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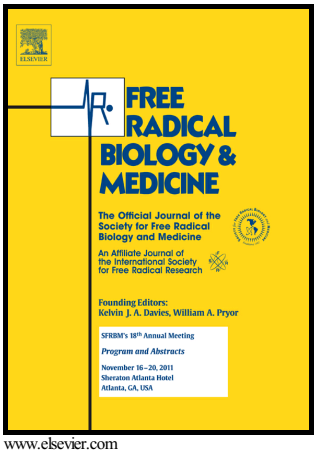

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Oxidative stress in autoimmune rheumatic diseases.

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Abstract

The management of patients with autoimmune rheumatic diseases such as rheumatoid arthritis (RA) remains a significant challenge. Often the rheumatologist is restricted to treating and relieving the symptoms and consequences and not the underlying cause of the disease. Oxidative stress occurs in many autoimmune diseases, with the excess production of reactive oxygen species (ROS) and reactive nitrogen species (RNS). The sources of such reactive species include NADPH oxidases (NOXs), the mitochondrial electron transport chain, nitric oxide synthases, nitrite reductases, and the hydrogen sulfide producing enzymes cystathionine-β synthase and cystathionine-γ lyase. Superoxide undergoes a dismutation reaction to generate hydrogen peroxide which, in the presence of transition metal ions (e.g. ferrous ions), forms the hydroxyl radical. The enzyme myeloperoxidase, present in inflammatory cells, produces hypochlorous acid, and in healthy individuals ROS and RNS production by phagocytic cells is important in microbial killing. Both low molecular weight antioxidant molecules and antioxidant enzymes, such as superoxide dismutase, catalase, glutathione peroxidase, and peroxiredoxin remove ROS. However, when ROS production exceeds the antioxidant protection, oxidative stress occurs.

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