Global Scientific JOURNALS

GSJ: Volume 11, Issue 7, July 2023, Online: ISSN 2320-9186

www.globalscientificjournal.com

Stability Analysis of Ebola Virus Epidemic with Treatment in the Presence of Two Infectious Compartment

∗[1]Abdullahi M. Auwal, [2]Luka Joshua,[3]Salisu Lukunti and [4]Kalabu S. Ahmad

[1,2,3, 4]Mathematics and statistics, Federal Polytechnics Bauchi

auwal2gga@yahoo.com

Abstract

In this paper, we analyzed the Ebola virus endemic model with treatment in the presence of two infectious or infective compartments, the model allows for some infected individuals and infectious deceased individual to move from treatment compartment to recovery compartment. We prove that the propose mathematical model is biological and meaningfully well-posed. We also compute the basic reproduction number via next generation method. Stability analysis of the disease-free equilibrium, endemic equilibrium and global stability of Ebola virus -free equilibrium has being establishes. Furthermore, the model analysis shows that the disease-free steady state is locally stable when the threshold parameter reproduction number is less than unity.

Keywords: Ebola virus, mathematical model, basic reproduction number, stability analysis, Control Parameters.

Introduction

The first recorded Ebola outbreak dates to 1976 in a remote village Zaire [1, 2], and its name is from the Ebola River close to the village [3]. Since then, 24 Ebola outbreaks have been recorded in different African countries [4, 5]. It is believed that Ebola virus originated from wild animals, which transmit the pathogen to humans [6–8]. The 2014-15 Ebola outbreak in West Africa presents a serious threat to the public health worldwide [9] and is the largest and longest since the first identification of the disease [10], Since then, many other countries were affected, including Nigeria, Senegal, United States, Spain, Mali, United Kingdom and Italy [10]. Ebola virus disease (EVD), also known as Ebola hemorrhagic fever, is a potentially severe illness with high case fatality rate in human [4,11].

The Ebola virus is transmitted among humans through close contact with bodily fluids of infected ill and dead persons, including blood, secretions, semen [12], breast milk [13]. Symptoms of Ebola infection start from 3 to 21 days and commonly include, fever, fatigue, loss of appetite, vomiting, diarrhea, headache, as well as specific hemorrhagic symptoms, unexplained bleeding at different organs [14]. Ebola transmission, unlike most other infectious diseases, contains three major components, including their community, hospital and post-death transmission [15–18]. Transmission among people in community is the major component in Ebola outbreaks. Absent awareness of the disease, Ebola cases grow exponentially without any prevention and intervention [10]. Most of time, people are infected by Ebola virus without accurate diagnosis, or are wrongly diagnosed as other prevalent infectious disease,

Many healthcare workers, visitors and other non-Ebola patients are at the risk of infected by Ebola virus in hospital as reported by [4], this affectionate help in triggering the obstacles to mitigate the spread of the disease.

Thus, the post-death transmission of the Ebola virus from the deceased who has not yet been buried is essential. Because of this, if the victim of the Ebola virus sickness is not properly buried, the infectious viruses are still present in their bodily fluids and can cause the disease to spread. Ancient burial customs that promote the dissemination include washing, touching, and kissing the body [17]. Organization for World Health (WHO) considerations The rapid and covert spread of EVD was facilitated by high population mobility across permeable borders, cultural beliefs, community resistance, health worker strikes, and messages encouraging hopelessness [10].

Mathematical model and simulation are practical essential tool that helps us to improve our understanding of the real-world problem. It can help to determine the characteristics and magnitude of epidemic disease transmission, to predict its outbreak and to see which parameters are more influential in the dynamics of the disease. Over the decade, mathematical models have become vital tools in studying the dynamics of diseases in a given population. The recent development of the use of mathematical models has covered many fields, using different method, for examples of these studies on Ebola virus see [2–12], and the references therein. Although the aforementioned model above was based on bilinear incident rate, and they did not take cognizant of deceased compartment.

According to Weitz *et al.* [19], the number of Ebola cases is understated if the deceased's role in EVD infection is ignored. Since then, numerous mathematical models of Ebola virus diseases that included deceased compartments have been created and examined; for examples, see [14,17-19]. These models were created to provide answers to particular queries in an effort to advance our understanding of the epidemiology of the disease under investigation. More particularly, Chowell *et al.* [31] look into how behavior modification affects EVD transmission when there is a linear force of infection. The effectiveness of social mobilization in containing the Ebola virus in Lofa County, Liberia, was researched by Fast *et al.* [21]. The Evidence of behavior change during an Ebola virus disease outbreak, Sierra Leone, is examined by Jalloh *et al.* [23]. The effect of prevention measures and behavioral modifications on the eradication of Ebola from Lofa County, Liberia, is examined by Funk *et al.* [24]. According to Sylvie *et al.* [22], the new incidence function (new exponential nonlinear incidence function), which lessens the spread of disease as a result of human behavior, can capture the effectiveness and speed of behavior change. The steady states of the model were established by suitable Lyapunov functions along with the global stability and equilibrium points.

Hence, In the absence or limited access to pharmaceutical interventions for EVD such as vaccines and treatment, behavioral changes remain one of the best choices of control strategy to reduce the transmission rate of infectious disease. However, the best chance of stopping the disease's transmission lies in early treatment and behavioral changes, particularly in societies where washing or touching deceased, kissing the dead, and eating bush meat are commonplace [17,25]. Thus, this paper extends the model by Sylvie *et al.* [22] with aim to engross on transmission dynamics of Ebola virus diseases with assessment of treatment in the presence of two infectious compartments, concerning the progression of the infection in the human population. The efficacy of treatment compartment may give us a better understanding of the epidemiological structure of Ebola virus, which may evaluate the level of control in the behavior of the affected population as a result of contracting the Ebola virus diseases through corpse or death infectious human.

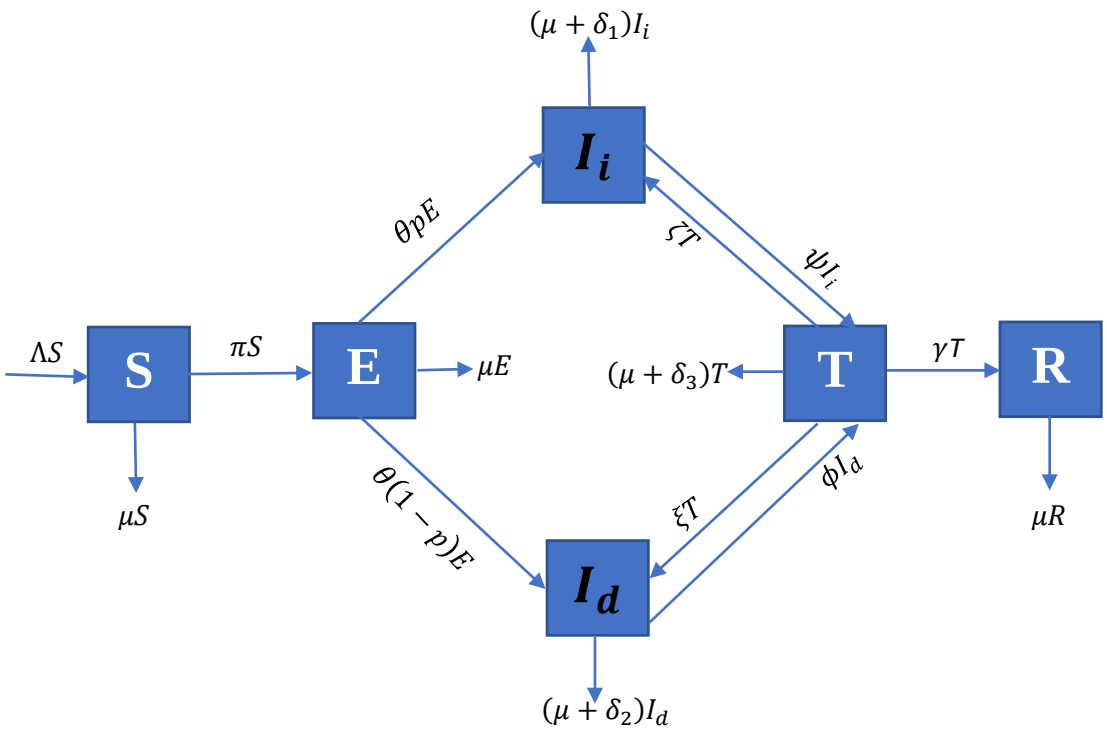

Figure 1. the pictorial diagrammatical representation of the flow chart model system

Description of the state variables and the parameters of the flow chart model

Symbol of the Variable	Description
$S(t)$	Susceptible population
$E(t)$	Exposed population
$I_i(t)$	Infected population
$I_d(t)$	Infectious deceased population (individual contacted the disease through corpse or death infectious human)
$T(t)$	Treatment compartment

Table 1. The state variables of the flow chart model system

Symbol of the Parameters	Descriptions
Λ	Rate of recruitment of susceptible population
μ	Natural death rate
γ	Recovery rate of infected and infectious deceased population
$\delta_1.\delta_2$ and δ_3	Disease induced death rates of individuals in $I_i(t)$, $I_d(t)$ and $T(t)$ respectively
ψ and ϕ	Progression rates at which infected and infectious deceased individuals move to treatment class, (Treatment rate).
$\zeta$and ξ	Rate at which treated individuals move to $I_i(t)$ and $I_d(t)$ compartment respectively, (Rate of treatment failure).
$p\epsilon(0,1)$	Proportion of new exposed individual that become symptomatically infected
θ	Rate at which an exposed individual becomes infectious
ω	Transmission rates due to contact with infectious deceased population (I_d) relative to infectious human corpse yet to be buried
β_1	the contact rate between the susceptible and the infected
β_2	the contact rate between the susceptible and the infectious deceased population

Table 2. The parameters of the flow chart model system

The model System description and formulation

A mathematical model of Ebola virus by in cooperating infectious deceased compartment with treatment is introduced. The total population $N(t)$ is divided into six compartments, namely, $S(t)$ represents the number of susceptible individuals, $E(t)$ represents the number of exposed individuals in the stage of Ebola infection, $I_i(t)$ represents the number of individuals with full-blown Ebola virus, $I_d(t)$ represents the number of individuals that contracted the Ebola virus diseases through corpse or death infectious human, $T(t)$ represents the number of individuals

being treated; $R(t)$ represents the number of individuals who have recovered. A susceptible individual may get infected through contacting with infectious individuals or infectious deceased population. Although the cured of Ebola virus is not realistic or guaranteed, the treatment is significantly important. Hence, in this paper, we extend the model proposed by Sylvie *et al.* [22] in the following sense:

i. Incorporating exposed and infectious deceased compartments.
ii. Incorporating treatment as control measure.
iii. A nonlinear force of infection described by Ricker function was used in [22], while we used Standard incidence rate.

Thus, the proposed extended model is formulated based on the following assumption.

(a) Homogeneous mixing of members of the population under consideration with equal chances of transmitting the virus.
(b) the Probability of susceptible individuals to be becomes infectious in the population under consideration by the proportion of $(1-p)$, with the transmission rate β and $0 \leq p \leq 1$.
(c) Successful treatment of individuals to becomes recovered, does not guarantee permanent immunity.

Hence, the total population $N(t)$ is given by: $N(t) = S(t) + E(t) + I_i(t) + I_d(t) + T(t) + R(t)$, and the pictorial diagrammatical representation of the model is shown in Fig. 1. According to Fig. 1, we have following model equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \pi S - \mu S \\
\frac{dE}{dt} &= \pi S - \theta E - \mu E \\
\frac{dI_i}{dt} &= \theta p E + \zeta T - \psi I_i - (\mu + \delta_1) I_i \\
\frac{dI_d}{dt} &= \theta(1-p)E + \xi T - \phi I_d - (\mu + \delta_2) I_d \\
\frac{dT}{dt} &= \psi I_i + \phi I_d - \zeta T - \xi T - \gamma T - (\mu + \delta_3) T \\
\frac{dR}{dt} &= \gamma T - \mu R
\end{aligned} \tag{1}$$

With $\pi = \frac{\beta_1 I_i + \beta_2 \omega I_d}{N}$

We assume that all parameters in the model are constants. Where Λ is recruitment rate. μ is the natural death rate; $\beta_1 and\ \beta_2$ are the contact rate between the susceptible and the infected or infectious deceased individuals respectively, ω indicate the transmission rates due to contact with infectious deceased population (I_d) relative to infectious human corpse yet to be buried. $\delta_1 . \delta_2$ and $\delta_3$are disease induced death rates of individuals in $I_i(t)$, $I_d(t)$ and $T(t)$ in compartments respectively. $\psi\ and\ \phi$ Progression rates at which infected and infectious deceased individuals move to treatment class, (Treatment rate). while $\zeta$and ξ are the Rate at which treated individuals move to $I_i(t)$ and $I_d(t)$ compartment respectively, (Rate of treatment failure).$p\epsilon(0,1)$ Proportion of new exposed individual that become symptomatically infected, the rate at which an exposed individual becomes infectious is given as θ.where γ stand as the recovery rate of infected and infectious deceased population respectively.

Analysis of the Model

Positivity and Boundedness

To ensure that the system of differential equations in (1), is mathematically well defined and biologically meaningful, we prove the positivity and boundedness of the solutions of our model as follows

Theorem1.

Let the initial coditions $S(0) > 0, E(0) > 0, I_i(0) > 0,\ I_d(0) > 0, T(0) > 0, R(0) > 0$, *then the solution of* $S(t), E(t), I_i(t), I_d(t), T(t), R(t)$ *of the model system* (1) are positive for all $t \geq 0$.

Proof. Suppose $S(t)$ is not positive, then there exists a first time, say $t^* > 0$, such that $S(t) > 0$ For all $t \in [0, t^*)$ and $S(t^*) = 0$. By inspection of the equation of $E(t)$, we have that

$$\frac{dE}{dt} \geq -(\theta + \mu +)E(t), for\ \ t \in [0, t^*),$$

Hence, it follows that, $E > 0\ for\ t \in [0, t^*)$.

Thus, it is clear from the first equation of model system (1) that

$$\frac{dS}{dt} \geq -(\pi + \mu)S(t), for\ t \in [0, t^*).$$

It follows that $S(t^*) > 0$ which contradicts $S(t^*) = 0$. therefore, $S(t)$ is positive. Using similar approach as that for $S(t)$,it is easy to show that $E(t) > 0, I_i(0) > 0,\ I_d(0) > 0, T(t) > 0,$ $R(t) > 0$.Hence the proof.

Invariant Region

In order to retain the biological feasible region of the model system (1) we will be analyzed in a biologically feasible region as follows. Now let us consider the biologically feasible region consisting of $\Omega = \Omega \in \Re_+^6$

$\Omega = \{(S, E, I_i, I_d, T, R)\ \in \Re_+^6 : N \leq \frac{\Lambda}{\mu}\ \}$

It can be shown that the set Ω is a positively invariant set and global attractor of this system. This implies any phase trajectory initiated anywhere in the nonnegative region $\Re_+^6$ enters the feasible region Ω and remains in thereafter.

Lemma 1. The biological feasible region $\Omega = \Omega \subset \Re_+^6$ of the Ebola virus model (1) is positively invariant with nonnegative initial conditions in $\Re_+^6$.

Proof. The following steps are followed to establish the positive invariance of Ω (i.e., solutions in Ω remain in Ω for all $t > 0$). The rate of change of the total populations N respectively, are obtained by adding the respective components of model (1) which result to

$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - \{\mu E(t) + -(\mu + \delta_1)I_i + (\mu + \delta_2)I_d + (\mu + \delta_3)T(t) + \mu R(t)\}$ so that,

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t) \qquad (2)$$

Hence, $N(t) \leq \mu N(0)e^{\mu t}\ + \frac{\Lambda}{\mu}(1 - e^{-\mu t})$. In particular, $N(t) \leq \frac{\Lambda}{\mu}$ if the total population at the initial instant of time, $N(0) \leq \frac{\Lambda}{\mu}$. So, the region Ω is positively invariant. Thus, it is consequently adequate to consider the dynamics of Ebola virus governed by model (1) in the

biological feasible region Ω , where the model is considered to be epidemiologically and mathematically well posed [26].

Existence and Stability of Ebola virus free equilibrium (LFFE)

The Ebola virus free equilibrium of model (1) is obtained at the steady-state solution in the absence of Lassa fever infection, there in by setting the right-hand side of equation (1) equal to zero, such that $S^* = E^* = I_i^* = I_d^* = T^* = R^* = 0$ and solve it simultaneously we get the disease-free equilibrium state denoted by $\mathcal{E}_0$ and is given by

$$\mathcal{E}_0 = (S^*, E^*, I_i^*, I_d^*, T^*, R^*) = \left(\frac{\Lambda}{\mu}, 0,0,0,0,0\right) \quad (3)$$

Basic reproduction number

By considering the most relevant equations in the model system (1) say, $E(t),\ I_i(t),\ I_d(t)\ and\ T(t)$ thus, the gain (the new infection matrices F) and losses (the transfer or transition matrices V) expressions associated with the model (1) using next generation matrix method [27, 28, 29], it follows that the basic reproduction number of model system (1) is computed as

$$F = \begin{pmatrix} 0 & \beta_1 & \beta_2\omega & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_r & 0 \end{pmatrix}, \quad V = \begin{pmatrix} Q_1 & 0 & 0 & 0 \\ -\theta\rho & Q_2 & 0 & -\zeta \\ -K & 0 & Q_3 & 0 \\ 0 & -\psi & -\phi & Q_4 \end{pmatrix}$$

Where $K = \theta(1-\rho), Q_1 = \theta + \mu, Q_2 = \psi + \mu + \delta_1, Q_3 = \phi + \mu + \delta_2 and\ Q_4 = \zeta + \xi + \gamma + \mu + \delta_3$

Thus, the spectral radius of the next-generation FV^{-1},is the basic reproduction $\mathcal{R}_0$ of the model system (1) it follows then that the associated reproduction number denoted by $\mathcal{R}_0$, is given by

$$\mathcal{R}_0 = \frac{K\omega\psi\zeta\beta_2 - K\omega\beta_2 Q_2 Q_4 + \phi\rho\theta\xi\beta_1 - \rho\theta Q_3 Q_4 \beta_1 - K\phi\zeta\beta_1 - \omega\psi\rho\theta\xi\beta_2}{Q_1(\phi\xi Q_2 + \psi\zeta Q_3 - Q_2 Q_3 Q_4)} \quad (4)$$

The basic reproduction $\mathcal{R}_0$ measures the contribution of treatment in managing the Ebola virus risk caused by a lot of time wasted before the patient actually presents for treatment, and the prognosis gets very bad if treatment is not commenced within six days from the onset of symptoms, we can easily observe that the upswing or rise in any of the threshold quantity will directly boom or rising the risk of Ebola virus in the population.

Stability Analysis

Lemma 2: The disease – free equilibrium (DFE) of the Ebola virus model system (1), given by $\mathcal{E}_0$, is locally asymptotically stable if $\mathcal{R}_0 < 1$ and ε_0 is unstable if $\mathcal{R}_0 > 1$.

Proof. Using linearization method, the Jacobian matrix associated with the Ebola virus model at the DFE, $\mathcal{E}_0 = (S^*, E^*, I_i^*, I_d^*, T^*, R^*) = \left(\frac{\Lambda}{\mu}, 0,0,0,0,0\right)$ is obtain as

$$J(\varepsilon_0) = \begin{pmatrix} -Q_1 & 0 & 0 & 0 & 0 & 0 \\ \pi & -Q_2 & 0 & 0 & 0 & 0 \\ 0 & \theta\rho & -Q_3 & 0 & \zeta & 0 \\ 0 & \theta(1-\rho) & 0 & -Q_4 & \xi & 0 \\ 0 & 0 & \psi & \phi & -Q_5 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu \end{pmatrix} \quad (5)$$

$Q_1 = (\pi + \mu), Q_2 = (\theta + \mu), Q_3 = (\psi + \mu + \delta_1), Q_4 = (\phi + \mu + \delta_2) \quad and\ Q_5 = (\zeta + \xi + \gamma + \mu + \delta_3)$ (6)

Then the eigenvalues can be determined by solving the characteristic equation $|J - \lambda I| = 0$, evaluating the eigenvalues of the Jacobian matrix J we found that the following first three eigenvalues say$-(\pi + \mu), -(\theta + \mu)$ and $-\mu$ are negative.

Now let us consider sub matrix J_1 bellow

$$J_1 = \begin{pmatrix} -Q_3 & 0 & \zeta \\ 0 & -Q_4 & \xi \\ \psi & \phi & -Q_5 \end{pmatrix} \quad (7)$$

and the characteristic polynomial is calculated as follows

$$\lambda^3 - (-Q_5 - Q_4 - Q_3)\lambda^2 - (\zeta\psi - Q_3Q_4 - Q_3Q_5 - Q_5Q_4 + \xi\phi)\lambda - \zeta Q_4\psi + Q_5Q_4Q_3 - Q_3\xi\phi \quad (8)$$

Hence, the roots of the equation are $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$,

Where $a_1 = (Q_5 + Q_4 + Q_3)$, $a_2 = (\zeta\psi + Q_3Q_4 + Q_3Q_5 + Q_5Q_4 - \xi\phi)$ and $a_3 = -\zeta Q_4\psi + Q_5Q_4Q_3 - Q_3\xi\phi$.

Using Routh – Hurwitz criteria, ε_0 is locally asymptotically stable when $\mathcal{R}_0 < 1$, or if $a_1 > 0, a_3 > 0$ and $a_1a_2 > a_3$. Thus, the above lemma has been proved accordingly.

Global stability of Ebola virus -free equilibrium

The global stability of the Ebola virus-free equilibrium $\mathcal{E}_0$ of the model system (1), can be examine using the idea in [30]. Now let us, re-write the Ebola virus model (1) in the form

$$\frac{dX}{dt} = F(X, Z), \quad \frac{dZ}{dt} = G(X, Z) \quad and\ G(X, 0) = 0 \quad (9)$$

where $X = (S, R)$ is the uninfected population and $Z = (E, I_i, I_d, T)$ is the infected population with the component of $X \in \mathcal{R}^2$ and $Z \in \mathcal{R}^4$. The Ebola virus-free equilibrium is obtained as

$$\varepsilon_0^* = (X^*, 0) = \left(\frac{\Lambda}{\mu}, 0\right) \quad (10)$$

The point $\varepsilon_0^* = (X^*, 0)$ is said to be globally asymptotically stable, if and only if the following criterion are satisfied.

(C1): $\frac{dX}{dt} = F(X, 0)$, X^* is globally asymptotically stable (GAS),

(C2): $G(X, Z) = QZ - \hat{G}(X, Z)$ with $\hat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$.

Where $Q = B_Z G(X^*, 0)$ is an M-matrix (the off-diagonal elements of B are non-negative) and Ω is the biologically feasible region of the model system. Then the bellow result holds, whenever model system (1) satisfies the above criterion.

Theorem 2. The DFE of the model system (1) at a fixed point $\varepsilon_0^* = (X^*, 0)$ is globally asymptotically stable (GAS), if $\mathcal{R}_0 < 1$, and the criterion (C1) and (C2) are satisfied.

Proof. By considering the model system (1), we have $F(X, Z)\ and\ G(X, Z)$ as

$$\frac{dX}{dt} = F(X, Z) = \begin{pmatrix} \Lambda - Q_1S \\ \gamma T - \mu R \end{pmatrix}, \quad \frac{dZ}{dt} = G(X, Z) = \begin{pmatrix} \pi S - Q_2E \\ \theta pE + \zeta T - Q_3I_i \\ \theta(1-p)E + \xi T - Q_4I_d \\ \psi I_i + \phi I_d - Q_5T \end{pmatrix} \quad (11)$$

Where $Q_1,\ Q_2, Q_3, Q_4\ and\ Q_5$ are same as defined in (6)

We now have the following reduced system:

$$\left.\frac{dX}{dt}\right|_{Z=0} = F(X,Z) = \begin{pmatrix} \Lambda - \mu S \\ 0 \end{pmatrix} \quad (12)$$

From equation (11), we can obviously say that $\varepsilon_0^* = \left(\frac{\Lambda}{\mu}, 0\right)$, is the GAS equilibrium point for the reduced system (12). This is trivia by solving $\frac{dS}{dt} = \Lambda - \mu S$ to have $S(t) = \frac{\Lambda}{\mu} + \left(S(t) - \frac{\Lambda}{\mu}\right) exp^{-\mu t}$, which implies that $S(t) \to \frac{\Lambda}{\mu}$ as $t \to \infty$. Hence, the convergence of solutions is global in the region Ω.

Let, $Q = B_Z G(X^*, 0) = \begin{pmatrix} -Q_3 & 0 & \zeta \\ 0 & -Q_4 & \xi \\ \psi & \phi & -Q_5 \end{pmatrix}$ (13)

Where $Q_3, Q_4 \; and \; Q_5$ are same as defined in (6)

Then, we verify the second criteria (C2):

$$\hat{G}\,(X,Z) = \begin{pmatrix} \widehat{G_1}\,(X,Z) \\ \widehat{G_2}\,(X,Z) \\ \widehat{G_3}\,(X,Z) \\ \widehat{G_4}\,(X,Z) \end{pmatrix} = \begin{pmatrix} (\beta_1 I_i + \beta_2 \omega I_d)\left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Since $S \leq 0$, it is clear that $\hat{G}\,(X,Z) \geq 0$. Thus, the Ebola virus-free with the fixed point $\varepsilon_0^* = (X^*, 0)$ is globally asymptotically stable when $\mathcal{R}_0 < 1$.

Hence, this indicate that regardless of the initial sizes of the sub-populations of the system, is possible to eradicate Ebola virus disease, whenever the reproduction number is less than unity.

Existence and stability of endemic equilibria

Ebola virus endemic equilibrium points of model (1) is obtained at the steady-state solution in the presence Ebola virus infection in the population. there in by setting the right-hand side of equation (1) equal to zero we have

$$\Lambda - Q_1 S = 0$$
$$\lambda S - Q_2 E = 0$$
$$\theta p E + \zeta T - Q_3 I_i = 0 \quad (14)$$
$$\theta(1-p)E + \xi T - Q_4 I_d = 0$$
$$\psi I_i + \phi I_d - Q_5 T = 0$$
$$\gamma T - \mu R = 0$$

With $\pi = \frac{\beta_1 I_i + \beta_2 \omega I_d}{N}$

Where $Q_1,\; Q_2\,, Q_3, Q_4 \; and \; Q_5$ are same as defined in (6)

and solve it simultaneously in terms of the associated form of infection we get the Lassa fever endemic equilibrium points state denoted by $\mathcal{E}_1$ and is given by $\mathcal{E}_1 = (S^{**}, E^{**}, {I_i}^{**}, {I_d}^{**}, T^{**}, R^{**})$. Therefore, the endemic equilibrium points of the model system (1) are

$$S^{**} = \frac{\Lambda}{Q_1}$$

$$E^{**} = \frac{\pi^{**}\Lambda}{Q_2 Q_1}$$

$$I_i{}^{**} = \frac{\theta\,\pi^{**}\Lambda(\phi\rho\xi + \phi\rho\zeta - \rho Q_4 Q_5 - \phi\zeta)}{(\phi\xi Q_3 + \psi\zeta Q_4 - Q_3 Q_4 Q_5)Q_1 Q_2}$$

$$I_d{}^{**} = \frac{\theta\,\pi^{**}\Lambda(-\psi\rho\xi - \psi\rho\zeta + \rho Q_3 Q_5 + \psi\zeta - Q_3 Q_5)}{(\phi\xi Q_3 + \psi\zeta Q_4 - Q_3 Q_4 Q_5)Q_1 Q_2}$$

$$T^{**} = \frac{\theta\,\pi^{**}\Lambda(\phi\rho Q_3 - \psi\rho Q_4 - \phi Q_3)}{(\phi\xi Q_3 + \psi\zeta Q_4 - Q_3 Q_4 Q_5)Q_1 Q_2}$$

$$R^{**} = \frac{\gamma\theta\,\pi^{**}\Lambda(\phi\rho Q_3 - \psi\rho Q_4 - \phi Q_3)}{\mu(\phi\xi Q_3 + \psi\zeta Q_4 - Q_3 Q_4 Q_5)Q_1 Q_2}$$

With $\pi^{**} = \frac{\beta_1 I_i{}^{**} + \beta_2 \omega I_d{}^{**}}{N^{**}}$ and $N^{**} = S^{**} + E^{**} + I_i{}^{**} + I_d{}^{**} + T^{**} + R^{**}$ (16)

We can now rewrite (16) in the following form

$$S^{**} + E^{**} + \left(1 - \frac{\beta_1}{\pi^{**}}\right) I_i{}^{**} + \left(1 - \frac{\beta_2 \omega}{\pi^{**}}\right) I_d{}^{**} + T^{**} + R^{**} = 0 \quad (17)$$

Subtituting equation (15) into (17) for π^{**} we obtain

$$\frac{\Lambda}{Q_1} + \frac{\pi^{**}\Lambda}{Q_2 Q_1} + \frac{\left(1 - \frac{\beta_1}{\pi^{**}}\right)\theta\,\pi^{**}\Lambda(\zeta\phi\rho + \phi\rho\xi - \rho Q_4 Q_5 - \zeta\phi)}{(\zeta\psi Q_4 + \phi\xi Q_3 - Q_3 Q_4 Q_5)Q_1 Q_2} + \frac{\left(1 - \frac{\beta_2 \omega}{\pi^{**}}\right)\theta\,\pi^{**}\Lambda(-\zeta\psi\rho - \psi\rho\xi + \rho Q_3 Q_5 + \zeta\psi - Q_3 Q_5)}{(\zeta\psi Q_4 + \phi\xi Q_3 - Q_3 Q_4 Q_5)Q_1 Q_2} + \frac{\theta\,\pi^{**}\Lambda(\phi\rho Q_3 - \psi\rho Q_4 - \phi Q_3)}{(\zeta\psi Q_4 + \phi\xi Q_3 - Q_3 Q_4 Q_5)Q_1 Q_2} + \frac{\gamma\theta\,\pi^{**}\Lambda(\phi\rho Q_3 - \psi\rho Q_4 - \phi Q_3)}{\mu(\zeta\psi Q_4 + \phi\xi Q_3 - Q_3 Q_4 Q_5)Q_1 Q_2} = 0 \quad (18)$$

Hence, the model system (1) has endemic equilibria correspond to positive solutions of the equation (18), which shows that there is at least one endemic equilibrium whenever $R_0 > 1$.

Conclusion

In this paper, a non-linear deterministic mathematical model of Ebola virus epidemic with treatment in present of two infectious compartments was formulated and analyzed. We obtain the basic reproduction number $\mathcal{R}_0$ via next generation matrix method, we have shown that if $\mathcal{R}_0$ is greater than one, means the disease is spreading and also the number of infected individual increases as well. The disease-free equilibrium points and the endemic equilibrium point has been established. We have proven that the disease – free equilibrium (DFE) of the Ebola virus model system is locally asymptotically stable (LAS) if $\mathcal{R}_0 < 1$ and ε_0 is unstable if $\mathcal{R}_0 > 1$, and globally-asymptotically stable (GAS) whenever all new recruits are effectively or properly treated ,this indicate that the treatment rate $\psi\ and\ \phi$ should be greater than one, and the treatment failure rate $\zeta$and ξ should be equal to zero by Keeping the remaining parameters constant.

This show that the early treatment of Lassa fever is necessary and meaningful also It will significantly lessen the disease's negative consequences on the populace. Finally, the public health implication of the aforementioned finding is that if the Ebola virus disease control approach can reduce the reproduction rate to below the value of one, then Ebola virus can be eradicated from the community.

References

[9] "WHO Statement on the 1st meeting of the IHR Emergency Committee on the 2014 Ebola outbreak in West Africa," http://www.who.int/mediacentre/news/ statements/2014/ebola-20140808/en/, [Online; accessed 2016-02-03].

[10] "One year into the Ebola epidemic: a deadly, tenacious and unforgiving virus," http://www.who.int/csr/disease/ebola/one-year-report/introduction/en/, [Online; accessed 2016-02-03].

[4] "WHO | Ebola virus disease," http://www.who.int/mediacentre/factsheets/ fs103/en/, [Online; accessed 2016-04-03].

[11] S. K. Singh and D. Ruzek, Viral Hemorrhagic Fevers. CRC Press, Jul. 2013.

[1] "Ebola haemorrhagic fever in Zaire, 1976," Bull World Health Organ, vol. 56, no. 2, pp. 271–293, 1978.

[2] E. T. W. Bowen, G. Lloyd, W. J. Harris, G. S. Platt, A. Baskerville, and E. E. Vella, "Originally published as Volume 1, Issue 8011viral HÆMORRHAGIC FEVER IN SOUTHERN SUDAN AND NORTHERN ZAIRE," The Lancet, vol. 309, no. 8011, pp. 571–573, Mar. 1977.

[3] H. Feldmann and T. W. Geisbert, "Ebola haemorrhagic fever," Lancet, vol. 377, no. 9768, pp. 849–862, Mar. 2011.

[5] M. D. Van Kerkhove, A. I. Bento, H. L. Mills, N. M. Ferguson, and C. A. Donnelly, "A review of epidemiological parameters from Ebola outbreaks to inform early public health decision-making," Scientific Data, vol. 2, p. 150019, May 2015.

[6] G. Chowell and H. Nishiura, "Transmission dynamics and control of Ebola virus disease (EVD): a review," BMC medicine, vol. 12, no. 1, p. 196, 2014.

[7] E. M. Leroy, B. Kumulungui, X. Pourrut, P. Rouquet, A. Hassanin, P. Yaba, A. D´elicat, J. T. Paweska, J.-P. Gonzalez, and R. Swanepoel, "Fruit bats as reservoirs of Ebola virus," Nature, vol. 438, no. 7068, pp. 575–576, Dec. 2005.

[8] X. Pourrut, A. D´elicat, P. E. Rollin, T. G. Ksiazek, J.-P. Gonzalez, and E. M. Leroy, "Spatial and temporal patterns of Zaire ebolavirus antibody prevalence in the possible reservoir bat species," J. Infect. Dis., vol. 196 Suppl 2, pp. S176–183, Nov. 2007.

[12] "Preliminary study finds that Ebola virus fragments can persist in the semen of some survivors for at least nine months | CDC Online Newsroom | CDC," http: //www.cdc.gov/media/releases/2015/p1014-ebola-virus.html, [Online; accessed 2016-04-12]. 121

[13] "Recommendations for Breastfeeding/Infant Feeding in the Context of Ebola | Ebola Hemorrhagic Fever | CDC," http://www.cdc.gov/vhf/ebola/hcp/ recommendations-breastfeeding-infant-feeding-ebola.html, [Online; accessed 2016-04-12].

[14] WHO Ebola Response Team, "Ebola Virus Disease in West Africa — The First 9 Months of the Epidemic and Forward Projections," New England Journal of Medicine, vol. 371, no. 16, pp. 1481–1495, Oct. 2014.

[15] S. F. Dowell, R. Mukunu, T. G. Ksiazek, A. S. Khan, P. E. Rollin, and C. J. Peters, "Transmission of Ebola hemorrhagic fever: a study of risk factors in family members, Kikwit, Democratic Republic of the Congo, 1995. Commission de Lutte contre les Epid´emies `a Kikwit," J. Infect. Dis., vol. 179 Suppl 1, pp. S87–91, Feb. 1999.

[16] T. H. Roels, A. S. Bloom, J. Buffington, G. L. Muhungu, W. R. Mac Kenzie, A. S. Khan, R. Ndambi, D. L. Noah, H. R. Rolka, C. J. Peters, and T. G. Ksiazek, "Ebola hemorrhagic

fever, Kikwit, Democratic Republic of the Congo, 1995: risk factors for patients without a reported exposure," J. Infect. Dis., vol. 179 Suppl 1, pp. S92–97, Feb. 1999.

[17] B. S. Hewlett and R. P. Amola, "Cultural Contexts of Ebola in Northern Uganda," Emerg Infect Dis, vol. 9, no. 10, pp. 1242–1248, Oct. 2003.

[18] J. Legrand, R. F. Grais, P. Y. Boelle, A. J. Valleron, and A. Flahault, "Understanding the dynamics of Ebola epidemics," Epidemiol. Infect., vol. 135, no. 4, pp. 610–621, May 2007.

[19] S. Weitz and J. Dushoff, "Modeling post-death transmission of Ebola: challenges for inference and opportunities for control," Scientific Reports, vol. 5, no. 1, 2015.

[20] G. Chowell and J. M. Hyman, Mathematical and Statistical Modeling for Emerging and Re-emerging Infectious Diseases, Springer International Publishing, Switzerland, 2016.

[21] S. M. Fast, S. Mekaru, J. S. Brownstein, T. A. Postlethwaite, and N. Markuzon, "The role of social mobilization in controlling Ebola virus in Lofa County, Liberia," PLoS Currents, vol. 7, 2015.

[22] Sylvie Diane Djiomba Njankou and Farai Nyabadza (2022) . Modelling the Role of Human Behaviour in Ebola Virus Disease (EVD) Transmission Dynamics. Hindawi Computational and Mathematical Methods in Medicine Volume 2022, Article ID 4150043, 11 pages https://doi.org/10.1155/2022/4150043.

[23] M. F. Jalloh, P. Sengeh, R. E. Bunnell et al., "Evidence of behaviour change during an Ebola virus disease outbreak, Sierra Leone," Bulletin of the World Health Organization, vol. 98, no. 5, pp. 330–340B, 2020.

[24] S. Funk, I. Ciglenecki, A. Tiffany et al., "The impact of control strategies and behavioural changes on the elimination of Ebola from Lofa County, Liberia," Philosophical Transactions of the Royal Society B: Biological Sciences, vol. 372, 2017.

[25] K. A. Alexander, C. E. Sanderson, M. Marathe et al., "What factors might have led to the emergence of Ebola in West Africa?," PLoS Neglected Tropical Diseases, vol. 9, no. 6, p. e0003652, 2015.

[26]Lakshmikantham, V., Leela, S., & Martynyuk, A.A. (1989). Stability Analysis of Nonlinear Systems. *Springer, Berlin*.

[27] Diekmann O. and Heesterbeek J. A. P. (2000), Mathematical epidemiology of infectious diseases: model building, analysis and interpretation, Chichester-Oxford Academic

[28] Diekmann O., Heesterbeek J. A. P. and Metz J. A. J. (1990), On the defenition and the computation of the basic reproduction ratio R0 in models for infectious diseases, Journal of Mathematical Biology, 35, 503-522.

[29] Van den Driessche P. and Watmough J. (2002), Reproduction numbers and sub threshold endemic equilibria for compartmental models of disease transmission, Mathematical Biosciences, 180, 29-48.

[30] Castillo-Chavez, C., Blower, S., Van den Driessche, P., Kirschner, D., Yakubu, A.-A.: Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory, vol. 126. Springer, Berlin (2002)

[31] G. Chowell, N. W. Hengartnerb, C. Castillo-Chavez, P. W. Fenimorea, and J. M. Hyman, "The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda," Journal of Theoretical Biology, vol. 229, no. 1, pp. 119–126, 2004.