P-ISSN : 2527-4627
Warmadewa Medical Journal
Available online http://ejournal.warmadewa.ac.id/index.php/warmadewa_medical_journal

WMJ (Warmadewa Medical Journal), Vol. 11, No.1, May 2026, Page. 54-62

The Role of Micronutrients in Human Immunodeficiency Virus Infection: A Narrative Review

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Abstract

Human Immunodeficiency Virus (HIV) remains a major global health burden, with micronutrient deficiency being a frequent complication that worsens disease progression. Deficiencies in vitamins A, D, E, and zinc have been linked to impaired immune responses, increased susceptibility to opportunistic infections, and poor treatment outcomes. This narrative review aims to explore the role of micronutrients, particularly vitamins A, D, E, and zinc, in the clinical course and management of HIV infection. A literature-based narrative review was conducted using PubMed, Scopus, and Google Scholar. Articles discussing the association between micronutrients and HIV progression, immune response, and treatment outcomes were synthesized. A total of 847 articles were initially identified; after screening and applying inclusion criteria, 42 articles were included in this review. Evidence shows that vitamin A influences mucosal immunity and reduces mother-to-child transmission; vitamin D modulates immune cell proliferation and autophagy; vitamin E acts as an antioxidant reducing oxidative stress; and zinc supports T-cell function and reduces opportunistic infections. Vitamin D supplementation significantly increased serum 25(OH)D levels ($p<0.0001$) but did not significantly alter CD4 counts or viral load in clinical trials. Overall, supplementation outcomes remain inconsistent. While multiple micronutrient supplementation may improve immune markers, its impact on mortality and viral load reduction is still inconclusive. Micronutrient deficiency is common among HIV patients and negatively affects disease progression. Although supplementation may improve nutritional and immunological status, robust evidence regarding long-term survival and virological outcomes is lacking. Further high-quality studies are required to establish clear recommendations.

Keywords: HIV, Micronutrients, Vitamin A, Vitamin D, Vitamin E, Zinc

INTRODUCTION

Human Immunodeficiency Virus (HIV) remains a major global public health burden, with profound epidemiological, clinical, and socioeconomic implications. In 2019, more than 38 million people were living with HIV worldwide, and 1.7 million new infections were reported in that year alone. Sub-Saharan Africa accounted for nearly 61% of these new infections, while significant increases were also observed in Eastern Europe, Central Asia, the Middle East, and Latin America (1). In 2016, the United Nations General Assembly endorsed the Fast-Track strategy aimed at ending AIDS as a public health threat by 2030; however, by 2018, HIV still accounted for approximately 770,000 deaths globally (2–4).

In Indonesia, HIV prevalence continues to rise. By 2021, national surveillance data recorded 427,201 cases of HIV infection and 131,417 cases of AIDS. Bali Province ranked sixth nationally, with 31,363 reported cases. Hospital-based registry data from Prof. Dr. I.G.N.G. Ngoerah General Hospital documented 16 new HIV cases between July and September 2022, further highlighting HIV as a persistent local public health concern (5,6).

Beyond viral replication, nutritional status plays a critical role in HIV disease progression. Both early- and late-stage HIV infection adversely affect micronutrient status due to increased metabolic requirements and impaired nutrient absorption (7). Micronutrient deficiencies, particularly involving vitamins A, D, E, and zinc, impair im-

mune responses, promote oxidative stress, accelerate $CD4^+$ T-cell apoptosis, and indirectly stimulate HIV replication (8–10). Despite the substantial benefits of antiretroviral therapy (ART), weight loss and wasting remain prevalent, and the clinical impact of micronutrient supplementation remains controversial (5,11).

This narrative review synthesizes current evidence on the immunomodulatory roles of vitamins A, D, E, and zinc in HIV infection, with particular emphasis on their immunological effects, clinical outcomes, and key research gaps.

METHOD

This study was conducted as a narrative review by synthesizing evidence from previously published studies and official guidelines. Literature searches were performed in PubMed, Scopus, and Google Scholar, as well as in relevant reports from the World Health Organization (WHO) and the Indonesian Ministry of Health, for articles published between January 2000 and December 2024. The search employed the following Boolean keywords: ('HIV' OR 'Human Immunodeficiency Virus') AND ('micronutrients' OR 'vitamin A' OR 'vitamin D' OR 'vitamin E' OR 'zinc'). The search was limited to English-language articles and studies involving human subjects.

A total of 847 articles were initially identified. After removal of duplicates (n = 203), 644 titles and abstracts were screened. Full-text review was conducted for 112 articles that met the preliminary inclusion criteria. Ultimately, 42 articles were included in the narrative synthesis.

The inclusion criteria comprised peer-reviewed articles published in English, including randomized controlled trials, cohort studies, systematic reviews, and narrative reviews that examined the association between micronutrient status and HIV infection. Articles were excluded if they did not provide full-text access, were not written in English, involved animal models, or consisted of editorials or commentaries unrelated to the role of micronutrients in HIV. Extracted findings were analyzed narratively to describe the effects of micronutrient deficiencies and supplementation on immune function, HIV disease progression, and treatment outcomes. No formal statistical pooling or meta-analysis was performed because of heterogeneity in study designs, populations, and outcome measures across the included studies.

RESULT AND DISCUSSION

Micronutrient Deficiency in HIV Infection

Micronutrient deficiency is a well-documented complication across all stages of HIV infection, including asymptomatic disease. Serum concentrations of fat-soluble vitamins (A, D, and E) and trace elements such as zinc are frequently lower in people living with HIV than in HIV-negative controls (12,13). Multiple mechanisms contribute to this phenomenon, including malabsorption due to chronic diarrhea and intestinal barrier disruption, increased metabolic demand resulting from systemic inflammation and fever, anorexia (including ART-related nausea and esophagitis), and drug–nutrient interactions that alter nutrient utilization and excretion (14–16).

Prospective observational evidence has linked micronutrient insufficiency with immune decline. In a cohort of drug users followed for 18 months, reductions in CD4 counts were strongly associated with evolving deficiencies in vitamins A and B12. Low vitamin E and B12 levels were also observed among individuals who progressed to AIDS. Selenium and zinc depletion have similarly been associated with accelerated HIV disease progression and increased mortality (12,13). Collectively, these findings suggest that micronutrient deficiencies are not merely epiphenomena of malnutrition but active contributors to immune dysfunction and adverse clinical outcomes in HIV.

Table 1. WHO Clinical Staging of HIV Infection (17).

Stage	Core Features
Stage 1 – Asymp-	No weight loss; no symptoms, or only persistent generalized lymphadenopathy.
Stage 2 – Mild disease	Weight loss of 5–10%; recurrent upper respiratory tract infections; herpes zoster; angular cheilitis; recurrent oral ulcers; pruritic papular eruption; seborrheic dermatitis; fungal nail infec-
Stage 3 – Advanced disease	Weight loss >10%; chronic diarrhea (>1 month) and/or unexplained fever (>1 month); oral or vaginal candidiasis; oral hairy leukoplakia; pulmonary tuberculosis; severe bacterial infections;
Stage 4 – Severe disease (AIDS)	HIV wasting syndrome; Pneumocystis pneumonia; chronic herpes simplex ulcers (>1 month); esophageal candidiasis; extrapulmonary tuberculosis; Kaposi's sarcoma; CMV retinitis; cerebral toxoplasmosis; HIV encephalopathy; cryptococcal meningitis; disseminated non-tuberculous mycobacterial infection; progressive multifocal leukoencephalopathy (PML); invasive cervical carcinoma; primary CNS lymphoma; peripheral neuropathy or cardiomyopathy attributable to HIV.

Table 2. Summary of Key Micronutrients in HIV Infection.

Micronutrient	Deficiency Prevalence in HIV	Key Mechanism(s)	Supplementation Outcomes	Guideline Recommendation
Vitamin A	12–19% in asymptomatic adults; up to 63% in HIV-infected pregnant women (Malawi)	Maintains epithelial integrity; regulates T-cell differentiation (Th2/Th17, Treg); enhances NK-cell activity and antibody responses via retinoic acid signaling	Modest reduction in MTCT reported in meta-analyses; no consistent effect on CD4 count or viral load; benefits appear greatest in VAD-endemic settings	WHO endorses supplementation for infants and children in VAD-endemic settings; no HIV-specific adult dosing recommendations
Vitamin D	Widespread, particularly among individuals receiving ART and those with limited sun exposure	Suppresses T-cell proliferation via VDR–RXR binding; promotes macrophage autophagy and inhibits HIV replication; activates cathelicidin-dependent antiviral pathways	Significantly increases 25(OH)D levels ($p < 0.0001$); no significant effect on CD4 count or viral load in multiple RCTs	Supplementation 400–2000 IU/day for extra-skeletal benefits; important for bone health and possible TB risk reduction
Vitamin E	Common; low α-tocopherol levels associated with wasting and higher viral loads	Principal lipid-phase antioxidant; terminates peroxyl radical propagation; regenerated by vitamin C; reduces ART-associated oxidative stress	Inconsistent; some cohorts show no benefit; paradoxical findings (higher pre-infection levels linked to greater mortality in one Kenyan cohort)	No HIV-specific dosing recommendations; nutritional adequacy should be ensured (RDA ≈15 mg/day; UL 1000 mg/day)
Zinc	29% frank deficiency among hospitalized AIDS patients; 21% borderline deficiency	Essential for thymic development, T-cell maturation, cytokine balance (Th1/Th2), phagocytosis, and oxidative burst regulation	≈0.4 mg/kg/day increased CD4/CD3 and weight in adults with AIDS; intakes >11.6 mg/day associated with faster disease progression	Target documented deficiency; avoid chronic high dosing; use thymulin activity as sensitive biomarker where available

Vitamin A and HIV

Vitamin A maintains epithelial integrity, shapes T-cell differentiation (Th2/ Th17 and Treg), enhances NK-cell activity and phagocytosis, and supports antibody responses via retinoic acid signaling (18,19). In HIV, a bidirectional cycle exists in which infection impairs appetite, absorption, utilization, and retention of nutrients, while poor nutritional status increases infection risk and severity (14,15). Vitamin A deficiency has been documented in 12–19% of asymptomatic HIV-positive adults; in Malawi, 63% of 474 HIV-infected pregnant women were deficient, and approximately 70% of their infants, regardless of HIV status, were born deficient (16,20). Deficiency correlates with diarrhea, respiratory infections, and more rapid HIV progression (14,15).

A meta-analysis reported that antenatal vitamin A modestly reduced mother-to-child transmission (MTCT), although the effects were heterogeneous across trials (18). A single study from Zimbabwe suggested that a postpartum 200,000-IU dose administered within ≤8 weeks reduced breast-milk transmission (21). However, several cross-sectional and intervention studies found no consistent effects on CD4 counts or viral load (14–16). Current WHO guidance endorses specific dosing for infants and children in VAD-endemic settings but does not provide HIV-specific dosing recommendations for adults (20). In practice, vitamin A should be used to correct proven deficiency as part of comprehensive nutritional support alongside ART.

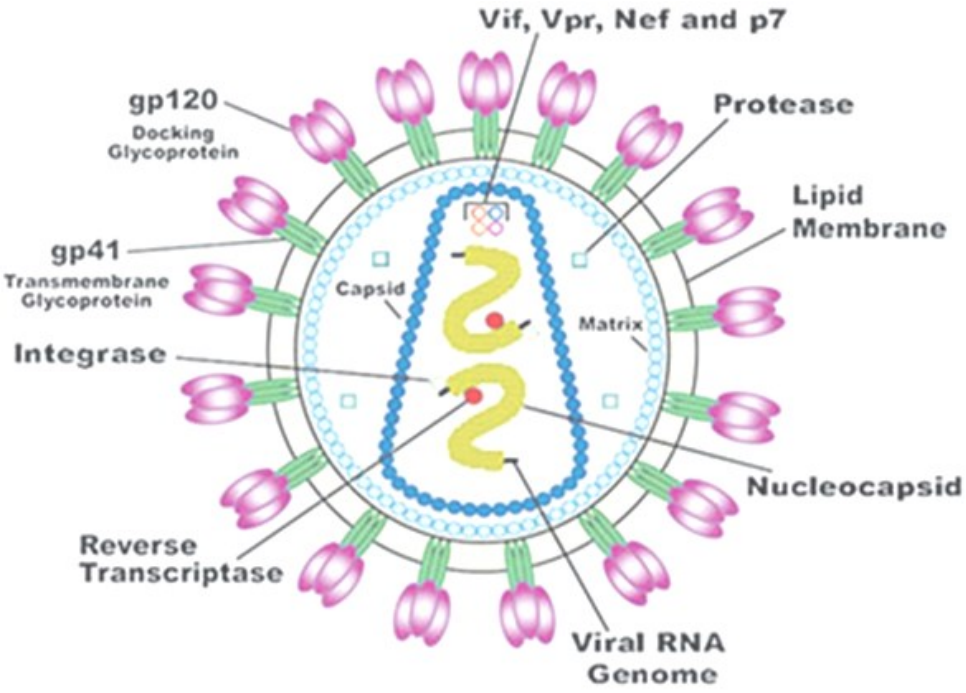

Figure 1. Structure of Human Immunodeficiency Virus (22).

Vitamin D and HIV

Beyond skeletal health, 1,25(OH)$_2$D$_3$ exerts potent immunomodulatory effects: it suppresses T-cell proliferation and IL-2/IFN-γ expression via VDR–RXR binding to VDREs, enhances regulatory T-cell suppression, and promotes autophagy in macrophages—mechanisms that may inhibit HIV replication (18,19). In vitro and ex vivo studies implicate cathelicidin-dependent, TLR-8–linked pathways in vitamin D-mediated antiviral autophagy, including in HIV–TB coinfection (23). HIV itself can perturb vitamin D metabolism and epigenetically downregulate VDR, potentially promoting T-cell apoptosis

(24,25).

Clinical trials in HIV have shown robust biochemical responses with limited immunovirologic change. In U.S. youth with HIV, bimonthly 100,000-IU cholecalciferol plus daily calcium significantly increased 25(OH)D ($p < 0.0001$) but did not alter CD4 count ($p = 0.18$), CD4% ($p = 0.09$), viral load ($p = 0.66$), or ART failure over 12 months (29). Similar findings were reported in Danish adults and Italian children, in whom 25(OH)D improved significantly without meaningful CD4 gains (30,31). Despite modest immune effects, vitamin D remains important for bone health and may reduce TB risk, both of which are clinically relevant in HIV management.

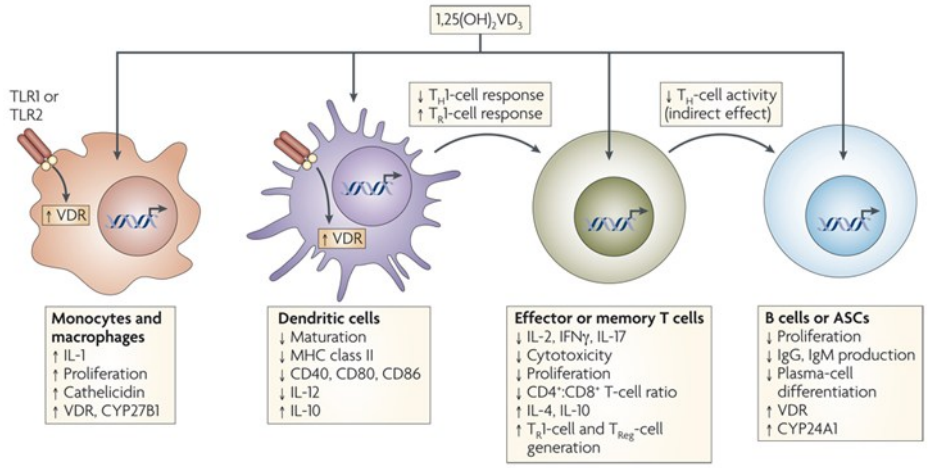

Figure 2. Mechanism of Vitamin D Immunomodulation (26)

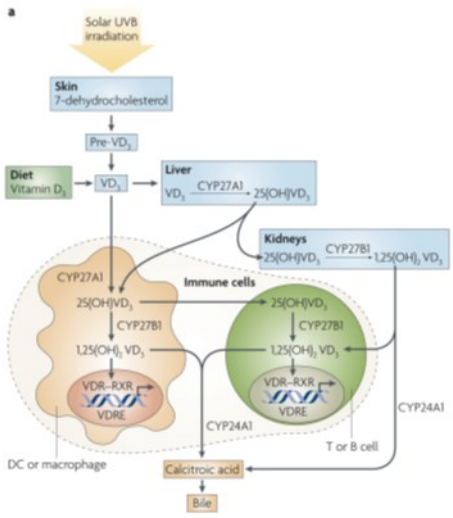

Figure 3. Vitamin D Metabolism (26)

Vitamin E and HIV

Vitamin E (α-tocopherol) is the principal lipid-phase, chain-breaking antioxidant that terminates peroxyl radical propagation and is recycled back to its reduced state by vitamin C (32). In HIV, oxidative stress driven by both the virus and ART depletes vitamin E; low α-tocopherol levels are associated with wasting, higher viral loads, and impaired immune recovery (34,35). However, outcomes with supplementation are inconsistent. The Baltimore cohort did not show a clear link between low vitamin E and AIDS progression, whereas a Kenyan cohort study paradoxically associated higher pre-infection vita-

min E levels with greater mortality in women (35). These apparently conflicting findings likely reflect differences in baseline oxidative stress, dosing, timing, and confounding by lipid levels. Current WHO guidance does not provide HIV-specific vitamin E dosing recommendations; therefore, nutritional adequacy should be ensured (adult RDA ≈15 mg/day; UL 1000 mg/day) (32,36).

Zinc and HIV

Zinc is indispensable for thymic development, T-cell maturation, cytokine balance (Th1/Th2), chemotaxis, phagocytosis, and regulation of the oxidative burst (37,38). In HIV, zinc deficiency is common and is exacerbated by acute-phase sequestration, malabsorption, anorexia, and diarrhea. In 228 hospitalized AIDS patients, 29% had frank zinc deficiency and 21% had borderline levels; low baseline zinc correlated with higher rates of bacterial infections and mortality. Dietary zinc intake has been positively associated with CD4 counts, and lower baseline serum zinc has been shown to predict progression to AIDS in prospective cohorts (12).

Interventions show benefit but require caution in dosing. In adults with AIDS, approximately 0.4 mg/kg/day for 10 months increased CD4 and CD3 counts and produced clinically relevant weight gain. In children with HIV, 1.8–2.2 mg/kg/day for 3–4 weeks improved CD4 counts in some studies and enhanced clinical scores. However, daily intakes exceeding approximately 11.6 mg/day in one cohort of HIV-1-seropositive men were associated with faster disease progression (12,13). Therefore, zinc repletion should target documented deficiency and avoid chronic high-dose supplementation.

Single versus Multiple Micronutrient Supplementation

Evidence comparing single-nutrient versus multiple-micronutrient strategies remains heterogeneous. A comprehensive systematic review reported that routine multiple micronutrient (MMN) supplementation had little or no effect on mortality in adults living with HIV (low-certainty evidence) across ART-naïve, ART-treated, and TB-coinfected populations. Over periods of up to two years, MMN supplementation did not consistently improve mean CD4 counts (low-certainty evidence) or viral load (moderate-certainty evidence) (39). A Botswana randomized controlled trial noted a reduced risk of CD4 decline to <250 cells/mm³ over two years only in the high-dose MMN plus selenium arm, with no consistent effect on viral load or mortality, and this finding has had limited replication in similar trials (40).

Trials of single or dual micronutrient supplementation generally did not demonstrate clinically important effects on mortality, hospitalization, or persistent diarrhea; benefits were largely restricted to biochemical correction (41,42). These findings underscore that micronutrients should be positioned as adjuncts to ART within comprehensive nutritional support tailored to individual deficiencies and local dietary contexts (39).

LIMITATIONS

Several limitations of this review merit acknowledgment. First, the included studies were heterogeneous in design (randomized controlled trials, cohort studies, and narrative reviews), duration, micronutrient doses, and study populations, precluding formal statistical pooling and limiting the generalizability of the conclusions. Second, as a narrative review, this synthesis is subject to potential selection bias in article inclusion, and the absence of a meta-analytic approach means that effect estimates cannot be precisely quantified. Third, the majority of the evidence derives from Sub-Saharan African and Western populations, with a notable paucity of data from Southeast Asian populations, including Indonesia, where HIV burden, dietary patterns, and baseline micronutrient status may differ substantially. Future studies from these regions are warranted to inform locally applicable clinical guidelines.

CLINICAL IMPLICATIONS

The findings of this review carry several practical implications for clinicians managing patients with HIV. Routine multiple micronutrient supplementation is not recommended for all patients with HIV; rather, supplementation should be targeted to those with documented deficiencies confirmed through appropriate laboratory assessment. Vitamin D screening should be considered in patients with HIV who have low bone density, prolonged ART use, or a high risk of TB, given the consistent biochemical response to supplementation despite the absence of marked immunologic benefit. Zinc supplementation should be used with caution and should not exceed approximately 11.6 mg/day to avoid potential adverse effects on disease progression. Vitamin A supplementation remains relevant primarily for the correction of documented deficiency in VAD-endemic settings, particularly in pregnant women and children. Overall, micronutrient interventions should be integrated into a comprehensive nutritional assessment framework as part of holistic HIV care, rather than applied as uniform supplementation protocols.

CONCLUSION

This narrative review highlights that deficiencies in vitamins A, D, E, and zinc are common among individuals living with HIV and negatively affect disease progression, immune function, and clinical outcomes. While supplementation may improve nutritional and immunological status, consistent evidence regarding long-term survival and virological control remains limited. These findings support the role of micronutrients as adjunctive, but not substitute, interventions alongside antiretroviral therapy. Further research is required to determine optimal supplementation strategies, clarify long-term clinical benefits, and establish standardized recommendations for integrating micronutrient support into comprehensive HIV management.

ACKNOWLEDGEMENT

The authors would like to express their gratitude to the Faculty of Medicine and Health Sciences, Universitas Warmadewa, for providing academic support and access to reference materials during the preparation of this manuscript. Appreciation is also extended to colleagues who provided constructive feedback during the early stages of writing.

CONFLICT OF INTEREST

The authors declare no conflict of interest related to the preparation and publication of this manuscript.

FUNDING STATEMENT

This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The work was conducted independently by the authors as part of academic requirements.

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