

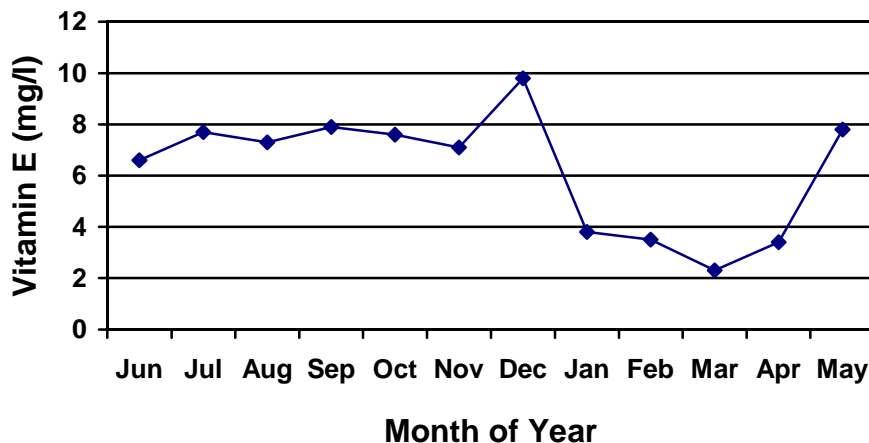
The Role of Selenium and Vitamin E in Milk Quality

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Introduction

Selenium and *vitamin E* are essential dietary nutrients in ruminants. The majority of dairy cows in the United States are located in areas with selenium deficient soils. Plants grown in these soils do not contain adequate levels of dietary selenium to meet the nutritional requirements of dairy cows. The primary source of vitamin E for dairy cows is forage. Processing and storage of forage can result in considerable loss of vitamin E. Serum vitamin E levels in cows have been demonstrated to decrease seasonally, corresponding approximately with length of feed storage (Fig. 1).¹ Therefore, it is not uncommon for inadequate dietary levels of selenium and vitamin E to be fed in many dairy herds.²

**Seasonal Distribution of Serum Vitamin E
in Dairy Cows**



Adapted from Braun, 1991

It is well documented that selenium and vitamin E play important roles in maintaining herd health of dairy cattle^{3,4,5} White muscle disease is known to be caused by a primary selenium deficiency.^{3,4} A number of selenium and vitamin E responsive reproductive conditions such as retained placenta and metritis have also been related to subclinical selenium deficiency.⁶ More recently, a role for selenium and vitamin E in mammary gland health has been defined.^{2,5,7,8,9,10,11,12}

Terminology, Measurement and Biology

Selenium is located within cells as an important component of the enzyme *glutathione peroxidase*. Vitamin E is a fat-soluble vitamin that is associated with cell wall membranes. The most biologically active form of vitamin E is referred to as *alpha-tocopherol*. Glutathione peroxidase and alpha-tocopherol are important components of the cellular defense system. Together they function to protect the cell membrane, and cell content from oxidative damage. One example of oxidative damage is the reaction of white blood cells (WBC's) to invasion of bacteria into the udder. When bacteria gain

entrance to the udder, an influx of white blood cells occurs to fight off the infection.¹³ Peroxides (and other substances) are produced by the WBCs to help destroy the pathogens. Uncontrolled, these peroxides can be dangerous to healthy cells and tissue. Vitamin E and selenium are necessary to help the white blood cells reduce the peroxides to safe substances and to continue destruction of invading pathogens. When an animal is deficient in vitamin E and/or Selenium, this function of WBC's is impaired.

Selenium status of cows can be measured directly in whole blood, plasma or serum. Selenium status can also be measured by testing glutathione peroxidase in whole blood. Serum and plasma Se concentrations reflect current nutritional conditions whereas glutathione peroxidase levels are reflective of diets fed several weeks previously. The Wisconsin Animal Health Laboratory (WAHL) recommends testing whole blood samples (purple top tubes) for selenium and serum samples (red top tubes) for vitamin E (Table 1). As with any other test, it is important to send samples from enough animals to be truly representative of the herd or group in question.

Table 1: Wisconsin Animal Health Laboratory Selenium and Vitamin E Testing

Test	Cost ^a	Sample	Interpretation
Selenium	\$7.00	Whole Blood (purple top tube)	Deficient: .004-0.08µg/ml Marginal: .060-0.16 µg/ml Adequate: .200-1.20µg/ml
Vitamin E	\$15.00 ^b	Serum (red top tube)	Deficient: <2.50 µg/ml Adequate: 3.0-10.0 µg/ml

^a1997-1998 in-state fee; ^bincludes vitamin A testing

The WAHL does not have sufficient computer capabilities to easily report the distribution of results from the numerous samples that it receives. A summary of selenium and vitamin E samples from adult cattle in Wisconsin sent to the Michigan Animal Health Diagnostic labs (AHDL) since 1994 indicates that up to 25% of samples from WI had marginal levels of these nutrients (Table 2). Because vitamin E is bound to fat, the Michigan AHDL reports the vitamin E-cholesterol ratio in addition to serum vitamin E determinations. They believe this ratio is a more sensitive indicator of biologically active vitamin E than serum vitamin E concentration alone.

Table 2: Result of Selenium and Vitamin E testing from Wisconsin Cows at the MI AHDL

	Selenium		Vitamin E	
	Whole Blood (ng/ml)	Serum (ng/ml)	Vit :Cholesterol Ratio (x10 ³)	Serum (µg/ml)
Number of samples	149	628	390	807
Average	182	74	3.0	4.18
25 th percentile	160	61	2.12	2.32
50 th percentile	183	72	2.88	3.78
75 th percentile	198	84	3.76	5.32
Interpretation	Deficient: <50	Deficient: <35	Deficient: <1.5	
Guideline ^a	Adequate: 120-150	Adequate: 70 – 100	Adequate: 2.5 – 6.0	

^aDeficient means the animal is at risk for deficiency syndrome; adequate means there is little possibility of benefit from additional supplementation

Impact of Selenium and Vitamin E on Udder Health

The first evidence of a relationship between selenium/vitamin E and udder health was reported in 1984.⁵ Since that time, numerous studies have explored this relationship. Initially researchers focused on the effect of *dietary deficiencies* of vitamin E and selenium on the occurrence of clinical mastitis.¹⁴ Later it was recognized that plasma alpha-tocopherol levels in dairy cows drop dramatically near calving (Figure 2).⁷

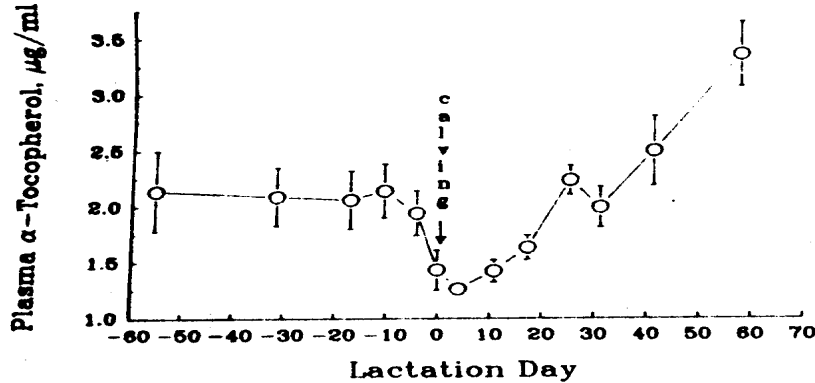


Figure 6. Changes in plasma tocopherol values over time. Points are averages of cows from nine different herds.

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This depression may be due a combination of reduced dry matter intake in dry cows and loss of vitamin E into milk of lactating cows. The depression coincides with a high-risk period for both clinical and subclinical intramammary infections.

Clinical Mastitis

Supplementation with vitamin E during the dry period has been demonstrated to decrease clinical mastitis in the following lactation. An Ohio study that compared feeding 740 IU/day vitamin E during the dry period to no vitamin supplementation reported a 37% reduction in clinical mastitis in the subsequent lactation.⁵ In that study, the duration of clinical symptoms was reduced by 46% for animals supplemented only with selenium, 44% for animals supplemented solely with vitamin E and 62% for animals supplemented with both vitamin E and selenium. Additional studies, by the same group reported a large reduction in cases of clinical mastitis in cows supplemented with vitamin E while receiving a low selenium diet (Table 3).¹¹

Table 3. Effect of vitamin E supplementation on the rate of clinical mastitis^a

Group	Prepartum Vitamin E ^b	Postpartum Vitamin E ^b	Clinical Mastitis Rate ^a
1	100 IU/d	100 IU/d	25.0%
2	1000 IU/d	500 IU/d	16.7%
3	1000 IU/d for 6 weeks 4000 IU/d for 2 weeks	2000 IU/d	2.6%

^apercent of quarters infected in the first 7 days of lactation, ^bfed in diets; from Weiss, et al, 1997

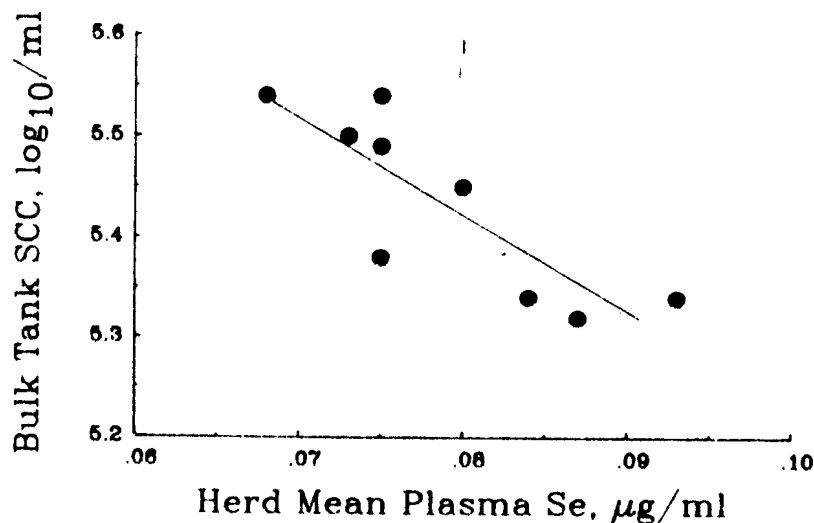
Selenium supplementation has been independently associated with clinical mastitis. The effect of selenium on clinical mastitis appears to be more significant for the prevention and recovery of infections caused by environmental pathogens as compared to infections caused by contagious pathogens. Experimental challenge studies have demonstrated a reduction in the severity and duration of clinical mastitis caused by *E. coli* but not *Staph aureus* in cows fed selenium supplemented diets as compared to cows that received selenium deficient diets.^{9,10}

Selenium and vitamin E act in an apparently synergistic way to protect the mammary gland. Some studies have been unable to demonstrate a beneficial effect of vitamin E in selenium deficient cows. The effect of supplemental vitamin E on clinical mastitis rates in herds with marginal to deficient levels of selenium was unremarkable in at least two reports.^{15,16} In cows with adequate levels of selenium and vitamin E, supplementation of either nutrient in excess of requirements does not seem to provide additional protection against clinical mastitis. Injections of vitamin E during the dry period did not decrease the rate of clinical mastitis during the following lactation in cows that were not deficient in either nutrient.¹⁷

Subclinical Intramammary Infections and Somatic Cell Count

Subclinical mastitis can be measured directly by culturing milk for bacteria or indirectly by measuring the somatic cell count (SCC). Using both of these measures, selenium levels have been clearly associated with subclinical infections of the udder. A Pennsylvania study found considerably lower concentrations of selenium in herds with high SCC ($\geq 700,000$) as compared to herds with low SCC ($\leq 150,000$).⁸ Mean herd bulk tank somatic cell counts have been shown to decrease as plasma selenium increases (Figure 2).⁷

Figure 2: Relationship between Selenium and bulk tank SCC



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In cows fed a selenium deficient diet, the level of vitamin E fed to dry cows has been shown to reduce the rate of subclinical intramammary infection at calving.¹¹ In this study, only the highest rate of supplementation (group 3 in table 2) had an effect on subclinical infection rates. There may not have been an adequate number of cases of subclinical mastitis in this study to demonstrate an effect at the lower supplementation rates.

Conclusions and Recommendations

Vitamin E and selenium play important and synergistic roles in maintaining udder health defense mechanisms. Depressed dry matter intakes, feed storage and processing, and the demands of lactation can contribute to dietary levels of selenium and vitamin E that are inadequate to optimize udder health in recently fresh cows. Clinical and subclinical mastitis rates may be increased in herds with low dietary levels of selenium and/or vitamin E. To ensure optimal udder health, the maximum allowable level of dietary selenium should routinely be fed to lactating and non-lactating dairy cows. Cows should be fed high enough levels of vitamin E to ensure that plasma levels of α -tocopherol are at least 3 μ g/ml at calving. Cows that are dependent upon stored forages may need to be supplemented with vitamin E at >1000 IU/d to maximize udder immunity during the critical post-parturient period.

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⁴ Smith, H.A., T. C. Jones and R. D. Hunt. 1972. *Veterinary Pathology*, 4th ed., Lea and Febiger, Philadelphia, PA

⁵ Smith, K.L., J. H. Harrison, D. D. Hancock, D. A. Todhunter, and H. R. Conrad. 1984. Effect of vitamin E and selenium supplementation on incidence of clinical mastitis and duration of clinical symptoms. *J Dairy Sci.* 67:1293.

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⁹ Erskine, R. J., Eberhart, R. J., and R. W. Scholz. 1990. Experimentally induced *Staphylococcus aureus* mastitis in selenium-deficient and selenium-supplemented dairy cows. *Am. J Vet. Res.* 51:1107.

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¹³ Craven, N., and M. R. Williams. 1985. Defenses of the bovine mammary gland against infection and prospects for their enhancements. *Vet. Immunol. Immunopath.* 2:71.

¹⁴ Smith, K. L., J. H. Harrison, D. D. Hancock, D. A. Todhunter, and H. R. Conrad. 1984. Effect of vitamin E and selenium supplementation on incidence of clinical mastitis and duration of clinical symptoms. *J. Dairy Sci.* 67:1293.

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¹⁷ Erskine, R.J., P. C. Bartlett, T.Herd, and P. Gaston. Effects of parenteral administration of vitamin E on health of periparturient dairy cows. 1997. *J. Am. Vet. Med. Assoc.* 211:466.