Suobei Li, J Diabetes Metab 2014, 5:10
http://dx.doi.org/10.4172/2155-6156.S1.026

OMICS Group Conferences
Accelerating Scientific Discovery

5th World Congress on
Diabetes & Metabolism
November 03-05, 2014 Embassy Suites Las Vegas, USA

# Sevoflurane pretreatment reduces high glucose-induced oxidative stress and inflammatory injury in human umbilical vein endothelial cells

**Suobei Li**
Central South University, China

**Background:** Hyperglycemia induced oxidative stress and inflammatory injury result in endothelial dysfunction, which is a risk factor for cardiovascular disease. Sevoflurane, one of the most commonly used volatile anesthetics, has been proven to be effective in combating oxidative stress and protecting organs against inflammatory injury in various conditions. We hypothesized that sevoflurane pretreatment could protect endothelial cells against high glucose-induced endothelial dysfunction, and that the protective effects of sevoflurane preconditioning might be associated with the reduction of inflammatory cytokines.

**Methods:** Primary cultured human umbilical vein endothelial cells (HUVECs) were exposed to sevoflurane (0.5, 1.5 and 2.5 minimum alveolar concentration, MAC) for 30 min with or without N(G)-nitro-L-arginine methyl ester (L-NAME, a nitric oxide synthase inhibitor) 1 mMfor 1 hour, and then incubated with high glucose (HG, 25 mM) for 12 hours, respectively. Tumor necrosis factor-α(TNF-α) release in culture medium was detected by using ELISA. Expression of intracellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) (cell ELISA and Western blot) and activation of NF-κB (Western blot) were also assessed. Adhesion of HRP-labeledHL-60 to HUVECs was measured by a spectrophotometer. The intracellular reactive oxygen species (ROS) production was assessed by monitoring the DCF fluorescence level after incubation with 25 mM glucose for 1 hour.

**Results:** In our study, the high glucose-induced increases in TNF-α release and expression of ICAM-1 and VCAM-1 were significantly attenuated by pretreatment with sevoflurane in a dose-dependent manner in cultured HUVECs. Enhanced cell adhesion caused by high glucose in co-cultured HL-60 and HUVEC was also blocked by pretreatment with sevoflurane. Pretreatment with sevoflurane also blocked formation of high glucose-induced ROS in HUVECs. In addition, sevoflurane suppressed the transcriptional activity of NF-κB and IκB phosphorylation under high glucose conditions. Pretreatment with L-NAME attenuated the protective action of sevoflurane on high glucose-induced ICAM-1 and VCAM-1 expression, activation of NF-κB and leukocytes adhesion to HUVECsαsuggesting a potential role of nitric oxide (NO) signaling.

**Conclusion:** The present data suggest that sevoflurane could suppress high glucose-induced oxidative stress and vascular inflammatory processes, and sevoflurane confers cytoprotective effects likely through inhibition of ROS and NF-κB activation in aNO-dependent manner in HUVECs.

lisuobei@hotmail.com