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Angiotensin II induces Reactive Oxygen Species (ROS) generation and apoptosis in lymphoma EL4 cells

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Background/aim: Renin angiotensin system (RAS) is implicated in cardiovascular diseases. Ang II infusion induces hypertension and vascular damage especially endothelial dysfunction, inflammation and oxidative stress. Angiotensin II receptors are expressed in immune cells like T cells and monocytes. Tumour become more aggressive and dangerous in parallel to angiogenesis development witch induce intravasation and extravasation cells going from primary tumour to blood and then from blood to colonize new site and induce metastasis. Macrophages play a pivotal role in inflammation and cancer microenvironment. We aim to study the effect of angiotensin II in Macrophage polarization and cancer microenvironment related to innate immunity.

Materials and methods: Malignant lymphoma EL4 cells were incubated for 24h, 48h and 72h with conditioned media of macrophages stimulated with Angiotensin II (1Um) for 24h. We determine the effect of Angiotensin II stimulate macrophages in Reactive Oxygen Species (ROS) generation using CellRox kit, viability and apoptosis using immunofluorescence and cell phenotyping using flow cytometry.

Results and conclusion: There was a significant increase in ROS generation in cells incubated with Angiotensin II compared to control. Angiotensin II increases significantly apoptosis time dependently from 24 to 72h and change cell CD inflammatory markers after 24h incubation. These differences may be caused by significant increase in ROS generation in cells incubated with Angiotensin II compared to control.

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