VOLUME 8 ◆ SUPPLEMENT 5 ◆ DECEMBER 2011

PROCEEDINGS FROM THE 14TH ANNUAL CONGRESS OF
THE EUROPEAN SOCIETY FOR SEXUAL MEDICINE
MILAN, ITALY DECEMBER 1–4 2011

BASIC SCIENCE & CLINICAL RESEARCH IN MALE & FEMALE SEXUAL FUNCTION & DYSFUNCTION

THE JOURNAL OF

Sexual Medicine

BASIC SCIENCE & CLINICAL RESEARCH IN MALE & FEMALE SEXUAL FUNCTION & DYSFUNCTION

Official Journal of
The International Society for Sexual Medicine

Asia Pacific Society for Sexual Medicine (APSSM)
European Society for Sexual Medicine (ESSM)
Latin American Society for Sexual Medicine (SLAMS)
Sexual Medicine Society of North America (SMSNA)
International Society for the Study of Women's Sexual Health (ISSWSH)

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with Control group. However, SM/collagen ratios in L-citrulline group were increased compared with Ligation group. NOx levels of Ligation group were significantly lower than that of Control group ($P < 0.05$), and that of L-citrulline group were significantly higher than that of Ligation group ($P < 0.01$).
Conclusion: Max ICP/MAP ratios were restored by oral L-citrulline supplementation. Thus, we suggest that oral L-citrulline supplementation in early ischemic period may be a new novel therapy for acute arteriogenic ED.
Policy of full disclosure: None.

PS-02-007

PROTEIN STRUCTURAL ALTERATIONS IN DIABETIC CAVERNOSAL TISSUE—THEIR ROLE IN ERECTILE DYSFUNCTION PROGRESSION

Castela, A.[1]; Soares, R.[2]; Gomes, P.[2]; Coelho, P.[3]; Fernandes, R.[4]; Costa, R.[2]; Vendeira, P.[1]; Costa, C.[2]
[1]University of Porto, Institute for Molecular and Cell, Portugal; [2]University of Porto, Department of Biochemistry (U38-FCT), Portugal; [3]Instituto Politécnico do Porto, Ciências Químicas e das, Vila Nova de Gaia, Portugal; [4]Instituto Politécnico do Porto, Ciências Químicas e das, Portugal

Objective: Erectile dysfunction (ED) is one of the most prevalent complications in diabetic men. Hyperglicemia contributes to increased oxidative stress (OS) in diabetic corpus cavernosum (CC), promoting alterations in cavernosal cellular components. However, it remains unclear the mechanisms by which OS induces modifications in diabetic penile tissue with the progression of diabetes and its role in the development of ED. We intended to evaluate/quantify CC protein structural modification caused by OS in an early and late stage of diabetes.
Methods: Male Wistar rats were divided into groups (N = 5/group): 2- and 8-week-streptozotocin-induced type 1 diabetes and age-matched controls. Systemic OS was evaluated in blood samples by chromatographic detection of oxidized glutathione (GSSG)/reduced glutathione (GSH). Penile OS-induced protein damage was assessed by oxidative structural changes detected by western blotting of 3-nitrotyrosine (3-NT) and protein carbonylation.
Results: Our results revealed a significant increase in blood GSSG/GSH ratio at 8 weeks of diabetes (diabetic rats 1.619 ± 0.216 vs. controls 0.779 ± 0.238, $P < 0.05$), suggesting a systemic OS increment. Consistently, a significant augmentation in protein nitration (diabetic group: 3.398 ± 0.332 vs. controls 2.284 ± 0.092; $P < 0.05$) and carbonylation (diabetic animals: 12.620 ± 0.224 vs. controls 11.240 ± 0.398; $P < 0.05$) was observed only in 8-week diabetic CC, indicating more severe protein oxidative modifications at a late stage of the disease.
Conclusion: We demonstrated that systemic and penile OS effects are detected mainly in established diabetes. OS-induced penile protein modifications seem to occur only in the advanced stage of the disease and may be responsible for promoting structural/functional deregulations in cellular/molecular mechanisms essential for normal erectile process, contributing to the development and progression of diabetic-associated ED.
Policy of full disclosure: None.

PS-02-008

EXPRESSION AND DISTRIBUTION OF THE TRANSIENT RECEPTOR POTENTIAL ION CHANNEL A1 (TRPA1) IN HUMAN PENILE ERECTILE TISSUE

Ückert, S.[1]; Waldkirch, E.[1]; Sonnenberg, J.[2]; Boeck, N.[2]; Kuczyk, M.[1]; Hedlund, P.[3]
[1]Hannover Medical School, Department of Urology, Germany; [2]IBFA, Sexual Function Research Unit, Barsinghausen, Germany; [3]University Vita Salute, Department of Urology, Milano, Italy

Objective: The transient receptor potential ion channel A1 (TRPA1) has been suggested to be involved in mechano-afferent/efferent signaling in the bladder, prostate, and urethra. Up until today, no study has addressed the expression of this receptor in male genital and reproductive tissues. Thus, it was the aim of the present study to evaluate in human penile erectile tissue by means of molecular biology and immunohistochemistry the expression and localization of TRPA/TRPA1.
Methods: Human penile erectile tissue (corpus cavernosum penis) was obtained from five subjects who had undergone gender reassignment surgery. The expression of messenger ribonucleic acid (mRNA) encoding sequences specific for the TRPA receptor protein (hTRPA01FWD [985-1003], hTRPA02REV [1641–1623]) was elucidated by means of reverse transcriptase polymerase chain reaction (RT-PCR). Using immunohistochemical methods (double-labeling technique, laser fluorescence microscopy), the distribution of TRPA1 in relation to neuronal nitric oxide synthase (nNOS), the neuropeptide vasoactive intestinal polypeptide (VIP), and vesicular acetylcholine transporter protein (VAChT) was examined.
Results: RT-PCR revealed a faint but distinct signal related to the expected molecular size of 656 bp. Immunoreactivity for TRPA1 was registered in nerves transversing the cavernous sinusoidal space. These nerves also displayed the expression of VAChT. Signals specific for TRPA1 were also observed in meshworks of nerve fibers running alongside the walls of cavernous arteries. Varicose nerves containing nNOS or VIP were not immunoreactive for TRPA1. Cavernous vascular and non-vascular smooth muscle did not present immunosignals related to TRPA1.
Conclusion: The distribution of TRPA1 and VAChT receptors in penile erectile tissue suggests a role for TRPA1 in the mechanism of cholinergic signaling in the human penis.
Policy of full disclosure: None.

PS-02-009

NEURO-INFLAMMATION FOLLOWING CAVERNOUS NERVE INJURY IS ACCOMPANIED BY SIGNIFICANT CHEMOKINE-GENE UPREGULATION IN VIVO AND IN VITRO

Albersen, M.[1]; Berkers, J.[1]; Dekoninck, P.[2]; Deprest, J.[2]; De Ridder, D.[1]; Van der Aa, F.[1]
[1]University Hospitals Leuven, Experimental Urology, Belgium; [2]University Hospitals Leuven, Experimental Gynecology, Belgium

Objective: We have recently demonstrated the essential role of adipose tissue-derived stem cell (ADSC) recruitment toward the major pelvic ganglion (MPG) in rats following cavernous nerve injury (CNI). The interaction between chemokines and their receptors plays a major role in this process. The objectives of this study were to examine chemokine subtype expression in neuro-inflammation of the rat MPG following cavernous nerve injury (CNI), and to evaluate the usefulness of tumor necrosis factor alpha (TNFA)-stimulated rat Schwann cells as an in vitro model for neuronal chemokine production.
Methods: Six male 12-week-old Sprague Dawley rats underwent laparotomy and bilateral crush injury of the cavernous nerves. Six rats served as sham controls (laparotomy and periprostatic dissection only). Twenty-four hours after CNI, the MPGs were harvested, RNA was isolated and subjected to qPCR analysis in triplicate. A rat peripheral nerve schwannoma derived Schwann cell line was acquired (RT4-D6P2T) and cultured in the presence of TNFA in dosages of 0, 1, 10, and 100 nM for 24 hours in triplicate. Cells were then harvested and RNA was isolated and subjected to qPCR analysis.
Results: Twenty-four hours following CNI, neuro-inflammation was present in the rat MPG as illustrated by significant upregulation of TNFA and transforming growth factor beta (TGFB) 1 and 2. Crush injury further resulted in significant upregulation of the chemokines CCL2-22-28, CXCL12, CX3CL1, and XCL1. The in vitro stimulation of Schwann cells with TNFA mimicked this neuro-inflammatory condition at 24 hours as illustrated by a similar TGFB and chemokine RNA expression profile.
Conclusion: CNI-related neuro-inflammation in vivo and in vitro is accompanied by the expression of various chemokines. These chemokines may be responsible for the recruitment of ADSC toward the