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 *hKLK1* 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

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AB088. FTY720 supplementation partially improves erectile dysfunction in rats with streptozotocin-induced 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) 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 over-activated 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

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