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SMILS III

Integrating Disease Dynamics and Environmental Sanitation: A Mathematical Model of Tuberculosis Spread in Communities with Compromised Water and Waste Management

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Abstract. Tuberculosis (TB) remains a global health challenge, particularly in areas with inadequate environmental sanitation. Poor water quality and ineffective waste management can indirectly exacerbate TB transmission by weakening public health resilience, increasing vulnerability to infection, and complicating recovery. This study develops a compartmental mathematical model that integrates TB disease dynamics with environmental sanitation factors, specifically focusing on communities facing disrupted water supply and waste disposal systems. TB is a disease caused by infection with the bacterium Mycobacterium tuberculosis. The developed mathematical model of TB spread is a system of nonlinear differential equations with six variables: susceptible, vaccinated, latent, infected, drug-resistant, and cured, while incorporating sanitation-related parameters that influence susceptibility and recovery levels. The basic reproduction number (R_0) is then derived as a function of epidemiological and sanitation parameters, using a next-generation matrix. Improved sanitation results in a smaller R_0. Furthermore, local stability analysis around non-endemic and endemic equilibrium points is performed using the Routh-Hurwitz method. If R_0 is less than one, the non-endemic equilibrium is asymptotically stable, indicating that TB will disappear from the community over time. If R_0 is greater than one, the endemic equilibrium is asymptotically stable, indicating that TB will spread throughout the community over time. Simulation results show that if parameters related to sanitation are improved, the number of individuals recovering will increase. These results highlight the link between public health and the environment, which policymakers can use to evaluate interventions to reduce TB transmission in the community.

1 Introduction

Tuberculosis (TB) is an infectious disease that remains a significant public health issue. It is caused by the bacterium Mycobacterium tuberculosis. While it typically attacks the lungs, it can also affect other organs. The spread of TB is also greatly influenced by environmental conditions [1].

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Environments with poor sanitation, inadequate ventilation, and Poor air conditions have the potential to increase disease transmission. Although TB is generally transmitted through the air (airborne transmission), its spread can occur more rapidly in environments with poor sanitation [2]. Inadequate sanitation conditions can reduce public health, weaken the immune system, and increase the frequency of interactions in closed spaces or densely populated areas, allowing the Mycobacterium tuberculosis bacteria to survive longer [3]. In addition, dirty and humid environments can be ideal places for bacteria to grow and weaken the immune system of residents, making them more susceptible to infection[4].

Poor water and waste management can exacerbate the spread of TB. Suboptimal water management can be an indirect factor that can trigger the spread of TB because environmental contamination helps maintain disease transmission. Limited clean water supplies hinder personal and environmental hygiene, increasing the opportunity for transmission of various diseases, including TB. Poor sanitation often creates densely populated and humid environments, which support the growth of airborne Mycobacterium tuberculosis bacteria, especially in enclosed spaces with minimal air circulation. A lack of clean water also has the potential to weaken the immune system by increasing the risk of exposure to other diseases, making people more susceptible to contracting or experiencing a relapse of TB. Therefore, good water management and the provision of adequate sanitation facilities are strategic steps in preventing and controlling this disease.

Good sanitation plays a crucial role in TB control through improved air quality, ventilation, and proper waste management, thus reducing the risk of transmission and improving general health and immunity [5]. Accumulated garbage, improper medical waste disposal, poor ventilation, high humidity, and overcrowding can degrade environmental quality, increase re-exposure to TB bacteria, and increase the risk of latent TB developing into active TB. Poor environmental conditions can also accelerate the spread of the disease, particularly in densely populated areas with limited health facilities. Therefore, good water management and the provision of adequate sanitation facilities are strategic steps to prevent and control this disease.

In Indonesia, TB remains a serious health problem [6]. Then the emergence of drug-resistant tuberculosis (MDR-TB) cases exacerbates the challenge of controlling this disease. MDR-TB occurs when the bacteria that cause TB mutate or acquire resistance to first-line drugs, such as isoniazid and rifampicin, making treatment more difficult, longer-lasting, and more expensive.

In an effort to understand and control the spread of disease, an effective approach can be taken by using mathematical models, because they are able to describe the dynamics of transmission and the impact of interventions. Mathematical modelling is used to predict the spread of disease, the number of active sufferers, and the length of the pandemic [7]. Various mathematical models, such as SIS, SIR, SEIR, and their variations, have been used to predict the spread of TB disease. Previous research has utilized mathematical models to explore various control strategies for preventing the spread of tuberculosis disease at different stages of infection. One of the primary preventive measures is the administration of the BCG vaccine to individuals who remain in a vulnerable condition [8]. In addition, other strategies include providing treatment at an early stage, before the infection develops further [9], as well as medical therapy for individuals who are already in the latent phase or infected [10-11]. However, in some cases, this treatment can lead to the emergence of drug resistance, which is characterized by the emergence of first-line drug resistance (MDR-TB) [7-12], even to resistance to second-line drugs (XDR-TB) [7]. In severe stages of infection, intensive treatment in hospital or medical quarantine is a highly recommended step to prevent further transmission [13-14].

Unlike previous TB models that primarily emphasize biological aspects (infection, latent, resistance, and recovery), this study explicitly incorporates environmental sanitation into the disease dynamics. The model highlights that proper sanitation can accelerate recovery by enhancing immunity, whereas poor sanitation, limited access to clean water, and water pollution may increase the risk of transmission. Thus, this model not only describes epidemiological dynamics but also links them to freshwater sustainability issues, where clean water management and pollution prevention constitute essential components of TB control strategies.

Based on this, the author aims to study a mathematical model of tuberculosis's stability. The model uses six variables: susceptible, vaccinated, latent, infected, drug-resistant infected, and cured. Individuals receiving the vaccine may move to the cured group if the vaccine is fully effective. They may move to a latent immunity decreases. Latent individuals may recover if good sanitation supports immunity and suppresses infection activation. This analysis highlights the link between public health and the environment. Policymakers can use these results to evaluate TB interventions in the community.

From this model, we will find the non-endemic and endemic equilibrium points and the basic reproduction number. Next, we will conduct a local stability analysis for both non-endemic and endemic equilibrium points. Numerical simulations will then be performed to visualize the model.

2 Formulation of the Model

The model studied in this study regarding the distribution of tuberculosis explains the relationship between six subpopulations, namely the class of individuals susceptible (S), the class of individuals vaccinated (V), the class of individuals Latent (L), the class of individuals infected (I), the class of individuals infected with MDR-TB (M), and the class of individuals Recovered (R). Thereforethe population at time t is expressed by $N(t) = S(t) + V(t) + L(t) + I(t) + M(t) + R(t)$**.** In this model, Λ represents the rate of recruitment of individuals into class S, while other parameters include β is the rate of disease spread, μ is the natural death rate, η is the vaccination rate from S to V, $(1-\tau)\kappa VI$ is the fraction of contact rate from V to L, $\tau\kappa VI$ is the fraction of progression to the recovery stage, θ is the rate of progression from L to I, ω is the rate of recovery due to good sanitation factors, σ is the rate of progression from I to M, γ_1 is the recovery rate of individual I, γ_2 is the recovery rate of individual M, δ_1 is the rate of death due to disease in individual I, δ_2 is the rate of death due to disease in individual M. The following is Figure 1. Schematic of the TB disease transmission model.

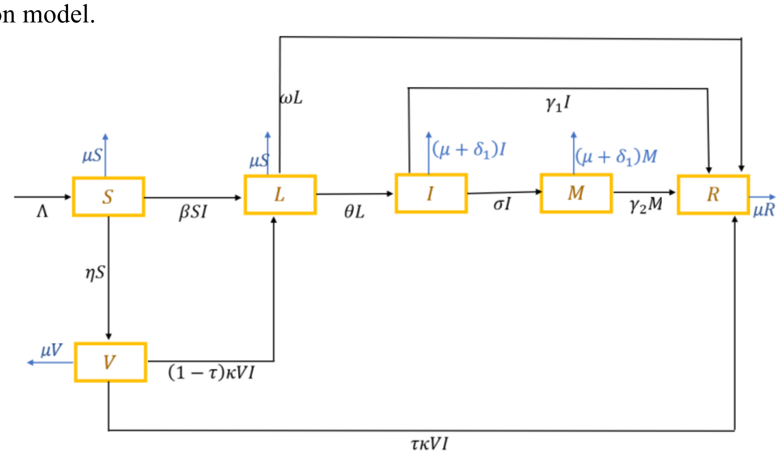

Fig.1. Schematic of the TB disease transmission model

From the image above, it can be written as a non-linear differential equation as follows:

$$
\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - (\mu + \eta)S \\
\frac{dV}{dt} &= \eta S - \kappa VI - \mu V \\
\frac{dL}{dt} &= \beta SI + (1-\tau)\kappa VI - (\theta + \omega + \mu)L \\
\frac{dI}{dt} &= \theta L - (\sigma + \gamma_1 + \delta_1 + \mu)I \\
\frac{dM}{dt} &= \sigma I - (\gamma_2 + \delta_2 + \mu)M \\
\frac{dR}{dt} &= \tau\kappa VI + \omega L + \gamma_1 I + \gamma_2 M - \mu R
\end{aligned}
\qquad (2.1)
$$

with $S(0) \geq 0, V(0) \geq 0, L(0) \geq 0, I(0) \geq 0, M(0) \geq 0, R(0) \geq 0$, and parameters $\Lambda > 0, 0 < \beta, \mu, \eta, \kappa, \tau, \theta, \omega, \sigma, \gamma_1, \delta_1, \gamma_2, \delta_2 < 1$.

3 Mathematical Model analysis

3.1 The Equilibrium State

The equilibrium point is a state when the total number of subpopulations does not change over time. A TB distribution model is said to be in equilibrium if the following conditions are met:

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dM}{dt} = \frac{dR}{dt} = 0 \qquad (3.1)$$

The system of equations (3.1) consists of two equilibrium points: disease-free/non-endemic equilibrium (DFE), denoted by $E_0(S_0, V_0, L_0, I_0, M_0, R_0) = \left(\frac{\Lambda}{(\mu+\eta)}, \frac{\eta\Lambda}{\mu(\mu+\eta)}, 0,0,0,0\right)$ and the endemic equilibrium (EE), denoted by $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$.

There is a condition called the endemic equilibrium point, which is a condition where the spread of TB disease continues so that the variables S, V, L, I, M, R, are not equal to zero. Then, $E_1(S^*, V^*, L^*, I^*, M^*, R^*)$ is obtained with the following values:

$$
\begin{aligned}
S^* &= \frac{\Lambda}{(\beta I^* + \mu + \eta)} \\
V^* &= \frac{\eta\Lambda}{(\beta I^* + \mu + \eta)(\kappa I + \mu)} \\
L^* &= I^*\left(\frac{\beta\Lambda(\kappa I^* + \mu) + (1-\tau)\kappa\eta\Lambda}{(\beta I^* + \mu + \eta)(\kappa I^* + \mu)(\theta + \omega + \mu)}\right) \\
I^* &= \frac{\mu(\eta + \mu)(\sigma + \gamma_1 + \delta_1 + \mu)}{\beta\kappa(\sigma + \gamma_1 + \delta_1) + \mu} - (R_0 - 1) \\
M^* &= \frac{\sigma I^*}{(\gamma_2 + \delta_2 + \mu)} \\
R^* &= \tau\kappa V^* I^* + \omega L^* + \gamma_1 I^* + \gamma_2 M^* - \mu R^*
\end{aligned}
$$

3.2Basic Reproduction Number

The basic reproduction number (R_0) is an epidemiological indicator that states the average number of new individuals infected by one infectious individual in a susceptible population. If the basic reproduction number $R_0 < 1$ means that one infected individual can infect less than one susceptible individual so that the disease cannot develop in a population, but if the basic reproduction number $R_0 > 1$ means that one infected individual can infect more than one susceptible individual so that the disease can spread in a population and its spread is endemic.

Determination of the basic reproduction value (R_0) is carried out using the next generation matrix (NGM) method. The application of the NGM method is carried out by finding the largest eigenvalue of the Jacobian matrix in a non-endemic equilibrium. In this dynamic system model, individuals who fall into the infected category are in the latent (L), infectious (I), and MDR-TB (M) compartments.

For example, $x = [L, I, M]^T$ thus it can be formulated as

$$\frac{dx}{dt} = F(x) - V(x) = \begin{bmatrix} \beta SI + (1-\tau)\kappa VI - (\theta + \omega + \mu)L \\ \theta L - (\sigma + \gamma_1 + \delta_1 + \mu)I \\ \sigma I - (\gamma_2 + \delta_2 + \mu)M \end{bmatrix}$$

where $F(x)$ is a matrix containing individuals infected with TB in the early stages and $V(x)$ is a matrix containing individuals infected with TB.

Then the eigenvalues in the NGM matrix are searched using the formula $NGM = F(E_0) \cdot V^{-1}$, where the basic reproduction number (R_0) is obtained from the spectral radius of the NGM determined using the non-endemic equilibrium point, so that the results of the R_0 calculation are obtained as follows:

$$R_0 = \frac{\Lambda\theta\big(\beta\mu + \eta\kappa(1-\tau)\big)}{\mu(\eta+\mu)(\theta+\omega+\mu)(\sigma+\gamma_1+\delta_1+\mu)},$$

with $0 < \tau < 1$.

The R_0 value above shows that the ω parameter, which represents the recovery rate due to good sanitation, has an inverse relationship with the basic reproduction rate. Increasing the ω value will decrease the R_0value, thereby reducing the potential for an epidemic. This parameter plays a crucial role in increasing the recovery rate, reducing the number of infected individuals, and accelerating the achievement of a TB-free state when $R_0 > 1$.

3.3Stability Analysis

Dynamics of disease transmission around equilibrium conditions, at $E_0(S_0, V_0, L_0, I_0, M_0, R_0)$ and $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$, can be determined using stability analysis. After that, a stability analysis was carried out using the Jacobian matrix to determine the eigenvalues $E_0(S_0, V_0, L_0, I_0, M_0, R_0)$ and $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$.

The eigenvalues are obtained via the Jacobian $\big(J(E_0)\big)$ Useful to help with stability $E_0(S_0, V_0, L_0, I_0, M_0, R_0)$. Using the Routh-Hurwitz criteria.

Theorem 3.1. Given $R_0 = \frac{\Lambda\theta(\beta\mu+\eta\kappa(1-\tau))}{\mu(\eta+\mu)(\theta+\omega+\mu)(\sigma+\gamma_1+\delta_1+\mu)}$

i. If $R_0 < 1$, then $E_0(S_0, V_0, L_0, I_0, M_0, R_0) = \left(\frac{\Lambda}{(\mu+\eta)}, \frac{\eta\Lambda}{\mu(\mu+\eta)}, 0,0,0,0\right)$ is locally asymptotically stable

ii. If $R_0 > 1$, then $E_0(S_0, V_0, L_0, I_0, M_0, R_0) = \left(\frac{\Lambda}{(\mu+\eta)}, \frac{\eta\Lambda}{\mu(\mu+\eta)}, 0,0,0,0\right)$ is unstable.

Proof:

Assume $E_0(S_0, V_0, L_0, I_0, M_0, R_0) = \left(\frac{\Lambda}{(\mu+\eta)}, \frac{\eta\Lambda}{\mu(\mu+\eta)}, 0,0,0,0\right)$ then by using the following formula we can calculate the eigenvalues of the Jacobian matrix in $E_0(S_0, V_0, L_0, I_0, M_0, R_0)$:

$$|J(E_0) - xI| = 0$$

After obtaining the matrix determinant, the next step is to determine the characteristic equation.

$$(\mu+\eta+x)(\mu+x)(\gamma_2+\delta_2+\mu+x)\left((\theta+\omega+\mu-x)(\sigma+\gamma_1+\delta_1+\mu+x) - \theta\left(\frac{\beta\Lambda}{(\mu+\eta)} + \frac{(1-\tau)\kappa\eta\Lambda}{(\mu+\eta)\mu}\right)\right)$$

From the equation, the eigenvalues $x_1 = -(\mu+\eta), x_2 = -\mu, x_3 = -(\gamma_2+\delta_2+\mu)$are obtained and the polynomial equation $p(x) = a_0x^2 + a_1x + a_2$, with

$$a_0 = 1$$
$$a_1 = (\theta+\omega+\mu) + (\sigma+\gamma_1+\delta_1+\mu)$$
$$a_2 = (\theta+\omega+\mu) + (\sigma+\gamma_1+\delta_1+\mu) - \theta\left(\frac{\beta\Lambda}{(\mu+\eta)} + \frac{(1-\tau)\kappa\eta\Lambda}{(\mu+\eta)\mu}\right)$$

Based on the Routh-Hurwitz criterion, local asymptotic stability at a non-endemic equilibrium point is achieved if the coefficients of the equation satisfy the conditions $a_1 > 0$ and $a_1 > 0$, which can be written as follows:

$$a_1 = (\theta+\omega+\mu) + (\sigma+\gamma_1+\delta_1+\mu) > 0$$

It will be shown that $a_2 > 0$, with

$$a_2 = \left(\frac{(\theta+\omega+\mu)(\sigma+\gamma_1+\delta_1+\mu)(\mu+\eta)\mu}{(\mu+\eta)\mu} - \frac{\theta(\beta\Lambda\mu + (1-\tau)\kappa\eta\Lambda)}{(\mu+\eta)\mu}\right)$$

Suppose

$$D = (\theta+\omega+\mu)(\sigma+\gamma_1+\delta_1+\mu)(\mu+\eta)\mu$$
$$E = \theta(\beta\Lambda\mu + (1-\tau)\kappa\eta\Lambda)$$

Then we get:

$$a_2 = \frac{D}{(\mu+\eta)\mu} - \frac{E}{(\mu+\eta)\mu}$$

Since the denominator is positive, then $a_2 > 0$ if it is fulfilled $D > E$

In the calculations that have been carried out, it is proven that $a_1 > 0$. then the condition $a_2 > 0$ if $D > E$. Therefore, the results of the analysis show that the non-endemic equilibrium point is locally asymptotically stable when $R_0 < 1$. This indicates that each infected individual is only capable of transmitting the disease to less than one susceptible person, so that within a certain period of time the disease will disappear naturally (non-endemic state). ■

Stability analysis on at $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$ can be done as follows.

Theorem 3.2. Given $R_0 = \frac{\Lambda\theta(\beta\mu+\eta\kappa(1-\tau))}{\mu(\eta+\mu)(\theta+\omega+\mu)(\sigma+\gamma_1+\delta_1+\mu)}$

i. If $R_0 > 1$, then $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$ is locally asymptotically stable

ii. If $R_0 < 1$, then $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$ is unstable.

Proof:

Assume $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$, then by using the following formula we can calculate the eigenvalues of the Jacobian matrix in $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$

$$|J(E^*) - xI| = 0$$

After determining the matrix determinant, the next step is to obtain the characteristic equation.

$$(\gamma_2 + \delta_2 + \mu)(x^4 + a_1x^3 + a_2x^2 + a_3x + a_4)$$

In the characteristic equation above, the eigenvalue $x_1 = -(\gamma_2 + \delta_2 + \mu)$ is obtained and the polynomial equation $P(x) = x^4 + a_1x^3 + a_2x^2 + a_3x + a_4$ is obtained.

with,

$$
\begin{aligned}
a_0 &= 1\\
a_1 &= I^*(\beta + \kappa) + \delta_1 + \eta + \gamma_1 + 4\mu + \omega + \sigma + \theta\\
a_2 &= I^{*2}\beta\,\kappa - I^*\beta\delta_1 - I^*\beta\gamma_1 - 3I^*\beta\mu - I^*\beta\omega\\
&\quad -I^*\beta\sigma - I^*\beta\theta - I^*\delta_1\kappa - I^*\eta\kappa - I^*\gamma_1\kappa - 3I^*\kappa\mu\\
&\quad -I^*\kappa\omega - I^*\kappa\sigma - I^*\kappa\theta - S^*\beta\theta + V^*\kappa\tau\theta - V^*\kappa\theta\\
&\quad +\delta_1\eta + 3\delta_1\mu + \delta_1\omega + \delta_1\theta + \eta\gamma_1 + 3\eta\mu + \eta\omega + \eta\\
&\quad +\eta\theta + 3\gamma_1\mu + \gamma_1\omega + \gamma_1\theta + 6\mu^2 + 3\mu\omega + 3\mu\sigma\\
&\quad +3\mu\theta + \omega\sigma + \sigma\theta\\
a_3 &= \left(I^{*2}\beta\kappa(\delta_1 + \gamma_1 + \omega + \sigma + \theta + 2\mu)\right)\\
&\quad +I^*(\beta\kappa\theta(S^* + (1-\tau)V^*) - \beta(\,(\delta_1 + \gamma_1)(\omega + \theta + 2\mu) + 3\mu^2 + 2\mu(\omega + \sigma + \theta) + \omega\sigma\\
&\quad + \sigma\theta)\\
&\quad - \kappa[(\delta_1 + \gamma_1 + \eta)(\omega + \theta + 2\mu) + 3\mu^2 + 2\mu(\omega + \sigma + \theta) + \omega\sigma + \sigma\theta])\\
&\quad + (-\beta\theta(\eta + 2\mu)S^* + \kappa\theta(\eta + 2\mu)(\tau - 1)V^*) + 2\delta_1\eta\mu + \delta_1\eta(\omega + \theta)\\
&\quad + 3\delta_1\mu^2 + 2\delta_1\mu(\omega + \theta) + 2\eta\gamma_1\mu + \eta\gamma_1(\omega + \theta) + 3\eta\mu^2\\
&\quad + 2\eta\mu(\omega + \sigma + \theta) + \eta(\omega\sigma + \sigma\theta) + 3\gamma_1\mu^2 + 2\gamma_1\mu(\omega + \theta) + 4\mu^3\\
&\quad + 3\mu^2(\omega + \sigma + \theta) + 2\mu(\omega\sigma + \sigma\theta)\\
a_4 &= \left(I^{*2}\beta\kappa(\delta_1 + \gamma_1 + \mu + \sigma)(\mu + \omega + \theta)\right.\\
&\quad + I^*\left(-\beta\mu\big((\delta_1 + \gamma_1 + \mu)(\mu + \omega + \theta) - \mu^2\sigma - \mu\omega\sigma - \mu\sigma\theta\big)\right.\\
&\quad + \kappa(-\delta_1\eta\mu - \delta_1\eta\omega - \delta_1\eta\theta - \delta_1\mu^2 - \delta_1\mu\omega - \delta_1\mu\theta - \eta\gamma_1\mu - \eta\gamma_1\omega\\
&\quad - \eta\gamma_1\theta - \eta\mu^2 - \eta\mu\omega - \eta\mu\sigma - \eta\sigma\theta - \gamma_1\mu^2 - \gamma_1\mu\omega - \gamma_1\mu\theta - \mu^3 - \mu^2\omega\\
&\quad - \mu^2\sigma - \mu^2\theta - \mu\omega\sigma - \mu\sigma\theta) + S^*(\beta\eta\kappa\tau\theta + \beta\kappa\mu\theta)\\
&\quad \left. + V^*(-\beta\kappa\mu\tau\theta + \beta\kappa\mu\theta)\right) + \big(-\beta\mu\theta(\eta + \mu)\big)S^* + \kappa\mu\theta(\eta + \mu)(\tau - 1)V^*\\
&\quad + \mu(\eta + \mu)(\mu + \omega + \theta)(\delta_1 + \gamma_1 + \mu + \sigma)
\end{aligned}
$$

Based on the Routh-Hurwitz criterion, the equilibrium point $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$ is locally asymptotically stable if all the roots of the characteristic polynomial are negative, with the following conditions:

$$x_1 + x_2 + x_3 + x_4 = -\frac{a}{b} < 0$$

with $b = a_0 = 1$ dan $a = a_1$, then obtained:

$$-(I^*(\beta + \kappa) + \delta_1 + \eta + \gamma_1 + 4\mu + \omega + \sigma + \theta) < 0$$

$$\Leftrightarrow -\left(\frac{\mu(\eta+\mu)(\sigma+\gamma_1+\delta_1+\mu)}{\beta\kappa(\sigma+\gamma_1+\delta_1)+\mu} - (R_0 - 1)\right)(\beta + \kappa) - (\delta_1 + \eta + \gamma_1 + 4\mu + \omega + \sigma + \theta) < 0$$

suppose:

$$A = (\delta_1 + \eta + \gamma_1 + 4\mu + \omega + \sigma + \theta)$$
$$B = \frac{\mu(\eta + \mu)(\sigma + \gamma_1 + \delta_1 + \mu)}{\beta\kappa(\sigma + \gamma_1 + \delta_1) + \mu}$$

conditions are met if $R_0 > 1$

In a similar way, it is obtained

$$x_1\, x_2 + x_1\, x_3 + x_1\, x_4 + x_2\, x_3 + x_2\, x_4 + x_3\, x_4 = \frac{c}{b} > 0$$

with $b = a_0 = 1,$ dan $c = a_2$.

$$x_1x_2x_3 + x_1x_2x_4 + x_1x_3x_4 + x_2x_3x_4 = -\frac{d}{b} < 0$$

with $b = a_0 = 1,$ dan $d = a_3$.

$$x_1x_2x_3x_4 = \frac{e}{b} > 0$$

with $b = a_0 = 1,$ dan $e = a_4$.

Based on the analysis shows that the fourth-degree polynomial satisfies the necessary conditions and, for $R_0 > 1$, has roots with negative real parts. Thus, the endemic equilibrium point $E^*(S^*, V^*, L^*, I^*, M^*, R^*)$ is proven to be locally asymptotically stable when $R_0 > 1$. Biologically, this indicates that each actively infected individual is capable of transmitting the disease to more than one susceptible individual, so that the spread of the disease can persist in the population endemically.

4 Numerical Simulation

The proposed dynamic model of TB disease spread was verified using monthly data from the Demak District Health Office from January 2022 to June 2024. Model parameters were estimated using the Non-Linear Least Squares method, which is a commonly applied method. The parameter estimation results are presented in Table 1.

Table 1. Parameter values in the TB Transmission Model

Parameters	Values	Reference
Λ	122	[estimated]
β	0,001	[estimated]
μ	0,00119	[15]
η	0,3	[estimated]
κ	0,000005	[estimated]
τ	0,1	[estimated]
θ	0,01	[estimated]
ω	0,1	[estimated]
σ	0,02	[estimated]
δ_1	0,005	[estimated]
δ_2	0,002	[estimated]
γ_1	0,00217	[estimated]
γ_2	0,00049	[estimated]

The parameter values listed in Table 1 were substituted into the model, and endemic simulations were performed with the initial conditions $S(0) = 99990, V(0) = 1557, L(0) =$

$858, I(0) = 186, M(0) = 9, and\ R(0) = 168$, we obtain the following dynamic model of TB disease spread.

$$\frac{dS}{dt} = 122 - 0{,}05SI - 0{,}10119S$$
$$\frac{dV}{dt} = 0{,}10S - 0{,}000001VI - 0{,}00119V$$
$$\frac{dL}{dt} = 0{,}05SI + 0{,}00000095VI - 0{,}10119L$$
$$\frac{dI}{dt} = 0{,}05L - 0{,}01536I$$
$$\frac{dM}{dt} = 0{,}01I - 0{,}00268M$$
$$\frac{dR}{dt} = 0{,}00000005VI + 0{,}05L + 0{,}00217I + 0{,}00049M - 0{,}00119R$$

Then, the parameter value is used in calculating the basic reproduction number to determine the number of susceptible individuals that can be infected by one infected individual.

$$R_0 = \frac{\Lambda\theta\big(\beta\mu + \eta\kappa(1-\tau)\big)}{\mu(\eta+\mu)(\theta+\omega+\mu)(\sigma+\gamma_1+\delta_1+\mu)} \approx 2{,}85$$

$R_0 \approx 2{,}85 > 1$ is obtained, this shows that, on average, an infected individual is able to transmit the disease to two susceptible individuals. The following figure shows the dynamics of the SVLIMR model related to the level of TB disease spread.

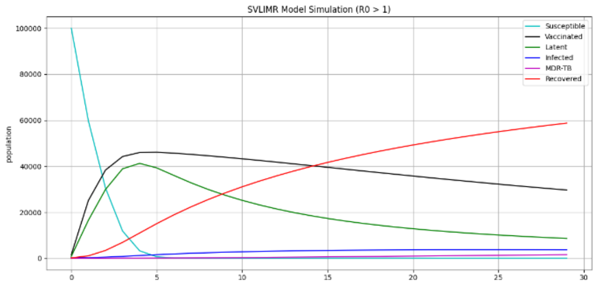

Fig.2. Simulation of TB endemic conditions

Based on the simulation results in Figure 2, the number of susceptible individuals experienced a sharp decline early in the period due to transmission and the vaccination program. The number of vaccinated individuals increased until the fourth month before gradually declining, while the number of latent individuals increased initially and then declined due to the transition to the infected or MDR-TB class. Infected and MDR-TB cases remained low thanks to the implementation of good sanitation, while the number of recovered individuals increased steadily. This pattern confirms that the combination of vaccination and sanitation is effective in suppressing the spread of TB, although the latent and MDR-TB classes still have the potential to be long-term sources of infection.

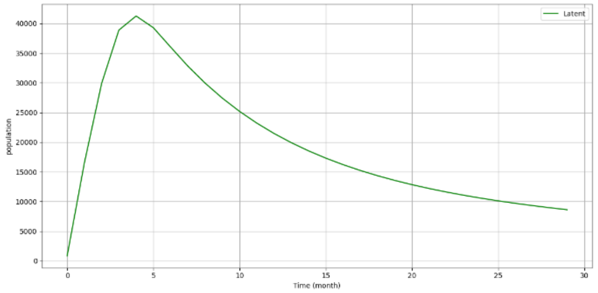

Fig.3. Simulation of the number of latent individuals in TB endemic conditions with $\omega = 10\%$

Figure 3 shows that when the parameter ω is only 10%, the number of individuals in the latent condition increases. This Results in an increase in the number of latent individuals up to the 4th month.

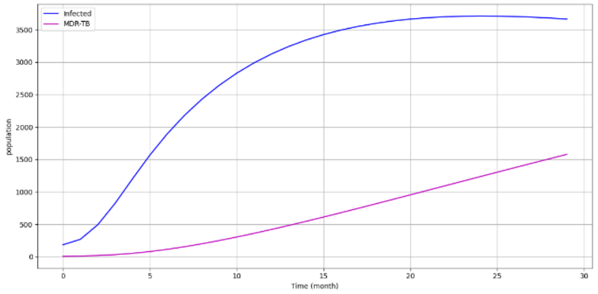

Fig.4. Simulation of the number of infected individuals and MDR-TB in TB endemic conditions

Figure 4 depicts the trend in the number of infected individuals under endemic conditions, where the number continues to increase and reaches its peak in the 25th month and then tends to stabilize, while MDR-TB cases show a consistent, gradual increase over time without appearing to decrease, although at a lower level.

The effect of the parameter value ω (recovery rate due to good sanitation factors) with other parameters remaining constant, namely $\omega = 0.1$; $\omega = 0.35$; $\omega = 0.7$ changes in the population of individuals recovery can be seen in Figure 5.

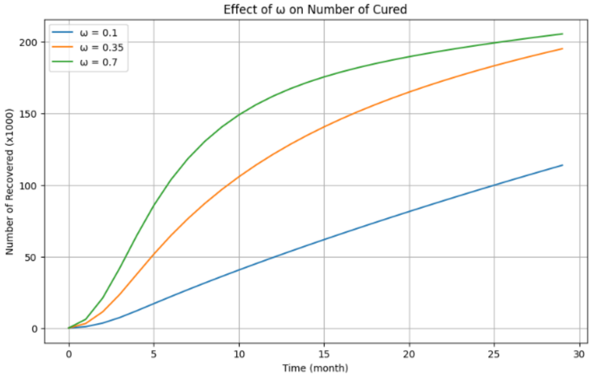

Fig.5. The effect of variations in the parameter ω (the rate of recovery due to good sanitation factors) on the number of people who recovered

Simulation results fig. 5, show that higher ω values lead to an increase in the cumulative number of recovered individuals. This illustrates that improved sanitation, such as the provision of clean water, effective waste management, and a healthy environment, can accelerate the patient recovery process, allowing more individuals to transition into the recovering group over time, in accordance with the objectives, namely that good sanitation can expedite the recovery process.

Numerical simulations showed that the model exhibits an endemic equilibrium. In the initial phase, there is an increase in the number of individuals exposed to and infected with TB, as indicated by a basic reproduction number of 2,85, which means that one infected person can transmit TB to an average of two susceptible individuals. To lower the reproduction number, improving sanitation practices can increase the number of recovered individuals. This means that increasing the number of latent individuals practicing good sanitation can indirectly reduce the number of cases of infection has changed significantly.

From the simulation numeric, by increasing sanitation to 70%, we found that $R0 = 0.2295 < 1$. This condition shows the existence of a non-endemic equilibrium that is asymptotically stable, so that tuberculosis transmission is reduced. This condition is given in Figure 6.

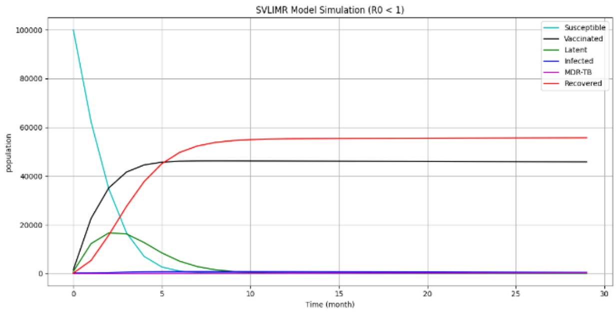

Fig.6. Simulation of non-endemic TB conditions

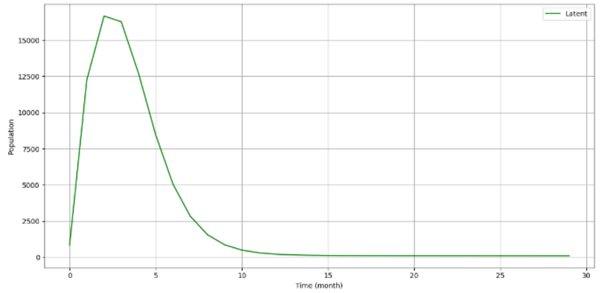

Fig.7. Simulation of the number of latent individuals in non-endemic TB conditions with $\omega = 70\%$

Figure 7 shows that when the ω parameter is 70%, the number of individuals in the latent state decreases. This indicates that increasing the ω value accelerates the transition of individuals from the latent phase, resulting in fewer latent cases in the population.

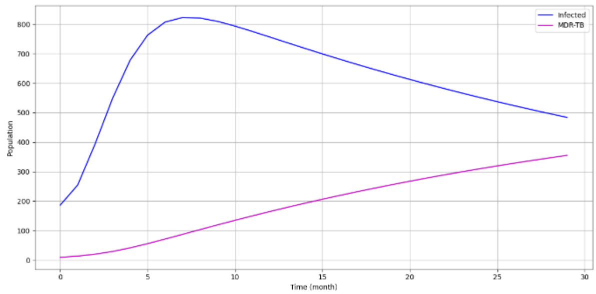

Fig.8. Simulation of the number of infected individuals and MDR-TB in non-endemic TB conditions

Based on Figure 8, it is shown that infected individuals reached a peak in the 7th month and then decreased, while MDR-TB continued to increase gradually throughout the simulation time period.

Thus, the results of this study confirm that improved sanitation, including the provision of clean water, adequate sanitation facilities, and integrated waste management, is a key factor in accelerating patient recovery while suppressing the spread of TB. Therefore, public health policies should prioritize sanitation-based interventions, which can then be integrated with vaccination strategies and other medical efforts to support accelerated TB elimination.

5 Conclusion

The SEIR model was modified to create a $SVLIMR$ model for TB transmission by adding variables for vaccination (V) and drug resistance (M). In this case, the population is divided into six subpopulations: susceptible, vaccinated, latent, infected, infected with drug resistance, and recovered. This TB transmission dynamics model has two equilibrium points: non-endemic and endemic. Local stability analysis at these two points was performed using the Routh-Hurwitz criterion. The results indicate that the non-endemic equilibrium point is locally asymptotically stable if R_0 less than one. Meanwhile, the endemic equilibrium point is locally asymptotically stable if R_0 greater than one.

Next, numerical simulations were conducted to verify the proposed model. The parameter ω, representing the recovery rate due to improved sanitation, has a positive effect on the Increase individual recovery rates, resulting in a significant increase in the number of recovered individuals. This means that overall good sanitation interventions can improve the immune system, suppress the activation of infections, and able to reduce the rate of spread of TB disease. Therefore, public health policies need to prioritize improving sanitation quality, combined with vaccination and treatment, to support TB elimination efforts.

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