Role of Mineral and Vitamin Status on Health of Cows and Calves

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■ Take Home Messages

- ‣ Maximizing the immune response of dairy cows during the transition period is critical to minimizing health problems in early lactation
- ‣ A deficiency of vitamin E, vitamin A, selenium, copper, or zinc will impair the immune response
- ‣ Low vitamin E and/or selenium status can increase incidence of retained placenta and mastitis
- ‣ Biotin supplementation may be beneficial in dairy herds with hoof health problems
- ‣ Supplementing minerals and vitamins at levels well above dietary requirements does not improve health, and may cause toxicity in the case of copper and selenium

■ Introduction

Deficiencies of many minerals and vitamins required by cattle can result in metabolic disorders that impair animal health. Nutrient deficiencies during pregnancy in cows often result in metabolic disorders and increased mortality in their calves. A number of trace minerals and vitamins are involved in the immune system, and impaired immunity can increase the susceptibility of cattle to infectious diseases. The transition period, from 3 weeks before to 3 weeks after parturition, is a stressful time for dairy cows. During the transition period the immune response is suppressed and cows exhibit greater susceptibility to a number of diseases. Assuring adequate trace mineral and vitamin status is critical for optimizing the immune response during the transition period. This paper will discuss the role of minerals and vitamins on health of dairy cattle.

■ Vitamin E

Vitamin E is an important lipid soluble antioxidant in the body. A deficiency of vitamin E in young calves results in white muscle disease. Low vitamin E status in cows will reduce the immune response and likely increase the incidence of certain diseases, especially mastitis. Fresh green forages are excellent sources of vitamin E, usually containing 80-200 IU of vitamin E/kg DM. However, concentrates and stored forages (hays, haylages, and silages) are generally low in vitamin E (NRC, 2001). In cows consuming harvested forages, the most recent Dairy Cattle NRC recommends that dry cows receive 1.6 IU of supplemental vitamin E/kg of body weight (approximately 80 IU/kg of DM intake) the last 60 days of gestation. In lactating dairy cows the recommendation is 0.8 IU of supplemental vitamin E/kg of body weight (approximately 20 IU/kg DM intake). On a per cow per day basis these levels correspond to roughly 1,000 and 500 IU of supplemental vitamin E for dry and lactating cows, respectively.

Plasma concentrations of alpha tocopherol or vitamin E decrease dramatically in dairy cows fed stored forages in late gestation and early lactation (Golf and Stabel, 1990). The lowest plasma alpha tocopherol concentrations are generally observed between 1 week prepartum and 2 weeks postpartum. Plasma vitamin E concentrations will drop to levels that reduce the immune response around parturition if vitamin E status is inadequate. Research has indicated that a plasma alpha tocopherol concentration of approximately 3 µg/ml is needed to maximize health and immune response in dairy cows (Weiss et al., 1997).

It is well known that the functions of vitamin E and selenium are related and that responses in animal health to supplementation of either nutrient can depend on the animal's status of the other nutrient. Supplementing vitamin E during the dry period has reduced the incidence of mastitis in cows fed diets low or marginal in selenium in some studies. Supplementing 740 IU of vitamin E/cow/day throughout the dry period reduced incidence of clinical mastitis by 37% and duration of mammary infections by 44% in cows fed a diet low in selenium (0.06 mg/kg DM) and vitamin E (Smith et al., 1984). Weiss et al. (1997) found that supplementing 4,000 IU of vitamin E/day for 14 days prepartum reduced new intramammary gland infections and incidence of clinical mastitis in early lactation by 63% and 89%, respectively, compared to control cows supplemented with 100 IU of vitamin E/day. Supplementation of cows in this study with 1,000 IU of vitamin E/day was less effective (30% vs. 89% reduction) in preventing mastitis than 4,000 IU/day (Weiss et al., 1997). In this study all cows were supplemented with 0.10 mg of selenium/kg diet and the dry cow diet analyzed 0.14 mg of selenium/kg DM. This level of selenium would be considered low or marginal relative to requirements. In Canada, Batra et al. (1992) found that supplementing 1,000 IU of vitamin

E/day did not affect incidence of clinical mastitis in dairy cow fed diets low in selenium (0.10 – 0.14 mg/kg DM).

Responses to supplementing high levels of vitamin E during the transition period in regard to mammary gland health have been highly variable. Injecting or feeding high levels of vitamin E have not reduced incidence of mastitis in many studies where cows appeared to be receiving adequate selenium. LaBlane et al. (2002) injected cows with 0 or 3,000 IU of vitamin E at one week prior to calving on 21 commercial dairy herds in Canada. Vitamin E injections increased plasma alpha tocopherol concentrations at 7 and 14 days after injection but did not affect incidence of clinical mastitis. Similar results were obtained on commercial dairy farms in England when cows were injected with 0 or 2,100 mg of vitamin E at 2 weeks prepartum and at calving (Bourne et al., 2008).

Recent studies in Sweden (Persson Waller et al., 2007) and the Netherlands (Bouwstra et al., 2010) evaluated supplementing high levels of vitamin E on commercial dairy herds with a history of a high incidence of mastitis. Persson Waller et al. (2007) supplemented cows with an additional 2,400 IU of vitamin E/day from 4 weeks prepartum until 2 weeks postpartum. Vitamin E supplementation for control cows in this study averaged 386 IU/day at 1 month prepartum and 664 IU/day at calving. Supplementing additional vitamin E did not affect incidence of clinical mastitis or somatic cell count. However, vitamin E supplementation at the high level lowered the risk of stillborn calves or calf deaths within 24 hours of birth.

It has generally been assumed that supplementation with high levels of vitamin E will either have a positive effect or no effect on mammary gland health. However, a recent study (Bouwstra et al., 2010) conducted on commercial dairy farms in the Netherlands indicated that supplementing dry cows with high levels of vitamin E can have an adverse effect on incidence of mastitis. In this study cows on 5 commercial farms were divided into two groups, and supplemented with either 125 or 3,000 IU of vitamin E/day during the dry period. Cows on all farms were fed wilted grass silage and limited corn silage during the dry period. Incidence of clinical and subclinical mastitis during the first 100 days in milk was higher in cows supplemented with high vitamin E. Cows in this study had relatively high plasma concentrations of vitamin E at dry off and this may explain the adverse effects of high vitamin E on incidence of mastitis (Bouwstra et al., 2010). In this study serum vitamin E concentrations were 6.6 µg/ml at dry-off and 4.3 µg/ml at 2 weeks prepartum. In the Ohio study where vitamin E supplementation reduced clinical mastitis, plasma vitamin E concentrations in control cows (fed 100 IU vitamin E/day) were 3.1 µg/ml at 60 days prepartum and 2.6 µg/ml at 2 weeks prepartum (Weiss et al., 1997).

Incidence of retained placenta also has been reduced by oral (Miller et al., 1993) and intramuscular injections of vitamin E (Bourne et al., 2008). Bourne et al. (2008) found that injecting 2,100 mg of vitamin E at 2 weeks prepartum and at calving reduced the incidence of retained placenta from 6.5 to 3%. Erskine et al. (1997) reported that injection of 3,000 IU of vitamin E prepartum reduced the incidence of metritis as well as retained placenta. Similarly to mastitis, initial vitamin E status determines whether incidence of retained placenta is reduced by vitamin E administration. LeBlanc et al. (2002) reported that injection of vitamin E 1 week before calving reduced the incidence of retained placenta in cows with marginal pre-treatment serum vitamin E concentration but not in cows with adequate serum vitamin E concentrations.

■ Selenium

The selenium requirement of dairy cattle is approximately 0.3 mg/kg DM (NRC, 2001). Although the requirement for selenium is low, feedstuffs produced in many areas are deficient in selenium. Selenium functions in the antioxidant system as a component of the enzyme glutathione peroxidase.

White muscle disease in calves is a common clinical sign of selenium deficiency that results in degeneration and necrosis in both skeletal and cardiac muscle. Affected animals may show stiffness, lameness, or even cardiac failure. Unthriftiness, weight loss, and diarrhea are other signs of selenium deficiency that can be seen in young calves. Selenium deficiency in calves is caused by their dam receiving inadequate selenium during gestation.

It is well documented that selenium deficiency reduces the immune response. Selenium deficiency in dairy cows reduces the ability of blood (Hogan et al, 1990) and milk (Grasso et al., 1990) neutrophils to kill bacteria. Blood concentrations of selenium in dairy herds have been related to mammary gland health (Erskine et al., 1987; Weiss et al., 1990). Low blood selenium concentrations were related to greater prevalence of intramammary infection in a study involving 32 dairy herds in Pennsylvania (Erskine et al., 1987). In a study involving 9 commercial dairy herds in Ohio, herds with high serum selenium concentrations had reduced rates of mastitis and lower bulk tank somatic cell counts (Weiss et al., 1990).

Intramuscular injection of 0.1 mg selenium/kg of body weight at 21 days prepartum did not affect the incidence of clinical mastitis but reduced the duration of clinical symptoms in cows with clinical mastitis by 46% (Smith et al., 1984). Cows in this study received diets low in selenium and vitamin E. Selenium administration in combination with oral supplementation of 740 IU of vitamin E/day in this study was more effective than selenium or vitamin E

alone, reducing incidence of mastitis by 37% and duration of clinical symptoms by 62% (Smith et al., 1984). Selenium supplementation to diets deficient in selenium reduced the rate of new intramammary gland infections in dairy cows (Malbe et al., 1995). A number of studies have indicated that prepartum selenium supplementation can reduce the incidence of retained placenta in dairy cows fed diets low in selenium (Julien et al., 1976).

Supplementing selenium to diets adequate in selenium does not result in additional improvements in health (Schingoethe et al., 1982). Immune response is also not enhanced by supplementing high levels of selenium (Ellis et al., 1997). Selenium toxicity may also be a problem when selenium is supplemented at levels well above requirements.

High levels of sulfur in the diet reduce absorption of selenium and may increase selenium requirements (Ivancic and Weiss, 2001). Selenium naturally present in legume hay appears to be less available than selenium in grass hay or concentrates (Spears, 2003). Selenium is usually supplemented to diets as sodium selenite or selenized yeast. Selenomethionine is the predominant form of selenium that occurs naturally in feedstuffs and in high selenium yeast. Selenomethionine and selenized yeast are considerably more bioavailable than selenite when fed to selenium-deficient cattle (Pehrson et al., 1989). Supplemental selenium from selenized yeast results in higher plasma and milk selenium concentrations than a similar level of selenium from selenite (Ortman and Pehrson, 1999).

■ Vitamin A and β-Carotene

Vitamin A deficient animals are more susceptible to bacterial, viral, and parasitic infections than animals with sufficient vitamin A (Chew, 1987). A deficiency of vitamin A can cause abortions and increased incidence of retained placenta in cows, and increased morbidity and mortality in calves. The dairy cattle NRC recommends 75,000 IU of vitamin A/day for lactating cows and 80,000 IU/day for dry cows. The current recommended levels of vitamin A appear to be adequate for maximizing health in dairy cattle. Chew and Johnson (1985) reported that increasing the intake of vitamin A from 53,000 (66% of current recommendation) to 173,000 IU/day in dairy cows starting at 30 days prepartum decreased somatic cell counts in milk during early lactation. In contrast, Oldham et al. (1991) found that increasing vitamin A from 50,000 to 170,000 IU/day did not affect somatic cell count or mammary gland health in cows. Compared to cows receiving no supplemental vitamin A, cows supplemented with 120,000 IU of vitamin A/day for 4 weeks prepartum had reduced incidence of retained placenta. Young calves fed milk replacer with high vitamin A concentrations (87,000 IU/kg DM) had improved fecal consistency compared with calves fed milk replacer low (7,000 IU/kg DM) in vitamin A (Eicher et al., 1994).

Beta-carotene is the major precursor of vitamin A that occurs naturally in feedstuffs. Fresh green forage is an excellent source of β-carotene. Much of the β-carotene in silages and hays is loss during harvest and storage. Cereal grains and grain-byproducts are extremely low in β-carotene. In addition to providing vitamin A, β-carotene can also serve as an antioxidant while vitamin A is not an important antioxidant. Some studies have indicated that β-carotene supplementation at 300 mg/day to dry cows may improve mammary gland health (Chew, 1993) and reduce the incidence of retained placenta and metritis (Michal et al., 1994). In these studies vitamin A was only supplemented at 66% of the current recommendation for dry cows. It is unclear if supplementing β-carotene will improve health of dry cows receiving adequate vitamin A.

■ Zinc

Severe zinc deficiency greatly impairs immune responses and reduces disease resistance (Spears and Weiss, 2008). In dairy cattle severe zinc deficiency would be very rare. A marginal deficiency of zinc would be more likely in dairy cows. Lactating dairy cows require 45 to 65 mg zinc/kg of DM (NRC, 2001). Increasing dietary zinc from 40 to 65 mg/kg DM reduced somatic cell counts and milk amyloid A concentrations in dairy cows (Cope et al., 2009). Milk amyloid A is an acute phase protein that increases in response to inflammation.

Some research suggests that organic forms of zinc may affect mammary gland health status. Supplementation of dairy diets with zinc methionine has reduced somatic cell counts in some studies but not in others (Kellogg, 1990). Spain (1993) found that lactating cows supplemented with zinc proteinate had a lower rate of new intramammary infections than those supplemented with inorganic zinc oxide. He suggested that zinc proteinate may enhance resistance to mammary infections by increasing keratin synthesis in the teat canal.

■ Copper

Copper requirements of cattle can vary from less than 10 to 20 mg of copper/kg DM, depending on the level of other minerals present in the diet. Relatively low concentrations of molybdenum (5 mg/kg DM or higher) in combination with normal levels of sulfur (0.2 – 0.25%) greatly reduces the bioavailability of copper. Elevated sulfur (greater than 0.30%) in the diet will reduce bioavailability of copper even if molybdenum levels are low. High concentrations of iron (250 mg/kg DM or higher) will also reduce bioavailability of copper. Copper can be supplemented to cattle diets as

copper sulfate, basic copper chloride or various organic copper sources. Copper oxide is very poorly absorbed and should not be used in mineral supplements.

Depigmentation or bleaching of hair is usually the earliest visual sign of copper deficiency. Loss of hair pigmentation in copper-deficient cattle is associated with a rough hair coat. Copper deficiency can also result in fragile bones, anemia, sudden death due to heart failure, and reduced immune response. The ability of neutrophils to kill microorganisms is reduced during copper deficiency (Spears and Weiss, 2008).

Heifers fed diets marginal in copper (6 – 7 mg/kg DM) had a greater percentage (60% vs. 36%) of infected quarters at calving than heifers supplemented with 20 mg of copper/kg DM (Harmon, 1998). In a separate study, heifers were fed a control diet (6 – 7 mg copper/kg DM) or the control diet supplemented with 10 mg copper/kg DM from either copper sulfate or copper proteinate for 120 days prior to calving (Harmon, 1998). Heifers supplemented with copper proteinate had a greater proportion of uninfected quarters at calving than heifers fed the control or copper sulfate supplemented diet. Scaletti et al. (2003) evaluated the effect of dietary copper on responses of heifers to an intramammary *E. coli* challenge at 34 days of lactation. Heifers were fed a control diet low in copper (6.5 mg/kg) or the control diet supplemented with 20 mg copper/kg DM from 60 days prepartum through 42 days of lactation. Following the *E. coli* challenge, heifers supplemented with copper had lower *E. coli* numbers and somatic cell counts in milk, lower clinical scores, and lower peak rectal temperatures than control heifers. Although the severity of *E. coli* infection was decreased by supplemental copper, the duration of infection was not affected by copper (Scaletti et al., 2003).

Supplementing copper well above animal requirements results in accumulation of copper in the liver, and may lead to death due to copper toxicity. Total mixed rations containing as little as 30 to 40 mg of copper/kg DM may cause toxicity in adult cattle if fed for a prolonged period of time (NRC, 2005). Young calves are more susceptible to copper toxicity because copper absorption is much higher in calves prior to development of the rumen.

■ Biotin

B-vitamins are synthesized by rumen microorganisms. It has generally been assumed that B-vitamins do not need to be supplemented to dairy diets. However, a number of recent long-term studies indicate that biotin supplementation can reduce the prevalence of hoof lesions and lameness in dairy cows (Weiss and Ferreira, 2006).   Biotin has usually been

supplemented at 20 mg/cow/day, and several months of biotin supplementation is needed before hoof health improvements are observed.

■ Conclusion

A number of minerals and vitamins can affect immune function and health in cattle if they are present in the diet in inadequate amounts. It is well documented that low vitamin E and/or selenium status can increase incidence of mastitis and retained placenta. A deficiency of copper may affect mammary gland health. Copper status in cattle is affected not only by the level of copper in the diet but also by high levels of other minerals, such as sulfur, molybdenum, and iron that reduce the bioavailability of copper. In herds with a history of lameness and hoof lesions, biotin supplementation may improve hoof health. Supplementing dairy cattle with minerals and vitamins at levels well above requirements will not result in further improvements in health and may produce adverse effects on animal health.

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