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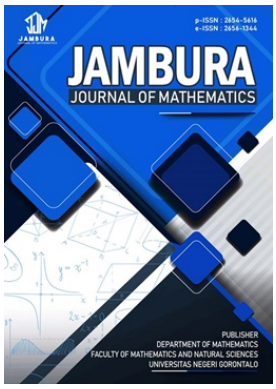
JAMBURA JOURNAL OF MATHEMATICS

Volume 8, Issue 2, Pages 265–272, August 2026

Received 15 June 2026, Revised 8 August 2026, Accepted 17 August 2026, Published 23 August 2026

To Cite this Article : R. A. D. Prasetyo, N. C. Muhammad, D. K. Arif, and M. Mardlijah, "HIV Transmission Dynamics and Workforce Productivity in Indonesia: A Nonlinear Modeling and Parameter Estimation Study", *Jambura J. Math*, vol. 8, no. 2, pp. 265–272, 2026, doi: *https://doi.org/10.37905/jjom.v8i2.39823*.

JOURNAL INFO • JAMBURA JOURNAL OF MATHEMATICS



Homepage	:	http://ejurnal.ung.ac.id/index.php/jjom/index
Journal Abbreviation	:	Jambura J. Math.
Frequency	:	Biannual (February and August)
Publication Language	:	English (preferable), Indonesia
DOI	:	https://doi.org/10.37905/jjom
Online ISSN	:	2656-1344
Editor-in-Chief	:	Hasan S. Panigoro
Publisher	:	Department of Mathematics, Universitas Negeri Gorontalo
Country	:	Indonesia
OAI Address	:	http://ejurnal.ung.ac.id/index.php/jjom/oai
Google Scholar ID	:	iWLjgaUAAAAJ
Email	:	info.jjom@ung.ac.id

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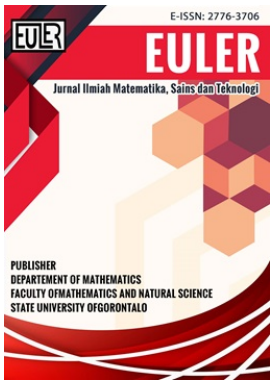

EULER : Jurnal Ilmiah Matematika, Sains, dan Teknologi

Jambura Journal of Mathematics, Volume 8, Issue 2, Pages 265–272, August 2026 https://doi.org/10.37905/jjom.v8i2.39823

Research Article

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HIV Transmission Dynamics and Workforce Productivity in Indonesia: A Nonlinear Modeling and Parameter Estimation Study

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ARTICLE HISTORY
Received 15 June 2026
Revised 8 August 2026
Accepted 17 August 2026
Published 23 August 2026

KEYWORDS
HIV transmission
Mathematical modeling
Stability analysis
Sensitivity analysis
Least squares method

ABSTRACT. *This article addresses limited multi-compartment clinical data by developing a six-compartment nonlinear mathematical model consisting of susceptible (S), protected (P), exposed (E), non-productive infected (I_n), productive infected (I_p), and AIDS phase (A) to analyze HIV transmission dynamics and workforce productivity in Indonesia. Utilizing empirical data from 2006 to 2023, parameter estimation via nonlinear least squares yielded a robust Mean Absolute Percentage Error (MAPE) of 14.20%. The system's local stability is governed by the basic reproduction number, where the baseline estimation $\mathcal{R}_0 = 0.831332 < 1$ theoretically guarantees long-term disease eradication. Linearization around the disease-free equilibrium (E_0) proved a stable focus behavior, showing trajectories that approach the steady state via damped oscillations due to clinical progression delays. Sensitivity analysis and numerical simulations identified the transmission rate from exposed individuals (β_e) and the transition rate from exposed to non-productive infected (γ) as the most critical parameters controlling $\mathcal{R}_0$. While elevated transmission from the exposed compartment forces a continuous rise in the exposed cohort, accelerating the clinical transition rate shifts the non-productive infected peak earlier and rapidly suppresses active clusters to zero. These findings provide critical insights into how clinical manifestation timing and transmission from the exposed compartment interact, which is vital for planning healthcare resource windows and safeguarding workforce productivity.*

1. Introduction

Human Immunodeficiency Virus (HIV) is an RNA virus that attacks the human immune system by targeting cells expressing the CD4 surface antigen, particularly T lymphocytes, which are crucial for regulating the immune response [1]. Damage to these cells leads to a gradual decline in immune function and weakening of the body's defenses against infection. The most advanced stage of this infection is acquired immunodeficiency syndrome (AIDS), which signifies a drastic decline in the efficiency of the immune system [2].

HIV transmission is facilitated by several factors, such as contact with blood, semen, vaginal/cervical fluids, and breast milk from an infected mother. Therefore, this virus can be transmitted through sexual intercourse, the use of contaminated needles (including as a result of workplace accidents in healthcare facilities), blood transfusions, and organ donation [3].

To date, HIV/AIDS remains an incurable disease. The available treatments only serve to inhibit viral replication to prevent complications, with Antiretroviral Therapy (ART) being the most effective method. However, access to treatment remains a global challenge; in 2018, ART coverage worldwide reached only 62% of the total 37.9 million people living with HIV/AIDS [4]. Globally, UNAIDS reported that approximately 38 million people were living with HIV in 2019 [5]. Indonesia itself is one of the countries in Southeast Asia with a significant burden of cases. Although 2021 data indicated a decline in new infections to 25,000–28,000 cases, the cumulative trend over the past decade has continued to fluctuate. According to data from the Indonesian Ministry of Health, the cumulative number of reported HIV infections from 1987 to March 2019 reached 338,363 people [6].

The most critical factor in an economic context is the age distribution of those affected, as approximately 75% of all HIV cases in Indonesia are concentrated among the productive age group [6]. The high prevalence in this demographic cluster makes HIV a real threat to social stability and the national economy. The International Labour Organization (ILO) emphasizes that workplaces play a vital role in controlling this epidemic because a decline in workers' health status reduces institutional productivity due to decreased efficiency, increased absenteeism, and a surge in corporate expenditures for healthcare, recruitment, and training [6, 7]. At the macro level, global spending on managing this disease reached approximately US$19 billion in 2018, reflecting a massive financial burden [5]. Given the close link between clinical aspects and economic stability, mathematical modeling serves as a strategic method to investigate disease transmission dynamics and predict its consequences on non-clinical aspects, particularly labor productivity.

Several studies have developed mathematical frameworks to evaluate the impact of public awareness, clinical screening, treatment capacities, optimal control, and fractional-order dynamics on HIV transmission [8–12]. However, most existing literature overlooks the integration of workforce management with

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Homepage : http://ejurnal.ung.ac.id/index.php/jjom/index / E-ISSN : 2656-1344

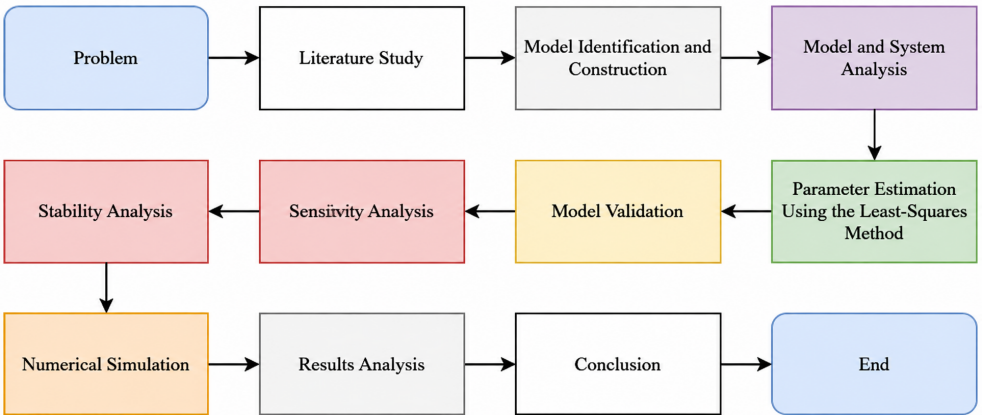

Figure 1. Research methods.

epidemiology to separate infected individuals into productive and non-productive states [13], which is critical because variations in clinical stages directly dictate macro-economic fluctuations. Furthermore, data-driven parameter estimation using comprehensive longitudinal data remains limited in frameworks tracking workforce stabilization.

Therefore, this study developed a data-driven mathematical model for HIV transmission on workforce productivity using empirical data from Indonesia spanning 2006 to 2023. This study aimed to formulate the transmission model, analyze its equilibrium points and stability, derive the basic reproduction number, estimate and validate parameters using the least squares method, and perform a local sensitivity analysis. The main contribution lies in creating an Indonesian data-driven framework that explicitly couples labor recruitment, individuals practicing effective protective behavior, and subdivided work-productivity states under an epidemic burden.

2. Methods

The research methodology used in this study combines mathematical modeling, data analysis, and computational simulation to examine the dynamics of HIV transmission and individuals practicing effective protective behavior strategies in Indonesia. Initially, the study design was based on a compartmental epidemiological model that categorizes the population into six compartments: susceptible (S), protected (P), exposed (E), non-productive infected (I_n), productive infected (I_p), and AIDS phase (A). This model framework facilitates the integration of protection dynamics, loss of protection effectiveness, and various economic productivity impacts among infected workers. The model is formulated as a system of nonlinear differential equations describing inter-compartment transitions over time.

The research methodology in this study follows a systematic approach, beginning with a comprehensive literature review to establish a theoretical framework and identify existing research gaps regarding HIV transmission models and workforce productivity. This was followed by a data collection phase, where longitudinal data on AIDS cases among the productive age group from Indonesian public health records were compiled for empirical analysis. Subsequently, the model identification and construction phase involved formulating a compartmental epidemiological model describing population dynamics among the susceptible, protected, exposed, infected, and advanced AIDS cohorts.

The system analysis phase includes examining the model's equilibrium points and local stability to explain disease behavior. Parameter estimation was then performed using the least squares method to ensure an accurate fit with field observation data, followed by a sensitivity analysis to determine the most influential parameters affecting transmission and control. Finally, numerical simulations were conducted to investigate various protection scenarios and their impact on HIV dynamics. The results were analyzed to interpret the findings, evaluate the effectiveness of education or protection strategies, and summarize key insights for HIV control in Indonesia. This structured workflow is illustrated in Figure 1.

3. Results and Discussion

3.1. Mathematical modeling of HIV/AIDS transmission dynamics

The mathematical model describes the dynamics of HIV transmission within the working-age population aged 20 years and older. The total studied population is systematically classified into six distinct compartments based on health status, awareness levels, and workforce productivity: susceptible individuals (S), individuals practicing effective protective behavior (P), exposed individuals (E), non-productive infected individuals (I_n), productive infected individuals (I_p), and individuals in the advanced AIDS stage (A).

In constructing this model, several basic assumptions are established to reflect the transmission mechanisms and their impact on productivity. The model assumes that recruitment influx enters the susceptible class (S) exclusively, while healthy individuals can transition bidirectionally between the susceptible and protected (P) groups based on preventive behaviors. HIV transmission occurs solely through sexual or risk-related contacts between susceptible individuals and virus-carrying groups, namely the exposed (E), non-productive infected (I_n), and productive infected (I_p) cohorts, meaning individuals practicing effective protective behavior cannot be infected directly without first reverting to the susceptible pool. Upon infection, individuals enter the exposed phase (E) before clinically progressing to the non-productive group (I_n) due to initial health decline or psychological distress. Individuals in this non-productive state can restore their work capacity and transition to the productive infected group (I_p) through Antiretroviral (ARV) therapy. However,

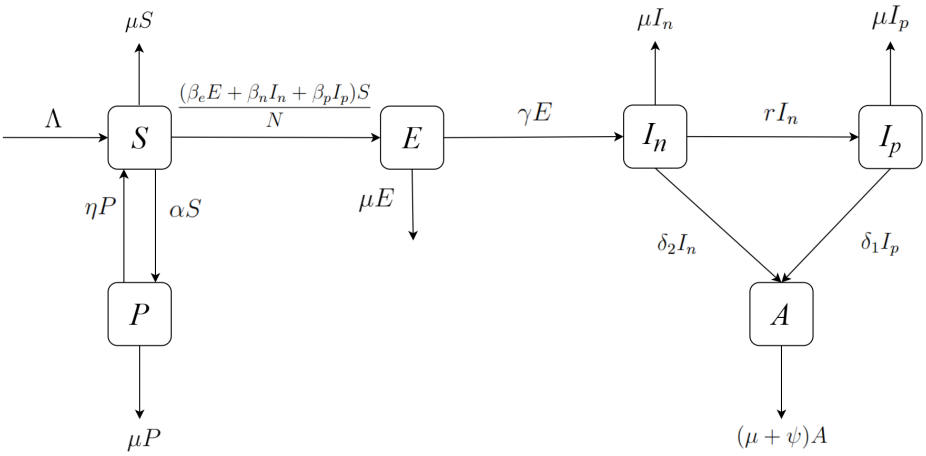

Figure 2. Compartment diagram of HIV/AIDS mathematical model.

Table 1. Model Parameters

Parameter	Definition	Unit	Values
Λ	Recruitment rate of the new workforce	People/year	Estimated
μ	Natural death rate	Per year	Estimated
α	Protection rate of the susceptible group	Per year	Fitted
η	Rate of loss of protection effectiveness	Per year	Fitted
β_e	Transmission rate from exposed phase individuals	Per year	Fitted
β_n	Transmission rate from non-productive individuals	Per year	Fitted
β_p	Transmission rate from productive individuals	Per year	Fitted
γ	Transition rate from exposed to non-productive	Per year	Fitted
r	Rate of workforce productivity restoration via ARV	Per year	Fitted
δ_1	Rate of decline from the productive group to AIDS	Per year	Fitted
δ_2	Rate of decline from the non-productive group to AIDS	Per year	Fitted
ψ	Disease-induced death rate due to AIDS	Per year	Fitted

individuals from both infected classes can deteriorate into advanced AIDS (A), resulting in an irreversible total loss of productivity. Finally, natural mortality applies uniformly across all compartments, whereas disease-induced death is strictly confined to the AIDS phase. Based on these core assumptions, the compartmental framework is systematically illustrated in Figure 2.

The transmission intensity within the productive workforce depends on the total population, $N = S + P + E + I_n + I_p + A$. Consequently, the dynamic propagation of HIV is formulated into the following nonlinear system of ordinary differential equations.

$$\frac{dS}{dt} = \dot{S} = \Lambda + \eta P - \frac{(\beta_e E + \beta_n I_n + \beta_p I_p)S}{N} - (\alpha + \mu)S, \quad (1)$$

$$\frac{dP}{dt} = \dot{P} = \alpha S - (\eta + \mu)P, \quad (2)$$

$$\frac{dE}{dt} = \dot{E} = \frac{(\beta_e E + \beta_n I_n + \beta_p I_p)S}{N} - (\gamma + \mu)E, \quad (3)$$

$$\frac{dI_n}{dt} = \dot{I}_n = \gamma E - (r + \delta_2 + \mu)I_n, \quad (4)$$

$$\frac{dI_p}{dt} = \dot{I}_p = rI_n - (\delta_1 + \mu)I_p, \quad (5)$$

$$\frac{dA}{dt} = \dot{A} = \delta_2 I_n + \delta_1 I_p - (\mu + \psi)A. \quad (6)$$

3.1.1. Disease-Free Equilibrium

The disease-free equilibrium point (E_0) represents a steady-state condition where the infection is completely absent from the workforce population. This state is analyzed by setting all infected compartments ($E = I_n = I_p = A = 0$) and all rate of change equations to zero, following the condition:

$$\frac{dS}{dt} = \frac{dP}{dt} = \frac{dE}{dt} = \frac{dI_n}{dt} = \frac{dI_p}{dt} = \frac{dA}{dt} = 0.$$

By substituting these conditions into the model, the system simplifies into a set of linear algebraic equations for the non-infected sub-populations. Solving this system yields the explicit coordinates for the disease-free equilibrium point E_0:

$$\begin{aligned} E_0 &= \left(S_0^*, P_0^*, E_0^*, I_{n0}^*, I_{p0}^*, A_0^*\right) \\ &= \left(\frac{\Lambda(\eta + \mu)}{\mu(\alpha + \eta + \mu)}, \frac{\Lambda\alpha}{\mu(\alpha + \eta + \mu)}, 0, 0, 0, 0\right). \end{aligned}$$

3.1.2. Basic reproduction number

From an epidemiological perspective, the basic reproduction number ($\mathcal{R}_0$) derived from this model acts as the critical

threshold governing the long-term behavior of the epidemic. When targeted interventions successfully reduce this threshold below unity ($\mathcal{R}_0 < 1$), the disease-free equilibrium point becomes locally asymptotically stable, which mathematically guarantees the eradication of the infection from the population as transmission chains collapse. On the other hand, if $\mathcal{R}_0 > 1$, the infection will successfully establish itself within the population, maintaining a persistent endemic state [14].

To derive this threshold, the next-generation matrix approach is utilized to rigorously isolate the transmission component (F) from the non-transmission transition interactions (V). This framework ensures that the spectral radius of the operator matrix FV^{-1} precisely quantifies the expected number of secondary infections generated by a single infectious individual throughout their entire infectious duration [15]. The mathematical procedure to compute $\mathcal{R}_0$ focuses on the disease compartments (E, I_n, I_p). Evaluated at the disease-free equilibrium (E_0), the total population reaches a steady state defined by $N_0 = \frac{\Lambda}{\mu}$, yielding a normalized susceptible population fraction that satisfies:

$$\frac{S_0^*}{N_0} = \frac{\eta + \mu}{\alpha + \eta + \mu}.$$

The new infection matrix (F) and the transition matrix (V) evaluated at E_0 are derived as follows:

$$F = \begin{bmatrix} \beta_e \left(\frac{S_0^*}{N_0}\right) & \beta_n \left(\frac{S_0^*}{N_0}\right) & \beta_p \left(\frac{S_0^*}{N_0}\right) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \gamma + \mu & 0 & 0 \\ -\gamma & r + \delta_2 + \mu & 0 \\ 0 & -r & \delta_1 + \mu \end{bmatrix}.$$

The inverse of the transition matrix V is formulated as:

$$V^{-1} = \begin{bmatrix} \frac{1}{\gamma+\mu} & 0 & 0 \\ \frac{\gamma}{(\gamma+\mu)(r+\delta_2+\mu)} & \frac{1}{r+\delta_2+\mu} & 0 \\ \frac{\gamma r}{(\delta_1+\mu)(\gamma+\mu)(r+\delta_2+\mu)} & \frac{r}{(\delta_1+\mu)(r+\delta_2+\mu)} & \frac{1}{\delta_1+\mu} \end{bmatrix}.$$

The basic reproduction number ($\mathcal{R}_0$) is obtained from the spectral radius of the next-generation operator matrix, $\mathcal{R}_0 = \rho\left(FV^{-1}\right)$. Analytically, the explicit formulation of $\mathcal{R}_0$ is expressed as follows:

$$\mathcal{R}_0 = \left(\frac{\eta + \mu}{\alpha + \eta + \mu}\right) \frac{1}{\gamma + \mu} \left[\beta_e + \frac{\beta_n \gamma}{r + \delta_2 + \mu} + \frac{\beta_p \gamma r}{(r + \delta_2 + \mu)(\delta_1 + \mu)}\right]. \tag{7}$$

3.1.3. Endemic Equilibrium Point

The endemic equilibrium point (E_1) represents the persistence of HIV infection within the population. Let $K_1 = r + \delta_2 + \mu$ be defined as a constant. Through algebraic substitution in the steady-state system using the $\mathcal{R}_0$ formulation, the explicit coordinates for the equilibrium point E_1 are obtained as follows:

$$S_1^* = \frac{N^*(\eta + \mu)}{\mathcal{R}_0(\alpha + \eta + \mu)},$$

$$P_1^* = \frac{N^*\alpha}{\mathcal{R}_0(\alpha + \eta + \mu)},$$

$$E_1^* = \frac{\Lambda\mathcal{R}_0 - N^*\mu}{\mathcal{R}_0(\gamma + \mu)},$$

$$I_{n1}^* = \frac{\gamma(\Lambda\mathcal{R}_0 - N^*\mu)}{\mathcal{R}_0(\gamma + \mu)K_1},$$

$$I_{p1}^* = \frac{\gamma r(\Lambda\mathcal{R}_0 - N^*\mu)}{\mathcal{R}_0(\delta_1 + \mu)(\gamma + \mu)K_1},$$

$$A_1^* = \frac{\gamma(\Lambda\mathcal{R}_0 - N^*\mu)(\delta_1\delta_2 + \delta_2\mu + \delta_1 r)}{\mathcal{R}_0(\delta_1 + \mu)(\gamma + \mu)(\mu + \psi)K_1},$$

where N^* represents the total population at the steady state. The endemic equilibrium point E_1 exists and is positive if and only if $\mathcal{R}_0 > 1$.

3.2. Parameter Estimation

3.2.1. HIV/AIDS Cases in Indonesia

This study utilizes secondary data concerning the cumulative annual clinical AIDS cases among the productive age workforce aged 20 years and older in Indonesia from 2006 to 2023, as detailed in Table 2. These data are derived from the official epidemiological monitoring records published by the Ministry of Health of the Republic of Indonesia [16–18]. These empirical data serve as the baseline reference for parameter estimation using the least squares method to evaluate the goodness-of-fit between the mathematically simulated trajectories and real-world dynamics.

Table 2. Reported cumulative AIDS cases among individuals aged 20 years and older, 2006-2023.

Year	Case	Year	Case
2006	2,909	2015	8,249
2007	3,739	2016	9,303
2008	4,340	2017	9,693
2009	5,323	2018	9,396
2010	5,470	2019	6,016
2011	6,248	2020	7,386
2012	8,927	2021	4,916
2013	9,596	2022	8,465
2014	7,879	2023	14,638

The model parameters are classified into demographic parameters calculated directly from empirical data and epidemiological parameters estimated via optimization. Based on Indonesia's life expectancy of 68.47 years in 2006 [19], the natural death rate μ is determined by:

$$\mu = \frac{1}{68.47} \text{ per year.}$$

Given an initial workforce of 106,400,000 individuals in the baseline year [20], the constant recruitment rate Λ is established as:

$$\Lambda = \frac{106,400,000}{68.47} \text{ individuals per year.}$$

Furthermore, the initial conditions for each compartment in the baseline year 2006 are extracted proportionally from these benchmarks. To match the first entry in Table 2, the initial AIDS compartment is set to $A(0) = 2,909$. From the 7,195 recorded

HIV cases at that time, the infected classes are distributed as $I_n(0) = 1,000$ and $I_p(0) = 6,195$. The initial values for the latent class and the protected group are set to $E(0) = 14,390$ and $P(0) = 5,000,000$. By subtracting these sub-populations from the total initial workforce, the initial condition for the susceptible group $S(0)$ is obtained as:

$$\begin{aligned} S(0) &= 106,400,000 - 2,909 - 1,000 - 6,195 \\ &\quad - 5,000,000 - 14,390 \\ &= 101,375,506. \end{aligned}$$

3.2.2. Parameter Estimation Using Nonlinear Least-Squares Method

The nonlinear least-squares method is applied to determine the optimal parameter values by minimizing a least-squares objective function between the observed empirical data and the model predictions, where the optimization process focuses on minimizing an objective function defined as the sum of squared differences between the empirical cumulative AIDS cases recorded annually and the model's corresponding predicted trajectory for the AIDS compartment (A) at each specific time step. In this study, this estimation process is computationally executed via the nonlinear least squares estimation solver, specifically utilizing the `lsqcurvefit` function in MATLAB, which is intentionally designed to visualize long-term trends alongside reported annual fluctuations to extract realistic, robust, and data-driven parameter estimates [21] since the empirical datasets obtained exclusively provide the numbers for clinically reported symptomatic cases that directly correspond to the active AIDS compartment in our dynamic system model.

Conversely, direct empirical observations for several historical sub-populations—such as the latent or exposed phase individuals (E), protected groups (P), or specific active infected stages (I_n, I_p) are completely unavailable in public datasets. To overcome the absence of complete empirical data, these unobserved compartments are mathematically reconstructed through the model's internal dynamic structure. This reconstruction is accomplished by leveraging established parameter baseline boundaries from existing epidemiological literature and systematically adjusting the initial conditions. This integrated mathematical approach ensures that reliable parameter estimation and comprehensive policy scenario analyses remain highly feasible and analytically valid, even when dealing with partially unobserved biological systems [22].

3.2.3. Model Fitting Evaluation

The remaining transmission and transition parameters are optimized iteratively using the Least Squares method based on the time-series data of AIDS cases. The comprehensive summary of the calibrated and assigned model parameter values is compiled in Table 3.

Once all demographic values and Least Squares estimation results are acquired, numerical simulations are executed using the fourth-order Runge-Kutta method to project the dynamics of the AIDS compartment (A) from 2006 to 2023. A visual comparison between the model's trajectory and the real AIDS case data in Indonesia is illustrated in Figure 3. As shown in Figure 3, the model simulation curve generally succeeds in tracking the long-term growth trend of the real AIDS case data. During the initial phase (2006–2011), the model exhibits an adequate level of accuracy in capturing empirical fluctuations. Although minor deviations occur in certain periods due to external field factors, the simulation line remains tightly aligned with the long-term trend of the reported data, yielding an in-sample fitting error of 14.20% in terms of Mean Absolute Percentage Error (MAPE). Consequently, the combination of initial conditions and optimized parameters provides an acceptable fit to the observed data on HIV/AIDS transmission within the Indonesian workforce, providing a reliable foundation for policy sensitivity analysis.

Table 3. Parameter estimation results.

Parameter	Values	Parameter	Values
Λ	$\frac{106,400,000}{68_{1}47}$	β_p	0.190065
μ	$\frac{1}{68.47}$	γ	0.338211
α	0.448293	r	0.354515
η	0.190000	δ_1	0.559315
β_e	0.750000	δ_2	0.190000
β_n	0.190000	ψ	0.750000

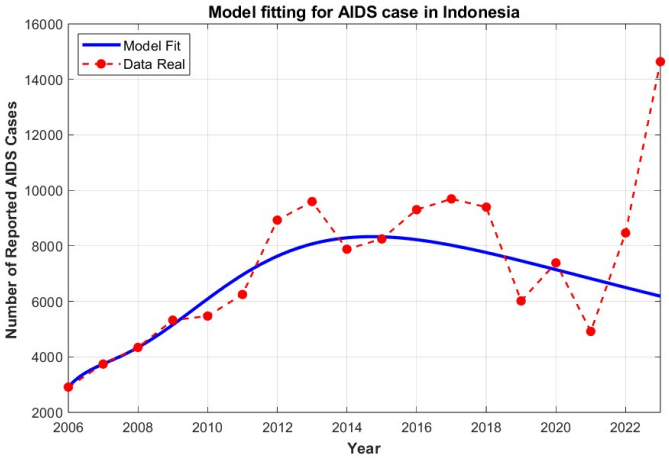

Figure 3. Comparison of observed and fitted annual AIDS cases in Indonesia.

3.3. *Stability Analysis*

The local stability of the system is evaluated by analyzing the eigenvalues of the Jacobian matrix evaluated at the disease-free equilibrium point (E_0). By applying partial differentiation with respect to the state variables (S, P, E, I_n, I_p, A), the general symbolic form of the Jacobian matrix (J) for the system is formulated as follows:

$$J = \begin{bmatrix} J_{11} & J_{12} & J_{13} & J_{14} & J_{15} & J_{16} \\ \alpha & J_{22} & 0 & 0 & 0 & 0 \\ J_{31} & -\phi\frac{S}{N} & J_{33} & J_{34} & J_{35} & -\phi\frac{S}{N} \\ 0 & 0 & \gamma & J_{44} & 0 & 0 \\ 0 & 0 & 0 & r & -(\delta_1+\mu) & 0 \\ 0 & 0 & 0 & \delta_2 & \delta_1 & -(\mu+\psi) \end{bmatrix}, \quad (8)$$

where

$$J_{11} = -\phi\left(1 - \frac{S}{N}\right) - (\alpha + \mu), \quad J_{12} = \eta + \phi\frac{S}{N},$$
$$J_{13} = -\frac{\beta_e S}{N} + \phi\frac{S}{N}, \quad J_{14} = -\frac{\beta_n S}{N} + \phi\frac{S}{N},$$
$$J_{15} = -\frac{\beta_p S}{N} + \phi\frac{S}{N}, \quad J_{16} = \phi\frac{S}{N},$$
$$J_{22} = -(\eta + \mu), \quad J_{31} = \phi\left(1 - \frac{S}{N}\right),$$
$$J_{33} = \frac{\beta_e S}{N} - \phi\frac{S}{N} - (\gamma + \mu), \quad J_{34} = \frac{\beta_n S}{N} - \phi\frac{S}{N},$$
$$J_{35} = \frac{\beta_p S}{N} - \phi\frac{S}{N}, \quad J_{44} = -(r + \delta_2 + \mu).$$

By substituting the optimized and demographic parameter values from Table 3 into eq. (8) at the disease-free equilibrium state, the non-infected population ratios stabilize at their respective steady-state benchmarks. This yields the numerical Jacobian matrix evaluated at the disease-free equilibrium:

$$J(E_0) = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} & a_{15} & 0 \\ a_{21} & a_{22} & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{33} & a_{34} & a_{35} & 0 \\ 0 & 0 & a_{43} & a_{44} & 0 & 0 \\ 0 & 0 & 0 & a_{54} & a_{55} & 0 \\ 0 & 0 & 0 & a_{64} & a_{65} & a_{66} \end{bmatrix},$$

where

$$a_{11} = -0.46290, \quad a_{12} = 0.19000, \quad a_{13} = -0.23503,$$
$$a_{14} = -0.05954, \quad a_{15} = -0.05956, \quad a_{21} = 0.44829,$$
$$a_{22} = -0.20460, \quad a_{33} = -0.11778, \quad a_{34} = 0.05954,$$
$$a_{35} = 0.05956, \quad a_{43} = 0.33821, \quad a_{44} = -0.55912,$$
$$a_{54} = 0.35452, \quad a_{55} = -0.57392, \quad a_{64} = 0.19000,$$
$$a_{65} = 0.55932, \quad a_{66} = -0.76460.$$

Through characteristic polynomial evaluation of $J(E_0)$, the calculated eigenvalues are determined as $\lambda_1 = -0.764605$, $\lambda_2 = -0.014605$, $\lambda_3 = -0.652898$, $\lambda_4 = -0.599796 + 0.113989i$, $\lambda_5 = -0.599796 - 0.113989i$, and $\lambda_6 = -0.051230$. Since all calculated eigenvalues possess strictly negative real parts ($\mathrm{Re}(\lambda_i) < 0$), these negative values dictate that any trajectory starting near the steady state will naturally decay over time, while the distinct complex conjugate pair $\lambda_{4,5}$ indicates damped oscillatory behavior in a portion of the local dynamics. Epidemiologically, this behavior implies that under the baseline threshold $\mathcal{R}_0 < 1$, the eradication of the infected sub-populations (E, I_n, I_p, A) will not follow a strictly monotonic decline but will instead experience minor, temporary fluctuations. Crucially, because all transient perturbations rapidly decay toward zero, the disease-free equilibrium (E_0) is proved to be locally asymptotically stable, theoretically guaranteeing long-term HIV eradication within the national workforce population.

3.4. *Sensitivity Analysis*

The sensitivity analysis conducted in this research is a local sensitivity analysis. The normalized sensitivity index of variable X, continuously differentiable with respect to a given parameter p, is defined as follows:

$$C_p^X = \frac{\partial X}{\partial p} \times \frac{p}{X},$$

where X is the variable to be analyzed and p is the parameter. The higher the absolute value of the sensitivity index, the more influential the parameter is to the system. Positive and negative signs indicate the direction of the relationship between the parameter and the analyzed variable, specifically the basic reproduction number ($\mathcal{R}_0$).

This section focuses on analyzing the sensitivity of parameters relative to the basic reproduction number. A sensitivity analysis of $\mathcal{R}_0$ was conducted to determine which parameters exert the most significant influence on $\mathcal{R}_0$. Substituting the parameter values obtained from the Least Squares estimation yields the baseline value of $\mathcal{R}_0 = 0.831332$. The sensitivity index of the parameter α is:

$$C_\alpha^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathcal{R}_0}.$$

Using the same calculus procedure, the sensitivity indices for the other remaining system parameters were analogously obtained. The parameter values in Table 3 are used to calculate each sensitivity index value. To evaluate the consistency of the relationship between the parameters and the basic reproduction number, the parameter values were increased and decreased by 10%, and the resulting variations in the actual value of the basic reproduction number were analyzed. The comprehensive calculation results are summarized in Table 4.

Table 4. Sensitivity indices of model parameters relative to $\mathcal{R}_0$.

Parameter	Sensitivity Index	$\mathcal{R}_0$: $p - 10\%$	$\mathcal{R}_0$: $p + 10\%$
α	-0.686620	0.892622	0.777919
β_e	0.801326	0.764716	0.897949
η	0.637609	0.776737	0.882840
β_n	0.122796	0.821124	0.841541
β_p	0.075878	0.825024	0.837640
δ_2	-0.067513	0.837142	0.825904
γ	-0.759931	0.901206	0.773683
δ_1	-0.073947	0.838144	0.825731
r	-0.050093	0.835779	0.827416
μ	0.000496	0.831281	0.831363

The sensitivity index results indicate that the most influential parameters on $\mathcal{R}_0$ are the transmission rate β_e and the transition rate γ. Specifically, β_e has a positive effect on $\mathcal{R}_0$, whereas γ has a negative effect. To test whether the relationship between the parameters and the basic reproduction number is consistent, a 10% increase or decrease in the parameter value is applied to observe changes in the basic reproduction number. Increasing the beta parameter (β_e) by 10% increases the $\mathcal{R}_0$ value from 0.831332 to 0.897949, while decreasing it by 10% reduces the $\mathcal{R}_0$ value to 0.764716. Conversely, for the gamma parameter (γ) which exhibits a negative sensitivity index of -0.759931, increasing its value by 10% decreases the $\mathcal{R}_0$ value to 0.773683, whereas a 10% reduction shifts the $\mathcal{R}_0$ value upward to 0.901206. This demonstrates that the analysis results are consistent with the experimental results on the $\mathcal{R}_0$ value.

3.5. Numerical Simulation

In this section, numerical simulations are carried out to analyze the dynamics of the HIV transmission model and its impact on workforce productivity in Indonesia. The simulations were run using MATLAB software to observe population trajectories over the period from 2006 to 2023, matching the timeline of the available empirical data. The simulation applies the calibrated parameter set from Table 3 and the initial conditions representing the 2006 demographic and epidemiological benchmarks.

Based on the core dynamics of disease transmission and clinical progression, the transmission rate from exposed individuals β_e and the transition rate from exposed to non-productive infected γ were identified as key strategic parameters controlling system trajectories. Therefore, the simulation focused on two hypothetical scenarios to examine the sensitivity of the exposed population (E) to changes in transmission rates from the exposed phase, as well as the infected non-productive population (I_n) to variations in the clinical progression rate from the latent stage.

The parameter β_e measures the transmission intensity originating from the exposed compartment. To test the system's resilience against increased transmission from this latent phase, a simulation was carried out by varying the value of β_e across five discrete levels: $\beta_e = 0.1000$, $\beta_e = 0.3000$, $\beta_e = 0.5000$, $\beta_e = 0.7500$, and $\beta_e = 1.0000$. The temporal response of the exposed population (E) over the simulated timeline is presented in Figure 4.

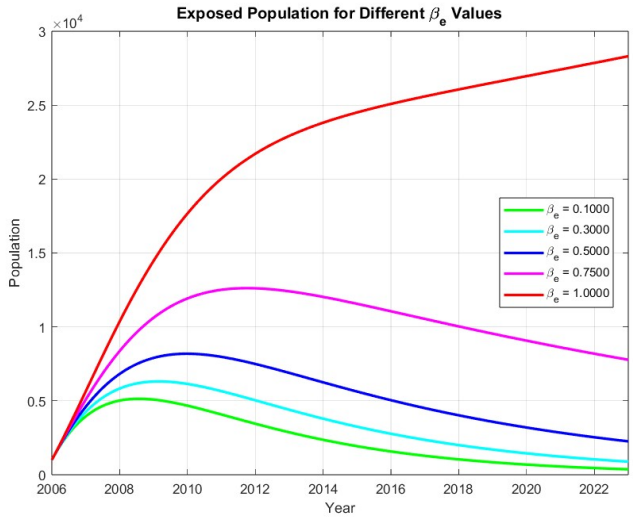

Figure 4. Dynamics of the exposed population under different values of β_e.

The simulation results in Figure 4 show that at lower transmission rates ($\beta_e = 0.1000$ to $\beta_e = 0.7500$), the exposed population experiences a relatively transient peak within the first few years (around 2009–2011) before gradually declining toward control levels by 2023. This early peak occurs due to the rapid conversion of the initial highly vulnerable sub-populations. An interesting phenomenon occurs when the transmission rate is increased to the maximum threshold ($\beta_e = 1.0000$). Theoretically, a sufficiently large increase in β_e disrupts the declining trend observed under the lower-transmission scenarios, causing the exposed population curve to rise aggressively and continuously without reaching a turnaround point, approaching nearly 2.8×10^4 individuals by the end of 2023. This indicates that high contact intensities prevent transmission saturation within this 17-year observation window, leading to a persistent epidemiological burden.

Meanwhile, the parameter γ represents the transition rate from the exposed phase to the infected non-productive population (I_n). Through this scenario analysis, we simulate the structural impact of varying γ from a baseline value of $\gamma = 0.3382$ towards more accelerated clinical progression rates, namely: $\gamma = 0.3382$, $\gamma = 0.4000$, $\gamma = 0.6000$, $\gamma = 0.8000$, and $\gamma = 1.0000$. The dynamic response of the infected non-productive group is illustrated in Figure 5.

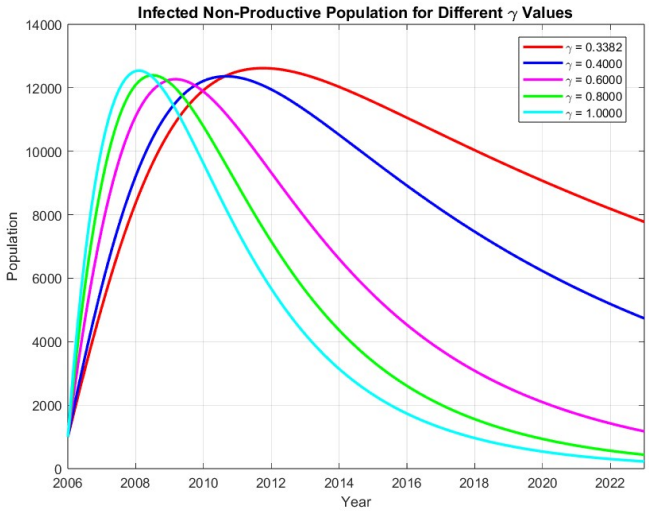

Figure 5. Dynamics of the non-productive infected population under different values of γ.

Figure 5 confirms the epidemic theory that variations in the clinical progression rate from the latent phase heavily alter the timing and magnitude of peak infections. In the baseline scenario ($\gamma = 0.3382$), the infected non-productive population reaches its maximum peak latest (around 2011–2012) at approximately 12,600 individuals before exhibiting a slow decay. As γ is sequentially increased to higher levels ($\gamma = 0.6000$ to $\gamma = 1.0000$), a notable shift occurs: the peak of the I_n population is forced to occur significantly earlier (shifting backward to 2008) and drops drastically thereafter. Ramping up γ to 1.0000 causes the curve to plunge sharply, reaching a near-zero steady state well before 2022. This occurs because the influx from the exposed class is rapidly concentrated in the earlier period, exhausting the latent reservoir quicker and accelerating the transition towards subsequent stages. This experiment provides critical insights into how the acceleration of clinical manifestation shifts the epidemiological timeline, which is vital for planning healthcare resource windows and workforce stabilization.

4. Conclusion

This research successfully addresses the challenge of limited multi-compartment clinical data by developing a nonlinear mathematical model of HIV transmission divided into six epi-

demiological compartments: susceptible (S), exposed (E), non-productive infected (I_n), productive infected (I_p), AIDS (A), and protected (P). The differentiation within the infected workforce classes proved crucial for capturing the long-term clinical and economic impacts of HIV dynamics on workforce productivity. By integrating demographic benchmarks and epidemiological data spanning from 2006 to 2023, the parameter estimation yielded an in-sample fitting error of 14.20%, confirming the model's accuracy in capturing empirical fluctuations. Theoretical system analysis confirmed the mathematical criteria for disease control. The derived basic reproduction number ($\mathcal{R}_0$) serves as the threshold parameter governing the stability of the disease-free equilibrium. With the baseline estimation yielding $\mathcal{R}_0 = 0.831332$, the system theoretically guarantees that transmission chains can be broken since $\mathcal{R}_0 < 1$, leading to the gradual eradication of the infection in the long term. Furthermore, local sensitivity analysis identified the transmission rate from exposed individuals (β_e) and the transition rate from exposed to non-productive infected (γ) as the two most sensitive parameters wielding dominant control over the epidemic's reproduction index and trajectory fluctuations.

These analytical findings are strongly verified by numerical simulations of hypothetical scenarios from 2006 to 2023. Simulating an increased transmission rate from the latent phase (β_e) reveals that elevated contact intensity forces the exposed population (E) to rise continuously without reaching short-term saturation. Conversely, accelerating the clinical transition rate (γ) demonstrates that faster progression from the latent reservoir effectively shifts the infected non-productive (I_n) peak significantly earlier, rapidly depleting the exposed pool and suppressing active clusters toward a near-zero steady state in the long run. Despite these valuable insights, the study's primary limitation lies in its deterministic framework, which omits stochastic behavioral variations and localized demographic disparities within the national workforce, providing critical insights for epidemiological forecasting to safeguard workforce productivity.

Author Contributions. **Rizqi Aridh Dwi Prasetyo**: Conceptualization, methodology, software, validation, formal analysis, investigation, data curation, writing—original draft preparation, visualization. **Nuansa Cahaya Muhammad**: Conceptualization, methodology, software, validation, formal analysis, investigation, data curation, writing—original draft preparation. **Didik Khusnul Arif**: Conceptualization, methodology, validation, formal analysis, writing—review and editing, supervision. **Mardlijah**: Conceptualization, methodology, validation, resources, project administration, writing—review and editing, supervision.

Acknowledgement. The authors thank the Department of Mathematics, Institut Teknologi Sepuluh Nopember, Surabaya, for its institutional support.

Funding. This research was not funded by any institution.

Conflict of interest. There are no conflicts of interest regarding the publication of this paper.

Data availability. The data used in this study were obtained from the publicly available reports cited in References [16–20].

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