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Research Article

Investigating Lie Group Symmetry and Analyzing Stability in Mathematical Modeling of the HIV Epidemic

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Abstract: This study explores the dynamics of the Human Immunodeficiency Virus (HIV)/Acquired Immunodeficiency Syndrome (AIDS) pandemic using stability and Lie symmetry analysis. The stability analysis indicates a marked decline in the susceptible population in the absence of treatment, which ultimately leads to the stabilization of the AIDS population due to increased mortality among untreated AIDS patients. Furthermore, the study identified a direct relationship between the number of HIV-positive individuals and the progression to AIDS. Lie point symmetries are applied to reveal a three-dimensional Lie algebra, facilitating the derivation of model reductions and closed-form solutions. In particular, the death rate of AIDS patients is analyzed in conjunction with the HIV infection rate and the transmission probability per partner contact. The study employs the most general Lie symmetry generator to obtain both reductions and numerical solutions, improving the understanding of the model behavior.

***Keywords*:** HIV epidemic, lie symmetry theory, invariant solutions, stability analysis

MSC: 34C14, 92D30, 34D20

1. Introduction

In [1], May emphasizes the critical role of mathematical models in understanding the dynamics of Human Immunodeficiency Virus (HIV)/Acquired Immunodeficiency Syndrome (AIDS) transmission. In particular, after HIV infection occurs, there can be a prolonged and variable incubation period before the onset of clinically detectable AIDS [2]. This results in a large percentage of HIV-infected individuals remaining undetected, leading to a lack of comprehensive data on HIV prevalence and incidence trends. Recognizing these characteristics is essential to predict the long-term impact of HIV infection [3]. However, many challenges arise when modeling the transmission dynamics of HIV/AIDS, largely due to the many complex factors that are difficult to quantify and estimate [3]. Despite these difficulties, various models of differing complexity have been developed to address these issues [4, 5].

In this article, we apply the Lie group analysis to examine the impact of Antiretroviral Therapy (ART) on HIV infection. ART comprises medications that reduce the rate of HIV replication in the body, thereby preventing progression to AIDS. The model consists of three key variables: (S) represents susceptible individuals, (I_H) denotes individuals infected with HIV but not yet exhibiting clinical symptoms of AIDS, and (A_H) refers to individuals infected with HIV

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who display AIDS symptoms. The parameters of the model are defined as follows: Λ represents the recruitment rate; λ_H is the force of infection; μ is the HIV-related mortality rate; d_A is the AIDS-related mortality rate; β_H denotes the probability of transmission per partner contact; and η_H represents the effective rate of partner change. The model is described by a system of three ordinary differential equations, as shown below.

$$\frac{dS}{dt} = \Lambda - (\lambda_H + \mu)S, \tag{1}$$

$$\frac{dI_H}{dt} = \lambda_H S - (\rho_1 + \mu)I_H, \tag{2}$$

$$\frac{dA_H}{dt} = \rho_1 I_H - (\mu + d_A)A_H. \tag{3}$$

The model is initiated with the following initial conditions: $S(0) = S_0 \geq 0,\ I_H(0) = I_{H0} \geq 0$ and $A_H(0) = A_{H0} \geq 0$. The force of infection, denoted by λ_H, is expressed as follows:

$$\lambda_H = \frac{\beta_H \eta_H I_H}{N_H}. \tag{4}$$

The total population of the model is represented by $N_H(t) = S(t) + I_H(t) + A_H(t)$. In accordance with biological considerations as discussed in [6], the sub-model system (1) is examined within given region

$$\mathscr{G}_\varepsilon = \left\{(S, I_H, A_H) \in \mathscr{R}^3_+ : N_H(t) \leq \frac{\Lambda}{\mu}\right\} \tag{5}$$

which is positively invariant with respect to system (1).

The structure of this paper is organized as follows. In Section 2, a comprehensive Lie symmetry analysis of the proposed model is presented. This section identifies the symmetries, derives the corresponding Lie algebra, and utilizes these symmetry properties to simplify the model and obtain analytical solutions where applicable.

Section 3 explores the persistence properties of the model. This involves a detailed examination of the existence, uniqueness, positivity, and boundedness of the solutions, ensuring that the model remains biologically meaningful. Furthermore, this section analyzes the existence and global stability of equilibrium points and provides a systematic calculation of the control reproduction number (R_0), assessing the potential for disease persistence or eradication.

In Section 4, the focus shifts to numerical analysis, covering essential aspects such as the elasticity index and global sensitivity analysis to identify the parameters that most significantly influence the model's behavior. Numerical simulations are also conducted to validate the theoretical results and to provide additional insights into the model's dynamics under various scenarios.

Finally, Section 5 offers concluding remarks summarizing the key findings of the study. This section highlights the contributions made by the Lie symmetry and stability analyses and suggests potential avenues for future research. Throughout, the paper systematically progresses from theoretical analysis to numerical validation, providing a clear and structured exploration of the model.

2. Application of Lie group theory

2.1 *Lie symmetry analysis of the HIV/AIDS model*

When we substitute (4) into (1), the HIV/AIDS model system becomes:

$$\frac{dS}{dt} = \Lambda - \frac{\beta_H \eta_H I_H}{N_H} S - \mu S, \tag{6}$$

$$\frac{dI_H}{dt} = \frac{\beta_H \eta_H I_H}{N_H} S - (\rho_1 + \mu) I_H, \tag{7}$$

$$\frac{dA_H}{dt} = \rho_1 I_H - (\mu + d_A) A_H. \tag{8}$$

We derive A_H from (6), i.e.

$$A_H = \frac{(\dot{S} - \Lambda)(S + I_H) + \mu S^2 + \mu S I_H + \beta_H \eta_H S I_H}{\Lambda - \mu S - \dot{S}}. \tag{9}$$

The derivative of (9) gives

$$\begin{aligned} \dot{A}_H = \Big[& - \dot{s}^3 \dot{I}_H - \Lambda^2 \dot{I}_H + \Lambda(\beta_H \eta_H + \mu) S \dot{I}_H + \Lambda \mu S \dot{I}_H - \mu(\beta_H \eta_H + \mu) S^2 \dot{I}_H \\ & + 2\Lambda \dot{S}^2 - 2\mu S \dot{S}^2 - \beta_H \eta_H \dot{S}^2 I_H - \dot{S}^2 \dot{I}_H - \mu^2 \dot{S} S^2 - \Lambda^2 \dot{S} + \beta_H \eta_H \dot{S} I_H \\ & + 2\dot{S} \dot{I}_H + 2\mu \Lambda S - (\beta_H \eta_H + 2\mu) S \dot{I}_H + \beta_H \eta_H S \ddot{S} I_H \Big] \Big/ (\dot{S} + \mu S - \Lambda)^2. \end{aligned} \tag{10}$$

The substitution of (10) into (8) and the sum (6) + (7) give a system of one equation of second order in S and one of first order in I_H, namely

$$\begin{aligned} \ddot{S} = \Big[& d_A \mu^2 S^3 + S(\Lambda - \dot{S})(d_A \Lambda - (d_A \beta \eta + 2 d_A \mu + 2\mu \rho_1) I_H - (d_A + 2\mu) \dot{S} \\ & - \beta_H \eta_H \dot{I}_H - 2\mu \dot{I}_H) + \mu S^2 (-2 d_A \Lambda + (d_A(\beta_H \eta_H + \mu) + \mu \rho_1) I_H \\ & + (2 d_A + \mu) \dot{S} + \beta_H \eta_H \dot{I}_H + \mu \dot{I}_H) + (\Lambda - \dot{S})(I_H(\Lambda(d_A + \rho_1) \\ & - (d_A + \beta_H \eta_H + \rho_1) \dot{S}) + (\Lambda - \dot{S})(\dot{S} + \dot{I}_H)) \Big] \end{aligned} \tag{11}$$

$$\dot{I}_H = -(\dot{S} + \mu S + \mu I_H + \rho_1 I_H - \Lambda). \tag{12}$$

When applying SYM package [7] for Lie group analysis of the aforementioned nonlinear system, an intriguing scenario emerges. Specifically, when Λ equals zero (indicating a situation where the recruitment rate is zero), this model has shown substantial success in reproducing findings documented in studies on HIV transmission in the San Francisco area, as reported in works such as those by Hassan [8, 9]. Furthermore, Torrisi and Nucci [10] have determined the general solution for the HIV model (1) under the condition of $\Lambda = 0$. In this case, if we define the death rate of AIDS patients, denoted as d_A, as the sum of the death rate of HIV-infected individuals plus the product of the probability of transmission per partner contact and the effective rate of partner change, it can be expressed as $d_A = \mu + \beta_H \eta_H$.

The analysis yields a linear parabolic partial differential equation, and its characteristic curve is provided by $S + I_H$. As a result, the new dependent variable, $u = S + I_H$, is introduced to obtain a new system, which in the case $d_A = \mu + \beta_H \eta_H$ admits an eight-dimensional Lie symmetry algebra. Actually it becomes separable, i.e.

$$\ddot{S} = \Big[\beta_H \eta_H \mu S^2 + \beta_H \eta_H S\dot{S} + \mu^2 S^2 - \mu \rho_1 S^2 + 2\mu S\dot{S} - \rho_1 S\dot{S} + 2\dot{S}^2\Big] \Big/ S \tag{13}$$

$$\dot{u} = -(\mu + \rho_1)u + \rho_1 S. \tag{14}$$

Torrisi and Nucci [10] finds for the Lie point symmetry of equation (13) that the coefficients of the generator infinitesimal transformation are given by

$$\xi(t, S) = \frac{\exp[-(\beta_H \eta_H + \mu - \rho_1)t]C_1}{S} + \frac{\exp[-\mu t]C_2}{S} + \exp[(\beta_H \eta_H + \mu - \rho_1)t]S^2 C_3 + \exp[\mu t]S^2 C_4 + SC_5 - \exp[(\beta_H \eta_H - \rho_1)t](C_7 - C_8)$$

$$\eta(t, S) = -\mu \exp[-(\beta_H \eta_H + \mu - \rho_1)t]C_1 - \frac{(\beta_H \eta_H + \mu - \rho_1)\exp[-\mu t]C_2}{S} + \exp[(\beta_H \eta_H - \rho_1)t]S\Big[(\beta_H \eta_H + \mu - \rho_1)C_7 - \mu C_8\Big]$$

which mean that, if $\beta_H \eta_H \neq \rho_1$, one finds an eight-dimensional Lie symmetry algebra generated by the following eight operators:

$$G_1 = \exp[-(\beta_H \eta_H + \mu - \rho_1)t]\Big[\frac{1}{S}\partial_t - \mu \partial_S\Big],$$

$$G_2 = \exp[-\mu t]\Big[\frac{1}{S}\partial_t - (\beta_H \eta_H + \mu - \rho_1)\partial_S\Big],$$

$$G_3 = \exp[(\beta_H \eta_H + \mu - \rho_1)t]S^2 \partial_S,$$

$$
\begin{aligned}
G_4 &= \exp[\mu t] S^2 \partial_S, \\
G_5 &= S\partial_S, \\
G_6 &= \partial_t, \\
G_7 &= \exp[(\beta_H \eta_H - \rho_1)t]\Big[-\partial_t + (\beta_H \eta_H + \mu - \rho_1) S\partial_S\Big], \\
G_8 &= \exp[-(\beta_H \eta_H - \rho_1)t]\Big[\partial_t + \mu S\partial_S\Big].
\end{aligned} \tag{15}
$$

If $\beta_H \eta_H = \rho_1$, one finds the eight Lie point symmetries

$$
\begin{aligned}
\hat{G}_1 &= \exp[-\mu t]\Big[\frac{1}{S}\partial_t - \mu\partial_S\Big], \\
\hat{G}_2 &= \exp[-\mu t]\Big[\frac{1}{S}\partial_t - \mu\partial_S\Big], \\
\hat{G}_3 &= \exp[\mu t] S^2 \partial_S, \\
\hat{G}_4 &= \exp[\mu t] S^2 \partial_S, \\
\hat{G}_5 &= S\partial_S, \\
\hat{G}_6 &= \partial_t, \\
\hat{G}_7 &= \Big[-\partial_t + \mu S\partial_S\Big], \\
\hat{G}_8 &= \Big[\partial_t + \mu S\partial_S\Big].
\end{aligned} \tag{16}
$$

Therefore equation (13) is linearisable by means of a point transformation [11].

To identify the linearizing transformation, one seeks a two-dimensional abelian intransitive sub-algebra. Following Lie's classification of two-dimensional algebras in the real plane, as outlined in Matadi's work [11], it is then converted into the canonical form, which we'll refer to

$$
\partial_{\tilde{v}}, \quad \tilde{t}\partial_{\tilde{v}}
$$

Using the new dependent variable $\tilde{v}$ and the new independent variable $\tilde{t}$, the sub-algebra is formed by the generators G_3 and G_4 when $\beta_H \eta_H$ is not equal to ρ_1, or alternatively, by the generators $\hat{G}_3$ and $\hat{G}_4$ when $\beta_H \eta_H$ equals ρ_1. It is straightforward to deduce that the transformation turning (13) into a linear ordinary differential equation can be achieved by setting either

$$\tilde{t} = \exp\left[(\rho_1 - \beta_H \eta_H)t\right], \quad \tilde{v} = -\frac{\exp\left[(\rho_1 - \beta_H \eta_H - \mu)t\right]}{S} \tag{17}$$

if $\beta_H \eta_H \neq \rho_1$ or

$$\tilde{t} = t, \quad \tilde{v} = -\frac{e^{-\mu t}}{S} \tag{18}$$

if $\beta_H \eta_H = \rho_1$.

Thus equation (13) becomes:

$$\frac{d^2\tilde{v}}{d\tilde{t}^2} = 0 \tag{19}$$

Consequently, its general solution is straightforwardly expressed as

$$\tilde{v} = a_1\tilde{t} + a_2, \tag{20}$$

with a_1 and a_2 representing arbitrary constants. This results in the following general solution for the system (1)

$$\begin{aligned} S(t) &= \frac{f_1(t)}{f_2(t)}, \\ I_H(t) &= \frac{1}{f_5(t)} \int \frac{f_3(t)}{f_4(t)} dt, \\ A_H(t) &= \frac{f_6(t)}{f_7(t)} - \frac{f_1(t)}{f_2(t)} + \frac{f_8(t)}{f_{10}(t)} \int \frac{f_9(t)}{f_4(t)} dt, \end{aligned} \tag{21}$$

where

$$f_1(t) = \exp[\rho_1 t] c_2,$$

$$f_2(t) = \exp[\mu t] \Big[\exp[\rho_1 t](\beta_H \eta_H - \rho_1) c_1 + \exp[\beta_H \eta_H t] \beta_H \eta_H \Big]$$

$$f_3(t) = (\beta_H \eta_H - \rho_1) \beta_H \eta_H c_2 \exp[\beta_H \eta_H t + 2\rho_1 t]$$

$$f_4(t) = (\exp[\beta_H \eta_H t]\beta_H \eta_H + \exp[\rho_1 t]\beta_H \eta_H c_1 - \exp[\rho_1 t]c_1\rho_1)^2$$

$$f_5(t) = \exp[(\mu + \rho_1)t]$$

$$f_6(t) = (\exp[\rho_1 t](\beta_H \eta_H - \rho_1)c_1 + \exp[\beta_H \eta_H t]\rho_1)c_3$$

$$f_7(t) = (\beta_H \eta_H - \rho_1)\exp[(\beta_H \eta_H + \mu + \rho_1)t]$$

$$f_8(t) = \beta_H \eta_H c_2(\exp[\rho_1 t](\beta_H \eta_H - \rho_1)c_1 + \exp[\beta_H \eta_H t]\rho_1)$$

$$f_9(t) = \exp[(\beta_H \eta_H + 2\rho_1)t]$$

$$f_{10}(t) = \exp[(\beta_H \eta_H + \mu t + \rho_1)t].$$

If $\beta_H \eta_H = 2\rho_1$, then the general solution is simply

$$S(t) = \frac{c_1}{\exp[\mu t]\left(2\exp[\rho_1 t] + c_1 c_2\right)},$$

$$I_H(t) = \Big[2\exp[\rho_1 t]\log(2\exp[\rho_1 t] + c_1 c_2)c_1 - 2\exp[\rho_1 t]c_1 + 4\exp[\rho_1 t]c_3$$

$$+\log(2\exp[\rho_1 t] + c_1 c_2)c_1^2 c_2 + 2c_1 c_2 c_3\Big] \Big/ \Big[2\exp[(\mu + \rho_1)t](2\exp[\rho_1 t] + c_1 c_2)\Big],$$

$$A_H(t) = \Big[\exp[\rho_1 t]\log(2\exp[\rho_1 t] + c_1 c_2)c_1 - 2\exp[\rho_1 t]c_1 + 2\exp[\rho_1 t]c_3$$

$$+\log(2\exp[\rho_1 t] + c_1 c_2)c_1^2 c_2 + 2c_1 c_2 c_3\Big] \Big/ \Big[2\exp[(\mu + \rho_1)t]\Big].$$

The alignment of a connection between system parameters, enhancing integrability, and the practical manifestation of this relationship in real-world modeling is captivating. It should sufficiently arouse one's curiosity to explore the potential occurrence of such a coincidence in alternative models.

2.2 *Biological interpretation of lie symmetries and reductions*

While the Lie symmetry analysis in this study primarily serves to reduce the complexity of the model equations and facilitate closed-form solutions, it also provides important biological insights that deepen our understanding of HIV transmission dynamics.

The identification of Lie point symmetries reveals invariant structures within the HIV transmission system—conditions under which the qualitative behavior of the system remains unchanged despite mathematical transformations. In particular, the existence of an eight-dimensional Lie symmetry algebra when the condition $d_A = \mu + \beta_H \eta_H$ holds,

uncovers a critical relationship among the AIDS-related mortality rate (d_A), the natural HIV-induced death rate (μ), the probability of transmission per contact (β_H), and the effective rate of partner change (η_H). This condition suggests a balance point in the epidemic dynamics, where the rates of infection and disease-induced mortality are harmonized.

Biologically, this symmetry reflects a scenario in which the rate at which AIDS patients die is matched by the rate of new infections generated through partner interactions. When this balance is achieved, the system exhibits symmetry, allowing for the derivation of reduced subsystems. For instance, using the new variable $u = S + I_H$, the model simplifies to describe invariant dynamics that are biologically interpretable as the conserved potential for transmission within the population.

Moreover, the linearizability of the nonlinear system under these symmetry conditions enables explicit analytical solutions that predict the evolution of susceptible, infected, and AIDS populations over time. These closed-form solutions offer practical benefits:

• They allow for precise estimation of epidemiological thresholds where interventions could be most effective.

• They highlight the effect of behavioral and treatment-related parameters (e.g., η_H, d_A) on the overall dynamics.

• They provide a tractable framework for public health modeling, enabling long-term projections from initial conditions and estimated parameter values.

The Lie symmetry analysis not only enhances the mathematical tractability of the model but also uncovers biologically significant structures that correspond to critical epidemiological scenarios. These insights can support the design of intervention strategies aimed at reducing transmission and stabilizing disease prevalence.

3. Persistence

Within this section, we examine the criteria for the persistence of both the host population and the disease. We present Lemma 1 and Lemma 2, with their respective proofs available in [12]. These theorems were instrumental in establishing the proof for Theorem 1.

Lemma 1 Consider a locally compact metric space X equipped with the metric d. X is represented as the disjoint union of two sets, denoted as X_1 and X_2, with the additional condition that X_2 is compact. Suppose ϕ represents a continuous semiflow on X_1. In this context, it is established that X_2 serves as a uniform strong repeller for X_1 if and only if it functions as a uniform weak repeller for X_1, as stated in the theorem.

Lemma 2 Suppose D is a bounded interval in $\mathbb{R}$, and let g: $t_0, \infty) \times D \longrightarrow \mathbb{R}$ be both bounded and uniformly continuous. Additionally, consider a solution x: $(t_0, \infty) \longrightarrow D$ of the differential equation $\dot{x} = g(t, x)$, defined on the entire interval (t_0, ∞). Then, there exist sequences s_n, $t_n \longrightarrow \infty$ such that both of the following limits hold:

$$lim_{n\longrightarrow\infty} g(s_n, x_\infty) = 0$$

and

$$lim_{n\longrightarrow\infty} g(t_n, x^\infty) = 0.$$

Now, we present Corollary 1, which will be employed in the proof of Theorem 1.

Corollary 1 If the conditions outlined in Theorem 1 are met, then the following statements hold:

1. $\liminf_{t\to\infty} g(t, x_\infty) \geq 0 \geq \limsup_{t\to\infty} g(t, x_\infty)$.
2. $\liminf_{t\to\infty} g(t, x^\infty) \geq 0 \geq \limsup_{t\to\infty} g(t, x^\infty)$.

Upon re-configuring model (1), we have

$$\frac{dS}{dt} = \Lambda - \frac{\beta_H(I_H + \eta_A A_H)}{N_H} S - \mu S,$$

$$\frac{dI_H}{dt} = \frac{\beta_H(I_H + \eta_A A_H)}{N_H} S - (\rho_1 + \mu) I_H, \tag{22}$$

$$\frac{dA_H}{dt} = \rho_1 I_H - (\mu + d_A) A_H,$$

$\beta_H(N_H)$ can exhibit diverse forms, and taking that into consideration, we introduce the following assumptions [13]:

1. The function $\beta_H(N_H)$ possesses continuous differentiability for $N_H > 0$.
2. $\beta_H(N_H)$ exhibits a monotone non-decreasing behavior with respect to N_H.
3. $\beta_H(N_H)$ is greater than zero for all N_H greater than zero.

Reformulating the model in terms of the proportions of the susceptible, infected, and ill individuals within the population, denoted as

$$x_1 = \frac{S}{N_H},\ x_2 = \frac{I_H}{N_H} \text{ and } x_3 = \frac{A_H}{N_H}. \tag{23}$$

Employing the rescalling equation (23), equation (22) can be rewritten as the subsequent nonlinear system of differential equations:

$$\frac{dN_H}{dt} = \Lambda - (\mu + d_A x_1) N_H,$$

$$\frac{dx_1}{dt} = \frac{\Lambda}{\mu}(1 - x_1) - \beta_H(N_H)(x_1 x_2 + \eta_A x_1 x_3) + d_A x_1 x_3, \tag{24}$$

$$\frac{dx_2}{dt} = \beta_H(N_H)(x_1 x_2 + \eta_A x_1 x_3) - \left(\rho_1 + \frac{\Lambda}{N_H}\right) x_2 + d_A x_2 x_3,$$

$$\frac{dx_3}{dt} = \rho_1 x_2 + d_A x_3 (x_3 - 1) - \frac{\Lambda}{N_H} x_3.$$

Equation (23) indicates that

$$x_1 + x_2 + x_3 = 1. \tag{25}$$

The manifold $x_1 + x_2 + x_3 = 1$, with $x_1, x_2, x_3 \geq 0$, remains invariant under the solution of the flow of (24). This implies that for any initial data meeting the conditions in (25), the system (24) has a global solution that satisfies (23). Now, we proceed to outline the conditions for the persistence of the host population.

Theorem 1 Suppose $\beta_H(0) = 0$ and $N_H(0) > 0$. In this case, the population exhibits uniform persistence, meaning that

$$\liminf_{t\to\infty} N_H(t) \geq \varepsilon$$

holds, where $\varepsilon > 0$ is a constant that does not rely on the initial data.

Proof. It is essential to establish that the set

$$X_1 = \{N_H = 0, x \geq 0, y \geq 0, z \geq 0, x_1 + x_2 + x_3 = 1\}$$

acts as a uniform strong repeller for

$$X_1 = \{N_H > 0, x_1 \geq 0, x_2 \geq 0, x_3 \geq 0, x_1 + x_2 + x_3 = 1\}.$$

Since the conditions of Lemma 2 are met it suffices to demonstrate that X_1 functions as a uniform weak repeller for X_1. Consider defining $r = x_1 + x_3$. Then,

$$\begin{aligned}\dot{r} =& \beta_H(N_H)(x_1x_2 + \eta_{Ax_1x_3}) - \frac{\Lambda}{N_H}r + d_Ax_3r - d_Ax_3\\ \leq& \beta_H(N_H)(1+\eta_A) - \frac{\Lambda}{N_H}r + d_A(r-1)\end{aligned} \tag{26}$$

Utilizing the fact that $x_1, x_2, x_3, r \leq 1$, it follows that

$$\begin{aligned}&\frac{\Lambda}{N_H^\infty}r^\infty + (1-r^\infty)d_A \leq \beta_H(N_H^\infty)(1+\eta_A)\\ \Rightarrow& \beta_H(N_H^\infty) \geq \frac{\Lambda r^\infty}{N_H^\infty(1+\eta_A)} + \frac{(1-r^\infty)d_A}{1+\eta_A}.\end{aligned} \tag{27}$$

Derived from the equation for N_H, as given in (24), we obtain

$$\liminf_{t\to\infty} \frac{1}{N_H}\frac{dN_H}{dt} \geq \frac{\Lambda}{N_H^\infty} - (\mu + d_Az^\infty) \geq \frac{\Lambda}{N_H^\infty} - (\mu + d_Ar^\infty).$$

Consequently, N_H experiences exponential growth unless

$$\frac{\Lambda}{N_H^\infty} \leq \mu + d_Ar^\infty, \text{ that is } \frac{1}{d_A}\left(\frac{\Lambda}{N_H^\infty} - \mu\right) \leq r^\infty. \tag{28}$$

By merging equations (27) and (28), we derive the following equation:

$$\beta_H(N_H^\infty) \geq \left(\frac{\Lambda}{d_A N_H^\infty(1+\eta_A)} - 1\right)\left(\frac{\Lambda}{N_H^\infty} - \mu\right) + \frac{d_A}{1+\eta_A}. \tag{29}$$

□

Since $\beta_H(0) = 0$ and $\beta_H(N_H)$ is continuous at 0, we can establish that $N_H^\infty \geq \varepsilon > 0$, where ε is independent of the initial data. Observing (30), it becomes apparent that we can relax the condition $\beta_H(0) = 0$ and require

$$\beta_H(N_H^\infty) < \left(\frac{\Lambda}{d_A N_H^\infty(1+\eta_A)} - 1\right)\left(\frac{\Lambda}{N_H^\infty} - \mu\right) + \frac{d_A}{1+\eta_A}. \tag{30}$$

It is crucial to explore the conditions that lead to the persistence or endemicity of the disease within the population. The disease is deemed persistent if the fractions of infected individuals and individuals with AIDS do not approach zero. Even if the overall population decreases, as long as the proportions of infected and AIDS cases remain non-zero, we would still conclude that the disease persists within the population.

Proposition 1 Suppose $\beta_H(\infty)(1+\eta_A) \geq \Lambda r^\infty / N_H^\infty$. In that case, the disease is uniformly weakly persistent, meaning that

$$r^\infty = \limsup_{t\to\infty} r(t) \geq \varepsilon$$

holds, and ε is independent of the initial data, provided that $r(0) > 0$.

The proof of Proposition 1 is described in [12].

3.1 *Equilibrium state in the absence of disease and analysis of its stability*

The equilibrium State of the model (22) in the absence of disease of the is given by

$$A_0 = (S_0, I_{H0}, A_{H0}) = \left(\frac{\Lambda}{\mu}, 0, 0\right). \tag{31}$$

The spectral radius, denoted as R_1, serves as a pivotal threshold value for controlling diseases, as it signifies the count of new infections initiated by a single infected individual within a completely susceptible population, as outlined in reference [14]. In our scenario, the reproductive number, denoted as R_1, within the model system (22), represents the quantity of subsequent HIV/AIDS cases originating from a single HIV-positive individual throughout their entire lifetime. We employ the methodology introduced by Van den Driessche and Watmough [14] to ascertain the reproductive number for the model system (1). Hence, the following is obtained

$$\mathscr{F} = \begin{pmatrix} \lambda_{H1} \\ 0 \\ 0 \end{pmatrix} \text{ and } \nu = \begin{pmatrix} (\rho_1+\mu)I_H \\ -\rho_1 I_H + (\mu+d_A)A_H \\ (\lambda_H+\mu)S - \Lambda \end{pmatrix}. \tag{32}$$

The infected compartments solely consist of I_H and A_H. Consequently, we can deduce the following:

$$F = \begin{pmatrix} \beta_H & \eta_H \\ 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \rho_1 + \mu & 0 \\ -\rho_1 & \mu + d_A \end{pmatrix}. \tag{33}$$

As a result, the HIV/AIDS-induced reproductive number, represented by the spectral radius of the dominant eigenvalue, is denoted as

$$\rho(F.V^{-1}) = R_1 = \frac{\beta_H(\mu + d_A + \eta_A\rho_1)}{(\rho_1 + \mu)(\mu + d_A)}. \tag{34}$$

Concerning the model system (22), it can be determined that the disease-free equilibrium is locally asymptotically stable when $R_1 < 1$ and unstable when $R_1 > 1$.

Theorem 2 The disease-free equilibrium, denoted as A_0, exhibits local asymptotic stability when $R_1 < 1$ and instability when $R_1 > 1$.

Proof. The Jacobian matrix at A_0 for the model system (22) is represented as

$$J(A_0) = \begin{pmatrix} -\mu & -\beta_H & \beta_H\eta_A \\ 0 & \beta_H - (\rho_1 + \mu) & \beta_H\eta_A \\ 0 & \rho_1 & -(\mu + d_A) \end{pmatrix}.$$

$$\text{Trace}[J(A_0)] = -(2\mu + d_A) + \beta_H - (\rho_1 + \mu) < 0$$

$$\text{when } \rho_1 + \mu > \beta_H.$$

$$\text{Det}[J(A_0)] = -\mu((\mu + d_A)(\rho_1 + \mu) - \beta_H(\mu + d_A + \rho_1\eta_A)) < 0$$

$$\text{when } (\mu + d_A)(\rho_1 + \mu) > \beta_H(\mu + d_A + \rho_1\eta_A)$$

$$\Longrightarrow 1 > \frac{\beta_H(\mu + d_A + \rho_1\eta_A)}{(\mu + d_A)(\rho_1 + \mu)}.$$

As a result, A_0 is characterized by local asymptotic stability when $R_1 < 1$ and instability in all other cases. □

We now enumerate two requirements that, if met, guarantee the disease-free state's global asymptotic stability. The model system (22) is rewritten as follows [13].

$$\frac{dX}{dt} = F(X, Z),$$

$$\frac{dZ}{dt} = G(X, Z), \;\; G(X, 0) = 0, \tag{35}$$

In this context, where X corresponds to S and Z pertains to (I_H, A_H), X is an element of $\mathbb{R}^1$, signifying the quantity of uninfected individuals, and Z is an element of $\mathbb{R}^2$, signifying the quantity of infected individuals, encompassing both latent and infectious cases.

The disease-free equilibrium is presently represented as

$$A_0 = (X^*, 0), \;\; \text{where } X^* = \left(\frac{\Lambda}{\mu}\right).$$

To ensure local asymptotic stability, the following conditions, denoted as (H_1) and (H_2), must be satisfied [13].

$$(H_1) \;\; \frac{dX}{dt} = F(X, 0) \;\; X^* \;\; \text{is globally asymptotically stable}$$

$$(H_2) \;\; G(X, Z) = AZ - \hat{G}(X, Z), \;\; \hat{G}(X, Z) \geq 0 \text{ for } (X, Z) \in \mathscr{G}_\varepsilon, \tag{36}$$

Here, $A = D_Z G(X^*, \; 0)$ is considered an M-matrix, with the off-diagonal elements of A being nonnegative. Additionally, $\mathscr{G}$ represents the region where the model is biologically meaningful. If the system (35) fulfills conditions (H_1) and (H_2), then (1) is applicable.

Theorem 3 The fixed point $A_0 = (X^*, 0)$ represents a globally asymptotically stable equilibrium for the system (35) when $R_1 < 1$ and the conditions outlined in (36) are met.

Proof. In Theorem 2, we demonstrated that, under the condition $R_1 < 1$ and A_0, the model system (22) attains local asymptotic stability. Considering

$$F(X, 0) = [\Lambda - \mu S],$$

$$G(X, Z) = AZ - \hat{G}(X, Z), \;\; A = \begin{bmatrix} \beta_H - (\rho_1 + \mu) & \beta_H \eta_A \\ \rho_1 & -(\mu + d_A) \end{bmatrix},$$

it follows that

$$\hat{G}(X, Z) = \begin{bmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \end{bmatrix} = \begin{bmatrix} \beta_H (1 - \frac{1}{N})(I_H + \eta_A A_H) \\ 0 \end{bmatrix}.$$

Hence, when $\hat{G}1(X, Z) \geq 0$ and $\hat{G}2(X, Z) \geq 0$, it implies that $\hat{G}(X, Z) \geq 0$. The criteria outlined in (36) are met, and A_0 attains global asymptotic stability when $R_1 < 1$. □

3.2 *Analysis of the stability of the endemic equilibrium*

The equilibrium with endemic disease is given by

$$A^* = (S^*, I_H^*, A_H^*) = \left(\frac{N_H}{R_1}, \frac{\Lambda R_1 - \mu N_H}{(\rho_1 + \mu)R_1}, \frac{\rho_1(\Lambda R_1 - \mu N_H)}{R_1(\rho_1 + \mu)(\mu + d_A)}\right). \tag{37}$$

Theorem 4 The endemic equilibrium, designated as A^*, exhibits local asymptotic stability when $R_1 > 1$, and instability in other cases.

Proof. The disease reaches an endemic state when both $\dot{I}H(t) > 0$ and $\dot{A}H(t) > 0$, represented as

$$I_H < \frac{\beta_H}{N_H}\frac{I_H + \eta_A A_H}{\rho_1 + \mu}S < \frac{\beta_H}{\rho_1 + \mu}(I_H + \eta_A A_H)$$

$$\Rightarrow I_H < \frac{\beta_H}{\rho_1 + \mu}(I_H + \eta_A A_H) \tag{38}$$

Following the utilization of the condition $\frac{S}{N_H} < 1$ and

$$A_H > \frac{\rho_1 I_H}{\mu + d_A}, \tag{39}$$

and subsequently substituting (39) into (38), we derive the following

$$I_H < \frac{\beta_H}{\rho_1 + \mu}(I_H + \eta_A A_H) < \frac{\beta_H}{\rho_1 + \mu}\left(I_H + \frac{\rho_1 \eta_A I_H}{\mu + d_A}\right)$$

$$\Rightarrow I_H < \frac{\beta_H(\mu + d_A + \rho_1 \eta_A)}{(\rho_1 + \mu)(\mu + d_A)}I_H = R_1 I_H$$

$$\Rightarrow 1 < R_1.$$

Hence, the equilibrium with endemic disease attains local asymptotic stability when $R_1 > 1$. □

4. Global sensitivity analysis

The global sensitivity analysis was carried out using the Partial Rank Correlation Coefficient (PRCC) method implemented in R software (version 4.3.2) in combination with Latin Hypercube Sampling (LHS). A total of 1,000 sample points were generated to evaluate the sensitivity of the basic reproduction number R_1 and the cumulative number of new HIV infections.

The parameter ranges used in the sampling process are presented below in Table 1. These ranges were selected based on literature sources and plausible biological variability in HIV transmission dynamics.

Table 1. Parameter ranges used for sensitivity analysis

Parameter	Description	Min Value	Max Value
β_H	Probability of transmission per partner contact	0.01	0.05
μ	HIV-related natural death rate	5×10^{-5}	8×10^{-5}
ρ_1	HIV progression rate to AIDS	2×10^{-7}	5×10^{-7}
η_H	Effective rate of partner change	0.2	1.0
d_A	AIDS-related death rate	5×10^{-6}	1.5×10^{-5}

For each parameter, the PRCC value was computed with respect to both R_1 and cumulative new infections over the simulation time horizon. Parameters with PRCC values near $\pm$ 1 were interpreted as having a strong influence on the outcome, and these are highlighted in Figure 1.

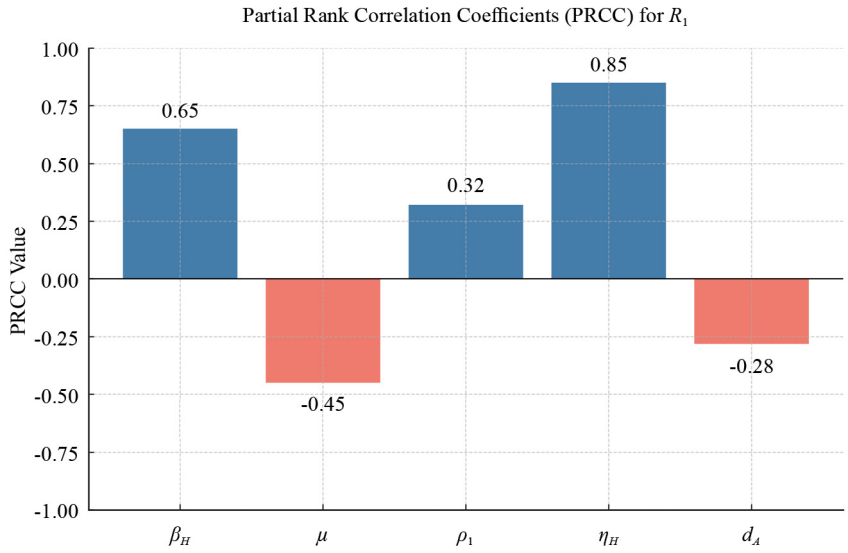

Figure 1. The global sensitivity analysis reveals the PRCC pertaining to the control reproduction number R_1 within the HIV-infected population

Figure 1 illustrates the PRCC values derived from Latin Hypercube Sampling, quantifying the sensitivity of R_1 to parameter variations.

• η_H shows the strongest positive correlation (PRCC ≈ 0.85), indicating that higher partner turnover significantly raises R_1. Public health interventions that reduce η_H may be particularly effective.

• β_H also positively influences R_1 (PRCC ≈ 0.65), underscoring the importance of reducing transmission probability through preventive measures.

• The HIV progression rate ρ_1 has a moderate positive correlation (PRCC ≈ 0.32), reflecting its role in determining how long infected individuals remain in the infectious class.

• The parameters μ (natural HIV mortality rate) and d_A (AIDS-related death rate) have negative PRCCs (approximately -0.45 and -0.28, respectively), as higher death rates shorten the infectious period and thus reduce R_1.

These results emphasize that controlling behavioral parameters, especially η_H and β_H, is crucial to mitigating the spread of HIV. The findings can guide public health efforts aimed at reducing R_1 below the epidemic threshold.

5. Numerical solutions

In this section, the Matlab solver ode45 is utilized to establish the numerical solutions of system of first-order ODEs (1) subject to the initial conditions $S(0) = 0.9$, $I_H(0) = 0.6$, $A_H(0) = 0$. The parameters used for numerical simulations (unless varied in sensitivity analyses) were those listed in Table 2. These represent typical values drawn from previous studies and are consistent with those used in the sensitivity setup.

Table 2. Baseline parameter values used for numerical simulation

Parameter	Description	Value
β_H	Probability of transmission per partner contact	0.02
μ	HIV-related natural death rate	6.3×10^{-5}
ρ_1	HIV progression rate to AIDS	3.9×10^{-7}
η_H	Effective rate of partner change	0.64
d_A	AIDS-related death rate	1.0×10^{-6}

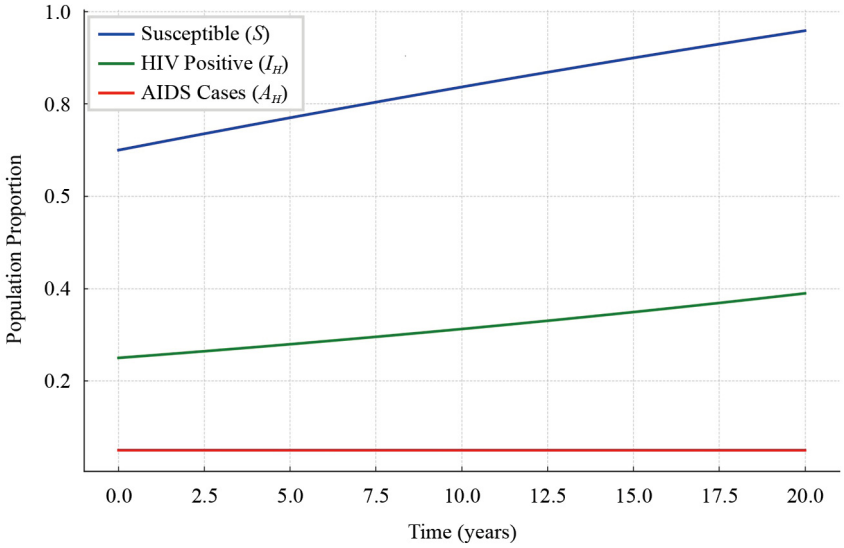

Figure 2. Graph showing the susceptible, S, the HIV positive, (I_H), the AIDS cases, (A_H)

Figure 2 illustrates the time evolution of the HIV/AIDS model over a 20-year period under modified parameter conditions designed to elicit oscillatory behavior. The susceptible population (S) shows a gradual but non-linear decline characterized by periodic fluctuations. These oscillations result from the interplay between recruitment into the susceptible class and new infections driven by transmission dynamics. The HIV-positive class (I_H) initially rises and then exhibits mild waves, capturing alternating phases of intensified and reduced transmission. This wavy behavior reflects the biological reality where infection rates vary due to behavioral, demographic, or treatment-driven changes in the population. The AIDS-affected class (A_H) grows slowly over time, with subtle undulations resulting from delayed progression from HIV infection to AIDS. The oscillatory nature of the curves demonstrates that even in a deterministic setting, non-equilibrium dynamics can emerge when key parameters such as the partner change rate and progression rate are varied. These results emphasize the importance of dynamic monitoring and adaptable intervention strategies in managing long-term HIV/AIDS epidemics.

6. Comparative insights with existing models and empirical data

The results obtained in this study align with and extend existing findings in the literature. For instance, our observation that the susceptible population declines over time while the HIV-positive population stabilizes under treatment is consistent with the outcomes reported by Ayele et al. [3], who modeled HIV/AIDS dynamics in the Ethiopian context using optimal control theory. Similarly, the sensitivity of the basic reproduction number R_1 to behavioral parameters such as the effective rate of partner change (η_H) and the transmission probability per contact (β_H) confirms earlier studies by Garnett and Anderson [4], who emphasized the importance of sexual network structures in driving HIV transmission.

Unlike traditional compartmental models, our use of Lie symmetry techniques introduces novel analytical perspectives, allowing for exact solutions under biologically meaningful parameter constraints. This offers a significant advantage over purely numerical approaches, especially in identifying threshold conditions (e.g., $d_A = \mu + \beta_H \eta_H$) that define structural properties of the epidemic dynamics. This reduction and solvability insight builds on the symmetry-based framework developed by Torrisi and Nucci [10], and further demonstrates its applicability in modeling real-world HIV dynamics.

In terms of empirical consistency, the model's parameters and qualitative trends were selected to reflect those observed in public health datasets, such as those provided by the CDC [15]. For instance, our simulation shows a slow rise in AIDS cases following an initial period of relative stability among HIV-positive individuals—a trend consistent with longitudinal data showing delayed disease progression under ART rollout. Moreover, the global sensitivity analysis indicates that modest changes in partner behavior or ART coverage could significantly shift epidemic outcomes, echoing conclusions drawn in studies of intervention effectiveness across sub-Saharan Africa.

These findings collectively suggest that the proposed Lie symmetry-based approach not only agrees with previous models but also provides enhanced tools for exploring structural features and informing intervention strategies in HIV epidemiology. Future work may consider calibrating the model directly to region-specific datasets to quantify its predictive accuracy and policy relevance.

7. Conclusion

This study presents a mathematical model for the transmission dynamics of HIV/AIDS that incorporates the effects of Antiretroviral Therapy (ART), with a particular focus on patients receiving treatment during the AIDS stage. The analysis integrates Lie group symmetry methods and classical stability theory to explore both analytical and qualitative behaviors of the system. By applying Lie symmetry techniques, a general solution to the nonlinear model is derived under the biologically meaningful condition that the AIDS-related mortality rate equals the sum of the natural mortality rate among HIV-infected individuals and the product of the transmission probability per partner contact with the effective partner change rate. This symmetry condition not only facilitates model reduction but also highlights a structural equilibrium in the transmission dynamics, offering biologically interpretable thresholds for disease control.

In addition to identifying the equilibrium points and control reproduction number (R_1), the study rigorously assesses the local and global stability of both the disease-free and endemic equilibria. Numerical simulations conducted using adaptive solvers reveal the temporal evolution of susceptible, infected, and AIDS populations. Global sensitivity analysis—based on PRCC and Latin Hypercube Sampling—quantifies the influence of key parameters and underscores the dominant role of behavioral factors such as partner change rate (η_H) and transmission probability (β_H) in shaping the epidemic's trajectory. The simulation results are consistent with empirical trends observed in CDC data and align with existing epidemiological models, while offering new analytical perspectives through symmetry-based reductions.

Importantly, the findings highlight a nuanced insight: although antiretroviral treatment improves individual health outcomes by delaying progression to AIDS, it may inadvertently extend the infectious period, thereby increasing the potential for secondary transmission if not paired with effective preventive measures. These results stress the need for integrated public health strategies that combine treatment access with behavioral interventions and continuous surveillance. Future research may involve calibrating the model with region-specific data to improve predictive power and to further explore optimal control strategies in different epidemiological contexts.

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Conflict of interest

The author declares no competing financial interest.

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