# Comparative vitamin E requirements and metabolism in livestock

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Review article

# Comparative vitamin E requirements and metabolism in livestock

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**Summary** — It has been over 50 years since vitamin E was originally described as a lipid-soluble dietary constituent required for normal reproduction in rats. Vitamin E is recognized as an essential vitamin required for all classes of animals functioning predominantly as an intracellular antioxidant in maintaining the integrity of biological cell membranes. Although a wealth of information has been gathered on clinical signs of vitamin E deficiency, establishing its requirements for animals has been exceedingly difficult because of interrelationships with other dietary constituents. Vitamin E requirements for animals cannot be defined in isolation. Requirements are influenced by the amount and type of fat (particularly with monogastrics) and degree of fat oxidation in the diet; the presence of antioxidants; dietary selenium (closely interrelated with vitamin E), iron, copper, and sulphur amino acids, as well as the physiological status of the animal. Other factors to be considered in assessing vitamin E needs of animals under commercial production conditions include: a) variability of vitamin E content in feedstuffs; b) poor stability of vitamin E during processing and storage of feeds; and c) management practices resulting in overstressed animals. Information on the function of or requirements for vitamin E in animals is very incomplete. Estimated dietary vitamin E requirements for most animal species are in the range of 10–40 IU/kg of diet. Of particular concern is the lack of vitamin E requirement information regarding young dairy and beef calves. Although good experimental evidence indicates a benefical role of supplemental vitamin E above physiological levels on overall performance, enhanced immunocompetence and preservation of meat and milk products, levels of vitamin E required to produce these desired effects needs to be firmly established. Present estimated dietary requirements for vitamin E across species may need to be redefined as new information becomes available about the role this nutrient plays in growth, health and overall metabolism.

vitamin E / requirement / metabolism / supplementation

**Résumé — Comparaison entre les besoins en vitamine E du bétail et le métabolisme.** Il y a plus de 50 ans que la vitamine E a été décrite pour la première fois comme étant un lipide soluble présent dans l'alimentation et nécessaire pour la reproduction des rats. Il est reconnu que la vitamine E est une vitamine essentielle, nécessaire pour toutes les espèces animales et qui fonctionne principalement comme anti-oxydant en maintenant l'intégrité des membranes cellulaires biologiques.

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* Correspondence and reprints

*Bien qu'u.n très grand nombre d'informations ait été rassemblé en ce qui concerne les signes clini-
ques induits par la déficience en vitamine E, il a été excessivement difficile d'établir les besoins des
animaux en vitamine E du fait des interrelations avec les autres constituants alimentaires. Les be-
soins des animaux en vitamine E ne peuvent être définis isolément. Ces besoins sont influencés par :
1) la quantité et le type de graisse (particulièrement chez les monogastriques) et le degré d'oxydation
des graisses dans l'alimentation, 2) la présence d'anti-oxydants, 3) le sélénium alimentaire (en rela-
tion étroite avec la vitamine E), le fer, le cuivre et les acides aminés sulfurés, aussi bien que l'état
physiologique de l'animal. Les autres facteurs à considérer dans l'évaluation des besoins en vitamine
E chez les animaux élevés pour le commerce comprennent a) la variabilité du contenu en vitamine E
dans la nourriture, b) la faible stabilité de la vitamine E au cours du traitement et de la conservation
des aliments et c) des habitudes d'exploitation qui provoquent un stress important chez les animaux.
Les informations sur la fonction de la vitamine E et les besoins des animaux en cette vitamine sont
très incomplètes. Pour la plupart des animaux, les besoins en vitamine E alimentaire ont été estimés
à des doses allant de 10 à 40 UI/kg d'aliment. En particulier, on manque d'informations concernant
les besoins en vitamine E chez les jeunes vaches laitières et chez les veaux. Bien que de bonnes
preuves expérimentales indiquent qu'un supplément en vitamine E a un rôle bénéfique (au niveau
physiologique) sur les performances globales, l'augmentation de l'immunocompétence et la conserva-
tion de la viande et des produits laitiers, il est indispensable de s'assurer des taux de vitamine E né-
cessaires pour produire les effets désirés. Les besoins en vitamine E alimentaire estimés actuelle-
ment doivent être redéfinis en fonction des connaissances disponibles sur le rôle que joue ce
nutriment dans la croissance, la santé et le métabolisme en général.*

**vitamine E / besoin / métabolisme / supplémentation**

## INTRODUCTION

Existence of an anti-sterility vitamin was brought to light in the early 1920s when evidence was obtained that female rats reared on a diet containing all the vitamins known at the time failed to reproduce, although they were apparently normal in other respects (Mattill and Conklin, 1920; Evans and Bishop, 1922; Sure, 1924). Although rats would mate and conceive, pregnancy was invariably terminated by fetal death followed by resorption. The missing factor was characterized as a vitamin (Evans and Bishop, 1922), and designated vitamin E. Following its isolation and purification from wheat germ oil, Evans *et al* (1938), proposed the name tocopherol originating from the Greek "to bear offspring". Two years later its structure was elucidated (Fernholz, 1938) and shortly thereafter it was synthesized (Karrer *et al*, 1938).

The term "vitamin E" applies to a group of lipid soluble compounds known as tocopherols and tocotrienols possessing varying degrees of vitamin activity (antioxidants) of which D-$\alpha$-tocopherol is the most active (McDowell, 1989). During the period 1930–1950, multiple varied deficiency disorders in animals were reported to be cured by vitamin E including exudative diathesis and encephalomalacia in chicks; liver necrosis and mulberry heart disease in pigs; anemia in monkeys; steatitis in a number of animal species; white muscle disease and cardiomyopathy in sheep and cattle.

Vitamin E displays a great versatility in the range of deficiency signs among species and even within the same species. In a single species, the chick, 3 distinct vitamin E deficiency diseases have been noted, including exudative diathesis, encephalomalacia and muscular dystrophy. It was reported in 1944 that clinical signs in the

chick could be enhanced or suppressed by dietary changes unrelated to the vitamin E content of the diet (Dam, 1944).

Following the recognition that vitamin E was an essential nutrient for all species of animals, numerous interrelationships were identified later between it and other dietary factors such as selenium, synthetic antioxidants and sulfur amino acids in preventing many varied animal diseases while polyunsaturated fatty acids (linoleate series) could exacerbate deficiency states (Mason and Horwitt, 1972; Scott, 1978; Machlin, 1980; Combs, 1981; Machlin, 1984). These diseases include those prevented by vitamin E or certain synthetic antioxidants (eg, encephalomalacia in chicks, fetal death and resorption in rats and muscular dystrophy in rabbits); those prevented by vitamin E or selenium (eg, dietary liver degeneration in rats, exudative diathesis in chicks, and nutritional muscular dystrophy in lambs, calves and turkeys); and those prevented only by vitamin E (eg, testicular degeneration in rats, guinea pigs and chickens and nutritional muscular dystrophies in rabbits and pigs (Machlin, 1984; National Research Council, 1987). Vitamin E functions in at least 2 different metabolic roles: 1) as a fat soluble antioxidant; and 2) in one or more specific roles interrelated with the metabolism of selenium and sulfur amino acids (Scott, 1980).

Establishing vitamin E requirements for animals is exceedingly difficult to determine because of the interrelationships with dietary factors therefore, its requirement is dependent on dietary levels of polyunsaturated fatty acids (PUFA), antioxidants, sulfur amino acids and selenium (McDowell, 1989). Other factors influencing vitamin E needs of animals under commercial production conditions include: a) variability in vitamin E content in feedstuffs; b) poor quality feedstuffs supplying limited amounts of vitamin E; c) poor stability of vitamin E feedstuffs during processing and

storage; and d) management practices resulting in stressed situations.

Estimated dietary vitamin E requirements for most animal species are in the range of 10–40 IU/kg of diet. Generally, these requirements have been established by determining the level necessary to prevent vitamin E-deficiency signs. Recent findings have shown a beneficial effect with increased animal performance and immunocompetence following supplementation of vitamin E over its minimum requirement.

## METABOLISM OF VITAMIN E

The metabolism of vitamin E in relation to absorption, transport, storage and excretion has been summarized in a number of publications (Machlin, 1984; McDowell, 1989). Information on the absorption and excretion of tocopherols by farm animals is extremely sparse.

### Absorption and transport

Mechanism of absorption of vitamin E is similar to that of other fat-soluble vitamins (Weber, 1983). Its absorption is closely associated with that of fat, and is accelerated by the presence of bile. It is clear that species differ in their ability to absorb tocopherols, especially since great variation has been reported for a single species. Also, with labile substances like the tocopherols, a simple estimation of absorption based upon amounts present in the food and feces may be inaccurate.

In unpublished work with young calves, Blaxter and Brown (1952) indicated that about 25% of α-tocopherol added as the acetate to a diet of dried skimmed milk was excreted in the feces when the daily intake of the ester was from 25–100 mg. These and other metabolic studies have

shown that tocopherols are incompletely absorbed. The amount absorbed seems to depend on the requirement of the organism (Klatskin and Molander, 1952). Absorption and elimination also seem to depend on the amount in the diet. Rate of absorption of tocopherols depends on various factors, eg: 1) pancreatic enzymes; 2) bile acids; 3) pH level of the intestinal contents; 4) intestinal motility; 5) other food components, in particular the fatty acids (Simon-Schmoss et al, 1984).

Griffiths (1960) noticed a linear relation between the logarithms of serum tocopherol and dietary concentration of vitamin E in chickens. Only $\alpha$-tocopherol was identified. Gray (1959) found considerably lower tocopherol levels in rat plasma at high levels of tocopherol intake than Griffiths (1960) found in chicken serum.

The work of Dju et al (1950) with hens indicated that $\alpha$-tocopherol was absorbed to a much greater extent than the $\gamma$- and $\delta$-compounds. The serum $\alpha$-tocopherol values of 2 hens which received weekly supplements of 1.6 and 2.0 g $\alpha$-tocopherol were 20.0 and 20.1 mg per 100 ml, but 2 hens that received weekly supplements of 0.8 and 1.0 g $\delta$-tocopherol had serum tocopherol values of only 1.35 and 2.1 mg per 100 ml.

Tocopherol esters are hydrolyzed prior to absorption, and both bile and pancreatic juice are necessary for absorption to proceed (Gallo-Torres, 1970). These facts support the idea that free tocopherol is absorbed by diffusion from a mixed micelle of fatty acids, monoacyl glycerols, bile salts and acids, cholesterol and other fat-soluble vitamins. Maximal absorption occurs at the junction of the upper and middle thirds of the small intestine (Hollander et al, 1975). After crossing the brush border into intestinal mucosal cells, tocopherol is not re-esterified but is incorporated as the alcohol into chylomicrons in mammals and enters the plasma via

the lymphatic system (Behrens et al, 1982).

Desai et al (1965) showed, in confirmation of studies by Weber et al (1962) with rats, that L-$\alpha$-tocopherol (2S, 4'RS, 8'RS, synthetic) was absorbed as well as or better than the D-form (2R, 4'R, 8'R, natural) of the vitamin. It appeared, therefore, that the differences in biopotency must be due to differences in retention whereby the D-epimer is retained much better than the L-epimer in the blood and perhaps in other body tissues. These results indicated the existence of an active carrier of D-$\alpha$-tocopherol in the blood and tissues which has a greater affinity for the D-epimer than for L-$\alpha$-tocopherol. Recently it has been reported that specific binding proteins exist for $\alpha$-tocopherol in the cytosol and nuclei of rat liver tissue (Catignani and Bieri, 1977; Guarnieri et al, 1980; Prasad et al, 1981) as well as in human erythrocytes (Kitabchi and Wimalasena, 1983) which are fairly specific for the natural stereoisomer. Experiments by Desai et al (1965) and Scott (1965), comparing the oral administration of D-$\alpha$ and L-$\alpha$-tocopheryl acetates in the presence of graded levels of dietary selenium have indicated that selenium is involved in some unknown way in the retention of D-$\alpha$-tocopherol in plasma. It remains to be determined if the differences in plasma levels of D- and L-epimers of $\alpha$-tocopherol are the result of differences: 1) in the rate of excretion; 2) in the rate of destruction; 3) in the affinity of the epimers for specific carriers; or 4) in chemical activity influenced by structural configurations.

Gallo-Torres (1970) reported the obligatory role of bile for the intestinal absorption of vitamin E into the lymph of rats. Only negligible amounts of radioactivity could be detected in the thoracic duct lymph when both bile and pancreatic juice were absent from the duodenum.

Apparently only small amounts of tocopherol are transported from the intestine via the portal vein in mammals, whereas

all of the tocopherol absorption in birds occurs *via* the portal vein directly to the liver (Machlin, 1984).

The absorption of orally fed vitamin E follows the pattern of lipids in general and of fat-soluble vitamins in particular (Wiss *et al*, 1962; Desai *et al*, 1965). The specific site of absorption is not well established. The small intestine is thought to be the major site of absorption for tocopherol even though some absorption takes place from the stomach of nonruminants and the rumen of ruminants (Blaxter and Brown, 1952; Roles, 1967). Generally, in animals the uptake of vitamin E from the small intestine is lowered when tocopherol is fed in an oily form. The uptake is facilitated by bile salts (Simon *et al*, 1956). The presence of vitamin E in both the blood and lymph of animals suggests that absorbed vitamin E can be transferred by either the blood or lymphatic route (Roles, 1967).

Wiss *et al* (1962) were also able to establish a mathematical relationship between the logarithms of tocopherol intake and plasma and liver concentration in chickens fed high doses of D-α-tocopheryl acetate (2 000 to 20 000 IU/kg of feed). Using [$C^{14}$]-DL-α-tocopheryl acetate, they demonstrated that maximal liver concentration was reached only after several hours, and persisted longer than the synthetic antioxidant ethoxyquin which attained a maximum within 30 min before declining rapidly. Most of the tocopherol was bound to the structural components of the liver cells, primarily the mitochondria and microsomes.

Vitamin E is transported in the blood *via* lipoproteins. A rapid exchange among lipoprotein classes occurs with vitamin E after it enters the circulation *via* the chylomicrons. Since plasma tocopherol level is correlated with the total plasma lipid content, low density lipoprotein (LDL), the most common lipoprotein in human plasma, carries most of the plasma vitamin E. There

is also a rapid exchange between tocopherol in the erythrocyte membrane and lipoproteins such that approximately 20% of the plasma tocopherol concentration is carried by red blood cells. Delivery to other tissue cells appears to be in association with the receptor-mediated uptake of LDL (Traber and Kayden, 1984).

Type and composition of diet influence absorption of vitamin E from the gut. Pudelkiewicz *et al* (1960) reported that only about one-third of the D-α-tocopherol in alfalfa is available to chicks. The poor utilization is attributed to a fat-soluble compound in alfalfa that acts antagonistically to α-tocopherol, decreasing its availability. An antagonistic relationship also exists between absorption of vitamin E and polyunsaturated fatty acids (Bunyan *et al*, 1968).

### Storage

Vitamin E is stored throughout all body tissues with adipose tissue, liver and muscle representing the major storage deposits. Rates of depletion of α-tocopherol from tissue of animals given vitamin E deficient diets vary considerably from tissue to tissue within a particular species (Diplock, 1985). Studies on the depletion of α-tocopherol in tissues of rats varied considerably, with the fastest loss occurring in the plasma, liver and heart muscle, intermediate for testes and heart muscle, while the slowest loss was found in the adipose tissue over a 6-wk depletion period (Bieri, 1972). There appears to be 2 different pools of vitamin E in the body, a labile pool and a fixed pool which is retained for long periods of time.

### Excretion

The major route of excretion of vitamin E is fecal elimination. Usually less than 1% of

orally ingested vitamin E is excreted in the urine (Machlin, 1984). Excess tocopherol is eliminated in the feces. Simon *et al* (1956) observed that rabbits receiving a single dose of 10–15 mg 5-methyl-$C^{14}$-D-α-tocopherol succinate eliminated 65% of the dose *via* the feces in 3 days and 80% in 6 days; 90% of the radioactivity was identified as free α-tocopherol by isotope dilution. It was concluded that the vitamin was re-secreted into the intestinal tract from the blood or *via* the bile. Mellors and McBarnes (1966) demonstrated that no significant amount of either tocopherol or its metabolites was introduced into the lumen of the rat guts *via* the bile or through secretion from mucosal cells. By using $C^{14}$ α-tocopherol, Shantz (quoted by Harris and Ludwig, 1949) remarked that 80% of the vitamin E given in oil solution was excreted in the feces of rats.

Dju *et al* (1950) observed that chickens receiving 1 g of α-tocopherol/day for a prolonged period eliminated 75% of the ingested quantity unchanged *via* the feces by 24 h. In contrast, chickens receiving a diet supplemented with 17.5 and 35 mg of α-tocopherol/kg only eliminated about 23% in the feces (Pudelkiewicz *et al*, 1960).

### Vitamin E in feedstuffs

Natural tocopherols and tocotrienols are widely distributed in plants occurring mainly as free alcohols in lipid-containing fractions of green leaves and seeds. Estimates for vitamin E activity in animal feedstuffs are dependent upon the reliability and applicability of the assay procedure and also upon the relative biopotency of the various structural and epimeric forms of vitamin E (Ullrey, 1981). Early characterization for total vitamin E in feedstuffs was based on separation of tocopherols by column, paper or thin-layer chromatography, followed by a colorimetric reaction,

predominately the Emmerie and Engle method. In addition to the lack of separation of individual tocopherols, colorimetric determinations resulted in overestimating total vitamin E levels, due to interfering substances. Total vitamin E analysis of feedstuffs is of limited value in providing a reliable estimated of the biological value of the vitamin.

The advent of high pressure liquid chromatography (HPLC) has provided a method which offers sensitivity, rapidity and accuracy for the analysis of individual tocopherols (α, β, γ, δ) in a range of materials of biological origin including feedstuffs. Tocopherol concentrations shown in table I provide a more accurate evaluation of individual tocopherols (*via* HPLC) in a few selected feedstuffs.

As a result of α-tocopherol being the most biologically active isomer (table II) many nutritionists prefer listing only α-tocopherol in feedstuffs, though a great diversity exists in the proportion of individual tocopherols of plant origin (table I). The D-

**Table I.** Tocopherols in selected feedstuffs (ppm)[a].

| Feedstuff | α | β | γ | δ |
|---|---|---|---|---|
| Barley | 4 | 3 | 0.5 | 0.1 |
| Corn | 6 | –[b] | 38 | Tr[c] |
| Oats | 7 | 2 | 3 | – |
| Rye | 8 | 4 | 6 | |
| Wheat | 10 | 9 | – | 0.8 |
| Corn oil | 112 | 50 | 602 | 18 |
| Cottonseed oil | 389 | – | 387 | – |
| Palm oil | 256 | – | 316 | 70 |
| Safflower oil | 387 | – | 174 | 240 |
| Soybean oil | 101 | – | 593 | 264 |
| Wheat germ oil | 1 330 | 710 | 260 | 271 |

[a] Modified from Ullrey (1981). [b] No value reported. [c] Trace.

**Table II.** Biopotency of tocopherols[a].

| Abbreviation | Structure | Fetal resorption rate (%) | Hemolysis (rat) (%) | Muscle dystrophy (chicken) (%) |
|---|---|---|---|---|
| α | 5, 7, 8-Trimethyl tocol | 100 | 100 | 100 |
| β | 5, 8-Dimethyl tocol | 25-40 | 15-24 | 12 |
| γ | 7, 8-Dimethyl tocol | 1-11 | 3-20 | 5 |
| δ | 8-Methyl tocol | 1 | 0.3-2 | – |

[a] Modified from Machlin (1984).

α-tocopherol content of various animal feed ingredients is given in table III.

While various feedstuffs contain a wide variety of tocopherols and tocotrienols, only α-tocopherol appears in significant levels in blood and tissue of animals. Studies with steers fed diets containing varying levels of different vitamin E isomers results in only α-tocopherol being present in significant amounts in blood, liver and adipose tissue (Rice and McMurray, 1982). In calves and dairy cows, 92–97% of total blood vitamin E concentration was α-tocopherol (Pehrson and Hakkarainen, 1986). Similar findings were observed in swine fed normal mixtures of tocopherols and tocotrienols (Rice and McMurray, 1982). In view of the fact that α-tocopherol predominates in animal tissues, regardless of the presence of other isomers (β, γ, δ) in feeds, the α-tocopherol isomer should be considered in determining the vitamin E activity supplied by feedstuffs.

Table IV provides data on α-tocopherol content of feedstuffs and complete diets obtained in a survey of feedyards in the United States (Adams, 1982). The α-tocopherol content of shelled, rolled and high moisture corn was 36–63% lower than published values. In addition, milo and alfalfa hay values were 54 and 58% of those found in the literature, respectively.

**Table III.** α-Tocopherol content of feeds (ppm)[a].

| Source | Mean | Range |
|---|---|---|
| Alfalfa meal, dehydrated 17% protein | 73 | 28-121 |
| Alfalfa meal, sun-cured 13% protein | 41 | 18-61 |
| Alfalfa hay | 53 | 23-102 |
| Barley, whole | 36 | 22-43 |
| Brewer's grains, dried | 27 | 17-48 |
| Corn, whole | 20 | 11-35 |
| Cottonseed meal | 9 | 2-16 |
| Distiller's grains, dehydrated | 30 | 17-40 |
| Lard | 12 | 2-30 |
| Linseed meal | 8 | 3-10 |
| Milo | 12 | 10-16 |
| Molasses, cane | 5 | 3-9 |
| Oats, whole | 20 | 18-24 |
| Poultry by-producted meal | 2 | 1-4 |
| Sorghum, grain | 12 | 10-16 |
| Soybean meal, solvent process | 3 | 1-5 |
| Wheat, whole | 11 | 3-15 |
| Wheat, bran | 17 | 15-19 |

[a] Adapted from Bauernfeind (1980) and Ullrey (1981).

Vitamin E levels supplied by feedstuffs in commercial diets cannot be accurately estimated from earlier published vitamin E or tocopherol values. In addition, a wide variation exists in the vitamin E content within the same feedstuff. Varietal differences in α-tocopherol content of 42 different vari-

**Table IV.** $\alpha$-Tocopherol content of feedstuffs and rations[a].

| | No of samples | $\alpha$-Tocopherol | | | | Average IU found as % of published values |
| | | Average (Mg) | (IU) | Range (IU) | Published values [b] (IU) | |
| --- | --- | --- | --- | --- | --- | --- |
| | | per pound | | | | |
| Corn, shelled | 8 | 5.7 | 8.5 | 4.9–16.4 | 13.5 | 63 |
| Corn, rolled | 1 | 3.3 | 4.9 | – | 13.5 | 36 |
| Corn, high moisture[c] | 17 | 5.1 | 7.7 | 0.0–10.9 | 13.5 | 57 |
| Corn gluten feed | 1 | 1.1 | 1.6 | – | 8.1 | 20 |
| Corn, ground ear | 3 | 3.1 | 4.7 | 2.4–6.0 | – | – |
| Silage, corn | 13 | 5.5 | 8.2 | 3.7–17.3 | – | – |
| Cottonseed hulls | 1 | 3.0 | 4.5 | – | – | – |
| Milo (rolled) | 2 | 2.5 | 3.7 | 2.0–4.5 | 6.8 | 54 |
| Hay, alfalfa | 10 | 13.8 | 20.6 | 7.9–25.0 | 35.6 | 58 |
| Feedlot rations | 18 | 5.6 | 8.3 | 2.0–22.4 | -- | – |
| Sudan hay | 1 | 8.0 | 11.9 | – | – | – |

[a] Modified from Adams (1982). [b] Published values–*Atlas of Nutritional Data on US and Canadian Feed*–National Research Council–National Academy of Sciences (National Research Council, 1982). [c] High moisture corn corrected to 90% dry matter basis.

eties of corn have been reported, with an average of 4.2 IU/kg and a range from 1.7–6.9 IU/kg (Combs and Combs, 1984).

Vitamin E concentrations shown in tables of feed composition represent only average values with actual vitamin E levels of each feedstuff varying over a fairly wide range. Methods of analysis, as mentioned previously, account for some of the variability as well as differences resulting from processing and storage of feedstuffs.

### Stability of vitamin E in feedstuffs

In general, stability of tocopherols in feedstuffs is quite poor, arising from their susceptibility to destruction by oxygen, heat, moisture, oxidizing fats and certain trace elements (*eg*, Fe and Cu) with substantial losses in processed and stored as well as in manufacturing and storage of finished feeds. With concentrates, oxidation increases following grinding, mixing with minerals, the addition of fat (particularly polyunsaturated fats) and pelleting. For pelleted feeds, destruction of both vitamins E and A may occur if the diets are not sufficient in antioxidants to prevent their accelerated oxidation under conditions of moisture and high temperatures.

When harvested forage crops are exposed to natural sunlight there is quite a rapid decline in the tocopherol content the speed and extent of the decline depending on the method and duration of the drying process. For example, in one study up to 80% of the vitamin E was lost in haymaking (King *et al*, 1967), whereas silage making and rapid dehydration of forages retain most of the vitamin E content. Destruction of vitamin E in forages is influ-

enced by the stage of maturity at time of forage cutting as well as the period of time from cutting to dehydration. Losses during storage can reach 50% in one month, while drying in the swath can account for up to 60% loss within 4 days.

Artificial drying of grains results in a much lower vitamin E content. Artificially dried corn resulted in only 9.3 ppm α-tocopherol *versus* 20 ppm in undried corn (Young *et al*, 1975). The addition to grains of antifungal agents such as 1% propionic acid results in large losses of tocopherol content. Corn stored as acid-treated (propionic or an acetic–propionic mixture), high moisture corn contained a level of approximately one ppm of α-tocopherol whereas similar corn articificially dried following harvesting contained 5.7 ppm of α-tocopherol (Young *et al*, 1978).

## REQUIREMENTS OF VITAMIN E

### Pigs

In general, vitamin E usually is used to include 8 naturally occurring structurally related compounds (Kasparek, 1980; McDowell, 1989). The composition of common pig feedstuffs appears to indicate that the tocotrienols are much less important than the tocols (Bauernfeind and Cort, 1974), and that large quantities of tocols are available from vegetable oils. This, together with the accepted biopotencies of the various natural forms (Brubacher and Wiss, 1972) may influence the swine requirements for vitamin E. In all but a few exceptions, vitamin E nutrition of pigs has concerned α-tocopherol (Bratzler *et al*, 1950; Jensen *et al*, 1988b), even though γ-tocopherol is the most abundant isomer in corn (Green, 1958; Ullrey, 1981).

The vitamin E requirement for pigs cannot be defined in isolation. The require-

ment for vitamin E is influenced by the amount and type and degree of oxidation of fat in the diet, the presence of antioxidants, natural or synthetic, and the dietary selenium, iron, copper and sulfur amino acid levels (Dam, 1962; Tollerz, 1973; Najman *et al*, 1976; Draper, 1980; McDowell, 1989).

The addition of fat to swine diets is a way of increasing the caloric density of the diet (Freeman, 1983). However, when this fat is in the form of vegetable oil with a high polyunsaturated fatty acid level, the risk of formation of oxidation products is increased. Synthetic antioxidants can be added to prevent formation of these products in the diet, but the tocopherols, which are natural antioxidants can also serve this function, although this would reduce the amount available to the pig. Pigs eating diets containing polyunsaturated fatty acids have increased polyunsaturation at the tissue level (Koch *et al*, 1968; Castell and Falk, 1980). Adding vitamin E to swine diets has been shown to increase tissue vitamin E levels and to limit rancidity in pork products (Marusich, 1980).

The formation of peroxides and free radicals from polyunsaturated fatty acids in tissue can be damaging. Selenium functions as a component of glutathione peroxidase found in the cytosol and mitochondrial matrix, and as such participates in the enzymatic detoxification of peroxides and free radicals (Rotruck *et al*, 1973). Because of their chemical structure, tocopherols act as free radical scavengers at the cell membrane level (Ullrey, 1981; Hennig *et al*, 1987). This overlap in function explains the interrelationship in the nutrient requirement of selenium and vitamin E that has been well documented in swine (Bengtsson *et al*, 1978a, b; McCay and King, 1980).

The vitamin E requirements for swine has been set by the National Research Council (1988) as illustrated in table V. There may be several occasions when pig

metabolism may result in an increased requirements for vitamin E compared to other species. For example, the newborn pig is practically devoid of circulating antibodies until it receives colostrum and milk, and is therefore very susceptible to disease and infection (Payne and Marsh, 1962; Klobasa and Werhahn, 1981). This may explain the relatively high levels of vitamin E found in cow colostrum compared to cow's milk produced later (Malm et al, 1976; Young et al, 1977), since vitamin E has been shown to interact with the immune system and therefore to assist in resistance to disease and infection in a number of species (Nockels, 1979; Tengerdy

**Table V.** Vitamin E requirements (IU/day) for different classes of pigs (kg).

| | |
|---|---|
| Growing swine [a, b] | |
| 1–5 | 4 |
| 5–10 | 7 |
| 10–20 | 10 |
| 20–50 | 21 |
| ·50–100 | 34 |
| | |
| Adult boars [c] | |
| 162.5 | 41.8 |
| | |
| Gestation: gilts and sows [d] | |
| 120 | 39.6 |
| 162.5 | 41.8 |
| 182.5 | 44 |
| | |
| Lactation: gilts and sows [e] | |
| 145 | 96.8 |
| 165 | 116.6 |
| 185 | 134.2 |

[a] Assumes *ad lib* feeding, and 0.1 mg selenium/day, and level of performance defined by the National Research Council (NRC) (1988). [b] 1 mg of DL-$\alpha$-tocopherol = 1 IU. [c] Assumes feed intake of 1.9 kg, and level of performance defined by NRC. [d] Assumes feed intakes of 1.8, 1.9, and 2.0 kg, respectively, and level of performance defined by the NRC. [e] Assumes feed intakes of 4.4, 5.3, and 6.1 kg, respectively, and level of performance defined by the NRC.

et al, 1981). The high intake of vitamin E by the piglet between birth and weaning corresponds to a period of rapid fat accretion, the magnitude of which is almost unique in the animal world (Farnworth and Kramer, 1987). This build-up of body fat may require additional vitamin E to provide protection at the tissue level. It may be important to note that sow milk vitamin E levels can be affected by the dietary intake of the sow (Young et al, 1977; National Research Council, 1988;).

In the weaned pig the transition from high fat milk diets to low fat, high carbohydrate solid feeds is accompanied by enzymatic changes (Mersmann et al, 1973); feed consumption is characteristically low and loss of weight and diarrhea often occur (Holme, 1969; Seve, 1982; Ball and Aherne, 1987). At the same time a depletion of vitamin E body stores occurs (Mahan and Moxon, 1980). It has been suggested that increased vitamin E would be beneficial to the pig at this time.

Pigs can be exposed to a variety of stresses, including those related to environment, nutrition, management and housing. At the same time a host of seemingly unrelated conditions in swine such as stomach ulcers, poor appetite and growth, decreased resistance to disease and infection, and death have been attributed to "stress" (Kelley, 1980; Mitchell and Heffron, 1982; Belschner and Love, 1984; Griffin, 1989). It has been suggested that vitamin E requirements may be increased by a variety of stresses (Ullrey, 1981). Vitamin E may well have specific therapeutic uses, but ambiguous and improper use of the term stress (Fraser et al, 1975) may overestimate the benefits of vitamin E.

Recently, 2 areas of research have been reported to affect current thinking about vitamin E nutrition in swine. Several reports have demonstrated the beneficial effects of adding vitamin E above the recommended levels to swine diets, as a

means of enhancing the immune system of the pig. Both cell-mediated and humoral immunity have been improved in swine consuming increased levels of vitamin E (Ellis and Vorhies, 1976; Peplowski et al, 1981; Bonnette et al, 1988; Jensen et al, 1988a). Mechanisms for this enhancement have been proposed but have not been proven (Tengerdy et al, 1981).

Research with partitioning agents– either somatotropin or the β-adrenergic agonist–has shown that swine growth and body composition can be significantly altered. In particular, growth is increased, carcass protein is increased, and fat decreased (McKeith, 1987). If repartitioning agents become popular with the swine industry, the nutrient requirements (including that of vitamin E) of faster growing leaner pigs will have to be established (Easter, 1987).

## Poultry

Minimum poultry requirements of vitamin E as established by the Nutrition Research Council (National Research Council, 1984b) are shown in table VI. These values were established from research conducted with semi-purified selenium-adequate diets under ideal environmental conditions (Jensen and McGinnis, 1960;

Machlin and Gordon, 1962; Bartov and Bornstein, 1972; Combs and Scott, 1974). The minimum levels of vitamin E are those that will yield optimum growth in poultry or sustained maximum egg production. In establishing vitamin E level for practical diets it is necessary to consider several factors which may alter the dietary requirements: 1) possible genetic differences in requirements; 2) variations in absorbability of vitamin E; 3) destruction of vitamin E in the gastrointestinal tract or in the feed; 4) variation in the quantity of vitamin E transferred from breeder hen to chick; and 5) increase in requirement due to disease or other stress.

Heavy breeds of fowl were reported to be more susceptible to encephalomalacia than White Leghorn chicks (Howes and Hutt, 1952). This difference in susceptibility to vitamin E deficiency may result from the greater demands of a rapid growth rate or to differences in initial vitamin E stores. This should be given consideration when extrapolating requirement data obtained with birds of the White Leghorn breed to practical feed levels for other breeds of fowl.

The proportion of dietary vitamin E that is absorbed and retained is influenced by the identity of the vitamin E isomers present and by the nature and quantity of other ingredients of this feed.

**Table VI.** Vitamin E requirements of poultry (IU per kg) [a].

| Species | Age (wk) | Starter | Age (wk) | Grower | Layer | Breeder |
|---------|----------|---------|----------|--------|-------|---------|
| Fowl, Leghorn type | (0–6) | 10 | (7–20) | 5 | 5 | 10 |
| Fowler, broiler | (0–3) | 10 | (3–8) | 10 | – | – |
| Turkey | (0–8) | 12 | (9–24) | 10 | – | 25 |
| Quail | – | 12 | – | – | – | 25 |

[a] National Research Council (1984b).

The biological activities of the natural and L isomers of vitamin E determined according to their potency in preventing muscular dystrophy in chicks indicated relative potencies, compared with DL-α-tocopheryl acetate (= 1.0), of D-α-tocopherol = 1.46 to 1.67, L-α-tocopherol = 0.36, β-tocopherol = 0.12 to 0.32, δ-tocopherol = 0.05 to 0.07 and α-tocopherol = 0.02 (Bruggemann *et al,* 1963; Scott and Desai, 1964; Hakkarainen *et al,* 1984). The aggregate potency of the vitamin E in the barley oil was 0.49 (Hakkarainen *et al,* 1984). The availability of vitamin E from feedstuffs may be less than 100%; for barley grain, availability was 75% of that from barley oil (Hakkarainen *et al,* 1984). Earlier evaluations of liver storage of vitamin E from alfalfa did not separate potency from availability. Biological values of 0.25 to 0.34 have been reported for alfalfa (Bunnell, 1957; Pudelkiewicz *et al,* 1957).

A minimum dietary content of fat is required for efficient absorption of fat-soluble vitamins. At both marginal and adequate levels of dietary vitamin E, the plasma level of vitamin E increased linearly in broiler chicks as the dietary content of saturated animal fat was increased from 0 to 6% (Abawi *et al,* 1985). One aspect of the vitamin-E–selenium interrelationship concerns the adequacy of dietary selenium to support functional integrity of the pancreas and thereby to maintain normal digestion of fats (Thompson and Scott, 1970). Without adequate provision of this trace element (National Research Council requirement 0.10–0.25 mg/kg) the plasma level of vitamin E is depressed even at high (100 mg/kg) dietary vitamin levels (Thompson and Scott, 1969). As zinc (Lu and Combs, 1988) and other heavy metals (Kling *et al,* 1987) interfere with selenium uptake, excessive levels may reduce plasma vitamin E level and precipitate a deficiency condition (Lu and Combs, 1988). Bile is essential to micelle formation and may be a limi-

tation to lipid absorption in chicks at an age when bile production is not fully developed. Feedstuffs containing soluble gums such as barley, oats (glucan), rye (pectin) or guarmeal (galactomannen) may, through their ability to bind bile salts, reduce absorption of fats and fat-soluble vitamins (Vahouny and Cassidy, 1985). Administration of water-miscible forms of fat-soluble vitamins has partially overcome a growth depression encountered in young chicks on feeding high levels of naked oats (Cave *et al,* 1990).

Fats and oils are widely used to raise the metabolizable energy content of poultry feeds to complement high fiber feedstuffs included for their cost advantage, and less extensively to modify the lipid composition of eggs or poultry meat, so as to meet market demands for health-sensitive foods. The presence of fats containing polyunsaturated fatty acids (PUFA) involves the risk of the development of oxidative rancidity as the fatty acids become peroxidized in the feed or in the gastrointestinal tract. The problem is more serious with oils of high PUFA content and high levels of the more highly unsaturated fatty acids, *ie*, plant and fish oils, including animal–vegetable fat blends (Abawi *et al,* 1985). Of the 3 vitamin E deficiency conditions found in chickens, encephalomalacia occurs when the deficiency is associated with a high dietary PUFA level.

Characteristically, PUFA plant oils also have a high content of vitamin E, and little additional supplementation of the vitamin may be required (Jager, 1972). In fish oils, however, the ratio of vitamin E:PUFA is lower than most plant oils and particular fatty acids (*eg*, 22:6) are more highly unsaturated. The requirement for vitamin E in chick diets containing safflower oil, of high–74%–linoleic acid, was recommended at 0.3 mg of DL-α-tocopherol acetate per g of dietary oxidized oil for prevention of encephalomalacic ataxia (Dror and Bar-

tov, 1982). Thus diets of 3–7% fat would require 9–20 IU vitamin E per kg to be sufficient against the hazard of fat usage. In contrast, the addition of 24 IU vitamin E was recommended for a diet containing the more highly unsaturated fish oil at 2% of the diet (Singsen et al, 1955). Other synthetic antioxidants such as ethoxyquin may be used to prevent peroxidation of PUFA in oils and fats and are normally added to commercial oils to maintain quality during storage and on mixing into feeds. When included at 0.125 g/kg diet, ethoxyquin prevents encephalomalacia without, however, being effective against other vitamin E-deficiency conditions of the chicken.

Vitamin A is often added to poultry diets at a level several times that required to prevent deficiency signs in an ideal environment. This may be done either as a measure to anticipate risks of stress or to modify broiler pigmentation (Vahl and Van't Klooster, 1987). Vitamin A levels of 16 000 IU/kg and higher antagonized absorption of vitamin E, resulting in depressed plasma level and liver storage of vitamin E (Pudelkiewicz et al, 1964; Frigg and Broz, 1984; Vahl and Van't Klooster, 1987) and increased the incidence of encephalomalacia (Dror et al, 1980). Dietary supplementation of 30–50 IU vitamin E per kg increased plasma vitamin E very little. However, a moderate increase in plasma vitamin E was obtained at high dietary vitamin A levels when dietary E was increased by one order of magnitude to 100 IU/kg (Sklan, 1983).

Young chicks obtain vitamin E from their liver stores in addition to that available from the diet. The vitamin E content of the liver of a chick depends on that in the hatching egg (Bartov et al, 1965) which in turn is correlated with the vitamin E content of the breeder hen diet (Richter et al, 1986). Although a high liver content of vitamin E may be assured in the day-old chick by supplementation of the breeder diet to 10 IU/kg, its adequacy for prevention of en-

cephalomalacia may be put at risk by a high PUFA content of the egg resulting from the lipid in the breeder diet. Where unsaturated or oxidized oil is used in a breeder diet, a higher level of supplementation up to 35 IU/kg may be required (Bartov and Bornstein, 1980).

There are certain growth inhibiting effects identified with feedstuffs or with adventitious components of feed which impair the vitamin E status of poultry consuming them, by interfering with tissue metabolism of the vitamin. Examples are bean vicine, which increased liver peroxidation decreasing plasma level of vitamin E (Hintz and Hogue, 1964; Muduuli et al, 1982) and the grain mycotoxin T-2 which results in depressed plasma concentration of lipoprotein and vitamin E (Coffin and Combs, 1981). These and other secondary vitamin E deficiency conditions may or may not respond to an increase in dietary vitamin E.

A contingency which must be anticipated by poultry producers is that chicks will be exposed to infection. Unless protective mechanisms are operative, production losses will be incurred as a disease spreads through the flock. Development of primary immune organs of the chick is specifically dependent on vitamin E (Marsh et al, 1981). Furthermore, 2 phases of the subsequent functional development of immunity, clonal expansion of lymphocytes (Marsh et al, 1981, 1986) and lymphocyte production of antibody in response to challenge in both young (Tengerdy and Brown, 1977; Franchini et al, 1986) and adults (Jackson et al, 1978) may be enhanced by vitamin E at a level of intake of 100–300 IU/kg in the diet, which is well above that required to support full growth potential and prevent encephalomalacia. In comparison, the requirement of vitamin E for clonal expansion of lymphocytes in mammalian species, estimated by a dose–response technique, was 5-fold that required for pre-

vention of muscular dystrophy (Bendich et al, 1986). Whereas chicks displayed detrimental effects of excessive vitamin E intake at 4 000 IU/kg and above (Nockels et al, 1976), enhancement of immune response was obtained at 1–2 orders of magnitude less than this value. Determination of the level of vitamin intake giving optimum immune function has yet to be established for the various classes of poultry.

A final aspect relates to product quality in poultry meat. During cold storage muscle lipids continue to be subject to peroxidation to an extent depending on the PUFA and vitamin E contents in addition to temperature and time. A dietary level > 10 (Csallany et al, 1988) or 16.7 IU/kg vitamin E (Combs and Regenstein, 1980) has been recommended so as to obtain a level in the carcass of not less than 3 IU/g muscle (Marusich et al, 1975) that would ensure lipid stability.

## Cattle

The Nutrient Requirements of Dairy Cattle (NRC, 1989) and Beef Cattle (NRC, 1984a) estimate the vitamin E requirement for young calves to be between 15–60 IU/kg of dry matter intake (table VII).

Recent estimated requirements for vitamin E for young weaned calves have been proposed at 2.4 IU/kg body weight, and for calves 10–24 weeks of age at 3.4 IU/kg body weight (Reddy et al, 1987).

Several factors affect the amount of vitamin E required by calves including body stores, growth rate, stress conditions endured by confinement, diseases, weaning, transport as well as the interrelationships with dietary polyunsaturated fatty acids, selenium and vitamin A. Newborn animals generally have low blood tocopherol levels probably as a result of poor placental transfer of the vitamin (Paulson et al, 1968). Vitamin E is needed during the rapid growth phase of the newborn (Farrell, 1980). If conventional calf diets are limiting in vitamin E, intake by calves may not be adequate to provide a sufficient rate of deposition in newly-formed membranes to prevent free radical-initiated peroxidative changes, which may result in preclinical cases of muscular dystrophy.

Assessing requirements for vitamin E had been based predominately on growth rates or the amount of the vitamin needed to prevent clinical signs of deficiencies including nutritional muscular dystrophy. There is increasing evidence that marginal deficiencies of vitamin E occur in the field which seldom manifest themselves in easily detectable signs and are masked by poor health and decreased performance (Adams, 1982). Vitamin E supplementation of weaned heifers (8 months) for 6 months prior to breeding significantly increased pregnancy rate over controls (83 vs 33%, respectively) (LaFlamme and Hidiroglou, 1991). Age of first heat, breeding and calving were unaffected by vitamin E supplementation.

Nutritional status with respect to vitamin E is commonly estimated from plasma (or

**Table VII.** Vitamin E requirements for [a] dairy and [b] beef cattle (IU/kg of diet).

| | |
|---|---|
| *Dairy cattle* | |
| Lactating cows | 15 |
| Growing heifers | 25 |
| Calf milk replacer | 40 |
| *Beef cattle* | |
| Growing | 15–60 |
| Feedlots | – |
| Pregnant heifers and cows | – |

[a] National Research Council (1989); *Nutrient Requirements of Dairy Cattle.* [b] National Research Council (1984a); *Nutrient Requirements of Beef Cattle.*

serum) concentration (McDowell and Williams, 1991). There is a relatively high correlation between plasma and liver levels of α-tocopherol (and also between amount of dietary α-tocopherol administered and plasma levels). Plasma tocopherol concentrations of 0.5–1 μg/ml are considered low in most animals species, with less than 0.5 μg/ml generally considered a vitamin E deficiency. Adams (1982) reported that plasma tocopherol concentrations between 0.60 to 1.6 μg/ml were associated with calves diagnosed with nutritional muscular dystrophy. Serum α-tocopherol concentrations of 1.0 to 1.5 μg/ml were reported by McMurray and Rice (1982) as associated with clinical lesions of white muscle disease, with values < 2 μg/ml (0.2 mg/dl) considered deficient. Serum α-tocopherol concentrations > 4.0 μg/ml have been considered to indicate adequacy in adult cattle. Similarly, marginal vitamin E status in adult cattle was associated with plasma tocopherol concentrations between 2.0 to 3.0 μg/ml (Adams, 1982). A guideline from limited data on vitamin E status based on plasma concentrations of α-tocopherol is given for cattle in table VIII.

Recent studies with young calves have shown enhanced immune response with increased performance following supplementation with vitamin E (Tikriti, 1969; Reddy et al, 1986; St-Laurent et al, 1990). Heifer dairy calves had overall greater weight gains when fed typical calf diets supplemented with 125 or 250 IU of vitamin E/calf day over a 24-week period than unsupplemented calves (Reddy et al, 1985). Blood profiles for α-tocopherol, creatine kinase, glutamic oxaloacetic transaminase and lactic dehydrogenase enzymes indicated a deficiency status in unsupplemented calves (Reddy et al, 1985).

Trends toward a higher cell-mediated immune response was observed in dairy calves following 1 g supplementation of vitamin E/day (St-Laurent et al, 1990). Stud-

ies 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). Similar observations of improved gain and feed efficiency with stressed beef calves have been noted following supplementation of vitamin E (Goering et al, 1976; National Research Council, 1989). 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).

Optimal allowance of vitamin E for milking cows would be the amount that transfers an adequate amount of vitamin E into cow's milk for maintaining freshness and a pleasant taste until the milk is consumed. Even in refrigerated storage milk fat is prone to autoxidation by a free radical mechanism. Oxidized fat imparts to milk an off flavour which varies from metabolic to tallowy to cardboardy. Vitamin E is a natural antioxidant which appears to terminate the chain reaction of the free radical mechanism.

Fresh forage like pearl millet or late cut alfalfa, in quantities usually consumed by milking cows, provided 4–5 g of vitamin E

Table VIII. Plasma tocopherol levels and status in cattle [a].

| Nutritional status | Plasma (α-tocopherol, ppm) |
|---|---|
| Adequate | ≥ 4.0 |
| Minimal | 3.0–4.0 |
| Marginal | 2.0–3.0 |
| Deficient | ≤ 2.0 |

[a] Modified from Adams (1982).

daily (King, 1967). Milk of cows grazing good pastures in New Zealand contained 1.45 mg to > 2 mg α-tocopherol/l (Miller *et al*, 1973). On an average cow's milk contains 3.5% fat. Thus vitamin E content of cow's milk in New Zealand ranged from 4.0–5.0% fat, which is equivalent to that of human breast milk (George and Lebenthal, 1981).

Vitamin E content of forage stored as early cut hay or silage may decrease to less than one-third within a few months. Consequently in winter months ≈ 10% of the dairy farms in areas like the northern states of the USA may be providing processors with oxidized flavored milk. If most of the milk produced is for the fluid milk market, serious outbreaks of an off flavor in milk could result (St-Laurent *et al*, 1990). Transfer of dietary vitamin E to cow's milk is very low and diminishes with increased intake. About 1–2% of total vitamin E was transferred into milk of cows consuming about 600 mg α-tocopherol in basal ingredient diets plus 1 g/day D-α-tocopheryl acetate as a supplement. The efficiency of transfer decreased to 0.3–0.6% for a daily supplement of 16 g (Tikriti, 1969). To maintain 40–50 µg vitamin E/g milk fat, a cow producing 40 kg milk containing 3.5% fat and transferring 1% of dietary vitamin E into milk would require 5.6 to 7.0 g of DL-α-tocopheryl acetate daily. Thus the National Research Council (NRC, 1989) recommendation of 15 IU vitamin E/kg feed is highly inadequate (table VII). In recent studies 40–50 µg vitamin E/g milk fat was attained by feeding 400 IU DL-α-tocopheryl acetate/kg dietary dry matter (Atwal *et al*, 1991) *ie*, about 8 g/day, whereas supplementation of 0.700 and 3 000 IU vitamin E daily produced milk containing 12.8, 15.8 and 22 µg vitamin E/g fat, respectively (St-Laurent *et al*, 1990).

It may be noted that when cows are deriving a part of milk fat from body fat as in early lactation or from fats and oils fed to improve energy intake, milk fat will be higher in unsaturated fatty acids (Atwal *et al*, 1990, 1991). Consequently, more vitamin E would be required to control autoxidation. Optimal requirement should also be related to the level of production. A supply of 200 IU vitamin E/kg milk (3.5% fat) would ensure 40–50 µg vitamin E/g milk fat. It should be remembered that for proper action of vitamin E, other nutrients like energy, protein and selenium must be supplied in adequate amounts.

The cause of the low rate of transfer of dietary vitamin E to cow's milk is not fully understood. In one study feeding 5 g D-α-tocopheryl acetate daily for 5 d increased vitamin E in milk fat from about 16 to 24 µg. For cows fed 540 g/day safflower oil coated with formaldehyde treated casein for 2 months prior to the vitamin E response test the comparable increase was from 20 to 60 µg/g milk fat (Goering *et al*, 1976). The linoleic acid content of milk fatty acids also increased from 2.7 to 13.2% by feeding protected safflower oil. However, the effect of protected oil on vitamin E in milk may not relate to this increase in linoleic acid content of milk because even for milk of low linoleic acid, vitamin E content could reach about 50 µg/g level. The effect of protected oil most likely related to increased transport of vitamin E in plasma. Feeding of calcium soaps of palm oil increased oleic acid contents of cholesterol esters and phosphatidyl choline of high density lipoproteins (HDL) in plasma of milking cows. The amount of vitamin E associated with HDL was also increased from 4.3 to 6.3 mg/g lipid (Atwal, unpublished data). Thus the low supply of unsaturated fatty acids due to biohydrogenation in the rumen seems to reduce the vitamin E transport by plasma lipoproteins of cows. This may be the major reason for low rate of transfer of dietary vitamin E to milk. Feeding of liberal amounts of vitamin E to

milking cows will also have a beneficial effect in reducing the incidence of mastitis.

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.

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 diseased state, there is an increased production of glucocorticoids, epinephrine, eicosanoids, as well as elevated phagocytic activity (Nockels, 1991), 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, 1987).

## CONCLUSION

Establishing vitamin E requirements for animals has been exceedingly difficult because of interrelationships with other dietary constituents. Requirements are influenced by the amount and type of fat (particularly with monogastrics) and degree of fat oxidation in the diet; the presence of antioxidants; dietary selenium, iron, copper, and sulphur amino acids, as well as the physiological status of the animal. Estimated dietary vitamin E requirements for most animal species are in the range of 10–40 IU/kg of diet. A beneficial role of supplemental vitamin E above physiological levels on overall performance, enhanced immunocompetence and preservation of meat and milk products has been shown but more research is needed to establish optimum dietary concentration.

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