

Fatty Liver in Birds

By Debra McDonald

Fatty liver is a common disease in psittacines associated with an accumulation of a large amount of abdominal and hepatic (liver) fat. An overweight bird with a high accumulation of fat is indicative of the disease. High cholesterol and cessation of egg production are characteristic symptoms of fatty liver. Although there are some hereditary tendencies towards the disease, nutrition plays a major role in its development and it is generally a consequence of an imbalance in energy metabolism.

Lipid infiltration into the liver results in an enlarged liver (hepatomegaly), with a weakened cellular structure that is easily damaged. The numerous blood vessels of the liver are easily ruptured during egg laying and a hen with an enlarged, friable liver that is straining to lay, is more vulnerable to injury and rupture of large blood vessels that can lead to sufficient blood loss to result in death.

Nutritional Implications for Development of Fatty Liver: Biotin

It is generally perceived that a diet low in fat will alleviate or prevent symptoms. However, long chain fatty acids, especially those of the *n*-3 family (concentrated in flaxseed/linseed), are beneficial in the diet as a preventative measure. Low dietary protein predisposes chicks to develop fatty liver but high dietary protein can lead to biotin deficiency and diets containing excess protein are not recommended. Biotin is an essential coenzyme in carbohydrate, fat and protein metabolism. Biotin deficiency is most severe in young chicks of heavier strain and greater rate of body weight gain so promoting higher growth rates in psittacines may predispose birds to fatty liver. Biotin is rapidly destroyed as feeds become rancid, with the addition of α -tocopherol decreasing inactivation of biotin. Higher biotin intake is required if birds are fed diets of sunflower seeds.

Nutritional Implications for Development of Fatty Liver: SAME

An important nutrient in fatty liver disease is a natural metabolite of the amino acid methionine known as S-adenosylmethionine (SAME). SAME maintains mitochondrial function, prevents DNA mutations and restores cellular membrane fluidity so that cell receptors become better able to bind hormones and other factors. SAME's methyl groups make possible the production of the "fat burner" *carnitine*; the neuronutrient *acetyl L-carnitine*; the primary ATP energy reservoir, *creatine phosphate*; the stress hormone and neurotransmitter, *adrenaline*; the neuronutrient and chief membrane phospholipid, *phosphatidyl choline*; and the DNA bases *methyladenine* and *methylcytosine*. While healthy livers synthesise sufficient methionine, liver disease can impair SAME synthetase. Deficiencies of any of the active coenzyme forms of vitamins B₂, B₆, B₁₂ and folic acid will disrupt SAME production. SAME production decreases with age so dietary supplementation may be required for aged birds prone to fatty liver disease. Without

SAMe, the liver protective agent glutathione cannot be synthesised. While increasing glutathione levels through supplementation is desirable, glutathione alone is not a substitute for the combined actions of SAMe and glutathione. Anhydrous betaine is a substance made from beet sugar that increases SAMe levels. Increasing levels of betaine reduces fatty infiltration and provides the precursors for the free radical quencher glutathione. Betaine is added to all **Dr Mac's Organic Origins** diets.

Nutritional Implications for Development of Fatty Liver: Silymarin

Silymarin is a collective group of polyphenolic flavanolignans extracted from the seeds of the milk thistle (*Silybum marianum*). The flavanoids are powerful antioxidants, increasing levels of glutathione and protecting the liver from oxidative damage, and may promote growth of new, healthy liver cells.

Nutritional Implications for Development of Fatty Liver: Pesticides

While pesticide levels of individual ingredients may be deemed safe, a combination of a variety of pesticides or an accumulation of pesticides in tissues can result in pesticide toxicity. PCBs (polychlorinated biphenyls) increase liver and body weights of birds associated with fatty liver syndrome and can increase total cholesterol. In addition, many pesticides have estrogenic actions and high estrogen levels are associated with fatty liver syndrome. These estrogenic pesticides mimic the action of normal endogenous hormones and influence normal ovarian function. A combination of estrogen and a positive energy balance (excess dietary energy) create sufficient fat deposition in the liver for fatty liver syndrome to occur. Force-feeding birds that do not need to be force fed can also increase liver fat and plasma estradiol, producing fatty liver syndrome. This condition has also been observed in cockatoos and cockatiels fed by inexperienced aviculturists or provided with improperly formulated diets.