

that basal PKG does not play a role in the regulation of BK channel activity in UBSM cells. In conclusion, basal PKA plays an essential role in maintaining BK channel activity, and thereby controlling UBSM excitability and contractility.

Keywords: Detrusor; PKA; H-89; KT-5720; PKI 14-22

doi: 10.3978/j.issn.2223-4683.2014.s207

Cite this abstract as: Li N, Li Z, Wang P. Constitutive PKA activity is essential for maintaining the excitability and contractility in guinea pig urinary bladder smooth muscle: central role played by the BK channel. *Transl Androl Urol* 2014;3(S1):AB207. doi: 10.3978/j.issn.2223-4683.2014.s207

AB208. Is abnormal expression of semenogelin I involved with seminal vesiculitis?

Bianjiang Liu, Zhen Song, Aiming Xu, Shifeng Su, Zengjun Wang

State Key Laboratory of Reproductive Medicine and Department of Urology, The First Affiliated Hospital of Nanjing Medical University, Nanjing 210029, China

Abstract: Seminal vesiculitis is the common disease of male urogenital system. However, the pathogenesis of seminal vesiculitis remains unclear. Semenogelin I (Sg I) is mainly synthesized and secreted by seminal vesicle and has antibacterial activity. We thus postulate that Sg I plays an important role during the and development of seminal vesiculitis. In the present study, we analyzed the expression of Sg I in normal seminal vesicle tissues and seminal vesiculitis tissues through immunohistochemistry. The results showed down-regulated expression of cluster in at protein level in seminal vesiculitis tissues compared with normal seminal vesicle tissues. Our preliminary data suggest that the abnormal expression of cluster in is closely related to seminal vesiculitis. Down regulation of Sg I expression may weaken the antibacterial activity of the seminal vesicle and then induce the occurrence of disease. This is the first study to focus on the relationship between Sg I and human

seminal vesiculitis.

Keywords: Seminal vesiculitis; semenogelin I (Sg I); abnormal expression

doi: 10.3978/j.issn.2223-4683.2014.s208

Cite this abstract as: Liu B, Song Z, Xu A, Su S, Wang Z. Is abnormal expression of semenogelin I involved with seminal vesiculitis? *Transl Androl Urol* 2014;3(S1):AB208. doi: 10.3978/j.issn.2223-4683.2014.s208

AB209. Co-incubation of human spermatozoa with anti-VDAC antibody reduced sperm motility

Bianjiang Liu, Min Tang, Zhijian Han, Jie Li, Jiexiu Zhang, Pei Lu, Ninghong Song, Zengjun Wang

State Key Laboratory of Reproductive Medicine and Department of Urology, The First Affiliated Hospital of Nanjing Medical University, Nanjing 210000, China

Background: Voltage-dependent anion channel (VDAC), a channel protein, exists in the outer mitochondrial membrane of somatic cells and is involved in multiple physiological and pathophysiological processes. Up until now, little has been known about VDAC in male germ cells. In the present study, the relationship between VDAC and human sperm motility was explored.

Methods: Highly motile human spermatozoa were incubated in vitro with anti-VDAC antibody. Total sperm motility, straight line velocity (VSL), curvilinear velocity (VCL), and average path velocity (VAP) were recorded. Intracellular free calcium concentration $[(Ca)_{i}]$, pH value (pHi), and ATP content were determined.

Results: Co-incubation with anti-VDAC antibody reduced VSL, VCL, and VAP of spermatozoa. Co-incubation further reduced $[(Ca)_{i}]$. Anti-VDAC antibody did not significantly alter total sperm motility, pHi and intracellular ATP content.

Conclusions: The data suggest that co-incubation with anti-VDAC antibody reduces sperm motility through inhibition of Ca transmembrane flow. In this way, VDAC participates in the modulation of human sperm motility through mediating Ca transmembrane transport.

Keywords: Voltage-dependent anion channel (VDAC); human; sperm motility; calcium; pH; ATP

doi: 10.3978/j.issn.2223-4683.2014.s209

Cite this abstract as: Liu B, Tang M, Han Z, Li J, Zhang J, Lu P, Song N, Wang Z. Co-incubation of human spermatozoa with anti-VDAC antibody reduced sperm motility. *Transl Androl Urol* 2014;3(S1):AB209. doi: 10.3978/j.issn.2223-4683.2014.s209

AB210. The cytochrome P4501A1 gene polymorphisms and idiopathic male infertility risk: a meta-analysis

Jianzheng Fang, Shangqian Wang, Hainan Wang, Shengli Zhang, Shifeng Su, Zhen Song, Yunfei Deng, Jian Qian, Jinbao Gu, Bianjiang Liu, Jingyi Cao, Zengjun Wang

State Key Laboratory of Reproductive Medicine, Department of Urology, First Affiliated Hospital of Nanjing Medical University, Nanjing 210029, China; Department of Urology, Xuzhou Third People's Hospital, Xuzhou 221000, China

Abstract: Meta-analysis Polymorphism Studies of the relationship between male infertility and CYP1A1 polymorphisms are inconclusive. To drive a more precise estimation, we performed a meta-analysis based on 1,060 cases and 1,225 controls from 7 published case-control studies. PubMed and CNKI literature search were conducted to identify all eligible studies investigating such a relationship. Crude odds ratios (ORs) with 95% confidence intervals (CIs) were used to assess the strength of association in the additive model, dominant model, recessive model, and allele-frequency genetic model. In the

overall analysis, the frequency of CYP1A1*2A genotype was significantly associated with susceptibility to idiopathic male infertility. Further stratified analysis by ethnicity showed notable association between the polymorphism and risk of idiopathic male infertility in Asians. In conclusion, these results support that the CYP1A1*2A genotype polymorphism mainly contributes to idiopathic male infertility susceptibility in Asians but not in Caucasians.

Keywords: CYP1A1; male infertility; meta-analysis

doi: 10.3978/j.issn.2223-4683.2014.s210

Cite this abstract as: Fang J, Wang S, Wang H, Zhang S, Su S, Song Z, Deng Y, Qian J, Gu J, Liu B, Cao J, Wang Z. The cytochrome P4501A1 gene polymorphisms and idiopathic male infertility risk: a meta-analysis. *Transl Androl Urol* 2014;3(S1):AB210. doi: 10.3978/j.issn.2223-4683.2014.s210

AB211. Association of the glutathione S-transferase M1, T1 polymorphisms with cancer: evidence from a meta-analysis

Jianzheng Fang, Shangqian Wang, Shengli Zhang, Shifeng Su, Zhen Song, Yunfei Deng, Hongqing Cui, Hainan Wang, Yi Zhang, Jian Qian, Jinbao Gu, Bianjiang Liu, Pengchao Li, Rui Zhang, Xinnong Liu, Zengjun Wang

State Key Laboratory of Reproductive Medicine, Department of Urology, Department of Neurosurgery, The First Affiliated Hospital of Nanjing Medical University, Nanjing 210000, China; Department of General Surgery, Qilu Hospital, Shandong University, Jinan 250000, China

Background: Glutathione S-transferases (GSTs) are a family of multifunctional enzymes that are involved in the metabolism of many xenobiotics, including a wide range of environmental carcinogens. While the null genotypes in GSTM1 and GSTT1 have been implicated in tumor genesis, it remains inconsistent and inconclusive. Herein, we aimed to assess the possible associations of the GSTM1