

**Title:** Effects of propofol on Ang II-increased NADPH oxidase activity and reactive oxygen species formation in cardiac fibroblasts

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**Aims:** In our previous report that propofol could inhibited cardiac fibroblast proliferation. However, the intracellular mechanism of propofol still remains unclear. The aims of this study were to test the anti-oxidative influence of propofol on Ang II-increased NADPH oxidase activity and reactive oxygen species (ROS) formation in cardiac fibroblasts

**Methods:** Cultured cardiac fibroblasts were exposed to Ang II in the presence of propofol. The NADPH oxidase activity in human endothelial cells was measured by NADPH oxidase assay(*Am J Physiol Heart Circ Physiol* 2004;**287**:H1254). Superoxide was measured by lucigenin-amplified chemiluminescence. The level of ROS are measured using as previously.(*J Mol Cell Cardiol*33:1805-14) The DCF level in cardiac fibroblasts were also measured by Flow cytometric histogram. The p value less than 0.05 were considered significant(ANOVA).

**Results:** Pretreatment of cultured cardiac fibroblasts with propofol (10  $\mu$  M) significantly inhibited Ang II-induced NADPH oxidase activity, superoxide formation, and ROS formation as measured after Ang II treatment. These findings support that propofol inhibited Ang II-increased NADPH oxidase activity and intracellular ROS levels in cardiac fibroblasts

**Discussion:** Propofol could significantly decreased Ang II-induced intra-cellular ROS formation in Ang-II stimulated cardiac fibroblasts which possessing an anti-proliferative effect. These results indicated that beside the anti-hypertensive effect, propofol also possess an anti-proliferative activity and may have much therapeutic potential in cardio-vascular diseases.