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**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 triiodothyronine ($T_3$) injections. These studies showed that $T_3$ advanced Leydig and Sertoli cell development, while PTU induced hypothyroidism delayed the development of these cell types. 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 of thyroxine ($T_4$) were added. After parturition dams and offspring were kept on this diet until sacrifice. Pups were killed between day 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 for 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 hyperthyroid pups. Moreover, also the percentage of tubules that contained pachytene spermatocytes was reduced in these animals at the age of 21 days. Leydig cell proliferation, as identified by BrdU and 3ß-HSD labelling, was slightly lower in the hyperthyroid animals up to day 15, but significantly increased above control levels from day 21 onwards. The hypothyroid testis data are being analysed at present and will be presented at the workshop. The data suggest that mild forms of hyper- and hypothyroidism influence testis maturation.