UPDATE ON VITAMIN E AND SELENIUM NUTRITION FOR RUMINANTS

by

Lee R. McDowell
Animal Science Department
University of Florida
Gainesville, Florida

Scot N. Williams
Hoffmann-La Roche Inc.
Nutley, New Jersey

Introduction

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 selenium is closely linked to vitamin E. Both selenium and vitamin E function to protect biological membranes from oxidative degeneration. 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 glutathione peroxidase (GSH), 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 selenium are available (Combs and Combs, 1986; McDowell, 1989, 1991). The present paper will emphasize the effects of deficiency of these nutrients as well as the more recent information on placental and mammary transfer, immune response, mastitis, meat color, status detection and finally requirements and methods of supplementation.

Selenium-Vitamin E Deficiency

The most significantly important result of selenium and vitamin E deficiency is tissue degeneration. White muscle disease (WMD) is a degeneration of striated muscles that occurs without neural involvement and is the major clinical sign of selenium and vitamin E deficiency in newborn ruminants. The disease is characterized by generalized weakness, stiffness and deterioration of muscles with affected animals having difficulty standing. Ultimately, they lose body weight and become prostrate before death. Calves with WMD have chalky white striations, degeneration, and necrosis in the skeletal muscles and heart. In calves the tongue musculature may be affected, therefore not allowing suckling. In milder cases with calves where the chief clinical signs are stiffness and difficulty standing, dramatic, rapid improvement can result from selenium-vitamin E injections. During some years, incidence of WMD in certain regions is sporadic, with less than 1% of the

Presented at the 2nd Florida Ruminant Nutrition Symposium, Gainesville, January 23-24, 1991

herds affected. In other areas such as Turkey and New Zealand, a 20-30% incidence of WMD may occur.

An acute and chronic as well as a peracute form of the disease can be distinguished in older calves, usually already in the finishing period. In particular, stress situations such as transport, regrouping or abrupt changes in feed composition are generally considered as precipitating factors. Sudden death without previous unmistakable signs of disease are main features of the peracute condition. The cause is usually to be found in advanced degeneration of myocardium. Motor disturbances such as an unsteady gait or stiff calf disease, hard lumbar, neck and forelimb muscles, muscle tremor and perspiration are encountered in acute form. In lambs, WMD takes a similar course to that found in calves.

From Florida, in feeder calves the condition is seen most commonly as "buckling;" calves come off the truck, or out of the processing chute with weakness of rear legs, buckling of fetlocks and frequently, generalized shaking or quivering of muscles. Many calves become progressively worse until they are unable to rise and may appear to be paralyzed. Many animals will be down or continue to buckle for extended periods. Death loss is high in severe cases. Calves with excitable temperaments appear to be most commonly affected. Post-mortem examination of affected calves reveals pale, chalky streaks in muscles of the hamstring and back. The heart, rib muscles and diaphragm may also be affected (McDowell et al., 1985).

Recent research indicates that unsaturated fatty acids from lipids in grasses can act as a peroxidative challenge in WMD in calves. Nutritional degenerative myopathy in older calves occurs most frequently at turnout to spring pasture. McMurray et al. (1980) showed that polyunsaturated fatty acids were capable of escaping ruminal hydrogenation at turnout, resulting in a three-fold increase of plasma linolenic acid within three days of turnout. Rice et al. (1981) showed that linolenic acid, if protected from ruminal hydrogenation, rapidly reaches high levels in blood and is associated with a rise in plasma creatine phosphokinase indicating muscular degenerative myopathy.

Most nutritional myopathy cases have involved young ruminants with effects less fully described for adult species. However, degenerative myopathy in adult cattle has been reported. Rapid growth in heifers coupled with stresses of late pregnancy and parturition may contribute to this deficiency. A myopathic condition affecting yearling cattle has been reported by Barton and Allen (1973). The condition was associated with animals fed grains treated with propionic acid, which is known to be destructive to vitamin E.

Other selenium-responsive conditions are not restricted to young animals and relate to unthriftiness ("illthrift"), occurring in lambs and hoggets at pasture and can occur in beef and dairy cattle of all ages (Underwood, 1981). Cattle grazing on peaty muck soils in the Florida Everglades developed anemia associated with the presence of Heinz bodies and suboptimal blood selenium (Morris et al., 1984). Selenium supplementation of these cattle corrected anemia, prevented Heinz body formation, and increased body weight of both cows and calves. Further reports in Northern Florida have associated Heinz body anemia and diarrhea with cattle on rye pastures with low selenium status (W.

Kunkle and F. Bullock, 1990, personal communication). There appeared to be beneficial effects from adequate levels of selenium supplementation. Wolf et al. (1963) reviewed the therapeutic effect on diarrhea as the result of selenium treatment in lambs and calves. Cattle have been successfully treated with selenium for scours that had not responded to antibiotics or anthelmintics. Hartley and Grant (1961) mentioned a beneficial response to selenium by beef and dairy calves in New Zealand that had exhibited severe and rapidly progressive unthriftiness associated with profuse diarrhea.

In subclinical selenium-vitamin E deficiency, performance may be reduced, with slower gains and lowered reproduction involving an increased number of services needed per conception. In cattle, selenium supplementation was beneficial to maintaining sperm mobility in vitro. Hartley and Grant (1961) reported the incidence of barren ewes was reduced from over 30 to 5% with selenium administration. Farms in New Zealand have had lamb losses as high as 40 to 50%. Vitamin E reduced these losses by only 60%, while selenium reduced losses by 96%.

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 selenium, as shown by research in the United States, Scotland and Brazil. From Ohio, incidence of retained placenta was reduced from a mean of 51.2% in untreated cows to 8.8% in cows injected with a combination of selenium-vitamin E (Julien et al., 1976). Harrison et al. (1984) indicated a 17.5% retained placenta for control dairy cows, with no incidence for cows receiving both selenium and vitamin E (neither vitamin E nor selenium was effective alone). From the same study, control versus selenium administration reduced cystic ovaries (47 versus 19%) and incidence of metritis (84 versus 60%). Other research indicates incidence of retained placenta (22.1%) was not affected by a combination of selenium-vitamin E or selenium alone (Hidiroglou et al., 1987). In high-producing dairy goats, selenium-vitamin E deficiency manifests itself in poor involution of the uterus with accompanying retained placenta and metritis following kidding (Guss, 1977). Other studies on the effects of selenium and vitamin E on retained placenta have been inconclusive but do suggest that supplemental level, timing of administration, and the interaction of these nutrients with beta-carotene, vitamin A and other nutrients needs to be further defined.

Placental and Mammary Transfer

Selenium is readily transmissible through the placenta to the fetus. A selenium concentrating ability was revealed for the fetal liver, suggesting possibly a storage role for selenium mobilization in postnatal life (Van Saun et al., 1989a). Fetal and newborn calf whole blood selenium concentrations were influenced by maternal selenium status with a concentrating ability found in offspring from selenium-deficient dams (Van Saun et al., 1989a; Koller et al., 1984).

Selenium passes the mammary barrier but is less efficiently transferred to later produced milk than to colostrum or transferred via the placenta. Maternal selenium status influences colostral selenium concentration and calf selenium status in the early postnatal period (Koller et al., 1984; McDowell et al., 1990). For Brahman cattle in Florida selenium colostrum concentration

for control animals was .015 ppm compared to .031 ppm for dietary selenium supplementation (.25 ppm) and .049 for dietary plus injectable selenium (5 mg selenium) supplementation (McDowell et al., 1990).

A milk diet alone, even if the dam is supplemented with selenium may require the calf to mobilize selenium reserves obtained from placental transfer and colostrum ingestion. Koller et al. (1984) revealed a greater decline in nursing beef calf's selenium status from selenium deficient compared with selenium adequate dams.

These data emphasize the importance of selenium supplementation throughout pregnancy to ensure adequate placental transfer in an effort to maximize neonatal selenium reserves and to minimize the potential of antenatal or postnatal selenium deficiency disease. Additionally, the role of adequate colostrum ingestion should not be overlooked (Van Saun, 1990).

Contrary to selenium, vitamin E does not cross the placenta in any appreciable amounts, however, it is concentrated in colostrum (Van Saun et al., 1989b). With respect to neonatal ruminants, several investigators (Hidiroglou et al., 1969; Van Saun et al., 1989b) 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 a source of vitamin E. Van Saun et al. (1989b) 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.

Immune Response, Mastitis and Meat Color

Levels of selenium and vitamin E above the generally accepted requirements have been shown to enhance 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 selenium 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, selenium deficiency has been associated with decreased immune system function. Decreased GSH activity in phagocytic cells has been reported in selenium-deficient heifers (Boyne and Arthur, 1979). In bovine neutrophils, the bactericidal capacity for *Candida albicans* and *Staphylococcus aureus* is lowered in selenium-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 which challenge the animals antioxidant system. 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 immunoenhancing 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, 1989).

The effects of oral vitamin E supplementation in young calves was evaluated by Cipriano et al. (1982). Calves were fed skimmed colostrum and supplemented with either 0 or 1000 mg dl-α-tocopheryl acetate for six weeks in a vitamin E deficient diet. Conventionally managed calves were included as positive controls. Vitamin E supplemented calves had greater plasma α-tocopherol concentrations as well as mean lymphocyte blastogenesis response to phytohemagglutinin (PTH) expressed as mean lymphocyte stimulation indices (LSI) (Table 2) at six weeks. These authors suggested that the enhancing effect of vitamin E on immune response of cattle could have been partially masked in this study by feeding of diets high in emulsified fats.

Vitamin E administration to calves enhanced immune response and weight gain while enzymes of muscle origin (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 (Pruiett et al., 1989).

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 gain and feed efficiency in this trial (Table 3).

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 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 supplemental stressed cattle (Table 4). 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 4).

An increased incidence and severity of mastitis is associated with selenium deficiency and/or vitamin E deficiency (Smith et al., 1984; Erskine et al., 1989; Hogan et al., 1990; Weiss et al., 1990). Smith and Conrad (1987) reported that intramammary infection was reduced 42.2% in vitamin E-selenium supplemented versus unsupplemented controls. The duration of all intramammary infections in lactation was reduced 40 to 50% in supplemented heifers. Weiss et al. (1990) reported that clinical mastitis was negatively related to plasma selenium concentration and concentration of vitamin E in the diet. Diets of multiparous dairy cows were supplemented with either 0 or 1000 IU vitamin E (as dl-α-tocopheryl acetate) during the dry period (Smith et al.,

1984). Cows were additionally administered selenium at the rate of 0 or .1 mg/kg body weight via i.m. injection 21 days prepartum. No vitamin E or selenium were supplemented 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 selenium but not supplemented with dietary vitamin E. These authors also reported that clinical cases in the vitamin E supplemented-selenium injected cows were consistently of shorter duration than those occurring in all other groups. Erskine et al. (1989) investigated specific effects of selenium status of dairy cattle on the induction of mastitis by *E. coli*. Bacterial concentrations were significantly higher in selenium-deficient than in selenium-adequate cows and selenium supplementation reduced both severity and duration of clinical mastitis.

Dramatic effects of vitamin E supplementation (370 IU/head daily) on the stability of beef color were observed, although color score and pigmentation intensity were unaffected (Faustman et al., 1989a). Loin steaks of control steers discolored two to three days sooner than those supplemented with vitamin E. Supplemental dietary vitamin E extended the color shelf life of loin steaks from 3.7 to 6.3 days. This was most likely due to the increased α-tocopherol content of the loin tissue of the supplemented animals, which was approximately 4-fold greater than controls (Faustman et al., 1989a). Color is an extremely critical component of fresh red meat appearance and greatly influences the customer's perception of meat quality. In a subsequent report, Faustman et al. (1989b) observed that vitamin E stabilized the pigments and lipids of meat from the supplemented steers. Perhaps the vitamin E supplemented steers were able to incorporate a greater amount of vitamin E into cellular membranes where it can perform its antioxidant function. The effects of vitamin E as an in vivo lipid stabilizer and its effect on flavor and storage properties of various meats and milk have been reviewed.

Assessment of Status

Confirmation of a low selenium 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 selenium 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 nutritional muscular dystrophy 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 selenium dependent enzyme, GSH, is a relatively good status indicator of this element. Liver and plasma GSH activities increase or decrease rapidly during repletion or depletion of selenium, therefore, concentrations of the enzyme serve as a relatively

accurate indicator of the sufficiency of selenium. GSH 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).

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 .5-1 ug/ml are considered low in most animal species, with less than .5 ug/ml generally considered a vitamin E deficiency. Adams (1982) reported that plasma tocopherol concentrations between .60 to 1.6 ug/ml were associated with calves diagnosed with nutritional muscular dystrophy. Serum α-tocopherol concentrations of 1.0 to 1.5 ug/ml were reported by McMurray and Rice (1982) as associated with clinical lesions of white muscle disease, with values <2 ug/ml (.2 mg/dl) considered deficient. Serum α-tocopherol concentrations >4.0 ug/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 ug/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 concentration 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).

It is debated as to which assay is best in accurately predicting selenium status or potential presence of selenium 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 selenium, interpretation of the animal's selenium status may differ based on the assay used.

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

In many dairy herds, selenium-vitamin E supplementation stops when the cow no longer receives the lactation grain. If not receiving any dietary selenium and vitamin E, the cow will rapidly mobilize stores of these nutrients during the dry period. Depending upon her prior selenium and vitamin E status, the dry cow may become depleted by freshening with the possibility of selenium-vitamin E deficient disease affecting either the cow and/or calf. Analysis of the selenium and vitamin E status by serum concentrations of these nutrients (or GSH for selenium) may not accurately reveal this decline in selenium and vitamin E.

Requirements and Supplementation

Review of the literature would suggest the selenium requirement for most species of livestock is between 0.05-0.3 ppm (mg/kg) of dietary dry matter. 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.

The National Research Council (NRC) estimated vitamin E requirements for beef cattle, dairy cattle and sheep range from 15-40 ppm. There is much controversy. However, recent research suggests that relatively high levels of supplemental vitamin E may improve performance and that "mega-dose" levels of supplemental vitamin E may improve carcass quality.

The need for supplementation of selenium and/or vitamin E for ruminants would be dependent on the requirement of individual species, under different conditions of production, in relation to available selenium and vitamin E in feed sources. A series of studies have been undertaken to determine the utilization of different chemical forms of vitamin E by sheep and cattle (Hidiroglou et al., 1988a,b). The vitamin E form of d-α-tocopherol and its acetate ester was of higher biological value than racemic products, with greatest response occurring with d-α-tocopherol. Factors of primary importance to influence selenium and vitamin E supplementation include: (1) selenium and/or vitamin E deficient concentrates and roughages; (2) excessively dry ranges or pastures for grazing livestock; (3) confinement feeding where selenium 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 selenium (i.e., heavy metals, aflatoxines, 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 selenium and/or vitamin E; (7) accelerated rates of gain, production and feed efficiency may increase metabolic demands for selenium and vitamin E; and (8) intensified production may also indirectly increase selenium and vitamin E needs of animals by elevating stress, which often increases susceptibility to various diseases (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. A vitamin E supplementation program utilizing both parenteral and oral administration is often suggested. Injectable vitamin E (with or without selenium) is available for calves and lambs at birth and two to four weeks after birth. Cows and ewes should receive the product two weeks prior to parturition. The product is also indicated for early arrival feedlot cattle and lambs as well as those animals which are not consuming adequate vitamin E supplemented diets or which are showing signs of a vitamin E deficiency. For continued protection of all cattle and sheep from a possible vitamin E deficiency, adequate supplemental vitamin E in the feed is essential. Roche recommended vitamin E fortification guidelines are shown in Table 5.

For feedlot animals and dairy cows that receive concentrates, the most efficient method of providing supplemental selenium is through use of selenium-containing mineral supplements that are combined with concentrate feeds. Typically these complete diets would be formulated to contain 0.1-0.3 ppm of supplemental selenium. The principal methods of increasing selenium intake by grazing livestock include 1) a free-choice selenium mineral supplement, 2) selenium fertilization, 3) injections of selenium, 4) as a drench, and 5) selenium ruminal pellets (heavy boluses) (McDowell, 1991). A recent advance has been the development of an intraruminal osmotic pump, which actively disperses selenium at a rate of 3 mg per day (Campbell et al., 1990).

In areas where soils are low in selenium, certain agricultural practices may have some effect in increasing the level available. Applying manure to low-selenium soils from animals fed imported selenium-adequate feeds increases the soil selenium content slightly. Liming some soils deficient in selenium results in only a very small increase in selenium uptake by plants. The direct approach to increasing intake by animals through use of a selenium-containing fertilizer or spraying of the foliage with selenium compounds was not until recently widely practiced because the added selenium 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, 1-2 ozs/acre (as sodium selenite) should present no hazard. Fertilizer application with selenium to crops in New Zealand and Finland has increased selenium in meat by-products (Rimmer et al., 1990; Ekholum et al., 1990).

References

Adams, R.C. 1982. Feedlot cattle need supplemental vitamin E. Feedstuffs 54(18):24.

Barton, C.R., and W.M. Allen. 1973. Possible paralytic myoglobinuria of unknown etiology in young cattle. Vet. Rec. 92:288.

Boyne, R., and J.R. Arthur. 1979. Alterations in neutrophil function in selenium-deficient cattle. J. Comp. Pathol. 89:151.

Campbell, D.T., J. Mass, D.W. Weber, O.R. Hedstrom and B.B. Norman. 1990. Safety and efficacy of two sustained-release intrareticular selenium supplements and the associated placental and colostral transfer of selenium in beef cattle. Am. J. Vet. Res. 51:813.

Cipriano, J.E., J.L. Morrill and N.V. Anderson. 1982. Effect of dietary vitamin E on immune responses of calves. J. Dairy Sci. 65:2357.

Combs, S.B., and G.F. Combs Jr. 1987. Varietal differences in the vitamin E content of corn. In: Proc. 1984 Cornell Nutrition Conference for Feed Manufacturers. Ithaca, New York.

Ekholm, P., M. Zlinin, P. Koivistornen and P. Varo. 1990. Effects of general soil fertilization with sodium selenate in Finland on the selenium content of meat and fish. J. Agr. and Food Chem. 38:695.

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

Faustman, C., R.G. Cassens, D.M. Schaefer, D.R. Buege, and K.K. Scheller. 1989a. Vitamin E supplementation of Holstein steer diets improves sirloin steak color. J. Food Sci. 54(2):485.

Faustman, C., R.G. Cassens, D.M. Schaefer, D.R. Buege, S.N. Williams and K.K. Scheller. 1989b. Improvement of pigment and lipid stability in

Holstein steer beef by dietary supplementation with vitamin E. J. Food Sci. 54(4):858.
Gill, D.R., R.A. Smith, R.B. Hicks and R.L. Ball. 1986. The effect of vitamin E supplementation on the health and performance of newly arrived stocker cattle. Ok. Agr. Exp. Sta. Res. Rep. MP 118:240.
Golub, M.S. and M.E. Gershwin. 1985. Stress-inducted immunomodulation: what is it, if it is? In: G.P. Moberg (Ed.), Animal Stress. Am. J. Physiol. Soc., Bethesda, MD, USA.
Guss, S.B. 1977. Management and Diseases of Dairy Goats. Dairy Goat Journal Publishing Corporation, Scottsdale, Arizona.
Hadden, J.W. 1987. Neuroendocrine modulation of the thymus-dependent immune system. Ann. N.Y. Acad. Sci. 496:39.
Harrison, J.H., D.D. Hancock, and H.R. Conrad. 1984. Vitamin E and selenium of the dairy cow. J. Dairy Sci. 67:123.
Hartley, W.J., and A.B. Grant. 1961. A review of selenium responsive diseases of New Zealand livestock. Fed. Proc. Fed. Am. Soc. Exp. Biol. 20:679.
Hidiroglou, N., L.F. LaFlamme and L.R. McDowell. 1988a. Blood plasma and tissue concentrations of vitamin E in beef cattle as influenced by supplementation of various tocopherol compounds. J. Anim. Sci. 66:3227.
Hidiroglou, N., L. R. McDowell and R. Pastrana. 1988b. Bioavailability of various vitamin E compounds in sheep. Intl. J. Vit. Nutr. Res. 58:189.
Hidiroglou, M., A.J. McAllister and C.J. Williams. 1987. Prepartum supplementation of selenium and vitamin E to dairy cows: assessment of selenium status and reproductive performance. J. Dairy Sci. 70, 1281.
Hidiroglou, M., I. Hoffmann and K.J. Jenkins. 1969. Selenium distribution and radiotocopherol metabolism in the pregnant ewe and fetal lamb. Can. J. Physiol. Pharmacol. 47:953.
Hoffmann-La Roche. 1989. Vitamin Fortification Guidelines. Update No. 2. Animal Health and Nutrition, Hoffmann-La Roche Inc., Nutley, NJ.
Hogan, J.S., K.L. Smith, W.P. Weiss, D.A. Todhunter and W.L. Schockey. 1990. Relationships among vitamin E, selenium and bovine blood neutrophils. J. Dairy Sci. 73:2372.
Julien, W.E., H.R. Conrad, J.E. Jones and A.L. Moxon. 1976. J. Dairy Sci. 59:1954.
Koller, L.D., G.A. Whitbeck and P.J. South. 1984. Transplacental transfer and colostral concentrations of selenium in beef cattle. Am. J. Vet. Res. 45:2507.
Lee, R.W., R.L. Stuart, K.R. Perryman and K.W. Ridenour. 1985. Effect of vitamin supplementation on the performance of stressed beef calves. J. Anim. Sci. 61(Supp. 1):425.
McDowell, L.R. 1985. Nutrition of Grazing Ruminants in Warm Climates. Academic Press, Inc., New York.
McDowell, L.R. 1989. Vitamins in Animal Nutrition. Academic Press, Inc., New York.
McDowell, L.R. 1991. Minerals in Animal Nutrition. Academic Press, Inc., New York (accepted for publication).
McDowell, L.R., J.H. Conrad, G.L. Ellis and R.M. Mason Jr. 1985. Copper, selenium and zinc supplementation for grazing cattle. In: Proc. Florida Nutrition Conference, p. 79. Univ. of Fla., Gainesville.
McDowell, L.R., Y. Salih, J.F. Hentges and C.J. Wilcox. 1990. Selenium supplementation and concentrations of selenium in cattle tissues and fluids. Nutrition Research 10:793.

McMurray, C.H., and D.A. Rice. 1982. Vitamin E and selenium deficiency diseases. Irish Vet. J. 36:57.

McMurray, C.H., D.A. Rice and W.J. Blanchflower. 1980. Changes in plasma levels of linoleic acids in calves recently introduced to spring pasture. Proc. Nutr. Soc. 39:65A (Abstr).

Morris, J.G., W.S. Cripe, H.L. Chapman Jr., D.F. Walker, J.B. Armstrong, J.D. Alexander Jr., R. Miranda, A. Sanchez Jr., B. Sanchez, J.R. Blair-West, and D.A. Denton. 1984. Selenium deficiency in cattle associated with Heinz bodies and anemia. Science 223:491.

Nockels, C.F. 1989. Stress, disease and vitamin E requirements. Proc. Twentieth Annual Convention of American Assoc. of Bovine Prac. Kansas City, MO, USA.

Oldfield, J.E. 1990. Uses of selenium in agriculture, environment: an update. Feedstuffs 62(46):11.

Pruiett, S.P., J.L. Morrill, F. Blecha, P.G. Reddy, J. Higgins and N.V. Anderson. 1989. Effect of supplemental vitamin C and E in milk replacer on lymphocyte and neutrophil function in bull calves. J. Anim. Sci. 67(Suppl. 1):243.

Reddy, P.G., J.L. Morrill, H.C. Minocha and J.S. Stevenson. 1987. Vitamin E is immunostimulatory in calves. J. Dairy Sci. 70:993.

Rice, D.A., W.J. Blanchflower and C.H. McMurray. 1981. Reproduction of nutritional degenerative myopathy in the post ruminant calf. Vet. Record 109:161.

Rimmer, D.L., R.S. Shiel, J.K. Syers and M. Wilkinson. 1990. Effects of soil application of selenium on pasture composition. J. Sci. Food and Agr. 51:407.

Smith, K.L., and H.R. Conrad. 1987. Vitamin E and selenium supplementation for dairy cows. Proc. Roche Technical Symposium, The Role of Vitamins on Animal Performance and Immune Response, Daytona Beach, FL, USA. RCD 7442.

Smith, K.L., J.H. Harrison, D.D. Hancock, et al. 1984. Effect of vitamin E and selenium supplementation on incidence of clinical mastitis and duration of clinical symptoms. J. Dairy Sci. 67:1293.

Swecher Jr., W.S., D.E. Eversole, C.D. Thatcher, D.J. Blodgett, G.G. Schurig and J.B. Meldrum. 1989. Influence of supplemental selenium on humoral immune response in weaned beef calves. Am. J. Vet. Res. 50:1760.

Underwood, E.J. 1981. The Mineral Nutrition of Livestock. Commonwealth Agricultural Bureaux, London.

Van Saun, R.J. 1990. Rational approach to selenium supplementation essential. Feedstuffs 62(3):15.

Van Saun, R.J., T.H. Herdt and H.D. Stowe. 1989a. Maternal and fetal selenium concentrations and their interrelationships in dairy cattle. J. Nutr. 119:1128.

Van Saun, R.J., T.H. Herdt and H.D. Stowe. 1989b. Maternal and fetal vitamin E concentrations and Se-vitamin E interrelationships in dairy cattle. J. Nutr. 119:1156.

Weiss, W.P., J.S. Hogan, K.L. Smith, and K.H. Hoblet. 1990. Relationships among selenium, vitamin E and mammary gland health in commercial dairy herds. J. Dairy Sci. 73:381.

Wolf, E., V. Kollonitsch and C.H. Kline. 1963. J. Agr. Food Chem. 11:355.

TABLE 1. Effect of selenium supplementation on serum and milk selenium concentrations (ug/ml) of Brahman cattle[a]

	Sample No. per Treatment	Control	Injectable and Dietary Selenium[b]	Dietary Selenium[c]
Cow serum	66	0.024[d]	0.055[e]	0.045[e]
Colostrum	24	0.015[d]	0.049[f]	0.031[e]
Milk	44	0.0046[d]	0.0097[e]	0.0062[e]
Calf serum	58	0.030[d]	0.053[e]	0.049[e]

[a] McDowell et al. (1989).
[b] Control + 0.25 ppm Se + intramuscular injection of 5 mg Se plus 1,500 mg vitamin E.
[c] Control + 0.25 ppm selenium.
[d-f] Means in the same column with different letters in their superscripts differ (P<0.05).

TABLE 2. Effect of dietary vitamin E on plasma α-tocopherol concentration and immune response of young calves[a]

Item	Treatment Group[b]		
	-E	+E	Control
Plasma α-tocopherol, mg/dl	.071[c]	.639[d]	.155[c]
Serum immunoglobulin, mg/dl			
IgG1	1079	1168	1315
IgG2	488	562	432
IgM	151	118	100
IgA	37	53	85
Lymphocyte stimulation index	76	220	152

[a] From Cipriano et al. (1982).
[b] -E = vitamin E deficient diet, no supplemental vitamin E; +E = vitamin E deficient diet, 100 mg supplemental vitamin E per head per day; control = no supplemental vitamin E, conventionally managed.
[c,d] Means with different superscripts differ (P<.05).

TABLE 3. Effect of vitamin supplementation on 28-day performance of stressed beef cattle[a]

Treatment	Average Daily Gain (kg)	Average Daily Feed Intake (kg as fed)	Feed Conversion
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 4. 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	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 5. Recommended vitamin E fortification guidelines for cattle and sheep[a]

Class	Vitamin E Activity (IU/head/day)
Calves	200-400
Feedlot cattle (growing and finishing)	200-500
Beef cows and bulls	150-340
Fattening lambs	50-80
Dairy cows	500-1200[b]

[a] From Hoffmann-La Roche (1989).
[b] Dry cows should be supplemented 1000 IU vitamin E per head per day.