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RESEARCH ARTICLE

Mathematical Analysis of the Role of Detection Rate on Dynamical Spread of Ebola Virus Disease

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ABSTRACT

In this paper, a non-linear mathematical model of the Ebola virus disease with detection rate is proposed and analyzed. The whole population under consideration is divided into five compartments e.g. susceptible, latently infected, infected undetected, infected detected, and recovered to study the transmission dynamics of the Ebola virus disease. Based on the immunity level, susceptible individuals move to exposed class or directly to infected detected class once they come into contact with an infective. This has been incorporated through the progression rate which is slow. The equilibria of the model and the basic reproduction number R_0 are computed. It is observed that the disease-free equilibrium of the model is locally asymptotically stable when $R_0<1$. The model exhibits forward bifurcation under certain restrictions on parameters, which indicate that the model has a single endemic equilibrium for $R_0<1$. This suggests that an accurate estimation of parameters and the level of control measures are required to reduce the infection prevalence of the Ebola virus in the endemic region and just $R_0<1$ is enough to eliminate the disease from the population. R_0 needs to be lowered much below one to confirm the global stability of the disease-free equilibrium. Numerical simulation is performed to demonstrate the analytical results. It is found that the increase in the rate of detection rate leads to a decrease in the threshold value of R_0. Numerical simulations have been carried out to support the analytic results.

Keywords: Nonlinear system, Reproduction number, Sensitivity and Bifurcation analysis.

1 Introduction

The disease Ebola virus epidemic first broke out in 1976 in two countries; Nzaire (a town in South Sudan) and Yamuku in Democratic Republic of Congo at a village near the Ebola river [9,11] though the Filoviruses was first discovered in 1969.7 Initially, the virus was thought to be native to East Africa [1].In recent time, the outbreaks of it have shown epicenters outside this region and may be attributed to the migratory effect of the bats [4]. Other than the fact that the bats potentially migrate in epidemic epicenters; there are frequent epidemic outbreaks in countries undergoing civil strife or emerging from clash states in East and Western Africa. These are also countries with poor socioeconomic status, suggesting ill-prepared public health responses to the epidemics. Internationally, however, travelling including ambulance of infected people has been the mode of spread to Europe and the USA [7]. The risk to EVD in children is attributed to contact with their sick parent(s) which can either be vertically spread through breast-feeding has also been described or horizontally transmission, through caretakers and relatives. It was reported by [12] that the pediatric EVD data from various recent outbreaks [8], these data suggests that the proportion of children infected has varied in different settings and overtime. For instance, the proportions of children involved was 27/315(9%) in the Zaire EVD in 1995, 90/218 (41%) in Gulu, Uganda (Sudan EVD) in 2000–2001and 147/823 (18%) in the current outbreak in the 4 most affected West African countries as of August 2014.9 Furthermore, data from Gulu in Uganda precisely, indicated that female children had a higher risk of

developing EVD compared with their male counterparts [24]. In Gulu, Uganda it was observed that children were in isolation rooms and in contact with their EVD-infected parents [24] but, the pediatric EVD's sparing nature is attributed to low-risk exposures [25].

The outbreak of Ebola Virus Disease in West Africa happens to be the most severe in recorded history [9]; hence, there is a need to explore the dynamics of this disease through mathematical modeling, in order to control further outbreak of the disease in World [3]. Several researchers have developed some mathematical models to better improve our understanding of the dynamics and spread of Ebola Virus Disease in order to curb its prevalence and curtain the incessant outbreaks of the virus [3,7]. This research was formulated and analyzed a mathematical model that studied the effect of each parameter and impart of detection rate on the dynamical spread of Ebola virus diseases in the population.

2 Mathematical Model

The study uses five (5) compartmental deterministic mathematical model of the S, L, I_u, I_d, R to have better understanding of the dynamical spread of Ebola virus diseases in the population. The population size $N(t)$ is sub–divided into sub–classes of individuals who are Susceptible $S(t)$, Latent L(t), Infected undetected $I_u(t)$, Infected detected $I_d(t)$, and Recovered $R(t)$,

Where

$$N(t) = S(t) + L(t) + I_u(t) + I_d(t) + R(t) \quad (1)$$

Susceptible individual is a member of a population who is at risk of becoming infected by a disease, Ebola virus diseases. The population of susceptible individuals increases by the recruitment of active individuals at the rateπ. The population decreased by natural death at a rate μ also, by force of infection of infected detected individualsλ. Also, Latent individual is a member of a population who is infected individual but not infectious of the disease Ebola virus. The population of latent individuals increases through the product of slow progression and infection of susceptible and are assumed to show no disease symptoms at this time. The population of latent class diminished by the progression rate of infected individual to infectious class I_d, disease induced death and natural death at a rateμ. In addition, Infected detected individual is a member of a population who is infected and capable of transmitting the disease, Ebola virus in the population. The population of infected detected individuals increases through the infection of susceptible, detection rate of infected individual and the progression rate of infected individual to infectious class $I_d$from latent. The population is decreased by recovery rate of infectious, natural death, disease induced death and endogenous reactivation with progression rate (τ_2), (μ), (δ) and ($\alpha\tau_1$) respectively. They are those under treatment or isolation center. Furthermore, Infected undetected individual is a member of a population who is infected and capable of transmitting the disease, EVB. The population of infected undetected individuals increases through the endogenous reactivation with progression rate. The population is decreased by recovery rate of infected, natural death, disease induced death and detection rate (τ_3), (μ), (δ) and (r) respectively. Finally, Recovered individual is a member of a population who recovered from the disease. The population of recovered individual is increased by the treatment of infectious individual at a rate (τ_2) and treatment of infected individual at a rate (τ_3), this population later decreased by natural death at the rate (μ).

The diagrammatic representation of the Ebola virus disease model dynamics can be seen in Figure 1 and the corresponding model is governed by the following system of nonlinear ordinary differential equations (1). Also, the associated variables and parameters of the model are tabulated in Table 1 and Table 2 respectively.

2.1 Model Equation

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \lambda S(t) - \mu S(t) \\
\frac{dL}{dt} &= \varepsilon\lambda S(t) - (\tau_1 + \delta_L + \mu)L(t) \\
\frac{dI_d}{dt} &= (1-\varepsilon)\lambda S(t) - (\tau_2 + \delta_{I_d} + \mu)I_d(t) + rI_u(t) + (1-\alpha)\tau_1 L(t) \\
\frac{dI_u}{dt} &= \alpha\tau_1 L(t) - (r + \tau_3 + \delta_{I_U} + \mu)I_u(t) \\
\frac{dR}{dt} &= \tau_2 I_d(t) + \tau_3 I_u(t) - \mu R(t)
\end{aligned}\right\} \quad (2)$$

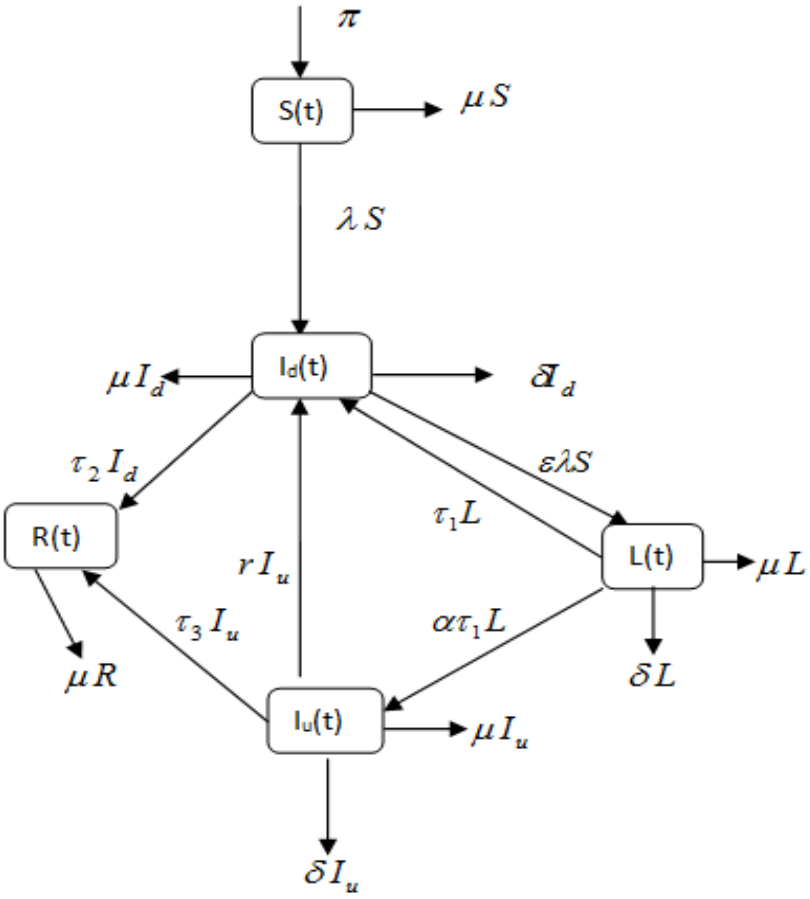

Figure 1: Schematic diagram for Ebola virus disease dynamics.

Table 1. Description of Variables

Variables	Definitions
S	Susceptible individuals
L	Latently infected individual
I_u	Infected individual undetected
I_d	Infected individual detected
R	Recovered individual

Table 2. Description of parameters

Parameters	Definitions
τ_1	Progression rate of infected individual to infectious individual
τ_2	Recovery rate of infected detected individual due to treatment
τ_3	Recovery rate of infected undetected individual due to treatment
r	Detection rate of infected undetected individual
π	Recruitment rate
μ	Natural death rate
α	Endogenous reactivation rate
θ	Modification parameter
δ	Induced mortality rate
β	Effective contact rate
N	Total population
λ	Force of infection
ε	Slow progressor

3 Basic Property

3.1 Positivity and Boundedness of Solutions

Since model (2) monitors human population, all the parameters are non-negative. Therefore, it is needful to show that all the state variables are also non-negative for all time t > 0.

3.1.1 Theorem 1

The state variables, S(t); L(t); $I_U(t)$; $I_d(t)$; and R(t), of the autonomous version of the Ebola Virus disease of model (2), with the non-negative initial data, remain non-negative for all t > 0.

3.1.2 Proof

Recalling the equation in system (2)

$$\frac{dS}{dt} = \pi - \lambda S(t) - \mu S(t)$$
$$\frac{dL}{dt} = \varepsilon\lambda S(t) - (\tau_1 + \delta_L + \mu)L(t)$$
$$\frac{dI_d}{dt} = (1-\varepsilon)\lambda S(t) - (\tau_2 + \delta_{I_d} + \mu)I_d(t) + rI_u(t) + (1-\alpha)\tau_1 L(t)$$
$$\frac{dI_u}{dt} = \alpha\tau_1 L(t) - (r + \tau_3 + \delta_{I_U} + \mu)I_u(t)$$
$$\frac{dR}{dt} = \tau_2 I_d(t) + \tau_3 I_u(t) - \mu R(t) \quad (3)$$

where

$$\lambda = \frac{\beta(\theta I_d + I_u)}{N} \quad (4)$$

One can see from the first equation of (3) that

$$\frac{dS}{dt} \geq -(\lambda + \mu)S(t) \quad (5)$$

So that,

$$\frac{d}{dt}(S(t)\, exp(\mu t + \int_0^t \lambda(\varpi)d\varpi)) \geq 0 \quad (6)$$

From which follows that

$$S(t) \geq S(0)\, exp(-(\mu t + \int_0^t \lambda(\varpi)d\varpi)) > 0 \quad (7)$$

It can be shown, using similar approach, that other state variables, L(t); $I_u(t)$; $I_d(t)$; and R(t), are non-negative for all t > 0.

Next, consider the biologically feasible region, define by $\Gamma \subset R_+^5$

Where:

$$\Gamma = \left\{(S, L, I_u, I_d, R) \in R_+^5 : N \leq \frac{\pi}{\mu}\right\} \tag{8}$$

It can be shown that Γ is positively invariant region.

3.1.3 Theorem 2

The region $\Gamma$is positively invariant with respect to the model (2)

3.1.4 Proof:

The rate of change of the total population is given by

$$\frac{dN}{dt} = \pi - (S + L + Iu + Id + R)\mu - (L + Iu + Id)\delta \tag{9}$$

It results into the solution;

$$N(t) = N(0)\, exp(-\mu t) + \frac{\pi}{\mu}(1 - exp(-\mu t)) \tag{10}$$

It follows that

$$N(t) \to \frac{\pi}{\mu} \text{ as } \quad t \to \infty$$

In particular,

$$N(t) \leq \frac{\pi}{\mu} \tag{11}$$

If

$$N(0) \leq \frac{\pi}{\mu} \tag{12}$$

with respect to the Ebola Virus model (3). Hence, it suffices to consider the dynamics of the model in Γ. In this region, the Ebola Virus model can be considered as being mathematically well-posed [14].

4 Stability Property

4.1 Disease Free Equilibrium (DFE)

Disease free means when there is disease in the population, i.e, $I_u = I_d = 0$. At equilibrium points, all compartments are set to be zero;

$$\frac{dS}{dt} = \frac{dL}{dt} = \frac{dI_u}{dt} = \frac{dI_d}{dt} = \frac{dR}{dt} = 0 \tag{13}$$

Let E_0 denotes the disease-free equilibrium. Thus; the model in (2) has disease free equilibrium given by

$$E_0 = (S, L, I_u, I_d, R) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right) \tag{14}$$

4.2 Endemic Equilibrium

The endemic equilibrium of the model (2) is given below;

$$\left.\begin{aligned} S^* &= \frac{\pi}{\mu R_0} \\ L^* &= \frac{\varepsilon\pi(R_0 - 1)}{d_1 R_0} \\ I_u{}^* &= \frac{\alpha\tau_1 \rightleftarrows \varepsilon\pi(R_0 - 1)}{K_1 K_3 R_0} \\ I_d{}^* &= \frac{(K_1 K_3(1-\varepsilon) + K_3\tau_1\varepsilon(1-\alpha) + r\alpha\tau_1\varepsilon)\pi(R_0 - 1)}{K_1 K_2 K_3 R_0} \\ R^* &= \frac{(\tau_2(K_1 K_3(1-\varepsilon) + K_3\tau_1\varepsilon(1-\alpha)) + \alpha\tau_1\varepsilon(r\tau_2 + d_2\tau 3))\pi(R_0 - 1)}{K_1 K_2 K_3 \mu R_0} \end{aligned}\right\} \tag{15}$$

Where

$$K_1 = \tau_1 + \delta_L + \mu$$
$$K_2 = \tau_2 + \delta_{I_d} + \mu$$
$$K_3 = r + \tau_3 + \delta_{I_u} + \mu$$

4.3 Basic Reproduction Number (R_0)

Using next generation matrix [10],[13] the non-negative matrix F (new infection terms) and non-singular matrix V (other transferring terms) of the model are given, respectively by;

$$F = \begin{pmatrix} \frac{\beta(\theta I_d + I_u)S}{N} \\ 0 \\ 0 \end{pmatrix} \text{and } V = \begin{pmatrix} (\tau_1 + \delta_L + \mu)L \\ (\tau_2 + \delta_{I_d} + \mu)I_d - rI_u - (1-\alpha)\tau_1 L \\ (r + \tau_3 + \mu + \delta_{I_u})I_u - \alpha\tau_1 L \end{pmatrix} \quad (16)$$

After taking partial derivatives of F and$V$, we have:

$$F = \begin{pmatrix} 0 & \varepsilon\beta & \varepsilon\beta\theta \\ 0 & (1-\varepsilon)\beta & (1-\varepsilon)\beta\theta \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} K_1 & 0 & 0 \\ -(1-\alpha)\tau_1 & -r & K_2 \\ -\alpha\tau_1 & K_3 & 0 \end{pmatrix} \quad (17)$$

Thus;

$$R_0 = \frac{\beta(\theta\varepsilon K_3\tau_1(1-\alpha) + \theta K_1 K_2(1-\varepsilon) + \alpha\varepsilon\tau_1(K_2 + \theta r))}{K_1 K_2 K_3} \quad (18)$$

The threshold quantity R_0 is the basic reproduction number of the model system (2) for Ebola infection. It is the average number of new secondary infections generated by a single infected individual in his or her infectious period. [1].

4.4 Local Stability

4.4.1 Theorem 3

The disease-free equilibrium of the modeled in equation (2) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

4.4.2 Proof:

To determine the local stability ofE_0, the following Jacobian matrix is computed corresponding to equilibrium pointE_0.

Considering the stability of the disease-free equilibrium at the critical point $\left(\frac{\pi}{\mu}, 0,0,0,0\right)$.

$$J = \begin{pmatrix} -\mu & 0 & -\beta & -\beta\theta & 0 \\ 0 & -K_1 & \varepsilon\beta & \varepsilon\beta\theta & 0 \\ 0 & (1-\alpha)\tau_1 & r + (1-\varepsilon)\beta - K_2 & -K_2 & 0 \\ 0 & \alpha\tau_1 & -K_3 & 0 & 0 \\ 0 & 0 & \tau_3 & \tau_2 & -\mu \end{pmatrix} \quad (19)$$

A necessary and sufficient condition for local asymptotic stability is for the real part of the eigenvalue to be in the negative half plane [10]. Thus, it can show that$J(\varepsilon_0)$ given by (19) has eigenvalues all have a negative real part.

To this purpose, it is obvious from (19) that $-\mu$ (twice) are the two of the five eigenvalues of $J(E_0)$ since the first and fifth columns contain only the diagonal terms. Hence, the other three eigenvalues can be obtained from the sub-matrix of 3 by 3 matrix, $J^*(E_0)$ given by

$$J^* = \begin{pmatrix} -K_1 & \varepsilon\beta & \varepsilon\beta\theta \\ (1-\alpha)\tau_1 & r + (1-\varepsilon)\beta - K_2 & -K_2 \\ \alpha\tau_1 & -K_3 & 0 \end{pmatrix} \quad (20)$$

In what follows, the characteristic equation of $J^*(E_0)$ is of the form $|J^* - \lambda| = 0$ is given by:

$$J^* = \begin{pmatrix} -K_1 - \lambda & \varepsilon\beta & \varepsilon\beta\theta \\ (1-\alpha)\tau_1 & r + (1-\varepsilon)\beta - K_2 - \lambda & -K_2 \\ \alpha\tau_1 & -K_3 & -\lambda \end{pmatrix} \quad (21)$$

Simplifying matrix (21), can be written as:

$$B_3\lambda^3 + B_2\lambda^2 + B_1\lambda + B_0 = 0 \quad (22)$$

And

$$B_0 = K_1K_2K_3 - K_1(1-\varepsilon)\beta\theta K_3 - \varepsilon\beta K_2\alpha\tau_1 - \varepsilon\beta\theta K_3(1-\alpha)\tau_1 - \varepsilon\beta\theta\alpha\tau_1 r$$

It is easy to see that B_0 can be written in terms of R_0 as :

$$B_0 = 1 - \frac{\beta(\theta\varepsilon K_3\tau_1(1-\alpha)+\theta K_1K_2(1-\varepsilon)+\alpha\varepsilon\tau_1(K_2+\theta r))}{K_1K_2K_3} \quad (23)$$

If in (23) $R_0 < 1$, then $B_0 > 0$. Since the coefficientsB_i, i = 1,2,3 and the Hurwitz matrices of the polynomial (22) are positive, using Routh-Hurwitz criterion (see,[15]), all the eigenvalues of (22) have negative real parts. Therefore, the disease-free equilibrium, ε_0, is stable. Otherwise, whenever $R_0 > 1$, then$B_0 < 0$. By Descartes' rule of signs [16], there exists one eigenvalue with positive real part. Hence, ε_0 is unstable for$R_0 > 1$.

The implication of Theorem 3 is that reduction or elimination of Ebola Virus diseases governed by model (2) can be eliminated from the population whenever an influx by infected individual is small such that $R_0 < 1$.

4.5 Global Stability

4.5.1 Theorem 4

The disease free-equilibrium of the system in (2) is globally asymptotically stable (GAS) whenever $R_0 < 1$and unstable if$R_0 > 1$.

4.5.2 Proof:

Consider the linear Lyapunov function $V: \Gamma \to R_0$defined by

$$V = A_1L(t) + A_2I_d(t) + A_3I_u(t) \quad (24)$$

where $A_1 = \frac{(1-\alpha)\tau_1K_3+r\alpha\tau_1}{K_1K_3}$, $A_2 = 1$and $A_3 = \frac{r}{K_3}$The time derivative of (24) along the solution path of the system (2) is given by

$$V' = \left[\frac{(1-\alpha)\tau_1K_3+r\alpha\tau_1}{K_1K_3}\right]\left(\varepsilon\lambda S(t) - K_1L(t)\right) + \left((1-\varepsilon)\lambda S(t) - K_2I_d(t) + rI_u(t) + (1-\alpha)\tau_1L(t)\right) + \frac{r}{K_3}(\alpha\tau_1L(t) - K_3I_u(t)) \quad (25)$$

$$V' = \frac{(1-\alpha)\tau_1\varepsilon\lambda}{K_1}S(t) + \frac{r\alpha\tau_1\varepsilon\lambda}{K_1K_3}S(t) + \frac{K_2\alpha\tau_1\varepsilon\lambda}{\theta K_1K_3}S(t) + \frac{(1-\varepsilon)K_2\lambda}{K_3}S(t) - K_2I_d(t)$$

$$V' = \frac{(1-\alpha)\tau_1\varepsilon\beta\theta}{K_1}I_d(t) + \frac{r\alpha\tau_1\varepsilon\beta\theta}{K_1K_3}I_d(t) + \frac{K_2\alpha\tau_1\varepsilon\beta}{K_1K_3}I_d(t) + \frac{(1-\varepsilon)K_2\beta\theta}{K_3}I_d(t) - K_2I_d(t)$$

$$V' \leq \left[\frac{\beta(\theta\varepsilon K_3\tau_1(1-\alpha) + \theta K_1K_2(1-\varepsilon) + \alpha\varepsilon\tau_1(K_2+\theta r))}{K_1K_3} - K_2\right]I_d(t)$$

$$V' \leq K_2[R_0 - 1]I_d(t) \quad (26)$$

Thus, $V' \leq 0$ if $R_0 \leq 0$ with $V' = 0$if and only if $I_d = 0$. This shows that as $t \to \infty$, then (S(t), L(t), I_u(t), I_d(t), R(T)) $\to\left(\frac{\pi}{\mu}, 0,0,0,0\right)$. It follows that the largest compact invariant set in {(S(t), L(t), I_u(t), I_d(t), R(T)) $\in \Gamma$:V'=0} is the singletonE_0. Therefore, by LaSalle's Invariance Principle [17], the DFE given by E_0 is GAS in Γ if $R_0 \leq 0$.

The implication of Theorem 4 is that reduction or elimination of Ebola Virus disease is independent of the initial sizes of the sick people in the population. Hence, Ebola Virus disease can be eliminated if the associated reproduction number is less than unity.

4.6 Bifurcation Analysis

Bifurcation analysis is used to explore how the asymptotic stability of disease-free equilibrium is exchanged for asymptotic stability of endemic equilibrium of model (2) as the threshold quantity, R_o, cross the unity. In other words, to investigate the bifurcation at $R_o = 1$, using a center manifold theory of bifurcation analysis described by [18], used in some literatures like [19], [20], [21],[22], [23].

Choosing β as the bifurcation parameter, then at $R_o = 1$.

$$R_0 = \frac{\beta(\theta\varepsilon K_3\tau_1(1-\alpha)+\theta K_1K_2(1-\varepsilon)+\alpha\varepsilon\tau_1(K_2+\theta r))}{K_1K_2K_3} = 1 \tag{27}$$

then,

$$\beta^* = \frac{K_1K_2K_3}{(\theta\varepsilon K_3\tau_1(1-\alpha)+\theta K_1K_2(1-\varepsilon)+\alpha\varepsilon\tau_1(K_2+\theta r))} \tag{28}$$

So that the disease-free equilibrium, D_o, is locally stable when$\beta < \beta^*$, and is unstable when$\beta > \beta^*$, this,β^*, is bifurcation value.

The linearized matrix of the system (2) around the disease-free equilibrium E_o and evaluated at β^* is given by;

$$J(E_0,\beta^*) = \begin{pmatrix} -\mu & 0 & -\beta^* & -\beta^*\theta & 0 \\ 0 & -K_1 & \varepsilon\beta^* & \varepsilon\beta^*\theta & 0 \\ 0 & (1-\alpha)\tau_1 & r+(1-\varepsilon)\beta^*-K_2 & -K_2 & 0 \\ 0 & \alpha\tau_1 & -K_3 & 0 & 0 \\ 0 & 0 & \tau_3 & \tau_2 & -\mu \end{pmatrix} \tag{29}$$

The eigenvalues (λ) , of $J(E_o,\beta^*)$given by (29) are the roots of the characteristic equation of the form:

$$(\lambda+\mu)^2P(\lambda) = 0 \tag{30}$$

Where $P(\lambda)$is a polynomial of degree three whose roots are real and negative except one zero eigenvalue.

4.6.1 Determination of Right Eigen-vector and Left Eigen-vector

The right eigenvector,$w = (w_1, w_2, w_3, w_4, w_5)^T$, associated with this simple zero eigenvalue can be obtained from $J(D_o,\beta^*)w = 0$. Furthermore, the left eigenvector, $v = (v_1, v_2, v_3, v_4, v_5)$, corresponding to the simple zero eigenvalue of (29) is obtained from $vJ(D_o,\beta^*) = 0$

4.6.2 Computation of Bifurcation Coefficient

The direction of the bifurcation at $R_o = 1$ is determined by the signs of bifurcation coefficient "a" and "b", obtained from the above partial derivatives, given, respecting, by

$$a = \frac{DK_1[AC+\overleftrightarrow{B}K_1K_2K_3\theta(1-\varepsilon)]}{CY^2\pi}v_2w_2^2 \tag{31}$$

Similarly,

$$b = \frac{\varepsilon K_2\alpha\tau_1(1+\theta)-\theta Y}{K_2K_3} + \frac{\varepsilon K_1K_2\theta\alpha\tau_1(1-\varepsilon)(1+\theta)-K_1\theta^2Y(1-\varepsilon)}{K_2Y-K_1K_2K_3\theta(1-\varepsilon)}v_2w_2 \tag{32}$$

Where:

$$A = K_1Y - K_1K_2\alpha\tau_1(1-\varepsilon)$$
$$B = K_1K_2\alpha\tau_1(1-\varepsilon) - K_1Y$$
$$C = K_1K_2K_3\theta(1-\varepsilon) - K_2Y$$
$$Y = \theta\varepsilon K_3\tau_1(1-\alpha) + \theta K_1K_2(1-\varepsilon) + \alpha\varepsilon\tau_1(K_2+\theta r) \tag{33}$$

By numerical evaluation, using value of parameter in Table 3, it was found that $a < 0$ and$b > 0$, which follows from the theorem of [18] that the model (2) exhibits a supercritical (forward) bifurcation and the endemic equilibrium E* is locally asymptotically stable.

Table 3. Parameters Value and Source

Parameters	Value	Baseline	Source
τ_1	0.9 – 0.4	0.6	[2, 4]
τ_2	0.9 – 0.4	0.7	[5, 12]
τ_3	0.9 – 0.4	0.7	[9,12]
r	0.2 – 0	0.05	[5, 11]
π	1 – 0.2	0.9	[4, 6]
μ	0.2 – 0	0.1	[2,5]
α	0.8 – 0.4	0.5	Assumed
θ	0.9 – 0.2	0.6	[2, 5]
δ	0.2 – 0	0.01	Assumed
β	0.9 – 0.2	0.7	[6, 12]
ε	0.4 – 0.1	0.2	[10, 12]

5 Sensitivity Analysis

To determine how changes in parameters affect the transmission and spread of the disease, a sensitivity analysis of model (2) is carried out in the sense of [10],[17]. This was done to examines changing effects of the model parameters with respect to basic reproduction number, R_o, of the model (2).

Definition 1. The normalized forward-sensitivity index of a variable, v, depends differentiable on a parameter, p, is defined as:

$$\Upsilon_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v} \tag{34}$$

In particular, sensitivity indices of the basic reproduction number, R_o, with respect to the model parameter. The following results were obtained using the parameter value in

Table 4. Sensitivity indices with the Parameters

Parameter	Sign
β	Positive
θ	Positive
ε	Negative
α	Positive
τ_1	Positive
τ_2	Negative
τ_3	Negative
r	Negative

The positive sign of S.I of R_o to the model parameters shows that an increase (or decrease) in the value of each of the parameter in this case will lead to an increases (or decrease) in R_o of the model (2) and asymptotically results into persistence (or elimination) of the disease in the community . On the contrary, the negative sign of R_o to the model parameters indicates that an increase (or decrease) in the value of each of the parameter in this case leads to a corresponding decrease (or increases) on R_o of the model (2). Hence, with sensitivity analysis, one can get insight on the appropriate intervention strategies to prevent and control the spread of the disease described by model (2).

Table 5. Sensitivity value with the Parameters

Parameter	Sign
β	+ 1
θ	+ 0.7361563518
α	+ 0.2159609121
τ_1	+ 0.03905979791
r	- 0.04219721649
τ_2	- 0.02054932245
τ_3	- 0.02054932245
ε	- 0.01768264309

The most sensitive parameter isβfollow by$\theta$and the least sensitive parameter is ε.
All these eight parameters play an important role in the dynamical spread of the Ebola Virus disease in the population. The effect of some of them will be graphically illustrated below.

6 Results and Discussion

Numerical simulation was carried out by MAPLE 18 software using Runge-Kutta method of order four with the set of parameter values given in Table 3. Dynamic spread of Ebola is checked simultaneously on Recovered, Susceptible, Infected undetected, Infected detected and Latent individuals since the spread of Ebola is a function of time. S(0) =7, Id(0) =0, $I_u(0)$=0, R(0)=0, L(0)=0 Figs 2-6 below are the results obtained from numerical simulation of the Ebola model with the dynamic spread.
In this study, five (5) deterministic epidemiological model of (S, L, I_u, I_d, R) are presented to gain insight into the dynamical spread of Ebola virus disease. Positivity of solution shows that, the model presented is mathematically and epidemiologically well posed. Local and global stability of the model shows that, disease-free equilibrium is asymptotically stable whenever the threshold quantity 'R_0' is less than unity and otherwise endemic when it is greater than unity.
The sensitivity analysis reveals that eight (8) parameters plays an important role in the dynamical spread of Ebola Virus disease according to the model (2), the parameters are τ_1,β,θ,ε,α,r,τ_2 and τ_3. Four (4) were positive and four (4) were negative as it can be seen in Table 4 and Table 5, increasing those with positive index will result in the higher spread of the disease in the population, so effects must be made to keep it loss while increasing those with negative index will result in the reducing the spread of the disease in the population, so effects must be made to raise it up.
The bifurcation analysis was a forward which shows that the disease can be control if all effect is put in place to force $R_0$to be less than one.
Figures. 2-5 of numerical simulation showed that, shows the behavior of some parameters on the dynamical spread of Ebola Virus diseases.

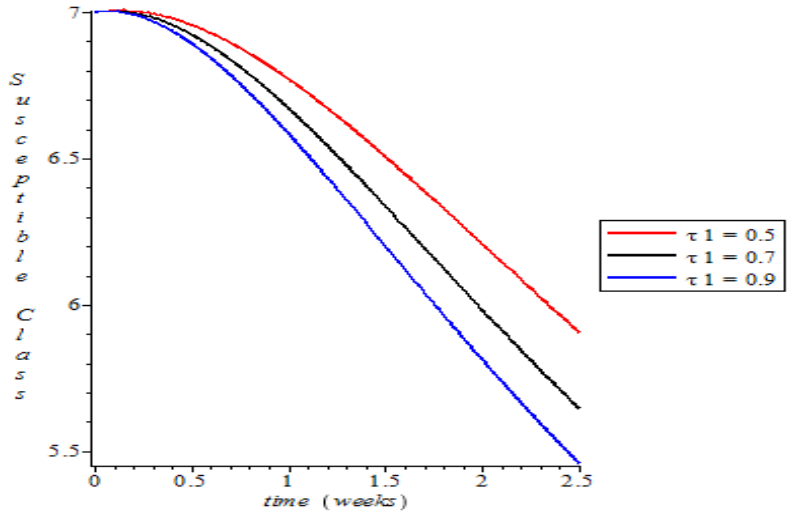

Figure 2(a) Effect of Progression rate of infected individual on susceptible class.

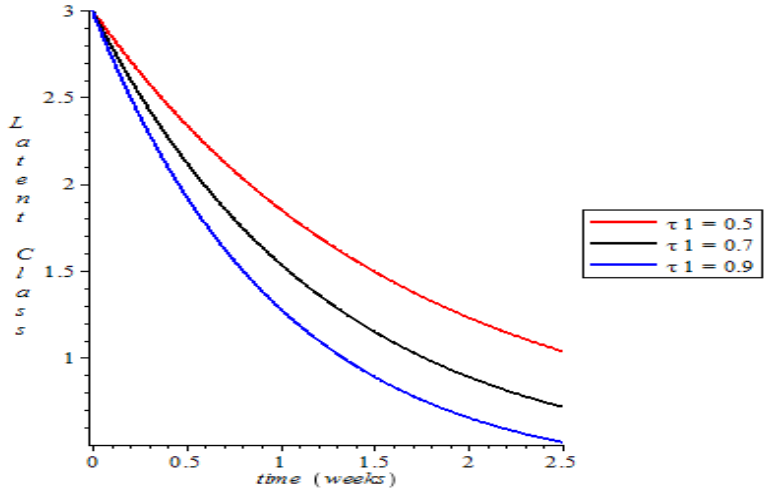

Figure 2(b) Effect of Progression rate of infected individual on latent class.

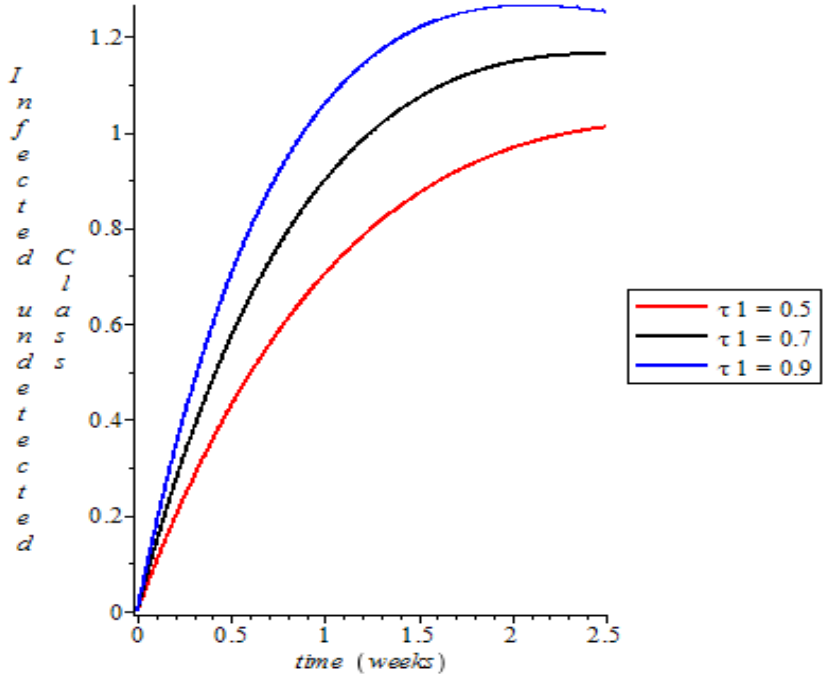

Figure 2(c) Effect of Progression rate of infected individual on infected undetected class.

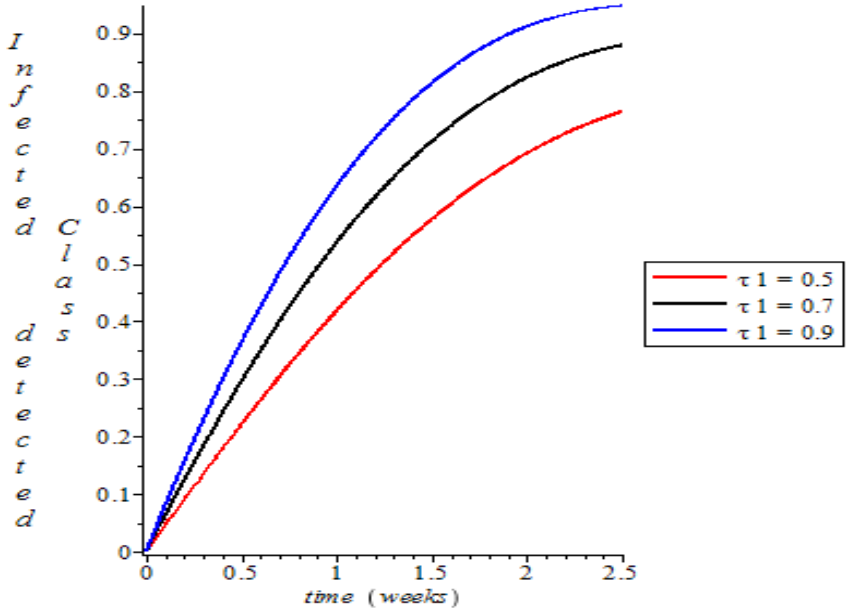

Figure 2(d) Effect of Progression rate of infected individual on infected detected class.

Figure 2a-d, shows the effect of progression rate of infected individual to infectious individual (τ_1): **Figure 2a** reveals it effect on susceptible individuals (S), as$\tau_1$increases S decreases with time, **Figure 2b** shows the effect of $\tau_1$on latently infected individuals (L), as$\tau_1$increases L decreases with time, **Figure 2c**Pointed out the effect of $\tau_1$on infected undetected individuals (I_u), asτ_1 increases I_u increases with time and **Figure 2d** depicted the effect of $\tau_1$on infected detected individuals (I_d), asτ_1 increases I_d increases with time. This confirmed the sensitivity analysis results.

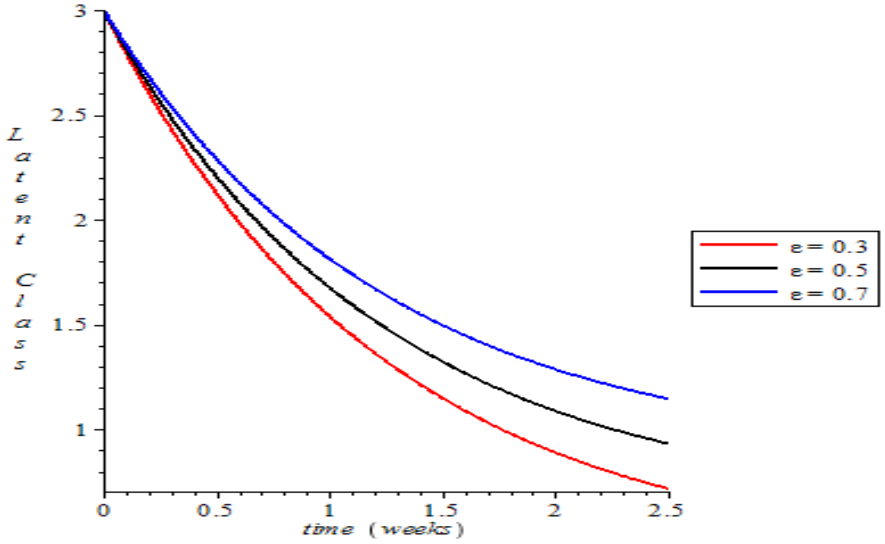

Figure 3(a) Impact of Slow progressor on latent class

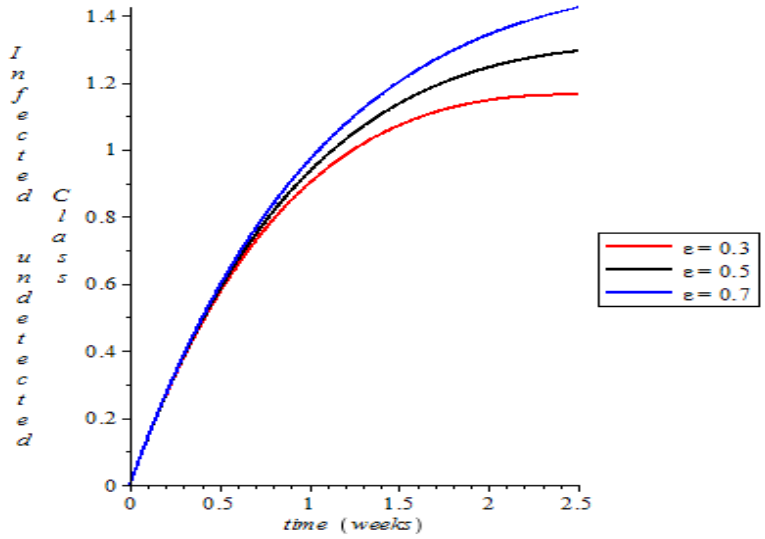

Figure 3(b) Impact of Slow progressor on infected undetected class

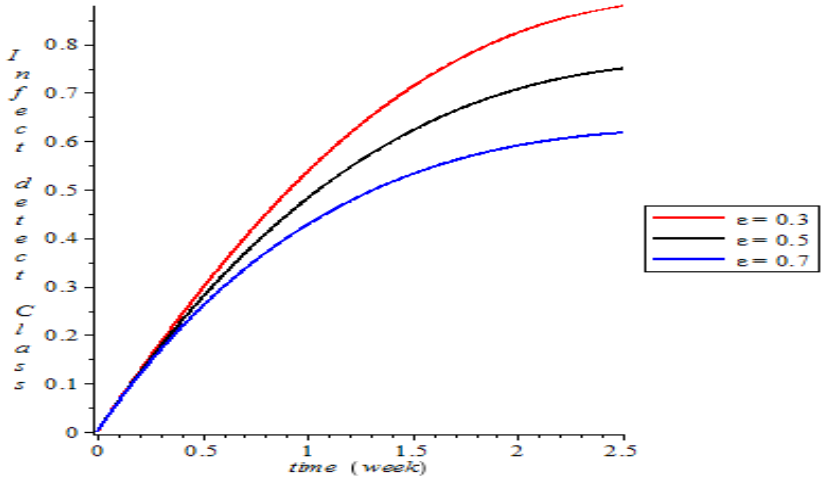

Figure 3(c) Impact of Slow progressor on infected detected class

Figure 3a-c, reveals the impact of slow progressor (ε):**Figure 3a** depicted the effect of εon latently infected individuals (L), as ε increases L increases with time, **Figure 3b** shows the effect of εon infected undetected individuals (I_u), asε increases I_u increases with time and **Figure 3c** pointed out the effect of εon infected detected individuals (I_d), as ε increases I_d increases with time. Sensitivity analysis was established with this result.

Figure 4a-b, pointed out the significant of detection rate of infected undetected individual (r): **Figure 4a** reveals the effect of $r$on infected undetected individuals (I_u), as$r$increases I_u decreases with time and **Figure 4b** shows the effect of $r$on infected detected individuals (I_d), as$r$increases I_d increases with time. This is in agreement with the sensitivity and bifurcation analysis results.

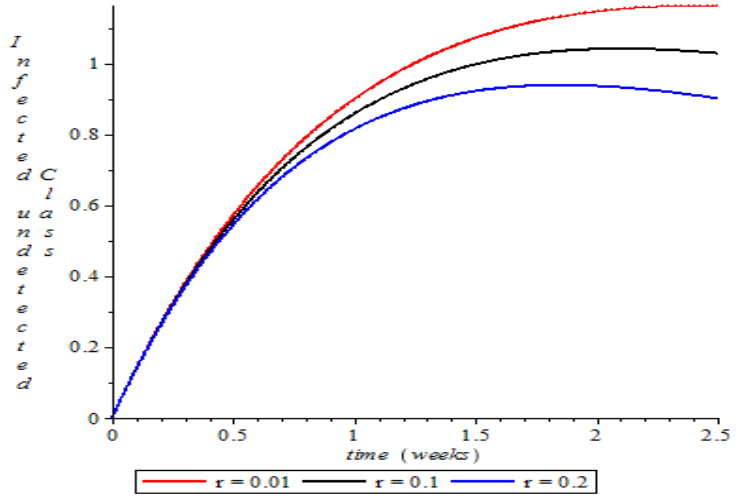

Figure 4(a) Significant of detection rate of infected undetected individual on infected undetected class

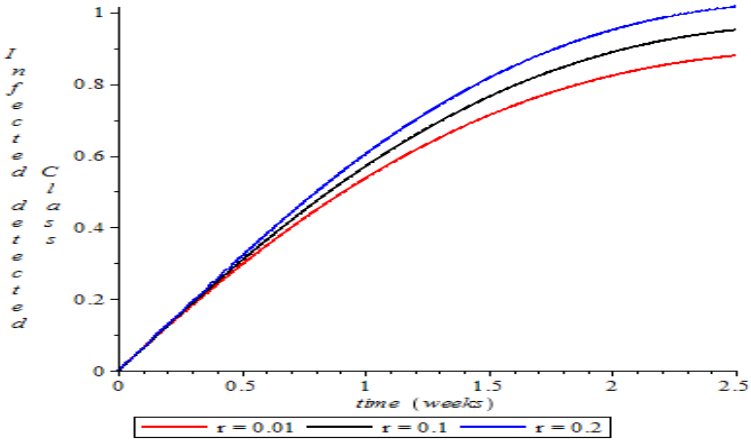

Figure 4(b) Significant of detection rate of infected undetected individual on infected detected class

Figure 5a-c, shows the effects of endogenous reactivation rate on the population (α):**Figure 5a** reveals it effect on susceptible individuals (S), as α increases S decreases with time, **Figure 5b** pointed out the effect of on infected undetected individuals (I_u), as decreases I_u increases with time, and **Figure 5c** Pointed out the effect of α on infected undetected individuals (I_d), as α decreases I_d increases with time..

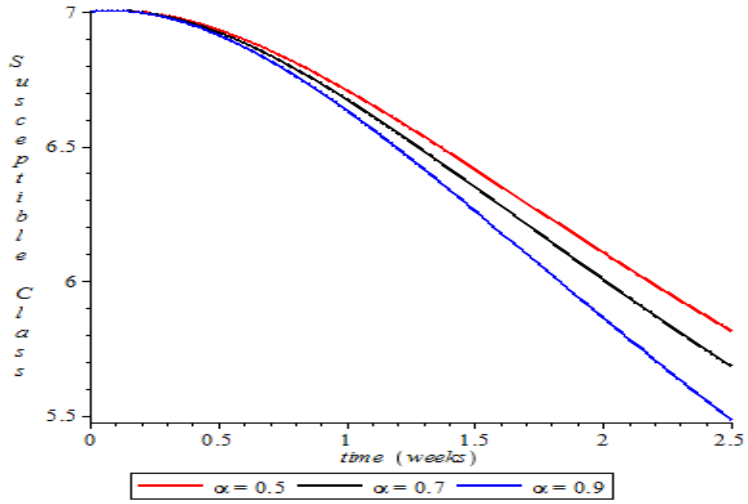

Figure 5(a) Effect of endogenous reactivation rate on susceptible class

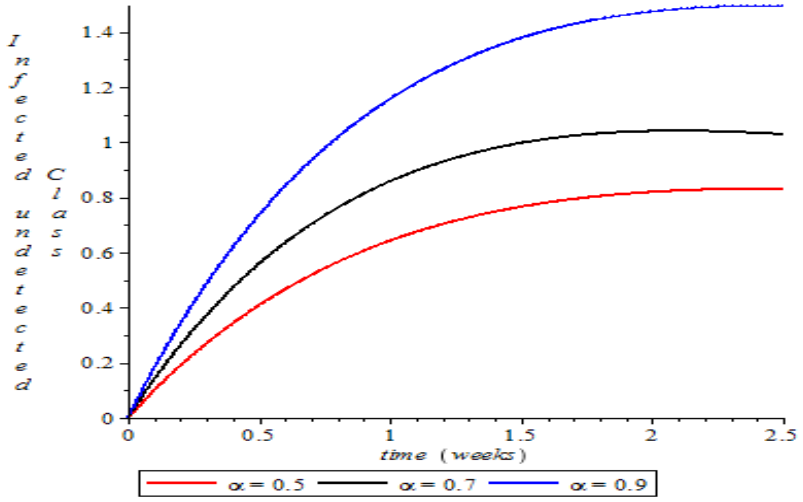

Figure 5(b) Effect of endogenous reactivation rate on infected undetected class

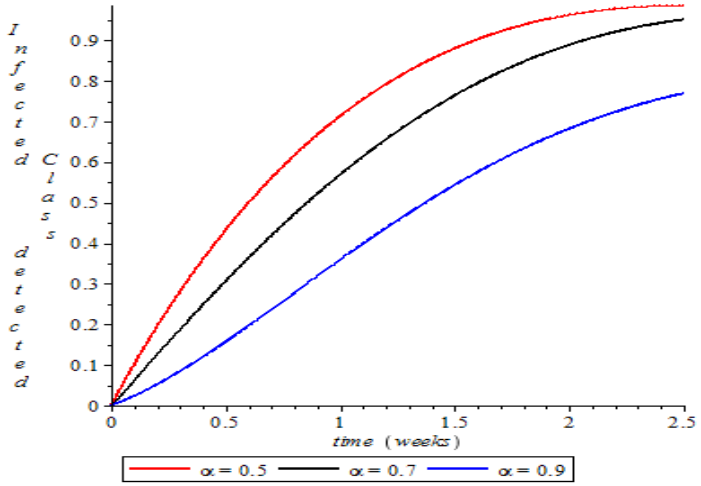

Figure 5(c) Effect of endogenous reactivation rate on infected undetected class

7 Conclusion

In conclusion, reduction or elimination of Ebola Virus diseases governed by model (2) can be eliminated from the population whenever an influx by infected individual is small such that$R_0 < 1$, also reduction or elimination of Ebola Virus disease is independent of the initial sizes of the sick people in the population. Hence, Ebola Virus disease can be eliminated if the associated reproduction number is less than unity. The bifurcation analysis was a forward which shows that the disease can be control if all effect is put in place to force R_0 to be less than one. The sensitivity analysis reveals that four (4) were positive, which are τ_1, β, θ and, α; increasing these one will result in the more spread of the disease in the population, all hand must be on deck to keep it loss. Four (4) were negative are ε,r,τ_2 and τ_3; increasing those with negative index will result in the reducing the spread of the disease in the population, so effects must be made to raise it up.

8 Competing Interests

The author declared that no conflict of interest exists in this publication.

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References

[1] Bishop B.M. Potential and emerging treatment options for Ebola virus disease. Ann Pharmacother(2014) 143 -161.
[2] Marsh G. A., Haining J. and Robinson R. Ebola:Reston virus infection of pigs:clinical signifcance and transmission potential. J Infect Dis., 204(Suppl 3):(2011)S804–S809.
[3] Amira Rachah. A mathematical model with isolation for the dynamics of Ebola virus, IOP Conf. Series: Journal of Physics: Conf. Series **1132** (2018) 012058, doi:10.1088/1742-6596/1132/1/012058.
[4] J. Astacio, D. Briere, M. Guillen, J. Martinez, F. Rodriguez and N. Valenzuela-Campos. Mathematical models to study the outbreaks of Ebola, Biometrics Unit Technical Report, Numebr BU-1365-M, Cornell University (1996) 231 pages.
[5] G. Chowell, N. W. Hengartner, C. Castillo-Chavez, P. W. Fenimore, and J. M. Hyman. The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda, J.Theor. Biol., vol 229, no. 1, (2004) pp. 119-126.
[6] C. Rizkalla, F. Blanco-Silva, and S. Gruver. Modeling the impact of Ebola and bushmeathunting on western lowland gorillas, EcoHealth J. Consortium, vol 4, (2007) pp. 151-155.
[7] Mhlanga, A. Dynamical analysis and control strategies in modeling Ebola virus disease. Advance differential equation 2019, 458 (2019).
[8] Qiu X., Wong G. and Audet J. Reversion of advanced Ebola virus disease in nonhuman primates with ZMapp. Nature. 514: (2014). 47–53
[9] Abdulrahman N., Sirajo A., and Abdulrazaq A. A mathematical model for the controlling the spread of Ebola virus disease in Nigeria. International Journal of Humanities and Management Sciences (IJHMS) Volume 3, Issue 3, (2015). ISSN 2320–4044.
[10] J. A. Lewnard, M. L. NdeffoMbah, J. A. and Alfaro-Murillo. Dynamics and control of Ebola virus transmission inMontserrado, Liberia: a mathematical modelling analysis, The Lancet Infectious Diseases, vol. 14, no. 12, (2015) pp. 1189–1195.
[11] Kelly, J., et al. Projection of Ebola outbreak size and duration with and without vaccine use in Equateur, Democratic Repulic of Congo, as of May 27, 2018. PLoS ONE14, e0213190 (2019).
[12] A. Rachah and D. F.M. Torres. Mathematical Modelling, Simulation, and Optimal Control of The 2014 Ebola Outbreak in West Africa. Hindawi Publishing Corporation, Discrete Dynamics in Nature and Society, Volume 2015, Article ID 842792, (2015)9 pages.
[13] Akanni J.O. and Akinpelu F.O. An HIV/AIDs model with vertical transmission, treatment and progression rate. Asian Research Journal of Mathematics, 1(4):(2016)1-17, Article no.ARJOM.28549.
[14] Lineberry T. W. and Bostwick J. M. Methamphetamine abuse: A perfect storm of complications Mayo Clin Proc. 2006; 81:(2006)77–84.
[15] Plüddemann A., Dada S. and Parry C. Monitoring alcohol and substance abuse trends in South Africa. SACENDU Research brief,13 (2):(2010)1–16.
[16] A. Plüddemann and C. D. H. Parry. Methamphetamine use and associated problems among adolescents in the Western Cape province of South Africa. MRC South Africa.(2012) 211 pages.
[17] LaSalle JP (1976). The stability of dynamical systems. In: Regional conference series in applied mathematics. SIAM, Philadelphia, Pa.
[18] Castillo-Chavez C. and Song B. Dynamical models of tuberculosis and theirapplications. Math Biosci Eng.,1:(2004) 361–404.
[19] Chitnis, N.; Cushing, J. M. and Hyman, J. M. Bifurcation Analysis of a Mathematical model for malaria transmission. SIAM J. Appl. Math. 67 (1), (2006) 24-45.
[20] J. Arino, C. C. McCluskey, and P. van den Driessche. Global results for an epidemic model with vaccination that exhibits backward bifurcation, SIAM Journal on Applied Mathematics, vol. 64, no. 1,(2003) pp. 260–276.
[21] S. Olaniyi and O.S. Obabiyi. Qualitative analysis of malaria dynamics with nonlinear incidence function, Applied Mathematical Sciences, 8, No 78, (2014)3889–3904.
[22] Garba S. andGumel A., Bakar M. Backward bifurcation in dengue transmissiondynamics. Math Biosci., 215:(2008) 11–25.
[23] A. D. Adediipo, J. O. Akanni and O. M. Shangodare. Bifurcation and Stability Analysis of the Dynamics of Gonorrhea Disease in the Population, World Scientific News 143 (2020) 139-154.
[24] Mupere E., Kaducu O.F. andYoti Z. Ebola haemorrhagic fever among hospitalised children and adolescents in northern Uganda: epidemiologic and clinical observations. Afr Health Sci.1:(2001) 60–65.
[25] Dowell S. F. Ebola hemorrhagic fever: why were children spared? Pediatr Infect Dis J. 15:(1996)189–191.