

MITOCHONDRIAL GENERATION OF OXYGEN RADICALS BY PERIPHERAL BLOOD MONONUCLEAR CELLS (PBMC's) IN A MODEL OF CHRONIC HEART FAILURE

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The usefulness of finding the source for the generation of reactive oxygen species will provide a greater understanding about the role of aldosterone in the neuroendocrine-immune interface in chronic heart failure. This will lead to a better understanding of the pathophysiology of chronic heart failure. ROS are both free radicals and reactive anions containing oxygen atoms, or molecules containing oxygen atoms that can either produce free radicals or are chemically activated by them. Examples are hydroxyl radical, superoxide, hydrogen peroxide, and peroxynitrite. ROS are primarily responsible for myocardial cell apoptosis. Peripheral Blood Mononuclear Cells (PBMC) treated with aldosterone, a mineralocorticoid hormone, generates increase levels of ROS, primarily H₂O₂ and invades the heart causing lesions similar to those seen in patients with chronic heart failure. Since previous research has shown that PBMC of mitochondria of aldosterone/salt treated rats generate increased level of ROS, we hypothesized that the intracellular source of the ROS are the PBMC of mitochondria. To test this hypothesis we used mitochondria specific probe MitoTracker Red (CM-H₂XROS) assay to assess mitochondrial ROS production by PBMC from aldosterone/salt treated rats compared to age/gender-matched untreated control rats. The PBMC fluorescence was then analyzed by single cell flow cytometry using a FACS cytometer. The results of the study show direct evidence for the increased generation of ROS by the peripheral blood mononuclear cells of mitochondria.