



Types of Cardiovascular Diseases due to Reactive Oxygen Species

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ARTICLE HISTORY

Received: 01-Nov-2022, Manuscript No. EJMOAMS- 22-79584;
Editor assigned: 04-Nov -2022, PreQC No. EJMOAMS- 22-79584 (PQ);
Reviewed: 21-Nov -2022, QC No. EJMOAMS- 22-79584;
Revised: 28-Nov-2022, Manuscript No. EJMOAMS- 22-79584 (R);
Published: 05-Dec-2022

Description

ROS are molecules containing one or more unpaired electrons in their orbital that provide a significant degree of reactivity towards ROS. Reactive Oxygen Species (ROS) have been linked to the pathogenesis of several diseases, including cardiovascular diseases. The presence of increased amounts of ROS has been found to cause damage to major macromolecules in cells including vascular Endothelial Cells (EC) and other cell types in the cardiovascular system, leading to cellular and systemic dysfunction. For example, endothelial dysfunction caused by increased ROS has been shown to contribute to the development of atherosclerosis, hypertension, ischemic heart disease, ischemia-reperfusion injury, and other vascular diseases.

ROS have been reported to affect almost all organs in the body, leading to pathological conditions such as pulmonary fibrosis and vascular retinopathy. Many studies indicate the essential role of antioxidants in improving pathophysiological conditions. Several studies from different groups demonstrated that the reduction of ROS had rather deleterious effects on the cardiovascular system, including endothelial cells [1,2]. Several recent studies have supported a beneficial role of increased ROS in the vasculature, depending on the source and duration of subcellular ROS [3–6].

There are several different intracellular sources of ROS, including Dinucleotide Phosphate (NADPH) oxidases, mitochondria, peroxisomes, lysosomes, xanthine oxidases, cytochrome P450, etc. Originally, ROS-producing enzymes were discovered in phagocytic cells, where large amounts of ROS are produced physiologically by the membrane-bound enzymes NADPH oxidase (NOX) to kill microbes.

Types

Pulmonary and renovascular hypertension: ROS are also involved in renovascular hypertension and chronic

pulmonary hypertension associated with hypoxia. In renovascular hypertension, which is caused by increased renal renin release, ROS are thought to impair endothelium-dependent vasorelaxation [7]. The finding that *in vivo* administration of polyethylene glycol-conjugated superoxide dismutase (PEG-SOD) or *in vitro* treatment with the antioxidant tirone improved endothelial function in an AngII animal model also supports a critical role for ROS. ROS levels have been found to correlate with the level of hypoxia, which in turn plays an important role in the pathogenesis and development of pulmonary hypertension.

Atherosclerosis and ischemic heart disease: Endothelial dysfunction is one of the initial events that accompany atherosclerosis and coronary heart disease [8]. In conditions predisposing to atherosclerosis, such as hypercholesterolemia, ROS react with Low-Density Lipoproteins (LDL), leading to the formation of oxidized LDL (oxLDL), which is the main effector in the development of atherosclerosis. Increased NADPH oxidase activity and high ROS levels have been reported in atherosclerotic lesions. Increased p22phox subunit expressions along with elevated ROS levels in atherosclerotic lesions of patients with unstable angina have been reported.

Ischemic-reperfusion injury: Ischemic damage to the myocardium causes hypoxia and reduced oxygenation of the area supplied by the stenosis or blocked coronary vessel. It most often occurs in atherosclerosis. Deoxygenated cells are subject to reversible cell damage if the lack of blood is transient or short-term. Cardiac muscle cells are able to withstand ischemia for a maximum of 30 minutes, after which they undergo irreversible injury followed by death by necrosis. Necrotic tissue releases its cellular contents, including enzymes and ROS, into the surrounding environment. Reperfusion at this stage is more harmful than beneficial; simply put, the spread of these toxic substances into the environment causes further damage to the surrounding healthy cells.

Beneficial effects of ROS

Several researchers have hypothesized that stopping the abnormal production of ROS and increasing the natural protective activity of antioxidants may lead to beneficial outcomes in the treatment of cardiovascular disease.

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