AB156. Taurine ameliorates erectile function in streptozocin-induced type 1 diabetic rats via multiple signaling pathways

Mingchao Li, Yajun Ruan, Tao Wang, Jun Yang, Ke Rao, Shaogang Wang, Weimin Yang, Jihong Liu

Tongji Hospital, Wuhan 430030, China

Objective: PDE5 inhibitors represent the first line therapy for treatment of ED. However, diabetic patients have poorer response compared with normal patients. The aim of this study was to determine whether taurine, a sulfur-containing amino acid, affects diabetic erectile dysfunction.
Methods: Type 1 diabetes mellitus was induced in male rats by streptozotocin (60 mg/kg, intraperitoneally). After 12 weeks, apomorphine test was conducted to confirm diabetic erectile dysfunction (DED). Only DED rats were administered taurine (400 mg/kg/day, intraperitoneally) or vehicle, for 4 weeks. Age-matched control, nondiabetic, rats were treated with saline intraperitoneally for 4 weeks. At week 16, after a 2-day washout, erectile function was evaluated. Peniles were harvested for Western blot analysis of RhoA, ROCK-1, ROCK-2, eNOS, nNOS, NADPH oxidase subunit gp91phox.
Results: Erectile function was significantly destroyed in diabetic rats compared with nondiabetic rats and was ameliorated in diabetic rats treated with taurine. In diabetic rats, RhoA, ROCK-1, ROCK-2 and gp91phox protein expressions were increased, whereas eNOS and nNOS expressions were decreased compared with nondiabetic rats, and both were reversed in diabetic rats treated with taurine.
Conclusions: Taurine improves erectile function in diabetic rats probably by inhibiting RhoA/Rock and activating NOS/NO signaling pathways. This may provide a potential new therapy for diabetic ED.
Keywords: Taurine; diabetes; erectile dysfunction; RhoA/Rock

doi: 10.3978/j.issn.2223-4683.2015.s156

Cite this abstract as: Li M, Ruan Y, Wang T, Yang J, Rao K, Wang S, Yang W, Liu J. Taurine ameliorates erectile function in streptozocin-induced type 1 diabetic rats via multiple signaling pathways. Transl Androl Urol 2015;4(S1):AB156. doi: 10.3978/j.issn.2223-4683.2015.s156

AB157. Treatment of diabetic erectile dysfunction by using endothelial progenitor cells genetically modified by human telomerase reverse transcriptase

Yan Zhang, Tao Wang, Jun Yang, Rui Li, Zhi Chen, Shaogang Wang, Jihong Liu, Zhangqun Ye

Tongji Hospital, Wuhan 430030, China

Objective: Erectile dysfunction (ED) is one of the most common complications of diabetes mellitus (DM). Under the background that effectivity of treatments for diabetic ED are quite poor, stem cell therapy is emerging a useful method. The aim of this study is to evaluate the possibility and mechanism of treatment of diabetic ED with endothelial progenitor cells (EPCs) genetically modified by human telomerase reverse transcriptase (hTERT).
Methods: Rat EPCs were isolated and transfected with hTERT (EPCs-hTERT). The paracrine characters and resistance to oxidative stress of EPCs-hTERT was determined *in vitro*. Thirty SD male rats were divided into five groups: diabetic ED rats, diabetic ED rats treated with EPCs, diabetic ED rats treated with EPCs-control, diabetic ED rats treated with EPCs-hTERT, normal control rats. Diabetes was induced by intraperitoneal injection of streptozotocin. After 8 weeks, diabetic ED rats were selected by apomorphine (APO) test. After injection of EPCs for 2 weeks, intracavernosal pressure (ICP) was

measured by means of electrical stimulation for each group. Concentration of growth factors, the amount of EPCs and endothelium/smooth muscle content were evaluated *in vivo*.

Results: EPCs-hTERT showed stable expression of hTERT at the level of, mRNA and protein for more than 40 passages. EPCs-hTERT could paracrine more VEGF, HGF and bFGF. EPCs' ability of resistance to oxidative stress was dramatically improved by tranfection of hTERT. ICP/mean arterial pressure (MAP) ratio induced by electrical stimulation in diabetic ED treated with EPCs-hTERT was markably higher than diabetic ED rats treated with EPCs or EPCs-control. Immunofluorescence demonstrated that more cells survived in penile tissues after implantation of EPC-hTERT. More growth factors were detected, and the endothelium/smooth muscle content was increase in penile tissues.

Conclusions: The paracrine effect and resistance to oxidative stress of EPCs-hTERT contributed to the improvement of erection of diabetic ED rats.

Keywords: Diabetes mellitus (DM); erectile dysfunction (ED); human telomerase reverse transcriptase (hTERT); endothelial progenitor cells (EPCs)

doi: 10.3978/j.issn.2223-4683.2015.s157

Cite this abstract as: Zhang Y, Wang T, Yang J, Li R, Chen Z, Wang S, Liu J, Ye Z. Treatment of diabetic erectile dysfunction by using endothelial progenitor cells genetically modified by human telomerase reverse transcriptase. Transl Androl Urol 2015;4(S1):AB157. doi: 10.3978/j.issn.2223-4683.2015.s157

AB158. A total of 213 cases with male isolated gonadotropin-releasing hormone deficiency: clinical feature analysis and gene mutation study in some cases

Jihong Liu, Hao Xu, Yonghua Niu

Tongji Hospital, Wuhan 430030, China

Objective: Isolated gonadotropin-releasing hormone (GnRH) deficiency (IGD) is a rare clinical condition with a broad spectrum of human reproductive disorders which varies from congenital isolated hypogonadotropic hypogonadism (IHH) to constitutional delay of puberty (CDP) and adult-onset isolated hypogonadotropic hypogonadism (AHH) in male. In the presence of anosmia or hyposmia, IHH is defined as Kallmann syndrome (KS), whereas in the presence of a normal sense of smell, it is termed normosmic IHH (nIHH). In this study, we described the clinical features of 213 Chinese IGD males, and performed genetic study in some of these cases, and analyzed their genotype phenotype correlations.

Methods: We collected the male isolated GnRH deficiency patients in our outpatient and filed their medical and family history. The physical examinations, laboratory and imaging studies were performed. The PCR and agarose gel electrophoresis, whole genome chip and semiconductor target areas sequencing were used to reveal the molecular defects of two KS and ichthyosis brothers. We also used semiconductor target areas sequencing and Sanger sequencing to detect the mutations in 15 IHH causative genes in 4 KS patients with cleft lip/palate (CLP) and 8 IGD patients (including 5 KS patients, one nIHH patients and two AIHH patients) without CLP. Functional prediction and conservation analysis were performed to predict the consequence of the three mutations.

Results: The reproductive phenotypes in the IHH patients included Gynecomastia (23/210), microphallus (42/210), cryptorchidism (21/210), unilateral testicular agenesis (2/210), scrotum dysplasia (3/210). The non-reproductive phenotypes in our patients contained ichthyosis (3/210), unilateral renal agenesis (2/210), cleft lip (2/210), cleft lip and dental agenesis (1/210), cleft lip and palate combined with dental agenesis and high arched palate (1/210), nystagmus and iris hypoplasia (1/210). These non-reproductive phenotypes only presented in KS patients. The results of karyotype analysis showed that 15 out of the 210 IHH patients (7.1%) had polymorphic chromosomal variants.. The phenotypes of the two Chinese brothers with KS and X-linked ichthyosis were characterised by bilateral cryptorchidism, unilateral renal agenesis in one patient but normal kidney development in another. Genetic study showed that the two affected siblings had the same novel deletion at Xp22.3 including exons 9-14 of *KAL1* gene and entire STS gene. We also identified two novel heterozygous missense mutations in FGFR1, (NM_001174066): c.776G>A (p.G259E) and (NM_001174066): c.358C>T (p.R120C), in a 23-yr-old KS male with cleft lip and an

