ZINC IN THE NUTRITION OF DAIRY CATTLE

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Zinc is well established as an essential element in animal nutrition. Nearly 30 years ago it was shown that the skin lesions of zinc deficiency in pigs were prevented by supplementation with zinc (Tucker and Salmon, 1955). Within five years zinc was also shown to prevent the symptoms of what is now known as zinc deficiency in cattle. Much of our knowledge of the clinical signs of zinc deficiency has arisen from studying the signs and lesions of experimentally reproduced zinc deficiency from feeding specially formulated diets or by examining offspring with a heritable zinc malabsorption syndrome.

Biological Functions of Zinc

Zinc is a constituent of a number of metalloenzymes. These include carbonic anhydrase, calf thymus DNA polymerase, carboxypeptidase A and B, alcohol dehydrogenase, α mannosidase, lactic dehydrogenase, alkaline phosphatase, and many others. In addition several other enzyme complexes are activated by zinc. Zinc is also important in the activity of some hormones eg. insulin.

Probably because of its importance in so many enzyme and hormone systems and in structural complexes, zinc is involved in the metabolism of nucleic acids, carbohydrates, lipids and proteins. The homeostatic mechanism involving zinc disposition and its role in metabolism generally is still very poorly understood.

Laboratory and field trials have indicated that zinc is essential for

(a) growth
(b) maintenance
(c) reproduction
(d) defence against injury and infection and repair.

and that deficiencies of zinc from whatever cause will result in perturbation of one or more of these functions.

Clinical Consequences of Zinc Deficiency in Cattle

Laboratory and field experimentation have resulted in the following clinical signs or lesions of zinc deficiency being identified

- lethargy.
- cessation or retardation of growth.
- slowing or ceasing of hair growth with formation of brittle hairs.
- transient frothy or viscous saliva.
- pallour of tongue dorsum (increased numbers of bacteria) and dental pad ulceration.
- loss of taste perception.
- anorexia
- distortion of horn or hoof growth.
- thymic hypoplasia resulting in abnormal cell-mediated immune response and susceptibility to infectious disease. This is especially evident in calves suffering from Adema disease.
- abnormal keratogenesis with skin thickening, cracking and infection. Initially these lesions show about mouth, nose, eyes and other orifices and later about the neck, legs, head and scrotum and base of teats.
- retarded testicular growth and development.

Causes of Zinc Deficiency

Nutritional

Low zinc concentration in the feed is the most common cause of zinc deficiency overseas. Differing figures for dairy feed zinc concentrations low enough to cause zinc deficiency have been quoted by different investigators.

Therefore a conservative lower limit has been suggested for zinc concentration in the total ration of beef and dairy cattle of 25 mg Zn/kg of dry matter (DM) (Grace, 1983). An even more conservative lower limit of 50 mg Zn/kg of DM has been set by the Nutritional Council of the Canadian Feed Industry Association (Smart et al., 1981). In the absense of interfering substances in the diet this requirement for zinc is probably excessive. Several other nutritional factors are known to influence zinc absorption or metabolism although their importance in ruminant nutrition is unknown. These are phytic acid, calcium, copper, iron and molybdenum.

Genetic

An inherited condition of Holstein/Friesian cattle known as A46 or Adema disease has been observed initially in Scotland but also in Italy, Holland, Germany and Denmark. The disease is an autosomally recessive defect of zinc absorption and has many similarities to the similarly inherited condition acrodermatitis enteropathica of man (Weismann and Flagstad, 1976).

Sources of Zinc

The first and most important source of zinc to the new-borne calf is colostrum. Colostrum has high levels of zinc - upwards of 20 mg Zn/kg milk (Schwarz and Kirchgessner, 1975b). These decline rapidly after the first few days of lactation reaching 5 mg Zn/kg milk by about 10 days and declining throughout lactation to about 3 mg Zn/kg milk towards the end of lactation in autumn (N.R. Towers, personal communication). Pasture levels of zinc have been shown to vary within the range (Grace, 1972) 23-70 mg Zn/kg DM (mean 38) for the North Island and 17-27 mg Zn/kg DM (mean 22) for the South Island. The range from 40 Southland and Otago properties (Cornforth, personal communication) was similar, 7-50 mg Zn/kg DM (mean 24). Pasture samples collected at Ruakura throughout the year gave an overall mean ± SD of 38±10 mg Zn/kg DM (range 22-79). A large number of pasture samples submitted to Ruakura for the Auckland and Waikato regions gave a mean ± SD of 39±15 mg Zn/kg DM (mainly in the range 25-50). The lowest level was found in maize and the highest from a weed sample (12 and 157 mg Zn/kg DM respectively). The main causes of variation appear to be composition (legumes are higher in zinc than grasses and cereals) and maturity (mature grass has a lower content than young grass). There appears to be no seasonal variation in zinc content of pastures.

It is important to realise that the body has very limited stores of zinc for meeting long term deficits in zinc. Therefore a continuous supply of zinc is essential.

Most ingested zinc is excreted in faeces and negligible amounts are found in urine. Most control of body zinc homeostasis is at the gut level.

Zinc Deficiency in New Zealand Dairy Cattle?

No cases of zinc deficiency of either nutritional or genetic origin have been recorded in New Zealand dairy cattle. However a perusal of the records of New Zealand pasture zinc concentration suggests that there may be times when transient zinc deficiencies or marginal intakes might exist. It would not be surprising if occasionally zinc responsive conditions did occur under some unusual combination of conditions eg. alkaline soils, grass dominated pasture, high calcium intake, about parturition. Increased demands for zinc in productive animals may arise from

(a) improved genetic and technical advances resulting in further and more efficient animal production eg. breeding, genetic engineering, vaccinating for faster growth, use of growth stimulants;

* 1 mmole Zn = 65 mg Zn

(b) modification of feeding patterns and methods eg. use of maize or urea;

(c) movement from galvanised piping and fittings to plastic.

Diagnosis of Zinc Deficiency

As zinc deficiency has not been recognised as a problem in dairy cattle in New Zealand considerable caution must be exercised in investigating any disease where zinc deficiency is suspected. It must be remembered that zinc homeostasis is upset in several disease and some physiological processes and that low zinc levels in some body fluids and organs may be secondary to some other condition. It is suggested that all the following be considered in arriving at a diagnosis of zinc deficiency.

(a) Clinical signs and pathological findings.

(b) History and environmental conditions including analysis of plant or other fodder.

(c) Zinc concentration in serum, whole blood and liver.

(d) Response to zinc treatment.

(a) Clinical Signs

Clinical signs have been described. Skin lesions, slow growth and possibly reproductive problems should be examined critically to exclude differential diagnoses.

(b) History and Environment

Consider the age, maturity and physiological condition of the animal and the condition, composition and maturity of the pasture, the soil pH and recent topdressing history. Analysis of feed for zinc should be carried out. Analyses showing pasture zinc between 20 and 50 mg Zn/kg DM have been associated occasionally with responses to zinc supplementation although these have not been clearly defined as simple zinc deficiency.

(c) Zinc Concentration in Animal Fluids and Tissues

(i) Blood

During zinc depletion studies in lactating dairy cows (Schwarz and Kirchgessner, 1975a) showed that zinc content of whole blood only showed a definite decline after zinc deficiency lesions were well advanced. However serum zinc concentration decreased to 50% after one week of depletion and 25% of initial levels after 19 weeks. This suggests that serum zinc concentrations give a better indication of zinc deficiency than do whole blood determinations. It must be remembered that other disease conditions (liver disease, chronic and acute infection, acute tissue injury) and the immediate post-partum period are associated with falls in serum zinc concentrations. Table 1 shows the serum and plasma levels for some Ruakura dairy stock. Plasma zinc concentrations for 3-6 month old calves have also been given by Towers et al. (1981).

Stock	N		Mean±SD	Range
Steer and Heifer Yearlings	5	serum	0.94±0.05	0.80-1.05
Mixed Age Lactating Cows	28	serum	1.07±0.14	0.70-1.30
Mixed Age Lactating Cows	12	plasma	0.92±0.14	0.709-1.17

Table 1: Serum and plasma zinc concentrations (µg Zn/ml) for dairy stock at Ruakura.

(ii) Organs

Both liver and kidney show some decline in zinc concentration during zinc deficiency (Miller, 1968; 1970; Neatherey et al., 1973) and the decline may reach 30 to 60% when clinical symptoms of zinc deficiency are apparent. Kidney is not recommended for zinc analyses because of the variation in concentration of zinc between cortex and medulla and the difficulty of obtaining a uniform sample.

A wide ranging survey of some organ zinc concentrations has been reported by Solley et al. (1982). Figure 1 shows the distribution of zinc concentration in cattle livers. Solley (quoted by Towers, 1977) also showed that the mean liver zinc concentration for calves (394 mg Zn/kg; n = 253) was higher than that for other cattle (158 mg Zn/kg; n = 136). Figures from Ruakura dairy cattle indicate that mean liver levels ± SD of 121±25 mg Zn/kg (n = 4), 108±11 (n = 4) and 175±28 (n = 5) for lactating cows, non lactating cows and yearling steers respectively.

Hair is not a good indicator of zinc deficiency.

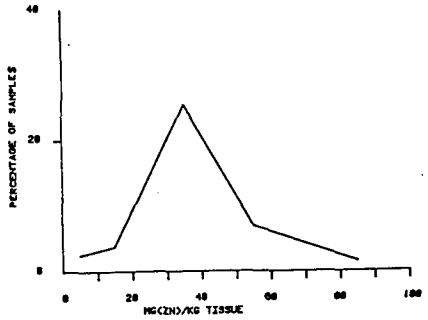

Fig. 1. Frequency distribution curve for zinc concentration in cattle livers throughout New Zealand (from Solley et al. 1981).

(d) Response to zinc treatment

Zinc deficient ruminants usually respond rapidly to the administration of supplementary zinc. Response may be measured by clinical appraisal, weight changes or possibly by increasing activity of serum alkaline phosphatase. However the latter approach should only be incorporated in the investigation of a significant field problem and then in consultation with a suitable laboratory. In order to overcome the influence of possible interfering factors or absorption problems the administration rate of zinc for a zinc response trial should be higher than the rate considered adequate to prevent zinc deficiency. A daily dose rate of 2-3 mg Zn/kg body weight as the oxide or sulphate is suggested. This is equal to a concentration in feed of about 100-150 mg Zn/kg DM.

Calcium, copper, cadmium, selenium, molybdenum and chelates (eg. phytate or EDTA), are known to affect the absorption, availability or metabolism of zinc in monogastric animals but the evidence for such actions in ruminants is limited. Zinc in large quantities may also interfere with the metabolism of some of these chemicals.

Other Zinc Interrelationships

Facial eczema

The prophylactic effect of zinc in preventing facial eczema and some other toxicoses in ruminants is well established. The dose rates of zinc required to cause significant protection are high, about 20 to 30 times normal daily intake.

Wound healing

Supplementing a normal practical diet for dairy cattle with large amounts of zinc oxide or sulphate has been shown to significantly increase the rate of wound healing (Miller et al. 1967). However this effect of zinc on wound healing has been a controversial subject, with some investigators often obtaining no effect of zinc on wound healing. Win (1975) concluded that the positive effects of zinc on wound healing probably occurred in cattle which were marginally zinc deficient.

Copper

Although prolonged zinc administration has been shown to reduce serum levels of copper in cattle and calves (Towers et al. 1981) it has not been shown to cause clinical copper deficiency in sheep or cattle during experiments designed to test the facial eczema prophylaxis effect of zinc. Because preliminary findings suggest that zinc may cause its prophylactic effect through an effect on copper metabolism, (R. Munday, personal communication), it is suggested that copper supplements should not be administered along with zinc in facial eczema control.

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