Mathematical Modelling of Tuberculosis with Vaccination and Saturated Incidence Rate in Indonesia

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Abstract. This study is motivated by the high incidence of tuberculosis (TB) in Indonesia. Various preventive intervention strategies have been developed to reduce the spread of TB; however, the disease continues to contribute to a high mortality rate. Therefore, this study aims to develop a deterministic mathematical model to analyze TB transmission dynamics by incorporating vaccine efficacy and vaccination rates. The proposed model involves infectious and latent (non-infectious) populations. The latent population represents individuals infected with *Mycobacterium tuberculosis* who do not exhibit symptoms and are not yet infectious, but have the potential to progress to active TB when the immune system weakens. Thus, the latent population acts as a reservoir of infection that contributes to the emergence of new TB cases. The model is analyzed through the determination of equilibrium points and stability analysis, followed by sensitivity analysis and numerical simulations to examine the impact of variations in vaccine efficacy and vaccination rates on TB transmission dynamics involving latent and infectious populations. The results show that increasing vaccine efficacy significantly reduces the latent population, which subsequently decreases the number of infectious individuals. In addition, higher vaccine efficacy reduces the controlled reproduction number influenced by latent and infectious individuals, thereby limiting disease transmission. Sensitivity analysis also indicates that vaccine-related parameters have a strong influence on transmission dynamics. In conclusion, improving vaccine efficacy and increasing vaccination rates are key factors in controlling and eliminating tuberculosis in the population.
Keywords: Tuberculosis, Mathematical modeling, Vaccine efficacy, Vaccine rate, Latent infection.

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1. Introduction

Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis and primarily affects the lungs, although it can also infect other organs such as the kidneys, bones, and brain (WHO, 2023). This disease remains a major challenge in global public health. In 2023, TB ranked as the second leading cause of death from infectious diseases worldwide after COVID-19. Globally, TB continues to exhibit a high incidence rate with an uneven distribution across countries. In 2023, an estimated 10.8 million TB cases were reported, with approximately 1.1 million deaths. Ten countries accounted for about two-thirds of the global TB burden, namely India (26%), Indonesia (10%), China (7%), Philippines (7%), Pakistan (6%), Nigeria (5%), Bangladesh (3%), Democratic Republic of the Congo (3%), Myanmar (3%), and South Africa (2%). Among these countries, Indonesia ranks second after India, indicating a very high TB burden (Ministry of Health of the Republic of Indonesia, 2023). Tuberculosis remains a serious public health issue in Indonesia. According to the 2023 report from the Ministry of Health, TB prevention efforts include vaccination, screening of high-risk individuals, early detection, and treatment. However, TB case notifications have increased in recent years.

Reported cases rose from 393,323 in 2020 to 821,200 in 2023, and are estimated to reach approximately 889,133 cases in 2024, indicating that TB control efforts are not yet optimal.

Tuberculosis remains a major public health challenge in Indonesia. According to the 2023 report from the Ministry of Health of the Republic of Indonesia, TB prevention efforts include vaccination, screening of high-risk individuals, early detection, and treatment. However, TB case notifications have increased since the COVID-19 pandemic. In 2020, there were 393,323 reported cases, increasing to 443,235 in 2021 and 724,309 in 2022. The 2023 report recorded 821,200 cases, and the 2024 estimate suggests that the number may reach approximately 889,133 cases.

One of the main interventions in TB control is vaccination. The effectiveness of vaccination programs is strongly influenced by vaccine efficacy and the vaccination rate within the population. Vaccine efficacy measures how effectively a vaccine prevents disease, while the vaccination rate determines how quickly protection spreads throughout the population. Without efficacy data, it is difficult to estimate the reduction in infection risk or progression to active disease, leading to potentially inaccurate epidemiological models. Conversely, without considering the vaccination rate, even a highly efficacious vaccine may fail to significantly reduce disease transmission if its distribution is slow or coverage remains low (Hawn, 2014).

The Bacillus Calmette–Guérin (BCG) vaccine is a cornerstone of the national immunization program in Indonesia and has been administered to infants since birth. The increase in TB cases among children has been associated with limitations in BCG protection, highlighting the importance of monitoring vaccine coverage and efficacy (Nataprawira et al., 2022). Although the BCG vaccine has proven effective in preventing severe forms of TB, particularly in infants and children, it has not been able to eliminate TB entirely. One of its main limitations is that it does not fully prevent TB infection. In many cases, infection does not immediately progress to active disease but instead remains in the form of latent tuberculosis infection. Latent TB is a condition in which individuals are infected with TB bacteria but do not exhibit symptoms and are not infectious. However, individuals with latent TB have the potential to develop active TB when their immune system weakens.

On the other hand, the effectiveness of vaccination programs is not solely determined by vaccine availability, but also by vaccine efficacy and vaccination rate. Therefore, research on vaccine efficacy and vaccination rate is highly necessary in Indonesia, given the high TB burden and the suboptimal performance of current control programs. These parameters play a crucial role in determining TB transmission dynamics and the effectiveness of control strategies. In the context of mathematical modeling, vaccination is an important research topic because vaccine efficacy and vaccination rate are not constant parameters. They are influenced by epidemiological, social, and policy factors. Therefore, a mathematical approach is required to evaluate how variations in vaccination parameters affect TB transmission dynamics and to determine optimal conditions for effective disease control.

In TB transmission models, the incidence function plays an important role in determining the transmission rate. Previous studies have commonly used the bilinear incidence function βSI, as applied by Side et al. (2016), Koriko and Yusuf (2008), Faruk (2016), and Bahari et al. (2023), where the infection rate is assumed to be proportional to the interaction between susceptible and infected individuals. According to Keeling and Rohani (2008), the bilinear function is more suitable for small-scale populations where contact rates increase with population density, such as in rapidly spreading diseases like influenza. As an alternative, the saturated incidence rate function $\beta S(t)I(t)/(1 + bI(t)$ bI(t)) is used. In this formulation, β represents the transmission rate when the disease enters a fully susceptible population, while the term $1/(1 + bI(t)$ captures the inhibitory effect due to behavioral changes or limited effective contact as the number of infected individuals increases (Mengistu et al.,

2020). This function is considered more realistic than the bilinear form because it accounts for limitations in effective contact and changes in population behavior.

Based on the above considerations, this study aims to develop and analyze a mathematical model of TB transmission incorporating vaccination and a saturated incidence rate. The analysis focuses on examining the effects of vaccine efficacy and vaccination rate on the dynamics of latent and actively infected populations. The results are expected to provide quantitative insights and strategic recommendations for TB control.

2. Method

This study began by developing a model for the spread of tuberculosis (TB), which continues to show a high number of cases from year to year. The population was divided into five groups, and the dynamics of each group were expressed as a system of nonlinear autonomous differential equations.

To control the spread of TB, two strategies were incorporated into the model: vaccine efficacy and vaccination rate. Next, disease-free and endemic equilibrium points were determined to analyze the likelihood of disease spread. Because the resulting model is nonlinear and difficult to solve analytically, an approximation method was used to linearize the model around the equilibrium points using the Jacobian matrix. Based on this Jacobian matrix, the local stability of each equilibrium point was analyzed using the Routh-Hurwitz stability criterion.

The basic reproduction number was obtained using the next-generation matrix method and used as an indicator of the potential spread of TB in the community. Next, a sensitivity analysis was conducted to determine the positive and negative effects of model parameters on the dynamics of TB spread and to identify the parameters with the most dominant influence. The analysis results were confirmed through simulations based on several population data in Indonesia.

3. Results and Discussion

3.1 Construction of the Model

Tables and figures are presented systematically to support data clarity and discussion, with numbering using Arabic numerals according to their order of appearance in the text. Table titles are placed above the tables, while figure captions are placed below the figures, both written concisely, clearly, and informatively. Tables are arranged in a simple format using horizontal lines only, without vertical lines, and include units in the column headings as well as notes, when necessary, whereas figures must be of clear and readable quality. Each table and figure must be cited and explained in the text, must not be split across pages, and the language used must be consistent with the language of the scientific manuscript.

To formulate a tuberculosis (TB) transmission model, the total population N(t) is devided into five compartments: susceptible (S), vaccinated (V), latent (L), infectious (I), and recovered (R). The development of the model is based on several following assumptions: Individuals in the susceptible (S) class are those who have not been infected with TB but remain at risk of contracting the disease. The vaccinated (V) class represents individuals who have received the Bacillus Calmette–Guérin (BCG) vaccine. Those in the latent (L) class are infected but do not transmit the disease, whereas individuals in the infectious (I) class are actively infected and capable of spreading TB. The recovered (R) class consists of individuals who have successfully completed treatment.

The susceptible population increases through recruitment at a rate Λ. In line with previous studies (Sahu & Dhar, 2012; Baba et al., 2020; Kar & Jana, 2013), the transmission process is described

using a saturated incidence function of the form $\frac{\beta I}{1+bI}$, where β denotes the effective contact rate and b represents the saturation effect. Vaccination is administered to susceptible individuals at a rate φ, with $0 \leq \varphi \leq 1$. Since the vaccine does not confer complete immunity, vaccinated individuals may still acquire infection, although at a reduced rate determined by the efficacy parameter ε. Consequently, the infection rate among vaccinated individuals is scaled by $(1-\varepsilon)$.

Treatment is provided to individuals in both latent and infectious stages. The rate at which latent individuals progress to the infectious stage is given by k, while treatment rates for latent and infectious individuals are denoted by α and r, respectively. Among those receiving treatment in the infectious class, a proportion p recovers and moves to the recovered class, whereas the remaining proportion $(1-p)$ returns to the latent class.

Recovered individuals are not assumed to be completely free of infection, as relapse into the latent stage may occur. The natural death rate μ is assumed to be uniform across all compartments, while disease-induced mortality occurs only in the infectious class at a rate d. All parameters involved in the model are assumed to be non-negative.

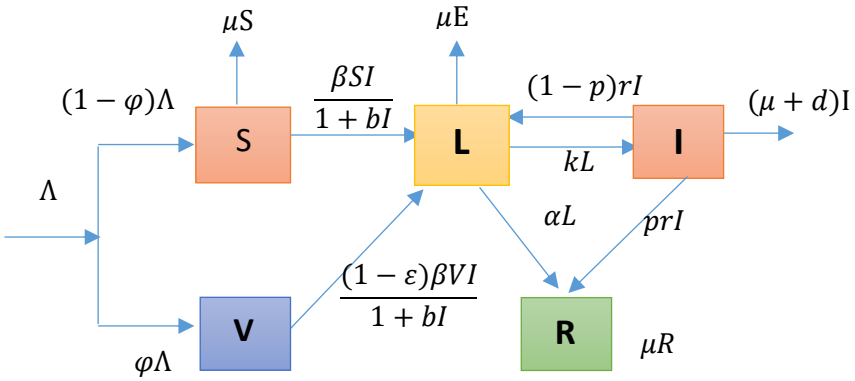

Figure 1. Flow chart model for pulmonary tuberculosis disease.

Based on the above assumptions, we describe the dynamics of TB with the following system of ordinary differential equations (ODEs) with four compartments.

$$\begin{aligned}
\frac{dS(t)}{dt} &= (1-\varphi)\Lambda - \frac{\beta S(t)I(t)}{1+bI(t)} - \mu S(t),\\
\frac{dV(t)}{dt} &= \varphi\Lambda - \frac{(1-\varepsilon)\beta V(t)I(t)}{1+bI(t)} - \mu V(t),\\
\frac{dL(t)}{dt} &= \frac{\beta S(t)I(t)}{1+bI(t)} + \frac{(1-\varepsilon)\beta V(t)I(t)}{1+bI(t)} + (1-p)rI(t) - \alpha L(t) - kL(t) - \mu L(t),\\
\frac{dI(t)}{dt} &= kL(t) - (1-p)rI(t) - prI(t) - (\mu+d)I(t),\\
\frac{dR(t)}{dt} &= \alpha L(t) + prI(t) - \mu R(t).
\end{aligned} \tag{1}$$

With the initial conditions illustrated by

$$S(0) \geq 0, V(0) \geq 0, L(0) \geq 0, I(0) \geq 0, R(0) \geq 0.$$

The complete transfer flow of the model parameters is shown in Figure 1. The time unit "t" has been considered in "years".

3.2. Basic Properties of the Model

3.2.1. Positivity of Solutions

Since model system (1) involves a human population, it is necessary to prove that all variables and their associated parameters are nonnegative, so we have the following theorem.

Lemma 1. Suppose the initial values S_0, V_0, L_0, I_0,and R_0 are nonnegative. Then, the solution set $\Omega = \{S(t), V(t), L(t), I(t), R(t)\}$ is nonnegative for all $t \geq 0$.

Proof. If we let $\frac{\beta I(t)}{1+bI} - \mu = K(t)$, then it follows from the first equation of the model (1) that

$$\frac{dS(t)}{dt} + K(t)S(t) = (1-\varphi)\Lambda N. \tag{2}$$

Multiplying both sides of (2) by M(t)

$$M(t)\frac{dS(t)}{dt} + M(t)K(t)S(t) = (1-\varphi)\Lambda N\, M(t). \tag{3}$$

If $M(t)K(t) = M'(t)$,
so

$$\frac{d}{dt}\big(S(t)M(t)\big) = (1-\varphi)\Lambda N\, M(t). \tag{4}$$

Because if $\mathrm{M(t)K(t) = M'(t)}$ then

$$\frac{M'(t)}{M(t)} = K(t),$$

so that

$$M(t) = \exp\left(\int_0^t K(\tau)d\tau\right).$$

Substitute M(t) in equation (4)

$$\frac{d}{dt}\left[S(t)\exp\left(\int_0^t K(\tau)d\tau\right)\right] = (1-\varphi)\Lambda N\ \exp\left(\int_0^t K(\tau)d\tau\right). \tag{5}$$

Integrating both sides of equation (5) from 0 to t, and using the initial condition $S(0) = S_0$

$$S(t)\exp\left(\int_0^t K(\tau)d\tau\right) - S_0 = (1-\varphi)\Lambda\, N\int_0^t \left(\exp\left(\int_0^\tau K(u)du\right)d\tau\right),$$

thus

$$S(t) = S_0\exp\left\{-\int_0^t K(\tau)d\tau\right\} + (1-\varphi)\Lambda N\ \left(\int_0^t \exp\left(\int_0^\tau K(u)du\right)d\tau\right)\exp\left\{-\int_0^t K(\tau)d\tau\right\} \geq 0. \tag{6}$$

Similarly, it can be shown that $V(t), L(t), I(t)$, and $R(t)$ are nonnegative for all time $t \geq 0$. his completes the proof.

3.2.2 Invariant region

The dynamical system (1) describes the evolution of the human population over time. The invariant region ensures that the solutions of the model remain nonnegative and bounded for all time. Therefore, the TB model (1) is studied in a biologically feasible region defined as follows:

$$\Omega = \left\{(S(t), V(t), L(t), I(t), R(t)) \in {R_+}^5 \middle| 0 \leq S(t) + V(t) + L(t) + I(t) + R(t) \leq \frac{\Lambda}{\mu}\right\}. \tag{7}$$

Lemma 2. The region $\Omega \subset {R_+}^5$ is positively invariant for model (1) with nonnegative initial conditions in ${R_+}^5$.

Proof. For this model, the total population is given by

$$N(t) = S(t) + V(t) + L(t) + I(t) + R(t), \tag{8}$$

then

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dV(t)}{dt} + \frac{dL(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt}. \tag{9}$$

Substituting the values from the model (1), we get

$$\frac{dN(t)}{dt} = \Lambda - \mu(S(t) + V(t) + L(t) + I(t) + R(t)) - dI(t)$$

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dI(t). \quad (10)$$

Because $dI(t) \geq 0$ then

$$-dI(t) \leq 0,$$

therefore

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dI(t) \leq \Lambda - \mu N(t). \quad (11)$$

Thus, integrating both sides and taking as $t \to \infty$, we get

$$\frac{dN(t)}{dt} + \mu N(t) = \Lambda\,. \quad (12)$$

Using the integrating factor $e^{\mu t}$, multiply both sides of equation (12) by this factor to obtain

$$e^{\mu t}\frac{dN(t)}{dt} + e^{\mu t}\mu N(t) = \Lambda e^{\mu t}.$$

This can be rewritten as

$$\frac{dN(t)e^{\mu t}}{dt} = \Lambda e^{\mu t}. \quad (13)$$

Next, integrate equation (13) to obtain N(t)

$$0 \leq N(t) \leq \frac{\Lambda}{\mu}.$$

Therefore, the feasible solution set for the model is given by

$$\Omega = \left\{(S(t), V(t), L(t), I(t), R(t)) \in {R_+}^5 \middle| 0 \leq S(t) + V(t) + L(t) + I(t) + R(t) \leq \frac{\Lambda}{\mu}\right\}. \quad (14)$$

3.3. Equilibria

The long-term behavior of a model is crucial for analysis. When left undisturbed for a long time without external disturbances, it is important to determine whether the system will reach a stable state. Eventually, a stable state will be reached by the system. If the disease consistently disappears from the population, the system is said to be converging toward a disease-free equilibrium. Conversely, there is a possibility that the disease persists in the population, where the size of each population class tends to converge toward a stable point called the endemic equilibrium point.

3.3.1 Disease-Free and Endemic Equilibria

It is easy to verify that model (1) always has a disease-free equilibrium (DFE). To obtain a disease-free equilibrium point, equation (1) must satisfy

$$\frac{dS(t)}{dt} = \frac{dV(t)}{dt} = \frac{dL(t)}{dt} = \frac{dI(t)}{dt} = \frac{dR(t)}{dt} = 0.$$

So the following equation is obtained

$$(1-\varphi)\Lambda - \frac{\beta S(t)I(t)}{1+bI} - \mu S(t) = 0, \quad (15)$$

$$\varphi\Lambda - \frac{(1-\varepsilon)\beta V(t)I(t)}{1+bI(t)} - \mu V(t) = 0, \quad (16)$$

$$\frac{\beta S(t)I(t)}{1+bI(t)} + \frac{(1-\varepsilon)\beta V(t)I(t)}{1+bI(t)} + (1-p)rI(t) - \alpha L(t) - kL(t) - \mu L(t) = 0, \quad (17)$$

$$kL(t) - (1-p)rI(t) - prI(t) - (\mu + d)I(t) = 0, \quad (18)$$

$$\alpha L(t) + prI(t) - \mu R(t) = 0. \tag{19}$$

The non-endemic equilibrium point is a condition where there is no spread of disease in the population, so that $I(t) = 0$ equations (15) to (19) become

$(1-\varphi)\Lambda - \mu S(t) = 0,$
$\varphi\Lambda - \mu V(t) = 0,$
$-\alpha L(t) - kL(t) - \mu L(t) = 0,$
$kL(t) = 0,$
$\alpha L(t) - \mu R(t) = 0.$

Because $I(t) = 0$, then from equations (18) and (19) we obtain $L(t) = 0$ and $R(t) = 0$.

By setting I =0, the system admits a disease-free equilibrium given by

$$E_0 = (S_0, V_0, L_0, I_0, R_0) = \left(\frac{(1-\varphi)\Lambda}{\mu}, \frac{\varphi\Lambda}{\mu}, 0,0,0\right), \tag{20}$$

and the endemic equilibrium point is

$$E^* = (S^*, V^*, L^*, I^*, R^*),$$

with

$$S^* = \frac{(1-\varphi)\Lambda(1+bI^*)}{\beta I^* + \mu(1+bI^*)},$$
$$V^* = \frac{\varphi\Lambda(1+bI^*)}{(1-\varepsilon)\beta I^* + \mu(1+bI^*)},$$
$$L^* = \frac{(r+\mu+d)}{k} I^*,$$
$$R^* = \frac{prI^* + \alpha L^*}{\mu} = \frac{prI^* + \alpha\left(\frac{(r+\mu+d)}{k} I^*\right)}{\mu},$$
$$I^* = \frac{\frac{\beta\Lambda}{\mu}(1-\varepsilon\varphi) - \left[\frac{(\alpha+k+\mu)(r+\mu+d)}{k} - (1-p)r\right]}{b\left[\frac{(\alpha+k+\mu)(r+\mu+d)}{k} - (1-p)r\right]}, \tag{21}$$

3.3.2 Basic Reproduction Number

Next, we introduce the basic reproductive number $\mathcal{R}_0$, defined as the average number of new TB cases produced by a single infected individual during its infectious period, when that individual interacts with a fully susceptible population. The basic reproductive number plays a crucial role in determining the global dynamics of disease in epidemiological studies. It helps us determine whether a disease will spread and persist or whether it will eventually disappear from the population. If the reproductive number is less than one, it indicates that the disease is not spreading in the community. If the value is greater than one, the disease is likely to infect the community, that is, it is endemic and will cause deaths and possibly epidemics. We obtained $\mathcal{R}_0$ using the next-generation matrix method presented in (Yang, 2014).

Next, the matrix K is defined in Equation (22), namely

$$K = FV^{-1}, \tag{22}$$

with F and V indicating the rate of emergence of new infections in I, forming an $n \times n$ matrix,

$$F = \frac{\partial \mathcal{F}_i}{\partial x_j}(x^*), \qquad i,j = 1,2,\ldots,m,$$

and

$$V = \frac{\partial \mathcal{V}_i}{\partial x_j}(x^*), \quad i,j = 1,2,\dots,n.$$

The $\mathcal{F}_i(x,y)$ matrix is the infection rate, which contains the subpopulation of individuals who become infected with the disease in the early stages due to contact with an infected individual. Meanwhile, the $\mathcal{V}_i(x,y)$ matrix is the disease progression rate, which contains the subpopulation of individuals who become infected and progress to later stages of the disease.

From model (1), consider only the infected subpopulation, or the subpopulation that causes the spread of the disease, namely

$$\frac{dL(t)}{dt} = \frac{\beta S(t)I(t)}{1+bI(t)} + \frac{(1-\varepsilon)\beta V(t)I(t)}{1+bI(t)} + (1-p)rI(t) - \alpha L(t) - kL(t) - \mu L(t). \tag{23}$$

$$\frac{dI(t)}{dt} = kL(t) - (1-p)rI(t) - prI(t) - (\mu+d)I(t). \tag{24}$$

Based on this system, the functions $\mathcal{F}_i$ and $\mathcal{V}_i$ are obtained as follows

$$\mathcal{F}_i = \begin{bmatrix} \frac{\beta SI}{1+bI} + \frac{(1-\varepsilon)\beta VI}{1+bI} \\ 0 \end{bmatrix} = \begin{bmatrix} \mathcal{F}_1 \\ \mathcal{F}_2 \end{bmatrix}, \qquad \mathcal{V}_i = \begin{bmatrix} (\alpha+k+\mu)L - (1-p)rI \\ -kL + (r+\mu+d)I \end{bmatrix} = \begin{bmatrix} \mathcal{V}_1 \\ \mathcal{V}_2 \end{bmatrix}.$$

Next, a Jacobian matrix is formed from F and V which is evaluated against the disease-free equilibrium point, namely

$$F = \begin{bmatrix} \frac{\partial \mathcal{F}_1}{\partial L} & \frac{\partial \mathcal{F}_1}{\partial I} \\ \frac{\partial \mathcal{F}_2}{\partial L} & \frac{\partial \mathcal{F}_2}{\partial I} \end{bmatrix} = \begin{bmatrix} 0 & \frac{\beta S^* + (1-\varepsilon)\beta V^*}{(1+bI^*)^2} \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \frac{\partial \mathcal{V}_1}{\partial L} & \frac{\partial \mathcal{V}_1}{\partial I} \\ \frac{\partial \mathcal{V}_2}{\partial L} & \frac{\partial \mathcal{V}_2}{\partial I} \end{bmatrix} = \begin{bmatrix} (\alpha+k+\mu) & -(1-p)r \\ -k & (r+\mu+d) \end{bmatrix},$$

$$V^{-1} = \frac{1}{\det V}\begin{bmatrix} (r+\mu+d) & (1-p)r \\ k & (\alpha+k+\mu) \end{bmatrix},$$

$$V^{-1} = \frac{1}{(\alpha+k+\mu)(r+\mu+d) - (1-p)rk}\begin{bmatrix} (r+\mu+d) & (1-p)r \\ k & (\alpha+k+\mu) \end{bmatrix}.$$

The matrix FV^{-1} is obtained

$$FV^{-1} = \frac{1}{(\alpha+k+\mu)(r+\mu+d) - (1-p)rk}\begin{bmatrix} k(\beta S^* + (1-\varepsilon)\beta V^*) & (\alpha+k+\mu)(\beta S^* + (1-\varepsilon)\beta V^*) \\ 0 & 0 \end{bmatrix}.$$

Because

$$S^* = \frac{(1-\varphi)\Lambda}{\mu}, V^* = \frac{\varphi\Lambda}{\mu},$$

using the next generation matrix approach, the basic reproduction number of the model is obtained as

$$\mathcal{R}_0 = \rho(FV^{-1}),$$

$$\mathcal{R}_0 = \frac{\beta\Lambda k(1-\varepsilon\varphi)}{\mu[(\alpha+k+\mu)(r+\mu+d) - (1-p)rk]}.$$

Since ε and φ are parameters with valuaes between 0 and 1, $(\alpha+k+\mu)(r+\mu+\delta) - (1-p)rk = (\alpha+k+\mu)(r+\mu+\delta) - rk + prk$, where $(\alpha+k+\mu)(r+\mu+\delta) > rk$ then $(\alpha+k+\mu)(r+\mu+\delta) - (1-p)rk > 0$, so that $\mathcal{R}_0 > 0$.

3.3.3 Sensitivity Analysis

In this subsection, we investigate the effect of the various model parameters on the basic reproduction number, $\mathcal{R}_0$. For this purpose, we employ the normalized forward sensitivity index, also known as elasticity (Helton, 1985). If $\mathcal{R}_0$ is assumed to be a differentiable function with respect to a parameter π, then the normalized forward sensitivity index of $\mathcal{R}_0$ with respect to π is defined by

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

This normalized sensitivity index measures the relative change in $\mathcal{R}_0$ with respect to changes in the parameter π.

A sensitivity analysis is conducted to evaluate the effect of model parameters on the basic reproduction number. The results of the sensitivity analysis are presented in Table 1.

Table 1 Sensitivity Index $\mathcal{R}_0$.

Parameters	Sensitivity Index
Λ	$C_\Lambda^{\mathcal{R}_0} = 1 > 0$
φ	$C_\varphi^{\mathcal{R}_0} = -\frac{\varepsilon\varphi}{(1-\varepsilon\varphi)} < 0$
β	$C_\beta^{\mathcal{R}_0} = 1 > 0$
ε	$C_\varepsilon^{\mathcal{R}_0} = -\frac{\varepsilon\varphi}{(1-\varepsilon\varphi)} < 0$
p	$C_p^{\mathcal{R}_0} = -\frac{prk}{(\alpha+k+\mu)(r+\mu+\delta)-(1-p)rk} < 0$
α	$C_\alpha^{\mathcal{R}_0} = -\frac{\alpha(r+\mu+d)}{(\alpha+k+\mu)(r+\mu+d)-(1-p)rk} < 0$
k	$C_k^{\mathcal{R}_0} = 1 - \frac{k(\alpha+r+\mu+d)}{(\alpha+k+\mu)(r+\mu+d)-(1-p)rk} > 0$
r	$C_r^{\mathcal{R}_0} = -\frac{r(\alpha+\mu+pk)}{(\alpha+k+\mu)(r+\mu+d)-(1-p)rk} < 0$
d	$C_d^{\mathcal{R}_0} = -\frac{d(\alpha+k+\mu)}{(\alpha+k+\mu)(r+\mu+d)-(1-p)rk} < 0$
μ	$C_\mu^{\mathcal{R}_0} = -1 - \frac{\mu(\alpha+k+r+2\mu+d)}{(\alpha+k+\mu)(r+\mu+d)-(1-p)rk} < 0$
b	$C_b^{\mathcal{R}_0} = 0$

Table 2. List of initial values and parameter values used for the model simulations.

Variables and parameters	Description	Value	Unit	Referensi
$N(0)$	Initial total population	278,696,200	Individuals	(BPS,2023)
$S(0)$	Number of susceptible individuals	241,687,971	Individuals	Estimated
$V(0)$	Number of vaccinated individuals	4,065,600	Individuals	(BPS Proyeksi Penduduk Indonesia 2020-2050)
$L(0)$	Number of latent individuals	10,900,000	Individuals	(WHO,2023)
$I(0)$	Number of actively infected individuals	1,090,000	Individuals	(Kemenkes RI, 2023)

Variables and parameters	Description	Value	Unit	Referensi
$R(0)$	Number of recovered individuals	552,629	Individuals	(Kemenkes RI, 2023)
Λ	Recruitment (birth) rate	4.289.346	Individuals /year	Laporan Statistik Hayati,2023
φ	Vaccination rate	0,87	1/year	WHO
β	The Transmission coefficient	$9{,}9 \times 10^{-8}$	1/ (Individuals.year)	Estimated
b	Saturation rate	8.63×10^{-6}	1/ Individuals	Estimated
ε	Vaccine efficacy	0.71	-	(Machlaurin, 2023)
p	Treatment success rate	0.87	-	(Kemenkes RI, 2023)
α	Treatment rate of latent individuals	0.2	1/year	Estimated
k	Progression rate from latent to infectious individuals	0.0128	1/ year	(Kemenkes RI, 2023)
r	Treatment rate of infectious individuals	0.673	1/ year	(Kemenkes RI, 2023)
d	TB induced death rate	0.028	1/ year	(Kemenkes RI, 2023)
μ	The natural death rate	0.003646	1/ year	(BPS,2023), (Laporan Statistik Hayati,2023)

Discretization was performed using a time step of $h = 3$ years to obtain an explicit relationship between the initial conditions in 2020 and the final conditions in 2023 in the parameter estimation process. Data on the number of individuals in each compartment of the SVLIR model for the years 2020 and 2023 are provided.

Table 3 Population data for 2020-2023.

Variable	2020	2021	2022	2023
S	257,085,644	258,008,093	260,505,425	262,087,971
V	3,731,731	3,218,047	3,264,287	4,065,600
L	8,240,000	9,690,000	10,600,000	10,900,000
I	824,000	969,000	1,060,000	1,090,000
R	322,524,86	318,760	344,088	552,629
Total	270,203,900	272,203,900	275,773,800	278,696,200

The epidemiology of latent tuberculosis (TB) infection indicates that the risk of progression to active disease is highest within 2–5 years after initial infection (WHO, 2015; Price & Nguyen, 2018). Because the latent phase cannot be directly observed, many TB dynamics models assume an average latency duration of several years, with a value of approximately five years often used as a biologically realistic approximation.

The value of the parameter α in this study was determined based on the tuberculosis epidemiology literature, where the rate of progression from the latent phase to active infection is generally on the order of 0.1–0.3 per year. Some studies explicitly use a value of 0.2 per year, while data-driven estimates suggest a very close value of approximately 0.216 per year (Moualeu-Ngangue et al., 2015). Therefore, in this study, α = 0.2 per year is used, representing an average latency duration of 1/α= 5 years.

The parameter β is calibrated from the latent class dynamics equation (L) based on Indonesian TB data for the 2020–2023 period, as presented in Tables 2 and 3. The calibration process is performed by discretizing the system of differential equation using a forward difference scheme with a time step of 3 years.

$$\frac{L_{n+1}-L_n}{h}=\frac{\beta S_n I_n}{1+bI_n}+\frac{(1-\varepsilon)\beta V_n I_n}{1+bI_n}+(1-p)rI_n-(\alpha+k+\mu)L_n,$$
$$\frac{L_{2023}-L_{2020}}{3}=\frac{\beta S_{2020} I_{2020}}{1+bI_{2020}}+\frac{(1-\varepsilon)\beta V_{2020} I_{2020}}{1+bI_{2020}}+(1-p)rI_{2020}-(\alpha+k+\mu)L_{2020}. \quad (25)$$

With α = 0.2/year, the value of parameter β from equation (25) yaitu $\beta = 9.9 \times \frac{10^{-8}}{year}$.

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

Parameter	Sensitivity Index
Λ	1
φ	-1.62
β	1
ε	-1.62
p	-0.049
α	-0.93
k	0.92
r	-0.96
d	-0.04
μ	-1.02
b	0

Based on the sensitivity analysis results for the basic reproduction number ($\mathcal{R}_0$), parameters related to vaccination showed the most dominant influence in reducing the value of $\mathcal{R}_0$. Specifically, vaccine efficacy (ε) and vaccination rate (φ) had the largest negative sensitivity indices, while the transmission rate parameter (β) had the largest positive sensitivity indices. A 10% increase in the parameters ε and ϕ would decrease the value of $\mathcal{R}_0$ by approximately 16.2% each, while a 10% decrease in the parameter β would decrease $R0$ by 10%. This indicates that proportional changes in these parameters significantly affect the potential for tuberculosis transmission.

These modeling results align with the direction of tuberculosis control policy in Indonesia, which emphasizes strengthening immunization programs, increasing treatment coverage and success, and expanding TB preventive therapy to at-risk groups. Therefore, the developed model provides quantitative support for national policies in efforts to achieve the tuberculosis elimination target.

3.3.4. Stability Analysis

In this section, the stability analysis of the model's equilibrium point is summarized in the following theorem.

Lemma 3*. The disease-free equilibrium point E_0 in equation (20) of model (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

Proof. The Jacobi matrix of model (1) evaluated at the disease-free equilibrium point E^* is as follows

$$J(E_0)=\begin{pmatrix} -\mu & 0 & 0 & -\beta S_0^* & 0 \\ 0 & -\mu & 0 & -(1-\varepsilon)\beta V_0^* & 0 \\ 0 & 0 & -(\alpha+k+\mu) & \beta S_0^*+(1-\varepsilon)\beta V_0^*+(1-p)r & 0 \\ 0 & 0 & k & -(r+\mu+d) & 0 \\ 0 & 0 & \alpha & pr & -\mu \end{pmatrix}.$$

The associated characteristic equation of $J(E_0)$ is given by

$$f(\lambda)=(\lambda+\mu)^3[\lambda^2+a_1\lambda+a_0], \tag{26}$$

where

$$a_1=(\alpha+k+\mu)+(r+\mu+d)>0,$$
$$a_0=(\alpha+k+\mu)+(r+\mu+d)-k\left(\frac{\beta\Lambda(1-\varepsilon\varphi)}{\mu}+(1-p)r\right),$$

From equation (26), we obtain $\lambda_1=-\mu$, $\lambda_2=-\mu$, and $\lambda_3=-\mu$. If $\mathcal{R}_0=\frac{\beta\Lambda k(1-\varepsilon\varphi)}{\mu[(\alpha+k+\mu)(r+\mu+d)-(1-p)rk]}<1$, then $a_0=(\alpha+k+\mu)+(r+\mu+d)-k\left(\frac{\beta\Lambda(1-\varepsilon\varphi)}{\mu}+(1-p)r\right)>0$. Since $a_1, a_0>0$,

according to the Routh-Hurwitz criterion (Chitnis, et al., 2008), we obtain $Re(\lambda 4)<0$ and $Re(\lambda 5)<0$. If $\mathcal{R}_0<1$, then the real part of all eigenvalues of the Jacobi matrix J(E_0) is negative so that the stability of the equilibrium point without addiction E^* of model (1) is locally asymptotically stable.

Lemma 4. *The endemic equilibrium point E^* in equation (21) of model (1) is locally asymptotically stable, if $\mathcal{R}0>1$.*

Proof. The Jacobi matrix of model (1) evaluated at the Addiction equilibrium point E^* is as follows

$$J(\boldsymbol{E}^*)=\begin{pmatrix} -\frac{\beta I^*}{1+bI^*}-\mu & 0 & 0 & -\frac{\beta S^*}{(1+bI^*)^2} & 0 \\ 0 & -(1-\varepsilon)\frac{\beta I^*}{1+bI^*}-\mu & 0 & -(1-\varepsilon)\beta V^* & 0 \\ \frac{\beta I^*}{1+bI^*} & (1-\varepsilon)\frac{\beta I^*}{1+bI^*} & -(\alpha+k+\mu) & \frac{(\beta S^*+(1-\varepsilon)\beta V^*)}{(1+bI^*)^2}+(1-p)r & 0 \\ 0 & 0 & k & -(r+\mu+d) & 0 \\ 0 & 0 & \alpha & pr & -\mu \end{pmatrix}.$$

The associated characteristic equation of Jacobian matrix evaluated at the endemic equilibrium $J(\boldsymbol{E}^*)$ is given by

$$f(\lambda)=(\lambda+\mu)(\lambda^4+b_1\lambda^3+b_2\lambda^2+b_3\lambda+b_4), \tag{27}$$

where $b_1=a_1+a_2+a_3+a_4, b_2=(a_1+a_2)(a_3+a_4)+a_1a_2+a_3a_4+k(C+(1-p)r, b_3=a_1a_2(a_3+a_4)+a_3a_4(a_1+a_2)+k[(C+(1-p)r)(a_1+a_3)-BE]+AkD, b_4=a_1a_2a_3a_4+ka_1[a_3(C+(1-p)r)-BE]+AkDa_3$, The parameters are defined as $a_1=A+\mu$, $a_2=\alpha+k+\mu$, $a_3=B+\mu$, $a_4=r+\mu+d$, $a_1=A+\mu$, with $A=\frac{\beta I^*}{1+bI^*}>0$, $B=(1-\varepsilon)\frac{\beta I^*}{1+bI^*}>0$, $C=\frac{(\beta S^*+(1-\varepsilon)\beta V^*)}{(1+bI^*)^2}>0$, $D=\frac{\beta S^*}{(1+bI^*)^2}>0$, $E=(1-\varepsilon)\beta V^*>0$.

The roots of equation (27) are $\lambda_1=-\mu$, while the remaining eigenvalues are determined by the quartic polynomial $g(\lambda)=(\lambda^4+b_1\lambda^3+b_2\lambda^2+b_3\lambda+b_4)$. (28)

Note that the basic reproduction number is given by $\mathcal{R}_0=\frac{\beta\Lambda k(1-\varepsilon\varphi)}{\mu[(\alpha+k+\mu)(r+\mu+d)-(1-p)rk]}$. If $\mathcal{R}_0>1$, then $\beta\Lambda k(1-\varepsilon\varphi)>\mu[(\alpha+k+\mu)(r+\mu+d)-(1-p)rk]$, this implies $\beta\Lambda k(1-\varepsilon\varphi)-\mu[(\alpha+k+\mu)(r+\mu+d)-(1-p)rk]>\mathbf{0}$, or $\beta\Lambda k(1-\varepsilon\varphi)-\mu\big[(\alpha+\mu)(r+\mu+d)\big)+k(u+d+pr)\big]>0$. Consequently, the endemic equilibrium satisfies

$$I^* = \frac{\frac{\beta\Lambda}{\mu}(1-\varepsilon\varphi) - \left[\frac{(\alpha+k+\mu)(r+\mu+d)}{k} - (1-p)r\right]}{b\left[\frac{(\alpha+k+\mu)(r+\mu+d)}{k} - (1-p)r\right]}.$$

Thus, it can be simplified as

$$I^* = \frac{(R_0-1)}{b} > 0.$$

Therefore, the endemic equilibrium E^* is given by

$$S^* = \frac{(1-\varphi)\Lambda(1+bI^*)}{\beta I^* + \mu(1+bI^*)} = \frac{(1-\varphi)\Lambda(1+bI^*)}{\beta I^* + \mu(1+bI^*)} > 0,$$

$$V^* = \frac{\varphi\Lambda(1+bI^*)}{(1-\varepsilon)\beta I^* + \mu(1+bI^*)} > 0,$$

$$L^* = \frac{(r+\mu+d)}{k} I^* > 0,$$

$$R^* = \frac{prI^* + \alpha L^*}{\mu} = \frac{prI^* + \alpha\left(\frac{(r+\mu+d)}{k} I^*\right)}{\mu} > 0.$$

Next, we verify the Routh-Hurwitz conditions for the quartic polynomial (28) it will be shown that $b_1 > 0,\ b_2 > 0, b_3 > 0, b_4 > 0, b_1b_2 - b_3 > 0,$ and $b_2b_3 - b_1^2b_4 - b_3^2 > 0$. It is known that $b_1 = a_1 + a_2 + a_3 + a_4 > 0$, since all parameters $a_i > 0$, i = 1,2,3,4. Similarly, from the definitions of $b_2, b_3,$ and $b_4,$ and the positivity of all model parameters and state variables at the endemic equilibrium, it follows that

$$b_2 > 0, b_3 > 0, b_4 > 0.$$

To show that $b_3 > 0$ and $b_4 > 0$, note that the term $a_3(C + (1-p)r) - BE$ can be expressed in terms of endemic equilibrium quantities. Using the equilibrium relation

$\beta S^* + (1-\varepsilon)\beta V^* = (1+bI^*)(\mu+r+d)$ or $(1-\varepsilon)\beta V^* = (1+bI^*)(\mu+r+d) - \beta S^*$.

Because $S^* > 0$ then

$$(1-\varepsilon)\beta V^* < (1+bI^*)(\mu+r+d).$$

Multiply both sides by $(1-\varepsilon)\beta I^*(1+bI^*)^2$,

$$(1-\varepsilon)^2\beta^2 I^* V^*(1+bI^*)^2 < (\mu+r+d)(1-\varepsilon)\beta I^*(1+bI^*)(1+bI^*)^2$$

Because $(1+bI^*)^2 > 1$ and $(\mu+r+d)(1-\varepsilon)\beta I^*(1+bI^*)$ part of the positive term then $a_3(C+(1-p)r) - BE > 0$ consequently $b_3 > 0$, $b_4 > 0$.

Furthermore, by grouping positive terms in the expression of $b_1b_2 - b_3$ it can be shown that

$$b_1b_2 - b_3 > 0,$$

since the dominant positive terms outweigh the negative components. Next, consider

$$\Delta = b_3(b_1b_2 - b_3) - b_1^2b_4 > 0.$$

Using the relation $I^* = \frac{(\mathcal{R}_0-1)}{b} > 1$, we can write $b_3 = I^*C_1$, and $b_4 = I^*C_2$, where $C_1 > 0$ and $C_2 > 0$. Thus,

$$\Delta = b_1b_2b_3 - b_1^2b_4 - b_3^2$$
$$= b_1b_2(I^*C_1) - b_1^2I^*C_2 - (I^*C_1)^2$$
$$= I^*(b_1b_2C_1 - b_1^2C_2 - C_1^2I^*)$$
$$\Delta = \frac{(\mathcal{R}_0-1)}{b}(b_1b_2C_1 - b_1^2C_2 - C_1^2I^*).$$

Or $\Delta = (\mathcal{R}_0 - 1)\left(\frac{1}{b}b_1b_2C_1 - \frac{1}{b}b_1^2C_2 - C_1^2\frac{(\mathcal{R}_0-1)}{b}\right)$.

For $\mathcal{R}_0 > 1$, we have I^* and $(\mathcal{R}_0 - 1) > 0$,

for $\mathcal{R}_0$ sufficiently close to 1, we have

$$\left(\frac{1}{b}b_1b_2C_1 - \frac{1}{b}b_1^2C_2 - C_1^2\frac{(\mathcal{R}_0-1)}{b}\right) > 0.$$

which implies that $\Delta > 0$, Therefore, all the Routh–Hurwitz conditions (Chitnis et al., 2008) are satisfied, namely $b_1 > 0,\ b_2 > 0, b_3 > 0, b_4 > 0, b_1b_2 - b_3 > 0,$ and $\Delta = b_1b_2b_3 - b_1^2b_4 - b_3^2 > 0$, Hence, all eigenvalues satisfy $\mathrm{Re}(\lambda_2) < 0, \mathrm{Re}(\lambda_2) < 0, \mathrm{Re}(\lambda_3) < 0,$ and $\mathrm{Re}(\lambda_4) < 0$. This

analysis shows that, whenever $\mathcal{R}_0 > 1$ maka $\mathrm{Re}(\lambda_i) < 0$, i = 1,2,3,4,5. the endemic equilibrium point E* **is** locally asymptotically stable**.**

3.3.5 Numerical Simulation

To illustrate some of the analytical results obtained previously, a numerical simulation was conducted using the initial values and parameter values listed in Table 2. In this study, the initial values and parameter values of the TB model were estimated based on Indonesian data. The initial values were obtained from World Health Organization (WHO) data, the 2023 Indonesian Ministry of Health TB Control Program Report, the Indonesian Biostatistics Report, and the Indonesian Central Statistics Agency (BPS) in 2023. In this study, these initial values and parameters were used so that the simulation could be applied to Indonesia.

Numerical simulations were performed to illustrate the system dynamics around the disease-free equilibrium point and to verify the results of the analytical stability analysis. Based on the parameter values provided in Table 2, with initial conditions $N(0) = 278{,}696{,}200$, $S(0) = 262{,}087{,}971, V(0) = 4{,}065{,}600$, $L(0) = 10{,}900{,}000, I(0) = 1{,}090{,}000$, $R(0) = 552{,}629$. and $\beta = 9.9 \times 10^{-8}$, the basic reproduction number was found to be $\mathcal{R}_0 = 3.76 > 1$ indicating that the system tends toward an endemic equilibrium state. The simulations are conducted within a time range of 0 and 50 years since pulmonary tuberculosis infections take a longer time to reactivate into a chronic disease and subsequently cured. The simulation results are presented graphically in Figures. 2–6.

3.3.5.1. Effect of Vaccine Efficacy Variation on Latent, Actively Infected, and Recovered Populations

In the model flow diagram shown in Figure 1, ε represents the vaccine efficacy. Figure 2 illustrates the effect of varying vaccine efficacy on the latent infected population. It is observed that an increase in vaccine efficacy leads to a reduction in the number of individuals in the latent infection compartment.

This reduction occurs because vaccines with higher efficacy enhance the immune response, thereby decreasing the probability that susceptible or vaccinated individuals progress to the latent infection stage after exposure to the pathogen. Therefore, the development of highly effective vaccines plays a crucial role in reducing the number of latent infection cases within the population.

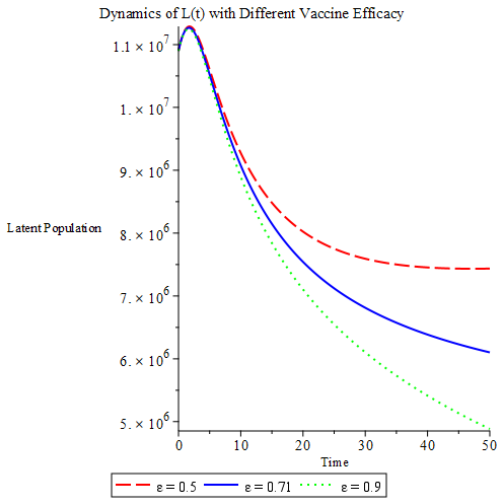

Figure 2. Effects of varying vaccine efficacy (ε) on the laten population.

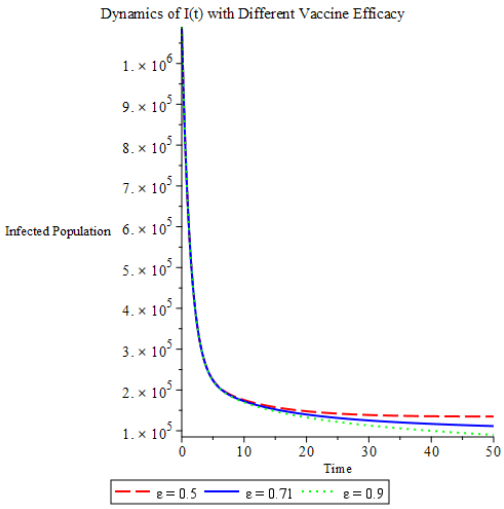

Figure 3. Effects of varying vaccine efficacy (ε) on the infectious population undergoing treatment.

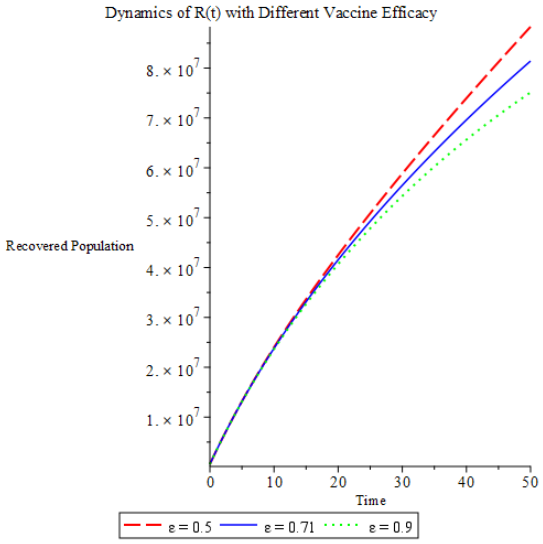

Figure 4. Effects of varying vaccine efficacy (ε) on the recovered population.

The study of the latent population in tuberculosis is of great importance because individuals in the latent stage do not exhibit clinical symptoms, yet they still harbor *Mycobacterium tuberculosis* within their bodies. Individuals in this condition have the potential to progress to active tuberculosis if their immune system becomes weakened. Therefore, the latent population can act as a reservoir of infection that contributes to the emergence of new tuberculosis cases in the future.

Moreover, a high number of individuals in the latent stage may prolong the persistence of the disease within the population, even when the number of active infection cases appears to decline. Hence, understanding the dynamics of the latent population through a mathematical modeling approach is essential for identifying effective control strategies, including improving vaccine efficacy, expanding vaccination coverage, and implementing medical interventions aimed at preventing the progression from latent infection to active tuberculosis.

Figure 3 illustrates that an increase in vaccine efficacy (ε) leads to a reduction in the population of actively infected individuals undergoing treatment. Furthermore, the results indicate that the number of individuals receiving treatment decreases after five years when a vaccine with 90% efficacy is administered. Higher vaccine efficacy enhances individual immunity, thereby reducing infection transmission and consequently decreasing the number of individuals requiring treatment significantly. This reduction not only lowers the risk of transmission to healthcare workers but also decreases the resources required for treatment, particularly in Indonesia.

Figure 4 illustrates the effect of vaccine efficacy on the recovered population. It is observed that increasing vaccine efficacy results in a decrease in the number of individuals in the recovered class. This phenomenon occurs because fewer individuals become infected due to enhanced immunity. Thus, it is evident that vaccines with higher efficacy effectively reduce infection transmission within the population.

3.3.5.2. Effect of Vaccine Efficacy and Vaccination Rate Variation on the Control Reproduction Number Due to the Actively Infected Population

Figure 6 shows that an increase in vaccine efficacy leads to a decrease in the control reproduction number associated with the actively infected population. This reduction occurs as the number of

infected individuals declines, which in turn lowers the transmission rate of the disease to the susceptible population.

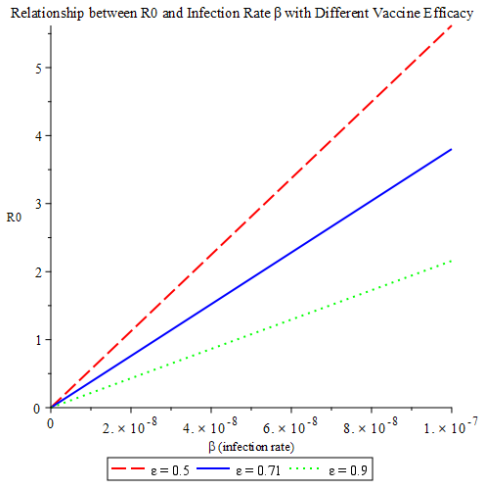

Figure 5. Effects of varying vaccine efficacy (ε) on the control reproduction number due to asymptomatic infectious population.

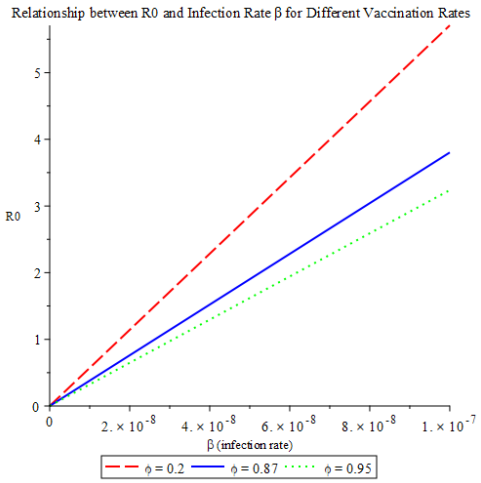

Figure 6. Effects of varying vaccination rate (φ) on the control reproduction number due to the asymptomatic infectious population.

Referring to the model flow diagram in Figure 1, φ represents the vaccination rate against tuberculosis. Figure 6 illustrates the effect of the vaccination rate, using a vaccine with 90% efficacy, on the control reproduction number associated with the latent infected population. It is observed that an increase in the vaccination rate leads to a decrease in the control reproduction number, thereby reducing the level of infection transmission. An increase in vaccination coverage raises the proportion of individuals with improved immunity, which in turn contributes to lowering the spread of infection within the population.

4. Conclusion

In this study, a mathematical model is developed to investigate the effects of vaccine efficacy and vaccination rate on both the latent and actively infected populations. The focus on the latent population is motivated by their critical role in disease dynamics, as these individuals do not exhibit clinical symptoms but continue to carry *Mycobacterium tuberculosis* within their bodies. They may progress to active tuberculosis when their immune system weakens. Meanwhile, the actively infected population is targeted due to their role in continuously transmitting the infection to susceptible individuals, thereby contributing to a high transmission rate.

The proposed model examines the dynamic behavior of tuberculosis (TB) transmission by incorporating vaccination and a saturated incidence rate. The total population is divided into five compartments: susceptible (S), vaccinated (V), latent (L), actively infected (I), and recovered (R). Based on numerical simulations and theoretical analysis, vaccine efficacy (ε) is found to have a significant impact on the disease dynamics, as reflected in the behavior of $L(t)$, $I(t)$, and $R(t)$, as well as in the value of the basic reproduction number $\mathcal{R}_0$. Under the baseline condition $\varepsilon = 0.71$, the system exhibits endemic behavior, where the latent and infected populations initially increase, reach a peak, and then decline toward a steady state, while the recovered population gradually increases until it stabilizes.

When vaccine efficacy is reduced to $\varepsilon = 0.5$ (a decrease of approximately 29.6%), the infected population $I(t)$ reaches a higher peak and declines more slowly. This leads to an increase in the latent population $L(t)$ and prolongs the duration of infection within the population. Consequently,

the recovered population $R(t)$ becomes larger, reflecting a higher number of individuals who were previously infected. This condition indicates that lower vaccine efficacy increases the overall disease burden.
In contrast, increasing vaccine efficacy to ε = 0.9 (an increase of approximately 26.8%) results in a significant reduction in both the latent and infected populations. The infection peak becomes lower, and the system reaches equilibrium more quickly. In this case, the recovered population $R(t)$ grows more slowly and attains a lower final value, indicating that fewer individuals experience infection over time. Thus, a lower value of $R(t)$ in this context reflects a more controlled epidemiological situation.

Based on the sensitivity analysis results for the basic reproduction number ($\mathcal{R}_0$), parameters related to vaccination showed the most dominant influence in reducing the value of $\mathcal{R}_0$. Specifically, vaccine efficacy (ε) and vaccination rate (φ) had the largest negative sensitivity indices, while the transmission rate parameter (β) had the largest positive sensitivity indices. A 10% increase in the parameters ε and ϕ would decrease the value of $\mathcal{R}_0$ by approximately 16.2% each, while a 10% decrease in the parameter β would decrease $\mathcal{R}_0$ by 10%. This indicates that proportional changes in these parameters significantly affect the potential for tuberculosis transmission.

Furthermore, the relationship between $\mathcal{R}_0$ and the transmission rate β exhibits an increasing linear pattern for each level of vaccine efficacy. However, the slope of the curve depends on the value of ε, where lower vaccine efficacy results in significantly higher $\mathcal{R}_0$ values for the same β. Conversely, higher vaccine efficacy reduces the slope, yielding lower $\mathcal{R}_0$ values for identical transmission rates. This demonstrates that vaccine efficacy can mitigate the impact of increased transmission rates on disease spread.

Overall, there is strong consistency between the simulation results, sensitivity analysis, and the relationship between $\mathcal{R}_0$ and transmission parameters. Vaccine efficacy and vaccination rates not only reduces the epidemic threshold $\mathcal{R}_0$, but also directly decreases the number of latent and infected individuals, while influencing the accumulation pattern of recovered individuals. Therefore, improving vaccine efficacy and vaccination rates is a highly effective strategy for disease control, as it simultaneously reduces transmission and the overall infection burden within the population.

For future research, several extensions can be considered: (1) incorporating population heterogeneity, such as differences in age, region, or vaccination compliance; (2) examining the role of comorbidities in TB transmission; (3) evaluating additional control strategies, including treatment, active screening, and mask usage within the modeling framework; and (4) analyzing bifurcation phenomena and the possibility of multiple equilibria to gain deeper insight into system dynamics.

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