

Nutrition and Iron Storage Disease

By Debra McDonald

Iron storage disease is prevalent in many frugivorous and insectivorous birds maintained on commercially formulated foods as well as in some seed-eating psittacines. Various factors have been implicated in the development of this disease including genetic predisposition, immunological stress, viruses and nutrition. However, these theories do not hold true for all species, nor even all individuals of the same species. A common factor with many commercially formulated products is the high vitamin A content and it is plausible that this excess vitamin A is influencing iron uptake in birds. These high vitamin A levels are in contrast to vitamin A content of wild food resources, with negligible levels in many invertebrates and a distinct absence in plants, with vitamin A activity arising from plant carotenoids.

While iron is an essential nutrient it also acts as a catalyst for chemical reactions involving free radical formation, potentially leading to cell damage. Iron storage disease results from the accumulation of iron in various tissues with the liver being the organ most frequently affected. Animals have no means of eliminating excess iron so chronic ingestion of large amounts of absorbable dietary iron results in liver storage.

Nutritional Implications for Iron Storage Disease

Iron storage disease is generally not reported for wild birds or those maintained in captivity on fruit-based diets. There are a number of nutritional factors that may contribute to iron storage disease including:

a) Dietary Iron Content

Excess dietary iron has long been implicated as a causative factor in the development of iron storage disease, with many commercial foods containing high levels of iron. It is generally recommended that commercial diets contain less than 100 mg kg⁻¹. However, iron content of wild fruits and insects often exceeds this concentration. This brings into question whether levels of dietary iron alone are responsible for the development of iron storage disease. With the focus on absolute levels of dietary iron, some other potentially problematic aspects of nutrient composition are often ignored.

b) Ascorbic Acid (Vitamin C)

Iron is absorbed in the duodenum after ferric iron is reduced to the ferrous form and ascorbic acid (vitamin C) reduces and chelates nonhaeme (plant-based) iron, increasing absorption. Ascorbic acid also maintains a soluble complex when intestinal pH increases enhancing the solubility of ferric iron. Vitamin C content of commercially available fruits commonly fed to pet birds is significantly higher than that of wild fruits. However, not all studies support the view that excess vitamin C contributes to the development of iron storage disease in all species.

c) Phenolics and Tannins

Tannins reduces the absorbance of bioavailable iron by interfering with complex formation in gut. Many wild birds drink rainwater caught in tree cavities, which may contain tannins leached from plants and some aviculturists incorporate tannins in diets or drinking water of birds susceptible to iron storage disease. However, studies of starling do not support this practice and tannins can also bind to proteins and render them insoluble. Condensed tannins are less likely to interfere with iron absorption than galloyl tannins so it is important to know the nature of the tannin being provided.

d) Fats

Saturated fats increase iron absorption, which may be accredited to changes in fatty acid composition of the intestinal lining. Formulated diets are unlikely to be high in saturated fats unless pellet coatings such as palm oil (high in saturated fats) are used. Commercial dog foods being used as dietary substitutes for invertebrates may also be high in saturated fats.

e) Vitamin E

One factor that is often overlooked when formulating dietary rations is that of vitamin E content. Vitamin E is a potent antioxidant and any compound that induces oxidative stress is considered to have a potential negative effect on vitamin E concentration in both blood and tissues. While a minimum of 10 mg kg⁻¹ (DM) is generally recommended for poultry, more recent studies suggest up to 100 mg kg⁻¹ (DM) may be required and it is possible that dietary requirements of birds susceptible to iron storage disease are as high as 200-300 IU kg⁻¹ (DM). Many commercial products fall well below these recommendations. Manufacturers' data commonly reflect calculated amounts provided through vitamin premixes and does not take into account variations due to poor packaging or lipid peroxidation from other ingredients. Requirements increase with the addition of polyunsaturated fats to the diet common in some nuts and sunflower seeds as well as environmental factors such as atmospheric pollutants, toxins, infectious agents and other stressors.

f) Vitamin A

Plants do not contain vitamin A, with wild birds ingesting provitamin A carotenoids and converting these to vitamin A on an 'as needs' basis. Even insects are very low in vitamin A. However, vitamin A increases iron absorption from grains such as rice, wheat and corn, common ingredients in formulated products. In contrast to vitamin A levels of wild food resources, vitamin A content of commercially formulated foods is generally high, exceeding recommendations for cockatiels of 2-4,000 IU kg⁻¹. Vitamin A content of commercial diets for parrots can be as high as 17,000 IU kg⁻¹, with values for some nectar replacement products higher than 60,000 IU kg⁻¹, flamingo diets up to 36,110 IU kg⁻¹ and some hand-rearing formulas as high as 47,000 IU kg⁻¹. Vitamin A will also minimise uptake of vitamin E, while the presence of carotenoids in cell membranes partially inhibits the loss of vitamin E and ingestion of carotenoids from wild food resources may protect membrane stores of vitamin E. Dietary tannins invert catalysis from pro-oxidation to antioxidation but excess vitamin A can inhibit this effect and also partially inhibits the effect of phytate on iron absorption.