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Impaired learning and memory performance in subclinical hypothyroidism rat model induced by hemi-thyroid electrocauterization.

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Abstract

It is well known that clinical hypothyroidism (CH) could induce cognitive deficits and the decision to start treatment for CH with thyroxine is usually easy. However, the relationship of cognition dysfunction with subclinical hypothyroidism (SCH) is inconsistent and whether need to treat SCH is controversial. In the present study, we induced SCH rat model by hemi-thyroid electrocauterization, then employed a serial of behavior tests including beam balance, open field, and Morris water maze (MWM) to investigate the behavior performance of SCH rats, and explored the protein expression of phosphorylated ERK1/2 in hippocampus by western blot. Our results elucidated that hemi-thyroid electrocauterized rats had elevated plasma thyrotropic-stimulating hormone (TSH) level with normal free thyroxine (fT4) and triiodothyronine (T3) concentrations, which defines SCH in humans. If diagnosed rat SCH according to the measures that both plasma TSH higher than 97.5 percentile of sham group and fT4 among 2.5 and 97.5 percentile of sham group, the success rate of SCH modeling was 66.6%. SCH decreased exploratory behavior but did not affect motor function in rats, with the negative correlation of exploratory behavior with plasma TSH concentration. Moreover, SCH rats displayed impairment of learning and memory ability in MWM task, with the longer escape latency in the acquisition phase and shorter duration in target quadrant in the test phase than that of sham rats, the mechanism of which might be related with the increased plasma TSH concentration, the decreased hippocampal T3 level, and the enhanced expression of phosphorylated ERK1/2 in hippocampus. Our results, together with the results from other studies, suggest that treatment is necessary for SCH.

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