

Vitamin E and Other Good Stuff in Dairy Nutrition

H. Fred Troutt, VMD, PhD
Department of Veterinary Clinical Medicine
College of Veterinary Medicine
University of Illinois at Urbana-Champaign
Urbana, Illinois 61801

INTRODUCTION

We've known for a considerable period of time that the dairy cow in early lactation *walks* a nutritional-metabolic *tightrope* in terms of her capacity to maximize milk production, conceive efficiently, and resist metabolic disease. We've been concerned particularly for those metabolic disorders associated with altered carbohydrate and lipid metabolism. However, we are now fundamentally aware that the recently post-parturient cow is increasingly susceptible to other disease states that etiologically can have a metabolic basis (udder edema) or a metabolic-infectious interaction (mastitis and retained placenta-metritis). Statistically these conditions may have common and related causal associations (Gröhn et al., 1989) dealing with nutritional deficiencies of antioxidants, such as vitamin E and selenium, that allow clinical expression of oxidative stress (Miller et al., 1993). The relationship between an increased incidence of retained placenta and diminished vitamin E and selenium nutrition is well established (Harrison et al., 1984). And the efficacy of supplemental vitamin E and selenium to reduce the incidence of clinical mastitis has been known since at least 1984 (Smith et al., 1984).

These oxidative stress conditions--udder edema, retained placenta, and mastitis--are more than likely initiated in the dry period and, at least with regard to mastitis, involve some suppression of the cow's immune system (Hogan et al., 1993). Chandler (1990) has emphasized that the dry cow period on many dairy farms, although forming the

basis for optimal production, is an "abused and mismanaged" period with respect to nutritional programs in general, but also programs that foster enhancement of the immune system.

As we continue to emphasize maximal production through the increased use of energy dense, silage based, early lactation rations, and later production enhancers; it is likely that clinical conditions, precipitated by oxidative stress due to relative deficiencies of antioxidants such as vitamin E, will be exacerbated.

OXIDATIVE STRESS

Miller et al. (1993) offer a detailed explanation of oxidative stress, the production of reactive oxygen metabolites (ROM), and antioxidant defense. Oxidative stress results when ROM are not effectively removed from biological tissues because of a deficiency of antioxidants (Miller et al., 1993), such as vitamin E and/or selenium. The production of ROM can be the result of usual metabolic activities (endogenous) or they may be induced by exogenous (xenobiotic) influences. Examples of xenobiotic influences producing ROM include mycotoxins (Miller et al., 1993) and Maillard or browning reaction products (Zanzalari et al., 1989) present in dairy cattle feed.

ROM, if not effectively removed by an antioxidant (e.g. vitamin E), can induce cellular damage by oxidizing cellular molecules including proteins, polysaccharides, and DNA; and enzyme systems can be interrupted (Miller et al., 1993).

Table 1. Reactive Oxygen Metabolites*

Name	Symbol	Name	Symbol
Superoxide	(O ₂ ⁻)	Peroxy radical	(RO ₂ [•])
Hydrogen peroxide	(HOOH)	Lipid peroxide	(LOOH)
Hydroxyl radical	(•OH)	Reduced iron	(Fe ⁺⁺)
Oxidized fatty acid	(L [•])		

*Modified from Table 1 presented by Miller, et al (1993).

ROM can attract a hydrogen atom and its electron from other molecules, such as polyunsaturated fatty acids, establishing a chain reaction of electron transfers (Gardner, 1989). If not controlled, cell membranes will be altered, affecting cell membrane integrity and permeability. Antioxidants quench ROM or free radicals (molecules, single reactive electron outer ring or shell; Southorn and Powis, 1988).

The potential damage as a consequence of ROM include (Miller et al., 1993):

- a. Reduced supplies of NADPH and diversion of glucose.
- b. Peroxidation of steroidogenic enzymes and inhibition of androgen and estrogen production with possible elevation of corticosterone, which would tend to increase sodium and water retention as in udder edema.
- c. Peroxidation of polyunsaturated fatty acids with loss of membrane integrity.

ANTIOXIDANT SYSTEMS

Under usual circumstances the mammalian system contains an integrated antioxidant defense system against ROM. The components of this system have been classified by Miller et al. (1993) as "preventive or chain breaking" and include:

1. Preventive

- a. transferrin, ceruloplasmin, albumin--remove metal radicals in extracellular fluids

- b. superoxide dismutases with manganese, copper, and zinc as cofactors--remove intracellular superoxide (O₂⁻) and hydrogen peroxide (H₂O₂)
- c. glutathione peroxidase with selenium as cofactor--removes intracellular O₂⁻ and H₂O₂
- d. catalase with iron as cofactor--removes intracellular O₂⁻ and H₂O₂

2. Chain breaking

- a. vitamin E
- b. beta carotene
- c. ubiquinone
- d. ascorbate (vitamin C)
- e. urate
- f. GSH (reduced glutathione)

VITAMIN E (alpha-tocopherol)

Adequacy. Due to losses of vitamin E in forages that are mature and have been stored, it is likely that vitamin E intakes in many dairy herds are inadequate (Hogan et al., 1993).

Mastitis. Hogan, et al. (1993) have pointed out that plasma alpha-tocopherol tends to decrease seven to ten days before parturition and remains decreased for two to three weeks into lactation when animals receive a constant level of vitamin E in the ration. Subcutaneous injections of vitamin E (3000 IU vitamin E), at ten and five days before anticipated calving, successfully increased alpha-tocopherol concentrations in

plasma and neutrophils during late gestation and early lactation. Also cows receiving vitamin E injections during the dry period demonstrated greater neutrophil capabilities to kill bacteria at parturition (Hogan et al., 1993).

Overall, cattle supplemented with vitamin E and selenium demonstrate reduced rates of mastitis and a reduction in duration of clinical signs (Hogan et al., 1993). And dietary vitamin E and selenium reduced the prevalence of intramammary infection in first calf heifers (Smith et al., 1993).

Retained placenta. Supplemental vitamin E appears to be more effective in preventing retained placenta when selenium is also provided. Miller, et al. (1993) presented data that the incidence of retained placenta was consistently, and for the most part significantly, less than control animals when fed diets containing greater than 0.12 ppm selenium with or without 1000 IU vitamin E supplemented during the last 40 days of gestation.

Udder edema. Vitamin E (1000 IU per day) reduced the severity of udder edema when adequate (at least 0.12 ppm) selenium was also in the diet (Miller et al., 1993).

CONCLUSIONS ABOUT VITAMIN E

1. It is recommended that dry cows receive at least 1000 IU of supplemental vitamin E per day and lactating cows 500 IU per day while receiving diets containing 0.3 ppm selenium (Aseltine, 1991).
2. The overall antioxidant requirements may be greater for high performance dairy cows than generally accepted (Miller et al., 1993).
3. The form of vitamin E supplemented must be considered. According to Patton (1989) the natural acetate form in research trials is superior to the synthetic acetate form.

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