

# Evaluation of the effects of angiotensin II and mitochondria transplantation on IL-1 $\beta$ , IL-6 and IL-10 cytokines in rat cardiomyoblast cells

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Angiotensin II (Ang II) causes mitochondrial dysfunction in the cardiovascular system, which is one of the underlying causes of cardiovascular pathologies. This study aimed to investigate the effects of Ang II and mitochondria transplantation on the proliferation, apoptosis, inflammatory and anti-inflammatory cytokines of rat cardiomyoblasts (H9c2). Mitochondria were isolated from rat mesenchymal stem cells (MSCs) with a commercial kit. Total protein and ATP levels were measured. Ang II (0.1  $\mu$ M) and Mitochondria (5  $\mu$ g/mL, 10  $\mu$ g/mL) cytotoxicity was evaluated by MTT method. IL-1 $\beta$ , IL-6, IL-10 and caspase-3 levels were measured with ELISA. It was determined that decreasing doses of Ang II starting from 10  $\mu$ M increased cell proliferation. H9c2 proliferation increased in the group that received Ang II (0.1  $\mu$ M)+Mitochondria (10  $\mu$ g/mL) compared to Ang II (0.1  $\mu$ M) group. IL-6 levels showed a partial decrease with Ang II alone (0.1  $\mu$ M) compared to the control. In addition, with the combined application of Ang II (0.1  $\mu$ M) + Mitochondria (5  $\mu$ g/mL), IL-6 levels decreased compared to both Ang II alone (0.1  $\mu$ M) and the control group. On the other hand, it was found that IL-10 levels increased, on the contrary. These results suggest that mitochondria transplantation MT may enhance anti-inflammatory activity by Ang II type-2 receptors and affect in various apoptotic and proliferative pathways. Further studies are needed to elucidate the relationship between Ang II receptors and mitochondria on intracellular inflammatory signaling processes.

**Keywords:** Apoptosis, Inflammatory, Anti-inflammatory cytokines, Angiotensin II, Mitochondria isolation