3. Copper

Copper is an essential trace element, being involved in at least 10 enzymes which catalyze oxidase type reactions in both plants and animals. Animals have a higher requirement for copper than plants and are affected by copper deficiency at plant copper levels which do not affect plants and plant growth (Arthur et al. 1981). In animals, copper is required for body, bone and wool growth, for pigmentation, myelination of nerve fibres and leucocyte function (Underwood 1977, Arthur et al. 1981). In plants, copper is required for photosynthesis and nitrogen metabolism, cell wall structure, growth and seed set.

3.1 Occurrence of copper deficiency in Victoria

Copper deficiency in pasture was first diagnosed in Victoria in 1945 (Savage 1974). Most of the areas where copper deficiency in plants and animals has occurred have been recognised for many years (figure 3.1). Local detail is readily available from Departmental offices. Deficient areas have been defined from pasture response trials, and their related soil type, from clinical signs in animals and responses to treatment, and from liver and blood copper concentrations in sheep and cattle. Suspect soil types include coastal and other sands (except Mallee sands), sandy loams of granite or Pliocene origin, loams from sandstone and peaty swamp land (Savage 1974).

Much of this data is historical and should be used only as a guide. It may not reflect the current situation due to more recent fertiliser and pasture renovation practices.

Many of the pastures in the deficient areas have now been top-dressed with copper, and the incidence and severity of copper deficiency in animals and pastures has been greatly reduced. It has been estimated that, in the period of 1971 to 1974 alone, the area of pasture in Victoria top-dressed with cop-per was between 250 000 and 500 000 ha (Savage 1974). The total area where copper may be deficient for livestock is about 2.1 million ha. A single copper top-dressing has provided adequate copper for animal and pasture production for at least 13 years according to recent research in Western Australia (Loneragan et al. 1981).

The level of interest of farmers, veterinarians, and agribusiness in copper supplementation for animal health and production is indicated by the fact that 60% of copper supplements marketed for animals in Australia are sold in southern Victoria.

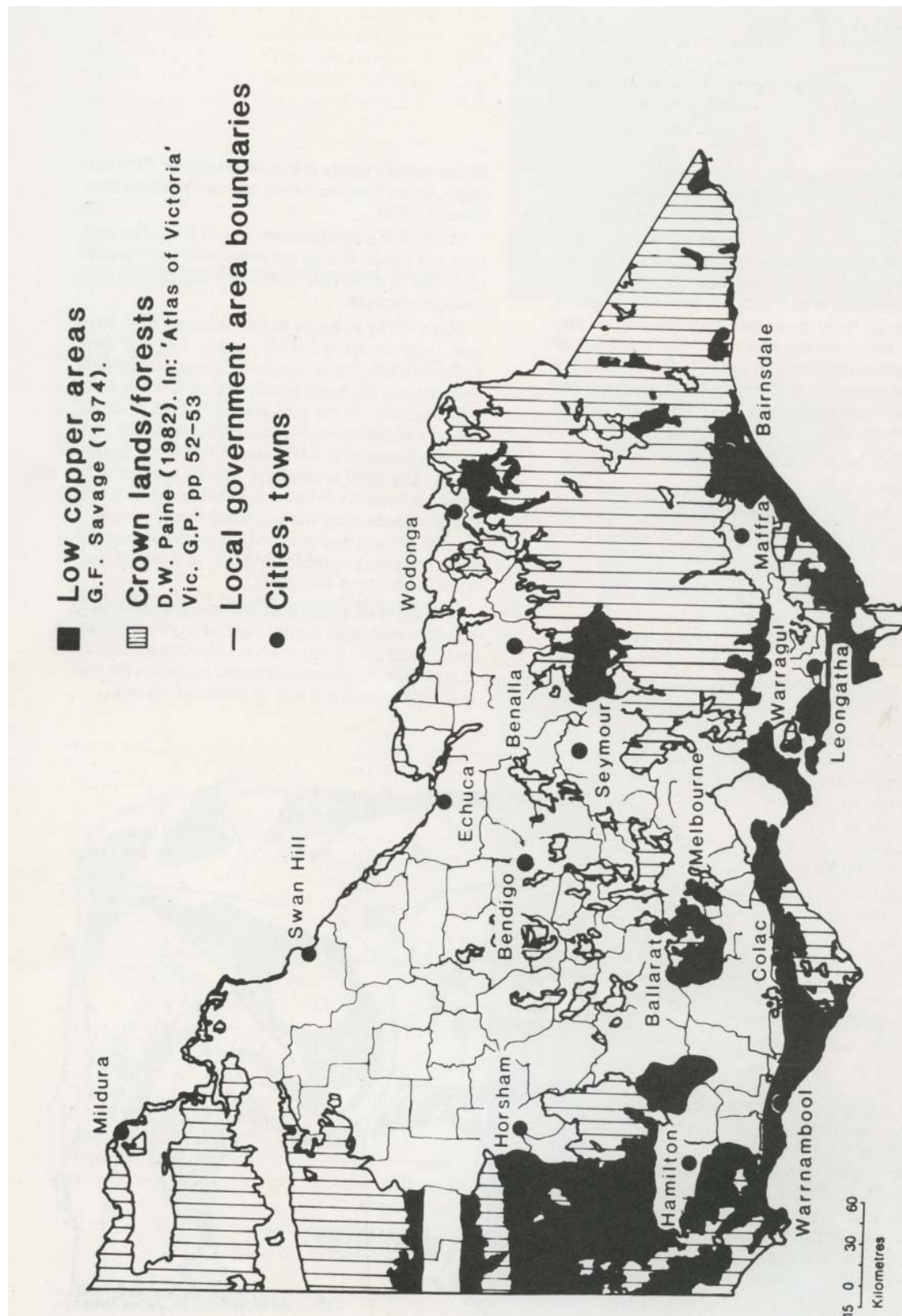Herbage and soil copper (Mo, S and Fe)
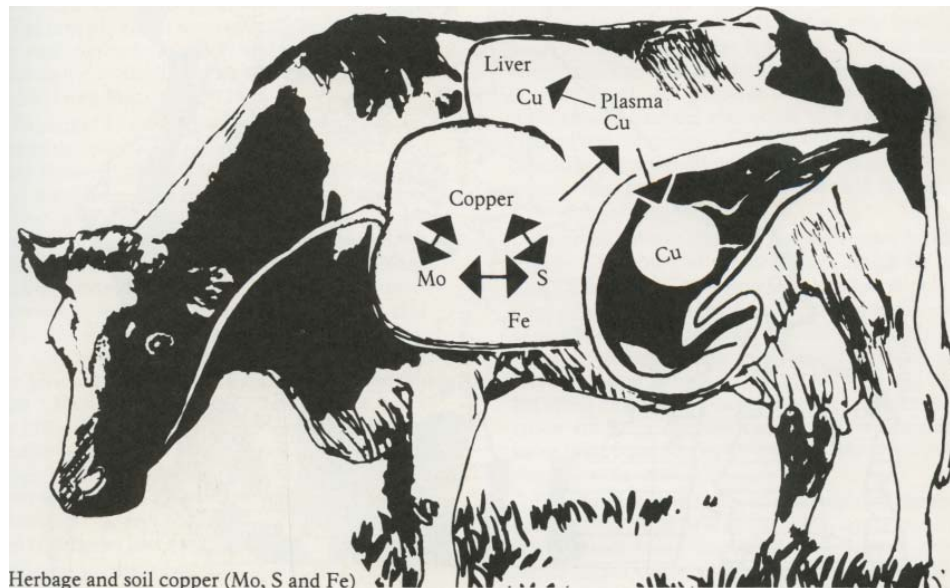
Figure 3.1: Areas where copper deficiency in pasture and livestock has been identified in Victoria, based on the data published by Savage (1974). Many of the pastures in the most deficient areas have now been treated with copper.

Copper concentration in a pasture is affected by the botanical composition, stage of maturity and the season. Grasses generally have lower copper concentrations than clovers and the copper concentration in both declines from winter through to late spring. The copper nutrition of grazing animals depends not only on the herbage copper concentration. It also depends upon the amount of herbage available (which varies markedly with the season, with most being available between August and December) and the availability to the animals of the copper in the herbage.

Over the summer period, the availability to animals of copper present in pastures increases as pastures dry off. This is due at least partially to changes in the forms of copper present (to copper-amino acid forms which are more readily absorbed) and to decreases in feed digestibility which decrease ruminal sulphide production (Underwood 1981). For these reasons, copper deficiency in animals occurs seasonally in Victoria in the winter-spring period and often resolves itself during summer.

High dietary intakes of molybdenum, sulphur, zinc, iron, cadmium and calcium have all been shown to decrease the availability of dietary copper to animals (ARC 1980, Underwood 1977). The severity with which these various factors interact, and the effect they have on specific animal tissues, leads to a variety of copper responsive conditions in animals (Underwood 1977). In the Victorian situation, the interaction between copper and molybdenum is particularly important in determining copper availability to animals (see Mo section). Many of the areas which are marginal or deficient in copper are also deficient in molybdenum (Savage 1974) and molybdenum applications to these areas can exacerbate the copper deficiency. Liming of pastures can result in an overall decrease in cop-per available to both plants and animals and can increase the availability of molybdenum (Drake and Kehoe 1954, Mahoney 1982).

Reports of 41 copper supplementation trials con-ducted on cattle in Victoria between 1970 and 1981 were presented at the regional workshops associated with this review (see bibliography). Increased growth of copper-supplemented animals was observed in 10 of 30 trials conducted in beef herds. Three of these responses were obtained on known high-molybdenum pastures, and the other seven were obtained in trials conducted during the critical period between May and October. Where cop-per supplementation increased growth of cattle, the serum copper concentrations have been less than 5 umol/l, and liver copper less than 0.3 mmol/ kg DM. Cattle responding to copper treatments usually had clinical signs of poor body condition, rough coats, and diarrhoea. In some trials it was observed that the bodyweight differences between treated and control groups had disappeared by the end of summer and autumn.

Eleven of the trials were conducted on dairy herds.

None of the herds had low copper status, according to the serum copper concentrations (all above 8 umol/l) at the beginning of trials. There were no increases in milk production or improvements in fertility due to copper supplementation.

In sheep, growth responses to copper supplementation are less common than in cattle grazing similar pastures. Reports of 15 copper supplementation trials conducted on sheep in Victoria between 1959 and 1982 were presented at the regional workshops (see bibliography). A growth response was observed in only two of the 15 trials. Many of the trials were conducted on properties where symptoms of copper deficiency were either absent or mild, for example, steely wool. These findings suggest that direct copper supplementation of sheep should be restricted to situations where more severe signs of copper deficiency, such as bone fragility or swayback are observed, and that treatment is rarely justified on the assumption that growth alone may be improved.

3.2   Signs of copper deficiency in livestock and pastures

3.2.1   Cattle

Hair coat abnormalities/rough coats, fading of coat color and the development of thin, sparse dry hair are some of the earliest signs associated with cop-per deficiency in young cattle, for example, sandy-colored Herefords and bronze-tinged black cattle. These signs are usually seen with copper deficiency before growth is affected. Facial hair, particularly around the ear margins and eyes, usually shows the first changes.

Treatment of affected calves with copper is considered important by farmers who sell their stock by auction through saleyards and so rely heavily upon animal appearance. It should be noted that changes in hair coat color and texture are not sufficiently specific for diagnosis of copper deficiency, since similar changes also occur with cobalt deficiency, and retention of the winter coat due to debilitation from underfeeding and intestinal parasitism. Hair coat changes are also seen in lactating cows with sodium deficiency.

Retarded growth/illthrift, plasma copper levels usually have to be in the deficiency range for at least a month before weight gains to copper supplementation occur. Responses to supplementation are generally small (10 to 15%), and are most likely to occur in young rapidly growing cattle (3 to 12 months of age) during the spring (Paynter and Allen 1981). Growth responses to copper supplementation are more common when pastures contain more than 3 mg Mo/kg DM or Cu: Mo ratios less than 2 (Suttle 1983).

Diarrhoea is a variable clinical sign of copper deficiency in cattle. It is non-specific, and is usually suspected of being associated with copper deficiency when cattle with diarrhoea have not responded to

anthelmintic treatments. High molybdenum concentrations (above 5 mg Mo/kg

DM) in lush herbage are commonly associated with diarrhoea that responds rapidly to injectible copper. On pastures with high molybdenum content, cattle may develop diarrhoea before liver reserves are depleted and blood copper fractions decrease (Anon. 1982).

Skeletal defects in calves with copper deficiency may be seen as swelling and stiffness of the fetlock joints.

Infertility has been associated with severe cop-per deficiency, but the evidence is rarely conclusive. Few studies have definitely related copper deficiency to infertility and it is considered that copper deficiency alone would have to be prolonged before an effect on fertility would be observed. However, copper treatment of beef heifers at the start of mating in Victoria has been shown to suppress fertility in the short term (Cummins 1977).

Anaemia is a relatively rare sign of copper deficiency, and develops late in the disorder (Anon. 1982).

Cardiovascular disorders/"falling disease", first noted in cattle in Western Australia, is due to degeneration of the myocardium with replacement fibrosis. Elsewhere, this disorder is rare in cattle with copper deficiency.

3.2.2   Sheep
Wool abnormalities, loss of wool crimp is one of the first clinical signs of copper deficiency in sheep. The wool lacks character and develops a sheen or lustre ("steely wool") with crimps 3-4 times normal width, and it has greatly reduced tensile strength and elasticity.

Greying of black-woolled sheep is a sensitive sign of copper deficiency, and is one of the first signs observed with excessive molybdenum and sulphate intake.

Enzootic ataxia (swayback), occurs in spring-born lambs in southern Australia in areas low in copper, and where ewes have a high intake of molybdenum, or other factors which reduce the absorption of copper. The nervous tissue of the lamb has a special requirement for copper in the last two months of gestation, when it is rapidly developing, and in the immediate post-natal period. The ataxia is seen as paralysis or a staggering gait in newborn lambs, or develops up to six weeks after birth. It has been observed principally in the Western District and in South Gippsland (Savage 1974).

The greatest demand for copper by grazing sheep is by the ewe in late pregnancy and early lactation. An adult ewe needs about 3.7 mg/day, but in late pregnancy with twins the requirement almost trebles to 10.5 mg and almost doubles again in lactation (to 20.7 mg/day) for a ewe giving 3 kg milk (Underwood 1977). All of these requirements can be met on a diet providing 4.5-8.0 mg Cu/kg dry matter providing the ewe gets enough to eat and utilizes 6% of the dietary copper. On a pasture containing high molybdenum and sulphur levels, only 2% or less of the dietary copper may be utilised (Lewis 1982). On such pastures the ewe in late pregnancy, during the winter months of June, July and August in southern Australia, may have great difficulty in meeting its requirement for copper, with resulting detriment to the lamb.

Table 3.1: Biochemical values used to assess copper nutrition

	Sheep		Cattle	
	Deficient	Adequate[1]	Deficient	Adequate[1]
Liver				
Copper				
(mmol/kg DM)	< 0.2	0.6-10.0	< 0.1	0.3-6.3
(mmol/kg wet wt)	—	0.2-3.7	—	0.1-2.1
Plasma				
Copper (umol/l)	< 5	8-20	< 5	8-20
Ceruloplasmin (U/1)	< 5	40-90	< 5	50-90
Erythrocyte				
Copper (umol/l)	—	—	< 8	8-14
CUSOD (U/g Hb)	< 200	450	< 300	500
Pasture				
Copper[2] (mg/kg DM)	—	6	—	7

1.   Marginal nutritional status defined as the range between deficient and adequate levels (see section 1.5).
2.   Approximate only because of the importance of sulphur and molybdenum concentrations.
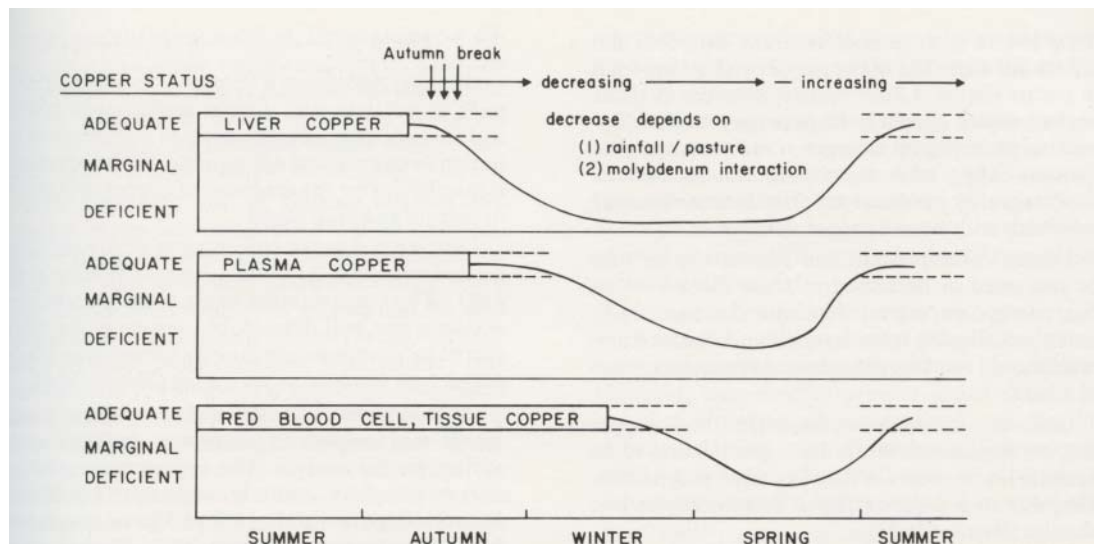
Figure 3.2: Schematic diagram of the seasonal changes in copper in liver, plasma and red blood cells expected in live stock in Victoria in a marginally deficient area.

Anaemia in sheep can occur with extreme copper deficiency, but can be induced more readily by adding molybdenum and sulphate to a low-copper diet (Lewis 1982). In grazing sheep, parasitism, cobalt deficiency or malnutrition are more likely to be causes of anaemia than copper deficiency alone.

Reduced growth is not a feature of copper deficiency in grazing sheep, but occurs when dietary molybdenum and sulphate are greatly increased.

Bone fragility in lambs is often associated with copper deficiency, with increased incidence of fractures in long bones and rib bones. In Victoria, bone fragility is more characteristic of a molybdenum/sulphate induced deficiency than of a copper deficiency alone.

Infertility)s not a feature of copper deficiency in grazing sheep.

3.2.3 Pastures

Chlorosis, necrosis, leaf distortion, that is, cup-ping in clover, terminal die-back in young leaf tissues and white, empty seed heads in grasses are features of severe copper deficiency. Less severe deficiency signs observed are similar to those of other marginal plant deficiencies, for example, relative stunting with delayed leaf senescence and reduced seed production (Loneragan *et al.* 1981).

3.3 Diagnosis of copper deficiency

3.3.1 Livestock

For the diagnosis of copper deficiency the first aim is to determine the copper status of the animals, then determine if the deficiency is due to a copper deficiency alone, or to the presence of interacting factors such as molybdenum and sulphate. In most field investigations, the only practical laboratory tests available are analyses of liver and blood to assess the copper status of the animals (table 3.1 and figure 3.2). Pasture samples provide useful supplementary information if they can be assayed for copper, molybdenum and sulphate.

A clinical response, seen as an improvement in health and production after copper supplementation, is the most definitive way of confirming copper deficiency in animals.

Copper deficiency in cattle in southern Australia has been most prevalent in favorable seasons with lush grass pasture growth in spring (Underwood 1981). In Victoria, a marginal copper deficiency is often observed in which plasma copper concentrations are decreased for a short period without erythrocyte copper being affected (Paynter and Allen 1981). This period may occur between May and November (winter and spring) depending on the year. The extent and duration of the depletion of copper in liver, plasma, erythrocytes and other tissues in this period determines the severity of deficiency (figure 3.2), and whether production and health problems

due to copper deficiency will occur. The copper nutrition of grazing cattle increases as pastures dry off over the summer period, and a marginal deficiency detected in mid-spring is usually resolved in mid-summer.

Liver copper

Copper that is absorbed in excess of tissue requirements accumulates in the liver. When intake is below this requirement the first change observed in the copper status is a decrease in liver copper concentration (figure 3.2). When liver copper has decreased to the extent that copper required for ceruloplasmin synthesis by the liver becomes limiting, plasma ceruloplasmin and copper concentration decrease (Suttle 1983).

Plasma copper

Ceruloplasmin is the major copper-containing enzyme and contains 70 to 90% of the plasma copper. Low plasma copper or ceruloplasmin reflects depletion of liver copper reserves, but does not reflect the depletion of copper-dependent enzymes in tissues (figure 3.2). It is these changes in tissue enzyme which appear to be necessary for development of pathological changes or reductions in production. They take about a month to become important after plasma copper has decreased to very low levels with simple copper deficiency.

Plasma ceruloplasmin and plasma copper may be increased or decreased in some disease states, including ostertagia infections (Savage 1974). Samples collected from feverish and diseased animals should not be used to assess the copper status of a herd.

Collection methods are important; both copper and ceruloplasmin values have been found to be variably lower in serum than in paired plasma samples, due to a sequestering of ceruloplasmin into the clot (Paynter 1981).

Erythrocyte copper

The copper concentration in erythrocytes may better reflect the tissue activities of copper enzymes (Andrewartha and Caple 1980). In erythrocytes, 75% of the copper is in the copper-superoxide dismutase enzyme (CUSOD). The lifespan of erythrocytes is about 120 days and since CUSOD appears to be synthesized at erythrocyte initiation, erythrocyte copper concentration appears to be dependent on the copper intake of the animal over the preceding 2–3 months (Paynter and Allen 1981, Paynter et al. 1982).

Pasture copper

An **available** copper concentration of 5 mg Cu/ kg DM diet appears to be required for adequate copper nutrition of animals (Underwood 1977). In the Victorian situation, a total herbage copper concentration of 7 mg Cu/kg DM has been suggested to satisfy this requirement (Skene 1964), but the required level is not well defined (Brown 1982a). Herbage copper levels in Victoria commonly range between 3 and 20 mg/kg DM, with about 50%

of all samples tested being 7 mg/kg or less (Brown 1982b).

Where a high concentration of molybdenum relative to that of copper occurs in herbage, an application of molybdenum may induce copper deficiency in animals. As an approximation, the copper availability is reduced by 50% for every increase of 4 mg Mo/kg DM in the diet (Loneragan *et al*. 1981). In Victoria, 7% of 260 mixed herbage samples analysed between 1970 and 1982 had molybdenum concentrations between 2 and 4 mg Mo/kg DM and 3% had greater than 4 mg Mo/kg DM (Brown 1982c).

Soil copper

Soil copper concentration is poorly correlated with animal copper nutrition (McDonald and Mahoney 1982).

3.3.2   Plants

If copper deficiency is suspected in plants on the basis of soil type and district responses in plants and animals then measurements of copper concentration in pastures are appropriate. Visual appraisal is insufficient for the diagnosis of copper deficiency in pasture and crop plants.

Deficiency signs in clover occur where the copper concentration is less than 3 mg/kg (Reuter *et al*. 1981). The concentration where a pasture response occurs is not well defined, but responses have occurred in Victoria where the concentration in whole clover tops was less than 6 mg/kg (Skene 1964).

At present, the State Chemistry Laboratory uses whole top samples of sub-clover, collected mid-spring, for Cu analysis. The copper concentration in these samples commonly ranges from 4 to 20 mg/kg, with approximately 18% of Victorian samples being 6 mg/kg or less (Brown 1982). Work in other States indicates that a concentration of less than 3 mg/kg in youngest-fully-open subterranean clover leaves more accurately indicates deficiencies in plants because this measure, unlike whole tops analysis, takes account of changes in copper concentration which occur with stage of growth or maturation (Reuter *et al*. 1981).

Where whole tops of sub clover have a copper concentration less than 6 mg/kg, it is recommended that strip trials be conducted to ascertain if pasture production responses occur.

Soil copper concentration is poorly correlated with plant copper concentrations (Conley 1983).

3.3.3   Analyses available

Ceruloplasmin assays and copper analyses of plasma and liver are conducted at all veterinary laboratories. The copper-superoxide dismutase assays are conducted, for special investigations, at Benalla Regional Veterinary Laboratory. Pasture analysis is available through the State Chemistry Laboratory. Soil analysis is not recommended for diagnostic purposes.

3.4   Treatment for copper deficiency

3.4.1   Immediate treatment of animals

Immediate treatment of copper deficiency in sheep or cattle may be given by injections with commercially available organic copper compounds, or by oral drenches (table 3.2). Problems associated with injectible copper compounds in Victoria have included: abscesses at the site of injection due to poor technique (see the agnote "Copper for pasture and grazing animals"); anaphylactic reactions, including respiratory distress and sudden death occurring within an hour of injection; death due to liver damage; decreased milk production; and suppression of fertility when cows have been treated at mating (Cummins 1977). With these reservations, injectible copper therapy is suitable for both primary and conditioned, for example, molybdenum induced, forms of copper deficiency, and a single treatment provides adequate copper for 2-3 months. Animals should not be treated with copper unless copper deficiency has been diagnosed, for example, plasma copper concentration less than 5 umol/l (table 3.1.)

Oral drenching with an aqueous solution containing copper sulphate (table 3.2) is useful in primary copper deficiency only. Copper sulphate may be mixed with levamisole-based and oxfendbendazole anthelmintic drenches, and should be used within 24 hours of mixing. Copper is not compatible with many other drenches. With primary copper deficiency, a single oral treatment is adequate for 1-2 months. However, for molybdenum-induced deficiency weekly treatments may be required, making this form of treatment generally impractical (Underwood 1981). Metering devices developed for addition of minerals to drinking water have been successfully used to supplement cattle with copper (Macpherson 1981).

Copper oxide needles and glass bullets containing copper have been developed for introduction to the rumen and are being tested before commercial release. Work conducted in South Australia has indicated that calves given 50 g copper oxide needles had adequate copper reserves over 184 days, whereas a group of calves given a standard copper glycinate injection lost weight towards the end of the trial (Deland *et al*. 1979).

Copper-containing salt licks may be used where individual animal treatment is impractical. Recommended concentrations are 2% copper sulphate for cattle and 0.25-0.5% for sheep (Savage 1974). Some or all animals may fail to lick the salt block or mineral mix and this reduces the efficacy of this method of treatment.

Strategic treatment to cover the period from mid-winter to mid-spring is usually sufficient for all but the most severely deficient animals in Victoria. However,

treatment to prevent ataxia should be aimed at providing adequate copper nutrition for at least six weeks prior to lambing. This ensures adequate copper for the high amount of myelination occurring in the foetus during this period and post-natally.

3.4.2 Long-term treatment and prevention of copper deficiency in animals and pastures

Where herbage copper is limiting for animals, and the stocking rate warrants it, pasture topdressing with copper is the preferred long-term treatment. In the southern high-rainfall areas of Victoria, on newly cleared copper-deficient country, copper fertiliser is applied immediately before or after sowing pasture. It can also be applied successfully to established pasture. The standard recommendation has been to apply 2 kg of copper per ha every five to seven years. However, there is little Victorian data available to determine how often copper should be reapplied. Work in other States indicates that at this rate of application, reapplication may not be necessary, at least in terms of copper status of pastures and sheep, for at least 13 years (Loneragan et al. 1981). Victorian experience suggests that the more frequent application is necessary to prevent the appearance of copper deficiency in stock.

Fertilisers mixed with copper oxide are available commercially and offer a range of concentrations of copper. The concentration used will depend on the rate of copper needed and on the rate of the other fertilisers to be applied in the mixtures. Copper is commonly applied at 0.5, 1 or 2 kg/ha, at intervals of one to several years. (For details see the agnote "Copper for pastures and grazing animals").

Table 3.2: Copper treatments for animals

Injectible remedies

1 Copper glycinate
   Cuprate   Heriot Agencies Pty Ltd,
                                       16 Macquarie Place, Boronia
                                       Bottles containing 100 ml copper glycinate
2 Organic copper complexes
   Cujec   ICI Australia Ltd,
                                       1 Nicholson Street, Melbourne
                                       Diethylamine cupro-ocyquinolone sulphonate available copper 6 mg/ml
                                       Cujec is not registered for administration to cattle.

Oral remedies

1 Copper sulphate
   Dose – cattle 4g per head
        - sheep 1.5 g given at weekly intervals in deficient period
   Products available:
    Admin mineral supplement – for addition to *Systamex* for sheep
    Wellcome Australia Ltd, 145 Heidelberg Road, Northcote
2 Mineral salt blocks, licks
   Etc
   Many suppliers

Trade names are shown in italics.

Cautionary note
1.   The manufacturers' "Directions for Use" of remedies should be read and followed.
2.   Intramuscular injections should not be given to animals intended for slaughter as sterile nodules may be caused and the meat condemned. Subcutaneous injections are preferable. A large swelling often develops at injection sites. This swelling usually disappears after 2 to 3 weeks. On properties where blackleg occurs, susceptible animals should be vaccinated at least 2 weeks before injections of copper are given.

When topdressing molybdenum-deficient pasture with molybdenum, it is important to recognise that overall production gains through increased pasture production will be far greater than any production losses incurred through a molybdenum toxicity. Copper should be applied with the molybdenum if the concentration of copper in the clover is marginal.

3.5   *Copper toxicity*

Both chronic and acute forms of copper toxicity have been observed in sheep in Victoria. The main form of chronic copper poisoning in sheep is associated with long-term ingestion of heliotrope and occurs principally in northern Victoria. Pyrrolizidine alkaloid damage to the liver following ingestion of heliotrope predisposes to greatly increased liver copper concentrations even at normal dietary copper concentrations (Savage 1974). The excessive accumulation of copper, usually over several months, results in a sudden release of copper into the blood. The form of copper released is highly toxic to erythrocytes and other tissues, and haemolysis, haemoglobinuria, jaundice and muscle damage result.

Chronic copper toxicity without predisposing liver damage has also been observed in grazing sheep in Victoria. Known as phytogenous copper toxicity, the disease is prevalent in seasons which favor the early germination and continued dominance of

subterraneum clover within the pasture sward (Albiston 1975). Outbreaks have been re-corded in 1939, 1946, 1947, 1973 and most recently in 1983 (in North East Victoria and the Western District). Toxic pastures generally contain a normal or marginally elevated copper concentration (10-20 mg/kg DM) and a very low molybdenum concentration (less than 0.2 mg/kg DM) for extended periods. This favors the accumulation of copper in the liver.

Chronic copper toxicity can also be a problem in housed sheep. Dry feeds having a high concentration of available copper and a low concentration of molybdenum, or mineral premixes containing high copper levels, are usually the cause.

Acute copper toxicity usually follows within a few days of dosing with copper, and is evident as gastroenteritis with associated abdominal pain, severe diarrhoea and signs of shock (Savage 1974). Faeces are generally bluish-green and contain mucus. Affected sheep pass red-colored urine. Dead animals should be submitted for examination by a pathologist. This form of copper toxicity has been seen in sheep given therapeutic doses of copper in the summer and autumn. It is re-emphasised that sheep should not be treated with copper in this period unless copper deficiency has been diagnosed.

Indications of copper toxicity are high liver and

kidney copper concentrations, with elevated plasma copper concentrations and high liver enzyme activities in plasma during the acute phase of toxicity. Histological examination of liver can also be used to confirm the toxicity and may be useful in differentiating the involvement of hepatotoxins in causing the liver copper accumulation (Savage 1974).

Cattle are not as susceptible to copper toxicity as sheep, but deaths in calves one to two months old occurred after they were given 12 to 24 ml injections of *Cujec* containing 6 mg Cu/ml (Mylrea and Byrne 1974). All 44 calves died from a combination of hepatotoxicity and toxic renal tubular nephrosis over a period of 12 days after the injection.

Chronic copper poisoning has occurred in dairy cows by over-supplementing prepared feed with up to 22 g copper sulphate/day (Stogdale 1978).

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