Nutritional Diseases

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Due to their rapid metabolism, birds have high nutritional needs and, concomitantly, manifest signs of malnutrition more quickly than many other animals. Indeed, malnutrition is a frequently diagnosed disease condition in pet birds, and many of the other diseases that are diagnosed actually may be due to a lowered resistance to pathogenic (disease-causing) organisms resulting from a poor level of nutrition.

Nutritional problems seem to be prevalent in pet birds undergoing reproductive cycles and in pet birds with special nutritional requirements. Common problems include calcium deficiency (often associated with a number of reproductive complications, such as egg binding and oviductal prolapse), vitamin D3 deficiency (and associated calcium problems), vitamin A deficiency, and goiter. A number of less well defined complaints also may have a nutritional component, including poor feathering, crooked keels and backs, rotated tibia (splay leg), fatty liver syndromes, pododermatitis (foot inflammation), and several ataxic (neurologic) syndromes.

The dietary needs of the many species of avian pet or breeding psittacines that are relatively rare in captivity may differ significantly from those of other psittacines. Research has shown, for example, that hand-fed palm cockatoos may suffer from maldigestive syndromes. Careful review of the nutritional history is indicated when considering the etiology of such complaints.

Some of the more common nutritional problems seen in pet birds are described in the following sections.

Obesity/Hepatic Lipidosis

Obesity with resultant hepatic lipidosis is one of the more common nutritional diseases seen in pet birds. It is particularly common in budgerigars due to the high-fat seed diet these birds normally consume. Not only can this type of diet lead to obesity, but also the fat can bind to calcium, leading to lowered blood calcium. Seed is also low in vitamin A, which can lead to lowered resistance to disease.

Even more detrimental, fat infiltration can severely compromise the functioning of the body's organs. Two organs that are particularly affected are the liver and the heart. Over time, as fat that has accumulated in the bloodstream infiltrates the liver, the amount of functional liver tissue decreases, and liver function is greatly compromised. This condition, called hepatic lipidosis, can also result in the liver becoming quite enlarged. Fat may also accumulate around the heart, interfering with normal function. Due to the excessive weight, the bird may be unable to fly or may tire easily.

The following may be characteristic signs of hepatic lipidosis in the budgerigar:

- *Obesity.* The bird may be overweight, with fat deposits on the chest and abdomen, so that the bird has a buxom chest or abdomen.

- *Overgrown beak.* With hepatic lipidosis, the beak sometimes grows rapidly and abnormally. Technicians should be trained to recognize signs of hepatic lipidosis, so that when an owner brings a bird in for a beak trim, he or she can recommend an examination with the practitioner if there is evidence of the disease. The beak trim should not be done on these birds, as they can easily become stressed or die during the routine procedure due to the abnormal liver condition.

- *Black spots on the beak and toenails.* Due to the compromised liver function, the bird's blood may not clot properly, and black spots may be seen on the beak and toenails. The spots are areas of hemorrhage, like bruising.

- *Enlarged fatty liver.* Though an enlarged liver cannot always be detected with the naked eye, it can be palpated manually during a physical examination. In small avian patients, the liver can be seen through the skin if the feathers and skin are moistened with a small amount of alcohol.

Most birds that show all or most of these clinical signs are in serious condition. A high fat level may disrupt the body's normal metabolism, compromise the functioning of its organs, and subject the bird to secondary disease. The bird may not be able to withstand even simple stress, and sudden death can occur.

However, the disease can be detected before it gets to a serious state through regular physical examinations that include blood testing. Many times, the blood tests will show lipemia, anemia, and icteric (jaundiced) plasma, indicating liver disease.

Identified early, the disease can be treated with nutritional management. Ideally, it can be prevented entirely through proper nutrition with an adequate homemade or commercially prepared diet, such as a formulated diet. If the bird is on an all seed or high fat diet, dietary change to a more balanced diet is of utmost importance. A conversion to such a diet needs to be done carefully as some birds do not readily accept such change. In more severe cases medications may be prescribed, such as colchicine/probenecid used as a "liver sparing" drug.

Vitamin A Imbalances

Hypovitaminosis A

Birds on all-seed diets will likely suffer from a vitamin A deficiency, since peanuts and most seeds and grains (except yellow corn) are devoid of this vitamin. Because vitamin A maintains mucous membranes and other epithelial surfaces, low levels of vitamin A will result in decreased resistance of these tissues to disease-producing organisms. As a result, respiratory and sinus infections are more severe in birds with deficient levels of vitamin A. Vitamin A deficiency can also lead to hyperkeratosis, or thickening of the skin with excessive scaliness and flakiness of the feet.

Signs of hypovitaminosis A often seen by avian veterinarians include white plaques on the roof of the mouth, excessive oral mucus (caused by changes in the salivary and tear glands), and

blunting of the choanal papillae. Quite often, birds brought to the hospital for some type of respiratory difficulty described by the owner as "sneezing" or "coughing". They have developed the condition as a result of the excessive mucus and mouth lesions caused by vitamin A deficiency. If secondary infections have developed, pustules or abscesses may be seen in the oral cavity, crop, or respiratory tract. In severe cases of vitamin A deficiency there may also be changes in the kidneys that can lead to gout. Other signs of vitamin A deficiency are fading of the pigmentation of the skin and feathers, failure of young birds to gain weight, low hatchability rates, and high hatching mortality.

The best and safest way to ensure that a bird will not suffer from a vitamin A deficiency is to provide it with adequate nutrition. Birds that prefer seeds should be given a commercial vitamin supplement, administered on the food or in the water. All birds should also be provided with foods that are rich in vitamin A, such as red or orange vegetables (carrots, sweet potatoes, red peppers, squash, mango, papaya) and dark green leafy vegetables (spinach, broccoli, collards). In addition, beta-carotene supplementation is effective. Readily converted by birds to the active form of vitamin A, beta-carotene cannot be oversupplemented to produce toxicity. Injectable vitamin A is usually used for clinical cases.

Another means of supplementation is to periodically add a few drops of vitamin A from a punctured vitamin A gel capsule to the bird's diet. Some breeders add a teaspoonful of cod liver oil to each pound of seed, since vitamin A has been shown to improve reproductive results. The use of cod liver oil is not recommended, however, as it can become rancid and promote the destruction of vitamin E. In all of the other forms noted, vitamin A supplementation is an effective means of increasing birds' resistance to disease and yielding better reproductive results.

Hypervitaminosis A

Oversupplementation of vitamin A has the potential to be toxic in birds (as in other animals), but this condition is not well documented in birds. In other animals, overdoses of vitamin A have been shown to cause a wide variety of signs, including weakness and bone abnormalities.[5]

Calcium, Phosphorus, and Vitamin D3 Imbalances

Calcium/Phosphorus Imbalances

Diets that consist of seeds and grains, especially oily ones, are deficient in calcium, have a low ratio of calcium to phosphorus, and have low levels of vitamin D3. In addition, the oils in these seeds and grains may bind calcium in the intestine to form insoluble "soaps" that prevent its absorption. Due to the high-grain diet most birds are fed, the most common mineral disorders in birds are calcium and phosphorus deficiencies and imbalances.

Calcium, as the chief mineral constituent of the bird's body and of the whole egg, is required in the avian diet in larger amounts than any other mineral. Among its other purposes, it is essential for the mineralization of the skeleton; if insufficient calcium, phosphorus, or both are absorbed from the intestine, bone development will be abnormal or extremely fragile. Calcium is also required for muscle contraction, nerve impulse transmission, and many other metabolic processes.

Because calcium metabolism by the body is linked to phosphorus and vitamin D, adequate amounts of these nutrients are necessary for the proper utilization of calcium. Calcium and phosphorus are in equilibrium in the body at a ratio of 2:1. In the diet, the optimal ratio should be 2:1, although a range of 0.5:1 to 2.5:1 can be tolerated. Vitamin D3 is required for the absorption of calcium into the bloodstream from the intestinal tract.

Adult birds suffering from calcium deficiencies may become weak or ataxic (uncoordinated) and may develop bone weakness, osteoporosis, and pathological fractures. Laying hens that are not properly supplemented are at highest risk for calcium deficiency disorders. Acute hypocalcemia in laying hens is thought to result in partial paresis (weakness) and egg binding. Seen in excessive egg layers (notably unpaired cockatiels, lovebirds, and finches), this condition is often alleviated with calcium supplementation, including intramuscular injections of calcium. An acute hypocalcemic syndrome has also been seen in the African gray parrot, which may present with seizures and blood calcium levels of under 6 mg%.

In chicks and immature birds (particularly cockatiels, grass parakeets, rosellas, doves, toucans, and orphaned wild birds), calcium and vitamin D3 deficiencies are often the result of poor dietary supplementation of the hen and will often manifest as bone and joint deformities. Rickets (vitamin D3 deficiency), in the young chicken, is characterized by long-bone deformities (bowing) and folding fractures. Splay-leg deformities are not well understood, but in ratites, they have been linked to rapid growth rates and high relative protein levels in the diet.

To overcome calcium deficiencies, supplementation is essential, but it must be done properly. An excess of either calcium or phosphorus can lead to problems.

High calcium in the diet can cause other problems as well. Young, growing birds fed high-calcium diets can develop kidney problems leading to kidney failure and mineralization. In addition, high calcium levels without increased levels of manganese and zinc will interfere with the absorption of these trace elements.

If too much phosphorus is provided, low calcium levels will result. The excess phosphorus will bind with calcium in the intestine to form an insoluble chemical, calcium phosphate. The remaining phosphorus is still absorbed, but blood calcium levels will be low.

Vitamin D Imbalances

The primary function of vitamin D is to regulate calcium metabolism. Vitamin D toxicosis occurs when vitamin D is present in the diet at high enough levels to stimulate the excessive absorption of calcium from the diet or its resorption from the bone. Hypercalcemia occurs, but initially (before calcium levels get too high and with a normal glomerular filtration rate [GFR] by the kidneys) the calcium can be excreted. The GFR will fall, however, when nephrolithiasis (kidney stones) occurs due to prolonged hypercalciuria (high levels of calcium in the urine), producing the elevated blood calcium levels that occur with vitamin D toxicosis.

The level of vitamin D that causes toxicosis varies with the form of vitamin D, amounts of vitamin A and calcium in the diet, and the health of the kidneys. Cholecalciferol (vitamin D3), which is used in most diets and supplements, is 10–20 times more toxic than ergocalciferol

(vitamin D2) in birds that have been tested. However, ergocalciferol is not effective in replacing vitamin D3 and its function.

Oversupplementation with vitamin D3 can cause kidney mineralization and widespread metastatic calcification due to the increased calcium absorption. Decreasing calcium in the diet can slow the rate of nephrolithiasis if toxic levels of vitamin D3 have been fed.

The safest and most effective way to help overcome mineral imbalances is to provide a nutritionally adequate diet, either homemade or commercially prepared (such as a formulated diet). In seedeaters, a balanced calcium/phosphorus/vitamin D3 supplement should be provided. Other sources of minerals that have been shown to be safe for birds are calcium carbonate (found, for example, in cuttlebones, plaster blocks, mineral blocks, and oyster shells), calcium gluconate supplements, liquid-combination calcium available commercially, and such natural sources as spinach, broccoli, cheese, yogurt, and milk. Crushed eggshells may also be used but must not be raw due to the risk of salmonellosis.

Care must be taken when supplementing young birds. Most commercial hand-feeding diets contain adequate levels of minerals; adding additional amounts, unless clinically indicated, can lead to mineralization of the kidneys.

Iodine Deficiency/Goiter

Many seeds are deficient in iodine, which is a building block of thyroxine (thyroid hormone) and is essential for normal thyroid gland function. For that reason, birds fed seed diets must receive supplemental iodine as a part of a balanced vitamin/mineral source added to the food or water. (Iodine appears to be provided in adequate amounts in formulated diets.) Iodine is especially important in budgies, which appear to be particularly susceptible to thyroid problems. The same condition has occasionally been reported in other species of birds.

Iodine deficiency can lead to thyroid dysplasia (a malformation of the thyroid gland), which is commonly termed goiter. Because the thyroid glands are located at the branching of the trachea into each lung, just above the heart, the enlarged, dysplastic glands put pressure on the trachea and syrinx(voicebox), with resultant displacement and breathing difficulties. A click, wheeze, or the incessant “squeaking” in a budgerigar, heard on both inspiration and expiration, will point to this condition. Any budgerigar presented for respiratory wheezing should be evaluated for thyroid hyperplasia. Other clinical signs can include vomiting and engorgement of the jugular vein, due to the partial occlusion of the thoracic inlet.

The development of thyroid dysplasia is gradual, and the condition worsens over time, with the respiratory noises becoming more noticeable as the thyroids enlarge. Quite often, the bird will be in extreme distress, sometimes forced to hold its head upright to facilitate breathing. Further, the condition can be complicated by secondary invasion of bacteria and fungus. The iodine deficiency may also produce some degree of hypothyroidism, which is manifested by weight gain, the development of fat deposits, lethargy, and poor feather quality.
Although thyroid dysplasia is mainly seen in budgies and occurs in rare occasions in cockatiels and canaries, the potential exists for all birds to suffer from iodine deficiency. The severity of the condition will dictate treatment: A mild case can be corrected by the addition of supplemental

iodine in the diet; a severe case may require hospitalization and daily injections of sodium iodide until the condition resolves. The best manner of treatment, however, is prevention through the proper provision of trace elements in a formulated diet or the use of a supplement for birds on a seed diet.

Budgerigars with thyroid tumors may have clinical signs identical to goiter. However, while goiter will resolve quickly with iodine supplementation, thyroid tumors will not.

Hemochromatosis

Hemochromatosis, or iron storage disease, develops from an inability to eliminate iron, leading to liver, renal, and heart damage. Little more than that is understood about the condition, however. Chronic stress with resultant blood breakdown, a lack of certain enzymes, genetic predisposition, and combination etiologies have all been suggested as causes. Hemochromatosis is mainly seen in mynahs and toucans and often results in their presentation for dyspnea (difficulty breathing). Other signs include a distended abdomen (ascites and liver changes) and discolored droppings.

Birds with this condition are generally treated with long-term weekly phlebotomies (blood letting) to reduce the iron load. In the procedure, 10% of the bird's blood volume can be removed safely at one time, which equals approximately 1% of the bird's body weight. In birds with ascites (abdominal fluid), this percentage should be decreased, as their body weight may be artificially elevated. Serum iron levels should be monitored to ensure that they are 150 mg% or less, and a CBC or hematocrit should be used to make certain that the bird has recovered from each phlebotomy. Deferoxamine, an iron chelator, has also been used in the treatment of this condition.

Prevention of iron storage disease can be achieved through dietary management, which has been facilitated by the availability of better diets. Low-iron diets and bottled water are recommended for birds in any species that are susceptible to the condition that have not developed the disease. Long-lived mynahs that have not developed the condition have been found to eat a diet high in human food with little reliance on mynah pellets.

Adapted from *Essentials of Avian Medicine: A Guide for Practitioners, Second Edition* by Peter S. Sakas, DVM, MS. Published by the American Animal Hospital Association Press. (2002)

[5] Fraser, C.M., Bergeron, J.A., Mays, A., and Aiello, S., eds. The Merck veterinary manual, 7th ed., p. 591. Rahway, NJ: Merck & Co.; 1991. ***(LB: Is there a chapter title for what's on p. 591? And is NJ the right state—I added it.)***