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Immunometabolism in HIV Infection: Mechanisms, Biomarkers, and Therapeutic Opportunities

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Abstract

Human immunodeficiency virus (HIV) infection induces profound and persistent alterations in host cell metabolism that extend well beyond viral replication, reshaping how immune cells generate energy and use nutrients to support proliferation, effector function, and survival. This phenomenon, termed immunometabolism, has emerged as a central axis linking chronic immune activation, oxidative stress, and disease progression in people living with HIV (PLHIV), including those receiving suppressive antiretroviral therapy (ART). Infected and bystander CD4+ T cells, monocytes/macrophages, and other immune subsets undergo a shift toward aerobic glycolysis, mitochondrial dysfunction, and dysregulated reactive oxygen and nitrogen species (RONS) production, sustaining inflammation, exhausting antioxidant reserves, and favouring viral persistence within long-lived cellular reservoirs. Despite virologic suppression, many of these metabolic and redox abnormalities are incompletely normalized, contributing to residual inflammation and premature onset of non-AIDS comorbidities. This review synthesizes current evidence on the mechanistic links between immunometabolic reprogramming and oxidative stress in HIV infection, catalogues candidate biomarkers of immunometabolic dysfunction, and appraises emerging metabolic and antioxidant-based therapeutic strategies as adjuncts to ART. Population-specific factors relevant to Sub-Saharan African cohorts are also considered. A clearer understanding of immunometabolic pathways in HIV infection may open avenues for biomarker-guided, host-directed adjunct therapies aimed at reducing chronic inflammation and improving long-term outcomes for PLHIV.

Keywords: *HIV, immunometabolism, oxidative stress, glycolysis, mitochondrial dysfunction, antiretroviral therapy, biomarkers.*

1. Introduction

HIV infection remains a major global health burden despite the transformative impact of combination antiretroviral therapy (ART), which has converted a once uniformly fatal disease into a manageable chronic condition. Even when plasma viremia is durably suppressed, people living with HIV (PLHIV) continue to experience elevated rates of cardiovascular disease, neurocognitive impairment,

and accelerated aging compared with HIV-negative counterparts, phenomena largely attributed to persistent low-grade immune activation and chronic inflammation [1].

Over the past decade, immunometabolism is the study of how metabolic pathways shape and are shaped by immune cell function, has provided a mechanistic framework for understanding this residual inflammatory state. Immune cells rely on distinct metabolic programs to support their proliferative and effector demands: resting lymphocytes and monocytes largely depend on oxidative phosphorylation (OXPHOS) and fatty acid oxidation, whereas activated, proliferating, or effector cells preferentially switch to aerobic glycolysis, reminiscent of the Warburg effect described in cancer cells [2,3].

HIV infection perturbs this metabolic equilibrium at multiple levels. The virus preferentially infects and persists within metabolically active, glycolytic CD4+ T cells, and chronic antigenic stimulation, microbial translocation from a damaged gut barrier, and persistent low-level viral replication in sanctuary sites drive sustained immune activation that further skews immune cell metabolism toward glycolysis and away from efficient mitochondrial respiration [3,4].

This review aims to (i) summarize current mechanistic understanding of immunometabolic reprogramming across key immune cell subsets in HIV infection, (ii) examine the bidirectional relationship between immunometabolism and oxidative stress, (iii) catalogue candidate biomarkers of immunometabolic dysfunction with translational potential, (iv) evaluate the impact of ART on restoring metabolic and redox homeostasis, and (v) appraise emerging therapeutic strategies as adjuncts to conventional HIV care.

2. Metabolic Reprogramming of Immune Cells in HIV Infection

2.1 The Glycolytic Switch in HIV-Infected and Bystander CD4+ T Cells

Activated CD4+ T cells, whether directly infected or bystander cells responding to chronic antigenic and inflammatory stimuli, characteristically upregulate glucose transporters and glycolytic enzymes to meet the bioenergetic and biosynthetic demands of proliferation. Cellular metabolic state is a major determinant of HIV-1 susceptibility: CD4+ T cells with higher glycolytic and glucose-uptake capacity are preferentially and more efficiently infected and support more robust reservoir seeding than cells relying predominantly on oxidative metabolism [5–7].

This creates a self-reinforcing cycle in which chronic immune activation increases the pool of glycolytically active target cells, which in turn supports ongoing viral persistence even under suppressive ART [7,8].

2.2 Mitochondrial Dysfunction and Impaired Oxidative Phosphorylation

Mitochondria are not merely bioenergetic organelles but also central hubs of innate and adaptive immune signalling, contributing to inflammasome activation, cytokine production, and antiviral responses. In HIV infection, mitochondrial membrane potential, respiratory capacity, and mitochondrial DNA (mtDNA) integrity are progressively compromised in both infected and uninfected bystander cells, a consequence of direct viral protein effects, chronic inflammatory signalling, and, in some cases, off-target effects of certain antiretroviral drug classes [9,10].

Immunometabolic profiling of peripheral blood mononuclear cells (PBMCs) has demonstrated that alterations in central carbon and lipid metabolism persist despite years of suppressive ART, indicating incomplete normalization of the immunometabolic phenotype even in virologically controlled PLHIV. This residual dysfunction is thought to sustain low-grade inflammation and contribute to the excess burden of cardiometabolic and neurocognitive comorbidities observed in treated PLHIV [4,8].

2.3 Macrophage Metabolic Polarization and Viral Reservoirs

Monocytes and macrophages display remarkable metabolic plasticity, shifting between glycolysis-dominant, pro-inflammatory (M1-like) and OXPHOS-dominant, reparative (M2-like) phenotypes depending on the microenvironment. HIV infection biases macrophage populations toward a pro-inflammatory, glycolytically active state that supports viral replication in tissue reservoirs, including the central nervous system and lung, while also impairing key macrophage antimicrobial and antioxidant functions [6,11]. Figure 1 presents the Immunometabolic reprogramming in uninfected versus HIV-infected/chronically activated CD4+ T cells and macrophages.

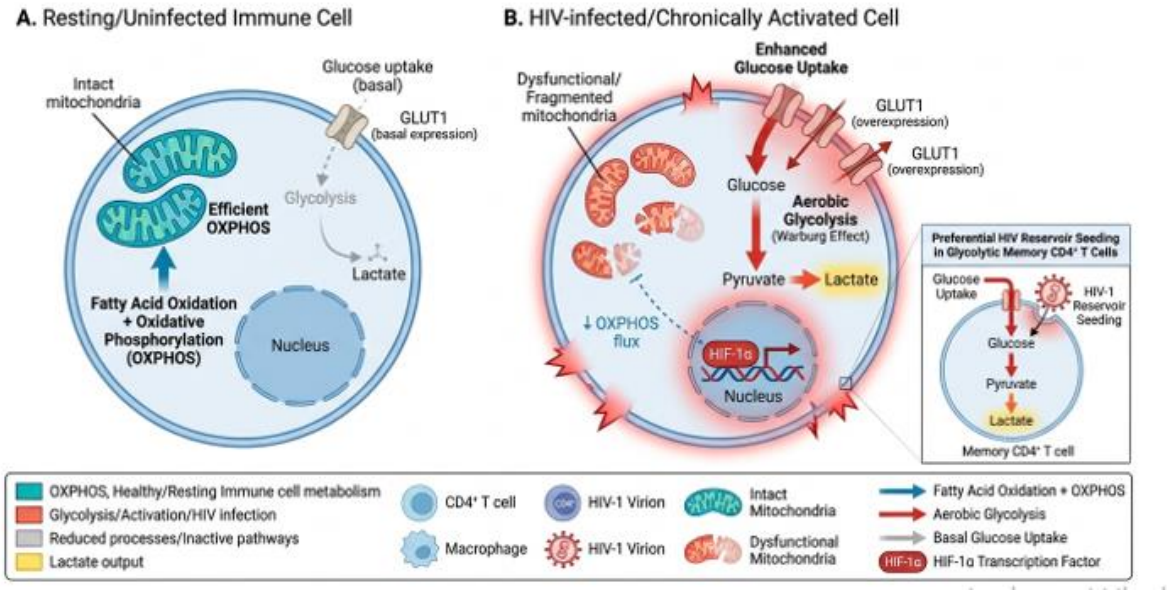

Figure 1. Immunometabolic reprogramming in uninfected versus HIV-infected/chronically activated CD4+ T cells and macrophages.

"***Caption:*** *A scientific schematic diagram illustrating immunometabolic reprogramming in HIV-infected versus uninfected CD4+ T cells and macrophages. Panel A: a resting/uninfected immune cell relying on oxidative phosphorylation (OXPHOS) and fatty acid oxidation, with intact, efficient mitochondria. Panel B: an HIV-infected or chronically activated bystander cell showing a shift toward aerobic glycolysis (Warburg-like effect), increased GLUT1 transporter expression, elevated lactate output, and dysfunctional/fragmented mitochondria. Include labelled arrows for glucose uptake, glycolysis, and reduced OXPHOS flux; label key molecules (GLUT1, HIF-1α, lactate); indicate preferential HIV-1 reservoir seeding in glycolytic memory CD4+ T cells".*

Sources: Figure adapted from the mechanisms described in references [5–9]

HIV-associated suppression of nuclear factor erythroid 2-related factor 2 (Nrf2) signalling, mediated in part through microRNA-144 upregulation, compromises alveolar macrophage antioxidant capacity and innate defense, illustrating a direct mechanistic link between immunometabolic reprogramming, oxidative stress, and impaired tissue immunity in HIV infection [11]. Table 1 shows the Metabolic reprogramming across immune cell subsets in HIV infection.

Table 1. Metabolic reprogramming across immune cell subsets in HIV infection.

Immune cell type	Predominant metabolic shift	Functional consequence	Key reference(s)
Activated / bystander CD4+ T cells	Increased aerobic glycolysis; upregulated GLUT1 and glycolytic enzymes	Enhanced HIV-1 susceptibility and reservoir seeding	[5–7]
Latently infected CD4+ T cells	Preferential seeding within glycolytically active memory subsets	Sustains a long-lived, ART-resistant viral reservoir	[7,8]
PBMCs, ART-suppressed PLHIV	Persistent alterations in central carbon and lipid metabolism	Incomplete immunometabolic normalization despite viral suppression	[8]
Tissue macrophages (e.g., alveolar)	Glycolytic, pro-inflammatory (M1-like) polarization; Nrf2 suppression	Impaired antioxidant defense and antimicrobial function; reservoir maintenance	[6,11]

Immune cell type	Predominant metabolic shift	Functional consequence	Key reference(s)
CD8+ T cells	Reduced mitochondrial spare respiratory capacity	Metabolic exhaustion; impaired antiviral cytotoxic function	[6,9]

3. Oxidative Stress as a Driver and Consequence of Immunometabolic Dysfunction

Oxidative stress arises when the generation of reactive oxygen and nitrogen species (RONS) exceeds the buffering capacity of endogenous enzymatic (superoxide dismutase, catalase, glutathione peroxidase) and non-enzymatic (glutathione, vitamins A, C, E, selenium, zinc) antioxidant systems. Chronic immune activation and persistent inflammation are increasingly recognized as key drivers of oxidative stress in HIV disease, independent of ongoing plasma viral replication [12,13].

In HIV infection, RONS are generated through multiple, interconnected mechanisms: direct effects of viral proteins (e.g., Tat, Nef, gp120/Env) on mitochondrial function and NADPH oxidase activity; chronic pro-inflammatory cytokine signalling; and dysregulated glycolytic and mitochondrial metabolism. This creates a self-amplifying cycle in which glycolytic reprogramming and mitochondrial dysfunction promote RONS overproduction, while RONS in turn further impair mitochondrial integrity and amplify inflammatory signalling pathways such as NF-κB [12,14].

HIV-1 envelope glycoprotein (Env) has been shown to induce pexophagy; the selective autophagic degradation of peroxisomes, key organelles involved in RONS detoxification, in uninfected bystander CD4+ T cells, leading to oxidative stress-mediated cell death and contributing to the profound CD4+ T-cell depletion characteristic of untreated HIV infection. This mechanism illustrates how oxidative stress in HIV infection is not confined to directly infected cells but extends to the broader immune compartment through paracrine and organelle-targeted mechanisms [15]. Figure 2. Illustrates the self-amplifying cycle of oxidative stress and immunometabolic dysfunction in HIV infection.

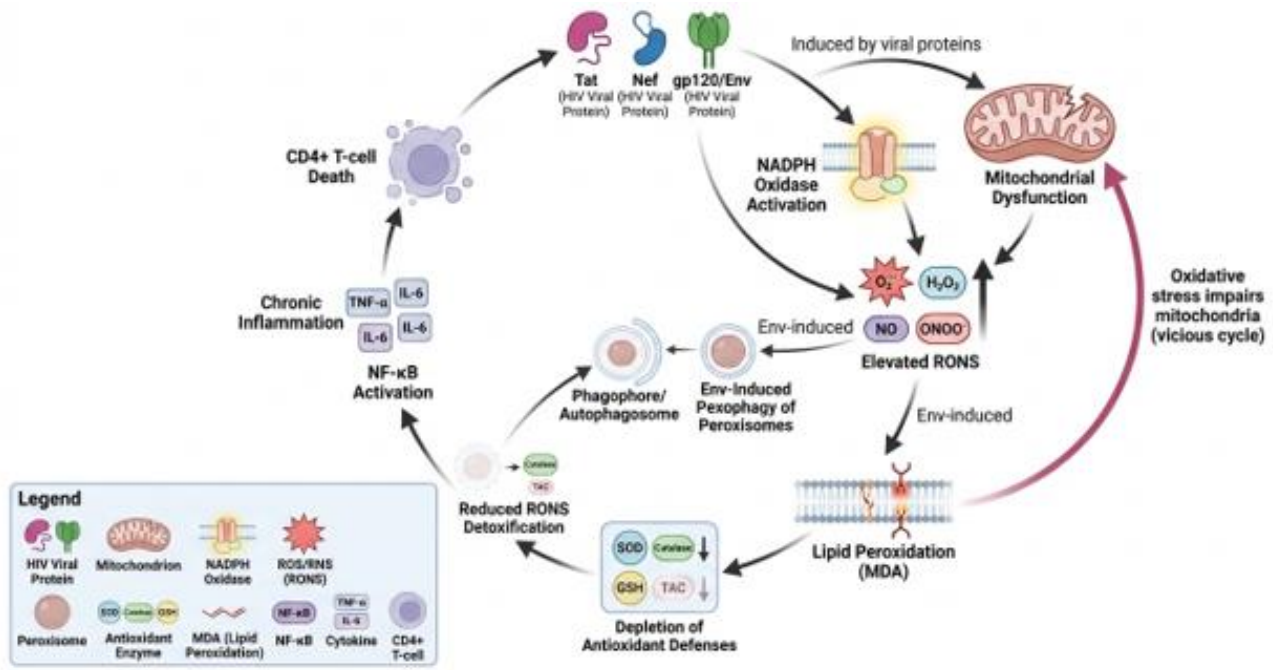

Figure 2. The self-amplifying cycle of oxidative stress and immunometabolic dysfunction in HIV infection.

Caption: *A circular scientific pathway diagram depicting the oxidative stress cycle in HIV infection. Show HIV viral proteins (Tat, Nef, gp120/Env) triggering mitochondrial dysfunction and NADPH oxidase activation, leading to increased reactive oxygen and nitrogen species (RONS) production. Depict Env-induced pexophagy of peroxisomes, reducing RONS detoxification capacity. Show depletion of antioxidant*

defences (SOD, catalase, glutathione, total antioxidant capacity) and downstream effects: lipid peroxidation (MDA), NF-κB activation, chronic inflammation, and CD4+ T-cell death. Include a feedback-loop arrow showing how oxidative stress further impairs mitochondrial function.

Sources: Adapted from references [11,12,14,15].

Clinical studies corroborate these mechanistic findings. Cross-sectional assessments of PLHIV consistently report elevated markers of lipid peroxidation, such as malondialdehyde (MDA), alongside reduced total antioxidant capacity (TAC) and depleted levels of key micronutrient antioxidants compared with HIV-negative controls [14,16,17].

Reduced TAC and elevated oxidative stress indices have also been documented among PLHIV in Sub-Saharan African cohorts, where background micronutrient insufficiency may compound HIV-associated oxidative burden. Alterations in lipoperoxidation and antioxidant balance have further been implicated in HIV-associated complications during pregnancy, suggesting organ- and life-stage-specific consequences of oxidative stress-driven immunometabolic dysfunction [16–18].

4. Immune Cell-Specific Metabolic Alterations and Viral Persistence

Beyond CD4+ T cells and macrophages, emerging evidence implicates immunometabolic dysregulation in the function of natural killer cells, CD8+ T cells, and B cells in HIV infection, although mechanistic data in these subsets remain comparatively limited. Chronic activation-induced metabolic exhaustion, characterised by reduced mitochondrial spare respiratory capacity and increased reliance on glycolysis, has been proposed as a contributor to CD8+ T-cell dysfunction and impaired viral control in progressive HIV disease [6,9].

The persistence of a small, transcriptionally quiescent but metabolically primed pool of latently infected CD4+ T cells is influenced by cellular metabolic state, with glycolytically active memory CD4+ T-cell subsets serving as preferential sites of reservoir seeding. These observations position immunometabolic reprogramming not merely as a bystander consequence of infection but as an active contributor to viral reservoir maintenance and a potential target for HIV cure strategies [7,8].

5. Biomarkers of Immunometabolic Dysfunction in HIV

Given the mechanistic centrality of metabolic and oxidative dysregulation in HIV pathogenesis, several circulating and cellular biomarkers have been proposed to capture residual immunometabolic dysfunction in PLHIV, particularly those on suppressive ART (Table 2). These include markers of lipid peroxidation (MDA, protein carbonyls), antioxidant reserve (TAC, glutathione, superoxide dismutase and catalase activity, micronutrient status), mitochondrial integrity (circulating cell-free mtDNA), and metabolic flux (plasma lactate, glucose-uptake indices) [12,14,18,19].

Diverse dietary and supplement-derived antioxidant sources are being investigated as adjuncts to correct the oxidative imbalance observed in PLHIV, underscoring the translational relevance of these biomarkers for monitoring intervention response. Work examining antioxidant enzyme activity and micronutrient status in Sub-Saharan African PLHIV cohorts further highlights the potential of combined biomarker panels, rather than single analytes, to more accurately capture the heterogeneity of immunometabolic dysfunction across populations with differing baseline nutritional status [14,19].

Despite this progress, standardisation of assay methods, reference ranges, and validation in longitudinal cohorts remain necessary before these biomarkers can be incorporated into routine clinical monitoring of PLHIV.

Table 2. Candidate biomarkers of immunometabolic dysfunction in HIV infection.

Biomarker category	Representative markers	Clinical/biological significance	Key reference(s)
Lipid peroxidation	Malondialdehyde (MDA), protein carbonyls	Indicates oxidative membrane and protein damage	[12,14]
Antioxidant reserve	Total antioxidant capacity (TAC), SOD, catalase, glutathione	Reflects capacity to buffer RONS	[14,16–18]
Micronutrient status	Vitamins A, C, E; zinc; selenium	Depletion associated with increased oxidative burden	[14,19]

Biomarker category	Representative markers	Clinical/biological significance	Key reference(s)
Mitochondrial integrity	Circulating cell-free mtDNA, mitochondrial membrane potential	Marker of mitochondrial injury and dysfunction	[8,20]
Metabolic flux	Plasma lactate, glucose-uptake indices	Reflects glycolytic reprogramming of immune cells	[7,8]

6. Effect of Antiretroviral Therapy on Immunometabolism

ART, particularly integrase strand transfer inhibitor (INSTI)-based regimens, has been shown to significantly reduce markers of oxidative stress compared with pre-treatment levels, suggesting that viral suppression itself is a major determinant of redox and metabolic recovery [21].

However, this normalisation is frequently incomplete: studies assessing mitochondrial and oxidative parameters after short- and long-term highly active antiretroviral therapy (HAART) report persistent mitochondrial toxicity and oxidative imbalance in a subset of patients, with some effects related to specific drug classes known to impair mitochondrial DNA polymerase-γ [20].

Consistent with this, immunometabolic profiling of PBMCs from ART-suppressed PLHIV reveals that alterations in central metabolic and lipid pathways persist despite years of virologic control, indicating that ART alone is insufficient to fully restore immunometabolic homeostasis. These findings support the rationale for host-directed adjunct therapies aimed specifically at correcting residual metabolic and oxidative dysfunction in ART-treated PLHIV [8].

7. Therapeutic Opportunities Targeting Immunometabolic Pathways

Recognition that immunometabolic and oxidative dysregulation persists despite viral suppression has stimulated interest in adjunct therapeutic strategies operating alongside conventional ART (Table 3). Broadly, these approaches fall into three categories: (i) antioxidant and micronutrient supplementation strategies aimed at restoring redox balance and replenishing depleted vitamins and trace elements [14,19];

(ii) metabolic modulators that target glycolytic flux or restore mitochondrial oxidative capacity in immune cells, informed by mechanistic work on the glycolytic dependence of HIV reservoir cells [7,8];

and (iii) agents targeting specific redox-regulatory nodes, such as Nrf2 pathway activators, which could reverse HIV-associated suppression of endogenous antioxidant gene expression in macrophages and other innate immune cells [11].

Dietary and nutraceutical antioxidant sources have received particular attention in resource-limited settings, where both HIV-associated and background nutritional oxidative stress may be more pronounced, and where affordable, accessible interventions are especially valuable. Nonetheless, most evidence to date derives from observational or small interventional studies, and robust randomised controlled trials are needed to establish clinical efficacy, optimal dosing, and safety before these adjunct approaches can be recommended as standard practice [14,19].

Table 3. Emerging therapeutic strategies targeting immunometabolism in HIV infection.

Strategy	Proposed mechanism	Evidence stage	Key reference(s)
Micronutrient/antioxidant supplementation (vitamin C, E, selenium, zinc; dietary sources)	Replenishes depleted antioxidant reserves; reduces lipid peroxidation	Observational / small interventional studies	[14,19]
Nrf2 pathway activators	Restores endogenous antioxidant gene expression suppressed by HIV	Preclinical/mechanistic	[11]

Strategy	Proposed mechanism	Evidence stage	Key reference(s)
INSTI-based ART optimization	Reduces oxidative stress more effectively than some other ART classes	Clinical comparative studies	[21]
Mitochondrial-protective agents	Mitigate mitochondrial toxicity associated with certain ART classes	Clinical/observational	[20]
Metabolic modulators (glycolysis-targeting agents)	Reduce glycolytic support for reservoir-harbouring cells	Preclinical/conceptual	[7,8]

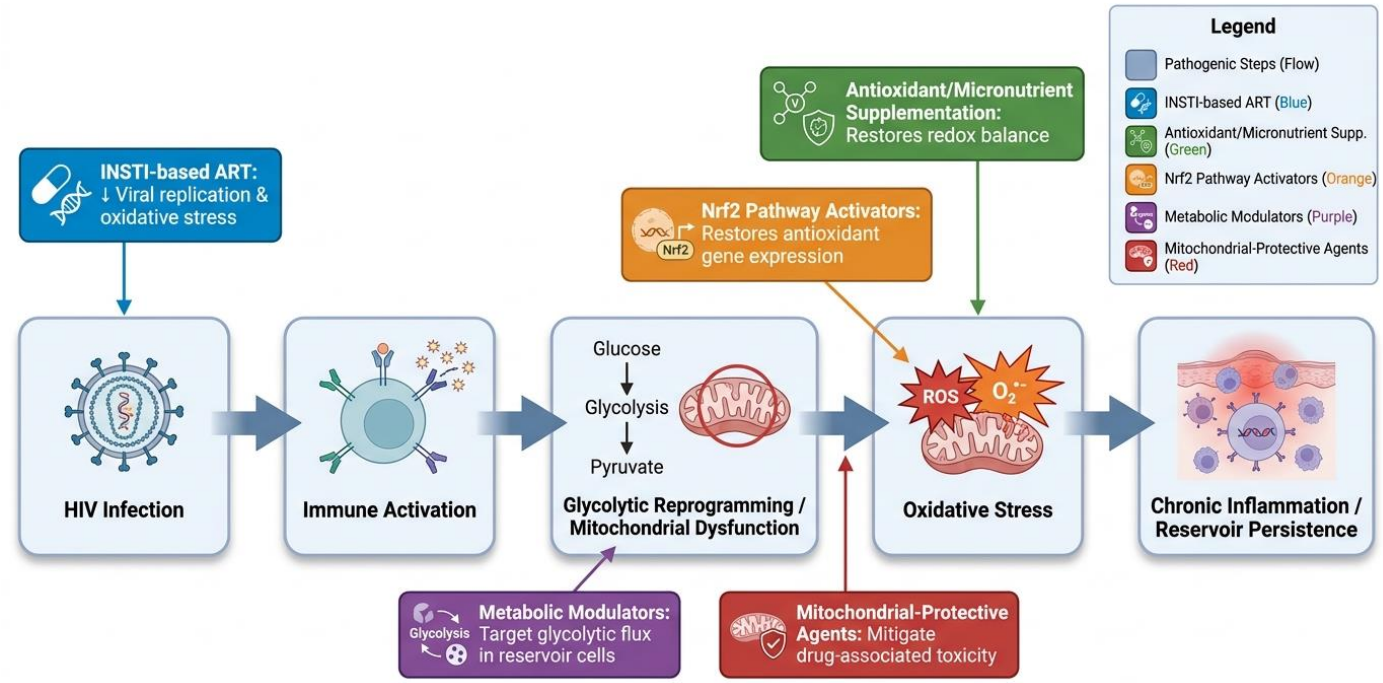

Figure 3. Therapeutic targets along the HIV immunometabolism pathway.

Caption: *A schematic pathway showing therapeutic targets in the HIV immunometabolism pathway: HIV infection → immune activation → glycolytic reprogramming/mitochondrial dysfunction → oxidative stress → chronic inflammation/reservoir persistence. There are five labelled intervention points in the diagram: (1) INSTI-based ART reducing viral replication and oxidative stress; (2) antioxidant/micronutrient supplementation restoring redox balance; (3) Nrf2 pathway activators restoring antioxidant gene expression; (4) metabolic modulators targeting glycolytic flux in reservoir cells; (5) mitochondrial-protective agents mitigating drug-associated toxicity.*

Sources: Adapted from references [11,14,19–21].

8. Population-Specific Considerations: Sub-Saharan Africa

Sub-Saharan Africa bears a disproportionate share of the global HIV burden, and PLHIV in this region often present with compounding factors, including background micronutrient insufficiency, co-endemic infections, and variable access to consistent ART, that may exacerbate immunometabolic and oxidative dysfunction beyond that attributable to HIV infection alone [18,19].

Cross-sectional data from Nigerian cohorts have documented significantly reduced plasma antioxidant capacity and elevated oxidative stress markers among PLHIV compared with HIV-negative controls, correlating with markers of disease severity. Reviews focused on antioxidant enzyme activity and micronutrient status in Sub-Saharan African populations further emphasise the potential value of regionally tailored nutritional and antioxidant supplementation strategies, given the interplay

between endemic nutritional deficiencies and HIV-associated oxidative burden [18,19].

These population-specific findings underscore the importance of context-sensitive biomarker validation and intervention design rather than uncritical extrapolation of findings from higher-resource settings.

9. Conclusion and Future Directions

Immunometabolic reprogramming is now recognised as a central, interconnected feature of HIV pathogenesis, tightly linked to oxidative stress, chronic immune activation, and viral reservoir persistence. Even in the era of effective ART, residual immunometabolic and redox abnormalities appear to contribute to ongoing inflammation and the excess burden of non-AIDS comorbidities experienced by PLHIV.

Candidate biomarkers spanning oxidative stress, antioxidant reserve, mitochondrial integrity, and metabolic flux offer promising, if still incompletely validated, tools for stratifying residual risk and monitoring adjunct interventions. Emerging therapeutic strategies, ranging from targeted antioxidant and micronutrient supplementation to metabolic modulators and Nrf2 pathway activators, hold potential to complement ART by directly addressing the immunometabolic drivers of chronic inflammation.

Future research should prioritise longitudinal, multi-omic characterisation of immunometabolic trajectories across diverse populations, standardisation of biomarker assays, and well-powered randomised controlled trials of adjunct metabolic and antioxidant therapies, with particular attention to resource-limited, high-burden settings such as Sub-Saharan Africa.

Conflict of Interest

The authors declare no conflict of interest.

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