NUTRITIONAL DISEASES OF AMPHIBIANS

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Introduction

Basic knowledge of amphibian nutrition lags far behind that of reptiles and even fish. Recent documentation of worldwide declines and extinctions of amphibian populations and subsequent development of a variety of *ex-situ* and *in-situ* captive propagation programs will almost certainly increase the demand for scientifically based programs for nutritional management of these animals. Because the class Amphibia has such rich species diversity, development of a satisfactory "one size fits all" approach to dietary husbandry is unlikely. Approaches that incorporate careful observations of species natural history along with experimental studies as has been documented for some reptile species will be necessary.[3] This paper reviews common and emerging nutritional diseases of amphibians observed in the author's diagnostic practice.

Metabolic Bone Disease

Metabolic bone disease (MBD) is one of the best-recognized amphibian nutritional diseases and is usually due to calcium deficiency as a result of inadequately supplemented insect-based diets.[7,9] Specifics of vitamin D metabolism including the need for UV light supplementation are poorly understood for most amphibian species (but light supplementation is still encouraged). When investigating cases of MBD attention should be given to factors such as water hardness and water phosphate levels as well as dietary management.[5,8] There is one report of MBD possibly associated with hypervitaminosis A in African clawed frogs (*Xenopus laevis*).[2] This observation should not discourage vitamin A supplementation in amphibians on insectivorous diets as affected clawed frogs were fed a diet expected to be very rich in vitamin A (liver). Early clinical signs of calcium deficiencies in amphibians may be limited to tetany or subcutaneous (lymph sac) fluid accumulation. Advanced or chronic cases may have decreased bone mineralization and skeletal deformities.

"Short Tongue Syndrome" (Suspected hypovitaminosis A)

A recently described condition most often observed in bufonids (toads) in which affected animals lose the ability to prehend prey despite vigorous effort.[4] The author has noted this condition in species of special conservation concern including Wyoming toads, Puerto Rican crested toads, Kihansi spray toads and Panamanian golden frogs. Histologic lesions can include replacement of normal mucus-secreting epithelium of the tongue with squamous epithelium (squamous metaplasia) consistent with vitamin A deficiency. Captive Wyoming toads with lingual squamous metaplasia had a mean liver retinol of 1.6 micrograms/gram compared with a mean of 104.6 micrograms/gram for free-ranging toads without histologic evidence of squamous metaplasia. Treatment of affected animals has been reported successful using human vitamin A liquid gel caps diluted in propylene glycol or vegetable oil to provide a dose of 1 IU/gram body

weight daily for 2 weeks. Crickets used as a mainstay for many amphibian diets are known to be deficient in vitamin A and require supplementation by gut loading and/or dusting with vitamin powders. Review of dietary supplementation practices (including length and method of supplement storage) may be helpful in nutritional management of captive amphibian colonies. Feeding trials to experimentally reproduce short tongue syndrome and lingual squamous metaplasia in toads as well as studies of carotenoid metabolism in amphibian species are needed.

Lipid Keratopathy (Corneal Lipidosis)

Lipid keratopathy is a frequently observed disease in captive frogs. Clinical signs include progressive white discoloration and opacity of the cornea. Histologically, there are corneal deposits of cholesterol and infiltrates of foamy (lipid-laden) macrophages. The condition has been experimentally associated with a high cholesterol diet in Cuban tree frogs.[6] Affected animals have elevated serum cholesterol. Domestic crickets may have cholesterol contents higher than foods typically consumed by many amphibian species and a possible relationship to corneal lipidosis should be explored.

Inanition and Maladaption Syndromes

Postmortem evaluation of overall body (nutritional) condition most often relies on the size of the abdominal fat bodies. Inanition with depleted fat stores is a common finding especially in recently imported animals. This may become more of a challenge for zoo nutritionists as more novel and specialized amphibian species are brought into captivity for conservation programs.

Gastric Overload, Impaction and Foreign Bodies

Amphibians can be voracious feeders and occasionally will overestimate their capability to ingest large prey items resulting in gastric overload or impaction. Ingestion of foreign bodies such as stones or other cage substrate can occur as prey items are consumed. The author has observed intestinal obstruction associated with ingestion of indigestible acrylamide-based products that are available commercially for providing hydration to domestic crickets. Occasionally, insect mouthparts or ovipositors can traumatize or perforate the stomach wall.

Suspected B-Vitamin Deficiencies

Thiamine deficiencies associated with the feeding of fish containing thiaminases is a possible cause of neurologic signs.[7] Possible thiamine or other B-vitamin deficiencies have also been suspected in association with a paralysis syndrome in dendrobatid frogs.[7] Finally, spindly leg syndrome is a common developmental problem of the limbs that reportedly can be reduced in frequency with vitamin B complex supplementation.[7] Experimental evaluation of B-vitamin deficiencies in amphibians may be useful.

Gout and Urinary Calculi

With the exception of some uricotelic species such the waxy tree frogs (*Phyllomedusa sp.*) most amphibians produce either ammonia or urea as the primary nitrogenous waste product. Hence, in

contrast to reptiles, gout is only a very rarely encountered condition. Urinary bladder stones composed of urates can be observed in the uricotelic species and could be associated with hydration status. Calcium oxalate associated renal calculi or nephrosis has been observed in some tadpoles and postmetamorphic animals with dietary exposure (directly or via prey items) to plant oxalates.

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