Beef Session V

Cow-Calf

Duane Miksch, *Presiding*

Trace Minerals: Their Role in Neonatal Diarrhea in West-Central Nebraska

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Introduction

All forms of living matter require inorganic elements or minerals for their normal life processes. Virtually all elements have been found in animal tissues and several additional ones may possess physiological significance. The functions of minerals in animal physiology are interrelated; seldom can they be considered as single elements with independent and self-sufficient roles. Ninety-one elements occur naturally on earth with unequal distribution and occurrence in nature.

It is now thought that at least 24 elements are essential to living matter. The four most abundant in the human body are hydrogen, oxygen, carbon, and nitrogen. The seven next most common, in order of abundance, are calcium, phosphorus, chlorine, potassium, sulfur, sodium, and magnesium. Iron, copper, zinc, silicon, iodine, cobalt, manganese, molybdenum, fluorine, tin, chromium, selenium, and vanadium are needed and play a role in living matter. Boron is also thought essential for some plants, and it is possible that aluminum, nickel, and germanium may turn out to be necessary.[6]

Nutritional disorders involving the mineral elements may arise as single deficiencies or excesses of particular elements, but more often as deficiencies or toxicities conditioned by the extent to which other organic or inorganic nutrients are present in the diet. These conditioning factors may be a reflection on the soils on which plants are grown, or they may be related to the presence of specific plants which are seleniferous or goitrogenic.[7]

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Gastrointestinal irritation is a common entity in trace mineral toxicosis. Diarrhea has been reported with excesses of arsenic (As), boron (B), copper (Cu), fluorine (F), iron (Fe), lead (Pb), magnesium (Mg), mercury (Hg), molybdenum (Mo), phosphorus (P), selenium (Se), sulfur (S), tin (Sn), tungsten (W), and vanadium (V).[11] The most common trace mineral deficiencies that may be associated with diarrhea are copper[15] and selenium.[13 14 17] Interactions are generally involved in calf diarrheas in west-central Nebraska, and these usually include, copper, molybdenum, sulfates, iron, selenium, and zinc.

Observations

To determine the extent of trace mineral involvement in west-central Nebraska beef calves, trace elements analyses were performed on 144 livers from perinatal calves submitted to the Veterinary Science Laboratory, North Platte, Nebraska during 1983 and 1984. All calves originated from herds in west-central Nebraska and ranged in age from eight months of gestation up to six-week-old neonates. Perinates were subjected to complete gross and microscopic, bacteriologic, virologic and toxicologic examinations. The purpose of this preliminary study was to determine if trace mineral problems existed in west-central Nebraska, and if any relationship existed with potential disease syndromes.

Calves were divided into five groups based on objective gross examination and ocular fluid evaluation: abomasal ulcers, abomasal bloat, diarrhea, excessive nitrate exposure, and weak or premature calves. Ocular fluids from all submissions were examined qualitatively for nitrate using the diphenylamine/sulfuric acid method, with quantitative NO_3 analysis by cadmium column reduction. Liver samples of approximately 50 gm were taken from the reticular impression of the liver and submitted to Michigan State

University for multi-element analyses. Concentrations of Al, As, Ca, Cd, Cr, Cu, Fe, Hg, K, Mg, Mn, Mo, Na, P, Pb, Tl, Zn, and B were determined using inductively coupled plasma-atomic emmission spectrometry (ICP-AES). Selenium concentrations were determined using spectrophotofluorometric methodology.

Elemental concentrations of 41 livers from calves with diarrhea, representing 24 different ranches are listed in Table 1. Animal age varied from 6 to 60 days. Mean concentrations for all elements fell within the expected ranges. Individually, seven elements were either above or below the suspected range. Thirty-seven variations occurred in the 41 livers. Excess storage of elements was recorded for aluminum (1), arsenic (1), copper (3), iron (8), selenium (2), and zinc (3). Deficiencies of copper (1), iron (4), sodium (7), selenium (2), and molybdenum (5) were found. Numerous low and high expected results occurred; these concentrations could cause animal health problems if interaction with other imbalanced trace minerals occurred.

TABLE 1. Liver multi-element analyses (wet weight) from calves with diarrhea in west-central Nebraska.

Element	Expected Ranges PPM*	N	Mean	Minimum Value	Maximum Value
Al	1-5	9	3.40	1.10	17.00
As	0.03-.40	1	1.30	1.30	1.30
Ca	30-100	41	63.60	40.00	91.00
Cd	0.02-1.0	—	—	—	—
Cr	——	7	0.21	0.20	0.30
Cu	35-200	41	72.20	25.00	210.00
Fe	45-300	41	206.00	36.00	1540.00
Hg	<0.01-0.06	—	—	—	—
K	1200-4000	41	2834.00	1800.00	3700.00
Mg	90-200	41	163.00	118.00	206.00
Mn	0.5-3.5	39	1.90	0.90	3.70
Mo	0.37-1.54	39	0.47	0.20	2.20
Na	1000-2000	41	1204.00	634.00	2000.00
P	2000-4400	41	3542.00	2600.00	4600.00
Pb	0.0- 1.0	—	—	—	—
Se	0.25-1.2	41	0.54	0.23	1.40
Tl	——	—	—	—	—
Zn	20-180	41	110.00	27.00	229.00
B	——	2	0.20	0.10	0.30

* Literature Cited 16, 21.

Liver concentrations indicated above normal iron content as the most common trace mineral finding in calves with diarrhea. High iron content (8) was observed on seven different ranches and ranged from 314 to 1540 ppm. Forage analyses generally indicated high molybdenum content in most feedstuffs. Ideal copper/molybdenum ratios should be 6:1 with an adequate ratio 3:1. Ratios of feedstuffs from Nebraska ranches ranged from 1.5:1 to <1:1. Blood sera from cattle on these ranches routinely indicated marginal to low serum copper concentrations.

No single element appears responsible for neonatal calf diarrhea, but the variety of fluctuations indicate that trace elements play a role in many enteric syndromes. Functions of trace elements are interrelated, and the copper/molybdenum/sulfur interaction has the greatest frequency in the Sandhills of west-central Nebraska. Iron, selenium, and zinc are also affected.

Supplementation of necessary elements in a proper protein and energy diet has improved production and lessened disease effects by reduced morbidity and mortality. A field trial of a 690 cow herd that had low serum copper concentrations showed decreased mortality and morbidity in cows supplemented with copper (Table 2). Copper deficiency was apparently caused by high iron content in feedstuffs. Supplementation was in the form of copper injection (cupric glycinate) approximately 30 days before calving. Every other cow through the chute was injected; all were maintained together.

TABLE 2. Mobidity and mortality of calves on copper field study.

	Number of Animals	Percent
Total Copper Injected Cows	345	
Calf Results		
Dead	7	2.0
One Treatment	17	4.9
Two Treatments	3	0.9
Three Treatments	3	0.9
Four Treatments	1	0.3
Total Dead and/or Sick	31	9.0
Total Untreated	314	91.0
	Number of Animals	**Percent**
Total Non-Injected Cows	345	
Calf Results		
Dead	19	5.5
One Treatment	50	14.5
Two Treatments	12	3.5
Three Treatments	6	1.7
Four Treatments	2	0.6
Total Dead and/or Sick	89	25.8
Total Untreated	256	74.2

TABLE 3. Cow hematology values.

	Normal Range*	2-28-84 CU Cows	3-01-84 CU Cows	Weaning 9-27-84 CU Cows	Weaning 9-27-84 Non CU Cows
WBC	7.5-13 k/mcl	6.9	7.8	10.5	9.4
RBC	6.5-8.5 m/ul	8.8	8.3	7.7	7.8
Hgb	11-14 gm/dl	12.8	14.4	16.3	15.3
Hct	32-38%	49.1	49.4	49.9	49.9
MCV	40-60 fl	64.0	65.0	52.7	54.6

* Literature Cited 7, 18, 20.

Hematology values were within normal ranges except for hemoglobin (Hgb) and hematocrit (Hct), which were above normal in both groups (Table 3 and 4). At weaning time, the copper treated cows had a higher WBC count than the non-

copper supplement cows. IgG and IgM evaluations on 82 cows and 61 calves at weaning revealed only one abnormality, which was low IgM in a calf from a cow not injected with copper.

Other problems attributed to trace minerals include sudden death in calves, white muscle disease, poor nursing and unthriftiness in calves, retained placentas, infertility, achromotrichia and downer cows.

TABLE 4. Calf hematology values at weaning 9-27-84.

	Normal Range*	CU Calves	Non CU Calves
WBC	7.5-13 k/mcl	12.30	12.50
RBC	6.5-8.5 m/ul	8.13	8.09
Hgb	11-14 gm/dl	14.60	15.00
Hct	32-38%	39.90	40.70
MCV	40-60 fl	41.00	41.20

* Literature Cited 7, 18, 20.

Literature Review

Copper

Copper is an essential trace element whose presence in biological systems was first reported over 150 years ago. Copper participates in the activity of over a dozen enzyme systems. Low serum copper concentrations have been related to disturbances in iron absorption, failures in erythropoiesis, mental deterioration, and death.[8]

Copper is necessary for the utilization of iron in hemoglobin synthesis, and anemia develops during a copper deficiency. Adequate dietary copper is essential for synthesis of myelin that surrounds peripheral nerves. Copper-containing enzymes in conversion of tyrosine to melanin is the probable cause of failure of normal hair pigmentation when insufficient quantities of copper are fed. Loss of crimp and tensile strength in wool also occurs because of failure to maintain normal disulfide groups that provide cross linkages within the wool fiber. Copper also is involved in bone formation, reproduction, elastin formation and in the prevention of degeneration of bovine myocardium.[1 2 7 19]

There is little placental transfer of copper; therefore, the calf is dependent on copper in colostrum. Concentrations of copper in milk drop significantly 48 hours after calving. Copper deficiency in several species has produced anatomical, chemical, and physiological abnormalities similar to those found in ischemic heart disease. A mild microcytic normochromic anemia has been reported by Canadian workers. In copper deficiency iron release from reticuloendothelial cells is improved because of a decreased activity or production of ceruloplasmin and ferroxidase II. Diarrhea associated with copper deficiency has been also related to depletion of cytochrome oxidase in the small intestine, resulting in partial villus atrophy.[17]

Chronic stress or infections can lead to an increase of plasma copper. Plasma or serum concentrations alone are not totally reliable in diagnosis of copper deficiency. Serum and liver concentrations should be done concomitantly when possible.

Molybdenum

Naturally growing herbage usually reflects soil molybdenum content. Plants growing on soils industrially contaminated with molybdenum or containing naturally high concentrations of molybdenum have contained up to 231 ppm molybdenum.[5]

Cattle have the least tolerance to molybdenum of farm animals, followed by sheep, horses, and pigs.[19] A higher turnover rate of ceruloplasmin in cattle than sheep may account for the difference in molybdenum tolerance. A wide tolerance range occurs in ruminants: 6.2 ppm for cattle compared to 1,000 ppm for mule deer. Dietary copper concentrations of 8-10 ppm protect cattle against dietary molybdenum concentrations of approximately 5-6 ppm.[11]

Clinical signs of molybdenum toxicity vary among different species. Growth retardation or loss of body weight occur in all species but diarrhea occurs only in cattle. Adult cows retain liver molybdenum concentrations when ingesting large or moderately large amounts of the element but rapidly return to normal when the source is removed. Other disorders include lameness, joint abnormalities and osteoporosis due to disturbance of phosphorus metabolism, connective tissue changes and spontanenous bone fractures, infertility in cows and lack of libido in young males.[7 11 19]

A reciprocal antagonism exists between molybdenum and copper. Molybdenum, in the presence of sulfates, limits copper retention in cattle and sheep; however, neither molybdenum nor sulfates alone affect copper retention.[7] Effects of excess molybdenum are essentially those of copper deficiency:

1) Integumental changes (rough hair coat, achromotrichia, loss of crimp in wool) related to deficiency of copper dependent enzyme tyrosinase.

2) Anemia—deficiency of copper deficient enzyme ferroxidase.

3) Skeletal or collagenous manifestations—due to deficiency of copper-dependent enzyme dopamine B hydroxylase.

4) Growth retardation and anorexia due to copper deficient enzyme cytochrome C oxidase.[7 11 12 19]

Effects of molybdenum are influenced by manganese, zinc, iron, lead, tungstate, ascorbic acid, methionine, cysteine, protein and alkalinity of soils. Most dietary forms of molybdenum except molybdenite (MoS_2) are absorbed from the gastrointestinal tract.[19] Molybate is rapidly absorbed and excreted in urine and feces. Because of this, molybdenum content is a poor indicator of dietary molybdenum status. High molybdenum content in swine diets tends to promote copper storage in liver and kidney, in contrast to an opposite effect in ruminants.[11] Peak plasma molybdenum concentrations in cattle may not occur until 7 months after initial molybdenum intake.

Molybdenum has been identified as a cofactor of several metalloenzyme systems including xanthine oxidase, aldehyde oxidase, nitrate reductase, and hydrogenase. Its occurrence in these metalloflavoproteins suggests that it is an essential micronutrient.[7] Molybdenum readily changes its oxidation state and can act as an electron transfer agent in oxidation-reduction reactions. The major biochemical role of molybdenum in animals is currently thought to be in the formation and activity of xanthine oxidase (xanthine dehydrogenase) essential for metabolic degradation of purines to uric acid.[11]

Iron

Iron toxicity is seldom a problem in domestic animals, and maximum tolerable dietary concentrations are given as 1000 ppm for cattle and 500 ppm for sheep.[12] It is difficult to determine maximum safe dietary iron content because high intakes of cobalt, copper, zinc, manganese and low intakes of nickel depress iron absorption. Availability of the form of iron in the diet is also of great importance. Concentrate and forages generally contain iron in quantities adequate to meet dietary needs.[1]

Iron is intricately involved with oxygen transport and oxygen storage in tissues, because its presence in hemoglobin and myoglobin facilitates oxygen-binding capacity. In addition, iron-containing cytochrome enzymes facilitate oxygen utilization at the cellular level. A reduction in circulating hemoglobin is one of the early indications of dietary iron insufficiency.[1]

Selenium

Selenium is a semimetal which is similar to sulfur in its chemical characteristics. It is found mainly in Cretaceous rocks, volcanic material and some sea floor deposits.

In the middle 1800's a condition was described in cavalry horses at Fort Randall, SD, which resulted in sloughing of hooves, mane and tail. A similar condition was recorded several hundred years earlier by Marco Polo during his travels through western China.[1] In recent years, selenium deficiency is recognized as a much more serious problem among grazing animals than is the toxicity of the element.[1 2 10 13 14]

Legumes are generally considered to be poor absorbers of selenium. Selenium can also be leached from soil by excessive rainfall or irrigation. Thus, cattle that pasture on fertilized soil and fertilized and irrigated winter-fed alfalfa hay should be expected to be most severely deficient in selenium.[9]

In animal diets, selenium availability for absorption is important, since no mechanism for its storage has been recognized. Animals are able to utilize both inorganic salts (selenite, selenate) and organic forms (plant, animal) of selenium. The organic form of plant origin has the best biological availability. However, inorganic selenite is the form most often used for supplementation, since it is least expensive.[10 19]

Selenium deficiency causes nutritional myopathy (white muscle disease) in young calves and yearlings. It is also implicated in a sudden death syndrome in calves less than three months old because of acute myocardial degeneration.[10]

Another syndrome in calves, seemingly unrelated to myopathy, is a selenium-responsive unthriftiness in dairy and beef calves. This syndrome varies from a subclinical growth deficit to a sudden loss of condition, and is commonly associated with a profuse diarrhea of undetermined cause.[10 13 14]

In yearlings and adult cattle, a nutritional myopathy with myoglobinuria is associated with selenium deficiency. It is more common in yearlings and is induced by stress due to transportation, bad weather, or turning out to pasture in the spring. Main clinical signs are locomotor disturbance and myoglobinuria. Also in adult cattle, two reports describe a post-parturient downer cow syndrome resembling milk fever. Affected cows were reluctant to walk, and recumbent in severe cases.[10]

Other problems possibly linked to selenium deficiency in cows include higher incidence of retained placenta, abortion, and birth of weak calves.[2 7 10 12 19] Selenium readily crosses the placenta of beef cattle. Low selenium concentrations in dams could result in sequestering of the element in the fetus, with the fetus actually attaining blood selenium concentrations greater than the mother. Colostrum can accumulate concentrations of selenium proportionate to selenium intake, but milk 7 days postpartum contains much lower amounts of selenium than does the colostrum.[10]

Selenium, through its activity as glutathione peroxidase (GSH-Px), works with vitamin E in cytosol to prevent cell membrane damage.[1 10 22] Through its metabolism of hydroperoxides, GSH-Px may be involved with synthesis of various prostaglandin derivatives, normal function of lymphocytes, neutrophils, and macrophages, and maintenance of erythrocyte integrity. Moreover, GSH-Px prevents sarcolemma damage and, therefore, muscle fiber damage.[1 3]

The most widely used assessment of selenium status is blood selenium concentrations. Low blood selenium is always found in selenium deficient conditions. Selenium is incorporated into the erythrocyte GSH-Px at the time of erythropoiesis.[17]

Needs for selenium are greatest when protein requirements are greatest. Response to supplemental selenium was greatest for cattle fed diets that provided less than optimum amounts of protein.[4]

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