

ROLE OF DIETARY ANTIOXIDANTS VITAMINS AND MINERALS IN RUMINANTS – A REVIEW

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Antioxidant vitamins and minerals are natural compounds that help protect the body from harmful free radicals. Free radicals are atoms or groups of atoms that can cause damage to cells, impairing the immune system and leading to infections and various degenerative diseases. The best known antioxidant vitamins and minerals are vitamin A, beta-carotene, vitamins C and E, the minerals Selenium, Zinc, Manganese, Copper, and Iron. The body uses these elements to make free radical enzyme scavengers; which neutralize the free radicals. The present paper describes these nutrients in relation to their antioxidant activity and also explain the need of dietary supplementation of such vitamin and mineral in ruminants for maintenance of health and productivity.

Keywords: Antioxidant, Vitamins, Minerals, Ruminant

Vitamin E, vitamin C, Carotenoids, Selenium and other trace minerals are important antioxidant components of animal diets and their roles in animal health and immune function are indispensable as free radical scavengers (Rifici and khachadurian,1993, Baldi et al.,2000).

Free radicals are atoms or molecules containing one or more unpaired electrons, making them very “reactive” (Badwey, 1980). The extreme reactivity of these free radicals is driven by a desire to acquire another electron and thus impart ability to interact with cellular components such as DNA, proteins, lipids and carbohydrates and ultimately leads to tissue damage. Although, the term ‘free radical’ collectively includes reactive oxygen species (ROS) and reactive

nitrogen species (RNS), but some non-radical reactive derivatives of oxygen and nitrogen are also considered to be free radicals (Badwey and Karnovsky,1980).

Free radicals (ROS/RNS) are constantly produced during normal physiological metabolism in tissues. In the process, oxygen is utilized within the mitochondria to generate ATP. As a result, the majority of oxygen is reduced to form water. However, a small quantity (2-5%) is not completely reduced resulting in the formation of reactive oxygen intermediates. The super oxide radical is the main free radical produced in living cells and the electron transport chain in the mitochondria is considered to be responsible for it. Free radicals generated naturally during oxidative metabolism if present in excess react with fatty acids to form fatty acid hydroperoxides which can then induce a chain reaction forming further free radicals and hydroperoxides: if the chain reaction is not terminated cell damage will occur. In addition, the activation of macrophages in stress conditions is another important source of free radical generation. Immune cells produce ROS/RNS and use them as an important weapon to destroy pathogens (Schwarz, 1996).

Thus, under normal conditions the deleterious effects of ROS/RNS are counteracted by the body’s antioxidant defenses, which are contributed to through dietary intake of key nutrients like vitamins and trace minerals. Antioxidants serve to stabilize these highly reactive free radicals, thereby maintaining the structural and functional integrity of cells. Therefore,

antioxidants are very important to immune defense and health of humans and animals. Oxidative stress occurs when the production of reactive oxygen metabolites exceeds the capacity of the antioxidant system of the cell, tissue or body. As the free radicals are toxic to cells, the body is attributed by a sophisticated antioxidant system that depends on antioxidant nutrients. Tissue defense mechanism against free-radical damage generally includes vitamin C, vitamin E, and β -carotene (and other carotenoids) as the major vitamin antioxidant sources. Further, several metalloenzymes such as glutathione peroxidase (Se), catalase (Fe), and superoxide dismutase (Cu, Zn, and Mn) are also critical in protecting the internal cellular constituents from oxidative damage. The animal body synthesizes these antioxidant enzymes only when these metals are delivered in the diet in sufficient amounts. In contrast, deficiency of such elements causes oxidative stress and damage to biological molecules and membranes. There is a delicate balance between the amount of free radicals generated in the body and the antioxidants needed to provide protection against them. An excess of free radicals, or lack of antioxidant protection, can shift this balance resulting in oxidative stress. The dietary and tissue balance of all these nutrients (vitamins and trace elements) are important in protecting tissues against free-radical damage as well as participating in immune function.

Antioxidant vitamins and selenium

The principal antioxidant vitamins for tissue defense against free-radical damage includes vitamins E, C and β -carotene. Selenium would be the mineral most specifically related to antioxidant function. There are at least 35 antioxidant Se proteins and include selenoprotein P, five glutathione peroxidases and three thioredoxin reductases (TrXR1). Most recent research indicates TrXR1 reduces harmful ROS and facilitates gene expression of other cytoprotective antioxidants (Nakamura, 2005).

Vitamin E functions as a membrane-bound antioxidant, trapping lipid peroxyl free radicals produced from unsaturated fatty acids under conditions of 'oxidative stress'.

It is a chain-breaking antioxidant, neutralizing free radicals and preventing oxidation of lipids within membranes (Baldi et al, 2000). Vitamin E serves as the first line of defense against peroxidation of phospholipids. Selenium as part of glutathione peroxidase (GSH-Px) is the second line of defense as the enzyme destroys peroxides and hydroperoxides. The various GSH-Px enzymes are characterized by different tissue specificities and are expressed from different genes. In general, different forms of GSH-Px perform their protective functions in concert, with each providing antioxidant protection at different sites of the body. The principal vitamin E form with antioxidant and immune functions is α -tocopherol. However, although studies are limited, non α -tocopherol and tocotrienols have important functions. α -tocopherol has been shown to be a more effective inhibitor of peroxy nitrite-induced lipid peroxidation (McCormick and Parker, 2004). Moreover, α -tocopherol is found to be more effective in inhibiting inflammatory reactions. Similarly, Tocotrienols possess excellent antioxidant activity *in vitro* and have been found to be efficient suppressor of ROS than tocopherols (Schaffer et al., 2005). Further, tocotrienols were found to be more effective for reducing the ageing process and age-related diseases. In the lipid environment of biological membranes a combination of carotenoids and other antioxidants, especially tocopherols, may provide better antioxidant protection than tocopherols alone. Antioxidant properties of carotenoids include scavenging singlet oxygen and peroxyl radicals, sulfur, thiyl, sulfonyl and NO₂ radicals and provide protection of lipids from superoxide and hydroxyl radical attack (Schaefer et al, 1995). Therefore, carotenoids can actively quench singlet oxygen (O₂) and prevent lipid peroxidation caused by O₂ and can intercept the propagation step of lipid peroxidation. One molecule of β -carotene is able to quench 1000 molecules of O₂ before it reacts chemically and forms products. Although the principal antioxidant carotenoid is β -carotene, other carotenoids like lutein, lycopene and zeaxanthin) have strong antioxidant activities. Vitamin C is the most

important antioxidant in extracellular fluids and can protect bio-membranes against lipid peroxidation damage by eliminating peroxy radicals in the aqueous phase before the latter can initiate peroxidation. Vitamin C is located in the aqueous phase of cells, where it contributes to radical scavenging. Vitamin C is a potent antioxidant; it easily gives up electrons to provide stability to the reactive oxygen (ROS).

Mechanisms of action of antioxidants

Antioxidants are effective via various mechanisms viz. 1) preventive antioxidants, 2) free radical scavengers, 3) sequestration of elements by chelation and 4) quenching active oxygen species (Weisburger, 1999).

- *Preventive antioxidants:* These antioxidants suppress formation of free radicals; e.g. catalase (Fe containing) and glutathione peroxidase (Se containing). These two antioxidant enzymes, decompose hydrogen peroxide, preventing the formation of oxygen radicals.
- *Free radical scavengers:* These antioxidants confer stability to the 'reactive' species by donating an electron and become oxidized themselves to form a more stable radical. e.g. α -tocopherol (vitamin E) scavenges peroxy radicals and is converted to a tocopherol radical.
- *Sequestration of metal by chelation:* Although trace minerals are important dietary constituents, they can act as pro-oxidants (promote free radical formation). Since trace minerals such as Fe and Cu can propagate the formation of more reactive radicals they are kept bound to transport proteins such as transferrin or ceruloplasmin, as these render them less available to contribute to radical or pro-oxidant formation.
- *Quenching of active oxygen species:* Antioxidants can convert active oxygen species to more stable forms, for example, carotenoids and vitamin E stabilize singlet oxygen radicals, forming less reactive hydrogen peroxide.

Role of antioxidants in stress conditions

In equilibrium, free radical generation is neutralized by the antioxidant system. Natural and synthetic antioxidants in the feed as well as optimal levels of Mn, Cu, Zn, Fe and Se help to maintain the efficient levels of endogenous antioxidants in the tissues. Optimal nutrient composition allows the food antioxidants to be efficiently absorbed and metabolized. Optimal temperature, humidity and other environmental conditions are also needed for effective protection against free radical production. Prevention of different diseases by antibiotics and other drugs is an integral part of the efficient antioxidant defense as well. Different stress conditions are associated with overproduction of free radicals and cause oxidative stress i.e., a disturbance in the pro-oxidant-antioxidant balance leading to potential tissue damage. Stress conditions can be generally divided into three main categories: nutritional, environmental and disease related.

The most important part is nutritional stress conditions including high levels of PUFAs, deficiencies of vitamin E, Se, Zn or Mn, Fe-overload, hypervitaminosis A and presence of different toxins and toxic compounds. Other stress factors include environmental conditions like increased temperature or humidity, radiation, etc. The third group of stress factors is internal and includes various bacterial or viral diseases as well as allergy. All animals protect themselves from invasion of microorganisms, parasites, fungi, viruses and any foreign molecules. Phagocytosis is the major mechanism by which microbes are removed from the body and is especially important for defense against extracellular microbes. The mechanism of the phagocytic process is the killing of microbes by bombarding them with oxidants (superoxide and hydroxyl radicals, hydrogen peroxide, nitric oxide, etc.). Without adequate antioxidant nutrient reserves, cellular machinery will be damaged by the free radicals, thereby reducing the effectiveness of the immune cell. When antioxidant capacity is limited, the lifespan of immune cells is reduced and an infection can become established or severity of an infection can increase (Weiss,

2005). Intensified production increases stress and subclinical disease level conditions because of higher densities of animals in confined areas. Stress and disease conditions in ruminants may increase the basic requirement for certain vitamins and minerals. A number of studies indicate that nutrient levels that are adequate for growth, feed efficiency, gestation and lactation may not be adequate for normal immunity and for maximizing the animal's resistance to disease (Wuryastuti et al.,1993). Diseases or parasites affecting the gastrointestinal tract will reduce intestinal absorption of vitamins, both from dietary sources and those synthesized by microorganisms. If they cause diarrhoea or vomiting this will also decrease intestinal absorption and increase needs. Vitamin A deficiency is often seen in heavily parasitized animals that supposedly were receiving an adequate amount of the vitamin.

Mycotoxins in feed can substantially decrease antioxidant nutrient assimilation from the feed and increase their requirements to prevent damaging effects of free radicals and toxic products of their metabolism. It is now increasingly recognized that at least 25% of the world's grains are contaminated with mycotoxins. Mycotoxins are known to cause digestive disturbances such as vomiting and diarrhea as well as internal bleeding, and interfere with absorption of dietary vitamins A, D, E and K. Environmental pollutants (e.g. heavy metals, pesticides, fungicides, herbicides, etc.) can cause oxidative stress.

Role of antioxidant vitamins and trace minerals in immunity

Antioxidant vitamins generally enhance different aspects of cellular and non-cellular immunity. The antioxidant function of these vitamins could, at least in part, enhance immunity by maintaining the functional and structural integrity of important immune cells. A compromised immune system will result in reduced animal production efficiency through increased susceptibility to diseases, thereby leading to increased animal morbidity and mortality.

One of the protective effects of vitamin C may partly be mediated through its ability to reduce circulating glucocorticoids. The

suppressive effect of corticoids on neutrophil function in cattle had been alleviated with vitamin C supplementation. Vitamin C and E supplementation resulted in a 78% decrease in the susceptibility of lipoproteins to mononuclear cell-mediated oxidation (Rifici and Khachadurian, 1993). Ascorbic acid is reported to have a stimulating effect on phagocytic activity of leukocytes, on function of the reticulo-endothelial system, and on formation of antibodies. Ascorbic acid levels are very high in phagocytic cells, which uses free radicals and other highly reactive oxygen molecules to kill pathogens that invade the body. In the process, however, cells and tissues may be damaged by these reactive species. Ascorbic acid helps to protect these cells from oxidative damage.

Considerable attention is directed to the role of vitamin E and Se in protecting leukocytes and macrophages during phagocytosis, the mechanism whereby animals immunologically kill invading bacteria. Both vitamin E and Se may help these cells to survive the toxic products that are produced in order to effectively kill ingested bacteria (Badwey and Karnovsky, 1980). Macrophages and neutrophils from vitamin E-deficient animals had decreased phagocytic activity. Since vitamin E acts as a tissue antioxidant and aids in quenching free-radicals produced in the body, any infection or other stress factors may exacerbate depletion of the limited vitamin E stores from various tissues. With respect to immune-competency, dietary requirements may be adequate for normal growth and production; however, higher levels have been shown to positively influence both cellular and humoral immune status of ruminant species. During stress and disease, there is an increase in production of glucocorticoids, epinephrine, eicosanoids, and phagocytic activity. Eicosanoid and corticoid synthesis and phagocytic respiratory bursts are prominent producers of free radicals which challenge the animal antioxidant systems. Vitamin E has been implicated in stimulation of serum antibody synthesis, particularly IgG antibodies. Accordingly, vitamin E supplementation was observed to have increased -tocopherol

status and immune response of weaned piglets (Lauridsen and Jensen, 2005). The effects of vitamin E and Se supplementation on protection against infection by several types of pathogenic organisms, as well as antibody titers and phagocytosis of the pathogens have been reported for calves and lambs (Reffett et al., 1988). As an example, calves receiving 125 IU of vitamin E daily were able to maximize their immune responses compared to calves receiving low dietary vitamin E (Ready et al., 1987). Bovine respiratory disease (BRD) is the most economically important disease affecting feedlot cattle, causing approximately 75% of morbidity and over 50% of mortality. Rivera et al. (2002) suggest that vitamin E supplementation may enhance recovery from BRD. Interestingly when given as an adjuvant at vaccination, vitamin E is highly effective in enhancing antibody titers, implying that this may be an effective way of obtaining immunological response following vaccination. Cows given a single subcutaneous injection of 3,000 IU of vitamin E one week before expected calving tended to have reduced risk of retained placenta (LeBlanc et al., 2002).

Antioxidants, including vitamin E, play a role in resistance to viral infection. Vitamin E deficiency allows a normally benign virus to cause disease (Beck et al., 1994). In mice, enhanced virulence of a virus resulted in myocardial injury that was prevented with vitamin E adequacy. A Se or vitamin E deficiency leads to a change in viral phenotype, such that an avirulent strain of a virus becomes virulent and a virulent strain becomes more virulent (Beck, 1997).

Carotenoids have been shown to have biological actions independent of vitamin A. Recent animal studies indicate that certain carotenoids with antioxidant capacities, but without vitamin A activity, can enhance many aspects of immune functions, can act directly as antimutagens and anticarcinogens, can protect against radiation damage, and can block the damaging effects of photosensitizers. Also, carotenoids can directly affect gene expression and this mechanism may enable carotenoids to modulate the interaction between B cells and T cells, thus regulating

humoral and cell-mediated immunity. Vitamin A and β -carotene have important roles in protection against numerous infections including mastitis. Potential pathogens are regularly present in the teat orifice, and under suitable circumstances can invade and initiate clinical mastitis. Any unhealthy state of the epithelium would increase susceptibility of a mammary gland to invasion by pathogens. There are reports of improved mammary health in dairy cows supplemented with β -carotene and vitamin A during the dry and lactating periods (Chew and Johnston, 1985).

Polymorphonuclear neutrophils (PMN) are the major line of defense against bacteria in the mammary gland. β -carotene supplementation seems to exert a stabilizing effect on PMN and lymphocyte function during the period around dry off (Tjoelker et al., 1990). Daniel et al. (1991) reported that β -carotene enhanced the bactericidal activity of blood and milk PMN, against *S. aureus* but did not affect phagocytosis. Vitamin A either had no effect or suppressed bactericidal activity and phagocytosis. Control of free radicals is important for bactericidal activity but not for phagocytosis. The antioxidant activity of vitamin A is not important; it does not quench

or remove free radicals. β -carotene, on the other hand, does have significant antioxidant properties and effectively quenches singlet oxygen free radicals (Mascio et al., 1991).

Supplemental levels of vitamin E higher than recommended by the dairy cattle NRC (2001) have been beneficial in the control of mastitis. It is reported that intramammary infection is reduced by 42.2 % in vitamin E-Se supplemented versus unsupplemented controls. Clinical mastitis is negatively related to plasma Se concentration and concentration of vitamin E in the diet. A known consequence of vitamin E and Se deficiency is impaired PMN activity and postpartum vitamin E deficiencies are frequently observed in dairy cows. Dietary supplementation of cows with Se and vitamin E results in a more rapid PMN influx into milk following intramammary bacterial challenge and increased intracellular kill of ingested bacteria by

PMN. Subcutaneous injections of vitamin E approximately 10 and 5 days before calving successfully elevated PMN α -tocopherol concentrations during the periparturient period and negated the suppressed intracellular kill of bacteria by PMN that commonly is observed around calving. Plasma α -tocopherol decreased at calving for cows fed dietary treatments with low or intermediate concentrations of vitamin E, but not for cows fed high vitamin E treatment (Weiss et al., 1997). High dietary vitamin E increased concentration of α -tocopherol in blood neutrophils at parturition. The high vitamin E treatment was 1,000 IU/day of vitamin E during the first 46 days of the dry period, 4,000 IU/d during the last 14 day of the dry period, and 2,000 IU/day during lactation. (Weiss et al., 1997). Based on neutrophil function and clinical mastitis, the minimal plasma vitamin E level for peripartum dairy cows is 3.0 to 3.5 $\mu\text{g}/\text{mL}$. Somatic cell counts (SCC) are a primary indicator of mastitis and milk quality in dairy herds. Baldi et al. (2000) reported significantly lower SCC (log 10) in cows fed 2,000 IU/d supplemental vitamin E versus 1,000 IU/d from 14 d pre-calving through 7 d post calving in diets with 0.3 ppm added Se. Plasma and milk vitamin E levels were increased in cows fed 2,000 vs 1,000 IU/d. Taken together, these studies substantiate the cooperative roles of vitamin E and Se in maintaining mammary immune function and suggest that 2,000 to 4,000 IU vitamin E/d during the peripartum period can be beneficial to dairy cows in terms of udder health and milk quality (Seymour, 2001).

Effects of vitamin E supplementation

Supplementing vitamin E in well balanced diets has been shown to increase humoral immunity for ruminant in addition to increasing the antioxidant defense (Rivera et al., 2002; Wuryastuti et al., 1993). Gill et al. (1986) supplemented newly received feedlot cattle with 1,600 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 supplemented stressed cattle.

Baldi et al. (2000) supplemented cows with 1,000 or 2,000 IU vitamin E starting 14 d prepartum through 7 d postpartum and reported significant reduction in number of services per conception (1.3 vs. 2.2). Kim et al. (1997) used 120 Holstein cows to test the effects of injecting 500 IU vitamin E and 40 mg Se at 20 d prepartum on reproductive performance. The combination of vitamin E and Se significantly reduced the incidence of retained placenta (30 vs 13.3%) and days to first service (103 vs 60 d). Olson and Seidel (2000) reported that vitamin E enhanced the development of bovine embryos both *in vitro* and after their transfer to recipient cows. Selenium source for heat stressed dairy cows (Florida) were fed supplemental Se as yeast Se or as selenite for 81 days (beginning 26 d prepartum) (Thatcher, 2006). The benefits of organic Se were: 1) elevated plasma Se, 2) phagocytosis and killing activity of neutrophils increased, immunocompetence increased at parturition, milk yield increased, uterine health improved and 2nd service pregnancy rate increased.

Recommended vitamin and mineral allowances

The National Research Council (NRC) requirements for vitamins and minerals are usually close to minimum levels required to prevent deficiency signs and for conditions of health and adequate performance, provided sufficient amounts of all other nutrients are supplied. Allowances of vitamins and minerals are those total levels from all sources fed to compensate for factors influencing the needs of animals. These “influencing factors” include those that may lead to inadequate levels of dietary vitamins and minerals. Influencing factors can include stress, infectious diseases, parasites, biological variations, diet composition, and bioavailability of sources, stability and nutrient interrelationships. Mineral levels vary in feed ingredients because of crop location, fertilization, plant genetics, plant disease and weather. For example, corn and other grains will vary in their concentration of vitamins and minerals depending on varieties, soils, levels of fertilization, maturity at harvest, processing methods and storage. Intensive cropping

practices and use of new crop varieties may result in reduced levels of these nutrients in many feedstuffs. In forage crops, factors that favor production of lush green plants also favor production of higher levels of vitamins and minerals, as most plants decline in their vitamin and mineral content as they mature. Harvesting conditions, processing and storage will affect the vitamin and mineral content of both feedstuffs and supplements.

Vitamin Supplementation to ruminants

Supplementation allowances need to be set at levels that reflect different management systems and that are high enough to take care of fluctuations in environmental temperatures, energy content of feed, or other factors that might influence feed consumption or the vitamin requirements in other ways. Grazing ruminants generally only need supplemental vitamin A, if pastures are low in carotene, and possibly vitamin E (influenced by Se status). Vitamin D is provided by ultraviolet light activity on the skin, while all other vitamins can be provided by ruminal or intestinal microbial synthesis. Ruminants housed under more strict confinement conditions generally require vitamins A and E and may require vitamin D if deprived of sunlight. Additional supplemental vitamin E would be needed to stabilize the meat color of finishing animals. Under specific conditions, relating to stress and high productivity, ruminants may be benefitted by supplemental B vitamins, particularly thiamin and niacin. Biotin deficiency has been linked to lameness in cattle (Budras et al., 1997). Increased plasma biotin levels have been associated with hardness and positive conformational changes in bovine hooves. Future research may find a need for carnitine supplementation. Adding a complete B-vitamin mixture to cattle diets entering the feedlot during the first month can reduce stress and increase gains. In one study, supplemental B vitamins given feedlot calves tended to reduce morbidity of animals (Zinn et al., 1987). Apparently under stress conditions of feedlots, the microbial population in the rumen is not synthesizing certain B vitamins at adequate levels.

Mineral Supplementation required for grazing ruminants

Ruminants that depend primarily on pasture have different mineral supplementation needs than those receiving concentrates. Phosphorus is generally most limiting, with free-choice mineral supplements containing Ca, P, common salt and selected trace minerals (Seymour, 2001). The trace elements most likely to be deficient for grazing livestock are Cu, Co, I, Se and Zn. Iron is generally not required unless animals are losing blood due to parasites; Mn deficiency is restricted to few regions of the world. Animals in some tropical regions may benefit from S supplementation particularly if non-protein nitrogen (e.g., urea) supplements are fed. Magnesium supplementation is warranted when grass tetany is a problem. Potassium may be needed in winter or the dry season when forage potassium is less than required.

Mineral supplementation for concentrates fed ruminants

The best means of providing minerals to ruminants receiving concentrates would be to combine the minerals with the concentrate diets, including Ca, P and a trace mineralized salt (NaCl plus, Co, Cu, I, Fe, Mn, Se and Zn). Chromium has been shown to be beneficial to stressed calves (e.g., transport stress). The less dietary forage, the higher the level of Ca required. Some studies would recommend K supplementation for high-producing dairy cows and finishing ruminants when high quality K-rich forages are limited; Mg is often added to high concentrate diets. Sulfur often is needed when non-protein nitrogen (e.g., urea) is used. To prevent milk fever, dairy cattle need special diets that regulate the Ca and P concentration and increase acidity. Normally the Ca:P ratio is less important than that of monogastric diets, assuming the P concentration is adequate. Diets that limit forage in relation to concentrates need some mineral salts as buffers as well as individual elements. High-producing dairy cows, finishing cattle and sheep often receive high concentrate diets. Feeding large amounts of concentrate with small amounts of forage or with forages that have been fermented or finely chopped will decrease ruminant saliva

output and increase the acid load of these ruminants.

CONCLUSION

Oxidative stress occurs when production of free radicals exceeds the capacity of the antioxidant system of body cells. Certain nutrients serve as antioxidants or are components of antioxidant enzymes. Antioxidants include vitamins C and E, carotenoids, and antioxidant enzymes containing Se, Cu, Mn, Fe or Zn. Stress conditions that generate free radical production can be classified as nutritional (e.g., high PUFA and or excesses of certain minerals and vitamins), environmental (e.g., temperature and humidity) and internal (e.g., bacterial, viral and fungal diseases). Antioxidant nutrients play important roles in animal health by inactivating harmful free radicals produced through normal cellular activity and from various stressors. A compromised immune system will result in reduced animal production efficiency through increased susceptibility to disease, thereby leading to increased animal morbidity and mortality. A number of factors influence vitamin and mineral requirements and utilization and include: physiological make-up and production function; confinement rearing without pasture; stress, disease and adverse environmental conditions; vitamin antagonists, use of antimicrobial drugs and body vitamin reserves. Under commercial ruminant production conditions, vitamin and mineral allowances higher than NRC requirements may be needed to allow optimum performance. Generally, the optimum vitamin and mineral supplementation level is the quantity that achieves the best growth rate, feed utilization, health (including immune competency), and provides adequate body reserves.

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