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Cobalt Deficiency in Ruminants

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All ruminants (including sheep, cattle and goats) require cobalt in their diet for the synthesis of vitamin B12. Vitamin B12 is an essential part of several enzyme systems that carry out a number of basic metabolic functions and is required for energy metabolism, the production of erythrocytes and maintaining nervous system integrity. These functions are significantly affected in the deficient state. Cobalt deficiency in soils is directly related to vitamin B12 deficiency in livestock as plants have no essential requirement for cobalt, so uptake by plants is variable.

Most soils contain more cobalt than the plants growing on them. Deficiency most commonly occurs on lush grazing or pasture grown in cobalt deficient soils due to multiple factors, including dilution of cobalt in rapidly growing plants and reduced soil contamination of lush plant material.

Soil contamination of grazing may constitute 10-25% of the total dry matter intake of livestock on short and dry grazing. Schneider *et al.* (1991) noticed paradoxically in cobalt deficient areas in the southwestern Cape that sheep were in the best condition during autumn when the grazing was dry and of poor quality, but in poor condition when on lush spring grazing.

Sheep and goats are more susceptible to cobalt deficiency than cattle, and deficiency is more frequently reported in weaners and young animals due to their increased energy demand for growth.

Animal factors influencing the requirement for cobalt

- **Age.** Newborn animals have low reserves and a high requirement for cobalt. Colostrum provides vitamin B12, but milk has low levels. Where grazing is likely to be lacking in cobalt, young animals should be treated at 6-8 weeks of age with injectable vitamin B12. Weaner sheep are also more prone to cobalt deficiency than adult animals.

- **Species.** Sheep and goats appear to be more susceptible to cobalt deficiency than cattle.

- **Worm burdens.** Barbers pole worm (*Haemonchus*) and other diarrhoea causing worms (*Ostertagia*, *Telodorsagia*, *Trichostrongylus*, *Nematodirus*, *Fasciola*) cause loss of protein from the gut and a reduction in feed intake. Young animals with heavy worm burdens will be more susceptible to cobalt deficiency. Avoid weaning young animals onto pastures that are likely to have high worm burdens.

Plant and climate factors affecting cobalt availability

- **Soil type.** Cobalt deficiency mainly occurs on deep sandy soil, weathered leached soils, sandy soils with clay subsoils, loamy and gravelly sands. The level of soil contamination of grazing also significantly impacts cobalt availability.

- **Pasture species.** Grasses contain about half the cobalt found in legumes growing on the same soil. Cobalt deficiency usually occurs in animals grazing rapid growing, grassy pastures.

- **Winter rainfall.** There is a close relationship between climate and the incidence of cobalt deficiency. While deficiency is generally only seen in areas with a greater than 500-millimetre annual rainfall, the winter rainfall in the current year has the most influence. Winter rainfall influences cobalt availability in two ways: it leaches the cobalt from the soil and affects the pasture growth rate in the spring flush. Cobalt is not an essential nutrient for plants, so high growth rates in spring dilute the amount of cobalt in the plant.

Clinical Signs

Ill-thrift, poor growth rates, reduced appetite, anaemia and scaling dermatitis of the ear pinnae are consistent signs described in all ruminants.

Additional signs described in sheep include excessive lacrimation with matting of wool below eyes, break in the wool, and ewes may have lambs of low body weight. Additional signs in cattle include a rough, pale coat, reduced milk production, diarrhoea in calves and 'pica' with affected animals eating soil, bark, leaves etc.

Pathology

In all ruminants (sheep, goats, cattle), severe emaciation with marked depletion of body fat stores, serous atrophy of fat, moderate to severe anaemia and hypoproteinaemic body effusions (hydrothorax, hydropericardium, ascites) are usually observed. A unique cobalt associated hepatopathy (white liver disease) is described in sheep and goats, but not cattle.

There is severe hepatomegaly in acute white liver disease with increased friability and marked fatty hepatosis (liver biopsies usually float in formalin). Hepatic fatty change in chronic white liver disease is less severe and not always grossly visible. Icterus is variably observed.

The hallmark features of the hepatic histopathology of white liver disease are fatty change in hepatocytes, portal bile duct hyperplasia and mesenchymal proliferation and the presence of ceroid pigment in both hepatocytes and macrophages (Figures 1, 2, 3).

The degree and distribution of these changes vary with the stage of the disease. In the initial acute stages, the distribution of fatty change in primarily centri-zonal and portal proliferative changes is mild. With increasing chronicity, fatty change becomes more diffuse, and portal proliferative changes are marked.

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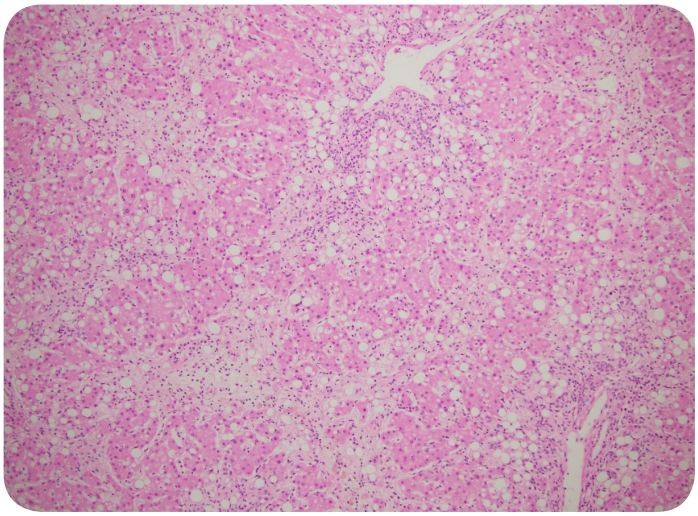


Figure 1. Sheep live: marked hepatocellular vacuolar degeneration (fatty change) with marked portal mesenchymal proliferation. A few pigment engorged cells (hepatocytes and macrophages) are just visible at this magnification.

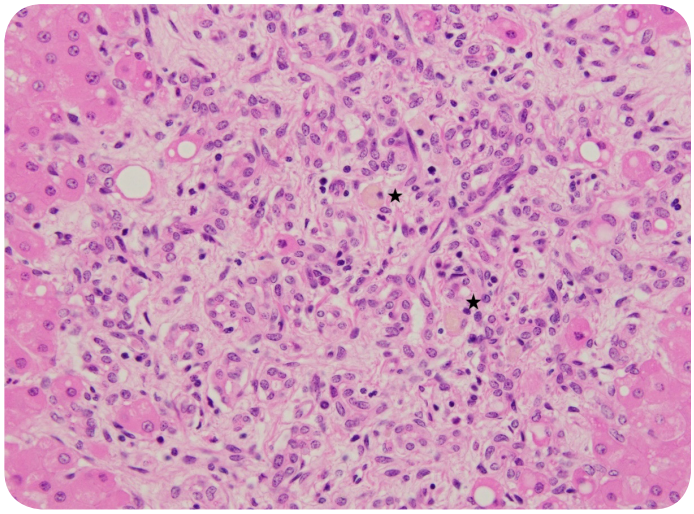


Figure 2. Sheep liver higher power view of portal area exhibiting biliary hyperplasia, mesenchymal proliferation with pigment engorged macrophages in the portal tracts (*). Mild inflammatory infiltrate of individual mononuclear cells (H&E stain 40 X magnification).

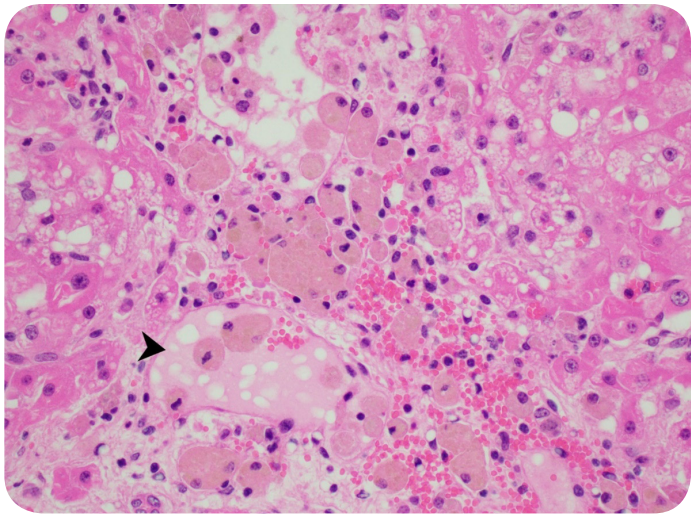


Figure 3. Sheep liver with clusters of ceroid-lipofuscins engorged macrophages and hepatocytes, sloughing into hepatic sinusoids and central veins (arrowhead). Prominent fatty change of the surrounding hepatocytes (H&E stain 40 X magnification).

Diagnostics

Plasma methylmalonic acid (MMA) quantification has been developed into a highly sensitive and specific indicator of vitamin B_{12} and cobalt deficiency, with target plasma levels being < 3 µmol/L, deficiency inducing raised levels of plasma MMA. As ruminants require dietary cobalt to synthesise vitamin B_{12}, cobalt deficiency effectively causes vitamin B12 deficiency.

Plasma MMA is currently considered the most reliable diagnostic assay for cobalt deficiency in live animals. This analysis is also widely employed to evaluate the efficacy of supplementation with injectable vitamin B_{12} or oral cobalt in deficient animals.

Effective supplementation with vitamin B_{12} reduces plasma MMA levels to normal within ten (10) days, while with adequate oral supplementation MMA levels only reduce to normal after around 30 days.

At post-mortem, quantitative analysis of fresh liver tissue for vitamin B12 and cobalt can be performed. The critical minimal tissue level for vitamin B12 on a dry weight basis is 0.20 ppm, while the hepatic target range for cobalt is 0.03 to 0.1 ppm.
Pasture mineral analyses do not routinely check for cobalt levels as it is not an essential nutrient for plants, so locating a pasture laboratory with a validated cobalt assay can prove problematic. Soil tests for cobalt are unreliable.

Treating and preventing cobalt deficiency

Vitamin B12 injection provides the most rapidly acting therapeutic option. A single injection of vitamin B12 at the manufacturer's recommended dose will prevent the development of deficiency for 6–8 weeks. Injectable vitamin B12 is the preferred method used in lambs and calves pre-weaning. It can also be used in animals due for sale in the following two months (prime lamb, beef weaners).

Intraruminal cobalt pellets, applied via a 'bulleting gun', are better for long-term prevention of deficiency. In sheep, pelleting is best done when lambs are weaned. Pellets are not suitable for lambs less than eight weeks old. Cattle should be older than two (2) months with a functional rumen. One pellet is effective for 1–3 years.

Cobalt licks are available, but the intake is highly variable; drenches are only effective for about two (2) weeks and therefore impractical. Pasture topdressing has proven unsuccessful in most countries.

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QUESTION 1

Cobalt is essential for which metabolic process in ruminants?
a. Synthesis of vitamins B1
b. Synthesis of vitamin B2
c. Activation of vitamin C
d. Synthesis of vitamin B12
e. Activation of vitamin B 6

QUESTION 2

Why is cobalt deficiency most commonly reported in ruminants grazing lush pasture?
a. The rapid uptake of other elements that bind cobalt in the plant makes it unavailable
b. There is reduced soil contamination of lush pasture, reducing the amount of soil making up the dry matter intake
c. Fertilisation of pasture binds cobalt in the soil
d. Irrigation or high rainfall depress the uptake of cobalt by the pasture
e. Rapidly growing pasture has no cobalt requirement and reduces uptake to replace with other essential minerals

QUESTION 3

Which of the following ages of sheep have the highest requirement for cobalt?
a. Neonatal lambs
b. 6 to 8-week old lambs
c. Weaners
d. Yearling sheep
e. Adults

QUESTION 4

In which type of soil is cobalt deficiency most likely to occur?
a. Clay soils
b. Peaty soils
c. Sandy soils
d. Silty soils
e. Loamy soils

QUESTION 5

Which of the following clinical signs of cobalt deficiency is unique to sheep?
a. Ill thrift
b. Weight loss
c. Reduced appetite
d. Anaemia
e. Excessive lacrimation

QUESTION 6

Which of the following gross post-mortal lesions is a consistent finding in sheep but not cattle with cobalt deficiency?
a. Emaciation with serous atrophy of fat
b. Anaemia
c. Hydrothorax and hydropericardium
d. Pale white liver
e. Ascites

QUESTION 7

What are the characteristic histological features of WLD?
a. Fatty liver with ceroid lipofuscin pigment in hepatocytes and macrophages
b. Hepatic cirrhosis with iron pigment in portal macrophages
c. Hepatic hydropic degeneration with portal fibrosis and centrilobular accumulation of copper pigment
d. Centrilobular hepatic necrosis with haemorrhage and hemosiderin pigment in sinusoidal macrophages
e. Periportal hepatic necrosis with cholestasis

QUESTION 8

Which is considered the most reliable assay for diagnosing cobalt deficiency in live ruminants?
a. Serum cobalt
b. Serum vitamin B12
c. Plasma methylmalonic acid (MMA)
d. Plasma cobalt
e. Plasma vitamin B12

QUESTION 9

When evaluating a flock or herd for evidence of cobalt deficiency, which of the following assays are considered unreliable?
a. Liver tissue cobalt levels
b. Liver tissue vitamin B12 levels
c. Evaluation of levels of cobalt and pasture
d. Serum vitamin B12 levels
e. Soil testing for cobalt

QUESTION 10

Which of the following treatment regimes provide the most rapidly acting therapeutic and preventive option for addressing cobalt deficiency?
a. Cobalt drenches
b. Intra-ruminal cobalt pellets
c. Vitamin B12 injection
d. Cobalt licks
e. Pasture top dressing with cobalt

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