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Longitudinal Modeling of CD4 Cell Count Trajectories among HIV Patients Receiving Antiretroviral Therapy in Pretoria, South Africa

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Abstract

To assess immune recovery among HIV patients undergoing antiretroviral therapy (ART), it is essential to monitor the CD4 cell count. Optimizing patient management is critical for understanding CD4 progression and its determinants. A longitudinal dataset comprising 4,317 observations from 841 HIV patients was analyzed using linear mixed-effects models. Random intercept and slope models were fitted to account for within-patient correlation. Nonlinear CD4 trajectories were modeled using quadratic time effects, and covariates including age, sex, tuberculosis status, and adherence were incorporated. CD4 count increased significantly over time, with an initial rapid increase followed by a plateau ($\beta_1 = 97.88$, $p < 0.001$; $\beta_2 = -58.14$, $p < 0.001$). Significant heterogeneity occurred in baseline levels and rates of change. Male patients had lower CD4 counts than females ($\beta = -32.30$, $p < 0.001$). Tuberculosis status had a negative but borderline effect, while age and adherence were not statistically significant. The findings support individualized treatment strategies and continuous monitoring.

Keywords: HIV; CD4 count; longitudinal data; mixed-effects model; nonlinear trajectory; antiretroviral therapy; immune recovery; heterogeneity.

1. Introduction

Human Immunodeficiency Virus (HIV) remains a major global public health challenge, particularly in sub-Saharan Africa. As of 2020, the population of persons living with HIV/AIDS in

west and central sub-Africa alone is over 7 million. This accounted for over 4.94% of the global HIV/AIDS death rate [1].

Despite remarkable advancements in the global fight against HIV and the aggressive scale-up of antiretroviral therapy (ART), South Africa continues to experience the world's heaviest burden of the virus [2]. As of recent estimates, the country is home to approximately 7.7 million people living with HIV, with women disproportionately bearing the brunt of the epidemic [3]. This heavy disparity is particularly evident in maternal health, where one in three women attending antenatal care is living with [3]. While the vast expansion of ART programs has successfully lowered mortality rates and vertical transmission risks, persistent structural challenges such as deep-seated economic inequalities, gender-based violence, and localized social stigmas continue to hinder the complete eradication of new infections [2].

The regional landscape of the epidemic in South Africa reveals extensive spatial and demographic variations that complicate public health interventions. Highly populated and economically active hubs like the Gauteng province exhibit massive densities of individuals living with the virus, while other regions like the KwaZulu-Natal and Eastern Cape provinces maintain staggeringly high prevalence [3, 4]. Furthermore, research highlights that a lack of adequate healthcare infrastructure in rural or informal tribal areas leaves a massive proportion of the vulnerable population underserved, severely limiting their access to timely testing and treatment [4]. To effectively align with the United Nations Joint Programme on HIV/AIDS (UNAIDS) target of ending the epidemic as a public health threat, South African strategies must increasingly address these geographic gaps and aggressively tackle the social determinants driving new infections among its youth [3].

In Nigeria for example, 1.5% prevalence of HIV infection was reported in 2020 [5]. This suggests that approximately 1.9 million of the population is infected with HIV/AIDS [6].

CD4 cells are white blood cells that act as key coordinators of the immune system, signaling other cells to fight infections. CD4 count is crucial for evaluating immune system health in people with HIV, which destroys these cells. The higher the body CD4 counts, the better the immune system. This study used the CD4 count as determinant of severity of a patient's condition. This is because CD4 cells are the cells attacked by the HIV virus, such that as the HIV infection progress, the number of these cells decreases. The normal range for CD4 cells in the body is about 500 – 1500, and a patient is diagnosed with AIDS if the CD4 counts drop below 200 [7].

A number of studies have been carried out on the change in CD4 cell counts of HIV patients on ART. Almost all the initial studies attempted to identify factors, in addition to HIV, that causes decline in CD4 cell counts in HIV patients who are undergoing antiretroviral therapy. Some of these factors are found to be demographic, such as, sex, age, location, WHO clinical stage, baseline CD4 cell count, etc. [8-13]. A good knowledge of factors which influence CD4 counts other than HIV and ART help health professionals, care givers and even patients to facilitate necessary health monitoring and management techniques of care interventions.

Some studies employed linear regression models to study some predictors of CD4 cell counts recovery in HIV-1 positive patients who are undergoing ART for over five (5) years [14, 15]. These studies majorly assumed that the change in CD4 cell counts is linear. Adams & Luguterah and Belege et al. [16, 17] suggested a longitudinal approach to studying change in CD4 cell counts of HIV-1 patients on active ART in the Builsa district hospital.

Although Antiretroviral therapy (ART) has significantly improved the prognosis of HIV patients, variability in treatment outcomes remains a major concern. Not all patients experience optimal immune recovery, and some continue to have low CD4 counts despite sustained treatment.

This heterogeneity in response poses challenges for clinicians and policymakers in optimizing treatment strategies.

Many previous studies on CD4 progression have relied on cross-sectional analyses or simple regression models that fail to capture the longitudinal and correlated nature of the data. Such approaches often assume a linear trend in CD4 recovery, which does not reflect the true biological process. Consequently, important features such as nonlinear trajectories and individual variability may be overlooked.

Furthermore, limited attention has been given to the combined effects of time and patient-specific factors in shaping CD4 dynamics, particularly in resource-limited settings. There is also a scarcity of studies that incorporate both random intercepts and random slopes to fully account heterogeneity in patient responses. Therefore, there is need for a comprehensive longitudinal analysis that accurately models CD4 progression, captures nonlinear trends, and evaluates the influence of relevant covariates.

This study therefore aims to model CD4 trajectories using advanced longitudinal methods that capture both nonlinear trends and patient-specific variability, while also identifying key determinants of immune recovery.

2. Methods

2.1. Study Design and Data

This study utilized a retrospective longitudinal dataset consisting of repeated CD4 measurements from HIV patients receiving ART. The dataset included 4,317 observations from 841 patients, with measurements recorded over multiple time points.

Let $i = 1,\ 2, \ldots, N$ denote patients, and

$j = 1,\ 2, \ldots, n_i$ denote repeated measurement for patient i

The total number of observations is therefore denoted by

$\sum_{i=1}^{N} n_i$ (1)

2.2. Variables

The variables utilized in this study include:

i. Y_{ij}: This is the CD4 cell count (cells/mm^3) of patient i at time j. It is the response variable in this study

ii. t_{ij}: Time (in months) since ART initiation. The time variable was centered in order to improve numerical stability and make intercept interpretable. The centered time variable is given by:

$t_{ij}^{*} = \frac{t_{ij} - \bar{t}}{s_t}$ (2)

where:

$\bar{t}$ is the mean time

s_t is the standard deviation of time

iii. Covariates: The covariates included as explanatory variables in this study are age, sex, tuberculosis status (yes / no) and patient's treatment adherence status.

2.3. Data Analysis

Linear mixed-effects models were employed to account for repeated measurements within patients. The modeling strategy proceeded as follows:

1. **Null model** to estimate variance components and intra-class correlation

   $$CD4 = \beta_0 + b_{0i} + e_{ij} \quad (3)$$

2. **Linear time model** to assess temporal trends

   $$CD4_{ij} = \beta_0 + \beta_1 \,\text{month}_{ij} + b_{0i} + e_{ij} \quad (4)$$

3. **Random slope model** to capture individual-specific trajectories

   $$CD4_{ij} = \beta_0 + \beta_1 \text{month}_{ij} + \; b_{0i} + b_{1i}\text{month}_{ij} + e_{ij} \quad (5)$$

4. **Nonlinear model** incorporating quadratic time effects

   $$CD4_{ij} = \beta_0 + \beta_1 \text{month}_{ij} + \beta_2 \,\text{month}_{ij}^2 + b_{0i} + b_{1i}\text{month}_{ij} + e_{ij} \quad (6)$$

5. **Covariate-adjusted model** to identify predictors of CD4 progression

$$CD4_{ij} = \beta_0 + \beta_1 \text{month}_{ij} + \beta_2 month_{ij}^2 + \beta_3 \text{age} + \beta_4 \text{sex} + \beta_5 \text{tb} + \beta_6 \,\text{adherence} + \; b_{0i} + b_{1i}\text{month}_{ij} + e_{ij} \quad (5)$$

Model estimation was performed using restricted maximum likelihood (REML).

3. Results

3.1. Descriptive Findings

The mean CD4 count at baseline was low, indicating compromised immune status at ART initiation. Substantial variability was observed across patients.

Model 1: Null Linear mixed-effects model

A null linear mixed-effects model was fitted to assess the extent of variability in CD4 count attributable to differences between patients.

$$CD4 = \beta_0 + b_{0i} + e_{ij}$$

Table 1: Random Effect

Groups Name	Variance	Standard Deviation
Intercept (Between Patient)	15458	124.3
Residual (Within Patient)	25238	158.9

Table 2: Fixed Effect

	Estimate	Standard Error	t-value
Intercept	274.184	5.071	54.06

$$ICC = \frac{15458}{15458 + 25238} = 0.38$$

From Table 1 and 2, the estimated mean CD4 count was 274.18 cells/mm^3. The variance components indicated substantial between-patient variability (σ^2 = 15458) and within-patient variability (σ^2 = 25238). The intra-class correlation coefficient (ICC) was 0.38, suggesting that 38% of the total variation in CD4 count is attributable to differences between patients while 62% is due to within patient variation over time. This shows that the data exhibits strong clustering which justifies the use of mixed effect model.

Model 2

In order to assess the within patient variation over time, we introduce the time variable (month) into the model. This gives the Model 2 as shown below

$$CD4_{ij} = \beta_0 + \beta_1 \,\mathrm{month}_{ij} + b_{0i} + e_{ij}$$

This tells us how CD4 changes over time across patients

Table 3: Random Effect

Groups Name	Variance	Standard Deviation
Intercept (Patient No.)	14020	118.4
Residual	13680	117.0

Table 4: Fixed Effect

	Estimate	Standard Error	t-value
Intercept	160.0668	4.9573	32.29
Month	7.6266	0.1353	56.38

Correlation of Fixed Effects: (Intr)

month -0.393

Table 3 and 4 shows the estimates of the parameters in Model 2. From the table, we see that at baseline (month = 0), the average CD4 count is approximately 160 cells/mm^3. The time (in month) effect is 7.63. This means that on average, CD4 count increases by 7.63 cells/ mm^3 per

month after ART initiation. The t-value is 56.38 which is highly significant at 5% level. This means that the increase in CD4 count over time is highly significant at 5% level of significance and shows that patients on ART experience a steady and significant increase in CD4 count over time, indicating immune recovery. However, Model 2 does not account for variation in the rate of patient recovery, that is, it assumes that all patients recover at the same rate. In order to account for this variation, we introduced additional parameters which specifies different baseline CD4 for each patient and accounts for their different rate of CD4 change.

Model 3

$$CD4_{ij} = \beta_0 + \beta_1 \text{month}_{ij} + \ b_{0i} + b_{1i}\text{month}_{ij} + e_{ij}$$

Table 5: Random Effect

Groups Name	**Variance**	**Standard Deviation**
Intercept (Patient No.)	37160	192.77
Month	9900	99.50
Residual	9608	98.02

Table 6: Fixed Effect

	Estimate	**Standard Error**	**t-value**
Intercept	412.032	7.919	52.03
Month	164.692	4.818	34.18

From Table 5 and 6, random slope linear mixed-effects model was fitted to allow individual-specific trajectories. The results showed a significant positive effect of time on CD4 count (β = 164.69, p < 0.001). There was substantial variability in both baseline CD4 levels (σ^2 = 37160) and rates of change over time (σ^2 = 9900). The strong positive correlation (ρ = 0.91) between intercepts and slopes suggests that patients with higher baseline CD4 counts tend to experience faster immunological recovery.

Model 4

$$CD4_{ij} = \beta_0 + \beta_1 \text{month}_{ij} + \beta_2 \, \text{month}_{ij}^2 + b_{0i} + b_{1i}\text{month}_{ij} + e_{ij}$$

This model accounts for individual trajectory and equally allows for nonlinear CD4 progression

Table 7: Random Effect

Groups Name	**Variance**	**Standard Deviation**
Intercept (Patient No.)	35651	188.81
Month	8898	94.33
Residual	8548	92.45

Table 8: Fixed Effect

	Estimate	Standard Error	t-value
Intercept	420.241	7.693	54.62
Month	96.848	5.509	17.58
Month^2	-58.584	2.704	-21.67

A quadratic mixed-effects model was fitted to capture nonlinear CD4 trajectories over time. The results as seen in Table 7 and 8 revealed a significant positive linear effect of time (β = 96.85, p < 0.001) and a significant negative quadratic effect (β = -58.58, p < 0.001), indicating that CD4 count increases rapidly at the early stages of treatment and subsequently plateaus. The model provided a better fit compared to the linear model, as evidenced by a lower REML value. Substantial variability was observed in both baseline CD4 levels and rates of change across patients, suggesting heterogeneity in treatment response.

Model 5

$$CD4_{ij} = \beta_0 + \beta_1 \text{month}_{ij} + \beta_2 month_{ij}^2 + \beta_3 \text{age} + \beta_4 \text{sex} + \beta_5 \text{tb} + \beta_6 \,\text{adherence} + b_{0i} + b_{1i} \text{month}_{ij} + e_{ij}$$

Table 9: Random Effect

Groups Name	Variance	Standard Deviation
Intercept (Patient No.)	34053	184.53
Month	8756	17.686
Residual	8587	92.66

Table 10: Fixed Effect

	Estimate	Standard Error	t-value
Intercept	455.6031	17.4376	26.128
Month	97.8847	5.5346	17.686
Month^2	-58.1350	2.7257	-21.329
Age	-0.5511	0.4169	-1.322
Sex (Male)	-32.3039	7.9803	-4.048
tb	-14.4336	7.9892	-1.807
Non-adherence	-1.0762	10.8911	-0.099

The results of the adjusted mixed-effects model from Table 9 and 10 indicated that CD4 count increased significantly over time, with a nonlinear trajectory characterized by an initial rapid increase followed by a plateau (β_1 = 97.88, β_2 = -58.14, p < 0.001). Among the covariates, sex was found to be a significant predictor, with male patients having significantly lower CD4 counts compared to females (β = -32.30, p < 0.001). TB co-infection showed a negative but borderline significant association with CD4 count, while age and adherence were not statistically significant predictors. Substantial variability remained in both baseline CD4 levels and rates of change across patients.

3.2. Longitudinal Trajectory of CD4

The study established that the CD4 cell count of patients increased significantly over time following the initiation of antiretroviral therapy. Rather than following a strictly constant or linear progression, the longitudinal trajectory of immune recovery was found to be distinctly nonlinear. The fitted quadratic mixed-effects model revealed a strong, positive linear time effect in the early months of treatment (β_1 = 97.88, p < 0.001), pointing to a phase of rapid and aggressive initial CD4 cell count recovery. However, this period of sharp growth was accompanied by a statistically significant negative quadratic effect (β_2 = -58.14, p < 0.001), which mathematically dictates that the rate of increase diminishes as time progresses. Clinically or biologically, this indicates that while patients experience substantial and swift immune system improvements immediately after starting therapy, this recovery eventually decelerates and reaches a plateau over time.

3.3. Random Effects

To properly account for the inherent correlation in repeated measurements and assess individual differences among the cohort, random effects were incorporated into the mixed-effects models. The results revealed substantial and highly statistically significant variability in both the baseline CD4 levels and the rates of immune recovery across patients. Specifically, the variance components indicated a massive spread at baseline, with an estimated intercept variance of approximately 34,053. This high degree of between-patient variability at the start of the study reflects a diverse spectrum of immune suppression among patients arriving for antiretroviral therapy initiation.

In addition to baseline differences, the rates of CD4 change over time also exhibited notable inter-individual variation, yielding an estimated slope variance of approximately 8,756. This proves that patients do not recover at a uniform pace; instead, some experience much steeper or more gradual immune reconstitution trajectories than others. Notably, a strong positive correlation of approximately 0.90 was observed between the random intercepts and random slopes. This powerful relationship indicates that patients who enter treatment with higher baseline CD4 counts generally tend to experience a faster and more robust rate of immunological recovery over time. Together, these random effects highlight a profound heterogeneity in patient treatment responses and strongly reinforce the necessity of shifting toward highly individualized clinical monitoring strategies.

3.4. Effects of Covariates

The study evaluated the influence of several patient-specific covariates on CD4 count trajectories among HIV patients undergoing antiretroviral therapy. Among these factors, biological sex emerged as a highly statistically significant predictor of immunological recovery. Specifically, male patients exhibited significantly lower CD4 counts compared to their female counterparts, showing a negative effect size of approximately 32.30 cells/mm^3 (β = -32.30, p < 0.001). This finding underscores a clear disparity in treatment outcomes between the sexes and suggests that females experience a generally higher level of immune reconstitution during the course of therapy.

Other evaluated clinical and demographic factors did not demonstrate statistically significant associations with CD4 progression at standard confidence levels. Tuberculosis (TB) co-infection

showed a negative effect of 14.43 cells/mm^3 (β = -14.43), which aligns with clinical expectations regarding the burden of co-infections, but this association was only borderline significant and did not clear the threshold for definitive statistical significance in this model. Similarly, the patient's age and treatment adherence status were not established as significant predictors of the CD4 trajectory. Age exerted a very small, non-significant negative effect (β = -0.55). Non-adherence also displayed a marginal negative impact (β = -1.08) that failed to reach statistical significance, indicating that within this specific cohort and modeling framework, adherence and age did not explain the substantial remaining variability observed in the rates of patient recovery.

3.5. Graphical Summary of Model Results

Figure 1 compares the fitted mean trajectories implied by the reported fixed-effect coefficients. Because the manuscript reports centered/scaled time for the nonlinear models but does not provide the original time mean and standard deviation, the horizontal axis is shown as a time index rather than raw months.

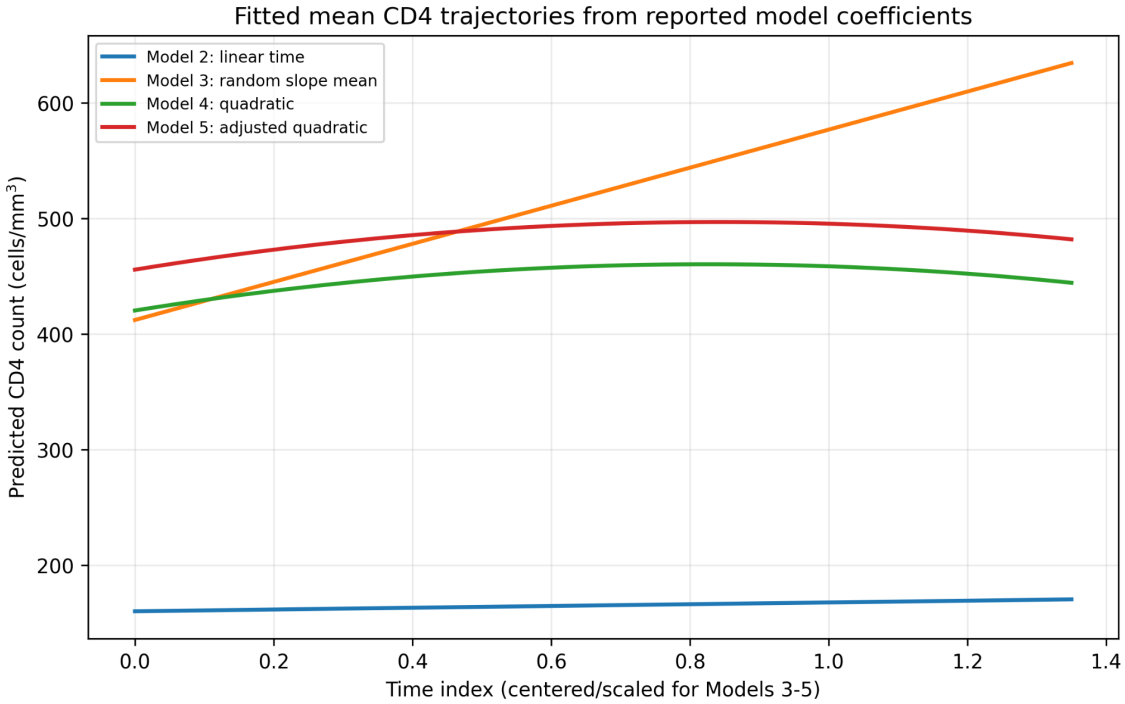

Figure 1: Fitted mean CD4 trajectories reconstructed from the reported coefficients of Models 2–5.

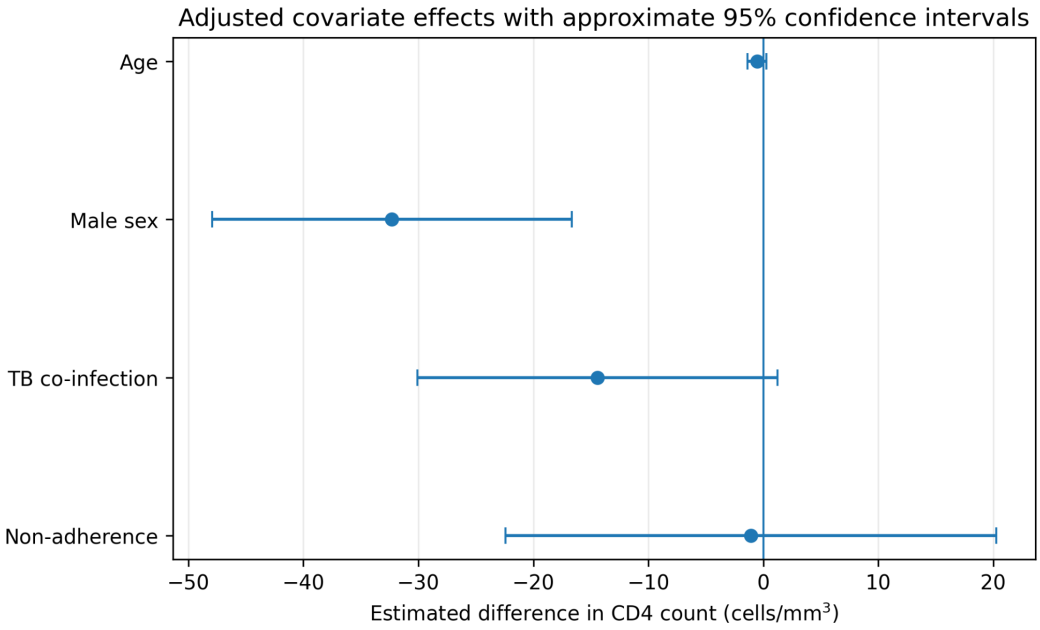

Figure 2: Adjusted covariate effects from Model 5 with approximate 95% confidence intervals calculated as estimate $\pm 1.96\times$ standard error.

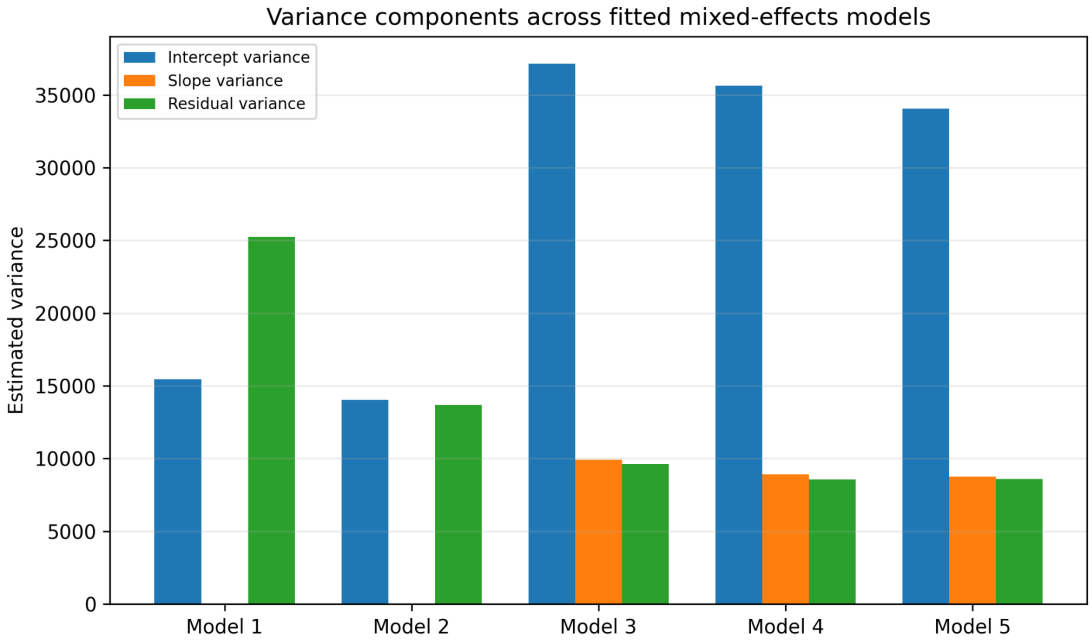

Figure 3: Reported variance components across the five fitted mixed-effects models.

4. Discussion

The results of this study demonstrate that CD4 cell count recovery among HIV patients receiving antiretroviral therapy (ART) in Pretoria follows a distinctly nonlinear trajectory. Instead of a uniform, straight-line improvement, patients typically experience a phase of rapid and aggressive immune reconstitution in the months immediately following the initiation of treatment, which

eventually decelerates and reaches a plateau over time. This pattern is highly consistent with clinical expectations and aligns closely with previous literature on immune restoration, which posits that the body undergoes a robust initial wave of T-cell expansion upon the suppression of the viral load, before gradually stabilizing at a new immunological baseline. The strong positive correlation observed between baseline CD4 counts and the subsequent rate of recovery further emphasizes that initiating ART at an earlier stage of the disease allows patients to achieve faster and more substantial immune protection.

Among the various demographic and clinical covariates assessed, biological sex was identified as a highly significant predictor of immunological outcomes. Male patients were found to have significantly lower CD4 counts compared to female patients throughout the treatment timeline. This disparity could be rooted in a combination of biological factors and behavioral differences, such as health-seeking behaviors or varying rates of true treatment engagement. Females often show better adherence or earlier presentation in clinical settings, which contributes to more favorable CD4 trajectories. Conversely, tuberculosis (TB) co-infection exhibited a negative but only borderline significant association with CD4 progression in this cohort. While it did not meet the strict threshold for statistical significance in this specific model, the direction of the effect aligns with the well-documented detrimental impact that opportunistic infections impose on the capacity of the immune system to recover.

Interestingly, variables such as age and recorded adherence did not show statistically significant associations with CD4 recovery in this analysis. This lack of significance may stem from measurement limitations, such as the potential for self-reported adherence data to mask the true extent of medication compliance, or from underlying confounding factors that were not fully captured in the current dataset. Above all, the most striking feature of the data was the profound degree of heterogeneity observed in both the starting immune status and the individual recovery rates of the patients. Because patients vary so wildly in how they respond to ART, these findings heavily discourage a "one-size-fits-all" clinical framework. Instead, they highlight a critical need for targeted, individualized monitoring strategies and patient-specific care plans to optimize long-term immune recovery in diverse populations.

5. Conclusion

This study demonstrates that CD4 cell count progression among HIV patients receiving antiretroviral therapy (ART) in Pretoria follows a distinctly nonlinear trajectory characterized by substantial inter-individual variability. Biological sex was identified as a key determinant of immune recovery, with male patients experiencing significantly poorer outcomes compared to females. These findings underscore a critical need for shifting away from a uniform approach to treatment, highlighting the necessity for personalized clinical management and individualized monitoring strategies. Ultimately, these results call for further research to investigate additional factors that influence patient treatment response to help optimize long-term immune recovery outcomes.

6. Recommendations

Based on the manuscript's findings regarding the nonlinear trajectory of CD4 cell counts, the rapid initial rise followed by a plateau, and the significant effects of biological sex, tuberculosis

co-infection, and treatment adherence, the following recommendations are provided to enhance clinical management.

Public health strategies should prioritize early HIV diagnosis and immediate enrollment into antiretroviral therapy to maximize the initial phase of rapid immune recovery. Because the study established that CD4 trajectories feature a sharp initial increase followed by a plateau, delaying treatment reduces the opportunity for patients to secure an optimized long-term immune ceiling. Immediate treatment initiation is strongly supported by findings that starting therapy at higher baseline CD4 counts ensures patients achieve healthier absolute immune levels. Furthermore, targeted male-focused interventions must be developed within healthcare frameworks to bridge the gap in immune outcomes. Since the study's results showed that male patients have significantly lower CD4 counts compared to females, active community outreach and male-friendly clinical environments are necessary to improve their treatment seeking and retention behaviors. This observation aligns with existing evidence that men consistently present with a lower hazard of CD4 recovery, thus emphasizing the need for focused, differentiated clinical care.

Furthermore, clinical programs must strictly enforce integrated medical management for individuals dealing with both HIV and tuberculosis co-infection. Given the study's findings that tuberculosis status had a negative impact on CD4 count progression, treatment protocols must treat the infections concurrently rather than sequentially. This is heavily backed by global public health observations showing that patients with active tuberculosis co-infections have a drastically higher risk of severe immune depletion and death. Lastly, intensive patient counseling and continuous adherence monitoring must be standard practice throughout the care process. The study identified adherence as a major determining factor for CD4 trajectories, meaning that even small gaps in medication compliance can severely restrict proper immune reconstitution. Robust adherence is widely recognized as the strongest modifiable predictor of positive health outcomes, and it must be actively reinforced through localized support groups or cell-phone reminder systems to stop patients from dropping into treatment failure.

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Conflict of Interest

The authors declare no conflict of interest.

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