Thyroid Diseases in Rodent Species

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KEYWORDS

• Thyroid disease • Hyperthyroidism • Hypothyroidism • Thyroid neoplasia

KEY POINTS

- There are abundant studies of thyroid disease in rodents in a laboratory setting; however, research is limited in pet rodent populations.
- Validated reference intervals for thyroid testing are scarce, making interpretation of blood work difficult.
- Hyperthyroidism and hypothyroidism has been reported in multiple rodent species, with the most data available for guinea pigs.
- Thyroid neoplasia has been reported in the most common rodent species although often as an incidental finding at necropsy.
- Further research is needed to determine frequencies of disease, methods of diagnosing, and treatment options with the best efficacy.

INTRODUCTION

Information available on naturally occurring thyroid disease in small mammals is extremely limited. Although there is an abundance of research with experimentally induced thyroid disease in laboratory rodents, this does not follow the pattern of naturally occurring diseases in these species and often has limited value when evaluating pet rodents. Overall, naturally occurring thyroid gland dysfunction is poorly documented in rodents, and more research is needed to explore these diseases. The expense associated with diagnostics is a limiting factor for many exotic pet owners, often preventing clinicians from being able to diagnose thyroid diseases definitively. Furthermore, due to the small size of most rodents, it may difficult to obtain sufficient blood volume to run multiple diagnostics. Even if enough blood is collected, it is also quite challenging to interpret the results because the assays have not been validated in most rodent species.

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ANATOMY

The thyroid gland, located at the cranial-ventral aspect of the trachea, is bilobed and connected by an isthmus.[1] It is closely associated with the parathyroid glands, which are responsible for secretion of parathyroid hormone. Based on embryologic development, it is possible to have ectopic thyroid tissue in other locations, ranging from the larynx to the diaphragm[2]; this becomes important if the ectopic tissue begins secreting excess thyroid hormones.

Histologically, the thyroid gland is composed of 2 cell types: follicular cells, which predominate, and C cells, which are also known as parafollicular cells.[2] The main functional units of the thyroid gland are the numerous follicles, which are lined by low cuboidal epithelium (follicular cells).[1,2] C cells are found in small groups of 10 cells or fewer scattered throughout the gland, either in connective tissue between follicles or at the base of the follicular cells. These cells account for 0.1% of thyroid epithelial cells.[1]

PHYSIOLOGY

Thyroid hormones are essential for normal body function including growth and the development of neurologic and skeletal systems.[2] They increase normal metabolism in most tissues and have catabolic effects on muscle and fat. They also increase the number and affinity of β-adrenergic receptors, which have inotropic and chronotropic effects on the heart. The hormones are also responsible for erythropoiesis and cholesterol synthesis and breakdown.[3] In rodents, thyroid hormones, along with catecholamines, play an important role in thermogenesis of brown fat, allowing them to adapt to cold temperatures.[4]

As stated previously, the thyroid gland consists of 2 primary cell types. Follicular cells produce thyroglobulin and are responsible for the uptake and oxidation of iodine. Once oxidized, the iodine is released into the follicular lumen and interacts with thyroglobulin, producing mostly thyroxine (T4) and a small amount of triiodothyronine (T3). T4 is the main product of the thyroid gland and is solely produced there. In contrast, only a small portion of T3 is made in the thyroid gland; the remainder is produced from deiodination of T4 to T3 within target cells.[3] When signaled to be released, the iodinated thyroglobulin undergoes hydrolysis, releasing T3 and T4 into the bloodstream.[2]

The vast majority (>99%) of serum thyroid hormones are stored in the biologically inactive, protein-bound state.[3] It is the unbound, free T4 (fT4) and T3 (fT3) that are biologically active and able to enter cells and stimulate negative feedback on the pituitary gland and hypothalamus. Circulating fT3 enters cells more rapidly and has a faster onset of action than T4.[3] Once in cells, T3 is able to act directly, and T4 is deiodinated to the more bioactive T3, which acts on the cell to exert its effects.

Thyroid hormone production and release are controlled by the hypothalamic-pituitary-thyroid axis. The hypothalamus detects decreased circulating levels of thyroid hormones and releases thyrotropin-releasing hormone (TRH). TRH acts on the anterior pituitary gland to release thyrotropin-stimulating hormone (TSH), which stimulates T3 and T4 release from the thyroid gland. Rising levels of thyroid hormones in the blood initiates a negative feedback loop, which inhibits additional release of TSH.[3]

The thyroid also contains C cells, which are responsible for the production of calcitonin. Calcitonin is responsible for controlling calcium deposition in bone by inhibiting bone resorption by osteoclasts, leading to decreased serum calcium levels. Calcitonin acts in opposition to parathyroid hormone.[2]