

Bon Appetit! Nutritional Considerations for Exotic Pets

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Exotic pets come in all shapes and sizes and can be herbivorous, omnivorous, or carnivorous, depending on the species. An ideal diet for an exotic pet in captivity should mimic their natural diet as closely as possible and provide a diversified selection of food. Some herbivorous species will readily eat an omnivorous diet, but eventually these animals will reveal signs of nutritional deficiencies. Therefore, it is important to remember that food preferences do not always correlate with appropriate nutrition.

Herbivorous species are primarily classified as hindgut fermenters or ruminants, with microbial fermentation occurring in the large intestine or stomach, respectively. Consequently, the bulk of the diet of herbivorous species should be plant fiber. The plant fiber offered should be rich in vitamins A and D₃ and should have more available calcium than phosphorous. An ideal Ca:P ratio of at least 1.5-2:1 should be present. The diet should also be low in fats, oils, proteins, thiocyanates, and oxylates. Herbivorous species housed outdoors will forage for themselves if provided with an appropriately planted enclosure, but additional food is usually required. It is important to periodically peruse the yard and rule-out the presence of any poisonous plants. A variety of foods should be offered and can be mixed with calcium, iodine, vitamin D₃, and vitamin A supplementation. It is important to remember that grocery greens are generally higher in protein, lower in fiber, and may have an inverse calcium: phosphorus ratio when compared to natural forage. Spinach, cabbage, and beet greens should not be fed in excess due to their high oxylate content. The majority of foods designed for dogs, cats, humans, and other mammals should not be fed to herbivorous exotic pets. Debilitated herbivorous species requiring force-feeding or tube-feedings should be fed a critical care diet designed for their specific needs.

It has been suggested that omnivorous exotic pets do best when offered plant and animal matter in proportions that range from 75:25 to 90:10. Dietary requirements in these species tend to change with age, with most juveniles requiring a diet comprised of a higher proportion of animal matter. As the juveniles mature, their dietary requirements shift to a more herbivorous diet. The primary animal proteins offered should mimic a natural diet, including earthworms, slugs, snails, millipedes, pupae, and maggots (mealworms). It is essential to monitor the diets of captive invertebrates in order to avoid nutritional deficiencies in the animals eating them. Offering the invertebrates a diet rich in minerals and vitamins will help to ensure that the prey is “gut-loaded”. Liver and yellow or dark orange colored vegetables (squash, carrots, and sweet potatoes) are excellent sources of vitamin A, and Swiss chard, kale, beet greens, escarole, parsley, watercress, and green beans all have a positive Ca:P ratio.

Carnivorous exotic pets are generally the easiest group to provide food for in captivity, as there is a range of invertebrate and vertebrate prey species that can be offered. However, as was mentioned previously, those carnivores that specifically hunt invertebrates do need to have their prey species “gut-loaded”. Most carnivorous aquatic species are piscivorous. If frozen fish are offered, then the diet needs to be supplemented with thiamine, as frozen-thawed fish can produce thiaminases.

Nutritional disorders in exotic pets commonly present as a chronic problem, and the diet is often times centered around limited food sources or human convenience. In most cases, deficient diets are comprised of limited numbers of food items and/or are not supplemented with calcium and vitamin powders.

Vitamin A is a critical component in the production and maintenance of epithelial cells, and is intimately associated with several structures related to vision. Hypovitaminosis A is a common clinical entity in exotic pet medicine, especially in birds and reptiles fed predominately vitamin A deficient foods. The most obvious clinical abnormality associated with hypovitaminosis A is squamous metaplasia, which results in the degeneration of epithelial surfaces (e.g., conjunctiva, gingiva, pancreatic ducts, renal tubules, and skin). Due to the multiple epithelial surfaces of the body, squamous metaplasia can manifest itself in several different ways. Blepharospasm, conjunctivitis, blepharodema, blindness, rhinitis, blepharitis, lower respiratory tract disease (nasal discharge, depression, dyspnea, open-mouth breathing), and/or cutaneous abnormalities may be observed. Middle ear infections and aural abscesses have also been linked with hypovitaminosis A. The diagnosis of hypovitaminosis A can be met via dietary history, clinical signs, measuring vitamin A concentrations, or histopathology of tissue samples (squamous metaplasia of the epithelia surfaces). Supportive treatment should be utilized concerning the clinical manifestations of vitamin A deficiency, and appropriate husbandry and dietary changes should be instituted. Vitamin A deficiency can be corrected by oral supplementation with vitamin A products, or by offering small amounts of liver once per week. Injectable vitamin A should be used very cautiously, as hypervitaminosis A can occur with a single injection.

Metabolic bone disease (MBD) is defined as any metabolic defect that alters the morphology and functioning of bones. MBD is usually related to low levels of calcium or excessive levels of phosphorus, which consequently bind to calcium and render it physiologically unavailable. Decreased calcium availability results in increased parathyroid activity and mobilization of stored calcium from the shell and bone cortices. Factors predisposing exotic pets to the development of MBD include: dietary deficiency of calcium and/or suitable vitamin D₃, inappropriate calcium: phosphorus ratio in the diet, lack of exposure to ultraviolet light (ultraviolet B radiation increases activation of vitamin D precursors and facilitates gastrointestinal absorption of calcium), dietary excess of protein during rapid growth periods, anorexia, and abnormal vitamin D₃ metabolism secondary to renal, hepatic, intestinal, or parathyroid disease. MBD is commonly observed in rapidly growing juvenile animals and reproductively active females. Clinical signs consistent with MBD vary depending on the age and species of the patient. The most common clinical findings in affected animals include muscle tremors/fasciculations, seizures, soft-shells, pathologic fractures and acute death. A thorough history is required before a diagnosis of MBD can be met. Diagnostically, radiographs and blood work can provide insight into the animal's disease state. Radiography may reveal fibrous osteodystrophy and pathologic fractures. Low blood calcium levels are highly suggestive of MBD, but calcium blood levels are frequently not low in cases of MBD because of hyperparathyroid activity. It must be remembered that blood levels of calcium are not reflective of physiologically available levels of calcium. Ionized levels of calcium are more indicative of the availability of calcium, but, unfortunately, published reference levels are difficult to find in the literature. Treatment of MBD is dependent upon the correction of inappropriate husbandry. An unsuitable calcium: phosphorus ratio of the diet should be corrected, the proper provision of

ultraviolet light should be instituted and oral supplementation of calcium and vitamin D₃ should be initiated. Supplemental calcium during the treatment period is also strongly recommended.

Gout is defined as the deposition of uric acid and urate salts within visceral tissues and on articular surfaces. Gout occurs as a result of hyperuricemia, which arises secondary to increased production or decreased excretion of uric acid. Increased production of uric acid may occur secondary to the ingestion of excessive amounts of protein (e.g., an herbivorous chelonian that is regularly offered animal protein). Decreased excretion of uric acid may occur secondary to reduced perfusion of renal tissues, which may be a result of dehydration, hemoconcentration, water deprivation, or renal disease. Reduced glomerular filtration eventually leads to a decrease in the overall excretion of urate salts, which results in hyperuricemia. Hyperuricemia, in turn, leads to the precipitation of urate complex microcrystals within tissues. These deposits are known as “gout tophi”. Common sites of deposition of uric acid include articular joints and viscera. Clinical signs associated with gout include joint swelling and pain, depression, and dehydration. Affected animals are also commonly anorectic and lethargic. The diagnosis of gout may be done with blood work and radiographs/ultrasound. The mainstay of therapy is rehydration to correct the hyperuricemia, and the correction of any dietary imbalances or other predisposing causes of gout. Allopurinol, a urease inhibitor, is commonly used in hyperuricemic animals to reduce uric acid production. It must be mentioned that studies concerning the efficacy of this drug and the possible long-term effects of the drug in exotic pets have not been conducted. Probenecid, which increases the renal excretion of uric acid, should not be used until the glomerular filtration rate is considered acceptable. Any concurrent infections in affected joints or organs that occur secondary to gout deposition should be treated appropriately. Surgery is occasionally indicated when uric acid deposits are compromising joints.

Providing appropriate nutrition is key to having success with exotic pets. By providing the essentials, we can minimize the likelihood for these common nutritional diseases outlined previously. Take the time to learn about the needs of the species you work with, so that you can make the best recommendations for your clients.

Reference

Carpenter JW. 2018. Exotic Animal Formulary. Elsevier, St. Louis, MO, pp: 701.