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Oxidative Stress as a Central Mediator of Chronic Inflammation: Mechanistic Insights and Therapeutic Targets

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ABSTRACT

Oxidative stress, characterized by an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defense system, plays a pivotal role in the initiation and perpetuation of chronic inflammation. ROS activate redox-sensitive transcription factors, modulate intracellular signaling cascades, and promote the release of pro-inflammatory mediators, leading to sustained immune activation and tissue damage. This review elucidates the molecular mechanisms linking oxidative stress to chronic inflammation, including mitochondrial dysfunction, NADPH oxidase activation, and impaired antioxidant defenses. We highlight the interplay between oxidative stress and immune cell function, the amplification of inflammatory signaling through NF-κB and NLRP3 inflammasome activation, and the resultant pathophysiological changes in chronic diseases such as atherosclerosis, rheumatoid arthritis, neurodegenerative disorders, and metabolic syndrome. Furthermore, we explore emerging therapeutic strategies targeting oxidative stress, including antioxidant therapies, Nrf2 activators, redox enzyme inhibitors, and lifestyle interventions. Understanding these mechanisms offers novel insights into the prevention and management of chronic inflammatory conditions.

Keywords: Oxidative stress, Chronic inflammation, Reactive oxygen species, NF-κB, Antioxidant therapy

INTRODUCTION

Chronic inflammation is a persistent and dysregulated immune response that remains active long after the initiating stimulus has been removed [1]. Unlike acute inflammation, which is protective, short-lived, and aimed at eliminating pathogens or repairing tissue damage, chronic inflammation is pathological and often results in progressive tissue injury, remodeling, and loss of function [2]. It is widely recognized as a major contributor to the pathogenesis of numerous chronic diseases, including cardiovascular disorders such as atherosclerosis and hypertension, autoimmune conditions like rheumatoid arthritis and systemic lupus erythematosus, neurodegenerative diseases including Alzheimer's and Parkinson's, and metabolic disorders such as type 2 diabetes, non-alcoholic fatty liver disease, and obesity-associated metabolic syndrome. Among the various factors driving this prolonged inflammatory state, oxidative stress has emerged as a central mediator that connects environmental, metabolic, and infectious insults to sustained inflammatory signaling. Oxidative stress occurs when the generation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) overwhelms the neutralizing capacity of endogenous antioxidant defense systems [3]. Under normal physiological conditions, ROS and RNS are produced at controlled levels and serve important functions in cell signaling, immune defense, and maintenance of vascular tone. Key antioxidant enzymes, including superoxide dismutase (SOD), catalase, and glutathione peroxidase, maintain a balance between oxidant production and elimination. When this balance is disrupted, excessive ROS and RNS levels activate redox-sensitive

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transcription factors such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), activator protein-1 (AP-1), and hypoxia-inducible factor-1 alpha (HIF-1α) [4]. These transcription factors promote the expression of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), along with chemokines and adhesion molecules that recruit and activate immune cells [5]. The interplay between oxidative stress and inflammation forms a self-perpetuating cycle. Activated immune cells, including macrophages, neutrophils, and microglia, produce high levels of ROS and RNS through mechanisms such as the respiratory burst [6]. While this is beneficial during acute infections for pathogen clearance, in chronic inflammation, persistent oxidative activity damages lipids, proteins, and nucleic acids. These oxidative modifications generate damage-associated molecular patterns (DAMPs) that are recognized by pattern recognition receptors (PRRs) such as Toll-like receptors (TLRs), further amplifying inflammatory responses. Beyond localized tissue injury, oxidative stress has systemic effects [7]. In the vasculature, it reduces nitric oxide bioavailability, leading to endothelial dysfunction and increased vascular permeability. In the nervous system, oxidative damage to neuronal membranes and synaptic proteins contributes to neuroinflammation and neuronal loss. In metabolic tissues, ROS interfere with insulin receptor signaling, promote adipocyte dysfunction, and sustain low-grade inflammation, which exacerbates metabolic syndrome [8].

Given its central role in multiple pathological processes, oxidative stress represents an attractive target for therapeutic intervention. A detailed understanding of its cellular sources and regulatory mechanisms is crucial for designing strategies to break the cycle of inflammation and oxidative damage [9].

2. Sources and Regulation of Reactive Oxygen Species in Inflammation

2.1 Mitochondrial ROS Production

Mitochondria are primary sources of ROS in most cells, producing superoxide anions as by-products of oxidative phosphorylation [10]. Electron leakage from complexes I and III of the electron transport chain results in the partial reduction of oxygen to form superoxide [11]. Under physiological conditions, mitochondrial ROS act as signaling molecules that regulate processes such as autophagy and immune cell activation. However, in inflammatory states, mitochondrial dysfunction increases electron leakage and ROS production. Excessive mitochondrial ROS activate redox-sensitive pathways, promote the release of pro-inflammatory cytokines, and trigger mitochondrial DNA damage, which itself acts as a DAMP to stimulate further immune activation [12].

2.2 NADPH Oxidase Activation

NADPH oxidases (NOX enzymes) are membrane-bound enzyme complexes dedicated to ROS generation [13]. In immune cells such as neutrophils and macrophages, NOX2 generates superoxide during the respiratory burst to destroy pathogens. However, chronic or inappropriate NOX activation results in excessive ROS production that damages host tissues. NOX-derived ROS also activate NF-κB and other inflammatory transcription factors, sustaining the inflammatory process [14].

2.3 Uncoupled Nitric Oxide Synthase (NOS)

Nitric oxide synthases (NOS) produce nitric oxide (NO), a molecule with vasodilatory, anti-inflammatory, and antimicrobial properties. Under oxidative stress, endothelial NOS (eNOS) can become uncoupled due to limited availability of its cofactor tetrahydrobiopterin (BH4) [15]. This uncoupling causes eNOS to produce superoxide instead of nitric oxide, contributing to oxidative stress, endothelial dysfunction, and vascular inflammation [16].

2.4 Peroxisomal and Endoplasmic Reticulum Stress

Peroxisomes contribute to ROS production during fatty acid β-oxidation, generating hydrogen peroxide as a by-product [17]. Although peroxisomes contain catalase to detoxify hydrogen peroxide, excessive fatty acid oxidation or impaired catalase activity can elevate ROS levels. Similarly, endoplasmic reticulum (ER) stress, often triggered by protein misfolding, leads to the activation of the unfolded protein response (UPR) [18]. Persistent ER stress increases ROS production, disrupts calcium homeostasis, and activates inflammatory pathways such as the NLRP3 inflammasome. The combined oxidative and inflammatory stress from peroxisomal and ER dysfunction amplifies tissue injury in chronic diseases [19].

3. Molecular Mechanisms Linking Oxidative Stress to Chronic Inflammation

3.1 Activation of Redox-Sensitive Transcription Factors

Reactive oxygen species (ROS) act as critical signaling molecules that modulate the activity of redox-sensitive transcription factors, including nuclear factor-kappa B (NF-κB), activator protein-1 (AP-1), and hypoxia-inducible factor-1α (HIF-1α). NF-κB, a master regulator of inflammation, is normally sequestered in the cytoplasm by inhibitor proteins (IκBs) [20]. ROS promote IκB phosphorylation and degradation, enabling NF-κB to translocate into the nucleus and induce the transcription of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, as well

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as adhesion molecules that recruit immune cells to inflamed tissues. Similarly, AP-1 is activated through ROS-dependent mitogen-activated protein kinase (MAPK) pathways, enhancing the expression of genes involved in inflammation and tissue remodeling. HIF-1α, which is stabilized under oxidative and hypoxic conditions, drives glycolytic metabolism in immune cells, supporting sustained inflammatory responses [21].

3.2 NLRP3 Inflammasome Activation

The NOD-like receptor pyrin domain-containing 3 (NLRP3) inflammasome is a cytosolic multiprotein complex that plays a pivotal role in innate immunity [22]. ROS act as upstream activators of NLRP3 by promoting mitochondrial dysfunction, potassium efflux, and lysosomal damage. This activation triggers the recruitment and activation of caspase-1, which cleaves pro-IL-1β and pro-IL-18 into their mature, biologically active forms. These cytokines are potent amplifiers of inflammation, promoting fever, leukocyte recruitment, and tissue injury. Persistent ROS-driven inflammasome activation has been implicated in the pathogenesis of chronic diseases such as gout, type 2 diabetes, and atherosclerosis [23].

3.3 Oxidative Modification of Biomolecules

Excessive ROS production results in oxidative damage to lipids, proteins, and nucleic acids [24]. Lipid peroxidation yields reactive aldehydes such as malondialdehyde (MDA) and 4-hydroxynonenal (4-HNE), which form adducts with proteins, altering their function and immunogenicity. Oxidized proteins can lose enzymatic activity or become aggregation-prone, contributing to tissue dysfunction. Oxidative damage to DNA generates lesions such as 8-hydroxy-2'-deoxyguanosine (8-OHdG), which can lead to mutations and genomic instability. Many of these oxidized macromolecules act as damage-associated molecular patterns (DAMPs), engaging pattern recognition receptors (PRRs) on immune cells and perpetuating inflammatory signaling [25].

3.4 Crosstalk Between Oxidative Stress and Immune Cells

Oxidative stress significantly influences immune cell behavior. In macrophages, ROS regulate polarization between the pro-inflammatory M1 phenotype and the anti-inflammatory M2 phenotype, often skewing the balance toward M1 dominance in chronic inflammation [26]. In T cells, ROS modulate activation thresholds, proliferation, and cytokine production, with high ROS levels often favoring pro-inflammatory Th1 and Th17 responses over regulatory T cell (Treg) functions. Neutrophils, upon activation, release neutrophil extracellular traps (NETs), which are ROS-dependent chromatin structures that trap pathogens but also damage surrounding tissues and exacerbate inflammation. This oxidative–immune interplay sustains the inflammatory microenvironment, making resolution of inflammation more difficult [27].

4. Oxidative Stress and Chronic Inflammatory Diseases

4.1 Cardiovascular diseases

In atherosclerosis, oxidative stress promotes the oxidative modification of low-density lipoprotein (LDL) into oxidized LDL (oxLDL), which is avidly taken up by macrophages via scavenger receptors to form foam cells [28]. This process contributes to the growth of atherosclerotic plaques. ROS also impair endothelial nitric oxide (NO) bioavailability, leading to endothelial dysfunction and increased vascular tone. In advanced disease, oxidative stress destabilizes plaques by activating matrix metalloproteinases (MMPs) that degrade the fibrous cap, increasing the risk of plaque rupture and thrombosis [29].

4.2 Rheumatoid Arthritis

In rheumatoid arthritis, excessive ROS production within the inflamed synovium accelerates joint destruction by stimulating the production of MMPs, which degrade cartilage, and by enhancing osteoclast differentiation, which erodes bone. ROS also promote the activation of synovial fibroblasts and immune cell infiltration, leading to chronic pannus formation and sustained joint inflammation [30].

4.3 Neurodegenerative Disorders

In Alzheimer's disease, oxidative stress promotes amyloid-beta aggregation, tau hyperphosphorylation, and synaptic dysfunction. In Parkinson's disease, ROS derived from mitochondrial complex I dysfunction and dopamine metabolism contribute to dopaminergic neuron loss in the substantia nigra. In both cases, oxidative stress activates microglia, which release further ROS and pro-inflammatory cytokines, creating a self-perpetuating cycle of neuroinflammation and neurodegeneration [31].

4.4 Metabolic Syndrome and Diabetes

In metabolic syndrome and type 2 diabetes, hyperglycemia and elevated free fatty acids increase mitochondrial ROS production and activate NADPH oxidases [32]. This oxidative stress triggers inflammatory pathways in adipocytes, vascular endothelial cells, and pancreatic β-cells, leading to insulin resistance, vascular inflammation, and β-cell apoptosis [33].

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5. Therapeutic Strategies Targeting Oxidative Stress in Chronic Inflammation

5.1 Antioxidant Therapy

Antioxidant therapy seeks to neutralize excessive reactive oxygen species (ROS) and restore redox balance. Dietary antioxidants such as vitamins C and E, polyphenols (e.g., resveratrol, quercetin), and carotenoids (e.g., β-carotene, lycopene) scavenge free radicals and modulate inflammatory signaling. Endogenous antioxidant enhancers like N-acetylcysteine (NAC) serve as precursors for glutathione synthesis, reinforcing the body's primary defense system against oxidative injury [34].

5.2 Nrf2 Pathway Activation

The transcription factor nuclear factor erythroid 2–related factor 2 (Nrf2) orchestrates cellular antioxidant responses by upregulating genes encoding detoxifying enzymes, heme oxygenase-1, and glutathione-related proteins. Natural compounds such as curcumin, sulforaphane, and epigallocatechin gallate (EGCG) have been shown to activate Nrf2, while synthetic activators are being developed for clinical use to mitigate inflammation-related oxidative damage [35].

5.3 NOX Inhibitors

NADPH oxidases (NOX enzymes) are major sources of ROS in inflammatory states. Selective pharmacological NOX inhibitors aim to suppress pathological ROS generation while preserving its physiological role in immune defense. These agents hold promise in conditions such as cardiovascular disease and neuroinflammation [36].

5.4 Mitochondria-Targeted Antioxidants

Since mitochondrial dysfunction is a key source of ROS in chronic inflammation, specialized antioxidants such as MitoQ and SkQ1 are designed to accumulate within mitochondria. These compounds protect mitochondrial integrity, improve energy metabolism, and dampen ROS-driven inflammatory signaling [37].

5.5 Lifestyle Interventions

Non-pharmacological strategies such as adopting antioxidant-rich diets, engaging in regular moderate exercise, managing psychological stress, and avoiding environmental pollutants enhance redox homeostasis and contribute to long-term inflammation control [38].

CONCLUSION

Oxidative stress plays a pivotal role in sustaining and amplifying chronic inflammation by triggering redox-sensitive transcription factors, such as NF-κB and AP-1, promoting dysregulated immune cell activation, and accelerating tissue damage. This persistent oxidative–inflammatory cycle underlies the pathogenesis of numerous chronic diseases, including cardiovascular disorders, neurodegenerative conditions, metabolic syndrome, and autoimmune pathologies. Therapeutic strategies that directly reduce excessive reactive oxygen species or enhance endogenous antioxidant capacity, coupled with approaches that restore mitochondrial function, offer significant potential for disease modification. Equally, lifestyle-based interventions—nutritional optimization, regular physical activity, stress management, and environmental toxin avoidance—represent accessible, cost-effective means of improving redox balance. Moving forward, disease-specific research into redox biology and biomarker-driven patient stratification will be crucial for developing precision antioxidant therapies that achieve meaningful clinical outcomes while minimizing adverse effects. By targeting oxidative stress, it may be possible to disrupt the vicious cycle of inflammation and restore tissue homeostasis.

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