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Dietary and Endogenous Antioxidants in Immune Homeostasis: A Translational Perspective

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ABSTRACT

Immune homeostasis relies on a dynamic equilibrium between oxidative stress, redox signaling, and the antioxidant systems that buffer reactive oxygen and nitrogen species generated during normal metabolism and inflammatory responses. Both endogenous antioxidants-such as glutathione, thioredoxin, uric acid, and enzymatic systems-and dietary antioxidants derived from fruits, vegetables, spices, and phytochemicals collectively maintain this balance. Disruption of redox homeostasis leads to immune dysfunction characterized by exaggerated inflammation, impaired pathogen clearance, altered lymphocyte differentiation, and progression of chronic metabolic and inflammatory diseases. This review synthesizes current mechanistic insights and emerging translational evidence on how dietary and endogenous antioxidants regulate innate and adaptive immunity through redox-sensitive pathways, including Nrf2, NF-κB, HIF-1α, and mitochondrial signaling. It further highlights clinical implications, therapeutic opportunities, and remaining research gaps required to bridge experimental knowledge with real-world clinical application. Understanding the interplay between antioxidant systems and immune regulation offers an evidence-based foundation for developing targeted dietary, pharmacological, and lifestyle interventions to restore immune balance in diverse pathological contexts.

Keywords: Antioxidants, Redox Homeostasis, Immune Regulation, Dietary Phytochemicals, Oxidative Stress

INTRODUCTION

Oxidative stress and immune function are deeply intertwined, forming a continuous feedback loop that influences nearly every phase of immune activity [1-5]. During immune activation, phagocytes and other leukocytes generate reactive oxygen species (ROS) and reactive nitrogen species (RNS) as essential antimicrobial agents and as intracellular signaling molecules that regulate gene expression, cytokine production, and cell differentiation [6-10]. Although these oxidants play indispensable physiological roles, their excess can rapidly shift the immune environment toward pathology. Elevated ROS levels promote lipid peroxidation, protein carbonylation, DNA strand breaks, mitochondrial dysfunction, and dysregulated inflammatory signaling [11-16]. These changes compromise cellular integrity and lead to chronic activation of inflammatory pathways, ultimately disrupting the delicate balance required for effective immunity [17-23]. To counter these potentially damaging effects, the body relies on a sophisticated array of endogenous antioxidant systems and complementary dietary antioxidants. Immune homeostasis, defined as the coordinated equilibrium between immune activation, tolerance, and resolution of inflammation, depends on the efficiency and responsiveness of these antioxidant defenses [24-29]. When antioxidant capacity is insufficient or overwhelmed-due to infection, metabolic stress, nutrient deficiencies, aging, or environmental exposures-redox imbalance drives the onset and progression of a wide spectrum of diseases [30-36]. These include autoimmune disorders, persistent infections, cardiometabolic diseases, certain cancers, and neuroinflammatory conditions [37-44]. Conversely, enhancing antioxidant status through endogenous

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upregulation or dietary intake can modulate immune signaling pathways, improve host resilience to pathogens, reduce tissue damage, and restore homeostatic balance [45-54].

A growing body of evidence demonstrates that redox regulation is not simply a protective mechanism against oxidative injury but also a fundamental determinant of immune cell development, differentiation, metabolism, and effector function [55-60]. Understanding how endogenous and dietary antioxidants interact with immune pathways provides a powerful framework for developing nutritional and pharmacological interventions that promote health and prevent chronic disease [61-67]. This review integrates mechanistic, nutritional, and translational perspectives to highlight the roles of endogenous antioxidant systems in immune regulation and their broader implications for clinical practice and public health.

2. Endogenous Antioxidant Systems in Immune Regulation

2.1 Glutathione System

Glutathione (GSH) is the most abundant intracellular antioxidant and a central regulator of redox-sensitive immune processes [68-74]. The reduced form of glutathione directly neutralizes ROS and supports the activity of glutathione peroxidases, which detoxify hydrogen peroxide and lipid peroxides. The ratio of reduced to oxidized glutathione is a widely used indicator of cellular redox balance and immune competence [75-80]. GSH controls T cell activation by regulating redox-sensitive transcription factors, supports antigen presentation by protecting dendritic cells from oxidative injury, influences macrophage polarization toward the anti-inflammatory M2 pathway, and safeguards lymphocytes from activation-induced cell death. Deficiency in GSH impairs lymphocyte proliferation, reduces cytokine production, and increases susceptibility to infections.

2.2 Thioredoxin and Peroxiredoxin Systems

Thioredoxin and peroxiredoxins form a complementary antioxidant network that maintains the redox state of protein thiols [81-84]. These systems play key roles in DNA synthesis during lymphocyte expansion, regulation of inflammasome activation, and modulation of cytokine signaling via reversible oxidation of cysteine residues [85-90]. Extracellular thioredoxin also functions as a cytokine-like molecule capable of influencing leukocyte recruitment and chemotaxis, linking redox buffering to immune communication.

2.3 Enzymatic Antioxidants

Superoxide dismutase, catalase, and glutathione peroxidases constitute the core enzymatic antioxidant machinery [91-96]. Together, they detoxify superoxide radicals and hydrogen peroxide, protect immune cells from self-inflicted oxidative damage during respiratory bursts, and preserve the integrity of epithelial and endothelial barriers essential for innate defense [91-96].

2.4 Endogenous Small-Molecule Antioxidants

Small molecules such as uric acid, bilirubin, lipoic acid, and coenzyme Q10 contribute significantly to systemic antioxidant capacity [17]. These molecules possess strong radical-scavenging abilities and modulate inflammation, mitochondrial function, and vascular homeostasis [18]. Despite their modest size, their physiological influence on redox and immune regulation is substantial, supporting the broader antioxidant network that sustains immune homeostasis.

3. Dietary Antioxidants and Their Impact on Immunity

Dietary antioxidants comprise a wide range of nutrients and bioactive compounds, including vitamins such as C, E, and A, trace minerals like selenium and zinc, polyphenols, carotenoids, and various plant-derived phytochemicals [19]. Together, these molecules fortify endogenous antioxidant systems and exert direct regulatory effects on immune pathways [20]. Their influence extends beyond simple free radical scavenging; many act as signaling modulators that shape cellular differentiation, cytokine release, membrane stability, and gene expression patterns involved in both innate and adaptive immunity [21]. Adequate intake of these antioxidants is therefore essential for maintaining immune resilience, preventing chronic inflammation, and supporting recovery from infection or tissue injury.

3.1 Vitamin C (Ascorbate)

Vitamin C is one of the most important water-soluble antioxidants and accumulates in leukocytes at concentrations far higher than in plasma [22]. It enhances innate immunity by improving neutrophil chemotaxis, promoting the formation of reactive oxygen species required for microbial killing, and facilitating clearance of spent neutrophils from inflammatory sites [23]. Vitamin C also regenerates oxidized vitamin E and sustains glutathione levels, amplifying systemic antioxidant capacity. By modulating cytokine production and dampening excessive inflammatory responses, it contributes to balanced immunity. Deficiency leads to impaired epithelial barrier function, reduced lymphocyte activation, and an increased susceptibility to infections.

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3.2 Vitamin E (Tocopherols and Tocotrienols)

Vitamin E is a lipid-soluble antioxidant that protects cellular membranes from peroxidation [24]. It improves T cell responsiveness by maintaining membrane fluidity and enhancing signal transduction. Vitamin E supplementation has been shown to reduce age-related immune decline, partly by revitalizing T cell–mediated responses [25]. Its regulatory effects on prostaglandin synthesis and inflammatory signaling support a more controlled inflammatory response and improved immune coordination.

3.3 Vitamin A and Carotenoids

Vitamin A and its derivatives play central roles in mucosal immunity, promoting epithelial integrity, supporting IgA secretion, and guiding the differentiation of lymphocyte subsets [26]. Retinoids influence the balance between Th1, Th2, and regulatory T cells, thereby shaping adaptive immunity [27]. Carotenoids such as beta-carotene enhance natural killer cell activity, improve antioxidant capacity within immune cells, and contribute to resistance against oxidative stress encountered during immune activation.

3.4 Selenium

Selenium is indispensable for the activity of glutathione peroxidases and thioredoxin reductases, enzymes crucial for maintaining cellular redox stability [28]. Selenium deficiency compromises lymphocyte proliferation, weakens antiviral defenses, and heightens vulnerability to oxidative damage during inflammation [29]. Optimal selenium intake enhances cytotoxic T cell and NK cell activity, supporting both innate and adaptive immune responses.

3.5 Polyphenols

Polyphenols, widely found in fruits, vegetables, teas, and spices, are among the most potent dietary modulators of immune function [30]. Compounds such as quercetin, catechins, curcumin, and resveratrol operate through multiple mechanisms. They neutralize ROS, activate the Nrf2 pathway to induce endogenous antioxidant genes, inhibit NF-κB-driven inflammatory cytokine expression, and promote anti-inflammatory macrophage phenotypes [31]. Many polyphenols also facilitate the development of regulatory T cells, enhancing immune tolerance and helping to prevent chronic inflammatory disorders.

3.6 Dietary Fiber and Microbiota-Derived Antioxidants

Dietary fiber indirectly contributes to antioxidant and immune regulation through fermentation by gut microbiota [32]. The resulting short-chain fatty acids, especially butyrate, serve as metabolic substrates for intestinal cells, strengthen epithelial barrier function, modulate inflammatory gene expression, and act as epigenetic regulators [33]. Through these mechanisms, fiber-derived metabolites enhance systemic antioxidant defenses and sustain optimal immune homeostasis.

5. Antioxidant Regulation of Innate Immunity

Innate immune cells are particularly sensitive to redox conditions. In macrophages, oxidative stress favors pro-inflammatory M1 polarization, whereas reducing environments promote M2 phenotypes involved in tissue repair and resolution [34]. Dietary antioxidants therefore support balanced macrophage responses.

Neutrophils rely on ROS for antimicrobial activity, yet excessive generation damages host tissues. Antioxidants improve the efficiency of microbial killing, protect neutrophils from premature apoptosis, and limit collateral tissue injury.

Natural killer cells require an optimal redox environment for cytotoxicity and cytokine release. Nutrients such as vitamins C and E, as well as polyphenols like quercetin and resveratrol, enhance NK cell performance.

Dendritic cells also depend on controlled redox signaling for antigen processing and T cell priming. Elevated ROS impair their function, while antioxidants help maintain antigen presentation and promote regulatory immune responses.

6. Antioxidant Regulation of Adaptive Immunity

6.1 T Lymphocytes

T lymphocyte function is tightly coupled to intracellular redox status, as activation, differentiation, and survival all depend on controlled ROS signaling. Low to moderate ROS levels act as second messengers that facilitate TCR signaling and metabolic reprogramming, but excessive ROS disrupt these processes [35]. High oxidative burden inhibits proliferation by blocking key kinase cascades, reduces IL-2 and other cytokines, and can drive T cells toward anergy or apoptosis through mitochondrial damage. Adequate antioxidant buffering preserves the signaling window necessary for effective T cell responses. Dietary and endogenous antioxidants promote balanced Th1/Th2 differentiation, support the expansion and suppressive function of regulatory T cells, and counteract the differentiation of pro-inflammatory Th17 cells [36]. Redox homeostasis also enhances the formation and longevity of memory T cells by sustaining mitochondrial fitness and preventing oxidative exhaustion, improving long-term immune protection.

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6.2 B Lymphocytes

B cell maturation, germinal-center reactions, and antibody class switching rely on precise redox control. Antioxidants help maintain the integrity of B cell receptor signaling and support the survival of activated B cells [37]. Polyphenols and vitamins A and E are particularly influential, as they enhance B cell responsiveness, improve somatic hypermutation efficiency, and promote the generation of high-affinity antibodies. By stabilizing cellular redox balance, antioxidants ultimately reinforce humoral immunity and contribute to more robust, durable antibody-mediated protection.

CONCLUSION

Antioxidants-both endogenous and dietary-play indispensable roles in maintaining immune homeostasis through regulation of redox-sensitive pathways, modulation of immune cell function, and preservation of tissue integrity. Their influence spans innate and adaptive immunity, metabolic and inflammatory signaling, and resilience against infections and chronic disease. Translational evidence supports targeted use of antioxidants as complementary strategies to enhance immune function, reduce inflammation, and improve health outcomes. Continued integrative research bridging molecular biology, nutrition, and clinical medicine will advance the development of precision antioxidant therapies tailored to individual redox and immunological profiles.

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