6. Cobalt/Vitamin B12

Vitamin B12 is synthesized by rumen microbes from dietary cobalt and the clinical signs of cobalt deficiency in sheep and cattle are due to reduced vitamin B12 concentrations in tissue. Vitamin B12 coenzyme is required by methylmalonyl-CoA mutase, a mitochondrial enzyme involved in the major pathway through which propionate and several amino acids are metabolised. Propionate, derived from fermentation of plant cellulose in the rumen, is the major source of energy in the ruminant (Mills 1981).

In plants, cobalt appears to be required only for the vitamin B12 nutrition of the nitrogen-fixing Rhizobia in legume nodules.
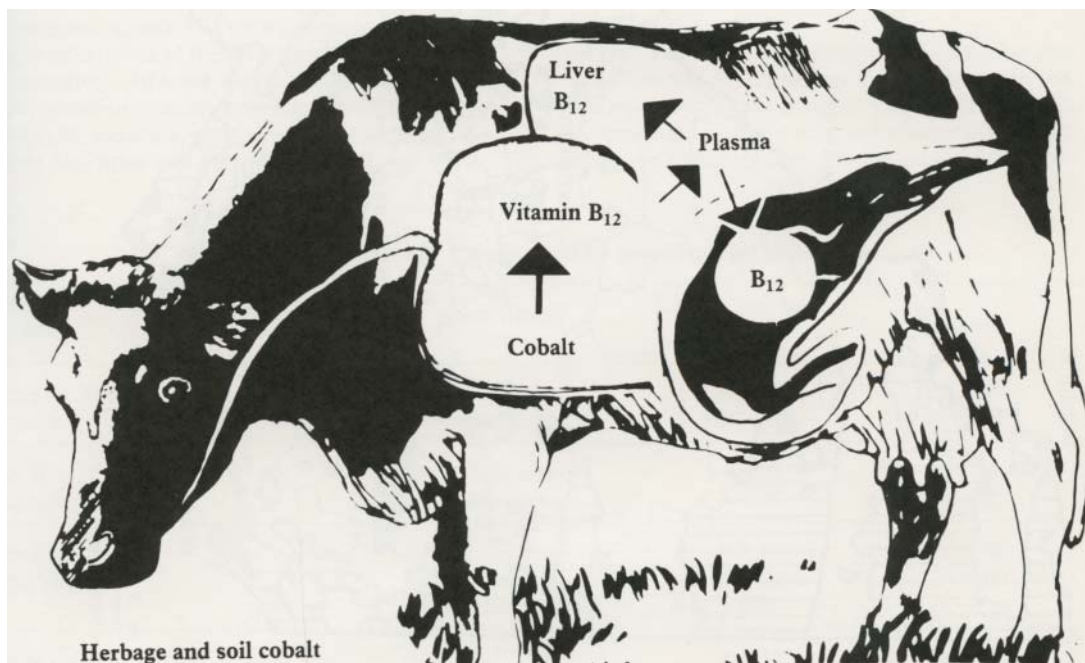
6.1 Occurrence of cobalt deficiency in Victoria

Cobalt deficiency in sheep was first described in the Dandenong Ranges in 1957 by Skerman and Sutherland. Since then, other areas deficient in cobalt for livestock have been identified (figure 6.1) in the Western District, the Otways, South and East Gippsland and in the Strathbogie Ranges of Central Victoria (Allen 1979, Allen and Love 1982, Edwards 1962, Halpin 1979, Hardefeldt et al. 1982, Margetts *et al.* 1961, Mitchell *et al.* 1982, Napthine *et al.* 1982, Rentsch 1980).

Pasture dry matter responses to cobalt application have been recorded at six sites and a lucerne response at one site, all in the west and south-west Wimmera (Eales and Dykstra 1982).

Coastal calcareous sands are commonly associated with cobalt deficiency in grazing animals (Margetts *et al.* 1961). Generally, however, soil type, soil cobalt concentrations and soil cobalt availability to plants are poorly correlated. For example, the red kraznosem soils of Gippsland have a high total cobalt concentration but a low level of cobalt available to plants (Nicolls and Honeysett 1964). Manganese oxide minerals have been shown to strongly bind free soil cobalt to their surfaces rendering it unavailable to plants (Anon. 1969).

Liming has been associated with reduced cobalt uptake in pastures (Adams *et al.* 1969) and applications of superphosphate have been related to reduced vitamin B12 concentrations in grazing sheep (Halpin *et al.* 1981). On two properties in Victoria, cobalt deficiency has occurred in flocks grazing newly established pastures sown on paddocks where potatoes had previously been grown on Krasnozem soil with heavy superphosphate use (Allen and Love 1982).
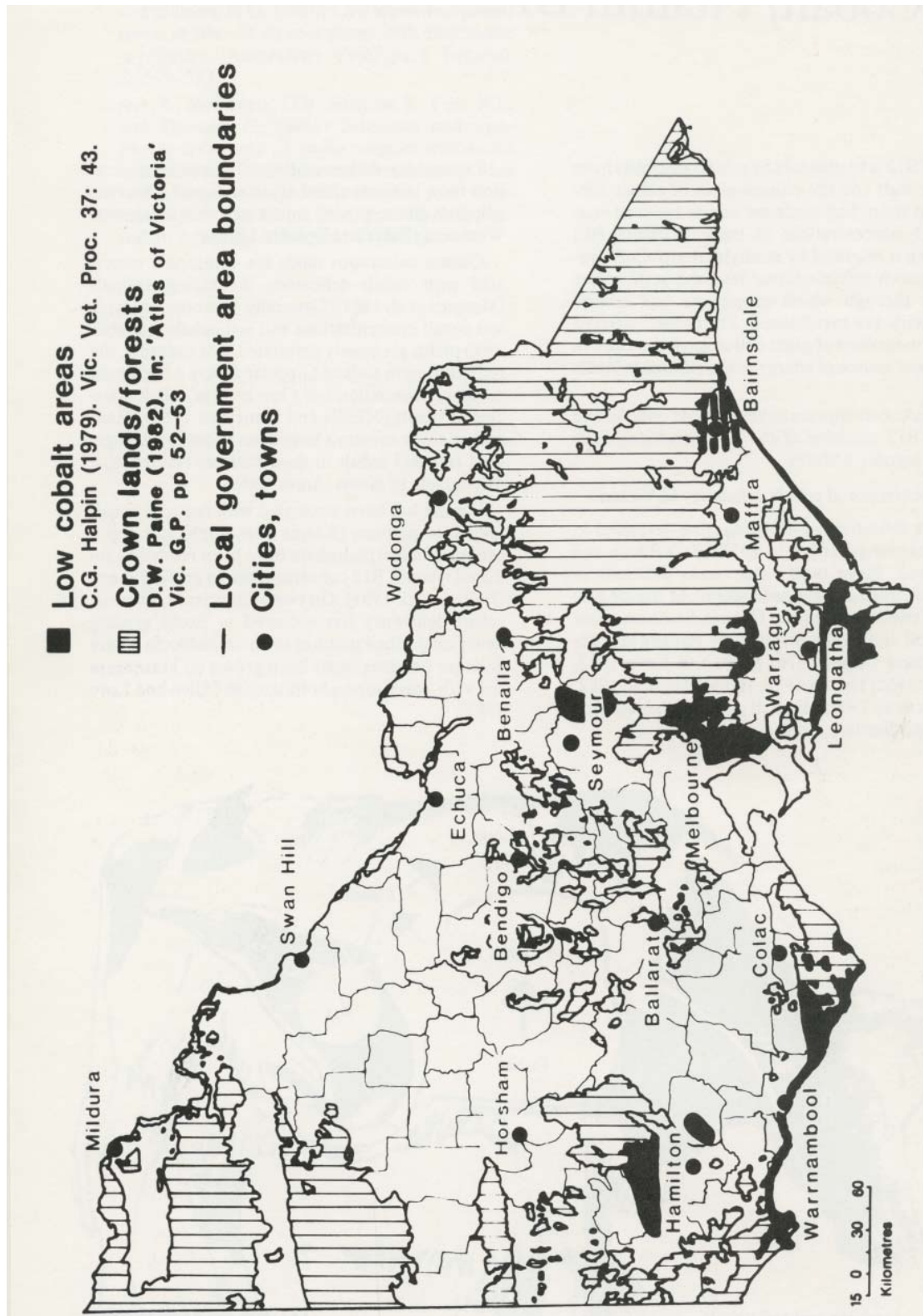

Herbage and soil cobalt

Figure 6.1: Areas where cobalt deficiency has been detected in livestock in Victoria based on original data by Halpin (1979).

Legend:
- **Low cobalt areas**
  C.G. Halpin (1979). Vic. Vet. Proc. 37: 43.
- **Crown lands/forests**
  D.W. Paine (1982). In: 'Atlas of Victoria'
  Vic. G.P. pp 52–53
- **— Local government area boundaries**
- **● Cities, towns**

Seasonal variations in cobalt nutrition are significant, concentrations of cobalt in pastures and plasma vitamin B12 in livestock being lowest in spring. In addition, there are large fluctuations in available cobalt between years (Gardiner 1977). Seasons favoring lush pasture growth favor development of cobalt deficiency. This effect has been attributed to animals ingesting less soil when grazing lightly stocked, rapidly growing pastures. Soil provides a more concentrated source of cobalt to the ruminant than pastures.

More than 60 documented cobalt supplementation trials have been conducted on sheep throughout Victoria during the past 20 years, and less than 10 with cattle. Large body weight responses (5.6 kg in 5 weeks) and wool yield gains (0.7 kg in 6 weeks) in treated lambs were recorded at Lavers Hill in the Otways Ranges (Edwards 1962, Allen and Love 1982). Even in known low-cobalt areas clinical signs of cobalt deficiency, or responses to cobalt supplementation, do not occur every year.

6.2   Signs of cobalt deficiency in livestock

Cobalt deficiency causes unthriftiness and eventually death if sufficiently severe and prolonged. The signs of cobalt deficiency are similar to those of malnutrition and include emaciation, poor appetite, weeping 'rheumy' eyes, anaemia and de-creased milk production. Young animals are most susceptible and lambs are more susceptible than calves. Cobalt deficiency has not been reported in grazing horses, even in areas where sheep and cattle soon waste and die.

Cobalt deficiency predisposes sheep to white liver disease (Mitchell *et al.* 1982, Napthine *et al.* 1982). This disease has been observed in flocks in most of the low cobalt areas of Victoria and is characterised by fatty infiltration of the liver and low serum B12 concentration. Affected sheep are unthrifty and show photosensitization associated with the liver damage, for example, scaly ears. Nervous signs including blindness and convulsions have also been observed. Severe cobalt deficiency has not been observed in cattle in Victoria, although marginal cobalt nutrition has been associated with a high incidence of ketosis and poor body condition in lactating cows, and poor growth of heifers (Blockey 1975).

6.3   Diagnosis of cobalt deficiency 6.3.1 Livestock

In sheep, cobalt nutrition is most conveniently assessed by determination of the vitamin B12 concentration in blood plasma or serum (table 6.1). No differences are observed between vitamin B12 concentrations in plasma and serum. Only blood samples from weaned lambs or adult sheep should be used to assess the cobalt nutrition of a flock because plasma vitamin B12 concentrations are normally low in preruminant lambs (see figure 6.2).

While cobalt intake is the major determinant of synthesis of vitamin B12 in the rumen, other factors are important. As well as vitamin B12 (cyanocobalamin), several natural analogues of the vitamin are also synthesised. In sheep, they are not absorbed and cannot be detected in the plasma. This situation is different in cattle where the analogues appear to be absorbed, and may constitute a variable proportion of the total plasma vitamin B12 level as measured by many laboratory assays (Halpin 1982, Mills 1981). In cattle, the relationship between cobalt nutrition and plasma vitamin B12 concentration is less precise than for sheep.

Plasma vitamin B12 concentrations in sheep respond rapidly (24 hours) to oral cobalt supplements. The same is not true for cattle but the mechanism for this difference has not been explained.

Liver vitamin B12 concentration indicates the tissue vitamin B12 reserves and is the preferred index of cobalt intake in cattle. Under normal grazing conditions, where seasonal changes in cobalt intake are relatively slow, plasma values closely reflect liver values in sheep. However, poor correlations between plasma and liver vitamin B12 values have been observed following supplementation of deficient animals, because of the differences in half life of vitamin B12 within these tissues.

Measurement of total liver cobalt provides an indirect measure of the biologically active cobalt, that is, vitamin B12. In sheep with adequate cobalt nutrition most of the liver cobalt can be accounted for as vitamin B12, whereas in cobalt deficiency only about one-fifth of the liver cobalt exists in this form.

Cobalt deficiency in sheep and cattle occurs when pasture cobalt concentrations are less than 0.10 mg/kg dry weight (Halpin and Caple 1982).

Table 6.1: Biochemical values used to assess cobalt nutrition

Indicator	Nutritional Status[1]	Sheep	Cattle
Plasma vit B12 nmol/l	Deficient	< 0.40	< 0.10
	Adequate	0.70-7.00	0.10-2.00
Liver vit B12 nmol/kg wet wt	Deficient	<100	<75
	Adequate	> 220	> 220
Liver cobalt umol/kg DM	Deficient	< 1.00	< 1.00
	Adequate	1.00-10.00	> 1.00
	Toxic	15.00	—
Pasture cobalt mg/kg DM	Deficient	< 0.10	< 0.10

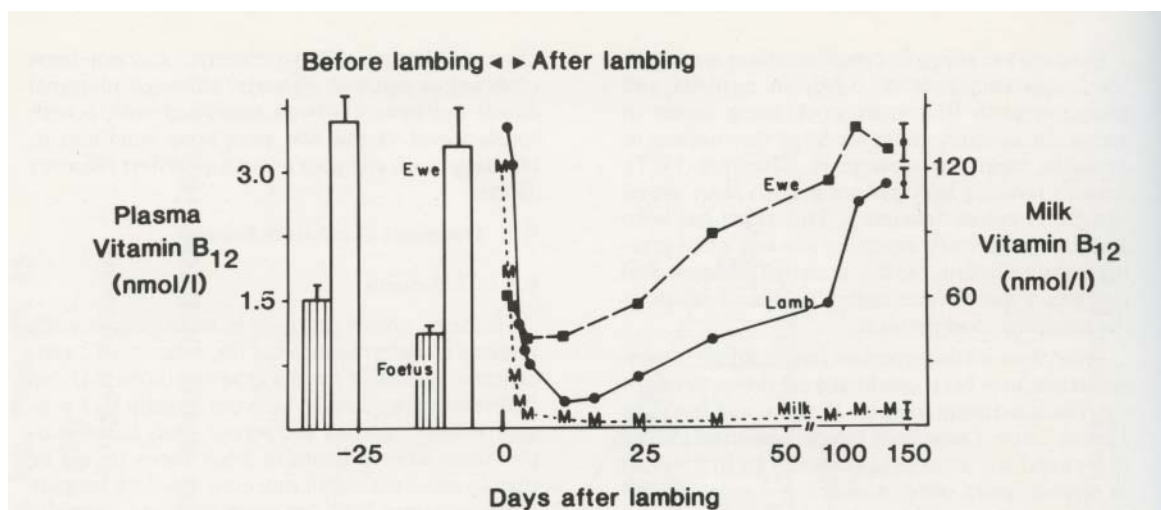1. Marginal nutritional status defined as range between adequate and deficient levels (see section 1.5).

Figure 6.2: Changes in plasma vitamin B12 concentration in lambs before and after birth and in plasma and milk of ewes (Halpin and Caple 1982).

6.3.2   Plants

Critical concentrations of cobalt for clover growth are in the order of 0.04 mg Co/kg dry matter (Ozanne *et al*. 1963). Pasture test strips could be used for diagnosing Co deficiency for legume species in specific areas (Eales and Dykstra 1982), but responses are likely to be rare. No responses have been obtained in grasses even with levels as low as 0.01 mg Co/kg (Ozanne *et al*. 1963).

6.3.3   Soils

The relationship between the various soil tests for cobalt and cobalt availability to herbage and animals is poorly defined and of little diagnostic significance (Gardiner 1977).

6.3.4   Analyses available

Vitamin B12 assays are available at 'Attwood' Veterinary Research Laboratory and Bairnsdale Regional Veterinary Laboratory.

Liver cobalt assays are performed at the Hamilton Regional Veterinary Laboratory.

Soil and pasture cobalt analyses are not available from the State Chemistry Laboratory for diagnostic purposes.

6.4   Treatment of cobalt deficiency

6.4.1   Immediate treatment for animals

Vitamin B12 injections are recommended for the immediate treatment of deficient animals showing clinical signs of malnutrition. Injections of 2 mg to lambs and sheep and 4 mg to calves provide adequate vitamin B12 for three months. Cobalt pellets may also be used for immediate treatment of deficiency and last 12 months. The choice between using pellets or vitamin B12 injections is dependent on relative cost effectiveness and the time period for which animals require supplementation.

6.4.2   Long-term treatment and prevention

Cobalt deficiency can be prevented by a range of cobalt supplements—drenches, licks, foliar sprays and fertiliser top-dressing, as well as pellets and vitamin B12 injections. Vitamin B12 is only syrithesised from dietary cobalt in the rumen and is poorly absorbed. Therefore, animals require regular and frequent oral dosing with cobalt to satisfy their

vitamin B12 requirements and drenches must be administered weekly or at most fortnightly to be completely effective.

Salt licks or mineral mixes containing cobalt should provide a minimum of 0.05 mg cobalt (equivalent to 0.24 mg cobalt sulphate) per sheep per day. Some or all animals may fail to lick the salt block or mix and this often compromises the efficacy of this method of treatment.

Pasture top-dressing has been the recommended method of treatment of cobalt deficiency in New Zealand, particularly on pumice land. For many years an annual application of 350 g cobalt sulphate per hectare, in superphosphate, has been recommended. More recently it has been found in New Zealand that where previous cobalt applications have raised cobalt levels in the pasture to an adequate concentration, for example, after ten applications, the frequency and rate of application can be reduced to 175 g/ha every three years, (Sherrell and Percival 1984, pers. comm.).

Australian and overseas data indicate that pasture topdressing is often not successful in preventing cobalt deficiency in animals (Anon. 1969, McLaren *et al*. 1979) and suggests that the short-term benefits of cobalt fertilisers may be the result of direct ingestion of the fertilisers. The efficacy of cobalt fertilisers is strongly affected by soil type. Uptake of cobalt by plants on calcareous soils, and on soils high in manganese (above 1000 ppm total soil manganese) may be negligible (Anon. 1969).

Cobalt pellets introduced orally are the most efficient method of prevention because they continually release trace amounts of the element directly into the rumen and should last the lifetime of the animal. The pellets do have some disadvantages. A small percentage may be regurgitated and lost. Pellets should only be administered to animals having a functional rumen (older than two months). In animals grazing on calcareous soils the pellet may become coated with a calcium phosphate deposit which reduces the rate of cobalt release from the pellet. The provision of a grinder with the pellet helps to remove the deposit.

Cobalt supplementation of ewes after parturition is not a suitable means of supplementing lambs. Ewe's milk, even in ewes supplemented with cobalt, provides only a fraction of the lamb's daily requirement. Ewes grazing cobalt deficient pastures should receive cobalt bullets at least eight weeks before lambing to ensure adequate vitamin B12 reserves in the foetal liver and colostrum. An injection of 2 mg of vitamin B12 at six to eight weeks of age will provide adequate vitamin B12 for lambs until they are weaned.

6.5   Cobalt toxicity

Cobalt is not very toxic, for example, daily doses of 3 mg Co/kg bodyweight can be tolerated by sheep for many weeks (Andrews 1965). Daily doses of 10 mg

Co/kg bodyweight depress appetite and bodyweight and some deaths may occur. A single dose of 40-60 mg Co/kg bodyweight, as a soluble salt, may be fatal to sheep (Andrews 1965). Cattle appear to be less tolerant to cobalt than sheep.

6.6   Phalaris staggers

Phalaris staggers is a disease mainly affecting sheep grazing pastures containing Phalaris aquatica. The disease is characterised as a chronic nervous disorder from which sheep never recover (Southcott 1956). The etiology of phalaris staggers is unknown. Although tryptamine alkaloids were thought to be associated with phalaris staggers, the infusion of tryptamine alkaloids isolated from phalaris into normal or cobalt-deficient sheep has not reproduced the syndrome (Seawright 1982).

In cobalt-deficient areas, phalaris staggers has been prevented by treatment of animals with cobalt drenches or pellets, or by top-dressing pastures with cobalt. Injectible vitamin B12 preparations are not effective in preventing the disorder and cobalt is not effective in treating affected sheep. The conclusion is that sheep in the low cobalt areas should receive cobalt pellets to prevent phalaris staggers if they have access to phalaris pastures.

Other syndromes in sheep and cattle grazing phalaris pastures include sudden death due to heart failure, and an acute nervous disorder from which affected animals may recover. These other syndromes have been attributed to poisoning by tryptamine alkaloids in phalaris. Cobalt does not appear to protect against the sudden death or acute nervous syndrome.

Where sheep show nervous signs while grazing phalaris in areas other than those recognised as being low in cobalt, the sheep should be examined by a pathologist. Pathology consistent with phalaris staggers, and low cobalt concentrations in the sheep, indicate that sheep should receive cobalt bullets. Where the phalaris cobalt levels are greater than 0.3 mg/kg, or plasma vitamin B12 concentrations are greater than 0.7 nmol/l, sheep are unlikely to benefit from cobalt supplementation (Southcott 1956).

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