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The Importance of ROS and Redox Signalling in Tissue and Oxidative Stress

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Editorial

Inflammation is linked to the production of soluble mediators that promote cellular activation and migration of inflammatory leukocytes to the site of damage, as well as endothelial expression of adhesion molecules and enhanced vascular permeability. It is a strictly regulated step-by-step process that has evolved to deal with a wide range of various inflammatory stimuli. However, under certain physio pathological conditions, the inflammatory response overwhelms local regulatory mechanisms and leads to systemic inflammation, which may affect metabolism in distant tissues and organs, as mitochondria are able to perceive signals of inflammation. Because mitochondria are one of the first organelles to be affected by a dysregulation in the systemic inflammatory response, it has been associated with the progression of the physio pathological mechanisms [1].

Mitochondria are a key source of within most mammalian cells and hence play an important role in oxidative. Production may contribute to mitochondrial damage in a variety of diseases, and it also plays a role in a complicated redox signalling network that connects the organelle to the rest of the cell. As a result, a role for ROS produced by mitochondria in modulating inflammatory signals was proposed, and mitochondria have been implicated in numerous aspects of the inflammatory response. Exposure to air particle matter is an inflammatory disease that disrupts mitochondrial activity in various organs. PM absorption by alveolar macrophages has been shown to produce local cell activation and recruitment, cytokine release, and lung inflammation following both acute and chronic pollutant exposure [2]. Following that, inflammatory mediators have been found to impaired mitochondrial activity, which leads to bioenergetics dysfunction and increased oxidant generation, has been found to influence tissue homeostasis and organ function in this proinflammatory milieu.

The purpose of this review is to explain the most recent findings in the cellular and molecular pathways that link systemic inflammation and mitochondrial dysfunction in several organs, using air pollution as a case study. By linking the oxidation of fatty acids and pyruvate with the synthesis of adenosine triphosphate via electron transport, mitochondria have historically been regarded as the primary source of cellular energy. They are complicated organelles that regulate homeostasis, apoptosis, and differentiation, among other things [3]. Mitochondria are a significant source of energy in most mammalian cells and are thus heavily implicated in oxidative stress, where increased production may contribute to mitochondrial damage in a variety of diseases. They also serve an important part in a redox signalling system in which the organelle communicates with the remainder of the cell. Recently, new mitochondrial functions have been proposed, specifically linking alterations in the mechanisms linked to generation with the inflammatory responses involved in different pathological conditions the cellular and molecular mechanisms that link systemic inflammation and mitochondrial dysfunction in different organs, taking air pollution [4].

The term ROS was first used to characterise the luminol chemiluminescence of activated human monocytes. Despite the fact that the mentioned work did not offer a characterization of the involved chemical species, the concept was quickly adopted by the scientific community. Originally, superoxide anion hydrogen peroxide H_2O_2 and hydroxyl radical HO were present, which resulted from the partial reduction of molecular O_2. When molecule O_2 takes an electron in its basal state, the result is a reactive chemical species with only one unpaired electron. When a second electron is added, the peroxide ion is formed, of which H_2O_2 is the most prevalent type at physiological pH. H_2O_2 is less reactive because it lacks unpaired electrons [5]. However, this molecule is regarded as because the O-O bond has low bond energy of 138 kJ/mol, it produces a reactive O_2 species. As a result, it can breakdown, resulting in the generation of HO, which has such a high reactivity that it reacts very close to the site of formation.

Conflict of Interest

None.

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