A Mathematical Model For The Dynamical Behavior Of Ebola Virus Transmission In Human-Bat Population: Implication Of Immediate Discharge Of Recovered Individuals

Joshua Agbomola (agbomolajoshua@gmail.com)
Tai Solarin University of Education

Adedapo Loyinmi
Tai Solarin University of Education

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A Mathematical Model for the Dynamical Behavior of Ebola virus Transmission in Human-Bat Population: Implication of immediate discharge of Recovered Individuals

Joshua Oluwasegun Agbomola¹, Adedapo Chris Loyinmi²

¹Department of Mathematics, Tai Solarin University of Education, Ijagun, Ogun State, Nigeria

Corresponding Email Address: agbomolajoshua@gmail.com & loyinmiac@tasued.edu.ng (Loyinmi, AC)

²Department of Mathematics, Tai Solarin University of Education, Ijagun. Ogun State. Nigeria

Email Address: loyinmiac@tasued.edu.ng

ORCID: 0000-0002-7637-1591; 0000-0002-6171-4256

Abstract

Ebola virus is zoonotic. Earlier research has paid less attention to vector–host transmission dynamics of the virus. In this study, we, therefore, proposed an unprecedented coupled SEIR-SEI epidemic model of the type $S_b E_h I_h R_{bh} R_{his} - S_b E_h I_b$ which predicts the prevalence and virulence of the Ebola virus from bats to humans. More so, since remnants of the virus can still last for days in the fluidic parts of the body after recovery, the recovery class $R_h$ is subdivided into two ($R_{his}$ & $R_{bhi}$). The sensitivity analysis investigation reveals parameters that have profound effects on the reproduction number. The system is proven to be positive and bounded at all times and the fundamental parameters for invasion, $R_{bh}$ and $R_{hb}$ for both human and bat populations respectively were established to be less than unity. Also, persistence (Endemic) and Disease Free Equilibrium points of the model were ascertained to be asymptotically stable both locally and globally.
Keywords: zoonotic, bifurcation, recovered with and without isolation, persistence equilibrium.
1.0 Introduction

Ebola virus is known to have been transmitted from wild and domestic animals to humans in their respective habitats. It is confirmed to have been spread from animal to human and human to human. Ebola virus disease (EVD) is erstwhile known as Ebola Haemorrhagic fever, rare but perilous, frequently causing fatal illness in humans. The first recorded appearance of the Ebola virus disease was in 1976 in 2 coincided outbreaks, which were in Nzara (South Sudan), and Yambuku (DRC). Then it was said to spring up again in a village close to the Ebola River, which was what brought it denotations. From discovery, there are six species of Ebola virus generated from three virus families called Filoviridae which are Cuevavirus, Marburgvirus, and Ebolavirus. Amongst the six (6), only four (4) of them were traced to have caused Ebola virus disease in the human population. They are Tai Forest virus, Ebola-Zaïre virus, Bundibugyo Ebola virus, and Sudan Ebola virus. There are accounts of the upsurge of the virus in Africa. As at the 2021 approximate estimation of the decimation, it is said to be 50% but the variation of the upsurge from times past falls between 25%-90%. From statistics, it was ascertained that the outbreaks of the Ebola virus through the year 2014-2016 were the largest narrowing it down to the year 1976 when the Ebola virus disease began. It all started in Guinea then stirred across land borders to Liberia, Mali, USA, Spain, Sierra Leone, etc. Through inductive definition, the Ebola virus is said to have been transmitting from fruit bats of the Pteropodidae family which is ascertained as the natural Ebola hosts. This virus enters into the Human population via narrow contact with a fluidic component of the body which could be secretions, blood, organs, etc., of infected animals such as chimpanzees, porcupines, monkeys, gorillas, forest antelope, or fruit bats found sick or succumbed or in the rainforest. Sequel to the available information on the Ebola virus, serious contamination routes abound: Healthcare workers have without numbers been infected while treating patients with either confirmed or suspected cases of the Ebola Virus. This was narrowed to the close association with the patients portraying that the infection control precautions are not intensely taken note of; Burial rites or practices of some ethnic groups or cities which primarily involved the direct contact with the
dead body have also contributed greatly to the transmission of Ebola virus because people remain infectious as long as their blood, tissues or fluidic part of the body contain the virus. An infected pregnant woman who gets recovered from Ebola infection on treatment may still be a carrier of the virus precisely in her breast-milk. Deductively, there would be a risk of transmission to the baby she carries, and if the baby is infected, then there is a high probability of transmission to anyone who carries the baby afterward. Consumption of contaminated bush meat also resulted in the reoccurrence of the virus. EVD incubations period is calculated to be 2-21 days and noticeable symptoms of EVD fever include headache, fatigue, sore throat, vomiting, diarrhea, muscular pain, internal and external bleeding, low white blood cell, rashes, and so on. Bi-Monoclonal antibodies called Inmazeb and Ebanga were approved by the US Food and Drug Administration at the concluding part of the year 2021 for the treatment of the Ebola virus precisely the class of Zaire ebolavirus. Also, Ervebo has been verified to be very effective during the process of vaccination of Zaire ebolavirus. The vaccine was recommended by the Strategic Advisory Group of Experts on immunization as part of a broader set of EVD outbreak response tools. It was later approved by the US Food and Drug Administration in December 2020 coupled with the direction stated by the World Health Organization (WHO). [27]

It is observed that many works done on Ebola Virus Disease (EVD) involve mainly the human populations. In the work of [6], four compartmental variables SEIR for human population was considered and R$_0$ was estimated as 1.34 for Uganda and 1.83 for Congo. Noticeable research was also done by [14] in which the reproduction number R$_0$ for Uganda and Congo was approximately calculated to be 1.33 and 1.35 respectively. A distinctive work was also carried out by [13] wherein one more compartment (Death-D) was added to the existing one and the resulting reproduction number was 2.7 for both the 1995 Congo and 2000 Uganda outbreaks. The work was followed by [20] wherein the statistical data analysis of the 1995 Ebola upsurge in the Democratic Republic of Congo was worked on. The authors implement the Markov Chain Monte Carlos method to appraise the parameters of their SEIRD model and estimated the pre-interference
R₀ at 2.1 and the post-interference R₀ at 0.1339 for the outbreak which occurred in Congo 1995. Perusing the work done by some authors in 2014, they are conclusively similar to the ones that were done in the past [2, 8]. A quantifiable shift was done by [1, 11] wherein demographic dynamics which have been missing from the previous works were considered. Also, the development of meta-population models through internationalization in West Africa was done by [11]. Contamination of the Ebola virus was strictly narrowed to eating contaminated bush meat hunted for food or coming in contact with fruit bats since 1995 as stated by [16], [15], and [5]. And more figuratively, further constraints such as environmental transmission (direct and indirect), persistence, and survival of the virus were added by [5], [21], and [17] which made their proposed model more voluminous. A distinguished model was proposed by [10], where quarantine and non-quarantine compartmental variables were considered and it was proved that R₀ is conditionally less than unity. In [3], a model with five (SIPRD) compartmental variables was considered to answer fundamental questions about if consumption of the contaminated bushmeat, environmental contamination, and some ethnical funeral practices can justify the deluge of transmission of the Ebola virus. The reproduction number was obtained and compared with or without environment-class P and vital dynamic, and is between 1.51-2.7 for $R_0^{SIPRD}$ and 1.02-1.58 for $R_0^{SIRD}$. Considering [22], a distinctive model mixed with hospital isolation and application of medication was formulated. They analyzed their effect on resisting the spread of the Ebola virus. They obtained their R₀ to be 1.754. In [12], a mathematical model for Ebola virus infection in humans with the effectiveness of drug usage was discussed. They described the cellular description of dynamical systems of the Ebola virus incorporating cytotoxic T-lymphocyte cells and the study as well established the reproductive ratio, $R_0<1$. A generalized epizootic model was developed by [28]. In the work, they take into account the environmental contamination and incidence function encapsulating many incidence rates for infectiousness. From [23], a model that added control strategies such as education campaigns, quarantine, safe burial, and treatment was proposed. A differential equation based on the two-patch model was formulated and explained by [18]. In this study, patches were connected with
migration restricted to both susceptible and recovered individuals. Here also, the threshold parameter $R_0$ is less than unity.

Earlier research on Ebola transmission dynamics has been on either the human population or bat population. These limited researches have motivated this research. We have proposed an unprecedented and feasible model to couple bats and humans (as host and vector) to analytically explain the transmission dynamics of the Ebola virus between humans and bats. The flow diagram in figure 1 is the bifurcation description containing eight (8) compartments. Recent research showed that any infected individual that is completely treated and has recovered may still have the virus for some days or months in the fluidic part (semen, eyeball, blood, breast milk, etc.) of the body. This is an important constraint that should be seriously considered to reduce the effect of relapse which will promote the virulence of the Ebola virus. So, to capture this in our proposed model, we, therefore, divide the recovered class into two: Recovered with isolation and Recovered without isolation. By so doing, we have our compartmental variables as $S_h E_h I_h R_{his} R_{hui} - S_b E_b I_b$. The conditions for Persistence (Endemic) and Disease Free Equilibrium were clearly stated.

2.0 Conceptual Model Formulation and Equations
\( \beta \) be effective transmission rate between humans and bat
\( \beta_2 \) be effective transmission rate for susceptible bats and wild bats (Chiroptera)
\( \beta_3 \) be effective transmission rate for susceptible bats and wild bats (Chiroptera)
\( m \) be saturation factor
\( \tau \) be the treatment rate of infectious individual s.
\( \phi_1 \) be the saturated incidence function or agent of infection for humans.
\( \phi_2 \) be the saturated incidence function or agent of infection for bats.
\( \theta_h \) be the transition rate from exposed individual s to infectious.
\( \Lambda_h \) be the recruitment/immigration rate for humans
\( \Lambda_b \) be the recruitment/immigration rate for bats
\( \theta_b \) be the transition rate from exposed bats to infectious bats
\( \mu_h \) be the natural mortality rate in the human's population
\( \mu_b \) be the natural mortality rate in the bat's population
\( \delta \) be the disease-prompted mortality rate
\( S_h \) be the susceptible human population
\( E_h \) be the exposed human population
\( I_h \) be infectious human population
\( R_{his} \) be the recovered human population under isolation
\( R_{hn} \) be the recovered human population not under isolation
\( S_b \) be the susceptible bat's population
\( E_b \) be the exposed bat's population
\( I_b \) be the infective bat's population

Bifurcation analysis and dynamical behavior of an Ebola virus model with a saturated incidence rate

\[
\phi_1 = \frac{\beta_1 I_h}{1 + mI_h} + \frac{\beta_2 I_b}{1 + mI_b}
\]

Where

\[
\phi_2 = \frac{\beta_3 I_b}{1 + mI_b}
\]

Let the probability distribution of those that recovered be 1. That is, \( P(R_{hi} + R_{his}) = 1 \).

Let \( c \) be the probability of just recovering in isolation till the remains of the virus completely leave the fluidic part of their body.
Then \((1 - c)\) be the probability of recovered humans that are not in isolation till the remains of the virus completely leave the fluidic part of their body.

From the Figure 1, we have the following systems of the equation:

For human

\[
\begin{align*}
\frac{dS_h}{dt} &= \lambda_h - \mu_h S_h - \phi_i S_h \\
\frac{dE_h}{dt} &= \phi_i S_h + (\mu_h + \theta_i) E_h \\
\frac{dI_h}{dt} &= \theta_i E_h + \rho R_{hi} - (\mu_h + \delta + c \tau + (1 - c) \tau) I_h \\
\frac{dR_{hi}}{dt} &= c \tau I_h - \mu_h R_{hi} \\
\frac{dR_{hni}}{dt} &= (1 - c) \tau I_h - (\mu_h + \rho) R_{hni}
\end{align*}
\]

(2)

For Bats

\[
\begin{align*}
\frac{dS_b}{dt} &= \lambda_b - (\mu_b + \phi_2) S_b \\
\frac{dE_b}{dt} &= \phi_2 S_b - (\mu_b + \theta_b) E_b \\
\frac{dI_b}{dt} &= \theta_b E_b - \mu_b I_b
\end{align*}
\]

(3)

Where;

\[
\begin{align*}
S_h + E_h + I_h + R_{hi} + R_{hni} &= N_h \\
S_b + E_b + I_b &= N_b \\
S_h(t = 0) &\geq 0, E_h(t = 0) \geq 0, I_h(t = 0) \geq 0, R_{hi}(t = 0) \geq 0, R_{hni}(t = ) \geq 0 \\
S_b(t = 0) &\geq 0, E_b(t = 0) \geq 0, I_b(t = 0) \geq 0
\end{align*}
\]

(4)

\(N_h\) is the human population size and \(N_b\) bat total population size.
3.0  **Positivity and Boundedness of solutions**

Sequel to the human and bat populations, all related constraints used in the model \((2-3)\) must be arbitrarily non-negative to create the dynamic transmission model of Ebola epidemiologically significant. Thus analytic solutions obtained remain positive for all time \(t \geq 0\) and bounded referencing [3] in a feasible region.

\[
\alpha_h = \left( S_h, E_h, I_h, R_{hni}, R_{his} \right) \in \mathbb{R}^5_+, N_h \leq \frac{\Lambda_h}{\mu_h}
\]

\[
\alpha_b = \left( S_b, E_b, I_b \right) \in \mathbb{R}^3_+, N_b \leq \frac{\Lambda_b}{\mu_b}
\]

and

\[
\alpha_f = \left( S_h, E_h, I_h, R_{hni}, R_{his}, S_b, E_b, I_b \right) \in \mathbb{R}^8_+, N_h \leq \frac{\Lambda_h}{\mu_h}, N_b \leq \frac{\Lambda_b}{\mu_b}
\]

### 3.1 Positivity of solution

The solutions \(S_h, E_h, I_h, R_{hni}, R_{his}, S_b, E_b, I_b\) of the system \((2-3)\) are positive for all \(t \geq 0\) with non-negative initial conditions.

**Theorem 1:** Regarding the initial conditions as given in the equation (4). Then the solutions \((S_h, E_h, I_h, R_{hni}, R_{his}, S_b, E_b, I_b)\) of the system \((2-3)\) are positive for all \(t > 0\).

**Proof:**

Let \(\bar{\chi} = \text{supremum}\left\{t > 0 : S_h(t) \geq 0, E_h(t) \geq 0, I_h(t) \geq 0, R_{hni}(t) \geq 0, R_{his}(t) \geq 0, S_b(t) \geq 0, E_b(t) \geq 0, I_b(t) \geq 0\right\}\)

Succinctly, \(\bar{\chi} > 0\).

Assume \(S_h(0) \geq 0\), the first equation in \((2)\) becomes

\[
\frac{dS_h}{dt} = \Lambda_h - (\mu_h + \phi_h)S_h
\]
\[ S_h(t) = \int_0^t e^{\int_0^t (\mu_h + \phi_h) \, dt} \, dt + C \quad \Rightarrow \quad S_h(t) = \sum_h e^{\int_0^t (\mu_h + \phi_h) \, dt} \int_0^t e^{\int_0^t (\mu_h + \phi_h) \, dt} \, dt + S_h(0)e^{\int_0^t (\mu_h + \phi_h) \, dt} \]

Therefore \( S_h(t) = \sum_h e^{\int_0^t (\mu_h + \phi_h) \, dt} \int_0^t e^{\int_0^t (\mu_h + \phi_h) \, dt} \, dt + S_h(0)e^{\int_0^t (\mu_h + \phi_h) \, dt} \)

As \( S_h(0) \geq 0 \), the sum of the non-negative terms \( S_h \) is non-negative.

From first equation in (3),

\[ \frac{dS_h}{dt} = \sum_h - (\mu_h + \phi_2)S_h \]

Using a similar process above with \( S_b(0) \geq 0 \),

\[ S_b(t) = \sum_b e^{\int_0^t (\mu_b + \phi_2) \, dt} \int_0^t e^{\int_0^t (\mu_b + \phi_2) \, dt} \, dt + S_b(0)e^{\int_0^t (\mu_b + \phi_2) \, dt} \]

Comparatively, we can prove that the compartmental variables \( E_h, I_h, R_{hni}, R_{his}, E_b, I_b \) are positive for all \( t > 0 \).

### 3.2 Boundedness of the solutions

**Theorem 2:** The analytic solutions of all systems of equations (2 – 3) are bounded.

**Proof**

The total population for humans is denoted by \( N_h \) while for bat is \( N_b \). From equation 4, i.e.

\[ S_h + E_h + I_h + R_{hni} + R_{his} = N_h \text{ and } S_b + E_b + I_b = N_b \]
$$\frac{dN_h}{dt} = dS_h + dE_h + dI_h + dR_{his} + dR_{his}$$

$$\frac{dN_b}{dt} = dS_b + dE_b + dI_b$$

Using equation (2) – (3)

$$\frac{dN_h}{dt} = \Lambda_h - \mu_b(S_h + E_h + I_h + R_{his} + R_{his}) - \delta h$$

$$\frac{dN_b}{dt} = \Lambda_b - \mu_b N_h$$

$$\Rightarrow \frac{dN_b}{dt} = \Lambda_b - \mu_b(S_b + E_b + I_b)$$

(5)

$$\frac{dN_b}{dt} = \Lambda_b - \mu_b N_b$$

(6)

We state that $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ and $N_b \leq \frac{\Lambda_b}{\mu_b}$ for all $t \geq 0$. It then follows from equations (5) and (6) that

$$\frac{dN_h}{dt} \leq \Lambda - \mu_h N_h$$

$$\frac{dN_b}{dt} \leq \Lambda - \mu_b N_b$$

Implementing Grownwall’s inequality in [3], we have;

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} + \left( N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_b t}$$

$$N_b(t) \leq \frac{\Lambda_b}{\mu_b} + \left( N_b(0) - \frac{\Lambda_b}{\mu_b} \right) e^{-\mu_b t}$$

And hence $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ and $N_b(t) \leq \frac{\Lambda_b}{\mu_b}$ for all $t \geq 0$ whenever $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$ and $N_b(0) \leq \frac{\Lambda_b}{\mu_b}$.

Succinctly, $\lim_{t \to \infty} \sup \remum N_h \leq \frac{\Lambda_h}{\mu_h}$ & $\lim_{t \to \infty} \sup \remum N_b \leq \frac{\Lambda_b}{\mu_b}$
Showing that \( N_h(t), \ S_h(t), \ E_h(t), \ I_h(t), \ R_{hm}(t), \ R_{hs}(t), \ S_b(t), \ E_b(t) \) and \( I_b(t) \) are bounded, which completes the proof.

Then \( \alpha = \begin{bmatrix} (S_h, E_h, I_h, R_{hm}, R_{hs}, S_b, E_b, I_b) \in \mathbb{R}_+^8, N_h(t) \leq \frac{\hat{\lambda}_h}{\mu_h}, N_b(t) \leq \frac{\hat{\lambda}_b}{\mu_b}, S_h(t) \geq 0, E_h(t) \geq 0, I_h(t) \geq 0, R_{hm}(t) \geq 0, R_{hs}(t) \geq 0, S_b(t) \geq 0, E_b(t) \geq 0, I_b(t) \geq 0 \end{bmatrix} \) \( \) \( (7) \)

### 4.0 Basic reproductive number, \( R_0 \)

A sacrosanct parameter in the epidemiological model is the basic reproductive number \( R_0 \). It determines if a disease will invade a population or not. This research aims to sensitize all parameters which will make the value of \( R_0 \) less than unity. Representing the reproductive number for human by \( R_{0h} \), and bat by \( R_{0b} \) and the using the popular generation matrix defined by [25], that is \( R_{0b} = \rho(FV^{-1}) \) we have;

Considering equation (2);

Let \( \chi = [E_h, I_h, R_{hm}]^T, \chi = (F_h + V_h)\chi \)

\[
F = \begin{pmatrix}
0 & \frac{\beta_h \cap h}{\mu_h} & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix}, \quad V = \begin{pmatrix}
(\mu_h + \phi_h) & 0 & 0 \\
-\phi_h & (\mu_h + \delta + c\mu + (1-c)\tau) & -\rho \\
0 & -c\tau & \mu_h
\end{pmatrix}
\]

\[
FV^{-1} = \begin{pmatrix}
\beta_h \cap h \theta_h & \beta_h \cap h & \beta_h \cap h \rho \\
\mu_h(\mu_h + \theta_h)(\mu_h + \delta + c\tau + (1-c)\tau) & \mu_h(\mu_h + \delta + c\tau + (1-c)\tau) & \mu_h(\mu_h + \delta + c\tau + (1-c)\tau) \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix}
\]

(8)
$$R_{0h} = \rho(FV^{-1}) = \frac{\beta_1 \wedge_h \theta_h}{\mu_h(\mu_h + \theta_h)(\mu_h + \delta + c \tau + (1 - c)\tau)}.$$  \hfill (9)

Comparably, by considering equation (3) and using the next generation matrix approach as above, i.e.,

$$R_{0b} = \rho(FV^{-1}).$$

We let \( \chi = [E_b, I_b]^T \), \( \chi^* = (F_b + V_b)\chi \)

Where \( F_b = \begin{pmatrix} 0 & \frac{\beta_3 \wedge_b}{\mu_b} \\ \frac{\beta_3 \wedge_b}{\mu_b} & 0 \end{pmatrix} \), \( V_b = \begin{pmatrix} (\mu_b + \theta_b) & 0 \\ -\theta_b & \mu_b \end{pmatrix} \)

$$FV^{-1} = \begin{pmatrix} \frac{\theta_b \beta_1 \wedge_b}{\mu_b^2(\mu_b + \theta_b)} & \frac{\beta_3 \wedge_b (\mu_b + \theta_b)}{\mu_b^2(\mu_b + \theta_b)} \\ 0 & \frac{\beta_3 \wedge_b (\mu_b + \theta_b)}{\mu_b^2(\mu_b + \theta_b)} \end{pmatrix}$$ \hfill (10)

With \( R_{0b} = \frac{\theta_b \beta_3 \wedge_b}{\mu_b^2(\mu_b + \theta_b)} \) \hfill (11)

5.0 Stability Analysis of the Disease Free Equilibrium (DFE)

The existence of the DFE points is the stable–state – solution where the Ebola virus hemorrhage infection dies out from the population and this implies the entire population is completely susceptible.

Then \( S_h^0 \neq 0 \) and \( S_v^0 \neq 0 \)

At the DFE point at \( E^0 \), \( S_h = E_h = I_h = R_{hiv} = R_{hvi} = S_b = E_b = I_b = 0 \)
\[ v_h - \mu_h S_h^0 - \phi_1 S_h^0 = 0 \]
\[ \phi_1 S_h^0 - (\mu_h + \theta_h) E_h^0 = 0 \]
\[ \theta_h E_h^0 + \rho R_{hasi}^0 - (\mu_h + \delta + \epsilon \tau + (1 - c) \tau) I_h^0 = 0 \]
\[ c d_h^0 - \mu_i R_{hasi}^0 = 0 \]
\[ (1 - c) d_h^0 - (\mu_h + \rho) R_{hasi}^0 = 0 \]
\[ \wedge_b - (\mu_b + \phi_2) S_b^0 = 0 \]
\[ \phi_2 S_b^0 - (\mu_b + \theta_b) E_b^0 = 0 \]
\[ \theta_b E_b^0 - \mu_i I_b^0 = 0 \]

Since \( S_h^0 \neq 0 \) at DFE, then \( E_h^0 = 0, I_h^0 = 0, R_{hasi}^0 = 0, R_{hasi}^0 = 0 \)

\[
E^0 = \left( S_h^0, E_h^0, I_h^0, R_{hasi}^0, R_{hasi}^0, S_b^0, E_b^0, I_b^0 \right) = \left( \frac{\wedge_h}{\mu_b}, 0, 0, 0, 0, \frac{\wedge_h}{\mu_b}, 0, 0 \right)
\]

(13)

5.1 Local Stability Analysis of the Model

Theorem 2

The DFE \( E^0 = \left( \frac{\wedge_h}{\mu_b}, 0, 0, 0, 0, \frac{\wedge_h}{\mu_b}, 0, 0 \right) \) of the system is locally asymptotically stable if and only if all Eigenvalues of the system’s Jacobian are non-positive real values.

Proof

Owing to the above theorem, Jacobian matrix of the systems of equation (2)- (3) at DFE (13), \( J(S_h, E_h, I_h, R_{hasi}, R_{hasi}, S_b, E_b, I_b) \) we have;
\[ J = \begin{bmatrix}
-\phi - \mu_b & 0 & -\beta_1 \hat{S}_h^{*} & 0 & 0 & 0 & 0 & -\beta_2 \hat{S}_h^{*} \\
\phi_1 & -(\mu_b + \theta_b) & \frac{\beta_1 \hat{S}_h^{*}}{1+ml_b} & 0 & 0 & 0 & 0 & \frac{\beta_2 \hat{S}_h^{*}}{1+ml_b} \\
0 & \theta_b & -(\mu_b + \delta + c \tau + (1-c)\tau) & 0 & \rho & 0 & 0 & 0 \\
0 & 0 & \frac{c \tau}{1+ml_b} & -\mu_h & 0 & 0 & 0 & 0 \\
0 & 0 & (1-c)\tau & 0 & -(\mu_b + \rho) & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -(\mu_b + \phi_2) & 0 & -\beta_1 \hat{S}_h^{*} & \frac{\beta_2 \hat{S}_h^{*}}{1+ml_b} \\
0 & 0 & 0 & 0 & \phi_2 & -(\mu_b + \theta_b) & \frac{\beta_1 \hat{S}_h^{*}}{1+ml_b} & -\mu_b \\
0 & 0 & 0 & 0 & 0 & 0 & \theta_b & -\mu_b 
\end{bmatrix} \]

At DFE, \( E^0 = \left( \frac{\mu_h}{\mu_b}, 0, 0, 0, 0, 0, 0, 0 \right) \)

Let \( X = (\mu_b + \theta_b), \ Y = (\mu_h + \delta + c \tau + (1-c)\tau), \ Z = (\mu_b + \rho) \) and \( U = (\mu_b + \theta_b) \)
\[ J = \begin{bmatrix} -\mu_h & 0 & -\beta_1 \wedge h & 0 & 0 & 0 & 0 & -\beta_2 \wedge h \\ 0 & -X & \beta_1 \wedge h & \mu_h & 0 & 0 & 0 & \beta_2 \wedge h \\ 0 & \theta_h & -Y & 0 & \rho & 0 & 0 & 0 \\ 0 & 0 & c \tau & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-c)\tau & 0 & -Z & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_b & 0 & -\beta_3 \wedge h & \mu_h \\ 0 & 0 & 0 & 0 & 0 & U & \beta_3 \wedge h & \mu_b \\ 0 & 0 & 0 & 0 & 0 & \theta_b & -\mu_b & -\mu_b \end{bmatrix} \] (15)

\[ |J - \lambda I| = \begin{bmatrix} -\mu_h - \lambda & 0 & -\beta_1 \wedge h & 0 & 0 & 0 & 0 & -\beta_2 \wedge h \\ 0 & -X - \lambda & \beta_1 \wedge h & \mu_h & 0 & 0 & 0 & \beta_2 \wedge h \\ 0 & \theta_h & -Y - \lambda & 0 & \rho & 0 & 0 & 0 \\ 0 & 0 & c \tau & -\mu_h - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-c)\tau & 0 & -Z - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_b - \lambda & 0 & -\beta_3 \wedge h & \mu_b \\ 0 & 0 & 0 & 0 & 0 & U - \lambda & \beta_3 \wedge h & \mu_b \\ 0 & 0 & 0 & 0 & 0 & \theta_b & -\mu_b - \lambda \end{bmatrix} \] (16)

\(-\mu_h - \lambda\) is the only non-zero entry in the first column, hence \(\lambda_1 = -\mu_h\).

Deleting the perpendicular row and column gives

\[ J_0 = \begin{bmatrix} -X - \lambda & \beta_1 \wedge h & 0 & 0 & 0 & 0 & 0 & \beta_2 \wedge h \\ \theta_h & -Y - \lambda & 0 & \rho & 0 & 0 & 0 & 0 \\ 0 & c \tau & -\mu_h - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-c)\tau & 0 & -Z - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_b - \lambda & 0 & -\beta_3 \wedge h & \mu_b \\ 0 & 0 & 0 & 0 & 0 & U - \lambda & \beta_3 \wedge h & \mu_b \\ 0 & 0 & 0 & 0 & 0 & \theta_b & -\mu_b - \lambda \end{bmatrix} \] (17)
Similarly, row 3, \(-\mu_h - \lambda\) is the only non-zero entry, hence \(\hat{\lambda}_2 = -\mu_h\). Therefore:

\[
J_1 = \begin{bmatrix}
-X - \lambda & \frac{\beta_1 \wedge h}{\mu_h} & 0 & 0 & 0 & \frac{\beta_2 \wedge h}{\mu_h} \\
\theta_h & -Y - \lambda & \rho & 0 & 0 & 0 \\
0 & (1-c)\tau & -Z - \lambda & 0 & 0 & 0 \\
0 & 0 & 0 & -\mu_h - \lambda & 0 & -\frac{\beta_3 \wedge h}{\mu_h} \\
0 & 0 & 0 & 0 & -U - \lambda & \frac{\beta_3 \wedge h}{\mu_h} \\
0 & 0 & 0 & 0 & 0 & \theta_b - \mu_b - \lambda
\end{bmatrix}
\]  

(18)

Similarly at row 4, \(-\mu_b - \lambda\) is the only entry, hence \(\hat{\lambda}_3 = -\mu_b\), new \(J_2\) is:

\[
J_2 = \begin{bmatrix}
-X - \lambda & \frac{\beta_1 \wedge h}{\mu_h} & 0 & 0 & \frac{\beta_2 \wedge h}{\mu_h} \\
\theta_h & -Y - \lambda & \rho & 0 & 0 \\
0 & (1-c)\tau & -Z - \lambda & 0 & 0 \\
0 & 0 & 0 & -U - \lambda & \frac{\beta_3 \wedge h}{\mu_b} \\
0 & 0 & 0 & 0 & \theta_b - \mu_b - \lambda
\end{bmatrix}
\]  

(19)

Splitting matrix (19) into two for scrutiny, that is:

\[
J_2^* = \begin{bmatrix}
-X & \frac{\beta_1 \wedge h}{\mu_b} & 0 & 0 & \frac{\beta_2 \wedge h}{\mu_b} \\
\theta_b & -Y & \rho & 0 & 0 \\
0 & (1-c)\tau & -Z & 0 & 0 \\
0 & 0 & 0 & -U & \frac{\beta_3 \wedge h}{\mu_b} \\
0 & 0 & 0 & 0 & \theta_b - \mu_b
\end{bmatrix}
\]  

and \(I\hat{\lambda} = \begin{bmatrix} \lambda & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 \\ 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & \lambda \end{bmatrix}\)  

(20)

Reducing \(J_2^*\) in row echelon form by the row operations besides it
\[
-X \begin{pmatrix}
\beta_1 \wedge h & 0 & 0 \\
\mu_h & -Y & 0 \\
0 & -Z & \rho
\end{pmatrix} \theta_h + 0 \begin{pmatrix}
\beta_2 \wedge h \\
\mu_h & 0 \\
0 & 0
\end{pmatrix} \theta_h + XR
\]

(21)

\[
= -X \begin{pmatrix}
\beta_1 \wedge h \\
\mu_h & -Y \\
0 & -Z \\
0 & 0 & (1-c)\tau
\end{pmatrix} X \rho \begin{pmatrix}
\theta_h \beta_1 \wedge h \\
\mu_h & -X Y \\
0 & 0 \\
0 & 0 & 0
\end{pmatrix} \theta_h + 0 \begin{pmatrix}
\beta_2 \wedge h \\
\mu_h & -Y \\
0 & 0 \\
0 & 0 & 0
\end{pmatrix} \theta_h + \left(1-c\right)\tau \beta_3 \wedge h + R_3 \rightarrow \left(1-c\right)R_2 - \begin{pmatrix}
\theta_h \beta_1 \wedge h \\
\mu_h & -X Y \\
0 & 0 & 0
\end{pmatrix} \theta_h + \left(1-c\right)\tau \beta_2 \wedge h + UR
\]

(22)

Relating to matrices in (19), that is;
\[ R_s(J^*_2) - 1 \lambda = \begin{bmatrix} -X - \lambda & \frac{\beta_1 \wedge h}{\mu_h} & 0 & 0 & \frac{\beta_1 \wedge h}{\mu_h} \\ 0 & \frac{(\theta_b \beta_1 \wedge h)}{\mu_h} - XY - \lambda & X\rho & 0 & \frac{\theta_b \beta_2 \wedge h}{\mu_h} \\ 0 & 0 & (1 - c)\tau XP + Z\left(\frac{\theta_b \beta_1 \wedge h}{\mu_h} - XY\right) - \lambda & 0 & \frac{(1 - c)\tau \theta_b \beta_2 \wedge h}{\mu_h} \\ 0 & 0 & 0 & -U - \lambda & \frac{\beta_1 \wedge h}{\mu_h} \\ 0 & 0 & 0 & 0 & \left[\frac{\theta_b \beta_3 \wedge h}{\mu_b} - \mu_b U\right] - \lambda \end{bmatrix} \]

\[ \lambda_4 = -X, \quad \lambda_3 = \frac{\theta_b \beta_1 \wedge h}{\mu_h} - XY, \quad \lambda_6 = X\rho(1 - c)\tau + Z\left(\frac{\theta_b \beta_1 \wedge h}{\mu_h} - XY\right), \quad \lambda_7 = -U, \quad \lambda_8 = \frac{\theta_b \beta_3 \wedge h}{\mu_b} - \mu_b U, \]

\[ \lambda_1 = -\mu_h, \quad \lambda_2 = -\mu_h, \quad \lambda_3 = -\mu_h, \quad \lambda_4 = -(\mu_h + \theta_h), \quad \lambda_5 = \frac{\theta_b \beta_1 \wedge h}{\mu_h} - (\mu_h + \theta_h)(\mu_h + \delta + c\tau + (1 - c)\tau), \]

\[ \lambda_6 = \frac{(\mu_h + \theta_h)\rho(1 - c)\tau + (\mu_h + \rho)(\theta_h \beta_1 \wedge h) - (\mu_h + \theta_h)(\mu_h + \delta + c\tau + (1 - c)\tau)}{\mu_h}, \quad \lambda_7 = - (\mu_h + \theta_h), \]

\[ \lambda_8 = \frac{\theta_b \beta_3 \wedge h}{\mu_b} - \mu_b (\mu_b + \theta_h). \]

**Theorem 3:** The disease-free equilibrium is locally asymptotically stable when \( R_s < 1 \)

**Proof:**

We proceed from the Eigenvalues \( \lambda_3 = \frac{\theta_b \beta_1 \wedge h}{\mu_h} - (\mu_h + \theta_h)(\mu_h + \delta + c\tau + (1 - c)\tau) \) and

\[ \lambda_8 = \frac{\theta_b \beta_3 \wedge h}{\mu_b} - \mu_b (\mu_b + \theta_h) \] obtained above. We already have \( R_{0h} = \frac{\beta_1 \wedge h \theta_h}{\mu_h (\mu_h + \theta_h)(\mu_h + \delta + c\tau + (1 - c)\tau)}. \)
\[ \lambda_g = \frac{\theta_b \beta_3 \wedge_h}{\mu_h} - (\mu_h + \theta_b)(\mu_h + \delta + c\tau + (1-c)\tau) \]

\[ = -\left(\mu_h + \theta_b)(\mu_h + \delta + c\tau + (1-c)\tau) - \frac{\theta_b \beta_3 \wedge_h}{\mu_h}\right) \]

\[ = -(\mu_h + \theta_b)(\mu_h + \delta + c\tau + (1-c)\tau) \left[1 - \frac{\beta_3 \wedge_h \theta_h}{\mu_h(\mu_h + \theta_b)(\mu_h + \delta + c\tau + (1-c)\tau)}\right] \]

\[ = -(\mu_h + \theta_b)(\mu_h + \delta + c\tau + (1-c)\tau) [1 - R_{0b}] \]

\[ \leq -[(\mu_h + \theta_b)(\mu_h + \delta + c\tau + (1-c)\tau)(1 - R_{0b})] \quad (24) \]

Since \( R_{0b} = \frac{\theta_b \beta_3 \wedge_h}{\mu_b^2 (\theta_b + \mu_b)} \) and from above;

\[ \lambda_a = \frac{\theta_b \beta_3 \wedge_h}{\mu_b} - \mu_b(\mu_b + \theta_b) \]

\[ = -(\mu_b(\mu_b + \theta_b) - \frac{\theta_b \beta_3 \wedge_h}{\mu_b}) \]

\[ = -(\mu_b(\mu_b + \theta_b)) \left[1 - \frac{\theta_b \beta_3 \wedge_h}{\mu_b^2 (\mu_b + \theta_b)}\right] \]

\[ = -(\mu_b(\mu_b + \theta_b)) [1 - R_{0b}] \]

\[ \leq -\mu_b(\mu_b + \theta_b)(1 - R_{0b}) \quad (25) \]

Eigenvalues \( \lambda_g \) and \( \lambda_a \) remain negative justifying that \( R_{0b} < 1 \) and \( R_{0h} < 1 \). And since \( R_{0b} < 1 \) and \( R_{0h} < 1 \), then \( R_{0G} = R_{0b} \times R_{0h} < 1 \). Therefore \( R_{0G} < 1 \).

### 5.2 Global stability analysis of the model

The disease-free equilibrium (DFE) of the systems (1) – (8) is globally stable on \( M \) if \( R_G < 1 \).

**Proof**

We implement Castillo – Chavez and co-authors method described in [4, 15] to prove this theorem.

We define some new variables and partition our systems of equations (1) – (8) into two systems.
Let \( X = (S_h, R_{bhi}, R_{bai}, S_b) \)
\( I = (E_h, I_h, E_b, I_b) \) \hspace{1cm} (26)

\[
\frac{dX}{dt} = F(X,I)
\]
And
\[
\frac{dl}{dt} = G(X,I), G(X,0) = 0 \hspace{1cm} (27)
\]

Where \( X \in \mathbb{R}^4 \) represents the number of compartments without infection and \( I \in \mathbb{R}^4 \) represents the numbers of compartments with infection. The double vector value functions \( F(X,I) \) and \( G(X,I) \) are defined as follows:

\[
F(X,) = \begin{bmatrix}
\wedge_h - \mu_h S_h - \phi_1 S_h \\
c \phi_1 I_h - \mu_h R_{bhi} \\
(1-c) \phi_1 I_h - (\mu_h + \rho) R_{bai}
\end{bmatrix}
\] \hspace{1cm} (28)

\[
G(X,I) = \begin{bmatrix}
\phi_1 S_h - (\mu_h + \theta_h) E_h \\
\theta_h E_h + \rho R_{bai} - (\mu_h + \delta + c \tau + (1-c) \tau) I_h \\
\phi_2 S_b - (\mu_b + \theta_b) E_b \\
\theta_b E_b - \mu_b I_b
\end{bmatrix}
\] \hspace{1cm} (29)

With \( G(X,0) = 0 \) and \( E^0 = (X^*,0) \) which represent the disease-free equilibrium of the subsystems

\[
X^* = \begin{pmatrix}
\wedge_h \\
\mu_h \\
\wedge_b \\
\mu_b
\end{pmatrix}
\]

The conditions \( H_1 \) and \( H_2 \) below must be completely satisfied for global stability.

\[
H_1 : \left( \frac{dX}{dt} \right) = F(X,0)
\]

\[
H_2 : G(X,I) = BI \Rightarrow G(X,I)
\] \hspace{1cm} (30)

Where B is an M-matrix (the off-diagonal element of B is non-negative).

Now, considering the reduced system, \( \frac{dX}{dt} = F(X,0) \), i.e. \( I = 0 \). Then,
\[ \begin{align*}
\frac{dS_h}{dt} & = \beta_1 \frac{S_h}{\mu_b} - \mu_h S_h \\
\frac{dR_{his}}{dt} & = -\mu_h R_{his} \\
\frac{dR_{his}}{dt} & = -(\mu_h + \rho) R_{his} \\
\frac{dS_b}{dt} & = \mu_b - \mu_b S_b
\end{align*} \] (31)

\[ X^* = (S_h, R_{his}, R_{his}, S_b) = \left( \frac{\beta_1 S_h}{\mu_b}, 0, 0, \frac{\beta_2 S_h}{\mu_b} \right) \]

is the GAS equilibrium points for the reduced system \( \frac{dX}{dt} = F(X,0) \).

The solution of \( X^* \) has been resolved when obtaining the positive invariant region. At \( t \to \infty \), we have the respective values at DFE. Hence, the convergence of solutions of equations (29) – (31) is global in \( M \) and

\[ G(X,I) = [B I - \hat{G}(X,I) | \hat{G}(X,I) \geq 0, \forall (X,I) \in M] \] (32)

Where \( B = \begin{bmatrix}
- (\mu_h + \theta_h) & \frac{\beta_1 S_h}{\mu_b} & 0 & \frac{\beta_2 S_h}{\mu_b} \\
\theta_h & - (\mu_h + \delta + c \tau + (1 - c) \tau) & 0 & 0 \\
0 & 0 & - (\mu_b + \theta_b) & \frac{\beta_1 S_h}{\mu_b} \\
0 & 0 & 0 & - \mu_b
\end{bmatrix} \)

\[ I = \begin{bmatrix}
E_h \\
I_h \\
E_b \\
I_b
\end{bmatrix}, \quad \hat{G}(X,I) = \begin{bmatrix}
\frac{\beta_1 S_h}{\mu_b} + \beta_2 S_h \left( \frac{\beta_1 S_h}{\mu_b} - S_h \right) \\
\beta_3 \left( \frac{\beta_1 S_h}{\mu_b} - S_h \right)
\end{bmatrix} \] (33)

5.2.1 Endemic Equilibrium state (EES)

**Theorem 4:** If \( R_0 > 1 \), system (2) – (3) express a distinct EES, \( E^* = \{ S_h^*, E_h^*, I_h^*, R_{his}^*, R_{his}^*, S_b^*, E_b^*, I_b^* \} \).

At equilibrium:
\[ \begin{align*}
\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dR_{his}}{dt} = \frac{dR_{his}}{dt} = \frac{dS_b}{dt} = \frac{dE_b}{dt} = \frac{dI_b}{dt} = 0.
\end{align*} \]
Let \( \frac{dS_b}{dt} = \frac{dE_b}{dt} = \frac{dI_b}{dt} = 0 \), then we have;

\[
\land_b - (\mu_b + \phi_2)S_b = 0 \quad (34)
\]
\[
\phi_2S_b - (\mu_b + \theta_b)E_b = 0 \quad (35)
\]
\[
\theta_bE_b - \mu_bI_b = 0 \quad (36)
\]

Recall \( \phi_2 = \frac{\beta_3I_b}{1 + ml_b} \) at \( m = 0 \) for feasibility, and then we have;

From (36)

\[
E_b = \frac{\mu_bI_b}{\theta_b} \quad (37)
\]

Putting (37) into (35) gives;

\[
I_b \left[ \beta_3S_b - \left( \frac{(\mu_b + \theta_b)\mu_b}{\theta_b} \right) \right] = 0
\]

\[
\Rightarrow I_b = 0 \quad \text{or} \quad \beta_3S_b - \left( \frac{(\mu_b + \theta_b)\mu_b}{\theta_b} \right) = 0
\]

Therefore, \( S_b = \frac{(\mu_b + \theta_b)\mu_b}{\beta_3\theta_b} \quad (38) \)

Putting (38) into (34) gives:

\[
I_b^* = \frac{\land_b \beta_3\theta_b - \mu_b^2(\mu_b + \theta_b)}{\beta_3\mu_b(\mu_b + \theta_b)} \quad (39)
\]

Putting (39) into (36), we have:

\[
E_b^* = \frac{\mu_bI_b^*}{\theta_b} = \frac{\land_b \beta_3\theta_b - \mu_b^2(\mu_b + \theta_b)}{\beta_3\mu_b(\mu_b + \theta_b)} \quad (40)
\]
From equation (2)

\[ \wedge_b - \mu_h S_h - \beta_h I_h S_h = 0 \]  
\[ \beta_h I_h S_h - (\mu_h + \theta_h)E_h = 0 \]  
\[ \theta_h E_h + \rho R_{hni} - (\mu_h + \delta + c \tau + (1-c)\tau)I \]  
\[ c \mu_h - \mu_h R_{his} = 0 \]  
\[ (1-c)\mu_h - (\mu_h + \rho)R_{hni} = 0 \]  

From (44), we have \[ R_{hni}^* = \frac{(1-c)\mu_h^*}{(\mu_h + \rho)} \]  

Putting (45) into (42), we have,

\[ E_h^* = \left[ (\mu_h + \delta + c \tau + (1-c)\tau)(\mu_h + \rho) - \rho \tau (1-c) \right] \frac{I_h^*}{\mu_h + \rho} \]  

Putting (46) into (42) gives

\[ \beta_h I_h^* S_h - \frac{I_h^* [(\mu_h + \theta_h)[(\mu_h + \delta + c \tau + (1-c)\tau)(\mu_h + \rho) - \rho \tau (1-c)]]}{(\mu_h + \rho)} = 0 \]

This implies either \[ I_h^* = 0 \] or 
\[ \beta_h S_h - \frac{[(\mu_h + \theta_h)[(\mu_h + \delta + c \tau + (1-c)\tau)(\mu_h + \rho) - \rho \tau (1-c)]]}{(\mu_h + \rho)} = 0 \]

Therefore 
\[ S_h = \frac{[(\mu_h + \theta_h)[(\mu_h + \delta + c \tau + (1-c)\tau)(\mu_h + \rho) - \rho \tau (1-c)]]}{\beta_h (\mu_h + \rho)} \]  

To get \[ I_h^* \], we put (48) into (41) and we have the expressions below justifying the equilibrium states of (2) – (3).

Solving equations (2) – (3) considering the equilibrium state, we have:

\[ I_h^* = \frac{\wedge_b (\mu_h + \theta_b)[(\mu_h + \delta + c \tau + (1-c)\tau)(\mu_h + \rho) - \rho \tau (1-c)] - \wedge_b \beta_h (\mu_h + \rho)}{\beta_h (\mu_h + \theta_b)[(\mu_h + \delta + c \tau + (1-c)\tau)(\mu_h + \rho) - \rho \tau (1-c)]} \]  

\[ I_h^* = \frac{\wedge_b \beta_h \theta_h - \mu_h^* (\mu_h + \theta_b)}{\beta_h (\mu_h + \theta_b)} \]
Sensitivity Analysis of the Model

The parameters which are most responsible for the prevalence and virulence of Ebola virus diseases can be determined by sensitizing all incorporated parameters. This sensitivity analysis is commonly used to determine the robustness of model prediction to parameter values. The normalized forward-sensitivity index of a variable $R_0$ that differentially depends on a parameter $f$ is defined as: 

$$\Gamma_f = \frac{\partial R_0}{\partial f} \times \frac{f}{R_0}$$

Table 1

<table>
<thead>
<tr>
<th>S/N</th>
<th>Parameters</th>
<th>Signs</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$\Lambda_h$</td>
<td>+</td>
<td>0.5</td>
</tr>
<tr>
<td>2</td>
<td>$\Lambda_b$</td>
<td>+</td>
<td>0.5</td>
</tr>
<tr>
<td>3</td>
<td>$\beta_1$</td>
<td>+</td>
<td>0.5</td>
</tr>
<tr>
<td>4</td>
<td>$\beta_2$</td>
<td>+</td>
<td>0.5</td>
</tr>
<tr>
<td>5</td>
<td>$\beta_3$</td>
<td>+</td>
<td>0.5</td>
</tr>
<tr>
<td>6</td>
<td>$\theta_h$</td>
<td>+</td>
<td>0.00199</td>
</tr>
</tbody>
</table>
Interpretation of sensitivity indices

Table 1 shows the numerical result of our sensitivity analysis carried out. The above result shows that the effective transmission rates $\beta_1, \beta_2, \beta_3$, and the recruitment rates for human and bats populations $\Lambda_h, \Lambda_b$ respectively have prominent effects in prevalence and virulence of the Ebola virus since the values are predominantly positive (+0.5 each) concerning graph figure 14 and 15. So, an increase (or decrease) in their values by some certain percentage will result in an increase (or decrease) in the effective basic reproduction number. And the two remaining positive sensitized values which are considerably small have traceable effects on the $R_0$. Contrarily, the negative indices in order of decrease are as follows: Rodents’ natural mortality rate ($\mu_b$), treatment rate ($\tau$), humans’ natural mortality rate ($\mu_h$), and disease-prompted mortality rate ($\delta$) which are -1.02189, -0.691, -0.51, -0.189 as clearly stated in Table 1. These negative values infer that an increase (or decrease) of any of these parameters will bring about a decrease (or increase) in the basic reproduction number.

Thus, these parameters with negative indices suggest that the decimation of bats by nature or man, quick treatment of the infected humans, immunization (sterilized or partial) should be drastically increased while the positive indices suggest that all possible ways of transmitting the virus should be avoided. Recovered pregnant and breastfeeding women, and all recovered individuals should be advised to isolate themselves till
the remaining component of the virus leaves their body. Lastly, putting other preventive measures into consideration would make the environment achieve EBOV-free equilibrium.

Results and Discussions

In this study, we have examined and analyzed the simulated results of the bat-human model of Ebola virus disease. This is to justify the analytical solutions as well as interpret the dynamical behavior of the model system. Using Ordinary Differential Equation 45 (ODE-45) in MATLAB software, we have simulated the whole and individual compartments of the model concerning the values of the state variables whose initial conditions are provided. Table 2 and Table 3 contained the state variables and parameter values.

Table 2

<table>
<thead>
<tr>
<th>STATE</th>
<th>VALUES</th>
<th>SOURCES</th>
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<tbody>
<tr>
<td>$S_h$</td>
<td>6000</td>
<td>Estimated</td>
</tr>
<tr>
<td>$E_h$</td>
<td>4300</td>
<td>Assumed</td>
</tr>
<tr>
<td>$I_h$</td>
<td>990</td>
<td>Assumed</td>
</tr>
<tr>
<td>$R_{his}$</td>
<td>500</td>
<td>Estimated</td>
</tr>
<tr>
<td>$R_{uni}$</td>
<td>150</td>
<td>Estimated</td>
</tr>
<tr>
<td>$S_b$</td>
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<td>Assumed</td>
</tr>
<tr>
<td>$E_b$</td>
<td>80</td>
<td>Assumed</td>
</tr>
<tr>
<td>$I_b$</td>
<td>35</td>
<td>Assumed</td>
</tr>
</tbody>
</table>

Table 3

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>VALUE</th>
<th>DIMENSION</th>
<th>SOURCE</th>
</tr>
</thead>
<tbody>
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<td>$\Lambda_h$</td>
<td>1.2</td>
<td>Humans x day$^{-1}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>Parameter</td>
<td>Value</td>
<td>Unit</td>
<td>Source/Note</td>
</tr>
<tr>
<td>-----------</td>
<td>-------</td>
<td>---------------</td>
<td>-------------------------</td>
</tr>
<tr>
<td>$\Lambda_b$</td>
<td>0.7</td>
<td>Bat x day$^{-1}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>0.04-0.004</td>
<td>Dimensionless</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>0.04-0.004</td>
<td>Dimensionless</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\beta_3$</td>
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<tr>
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<td>$c$</td>
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Figure 2: Bats- Humans Population Density

Figure 3: Dynamical behavior of the humans in the population density

Figure 4: Dynamical behavior of the bats in the population density.

Figure 2-4 explicitly shows the dynamical behavior of human-bats in the population density. Figure 2 shows the overall dynamical behavior of the deterministic model incorporating both the human-bats curves while Figure 3 & 4 demonstrate the dynamical behavior of the human and bat curves accordingly.
Figure 5: Dynamical behaviors of eliminating infectious bats

This curve in Figure 5 depicts what happens when the natural mortality rate increases. When the death rate of infectious bats is increased either by nature, hunting, or other control measures, the population dynamic curves will be stabled hence, the DFE is achieved which promotes avirulence in the population density. And if otherwise, the curves approach endemic equilibrium.

Figure 6: The graph showing the dynamism of recovered humans that is not under isolation
Figure 7: The graph shows the dynamism of recovered humans that are under isolation.

Figure 8: The graph depicts the effect of relapse rate on the infectious individuals.
Research shows that some percentage of recovered individuals still have the virus in the fluidic part of their bodies - the blood, eyeball, semen, sperms, and breast-milk to mention a few. This could last for hours, days, and even months. So these sets of individuals are expected to be isolated till the virus finally leaves their body to avoid the prevalence of the virus. This study raises awareness of possible reinfection since the majority of the populations are not aware of this mode of reinfection “relapse rate” by showing the implication of these individuals in the population density. The above figure 6-10 validates and demonstrates the huge effect of not isolating recently recovered individuals. Figure 6-7 shows the dynamical behavior of the isolation parameters. And embracing the isolation idea will promote avirulence. If the probability distribution of isolation is unity, the curve of the recovered individuals that are not under isolation $R_{ini}$ as shown in figure 6 is completely flattened “to zero” which affirms that there will be no reinfection of Ebola virus diseases in Nigeria and concerned countries in Africa at large. So, we suggest that Governing bodies should make it a MUST for all public health workers and medical practitioners to instill the attribute of isolation. Also, figure 8-10 clearly shows on the curve the huge effect of relapse rate on the population dynamics. Also, doubling the relapse rate depopulated recovered compartments that are not isolated by drastically populating the infectious class which is a big threat to humanity. Though the recovered individuals in isolation are increased which is advisable if and only if reinfection is no longer recorded. It is
crystal clear that **figure 6-10** are intertwined; creating awareness of the unknown aspect of EBOV and also validating what will happen if “isolation” is completely embraced without complacency.

**Figure 11:** The effects of instantaneously administering drugs

**Figure 11** shows the efficacy of drugs “Inmazeb and Ebanga” being immediately administered during the treatment of infectious individuals was shown in the above graph. Having these two drugs available with(out) vaccination, and administering them accurately would ameliorate the prevalence of EBOV instantaneously. And increasing the treatment rate of the affected population, the infectious curves then approach the disease-free equilibrium after a few days of the outbreak.

**Figure 12:** The dynamical behaviors of the saturation factors on susceptible population

**Figure 12** reveals the drastic changing effect of the susceptible individuals with or without the “saturation effect” $m$. This demonstrates that the rate at which humans are susceptible to Ebola virus disease is reduced when saturation is being introduced.
**Figure 13:** The dynamical behaviors of the saturation factors on the exposed population

**Figure 13** shows the drastic changing effect of the susceptible individuals with or without the “saturation effect (m)”. This demonstrates that the rate at which humans are being exposed to Ebola virus disease from bats (Chiroptera) is reduced when saturation set-in in the exposed compartment.

**Figure 14:** The Histogram plots of Sensitivity analysis against parameters (see Interpretation of Sensitivity Indices for reference)
Conclusion

In this research, we have analytically investigated the grievously devastating but avoidable consequences of not isolating newly recovered individuals from Ebola infection by proposing an unprecedented and feasible mathematical model - $SEIR_{hh} R_{hh} - SEI$, considering the human and bat populations. We further established the positivity and boundedness of the system. The reproduction number $R_{G}$ comprising both the humans $R_{oh}$ and bats $R_{ob}$ populations was also established to be less than unity. The persistence (Endemic) and Disease Free Equilibrium point of the model were ascertained to be asymptotically stabled both locally and globally, stating the longevity and transitory of the Ebola virus disease in the host population depending if $R_{G} < 1$ or $R_{G} > 1$. Also, the parameters were clinically sensitized and their sensitivity indices were stated. Numerical solution for the global stability at DFE shows that the disease would disappear in the host population at an instantaneous time t.
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Statements And Declaration

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Author’s Contribution

Corresponding Author’s Name: Agbomola Oluwasegun Joshua
Authorship roles: Conceptualization
Software
Data curation
Writing-review editing
Formal Analysis
Methodology
Writing Original draft

Author’s Name: Loyinmi Adedapo Chris
Authorship roles: Project Administration
Funding Acquisition
Investigation
Visualization
Resources Validation
Supervision

Competing Interest

The authors declare that they have no competing interests.

Data Availability
The datasets generated during and/or analyzed during the current study are available from the corresponding author on plausible request.

References


