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PERSPECTIVE

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Activation of Adipose tissue thermogenesis induced by thyroid hormone (T3, T4)

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Description

Each warm-blooded animal produces heat through a process called thermogenesis. There is mandatory thermogenesis resulting from body metabolism during cold exposure, as well as adaptive thermogenesis via shivering and non-shivering processes. Nevertheless, White Adipose Tissue (WAT) can also go through browning via adrenergic stimulation to gain thermogenic potential. The latter mostly happens in Brown Adipose Tissue (BAT) and muscle. Due to the fact that hypothyroidism and hyperthyroidism, respectively, cause a drop in body temperature and an increase in body temperature, Thyroid Hormone (TH) also has significant impacts on thermoregulation. We refer to the thermogenesis that is TH-mediated and occurs under thermo neutral settings as "activated" thermogenesis. By activating the Un Coupling Protein (Ucp1), TH causes the brown and/or white adipose tissues to engage in uncoupled respiration, which produces heat. With its central action, TH stimulates browning and the BAT via the sympathetic nervous system. Recent research, however, also demonstrates that TH directly stimulates Ucp1 expression and thermogenesis in the BAT through an autophagy-dependent mechanism. Moreover, THs can influence thermogenesis in a way that is independent of Ucp1, most likely by activating exothermic metabolic pathways. The thermogenic effects of THs on adipose tissues are summarised in this paper.

Thermogenesis is a crucial survival strategy. When the environment is at the thermo neutral temperature, which for an adult man is roughly 23 °C, obligatory thermogenesis is adequate to sustain body temperature and proper bodily function. Adaptive (sometimes referred to as facultative) thermogenic processes that need or do not require shivering are activated when the surrounding temperature drops below thermo neutrality. By the contraction of skeletal muscles, shivering thermogenesis enhances heat generation in reaction to cold.

Adipose tissue comes in two main varieties: White Adipose Tissue (WAT) and Brown Adipose Tissue (BAT), which, respectively, store energy and produce body heat. When it surrounds intra-abdominal organs like the liver, pancreas, and intestines, WAT is stored viscerally and subcutaneously. Visceral WAT accumulation is strongly linked to both insulin resistance and diabetes. On the other hand, obesity and the risk of developing non-alcoholic fatty liver disease are adversely correlated with the quantity of the active BAT. By stimulating the Sympathetic Nervous System (SNS), which raises the amount of cyclic Adenosine Monophosphate (cAMP) inside BAT cells, cold exposure causes BAT to become acutely activated. The increased lipolysis of intracellular TAG into Free Fatty Acids (FFA) by Protein Kinase A (PKA) in response to the elevated cAMP level then serves as an energy source for -oxidation.

It's interesting to note that Thyroid Hormone (TH) and its analogues, by increasing BAT activity and browning under thermo neutral settings, can stimulate tissue remodelling in both BAT and WAT. To distinguish it from cold-induced adaptive thermogenesis, we previously referred to this mechanism as "activated" thermogenesis. While both cold exposure and TH can increase thermogenesis in the BAT, we discovered that there are fundamental distinctions in how the metabolic processes are controlled and maintained. Via a direct increase in metabolic rate in certain tissues, the two forms of TH—thyroxin (T4) and its active metabolite 3,5,30-triiodothyronine (T3)—regulate both obligatory and adaptive thermogenesis. Although hypothyroid people are more likely to experience hypothermia after extended cold exposure, normal thyroid level is crucial for adaptive thermogenesis in response to the cold.

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The nuclear receptor superfamily includes thyroid hormone receptors, which are ligand-inducible transcription factors. Thyroid hormone receptors a and b (TR and TR) are the two main TR isoforms. Target genes' promoter regions contain TH response elements, which ligand-bound TRs bind to to cause the production of the target genes.

In conclusion, TH and other TH analogues can boost the BAT and WAT's capacity for thermogenesis by promoting fatty acid -oxidation, lipogenesis, mitochondrial biogenesis, and autophagy. The negative effects of TH on other tissues, such the heart and bone, may be lessened by careful titration of TH and focused administration. With the present obesity pandemic, TH or its analogues may be viable therapeutic agents to boost energy expenditure and prevent weight gain. This is because TH stimulates the BAT activity and browns the WAT.