Update on Common Nutritional Disorders of Captive Reptiles

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KEYWORDS

• Nutritional secondary hyperparathyroidism • Hypovitaminosis A • Obesity
• Dehydration • Constipation • Hypothyroidism

KEY POINTS

- Nutritional secondary hyperparathyroidism is the most important nutritional disorder in captive reptiles and is either caused by a deficiency in dietary calcium and/or vitamin D_3 or inadequate exposure to UV-B radiation.
- Hypovitaminosis A is predominately a disorder of omnivorous and carnivorous reptiles that receive insufficient dietary vitamin A. Herbivorous reptiles rarely suffer from this disorder because of their ability to endogenously synthetize vitamin A from plant-derived provitamins contained in their diet.
- Nutritional secondary hypothyroidism caused by excessive intake of goiterogenic plants is predominately a disorder of herbivorous reptiles. The diagnosis should be made carefully because evaluation of thyroid function has not been reported in reptiles and visualization of the thyroid gland may be challenging because of the intracoelomic location of the gland.

INTRODUCTION

Nutritional disorders of captive reptiles remain very common despite the increasing knowledge about reptile husbandry and nutrition. Many nutritional disorders are diagnosed late in the disease process and often secondary complications have occurred. Therefore, every attempt should be made to educate reptile owners and keepers about the proper care and, in particular, dietary needs of reptiles under their care because all nutritional disorders seen in captive reptiles are preventable.

However, because the class Reptilia is so diverse and reptiles have adapted to such a large variety of climates, habitats, and natural diets, making general applicable

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recommendations challenging. A detailed knowledge about the natural habitat and diet of reptiles commonly seen by veterinarians is important in order to provide the appropriate advice to reptile owners and keepers. Although the recommendations on appropriate captive diets for reptiles are based predominately on experience and anecdotal information, a growing body of scientifically derived knowledge is available to support dietary and husbandry recommendations. This article attempts to summarize the current information on the most common nutritional disorders of reptiles. Excellent reviews on basic nutrition of reptiles have been published in the literature.[1,2] The reader is encouraged to use this article as a supplement and update to the existing literature.

NUTRITIONAL SECONDARY HYPERPARATHYROIDISM

Nutritional secondary hyperparathyroidism (NSHP) in captive reptiles occurs because of dietary calcium and/or vitamin D_3 deficiencies. Deficiencies in vitamin D_3 (cholecalciferol) can occur because of insufficient dietary intake in nonherbivorous reptile species or in most cases because of a lack of adequate exposure to UV-B radiation, which is required for endogenous vitamin D_3 synthesis. NSHP is the most common nutritional disorder of captive reptiles, particularly in lizards and chelonians as well as herbivorous and insectivorous species. Animals feeding exclusively on whole prey (ie, snakes) are not affected by NSHP because whole vertebrate prey contains adequate amounts of calcium and vitamin D_3.

Bone is the major storage site of body calcium, with about 99% of body calcium stored. Vitamin D_3, calcitonin, and parathyroid hormone (PTH) control storage and release of calcium from the bone.[3] Chronic reduced blood calcium levels will trigger increased secretion of PTH from the parathyroid glands, resulting in hyperparathyroidism. Increased secretion of PTH leads to increased resorption of calcium from the bones, resulting in osteomalacia (syn fibrous osteodystrophy, nutrition-related metabolic bone disease).

Dietary factors that can lead to NSHP include insufficient calcium intake; inappropriate calcium/phosphorous (Ca:P) ratio of the diet; insufficient calcium absorption from the intestine, which is vitamin D_3 dependent; insufficient dietary vitamin D intake; or insufficient exposure to UV-B radiation (280–315 nm). The biological active form of vitamin D_3 is 1,25-dihydroxycholecalciferol (calcitriol), which regulates calcium metabolism predominately by increasing intestinal calcium absorption and renal calcium reabsorption.[3] UV-B is necessary to activate the cholecalciferol pathway in species that rely not on dietary vitamin D_3 intake, which is the case in herbivorous reptiles. If vitamin D_3 is predominately of dietary origin or endogenously synthetized differs between reptile species based on their nutritional (carnivorous, omnivorous, herbivorous) strategy and natural behavior (eg, nocturnal vs diurnal) and natural habitat.

Effect of UV-B Radiation

In omnivorous and carnivorous reptiles, the need for provision of artificial UV-B radiation in captivity remains controversial. In most reptile species evaluated, exposure to UV-B radiation will lead to increased plasma vitamin D levels. Exposure to artificial UV-B in panther chameleons (*Furcifer pardalis*) had a significant effect on plasma 25-hydroxycholecalciferol.[4] Red-eared sliders (*Trachemys scripta elegans*) had significantly higher plasma 25-hydroxycholecalciferol concentrations if turtles were exposed to artificial UV-B radiation for 4 weeks.[5] In corn snakes, exposure to artificial UV-B radiation for 4 weeks significantly increased plasma 25-hydroxycholecalciferol levels.[6] In contrast, exposing ball pythons (*Python regius*) to artificial UV-B radiation