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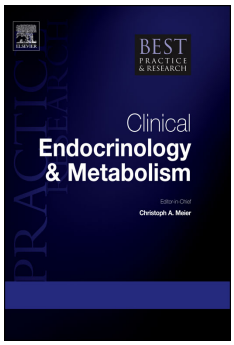

Genetically modified mouse models to investigate thyroid development, function and growth

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Genetically modified mouse models to investigate thyroid development, function and growth

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Abstract

The thyroid gland produces thyroid hormones (THs), which are essential regulators for growth, development and metabolism. The thyroid is mainly controlled by the thyroid-stimulating hormone (TSH), which binds to its receptor (TSHR) on thyrocytes and mediates its action via different G protein-mediated signaling pathways. TSH primarily activates the G_s- pathway, and at higher concentrations also the $G_{q/11}$-pathway, leading to an increase of intracellular cAMP and Ca^{2+}, respectively. To date, the physiological importance of other G protein-mediated signaling pathways in thyrocytes is unclear. Congenital hypothyroidism (CH) is defined as the lack of THs at birth. In familial cases, high-throughput sequencing methods have facilitated the identification of novel mutations. Nevertheless, the precise etiology of CH yet remains unravelled in a proportion of cases. Genetically modified mouse models can reveal new pathophysiological mechanisms of thyroid diseases. Here, we will present an overview of genetic mouse models for thyroid diseases, which have provided crucial insights into the thyroid gland development, function, and growth with a special focus on TSHR and microRNA signaling.

KEY WORDS Thyroid, thyroid-stimulating hormone receptor, G protein, Cre/LoxP-system, microRNA, Dicer1, congenital hypothyroidism