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ROS: Dual Roles in Health and Disease

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Abstract

Reactive *Oxygen Species* (ROS) exert multifaceted roles in biology, ranging from essential signaling to mediating oxidative stress in various pathologies. This compilation explores ROS's dual nature in cancer, their involvement in neurodegenerative, cardiovascular, and metabolic diseases, and their contribution to renal fibrosis. It also highlights their intricate regulation of autophagy and critical function in plant immunity. Understanding ROS mechanisms and their balance is crucial for developing therapeutic strategies across diverse biological systems.

Keywords

Reactive Oxygen Species; Oxidative Stress; Cancer; Neurodegenerative Diseases; Cardiovascular Diseases; Metabolic Diseases; Autophagy; Ferroptosis; Renal Fibrosis; Plant Immunity; Therapeutic Strategies

Introduction

Reactive oxygen species play a dual role in cancer, acting as signaling molecules that promote cell proliferation, survival, and metastasis, but also as mediators of oxidative stress that can induce cell death. This dual nature allows for therapeutic strategies that either suppress ROS to protect normal cells or enhance ROS to selectively kill cancer cells, making ROS a significant target for drug development [1].

The review highlights the complex relationship between ROS, inflammation, and autophagy in neurodegenerative diseases like Alzheimer's and Parkinson's. It explains how oxidative stress contributes to disease progression, but also discusses the protective roles of autophagy in clearing damaged cellular components and modulating inflammation, suggesting therapeutic avenues targeting these pathways [2].

This review explores the critical involvement of ROS and oxidative stress in the pathogenesis of various cardiovascular diseases, including atherosclerosis, hypertension, and heart failure. It emphasizes the potential therapeutic benefits of antioxidant strategies, discussing both endogenous and exogenous antioxidants in mitigating cardiovascular damage and improving outcomes [3].

This paper focuses on the critical role of mitochondrial ROS (mtROS) in the development and progression of various metabolic diseases, including obesity, type 2 diabetes, and non-alcoholic fatty liver disease. It details the mechanisms by which mtROS imbalance contributes to metabolic dysfunction and explores current and potential therapeutic interventions targeting mtROS production and scavenging pathways [4].

This article delves into the significant role of reactive oxygen species and the resulting oxidative stress in the pathogenesis of various neurological disorders, such as stroke, Parkinson's, and Alzheimer's disease. It clarifies how oxidative damage to lipids, proteins, and DNA contributes to neuronal cell death and dysfunction, highlighting potential therapeutic targets aimed at modulating redox balance in the brain [5].

This article examines the intricate relationship between reactive oxygen species and ferroptosis, a distinct form of regulated cell death characterized by iron-dependent lipid peroxidation, in the context of cancer. It elucidates how ROS generation is central to initiating and executing ferroptosis, presenting this pathway as a promising therapeutic strategy for overcoming drug resistance and treating various cancers [6].

This review discusses the critical and multifaceted role of reactive oxygen species in orchestrating plant immune responses. It explains how ROS act as primary messengers, triggering defense signaling cascades, and also contribute to direct antimicrobial action, demonstrating their crucial function in protecting plants against pathogen invasion [7].

This article explores the central involvement of reactive oxygen species in the pathogenesis and progression of renal fibrosis, a common pathway to end-stage kidney disease. It details how oxidative stress contributes to cellular damage, inflammation, and extracellular matrix accumulation in the kidney, and discusses various antioxidant-based therapeutic strategies aimed at mitigating fibrosis and preserving renal function [8].

This paper investigates the intricate regulatory roles of reactive oxygen species in autophagy, a crucial cellular process for degrading and recycling damaged components. It describes how ROS can both induce and inhibit autophagy depending on their concentration and cellular context, influencing cell survival, death, and disease progression, offering insights into therapeutic interventions [9].

This comprehensive review provides a balanced perspective on the role of reactive oxygen species in human health and disease. It highlights the physiological functions of ROS in cellular signaling, immune defense, and homeostasis, while also detailing their pathological contributions to oxidative stress-related diseases such as cancer, neurodegeneration, and cardiovascular disorders, suggesting that maintaining ROS balance is crucial [10].

Description

Reactive oxygen species (ROS) play a crucial and multifaceted role in human health and disease, encompassing physiological functions like cellular signaling and immune defense, alongside pathological contributions to various oxidative stress-related conditions [10]. Maintaining ROS balance is therefore critical. Specifically in cancer, ROS exhibit a dual nature; they act as signaling molecules promoting cell proliferation, survival, and metastasis, but also mediate oxidative stress leading to cell death [1]. This dual characteristic opens avenues for therapeutic strategies that either suppress ROS to protect normal cells or enhance ROS to selectively kill cancer cells, making ROS a significant target for drug development [1].

Beyond the general dual role, the intricate relationship between Reactive Oxygen Species and ferroptosis, a distinct form of iron-dependent lipid peroxidation-driven cell death, is central to understanding cancer progression and resistance [6]. ROS generation is key to initiating and executing ferroptosis, presenting this pathway as a promising therapeutic strategy for overcoming drug resistance and treating various cancers [6]. Furthermore, the complex interplay involving ROS, inflammation, and autophagy is particularly relevant in neurodegenerative diseases like Alzheimer's and Parkinson's [2]. Oxidative stress significantly contributes to disease progression, yet autophagy also offers protective roles by clearing damaged cellular components and modulating inflammation, pointing to therapeutic opportunities targeting these pathways [2].

Reactive Oxygen Species and the resultant oxidative stress are deeply involved in the pathogenesis of various neurological disorders, including stroke, Parkinson's, and Alzheimer's disease [5]. Oxidative damage to lipids, proteins, and DNA contributes significantly to neuronal cell death and dysfunction, underscoring the importance of modulating redox balance in the brain as a therapeutic target [5]. In the cardiovascular system, ROS and oxidative stress are critically involved in conditions such as atherosclerosis, hypertension, and heart failure [3]. Antioxidant strategies, both endogenous and exogenous, show potential therapeutic benefits in mitigating cardiovascular damage and improving outcomes [3].

Mitochondrial Reactive Oxygen Species (mtROS) play a critical role in the development and progression of metabolic diseases, including obesity, type 2 diabetes, and non-alcoholic fatty liver disease [4]. An imbalance in mtROS contributes to metabolic dysfunction, leading to therapeutic interventions targeting mtROS production and scavenging pathways [4]. Similarly, ROS are centrally involved in the pathogenesis and progression of renal fibrosis, a common pathway to end-stage kidney disease [8]. Oxidative stress contributes to cellular damage, inflammation, and extracellular matrix accumulation in the kidney, with antioxidant-based therapeutic strategies showing promise in mitigating fibrosis and preserving renal function [8].

Furthermore, Reactive Oxygen Species intricately regulate autophagy, a crucial cellular process for degrading and recycling damaged components [9]. ROS can both induce and inhibit autophagy based on their concentration and cellular context, influencing cell survival, death, and disease progression, thereby offering insights into potential therapeutic interventions [9]. Looking beyond human health, ROS also play a critical and multifaceted role in orchestrating plant immune responses [7]. They act as primary messengers, triggering defense signaling cascades, and contribute to direct antimicrobial action, demonstrating their crucial function in protecting plants against pathogen invasion [7].

Conclusion

Reactive Oxygen Species (ROS) are fundamental molecules with complex roles in biology, acting as both essential signaling mediators and potent drivers of oxidative stress. In human health, ROS contribute to normal physiological functions like cellular signaling and immune defense, but their imbalance is implicated in a wide array of pathological conditions. These include cancer, where ROS have a dual nature—promoting cell growth yet also inducing cell death, making them a key target for therapeutic strategies like ferroptosis induction [1, 6]. ROS are also central to neurodegenerative diseases such as Alzheimer's and Parkinson's, where they interact with inflammation and autophagy to drive disease progression, though autophagy can also be protective [2, 5].

Beyond neurological disorders, ROS and oxidative stress are critical in cardiovascular diseases like atherosclerosis and heart failure, prompting interest in antioxidant therapies [3]. Mitochondrial ROS (mtROS) specifically contribute to metabolic diseases, including obesity and type 2 diabetes, highlighting mtROS-targeting interventions [4]. The progression of renal fibrosis, leading to end-stage kidney disease, is also heavily influenced by oxidative stress, with antioxidant strategies offering potential [8]. On a cellular level, ROS intricately regulate autophagy, either inducing or inhibiting it depending on context, affecting cell fate [9]. Remarkably, ROS also play a crucial role in plant immunity, acting as key messengers and antimicrobial agents against pathogens [7]. Overall, understanding and modulating ROS balance is vital across diverse biological systems and disease contexts [10].

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