Background: Childhood obesity is a global health problem and co-morbidities develop already during childhood and adolescence. Male obesity impacts negatively on the reproductive function. Testosterone is decreased, sperm quality reduced, and the physical and molecular structure of germ cells altered in obese males. However, less is known about the role of prepubertal obesity on future reproductive function.

Objective and Hypotheses: The aim of our study was to explore the influence of prepubertal obesity on reproductive potential and androgenic status in adult male rats.

Material and Methods: Lewis male rats were exposed to high fat diet (HFD) and standard chow (SC) from day 21 until 3 (group 1) and 9 months (group 2). Various anthropometric data including fat mass and adipocyte diameter were analyzed. Mating studies and semen analyses were performed. Sex steroids and gonadotropin levels were determined by immunoaassays. Testis morphology was evaluated by microscopy. Expression of Leydig-, Sertoli- and germ cell specific genes were analyzed at the transcriptional (q-PCR) level.

Intratesticular testosterone was measured and did not show significant differences in group 1 but was decreased by 31.5% (p=0.004) in group 2 (Figure 6).

We observed an upregulation of steroidalogenic enzymes in group 1, while 9-months treatment with HFD (group 2) down-regulated steroidalogenic enzymes (Figure 7, 8).

Conclusion: Long-term (9 months) obesity developed in the prepubertal period significantly suppressed Leydig cell capacity to produce testosterone and altered the T/E2 ratio in obese rats. Furthermore steroidalogenic enzymes were downregulated and intratesticular testosterone significantly decreased. In addition sperm motility was negatively affected in obese animals on HFD and litters were smaller and had a lower birth weight. If translated into human medicine, the observed perturbations of sex hormone levels may indicate a disturbed spermatogenesis and attenuated reproductive potential and fertility in obese males.

Karolinska Institutet Dr. Isabel Wagner Department of Women’s and Children’s Health Pediatric Endocrinology Unit

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