BENEFITS OF SUPPLEMENTAL VITAMIN E INCLUDING RELATIONSHIP TO GOSSYPOL

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INTRODUCTION

Vitamin E is essential for such body functions as growth, reproduction, prevention of various diseases, and protection of the integrity of tissues. The metabolic function of selenium is closely linked to vitamin E. Both selenium and vitamin E function to protect biological membranes from oxidative degeneration. Vitamin E and the Se containing enzyme glutathione peroxidase (GSH) are an integral part of the antioxidant system present in all cells. Vitamin E is a very efficient scavenger of both reactive oxygen species and lipid hydroperoxides, converting both to nonreactive forms (Putnam and Comben, 1987).

White muscle disease is the major clinical sign of vitamin E and Se deficiency. In Florida, the condition in feeder calves is seen most commonly as "buckling"; calves after transportation or handling show weakness of rear legs, buckling of fetlocks and frequently, generalized shaking or quivering of muscles (McDowell et al., 1985). Poor reproductive performance in cattle has been shown to include retained placenta, with high incidence of retained placentas greatly reduced by the administration of adequate dietary levels of vitamin E and Se. Harrison et al. (1984) indicated a 17.5% incidence of retained placenta for control dairy cows, with no incidence for cows receiving both Se and vitamin E (neither vitamin E nor selenium was effective alone). Other research indicates incidence of retained placenta (22.1%) was not affected by a combination of Se-vitamin E or Se alone (Hidiroglou et al., 1987).

Comprehensive reviews of the functions and metabolism of vitamin E and Se are available (Hidiroglou et al., 1992; McDowell, 1985, 1989, 1992). The present paper

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will emphasize under what conditions vitamin E supplementation is required for ruminants, as well as the more recent information on placental and mammary transfer, immune response, mastitis, and meat color. A role of supplemental vitamin E to alleviate gossypol toxicity will be noted. Likewise, the need for Se supplementation will also be stressed where appropriate. The NRC has estimated the vitamin E requirements for ruminants to range from 15-40 mg/kg, however higher supplemental levels will be shown to be beneficial.

PLACENTAL AND MAMMARY TRANSFER

Selenium is readily transmissible through the placenta to the fetus and passes the mammary barrier but is less efficiently transferred to later produced milk than to colostrum. Maternal Se status influences colostral Se concentration and calf Se status in the early postnatal period (McDowell et al., 1990). For Brahman cattle in Florida, Se colostrum concentration for control animals was 0.015 mg/kg compared to 0.031 mg/kg for dietary selenium supplementation (0.25 mg/kg) and 0.049 for dietary plus injectable Se (5 mg selenium) supplementation (McDowell et al., 1990). These data emphasize the importance of Se supplementation throughout pregnancy to ensure adequate placental transfer in an effort to maximize neonatal Se reserves and to minimize the potential of postnatal Se deficiency disease. Additionally, the role of adequate colostrum ingestion should not be overlooked.

Contrary to Se, vitamin E does not cross the placenta in any appreciable amounts, however, it is concentrated in colostrum (Van Saun et al., 1989). With respect to neonatal ruminants, several investigators (Hidiroglou et al., 1969; Van Saun et al., 1989) have reported limited placental transport of α-tocopherol, making neonates highly susceptible to vitamin E deficiency. This may be related to either a decreasing efficiency in placental vitamin E transfer as gestation proceeds, a dilution effect as a result of rapid fetal growth or possibly a decrease in available maternal vitamin E. With limited placental transfer of vitamin E, neonatal ruminants must rely heavily on ingestion of colostrum as source of vitamin E. Van Saun et al. (1989) reported decreased fetal serum vitamin E concentrations with increasing fetal age and increased fetal vitamin E status with greater maternal vitamin E concentration in dairy calves and cows. Additionally, these authors reported less of a decline in fetal serum vitamin E concentration during gestation in fetuses from vitamin E adequate dams.

Although placental transfer of vitamin E is inefficient, high levels of the vitamin have been shown in calves (Nockels, 1991) and lambs (Njeru et al., 1994) after consumption of colostrum. Nockels (1991) reported α-tocopherol levels in plasma from beef calves prior to colostrum consumption, and for several days thereafter. Precolostral plasma vitamin E levels averaged 0.2 µg/ml and increased to 3.3 µg/ml at 5 to 8 days of age. Njeru et al. (1994) fed ewes DL-α-tocopheryl acetate at graded levels (0, 15, and 60 IU/head daily) to study placental and mammary gland transfer. Supplemental vitamin E had no effect on serum α-tocopherol of lambs prior to nursing, averaging .35 µg/ml. By day 3, lamb serum tocopherol increased to 1.41, 1.84, 2.43 and 4.46 µg/ml, respectively, for the four supplemental dietary levels of vitamin E (Table 1). Vitamin E at the given levels of supplementation increased colostral-α-tocopherol at a linear rate of 3.3, 6.8, 8.0 and 9.6 µg/ml, respectively. The importance

of providing colostrum rich in vitamin E is quite apparent as both calves and lambs are born with low levels of vitamin E (Nockels, 1991; Njeru et al., 1994). Other research that also found this very low blood vitamin E concentrations at birth for ruminants include Whiting and Loosli (1948), Hidiroglou et al. (1969), and Van Saun et al., (1989). Low blood vitamin E may lead to diminished disease resistance and immune response in the neonate (Nockels, 1991). Also important is the concept that colostrum can be increased significantly in vitamin E by supplementing the vitamin to the dam (Table 1).

VITAMIN E AND GOSSYPOL TOXICITY

Cotton by-products (cottonseed, cottonseed meal, cottonseed hulls) have been fed to ruminants for over 100 years. Although whole cottonseed (WCS), and cottonseed meal (CSM) are an important source of protein for ruminants, they contain the toxic polyphenolic pigment gossypol. Gossypol has been found to affect liver functions, erythrocyte oxygen carrying or releasing capacity, respiration rate, intake and production and reproductive capacity (Lindsey et al., 1980; Calhoun et al., 1990; Gray et al., 1990). Cottonseed containing 1.3% gossypol was recently implicated in the deaths of dairy cows (Price et al., 1993). In relation to female reproduction, gossypol has been shown to inhibit embryo implantation (Lagerlof and Tone, 1985), delay embryo development (Zirkle et al., 1988) and decrease progesterone production in bovine luteal cells (Gu et al., 1990). Wyse et al. (1991) found that feeding 5.0 g free gossypol from cottonseed meal increased the number of degenerative embryos collected from superovulated Brangus heifers.

In ruminants, gossypol has been found to affect sperm motility associated with damage to the spermatogenic epithelium, leading to reduced germinal cell layers (Arshami and Ruttle, 1988; Chase et al., 1989). Chase et al. (1989) found delay in age at puberty in ruminants fed 60 mg free gossypol per kg body weight, but not when fed at 6 mg. After five weeks, Risco et al. (1993) found that the percentage of normal spermatozoa was lower in bulls fed 8.2 g free gossypol than in bulls fed 0 g. It is unlikely that gossypol toxicity will occur in mature cattle fed diets containing up to 25% whole cottonseed (Calhoun, 1995).

Generally, no problems result from feeding relatively high levels of cotton products to mature ruminants. Although ruminants seem to have a large capacity to the detoxify gossypol, toxicity resulting from consumption of this compound has been observed. Gossypol intake by mature ruminants may overwhelm ruminal detoxification and become absorbed at levels potentially toxic (Randel et al., 1992). Very high levels of cottonseed products are currently being fed to beef and high producing dairy cattle in early lactation (e.g. 3.2-3.3 kg per head daily). Typically gossypol varies from 0.9-1.2% in whole cottonseeds. Cattle consuming 3.3 kg of whole cottonseeds would ingest 40 g of free gossypol, problems have been reported with 4 to 5 g of free gossypol.

Since cottonseed products are economical, least cost formulations result in high levels of these products in cattle diets. For optimum cattle production it would seem logical to restrict gossypol intake or modify the toxicity. Vitamin E has a great

potential to modify gossypol toxicity. Formation of free radicals due to stress conditions has been associated with many reproductive problems such as retention of fetal membranes, reduction of embryo development, and production of hormones. Vitamin E is an antioxidant that prevents the damage of free radicals at the cellular level. It seems that gossypol as a promoter of free radical formation may reduce cellular antioxidants. Bender et al. (1988) found that vitamin E, ascorbate, glutathione peroxidase, and other antioxidants were reduced by feeding rats high levels of gossypol. In dairy cattle, Lane and Stuart (1990) found that feeding high amounts of gossypol decreased plasma vitamin E levels, indicating a possible relationship between gossypol toxicity and vitamin E. Pre-and postpartum consumption of free gossypol impaired aspects of calf skeletal development with lowered serum vitamin E and ß-carotene (Willard et al., 1995).

The University of Florida has undertaken experiments to study relationships between gossypol and supplemental vitamin E. Velasquez-Pereira et al. (1996) studied long term feeding of gossypol from cottonseed meal (CSM) on reproductive parameters of Holstein bulls and the use of vitamin E to counteract detrimental effects of gossypol. One treatment group received a high dietary level of gossypol from one week until 16 months of age. Treatments were modified at 6 months of age and were as follows: 1) control (SBM + corn) 2) CSM +corn and 3) CSM + 4000 IU vitamin E (DL-α-tocopheryl acetate) per head daily. Treatments 2 and 3 provided 14 mg free gossypol/kg body weight (from 2.3 g at 6 months to 5.3 g daily at 16 months). Final weights approached statistical significance ($P<.10$) with best results from the E treatment followed by the control compared to the gossypol alone (400, 388 and 372 kg, respectively).

Percentage of animals that reached puberty (production of 50 X 10^6 sperm with at least 10 % motility) is shown in Figure 1. During the experimental period, four animals from the gossypol group did not produce enough semen to be pubertal according to the standards of experimental protocol. Vitamin E feeding seemed to improve age at sexual maturity in this study. Chase et al. (1994) reported that bulls fed whole cottonseeds from weaning through puberty had lower body weight gains and reached sexual puberty at an older age than bulls fed CSM or SBM.

Velasquez-Pereira et al. (1996) reported that bulls which received 14 mg free gossypol /kg body weight had a lower ($P<.05$) percentage of normal sperm than those which also received supplemental vitamin E, 31 vs 55%, respectively (Table 2). Likewise, sperm production per gram of parenchyma and total daily sperm production were higher ($P<.05$) when gossypol treated animals also received vitamin E. For these same animals, sex-drive traits were studied in relation to the control, gossypol and gossypol + vitamin E treatments (Table 3). At 12 and 16 months of age animals were given two assessments for sex-drive.

Bulls receiving gossypol exhibited more sexual inactivity ($P < .05$) at the first test than bulls in other treatments. Vitamin E supplementation to bulls receiving gossypol improved number of mounts in the first test and time of first service in the second test. The results of gossypol treatment for the first test may indicate lack of sexual maturity, which agrees with sperm production data. At time of first test (12 months of age)

none of the gossypol, two of control and six of the vitamin E supplemented bulls had reached puberty as per experimental protocol. The final conclusion of the Florida data is that vitamin E is effective in reducing or eliminating the important gossypol toxicity effects for male cattle.

PERFORMANCE AND IMMUNE RESPONSE FOR RUMINANT HEALTH, INCLUDING MASTITIS CONTROL

Deficiencies of vitamin E and Se result in disease conditions as a result of an inadequate immune response. Likewise, levels of Se and vitamin E above the generally accepted requirements have enhanced the immune response in several species. 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 help these cells to survive the toxic products that are produced in order to effectively kill ingested bacteria (Badwey and Karnovsky, 1980). In a number of mammalian species, Se deficiency has been associated with decreased immune system function. Decreased GSH 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; Gyang et al., 1984). Inefficient immune cell function may predispose cattle to infectious diseases.

The protective effects of vitamin E 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 immuno enhancing effect by virtue of altering arachidonic acid metabolism and subsequent synthesis of prostaglandin, thromboxanes and leukotrienes. Under stress conditions, increased levels of these compounds by endogenous synthesis or exogenous entry may adversely affect immune cell function (Hadden, 1987).

The effects of vitamin E and Se supplementation on protection against infection by several types of pathogenic organisms, as well as antibody titers and phogocytosis of the pathogens, have been reported for calves (Cipriano et al., 1982; Reddy et al., 1987) and lambs (Reffett et al., 1988; Finch and Turner, 1989). Calves receiving 125 IU of vitamin E daily were able to maximize their immune responses compared to calves receiving low dietary vitamin E (Reddy et al., 1987).

Vitamin E administration to calves enhanced immune response and weight gain while enzymes indicative of muscle breakdown (e.g., creatine kinase and serum glutamic oxaloacetic transaminase) and plasma cortisol concentration were decreased (Reddy et al., 1987). Vitamin E also positively influenced neutrophil-mediated antibody dependent cellular cytotoxicity and phagocytosis as well as lymphocyte stimulation in calves fed milk replacer with 57 IU vitamin E per kilogram for an eight-week period (Pruiett et al., 1989).

Trends toward a higher cell-mediated immune response was observed in dairy calves following 1 g supplementation of vitamin E per day (St. Laurent et al., 1990). Studies with Holstein heifer calves supplemented weekly with 1,400 or 1,800 mg of vitamin E enhanced both cell-mediated and humoral immune response (Reddy et al., 1986). These improved calf responses suggest that the criteria for minimum requirements should not be based entirely on growth rates or the amounts needed to prevent clinical signs of deficiencies, but also on the amounts needed for optimal immune competence (Reddy et al., 1986).

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 (as DL-α-tocopheryl acetate) per head per day that were stressed by long distance shipment and changes from green forages to high grain feedlot diets. Perhaps depression of circulating cortisol concentrations may explain the improved gained and feed efficiency in this trial (Table 4).

Gill et al. (1986) supplemented newly received feedlot cattle with 1600 IU vitamin E (as DL-α-tocopheryl acetate) per head per day for the first 21 days and 800 IU vitamin E per head per day 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 (Table 5). The number of sick pen days per head was reduced by 15.6% and morbidity was reduced by 13.4% with vitamin E supplementation (Table 5). 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, and likewise Se via GSHPx in destroying these free radicals. Increasing stress apparently increases the potential for vitamin E to improve cattle performance. Vitamin E supplementation of feedlot cattle has not consistently affected performance, partially because of dietary adequacy of the vitamin, level of stress of cattle, and previous dietary history. In a series of six trials conducted with cattle moving from pasture to high-energy diets fed in confinement (Hill and Williams, 1993), gain and feed conversion efficiency tended to be improved, but in some trials performance was unaffected by dietary vitamin E supplementation. In trials completed in 1993 and 1994 (G.M. Hill, personal communications), performance of steers was improved by vitamin E supplementation at 1000 or 1500 IU/hd daily. Hutcheson and Cole (1985) reported improved gain in cattle fed 50, 100, 300 IU of vitamin E daily. These reports may be contrasted with others that showed no improvement in feedlot performance when vitamin E was supplemented at 200 IU/hd daily or higher levels (Schaefer et al., 1989; Arnold et al., 1992; Arnold et al., 1993). In two trials conducted with steers subjected to mild stress associated with abrupt movement from pasture to a slatted floor feedlot, Hill (1987) reported no improvement in gain or feed efficiency in Trial 1 when supplemental vitamin E was at 0 to 200 IU/hd daily for 130d. However, in Trial 2, steers supplemented with 500 or 1000 IU of vitamin E daily had significantly improved 28-d gains and feed conversion efficiencies compared with controls provided with no supplemental vitamin E. Gain performance

for both supplemental treatments tended to be higher than controls throughout the 84-d trial.

In subsequent trials (Hill et al., 1990) effects of vitamin E supplementation (0 or 1000 IU/hd daily) on feedlot performance, carcass traits and liver abscesses were determined. All diets were supplemented with 0.1 mg/kg Se per kg of diet. In Trial 1, pelleted peanut hulls were fed at 15% of the diet as the roughage source, because in previous trials they induced high levels of liver abscesses when fed to feedlot cattle. Slight improvements in ADG and feed/gain were recorded for the vitamin E supplemented steers and liver abscesses were reduced from 41.4% (12 of 29 livers) for controls to 21.4% (6 of 28 livers) for vitamin E steers. In Trial 2, coarsely ground peanut hulls were fed at 15% of the diet, resulting in 14% improvements in both 28-d gain and feed efficiency for vitamin E supplemented steers, and 126-d performance tended to be higher for vitamin E supplemented steers. Liver abscess incidence was low for these steers on the coarsely ground peanut hull diets (Control steers: 1 of 18=5.5%; vitamin E steers: 2 of 17=11.8%).

Two feedlot experiments conducted with steers fed high levels of vitamins A and E revealed substantial effects on performance and on plasma, liver and muscle α-tocopherol concentrations (Hill et al., 1995a,b). Corn-based 83% concentrate diets were fed to 84 steers for 133 d in Exp. 1, and to 72 steers for 105 d in Exp 2 with supplemental vitamin A and E. A 2 x 2 factorial arrangement of treatments was used in each experiment, and specific treatments supplied supplemental vitamin A and E (IU/steer daily) at the following respective levels: A=Lo A, Lo E, 24,000 and 150; B=Hi A, Lo E, 70,000 and 150; C=Lo A, Hi E, 24,000 and 1,500 (1,000 in Exp. 2); D=Hi A, Hi E, 70,000 and 1,500 (1,000 in Exp. 2). All diets were supplemented with vitamin E (7,000 IU/d). Se at .3 ppm/steer daily, and lasalocid (Bovatec®) at 30 g/ton. All steers were implanted with Synovex-S® on d 1 of each experiment. High level vitamin A supplementation at 70,000 IU/d, which is approximately three time current NRC (1984) recommendations for growing-finishing beef cattle, reduced steer ADG ($P < .05$) in Exp. 1, and it resulted in less efficient ($P < .05$) conversion of feed to gain in Exp. 1 and 2. High-level vitamin E supplementation did not affect performance in Exp. 1, but it increased ADG ($P < .05$) and improved feed conversion efficiency in Exp. 2. Feeding high levels of vitamin A tended to depress ($P > .10$) plasma α-tocopherol at d 28, and depressed plasma α-tocopherol at d 112 ($P < .06$) in Exp. 1, and at d 105 ($P < .05$) in Exp.2. High levels of vitamin E increased ($P < .01$) plasma α-tocopherol at d 28 in both experiments, and increased ($P < .05$) plasma α-tocopherol at d 112 in Exp. 1 and at d 105 in Exp. 2. Muscle α-tocopherol was increased by HI E in Exp. 1 ($P < .05$) and Exp. 2 ($P < .01$). Liver α-tocopherol was increased in Exp. 2 ($P < .01$). Patterns of α-tocopherol concentrations in muscle and liver were very similar to those observed for plasma for the four treatments. There were no interacitons ($P > .10$) for vitamin A and E treatments in either experiment for performance data or for tissue samples. Control (Lo A, Lo E) treatments in both of these experiments produced muslce α-tocopherol concentrations at or above the 3.3 μg/g threshold concentration for color stability proposed by Arnold et al. (1993); therefore, differences in color retention or stability were not observed for vitamin E treatments. Supplementation of feedlot diets with approxiamtely three times NRC recommended levels of vitamin A could potentially reduce feedlot performance and interfere with vitamine E metabolism.

Mastitis control is based on two principles: reducing teat end exposure to pathogens and increasing the cow's resistance to infection. The skin and keratin plug within the teat canal provide a physical barrier to potential pathogens, however beyond this the non-specific immune system comes into play. Once an antigen (e.g., mastitis organism) has reached the teat and entered the lumina of the udder, there is a host of cellular and humoral defense systems that may take part in its destruction. Vitamins A and E, β-carotene and selenium have all been implicated in enhancement of the defense system against mastitis.

Mastitis incidence has been shown to be related to vitamin E and Se status of dairy herds. Weiss et al. (1990) surveyed dairy herds and observed a negative correlation between dietary vitamin E intake and rates of clinical mastitis. Vitamin E and Se supplementation of dairy cows resulted in reduced rates and duration of intramammary infections and incidence of clinical mastitis (Smith et al., 1984).

Supplemental levels of vitamin E higher than recommended by the dairy cattle NRC (1989) have been beneficial in the control of mastitis. Smith and Conrad (1987) reported that intramammary infection was reduced 42.2% in vitamin E-Se supplemented versus unsupplemented controls. The duration of all intramammary infections in lactation was reduced 40 to 50% in supplemented heifers. Lactation mean somatic cell count was lower in supplemented heifers and there was a 68% reduction in heifers with lactation mean somatic cell counts greater than 200,000 cell per ml. Clinical mastitis during the first 4 days of lactation was reduced by 57%. Weiss et al. (1990) reported that clinical mastitis was negatively related to plasma Se concentration and concentration of vitamin E in the diet. Diets of multiparous dairy cows were supplemented with either 0 or 1,000 IU vitamin E (as DL-α-tocopheryl acetate) during the dry period (Smith et al., 1984). Cows were additionally administered with Se at the rate of 0 or .1 mg per kilogram body weight via i.m. injection 21 days pre-partum. No vitamin E or Se was supplemented during lactation. Incidence of new clinical cases of mastitis was reduced by 37% in both groups receiving vitamin E compared to controls. 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. Erskine et al. (1989) investigated specific effects of Se status of dairy cattle on the induction of mastitis by E. coli. Bacterial concentrations in milk were significantly higher in Se-deficient than in Se-adequate cows and Se supplementation reduced both severity and duration of clinical mastitis. Other field surveys throughout the world, not previously reviewed above, have also confirmed the practical importance of dietary vitamin E and selenium supplementation of dairy cows in maintaining udder health (Atroshi et al., 1986; Ropstad et al., 1987).

Vitamin E and Se appear to enhance host defenses against infections by improving phagocytic cell function. Both vitamin E and GSH are antioxidants that protect phagocytic cells and surrounding tissues from oxidative attack by free radicals produced by the respiratory burst of neutrophils and macrophages during phagocytosis (Baboir, 1984; Baker and Cohen, 1983). Hogan et al. (1992) reported that vitamin E

supplementation of diets increased intracellular kill of Staphylococcus aureus and Escherichia coli by neutrophils.

Dietary supplementation of cattle with Se results in a more rapid neutrophil influx into milk following intramammary bacterial challenge and increased intracellular kill of ingested bacteria by neutrophils (Hogan et al., 1993). Dietary supplementation with vitamin E for early lactation cows increased bactericidal activity by bovine blood neutrophils. Recently completed trials have shown that subcutaneous injections of vitamin E approximately 10 and 5 days prior to calving successfully elevated neutrophil α-tocopherol concentrations during the periparturient period and negated the suppressed intracellular kill of bacteria by neutrophils that is commonly observed at calving (Hogan et al., 1993).

STABILIZING EFFECT OF VITAMIN E ON BEEF COLOR AND LIPIDS

Color is a primary factor used by consumers to judge beef quality, fresh meat should be bright-red or bright-pink and that any deviation from this is unacceptable. Myoglobin is the pigment mainly responsible for meat color since most hemoglobin is removed when the animal is slaughtered. 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 related (Greene, 1971; Faustman et al., 1989a) suggesting that delaying the breakdown of lipid may result in similar delay of meat discoloration. It is 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 (Williams et al., 1993).

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. Additional research has been reported from the University of Georgia (Hill et al., 1990; Hill et al., 1992; Hill and Williams, 1993) and at the University of Idaho (Comstock et al., 1991; Garber et al., 1992). In the University of Wisconsin, Holstein and crossbred beef steer calves of various weights and ages were utilized with a variety of vitamin E supplementation regimens.

The basal diet in all trials consisted of 100 g/kg corn silage and 900 g/kg concentrate (dry matter basis). The concentrate portion of the diet was composed of 742 g/kg high-moisture corn, 88 g/kg soybean meal, 35 g/kg vitamin and mineral supplements and 35 g/kg vitamin E supplement. The basal diet was formulated to

contain 0.1 mg/kg 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 6. Both length of time and level of vitamin E supplementation affected tissue concentration of α-tocopherol as compared to animals receiving only the 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 7). Vitamin E supplementation, regardless of level or duration, extended the color shelf-life of loin (or sirloin, data not presented here: see Faustman et al., 1989a; Faustman et al., 1989b) and steaks aged 7 or 21 days in both Holstein and crossbred beef steers (Hoffmann-La Roche, 1991).

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, 1991; Schaefer et al., 1991; Hill and Williams; 1993; Kiser et al., 1993). 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 .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). It would appear that dietary 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 increased tissue α-tocopherol concentrations in terms of reduced accumulation of metmyoglobin and TBA reactive substances. Liu et al. (1995) suggest, from cumulative experiments conducted to date, that beef from animals that receive 500 IU/steer daily of vitamin E for 126 days would greatly extend color display life.

Adding vitamin E at the feedlot requires an administration of 500 IU per day during the last 100 days of the finishing period at a cost of $1.50 per head (Williams, S. N., personal communication, 1994). A higher, longer incorporated dose is recommended by Williams for beef destined to the export market, because of the longer time the beef is vulnerable to oxidation and because the increased cost is recoverable in increased prices. There has been a development of an inexpensive in-slaughter facility analysis to verify vitamin E supplementation to finishing cattle (Smith, 1994). The spectrophotometric method can be performed by an employee trained to do so on the processing line. It is quick and reduces cost from $80 per sample to $3.00 to $3.50.

There are human health benefits of having higher levels of vitamin E in meat, other than the fat stability concept (McDowell, 1989; VERIS, 1993). Recently, antioxidant nutrients, vitamin E, vitamins C and beta-carotene have become the focus for their protective role in disease prevention, particularly cardiovascular disease (atherosclerosis) and cancer. Vitamin E also has a beneficial effect in preventing eye disorders, skin diseases, ulcers and intermittent claudication (inadequate blood flow). Vitamin E may reduce atherosclerosis by preventing low density lipoprotein (LDL) cholesterol from oxidizing and causing arterial injury.

SUMMARY

The most widely known result of Se and vitamin E deficiency is tissue degeneration (e.g., white muscle disease). Vitamin E does not cross the placenta in any appreciable amounts; however, it is concentrated in colostrum. Supplemental vitamin E can greatly increase colostral tocopherol. The importance of providing colostrum rich in vitamin E is essential as both calves and lambs are born with low levels of the vitamin. Detrimental effects of gossypol on bull reproduction can be eliminated or greatly reduced with supplemental vitamin E. Vitamin E has been shown to increase performance of feedlot cattle, to increase immune response for ruminant health including being beneficial for mastitis control. Vitamin E at higher than NRC requirements to finishing cattle has dramatically maintained the red color (oxymyoglobin) versus the oxidized color metmyoglobin of beef. It would appear that supplementation of 500 I.U. vitamin E per head daily for 84 to 126 days would yield tissue α-tocopherol that would maintain a favorable level of oxymyoglobin, to increase the value of meat. The NRC estimates vitamin E requirements for beef cattle, dairy cattle, and sheep to range from 15-40 mg/kg, however higher levels will likely improve performance and megadose levels will improve carcass quality. Higher amounts of

supplemental vitamin is also benefitial to modify or eliminate gossypol toxicity effects in male reproduction.

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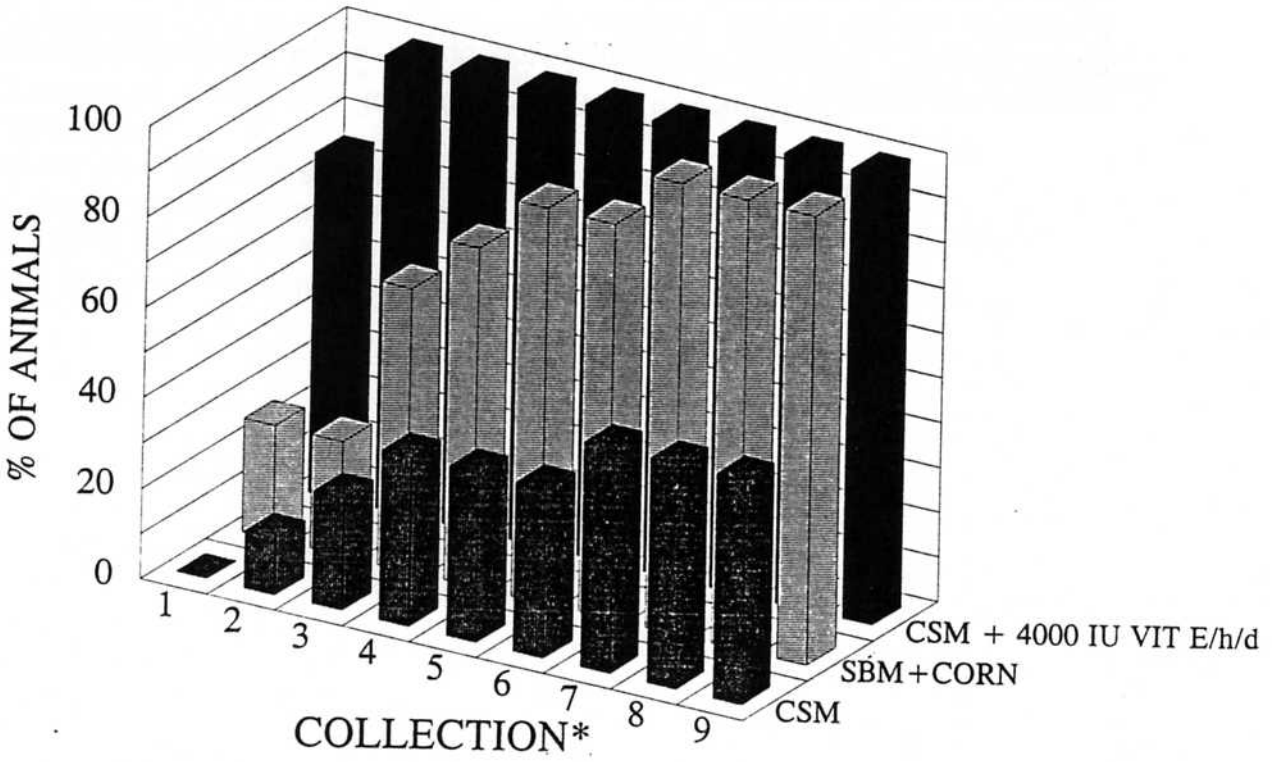
Figure 1. Percentage of animals that reached puberty at each collection



*Collections started at 12 mo. of age and continue weekly for nine wks.

Table 1. Effect of supplemental vitamin E to prepartum ewes on α-tocopherol concentration in serum and colostrum (μg/ml)[a,b]

	Ewes		Lambs	
Supplemental vitamin E (IU/day)	Serum at Parturition	Colostrum Day 1	Serum Prior to Nursing	Serum Day 3
0	0.94	3.3	0.4	1.41
15	1.94	6.8	0.4	1.84
30	2.53	8.0	0.38	2.43
60	4.07	9.6	0.23	4.46

[a] Njeru et al. (1994b). Treatments administered as DL-α-tocopheryl acetate 28 days prepartum through 28 days postpartum.

[b] Linear ($P<0.05$) treatment effects for α-tocopherol in ewe serum and colostrum and lamb serum at day 3.

Table 2. Relationship of gossypol and vitamin E on semen characteristics of dairy bulls[a]

Item	Treatment		
	TRT1[b]	TRT2[c]	TRT3[d]
Normal, %	64.7 ± 6.4[h]	31.4 ± 7.4[i]	54.6 ± 6.4[h]
Abnormal (DIC)[e], %	4.4 ± 1.3[h]	13.4 ± 1.5[i]	4.8 ± 1.2[h]
DSPG[f] ($x10^6$/g)	14.6 ± 1.0[h]	10.2 ± 1.0[i]	17.6 ± 1.0[h]
DSP[g] ($x10^9$)	3.2 ± 3.0[h]	2.2 ± 3.0[i]	4.1 ± 3.0[h]

[a]Least square means ± SEM.
[b]Diet based on SBM, corn and 30 IU vitamin E/kg of supplement.
[c]Diet containing 14 mg free gossypol/Kg BW/d and 30 IU vitamin E/kg of supplement.
[d]Diet containing 14 mg free gossypol/Kg/ BW/d and 4000 IU vitamin E/bull/d.
[e]Midpiece abnormalities evaluated in isotonic formal saline using DIC.
[f]Daily sperm production per gram of parenchyma.
[g]Daily sperm production total.
[hi]Means in a row with different superscript differ $P < .05$.

Table 3. Effects of Gossypol and Vitamin E on Sex-drive of Holstein bulls[1]

Item	Mo.	Control	+Gossypol[2]	+Gossypol+Vit. E[3]
Libido Score	12	7.9 ± 1.2	6.8 ± 1.0	9.1 ± 1.1
	16	10.4 ± .7	8.9 ± .5	10.0 ± .6
No. of Mounts	12	5.7 ± 1.2	3.2^{b} ± 1.1	6.3^{a} ± 1.2
	16	9.4 ± 1.3	9.5 ± .9	7.9 ± 1.1
No. of Services	12	1.4 ± .5	1.4 ± .5	1.9 ± .5
	16	2.4 ± .5	1.7 ± .4	2.3 ± .4
Time of Mounts (sec)	12	156 ± 60	225 ± 57	135 ± 57
	16	38^{a} ± 7	29^{ab} ± 5	21^{b} ± 5
Time of 1st service (mo)	12	223 ± 94	232 ± 69	206 ± 69
	16	154^{ab} ± 71	213^{a} ± 52	69^{b} ± 60
Sexual Activity (min)	12	1.2^{d} ± .8	3.9^{c} ± .7	1.7^{d} ± .8
	16	.1 ± .2	.1 ± .1	.2 ± .2

[ab] Means with different superscript in the same row differ (P<.1)
[cd] Means with different superscript in the same row differ (P<.05)

[1] Each treatment represented 8 animals.
[2] Animals received 14 mg free gossypol per kg body weight.
[3] Receeved 4,000 IU vitamin E per day as DL-α-tocoperyl acetate.

Table 4. Effect of vitamin E supplementation on 28-day performance of stressed beef cattle[a]

Treatment	Average Daily Gain (kg)	Average Daily Feed Intake (kg as fed)	Feed Conversion Ratio
Control	1.18[b]	7.47	6.33[e]
450 IU Vitamin E	1.25[c]	7.37	5.90[f]
450 IU Vitamin E + B-Vitamins	1.31[d]	6.95	5.31[f]

[a] From Lee et al. (1985).
[b,c,d] Means with different superscripts differ (P<.08).
[e,f] Means with different superscripts differ (P<.02).

Table 5. Effect of vitamin E supplementation on performance, morbidity and mortality in stressed cattle[a]

Item	Control	Vitamin E
Number of head	252	250
Average daily gain, kg	.43[c]	.53[d]
Feed conversion ratio	18.56	15.06
Sick days	3.2	2.7
Morbidity, %	43.2	37.5
Mortality, %	1.8	1.6

[a] From Gill et al. (1986).
[b] 1600 IU vitamin E per head per day for first 21 days and 800 IU for the last seven days.
[c,d] Means with different superscripts differ (P<.01).

Table 6. 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	.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

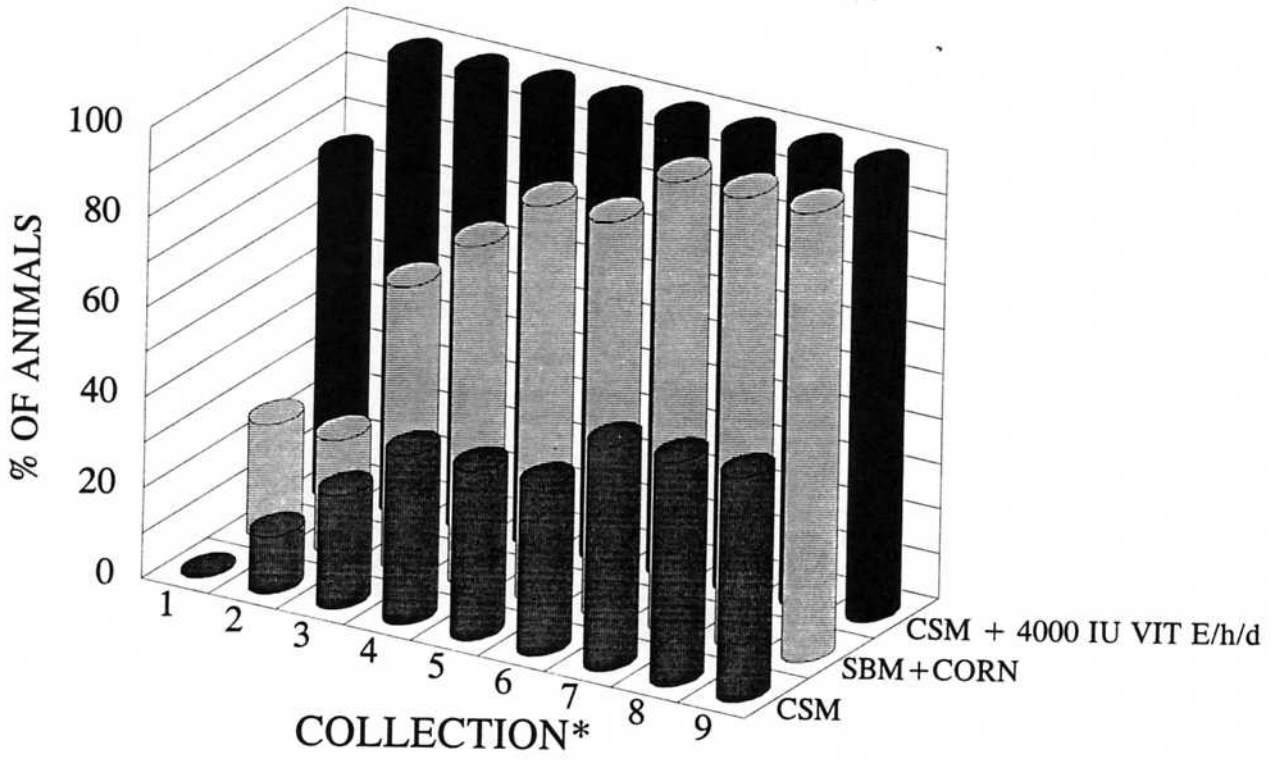
[a] From Hoffmann-La Roche (1991), Schaefer et al. (1991), Arnold et al. (1992).
[b] Total dietary vitamin E intake minus vitamin E content of feedstuffs
[c] Liver, subcutaneous fat and sirloin (µg/mg), plasma (µg/ml).
[d] Tissue α-tocopherol concentrations as follows: left number = 42 days, right number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

Table 7. 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 (1991), Schaefer et al. (1991), Arnold et al. (1992).
[b] Total dietary 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.

Figure 1. Percentage of animals that reached puberty at each collection



*Collections started at 12 mo. of age and continue weekly for nine wks.