Thyroid Hormones and Adipose Tissues

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Received: 4-July-2022, Manuscript No. jshs-22-18706; **Editor assigned:** 18-July-2022, PreQC No. jshs-22- 18706 (PQ); **Reviewed**: 25-July-2022, QC No. jshs-22-18706 (Q); **Revised**: 27-July-2022, Manuscript No. jshs-22- 18706 (R); **Published**: 28-July-2022, doi no. 10.35248/ 2157-7536-22.13.4.241

Abstract

Complex homeostatic mechanisms, including those originating in adipose tissue, regulate energy balance maintenance. The primary function of adipose tissue is to store excess metabolic energy as fat. During periods of energy deprivation, the energy stored as fat can be mobilized (hunger, fasting, diseases). Adipose tissue also plays a homeostatic role, regulating energy balance and acting as an endocrine organ, secreting substances that regulate body homeostasis. White and Brown Adipose Tissues (WAT and BAT) with distinct phenotypes, functions, and regulation have been identified. WAT stores energy, whereas BAT releases it as heat. Brown and white adipocytes have distinct ontogenetic origins and lineages, and distinct WAT and BAT markers have been identified. WAT has been found to contain "brite" or beige adipose tissue, which shares some properties with BAT. Thyroid hormones have pleiotropic effects, regulating differentiation in many tissues, including adipose tissue. Adipogenesis is the process by which mature adipocytes are produced. It is regulated by several transcription factors (c/EBPs, PPARs) that coordinately activate specific genes, resulting in the adipocyte phenotype. T3 controls many genes involved in lipid mobilization and storage, as well as thermogenesis. Thyroid hormones regulate genes important for WAT and BAT function, including lipogenesis, lipolysis, thermogenesis, mitochondrial function, transcription factors, and nutrient availability. T3 regulates transcription factors directly through specific TREs in gene promoters. T3 availability is regulated by the deiodinases D3, D2, and D1. D3 is activated during proliferation, whereas D2 is associated with the adipocyte differentiation programme, supplying T3 for lipogenesis and thermogenesis.

Introduction

Thyroid hormones are important during developmental processes because they regulate multiple physiological systems in many tissues. T3 controls the development of many tissues by acting on specific cells, such as those in the cochlea and retina. Thyroid hormone supply is finely tuned and dose-specifically regulated in specific areas of the brain via sequential increases or decreases in D2 and D3 deiodinases, as studied in human foetal brain, cochlea, and amphibian and fish metamorphosis. Thyroid hormones work by regulating genes that are involved in the differentiation of many tissues. Thyroid hormones regulate the function of many tissues in adults, including the brain, muscle, heart, liver, adipose tissue, and skin, by controlling carbohydrate and lipid metabolism, protein transcription, and the basal metabolic rate. T3 acts via nuclear receptors, which are encoded by two genes, TR and TR, and have three isoforms: TR-1, TR-2, and TR-1. They form heterodimers with RXR by binding to Thyroid Response Elements (TREs) found in the promoters of target genes. Corepressors and coactivators influence T3 action. Thyroid hormone concentrations in tissues are modulated by the action of the deiodinases D1, D2, and D3, which regulate the amount required in each tissue. Thyroid hormones have a number of important targets, including adipose tissue. Aside from its role in lipid transport, synthesis, and mobilization, adipose tissue is the primary site of lipid storage. Adipose tissue stores energy in the form of fat so that it can be used during times of hunger or illness. Furthermore, adipose tissue functions as a homeostatic mechanism, regulating energy reserves and releasing many substances that maintain the organism's homeostasis; some of these substances, such as leptin, act as adipostats, regulating the amount of fat stored. White and brown adipose tissue (WAT and BAT) exist in mammals and have distinct phenotypes, functions, and regulation. For many years, White Adipose Tissue (WAT) was thought to be a lipid storage site. White adipocytes have a large lipid droplet that fills the cellular space, and the cellular structures (nuclei, mitochondria) are close to the cellular membrane. WAT is distributed in various anatomical locations, which have been broadly classified as subcutaneous, visceral, and intra-abdominal fat. Because an increase in visceral fat is associated with insulin resistance, metabolic syndrome, and cardiovascular diseases, each location has a different lipolytic sensitivity in response to hormones. Fat also covers other organs such as the kidney, heart, and gonads (perirenal or perigonadal depots). These adipose sites are not pure WAT, and some of them are in the primitive BAT sites found in hibernating animals. In humans, WAT is one of the largest tissues and is found in numerous depots throughout the body; it accounts for about 10%-15% of total body weight in healthy subjects and up to 50% in obese subjects.

The adaptive or facultative thermogenesis is controlled by Brown Adipose Tissue (BAT). In order to maintain energy balance, BAT is activated in response to cold exposure or fat diets, providing extra heat in stressful situations. BAT is abundant in small rodents, hibernating animals, and especially newborns; it is found in small pads in the interscapular and cervical region, protecting organs such as the heart, aorta, kidneys, and other organs that should be heated up during hibernation arousal. The primary function of BAT is to generate heat. The activation of the Uncoupling Protein 1 (UCP1), a mitochondrial protein that acts as a proton channel, uncoupling oxidative phosphorylation and producing heat instead of ATP, makes this possible. This activation is triggered by adrenergic stimulation, which increases following cold exposure. The BAT is an extremely innervated and irrigated tissue. BAT morphology is distinguished by multilocular lipid droplets that are easily mobilized, as well as numerous and active mitochondria, the number and activity of which increases in response to cold exposure (mitochondriogenesis). BAT activation is now being considered as a potential therapeutic tool in the fight against obesity. The analysis of the white and brown adipocyte lineages reveals that both cells have a distinct embryological origin. Brown adipocytes, unlike white adipocytes, have a myogenic origin, as evidenced by the expression of the myogenic marker, myogenic factor 5, Myf5+, which is also found in myoblasts. Several genes, including UCP1 and D2, have been identified to trace the presence of white and brown adipocytes, as well as markers of terminal differentiation.

Furthermore, a new type of adipose tissue known as "beige" or "brite" adipose tissue has recently been identified. WAT contains small clusters of brown-like adipocytes that express UCP-1 under certain conditions, which have been dubbed "brite" (brown-white) or "beige" adipocytes. They are multilocular and express UCP-1, Cidea (Cell Death Activator CIDE-A), and brown adipocyte markers such as PGC1 (PPAR Coactivator 1α). They are more common in specific anatomical locations, such as the inguinal fat. "Brite" adipocytes appear to be derived from different embryonic precursors than brown adipocytes and have distinct gene signatures. Its presence, abundance, and activity increase are regulated differently than brown adipocytes.

Cite this article: Stan L. S Thyroid Hormones and Adipose Tissues J Steriod Horm Sci. 2022,13(4), 001.