

AB206. Urinary nerve growth factor levels could be a biomarker for overactive bladder symptom: a meta-analysis

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Abstract: To examine whether urinary tract nerve growth factor (uNGF) could be a biomarker for overactive bladder (OAB) symptom, we conducted a comprehensive meta-analysis of 8 case-control studies. In all the studies considered, patients with OAB symptom had a higher uNGF level compared to healthy people. In addition, patients had a significantly lower uNGF level after successful treatment. In the subgroup analysis, we found that patients with OAB-wet symptom had a higher uNGF level than patients with OAB-dry symptom. However, no significant difference was found between patients with OAB symptom and patients with interstitial cystitis/painful bladder syndrome (IC/PBS) symptom in uNGF/Cr levels. In conclusion, uNGF level could be a useful biomarker for the diagnosis of OAB, a possible biomarker for differentiation between OAB subtypes (wet or dry), and a predictive biomarker for a specific treatment, but it cannot be used as the urinary biomarker for the differential diagnosis of IC/PBS and OAB.

Keywords: Urinary nerve growth factor (uNGF); overactive bladder (OAB); met

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AB207. 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

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Abstract: The large conductance voltage- and Ca-activated K(BK) channel is a critical regulator of urinary bladder smooth muscle (UBSM) excitability and contractility in many species. BK channel activation can hyperpolarize the cell resting membrane potential (RMP), decrease the intracellular Ca level, and attenuate UBSM cell excitability, therefore cause UBSM relaxation. Elevation of cellular cAMP levels by β -adrenergic receptor activation or phosphodiesterase (PDE) inhibition suppresses guinea pig UBSM excitability and contractility via BK channel activation. The current study aimed at determining the mechanism by which basal PKA, without elevation of cellular cAMP levels, controls UBSM excitability, and contractility via BK channel activation. UBSM strips (—2–4 mm wide and —5–8 mm long) were harvested from guinea pig bladder following removing urothelium. We used perforated patch-clamp and line-scanning confocal techniques on freshly-isolated guinea pig UBSM cells, and isometric tension recordings of UBSM isolated strips. Our data show that PKA inhibitors, H-89, PKI 14-22, or KT-5720: (I) reduced Ca spark frequency, eliminated spontaneous transient BK channel currents, abolished the transient hyperpolarizations, and depolarized the cell RMP; (II) increased the spontaneous phasic contraction amplitude, force, duration, and decreased the phasic contraction frequency; (III) in the presence of PKA inhibitor, H-89, PDE1 inhibition with 8MM-IBMX did not affect BK channel activity or DSM contractility. Using a selective PKG inhibitor, DT-2, we provided direct evidence at cellular level

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

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

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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

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

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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.