CAUCHY – Jurnal Matematika Murni dan Aplikasi
Volume 11 (1) (2026), Pages 628-647
p-ISSN: 2086-0382; e-ISSN: 2477-3344

Deterministic Modeling of HIV–Malaria Co-Infection and Sensitivity Analysis in Papua

Vina Lusiana*, Diva Hardiestya Fanny, and Muhammad Syazali

Department of Mathematics, Faculty of Military Mathematics and Natural Sciences, Indonesia Defence University, Bogor Regency, West Java, 16810, Indonesia

Abstract

This study develops and analyzes a deterministic compartmental model for HIV–malaria co-infection in a dual-endemic setting. The model incorporates direct HIV transmission, vector-mediated malaria transmission, and epidemiological interaction through co-infected compartments. The basic reproduction number R_0 is derived to characterize the threshold behavior of the system, and a normalized forward sensitivity analysis is performed to identify key parameters influencing transmission potential. The results show that R_0 is most sensitive to the HIV transmission rate and the mosquito biting rate, indicating the dominant role of transmission intensity parameters. Conversely, mosquito mortality and HIV disease progression rates contribute negatively to transmission potential. Numerical simulations under high-endemic baseline conditions representative of Papua, Indonesia, demonstrate distinct epidemiological time scales and convergence toward an endemic steady state. The findings provide a theoretical description of the interaction between HIV and malaria transmission dynamics within a deterministic modeling framework.

Keywords: HIV–Malaria Co-Infection; Mathematical Modeling; Basic Reproduction Number; Sensitivity Analysis; Papua.

1. Introduction

Human Immunodeficiency Virus (HIV) and malaria remain two major infectious diseases that continue to impose a substantial global public health burden, particularly in tropical and subtropical regions [1], [2]. Despite notable advances in prevention and treatment, both infections persist in many low- and middle-income countries, requiring sustained and coordinated public health responses. HIV is a chronic viral infection that progressively weakens the immune system and may develop into Acquired Immunodeficiency Syndrome (AIDS) in the absence of effective antiretroviral therapy. Malaria, on the other hand, is a vector-borne parasitic disease transmitted through the bite of infected female *Anopheles* mosquitoes and remains endemic in several regions of Indonesia [3].

The coexistence of HIV and malaria within the same population creates conditions under which co-infection may occur. Clinical and biological evidence suggests the presence of bidirectional interactions between the two diseases. Acute malaria infection may induce immune activation that enhances HIV replication, while HIV-related immunosuppression may increase susceptibility

---

[*] **Corresponding author**. E-mail: vina.lusiana@idu.ac.id

Submitted: February 05, 2026 Reviewed: February 19, 2026 Accepted: March 06, 2026
DOI: `https://doi.org/10.18860/cauchy.v11i1.40964`

to malaria infection and severity of disease outcomes [4], [5]. Although these interactions operate at the individual level, their cumulative effects can influence disease transmission and persistence at the population level. Mathematical modeling offers a systematic framework for representing such interactions through compartmental structures that explicitly include co-infected classes and modified transmission or progression processes.

In Indonesia, substantial regional heterogeneity characterizes the epidemiology of both HIV and malaria. National surveillance reports indicate that eastern provinces continue to experience relatively high HIV incidence and prevalence [6]. Similarly, malaria annual reports consistently show that eastern Indonesia remains an area of persistent endemic transmission [3]. Papua Province, in particular, faces sustained public health challenges related to the concurrent presence of both infections.

According to regional health statistics, Papua Province recorded 2,541 newly reported HIV cases, alongside a malaria morbidity rate of 199.22 per 1,000 population. Official demographic data further indicate that the total population of Papua exceeds one million inhabitants [7]. These figures demonstrate the coexistence of HIV and malaria within the same demographic system. While coexistence alone does not imply direct causal interaction, it provides a relevant epidemiological context for investigating potential interaction mechanisms through mathematical modeling.

Deterministic mathematical models of HIV–malaria co-infection have been widely studied using systems of nonlinear ordinary differential equations. Early analytical work established threshold dynamics, equilibrium behavior, and persistence conditions through derivation of the basic reproduction number [8], [9]. These foundational studies demonstrated how interactions between HIV and malaria infections may alter disease dynamics at the population level.

Subsequent modeling efforts incorporated treatment-related mechanisms and behavioral effects. The epidemiological implications of antiretroviral therapy for HIV–malaria co-infection were examined in [10], while optimal control frameworks were employed to explore the theoretical effectiveness of combined intervention strategies [11]. Sensitivity and elasticity analyses have also been applied to coinfection models to identify parameters that exert the strongest influence on transmission potential and threshold quantities [12], [13].

Beyond theoretical modeling, several empirical and epidemiological studies have highlighted the public health relevance of HIV and malaria co-occurrence. Clinical investigations have reported increased morbidity and altered disease progression among coinfected individuals [4], [5]. In the Indonesian context, statistical and epidemiological analyses have documented regional disparities in HIV and malaria burden, with eastern provinces exhibiting higher prevalence and incidence rates [14], [15]. Environmental and climatic factors contributing to malaria transmission in Papua have also been examined [16], [17], while clinical determinants of recurrence and mortality among HIV–malaria coinfected patients in Jayapura have been reported in [18].

Specific to Papua Province, logistic growth modeling has been used to describe the temporal evolution of HIV/AIDS cases [19]. However, deterministic compartmental models that explicitly capture HIV–malaria co-infection dynamics, combined with systematic analysis of the disease-free equilibrium, endemic equilibrium, basic reproduction number, and parameter sensitivity, remain limited for this regional setting. In particular, mathematically consistent formulations that clearly link model assumptions, parameter definitions, and threshold analysis are still needed.

Motivated by these gaps, the present study develops a deterministic HIV–malaria co-infection model formulated as a system of nonlinear ordinary differential equations that partition human and vector populations according to infection status. The analysis includes derivation of the disease-free equilibrium, characterization of the endemic equilibrium, computation of the basic reproduction number using the next-generation matrix approach, and normalized sensitivity analysis of key parameters with respect to R_0. Numerical simulations are conducted to illustrate the qualitative behavior of the model under parameter variation. Optimal control strategies and intervention optimization are not considered in this work. The results aim to provide a mathematically consistent theoretical framework for understanding HIV–malaria co-infection dynamics and to contribute insight relevant to dual-endemic settings such as Papua Province.

2. Methods

This study develops a deterministic compartmental model to describe the transmission dynamics of HIV–malaria co-infection in a structured population. The model is formulated as a system of nonlinear ordinary differential equations (ODEs) governing the temporal evolution of epidemiological compartments in both human and mosquito populations.

2.1. Model Formulation

Let $N_H(t)$ and $N_V(t)$ denote the total human and mosquito populations at time t, respectively. The human population is divided into epidemiological compartments according to infection status, while the mosquito population is structured into classes relevant to malaria transmission. The model assumes homogeneous mixing among humans with frequency-dependent HIV transmission, whereas malaria transmission occurs through mosquito bites. Humans are recruited into the susceptible class at rate Λ_H, assuming no vertical transmission from mother to child so that all newborns are considered susceptible, and natural mortality occurs at rate μ. Susceptible individuals may acquire malaria through bites from infectious mosquitoes or HIV through effective contact with infectious humans. Malaria infection is recoverable at rate γ, whereas HIV infection is chronic and may progress to AIDS at rate α_1. Co-infection occurs when individuals infected with one disease acquire the other, and individuals in the AIDS class are assumed not to contribute to further HIV transmission. Mosquitoes are recruited at rate Λ_V, experience natural mortality at rate μ_V, and once infected with malaria remain infectious for life.

The epidemiological state variables are summarized in Table 1. The human population consists of susceptible (S), malaria-exposed (E_M), malaria-infected (I_M), HIV-infected (I_H), co-infected (I_{HM}), AIDS-only (A_H), and AIDS with malaria (A_{HM}) compartments. The mosquito population consists of susceptible (S_V), exposed (E_V), and infectious (I_V) compartments.

Table 1: List of Model Variables

No	Variable	Description
1	$S(t)$	Number of susceptible human population at time t
2	$E_M(t)$	Number of humans exposed to malaria at time t
3	$I_M(t)$	Number of humans infected with malaria at time t
4	$I_H(t)$	Number of humans infected with HIV at time t
5	$I_{HM}(t)$	Number of humans co-infected with HIV and malaria at time t
6	$A_H(t)$	Number of humans with HIV/AIDS at time t
7	$A_{HM}(t)$	Number of humans with AIDS and malaria at time t
8	$S_V(t)$	Number of susceptible mosquito population at time t
9	$E_V(t)$	Number of mosquitoes exposed to malaria at time t
10	$I_V(t)$	Number of mosquitoes infected with malaria at time t

Model parameters and their descriptions are provided in Table 2. Parameter values are derived from published epidemiological reports where available, while remaining parameters are estimated to reflect endemic conditions in Papua and to maintain epidemiological plausibility consistent with prior HIV–malaria modeling studies [8], [10], [11].

Table 2: Model parameters and their epidemiological interpretation

No	**Parameter**	**Value**	**Description**	**Source / Note**
1	Λ_H	45	Human recruitment rate (per day, Papua)	Estimated
2	μ	3.8×10^{-5}	Natural human mortality rate (per day)	[7]

Continued on next page

Table 2 continued from previous page

No	Parameter	Value	Description	Source / Note
3	Λ_V	500	Mosquito recruitment rate (per day)	Estimated
4	μ_V	0.04	Natural mosquito mortality rate (per day)	[20]
5	θ_V	0.10	Progression rate from exposed to infectious mosquitoes (per day)	[2], [3]
6	b_M	0.80	Mosquito biting rate on humans (per day)	[20]
7	β_M	0.15	Malaria transmission probability (mosquito to human)	[2], [3]
8	β_V	0.08	Malaria transmission probability (human to mosquito)	[2], [3]
9	β_H	0.003	Effective HIV transmission rate (per day)	[1], [6]
10	p	0.0833	Progression rate from exposed to infectious malaria in humans (per day)	[2], [3]
11	γ	0.05	Recovery rate from malaria infection (per day)	[2], [3]
12	ϕ	0.01	Early recovery rate from exposed malaria (per day)	Estimated
13	α_1	6.84×10^{-4}	Progression rate from HIV infection to AIDS (per day)	[1], [6]
14	α_2	0.00137	Progression rate from HIV–malaria co-infection to AIDS (per day)	Estimated
15	δ_M	1.0×10^{-4}	Malaria-induced mortality rate in humans (per day)	[2], [3]
16	δ_H	1.4×10^{-4}	HIV-induced mortality rate (per day)	[1], [6]
17	δ_A	2.7×10^{-4}	AIDS-related mortality rate (per day)	Estimated
18	ω	0.70	Modification factor for HIV susceptibility due to malaria infection	Estimated
19	τ	1.50	Malaria mortality amplification under co-infection	Estimated
20	ε_1	0.50	Reduction factor of malaria recovery in HIV-infected individuals	Estimated
21	ε_2	0.30	Reduction factor of malaria recovery in AIDS individuals	Estimated
22	η_1	1.30	Enhancement factor of malaria susceptibility in HIV-infected individuals	Estimated
23	η_2	1.50	Enhancement factor of malaria susceptibility in AIDS individuals	Estimated

2.2. Model Structure

A schematic transmission diagram illustrating the flow of individuals among compartments and the malaria transmission cycle in mosquitoes is presented in Fig. 1. The diagram summarizes the interaction pathways described in the assumptions above.

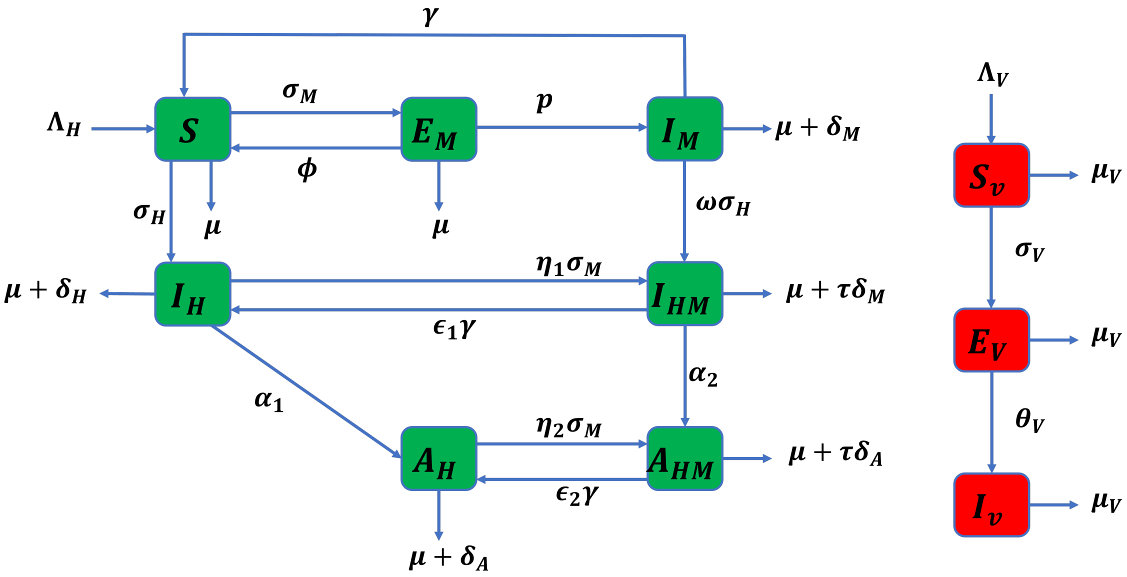


Fig. 1: Transmission diagram of the HIV–malaria co-infection model

Based on the transmission pathways depicted in Fig. 1, the complete system of nonlinear ordinary differential equations governing the HIV–malaria co-infection dynamics is formulated in the subsequent subsection.

2.2.1. Human Population

The dynamics of the human population are governed by the following system of ordinary differential equations:

$$\frac{dS}{dt} = \Lambda_H + \phi E_M + \gamma I_M - (\sigma_M + \sigma_H + \mu)S, \tag{1}$$

$$\frac{dE_M}{dt} = \sigma_M S - (\phi + p + \mu)E_M, \tag{2}$$

$$\frac{dI_M}{dt} = pE_M - (\gamma + \omega\sigma_H + \mu + \delta_M)I_M, \tag{3}$$

$$\frac{dI_H}{dt} = \sigma_H S + \varepsilon_1 \gamma I_{HM} - (\eta_1 \sigma_M + \alpha_1 + \mu + \delta_H)I_H, \tag{4}$$

$$\frac{dA_H}{dt} = \alpha_1 I_H + \varepsilon_2 \gamma A_{HM} - (\eta_2 \sigma_M + \mu + \delta_A)A_H, \tag{5}$$

$$\frac{dI_{HM}}{dt} = \eta_1 \sigma_M I_H + \omega\sigma_H I_M - (\varepsilon_1 \gamma + \alpha_2 + \mu + \tau\delta_M)I_{HM}, \tag{6}$$

$$\frac{dA_{HM}}{dt} = \alpha_2 I_{HM} + \eta_2 \sigma_M A_H - (\varepsilon_2 \gamma + \mu + \tau\delta_A)A_{HM}. \tag{7}$$

2.2.2. Mosquito Population

The malaria transmission dynamics in the mosquito population are described by the following equations:

$$\frac{dS_V}{dt} = \Lambda_V - (\sigma_V + \mu_V)S_V, \tag{8}$$

$$\frac{dE_V}{dt} = \sigma_V S_V - (\theta_V + \mu_V)E_V, \tag{9}$$

$$\frac{dI_V}{dt} = \theta_V E_V - \mu_V I_V. \tag{10}$$

2.2.3. Initial Conditions

The system is supplemented with the following non-negative initial conditions for the human population:

$$S(0) = S_0 > 0, \quad E_M(0) = E_{M0} \geq 0, \quad I_M(0) = I_{M0} \geq 0,$$

$$I_H(0) = I_{H0} \geq 0, \quad I_{HM}(0) = I_{HM0} \geq 0, \quad A_H(0) = A_{H0} \geq 0, \quad A_{HM}(0) = A_{HM0} \geq 0.$$

Similarly, for the mosquito population:

$$S_V(0) = S_{V0} > 0, \quad E_V(0) = E_{V0} \geq 0, \quad I_V(0) = I_{V0} \geq 0.$$

All initial conditions are assumed to be non-negative to ensure biological feasibility and epidemiological consistency of the model.

2.2.4. Forces of Infection

The transmission rates governing interactions between the human and mosquito populations are defined as follows. The malaria transmission rate from infectious mosquitoes to humans is given by

$$\sigma_M = \beta_M b_M \frac{I_V}{N_V},$$

where

$$N_V = S_V + E_V + I_V$$

denotes the total mosquito population.

The HIV transmission rate among humans is defined as

$$\sigma_H = \beta_H \frac{I_H + I_{HM}}{N_H},$$

where

$$N_H = S + E_M + I_M + I_H + I_{HM} + A_H + A_{HM}$$

represents the total human population. The malaria transmission rate from infectious humans to mosquitoes is expressed as

$$\sigma_V = \beta_V b_M \frac{I_M + I_{HM}}{N_H}.$$

2.3. Positivity and Boundedness of Solutions

The biological relevance of the HIV–malaria co-infection model requires that all state variables remain non-negative and bounded for all $t \geq 0$. In this subsection, it is shown that the solutions of system Eqs. (1)–(10) satisfy these properties.

Lemma 2.1 (Positivity of Solutions)**.** *For any non-negative initial conditions, the solutions of system Eqs.* (1)–(10) *remain in the positive orthant* $\mathbb{R}^{10}_{\geq 0}$ *for all* $t \geq 0$*.*

Proof. Each equation in the model can be written in the general form

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

where $F(X) \geq 0$ represents recruitment or inflow terms and $G(X) \geq 0$ denotes the per capita outflow rate.

If $X(0) \geq 0$, then whenever $X(t) = 0$, we obtain

$$\left.\frac{dX}{dt}\right|_{X=0} = F(X) \geq 0.$$

Thus, the vector field on the boundary of the positive orthant points inward or is tangent to it, implying that the solution cannot cross into negative values.

As an illustration, consider the susceptible mosquito population:

$$\frac{dS_V}{dt} = \Lambda_V - (\sigma_V + \mu_V)S_V.$$

This linear equation has the explicit solution

$$S_V(t) = S_V(0)e^{-(\sigma_V+\mu_V)t} + \frac{\Lambda_V}{\sigma_V + \mu_V}\left(1 - e^{-(\sigma_V+\mu_V)t}\right),$$

which satisfies $S_V(t) \geq 0$ for all $t \geq 0$.

Applying the same argument to all remaining compartments yields

$$S,\ E_M,\ I_M,\ I_H,\ A_H,\ I_{HM},\ A_{HM},\ S_V,\ E_V,\ I_V \geq 0 \quad \text{for all } t \geq 0.$$

Therefore, all state variables remain non-negative for all time, and the solutions are biologically feasible. ☐

Lemma 2.2 (Boundedness of Solutions)**.** *The total human population and the total mosquito population remain bounded for all* $t \geq 0$.

Proof. Let the total human population be

$$N_H = S + E_M + I_M + I_H + A_H + I_{HM} + A_{HM}.$$

Summing equations Eqs. (1)–(7) gives

$$\frac{dN_H}{dt} = \Lambda_H - \mu N_H - D_H(t),$$

where $D_H(t) \geq 0$ represents disease-induced deaths. Hence,

$$\frac{dN_H}{dt} \leq \Lambda_H - \mu N_H.$$

Solving the comparison equation $\frac{dY}{dt} = \Lambda_H - \mu Y$ yields

$$N_H(t) \leq N_H(0)e^{-\mu t} + \frac{\Lambda_H}{\mu}(1 - e^{-\mu t}),$$

which implies $0 \leq N_H(t) \leq \frac{\Lambda_H}{\mu}$ as $t \to \infty$.
Similarly, for the mosquito population

$$N_V = S_V + E_V + I_V,$$

summing equations Eqs. (8)–(10) gives

$$\frac{dN_V}{dt} = \Lambda_V - \mu_V N_V,$$

with solution

$$N_V(t) = N_V(0)e^{-\mu_V t} + \frac{\Lambda_V}{\mu_V}(1 - e^{-\mu_V t}),$$

so that $0 \leq N_V(t) \leq \frac{\Lambda_V}{\mu_V}$.
Therefore, all solutions remain in the positively invariant region

$$\Omega = \left\{ X \in \mathbb{R}^{10}_{\geq 0} \mid N_H \leq \frac{\Lambda_H}{\mu},\ N_V \leq \frac{\Lambda_V}{\mu_V} \right\},$$

and the model solutions are bounded. □

2.4. Equilibrium Points

In epidemiological model analysis, equilibrium points represent states in which all population compartments remain constant over time, indicating that no changes occur in the number of individuals within each compartment. Mathematically, an equilibrium point is characterized by the condition

$$\frac{dX}{dt} = 0$$

for all state variables X.

In the HIV–malaria co-infection model, equilibrium points provide insight into the long-term behavior of the system and indicate whether the infection will die out or persist in the population. In general, the model admits two principal types of equilibrium points: the disease-free equilibrium

(DFE) and the endemic equilibrium (EE).

2.4.1. Disease-Free Equilibrium (DFE)

The disease-free equilibrium corresponds to a state in which there are no individuals infected with HIV, malaria, or co-infection in either the human or mosquito populations. Thus,

$$E_M^* = I_M^* = I_H^* = A_H^* = I_{HM}^* = A_{HM}^* = 0, \qquad E_V^* = I_V^* = 0.$$

Therefore, the disease-free equilibrium of the HIV–malaria co-infection model is given by

$$\mathcal{E}_0 = \left(\frac{\Lambda_H}{\mu}, 0, 0, 0, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0 \right).$$

This equilibrium represents a population state in which HIV, malaria, and their co-infection are completely absent from both the human and mosquito populations. The local stability of the disease-free equilibrium will be determined in the next section through the computation of the basic reproduction number R_0 using the next-generation matrix method.

Theorem 2.3 (Local Stability of the Disease-Free Equilibrium). *The HIV–malaria co-infection model admits a disease-free equilibrium (DFE) given by $\mathcal{E}_0$. The disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable if and only if $R_0 < 1$, and it is unstable whenever $R_0 > 1$.*

Proof. Let J denote the Jacobian matrix of the full system. Evaluating J at $\mathcal{E}_0$ yields a block triangular matrix

$$J(\mathcal{E}_0) = \begin{pmatrix} J_H & 0 \\ 0 & J_V \end{pmatrix},$$

where J_H corresponds to the human compartments and J_V to the mosquito compartments. The vector submatrix is

$$J_V = \begin{pmatrix} -\mu_V & 0 & 0 \\ 0 & -(\theta_V + \mu_V) & 0 \\ 0 & \theta_V & -\mu_V \end{pmatrix}.$$

Its eigenvalues are

$$-\mu_V, \quad -(\theta_V + \mu_V), \quad -\mu_V,$$

which are strictly negative for biologically meaningful parameter values.
The human submatrix can be written in compact form as

$$J_H = \begin{pmatrix} -A_1 & \phi & \gamma & 0 & 0 & 0 & 0 \\ 0 & -A_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \rho & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_4 & 0 & \varepsilon_1\gamma & 0 \\ 0 & 0 & 0 & \alpha_1 & -A_5 & 0 & \varepsilon_2\gamma \\ 0 & 0 & 0 & 0 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_2 & -A_7 \end{pmatrix},$$

where

$$A_1 = \mu, \qquad A_2 = \mu + \phi + \rho,$$
$$A_3 = \gamma + \mu + \delta_M, \qquad A_4 = \mu + \alpha_1 + \delta_H,$$
$$A_5 = \mu + \delta_A, \qquad A_6 = \iota\delta_M + \varepsilon_1\gamma + \mu + \alpha_2,$$
$$A_7 = \iota\delta_A + \varepsilon_2\gamma + \mu.$$

The matrix J_H is block triangular and decomposes into three epidemiological components: malaria, HIV, and co-infection dynamics.
The malaria sub-block yields eigenvalues

$$-\mu, \quad -(\mu+\phi+\rho), \quad -(\gamma+\mu+\delta_M),$$

which are all negative.
The co-infection sub-block produces

$$-(\iota\delta_M+\varepsilon_1\gamma+\mu+\alpha_2), \qquad -(\iota\delta_A+\varepsilon_2\gamma+\mu),$$

which are likewise strictly negative.
The remaining eigenvalues arise from the HIV transmission sub-block

$$\begin{pmatrix} -(\mu+\alpha_1+\delta_H) & \varepsilon_1\gamma \\ \alpha_1 & -(\mu+\delta_A) \end{pmatrix}.$$

Its characteristic polynomial is

$$\lambda^2+a_1\lambda+a_0=0,$$

where

$$a_1=2\mu+\alpha_1+\delta_H+\delta_A>0, \quad \textit{and} \quad a_0=(\mu+\alpha_1+\delta_H)(\mu+\delta_A)-\alpha_1\varepsilon_1\gamma.$$

By the Routh–Hurwitz criterion, both eigenvalues have negative real parts if and only if $a_0>0$. □

2.4.2. Endemic Equilibria

The endemic equilibrium (EE) represents a situation in which the disease persists in the population at a stable level over time rather than dying out. At this equilibrium state, infection, recovery, mortality, and transition processes are balanced, yielding positive and non–trivial steady–state solutions of the system under the condition

$$\frac{dX}{dt}=0,$$

where X denotes the vector of all state variables.

In the HIV–malaria co-infection model, the existence of endemic equilibria is more intricate due to biological interactions between the two pathogens. These include enhanced susceptibility to secondary infection, altered immune responses, and modified disease progression, all of which influence long–term persistence of the infections in the population.

Existence and Local Stability of Endemic Equilibria. Consider the HIV–malaria co-infection model defined by system Eqs. (1)–(10). Let $R_0^{(M)}$ and $R_0^{(H)}$ denote the basic reproduction numbers associated with malaria and HIV transmission, respectively. Then the system admits the following endemic equilibria.

Endemic Equilibrium 1 (END–1): Malaria-Only Endemic State. If $R_0^{(M)}>1$ and HIV transmission is absent, i.e.

$$I_H^*=I_{HM}^*=A_H^*=A_{HM}^*=0, \qquad \sigma_H=0,$$

then the malaria-only endemic equilibrium is given by

$$S^* = \frac{\Lambda_H(\gamma+\mu+\delta_M)(\phi+p+\mu)}{\mu(\phi+p+\mu)(\gamma+\mu+\delta_M)+\sigma_M\left[\mu(p+\gamma+\mu)+(p+\mu)\delta_M\right]},$$

$$E_M^* = \frac{\sigma_M\Lambda_H(\gamma+\mu+\delta_M)}{\mu(\phi+p+\mu)(\gamma+\mu+\delta_M)+\sigma_M\left[\mu(p+\gamma+\mu)+(p+\mu)\delta_M\right]},$$

$$I_M^* = \frac{p\sigma_M\Lambda_H}{\mu(\phi+p+\mu)(\gamma+\mu+\delta_M)+\sigma_M\left[\mu(p+\gamma+\mu)+(p+\mu)\delta_M\right]}.$$

Endemic Equilibrium 2 (END–2): HIV-Only Endemic State. If $R_0^{(H)} > 1$ and malaria transmission is absent, i.e.

$$E_M^* = I_M^* = I_{HM}^* = A_{HM}^* = 0, \qquad \sigma_M = 0,$$

then the HIV-only endemic equilibrium is given by

$$S^* = \frac{\Lambda_H}{\sigma_H^*+\mu}, \qquad I_H^* = \frac{\Lambda_H\sigma_H^*}{(\alpha_1+\mu+\delta_H)(\sigma_H^*+\mu)},$$

$$A_H^* = \frac{\Lambda_H\alpha_1\sigma_H^*}{(\alpha_1+\mu+\delta_H)(\mu+\delta_A)(\sigma_H^*+\mu)}.$$

Endemic Equilibrium 3 (END–3): HIV–Malaria Co-infection Endemic State. If both reproduction numbers satisfy

$$R_0^{(M)} > 1, \qquad R_0^{(H)} > 1,$$

then the system admits a coexistence endemic equilibrium in which

$$E_M^*, I_M^*, I_H^*, I_{HM}^*, A_H^*, A_{HM}^* > 0.$$

The coexistence equilibrium is given by

$$S^* = \frac{(\phi+p+\mu)(\omega\sigma_H+\delta_M+\gamma+\mu)\Lambda_H}{\mathcal{D}}, \qquad E_M^* = \frac{(\omega\sigma_H+\delta_M+\gamma+\mu)\Lambda_H\sigma_M}{\mathcal{D}},$$

$$I_M^* = \frac{p\Lambda_H\sigma_M}{\mathcal{D}}, \qquad I_H^* = \frac{\mathcal{N}_{I_H}\sigma_H\Lambda_H}{\mathcal{D}\mathcal{K}},$$

$$A_H^* = \frac{\mathcal{N}_{A_H}\sigma_H\Lambda_H}{\mathcal{D}}, \qquad I_{HM}^* = \frac{\mathcal{N}_{I_{HM}}\sigma_M\sigma_H\Lambda_H}{\mathcal{D}\mathcal{K}},$$

$$A_{HM}^* = \frac{\mathcal{N}_{A_{HM}}\sigma_M\sigma_H\Lambda_H}{\mathcal{D}}.$$

For the mosquito population, the equilibrium values are

$$S_V^* = \frac{\Lambda_V}{\sigma_V+\mu_V}, \qquad E_V^* = \frac{\sigma_V\Lambda_V}{(\sigma_V+\mu_V)(\theta_V+\mu_V)},$$

$$I_V^* = \frac{\sigma_V\theta_V\Lambda_V}{\mu_V(\sigma_V+\mu_V)(\theta_V+\mu_V)}.$$

where

$$\mathcal{D} = (\omega\sigma_H+\delta_M+\gamma+\mu)(\mu^2+\mu p+\mu\phi+\mu\sigma_H+\mu\sigma_M+p\sigma_H),$$

$$\mathcal{K} = (\delta_M\tau+\gamma\epsilon_1+\mu+\alpha_1)(\eta_1\sigma_M+\alpha_2+\delta_H+\mu).$$

$$\begin{aligned}
\mathcal{N}_{I_H} &= \delta_M \mu\omega\tau + \delta_M \omega p\tau + \delta_M \omega\phi\tau + \gamma\mu\omega\epsilon_1 + \gamma\omega p\epsilon_1 + \gamma\omega\phi\epsilon_1 \\
&\quad + \alpha_2\mu\omega + \alpha_2\omega p + \alpha_2\omega\phi + \mu^2\omega + \mu\omega p + \mu\omega\phi, \\
\mathcal{N}_{A_H} &= \alpha_1\delta_M\mu\tau + \alpha_1\delta_M p\tau + \alpha_1\delta_M\phi\tau + \alpha_1\gamma\mu\epsilon_1 + \alpha_1\gamma p\epsilon_1 + \alpha_1\gamma\phi\epsilon_1 \\
&\quad + \alpha_1\mu^2 + \alpha_1\mu p + \alpha_1\mu\phi, \\
\mathcal{N}_{I_{HM}} &= \eta_1\mu\omega + \eta_1\omega p + \eta_1\omega\phi + \delta_M\eta_1\mu + \delta_M\eta_1 p + \delta_M\eta_1\phi \\
&\quad + \eta_1\gamma\mu + \eta_1\gamma p + \eta_1\gamma\phi + \eta_1\mu^2 + \eta_1\mu p + \eta_1\mu\phi, \\
\mathcal{N}_{A_{HM}} &= \alpha_2\eta_1\mu + \alpha_2\eta_1 p + \alpha_2\eta_1\phi + \alpha_2\mu^2 + \alpha_2\mu p + \alpha_2\mu\phi.
\end{aligned}$$

The endemic equilibria are obtained by setting all derivatives of the system Eqs. (1)–(10) equal to zero and solving the resulting algebraic equations. For the malaria-only equilibrium, HIV-related compartments are set to zero and the reduced malaria subsystem is solved. Similarly, the HIV-only equilibrium is obtained by eliminating malaria compartments. The coexistence equilibrium is obtained by solving the complete steady-state system in which all infected compartments are positive. Local stability can be established by evaluating the Jacobian matrix of the system at each equilibrium point and applying the Routh–Hurwitz stability criteria.

2.5. Basic Reproduction Number (R_0)

The basic reproduction number R_0 is defined as the average number of secondary infections produced by a single infectious individual introduced into a completely susceptible population. For the HIV–malaria co-infection model, which incorporates both host–vector interactions and pathogen–pathogen coupling, the Next-Generation Matrix (NGM) method proposed by van den Driessche and Watmough (2002) is employed to derive an explicit analytical expression for R_0.

The NGM approach decomposes the dynamics of the infected compartments into two components: the rate of appearance of new infections, denoted by $\mathbf{F}(x)$, and the rate of transfer out of infected compartments, denoted by $\mathbf{V}(x)$. These components are linearized at the disease-free equilibrium (DFE).

The vector of infected compartments is defined as

$$\mathbf{x} = (E_M, I_M, I_H, I_{HM}, E_V, I_V)^\top,$$

with notation consistent with system (2.1)–(2.10). At the DFE, the susceptible populations satisfy

$$S^* = \frac{\Lambda_H}{\mu}, \qquad S_V^* = \frac{\Lambda_V}{\mu_V},$$

and all infected compartments are zero.

The vector of new infection terms is given by

$$\mathbf{F}(x) = \begin{pmatrix} \beta_M b_M \dfrac{S I_V}{N_V} \\ 0 \\ \beta_H \dfrac{S(I_H + I_{HM})}{N_H} \\ 0 \\ \beta_V b_M \dfrac{S_V((I_H + I_{HM})}{N_H} \\ 0 \end{pmatrix},$$

The transition vector $\mathbf{V}(x)$, which represents progression, recovery, and disease-induced mortality, is expressed as

$$\mathbf{V}(x) = \begin{pmatrix} (\phi + p + \mu)E_M \\ (\gamma + \mu + \delta_M)I_M - pE_M \\ (\alpha_1 + \mu + \delta_H)I_H \\ (\alpha_2 + \varepsilon_1\gamma + \mu + \tau\delta_M)I_{HM} - \eta_1\sigma_M I_H - \omega\sigma_H I_M \\ (\theta_V + \mu_V)E_V \\ \mu_V I_V - \theta_V E_V \end{pmatrix}$$

The Jacobian matrices

$$\mathbf{F} = D\mathbf{F}(E_0), \qquad \mathbf{V} = D\mathbf{V}(E_0),$$

are evaluated at the DFE. The next-generation matrix is defined as

$$\mathbf{K} = \mathbf{F}\mathbf{V}^{-1},$$

and the basic reproduction number R_0 is defined as the spectral radius of the next-generation matrix $\mathbf{K}$. Thus, we have

$$R_0 = \rho(\mathbf{K}) = \max\left\{R_0^{(M)},\, R_0^{(H)}\right\}.$$

Here,

$$R_0^{(M)} = \sqrt{\frac{\beta_M\beta_V b_M^2\theta_V p}{\mu_V(\theta_V + \mu_V)(\phi + p + \mu)(\gamma + \mu + \delta_M)}}$$

denotes the basic reproduction number associated with malaria transmission, while

$$R_0^{(H)} = \frac{\beta_H}{(\mu + \alpha_1 + \delta_H)}$$

represents the basic reproduction number corresponding to HIV transmission through direct human-to-human contact.

Biologically, $R_0^{(M)}$ represents the average number of secondary malaria infections generated through the complete mosquito–human–mosquito transmission cycle by a single infectious individual introduced into a wholly susceptible population. The square-root structure reflects the two-stage transmission process between humans and vectors. Meanwhile, $R_0^{(H)}$ denotes the average number of secondary HIV infections produced by one infectious individual through direct human-to-human contact. The overall threshold parameter is determined by the dominant transmission pathway. Co-infection interaction parameters influence endemic dynamics but do not affect the invasion threshold at the disease-free equilibrium.

2.5.1. Sensitivity Analysis of the HIV–Malaria Co-Infection Model

Sensitivity analysis is performed to identify key parameters that most strongly influence the basic reproduction number R_0, which represents the epidemic threshold and transmission potential of the HIV–malaria co-infection system. Determining the relative importance of model parameters is essential for guiding effective intervention strategies and prioritizing public health control measures.

In this study, a normalized forward sensitivity analysis, also known as elasticity analysis, is employed. For a given parameter x, the normalized sensitivity index of R_0 with respect to x is defined as

$$\Upsilon_x^{R_0} = \frac{\partial R_0}{\partial x} \cdot \frac{x}{R_0}.$$

This index quantifies the proportional change in R_0 resulting from a proportional change in parameter x. Consequently, a sensitivity index of magnitude $|\Upsilon_x^{R_0}|$ indicates that a 1% increase in x produces an approximate $|\Upsilon_x^{R_0}|$% change in R_0, providing a clear and interpretable measure of parameter influence.

The computed normalized sensitivity indices for the HIV–malaria co-infection model are summarized in Table 3.

Table 3: Normalized sensitivity indices of the basic reproduction number R_0

No.	Parameter Description	Symbol	$\boldsymbol{\Upsilon_x^{R_0}}$
1	Human recruitment rate	Λ_H	-0.01027
2	Malaria transmission (vector to human)	β_M	$+0.25035$
3	Malaria-induced death rate	δ_M	-0.21906
4	Progression to infectious malaria	p	$+0.00150$
5	HIV transmission rate	β_H	$+0.50956$
6	Malaria recovery rate	γ	-0.01825
7	Natural human death rate	μ	-0.02640
8	Progression from HIV to AIDS	α_1	-0.41350
9	HIV-induced death rate	δ_H	-0.08270
10	Mosquito biting rate	b_M	$+0.49044$
11	Malaria transmission (human to vector)	β_V	$+0.25035$
12	Mosquito incubation progression rate	θ_V	$+0.12518$
13	Natural mosquito death rate	μ_V	-0.37553

Positive sensitivity indices indicate that increases in the corresponding parameters lead to higher values of the basic reproduction number R_0, whereas negative indices indicate a reduction in transmission potential. The results show that the HIV transmission rate (β_H) and the mosquito biting rate (b_M) have the largest positive sensitivity indices, suggesting that HIV transmission intensity and human–vector contact play a major role in determining R_0. In contrast, parameters related to disease progression and mortality, particularly the progression rate from HIV infection to AIDS (α_1) and the natural mosquito death rate (μ_V), exhibit relatively large negative sensitivity indices, indicating their association with reduced transmission potential.

2.5.2. Numerical Simulation of the Model

Numerical simulations are performed to investigate the baseline temporal dynamics of the HIV–malaria co-infection system under high-endemic conditions representative of Papua, Indonesia. Papua reports the highest combined burden of HIV and malaria nationally, with an estimated total population of $N_H = 1{,}197{,}497$, 2,541 documented HIV cases, and an Annual Parasite Incidence (API) of 199.22 per 1,000 population [3], [6], [7].

Initial conditions are selected to reflect a high-transmission environment while maintaining demographic consistency:

$$S(0) = 704{,}946, \quad E_M(0) = 250{,}000, \quad I_M(0) = 238{,}600,$$

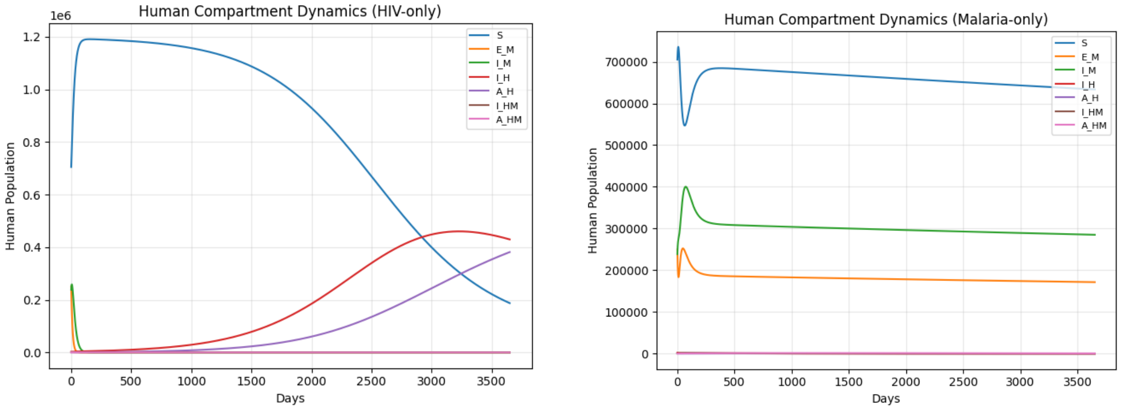
$$I_H(0) = 2{,}451, \quad A_H(0) = 500, \quad I_{HM}(0) = 1{,}200, \quad A_{HM}(0) = 300,$$

and for the mosquito population,

$$S_V(0) = 50{,}000, \quad E_V(0) = 10{,}000, \quad I_V(0) = 5{,}000.$$

All model parameters are fixed at their baseline values in order to examine the intrinsic dynamic behavior of the coupled HIV–malaria system in the absence of intervention measures.

Single-Disease Dynamics. To distinguish the intrinsic transmission mechanisms of each pathogen, the model is first simulated under isolated single-disease scenarios: (i) HIV-only transmission, obtained by suppressing malaria transmission parameters, and (ii) malaria-only transmission, obtained by excluding HIV transmission pathways. The full compartmental structure is retained to ensure structural consistency across simulations.

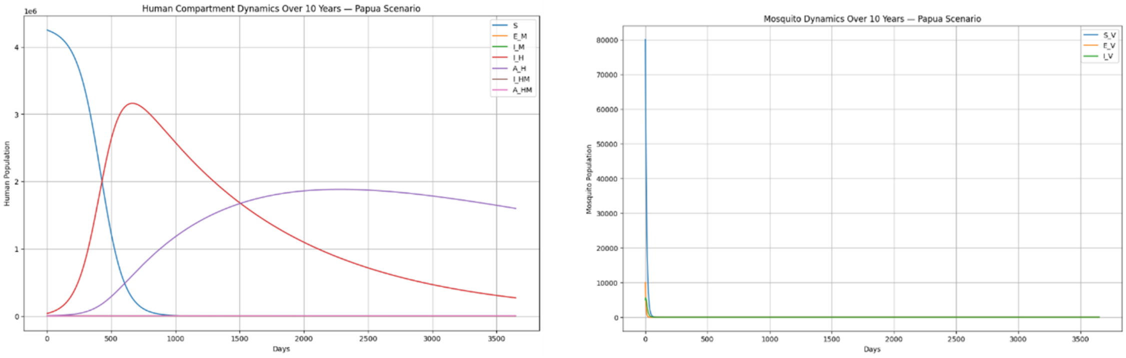


(a) HIV-only dynamics **(b)** Malaria-only dynamics

Fig. 2: Temporal dynamics under single-disease scenarios: (a) HIV-only and (b) malaria-only.

Fig. 2 illustrates the temporal behavior under these scenarios. HIV-only dynamics evolve on a relatively slower time scale, characterized by gradual progression from HIV infection to AIDS-related compartments. In contrast, malaria-only dynamics exhibit faster transient behavior driven by vector-mediated transmission, with rapid changes in exposed and infectious classes. These contrasting temporal patterns highlight the fundamentally different epidemiological time scales governing HIV and malaria transmission.

HIV–Malaria Co-infection Dynamics. The full co-infection model is subsequently simulated with all transmission pathways active over a 10-year time horizon. This configuration represents a dual-endemic environment in which HIV and malaria circulate simultaneously within human and vector populations.



(a) Human compartment dynamics over a 10-year horizon **(b)** Mosquito compartment dynamics over a 10-year horizon

Fig. 3: Baseline temporal dynamics of the HIV–malaria co-infection model under high-endemic conditions in Papua: (a) human compartments and (b) mosquito compartments.

The human population dynamics presented in Fig. 3a reveal nonlinear interactions between HIV and malaria. In particular, the co-infected compartments (I_{HM} and A_{HM}) initially increase before gradually approaching steady-state levels. This transient amplification reflects the synergistic influence of HIV-induced immunosuppression combined with sustained malaria exposure.

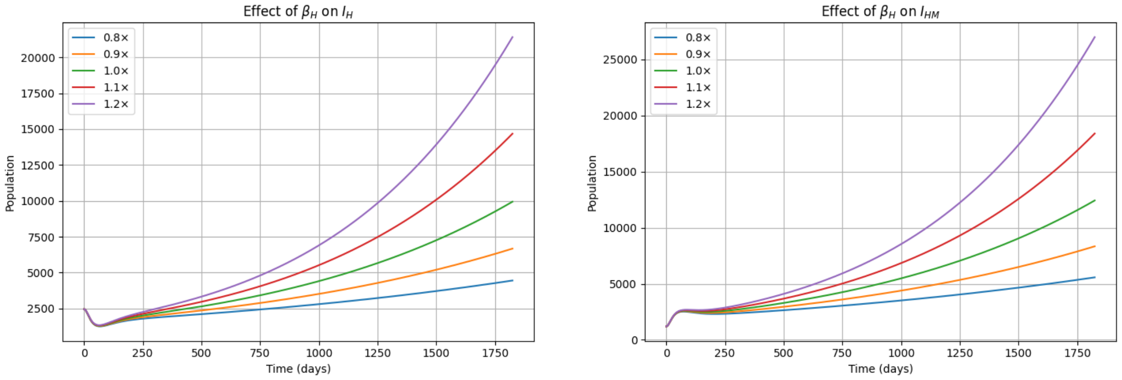
Meanwhile, the mosquito dynamics shown in Fig. 3b demonstrate persistent malaria transmission within the vector population. Although the infectious mosquito compartment (I_V) declines from its initial value, it stabilizes at a positive level, thereby maintaining continuous transmission pressure on the human population.

Overall, the numerical trajectories suggest convergence toward an endemic steady state under baseline parameter values. This equilibrium-like behavior arises from the balance between infection, disease progression, recovery, and natural or disease-induced mortality. These baseline simulations provide a reference framework for subsequent sensitivity analyses and evaluation of parameter-driven intervention strategies.

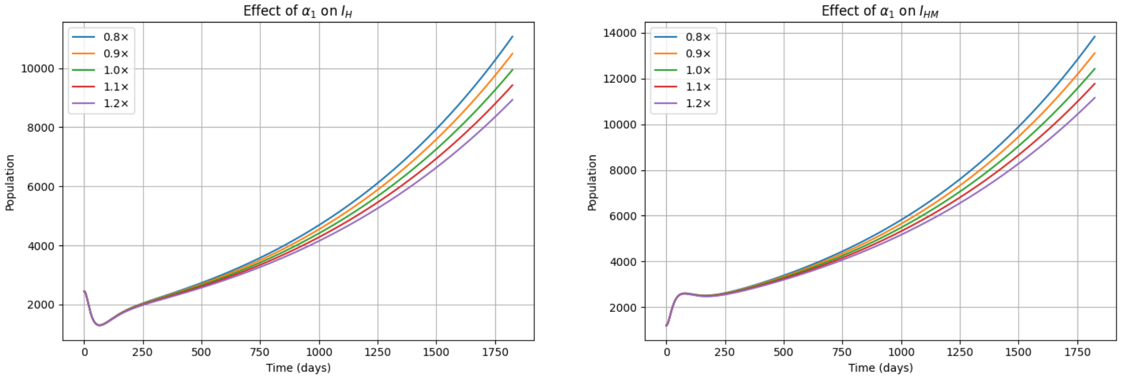
2.5.3. Numerical Simulations with Parameter Variations

Following the baseline simulations, a normalized forward sensitivity analysis was conducted to determine the parameters that most strongly influence the basic reproduction number R_0. To further illustrate the epidemiological implications of these dominant parameters, additional numerical simulations were performed by varying each parameter around its baseline value while keeping all other parameters fixed. The simulations focus on how variations in key HIV- and malaria-related parameters affect the infected human populations.

Effects of HIV-Related Parameters on I_H and I_{HM} Fig. 4 illustrates the impact of variations in the HIV transmission rate (β_H) and the HIV progression rate to AIDS (α_1) on the dynamics of HIV-only infected individuals (I_H) and co-infected individuals (I_{HM}). An increase in the HIV transmission rate β_H leads to a substantial rise in both I_H and I_{HM}. Epidemiologically, a higher transmission rate strengthens the force of HIV infection, increasing the number of newly infected individuals. As the HIV-infected population expands, the pool of individuals susceptible to malaria infection also grows, thereby elevating the number of co-infected individuals.



(a) Effect of β_H on I_H **(b)** Effect of β_H on I_{HM}

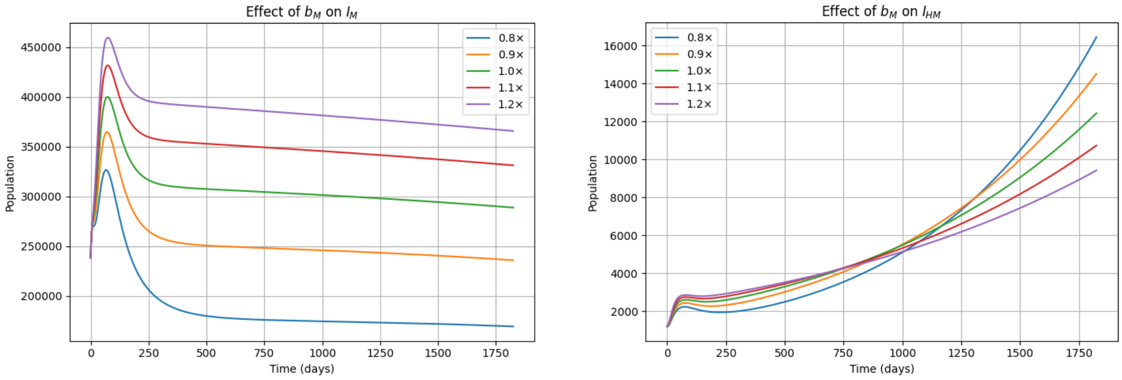


(c) Effect of α_1 on I_H **(d)** Effect of α_1 on I_{HM}

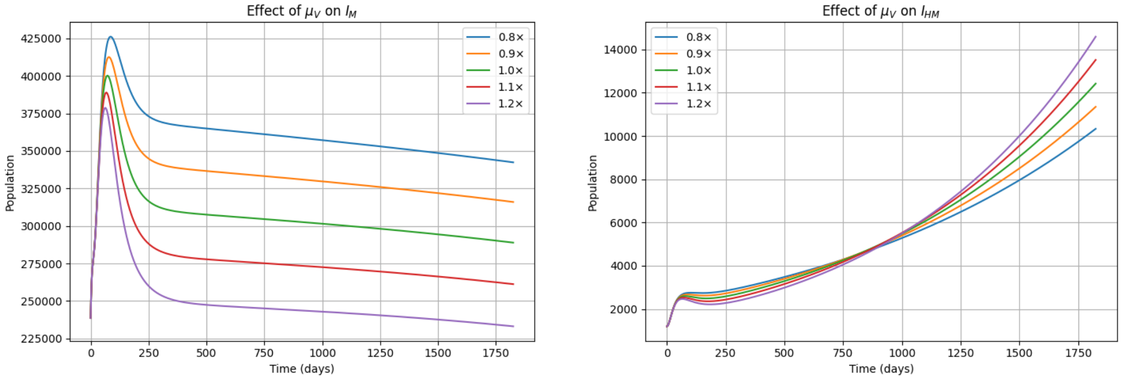
Fig. 4: Impact of dominant HIV-related parameters on the HIV-only infected population (I_H) and the co-infected population (I_{HM}). The first row illustrates the effect of the HIV transmission rate (β_H), while the second row illustrates the effect of the HIV progression rate to AIDS (α_1).

The amplification observed in I_{HM} demonstrates the strong epidemiological coupling between HIV transmission dynamics and malaria exposure in endemic regions. In contrast, increasing the progression rate α_1 accelerates the transition from the HIV-infected class to the AIDS class. Consequently, the size of the I_H compartment decreases due to faster disease progression. This reduction indirectly affects the co-infected population I_{HM}, since fewer individuals remain in the HIV-infected stage where malaria co-infection may occur. These results highlight the important role of HIV disease progression in shaping the long-term co-infection dynamics.

Effects of Malaria-Related Parameters on I_M and I_{HM}. Additional simulations examining the effect of the malaria transmission probability β_M on I_M and I_{HM} (see Figures corresponding to β_M) show a similar trend. Increasing β_M strengthens the malaria force of infection, leading to a higher peak of malaria-only infections (I_M) and a substantial increase in the co-infected population (I_{HM}). This occurs because stronger malaria transmissibility increases the likelihood that HIV-infected individuals acquire malaria, thereby enlarging the co-infection compartment.



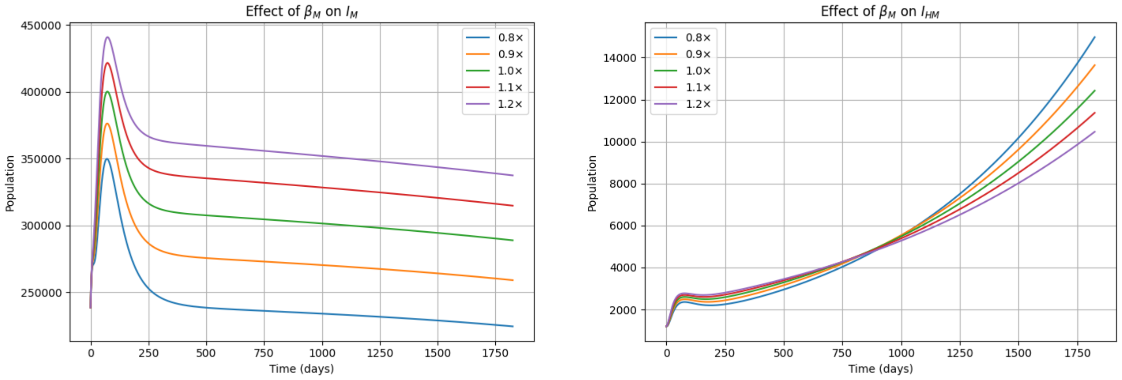
(a) Effect of b_M on I_M **(b)** Effect of b_M on I_{HM}



(c) Effect of μ_V on I_M **(d)** Effect of μ_V on I_{HM}

Fig. 5: Impact of malaria-related parameters on malaria-only infection (I_M) and HIV–malaria co-infection (I_{HM}). The first row shows the effect of mosquito biting rate (b_M), while the second row shows the effect of mosquito natural death rate (μ_V).

Fig. 6a and Fig. 6b illustrate the impact of the malaria transmission probability β_M on the dynamics of malaria-only infection (I_M) and HIV–malaria co-infection (I_{HM}). An increase in β_M strengthens the malaria force of infection, resulting in a higher peak of I_M and a substantial growth in the co-infected population I_{HM}. This occurs because higher malaria transmissibility increases the likelihood that HIV-infected individuals acquire malaria, thereby enlarging the co-infection compartment.



(a) Effect of β_M on I_M **(b)** Effect of β_M on I_{HM}

Fig. 6: Effect of the malaria transmission probability β_M on malaria-only infection (I_M) and HIV–malaria co-infection (I_{HM}).

2.6. Discussion

A deterministic compartmental model for HIV–malaria co-infection has been formulated and analyzed through reproduction number analysis, normalized forward sensitivity indices, and numerical simulations. The model incorporates direct HIV transmission, vector-mediated malaria transmission, and epidemiological interaction via co-infected compartments.

The sensitivity results indicate that the basic reproduction number R_0 is highly responsive to the HIV transmission rate and the mosquito biting rate, highlighting the dominant influence of transmission intensity parameters. In contrast, the natural mosquito death rate and the progression rate from HIV infection to AIDS have negative impacts on R_0, reflecting the role of removal and transition mechanisms in reducing transmission potential.

Numerical simulations demonstrate distinct epidemiological time scales, with HIV exhibiting slower progression dynamics and malaria showing faster transient behavior. When both infections circulate simultaneously, the system converges to an endemic equilibrium under baseline parameters, indicating sustained coexistence within the modeled population. Nonlinear coupling effects are also observed, where malaria transmission parameters indirectly affect co-infection prevalence through interaction terms.

3. Conclusion

This study provides a mathematical characterization of HIV–malaria co-infection dynamics in a dual-endemic setting. The analysis shows that transmission intensity parameters strongly determine the magnitude of R_0, while vector mortality and disease progression mechanisms contribute to reducing transmission potential. Under baseline conditions, the model predicts convergence toward an endemic steady state.

These findings offer a theoretical foundation for understanding the interaction between HIV and malaria within a deterministic modeling framework and may support further analytical extensions of the model.

CRediT Authorship Contribution Statement

Vina Lusiana served as the corresponding author and had the primary role in conceptualization, data curation, formal analysis, investigation, methodology, project administration, validation, visualization, supervision, writing the original draft, and reviewing and editing the manuscript. **Diva Hardiestya Fanny** contributed to conceptualization, formal analysis, investigation, methodology, software development, resources, and review and editing of the manuscript. **Muhammad Syazali** contributed to data curation, investigation, resources, validation, visualization, and review and editing of the manuscript. All authors discussed the results and approved the final version of the manuscript.

Declaration of Generative AI and AI-assisted technologies

The authors declare that generative AI tools (e.g., ChatGPT) were used only for language refinement and formatting, including spelling, grammar, and clarity. All scientific content, analyses, interpretations, and conclusions were developed entirely by the authors. The final manuscript was reviewed and approved by all authors.

Declaration of Competing Interest

The authors declare no conflicts of interest that could influence the research.

Funding and Acknowledgments

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

The authors would like to thank all individuals and institutions who provided support during the research and manuscript preparation, including language assistance, proofreading, and constructive academic feedback.

Data and Code Availability

All data used in this study are obtained from publicly accessible official sources. The datasets are available from the World Health Organization (WHO), the Ministry of Health of the Republic of Indonesia (Kementerian Kesehatan RI), and the Indonesian Central Bureau of Statistics (BPS) through their official publications and repositories.

Specifically, the data were retrieved from:

- **World Health Organization**, *HIV Country Profile: Indonesia* (2023). Available at: `https://www.who.int/publications/m/item/hiv-country-profile-2023-indonesia`.
- **World Health Organization**, *World Malaria Report 2024*. Available at: `https://www.who.int/publications/i/item/9789240095047`.
- **Kementerian Kesehatan Republik Indonesia**, *Laporan Tahunan Malaria Indonesia 2023*. Available at: `https://www.kemkes.go.id/resources/download/pusdatin/laporan/malaria-2023.pdf`.
- **Kementerian Kesehatan Republik Indonesia**, *Laporan Perkembangan HIV/AIDS dan IMS Triwulan IV Tahun 2024*. Available at: `https://www.kemkes.go.id/resources/download/pusdatin/laporan/hiv-ims-q4-2024.pdf`.
- **Badan Pusat Statistik Provinsi Papua**, *Statistik Papua*. Available at: `https://papua.bps.go.id/`.
- **Badan Pusat Statistik (BPS)**, *Statistik Indonesia 2024*. Available at: `https://www.bps.go.id/publication/2024/01/01/xxxxxx/statistik-indonesia-2024.html`.

No proprietary datasets were used in this study.

References

[1] World Health Organization, “Hiv country profile: Indonesia,” WHO, 2023. `https://www.who.int`.

[2] World Health Organization, “World malaria report 2024,” WHO, 2024. `https://www.who.int/publications/i/item/9789240095047`.

[3] Kementerian Kesehatan Republik Indonesia, "Laporan tahunan malaria indonesia 2023," Direktorat P2P, 2023. `https://www.kemkes.go.id`.

[4] T. E. Kwenti, "Malaria and hiv coinfection in sub-saharan africa: Prevalence, impact, and treatment strategies," *Research and Reports in Tropical Medicine*, vol. 9, pp. 123–136, 2018. DOI: `10.2147/RRTM.S154001`.

[5] J. Oyieko et al., "Longitudinal and cross-sectional analyses of asymptomatic hiv-1/malaria co-infection in kisumu county, kenya," *American Journal of Tropical Medicine and Hygiene*, vol. 108, no. 1, pp. 85–92, 2023. DOI: `10.4269/ajtmh.22-0035`.

[6] Kementerian Kesehatan Republik Indonesia, "Laporan perkembangan hiv/aids dan ims triwulan iv tahun 2024," Direktorat P2P, 2024. `https://www.kemkes.go.id`.

[7] Badan Pusat Statistik, "Statistik indonesia 2024," BPS, 2024. `https://www.bps.go.id`.

[8] Z. Mukandavire et al., "Mathematical analysis of a model for hiv–malaria co-infection," *Mathematical Biosciences and Engineering*, vol. 6, no. 2, pp. 333–362, 2009. DOI: `10.3934/mbe.2009.6.333`.

[9] B. Seidu, O. D. Makinde, and I. Y. Seini, "Mathematical analysis of the effects of hiv–malaria co-infection on workplace productivity," *Acta Biotheoretica*, vol. 63, no. 2, pp. 151–182, 2015. DOI: `10.1007/s10441-015-9255-y`.

[10] F. Nyabadza et al., "The implications of hiv treatment on the hiv–malaria co-infection dynamics: A modeling perspective," *BioMed Research International*, 2015. DOI: `10.1155/2015/659651`.

[11] F. Fatmawati, A. Windarto, and L. Hanif, "Application of optimal control strategies to hiv-malaria co-infection dynamics," *Journal of Physics: Conference Series*, vol. 974, p. 012 057, 2018. DOI: `10.1088/1742-6596/974/1/012057`.

[12] M. Rabiu, R. Willie, and N. Parumasur, "Optimal control strategies and sensitivity analysis of an hiv/aids-resistant model with behavior change," *Computational and Mathematical Methods in Medicine*, 2021. DOI: `10.1155/2021/6690215`.

[13] A. O. Oladapo et al., "Mathematical analysis of sensitive parameters on hiv–malaria co-infection," *Jambura Journal of Biomathematics*, vol. 4, no. 1, pp. 37–45, 2023. DOI: `10.34312/jjbm.v4i1.18972`.

[14] P. Paisal and A. D. Laksono, "Regional disparities of tb–hiv coinfection among national health insurance members in indonesia," *Clinical Epidemiology and Global Health*, vol. 36, p. 102 201, 2025. DOI: `10.1016/j.cegh.2025.102201`.

[15] H. Hasmi and S. P. Irab, "Uncovering the risk factors of hiv and aids in papua," *Universal Journal of Public Health*, vol. 13, no. 4, pp. 930–944, 2025. DOI: `10.13189/ujph.2025.130416`.

[16] Y. K. Ashar, A. M. Lauchan, and P. Safira, "Analysis of environmental risk factors associated with malaria incidence in papua, indonesia," *Jurnal Kesehatan Lingkungan*, vol. 17, no. 3, pp. 287–293, 2025. DOI: `10.20473/jkl.v17i3.2025.287-293`.

[17] L. Rahmah, Z. Parinding, and D. M. I. Kaseroan, "Impact of climate change on the spread of malaria in endemic tropical areas," *The Journal of Academic Science*, vol. 2, no. 5, pp. 1459–1467, 2025. DOI: `10.59613/80bwbd56`.

[18] D. Winiarti, A. Mudigdo, and B. Murti, "Determinants of recurrence and death in hiv-malaria co-infection patients in jayapura, papua, indonesia," *Journal of Epidemiology and Public Health*, vol. 4, no. 3, pp. 138–155, 2019. DOI: `10.26911/jepublichealth.2019.04.03.01`.

[19] A. Abraham and T. Tandiangnga, "Aplikasi model logistik terhadap pertumbuhan penyakit hiv/aids di provinsi papua," *Journal of Health, Education, Economics, Science, and Technology (J-HEST)*, vol. 6, no. 2, pp. 219–224, 2024. DOI: `10.37830/jhest.v6i2`.

[20] D. L. Smith and F. E. McKenzie, "Statistical analysis of the relationship between infectious diseases and environmental factors: Malaria as a case study," *Proceedings of the National Academy of Sciences*, vol. 101, no. 16, pp. 6147–6152, 2004. DOI: `10.1073/pnas.0305326101`.