

# SEIR Epidemic Model of the Spread of Tuberculosis in Samarinda City with the Addition of Vaccination Parameters

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## ABSTRACT

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Tuberculosis (TB) is a contagious, dangerous disease that infects the human body through the respiratory tract. The city of Samarinda itself is the city with the second highest rate of tuberculosis transmission in East Kalimantan. The aim of this research is to build a SEIR mathematical model of the spread of tuberculosis, analyze the stability of the model, and simulate the model. This research uses the Runge-Kutta method, which has high accuracy in estimating solutions and is relatively stable. This data is secondary data obtained through BPS as well as articles from previous researchers. The results of the research showed that the percentage of increase in the spread of the virus in the susceptible population (S) during the first week was 2,048%, then the 4th week to the 100th day decreased by approximately 164,100%, then the percentage of the population that was exposed but did not spread it (E) decreased in the first week, namely 34,525%, then in the 4th week to the 100th day there was a very significant decrease, namely 7,600%, then the percentage of the population infected and infectious (I) in the first week rose to 19,138%, then in the 4th week to the 2nd week -100 experienced a decrease of approximately 716,900%, and finally, the percentage of the population who recovered in the first week began to increase due to the very influential vaccine, namely increasing by 26,860%, then the 4th week to the 100th week also experienced a significant increase, namely by 81.681%.

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## I. Introduction

Tuberculosis (TB) is a contagious, dangerous disease that infects the human body through the respiratory tract. This disease is caused by the bacteria *Mycobacterium tuberculosis* (Mtb), which is transmitted through the air [1]. This infection is caused by inhaling saliva containing the bacteria *Mycobacterium tuberculosis*. Transmission is usually through exposure to tuberculosis bacilli through fluids from people with pulmonary TB while talking, coughing, or sneezing. Common symptoms are cough, fever, hemoptysis, chest pain, fatigue, and weight loss [2].

East Kalimantan is a province in Indonesia with a significant increase in BTA (+) TB cases, ranking 17th in the notification of TB cases per 100,000 population in Indonesia in 2016, an increase of 7 places compared to 2012. The highest prevalence of pulmonary tuberculosis sufferers in Samarinda City is 457 people per year [3]. The first factor that causes tuberculosis is the habit of smoking, which can reduce the body's immune system, making it easy to get the disease, especially in men who have a smoking habit [4]. The second factor is the level of knowledge. The level of knowledge plays an important role in fighting tuberculosis. A person's understanding and knowledge about TB include the routes of transmission and the risks a person faces when taking preventive measures. Therefore, a better understanding of the factors that prevent TB transmission in TB patients is important to reduce the transmission of this infection [5].

Mathematical models for epidemic disease cases cannot accurately describe all aspects of the original epidemic, but mathematical modeling can provide good hope for comparing strategies that



can be used to reduce the rate of infection. Although the mathematical model is unable to predict and control epidemic diseases in the future [6].

Several researchers have also conducted research on models of the spread of tuberculosis ([7]; [8]; [9]; [10]). [7] developed the SIR mathematical model for the spread of tuberculosis by taking into account transmigration factors. Initially, [9] developed the SEIR mathematical model by taking into account treatment factors. [9] discuss the formation and analysis of mathematical models for the transmission of tuberculosis, which focus on cases of detection and treatment. This research makes modifications to the model from previous research. This model was developed from the SEIR model researched by Das (2021) [11].

In 2020, the city of Samarinda will be one of the cities with the largest spread of TB disease in East Kalimantan, which is second after Balikpapan. Therefore, in this article, researchers want to model the SEIR Epidemic and simulate a model of the spread of TB disease in Samarinda using the Runge-Kutta method of order 4 by adding vaccination parameters or vaccination therapy to test whether adding vaccination parameters to the susceptible population (S) can have a significant effect. significant impact on the level of TB spread in Samarinda city. Then, in this article, we will compare the level of TB spread in Samarinda without and using vaccination therapy.

The model is structured in the form of a system of differential equations that depends on the variables that represent each population. Next, parameter analysis was carried out and looked for the basic reproduction ratio ( $R_0$ ) and for numerical simulation using the Runge-Kutta Order 4 Method. The choice of this method was based on the fact that it has high accuracy in estimating solutions to differential equations and is relatively stable. The difference between this article and other articles lies in the parameters, where this article adds vaccination parameters to the distribution model. The aim of this research is to build a SEIR mathematical model of the spread of tuberculosis, analyze the stability of the model, and simulate the model.

## II. Method

This research began with a library and literature study to build a mathematical model that represents the dynamics of the spread of tuberculosis in the population of Samarinda City. The data used is secondary data from the BPS (Central Statistics Agency) and news articles, which are data on the population of people suffering from tuberculosis and the number of people who were vaccinated against tuberculosis in 2021. Several methods are used to study the SEIR model with vaccination parameters, including the New Generation Method, Matrix, linearization, stability analysis, the Runge-Kutta method, and numerical simulation for solution visualization with the Matlab program.

The amount  $R_0$  is defined as the expected number of secondary cases resulting from one patient who has the ability to transmit the disease when he enters a population that is all healthy during the infectious period or infection period. In other words, this quantity is a multiple factor (multiplication factor) of the initial case (primary case). This quantity can also be defined as the expected number of secondary cases per primary case in a 'virgin' population [12].

In the epidemic model, it is known that an infection will become extinct if  $R_0 < 1$ ; while the infection continues if  $R_0 > 1$ . Based on this concept, to determine long-term behavior in suspect populations, it is necessary to calculate the basic reproduction number ( $R_0$ ). The basic reproduction number is the expected number of infections or the number of new infections resulting from one initial infection that appears in a susceptible population. Therefore, calculating  $R_0$  involves multiplying the infection rate by the duration of infection. The method used to determine the basic reproduction number ( $R_0$ ) is the Next Generation Matrix Method [13].

In very large populations with a relatively smaller total infected population, it can be assumed that the number of susceptible populations is constant. In this case, the calculation of the next generation matrix can be done with  $FV^{-1}$ , where the entries in this matrix represent the expected value of infection generated in the  $i$  compartment by the  $j$  compartment. The New Generations Matrix method has positive eigenvalues with a modulus at least the same as the modulus of the other eigenvalues. On this basis, this positive eigenvalue can be a candidate for the basic reproduction number  $R_0$  [13].

The Runge-Kutta method is a method commonly used in solving ordinary differential equations. The calculations in this method have four function evaluations in one iteration process [14]. The Runge-Kutta method is another alternative to other numerical methods that does not require derivative calculations. This method seeks to obtain a higher degree of accuracy and, at the same time, avoids the need to look for higher derivatives ([15];[16]).

### III. Results and Discussion

#### A. Mathematical Model

In the SEIR model of the spread of tuberculosis in Samarinda City, the human population is divided into sub-populations, namely: The susceptible (S) class states the number of individuals who are susceptible to tuberculosis; the exposed (E) class states individuals who are infected with tuberculosis but cannot transmit it; the infected (I) class represents the number of individuals who are infected and can transmit tuberculosis; and the recovered (R) class represents individuals who have recovered from tuberculosis. The model formulation in this study adds the parameter that susceptible humans can recover and be immune from contracting tuberculosis with vaccine therapy ( $v$ ).

In this research, the model formulation will first be determined, and the steps taken are:

- Determine a system of differential equations based on the growth rate per unit of time for each subpopulation S, E, I, and R by adding the parameter  $v$  (therapy using vaccines) involving the total  $R_0$ .
- Determine the general form of the basic reproduction number  $R_0$  and the basic reproduction number with  $R_r$  therapy.

Then, after determining the model formulation, numerical simulations were carried out using the Runge-Kutta method of order 4, with the aim of finding out how the distribution affects the model that has been developed and analyzing the stability of the model. The SEIR model transmission scheme for TB in Samarinda can be seen in Figure 1 as follows:

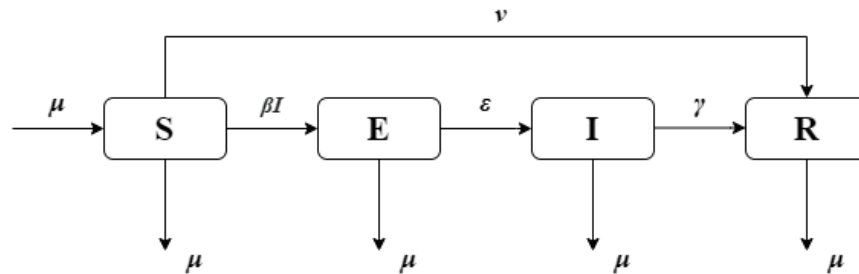


Fig 1. Scheme of Transmission Model for TB

Based on the model diagram image, a SEIR model for the spread of TB is obtained by considering vaccination as a model parameter in the form of a system of non-linear ordinary differential equations consisting of four dependent variables, namely S, E, I, and R, and one independent variable, namely t, as follows:

$$\frac{dS}{dt} = \mu N - \left( \beta \frac{I}{N} + \mu + v \right) S \quad (1)$$

$$= \mu N - \frac{\beta IS}{N} - \mu S - vS$$

$$\frac{dE}{dt} = \beta \frac{I}{N} S - (\varepsilon + \mu) E \quad (2)$$

$$= \beta \frac{I}{N} S - \varepsilon E - \mu E$$

$$\frac{dI}{dt} = \varepsilon E - (\gamma + \mu) I \quad (3)$$

$$= \varepsilon E - \gamma I - \mu I$$

$$\frac{dR}{dt} = \gamma I - \mu R + vS \tag{4}$$

where:

- $N(t)$  : Total population ( $S+E+I+R$ ) per unit time
- $\mu$  : Population rate (natural births/deaths)
- $\beta$  : Tuberculosis transmission rate
- $\varepsilon$  : rate of change in susceptibility to becoming infected with the virus
- $\gamma$  : healing rate of those infected with the tuberculosis virus
- $v$  : rate of vaccine administration to vulnerable populations

For example:

$$s = \frac{S}{N}; e = \frac{E}{N}; i = \frac{I}{N}; r = \frac{R}{N}$$

Where:  $N = s + e + i + r = 1$ . The previous equation model can be stated as follows:

$$\frac{ds}{dt} = \mu - (\beta i + \mu + v)s \tag{5}$$

$$= \mu - \beta is - \mu s - vs$$

$$\frac{de}{dt} = \beta is - (\varepsilon + \mu)e \tag{6}$$

$$= \beta is - \varepsilon e - \mu e$$

$$\frac{di}{dt} = \varepsilon e - (\gamma + \mu)i \tag{7}$$

$$= \varepsilon e - \gamma i - \mu i$$

$$\frac{dr}{dt} = \gamma i - \mu r + vs \tag{8}$$

*B. Equilibrium Point and Stability*

To determine two equilibrium points, each equation in the system of equations (5) and (8) must be equal to zero, or  $(\frac{ds}{dt}, \frac{de}{dt}, \frac{di}{dt}, \frac{dr}{dt}) = (0,0,0,0)$ , so we get:

$$\mu - (\beta i + \mu + v)s = 0 \tag{9}$$

$$\beta is - (\varepsilon + \mu)e = 0 \tag{10}$$

$$\varepsilon e - (\gamma + \mu)i = 0 \tag{11}$$

$$\gamma i - \mu r + vs = 0 \tag{12}$$

Next, it can be determined Disease-free Equilibrium Point

*C. Disease-free Equilibrium Point and Its Stability*

The disease-free equilibrium point is a steady-state solution where there is no TB disease in the population. So, no one is exposed or infected; this means  $e = i = 0$ . So, by solving equations (5) and (8), the TB disease-free equilibrium point is obtained:

$$E_0 = \left( \frac{\mu}{\mu + v}, 0, 0, \frac{v}{\mu + v} \right)$$

#### D. Endemic Equilibrium Point

The endemic equilibrium point  $E^* = (s^*, e^*, i^*, r^*)$  is a steady-state solution that shows the possibility of the spread of a disease. This results in the population  $S \neq 0, E \neq 0, I \neq 0,$  and  $R \neq 0$ . From Equations (5) – (8), the endemic equilibrium point is obtained as follows:

$$\begin{aligned} E^* &= (s^*, e^*, i^*, r^*) \\ &= \left( \frac{\mu}{R_0(\mu + v)}, \frac{\mu(R_0 - 1)}{R_0(\varepsilon + \mu)}, \frac{(\mu + v)(R_0 - 1)}{\beta}, \frac{\gamma(R_0 - 1)(\mu + v)^2 + \beta\mu v}{\beta\mu R_0(\mu + v)} \right) \end{aligned}$$

#### E. New Generation Matrix Method

New Generation Matrix method for linearization of infected subsystems, namely  $i$  and  $e$

$$\begin{aligned} J(E, I) &= \begin{pmatrix} \frac{\partial}{\partial i} (\beta is - \varepsilon e - \mu e) & \frac{\partial}{\partial e} (\beta is - \varepsilon e - \mu e) \\ \frac{\partial}{\partial i} (\varepsilon e - \gamma i - \mu i) & \frac{\partial}{\partial e} (\varepsilon e - \gamma i - \mu i) \end{pmatrix} \\ &= \begin{pmatrix} \beta s & -\varepsilon - \mu \\ -\gamma - \mu & \varepsilon \end{pmatrix} \end{aligned}$$

- Substitution to equilibrium point

$$\begin{aligned} J\left(\frac{\mu}{\mu + v}, 0, 0, \left(\frac{v}{\mu + v}\right)\right) &= \begin{pmatrix} \frac{\beta\mu}{\mu + v} & -\varepsilon - \mu \\ -\gamma - \mu & \varepsilon \end{pmatrix} \\ &= \begin{pmatrix} \frac{\beta\mu}{\mu + v} & -\varepsilon - \mu \\ -\gamma - \mu & \varepsilon \end{pmatrix} \end{aligned}$$

- Be Composed  $J = F - V$

$$J = F - V = \begin{pmatrix} \frac{\beta\mu}{\mu + v} & 0 \\ 0 & 0 \end{pmatrix} - \begin{pmatrix} 0 & \varepsilon + \mu \\ \gamma + \mu & -\varepsilon \end{pmatrix}$$

- Calculate  $R_0$  with  $R_0 = \rho(F \cdot V^{-1})$

$$\begin{aligned} R_0 &= \rho\left(\begin{pmatrix} \frac{\beta\mu}{\mu + v} & 0 \\ 0 & 0 \end{pmatrix} \left(-\frac{1}{(\gamma + \mu)(\varepsilon + \mu)} \begin{pmatrix} -\varepsilon & -\varepsilon - \mu \\ -\gamma - \mu & 0 \end{pmatrix}\right)\right) \\ &= \rho\left(-\frac{1}{(\gamma + \mu)(\varepsilon + \mu)} \begin{pmatrix} -\frac{\beta\mu\varepsilon}{\mu + v} & -\frac{\beta\mu(\varepsilon + \mu)}{\mu + v} \\ 0 & 0 \end{pmatrix}\right) \\ &= \rho\left(\frac{1}{(\gamma + \mu)(\varepsilon + \mu)} \begin{pmatrix} \frac{\beta\mu\varepsilon}{\mu + v} & \frac{\beta\mu(\varepsilon + \mu)}{\mu + v} \\ 0 & 0 \end{pmatrix}\right) \\ &= \rho\left(\begin{pmatrix} \frac{\beta\mu\varepsilon}{(\gamma + \mu)(\varepsilon + \mu)(\mu + v)} & \frac{\beta\mu(\varepsilon + \mu)}{(\gamma + \mu)(\varepsilon + \mu)(\mu + v)} \\ 0 & 0 \end{pmatrix}\right) \end{aligned}$$

$$= \rho \left( \begin{array}{cc} \frac{\beta\mu\varepsilon}{(\gamma+\mu)(\varepsilon+\mu)(\mu+v)} & \frac{\mu\beta}{(\gamma+\mu)(\mu+v)} \\ 0 & 0 \end{array} \right)$$

• The largest eigenvalue of the  $R_0$  matrix will be calculated

$$|A - \lambda I| = 0$$

$$\left| \begin{array}{cc} \frac{\beta\mu\varepsilon}{(\gamma+\mu)(\varepsilon+\mu)(\mu+v)} - \lambda & \frac{\mu\beta}{(\gamma+\mu)(\mu+v)} \\ 0 & 0 - \lambda \end{array} \right| = 0$$

$$-\lambda \left( \frac{\beta\mu\varepsilon}{(\gamma+\mu)(\varepsilon+\mu)(\mu+v)} - \lambda \right) = 0$$

then,

$$\lambda_1 = 0, \lambda_2 = \frac{\beta\mu\varepsilon}{(\gamma+\mu)(\varepsilon+\mu)(\mu+v)}$$

So, we get value  $R_0$ :

$$R_0 = \frac{\beta\mu\varepsilon}{(\gamma + \mu)(\varepsilon + \mu)(\mu + v)} \tag{13}$$

F. Model Stability Analysis

$$J = \begin{pmatrix} -(\beta i + \mu + v) & 0 & -\beta s & 0 \\ \beta i & -(\varepsilon + \mu) & \beta s & 0 \\ 0 & \beta & -(\gamma + \mu) & 0 \\ v & 0 & \gamma & -\mu \end{pmatrix}$$

G. Model Stability Analysis at the Disease-Free Equilibrium Point

Based on the Jacobian matrix in the equation above, the stability of the model at the disease-free equilibrium point ( $E_0$ ) is then determined.

$$J_{E_0} = \begin{pmatrix} -(\mu + v) & 0 & \frac{-\beta\mu}{\mu + v} & 0 \\ 0 & -(\varepsilon + \mu) & \frac{\beta\mu}{\mu + v} & 0 \\ 0 & \varepsilon & -(\gamma + \mu) & 0 \\ v & 0 & \gamma & -\mu \end{pmatrix}$$

By using the characteristic equation, namely

$$|J_{E_0} - \lambda I| = (\mu + \lambda)(\mu + v + \lambda) \left\{ (\varepsilon + \mu + \lambda)(\gamma + \mu + \lambda) - \frac{\alpha\beta\mu}{\mu + v} \right\} = 0$$

Then the eigenvalues are obtained as follows:

$$\lambda_1 = -\mu < 0; \lambda_2 = -(\mu + v) < 0$$

or

$$(\beta + \mu + \lambda)(\mu_1 + \delta + \mu + \lambda) - \alpha\beta\mu\mu + v = 0$$

The above equation is used to determine the values of  $\lambda_3$  and  $\lambda_4$ , ie

$$a_0\lambda + a_1\lambda + a_2 = 0$$

with:

$$\begin{aligned}
 a_0 &= 1 > 0 \\
 a_1 &= \varepsilon + \sigma + 2\mu > 0 \\
 a_2 &= (\varepsilon + \mu)(\gamma + \mu) - (\beta\varepsilon\mu + v) \\
 &= (\varepsilon + \mu)(\gamma + \mu)(1 - R_0) > 0, \text{ jika } R_0 < 1
 \end{aligned}$$

Based on the previous equation, by using the Routh-Hurwitz criterion, it can be seen that the real part of  $\lambda_3$  and  $\lambda_4$  is negative with the condition that  $R_0 < 1$ . Then, because the real part of  $\lambda_1$ ,  $\lambda_2$ ,  $\lambda_3$ , and  $\lambda_4$  is negative, then based the stability analysis it is found that the disease-free equilibrium is stable provided that  $R_0 < 1$ .

The stability of the model at the endemic equilibrium point ( $E^*$ ) is determine follows.

$$J_{E^*} = \begin{pmatrix} -((\mu + v)R_0) & 0 & \frac{-\beta\mu}{R_0(\mu + v)} & 0 \\ (\mu + v)(R_0 - 1) & -(\varepsilon + \mu) & \frac{\beta\mu}{R_0(\mu + v)} & 0 \\ 0 & \varepsilon & -(\gamma + \mu) & 0 \\ v & 0 & \gamma & -\mu \end{pmatrix}$$

By using the characteristic equation, namely

$$|JE^* - \lambda I| = (\mu + \lambda) \{ [((\mu + v)R_0 + \lambda)(\varepsilon + \mu + \lambda)(\gamma + \mu + \lambda)] - [\beta\varepsilon\mu R_0 + \beta\varepsilon\mu\lambda R_0(\mu + v)] \} = 0,$$

From Equation (17), assume:

$$A = ((\mu + v)R_0) > 0;$$

$$B = (\beta + \mu) > 0;$$

$$C = (\gamma + \mu) > 0;$$

$$D = \beta\varepsilon\mu R_0(\mu + v) > 0; \text{ and}$$

$$E = \beta\varepsilon\mu R_0 > 0, \text{ So:}$$

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

With:

$$a_0 = 1 > 0;$$

$$a_1 = (A + B + C) > 0;$$

$$a_2 = AB + AC + BC - D;$$

$$a_3 = (ABC - E) > 0, \text{ if } R_0 > 1$$

Based on the previous equation using the Routh-Hurwitz criterion, it can be seen that the real part of  $\lambda_2$ ,  $\lambda_3$ , and  $\lambda_4$  is negative with the condition that  $R_0 > 1$ . Because the real part of  $\lambda_1$ ,  $\lambda_2$ ,  $\lambda_3$ , and  $\lambda_4$  is negative, then based on the stability analysis, it is obtained that the endemic equilibrium point is stable, provided that  $R_0 > 1$ .

**Runge-Kutta Orde 4 Method**

The solution to the TBC spread model is sought using the 4th Order Runge-Kutta Method, namely,  $Y_{i+1} = Y_i + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4)$ , so that:

$$S_{i+1} = S_i + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4) \tag{14}$$

$$E_{i+1} = E_i + \frac{1}{6}(l_1 + 2l_2 + 2l_3 + l_4) \tag{15}$$

$$I_{i+1} = I_i + \frac{1}{6}(m_1 + 2m_2 + 2m_3 + m_4) \tag{16}$$

$$R_{i+1} = R_i + \frac{1}{6}(n_1 + 2n_2 + 2n_3 + n_4) \tag{17}$$

With,

$$\begin{aligned} k_1 &= h * f(t_i, S_i, E_i, I_i, R_i) \\ &= h * \left( \mu N - \frac{\beta I_i S_i}{N} - \mu S_i - v S_i \right) \end{aligned} \tag{18}$$

$$\begin{aligned} l_1 &= h * g(t_i, S_i, E_i, I_i, R_i) \\ &= h * \left( \beta \frac{I_i S_i}{N} - \varepsilon E_i - \mu E_i \right) \end{aligned} \tag{19}$$

$$\begin{aligned} m_1 &= h * b(t_i, S_i, E_i, I_i, R_i) \\ &= h * (\varepsilon E_i - \gamma I_i - \mu I_i) \end{aligned} \tag{20}$$

$$\begin{aligned} n_1 &= h * d(t_i, S_i, E_i, I_i, R_i) \\ &= h * (\gamma I_i - \mu R_i + v S_i) \end{aligned} \tag{21}$$

$$\begin{aligned} k_2 &= h * f\left(t_i + \frac{h}{2}, S_i + K_1 \frac{h}{2}, E_i + l_1 \frac{h}{2}, I_i + m_1 \frac{h}{2}, R_i + n_1 \frac{h}{2}\right) \\ &= h * \left( \mu N - \frac{\beta(l_i + m_1 \frac{h}{2})(S_i + K_1 \frac{h}{2})}{N} - \pi\left(S_i + K_1 \frac{h}{2}\right) - v\left(S_i + K_1 \frac{h}{2}\right) \right) \end{aligned} \tag{22}$$

$$\begin{aligned} l_2 &= h * g\left(t_i + \frac{h}{2}, S_i + K_1 \frac{h}{2}, E_i + l_1 \frac{h}{2}, I_i + m_1 \frac{h}{2}, R_i + n_1 \frac{h}{2}\right) \\ &= h * \left( \frac{\beta(l_i + m_1 \frac{h}{2})(S_i + K_1 \frac{h}{2})}{N} - \varepsilon\left(E_i + l_1 \frac{h}{2}\right) - \mu\left(E_i + l_1 \frac{h}{2}\right) \right) \end{aligned} \tag{23}$$

$$\begin{aligned} m_2 &= h * b\left(t_i + \frac{h}{2}, S_i + K_1 \frac{h}{2}, E_i + l_1 \frac{h}{2}, I_i + m_1 \frac{h}{2}, R_i + n_1 \frac{h}{2}\right) \\ &= h * \left( \varepsilon\left(E_i + l_1 \frac{h}{2}\right) - \gamma\left(I_i + m_1 \frac{h}{2}\right) - \mu\left(I_i + m_1 \frac{h}{2}\right) \right) \end{aligned} \tag{24}$$

$$\begin{aligned} n_2 &= h * d\left(t_i + \frac{h}{2}, S_i + K_1 \frac{h}{2}, E_i + l_1 \frac{h}{2}, I_i + m_1 \frac{h}{2}, R_i + n_1 \frac{h}{2}\right) \\ &= h * \left( \gamma\left(I_i + m_1 \frac{h}{2}\right) - \mu\left(R_i + n_1 \frac{h}{2}\right) + v\left(S_i + K_1 \frac{h}{2}\right) \right) \end{aligned} \tag{25}$$

$$\begin{aligned}
k_3 &= h * f \left( t_i + \frac{h}{2}, S_i + K_2 \frac{h}{2}, E_i + l_2 \frac{h}{2}, l_i + m_2 \frac{h}{2}, R_i + n_2 \frac{h}{2} \right) \\
&= h * \left( \mu N - \frac{\beta(l_i+m_2 \frac{h}{2})(S_i+K_2 \frac{h}{2})}{N} - \pi \left( S_i + K_1 \frac{h}{2} \right) - V \left( S_i + K_1 \frac{h}{2} \right) \right)
\end{aligned} \tag{26}$$

$$\begin{aligned}
l_3 &= h * g \left( t_i + \frac{h}{2}, S_i + K_2 \frac{h}{2}, E_i + l_2 \frac{h}{2}, l_i + m_2 \frac{h}{2}, R_i + n_2 \frac{h}{2} \right) \\
&= h * \left( \frac{\beta(l_i+m_2 \frac{h}{2})(S_i+K_2 \frac{h}{2})}{N} - \varepsilon \left( E_i + l_2 \frac{h}{2} \right) - \mu \left( E_i + l_2 \frac{h}{2} \right) \right)
\end{aligned} \tag{27}$$

$$\begin{aligned}
m_3 &= h * b \left( t_i + \frac{h}{2}, S_i + K_2 \frac{h}{2}, E_i + l_2 \frac{h}{2}, l_i + m_2 \frac{h}{2}, R_i + n_2 \frac{h}{2} \right) \\
&= h * \left( \varepsilon \left( E_i + l_2 \frac{h}{2} \right) - \gamma \left( l_i + m_2 \frac{h}{2} \right) - \mu \left( l_i + m_2 \frac{h}{2} \right) \right)
\end{aligned} \tag{28}$$

$$\begin{aligned}
n_3 &= h * d \left( t_i + \frac{h}{2}, S_i + K_2 \frac{h}{2}, E_i + l_2 \frac{h}{2}, l_i + m_2 \frac{h}{2}, R_i + n_2 \frac{h}{2} \right) \\
&= h * \left( \gamma \left( l_i + m_2 \frac{h}{2} \right) - \mu \left( R_i + n_2 \frac{h}{2} \right) + v \left( S_i + K_2 \frac{h}{2} \right) \right)
\end{aligned} \tag{29}$$

$$\begin{aligned}
k_4 &= h * f \left( t_i + \frac{h}{2}, S_i + K_3 \frac{h}{2}, E_i + l_3 \frac{h}{2}, l_i + m_3 \frac{h}{2}, R_i + n_3 \frac{h}{2} \right) \\
&= h * \left( \mu N - \frac{\beta(l_i+m_3 \frac{h}{2})(S_i+K_3 \frac{h}{2})}{N} - \pi(S_i + K_3 h) - v(S_i + K_3 h) \right)
\end{aligned} \tag{30}$$

$$\begin{aligned}
l_4 &= h * g(t_i + h, S_i + K_3 h, E_i + l_3 h, l_i + m_3 h, R_i + n_3 h) \\
&= h * \left( \beta \frac{(l_i+m_3 h)(S_i+K_3 h)}{N} - \varepsilon(E_i + l_3 h) - \mu(E_i + l_3 h) \right)
\end{aligned} \tag{31}$$

$$\begin{aligned}
m_4 &= h * b(t_i + h, S_i + K_3 h, E_i + l_3 h, l_i + m_3 h, R_i + n_3 h) \\
&= h * (\varepsilon(E_i + l_3 h) - \gamma(l_i + m_3 h) - \mu(l_i + m_3 h))
\end{aligned} \tag{32}$$

$$\begin{aligned}
n_4 &= h * d(t_i + h, S_i + K_3 h, E_i + l_3 h, l_i + m_3 h, R_i + n_3 h) \\
&= h * (\gamma(l_i + m_3 h) - \mu(R_i + n_3 h) + v(S_i + K_3 h))
\end{aligned} \tag{33}$$

where  $S_{i+1}, E_{i+1}, I_{i+1}, R_{i+1}$  respectively are equations that will later be used in determining the susceptible population, exposed population, infected population, and recovered population in the next iteration.  $k_1, k_2, k_3, k_4, l_1, l_2, l_3, l_4, m_1, m_2, m_3, m_4, n_1, n_2, n_3, n_4$  are constants in the fourth order runge-kutta formula.  $k_1, k_2, k_3, k_4$  are constants from equations (18), (22), (26), and (30).  $l_1, l_2, l_3, l_4$  are constants from equations (19), (23), (27), and (31).  $m_1, m_2, m_3, m_4$  are constants from equations (20), (24), (28), and (32).  $n_1, n_2, n_3, n_4$  are constants from equations (21), (25), (29), and (33).

### Simulation of model

The time required to complete the model is 100 days with a time interval and step distance of  $h=0.1$  days, so that iteration is carried out 1000 times. Where the calculation for each iteration uses the Matlab program which is then simulated in graphic form.

The initial values and parameters used are as follows:

Table 1. Initial Values of Model Variables

Variable	Value
$N_0$	825.490 people
$S_0$	268.284 people
$E_0$	557.206 people
$I_0$	191 people
$R_0$	40 people

The parameters used for the SEIR model simulation are given in Table 2, with the interval [0,100] and  $h = 0.1$ .

Table 2. Parameter of Model SEIR

$\mu$	$\beta$	$\epsilon$	$\gamma$	$\nu$
0.001	0.2	0.1	0.05	0.5

A model simulation of the spread of tuberculosis in Samarinda City was carried out after carrying out model analysis, variable identification, and data collection. The model formed is then analyzed. After analyzing the model, variable identification is carried out. The data that has been obtained is then obtained and simulated, as seen in Figures 2 and 3.

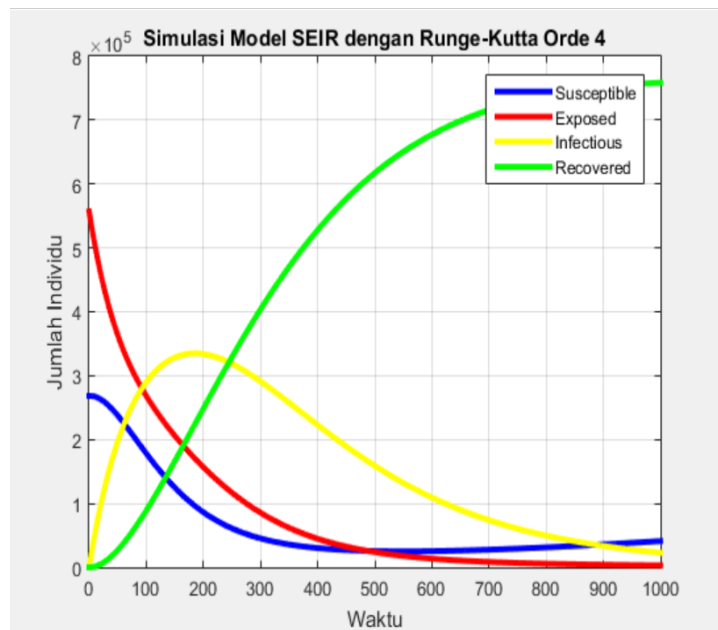


Fig 2. SEIR Model Simulation Without Adding Vaccination Parameters

In Figure 2, it is explained that the x-axis represents time in 100 days containing 1000 iterations, while the y-axis depicts the number of individuals in each category: susceptible (S), exposed (E), infected (I), and recovered (R) with the colors are blue, red, yellow, and green, respectively. With a population of 825,490 people. At the beginning of data entry, the number of susceptible individuals was 268,284, the number of exposed individuals was 557,206, the number of infected individuals was 191, and the number of recovered individuals was 40.

In the first week, the number of individuals susceptible to the virus was 238,720, which decreased. Meanwhile, the number of individuals who have been exposed but have not shown symptoms (exposed), 366,320 people, has also decreased. However, the number of infected individuals reached 194,400, which has increased due to the spread of the virus. On the other hand, individuals who have recovered also experience an increase, even though it is at a normal level. This is caused by handling without additional vaccination. The number of individuals who have recovered has reached around 26,284 people.

On the 100th day, the number of susceptibles reached 41,083 people, which experienced a decrease; the number of exposed people reached around 3,109 people, which experienced a decrease; the number of infected people reached 23,013 people, which experienced a decrease; and the number of recovered people reached 758,490 people, which experienced an increase.

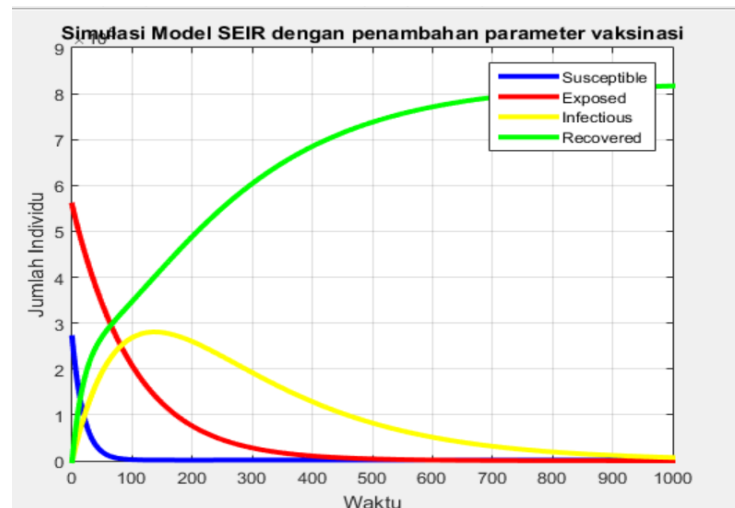


Fig 3. SEIR Model Simulation with Addition of Vaccination Parameters

In Figure 3, it is explained that the  $x$ -axis represents time in 100 days containing 1000 iterations, while the  $y$ -axis depicts the number of individuals in each category: susceptible (S), exposed (E), infected (I), and recovered (R) with the colors are blue, red, yellow, and green, respectively. With a population of 825,490 people. At the beginning of data entry, the number of susceptible individuals was 268,284, the number of exposed individuals was 557,206, the number of infected individuals was 191, and the number of recovered individuals was 40.

In the first week, the number of individuals who were susceptible, as many as 20,484 people, experienced a significant decrease, while individuals in the exposed stage, as many as 345,250 people, experienced a decrease. The number of infected individuals is 191,380, which has increased due to the spread of the virus. On the other hand, recovered individuals experienced a significant increase, reaching 268,600 people.

On the 100th day, the situation changed, namely the number of susceptible of around 1,641 people decreased, and this was probably caused by several people recovering from the disease. The number of exposed people was around 76, a significant decrease, and individuals in this exposed stage had entered the infected stage or had recovered over time. The number of infected people, around 7,169, has decreased, and this could be due to recovery, and the number of recovered people has experienced a significant increase of around 816,810 people. This suggests that many infected individuals recover from the disease and are no longer susceptible to infection. Reasons for this significant increase may include recovery from previously infected individuals and individuals who received vaccinations before becoming infected.

#### IV. Conclusion

Tuberculosis (TB) is a contagious, dangerous disease that can infect the human respiratory tract. This disease is caused by the bacteria *Mycobacterium tuberculosis* (Mtb), which is transmitted through the air. The methods used to study the SEIR model with vaccination parameters are the New Generation Matrix Method, linearization, stability analysis, Runge Kutta Method, and numerical

simulation for solution visualization with the Matlab program. With a population of 825,490 people, calculation results were obtained using the Matlab program, namely that at the beginning of data entry, the number of susceptible individuals was 268,284 people, the number of exposed individuals was 557,206 people, there were 191 infected individuals, and there were 40 recovered individuals. The main difference between those who use vaccination parameters and those who do not use vaccination parameters lies in recovering, so that the chance of recovery using vaccines is greater than that of those who do not use vaccines, which occurs because the death parameters in the population are not calculated.

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