AB195. Role of manchette in elongated sperm development with the Hook 1 mutant of decapitated sperm in human

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Objective: Teratospermia seriously affect male infertility. The production of abnormal sperm is closely related to the process of the sperm spermiagenesis, but the molecular mechanism is uncovered.
Design: In our study, DNA sequencing was used to find the potential mutant on the genome and then the expression of the interested genes were identified by immuno-fluorescence. **Materials and methods:** Seven male with decapitated sperm and 100 normal were inclusion in this study. DNA isolated from the blood and the whole exons of the SPATA6 and Hook1, both of which are associated genes found in defect mouse, were sequenced in human.
Results: The results showed the presence of 1 SNP loci of totally 13 exons in SPATA6; while there were1 SNP loci and 1 mutant in Hook1 gene with 22 exons. Furthermore, the mutation in exon 10 of Hook1 has never been reported in other SNP databases and the protein structure prediction also showed the damage affection on the protein 3D-structural of HOOK1. Hook1 gene is connected to the microtubule protein, and it expressed mainly in the haploid sperm cells in testis. The locations of the expression were around the manchette structure.
Conclusions: Thus, the HOOK1 protein is closely related to the decapitated sperm during the production of the sperm. Our research offer new clues to the cause of decapitated sperm also provided the foundation to reveal the molecular mechanism of sperm agenesis.
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Keywords: Teratospermia; infertility; decapitated sperm; *SPATA6*; *HOOK1*

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AB196. Sulforaphane reduction of testicular apoptotic cell death in diabetic mice is associated with the up-regulation of Nrf2 expression and function

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Abstract: Diabetes-induced testicular cell death is predominantly due to oxidative stress. Nuclear factor (erythroid-derived 2)-like 2 (*Nrf2*) is an important transcription factor in controlling the anti-oxidative system and is inducible by sulforaphane (SFN). To test whether SFN prevents diabetes-induced testicular cell death, an insulin-defective stage of type 2 diabetes (IDS-T2DM) was induced in mice. This was accomplished by feeding them a high-fat diet (HFD) for 3 months to induce insulin resistance, and then giving one intraperitoneal injection of streptozotocin to induce hyperglycemia while age-matched control mice were fed a normal diet (ND). IDS-T2DMand ND-fed control mice were then further subdivided into those with or without 4-month SFN treatment. IDS-T2DM induced significant increases in testicular cell death presumably through receptor and mitochondrial pathways, shown by increased ratio of Bax/Bcl2 expression and cleavage of caspase-3 and caspase-8 without significant change of endoplasmic reticulum stress. Diabetes also significantly increased testicular oxidative damage and inflammation. All these diabetic effects were significantly prevented by SFN treatment with up-regulated

Nrf2 expression. These results suggest that IDS-T2DM induces testicular cell death presumably through caspase-8 activation and mitochondria-mediated cell death pathways, and also by significantly down-regulating testicular *Nrf2* expression and function. SFN up-regulates testicular *Nrf2* expression, and its target antioxidant expression, which was associated with significant protection of the testis from IDS-T2DM-induced germ cell death.

Keywords: High fat diet; male germ cells; *Nrf2*; sulforaphane; type diabetes

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AB197. The prevalence of FSH autoantibodies in the aging male

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Aim: This study was conducted to evaluate the prevalence of FSH autoantibodies in aging male and further observe the correlation between FSH autoantibodies and reproductive hormones.

Methods: The serum samples were collected from 192 normal men whose mean age was 49.47±18.51 (range, 18-88) years and the level of sera FSH, LH, T, SHBG and FSH antibody was detected by RIA and ELISA assay, respectively. Free testosterone index (FTI) was analyzed based on the serum level of total T and SHBG.

Results: The positive incidence of anti-sera against FSH in the group, aged 60-89 years, was significantly higher than that in the group, aged 18-59 years (20.00%, 14/70 *vs.* 9.84%, 12/122) (P<0.05). There was positive correlation between age and the concentration of serum FSH or LH (r=0.306, P=0.0001; r=0.246, P=0.002). Meanwhile, a negative correlation between age and the level of serum T or FTI was also found (r=–0.461, P=0.0001; r=–0.407, P=0.0001). The FSH autoantibodies in different age men do not have effect on the level of serum LH, T, SHBG and FTI. However, in the group aged 60-89 years, the level of serum FSH in the positive FSH autoantibodies samples was lower than that in the negative samples (P<0.05).

Conclusions: Aged men were associated with higher incidence of FSH autoantibodies. The level of serum FSH in aged man could be affected by FSH autoantibodies. Anti-sera against FSH in men might be involved in a certain physiologic process of male aging.

Keywords: FSH autoantibodies; FSH; male aging

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AB198. Nodal regulates the differentiation of iPS cells to male germ cells via Smad2/3, Oct4 and Foxh1 activation

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Objective: The differentiation of male germ cells from iPS cellsprovides an ideal model for unveiling molecular

