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Systemic Redox Imbalance in Chronic Disease: Are Antioxidants the Missing Link in Multiorgan Protection?

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ABSTRACT

Chronic diseases such as cardiovascular disease, diabetes mellitus, chronic kidney disease, neurodegenerative disorders, and chronic inflammatory conditions share a common pathological thread: systemic redox imbalance. This imbalance reflects a disruption between the generation of reactive oxygen and nitrogen species (ROS/RNS) and the body's antioxidant defenses, resulting in sustained oxidative stress that drives cellular dysfunction, inflammation, and tissue injury across multiple organs. While ROS act as essential mediators of physiological signaling, excessive ROS accumulation overwhelms endogenous antioxidant systems, contributing to mitochondrial dysfunction, endothelial damage, immune dysregulation, genomic instability, and metabolic disturbances. Recent research has increasingly focused on whether augmenting antioxidant capacity, either through dietary, pharmacological, or novel molecular approaches, can mitigate oxidative damage and confer multiorgan protection. Clinical and preclinical studies suggest that enhancing antioxidant defense may bolster tissue resilience and reduce complications in chronic disease. However, clinical trials have yielded mixed results, and unresolved questions remain regarding optimal antioxidant types, dosing strategies, bioavailability, timing of intervention, and organ-specific targeting. This review synthesizes current understanding of systemic redox imbalance in chronic disease, critically examines the role of antioxidants in modulating redox homeostasis, and explores future directions for integrating antioxidant strategies into multiorgan protection paradigms.

Keywords: oxidative stress, redox imbalance, chronic disease, antioxidants, multiorgan protection.

INTRODUCTION

Reactive oxygen species and reactive nitrogen species are continuously generated as byproducts of cellular metabolism, particularly during mitochondrial oxidative phosphorylation and enzymatic reactions [1]. Under healthy conditions, ROS/RNS serve vital roles in redox signaling, host defense, and regulation of cellular proliferation and apoptosis. A tightly regulated balance between pro-oxidant production and antioxidant defense mechanisms maintains redox homeostasis [2]. However, when ROS generation exceeds the capacity of antioxidant systems, a state of oxidative stress ensues, marked by oxidative damage to lipids, proteins, and DNA, and dysregulation of critical cellular pathways [3]. This systemic redox imbalance is increasingly recognized as a central feature in the pathophysiology of diverse chronic diseases spanning cardiovascular, metabolic, renal, neurodegenerative, and inflammatory disorders. Systemic oxidative stress contributes to chronic disease progression via multiple mechanisms, including activation of pro-inflammatory signaling, endothelial dysfunction, mitochondrial damage, and immune dysregulation [4]. It affects not only the primary diseased organ but also secondary targets through circulating ROS, altered redox signaling, and chronic inflammatory mediators. The cumulative nature of oxidative stress means that redox imbalance in one organ system can exacerbate dysfunction

in distant tissues, contributing to complex comorbidity patterns observed in aging populations and individuals with multimorbidity [5]. Antioxidant defenses encompass a wide network of enzymatic mechanisms, such as superoxide dismutase, catalase, and glutathione peroxidase, as well as non-enzymatic molecules including glutathione, vitamins C and E, carotenoids, and polyphenols [6]. These systems act synergistically to neutralize ROS, repair oxidative damage, and support redox signaling. In chronic disease states, however, antioxidant capacity is often overwhelmed or depleted, creating conditions in which oxidative stress accelerates disease progression and tissue injury [7]. Given the central role of oxidative stress in chronic disease pathogenesis, researchers and clinicians have long debated whether antioxidant interventions could provide multiorgan protective benefits [8]. While preclinical studies often show promising effects of antioxidants in restoring redox balance and reducing pathological features, translation to clinical success has been inconsistent, with varying outcomes across patient populations and disease contexts [9]. This review explores systemic redox imbalance in chronic disease and scrutinizes the potential of antioxidants as a missing link in comprehensive multiorgan protection.

2. Systemic Redox Imbalance in Chronic Disease

2.1 Oxidative Stress as a Common Landscape in Chronic Pathologies

Emerging evidence indicates that systemic redox imbalance is present across a spectrum of chronic diseases, not confined to isolated organs [10]. Elevated ROS levels and markers of oxidative damage are consistently observed in chronic kidney disease (CKD), cardiovascular disease, diabetes, chronic lung disease, neurodegenerative conditions, and aging-related disorders. In patients with CKD, systemic oxidative stress develops early in the disease course and worsens as renal function declines [11]. Increased lipid peroxidation and protein oxidation products, along with depletion of glutathione and alterations in antioxidant enzyme function, reflect a profound imbalance between pro-oxidants and antioxidants. These redox disturbances contribute to vascular dysfunction, inflammation, anemia, and progressive renal injury [12]. Cardiovascular disease is similarly linked to oxidative stress. Excess ROS promotes endothelial dysfunction by reducing nitric oxide bioavailability, enhancing pro-inflammatory gene expression, and facilitating atherogenic transformation of vascular cells [13]. Systemic redox imbalance thereby accelerates atherosclerosis, myocardial injury, and vascular stiffening. In metabolic diseases like diabetes, chronic hyperglycemia amplifies ROS production through glucose auto-oxidation, activation of the polyol pathway, and mitochondrial electron leakage, overwhelming antioxidant defenses and leading to widespread oxidative damage [14]. In neurodegenerative disorders such as Alzheimer's and Parkinson's disease, oxidative stress is implicated in mitochondrial dysfunction, protein misfolding, and synaptic degeneration, contributing to progressive neuronal loss [15]. This pervasive occurrence of oxidative imbalance across distinct diseases suggests that redox dysregulation is not merely a downstream effect of pathology but a core component of chronic disease biology, underlying shared mechanisms of inflammation, tissue injury, and organ failure.

3. Molecular and Cellular Mechanisms of Redox Imbalance

3.1 Sources of Reactive Oxygen and Nitrogen Species

ROS originate from endogenous metabolism and exogenous exposures. Mitochondria are primary ROS generators through electron transport chain activity [16]. Other sources include NADPH oxidases, xanthine oxidase, and uncoupled nitric oxide synthase. Additionally, environmental factors such as pollution, smoking, radiation, and certain drugs can enhance ROS production [17]. RNS, including peroxynitrite and nitric oxide derivatives, arise from interactions between superoxide and nitric oxide [18]. While low levels of these species regulate vasodilation and immune responses, chronic excess leads to nitrosative stress, protein nitration, and cellular dysfunction [19].

3.2 Antioxidant Defense Networks

Antioxidant systems counteract ROS/RNS through enzymatic and non-enzymatic mechanisms. Superoxide dismutase converts superoxide radicals to hydrogen peroxide, which is then detoxified by catalase and glutathione peroxidase [20]. Non-enzymatic molecules like glutathione, vitamins C and E, polyphenols, and carotenoids scavenge free radicals and regenerate oxidized antioxidants, maintaining redox balance. In chronic disease states, antioxidant enzyme activities and non-enzymatic antioxidant levels are often compromised, either through consumption by excess ROS or through diminished synthesis and recycling [21]. Reduced glutathione levels, for instance, are commonly observed in CKD and metabolic diseases, reflecting impaired cellular redox buffering capacity.

3.3 Redox Signaling and Pathological Pathways

ROS serve as signaling molecules that modulate transcription factors such as NF-κB, AP-1, and Nrf2[22]. Under oxidative stress, persistent activation of pro-inflammatory pathways drives chronic inflammation, while impaired

Nrf2 signaling reduces expression of antioxidant genes[23]. Dysregulated mitochondrial dynamics and apoptosis pathways further exacerbate tissue injury.

4. Systemic Consequences of Redox Imbalance Across Organ Systems

4.1 Cardiovascular and Endothelial Dysfunction

The cardiovascular system is particularly vulnerable to systemic redox imbalance [24]. Excessive ROS reduces nitric oxide bioavailability through direct chemical inactivation and by promoting endothelial nitric oxide synthase uncoupling. This impairs vasodilation and fosters endothelial dysfunction, a critical early event in atherosclerosis and hypertension [25]. Oxidative stress also enhances expression of adhesion molecules and chemokines, facilitating leukocyte recruitment to the vascular wall and promoting chronic vascular inflammation [26]. Lipid peroxidation further contributes to cardiovascular pathology by generating oxidized low-density lipoproteins, which are readily taken up by macrophages to form foam cells [27]. This process accelerates plaque formation and destabilization, increasing the risk of myocardial infarction and stroke [28]. Importantly, systemic oxidative stress originating from metabolic or renal disease can amplify cardiovascular risk, highlighting redox imbalance as a unifying mechanism linking multiorgan dysfunction [29].

4.2 Metabolic and Hepatic Effects

In metabolic disorders such as obesity and type 2 diabetes, redox imbalance plays a dual role as both a cause and consequence of insulin resistance. ROS interfere with insulin signaling by activating stress-responsive kinases that inhibit insulin receptor substrate function [30]. At the hepatic level, oxidative stress promotes lipid accumulation, mitochondrial dysfunction, and inflammatory signaling, contributing to non-alcoholic fatty liver disease and its progression to steatohepatitis. Systemic oxidative stress also alters adipokine secretion, favoring a pro-inflammatory endocrine profile that further exacerbates metabolic dysregulation [31]. These interactions illustrate how redox imbalance integrates metabolic, inflammatory, and endocrine pathways across organs.

4.3 Renal and Pulmonary Involvement

In chronic kidney disease, oxidative stress contributes to glomerular injury, tubular dysfunction, and progressive fibrosis [32]. Reduced renal clearance of oxidative byproducts further amplifies systemic redox imbalance, creating a feedback loop that accelerates cardiovascular and metabolic complications [33]. Similarly, in chronic lung diseases such as chronic obstructive pulmonary disease and pulmonary fibrosis, excessive ROS derived from inflammatory cells and environmental exposures damage alveolar structures and impair gas exchange, with systemic spillover effects on redox homeostasis.

4.4 Neurodegeneration and Cognitive Decline

The central nervous system is highly sensitive to oxidative damage due to its high oxygen consumption and lipid-rich environment [34]. Systemic redox imbalance contributes to neuroinflammation, mitochondrial dysfunction, and synaptic loss, which are hallmarks of neurodegenerative diseases. Oxidative modification of proteins and nucleic acids promotes misfolding and aggregation, processes implicated in Alzheimer's and Parkinson's diseases [35]. Importantly, systemic inflammation and oxidative stress originating from peripheral chronic diseases can exacerbate neurodegenerative processes, reinforcing the concept of multiorgan redox crosstalk.

5. Antioxidants as Potential Mediators of Multiorgan Protection

5.1 Endogenous Antioxidant Pathways

Endogenous antioxidant systems represent the first line of defense against systemic oxidative stress. Transcriptional regulation of antioxidant genes by nuclear factor erythroid 2–related factor 2 plays a central role in maintaining redox balance [36]. Activation of this pathway enhances expression of detoxifying enzymes, glutathione synthesis, and cellular stress resistance. In many chronic diseases, however, Nrf2 signaling is impaired, limiting adaptive antioxidant responses and predisposing tissues to injury [37]. Therapeutic strategies that restore or enhance endogenous antioxidant capacity may offer more physiologically relevant protection than exogenous supplementation alone. These approaches aim to reestablish redox homeostasis without disrupting essential ROS-mediated signaling.

5.2 Dietary and Nutritional Antioxidants

Diet-derived antioxidants, including vitamins C and E, carotenoids, flavonoids, and polyphenols, have long been investigated for their potential protective effects [38]. Epidemiological studies often associate diets rich in fruits, vegetables, and whole grains with reduced incidence of chronic diseases, suggesting a role for dietary antioxidants in long-term health maintenance. However, clinical trials of isolated antioxidant supplements have produced inconsistent results [39]. Factors such as poor bioavailability, inappropriate dosing, timing of intervention, and

disease stage may explain these discrepancies. Additionally, excessive antioxidant supplementation may blunt physiological redox signaling or exert pro-oxidant effects under certain conditions.

5.3 Pharmacological and Targeted Antioxidants

Advances in redox biology have prompted the development of targeted antioxidant therapies aimed at specific cellular compartments, such as mitochondria [40]. Mitochondria-targeted antioxidants seek to neutralize ROS at their primary source, thereby preserving mitochondrial function and reducing downstream damage. Early studies suggest potential benefits in models of cardiovascular, neurodegenerative, and metabolic diseases, although robust clinical evidence remains limited. Other pharmacological agents indirectly modulate redox balance by reducing ROS production or enhancing endogenous antioxidant pathways. These include inhibitors of NADPH oxidases, modulators of nitric oxide synthase coupling, and activators of Nrf2 signaling.

CONCLUSION

Systemic redox imbalance is a unifying pathological feature of chronic diseases affecting multiple organ systems. Excessive oxidative stress disrupts cellular signaling, immune regulation, and tissue integrity, driving disease progression and comorbidity. While antioxidants are not a universal remedy, growing evidence suggests that restoring redox balance particularly by enhancing endogenous antioxidant defenses, may represent a critical missing link in multiorgan protection. A nuanced understanding of redox biology, coupled with targeted and personalized interventions, holds promise for improving outcomes in chronic disease.

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