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# Implications of nutritional intervention in hiv disease progression and oxidative stress management: A comprehensive review

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## Abstract

HIV infection induces chronic immune activation, oxidative stress, and nutrient depletion, collectively influencing disease progression. Although antiretroviral therapy (ART) improves survival, it may exacerbate oxidative damage through mitochondrial toxicity. This narrative review synthesizes evidence from 1990 to 2024 on relationships among HIV infection, ART-induced oxidative stress, antioxidant status, and nutrition, with emphasis on sub-Saharan Africa. Findings indicate that HIV and ART increase reactive oxygen species, weaken antioxidant defenses, and disrupt nutrient metabolism. Micronutrient deficiencies, particularly vitamins A, C, and E, selenium, and zinc, are common among people living with HIV and associate with accelerated CD4 decline and increased opportunistic infections. Supplementation studies suggest that improving antioxidant and micronutrient status may reduce oxidative stress, enhance immune function, and improve ART tolerability, although outcomes vary by baseline nutrition, ART regimen, and adherence. Integrating targeted nutritional interventions into HIV care may mitigate oxidative damage and improve long-term outcomes, but further trials are required.

***Keywords:*** *HIV, Oxidative stress, Antiretroviral therapy, Nutrition-antioxidants*



## 1. Introduction

Human Immunodeficiency Virus (HIV) remains one of the most significant public health challenges globally,

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with an estimated 39.0 million people living with HIV at the end of 2022, two-thirds of whom reside in sub-Saharan Africa (World Health Organization, 2022). Since the start of the epidemic, approximately 40.4 million people have died from HIV-related causes. In Nigeria, which bears the second-largest HIV burden worldwide, about 1.9 million people were living with HIV in 2022 (UNAIDS, 2022). Six states, including Kaduna State, account for over 40% of Nigeria's HIV cases, with a prevalence of 1.1% (Joshua *et al.*, 2021).

Although antiretroviral therapy (ART) has transformed HIV into a manageable chronic condition, long-term complications persist due to ongoing immune activation, inflammation, and oxidative stress. Oxidative stress an imbalance between Reactive Oxygen Species (ROS) production and antioxidant defenses plays a pivotal role in HIV pathogenesis. It contributes to immune cell apoptosis, reduced CD4+ T cell counts, and progression to Acquired Immunodeficiency Syndrome (AIDS) (Allard *et al.*, 2006).

Nutrition is increasingly recognized as a modifiable factor influencing HIV outcomes. Micronutrient deficiencies are common among people living with HIV / AIDS (PLWHA), often resulting from malabsorption, altered metabolism, anorexia, and increased nutrient losses (Semba and Tang, 1999). These deficiencies can exacerbate oxidative stress, impair immune responses, and hasten disease progression. Conversely, adequate nutritional support particularly antioxidant-rich diets can mitigate oxidative damage, enhance immune recovery, and improve ART adherence (Fawzi *et al.*, 1998; Baum *et al.*, 2011).

This review synthesizes current evidence on the interplay between HIV progression, oxidative stress, and nutritional interventions. It highlights the potential benefits, limitations, and implementation challenges of integrating targeted nutrition strategies into HIV care, with a particular focus on resource-limited settings such as Nigeria

## 2. Methods of literature search

This review followed a narrative synthesis approach to collate and interpret evidence on the interplay between HIV disease progression, oxidative stress, and nutritional interventions. A comprehensive literature search was conducted between January and March 2025 across four major electronic databases: PubMed, Scopus, Web of Science, and Google Scholar. The search covered publications from January 1998 to December 2024 to capture both foundational and recent advances in the field.

### *2.1. Search terms and boolean strategy*

A combination of Medical Subject Headings (MeSH) and free-text keywords was used, including:

- "HIV" OR "Human immunodeficiency virus".
- "AIDS" OR "Acquired immunodeficiency syndrome".
- "Oxidative stress" OR "Reactive oxygen species" OR "Antioxidants".
- "Nutrition" OR "Nutritional support" OR "Micronutrients" OR "Macronutrients".
- "Antiretroviral therapy" OR "ART" OR "HAART".

These terms were combined using Boolean operators "AND" and "OR" to refine search results (e.g., HIV and oxidative stress and nutrition).

### *2.2. Inclusion and exclusion criteria*

Inclusion criteria were:

1. Peer-reviewed articles in English.
2. Clinical trials, cohort studies, case-control studies, systematic reviews, meta-analyses, and authoritative guidelines.
3. Studies addressing at least one of the following:
    - Mechanisms linking HIV and oxidative stress.
    - Effects of ART on oxidative stress.

- Impact of nutritional interventions on HIV progression, oxidative stress, or ART outcomes.

Exclusion criteria were:

1. Non-peer-reviewed reports, opinion pieces, or conference abstracts without full data.
2. Studies focused solely on non-HIV-related oxidative stress.
3. Articles without clear methodological descriptions or outcome measures.

### *2.3. Additional search and screening*

Reference lists of relevant studies were manually screened to identify additional eligible publications. Grey literature, such as WHO and UNAIDS technical reports, was included when they provided epidemiological or policy-relevant context. Screening of titles and abstracts was performed independently by two reviewers to minimize selection bias, followed by full-text appraisal.

### *2.4. Data extraction and synthesis*

Key data extracted included study location, design, population characteristics, type of intervention (if applicable), outcomes measured, and main findings. Given the heterogeneity of study designs and outcome measures, results were synthesized narratively rather than through meta-analysis

## 3. HIV disease progression

HIV is a retroviral infection that primarily targets CD4+ T lymphocytes, key regulators of immune defense. Without treatment, the infection follows a predictable course, progressing through distinct clinical stages before culminating in Acquired Immunodeficiency Syndrome (AIDS) (Harper, 2017).

### *3.1. Stages of progression*

1. **Primary (Acute) HIV infection:** Occurring within 2-6 weeks of exposure, this stage is characterized by rapid viral replication, high plasma viral load, and a sharp decline in CD4+ T cell count. Symptoms, if present, resemble influenza fever, lymphadenopathy, sore throat, rash, and malaise—and typically resolve within weeks (Friis, 2005).
2. **Clinical latency (Chronic HIV infection):** Following immune partial recovery, the virus persists at lower replication levels. This asymptomatic phase can last several years, though ongoing viral activity gradually erodes immune function (Garrido, 2022).
3. **Symptomatic HIV infection:** As CD4+ counts decline, opportunistic infections and HIV-related conditions (e.g., oral thrush, chronic diarrhea, persistent fever) emerge. The rate of progression varies by host genetics, viral subtype, and healthcare access (Denis *et al.*, 2021).
4. **AIDS:** Defined by a CD4+ T cell count below 200 cells / $mm^3$ and / or the presence of AIDS-defining illnesses (e.g., Pneumocystis jirovecii pneumonia, cryptococcal meningitis, Kaposi's sarcoma) (Ross, 2018). Without intervention, mortality risk is extremely high.

### *3.2. Immunological and virological drivers*

Disease progression reflects the interplay of:

- CD4+ T cell depletion via direct viral killing, apoptosis, and immune-mediated destruction.
- Chronic immune activation leading to immune exhaustion.
- High viral load correlating with faster CD4+ decline.
- Viral diversity and drug resistance mutations driven by rapid replication (Jacob *et al.*, 2022).

### *3.3. Consequences of untreated HIV*

In the absence of ART, HIV leads to profound immunosuppression, frequent opportunistic infections, wasting syndrome, neurocognitive decline, and higher rates of certain cancers. Co-infections such as tuberculosis and

hepatitis accelerate progression (Repina and Pavelets, 2020). Mortality can occur within 1-3 years of developing AIDS.

## 4. Antiretroviral Therapy (ART)

### *4.1. Development and evolution*

The introduction of ART revolutionized HIV management, transforming it from a fatal disease to a manageable chronic condition. In the late 1980s, monotherapy with zidovudine (AZT) offered limited and short-lived benefits, hampered by rapid resistance and toxicity (Jha, 2018). The mid-1990s saw the advent of Highly Active Antiretroviral Therapy (HAART) combinations of three or more drugs targeting different stages of the viral life cycle which markedly reduced AIDS-related morbidity and mortality (Shahid and Bathala, 2023).

Since then, ART regimens have become more potent, better tolerated, and simpler to administer, with fixed-dose combinations improving adherence and long-term viral suppression. WHO currently recommends first-line regimens based on two Nucleoside Reverse Transcriptase Inhibitors (NRTIs) plus an Integrase Strand Transfer Inhibitor (INSTI) or Non-Nucleoside Reverse Transcriptase Inhibitor (NNRTI).

### *4.2. Classes of antiretroviral drugs*

1. **Entry inhibitors:** Block HIV from binding or fusing with host cells (e.g., maraviroc, enfuvirtide).
2. **NRTIs:** Mimic natural nucleosides, terminate viral DNA chain elongation (e.g., zidovudine, lamivudine, tenofovir).
3. **NNRTIs:** Bind directly to reverse transcriptase, altering its structure and inhibiting function (e.g., efavirenz, nevirapine).
4. **Protease inhibitors (PIs):** Prevent maturation of viral proteins, producing non-infectious virions (e.g., atazanavir, darunavir).
5. **INSTIs:** Block integration of viral DNA into the host genome (e.g., dolutegravir, bictegravir).
6. **Pharmacokinetic enhancers:** Boost plasma concentrations of companion drugs by inhibiting drug metabolism (e.g., ritonavir, cobicistat).

### *4.3. Benefits*

- Sustained viral suppression and immune reconstitution.
- Reduced morbidity, mortality, and risk of HIV transmission (“U=U” principle).
- Improved quality of life and life expectancy approaching that of HIV-negative individuals.

### *4.4. Challenges*

- **Adherence:** Complex dosing, side effects, stigma, and food insecurity can compromise adherence.
- **Drug resistance:** Emerges from incomplete viral suppression.
- **Toxicities:** Mitochondrial dysfunction, metabolic disturbances, and oxidative stress can result from certain ART agents.
- **Access barriers:** Limited drug availability and high costs in low-resource settings remain obstacles (Bai *et al.*, 2020)

## 5. Oxidative stress in HIV

### *5.1. Definition and mechanisms*

Oxidative stress occurs when the production of Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS) exceeds the capacity of endogenous antioxidant defenses, leading to cellular damage (Chelikani *et al.*, 2004). In HIV infection, oxidative stress is driven by multiple factors:

- Chronic immune activation leading to overproduction of ROS by activated macrophages and neutrophils.
- Direct viral effects, as HIV proteins such as gp120 and Tat promote mitochondrial dysfunction and ROS generation (Awodele *et al.*, 2012).
- ART-induced toxicity, particularly from NRTIs and PIs, which can impair mitochondrial DNA replication and increase ROS production (Bhatia *et al.*, 2015).

### *5.2. Cellular and molecular impacts*

Excessive ROS damages lipids, proteins, and nucleic acids, leading to:

- Lipid peroxidation, compromising membrane integrity.
- DNA strand breaks and base modifications, potentially accelerating mutagenesis.
- Protein oxidation, impairing enzyme function and structural stability.
- Apoptosis of immune cells, especially CD4+ T lymphocytes, exacerbating immunosuppression (Allard *et al.*, 2006).

### *5.3. Evidence linking HIV and oxidative stress*

Studies have consistently shown elevated oxidative stress biomarkers in HIV-positive individuals compared to HIV-negative controls, including increased malondialdehyde (MDA) levels and reduced antioxidant enzyme activities (SOD, catalase, glutathione peroxidase) (Akinmoladun *et al.*, 2019; Sharma, 2021). ART, while suppressing viral load, does not fully normalize oxidative stress markers, suggesting persistent pro-oxidative mechanisms independent of active viral replication.

### *5.4. Role in disease progression*

Oxidative stress contributes to:

- Immune dysfunction by promoting apoptosis and impairing lymphocyte proliferation.
- Viral replication enhancement, as oxidative conditions may activate NF-κB, a transcription factor that promotes HIV gene expression.
- Comorbidity risk, including cardiovascular disease, neurocognitive decline, and cancer.

### *5.5. Conceptual framework*

The conceptual framework below illustrates the interplay between HIV infection, Antiretroviral Therapy (ART),

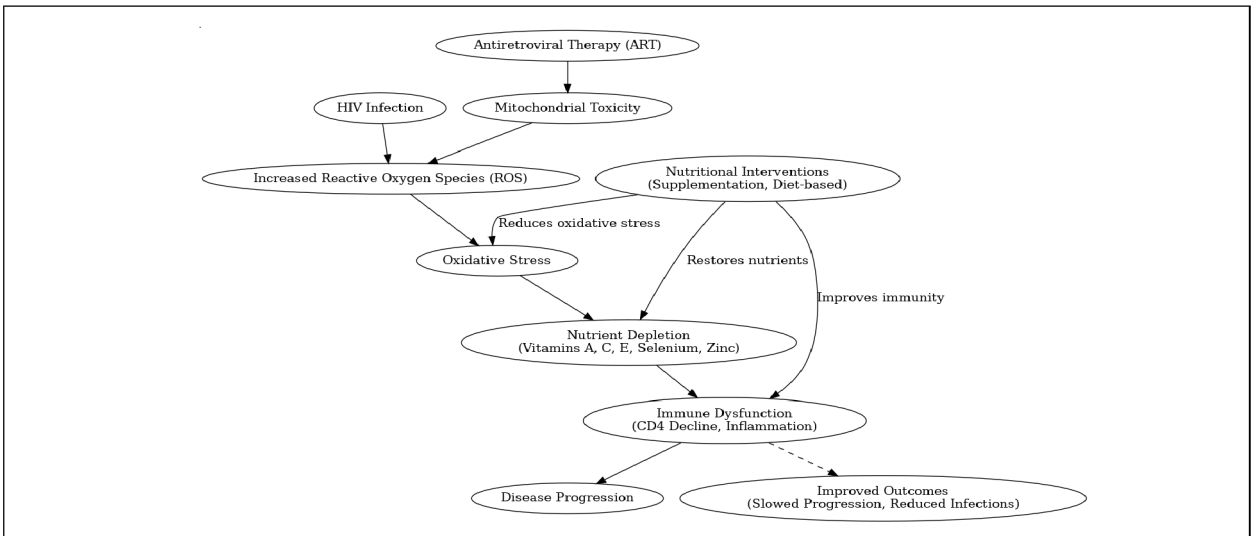


**Figure 1: Illustrates the interplay between HIV infection, antiretroviral therapy (ART), oxidative stress, and nutritional interventions**

oxidative stress, and nutritional interventions. It demonstrates how HIV and ART contribute to oxidative stress, which in turn impacts immune function and disease progression, while nutrition serves as a modifiable factor that can break this cycle.

## 6. Nutritional status, HIV and oxidative stress

### *6.1. Overview*

Nutritional status is a critical determinant of health outcomes in people living with HIV/AIDS (PLWHA). HIV infection increases energy expenditure, alters nutrient metabolism, and often coincides with reduced dietary intake due to anorexia, oral lesions, gastrointestinal disturbances, or socioeconomic constraints (Semba and Tang, 1999). Oxidative stress is both a cause and consequence of nutrient depletion: Reactive Oxygen Species (ROS) consume antioxidants, while inadequate nutrient reserves impair the body's ability to counteract oxidative damage.

### *6.2. Micronutrients with antioxidant functions*

*6.2.1. Vitamin A (Retinoid and carotenoids)*

- **Role:** Maintains epithelial integrity, supports immune function, and modulates oxidative processes.
- **Evidence:** Supplementation has been associated with reduced morbidity in HIV-infected children and pregnant women but may increase viral shedding in some cases (Fawzi *et al.*, 1999).
- **Sources:** Liver, dairy products, orange and dark green vegetables.

*6.2.2. Vitamin C (Ascorbic acid)*

- **Role:** Potent water-soluble antioxidant; regenerates oxidized vitamin E; scavenges ROS.
- **Evidence:** HIV-positive individuals often exhibit reduced plasma vitamin C; supplementation improves antioxidant status and may reduce oxidative DNA damage (Allard *et al.*, 1998).
- **Sources:** Citrus fruits, tomatoes, peppers, leafy greens.

*6.2.3. Vitamin E ($\alpha$-Tocopherol)*

- **Role:** Lipid-soluble antioxidant protecting cell membranes from lipid peroxidation.
- **Evidence:** Low vitamin E levels correlate with faster HIV disease progression; supplementation improves immune parameters and reduces oxidative stress markers (Baum *et al.*, 1994).
- **Sources:** Nuts, seeds, vegetable oils.

*6.2.4. Selenium*

- **Role:** Integral component of glutathione peroxidase, a key antioxidant enzyme.
- **Evidence:** Deficiency is linked to increased mortality risk in PLWHA; supplementation improves immune function and may slow disease progression (Baum *et al.*, 1997).
- **Sources:** Brazil nuts, seafood, cereals.

*6.2.5. Zinc*

- **Role:** Cofactor for antioxidant enzymes (e.g., superoxide dismutase) and critical for immune cell proliferation.
- **Evidence:** Deficiency is common in HIV and associated with increased opportunistic infections; supplementation requires caution as excessive zinc may promote viral replication (Tang *et al.*, 1993).
- **Sources:** Meat, legumes, whole grains.

### *6.3. Phytochemicals and dietary antioxidants*

Plant-derived compounds such as flavonoids, carotenoids, and polyphenols possess strong ROS-scavenging abilities. Diets rich in fruits, vegetables, whole grains, and legumes provide synergistic antioxidant effects beyond isolated nutrient supplementation (Serafini and Peluso, 2016).

### 6.4. Macronutrient considerations

Adequate protein intake is essential for immune cell synthesis and antioxidant enzyme production. HIV infection often induces hypermetabolism, requiring higher caloric intake to maintain lean body mass (Kotler, 2000). Balanced macronutrient provision supports ART tolerance and metabolic stability.

### 6.5. Implications for intervention

Targeted nutritional interventions can restore antioxidant capacity, improve immune markers, and potentially delay disease progression. However, supplementation strategies must be context-specific, considering baseline nutritional status, ART regimen, and potential drug nutrient interactions.

#### 6.5.1. Interactions and implications

The interplay between HIV infection, Antiretroviral Therapy (ART), oxidative stress, and nutrition forms a complex network that influences disease progression and treatment outcomes. HIV infection increases oxidative stress through chronic immune activation, viral protein-induced ROS generation, and depletion of antioxidant reserves. ART, while suppressing viral replication, can further exacerbate oxidative stress via mitochondrial toxicity, metabolic disturbances, and altered nutrient metabolism.

Nutritional interventions can modulate this cycle by replenishing antioxidant defenses, supporting immune cell function, and improving ART tolerability. However, their effectiveness depends on several factors, including the stage of HIV infection, baseline nutritional status, ART regimen, and adherence levels.

The implications for clinical practice are clear: nutrition should be integrated into HIV care as a core component, not an adjunct. This integration requires interdisciplinary collaboration among clinicians, dietitians, and community health workers, especially in resource-limited settings where food insecurity is prevalent.

**Table 1: Interaction between HIV, ART, Oxidative stress and Nutrition**

| Factors | Effect on oxidative stress | Impact on nutrition | Clinical implications |
|---|---|---|---|
| HIV infection | ↑ ROS production via immune activation and viral proteins | Nutrient malabsorption, anorexia, increased metabolic demand | Antioxidant depletion, micronutrient deficiencies |
| ART | Mitochondrial toxicity → ↑ ROS | Altered nutrient metabolism | Persistent oxidative stress despite viral suppression |
| Oxidative stress | Damages lipids, proteins, DNA | Impaired nutrient utilization | Immune suppression, faster disease progression |
| Nutrition | Antioxidants neutralize ROS, support immunity | Nutrient adequacy improves ART adherence | Reduced inflammation, slower disease progression |

**Note:** ↑ = Increase.

## 7. Research gaps

Despite substantial evidence linking oxidative stress, nutrition, and HIV progression, several knowledge gaps remain:

1. **Optimal nutrient combinations and doses:** Most studies examine single micronutrients, but synergistic effects of combined supplementation are underexplored.
2. **Long-term clinical outcomes:** Few trials track the sustained effects of nutritional interventions on mortality, ART adherence, and comorbidity prevention.
3. **Population-specific needs:** There is limited evidence for context-specific interventions tailored to pregnant women, children, and older adults living with HIV in sub-Saharan Africa.
4. **Mechanistic insights:** More research is needed on how specific nutrients modulate HIV-related inflammatory and oxidative pathways at the molecular level.
5. **Drug–nutrient interactions:** The impact of ART regimens on micronutrient absorption and metabolism remains poorly characterized.

## 8. Conclusion and recommendations

### *8.1. Conclusion*

HIV infection induces chronic oxidative stress that persists even under effective ART, contributing to immune dysfunction and accelerated disease progression. Nutritional interventions—particularly those rich in antioxidants such as vitamins C and E, selenium, and carotenoids—can reduce oxidative damage, improve immune recovery, and enhance ART tolerability. However, implementation must consider individual nutritional status, ART regimen, and local food availability.

Integrating targeted nutrition support into HIV care could significantly improve clinical outcomes, especially in resource-limited settings where malnutrition and food insecurity are prevalent.

### *8.2. Recommendations*

#### *8.2.1. Policy and programmatic*

- Incorporate nutritional assessment and counseling into routine HIV care protocols.
- Strengthen food support programs for PLWHA, particularly in high-prevalence, low-income regions.
- Support local production and fortification of antioxidant-rich foods.

#### *8.2.2. Research*

- Conduct large-scale, long-term randomized trials to define optimal supplementation strategies.
- Investigate nutrient–drug interactions in different ART regimens.
- Explore cost-effective, culturally appropriate dietary interventions in sub-Saharan Africa.

### *8.3. Practical recommendations for clinicians*

1. **Assess nutritional status regularly:** Include anthropometry, dietary history, and laboratory markers of key micronutrients.
2. **Encourage whole-food sources:** Promote diets rich in fruits, vegetables, nuts, seeds, legumes, and whole grains to provide a spectrum of antioxidants.
3. **Monitor for deficiencies:** Particularly vitamins A, C, and E, selenium, and zinc; address with supplementation when indicated.
4. **Consider ART–nutrition interactions:** Adjust nutritional advice based on regimen side effects (e.g., gastrointestinal disturbances, lipid metabolism changes).
5. **Integrate counseling into HIV clinics:** Collaboration between clinicians, dietitians, and pharmacists improves patient adherence and outcomes.

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## Conflicts of interest

The authors declare no conflicts of interest.

## Ethics statement

This study did not involve human participants, animal subjects, or materials requiring ethical approval.

## Informed consent

As no human participants were involved, informed consent was not applicable.

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