ON SITR THEORETICAL MODEL OF EBOLA VIRUS PROPAGATION WITH RELAPSE AND REINFECTION

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Abstract

Despite the national government's commitment to eradicating the Ebola virus epidemic and the WHO's combined efforts in this regard in Ebola-torn countries in Africa, many people are still not aware of some possible modes of transmission of Ebola. Many researchers also failed to include these possible modes of transmission, reinfection, and relapse of EVD in their studies. We studied the dynamics of Ebola infection with relapse and reinfection by employing a non-linear SITR mathematical model. We computed the fundamental reproduction ratio, examined the model equilibria, and carried out a sensitivity analysis. Lassalle's invariance principle was employed to examine the global stability of the Ebola-free equilibrium. Further analysis indicates that the model exhibits a backward bifurcation. We therefore employed the centre manifold theorem to prove the model's asymptotic stability. Our analysis indicates that Ebola-free equilibrium was locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The global analysis conducted on the model revealed that the Ebola-free equilibrium was globally stable if $R_0 < 1$. Our model revealed that Ebola can be controlled by increasing the number of infectious people who go for treatment and reducing the transmission rate of people who are prone to the disease through an intensive educational campaign and vaccination of susceptible individuals.

Keywords**:** *STIR model, reproduction ratio, backward bifurcation, asymptotically stable, global stability.*

1.0 Introduction

Humans can contract the deadly Ebola virus disease (EVD), which has been known to have mortality rates in cases as high as 90% during previous occurrences (Lokuge et al., 2016). Ebola was named after the adjacent Ebola River, and it was discovered in 1976 in Zaire. A related outbreak involving a different strain of EVD happened in Sudan that same

year. Five (5) distinct Ebola virus species have been found, with more than 25 confirmed outbreaks of the disease occurring in Africa since 1976. Since 2000, there have been more outbreaks and cases of Ebola virus hemorrhagic fever, which continues to plague the people of equatorial Africa (Anna et al., 2015).

In 2014, Ebola virus outbreak resulted in over 6,000 clinically confirmed cases and left almost seventy percent of those infected with the disease dead in West Africa. According to Berge et al. (2016), the 2014 outbreak had the highest death toll among the 20 Ebola outbreaks that have happened since 1976. EVD symptoms are fever, weakness, diarrhoea, unexplained haemorrhage (bleeding or bruising) (Conrad et al., 2016; CDC, January 14, 2021), vomiting, severe headache, muscle pain, fatigue, and abdominal (stomach) pain (Sun et al., 2022; CDC, January 14, 2021).

Blood, faeces, and vomit are the most contagious body fluids (Osterholm et al., 2015). However, other fluids, including urine, saliva, breast milk, semen, and theoretically perspiration, may also be involved in the transmission of the virus (Roca et al., 2015). In several EVD outbreaks, funeral customs like washing dead bodies have been crucial to the spread of infection (Chan et al., 2014). Because the virus can live on surfaces or in items for hours or days, there may be more ways to spread it (Roca et al., 2015; January 14, 2021). Despite the virus being eliminated from the blood, clinically recovered patients can still have symptoms of the Ebola virus (MacIntyre et al., 2016; Chughtai et al., 2016). With over 10,000 Ebola survivors, WHO has focused its efforts in handling individuals who have survived the disease and the potential for an epidemic to recur as a result of the chronic persistence of the virus (WHO, 2016). 'Recurrence, also known as recrudescence, is the recurrence of symptoms in survivors as a result of the survival of viruses in immunologically protected body sites, whereas "reinfection" is the propensity of survivors to become infected again after recovery. Both of these events put a strain on disease prevention efforts and raise the possibility of Ebola virus epidemics (MacIntyre et al., 2016).

Examples of immunologically protected sites where the Ebola virus can survive include semen, perspiration, vaginal fluids, aqueous humour, urine, and liquid from the breast (MacIntyre et al., 2016; Chughtai et al., 2016).

Ninety-three Ebola survivors were all found to carry the virus' RNA in their semen 2-3 months after recovering from the illness, according to a study. This number decreased to sixty-five percent at 4-6 months and twenty-six percent at 7-9 months. (MacIntyre et al., 2016; Dean et al 2015). RT-PCR tests for Ebola can be positive for over nine months in certain survivors, according to studies on viral persistence (Sun et al., 2022). A positive PCR (polymerase chain reaction) test result might or might not show that a virus is still alive. Only a virus culture that is positive can determine an infection risk (MacIntyre et al., 2016). It is a rare but reported occurrence for someone who has recovered from EVD to experience relapse-symptomatic illness as a result of enhanced virus replication in a particular place. It is still unclear why this occurrence occurs. (WHO, February 23, 2021).

Theoretically, a re-challenge with a high viral load may result in EVD reinfection, as could a combination of both of these variables and waning or weakened immunity. Studies reveal that after a few years of recovery, some Ebola virus survivors experience a decline in their

antibody levels. (Maclntyre & Ahmed, 2016). Ebola has been declared eradicated in Liberia, Sierra Leone, and Guinea, although it has periodically returned. The virus' persistence in those who have survived is probably the cause of new infections (WHO, March 2016).

Experimental therapeutics are being researched for Ebola virus infections. (Passi et al., 2015; Ansari, 2014). Supportive care therapy is the mainstay of treatment for EVD, involving fluid and electrolyte balance, anticoagulants, procoagulants, oxygen status, and pain management (Passi et al., 2015; Choi, 2013). Due to the absence of effective antiviral medications or vaccines for the disease, contact tracking and early diagnosis of sick people for isolation and care in treatment facilities are essential for controlling an epidemic (Beeching et al., 2014; Chowell et al., 2015). Early treatment increases the likelihood that a patient will survive (Passi et al., 2015). Large numbers of virions in body fluids, tissues, and particularly skin are indicative of the late stages of the Ebola virus disease. Without an adequate barrier, those who are exposed to Ebola patients have a significant risk of contracting the disease. (Alehegn, 2014). Additionally, the findings of the research by Jian et al. (2017) and Messaoudi (2015) showed that the late stage of EBOV infection is associated with higher mortality.

In recent years, a number of authors have studied epidemiological models of EVD but almost all their studies on EVD propagations are carried out in classical settings (Berge et al., 2016). For instance, SI-model (Berge, 2016; Wang, 2016), SI-model (Fisman, 2014; Rachah & Torres, 2015), *SEIR* - model (Althaus, 2014; Chowell et al., 2004; Lekone & Finkenstdt, 2006; Rachah & Torres, 2016; Webb & Browne, 2016; Siettos, 2015), *SEIRD* - model (Fsina, et al., 2014; Ndanguza et al., 2013), $SEI_EI_LR_1R_2$-model(Agusto, 2017), *SEIRHD* - model (Agusto, 2015; Ivorra et al., 2015) where S, $E, I, I_E, I_L, H, R, R_1, R_2$-and denote the Susceptible, Exposed, Infectious, Symptomatic in early infection, Symptomatic in late infection, Hospitalized, Recovered/Removed(R andR_1), Immune and Dead/ Deceased respectively.

In addition to the above-mentioned compartments that were incorporated by researchers in their models, Imran et al. (2017) divided the dead/deceased *(D)* compartment in their model into two distinct compartments. Namely, dead but unburied *(F)* and dead and buried *(R_D)*. Furthermore, Berge et al. (2016) incorporated the infection of EVD through a contaminated environment resulting from the continuous existence of the Ebola virus in the surroundings of the deceased Ebola victims in their *SIRPD* model. Where *P* represents Ebola virus pathogens in the environment.

Another piece of research that is relevant to the current research is the work of Shashank et al. (2016). They conducted research on the SITR model for wireless sensor networks. They computed the threshold parameter and discussed the equilibria together with global stability analysis in their model. Their results, indicate that a mathematical model can be employed to analyse the transmission of worms. Their proposed model can be employed to reduce battery overhead and increase wireless sensor network lifetime. Again, Shashank et al. (2016) did not consider the recurrence (relapse) of recovered sensor nodes from malware attacks after the application of antivirus. They denoted as the crash rate of sensor nodes, but they fail to consider the disease-induced death rate in their model.

The above-related works reviewed discuss the transmission of Ebola and how the disease can be controlled. However, we have observed that none of the articles considered the existence of the Ebola virus in survivors' immunologically protected sites in their bodies and their associated recurrence or relapse-symptomatic illness (MacIntyre et al., 2016, Chughtai et al., 2016; Dean et al., 2015; Agusto, 2017). To the best of our knowledge, no one has conducted research on the susceptible, infectious, treatment, and recovered (SITR) model of Ebola virus disease. This research work seeks to fill that gap.

The objective of this research is to model Ebola transmission using the STIR model, taking into account recurrent or relapse illness in Ebola survivors. We assumed that some people who are infected with EVD enter treatment centers through interpersonal interaction with recovered Ebola victims who are still in the treatment compartment. This interaction can cause the transmission of EVD. Furthermore, Ebola patients at their late stage of infection may not be successful at the treatment compartment, so they may proceed to the infectious compartment until they die, since the late stage of Ebola infection is associated with a fatal outcome compared to the early stage of Ebola infection. (Jian T, et al., 2017). These factors have been captured in the current study, along with the reinfection of EVD after successful recovery from the treatment compartment.

The research is arranged in the following manner: The model description and the presumptions are in Section 2. In Section 3, we analys the basic properties of the model, equilibria and stability analysis, the existence of equilibria, and bifurcation analysis. Section 4 is devoted to the discussion of results and conclusions.

2.0 Description of the Model

We used *SITR* model to study the spread of EVD. The entire populace is categorised into four mutually independent subgroups: susceptible *S(t),* infectious *I(t)*, treatment *T(t)* and recovered *R(t)* individuals. We presume that people are introduced into the susceptible class either birth or immigration at the rate ψ and removed at the rate μ. People become infected via contacts with Ebola patients at the effective contact rate α_1. It is assumed that some infectious individuals enter the treatment compartment from interpersonal interaction with recovered Ebola victims at the effective contact rate α_2. η represents a section of recovered victims who become susceptible to the disease again. δ_1 denotes the Ebola related death rate. ϕ represents the recovered rate of Ebola infectious individuals. The infectious Ebola victims enter the treatment class at the rate β and k is the relapse rate of the Ebola infectious individuals who were not successful at the treatment class. Transition between the state variables of the model is displayed in Figure 1 as

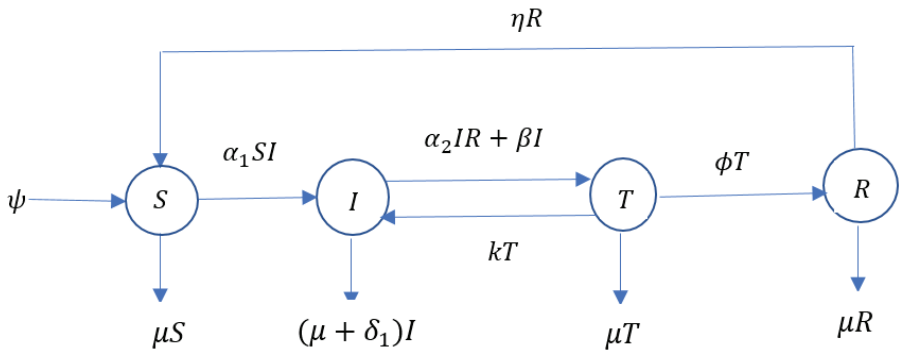


Figure 1: Transitions within the state variables

Table 1: State Variables

Variable	Description
S	Susceptible populace
I	Infectious populace
T	Infectious populace under treatment
R	Recovered populace

Parameter	Description	Value	Range	Source
ψ	Recruitment rate	1.2	[1,3]	(Imran et al., 2017)
α_1	Effective contact rate of susceptible individuals	0.03335	[0.02,0.99]	(Adu et al., 2023)
α_2	Effective contact rate of infectious individuals	0.298	[0.01,0.3]	Assumed
μ	Natural death rate	0.0633	[0.02,0.1]	(Adu et al., 2023)
δ_1	Ebola-related death rate	0.3	[0.01,0.8]	(Adu et al., 2023)
β	Transfer rate of infectious individuals to enter treatment	0.785	[0.3,0.9]	Assumed
η	Immunity loss rate of recovered individuals	0.031	[0.01, 0.08]	Assumed
ϕ	Recovery rate of infectious individuals	0.1667	[0.03, 0.5]	(Chowell et al., 2015)
k	Relapse rate of infectious individuals under treatment	0.009	[0.001, 0.1]	Assumed

Table 2: Model Parameters

The model that governs the presumptions results in the following four(4) nonlinear equation

$$\begin{aligned}
\frac{dS}{dt} &= \psi + \eta R - \alpha_1 SI - \mu S, \\
\frac{dI}{dt} &= \alpha_1 SI + kT - \alpha_2 IR - (\beta + \mu + \delta_1)I, \\
\frac{dT}{dt} &= \alpha_2 IR + \beta I - (\mu + k + \phi)T, \\
\frac{dR}{dt} &= \phi T - (\mu + \eta)R,
\end{aligned} \tag{1}$$

where $S > 0, I \geq 0, T \geq 0, R \geq 0,$

and

$$N(t) = S(t) + I(t) + T(t) + R(t),$$

is the entire populace at time. We then add all equations of model (1) and obtain

$$\frac{dN}{dt} = \psi - \mu N - \delta_1. \qquad (2)$$

3.0 ANALYSIS OF THE EBOLA MODEL

3.1 Basic Properties

Inasmuch as system (1) represents the dynamics of human population, all parameters and variables must not be negative. Using the method employed by (Chowel et al., 2015), we prove the results below:

Theorem 1: *All of the variables in model 1 are non-negative.*

Lemma 1: *The closed set*

$$\Sigma = \left\{(S, I, T, R) \in R_+^4 : \frac{\psi}{\mu + \delta_1} \leq S + I + T + R \leq \frac{\psi}{\mu}\right\},$$

is positively invariant for model (1) and is absorbing.

Proof: Eqn. (2) means that

$$\frac{dN}{dt} \leq \psi - \mu N. \qquad (3)$$

$$\frac{dN}{dt} \geq \psi - (\mu + \delta_1)N. \qquad (4)$$

It follows from equation (3) that

$$N(t) \leq \frac{\psi}{\mu} + \left(N(0) - \frac{\psi}{\mu}\right) e^{-\mu t}, \qquad (5)$$

and from Eqn. (4) we have

$$N(t) \geq \frac{\psi}{\mu+\delta_1} + \left(N(0) - \frac{\psi}{\mu+\delta_1}\right) e^{-(\mu+\delta_1)t}. \qquad (6)$$

If we consider $N(0) > \psi/\mu$, then $dN/dt < 0$ and therefore (based on inequality (5)), $N(t)$ decreases steadily until reaching ψ/μ when $t \longrightarrow \infty$. Similarly, if we consider $N(0) > \psi/(\mu + \delta_1)$, then $dN/dt > 0$ and therefore (based on inequality (6)), $N(t)$ rises steadily until it reaches a maximum at $\psi/(\mu + \delta_1)$ when $t \longrightarrow \infty$. Next, we investigate the case if $N(0)$ lies in the phase between $\psi/(\mu + \delta_1)$ and ψ/μ . We now combine the two inequalities (5) and (6) and obtain

$$\frac{\psi}{\mu + \delta_1} + \left(N(0) - \frac{\psi}{\mu + \delta_1}\right) e^{-(\mu+\delta_1)t} \leq N(t) \leq \frac{\psi}{\mu} + \left(N(0) - \frac{\psi}{\mu}\right) e^{-\mu t},$$

after taking the limit when t approaches ∞, we find that *N(t)* stays within the same phase. Thus, the set Σ is positively invariant and absorbing.

3.2 Ebola-Free Equilibrium and Reproduction Number

The Ebola-free equilibrium of model (1) is given by $E_0 = \left(\frac{\psi}{\mu}, 0,0,0\right)$. Following the approach used by (van den Driesshe & Watmough, 2002) for model (1), The matrices F and V are, respectively, for the new infection terms and the residual transfer terms and are given by

$$F = \begin{bmatrix} \alpha_1 \frac{\psi}{\mu} & k \\ 0 & 0 \end{bmatrix} \text{ and } \quad V = \begin{bmatrix} \beta + \mu + \delta_1 & 0 \\ -\beta & \mu + k + \phi \end{bmatrix}. \tag{7}$$

The basic reproduction number R_0 is defined to be denoted by $\rho(FV^{-1})$. Thus,

$$R_0 = \rho(FV^{-1}) = \frac{\alpha_1 \psi}{\mu(\beta+\mu+\delta_1)} + \frac{\beta k}{(\beta+\mu+\delta_1)(\mu+k+\phi)}. \tag{8}$$

The R_0 refers to the anticipated number of subsequent infections that an Ebola patient will produce in a community that is completely susceptible during his infectious period (van den Driessche & Watmough, 2002). Epidemiologically, R0 determines the intensity of an outbreak. R0 value below 1 indicates an end to an outbreak of a disease, while an R0 value greater than 1 suggests potential epidemics. Whenever the reproduction number reduces below 1 as a result of efforts like vaccination, social isolation, or quarantine measures, it indicates that the epidemic is being contained. For the planning of resources and public health initiatives to reduce the effects of the outbreak, this information is essential.

3.3 Analysis of Ebola-Free Equilibrium

We prove the local stability of E_0 in this subsection. We also present the following lowing results:

Theorem 2: *The Ebola-free equilibrium of model (1), is locally asymptotically stable if* $R_0 < 1$ *and* $\alpha_1 \frac{\psi}{\mu} < (\beta + \mu + \delta_1) + (\mu + k + \phi)$ *and unstable if* $R_0 < 1$

Proof: The associated Jacobian matrix of the model (1) at $E_0 = \left(\frac{\psi}{\mu}, 0,0,0\right)$ is given by

$$J(E_0) = \begin{bmatrix} -\mu & -\alpha_1 \frac{\psi}{\mu} & 0 & \eta \\ 0 & \alpha_1 \frac{\psi}{\mu} - (\beta + \mu + \delta_1) & k & 0 \\ 0 & \beta & -(\mu + k + \phi) & 0 \\ 0 & 0 & \phi & -\mu - \eta \end{bmatrix}. \tag{9}$$

The characteristic equation of model (1) at E_0 is of the form

$$(\lambda + \mu)(\lambda + \eta + \mu)[\lambda^2 + \left((\beta + \mu + \delta_1) + (\mu + k + \phi) - \alpha_1 \frac{\psi}{\mu}\right)\lambda + (\beta + \mu + \delta_1) \times (\mu + k + \phi)(1 - R_0)]. \tag{10}$$

It can be seen that Equation (10) has two negative roots $\lambda_1 = -\mu, \lambda_2 = -\mu - \eta,$ The other roots λ_3 and λ_4 of equation (10) can be determined using the following equation

$$\lambda^2 + \left((\beta+\mu+\delta_1)+(\mu+k+\phi)-\alpha_1\frac{\psi}{\mu}\right)\lambda + (\beta+\mu+\delta_1)(\mu+k+\phi)(1-R_0) = 0\,, \quad (11)$$

$$\lambda_3\lambda_4 = (\beta+\mu+\delta_1)(\mu+k+\phi)(1-R_0),$$

$$\lambda_3+\lambda_4 = \alpha_1\frac{\psi}{\mu} - (\beta+\mu+\delta_1) - (\mu+k+\phi).$$

If $R_0 < 1$ Equation (11) has one positive real root. Hence, E_0, is unstable. If $R_0 < 1$ and $\alpha_1\frac{\psi}{\mu} < (\beta+\mu+\delta_1)+(\mu+k+\phi)$, then $\lambda_3\lambda_4 > 0$ and $\lambda_3+\lambda_4 < 0$. Hence, the characteristic roots of equation $R_0 < 1$.

3.4 Global Analysis of Ebola-Free Equilibrium

Theorem 2 implies that Ebola can be eliminated when $R_0 < 1$ if the initial sizes of the sub-populations of the model are in the basin of attraction of E_0. In other to ensure that Ebola eradication does not depend on the initial sizes of the sub-populations of the model, we study the global stability of Ebola-free equilibrium E_0 as follows.

E_0

$\Sigma \quad R_0 < 1$

The Lyapunov function

$L = g_1 I + g_2 T.$

where

$g_1 = \mu + k + \phi$ and $g_2 = k.$

The derivative of is given by

$$\frac{dL}{dt} = g_1 I' + g_2 T'.$$

$$\frac{dL}{dt} = (\mu+k+\phi)I' + kT'.$$

$$\frac{dL}{dt} = (\mu+k+\phi)[\alpha_1 SI + kT - \alpha_2 IR - (\beta+\mu+\delta_1)I] + k[\alpha_2 IR + \beta I - (\mu+k+\phi)T],$$

$$= [(\mu+k+\phi)\alpha_1 S + \left(k\beta - (\beta+\mu+\delta_1)(\mu+k+\phi)\right) + (k-(\mu+k+\phi))\alpha_2 R]I,$$

$$= (\beta+\mu+\delta_1)(\mu+k+\phi)\left(\frac{\alpha_1\psi}{\mu(\beta+\mu+\delta_1)} + \frac{\beta k}{(\beta+\mu+\delta_1)(\mu+k+\phi)} - 1\right) - \alpha_2 R(\mu+\phi)I,$$

$$= (\beta+\mu+\delta_1)(\mu+k+\phi)(R_0-1) - \alpha_2 R(\mu+\phi)I.$$

Thus, $\frac{dL}{dt} < 0$ whenever $R_0 < 1$ and $\frac{dL}{dt} < 0$, if and $I = 0$. In addition to this, $T \to 0$ as $t \to \infty$ if $I = 0$. We conclude with Lasalle's Invariance Principle (Hale, 1969; Busenberg et al., 1990), that all the solutions to model (1) with initial data in Σ approaches E_0 as $t \to \infty$. This completes the proof.

3.5 Existence of Endemic

Here, we examine the requirements for model (1)'s endemic equilibrium. The endemic equilibrium denoted by $E^* = (S^*, I^*, T^*, R^*)$ is obtained by substituting the derivatives in the left-hand-side of model (1) and equate it to zero. We then solve the associated system of S^*, I^*, T^*, and R^*, we obtain

$$S^* = \frac{\psi(\mu+\eta)(\mu+k+\phi)+\phi I^*(\psi\alpha_2+\eta\beta)}{(\alpha_1 I^*+\mu)[(\mu+\eta)(\mu+k+\phi)-\alpha_2\phi I^*]}, T^* = \frac{\beta(\mu+\eta)I^*}{(\mu+\eta)(\mu+k+\phi)-\alpha_2\phi I^*}, R^* = \frac{\beta\phi I^*}{(\mu+\eta)(\mu+k+\phi)-\alpha_2\phi I^*}. \quad (12)$$

The endemic equilibrium (12) satisfies

$$Q(I^*) = I^*(A_1(I^*)^2 + A_2 I^* + A_3) = 0, \quad (13)$$

$$A_1 = \alpha_1\alpha_2\phi(\mu+\delta_1),$$

$$A_2 = \alpha_2\phi[\psi\alpha_1 + \mu(\mu+\delta_1)] + \alpha_1\eta\phi + \alpha_1(\eta+\mu)[k\beta - (k+\mu+\phi)(\beta+\mu+\delta_1)],$$

$$A_3 = (k+\mu+\phi)(\beta+\mu+\delta_1)(\eta+\mu)(R_0-1).$$

The root $I^* = 0$ of equation (13) corresponds to Ebola-free equilibrium. Thus, we regard the quadratic equation

$$Q(I^*) = A_1(I^*)^2 + A_2 I^* + A_3 = 0, \quad (14)$$

in determining the existence of endemic equilibrium. It should be noted that the positive root of the equation provides the endemic equilibrium (14).

One can easily see that $A_1 > 0$ whether $R_0 > 1$ or not. If , $R_0 > 1$, $A_3 > 0$ and $A_2 < 0$ if when $R_0 > 1$, then the graph of the polynomial (14) indicates that model (1) has one endemic equilibrium. If $R_0 < 1$ and $A_3 < 0$ Then equation (14) has no endemic equilibrium. If $R_0 = 1$, $A_2 > 0$ and $A_3 = 0$ then equation (14) has no positive root. In conclusion, we arrive at the results below.

Theorem 4: *The model (1) has a unique endemic equilibrium if* $A_2 < 0$ *and* $R_0 > 1$, and no *endemic equilibrium when* $R_0 \leq 1$.

3.6 Local Stability of Endemic Equilibrium and Bifurcation Analysis

We examine the possibility of bifurcation and discuss the local stability of endemic equilibrium. The bifurcation phenomenon is established in this section by using the centre manifold theory as explained in Theorem 4.1 by both Carlos Castillo-Chavez and Song (2014) and B. Buonomo and D. Lacitignola (2010) respectively as follows:

We consider the transmission rate of Ebola α_1 as the bifurcation parameter so that $R_0 = 1$ if and only is

$$\alpha_1 = \alpha_1^* = \frac{\mu(\beta+\mu+\delta_1)(\mu+k+\phi) - \beta k\mu}{\psi(\mu+k+\phi)}.$$

Introducing $S = x_1$, $I = x_2$, $T = x_3$, and $R = x_4$, model (1) becomes

$$\frac{dx_1}{dt} = \psi + \eta x_4 - \alpha_1 x_1 x_2 - \mu x_1 := f_1,$$

$$\frac{dx_2}{dt} = \alpha_1 x_1 x_2 + k x_3 - \alpha_2 x_2 x_4 - (\beta + \mu + \delta_1) x_2 := f_2 \qquad (16)$$

$$\frac{dx_3}{dt} = \alpha_2 x_2 x_4 + \beta x_2 - (\mu + k + \phi) x_3 := f_3,$$

$$\frac{dx_4}{dt} = \phi x_3 - (\mu + \eta) x_4 := f_4.$$

We know that the Ebola-free equilibrium is $\left[x_1^* = \frac{\psi}{\mu}, x_2^* = 0, x_3^* = 0, x_4^* = 0\right]$ We linearized the matrix of the model (1) around the Ebola-free equilibrium when $\alpha_1 = \alpha_1^*$ and obtained

$$J(E_+^0) = \begin{bmatrix} -\mu & -\alpha_1^* \frac{\psi}{\mu} & 0 & \eta \\ 0 & \alpha_1^* \frac{\psi}{\mu} - (\beta + \mu + \delta_1) & k & 0 \\ 0 & \beta & -(\mu + k + \phi) & 0 \\ 0 & 0 & \phi & -\mu - \eta \end{bmatrix}. \qquad (17)$$

The matrix $J(E_+^0)$ possesses a simple eigenvalue, with other eigenvalues endowed with negative real parts. Therefore, centre manifold theorem (Castillo-Chavez & Song, 2014) can be applied. We therefore need to derive the values of *a* and *b*. We begin this by calculating the right and left eigenvalues of $J(E_+^0)$ denoted by

$W = [w_1, w_2, w_3, w_4]^T$ and $V = [v_1, v_2, v_3, v_4]$ respectively. We obtain

$$w_1 = \frac{-\psi\alpha_1^*(\mu+k+\phi)(\mu+\eta)+\phi\eta\beta\mu}{\mu^2\beta\phi}, w_2 = \frac{(\mu+k+\phi)(\mu+\eta)}{\beta\phi}, w_3 = \frac{(\mu+\eta)}{\phi}, w_4 = 1,$$

and

$$v_1 = 0, v_2 = \frac{(\mu+k+\phi)}{k}, v_3 = 1 \text{ and } v_4 = 0.$$

Next, we compute the values of *a* and *b*. From model (16), all the associated partial derivatives of $F = (f_1, f_2, f_3, f_4)^T$ in (16) are zero at the Ebola-free equilibrium (DFE) except the following

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_2} = \frac{\partial^2 f_1}{\partial x_2 \partial x_1} = -\alpha_1^*, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = \alpha_1^*, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = -\alpha_2,$$

$$\frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial x_2} = \alpha_2, \quad \frac{\partial^2 f_2}{\partial x_2 \partial \alpha_1^*} = \frac{\psi}{\mu}.$$

Substituting the above equations into a and b in

$$a = \sum_{k,i,j=1}^{n} v_k \omega_i \omega_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$b = \sum_{k,i=1}^{n} v_k \omega_i \frac{\partial^2 f_k}{\partial x_i \partial \emptyset}(0,0).$$

it follows that

$$a = 2v_2 w_1 w_2 \frac{\partial^2 f_2}{\partial x_1 \partial x_2} + 2v_2 w_2 w_4 \frac{\partial^2 f_2}{\partial x_2 \partial x_4} + 2v_3 w_2 w_4 \frac{\partial^2 f_3}{\partial x_2 \partial x_4},$$

$$a = 2v_2 w_1 w_2 \alpha_1^* - 2v_2 w_2 w_4 \alpha_2 + 2v_3 w_2 w_4 \alpha_2,$$

$$a = \frac{2(\mu + k + \phi)^2(\mu + \eta)}{k\beta^2\mu^2\phi^2}\left[\frac{\alpha_2}{(\mu + k + \phi)} - (\psi \alpha_1^{*2}(\mu + k + \phi)(\mu + \eta) + \beta\mu(\alpha_2\mu + \phi\eta)\right] > 0,$$

$$b = v_2 w_2 \frac{\partial^2 f_2}{\partial x_2 \partial \alpha_1^*} + v_3 w_3 \frac{\partial^2 f_2}{\partial x_3 \partial \alpha_1^*},$$

$$b = \frac{\psi(k + \phi + \mu)^2(\mu + \eta)}{\beta\mu k\phi} > 0.$$

It can be seen that $b > 0$. It follows from the results given in (Chen et al., 2016), that the model undergoes backward bifurcation if $a > 0$. The Ebola-Present equilibrium, which exists whenever $R_0 > 1$, is locally asymptotically stable whenever $R_0 > 1$ and $\alpha_1^* < \alpha_1$ with α_1 close to α_1^*. The significance of the backward bifurcation is that it leads to a situation where a stable endemic equilibrium coexists with a disease-free equilibrium. In other words, because of the presence of backward bifurcation in the model, even if $R_0 > 1$ (indicating disease extinction), the disease might not disappear right away since it still has an unstable endemic state that can be stabilized by particular variables. This can help the disease control authorities to know that reducing R_0 below 1 is not sufficient for controlling the epidemic. Further disease elimination strategies must be considered in order to control the epidemic. We arrived at the results below.

Theorem 6: *The unique endemic equilibrium of model (1) is locally asymptotically stable when* $R_0 > 1$.

5.1.4 Sensitivity Analysis of the basic reproduction number

We study the impact of the model parameters on the in the current subsection. Given that data collection and estimated values are typically subject to uncertainty, sensitivity analysis is frequently performed to assess how robust model predictions are to parameter values (Chitnis et al., 2008). We are concerned with the parameter values that have a considerable impact on reproduction ratio, since these are the factors that should be considered when developing preventive measures.As a result, the fundamental reproduction number is a differentiable function of the input parameters. Partial derivatives can also be used for calcu-

lating sensitivity indices. For example, the normalized forward of index with respect to μ using the values in Table 2.

In Table 3, the specific sensitivity indices of R_0 according to the examination of other model parameters are shown.

Table 3. Sensitivity analysis of the R_0

Number	Parameter	Index
1	ψ	+0.95533
2	α_1	+0.95533
3	k	+0.04299
4	β	+0.66722
5	δ_1	+2.66283
6	μ	−0.99370
7	ϕ	−0.00745

While parameters with positive signs rise, R_0 values rise, those with negative signs lower R_0 value as their values rise. The parameters with the most sensitive indexes in this model are δ_1, ψ, α_1 and μ. For example raising (lowering) the value of by 10% raising (lowering) the value of R_0 by 26.6283%. Similarly, raising (lowering) the value of δ_1 by 10% increasing (decreasing) the value of R_0 by 9.5533%. Also, raising (lowering) the value of α_1 and μ by 10% increasing (decreasing) the value of R_0 by 9.5533% and 9.9370% respectively.

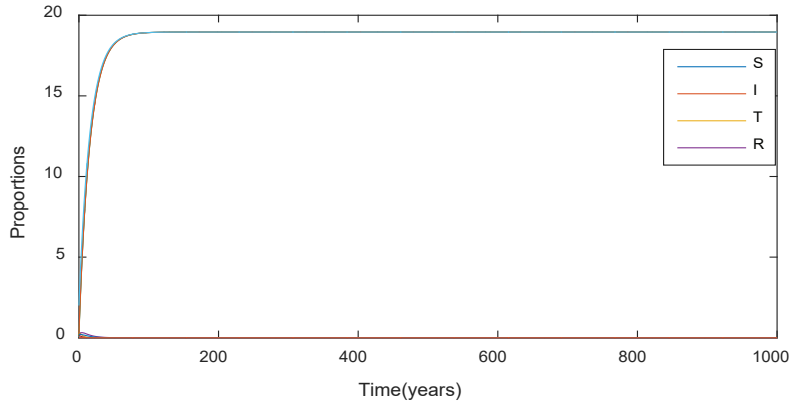
4.0 DISCUSSION AND CONCLUSIONS

We introduced a *SITR* epidemiological model capturing the dynamics of Ebola transmission with relapse and reinfection. We assumed in the full model that some Ebola infectious individuals enter the treatment class from interpersonal interactions with recovered Ebola victims. This allows some recovered individuals to be recruited into the infections class. We also assumed that this interaction can also results in recruiting infectious individuals who have recovered from Ebola and still in the treatment class into the infections class. Ebola infectious individuals at their late stage of infection are assumed to be transferred to the infectious class in this model since treating Ebola at its late stage of infection is difficult. We used MATLAB software to carry out the numerical simulations based on data from some already published works and assumption because we did not get access to accurate data for our model in related literature.

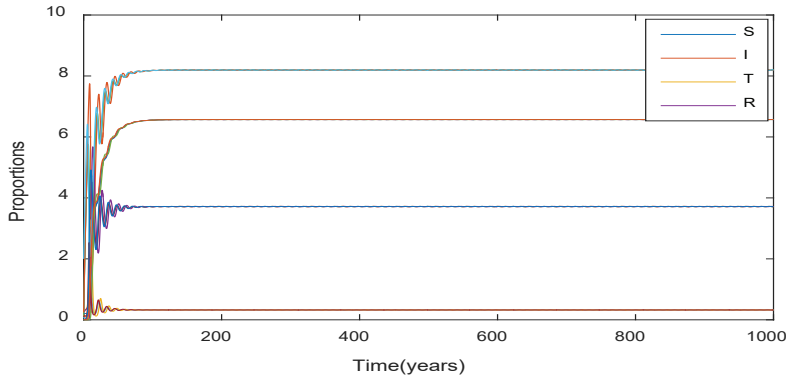
We calculated the R_0, analysed the stability of Ebola-free and endemic equilibria and performed the sensitivity analysis of R_0. We also discuss the global stability of Ebola-free equilibrium. Furthermore, we identified the factors that could lead to bifurcation. Our further analysis indicates that our model exhibits backward bifurcation since $a = 21770.99903119366 \times 10^4 > 0$ and $b = 86.703626864005471 > 0$. This indicates that reducing R_0 below 1 is not enough to control Ebola infection. Other disease control strategies need to be employed. Using the values in Table 1, the $R_0 < 1$. This indicates the absence of Ebola virus disease (EVD) and that only the susceptible are present in the populace. This also implies that the model is locally asymptotically stable if $R_0 < 1$ at Ebola-free equilibrium. This has been confirmed numerically in Fig 2(a). Upon increasing the value

of α_1 from 0.03335 to 0.3335, $R_0 > 1$. This indicates situation in which all susceptible individuals, infectious individuals, individuals in treatment and recovered individuals coexist in the population. This situation indicates the presence of EVD in the population. Ebola infectious individuals will continue to spread the infection to other susceptible individuals in the population and the Ebola-free equilibrium becomes unstable at $R_0 > 1$. This is concurrent with our analytic solution which has been confirmed numerically with Fig 2(b). The reproduction number (R_0) is directly proportional to parameters α_1 and ψ and according to our sensitivity analysis, whenever the value of any of these parameters is increased R_0 increases accordingly. Also decreasing the value of any of these parameters decreases the value of R_0. μ and δ_1 are inversely proportional to the R_0 and that increasing the value of any of them reduces the value of R_0 but decreasing any of them increase the value of R_0.

The impact of β, ϕ and α_1 was investigated and found that the value of R_0 decreases whenever either β or ϕ increases. This has been shown in Fig. 3(a), Fig. (3b), Fig. 4(a) and Fig. 4(b) respectively. This implies that Ebola can be controlled if either more infectious individuals are encouraged to go for treatment or very effective treatment is given at the Ebola treatment centres. These would help more infectious people under treatment to recover from Ebola.

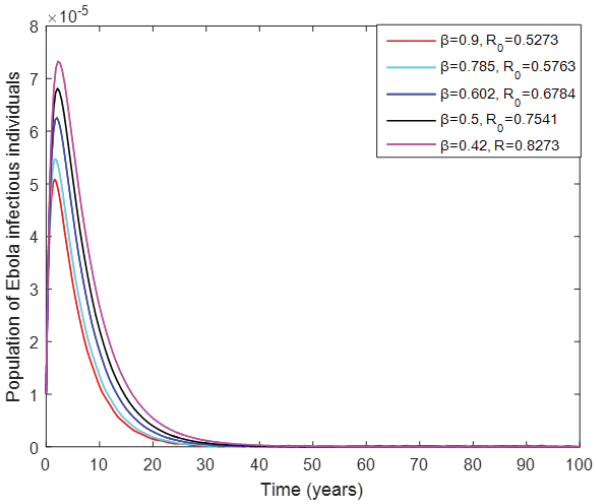


(a)

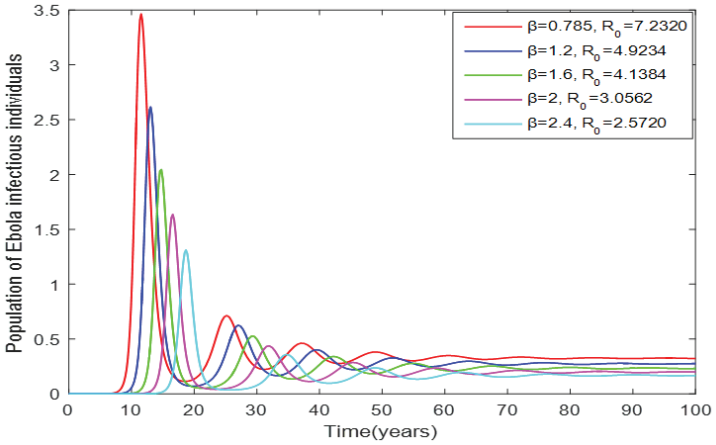


(b)

Figure 2: Time series plot of the model (1) illustrating (a) $R_0 = 0.5763$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.03335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298$, $\beta = 0.785$, $\delta_1 = 0.3, \phi = 0.1667$ and (b) $R_0 = 7.2320$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.3335$,$\mu = 0.0633$,$k = 0.009$,$\alpha_2 = 0.298$,$\beta = 0.785$,$\delta_1 = 0.03, \phi = 0.1667$.

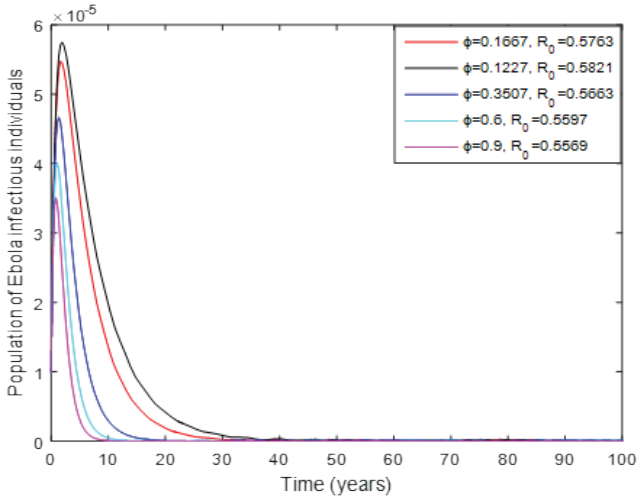


(a)

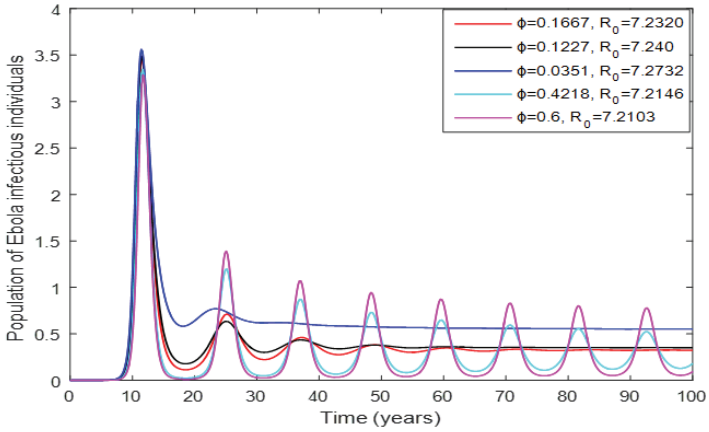


(b)

Figure 3: Time series plot of the system (1) illustrating (a) the effect of β, when $R_0 < 1$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.03335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298, \beta = 0.785, \delta_1 = 0.3, \phi = 0.1667$ and (b) the effect of β, when $R_0 > 1$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.3335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298$, $\beta = 0.785, \delta_1 = 0.03, \phi = 0.1667$.

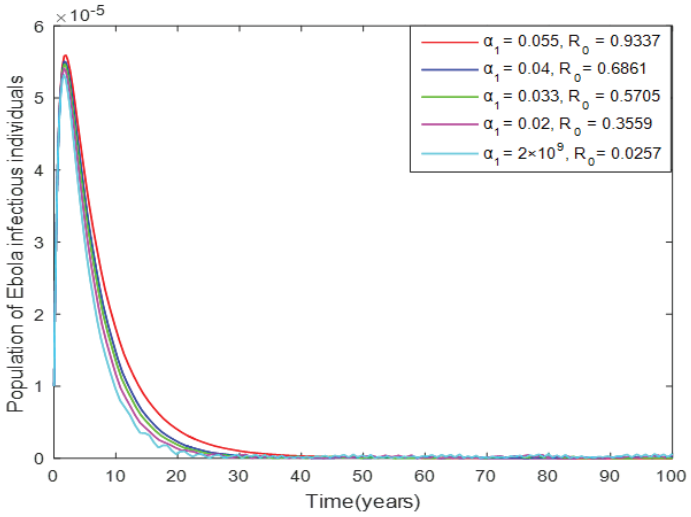


(a)

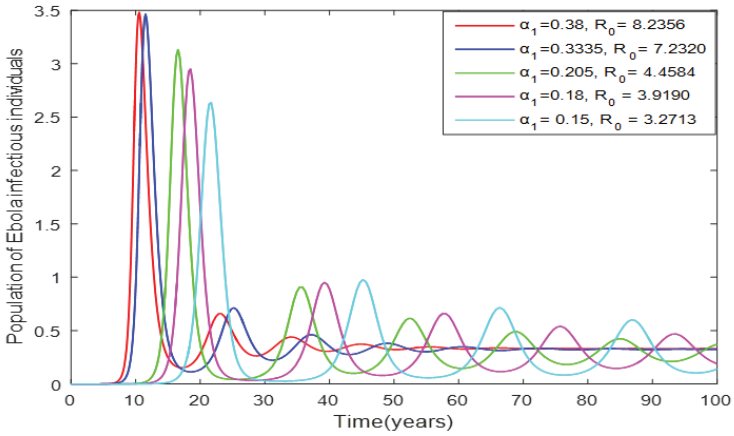


(b)

Figure 4: Time series plot of the system (1) illustrating (a) the effect of ϕ, when $R_0 < 1$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.03335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298, \beta = 0.785, \delta_1 = 0.3, \phi = 0.1667$ and (b) the effect of ϕ, when $R_0 > 1$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.3335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298$, $\beta = 0.785, \delta_1 = 0.03, \phi = 0.1667$.



(a)



(b)

Figure 5: Time series plot of the system (1) illustrating (a) the effect of α_1, when $R_0 < 1$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.03335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298, \beta = 0.785, \delta_1 = 0.3, \phi = 0.1667$ and (b) the effect of α_1, when $R_0 > 1$ with parameter values $\psi = 1.2$, $\eta = 0.031$, $\alpha_1 = 0.3335$, $\mu = 0.0633$, $k = 0.009$, $\alpha_2 = 0.298$, $\beta = 0.785, \delta_1 = 0.03, \phi = 0.1667$.

Also, the value of R_0 increases whenever α_1 increases and R_0 decreases whenever α_1 decreases. This has been shown numerically in Fig. 5(a) and Fig. 5(b). This implies that Ebola disease can be eradicated if R_0 is decreased to a value below unity. Effective public education and campaigns among individuals in the community to prevent with infected people must therefore be taken into consideration in order to eradicate the disease. Finally, our model shows that, EVD can be prevented by increasing the infectious people that enter treatment and also by lowering the effective contact rate of the susceptible through intensive educational campaign and vaccination of the susceptible.

There are several limitations to this research that we hope to overcome in future work. The memory effect is a crucial characteristic of biological systems. The use of fractional order models allowed for the realisation of this; however, the deterministic approach that was employed in this research was unable to do that. We also used some parameter values from existing literature, and others were chosen based on assumptions for our analysis in this research. Real data can be used to conduct similar research.

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