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Quantification of Plasma miRNA-150 Across HIV-Infected Cohorts and its Correlation with CD4 and CD8 T-Cell Counts

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Abstract

Introduction: Plasma microRNAs have emerged as potential biomarkers in HIV infection, but the role of miRNA-150 across clinically stratified HIV groups remains uncertain.

Aim of the study: To quantify plasma miRNA-150 among different groups of HIV-infected patients and to evaluate its correlation with CD4 and CD8 T-lymphocyte count.

Methods: This cross-sectional study used archived plasma samples from HIV-1-infected adults preserved at the Department of Virology, Bangladesh Medical University, Bangladesh from July, 2024 to June, 2025. Participants were categorized into four protocol-defined groups: ART naïve with CD4 count <200 cells/µL, ART naïve with CD4 count 200–499 cells/µL, ART naïve with CD4 count ≥500 cells/µL, and ART receivers for more than 6 months. Plasma miRNA-150 was quantified by qRT-PCR using cel-miR-39-3p as an exogenous spike-in control. Quantified values were expressed as log10 copies/µL plasma. Group differences were described using summary statistics, and correlations with CD4 and CD8 counts were assessed using Pearson's correlation coefficient.

Results: A total of 86 HIV-infected participants were analyzed. Mean plasma miRNA-150 was 2.94 ± 0.65 log10 copies/µL in the ART-naïve CD4 <200 group, 2.66 ± 0.68 in the ART-naïve CD4 200–499 group, 2.54 ± 0.64 in the ART-naïve CD4 ≥500 group, and 2.97 ± 0.88 in the ART receiver group. Plasma miRNA-150 showed a weak, non-significant correlation with CD4 count, $r = 0.086$, $P = .428$, and a negligible, non-significant correlation with CD8 count, $r = 0.025$, $P = .817$.

Conclusion: Plasma miRNA-150 was measurable across HIV-infected groups, but between-group differences were modest and no significant correlation with CD4 or CD8 T-lymphocyte count was observed. Plasma miRNA-150 may therefore have limited utility as a stand-alone cross-sectional biomarker of immune status in this cohort.

Keywords: HIV, miRNA-150, plasma biomarkers, CD4 T-lymphocyte count, CD8 T-lymphocyte count

Introduction

Human immunodeficiency virus, HIV, infection remains a major global public health challenge, and contemporary epidemic estimates continue to depend on standardized surveillance and modeling frameworks because the burden of disease, treatment coverage, and survival patterns vary substantially across settings.[1] In Bangladesh, HIV prevalence has historically remained low in the general population, yet the epidemic has shown important concentration within specific vulnerable groups, making locally generated clinical and biomarker data especially valuable for monitoring disease status and treatment response.[2] At the same time, routine HIV care has increasingly moved beyond simple case detection toward more refined assessment of immune recovery, residual immune dysregulation, and risk stratification during antiretroviral therapy, ART. Although CD4 T-lymphocyte count remains central to clinical staging and immune assessment, evidence now indicates that CD8 count and the CD4/CD8 ratio also carry independent prognostic value, particularly in treated populations, where persistent immune activation may not be fully captured by CD4 recovery alone.[3–5]

MicroRNAs, miRNAs, are short non-coding RNAs that regulate gene expression post-transcriptionally and participate in multiple immune and inflammatory pathways. Among them, miR-150 is of particular interest in HIV-related research because of its established role in lymphocyte biology. Experimental studies have shown that miR-150 regulates B-cell differentiation through targeting c-Myb and also influences memory CD8 T-cell differentiation, supporting its biological relevance to adaptive immune function and making it a plausible candidate biomarker in studies that examine immunologic status in HIV infection.[6,7] In parallel, circulating miRNAs have emerged as analytically attractive biomarkers because they can be measured in plasma with relative stability using quantitative reverse transcription polymerase chain reaction, qRT-PCR based approaches. Foundational work on circulating miRNAs demonstrated that plasma miRNAs are sufficiently stable for blood-based biomarker applications, while subsequent methodological papers clarified practical approaches for extraction, normalization, and qRT-PCR analysis in plasma and serum samples.[8,9] These analytic developments have made plasma-based miRNA quantification increasingly feasible in translational and clinical studies.

Within HIV research, the circulating miRNA field has advanced from broad profiling studies to more focused biomarker investigations. Distinct plasma miRNA signatures have been reported in HIV-infected individuals compared with HIV-negative controls, supporting the concept that systemic miRNA patterns reflect HIV-associated immune perturbation.[10] In treated populations, plasma miRNA panels have also been associated with immune response after ART, suggesting that circulating miRNAs may contribute information that complements standard immunologic markers rather than replacing them.[11] Among individual candidates, miR-150 has received particular attention. Munshi et al. identified miR-150 as a potential biomarker of HIV/AIDS disease progression and therapy, and reported an association between miR-150 and CD4 cell count, thereby providing a strong rationale for evaluating this marker in clinically stratified HIV populations.[12] However, the available literature remains limited, especially in South Asian settings, and the direction and strength of association between plasma miR-150 and conventional immune markers may vary across study populations, treatment exposure, and assay approaches.

Against this background, assessing plasma miR-150 across protocol-defined HIV patient groups may help clarify whether this marker reflects differences in immune status across ART-naive and ART-experienced individuals, and whether it shows meaningful correlation with CD4 and CD8 T-lymphocyte counts. Such a question is clinically relevant because a useful plasma biomarker should demonstrate both analytic feasibility and biologic or clinical coherence with

established measures of immune function. The present study therefore aimed to quantify plasma miR-150 among different groups of HIV-infected patients and to evaluate its correlation with CD4 and CD8 T-lymphocyte count in a Bangladeshi study population.

Methods

This cross-sectional study was conducted using archived plasma samples from HIV-1-infected adults preserved at the Department of Virology, Bangladesh Medical University, Bangladesh from July, 2024 to June, 2025. The source population originally included serologically confirmed HIV-infected patients and healthy controls; however, the present manuscript analyzed only the HIV-infected groups relevant to the study title. Eligible patients were aged more than 18 years, had documented ART status, and had available CD4 and CD8 T-lymphocyte counts in the laboratory records. HIV-infected participants were categorized into four protocol-defined groups: ART naïve with CD4 count <200 cells/μL, ART naïve with CD4 count 200–499 cells/μL, ART naïve with CD4 count ≥500 cells/μL, and ART receivers who had received ART for more than 6 months. Hemolyzed or inadequate samples were excluded. Archived specimens had been collected in 2017 and stored at −70 °C until analysis. These grouping and sample handling procedures followed the study protocol.

Total RNA, including miRNA, was extracted from 200 μL plasma using a commercial serum and plasma RNA extraction kit. Cel-miR-39-3p was added to each plasma sample as an exogenous spike-in control before extraction. RNA concentration and purity were assessed by NanoDrop spectrophotometry, and cDNA was synthesized from extracted RNA using stem-loop reverse transcription primers for hsa-miR-150 and cel-miR-39-3p. Quantitative real-time PCR was then performed using SYBR Green chemistry on the StepOnePlus platform. Each sample was assayed in duplicate for target miR-150 and in duplicate for cel-miR-39-3p. No-template controls and melt-curve analysis were included in the PCR workflow. The use of plasma miRNA quantification by qRT-PCR and the analytical approach for circulating miRNA measurement were consistent with established methods for circulating microRNA biomarker studies.[8,9] The laboratory workflow and assay structure also followed the study protocol.

For data processing, average Ct values were calculated for hsa-miR-150 and cel-miR-39-3p. miR-150 Ct values were normalized using the median cel-miR-39-3p Ct across samples, according to the protocol formula: normalized Ct = raw miR-150 Ct − [(sample cel-miR-39 Ct) − (median cel-miR-39 Ct)]. Absolute quantification was then derived from the protocol-defined standard curve equation, $y = -4.814x + 41.85$, where y represents normalized Ct and x represents miR-150 copies per microliter of plasma. Quantified values were expressed as log10 copies/μL plasma for analysis. This normalization and absolute quantification strategy was based on spike-in control normalization and standard-curve conversion methods described for circulating plasma miRNA analysis, and matched the protocol used in the present study.[8] CD4 and CD8 T-lymphocyte counts were obtained from the laboratory record book and were not remeasured as part of the present study.

Statistical analysis was performed on the HIV-infected groups only. Quantitative variables were summarized as mean ± standard deviation and median with interquartile range, as appropriate. Between-group differences in plasma miR-150 were assessed using one-way ANOVA, with non-parametric comparison also considered where relevant. Correlations between plasma miR-150 level and CD4 and CD8 T-lymphocyte counts were evaluated using Pearson's correlation coefficient. All tests were two-sided, and $P < .05$ was considered statistically significant. The main statistical framework was consistent with the analytic plan specified in the study protocol.

Results

Mean age ranged from 34.2 to 39.8 years across groups. Male participants were more common in the three ART-naïve groups, while the ART receiver group showed a 1:1 male to female distribution. Mean CD4 count increased from 99 cells/μL in the ART-naïve CD4 <200 group to 976 cells/μL in the ART receiver group. Mean CD8 count was lowest in the ART-naïve CD4 <200 group, 795 cells/μL, and highest in the ART-naïve CD4 ≥500 group, 1268 cells/μL. The CD4:CD8 ratio rose steadily across groups, from 0.17 to 0.94. Mean miRNA-150 level ranged from 2.54 to 2.97 log10 copies/μL, with the lowest value in the ART-naïve CD4 ≥500 group and the highest in the ART receiver group [Table 1].

Mean plasma miRNA-150 was 2.94 log10 copies/μL in the ART-naïve CD4 <200 group, 2.66 in the ART-naïve CD4 200 to 499 group, 2.54 in the ART-naïve CD4 ≥500 group, and 2.97 in the ART receiver group. Median values followed the same general pattern, 2.83, 2.77, 2.39, and 3.00, respectively. The ART receiver group had the highest central value, while the ART-naïve CD4 ≥500 group had the lowest. The spread of values remained broad in all groups [Table 2].

Median plasma miRNA-150 was highest in the ART receiver group and lowest in the ART-naïve CD4 ≥500 group. The ART-naïve CD4 <200 and ART-naïve CD4 200 to 499 groups showed intermediate values. Interquartile ranges overlapped across all four groups. The ART receiver group showed one low outlier [Figure 1].

The correlation between plasma miRNA-150 and CD4 count was weak, $r = 0.086$, with a 95% CI from –0.128 to 0.293 and $P = .428$. The correlation between plasma miRNA-150 and CD8 count was negligible, $r = 0.025$, with a 95% CI from –0.188 to 0.236 and $P = .817$. Neither association reached statistical significance [Table 3].

Plasma miRNA-150 values were widely scattered across the full CD4 range. The fitted line showed only a minimal upward slope. No clear concentration gradient was visible across increasing CD4 counts [Figure 2].

Table 1: Baseline characteristics of HIV-infected participants by study group

Characteristic	ART naïve CD4 <200 (*n* = 21)	ART naïve CD4 200–499 (*n* = 17)	ART naïve CD4 ≥500 (*n* = 24)	ART receiver >6 mo (*n* = 24)
Age, years, M ± SD	37.3 ± 10.5	37.4 ± 6.5	34.2 ± 9.0	39.8 ± 9.1
Male, *n* (%)	13 (61.9)	11 (64.7)	17 (70.8)	12 (50.0)
Female, *n* (%)	8 (38.1)	6 (35.3)	7 (29.2)	12 (50.0)
CD4 count, cells/μL, M ± SD	99 ± 55	268 ± 38	691 ± 93	976 ± 211
CD8 count, cells/μL, M ± SD	795 ± 389	1018 ± 371	1268 ± 485	1123 ± 390
CD4:CD8 ratio, M ± SD	0.17 ± 0.19	0.31 ± 0.15	0.71 ± 0.56	0.94 ± 0.32
miRNA-150, log10 copies/μL, M ± SD	2.94 ± 0.65	2.66 ± 0.68	2.54 ± 0.64	2.97 ± 0.88

Table 2: Quantification of plasma miRNA-150 among different groups of HIV-infected patients

Group	*n*	M ± SD	Median (IQR)
ART naïve CD4 <200	21	2.94 ± 0.65	2.83 (2.54, 3.30)
ART naïve CD4 200–499	17	2.66 ± 0.68	2.77 (2.09, 3.03)
ART naïve CD4 ≥500	24	2.54 ± 0.64	2.39 (2.13, 3.17)
ART receiver >6 month	24	2.97 ± 0.88	3.00 (2.60, 3.45)

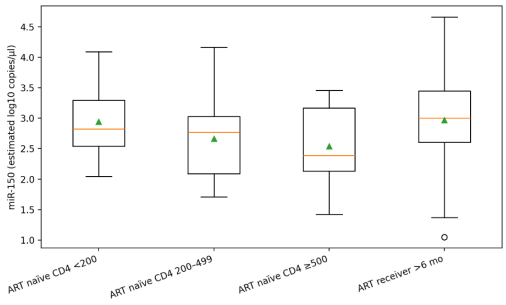

Figure 1: Plasma miR-150 (estimated log10 copies/µl) by HIV group (boxplot).

Table 3: Correlation of plasma miRNA-150 with CD4 and CD8 T-lymphocyte counts among HIV-infected participants

Variable	*n*	Pearson's *r*	95% CI	*P*
CD4 count	86	0.086	–0.128, 0.293	.428
CD8 count	86	0.025	–0.188, 0.236	.817

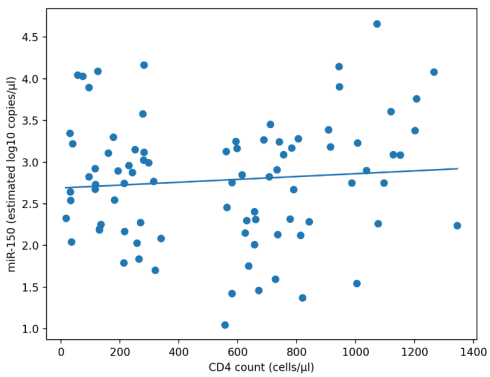

Figure 2: Scatter plot of plasma miR-150 (estimated log10 copies/µl) versus CD4 count.

Plasma miRNA-150 values were broadly dispersed across the CD8 range. The fitted line was nearly horizontal. No visible linear relationship was present between miRNA-150 and CD8 count [Figure 3].

Discussion

In this study of 86 HIV-infected participants grouped by protocol-defined immunologic

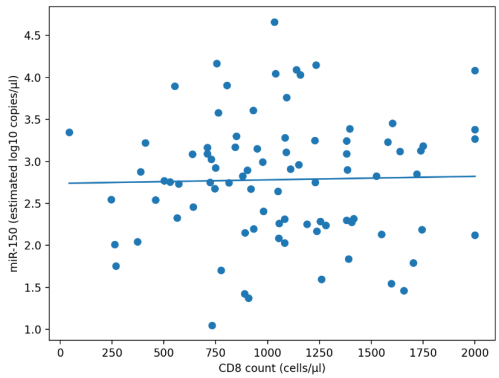

Figure 3: Scatter plot of plasma miR-150 (estimated log10 copies/µl) versus CD8 count.

and treatment categories, plasma miRNA-150 showed only modest variation across groups and no meaningful linear association with either CD4 or CD8 T-lymphocyte count. Mean miRNA-150 ranged from 2.54 ± 0.64 log10 copies/µL in the ART-naïve CD4 ≥500 group to 2.97 ± 0.88 log10 copies/µL in the ART receiver group, while the ART-naïve CD4 <200 group showed a similarly elevated mean value of 2.94 ± 0.65 log10 copies/µL. Median values followed the same pattern, with the highest central value in the ART receiver group, 3.00, and the lowest in the ART-naïve CD4 ≥500 group, 2.39. However, the interquartile ranges overlapped across all groups, and the overall pattern was not one of clear biological separation. This indicates that, within this dataset, plasma miRNA-150 did not discriminate strongly between protocol-defined HIV groups despite differences in immune status.

The most clinically relevant finding is the absence of significant correlation between plasma miRNA-150 and standard immunologic markers. The correlation with CD4 count was weak, r = 0.086, 95% CI [–0.128, 0.293], P = .428, and the correlation with CD8 count was negligible, r = 0.025, 95% CI [–0.188, 0.236], P = .817. The fitted lines in the scatter plots were nearly flat, and the data remained widely dispersed across the full CD4 and CD8 ranges. This suggests that plasma miRNA-150, as quantified here, was not tightly

aligned with contemporaneous peripheral T-lymphocyte counts. In practical terms, the marker did not behave as a direct surrogate for either CD4 depletion or CD8 expansion in this cohort.

This result differs from the study by Munshi et al., which identified miR-150 as a potential biomarker of HIV/AIDS disease progression and therapy and reported a positive association between miR-150 and CD4 cell count.[12] That paper remains the closest disease-specific comparator for the present manuscript, and the difference is important. One explanation is specimen type and analytic context. Munshi et al. evaluated both PBMC and plasma miRNAs, whereas the present study is restricted to plasma. A marker may track cellular immune state more closely in PBMC than in cell-free plasma, where pre-analytic variation, extracellular packaging, and release dynamics may weaken simple correlation with lymphocyte counts. Another explanation is population structure. The present dataset includes four protocol-defined HIV groups with both ART-naïve and ART-experienced individuals, and the observed pattern suggests that treatment exposure and immune category do not translate into a monotonic plasma miRNA-150 gradient. This is consistent with the view that circulating miRNAs may reflect broader immune perturbation rather than a single linear immunologic axis.

The present findings are more consistent with broader HIV circulating-miRNA literature showing heterogeneity rather than uniform directional change. Narla et al. demonstrated that HIV infection is associated with a distinct circulating miRNA profile, but that work emphasized multimiRNA patterning rather than reliance on a single marker.[10] Similarly, Lv et al. reported that a plasma five-miRNA panel predicted poor immune response after ART, again indicating that informative signal may be distributed across a panel rather than concentrated in one miRNA.[11] Marquez-Pedroza et al. also examined plasma miRNA expression in treated HIV-1-positive patients and related miRNA levels to markers of response to antiretroviral therapy, supporting the broader concept that plasma miRNAs are biologically relevant in HIV but may show context-specific associations depending on treatment status, resistance patterns, and the selected miRNAs.[13] Against that background, the current null correlations are not unexpected. They suggest that plasma miRNA-150 alone may have limited utility as a stand-alone immunologic marker in cross-sectional HIV stratification.

The group pattern itself is also notable. Mean miRNA-150 was not lowest in the most immunosuppressed participants. Instead, the ART-naïve CD4 ≥500 group had the lowest value, while the ART receiver and ART-naïve CD4 <200 groups had the highest values. This non-monotonic distribution argues against a simple interpretation of miRNA-150 as a direct quantitative reflection of disease severity. It is more plausible that plasma miRNA-150 is influenced by multiple biological processes, including lymphocyte differentiation, immune activation state, cellular turnover, and treatment-related immune remodeling. Experimental work supports such complexity. Xiao et al. showed that miR-150 regulates lymphocyte differentiation through c-Myb, and Chen et al. demonstrated that miR-150 modulates memory CD8 T-cell differentiation through the same pathway.[6,7] These studies provide biological plausibility for investigating miR-150 in HIV, but they do not imply that plasma abundance should correlate linearly with bulk CD4 or CD8 counts in clinical samples.

The CD4, CD8, and CD4/CD8 distributions in this study still confirm that the clinical grouping was meaningful. Mean CD4 increased from 99 ± 55 cells/μL in the ART-naïve CD4 <200 group to 976 ± 211 cells/μL in the ART receiver group, and the CD4:CD8 ratio increased from 0.17 ± 0.19 to 0.94 ± 0.32 across the same categories. This is relevant because contemporary HIV literature recognizes CD8 count and the CD4/CD8 ratio as clinically important markers of immune recovery and non-AIDS risk, not merely background

covariates. Serrano-Villar et al. showed that elevated CD8 count and low CD4/CD8 ratio after successful ART predict subsequent clinical progression, and Martínez-Sanz et al. further supported the value of CD4, CD8, and CD4/CD8 ratio in predicting non-AIDS events.[3,4] Ma et al. similarly emphasized the prognostic relevance of combined CD4, CD8, and CD4/CD8 trajectories after therapy.[5] In the present study, those conventional immune measures behaved as expected across groups, whereas plasma miRNA-150 did not track them closely. That contrast strengthens the interpretation that miRNA-150 is not a surrogate replacement for established immune monitoring markers in this dataset.

Taken together, the present findings support a cautious conclusion. Plasma miRNA-150 was detectable and quantifiable across all HIV groups, but its between-group variation was modest and its correlations with CD4 and CD8 counts were not significant. The marker therefore appears analytically feasible but clinically limited as a stand-alone cross-sectional indicator of immune status in this cohort. Its value may lie more in multimarker panels, longitudinal monitoring, or integration with additional virologic and inflammatory variables than in isolated interpretation against CD4 or CD8 alone.

Limitations of the Study

This study had a cross-sectional design, so temporal or causal relationships between plasma miRNA-150 and immunologic markers could not be established. The sample size within each subgroup was modest, and analysis was limited to archived HIV-infected samples with available CD4 and CD8 data, which may have reduced power to detect weak associations. In addition, viral load and other inflammatory or treatment-related variables were not included in the final analysis, limiting broader interpretation of plasma miRNA-150 as a clinical biomarker.

Conclusion

Plasma miRNA-150 was successfully quantified in all protocol-defined HIV-infected groups, confirming the analytical feasibility of qRT-PCR-based measurement from archived plasma samples. However, the between-group variation was modest, with the highest central values observed in the ART receiver group and the lowest in the ART-naïve CD4 ≥500 group, and substantial overlap remained across groups. Plasma miRNA-150 showed no significant correlation with CD4 count or CD8 T-lymphocyte count in this cohort. These findings suggest that plasma miRNA-150 alone has limited value as a stand-alone cross-sectional marker of immune status in HIV-infected patients, although it may still have value within multimarker or longitudinal biomarker approaches.

Ethical Approval

The study was approved by the Institutional Ethics Committee.

Authors' Contribution

The study was conceptualized by Dr. Nusrat Fatema and Prof Saif Ullah Munshi. Data collection was conducted by Dr. Nusrat Fatema. Formal analysis and methodology were prepared by Dr. Nusrat Fatema. Laboratory work was performed by Dr. Nusrat Fatema and Dr. Shafinaz Khan. Dr. Nusrat Fatema reviewed and edited the final draft. Dr. Zannatul Ferdous Tania helped in writing, review & editing. Prof. Shahina Tabassum– review & editing, Supervision, and Formal analysis.

Data Availability

Data are available from the corresponding author upon reasonable request.

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