

Deiodinases and Developmental Hypothyroidism

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Thyroid hormones (THs) are crucial for normal organ development [1-30]. The levels of 3,5,3'-triiodothyronine (T3) in tissues is regulated by the action of the iodothyronine deiodinases (DI, DII, and DIII) [4,31-33]. In 2015b, I reported that any defects in fetal Ds may have more impact on fetal brain since they can result in intracellular T3 deficiency despite sufficient maternal TH supply. Also, my group [24] reported that hypothyroidism during the critical developmental period caused elevation in D2 activity in the diencephalon and mesencephalon, revealing of a compensatory response. This reflects that the compensatory action of Ds to changed TH level is not yet mature in young embryos or fetuses [32-34]. However, MMI treatment did not meaningfully change the activities of D1 or D3 in maternal liver and kidney. Thus, further studies are needed to elucidate the tissue-, cell-, and sex-specific expression of individual Ds during the development of both human and animals, in the adult, during aging and when sick.

Conflict of Interest

The author declares that no competing financial interests exist.

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