

Mathematical Analysis of an Ebola Differential Infectivity Model

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Abstract

West Africa and the Democratic Republic of Congo have in the recent years experienced majority of the Ebola Virus Disease (EVD) burden. Deaths from EVD occur yearly but naturally peak during the dry season. The average EVD fatality rate is approximated at 50%. Studies done on the dynamics of EVD transmission have not captured the differential infectivity aspects of the disease. In this work, a nonlinear differential infectivity model with variation in infectiousness that captures the dynamics of EVD to assess the role of varied infectivity on EVD with possible intervention measures is formulated. Stability analysis of the model shows that the model is conditionally locally and globally stable. The model is also shown to exhibit hopf bifurcation, which shows that the transmission dynamics of the disease are periodic in nature. Numerical simulation results showed that intervention measures to control the disease is more efficient at the onset of the infection to reduce the spread of the disease. The findings are significant in designing intervention measures aimed at reducing infections.

Keywords: Differential Infectivity; Stability Analysis; Ebola Virus Disease

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1 Introduction

Ebola virus disease(EVD) is a zoonotic disease with five different viral strains with majority of the past outbreaks linked to EBOV, SUDV and BDBV [2]. EBOV is the most virulent with a case fatality ranging between (70 – 90%) followed by SUDV (50 – 55%) and BDBV (40 – 48%) [11]. The first outbreaks of EVD occurred in DRC and Sudan simultaneously in 1976, the two outbreaks were caused by two different strains, EBOV in DRC outbreak and SUDV in the Sudan outbreak [12]. EVD outbreaks among humans have been associated with direct human contact with intermediate infected hosts which include; gorillas, chimpanzees and monkeys and transmitted by direct human to human contact via body fluids

or indirect contact with contaminated surfaces. In Dowell [6], individuals become symptomatic after an average incubation period mostly ranging from 2-21 days and infectiousness is increased during the later stages of the disease. Characteristic symptoms of EVD are nonspecific and include sudden onset of fever, weakness, vomiting, diarrhea, headache and a sore throat, while only a fraction of the symptomatic individuals present with haemorrhagic manifestations. The fatality rate of EVD ranges from 30% to 90% depending on the virus species [16]. Treatment against EVD mainly consists of providing medical care based on symptomatic therapy to maintain the vital respiratory, cardio-vascular and renal functions [12].

The use of mathematical models in disease description is instrumental in providing guidance as to the future projections of ongoing public health crises, and in assessing the potential impact interventions might have towards transmission control. For instance, models with differential infectivity bring in a sense of disease progression, where there is a single susceptible compartment and the infectious are to progress through stages. Studies have shown that the viral levels of EVD vary widely between individuals and within the same individual over time. This variation determines the severity of the symptoms and the intervention to be applied. For example, during an Ebola infection, an infected host progresses through initial non-specific symptoms (fever, headache) to gastro-intestinal (diarrhoea, vomiting) and then to either deterioration phase (bleeding) or recovery [9]. Furthermore, individuals with high viral loads have been shown to be more infectious [6]. These transmission characteristics can be well represented using differential infectivity. This variation allows for the prediction of the possible effects of interventions. Other closely related studies can be found in [1, 4, 15].

A study by Harta et al [9] suggests that variations in symptoms during EVD could alter the type of intervention strategy to be applied. This variation may suggest that a small subset of infected people may be responsible for disproportionate number of infections. This study therefore develops a differential infectivity model to study the impact of variations in EVD on the infectiousness of an individual in order to determine the virulence of each infective group and to provide balance between prevention and treatment methods in the control of an EVD outbreak.

2 Model Formulation

In this section, a differential infectivity Ebola model that categorizes the population into five compartments, namely; the Susceptible individuals $S(t)$, the Infected population $I_1(t)$ (individuals with non-specific symptoms), $I_2(t)$ (individuals in the gastro-intestinal phase), $I_3(t)$ (individuals in the deterioration phase) and the Recovered population $R(t)$ is developed. The susceptible population is recruited at the rate Λ , while the population can decrease due to natural deaths at a rate μ . Infected individuals die naturally or as a result of infection at the rate δ . The system of differential equations describing the dynamics of the model is as follows;

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - \frac{\beta S(I_1 + k_1 I_2 + k_2 I_3)(1 - \sigma)}{N} - \mu S \\
 \frac{dI_1}{dt} &= \frac{\beta S(I_1 + k_1 I_2 + k_2 I_3)(1 - \sigma)}{N} - (\mu + \gamma_1) I_1 \\
 \frac{dI_2}{dt} &= \gamma_1 I_1 - (\mu + \delta_1 + \gamma_2 + \omega) I_2 \\
 \frac{dI_3}{dt} &= \gamma_2 I_2 - (\mu + \delta_2 + \alpha) I_3 \\
 \frac{dR}{dt} &= \omega I_2 + \alpha I_3 - \mu R
 \end{aligned} \tag{1}$$

and takes the initial conditions;

$$S(t_0) = S(0), I(t_0) = I(0), R(t_0) = R(0); t_0 = 0 \tag{2}$$

where $k_1 > k_2$ represents the modification parameter of reduced rate of infectivity for classes I_2 and I_3 respectively as a result of reduced morbidity due to the specific symptoms associated to the disease.

3 Qualitative Analysis of the Model

3.1 Positivity and boundedness of Solutions

Since the model describes human population, the state variables of model (1) are assumed to be non-negative and ultimately bounded at all time. Therefore, the model is biologically meaningful and mathematically well posed.

3.2 Basic Reproduction Number

Definition 3.1. *The basic reproduction number (R_0) is the average number of secondary infections due to a single infectious individual introduced in a fully susceptible population over the course of the infectious period. If $R_0 < 1$, it means that on average an infected individual produces less than one new infected individual while $R_0 > 1$ means that each infected individual produces more than one new infection on average.*

The R_0 of model (1) determined by the method of next generation matrix approach [14] is given by:

$$R_0 = \beta \frac{(\mu + \delta_1 + \gamma_2 + \omega)(\mu + \delta_2 + \alpha) + k_1 \gamma_1 (\mu + \delta_2 + \alpha) + k_2 \gamma_1 \gamma_2}{(\mu + \gamma_1)(\mu + \delta_2 + \alpha)(\mu + \delta_1 + \gamma_2 + \omega)}. \tag{3}$$

Which can be expressed as;

$$R_0 = \beta \frac{BC + k_1 \gamma_1 B + k_2 \gamma_1 \gamma_2}{ABC} \tag{4}$$

where; $A = (\mu + \gamma_1)$, $B = (\mu + \delta_2 + \alpha)$, $C = (\mu + \delta_1 + \gamma_2 + \omega)$. This can as well be written as;

$$R_0 = [R_{01} + R_{02} + R_{03}],$$

with terms on the RHS representing the contribution of infections from each of the infective classes I_1, I_2 , and I_3 .

4 Equilibrium Points of the Model

4.1 Disease Free Equilibrium (DFE)

The disease free equilibrium (DFE) is defined as the state at which the disease under study is not present in the population.

Proposition 4.1. *The model in system (1) has a disease free equilibrium given by:*

$$E^0 = (S^0, I_1^0, I_2^0, I_3^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$$

4.2 Existence of the Endemic Equilibrium (EE)

Proposition 4.2. *If $R_0 > 1$, then there exists a unique endemic equilibrium $E^*(S^*, I_1^*, I_2^*, I_3^*, R^*)$.*

Proof. The Endemic equilibrium point (E^*) of equation 1 is found by equating its RHS to 0 and solving for the variables. Thus;

$$\begin{aligned} S^* &= \frac{N}{R_0} \\ I_1^* &= \frac{\Lambda}{\mu + \gamma_1}(R_0 - 1) \\ I_2^* &= \frac{\gamma_1 \Lambda}{(\mu + \gamma_1)(\mu + \delta_1 + \gamma_2 + \omega)}(R_0 - 1) \\ I_3^* &= \frac{\gamma_1 \gamma_2 \Lambda}{(\mu + \gamma_1)(\mu + \delta_1 + \gamma_2 + \omega)(\mu + \delta_2 + \alpha)}(R_0 - 1) \\ R &= N(R_0 - 1) \left(\frac{\omega \gamma_1 - \alpha(\mu + \delta_1 + \gamma_2 + \omega)}{\mu + \delta_1 + \gamma_2 + \omega} \right) \end{aligned} \quad (5)$$

If $R_0 > 1$ then there exists $I_1^* > 0$, $I_2^* > 0$ and $I_3^* > 0$, hence the model has a positive endemic equilibrium. $\square$

5 Stability Analysis of the Model

5.1 Local Stability of the DFE

Proposition 5.1. *For any time $t \geq 0$, the disease free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$ of system (1) is asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.*

Proof.

$$J_{DFE} = \begin{pmatrix} -\mu & -\beta\frac{\Lambda}{\mu} & -\beta k_1\frac{\Lambda}{\mu} & -\beta k_2\frac{\Lambda}{\mu} & 0 \\ 0 & \frac{\beta\Lambda}{\mu} - (\gamma_1 + \mu) & 0 & 0 & 0 \\ 0 & \gamma_1 & -(\mu + \delta_1 + \gamma_2 + \omega) & 0 & 0 \\ 0 & 0 & \gamma_2 & -(\mu + \delta_2 + \alpha) & 0 \\ 0 & 0 & \omega & \alpha & -\mu \end{pmatrix} \quad (6)$$

The eigenvalues of the matrix (6) are;

$$\begin{aligned} \lambda_{1,2} &= -\mu \\ \lambda_3 &= -(\mu + \delta_2 + \alpha) \\ \lambda_4 &= -(\mu + \delta_1 + \gamma_2 + \omega) \\ \lambda_5 &= \frac{\beta\Lambda}{\mu} - (\mu + \gamma_1) \end{aligned} \quad (7)$$

λ_5 is negative whenever

$$\frac{\beta\Lambda}{\mu(\mu + \gamma_1)} < 1$$

which is equivalent to;

$$R_{01} \frac{\Lambda}{\mu} < 1$$

□

For local asymptotic stability, all real parts of λ should be negative. Clearly all roots are negative provided $R_0 < 1$. This implies that when a small number of infected individuals at class I_1 are introduced into the population, after sometime the system returns to the DFE.

5.2 Global Stability of the Disease Free Equilibrium

To study the global stability of the DFE, we use the technique by Castillo Chavez [3], which requires that the system be written in the form:

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z) \\ \frac{dZ}{dt} &= G(X, Z), G(X, 0) = 0 \end{aligned}$$

where $X \in R$ and $X = (S)$ denotes the number of uninfected individuals and $Z \in R$, where $Z = (I_1, I_2, I_3)$ denotes the number of infected individuals. The following condition must be met to guarantee global asymptotic stability.

$$\frac{dX}{dt} = F(X, 0), X^* G.A.S$$

$G(X, Z) = AZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0$ for all $(X, Z) \in \Gamma$ where $A = D_Z G(X^*, Z)$ is an M-matrix and Γ is the region where the model makes biological sense.

Theorem 5.1. *The disease free equilibrium E^0 of a system is GAS if $R_0 < 1$ and unstable if $R_0 > 1$.*

Since R is decoupled from the other equations in equation 1, we study the reduced system given by;

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta S(I_1 + k_1 I_2 + k_2 I_3) - \mu S \\ \frac{dI_1}{dt} &= \beta S(I_1 + k_1 I_2 + k_2 I_3) - (\mu + \gamma_1) I_1 \\ \frac{dI_2}{dt} &= \gamma_1 I_1 - (\mu + \delta_1 + \gamma_2 + \omega) I_2 \\ \frac{dI_3}{dt} &= \gamma_2 I_2 - (\mu + \delta_2 + \alpha) I_3 \end{aligned} \quad (8)$$

Therefore;

$$\frac{dX}{dt} = F(X, Z) = \Lambda - \mu S$$

At $S = S^0$, $F(X, 0) = 0$ and $\frac{dX}{dt} = F(X, 0) = \Lambda - \mu S$ as $t \rightarrow \infty$, $X \rightarrow X^0$, hence $X = X^0 = (S^0)$ is GAS.

$G(X, Z) = AZ - \hat{G}(X, Z) = \text{RHS of equation two, three and four in system 1.}$ Differentiating the equations in the system with respect to (I_1, I_2, I_3) at DFE gives;

$$\begin{pmatrix} \beta S - (\mu + \gamma_1) & \beta k_1 S & \beta k_2 S \\ \gamma_1 & -(\mu + \delta_1 + \gamma_2 + \omega) & 0 \\ 0 & \gamma_1 & -(\mu + \delta_2 + \alpha) \end{pmatrix} \begin{pmatrix} I_1 \\ I_2 \\ I_3 \end{pmatrix} - \begin{pmatrix} \beta S(I_1 + k_1 I_2 + k_2 I_3) - (\mu + \gamma) I_1 \\ \gamma_1 I_1 - (\mu + \delta_1 + \gamma_2 + \omega) I_2 \\ \gamma_2 I_2 - (\mu + \delta_2 + \alpha) I_3 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}$$

$\hat{G}(X, Z) = 0$. Since $\hat{G}(X, Z) \leq 0$, then the DFE of the system is globally asymptotically stable. This means that if the system is perturbed by introducing a large number of infectives in a population at DFE then eventually the system returns to DFE. Epidemiologically, the disease does not spread in the population even if an infective is introduced into the population provided $R_0 < 1$.

5.3 Local Stability of Endemic Equilibrium (EE) point

The Jacobian matrix of equation (1) evaluated at endemic equilibrium is given by;

$$J_{EE} = \begin{pmatrix} -(\frac{\beta\Lambda(R_0-1)}{AN}[1 + \frac{k_1\gamma_1}{B}] + \frac{k_2\gamma_1\gamma_2}{BC} + \mu) & \frac{-\beta}{R_0} & \frac{-\beta k_1}{R_0} & \frac{-\beta k_2}{R_0} & 0 \\ \frac{\beta\Lambda(R_0-1)}{AN}[1 + \frac{k_1\gamma_1}{B}] + \frac{k_2\gamma_1\gamma_2}{BC} & -(\frac{\beta}{R_0} + A) & \frac{\beta k_1}{R_0} & \frac{\beta k_2}{R_0} & 0 \\ 0 & \gamma_1 & -B & 0 & 0 \\ 0 & 0 & \gamma_2 & -C & 0 \\ 0 & 0 & \omega & \alpha & -\mu \end{pmatrix} \quad (9)$$

where

$$A = (\mu + \gamma_1), B = (\mu + \delta_1 + \gamma_2 + \omega), C = (\mu + \delta_2 + \alpha)$$

This can be expressed as;

$$J_{EE} = \begin{pmatrix} -a - \mu & \frac{-\beta}{R_0} & \frac{-\beta k_1}{R_0} & \frac{-\beta k_2}{R_0} & 0 \\ a & -b & \frac{\beta k_1}{R_0} & \frac{\beta k_2}{R_0} & 0 \\ 0 & \gamma_1 & -B & 0 & 0 \\ 0 & 0 & \gamma_2 & -C & 0 \\ 0 & 0 & \omega & \alpha & -\mu \end{pmatrix} \quad (10)$$

where

$$a = \frac{\beta\Lambda(R_0-1)}{AN}[1 + \frac{k_1\gamma_1}{B} + \frac{k_2\gamma_1\gamma_2}{BC}]$$

and

$$b = (\frac{\beta}{R_0} - A)$$

Since the diagonal elements of the J_{EE} matrix (10) are negative, the matrix is said to be stable if it is diagonally dominant in columns.

Set $\phi = \max\{g_1, g_2, g_3, g_4, g_5\}$ where;

$$\begin{aligned} g_1 &= |-a - \mu| > |a| \\ g_2 &= |-b| > |\frac{\beta}{R_0} + \gamma_1| \\ g_3 &= |-(\mu + \delta_1 + \gamma_2 + \omega)| > |-\frac{\beta k_1}{R_0} + \frac{\beta K_1}{R_0} + \gamma_2 + \omega| \\ g_4 &= |-(\mu + \delta_2 + \alpha)| > |-\frac{\beta k_2}{R_0} + \frac{\beta K_2}{R_0} + \alpha| \\ g_5 &= |-\mu| > 0 \end{aligned} \quad (11)$$

which indicates that matrix (10) is diagonally dominant in columns, then by the Gershgorin disc argument [8], the eigenvalues of matrix (10) lie within atleast one Gershgorin disc.

Therefore the endemic equilibrium is locally asymptotically stable whenever $R_0 > 1$. Epidemiologically, if a small number of infected individuals are introduced into a susceptible population, each infected individual will produce more than one infected individual on average in the entire infectious period.

5.4 Global Stability of Endemic Equilibrium (EE) point

In this section, we prove the global stability of the endemic equilibrium E^* when $R_0 > 1$.

Theorem 5.2. *If $R_0 > 1$, then system (1) has a globally asymptotically stable endemic equilibrium E^* .*

Proof. Rewrite the equilibrium equations for $E^* = (S^*, I_1^*, I_2^*, I_3^*)$ as;

$$\begin{aligned}\Lambda &= (\pi + \mu)S^* \\ \pi S^* &= (\mu + \gamma_1 + \alpha_1)I_1^* \\ I_1^* &= \frac{(\mu + \delta_1 + \gamma_2 + \alpha_2)I_2^*}{\gamma_1} \\ I_2^* &= \frac{(\mu + \delta_2 + \alpha_3)I_3^*}{\gamma_2}\end{aligned}$$

where $\pi = \frac{\beta(I_1 + k_1 I_2 + k_2 I_3)}{N}$

Consider the following non-linear Lyapunov function of Volterra type defined by;

$$W = W(x) = (S - S^* - S^* \frac{\ln S}{S^*}) + (I_1 - I_1^* - I_1^* \frac{\ln I_1}{I_1^*}) + (I_2 - I_2^* - I_2^* \frac{\ln I_2}{I_2^*}) + (I_3 - I_3^* - I_3^* \frac{\ln I_3}{I_3^*})$$

where $x^* = E^* = (S^*, I_1^*, I_2^*, I_3^*)$

Computing the derivative of W along solutions of system (1), we obtain;

$$\frac{dW}{dt} = (1 - \frac{S^*}{S})S' + (1 - \frac{I_1^*}{I_1})I_1' + (1 - \frac{I_2^*}{I_2})I_2' + (1 - \frac{I_3^*}{I_3})I_3'$$

Substituting the derivatives (S', I_1', I_2', I_3') from system (1)

$$\begin{aligned}\frac{dW}{dt} &= (1 - \frac{S^*}{S})(\Lambda - \pi S - \mu S) + (1 - \frac{I_1^*}{I_1})(\pi S - (\mu + \gamma_1 + \alpha_1)I_1) + (1 - \frac{I_2^*}{I_2})(\gamma_1 I_1 - (\mu + \delta_1 + \gamma_2 + \alpha_2)I_2) \\ &\quad + (1 - \frac{I_3^*}{I_3})(\gamma_2 I_2 - (\mu + \delta_2 + \alpha_3)I_3) \\ \frac{dW}{dt} &= \mu S^* (2 - \frac{S}{S^*} - \frac{S^*}{S}) + 0 \leq 0\end{aligned}$$

□

Therefore; $\frac{dW}{dt} \leq 0$ only holds at $S = S^*$ and therefore W is a Lyapunov function and it follows by Lasalle's Invariance principle [10] that every solution to the equation of the model (1) approaches the associated endemic equilibria E^* of the model as $t \rightarrow \infty$ for $R_0 > 1$. This implies that if the disease exists in the population, then the disease will persist in the population.

5.5 Existence of Periodic Solutions

Differential equations depend on parameters and the qualitative behaviour of a solution system can be different depending on the change in parameter values. In this section this study seeks to apply a complete mathematical characterization of coefficients criteria [1] for a system of dimensions $n = 4$, that seeks to determine existence or non-existence of Hopf-bifurcation in the system developed. $\lambda_1 = -\mu$ is one of the eigen values of matrix (10) and therefore the reduced $J_{4 \times 4}$ matrix obtained from matrix (10) is given by;

$$J_{4 \times 4} = \begin{pmatrix} -a - \mu & \frac{-\beta}{R_0} & \frac{-\beta k_1}{R_0} & \frac{-\beta k_2}{R_0} \\ a & -b & \frac{\beta k_1}{R_0} & \frac{\beta k_2}{R_0} \\ 0 & \gamma_1 & -B & 0 \\ 0 & 0 & \gamma_2 & -C \end{pmatrix} \quad (12)$$

Theorem 5.3. *A polynomial equation of the form;*

$$\lambda^4 + B_1\lambda^3 + B_2\lambda^2 + B_3\lambda + B_4 = 0 \quad (13)$$

has a pair of pure imaginary roots and two roots with negative real parts if and only if; $B_1 > 0$, $B_3 > 0$, $B_4 > 0$ and $B_1B_2B_3 - B_1^2B_4 - B_3^2 = 0$

Proof. The characteristic equation of the matrix (12) is given by equation (13); where;

$$B_1 = C + B + a + b + \mu$$

$$B_2 = C(B + a + b + \mu) + B(a + b + \mu) + a(b + g) - b\mu - ge\gamma_1$$

$$B_3 = CB(a + b + \mu) + (ab + ag + b\mu)(C + B) + \gamma_1ge(C - \mu) - gf\gamma_1\gamma_2$$

$$B_4 = CB(ab + ag + b\mu) - gm\mu\gamma_1(Ce - \gamma_2f)$$

$$g = \frac{\beta}{R_0}, \quad e = \frac{\beta k_1}{R_0}, \quad f = \frac{\beta k_2}{R_0}$$

It is clear that;

$B_1 > 0$, $B_3 > 0$ whenever $CB(a + b + \mu) + (ab + ag + b\mu)(C + B) + \gamma_1ge(C - \mu) > gf\gamma_1\gamma_2$ and $B_4 > 0$ whenever $CB(ab + ag + b\mu) > gm\mu\gamma_1(Ce - \gamma_2f)$ and $B_1B_2B_3 - B_1^2B_4 - B_3^2 = 0$. Because the conditions in theorem (5.3) are met then model (1) undergoes hopf-bifurcation. $\square$

This hopf-bifurcation property of model (1) is illustrated by simulating the model using a set of parameter values given in the next section. This indicates the reason for the possible recurrence of the disease in the population.

6 Numerical Simulation and Discussion

In this section, model (1) is simulated and discussed to investigate the impact of the different infectious groups during an epidemic in order to determine the appropriate intervention measures.

Table 1: Parameters and initial conditions for DRC

Parameters	Description	Range	Source
N	Population size	230,000	Estimated
β	Infection rate	$1.6 \times 10^{-6} week^{-1}$	[9]
k_1	Mid infection	0.8	Varies
k_2	Late infection	0.9	Estimated
μ	Natural death rate	$[0, 1] day^{-1}$	[9]
δ	Disease induced death rate	$[0.5] day^{-1}$	[9]
α	Recovery rate	0.18(0.16-0.202)	Varies
$\frac{1}{\gamma}$	Incubation period	1 week	[9]

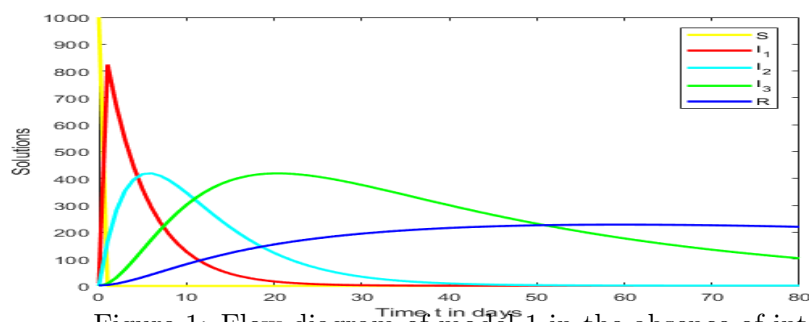


Figure 1: Flow diagram of model 1 in the absence of intervention

Figure (1) represents the behaviour of the infective in the absence of an intervention measure. Curves I_1 , I_2 and I_3 rise above the R curve. This implies that in the absence of intervention there is a high number of infections and lower recoveries. This implies that the disease will become a pandemic. The virulence of an infection can be determined by; the number of infections caused, the severity of the disease or the number of deaths caused by an infection.

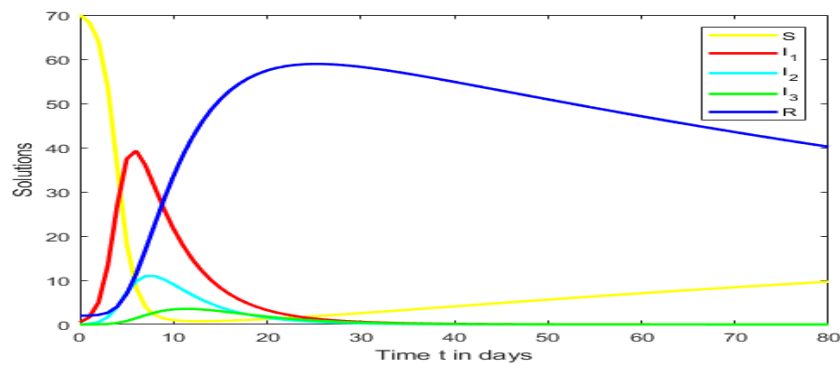


Figure 2: Flow diagram of model 1 in the presence of an intervention

Figure (1) also shows that in the early stages of an epidemic, class I_1 , with non-specific symptoms, appears to be the most virulent as it causes the bulk of the infections. If no intervention measure is applied then the disease progresses to other classes.

The number of infections in class I_1 in figure (2) is lower than that in figure (1). As the epidemic progresses to classes I_2 and I_3 , this groups have a lesser impact to the number of infections as compared to class I_1 as shown from figure (2). This can be attributed to the fact that infected individuals in this two stages have progressed through the infection and have developed more specific and serious symptoms and therefore the susceptible population take precautionary measure to avoid direct interaction. In the presence of an intervention measure such as hand washing, detection and case isolation, the number of infections are reduced.

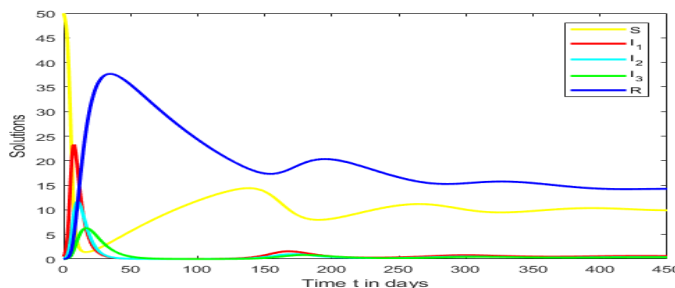


Figure 3: Periodicity of Ebola Virus Disease

Fruit bats are the main source of EVD which peaks periodically especially during their migratory period. The seasonal migration of bats has been attributed to environmental conditions and their pursuit of better climatic conditions, i.e interchanging of rainy and dry seasons [13]. This season is incidentally a bushmeat trade boom for local hunters. Migration of wild animals together with hunting promote the transmission of zoonotic diseases [17]. According to the findings in [7], the author concludes that the occurrence of EVD is closely related to ecological factors and seasons have an impact on human behaviours. Figure (3) implies periodicity of the disease over a given period of time. This indicates the reasons as to why the EVD infection has been recurrent in the DRC.

6.1 Conclusion

EVD is a zoonotic disease and its previous outbreaks could have been as a result of seasonality of bats and the human lifestyle. The results of the study show that the major propeller of the epidemic is class I_1 which has non-specific symptoms. However in the absence of intervention, infections in class I_3 prolongs the epidemic, i.e class I_1 increases the number of infections sharply where as class I_3 prolongs the infectivity period. This in turn was ballooned by the fact that at the onset of the infection the disease has non-specific symptoms which are sometimes mistaken to be other infections. Although it may seem possible that viral levels and transmissibility are directly correlated, it is important to note that such a correlation does not exist. This is shown by the number of infections arising from class I_1 as compared to their viral levels. Therefore health practitioners should engage in disease surveillance at the onset of the infection. To stem the repeated episodes, it is recommended that prompt intervention measures be made at the beginning of an epidemic or before.

The study recommends the analysis of the Ebola dynamics using the effective contact rate as a periodic function to determine the threshold dynamics and the possible drivers of the infection.

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