



# Can Indonesia Eliminate Tuberculosis by 2030? A Deterministic Epidemic Model Approach

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## ABSTRACT

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Indonesia, bearing the world's second-highest tuberculosis (TB) burden, has mandated a national target to eliminate TB by 2030, aiming for an incidence rate of 65 per 100,000 population. This study aims not only to project future transmission dynamics but also to systematically explore the specific epidemiological barriers, namely, drug resistance and relapse mechanisms, that hinder achieving this goal. To address the heterogeneity of TB transmission, we developed a novel deterministic SVE3I3R model. This framework stratifies the population into vaccinated, latent Tuberculosis Infection (LTBI), and infectious compartments, explicitly distinguishing among Drug-Susceptible (DS-TB), Multidrug-Resistant (MDR-TB), and Extensively Drug-Resistant (XDR-TB) strains. The resulting system of ordinary differential equations was solved numerically using the fourth-order Runge-Kutta (RK4) method to ensure stability and accuracy in simulating long-term epidemiological trends from 2023 to 2030. Parameters were calibrated using national reports and literature specific to the Indonesian context. Projections indicate that Indonesia will miss the 2030 elimination target by a significant margin. The model forecasts a TB incidence rate of 321 per 100,000 population by 2030, nearly five times the national benchmark. The analysis reveals that failure to reach the target is mechanistically driven by a "relapse trap" among recovered individuals and an alarming exponential surge in resistant strains (MDR-TB and XDR-TB). These findings suggest that current control strategies are insufficient not merely in scale but in structure. Evidence-based policy must urgently shift from standard intervention to aggressive interruption of resistance pathways and enhanced management of the latent reservoir to prevent the projected demographic resurgence.



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## A. INTRODUCTION

Tuberculosis (TB) remains a leading global cause of death from a single infectious agent, resulting in an estimated 1.25 million fatalities in 2023, nearly double those from HIV/AIDS. Despite being preventable and curable, TB incidence reached over 10 million new cases (134 per 100,000 people), with 87% concentrated in 30 high-burden countries. India, Indonesia, China, the Philippines, and Pakistan account for 56% of cases. Affected individuals were 55%

men, 33% women, and 12% children. The rise in new diagnoses to 8.2 million is linked to COVID-19-related delays. A major concern is multidrug- or rifampicin-resistant TB (MDR/RR-TB); while 175,923 cases were diagnosed in 2023, the estimated incidence was 400,000 cases (Saputra et al., 2024; WHO, 2024).

Indonesia faces a critical tuberculosis (TB) burden, ranking as the world's second-largest contributor to the global epidemic and accounting for 10% of all cases. With an estimated national incidence of 387 per 100,000 people, the country had approximately 1,090,000 cases and 130,927 deaths in 2023, a mortality rate equivalent to 14 deaths per hour. Furthermore, an estimated 29,535 cases of rifampicin-resistant TB (RR-TB) were reported. This urgent public health crisis underscores the necessity of meeting the ambitious targets of the WHO's End TB Strategy and the UN Sustainable Development Goals, which aim to drastically reduce TB incidence and mortality by 2030 and 2035, respectively (Meiyanti et al., 2024; Sasmita et al., 2025; WHO, 2024).

Indonesia's Presidential Regulation No. 67 of 2021 mandates a national strategy to eliminate TB by 2030, targeting reductions in incidence to 65 per 100,000 and mortality to 6 per 100,000 (Presidential Regulation Number 67 of 2021 Concerning Tuberculosis Control, 2021). The plan prioritizes improving healthcare access, early diagnosis, treatment, and preventive measures. However, persistent constraints in healthcare systems and funding hinder progress. Overcoming these barriers and addressing the complex dynamics of TB transmission will therefore require innovative and effective approaches.

Deterministic epidemic models are instrumental for analyzing TB transmission and evaluating interventions. These models employ mathematical and computational techniques to simulate disease spread, incorporating demographic, environmental, and health data (Sofonea et al., 2022). Established frameworks like the SIR and SEIR models have been applied to predict TB dynamics, with studies in Ghana recommending improved early detection (Mettle et al., 2020). Research in China and Pakistan using SVEIR and SLITR models further identified increasing treatment rates as a cost-effective elimination strategy (Liu et al., 2020; Ullah et al., 2019).

Recent advancements include modified frameworks such as SVIR, SE3I3R, and SVEIR models that investigate the roles of vaccination, treatment failure, cure rates, and improve prediction accuracy (Ginting et al., 2024; Sasmita et al., 2019; Sulayman et al., 2021). To effectively translate this national mandate into achievable outcomes, a robust analytical framework is required to forecast epidemiological trends under current interventions. Deterministic epidemic models are instrumental for analyzing these complex transmission dynamics and evaluating the feasibility of policy targets

This study aims to evaluate Indonesia's prospects of achieving its 2030 TB elimination target, as mandated by Presidential Regulation No. 67 of 2021. We develop a novel deterministic compartmental model for TB transmission. Building on the SEIR framework, our model introduces a Susceptible-Vaccinated-Three Exposed-Three Infectious-Recovered (SVE3I3R) structure to enhance realism by more accurately reflecting TB pathogenesis. However, existing models often overlook the detailed stratification of drug resistance levels, specifically the distinct transmission dynamics of Multidrug-Resistant (MDR) and Extensively

Drug-Resistant (XDR) TB. This study addresses this critical gap by developing a comprehensive SVE3I3R model tailored to the Indonesian context.

Unlike standard SEIR frameworks, the proposed SVE3I3R model explicitly differentiates between Drug-Susceptible (DS), MDR, and XDR tuberculosis strains. This granular stratification provides a significant conceptual advantage, enabling a more precise assessment of how drug resistance impedes elimination efforts and capturing the impact of relapse dynamics often simplified in previous studies. This innovative approach, not yet widely applied in Indonesia, provides a critical tool for understanding local transmission dynamics, developing effective control strategies, and informing public health policy to meet the TB elimination target.

## B. METHODS

### 1. Study Procedure

This study utilizes a quantitative research design based on deterministic mathematical modeling to simulate epidemic scenario. This study will follow a systematic procedure, starting with developing a disease transmission flowchart to construct an SVEIR compartmental model. This model will be defined by a system of ordinary differential equations (ODEs). Subsequently, constant initial values for each compartment and parameters, such as infection and recovery rates, will be assigned using Indonesian epidemiological data and values from previous studies.

The system of ordinary differential equations (ODEs) was solved numerically using the fourth-order Runge-Kutta (RK4) method to ensure high accuracy and stability, which is critical for dynamic epidemiological compartmental models. For a system defined as  $\frac{dy}{dt} = f(t, y)$ , where  $y$  represents the vector of state variables and  $t$  is time, the RK4 method iteratively computes the solution at each time step  $h$  by combining four slope estimates:  $k_1$ , calculated at the beginning of the interval using the current state  $y_n$ ;  $k_2$ , evaluated at the midpoint using  $y_n$  adjusted by half of  $k_1$ ;  $k_3$ , recalculated at the midpoint with  $y_n$  updated by half of  $k_2$ ; and  $k_4$ , estimated at the interval's end using  $y_n$  fully adjusted by  $k_3$ . The updated state  $y_{n+1}$  is derived from a weighted average of these slopes. This iterative process produced estimates of tuberculosis incidence from 2023 to 2030 with an accuracy of  $\Delta \text{ dataset} \leq 1 \times 10^{-3}$ , which served as the convergence criterion for terminating the iterations (Lenhart & Workman, 2007). All equations for the RK4 are defined by the following equations from 1 to 5.

$$k_1 = hf(t_n, y_n) \tag{1}$$

$$k_2 = hf\left(t_n + \frac{h}{2}, y_n + \frac{k_1}{2}\right) \tag{2}$$

$$k_3 = hf\left(t_n + \frac{h}{2}, y_n + \frac{k_2}{2}\right) \tag{3}$$

$$k_4 = hf(t_n + h, y_n + k_3) \tag{4}$$

$$y_{n+1} = y_n + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4) \tag{5}$$

The dataset will be analyzed using descriptive statistics, including tabulation and visualization, to identify key trends (Reskiaddin et al., 2025). The estimated incidence will then be compared to Indonesia's 2030 TB elimination targets to evaluate progress and develop data-

driven recommendations. All analyses will be performed using R software, and the results from the SVEIR model will be summarized.

## 2. Assumptions and Model Formulation

TB pathogenesis begins when susceptible individuals ( $S$ ) inhale *Mycobacterium tuberculosis* (Mtb). Following exposure, some develop latent TB infection (LTBI), wherein the immune system controls bacterial replication, resulting in asymptomatic, exposed individuals ( $E$ ) who test positive on tuberculin or interferon-gamma release assays but exhibit normal chest X-rays and negative sputum tests (MoH Indonesia, 2020; Sasmita et al., 2024). These individuals remain at risk of progressing to active TB. The infection rate  $\alpha$  governs the transition from  $S$  to  $E$ , with the exposed compartment further stratified into three subcategories based on drug resistance:  $E_1$  as exposed individuals for drug-susceptible TB (DS-TB),  $E_2$  as exposed individuals for multidrug-resistant TB (MDR-TB), and  $E_3$  as exposed individuals for extensively drug-resistant TB (XDR-TB). The model also includes a vaccinated compartment ( $V$ ), comprising individuals with temporary immunity acquired through vaccination. This immunity reduces susceptibility to infection but wanes over time at a rate  $\xi$ , causing vaccinated individuals to re-enter the susceptible compartment. Susceptible individuals enter  $V$  upon vaccination at a rate  $\tau$ .

Individuals in any exposed compartment ( $E$ ) may progress to an active, infectious state. Infectious individuals are characterized by clinical symptoms (e.g., cough, fever, weight loss) and the ability to transmit Mtb to susceptible individuals through close contact, central to TB transmission (Ma et al., 2024). These individuals are classified into three compartments based on drug resistance profiles:  $I_1$  (drug-susceptible TB, DS-TB),  $I_2$  (multidrug-resistant TB, MDR-TB), and  $I_3$  (extensively drug-resistant TB, XDR-TB), with progression rates from exposed to infectious states denoted by parameters  $\beta_1$ ,  $\beta_2$  dan  $\beta_3$ .

DS-TB is treatable with first-line drugs, while MDR-TB resists at least isoniazid and rifampicin. XDR-TB, a more severe form of MDR-TB, shows additional resistance to fluoroquinolones and second-line injectable drugs such as bedaquiline or linezolid (Sikandar & Xing, 2025). Consequently, XDR-TB involves more complex treatment, higher mortality, and greater public health burden compared to MDR-TB or DS-TB. Treatments for XDR-TB are more toxic, costly, and less effective than those for MDR-TB or DS-TB. A key assumption of this model is that all susceptible individuals must pass through an exposed stage before becoming infectious.

The model incorporates two pathways to recovery: spontaneous resolution and/or treatment-mediated clearance for LTBI, and treatment-mediated recovery for active TB. Individuals entering the recovered compartment ( $R$ ) are considered fully healed. The first pathway involves spontaneous clearance or treatment-driven recovery from latent infection (compartments  $E_1$ ,  $E_2$ , and  $E_3$ ), represented by rates  $\rho_1$ ,  $\rho_2$ , and  $\rho_3$ . Standard TB Prevention Therapy (TPT) in Indonesia includes short-course regimens such as 3HP, 3HR, or longer 6H/9H courses. These treatments are available at public health facilities for eligible non-TB patients with normal diagnostic results (MoH Indonesia, 2020).

The second recovery pathway occurs through successful treatment, where infectious individuals ( $I_1$ ,  $I_2$ , and  $I_3$ ) achieve cure or treatment completion upon adherence to therapeutic

regimens. These transitions, quantified by parameters  $\delta_1$ ,  $\delta_2$ , and  $\delta_3$ , reflect positive clinical outcomes dependent on strict treatment adherence. Together with spontaneous or treatment-mediated clearance from latency, this dual-recovery mechanism highlights the combined role of natural immunity and effective healthcare interventions in reducing TB prevalence.

In Indonesia, drug-susceptible TB (DS-TB) is treated with a standardized 6-month regimen (2HRZE/4HR), comprising two months of isoniazid (H), rifampicin (R), pyrazinamide (Z), and ethambutol (E), followed by four months of H and R (Ministry of Health Indonesia, 2019, 2025). MDR-TB is managed using all-oral regimens: a shorter 9-month course including bedaquiline and a fluoroquinolone, or an 18–20-month individualized regimen for ineligible cases (Kementerian Kesehatan RI, 2023; WHO, 2020). XDR-TB and complex cases are treated with newer 6-month regimens such as BPaL or BPaLM or individualized regimens based on drug susceptibility testing (Ministry of Health Indonesia, 2023, 2024b). All treatments require rigorous clinical and laboratory monitoring for safety and efficacy, as stipulated in Indonesian national guidelines.

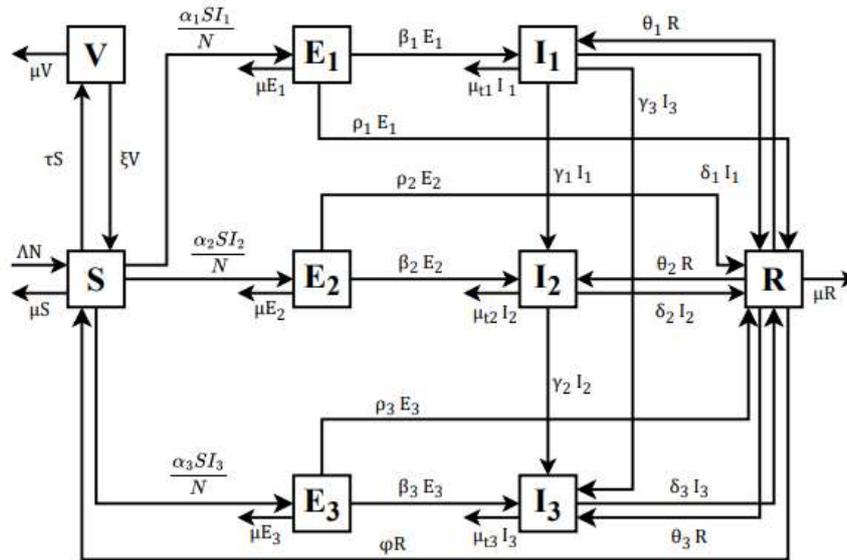
The model incorporates the progression of infectious individuals toward increasingly drug-resistant TB forms. Specifically, individuals with DS-TB may develop MDR-TB or XDR-TB, while those with MDR-TB may progress to XDR-TB. These transitions result primarily from inadequate treatment, such as misuse of second-line drugs or non-adherence, compounded by systemic challenges including limited diagnostics, treatment delays, and fragmented health systems (Chin et al., 2024). Parameters quantify progression rates:  $\gamma_1$  (DS-TB to MDR-TB),  $\gamma_3$  (DS-TB to XDR-TB), and  $\gamma_2$  (MDR-TB to XDR-TB).

The model explicitly incorporates tuberculosis relapse, a critical epidemiological feature reflecting the heightened risk of reinfection or reactivation among recovered individuals ( $R$ ). Unlike many infections, TB does not confer permanent immunity. Recovered individuals remain susceptible to reinfection or reactivation of latent infection, a key factor sustaining transmission in endemic settings. Two relapse mechanisms are defined: (1) loss of immunity (parameter  $\varphi$ ), where recovered individuals gradually revert to the susceptible compartment ( $S$ ) due to waning protection; and (2) direct reactivation (parameters  $\theta_1, \theta_2, \theta_3$ ), where individuals relapse directly into infectious compartments ( $I_1, I_2, I_3$ ) without returning to susceptibility, capturing scenarios such as latent infection reactivation or recurrence from incomplete treatment. This structure enhances the model's epidemiological realism by accounting for both reinfection and endogenous relapse pathways.

This epidemiological model, structured on an SVEIR framework, integrates a natural birth rate ( $A$ ) and two mortality components: a natural death rate ( $\mu$ ) affecting all compartments except active infectious cases, and a TB-specific death rate ( $\mu_t$ ) for individuals in infectious compartments before or during treatment. The population is stratified into nine compartments representing distinct epidemiological states: Susceptible ( $S$ ), Vaccinated ( $V$ ), Exposed ( $E_1, E_2, E_3$  for DS-TB, MDR-TB, and XDR-TB, respectively), Infectious ( $I_1, I_2, I_3$ ), and Recovered ( $R$ ). Each compartment corresponds to a disease progression stage, with its size at time  $t$  (denoted  $S(t), V(t)$ , etc.) governed by a system of differential equations.  $N_t$  gives the total population at time ( $t$ ). The model is formally defined by the following set of equations:

$$N(t) = S(t) + V(t) + E_1(t) + E_2(t) + E_3(t) + I_1(t) + I_2(t) + I_3(t) + R(t) \tag{6}$$

Based on the stated assumptions, this study developed a tuberculosis (TB) transmission flowchart, as Figure 1, to visualize the movement of individuals across epidemiological compartments. The model assumes that the rate of change in each compartment equals the net flow of individuals entering and exiting per unit time. This principle is formalized through the following system of differential equations:



**Figure 1.** TB transmission dynamics within an SV3E3IR compartmental framework.

$$\frac{dS}{dt} = \lambda N + \xi V + \varphi R - \frac{S}{N} \sum_{i=1}^3 \alpha_i I_i - \mu S - \tau S \tag{7}$$

$$\frac{dV}{dt} = \tau S - V(\xi + \mu) \tag{8}$$

$$\frac{dE_1}{dt} = \frac{\alpha_1 S I_1}{N} - E_1(\beta_1 + \rho_1 + \mu) \tag{9}$$

$$\frac{dE_2}{dt} = \frac{\alpha_2 S I_2}{N} - E_2(\beta_2 + \rho_2 + \mu) \tag{10}$$

$$\frac{dE_3}{dt} = \frac{\alpha_3 S I_3}{N} - E_3(\beta_3 + \rho_3 + \mu) \tag{11}$$

$$\frac{dI_1}{dt} = \beta_1 E_1 + \theta_1 R - I_1(\delta_1 + \gamma_1 + \gamma_3 + \mu_{t1}) \tag{12}$$

$$\frac{dI_2}{dt} = \beta_2 E_2 + \theta_2 R + \gamma_1 I_1 - I_2(\delta_2 + \gamma_2 + \mu_{t2}) \tag{13}$$

$$\frac{dI_3}{dt} = \beta_3 E_3 + \theta_3 R + \gamma_3 I_1 + \gamma_2 I_2 - I_3(\delta_3 - \mu_{t3}) \tag{14}$$

$$\frac{dR}{dt} = \sum_{i=1}^3 (\delta_i I_i + \rho_i E_i) - R(\varphi + \sum_{i=1}^3 \theta_i + \mu) \tag{15}$$

### 3. Initial Values of State Variables and Parameters

The initial values for state variables and parameters were obtained through a comprehensive literature review and national reports, prioritizing data specific to the Indonesian context. Initial compartment populations are provided in Table 1. To ensure the reliability of the simulation, state les and variabeparameters labeled as 'Data fitted' were estimated by calibrating the model against historical TB incidence data from national reports (2018–2024). Specifically, the number of DS-TB cases ( $I_1$ ) was calculated using WHO data as  $I_1 = I - I_2 - I_3$ , where  $I$  represents total infectious individuals. Similarly, the exposed compartments were disaggregated proportionally:  $E_1 = E \times (I_1 / I)$ ,  $E_2 = E \times (I_2 / I)$ , and  $E_3 = E \times (I_3 / I)$ . The susceptible population was estimated as  $S = N - (V + E + I + R)$ , where  $N$  denotes the total population.

**Table 1.** Initial values of state variables for TB disease transmission in Indonesia

No.	Symbols	Size*	References
1	$S$	156,288,179	Data fitted
2	$V$	3,069,255	(Ministry of Health Indonesia, 2024a)
3	$E$	120,000,000	(Lye Koh et al., 2019; WHO, 2024)
4	$E_1$	116,633,945	Data fitted
5	$E_2$	3,302,752	Data fitted
6	$E_3$	63,303	Data fitted
7	$I$	1,090,000	(WHO, 2024)
8	$I_1$	1,059,425	Data fitted
9	$I_2$	30,000	(WHO, 2024)
10	$I_3$	575	(WHO, 2024)
11	$R$	552,566	(Ministry of Health Indonesia, 2024a)
12	$N$	281,000,000	(WHO, 2024)

\*The unit of state variable is the number of individuals

Certain model parameters were estimated in the absence of direct empirical data. Based on the established epidemiological range indicating that a single infectious individual can infect 10–15 people per year (Moghaddam et al., 2016), strain-specific infection rates ( $\alpha_1, \alpha_2, \alpha_3$ ) were calculated as  $\alpha_1 = (I_1 \times 15) / S$ ,  $\alpha_2 = (I_2 \times 15) / S$ , and  $\alpha_3 = (I_3 \times 15) / S$ . These parameters, detailed in Table 2, are essential for simulating TB transmission dynamics and assessing the potential impact of public health interventions in Indonesia.

**Table 2.** Values of parameters for TB disease transmission in Indonesia

No.	Symbols	Value	References
1	$\lambda$	$1.659 \times 10^{-1}$	(BPS-Statistics Indonesia, 2020)
2	$\mu$	$5.920 \times 10^{-2}$	(BPS-Statistics Indonesia, 2020)
3	$\tau$	$1.9268 \times 10^{-2}$	(Widyaningsih et al., 2024)
4	$\xi$	$6.6667 \times 10^{-2}$	(Widyaningsih et al., 2024)
5	$\alpha_1$	$1.017 \times 10^{-1}$	Data fitted
6	$\alpha_2$	$2.9 \times 10^{-3}$	Data fitted
7	$\alpha_3$	$1.0 \times 10^{-4}$	Data fitted
8	$\beta_1$	$3.0 \times 10^{-3}$	(Ojo et al., 2023)
9	$\beta_2$	$3.0 \times 10^{-3}$	(Ojo et al., 2023)
10	$\beta_3$	$3.0 \times 10^{-3}$	(Ojo et al., 2023)
11	$\gamma_1$	$5.0 \times 10^{-2}$	(Dheda et al., 2017)

No.	Symbols	Value	References
12	$\gamma_2$	$1.0 \times 10^{-1}$	(Dheda et al, 2017)
13	$\gamma_3$	$5.0 \times 10^{-2}$	(Dheda et al, 2017)
14	$\delta_1$	$8.7 \times 10^{-1}$	(WHO, 2024)
15	$\delta_2$	$5.7 \times 10^{-1}$	(WHO, 2024)
16	$\delta_3$	$4.9 \times 10^{-1}$	(WHO, 2024)
17	$\theta_1$	$8.843 \times 10^{-3}$	(Widyaningsih et al., 2024)
18	$\theta_2$	$8.843 \times 10^{-3}$	(Widyaningsih et al., 2024)
19	$\theta_3$	$8.843 \times 10^{-3}$	(Widyaningsih et al., 2024)
20	$\rho_1$	$7.9 \times 10^{-1}$	(Widyaningsih et al., 2024)
21	$\rho_2$	$7.9 \times 10^{-1}$	(Widyaningsih et al., 2024)
22	$\rho_3$	$7.9 \times 10^{-1}$	(Widyaningsih et al., 2024)
23	$\varphi$	$9.65870 \times 10^{-1}$	Data fitted
24	$\mu_{t1}$	$2.0 \times 10^{-1}$	(Dheda et al, 2017; Soodejani et al., 2024)
25	$\mu_{t2}$	$4.0 \times 10^{-1}$	(Dheda et al, 2017; Soodejani et al., 2024)
26	$\mu_{t3}$	$6.0 \times 10^{-1}$	(Dheda et al, 2017; Soodejani et al., 2024)

### C. RESULT AND DISCUSSION

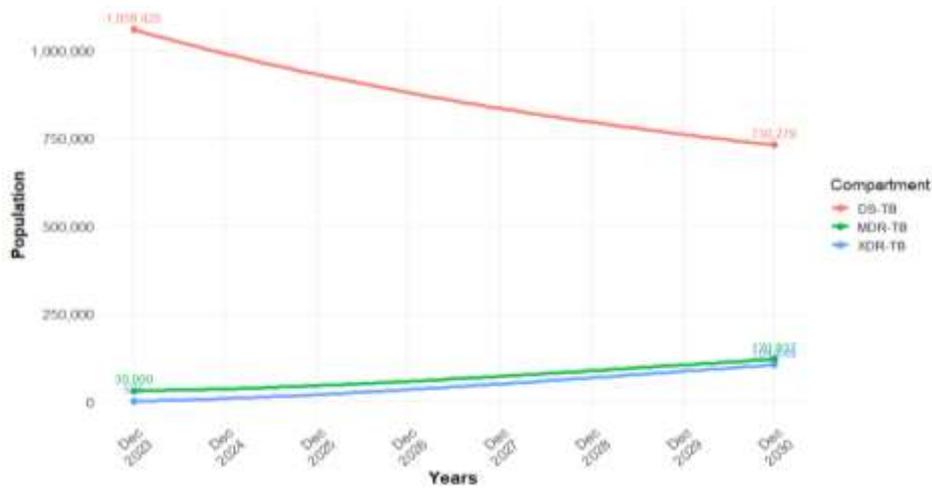
The ODE system was solved numerically using the RK4 method to simulate compartmental dynamics over time. Iterations continued until convergence was achieved, defined as a maximum compartmental change below the termination threshold of  $1 \times 10^{-3}$ . The simulation reached high numerical stability, with a final  $\Delta$  value of  $1.29 \times 10^{-4}$ . Using this deterministic SVE3I3R model, compartment sizes were projected from December 2023 to December 2030 within the Indonesian population. Results are presented in Table 3 and Figures 2, providing epidemiological insights into the future trajectory of TB burden under current conditions. Table 3 presents the core output of the deterministic SVE3I3R model simulation run from December 2023 to December 2030. Its purpose is to quantify the projected epidemiological trajectory of tuberculosis in Indonesia under current intervention strategies. The table moves beyond point estimates by providing a distribution of values for each compartment, offering a robust understanding of the range, central tendency, and variability of the model's predictions. This is critical for assessing the certainty of the projections and the potential burden on the healthcare system.

**Table 3.** Summary statistics for each compartment (in people)

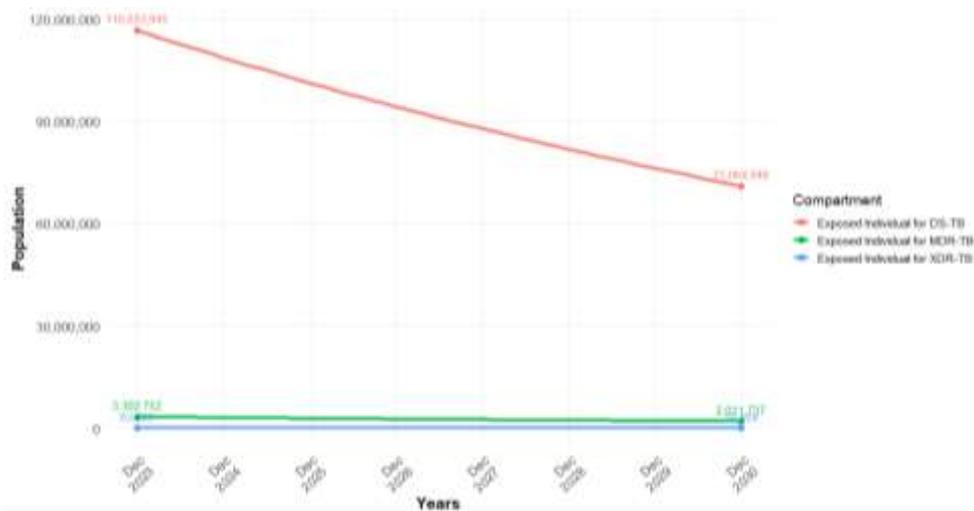
Symbol	Min	Mean	Max	SD	IQR
$S$	156,288,179	167,626,665	181,710,968	7,602,514	13,060,732
$V$	3,069,255	6,128,971	9,306,950	1,830,123	3,109,348
$E_1$	71,064,048	91,996,915	116,633,945	13,364,902	22,611,271
$E_2$	116,633,945	2,604,747	3,302,752	378,633	640,582
$E_3$	38,559	49,925	63,303	7,257	12,278
$I_1$	730,279	869,775	1,059,425	95,664	158,771
$I_2$	30,000	67,851	120,937	28,137	49,396
$I_3$	575	45,132	104,649	31,943	55,901
$R$	552,566	19,988,450	32,828,176	9,447,841	15,605,884

Min is the minimum value; mean is the average value; max is the maximum value; SD is the standard deviation value; IQR is the interquartile range value. The projected outcomes in Table 3 are a direct computational result of the novel SVE3I3R model's structure and initial

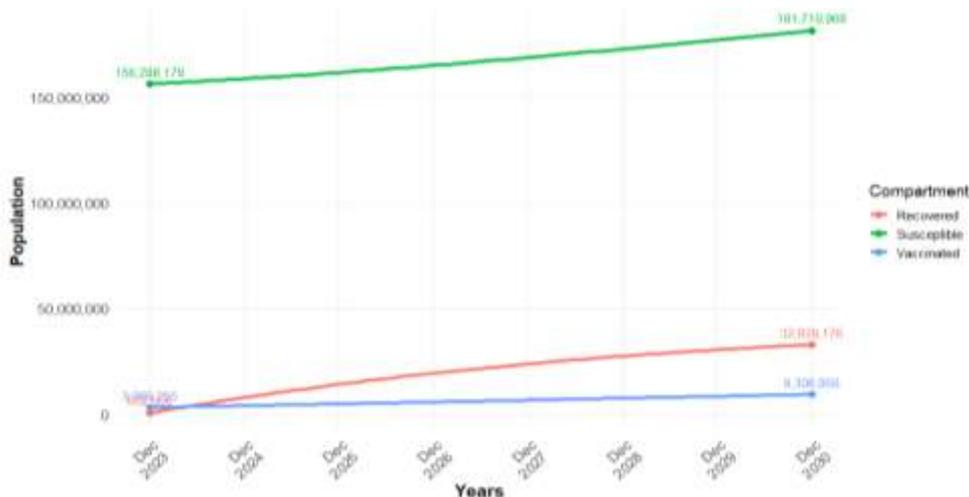
parameters, validating the epidemiological concerns raised in the study's background. The model forecasts a persistently high burden of exposed individuals for DS-TB ( $E_1$ ), with approximately 116 million individuals, stemming from the initial large susceptible individual ( $S$ ) and the high infection rate. This vast reservoir underscores the challenge of Indonesia's LTBI epidemic. While a decline in active DS-TB cases ( $I_1$ ) is observed, attributable to the high recovery rate ( $\delta_1 = 8.7 \times 10^{-1}$ ) integrated into the model, this progress is dramatically offset by an alarming surge in drug-resistant forms. The model projects a fourfold increase in MDR-TB ( $I_2$ ) and a 182-fold increase in XDR-TB ( $I_3$ ), a dire consequence directly driven by the progression parameters ( $\gamma_1, \gamma_2, \gamma_3$ ) that mathematically represent the development of resistance due to inadequate treatment and systemic healthcare failures. Furthermore, the model predicts a significant expansion of the recovered compartment ( $R$ ), which, due to the incorporated relapse mechanisms (parameters  $\theta_1$  and  $\varphi$ ), paradoxically represents a large population at risk of reinfection or reactivation, thereby perpetuating the transmission cycle and hindering elimination efforts.



(a)



(b)



(c)

**Figure 2.** (a) trend of infectious individuals, (b) trend of exposed individuals, (c) trend of susceptible, vaccinated, and recovered individuals from 2023 to 2030

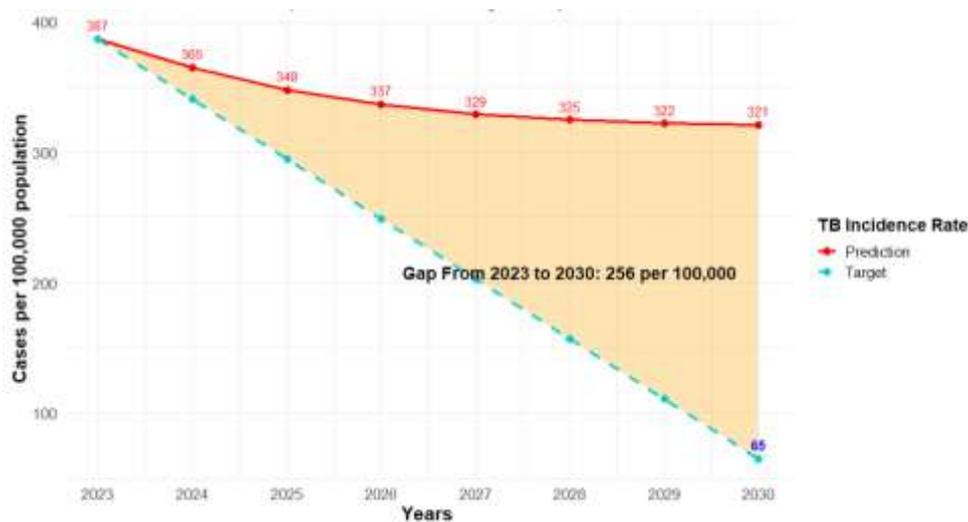
Table 4 and Figure 3 are the pivotal outputs of the deterministic SVE3I3R model, presenting the core epidemiological forecast for Indonesia's TB burden through to 2030. Their purpose is to quantify the projected number of infectious individuals and the TB incidence rate (per 100,000 population) under the current trajectory of intervention strategies. This data is the primary evidence used to evaluate the feasibility of achieving the national elimination target of Presidential Regulation No. 67 of 2021.

The data presented in Table 4 and visualized in Figure 3 reveal a story of stalled progress and a looming public health crisis. The model's projection shows a TB incidence rate that declines marginally from 387 per 100,000 in 2023 to 321 per 100,000 in 2030. This trajectory is characterized by a rapid diminishment in the rate of improvement; the most significant drop occurs between 2023 and 2024 (22 cases per 100.000), after which the annual declines become increasingly shallow. This stagnation indicates that the current suite of interventions has reached the limits of its effectiveness and cannot induce the exponential decline required for elimination. The apparent improvement in absolute case numbers is also affected by population growth, resulting in a persistently high disease burden that continues to fuel community transmission.

**Table 4.** Prediction of TB incidence rate based on the SVE3I3R Model

Years	Total Population	Total Infectious Individuals	Proportion in 100.000
2023	281,000,000	1,090,000	387
2024	283,357,176	1,034,191	365
2025	285,734,347	995,591	348
2026	288,131,326	970,717	337
2027	290,548,016	956,611	329
2028	292,984,382	950,776	325
2029	295,440,453	951,112	322
2030	297,916,303	955,866	321

When this projected trajectory is juxtaposed with the linear path required to meet the national target of 65 per 100,000, the analysis reveals a profound and growing strategic divergence. The target line represents an ambitious. In stark contrast, the model's output shows a pathway that begins to diverge almost immediately and evolves into a chasm by 2030. The projected incidence for 2030 is not merely above the target; it is nearly five times higher, a clear quantitative verdict on the inadequacy of the status quo. This widening gap is not an abstraction but a measure of the future morbidity, mortality, and economic cost incurred if policies remain unchanged. It underscores a critical failure to translate a high-level political commitment into an operational strategy powerful enough to alter the fundamental dynamics of TB transmission and progression within the population.



**Figure 3.** Prediction versus target on TB incidence rate in Indonesia from 2023 to 2030

The overall interpretation of these results is that Indonesia's current TB control program, while making strides in case detection and treatment for DS-TB, is structurally insufficient to alter the fundamental dynamics required for elimination. Our findings align with the concerns raised by global reports, such as those from the WHO, which highlight the escalating threat of drug-resistant TB in high-burden countries (WHO, 2024). However, they stand in contrast to more optimistic linear projections that may not fully account for the non-linear complexities of TB transmission, such as relapse and the development of resistance. The divergence can be attributed to our model's explicit incorporation of these critical pathways, specifically, the transitions between drug resistance classes ( $\gamma$  parameters) and the relapse mechanisms ( $\theta$  and  $\varphi$  parameters). While previous studies in other regions using SVEIR or SEIR frameworks have emphasized treatment rate improvements (Dicko et al., 2024; Hajji & Albargi, 2022), our results suggest that in the Indonesian context, these gains are being systematically undermined by the twin crises of drug resistance and reactivation, a nuance that simpler models may not capture.

The generalizability of these findings must be considered cautiously. The model is particular to the Indonesian epidemiological context, calibrated with local data on population demographics, initial compartment sizes, and parameter estimates. Therefore, the precise numerical projections are not directly transferable to other countries. However, the model's structure and the identified dynamics possess strong theoretical generalizability. The

mechanisms driving the results, specifically, how high relapse rates in a large recovered population and inadequate treatment leading to explosive drug-resistant TB growth, are likely applicable to other high-burden, resource-limited settings with similar healthcare system challenges and large LTBI pools. The SVE3I3R framework can serve as a template for analogous studies in other countries aiming to evaluate their elimination targets against complex, realistic transmission dynamics.

The primary theoretical implication of this work is the advancement of deterministic compartmental modeling for TB. By stratifying the exposed and infectious compartments by drug-resistance profile ( $E_1, E_2, E_3$ , and  $I_1, I_2, I_3$ ) and explicitly modeling transitions between them ( $\gamma$  parameters), our SVE3I3R model provides a more granular and realistic representation of the TB epidemic than traditional SEIR or SVEIR models. Furthermore, explicitly incorporating a dual relapse mechanism, both a return to susceptibility ( $\varphi$ ) and direct reactivation to infectious states ( $\theta$ ), adds significant epidemiological fidelity. This model contributes to the theoretical understanding that achieving TB elimination requires frameworks that move beyond tracking overall incidence and instead capture the internal shifts within the epidemic, particularly the escalating threat of drug resistance.

This study has several critical implications for future research. First, it underscores the necessity of moving from aggregate TB models to those that disaggregate by resistance strain to forecast future burdens accurately. Subsequent research should focus on refining the parameter estimates for resistance development and relapse through longitudinal cohort studies within Indonesia. Second, the model provides a platform for in-silico testing of intervention strategies. Future work should employ this SVE3I3R model to conduct cost-effectiveness analyses of various scenarios, such as the impact of scaling up universal drug susceptibility testing (DST), enhancing infection control, or expanding TB Preventive Therapy (TPT) to different target groups. Finally, there is a need to integrate spatial heterogeneity or agent-based approaches to identify subnational hotspots and effectively tailor interventions.

For public health practice, the implications are urgent and clear. A "business-as-usual" approach focused primarily on diagnosing and treating DS-TB will fail. Practice must pivot to a dual-strategy: aggressively containing drug-resistant TB while simultaneously draining the latent reservoir (Chaw et al., 2020). This necessitates a massive scale-up of laboratory capacity to ensure universal DST for all diagnosed cases, enabling prompt initiation of appropriate regimens. Concurrently, a nationwide expansion of TPT, targeting high-risk groups and the vast latent pool, is imperative to prevent reactivation. Infection control measures in healthcare settings and congregate living environments must be strengthened to break transmission chains, especially drug-resistant strains (Marme et al., 2023; Vigneschow et al., 2021).

At the policy level, these findings serve as a critical evidence base for a fundamental overhaul of Indonesia's TB control strategy, as mandated by Presidential Regulation No. 67 of 2021. Policymakers must recognize that the current trajectory will not meet the 2030 target. Policy implications include: (1) **Reallocating Resources:** Directing significant funding towards strengthening the laboratory network for DST and securing sustainable access to shorter, more effective regimens for MDR-TB/XDR-TB. (2) **Programmatic Expansion:** Formulating and funding a comprehensive, large-scale TPT rollout policy integrated into primary healthcare. (3) **Regulatory Action:** Enforcing stronger regulations for infection control and mandating rapid

molecular diagnostics as the initial test for all TB suspects. This evidence demands that the ambitious political commitment be matched with an equally ambitious and evidence-informed operational plan.

This study has several limitations. First, as a deterministic model, it does not account for stochastic events, which can influence outbreak dynamics, especially in low-prevalence settings nearing elimination. Second, the model assumes homogeneous mixing within the population, which may oversimplify complex contact networks and spatial heterogeneity in transmission risk across Indonesia's diverse archipelago. Third, several parameters, for instance, for relapse ( $\theta$ ) and resistance development ( $\gamma$ ), were estimated from international literature or fitted due to a lack of robust local data; this could introduce bias. The direction of this potential bias is likely towards underestimating the complexity of the epidemic, meaning the actual situation may be more challenging than projected. Finally, the model does not incorporate potential future advancements in vaccines or therapeutics, which could alter the trajectory if successfully deployed before 2030.

#### **D. CONCLUSION AND SUGGESTIONS**

This study's findings show that Indonesia will not achieve the 2030 elimination target of 65 cases per 100,000 population. Instead, the incidence rate is forecasted to reach 321 per 100,000 by 2030, nearly five times the national goal. The surge in Multidrug-Resistant (MDR) and Extensively Drug-Resistant (XDR) cases, projected to exceed 100,000 cases respectively, highlights the critical failure of current control strategies to contain resistant strains. Theoretically, this research contributes to the field by demonstrating that incorporating detailed resistance stratification (SVE3I3R) significantly alters projection outcomes compared to traditional aggregate models. Practical implications suggest that without an urgent shift to universal drug susceptibility testing and aggressive preventive therapy, the epidemic will persist.

The findings consistently address the study's primary objective where to project TB transmission and assess the 2030 target. The results unequivocally demonstrate that without an immediate and paradigm-shifting response that prioritizes the containment of drug-resistant TB and the prevention of reactivation, the goal of TB elimination in Indonesia will remain out of reach. This research delivers a clear and urgent call to action for policymakers and public health practitioners. Future research should employ stochastic modeling to account for random variation in low-incidence scenarios and incorporate spatial heterogeneity to refine these projections further.

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