# Mineral Nutrition of the Beef Cow to Impact Immunologic Response

C.K. Clark, R.P. Ansotegui, J.A. Paterson

Animal & Range Sciences Department Montana State University Bozeman, Montana

The role of minerals in beef cattle diets continues to be a strong area of interest for producers, veterinarians and scientists. Minerals are required for a variety of metabolic functions including the immune system's response to pathogenic challenges. Maintaining the immune response through proper mineral supplementation may have a positive impact on herd health and ultimately, profitability of cow-calf operations. However, mineral supplementation strategies quickly become complex because of differences in forage mineral bioavailability, interactions among minerals which can inhibit mineral absorption, and difficulty in easily assessing cow mineral status. The effects of copper, zinc, cobalt and chromium on the immune system are the primary focus of this paper.

## **Immune System Background**

When an animal is exposed to a foreign substance (antigen), the defense mechanism of the immune system has three ways to respond; 1) cell-mediated immunity, 2) humoral immunity and 3) the phagocytic system. Resistance to a disease challenge is dependent upon the speed and effectiveness of each response mechanism. The immune system is primarily comprised of lymphocytes and phagocytes which are produced from cells in the bone marrow. Lymphocyte production and differentiation is regulated by lymphoid organs, such as the spleen and lymph nodes. Vitamins A and D are important for differentiation of lymphocyte cells into Tlymphocytes and B-cells. Cell-mediated immunity requires T-lymphocytes and these cells mature in the thymus and then accumulate in the lymph nodes from where they respond to antigens. The functions of the Tcells include killing virus-infected cells, helping B-cells build antibodies, regulating the level of immune response and stimulating activity of other immune cells, such as macrophages. One routine method for evaluating a cell-mediated response is intradermal injection of a foreign substance such as phytohemagglutinin (PHA-P, a bean protein) and then measuring the swelling of the skin at specific hours post-injection.

The second type of response is referred to as humoral immunity. B-cells are processed in intestinal lymphoid tissue and differentiate into antibody-producing cells. The B-cells migrate to areas other than the thymus and produce antibodies in the presence of an antigen and help form antigen-specific T-cells. Some antigens are T-independent and can directly stimulate B-cells. The humoral immune response is measured as an antibody titer in blood serum.

In the process of vaccinating animals against diseases, an antigen is introduced as either a modified live or killed organism. The B-cells respond to the vaccine and build antibodies. The level of antibodies produced is at a maximum level approximately 10 days after injection and is referred to as the "primary response." When the vaccine is injected a second time, there is a much more rapid response by B-cells and higher levels of antibodies are produced which persist for months. The response to a booster vaccination is called the "secondary response."

The maximum response by T-cells and B-cells may require days, while the phagocytic system responds immediately and utilizes both neutrophils and macrophages. Neutrophils are responsible for ingesting pathogens and degrading them (phagocytosis). Neutrophils produce free radicals and oxidants to destroy the membrane of the engulfed pathogen. In the absence of adequate antioxidants, the membrane of neutrophils may also be destroyed and reduce the function of the neutrophil. Actions of the macrophage include phagocytosis, processing and presenting antigens to lymphocytes, and releasing substances which promote responses of the immune system.

Nutritional status influences an animal's immune

Paper presented at the Annual Conference for Veterinarians and Veterinary Technicians, Food Animal Program; University Minnesota, October 25-26, 1994. system and its ability to respond to pathogenic challenge. In order for the immune system to function properly, energy, protein, vitamins, and minerals must be provided to the animal in adequate amounts. Nutrient deficiencies have resulted in depressed immunocompetence and increased susceptibility to infectious disease (Beisel, 1982). Energy is required for protein synthesis and cell replication. Protein provides amino acids which are needed for acute phase proteins, antibodies, and cytokines. Vitamins A and D are necessary for lymphocyte differentiation into T-cells and B-cells. Vitamin E serves as an antioxidant to protect cellular membrane polyunsaturated fatty acids in the process of phagocytosis by neutrophils. Minerals also contribute to the antioxidant enzyme system.

## **Trace Elements Role in Immunity**

Trace elements that are thought to have an impact on productivity of grazing cattle include copper, zinc, cobalt, manganese, selenium and iodine. Deficiencies of these minerals have been shown to alter various components of the immune system (Suttle and Jones, 1989). Specific trace mineral deficiencies which have been shown to decrease neutrophil function in ruminants include copper, cobalt, selenium and molybdenum (Jones and Suttle, 1981; Aziz and Klesius, 1986; MacPherson *et al.*, 1987). Decreased cellular immunity, lowered antibody response and disrupted growth of Tdependent tissue have resulted from inadequate intake of zinc (Fletcher *et al.*, 1988). Trace mineral requirements for beef cattle are given in Table 1.

## Table 1.Trace Mineral Requirements of Beef Cattle<br/>Given as Dietary Concentration (ppm).

e Cu	Zn	Mn	Co	-1	Fe	Se	Cr
4 8	30	40	0.1	0.5	50	0.2	
0 12	30	25	0.1	0.5	30	0.1	
0 10 <sup>₫</sup>	45 <sup>e</sup>	40	0.1	0.5	100	0.3	
	4 8 0 12	4 8 30 0 12 30	4 8 30 40 0 12 30 25	4 8 30 40 0.1   0 12 30 25 0.1	4 8 30 40 0.1 0.5   0 12 30 25 0.1 0.5	4 8 30 40 0.1 0.5 50   0 12 30 25 0.1 0.5 30	4 8 30 40 0.1 0.5 50 0.2   0 12 30 25 0.1 0.5 30 0.1

<sup>a</sup>National Research Council Beef Cattle Requirements <sup>b</sup>Agricultural Research Council

Puls, 1990, Mineral Levels in Animal Health

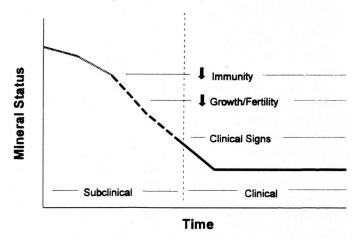
<sup>d</sup>Copper: Molybdenum ratio; minimum 3.0, adequate 4.3, ideal 6.0-10.0 <sup>°</sup>Requirement is considered adequate with 0.3% calcium also in the diet, however; for each additional 0.1% increase in calcium add 16 ppm zinc.

Subclinical mineral deficiencies in cattle may be a larger problem than acute mineral deficiency because specific clinical symptoms are not evident so as to allow the producer to recognize the deficiency (Figure 1). Animals with a subclinical status can continue to reproduce or grow but at a reduced rate, have decreased feed efficiency, and the immune system may be depressed.

#### Copper

Copper deficiency has been identified as the most common trace mineral deficiency of cattle in the world (McDowell, 1985). Copper is an essential trace element required for numerous enzyme systems; iron metabolism, mobilization and connective tissue metabolism, integrity of the central nervous system and the immune system. Copper functions in the immune system through the following: energy production, neutrophil production and activity, antioxidant enzyme production, development of antibodies and lymphocyte replication (Nockels, 1994). The importance of copper for maintaining the functions of the immune system has been demonstrated in several studies. Copper deficiency in cattle can alter the immune system and increase an animal's susceptibility to disease (Smart, 1981). Viral and bacterial challenges have been shown to increase serum ceruloplasmin and plasma copper in copper-repleted cattle indicating a major protective role for copper in infectious diseases (Stable et al., 1993). Low copper status has resulted in decreased humoral and cell-mediated immunity, as well as decreased neutrophil bactericidal capability in steers (Jones and Suttle, 1981; Xin et al., 1991).

**Figure 1.** Effects of Trace Mineral Deficiencies on Immune Function in Cows and Calves



Source: Wikse, 1992, TAMV Beef Cattle Short Course

During gestation, the developing calf is dependent on its dam for supplying copper, manganese, zinc and selenium. The fetus accumulates copper in the liver at the expense of the dam with most of the accumulation occurring after 180 days of gestation. Lower than normal tissue reserves in the fetal calf as a result of deficiency in the dam can impair development and growth (Abdebrahman and Kincaid, 1993). Deficient copper levels in cows have resulted in increased incidence of scours in their calves (Smart *et al.*, 1986). Occurrence of abomasal ulcers shortly after birth and respiratory problems have both been attributed to inadequate copper levels in calves. (Naylor *et al.*, 1989)

Copper deficiency in cattle may be caused by low levels of copper in forage. Absorption of copper is higher for dried forage compared to fresh grasses due to a slower passage rate through the gut allowing for more absorption. Interactions with other dietary mineral sources must be given consideration; particularly high levels of molybdenum or sulfur. Molybdenum ties up copper making it unavailable to the animal and creates problems when dietary molybdenum levels are in excess of 1-3 ppm or the copper to molybdenum ratio falls below 3:1. The interaction of molybdenum and copper can be intensified with sulfur. Sulfur forms thiomolybdates which bind to copper in the rumen to form insoluble complexes that are poorly absorbed (Gooneratne et al., 1989; Spears, 1991). In general, 10 ppm copper (dry matter basis) in the diet is considered adequate. Providing 0.2-0.5% copper in a mineral mix can correct a copper deficiency caused by low forage copper. However, an excess of 0.5% may be required if interactions with other minerals decrease copper absorption. It is important to assess the copper status of cattle before routinely adding excess copper to diets because ruminants are more sensitive to copper toxicity than are nonruminants. Copper levels in the diet exceeding 200-800 ppm for cattle and 115 ppm for calves are potentially toxic (Corah, 1994).

Liver biopsy provides the best indication of copper status in live cattle. Puls (1990) suggests liver copper levels of 25-100 ppm (wet weight basis) are adequate with 0.5-10 ppm being deficient and 250-800 ppm being toxic. Serum copper levels are not a good indication of copper status. All copper circulating in the blood is not available to the animal, and serum copper values can be affected by a number of factors which include dietary molybdenum and sulphate, infection, trauma, and stage of gestation (Puls, 1990). In addition, serum copper levels do not correspond to liver copper, and are not considered a reliable indicator of copper status in cattle (Clark et al., 1993). Cattle with low plasma copper levels have been found to have adequate liver copper levels (Mulryan and Mason, 1992). If serum copper values are used, a value below 0.6 ppm would indicate a potential deficiency. Given the complexity of determining copper status in cattle because of interactions with other minerals, variability of available copper in forage and bioavailability of copper in the animal; dependence on a single variable of copper status, can result in erroneous diagnosis. For a more accurate understanding of copper status, correlations must be made between copper tissue levels, forage analysis, water analysis and possibly plasma ceruloplasmin activity (Wikse et al., 1992). The most obvious clinical symptom is a change

in the appearance of the hair coat. The color may look faded and overall hair condition is rough. Other symptoms include general anemia, abnormal bone and ligament development and a reduction in growth rate.

### Zinc

Zinc is actively involved in enzyme systems through metabolism of feed constituents such as protein and carbohydrate as components of insulin. As in the case of copper, zinc is also required for maintaining responsiveness of the immune system. Zinc functions in the immune system through energy production, protein synthesis, stabilization of membranes against bacterial endotoxins, antioxidant enzyme production, and maintenance of lymphocyte replication and antibody production (Nockels, 1994). Zinc deficiency has been shown to have an important impact on immunity. Calves with a genetic disorder which interferes with zinc metabolism resulting in zinc deficiency exhibit thymus atrophy and impaired lymphocyte response to mitogen stimulation (Perryman et al., 1989). A lower percentage of lymphocytes and higher percentage of neutrophils in the blood has been observed in animals consuming a diet deficient in zinc (Droke and Spears, 1993).

Zinc has been shown to have a positive impact on immunity in stocker and feedlot cattle with limited research in beef cows. Weaned calves normally experience stress due to transportation, changes in feed and handling which increases susceptibility to infectious diseases. During this period of stress, providing adequate dietary zinc may be critical because stress has been shown to have a negative impact on zinc retention (Nockles et al., 1993). Infection can also have a detrimental effect on zinc status in cattle. Infecting cattle with a bovine rhinotracheitis challenge increased urinary zinc excretion which caused a negative balance (Orr et al., 1990). Feed intake is often depressed when feeder cattle are stressed and the reduction in intake results in a decrease of trace minerals ingested. Supplying zinc to steer calves which had undergone stress (weaning, transportation, exposure to new cattle and vaccination) was shown to increase feed intake (Spears et al., 1992). Serum zinc levels were lowest during the peak morbidity in a natural outbreak of bovine respiratory disease and steer calves challenged with infectious bovine rhinotrachetis virus had a decline in serum zinc (Hutcheson, 1989). It has been suggested that immune capacity can be substantially lowered to inadequate dietary levels of zinc before any clinical symptoms appear (Fraker, 1983). Zinc supplementation for stressed cattle enhanced recovery rate in infectious bovine rhinotracheitis virus-stressed cattle (Chirase et al., 1991). Zinc methionine has also been shown to increase antibody titer against bovine herpesvirus-1 (Spears et

### al., 1991).

Limited research has been done to determine the effects of zinc supplementation for beef cows. Dietary zinc requirement for gestating beef cows may increase during the last trimester of pregnancy. Liver and plasma zinc levels have been shown to decrease prior to parturition due to demands for deposition in fetal tissue (Xin *et al.*, 1993). Supplementing zinc to dairy cows during lactation resulted in fewer infections of mammary glands (Spain *et al.*, 1993). In grazing cows with suckling calves, zinc supplementation resulted in a 6% improvement in weight gain for calves with no change in cow weight gain (Maryland *et al.*, 1990).

Zinc status of cattle is not easily assessed. Currently there is not a good indicator for determining marginal deficiencies. Levels of 25-100 ppm (wet basis) in liver and 0.8-1.4 ppm in serum have been suggested to be adequate (Puls, 1990). Collecting blood for zinc analysis requires a specialized blood tube for mineral analysis. Analyzing hair for zinc may also be useful for long term monitoring. Clinical signs of zinc deficiency included parakeratosis, rough hair coat, joint stiffness and reduced immune response. Feedlot cattle can have reduced gain, but minimal clinical signs.

The recommended dietary level is 30-40 ppm (NRC, 1984) and zinc movement in the body is precisely regulated within this requirement range. A level of 80 ppm zinc has been suggested for stressed and sick feedlot cattle due to decreased feed intake and increased excretion (Hutcheson, 1989). Readily available stores are not available and zinc plasma levels can decrease rapidly if animals are fed a deficient diet (NRC, 1980). High dietary calcium decreases the absorption of zinc, therefore, zinc requirement in the diet increases. Other minerals (copper, phosphorus and iron) can also interact and reduce bioavailability of zinc. Excess zinc inhibits copper and iron absorption and utilization. Zinc toxicity is fairly rare in ruminants with the maximum tolerable level reported to be 500 ppm (NRC, 1984).

## Cobalt

Cobalt functions primarily as a component of vitamin  $B_{12}$  which is synthesized by rumen microorganisms. Cobalt also appears to have a role in the immune system of cattle. Neutrophils isolated from cobalt deficient calves had reduced ability to kill a pathogen (MacPherson *et al.*, 1989). Weight gain of cattle consuming a low cobalt diet was unaffected until 40-60 weeks, but the immune status (measured as neutrophil function) was reduced after 10 weeks (Paterson and MacPherson, 1991).

Cobalt serum levels are not considered to be a good indicator of cobalt status, however, liver levels reflect status better than serum values. Vitamin  $B_{12}$  concentrations in serum and liver are more reliable indicators

of cobalt status. Puls (1990) reports vitamin  $B_{12}$  values which indicate adequate cobalt levels were between 0.25-2.50 ppm (wet basis) in liver and 0.40-0.90 mg/L in serum. If hair samples are analyzed, a normal value for Co is 0.03 ppm (dry weight). Forage cobalt concentrations provide a reliable indicator of cobalt availability (Spears; 1994), but levels in forage can be influenced by cobalt level in soil and soil pH.

Clinical symptoms of cobalt deficiency include loss of appetite (an early symptom), anemia, loss of condition, weakness, rough hair coat and reduced conception rates. Animals in a deficient state respond rapidly to dietary cobalt supplementation. Appetite increases in a week, weight gain follows quickly but remission from anemia occurs more slowly. Toxicity symptoms are similar to those for deficiency. Cobalt levels are considered toxic if above 10 ppm in the diet.

#### Chromium

Chromium has been shown to play a role in the immune system. Chromium aids insulin function, increases serum immunoglobulins and retention of other trace minerals, and reduces serum cortisol. Cortisol has been found to inhibit production and actions of antibodies, lymphocyte function and leukocyte population (Munck *et al.*, 1984). Chromium supplementation in calves reduced serum cortisol and increased serum immunoglobulins (Chang and Mowatt, 1992). Effectiveness of vaccines may be improved with adequate levels of chromium by reducing cortisol levels and their inhibitory effects on the immune system.

Stressed feeder calves receiving supplemental chromium had improved weight gain and reduced morbidity the first 28 days while on a corn silage diet (Moonsie-Shageer and Mowat, 1993) suggesting that chromium may be limited in corn silage diets. Chromium supplementation reduced rectal temperature and increased blood antibody titers. Implications from this study suggested inclusion of chromium in preconditioning diets, during marketing and shipping, or receiving rations may improve performance and immunocompetence.

Dairy cows fed diets based on grass hay, haylage and corn silage were either supplemented with chelated chromium (0.5 ppm) six weeks prior to parturition and until 16 weeks of lactation, or received no supplement. A humoral response was measured and supplemented cows had an increase in antibody titer. It appeared that chromium had a significant immunomodulatory effect in cows (Burton *et al.*, 1993).

Investigating the use of chromium supplementation is fairly new and little is known about the dietary requirement of ruminants. Puls (1990) does not indicate a required dietary level, but does suggest 30-40 mg/kg zinc chromate for one month. Normal serum chromium is given as  $0.25-0.30\mu$ g/L and liver chromium as 0.04-3.8 ppm on a wet basis. Chromium interactions with other minerals appears to be positive. It has been suggested that supplemental chromium prevents stress-induced losses of copper, zinc, manganese and iron (Schrauzer *et al.*, 1986).

## **Trace Mineral Sources**

Trace minerals for supplements are available in different forms: inorganic, chelates, proteinates and complexes. Chelates, proteinates and complexes are often grouped together and are referred to as protected minerals. However, there are differences among them. Chelated minerals are associated with one to three moles of amino acids and form a closed ring structure. Proteinated minerals are associated with either amino acids or small peptides which result in an open ring structure. Complexes are minerals bonded to an organic compound. All forms of protected minerals have a neutral electrical charge while inorganic minerals possess either positive or negative charges.

The process of chelating minerals to organic compounds occurs naturally through digestive metabolic pathways. Positive or negative charges common to inorganic sources enhance the possibility of ionic binding to transport or enzymatic proteins. The problem with minerals chelated in this manner is that minerals may be bound to compounds with increased molecular size and decreased absorption. Protected minerals have a neutral charge, therefore, the minerals are not bound to large molecules and bioavailability of the minerals are increased.

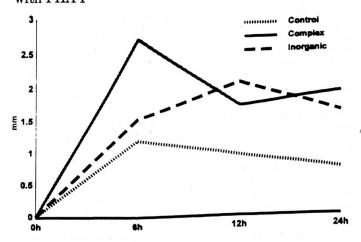
The availability of copper oxide, copper sulfate and organic copper has been compared in several studies. Source of copper (copper sulfate and copper lysine) did not affect soluble copper concentrations when using an in vitro system to estimate availability (Ward and Spears, 1993). Copper lysine appeared to be equal to copper sulfate when growth rate, feed intake, feed efficiency, plasma copper, ceruloplasmin activity and immune response of growing steers were used as indicators of utilization (Ward et al., 1993). Lactating beef cows at a commercial cow-calf operation deficient in copper had increased liver copper from either copper proteinate or copper sulfate supplements when compared to copper oxide supplement (Clark, et al., 1993). These results suggested copper availability from proteinate or sulfate forms was higher than the oxide form. Contrary to these findings, calves fed copper lysine had 53% greater apparent copper absorption and increased retention than calves fed copper sulfate during a repletion period (Nockels et al., 1993). Zinc source was also evaluated in this study and no differences between zinc methionine and zinc sulfate were found.

Zinc methionine supplementation enhanced recovery rate of IBR stressed cattle compared to supplementing zinc oxide (Chirase *et al.*, 1991). When cattle were challenged with bovine herpesvirus, antibody titer formation following vaccination was enhanced with zinc methionine compared to the basal diet without additional zinc or zinc oxide additions.

## Montana State University Research

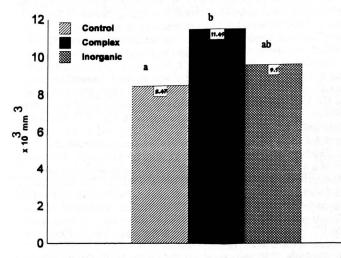
A study was recently completed at the Montana State University to determine if form of supplemental trace minerals (complexed versus inorganic) fed during the last trimester of gestation influenced cellular immunity, blood cell counts and serum copper and zinc concentrations of heifers and their calves. Pregnant cross-bred beef heifers were allotted to pasture according to expected calving date and weight. Treatments compared were control, complexed mineral and inorganic mineral fed ad libitum in mineral feeders. The control heifers received trace mineralized salt only while the inorganic treatment heifers received a supplement which contained 8818 ppm zinc, 2472 ppm copper, 4987 ppm manganese and 704 ppm cobalt in the inorganic sulfate form. The complex-treatment heifers received a supplement with zinc methionine, copper lysine, manganese methionine and cobalt glucoheptonate providing the same level of each mineral described for the inorganic supplement. Blood samples were collected 30 days following the start of supplementation to measure serum copper, zinc and complete blood cell counts in heifers, and in calves within 24 hours of birth. A skinfold test was conducted to measure a cell-mediated immune response on the same day blood samples were collected. Phytohemagglutinin (PHA-P) was injected intradermally at two locations on the neck and the swelling response was measured at 0, 6, 12 and 24 hours after infection for cows and 0, 4, 8, 12 and 24 h after injection for calves. As heifers calved they were removed from supplemental mineral-treatment groups and placed in a common pasture. The heifers were bled a second time one week after the last calf was born to determine blood cell counts. The time interval from when heifers were removed from mineral treatments until this blood collection ranged from seven to 42 days.

Heifers consuming either complex or inorganic mineral supplements had a greater swelling response than did the control-supplement heifers at 6 h post-injection. Maximum swelling appeared sooner in heifers fed the complex mineral supplement (at 6 h) with the inorganic-mineral supplement groups reaching the maximum response at 12 h (Figure 2). Swelling response in the calves was not affected by mineral treatments. These data suggest that chemical form of mineral supplement may influence the initiation of cell medicated response to pathogens. **Figure 2.** Effects of Mineral Supplementation on Heifer Skinfold Thickness 6, 12 and 24 h Post Injection with PHA-P

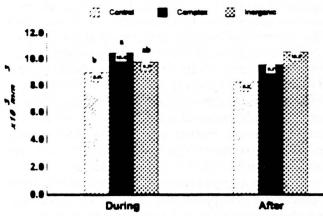


White blood cell counts (Figures 3 and 4) were highest for complex-supplemented heifers and their calves compared to those supplemented with inorganic minerals or control. Both complex and inorganic supplemented heifers had higher segmented neutrophils than controls (Figure 5). Other studies have reported an influence on blood cells from mineral supplementation. Blood samples collected after removal from supplement were similar for all cell counts. Results indicate that mineralsupplementation influenced cell counts, however, changes in cell types were dependent on continued supplementation and not on body mineral storage. Movement of minerals within the body may be regulated within the requirement range. Readily available stores of zinc are quite small as indicated by drop in plasma zinc values to the deficiency range within 24 hours after switching animals to diets very low in zinc (NRC, 1980).

**Figure 3.** Influence of Cow Mineral Supplementation on Calf White Blood Cell Counts



**Figure 4.** Influence of Mineral Supplementation on Heifer White Blood Cell Counts During Supplementation vs After Supplementation.



**Figure 5.** Influence of Mineral Supplementation on Heifer Neutrophil Counts During Supplementation vs After Supplementation

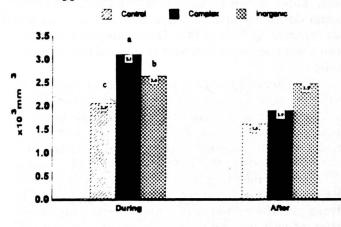


Table 2.Influence of Mineral Supplementation on<br/>Copper and Zinc Serum Levels in Heifers and<br/>Their Calves.

Item	Complex <sup>a</sup>	Inorganic <sup>b</sup>	Control
Serum copper levels, mg/L	The and the second	teats is as d	See Contractor
Heifers	.60	.61	.63
Calves	.34°	.33°	.31ª
Serum zinc levels, mg/L			
Heifers	.73	.70	.68
Calves	1.23	.88	1.00

<sup>a</sup>Complex supplement contained the following organic mineral complexes; zinc methionine (8818 ppm zn), copper lysine (2472 ppm cu), cobalt glucoheptonate (704 ppm co) and manganese methionine (4987 ppm mn).

<sup>b</sup>Inorganic supplement contained zinc, copper, cobalt and manganese in the sulfate forms and provided minerals at the same level as the complex supplement.

<sup>cd</sup>Means in the same row with different superscripts are different (P<.07).

Serum copper and zinc levels for both heifers and calves are given in Table 2. Serum copper concentrations for the calves were increased when cows were supplemented with added minerals, however; the levels measured are below those considered adequate (0.6 mg/L). Previous studies have shown that fetal calves accumulate copper in the liver and are born with lower serum copper than adults (Puls, 1990; Abdebrahman and Kincaid, 1993). During the first three days postpartum, liver copper decreases and serum copper levels increase as liver copper is converted to the copper dependent enzymes. Serum zinc was similar among supplement treatments and levels of zinc were in the normal range of 0.80-1.00 mg/L (Puls, 1990).

Serum copper and zinc values in the heifers were not influenced by supplementation. The validity of utilizing blood serum as an indication of mineral status in the heifers may be questionable. Supplemented heifers were consuming levels of copper (24 ppm) and zinc (88 ppm) which were twice NRC (1984) requirements, however, heifer serum mineral levels indicate marginal copper deficiency and adequate zinc according to values reported by Puls (1990). Liver samples may have been a more accurate indicator of mineral status in the heifers.

Supplementing 2-year old heifers in late gestation with zinc, copper, manganese and cobalt influenced cellmediated immunity in heifers and blood cell counts in heifers and their calves. Heifers receiving complexed minerals had the highest response to cell-mediated immunity, and number of white blood cell and neutrophils while heifers receiving inorganic minerals had intermediate responses. Future research will continue to investigate the influence of trace mineral supplementation of beef production with emphasis on cow-calf operations.

Minerals can have a substantial influence on herd health, production and profits for the producer. The importance of minerals dictates the need for continued research in several different areas such as evaluating mineral source to determine levels required in order to maintain immunity responses, methods to more easily assess animal mineral status, and determining optimum levels of specific minerals according to animal performance goals. The following question needs to be addressed. Is it possible to develop optimum mineral feeding strategies which maximizes production and minimizes financial cost to the producer?

#### References

Abdebrahman, M.M. and R.L. Kincaid. 1993. Deposition of copper, manganese, zinc and selenium in bovine fetal tissue at different stages of gestation. J. Dairy Sci. 76:3588 Aziz, E. and P.H. Klesius. 1986.

Effect of selenium deficiency on caprine polymorphonuclear leukocyte production of leukotriene B4 and its neutrophil chemotactic activity. Am. J. Vet. Res. 47:426. Beisel, W. 1982. Single nutrients and immunity. Am. J. Clin. Nutr. 35:417. Burton, J.L., B.A. Mallard and D.N. Mowat. Effects of supplemental chromium on immune responses of periparturient and early lactation dairy cows. J. Anim. Sci. 71:1532. Chang, X. and D.N. Mowat. 1992. Supplemental chromium for stressed and growing feeder calves. J. Anim. Sci. 70:559. Chirase, N.K., D.P. Hutcheson and G.B. Thompson. 1991. Feed intake, rectal temperature, and serum mineral concentrations of feedlot cattle fed zinc oxide or zinc methionine and challenged with infectious bovine rhinotrachetis virus. J. Anim. Sci. 69:4137. Clark, T.W., Z. Xin, Z. Du and R.W. Hemken. 1993. A field trial comparing copper sulfate, copper proteinate and copper oxide as copper sources for beef cattle. J. Dairy Sci. 76(Suppl.):334(Abstr.). Corah, L. 1993. Trace mineral requirements of grazing cattle. In: Proceedings: 28th Annual Pacific Northwest Animal Nutrition Conference. Boise, Idaho pp. 135. Droke, E.A. and J.W. Spears. 1993. In vitro and in vivo immunological measurements in growing lambs fed diets deficient, marginal or adequate in zinc. J. Nutr. Immunol. 2:71. Fletcher, M.P., M.E. Gershwin, C.L. Keen and L. Hurley. 1988. Trace element deficiencies and immune responsiveness in human and animal models. Nutrition and Immunology. R.K. Chandra, ed. Alan R. Liss, Inc., New York, NY. pp 215-239. Fraker, P.J. 1983. Zinc deficiency: A common immunodeficiency state. Sur. Immunol. Res. 2:155. Gooneratne, S.R., W. T. Cuckly and D.A. Christensen. 1989. Reveiw of copper deficiency and metabolism in ruminants. Can. J. Anim. Sci. 69:819. Hutcheson, D.P. 1989. Nutritional factors affecting the immune response in cattle: In: Proc. of the Southwest Nutrition and Management Conf., Temple, Arizona; Jones, D.G. and N.F. Suttle. 1981. Some effects of copper deficiency on leucocyte function in sheep and cattle. Res. Vet. Sci. 31:151. MacPherson, A., D. Gray, G.B.B. Mitchell and C.N. Taylor. 1987. Ostertagia infection and neutrophil function in cobalt-deficient and cobalt-supplemented cattle. Br. Vet. J. 143:348. MacPherson, A., G. Fisher and J.E. Paterson. 1989. Effect of cobalt deficiency on the immune function of ruminants. In: Trace elements in man and animals. L.S. Hurley, B. Lonnerdal, C.L. Keen and R.B. Rucker (ed.) Plenum Press, New York. pp 397. Mayland, H.F., R.C. Rosenau and A.R. Florence 1980. Grazing cow and calf responses to zinc supplementation. J. Anim. Sci. 51:966 McDowell, L.R. 1985. Copper, molybdenum and sulfur. In: McDowell LR, ed. Nutrition of grazing ruminants in warm climates. Academic Press Inc., Orlando, Florida pp237. Mossie-Shageer, S. and D.N. Mowat. Effect of level of supplemental chromium on performance, serum constituents, and immune status of stressed feeder calves. J. Anim. Sci. 71:232. Mulryan, G. and F. Mason. 1992. Assessment of liver copper status in cattle from plasma copper and plasma copper enzymes. Ann. Rech. Vet. 23:233. Munck, A., P.M. Guyre and N.J. Holbrook. 1984. Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. Endocrinol. Rev. 5:25. National Research Council. 1980. Mineral tolerance of domestic animals. National Academy of Sciences, Washington, D.C. National Research Council. 1984. Nutrient requirements of beef cattle. National Academy of Sciences, Washington, D.C. Naylor, J.M., T.R. Kasari, and B.R. Blakley. 1989. Diagnosis of copper deficiency and effects of supplementation in beef cows. Can. J. Vet. Res. 53:348. Nockels, C.F. 1994. Micronutrients and the immune response. In: Montana Nutrition Conference Proceedings., Bozeman, Montana. pp3.1. Nockels, C.F., J. DeBonis and J. Torrent. 1993. Stress induction affects copper and zinc balance in calves fed organic and inorganic copper and zinc sources. J. Anim. Sci. 71:2539. Paterson, J.E. and A. MacPherson. 1990. The influence of a low cobalt intake on the neutrophil function and severity of Ostertagia infection in cattle. British Vet. J. 146:519. Perryman, L.E., D.R. Leach, W.C. Davis, W.D. Mickelson, S.R. Heller, H.D. Ochs, J.A. Ellis and E. Brummerstedt. 1989. Lymphocyte alterations in zincdeficient calves with lethal trait A46. Vet. Immuno. Immunopath. 21:239. Puls, R. 1990. Mineral levels in animal health. Sherpa international. Clearbrook, British Columbia, Canada. Schrauzer, G.N., K.P. Shrestha, T.B. Molenaar and S. Meade. 1986. Effects of chromium supplementation on food energy utilization and the trace element composition in the liver and heart of glucose exposed young mice. *Biol. Trace Elem. Res.* 9:79. Smart, M.E., R. Cohen, and D.A. Christensen. 1986. The effects of sulphate removal from the drinking water on the plasma and liver copper and zinc concentrations of beef cows and their calves. *Can. J. Anim. Sci.* 66:669. Spain, J.N., D. Hardin, B. Steevens and J. Thorne. 1993. Effect of organic zinc supplementation on milk somatic cell count and incidence of mammary gland infections of lactating dairy cows. *J. Dairy Sci.* 76(Suppl):265(Abstr;) Spears, J.W. 1991. Advances in mineral nutrition in grazing ruminants. In *Proceedings of the Grazing Livestock Nutrition Conf.* Steamboat Springs, Colorado. pp 138. Spears, J.W., R. W. Harvey and T.T. Brown. 1991. Effects of zinc methionine and zinc oxide on performance, blood characteristics, and antibody titer response to viral vaccination in stressed feeder calves. J. Amer. Med. Assoc. 199:12:1731. Spears, J.W. 1993. Minerals in forages. In: Forage quality, evaluation and utilization, G.C. Fahey, Jr. (ed.), American Society of Agronomy, Inc. Madison, WI. pp. 281. Suttle, N.F. and D.G. Jones. 1989. Recent developments in trace element metabolism and functions: Trace elements, disease resistance and immune responsiveness in ruminants. J. Nutr. 119:1055. Wikse, S.E., Herd, D., Field, R. and Holland P. 1992. Diagnosis of copper deficiency in cattle. J. Amer. Vet. Med. Assoc. 200:11:1625. Wikse, S.E. 1992. Proceedings 1992 Texas Beef Cattle Short Course. Xin, Z., D.F. Waterman, R.W. Hemken and R.J. Harmon. 1993. Copper status and requirement during the dry period and early lactation in multiparous Holstein cows. J. Dairy Sci. 76:2711.

## Abstracts

## **Developing group practices: a management challenge**

## **Richard B. Hays, Leonie Sanderson**

Australian Veterinary Record (1995) 72, 145-147

The advantages and disadvantages of forming larger professional practices are often debated. This paper reports an exploration of the issues through three case studies involving clusters of Sydney general medical practitioners who had expressed a desire to amalgamate their solo or small group practices. Their most frequently stated goals were to reduce financial overheads, to improve the range of services offered to their patients and to improve the opportunities for recreational and study leave. Several barriers to successful amalgamation were identified, and methods of overcoming these were explored. Practices can successfully amalgamate, but only where there is a group of likeminded general practitioners who are willing to invest time to achieve mutually agreed objectives. Amalgamation will not be appropriate in all circumstances. Larger group practices should benefit from the employment of a professional practice manager. These findings may be relevant to veterinary and dental practices.

## Ultrasonographic examination of the small intestine of cows

### U. Braun, O. Marmier

Veterinary Record 136, 239-244

The small intestine of 50 cows was examined ultrasonographically with a 3.5 MHz linear transducer. The cows were examined on the right side, from the tuber coxae to the eighth intercostal space and from the transverse processes of the vertebrae to the linea alba. The appearance of loops of small intestine and their contents and motility were assessed. In the majority of cases, the intestine contained feed and the contents appeared hyperechoic, but in some cows, the intestinal lumen contained mucus or fluid which was hypoechoic. The cranial part of the duodenum could be viewed longitudinally and in cross-section; its largest diameter varied from 1.1 to 5.4 cm and it could be identified with certainty only medial to the gall bladder, which served as an acoustic window. The descending part of the duodenum was adjacent to the abdominal wall and was enveloped in the hyperechoic greater omentum, differentiating it from the jejunum and ileum. The largest diameter of the descending part of the duodenum varied from 0.9 to 3.7 cm. The ascending duodenum could not be identified because of its anatomical position. Loops of jejunum and ileum were usually viewed in cross-section and sometimes longitudinally; in contrast with the duodenum, they were constantly moving. The average diameter of the jejunum and ileum varied from 2.2 to 4.5 cm.