Natural Antioxidants in Poultry Production

Neospark

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Antioxidants are involved in the prevention of cellular damage, which is commonly responsible for aging and a variety of diseases. Antioxidants are molecules, which can safely interact with free radicals and terminate the chain reaction before vital molecules are damaged. Although there are several enzyme systems within the body that scavenge free radicals, the principle micronutrient (vitamin) antioxidants are vitamin E, beta-carotene, and vitamin C. Additionally, selenium, a trace metal that is required for proper function of one of the body's antioxidant enzyme systems, is sometimes included in this category. The body cannot manufacture these micronutrients so they must be supplied in the diet.

Vitamin E

Introduction

- Vitamin E is essential for good reproductive performance.
- The standard tests for potency of forms of vitamin E (tocopherols) measure the number of rats giving birth to at least one live offspring, or the amount of vitamin E required to prevent resorption during gestation.
- The term "tocopherol" derives from the Greek words "tokos" (childbirth) and "pherein" (to bear).
- The alpha tocopherol form possesses the most activity. Compared to d-alpha tocopherol, the so-called "mixed tocopherols" possess substantially less vitamin activity:
- d-beta tocopherol; 15% to 40%,
- d-gamma; 1% to 20%, and
- d-delta; 1%.
- The World Health Organization and others have established that 1 mg. of dl-alpha tocopheryl acetate equals 1 IU vitamin E. The relative activities of the various forms are:

Milligram	Equals
d-alpha tocopherol	1.49 IU
d-alpha tocopheryl acetate	1.36 IU
d-alpha tocopheryl acid succinate	1.21 IU
dl-alpha tocopherol	1.10 IU
dl-alpha tocopheryl acetate	1.00 IU
dl-alpha tocopheryl succinate	0.89 IU

Because of its outstanding stability and resistance to oxidation outside the body, dl-alpha tocopheryl acetate is the preferred form for animal supplementation and is widely used in capsule supplements for people.

Free radicals and water-soluble and fat-soluble antioxidants

- Normal compounds produce their chemical bonds by sharing pairs of electrons, one from each atom.
- Free radicals pick up another electron from normal compounds (typically, oxygen), thus producing a stable product by pairing their unpaired electron. But in doing so they alter the "donor" compound and create another radical, since the process is still short one electron.
- In this way, free radicals alter tissue proteins and lipids (fats), causing cell damage and death. Free radical induced reactions tend to continue to produce unstable forms that go on to do further damage and release more radicals.
- The antioxidants convert free radicals to relatively stable compounds and stop or prevent the chain reaction of free radical damage. The most damaging free radical molecules are the superoxide anion (0_2^0), the hydroxyl radical (HO^0) and the peroxide radical (OH-).

- Hydrogen peroxide (H_2O_2) while not a true radical, is unstable and is likely to be converted to the hydroxy radical, which is the most potent oxidizing agent known. So it, too, must be converted by antioxidants to water.
- Superoxide, hydroxyl, peroxide radicals and hydrogen peroxide are detoxified by the enzymes superoxide dismutase, catalase and glutathione peroxidase in the water-based areas of the cell.
- Vitamin E is fat-soluble and is found mainly in the cell membranes and fatty structures of most cells. In adipose (body fat) tissue, vitamin E is found in large quantities.
- Vitamin E detoxifies peroxides, thus preventing generation of the even more toxic hydroxyl and superoxide radicals and singlet oxygen (O-).

The Relationship Of Vitamin E And Selenium (Or) Sparing Effect Of Vitamin E to Selenium

- Production of glutathione peroxidase for free radical scavenging depends upon the availability of selenium.
- The uptake of selenium here requires cysteine, either delivered directly in the diet or produced from methionine.
- The resulting form, **selenocysteine,** is the active antioxidant portion of glutathione peroxidase. Because glutathione peroxidase in the water-based areas of the cell can stop free radical reactions that would otherwise go on to attack lipid-based areas (e.g., the cell membrane), it tends to "spare" vitamin E by reducing its workload.
- Likewise, since vitamin E conversion of free radicals prevents leaks or complete breakdowns of cell membranes, it spares glutathione peroxidase from having to convert free radicals that would otherwise invade the cell.
- This co-operation is the basis for the so-called sparing effects of vitamin E and selenium.

Absorption and distribution of vitamin E

- Vitamin E is absorbed in a mixture with other dietary fats (mostly triglycerides) through the wall of the duodenum (beginning of the small intestine). Because of this mechanism, absorption of vitamin E is enhanced when dietary fats are present.
- As dl-alpha tocopheryl acetate passes through the intestinal lining, it is almost completely hydrolyzed to form alpha tocopherol.

Absorption of vitamin E is limited. In rats and humans only 20% to 30% of orally administered vitamin E is absorbed. The efficiency of vitamin E absorption decreases with higher feeding levels. (Other fat-soluble vitamins (A, D and K) have absorption efficiencies of 50% to 80%) Vitamin E absorption is poor in low fat diets.

- Absorption is also adversely affected by high levels of polyunsaturates or high levels of other fat-soluble vitamins, particularly vitamin A. However, high levels of vitamin E do not appear to affect vitamin A absorption, although they may create a somewhat higher need for vitamin A.

As much as 90% of the vitamin E in the body is stored in adipose (body fat) tissue, with the balance occurring primarily in the liver and skeletal muscles. The rate at which vitamin E is depleted varies tremendously with the type of tissue. In studies with rats, the turnover of alpha-tocopherol was 7 to 10 days in the lungs and liver, but 76 days in the spinal cord. In guinea pigs, adipose levels of vitamin E declined only slightly over a 4 month period of dietary vitamin E deprivation. However, the animals showed clear signs of vitamin E deficiency, indicating that vitamin E stored in fat cells is essentially not available to the rest of the body, which depends on vitamin E supplied in the diet.

Dietary levels in feedstuffs

- The richest food sources of vitamin E are vegetable oils, whole cereals, eggs, liver, legumes and most green plants.
- Green forages, especially alfalfa, are very good sources. The leaves of grasses contain 20 to 50 times the vitamin E found in the stems. Thus, vitamin E activity can decrease 70% to 90% from early growth to maturity in grasses, while in legumes the vitamin E activity decreases 34% to 65% from the early leafy to the post-flowering stages.
- Stability of all natural tocopherols is poor, and substantial losses occur when feeds are dried, processed and stored.
- Vitamin E is highly sensitive to heat, oxygen, moisture, oxidizing fats (especially polyunsaturates) and trace minerals.
- Oxidation of vitamin E is increased by grinding, pelleting, extruding or mixing minerals or fats in feeds. Losses from cutting, drying and baling hay range from 30% to 80%. From 54% to 73% of vitamin E activity is lost in alfalfa hay stored at 33E C for 12 weeks.
- The heaviest losses of vitamin E occur in high-moisture feeds.

Vitamin and Selenium- Source

Feed	**Se Content (mg/kg)**	**Feed**	**Se Content (mg/kg)**
Alfalfa	0.32 - 0.37	Barley grain	0.11 - 0.22
Brewer's Grain	0.70	Corn (distiller's grain)	0.48
Corn (gluten meal)	1.11	Corn (grain)	0.08
Cottonseed	10.0	Fish meal	1.4 - 2.4
Flax seed	0.9	Oats (grain)	0.26
Oats (hay)	0.17	Oats (silage)	0.01
Rape seed	1.05	Sorghum silage	0.21
Soybean seed	0.11	Sunflower meal	2.13
Wheat (soft, winter grain)	0.05	Wheat (hard, winter grain)	0.45
Whey (dehydr.)	0.06	Yeast	0.98 - 1.08

Selenium

General

- Selenium is a relatively scarce and widely distributed element
- The mean content in the crust of the earth is 0.09 ppm, making it the 66th in order of abundance
- Se content in most soils lies between 0.1 and 2 ppm

Functions

- Integral compound of glutathione peroxidase, an enzyme which catalyses the removal of hydrogen peroxide
- Glutathione peroxidase contains four selenium atoms and forms a second line defense after vitamin E
- Selenium has sparing effect of vitamin E by ensuring normal absorption of the vitamin. (This is due to the protection of integrity of pancreas and satisfactory fat digestion and absorption)

Mechanism of action of the glutathione peroxidase in the prevention of oxidation of cell membranes lipids

Step 1:

Microsomal NADPH-cytochrome P 450 reductase activity initiates lipid peroxidation in biological membranes. Because the enzyme generates super oxide anion radicals (O_2^-) when catalyzing the following reaction.

$$NADPH \longrightarrow NADP + O_2^-$$

This super oxide molecule was not a radical species responsible for the lipid peroxidation in the microsomes.

Step 2:

But the subsequent reaction of super oxide molecule bring down the deleterious effects

$$2\ O_2^- + 2\ H^+ \xrightarrow{\text{Super oxide dimutase}} H_2O_2 + O_2^-$$

This reaction is catalyzed by super oxide dimutase, which is mainly present in the all the respiring cells.

Step 3:

$$H_2O_2 + O_2^- \longrightarrow {}^*OH + O_2 + OH^-$$

According to this reaction the hydrogen peroxide generated by the super oxide mutase is not destroyed, it may react with super oxide ion in the presence of ferric ion to produce a hydroxyl free radical (*** OH)**

Glutathione peroxidase will markedly destroy the hydrogen peroxide in to water and organic hydroxy compounds.

Selenium spares vitamin E

By three ways:

1. By preserving the integrity of pancreas, which in turn allows normal fat digestion and vitamin E absorption
2. Selenium is an integral part of glutathione peroxidase, which takes care membrane lipid peroxidation there by reducing the requirement of vitaminE
3. Selenium aids in some unknown way in the retention of vitamin E in blood plasma.

Vitamin E reduces selenium requirement in two ways:

1. By maintaining body selenium in an active form or by preventing its loss from the body
2. By preventing a chain reactive autoxidation of the membrane lipids within the membrane itself, there by inhibit the production of hydrogen peroxide.

Requirement

- The requirement of selenium is depends up on the vitamin E content of feedstuffs.

	LAYERS			BROILERS		BREEDERS
	Chick	Grower	Layer	Broiler starter	Broiler finisher	Breeder
Selenium (mg/kg)	0.15	0.10	0.10	0.15	0.15	0.15

Ref: NRC 1994

Selenium has also been recently found in another enzyme, 5'-deiodinase. 5'-deiodinase is an enzyme that catalyzes the reaction of the inactive form of thyroxine to the active form. Thyroxine is a very important hormone from the thyroid that helps in regulating body temperature, metabolism, reproduction, circulation, and muscle function. It is known that Se protects the body from heavy metals such as cadmium, mercury, and silver by forming unreactive complexes with them.

Vitamin E and Selenium – Immunity

Vitamin E enhances specific humoral and cell-mediated immune responses as well as native resistance to disease, particularly phagocytosis. Supplemental levels of vitamin E have an immuno-stimulatory effect, increase delayed hypersensitivity and affect mitogenic responsiveness. Dietary supplementation of Se at levels above those recommended as nutritional requirements (0.1 ppm) enhances the primary immune responses. Vitamin E and Se appear to participate in similar nutritional and bio-chemical relationships. Supplementation of vitamin E in the diet of chicks enhances humoral immunity, which may be due to destruction of peroxides by vitamin E. Vitamin E and Se play a role in protecting against oxidative damage. Free radicals are scavenged by vitamin E as a first line of defense and then glutathione peroxidase of which Se is a part destroys any peroxides formed before they can damage the cell. Nutritional deficiencies of vitamin E or Se or both impair immune function as measured by humoral response to sheep red blood cells in young chicks.

Accordingly studies have been conducted with various levels of Se, vitamin E and their combinations to examine the effect on performance and immune response of broilers. Effect of supplementation of vitamin E, selenium and their combinations suggested that maximum body weight gain and best efficiency of feed utilization were observed in broilers fed diets containing 0.50 mg/kg Se and 300 IU/kg vitamin E.

Significantly higher antibody titres (HI and ELISA) at 10 d PI were attributed to 0.06 mg/kg and 150 IU/kg Se and vitamin E, respectively. Hence, optimum growth and immune response may be achieved at supplemental level of Se of 0.06 mg/kg and vitamin E at 150 IU/kg. The vitamin E level is higher than that of recommended by NRC (1984, 1994). Dietary supplementation with selenium or vitamin E reduced mortality and increased body weight gain of nonimmunized chickens infected with E. These studies suggest that immunization of chickens against coccidiosis is enhanced by Se or vitamin E supplementation.

Vitamin and Selenium- Deficiency

The nutrient requirement for poultry varies with the status of the animal. For young chicks, < 6 weeks, the requirement is 0.15 mg/kg; for the others, the requirement is 0.10 mg/kg. Several conditions affect poultry due to Se deficiency. One is exudative diathesis, which is the accumulation of fluid throughout the body, particularly in the abdomen and feet. This is caused by increased permeability of the capillaries and leakage of fluid from the capillaries.

Chicks with this condition are also anemic and are protein deficient. It occurs about 2-4 weeks after hatching and is easily diagnosed due to the edema and the blue-green tint to the skin after progressing to the hemorrhagic stage. Poultry are also affected by nutritional muscular dystrophy. They also suffer from pancreatic atrophy, which has been found to be caused solely by Se deficiency. Atrophy of the pancreas results in a reduction in the amounts of lipase, trypsinogen, and chymotrypsin--all enzymes that aid in digestion of food. Therefore, this leads to extremely reduced growth and feathering. Egg production decreases when hens are Se deficient; there is no evidence that suggests an effect on male reproduction.

A spectrum of diseases of chickens and turkeys, occasionally ducklings and other birds, seen worldwide, characterised by oxidation of various tissues and caused by Vitamin E deficiency. The problem is associated with feed rancidity typically in diets with high fat. Encephalomalacia and exudative diathesis tends to be seen in young birds of 1–5 weeks of age. Muscular dystrophy is seen more frequently in older and mature birds.

Signs of Vitamin E deficiencies are Imbalance, Staggering, Uncontrolled movements, Falling over, Paralysis, Ventral oedema, Green wings. Post mortem lesions are Swollen cerebellum with areas of congestion, Haemorrhage, Necrosis, Blood-stained or greenish subcutaneous oedema, Steatitis, White streaks in muscle.

For Further information please refer to our Product details of:

- **PeriVac Plus**
- **PeriVac Plus Forte**
- **PeriVac Plus Liquid**