RECENT FINDINGS IN TRACE ELEMENT NUTRITION

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I. INTRODUCTION

Studies in South Australia in the 1930-40's showed that the coastal areas and, to a lesser extent, the areas inland had inadequate levels of cobalt and copper for livestock health and production. Since these studies, selenium responsive conditions have been recognised in livestock depastured on the ironstone soils in the higher rainfall areas and also in isolated, but at present ill-defined, areas in the south-east of the State. The extent to which other trace element inadequacies are limiting livestock production in this State is not known but productivity responses to manganese and zinc supplements have been obtained in sheep at pasture.

The occurrence of severe copper, cobalt or selenium deficiency in livestock in South Australia has been reduced or prevented by applications of trace elements to soil or their administration to the animal. However, sub-clinical conditions resulting from marginal trace element inadequacies may occur over large areas of the State. Sub-clinical deficiencies often result in a greater economic loss to the producer than a frank deficiency of the element since the latter can be recognised and the disorder corrected. Laboratory findings are increasingly being relied upon to indicate marginal trace element inadequacies in livestock. The purpose of this paper is to review some of the laboratory methods currently used to assess the copper, cobalt and selenium status of animals and to comment on methods for correcting inadequacies of these trace elements in the grazing animal.

II. COPPER

(a) Assessment of Status

The copper status of ruminants is influenced not only by the amount of copper in the diet, but also by other factors notably dietary concentrations of molybdenum and sulphur.

Early indications of hypocuprosis in sheep are partial loss of the distinctive crimp in wool resulting in 'steely wool' and depigmentation in coloured fleece. A 10% loss in wool production can occur

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before steeliness in wool is apparent. Furthermore this defect may not be recognised by producers. The prevalence of steely wool in wool clips sold from South Australia was used by H.J. Lee between 1939 and 1946 and by Hannam and Reuter between 1972 and 1975 (Hannam and Reuter 1977) to indicate areas in this State where copper is likely to be inadequate for normal sheep production.

Achromotrichia is regarded as an early sign of hypocuprosis in cattle although it may be difficult to recognise in crossbred cattle and may be confused with the hair bleached by weathering and about to be shed. Recent work with calves also shows that hypocuprosis may affect productivity before changes in coat colour become apparent (Bingley and Anderson 1972; Suttle and Angus 1976).

The laboratory examination of blood or other tissues can provide useful information in the diagnosis of copper responsive conditions. The liver is the major reserve of copper and, in cases of dietary inadequacy, it will become depleted to maintain copper supplies to other tissues. Despite the development of simple biopsy techniques for the removal of liver for copper assay, liver biopsy is rarely used to assess copper status of live animals. Instead blood or occasionally hair or wool samples are employed. Plasma (or serum) copper estimations are preferred to whole blood copper estimations because the plasma copper is more labile than blood cell copper and hence more likely to change when the copper status of the animal is low. In normal animals, copper bound to caeruloplasmin with lesser amounts in loose combination with albumin accounts for the bulk of the plasma copper. However, in some instances of copper depletion in ruminants, induced by molybdenum in the presence of adequate dietary sulphur, the plasma copper concentrations may not fall and indeed may rise. This anomaly is probably due to the presence of copper-thiomolybdate complexes in plasma. These complexes are very stable and render the copper unavailable to the animal (Dick, Dewey and Gawthorne 1975; Bingley and Ruksan 1977; Bremner and Young 1978). Fortunately, the thiomolybdate-copper complexes appear to be insoluble in dilute trichloroacetic acid so that the assay of trichloroacetic acid soluble copper in plasma or the assay of plasma caeruloplasmin activity can be used to determine the copper available to tissues.

The assay of the copper-dependent enzymes, caeruloplasmin in plasma or cytochrome oxidase in liver, instead of copper do not appear to confer any advantage in the diagnosis of a copper-responsive condition since enzyme and element are often closely associated (Mills and Dalgarno 1970; Poole 1970). Plasma copper concentrations of less than about 8 \( \mu \text{mol/l} \) are usually considered indicative of hypocuprosis in ruminants. However, in recent field trials we have recorded plasma copper concentrations of 3-5 \( \mu \text{mol/l} \) in apparently healthy calves, assessed by measuring growth rate responses to copper supplementation. A minimum concentration of copper in tissues consistent with optimum production may vary depending on factors such as breed, age, physiological condition and health of the animal. Appreciation of some of these problems has resulted in Dutch advisory workers assigning minimal values for liver copper and for blood copper in cattle according to the stage of development of the animal and to environmental conditions (Committee on Mineral Nutrition, T.N.O. 1973).
(b) **Treatment and Prevention of Hypocuprosis**

Early studies in South Australia established that soils over wide areas, when newly developed, required copper fertilizer for optimum plant and livestock production. A common recommendation was to apply 4 to 8 kg/ha of copper sulphate to pasture every three to four years. The persistence of available copper in some South Australian soils following application has been underestimated. Recent work has shown that a total application of 4 kg/ha of copper sulphate to the ironstone soils of Kangaroo Island and the southern Adelaide Hills was effective in preventing copper-responsive conditions in pasture and sheep for at least 15 years (Reuter et al. 1977). Similar findings on the persistence of applied copper were also indicated from field trials conducted over the 1974-1977 period with weaner sheep depastured on a number of soil types in the south-east of the State. On some other soil types, however, the persistence of applied copper, is short-lived. Studies just completed indicate that the peat soils in the south-east of South Australia require 4 kg/ha of copper sulphate every three years to maintain pasture production (R.J. Hannam personal communication). Even this rate of copper application is insufficient to meet the requirements of animals grazing that pasture and direct administration of copper to dairy cattle has to be undertaken to prevent hypocuprosis (P.R. Cunningham personal communications).

A significant development in the provision of copper to livestock was the work of Dewey (1977) who showed that oral dosing of 10 g copper oxide particles to sheep resulted in a marked enhancement in liver copper stores. He showed that a large proportion of the dose quickly migrated to the abomasum and the residence time in that organ was sufficient to permit dissolution of the copper oxide with the absorption of copper.

Field experience with Dewey's copper oxide is encouraging. The plasma copper concentrations (trichloroacetic acid - soluble copper) of calves depastured on peat soils in the south-east of South Australia were maintained over a longer period in the calves treated with copper oxide (Table 1). This was associated with an apparent increase in haemoglobin concentrations ($P<0.05$, 't' test) 21 weeks after treatment. The mean live weight gains were consistently greater in the copper oxide group (Table 1), but this greater response was not significant ($0.10>P>0.05$, paired 't' test) at the twenty-first week of the trial.

This method of copper supplementation appears to be safe, as attempts to induce copper toxicity in sheep by frequent dosing with copper oxide have failed (D.W. Dewey personal communication). Commercial production of copper oxide particles is anticipated for the near future and should prove an effective and economical means of providing copper to livestock in the field.
TABLE 1: Live weight, plasma copper and blood haemoglobin values in calves given 120 mg copper (as copper glycinate) parenterally or 50 g copper oxide orally*

(Mean values with their standard deviation for 7 calves).

<table>
<thead>
<tr>
<th>Weeks after treatment</th>
<th>Copper Glycinate</th>
<th></th>
<th></th>
<th></th>
<th>Copper Oxide</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Live Weight kg</td>
<td>Plasma Cu μmol/l</td>
<td>Blood Hb g/100ml</td>
<td></td>
<td>Live Weight kg</td>
<td>Plasma Cu μmol/l</td>
<td>Blood Hb g/100ml</td>
</tr>
<tr>
<td>Pre-Treatment</td>
<td>118±33</td>
<td>2±0.4</td>
<td>122±33</td>
<td>2±0.8</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5</td>
<td>11±2.0</td>
<td>10±1.5</td>
<td>12±1.3</td>
<td>12±2.5</td>
<td></td>
<td></td>
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<tr>
<td>11</td>
<td>6±1.9</td>
<td>12±1.2</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>206±19</td>
<td>3±0.8</td>
<td>215±22</td>
<td>12±1.2</td>
<td>12±0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>221±22</td>
<td>2±0.8</td>
<td>235±19</td>
<td>8±1.3</td>
<td>14±0.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Calves were matched according to live weight within sex and allocated at random to treatment groups (data supplied by P.R. Cunningham).

+ Mean values for 3 calves.

III. COBALT

(a) Assessment of Status

Cobalt or vitamin B12 responsive conditions in sheep have received considerable attention but less is known about the disorder in cattle (see review by Gardiner 1977). Although cattle are regarded as being less susceptible to cobalt deficiency than sheep, in South Australia, growth rate responses to cobalt supplementation have been observed in apparently healthy beef cattle in the south-east (Skerman et al 1959) and more recently on Yorke Peninsula (M.E. Dodson personal communication).

Currently we are evaluating diagnostic aids for the detection of cobalt responsive conditions in cattle. Table 2 summarises some preliminary findings with B12 deficient calves depastured on coastal calcareous sands. The response in live weight to B12 supplementation suggests that the calves were probably born with sufficient B12 reserves for the first few months of life. After this period the B12 in the dam's milk, which was usually less than 1 μg/l, together with
<table>
<thead>
<tr>
<th>Weeks</th>
<th>Nil Treatment+</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>B12 Treatment+</th>
<th></th>
<th></th>
<th></th>
<th></th>
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<tr>
<td></td>
<td>Live Weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Live Weight</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>kg</td>
<td>63±27</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>66±29</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WBC 10³/mm³</td>
<td>82±26</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>88±30</td>
<td></td>
<td></td>
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<tr>
<td>12</td>
<td>91±24</td>
<td>10±4.3</td>
<td>12±2.2</td>
<td>41±3</td>
<td></td>
<td>102±30</td>
<td>12±0.6</td>
<td>12±2.7</td>
<td>42±4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>106±23</td>
<td>8±1.5</td>
<td>12±1.7</td>
<td>43±4</td>
<td></td>
<td>125±29</td>
<td>9±0.7</td>
<td>12±1.1</td>
<td>42±2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>117±26</td>
<td>9±0.7</td>
<td>13±1.1</td>
<td>41±4</td>
<td></td>
<td>158±31</td>
<td>10±1.6</td>
<td>11±0.4</td>
<td>53±5</td>
<td></td>
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</tr>
<tr>
<td>29</td>
<td>131±31</td>
<td>8±1.8</td>
<td>11±0.9</td>
<td>41±1</td>
<td></td>
<td>202±37</td>
<td>13±2.9</td>
<td>11±1.5</td>
<td>55±6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

+ WBC = White blood cells; Hb = haemoglobin; MCV = mean corpuscular volume. Mean values for 3 calves except at 29 weeks when mean values for 6 calves.

+ For each row and for each constituent, mean values with the same superscript 'a', 'b' or 'c' were significantly different at P<0.05, P<0.01, P<0.001 respectively.

* Hereford calves aged between 1 and 12 weeks were matched for live weight within sex and allocated at random to treatment - B12 treated calves received 6-weekly injections of cyanocobalamin (6 mg/50 kg live weight).
the little cobalt from ingested pasture was insufficient to meet their 
B12 requirements. One feature of cobalt deficiency is anaemia. In 
contrast to the normocytic, normochromic anaemia in B12 deficient sheep, 
the anaemia in the B12 deficient calves was of compensated microcytic 
normochromic type. It was associated with a depressed white blood cell 
count (see Table 2).

The onset of anaemia in sheep and cattle develops only in 
relatively severe deficiency and therefore evaluation of haematological 
parameters is of no value in assessing sub-clinical B12 inadequacy.

The B12 concentrations of liver and serum are responsive to 
cobalt supplementation and hence are of diagnostic value in advanced 
B12 deficiency. In general, serum B12 concentrations of less than 0.5 
μg/l in sheep and probably less than 0.2 μg/l in cattle are suggestive 
of cobalt inadequacy (see Gardiner 1977). In the field trial described 
above, preliminary assays indicate that the serum B12 values in the 
deficient calves were less than 0.1 μg/l.

Serum B12 concentrations are of less value for confirming 
marginal cases of cobalt deficiency. Serum B12 concentrations vary 
widely between sheep within groups and also within the same sheep on 
different days (Somers and Gawthorne 1969; Findlay 1972). There are 
also diurnal fluctuations in serum B12 concentrations.

Laboratory procedures which may prove superior to the assay of 
serum B12 are those which measure metabolic disturbances resulting 
from the reduced activities of B12-dependent pathways. Methylmalonate 
excretion has been proposed as a specific indicator of vitamin B12 
deficiency since its conversion to succinate requires a B12 dependent 
enzyme. Large amounts of propionate, a precursor of methylmalonate, 
are produced in the intestinal tract of ruminants and abnormal amounts 
of methylmalonate in urine or blood indicate a metabolic disturbance 
consistent with B12 insufficiency. For example in grossly cobalt 
deficient sheep at this laboratory serum methylmalonate concentrations 
were as high as 200 μmol/l whereas in cobalt sufficient sheep the 
concentration of methylmalonate in serum was in the order of 20 μmol/l 
(T.H. Benson personal communication). Gawthorne (1968) has shown that 
urinary excretion of formiminoglutamate, an intermediate in the meta-
bolism of histidine, was also a sensitive indicator of the disorder. 
Increased urinary excretions of methylmalonate and formiminoglutamate 
have been recorded in cobalt-deficient sheep at pasture (see Gardiner 
1977): Further studies are required to assess the usefulness of 
assaying these metabolites as indicators of marginal B12 inadequacy 
in ruminants under field conditions.

(b) Prevention and Treatment of Cobalt Deficiency

A common practice in South Australia is to administer cobalt 
pellets to livestock at weaning. The pellets introduced into the rumen, 
either singly or with an abrasive steel grinder, have proved an 
effective means of preventing B12 deficiency in sheep or cattle grazing 
cobalt-deficient pastures. The commercial pellet contains about 60% 
by weight of cobalt oxide, the active ingredient, and 40% iron powder. 
Recently attention has been directed towards reducing the cobalt oxide 
content of the pellet because of its high cost and short supply. The
release of cobalt from the pellet appears to be more than adequate. Serum B12 concentrations of 2 - 4 µg/l are frequently observed in sheep with pellets on cobalt-deficient pasture. Lee and Kuchel (1977) reported that a reduction of cobalt oxide from 60 to 30% resulted in an effective pellet for sheep. The concurrent increase in the proportion of iron powder was shown to raise the specific gravity of the pellet. This change should improve the retention of the pellet in the reticulo-rumen, since rejection of commercial pellets by regurgitation has been observed in cross-bred sheep.

The B12 reserves of lambs from B12-sufficient ewes are usually sufficient to meet the lambs, requirements for the first 2 - 3 months of life. Methods used to provide cobalt to the unweaned animal after this period include oral dosing of cobalt, provision of salt licks containing cobalt, the misting of a cobalt solution over the pasture grazed by livestock and soil applications of cobalt. These methods of cobalt supplementation will not be effective before the young animal has an established microflora in the alimentary tract. These methods will increase the B12 status of the dam but B12 in the dam's milk contributes only marginally to the B12 requirements of the lamb (Andrews and Stephenson 1966). One method of supplementation which has received some attention in New Zealand is the parenteral provision of B12 to the animal.

Parenteral injections of B12 as a means of preventing B12 deficiency in unweaned livestock at pasture are being investigated in South Australia. Table 3 presents some recent findings on the live weight and fleece weight response to cobalt and B12 supplementation in young sheep depastured on a coastal terra rossa sand. In general it was found that a single injection of 1 or 2 mg hydroxocobalamin was effective for at least 14 weeks in maintaining growth rates of young sheep in a severely cobalt deficient area. The slightly inferior growth rates in unweaned sheep receiving oral doses of cobalt is consistent with earlier findings that sheep require at least weekly doses of cobalt to maintain optimum growth rates. These and other results indicate that B12 supplementation is a convenient and practical means of preventing B12 deficiency in unweaned sheep at pasture.

IV. SELENIUM

(a) Assessment of Status

The selenium content of most tissues of ruminants is responsive to changes in the dietary intake of selenium. Blood is usually taken for assessing the selenium status of the animal and in ruminants about 65 to 80% of whole blood selenium is in the blood cells. The validity of using plasma or wool selenium values as indicators of the change in selenium status of sheep, over short intervals of time was recently questioned by White and Somers (1977). They found that lowering the sulphur content of a ration offered to sheep resulted in an increase in the selenium concentration in plasma and wool but this increase was not associated with a change in the selenium status of the animal as assessed by the apparent retention of dietary selenium. In fact it
### TABLE 3: Response in live weight and greasy fleece weight of young sheep to oral doses of cobalt or subcutaneous injections of hydroxocobalamin*

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mean live weight (kg), weeks after treatment</th>
<th>Mean fleece weight at weaning kg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretreatment 6 9 14 18</td>
<td></td>
</tr>
<tr>
<td>Nil</td>
<td>11.6 18.2 18.9 19.9 **</td>
<td>1.28</td>
</tr>
<tr>
<td>Co drench</td>
<td>11.6 18.5 **</td>
<td>1.41</td>
</tr>
<tr>
<td>0.5 mg B12</td>
<td>11.6 18.7 20.2 25.4 27.7</td>
<td>1.37</td>
</tr>
<tr>
<td>1 mg B12</td>
<td>11.6 18.8 21.2 24.9 26.8</td>
<td>1.35</td>
</tr>
<tr>
<td>2 mg B12</td>
<td>11.6 19.6 21.3 25.4 26.9</td>
<td>1.42</td>
</tr>
<tr>
<td>2 mg B12</td>
<td>11.7 19.9 **</td>
<td>1.53</td>
</tr>
<tr>
<td>LGD (P=0.05)</td>
<td>n.s. 1.2 1.4 1.7 1.0</td>
<td>0.13</td>
</tr>
</tbody>
</table>

* Department of Agriculture and Fisheries - I.M.V.S. joint project supported by the Australian Wool Corporation.

+ Lambs matched for live weight and allocated at random to treatment, 24 lambs/treatment. When sheep died missing value estimates were included in the analysis. Lambs receiving Co drench each received 22 mg Co orally at 0 and at 6 weeks. All lambs were weaned at 9 weeks.

+ Lambs in these groups were each given a cobalt pellet with grinder at weaning.

** Data not included in the analysis since half the sheep in this group died.
appeared that the selenium status of the animal had probably declined since at low selenium and sulphur intakes the wool selenium represented a significant 'sink' for the retained selenium.

In South Australia blood selenium values greater than 0.5 μmol/l in sheep and 0.2 μmol/l in cattle are considered to be 'normal'. The results of several studies, however, have shown that there is a considerable overlap in the selenium values for blood and other tissues from 'normal' and 'selenium-responsive' animals. This overlap is to be expected since, apart from factors such as age and physiological condition of the animal, other dietary components notably tocopherol can markedly alter the requirements for selenium. In many instances the laboratory diagnosis of a selenium responsive condition may well be improved by assessing tocopherol status and assaying serum enzymes indicative of muscular damage.

An alternative to the assay of selenium is the assay of glutathione peroxidase activity. Glutathione peroxidase is a seleno-enzyme and a number of studies have shown that, in ruminants, blood cell or whole blood glutathione peroxidase activity is positively correlated with whole blood selenium concentration (see Anderson, Berrett and Patterson 1978). The response in blood cell glutathione peroxidase activity to selenium supplementation suggests that the element is incorporated into the enzyme only during erythropoiesis (Paynter and McDonald 1976). A non selenium-dependent glutathione peroxidase activity has been demonstrated in tissues from a number of species including sheep (Lawrence and Burk 1978) but this enzyme does not appear to be present in the blood cells of ruminants. The assay of glutathione peroxidase activity in blood has been simplified (Board and Peter 1976) and should prove useful for evaluating the selenium status of animals in the 'field. The usefulness of the procedure will have to be assessed for each species since cattle and sheep differ in the relationship between whole blood selenium concentration and glutathione peroxidase activity in blood cells. As with selenium assays glutathione peroxidase activities alone can not confirm sub-clinical selenium-responsive conditions.

(b) Prevention and Treatment of Selenium Inadequacy

There are several possible methods available for supplementing dietary selenium. These include soil treatment to raise the selenium content of the pasture and the administration of selenium to the animal.

Studies in New Zealand and U.S.A. have indicated that soil applications of selenium were effective in preventing selenium-responsive disorders in sheep depastured on these soils for a number of years following application. A disadvantage with this method is that it would require extensive analytical control to ensure that potentially toxic amounts of selenium are not applied. Rates of application of selenium need to vary, higher rate being necessary for acidic soils than for alkaline or very sandy soils. For the ironstone soils of South Australia it is doubtful whether applied selenium would be available to pasture because of the high iron oxide content and acidic nature of this soil (see Reuter 1975).
The intraruminal selenium pellet developed by CSIRO for sheep has proved an effective means of supplementing the dietary selenium of animals at pasture. The pellets, which are made by compressing 0.5 g of elemental selenium with 9.5 g iron powder provide adequate amounts of selenium without toxicity for several years and are effective in preventing and correcting nutritional muscular dystrophy in lambs on a low selenium diet (Kuchel and Godwin 1976). The New Zealand experience with these pellets has not been so successful. Andrews et al. (1974) have reported that in Romney wethers about 15% of the treated animals rejected the pellets within one year and the pellets acquired an accretion of calcium phosphate. Such a high incidence of rejection of pellets has not been observed with Merino sheep and placing a steel grinder in the reticulo-rumen apparently overcomes any problem with salt deposition on the pellet (Kuchel and Godwin 1976).

Heavy selenium pellets for cattle, prepared by CSIRO, were tested in an area south of Adelaide where blood selenium concentrations are consistently low. These studies (R.E. Kuchel, M.E. Dodson and G.J. Judson unpublished findings) showed that two 30 g pellets containing 10% by weight of elemental selenium were effective in raising the selenium concentration in blood and milk of cows and in raising the selenium status of their calves; the blood selenium concentrations in the treated cows were greater than the blood selenium concentration in the untreated cows for periods up to 18 months. The evaluation of similar selenium pellets, prepared by a commercial firm is now underway using beef cows depastured on the ironstone soils of Kangaroo Island. Table 4 presents the initial findings which show that after 1 year there still remains a significant increase (P<0.01, 't' test) in blood selenium concentrations in cows given the selenium pellets.

TABLE 4: Effect of selenium pellets on blood selenium concentrations in beef cows*
(Mean values with their standard deviations for 6 - 15 cows)

<table>
<thead>
<tr>
<th>Weeks after giving pellets</th>
<th>Whole blood selenium umol/l</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Nil</td>
</tr>
<tr>
<td>Pre-treatment</td>
<td>0.35±0.07(15)</td>
</tr>
<tr>
<td>7</td>
<td>0.23±0.03(15)</td>
</tr>
<tr>
<td>11</td>
<td>0.37±0.04(14)</td>
</tr>
<tr>
<td>16</td>
<td>0.31±0.02 (7)</td>
</tr>
<tr>
<td>28</td>
<td>0.33±0.02 (6)</td>
</tr>
<tr>
<td>38</td>
<td>0.39±0.05 (6)</td>
</tr>
<tr>
<td>52</td>
<td>0.32±0.05 (6)</td>
</tr>
</tbody>
</table>

* Treated cows were each given two 30 g pellets containing 10% by weight elemental selenium and 90% iron grit. Pellets prepared by ICI Australia Limited. (G.J. Judson, K.H. Mattschoss and R.J. Clare unpublished findings).

+ Number of animals.
v. CONCLUSIONS

Considerable progress has been made in the prevention or treatment of trace element inadequacies in livestock at pasture. The detection of marginal or transient trace element inadequacies by laboratory diagnostic tests requires further attention. It is doubtful whether precise minimal values will be established for trace elements in blood, liver or other samples taken for diagnostic purposes since these values only indirectly reflect the adequacy of the element at those sites most sensitive to its deprivation. Biochemical lesions usually precede a pathological change and hence investigations of such lesions may be more fruitful in developing tests for the diagnosis of marginal trace element inadequacies in livestock.

VI. ACKNOWLEDGEMENTS

The helpful advice and criticisms of my colleagues is gratefully acknowledged.

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