Covenant Journal of Physical & Life Sciences (CJPL) Vol. 8 No. 2, Dec 2020
ISSN: *print 2354 - 3574 electronics 2354-3485DOI:*

An Open Access Journal Available Online

Stability Analysis of the Dynamical Spread of Ebola Virus Disease

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Received: 16.04.2020; Accepted: 19.11.2020
Date of Publication: December, 2020

***Abstract*:** The model was governed by a system of ordinary differential equations; with the total population sub-divided into Susceptible individuals (S), Latently individuals (L), Infected undetected individuals (I_u) , Infected detected individuals (I_d) and Recovered individuals (R). Theory of positivity and boundedness was used to investigate the well-posedness of the model. Equilibrium solutions were investigated analytically. The basic reproduction number (R_0) was calculated using the next generation method. Bifurcation analysis and global stability of the model were carried out using centre manifold theory and Lyapunov functions respectively. The effects of parameters such as Progression rate of infected individual to infectious individual (τ_1), Effective contact rate (β), Modification parameter (θ), Slow progressor (ε), Endogenous reactivation rate (α), Detection rate of infected undetected individual (r), Recovery rate of infected detected individual due to treatment (τ_2) and Recovery rate of infected undetected individual due to treatment (τ_3) on R_0 were explored through sensitivity analysis. To reduce the burden of Ebola virus disease in the population the following parameters, $\tau_1, \beta, \theta, \varepsilon, \alpha, r, \tau_2$, and τ_3 play a significant role in the spread of it in the population. Numerical simulation is analyzed by MAPLE 18 software using embedded Runge-Kutta method of order (4) which shows the dynamical spread of Ebola virus disease.

Keywords: Ebola virus disease; infected, reproduction number, critical points, bifurcation analysis and Lyapunov functions.

1.0 Introduction

Ebola virus is most commonly spread via personal contact, and it has an incubation period of two to twenty–one days [9]. It takes approximately eight hours for the virus to replicate, and can occur several times before the onset of symptoms. "Hundreds to thousands of new virus particles are then released during periods of hours to a few days, before it finally kills the cell."[1]. Ebola virus disease (EVD) also known as Ebola haemorrhagic fever which was named after the river in Democratic Republic of Congo (DRC, formerly Zaire) where it was firstly discovered in 1976, is a lethal virus for humans then [12] and a virulent filovirus that is known to affect humans and primates. The symptoms that occur within a few days after transmission include, high fever, headache, muscle aches, stomach pain, fatigue, diarrhea sore throat, hiccups, rash, red and itchy eyes, vomiting blood, bloody diarrhea [2]. The death rate of Ebola virus infection is somewhere between 50 % to 90 %, which means it is a deadly disease. Until now, there is no specific cure or vaccine for Ebola but, efforts are on-going to find a viable treatment [9].

The first known outbreak of Ebola was in 1976, it occurs the same time in the Democratic Republic of the Congo (DRC) and Sudan, and it was recorded that each has a death rate beyond 50%, this disease then disappeared after 1979 and did not re-appear again until 1994 [3]. As of October 8, 2014, the World Health Organization (WHO) reported 4656 cases of Ebola virus deaths, with most cases occurring in Liberia [10]. The extremely rapid increase of the disease and the high mortality rate make this virus a major problem for public health of the world [11]. The outbreaks have been occurring with increasing frequency, the most horrible outbreak of Ebola till date is currently occurring in West Africa, and it's been a long affair that has infected well over 24000 [9].

The present 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. A great many mathematicians have developed 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. In this study, a mathematical model was formulated and analyzed to see the effect of some parameter on the dynamical spread of Ebola virus diseases in the population.

2.0 Mathematical Model

The study used five (5) compartmental deterministic mathematical model of the S, L, I_u, I_d, Rto 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 $\boldsymbol{I_u(t)}$, Infected detected $\boldsymbol{I_d(t)}$, and recovered ***R(t)***,

Where

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

Susceptible (S): 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

rate π. The population decreased by natural death at a rate μ also, by force of infection of infected detected individuals λ.

Latent (E): 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 rate μ.

Infected detected (I_d): 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.

Infected undetected (I_u): 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.

Recovered (R): 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).

Hence, we have the following nonlinear system of differential equations:

2.1 Model Equation

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

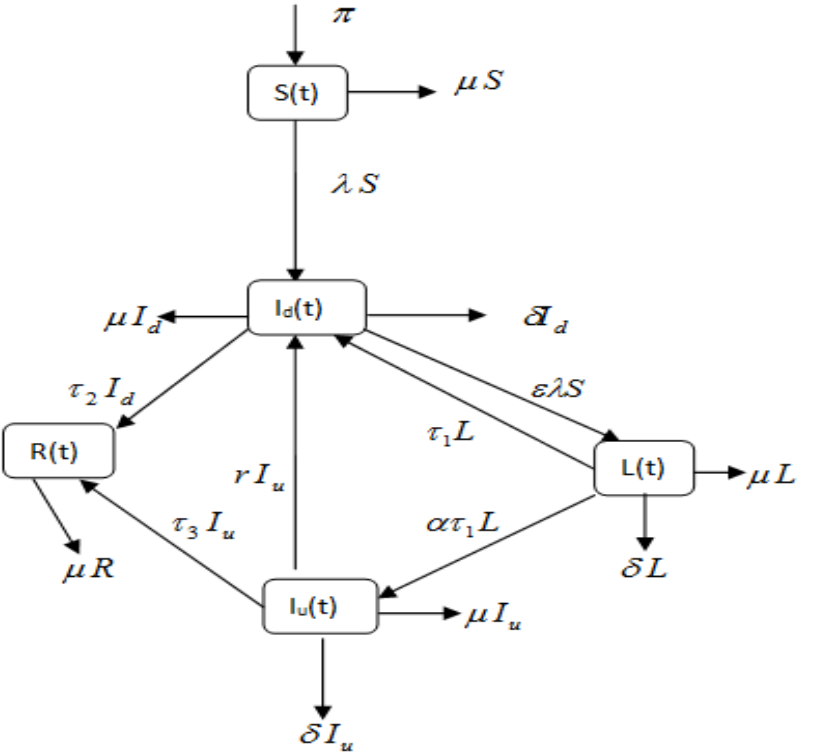

Figure 1. Flow Chat

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

3.0 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)

$$\frac{dS}{dt} = \pi - \lambda S(t) - \mu S(t)$$

$$\frac{dL}{dt} = \varepsilon \lambda S(t) - (\tau_1 + \delta_L + \mu) L(t)$$

$$\frac{dI_d}{dt} = (1-\varepsilon)\lambda S(t) - (\tau_2 + \delta_{I_d} + \mu) I_d(t) + r I_u(t) + (1-\alpha)\tau_1 L(t)$$

$$\frac{dI_u}{dt} = \alpha \tau_1 L(t) - (r + \tau_3 + \delta_{I_U} + \mu) I_u(t)$$

$$\frac{dR}{dt} = \tau_2 I_d(t) + \tau_3 I_u(t) - \mu R(t)$$

where

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

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

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

So that,

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

From which follows that

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

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:

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

It can be shown that Γ is positively invariant region.

3.1.3 Theorem 2

The region Γ is positively invariant with respect to the model (2)

3.1.4 Proof

The rate of change of the total population is given by

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

It results into the solution;

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

(3)

It follows that $N(t)\to\frac{\pi}{\mu}$ as $t\to\infty$, in particular, $N(t)\le\frac{\pi}{\mu}$, if $N(0)\le\frac{\pi}{\mu}$ 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].

$$\frac{dS}{dt}=\frac{dL}{dt}=\frac{dI_u}{dt}=\frac{dI_d}{dt}=\frac{dR}{dt}=0 \qquad (11)$$

4.0 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 the compartment are set to be zero;

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

$$E_0=(S,L,I_u,I_d,R)=\left(\frac{\pi}{\mu},0,0,0,0\right) \qquad (12)$$

4.2 Endemic Equilibrium

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

$$\left.\begin{aligned} S^* &= \frac{\pi}{\mu R_0} \\ L^* &= \frac{\varepsilon \pi (R_0 - 1)}{d_1 R_0} \\ I_u{}^* &= \frac{\alpha \tau_1 \varepsilon \pi (R_0 - 1)}{K_1 K_3 R_0} \\ I_d{}^* &= \frac{(K_1 K_3 (1-\varepsilon) + K_3 \tau_1 \varepsilon (1-\alpha) + r \alpha \tau_1 \varepsilon) \pi (R_0 - 1)}{K_1 K_2 K_3 R_0} \\ R^* &= \frac{(\tau_2 (K_1 K_3 (1-\varepsilon) + K_3 \tau_1 \varepsilon (1-\alpha)) + \alpha \tau_1 \varepsilon (r \tau_2 + d_2 \tau 3)) \pi (R_0 - 1)}{K_1 K_2 K_3 \mu R_0} \end{aligned}\right\} \quad (13)$$

Where

$$K_1 = \tau_1 + \delta_L + \mu$$
$$K_2 = \tau_2 + \delta_{I_d} + \mu$$
$$K_3 = r + \tau_3 + \delta_{I_u} + \mu \quad (14)$$

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;

$$F = \begin{pmatrix} \frac{\beta(\theta I_d + I_u)S}{N} \\ 0 \\ 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} (\tau_1 + \delta_L + \mu)L \\ (\tau_2 + \delta_{I_d} + \mu)I_d - r I_u - (1-\alpha)\tau_1 L \\ (r + \tau_3 + \mu + \delta_{I_u})I_u - \alpha \tau_1 L \end{pmatrix} \quad (15)$$

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

$$F = \begin{pmatrix} 0 & \varepsilon\beta & \varepsilon\beta\theta \\ 0 & (1-\varepsilon)\beta & (1-\varepsilon)\beta\theta \\ 0 & 0 & 0 \end{pmatrix} \text{ and } \quad V = \begin{pmatrix} K_1 & 0 & 0 \\ -(1-\alpha)\tau_1 & -r & K_2 \\ -\alpha\tau_1 & K_3 & 0 \end{pmatrix} \quad (16)$$

Thus;

$$R_0 = \frac{\beta(\theta\varepsilon K_3 \tau_1 (1-\alpha) + \theta K_1 K_2 (1-\varepsilon) + \alpha\varepsilon\tau_1 (K_2 + \theta r))}{K_1 K_2 K_3} \quad (17)$$

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 [13,21,23].

4.4 Local Stability

4.4.1 Theorem 3: The disease free equilibrium of the model (2) is locally

asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

To determine the local stability of E_0, the following Jacobian matrix is computed corresponding to equilibrium point E_0.

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

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

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 $J(E_0)$ given by (18) has eigenvalues all have a negative real part. To this purpose, it is obvious from (18) 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

4.4.2 Proof:

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

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

(18)

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

(20)

Simplifying matrix (20), can be written as:

$$B_3\lambda^3 + B_2\lambda^2 + B_1\lambda + B_0 = 0 \quad (21)$$

And

$$B_0 = K_1K_2K_3 - K_1(1-\varepsilon)\beta\theta K_3 - \varepsilon\beta K_2\alpha\tau_1 - \varepsilon\beta\theta K_3(1-\alpha)\tau_1 - \varepsilon\beta\theta\alpha\tau_1 r \quad (22)$$

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

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

If in (23) $R_0 < 1$, then $B_0 > 0$. Since the coefficients B_i, i = 1,2,3 and the Hurwitz matrices of the polynomial (21) are positive, using Routh-Hurwitz criterion (see,[15]), all the eigenvalues of (21) have negative real parts. Therefore, the disease free equilibrium, E_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, E_0 is unstable for $R_0 > 1$.

The implication of Theorem 3 is that the problem of Ebola Virus diseases governed by model (2) will be wiped out from the population, if the initial size of the sick sub-populations are in the basin of attraction of the E_0.

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

$$V = A_1 L(t) + A_2 I_d(t) + A_3 I_u(t) \qquad (24)$$

where $A_1 = \frac{(1-\alpha)\tau_1 K_3 + r\alpha\tau_1}{K_1 K_3}$, $A_2 = 1$ and $A_3 = \frac{r}{K_3}$

The time derivative of (24) along the solution path of the system (2) is given by

$$V' = \left[\frac{(1-\alpha)\tau_1 K_3 + r\alpha\tau_1}{K_1 K_3}\right](\varepsilon\lambda S(t) - K_1 L(t)) + ((1-\varepsilon)\lambda S(t) - K_2 I_d(t) + r I_u(t) + (1-\alpha)\tau_1 L(t))$$

$$+ \frac{r}{K_3}(\alpha\tau_1 L(t) - K_3 I_u(t))$$

(25)

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

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

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

$$V' = K_2[R_0 - 1] I_d(t)$$

(26)

Thus, $V' \le 0$ if $R_0 \le 0$ 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 singleton E_0. Therefore by LaSalle's Invariance Principle [17], the DFE given by E_0 is GAS in Γ if $R_0 \le 0$.

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 – 23].

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

$$\cdot\ R_0 = \frac{\beta(\theta\varepsilon K_3\tau_1(1-\alpha) + \theta K_1 K_2(1-\varepsilon) + \alpha\varepsilon\tau_1(K_2 + \theta r))}{K_1 K_2 K_3} = 1 \qquad (27)$$

then, $\beta^* = \frac{K_1 K_2 K_3}{(\theta \varepsilon K_3 \tau_1 (1-\alpha) + \theta K_1 K_2 (1-\varepsilon) + \alpha \varepsilon \tau_1 (K_2 + \theta r))}$ (28)

So that the disease-free equilibrium, E_0, is locally stable when $\beta < \beta^*$, and is unstable when $\beta > \beta^*$, this, β^*, is bifurcation value.

Then,

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

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

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

$(\lambda + \mu)^2 P(\lambda) = 0$ (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 $v\ J(D_o, \beta^*) = 0$

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

$$a = \frac{D K_1 [AC + B K_1 K_2 K_3 \theta (1-\varepsilon)]}{C Y^2 \pi} v_2 w_2^2$$

(31)

Similarly,

$$b = \frac{\varepsilon K_2 \alpha \tau_1 (1+\theta) - \theta Y}{K_2 K_3} + \frac{\varepsilon K_1 K_2 \theta \alpha \tau_1 (1-\varepsilon)(1+\theta) - K_1 \theta^2 Y (1-\varepsilon)}{K_2 Y - K_1 K_2 K_3 \theta (1-\varepsilon)} v_2 w_2$$

(32)

Where:

$$A = K_1 Y - K_1 K_2 \alpha \tau_1 (1-\varepsilon)$$
$$B = K_1 K_2 \alpha \tau_1 (1-\varepsilon) - K_1 Y$$
$$C = K_1 K_2 K_3 \theta (1-\varepsilon) - K_2 Y$$
$$Y = \theta \varepsilon K_3 \tau_1 (1-\alpha) + \theta K_1 K_2 (1-\varepsilon) + \alpha \varepsilon \tau_1 (K_2 + \theta r) \quad (33)$$

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

5.0 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 [9, 22-23]. This was done to examine changing effects of the model parameters with respect to basic reproduction number, R_o, of the model (2).

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

$$\Upsilon_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v} \quad (34)$$

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 3

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 is β followed by θ 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.

6.0 Numerical Simulation

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. $S(0) =300, I_d(0)=0.02, I_u(0)=150$,$R(0)=100, L(0)=100$ Figs 2-6 below are the results obtained from numerical simulation of the Ebola model with the dynamic spread

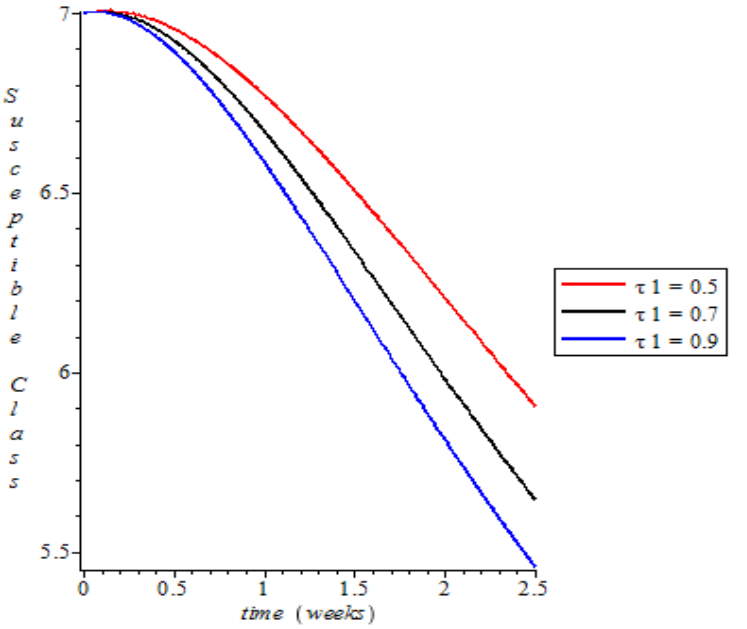

Figure 2(a) Graph of Progression rate of infected individual to infectious individual on susceptible class

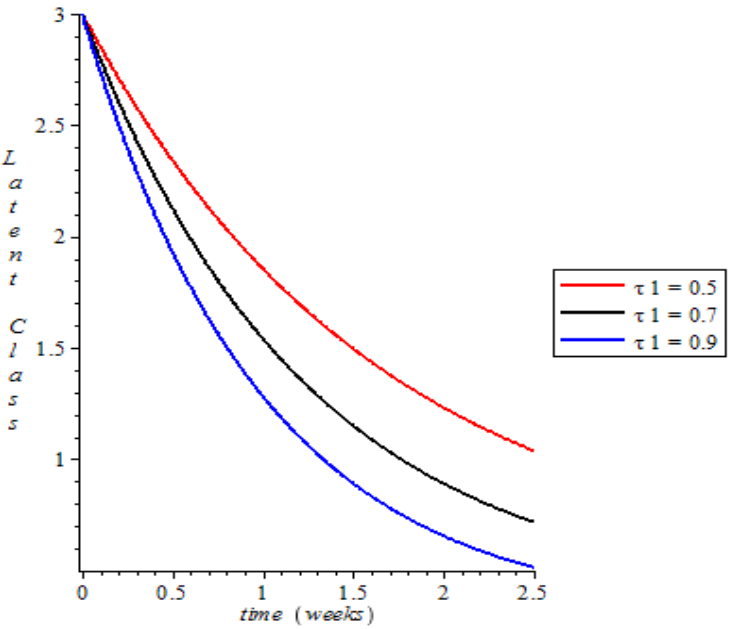

Figure 2(b) Graph of Progression rate of infected individual to infectious individual on latent class

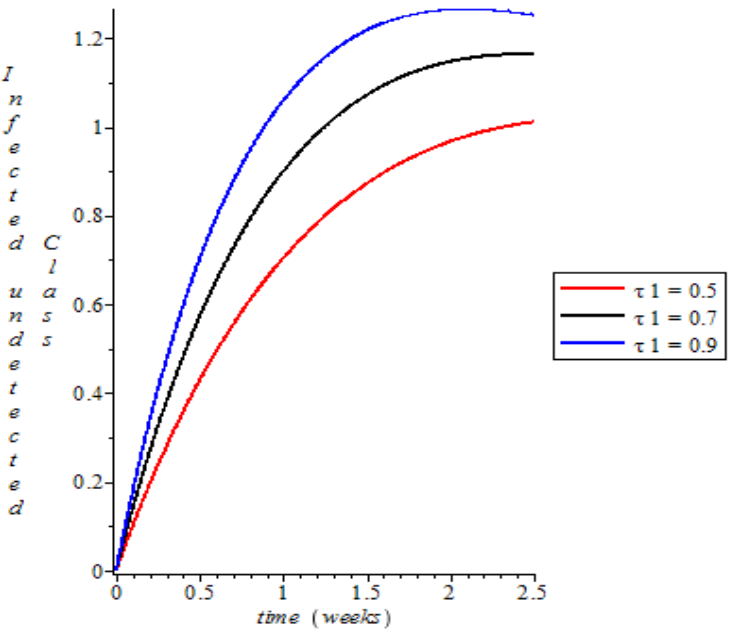

Figure 2(c) Graph of Progression rate of infected individual to infectious individual on infected undetected class

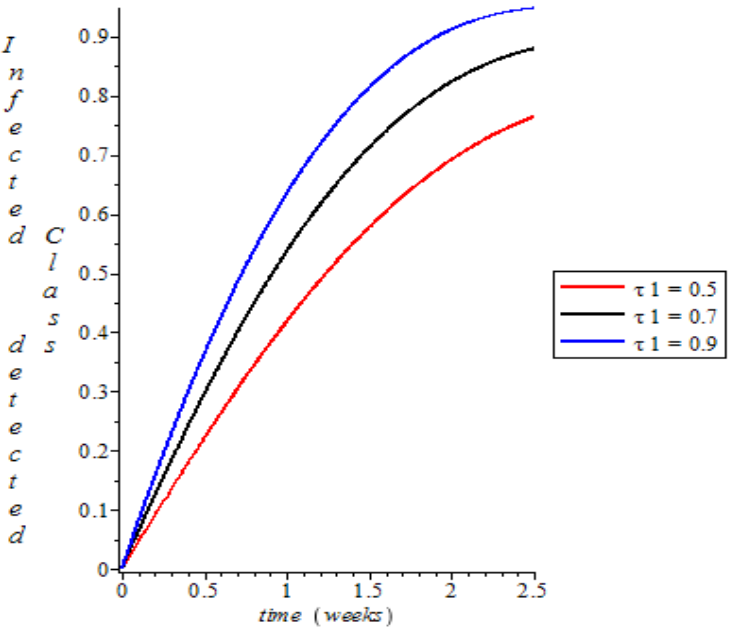

Figure 2(d) Graph of Progression rate of infected individual to infectious individual on infected detected class

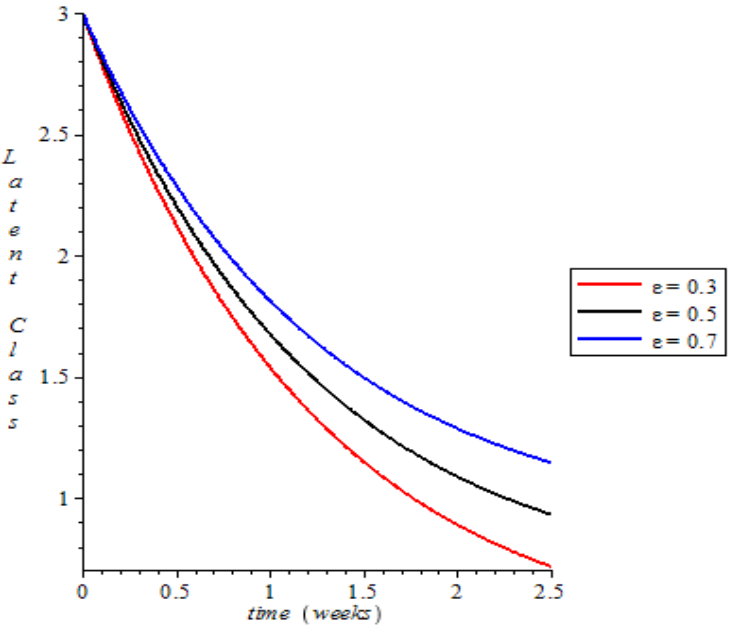

Figure 3(a) Graph of Slow progressor on latent class

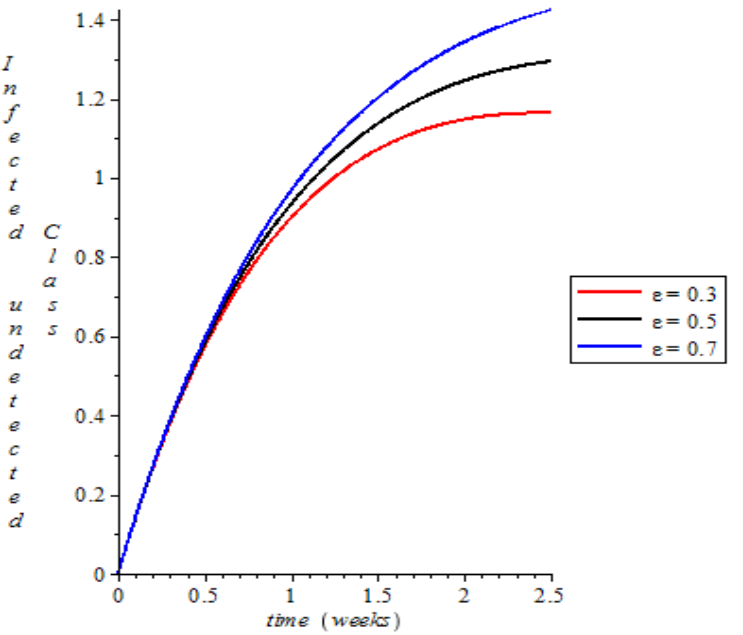

Figure 3(b) Graph of Slow progressor on infected undetected class

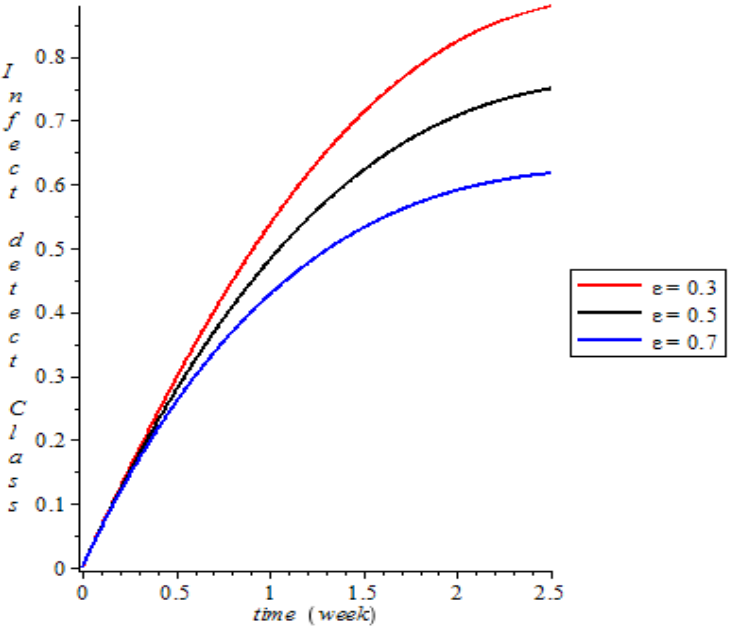

Figure 3(c) Graph of Slow progressor on infected detected class

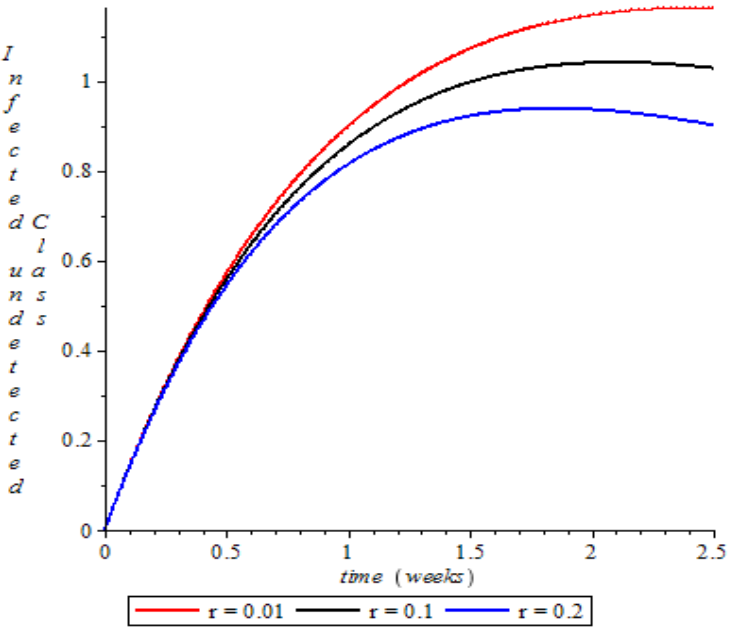

Figure 4(a) Graph of detection rate of infected undetected individual on infected undetected class

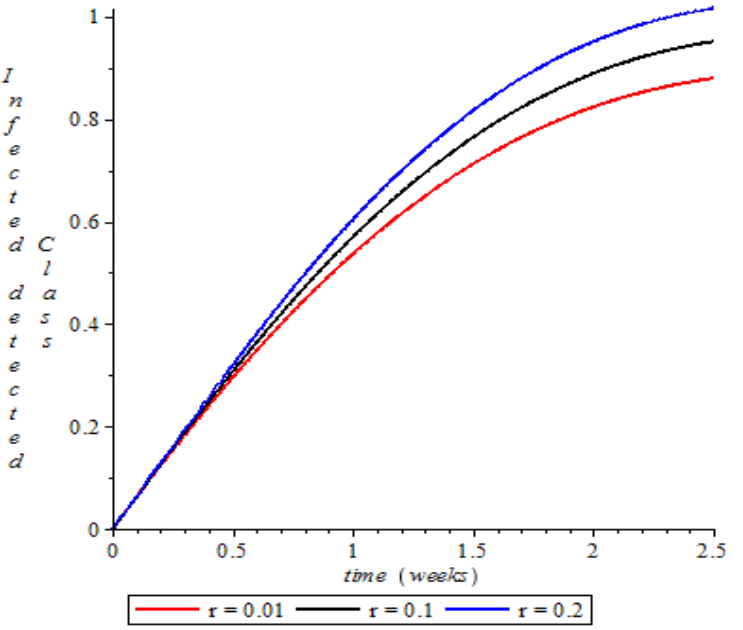

Figure 4(b) Graph of detection rate of infected undetected individual on infected detected class

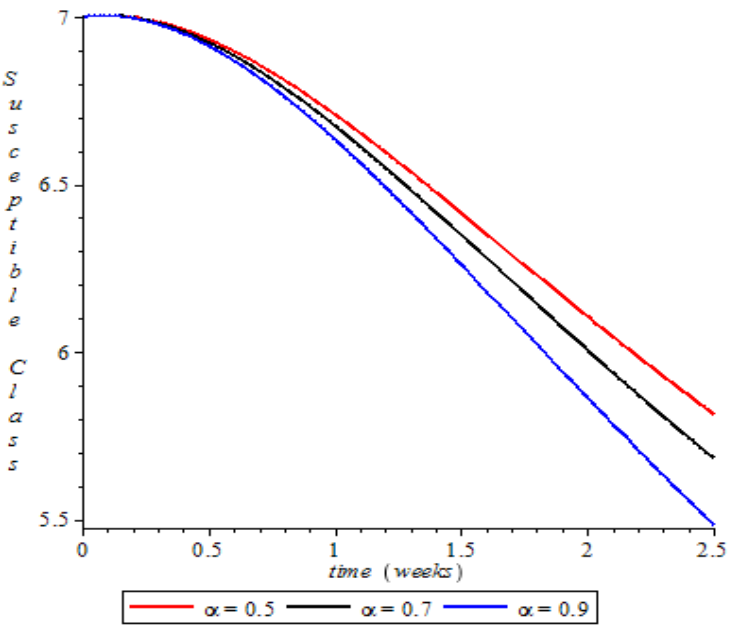

Figure 5(a) Graph of endogenous reactivation rate on susceptible class

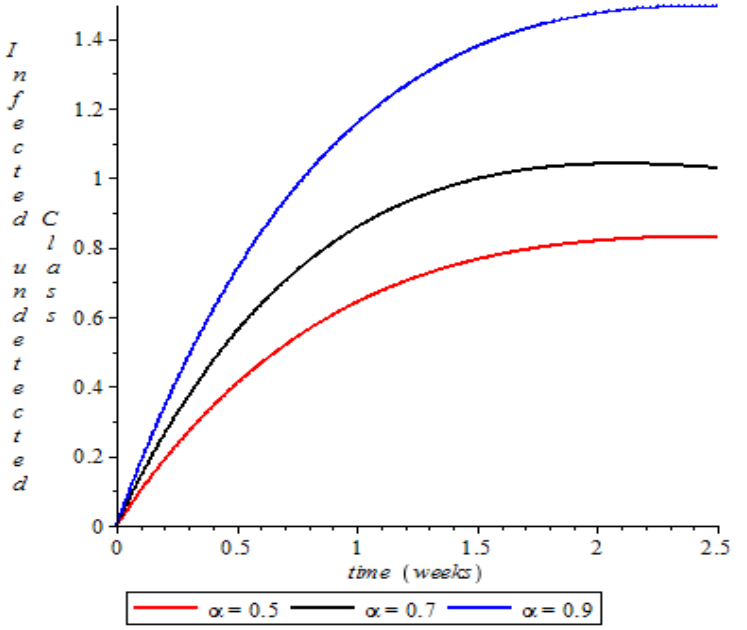

Figure 5(b) Graph of endogenous reactivation rate on infected undetected class

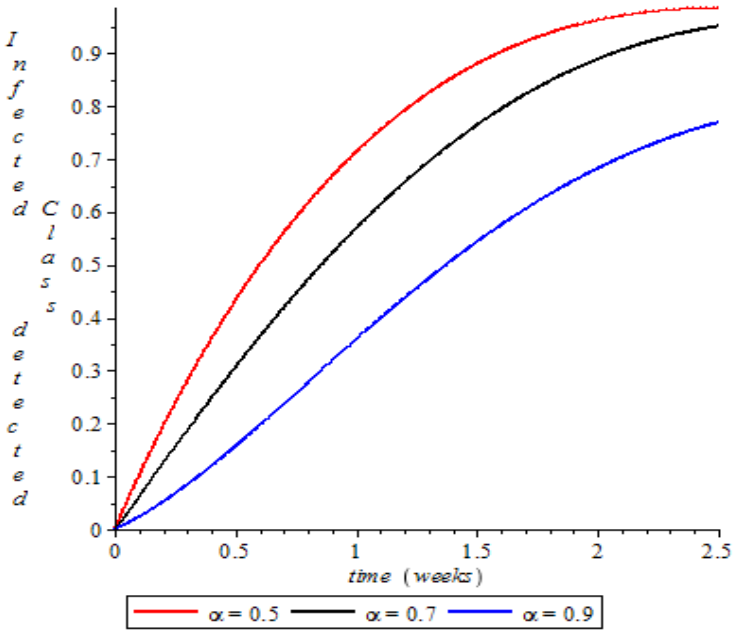

Figure 5(c) Graph of endogenous reactivation rate on infected undetected class

7. Results and Discussion

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 $\tau_1, \beta, \theta, \varepsilon, \alpha, r, \tau_2,$ 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 by the public and health workers to keep it loss while increasing those with negative index will result in the reducing the spread of the disease in the population.

The model exhibits forward bifurcation which shows that the disease can be control if all effect is put in place by the public and health workers to force R_0 below unity.

Figures. 2-5 of numerical simulation shows the behavior of some parameters on the dynamical spread of Ebola Virus diseases.

Figure 2a-e, shows the behavior of progression rate of infected individual to infectious individual.

(τ_1): (a) reveals it effect on susceptible individuals (S), as τ_1 increases S decreases with time, which means, it has a inverse effect on it. (b) shows the effect of τ_1 on latently infected individuals (L), as τ_1 increases L decreases with time, also, it has a inverse effect on it. (c) Pointed out the effect of τ_1 on infected undetected individuals (I_u), as τ_1 increases I_u increases with time, on this, it has a direct effect on it. (d) depicted the effect of $\tau_1$on infected detected individuals (I_d), as τ_1 increases I_d increases with time, also on this, it has a direct effect on it.

Figure 3a-c, reveals the effects of slow progressor (ε): (a) depicted the effect of ε on latently infected individuals (L), as ε increases L decreases with time, this reduces the burden of (L). (b) shows the effect of ε on infected undetected individuals (I_u), as ε increases I_u increases with time, this increases the burden of (I_u). (c) pointed out the effect of ε on infected detected individuals (I_d), as ε increases I_d increases with time this increases the burden of (I_d).

Figure 4a-b, pointed out the effects of detection rate of infected undetected individual (r): (a) the effect of r on infected undetected individuals (I_u), as r increases I_u decreases with time, this decreases the burden of (I_u). (b) the effect of r on infected detected individuals (I_d), as r increases I_d increases with time, this increases the burden of (I_u).

Figure 5a-c, shows the effects of endogenous reactivation rate on the population (α): (a) reveals it effect on susceptible individuals (S), as α increases S decreases with time, on this, it has an inverse effect on it. (b) shows the effect of α on latently infected individuals (L), as α increases L decreases with time, also on this, it has an inverse effect on it. (c) pointed out the effect of α on infected undetected individuals (I_u), as α decreases I_u increases with time, also on this, it has an inverse effect on it.

8.0 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.

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