Antioxidant Nutrients and Milk Quality[1]

W. P. Weiss
Department of Animal Sciences
Ohio Agricultural Research and Development Center
The Ohio State University, Wooster 44691

Milk quality is usually defined in terms of mastitis. Milk with a low somatic cell count (SCC) and is visibly normal (no clots) is considered high quality. Although these measures are clearly important, the definition of high quality milk must be expanded. To ensure a continued growing demand by consumers for dairy products, milk and dairy products must also taste good. A consumer that has a bad tasting glass of milk may be hesitant to purchase milk again.

Antioxidant Systems

Normal cell metabolism, environmental insults, and inflammatory responses produce compounds called reactive oxygen species or free radicals. Environmental insults include solar radiation, tobacco smoke (a bigger problem for humans than cows), and many toxins, including some mycotoxins. An inflammatory response occurs during a bacterial infection such as mastitis The major free radicals found in biological systems are superoxide, hydrogen peroxide, hydroxyl radical, and fatty acid radicals. These compounds can react with enzymes, cell membranes, and DNA causing cell damage or cell death. Because free radicals are toxic to cells, the body has developed a sophisticated antioxidant system that relies on antioxidant nutrients (Table 1). Several trace minerals (as part of enzymes) and some vitamins are integral components of the antioxidant system. The system includes water soluble antioxidants (found in cell cytosol) and fat soluble antioxidants (found in cell membranes). Both water and fat soluble antioxidants are needed because free radicals are found in both areas of cells. A free radical located in a cell membrane cannot be neutralized by an antioxidant located in the cytosol. Known antioxidant pathways suggests that the requirements of antioxidant nutrients are interrelated. A deficiency of one antioxidant may increase the requirement of another nutrient. However, a deficiency of a particular antioxidant nutrient cannot be alleviated fully by another nutrient.

Simply because a nutrient is directly involved with the antioxidant system does not mean that supplementing diets with that nutrient will improve cow health. Cows need to consume a certain quantity of biologically available minerals and vitamins to maintain optimal status. A cow is in optimal status when she has adequate amounts of trace minerals and vitamins for maximal production and to maintain good health. When

---

[1]Originally presented at the Roche Symposium, Pacific Northwest Nutrition Conference, October, 2002, Vancouver, British Columbia, Canada.

cows are below optimal status, supplementation of a biologically available form of the nutrient should elicit a positive response but once optimal status is obtained, no additional positive responses would be expected when the nutrient is supplemented.

Table 1. Some of the antioxidant systems found in mammalian cells.

Component (location in cell)	Nutrients Involved	Function
Superoxide dismutase (cytosol)	Copper and zinc	An enzyme that converts superoxide to hydrogen peroxide
Superoxide dismutase (mitochondria)	Manganese and zinc	An enzyme that converts superoxide to hydrogen peroxide
Ceruloplasmin	Copper	An antioxidant protein, may prevent copper from participating in oxidation reactions
Glutathione peroxidase (cytosol)	Selenium	An enzyme that converts hydrogen peroxide to water
Catalase (cytosol)	Iron	An enzyme (primarily in liver) that converts hydrogen peroxide to water
α-tocopherol (membranes)	Vitamin E	Breaks fatty acid peroxidation chain reactions
β-carotene (membranes)	β-carotene	Prevents initiation of fatty acid peroxidation chain reactions

Excessive supplementation may increase oxidative stress, decrease immune function, and increase health problems. The likelihood that a cow will respond to mineral or vitamin supplementation is a function of the amount of nutrient the cow would consume with an unsupplemented diet. Selenium in soil (and the resulting crops) is low in the major dairy areas of the U.S. which means that selenium supplementation often elicits a positive response. Many feeds, however, contain substantial concentrations of manganese so a response to supplemental manganese is much less likely.

Mastitis and Antioxidants

Numerous experiments have been conducted examining the influence of nutrients on immune function in vitro and in vivo; several of which used cattle. Data

from these experiments are useful in understanding mechanisms and to suggest possible links to health disorders; however, altered immune status does not necessarily mean altered health of a cow. Epidemiological studies also can suggest relationships between nutrient status and mammary health, but results are equivocal. Clinical trials are needed to determine whether a nutrient affects health. A well-designed clinical trial is one which uses enough animals for statistical validity and uses accepted disease measurement protocols. Clinical trials measuring the impact of nutrition on mammary gland health (e.g., intramammary gland infections, clinical mastitis, etc.) are limited.

B-carotene and Vitamin A. Two clinical trials to study the effects of vitamin A and (or) B-carotene on mammary gland health of dairy cows were conducted at Washington State University. In one study, cows were fed 53,000 IU of supplemental vitamin A/day, 173,000 IU/d or 53,000 IU/day plus 300 mg of supplemental B-carotene/day starting 3 wk before dry-off and continuing through the dry period (Dahlquist and Chew, 1985). Cows fed the vitamin A plus B-carotene treatment had fewer new infections during the early dry period (27% of previously uninfected quarters) than cows fed the other treatments (49% for low vitamin A and 50% for the high vitamin A treatments). In another trial the same treatments were used but supplementation started 3 wk before calving and continued through 10 wk of lactation (Chew and Johnston, 1985). The vitamin A plus B-carotene treatment reduced mean SCC during wk 2 to 8 of lactation (85,000 cells/ml compared with 225,000 and 125,000/ml for low and high vitamin A treatments). Oldham et al. (1991) conducted a study using similar treatments (50,000 IU supplemental vitamin A/day, 170,000 IU/day, and 50,000 IU/day plus 300 mg of B-carotene/day) starting 2 wk before dry-off and continuing until 6 wk of lactation. In that study, treatment did not affect new infections during the dry period (mean = 11.3% of quarters), new infections at calving (mean = 6.4%), total new infections during the experiment (27.5%), and cases of clinical mastitis (9.4% of quarters). A likely reason for the difference between the Washington State studies and Oldham et al. is differences in vitamin A and B-carotene status of the control cows. At the start of the Dahlquist and Chew (1985) experiment, mean plasma concentration of B-carotene was 2.5 mg/L, but in the Oldham et al. study, the mean concentration was 10 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.

Selenium and Vitamin E. Smith et al. (1984) reported that dry cows fed approximately 1000 IU of supplemental vitamin E/day during the 60 day dry period and (or) injected with approximately 50 mg of Se 21 day before expected calving had fewer cases of clinical mastitis with reduced duration compared with cows not fed vitamin E or injected with Se. Malbe et al. (1995) reported that supplementation of a very low Se diet with 0.2 ppm of Se from selenite or selenized yeast reduced SCC in lactating dairy cows. In a study from New Zealand (Wichtel et al., 1994) mean SCC over an entire lactation was reduced or tended to be reduced when cows were supplemented with 6 to 12 mg of Se/day, however, no difference in clinical mastitis was found between treatments. 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 1000 IU/d of supplemental vitamin E during a 60 day dry period to cows with low Se status reduced clinical mastitis during the first week of lactation compared with feeding 100 IU/day. In that same study, feeding 4000 IU of supplemental vitamin E/day during the last 14 days of the dry period further reduced clinical mastitis and reduced new infections at calving. Clinical mastitis incidence during the first week of lactation was 25.0. 16.7, and 3.6% of quarters for cows fed 100 IU/day, 1000 IU/day, or 1000 IU/day during the first 46 days of dry period followed by 4000 IU/day during the last 14 days of the dry period. An important feature of that study was that cows were fed a low Se diet (0.1 ppm) and had low plasma Se concentrations (0.048 mg/L).

Copper. No peer-reviewed publications were found from clinical trials on the effects of Cu on mammary gland health, but two reports that have not been peer-reviewed are available. Harmon and Torre (1994) used heifers that received no supplemental dietary Cu since weaning and fed them a diet with 0 or 20 ppm supplemental Cu (from Cu sulfate) from 84 days before calving through 105 days of lactation. At calving, heifers fed supplemental Cu had fewer quarters infected than unsupplemented animals. Following an *E. coli* endotoxin challenge, cows supplemented with Cu had lower clinical score and fewer abnormal quarters than unsupplemented cows. Supplementation had no effect on the response to a *S. aureus* challenge. Scaletti et al. (2000) fed heifers a diet with about 7 ppm total Cu or the same diet plus 20 ppm supplemental Cu (from Cu sulfate). Copper supplementation reduced the severity of mastitis following experimental challenge with *E. coli*. Based on liver Cu concentrations, the unsupplemented group was Cu deficient.

Zinc. Peer-reviewed clinical data are also extremely limited on the effects of dietary Zn on mammary gland health. Several studies (mostly non-peer reviewed) have examined the effect of supplemental Zn from Zn methionine on SCC. Supplementation was typically about 360 mg of Zn/day and in most studies the control and treatment diets were not equal in Zn concentrations (any effect could be caused by source or amount of supplemental Zn). In most studies Zn methionine supplementation statistically or numerically reduced SCC (see review by Kellogg, 1990). A recently published abstract (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 8 studies). In that summary, 4 of the experiments used a control diet that did not meet NRC (2001) requirements for Zn. Only one study on Zn was found in which incidence of mastitis and infection rates were measured. 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. are needed to determine whether increasing Zn intake of cows is directly related to mammary gland health.

Manganese. No clinical studies have been published on the effect of Mn on mammary gland health.

Antioxidants and Milk Flavor

On average, most fluid milk is judged to have a good flavor up to 14 d of storage but off-flavor of milk is still an important problem (Boor, 2001). Oxidized flavor (OF) is described as cardboard-like, metallic, or tallowy, and can develop over time because of improper storage and handling of the milk. In certain situations, OF can be detected in milk almost immediately following milking. Some milk is more susceptible to developing OF than other milk. The fatty acid profile of milk fat is a major factor in the development of OF. Oxidized flavor is more likely in milk that has a high concentration of polyunsaturated fatty acids such as linoleic or linolenic acid. The concentrations of those two fatty acids in milk can be increased by feeding certain oilseeds or rumen protected oils. Cows fed soybeans or linseeds can produce milk with very high concentrations of linolenic and linoleic acids (Charmley and Nicholson, 1994; Focant et al., 1998; Timmons et al., 2001). As the use of these types of products increase, OF may become a larger problem.

The antioxidant status of the cow can influence the development of oxidized flavor. Some antioxidants (for example, Cu) can increase susceptibility to oxidized flavor development, others reduce susceptibility. Milk with high concentrations of Cu is extremely susceptible to the development of OF (Barrefors et al., 1995), especially if the milk also is high in polyunsaturated fatty acids (Timmons et al., 2001). Within a reasonable range of dietary Cu concentrations, milk Cu is not highly correlated with Cu intake but at very high dietary Cu concentrations (about 80 ppm) milk Cu is elevated (Dunkley et al., 1968). The vitamin E concentration of milk is correlated with vitamin E intake but large changes in intake of vitamin E elicit only modest changes in milk vitamin E (St.-Laurent et al., 1990). Usually less than 2% of the vitamin E consumed by a cow is secreted in her milk and the efficiency of transfer decreases as vitamin E intake increases (Figure 1). Because of the low transfer of dietary vitamin E to milk, substantial amounts of vitamin E must be consumed by cows to reduce OF. In one study cows fed micronized soybeans and 8000 IU of vitamin E/d still produced OF milk (Charmley and Nicholson, 1994). Focant et al. (1998), reported that feeding about 10,000 IU/d of vitamin E eliminated OF in cows fed a mixture of canola and linseeds. Other studies have reported that cows fed between 700 and 3000 IU of vitamin E/d had reduced OF in milk but significant OF still occurred (St.-Laurent et al., 1990). Available data are not conclusive regarding the amount of dietary vitamin E needed to prevent OF when oilseeds are fed, but a generally at least 3000 IU of vitamin E/d is recommended when OF is a problem. The very high supplementation rates needed to eliminate OF

(about 10,000 IU/d) is costly but reduced consumption of dairy products caused by bad tasting milk also is costly.

NRC 2001 Requirements for Trace Minerals and Vitamins

For some trace nutrients, immune function and clinical mammary gland health data were used to establish requirements in the 2001 NRC. The requirements for trace minerals and vitamins published in that book should be the starting point when formulating diets. No currently available data suggests that supplementation above NRC (2001) requirements will improve mammary gland health except in specific, limited situations. The NRC requirements assume that bioavailability of minerals and vitamins are normal, but if excessive antagonists are consumed, NRC requirements may not be adequate. For example, diets (or drinking water) that provide excessive amounts of sulfur and molybdenum reduce availability of Cu and NRC requirements may not be adequate.

Trace mineral requirements in the NRC are given as milligrams of each mineral that must be absorbed each day, not as dietary concentrations. To convert NRC requirements to dietary concentrations, one must know dry matter intake and the absorption coefficient of the mineral. Intake can be measured on-farm or estimated using equations. Average absorption coefficients for common sources of supplemental minerals are in NRC (2001). For vitamins, NRC recommendations are expressed as IU of supplemental vitamin needed unit of body weight.

Vitamin A. The new NRC increased the vitamin A requirement of dry and lactating cows to 110 IU/kg of body weight (50 IU/lb). On a daily basis, an average lactating Holstein cow should consume about 72,000 IU of supplemental vitamin A/day, and an average dry Holstein cow should consume about 77,000 IU/day. Based on clinical data, no improvement in mammary gland health should be expected at supplementation rates greater than these requirements.

B-carotene. The NRC did not establish a requirement for B-carotene. Based on clinical data, mammary gland health may be improved with B-carotene supplementation when cows are in low B-carotene status. The cost of supplementing 300 mg of B-carotene/d is substantial and should not be done routinely. Diets that contain a large proportion of weathered hay may be low in B-carotene and supplementation might be useful. Diets based on good quality silage or fresh forage probably provide adequate B-carotene and supplementation would not be economical.

Vitamin E. The new NRC, based on clinical mammary gland health data, substantially increased the vitamin E requirement. The new requirement for a dry cow is 1.6 IU/kg of body weight (0.7 IU/lb) and 0.8 IU/kg of body weight (0.36 IU/lb) for a lactating cow. This equates to about 500 IU of supplemental vitamin E/day for a lactating Holstein cow and 1000 IU/day for a dry Holstein cow. Because fresh forage

usually has a very high concentration of vitamin E, cows that consume a substantial amount of pasture or green chop do not need as much supplemental vitamin E. The NRC suggest that grazing cows be supplemented with approximately 160 IU/day (lactating cows) or 330 IU/day (dry cows). Limited data suggest that supplementation of vitamin E above NRC might be necessary to optimize mammary gland health when periparturient cows are in marginal to poor Se status.

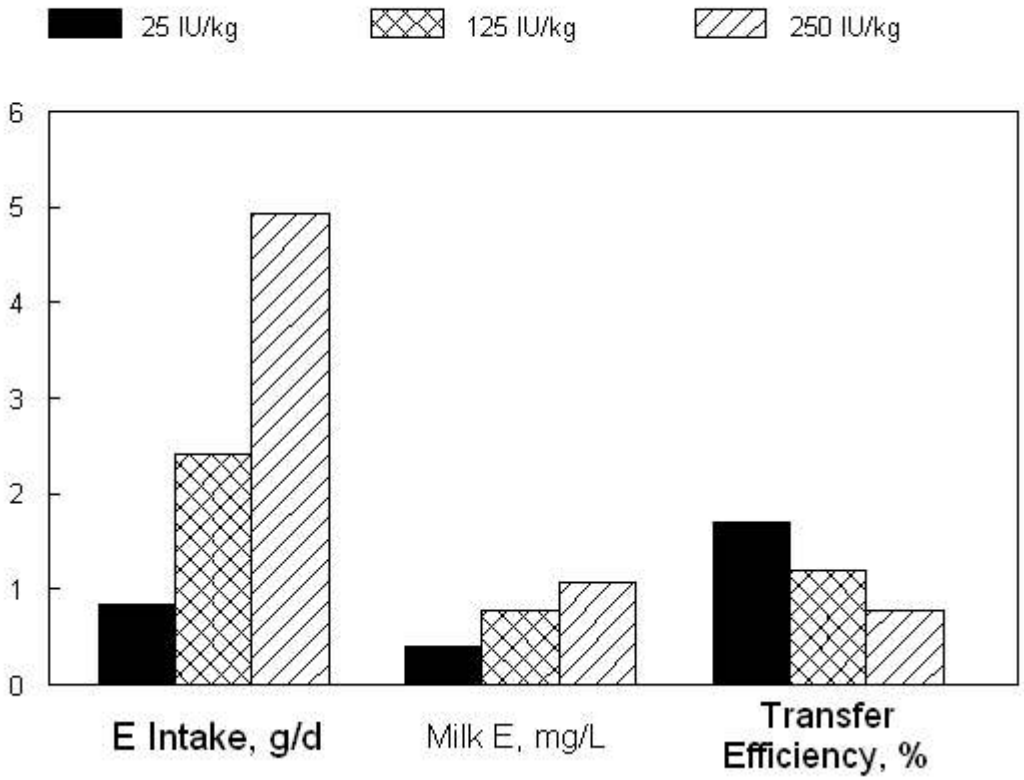

Figure 1. Effect of vitamin E intake on concentrations of vitamin E in milk and efficiency of transfer of consumed vitamin E to milk vitamin E. Treatments are in IU of supplemental vitamin E (all-rac a-tocopheryl acetate). Transfer efficiency calculated as milligrams of vitamin E consumed/d divided by milligrams of vitamin E secreted in milk/d times 100 (Weiss, unpublished).

Selenium. The concentration of supplemental Se in dairy diets is regulated by the U.S. FDA at 0.3 ppm and the new NRC set the Se requirement at 0.3 ppm for lactating and dry. No clinical data are available suggesting that more than 0.3 ppm supplemental Se will improve mammary gland health.

Copper. The new NRC increased the Cu requirement of lactating and dry cows. Assuming normal bioavailability and typical ingredients an average dry Holstein cow needs to consume 170 to 180 mg of Cu/day. An average Holstein cow producing 50 or 100 lbs of milk needs to consume 225 mg or 300 mg of Cu/day, respectively. These recommendations assume normal dietary concentrations of sulfur and molybdenum. When diets or drinking water contain excessive concentrations of sulfur and molybdenum these Cu recommendations will not be adequate. Clinical data showing positive responses to Cu supplementation at 20 ppm (total diet Cu was approximately 25 to 27 ppm) used control diets that were below NRC requirements. An improvement in mammary gland health to Cu supplementation would be less likely if control cows were fed Cu at NRC recommendations. Relative to requirements, Cu is the most toxic mineral routinely supplemented (recommended safe concentration is only four to five times the requirement); excessive supplementation of Cu should be avoided.

Zinc. Assuming normal bioavailability and common ingredients the new NRC Zn requirement is about 300 mg/day for an average Holstein dry cow, about 900 mg/day for a cow producing 50 lbs of milk, and 1400 mg/day for a cow producing 100 lbs of milk. Because clinical data are lacking, it is not known whether these concentrations are adequate for optimal mammary gland health. Cows can tolerate relatively high dietary concentrations of Zn (at least ten times requirements without adverse effects), however excessive Zn supplementation can cause a secondary Cu deficiency. Because of the interaction between Zn and Cu, dietary Zn should not exceed five times the concentration of dietary Cu.

Manganese. The new NRC requirements for Mn are about 240, 280, and 350 mg/day for a dry cow, a cow producing 50 lbs of milk, and a cow producing 100 lbs of milk, respectively. These values are substantially lower than previous recommendations. No data are available on the effects of Mn on mammary gland health. Manganese has a high safety factor (approximately 50 times requirement).

Recommended Dietary Concentrations

The approach followed by NRC of expressing requirements in either milligram or IU/day is scientifically correct and accounts for important sources of variation (i.e., variation in intake and bioavailability). However, the NRC was designed to evaluate the nutritional adequacy of diets rather than formulating diets. The NRC values should be considered minimum requirements--they do not include any safety margins. Because of variation in intake, environment and feed composition, diets should be formulated to provide more than recommended amounts of some minerals to ensure all cows consume adequate amounts of nutrients. The guidelines in Table 2 are based on NRC requirements plus adjustments for some expected variation.

Table 2. Suggested dietary concentrations (dry matter basis) of trace nutrients[1].

	Nonlactating cows			Lactating cow (milk yield)	
	Dry	Pre-fresh	Fresh cow	50 lbs	100 lbs
Est. intake, lbs/day	30	22	30	44	58
Vitamin A, IU/lb	3300	4500	3300	1850	1500
Vitamin E, IU/lb	35	50	25	12	10
Selenium, ppm	0.3	0.3	0.3	0.3	0.3
Copper, ppm	20	20	15 - 20	15 - 20	15 - 20
Manganese, ppm	30 - 50	40 - 50	40 - 50	30 - 40	30 - 40
Zinc, ppm	40 - 60	50 - 70	60 - 80	50 - 70	60 -80

[1]Values are for a Holstein cow with an average body weight for various stages of lactation and gestation. Pre-fresh is for cows in the last 2 wk of gestation. Fresh is for cows in the first 3 wk of lactation.

References

Barrefors, P., K. Granelli, L.-A. Appelqvist, and L. Bjoerck. 1995. Chemical characterization of raw milk samples with and without oxidative off-flavor. J. Dairy Sci. 78:2691-2699.

Boor, K. J. 2001. Fluid milk quality and safety: looking to the future. J. Dairy Sci. 84:1-11.

Charmley, E., and J. W. G. Nicholson. 1994. Influence of dietary fat source on oxidative stability and fatty acid composition of milk from cows receiving a low or high level of dietary vitamin E. Can. J. Anim. Sci. 74:657-664.

Chew, B. P., and L. A. Johnston. 1985. Effects of supplemental vitamin A and B-carotene on mastitis in dairy cows. J. Dairy Sci. 68 (suppl. 1):191 (abstr.).

Dahlquist, S. P., and B. P. Chew. 1985. Effects of vitamin A and B-carotene on mastitis in dairy cows during the early dry period. J. Dairy Sci. 68 (suppl. 1):191 (abstr.).

Dunkley, W. L., A. A. Franke, J. Robb, and M. Ronning. 1968. Influence of dietary copper and ethylenediaminetetraacetate on copper concentration and oxidative stability of milk. J. Dairy Sci. 51:863-866.

Erskine, R. J., R. J. Eberhart, P. J. Grasso, and R. W. Scholz. 1989. Induction of Escherichia coli mastitis in cows fed selenium-deficient or selenium-supplemented diets. Amer J Vet Res 50:2093.

Erskine, R. J., R. J. Eberhart, and R. W. Scholz. 1990. Experimentally induced Staphylococcus aureus mastitis in selenium-deficient and selenium-supplemented dairy cows. J Amer Vet Med Assoc 51:1107-1111.

Focant, M., E. Mignolet, M. Marique, F. Clabots, T. Breyne, D. Dalemans, and Y. Larondelle. 1998. The effect of vitamin E supplementation of cow diets containing rapeseed and

linseed on the prevention of milk fat oxidation. J. Dairy Sci. 81:1095-1101.
Harmon, R. J., and P. M. Torre. 1994. Copper and zinc: do they influence mastitis? Pages 54-65 in Proc. National Mastitis Council. Orlando, FL.
Jukola, E., J. Hakkarainen, H. Saloniemi, and S. Sankari. 1996. Blood selenium, vitamin E, vitamin A, and B-carotene concentrations and udder health, fertility treatments and fertility. J. Dairy Sci. 79:838-845.
Kellogg, D. W. 1990. Zinc methionine affects performance of lactating cows. Feedstuffs Aug 20, 1990 pg 15
Malbe, M., M. Klaassen, W. Fang, V. Myllys, M. Vikerpuur, K. Nyholm, W. Sankari, K. Suoranta, and M. Sandholm. 1995. Comparisons of selenite and selenium yeast feed supplements on Se-incorporation, mastitis, and leukocyte function in Se-deficient dairy cows. J. Vet. Med. (Ser. A) 42:111-121.
National Research Council. 2001. Nutrient Requirements of Dairy Cattle. Natl. Acad. Press, Washington DC.
Oldham, E. R., R. J. Eberhart, and L. D. Muller. 1991. Effects of supplemental vitamin A and B-carotene during the dry period and early lactation on udder health. J. Dairy Sci. 74:3775-3781.
Scaletti, R.W., D.S. Trammell, B.A. Smith, and R.J. Harmon. 2001. Role of dietary copper in altering response to intramammary E. coli challenge. Pages 29-33 in Proc. Int. Mastitis Symp., Natl. Mastitis Counc./Am. Assoc. Bovine Practitioners, Vancouver, BC, Canada.
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. 67:1293-1300.
St.-Laurent, A. M., M. Hidiroglou, M. Snoddon, and J. W. Nicholson. 1990. Effect of a-tocopherol supplementation to dairy cows on milk and plasma a-tocopherol concentrations and on spontaneous oxidized flavor in milk. Can J Anim Sci 70:561-570.
Timmons, J. S., W. P. Weiss, D. L. Palmquist, and W. J. Harper. 2001. Relationships among dietary roasted soybeans, milk components, and spontaneous oxidized flavor of milk. J. Dairy Sci. 84:2440-2449.
Tomlinson, D. J., M. T. Socha, C. J. Rapp, and A. B. Johnson. 2002. Summary of twelve trials evaluating the effect of feeding complexed zinc methionine on lactation performance of dairy cattle. J. Dairy Sci. 85 (suppl. 1): 106 (abstr.).
Weiss, W. P., J. S. Hogan, D. A. Todhunter, and K. L. Smith. 1997. Effect of vitamin E supplementation in diets with a low concentration of selenium on mammary gland health of dairy cows. J. Dairy Sci. 80:1728-1737.
Whitaker, D. A., H. F. Eayres, K. Aitchison, and J. M. Kelly. 1997. No effect of a dietary zinc proteinate on clinical mastitis, infection rate, recovery rate, and somatic cell count in dairy cows. Vet. J. 153:197-204.
Wichtel, J. J., A. L. Craigie, H. Varela-Alvarez, and N. B. Williamson. 1994. The effect of intra-ruminal selenium pellets on growth rate, lactation, and reproductive efficiency in dairy cattle. New Zealand Vet J 42:205-210.