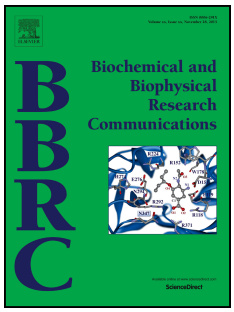
Accepted Manuscript



Neuroprotectant androst-3β, 5α, 6β-triol suppresses TNF-α-induced endothelial adhesion molecules expression and neutrophil adhesion to endothelial cells by attenuation of CYLD-NF-κB pathway

Min Yan, Tiandong Leng, Lipeng Tang, Xiaoke Zheng, Bingzheng Lu, Yuan Li, Longxiang Sheng, Suizhen Lin, Haitao Shi, Guangmei Yan, Wei Yin

PII: S0006-291X(17)30039-6

DOI: 10.1016/j.bbrc.2017.01.030

Reference: YBBRC 37092

To appear in: *Biochemical and Biophysical Research Communications*

Received Date: 28 December 2016

Accepted Date: 8 January 2017

Please cite this article as: M. Yan, T. Leng, L. Tang, X. Zheng, B. Lu, Y. Li, L. Sheng, S. Lin, H. Shi, G. Yan, W. Yin, Neuroprotectant androst-3β, 5α, 6β-triol suppresses TNF-α-induced endothelial adhesion molecules expression and neutrophil adhesion to endothelial cells by attenuation of CYLD-NF-κB pathway, *Biochemical and Biophysical Research Communications* (2017), doi: 10.1016/j.bbrc.2017.01.030.

Neuroprotectant androst-3β, 5*α*, 6β-triol suppresses TNF-α-induced endothelial adhesion molecules expression and neutrophil adhesion to endothelial cells by attenuation of CYLD-NF-κB pathway

Min Yan[a, 1], Tiandong Leng[b, 1], Lipeng Tang[c, 1], Xiaoke Zheng[a], Bingzheng Lu[d], Yuan Li[d], Longxiang Sheng[d], Suizhen Lin[e], Haitao Shi[e], Guangmei Yan[d], Wei Yin[f *]

[a]Department of Pathology, The First Affiliated Hospital, Sun Yat-Sen University, Guangzhou, GD 510080, P. R. China

[b]Neuroscience Institute, Morehouse School of Medicine, Atlanta, GA 30310, USA

[c]Deparment of Pharmacology of Traditional Chinese Medicine, Guangdong Province's Traditional Chinese Medical Hospital, Guangzhou, GD 510120, P. R. China

[d]Department of Pharmacology, Zhongshan School of Medicine, Sun Yat-sen University, Guangzhou，GD 510080, P. R. China

[e]Guangzhou Cellprotek Pharmaceutical, G Building F/4, 3 Lanyue Road, Science City, Guangzhou 510663, P. R. China

[f]Department of Biochemistry, Zhongshan School of Medicine, Sun Yat-sen University, Guangzhou，GD 510080, P. R. China

[1]These authors contributed equally to this work.