

Title: Ropivacaine prevents activation of NLRP3 inflammasome caused by high glucose in HUVECs

Authors: Xin Huang*, Jingyan Jiang, Lijun Huang, Qiusheng Ren, Xiang Gao, Shenghui Yu

Affiliations:

Department of Anesthesiology, The Affiliated People's Hospital of Ningbo University

***, Corresponding to:**

Xin Huang

Department of Anesthesiology, The Affiliated People's Hospital of Ningbo University, 315040, China

Address : No. 251, Baizhang East Road, Yinzhou, Ningbo, 315040, China

Tel/Fax: +86-0574-87016852

E-mail: Huangxin1784@163.com

Figure S1

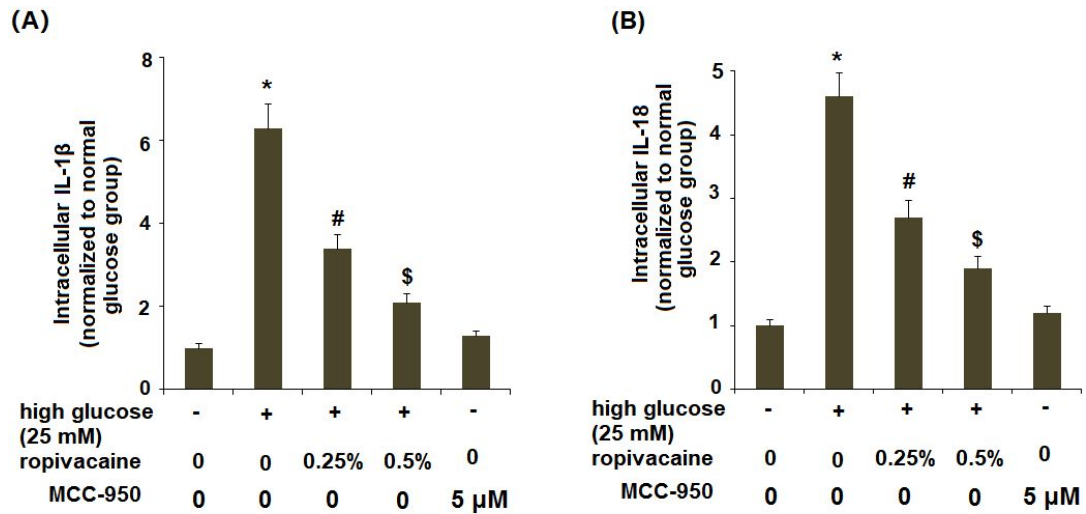


Figure S1. Ropivacaine inhibits high glucose-induced expression of intracellular IL-1 β and IL-18. HUVECs were treated with high glucose (25 mM) in the presence or absence of ropivacaine (0.25%, 0.5%) MCC-950 (5 μ M) for 24 h. (A). Intracellular IL-1 β was measured by ELISA analysis; (B). Intracellular IL-18 was measured by ELISA assay (*, P<0.01 vs normal glucose; #, P<0.01 high glucose only; \$, P<0.01 vs. high glucose+ropivacaine (0.25%), N=5-6).

Figure S2

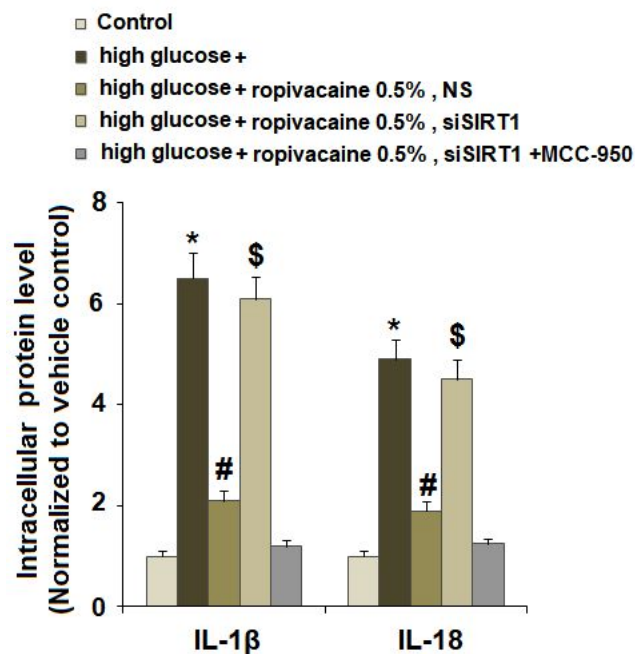


Figure S2. Silencing of SIRT1 abolished the inhibitory effects of ropivacaine on intracellular IL-1 β and IL-18 production. HUVECs were transfected with SIRT1 siRNA. At 12 h post transfection, cells were treated with high glucose (25 mM) in the presence or absence of ropivacaine (0.5%) for 24 h. NS, non-specific group; siSIRT1, SIRT1 siRNA. Intracellular IL-1 β and IL-18 were measured by ELISA assay (*, $P < 0.01$ vs normal glucose; #, $P < 0.01$ high glucose only; \$, $P < 0.01$ vs. high glucose+ropivacaine (0.5%)+NS group, N=5-6).