The Role of Selenium and Vitamin E in Mastitis and Reproduction of Dairy Cattle

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Role of Selenium and Vitamin E

Selenium was first recognized as an essential trace element in 1957 and identified as an essential component of the enzyme glutathione peroxidase (GSH-Px) in 1972. The highest concentration of selenium in the body of animals fed selenium-adequate diets is found in the kidney, followed by the liver. Erythrocytes and leukocytes contain approximately three fourths of whole blood selenium. However, almost all of the selenium associated GSH-Px activity in whole blood is associated with erythrocytes (Scholz and Hutchinson, 1979). Glutathione peroxidase catalyzes the conversion of hydrogen peroxide to water and converts lipid peroxides to lipid alcohols. Glutathione peroxidase functions in the cytosol of the cell. In addition to the role of GSH-Px as antioxidant, vitamin E also functions as an intercellular and intracellular antioxidant. Vitamin E protects polyunsaturated fatty acids, enzymes, and transports proteins in membranes from free radical attack. When acting as an antioxidant, vitamin E supplies become depleted.

Mastitis

Experiments have demonstrated that the selenium and/or vitamin E status of dairy cattle can affect the incidence, duration, and severity of clinical mastitis. Smith, *et al.*, 1984, assigned 80 non-lactating Holstein cows to one of four treatment groups: 1) Control, 2) Selenium (Se), 3) Vitamin E (E), and 4) Selenium and vitamin E (Se + E). Cows receiving selenium were injected with 0.1 mg selenium/kg of body weight i.m. 21 days prior to expected calving. Cows receiving vitamin E were supplemented with .74 g vitamin E per day in the ration. The incidence of clinical cases of mastitis per lactating quarter was reduced 12 - 37% in treated groups. The duration of clinical mastitis was reduced 13 - 41%. The predominance of clinical mastitis in this trial was associated with environmental pathogens. The plasma concentration of vitamin E during the prepartum period in the unsupplemented cows averaged 1.75 μg/ml and 2.74 μg/ml vitamin E supplemented cows. The serum concentration of selenium at calving for the control cows 0.0306 μg/ml and 0.0570 μg/ml for cows injected with selenium. Evidence from this trial would suggest that deficiencies of selenium and vitamin E may increase the incidence and duration of mastitis resulting from environmental pathogens. In an epidemiological study, Erskine, *et al.*, 1987, reported the blood selenium concentrations in dairy herds with high and low somatic cell counts. Two groups, each consisting of 16 dairy herds which were enrolled in Dairy Herd Improvement Association, were studied to evaluate the relationship between mean blood selenium concentrations and the prevalence of mastitis in the herd. The low somatic cell (SCC) herds had 12 month mean SCC of < 150,000 cells/ml and the high SCC herds had a 12 month mean herd SCC > 700,000 cells/ml. The herds with low SCC had significantly higher whole blood concentrations of selenium (0.133 μg/ml) and blood GSH-Px than herds with high SCC (0.074 μg/ml). The serum concentration of vitamin A, vitamin E, and beta-carotene were not significantly different between low and high SCC herds. The herds with high SCC had higher prevalence of *Streptococcus agalactia* and *Staphylococcus aureus*.

Erskine, *et al.*, 1989, experimentally challenged quarters of 20 Holstein heifers with coliform organisms in the 14th week of lactation. The heifers were divided into two groups; one group of 10 Holstein heifers was fed a selenium-deficient diet (0.04 mg of Se/kg of total ration on a dry matter basis) beginning 3 months before calving and continuing through the first lactation and one group of 10 heifers fed a diet supplemented with 2 mg selenium per day. The peak bacterial concentration in milk was higher and the duration of infection was longer for selenium-deficient cows. In addition, selenium-supplementation significantly reduced the number of agalactic and atrophied quarters compared to selenium-deficient heifers. Erskine, *et al.*, 1990, experimentally challenged quarters of 10 selenium-deficient (0.04 ppm Se in ration on a dry matter basis) cows and

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10 selenium-supplemented (0.14 ppm Se in ration on a dry matter) cows with *Staphylococcus aureus*. Infections were established in 14 of the 16 quarters from selenium-deficient cows and in 16 of the 19 quarters from selenium-supplemented cows. Peak bacterial concentrations occurred earlier and were higher in the selenium deficient cows. Similarly, somatic cell counts peaked earlier in selenium deficient cows. However, peak somatic cell counts did not differ between selenium-deficient and -supplemented groups. With respect to experimental staphylococcal mastitis, selenium status did not affect the percentage of challenge exposures resulting in infection, the duration of infection, or the severity of experimentally induced staphylococcal mastitis. From these experiments, the following conclusion can be drawn: 1) Selenium-supplementation of selenium-deficient cows is more effective in reducing the severity of environmental mastitis from coliform mastitis than staphylococcal mastitis. 2) Selenium-supplementation of selenium-deficient cows can reduce the severity of clinical signs including duration of febrile response, duration of clinical signs, and glandular atrophy and agalactia. 3) The mean whole blood selenium concentrations were significantly higher in herds with low somatic cell counts than in herds with high somatic cell counts. This suggests that low blood selenium concentrations are a risk factor for a higher prevalence of mastitis in the herd.

Reproduction

Retained Fetal Membranes

In a review of literature that summarized greater than 60,000 calvings, the average incidence of retained fetal membranes (RFM) was 10.3%. The causes of RFM can be divided into nutritional and non-nutritional categories. Selenium and vitamin E deficiencies are important nutritional causes of RFM. Trinder, *et al.*, first reported on the possible role of selenium in RFM in 1969. The incidence of RFM was higher at 42% (19/45) in untreated herdmates compared to 0% (0/20) in cows treated one month before calving with an injection of 15 mg selenium and 680 IU vitamin E i.m. In a later report (Trinder, *et al.*, 1973), the incidence of RFM for untreated cows was 44% (35/79), 5% (7/61) for cows treated with 15 mg Se and 680 IU vitamin E i.m., and 29% (9/31) for cows treated with only 15 mg Se i.m. The concentration of selenium in the diet of these cows was between 0.026 and 0.039 ppm of dry matter. The results of these trials suggest the incidence of RFM may further decrease when vitamin E supplementation is added. Gwazdauskas, *et al.*, 1979, randomly assigned 351 dairy cows to be either controls or to receive an injection of 21.9 mg Se and 500 mg vitamin E at 28 to 30 days prior to projected calving. The incidence of RFM in control cows was 10% (19/190) and 13.1% (21/160) for Se-vitamin E treated cows. This study did not report either the dietary intake of selenium or the concentration of selenium in plasma, serum or blood in control or treated cows. This study suggests there is no advantage to selenium-supplementation in a herd with a low incidence of RFM. Segerson, *et al.*, 1981, reported the results of a study where prepartum Holstein cows in four North Carolina herds were injected with 50 mg Se in combination with 680 IU vitamin E. The incidence of RFM was reduced in cows having borderline deficiencies of selenium prior to supplementation, but the incidence was not reduced in cows without deficiencies or in cows extremely deficient prior to selenium supplementation. Eger, *et al.*, 1985, reported on a series of five experiments involving selenium and vitamin E injected i.m. 3 weeks prepartum to Israeli-Holstein cows. The mean incidence of RFM for 348 control multiparous cows was 27.6% and 16.5% for control primiparous cows. Selenium, with or without vitamin E, significantly reduced the incidence of RFM to 14.7% in 428 multiparous cows and 7.0% in 186 primiparous cows. Selenium in the prepartum diet ranged from 0.035 to 0.109 ppm in the prepartum diet. Selenium alone was at least as effective as a combination of selenium and vitamin E in this study. Hidiroglou, *et al.*, evaluated the incidence of RFM in 627 parturitions. The herd was divided into 3 groups: 1) Control (n=217 cows), 2) Cows injected with 45 mg Se and 2040 IU of vitamin E (n=190), 3) Cows treated Se-containing intrarumenal boluses (n=220). The incidence of RFM (22.1%) was not reduced with either selenium supplement. The plasma selenium concentration at parturition in control cows was 0.0684 μg/ml and averaged 0.085 μg/ml in selenium-supplemented cows. Plasma selenium concentrations were adequate in control cows in this study thus explaining the failure of selenium-supplementation to reduce the incidence of RFM.

The following conclusions can be drawn from these studies relative to the benefit of selenium-supplementation: 1) Prepartum diets must be selenium deficient (<0.06 ppm of dry matter) if cattle are to benefit from selenium supplementation. 2) A benefit of selenium-supplementation on decreasing the incidence of RFM will not likely occur unless the incidence of RFM is greater than 20%. 3) The incidence of RFM will decrease with selenium-supplementation only if serum or whole blood selenium concentration are deficient before supplementation and Se- adequate post-supplementation. 4) Dietary supplementation of vitamin E of cows that are deficient may reduce the incidence of RFM, although the evidence of the benefit is not as strong as with Se. 5) The amount of selenium supplemented by injection to dry cows has ranged from 2.3 to 50 mg Se. 6) To effectively evaluate the benefit of selenium and vitamin E supplementation, the sele-

nium and vitamin E status of the animal must be determined both pre- and post-supplementation.

Cystic Ovarian Disease

Harrison, *et al.*, observed that cumulative incidence of cystic ovarian disease through the 14th post-partum week was 19% for cows supplemented with Se and 47% for cows not treated with selenium. Vitamin E status of these cows did not affect the incidence of cystic ovarian disease.

Fertility

There is limited information on the effect of selenium and vitamin E supplementation on fertility.

Evaluation of Selenium Status of Cattle

The clinician can evaluate the selenium status of animals either directly through laboratory determination of selenium concentrations in serum, plasma, or whole blood or glutathione peroxidase activity in whole blood, or indirectly through feed or ration analysis. The concentration of selenium in whole blood is about three times the concentration of selenium in serum or plasma. Since the concentration of selenium in erythrocytes is higher than in serum or plasma, hemolysis will result in an increase in serum or plasma selenium concentrations. Current recommendations suggest that whole blood be collected in either EDTA or heparin and used to estimate the Se status in cows (Maas, *et al.*, 1992). Since the life span of the bovine erythrocyte is 160 days and circulating erythrocytes do not have ability to corporate selenium into Se-dependent GSH-Px, whole blood selenium is an excellent indicator of long-term selenium status in cattle.

Table 1. Evaluation of Selenium Status

Selenium Status	Serum µg/ml	Whole Bld Se µg/ml
Deficient	<0.04	<0.11
Marginal	0.04 - 0.07	0.11 - 0.2
Adequate	>0.07	>0.2

Determining serum vitamin E concentrations may be the most efficient method of determining the vitamin E status of cattle.

Table 2. Evaluation of Vitamin E Status

Vitamin E Status	Serum µg/ml
Deficient	<1.5
Marginal	1.5 - 4
Adequate	>4

Selenium Supplementation

The literature would suggest that the selenium requirement for most species of livestock is between 0.05-0.3 ppm (mg/kg) of dietary dry matter. The current requirement for all classes of dairy cattle is 0.30 ppm (NRC, 1989). Selenium requirements are affected by dietary vitamin E intake, by disease process being prevented, and by the concentration of dietary antagonists including sulfur, iron, copper, and calcium.

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Escherichia Coli Bacterin For The Reduction of Clinical Signs of Toxic Mastitis In Cattle

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While herd surveys have indicated that 95% of mastitis in dairy cattle is caused by gram-positive bacteria such as *Streptococcus agalactiae* and *Staphylococcus aureus*,[1,2] mastitis caused by coliform organisms is gaining in recognition. Coliforms causing mastitis include gram-negative, lactose-fermenting organisms of the family Enterobacteriaceae in the genera *Escherichia, Klebsiella* and *Enterobacter*.[2,3] Routine mastitis control measures such as udder washing, teat dipping and milking machine sanitation have reduced the incidence of subclinical mastitis caused by the cocci, but they have not affected the incidence of coliform infections.[2,4,5] Herds in which mastitis caused by *Str. agalactiae* and *S. aureus* is under control may still have problems with clinical mastitis caused by coliforms.[3,6]

Prevalence of *E. coli* Mastitis

Investigators believe that *E. coli* is the most significant organism causing coliform mastitis because of its wide presence in the barnyard or dairy environment,[3,7] and studies across the United States have supported this concept.[1,3,8,9]

In a 1982 study in New York, pathogens were isolated from udder quarters of cows affected with clinical mastitis which were not responding to intramammary antibiotic treatment. Forty-four percent (32 of 72) of the cultures resulted in the discovery of *Escherichia coli*.[1] In a group of studies on eight dairy herds in California, 63% of the 158 coliform organisms cultured from cases of clinical mastitis were *E. coli*, 10% were *Enterobacter aerogenes* and 11% were *Klebsiella pneumonia*,[3] suggesting that *E. coli* is the predominant cause of coliform mastitis in California. Similar results were obtained from a single herd in Iowa.[8]

In 1985-86, *Escherichia coli* was isolated year-round, and was the predominant organism isolated during the summer in a Wisconsin study.[9] *Escherichia coli* was the principal cause of mastitis of cows in early and late lactation, while nearly equal numbers of *E. coli, Corynebacterium pyogenes*, streptococci and staphylococci were isolated at parturition. In this study, 45.5% (66 of 145) of the cultures obtained from cows with mastitis and anorexia resulted in the growth of *E. coli*. It was noted that the coliforms caused a more watery milk, higher rectal temperatures, less udder swelling and increased weakness and anorexia than did other mastitis pathogens.

Another investigator[10] found that most udders were resistant to *E. coli* infections during nonlactating periods, and that they became susceptible just before parturition. Other reports concur that *E. coli* mastitis is most prevalent during early lactation, and declines during late lactation to an insignificant level in nonlactating periods.[2,6]

Pathogenesis and Clinical Signs of *E. coli* Mastitis

Clinical mastitis caused by *Escherichia coli* begins with phagocytosis of the bacteria with resulting endotoxin release. Absorption of endotoxin in the mammary gland is then believed to cause the subsequent inflammatory and systemic reactions.[2] Investigators have shown that endotoxin can be detected in the blood following intramammary infusion,[11] and that acute

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