REVIEW ARTICLE

ARTICLE DE REVUE

A review of copper status of cattle in Canada and recommendations for supplementation

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Abstract

Primary and secondary copper deficiency has been identified in most Canadian provinces. Research has been done investigating the environmental and dietary factors that influence dietary copper availability and the effect of deficient copper status on the health and production of cattle. Across Canada, a high percentage of forages and grains are marginal to deficient in copper. In certain regions, high sulphates in the water and/or high molybdenum concentrations in the feed decrease dietary copper availability. The value of various oral and parenteral copper supplements in the treatment and prevention of copper deficiency is discussed.

Résumé

Revue de la question du cuivre chez les bovins au Canada incluant les recommandations comme additif alimentaire

Les déficiences en cuivre primaires et secondaires ont été identifiées dans presque toutes les provinces canadiennes. Des recherches ont été effectuées afin de déterminer les facteurs environnementaux et diététiques qui influencent la disponibilité alimentaire du cuivre de même que l'effet d'une telle déficience sur la santé et la production des bovins. À travers le Canada, les fourrages et les grains sont dans un pourcentage élevé légèrement déficients en cuivre. Dans certaines régions, des taux élevés de sulfate dans l'eau et/ou des concentrations élevées en molybdène dans la nourriture diminuent la disponibilité alimentaire du cuivre. Les auteurs discutent de la valeur des suppléments de cuivre administrés par voies orale et parentérale dans le traitement et la prévention des déficiences en cuivre.

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Introduction

By the early 1950's, copper (Cu) deficiency in cattle was reported in Europe, Australia, New Zealand, and the United States. In Canada, a Cu-responsive disease was described in cattle in the Swan River Valley of Manitoba in 1953 (1). The disorder had been recognized in the area for 40 years and was associated with pasture molybdenum (Mo) concentrations of up to 25.6 mg/kg dry matter (DM). The disease was characterized by severe diarrhea, stiffness, and emaciation; in some years, the mortality rate of calves reached 30%. However, affected cattle would respond to 2 g Cu sulphate given as a drench or in salt licks.

Based on provincial forage crop analyses, Bell *et al* (2) suggested that trace mineral deficiencies could occur in Saskatchewan. Henderson (3) identified a secondary Cu deficiency related to Mo excess in cattle of eastern Ontario. Miltimore *et al* (4) reported a Cu-responsive diarrhea in cattle of the British Columbia interior which was associated with depressed average daily gains (ADG) and emaciation. Many beef cattle in northern Ontario also were identified to have a low Cu status (5).

The purposes of this paper are to review the literature on Cu nutrition of cattle in Canada, from 1950 to July 1990, to identify the number of cattle at risk and factors which add to this risk, and to outline current recommendations for supplementation. Some papers may have been excluded from this review inadvertently or because we considered them of limited value. The literature was originally searched as part of a PhD thesis (M.E.S.), and has been updated on an ongoing basis. This review is a compilation of information from the files of the three authors.

Clinical disease

Signs of Cu deficiency can be overt or nonspecific. Often, the only sign seen is depression in production. Furthermore, cattle can be clinically normal although their liver and plasma Cu concentrations may indicate a deficient state (6). Research has shown that clinical signs seldom occur until the body's Cu reserves are depleted and a biochemical dysfunction has been created (7). The type and severity of signs depend on such factors as dietary Cu availability, age and breed of cattle, season of the year, and climatic conditions. The changes in tissue and plasma Cu concentrations

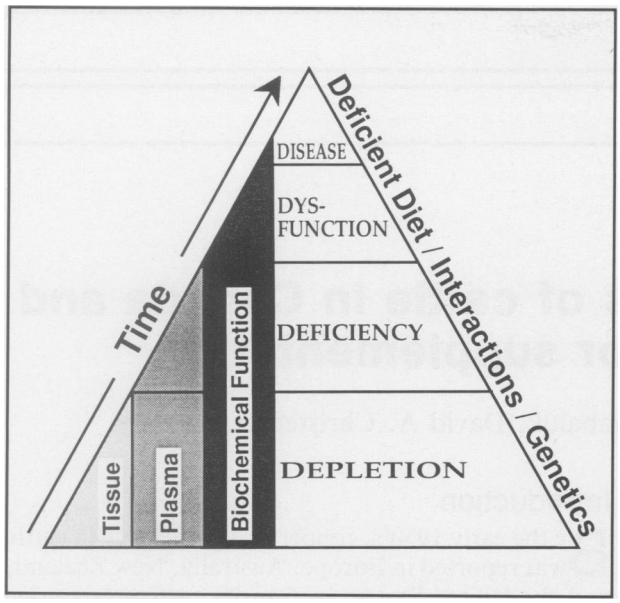

Figure 1. Diagrammatic illustration of tissue, plasma depletion, and the decline of biochemical function over time with a primary copper deficiency or when the availability of dietary copper is reduced by dietary interaction or breed (8). (*Reprinted with the kind permission of C.V. Mosby Company*).

and in biochemical function during inadequate Cu intake are represented diagrammatically in Figure 1 (8). As illustrated, clinical signs represent only a small part of the overall features of Cu deficiency.

Smart *et al* (9) described a field study of Cu deficiency in seven-month-old Simmental calves from north-central Manitoba. The calves were unthrifty, lame, and infected with *Dictyocaulus viviparus*, *Nematodirus*, and *Trichostrongylus* spp. Radiography showed flaring of the distal metaphyses and irregular, widened physes. At necropsy, the enlargement of the metacarpal and metatarsal bones was found to be an accumulation of osseous tissue on the periosteal surface surrounding the physis. The irregular thickness of the physis resulted from tongues of cartilage which extended into the metaphysis. In addition, the zone of hypertrophic chondrocytes was greatly thickened. The herd responded to an *ad libitum* salt mix of 2.5% Cu sulphate.

Copper deficiency has also been reported in Saskatchewan in three-to-four-month-old Simmental calves consuming high sulphate (1900 mg/L) water (10). The calves were unthrifty, lame, and had enlarged distal metacarpal and metatarsal metaphyses. A heavy louse infestation and coccidiosis were present. The calves responded to 5 g feed grade Cu sulphate given as an oral bolus daily for seven days. As well, a salt mix containing 5% feed grade Cu sulphate was available *ad libitum*. After clinical improvement, the Cu sulphate in the salt was reduced to 2% and this prevented recurrence of signs.

In a study of bovine enteric coccidiosis in western Canada, calves with neurological signs had lower liver Cu concentrations and higher plasma glucose concentrations than those without neurological signs (11). These authors suggested that hyperglycemia and Cu deficiency could increase the susceptibility of the central nervous system to damage. These three reports support Peck's (12) hypothesis that Cu deficiency may make calves more susceptible to internal parasites.

Furthermore, involvement of Cu in normal central nervous system function has been implied through studies of bovine polioencephalomalacia (PEM). In a PEM-affected steer, the combination of high sulphur (0.38%) and low Cu (7.1 mg/kg DM) in the diet was concluded to reduce blood thiamine and plasma Cu content (13). Copper supplementation produced substantial improvement in both the thiamine and Cu status of the herd.

Animal factors influencing copper status

Many factors influence plasma and liver Cu concentrations in cattle, including breed, age, season, feed composition, and dietary antagonists. Many of these factors have been investigated. Puls (14) has reviewed these studies and has published values for tissue and serum Cu concentrations that indicate a deficient, marginal, or adequate Cu state in cattle (Table 1). Although ceruloplasmin activity is highly correlated with serum or plasma Cu concentration in cattle, the relationship between ceruloplasmin activity and liver Cu concentration is not well defined (15) and will not be discussed in this review.

Age and breed affect plasma Cu concentrations in cattle. Cows less than four years of age had the lowest plasma Cu concentrations (16). In this Saskatchewan study, weanling Simmental-sired calves had lower plasma Cu concentrations than Angus and Hereford-sired calves. Weaned calves from two-year-old heifers had lower plasma Cu values than calves from older cows (16). These breed differences were confirmed by Smart (6), who found that Simmental cattle had higher Cu requirements than Herefords or Angus (6). Plasma Cu concentrations were lower in Simmental-sired heifers than in Hereford-sired and Angus-sired heifers (17). However, all the heifers in this study were Cu deficient.

Table 1. Plasma and liver copper concentrations for use in evaluating the copper status of adult cattle[a]

Copper status	Plasma		Liver	
	mg/L	μmol/L	mg/kg DM	μmol/g DM
Deficient	0.06–0.55	0.9–8.7	3.5–35.0	0.06–0.55
Marginal	0.55–0.70	8.7–11.0	17.5–70.0	0.28–1.10
Adequate	0.80–1.20	12.6–18.9	87.5–525.0	1.40–8.30

[a]Modified from Puls (14)

Seasonal fluctuations in plasma Cu concentrations have been reported (6). The lowest Cu concentrations occurred in February and March whereas highest concentrations were found in August and September (6). These fluctuations are modified by dietary Cu intake and stage of pregnancy, but are independent of liver Cu concentrations.

Stage of pregnancy affects the Cu kinetics of the fetus and of the dam. During the first trimester of pregnancy, Cu concentrations of the fetal calf's liver were 3.1 μmol/g DM and were independent of dam's age, breed, or Cu status (16). Previously, Smart and Christensen (18) had found that liver Cu content declined sharply after 180 days gestation in fetuses from cows who had deficient liver Cu concentrations (<0.47 μmol/g DM).

Although fetal liver Cu is unaffected by the dam's Cu status, dietary Cu intake of the cow during pregnancy modifies the liver Cu concentration of the neonatal calf (6). Higher Cu intakes by the cow result in greater liver Cu concentrations in the calf. However, caution must be used in supplementation of the dam because the amount of Cu which could contribute to fetal Cu toxicity has not been determined. The newborn calf has a naive Cu homeostatic system. The ceruloplasmin system does not mature until four days after birth when Cu is released from the liver and plasma Cu concentrations reach adult values. This postnatal increase in plasma Cu concentration of the calf was shown to be independent of cow or calf liver Cu concentrations (6).

Gooneratne *et al* (19) subsequently examined the effects of different dietary Cu and sulphur intakes on maternal and fetal liver Cu concentrations and on fetal growth. Copper associated with the metallothionein fraction increased during gestation in the livers of fetuses from cows fed a high Cu (30 mg/kg DM)-low sulphur (0.1%), or a low Cu (5 mg/kg DM)-low sulphur (0.1%) diet. Liver Cu declined during the last month of gestation in fetuses of cows fed a high sulphur (0.35%)-low Cu (5 mg/kg DM) diet. However, liver Cu content decreased during pregnancy in cows of all treatment groups. In a survey of pregnant cows slaughtered in Saskatchewan, fetal liver Cu concentrations declined when maternal liver concentrations fell below 0.79 μmol Cu/g DM (20). This effect was most marked in the last third of pregnancy.

Sixty-seven percent of the livers collected from cattle slaughtered in Saskatchewan between April and October had Cu concentrations below 0.55 μmol/g DM. These values suggest a Cu deficiency (21). In a national study evaluating tissue trace mineral concentrations of slaughtered cattle, the average hepatic Cu concentration was 1.5 μmol/g DM (22). Deficient liver Cu concentrations (<0.55 μmol/g DM) were found in 29% of the slaughtered cattle. An additional 35% had liver Cu concentrations between 0.55 and 1.7 μmol Cu/g DM, indicative of a marginal Cu status.

Trace mineral content in hair is highly variable. In pregnant Hereford cows fed a diet containing adequate Cu (19 mg/kg DM), unpigmented hair was lower in all minerals than pigmented hair (23). The average Cu content of the hair was 0.05 μmol/g DM.

Mineral interactions

Plasma direct and trichloracetic acid-soluble Cu concentrations, ceruloplasmin oxidase activity, and milk yield decreased more rapidly in cows fed diets containing 40 mg Mo/kg DM than in cows fed 0 or 20 mg Mo/kg DM (24). The Cu content in all diets was 6 mg/kg DM. Although milk from the cows fed the high Mo diet had a high Mo content, this did not affect the Cu status of the calves. However, plasma Mo values in these calves increased directly in relation to Mo concentration in the milk.

Holstein bull calves fed a basal diet containing 20 mg Cu/kg DM, to which 100 mg zinc (Zn)/kg DM was added, had lower liver Cu concentrations than calves fed the same diet containing 0 or 40 mg Zn/kg DM (25). Excess selenium (Se) intake (2.37 mg/kg DM) did not alter Cu metabolism in dairy cows over 276 days of lactation (26). A Cu and Se interaction has been postulated, but consistent results demonstrating this effect have not been seen.

Reducing the sulphur content from 500 to 42 mg/L in the drinking water of pregnant beef cows enhanced the individual cow's Cu status, but not that of her calf (27). Smart *et al* (28) subsequently found that the plasma Cu concentrations of cows injected with copper calcium edetate (CuCaEDTA), and provided with drinking water from which sulphur and iron were removed by reverse osmosis, were higher and more stable than in cows fed raw, untreated water and not given supplemental Cu (28). Although plasma Cu was affected, liver Cu concentrations were not influenced by treatment in these cows. Sulphate removal, but not iron removal, improved plasma Cu concentrations of both the cow and calf. Injecting pregnant cows with CuCaEDTA improved the plasma Cu status of the cow and calf, and the liver Cu concentration of the calf independent of water treatment (27).

Soil factors

In a slaughterhouse survey, low maternal and fetal Cu concentrations were found in pregnant beef cows originating from areas of grey-wooded soils and muskeg regions in Saskatchewan (20). Copper deficiency has been shown to prevail in grey-wooded soils, and was probable in seven other soil associations in Saskatchewan (29). For cereal crops, the critical level of extractable Cu in soil was about 0.4 mg/kg DM. However, plant uptake of Cu can be affected by species and cultivar of plant, precipitation, and generally is not highly correlated with soil-extractable Cu concentrations (30).

A study of the Cu and Mo status of pastures in eastern Saskatchewan found that forages grown on saline soils contained the lowest Cu concentrations (31). In Manitoba, Ashville, Favel, and the Vermillion River geological formations contain Mo-rich shales which can contribute to excess Mo in forages and to the secondary Cu deficiencies seen in livestock in these areas (32).

Feed factors

Molybdenum is one of the most important Cu antagonists in feeds. In British Columbia, a dietary

Cu:Mo ratio of less than 2 was concluded to be significant in creating a conditioned Cu deficiency in cattle (33, 34). The average Cu content of forages in British Columbia (1969–1984) was 8.4 ± 4.0 mg/kg DM, but ranged from 1.0–20 mg Cu/kg DM (35). Legume feeds tended to have higher Cu concentrations than corn silage or grass hays. The range of forage Cu values was similar for all areas, but, overall, forages sampled in the Kootenay region had the lowest Cu concentrations (35). Copper concentrations in corn silage were positively related to acid-detergent fiber, but this relationship did not hold for hays grown in the south coastal region of British Columbia (36). However, Cu concentrations were positively related to crude protein content in corn, grass silage, and hay.

In Alberta, Redshaw *et al* (37) found that 84% of wheat grain, 54% of barley grain, 28% of oat grain, 47% of legume roughage, 46% of grass-legume roughage, 44% of oat roughage, 42% of barley-oat roughage, and 27% of barley roughage contained less than 10 mg Cu/kg DM. However, copper distribution in the forages across the province did not show any consistent pattern. Weisenburger (38) found that 46% of the fresh and conserved forages sampled in the Medicine Hat area contained less than 5 mg Cu/kg DM. Ninety-five percent of the forages had less than 10 mg Cu/kg DM, with an overall average Cu content of 5.9 mg Cu/kg DM. The average Cu content of cereal grains was 5.1 mg/kg DM with a range of 0.6 to 18.2 mg/kg DM (38).

Copper concentrations of Saskatchewan pastures varied annually and were either deficient or adequate depending on rainfall (39). Copper-deficient herbage occurred with above normal rainfall; Cu in herbage was adequate with below normal rainfall. In Saskatchewan, the average Cu content of commonly used forages was determined to be about 10 mg/kg DM, but 63–67% of alfalfa hays and silages, 42% of alfalfa-brome hays, and 67% of brome hays contained less than 10 mg Cu/kg DM (40). Martin *et al* (41) found that rations of high producing dairy herds in Saskatchewan contained 7.9 mg Cu/kg DM compared to 10.7 mg Cu/kg DM for low producing herds. These researchers could not resolve the discrepancy of this observation because plasma and liver Cu concentrations were not analyzed.

Legumes grown in northwestern Manitoba generally have a higher Cu content than do grasses (32). Only 16% of grasses and 38% of legumes grown in north-central Manitoba supplied adequate Cu intakes for cattle.

In Ontario, forage Cu concentrations range from 4–7 mg/kg DM. Grains contain about 3–4 mg Cu/kg DM (42).

Shorthorn cows kept outside had higher plasma Cu concentrations than housed cows (43). Furthermore, feeding silage reduced plasma Cu values compared to feeding hay. These observations were confirmed by Ho *et al* who also observed that silage-fed cattle had lower plasma Cu concentrations than cattle fed hay (44).

Copper supplementation studies

In many areas of Canada, Cu supplementation of the diet is essential to maintain normal Cu status in cattle. Before the type and amount of Cu supplementation is chosen, the nutritionist must not only know the Cu concentration in the feed, but also the concentration of all dietary elements which might influence dietary Cu availability. Prolonged, excessive supplementation can result in Cu toxicity.

After a single oral dose of 15 g Cu sulfate, analysis of rumen fluid indicated that Cu supplementation did not affect live bacteria, total protozoa, or total volatile fatty acid concentrations (45). Cattle grazing a pasture containing 7.7 mg Cu/kg DM with an average Cu:Mo ratio of 1:1 showed an increase in ADG after injection of 100 mg Cu as CuCaEDTA (34). Hepatic Cu storage was then examined in cattle supplemented with injectable CuCaEDTA, oral Cu sulphate, or chelated Cu ($Na_2CuEDTA.3H_2O$) (46). In the first trial, Cu sulphate (0.5% Cu) was added to salt and the average daily voluntary salt intake was 35 g/head. Liver Cu in these yearling Jersey steers increased from 1.75 to 5.20 μmol/g DM after 333 days. In a subsequent study, the response to oral Cu sulphate (0.25% or 0.75% Cu), chelated Cu (0.05 or 0.25% Cu), or CuCaEDTA injections (100 mg Cu) was investigated. Daily voluntary intake of Cu-supplemented salt was 42 g/head. Copper source did not alter hepatic Cu storage over the 87-day study period (46).

Intraruminal commercial soluble glass boluses containing Se and Cu did not prevent a decrease in serum

Table 2. Beef and dairy cattle at risk of developing copper deficiency

	Total population[a]	Estimated cattle population at risk[b]	
Province	No. × 10^3	%	No. cattle × 10^3
British Columbia	672.0	10	67.2
Alberta	3800.0	10–15	570.0
Saskatchewan	2050.0	25	512.5
Manitoba	1080.0	10–15	162.0
Ontario	2250.0	10–15	337.5
Quebec	1492.0	?	?
Maritimes	357.4	5	17.9

[a]From Report on Livestock Surveys Catalogue 23.008 Statistics Canada (63)
[b]Estimates based on literature reviews and personal communication with researchers

Cu concentration of calves over a summer pasture period (47). In contrast to this study, Smart *et al* (unpublished data) found that Se-Cu boluses improved the liver Cu concentration of pregnant beef cows. In the latter study, liver Cu concentrations of the pregnant cows reached peak values five months after administration. Liver Cu concentrations of three-month-old calves also were positively affected.

The depression in plasma and milk Cu concentrations in cows fed grass silage was prevented by feeding salt either supplemented with Cu sulphate (0.14% Cu) or supplemented with chelated CuNaEDTA (0.018% Cu) free-choice (44). In this study, cattle that were fed hay consumed about 15 g mineral/head daily while those fed silage consumed about 26 g mineral daily.

Pregnant and open Shorthorn cows fed a grass silage diet known to produce hypocupremia were supplemented free-choice with a mineral containing a sequestered Cu (Cu polysaccharide complex, 0.024% Cu) (48). Daily mineral intakes by nonsupplemented pregnant cows, open cows, or supplemented pregnant cows were 27.4, 17.4, and 37.2 g, respectively. Plasma Cu concentrations of the nonsupplemented, pregnant cows declined over winter. Copper supplementation prevented hypocupremia in 80% of the cows. Open cows maintained a normal plasma Cu content throughout the trial. The authors suggested that the soluble protein in the silage reduced *in vivo* Cu availability.

Copper-deficient yearling heifers that were fed Cu-adequate diets gained weight while on pasture although plasma Cu concentrations decreased (49). As expected, heifers of normal Cu status did not show a weight response when fed an adequate Cu intake. Daily free choice mineral (0.5% Cu) intake was 16 g/head and was significantly less than anticipated (90 g/head).

Steacy *et al* (50) found, in a central Saskatchewan ROP bull station, that 76% of the bulls had plasma Cu concentrations below 8.7 μmol/L. Simmental and Galloway bulls had the lowest plasma Cu concentrations. When dietary Cu content was increased from 5 to 15 mg/kg DM, the plasma Cu concentration had returned to normal within 28 days. However, improvement in plasma Cu concentration was not associated with improved weight gains.

Copper oxide needles given orally to cows (20 g) and calves (4 g) had no effect on body weight over an 85-day grazing trial (51). The Cu content of the grass and legume pastures used in the trial were 3.9 ± 0.29 and 7.9 ± 0.58 mg/kg DM, respectively. Liver Cu concentrations increased over the grazing period and were highest in the Cu-supplemented cows and calves. Initial liver Cu concentrations indicated that the cows and calves had a normal Cu status. Average daily gains and liver Cu content of feedlot steers improved when a mineral containing Cu, Zn, manganese (Mn), and Se was fed (52). Liver Cu increased directly with level of supplementation.

In northwestern Manitoba, changes in plasma Cu concentration were examined in 40 selected beef herds (53). Twenty-seven herds were given a single intramuscular injection of Cu glycinate (60 mg available Cu) in the spring. The region of study was divided into an eastern section, where pasture Cu content was low, and a western section, where excess Mo existed in the forage. A single dose of Cu glycinate effectively supplemented cattle throughout the pasture season in the eastern area, but was ineffective in maintaining normal Cu status in cattle of the western region. The injectable Cu may have acted to increase serum Cu or may have prevented a drop in serum Cu concentration.

Injectable CuCaEDTA, Cu glycinate, and Cu methionate were then evaluated as Cu supplements for grazing cattle (54). After 90 days, serum Cu content was higher in all treated cattle than in controls, although there was no statistical difference among Cu treatments. Based on initial serum Cu values, the cattle in these studies were marginally or overtly Cu-deficient, yet there was no body weight response to Cu supplementation. The absorption coefficient for the forage Cu varied with the pasture site, but ranged from 0.024 to 0.054.

Eight herds of beef cattle from northwestern Manitoba were supplemented with Cu as injectable Cu glycinate, or 0.25 or 0.5% Cu as Cu sulphate, in a mineral supplement fed free-choice (54). A single dose of injectable Cu at the start of the pasture season or use of the 0.25% Cu supplement effectively elevated blood Cu when the dietary Mo and total sulphur (S) were less than 3 mg/kg DM and 3 g/kg DM, respectively. When dietary Mo and total S concentrations exceeded these values, injectable Cu had to be given at 90-day intervals or oral supplements had to be increased to 0.5% Cu (fed as Cu sulphate). Free choice mineral supplementation was satisfactory in maintaining plasma Cu concentration. Because the Cu status of the cattle was so varied, there was no uniform improvement in body weight with Cu supplementation.

Yearling steers grazing pastures containing 3.3 mg Cu/kg DM (grasses) or 6.9 mg/kg DM (alfalfa) and injected with a Cu (125 mg Cu oxide) and Zn preparation showed an increase in liver Cu concentration and also a slight elevation in serum Cu over a 113-day grazing period (55). The effect on serum Cu was not consistent and did not reflect liver Cu stores in some sampling periods. The liver Cu concentration of 0.63 μmol/g DM at the start of the trial did not indicate Cu deficiency; thus, no growth response was evident or expected.

Cattle population at risk of developing copper deficiency in Canada

The cattle population at risk in Canada by province is outlined in Table 2. In each province, Cu status depends on feed Cu content and those factors in the feed and water (for example, Mo, S or Fe) that decrease the availability of dietary Cu.

Korsrud *et al* found that 28.7% of the cattle slaughtered in Canada were deficient in Cu (22). Based on annual slaughter statistics, this means that about 895,000 head of cattle would benefit from Cu supplementation. An additional 35.4% of slaughtered cattle have a marginal Cu status and Cu supplementation would be recommended for these cattle. This

Table 3. Average copper, zinc, and manganese concentrations in feeds (mg/kg, 90% DM basis)

Province	Trace element	Forages				Grains			Pasture	Reference
		Grass hay	Legume hay	Cereal silage	Corn silage	Oats	Barley	Corn		
P.E.I.	Zn	—	27.0	—	—	—	27.0	—	—	64
1987	Mn	—	25.8	—	—	—	13.0	—	—	
N.B.	Cu	5.7	9.7	—	—	—	—	—	—	65
1978-79	Zn	33.5	42.5	—	—	—	—	—	—	
	Mn	53.0	68.0	—	—	—	—	—	—	
Ontario	Cu	6.0	7.3	6.0	5.2	3.5	5.3	2.3	—	66
1980-87	Zn	22.7	23.5	23.8	23.5	31.2	32.7	19.7	—	
	Mn	42.1	28.6	32.3	22.3	35.5	15.5	5.2	—	
Manitoba	Cu	4.4	7.2	—	1.0	—	2.3	—	2.7	67
1981	Zn	14.8	13.2	—	14.5	22.4	26.7	—	18.0	
	Mn	42.8	44.2	—	—	30.0	10.7	—	30.6	
Sask	Cu	10.0	9.5	10.0	7.5	3.0	5.0	—	—	40
1976-77	Zn	27.0	25.0	31.0	14.6	30.0	34.0	—	—	
	Mn	44.0	44.0	55.0	56.0	40.0	18.3	—	—	
Alberta	Cu	5.2	7.0	4.1	5.4	4.6	6.5	2.5	—	69
1976-86	Zn	24.4	23.5	25.3	52.9	32.6	41.2	22.8	—	
	Mn	75.0	35.7	47.3	51.9	43.9	18.8	6.8	—	
B.C.	Cu	6.2	9.8	6.5	7.1	6.3	5.5	6.3	—	35
1969-84	Zn	24.0	22.8	50.3	24.9	28.8	24.3	26.5	—	
	Mn	102.8	36.7	22.7	39.9	52.3	69.3	42.4	—	
NRC	Cu	—	11.0	6.0	10.0	7.0	9.0	4.0	—	68
1982	Zn	—	24.0	35.0	21.0	41.0	19.0	14.0	—	
	Mn	—	31.0	43.0	30.0	42.0	18.0	5.0	—	

NRC requirements for dairy cattle (56) in mg/kg DM: Cu-10; Zn-40; Mn-40 (value × 1.1 = 100% DM)

represents a further 1,100,000 head of cattle that might improve productivity with Cu supplementation.

Recommendations for supplementation

Most forms of Cu supplementation effectively improve the Cu status of cattle. The producer's acceptance of the supplement often depends on the ease by which the supplement can be used and the cost benefit. The National Research Council dairy (56) and beef cattle (57) requirement bulletins recommend dietary concentrations of 10 mg Cu/kg DM. However, experimental evidence suggests that, when dietary mineral interactions occur, up to 50 mg Cu/kg DM may be needed (20). The average trace mineral concentrations found in provincial feeds are summarized in Table 3.

The following are suggested routes of supplementation:

1. Soil and foliar application of copper

In certain Cu-deficient areas of the world, the Cu concentrations in pasture and forages have been effectively increased by soil application of 5.6 to 11.2 kg Cu sulphate per hectare (58,59). Foliar application of Cu sulphate also has been used. However, in Manitoba such techniques have not been useful in promoting Cu uptake by bromegrass or alfalfa because soil Cu is more available from acid soils than from alkaline soils which predominate in Manitoba (60). Care must be taken when foliar supplementation is used because toxic Cu concentrations can potentially accumulate in the soil and plant.

2. Parenteral injections of copper

Injectable Cu salt preparations can supply a slowly released Cu depot from subcutaneous tissues. The number of doses required during the year depends on the severity of the Cu deficiency and the type of Cu complex administered. The consensus is that these preparations must be given every three months to be effective in cattle fed Cu-deficient diets. However, if CuCaEDTA is given every three months over several years, there appears to be no apparent improvement in liver Cu with further use (6). The response of cattle to parenteral Cu supplements is variable. In our clinical experience, the best response with injectables occurs when treating overt Cu deficiency.

Parenteral forms of Cu can cause mild to severe local tissue reactions, particularly after repeated injections. The severity of the reaction depends on the preparation used. Copper EDTA caused minimal reaction, whereas Cu methionate and Cu glycinate can cause severe reactions (60).

These preparations are useful when oral supplementation is impractical. However, at present none of the parenteral Cu preparations are licensed for use in Canada. Newer preparations are being developed that reduce the associated tissue reaction and improve long-

term availability. Precautions should be taken when parenteral Cu is used in combination with other Cu supplements because Cu toxicity can develop.

3. Oral supplementation with copper

The most common method of Cu supplementation is through the diet. The chemical forms of Cu that can be used include Cu complexed to sulphate, oxide, a chelate, proteinate, or diamine peptides. The availabilities of these forms of Cu are quite different. The level of dietary supplementation needed depends on the total dietary Cu intake, diet composition, level of dietary antagonists, and the Cu requirements of the cattle (7).

Salt and mineral supplements specifically fortified with trace minerals are available commercially. The trace minerals in these supplements are commonly in balance with each other. Trace mineral salts are formulated to provide a specific amount of mineral when fed free-choice or by addition to a grain supplement. However, free-choice mineral intake is variable and depends on mineral palatability, the mineral content of water, the location of the mineral, and on individual animal preference. Free-choice supplementation is not ideal, but, if it is the only practical method of supplementation, then the mineral or salt intake must be monitored to determine if intake is adequate. The supplement should be located on pasture in areas where cattle congregate during a significant part of the day or in an area of the field or pen easily accessible to the cattle.

Inorganic copper chelates, or organic copper chelates of protein or amino acid diamines, have been developed for use where dietary interactions occur. These products are promoted to have a high biological Cu availability as they bypass complexation in the rumen. Copper availability from Cu proteinate was greater than from Cu sulphate for Holstein calves fed Mo-containing diets (61). More research is required to determine the efficacy of these products.

Copper oxide needles in a gelatin capsule, and sustained-release trace mineral glass bullets, have been developed to provide a slowly-released Cu supplement from the abomasum or rumen. An oral dose of 13 g Cu oxide needles/100 kg live weight was effective in maintaining a normal Cu status of cattle for up to six months (62). Neither of these products is currently licensed for use in Canada.

Maternal supplementation with copper

Little is known about the harm or benefit that Cu supplementation of the dam may have on the fetus or neonate. When the dam is Cu deficient, we know that, after 180 days of gestation, fetal liver Cu concentrations no longer increase as they do in Cu-replete cattle (18,20).

We know little of what happens to the fetus if the dam is oversupplemented. Questions that need to be answered are: 1) does the fetus indiscriminately accumulate Cu in the liver or do control mechanisms exist to restrict accumulation, and 2) if the accumulation is indiscriminate and toxic concentrations develop prenatally, will the maturation of the ceruloplasmin system in the neonate be associated with a sudden release of toxic concentrations of Cu that may be detrimental? CVJ

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