Dietary modification of omega-3 fatty acids for birds with atherosclerosis

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Atherosclerosis is characterized by hardening of the arteries and plaque formation and is widely reported in humans, other mammals, and birds. A high intake of cholesterol or an imbalance in dietary fatty acids can accelerate the development of atherosclerosis. Because many avian species naturally develop or are easily induced to develop atherosclerosis, they are frequently used in the study of this disease. However, lipid metabolism of birds differs in some respects from lipid metabolism of mammals. A key difference is the existence of portomicrons, rather than chylomicrons, that form in response to consumption of a meal that contains fats. Many studies have explored the effect of providing supplemental UFAs in an attempt to reduce atherosclerosis, with omega-3 fatty acids providing the most promising results. The purpose of the information provided here is to review atherosclerosis in birds, lipid metabolism in birds, and the effect that dietary UFAs may have on atherosclerosis.

Atherosclerosis in Birds

Atherosclerosis begins with fatty streaks and progresses to loss of arterial elasticity caused by thickening of the tunica intima, which is characterized by increases in smooth muscle cells and expansion of the extracellular matrix. Atheromatous plaques are fibrous tissue between the tunica intima and internal elastic lamina, which result in gross thickened pale lesions of the affected vessels. Circumferential arterial lesions may cause stenosis of the vessel lumen. In severe cases, there may be accumulation of lipid, cholesterol, foam cells, and macrophages and other leukocytes, as well as mineralization of the artery. Thickening of the arterial wall, roughening of the tunica intima, induration, and yellowish discoloration may be seen grossly during necropsy. However, necropsy surveys may underestimate the prevalence of this disease because it cannot be diagnosed solely on the basis of gross examination. Factors that predispose birds to the development of atherosclerosis remain unclear, but hyperlipidemia, endothelial inflammation, toxic substances, immune complex disease, species of bird, inappropriate long-term diet (high fat or cholesterol intake), and lack of exercise have been implicated. Median age of affected adult birds typically ranges from < 2 to 13 years. No sex predisposition is evident. Birds appear to be more susceptible to the development of atherosclerosis than any mammals, except for humans.

Avian species affected by atherosclerosis include many orders of birds, which range from domestic species to those maintained in zoologic collections. Most atherosclerosis studies in birds have involved investigations of turkeys, Japanese quails, chickens, or pigeons. Each order of bird develops atherosclerotic plaques in a different location, which suggests that slightly different mechanisms may exist for each species. The most common location is the aorta at the base of the heart, but plaques may also develop in the brachiocephalic artery, pulmonary artery, descending aorta, heart valves, and mural coronary arteries. Except for turkeys, all birds studied do not have aneurysmal dilation, which is in contrast to humans with atherosclerosis.

Risk factors for development of atherosclerosis include increases in plasma cholesterol, VLDL, intermediate-density lipoprotein, LDL, LDL₆, and apoprotein B concentrations. Decreased concentrations of HDL, particularly HDL₆, and apoprotein A₁ are also risk factors.

ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AA</td>
<td>Arachidonic acid</td>
</tr>
<tr>
<td>ALA</td>
<td>α-Linolenic acid</td>
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<tr>
<td>DHA</td>
<td>Docosahexaenoic acid</td>
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<tr>
<td>EPA</td>
<td>Eicosapentaenoic acid</td>
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<tr>
<td>HDL</td>
<td>High-density lipoprotein</td>
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<tr>
<td>LA</td>
<td>Linoleic acid</td>
</tr>
<tr>
<td>LDL</td>
<td>Low-density lipoprotein</td>
</tr>
<tr>
<td>PUFA</td>
<td>Polysaturated fatty acid</td>
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<tr>
<td>UFA</td>
<td>Unsaturated fatty acid</td>
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<tr>
<td>VLDL</td>
<td>Very-low-density lipoprotein</td>
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for development of atherosclerosis. However, most of the data are from domestic birds, rather than from zoologic or wild species.

Total vascular collagen content is not believed to be a good indicator of atherosclerosis in birds. However, atherosclerotic lesions in birds can contain cartilaginous or osteous metaplasia. The development of atherosclerosis is often associated with persistent and exaggerated postprandial lipemia. Therefore, postprandial metabolism of lipoproteins, rather than fasting lipoprotein concentrations, may offer more clues to the likelihood of developing atherosclerotic plaques.

Mural thrombi typically are scarce in birds and could explain the lack of thrombosis and ischemia in avian species. In contrast to atherosclerosis in mammals, atherosclerosis in birds may be linked with other diseases. For example, atherosclerotic lesions in chickens with herpesvirus infection (i.e., Marek’s disease) have similar characteristics and distribution of lesions as in humans with chronic arterial disease. Dietary PUFAs, especially omega-3 fatty acids, play an important role in the prevention of atherosclerosis by inhibiting inflammatory processes resulting from fatty streaks, atheroma accumulations, or other mechanisms.

**Lipid Metabolism**

Similar to the situation in mammals, the digestive tract of birds varies by species. Although some mammals have a cecum, many birds have paired ceca with variation in functionality among species, which results in slightly different digestive strategies among avian species. In addition to harboring bacteria and absorbing water, another possible function of the ceca is absorption and digestion of cholesterol. Psittaciformes (parrots), Apodiformes (hummingbirds and swifts), Coliiformes (collies-mousebirds), Coraciiformes (kingfishers and motmots), and Piciformes (woodpeckers) do not have ceca. Consequently, the common practice of basing diets on the nutritional needs of chickens, a species with well-developed ceca, may result in inadequate nutrition for some species.

A number of differences in lipid metabolism exist between mammals and birds. Avian pancreatic bile ducts enter the distal portion of the jejunum, rather than the proximal portion as in humans, dogs, and rats. This anatomic difference may affect the timing of lipid absorption during digestion. Additionally, the composition of avian bile acid differs from that of mammals. Although mammalian bile acid is composed of phospholipids and cholesterol, avian bile acid is mainly cholesteryl esters and triglycerides. In contrast to the situation in mammals, lipoproteins secreted by the small intestines of birds enter the portal blood and are thus called portomicrons. In mammals, lipoproteins enter the lymphatic system rather than the portal system and are commonly called chylomicrons. Portomicron remnants are cleared by the liver, as are chylomicron remnants after triglyceride hydrolysis. In birds, stored triglycerides are composed predominantly of 16- and 18-carbon UFAs, which make them more unsaturated than are triglycerides in mammals.

The liver is the primary site of fatty acid synthesis in chickens. In contrast, pigs mainly synthesize fatty acids in adipose tissue. Similar to birds, humans primarily synthesize fatty acids in the liver; although there is a low amount of fatty acid synthesis in adipose tissue; thus, birds can be used to study fatty acid synthesis in humans. When birds migrate and fly continuously for long distances, concentrations of free fatty acids in the blood increase to provide energy. These energy sources differ from those found in mammals. The predominant ketone in birds is β-hydroxybutyrate, whereas the predominate ketone in mammals is acetoacetate. The pentose shunt, a primary means for synthesis of NADPH in mammals, is replaced by malic enzyme and glucose-6-phosphate dehydrogenase in birds. Additionally, captive migratory species undergo the same changes in seasonal fat deposition that are seen in their wild counterparts.

Approximately 30% of the variation in plasma cholesterol concentrations in chickens is the result of heredity. Cholesterol-induced atherosclerosis is commonly used to study atherosclerosis in birds that do not normally develop atherosclerosis or to accelerate the disease process in susceptible birds. Avian species typically respond to exogenous cholesterol with an increase in blood cholesterol concentrations, whereas some vertebrates (including rats) have the ability to maintain blood cholesterol concentrations by reducing cholesterol synthesis. Birds have a predominant amount of cholesterol as HDL, as opposed to humans and some other mammals in which it is transported via LDL. In Japanese quails led a diet supplemented with 1% cholesterol (by weight) or a control diet with no added cholesterol, the males had significantly higher

**Table 1**—Mean ± SD values for blood lipid variables of birds.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Species</th>
<th>Value</th>
<th>Reference</th>
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<tbody>
<tr>
<td>VLDL (mg/dL)</td>
<td>Rhine goose</td>
<td>182 ± 86</td>
<td>8</td>
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<tr>
<td>Intermediate-density</td>
<td>Lands goose</td>
<td>76 ± 47</td>
<td>8</td>
</tr>
<tr>
<td>lipoprotein (mg/dL)</td>
<td>Rhine goose</td>
<td>25 ± 6</td>
<td>8</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>Lands goose</td>
<td>34 ± 8</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Rhine goose</td>
<td>109 ± 34</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Lands goose</td>
<td>106 ± 37</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Chicken and pigeon</td>
<td>128.8 ± 5.1</td>
<td>9</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>Rhine goose</td>
<td>530 ± 34</td>
<td>8</td>
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<tr>
<td></td>
<td>Lands goose</td>
<td>489 ± 58</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Chicken and pigeon</td>
<td>291.8 ± 4.8</td>
<td>9</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>Rhine goose</td>
<td>145 ± 9</td>
<td>8</td>
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<tr>
<td></td>
<td>Lands goose</td>
<td>155 ± 9</td>
<td>8</td>
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<tr>
<td></td>
<td>Parrot</td>
<td>119.9–340.3*</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>114.1 ± 11.4</td>
<td>10</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>Turkey</td>
<td>11.1 ± 1.0</td>
<td>10</td>
</tr>
<tr>
<td>Apolipoprotein A1 (mg/dL)</td>
<td>Turkey</td>
<td>227.1 ± 18.0</td>
<td>10</td>
</tr>
<tr>
<td>Apolipoprotein B (mg/dL)</td>
<td>Turkey</td>
<td>13.3 ± 0.2</td>
<td>10</td>
</tr>
<tr>
<td>LA (%)†</td>
<td>Chicken and pigeon</td>
<td>2.80 ± 2.1</td>
<td>9</td>
</tr>
<tr>
<td>ALA (%)†</td>
<td>Chicken and pigeon</td>
<td>2.85 ± 0.7</td>
<td>9</td>
</tr>
<tr>
<td>LA:ALA ratio</td>
<td>Chicken and pigeon</td>
<td>9.82†</td>
<td>9</td>
</tr>
</tbody>
</table>

*Value reported is the range. †Represents the percentage of total fatty acid content. ‡Value reported is the mean.
plasma cholesterol concentrations than those for the females and control groups. This could be the reason that male quails have significantly higher plaque scores than do females. However, this sex difference in atherosclerosis is not a consistent finding in all studies. Regardless of dietary treatment, female quails have significantly higher plasma triglyceride concentrations and amounts of cholesterol in the aorta, compared with results for male quails.

An important finding by the British Nutrition Foundation is that animal experiments in which researchers add 1% to 2% cholesterol (by weight) to a diet to induce atherosclerosis (as in the aforementioned study) are feeding approximately 50 to 100 times as much cholesterol as is contained in the average human diet. This approach has the potential to cause extreme effects that can alter the outcome of experiments from those seen with typical physiologic conditions and responses to cholesterol in the diet. Therefore, it is recommended that investigators use lower amounts of dietary cholesterol to best mimic the disorder.

In addition to cholesterol modification of diets, stimulating cholesterol metabolism in birds soon after hatching results in future resistance to diet-induced hypercholesterolemia. It has been estimated in humans that for each percentage of saturated fatty acid replaced by LA (which is a UFA), the total plasma cholesterol concentration decreases by 0.13 mmol/L (5 mg/dL). Food restriction affects plasma cholesterol concentrations and is believed to be protective against atherosclerosis. In 1 study, investigators provided 75-day-old male chickens food ad libitum or fed 80% of the food volume voluntarily consumed by another group of birds with a similar body weight. Birds were fed the diets for 37.5 months. Although the dietary-restricted group had a significantly lower body weight at the end of the study, they also had significantly less atherosclerosis. An interesting finding was that the group fed ad libitum had almost twice the cholesterol concentration in the abdominal aorta than was evident in the restricted group; however, no significant differences existed between plasma cholesterol concentrations of the 2 groups. The findings of that study are consistent with those in another epidemiological study in humans that detected a high positive correlation between caloric intake and death as a result of atherosclerosis.

Unsaturated Lipids and Atherosclerosis

Effects of UFAs on cells—The PUFAs improve health when substituted for saturated and monounsaturated fatty acids in the diet. The UFAs increase the fluidity of cell membranes, which results in upregulation of insulin receptors and a decrease in insulin resistance. Cell membranes become rigid when composed principally of saturated fatty acids and cholesterol. The PUFAs can act similarly to a second messenger by upregulating protein kinase C and activating macrophages and polymorphonuclear leukocytes. Cytokines and growth factors also rely on PUFAs for their function. The PUFAs have antibacterial, antiviral, and antifungal actions that may help prevent disease. Feeding chickens diets containing 8% palm oil, soybean oil, or flaxseed oil did not have a significant effect on antibody concentrations against infectious bursal disease. However, chickens fed diets rich in omega-3 or omega-6 fatty acids had a significantly greater antibody response to vaccination against infectious bursal disease. These findings may be attributed to nitroplids, which are formed by nitration of UFAs that can suppress inflammation. It is interesting that administration of aspirin results in conversion of AA, EPA, and DHA to their respective 15-epimer lipoxins, which are inhibitors of acute inflammation. Acetaminophen and EPA also act in combination to generate novel products, such as 15R-hydroxy-EPA and 18R-hydroxy-EPA, that aid in preventing microinflammation. Thus, consumption of omega-3 fatty acids in conjunction with some medications may be necessary to receive the full benefits.

Eicosapentaenoic acid can be elongated to form DHA. However, a review of studies conducted on dietary supplementation with ALA, EPA, or DHA found that only dietary supplementation with DHA resulted in increases in plasma DHA concentrations. Because DHA is more unsaturated than is ALA, it enhances cell membrane permeability and fusion more than does ALA. Fish oil can decrease the amount of oxygen-derived free radicals produced by neutrophils, thus increasing the bioavailability of nitric oxide. Additionally, acyl chains from DHA can bind to rhodopsin (a G-protein–coupled receptor) and weaken its interhelical packaging, which results in an increase in the kinetics of rhodopsin binding.

Specific dietary effects of UFAs on atherosclerosis in birds—With regard to the omega-6 fatty acids, conjugated LA is 1 form of LA that has health benefits. In 1 study, investigators used a basal diet supplemented (at 3% of the diet) with olive oil, corn oil, conjugated LA, and combinations of 2 of these oils. Analysis of the results indicated that a combination of olive oil to conjugated LA (1:1 ratio) resulted in greater growth but a similar rate of feed conversion because of increased feed intake of the chickens. There were no significant differences in carcass characteristics, except for weight of the breast muscles, between chickens fed the olive oil diet and those fed the olive oil with conjugated LA diet. Some significant carcass characteristics were detected for chickens fed the conjugated LA diet and corn oil and conjugated LA diet (1:1 ratio), compared with results for chickens fed the other diets.

In another study, investigators assessed the effects of conjugated LA on production and performance of laying hens. No significant differences in production or performance of laying hens were evident for diets supplemented with conjugated LA, fish oil, or a combination of the 2 in conjunction with yellow grease. Although the total fat comprised 3% of the diet (similar to the aforementioned study), the experimental oil comprised only 0.25% of the diet. This amount of supplementation may not have been enough to elicit differences in production and performance.

Substituting omega-3 fatty acids in place of omega-6 fatty acids results in alterations in fatty acid profiles in plasma and platelets, with a major shift from AA to EPA. Omega-3 fatty acids are strong anti-inflammatory agents because of their ability to prevent incorporation of AA into membranes and the subsequent conversion of AA to...
eicosanoids. Omega-3 fatty acids have antithrombotic properties and a favorable effect on endothelial function. They can also decrease platelet-derived growth factor, which is a necessary component for atherosclerotic plaque formation. Dietary trials in which investigators provided supplemental omega-3 fatty acids have been conducted on > 20,000 human subjects with no serious adverse effects reported.

Cardiovascular protective effects—Most cardiovascular protective effects of omega-3 fatty acids have been associated with EPA. Eicosanoids are responsible for regulating the duration and intensity of inflammatory responses. The EPA-derived eicosanoids are typically less inflammatory, whereas AA-derived eicosanoids generally are inflammatory. Free EPA results in leukotrienes of the 5 series and prostaglandins and thromboxanes of the 3 series. Metabolism of EPA typically decreases concentrations of thromboxane A₂, leukotriene B₄, and prostaglandin E₂ metabolites while increasing thromboxane A₂, prostacyclin prostaglandin I₁, and leukotriene B₄ concentrations. These combined effects result in decreased platelet adhesion and inhibition of vasoconstriction, which helps prevent atherosclerosis. In addition, EPA inhibits cell membranes from releasing AA and can antagonize AA-derived mediators. In higher doses, EPA can also decrease cytokine-induced expression of E-selectin and impair the adherent ability of ligand-bearing monocytes.

Dietary effect of ALA on birds and other species—Birds cannot form double bonds beyond the fatty acyl Δ-9 position because they lack the necessary desaturases. Thus, LA and ALA must be supplied in the diet. Birds require LA at 1% of the diet. However, excess amounts of LA may result in nutritional encephalomalacia. This condition results because LA is converted into AA and eicosanoids, such as prostaglandin E₂ and thromboxanes, which results in thrombogenesis. However, addition of ALA to the diet can block the biosynthesis of AA and the conversion of AA to eicosanoids. Thus, ALA may have a protective effect against the development of nutritional encephalomalacia. Wild parrots do not typically consume fish; thus, ALA is the major source of omega-3 fatty acids for free-living parrots.

Some functions of ALA in birds remain to be determined. For instance, preliminary analyses by our laboratory group of crop contents of wild Scarlet Macaw chicks revealed a range for crude fat content of 9.5% to 29.4% and a mean ± SD LA:ALA ratio of 8.3 ± 11.9. Content of LA ranged from 22.8% to 64.9% of total fatty acids, whereas ALA content ranged from 1.5% to 37.4% of total fatty acids. This is similar to the plasma concentrations in chickens and pigeons (Table 1). In contrast, many commercial diets for birds are based on corn and soybean components. Such diets have a preponderance of LA and contain only small amounts of ALA, which yields a relatively high LA:ALA ratio. It may be useful to evaluate diets that are similar in both LA and ALA content on the basis of their lipid metabolic effects in selected avian species to assess cardiovascular risk in these animals.

Rats fed diets with ALA as the predominant fatty acid had liver phosphoglycerides mainly composed of EPA and DHA. That study revealed the ability of rats to convert ALA into EPA and DHA. However, the efficiency of converting ALA to DHA is < 5% in humans. The 3 known metabolic pathways for ALA utilization depend on the amount of other fatty acids in the diet. α-Linolenic acid can be converted to long-chain omega-3 fatty acids or can undergo β-oxidation (with or without the resultant acetate completely oxidized). Fat acid profiles of bursal tissue in chickens reflect the fatty acid composition of the diet. This phenomenon was used to confirm conversion of ALA to long-chain omega-3 derivatives. Chickens fed a diet containing 8% flaxseed oil had EPA and DHA (which were not in the diet) in their tissues. Thus, chickens appear to be capable of converting ALA into EPA and DHA. Therefore, the ability to convert ALA into long-chain omega-3 fatty acids may support the potential benefit of all functionally active omega-3 fatty acids by supplementing the 18-carbon fatty acid.

Flaxseed is the most common form of ALA used to supplement diets because of the high concentration of this fatty acid. When flaxseed was added at 10%, 20%, or 30% to a growth diet for broilers, a significant decrease in body weight and an increase in omega-3 content in erythrocyte membranes were detected. The decreased growth of the broilers when fed the flaxseed-containing diets could have been attributable to factors that adversely affect diet quality; these effects can be prevented by heat treating the flaxseed. An addition of 10% ground flaxseed to the diet did not have a significant effect on feed intake or egg production in 18-week-old layers, compared with results for layers fed a control diet; however, feeding a diet in which 20% of the diet was ground flaxseed resulted in adverse effects.

A study in which investigators compared the fatty acid profile of breast muscle in broilers and degree of atherosclerosis revealed no association for LA, a negative but not significant correlation for ALA, and a significant negative correlation for palmitic acid. In another study, investigators conducted an experiment that evaluated the addition of flaxseed and lutein to diets on hen performance and liver hemorrhage in 18-week-old pullets. The addition of 10% flaxseed to the diet had no significant effect on feed intake, egg production, or liver hemorrhage and resulted in a decrease in liver fat. Investigators also conducted another study to evaluate the effect of supplementation with rapeseed or flaxseed on omega-3 fatty acids and the resulting ratio between omega-6 and omega-3 fatty acids in various muscles of 1-day-old chickens. However, even though many differences existed between the diets, the omega-6 and omega-3 content of each diet were not measured, which limits interpretation of the data.

A study was conducted in mice to evaluate effects for a diet high in ALA with those of a diet high in LA. Mice fed the diet high in ALA had significantly lower plasma total cholesterol and phospholipid concentrations. A meta-analysis of studies conducted for effects of supplementation with ALA on cardiovascular risk markers was performed; only 12 of 47 studies with similar outcomes were analyzed. It was found that ALA significantly lowered fibrinogen, fasting plasma glucose, and HDL concentrations. Furthermore, ALA had no sig-
nificant effect on total cholesterol, triglycerides, LDL, VLDL, and lipoprotein B concentrations; body weight or body mass index; and diastolic or systolic blood pressure. In addition, ALA also decreased concentrations of inflammatory markers, including C-reactive protein, interleukin-6, and serum amyloid A. Studies with differing ALA concentrations that did not result in changes in plasma cholesterol concentrations were mentioned in a review article.

Other dietary sources of ALA also exist and are being studied in relation to flaxseed. A study in which investigators compared diets containing flaxseed, rapeseed, chia seed, or chia meal at 15% of the diet in 1-day-old broiler chicks revealed that chia seed resulted in better performance because the flaxseed diet resulted in negative effects on growth. However, the flaxseed diet also resulted in lower amounts of abdominal fat. Thus, results of that study indicate that chia seed may be a viable alternative to flaxseed as a source of ALA.

Finally, the form of flaxseed in the diet has a large impact on the availability of fatty acids and other nutrients. Diets that differed in the form of flaxseed they contained were evaluated to determine their effects on plasma concentrations of fatty acids in humans. Only the milled and oil forms of flaxseed resulted in a significant increase in plasma ALA concentration. There was no significant difference in plasma triglyceride, cholesterol, EPA, or DHA concentrations among groups. All groups initially complained of gastrointestinal discomfort, but it only persisted in the group fed the diet containing whole flaxseed. Thus, it is important to indicate the form of flaxseed used in supplementation experiments along with the bioavailability of ALA. For example, a study in which whole flaxseed is used as the ALA source may have drastically less ALA available to the subjects than that calculated from the composition of flaxseed. This could be the reason why the group fed the diet containing whole flaxseed (which received the same amount [by weight] of flaxseed as the milled group) in the aforementioned study did not have an increase in plasma ALA concentration.

It is important to mention that some reports are inconsistent in providing critical information that could affect interpretation of the data. For example, many investigators conducting studies that involve supplementation with ALA may not have an adequate control that only ingests ALA (via dietary means) in order to determine the efficacy of supplementation. Thus, other factors that may contribute to increased ALA concentrations should be considered in the interpretation of research involving ALA supplementation. For this reason, it is important to use controlled supplementation studies in order to gather consistent data that can be applicable to the general population.

Conclusions

Atherosclerosis is fairly common in captive birds and is possibly prevalent because of a combination of dietary and housing management. Although lipid metabolism in birds typically differs from that in mammals, atheromatous plaques in birds resemble plaques in mammals. Consumption of PUFAs typically reduces the prevalence of atherosclerosis. Increasing the consumption of omega-3 fatty acids has the potential to decrease the amount of body fat, reduce the degree of insulin resistance, and boost the immune system and thus may be important in maintaining the health of captive birds. Although both omega-3 and omega-6 fatty acids are vital, the balance of omega-6 and omega-3 fatty acids in tissues continues to be an important determinant for optimal health and should be considered in studies conducted to investigate atherogenesis and lipid metabolism in avian species.

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