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Antioxidant Phytochemicals and Immune Modulation in HIV/AIDS: Mechanisms, Evidence, and Clinical Relevance

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ABSTRACT

Human Immunodeficiency Virus (HIV) infection is characterized by persistent immune activation, chronic inflammation, and oxidative stress, which collectively accelerate CD4+ T-cell depletion and contribute to progressive immune dysfunction. Oxidative stress, arising from excessive production of reactive oxygen species and impaired antioxidant defenses, plays a pivotal role in enhancing viral replication, disrupting cellular immunity, and driving the onset of comorbidities such as cardiovascular disease, neurocognitive impairment, and metabolic disorders in people living with HIV/AIDS (PLWHA). Despite the success of antiretroviral therapy (ART) in suppressing viral replication and extending lifespan, ART itself can exacerbate mitochondrial dysfunction and oxidative injury, underscoring the need for adjunctive therapeutic strategies. Antioxidant phytochemicals-bioactive compounds naturally present in fruits, vegetables, herbs, and spices-have attracted attention due to their potent free radical scavenging capacity, ability to restore redox balance, and immunomodulatory effects. Compounds such as flavonoids, carotenoids, and polyphenols have been shown to modulate key signaling pathways, reduce pro-inflammatory cytokine production, and protect immune cells from oxidative damage. This review examines the mechanistic basis of oxidative stress in HIV pathogenesis, summarizes experimental and clinical evidence on the role of phytochemicals in mitigating redox imbalance, and discusses their clinical relevance. Finally, it highlights research gaps and future directions for incorporating antioxidant phytochemicals into comprehensive HIV management strategies.

Keywords: Antioxidant phytochemicals, Oxidative stress, HIV/AIDS, Immune modulation, Reactive oxygen species (ROS), Polyphenols and flavonoids, Redox homeostasis

INTRODUCTION

Human Immunodeficiency Virus (HIV) infection and acquired immunodeficiency syndrome (AIDS) remain among the most pressing global health challenges, particularly in sub-Saharan Africa, where the disease burden is highest [1-4]. Despite the remarkable success of antiretroviral therapy (ART) in reducing viral load, improving survival, and transforming HIV from a fatal illness into a manageable chronic condition, multiple challenges persist [5-8]. These include the emergence of drug-resistant viral strains, incomplete immune reconstitution in many patients, and the rising incidence of non-AIDS comorbidities such as cardiovascular disease, metabolic disorders, and neurocognitive decline. Understanding the biological mechanisms that underlie these persistent complications is crucial for developing adjunctive interventions that go beyond viral suppression [9-11]. One central but often underappreciated factor in the pathophysiology of HIV is oxidative stress. Oxidative stress refers to an imbalance between the generation of reactive oxygen species (ROS) and the capacity of endogenous antioxidant defenses to neutralize them [12-15]. Excessive ROS production contributes to tissue damage, cellular dysfunction, and chronic inflammation [16-18]. In the context of HIV, oxidative stress both drives and results from immune dysregulation, thereby accelerating disease progression. It not only promotes viral replication but also amplifies immune cell apoptosis and contributes to systemic immune activation [19-23]. Furthermore, ART itself, while life-saving, can inadvertently intensify oxidative damage through mitochondrial toxicity, highlighting the dual burden of virus- and therapy-induced oxidative stress [24-27].

Growing evidence suggests that modulation of oxidative stress could improve immune function and clinical outcomes in people living with HIV/AIDS (PLWHA) [28-30]. Among potential interventions, antioxidant phytochemicals have received increasing scientific attention. These naturally occurring compounds, abundant in

fruits, vegetables, herbs, and spices, include polyphenols, flavonoids, carotenoids, terpenoids, and alkaloids. Their biological activities extend beyond simple free radical scavenging to include modulation of signaling pathways, regulation of gene expression, and restoration of redox balance [31-35]. Importantly, several phytochemicals also possess immunomodulatory and anti-inflammatory properties, making them attractive candidates for integrative HIV management strategies [36-39]. This review aims to synthesize current knowledge on the role of oxidative stress in HIV infection, examine how phytochemicals influence immune function in this context, and evaluate the clinical evidence supporting their therapeutic relevance [40-45]. By highlighting both mechanistic insights and translational potential, the discussion underscores the importance of antioxidant phytochemicals as adjuncts in HIV care, particularly in regions where dietary sources of these compounds are abundant but underutilized [46-49].

Oxidative Stress and Immune Dysfunction in HIV/AIDS

Mechanisms of Oxidative Stress in HIV

Oxidative stress arises in HIV infection through multiple, interconnected mechanisms. Viral replication itself is associated with increased ROS generation in infected CD4+ T cells and macrophages [50-55]. During active replication, viral proteins such as Tat and gp120 stimulate oxidative pathways, leading to excessive production of superoxide anions, hydrogen peroxide, and hydroxyl radicals [56-59]. These molecules overwhelm endogenous antioxidant defenses, including superoxide dismutase, catalase, and glutathione peroxidase.

ART, although indispensable for viral suppression, paradoxically contributes to oxidative stress. Nucleoside reverse transcriptase inhibitors (NRTIs) and protease inhibitors (PIs) are known to induce mitochondrial toxicity by inhibiting mitochondrial DNA polymerase gamma, resulting in impaired electron transport chain activity and enhanced leakage of electrons that form ROS [60-65]. This ART-induced mitochondrial dysfunction adds to the oxidative burden already present in HIV-infected individuals.

Inflammatory mediators further amplify oxidative damage. Pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) are elevated during chronic HIV infection [66-69]. These cytokines activate nicotinamide adenine dinucleotide phosphate (NADPH) oxidase and other ROS-producing enzymes, creating a vicious cycle in which oxidative stress sustains inflammation and inflammation perpetuates oxidative stress [70]. The resulting feedback loop accelerates immune exhaustion and tissue damage.

Impact on the Immune System

The immune system is particularly vulnerable to redox imbalance in HIV. Excessive ROS impairs antigen presentation by dendritic cells and macrophages, reducing their ability to mount effective adaptive responses [71-73]. In T lymphocytes, ROS trigger mitochondrial damage and activate pro-apoptotic signaling pathways, leading to premature death of CD4+ cells the central targets of HIV infection [14]. The loss of CD^{4+} T cells not only weakens adaptive immunity but also disrupts coordination between innate and adaptive arms of the immune system [74-75].

Oxidative stress also plays a direct role in enhancing HIV transcription and replication. Activation of redox-sensitive transcription factors such as nuclear factor kappa B (NF-κB) increases transcription of HIV proviral DNA integrated into host genomes [76-78]. This creates a feed-forward mechanism whereby oxidative stress fuels viral propagation, which in turn generates more oxidative stress through viral protein activity and immune activation.

Chronic redox imbalance contributes to the persistent immune activation that characterizes HIV pathogenesis, even in individuals on effective ART [79-80]. Continuous activation of T cells and macrophages drives immune exhaustion, increases susceptibility to opportunistic infections, and predisposes PLWHA to non-AIDS comorbidities [67-70]. Over time, this unresolved oxidative and inflammatory milieu undermines long-term immune reconstitution, limiting the full benefits of ART. In summary, oxidative stress in HIV/AIDS is both a cause and a consequence of immune dysfunction. It accelerates CD4+ T-cell depletion, enhances viral replication, and perpetuates chronic inflammation [71-73]. Addressing oxidative stress, therefore, represents a promising adjunctive strategy to improve immune health and clinical outcomes in HIV management.

Antioxidant Phytochemicals: Classification and Mechanisms

Antioxidant phytochemicals represent a diverse group of bioactive compounds derived from plants that exert protective effects against oxidative stress and immune dysfunction [74-78]. Their mechanisms of action extend beyond simple neutralization of free radicals to include modulation of intracellular signaling pathways, regulation of gene expression, and preservation of cellular integrity [79]. In the context of HIV/AIDS, where oxidative stress and immune dysregulation fuel disease progression, these compounds offer promising therapeutic potential.

Polyphenols

Polyphenols such as resveratrol, quercetin, and catechins are widely studied for their multifunctional biological effects. One of their most relevant mechanisms in HIV is the inhibition of nuclear factor kappa B (NF-κB) activation, a transcription factor that promotes HIV proviral DNA expression [80]. By suppressing NF-κB signaling, polyphenols indirectly reduce viral replication [60]. Additionally, their potent free radical scavenging activity protects immune cells from ROS-induced apoptosis, thereby preserving CD4+ T-cell survival [24]. Resveratrol

also activates sirtuin-1 pathways, which enhance cellular resistance to oxidative injury and regulate inflammatory responses, adding another layer of immune protection [25].

Carotenoids

Carotenoids, including beta-carotene and lycopene, are fat-soluble pigments with strong antioxidant properties. Their ability to stabilize cellular membranes protects lymphocytes and macrophages from lipid peroxidation, a common consequence of chronic oxidative stress in HIV [26]. Carotenoids also play an immunostimulatory role by supporting natural killer (NK) cell activity, enhancing antibody production, and promoting lymphocyte proliferation [27]. Beta-carotene, a precursor of vitamin A, contributes to mucosal immunity, which is often compromised in HIV-infected individuals [28]. These effects collectively enhance host defense against both HIV and opportunistic infections.

Flavonoids

Flavonoids, such as curcumin, apigenin, and kaempferol, exert broad immunomodulatory functions. They downregulate pro-inflammatory cytokines like TNF-α and IL-6, which are elevated in HIV infection and contribute to chronic immune activation [29]. Flavonoids also improve mitochondrial function, counteracting the oxidative damage induced by ART [30]. Curcumin, for example, has been shown to regulate multiple signaling pathways including mitogen-activated protein kinases (MAPKs) and Janus kinase/signal transducers and activators of transcription (JAK/STAT), leading to reduced inflammation and enhanced immune regulation [31]. Through these mechanisms, flavonoids not only attenuate oxidative stress but also support long-term immune reconstitution.

Alkaloids and Terpenoids

Alkaloids and terpenoids constitute another class of phytochemicals with therapeutic potential. Certain alkaloids exhibit direct antiviral activity by interfering with viral entry and replication [32]. Terpenoids, on the other hand, are known to enhance the functional activity of macrophages and dendritic cells, thereby strengthening innate immune responses [33]. Both classes of compounds also possess antioxidant properties, reducing oxidative injury in immune cells. Although less studied in the context of HIV compared to polyphenols or flavonoids, their dual role in antiviral defense and redox regulation makes them valuable candidates for further investigation. In summary, phytochemicals act through overlapping but distinct mechanisms, including inhibition of viral transcription, protection of immune cells from oxidative damage, modulation of inflammatory pathways, and enhancement of innate and adaptive immunity. Their multifunctionality highlights their potential as complementary strategies alongside ART in the management of HIV/AIDS.

CONCLUSION

Oxidative stress is a critical driver of immune dysfunction and disease progression in HIV/AIDS. Antioxidant phytochemicals offer promising avenues for restoring immune balance, reducing viral replication, and preventing comorbidities. While preclinical and preliminary clinical evidence is encouraging, robust trials are essential to translate these findings into practice. Integrating phytochemicals into HIV management could complement ART and contribute to holistic, patient-centered care.

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