Modeling Assumptions, Mathematical Analysis and Mitigation Through Intervention: An Application to Ebola Type Infectious Disease

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Abstract

Ebola virus is a life-threatening virus and has two major characteristics; one potential to have high mortality rate and the other infection transmission through newly infected dead bodies. There are some relevant features of Ebola that were observed during its recent outbreak: including varying rate of access to isolation facilities by patients and transmission of infection via improper handling of the dead bodies of infected diseased. Quick and safe burial may play an important role in the control and prevention of this virus. In this study, we consider mathematical modeling framework with four different cases for dynamics of Ebola virus with safe and unsafe burial practices, vaccination and treatment interventions with varying efficiency. The goal of this study is to show how timely treatment to Ebola leads to an effective control of the virus and, most importantly, how safe burial of dead bodies helps control the spread.

1 Introduction

Ebola virus is one of the virulent viruses that frequently emerges in both developing and developed nations and is a matter of serious concern for the entire world because of its high fatality ratio (WHO, 2014a; Summers et al., 2015). One of the transmissions of this disease is through body fluids of infected or dead humans or animals. Also, for this deadly virus disease, no effective drug has yet been discovered. Many researchers and biologists have conducted studies to understand the transmission dynamics and control of the disease but still not much is understood as to how to control the spread of this disease effectively. As per available literature, first case of Ebola virus (EV) outbreaks with documentation evidence detected in 1976 (Rathore et al., 2014). Hemorrhagic fever, normally known as Ebola virus disease (EVD), is taken to be the case that commenced in the mid-seventies in Guinea (Feldmann and Geisbert, 2011; Feldmann et al., 2003; Kortepeter et al., 2011). Due to this contingency, 3000 conditions and 1500 deceases were sustained in Guinea, Liberia, Nigeria and Sierra Leone. Considering these inventive conditions, there were more or less 20 major episodes through the year 2014 (CDC, 2014). The latest events which started in Guinea at the early 2014 and after that spread to Liberia and Sierra Leone, is the longest, largest and most widespread Ebola event. Therefore on August 8, 2014, WHO announced the epidemic to be a Public Health Emergency of International Concern (PHEIC) (WHO, 2014b). In accordance with the WHO report, as of Nov. 2, 2014, a total of 13,042 suspected cases and 4,818 affirmed deaths were registered. Amongst these dying, 4,791 (approx. 99.5%) were diagnosed in West African countries (CDC, 2014). The new broad expanded conflicts of EVD in Western Africa was due to Bundibugyo Ebola virus and Sudan Ebola virus, particularly the present conflict of 2014 is an attribute to the Zaire species (Du Toit, 2014). Ebola virus normally gets transmitted into the human population through close contact with blood, secretions, organs or other body fluids of infected animals such as chimpanzees, gorillas, fruit-bats, monkeys, forest antelope and porcupines found ill or dead in the rainforest. Countries like West Africa and South Asia are at high risk of Ebola virus. The main symptoms of Ebola virus such as fever fatigue muscular pain, vomiting, diarrhoea, can be seen within four to six days after a person becomes infected with this harmful disease. Due to non availability of antiviral drug for this deadly virus it is difficult to control the spread of this disease. However, early stage treatment as the symptoms of virus appears in human, basic interventions can significantly improve the chances of survival which include:

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giving more fluid and electrolytes through injection, supplying oxygen to balance the oxygen level and appropriate medicine
like ZMapp and rVSV-ZEBOV for early treatment. In a recent experiment, it has been found that nucleoside analogue- and
siRNA-based therapies are very much effective if a therapy with a > 50% controlling rate is administrated within a few days
post-symptom-onset (Martyushev et al., 2016). Effective drugs play very a vital role in controlling the dynamics of Ebola virus.
It has been worth mentioning that many times the symptoms of Ebola infection are mistaken by other disease like dengue fever
and other infectious diseases. The spread of Ebola virus poses a threat to many countries because the location and identification
of the virus is still unknown (Madubueze et al., 2018).

Over the past years, several mathematical models have been proposed and developed to describe the dynamics of Ebola virus
(EV) (Kabli et al., 2018; Singh et al., 2016; Althaus, 2014; Legrand et al., 2007; Mubayi et al., 2010; Wang and Zhong, 2015;
Rachah and Torres, 2015; 2016, 2017; Chowell et al., 2004; Grigorieva and Khailov, 2014; Area et al., 2015; Jones, 2017; Xiu et al.,
2015; Zhu et al., 2016). Agusto (2017) developed a mathematical model which exhibits that the infection in Ebola virus is
recurrence based. A recent new generalized epizootic model that describes the transmission dynamics of Ebola virus disease in
bat population is main reservoir of the Ebola virus (EL Rhoubari et al., 2018; Jiang et al., 2017; Yusuf and Benyah, 2012; Huo
and Feng, 2013; LaSalle, 1976; Feng and Velasco-Hernández, 1997; Abdelrazec et al., 2016; Singh et al., 2019; Singh and Sharma,
2018; Singh et al., 2018, 2019). Recently, many other mathematical models have been formulated and studied (Ullah et al., 2019;
Khan et al., 2019; Ahmad et al., 2019) to understand the transmission dynamics of many human killing diseases which is still
challenging and are open threat to medical practitioners.

Another aspect of study modelling of epidemics is to find an optimal solution of various problems. Mathematical modelling
regarding the epemics such as zika virus and hepatitis B virus etc. with optimal control solutions have intensively studied by
Wasim et al. (2019) and Ullah et al. (2019). The main contribution of this paper is to study the dynamical behaviour of the
transmission dynamics of Ebola virus in the treatment class which are not mentioned in earlier work. Weitz and Dushoff (2015)
particularly show in their work that reduction in transmission risk after death can have substantial epidemiological benefits.
The identifiability problem relevant to EVD is addressed in his work and shows that improved estimation of post death transmis-
sion would help control the spread of the disease and improve reliability in estimates of reproductive number. In the work of
Nielsen et al. (2015) improvement of burial practices and cemetery management has been addressed to control the spread of
EVD effectively.

In this paper, a mathematical model for the dynamics of Ebola virus with safe burial of disease dead bodies is proposed
and analyzed to know the primary facts and information about the Ebola virus. Ebola virus spreads by touching the bodies of
person who died of ebola disease. To prevent this infection, it is mandatory to make safe burial of the dead bodies (Weitz and
Dushoff, 2015). The safe burial of those who have died of Ebola is acknowledged as an important intervention for controlling
epidemics. Ebola virus spreads mainly through human to human transmission via direct contact with blood or body fluids of
a person who is sick with or has died from Ebola and objects that have been contaminated with body fluids (like blood, feces,
vomit) from a person sick with Ebola or the body of a person who died from Ebola. Health-care workers have frequently been
infect as well as patients suspected or confirmed Ebola virus (EVD). This occurs through close contact with patients
when infection control precautions are not strictly practiced. Safe burial ceremonies that involve direct contact with the body
of the deceased can also contribute in the transmission of Ebola. It has been observed that in absence of medical aid, people,
early diagnosis, isolation of suspected, awareness campaigns and sanitary burial practices are effective controls to stop disease
spread (Dickmann et al., 1990; Chowell et al., 2015; Chowell and Nishiura, 2014; Dhillon et al., 2015; Feng et al., 2000). We
consider four different mathematical models with different rates to understand the dynamics of this deadly virus with safe burial
of its deaths as we consider safe burial as one of the major controlling strategies. In this paper, our primary target is to build a
robust mathematical model to get insight about the impact of recovery rate and study the effect of limited resources on diseases
and how safe burial is important in controlling the diseases. It also provide insights where prevention and control interventions
could be targeted in future.

The organization of this paper is as follows. Section 2 presents the mathematical description of the proposed different model.
Section 3 provides the detailed analysis for all possible cases. Numerical results and simulation are carried out in Section 4.
Section 5 is devoted to discussion. The conclusion is drawn in Section 6.

2 Model Description: Capturing transmission dynamics of Ebola virus

The modeling population is divided into six categories based on infection status (the definition of categories is given in Table 2).
The total human population at time \( t \) is given by, 
\[
N(t) = A(t) + B(t) + C(t) + D(t) + E(t) + F(t),
\]
where susceptible is represented by \( A \), infected classes by \( B, C \) and \( D \), treatment class as \( E \), and removed class (that includes recovered individuals and disease-
related deaths) is represented by \( F \). Susceptible population is increased by the recruitment of individuals into the population
at a rate \( A \), through birth and net migration. The description of other parameters is provided in Table 3. The infected state
is further sub-divided into three classes: asymptomatic class, symptomatic and severely infected with Ebola class. Individuals
can die via natural causes or due to disease. It is further assumed that the treatments are given to symptomatic and severely infected
possible cases are given as: θ is recovery rate of limited health care such as beds in hospitals and non-availability of doctors on the dynamics of Ebola virus transmission, a non-linear as often, beds are shared and many patients are attended by same doctor in hospitals. To understand the impact leading to two sub-models in form of case /one.lf and case /two.lf. The primary assumptions are as follows:

Case 1 (see Figure 1): newly infected individuals individuals are first assumed to be asymptomatic (transition of A only to B), all infected individuals get treated and recover completely which means no death due to disease. B, C, D and E are all infected individuals but only D is assumed to transmit infection. Progression rate from C is β3 to D and γ1 to E.

Case 2 (see Figure 2): D and F individuals are both infectious with infectivity of F is assumed to be reduced by a factor ‘gd’ as compared to D. It is assumed the newly infected individuals either becomes latently infected(B) or become mildly symptomatic(C). The parameter p1 captures the assumption. p2 represents fraction of cases that becomes severe and (1 − p2) access treatment. Compartments D and E include disease deaths. Disease deaths are moved to removed class (R). The dead bodies are assumed to be cremated at the same rate δ. F includes recovered as well as newly dead and fractions of them are capable of infecting others due to culture related rituals.

Case 3 (see Figure 3): Here we assume both D and F individuals can infect. There are two ways individuals can be can be vaccinated (individuals vaccinated at birth are l and at the time of outbreak are ϕ.) Disease deaths are assumed for individuals under treatment.

Case 4 (see Figure 4): Here limited setting of resource is captured by assuming limited number of beds in treatment facilities. Only D individuals can infect and there is no disease related death.

Four sub models are formulated to get insight about the spread of Ebola virus and compare the role of various assumptions on transmission dynamics. In case 1, a simple mathematical model is formulated where we have assumed that the Ebola virus is in initials stage and causes no harm to population. No individual dies because of the virus. In case 2, the virus is assumed to have some effect on population and causing harm to the population. In case of Ebola virus, infection is likely to be spread by dead bodies if they are not buried safely, keeping the same view into consideration, we formulated a sub-model given by case 3. Many papers regarding the burial dead bodies of Ebola virus cases have been available in literature see (Hewlett and Amola, Peters and Peters, 1999). There is evidence that individuals (health-care workers, relatives) may become infected following contacts with patients’ body fluids or direct contact with patients during a visit at the hospital or participation in traditional burial ceremonies. Often during a outbreak of a disease, not all the patients get equal treatment because of non-availability of health care resources like shortage of beds and doctors in the hospitals. We have incorporated θ(b) as a special case of limited health care resources which is also an important aspect to study. In our model we incorporate asymptomatic class which is based on the facts that Ebola virus infection takes 2 to 21 days for its signs and symptoms to appear in human body. In case 2, it is assumed that a fraction of population are removed are dead because of Ebola as dead bodies are also source of infection. In case 3, we introduce vaccination rate and assume all population are getting similar treatment. In case 4, we assume that the population is not getting equal treatment facilities because of low availability of health resources like bed and doctors in hospitals see fig.(1c). Based on this assumption, we assume θ(b) = μ0 + (μ1 − μ0) \( \frac{b}{b+1} \). This type of function is also taken into consideration by Abdelrazec et al. (2016), but the denominator in a linear form where θ(b) = μ0 + (μ1 − μ0) \( \frac{b}{b+1} \). We have considered the denominator of θ(b) a non-linear as often, beds are shared and many patients are attended by same doctor in hospitals. To understand the impact of limited health care such as beds in hospitals and non-availability of doctors on the dynamics of Ebola virus transmission, recovery rate θ is taken as a function of b, where b is limited health care resources. The governing equations for all four different possible cases are given as:

### Table 1: Characteristics of the four different cases of the mathematical model.

<table>
<thead>
<tr>
<th>Model Characteristics</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease deaths in E</td>
<td>×</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Initiation to quick severity</td>
<td>×</td>
<td>✓</td>
<td>✓</td>
<td>×</td>
</tr>
<tr>
<td>Disease death in D</td>
<td>×</td>
<td>✓</td>
<td>✓</td>
<td>×</td>
</tr>
<tr>
<td>Vaccination</td>
<td>×</td>
<td>×</td>
<td>✓</td>
<td>×</td>
</tr>
<tr>
<td>Recovery as bed capacity</td>
<td>×</td>
<td>×</td>
<td>×</td>
<td>✓</td>
</tr>
<tr>
<td>Buried infection</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>×</td>
</tr>
<tr>
<td>Access rate of treatment of mild stage</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>×</td>
</tr>
<tr>
<td>Proportional to progression of mild to severe stage</td>
<td>×</td>
<td>✓</td>
<td>✓</td>
<td>×</td>
</tr>
</tbody>
</table>
Case 1: Model without disease induced death rate ($\delta = 0$)

\[
\begin{align*}
\frac{dA}{dt} &= \Lambda - \frac{\beta_1 A D}{N} + \sigma F - \mu A \\
\frac{dB}{dt} &= \frac{\beta_1 A D}{N} - (\beta_2 + \mu) B \\
\frac{dC}{dt} &= \beta_2 B - (\beta_3 + \mu + \gamma_1) C \\
\frac{dD}{dt} &= \beta_3 C - (\mu + \gamma) D \\
\frac{dE}{dt} &= \gamma_1 C + \gamma D - (\mu + \theta) E \\
\frac{dF}{dt} &= \theta E - (\sigma + \mu) F
\end{align*}
\] (1)

Case 2: Model with disease induced death rate

\[
\begin{align*}
\frac{dA}{dt} &= \Lambda - \frac{\beta_1 A (D + gdF)}{N} + \sigma F - \mu A \\
\frac{dB}{dt} &= \frac{p_1 \beta_1 A (D + gdF)}{N} - (\beta_2 + \mu) B \\
\frac{dC}{dt} &= \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \frac{\beta_1 A (D + gdF)}{N} \\
\frac{dD}{dt} &= p_2 \beta_3 C - (\mu + \gamma + \delta) D \\
\frac{dE}{dt} &= (1 - p_2) \beta_3 C + \gamma D - (\mu + \theta + \delta) E \\
\frac{dF}{dt} &= \theta E - (\sigma + \mu) F + \delta D + \delta E - \delta F
\end{align*}
\] (2)

Case 3: Model with vaccination rate $\varphi$

\[
\begin{align*}
\frac{dA}{dt} &= \Lambda (1 - \varphi) \frac{\beta_1 A (D + gdF)}{N} + \sigma F - \mu A - \varphi A \\
\frac{dB}{dt} &= \frac{p_1 \beta_1 A (D + gdF)}{N} - (\beta_2 + \mu) B \\
\frac{dC}{dt} &= \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \frac{\beta_1 A (D + gdF)}{N} \\
\frac{dD}{dt} &= p_2 \beta_3 C - (\mu + \gamma + \delta) D \\
\frac{dE}{dt} &= (1 - p_2) \beta_3 C + \gamma D - (\mu + \theta + \delta) E \\
\frac{dF}{dt} &= \Lambda + \varphi E - (\sigma + \mu) F + \delta D + \delta E - \delta F + \varphi A
\end{align*}
\] (3)

Case 4: Model with recovery rate as a special function of $\theta(b)$

\[
\begin{align*}
\frac{dA}{dt} &= \Lambda - \frac{\beta_1 A D}{N} + \sigma F - \mu A \\
\frac{dB}{dt} &= \frac{\beta_1 A D}{N} - (\beta_2 + \mu) B \\
\frac{dC}{dt} &= \beta_2 B - (\beta_3 + \mu + \gamma_1) C \\
\frac{dD}{dt} &= \beta_3 C - (\mu + \gamma) D \\
\frac{dE}{dt} &= \gamma_1 C + \gamma D - (\mu + \theta(b)) E \\
\frac{dF}{dt} &= \theta(b) E - (\sigma + \mu) F
\end{align*}
\] (4)

Figure 1: Flow diagram of Ebola virus among different compartments for case 1.
Figure 2: Flow diagram of Ebola virus among different compartments for case 2.

Figure 3: Flow diagram of Ebola virus among different compartments for case 3.

Figure 4: Flow diagram of Ebola virus among different compartments for case 4.
Table 2: Different compartment along with their description used in models in all four cases.

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A(t)$</td>
<td>Number of Susceptible individuals who can get Ebola</td>
</tr>
<tr>
<td>$B(t)$</td>
<td>Number of individuals who are latently infected and are asymptomatic</td>
</tr>
<tr>
<td>$C(t)$</td>
<td>Mildly Symptomatic individuals who are non-infectious and may receive treatment for Ebola</td>
</tr>
<tr>
<td>$D(t)$</td>
<td>Number of severely symptomatic individuals who are infectious</td>
</tr>
<tr>
<td>$E(t)$</td>
<td>Number of Individuals under treatment and are isolated</td>
</tr>
<tr>
<td>$F(t)$</td>
<td>Removed individuals that includes recovered cases disease related deaths (assumed dead bodies may be able to spread infection after death)</td>
</tr>
</tbody>
</table>

Table 3: Parameters and variables used in all models.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Definitions</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Lambda$</td>
<td>per capita recruitment rate of the population (newly recruited individuals are assumed to be susceptible)</td>
<td>0.55 years$^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\gamma_1, \gamma_2$</td>
<td>per capita rate of transition from C and D respectively</td>
<td>0.178</td>
<td>Chowell et al., 2015</td>
</tr>
<tr>
<td>$p_1$</td>
<td>proportion of infected individuals that are asymptomatic</td>
<td>0.35</td>
<td>Assumed</td>
</tr>
<tr>
<td>$p_2$</td>
<td>fraction of mildly symptomatic individuals that become severely infected</td>
<td>0.15</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>transmission coefficient from infected to susceptible</td>
<td>0.03 years$^{-1}$</td>
<td>Chowell et al., 2015</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>per capita progression rate from asymptomatic to mildly symptomatic</td>
<td>0.15 years$^{-1}$</td>
<td>Diekmann et al., 1990</td>
</tr>
<tr>
<td>$\beta_3$</td>
<td>per capita transition rate of patients from mildly symptomatic to severely infected</td>
<td>0.35 years$^{-1}$</td>
<td>Diekmann et al., 1990</td>
</tr>
<tr>
<td>$\theta$</td>
<td>recovery rate per capita</td>
<td>0.03 years$^{-1}$</td>
<td>Diekmann et al., 1990</td>
</tr>
<tr>
<td>$\delta$</td>
<td>diseases induced death rate per capita</td>
<td>0.0001 years$^{-1}$</td>
<td>Diekmann et al., 1990</td>
</tr>
<tr>
<td>$\overline{\delta}$</td>
<td>removal rate of disease deaths per capita</td>
<td>0.0002 years$^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\mu$</td>
<td>natural death rate per capita</td>
<td>0.0196 years$^{-1}$</td>
<td>Diekmann et al., 1990</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>per capita loosing rate of temporary recovery</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>$\varphi$</td>
<td>vaccination rate at older ages</td>
<td>0.4</td>
<td>Assumed</td>
</tr>
<tr>
<td>$g$</td>
<td>fraction of dead bodies that can generate new infection</td>
<td>0.5</td>
<td>Assumed</td>
</tr>
<tr>
<td>$d$</td>
<td>fraction of $g$ that are not safely buried</td>
<td>0.5</td>
<td>Assumed</td>
</tr>
<tr>
<td>$l$</td>
<td>proportion of individuals that are vaccinated at birth</td>
<td>0.4</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\mu_0$</td>
<td>minimum recovery rate per capita</td>
<td>0.1 years$^{-1}$</td>
<td>Carr, 2012</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>maximum recovery rate per capita</td>
<td>10 years$^{-1}$</td>
<td>Carr, 2012</td>
</tr>
<tr>
<td>$b$</td>
<td>fraction of health care resources available</td>
<td>0.20</td>
<td>Carr, 2012</td>
</tr>
</tbody>
</table>
3 The Analysis

Bounded and Invariant Region
To make our system (1)–(4) biologically meaningful, it is necessary to show the positivity and boundedness of the solutions.

Lemma 3.1 The feasible region \( \Omega \) defined by
\[
\Omega = \{ (A(t), B(t), C(t), D(t), E(t), F(t)) \in \mathbb{R}_+^6 : N(t) \leq \frac{A}{p} \}
\]
with initial conditions \( A(t) \geq 0, B(t) \geq 0, C(t) \geq 0, D(t) \geq 0, E(t) \geq 0, F(t) \geq 0 \) is positively invariant for system of equations (1)–(4).

Proof. Adding the equations of system (1)–(4), we obtain
\[
\frac{dN}{dt} \leq \Lambda - \mu N.
\]
Solving this differential equation (5), we have
\[
0 \leq N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t}
\]
where \( N(0) \) represents the initial values of the total population. Thus \( \lim_{t \to +\infty} N(t) \leq \frac{\Lambda}{\mu} \). It implies that the region \( \Omega = \{ (A(t), B(t), C(t), D(t), E(t), F(t)) \in \mathbb{R}_+^6 : N(t) \leq \frac{\Lambda}{\mu} \} \) is a positively invariant set for system (1)–(4). So existence and uniqueness of system (1)–(4) on the region given by set \( \Omega \). □

3.1 Case 1 Analysis
In this section we derive the equilibrium states and investigate their stability by using the reproduction number. Dynamical system (1)–(4) has disease-free equilibrium given by,

\[
E_0 = (A^0, B^0, C^0, D^0, E^0, F^0) = \left( \frac{\Lambda}{p}, 0, 0, 0, 0, 0 \right).
\]
The basic reproduction number, \( R_0 \) is calculated by using the Next Generation Matrix (van den Driessche and Watmough, 2002). Consider the matrices \( \mathcal{F} \) and \( \mathcal{U} \) which are defined due to the spread of new infection and transfer of individuals out of infective compartments, respectively.

Let \( x = (A, B, C, D, E, F)^T \), the system (1) can be written as
\[
\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{U}(x)
\]
where
\[
\mathcal{F}(x) = \begin{bmatrix}
0 \\
\frac{\beta A D}{N} \\
0 \\
0 \\
0 \\
0
\end{bmatrix}
\quad \text{and} \quad
\mathcal{U}(x) = \begin{bmatrix}
\frac{\beta A D}{N} - \sigma F + \mu A - \Lambda \\
(\beta_2 + \mu)B \\
(\beta_3 + \mu + \gamma_1)C - \beta_2 B \\
(\mu + \gamma)D - \beta_3 C \\
(\mu + \delta)E - \gamma_1 C - \gamma D \\
(\sigma + \mu)F - \delta E
\end{bmatrix}.
\]
The associated matrices after taking the partial derivatives of \( \mathcal{F}(x) \) and \( \mathcal{U}(x) \) at \( E_0 = \left( \frac{\Lambda}{p}, 0, 0, 0, 0, 0 \right) \) are
\[
D \mathcal{F}(E_0) = \begin{bmatrix}
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix}
\]
(6)
The reproduction number $R_0$ is obtained by computing largest eigenvalue of matrix, i.e.,

$$R_0 = \rho(WV^{-1}) = \max(|\lambda|, \lambda \in \rho(WV^{-1}))$$

$$= \frac{\beta_2 \beta_1 \beta_3 \Lambda}{\mu (\beta_2 + \mu) (\beta_3 + \mu + \gamma_1)(\mu + \gamma)}.$$

Biological interpretation of $R_0$: Since reproduction number $R_0$ defined as the effective number of secondary infection produced by single infected individual during his tenure of infectiousness. To understand biologically the meaning of $R_0$. We take

$$\psi_0 = \frac{\beta_2}{\beta_2 + \mu}, \quad \psi_1 = \frac{\beta_3}{\beta_3 + \mu + \gamma_1}, \quad \psi_2 = \frac{\beta_1}{\mu}, \quad \psi_3 = \frac{\Lambda}{\mu},$$

where $\psi_0$ shows that fraction of asymptomatic people becoming mildly symptomatic, $\psi_1$ shows that fraction of symptomatic people enter the treatment compartment for getting treatment. $\psi_2$ is the fraction of severely infected individuals entering the treatment compartment for treatment. Since $\mu$ is death rate, therefore $\tau = 1/\mu$ is an average time spent by a susceptible individual in $A(t)$ compartment. Therefore, $\psi_3 = \frac{\Lambda}{\mu}$ gives number of susceptible infected individuals by an infectious individual introduced into a completely susceptible population over its expected lifetime. Also computation of $\frac{dR_0}{d\gamma} < 0$ and $\frac{dR_0}{d\gamma} < 0$ shows $\gamma_1$ and $\gamma$ have negative effect on $R_0$, which indicates as early as symptomatic and severely infected individuals enter treatment class $E(t)$, the $R_0$ can be reduced. Biologically, it infers that early stage treatment can be a useful control strategy.

By the virtue of Theorem 2 of as cited by van den Driessche (2017), we have the following theorem.

**Theorem 3.2** The disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$ is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.

**Theorem 3.3** The disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$ is globally asymptotically stable for $R_0 < 1$ and unstable for otherwise.

**Proof.** The infected compartments of system (1) can be written as

$$\begin{bmatrix} B' \\ C' \\ D' \\ E' \end{bmatrix} = [F - V]\begin{bmatrix} B \\ C \\ D \\ E \end{bmatrix} - [1 - \frac{\Lambda}{\mu}]$$
Thus, we have
\[
\begin{bmatrix}
B' \\
C' \\
D' \\
E'
\end{bmatrix} \leq \begin{bmatrix}
F - V \\
C \\
D \\
E
\end{bmatrix}.
\] (9)

Since all eigenvalues of matrix \( F(x) - \mathcal{V}(x) \) have negative real part, the linearized differential inequality (9) is stable if \( \mathcal{R}_0 < 1 \).

Consequently, \((B, C, D, E) \rightarrow (0, 0, 0, 0)\) as \( t \rightarrow \infty \). Thus, by Comparison theorem, we have \((B, C, D, E) \rightarrow (0, 0, 0, 0)\) and \( A(t) \rightarrow \frac{\lambda}{\mu} \) as \( t \rightarrow \infty \). Hence, the disease-free equilibrium point \( E_0 \) is globally asymptotically stable for \( \mathcal{R}_0 < 1 \). \( \square \)

### 3.1.1 Endemic Equilibrium

Model system (1) has a unique endemic equilibrium point \( E_1 = (A^*, B^*, C^*, D^*, E^*, F^*) \)
\[
A^* = \frac{(b_2 + \mu)N (b_3 + \mu + \gamma_1) (\mu + \gamma)}{\beta_2 ((\mu + \gamma) \gamma_1 + \beta_3)}, \quad B^* = \frac{(b_3 + \mu + \gamma) (\mu + \gamma) (\mu + \gamma)}{\beta_2 (\mu + \gamma)} E^*,
\]
\[
C^* = \frac{(\mu + \gamma) \gamma_1 + \beta_3}{(\mu + \gamma)} E^*, \quad D^* = \frac{(\mu + \gamma) (\mu + \gamma) (\mu + \gamma)}{\beta_3 (\mu + \gamma)^2} E^*, \quad F^* = \frac{\theta}{\sigma + \mu} E^*.
\]

We resolve the above endemic equilibrium points \( E_1 \) to the Centre Manifold theory (Carr, 2012) and investigate the stability of \( E_1 \).

**Lemma 3.4** The system (1) is uniformly persistent on \( \Psi \).

**Proof.** Uniform persistence of system (1) implies that there exists a constant \( \xi > 0 \) such that any solution of (1)–(4) which starts in \( \Psi \), the interior of \( \Psi \) satisfies
\[
\xi \leq \lim \inf_{t \to \infty} A(t), \quad \xi \leq \lim \inf_{t \to \infty} B(t), \quad \xi \leq \lim \inf_{t \to \infty} C(t), \quad \xi \leq \lim \inf_{t \to \infty} D(t), \quad \xi \leq \lim \inf_{t \to \infty} E(t), \quad \xi \leq \lim \inf_{t \to \infty} F(t).
\]
\( \square \)

### 3.1.2 Global Stability of Endemic equilibrium point \( E_1 \)

In this section, we prove the global stability of endemic equilibrium point. Since, recovered class has least impact on the dynamics of system (1)–(4). To prove global stability of endemic equilibrium considers a Liapunov functional \( V \) of following types
\[
V = (A - A^* \ln A) + \phi (B - B^* \ln B) + \psi (C - C^* \ln C) + \xi (D - D^* \ln D) + \eta (E - E^* \ln E)
\]
which is continuous for all \((A, B, C, D, E, F) > 0\) and satisfies \( \frac{\partial V}{\partial A} = (1 - \frac{A^*}{A}), \frac{\partial V}{\partial B} = (1 - \frac{B^*}{B}), \ldots, \frac{\partial V}{\partial F} = (1 - \frac{F^*}{F}) \) by Korobeinikov and Wake (2002), we show that \( V' \leq 0 \).

**Proof.** Since
\[
V = (A - A^* \ln A) + \phi (B - B^* \ln B) + \psi (C - C^* \ln C) + \xi (D - D^* \ln D) + \eta (E - E^* \ln E),
\] (10)
taking derivative of (10) we obtain
\[
V' = A' \left(1 - \frac{A^*}{A}\right) + \phi B' \left(1 - \frac{B^*}{B}\right) + \psi C' \left(1 - \frac{C^*}{C}\right) + \xi D' \left(1 - \frac{D^*}{D}\right) + \eta E' \left(1 - \frac{E^*}{E}\right)
\]
\[
= \left(1 - \frac{A^*}{A}\right) \left( A' - \frac{\beta_1 AD}{N} + \sigma F - \mu A \right) + \frac{B^*}{B} \left( \frac{\beta_1 AD}{N} - (\beta_2 + \mu + \gamma) B \right)
\]
\[
+ \phi \left(1 - \frac{C^*}{C}\right) \left( \beta_2 B - (\beta_3 + \mu + \gamma) C \right) + \xi \left(1 - \frac{D^*}{D}\right) \left( \beta_3 C - (\mu + \gamma) D \right)
\]
\[
+ \eta \left(1 - \frac{E^*}{E}\right) \left( \gamma_1 C + \gamma D - (\mu + \gamma) E \right)
\]
\[
= \left(1 - \frac{A^*}{A}\right) \left( \frac{\beta_1 AD}{N} - \sigma F' + \mu A' - \frac{\beta_1 AD}{N} + \sigma F - \mu A \right)
\]
\[
+ \phi \left(1 - \frac{B^*}{B}\right) \left( \frac{\beta_1 AD}{N} - \frac{\beta_2 AD}{N} B \right) + \psi \left(1 - \frac{C^*}{C}\right) \left( \beta_2 B - \beta_2 B' \right)
\]
\[
+ \xi \left(1 - \frac{D^*}{D}\right) \left( \beta_3 C - \beta_3 C' D \right) + \eta \left(1 - \frac{E^*}{E}\right) \left( \gamma_1 C + \gamma D - \frac{\gamma_1 C' + \gamma D'}{E^*} \right).
\] (11)
Therefore, equation (11) becomes

\[
V' = \left(1 - \frac{1}{x} \right) \left( \frac{\beta_1 A^* D^*}{N} - \frac{\beta_1 A^* D^*}{N} x p - \frac{\beta_1 A^* D^*}{N} B^* y \right) + \phi \left(1 - \frac{1}{y} \right) \left( \frac{\beta_1 A^* D^*}{N} x - \frac{\beta_1 A^* D^*}{N} B^* y \right) \\
+ \psi \left(1 - \frac{1}{z} \right) \left( \phi_2 B^* y - \frac{\beta_2 B^*}{C^*} z \right) + \xi \left(1 - \frac{1}{p} \right) \left( \phi_1 C^* z - \frac{\beta_3 C^*}{D^*} y \right) \\
+ \eta \left(1 - \frac{1}{q} \right) \left( \gamma C^* p + \gamma D^* q - \frac{\gamma C^* + \gamma D^*}{E^*} \right) \\
= -\mu A^* \frac{(1-x)^2}{x} + \frac{\beta_1 A^* D^*}{N} \left(1 - \frac{1}{x} + p\right) + \phi \frac{\beta_1 A^*}{N} \left( px - y - \frac{x}{y} + 1 \right) \\
+ \psi \phi_2 B^* \left( y - z - \frac{z}{p} \right) + \phi_3 C^* \left( z - p - \frac{z}{q} \right) + \eta \gamma D^* \left( p - q - \frac{p}{q} + 1 \right).
\]

On combining terms, we get

\[
V' = -\mu A^* \frac{(1-x)^2}{x} + \frac{\beta_1 A^* D^*}{N} \left(1 - \frac{1}{x} + p\right) + \phi \frac{\beta_1 A^*}{N} \left( px - y - \frac{x}{y} + 1 \right) \\
+ \psi \phi_2 B^* \left( y - z - \frac{z}{p} \right) + \phi_3 C^* \left( z - p - \frac{z}{q} \right) + \eta \gamma D^* \left( p - q - \frac{p}{q} + 1 \right).
\]

The variable terms those appear in \( V' \) with positive coefficients are \( x p, y, z \) and \( p \). If the total of these coefficients is positive, then there is a strong possibility that \( V' \) is positive. Equating the coefficients of \( x p, y, z \) and \( p \) to zero, we have

\[
\begin{align*}
\frac{\beta_1 A^* D^*}{N} - \phi \frac{\beta_1 A^*}{N} &= 0, \\
\phi \frac{\beta_1 A^* D^*}{N} - \phi_2 B^* &= 0, \\
\psi \phi_2 B^* - \phi_3 C^* &= 0, \\
\phi_3 C^* - \phi_2 B^* &= 0, \\
\frac{\beta_1 A^* D^*}{N} - \phi_3 C^* &= 0.
\end{align*}
\]

From set of equations (13), we have \( \phi = 1, \psi = \frac{\beta_1 A^*}{\beta_2 B^*}, \xi = \frac{\beta_1 A^* D^*}{\beta_2 B^*} \). On substituting the values of all above parameters in equation 12

\[
V' = -\mu A^* \frac{(1-x)^2}{x} + \frac{2 \beta_1 A^* D^*}{N} \left( 2 - \frac{z}{p} - \frac{1}{x} \right) + \beta_1 A^* + \phi_1 A^* \left( x - \frac{y}{z} \right) + \eta \gamma D^* \left( 1 - \frac{z}{p} \right).
\]

Since the arithmetic mean is greater than or equal to the geometrical mean, than \( 2 - \frac{z}{p} - \frac{1}{x} \leq 0 \) for \( x, z, p > 0 \) and \( 2 - \frac{z}{p} - \frac{1}{x} = 0 \) iff \( z = p = x = 1 \). Also \( \frac{y}{z} - \frac{y}{z} = 0 \) for \( x = y = z = 1 \). Therefore, \( V' \leq 0 \) for \( x, y, z, p, q > 0 \) and \( V' = 0 \) for \( z = p = x = 1 \) and \( q = \frac{1}{z} \).

The maximum invariant set of system (1) on the set \( \{(x, y, z, p, q) : V' = 0\} \) is the singleton \( \{1,1,1,1,1\} \). Thus, for dynamical system (1), the endemic equilibrium is globally asymptotically stable by LaSalle invariant principal (LaSalle, 1976).

\[\square\]

### 3.2 Case 2 Analysis

In this section, we compute the existence of critical points of mathematical model with case 2 which has disease-free equilibrium given by \( E^* = (A^*, B^*, C^*, D^*, E^*) = (\frac{A^*}{p}, 0, 0, 0, 0) \).
3.2.1 Existence of Endemic Equilibria and its Stability Analysis

The eigenvalues of variational matrix corresponding to disease-free equilibrium are \(-\mu, -(\beta_2 + \mu), -(\beta_3 + \mu), -(\mu + \gamma + \delta), -(\mu + \theta + \delta), -(\sigma + \mu - \delta)\). Since all the eigenvalues are negative. Thus solution clearly decays to zero exponentially and the disease-free equilibrium point is not only stable but also asymptotically stable.

3.2.2 Stability of Endemic Equilibrium Point

Model system case (2) has a unique endemic equilibrium point \(E_2 = (A^*, B^*, C^*, D^*, E^*, F^*)\)

\[
A^* = \frac{N}{\beta_1 \left(\frac{p_2 \beta_2}{\mu + \theta + \gamma} - gd(F^*)\right)} \left\{\Lambda - \sigma \frac{1}{(\sigma + \mu)} \left(\frac{\beta_3 \gamma}{\mu + \gamma + \delta} + (\theta + \delta - \delta) \frac{(\mu + \gamma + \delta)(1 - p_2) \beta_3 + \gamma p_2 \beta_3}{(\mu + \gamma + \delta)(\mu + \theta + \delta)}\right)\right\} C^*;
\]

\[B^* = \frac{p_1 \beta_1}{(\beta_2 + \mu)} A^*(D + gdF^*) N, \quad D^* = \frac{p_2 \beta_2 C^*}{(\mu + \theta + \gamma)}, \quad E^* = \frac{1}{\sigma + \mu} \left(\frac{\gamma D}{\mu + \theta + \gamma}\right) C^*, \]

\[F^* = \frac{1}{\sigma + \mu} \left(\frac{\gamma D}{\mu + \theta + \gamma}\right) C^*.
\]

We resolve the above endemic equilibrium points \(E_2\) to the Centre Manifold theory and investigate their stability of \(E_2\).

The system of equation for model (2) is given below:

\[
\begin{align*}
\frac{dA}{dt} &= \Lambda - \beta_1 A(D + gdF) + \sigma F - \mu A & \frac{dD}{dt} &= p_2 \beta_3 C - (\mu + \gamma + \delta) D \\
\frac{dB}{dt} &= p_1 \beta_1 A(D + gdF) - (\beta_2 + \mu) B & \frac{dE}{dt} &= (1 - p_2) \beta_3 C + \gamma D - (\mu + \theta + \delta) E \\
\frac{dC}{dt} &= \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \beta_3 A(D + gdF) N & \frac{dF}{dt} &= \theta E - (\sigma + \mu) F + 2D + 2E - 2F.
\end{align*}
\]

From above system, it is clear that \(N = \Lambda - \mu N - \delta F\).

Substituting \(A = N - B - C - D - E - F\) in system (2) and omitting the compartment \(A\) from system we get

\[
\begin{align*}
\frac{dB}{dt} &= \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \beta_3 N - \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \beta_3 A(D + gdF) N \\
\frac{dC}{dt} &= \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \beta_3 N - \beta_2 B - (\beta_3 + \mu) C + (1 - p_1) \beta_3 A(D + gdF) N \\
\frac{dD}{dt} &= p_2 \beta_3 C - (\mu + \gamma + \delta) D \\
\frac{dE}{dt} &= (1 - p_2) \beta_3 C + \gamma D - (\mu + \theta + \delta) E \\
\frac{dN}{dt} &= \Lambda - \mu N - \delta E \\
\frac{dF}{dt} &= \theta E - (\sigma + \mu) F + 2D + 2E - 2F.
\end{align*}
\]

Since \(\frac{dN}{dt} \leq \Lambda - \mu N\), thus \(N(t) \leq N(0) \frac{\Lambda}{\mu}\) for sufficiently large \(t\) which ensure that \(A\) is non-negative. Thus the region \(\Gamma = \left\{B, C, D, E, F, N \in R_+^6 : B + C + D + E + F \leq N \leq \frac{\Lambda}{\mu}\right\}\), which is positive invariant set for system (2).
To discuss the stability of endemic equilibrium point of system (2), let us make following linearization $B = B + B^*$, $C = C + C^*$, $D = D + D^*$, $E = E + E^*$, $N = N + N^*$. The linearized system of equation becomes

\[
\frac{d\hat{B}}{dt} = \frac{p_1 \beta_1 (\hat{N} - \hat{B} - \hat{C} - \hat{D} - \hat{E} - \hat{F}) (\hat{D} + g \hat{F})}{N} - (\hat{\beta}_2 + \mu) \hat{B}
\]

\[
\frac{d\hat{C}}{dt} = \beta_2 \hat{B} - (\beta_3 + \mu) \hat{C} + (1 - p_1) \frac{\beta_1 (\hat{N} - \hat{B} - \hat{C} - \hat{D} - \hat{E} - \hat{F}) (\hat{D} + g \hat{F})}{N}
\]

\[
\frac{d\hat{D}}{dt} = p_2 \beta_3 \hat{C} - (\mu + \gamma + \delta) \hat{D}
\]

\[
\frac{d\hat{E}}{dt} = (1 - p_2) \beta_3 \hat{C} + \gamma \hat{D} - (\mu + \delta) \hat{E}
\]

\[
\frac{d\hat{N}}{dt} = \Lambda - \mu \hat{N} - \delta \hat{E}
\]

\[
\frac{d\hat{F}}{dt} = \sigma \hat{E} - (\sigma + \mu) \hat{F} + \delta \hat{D} + \delta \hat{E} - \delta \hat{F}.
\]

Since the last equation in model (2) has least impact on the dynamics of the system, therefore, we omit the last equation and construct the following Lyapunov functional. The Lyapunov function constructed in this section containing $I, m, n, p, q, r$ as constant is given by

\[
V = m \left( B - B^* \ln \frac{B}{B^*} \right) + n \left( C - C^* \ln \frac{C}{C^*} \right) + p \left( D - D^* \ln \frac{D}{D^*} \right) + \frac{q}{2} \left( \frac{(E - E^*)^2}{N} \right)
\]

\[+ r \int_{N^*}^{N} \frac{\beta_2}{t} \left( t - N^* \right) - \left( N^* - B^* - C^* - D^* - E^* \right) \left( \frac{\beta_2}{N} - \frac{\beta_2}{t} \right) \, dt.\]

The derivative of Lyapunov functional is

\[
V' = \frac{B'}{B} \left( B - B^* \right) + n \frac{C'}{C} \left( C - C^* \right) + p \frac{D'}{D} \left( D - D^* \right) + \frac{q}{2} \left( \frac{E - E^*}{N} \right) - \frac{q}{2} \left( \frac{(E - E^*)^2}{N^2} \right)
\]

\[+ r N' \left( \frac{\beta_2}{N} \left( (N - N^*) - (N^* - B^* - C^* - D^* - E^*) \left( \frac{\beta_2}{N} - \frac{\beta_2}{t} \right) \right) \right).\]

From system 15, we see that

\[
V' = m (N^* - B^* - C^* - D^* - E^*) \left( \frac{\beta_2}{N} - \frac{\beta_2}{t} \right) (B - B^*) + n (N^* - B^* - C^* - D^* - E^*) \left( \frac{\beta_2}{N} - \frac{\beta_2}{t} \right) (C - C^*)
\]

\[ - (\mu + \gamma + \delta)(D - D^*) + q (1 - p_2) \beta_2 (C - C^*) + \gamma (D - D^*) - \frac{q}{2} (2 \mu + 2 \delta - 3 \gamma) (E - E^*) \frac{E - E^*}{N}
\]

\[ - \frac{q}{2} \mu (N - N^*) \left( \frac{(E - E^*)^2}{N^2} - r \mu (N - N^*) \right) \left( \frac{\beta_2}{N} \left( (N - N^*) - (N^* - B^* - C^* - D^* - E^*) \left( \frac{\beta_2}{N} - \frac{\beta_2}{t} \right) \right) \right).\]

Since $B \leq N$ and $2 \delta \leq 2 \mu + 2 \delta$. Also $N(t) \leq \frac{\Delta}{\mu}$.

\[
V \leq -\hat{\beta}_2 m \frac{B - B^*}{N} - \eta \mu \frac{C - C^*}{N} - (\mu + \gamma + \delta) \frac{(D - D^*)}{N} - \frac{q}{2} (\mu + \delta + \gamma) \frac{(E - E^*)^2}{N} - r \mu (N - N^*).\]

Thus the function is negative definite. Thus endemic equilibrium is globally asymptotically stable.

### 3.3 Case 3 Analysis

Since this case is a special case of case 2. Therefore, similar analysis is followed for this case.

### 3.4 Case 4 Analyses

In this section, we compute the existence of critical points of mathematical model with case 4 which has disease-free equilibrium given by $E^2 = (A^2, B^2, C^2, D^2, E^2, F^2) = (\frac{\Delta}{p}, 0, 0, 0, 0, 0)$. To ensure the stability of the disease-free equilibrium point, we use next generation method.
Model system (/three.lf) has a unique endemic equilibrium point for Sierra Leone and (/five.lf) (/nine.lf) for Liberia. The details of the proof is in the Appendix.

**Theorem 3.5** The disease-free equilibrium point $E^2$ is locally stable if $\mathcal{R}_1 < 1$ and unstable if $\mathcal{R}_1 > 1$. 

**3.4.1 Existence and stability of Endemic equilibrium point**

Model system (3) has a unique endemic equilibrium point $E = (\tilde{A}, \tilde{B}, \tilde{C}, \tilde{D}, \tilde{E})$

$\tilde{A} = \frac{\Lambda + \sigma \left\{ \frac{\hat{C}}{\gamma + \mu} \right\}}{\hat{B} \left[ \frac{\hat{B} \left( \frac{\hat{B}}{\gamma + \mu} \right) \gamma + \mu}{\sigma + \hat{\vartheta}} \right] + \mu}$, $\tilde{B} = \left( \frac{\beta_3 + \mu + \gamma}{\gamma + \mu} \right) \tilde{C}$, $\tilde{D} = \frac{\hat{D}}{\gamma + \mu}$, $\tilde{E} = \frac{\gamma \left( \chi + \mu \right) + \gamma \hat{B} \hat{E}}{(\mu + \hat{\vartheta}(b))(\mu + \gamma)}$, $\hat{F} = \frac{\hat{B} \hat{E}}{\sigma + \mu}$.

### 4 Numerical Results

In this section, we present numerical simulation of first two dynamical system (1) and (2) by validating the analytical results. We employ Runge-Kutta method of fourth order to simulate the system model (1) and (2) using parameters which are taken from the 2014 West Africa Ebola Outbreak and fit the model to the data which has been reported by the WHO (2014b) as given in Table 3. With recruitment rate $\Lambda = 0.01$, the basic infection reproduction numbers are computed as $R_0 = 1.51$ for Guinea, 2.53 for Sierra Leone and 1.59 for Liberia.

By choosing the following set of parameters $\Lambda = 0.55$, $\gamma_1 = 0.75$, $p_1 = 0.5$, $\beta_1 = 0.46$, $\beta_2 = 0.85$, $\beta_3 = 0.25$, $\mu = 0.008$, $\vartheta = 0.06$, we compute the reproduction number in our case, the results are $R_0 = 1.79$ for Guinea, 2.73 for Sierra Leone and 1.99 for Liberia. It shows that our model is in good agreement with Althaus (2014).

By using above parameters from Table 3 in (1), we compute endemic equilibrium point as $(1102.2, 2, 954.881.95, 2975, 2152.38, 3811.90, 3212.87)$ and the eigenvalues corresponding to endemic equilibrium are as: $-0.3610 \pm 0.6861i$, $-0.0066 \pm 0.0243i$, $-0.1360 \pm 0.0026i$. Since all the eigenvalues have negative real parts, therefore, for the default set parameters $E_1$ is a locally asymptotically stable point. To assess the effects of various parameters on the dynamics of the models, we exhibit graphical representations for the dynamical system as shown in Figures 5–9.
Figure 7: Change of $R_0$, with parameters $\mu$ and $\beta_2$ in (1).

Figure 8: Variation of symptomatic class with time for different values of $\gamma_1 = 0.25, 0.50, 0.75$ in system (1).

Figure 9: Variation of population size with time for different values of $\bar{\delta} = 0.5, 0.7, 0.9$.

Figure 5 exhibits the relationship between reproduction number $R_0$ and recovery rate $\theta$ in (1). When $\theta$ increases the number of individuals getting treatment also increases and as a result the reproduction number decreases which is also biologically relevant. Increasing $\theta$ sufficiently reduces the reproduction number below to 1.

Figure 6 displays the variation of asymptomatic class (1) with transition rate $\beta_1$, which signifies that as the contact rate increases with time, the asymptomatic class grows exponentially.

Figure 7 depicts the relation among reproduction number ($R_0$), disease-induced death rate ($\mu$) and contact rate from asymptomatic stage to symptomatic stage ($\beta_2$) in (1). We observe that both the parameters namely the disease-induced death rate $\mu$ and contact rate affects the reproduction number to large extent. As the values both decrease, the reproduction number also decreases. It indicates that in order to eradicate the disease from environment, we need to control these parameters within a significant level. However, the curve does not asymptotically become zero. It means diseases still prevails in the system.

Figure 8 demonstrates the effect of transition rate $\gamma_1$ on symptomatic class in (1). It has been observed that as we increase the value of $\gamma_1 = 0.25, 0.50, 0.75$, the symptomatic population decreases.

Since, in case of Ebola infection spread, dead bodies play a very crucial role, safe burial of death bodies is necessary to control this deadly virus. Figure 9 depicts the impact of removal of deaths from system which is described in model (2). It is noteworthy that as we increase the value of $\bar{\delta}$, the susceptible population first decreases and then increases afterwards. The infected compartment as well decreases with increase in $\bar{\delta}$. 
5 Discussion

This study aims to simulate the ebola epidemic with safe burial of its deaths taking into consideration of symptomatic and asymptomatic population under four different possible assumptions (four cases) of a mathematical model. In order to get insight of the dynamics of the transmission of ebola virus, model analysis is carried via computation of epidemic threshold quantities, reproduction number $R_0$ ($<& R_1$ for the other cases) and stability of steady states. It is seen that the transitions rates from symptomatic (severed individuals) to individuals under treatment have negative effect on reproduction number as $\frac{\partial R_0}{\partial \gamma} < 0$ and $\frac{\partial R_0}{\partial \beta} < 0$, which indicates that earlier the symptomatic and severed infected individuals enter treatment class, the faster $R_0$ can be reduced. Epidemiologically, it infers that early treatment is critical to controlling the disease when completely safe burial is not possible.

Further, we compare our results of reproduction number (8) with the results of Althaus (2014). In 2014, Althaus developed a predictive SEIR mathematical model on Ebola virus and fitted the model to the data which has been reported from WHO (data is available see WHO (2014b)) of three African countries namely Guinea, Sierra Leone and Liberia. Basic reproduction number in Althaus (2014) is estimated as $R_0 = 1.51$ for Guinea, 2.53 for Sierra Leone and 1.59 for Liberia. We compute the reproduction number for our model with our estimated parameters and found higher but reasonable (in good agreement with Althaus (2014)) values of $R_0 = 1.79$ for Guinea, 2.73 for Sierra Leone and 1.99 for Liberia. This increase in higher estimate can be attributed to finer level of characterisation of our infection stages as seen in epidemiology of a patient.

Moreover, it has been observed that the parameters such as disease-induced death rate and contact rate from asymptomatic stage to symptomatic stage affecting the Reproduction number to larger extent as compared to the demographic and disease-progression related parameters. Our model is based on system of nonlinear ordinary differential equations (ODEs) which is analyzed using bifurcation analysis and provides some useful information on the spread of Ebola virus under the assumption of safe and unsafe burial practices of its deaths. The safe burial of those who have died of Ebola is acknowledged as an important intervention for controlling epidemics. In earlier work of Xia et al. (2015) and Chowell et al. (2015), authors have considered burial aspect but they have not emphatically considered safe burial as safety intervention either they have incorporated vaccination or resource limitation in their model, we have considered safe burial as one of the major controlling strategies as well as vaccination intervention. Our works is different from previous work, as we divide the infection stages into three sub-classes namely: asymptomatic, symptomatic and severed infected with safe burial which is in line with actual situation in case of ebola epidemic. We consider here four cases: (i) transmission of Ebola with safe burial (case 1), (ii) without safe burial and possibility of direct severed infection in some cases (case 2), (iii) role of availability of vaccination intervention in the population (case 3), and (iv) transmission dynamics of Ebola spread when treatment intervention is limited by resources. Our results have identified disease persistence threshold quantity, $R_0$, and endemic levels in each of the four cases. We have classified four distinct scenarios (corresponding to four cases) in terms of efficacy of various interventions (safe burial, unsafe burial, vaccination and treatment efficiency).

There has been many direction in which this work can further be extended by examining the influence of human cultural and movement behaviours on ebola virus transmission. However, it may be difficult to find appropriate data to parameterize such models. As with other diseases, appropriate empirical studies are in need to comprehensively understand dynamics of Ebola with varied practices and interventions in different parts of the world, potential driven by social determinants of health (such as cultures).

6 Conclusion

In this article, a deterministic mathematical model and its four cases for understanding the impact of treatment, vaccination, and burial practices related interventions on transmission dynamic of Ebola virus with limited and unlimited healthcare resources are analysed. The model cases has two equilibrium points namely disease-free and endemic equilibrium. It has shown that the disease-free equilibrium is a locally asymptotically stable when reproduction number is less than unity and endemic equilibrium point is asymptotically stable when reproduction number is greater than unity. Sensitivity analysis is carried out on the model outputs to assess the effects of timely treatment and safe burial dead bodies on control of Ebola. We conclude that re-emergence of infection after effective treatment is a valuable aspect which deserves more attention and intervention strategies to reduce the Ebola virus effect on human population. Further, the treatment efficacy used in treatment of Ebola virus plays a significant role in reduction of dynamics of the virus. Our study has not considered the rates as variable which is more realistic. The future extension of our investigation will be the variables as function of time. We will also incorporate demographic and geographic effects on the dynamics of the Ebola virus.
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Conflict of Interest

The work has no potential conflict of interest.

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### Appendix

**Calculation of reproduction number $R_1$ for case 4**

Now, we have

$$F = \begin{bmatrix} 0 & \beta_1 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} -a_1 & 0 & 0 \\ -b_1 & -b_1 & 0 \\ 0 & -b_3 & -c_1 \end{bmatrix}$$

where

$$a_1 = \beta_2 + \mu, \quad b_1 = \beta_3 + \mu + \gamma, \quad c_1 = \mu + \gamma.$$
We then compute

\[
V^{-1} = \begin{bmatrix}
\frac{1}{\beta_2 + \mu} & 0 & 0 \\
\frac{\beta_2}{(\beta_2 + \mu)(\beta_3 + \mu + \gamma_1)(\mu + \gamma)} & \frac{1}{\beta_3 + \mu + \gamma_1} & 0 \\
\frac{\beta_2 \beta_3}{(\beta_2 + \mu)(\beta_3 + \mu + \gamma_1)(\mu + \gamma)(\mu + \gamma)} & \frac{-\beta_2 \beta_3}{(\beta_3 + \mu + \gamma_1)(\mu + \gamma)(\mu + \gamma)} & 1
\end{bmatrix}
\]

\[
FV^{-1} = \begin{bmatrix}
0 & \beta_1 & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{bmatrix}
\begin{bmatrix}
\frac{1}{\beta_2 + \mu} & 0 & 0 \\
\frac{\beta_2}{(\beta_2 + \mu)(\beta_3 + \mu + \gamma_1)} & \frac{1}{\beta_3 + \mu + \gamma_1} & 0 \\
\frac{\beta_2 \beta_3}{(\beta_2 + \mu)(\beta_3 + \mu + \gamma_1)(\mu + \gamma)(\mu + \gamma)} & \frac{-\beta_2 \beta_3}{(\beta_3 + \mu + \gamma_1)(\mu + \gamma)(\mu + \gamma)} & 1
\end{bmatrix}
\]

\[
= \begin{bmatrix}
\frac{\beta_1 \beta_2}{(\beta_2 + \mu)(\beta_3 + \mu + \gamma_1)} & \frac{\beta_1}{\beta_3 + \mu + \gamma_1} & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{bmatrix}
\]

The reproduction number \( R_1 = \max(\left|\lambda\right|; \lambda \in \sigma(FV^{-1})) \) is spectrum of matrix \( FV^{-1} \). Therefore,

\[
R_1 = \frac{\beta_1 \beta_2}{(\beta_2 + \mu)(\beta_3 + \mu + \gamma_1)}.
\]