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Interleukin-8 Expression Under Highly Effective Host Immunological Control of HIV-1 Disease Progression

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Abstract- Background: ***Interleukin-8 (IL-8) is a pro-inflammatory chemokine involved in neutrophil recruitment and immune activation and has been implicated in HIV-1–associated inflammation and disease progression. Despite effective highly active antiretroviral therapy (HAART), residual immune activation persists in many people living with HIV, and the role of IL-8 across different stages of infection and treatment remains incompletely understood, particularly in sub-Saharan Africa.***

Methods: ***This cross-sectional study enrolled 40 participants in Osogbo, South-Western Nigeria, comprising HIV-negative controls (n=10), HAART-naïve HIV-positive individuals (OFF-HAART, n=10), HAART-treated individuals (ON-HAART, n=10), and AIDS progressors (n=10). Serum IL-8 levels were quantified using enzyme-linked immunosorbent assay, while CD4+ T-cell counts were determined by flow cytometry. Viral load, hematological indices, and selected biochemical parameters were also assessed. Associations between IL-8, immunological markers, and socio-demographic variables were analyzed using correlation and comparative statistics.***

Results: ***Interleukin-8 (IL-8) levels showed a downward trend from HIV-negative subjects (175.89 ± 84.58 pg/mL) and OFF-HAART individuals (211.14 ± 64.55 pg/mL) to ON-HAART participants (125.08 ± 39.73 pg/mL), with the lowest levels observed in AIDS progressors (11.40 ± 1.14 pg/mL), although this trend did not reach statistical significance ($p = 0.088$). $CD4^+$ T-cell counts differed significantly across groups ($p < 0.001$), with marked depletion in OFF-HAART subjects (166.10 ± 41.40 cells/μL) and partial immune reconstitution in ON-HAART individuals (395.20 ± 61.69 cells/μL). Among AIDS progressors, longer infection duration was significantly associated with higher IL-8 expression ($\chi^2 = 6.667$, $p = 0.010$).***

Conclusion: ***Elevated IL-8 is linked to impaired immune status and disease progression in HIV-1 infection, while reduced IL-8 accompanies immune recovery and advanced disease stages. IL-8 may serve as a useful biomarker of immune dysregulation and progression across HIV disease states, supporting its potential role in monitoring inflammation alongside conventional immunological markers.***

I. INTRODUCTION

Human Immunodeficiency Virus type 1 (HIV-1) remains a major global public health challenge, particularly in sub-Saharan Africa, where it contributes significantly to morbidity and mortality . In 2024, UNAIDS reported approximately 29.4 million people living with HIV in this region, representing nearly two-thirds of the global burden (UNIAD, 2025). HIV infection leads to progressive depletion of CD4+ T-lymphocytes, resulting in impaired cellular immunity and increased susceptibility to opportunistic infections, ultimately culminating in acquired immunodeficiency syndrome (AIDS) (Deeks et al., 2013). The introduction of highly active antiretroviral therapy (HAART) has

dramatically improved survival, immune restoration, and quality of life for people living with HIV (PLHIV), transforming a once-fatal infection into a manageable chronic condition (Palella et al., 2006)
Despite the clinical success of HAART, residual immune activation and chronic inflammation persist in many individuals, even under effective viral suppression (Mu et al., 2024). This persistent inflammation is associated with a spectrum of comorbidities, including cardiovascular disease, metabolic syndrome, neurocognitive impairment, and accelerated aging (Teer et al., 2025). Cytokines such as Interleukin-8 (IL-8), a pro-inflammatory chemokine involved in neutrophil recruitment and activation, have emerged as important markers of immune activation in HIV infection. Elevated IL-8 levels have been reported in untreated HIV patients and are linked to disease progression, while reductions are observed with successful HAART, though not always returning to baseline levels observed in HIV-negative individuals (Mtshali et al., 2021).

IL-8, also known as CXCL8, plays a critical role in mediating innate immune responses. It is produced by monocytes, macrophages, endothelial cells, and T-cells in response to viral proteins and inflammatory stimuli. In the context of HIV, viral proteins such as Tat and Nef can directly induce IL-8 production via activation of nuclear factor kappa B (NF-κB) and mitogen-activated protein kinase (MAPK) signaling pathways ((Xiong et al., 2022). Persistent elevation of IL-8 contributes to chronic immune activation, which may impair CD4+ T-cell recovery, exacerbate tissue inflammation, and promote comorbid conditions. Therefore, IL-8 has potential utility as a biomarker for monitoring inflammation, immune dysregulation, and disease progression in HIV-infected individuals, particularly in those on long-term HAART.

Socio-demographic factors, including age, gender, and duration of infection, significantly influence immune response and disease trajectory. Younger individuals generally exhibit more robust CD4+ T-cell recovery following ART initiation compared to older adults, reflecting the impact of immune senescence on treatment outcomes (Means et al., 2016). Gender-based differences in immune activation have also been reported; for instance, women often display higher immune activation despite comparable viral suppression, potentially influencing progression rates and comorbidity risk (Means *et al.,* 2016). Understanding the interplay between demographic factors, viral dynamics, therapy exposure, and inflammatory biomarkers is therefore critical for optimizing HIV management strategies.

In Nigeria, HIV remains a significant health concern, with treatment programs expanding under the National HIV/AIDS Strategic Framework. However, limited studies have investigated the role of inflammatory markers, such as IL-8, in predicting immune recovery or disease progression in treated and untreated individuals. Examining these markers in relation to HAART exposure, CD4+ T-cell counts, and socio-demographic variables provides insights into residual immune dysregulation, treatment effectiveness, and potential therapeutic interventions.
This study, therefore, aimed to evaluate the immunological and biochemical profiles of HIV-infected individuals at different stages of disease and treatment, with a particular focus on IL-8 expression. By comparing HAART-naïve, HAART-experienced, and AIDS progressors with HIV-negative controls, the study investigates the dynamics of immune activation, cytokine regulation, and their relationship with clinical outcomes.

II. MATERIALS AND METHOD

2.1 Methodology

A total of forty participants were enrolled in this study, comprising thirty HIV-1–infected individuals receiving care at the Hope Clinic, State Specialist Hospital, Asubiaro, Osogbo, Osun State, South-Western Nigeria, and ten HIV-negative individuals who served as controls. Among the HIV-positive participants, 50% were on effective oral highly active antiretroviral therapy (HAART), consisting of Tenofovir (300 mg/day), Lamivudine (150 mg/day), and Efavirenz (600 mg/day). Pregnant women and individuals co-infected with tuberculosis and/or hepatitis viruses were excluded. HIV-positive participants who were HAART-naïve were asymptomatic and had CD4+ T-cell counts greater than 200 cells/μL at the time of enrollment. Ethical

approval for the study was obtained from the Research Ethics Committee of the Osun State Hospitals Management Board, and written informed consent was secured from all participants.

Determination of Interleukin- 8

Serum concentration of IL-8 was determined using stardard commercially prepared kit purchased purchased from Raybiotech USA. The procedure was carried out according to manufacturer's instructions.

Sampling, Processing and Preservation

Blood sampling was done by venepunture while using appropriate standard method. Specimen was transported under ice- cold condition to the laboratory within one hour. Serum was separated from the whole blood by centrifugation at 1,000rpm for ten minutes and stored at -70^{0}c plasma was also stored at -70^{0}c.

Diagnosis of HIV Infection and CD4 T-cell cytometry

The diagnosis of HIV-1 infection was performed using Immunochromatographic membrane assay and confirmed by Enzyme linked Immunosorbent assay (USA). Subjects with indeterminate results were excluded from the study. Control subjects were also confirmed to be negative for antibodies to HIV. EDTA-anticougulated blood CD4T-cell was enumerated using Cyflow® Cytometer according to the manufacturer's instructions (Partec, Germany).

Statistical Analysis

Data were analyzed using graph prism version 5 software package (San Diego, CA) to determine Spearman correlation and Fisher's test were used to test the association between two variables. Results were expressed as Mean ± Standard Error of Means (SEM). The level of statistical significance were considered $p<0.05$.

Table 1: Socio-demographic and Clinical Characteristics of Participants by Study Group

Variable	Category	HIV-negative (n=10)	OFF-HAART (n=10)	ON-HAART (n=10)	AIDS progressors (n=10)	χ^2 (df), *p*-value
Age (years)	≤ 30	0 (0.0%)	0 (0.0%)	1 (10.0%)	6 (60.0%)	20.783 (6), 0.002
	31–50	9 (90.0%)	8 (80.0%)	5 (50.0%)	3 (30.0%)	
	≥ 51	1 (10.0%)	2 (20.0%)	4 (40.0%)	1 (10.0%)	
Gender	Male	3 (30.0%)	1 (10.0%)	4 (40.0%)	8 (80.0%)	10.833 (3), 0.013
	Female	7 (70.0%)	9 (90.0%)	6 (60.0%)	2 (20.0%)	
Infection duration (years)	1–10	–	–	6 (60.0%)	5 (50.0%)	0.202 (1), 0.653
	11–20	–	–	4 (40.0%)	5 (50.0%)	
HAART duration (years)	< 5	–	–	6 (60.0%)	1 (10.0%)	11.238 (3), 0.011
	6–10	–	–	0 (0.0%)	4 (40.0%)	
	11–15	–	–	4 (40.0%)	2 (20.0%)	
	16–20	–	–	0 (0.0%)	3 (30.0%)	

Nature of HAART: Tenofovir (300 mg/day), lamivudine (150 mg/day) and Efaviren (600 mg/day)

Table 1 shows significant socio-demographic and clinical differences across the study groups. Age distribution varied markedly by group (χ^2 = 20.783, df = 6, p = 0.002), with AIDS progressors predominantly ≤30 years (6; 60.0%), while HIV-negative (9; 90.0%) and OFF-HAART participants (8; 80.0%) were mainly aged 31–50 years. The ON-HAART group demonstrated a more even age spread, with a higher proportion aged ≥51 years (4; 40.0%). Gender distribution also differed significantly (χ^2 = 10.833, df = 3, p = 0.013), with females predominating among HIV-negative (7; 70.0%) and OFF-HAART (9; 90.0%) groups, whereas males were more common among AIDS progressors (8;

80.0%). Infection duration did not differ significantly between ON-HAART and AIDS progressors (χ^2 = 0.202, df = 1, p = 0.653), with similar proportions reporting 1–10 and 11–20 years of infection. In contrast, HAART duration showed a significant association with disease group (χ^2 = 11.238, df = 3, p = 0.011); most ON-HAART participants had received therapy for <5 years (6; 60.0%), whereas AIDS progressors more frequently had longer treatment durations, particularly 6–10 years (4; 40.0%) and 16–20 years (3; 30.0%). All treated participants received a standard Tenofovir–Lamivudine–Efavirenz regimen. Table 2 demonstrates marked differences in immunological and selected biochemical markers across the study groups. CD4 count and percentage varied significantly among groups (p = 0.000), with OFF-HAART subjects showing profound immunosuppression (166.10 ± 41.40 cells/µL; 1.66 ± 0.41%), while ON-HAART participants exhibited partial immune reconstitution (395.20 ± 61.69 cells/µL). Notably, AIDS progressors recorded the highest CD4 values (782.60 ± 60.41 cells/µL; 7.83 ± 0.60%), exceeding even HIV-negative subjects, suggesting possible immune activation or redistribution effects. Viral load differed significantly across infected groups (p = 0.002), being markedly elevated in OFF-HAART and ON-HAART subjects compared with HIV-negative controls, while values were not available for AIDS progressors. In contrast, total white blood cell count, neutrophil indices, lymphocyte percentages, myeloperoxidase activity, total protein, and globulin levels did not differ significantly between groups (p > 0.05), indicating relative stability of these parameters irrespective of treatment status. Interleukin-8 levels showed a progressive decline from HIV-negative and OFF-HAART subjects to AIDS progressors, although this trend did not reach statistical significance (p = 0.088). Albumin levels differed significantly across groups (p = 0.018), with lower concentrations observed in OFF-HAART subjects and relatively higher levels in AIDS progressors and HIV-negative controls.

Table 2: Immunological and Biochemical Markers Among Study Groups

Parameter	HIV Negative Subjects	OFF-HAART Subjects	ON-HAART Subjects	AIDS Subjects (Progressors)	p-Value
CD4 (cells/µL)	603.80 ± 69.18	166.10 ± 41.40	395.20 ± 61.69	782.60 ± 60.41	0.000
% CD4	6.04 ± 0.69	1.66 ± 0.41	3.95 ± 0.62	7.83 ± 0.60	0.000
Viral Load (copies/mL)	4202.70 ± 716.26	299173.90 ± 72209.63	171861.80 ± 57852.45	–	0.002
WBC (cells/µL)	4480.00 ± 460.87	4460.00 ± 543.90	4290.00 ± 498.32	3920.00 ± 367.21	0.824
Neutrophil (%)	46.20 ± 5.06	50.10 ± 5.76	41.00 ± 4.32	40.30 ± 4.30	0.455
Absolute Neutrophil	2123.40 ± 353.88	2366.10 ± 433.31	1794.00 ± 303.98	1584.80 ± 232.80	0.385
Lymphocyte (%)	53.80 ± 5.06	49.90 ± 5.76	59.00 ± 4.32	59.70 ± 4.30	0.455
Interleukin-8 (pg/mL)	175.89 ± 84.58	211.14 ± 64.55	125.08 ± 39.73	11.40 ± 1.14	0.088
Myeloperoxidase U	0.11 ± 0.02	0.08 ± 0.02	0.08 ± 0.02	0.07 ± 0.02	0.629
Total Protein (g/dL)	80.59 ± 3.51	79.15 ± 3.93	167.99 ± 80.84	85.28 ± 5.83	0.349
Albumin (g/dL)	56.46 ± 1.64	49.43 ± 2.88	52.84 ± 1.62	58.59 ± 1.85	0.018
Globulin (g/dL)	24.13 ± 3.57	29.72 ± 4.28	115.15 ± 81.27	27.69 ± 4.15	0.336

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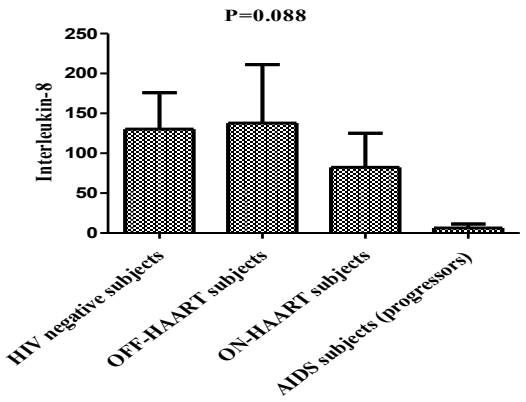


Figure 1: Interleukin-8 Levels Across Study Groups

Subject / Group	HIV-Negative Subjects	OFF-HAART Subjects	ON-HAART Subjects	AIDS Subjects (Progressors)	P-Value
Interleukin-8	175.89 ± 84.58	211.14 ± 64.55	125.08 ± 39.73	11.40 ± 1.14	0.088

Figure 1 illustrates a progressive reduction in interleukin-8 (IL-8) levels across the study groups, from HIV-negative subjects through OFF-HAART and ON-HAART participants to AIDS progressors. The highest IL-8 concentrations were observed among OFF-HAART subjects (211.14 ± 64.55 pg/mL), followed by HIV-negative controls (175.89 ± 84.58 pg/mL), while ON-HAART subjects exhibited comparatively lower levels (125.08 ± 39.73 pg/mL). In contrast, AIDS progressors showed markedly suppressed IL-8 levels (11.40 ± 1.14 pg/mL). Table 3 presents the correlation analysis between interleukin-8 (IL-8) levels and immune cell indices among AIDS progressor subjects. IL-8 showed weak, non-significant correlations with age ($r = 0.383$, $p = 0.274$), CD4 count ($r = -0.096$, $p = 0.793$), WBC ($r = 0.310$, $p = 0.384$), neutrophil percentage ($r = -0.253$, $p = 0.480$), absolute neutrophil count ($r = -0.060$, $p = 0.870$), lymphocyte percentage ($r = 0.253$, $p = 0.480$), and myeloperoxidase activity ($r = 0.196$, $p = 0.588$), indicating no statistically significant associations between IL-8 and these parameters. In contrast, strong and significant interrelationships were observed among immune cell indices themselves. Neutrophil percentage correlated positively with absolute neutrophil count ($r = 0.821$, $p = 0.004$) and showed a perfect inverse correlation with lymphocyte percentage ($r = -1.000$, $p = 0.000$). Similarly, absolute neutrophil count was strongly and negatively associated with lymphocyte percentage ($r = -0.821$, $p = 0.004$). Table 4 summarizes the correlation between interleukin-8 (IL-8), CD4 count, and hematological indices among ON-HAART subjects. IL-8 showed weak and non-significant correlations with age ($r = -0.041$, $p = 0.910$), CD4 count ($r = -0.354$, $p = 0.315$), WBC ($r = 0.020$, $p = 0.957$), neutrophil percentage ($r = -0.145$, $p = 0.689$), absolute neutrophil count ($r = -0.054$, $p = 0.883$), lymphocyte percentage ($r = 0.145$, $p = 0.689$), and myeloperoxidase activity ($r = -0.187$, $p = 0.604$), indicating no significant association between IL-8 and immune cell indices in treated individuals. In contrast, significant relationships were observed among hematological parameters themselves. WBC correlated strongly and positively with absolute neutrophil count ($r = 0.770$, $p = 0.009$), while neutrophil percentage correlated positively with absolute neutrophil count ($r = 0.749$, $p = 0.013$) and showed a perfect inverse correlation with lymphocyte percentage ($r = -1.000$, $p = 0.000$). Absolute neutrophil count also correlated negatively with lymphocyte percentage ($r = -0.749$, $p = 0.013$).

Table 3: Associations Between Interleukin-8 and Immune Cell Indices in AIDS Progressor Subjects

AIDS subjects (progressors)		Interlukin 8	Age	CD4	WBC	Neutrophil	Absolute neutrophil	Lmphocyte	Myeloperoxidase
Interlukin8	r	1	0.383	-0.096	0.310	-0.253	-0.060	0.253	0.196
	p		0.274	0.793	0.384	0.480	0.870	0.480	0.588

Age	r	0.383	1	0.001	-0.420	-0.333	-0.452	0.333	0.349
	p	0.274		0.998	0.227	0.347	0.189	0.347	0.323
CD4	r	-0.096	0.001	1	-0.063	0.247	0.271	-0.247	-0.391
	p	0.793	0.998		0.863	0.492	0.448	0.492	0.264
WBC	r	0.310	-0.420	-0.063	1	0.035	0.579	-0.035	-0.283
	p	0.384	0.227	0.863		0.923	0.079	0.923	0.427
Neutrophil	r	-0.253	-0.333	0.247	0.035	1	.821**	-1.000**	0.222
	p	0.480	0.347	0.492	0.923		0.004	0.000	0.537
Absolute neutrophil	r	-0.060	-0.452	0.271	0.579	.821**	1	-.821**	-0.026
	p	0.870	0.189	0.448	0.079	0.004		0.004	0.944
Lmphocyte	r	0.253	0.333	-0.247	-0.035	-1.000**	-.821**	1	-0.222
	p	0.480	0.347	0.492	0.923	0.000	0.004		0.537
Myeloperoxidase	r	0.196	0.349	-0.391	-0.283	0.222	-0.026	-0.222	1
	p	0.588	0.323	0.264	0.427	0.537	0.944	0.537	

Correlation is significant at the 0.05 level (2-tailed).
. Correlation is significant at the 0.01 level (2-tailed).

Table 4: Correlation Analysis of Interleukin-8 with CD4 Count and Hematological Indices in ON-HAART Subjects

ON-HAART subjects		Interlukin 8	Age	CD4	WBC	Neutrophil	Absolute neutrophil	Lmphocyte	Myeloperoxidase
Interlukin8	r	1	-0.041	-0.354	0.020	-0.145	-0.054	0.145	-0.187
	p		0.910	0.315	0.957	0.689	0.883	0.689	0.604
Age	r	-0.041	1	0.220	0.255	-0.368	-0.097	0.368	0.078
	p	0.910		0.542	0.478	0.296	0.790	0.296	0.830
CD4	r	-0.354	0.220	1	0.143	-0.029	0.189	0.029	0.583
	p	0.315	0.542		0.693	0.937	0.600	0.937	0.077
WBC	r	0.020	0.255	0.143	1	0.181	.770**	-0.181	-0.431
	p	0.957	0.478	0.693		0.617	0.009	0.617	0.214
Neutrophil	r	-0.145	-0.368	-0.029	0.181	1	.749*	-1.000**	-0.400
	p	0.689	0.296	0.937	0.617		0.013	0.000	0.252
Absolute neutrophil	r	-0.054	-0.097	0.189	.770**	.749*	1	-.749*	-0.449
	p	0.883	0.790	0.600	0.009	0.013		0.013	0.194
Lmphocyte	r	0.145	0.368	0.029	-0.181	-1.000**	-.749*	1	0.400
	p	0.689	0.296	0.937	0.617	0.000	0.013		0.252

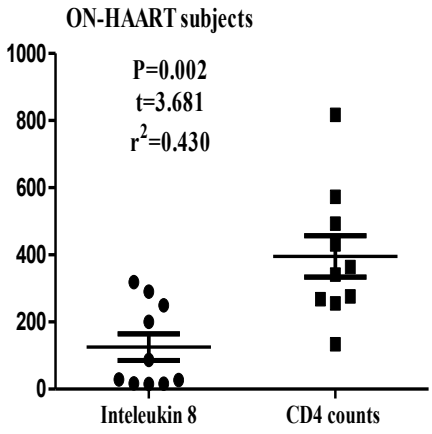
Myeloperoxidase	r	-0.187	0.078	0.583	-0.431	-0.400	-0.449	0.400	1
	p	0.604	0.830	0.077	0.214	0.252	0.194	0.252	

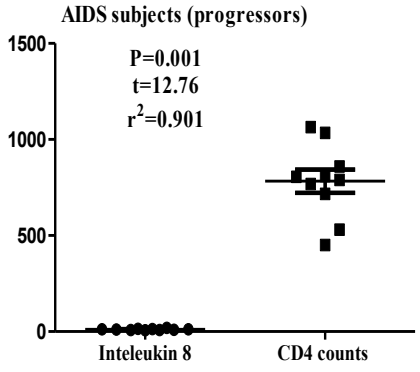
Correlation is significant at the 0.05 level (2-tailed).
. Correlation is significant at the 0.01 level (2-tailed).

Table 5 presents CD4 counts stratified by interleukin-8 (IL-8) expression across the study groups. Among HIV-negative subjects, individuals with low IL-8 levels exhibited higher mean CD4 counts (752.00 ± 135.44 cells/μL) compared with those with high IL-8 expression (505.00 ± 46.27 cells/μL). In the OFF-HAART group, all participants expressed high IL-8 levels and demonstrated markedly reduced CD4 counts (166.10 ± 41.40 cells/μL), reflecting severe immunosuppression in untreated infection. ON-HAART subjects also uniformly showed high IL-8 expression but displayed improved CD4 counts (395.20 ± 61.69 cells/μL), indicating partial immune recovery with therapy. Among AIDS progressors, CD4 counts were higher in those with low IL-8 expression (818.50 ± 90.57 cells/μL) compared with high IL-8 expressors (728.75 ± 72.48 cells/μL). In figure 2, the relationship between CD4 count and interleukin-8 varied across HIV disease stages. In HIV-negative subjects, CD4 was significantly associated with interleukin-8 (t = 3.916, df = 18, p = 0.001), explaining 46% of the variability (R^2 = 0.46). Among OFF-HAART subjects, no significant association was observed (t = 0.587, df = 18, p = 0.564; R^2 = 0.019), indicating minimal contribution of interleukin-8 to CD4 variability. In ON-HAART subjects, the association was statistically significant (t = 3.681, df = 18, p = 0.0017), with a moderate effect size (R^2 = 0.43). The strongest relationship was observed in AIDS subjects (progressors), where interleukin-8 explained 90% of CD4 variability (t = 12.76, df = 18, p < 0.0001; R^2 = 0.90), highlighting a progressive strengthening of the association with advancing disease.

Table 5: CD4 Counts According to Interleukin-8 Expression Across Study Groups

Group	Interleukin-8 Level	N	CD4 (Mean ± SEM)
HIV-Negative Subjects	High	6	505.00 ± 46.27
	Low	4	752.00 ± 135.44
OFF-HAART Subjects	High	10	166.10 ± 41.40
ON-HAART Subjects	High	10	395.20 ± 61.69
AIDS Subjects (Progressors)	High	4	728.75 ± 72.48
	Low	6	818.50 ± 90.57





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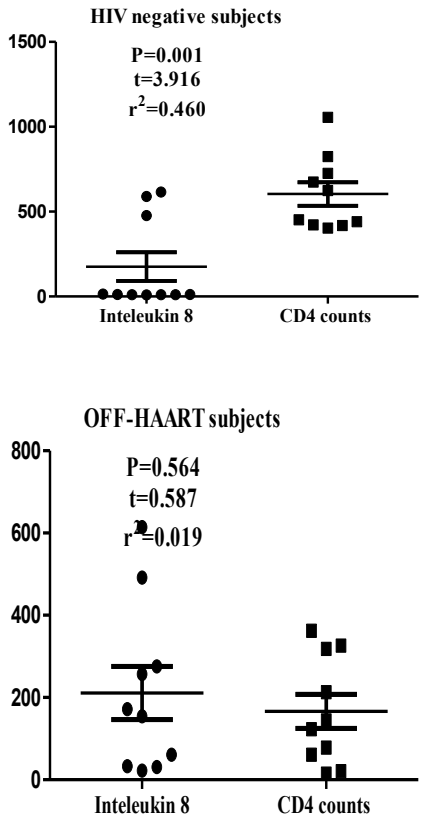

Figure 2: Stage-Dependent CD4 and IL-8 Relationship among the study group

cd4 and interleukin8	HIV negative subjects	OFF-HAART subjects	ON-HAART subjects	AIDS subjects (progressors)
P value	0.001	0.5642	0.0017	P<0.0001
t, df	t=3.916 df=18	t=0.5874 df=18	t=3.681 df=18	t=12.76 df=18
R squared	0.46	0.01881	0.4295	0.9005

Table 6 demonstrates that, among AIDS progressors, interleukin-8 (IL-8) expression is significantly associated with infection duration but not with age, gender, or HAART duration. High IL-8 expression was predominantly observed in participants with longer infection duration (11–20 years: 4, 100.0%), whereas low IL-8 levels were more common among those with shorter infection duration (1–10 years: 5, 83.3%), and this association was statistically significant ($\chi^2 = 6.667$, $p = 0.010$). Although younger age (≤30 years) and male gender were more frequent in the high IL-8 group, these differences did not reach statistical significance ($p = 0.153$ and $p = 0.197$, respectively). HAART duration showed a borderline association with IL-8 levels ($p = 0.065$), with high IL-8 expression tending to occur in individuals on long-term therapy (≥11 years), suggesting a possible cumulative inflammatory effect with prolonged infection and treatment.

Table 7 summarizes age, gender, and clinical characteristics across the broader study groups and confirms significant heterogeneity by disease and treatment status. Age distribution differed significantly across groups ($\chi^2 = 20.783$, $p = 0.002$), with AIDS progressors largely aged ≤30 years (6, 60.0%), while HIV-negative and OFF-HAART subjects were mainly within 31–50 years. Gender distribution also varied significantly ($\chi^2 = 10.833$, $p = 0.013$), with males predominating among AIDS progressors (8, 80.0%) and females among HIV-negative and OFF-HAART groups. In contrast, infection duration did not differ significantly between ON-HAART subjects and AIDS progressors ($p = 0.653$), whereas HAART duration showed a significant association with disease status ($\chi^2 = 11.238$, $p = 0.011$), indicating differing treatment exposure patterns across groups. Table 8 shows that IL-8 expression is inversely associated with CD4 counts, particularly in HIV-negative subjects and AIDS progressors, where lower IL-8 corresponds to higher CD4 levels. OFF-HAART subjects with uniformly high IL-8 had severe immunosuppression, while ON-HAART participants exhibited partial immune recovery. Viral load was highest in untreated and ON-HAART groups, reflecting ongoing viral replication, whereas hematological indices and most biochemical parameters were largely unchanged across IL-8 subgroups. Albumin differed significantly, being lower in OFF-HAART subjects, suggesting treatment and inflammation influence nutritional or inflammatory status. Overall, elevated IL-8 appears linked to impaired immunity and disease progression.

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Table 6: Demographic and Clinical Characteristics by Interleukin-8 Level AIDS subjects (progressors) (n=10)

Variable	Category	High IL-8 (n=4)	Low IL-8 (n=6)	Chi-Square (p-value)
Age (years)	≤30	3 (75.0%)	3 (50.0%)	3.750 (0.153)
	31–50	0 (0.0%)	3 (50.0%)	
	≥51	1 (25.0%)	0 (0.0%)	
Gender	Male	4 (100.0%)	4 (66.7%)	1.667 (0.197)
	Female	0 (0.0%)	2 (33.3%)	
Infection Duration (years)	1–10	0 (0.0%)	5 (83.3%)	6.667 (0.010)
	11–20	4 (100.0%)	1 (16.7%)	
HAART Duration (years)	<5	0 (0.0%)	1 (16.7%)	7.222 (0.065)
	6–10	0 (0.0%)	4 (66.7%)	
	11–15	2 (50.0%)	0 (0.0%)	
	16–20	2 (50.0%)	1 (16.7%)	

Table 7: Age and Gender Distribution by Interleukin-8 Level HIV negative subjects (n = 10)

Variable	Category	HIV Negative	OFF-HAART	ON-HAART	AIDS (Progressors)	χ^2	p-value
Age (years)	≤30	0	0	1 (10.0%)	6 (60.0%)	3	2
	31–50	9 (90.0%)	8 (80.0%)	5 (50.0%)	3 (30.0%)		
	≥51	1 (10.0%)	2 (20.0%)	4 (40.0%)	1 (10.0%)		
Gender	Male	3 (30.0%)	1 (10.0%)	4 (40.0%)	8 (80.0%)	10.833	0.013
	Female	7 (70.0%)	9 (90.0%)	6 (60.0%)	2 (20.0%)		
Infection Duration (years)	1–10	–	–	6 (60.0%)	5 (50.0%)	0.202	0.653
	11–20	–	–	4 (40.0%)	5 (50.0%)		
HAART Duration (years)	<5	–	–	6 (60.0%)	1 (10.0%)	11.238	0.011
	6–10	–	–	0 (0.0%)	4 (40.0%)		
	11–15	–	–	4 (40.0%)	2 (20.0%)		
	16–20	–	–	0 (0.0%)	3 (30.0%)		

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Table 8: Immunological and Biochemical Parameters by Group and Interleukin 8 Subgroups

Parameter	HIV Negative (High)	HIV Negative (Low)	OFF-HAART (High)	OFF-HAART (Low)	ON-HAART (High)	ON-HAART (Low)	AIDS Progressors (High)	AIDS Progressors (Low)	P-Value
Age (years)	41.67 ± 2.91	36.50 ± 1.19	43.50 ± 3.41	–	44.50 ± 3.61	–	31.25 ± 7.40	32.17 ± 4.24	0.035
CD4 (cells/μL)	505.00 ± 46.27	752.00 ± 135.44	166.10 ± 41.40	–	395.20 ± 61.69	–	728.75 ± 72.48	818.50 ± 90.57	0.000
Viral Load (copies/mL)	4671.17 ± 939.24	3500.00 ± 1169.58	299173.90 ± 72209.63	–	171861.80 ± 57852.45	–	–	–	0.002
WBC (cells/μL)	4900.00 ± 720.65	3850.00 ± 272.34	4460.00 ± 543.90	–	4290.00 ± 498.32	–	4800.00 ± 469.04	3333.33 ± 380.06	0.824
Neutrophil (%)	48.67 ± 6.50	42.50 ± 8.88	50.10 ± 5.76	–	41.00 ± 4.32	–	37.75 ± 6.25	42.00 ± 6.20	0.455
Absolute Neutrophil	2401.00 ± 494.05	1707.00 ± 482.37	2366.10 ± 433.31	–	1794.00 ± 303.98	–	1748.00 ± 177.70	1476.00 ± 379.52	0.385
Lymphocyte (%)	51.33 ± 6.50	57.50 ± 8.88	49.90 ± 5.76	–	59.00 ± 4.32	–	62.25 ± 6.25	58.00 ± 6.20	0.455
Interleukin-8 (pg/mL)	286.37 ± 123.92	10.18 ± 0.19	211.14 ± 64.55	–	125.08 ± 39.73	–	14.84 ± 1.50	9.10 ± 0.58	0.088
Myeloperoxidase (U)	0.085 ± 0.028	0.138 ± 0.023	0.077 ± 0.016	–	0.080 ± 0.021	–	0.087 ± 0.033	0.065 ± 0.026	0.629
Total Protein (g/L)	75.93 ± 4.61	87.58 ± 3.45	79.15 ± 3.93	–	167.99 ± 80.84	–	90.28 ± 7.35	81.95 ± 8.66	0.349
Albumin (g/L)	55.03 ± 0.99	58.60 ± 3.89	49.43 ± 2.88	–	52.84 ± 1.62	–	60.27 ± 2.37	57.47 ± 2.72	0.018
Globulin (g/L)	20.90 ± 4.32	28.99 ± 5.97	29.72 ± 4.28	–	115.15 ± 81.27	–	30.02 ± 6.55	26.15 ± 5.76	0.336

III. DISCUSSION

In this study, clear socio-demographic and immunological differences emerge across HIV disease stages and treatment status. Age distribution differs significantly among the groups ($\chi^2 = 20.783$, $p = 0.002$), with a higher proportion of relatively young individuals (≤ 30 years) among AIDS progressors and older adults in the HIV-negative and untreated (OFF-HAART) groups. This aligns with field evidence indicating that age influences immune recovery after antiretroviral therapy (ART) initiation, with younger patients showing better CD4+ T-cell gains (mean additional ~22 cells/μL) compared to older adults over the first year of therapy (Balestre *et al.,* 2012). Differential immune ageing also affects systemic inflammation and disease progression, underscoring age as a critical determinant of HIV immunological outcomes.

Gender differences also vary significantly between groups ($\chi^2 = 10.833$, $p = 0.013$), with male

predominance among AIDS progressors and female predominance in HIV-negative and OFF-HAART groups. Sex-specific immune responses in HIV infection have been observed, particularly in T-cell activation and inflammatory markers; women with suppressed viral loads had greater mucosal immune activation than men, which may influence progression trajectories (Santinelli *et al.,* 2020). These patterns may reflect both biological and socio-behavioural determinants of disease dynamics.

In this study, immunological markers show expected patterns of HIV immunopathogenesis, OFF-HAART subjects have profoundly low mean CD4 counts, reflecting uncontrolled viral replication and immune depletion, whereas ON-HAART participants exhibit partial CD4 recovery. This phenomenon of ART-mediated immune reconstitution is well documented: initiation of ART leads to increases in CD4+ T-cell counts and reduced immune activation, though the magnitude of recovery varies widely with baseline levels and duration on therapy (Manaye *et al.,* 2020). The significantly elevated viral loads seen in OFF-HAART and ON-HAART groups versus HIV-negative controls are consistent with ineffective viral suppression or early treatment effects prior to complete suppression, emphasizing that viremia remains a powerful driver of inflammation.

Despite ART, chronic inflammation persists. Interleukin-8 (IL-8) levels show a downward trend from untreated to treated participants and are lowest in AIDS progressors. Elevated IL-8 has been linked to chronic inflammation in HIV, including among individuals on ART who initiated therapy with low CD4 counts, reflecting incomplete resolution of immune activation (Ellwanger *et al.,* 2020). Persistent elevations in inflammatory markers such as IL-8, IL-6, and TNF-α despite viral suppression have been observed in aging ART cohorts, indicating that ART alone does not fully normalize cytokine profiles (Love *et al.,* 2024). Inflammation is also implicated in HIV-associated comorbidities such as cardiovascular disease, where chronic immune activation contributes to endothelial dysfunction and atherogenesis (Obare *et al.,* 2024).

Broad hematological indices such as total white blood cell counts and differential leukocyte percentages do not differ significantly across groups, suggesting that gross leukocyte counts are not sensitive to the subtle immune activation patterns that characterize chronic HIV infection and treatment responses. However, at the cellular subset level, ART and HIV status have been shown to influence innate lymphoid cell dysfunction and residual immune perturbation, even with long-term therapy (Nabatanzi *et al.,* 2021). These functional changes may not always be reflected in simple quantitative counts, highlighting the importance of deeper immunophenotyping in understanding disease progression.

Albumin levels vary significantly across study groups ($p = 0.018$), with lower concentrations in untreated subjects and relatively stable or higher levels in treated participants and HIV-negatives. Hypoalbuminemia is a marker of systemic inflammation and poorer prognosis in untreated HIV infection, and increases in albumin with ART reflect improvements in nutritional and inflammatory status following viremia reduction. Persistent inflammation, however, may continue to influence nutrient status and protein synthesis pathways even after prolonged therapy. The pattern of cytokine dysregulation observed in this study aligns with broader literature on HIV immunopathogenesis. HIV proteins such as Tat and Nef can directly induce pro-inflammatory cytokines including IL-8 and IL-6 via NF-κB and MAPK signaling pathways, contributing to sustained immune activation (Gunawan *et al.,* 2026). ART reduces, but does not eliminate, these signals, leading to residual inflammation that persists even with viral suppression, which has implications for immune recovery and comorbidity risk (Love *et al.,* 2024; Osuji *et al.,* 2018).

These findings underscore the multifactorial nature of immune activation in HIV, where socio-demographic factors (age, gender), viral dynamics (load and suppression), and treatment exposure converge to shape both immune reconstitution and inflammatory profiles. While ART remains the cornerstone of disease management improving CD4 counts and reducing viremia it does not fully resolve chronic inflammation, which continues to contribute to morbidity in treated populations. Integrating inflammatory biomarkers such as IL-8 into routine

monitoring may provide additional insights into residual immune dysregulation and long-term clinical risk.

CONCLUSION

In conclusion, this study demonstrates that HIV infection and antiretroviral therapy distinctly influence immunological, biochemical, and inflammatory profiles. CD4+ T-cell counts were profoundly suppressed in untreated participants, partially restored with HAART, and highest in AIDS progressors, highlighting stage-dependent immune dynamics. Interleukin-8 levels declined progressively from HIV-negative through treated and advanced disease groups, reflecting persistent but variable inflammation. Socio-demographic factors, including age and gender, modulated immune and cytokine patterns, while gross hematological indices remained largely unchanged.