Reactive oxygen species and superoxide dismutases: role in joint diseases.

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BACKGROUND: Reactive oxygen species (ROS) are produced in many normal and abnormal processes in humans, including atheroma, asthma, joint diseases, aging, and cancer. The superoxide anion $\text{O}_2^-$ is the main ROS.

DISCUSSION: Increased ROS production leads to tissue damage associated with inflammation. Superoxide dismutases (SODs) convert superoxide to hydrogen peroxide, which is then removed by glutathione peroxidase or catalase. Thus, SODs prevent the formation of highly aggressive ROS, such as peroxynitrite or the hydroxyl radical. Experimental models involving SOD knockout or overexpression are beginning to shed light on the pathophysiological role of SOD in humans. Although the antiinflammatory effects of exogenous native SOD (orgotein) are modest, synthetic SOD mimetics hold considerable promise for modulating the inflammatory response.

CONCLUSION: In this review, we discuss new knowledge about the role of the superoxide anion and its derivates as mediators of inflammation and the role of SODs and SOD mimetics as antioxidant treatments in joint diseases such as rheumatoid arthritis, osteoarthritis, and crystal-induced arthropathies.

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