The Impact of Relapse Rate on Deterministic Epidemiological Models with Pseudo-recovery

Anisa Fatta Qunia*, Ali Kusnanto, Paian Sianturi
Department of Mathematics, Faculty of Mathematics and Natural Science, IPB University, Bogor, West Java 16680, INDONESIA

*E-mail: anisa_fatta@apps.ipb.ac.id

Abstract. The deterministic epidemiological model with pseudo-recovery called the SEIRI model is a model that describes disease transmission in a population. Pseudo-recovery is a term for individuals who have recovered of infection, but some of them might be re-infected. This research aims to reconstruct the model, to analyse stability of fixed point and sensitivity of parameters. Also, to carry out numerical simulations upon combination of parameter values. The stability itself was determined using the Lyapunov functions. In this work, the sensitivity analysis was focussed on the effects of the effective contact rate and relapse rate on basic reproduction numbers. Both the effective contact rate and relapse rate increase with basic reproduction number. This may suggest that controlling the spread of the diseases can be done by decreasing both the effective contact rate or the relapse rate.

Keywords: basic reproduction number, Lyapunov, SEIRI, sensitivity analysis

1. Introduction
Disease is one of the factors that affect human health. The disease causes the body to become abnormal resulting in discomfort and dysfunction in the individual’s body. The disease can arise due to an unhealthy environment. One of the classifications of diseases that cause death is an infectious disease. Infectious disease is a disease caused by biological agents such as viruses, bacteria, or parasites [11].

There are several types of infectious diseases, including malaria, herpes, tuberculosis, and others. Each disease spreads in different ways. Therefore we need a model to depict transmission of infectious diseases in a population. This model is called a deterministic epidemiological model [8].

In [4], the population in epidemiological model is divided into three classes based on disease status, there are Susceptible (individuals who are susceptible to disease), Infectious (individuals who have infected the disease), and Recovery (individuals who are recovered from the disease and get immunity permanent) known as SIR epidemiological model. Based on this model, individuals who are in the Recovery class are considered not to be infected again by the disease because they have permanent immunity. But in the reality, many people can still be infected with the disease even after recovering. Therefore it is necessary to develop a more realistic model to learn the transmission of disease in the presence of a recurrence. Relapse is a condition where the signs and symptoms of a disease reappear after a period of healing. This condition is referred to as pseudo recovery that occurs due to incomplete treatment of the disease [8].

[11]
Recovery is a stage where the body from the infected phase becomes free. This recovery causes the individual to recover from the disease and will get a permanent body system. Pseudo recovery is a stage where the individual’s body condition has recovered from the disease, there is still the possibility of relapse of the disease. Therefore, in this study, the authors are interested in analyzing a deterministic epidemiological model with pseudo-recovery using the SEIRI model because it is suspected that incomplete treatment can cause a person to relapse or be re-infected by a disease. The assumptions used in this study are the birth rate equal to mortality rate, the number of populations is constant, there is a latent period, and individuals who have recovered can be re-infected.

2. Deterministic Epidemiological Model with Pseudo-recovery

In this study, a deterministic epidemiological model with pseudo-recovery will be discussed [8]. Pseudo recovery is a condition when a recovered individual can be re-infected by the disease due to incomplete treatment and decreased immunity. Pseudo recovery is described by individuals in recovered class \((R)\) who return to the infected class \((I)\).

\[
\begin{align*}
\frac{dS}{dt} &= \mu N - \frac{\beta S(t)I(t)}{N} - \mu S(t) \quad (1) \\
\frac{dE}{dt} &= \frac{\beta S(t)I(t)}{N} - (\alpha + \mu)E(t) \quad (2) \\
\frac{dI}{dt} &= \alpha E(t) - (\gamma + \mu)I(t) + \theta R(t) \quad (3) \\
\frac{dR}{dt} &= \gamma I(t) - (\mu + \theta)R(t) \quad (4)
\end{align*}
\]

where \(\mu, \beta, \alpha, \gamma, \theta > 0\) and \(N(t) = S(t) + E(t) + I(t) + R(t)\), with

- \(S(t)\) : number of susceptible individuals at time \(t\) (persons),
- \(E(t)\) : number of exposed individuals at time \(t\) (persons),
- \(I(t)\) : number of infected individuals at time \(t\) (persons),
- \(R(t)\) : number of individuals who recover at time \(t\) (persons),
- \(N(t)\) : total population at time \(t\) (persons),
- \(\mu\) : natural birth and death rates (1/time),
- \(\beta\) : effective contact rate (1/time),
- \(\alpha\) : individual transmission rate of Exposed class to Infected class (1/time),
- \(\gamma\) : individual transmission rate of Infected class to Recovered class (1/time),
- \(\theta\) : relapse rate (1/time).

\[2\]
This research will discuss a deterministic epidemiological model with pseudo-recovery. The model used is the SEIRI epidemic model. The SEIRI epidemic model is a disease spread model that illustrates that susceptible individuals will contract the disease by a sick individual so that the individual is exposed to the disease, then after being exposed to the disease the individual will become infected. The existence of medication and the immune system in the body causes the individual to be recovered, but due to incomplete treatment and over time the low immune system causes the recovered individual to become re-infected. The compartment diagram of the SEIRI epidemic model is illustrated in Figure 1 which is described by the system of differential equations in equations (1)-(4).

\[ S(0) = S_0, E(0) = E_0, I(0) = I_0, R(0) = R_0 \]

The initial step in this discussion is to transform the system of equations in equations (1)-(4) and divide each variable by the total population \((N)\) as follows:

\[ \bar{S} = \frac{S}{N}, \quad \bar{E} = \frac{E}{N}, \quad \bar{I} = \frac{I}{N}, \quad \bar{R} = \frac{R}{N}, \quad \text{and} \quad \bar{S} + \bar{E} + \bar{I} + \bar{R} = 1. \]

The system of equations (6) is derived with respect to \(t\), so that the following model is obtained:

\[
\begin{align*}
\frac{d\bar{S}}{dt} &= \frac{1}{N} \frac{dS}{dt} - \frac{S}{N^2} \frac{dN}{dt} \\
\frac{d\bar{E}}{dt} &= \frac{1}{N} \frac{dE}{dt} - \frac{E}{N^2} \frac{dN}{dt} \\
\frac{d\bar{I}}{dt} &= \frac{1}{N} \frac{dI}{dt} - \frac{I}{N^2} \frac{dN}{dt} \\
\frac{d\bar{R}}{dt} &= \frac{1}{N} \frac{dR}{dt} - \frac{R}{N^2} \frac{dN}{dt}
\end{align*}
\]

with

\[
\frac{dN}{dt} = \frac{ds}{dt} + \frac{de}{dt} + \frac{di}{dt} + \frac{dr}{dt}
\]

Furthermore, the substitution of system of differential equations in equations (1)-(4) to equation (11) is obtained

\[ \frac{dN}{dt} = 0. \]

From the system of differential equations in equations (1)-(4) and (11), the equation (7)-(10) can be written in equations (12)-(15).

\[
\begin{align*}
\frac{d\bar{S}}{dt} &= \mu - \beta \bar{S}(t)\bar{I}(t) - \mu \bar{S}(t), \\
\frac{d\bar{E}}{dt} &= \beta \bar{S}(t)\bar{I}(t) - (\alpha + \mu) \bar{E}(t), \\
\frac{d\bar{I}}{dt} &= \alpha \bar{E}(t) - (\gamma + \mu) \bar{I}(t) + \theta \bar{R}(t), \\
\frac{d\bar{R}}{dt} &= \gamma \bar{I}(t) - (\mu + \theta) \bar{R}(t).
\end{align*}
\]

For symbolic simplification, suppose \( \bar{S} = S, \bar{E} = E, \bar{I} = I, \) and \( \bar{R} = R, \) the equations (12)-(15) becomes as shown below
\[
\begin{align*}
\frac{dS}{dt} &= \mu - \beta S(t)I(t) - \mu S(t), \\
\frac{dE}{dt} &= \beta S(t)I(t) - (\alpha + \mu)E(t), \\
\frac{dI}{dt} &= \alpha E(t) - (\gamma + \mu)I(t) + \theta R(t), \\
\frac{dR}{dt} &= \gamma I(t) - (\mu + \theta)R(t),
\end{align*}
\]

where \(\mu, \beta, \alpha, \gamma, \theta > 0\) and \(N = 1\) with
\(S(t)\) : proportion of the number of susceptible individuals at time \(t\),
\(E(t)\) : proportion of the number of exposed individuals at time \(t\),
\(I(t)\) : proportion of the number of infected individuals at time \(t\),
\(R(t)\) : proportion of the number of recovered individuals at time \(t\).

3. Fixed Point and Basic Reproduction Number

The fixed point was determined by setting the rate of changes indicated in (16)-(19) to be zero.
\[
\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0
\]
The fixed points are of two types, namely disease-free fixed points and endemic fixed points. The fixed point obtained will be analyzed for its stability behavior.

3.1. Disease-free fixed point
The disease-free fixed point can be determined by assigning the population of \(E, I,\) and \(R\) are zero, as follows,
\[
E_0 = (S,E,I,R) = (1,0,0,0).
\]

3.2. Endemic fixed point
An endemic fixed point can be determined by assigning the population of \(E, I,\) and \(R\) are non-zero as follows,
\[
E_1 = (S,E,I,R) = (S^*,E^*,I^*,R^*),
\]
with
\[
S^* = \frac{\mu}{\alpha \theta + \alpha \mu} \left[ (\gamma + \theta + \mu) - \frac{\alpha \beta (\gamma + \theta + \mu)}{\beta (\alpha + \mu) (\gamma + \theta + \mu)} \right],
\]
\[
E^* = \frac{\mu}{\alpha \theta + \alpha \mu} \left[ (\gamma + \theta + \mu) - \frac{\alpha \beta (\gamma + \theta + \mu)}{\beta (\alpha + \mu) (\gamma + \theta + \mu)} \right],
\]
\[
I^* = \frac{\alpha \beta (\gamma + \theta + \mu)}{\beta (\alpha + \mu) (\gamma + \theta + \mu)},
\]
\[
R^* = \frac{\gamma (\alpha \beta + \alpha \beta \mu - \alpha \gamma \mu - \gamma \mu^2 - \gamma \mu^2 - \theta \mu^2 - \mu^3)}{\beta (\alpha + \mu) (\gamma + \theta + \mu)}.
\]

3.3. Basic Reproduction Number
Basic reproduction number, \(R_0\), is defined as an expected number of secondary infected individuals within the whole population is susceptible by introducing a single infected individual. The basic reproduction number can be obtained using the next-generation matrix. The next-generation matrix which is denoted by the matrix \(G\) is the multiplication between the matrix \(F\) and the inverse matrix \(V\), where \(F\) represents
the infection rate in each disease class and $V$ represents the transmission of each disease class (see [2],[8]). Based on equations (16)-(19) it is obtained

$$\frac{d}{dt} \begin{pmatrix} E \\ I \\ S \end{pmatrix} = \begin{pmatrix} \beta SI \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} (\alpha + \mu)E \\ (\gamma + \mu)I - \alpha E - \theta R \\ (\theta + \mu)R - \gamma I \end{pmatrix}$$

There are three compartments of the disease, namely $E, I,$ and $R$. The matrix $F$ and $V$ are defined as follows

$$F = \begin{pmatrix} 0 & \beta S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \alpha + \mu & 0 & 0 \\ -\alpha & \gamma + \mu & -\theta \\ 0 & -\gamma & \theta + \mu \end{pmatrix}$$

Each matrix element above is derived with respect to $E, I,$ and $R$, so that it obtained

$$F = \begin{pmatrix} 0 & \beta S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \alpha + \mu & 0 & 0 \\ -\alpha & \gamma + \mu & -\theta \\ 0 & -\gamma & \theta + \mu \end{pmatrix}$$

Having obtained a disease-free fixed point (8), then

$$F = \begin{pmatrix} 0 & \beta S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \alpha + \mu & 0 & 0 \\ -\alpha & \gamma + \mu & -\theta \\ 0 & -\gamma & \theta + \mu \end{pmatrix}$$

The next-generation matrix symbolized by $G$ can be determined using the following equation.

$$G = FV^{-1}$$

Before determining the matrix $G$, first, determine the inverse of the matrix $V$ using the adjoin matrix. \( \text{Adj}(V) = (\text{Kof}(V))^T \)

The adjoin matrix is obtained as follows:

$$\text{Adj}(V) = \begin{pmatrix} (\gamma + \mu)(\mu + \theta) - \gamma \theta & 0 & 0 \\ \alpha(\mu + \theta) & (\alpha + \mu)(\mu + \theta) & \theta(\alpha + \mu) \\ \gamma(\alpha + \mu) & \gamma(\alpha + \mu) & (\alpha + \mu)(\gamma + \mu) \end{pmatrix}$$

By using the Sarrus method, the determinant of the $V$ matrix is obtained as follows.

$$\text{det}(V) = \begin{vmatrix} \alpha + \mu & 0 & 0 \\ -\alpha & \gamma + \mu & -\theta \\ 0 & -\gamma & \theta + \mu \end{vmatrix} = (\alpha + \mu)(\gamma + \mu)(\theta + \mu) - \gamma \theta (\alpha + \mu)$$

The inverse of the matrix $V$ is determined using the following formula,

$$V^{-1} = \frac{1}{\text{det}(V)} \text{Adj}(V)$$

so that the inverse matrix $V$ is obtained as follows.

$$V^{-1} = \begin{pmatrix} (\gamma + \mu)(\mu + \theta) - \gamma \theta & 0 & 0 \\ \alpha(\mu + \theta) & (\alpha + \mu)(\mu + \theta) & \theta(\alpha + \mu) \\ \gamma(\alpha + \mu) & \gamma(\alpha + \mu) & (\alpha + \mu)(\gamma + \mu) \end{pmatrix}$$

Since $F$ and $V^{-1}$ matrices have been obtained, we will get the matrix $G$ as follows.

$$G = \begin{pmatrix} a\beta(\alpha + \mu) & 0 & 0 \\ 0 & b\beta(\alpha + \mu) & 0 \\ 0 & 0 & c\beta(\alpha + \mu) \end{pmatrix}$$

The basic reproduction number ($R_0$) is maximum eigenvalue of the next generation matrix $G$ so that it is obtained
3.4. Theorem 1
The solution of the set \{S, E, I, R\} in the system of differential equations in equations (16)-(19) with a non-negative initial value (5) will be non-negative for every \( t > 0 \).

Proof. Suppose given a non-negative initial value (5). Proof of Theorem 1 can be done using the integration factor method [8].

- **Susceptible population**
  \[
  \frac{dS}{dt} = \mu - \beta S(t)I(t) - \mu S(t)
  \]

- **Exposed population**
  \[
  \frac{dE}{dt} = \beta S(t)I(t) - (\alpha + \mu)E(t)
  \]

- **Infected population**
  \[
  \frac{dl}{dt} = \alpha E(t) - (\gamma + \mu)l(t) + \beta R(t)
  \]

\[
\mathcal{R}_0 = \frac{\alpha \beta (\mu + \theta)}{(\alpha + \mu)(\gamma + \mu)(\theta + \mu) - \gamma \theta (\alpha + \mu)} = \frac{\alpha \beta (\mu + \theta)}{\mu (\alpha + \mu)(\theta + \gamma + \mu)}
\]

\[
\text{Proof. } \text{Suppose given a non-negative initial value (5). Proof of Theorem 1 can be done using the integration factor method [8].}
\]

\[
\frac{dS}{dt} = \mu - \beta S(t)I(t) - \mu S(t)
\]

\[
\frac{dI}{dt} = \beta S(t)I(t) - \mu I(t) - \gamma I(t) + \alpha E(t)
\]

\[
\frac{dR}{dt} = \gamma I(t) - \mu R(t)
\]

\[
\text{Suppose} \ t = 0, \text{we can obtain:}
\]

\[
S(t) > S(0) \exp\left(-\left(\mu + \beta \int_0^t (I(\zeta)) d\zeta\right)\right) > 0, \forall t > 0, \text{because both sides are multiplied by}
\]

\[
\exp\left(-\left(\mu + \beta \int_0^t (I(\zeta)) d\zeta\right)\right)
\]

\[
\text{Exposed population}
\]

\[
\frac{dE}{dt} = \beta S(t)I(t) - (\alpha + \mu)E(t)
\]

\[
\frac{dE}{dt} + (\alpha + \mu)E(t) \geq 0,
\]

\[
\frac{d}{dt} \left[ E(t) \exp\left(\int_0^t (\alpha + \mu) d\zeta\right) \right] \geq 0, \text{because using the integration factor method}
\]

\[
\frac{d}{dt} \left[ E(t) \exp\left((\alpha + \mu) t\right) \right] \geq 0, \text{because } \int_0^t (\alpha + \mu) d\zeta = (\alpha + \mu)t
\]

\[
E(t) \exp((\alpha + \mu)t) \geq c, \text{because both sides are integrated}
\]

Suppose \( t = 0 \), we can obtain:

\[
E(t) \geq E(0), \forall t > 0, \text{because both sides are multiplied by}
\]

\[
\exp\left(\int_0^t (\alpha + \mu) t\right)
\]

\[
\text{Infected population}
\]

\[
\frac{dl}{dt} = \alpha E(t) - (\gamma + \mu)l(t) + \beta R(t)
\]
\[ \frac{dl}{dt} + (\gamma + \mu) l(t) \geq 0, \]
\[ \leftrightarrow \frac{d}{dt} \left[ l(t) \exp \int_0^t (\gamma + \mu) \, d\zeta \right] \geq 0, \text{ because using the integration factor method} \]
\[ \leftrightarrow \frac{d}{dt} \left[ l(t) \exp((\gamma + \mu) t) \right] \geq 0, \text{ because } \int_0^t (\gamma + \mu) \, d\zeta = (\gamma + \mu) t \]
\[ \rightarrow l(t) \exp((\gamma + \mu) t) \geq c, \text{ because both sides are integrated} \]

Suppose \( t = 0 \), we can obtain:
\[ l(t) \geq l(0) \exp(-(\gamma + \mu) t) > 0, \forall t > 0, \text{ because both sides are multiplied by} \]
\[ \exp(-(\gamma + \mu) t) \]

\[ \bullet \text{ Recovered population} \]
\[ \frac{dR}{dt} = \gamma I(t) - (\mu + \theta) R(t) \]
\[ \frac{dR}{dt} + (\mu + \theta) R(t) \geq 0, \]
\[ \leftrightarrow \frac{d}{dt} \left[ R(t) \exp \int_0^t (\mu + \theta) \, d\zeta \right] \geq 0, \text{ because using the integration factor method} \]
\[ \leftrightarrow \frac{d}{dt} \left[ R(t) \exp((\mu + \theta) t) \right] \geq 0, \text{ because } \int_0^t (\mu + \theta) \, d\zeta = (\mu + \theta) t \]
\[ \rightarrow R(t) \exp((\mu + \theta) t) \geq c, \text{ because both sides are integrated} \]

Suppose \( t = 0 \), we can obtain:
\[ R(t) \exp((\mu + \theta) t) \geq R(0), \]
\[ \rightarrow R(t) \geq R(0) \exp\left[-((\mu + \theta) t)\right] > 0, \forall t > 0, \text{ because both sides are multiplied by} \]
\[ \exp\left[-((\mu + \theta) t)\right] \]

So \( S(t), E(t), I(t), R(t) > 0 \) (see [8])

4. Stability Analysis

The fixed point stability can be done through the Jacobi matrix, namely matrix \( A \). Determination of fixed-point stability is obtained by looking at its eigenvalues, namely \( \lambda_i \) with \( i = 1, 2, 3, \ldots \), not obtained from \( \det(A - \lambda I) = 0 \). In general, the stability of fixed point has three behaviors, namely stable, unstable, and saddle. Stable if each eigenvalue is negative \( \lambda_i < 0 \) for all \( i \) and each component of the complex eigenvalues of the real part is less than or equal to zero \( \text{Re}(\lambda_i) \leq 0 \) for all \( i \). Unstable if each eigenvalue is positive \( \lambda_i > 0 \) for all \( i \) and each component of the complex eigenvalues of the real part is greater than or equal to zero \( \text{Re}(\lambda_i) \geq 0 \) for all \( i \). Saddle if there is a multiplication of two real eigenvalues is negative \( \lambda_i, \lambda_j < 0 \) for an \( i \) and \( j \) (see [10]).

According to [2], the disease-free fixed point, \( E_0 \), in equations (16)-(19) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \). Meanwhile, global stability will be proven in Theorem 2 and Theorem 3, which is the stability criterion for disease-free fixed point \( E_0 \) and endemic fixed point \( E_I \) by looking at the basic reproduction number. The complete proof will be discussed in the proof of Theorem 2 and Theorem 3.

4.1. Theorem 2

If \( R_0 \leq 1 \), the disease-free fixed point, \( E_0 \), in the system of differential equations in equations (16)-(19) is globally asymptotically stable in region \( \mathcal{D} \), with

\[ \mathcal{D} = \{(S,E,I,R) \in R^4_+ : S + E + I + R = 1\} \]
\[ Q = \frac{\alpha}{\alpha + \mu} E + I + \frac{\theta}{\theta + \mu} R \]

The derivative of the Lyapunov function is obtained as follows:

\[ \dot{Q} = \frac{\alpha}{\alpha + \mu} \dot{E} + \dot{I} + \frac{\theta}{\theta + \mu} \dot{R}. \]

With substitute the equations (16)-(19) into the derivative Lyapunov function, we can obtain:

\[
\begin{align*}
\dot{\theta} &= \frac{\alpha}{\alpha + \mu} \left[ \beta SI - (\alpha + \mu)E + [\alpha E - (\gamma + \mu)I + \theta R] + \frac{\theta y_1}{\theta + \mu} [\gamma I - (\theta + \mu)R] \right] \\
\dot{\gamma} &= \frac{\alpha}{\alpha + \mu} \left[ \gamma SI - \alpha E - (\gamma + \mu)I + \theta R + \frac{\theta y_1}{\theta + \mu} - \theta R \right] \\
\dot{\beta} &= \frac{\alpha}{\alpha + \mu} \left[ \beta SI - (\gamma + \mu)I + \frac{\theta y_1}{\theta + \mu} \right] \\
\dot{\gamma} &= \frac{\alpha}{\alpha + \mu} \left[ \gamma SI - (\gamma + \mu)I + \frac{\theta y_1}{\theta + \mu} \right] \\
\dot{\beta} &= \frac{\alpha}{\alpha + \mu} \left[ \beta SI - (\gamma + \mu)I + \frac{\theta y_1}{\theta + \mu} \right] \\
\dot{\gamma} &= \frac{\alpha}{\alpha + \mu} \left[ \gamma SI - (\gamma + \mu)I + \frac{\theta y_1}{\theta + \mu} \right]
\end{align*}
\]

\[ \dot{\gamma} \leq 0 \text{ for } R_0 \leq l. \]

So if \( l \to 0 \) for \( t \to \infty \), then \((S, E, R) = (1, 0, 0) \) for \( t \to \infty \). Thus, \( \{E_0\} \) is the largest invariant set of \( \{(S, E, I, R) \in \mathbb{D} : \dot{\gamma} = 0\} \). According to Lyapunov-LaSalle’s invariance principle (see [5]), the disease-free fixed point, \( E_0 \) is globally asymptotically stable in \( \mathbb{D} \) if \( R_0 \leq 1 \).

### 4.2. Theorem 3

If \( R_0 > 1 \), the endemic fixed point, \( E_l \) in the system of differential equations in equations (16)-(19) is a globally asymptotically stable.

**Proof.** There are several types of Lyapunov function any some type, such as Lotka-Volterra type Lyapunov function, common quadratic Lyapunov function, and composite quadratic Lyapunov function [6]. In this proof, we used the Lotka-Volterra type Lyapunov function. The Lyapunov function is defined as follows (see [8], [9]):

\[
\begin{align*}
Q &= S - S^* - S^* \ln \left( \frac{S}{S^*} \right) + E - E^* - \ln \left( \frac{E}{E^*} \right) + \frac{\alpha + \mu}{\alpha} \left[ I - I^* - \ln \left( \frac{I}{I^*} \right) \right] \\
&\quad + \frac{\theta (\alpha + \mu)}{\alpha (\theta + \mu)} \left[ R - R^* - \ln \left( \frac{R}{R^*} \right) \right] \\
\dot{Q} &= \dot{S} - \frac{SS^*}{S} + \dot{E} - \frac{E \dot{E}}{E} + \frac{\alpha + \mu}{\alpha} \left[ I - \frac{I^*}{I} \right] + \frac{\theta (\alpha + \mu)}{\alpha (\theta + \mu)} \left[ R - \frac{RR^*}{R} \right] \\
\dot{S} &= \hat{S} \left[ 1 - \frac{S^*}{S} \right] + \hat{E} \left[ 1 - \frac{E^*}{E} \right] + \left( \frac{\alpha + \mu}{\alpha} \right) i \left[ 1 - \frac{I^*}{I} \right] + \frac{\theta (\alpha + \mu)}{\alpha (\theta + \mu)} \hat{R} \left[ 1 - \frac{R^*}{R} \right]
\end{align*}
\]

With substitute the equations (16)-(19) into the derivative of Lyapunov function, we can get:
\[ \dot{N} = [\mu - \beta S] - \mu S \left[ 1 - \frac{S}{S} \right] + [\beta S] - (\alpha + \mu)E \left[ 1 - \frac{E}{E} \right] \]
\[ + \left( \frac{\alpha + \mu}{\alpha} \right) [\alpha E - (\gamma + \mu)I + \theta R] \frac{1 - \mu}{\mu} + \theta(\alpha + \mu) \frac{\gamma I - (\mu + \theta)R}{\alpha} \left[ 1 - \frac{R}{R} \right] \]
\[ \dot{S} = [\mu - \beta S] - \mu S - \mu S \left[ 1 - \frac{S}{S} \right] + [\beta S] - (\alpha + \mu)E \left[ 1 - \frac{E}{E} \right] \]
\[ + \left( \frac{\alpha + \mu}{\alpha} \right) [\alpha E - (\gamma + \mu)I + \theta R] \frac{1 - \mu}{\mu} + \theta(\alpha + \mu) \frac{\gamma I - (\mu + \theta)R}{\alpha} \left[ 1 - \frac{R}{R} \right] \]
\[ \dot{E} = [\mu - \beta S] - \mu S - \mu S \left[ 1 - \frac{S}{S} \right] + [\beta S] - (\alpha + \mu)E \left[ 1 - \frac{E}{E} \right] \]
\[ + \left( \frac{\alpha + \mu}{\alpha} \right) [\alpha E - (\gamma + \mu)I + \theta R] \frac{1 - \mu}{\mu} + \theta(\alpha + \mu) \frac{\gamma I - (\mu + \theta)R}{\alpha} \left[ 1 - \frac{R}{R} \right] \]
\[ \dot{I} = [\mu - \beta S] - \mu S - \mu S \left[ 1 - \frac{S}{S} \right] + [\beta S] - (\alpha + \mu)E \left[ 1 - \frac{E}{E} \right] \]
\[ + \left( \frac{\alpha + \mu}{\alpha} \right) [\alpha E - (\gamma + \mu)I + \theta R] \frac{1 - \mu}{\mu} + \theta(\alpha + \mu) \frac{\gamma I - (\mu + \theta)R}{\alpha} \left[ 1 - \frac{R}{R} \right] \]
when conditions are endemic, the system of differential equations in equations (16)-(19) can be written as follows.

\[ \mu - \beta S I - \mu S = 0 \rightarrow \mu = \beta S I + \mu S \]
(22)

\[ \beta S I - (\alpha + \mu)E = 0 \rightarrow \alpha + \mu = \frac{\beta S I}{E} \]
(23)

\[ \alpha E - (\gamma + \mu)I + \theta R = 0 \rightarrow \gamma + \mu = \frac{\alpha E + \theta R}{I} \]
(24)

\[ \gamma I - (\mu + \theta)R = 0 \rightarrow \mu + \theta = \frac{\gamma I}{R} \]
(25)

With substituting the equations Error! Reference source not found.,(25), we will get:
\[ \dot{N} = [\beta S I + \mu S] \left[ 1 - \frac{S}{S} \right] - \mu S \left[ 1 - \frac{S}{S} \right] + [\beta S] - \beta S I - \beta S I - \beta S I - \beta S I \frac{\alpha E + \theta R}{I} \]
\[ \dot{S} = [\beta S I + \mu S] - [\beta S I] - \mu S + \mu S + \beta S I - \beta S I - \beta S I - \beta S I \frac{\alpha E + \theta R}{I} \]
\[ \dot{E} = [\beta S I + \mu S] - [\beta S I] - \mu S + \mu S + \beta S I - \beta S I - \beta S I \frac{\alpha E + \theta R}{I} \]
\[ \dot{I} = [\beta S I + \mu S] - [\beta S I] - \mu S + \mu S + \beta S I - \beta S I - \beta S I \frac{\alpha E + \theta R}{I} \]
\[ \dot{Q} = 2\mu S^* - \mu S^* \frac{S^*}{S} - \mu S + 3\beta S^* I - \beta S^* I - \beta S I \frac{S^*}{S} - \beta SI E^* - \frac{\beta S^* I^* E^*}{E} + 2 \frac{\beta S^* I^* R^*}{\alpha E^*} - \frac{\beta S^* I^* \theta R^*}{\alpha E^*} \]

\[ \dot{S} = \mu S^* \left[ 2 - \frac{S^*}{S} - \frac{S}{S^*} \right] + \beta S^* I^* \left[ 3 - \frac{S^*}{S} - \frac{S}{S^*} \frac{E^*}{E} - \frac{E^* I^*}{E^*} \right] + \frac{\beta S^* I^* R^*}{\alpha E^*} \left[ 2 - \frac{I^* R^*}{IR^*} \right] - \frac{\beta S^* I^* \theta R^*}{\alpha E^*} \]

Using the AM-GM (Arithmetic Mean – Geometric Mean), it is obtained:

\[ \begin{align*}
 2 - \frac{S^*}{S} - \frac{S}{S^*} & \leq 0, \\
 3 - \frac{S^*}{S} - \frac{S}{S^*} \frac{E^*}{E} - \frac{E^* I^*}{E^*} & \leq 0, \\
 2 - \frac{I^* R^*}{IR^*} - \frac{I^* R^*}{IR^*} & \leq 0.
\end{align*} \]

So that \( \dot{Q} \leq 0 \) with \( \dot{Q} = 0 \) if \( S = S^* \), \( E = E^* \), \( I = I^* \) and \( R = R^* \). Based on [3] and [7], the AM-GM inequality can be written:

\[ \frac{x_1 + x_2 + \cdots + x_n}{n} \geq \sqrt[n]{x_1 x_2 \cdots x_n} \iff x_1 + x_2 + \cdots + x_n \geq n \sqrt[n]{x_1 x_2 \cdots x_n} \]

- **Proof** \[ 2 - \frac{S^*}{S} - \frac{S}{S^*} \leq 0 \]

\[ \begin{align*}
 \frac{S^*}{S} + \frac{S}{S^*} & \geq 2, \\
 - \frac{S}{S^*} - \frac{S^*}{S} & \leq -2, \\
 2 - \frac{S^*}{S} - \frac{S}{S^*} & \leq 0.
\end{align*} \]

- **Proof** \[ 3 - \frac{S^*}{S} - \frac{S}{S^*} \frac{E^*}{E^*} - \frac{E^* I^*}{E^*} \leq 0 \]

\[ \begin{align*}
 \frac{S^*}{S} + \frac{S}{S^*} \frac{E^*}{E^*} + \frac{E^* I^*}{E^*} & \geq 3, \\
 - \frac{S}{S^*} - \frac{S^*}{S} \frac{E^*}{E^*} - \frac{E^* I^*}{E^*} & \leq -3, \\
 3 - \frac{S^*}{S} - \frac{S}{S^*} \frac{E^*}{E^*} - \frac{E^* I^*}{E^*} & \leq 0.
\end{align*} \]

- **Proof** \[ 2 - \frac{I^* R^*}{IR^*} - \frac{I^* R^*}{IR^*} \leq 0 \]

\[ \begin{align*}
 \frac{I^* R^*}{IR^*} + \frac{I^* R^*}{IR^*} & \geq 2, \\
 - \frac{I^* R^*}{IR^*} - \frac{I^* R^*}{IR^*} & \leq -2, \\
 2 - \frac{I^* R^*}{IR^*} - \frac{I^* R^*}{IR^*} & \leq 0.
\end{align*} \]

Since \[ 2 - \frac{S^*}{S} - \frac{S}{S^*} \leq 0, \] \[ 3 - \frac{S^*}{S} - \frac{S}{S^*} \frac{E^*}{E^*} - \frac{E^* I^*}{E^*} \leq 0, \] and \[ 2 - \frac{I^* R^*}{IR^*} - \frac{I^* R^*}{IR^*} \leq 0, \] resulting in \( \dot{Q} \leq 0 \).

Based on Lyapunov-LaSalle’s invariance principle [5], the largest subset invariant where \( \dot{Q} = 0 \) is \( \{E_1 = (S^*, E^*, I^*, R^*)\} \) and it can be concluded that the endemic fixed point, \( E_1 \), is globally asymptotically stable.

### 5. Sensitivity Analysis

Sensitivity analysis is an analysis that is used to determine the effect of changing parameters on basic reproduction number. This analysis is carried out by changing one of the parameters in the simulation so that it can be seen how much influence these parameters have. As a consideration in conducting a sensitivity analysis, the sensitivity index can be determined by the following equation (see [1], [8]).
\[
\frac{\partial R_0}{\partial p} \times \frac{p}{R_0}
\]

with \( p \) is the parameter in the model.

The population dynamics of disease spread are influenced by several parameter values. Changes in parameter values can affect the simulation results. However, each parameter has a different level of influence depending on the level of sensitivity. The sensitivity analysis was carried out by considering the parameter sensitivity index value \( Y_p^{R_0} \). The sensitivity index is determined using equation (26) with \( R_0 \) is the equation (21).

\[
\begin{align*}
Y_{\beta}^{R_0} &= 1 \\
Y_{\mu}^{R_0} &= \frac{-\left( \alpha \theta^2 + 2 \theta^2 \mu + \alpha \theta \gamma + 2 \mu a \theta + 2 \mu \gamma + 4 \theta \mu^2 + a \mu^2 + \mu^2 \gamma + 2 \mu^3 \right)}{(\theta + \mu)(\alpha + \mu)(\theta + \gamma + \mu)} \\
Y_{\alpha}^{R_0} &= \frac{\mu}{(\alpha + \mu)} \\
Y_{\theta}^{R_0} &= \frac{\gamma \theta}{(\theta + \mu)(\theta + \gamma + \mu)} \\
Y_{\gamma}^{R_0} &= \frac{-\gamma}{(\theta + \gamma + \mu)}
\end{align*}
\]

The sensitivity index is shown in Table 1 by substituting each parameter. The parameter values used, among others \( \beta = 0.1, \mu = 0.01, \alpha = 0.02, \theta = 0.25, \) and \( \gamma = 0.6 \). Each parameter has a different sensitivity index mark.

| Parameter | The sensitivity index |
|-----------|-----------------------|
| \( \beta \) | 1                      |
| \( \mu \) | -2.64818               |
| \( \alpha \) | 0.333333              |
| \( \theta \) | 0.670841               |
| \( \gamma \) | -0.697674             |

Table 1 explains the parameters that affect \( R_0 \) are \( \mu \) and \( \beta \). The positive sign of sensitivity index means that increasing value of each of the parameter increases \( R_0 \), and a decreasing value of each of the parameter decreases \( R_0 \). The negative sign of sensitivity index indicates that a decrease in value of each of the parameter increases \( R_0 \) and an increase in value of each of the parameter decreases \( R_0 \). The highest sensitivity index values derived from \( \beta \) and \( \mu \), meaning that \( \beta \) and \( \mu \) have the greatest influence on changes or sensitivity of \( R_0 \) in causing endemic populations. The sensitivity index value of the \( \mu \) parameter is -2.64818, meaning that the parameter of \( \mu \) is very influential in changing the \( R_0 \) value. However, the \( \mu \) parameter was not simulated because it was assumed to be constant.

Next will be simulated the effect of the relapse rate \( (\theta) \) and the effective contact rate \( (\beta) \) on population dynamics in the presence of apparent recovery.

6. Numerical Simulation

Numerical simulation is performed to show the stability of a fixed point that has been obtained using Mathematica 12.0 Software. The parameter values used can be seen in Table 2 [8]. Parameters \( \mu, \gamma \), and \( \alpha \) is fixed values, while the changing parameter values are \( \beta \) and \( \theta \). The assumed initial value is \( S_0 = 0.95, E_0 = 0.01, I_0 = 0.04 \), and \( R_0 = 0 \).
Table 2. Parameter values of the model

| Parameter | Value   |
|-----------|---------|
| $\beta$   | [0,1]   |
| $\mu$     | 0.01    |
| $\alpha$  | 0.02    |
| $\theta$  | [0,1]   |
| $\gamma$  | 0.6     |

6.1. The effect of relapse rate ($\theta$) on the disease spread dynamics

This simulation was conducted to see the effect of the relapse rate on the dynamics of disease spread. The parameter values are taken in Table 2 with $\beta = 0.1$ and values of $\theta$ is varying. Each value of $\theta$ produces the basic reproduction number, eigenvalues, and stability points as shown in Table 3. The stability point depends on value of the basic reproduction number obtained. Table 3 is simulation results of changes in parameter value of $\theta$.

Table 3. The simulation results of changes in parameter value of $\theta$

| Parameter value $\theta$ | $R_0$   | Eigenvalues          | Stability point          |
|--------------------------|---------|-----------------------|--------------------------|
|                          |         | $\lambda_1 = -0.8608$,| (0.496,0.168,0.102,0.234) |
|                          |         | $\lambda_2 = -0.0421$,|                          |                          |
|                          |         | $\lambda_3 = -0.01$,   |                          |                          |
|                          |         | $\lambda_4 = -0.0072$  |                          |                          |
| 0.25                     | 2.0155  | $\lambda_1 = -0.8111$,| (0.579,0.140,0.073,0.208) |
|                          |         | $\lambda_2 = -0.0408$,|                          |                          |
|                          |         | $\lambda_3 = -0.01$,   |                          |                          |
|                          |         | $\lambda_4 = -0.0053$  |                          |                          |
| 0.25                     | 1.7284  | $\lambda_1 = -0.7124$,| (0.968,0.011,0.003,0.018) |
|                          |         | $\lambda_2 = -0.0376$,|                          |                          |
|                          |         | $\lambda_3 = -0.01$,   |                          |                          |
|                          |         | $\lambda_4 = -0.00026$  |                          |                          |
| 0.1                      | 1.0329  | $\lambda_1 = -0.6927$,| (1,0,0,0)                |                          |
|                          |         | $\lambda_2 = -0.0363$, |                          |                          |
|                          |         | $\lambda_3 = -0.01$,   |                          |                          |
|                          |         | $\lambda_4 = -0.0011$  |                          |                          |
| 0.08                     | 0.8696  | $\lambda_1 = -0.6629$,| (1,0,0,0)                |                          |
|                          |         | $\lambda_2 = -0.0336$, |                          |                          |
|                          |         | $\lambda_3 = -0.01$,   |                          |                          |
|                          |         | $\lambda_4 = -0.0035$  |                          |                          |
Based on Table 3, all eigenvalues of the model are negative and it is obtained $\mathcal{R}_0 < 1$ for $\theta$ which is 0.05 and 0.08, which means that in this state, the fixed point $(1, 0, 0, 0)$ is stable. In addition, it is also obtained $\mathcal{R}_0 > 1$ for $\theta$ which has a value of 0.1, 0.2, and 0.25 which results in an endemic stability point. The following shows the dynamics of the entire population when $\theta = 0.25$ and $\theta = 0.05$ in Figure 2 and Figure 3.

**Figure 2.** The population dynamics for $\mathcal{R}_0 > 1$ when $\theta = 0.25$

Figure 2 shows the population dynamics when $\theta = 0.25$. When $\theta = 0.25$, the value of $\mathcal{R}_0$ will be more than one, causing the population to endemic. The susceptible population and the infected population, which were originally valued at 0.95 and 0.04, will move towards 0.496 and 0.102 according to the endemic stability points.

**Figure 3.** The population dynamics for $\mathcal{R}_0 < 1$ when $\theta = 0.05$

Figure 3 shows the population dynamics when $\theta = 0.05$. The value of $\mathcal{R}_0$ will be less than one when $\theta = 0.05$ so that over time no individual is infected. The susceptible and infected populations, which were initially valued at 0.95 and 0.04, are moving towards 1 and 0.
Figure 2 and Figure 3 display the population stability points when $\theta = 0.25$ and $\theta = 0.05$ according to Table 3. The change in the proportion of each population depends on the parameter value of $\theta$. The number of population proportions will always be one even though the proportion value of each population changes according to the parameter value. Changes in the value of $\theta$ will affect population dynamics. The dynamics of the infected population from changes in parameter value of $\theta$ can be seen in Figure 4 below.

![Figure 4](image.png)

**Figure 4.** The dynamics of the infected population due to changes $\theta$ values

Figure 4 shows that changes in the value of $\theta$ will affect the dynamics of the infected population. Each value of $\theta$ has a different effect. The parameter $\theta$ which is valued at 0.05 and 0.08 results in $R_0$ value of less than one. This causes the infected population to go to zero, which means that any diseases present in the population will eventually become extinct and no individuals will be infected. So that in Figure 4 it can be seen that the curve will be stable towards a fixed point $E_0$ or a disease-free fixed point. Meanwhile, when the parameter $\theta$ is increased to a value of 0.1, 0.2, and 0.25 will result in $R_0$ value more than one. This causes the infected population to increase towards a point of endemic stability according to Table 3, which means that the disease continues to exist in the population. So, increasing the value of the parameter $\theta$ will increase the value of $R_0$ which will increase the number of infected population.

The simulation results on change in parameter $\theta$ show that when the relapse rate is decreased from $\theta = 0.1$ to $\theta = 0.08$, it will decrease $R_0$ from 1.03286 to 0.869565. In this case, the limit for endemic and disease-free occurrence can be determined analytically when the relapse rate is 0.096 assuming the values of other parameters are constant. Disease-free will occur when the parameter value of $\theta < 0.096$ and endemic occurs when the parameter value of $\theta > 0.096$.

6.2. The effect of effective contact rate ($\beta$) on the disease spread dynamics

The simulation of the effect of effective contact rate on the dynamics of disease spread was carried out using the parameter values according to Table 2 with a value of $\theta = 0.25$ and variable value of the parameter $\beta$. The change in the value of the parameter $\beta$ cause changes in $R_0$. This can be explained through the simulation results with changes in the parameter value of $\beta$ which are presented in Table 4 below.
| Parameter value $\beta$ | $\mathcal{R}_0$ | Eigenvalues | Stability Point |
|-------------------------|-----------------|-------------|-----------------|
| 0.2                     | 4.03101         | $\lambda_1 = -0.8609$, $\lambda_2 = -0.052$, $\lambda_3 = -0.0175$, $\lambda_4 = -0.01$ | (0.248,0.25,0.152,0.35) |
| 0.1                     | 2.0155          | $\lambda_1 = -0.8608$, $\lambda_2 = -0.0421$, $\lambda_3 = -0.01$, $\lambda_4 = -0.0072$ | (0.50,0.17,0.10,0.23) |
| 0.075                   | 1.51163         | $\lambda_1 = -0.8608$, $\lambda_2 = -0.0404$, $\lambda_3 = -0.01$, $\lambda_4 = -0.0038$ | (0.66,0.11,0.07,0.16) |
| 0.025                   | 0.50388         | $\lambda_1 = -0.8604$, $\lambda_2 = -0.0354$, $\lambda_3 = -0.01$, $\lambda_4 = -0.0042$ | (1,0,0,0) |
| 0.01                    | 0.20155         | $\lambda_1 = -0.8602$, $\lambda_2 = -0.0324$, $\lambda_3 = -0.01$, $\lambda_4 = -0.00738$ | (1,0,0,0) |

Based on Table 4, it can be explained that the $\mathcal{R}_0$ value will change along with the change in the parameter value of $\beta$. If the value of $\beta$ is enlarged, the value of $\mathcal{R}_0$ will increase. Conversely, if the value of $\beta$ is reduced, it will cause the value of $\mathcal{R}_0$ to decrease. For $\beta$ with a value of 0.01 and 0.025, the value of $\mathcal{R}_0$ is less than one, while for $\beta$ which has a value of 0.075, 0.1, and 0.2, the value of $\mathcal{R}_0$ is more than one. This means that with increasing in the value of $\beta$ causes an increase the value of $\mathcal{R}_0$, there are still individuals who are infected so that they have not reached a disease-free state. This indicates that efforts are needed to reduce the spread of disease by reducing the effective contact rate so that $\mathcal{R}_0$ becomes less than 1. The following shows the population dynamics when effective contact rates are 0.2 and 0.01 in Figure 5 and Figure 6.
Figure 5. The population dynamics for $\mathcal{R}_0 > 1$ when $\beta = 0.2$

Figure 5 shows the population dynamics when $\beta = 0.2$. The value of $\mathcal{R}_0$ will be more than one when $\beta = 0.2$. The susceptible and infected populations, which were originally valued at 0.95 and 0.04, become 0.248 and 0.152. This population proportion corresponds to the system stability point in Table 4.

Figure 6. The population dynamics for $\mathcal{R}_0 < 1$ when $\beta = 0.01$

Figure 6 shows the population dynamics when $\beta = 0.01$. The value of $\mathcal{R}_0$ will be less than one when $\beta = 0.01$ so that over time the population will be free from disease. The proportions of the susceptible and infected population which were initially valued at 0.95 and 0.04 moved towards 1 and 0 according to the point of the disease-free stability.

Changes the value of $\beta$ cause changes in the stability point of the system. The dynamics of the infected population from changes the value of $\beta$ can be seen in Figure 7 below.
Figure 7 shows that when $R_0 < 1$ is when $\beta = 0.01$ and $\beta = 0.025$ the dynamic model of disease spread will go to a disease-free state, while when $R_0 > 1$ is when $\beta = 0.075$, $\beta = 0.1$, and $\beta = 0.2$, dynamic model of disease spread will lead to endemic conditions so that infected individuals are still present.

The parameter values of $\beta = 0.01$ and $\beta = 0.025$ cause the infected population to zero so that after a long time no individual is infected and becomes a population free from disease. The parameter $\beta$ which is valued at $0.075$, $0.1$, and $0.2$ causes the infected population as shown in Figure 7 and will be stable at the endemic stability point shown in Table 4. The increase in $\beta$ value affects the number of infected populations so that over time the infected population will increase. The infected population will be stable according to the endemic stability point.

The result of the simulation on the change in parameter $\beta$ show that when the effective contact rate is decreased from $\beta = 0.075$ to $\beta = 0.025$, it will decrease $R_0$ from $1.51163$ to $0.503876$. In this case, the limit for endemic and disease-free occurrence can be determined analytically when the effective contact rate is $0.0496$ assuming the other parameter values are constant. Disease-free will occur when the parameter value of $\beta < 0.0496$ and endemic occurs when the parameter value of $\beta > 0.0496$.

Parameters $\beta$ and $\theta$ have a considerable influence on the dynamics of the infected population. This can be seen in Figure 4 and Figure 7. The higher the parameter values of $\beta$ and $\theta$ are given, the more population is infected. This is because the value of $R_0$ is increasing and has a value of more than one. When seen in Figure 4 and Figure 7, the change in the value of parameter $\beta$ makes the population endemic faster than parameter $\theta$. This is because the sensitivity index of parameter $\beta$ is higher than parameter $\theta$. The following is a graph of the relationship between the relapse rate $(\theta)$ and the effective contact rate $(\beta)$ which affects the basic reproduction number value.
Based on the value of the basic reproduction number, \( R_0 = \frac{\alpha \beta (\mu + \gamma)}{\mu (\alpha + \mu)(\theta + \gamma + \mu)} \), Figure 8 shows a nonlinear relationship between the relapse rate (\( \theta \)) and the effective contact rate (\( \beta \)). The equation \( \beta = \frac{\mu (\alpha + \mu)(\theta + \gamma + \mu)}{\alpha (\mu + \theta)} \) with the value of \( \alpha, \gamma, \) and \( \mu \) is constant according to Table 2. When the values of \( \alpha, \gamma, \) and \( \mu \) are substituted for the equation, we get \( \beta = A + x + \frac{0.000183}{0.02 \theta + 0.0002} \), where \( A = \frac{0.15}{1 + 0.01 \theta} \). The equation \( \beta \) graphs the relationship between the parameters \( \theta \) and \( \beta \) as in Figure 8. The shape of this graph is in accordance with the general form of the equation \( y = \frac{\theta}{x+1} \).

The value of \( \beta = A + x + \frac{0.000183}{0.02 \theta + 0.0002} \) becomes the limit of change in the value of \( R_0 \) which will then determine the occurrence of disease-free or endemic. If \( \beta > A + x + \frac{0.000183}{0.02 \theta + 0.0002} \), the value of \( R_0 \) will be greater than one, so that there will be a plague, while \( \beta < A + x + \frac{0.000183}{0.02 \theta + 0.0002} \) will make the value of \( R_0 \) less than one, so that it will be disease-free.

7. Conclusion

Controlling the spread of disease using a model in this study can be done by looking at the value of the basic reproduction number (\( R_0 \)). If the basic reproduction number is less than or equal to one, the population will be stable in disease free condition. If the basic reproduction number is greater than one, the population will be stable in endemic condition. One way to reduce the basic reproduction number is by decreasing relapse rate or effective contact rate. So that controlling the spread of the disease can be done by reducing the effective contact rate or reducing the relapse rate.

The limit value of the parameters \( \theta \) and \( \beta \) can be determined to calculate the limit of change in the value of \( R_0 \) to determine the occurrence of endemic or disease-free. The parameters \( \beta \) and \( \theta \) have a nonlinear relationship. This relationship is illustrated by an equation that becomes the limit of change in the value \( R_0 \) to determine the occurrence of disease-free or endemic.

References

[1] Chitnis N, Hyman J M and Cushing J M 2008 Determining important parameters in the spread of Malaria through the sensitivity analysis of a mathematical model Bulletin of Mathematical Biology 70 1272-96

[2] Driessche P Van Den and Watmough J 2002 Reproduction numbers and sub-threshold endemic
equilibria for compartmental models of disease transmission Mathematical Biosciences 180 29-48

[3] Hirschhorn M D 2007 The AM-GM Inequality The Mathematical Intelligencer 29 1

[4] Kermack W O and McKendrick A G 1927 A contribution to the mathematical theory of epidemics part I Proc. of the Royal Society of London Series A (London) vol 115 (London: The Royal Society) pp 700-721

[5] LaSalle J P 1976 The Stability of Dynamical Systems (Philadelphia: Regional Conference Series in Applied Mathematics, Society for Industrial and Applied Mathematics)

[6] Leon C V D 2009 Constructions of Lyapunov function for classic SIS, SIR, and SIRS epidemic models with variable population size Foro-Red-Mat 26 www.redmat.unam.mx/foro/volumenes/Vol026

[7] Maligranda L 2012 The AM-GM Inequality is equivalent to the Bernoulli Inequality The Mathematical Intelligencer 34 1-2

[8] Olaniyi S, Lawal M A and Obabiyi O S 2016 Stability and sensitivity analysis of a deterministic epidemiological model with pseudo-recovery IAENG International J. Appl. Math. 46

[9] Shuai Z and Driessche P Van Den 2013 Global stability of infectious disease models using Lyapunov functions SIAM J. Appl. Math. 73 1513-32

[10] Strogatz S H 1994 Nonlinear Dynamics and Chaos, with Applications to Physics, Biology, Chemistry, and Engineering (Massachusetts: Addison-Wesley Publishing Company)

[11] Sumampouw O J 2017 Pemberantasan Penyakit Menular (Yogyakarta: Deepublish)