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ROS: Diverse Roles, Disease, and Therapy

Dr. Claire L. Dupont*

Sorbonne University, Molecular Physiology Laboratory France

***Corresponding Author:** Dr. Claire L. Dupont, Sorbonne University, Molecular Physiology Laboratory France, E-mail: c.dupont@sorbonnebio.fr

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Abstract

Reactive *Oxygen Species* (ROS) exhibit diverse roles across biological systems, contributing to both cellular signaling and disease pathology. These molecules are critical in cancer progression, influencing autophagy, homeostasis, and ferroptosis. They are key regulators in inflammation, aging, and metabolic diseases, often via mitochondrial ROS. ROS also mediate plant immunity and T cell function. Furthermore, excessive ROS contribute to neurodegenerative conditions, acute kidney injury, and heart failure. Modulating ROS pathways offers significant therapeutic potential for treating a wide array of human diseases.

Keywords

Reactive Oxygen Species; Cancer; Inflammation; Aging; Plant Immunity; T Cell Function; Metabolic Diseases; Neurodegeneration; Kidney Injury; Heart Failure

Introduction

Reactive oxygen species (ROS) are fundamental to various biological processes, playing intricate and often dual roles in maintaining cellular health and contributing to disease pathogenesis. For instance, in cancer, ROS are pivotal in regulating autophagy, a process that can either promote tumor survival or induce cell death, depending on the cellular context and the specific levels of ROS present. Researchers suggest that targeting ROS-mediated autophagy could be a viable therapeutic strategy, opening avenues for novel drug development [1].

Furthermore, the role of mitochondrial reactive oxygen species (mtROS) is critical, particularly as key regulators in the progression of inflammation and the aging process. These molecules contribute significantly to cellular damage and activate signaling pathways that are deeply implicated in age-related diseases. Gaining insights into these roles provides a foundation for developing potential interventions aimed at delaying aging and mitigating chronic inflammatory conditions [2].

Beyond human physiology, ROS are essential in plant immunity, where plants employ them for both continuous surveillance and rapid, localized bursts when responding to pathogen attacks. These reactive molecules activate downstream signaling pathways, underscoring their vital function as mediators in plant defense mechanisms against various environmental threats [3].

In cancer biology, the complex interplay of ROS homeostasis and signaling is a major area of study. Cancer cells are known to manipulate ROS levels strategically to promote their own growth and survival. This understanding highlights the potential for therapies that modulate ROS pathways, offering new directions for cancer treatment [4].

Another significant interaction in cancer involves the crosstalk between ferroptosis, a distinct form of regulated cell death, and reactive oxygen species. ROS generation is a critical trigger for ferroptosis, and exploring this connection has important implications

for developing innovative therapeutic strategies. Manipulating oxidative stress through this pathway could lead to more effective cancer interventions [5].

The immune system also relies heavily on ROS for regulating T cell function. Both physiological and pathological concentrations of ROS influence T cell activation, differentiation, and overall immune responses. The insights gained from this area are invaluable for understanding immune disorders and for crafting targeted immunotherapies [6].

Metabolic diseases are another domain where mitochondrial reactive oxygen species (mtROS) play a significant role in their pathogenesis. Elucidating the mechanisms through which mtROS contribute to metabolic dysfunction is crucial for exploring potential therapeutic strategies focused on mitigating their harmful effects. This research provides a clear roadmap for addressing metabolic disorders through targeted antioxidant interventions [7].

Neurodegenerative diseases also involve ROS at their core. Reviews meticulously detail the mechanisms of ROS generation, the subsequent mitochondrial dysfunction, and the therapeutic implications for conditions such as Alzheimer's and Parkinson's. Researchers propose that targeting oxidative stress pathways represents a promising strategy for neuroprotection [8].

In acute kidney injury (AKI), current perspectives highlight the critical role of reactive oxygen species. Excessive ROS production is shown to contribute to renal cell damage and functional decline, involving complex underlying pathways. Identifying therapeutic opportunities to target ROS in AKI offers hope for improved management and treatment strategies for patients [9].

Finally, oxidative stress and the pivotal role of reactive oxygen species are central to the pathology of heart failure. Increased ROS levels contribute to cardiac dysfunction and remodeling through delineated mechanisms. Various therapeutic interventions aimed at modulating oxidative stress are discussed, providing potential strategies for preventing and treating this debilitating condition [10].

Description

Reactive Oxygen Species (ROS) are ubiquitous signaling molecules with profound impacts across diverse biological systems, often exhibiting dual functionality depending on their concentration and cellular context. In the realm of cancer biology, ROS play a multifaceted role. They are deeply involved in regulating autophagy, a process critical for cellular homeostasis, which, when mediated by ROS, can either facilitate tumor survival or trigger cell death [1]. Cancer cells frequently exploit and manipulate ROS levels to promote their own proliferation and survival, making ROS homeostasis and signaling a key area for therapeutic exploration. Modulating these pathways presents novel opportunities for cancer treatment [4]. Furthermore, the specific form of programmed cell death known as ferroptosis is intricately linked with ROS, where ROS generation serves as a fundamental trigger. Understanding this crosstalk offers new therapeutic avenues for cancer interventions by targeting oxidative stress [5].

Beyond cancer, mitochondrial reactive oxygen species (mtROS) emerge as central players in systemic conditions like inflammation and aging. They are significant contributors to cellular damage and activate specific signaling pathways that are intrinsically involved in age-related pathologies [2]. This highlights the potential for interventions that focus on delaying the aging process and alleviating chronic inflammatory states by modulating mtROS. Similarly, mtROS are critically implicated in the pathogenesis of various metabolic diseases. Research into the mechanisms by which mtROS drive metabolic dysfunction is paving the way for targeted antioxidant strategies to mitigate their harmful effects, offering a clear roadmap for therapeutic development in this area [7].

The influence of ROS extends to the intricate mechanisms of immunity, both in plants and animals. In plant immunity, ROS are utilized by plants for continuous surveillance against potential threats and for generating rapid, localized bursts of oxidative stress in response to pathogen attacks. These actions are crucial for activating downstream signaling pathways that underpin robust plant defense mechanisms [3]. In the human immune system, ROS are essential regulators of T cell function. Both normal physiological levels and pathological excesses of ROS significantly impact T cell activation, differentiation, and overall immune responses. These findings are invaluable for comprehending immune disorders and for developing precise immunotherapeutic approaches [6].

ROS are also intimately involved in the progression of several organ-specific diseases, underscoring their broad physiological relevance. In neurodegenerative diseases, including debilitating conditions like Alzheimer's and Parkinson's, ROS contribute significantly to their initiation and progression. This occurs through mechanisms involving ROS generation and subsequent mitochondrial dysfunction, suggesting that therapies targeting oxidative stress could offer promising neuroprotective strategies [8]. Acute kidney injury (AKI) similarly sees excessive ROS production leading to renal cell damage and functional decline. The complex pathways

involved make ROS a critical therapeutic target for improving the management and treatment of AKI patients [9]. Finally, in heart failure, oxidative stress and ROS play a pivotal role in pathology, contributing to cardiac dysfunction and remodeling. Investigating therapeutic interventions aimed at modulating this oxidative stress offers potential strategies for preventing and treating this severe condition [10]. Collectively, these insights demonstrate the pervasive nature of ROS as both harmful agents in disease and crucial mediators of biological functions, making their study fundamental for therapeutic advancement.

Conclusion

Reactive Oxygen Species (ROS) are critical molecules with diverse and often contrasting roles across biological systems. They are recognized for their involvement in various human pathologies, including cancer, inflammation, aging, metabolic diseases, and neurodegenerative conditions. In cancer, ROS regulate autophagy, influencing tumor survival or cell death depending on context, and cancer cells often manipulate ROS levels to promote growth and survival. Modulating ROS-induced autophagy and ROS homeostasis offers promising therapeutic strategies for cancer treatment [1, 4]. The interplay between ferroptosis, a distinct form of cell death, and ROS is also crucial in cancer, with ROS generation being a key trigger, suggesting new interventions by manipulating oxidative stress [5]. Beyond cancer, mitochondrial ROS (mtROS) are key regulators in inflammation and aging processes, contributing to cellular damage and signaling pathways implicated in age-related diseases. Understanding these roles is vital for interventions against aging and chronic inflammatory conditions [2]. Similarly, mtROS are central to the pathogenesis of metabolic diseases, and targeting these could lead to effective therapeutic strategies [7]. ROS also play significant roles in plant immunity, acting in both continuous surveillance and rapid, localized bursts against pathogens, mediating crucial defense mechanisms [3]. In the immune system, ROS influence T cell activation, differentiation, and overall responses, making their regulation important for understanding and treating immune disorders [6]. Furthermore, ROS are implicated in neurodegenerative diseases like Alzheimer's and Parkinson's, where their generation and mitochondrial dysfunction contribute to progression, highlighting oxidative stress as a therapeutic target [8]. Acute kidney injury and heart failure also involve ROS, with excessive production leading to renal cell damage and cardiac dysfunction. Therapeutic interventions aimed at modulating oxidative stress in these conditions offer hope for better management and treatment [9, 10]. Overall, understanding the intricate balance and specific roles of ROS is fundamental for developing novel therapeutic approaches across a wide spectrum of diseases.

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