

Vitamins and Supplementation Requirements

By Debra McDonald

Vitamins are classified into two groups: fat-soluble (vitamins A, D, E and K) and water-soluble (B complex, vitamin C). Fat-soluble vitamins require dietary lipids to be consumed simultaneously for optimal uptake and can be stored in tissue for extensive periods. As fat-soluble vitamins compete for mechanisms of uptake, an excess of one vitamin may lead to a deficiency of another even though dietary intake appears adequate. Carotenoids (provitamin A) are also absorbed in a similar fashion to fat-soluble vitamins so dietary excesses of fat-soluble vitamins can diminish uptake of carotenoids. In contrast, water-soluble vitamins are not stored for any period so regular intake is required.

Water-soluble Vitamins

As there have been no extensive studies undertaken on vitamin B requirements of pet and aviary birds, refer to publications for poultry.

Vitamin C

Vitamin C (ascorbic acid) is commonly known as the vitamin that prevents scurvy. However, it is also involved in collagen synthesis, steroid, fatty acid and drug metabolism, carnitine biosynthesis, tyrosine metabolism, catecholamine synthesis, prevention of peroxidation and histamine metabolism. Ascorbic acid plays an important role in many of the processes associated with the 'flight or fight' response, so stresses associated with high temperatures, growth and reproduction may increase requirements of birds in captivity as it may not be synthesized in sufficient quantities. The stress of high temperatures affects endocrine systems responsible for retention and proper metabolic functioning of vitamin C.

Sources of Vitamin C

Most birds are capable of synthesizing vitamin C if provided with adequate dietary precursors. Enzymatic activity occurs in the kidneys of birds in older orders and becomes localised in the liver of more advanced Passeriformes. However, some passerines such as the vented bulbul are unable to synthesize vitamin C due to a lack of *L-gulonolactone oxidase* and require a dietary source. Vitamin C is concentrated in fresh fruits and green leafy vegetables and animal organs such as liver and kidney, with only small amounts in meat.

Vitamin C Deficiency

Reproduction and growth increases the demand for protein including collagen (the synthesis of which involves vitamin C). Supplementation of young chicks and hens with 100-200 mg kg⁻¹ vitamin C, when exposed to high environmental temperatures improves

growth, egg production and eggshell strength. Dietary requirements may also vary with age as willow ptarmigan adults are able to synthesize sufficient vitamin C, whereas chicks require supplementation with 750 mg kg⁻¹. Berries that form part of the diet of the willow ptarmigan during the breeding season can contain up to 5000 mg kg⁻¹ vitamin C.

Vitamin C is susceptible to destruction with handling and processing. While it is stable when exposed to boiling water for short periods, a greater proportion is destroyed when heated at low temperatures for long periods. Any form of processing that ruptures tissue (such as freezing and thawing) exposes vitamin C to air losses due to oxidation but it is generally stable during normal pelleting processes.

Vitamin C Toxicosis

It is possible that birds have an intestinal block, similar to humans, that prevents excess absorption of vitamin C. If this is the case then metabolites of L-ascorbic acid such as oxalic acid may be problematic for birds susceptible to calcium deficiencies. Excesses of vitamin C can also bind copper, resulting in growth deficiencies, increase in the incidence of aortic rupture, and decrease in the elastin content of the aorta if diets are also deficient in copper. Levels of vitamin C vary significantly between foods and it is important to evaluate vitamin C content for species that are susceptible to iron storage disease, as vitamin C improves the absorption of iron by facilitating the reduction of the ferric form to the more absorbable ferrous state.

Fat-soluble Vitamins

Fat-soluble vitamins (A, D, E and K) are the vitamins that can accumulate in the body to excessively high levels and it is easy to overdose a bird on these vitamins. They compete for mechanisms of uptake so an excess of one can lead to a deficiency of another, despite adequate dietary intake of the latter.

Vitamin A

There is much confusion about vitamin A as it is generally perceived to be lacking in most foods but it is imperative that we provide the correct concentration of dietary vitamin A for birds in captivity.

In reality, plants do not produce vitamin A and vitamin A can only be obtained from animal foods or the conversion of provitamin A carotenoids such as β -carotene. Birds that feed only on plants must convert provitamin A carotenoids to vitamin A. This is done on an 'as needs' basis. Birds such as lorikeets that feed on brightly coloured fruits are not faced with shortages of provitamin A carotenoids in the wild and have not evolved with a need to store vitamin A in lean times. They are particularly susceptible to excesses of dietary vitamin A. Unfortunately, symptoms of vitamin A toxicosis often resemble those of deficiencies and distinguishing between the two requires careful evaluation of dietary intake, supplementation rates and other influencing factors.

Vitamin A is a key vitamin involved in:

- **vision:** Deficiencies of vitamin A can impair vision and lead to blindness. Vitamin A forms the prosthetic group of rhodopsin for dim light vision (rods) and the prosthetic group in iodopsin for bright light and colour vision (cones). At a late stage of vitamin A deficiency, it is possible to regenerate rods but cones eventually disintegrate and result in blindness.
- **reproduction:** defects in reproduction, including increased time between clutches, reduced hatchability, increased embryonic mortality and decreased survival time of progeny, decreased testis size and failure of spermatogenesis, and a decline in sexual activity in males are all correlated with deficiencies of vitamin A. These may be associated with failure to maintain healthy epithelium. Excesses of vitamin A may also interfere with uptake of vitamin E, compromising fertility, hatchability and survivorship of chicks.
- **immunity:** both deficiencies and excesses of dietary vitamin A suppress immune function. Vitamin A deficiency in chicks leads to a rapid loss of lymphocytes. Excess vitamin A in the hen can compromise antioxidant status of progeny as concentrations of vitamin E in egg yolks are depleted and decreases in vitamins C and E and carotenoids in embryonic liver increase susceptibility to lipid peroxidation.
- **membrane integrity:** Vitamin A is required for the maintenance of epithelial cells, which form protective linings on many of the body's organs. It maintains epithelial cells such as those lining the lungs, kidneys, gastrointestinal and reproductive tracts. Vitamin A penetrates lipoprotein membranes and, at optimum levels, may act as a cross-linkage agent between the lipid and protein, thus stabilizing the membrane. Weakening of the membrane results from excess vitamin A penetrating the inelastic protein portion of the membrane, which resists expansion. This can increase access to pathogens and infection. If dietary vitamin A is deficient, epithelial cells that make up the membranes will change their characteristic structure, altering membrane permeability of cells and intracellular particles. Hyperkeratosis results from both deficiencies and excess of vitamin A. This can result in a loss of function of the tissues involved including those of the alimentary, genital, reproductive, respiratory and urinary tracts. Coccidiosis can lead to the destruction of vitamin A in the gut and injures microvilli of intestinal wall, decreasing the absorbance of vitamin A. Vitamin A deficiency in chicks is characterised by poor feathering on head and neck and breast regions as well as facial dermatitis.
- **growth:** defects in bone growth result from changes in control of activity of osteoclasts of the epithelial cartilage. A deficiency in vitamin A results in reduced activity of osteoclast (reabsorbing bone), leading to excessive deposition of periosteal bone by the unchecked function of osteoblasts (depositing bone).

- **embryogenesis:** β -carotene cleavage enzymes are not detected in the duodenum of chicks and they may be reliant on maternal transfer of vitamin A to the embryo. Therefore, conversion efficiency of β -carotene to vitamin A needs to be evaluated in chicks before being incorporated into hand rearing mixes.
- **vocalisation patterns:** alterations in vocalization patterns are detected in cockatiels maintained on both zero and excess dietary vitamin A. These include exaggerated vocalisations in response to handling with greater intensity, number, duration and amplitude with stress calls. This may influence parental response to begging behaviour of chicks.
- **iron storage disease:** excess vitamin A enhances uptake of dietary iron and may contribute to iron storage disease in some species.
- **pancreatitis:** the pancreas is responsible for production of digestive enzymes to break down protein, fat and carbohydrates in the small intestine and releases glucagons and insulin, which regulate blood sugar levels. Pancreatitis is correlated with high dietary vitamin A and can lead to diabetes and digestive difficulties. Hypervitaminosis A also increases activity of the enzyme *sucrase* and eliminates the ability to regulate the activity of this enzyme by the duodenum in the small intestine.

Dietary Requirements for Vitamin A

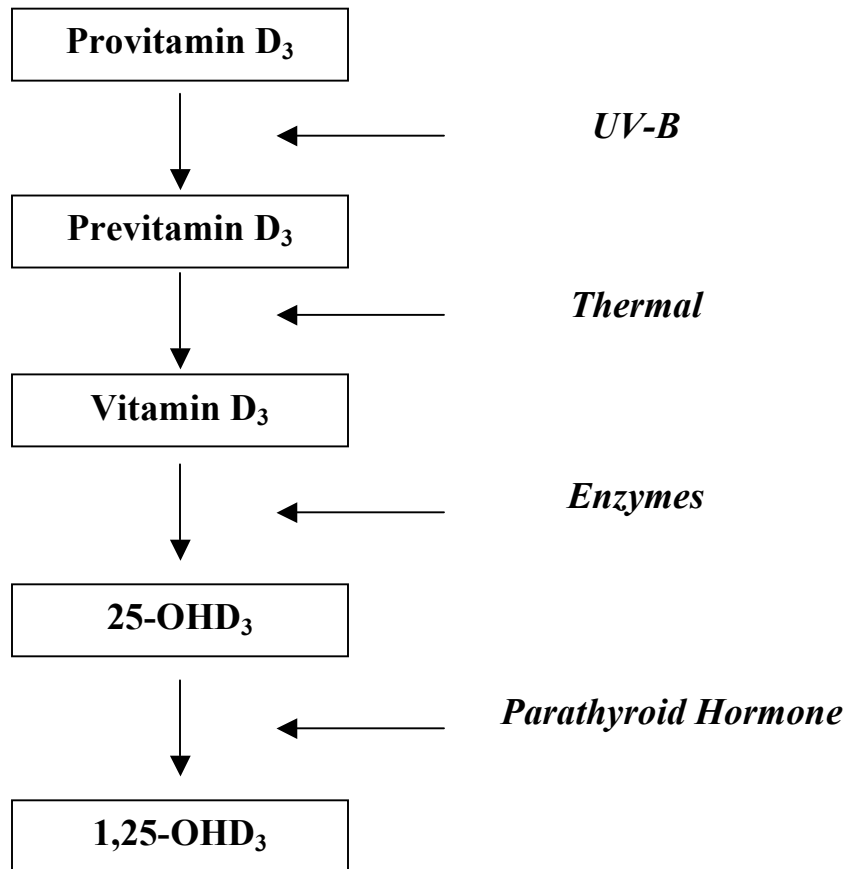
Vitamin A requirements vary taxonomically with the limited studies conducted on psittacines indicating that their dietary requirements do not exceed that of poultry. Despite this, many commercial products provide excessively high levels of vitamin A and concentrations exceeding 10,000 IU kg⁻¹ are toxic for cockatiels. Concentrations of 2,000-4,000 IU kg⁻¹ are generally recommended. Lorikeets are particularly susceptible to excesses of vitamin A, with vitamin A from egg powder alone proving to be toxic for these birds.

Vitamin D

Vitamin D is a group of closely related compounds that possess antirachitic activity. They are obtained directly from the diet or from irradiation of the body. The two major natural sources (provitamins) are cholecalciferol (D₃ in animals) and ergocalciferol (D₂, predominantly in plants). Cholecalciferol can be produced in the skin of most mammals from provitamin 7-dehydrocholesterol via activation with ultraviolet-B light in as little as 11-15 minutes daily. Both D₂ and D₃ forms can also be ingested and further metabolized to 25-hydroxyvitamin D through hydroxylation first by the liver, and then again to 1,25-dihydroxyvitamin D in the kidneys.

Function

The primary function of vitamin D is to enhance intestinal absorption and mobilisation, as well as retention and bone deposition of calcium and phosphorus. This function is manifested through its activity as a hormone (i.e., 1,25-dihydroxyvitamin D₃). The dietary requirement for vitamin D for poultry is 200 IU kg⁻¹ with the safe upper limit in chicks less than 60 days old 40,000 IU kg⁻¹ and as little as 2,800 IU kg⁻¹ if older than 60 days. Dietary recommendations for poultry have proven sufficient in scientific studies of African greys. Dogs and cats do not produce sufficient vitamin D when exposed to sunlight, requiring a dietary source of this nutrient and it is possible that carnivorous birds also have higher dietary requirements for vitamin D.



**Conversion of vitamin D₃ to active form.
Disruption at any stage can diminish uptake of calcium.**

Vitamin D Deficiency

Vitamin D deficiency may result from a dietary deficiency, compromised health status of key organs, dietary excesses of other fat-soluble vitamins or inadequate exposure to UVB

radiation. While birds only require limited exposure to sunlight each day to maintain adequate levels of vitamin D, many companion birds are maintained indoors and sunlight through ordinary window glass is inefficient for the production of vitamin D in the skin. Sunlight also provides most of its antirachitic powers during the four hours around noon so time of exposure to sunlight may influence its effectiveness. It is important to provide artificial lighting that provides UVB (290-320nm) as this is the wavelength at which vitamin D is converted.

Vitamin D synthesis can be affected by liver malfunction as this limits the production of the active forms, intestinal disorders can reduce absorbance of the vitamin, and kidney failure can prevent synthesis of 1,25-(OH)₂D. The first signs of vitamin D deficiency include decreased egg production, thinning of eggshells or no shells produced, a decrease in eggshell strength as hens age, decreased hatchability and an increased incidence of embryonic death, especially at around 18-19 days. D₃ and 25-OHD₃ are readily passed to the egg so problems may arise if hen's diet is deficient. Inadequate maternal transfer results in the failure of development of the upper mandible of chicks as well as ability of chicks to crack shells, leading to mortality.

Vitamin D Toxicosis

Macaws and budgerigars are particularly susceptible to excesses of dietary vitamin D. Widespread calcification of soft tissue can lead to inflammation, cellular degeneration, kidney insufficiency, bone thinning from demineralisation, leg problems due to calcium loss and general depression in performance in hens. Toxic levels can be transferred maternally to the embryo, leading to abnormalities in chick development.

Vitamin E

Vitamin E consists of a number of chemicals that are referred to as tocopherols (synthesized mainly by green parts of plants) and tocotrienols (synthesized mainly in the bran and germ fractions). As some forms of vitamin E have little biological activity, a total vitamin E analysis is not a reliable means of determining actual vitamin E activity. Synthetic vitamin E occurs as a number of isomers of varying activity with the natural source being more potent (and far more expensive).

Vitamin E Requirements

Requirements for dietary vitamin E are largely dependant on the nature of the diet as requirements are greater with increasing levels of polyunsaturated fatty acids (PUFA), especially those of the *n*-3 family, oxidizing agents, vitamin A, carotenoids and trace minerals. It is important to combat free-radical injury, enhancing the immune response and playing a role in the prevention of cancer, heart disease, cataracts, and a number of other diseases.

Antioxidant Function of Vitamin E

Vitamin E works in conjunction with the enzyme *glutathione peroxidase* to protect cells against the adverse effects of reactive oxygen and other free radicals that initiate the oxidation of polyunsaturated membrane phospholipids. This function cannot be replaced by synthetic antioxidants. Selenium, as part of the enzyme *glutathione peroxidase*, is a second line of defence that destroys peroxides before they damage membranes. Therefore, selenium, vitamin E and sulfur-containing amino acids, through different biochemical mechanisms, are capable of preventing some of the same nutritional diseases. Vitamin E prevents fatty acid hydroperoxide formation, sulfur-containing amino acids are precursors of *glutathione peroxidase* and selenium is a component of *glutathione peroxidase*. Oxygen-free radicals form naturally during metabolism and are promoted by external factors such as X-rays, ultraviolet radiation and pollution. Antioxidants such as vitamin E help to counter the detrimental effects of free radicals and a deficiency can be implicated in the development of several diseases including cancer, inflammatory conditions and heart disease, highlighting the need to consider antioxidant levels as part of preventative medicine.

Substituting Vitamin E with Selenium

Vitamin E and selenium act synergistically in the *glutathione peroxidase* system with higher levels of vitamin E reducing the requirement for selenium. While it has been suggested that vitamin E can be substituted with the far cheaper selenium, the two nutrients are not interchangeable, as they don't have the same actions. Selenium toxicosis can decrease hatchability, growth and reproductive success, also resulting in teratogenesis, deformed embryos, diminished immune function, abnormal feather loss, emaciation and liver lesions.

Dietary Requirements of Vitamin E

Vitamin E is one of the least toxic vitamins but high doses may antagonize other fat-soluble vitamins, resulting in reduced hepatic and egg yolk storage of vitamin A, impaired bone mineralization and coagulopathies as a result of decreasing absorption of vitamins A, D and K respectively. While dietary requirements for captive animals have been estimated at 200mg kg⁻¹ DM, many foods are deficient in this vitamin, especially formulated foods. The high PUFA contents of many nuts and marine fish can also increase the requirement for dietary vitamin E as well as heat stress.

Deficiencies of Vitamin E

The clinical manifestations of vitamin E deficiency vary markedly between species. In general, the neuromuscular, vascular and reproductive systems are affected most commonly. Signs of vitamin E deficiency are mostly attributed to membrane dysfunction because of the oxidative degradation of PUFA membrane phospholipids and disruption of other critical cellular processes.

Vitamin K

Vitamin K is a fat-soluble vitamin that receives little attention as it is readily available from most produce items and vitamin K deficiencies are not normally diagnosed. However, there are some bird species that demonstrate symptoms of vitamin K deficiency, particularly those that nest in termitaria (termite mounds).

Vitamin K plays a major role in blood clotting and is involved in the synthesis of osteocalcin, a protein that regulates the incorporation of calcium phosphates in growing bone. Since birds have such a short intestinal tract and so few microorganisms, it is assumed that they rely very little on vitamin K produced by bacteria and require a dietary source. It is possible that this supply needs to be increased during breeding to ensure sufficient supply to hatchlings. Dietary vitamin K is also particularly important for some species of fig parrots that show high mortality associated with cerebral hemorrhage, a condition that is prevented with vitamin K supplementation.

Forms of Vitamin K

Vitamin K is available from three different sources, all differing only in the side chain:

- a) Vitamin K₁: phylloquinone produced from plants
- b) Vitamin K₂: menaquinone produced by bacteria (has an additional double bond in the repeater side chain)
- c) Vitamin K₃: menadione produced synthetically with no side chain.
- d) Dietary Sources of Vitamin K

Vitamin K₁ is present as the fat-soluble portion of plant chlorophyll and must be converted to vitamin K₂ prior to absorption. Birds that nest in termitaria may have developed a dependence on the passively absorbed vitamin K₂, as they ingest termites during excavation of live termite mounds. Termites contain large populations of gut microbes to break down β -glycosidic bonds of plant fibres and may produce concentrated quantities of vitamin K₂. While vitamin K₁ is actively absorbed, vitamin K₂ is passively absorbed and birds may not be able to utilise plant-based vitamin K. The synthetic form is both passively and actively absorbed. Breeding hens must be provided with adequate dietary vitamin K to ensure adequate transfer to embryonic liver.