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Interaction of Cattle Health/Immunity and Nutrition[1,2]

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ABSTRACT: The usual means of assessing the health of newly received beef cattle susceptible to bovine respiratory disease (BRD) are subjective, typically involving visual evaluation aided by minimal clinical measurements. Recent evidence based on the occurrence of pneumonic lung lesions at slaughter indicates a need for more accurate methods of diagnosing BRD. Inadequate passive immune transfer at birth may be an important risk factor in susceptibility to BRD, suggesting the need for management to improve passive transfer success rates. Preweaning management and vaccination practices offer opportunities for beef cattle producers to improve the immune status of newly weaned calves and decrease postweaning BRD. Feeding diets with higher levels of concentrate typically improves performance by newly weaned or received cattle, as does feeding diets supplemented with protein; however, limited data suggest that increasing concentrate and protein in receiving diets increases the rate and severity of subjectively determined BRD morbidity. Research with receiving diet concentrate/protein level relative to humoral and cell-mediated immune function coupled with indicators of health and performance is needed. Supplemental B vitamins are sometimes useful in receiving diets, but the effects have been variable, presumably reflecting differences in stress and associated feed intake responses. Vitamin E added to receiving diets to supply ≥ 400 IU/animal daily seems beneficial for increasing gain and decreasing BRD morbidity; however, further dose titration experiments are needed. Supplemental Zn, Cu, Se, and Cr can alter immune function of newly received calves, and some field trials have shown decreases in BRD morbidity rate with supplementation; however, several experiments have shown no performance or health/immune benefits from supplementation of these trace minerals. Formulation of receiving diets should take into account decreased feed intake by highly stressed, newly received beef cattle and known nutrient deficiencies, but fortification of such diets with trace minerals beyond the levels needed to compensate for these effects is difficult to justify from present data.

Key Words: Beef Cattle, Immunity, Energy, Protein, Vitamins, Minerals

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Introduction

Economic losses resulting from morbidity and mortality associated with bovine respiratory disease (**BRD**) in newly weaned or received cattle continue to plague the beef cattle industry. A survey (USDA-APHIS, 1994) of small feedlots (100 to 1,000 animals marketed annually) throughout the United States indicated that death losses ranged from 1.5 to 2.7 per 100 animals marketed, and approximately 70% of these deaths are attributed to respiratory disease. Death loss is by no means the only economic cost of BRD and is often not the most significant cost. Perino (1992) noted that BRD also affects production economics through cost of treatment and cost of lost production and(or) salvage.

Lightweight (e.g., < 200 kg), newly received cattle typically face two primary problems that contribute to a high incidence of BRD. First, stress associated with weaning and transportation has a negative effect on the immune system (Blecha et al., 1984). Second, this stress typically occurs when the animal is exposed to a variety of infectious agents as a result of marketing/transporting/management procedures. Nutrition can interact with these two primary factors, most likely as a result of preweaning nutritional deficiencies or through decreased feed intake associated with stress. Feed intake by stressed calves is low (Cole, 1996), averaging approximately 1.5% of BW during the first 2 wk after arrival of lightweight feeder cattle (Galyean and Hubbert, 1995). Low feed, and thereby

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nutrient, intake makes correction of nutritional deficiencies difficult, which could further compromise immune function (Cole, 1996) and potentially increase susceptibility to infection. For older (e.g., yearling) cattle, feed intake is typically greater than that by lightweight cattle subjected to shipping stress, although outbreaks of BRD still occur, reflecting increased exposure to infectious agents and stress. This review will focus on potential interactions of nutrition with BRD morbidity and immune function of newly weaned or received beef cattle.

Assessment of Health/Immunity

Health is a general term that describes the overall well-being and condition of an animal. In much the same way that "beauty is in the eye of the beholder," health of beef cattle is often a subjective matter, typically determined by visual observation of the animal, often coupled with various clinical measurements (e.g., rectal temperature or serum clinical profiles) to aid in confirmation of disease states and diagnosis. In the context of BRD, practical determination of morbidity involves visual assessment of an animal to evaluate signs that are frequently associated with BRD, including anorexia, depression, lethargy, nasal and(or) ocular discharge, and sometimes altered gait. Rectal temperatures exceeding 39.7°C are usually considered to confirm a diagnosis of BRD when one or more of the visual signs are present. Because of a heavy reliance on visual signs for determination of BRD, the potential for an incorrect diagnosis may be high.

Immunity refers to reactions by an animal's body to foreign substances such as microbes and various macromolecules, independent of a physiological or pathological result of the reaction (Abbas et al., 1991). Immunity is generally classified as either innate (natural) or acquired (specific). Innate immunity includes physical/chemical barriers, the complement system, phagocytes such as macrophages, neutrophils, and natural killer cells, and macrophage-derived cytokines such as α and β interferons and tumor necrosis factor (Abbas et al., 1991). Acquired immunity, which is induced by natural exposure or vaccination, includes antibodies, lymphocytes, and lymphocyte-derived cytokines such as interleukin-2, interleukin-4, and transforming growth factor-β (Abbas et al., 1991). Acquired immunity is further subdivided into either humoral or cell-mediated immunity. Humoral immunity is mediated by B-lymphocytes, which respond to antigens to become antibody-producing cells and memory cells and provide defense against extracellular microbial infections. In cell-mediated immunity, the T-lymphocytes and associated cytokines provide defense against intracellular pathogens and tumor cells. A variety of in vitro immune function tests have been developed, but it should be noted that in many beef cattle experiments related to BRD such tests are often substitution variables that provide an indication of the potential effects of nutritional treatments on immune function, but that may not relate to the clinical occurrence of BRD. Hence, caution should be exercised in attributing clinical significance to many of the commonly used immune function tests, particularly those conducted independent of clinically defined disease states.

Limited evidence suggests that current methods of BRD diagnosis and treatment may be of questionable utility. Wittum et al. (1996) evaluated BRD morbidity in 469 crossbred steers born in three consecutive calving seasons at the USDA-MARC in Clay Center, NE. Health records were maintained from birth to weaning at approximately 6 mo of age. At weaning, calves were moved to the feedlot and fed for an average of 273 d, observed daily for signs of BRD, and treated as needed. Lungs were collected at slaughter and evaluated for gross lesions indicative of active or resolved pneumonia. Thirty-five percent of the steers were treated for BRD (8% before weaning, 29% in the feedlot, and 2% treated both before weaning and in the feedlot), but 72% had pulmonary lesions indicative of BRD evident at slaughter. Pulmonary lesions were evident in 78% of treated steers and 68% of steers not treated for BRD. The authors concluded that currently used methods of treating cattle for BRD are not adequate to prevent production losses and that improved methods of diagnosis for BRD are needed. Evaluation of lung lesions at slaughter may be a useful tool for monitoring the efficacy of BRD diagnosis and treatment.

Preweaning Factors Related to Health and Immunity

"A good start in life" may be critical to the health status of cattle throughout life. Passive transfer of colostral immunoglobulins is vital to short-term health and survival of neonates, and limited data suggest that inadequate transfer occurs in 10 to 25% of newborn beef calves (Perino, 1997). The success of passive colostral transfer, as measured by serum immunoglobulin concentrations, also seems to have predictive value for long-term health outcomes, both before and after weaning. Perino (1997) reviewed factors that influence the success of passive transfer. The effect of nutritional status of the dam before birth on the success of passive transfer is unclear (Perino, 1997) and needs further research. Wittum and Perino (1995) used 263 crossbred calves from various beef and dairy breeds at the USDA-MARC to evaluate the role of passive transfer in short- and long-term health. Blood samples were collected 24 h postpartum for measurement of plasma protein and serum immunoglobulin (**Ig**)G concentrations. Health was monitored from birth to weaning and throughout a

subsequent feeding period. Preweaning mortality was associated with lower ($P < .01$) IgG at 24 h after birth, as was neonatal morbidity and preweaning morbidity. Feedlot respiratory morbidity was associated with lower serum plasma protein, but not serum IgG, at 24 h after birth. Calves with inadequate plasma proteins at 24 h after birth had a greater risk of morbidity and respiratory tract morbidity in the feedlot (odds ratios of 3 and 3.1, respectively) compared with calves that had adequate plasma protein. These results suggest that beef cattle producers must manage cows and calves to facilitate effective passive transfer. Perino (1997) reviewed various management practices for calves at risk for failure of passive transfer.

Preconditioning or similar preweaning management efforts have been used to alter the health status of calves (Hansen et al., 1992). In an extensive review of preconditioning studies with feeder calves, Cole (1985) concluded that preconditioning programs can be subdivided into three categories: 1) vaccination; 2) surgery (castration, dehorning); and 3) feeding. Feeding is often the greatest cost associated with such programs. In the controlled experiments reviewed by Cole (1985), preconditioning decreased feedlot morbidity and mortality by approximately 6 and .7 percentage units, respectively. Although Cole (1985) concluded that extensive preconditioning programs are often difficult for cow-calf producers to justify economically, preweaning interventions need not be costly and complex to provide potential benefits. For example, vaccination of calves with a chemically altered vaccine against respiratory disease viruses 4 to 6 mo before weaning on western rangelands increased the serum neutralization titer response to a modified-live respiratory vaccine administered upon arrival at the feedlot (Parker et al., 1993). Kreikemeier et al. (1997) compared Kentucky ranch calves (252 kg) assigned to three treatments: 1) vaccination with a killed viral vaccine 2 to 4 wk before weaning and revaccination with a killed vaccine at the time of commingling at a sale barn; 2) vaccination with a modified-live viral vaccine at the sale barn, but before shipment to a feedlot in western Kansas; and 3) vaccination with a modified-live vaccine on arrival at the feedlot. Calves in the two modified-live vaccine treatment groups were given a modified-live booster after 21 d in the feedlot. Morbidity rate and treatments per morbid calf were decreased from 37% and 1.14, respectively, for those vaccinated on arrival and 33% and 1.36 for those vaccinated at the sale barn to 27% and 1 for those vaccinated before weaning. Additional research in this area would aid our understanding and potentially provide additional options to beef cattle producers.

Dietary Energy Concentration/Concentrate Level

Stressed beef calves seem to have an altered eating pattern compared with their unstressed counterparts (Lofgreen, 1983). Unstressed cattle typically consume feed in quantities sufficient to maintain adequate energy intake, and dilution of the diet with lower-energy feeds or bulk would be expected to increase feed intake until physical capacity becomes limiting. This response is reversed, however, in stressed calves (Lofgreen, 1983); voluntary intake of low-energy (high-roughage) diets is less than that of high-energy ($\geq$ 60% concentrate) diets. Given a choice among feed mixtures varying in concentrate level, stressed calves selected diets with 72% concentrate during the 1st wk after arrival (Lofgreen, 1983). As a result of this altered eating pattern, performance by newly received calves is typically optimized with higher-concentrate (in excess of 60%) diets. Fluharty and Loerch (1996) used 60 individually fed steers (212 kg) in a 28-d trial to compare diets with either 30, 40, 50, or 60% corn silage (85, 80, 75, or 70% concentrate). Daily DMI increased linearly ($P < .02$) with increasing concentrate level for the 28-d period, most notably in the 3rd and 4th wk after arrival. Daily gain and gain:feed increased with increasing concentrate during wk 2 after arrival but did not differ among concentrate levels for the 28-d period. In a second experiment with 77 individually fed steers, feeding 85 vs 70% concentrate (30 vs 60% corn silage) increased DMI ($P < .01$) for the first 2 wk after arrival, but not for the 28-d trial. Pritchard and Mendez (1990) reported increased DMI during a 28-d receiving period by calves fed a high-energy (60% concentrate) receiving diet compared with a lower-energy, corn silage-based diet; however, feed:gain was superior for calves fed the lower-energy diet. Based on the results of these experiments, receiving diets with 60% or more concentrates offer the potential to increase DMI, and possibly daily gain and gain:feed, by newly received, lightweight beef cattle. Nonetheless, adequacy of feed mixing and milling facilities should be evaluated carefully in determining the optimum type of receiving diet for a given production setting. Feeding good-quality hay plus protein supplement has worked well in some cases (Cole, 1996), but weight gains are typically low with such programs, and calves may not fully compensate for lower gains during the receiving period (Lofgreen, 1988).

One possible negative aspect of higher-concentrate receiving diets is an increased morbidity rate (percentage of animals treated for BRD) and(or) severity of morbidity (days of medical treatment per calf) with increasing dietary concentrate level. Lofgreen et al. (1975) noted no effect of concentrate levels of 20, 55, and 72% (rolled barley base) on morbidity rate or severity in one trial, but there was a trend for increased rate and severity in a second trial with 55, 72, and 90% concentrate levels fed to newly received, lightweight cattle. With steam-flaked milo-based diets fed to lightweight, stressed cattle, Lofgreen (1983) reported that morbidity rate was 47, 49, and 57% and

severity was 2.5, 2.7, and 3.3 treatment days per calf with concentrate levels of 25, 50, and 75%, respectively. In contrast, Fluharty and Loerch (1996) did not report marked effects of concentrate level on morbidity of newly received steer calves fed corn silage-based diets, with no differences in morbidity rate among 70, 75, 80, and 85% concentrate receiving diets (27, 40, 33, and 20% morbidity, respectively). Differences in source of cattle, time of year, nature of diet, management, and other unknown factors likely confound the relationship between concentrate level and BRD morbidity. To date, however, no research has been done to determine the possible effects of concentrate level in receiving diets on measurements of humoral and cell-mediated immune function, nor have disease-challenge models been used to test different concentrate levels. Such research might help define the degree to which changes in rate and severity of morbidity with altered concentrate level reported in the literature truly reflect an increased susceptibility to BRD or simply reflect our inability to accurately diagnose BRD in field trials.

Dietary Protein Concentration and Source

The NRC (1984) factorial equations and the NRC (1996) metabolizable protein system can be used to calculate the quantity of protein required by beef cattle. Requirements calculated with both of these approaches are largely a function of BW and feed (net energy) intake (energy intake drives BW gain and protein deposited in gain). Because newly weaned/received calves often have a low net energy intake, they are likely to have a low capacity for protein deposition. Conversely, calves that experience less severe depressions in feed intake would likely have greater protein needs. Thus, the quantity of protein required by newly received calves during the first few days after arrival may depend heavily on feed intake. Estimates of feed intake based on historical data for various types and sources of cattle should be useful in estimating protein requirements during receiving periods.

Several experiments have been conducted to evaluate protein level and source for newly received cattle. With lightweight, market-stressed calves (approximately 184 kg) fed 50 to 60% concentrate diets that contained either 12 or 16% CP, DMI and daily gain were increased with the 16% CP diet in one trial, but not in another (Cole and Hutcheson, 1990). Morbidity from BRD was high (> 50%) in both trials and not affected by protein level. Van Koevering et al. (1991) limit-fed 72% concentrate diets (14.5% CP) that contained various protein sources that differed in ruminal degradation characteristics, including soybean meal, soybean meal:blood meal, sorghum distillers dried grain plus solubles, or sorghum distillers dried grain plus solubles:blood meal to shipping-stressed steer, bull, and heifer calves (222 kg) for 28 d. For calves that were never treated for BRD, performance was superior with supplemental soybean meal. Among morbid calves, incidence of multiple treatments (repulls) and number of sick days were less for calves fed diets that contained sorghum distillers dried grains plus solubles. Mortality tended to be lower with supplemental blood meal. Van Koevering et al. (1992) fed 72% concentrate diets to 215-kg shipping-stressed calves. Daily gains were greatest with soybean meal, but as in the previous study (Van Koevering et al., 1991), diets that contained sorghum distillers dried grains plus solubles seemed to decrease repulls and sick days.

Fluharty and Loerch (1995) conducted three trials with newly arrived cattle to assess protein needs. Diets in Trials 1 and 2 contained 55 to 60% corn silage, and the diet in Trial 3 contained 40% corn silage (DM basis). In Trial 1, newly weaned Simmental × Angus steers (243 kg) were used in a 2 × 4 factorial arrangement. Factors were diet CP concentration (12, 14, 16, or 18%) and protein source (soybean meal vs spray-dried blood meal). Daily gain increased linearly with increasing CP during the 1st wk after arrival. For the 42-d trial, blood meal diets resulted in a 7.4% increase ($P < .13$) in gain relative to soybean meal diets. Gain:feed increased linearly ($P < .01$) with increasing CP for the 1st wk and for the 42-d period, and blood meal diets improved gain:feed by 11% ($P < .01$) for the overall 42 d. Morbidity increased linearly ($P < .02$) with increasing CP (38, 50, 45, and 68% morbidity for 12, 14, 16, and 18% CP, respectively), but protein source did not influence morbidity. In Trial 2, (246-kg newly weaned Simmental × Angus steers) dietary CP concentrations were 11, 14, 17, 20, 23, or 26%, with a mixture of spray-dried blood meal and soybean meal (equal CP basis) as the protein source. No differences in DMI were noted, and maximum gain and optimum feed:gain was observed with the 20% CP diet. Morbidity did not differ among CP levels (25, 15, 20, 35, 42, and 30%, receptively). Treatments in Trial 3 (238-kg Simmental × Angus newly weaned steers) were as follows: 1) 12.5% CP control diet based on soybean meal and 2) phase-feeding of 23% CP in wk 1, 17% CP in wk 2, and 12.5% CP in wk 3 and 4, with sources of either corn gluten meal, ring-dried blood meal, spray-dried blood meal, fish mean, or soybean meal. Morbidity was low (13.9, 13.9, 8.3, 8.3, 8.3, and 19.4% morbidity for the control, ring-dried blood meal, spray-dried blood meal, fish meal, corn gluten meal, and soybean meal diets, respectively). Based on the results of all three trials, the authors concluded that increased CP concentrations are needed early in the receiving period when DMI is low.

Galyean et al. (1993; Table 1) used calves shipped from Tennessee to New Mexico (19.5 h in transit, 6.8% shrink from pay weight) to evaluate protein

level in receiving diets. Calves were assigned randomly to one of three diets with CP concentrations of 12, 14, or 16% (supplemental CP from soybean meal) for a 42-d receiving period. Daily gain increased linearly ($P < .05$) with increasing CP concentration for the overall 42-d period. Feed intake tended to increase linearly as CP concentration increased during both 21-d periods, with a linear increase ($P < .10$) for the overall 42-d period. Percentage of calves treated for symptoms of BRD was 35.8% overall, and more ($P < .03$) calves required treatment on the 16% CP (47.5%) than on the 14% CP diet (22.5%). Morbidity was intermediate for calves fed the 12% CP diet (37.5%). After the 42-d receiving period, all calves were adapted to a common 14% CP, 85% concentrate diet. Calves fed the 12% CP diet during the 42-d receiving period compensated for decreased gain during the subsequent 42-d period, such that dietary CP concentration fed during the receiving period did not affect cumulative 84-d performance (Table 1).

As with increasing concentrate level, increased morbidity rates may occur as CP level in the receiving diet increases. Such increases are particularly evident in the experiment of Galyean et al. (1993) and in one of the three trials reported by Fluharty and Loerch (1995). Moreover, Nissen et al. (1989) fed 5.2, 6.4, 7.4, or 9.5% metabolizable protein (**MP**) to newly received calves and reported a linear increase in gain and improved feed:gain ($P < .05$) with increasing MP level; however, the percentage of untreated calves decreased linearly ($P < .05$) with increasing MP, as did percentage of calves responding to infectious bovine rhinotracheitis vaccine. Serum cortisol concentrations increased linearly ($P < .05$) with increasing MP, and the authors suggested that increased cortisol with increasing MP might explain some of the changes in health responses. In contrast, McCoy et al. (1996) reported a negative correlation between the number of calves treated for BRD and MP supply in receiving diets. To evaluate the relationship between BRD morbidity and receiving diet CP level, 15 trial means

Table 1. Influence of protein concentration on performance by calves during a 42-d receiving period (Galyean et al., 1993)

	Receiving diet CP concentration, %				
Item	12	14	16	Contrast[a]	SE[b]
Receiving period performance (d 0 to 42)					
No. of calves	40	40	40	—	—
Initial BW, kg	187.1	185.7	183.0	—	1.2
42-d BW, kg	236.0	243.5	243.7	—	2.5
Daily gain, kg					
d 0 to 21	.68	.84	.89	NS	.15
d 21 to 42	1.60	1.92	2.00	L*	.10
d 0 to 42	1.14	1.38	1.45	L*	.05
Daily DMI, kg/steer					
d 0 to 21					
Hay	.61	.62	.61	NS	.02
Concentrate	2.65	2.89	3.03	NS	.15
Hay + concentrate	3.25	3.50	3.63	NS	.16
d 21 to 42	5.69	5.63	6.03	NS	.18
d 0 to 42	4.47	4.56	4.83	L†	.12
Feed:gain					
d 0 to 21	5.64	4.72	4.35	NS	.94
d 21 to 42	3.61	2.95	3.04	L*	.17
d 0 to 42	3.95	3.32	3.35	Q*	.08
Calves treated for BRD, %[c]	37.5	22.5	47.5	—	—
Mortality, no.	2	1	0	—	—
Post-receiving performance (d 43 to 84)					
Daily gain, kg	1.58	1.54	1.46	NS	.08
Daily DMI, kg/steer	6.97	7.15	7.03	NS	.21
Feed:gain	4.44	4.66	4.83	NS	.16
Overall performance (d 0 to 84)					
Daily gain, kg	1.36	1.46	1.45	NS	.05
Daily DMI, kg/steer	5.72	5.86	5.93	NS	.15
Feed:gain	4.23	4.01	4.09	NS	.09

[a]Orthogonal contrasts: L = linear, Q = quadratic effect of CP concentration. NS = not statistically significant.
[b]Standard error of means, n = four pens per treatment.
[c]Distribution differs; for 12 vs 14% CP, $P < .15$; for 14 vs 16% CP, $P < .03$.
*$P < .05$.
†$P < .10$.

from Galyean et al. (1993) and Fluharty and Loerch (1995) were pooled for regression analysis. Dietary CP level ranged from 11 to 26% in these trials, and morbidity rate ranged from 15 to 68%. Morbidity rate within trial was indexed by dividing the morbidity rate for each CP level by the trial mean for morbidity rate. Trial-indexed morbidity was then analyzed as the dependent variable by stepwise regression analysis (SAS, 1995), with linear, quadratic, and cubic effects of CP level, duration of trial, DMI, and initial BW as possible independent selections. Indexing morbidity rate eliminated all variables except CP; hence, regression analysis was conducted with trial-indexed morbidity as the dependent variable and linear, quadratic, and cubic effects of CP level as the independent variables. Results of the analysis are shown in Figure 1. The model accounted for approximately 52% of the variation in trial-indexed morbidity and indicated that BRD morbidity rates tended to increase with increasing CP level. The paradox presented by this analysis is that although morbidity rate seemed to increase with CP level, performance by calves fed higher CP levels was equal to or superior to performance of those fed lower CP levels. As discussed previously with regard to concentrate level, this paradox could reflect inaccurate diagnosis. Alternatively, it may reflect increased performance by morbid calves with higher CP levels or superior performance by healthy calves within higher-CP diets that compensated for increased morbidity. Further evaluation of the relationship between BRD morbidity and dietary CP level seems warranted, as do measurements of immune function with varying CP levels.

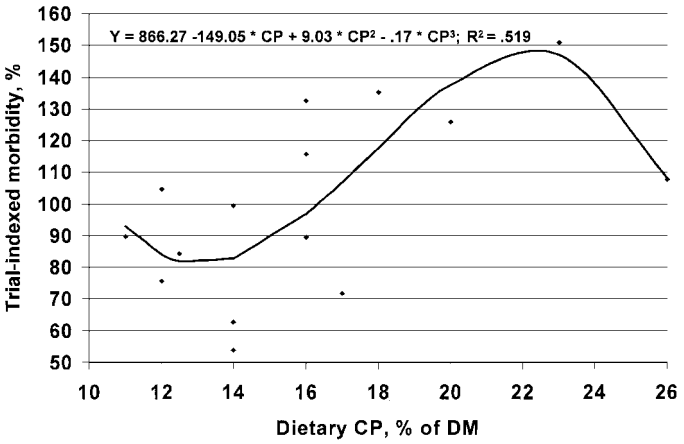

Figure 1. Relationship between CP level in receiving diets and trial-indexed morbidity rate. Data were trial means from Galyean et al. (1993) and Fluharty and Loerch (1995).

Vitamins

Experiments with B-vitamin supplementation to newly weaned or received cattle have resulted in variable responses; decreased morbidity and increased performance have been noted in some studies and little or no response in others (Cole, 1993, 1996). Cole et al. (1979) supplemented thiamin, riboflavin, pyridoxine, pantothenic acid, niacin, choline, and B_{12} in receiving diets for stressed feeder steers. Adding B vitamins decreased morbidity ($P < .05$) in calves that were weaned on the day of sale and fed hay in an order buyer barn but increased morbidity ($P < .05$) in calves that were preweaned. Feeding two different levels of these same B vitamins did not affect morbidity in a subsequent experiment (Cole et al., 1982). Zinn et al. (1987) supplemented the diets of stressed calves (116 kg) with various B vitamins and vitamin C at levels up to 10 times those required by growing pigs. Supplementation failed to alter gain and feed efficiency but decreased morbidity approximately 33% compared with unsupplemented controls. Dubeski et al. (1996b) fed 12 Hereford × Angus calves (6 to 8 mo of age; 153 kg) for 17 d after weaning at below maintenance, after which calves were deprived of feed for 3 d and given an intranasal inoculation of attenuated infectious bovine rhinotracheitis virus (**IBRV**). Injections of either saline or a B-vitamin mixture (thiamin, riboflavin, niacin, folic acid, pantothenic acid, B_6, B_{12}, and vitamin C) were given every 48 h for 14 d before and 14 d after inoculation with IBRV. Infection with IBRV decreased ($P = .001$) B_6, B_{12}, pantothenate, and vitamin C, but not folic acid, concentrations. Vitamin injections did not affect virus or interferon titers in nasal secretions, but 14 ($P = .115$) and 28 d ($P = .37$) after infection, vitamin injections tended to increase serum IgG titers to IBRV, suggesting enhanced humoral immune response with B-vitamin injections (Dubeski et al., 1996a). The authors suggested that B-vitamin status at the time of vaccination or infection may affect the success of the immune response. In a review of several experiments, Cole (1996) noted a 3% decrease in BRD morbidity, a 4.2% increase in gain, and a 5.1% increase in gain:feed with supplemental B vitamins. Variable responses to the feeding of B vitamins could, in part, be a result of differences in feed intake among experiments. Presumably, ruminal production of B vitamins would be greater in calves with high than in those with low feed intakes, potentially influencing results of supplementation.

Supplemental vitamin E in receiving diets seems to be beneficial for decreasing morbidity and improving performance. These benefits are likely to be mediated through effects of vitamin E on the immune system (Coelho, 1996). Secrist et al. (1997) pooled the results of five studies in which supplemental vitamin E was evaluated a levels of 450 to 1,600 IU/animal daily. Weighted means for daily gain tended ($P < .14$) to be increased (.92 vs .80 kg/d), with no change in

DMI, resulting in improved feed:gain (9 vs 12.4) with supplemental vitamin E. Morbidity tended to be less ($P < .14$) with vitamin E (48 vs 55%). These five trials included steers and steers plus bulls, with initial BW ranging from 242 to 272 kg. Percentage of concentrate in diets ranged from 10 to 74. Further research is needed to more accurately titrate the dose of vitamin E that provides health benefits to newly received cattle.

Because of possible injection-site reactions, injection of vitamin E with some commercial preparations may be less desirable than dietary supplements or drenches (Galyean et al., 1991). For example, Hays et al. (1987) reported that injection of 3,000 IU of vitamin E (d,l-α-tocopherol) at arrival processing of newly received steers, bull calves, and yearlings increased ($P < .07$) sick days per morbid calf, whereas feeding .9 kg of supplement that supplied 800 IU of vitamin E per animal increased daily gain ($P < .07$) and decreased morbidity and sick days per calf ($P < .07$).

Trace Minerals

Cole (1993) suggested that, with the exception of potassium, actual mineral requirements of stressed calves do not seem to be greater than those of unstressed calves. Nonetheless, concentrations of most minerals need to be increased in receiving diets to compensate for low feed intake by stressed calves. In addition, several trace minerals, most notably Zn, Cu, Cr, and Se, have been identified as possible supplemental nutrients in receiving diets because of potential effects on immune function. Recent information on each of these trace minerals will be reviewed in the following sections.

Effects of Supplemental Zn on Health and Immunity. Potentially low Zn concentrations for grazing cattle were noted by Corah et al. (1996), who surveyed forage samples from 327 cooperators in 18 states. Only 2.5% of samples were adequate in Zn content relative to NRC (1996) requirements for beef cattle, whereas 63.4% were considered deficient and 34.1% were considered marginal. Hence, Zn deficiencies may be common in practice. Readers are referred to Cole (1995), Galyean et al. (1995a), Greene (1995), and Spears (1995) for additional information on Zn supplementation practices and requirements of ruminants. Effects of Zn on immune function in nonruminants have been reviewed by Keen and Gershwin (1990), and Suttle and Jones (1989) reviewed the possible involvement of Zn in immune function of ruminants.

Zinc supplementation may be needed for stressed calves with a propensity to succumb to BRD, and source of Zn has been important in some studies but not in others. Chirase et al. (1991) reported that 3 d after an IBRV challenge, DMI was decreased 50% in steers fed a control diet with 31 mg of Zn/kg, compared with a 15% decrease in steers supplemented with 90 mg of Zn/kg from zinc methionine. Return to prechallenge DMI occurred 5 d sooner for steers fed zinc methionine than for control steers, and mean rectal temperature was lower for zinc methionine-supplemented steers than for controls on d 7 and 12. In a second experiment (Chirase et al., 1991), steers fed a control diet with 35 mg of Zn/kg had lower DMI on d 8 to 12 after an IBRV challenge than those fed a zinc methionine-supplemented diet (89 mg of Zn/kg). Similarly, on d 1 after an IBRV challenge in a third experiment, steers fed zinc methionine (170.7 mg of Zn/kg) had 65.5% greater DMI than controls (95.7 mg of Zn/kg), whereas steers supplemented with ZnO (163 mg/kg) had intermediate intakes (Chirase et al., 1991). Steers fed zinc methionine regained their pretrial DMI in 8 d, compared with 11 d for controls and 19 d for those fed supplemental ZnO.

Zinc retention became negative during stress caused by feed and water deprivation and ACTH (80 IU) injections (Nockels et al., 1993). Antibody titers against IBRV tended to be greater in steers supplemented with Zn from zinc methionine or ZnO (25 mg of supplemental Zn/kg) compared with controls (26 mg of Zn/kg), with the greatest titer response for zinc methionine (Spears et al., 1991). Zinc methionine supplemented to provide 71 mg of Zn/kg of diet decreased mean rectal temperature compared with a control diet (42 mg/kg), with intermediate responses for zinc proteinate (71 mg/kg) and $ZnSO_4$ (67 mg/kg) diets, on d 6 and 7 after an IBRV challenge in cattle (Blezinger et al., 1992).

Engle et al. (1995) fed one calf from each of five pairs of crossbred heifer calves (initial BW = 202 kg) a Zn-deficient diet (17 mg/kg), and the other calf was fed a Zn-adequate diet (40 mg/kg) diet for 28 d. Plasma Zn, feed efficiency, and phytohemagglutinin (**PHA**) skin-swelling response were less ($P < .05$) in Zn-deficient calves. In a second experiment, 208-kg crossbred heifers were fed a Zn-adequate diet for 30 d (40 mg of Zn/kg) and then allotted to either control (40 mg of Zn/kg) or Zn-deficient groups (17 mg/kg). After 21 d of depletion, the 17 mg/kg diets were supplemented with either zinc lysine, zinc methionine, or $ZnSO_4$ to bring the total Zn concentration up to 40 mg/kg. Zinc depletion decreased gain ($P < .05$) by an average of 45.6% and increased ($P < .05$) feed:gain by 97.5%. The cell-mediated skin swelling response to PHA was decreased ($P < .05$) by Zn depletion, but no differences were noted in plasma and liver Zn with Zn depletion. Differences in gain, intake, and feed:gain were not evident after 22 d of Zn repletion.

Kegley and Spears (1994) reported that supplemental zinc methionine increased ($P < .06$) in vitro unstimulated lymphocyte blastogenic response com-

pared with ZnO in lambs that were subjected to weaning and transport stress and fed a diet with 25 mg of supplemental Zn/kg. No differences in Zn source were evident in lymphocyte blastogenesis with PHA or pokeweed mitogen (**PWM**) stimulation. Lambs fed supplemental zinc methionine had smaller ($P < .08$) welt diameters than those fed supplemental ZnO at 46, 56, and 70 h after intradermal injection with PHA, but treatments did not differ before these times. Kegley et al. (1997a) used Angus crossbred heifers (176.4 kg initial BW) that were transported for 7 h and then assigned to control (no supplemental Zn), $ZnSO_4$ (360 mg of Zn/d), or Zn amino acid complex (360 mg of Zn/d) treatment groups while grazing fescue pasture (plus ad libitum access to bermudagrass hay) to evaluate performance and health responses. Serum Zn did not differ among treatments on d 28 and 56, nor did differential white blood cell counts on d 28. Cell-mediated immunity measured by an intradermal injection of PHA on d 70 was greater ($P < .07$) in heifers fed both sources of supplemental Zn than in controls 24 h after injection. In buffalo calves supplemented with 1,500 IU of dl-α-tocopherol and(or) 7 g of ZnO per animal on a weekly basis during an 18-wk trial, Mohamed et al. (1995) reported that vitamin E and(or) Zn supplementation increased the lymphocyte blastogenic response to PHA and that the interaction between vitamin E and Zn was positive.

In a field study, Galyean et al. (1995b) fed newly weaned steers four diets :1) 65% concentrate basal receiving diet supplemented with 30 mg of Zn/kg from ZnO; 2) basal + 35 mg of Zn/kg from zinc methionine; 3) basal + 70 mg of Zn/kg from $ZnSO_4$; and 4) basal + 70 mg of Zn/kg from zinc methionine. Morbidity from BRD during a 42-d receiving and subsequent concentrate adaptation period was decreased by approximately 52% (average of 22.9 vs 11.1%) for the two 70 mg/kg diets vs the basal and 35 mg/kg diets. Using newly received calves fed a prairie hay-based diet, Johnson et al. (1988) reported that supplementing 360 mg of Zn/animal daily as zinc methionine in a 70% concentrate pellet increased daily gain by 10.7%, decreased medical treatments per calf by 5.8%, and decreased morbidity (46 vs 51% for zinc methionine and control, respectively) when animals that were detected as sick during the first 3 d of the study were excluded from the analysis. When morbid calves were considered separately, there was a decrease ($P < .03$) in medical treatments per calf with zinc methionine vs control (4.45 vs 4.94). When all cattle were considered in the analysis, daily gain, feed intake, and feed efficiency did not differ between zinc methionine-supplemented calves and controls, although average number of medical treatments per calf (3.2 vs 3.7 for zinc methionine and control, respectively) was decreased with supplemental zinc methionine.

Not all experiments have noted positive effects of Zn supplementation on health and(or) immune function. With 27.6 mg of Zn/kg in a corn-cottonseed hull/isolated soy protein-based diet, supplemental Zn (25 mg/kg as zinc methionine or ZnO) did not alter the immune response either before or after lambs were subjected to stress (Gengelbach et al., 1992). Diets that were adequate (40 mg of supplemental Zn/kg), marginal (5 mg of supplemental Zn/kg), or deficient in Zn influenced the immune response of growing lambs (Droke and Spears, 1993); however, using the same dietary Zn concentrations, Droke et al. (1993) simulated stress with .2 mg of dexamethasone and reported that lymphocyte blastogenesis in response to PHA, concanavalin A (**ConA**), and PWM was not affected by treatments. Kincaid et al. (1997) used 40 Holstein heifer calves that were assigned to treatments at 6 wk of age to evaluate Zn level and source effects on growth and immune function. A calf starter was fed that provided four Zn treatments: 1) control at 60 mg of Zn/kg; 2) zinc methionine and zinc lysine added to supply 150 mg of supplemental Zn/kg; 3) zinc methionine and zinc lysine added to supply 300 mg of supplemental Zn/kg; and 4) ZnO added to supply 300 mg of supplemental Zn/kg. Actual concentrations of Zn averaged 65, 238, 400, and 340 mg/kg for the four treatments, respectively. Feed intakes and BW gains did not differ among treatments. Treatments did not affect lymphocyte blastogenesis (control, ConA, PHA, and PWM), lymphocyte interleukin-2 production, lymphocyte cytotoxicity, or phagocytic and intracellular killing ability of neutrophils, leading the authors to conclude that the Zn treatments did not affect in vitro measurements of immune response.

Several recent experiments have involved feeding Zn in combination with other trace minerals in an effort to modify performance and(or) immune response. Clark et al. (1996) allotted 60 heifers based on initial liver Cu status to three treatments (no supplement, inorganic trace minerals, and organic complexes of trace minerals). Equal dietary concentrations of Cu (24 mg/kg), Zn (64 mg/kg), Co (6 mg/kg), and Mn (49 mg/kg) were supplied by the organically complexed and inorganic (sulfate forms) treatments, and Mo (5 mg/kg), S (2,500 mg/kg), and Fe (1,000 mg/kg) were fed to all three treatment groups as antagonists. Grass hay was fed for ad libitum consumption until the heifers were moved to pasture at 60 d after calving. Differential white blood cell counts and PHA skin-swelling responses did not differ ($P > .10$) among treatments. George et al. (1997) shipped heifers (208.6 kg) that had been raised on eastern Colorado range and vaccinated only with clostridial bacterins 250 km to a research feedlot. All heifers were vaccinated for viral respiratory pathogens upon arrival. Treatments were a basal 55% concentrate receiving diet with 1) supplemental inorganic trace minerals (106 mg of Zn/kg from ZnO, 58 mg of Mn/kg from MnO, 37 mg of Cu/kg from $CuSO_4$, and 7 mg of Co/kg from $CoCO_3$); 2) the same

concentrations of elements supplied in the organic form (zinc methionine, manganese methionine, copper lysine, and cobalt glucoheptonate); or 3) organic complexes at a 3× level for the first 14 d after arrival, then switched to 1× for the remainder of the 42-d trial. Treatments did not affect gain, DMI, or feed:gain over the 42-d trial. Although calves already had a titer to parainfluenza virus-3 (**PI-3**) on arrival, the secondary PI-3 antibody titer response on d 14 and 28 after arrival vaccination was increased by organically complexed minerals ($P < .01$; ranking of 3× > 1× organic > inorganic). Skin swelling response to PHA on d 21 after arrival was greater for 3× vs inorganic at 12 ($P < .05$) and 24 h ($P < .01$) after PHA, with 3× > 1× > inorganic ($P < .01$) at 48 h after PHA. For IBR titers, 1× organic was greater than inorganic ($P < .01$) at d 14 after vaccination, but 3× did not differ from the other two treatments. At d 28, the IBR titer was greater for 1× organic ($P < .05$) than for the other two treatments. Calves fed the 3× treatment had a 17.2% decrease ($P < .05$) in the incidence of BRD compared with the other two treatments.

Angus and Simmental steer calves that were newly weaned were used in a 2 × 2 factorial design by Engle et al. (1997). Factors were 0 or .15% supplemental P and inorganic (sulfate) or inorganic + organically complexed (proteinate) trace minerals. Copper, Mn, and Zn were supplemented to a basal diet of corn silage at levels of 10, 25, and 25 mg/kg, respectively. For the organically complexed treatments, 50% of Cu and Mn and 66% of Zn were derived from proteinates, with the remainder from sulfates. Two days after weaning, calves were challenged with IBRV. Rectal temperature was not affected by treatment. Total immunoglobulin titers to porcine red blood cells were greater ($P < .05$) with no added P for Angus calves, but cell-mediated response to PHA was not affected by treatment or breed. Serum Cu, P, and Zn concentrations were not affected by treatment.

Although experiments with combinations of trace minerals provide valuable information on various commercial products, they provide little insight into the health or immune system effects of specific minerals and possible interactions among minerals. Additional research with individual trace minerals or with factorial arrangements of multiple minerals that allow testing of main effects and interactions is needed.

Effects of Copper on Health and Immunity. The concentration of Cu recommended by NRC (1996) for beef cattle is 10 mg/kg of diet, with a maximum tolerable level of 100 mg/kg; however, in practice, tolerance levels depend on interactions of Cu with other minerals (Underwood, 1977). A recent survey of forage samples in 18 states (Corah et al., 1996) indicated that more than half the samples collected were marginal to deficient in Cu. Evidence of impaired immune function in sheep with Cu deficiency induced by Mo (Suttle and Jones, 1989), coupled with potentially low Cu concentrations in forages in many areas, has led to increased interest in the role of Cu in health and immune function of beef cattle.

Serum Cu concentrations have been reported to increase with market and transit stress and after inoculation with IBRV (Orr et al., 1990; Stabel et al., 1993). Chirase et al. (1991) reported that serum Cu concentration was numerically greater on d 7 vs 0 and decreased by d 28 to concentrations lower than those on d 7 after a challenge with IBRV in beef steers supplemented with zinc methionine or ZnO. Chirase et al. (1994) reported that calves injected with 120 mg of copper glycinate 11 d before shipping had 30 and 6.9% lower DMI and BW change, respectively, after a challenge with IBRV than calves not injected with Cu. Differences in Cu retention as a result of Cu source have been noted under simulated stress in beef calves (Nockels et al., 1993).

Scottish Blackface Hill sheep, a breed that is naturally susceptible to Cu deficiency, and a genetic line selected for low plasma Cu produced lambs that were vulnerable to microbial infections when pasture management practices (liming and reseeding) lowered Cu status (Suttle and Jones, 1989). Copper supplementation was protective against infection; however, infected lambs were exceedingly Cu-deficient, even compared with contemporaries in the genetic line. In addition, pasture management changes increased pasture Mo and S contents, which could have triggered effects of thiomolybdates, with supplemental Cu simply protecting against thiomolybdates. Peripheral blood granulocytes from Cu-deficient sheep had a decreased ability to kill engulfed *Candida albicans*, and proliferative responses to antigens were lower in the hypocupremic, low-Cu-genotype lambs (PHA, ConA, and PWM antigens), with enhanced proliferative responses as a result of Cu supplementation. Suttle and Jones (1989) noted, however, that other workers have not shown lymphocyte responses in sheep depleted of Cu with tetrathiomolybdate. Boyne and Arthur (1986) evaluated the phagocytic ability of neutrophils from cattle fed adequate Cu or from Cu-deficient cattle (induced by Fe or Mo) and a feed-restricted control (80% of ad libitum). Neutrophil phagocytic activity was less ($P < .05$) for Fe and Mo groups than for the control, but it was also less for feed restriction. The Fe and Mo groups had lower ($P < .001$) phagocytic activity (*C. albicans* ingested/100 neutrophils) than either the control or feed-restriction groups.

Stabel et al. (1993) reported that Cu concentrations were decreased in liver, spleen, thymus, and lung by Cu deficiency, and they suggested that Cu-deficient animals are at a greater risk than nondeficient animals for infection. However, Stabel et al. (1993) failed to observe a consistent immune response with Holstein calves fed a semipurified diet

(1.5 mg of Cu/kg) supplemented with 0 or 10 mg of Cu/kg of diet in the form of $CuSO_4$. Saker et al. (1994) fed weaned calves (275 kg) a basal corn silage/soybean meal diet or the basal diet plus either copper lysine or $CuSO_4$. Average daily Cu intake for the three groups was 43 (basal), 97 (copper lysine), and 104 mg ($CuSO_4$). Copper lysine-supplemented calves had increased plasma Cu concentrations ($P < .05$), monocyte phagocytic activity ($P < .05$), and monocyte oxidative burst measurements ($P < .10$) compared with calves fed the basal diet. Dill et al. (1990) reported that humoral immune response did not increase with 3.25 mL of injectable ZnO suspension, Cu (2 mL of Moly-Cu), or Se (1 mL of Mu-Se/90.8 kg of BW) + Cu in steers fed diets deficient in these minerals. In a subsequent study, however, Dill et al. (1991) reported increased humoral immune response to Cu (2 mL of Moly-Cu) in steers fed diets deficient or marginal in Cu.

Arthington et al. (1995) allotted 12 Angus × Hereford heifers (183.6 kg) on the basis of initial liver Cu concentrations to either control (fed a basal diet supplemented to provide 10 mg of Cu/kg) or Mo-supplemented groups (fed the same basal diet but with sodium molybdate [Cu:Mo ratio = 1:2.5] and S [.3%] added). Diets were fed for 120 d, at which time Mo-supplemented heifers were Cu-deficient (286 vs 49 mg of Cu/kg of liver). Neutrophils were isolated before and 48 h after s.c. injection of Freund's complete adjuvant as an inflammatory stressor. Copper deficiency induced by Mo and S did not affect in vitro or in vivo measurements of neutrophil chemotaxis.

Gengelbach et al. (1997) assigned 38 2-yr-old heifers to one of four treatments during the last third of gestation. The basal diet (4.5 mg of Cu/kg) was corn silage with either 1) no supplemental Cu, Fe, or Mo; 2) 600 mg of Fe/kg; 3) 5 mg of Mo/kg; or 4) 10 mg of Cu/kg. Calves were allowed to consume their dam's diets and were weaned at an average age of 184 d. Superoxide dismutase (**SOD**) activity was less ($P < .06$) for Mo-supplemented than for control or Cu-supplemented calves at 170 d of age; SOD activity also was less ($P < .06$) for Fe-supplemented than for control calves at 170 d of age, but SOD activity in calves given Fe did not differ from that in calves given Mo or Cu. Two days after weaning, calves were given IBRV intranasally, followed in 5 d by intratracheal inoculation of *Pasteurella hemolytica*. Calves in the Fe and Cu groups had lower ($P < .06$) feed intakes and greater body temperatures following IBRV than the control and Mo-supplemented calves, which the authors interpreted as evidence of an improved immune response in the groups given Cu and Fe, perhaps mediated by cytokines. Conversely, decreased DMI and increased rectal temperature could be viewed as having a potentially negative effect on health and performance. Copper-supplemented calves had greater plasma tumor necrosis factor (**TNF**) concentrations than Mo-supplemented calves at weaning, and Cu-supplemented calves tended ($P = .11$) to have higher TNF 5 d after IRBV inoculation than calves given Mo or Fe. Plasma Cu was greater ($P < .01$) at weaning in Cu calves than in the other three groups, and ceruloplasmin absorbance was greater ($P < .01$) for Cu-supplemented calves than for the other three groups at weaning and 5 d after the IBRV challenge. On d 5 after IBRV, plasma Cu concentration was greater ($P < .05$) for Cu-supplemented calves than for other groups, least ($P < .05$) for calves given Mo, and intermediate for control and Fe-supplemented calves. The authors suggested that dietary level of Mo and Cu can alter body temperature and feed intake responses to disease by affecting TNF, and possibly other cytokines. In a follow-up study on immune function, Ward et al. (1997) used 38 bred heifers that were fed corn silage-based diets from the last third of gestation until their calves were weaned. Treatments were the same as those applied by Gengelbach et al. (1997). Secondary antibody response was greater ($P < .10$) in control calves than in calves from dams supplemented with Mo on d 7, 14, 21, and 28 after challenge with porcine red blood cells, whereas response in Cu-supplemented calves was lower on d 14 and 21, and the response in calves given Fe did not differ from that in controls. Calves from dams fed supplemental Cu had lower PHA-induced skinfold responses at all sampling times than controls, but calves given Fe or Mo did not differ from controls. In a second experiment, 18 Holstein bull calves were fed commercial milk replacer low in Cu for 49 d then were fed for 126 d a semipurified diet that contained 1.1 mg of Cu/kg, supplemented with either 5 mg of Mo/kg or 10 mg of Cu/kg. Lymphocyte viability did not differ among treatments, nor did lymphocyte blastogenic responses. Skinfold responses at 12 and 24 h after PHA were increased by Cu vs control, and at 2, 4, 6, 12, 24 h for Mo vs control ($P < .05$). The authors concluded that Cu deficiency and Cu deficiency coupled with high Mo and Fe had inconsistent effects on immune function and suggested that Cu deficiency may not affect specific immune function in calves.

Limited natural BRD challenge or field studies have been conducted with supplemental Cu. Galyean et al. (1995b) reported that supplemental copper lysine (5 mg of added Cu/kg) fed during the receiving period had a negative effect on daily gain ($P < .02$) and DMI ($P < .09$) during the subsequent growing and finishing period. Nonetheless, adding copper lysine to the receiving diet tended ($P < .17$) to decrease the percentage of morbid steers (13.9%) compared with the control diet (20.1%) that was formulated to supply 3.25 mg of supplemental Cu/kg from CuO. Brazle and Stokka (1994) observed that fewer calves required treatment for sickness between 29 to 56 d when administered a Cu-Zn-Vitamin E drench at arrival (250 mg of Cu from $CuSO_4$, 650 mg

of Zn from ZnO, and 400 IU of vitamin E). Wright et al. (1996) allotted 72 Hereford × Angus calves to four treatments: 1) basal diet (control); 2) basal + 500 IU of vitamin E + .3 mg of Se/kg; 3) basal + 10 mg of Cu/kg; and 4) basal + 10 mg of Cu/kg + 500 IU of vitamin E + .3 mg of Se/kg. Calves were separated from cows for 47 d before weaning and fed .91 kg/d of the respective diets. At weaning, calves were shipped 290 km and held overnight before a receiving period. Liver Cu was increased at weaning ($P < .05$) by the Cu + vitamin E + Se treatment. Treatments did not affect haptoglobin concentrations, and neither plasma ceruloplasmin nor lymphocyte proliferative responses differed among treatments. Weight gain during the receiving period was greater ($P < .10$) by calves given supplemental vitamin E and Se.

Effects of Selenium on Health and Immunity. Regional deficiencies of Se have been recognized for some time (NRC, 1996). Dargatz and Ross (1996) surveyed whole blood Se concentrations in cows and heifers from 253 cow-calf operations in 18 states. Overall, 7.8% of the samples were severely deficient, and 10.4% were marginally deficient. Among regions, cattle from southeastern states (AL, FL, GA, KY, MS, TN, and VA) were more likely to be deficient (18.6%) or marginal (23.8%) in Se. Corah et al. (1996) reported that 19.7% of the forage samples they surveyed from 18 states were adequate in Se content, 44.3% were deficient, 19.3% were marginal, and 16.7% were high relative to established requirements.

Reffett et al. (1988) evaluated the effects of Se deficiency on the primary and secondary humoral immune response in Holstein calves. For an 84-d period, Se-deficient calves were fed a diet with .03 mg of Se/kg, whereas Se-adequate calves were fed a diet that contained .2 mg of Se/kg. All calves were then challenged with an intranasal IBRV inoculation on d 0 and 35 of a 70-d experimental period. Serum antibody titers did not differ between treatments 14 d after the challenge but were greater in Se-adequate calves than in Se-deficient calves 14 d after the second IBRV challenge on d 35. Reffett Stabel et al. (1989) compared the immune response of newly weaned calves from dams fed either Se-deficient or Se-adequate diets, with or without an intratracheal inoculation with *P. hemolytica* 3 d after weaning and transport. Titers to *P. hemolytica* were lower ($P < .05$) in calves from Se-adequate dams than in calves from Se-deficient dams, and Se status did not affect weight gains during the study or body temperature after the challenge.

Experiments reported in the beef cattle literature often involve combined Se and vitamin E supplementation because of the interrelationship of these two nutrients. Droke and Loerch (1989) conducted five trials with steers new to the feedlot environment to evaluate one or two i.m. injections of Se and(or) vitamin E on performance, health, and antibody response to *P. hemolytica* vaccination. Although increases in serum IgG titers to *P. hemolytica* were noted with the combination of vitamin E and Se at various times after vaccination in four of the five trials, performance and health were not affected by treatments.

Wright et al. (1997) used 80 Hereford × Angus calves to evaluate Se and vitamin E combinations. The treatments were as follows: 1) basal diet only; 2) basal + .3 mg of Se/kg; 3) basal + .3 mg of Se/kg + 500 IU of vitamin E; 4) basal + .3 mg of Se/kg + 1,000 IU of vitamin E; and 5) basal + .3 mg of Se/kg + 1,500 IU of vitamin E. The basal diet was 60% rolled corn, 25% rolled oats, 10% soybean meal, and 5% molasses (as-fed basis). Calves were temporarily separated from their dams for 53 d before weaning and fed their assigned dietary treatments. All calves were vaccinated 17 d before weaning. At weaning, calves were revaccinated and shipped 418 km to a commercial feedlot. Antibody titers to IBR and BVD were not affected by treatment, and treatments did not affect serum haptoglobin concentrations. Moreover, treatments did not affect pre- or postweaning gain or transit shrink. The authors concluded that preweaning vitamin E and(or) Se supplementation did not influence postweaning performance, stress responses, or vaccination responses in beef calves with adequate vitamin E status.

Readers are referred to Suttle and Jones (1989) and Turner and Finch (1991) for additional information on the potential effects of Se on health and immune function in ruminants. In their review, Suttle and Jones (1989) concluded that there is little convincing evidence that Se deficiency affects resistance to infection in ruminants.

Effects of Chromium on Health and Immunity. Although no Cr requirement has been established for beef cattle, NRC (1996) suggested that results of recent experiments indicate supplemental Cr may be needed in some situations. Interest in the potential health or immune system effects of Cr in beef cattle was stimulated by the report by Chang and Mowat (1992) that gain by feeder calves during a 28-d receiving period was increased by feeding .4 mg of supplemental Cr/kg from high-Cr yeast when calves were not medicated with long-acting oxytetracycline (**LAOTC**) on arrival. In that experiment, however, gain was not affected by Cr when calves were medicated with LAOTC on arrival, the LAOTC × Cr interaction was not significant, and supplemental Cr had no effect on morbidity. Moonsie-Shageer and Mowat (1993) followed this initial study with additional research in which either 0, .2, .5, or 1 mg of Cr/kg from a high-Cr yeast product was supplemented to corn silage-based receiving diets for stressed feeder calves. Daily gain during a 30-d receiving period was increased ($P < .05$) for the .2 and 1 mg/kg levels vs unsupplemented controls, and morbidity was

decreased ($P < .05$) from 52.4% for controls to 14.3% for the .2 mg of Cr/kg level; however, morbidity did not differ from controls for the .5 and 1 mg/kg levels.

Results of subsequent research on the effects of Cr supplementation on health and(or) immune function of cattle have been variable. Lindell et al. (1994) conducted two experiments to evaluate either 0 or 4 mg/animal daily of Cr (yeast form) and revaccination (with or without a modified-live viral vaccine) on performance and health of British crossbred calves. In the first experiment with 224-kg calves, supplemental Cr did not affect performance but decreased morbidity ($P = .04$) from 34.4 to 21.6%. In the second experiment, supplemental Cr did not affect the ability of calves to withstand an intranasal IBR challenge. Ward et al. (1995) used 32 Suffolk lambs (31 kg) in a 2×2 factorial arrangement. Factors were 80 or 100% of CP requirements, with or without .4 mg of Cr/kg as chromium tripicolinate. In lambs fed adequate CP, the lymphocyte blastogenic response to PHA was increased when Cr was fed, but not in lambs fed inadequate CP (interaction, $P < .01$). In contrast to PHA, PWM decreased the blastogenic response by 15% in lambs fed Cr ($P < .01$), indicating the sensitivity of in vitro blastogenic assays to the mitogen(s) used.

Kegley and Spears (1995) assigned Angus and Angus crossbred steers (215 kg initial BW) obtained from feeder calf sales to four treatments: control; .4 mg of supplemental Cr/kg from $CrCl_3$; high-Cr yeast; and Cr-nicotinic acid complex in corn silage-based diets. Steers consumed diets ad libitum for 56 d, with no effects of treatments on performance. On d 52, PHA skinfold response was greater in the group given high-Cr yeast than in other three groups ($P < .10$) for 8 h after PHA injection. Lymphocyte blastogenic response to PHA was greater ($P < .05$) in steers supplemented with Cr-nicotinic acid than in steers given $CrCl_3$. Clearance of an i.v. infusion of glucose tended ($P < .11$) to be faster from 15 to 45 min after infusion in the steers fed the Cr-nicotinic acid diet. The increase in insulin in response to glucose infusion was greater 15 and 30 min after infusion in steers fed supplemental Cr-nicotinic acid than in those fed the other two Cr sources, and insulin in controls was lower than that in steers fed Cr-nicotinic acid at 30 min. The authors concluded that Cr-nicotinic acid and high-Cr yeast may affect immune response and that Cr-nicotinic acid affects insulin-related functions. Kegley et al. (1997b) fed 48 Angus crossbred steers (initial BW = 263 kg) 90% corn silage diets with either control or .4 mg of supplemental Cr/kg (as a Cr-nicotinic acid complex). After 56 d on treatments, half the steers in each treatment were transported 343 km and unloaded in an unfamiliar location, then returned to the feedlot (50 km) the following day. Feed was withheld during the 2 d of shipping and unloading. On return, all steers were inoculated with IBRV intranasally. The DMI (d 57 to 80) of shipped steers was less ($P < .07$) than that of unshipped steers after IRBV inoculation. Serum total IgG was decreased ($P < .10$) by Cr before and after shipping, but Cr did not affect serum glucose concentrations. Supplemental Cr did not affect rectal temperature after the IBRV challenge or the antibody response to either IBRV or porcine red blood cells; however, shipping decreased the IgG response to porcine red blood cells. The authors concluded that supplemental Cr did not affect the immune responses measured.

Holstein bull calves 6 to 8 wk of age (average BW = 84 kg) were fed diets supplemented with either no Cr or 3 mg/d of Cr from a high-Cr yeast product for 53 d (Arthington et al., 1997). After d 53, jugular blood samples were collected every 4 h for 24 h, and each calf was given an intranasal inoculation of IBRV, followed by collection of blood samples every 4 h for the next 6 d. Rectal temperatures were increased ($P < .05$) for 5 d after inoculation, but Cr did not affect the response. Treatment with Cr did not affect ACTH, cortisol, or plasma tumor necrosis factor-α. No differences were noted in daily excretion of Cu and Zn in the urine as a result of treatment, or in lymphocyte proliferative responses to mitogens or neutrophil bactericidal function. Ceruloplasmin and fibrinogen were not affected by treatment or viral inoculation.

Implications

Bovine respiratory disease negatively affects beef cattle performance and cattle feeding economics. Because current diagnostic methods are subjective and potentially inaccurate, improved methods of diagnosis under field conditions are needed. Additional research is needed on the effects of concentrate/protein level on immune function of beef cattle. The most appropriate conditions for supplementation of B vitamins and the dose titration response with vitamin E for receiving diets need to be defined. Supplemental zinc, copper, selenium, and chromium have altered immune function measurements and decreased respiratory disease morbidity under field conditions in some cases, but the results have been inconsistent. Nutritionists should formulate diets for newly received, stressed beef cattle to compensate for decreased feed intake and known nutrient deficiencies. However, nutrient fortification beyond compensation for these factors, especially with trace minerals, should be considered carefully.

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