Peripheral metabolism of thyroid hormones: a review.

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BACKGROUND: Peripheral metabolism of thyroid hormones is a critical component of the impact these hormones have on intracellular function. Thyroid hormones can be metabolized in peripheral tissue by deiodination, conjugation, deamination, and decarboxylation enzyme reactions. Therefore, alterations in these metabolic pathways might significantly impact the quantity of specific thyroid hormone metabolites influencing function at the cellular level.

DISCUSSION: Available evidence also suggests that, under some circumstances, the activity of hepatic antioxidant enzyme systems and lipid peroxidation might influence the peripheral metabolism of thyroid hormones. Several syndromes, such as "euthyroid sick syndrome" and "low T3 syndrome," have been classified within the medical literature. The common feature of these disorders is a low level of circulating T3, with generally normal to slightly elevated blood T4 levels and either normal or slightly suppressed TSH levels. This pattern of altered thyroid hormone levels is generally agreed to be a result of impairment in extra-thyroidal peripheral metabolism. Hepatic and renal pathology, as well as catabolic states such as those induced subsequent to severe injury, illness, or trauma result in consistent shifts in the thyroid hormone profile, secondary to their impact on peripheral enzyme pathways. Lifestyle factors, such as stress, caloric restriction, and exercise, influence peripheral metabolism of thyroid hormones. Exposure to toxic metals, chemical poisons, and several drugs can also influence the peripheral fate of thyroid hormones.

CONCLUSIONS: While the role of vitamins, minerals, and botanical extracts in thyroid hormone metabolism requires further elucidation, current evidence supports a role for selenium in the hepatic 5'-deiodination enzyme.

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