4. Selenium

Selenium is an essential element for animals, but not plants (Underwood 1981). Selenium is required by sheep and cattle for growth, and for prevention of selenium-responsive myopathy (white muscle disease). A number of other nutritional factors including vitamin E may be involved in the development of myopathy. Selenium also appears to have a role in the resistance of animals to disease, being involved in the production of antibodies and in the killing of micro-organisms engulfed by macrophages (Boyne and Arthur 1979). The dietary requirement of sheep and cattle is 0.05 to 0.1 mg Se/kg (Underwood 1981). Many pastures in Victoria contain much less than this concentration of selenium (Caple et al. 1980).

4.1 Occurrence of selenium deficiency in Victoria

Selenium deficiency, first identified in the early 1970s in Victoria, causes only minor production loss to the grazing industries on a statewide basis, but is important in sheep in some areas (Caple *et al*. 1980b, McDonald and Caple 1977).

The marginal selenium areas (figure 4.1) have been broadly defined based on the activity of the selenium-containing enzyme glutathione peroxidase (GSHPx) in blood of sheep and cattle (Caple *et al*. 1980). In the marginal selenium areas sheep may have mean blood GSHPx activity less than 50 units, and cattle less than 40 units (1 unit = 1 umol substrate oxidised per minute per gram haemoglobin). These activities correspond to a blood selenium concentration less than 0.5 umol/l, and a dietary selenium concentration of approximately 0.02 mg Se/kg dry matter (Caple *et al* 1980, Halpin *et al*. 1981).

There is a marked seasonal variation in the selenium nutrition of grazing livestock, with lowest levels occurring in spring and summer (Caple *et al*. 1980). There is also a variation between years. White muscle disease in lambs and calves in spring is most prevalent in years when there is good autumn rain-fall and abundant clover growth in spring (McDonald and Caple 1977). Heavy applications of superphosphate decrease the concentration of selenium in pastures and may also decrease the up-take of selenium by grazing livestock (Caple *et al*. 1980, Halpin *et al*. 1981) and predispose them to white muscle disease (McDonald and Caple 1977).

Apart from low selenium nutrition, other factors may be required to induce white muscle disease. These factors include stress, unaccustomed exercise, unsaturated fatty acids in clover pastures, and vitamin E deficiency. White muscle disease occurring in weaner lambs grazing oat stubbles or dry pastures in the autumn is thought to be due to low vitamin E nutrition (see section 5).

4.2 Signs of selenium deficiency in livestock

Selenium-responsive white muscle disease has been observed in lambs, calves, foals and kids in Victoria (Caple *et al*. 1980, McDonald and Caple 1977, Caple *et al*. 1978, Allen and Friend 1978). Lesions occur in skeletal and/or heart muscle. The clinical signs of white muscle disease vary according to the particular muscle groups affected. Skeletal muscle groups commonly affected include those of the upper fore and hind limbs, and affected animals walk with a stiff-legged gait or are unable to stand. Lesions in heart muscle may produce sudden death, and in intercostal muscles may produce respiratory distress.

In sheep, the disease is observed mainly in spring-born lambs on clover-dominant pastures, and also in weaner sheep grazing stubble pastures. The disease in calves may be precipitated by stresses such as yarding and weaning (Allen and Friend 1978). Usually only a small percentage of animals (less than 10% of a flock or herd) shows signs of white muscle disease. Lambs are more commonly affected in Victoria than other animals.

Selenium-responsive illthrift: More than 100 selenium response trials have been conducted during the past 20 years in Victoria. In low selenium areas, some treated lambs have shown increased body weight and wool weight gains and reduced mortality (McDonald 1975, Paynter *et al*. 1979, Paynter *et al*. 1982). The majority of trials have been con-ducted on Merino lambs. The increased body weight gains have been of the order of 2 kg in 10 weeks and the wool weight gains of the order of 0.4 kg in 12 months. These responses were obtained in flocks with very low selenium nutrition (blood GSHPx less than 20 units) (Paynter *et al*. 1979, Paynter *et al*. 1982). However, responses are not always obtained at this level of selenium nutrition (McCaughan 1979, Gill 1977).

Usually the lambs with illthrift in the low selenium areas have no evidence of myopathy, and there is a response to selenium but not to vitamin E. In contrast, some myopathies in lambs, such as those induced by feeding diets high in polyunsaturated fatty acids, are responsive to vitamin E and not to selenium. Myopathies in housed fine wool Merino sheep fed oat and lupin grain diets are responsive to vitamin E and not selenium. Increases in body-weight and wool growth have occurred after supplementation of these sheep with vitamin E (see section 5).

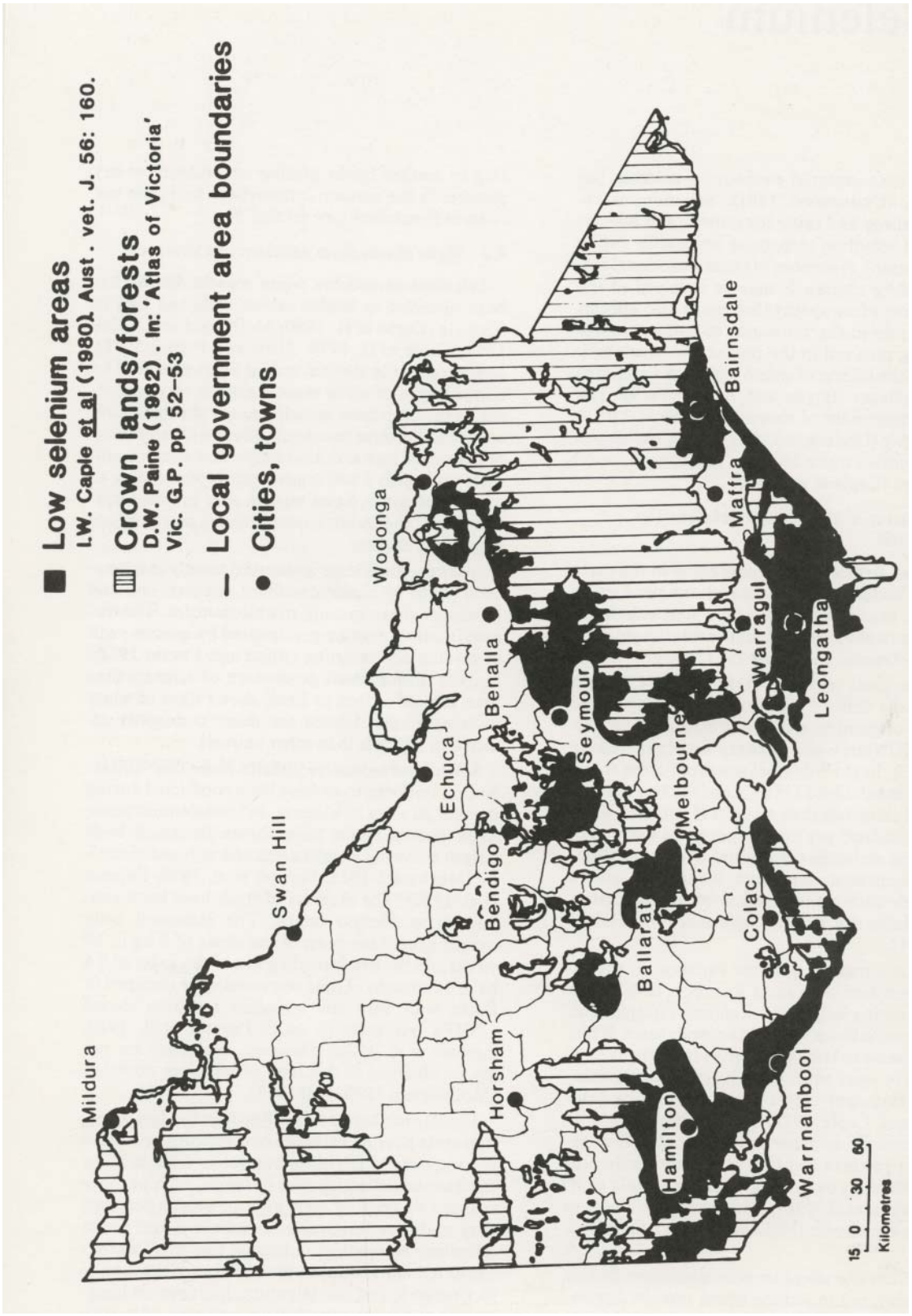

Figure 4.1: Map of Victorian showing marginal selenium areas based on original data by Caple *et. al.* (1980). Within the black shaded areas sheep may have blood glutathione peroxidase activities less than 50 units, cattle less than 40 units and blood selenium less than 0.5 umol/l in spring

Supplementation of calves with selenium has affected growth in only one of about 30 trials conducted in Victoria over the past 14 years (1970-1984) (Sully *et al*. 1982). The trials have included calves having very low blood GSHPx activities, for example, less than 10 units.

Infertility: Infertility in ewes due to increased embryonic mortality has been associated with selenium deficiency in other States, but trials conducted in the low selenium areas in the Western District have shown selenium supplementation to have no effect on ewe fertility (Hardefeldt 1978 a, b).

Retained foetal membranes in cows has not been associated with selenium deficiency in Victoria, and production responses have not been obtained in supplementation trials on dairy cows in low selenium areas of South Gippsland (Caple et al. 1982).

4.3 Diagnostic methods 4.3.1 Livestock

The selenium nutrition of grazing livestock can be assessed from blood and liver selenium levels (table 4.1). The discovery in 1972 that selenium was an essential component of the enzyme glutathione peroxidase revolutionised the laboratory diagnosis of selenium deficiency. Measurement of the enzyme activity is more convenient and more accurate than assays of the element in blood, especially at the levels found in selenium-deficient animals. The enzyme assay requires less sophisticated equipment (an ultraviolet spectrophotometer) than the elemental selenium assay. Greater than 95% of blood enzyme activity is associated with the erythrocytes and whole blood samples must be collected with anticoagulant.

In the diagnosis of selenium-responsive white muscle disease it is important to determine whether the animal has muscle damage, that other diseases such as arthritis are not the cause of lameness, and that blood glutathione peroxidase is low. Muscle damage in sheep and cattle can be assessed by an increase in the plasma activity of enzymes such as creatine kinase and aspartate amino transferase which are released into the plasma from damaged muscles and confirmed by histopathology. Creatine kinase is specific for heart and skeletal muscle damage whereas aspartate amino transferase is also released with liver damage.

Selenium appears to be incorporated into erythrocyte glutathione perioxidase only during formation of erythrocytes. Blood activities of the enzyme are therefore a reflection of the dietary intake several months previously, and of erythrocyte turn-over.

Blood glutathione peroxidase activity is not affected by age of the animal, at least for sheep, cattle and horses (Paynter *et al*. 1980).

The relationship between pasture selenium concentration and the selenium nutrition of animals grazing these pastures has been investigated in several areas of Victoria (Caple *et al*. 1980, McDonald and Caple 1977). In general, concentrations in pasture whole tops less than 0.02 mg Se/kg DM are associated with deficient levels of both selenium and glutathione peroxidase in blood of grazing animals, and pasture concentrations greater than 0.05 mg Se/kg DM are adequate for grazing livestock. Plant concentrations ranging down to 0.01 mg Se/ kg DM have been measured in Victorian pastures (Caple *et al*. 1980). However animal tests are the preferred method of diagnosis of selenium deficiency of livestock.

Table 4.1: Biochemical values used to assess selenium nutrition

Indicator	Nutritional Status[1]	Sheep	Cattle
Blood GSHPx	Deficient	< 20	< 10
Units[2]	Adequate	50-550	40-300
Blood selenium	Deficient	< 0.25	< 0.25
umol/l	Adequate	> 0.50	> 0.50
Liver selenium	Deficient	< 2.50	< 2.50
nmol/kg DM	Adequate	> 5.00	> 5.00
	Toxic	> 20.00	> 20.00
Pasture selenium	Deficient	< 0.03	< 0.02
mg/kg DM	Adequate	> 0.05	> 0.05
	Toxic	> 5.00	> 5.00

1. Marginal nutritional status is defined as the range between the adequate and deficient values (see section 1.5)
2. units = umol substrate oxidised per minute per gram haemoglobin

Soil selenium levels are insensitive indicators of animal selenium status.

4.3.2 Plants

Selenium is not essential for the growth of plants.

4.3.3 Analyses available

Blood glutathione peroxidase assays are conducted at Regional Veterinary Laboratories and the Attwood Veterinary Research Laboratory. Elemental selenium analyses on blood, tissue or herbage samples are not available as a routine diagnostic test, and are only conducted in special investigations.

Soil selenium analyses are not available.

4.4 Treatment for selenium deficiency

It is important that an assessment of the selenium nutrition be made before animals are treated because many selenium compounds are toxic in excess.

4.4.1 Immediate treatment of animals

Lambs and calves affected with white muscle disease are treated with intramuscular injections or oral drenches of selenium usually in the form of sodium selenite or sodium selenate (table 4.2). The dose rate is 0.1 mg Se/kg body weight. All lambs and calves in the affected group should be treated. Animals with muscle damage should be handled carefully during treatment to avoid precipitating further damage.

4.4.2 Long-term treatment and prevention of selenium deficiency

In the low selenium areas of the State, lambs can be treated with selenium at 4 to 6 weeks of age and again at 3 months of age. The dose rate is 0.1 mg Se/kg body weight. Lambs can receive further doses of selenium at intervals of three months until fully grown.

Selenium can be administered with pulpy kidney and pulpy kidney-tetanus vaccines which are available commercially. Selenium can be mixed with worm drenches, but the manufacturers' directions should be followed closely. These mixtures should be used within 24 hours of preparation.

In Victoria, no responses to selenium treatment have been observed in adult sheep or cattle. There appears to be no basis for recommendation of selenium treatment for adult animals except where pregnant ewes or cows are deficient in selenium and where their spring-born lambs and calves need to be protected from white muscle disease in the first few weeks after birth. For this situation, treatment four weeks before parturition is recommended.

Selenium pellets are available commercially. These pellets were developed by CSIRO and are designed to be retained in the forestomachs and to provide long-term (up to five years) selenium supplementation. Some commercial batches have not reproduced the efficacy of the original pellets, and have not released adequate selenium for extended periods. Until the efficacy of commercial pellets has been confirmed their use cannot be recommended as a substitute for regular selenium drenching. If pellets are used, their effectiveness should be monitored by measurement of blood glutathione peroxidase activity in treated sheep.

A mixture of selenium and superphosphate from the Phosphate Cooperative Company of Australia (commonly known as Pivot Fertilisers in Victoria) has now been registered for use in Victoria. The mixture has proved a useful alternative method of supplementing grazing livestock in low selenium areas of Victoria. Selenium in the fertiliser is en-capsulated within water-soluble pills, which readily break down following application to pasture, releasing the selenium for uptake by the plant roots.

Experiments at two sites in low selenium areas of Victoria have demonstrated that a single application of 10 g selenium per hectare, applied mixed with superphosphate in late autumn, was effective for sheep for at least 12 months and protected autumn-drop calves till they were sold in summer (Halpin *et al*. 1984). Pasture selenium concentrations responded within a few days to the fertiliser application and then gradually returned to base levels over approximately six to 12 months depending on the soil type. The effect on grazing animals was longer because of the selenium reserve built up in body tissues, and ensured adequate selenium for at least 12 months in sheep.

A range of selenium inclusion rates has been registered to satisfy the range of superphosphate rates usually applied to pasture in Victoria. The maximum permitted level (0.02%) corresponds to 200 grams of selenium per tonne of superphosphate and when the mixture is applied at the recommended rate of 50 kg/ha will provide the required 10 g selenium per hectare. Mixtures have been registered to supply 10 g selenium per hectare from superphosphate rates of 50, 100, 125, 200, 250 and 375 kg/ha.

4.5 Selenium toxicity

Selenium toxicity in animals is observed when dietary concentrations exceed 5 mg Se/kg (Under-wood 1981). Fortunately, there are no seleniferous (high selenium) areas or selenium accumulator plants in Victoria, unlike some areas of northern Australia (Seawright 1984).

Animals affected with chronic selenium poisoning show loss of appetite, lameness, sloughing of the hooves and loss of hair.

Acute selenium poisoning may be produced in animals with approximately 10 times the therapeutic dose. Signs of acute selenium toxicity include blindness, abdominal pain, excessive salivation, paralysis and death.

Selenium drenches and injections are currently listed under Schedule 4 of the Poisons Act. They may only be obtained by prescription from a veterinary surgeon.

Table 4.2: Selenium treatment for animals

Injectible remedies		
1	Selenium injection	
	Selvite E	Troy Laboratories Pty Ltd 98 Long Street Smithfield NSW
2	Selenium in vaccines	
	Pulpy kidney tetanus vaccine with selenium	Commonwealth Serum Labs 45 Poplar Road Parkville Vic
Oral remedies		
1	Selenium drenches	ICI Australia Ltd
	Selenium Drench Concentrate	1 Nicholson Street Melbourne
	Admin Se Mineral Supplement	Welcome Australia Ltd 145 Heidelberg Road Northcote
2	Selenium pellets	
	Permasel, Selenium pellets	ICI Australia Ltd 1 Nicholson Street
	Selenium pellets (Tri-sel)	Melbourne
3	Selenium fertiliser	Phosphate Co-Op Co. of Australia Ltd
	Super selenium	160 Queen Street Melbourne

Cautionary note

1. The manufacturers' Directions for Use" of remedies should be read and followed. This applies particularly when selenium is to be added to anthelmintics.
2. Care should be taken when calculating doses. The toxic dose is approximately 10 times the therapeutic dose.

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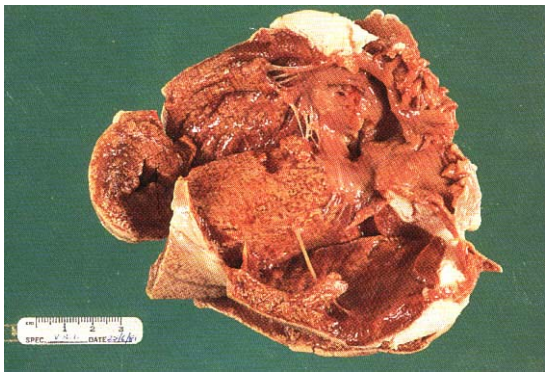
Selenium deficiency: note the pale lesions in this heart tissue which characterise white muscle disease.



In housed sheep, white muscle disease is more usually associated with poor vitamin E nutrition than selenium deficiency.

The watery eye discharge characteristic of cobalt deficiency in sheep.

Note the poor condition of the cobalt deficient sheep compared with the cobalt supplemented animal.