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MATHEMATICAL MODELING OF HIV TRANSMISSION DYNAMICS WITH ANTIRETROVIRAL THERAPY, TREATMENT ADHERENCE, AND TIME DELAY: STABILITY AND NUMERICAL ANALYSIS

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Abstract

This study presents a mathematical model to analyse the transmission dynamics of Human Immunodeficiency Virus (HIV) by incorporating antiretroviral therapy (ART), treatment adherence, and delay in treatment initiation. The model divides the population into susceptible, infected, and treated compartments, allowing a realistic representation of disease progression. A system of nonlinear differential equations with time delay is formulated and analyzed to determine equilibrium points and the basic reproduction number. The stability of the disease-free equilibrium is examined, showing that the infection dies out when the reproduction number is less than unity, while persistence occurs otherwise. Numerical simulations are performed to investigate the impact of key parameters on disease dynamics. The results demonstrate that ART significantly reduces infection levels; however, its effectiveness strongly depends on treatment adherence and timely initiation. Higher adherence leads to a rapid decline in infected individuals, whereas delays in treatment contribute to sustained transmission. The findings also highlight the importance of reducing transmission rates through preventive measures. The study provides valuable insights for designing effective intervention strategies and supports global efforts aligned with the Sustainable Development Goal 3 (Good Health and Well-being), particularly Target 3.3, which aims to end the HIV/AIDS epidemic by 2030**.**

1.Introduction to the Proposed Mathematical Model:

Human Immunodeficiency Virus (HIV) remains one of the most significant global public health challenges, particularly in developing countries where access to treatment and awareness programs is limited. The transmission dynamics of HIV are complex and influenced by multiple biological, behavioral, and socio-economic factors. Mathematical modeling has emerged as an effective tool for understanding the spread of infectious diseases and evaluating the impact of intervention strategies. Classical studies on HIV transmission dynamics have provided foundational insights into disease progression and control mechanisms [1–3]. Further research has incorporated detailed biological processes such as viral replication and immune response, enhancing the understanding of within-host and population-level dynamics [4].In recent years, the role of antiretroviral therapy (ART) has been widely recognized as a critical component in controlling HIV transmission. ART not only improves the health outcomes of infected individuals but also significantly reduces viral load, thereby lowering the probability of disease transmission [11,12]. Several mathematical models have been developed to study the impact of treatment strategies, including the effects of therapy initiation, drug scheduling,

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and optimal control approaches [7–9]. However, many of these models assume ideal conditions where treatment is immediately available and perfectly followed, which is not realistic in practical scenarios. One of the major challenges in HIV control is treatment adherence. In real-world settings, patients may not strictly follow prescribed ART regimens due to various factors such as lack of awareness, economic constraints, and social stigma. Previous studies have demonstrated that imperfect adherence can significantly affect treatment outcomes and disease dynamics [5,6]. In addition, delays in treatment initiation, often caused by late diagnosis or limited healthcare access, play a crucial role in sustaining disease transmission. Despite its importance, the combined effect of treatment adherence and delay has not been sufficiently explored in many existing models. From a global perspective, the control of HIV/AIDS is a key priority under the Sustainable Development Goal 3 (Good Health and Well-being), which aims to end the epidemics of major infectious diseases by 2030. In particular, SDG Target 3.3 emphasizes the need for effective strategies to combat HIV/AIDS through prevention, treatment, and awareness initiatives [19]. In countries like India, where HIV continues to pose a significant burden, understanding the interaction between treatment strategies and disease dynamics is essential for effective policy planning [13,14]. Motivated by these challenges, the present study proposes a mathematical model for HIV transmission dynamics that incorporates antiretroviral therapy, treatment adherence, and delay in treatment initiation. The model aims to provide a more realistic representation of HIV transmission by accounting for reduced infectivity among treated individuals, imperfect adherence to therapy, and time delays in treatment. Through qualitative analysis and numerical simulations, the study investigates the impact of these factors on disease control and highlights their importance in designing effective intervention strategies. The findings of this research are expected to contribute to ongoing efforts aimed at controlling HIV transmission and achieving global health targets aligned with SDG 3.

2. Literature Review:

Mathematical Modelling is very useful to study the real-life problems such as spread of various disease. [20-23]. It has been widely used to study the transmission dynamics of HIV/AIDS. Early work by Roy M. Anderson and Robert M. May [1] provided a foundational framework for understanding HIV spread, which was later extended to include staged progression and heterogeneous infectivity [2]. Further studies explored within-host dynamics and viral behavior, improving the theoretical understanding of HIV progression [3,4].The introduction of antiretroviral therapy (ART) significantly transformed HIV control strategies by reducing viral load and transmission rates [11,12]. Several mathematical models have been developed to evaluate treatment strategies, including optimal control and therapy scheduling approaches [7–9]. However, many of these models assume ideal treatment conditions, which may not reflect real-world scenarios.Treatment adherence is a critical factor influencing the effectiveness of ART. Studies have shown that incomplete adherence can lead to persistent infection and reduced treatment efficiency [5,6]. Additionally, delays in treatment initiation due to late diagnosis or limited healthcare access further contribute to sustained transmission, although this aspect has been less explored in modeling studies. Epidemiological studies

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indicate that HIV continues to be a major public health concern, particularly in countries like India [13,14]. From a global perspective, controlling HIV/AIDS is a key objective under the Sustainable Development Goal 3 (Good Health and Well-being), which aims to end the epidemic by 2030 [19].

Despite extensive research, there remains a need for models that simultaneously incorporate treatment, adherence, and delay. The present study addresses this gap by proposing a comprehensive mathematical model to better understand HIV transmission dynamics and support effective control strategies.

3. Model Formulation and Assumptions

In this study, a compartmental mathematical model is developed to describe the transmission dynamics of HIV by incorporating the effects of antiretroviral therapy (ART), treatment adherence, and delay in treatment initiation. The total population is divided into three mutually exclusive compartments: susceptible individuals S(t), infected individuals without treatment I(t), and infected individuals receiving ART T(t).

The total population at time t is given by:

$$N(t) = S(t) + I(t) + T(t) \tag{3.1}$$

Individuals enter the susceptible class through recruitment (birth or immigration) at a constant rate Λ. Susceptible individuals become infected through effective contact with infected individuals. The transmission rate from untreated infected individuals is denoted by β, while treated individuals have a reduced transmission rate denoted by $\beta 1$ where $\beta 1<\beta$, reflecting the effect of ART in reducing infectivity. A key feature of the model is the inclusion of **treatment adherence**, represented by the parameter η, where $0\leq\eta\leq 1$. This parameter represents the proportion of infected individuals who effectively respond to ART.In addition, a **time delay τ** is introduced in the treatment process to represent the lag between infection and the initiation of ART. This delay accounts for late diagnosis, healthcare access limitations, and social factors. Infected individuals move to the treated class at a rate α, modified by the adherence factor η and the time delay τ.All compartments are subject to natural death at rate μ, while infected individuals may experience disease-induced mortality at rate δ.

3.1Governing System of Differential Equations

$$\frac{\mathrm{dS}}{\mathrm{dt}} = \Lambda - \beta \mathrm{SI} - \beta_1 \mathrm{ST} - \mu \mathrm{S} \tag{3.2}$$

$$\frac{dI}{dt} = \beta SI + \beta_1 ST - \alpha\eta I(t-\tau) - (\mu+\delta)I \tag{3.3}$$

$$\frac{dT}{dt} = \alpha\eta I(t-\tau) - \mu T \tag{3.4}$$

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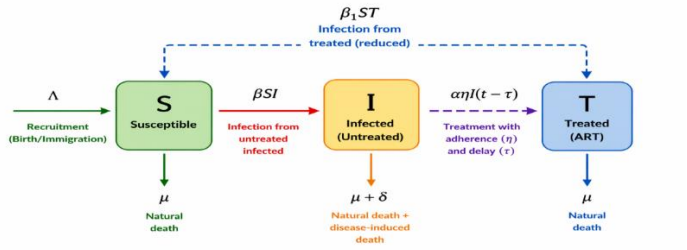

Figure 3.1: *Schematic diagram of the proposed HIV transmission model incorporating antiretroviral therapy (ART), treatment adherence, and time delay.*

Parameter Description

Parameter	Description
λ (Lambda)	Recruitment rate of susceptible population
β (Beta)	Transmission rate from untreated infected individuals
β_1 (Beta$_1$)	Transmission rate from treated individuals
α (Alpha)	Rate at which infected individuals receive ART
η (Eta)	Treatment adherence rate
τ (Tau)	Time delay in treatment initiation
μ (Mu)	Natural death rate
δ (Delta)	Disease-induced death rate

Note: All parameters are assumed to be positive.

Table 3.1: *Description of parameters used in the proposed HIV transmission model with antiretroviral therapy (ART), treatment adherence (η), and time delay (τ).*

3.2 Qualitative Analysis of the Model

3.2.1 Lemma 1: Non-negativity of Solutions

All solutions of the proposed HIV transmission model with non-negative initial conditions remain non-negative for all time t≥0.

Proof:

Let the initial conditions be:

$$S(0) \geq 0, \quad I(0) \geq 0, \quad T(0) \geq 0$$

Susceptible population

$$\frac{dS}{dt} = \Lambda - \beta SI - \beta_1 ST - \mu S$$

Since all parameters and variables are non-negative, we obtain:

$$\frac{dS}{dt} + \mu S \geq 0$$

Multiplying by integrating factor:

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$$\frac{d}{dt}(Se^{\mu t}) \geq 0$$

thus,

$$S(t) \geq S(0)e^{-\mu t} \geq 0 \qquad (3.5)$$

Hence, S(t) remains non-negative for all t ≥ 0

Similarly,

$$I(t) \geq I(0)e^{-(\mu+\delta)t} \geq 0 \qquad (3.6)$$

$$T(t) \geq T(0)e^{-\mu t} \geq 0 \qquad (3.7)$$

Hence all state variables remain non-negative for all time. Therefore, the model is biologically meaningful and mathematically well-defined.

3.2.2 Lemma 2: Boundedness of Solutions

All solutions of the model are bounded and remain in a feasible region for all t≥0.

Total population by (3.1) we get,

$$N(t) = S(t) + I(t) + T(t)$$

Differentiating both sides with respect to time, we obtain,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dT}{dt} \qquad (3.8)$$

Substituting the model equations, we get,

$$\frac{dN}{dt} = \Lambda - \beta SI - \beta_1 ST - \mu S + \beta SI + \beta_1 ST - \alpha\eta I(t-\tau) - (\mu+\delta)I + \alpha\eta I(t-\tau) - \mu T$$

$$\frac{dN}{dt} = \Lambda - \mu S - (\mu+\delta)I - \mu T$$

Thus,

$$\frac{dN}{dt} + \mu N \leq \Lambda \qquad (3.9)$$

The integrating factor is $e^{\mu t}$. Multiplying both sides, we obtain,

$$\frac{d}{dt}(Ne^{\mu t}) \leq \Lambda e^{\mu t}$$

Integrating with respect to time,

$$N(t)e^{\mu t} \leq \frac{\Lambda}{\mu}e^{\mu t} + C$$

Thus,

$$N(t) \leq \frac{\Lambda}{\mu} \qquad \forall t \geq 0 \qquad (3.10)$$

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Hence, the feasible region of the system is given by:

$$\Omega = \{(S, I, T) \in R_+^3 : N(t) \leq \frac{\Lambda}{\mu}\} \tag{3.11}$$

Hence all solutions of the model enter and remain in the region **Ω** for all t ≥0. Therefore, the system is bounded and biologically meaningful.

3.2.3 Disease-Free Equilibrium (DFE)

The disease-free equilibrium is obtained by setting the infected compartments equal to zero.

i.e $I = 0, \quad T = 0$

At equilibrium, all derivatives are zero. Thus, from the model equations:

$$\frac{dS}{dt} = 0, \quad \frac{dI}{dt} = 0, \quad \frac{dT}{dt} = 0$$

Thus, from the model equations:

$$\Lambda - \beta SI - \beta_1 ST - \mu S = 0 \tag{3.12}$$

Substitute,$I = 0, \quad T = 0$

Then,

$$\Lambda - \mu S = 0$$

Thus, $$S = \frac{\Lambda}{\mu} \tag{3.13}$$

Thus, the disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0\right)$ of the system is obtained when no infection is present in the population.

2.2.4 Basic Reproduction Number

The basic reproduction number, denoted by R_0, represents the average number of secondary infections produced by a single infected individual in a completely susceptible population.

The infected compartment of the model is I

The rate of appearance of new infections is given by:

$$F = \beta SI + \beta_1 ST \tag{3.14}$$

The transition term is given by:

$$V = (\mu + \delta + \alpha\eta)I \tag{3.15}$$

At DFE:

$$S^* = \frac{\Lambda}{\mu}, \quad I = 0, \quad T = 0$$

Now Compute derivative of F

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$$F = \beta S^* = \frac{\beta\Lambda}{\mu}$$

Now Compute derivative of V

$$V = \mu + \delta + \alpha\eta$$

Thus, the basic reproduction number is given by:

$$R_0 = \frac{\beta\Lambda}{\mu(\mu+\delta+\alpha\eta)} \tag{3.16}$$

Interpretation:

The value of R_0 determines the stability of the disease-free equilibrium:

- If $R_0 < 1$, the disease dies out over time
- If $R_0 > 1$ the disease persists in the population.

3.2.5 Local Stability of the Disease-Free Equilibrium

Theorem: The disease-free equilibrium$E_0 = \left(\frac{\Lambda}{\mu}, 0,0\right)$ locally asymptotically stable If $R_0 < 1$, and unstable If R_0>1.

Proof. To analyze the local stability, we compute the Jacobian matrix of the system.

The model equations are:

$$\frac{dS}{dt} = \Lambda - \beta SI - \beta_1 ST - \mu S$$

$$\frac{dI}{dt} = \beta SI + \beta_1 ST - \alpha\eta I(t-\tau) - (\mu+\delta)I$$

$$\frac{dT}{dt} = \alpha\eta I(t-\tau) - \mu T$$

The Jacobian matrix of the system is given by:

$$\begin{pmatrix} -\beta I - \beta_1 T - \mu & -\beta S & -\beta_1 S \\ \beta I + \beta_1 T & \beta S - (\mu + \delta + \alpha\eta) & \beta_1 S \\ 0 & \alpha\eta & -\mu \end{pmatrix} \tag{3.17}$$

At disease-free equilibrium:

$$S^* = \frac{\Lambda}{\mu}, \quad I = 0, \quad T = 0$$

Substituting into Jacobian:

$$\begin{pmatrix} -\mu & -\beta\Lambda/\mu & -\beta_1\Lambda/\mu \\ 0 & \beta\Lambda/\mu - (\mu + \delta + \alpha\eta) & \beta_1\Lambda/\mu \\ 0 & \alpha\eta & -\mu \end{pmatrix} \tag{3.18}$$

From the matrix, eigenvalues are:

$$\lambda_1 = -\mu$$

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$$\lambda_2 = \frac{\beta\Lambda}{\mu} - (\mu + \delta + \alpha\eta) \tag{3.19}$$

$$\lambda_3 = -\mu$$

Stability condition

For local stability, all eigenvalues must be negative.

from (3.19), we obtain,

$$\frac{\beta\Lambda}{\mu} < \mu + \delta + \alpha$$

In terms of R_0

$$R_0 = \frac{\beta\Lambda}{\mu(\mu+\delta+\alpha\eta)}$$

Thus,

$$R_0 < 1 \tag{3.20}$$

Hence, all the eigen values have negative real parts when $R_0 < 1$,and the disease-free equilibrium is locally asymptotical stable. If $R_0 > 1$,one eigen value becomes positive, and the equilibrium is unstable.

3.2.6 Global Stability of the Disease-Free Equilibrium

Theorem: The disease-free equilibrium E_0 is globally asymptotically stable in the feasible region Ω if $R_0 < 1$.

Proof: To prove the global stability of the disease-free equilibrium, we construct a Lyapunov function. Consider the function

$$V(I) = I \tag{3.21}$$

Clearly,$V(I) \geq 0$ and $V(I) = 0$ only when $I = 0$.

Differentiating V with respect to time,

$$\frac{dV}{dt} = \frac{dI}{dt} \tag{3.22}$$

Substituting from the model equations, we obtain

$$\frac{dV}{dt} = \beta SI + \beta_1 ST - \alpha\eta I(t-\tau) - (\mu + \delta)I \tag{3.23}$$

Since the fact that $S \leq \frac{\Lambda}{\mu}$,we obtain,

$$\frac{dV}{dt} \leq \left(\frac{\beta\Lambda}{\mu} - (\mu + \delta + \alpha\eta)\right) I$$

Since $R_0 = \frac{\beta\Lambda}{\mu(\mu+\delta+\alpha\eta)}$, and If $R_0 < 1$,then

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Then

$$\frac{dV}{dt} < 0 \tag{3.24}$$

Hence, V is a Lyapunov function. By LaSalle's Invariance Principle, the disease-free equilibrium is globally asymptotically stable in **Ω**.

3.2.7 Endemic Equilibrium

The endemic equilibrium of the system is obtained by setting all time derivatives equal to zero and considering the case when the infection persists in the population**.**

$$\frac{dS}{dt} = 0, \quad \frac{dI}{dt} = 0, \quad \frac{dT}{dt} = 0$$

From model equations, we obtain,

$$T^* = \frac{\alpha\eta}{\mu} I^* \tag{3.25}$$

$$S^* = \frac{\mu+\delta+\alpha\eta}{\beta+\frac{\beta_1\alpha\eta}{\mu}} \tag{3.26}$$

$$I^* = \frac{\Lambda-\mu S^*}{\beta S^*+\beta_1 S^*\frac{\alpha\eta}{\mu}} \tag{3.27}$$

Thus Endemic Equilibrium

$$E^* = (S^*, I^*, T^*) \tag{3.28}$$

Condition

$$I^* > 0 \quad \text{if} \quad R_0 > 1$$

Hence the endemic equilibrium exists when$R_0 > 1$, indicating sustained transmission of HIV in the population. The equilibrium levels are influenced by the transmission rate, ART treatment rate, adherence factor, and delay in treatment initiation, highlighting the crucial role of effective treatment strategies in controlling the disease.

4.Result and discussion

Numerical simulation and Graphical analysis

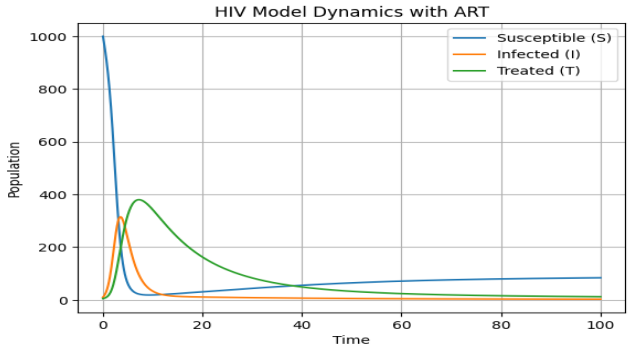

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Figure 4.1: *Time evolution of susceptible, infected, and treated populations in the proposed HIV model with ART. The infected population initially increases but decreases over time due to treatment, while the treated class grows and stabilizes. This behaviour demonstrates the impact of ART in reducing infection and controlling disease progression.*

The numerical simulation of the proposed HIV transmission model with ART provides important insights into the disease dynamics.

As observed in Figure 4.1, the susceptible population initially decreases due to increased exposure to infection, but gradually stabilizes over time. The infected population shows a rapid rise in the initial phase, indicating active transmission of the disease; however, it declines significantly as treatment is administered. The treated population increases sharply in the early stage, reflecting the impact of antiretroviral therapy, and later stabilizes at a lower level. This behaviour clearly demonstrates the effectiveness of ART in reducing the number of infected individuals and controlling the spread of HIV. Overall, the system approaches a stable state, confirming the theoretical results obtained in the qualitative analysis.

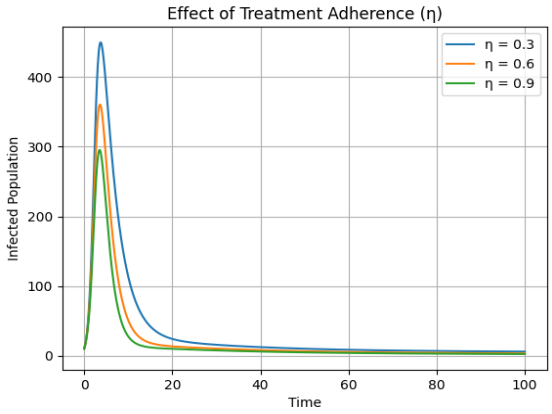

Figure 4.2: *Effect of treatment adherence (η) on the infected population. Higher adherence leads to a significant reduction in infection levels, demonstrating the importance of consistent ART usage.*

The effect of treatment adherence on the infected population is illustrated in Figure 4.2. It is observed that as the adherence level increases, the number of infected individuals decreases significantly over time. For lower values of adherence, the infected population remains relatively high, indicating that incomplete or irregular treatment is insufficient to control the spread of HIV.

On the other hand, higher adherence levels result in a rapid decline in the infected population, demonstrating the effectiveness of consistent antiretroviral therapy. This behaviour highlights the critical role of adherence in achieving viral suppression and reducing transmission.

Therefore, improving treatment adherence is essential for controlling HIV transmission, particularly in real-world settings where irregular treatment remains a major challenge. The

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results clearly indicate that adherence acts as a key control parameter in minimizing infection levels.

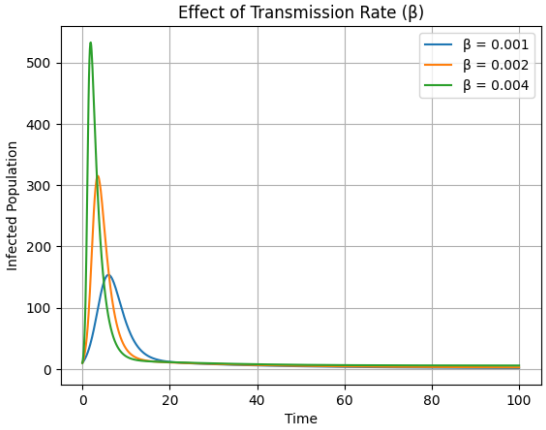

Figure 4.3: *Effect of transmission rate (β) on infection dynamics. An increase in transmission rate leads to a higher infected population, indicating faster disease spread.*

Figure 4.3, illustrates the effect of the transmission rate on the dynamics of the infected population. It is observed that as the transmission rate increases, the number of infected individuals rises significantly and reaches a higher peak. This indicates that higher contact rates or increased probability of transmission accelerate the spread of HIV within the population.

For lower values of the transmission rate, the infected population remains comparatively small and declines more rapidly, suggesting that reduced transmission can effectively control the disease. In contrast, higher transmission rates lead to sustained infection levels and delay the decline of the infected population. These results demonstrate that the transmission rate is a crucial factor in determining the severity of the epidemic. Therefore, preventive measures such as safe practices, awareness programs, and early diagnosis are essential to reduce the transmission rate and control the spread of HIV. The simulation results are consistent with the theoretical analysis, confirming that an increase in transmission rate leads to a higher basic reproduction number and sustained disease persistence. Overall, the simulation results highlight the significant role of antiretroviral therapy (ART), treatment adherence, and transmission rate in determining HIV dynamics. The findings indicate that ART effectively reduces the infected population over time; however, its success strongly depends on adherence levels, as higher adherence leads to a substantial decline in infection while poor adherence results in continued disease persistence [5,6,11,12]. The model further demonstrates that delays in treatment initiation contribute to increased infection levels, emphasizing the importance of early diagnosis and timely intervention. In addition, an increase in transmission rate leads to faster disease spread, indicating the need for preventive measures such as awareness programs and behavioural interventions. These findings are aligned with the Sustainable Development Goal 3 (Good Health and Well-being), particularly Target 3.3, which focuses on ending the HIV/AIDS epidemic by 2030 [19]. Overall, the study emphasizes the importance of integrating

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effective treatment strategies with public health interventions to achieve sustainable control of HIV.

5.Conclusion

This study presents a mathematical model to analyze the transmission dynamics of HIV in the presence of antiretroviral therapy (ART), incorporating key factors such as treatment adherence and delay in treatment initiation. The model captures the interaction between susceptible, infected, and treated populations, providing a realistic framework for understanding disease progression. The qualitative analysis establishes the positivity and boundedness of solutions, ensuring the biological feasibility of the model. The basic reproduction number R_0 is derived, which acts as a threshold parameter governing the spread of the infection. It is shown that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$, while an endemic equilibrium exists when$R_0 > 1$, indicating persistence of the disease. The numerical simulations support the analytical results and demonstrate that treatment adherence plays a significant role in reducing the infected population, whereas higher transmission rates lead to increased disease spread. The results highlight the importance of effective ART strategies in controlling HIV transmission. This study presents a mathematical model for HIV transmission dynamics incorporating antiretroviral therapy, treatment adherence, and delay in treatment initiation. The analysis establishes that the basic reproduction number acts as a threshold parameter governing disease dynamics, with the disease-free equilibrium being stable when the reproduction number is less than unity. The results demonstrate that while antiretroviral therapy plays a crucial role in reducing infection levels, its effectiveness is significantly influenced by treatment adherence and timely initiation. High adherence levels lead to a substantial decline in the infected population, whereas delays in treatment contribute to sustained transmission. These findings highlight the importance of strengthening healthcare systems to ensure early diagnosis, continuous treatment, and improved patient compliance. In the context of global health, the study contributes to the objectives of the Sustainable Development Goal 3 (Good Health and Well-being) by providing insights into effective strategies for controlling HIV transmission. The model emphasizes that a combination of treatment accessibility, adherence improvement, and transmission reduction is essential for achieving SDG Target 3.3. Overall, the proposed model offers a realistic and analytically tractable framework for understanding HIV dynamics and can serve as a useful tool for policymakers and researchers working towards sustainable disease control strategies.

6.Future Scope

The present model can be extended in several directions to enhance its applicability and realism. Future research may incorporate real epidemiological data to improve model validation and predictive accuracy. Additional compartments such as exposed or co-infected populations can be included to capture more complex disease dynamics. The model may also be extended by introducing time-dependent parameters, stochastic effects, and spatial heterogeneity to reflect real-world variability. Furthermore, the application of optimal control strategies and the integration of socio-economic and behavioral factors could provide deeper

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insights into effective intervention policies. Such extensions would contribute to more efficient and sustainable strategies for controlling HIV and achieving global health objectives aligned with the Sustainable Development Goal 3 (Good Health and Well-being).

Declarations:

Availability of Data and Materials:
No datasets were generated or analyzed during the current study.

Competing Interests:
The authors declare that they have no competing interests.

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Authors' Contributions:
Jayshri Nimje developed the mathematical model, performed the analysis, and prepared the manuscript. Dr. Kamal Wadhwa supervised the research work, reviewed the mathematical framework, and contributed to manuscript improvement.

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