

Oxidative Stress: Diseases, Mechanisms, Therapies

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Abstract

Oxidative stress is a critical factor in numerous pathologies, including neurodegeneration, cancer, cardiovascular diseases, diabetes, and age-related conditions. This comprehensive body of work highlights how reactive oxygen species damage cells, disrupt redox homeostasis, and fuel inflammation. It outlines the specific mechanisms underpinning these diseases, from mitochondrial dysfunction to gut microbiota interactions. Importantly, various therapeutic strategies are explored, including synthetic and natural antioxidants, Nrf2 pathway modulators, and mitochondrial-targeted interventions. Understanding these multifaceted roles of oxidative stress is vital for developing effective, targeted treatments and fostering healthy aging across diverse human diseases.

Keywords

Oxidative Stress; Neurodegeneration; Cancer; Cardiovascular Disease; Diabetes; Aging; Inflammation; Mitochondria; Antioxidants; Gut Microbiota

Introduction

This article offers a deep dive into the role of oxidative stress in neurodegenerative diseases like Alzheimer's and Parkinson's. It covers the mechanisms by which reactive oxygen species damage neurons and explores various therapeutic strategies aiming to mitigate this stress, including antioxidants, Nrf2 pathway modulators, and mitochondrial-targeted interventions. What this really means is that understanding these pathways is crucial for developing new treatments. This deep understanding of mechanisms like reactive oxygen species (ROS) damaging neurons and the exploration of various therapeutic strategies, including antioxidants and Nrf2 pathway modulators, provides crucial insights for developing new treatments targeting these complex neurodegenerative processes.[1].

This review explores the intricate relationship between oxidative stress, redox regulation, and cancer progression. It outlines how cellular redox imbalances contribute to cancer initiation, growth, metastasis, and drug resistance. The authors highlight the dual role of reactive oxygen species in promoting both cell proliferation and apoptosis, suggesting that carefully modulated redox therapies could be promising cancer treatments. The intricate balance of redox regulation is fundamental, and disruptions are clearly linked to cancer initiation, growth, metastasis, and drug resistance. This dual role of ROS, promoting both cell proliferation and apoptosis, suggests that precisely modulated redox therapies could hold significant promise as effective cancer treatments.[2].

Focusing on cardiovascular diseases, this article delves into how oxidative stress contributes to conditions like atherosclerosis, hypertension, and heart failure. It details the sources of reactive oxygen species in the cardiovascular system and the downstream cellular damage they cause. Importantly, it discusses emerging therapeutic strategies that target specific oxidative stress pathways to protect heart health, offering novel insights for treatment development. Specifically, the generation of reactive oxygen species within the

cardiovascular system leads to considerable cellular damage, contributing to conditions such as atherosclerosis, hypertension, and heart failure. Novel therapeutic strategies focusing on specific oxidative stress pathways are offering encouraging insights for protecting heart health and advancing treatment development.[3].

This paper investigates oxidative stress, redox homeostasis, and their connection to inflammaging – chronic low-grade inflammation associated with aging. It explains how disruptions in redox balance accelerate the aging process and contribute to age-related diseases. The findings here highlight the critical role of maintaining cellular redox balance to promote healthy aging and mitigate chronic inflammatory conditions. Disruptions in cellular redox balance are shown to accelerate the aging process and contribute to a range of age-related diseases. Therefore, the critical importance of maintaining this balance cannot be overstated in promoting healthy aging and effectively mitigating chronic inflammatory conditions associated with the aging process.[4].

The article focuses on mitochondrial oxidative stress and its profound impact on various diseases. Mitochondria are the primary site of reactive oxygen species production, and their dysfunction leads to a cascade of cellular damage. This work outlines current understanding of how mitochondrial oxidative stress underpins metabolic, neurodegenerative, and cardiovascular diseases, along with potential therapeutic strategies to target mitochondrial health. As the primary site of ROS production, mitochondrial dysfunction initiates a destructive cascade of cellular damage that underlies many metabolic, neurodegenerative, and cardiovascular diseases. Current understanding of this critical link is paving the way for targeted therapeutic strategies aimed at restoring and maintaining mitochondrial health.[5].

This paper examines the significant role of oxidative stress in the pathogenesis and complications of diabetes mellitus. It covers how hyperglycemia induces oxidative stress, leading to pancreatic beta-cell dysfunction and insulin resistance, and driving complications like retinopathy, nephropathy, and neuropathy. Here's the thing: understanding these mechanisms is vital for developing effective antioxidant-based therapies to manage diabetic complications. Hyperglycemia, a hallmark of diabetes, directly induces oxidative stress, which in turn impairs pancreatic beta-cell function and fosters insulin resistance. This leads to severe complications, including retinopathy, nephropathy, and neuropathy. Recognizing these specific mechanisms is truly vital for developing effective antioxidant-based therapies to manage and prevent diabetic complications.[6].

This article explores the immense potential of natural antiox-

idants in preventing and treating diseases by targeting oxidative stress. It discusses various classes of natural compounds, their antioxidant mechanisms, and their efficacy in mitigating oxidative damage in different disease models. What this really means is that leveraging dietary and natural sources of antioxidants could offer a promising approach to enhance health and combat pathology. These natural compounds exhibit diverse antioxidant mechanisms and have demonstrated efficacy in mitigating oxidative damage across various disease models. Leveraging these readily available dietary and natural sources of antioxidants could represent a highly promising and accessible approach to enhance overall health and effectively combat a wide range of pathologies.[7].

This review illuminates the 'vicious cycle' between oxidative stress and inflammation in chronic diseases. It explains how each process exacerbates the other, leading to persistent tissue damage and disease progression. Understanding this interplay is key, as breaking this cycle through targeted interventions could offer new avenues for managing chronic inflammatory conditions like autoimmune disorders and metabolic syndrome. This destructive interplay exacerbates tissue damage and drives disease progression in various chronic conditions. A deeper understanding of this vicious cycle is absolutely essential, as strategically breaking it through targeted interventions opens new and exciting avenues for effectively managing persistent inflammatory conditions such as autoimmune disorders and metabolic syndrome.[8].

This article focuses on the mechanisms of oxidative stress-induced brain damage and its implications for neurological diseases such as stroke, traumatic brain injury, and neurodegeneration. It outlines the specific cellular and molecular pathways through which oxidative stress harms brain tissue, emphasizing potential therapeutic targets. Let's break it down: effective neuroprotective strategies will likely need to address this oxidative damage. Oxidative stress inflicts specific cellular and molecular damage on brain tissue, with profound implications for neurological conditions including stroke, traumatic brain injury, and broader neurodegeneration. Recognizing these precise pathways is crucial, as effective neuroprotective strategies will undeniably need to comprehensively address and counteract this pervasive oxidative damage to the brain.[9].

This comprehensive review explores the fascinating and crucial interactions between oxidative stress and the gut microbiota in disease pathogenesis. It details how dysbiosis can either contribute to or be a consequence of oxidative stress, impacting various systemic diseases from inflammatory bowel disease to metabolic disorders. Understanding this bidirectional communication is essential for developing microbiota-targeted interventions. Dysbiosis, or an im-

balance in the gut microbiota, can either contribute to or arise as a consequence of oxidative stress, profoundly influencing a spectrum of systemic diseases from inflammatory bowel disease to various metabolic disorders. Appreciating this critical bidirectional communication is fundamental for developing innovative and effective microbiota-targeted interventions.[10].

Description

Oxidative stress, characterized by an imbalance between reactive oxygen species (ROS) production and the body's antioxidant defenses, plays a fundamental role in the pathogenesis of numerous human diseases. These reactive molecules cause cellular damage, impacting various biological functions across different organ systems. A deeper understanding of these underlying mechanisms, from cellular damage to systemic effects, is crucial for developing effective therapeutic interventions.

In neurodegenerative diseases like Alzheimer's and Parkinson's, oxidative stress directly damages neurons through the action of reactive oxygen species [1]. Effective neuroprotective strategies will therefore need to specifically address this oxidative damage to brain tissue, impacting conditions such as stroke and traumatic brain injury [9]. Mitochondria, being primary sites of ROS production, contribute significantly to this cascade of cellular damage in neurodegenerative conditions [5]. Understanding these pathways is crucial for developing new treatments [1].

The intricate relationship between oxidative stress, redox regulation, and cancer progression is well-established. Cellular redox imbalances contribute to cancer initiation, growth, metastasis, and drug resistance. Reactive oxygen species exhibit a dual role, promoting both cell proliferation and apoptosis, suggesting that carefully modulated redox therapies could be promising cancer treatments [2]. Beyond cancer, oxidative stress is a key contributor to cardiovascular diseases such as atherosclerosis, hypertension, and heart failure, causing significant cellular damage within the system [3]. Similarly, in diabetes mellitus, hyperglycemia-induced oxidative stress leads to pancreatic beta-cell dysfunction, insulin resistance, and severe complications like retinopathy, nephropathy, and neuropathy [6]. Targeting specific oxidative stress pathways offers novel insights for protecting heart health and managing diabetic complications [3, 6].

Oxidative stress is also intimately linked to inflammaging, which is chronic low-grade inflammation associated with aging. Disruptions in redox balance accelerate the aging process and contribute to age-related diseases. Maintaining cellular redox balance

is critical for healthy aging and mitigating chronic inflammatory conditions [4]. Furthermore, a vicious cycle exists between oxidative stress and inflammation in chronic diseases, where each process exacerbates the other, leading to persistent tissue damage [8]. Breaking this cycle through targeted interventions could offer new avenues for managing chronic inflammatory conditions like autoimmune disorders and metabolic syndrome [8]. Interestingly, the interactions between oxidative stress and the gut microbiota are crucial in disease pathogenesis. Dysbiosis can either contribute to or be a consequence of oxidative stress, impacting various systemic diseases. Understanding this bidirectional communication is essential for developing microbiota-targeted interventions [10].

Given the widespread impact of oxidative stress, therapeutic strategies are a major focus. Interventions include antioxidants, Nrf2 pathway modulators, and mitochondrial-targeted approaches [1, 5]. Moreover, the immense potential of natural antioxidants in preventing and treating diseases by mitigating oxidative damage has been explored. Leveraging dietary and natural sources of antioxidants could offer a promising approach to enhance health and combat pathology across various disease models [7]. This collective research underscores the importance of addressing oxidative stress for improved health outcomes.

Conclusion

Oxidative stress plays a central role in the development and progression of a wide array of human diseases. Research highlights its critical involvement in neurodegenerative conditions like Alzheimer's and Parkinson's, where reactive oxygen species directly damage neurons. The mechanisms linking redox imbalances to cancer initiation, growth, metastasis, and drug resistance are also extensively explored, with an emphasis on potential modulated redox therapies. The impact of oxidative stress extends to cardiovascular health, contributing to atherosclerosis, hypertension, and heart failure by damaging the cardiovascular system. It also significantly influences inflammaging, a chronic low-grade inflammation associated with aging, accelerating the aging process and age-related pathologies. Maintaining cellular redox balance, therefore, becomes key to promoting healthy aging. Mitochondrial dysfunction is frequently implicated, as mitochondria are major producers of reactive oxygen species, driving cellular damage across metabolic, neurodegenerative, and cardiovascular diseases. Oxidative stress is also a major factor in diabetes mellitus, where hyperglycemia-induced stress leads to beta-cell dysfunction, insulin resistance, and severe complications like retinopathy and nephropathy. Understanding these mechanisms is crucial for developing targeted antioxidant-based

therapies. Furthermore, the intricate interplay between oxidative stress and inflammation forms a vicious cycle in chronic diseases, exacerbating tissue damage. Breaking this cycle through targeted interventions could manage conditions like autoimmune disorders. The gut microbiota's role is also being investigated, revealing how dysbiosis can both cause and result from oxidative stress, impacting various systemic diseases. Lastly, the therapeutic potential of natural antioxidants in mitigating oxidative damage across various disease models is immense. Leveraging these dietary and natural sources offers a promising strategy to enhance health and combat pathology, underscoring the broad implications of oxidative stress research for novel treatment developments.

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