FETAL/NEONATAL HYPO- AND HYPERTHYROIDISM AFFECTS LEYDIG AND SERTOLI CELL DEVELOPMENT

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Previously the effects of neonatal hypo- and hyperthyroidism have been investigated using rather unphysiological approaches like propyl-thiouracil (PTU) treatment or daily tri-iodothyronine (T_3) injections. These studies showed that T_3 advanced Leydig and Sertoli cell development, while PTU induced hypothyroidism delayed the development of these cell types. Recent studies have demonstrated that PTU influences Leydig cell function and possibly development directly, making it difficult to interpret former hypothyroid data.
In the present study mild forms of hyper- and hypothyroidism were induced already during fetal development. Dams were fed an iodine-free diet to which 0.5% perchlorate was added to deplete endogenous iodine stores, or a diet to which different doses (3.5, 5.0, 7.0 µg T_4/100g BW) of thyroxine (T_4) were added. After parturition, dams and offspring were kept on this diet until sacrifice. Pups were killed between days 7 and 50 after birth.
Both hyper- and hypothyroidism resulted in a decrease in body and testis weight of the pups. Plasma TSH levels were significantly reduced in the hyperthyroid groups from day 21 after birth onwards, and were elevated in the hypothyroid offspring. In contrast to the controls, tubular lumen formation was significantly delayed in the testes of both hyper- and hypothyroid pups. Leydig cell proliferation, as identified by BrdU and 3ß-HSD labelling, was slightly decreased in the hyperthyroid animals up to day 16, but significantly increased above control levels from day 21 onwards. Leydig cell proliferation was decreased in hypothyroid rats at all ages. The data suggest that mild forms of hyper- and hypothyroidism influence testis maturation.

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