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On the Effects of Saturation Terms on A SEIR Epidemic Model with Infected and Susceptible Compartments

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ABSTRACT

The importance of the saturation term in an SEIR (Susceptible, Exposed, Infected, and Recovered) epidemic model was examined in this article. To estimate the basic reproduction number (R_0), examine the stabilities and run numerical simulations on the model, the next generation matrix, the Lyapunov function and Runge-Kutta techniques were used. The numerical simulation results reveal that, the saturation term has a significant influence in the model's susceptible and infected compartments. However, as demonstrated by the simulation results, saturation term has a greater influence on vulnerable people than on infected people. As a result, greater sensitization programs through seminars, media, and awareness will be more beneficial to the vulnerable class than the afflicted class during disease eradication.

Keywords:
Basic Reproduction Number; Local and Global Stabilities; Disease Free Equilibrium; Numerical Simulations

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1. Introduction

Epidemiology problems are usually formulated with mathematical models. These models are often applied to track the transmission dynamics of various diseases. Research on a mathematical model for the effective management of HIV infection was carried out in [1]. A paper on the numerical simulation of the SEIRS epidemic model with a saturated incidence rate considering the saturation term for susceptible individuals was presented in [2]. In their research, the disease-free and endemic equilibrium points are established. The basic reproduction number was derived using the next generation matrix. The local stability and global stability of both the disease-free and endemic equilibriums were also obtained using the next generation matrix. A conceptual investigation of the disease transmission coefficient was equally conducted in [3]. In their research, the Laplace Adomian decomposition method was

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applied to carry out the numerical simulation of a SEIR epidemic model. Their research proved that the technique applied is an efficient one and the disease-free equilibrium is a vital parameter to be considered during disease eradication. Similarly, the effect of the disease transmission coefficient on a disease-induced death SEIR epidemic model was conducted using the homotopy perturbation method in [4]. Their result showed that the homotopy perturbation method is an effective and productive method that distances itself, employ challenging computational work than other numeral methods.

The saturation term plays an important role in the spread of infectious diseases. Numerous researchers have worked on the impact of saturation terms in the eradication of diseases. For example, the analysis of saturation terms in mathematical models for malaria transmission was studied in [5]. They concluded in their research that increasing the saturation term by reducing the infection rate between humans and vectors through proper sensitization by health workers and using active anti-malaria drugs can reduce the prevalence of malaria. The law of mass action and saturation term in a SIR model with application to coronavirus modelling were studied in [6]. They concluded in their research that the saturation term is sufficient to capture the disease dynamics for many jurisdictions, including the overall world-wide disease curve progression. A Mathematical Model and Analysis of an SVEIR Model for Streptococcus Pneumonia with Saturated Incidence Force of Infection was studied in [7]. Their study indicated that improving the efficiency and enlarging the capacity of treatment are efficient ways to control the spread of disease. A mathematical analysis of the diarrhea model with a saturated incidence rate was studied in [8]. In their research, they presented a compartmental mathematical model of (SITR) to investigate the effect of saturation treatment on the dynamical spread of diarrhea in the community. Their study revealed that if drugs were made available to consumers at a saturation treatment rate of 99% at a very low cost on time, there would be a reduction in the dynamic spread of diarrhea in a community. A simple epidemiological model for typhoid with a saturated incidence rate and treatment effect was presented in [9]. They analyzed the impact of the saturation term on the incidence rate. Their research revealed that typhoid fever could be eradicated if control strategies associated with saturation parameters are increased. A biological mathematical model of vector-host disease with saturated treatment function and optimal control strategies was presented in [10]. In their research, they explored the dynamics of vector-host disease with a saturated treatment function. Their study showed that the best possible strategy that can minimize the number of infected humans is the application of their speculated control methods simultaneously. Advancing the research work in [10], an optimal control analysis of a vector-host model with saturated treatment was studied in [11]. Their research suggested different control intervention strategies useful during disease eradication.

Often, researchers usually model and analyze the effect of saturation term on incidence rate. For example, a research on the nonstandard numerical discretization of the SIR epidemic model with saturated incidence rate and vaccination was conducted in [12]. In their research, they proposed and analyzed an alternative nonstandard finite difference scheme by applying nonlocal approximation. It was verified that the proposed model is dynamically consistent with the corresponding continuous model. In advancing research on curbing the spread of infectious diseases, a paper on the mathematical modelling and analysis of an epidemic model with nonlinear incidence and general recovery functions was carried out in [13]. In addition, authors in [14] investigated the optimal control of a delayed SIRC epidemic model with a saturated incidence rate. The

analysis and optimal control of a multi-strain SEIR epidemic model with saturated incidence rate and treatment was carried out in [15]. The results of their research showed that with waning immunity in the absence of mitigating measures, each viral strain will reach an equilibrium after the peak of infections. In a paper presented in [16], the complex dynamics of a SEIR epidemic model with saturated incidence rate and treatment were studied. They analyzed the dynamics of the model through stability and bifurcation. The global stability of their endemic equilibrium was obtained using the geometric approach and the optimal control problem was designed. The dynamical analysis of a modified epidemic model with a saturated incidence rate and incomplete treatment was conducted in [17]. In their paper, a modified epidemic model with saturated incidence and incomplete treatment were addressed. The existence of all equilibrium points and stability analysis of the model were analyzed and different numerical methods were applied to the presented mathematical model to justify the conducted analysis. A mathematical model for Covid-19 disease transmission dynamics with the impact of saturated treatment, modeling, analysis and simulation were investigated in [18]. It was concluded in their research that decreasing the transmission rate for infectious alone is not sufficient to eradicate the disease because of the presence of backward bifurcation. They recommended that adherence to COVID-19 protocols can be helpful in mitigating the spread and demise of coronavirus. In [5],the impact of saturation term on malaria transmission was investigated. The basic reproduction number of the modified models was obtained using a next generation matrix. The stability of both the disease-free and endemic equilibriums were established. The results obtained via numerical simulation reveal that for proper treatment and eradication of malaria, the saturation term and other factors cannot be overemphasized. Authors in [19] conducted a mathematical analysis of COVID-19 using a SIR model with a convex incidence rate. Research on ways of reducing the incidence rate of diseases is still progressing. The dynamical behaviors of a modified SIR model with nonlinear incidence and recovery rates were presented in [20]. Their findings showed that successful treatment of patients at home can lead to minimizing the prevalence of the disease. A home-treatment algorithm based on anti-inflammatory drugs to prevent hospitalization of patients with early COVID-19 was studied in [21]. A mathematical model of COVID-19 spread with self-isolation at home and hospitalized compartments were presented in [22]. The impact of media-induced fear on a mathematical model of COVID-19 with a non-linear incidence rate was presented in [23].

The influence of saturation terms on either the infected or susceptible populations has been studied in all of these articles. In this research, we examined the influence of the saturation term on both the susceptible and infected classes in an SEIR mathematical model to determine where there is higher importance. We considered two cases namely First Case where we have $\alpha_1 S$ and Second Case where we have $\alpha_2 I$ which changes the incidence rate to saturated incidence rate. I.e.

$$\frac{\beta SI}{1+\alpha_1 S} \text{ and } \frac{\beta SI}{1+\alpha_2 I}.$$

2. Model

2.1. Model Formulation

In this paper, a four-compartment model was adopted and modified by incorporating saturated incidence rate to study its effect on the model.

2.1.1. Existing Model

Existing model by Al-Sheikh [24]:

$$\begin{aligned}
\frac{dS}{dt} &= A - \beta SI - \mu S, \\
\frac{dE}{dt} &= \beta SI - (\mu + \varepsilon) E, \\
\frac{dI}{dt} &= \varepsilon E - (\mu + r + d) I - cI, \\
\frac{dR}{dt} &= rI - \mu R + cI.
\end{aligned} \tag{1}$$

2.1.2. Proposed Model

This involves the application of the saturated incidence rate to study the effects of the saturation term on the infected and susceptible classes. Proposed model following the schematic diagram in Figure 1.

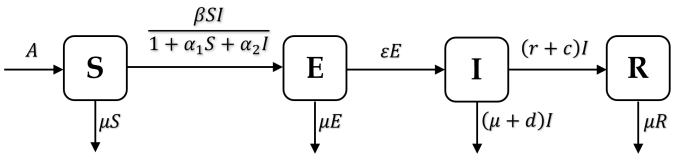

Figure 1. Schematic diagram of the proposed model

with the proposed model in equation (2):

$$\begin{aligned}
\frac{dS}{dt} &= A - \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I} - \mu S, \\
\frac{dE}{dt} &= \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I} - (\mu + \varepsilon) E, \\
\frac{dI}{dt} &= \varepsilon E - (\mu + r + d + c) I, \\
\frac{dR}{dt} &= (r + c) I - \mu R.
\end{aligned} \tag{2}$$

2.2. Model Description

The classes of the model are S, E, I, and R, represent the susceptible, exposed, infected and recovered classes respectively. We assume that the initial populations of each group in the model are $S(0) = s_0$, $E(0) = e_0$, $I(0) = i_0$, $R(0) = r_0$, where α_1 & α_2 represent the saturation terms. β represents the disease transmission coefficient, μ is the mortality rate, r is the recovery rate, ε is rate of losing immunity, d is disease induced death , c is the treatment rate and A is birth rate. A complete description of the parameters is given in Table 1. Since variable R does not appear in the first three equations, it is enough to analyze the following reduced system as shown in the equation (3).

Table 1. Table of parameters and their numerical values

Parameters	Description	Values	References
s_0	Initial susceptible population	25	Assumed
e_0	Initial exposed population	18	Assumed
i_0	Initial infected population	12	Assumed
r_0	Initial recovered population	7	Assumed
A	Birth rate	48	Assumed
c	Treatment rate	0.08	Assumed
β	Disease transmission coefficient	0.3	Assumed
ε	Rate of losing immunity	0.25	[25]
μ	Mortality rate	0.5	Assumed
d	Disease induced death	0.1	[25]
r	Treatment rate	0.15	[25]
α_1 , α_2	Saturation term	$0 \leq \alpha_n < 1 \quad n = 1, 2$	Calibrated

3. Results and Discussions

In this section, we present the analytical and numerical results including their biological interpretations. We first identify the existence and uniqueness of solution, investigate the biological equilibria, the basic reproduction number, stability and sensitivity analysis and ended by computing the numerical solutions to show the dynamical behaviors numerically. We present this analysis in 2 different model cases.

3.1. First Case

The model is inclusive of saturation term of the susceptible individual through a saturated incidence rate with a polynomial incorporation $\frac{1}{1+\alpha_1 S}$. Thus, as $\alpha_2 = 0$ from equation (2) we have;

$$\begin{aligned} \frac{dS}{dt} &= A - \frac{\beta SI}{1+\alpha_1 S} - \mu S, \\ \frac{dE}{dt} &= \frac{\beta SI}{1+\alpha_1 S} - (\mu+\varepsilon)\, E, \\ \frac{dI}{dt} &= \varepsilon E - (\mu + r + d + c)\, I, \end{aligned} \tag{3}$$

3.1.1. Existence and Uniqueness of Solution

Here, we apply a Lipchitz criterion to examine the existence and uniqueness of the model solution.

Thus, from equation (3), let:

$$B_1 = A - \frac{\beta SI}{1+\alpha_1 S} - \mu S; B_2 = \frac{\beta SI}{1+\alpha_1 S} - (\mu+\varepsilon)\, E; B_3 = \varepsilon E - (\mu + r + d + c)\, I \; ; B_4 = (r+c)\, I - \mu R.$$

The following partial derivatives are obtained:

For $B_1 = A - \frac{\beta SI}{1+\alpha_1 S} - \mu S$,

$$\left|\frac{\partial B_1}{\partial S}\right| = \left|-\frac{I\beta}{(1+\alpha_1 S)^2} - \mu\right| < \infty, \left|\frac{\partial B_1}{\partial E}\right| = |0| < \infty, \left|\frac{\partial B_1}{\partial I}\right| = \left|\frac{\beta S}{(1+\alpha_1 S)}\right| < \infty, \left|\frac{\partial B_1}{\partial R}\right| = |0| < \infty.$$

For $B_2 = \frac{\beta SI}{1+\alpha_1 S} - (\mu+\varepsilon)\, E$,

$$\left|\frac{\partial B_2}{\partial S}\right| = \left|\frac{\beta I}{(1+\alpha_1 S)^2}\right| < \infty, \left|\frac{\partial B_2}{\partial E}\right| = |(\mu+\varepsilon)| < \infty, \left|\frac{\partial B_2}{\partial I}\right| = \left|-\frac{\beta S}{(1+\alpha_1 S)^2}\right| < \infty, \left|\frac{\partial B_2}{\partial R}\right| = |0\,| < \infty.$$

For $B_3 = \varepsilon E - (\mu + r + d + c)\, I$,

$$\left|\frac{\partial B_3}{\partial S}\right| = |0| < \infty, \left|\frac{\partial B_3}{\partial E}\right| = |\varepsilon| < \infty, \left|\frac{\partial B_3}{\partial I}\right| = |-(\mu+r+d+c)| < \infty, \left|\frac{\partial B_3}{\partial R}\right| = |0| < \infty.$$

For $B_4 = (r+c)\, I - \mu R$,

$$\left|\frac{\partial B_4}{\partial S}\right| = |0| < \infty, \left|\frac{\partial B_4}{\partial E}\right| = |0| < \infty, \left|\frac{\partial B_4}{\partial I}\right| = |(r+c)| < \infty, \left|\frac{\partial B_4}{\partial R}\right| = |-\mu| < \infty.$$

The partial derivatives exist, continuous and are bounded, therefore the system of equation (3) exist and has a unique solution in $\Re^4$.

3.1.2. Biological Equilibria

1. **Disease-free equilibrium**
   At the disease free-equilibrium, $I = 0$. let:

$$\begin{aligned} A - \frac{\beta SI}{1+\alpha_1 S} - \mu S &= 0, \\ \frac{\beta SI}{1+\alpha_1 S} - (\mu+\varepsilon)\, E &= 0, \\ \varepsilon E - (\mu + r + d + c)\, I &= 0, \end{aligned} \tag{4}$$

   From equation (4),

$$\begin{aligned} A - \frac{\beta SI}{1+\alpha_1 S} - \mu S &= 0, \\ A - 0 &= \mu S, \\ S &= \frac{A}{\mu}, \end{aligned} \tag{5}$$

   Therefore, DFE;

$$(S^*, E^*, I^*) = \left[\frac{A}{\mu}, 0, 0\right]. \tag{6}$$

2. **Endemic Equilibrium**
   At the endemic equilibrium, $I \neq 0$. The endemic equilibrium points are:

$$(S, E, I) = (S^{**}, E^{**}, I^{**}), \tag{7}$$

Hence, solving

$$\begin{aligned}
S^{**} &= \frac{(\mu+\varepsilon)(\Lambda)}{\varepsilon\beta - \alpha_1(\mu+\varepsilon)(\Lambda)} \\
E^{**} &= \frac{(\Lambda)\left[A\varepsilon\beta - A\alpha_1(\mu+\varepsilon)(\Lambda) - \mu\varepsilon(\mu+\varepsilon)(\Lambda)\right]\left[(\mu+\varepsilon)(\Lambda)\right]}{\varepsilon^2\beta - \alpha_1\varepsilon(\mu+\varepsilon)(\Lambda)} \\
I^{**} &= \frac{A\varepsilon\beta(\mu+\varepsilon)(\Lambda) - A\alpha_1\left((\mu+\varepsilon)(\Lambda)\right)^2 - \mu\varepsilon\left((\mu+\varepsilon)(\Lambda)\right)^2}{\varepsilon\beta - \alpha_1(\mu+\varepsilon)(\Lambda)}
\end{aligned}$$

with

$$\Lambda = \mu + r + d + c.$$

3.1.3. The Basic Reproduction Number

There are two diseases state but only one way to create new infections. Hence, Exposed and Infected compartment of the model are concerned with from equation (3):

$$\begin{aligned}
\frac{dE}{dt} &= \frac{\beta SI}{1+\alpha_1 S} - (\mu+\varepsilon)E, \\
\frac{dI}{dt} &= \varepsilon E - (\mu+r+d+c)I.
\end{aligned} \tag{8}$$

We obtained the characteristics equation of matrix G, as: $|G - \lambda I| = 0$. Hence,

$$\begin{vmatrix} \frac{\beta A\varepsilon}{(\mu+\alpha_1 A)(\mu+\varepsilon)(\mu+r+d+c)} - \lambda & \frac{\beta A}{(\mu+\alpha_1 A)(\mu+r+d+c)} \\ 0 & 0-\lambda \end{vmatrix} = 0 \tag{9}$$

$$\left(\frac{\beta A\varepsilon}{(\mu+\alpha_1 A)(\mu+\varepsilon)(\mu+r+d+c)} - \lambda\right)(-\lambda) = 0$$

$$\lambda = \frac{\beta A\varepsilon}{(\mu+\alpha_1 A)(\mu+\varepsilon)(\mu+r+d+c)} \quad \vee \quad \lambda = 0.$$

Therefore, the dominant eigenvalue is the required basic reproduction number R_0. Hence,

$$R_0 = \frac{\beta A\varepsilon}{(\mu+\alpha_1 A)(\mu+\varepsilon)(\mu+r+d+c)}. \tag{10}$$

3.1.4. Effect of α_1 on the Basic Reproduction Number

To analyze the impact of α_1 on the basic reproduction number R_0, we evaluate equation (10) using the baseline parameters presented in Table 1, obtain:

$$R_0 = \frac{5.783132530}{0.5 + 48\alpha_1}.$$

Thus, as $0 \leq \alpha_1 < 1$, we have shown the effect of α_1 on R_0 in Table 2.

Table 2. Table showing the effect of α_1 on R_0

α_1	Effect on R_0
0.00	11.56626506
0.30	0.388129700
0.60	0.197376537
0.90	0.132337129

From Table 2, it could be observed that the basic reproduction number decreases drastically as α_1 progresses from 0 to 0.90. Hence, we can deduce that the disease will eventually dies out when α_1 is at peak because $R_0 < 1$.

3.1.5. Stability Analysis

1. **Local Stability of the Disease Free-Equilibrium**
   The system of equation (3) was linearized by setting

$$S - S_1 = x,\ E = E,\ I = I; \quad S = x + S_1.$$

From this setting, we obtained

$$\begin{aligned} \frac{dx}{dt} &= A - \beta I\left(x + S_1\right)\left(1 + \alpha_1 S\right)^{-1} - \mu\left(x + S_1\right), \\ \frac{dE}{dt} &= \beta I\left(x + S_1\right)\left(1 + \alpha_1 S\right)^{-1} - \left(\mu + \varepsilon\right) E, \\ \frac{dI}{dt} &= \varepsilon E - \left(\mu + r + d + c\right) I. \end{aligned} \tag{11}$$

The resulting Jacobian matrix is

$$\begin{pmatrix} \dot{x} \\ \dot{E} \\ \dot{I} \end{pmatrix} = \begin{pmatrix} -\mu & 0 & -\beta S_1 \\ 0 & -(\mu+\varepsilon) & 0 \\ 0 & \varepsilon & -(\mu+r+d+c) \end{pmatrix} \begin{pmatrix} x \\ E \\ I \end{pmatrix} + \text{Nonlinear Term}. \tag{12}$$

At DFE, $S = \frac{A}{\mu}$ therefore by substituting $S_1 = \frac{A}{\mu}$ yields

$$\begin{aligned} &(-\mu - \lambda)\left[\left(-\left(\mu+\varepsilon\right) - \lambda\right)\left(-\left(\mu+r+d+c\right) - \lambda\right) - 0\right] - \tfrac{\beta A}{\mu}\left[0\right] = 0 \\ &(-\mu - \lambda)\left(-\left(\mu+\varepsilon\right) - \lambda\right)\left(-\left(\mu+r+d+c\right) - \lambda\right) = 0 \end{aligned} \tag{13}$$

therefore,

$$\lambda = -\mu \ \vee\ \lambda = -\left(\mu+\varepsilon\right) \ \vee\ \lambda = -\left(\mu+r+d+c\right). \tag{14}$$

Since $R_0 < 1$, all the eigenvalue are all negatives, hence the disease free-equilibrium is locally asymptotically stable.

2. **Local Stability of the Endemic Equilibrium**
Let;

$$J=\begin{bmatrix} \frac{-\beta I^*-2\beta S^* I^*}{(1+\alpha_1 S^*)^2}-\mu & 0 & \frac{-\beta S^*}{1+\alpha_1 S^*} \\ \frac{\beta I^*}{(1+\alpha_1 S^*)^2} & -(\mu+\varepsilon) & 0 \\ 0 & \varepsilon & -(\mu+r+d+c) \end{bmatrix} \tag{15}$$

from equation (3). Analyzing the Jacobian matrix using the equilibrium points. The characteristics equation of J is $|J-\lambda I|=0$. The resulting characteristics polynomial is

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

where,
$a_0=1$
$a_1=2\mu+\varepsilon+r+d+c$
$a_2=\frac{\beta I^*+2\alpha_1\beta S^* I^*\mu}{(1+\alpha_1 S^*)^2}+\frac{\beta I^*+2\alpha_1\beta S^* I^*\varepsilon}{(1+\alpha_1 S^*)^2}+2\mu^2+2\mu\varepsilon+\mu r+\mu d+\mu c+\varepsilon r+\varepsilon d+\varepsilon c$
$a_3=\left(\frac{\beta I^*+2\alpha_1\beta S^* I^*}{(1+\alpha_1 S^*)^2}+\mu\right)(\mu+\varepsilon)(\mu+r+d+c)+\frac{\beta^2 S^* I^*\varepsilon}{(1+\alpha_1 S^*)^3}.$

Using the Routh-Hurwitz criterion, it can be seen that all the eigenvalues of the characteristics equation above have negative real part. Since $a_1a_2-a_3 > 0$, therefore the endemic equilibrium is locally asymptotically stable.

3. **Global Stability of Disease-Free Equilibrium**
Consider;

$$\begin{aligned} \frac{dE}{dt} &= \frac{\beta SI}{1+\alpha_1 S}-(\mu+\varepsilon)E, \\ \frac{dI}{dt} &= \varepsilon E-(\mu+r+d+c)I. \end{aligned} \tag{16}$$

where

$$R_o=\frac{\beta A\varepsilon}{(\mu+\alpha_1 A)(\mu+\varepsilon)(\mu+r+d+c)}.$$

Let $E=I_1$ and $I=I_2$. Constructing a Lyapunov function:
$V(t,S,E,I)=C_1I_1+C_2I_2$, where C_1, C_2 are constants,

$$
\begin{aligned}
\frac{dV}{dt} &= C_1 I_1^1 + C_2 I_2^1 \\
\frac{dV}{dt} &= C_1 \left(\frac{\beta S I_2}{1+\alpha_1 S} - (\mu+\varepsilon) I_1 \right) + C_2 \left(\varepsilon I_1 - (\mu+r+d+c) I_2 \right) \\
&\leq \left(C_2 \varepsilon - C_1 (\mu+\varepsilon) \right) I_1 + \left(C_2 \frac{\beta S I}{1+\alpha_1 S} - C_2 (\mu+r+d+c) \right) I_2 \\
&\leq \left(C_2 \varepsilon - C_1 (\mu+\varepsilon) \right) I_1 + \left(C_2 \frac{\beta A}{\mu+\alpha_1 A} - C_2 (\mu+r+d+c) \right) I_2 \\
&\leq C_2 \varepsilon I_1 - C_1 (\mu+\varepsilon) I_1 + C_2 \frac{\beta A}{\mu+\alpha_1 A} I_2 - C_2 (\mu+r+d+c) I_2 \\
&\text{Let } C_1 = \frac{1}{(\mu+\varepsilon)} \text{ and } C_2 = \frac{\beta A}{(\mu+\alpha_1 A)(\mu+r+d+c)} \\
&\leq \left(\frac{\beta A \varepsilon}{(\mu+r+d+c)(\mu+\varepsilon)(\mu+\alpha_1 A)} - 1 \right) I_1 \\
&+ \left(\frac{\beta A}{(\mu+\alpha_1 A)(\mu+\varepsilon)} - \frac{(\mu+r+d+c)}{(\mu+\alpha_1 A)(\mu+r+d+c)(\mu+\varepsilon)} \right) \\
V^1 &\leq (\mu+r+d+c)(\mu+\varepsilon) \left[R_o - 1 \right] I.
\end{aligned}
$$

It is imperative to note that $V^1 = 0$ only when $E = 0$ as $S_0 = \frac{A}{\mu}$ when $t \to \infty$. Based on LaSalle's Invariance principle. Hence, it is globally asymptotically stable whenever $R_0 < 1$.

4. **Global Stability of the Endemic Equilibrium**
For the global stability, Lyapunov function was constructed:

$$
\begin{aligned}
L(E, I) &= \varepsilon E + (\mu+\varepsilon) I \\
\dot{L}(E, I) &= \varepsilon \left[\frac{\beta S I}{1+\alpha_1 S} - \mu E - \varepsilon E \right] + (\mu+\varepsilon) \left[\varepsilon E - (\mu+r+d+c) I \right] \\
&= \frac{\varepsilon \beta S I}{1+\alpha_1 S} - (\mu+\varepsilon)(\mu+r+d+c) I
\end{aligned} \tag{17}
$$

Substituting $S = \frac{A}{\mu}$, we obtained:

$$
\begin{aligned}
\dot{L}(E, I) &= \frac{\varepsilon \beta A I}{\mu + A\alpha_1} - (\mu+\varepsilon)(\mu+r+d+c) I \\
&= (\mu+\varepsilon)(\mu+r+d+c) \left[\frac{R_0}{(\mu+A\alpha_1)} - 1 \right] I.
\end{aligned} \tag{18}
$$

now, if $I = 0$ and $R_0 < 1$ ensures that $\dot{L} = 0$. If $R_0 < 1$, the DFE is globally asymptotically stable and the disease may dies out.

3.1.6. Sensitivity Analysis

Sensitivity analysis is a financial model that determines how target variables are affected based on changes in other variables known as input variables. This model is also referred to as what if or simulation analysis. It is a way of predicting the outcome of a decision given a certain range of variables. In order to measure the impact of the model parameters, we utilized the sensitivity index analysis using normalized sensitivity index.

The sensitivity index with respect to the model parameter are obtained using

$$\chi_{R_0}^{\beta} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} \tag{19}$$

since

$$R_0 = \frac{\varepsilon \beta A}{(\mu+\varepsilon)(\mu + A\alpha_1)(\mu + r + d + c)}.$$

Therefore,

$$\begin{aligned}
\frac{\partial R_0}{\partial A} \times \frac{A}{R_0} &= \frac{\mu}{\mu + \alpha_1 A} \\
\frac{\partial R_0}{\partial \varepsilon} \times \frac{\varepsilon}{R_0} &= \frac{\mu}{\mu + \varepsilon} \\
\frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} &= \frac{\mu^2}{\varepsilon\,(\mu + \varepsilon)} \\
\frac{\partial R_0}{\partial \alpha_1} \times \frac{\alpha_1}{R_0} &= \frac{\mu \alpha_1}{(\mu + \varepsilon)\,\varepsilon} \\
\frac{\partial R_0}{\partial r} \times \frac{r}{R_0} &= \frac{r}{(\mu + r + d + c)} \\
\frac{\partial R}{\partial d} \times \frac{d}{R_0} &= \frac{d}{(\mu + r + d + c)} \\
\frac{\partial R_0}{\partial c} \times \frac{c}{R_0} &= \frac{c}{(\mu + r + d + c)} \\
\frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} &= 1.
\end{aligned}$$

By the parameter values in Table 1, we obtained the Sensitivity Index values for each parameter in Table 3.

We observed that the sensitivity index of α_1 in the mathematical model is highly significant. Thus it is a parameter which impact cannot be ignored in disease eradication.

Table 3. Sensitivity index of each parameter evaluated with their base values

Parameter	Sensitivity Index (R_0)
A	0.02262
ε	1.33333
β	1
r	-0.18722
d	-0.12048
c	-0.09639
α_1	1.20000

3.1.7. Numerical Simulation

This section shows several numerical simulation results for first case, which refers to the model equation (3). The results of the simulation are shown in Figure 2, Figure 3, and Figure 4.

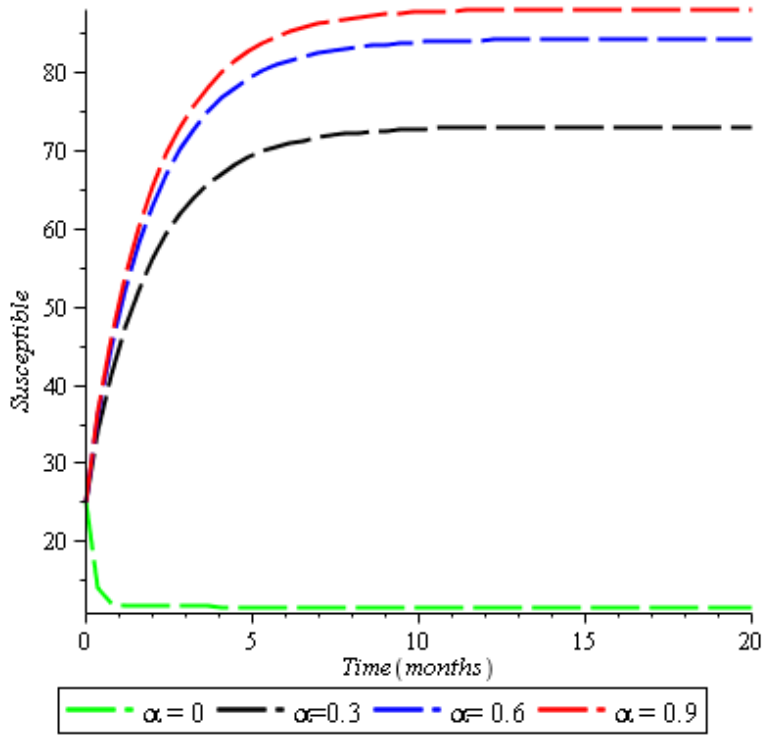

Figure 2. Effect of Saturation term on susceptible class with $A = 48$, $\mu = 0.5$, $\beta = 0.3$, $\varepsilon = 0.25$, $c = 0.08$, $d = 0.1$, $r = 0.15$

3.2. Second Case

The model involves saturation term for the infected individual to study its significant effect on the model. Hence the incident rate becomes $\frac{1}{1+\alpha_2 I}$ and as $\alpha_1 = 0$ from equation (2). Such that;

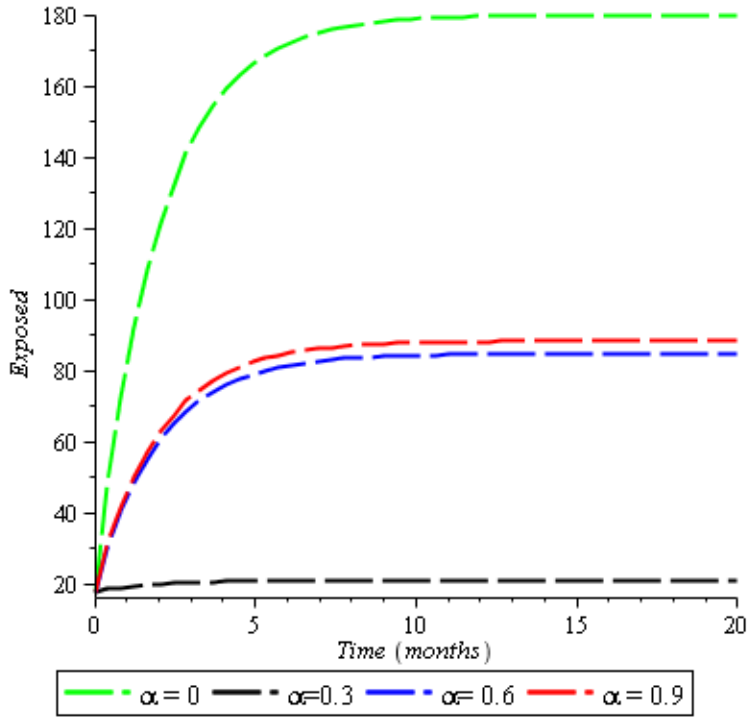

Figure 3. Effect of Saturation term on exposed class with $A = 48$, $\mu = 0.5$, $\beta = 0.3$, $\varepsilon = 0.25$, $c = 0.08$, $d = 0.1$, $r = 0.15$

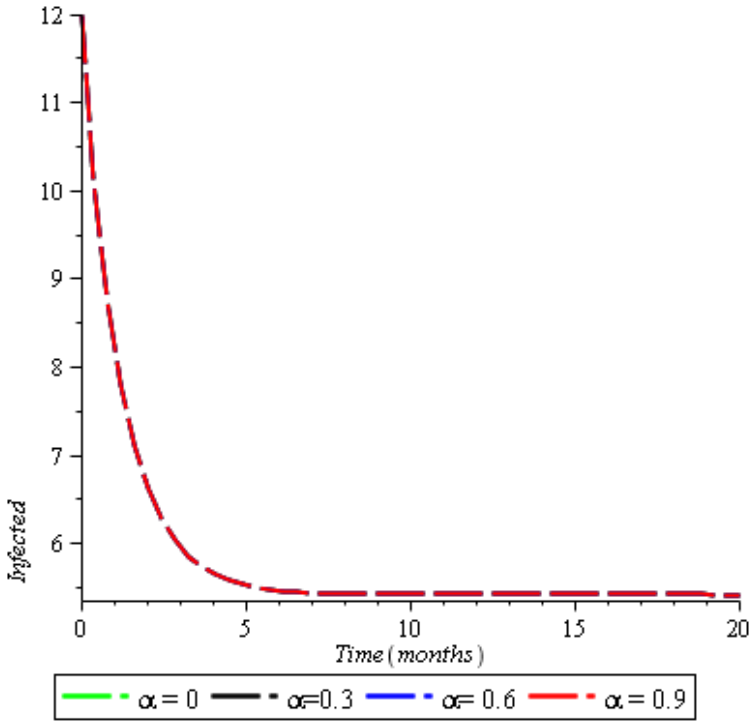

Figure 4. Effect of Saturation term on infected class with $A = 48$, $\mu = 0.5$, $\beta = 0.3$, $\varepsilon = 0.25$, $c = 0.08$, $d = 0.1$, $r = 0.15$

$$\begin{aligned}\frac{dS}{dt} &= A - \frac{\beta SI}{1+\alpha_2 I} - \mu S,\\ \frac{dE}{dt} &= \frac{\beta SI}{1+\alpha_2 I} - (\mu+\varepsilon)\,E,\\ \frac{dI}{dt} &= \varepsilon E - (\mu+r+d+c)\,I,\end{aligned} \tag{20}$$

3.2.1. Existence and Uniqueness of Solution

Similar to first case, we apply the Lipchitz criterion to verify the existence and uniqueness of the model. Hence, let:

Thus, from equation (20), let:

$$B_1 = A - \frac{\beta SI}{1+\alpha_2 S} - \mu I; B_2 = \frac{\beta SI}{1+\alpha_2 I} - (\mu+\varepsilon)\,E; B_3 = \varepsilon E - (\mu+r+d+c)\,I\ ; B_4 = (r+c)\,I - \mu R.$$

The following partial derivatives are obtained:

For $B_1 = A - \frac{\beta SI}{1+\alpha_2 I} - \mu S$,

$$\left|\frac{\partial B_1}{\partial S}\right| = \left|-\frac{I\beta}{(1+\alpha_2 I)^2} - \mu\right| < \infty, \left|\frac{\partial B_1}{\partial E}\right| = |0| < \infty, \left|\frac{\partial B_1}{\partial I}\right| = \left|\frac{\beta S}{(1+\alpha_2 I)}\right| < \infty, \left|\frac{\partial B_1}{\partial R}\right| = |0| < \infty.$$

For $B_2 = \frac{\beta SI}{1+\alpha_2 I} - (\mu+\varepsilon)\,E$,

$$\left|\frac{\partial B_2}{\partial S}\right| = \left|\frac{\beta I}{(1+\alpha_2 I)^2}\right| < \infty, \left|\frac{\partial B_2}{\partial E}\right| = |(\mu+\varepsilon)| < \infty, \left|\frac{\partial B_2}{\partial I}\right| = \left|-\frac{\beta S}{(1+\alpha_2 I)^2}\right| < \infty, \left|\frac{\partial B_2}{\partial R}\right| = |0\,| < \infty.$$

For $B_3 = \varepsilon E - (\mu+r+d+c)\,I$,

$$\left|\frac{\partial B_3}{\partial S}\right| = |0| < \infty, \left|\frac{\partial B_3}{\partial E}\right| = |\varepsilon| < \infty, \left|\frac{\partial B_3}{\partial I}\right| = |-(\mu+r+d+c)| < \infty, \left|\frac{\partial B_3}{\partial R}\right| = |0| < \infty.$$

For $B_4 = (r+c)\,I - \mu R$,

$$\left|\frac{\partial B_4}{\partial S}\right| = |0| < \infty, \left|\frac{\partial B_4}{\partial E}\right| = |0| < \infty, \left|\frac{\partial B_4}{\partial I}\right| = |(r+c)| < \infty, \left|\frac{\partial B_4}{\partial R}\right| = |-\mu| < \infty.$$

The partial derivatives exist, continuous and are bounded, therefore the system of equation (20) exist and has a unique solution in $\Re^4$.

3.2.2. Biological Equilibria

1. **Disease-free equilibrium**
   At the disease free-equilibrium (DFE), $I = 0$. If we let:

$$\begin{aligned} A - \frac{\beta SI}{1+\alpha_2 I} - \mu S &= 0, \\ A - 0 &= \mu S, \\ S &= \frac{A}{\mu}, \end{aligned} \tag{21}$$

Therefore, from equation (21), we obtained DFE

$$(S^*, E^*, I^*) = \left[\frac{A}{\mu}, 0, 0\right]. \tag{22}$$

2. **Endemic Equilibrium**
Similarly, we compute the endemic equilibria for case 2:

$$(S, E, I) = (S^{**}, E^{**}, I^{**}), \tag{23}$$

Hence, solving

$$\begin{aligned} S^{**} &= \frac{A}{\mu} - \frac{A\varepsilon\beta - \mu(\mu+\varepsilon)(\mu+r+d+c)}{\mu\varepsilon(\beta+\mu\alpha_2)} \\ E^{**} &= \frac{(\mu+r+d+c)(A\varepsilon\beta-\mu)}{\varepsilon(\beta+\mu\alpha_2)} \\ I^{**} &= \frac{A\varepsilon\beta - \mu(\mu+\varepsilon)(\mu+r+d+c)}{(\mu+\varepsilon)(\mu+r+d+c)(\beta+\mu\alpha_2)}. \end{aligned}$$

3.2.3. The Basic Reproduction Number

Here, we equally compute the basic reproduction number for case 2. Such that:

$$\begin{aligned} \frac{dE}{dt} &= \frac{\beta SI}{1+\alpha_2 I} - (\mu+\varepsilon)E, \\ \frac{dI}{dt} &= \varepsilon E - (\mu+r+d+c)I. \end{aligned} \tag{24}$$

We obtained the characteristics equation of matrix G, as: $|G - \lambda I| = 0$. Hence,

$$\begin{vmatrix} \frac{\beta A\varepsilon}{\mu(\mu+\varepsilon)(\mu+r+d+c)} - \lambda & \frac{\beta A}{\mu(\mu+r+d+c)} \\ 0 & 0-\lambda \end{vmatrix} = 0 \tag{25}$$

$$\left(\frac{\beta A\varepsilon}{\mu(\mu+\varepsilon)(\mu+r+d+c)} - \lambda\right)(-\lambda) = 0$$

$$\lambda = \frac{\beta A\varepsilon}{\mu(\mu+\varepsilon)(\mu+r+d+c)} \quad \vee \quad \lambda = 0.$$

Therefore, the dominant eigenvalue is the required basic reproduction number R_0. Hence,

$$R_0 = \frac{\beta A \varepsilon}{\mu\left(\mu+\varepsilon\right)\left(\mu+r+d+c\right)}. \tag{26}$$

3.2.4. Effect of α_2 on the Basic Reproduction Number

As observed in (26) the basic reproduction number does not contain parameter α_2. This shows that the saturation term on infected individuals have no significant effect in weighing down the basic reproduction number.

3.2.5. Stability Analysis

1. **Local Stability of the Disease Free-Equilibrium**
   The system of equation (20) was linearized by setting

$$S - S_1 = x,\ E = E,\ I = I; \quad S = x + S_1.$$

From this setting, we obtained

$$\begin{aligned} \frac{dx}{dt} &= A - \beta I\left(x+S_1\right)\left(1+\alpha_2 I\right)^{-1} - \mu\left(x+S_1\right), \\ \frac{dE}{dt} &= \beta I\left(x+S_1\right)\left(1+\alpha_2 I\right)^{-1} - \left(\mu+\varepsilon\right)E, \\ \frac{dI}{dt} &= \varepsilon E - \left(\mu+r+d+c\right)I. \end{aligned} \tag{27}$$

The resulting Jacobian matrix is

$$\begin{pmatrix} \dot{x} \\ \dot{E} \\ \dot{I} \end{pmatrix} = \begin{pmatrix} -\mu & 0 & -\beta S_1 \\ 0 & -\left(\mu+\varepsilon\right) & 0 \\ 0 & \varepsilon & -\left(\mu+r+d+c\right) \end{pmatrix} \begin{pmatrix} x \\ E \\ I \end{pmatrix} + \text{Nonlinear Term}. \tag{28}$$

At DFE, $S = \frac{A}{\mu}$ therefore by substituting $S_1 = \frac{A}{\mu}$ yields

$$\begin{aligned} &\left(-\mu-\lambda\right)\left[\left(-\left(\mu+\varepsilon\right)-\lambda\right)\left(-\left(\mu+r+d+c\right)-\lambda\right)-0\right] - \tfrac{\beta A}{\mu}\left[0\right] = 0 \\ &\left(-\mu-\lambda\right)\left(-\left(\mu+\varepsilon\right)-\lambda\right)\left(-\left(\mu+r+d+c\right)-\lambda\right) = 0 \end{aligned} \tag{29}$$

therefore,

$$\lambda = -\mu \ \vee\ \lambda = -\left(\mu+\varepsilon\right) \ \vee\ \lambda = -\left(\mu+r+d+c\right). \tag{30}$$

Since all the eigen value are negative, therefore $R_0 < 1$, and the disease free-equilibrium is locally asymptotically stable.

2. **Local Stability of the Endemic Equilibrium**
Let;

$$J=\begin{bmatrix} \frac{-\beta S^*}{1+\alpha_2 I^*}-\mu & 0 & \frac{-\beta S^*(1+\alpha_2 I^*)+\beta S^* I^* \alpha_2}{(1+\alpha_2 I^*)^2} \\ \frac{\beta S^*}{1+\alpha_2 I^*} & -(\mu+\varepsilon) & 0 \\ 0 & \varepsilon & -(\mu+r+d+c) \end{bmatrix} \tag{31}$$

To obtain the local stability of the endemic equilibrium, we set$|J-\lambda I|=0$ such that;

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

where,
$a_0=1$
$a_1=2\mu+\varepsilon+r+d+c$
$a_2=a_2=\frac{\beta I^*\mu}{1+\alpha_2 I^*}+\frac{\beta I^*\varepsilon}{1+\alpha_2 I^*}+2\mu^2+2\mu\varepsilon+\mu r+\mu d+\mu c+\varepsilon r+\varepsilon d+\varepsilon c$
$a_3=\left(\frac{\beta I^*}{1+\alpha_2 I^*}+\mu\right)(\mu+\varepsilon)(\mu+r+d+c)+\frac{\beta^2 S^* I^* \varepsilon}{(1+\alpha_2 I^*)^3}.$

Using the Routh-Hurwitz criterion, it can be seen that all the eigenvalues of the characteristics equation above have negative real part. Since $a_1a_2-a_3>0$, then the endemic equilibrium is locally asymptotically stable.

3. **Global stability of Disease-Free equilibrium**
To analyze the global stability of the model, we construct a Lyapunov function $L(E,I)=\varepsilon E+(\mu+\varepsilon)I$:

$$\begin{aligned} \dot{L}(E,I) &= \varepsilon\left[\frac{\beta SI}{1+\alpha_2 I}-\mu E-\varepsilon E\right]+(\mu+\varepsilon)\left[\varepsilon E-(\mu+r+d+c)I\right] \\ &= \frac{\varepsilon\beta SI}{1+\alpha_2 I}-(\mu+\varepsilon)(\mu+r+d+c)I \end{aligned} \tag{32}$$

Substituting $S=\frac{A}{\mu}$, we obtained

$$\begin{aligned} \dot{L}(E,I) &= \frac{\varepsilon\beta AI}{\mu(1+\alpha_2 I)}-(\mu+\varepsilon)(\mu+r+d+c)I \\ &= (\mu+\varepsilon)(\mu+r+d+c)\left[\frac{R_0}{(1+\alpha_2 I))}-1\right]I \end{aligned} \tag{33}$$

Now, if $I=0$ and $R_0<1$, this ensures that $\dot{L}=0$. If $R_0<1$, the DFE is globally asymptotically stable and the existence of disease will be wiped.

4. **Local stability of the Endemic Equilibrium**
Consider the following system of equations;

$$\begin{aligned}\frac{dS}{dt} &= A - \frac{\beta SI}{1+\alpha_2 S} - \mu S,\\ \frac{dE}{dt} &= \frac{\beta SI}{1+\alpha_2 S} - (\mu+\varepsilon)E,\\ \frac{dI}{dt} &= \varepsilon E - (\mu+r+d+c)I.\end{aligned} \tag{34}$$

Linearizing by applying the substitution $S = x + S^*$, $E = y + E^*$ and $I = z + I^*$ into above equations we have;

$$\begin{aligned}\frac{dx}{dt} &= A - \beta(x+S^*)(z+I^*) - [1+\alpha_2(z+I^*)]^{-1} - \mu(x+S^*),\\ \frac{dE}{dt} &= \beta(x+S^*)(z+I^*) - [1+\alpha_2(z+I^*)]^{-1} - (\mu+\varepsilon)(y+E^*),\\ \frac{dI}{dt} &= \varepsilon(y+E^*) - (\mu+r+d+c)(z+I^*).\end{aligned} \tag{35}$$

So,

$$\begin{aligned}\frac{dx}{dt} &= -\beta xz(1+\alpha_2 z)^{-1} - \mu x + \text{higherorder} + \text{non} - \text{linear terms},\\ \frac{dE}{dt} &= -\beta xz(1+\alpha_2 z)^{-1} - y(\mu+\varepsilon) + \text{higheroder} + \text{non} - \text{linear terms},\\ \frac{dI}{dt} &= y\varepsilon - z(\mu+r+d+c) + \text{higherorder} + \text{non} - \text{linear terms}.\end{aligned} \tag{36}$$

The Jacobian Matrix and Characteristic equations are resolved for, $|J_{E^*} - \lambda I| = 0$,

$$J_{E^*} = \begin{vmatrix} -\frac{\beta z}{1+\alpha_2 z} - \mu & 0 & \frac{\beta x}{1+\alpha_2} \\ \frac{\beta z}{1+\alpha_2 z} & -(\mu+\varepsilon) & \frac{\beta x}{1+\alpha_2} \\ 0 & \varepsilon & -(\mu+r+d+c) \end{vmatrix}$$

Where $|J_{E^*} - \lambda I| = 0$

$$\begin{vmatrix} -\left(\frac{\beta z}{1+\alpha_2 z} + \mu\right) - \lambda & 0 & \frac{\beta x}{1+\alpha_2} \\ \frac{\beta z}{1+\alpha_2 z} & -(\mu+\varepsilon) - \lambda & \frac{\beta x}{1+\alpha_2} \\ 0 & \varepsilon & -(\mu+r+d+c) - \lambda \end{vmatrix} = 0$$

The eigen values becomes;

$$\left[-\left(\frac{\beta z}{1+\alpha_2 z} + \mu\right) - \lambda\right][-(\mu+\varepsilon) - \lambda][-(\mu+r+d+c) - \lambda] = 0$$

Let,

$$A = -\left(\frac{\beta z}{1+\alpha_2 z} + \mu\right),\ B = -(\mu+\varepsilon), \text{ and } C = -(\mu+r+d+c)$$

then,

$$(A-\lambda)(B-\lambda)(C-\lambda)=0$$
$$\lambda^3-(A+B+C)\lambda^2+\left[(AB+C(A+B)\right]\lambda-ABC=0$$

Assume

$$a_1=(A+B+C),\ a_2=\left[(AB+C(A+B)\right],\ a_3=ABC$$

.

The characteristic equation becomes

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

The eigen values of the characteristic equation have negative real parts, hence the endemic equilibrium is Locally Asymptotically Stable.

5. Global Stability of the Endemic Equilibrium

Consider;

$$\begin{aligned}\frac{dE}{dt}&=\frac{\beta SI}{1+\alpha_2 I}-(\mu+\varepsilon)E,\\ \frac{dI}{dt}&=\varepsilon E-(\mu+r+d+c)I.\end{aligned}\tag{37}$$

where

$$R_0=\frac{\beta A\varepsilon}{\mu\left(\mu+\varepsilon\right)\left(\mu+r+d+c\right)}.$$

Let $E=I_1$ and $I=I_2$. Using Lyapunov algorithm:
$V(t,S,E,I)=C_1I_1+C_2I_2$, where C_1,C_2 are constant terms,

$$\begin{aligned}\frac{dV}{dt}&=C_1I_1^1+C_2I_2^1\\ \frac{dV}{dt}&=C_1\left(\frac{\beta SI_2}{1+\alpha_2I_2}-(\mu+\varepsilon)I_1\right)+C_2\left(\varepsilon I_1-(\mu+r+d+c)I_2\right)\\ &\leq\left(C_2\varepsilon-C_1(\mu+\varepsilon)\right)I_1+\left(C_2\frac{\beta SI}{(1+\alpha_2)}-C_2(\mu+r+d+c)\right)I_2\\ &\leq\left(C_2\varepsilon-C_1(\mu+\varepsilon)\right)I_1+\left(C_2\frac{\beta A}{\mu(1+\alpha_2)}-C_2(\mu+r+d+c)\right)I_2\\ &\leq C_2\varepsilon I_1-C_1(\mu+\varepsilon)I_1+C_2\frac{\beta A}{\mu(1+\alpha_2)}I_2-C_2(\mu+r+d+c)I_2\\ &\text{Let}\,C_1=\frac{1}{(\mu+\varepsilon)}\,\text{and}\,C_2=\frac{\beta A}{\mu(1+\alpha_2)(\mu+r+d+c)}\\ &\leq\left(\frac{\beta A\varepsilon}{(\mu+r+d+c)(\mu+\varepsilon)(1+\alpha_2)}-1\right)I_1\\ &+\left(\frac{\beta A}{\mu(1+\alpha_2)(\mu+\varepsilon)}-\frac{(\mu+r+d+c)}{\mu(1+\alpha_2)(\mu+r+d+c)(\mu+\varepsilon)}\right)\\ V^1&\leq\mu(\mu+r+d+c)(1+\alpha_2)\left[R_o-1\right]I.\end{aligned}$$

Then $V^1 = 0$ only when $E = 0$ in the model of equation (20) (second case) such that $S_o = \frac{A}{\mu}$ at $t \to \infty$. Based on LaSalle's Invariance principle. Hence, it is globally asymptotically stable whenever $R_o < 1$.

3.2.6. Sensitivity Analysis

Similar to first case, we compute the sensitivity index for second case. Such that,

$$\begin{aligned}
\frac{\partial R_0}{\partial A} \times \frac{A}{R_0} &= 1 \\
\frac{\partial R_0}{\partial \varepsilon} \times \frac{\varepsilon}{R_0} &= \frac{\mu}{\mu+\varepsilon} \\
\frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} &= -1 - \frac{\mu}{(\mu+\varepsilon)} - \frac{\mu}{(\mu+r+d+c)} \\
\frac{\partial R_0}{\partial \alpha_2} \times \frac{\alpha_2}{R_0} &= 0 \\
\frac{\partial R_0}{\partial r} \times \frac{r}{R_0} &= -\frac{r}{(\mu+r+d+c)} \\
\frac{\partial R}{\partial d} \times \frac{d}{R_0} &= \frac{d}{(\mu+r+d+c)} \\
\frac{\partial R_0}{\partial c} \times \frac{c}{R_0} &= \frac{c}{(\mu+r+d+c)} \\
\frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} &= 1.
\end{aligned}$$

By the parameter values in Table 1, we obtained the Sensitivity Index values for each parameter in Table 4.

Table 4. Sensitivity index of each parameter

Parameter	Sensitivity Index (R_0)
A	1
ε	0.99999
μ	0.99999
β	0.59999
r	-0.05556
d	-0.11111
c	-0.55555
α_2	0

In this case the sensitivity of α_2 in the model is 0. Thus in this case, its effect is negligible in eradicating the disease.

3.2.7. Numerical Simulation

This section shows several numerical simulation results for second case, which refers to the model equation (20). The results of the simulation are shown in Figure 5, Figure 6, and Figure 7.

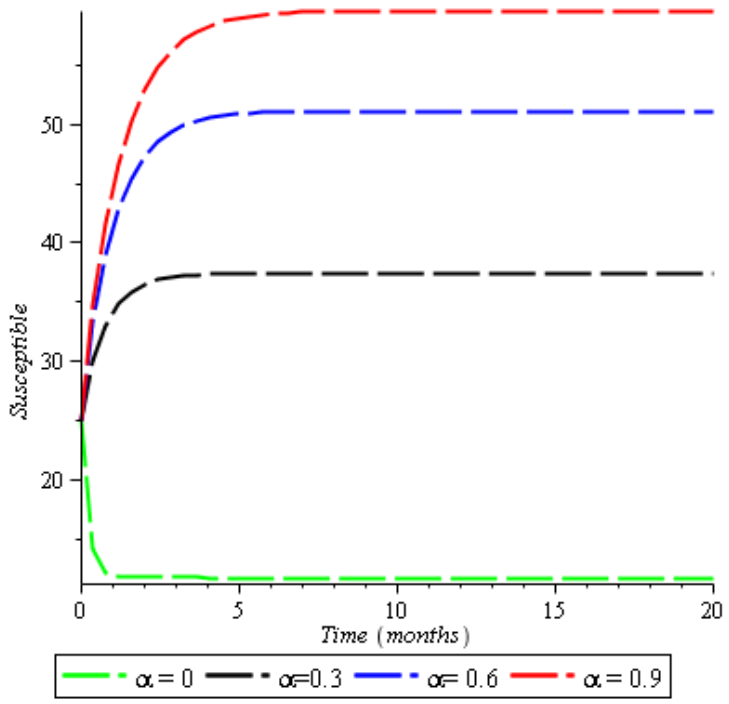

Figure 5. Effect of Saturation term on susceptible class with $A = 48$, $\mu = 0.5$, $\beta = 0.3$, $\varepsilon = 0.25$, $c = 0.08$, $d = 0.1$, $r = 0.15$

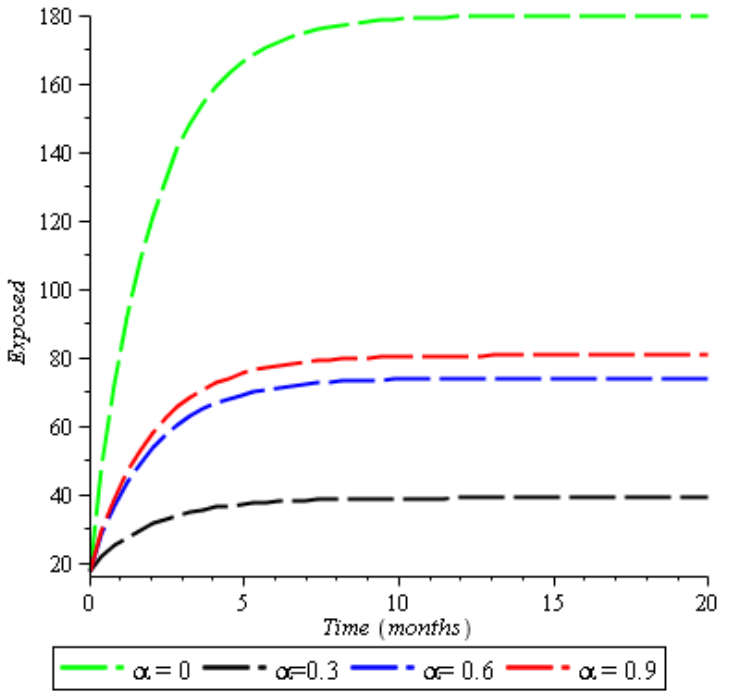

Figure 6. Effect of Saturation term on exposed class with $A = 48$, $\mu = 0.5$, $\beta = 0.3$, $\varepsilon = 0.25$, $c = 0.08$, $d = 0.1$, $r = 0.15$

3.3. Discussion

The simulation result presented in Figure 2-7 reveals the effect of saturation term on the present population of the model compartments in first and second case respectively. In

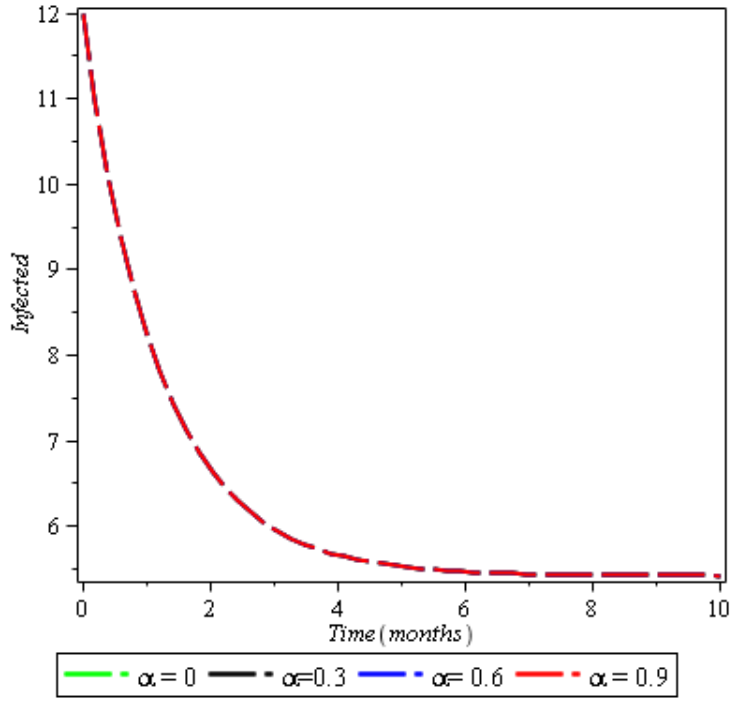

Figure 7. Effect of Saturation term on infected class with $A = 48$, $\mu = 0.5$, $\beta = 0.3$, $\varepsilon = 0.25$, $c = 0.08$, $d = 0.1$, $r = 0.15$

Figure 2, the role of saturation term as examined on the susceptible population revealed that the class population drastically increases to maximum as the saturation term increases from to . This shows that increasing the awareness level of people to factors capable of getting rid of diseases will stop them from moving from the susceptible class to the exposed or infected class. Figure 3 shows that saturation parameter also has effect on the exposed class. We observed that the exposed population was at its peak when the saturation level was 0. This interprets that if there is no strategy to curtail the spread of diseases, several people will get exposed. Figure 4 indicates that saturation term have little or no effect on the infected class i.e. the main factors associated to saturation term such as media induced fear, enlightenment and general knowledge of people against diseases have better role on the susceptible population than the already infected group. Figure 5 to 7 similarly revealed that saturation term is a parameter which cannot be ignored in disease eradication.

4. Conclusion

Saturation term play a vital role on susceptible and infected individuals. In our results, saturation term is more active on susceptible than infected individuals. Therefore, more sensitization program through seminar and media will be more useful to susceptible class than infected class during the eradication of diseases.

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