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Effects of experimentally-induced maternal hypothyroidism on crucial offspring rat brain enzyme activities.

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Abstract

Hypothyroidism is known to exert significant structural and functional changes to the developing central nervous system, and can lead to the establishment of serious mental retardation and neurological problems. The aim of the present study was to shed more light on the effects of gestational and/or lactational maternal exposure to propylthiouracil-induced experimental hypothyroidism on crucial brain enzyme activities of Wistar rat offspring, at two time-points of their lives: at birth (day-1) and at 21 days of age (end of lactation). Under all studied experimental conditions, offspring brain acetylcholinesterase (AChE) activity was found to be significantly decreased due to maternal hypothyroidism, in contrast to the two studied adenosinetriphosphatase (Na(+),K(+)-ATPase and Mg(2+)-ATPase) activities that were only found to be significantly altered right after birth (increased and decreased, respectively, following an exposure to gestational maternal hypothyroidism) and were restored to control levels by the end of lactation. As our findings regarding the pattern of effects that maternal hypothyroidism has on the above-mentioned crucial offspring brain enzyme activities are compared to those reported in the literature, several differences are revealed that could be attributed to both the mode of the experimental simulation approach followed as well as to the time-frames examined. These findings could provide the basis for a debate on the need of a more consistent experimental approach to hypothyroidism during neurodevelopment as well as for a further evaluation of the herein presented and discussed neurochemical (and, ultimately, neurodevelopmental) effects of experimentally-induced maternal hypothyroidism, in a brain region-specific manner.

KEYWORDS:

Acetylcholinesterase; Brain; Gestation; Hypothyroidism; Lactation; Mg(2+)-ATPase; Na(+),K(+)-ATPase; Propylthiouracil; Rat

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