SI	GLDH	B_{12}	MMA	SI	GLDH	B_{12}	MMA
CuO	5	23	÷	156	42	69	66	208	47	46	÷	162
CuO & B_{12}	41	73	÷	102	44	85	552	5	32	80	÷	4
B_{12}	49	55	÷	206	40	17	653	2	27	23	÷	8
Untreated	45	56	77	80	43	76	68	143	36	199	132	24

÷ not examined

In conclusion, B_{12} treatment caused quick clinical improvement, elevation of plasma glucose after 3 days, reduction of serum GLDH after 10 days, elevation of cholesterol and urea after 2 weeks, and lowering of Fe and Cu after 1–2 months. In contrast, OWLD lambs put on indoor feeding recovered slowly, and clinical pathology was unchanged for 2 months. Cu treatment prevented healing of liver damage as judged by serum GLDH.

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