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Vit E. ability to delay lipid oxidation of tallow products

Vitamin E and Selenium for Ruminants

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Introduction

Selenium (Se) is an essential micronutrient present in tissues throughout the body and is important physiologically (Rotruck *et. al.*, 1973) as it is an integral component of the enzyme glutathione peroxidase (GSHpx). Tissue concentrations of Se are highly correlated with GSHpx activity and directly related to dietary intake (Scholz and Hutchinson, 1979). Plants grown on Se deficient soils do not provide adequate dietary Se. In general forages grown on poorly drained acid soils are Se deficient. Selenium deficient soils are geographically widespread in the United States (US) and Europe, and approximately two-thirds of the dairy cattle in the US are in areas of known Se deficient soils.

The term vitamin E, according to the International Union of Pure and Applied Chemistry -International Union of Biochemistry (IUPAC - IUB) Commission on Biomedical Nomenclature, is used as a generic descriptor for all tocol and tocotrienol derivatives which qualitatively exhibit the biologic activity of α-tocopherol (IUPAC-IUB, 1973). Both the tocols (tocopherols) and tocotrienols consist of hydroquinone nucleus and an isoprenoid side chain (Machlin, 1984). Characteristically, tocols have a saturated side chain, whereas the tocotrienols have an unsaturated side chain containing three double bonds. Four principal compounds of each of these two classes of vitamin E exist, namely α, β, γ and δ, differentiated by the presence of methyl ($-CH_3$) groups at positions 5, 7 or 8 of the chroman ring. Alpha-tocopherol, the most biologically active of these compounds is the predominant vitamin E active compound in feedstuffs while the biological activity of the other tocols is limited.

According to the United States Pharmacopeia (1980), all-rac α-tocopheryl acetate is the international standard of vitamin E activity with one International Unit (IU) equivalent to one milligram of all-rac-α-tocopheryl acetate. This is the most widely available source of vitamin E activity for supplementation of animal feeds. The acetate ester is very stable to *in vitro* oxidation and has no activity as an *in vitro* antioxidant; however, it is readily hydrolyzed in the animal gut to nonesterified or free tocopherol which is the potent *in vivo* antioxidant.

Forages are a major source of vitamin E in ruminant diets (Kirmae and Carpena, 1973; Schingoethe *et. al.*, 1978). High levels of vitamin E are found in fresh green forages, however, the vitamin E content of stored forages is decreased significantly and the decrease is progressive with length of storage. The ensiling process is particularly destructive to vitamin E and corn silage is notoriously low in vitamin E.

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lipid & pigment oxidation are related closely.

Selenium and vitamin E are essential for such body functions as growth, reproduction, prevention of various diseases, and protection of the integrity of tissues. The metabolic function of Se is closely linked to vitamin E. Both Se and vitamin E function to protect biological membranes from oxidative damage. Lack of these nutrients results in tissue breakdown and degeneration. It now appears that vitamin E in cellular and subcellular membranes is the first line of defense against peroxidation of vital phospholipids. Even with adequate vitamin E, however, some peroxides are formed. Selenium, as part of the enzyme GSHpx, is a second line of defense that destroys these peroxides before they have an opportunity to cause damage to membranes.

Comprehensive reviews of the functions and metabolism of vitamin E and Se are available (Combs and Combs, 1986; McDowell, 1989, 1991). The objective of this paper is to review current concepts of vitamin E and Se nutrition as well as available research data on the influence of vitamin E and Se supplementation of ruminants.

Importance of Vitamin E and Selenium

Vitamin E and the Se containing enzyme GSHph are an integral part of the antioxidant system present in all cells (Hoekstra, 1975; Arthur, 1985). Both are important for optimum cell function because they help maintain low cellular and tissue concentrations of reactive oxygen molecules and lipid hydroperoxides. During the metabolism of oxygen within cells large quantities of superoxide and hydrogen peroxide are produced and these reactive oxygen species can severely damage membrane lipids, DNA, cellular proteins and enzymes. The specific function of GSHpx is the conversion of hydrogen peroxide to water and lipid hydroperoxides to the corresponding alcohol. Vitamin E is a very efficient scavenger of both reactive oxygen species and lipid hydroperoxides, converting both to nonreactive forms (Putnam and Comben, 1987).

Vitamin E and GSHpx function at two different levels within the cell. GSHpx functions in the cytosol of the cell, while vitamin E is an integral component of lipid membranes (Hoekstra, 1975). Vitamin E protects the polyunsaturated fatty acids (PUFA), enzymes, and transport proteins located in membranes from reactive oxygen species. The PUFA are present in all cellular membranes, but the concentration varies considerably from tissue to tissue. Membrane PUFA are extremely susceptible to attack from reactive oxygen species and the higher the concentration of membrane PUFA, the more susceptible the cell and tissue are to oxidant damage (Rice and Kennedy, 1988).

An important PUFA in cellular membranes is arachidonic acid (AA). Arachidonic acid can be metabolized to prostaglandins, thromboxane, and prostacyclin by the enzyme complex cyclo-oxygenase and to the leukotrienes by the lipoxygenase enzyme complex. Evidence clearly suggests that AA metabolism is altered in the animals deficient in vitamin E, Se, or both (Rice and Kennedy, 1988). Glutathione peroxidase participates directly in AA metabolism and vitamin E may function to control peroxidation of AA or its unstable metabolites.

Immune Response and Disease Resistance

Levels of Se and vitamin E above generally accepted requirements have been shown to enhance the immune response against a wide variety of particulate and soluble antigens in several species (Tengerdy, 1980). Currently considerable attention

is being paid to the role of these nutrients in protecting leukocytes and macrophages during phagocytosis, the mechanism whereby mammals immunologically kill invading bacteria. Vitamin E and Se may help these cells to survive the toxic products that are produced in order to effectively kill ingested bacteria. In a number of mammalian species, Se deficiency has been associated with decreased immune system function. Decreased GSHpx activity in phagocytic cells has been reported in Se-deficient heifers (Boyne and Arthur, 1979). In bovine neutrophils, the bactericidal capacity for *Candida albicans* and *Staphylococcus aureus* is lowered in Se-deficient cattle (Boyne and Arthur, 1979). Inefficient immune cell function may predispose cattle to infectious diseases.

The effects of vitamin E supplementation on protection against infection by several types of pathogenic organisms, as well as antibody titers and phagocytosis of the pathogens in various species has been thoroughly reviewed. When animals are in a stressed or disease state, there is an increased production of glucocorticoids, epinephrine, eiecosanoids, as well as elevated phagocytic activity (Nockels, 1989), which leads to production of free radicals (reactive oxygen molecules) which challenge the animals antioxidant system. The protective effects of vitamin E (and possibly Se) on animal health may be involved with its role in reduction of glucocorticoids, which are known to be immunosuppressive (Golub and Gershwin, 1985). Vitamin E also most likely has an immunoenhancing effect by virtue of altering AA metabolism and subsequent synthesis of prostaglandin, thromboxanes and leukotrienes as previously mentioned. Under stress conditions increased levels of these compounds by endogenous synthesis or exogenous entry may adversely affect immune cell function (Hadden, 1987).

Nockels (1991) reported due to vitamin E deficiencies occurring in neonatal dairy calves, it is also of interest to determine α-tocopherol levels in the plasma of beef calves. Blood samples were obtained from calves prior to colostrum consumption, and for several days thereafter (Nockels, 1991). Precolostral plasma vitamin E levels averaged 0.02 mg/dl and increased to 0.33 mg/dl at 5 to 8 days of age. The importance of providing colostrum containing the vitamin is quite apparent, as the calf has low levels at birth, similar to results reported in swine (Mahan, 1992). This very low blood vitamin E level at birth reflects limited placental transfer of this nutrient (Whiting and Loosli, 1948; Parrish *et. al.*, 1950; Hidiroglou *et. al.*, 1969; Van Saun *et. al.*, 1989) which may lead to diminished disease resistance and immune response in the neonate (Nockels, 1991).

Reddy *et. al.* (1985, 1986b) conducted a study to determine the level of vitamin E and the route of administration that would correct a vitamin E deficiency and improve immunity of dairy calves. Holstein heifer calves were allotted to one of four treatments either 0, 1400 or 2800 IU vitamin E fed orally weekly, or 1400 IU vitamin E injected intramuscularly (i.m.) weekly. The calf starter contained 0.44 ppm of Se. Blood serum was collected periodically from birth until 12 weeks of age for the determination of α-tocopherol, lymphocyte stimulation indices to phytohemagglutinin (PHA), immunoglobulin levels, and replication of IBR virus. Additionally, creatine kinase (CK) was used as a positive diagnosis of preclinical nutritional muscular dystrophy (NMD). Serum α-tocopherol was increased through week 8 in calves injected with vitamin E relative to the other treatments. However, no differences in serum α-tocopherol occurred among the treated calves at weeks 10 or 12 when blood vitamin E levels decreased in calves receiving the vitamin E injection.

Lymphocyte stimulation to PHA was increased at 4 and 8 weeks in the calves injected with vitamin E (Reddy *et. al.*, 1986). Serum factors that would inhibit IBR viral replication were compared among the treatment calves at 12 weeks. Both the calves fed the 2800 IU of vitamin E and those injected with vitamin E had lower viral titers. Serum immunoglobulin M (IgM) was increased at week 6 by feeding the highest vitamin E level. When averaged over the 12 week period, serum CK was reduced in calves by either oral or injected vitamin E supplementation compared to controls. These authors point out that higher CK concentrations detected in the unsupplemented calves is a preclinical indication of NMD which was not prevented by Se alone, and that both vitamin E and Se are necessary for prevention of NMD. In an additional experiment, 2000 IU of vitamin E was injected into yearling heifers and the serum α-tocopherol and lymphocyte stimulation indices were increased 7 days later (Reddy *et. al.*, 1986).

Reddy *et. al.*, (1987 a,b) in subsequent experiments, fed either 0, 125, 250 or 500 IU of vitamin E daily to Holstein calves in milk for the first 8 weeks. Thereafter, vitamin E was fed in 0.45 kg of dry feed daily until the calves were 24 weeks old. The objectives of these experiments were to determine if supplemental vitamin E would affect growth, immunity, blood serum vitamin E, CK and glutamic oxaloacetic trasaminase (GOT) levels. Blood sampling was periodic from birth to 24 weeks of age and serum vitamin E, CK, GOT, lymphocyte stimulation indices to different mitogens, primary and secondary antibody titers development to antibovine herpes virus type 1, and cortisol levels were determined. Vitamin E improved several immune responses in the calves. Overall weight gains were improved by vitamin E (125 and 250 IU vitamin E/d) supplementation. Serum glutamic oxalacetic transaminase and cortisol were decreased by 125 IU of vitamin E/day, while CK was unaffected. Supplementation of vitamin E increased serum α-tocopherol relative to controls. Serum tocopherol levels less than 0.2 mg/dl, as well as enzyme levels indicated a deficiency throughout the entire study for control calves. Incremental increases in vitamin E supplementation further increased serum vitamin E levels until 4 to 6 weeks of age when it peaked and then declined (Reddy *et. al.*, 1987a).

An increased incidence and severity of mastitis has been associated with Se deficiency and/or vitamin E deficiency (Smith *et. al.*, 1984; Erskine *et. al.*, 1989; Hogan *et. al.*, 1990; Weiss *et. al.*, 1990).

The influence of vitamin E and Se on mastitis was first reported by Smith *et. al.* (1984). Diets of multiparous dairy cows were supplemented with either 0 or 1000 IU vitamin E during the dry period. Cows were additionally administered Se at the rate of 0 or .1 mg/kg body weight via i.m. injection 21 days prepartum. No vitamin E or Se were supplemented in the diet during lactation. Incidence of new clinical cases of mastitis was reduced by 37% in both groups receiving vitamin E compared to control. The reduction in clinical mastitis was only 12% when cows were injected with Se but not supplemented with dietary vitamin E. These authors also reported that clinical cases of mastitis in the vitamin E supplemented-Se injected cows were consistently of shorter duration than those occurring in all other groups. Similar results have been recently reported by Finkelstein *et. al.* (1992) with multiparous Holstein cows supplemented with vitamin E and/or Se.

Growth and Performance

In a series of 28 day feedlot receiving trials, Lee *et. al.* (1985) observed an improvement in early performance of newly arrived growing cattle (250 kg)

supplemented with 450 IU vitamin E per head per day that were stressed by long distance shipment and changes in diet from green forages to high grain feedlot rations. Depression of circulating cortisol concentrations may explain the improved gain and feed efficiency in this trial. Carrica *et. al.* (1986) reported no performance response in heavy weight steers (366 kg) entering the feedlot when supplemented with 200 IU vitamin E per head per day. These authors concluded that vitamin E supplementation was more critical in lightweight calves or cattle that have undergone duress due to shipment and handling.

Hutcheson and Cole (1985) reported increased average daily gains in yearling cattle supplemented with either 100 or 300 IU vitamin E per head per day and .10 mg/kg Se. Plasma α-tocopherol levels were not influenced by vitamin E supplementation up to 300 IU vitamin E per head daily. Increasing body weight, altered absorptive efficiency or possibly ruminal destruction may partially explain these response, suggesting that supplementation should be increased with increased age and recommendations should be based on a body weight basis.

Gill *et. al.* (1986) supplemented newly received feedlot cattle with 1600 IU vitamin E per head per day for the first 21 days and 800 IU vitamin E for the remaining 7 days of a 28 day trial. Average daily gain and gain to feed ratios were improved by 23.2 and 28.6%, respectively, for vitamin E supplemented stressed cattle. The number of sick pen days per head was reduced by 15.6% and morbidity was reduced by 13.4% with vitamin E supplementation. The growth response to vitamin E could be related to the fact that young, rapidly growing animals are in a metabolically demanding state resulting from overall tissue growth which has a high energy demand. Vitamin E is an integral part of this response via its ability to quench free radicals which are generated during the course of metabolism.

Three 28 day feedlot receiving trials were conducted (May *et. al.*, 1987) to evaluate the effect of vitamin E supplementation on performance of steers and heifers. In trial one, 230 heifers were injected intramuscularly (i.m.) with either 0, 1250 or 2500 IU of vitamin E at processing; 192 steers in trial two were injected i.m. either 0 or 1250 IU vitamin E on day 0 and received 800 IU vitamin E orally per head per day; 360 steers in trial three received orally either 0 or 800 IU vitamin E per head per day. Animals in trials one and two moved from wheat pasture to the feedlot and those utilized in trial three were moved from native grass pastures. An initial single injection of vitamin E had no effect of 28 day performance in all three trials. Plasma alpha-tocopherol concentration at 28 days was not influenced by injection but was by oral vitamin E supplementation in both trials two and three.

Two trials were conducted with newly arrived feedlot cattle (Hill, 1987) to evaluate the effects of oral vitamin E supplementation on growth and performance. Steers were subjected to mild stress associated with abruptly moving animals from pasture to a slatted floor feedlot. In trial one, steers received either 0 or 200 IU supplemental vitamin E per head per day for 130 days. Steers in trials two received either 0, 500 or 1000 IU supplemental vitamin E daily for 84 days. Vitamin E supplementation had no effect on average daily gain or feed conversion in trial one, however, both parameter were improved in trial 2 during the first 28 days regardless of vitamin E supplementation level as compared to controls. Both vitamin E treatments in trial two tended to improve average daily gain throughout the trial. Vitamin E supplementation in trial one resulted in higher plasma α-tocopherol concentration at 56 and 112 days than controls, however,

concentration decreased over time across all treatments. The decrease in plasma α-tocopherol concentration may be related to change in diet (i.e., fresh forage, based to high gain) and depletion of body stores of vitamin E as the trial progressed. This is similar to the results obtained by May *et. al* (1987).

Hill *et. al.*(1989) subsequently reported the results of two additional feedlot trials which were conducted to determine the effects of vitamin E supplementation (1000 IU per head per day) on feedlot performance, carcass traits and the, incidence of liver abscesses. Steers were fed for 133 days in trial 1 and 126 days in trial 2. All diets were supplemented with 0.1 mg Se per kg. Oral supplementation of 1000 IU of vitamin E per head daily tended to improve feedlot steer performance in both trials, average daily gain increased 4.1 and 8.7% in trial 1 and 2, respectively; feed efficiency was improved 7.2 and 6.9% in trial 1 and 2, respectively. Plasma α-tocopherol concentrations were greatest for vitamin E supplemented steers. Vitamin E supplementation reduced the incidence of liver abscesses observed in trial 1 from 41.4 to 21.4%.

No improvement in average daily gain or incidence of liver abscesses was observed in steer calves fed 500 or 1000 IU supplemental vitamin E per head daily in a 113 day finishing trial (Nader, 1987). Feed efficiency was improved over the entire feeding period for steers receiving 500 IU vitamin E per head daily compared to controls. No difference in any carcass traits were observed between vitamin E treatment groups.

Two trials were conducted in Michigan (Metz and Rust, 1989) to evaluate supplementation of three levels of vitamin E (0, 500 or 1000 IU per head per day) to growing-finishing heifers. In both trials, cattle were fed corn silage diets without supplemental vitamin E for 100 days prior to trail initiation. Forty-five Maine Anjou crossbred heifers were fed a diet consisting of high moisture corn, corn silage and protein supplement for 140 days in trial 1 while 63 Hereford X Angus heifers were fed similarly in trial 2. Heifers were slaughtered at approximately 75% choice quality grade. No difference in average daily gain or feed efficiency were observed between the treatment groups. The incidence of liver abscesses was not different between treatment groups, however, with trail 2, the severity of liver abscesses was lower for cattle fed the highest level of vitamin E as compared to the intermediate group.

Brandt (1987) similarly reported no difference for 57- or 127-day steer feedlot performance as influenced by supplementation of 0, 500 or 1000 IU per head daily of vitamin E. Eighty-four Brahman crossbred steers were utilized in this 127 day finishing trial. Additionally, there were no effects of vitamin E supplementation on carcass traits or on the incidence and severity of liver abscesses.

Droke and Loerch (1989) conducted five trials with newly arrived feedlot steers to determine the effects of one or two i.m. injections of Se and (or) vitamin E on performance, health status and serum antibody response to *Pasteurella hemolytica* vaccination. Steers treated with 25 mg Se, 340 IU vitamin E, or 25 mg Se plus 340 IU vitamin E relative to controls after arrival, or the combination of Se and vitamin E 14 days prior to shipment and again on arrival showed no improvement in health or performance compared to controls. Serum IgG antibody titers to *P. hemolytica* vaccination was enhanced with the combination of Se and vitamin E.

In a follow-up study (Smith and Conrad, 1987), 55 first lactation Holstein and Jersey heifers were assigned to received either vitamin E and Se supplementation or to an unsupplemented control group 60 days prior to parturition. Supplemented heifers received prepartum, 2 IU vitamin E and 2 μg Se per kg body weight per day. These heifers were also injected subcutaneously (s.c.) 21 days prepartum with .1 mg Se per kg body weight. During lactation the supplemented group received 88 IU vitamin E and .3 mg Se per kg of concentrate.

Regardless of treatment group, plasma α-tocopherol concentration decreased from day 60 prepartum to calving although greater for unsupplemented heifers. The high initial plasma α-tocopherol values reported were most likely associated with the fact that the majority of heifers were on pasture up to this time. The overall plasma α-tocopherol is similar to that reported for gravid females of various species. Weiss *et. al.* (1990) reported that plasma α-tocopherol concentrations are essentially consistent from drying off until 7 days prepartum, then drop by about 50% and remain low until 20 to 30 days prepartum, returning to baseline levels of 60 days postpartum. Weiss *et. al.* (1990), speculate that this profile may be related to differences in absorptive efficiency for vitamin E between dry and lactating cows, the demand for vitamin E in milk and conceptus as well as differences in feed intake between dry and lactating cows. Weiss *et. al.* (1990) reported that clinical mastitis was negatively related to plasma Se concentration and concentration of vitamin E in the diet.

Smith and Conrad (1987) reported that intrammary infection was reduced 4.2% in vitamin E-selenium supplemented versus unsupplemented controls. The duration of all intrammary infections in lactation was reduced 40 to 50% in supplemented heifers. Lactation average mean somatic cell count was lower in supplemented heifers and there was a 68% reduction in heifers with lactation average mean somatic cell counts greater than 200,000 cells per ml. Clinical mastitis during the first 4 days of lactation was reduced by 57% in supplemented heifers. Incidence of clinical cases of mastitis throughout lactation was reduced by 32% in supplemented heifers compared to controls. The specific mode of action of vitamin E in enhancing mammary gland health remains to be elucidated, however, possible mechanisms may involve improvement in phagocytic cell function, improved gland immunocompetence as well as altered AA metabolism.

Studies have been conducted to evaluate the effects of vitamin E and Se on the incidence of retained placenta in dairy cows. Cows injected with vitamin E and Se at one month (Trinder *et. al.*, 1973) or 20 days (Julien and Conrad, 1976) prior to calving had lower incidence of retained placenta than cows not supplemented with these nutrients. Other work (Harrison *et. al.*, 1984) supplemented a practical diet based on legume-grass haylage with 1000 IU vitamin E per day per animal, beginning 21 days prepartum and used Se injection .1 mg/kg body weight 21 days before projected calving. This work indicated that while vitamin E and Se alone had no effect on the incidence of retained placenta, the combination of the two nutrients proved to be very effective. More recent research (Thomas *et. al.*, 1992) reported a lower incidence of retained placenta in cows which received 3 mg Se and 1000 IU vitamin E per head daily from 6 weeks prior to calving until parturition.

Other studies (Shigemoto *et. al.*, 1980; Segerson and Riviera, 1981; Schingoethe *et. al.*, 1982; Ishak *et. al.*, 1983 and Eger *et. al.*, 1985) in which the effects of selenium and vitamin E on the occurrence of retained placenta in cows was evaluated have been inconclusive but do suggest that supplemental level, timing

of administration, and the interaction of vitamin E and Se with beta-carotene, vitamin A and other nutrients needs to be further defined.

Stabilizing Effect of Vitamin E on Beef Color and Lipids

The visual appearance of any food product is an important sensory property by which consumers judge product quality (Cassens *et. al.*, 1988). Consumers have demonstrated that the color of fresh meat is bright-red or bright-pink and that any deviation from this is unacceptable. This is especially true in red-meat cuts were surface discoloration may be interpreted as unwholesomeness (Faustman and Cassens, 1989). Hood (1990) likewise indicated that fresh meat color continues to be of major interest to meat industry, especially the retail segment where a strong consumer preference persists for a bright red color.

The heme-containing proteins, hemoglobin and myoglobin are the primary pigments in muscle tissue associated with color. Myoglobin is the pigment mainly responsible for meat color since most hemoglobin is removed when the animal is exsanguinated. Myoglobin is located in muscle fibers and contributes some oxygen carrying capacity *in vivo*. The basic color problem associated with fresh beef is due to the inherently high oxidation potential of the reduced, deoxygenated myoglobin. The brown oxidized form, metmyoglobin, is relatively stable in air and gradually accumulates at the expense of the attractive red oxymyoglobin.

Lipid oxidation and pigment oxidation in fresh meat have been reported to be closely coupled (Greene, 1971; Faustman *et. al.*, 1989a) suggesting that delaying the breakdown of lipid may result in similar delay of meat discoloration. It is reasonable that any procedure which may enhance lipid stability and stabilize beef color, such as increasing the endogenous α-tocopherol content of beef tissues, may prolong product shelf-life and favorably impact its economic value and image in the marketplace.

A recent retail beef industry study (Booz-Allen and Hamilton, Inc., 1989) estimated that value deterioration of meat, expressed as a percent of the required retail gross margin, is on the order of 3.7% for the entire meat department and 5.4% for fresh meat. This loss in value consists of product price markdown, product conversion and rework, product discard, lowered stock inventory and inefficient use of labor (C. R. Bergh, 1992, personal communication). Value deterioration of fresh meat results from a number of different causes, however, loss of fresh beef "bloom" (i.e., the bright red color) is widely recognized as the primary cause of value deterioration in beef, the result of muscle pigment oxidation (Faustman *et. al.*, 1989b). Schaefer *et. al.* (1991) reported that increasing color shelf-life of beef by two days would save $175 million annually in retail beef sales. Other estimates have suggested a potential savings of up to $1 billion to the beef industry by increasing shelf-life of beef by one to two days (Hill *et. al.*, 1992).

To date, much research work in the area of vitamin E and beef quality has been performed at the University of Wisconsin-Madison. Both Holstein and crossbred beef steer calves of various weights and ages were utilized in these experiments and a variety of supplemental vitamin E regimens studied (Table 1).

The basal diet in all trials consisted of 10% corn silage and 90% concentrate (dry matter basis). The concentrate portion of the diet was composed of 74.2% high-moisture corn, 8.8% soybean meal, 3.5% vitamin and mineral supplements and

3.5% vitamin E supplement. The basal diet was formulated to contain 0.1 ppm Se. The vitamin E content of the basal diet was ca. 9 IU per kg (as-fed basis). Cattle were transported to a commercial slaughter plant and immediately slaughtered. After 24 hours of chilling, carcasses were quality and yield graded. Samples of strip-loin and top-sirloin butt were obtained, vacuum packaged and aged at 4°C for up to 21 days. Loin and sirloin steaks were sliced, placed on styrofoam trays, over-wrapped with PVC film and displayed under continuous cool-white fluorescent illumination at 2.5°C - 4°C to simulate retail display conditions. Meat color was monitored both subjectively by five trained panelists who evaluated both degree of redness and percentage of muscle surface areas which had discolored and objectively by reflectance spectroscopy to determine the percentage of total myoglobin present as metmyoglobin at various time intervals. Thiobartitric acid-reactive products (TBA), an index of lipid peroxidation, and α-tocopherol concentration were determined on each meat sample.

Comparisons of tissue α-tocopherol concentrations for liver, subcutaneous fat, sirloin and plasma over all trials are presented in (Table 2). Both length of time and level of vitamin E supplementation impacted tissue concentration of α-tocopherol as compared to animals receiving only basal diet. The accretion of vitamin E varied according to tissue, with liver showing the greatest increases followed by subcutaneous fat, sirloin and plasma.

Loin α-tocopherol concentrations were similar to those of sirloin α-tocopherol concentrations. The concentration of α-tocopherol in loin was increased for each increment of vitamin E supplementation. No differences were observed between Holstein and crossbred beef steers in loin α-tocopherol concentration in these studies. The increased loin concentrations of α-tocopherol are consistent with the observed responses in color shelf-life extension (Table 3). Vitamin E supplementation, regardless of level or length extended the color shelf-life of loin (or sirloin, data not presented here: see Faustman *et. al.*, 1989a; Faustman *et. al.*, 1989b) steaks aged 7 or 21 days in both Holstein and crossbred beef steers (Hoffmann-La Roche, 1991b).

Color shelf-life of loin steaks from vitamin E supplemented cattle was extended from 2 to 5 days. Metmyoglobin formation was reduced and delayed in all beef from vitamin E supplemented cattle compared to controls. Lipid oxidation was markedly inhibited in meat from vitamin E supplemented cattle (Hoffmann-La Roche 1991b, Schaefer *et. al.*, 1991; Liu *et. al.*, 1992a,b). Faustman *et. al.* (1989a) reported that metmyoglobin accumulation in fresh ground sirloin patties stored at 4°C for up to six days was greater for control animals than those supplemented with vitamin E. The oxidative stability of ground beef patties during refrigerated or frozen storage was also enhanced by vitamin E supplementation (Faustman *et. al.*, 1989a). These authors reported that α-tocopherol concentrations in excess of 0.3 mg/100 g meat (fresh basis) were necessary for reducing metmyoglobin or TBA accumulation in ground sirloin. These relationships have been further expanded by Arnold *et. al.* (1992). It would appear that the critical factor in using vitamin E supplementation to extend the shelf-life of beef is implementing a supplementation regimen which will achieve a minimum muscle tissue α-tocopherol concentration level of ca. 0.30 to 0.35 mg/100 g meat (fresh basis). The protective of muscle tissue α-tocopherol may be saturable (Arnold *et. al.*, 1992). Meat cuts which contain α-tocopherol in excess of 0.30 mg/100 g (fresh basis) do not appear to have any benefit in terms of reduced myoglobin accumulation or lipid oxidation (Faustman *et. al.*, 1989a). It would appear that supplementation of 500 IU vitamin E per head daily for 84 to 126 days

would yield tissue α-tocopherol concentrations of this magnitude (Schaefer *et. al.*, 1991). However, Schaefer (1992, personal communication) suggests that meat from cattle stored for prolonged periods (i.e., 56 days or longer) may benefit from increase tissue α-tocopherol concentrations in terms of reduced accumulation of metmyoglobin and TBA reactive substances.

Vitamin E supplementation did not alter microbial growth on fresh beef (Schaefer *et. al.*, 1991). Also, no differences in microbial growth were observed in beef Holstein or crossbred beef steers (Hoffmann-La Roche 1991b). These findings would suggest that an increased vitamin E concentration in beef muscle, does not mask any product unwholesomeness. Garber *et. al.* (1992) have also reported that microbial populations in gluteus medius steaks were not affected by vitamin E supplementation which are consistent with the Wisconsin results. Results of research trials conducted at the University of Wisconsin-Madison indicate that supplemental vitamin E is effective in stabilizing pigment and lipid components of beef thus extending color shelf-life of beef displayed under simulated retail conditions.

Similar results have been reported by other investigators. Hill *et. al.* (1991) observed that color retention scores were greater in rib steaks commercially packaged and stored over a 4 day period at 2.5°C from crossbred beef steers supplemented with 1000 IU vitamin E per head daily for 126 days. In a series of follow-up studies, Hill *et. al.* (1992) indicated that rib steaks from steers fed 1000 IU vitamin E per head daily for 126 days had greater color retention than unsupplemented controls. Additionally, these authors (Hill *et. al.*, 1992) reported that ground beef prepared from rib sections of steers supplemented with vitamin E had less discoloration after five days of storage as well as lower TBA values at ten days of storage.

Comstock *et. al.* (1991) conducted a study to determine effects of supplemental dietary or injectable vitamin E on fresh beef storage properties and shelf-life. Feedlot steers were provided 2000 IU vitamin E per head daily for 28 days prior to slaughter of 4000 IU vitamin E injected s.c. 24, 48 or 72 hours prior to slaughter. Gluteus medius muscle from supplemented steers tended to have reduced metmyoglobin accumulation and TBA values and improved Hunter 'a' values (redness). These authors provided no data on tissue α-tocopherol content. The somewhat diminished response observed by these researchers (Comstock *et. al.*, 1991) compared to results reported by Wisconsin workers may be due to an inadequate period of vitamin E supplementation whereby tissue α-tocopherol pools did not attain the necessary 'minimum' concentration to elicit the maximal response. In a more recent study conducted at Idaho (Garber *et. al.*, 1992), crossbred beef steers were fed 0, 250, 500, 1000 or 2000 IU vitamin E per head daily for ca. 126 days. These authors reported vitamin E supplementation delayed both metmyoglobin formation and lipid oxidation in a linear fashion and extended the color shelf-life of fresh sirloin steaks.

Based on university research results previously presented, a blind retail study (Williams *et. al.*, 1992) was conducted to provide information to assess the feasibility of feeding cattle vitamin E to reduce economic losses associated with discoloration of fresh beef. Holstein steers, weighing approximately 478 kg were supplemented with 500 IU of vitamin E for the last 100 to 120 days of the feeding period. All steers were fed whole corn with a protein supplement which contained vitamin E. Steers weighing 614 kg were slaughtered in a commercial slaughter facility.

Beef from these steers, along with beef derived from Holstein steers not receiving supplemental vitamin E was shipped to Las Vegas, NV; Lenexa, KS; and Houston, TX where meat products were processed and displayed for retail sale over a four-day period from Tuesday to Friday. Six meat cuts were evaluated in this test, namely strip-loin, t-bone, tenderloin, top-round and top-sirloin steaks as well as shoulder roasts. All meat products were coded; store employees were unaware of the purpose of comparing the two sets of meat cuts. All beef products were monitored for discoloration and any associated price discounts were recorded throughout the display period.

It should be noted that meat shipped to three locations differed in the length of aging period, thus pooling these data is inappropriate. Meat was aged for 7, 14 or 29 days when received at the Las Vegas, Lenexa and Houston locations, respectively. Both pounds available and total retail value of product for sale were similar for all locations. Both pounds of product as well as the value of product reduced in price due to discoloration were significantly lower in beef from vitamin E supplemented cattle (Table 6). This result was consistent across all locations studied and similar to results from the university studies. It should be noted, that when beef was discounted due to discoloration from either control of vitamin E supplemented animals, the value loss of discounted product was similar (i.e., $.80 and .85 per pound for control and vitamin E supplemented beef, respectively), indicating no discount bias between the test products.

Assessment of Vitamin E and Selenium Status

Confirmation of a low Se and/or vitamin E status in animals is obtained when specific deficiency diseases for various species associated with lack of these nutrients are present. Likewise, gross lesions and histopathological examinations provide definite evidence of selenium and/or vitamin E deficiency.

Muscular damage as a result of Se and/or vitamin E deficiencies causes leakage of intercellular contents into the blood. Thus, elevated levels of selected enzymes, above normal concentrations for particular species, serve as diagnostic aids in detecting tissue degeneration. Serum enzyme concentrations used to follow incidence of NMD include serum glutamic-oxalacetic-transaminase (SGOT), aspartate amino transferase (ASPAT), lactic dehydrogenase (LDH), creatine phosphokinase (CPK) and malic dehydrogenase (MDH). Enzyme tests are very sensitive, and an elevation of enzyme activity in serum is usually discovered before any pathological changes or clinical signs appear.

Low tissue concentration of the Se dependent enzyme, GSHpx, is a relatively good status indicator of this element. Liver and plasma GSHpx activities increase or decrease rapidly during repletion or depletion of Se, therefore, concentrations of the enzyme serve as a relatively accurate indicator of the adequacy of dietary Se. GSHpx activity in serum has been a suggested assay for selenium status of cattle; however, the small amounts present and enzyme stability limit its usefulness (Van Saun, 1990).

It is debated as to which assay is best in accurately predicting Se status or potential presence of Se deficiency disease. If an animal is maintained on a consistent selenium intake over a period of months, all tests will be of equal accuracy in defining selenium status. However, if an animal receives different levels of dietary Se, interpretation of the animal's Se status may differ based on the assay used.

Serum or plasma Se is considered a good status indicator, with less than 0.03 to 0.04 ppm considered critical for cattle (McDowell, 1985). However, more recently Swecker *et. al.* (1989) observed that blood selenium concentrations of at least 100 μg per liter were associated with optimal antibody production.

Nutritional status with respect to vitamin E is commonly estimated from plasma (or serum) concentration. There is a relatively high correlation between plasma and liver levels of α-tocopherol (and also between amount of dietary α-tocopherol administered and plasma levels). Plasma tocopherol concentrations of 0.5-1 μg/ml are considered low in most animal species, with less than 0.5 μg/ml generally considered indicative of a vitamin E deficiency. Adams (1982) reported that plasma tocopherol concentrations between .60 to 1.6 μg/ml were associated with calves diagnosed with NMD. Serum α-tocopherol concentrations of 1.0 to 1.5 μg/ml were reported by McMurray and Rice (1982) and values <2 μg/ml considered deficient. Serum α-tocopherol concentrations >4.0 μg/ml have been considered to indicate adequacy in adult cattle. Similarly, marginal vitamin E status in adult cattle was associated with plasma tocopherol concentrations between 2.0 to 3.0 μg/ml (Adams, 1982).

The use of blood serum (or plasma) α-tocopherol concentration as an indicator or an animal's vitamin E status should be critically interpreted in each individual case. The use of this parameter as an indicator of status is related to the ease in obtaining the sample tissue as well as the fact that vitamin E is not stored in appreciable concentrations in the body. However, blood serum (or plasma) α-tocopherol concentration is most likely reflective of recent dietary intake of vitamin E. Plasma vitamin E concentrations may have limited value for diagnosing vitamin E deficiency. Red blood cell or platelet concentration of vitamin E may more accurately reflect status of the vitamin (Nockels, 1989).

Confirmation of a low Se and/or vitamin E status in animals is obtained when specific deficiency diseases for various species associated with lack of these nutrients are present. Likewise, gross lesions and histopathological examinations provide definite evidence of selenium and/or vitamin E deficiency.

Requirements and Supplementation

Review of the literature would suggest the selenium requirement for most species of livestock is between 0.05 to 0.3 ppm (mg/kg) of dietary dry matter (McDowell, 1992). This range is given as a result of many factors which may influence an animal's selenium requirements and diverse opinions as to criteria used to establish a requirement. Factors such as selenium source, bioavailability, vitamin E status, dietary concentrations of antagonistic nutrients (especially sulfur, iron and copper), and methods used to assess status and response can all influence the selenium requirement.

Dietary vitamin E requirements of young beef and dairy calves have not been clearly defined as evidenced by a requirement range of 15 to 60 IU per kg diet dry matter (NRC, 1984; NRC, 1988) with no requirement defined for adult cattle. While cattle grazing green forages generally exhibit no signs of vitamin E deficiency (May *et. al.*, 1987 Stuart, 1987), feedlot cattle fed high grain diets which are low in vitamin E, often have depressed serum α-tocopherol concentrations suggesting subclinical vitamin E deficiency (Adams, 1982; Hill *et. al.*, 1991). Additionally, cattle consuming drought stricken or dormant pasture, hay, haylage or silage during winter months may be at risk (Schingoethe *et. al.*,

1978; Lynch, 1983; McDowell, 1989. Data in Tables 4 and 5 depict vitamin E concentrations in the various forages and 'typical' milo- and corn-based feedlot rations, respectively. Assuming a 227 kg steer consumed 5.9 kg forage dry matter per day, theoretical vitamin E intake would range from ca. 1000 to 4000 IU per day depending on forage type ingested. In contrast, growing-finishing steers weighing 350 kg consuming 8 kg of diet dry matter and gaining 1.3 kg per day would consume ca. 123 or 147 IU vitamin E per day, respectively, from milo- or corn-based theoretical feedlot rations. Generally, cattle on pasture far exceed NRC (1984; 1989) guidelines, whereas, feedlot cattle tend to consume far less vitamin E from natural feed ingredients.

Factors of primary importance to influence Se and vitamin E supplementation include: (1) Se and/or vitamin E deficient concentrates and roughages; (2) excessively dry ranges of pastures for grazing livestock; (3) confinement feeding where Se and vitamin E adequate forages are not included or only forages of poor quality are provided; (4) diets that predominate in ingredients that contain low levels of α-tocopherol and thereby less biologically active; (5) diets that include ingredients which increase Se (i.e., heavy metals, aflatoxins, etc.) and vitamin E requirements (i.e., unsaturated fats, waters high in nitrates, etc.); (6) harvesting, drying, or storage conditions of feeds that have resulted in destruction of Se and/or vitamin E; (7) accelerated rates of gain, production nd feed efficiency may increase metabolic demands for Se and vitamin E; and (8) intensified production may also indirectly increase Se and vitamin E needs of animals by elevating stress, which often increases susceptibility to various disease (McDowell, 1989, 1991).

The efficacy of vitamin E supplementation in ruminant animals is no longer questioned. The ability of this essential nutrient to affect animals growth, health and reproductive capabilities are well documented. The requirements for supplementation of vitamin E to ruminants depend on many factors that have been described in this bulletin. These factors must be considered when a vitamin E supplementation program is implemented as it is critical to assure proper vitamin E status in the animal. Based on currently available data, a vitamin E supplementation program utilizing both parenteral and oral administration is suggested. For continued protection of all cattle and sheep from a possible vitamin E deficiency, adequate supplemental vitamin E in the feed is essential.

For feedlot animals and dairy cows that receive concentrates, the most efficient method of providing supplemental Se, is through use of Se-containing mineral supplements that are combined with concentrate feeds. Typically, these complete diets would be formulated to contain 0.1 to 0.3 ppm of supplemental Se. The principal methods of increasing intake by grazing livestock include 1) a free-choice mineral supplement, 2) fertilization, 3) injection, 4) drench, and 5)

ruminal pellets (heavy boluses) (McDowell, 1991). A recent advance has been the development of an intraruminal osmotic pump, which actively disperses Se at a rate of 3 mg per day (Campbell *et. al.*, 1990).

In areas where soils are low in Se, certain agricultural practices may have some effect in increasing the level available. Applying manure to low-Se soils from animals fed imported Se-adequate feeds increases the soil Se content slightly. Liming some soils deficient in Se results in only a very small increase in selenium uptake by plants. The direct approach to increasing intake by animals through use of a Se-containing fertilizer or spraying of the foliage with Se

compounds was not until recently widely practiced because the added Se is poorly absorbed by most plants, especially from acid soils (Underwood, 1981). Supplementation by these procedures does upon occasion pose a toxic hazard. Nevertheless, with proper precautions to minimize pasture contamination, 1 to 2 ozs/acre (as sodium selenite) should present no hazard. Fertilizer application with Se to crops in New Zealand and Finland has increased selenium in meat by-products (Rimmer *et. al.*, 1990; Ekholum *et. al.*, 1990).

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Table 1. Effects of vitamin E supplementation on meat quality. Summary of Trial Descriptions at the University of Wisconsin-Madison[a].

Study No.	Trial Description[b]	Vitamin E Treatments	Net Vitamin E Dose[c] (IU/d)
1	108 Holstein steers - 110 kg, 300 d	0 or 500 IU/hd/d	300
2	8 Crossbred beef steers - 386 kg, 67 d	0 or 2000 IU/hd/d	1140
3	66 Holstein & crossbred beef steers - 182 kg, 270 d	0, 500 or 2000 IU/hd/d	360,1280
4	24 Holstein & crossbred beef steers - 182 kg, 270 d	0, 5.7 or 8.6 IU/kg BW,2000 IU/hd/d	2080, 3520, 1300
5	10 Holstein steers - 622 kg, 38 d	0 or 2000 IU/hd/d	1200
6	72 Holstein steers - 418 kg, 42 or 126 d	0, 250, 500 or 2000 IU/hd/d	232, 486, 2109
7	54 Holstein steers - 415 kg, 126 d	0, 250 or 500 IU/hd/d	194, 402

[a]From Hoffmann-La Roche (1991b), Schaefer et al. (1991), Arnold et al. (1992) and Liu et al. (1992a,b).
[b]Trial description indicates number an type of animals, initial weight and length of time on vitamin E treatment prior to slaughter. [c]Total vitamin E intake minus vitamin E content of feedstuffs.

Table 2. Effects of vitamin supplementation on meat quality. Comparison of tissue α-tocopherol[a].

Study No.	Net Vitamin E Dose[b] (IU/d)	Tissue α-tocopherol concentration[c]			
		Liver	Subcutaneous Fat	Sirloin	Plasma
Basal	0	2.9	2.7	1.8	1.9
1	300	-	-	4.4	2.9
2	1140	23.8	9.0	6.3	6.4
3	360	12.0	9.5	5.3	4.9
	1280	25.2	19.6	8.6	7.9
4	2080	24.9	19.0	8.2	7.6
	3520	31.2	22.5	-	7.7
5	1200	12.7	6.8	4.8	4.5
6[d]	232	-	-	1.29,2.01	0.94,1.20
	486	-	-	2.08,3.22	1.52,2.03
	2109	-	-	4.23,7.33	3.30,4.02
7	194	-	-	2.11	1.55
	402	-	-	3.90	2.43

[a]From Hoffmann-La Roche (1991b), Schaefer et al. (1991), Arnold et al. (1992) and Liu et al. (1992a,b).
[b]Total vitamin E intake minus vitamin E content of feedstuffs.
[c]Liver, subcutaneous fat and sirloin (μg/g, fresh); plasma (μg/ml).
[d]Tissue α-tocopherol concentrations as follows: left number = 42 days, right number = 126 days on respective vitamin E treatment.

Table 3. Effects of vitamin E supplementation on meat quality. Comparison of loin color stability - University of Wisconsin-Madison[a].

Study No.	Net Vitamin E Dose[b] (IU/d)	Loin α-tocopherol concentration (μg/g, fresh)	Extended Display Shelf Life[c] (days)
Basal	0	1.4	0
1	300	3.8	5.3
2	1140	6.2	2.0
3	360	4.1	2.5
	1280	6.8	4.0
4	2080	6.7	3.1
	3520	7.6	5.2
5	1200	3.5	4.8
6[d]	232	0.93, 1.49	1.1, 1.7
	486	1.34, 2.49	0.89, 2.34
	2109	2.93, 5.51	2.45, 4.12
7	194	1.67	0.44
	402	2.88	2.04

[a]From Hoffmann-La Roche (1991b), Schaefer et al. (1991), Arnold et al. (1992) and Liu et al. (1992a,b).
[b]Total vitamin E intake minus vitamin E content of feedstuffs.
[c]Beef from vitamin E supplemented cattle compared to beef from cattle receiving the basal diet.
[d]Tissue α-tocopherol concentration as follows: left number = 42 days, right number = 126 days on respective vitamin E treatment.

Table 4. Vitamin E Content of several fresh forages and estimated vitamin E intake of a 227 227 kg beef animal consuming 5.9 kg forage DM per day.

Item	IFN Number	Vitamin E Content[a] mg/kg DM	Vitamin E Content[a] IU/kg DM	Vitamin E Intake (IU/hd/d)
Fescue (fresh)	2-01-920	143	213	1,257
Orchardgrass (fresh)	2-03-451	436	649	3,832
Ryegrass, perennial (fresh)	2-04-086	178	265	1,564
Timothy (fresh)	2-04-912	110	165	975
Wheat (fresh)[b]	2-08-078	141	210	1,239

[a]From NRC (1982).
[b]From Hoffmann-La Roche (1988, unpublished data; SNW IOM 28-88).

Table 5. Estimated daily vitamin E intake of a 350 kg growing-finishing steer consuming a milo or corn based ration[a].

Ingredient	Lbs	Vitamin E Content[b] (IU)
Milo, Rolled	1,470	5,439
Alfalfa	200	7,160
Fat	40	156
Supplement[c]	80	1,255
Water	210	-
Total	2,000	14,010
Vitamin E (IU/lb)	-	7.01
Vitamin E Intake (IU/hd/d)	-	**123**
Corn, Rolled	1,614	10,007
Molasses	70	256[b]
Alfalfa Hay	180	6,444
Supplement	106	-
Sodium Bentonite	30	-
Total	2,000	16,707
Vitamin E (IU/lb)	-	8.35
Vitamin Intake[c] (IU/hd/d)	-	**147**

[a]Estimated from NRC (1976); 350 kg growing steers gaining 1.3 kg lb/hd/d consuming 8 kg lbs DM per day.
[b]From Cort et al. (1983).
[c]Contained 41.5% dehydrated alfalfa.

Table 6. Effects of vitamin E supplementation on meat quality. Results of a retail study conducted at various retail locations.

Location[a]	Product Available[b] (lbs) C[c]	+E	Total Retail Value ($) C	+E
Las Vegas, NV	259.12	274.56	976.54	1017.47
Lenexa, KS	383.37	355.97	1663.24	1516.24
Houston, TX	323.29	323.99	1225.86	1224.22
Total:	**965.78**	**954.54**	**3865.64**	**3757.93**

Location[a]	Product Discounted (lbs) C	+E	Discount ($) C	+E
Las Vegas, NV	81.52	30.16	88.72	33.29
Lenexa, KS	106.40	48.58	70.96	37.13
Houston, TX	83.45	9.00	57.42	3.87
Total:	**271.37**	**87.74**	**217.10**	**74.29**

Location[a]	Discount Loss (% of initial) C	+E	Discount Per lb for All Product Displayed (¢) C	+E
Las Vegas, NV	9.09	3.27	34.2	12.1
Lenexa, KS	4.27	2.45	18.5	10.4
Houston, TX	4.68	.32	17.8	1.2
Total:	**5.62**	**1.98**	**23.5**	**7.9**

[a]All tests in each location were conducted over a one-week period.
[b]Product evaluated consisted of strip steaks, T-bone steaks, Tenderloin steaks, shoulder roasts, Top-round steaks and Top sirloin steaks.
[c]C=control beef, cattle did not receive supplemental dietary vitamin E; +E = vitamin E supplemented beef cattle received 500 IU vitamin E per head daily for 100 days prior to slaughter.