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Commentary

Comprehensive Analysis of Genetic Polymorphisms in Thyroid Hormone Transporters

Noa Lieke*

Department of Epidemiology, Erasmus University Medical Center, Rotterdam, The Netherlands

DESCRIPTION

Thyroxine (T4) and Triiodothyronine (T3) are the two main Thyroid Hormones (THs), which are essential regulators of growth, development, and metabolism. Certain thyroid hormone transporters, including Sodium (Na^+)/Taurocholate Co-Transporting Polypeptides (NTCP), Organic Anion-Transporting Polypeptides (OATPs), and members of the Monocarboxylate Transporter (MCT) family, help move them across cellular membranes. These transporters affect the bioavailability and physiological effects of THs by ensuring their appropriate intracellular distribution. Thyroid hormone homeostasis and a number of thyroid-related illnesses can be impacted by genetic variants in the genes encoding these transporters, which can change their function, expression, or regulation. Clarifying these polymorphisms' effects on thyroid physiology and clinical consequences requires a thorough understanding of them.

The *SLC16A2* gene encodes MCT8, one of the most extensively researched thyroid hormone transporters. Transporting T3, the active form of thyroid hormone, into target cells is a critical function of MCT8. Thyroid hormone metabolism and function may be significantly impacted by genetic mutations or polymorphisms in *SLC16A2*. For instance, Allan-Herndon-Dudley Syndrome (AHDS), a rare X-linked condition marked by severe cognitive deficits and aberrant thyroid hormone levels, is associated with mutations in *SLC16A2*. Because of poor T3 absorption into tissues, people with AHDS have higher serum T3 levels and lower T4 and reverse T3 (rT3) levels. In addition to these serious mutations, *SLC16A2* Single-Nucleotide Polymorphisms (SNPs) have been linked to variations in thyroid function tests as well as vulnerability to neuropsychiatric and metabolic disorders. MCT10, which is encoded by the *SLC16A10* gene, is another essential transporter that helps move T3 and T4 across membranes. Changes in the kinetics and tissue distribution of thyroid hormones have been associated with polymorphisms in *SLC16A10*. For example, some SNPs may affect MCT10's affinity or capacity for thyroid hormones, changing the hormones' intracellular concentrations and subsequent consequences. Thyroid hormone signaling may be affected subtly but significantly by these polymorphisms, especially in organs where accurate TH control is essential, such the liver, muscles, and brain. Thyroid hormone trafficking is also significantly influenced by the Organic Anion-Transporting Polypeptides (OATPs), especially OATP1C1. T4 and rT3 absorption into the central nervous system is facilitated by OATP1C1, which is mostly expressed in the blood-brain barrier and encoded by the *SLCO1C1* gene. The effectiveness of thyroid hormone transport into the brain can be affected by variations in *SLCO1C1*, which may have an effect on neurological development and function. *SLCO1C1* polymorphisms have been investigated in connection with mood problems, cognitive deficits, and even the effectiveness of thyroid hormone replacement treatment in hypothyroid individuals.

The significance of OATP1C1 in preserving thyroid hormone homeostasis in the brain and its potential as a therapeutic target are highlighted by these findings. Another transporter related to thyroid hormone metabolism is the sodium-dependent bile acid transporter NTCP, which is encoded by the *SLC10A1* gene. Although its function in bile acid transport is its most well-known function, NTCP also helps T4 enter hepatocytes. Systemic thyroid hormone levels and hepatic thyroid hormone metabolism can be changed by genetic variations in *SLC10A1* that impact NTCP function. Variants of *SLC10A1* may be a factor in interindividual variations in thyroid hormone clearance rates and have been linked to change thyroid function test findings. Thyroid hormone transporter polymorphisms also affect medication interactions and therapeutic outcomes. To replicate the effects of genetic variants, several medicines, including statins, glucocorticoids, and antiepileptic drugs, can impede thyroid hormone transporters. Thyroid hormone levels or activity may change clinically significantly in people with certain transporter gene variations, making them more vulnerable to these drug-induced changes. Comprehending these variations might assist in customizing treatment plans, such as modifying doses of thyroid hormone replacement therapy or choosing substitute drugs to reduce side effects. Next-Generation

Correspondence to: Noa Lieke, Department of Epidemiology, Erasmus University Medical Center, Rotterdam, The Netherlands, E-mail: noa@lie.nl

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Sequencing (NGS) and Genome-Wide Association Studies (GWAS) are two examples of genomic technology advancements that have greatly improved our capacity to detect and describe variants in thyroid hormone transporter genes. These instruments have uncovered new variations linked to metabolic characteristics, illness risk, and thyroid function. To clarify the molecular effects of these variations on transporter activity and thyroid hormone homeostasis, functional investigations employing *in vitro* and *in vivo* models are essential.

CONCLUSION

Thyroid hormone homeostasis is modulated and the pathophysiology of thyroid-related illnesses is influenced by genetic variations in thyroid hormone transporters. Thyroid hormone transport efficiency, tissue distribution, and interactions with pharmacological and environmental variables can all be impacted by variations in transporter genes, including *SLC16A2*, *SLC16A10*, *SLCO1C1*, and *SLC10A1*.