Relationships Among Selenium, Vitamin E, and Mammary Gland Health in Commercial Dairy Herds

W. P. WEISS, J. S. HOGAN, K. L. SMITH, and K. H. HOBLET
Ohio Agricultural Research and Development Center
The Ohio State University
Wooster 44691

ABSTRACT

Nine well-managed dairy herds were monitored for 1 yr to determine if bulk tank SCC and rate of clinical mastitis were associated with dietary and plasma Se and vitamin E status. Intakes of Se and vitamin E were 1 to 16 mg/d and 100 to 900 mg/d, respectively. Plasma Se concentrations were correlated positively with intakes of Se below 5 mg/d but were independent of Se intakes above 5 mg/d. Feeding vitamin E increased plasma concentrations of tocopherol, but the influence of dietary vitamin E on plasma concentrations was four times greater for dry cows than for lactating cows probably due to secretion of tocopherol into colostrum and milk. Bulk tank SCC averaged 5.4 log_{10}/ml and decreased significantly as Se concentration in plasma increased. Plasma glutathione peroxidase (GSH-Px; EC 1.11.1.9) activity in whole blood and percentage quarters infected with major pathogens in 32 herds. The authors concluded that Se may be involved in the resistance of the mammary gland to infection with Streptococcus agalactiae and Staphylococcus aureus. However, data are limited on the associations among vitamin E, Se, and mastitis in commercial herds that have controlled contagious mastitis. Therefore, the objectives of this study were 1) to determine dietary and plasma concentrations of Se and vitamin E in well-managed dairy herds; and 2) to determine correlations between dietary and plasma Se and vitamin E and incidence of clinical mastitis in herds with low SCC.

INTRODUCTION

The nutrition program of a dairy herd has a major influence on cow productivity and health. Deficiencies of Se and vitamin E have been implicated in a host of diseases. Severe deficiencies of either of these nutrients can cause white muscle disease (1), whereas less severe deficiencies of one or both of these nutrients increases the incidence of retained placenta, metritis (8), and mastitis (23). The recent discovery that supplemental vitamin E and Se can reduce the incidence of some kinds of mastitis in a research herd (23) spawned interest in determining if these nutrients influenced incidence of mastitis in commercial herds. Erskine et al. (6) reported a negative relationship between mean herd glutathione peroxidase (GSH-Px; EC 1.11.1.9) activity in whole blood and percentage quarters infected with major pathogens in 32 herds. The authors concluded that Se may be involved in the resistance of the mammary gland to infection with Streptococcus agalactiae and Staphylococcus aureus. However, data are limited on the associations among vitamin E, Se, and mastitis in commercial herds that have controlled contagious mastitis. Therefore, the objectives of this study were 1) to determine dietary and plasma concentrations of Se and vitamin E in well-managed dairy herds; and 2) to determine correlations between dietary and plasma Se and vitamin E and incidence of clinical mastitis in herds with low SCC.

MATERIALS AND METHODS

Cooperating Herds

Nine commercial dairy herds were monitored for 1 yr to determine correlations among
dietary and herd plasma concentrations of vitamin E and Se, rates of clinical mastitis, and bulk tank SCC. Herds were selected by criteria that indicated that mastitis caused by Strep. agalactiae and Staph. aureus had been controlled. Selection criteria, breed distributions, production data, and housing management for herds were described previously (10).

Analytical Methods

Rations for dry and early lactation cows were sampled for chemical analyses during March, July, and November. Rations for early lactation cows were those fed during the first 60 d of lactation. Feeds were analyzed for CP, Ca, P, Mg (2), NDF (20), Se (19), feed tocopherol and tocopherol acetate (25), and NE\textsubscript{E} was estimated using equation [7] of Conrad et al. (4).

Plasma samples for vitamin E and Se analysis were obtained from cows in each herd during March, July, and November. Samples were collected from 10 cows in each herd by the practicing veterinarians. Cows sampled were chosen from the available pool of cows within 60 d prior to calving to 60 d postcalving. Blood plasma was assayed for Se (19), \(\alpha\)-tocopherol (25), and GSH-Px (12). Activity of GSH-Px was expressed in enzyme units (EU = micromoles of NAPDH oxidized/minute).

Clinical Mastitis and Somatic Cell Counts

Duplicate quarter foremilk samples were collected aseptically from all quarters of a cow with clinical mastitis prior to a course of antibiotic therapy. Primary culture of all milk samples was on trypticase blood esculin agar (.01 ml of milk) and on MacConkey agar (.1 ml of milk) to aid detection of coliform intramammary infections (24). Bacterial isolates were identified as reported by Smith et al. (24). Rates of clinical mastitis were determined retrospectively using all reports of clinical signs and culture results from foremilk samples (10). Rates of clinical mastitis were expressed as [(number of clinical cases/305 cow-d) + .001] for statistical analysis.

Samples of bulk tank milk were collected weekly from each of the nine farms for 52 consecutive wk. Samples were collected (9) and transported on ice to the laboratory for analysis within 6 h after collection. Somatic cell counts were electronically measured by a Coulter Counter (Model ZB1 Coulter Electronics, Inc., Hialeah, FL).

Statistical Analyses

Linear regression was used to determine relationships among variables (18). Regressions concerning diet and mastitis used herd as the experimental unit \((n = 9)\). Relationships between different plasma compounds (e.g., activity of GSH-Px in plasma and concentration of Se in plasma) used cow as experimental unit. Sample size varied depending on which population of cows \((n = 89\) for dry and \(n = 231\) for lactating cows) was used. Heifers and primiparous cows were excluded from the statistical analyses that had feed values as independent variables, since information concerning the diet fed to these animals prior to lactation was lacking. Stage of lactation ranged from \(-83\) d to \(68\) d (calving = 0 d). Average stage of lactation was \(-20\) d for dry cows and \(19\) d for lactating cows.

RESULTS AND DISCUSSION

Nutrient composition of the diets fed during the experiment is in Table 1. Dry cow diets tended to be higher in NE\textsubscript{E}, CP, Ca, and P than NRC (17) recommendations. Dietary Se concentrations were below NRC recommendations for herds 2, 4, 7, and 9 and equal or greater than NRC for herds 1, 3, 5, 6, and 8. Dietary Se in unsupplemented herds averaged .13 and .41 ppm for supplemented herds. Sodium selenate was the source of supplemental Se. Additionally, cows in herds 1, 3, 6, 7, and 8 received injections of ca. 50 mg Se approximately 14 d prior to calving. Herds could also be divided into vitamin E supplemented and unsupplemented herds. Cows from herds 2, 4, and 7 were fed very little supplemental vitamin E during the dry period (9.9 IU/kg); whereas cows from herds 1, 3, 5, 6, 8, and 9 received supplemental vitamin E (29.3 IU/kg).

Lactating cow diets were generally low in NE\textsubscript{E} and high in CP, Ca, and P as compared with NRC. All lactating cow diets contained supplemental Se, but herd 9 was still marginally low in Se. All other herds were fed diets...
TABLE 1. Nutrient composition of diets of dry and early lactation cows from nine survey herds.

<table>
<thead>
<tr>
<th>Herd</th>
<th>NEI (Mcal/kg)</th>
<th>CP (%)</th>
<th>Ca (%)</th>
<th>P (%)</th>
<th>Mg (%)</th>
<th>Se ppm</th>
<th>Vitamin E (IU/kg)</th>
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<table>
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<td>NRC 1</td>
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1Current NRC (17) recommendations.

Selenium

Both dietary Se and vitamin E have been related to incidence and severity of mastitis (23); therefore, these nutrients were examined in more detail. The concentration of Se in blood plasma was positively correlated to concentration of Se in the diet when the data set contained just dry cows or dry cows and lactating cows (Figure 1). No relationship existed between dietary Se and plasma Se in lactating cows. Regressing plasma Se (µg/ml) on dietary concentration (ppm) gave the following relationships:

All cows:

Plasma Se = .062 + .040 Feed Se  [1]

($r^2 = .40; P<.01$)

The discrepancy between dry and lactating cows is probably due to the differences in total intake of dietary Se (Figure 2). These data are based on estimated DM intake (based on amounts fed by producers); hence, the relatively large horizontal standard errors in Figure 2. Figure 2 reveals that plasma Se concentration reached a plateau at about .08 μg/ml. The inflection point occurs at an intake of about 5 mg Se/d. This is similar to the value (6 mg) obtained by Maus et al. (16); however, in the previous study, plasma Se reached a plateau at about .11 μg/ml. The discrepancy between Maus et al. (16) and the current experiment is probably due to a statistical aberration. The lactating cow data set included cows ranging from fresh to about 60 d in milk. Plasma Se values for cows just after freshening will reflect dry cow nutrition; therefore, the blood values were relatively low. These data points reduced the average plasma values for lactating cows. This is clearer when concentration of Se in plasma is plotted against lactation day (Figure 3).

The data in Figure 2 show that maximum plasma Se occurred when cows were fed about 5 mg Se/d. Higher dietary Se did not increase plasma Se. If maximum concentration of Se in plasma is indicative of optimal Se status in cows, dry cows in this study were either not receiving or were not absorbing adequate amounts of dietary Se, even though concentrations of Se in the diet exceeded NRC (17) in most of the herds. Lactating cows, however, generally consumed at least 5 mg Se/d. When plasma Se concentrations were plotted versus lactation day (Figure 3), differences in Se status between dry and lactation cows can be observed. During the dry period and the first 2 wk of lactation plasma Se averaged about .075 μg/ml. After 14 d in milk, plasma Se tended to increase. The increase was probably due to increased Se intake (dietary concentration of Se and DM intake increased when cows freshened). Previous research has shown that a 4- to 7-wk lag period occurs between the time of increasing dietary Se and maximum plasma concentrations (16, 21).

**Vitamin E**

The relationship between dietary concentration of vitamin E (α-tocopherol plus α-tocopherol acetate, IU/kg) and plasma concentration of α-tocopherol (μg/ml) is shown in Figure 4. Data from dry and lactating cows were distributed uniformly across dietary concentration of vitamin E but not across plasma concentrations of vitamin E. Regressing concentration of vita-
min E in plasma (µg/ml) against concentration of vitamin E in the diet (IU/kg) gave the following equations:

All cows:

Plasma vitamin E = .099 + .037 Feed [4]
(r² = .45; P<.01)

Lactating cows:

Plasma vitamin E = 1.09 + .026 Feed [5]
(r² = .64; P<.01)

Dry cows:

Plasma vitamin E = .87 + .049 Feed [6]
(r² = .52; P<.03)

Numerically, changes in plasma tocopherol were twice as sensitive to changes in concentration of vitamin E in the diet of dry cows as in lactating cows, but statistically, the slopes were similar (P>.21). The difference between dry and lactating cows was more pronounced when plasma concentrations of tocopherol were plotted against estimated intake of vitamin E (Figure 5). This was probably due to the greater DM intake of lactating cows versus dry cows. The regression equations for plasma tocopherol (µg/ml) versus daily intake of vitamin E (mg/d) were:

All cows:

Plasma = 1.45 + .0010 Feed [7]
(r² = .14; <.12)

Dry cows:

Plasma = .85 + .0038 Feed [8]
(r² = .56; P<.02)

Lactating cows:

Plasma = 1.19 + .0010 Feed [9]
(r² = .45; P<.05)

The slope of the dry cow equation was greater than the slope of the lactating cow equation (P<.03). This could be due to at least two factors. First, dry cows could absorb vitamin E more efficiently. There are no data supporting or refuting this. Second, the volume of distribution for vitamin E may be different between dry and lactating cows. Fat-corrected milk contains about 900 µg tocopherol/kg (5); therefore, cows in this study were secreting between 20 and 40 mg of tocopherol/d into milk. The milk pool does not exist in dry cows so it is possible that tocopherol that was destined for milk in lactating cows stayed in the plasma pool of dry cows.

Plasma tocopherol values were influenced by stage of lactation (Figure 6). Plasma values were essentially constant from drying off until about 7 d prepartum. Concentration of plasma tocopherol then dropped by about 50% and remained low until 20 to 30 d postpartum. Plasma tocopherol continued to increase up to 60 d postpartum. This profile was similar to that reported by Stowe et al. (25). The drop in
plasma tocopherol during the peripartent period could be due to decreased feed intake during this period (17) and, therefore, decreased intake of vitamin E. Plasma tocopherol is fairly sensitive to changes in consumption of vitamin E in rats and humans (13, 14). Tocopherol could also be transferred from the blood pool to the colostrum and milk pools, thereby decreasing plasma concentrations of tocopherol.

**Glutathione Peroxidase**

Glutathione peroxidase activity in plasma was monitored because of its relationship to Se status of animals (1). Activity of GSH-Px in plasma (all cows) was correlated weakly with concentration of Se in the diets (dry and lactation) and estimated intakes of Se:

\[
\text{GSH-Px (EU/ml)} = .18 + .18 \text{ Feed Se (ppm)} \quad [10]
\]

\[
(r^2 = .21, P < .06)
\]

\[
\text{GSH-Px (EU/ml)} = .16 + .011 \text{ Feed Se (mg/d)} \quad [11]
\]

\[
(r^2 = .29, P < .03)
\]

No relationships were found between dietary vitamin E and GSH-Px.

Activity of GSH-Px was relatively constant over stage of lactation except for a peak during the peripartent period (Figure 7). Excluding the peripartent period (parturition ± 4 d), GSH-Px activity averaged about .25 EU/ml, which is similar to that reported by Harrison et al. (8). Consumption of Se was similar between the two studies. No relationships (\(P > .50\)) were found between plasma Se concentration and plasma GSH-Px activity or plasma tocopherol concentration and plasma GSH-Px for dry cows, lactating cows, or the complete data set. Some studies have found correlations between plasma Se and plasma GSH-Px (8, 16) when plasma Se values were very low \(< .04 \mu g/ml\). Concentration of plasma Se in the present study averaged about .08 \(\mu g/ml\). Harrison et al. (8) found no relationship between plasma Se and plasma GSH-Px when plasma Se values were above .07 \(\mu g/ml\). This indicates that with respect to plasma GSH-Px activity, cows in the present study were at maximal plasma Se concentration.

The peak in GSH-Px activity shortly after parturition is noteworthy. A similar peak in GSH-Px activity was also reported when dry cows were given an injection of Se ca. 14 d prior to calving (8). The peak in GSH-Px was very short-lived, lasting 1 to 2 d in most herds. This suggests that it was not due to changes in intake of Se, because plasma GSH-Px activity in 9-mo-old calves remained elevated several months after a single injection of .1 mg Se/kg body weight (26). Several plausible explanations exist that could explain the peak in plasma GSH-Px, including the following. In rats, the source of plasma GSH-Px is liver (3); the source of the enzyme has not been identified in cattle. Events at calving could trigger a short-term increase in synthesis of plasma...
GSH-Px by the liver. Another possible explanation is leakage of GSH-Px from other tissues into plasma as a result of the stresses of parturition. Activity of GSH-Px in red blood cells is several hundred times higher than that of plasma (15, 22). Therefore, a small amount of hemolysis occurring either in the cow or during bleeding and sample preparation could increase plasma GSH-Px activities markedly. The peak in GSH-Px occurs at the same time relative to calving as does the nadir for plasma tocopherol; however, plasma tocopherol values were not correlated statistically \( (r^2 = .05) \) to plasma GSH-Px activity. Low concentrations of tocopherol in plasma increase fragility of red blood cells (22); therefore, a biological relationship between plasma tocopherol and GSH-Px may exist. This will require additional research.

**Mammary Gland Status**

A strong correlation existed between mean bulk tank SCC and herd mean serum Se concentration. Regression of bulk tank SCC on average herd serum Se concentration (including dry and lactating cows) gave the following equation (Figure 8):

\[
SCC = 6.06 - 7.64(Se, \mu g/ml) \quad [12]
\]

\( (r^2 = .70, P<.005) \)

Plasma GSH-Px activity also was negatively correlated to bulk tank SCC.

\[
SCC = 5.69 - .99(GSH-Px, IU/ml) \quad [13]
\]

\( (r^2 = .46, P<.05) \)

Correlation was stronger when both serum Se and GSH-Px were included in the regression equation:

\[
SCC = 6.18 - 7.59(Se, \mu g/ml) - .56 (GSH-Px, IU/ml) \quad [14]
\]

\( (r^2 = .82, P<.007) \)

Herd serum Se and GSH-Px activity were not correlated \( (P>.25) \). Serum vitamin E concentrations were not related to SCC \( (P>.25) \). Similar relationships were found when SCC values were regressed on serum concentrations of Se in lactating cows (greater than 3 d in milk) only.

The SCC data correspond well with data for clinical mastitis. Bulk tank SCC values are indicative of prevalence of intramammary infection. Both SCC and rate of clinical mastitis were correlated negatively with concentration of Se in plasma. Plasma activity of GSH-Px was not associated with rate of clinical mastitis but was negatively correlated with SCC.

To determine if Se and vitamin E status were associated with rate of clinical mastitis, data were grouped in different ways. First, blood and feed data for dry cows were regressed on rate of clinical mastitis during the first 21 d of lactation (CM21) to determine the influence of dry cow nutrition on CM in early lactation.
lactation. Second, the effect of lactation diet on CM for the entire lactation (TCM) was determined by regressing feed and blood data from cows that were at least 4 d in milk on TCM. Four days were chosen to represent the time most animals were receiving the normal early lactation diet. Third, the effect of general herd nutrition (all cows included in data set) on TCM was determined by regressing herd average feed and blood data on TCM.

Other than Se and vitamin E, the only nutrients related statistically to either CM21 or TCM were NE and Mg. Concentrations of energy and Mg were correlated positively to TCM ($r^2 = .34; P<.10$ for both nutrients). Concentrations of Se and vitamin E in the diet were not correlated to NE or Mg ($P>.25$).

Amount of Se or vitamin E in the dry cow diet had no effect ($P>.5$) on CM21; however, herds with relatively high concentrations of Se ($\mu$g/ml) in plasma tended to have lower CM21:

$$CM21 = 29.1 - 312.8 \text{ Plasma Se} \quad [17]$$

$$r^2 = .34, P<.10$$

Concentration of tocopherol in plasma did not influence CM21, but an interaction between plasma Se and tocopherol was evident:

$$CM21 = 8.84 - 136.58 \text{ Se} + 1.37 \text{ vitamin E} \quad [18]$$

$$r^2 = .52, P<.12$$

This relationship would mean high concentrations of tocopherol in plasma were associated with increased incidence of mastitis when plasma Se concentrations were also high. This relationship was not found when the data were grouped differently. When diet composition and blood data for lactating cows only (greater than 3 d in milk) were regressed on TCM, concentration of tocopherol in plasma had no effect on TCM but increasing plasma Se concentration ($\mu$g/ml) was associated with decreased TCM:

$$TCM = 1.49 - 13.08 \text{ Se} \quad [19]$$

$$r^2 = .46, P<.05$$

Concentrations of Se (ppm) and vitamin E (IU/kg) in the diet for lactating cows were related to TCM, but in opposite directions:

$$TCM = .58 - .01 \text{ Feed vitamin E} \quad [20]$$

$$r^2 = .36, P<.09$$

From Equations [19, 21, 22], an apparent paradox was evident for Se. High dietary Se was associated with an increased incidence of mastitis but high plasma concentrations of Se were associated with reduced incidence of mastitis. Data comparing feed Se with plasma Se (Figure 1 and 2), however, showed no relationship between them in lactating cows. Increasing concentration of vitamin E in the diet tended to be associated with decreased incidence of mastitis. Plasma vitamin E concentrations had no effect on mastitis even though a relatively strong correlation existed between feed and plasma vitamin E.

Across all herds, TCM averaged .399; solving Equation [22] for TCM = .399 gives a feed vitamin E concentration of about 23 IU/kg and a Se concentration of about .5 ppm. This means as dietary Se increased above .5 ppm or dietary vitamin E decreased below 23 IU/kg, TCM would increase above the mean for the experiment. This is in conflict with regression Equation [18] for CM21 and plasma Se and tocopherol. The two regression Equations [20, 22] imply that high dietary concentrations of vitamin E are beneficial, whereas equation [18] implies that high concentrations of plasma tocopherol are associated with increased mastitis, at least during the first 21 d of lactation. This suggests that plasma tocopherol may not be the most appropriate pool to examine with respect to mastitis. Data from experiments using other species of animals support this contention. In humans, plasma tocopherol concentrations (ranging from approximately 0 to 25 $\mu$g/ml) were not highly correlated to concentration of tocopherol in platelets, red blood cells, and lymphocytes (4). Due to the large difference in plasma tocopherol concentrations between cows (ca. 2 $\mu$g/ml) and humans (ca. 12 $\mu$g/ml) the data should be extrapolated with caution. If plasma tocopherol is not representative of concentration of tocopherol in other cellular pools, this could explain the relationship between plasma Se, plasma tocopherol, and CM21. Up-
red blood cells was greater in rats deficient in Se than in rats receiving supplemental Se (7). However, radioactivity in plasma was elevated in Se supplemented rats but only for about 8 h postdose. Fischer and Whanger (7) concluded from their data that vitamin E was taken up and metabolized by liver and red blood cells faster in Se-deficient rats than in Se-supplemented rats. Our data also may support that conclusion. Concentration of Se (μg/ml) was correlated in Se-deficient rats than in Se-supplemented rats. However, radioactivity in plasma was elevated with concentration of plasma tocopherol (μg/ml) in lactating cows:

\[
\text{Plasma tocopherol} = -0.85 + 30.7 \text{ plasma Se} \\
(r^2 = .36, P<.09)
\]

When plasma Se concentrations were high, uptake of plasma tocopherol into tissue could be reduced, thereby increasing plasma tocopherol. High plasma concentrations of tocopherol in the presence of high plasma concentrations of Se may not be indicative of high cellular concentrations of tocopherol. The interactions between vitamin E and Se need to be examined in more detail, especially in cows receiving relatively high doses of dietary Se.

CONCLUSIONS

Vitamin E and Se were related to rate of clinical mastitis and bulk tank SCC in herds that had controlled contagious mastitis. High serum Se values were associated with reduced rates of clinical mastitis and low bulk tank SCC. Concentration of Se in serum was positively related to concentration of Se in the diet until cows consumed more than about 5 mg Se/d. Above this value, serum Se was independent of Se intake. Concentration of vitamin E in the diet was negatively correlated to rate of clinical mastitis. Vitamin E intake was positively correlated to plasma vitamin E concentrations, but vitamin E intake had a greater effect on serum vitamin E values in dry cows than in lactating cows.

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