# PDGF-mediated PI3K/AKT/β-catenin signaling regulates gap junctions in corpus cavernosum smooth muscle cells

Xiang Zhang, Fan Zhao, Jian-Feng Zhao, Hui-Ying Fu, Xiao-Jun Huang, Bo-Dong Lv


www.elsevier.com/locate/yexcr

PII: S0014-4827(17)30627-4
DOI: http://dx.doi.org/10.1016/j.yexcr.2017.11.025
Reference: YEXCR10825

To appear in: *Experimental Cell Research*

Received date: 4 September 2017
Revised date: 17 November 2017
Accepted date: 18 November 2017

Cite this article as: Xiang Zhang, Fan Zhao, Jian-Feng Zhao, Hui-Ying Fu, Xiao Jun Huang and Bo-Dong Lv, PDGF-mediated PI3K/AKT/β-catenin signalin regulates gap junctions in corpus cavernosum smooth muscle cells, *Experimenta Cell Research,* http://dx.doi.org/10.1016/j.yexcr.2017.11.025

# PDGF-mediated PI3K/AKT/β-catenin signaling regulates gap junctions in corpus cavernosum smooth muscle cells

Xiang Zhang[a1], Fan Zhao[a1], Jian-Feng Zhao[b], Hui-Ying Fu[a,c], Xiao-Jun Huang[b], Bo-Dong Lv[b,c]*

[a]The Second Clinical Medical College, Zhejiang Chinese Medical University, Hangzhou, China

[b]Department of Urology, The Second Affiliated Hospital of Zhejiang Chinese Medical University, Hangzhou, China

[c]Andrology Laboratory on Integration of Chinese and Western Medicine, Zhejiang Provincial Key Laboratory of Traditional Chinese Medicine, Hangzhou, China

*Corresponding author. bodonglv0571@163.com

**Abstract**

Erectile dysfunction (ED) is the most common sexual disorder that men report to healthcare providers. Gap junctions (GJs) are thought to be responsible for synchronous shrinkage of corpus cavernosum smooth muscle cells (CCSMCs), and play thus an important role in the maintenance of an erection. Hypoxia has been suggested as a pathological mechanism underlying ED. Here we demonstrate that hypoxia increased the expression of platelet-derived growth factor (PDGF) and the main GJ component connexin (Cx)43 in CCSMCs. Inhibiting PDGF receptor (PDGFR) activity decreased Cx43 expression. Treatment with different concentrations of PDGF increased the levels of phosphorylated protein kinase B

[1] These authors contributed equally to this work