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MATHEMATICAL MODEL OF AN SIR EPIDEMIC SWITCHING WITH ZERO CO-INFECTIVES

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ABSTRACT

This paper studies the global dynamics of an SIR epidemic switching model with zero co-infectives and intervention programmes. The model considers two epidemics of non-specific nomenclature in which the first epidemic is a precondition to the outbreak of the second epidemic. Analytical study of the model exposed the two epidemic steady states, namely, epidemic-free equilibrium (EFE) and epidemic endemic equilibrium (EEE). Both equilibrium states are shown to be globally attractive points with respect to the criteria of the basic reproduction number using Lyapunov stability theory. Some sufficient conditions on the model parameters are obtained to show the existence of the forward bifurcation. Finally, numerical simulations are done to exemplify the qualitative results and the impact of switching and intervention programmes. The numerical results shown that switching reduces the susceptibility and infectivity of the first epidemic and increases that of the second epidemic. Also, depending on the severity of the both epidemics, the different levels of intervention programmes are needed to reduce the number of infectives in both epidemics. However, equal intervention programmes are recommended for both epidemics to avoid neglecting one epidemic during outbreaks of the two epidemics.

Contribution/Originality: This study is one of the few studies in mathematical epidemiology which have investigated the role of switching in an SIR model of two epidemics with zero co-infectives. In addition, Lyapunov functions theory and Center Manifold method is applied to the model for the global stability analysis and existence of forward bifurcation respectively.

1. INTRODUCTION

Infectious diseases continue to present epidemic and pandemic challenges around the world. For instance, the emergence of the 2003 SARS epidemic [1], 2009 A/HINI influenza pandemic [2], the 2014-15 Ebola epidemic in West Africa [3] and the recent outbreak of COVID-19 pandemic in 2019 [4] globally are worth mentioning. In epidemiology, epidemic refers to an increase in the number of cases of a disease above an expected value in a population at a given time. An epidemic takes place when an agent and susceptible hosts are present in adequate numbers with effective contact rates while pandemic refers to an epidemic that has spread over several countries or continents, usually with large numbers of infectives [5].

Different compartmental models form a key aspect of mathematical epidemiology in which the majority of the past models of two epidemics are focused on coinfections [6]. These coinfection models include HIV and malaria co-infection [7] , Hepatitis B and HIV co-infection [8], HIV/TB co-infection [9] and listeriosis and anthrax co-

infection [10]. The instance of switching from one form of the epidemic to the other without co-infection is almost a negligible area of research.

The switching of an epidemic has to do with the transition process or movement from one infectious disease to another infectious disease, that is, the first epidemic is a precondition to the outbreak of the second. The classical view of switched systems is that they evolve according to the mode-dependent continuous dynamics and experience transition between modes which are triggered by certain events [11]. The abrupt change in the structure or parameters of a dynamical system and the control of a continuous system with a switch controller are the two reasons that result in a switched system [12].

Modelling of the epidemic switched system has not been widely explored. However, Meng and Deng [13] studied the stability of stochastic switched SIR epidemic systems with discrete or distributed time delay. They made use of Lyapunov function and Ito's differential rule for the analysis of stochastic switched systems and further proved that switching the system can eradicate the disease. A regime-switching SIR epidemic model with degenerate diffusion was investigated by Jin, et al. [14]. They established the asymptotic behavior of the system using Markov semigroup theory. Rami, et al. [15] investigated the spread of disease in an SIS epidemiological model for a structured population. Their model was an extension of Fall, et al. [16]. The model considered a time-varying switched model, in which the parameters of the SIS model were subject to abrupt change. The stability analysis results were derived from the joint spectral radius based on the R_0. The paper recommends the extension of the work to the SIR compartmental type of disease switched system. On the other hand, Wang, et al. [17] proposed the threshold dynamics of switched multicity epidemic models with pulse control. The model developed was switched HIV models with transported-related infections. The Razumikhin-type stability theory was employed to show that the disease will go to extinction based on the condition that $R_0 < 1$. Naji and Hussien [18] formulated an epidemic model that describes the dynamics of two types of infectious diseases with both horizontal and vertical transmissions. The local and global stability of the equilibrium points of the model was analysed. Both local bifurcations analysis and Hopf bifurcation analysis for the four-dimensional epidemic model was studied.

Looking at the suggestion made by Rami, et al. [15] to extend the modelling of epidemic switching from SIS to the Susceptible-Infected-Recovered (SIR) epidemic, we are motivated to propose a SIR epidemic switching without co-infection. In our model, the two epidemics considered are of the nonspecific type and epidemic 1 is a prerequisite to the epidemic 2.

The rest of the paper is organized as follows: Section 2, is the model formulation and invariant region of the system while the existence of the equilibrium states and computation of the basic reproduction number are presented in Section 3. In Section 4, numerical simulations are carried out to display the effect of the switching rate and intervention programmes on the two epidemics. The discussion of the numerical simulation is described in Section 5 while Section 6 is the conclusion.

2. MODEL FORMULATION AND THE CLOSED DOMAIN

The host population $N(t)$ is compartmentalized into six classes; namely, Susceptible for Epidemic 1 (S_1), Infectious for Epidemic 1 (I_1), Recovery for Epidemic 1 (R_1), Susceptible for Epidemic 2 (S_2), Infectious for Epidemic 2 (I_2), and Recovery for Epidemic 2 (R_2). The assumptions below and the flow diagram in Figure 1 have been adopted for the model derivation. Table 1 describes the parameters of the model. The following are the assumptions of the model.

i. The two epidemics are not co-infected.
ii. An individual can only be susceptible to one epidemic at the same time.
iii. Without being infected with Epidemic 1, one cannot be infected by Epidemic 2. (i.e. Epidemic 1 is a pre-requisite to Epidemic 2).
iv. Epidemic 1 can switch to Epidemic 2 but not vice versa.
v. Infected individuals for epidemic 1 with very low immunity switches fast to epidemic 2 while those that recovered permanently from epidemic 1 immediately become susceptible to epidemic 2.

Table-1. Parameter description of the Model.

Parameters	Description
b_1	The number of individuals that enter into the susceptible class of epidemic 1 either by birth or immigration.
β_1	The transmission rate for Epidemic 1.
b_2	The number of individuals that enter into the susceptible class of epidemic 2 either by birth or immigration.
β_2	The transmission rate for Epidemic 2.
σ_s	Switching parameter.
m_1, m_2	Intervention Programs for epidemic 1 and epidemic 2 respectively.
δ_1	Mortality rate due to infection for Epidemic 1.
δ_2	Mortality rate due to infection for Epidemic 2.
μ	Natural death rate
γ_1	The rate at which individuals infected with Epidemic 1 recovered.
γ_2	The rate at which individuals infected with Epidemic 2 recovered.
q	The rate at which individuals recovered from Epidemic 2 becomes susceptible.
p	Probability of acquiring high immunity.

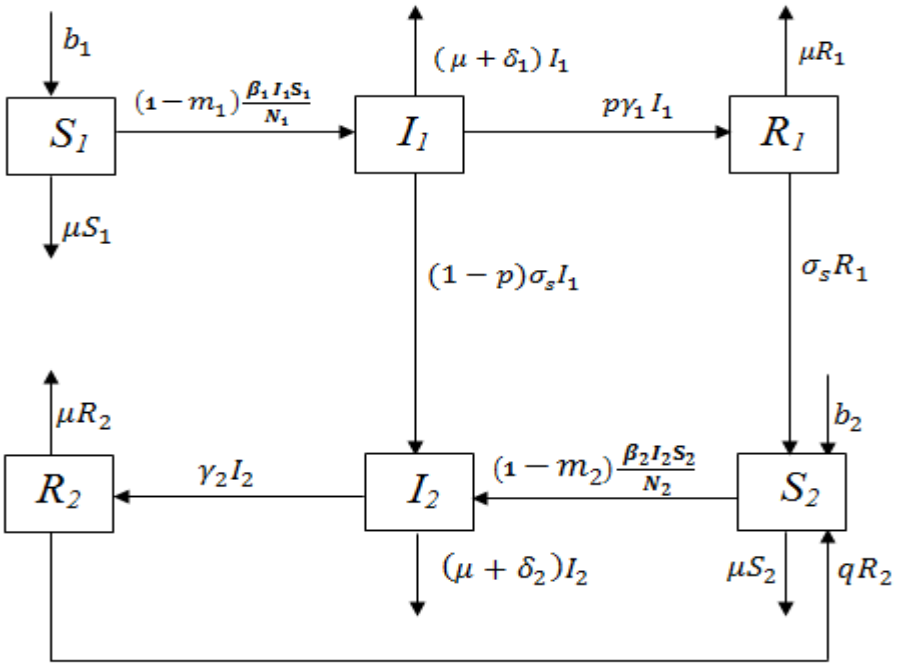


Figure-1. Flowchart for an SIR switching model without co-infection.

With the above assumptions, the set of the differential equation for the proposed SIR switched system is:

$$\left.\begin{aligned}
\frac{dS_1}{dt} &= b_1 - (1-m_1)\frac{\beta_1 I_1 S_1}{N_1} - \mu S_1 \\
\frac{dI_1}{dt} &= (1-m_1)\frac{\beta_1 I_1 S_1}{N_1} - (\mu + \delta_1 + p\gamma_1 + (1-p)\sigma_s) I_1 \\
\frac{dR_1}{dt} &= p\gamma_1 I_1 - (\mu + \sigma_s) R_1
\end{aligned}\right\} \text{epidemic 1}$$

$$\left.\begin{aligned}
\frac{dS_2}{dt} &= b_2 - (1-m_2)\frac{\beta_2 I_2 S_2}{N_2} - \mu S_2 + \sigma_s R_1 + qR_2 \\
\frac{dI_2}{dt} &= (1-m_2)\frac{\beta_2 I_2 S_2}{N_2} + (1-p)\sigma_s I_1 - (\mu + \delta_2 + \gamma_2) I_2 \\
\frac{dR_2}{dt} &= \gamma_2 I_2 - (\mu + q) R_2
\end{aligned}\right\} \text{epidemic 2}$$

(1)

with

$S_1(0) > 0, I_1(0) \geq 0,\ R_1(0) \geq 0,\ S_2(0) > 0,\ I_2(0) \geq 0, R_2(0) \geq 0,\ N(t) = N_1(t) + N_2(t),\ N_1(t) = S_1(t) + I_1(t) + R_1(t),$

and $N_2(t) = S_2(t) + I_2(t) + R_2(t)$.

Theorem 1. The closed domain $\bar{D} = \left\{(S_1, I_1, R_1, S_2, I_2, R_2) \in \mathbb{R}_+^6 : N \leq \frac{b_1+b_2}{\mu}\right\}$ is positive invariant and attractive to the system (1).

Proof. Assume $(S_1(t), I_1(t), R_1(t), S_2(t), I_2(t), R_2(t))$ to be any solution of the system (1) with any arbitrary initial condition. Then by summing together the entire system of Equation 1 yields:

$$\frac{dN}{dt} = b_1 + b_2 - \mu N(t) - \delta_1 I_1 - \delta_2 I_2 \tag{2a}$$

$$\leq b_1 + b_2 - \mu N(t)\,. \tag{2b}$$

By the standard comparison theorem [19], we obtain from Equations 2a and 2b that

$$N(t) \leq \frac{b_1+b_2}{\mu} + \left(N(0) - \frac{b_1+b_2}{\mu}\right) e^{-\mu t} \tag{3}$$

and consequently from Equation 3, it can be shown that

$$N(t) \leq \frac{b_1+b_2}{\mu}, \quad \text{when } N(0) \leq \frac{b_1+b_2}{\mu}. \tag{4}$$

Therefore, from Equation 4, it implies that $\bar{D}$ is a positive invariant. In addition, when $N(0) > \frac{b_1+b_2}{\mu}$, $N(t)$ approaches $\frac{b_1+b_2}{\mu}$ as $t \to \infty$. Hence, all solution in $\mathbb{R}_+^6$ approach, enter or stay in $\bar{D}$ (i.e. $\bar{D}$ is attracting).

From now onwards, it is sufficient to consider the dynamic of the switched system (1) in $\bar{D}$ since the equations are mathematically well-posed and epidemiologically sensible.

3. EXISTENCE OF THE EQUILIBRIUM STATES AND BASIC REPRODUCTION NUMBER

Two basic equilibrium states of the model (1) are investigated by setting the right-hand side of the model system to zero. The first equilibrium state is the epidemic-free equilibrium (EFE) that is represented by $E^0 = (S_1^0, 0, 0, S_2^0, 0, 0)$ with $S_1^0 = \frac{b_1}{\mu}$ and $S_2^0 = \frac{b_2}{\mu}$. Moreover, the basic reproduction number of the model system (1) denoted by R_0, is the maximum of the reproduction numbers computed using the Next-generation approach [20] related to each epidemic. We have the basic reproduction number, R_0, given as

$$R_0 = Max\,(R_{01}, R_{02}), \tag{5}$$

where

$$R_{01} = \frac{(1-m_1)\beta_1}{[\mu+\delta_1+p\gamma_1+(1-p)\sigma_s]} \tag{6}$$

and

$$R_{02} = \frac{(1-m_2)\beta_2}{(\mu+\delta_2+\gamma_2)} \tag{7}$$

R_{01} and R_{02} denote the reproduction numbers for epidemic 1 and epidemic 2 respectively in Equation 5.

3.1. Stability Analysis of Epidemic-Free Equilibrium State

Here, we examine the local asymptotic stability of system (1) at E^0 using the linearized stability theory.

Theorem 2. The epidemic-free equilibrium point of the model (1) is locally asymptotically stable if $R_0 < 1$.

Proof: The linearized form of the system (1) at the epidemic-free equilibrium state, E^0, is given by the Jacobian matrix, $J(E^0)$

$$J(E^0) = \begin{pmatrix} -\mu & -(1-m_1)\beta_1 & 0 & 0 & 0 & 0 \\ 0 & (1-m_1)\beta_1 - f_1 & 0 & 0 & 0 & 0 \\ 0 & p\gamma_1 & -g_1 & 0 & 0 & 0 \\ 0 & 0 & \sigma_s & -\mu & -(1-m_2)\beta_2 & q \\ 0 & (1-p)\sigma_s & 0 & 0 & (1-m_2)\beta_2 - f_2 & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & -g_2 \end{pmatrix}, \quad (8)$$

(8)

where

$$\begin{cases} f_1 = (\mu + \delta_1 + p\gamma_1 + (1-p)\sigma_s) \\ g_1 = (\mu + \sigma_s) \\ f_2 = (\mu + \delta_2 + \gamma_2) \\ g_2 = (\mu + q) \end{cases} \quad (9)$$

The eigenvalues of the Jacobian matrix of Equation 8 are

$$-\mu \text{ twice}, -g_i, i = 1,2, \frac{(1-m_1)\beta_1 S_1^0}{N_1^0} - f_1 = f_1(R_{01} - 1) \text{ and } \frac{(1-m_2)\beta_2 S_2^0}{N_2^0} - f_2 = f_2(R_{02} - 1). \quad (10)$$

The Equation 10 have negative eigenvalues if $R_{01} < 1$ and $R_{02} < 1$. This implies that the Epidemic-free equilibrium state, E^0, is locally asymptotically stable when $R_0 < 1$.

Hence, this completes the proof of Theorem 2.

3.2. Global Stability of Epidemic-Free Equilibrium State

The following theorem prove the global asymptotic stability of the system (1) at E^0 in the absence of a switching parameter.

Theorem 3. The model (1) exhibits a stable global asymptotic behaviour at epidemic-free equilibrium state when

$\sigma_s = 0$ and $R_0 \leq 1$

Proof: Using Lyapunov function adopted from Naji and Hussien [18]

$$L(\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2) = \sum_{i=1}^{2} I_i$$

The Lyapunov derivative along the trajectories of system (1) gives

$$\dot{L} = \left(\frac{(1-m_1)\beta_1 S_1}{N_1} - f_1^*\right) I_1 + \left(\frac{(1-m_2)\beta_2 S_2}{N_2} - f_2\right) I_2 \tag{11}$$

where $f_1^* = \mu + \delta_1 + p\gamma_1 \quad \text{at } \sigma_s = 0.$

Adding and subtracting of S_1^0 and S_2^0 in the first and second brackets respectively of Equation 11 and with little algebra yields

$$\dot{L} = \left(\frac{(1-m_1)\beta_1 S_1^0}{N_1^0} - f_1^*\right) I_1 - \left(\frac{(1-m_1)\beta_1 S_1^0}{N_1^0} - \frac{(1-m_1)\beta_1 S_1}{N_1}\right) I_1 + \left(\frac{(1-m_2)\beta_2 S_2^0}{N_2^0} - f_2\right) I_2 - \left(\frac{(1-m_2)\beta_2 S_2^0}{N_2^0} - \frac{(1-m_1)\beta_2 S_2}{N_2}\right) I_2$$

Since $\frac{S_1^0}{N_1^0} = \frac{S_2^0}{N_2^0} = 1$, and using Equations 6 and 7 at $\sigma_s = 0$, we have

$$\dot{L} = f_1^*(R_{01} - 1)I_1 + f_2(R_{02} - 1)I_2 - (1 - m_1)\beta_1\left(1 - \frac{S_1}{N_1}\right) I_1 - (1 - m_2)\beta_2\left(1 - \frac{S_2}{N_2}\right) I_2$$

But, $1 - \frac{S_1}{N_1} \geq 0, \text{ and } \quad 1 - \frac{S_2}{N_2} \geq 0.$

So,

$$\dot{L} \leq f_1^*(R_{01} - 1)I_1 + f_2(R_{02} - 1)I_2.$$

This implies that,

$$L' \leq 0 \quad \text{if } R_{01} \leq 1 \text{ and } R_{02} \leq 1 \quad (\Leftrightarrow R_0 \leq 1) \text{ and } L' = 0 \text{ when } I_1 = I_2 = 0.$$

Therefore, E^0 is a global attractive point and by LaSalle invariance principle [21] the epidemic-free equilibrium point is globally asymptotically stable whenever $R_0 \leq 1$ and $\sigma_s = 0$. This is shown graphically in Figure 2.

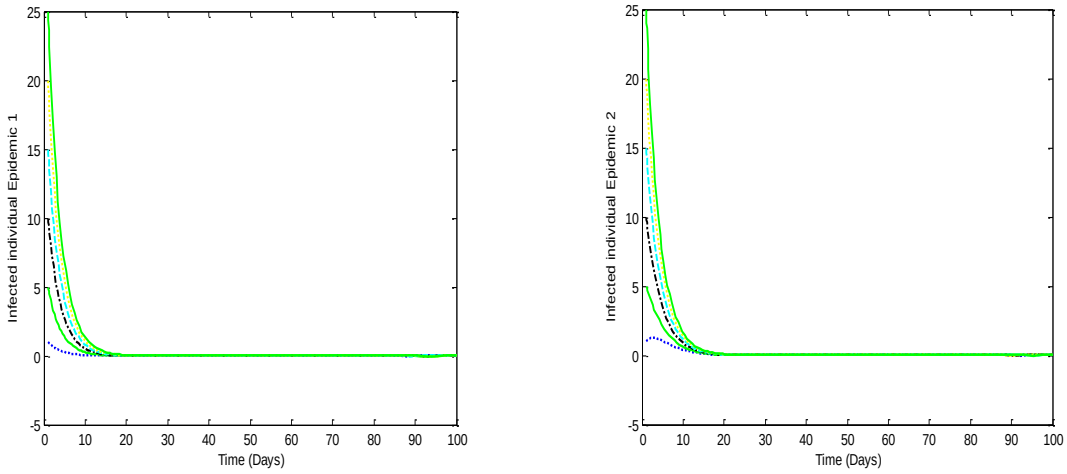


Figure-2. Global stability of the epidemic-free equilibrium state for the infected individuals in Epidemic 1 and 2.

The parameter values used in Figure 2 are

$$p = 0.3, \delta_1 = 0.1, \mu = 0.03, \gamma_1 = 0.3, \gamma_2 = 0.03, \sigma_s = 0.0, q = 0.3, m_1 = 0.2, m_2 = 0.2, b_1 = 20, b_2 = 10, \beta_1 = 0.05, \beta_2 = 0.05$$

with different initial conditions. (a) $\delta_2 = 0.3, R_0 = 0.08$. (b) $\delta_2 = 0.03, R_0 = 0.111.$

3.3. Existence and Stability of Epidemic Endemic State

The epidemic endemic equilibrium denoted by $\bar{E} = (\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2)$ can be obtained from the following:

$$\begin{cases} 0 = b_1 - (1 - m_1)\frac{\beta_1 I_1 S_1}{N_1} - \mu S_1 \\ 0 = (1 - m_1)\frac{\beta_1 I_1 S_1}{N_1} - f_1 I_1 \\ 0 = p\gamma_1 I_1 - g_1 R_1 \\ 0 = b_2 - (1 - m_2)\frac{\beta_2 I_2 S_2}{N_2} - \mu S_2 + \sigma_s R_1 + qR_2 \\ 0 = (1 - m_2)\frac{\beta_2 I_2 S_2}{N_2} + (1 - p)\sigma_s I_1 - f_2 I_2 \\ 0 = \gamma_2 I_2 - g_2 R_2 \end{cases} \quad (12)$$

From the second and third equations of Equation (12) when $I_1 \neq 0$, we have

$$f_1 = (1 - m_1)\frac{\beta_1 S_1}{N_1}$$

(13)

$$R_1 = \frac{p\gamma_1 I_1}{g_1}.$$

(14)

Substituting Equation (13) in the first equation of the system equation (12) gives

$$S_1 = \frac{b_1 - f_1 I_1}{\mu}.$$

(15)

Making use of the fact $N_1 = S_1 + I_1 + R_1$ in Equation (13), we get

$$f_1(I_1 + R_1) = (1 - m_1)\beta_1 S_1 - f_1 S_1 . \quad (16)$$

With S_1 of Equation (15) and R_1 of Equation (14) in Equation(16) and with little algebra gives

$$\bar{I}_1 = \frac{b_1 g_1 (R_{01} - 1)}{\mu(g_1 + p\gamma_1) + f_1 g_1 (R_{01} - 1)}.$$

(17)

Therefore, substituting Equation (17) into Equations (14) and (15) yields

$$\bar{R}_1 = \frac{p\gamma_1 b_1 (R_{01} - 1)}{\mu(g_1 + p\gamma_1) + f_1 g_1 (R_{01} - 1)} \quad \text{and} \quad \bar{S}_1 = \frac{b_1(g_1 + p\gamma_1)}{\mu(g_1 + p\gamma_1) + f_1 g_1 (R_{01} - 1)} .$$

Also, from the sixth and fifth equation of Equation (12), we get

$$R_2 = \frac{\gamma_2 I_2}{g_2}$$

(18)

and

$$\frac{(1 - m_2)\beta_2 I_2 S_2}{N_2} = f_2 I_2 - (1 - p)\sigma_s I_1.$$

(19)

Implementing Equations (14), (18), and (19) into the fourth equation of Equation (12) and simplifying yields

$$\bar{S}_2 = b_2 g_1 g_2 - g_1[q(\mu + \delta_2) + \mu f_2] I_2 + g_2(1 - p)\sigma_s \bar{I}_1.$$

(20)

Upon substitution of Equations (18) and (20) in (19) with $N_2 = S_2 + I_2 + R_2$ and a bit manipulation results to:

$$A\bar{I}_2 + B\bar{I}_2 - C = 0,$$

(21)

where

$$A = f_2 g_1 g_2 \delta_2 (R_{02} - 1) + \beta_2 g_1 \mu(\mu + \delta_2 + q)(1 - m_2) > 0 \quad if \quad R_{02} > 1,$$

$$B = g_1 g_2 \sigma_s \delta_2 \bar{I}_1 - f_2 g_2 [\sigma_s(\gamma_2 + g_1)\bar{I}_1 + b_2 g_1][R_{02} - 1],$$

$$C = g_2 {\sigma_s}^2 \delta_2 {\bar{I}_1}^2 (g_1 + p\gamma_1) + b_2 g_1 g_2 \sigma_s \bar{I}_1.$$

The Equation (21) is a quadratic polynomial equation with a positive solution

$$\bar{I}_2 = \frac{-B \pm \sqrt{(B^2 + 4Ac)}}{2A}$$

(22)

Hence, with Equation (22) the epidemic endemic equilibrium state, $\bar{E} = (\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2)$ exists when $R_{01} > 1$ and $R_{02} > 1$ which implies that $R_0 > 1$.

3.4. Local Stability of the Epidemic Endemic Equilibrium State

The local stability of the epidemic endemic equilibrium state is proved using the centre manifold theorem [22]. The theorem depends on the existence of bifurcation near $R_0 = 1$. The bifurcation can be forward or backward bifurcation. A forward bifurcation means that the endemic equilibrium is local asymptotically stable when $R_0 > 1$ and the disease-free equilibrium state is local asymptotically stable when $R_0 < 1$.

Since $R_0 = \max\{R_{01}, R_{02}\}$ and $R_0 < 1$, the bifurcation is considered in two ways:

i. when $R_{01} = 1, R_{02} < 1$. So that $R_0 = \max\{R_{01}, R_{02}\} = R_{01}$.

ii. when $R_{02} = 1, R_{01} < 1$. So that $R_0 = \max\{R_{01}, R_{02}\} = R_{02}$.

We rewrite Equation 1 by letting $S_1 = x_1, \quad I_1 = x_2, \; R_1 = x_3, \; S_2 = x_4, \; I_2 = x_5, \; R_2 = x_6$.

This yields

$$\left.\begin{aligned}
\frac{dx_1}{dt} &= b_1 - \frac{(1-m_1)\beta_1 x_1 x_2}{N_1} - \mu x_1 = k_1 \\
\frac{dx_2}{dt} &= \frac{(1-m_1)\beta_1 x_1 x_2}{N_1} - f_1 x_2 = k_2 \\
\frac{dx_3}{dt} &= p\gamma_1 x_2 - g_1 x_3 = k_3 \\
\frac{dx_4}{dt} &= b_2 - \frac{(1-m_2)\beta_2 x_4 x_5}{N_2} - \mu x_4 + (1-p)\sigma_s x_3 + q x_6 = k_4 \\
\frac{dx_5}{dt} &= \frac{(1-m_2)\beta_2 x_4 x_5}{N_2} + (1-p)\sigma_s x_2 - f_2 x_5 = k_5 \\
\frac{dx_6}{dt} &= \gamma_2 x_5 - g_2 x_6 = k_6
\end{aligned}\right\}$$

(23)

where f_1, g_1, f_2, g_2 are as defined in Equation (9), $N_1 = x_1 + x_2 + x_3$ and $N_2 = x_4 + x_5 + x_6$.

Theorem 4. The system (1) exhibits a forward bifurcation at $\mathrm{R}_0 = 1$. Hence, the endemic equilibrium state, $\bar{E}$ is locally asymptotically stable when $\mathrm{R}_0 > 1$ but close to 1.

Proof. This is proved in two ways as stated above using the concept of Castillo-Chavez and Song [22].

Case (i). when $\mathrm{R}_{01} = 1$, we choose $\beta_1 = \beta_1{}^*$ as the bifurcation parameter that occurs at $R_{01} = 1$. So from Equation 6, $\beta_1{}^* = \frac{f_1}{(1-m_1)}$.

The Jacobian matrix, $J(E^0)$, at EFE when $\beta_1 = \beta_1{}^*$ with $R_{01} = 1$ has a simple zero eigenvalue and negative eigenvalues in Equations (8) and (10).

Applying the Theorem 4.1 of Castillo-Chavez and Song [22] let $\boldsymbol{w} = (w_1, w_2, w_3, w_4, w_5, w_6)$ be the right eigenvector associated with the zero eigenvalue. Multiplying the Jacobian Matrix $J(E^0)$ of Equation (8) with $\boldsymbol{w}$ and equating to zero at $R_{01} = 1$, we have

$$w_1 = -\frac{(1-m_1)\beta_1{}^* w_2}{\mu},\ w_3 = \frac{p\gamma_1 w_2}{g_1},\ w_4 = \frac{(1-p)\sigma_s w_3 - (1-m_2)\beta_2 w_5 + q w_6}{\mu},\ w_5 = \frac{(1-p)\sigma_s}{((1-m_2)\beta_2 - f_2)} w_2 \quad ,$$

$$w_6 = \frac{\gamma_2}{g_2} w_3.$$

Similarly, the left eigenvector of the Jacobian $J(E^0)$ of (8) associated with the zero eigenvalue is given by $\boldsymbol{v} = (v_1, v_2, v_3, v_4, v_5, v_6)$ and it satisfies $\boldsymbol{v}.\boldsymbol{w} = 1$. Transposing Jacobian $J(E^0)$ first and multiply by $\boldsymbol{v}$, we have $\boldsymbol{v} = (0, v_2, 0,0,0,0)$.

The function $k_2 = \frac{(1-m_1)\beta_1 x_1 x_2}{N_1} - f_1 x_2$ will be used to find bifurcation coefficients or constants a and b from the system of Equation (23) since $v_2 > 0$. Note that a and b are calculated based on the relations given in Castillo-Chavez and Song [22]. The associated non-zero partial derivatives of k_2 at EFE, E^0 for the model are

$$\frac{\partial^2 k_2(E^0,\beta_1^*)}{\partial x_1 \partial x_8} = -\frac{(1-m_1)\beta_1^*}{N_1}, \quad \frac{\partial^2 k_2(E^0,\beta_1^*)}{\partial x_2^*} = -\frac{2(1-m_1)\beta_1^*}{N_1}, \quad \frac{\partial^2 k_2(E^0,\beta_1^*)}{\partial x_2 \partial \beta_1} = (1-m_1).$$

Substituting the respective partial derivatives into a and b using the properties that $v_2.w_2 = 1$, we have after simplifying that

$$a = -v_2 w_2{}^2 \frac{(1-m_1)\beta_1^*}{N_1}\left(\frac{g_1+2p\gamma_1}{g_1}\right) \quad \text{and} \quad b = v_2 w_2 (1-m_1). \tag{24a}$$

Case (ii). When $R_{02} = 1$, $\beta_2 = \beta_2{}^*$ is chosen as the bifurcation parameter that occurs at $R_{02} = 1$. So from Equation 7, $\beta_2{}^* = \frac{f_2}{(1-m_2)}$. Following this same method for case (i), we have $\boldsymbol{w} = \left(0,0,0,0,w_5,\frac{\gamma_2}{g_2}w_5\right)$ and $\boldsymbol{v} = (0,v_2,0,0,v_5,0)$. The Jacobian matrix, $J(E^0)$ of Equation (8), at EFE when $\beta_2 = \beta_2{}^*$ with $R_{02} = 1$ has a simple zero eigenvalue and negative eigenvalues in Equation 10.

The function, $k_5 = \frac{(1-m_2)\beta_2 x_4 x_5}{N_2} + (1-p)\sigma_s x_2 - f_2 x_5$ is used to find a and b from the system Equation (23) since $w_2 = 0$. The associated non-zero partial derivatives of k_5 at EFE, E^0 for the model are given by

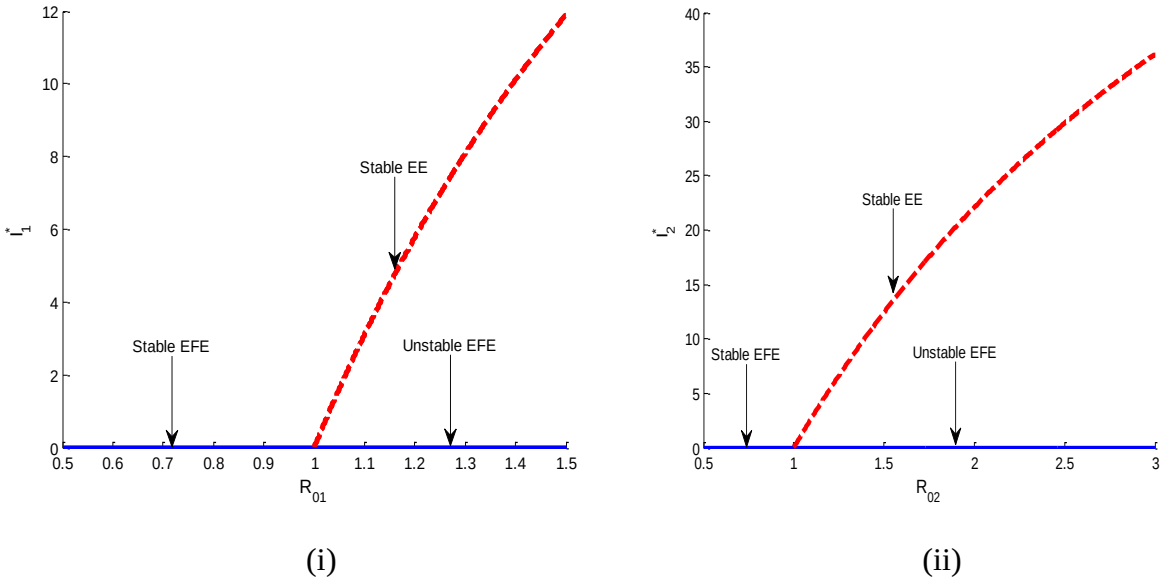
$$\frac{\partial^2 k_5(E_0,\beta_2^*)}{\partial x_5 \partial x_6} = -\frac{(1-m_2)\beta_2^*}{N_2}, \quad \frac{\partial^2 k_5(E_0,\beta_2^*)}{\partial x_5^*} = -\frac{2(1-m_2)\beta_2^*}{N_2}, \quad \frac{\partial^2 k_5(E_0,\beta_2^*)}{\partial x_5 \partial \beta_2} = (1-m_2).$$

Upon substituting the respective partial derivatives into a and b using the properties that $v_5.w_5 = 1$ yields

$$a = -v_5 w_5{}^2 \frac{(1-m_2)\beta_2^*}{N_2}\left(\frac{g_2+2\gamma_2}{g_2}\right) \quad \text{and} \quad b = v_5 w_5 (1-m_2). \tag{24b}$$

Since $v_2 w_2 > 1$ and $v_5 w_5 > 1$, we have from Equations (24a) and (24b) that $b > 0$ and $a < 0$ in both ways. Therefore, by Theorem 4.1 of Castillo-Chavez and Song [22] which stated that a forward bifurcation exists if $a < 0$ and $b > 0$. This implies that local asymptotic stability of endemic equilibrium state exists for $R_0 > 1$ but close to 1.

The above proved cases are graphically illustrated in Figure 3.



(i) (ii)

Figure-3. Forward bifurcation plot displaying (i) I_1^* as a function of R_{01} and (ii) I_2^* as a function of R_{02}.

Here, the parameter values used in Figure (3) are

$$p = 0.3, \delta_1 = 0.1, \delta_2 = 0.1, \mu = 0.3, \gamma_1 = 0.3, \gamma_2 = 0.3, \sigma_s = 0.01, q = 0.6, m_1 = 0.2, m_2 = 0.01, b_1 = 20, b_2 = 10, \beta_1 = 0.75, \beta_2 = 0.75$$

3.5. Global Stability of the Endemic Equilibrium State

Theorem 5. The epidemic endemic equilibrium state, $\bar{E}$ of the system (1) is globally asymptotically stable in $\bar{D}$ if $I_1 \leq \bar{I}_1, I_2 \leq \bar{I}_2$, and $R_{01}, R_{02} > 1$.

Proof: To prove this theorem, the work by Gupta, et al. [23] is used to construct a Lyapunov function L_f of the kind:

$$L_f(S_1, I_1, R_1, S_2, I_2, R_2) = \frac{U^2}{2} + c_1\left(I_1 - \bar{I}_1 - \bar{I}_1 \ln\frac{I_1}{\bar{I}_1}\right) + c_2\left(I_2 - \bar{I}_2 - \bar{I}_2 \ln\frac{I_2}{\bar{I}_2}\right), \quad (25)$$

where $U = (S_1 - \bar{S}_1) + (I_1 - \bar{I}_1) + (R_1 - \bar{R}_1) + (S_2 - \bar{S}_2) + (I_2 - \bar{I}_2) + (R_2 - \bar{R}_2)$

and c_1 and c_2 are the positive constants to be determined.

The time derivative of L_f in Equation (25) gives

$$L_f' = U(b_1 + b_2 - \mu(S_1 + I_1 + R_1 + S_2 + I_2 + R_2) - \delta_1 I_1 - \delta_2 I_2) + c_1\left(1 - \frac{\bar{I}_1}{I_1}\right)\left[(1 - m_1)\frac{\beta_1 I_1 S_1}{N_1} - f_1 I_1\right] + c_2\left(1 - \frac{\bar{I}_2}{I_2}\right)\left[(1 - m_2)\frac{\beta_2 I_2 S_2}{N_2} + \sigma_s I_1 - f_2 I_2\right]$$

. (26)

At equilibrium state,

$$b_1 + b_2 = \mu(\bar{S}_1 + \bar{I}_1 + \bar{R}_1 + \bar{S}_2 + \bar{I}_2 + \bar{R}_2) + \delta_1 \bar{I}_1 + \delta_2 \bar{I}_2. \quad (27)$$

So, substituting Equation (27) into Equation (26) gives

$$L_f' = U[-\mu(S_1 - \bar{S}_1) - \mu(S_2 - \bar{S}_2) - \mu(R_1 - \bar{R}_1) - \mu(R_2 - \bar{R}_2) - (\mu + \delta_1)(I_1 - \bar{I}_1) - (\mu + \delta_2)(I_2 - \bar{I}_2)] + c_1\left(1 - \frac{\bar{I}_1}{I_1}\right)\left[R_{01}\frac{S_1}{N_1} - 1\right] I_1 + c_2\left(1 - \frac{\bar{I}_2}{I_2}\right)\left[R_{02}\frac{S_2}{N_2} - 1\right] I_2$$

. We define $\eta =$ mean value of $(\mu,\ \mu + \delta_1,\ \mu + \delta_2)$ and $c_1 = c_2 = 1$ so that

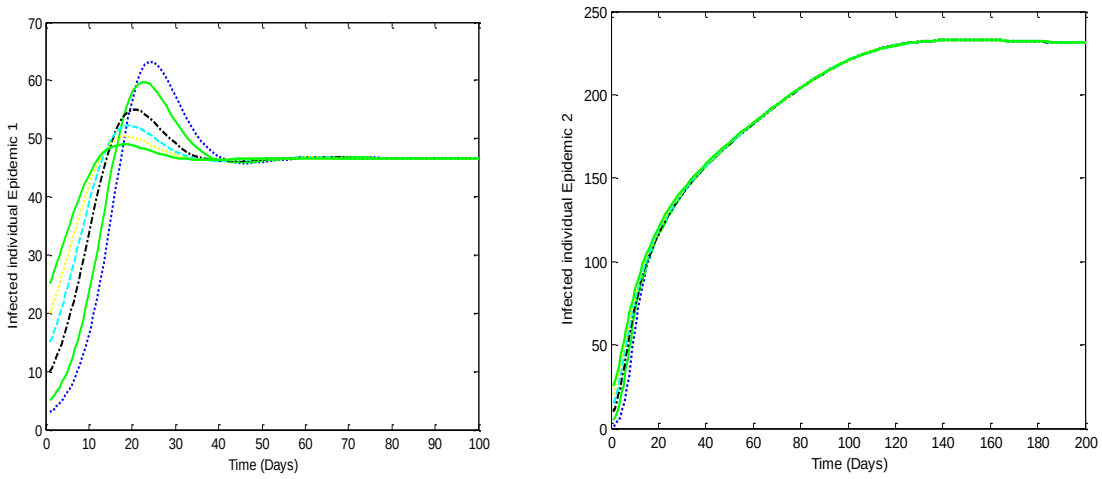
$$L_f' = -\eta U^2 + \left(1 - \frac{\bar{I}_1}{I_1}\right)\left[R_{01}\frac{S_1}{N_1} - 1\right] I_1 + \left(1 - \frac{\bar{I}_2}{I_2}\right)\left[R_{02}\frac{S_2}{N_2} - 1\right] I_2$$

Using the hypothesis that $I_1 \leq \bar{I}_1$ and $I_2 \leq \bar{I}_2$ yields

$L_f' < 0$ if $R_{01}, R_{02} > 1$ since $\frac{S_2}{N_2} \leq 1$ and $\frac{S_1}{N_1} \leq 1$.

On the other hand, $L_f' = 0$ if $S_1 = \bar{S}_1,\ I_1 = \bar{I}_1, R_1 = \bar{R}_1,\ S_2 = \bar{S}_2, I_2 = \bar{I}_2,$ and $R_2 = \bar{R}_2$.

Thus, by Lasalle invariant principle [21] the epidemic endemic equilibrium state, $\bar{E}$, is globally asymptotically stable when R_{01} and $R_{02} > 1$ which implies that $R_0 > 1$. Graphically, this is shown in Figure 4.



(a) (b)

Figure-4. Global stability of the epidemic endemic equilibrium state for the infected individuals in Epidemic 1 and 2.

The parameter values used in Figure 4 are

$$p = 0.3, \mu = 0.03, \gamma_1 = 0.3, \gamma_2 = 0.03, \sigma_s = 0.2, q = 0.3, m_1 = 0.2, m_2 = 0.3, b_1 = 20, b_2 = 10,$$

with different initial conditions. (a) $\beta_1 = 0.75, \beta_2 = 0.5, \delta_2 = 0.3, R_0 = 1.21.$ (b)

$\beta_1 = 0.5, \beta_2 = 0.75, , \delta_2 = 0.03, R_0 = 1.67.$

4. NUMERICAL SIMULATIONS

In this section, the numerical simulation of the system (1) is carried out using the set of parameters values given in Table 2. This is to study the impact of switching, intervention programmes and the proportion of infected people developing immunity on the epidemiology of infectious diseases. Some of the parameters' values are taken from Literature while some are assumed. The fourth-order Runge-Kutta scheme is used to solve the system (1).

Table-2. Parameter Values.

Parameters	Values	Source	Parameters	Values	Source
b_1	20	[18]	δ_1	0.1	[18]
b_2	10	Assumed	δ_2	0.1	[18]
β_1	0.75	[18]	μ	0.3	[18]
β_2	0.75	Assumed	γ_1	0.3	Assumed
σ_s	0.2	Assumed	γ_2	0.3	Assumed
m_1	(0,1)	Assumed	q	0.3	[18]
m_2	(0,1)	Assumed	p	(0,1)	[18]

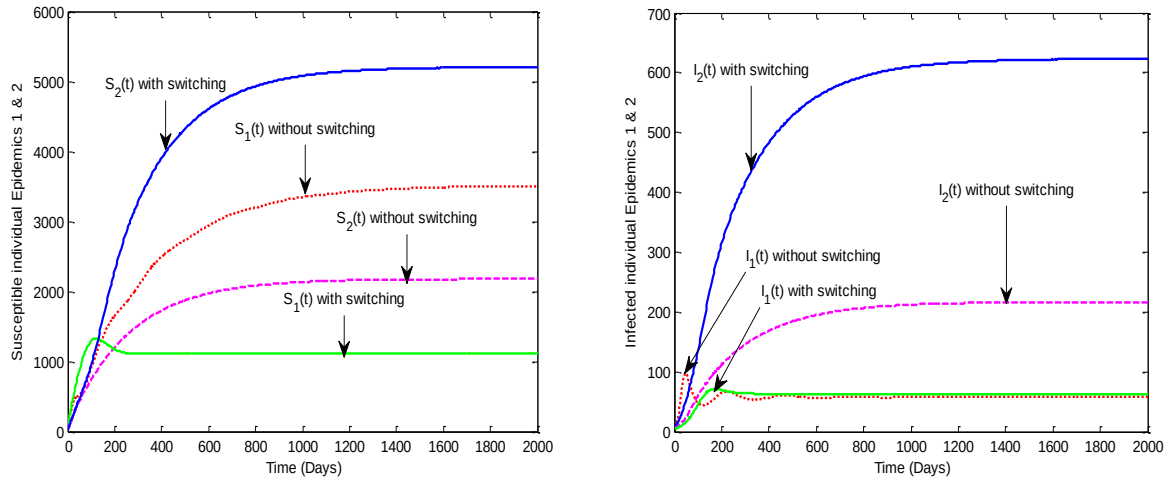


Figure-5. Simulation solution of the model (1) for the impact of switching on the Susceptible and Infected populations of the epidemic 1 and 2.

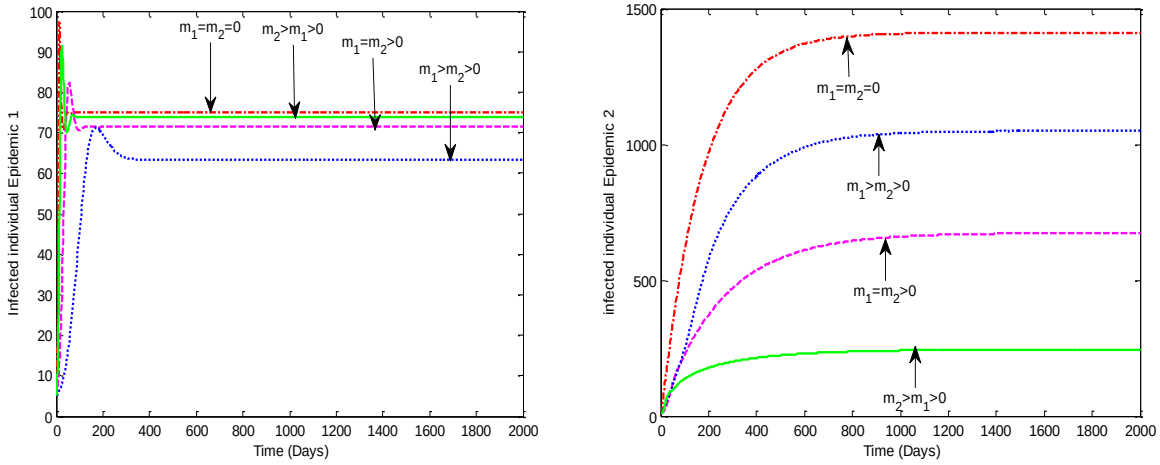


Figure-6. Simulation solution of the model (1) for the impact of interventional programmes on the infected population of the epidemic 1 and 2.

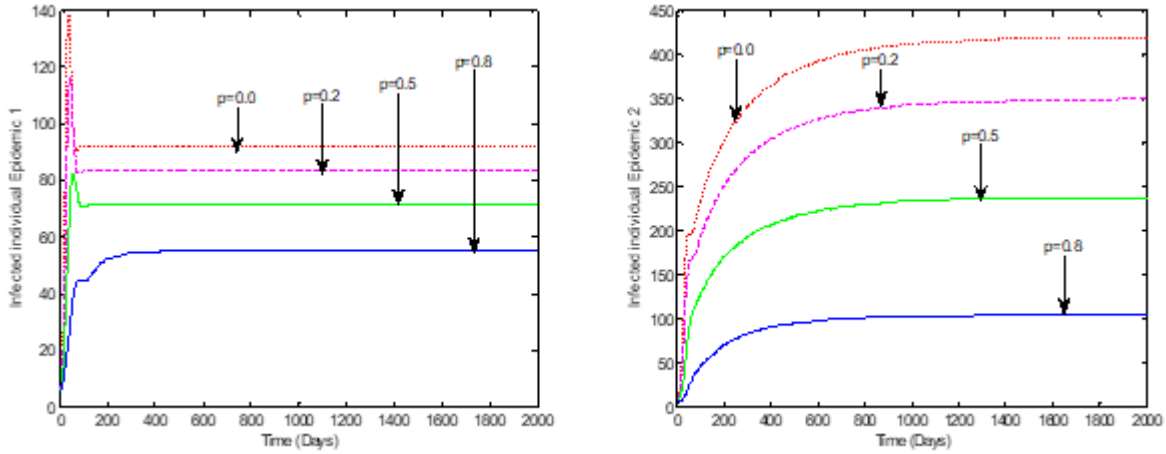
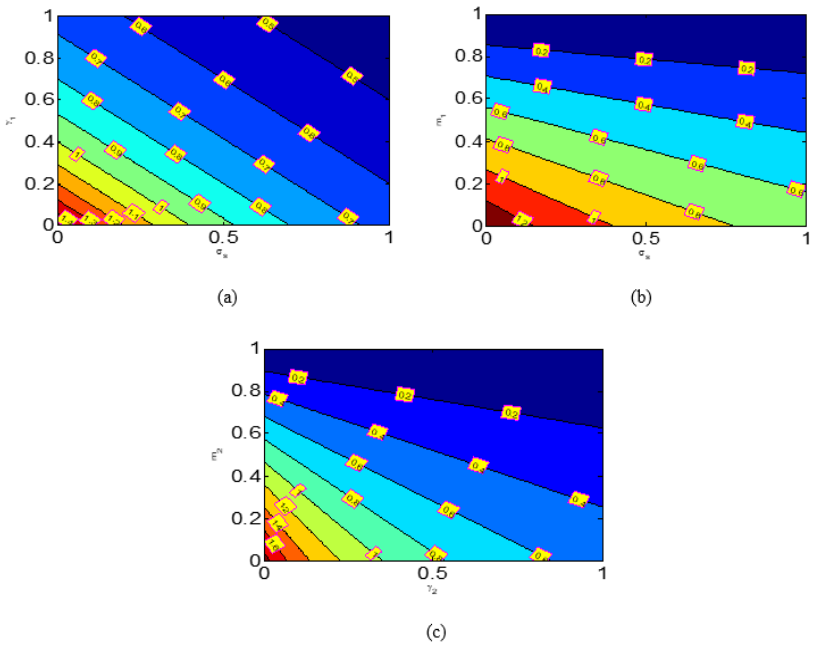


Figure-7. Simulation solution of the model (1) for the impact of interventional programmes on the infected population of the epidemic 1 and 2.



(a) (b)

(c)

Figure-8. Contour plot of the reproduction numbers for epidemics 1 and 2 (R_{01}, R_{02}). (a) R_{01} as a function of switching (σ_s) and recovery, (γ_1) (b) R_{01} as a function of switching (σ_s) and intervention progamme, (m_1). (c) R_{02} as a function of the intervention programme (m_2) and recovery, (γ_2).

5. DISCUSSION

Figure 5 demonstrates the impact of switching on the transmission dynamics of two epidemics. It is observed that when switching rate is increasing, the susceptible and infected populations for epidemic 1 decrease while the susceptible and infected population for epidemic 2 increases. This contributes to the endemicity of epidemic 2. It implies that the presence of switching in the epidemiology of the two epidemics reduces the susceptibility of the first epidemic and increases that of the second epidemic. Also, the process of transiting from epidemic 1 to 2 eliminates the infectivity tendencies of the first epidemic. This coincides with the result of Meng and Deng [13] that switching eradicates the spread of disease.

The impact of the intervention programmes (m_1, m_2) on the infected populations of epidemic 1 and 2 are illustrated in Figure 6. The intervention programmes are meant to minimize the transmission rates of the two epidemics. It is observed that when the intervention programme for epidemic 1 is given more priority over the intervention programme for epidemic 2 $(m_1 > m_2 > 0)$, it reduces the number of infectives of epidemic 1 as regards to epidemic 2. Conversely, the same argument holds for epidemic 2 when the intervention programme for epidemic 2 is given more priority over the intervention programme for epidemic 1 $(m_2 > m_1 > 0)$, it reduces remarkably the number of infectives for epidemic 2. However, when the intervention efforts of both epidemics are at an equal level $(i.e.\ m_1 = m_2 > 0)$, it reduces the number of the infected individuals of both epidemics but they are above when $m_1 > m_2 > 0$ for epidemic 1 and when $m_2 > m_1 > 0$ for epidemics 2. This implies that different levels of intervention programmes are needed to reduce the number of infectives in both epidemics that is the epidemic 1 requires intervention programme more than that of epidemic 2 to reduce its number of infectives while the epidemic 2 requires intervention programme greater than that of epidemic 1 to lower its number of infectives. However, we advise an equal level of intervention programmes for both epidemics so that one epidemic will not be neglected during another epidemic.

Figure 7 shows that the more infected individuals of epidemic 1 develop strong immunity, the more it lowers the number of infectives that switch to epidemic 2 and hence reduces the prevalence of both epidemics. This implies that any drugs/medications that can boost the immunity of the infected individuals in epidemic 1 will also help to reduce the infectives of epidemic 2.

Figure 8 displays the relationship between the reproduction numbers for epidemics 1 and 2. The reproduction number for epidemic 1 is less than unity, $(R_{01} < 1)$ in two ways: i. when the intervention programme for epidemic 1 is greater than 24% $(m_1 > 24\%)$, the recovery rate for infected individuals in epidemic 1 is greater than 0.4, $(\gamma_1 > 0.4)$ and switching rate is greater than or equal to 0.1 $(\sigma_s \geq 0.1)$ and ii. when $m_1 > 5\%, \sigma_s \geq 0.4$ and $\gamma_1 > 0.1$ (see Figure 8 a and b). For epidemic 2, the reproduction number for epidemic 2 is less than unity, $(R_{02} < 1)$ in two ways: when the intervention programme for epidemic 2 is greater than 36% $(m_2 > 36\%)$ and the recovery rate for infected individuals in epidemic 2 is greater than 0.15, $(\gamma_2 > 0.15)$ or when $m_2 > 2.5\%$

and $\gamma_2 > 0.35$ (see Figure 8a). For epidemic 1, it will be easy to achieve low switching rate, $(\sigma_s = 0.1)$ by increasing recovery rate and its intervention programme since low switching rate will reduce the number of infectives in epidemic 2 (see Figure 5). Also, the epidemic 2 requires high intervention programme and high recovery rate to eliminate the disease in epidemic 2. Thus, high intervention programmes and high recovery rates for the two epidemics are needed to bring the reproduction numbers of the two epidemics less than unity. Also, to have low switching rate means that more infectives in epidemic 1 will have to develop strong immunity by taking supplement and drugs that will boost their immunities when they recovered.

6. CONCLUSION

In this paper, we presented an SIR epidemic switched model and studied the dynamics of two infections with switching condition and intervention programmes. The analytical results of the model shown that there exists only two non-negative equilibrium points; the epidemic- free equilibrium (EFE) in the case of no infection and the epidemic-endemic equilibrium (EEE) denoting the presence of infection in the population. The EFE is locally asymptotically stable if the basic reproduction number is less than unity and globally stable when $R_0 \leq 1$ in the absence of switching parameter. Furthermore, the model undergoes forward bifurcation at $R_0 = 1$ in two ways: i. when the reproduction number for epidemic 1, $R_{01} = 1$, and the reproduction number for epidemic 2, $R_{02} < 1$ with $\beta_1 = \beta_1^*$ as the bifurcation parameter; ii. $R_{01} < 1, R_{02} = 1$, with the bifurcation parameter $\beta_2 = \beta_2^*$. Any of both conditions changes the stability behaviour of the system from stable to unstable around E^*. The analysis of the model shows that an epidemic endemic equilibrium is globally asymptotically stable whenever $R_0 > 1$. This gives the restriction of the epidemics within the endemic domain. The other results achieved (both analytical and numerical) suggest that the value of the reproduction numbers, R_{01} and R_{02}, can be less than one by increasing both the intervention programmes and the immunity rate. In addition, switching reduces the susceptibility and infectivity of the first epidemic and increases that of the second epidemic. Moreso, it emerges from our study that different levels of intervention programmes are needed for both epidemics. However, for one epidemic not to record more infectives, we advise equal intervention programmes for both epidemics. For future research, suitable epidemics that share similar characteristics to the dynamics of the proposed model can be applied.

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Acknowledgement: All authors contributed equally to the conception and design of the study.

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