COBALT DEFICIENCY DISEASE IN YOUNG SHEEP

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Since Australian workers **(1, 2)** demonstrated the essentiality of dietary cobalt some 20 years ago great progress has been made in the diagnosis and, control of a. wasting disease of cattle and sheep now known to be due. to a deficiency of cobalt in feed. In New Zealand the routine examination of animal livers for cobalt content has, with reservations to be discussed, proved a useful diagnostic aid. It has been shown that cobalt deficiency disease is usually associated with specific soil types low in cobalt, and so it has been possible to define cobalt deficient areas in broad outline (Fig. 1). Areas are defined in terms of cobalt available from the soil through the plant to the animal. The following criteria are used. **Severe Deficiency:** Prior to the advent of cobalt supplementation, sheep of all ages, and in places cattle, sickened and died. **Mild to Moderate Deficiency:** Mainly young sheep affected, but at times and in places mature sheep possibly unthrifty to a mild degree. **Marginal or Suspected Deficiency:** At times and in places young sheep may develop cobalt deficiency disease. Areas distinguished on the map are necessarily arbitrarily defined. For example land classified as mildly to moderately deficient will include areas where cobalt deficiency is at best only suspected; at present much of the land tentatively classified as marginally or suspected deficient is probably healthy.

Control of the disease by regular cobalt topdressing of affected land has become a common practice.

On the theoretical side it is now known that the physiological function of cobalt is largely if not wholly explicable in terms of the red cobalt-containing substance vitamin B12. All higher animals require the vitamin. In the ruminant vitamin B12 needs are met by microbial synthesis in the rumen. For this a dietary source of cobalt is necessary so that if cobalt intake is suboptimal elaboration of vitamin B12 is restricted, and unthriftiness results. Ruminants apparently require much more vitamin B12 than do other animals, which explains their susceptibility to disease when

grazing low-cobalt pastures on which horses, for example, remain healthy.

Young growing sheep are the most 'susceptible of all animals to cobalt deficiency. These are followed by mature sheep, calves, and mature cattle in that order. Because severely deficient areas such as the pumice lands of the North Island and granite soils of Nelson are now well recognised and regularly supplied with cobalt the disease in cattle and mature sheep is now no longer a major problem. There are, however, areas where the cobalt status of soils has been provisionally

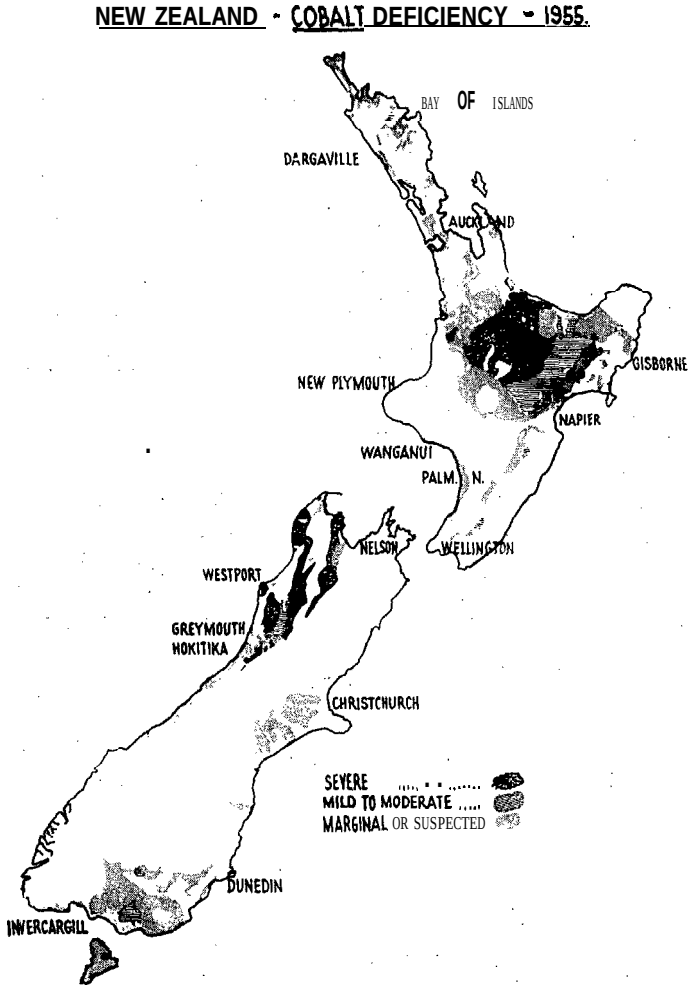

Figure 1.

classified as mildly to moderately deficient. Still other soils, amounting to several million acres, have been tentatively described as marginally or suspected cobalt-deficient. Areas represented by these two groups, (which for the purpose of the present paper may be considered together) include the gumlands and certain volcanic soils of North Auckland, soils formed in part from the margins of the great ash showers of the North Island, certain stony alluvial soils in Wairarapa, Westland and Canterbury, and leached soils in Otago and Southland. Within these areas the disease, which appears to be confined almost entirely to young sheep, often presents difficulties in diagnosis. Results from cobalt analyses are not always sufficiently conclusive, and at present the only sure means of diagnosing milder forms of cobalt deficiency disease is by measuring the weight-gain responses of animals to extra cobalt. For this and other reasons to be discussed the incidence of cobalt deficiency disease within the suspect areas is known with certainty only for isolated places and the economic loss from unrecognised or imperfectly controlled cobalt deficiency disease is probably still very great.

It is the purpose of this paper to describe cobalt deficiency disease as it occurs in young sheep on the less seriously deficient areas, to consider soil and pasture factors which may influence its course, and to discuss gaps in our knowledge which need to be filled to ensure an increased measure of control.

COBALT DEFICIENCY DISEASE IN YOUNG SHEEP

Signs of cobalt deficiency are essentially those of simple starvation. In lambs symptoms rarely appear during the first few months of life, possibly because of reserves of vitamin B12 existing at birth. Usually unthriftiness supervenes between the third and sixth months, depending upon the degree of deficiency. Not all lambs in a flock are, however, equally affected. A few might thrive; some fail only to gain as much weight as might be expected, and others waste away. Frequently the wool assumes a "washy" appearance and sometimes there is a discharge from the eyes. In mild cases mortalities are negligible, but if the disease is more severe the death rate can be quite high during the autumn months. During late winter and early spring surviving animals tend to recover, but as two-tooths they may again enter a second phase of unthriftiness in late spring and summer. Ultimately sheep tend to outgrow their susceptibility to mild cobalt deficiency. (Fig. 2.)

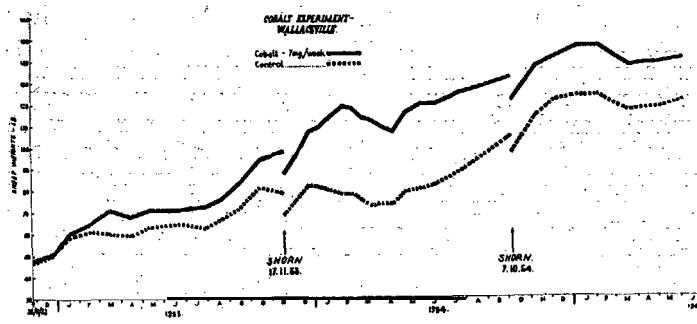

Figure 2.
Response of young growing sheep to cobalt on marginally cobalt-deficient land at Wallaceville.

The ailment can be variable in place and time. Some paddocks on the same farm or even the same soil type may be obviously worse than others and the degree of unthriftiness can vary from year to year.

THE EFFECT OF ENVIRONMENT ON COBALT DEFICIENCY DISEASE

From the considerable body of evidence available it is certain that the severity of the disease shown by grazing animals is closely paralleled by their cobalt intake. so far as this can be judged from chemical analyses of pasture samples. From this it follows that factors which govern the availability of soil cobalt to pasture plants or otherwise determine the amount of cobalt ingested via the pasture will influence the degree of unthriftiness.

SEASONAL EFFECTS'

Early investigations by McNaught and Paul (3) and subsequent work at Wallaceville has shown that in general the cobalt content of pastures tends to increase in late autumn and winter when plant growth is retarded and to decrease in spring and summer. These findings, together with the likelihood that the vitamin B_{12} requirement of young sheep is probably greatest during periods of more active growth, could explain their greater susceptibility to cobalt deficiency as weaned lambs during summer, and again as two-tooths during the following spring-summer period.

On grossly-deficient soils cattle and sheep are continuously subjected to an extreme deficiency of cobalt and ultimately die. But on less seriously affected land year to year fluctuations in the thrift of young sheep suggest that their cobalt intake can change signifi-

cantly from one year to the next. The most striking examples of year to year fluctuations. have been reported from South Australia and similar: though less profound effects have been experienced in New Zealand. Dixon and Kidson (5), investigating cobalt deficiency in the Morton Mains district, Southland, observed that in some years the trouble did not appear in obvious form, while on marginally cobalt-deficient land at Wallaceville year to year variations in response of lambs to cobalt have been found. New Zealand observations suggest that a wet season with lush growth of pasture favours development of the disease.

No complete explanation is as yet available to account for yearly fluctuations in disease incidence. It is probable that one major factor is variation. in availability to plants of soil cobalt, but it is not yet known how season governs availability. Undoubtedly a partial explanation must be in the fact that considerable differences are to be found in the cobalt concentrated in different plant species so that changes in the botanical composition of pastures from year to year, or from place to place, could influence the degree of deficiency to which grazing sheep are subjected.

COBALT CONTENT OF PURE SPECIES

From New Zealand and overseas work it is now well established that legumes 'take up considerably more cobalt than do grasses grown in the same environment. Perennial ryegrass, although not as high in cobalt as are the legumes, tend to absorb more of the- element than do other grasses, short rotation ryegrass, for example. Typical results are shown in- the accompanying table.

TABLE 1

Plant Species	Cobalt (p.p.m. D.M.)
Timothy	0.09
Cocksfoot	0.11
Meadow fescue	0.12
Short rotation ryegrass	0.13
Perennial ryegrass	0.16
Red clover	0.23
White clover.	0.24

It follows 'that, although cobalt deficiency can and does occur on pastures containing any of the usual mixtures, others things being equal; pastures dominated by clovers, or to a lesser extent perennial. ryegrass, are less, likely to be cobalt-deficient than those dominated by other grasses.

INTENSITY OF STOCKING

Field observations have suggested that cobalt deficiency disease tends to be more serious under conditions which favour greater length of pasture. This has been tested experimentally during two seasons on incipiently cobalt-deficient paddocks in Southland. It was found that, when paddocks were heavily stocked with lambs so that pastures averaged about a half to one inch in length, the lambs grew rather slowly, but consistently, and there was no evidence of cobalt deficiency disease. On the other hand when paddocks were lightly stocked so that pastures averaged three to five inches in length lambs grew more rapidly for the first four or five months of life but thereafter they lost weight. Analyses of livers and a controlled cobalt-dosing trial showed that weight losses were due to a deficiency of cobalt.

No definite explanation of these findings is yet available. But it seems probable that a reason lies in the fact that soil is much richer in cobalt than the herbage it grows, so that the protective effect of heavy grazing. was very likely due to the ingestion of soil-contaminated pastures. Whatever the cause or causes the experiment showed that heavy stocking will protect lambs against incipient cobalt deficiency whereas more lenient grazing with consequent greater length of pasture favours development of the disease.

OTHER FACTORS

On severely-deficient country the soil is very low in cobalt and probably always has been, at least within historical times On the other hand on mildlv-affected land it frequently appears that unthriftiness-in young sheep has developed. only within recent years. Restriction of the areas over which animals graze affords a partial explanation of this, Another possibility is that. the land was once healthy and disease has emerged because farming practices have in some way reduced the available cobalt content of the soil. Drainage. cultivation, irrigation, cropping and the use of fertilisers would be ‘expected to modifv soil characteristics and could conceivably influence the amount of cobalt available to pasture plants. At present very little is known concerning the impact of these factors on the development of cobalt deficiency. Work by the Cawthron Institute (6) and others (7, 8) suggests that liming tends to reduce cobalt uptake by pasture plants. Dixon and Kidson (5) however, found that lime was beneficial in controlling cobalt deficiency disease, presumably be-

cause of the relatively high cobalt content of the particular limestone used in their experiments.

Our own trials over a period of three years failed to show that high applications of lime significantly affected cobalt uptake. But this work was of necessity carried out on land which had already received lime. Possibly a different result might be obtained on more acid soils. The effect of superphosphate on the uptake of cobalt from incipiently deficient soils has so far received no attention, but it is of interest that American workers (9) state that "the cobalt content of forage may be reduced to (the) deficiency levels in heavy yields resulting from the use of chemical fertilisers as well as from poultry manure." We do not yet know whether molybdenum applications significantly affect cobalt uptake.

Time does not permit of speculations concerning the possible importance of nutritional and physiological stresses on the cobalt requirement of stock. It should, however, be mentioned that South Australian workers (10) found that cobalt dosing of sheep on incipiently-deficient land prevented phalaris staggers, a disease resulting from the consumption of *Phalaris tuberosa*. It was sugested that increased amounts of vitamin B12 resulting from cobalt dosing may have been required to destroy neurotoxic substances present in, or formed from, the plant.

CONCLUSION

To sum up, in general diagnosis and control of severe cobalt deficiency affecting cattle and mature sheep no longer presents special difficulties.' Affected soil types are well defined, cobalt content of soils, pastures and livers-are invariably low, and animals respond dramatically to cobalt. Present-day problems centre largely around the thrift of young sheep on marginally cobalt deficient land. Here diagnosis is more difficult. Because of the modifying influence of season, and imperfectly-understood man-made environmental factors, soil type becomes a much less reliable guide to the incidence of cobalt deficiency. Chemical analyses of samples for cobalt content are apt to be inconclusive. Properly conducted field trials will give a definite diagnosis although results may not be applicable every year, or beyond the boundary fence.

Current work is mainly concerned with the following :

1. Development of a more rapid and sensatiye laboratory technique for diagnosing cobalt deficiency

disease. -Es&nation of vitamin B12 in animal tissues may provide an improved test.

2. More precise knowledge of environmental factors including those of farm management which favour the emergence of cobalt deficiency. There is an accumulating body of information which suggests that cobalt deficiency disease in young sheep sometimes follows upon improved pastures and increased carrying capacity. If this is true, examination of farm management practices causing unthriftiness becomes of great importance, since much of the suspected potentially-deficient land such as the "pakihi" soils of the west coast of the South Island has yet to be developed. Determination of the way in which these factors work will depend upon knowledge gained from a number of scientific disciplines each occupied with some phase of the'soil-pasture-animal complex.

While it is true that solutions to important problems still lie in the future it should be emphasised that the application of already-existing knowledge has resulted in economic gains which can be measured in millions of pounds per annum. It can be expected that the application of results from current and future research will still further increase these gains.

ACKNOWLEDGMENTS

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DISCUSSION

Q. (Dr Mitchell). Where cobalt deficiency is suspected would the use of cobaltised super be justified as a form of insurance?

A. I do not recommend. cobalt applications unless cobalt has been diagnosed as deficient.

Q. (J. Holden). In the severe cobalt deficient areas of the North Island, cobalt has been applied for many years. Will these farmers have to keep on applying cobaltised superphosphate?

A. Work done by Dr. Filmer showed that on deficient areas after seven years of application of 5 oz of cobalt per year cobalt was still deficient. Finding out the answer to this question is a long term job.

Q. Has cobalt deficiency been found on peat soils ?

A. No, except for an area at Paki Paki on the Heretaunga plains on a soil composed of peat and pumice, where a cobalt response was obtained. The deficiency was most likely due to the pumice portion of the soil.

Q. (McNeil). If cobalt was diagnosed as adequate on liver analysis was it possible that cobalt was still deficient?

A. Yes. There is a big variability factor in the cobalt content of the livers of different animals. It was possible that even with normal levels of cobalt in a liver there may be a cobalt deficiency in the area.

Q. On large properties some paddocks are recognised as being good hogget rearing blocks whereas other paddocks are not. Would it be possible for one area to be slightly cobalt deficient?

A. It could be so. There are often many soil types on one farm. However it may not necessarily be due to cobalt deficiency. Only 5-10% of hogget livers sent in for analysis for possible cobalt deficiency were diagnosed as cobalt deficient.

Q. Is there any harm in putting cobalt on an area if it is not required?

A. Cobalt toxicity is extremely unlikely, but it has happened on two farms to my knowledge.