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THE ROLE OF ANTIOXIDANT NUTRIENTS IN RELATION TO BOVINE MASTITIS AND RETAINED FETAL MEMBRANES: A REVIEW

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ABSTRACT

Antioxidants play an important role in the health and production of cattle. Antioxidants deficiency has direct or indirect negative effects on growth, production and health of cattle. Some antioxidants (selenium, Vitamin E, Zinc, etc) when excessive in supplementation can lead to toxicity. Supplemental levels of vitamin E higher than recommended for dairy cattle have been beneficial in the control of mastitis and retained fetal membranes. The availability of antioxidants in sufficient quantities in the diet ensures the proper functioning of the immune and reproductive systems.

Keywords: antioxidants, cow, mastitis, retained fetal membranes

INTRODUCTION

Oxidative stress occurs when the production of reactive oxygen metabolites (ROM) exceeds the capacity of the antioxidant system of the cell, tissue, or body. Certain nutrients act as antioxidants or are components of antioxidant enzymes and have a direct effect on oxidative stress. The prevalence and severity of several important health disorders in dairy cows including retained fetal membranes, udder edema, and mastitis appear to be related to oxidative stress. In addition, antioxidant nutrients can affect milk quality. Milk quality is usually defined in terms of mastitis. Milk with a low somatic cell count (SCC) and visibly normal appearance (no clots) is considered high quality. Selenium deficiency favours the appearance of perinatal metritis and retention of placenta in dairy cattle (Sordillo, 2013).

Antioxidants and Mastitis

Mastitis is still an extremely prevalent and expensive problem for dairy farmers. On well-managed farms, approximately 50 cases of clinical mastitis can be expected per 100 cow-years (assuming 305 d lactation) Dahlquist and Chew (1985). Studies examining the effects of vitamin A and B-carotene on mastitis measures have been inconsistent. Dahlquist and Chew, 1985; Chew and Johnston, 1985 reported positive effects when cows were on diets that approximately met NRC (2001) requirements for vitamin A and supplemented with 300 mg/d of B-carotene, and one study reported no effect (Oldham *et al.*, 1991). Supplementation occurred during the dry period and early lactation. A likely reason for the difference between studies was differences in vitamin A and B-carotene status of the control cows. Control cows in the study that found no effect had high concentrations of plasma B-carotene (10 mg/L) whereas in one of the studies that reported an effect, plasma B-carotene was 2.5 mg/L. Jukola *et al.* (1996) suggested that plasma concentrations of B-carotene in dairy cows should be >3 mg/L to optimize udder health. LeBlanc *et al.* (2004), however, reported no statistical association between serum B-carotene concentrations and clinical mastitis during the first 30 DIM even though average B-carotene concentrations were below 3 mg/L.

Supplemental vitamin E and/or Se has been shown to reduce prevalence and severity of mastitis and reduce SCC (Malbe *et al.*, 1995; Smith *et al.*, 1984; Weiss *et al.*, 1997; Wichtel *et al.*, 1994; Achi, 2019). In general, supplementation rates were 0.3 ppm Se and 500 IU/d of supplemental vitamin E during lactation and 1000 IU/d during the dry period. Erskine *et al.* (1989) reported that feeding cows 2 mg of supplemental Se/day starting 3 months before calving and throughout lactation reduced the severity and duration of mastitis caused by

experimentally challenging cows with *Escherichia coli*. Control cows in that study were fed a diet with 0.04 ppm Se. In a similar study, Erskine *et al.* (1990) found no effects of supplemental Se on mastitis when cows were experimentally challenged with *Staphylococcus aureus*. Weiss *et al.* (1997) reported that feeding 4000 IU of supplemental vitamin E/day during the last 14 days of the dry period reduced clinical mastitis and new infections at calving compared with cows fed 1000 IU/d.

Diets with 20 ppm supplemental copper have been shown to reduce the severity of mastitis following an *E. coli* challenge compared to diets with 7 ppm (Scaletti *et al.*, 2000). Heifers that received no supplemental copper after weaning and then were fed a diet with no supplemental copper from 84 d prepartum to 108 d postpartum had more infected quarters during lactation than did animals fed 20 ppm supplemental copper from 84 d pre to 107 d postpartum (Harmon and Torre, 1994). Tomlinson *et al.* (2002) summarized results of 12 experiments and reported an overall significant reduction (196,000 vs. 294,000) in SCC when Zn-Met was supplemented (about 200 mg of Zn/d in 5 experiments and about 380 mg of Zn/d in 7 studies). In that summary, 4 of the experiments used a control diet that did not meet NRC (2001) requirements for Zn. Whitaker *et al.* (1997) compared the effects of providing supplemental Zn from a mixture of Zn proteinate (250 mg of Zn/day) and inorganic Zn (140 mg/day) or from all inorganic sources (390 mg of Zn/day). Diets contained approximately 50 ppm total Zn (about 25 ppm supplemental and 25 ppm from basal diet). Source of Zn had no effect on infection rate, new infections, clinical mastitis and SCC. More experiments similar to Whitaker *et al.* 1997 are needed to determine whether increasing Zn intake of cows is directly related to mammary gland health. Currently available data suggests that diets should contain about 20 ppm of copper (assuming no antagonists) and 50 to 60 ppm of Zn. Obtaining at least a portion of the supplemental zinc from zinc methionine may be beneficial. Vitamin C (ascorbic acid) is probably the most important water soluble antioxidant in mammals. Most forms of vitamin C are extensively degraded in the rumen, therefore the cow must rely on tissue synthesis of vitamin C. Cows can synthesize vitamin C and it is not considered an essential nutrient for cattle. The concentration of ascorbic acid is high in neutrophils (important immune cells with respect to mastitis) and increases as much as 30-fold when the neutrophil is stimulated. Santos *et al.* (2001) reported that plasma ascorbic acid concentrations in dairy cows were not correlated with somatic cell count (SCC). However, the range in SCC was limited (67,000 to 158,000/ml) and cows were only sampled once. Correlation analysis is a very weak statistical test for this type of data. Another experiment evaluated the therapeutic use of ascorbic acid following intramammary challenge with endotoxin (Chaiyotwittayakun *et al.*, 2002). One quarter from each cow was infused with endotoxin and the ascorbic acid was injected IV at 3 and 5 hours post challenge (25 g/dose). Vitamin C therapy had only limited effects on clinical signs.

Antioxidants and Retained Fetal Membranes (RFM)

Accumulating evidence strongly suggests that in many cases, RFM is an oxidative stress disease. The vitamin C concentration in maternal and fetal placental tissue is about 50% lower when cows have RFM than when they do not (Kankofer, 2001). Kimura *et al.* (2002) reported that neutrophils from cows with RFM had significantly less killing ability than neutrophils from cows without RFM. Neutrophils from Se-deficient cows have lower killing ability than neutrophils from Se-adequate cows (Hogan *et al.*, 1990). Supplementation of pro-oxidant nutrients (i.e., available iron) tends to increase the prevalence of RFM (Miller *et al.*, 1997).

Supplementation of certain antioxidant nutrients can reduce the prevalence of RFM. The majority of studies in which the control diet contained less than 0.1 ppm total Se have found that the incidence of RFM is reduced when Se is supplemented via the diet (0.1 to 0.3 ppm) Achi (2019) or via injections (about 50 mg of Se given 21 d prepartum), but Se supplementation had limited or no effect in studies in which the control diet contained more than 0.1 ppm Se. The effect of Se supplementation on RFM is influenced by the vitamin E status of cows. The incidence of RFM for cows that have low vitamin E status often is not influenced by Se supplementation (Harrison *et al.*, 1984). In a more recent study, a statistical association was found between the concentration of tocopherol in serum of periparturient cows and risk of RFM (LeBlanc *et al.*, 2004). They found that for every 1

ug/ml unit increase in serum tocopherol concentration (samples taken during the 1 wk before calving), the risk for RFM decreased by 21%. Cows that had a serum tocopherol: cholesterol ratio (ug tocopherol/ml divided by mg of cholesterol/ml) of <2 were three times more likely to have RFM than cows with a ratio >2 and cows with a ratio of <2.5 were twice as likely to have RFM as cows with a ratio >2.5. In that study (LeBlanc *et al.*, 2004) no associations were found between serum retinol and RFM or serum B-carotene and RFM.

CONCLUSION

From the review, role of antioxidant nutrients (Selenium and Vitamin E) in curbing cow mastitis and retained fetal membranes cannot be overemphasized; as such it is economically beneficial to supplement these nutrients in the diets of especially lactating and pregnant cows.

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