Global Stability Analysis of Sir Epidemic Model with Relapse and Immunity Loss

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Abstract

A deterministic mathematical model for the transmission dynamics of infectious disease with immunity loss and relapse was built and analyzed. The model was shown to exhibit two equilibria, namely, a disease free equilibrium and an endemic equilibrium. The computated basic reproductive number (R_0) *was used to establish that whenever* $R_0 < 1$*, the disease free equilibrium is locally asymptotically stable and the endemic equilibrium is locally asymptotically stable whenever* $R_0 > 1$*. Furthermore the global stability for the two equilibria was investigated using Lyapunov function. The model was simulated numerically to validate the analytical results.*

Keywords*: Epidemic Model, Relapse, Immunity Loss, Equilibria, Global Stability.*

1.0 Introduction

One of the major issues or events that has always attracted the attentions of large numbers of individuals worldwide apart from sport is infectious disease which is caused by pathogenic organisms such as bacteria, viruses, parasites or fungi (Shah and Gupta,2013; Akinyemi et al., 2015). Infectious diseases includes malaria, tuberculosis, cholera, AIDS, bird flu, lassa fever, ebola and could be transmitted through direct or indirect contact with contaminated body fluid or surface most especially through sex, blood transfusion, breast feeding, etc. (Shah and Gupta, 2013; James et al., 2015; Nguyen et al., 2015; Adewale et al., 2015; Al- Sheik et al.,2011). The emergence and reemergence of infectious diseases such as leprosy, plague, cholera, typhus, yellow fever, small pox diphtheria, tuberculosis, measles, ebola, pandemic influenza, severe acute respiratory (SARS), bovine tuberculosis, rinderpest, foot-and-mouth and others stated in (Hethcote et al., 2002; Sahu and Dhar, 2015; Safi, 2010), has continuously pose great challenges and threats to public health workers and individuals residing in endemic communities (Sahu and Dhar, 2015) since preventive, curative and control measures may not be hundred percent effective. Several other factors that may promote the persistence of infectious diseases includes absence of cure (e.g. HIV/AIDS), limited access to pharmaceutical interventions, disease induced stigma, poverty, etc. It is notable that recovered individuals may have temporal immunity which fades away over time or undergoes relapse. Thus capable to trigger disease burden. In 2012, it was published that infectious diseases were together responsible for the death of more than 8.7 million people worldwide (Global Health Observatory Data Repository, 2012). The socio economic impact of infectious diseases has made nations, health organisations, researchers and scientist to be at alert with the view to eradicate or contain its spread. Thus to

achieve this goal, it becomes imperative to design a framework to determine the optimal threshold needed to eradicate the spread of these life threatening diseases.

It is of great importance to state that in epidemiology, mathematical models have continously play important roles in increasing our understanding on mechanisms that influences the spread of infectious diseases ,suggesting the qualitative impact of disease control measures and forecasting disease incidences for both short and long term (Tripathi et al., 2007; Seidu and Makinde, 2014). Several epidemic models for infectious disease transmission dynamics with immunity loss are found in (Moghadas and Gumel,2003; Adda and Bichara,2012; Li et al.,1999, Peralta et al., 2015) while those with relapse are found in (Tudor, 1990; Blower, 2004;Van der Driessche et al.,2007a; Van der Driessche et al.,2007b).

The aim of this paper is to design and rigorously analyze a model that extends and complements the ones in (Moreira and Wang,1997; Korobeinikov and Wake,2002;Vargas-De-Leon,2009; Vargas-De-Leon,2011; Sajid et al., 2013; Freihat and Handam, 2014; Vargas-De-Leon,2013). The rest of this paper is organized as follows: Section 2 presents the model formulation. In Section 3, equilibria states and stability analysis of the model are presented while Section 4 presents numerical simulation and discussion of results. Section 5 concludes the paper.

2.0 Model Formulation

A non-linear deterministic model for the transmission dynamics of infectious diseases in the presence of immunity loss and relapse is built by dividing the total human population at time t, denoted by $N(t)$ into three disjoint epidemiological subpopulations, which are the susceptible population $S(t)$, infected population $I(t)$ and the recovered population $R(t)$. Thus $N(t)=S(t)+I(t)+R(t)$.

The following assumptions were considered to construct the model

1. Individuals are only recruited into the susceptible class.
2. The studied population varies with time and is homogenous.
3. Birth rate is not equal to death rate.
4. The force of infection is expressed as $\beta S(t)I(t)$.

The model is therefore governed by the following system of non-linear differential equations.

$$\frac{dS}{dt}=\pi-\beta SI-\mu S+\gamma R$$
$$\frac{dI}{dt}=\beta SI-(\mu+\delta+\theta)I+\varepsilon R$$
$$\frac{dR}{dt}=\theta I-(\mu+\gamma+\varepsilon)R \quad (1)$$

For convenience, we rewrite $S(t), I(t), R(t)$ and $N(t)$ as S, I, R and N respectively.

Table 1: Parameters Description and Hypothetical Values

Parameters	Symbols	Hypothetical Values	Source
Recruitment rate	π	5	Assumed
Disease transmission coefficient	β	0.001	Sajid et al., 2013; Freihat and Handam, 2014.
Natural death rate	μ	0.02	Safiel et al.,2012; Ibrahim et al.,2015.
Disease induced death rate	δ	0.09	Rahman and Zou, 2012.
Recovery rate	θ	0.1	Sajid et al., 2013
Relapse rate	ε	0.02	Assumed
Immunity loss rate	γ	0.05	Assumed

Lemma 1: The close set $\Omega = \left\{(S,I,Q,R) \in \mathbb{R}^4_+ : S+I+Q+R \le \frac{\pi}{\mu}\right\}$ is positively invariant and attracting with respect to the system (1)

Proof

From (1), we note that $\frac{dN}{dt} \le \pi - \mu N$ and establish that $N(t) \le N(0)e^{-\mu t} + \frac{\pi}{\mu}\left[1-e^{-\mu t}\right]$ by a standard comparism theorem (Lakshmikantham et al., 1989). $N(t)$ approaches $\frac{\pi}{\mu}$ as $t \to \infty$, thus the system (1) is positively-invariant and attracting in Ω.Thus the model is mathematically and epidemiologically meaningful in Ω (Hethcote,2000), and it is sufficient to consider solutions in Ω.

3.0 Equilibria States and Stability Analysis

The disease free equilibrium of the model is obtained as $E_0 = \left(S^*, I^*, R^*\right) = \left(\frac{\pi}{\mu}, 0, 0\right)$

The stability of E_0 can be analyzed by the method of Reproductive Number (R_0) which is determined by using the next generation method, on system (1) in the form of matrices F(non-negative) and V(non-singular) (Heffernan et al., 2005).Where F denote the new infection terms and V the transition term at E_0. Therefore

$$F = \begin{bmatrix} \frac{\beta\pi}{\mu} & 0 \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} K_1 & -\varepsilon \\ -\theta & K_2 \end{bmatrix}$$

The reproduction number is given by the spectral radius (the dominant eigenvalue) of the matrix FV^{-1} denoted by $\rho(FV^{-1})$.Thus

$$R_0 = \rho(FV^{-1}) = \frac{\beta\pi K_2}{\mu\left(K_1K_2 - \varepsilon\theta\right)} \qquad (2)$$

Where $K_1 = \mu + \delta + \theta$ and $K_2 = \mu + \gamma + \varepsilon$

The threshold R_0 is called the basic reproductive number, which is defined as the average number of secondary infections generated by a single infected individual in a totally susceptible population.

The endemic equilibrium of the model denoted by $E_1 = \left(S^{**}, I^{**}, R^{**}\right)$ as expressed in terms of R_0, is obtained as

$$S^{**} = \frac{\pi}{\mu R_0}, \qquad I^{**} = \frac{\mu\left(K_1K_2 - \theta\varepsilon\right)\left(R_0 - 1\right)}{\beta\left(K_1K_2 - \theta(\varepsilon + \gamma)\right)}, \qquad R^{**} = \frac{\theta I^{**}}{K_2}$$

Thus establishing the following results.

Proposition 1: If $R_0 < 1$, then the point E_1 does not exist and $E_1 = E_0$, when $R_0 = 1$.

Local Stability: First we investigate the local stability of the disease free equilibrium E_0.

Theorem 1: The disease-free equilibrium of system (1) is locally asymptotically stable whenever $R_0 < 1$ and unstable otherwise.

Proof. The variational matrix $J\left(E_0\right)$ of the system (1) corresponding to equilibrium E_0 is obtained as

$$J\left(E_0\right) = \begin{bmatrix} -\mu & -\dfrac{\beta\pi}{\mu} & \gamma \\ 0 & \dfrac{\beta\pi}{\mu} - K_1 & \varepsilon \\ 0 & \theta & -K_2 \end{bmatrix}$$

The characteristics equation corresponding of $J\left(E_0\right)$ is

$$f_1(\lambda) = (\lambda + \mu)\left(\lambda^2 + a_1\lambda + a_2\right) = 0$$

where

$$a_1 = K_1 + K_2 - \frac{\beta\pi}{\mu}$$

$$a_2 = K_1K_2 - \theta\varepsilon - \frac{\beta\pi K_2}{\mu}$$

Expressing a_1 and a_2 in terms of R_0, with the aid of (2) to have

$$a_1 = \frac{K_2^2 + R_0\varepsilon\theta + K_1K_2\left(1 - R_0\right)}{K_2}$$

$$a_2 = \left(K_1K_2 - \theta\varepsilon\right)\left(1 - R_0\right)$$

Thus by Routh Hurwitz criterion, we conclude that the system (1) is locally asymptotically stable since $a_i > 0$, $\forall i = 1, 2$ if and only if $R_0 < 1$.

The epidemiological implication of Theorem 1 is that the spread of an infectious disease can be effectively controlled in the community (when $R_0 < 1$) if the initial sizes of the sub-populations of the model are in the basin of attraction of the disease-free equilibrium E_0.

Theorem 2: The endemic equilibrium of system (1) is locally asymptotically stable whenever $R_0 > 1$ and unstable otherwise.

Proof. Linearizing the system at E_1, to obtain the variational matrix $J(E_1)$ as

$$J(E_1) = \begin{bmatrix} -(\beta I^{**} + \mu) & -\beta S^{**} & \gamma \\ \beta I^{**} & \dfrac{-\varepsilon R^{**}}{I^{**}} & \varepsilon \\ 0 & \theta & \dfrac{-\theta I^{**}}{R^{**}} \end{bmatrix}$$

where $\beta S^{**} - K_1 = \dfrac{-\varepsilon R^{**}}{I^{**}}$, and $-K_2 = \dfrac{-\theta I^{**}}{R^{**}}$.

The characteristics equation of $J(E_1)$ is

$$f_2(\lambda) = \left(\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0\right) = 0$$

where

$$b_2 = \frac{\theta I^{**2} + \varepsilon R^{*2} + \beta R^{**} I^{**2} + \mu R^{**} I^{**}}{R^{**} I^{**}}$$

$$b_1 = \frac{\beta\theta I^{**3} + \mu\theta I^{**2} + \varepsilon R^{**2}\left(\beta I^{**} + \mu\right) + \beta^2 R^{**} I^{**2} S^{**}}{R^{**} I^{**}}$$

$$b_0 = \frac{\beta\theta I^{**}\left(\beta I^{**} S^{**} - \gamma R^{**}\right)}{R^{**}}$$

It is obvious to note that b_2 and b_1 are greater than zero since components of the endemic equilibrium are positive provided $R_0 > 1$. We note that $\beta I^{**} S^{**} - \gamma R^{**} = \pi - \mu S^{**} = \dfrac{\pi(R_0 - 1)}{R_0}$, thus establishing that $b_0 > 0$ whenever $R_0 > 1$. Hence concluding the proof since Routh Hurwitz criterion is satisfied.

Theorem 3: The disease-free equilibrium of system (1) is globally asymptotically stable whenever $R_0 < 1$ and unstable otherwise.

Proof. Consider the Lyapunov function

$$V_1 = K_2 I + \varepsilon R \qquad (3)$$

Differentiating (3) with respect to time to obtain

$$\dot{V}_1 = K_2 \dot{I} + \varepsilon \dot{R}$$

$$\dot{V}_1 = K_2\left(\beta SI - K_1 I + \varepsilon R\right) + \varepsilon\left(\theta I - K_2 R\right) \qquad (4)$$

Simplifying (4) to get

$$\dot{V}_1 = \left(\beta S K_2 - K_1 K_2 + \varepsilon\theta\right) I \tag{5}$$

Since $S \le \frac{\pi}{\mu}$, (5) becomes

$$\dot{V}_1 \le \left(K_1 K_2 - \varepsilon\theta\right)\left(R_0 - 1\right) I$$

Clearly, $\dot{V}_1 \le 0$ when $R_0 \le 1$ and $\dot{V}_1 = 0$ if and only if $I = 0$. It follows from Lasalle's Invariance Principle (La Salle and Lefschetz,1961), that every solution to the system (1) with initial conditions in Ω approaches E_0 as $t \to \infty$. Thus, since the region Ω is positively-invariant, the disease free equilibrium is globally asymtotically stable in Ω if $R_0 \le 1$.

Theorem 4: The endemic equilibrium of system (1) is globally asymptotically stable whenever $R_0 > 1$ and unstable otherwise.

Proof. Consider the Lyapunov function

$$V_2 = \frac{1}{2}\left(\left(S - S^{**}\right) + \left(I - I^{**}\right) + \left(R - R^{**}\right)\right)^2 + \frac{2\mu + \delta}{\beta}\left(I - I^{**} - I^{**} In \frac{I}{I^{**}}\right) + \frac{\varepsilon\left(2\mu + \delta\right) R^{**}}{\beta\theta I^{**}}\left(R - R^{**} - R^{**} In \frac{R}{R^{**}}\right) + \frac{\left(2\mu + \delta\right)}{2\theta}\left(R - R^{**}\right)^2 \tag{6}$$

Differentiating (6) with respect to time to obtain

$$\frac{dV_2}{dt} = \left(\left(S - S^{**}\right) + \left(I - I^{**}\right) + \left(R - R^{**}\right)\right)\frac{dN}{dt} + \frac{2\mu + \delta}{\beta}\left(1 - \frac{I^{**}}{I}\right)\frac{dI}{dt} + \frac{\varepsilon\left(2\mu + \delta\right) R^{**}}{\beta\theta I^{**}}\left(1 - \frac{R^{**}}{R}\right)\frac{dR}{dt} + \frac{2\mu + \delta}{\theta}\left(R - R^{**}\right)\frac{dR}{dt}$$

$$\frac{dV_2}{dt} = \left(\left(S - S^{**}\right) + \left(I - I^{**}\right) + \left(R - R^{**}\right)\right)\left(\pi - \mu\left(S + R\right) - \left(\mu + \delta\right) I\right) + \frac{2\mu + \delta}{\beta}\left(1 - \frac{I^{**}}{I}\right)\left(\beta SI - K_1 I + \varepsilon R\right) + \frac{\varepsilon\left(2\mu + \delta\right) R^{**}}{\beta\theta I^{**}}\left(1 - \frac{R^{**}}{R}\right)\left(\theta I - K_2 R\right) + \frac{2\mu + \delta}{\theta}\left(R - R^{**}\right)\left(\theta I - K_2 R\right)$$

(7)

Using $\pi = \mu\left(S^{**}+R^{**}\right)+\left(\mu+\delta\right)I^{**}$, $K_1 = \beta S^{**}+\frac{\varepsilon R^{**}}{I^{**}}$, $K_2 = \frac{\theta I^{**}}{R^{**}}$ to simplify (7) as

$$\frac{dV_2}{dt} = -\left(\left(S-S^{**}\right)+\left(I-I^{**}\right)+\left(R-R^{**}\right)\right)\left(\mu\left(\left(S-S^{**}\right)+\left(R-R^{**}\right)\right)+\left(\mu+\delta\right)\left(I-I^{**}\right)\right)+$$

$$\frac{2\mu+\delta}{\beta}\left(I-I^{**}\right)\left(\beta\left(S-S^{**}\right)+\varepsilon\left(\frac{R}{I}-\frac{R^{**}}{I^{**}}\right)\right)+\frac{\varepsilon\left(2\mu+\delta\right)R^{**}}{\beta\theta I^{**}}\left(1-\frac{R^{**}}{R}\right)\left(\theta I-\frac{\theta R I^{**}}{R^{**}}\right)+$$

$$\frac{2\mu+\delta}{\theta}\left(R-R^{**}\right)\left(\theta\left(I-I^{**}\right)-K_2\left(R-R^{**}\right)\right)$$

$$\frac{dV_2}{dt} = -\left(\left(S-S^{**}\right)+\left(R-R^{**}\right)\right)^2-\left(2\mu+\delta\right)\left(I-I^{**}\right)\left(\left(S-S^{**}\right)+\left(R-R^{**}\right)\right)-\left(\mu+\delta\right)\left(I-I^{**}\right)^2+$$

$$\left(2\mu+\delta\right)\left(I-I^{**}\right)\left(S-S^{**}\right)+\frac{\varepsilon\left(2\mu+\delta\right)R^{**}}{\beta}\left(1+\frac{R}{R^{**}}-\frac{I}{I^{**}}-\frac{RI^{**}}{R^{**}I}\right)+\frac{\varepsilon\left(2\mu+\delta\right)R^{**}}{\beta}\left(1-\frac{R}{R^{**}}+\frac{I}{I^{**}}-\frac{IR^{**}}{I^{**}R}\right)$$

$$+\frac{2\mu+\delta}{\theta}\left(R-R^{**}\right)\left(\theta\left(I-I^{**}\right)-K_2\left(R-R^{**}\right)\right)$$

Thus after many tedious algebraic simplifications, we get

$$\frac{dV_2}{dt} = -\left(\left(S-S^{**}\right)+\left(R-R^{**}\right)\right)^2-\left(\mu+\delta\right)\left(I-I^{**}\right)^2-\frac{\varepsilon\left(2\mu+\delta\right)R^{**}}{\beta}\left(\sqrt{\frac{RI^{**}}{R^{**}I}}-\sqrt{\frac{IR^{**}}{I^{**}R}}\right)^2-\frac{K_2\left(2\mu+\delta\right)}{\theta}\left(R-R^{**}\right)^2$$

Thus, for $R_0>1, \dot{V}_2 \le 0$, where $\dot{V}_2 = 0$ holds only when $S = S^{**}, I = I^{**}$ and $R = R^{**}$. The only largest invariant set in $\left\{\left(S,I,R\right)\in\Omega : \dot{V}_2 = 0\right\}$ is the endemic equilibrium. Therefore the endemic equilibrium E_1 is globally asymptotically stable in the interior Ω, by LaSalle's invariance theorem principle (La Salle and Lefschetz,1961).

4.0 Numerical Simulation and Discussion

In this section, some numerical solutions of the model for different initial population sizes is presented using the various values of the parameters stated in Table.1 and to validate that these solutions are in agreement with the qualitative behaviours of the model obtained in section 2.Thus ,we choose different initial population sizes such that the total population, $S+I+R=250$ as follows

1____ $S\left(0\right)=200, I\left(0\right)=30, R\left(0\right)=20,$

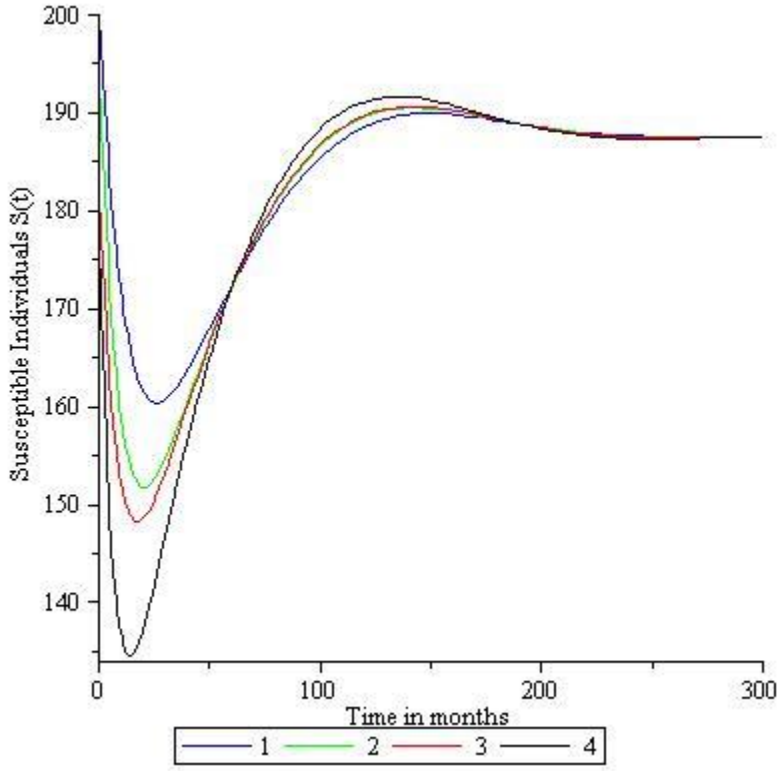
2____ $S\left(0\right)=195, I\left(0\right)=40, R\left(0\right)=15,$

3____ $S\left(0\right)=183.1, I\left(0\right)=43.6, R\left(0\right)=23.3,$

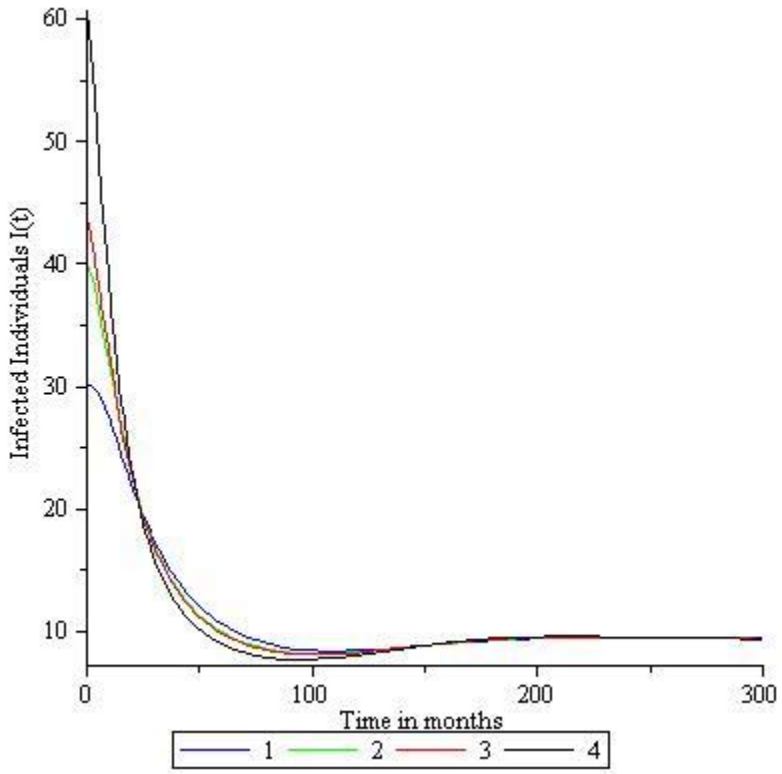
4____ $S\left(0\right)=179.38, I\left(0\right)=60.67, R\left(0\right)=9.95.$

In Fig.1, the three figures depict the numerical solution curve of the system (1) for $R_0 = 7.7219 > 1$. Figure 1(a) shows that the population of susceptible individuals $S\left(t\right)$ at first decreases, then it increases and later decreases to approach S^{**}. In figure 1(b), the population of

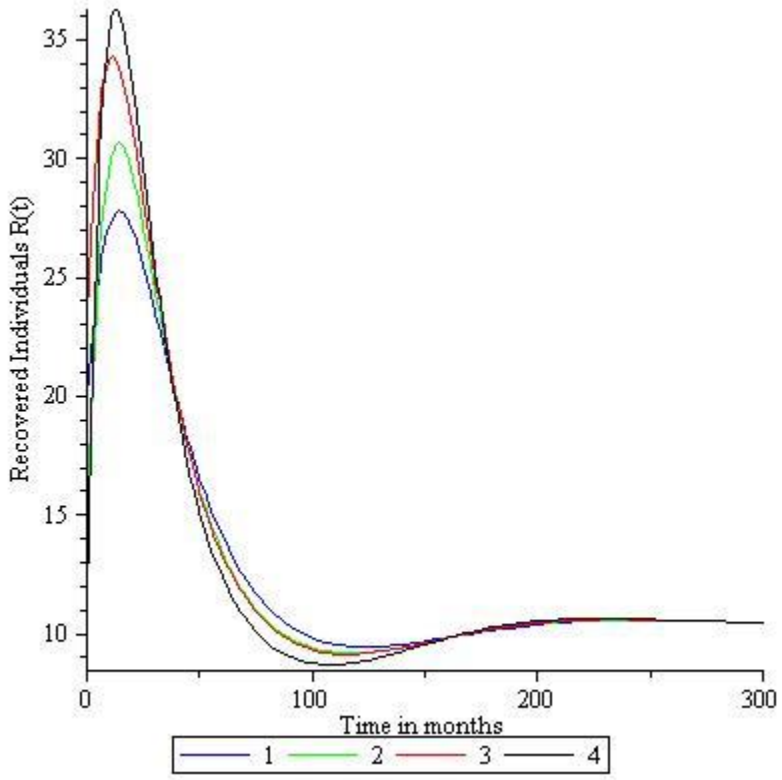
infected individuals $I(t)$ decreases at first, then it increases to approach I^{**}. In figure 1(c) the population of infective individuals $R(t)$ increases at first, then decreases and later increases to approach R^{**}. We note that the solution curves of these figures tends to the equilibrium E_1 for any initial values when $R_0 > 1$. Thus, the system (1) is locally-globally asymptotically stable about E_1 for the aforementioned parameter value.



(a)


Infected Individuals I(t)
60
50
40
30
20
10
0
100
200
300
Time in months
1
2
3
4

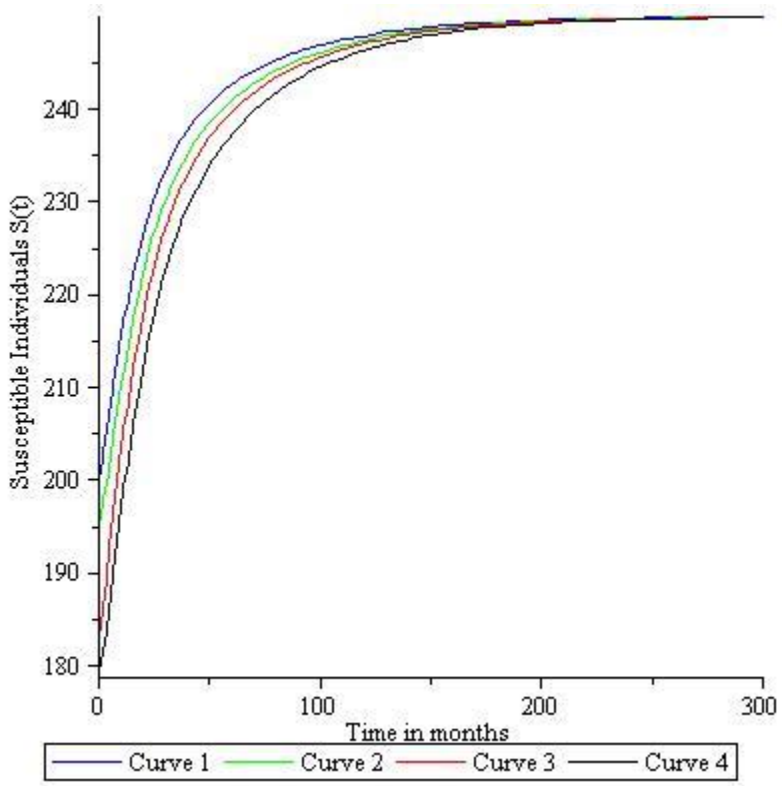
(b)


Recovered Individuals R(t)
35
30
25
20
15
10
0
100
200
300
Time in months
1
2
3
4

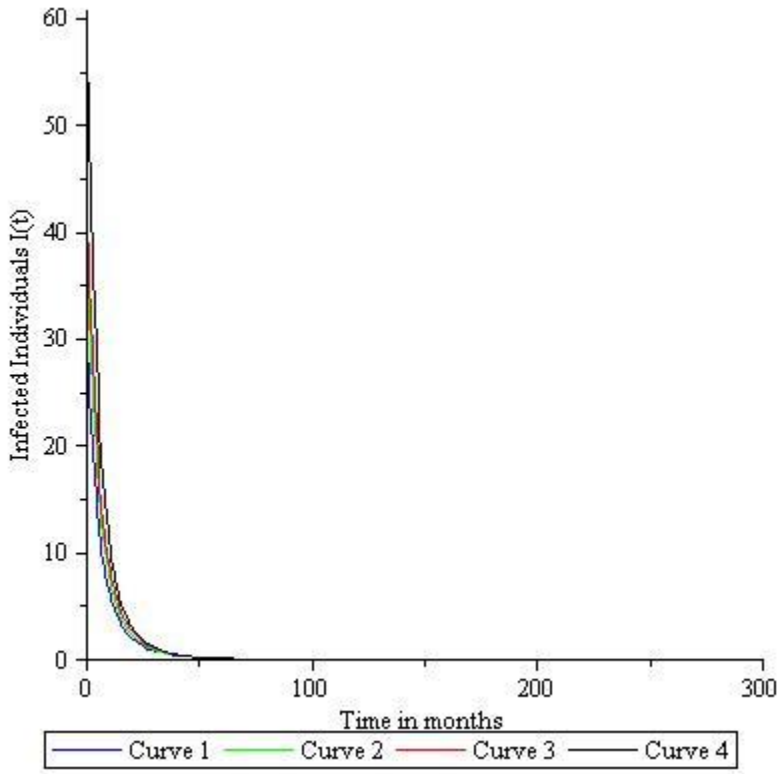
(c)

Fig. 1. Time plots of system (1) with different initial conditions for $R_0 > 1$: (a) Susceptible population; (b) Infected Population; (c) Recovered Population.

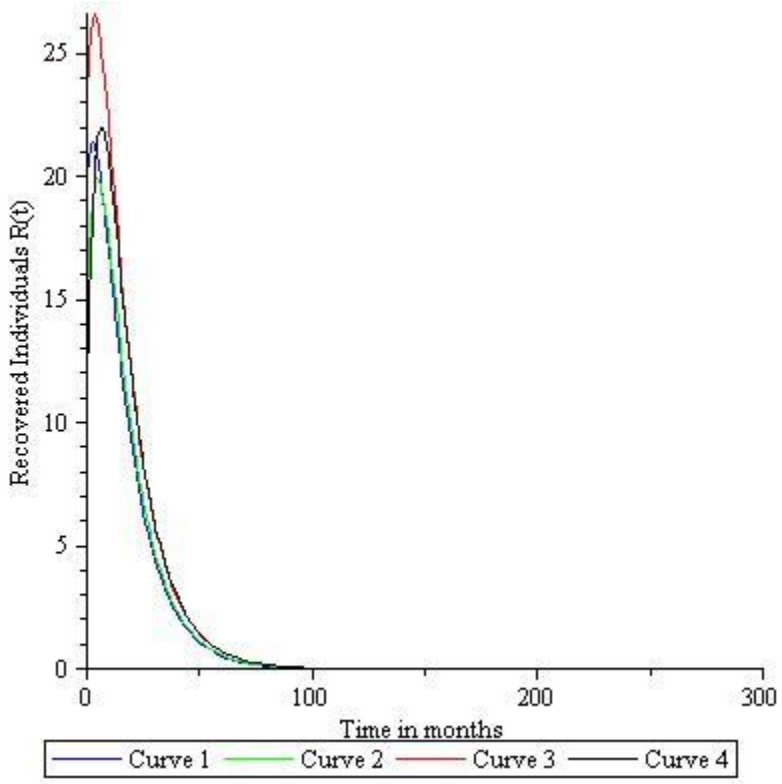
In Fig.2, the three figures depict the numerical solution curve of the system (1) for $\beta = 0.0001$ and $R_0 = 0.7722 < 1$. Figure 2(a) shows that the population of susceptible individuals $S(t)$ increases to approach S^* $\left(i.e. \frac{\pi}{\mu} = 250 \right)$. In figure 2(b), the population of infected individuals $I(t)$ decreases to to approach $I^*(i.e.\ zero)$. In figure 2(c) the population of recovered individuals $R(t)$ increases at first, then decreases to approach $R^*(i.e.\ zero)$. We note that the solution curves of these figures tends to the equilibrium E_0 for any initial values when $R_0 < 1$. Thus, the system (1) is locally-globally asymptotically stable about E_0 for the aforementioned parameter value.



(a)


Infected Individuals I(t)
60
50
40
30
20
10
0
0
100
200
300
Time in months
Curve 1
Curve 2
Curve 3
Curve 4

(b)


Recovered Individuals R(t)
25
20
15
10
5
0
0
100
200
300
Time in months
Curve 1
Curve 2
Curve 3
Curve 4

(c)

Fig. 2. Time plots of system (1) with different initial conditions for $R_0 < 1$: (a) Susceptible population; (b) Infected Population; (c) Recovered Population.

5.0 Conclusion

A three-dimensional deterministic mathematical model for the transmission dynamics of infectious diseases in the presence of relapse and immunity loss is formulated and rigorously studied using stability theory of nonlinear system. Some of the main epidemiological and mathematical findings are summarized as follows.

1. The model has a locally disease free equilibrium whenever the associated reproductive number R_0 is less than unity.
2. The disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.
3. The endemic equilibrium exist whenever $R_0 > 1$ and then locally asymptotically stable.
4. The model's endemic equilibrium is globally asymptotically stable whenever $R_0 > 1$.

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