AB087. Preserved erectile function in the hyperhomocysteinaemia transgenic rats harboring human tissue kallikrein

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Background: To investigate the role of human tissue kallikrein 1 (hKLK1) gene on the erectile dysfunction (ED) of induced by hyperhomocysteinaemia (HHcy) in rats.

Methods: The HHcy rat model was formed by a methionine (Met)-rich diet in SD rats. Here, 32 rats, 10-week-age, were divided in four groups: control (n=8), low-dose (4% Met; n=8), high-dose (7% Met; n=8) and transgenic rats (TGR +7% Met; n=8). Thirty days later, erectile function and related targets were tested.

Results: ED, impaired endothelial and smooth muscle function, and pathological changes (a higher apoptosis level and a lower autophagy level) were showed in the 4% Met and 7% Met groups compared with the control, while were all markedly diminished by the *bKLK1* gene in the TGR +7% Met group.

Conclusions: These data suggested that hKLK1 might play an inhibition role on HHcy-induced ED in rats by protection of endothelial function and inhibition of oxidative stress and corporal fibrosis.

Keywords: Erectile dysfunction (ED); gene therapy; metabolism; endothelial function

doi: 10.21037/tau.2017.s087

Cite this abstract as: Cui K, Tang Z, Luan Y, Rao K, Wang T, Wang S, Chen Z, Liu J. Preserved erectile function in the hyperhomocysteinaemia transgenic rats harboring human tissue kallikrein. Transl Androl Urol 2017;6(Suppl 3):AB087. doi: 10.21037/tau.2017.s087

AB088, FTY720

supplementation partially improves erectile dysfunction in rats with streptozotocininduced type 1 diabetes through inhibition of endothelial dysfunction and corporal fibrosis

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Background: To investigate whether FTY720, approved in 2010 for the treatment of patients with the relapsing-remitting form of multiple sclerosis, could ameliorate erectile dysfunction induced by diabetes mellitus (DMED). **Methods:** Thirty-two Sprague-Dawley rats (8 weeks old)

Methods: Thirty-two Sprague-Dawley rats (8 weeks old) were induced type I DM and the other eight rats formed the control (n=8). Eight weeks later, 17 rats with DMED tested with an apomorphine test were divided in two groups: DMED (n=8) and DMED + FTY720 (1 mg/kg/d; n=9). Treatment of FTY720 lasted for 4 weeks.

Results: Impaired erectile function, inhibited S1P3/Akt/NO/cGMP activity, serious corporal fibrosis and overactivated pathways (the Smad and non-Smad) were found in the DMED group compared with the control, while FTY720 partly but significantly improved these pathological changes induced by DM.

Conclusions: FTY720 supplementation inhibited endothelial dysfunction and corporal fibrosis, ultimately leading to partial improvement of DMED in rats. This finding provides evidence for a potential treatment method for DMED.

Keywords: Erectile dysfunction; diabetes mellitus; endothelial function; corporal fibrosis

doi: 10.21037/tau.2017.s088

Cite this abstract as: Cui K, Ruan Y, Tang Z, Rao K, Wang T, Wang S, Chen Z, Liu J. FTY720 supplementation partially improves erectile dysfunction in rats with streptozotocin-induced type 1 diabetes through inhibition of endothelial dysfunction and corporal fibrosis. Transl Androl Urol 2017;6(Suppl 3):AB088. doi: 10.21037/tau.2017.s088

Keywords: Adenosine; erectile function; aging

doi: 10.21037/tau.2017.s089

Cite this abstract as: Yang X, Yuan J. Impaired adenosine signaling influences erectile function in aging rats. Transl Androl Urol 2017;6(Suppl 3):AB089. doi: 10.21037/tau.2017.s089

AB089. Impaired adenosine signaling influences erectile function in aging rats

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Background: As one of the most common disorders in old adult, erectile dysfunction (ED) remains attracting andrological physicians' attention. The aim of this study is to investigate the alterations of adenosine signaling in the penis of aging rats, and the influence to erectile function.

Methods: According to apomorphine test, the aging rats (18 months) with ED were selected as age-related erectile dysfunction (A-ED) group, and the young rats (2 months) were selected as normal control (NC) group. The intracavernosal pressure (ICP) measurements were conducted to evaluate the penile erectile function. Quantitative real-time polymerase chain reaction (RT-PCR) and Western Blot were used to detect the expression levels of genes and protein related to adenosine signaling in penis. Results: Compared to NC group, the outcomes of ICP showed a decreasing trend in A-ED group. Expression of adenosine A2B receptor, adenosine deaminase (ADA), and phosphodiesterase type (5PDE5) were increased in A-ED group, and AMP deaminase type 1 (AMPD1) and 2 (AMPD2) were decreased in A-ED group. The results of Western Blot also showed an increasing trend of A2B receptor in A-ED group.

Conclusions: Rats with erectile dysfunction showed an impaired adenosine signaling, our study may provide a new sight for further study to improve the erectile function of A-ED patients.

AB090. Tobacco smoking and erection dysfunction: a systematic review

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Background: The aim of this review was to investigate the correlation between smoking and erectile dysfunction (ED). **Methods:** ISI Web of Science. PubMed and Google Scholar databases (until June 2017) were searched for relevant publications on the correlation between smoking and erectile dysfunction.

Results: A total of 163 studies were reviewed. Tobacco smoke, an aerosol produced by the incomplete combustion of tobacco, is proved to be harmful to several organs. A wealth of researches showed tobacco smoking is a high-risk factor for ED. Multiple human studies and animal researches analyzed the correlation and possible mechanism between smoking/nicotine and ED.

Conclusions: Almost all the researches showed the clear evidence that tobacco smoking is indeed quite harmful to erectile function. Dose-response relation also confirmed that long term or high quantity of nicotine intake may lead to higher incidence of ED. Smoking may impact on penile vascular endothelial cells and the release of acetylcholine in cerebral cortex. Multiple signal pathways are involved in the smoking-induced ED. Researches also revealed that smoking cessation could, to a certain extent, improve erectile function.

Keywords: Tobacco smoking; erectile dysfunction (ED); mechanism

doi: 10.21037/tau.2017.s090