LOGISTIC MATHEMATICAL MODEL OF EBOLA VIRUS DISEASE WITH CONVALESCENCE

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Abstract – We construct a simple mathematical model on Ebola transmission. The model accounts for the interaction between infectious and susceptible humans leading to infection. The model consisting of a system of ordinary differential equations describes the evolution and propagation of Ebola disease. The model divides the human population into susceptible humans, latent, infectious, convalescent and recovered humans. The analysis includes establishment of the basic reproduction number', R_0, in which R_0<1 guarantees a disease free state that is locally and globally asymptotically stable. The analysis shows that convalescent humans play a great role in ebola virus transmission.

Keywords: Ebola, Logistic, Modelling, Convalescence.

1. Introduction

The Ebola virus disease which was first noticed in 1976in Southern Sudan and the Democratic Republic of Congo [1] has resurfaced in subsequent years. Especially, between 1976 and January 2003, 10 significant Ebola fever outbreaks have occurred in Africa involving more than 1600 cases of infection and 1100 fatalities [2]. Ebola virus is transmitted through direct contact with blood and body fluid of infected individuals including semen, vaginal fluids, sweat, aqueous humour, urine and breast milk [14]. During the recovery process of Ebola infection, Patients who are able to mount an immuneresponse to the virus will begin to recover in 7 to 10days and start a period of prolonged convalescence [4].Most researchers and modellers of Ebola virus disease have focused their concern on disease regime during outbreaks. A historical account of several mathematical models has been given in [3]. They propose a mathematical model incorporating some vital elements bothering on transmission of deceased individuals during funerals and infection through contaminated environment resulting from African practices, hospitality and poor hygienic conditions. Other aspects include the contribution of consumption of bats, hunted meat and fruits from rain-forests to Ebola virus transmission.In modelling the Ebola virus disease in 2014, [2] avers that in order to properly estimate the spread of an infection, every case of infection should be accounted for in the model hence the recommendation that subsequent model should include parameters that account for people who are not recorded as infected or removed. In recent times there is a growing concern of the persistence of Ebola virus in the fluids of both male and female survivors. Especially, WHO recommends for abstinence or condom use during sexual intercourse for at least 3 months after recovery from Ebola virus disease [12, 5, 15]. Also, in a research finding by [7], Ebola virus can persist in different body fluids after being cleared from the blood. They suggest that even though the infectious dose is low, infection risk in convalescence should be assumed, the precautionary principle applied and close contact of Convalescent Ebola patients should be given clear guidance in infection control.To the best of our knowledge, none of the mathematical models on Ebolaconsiders the effect of convalescence on the transmissionof the disease. Here we propose a simple mathematical model incorporating the contribution of convalescence in Ebola virus disease dynamics. Section 1 includes a brief introduction whereas the formulation of the model is given in section 2. In section 3, we present the model analysis followed by numerical simulations in section 4. The paper was rounded up in section 5 with a brief discussion and conclusion.We divide the human population into compartments of susceptible, Latent, Infectious, Convalescence and Recovered. State variables in the model are given in Table 1 and the movement between compartments is summarised in Figure 1, the individual pathways to be discussed below.

Table 1: The state variables in the model

State variable	Description
N	Total human population
S	Number of Susceptible humans
L	Latent human population
I	Number of Infectious humans
C	Number of humans in the convalescent state
R	Recovered human individuals

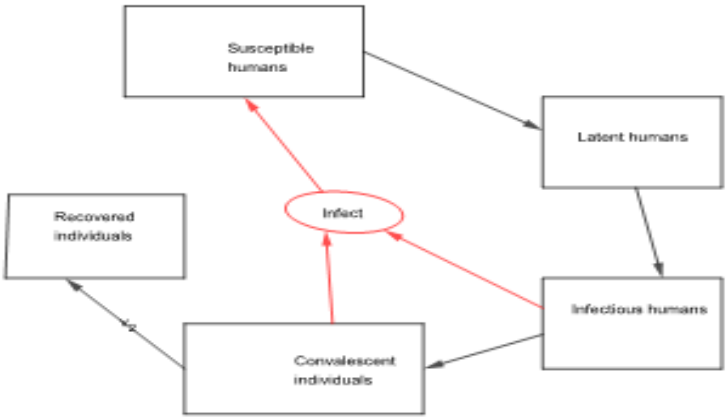


Figure 1:Schematic representation of Ebola transmission.

2 The formulation of the model

The entire human population is described by the equation $S + L + I + C + R = N$. Ebola virus is transmitted when a Susceptible human comes in contact with an Infectioushuman. Susceptible individuals die at a rate μS and the rest progress into the L compartment at ratesαSI and $\gamma SC$where$\alpha$and γ are rate constants. This follows from the principle of mass action which we have assumed that the transition of susceptible humans into the latent class is proportional to the contact between susceptiblehumans and individuals in the infectious and convalescent states. Individuals in the L class are non-infectious and remain in this state until they transit into the infectious compartment at a rate βL. We also assume that newly recovered individuals from Ebola only recover from disease symptoms but fall into a convalescent state whose infection dose has some significant risk of infection that cannot be ignored [12, 5, 15, 7]. We assume that individuals in the various compartments die at per capita rate μ, while some individual in the I class die at an additional rate $\Psi I$from the disease. Since there is no known literature that fully recovered individual can contact the disease again, we assume non transition of fully recovered individuals into the susceptible class and the only source of recruitment into the susceptible compartment is through natural birth, λN,where $\lambda$is the per capita birth rate constant. Using the above assumptions,we propose the following system of equations.

$$\frac{dS}{dt} = \lambda N - (\alpha I + \gamma C)S - \mu S - \theta N^2 (2.1)$$

$$\frac{dL}{dt} = (\alpha I + \gamma C)S - (\mu + \beta)L (2.2)$$

$$\frac{dI}{dt} = \beta L - (\mathrm{w} + \mu + \Psi)I \quad (2.3)$$

$$\frac{dC}{dt} = \mathrm{w}I - (\mu + \phi)C \quad (2.4)$$

$$\frac{dR}{dt} = \phi C - \mu R \quad (2.5)$$

$$\frac{dN}{dt} = (\lambda - \mu)N - \Psi I - \theta N^2 (2.6)$$

where (2.6) is derived from adding $(2.1) - (2.5)$. We impose

$$t = 0, N = N_0$$

as initial human population.

2.1 Parameter values

All the model parameters are listed in Table 2 together with values taken from various sources. We note that the values for these parameters have some variationsbut are within ranges compatible with values given by the various sources.

Table 2: Model parameters and their dimensions. Values marked with $(*)$ are assumed values and the rest are obtained from data.

Parameters	Description	Value	Unit	Source
α	Infection rate of susceptible humans by symptomatic individuals	0.006	$human^{-1}day^{-1}$	[3]
w	Rate of removal from disease death trap	0.060	day^{-1}	[6, 11]
γ	Infection rate of susceptible humans by convalescent individuals	0.0020*	$human^{-1}day^{-1}$	assumed
β	Rate of transition from latent state to infectiousness	0.20	day^{-1}	[6]
μ	Natural death rate	0.16	day^{-1}	[3]
ϕ	Full recovery rate of convalescentindividuals	0.072	day^{-1}	[12,5, 15]
λ	Natural birth rate	0.24*	day^{-1}	assumed
Ψ	Disease induced death rate	0.40	day^{-1}	[6, 8]
θ		0.00024*	$human^{-1}day^{-1}$	assumed

2.2 Nondimensionalisation

Since the variable N is the sum of the relevant compartment values, it is convenient to re-express the compartment values as population fractions using

$$\hat{S} = \frac{S}{N}, \ \hat{L} = \frac{L}{N}, \ \hat{I} = \frac{I}{N}, \ \hat{C} = \frac{C}{N}, \ \hat{R} = \frac{R}{N}$$

so that

$$\hat{S} + \hat{L} + \hat{I} + \hat{C} + \hat{R} = 1.$$

The time derivatives for the variables will become, using variable S as an example

$$\frac{dN\hat{S}}{dt} = N\frac{d\hat{S}}{dt} + \hat{S}\frac{dN}{dt} = N\frac{d\hat{S}}{dt} + \left(\lambda - \mu - \Psi\hat{I} - \theta N\right)N\hat{S},$$

A major control strategy adopted during Ebola Virus disease outbreak has been that of tracking and monitoring of all people who have come in contact with a first index case in order to treat and prevent them from transmitting the disease at the time they become infectious. The timescale in which an infected person remains in the latent class before eventually becoming sick or infectious is a very important determinant of the transmission of the disease. Hence we scale time with the transitionparameter β, from infection to infectiousness and write

$$t = \frac{\hat{t}}{\beta}$$

Assuming that N_0 is the initial population of humans, we write

$$N = N_0\widehat{N}.$$

By defining the following dimensionless parameters:

$$b = \frac{\lambda}{\beta}, \ a = \frac{\alpha N_0}{\beta}, \ d = \frac{\gamma N_0}{\beta}, \ e = \frac{\theta N_0}{\beta}, f = \frac{\Psi}{\beta}, g = \frac{w}{\beta}, h = \frac{\phi}{\beta}, p = \frac{\mu}{\beta}, t_0 = \frac{1}{\beta},$$

and by substituting these new parameters into $(2.1) - (2.6)$ and dropping the hats for clarity

we get

$$\frac{dS}{dt} = b(1-S) - (aI + dC)S + e(S-1)N + fSI, (2.7)$$

$$\frac{dL}{dt} = (aI + dC)S - (1+b)L + eLN + fLI, (2.8)$$

$$\frac{dI}{dt} = L - (g + f + b)I + eIN + fI^2, (2.9)$$

$$\frac{dC}{dt} = gI - (h+b)C + eCN + fCI \quad (3.0)$$

$$\frac{dR}{dt} = hC - bR + eRN + fRI \quad (3.1)$$

$$\frac{dN}{dt} = (b-p)N - fIN - eN^2. (3.2)$$

The dimensionless parameters and their values are given in Table 3.

Table 3: List of dimensionless parameters and their definitions in terms of the dimensional parameter values.

Dimensional form	Nondimensional parameter	Value
$\frac{\lambda}{\beta}$	b	1.20
$\frac{\alpha N_0}{\beta}$	a	3.76
$\frac{\gamma N_0}{\beta}$	d	1.28
$\frac{\theta N_0}{\beta}$	e	0.15
$\frac{\Psi}{\beta}$	f	0.20
$\frac{\omega}{\beta}$	g	0.067
$\frac{\phi}{\beta}$	h	0.36
$\frac{\mu}{\beta}$	p	0.8

3 Model Analysis

3.1 Establishing the basic reproduction Number, R_0.

Thebasic reproduction number is the expected number of secondary infection cases that would arise from the introduction of a single Ebola infected case into a fully susceptible or Ebola-free population[11]. A newly infectious Ebola patience in a disease-free population will infect people throughout his infectious period $\frac{1}{g+f+p}$ ata rate$(aI + dC)S$he method of next generation matrix usedin [12], [5]in determining the basic reproduction number of an infectious disease may be used in deriving C_{p_n}.

By considering a small perturbation of the kidnap-free state $(S = 1,\ L = 0,\ I = 0,\ C = 0,\ R = 0)$,

we investigate the linearised system expressed in the form

$$K' = FK - MK, \tag{3.3}$$

Where

$$K' = \frac{dR}{dt}, F = \begin{bmatrix} 0 & a & d \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, M = \begin{bmatrix} a_1 & 0 & 0 \\ -1 & a_2 & 0 \\ 0 & -g & a_3 \end{bmatrix}, K = \begin{bmatrix} L \\ I \\ C \end{bmatrix} \tag{3.4}$$

and $a_1 = 1 + p,\ a_2 = g + f + p,\ a_3 = h + p.$

Here, FK represents the matrix of new infection cases, MK is the transition of these cases between compartments and K the "reservoir of infection".This method assumes that there is a non-negative matrix $G =$

FM^{-1} that guarantees a unique, positive and real eigenvalue strictly greater than all others. Computing the inverse of M yields

$$G = \frac{1}{a_1 a_2 a_3}\begin{bmatrix} c_1 & c_2 & c_3 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, (3.5)$$

Where $c_1 = aa_3 + dg,\ c_2 = aa_1a_3,\ c_3 = da_1a_2.$

The characteristic equation of (3.3) in terms of the eigenvalue, σ, gives the largest

eigenvalue as $\sigma = \frac{c_1}{a_1a_2a_3}$. Thus the basic reproduction number is expressed as

$$\mathrm{R}_0 = \frac{a(h+p)+dg}{(1+p)(h+p)(g+f+p)}. (3.6)$$

3.2 Positivity, Existence and Uniqeness of Solution

The model is described in the domain

$$\Phi \in \mathcal{R}^5 = \left\{ \begin{matrix} S, L, I, C, R, N : S \geq 0\ , L \geq 0\ , I \geq\ 0\ , C \geq\ 0\ , R \geq\ 0\ , N > 0\ , \\ S + L + I + C + R = 1. \end{matrix} \right\} (3.7)$$

Suppose at $t = 0$ all variables are non-negative, then $S(0) + L(0) + I(0) + C(0) + R(0) = 1$. If $L = 0$, and all other variables are in Φ, then $\frac{dL}{dt} \geq 0$. This is also the case for all other variables in $(2.9) - (3.1)$. But if $S = 0$, $b\ >\ p$and $N < \frac{b-p}{e}$,then $\frac{dS}{dt} \geq 0$.If $N = 0$, then $\frac{dN}{dt} = 0$. But if $N > 0$ and

assuming $b\ >\ p$, then with appropriate initial conditions, $\frac{dN}{dt} > 0$ for all values of $t\ >\ 0$. We note that the right-hand side of $(2.7) - (3.2)$ is continuous with continuous partial derivatives, so solutions exist and are unique. The model is therefore mathematically and biologically well posed with solutions in Φ for all $t \in [0, \infty)$.

3.3 Steady state solution and stability analysis

It can easily be shown from the system that the kidnap free state is $(S, L, I, C, R) =\ (1, 0, 0, 0, 0)$. In the absence of the disease, $I = 0$ and substituting this into the right hand side of(2.9) and(3.0) we obtain$L = 0$. and$C = 0$ respectively. Further substitution of the values of $I$and C into (3.1) gives $R = 0$. For $N > 0$ and using the values of L and Cin (2.7) gives $S = 1$. Thus we are only left with the logistic equation

$$\frac{dN}{dt} = (b-p)N - eN^2, (3.8)$$

Which can be expressed in the special Bernouli form

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{k}\right) (3.9)$$

Where $r = b - p$ and$k = \frac{b-p}{e}$. Dividing (3.9) through by N^2,setting $n = \frac{1}{N}$ and $m = n - \frac{1}{k}$,we have the first order linear ode

$$\frac{dm}{dt} + rm = 0 (4.0)$$

By converting the solution of (4.0) to the original variables we get

$$N(t) = \frac{kN(0)}{N(0) + (k - N(0))e^{-rt}}$$

and as $t \to \infty$, $N(t) \to k = \frac{b-p}{e}$, the logistic curve or carrying capacity of the environment.

The solution to the logistic equation (3.8), shows that for $b > p, N(t)$ will grow to $\frac{b-p}{e}$ if $N(0) < \frac{b-p}{e}$ or $N(t)$ will decay to this value if $N(0) > \frac{b-p}{e}$. However, as we observe in the numerical solution thatin a situation where the basic reproduction number is less than unity, there is an initial reduction in the population before eventually increasing to the steady state. This shows that disease related deathr reduces the population in a fast timescale before the disease is brought under control. This behaviour may enable us to determine the number of deaths and the duration of the epidemic in dimensional terms.

The disease free state is locally asymptotically stable when $R_0 < 1$ and globally asymptotically stable When$R_0 < 1$, and unstable for$R_0 > 1$, where R_0 is as defined in (3.6). We note that $R_0 = 1$ is a bifurcation surface in which the system changes its stability status.

We derive sufficient conditions for local and global stability of the disease free state from all initial conditions in Φ. The Jacobian matrix obtained by linearising system $(2.7) - (3.1)$about the disease free equilibrium point, $(S, L, I, C, R) = (1, 0, 0, 0, 0)$is

$$J_{d_f} = \begin{bmatrix} -p & 0 & f-a & -d & 0 \\ 0 & -(1+p) & a & d & 0 \\ 0 & 1 & -(g+f+p) & 0 & 0 \\ 0 & 0 & g & -(h+p) & 0 \\ 0 & 0 & 0 & h & -p \end{bmatrix}.$$

Lemma 3.1*The disease free equilibrium is locally asymptotically stable if*$R_0 < 1$*and*

unstable if$R_0 > 1$.

Proof The characteristic polynomial equation of (3.6) with eigenvalues, K is

$$(p + \mathrm{K})^2\{\mathrm{K}^3 + (c_1 + c_2 + c_3)\mathrm{K}^2 + [c_3(c_1 + c_2) + c_1c_2 - a]\mathrm{K} + c_1c_2c_3 - (ac_3 + gd)\} = 0$$

With two of the eigenvalues strictly negative leaving the cubic equation

$$\mathrm{K}^3 + (c_1 + c_2 + c_3)\mathrm{K}^2 + [c_3(c_1 + c_2) + c_1c_2 - a]\mathrm{K} + c_1c_2c_3 - (ac_3 + gd) = 0, (4.1)$$

where

$c_1 = 1 + p, c_2 = g + f + p$ and $c_3 = h + p$.

Multiplying and dividing the coefficient of K by c_3, and also adding gd to and subtracting same from the coefficient of K gives

$$\mathrm{K}^3 + (c_1 + c_2 + c_3)\mathrm{K}^2 + \frac{1}{c_3}\{{c_3}^2(c_1 + c_2) + gd + c_1c_2c_3(1 - \mathrm{R}_0)\}\mathrm{K} + c_1c_2c_3(1 - \mathrm{R}_0) = 0, (4.2)$$

whereR_0 is as defined in (3.6) above.If$R_0 < 1$, then the coefficients of thecubic polynomial of (4.2) are all positive and non-zero; so by the Descartes' rule of signs there is no positive real eigenvalue. This means there are 3 negative real eigenvalues or 1 negative real eigenvalue and a complex conjugate pair. Thus by defining the

coefficients of K^3, K^2, K and the constant term in (4.2)as A_1, A_2, A_3 and A_4 respectively Routh Hurwitz stability condition for a cubic polynomial as stated in

[1] and given in this case by $A_2A_3 > A_1A_4$ is satisfied.

We observe that if$R_0 > 1$, the constant$A_4$is negative and the sequence of coefficients$\{A_n\}$

will have only one sign change irrespective of the sign of A_3. Thus, by using Descartes' rule of sign there exists one positive real eigenvalue and we conclude that the disease free state is unstable if

$R_0 > 1$. When$R_0 = 1$, $A_4 = 0$ and(4.2) has zero eigenvalue, which shows that $R_0 = 1$ is a bifurcation surface in (a, d, f, g, h, p) parameter space.

Lemma 3.2*The disease free equilibrium is globally asymptotically stable in Φ if*

$$R_0 < 1. \quad (4.3)$$

Proof Consider the function $\Psi : \{(S, L, I, C, R) \in \Phi\} \to \mathcal{R}$, where

$$\Psi = \frac{(1+p)(1-L)^2}{2(1+b)} + \frac{a(1-I)^2}{2(f+p)(g+f+b)} + \frac{d(1-C)^2}{2(h+p)(h+b)} + \frac{(1+p)(1-R)^2}{2h} \quad (4.4)$$

We note that $\Psi \geq 0$ and is continuously differentiable on the interior of Φ. We shall show that the disease free equilibrium is a global minimum of Ψ on Φ if (4.3) holds. The derivative of Ψ computed along solutions of the system is

$$-\left(\frac{1+p}{1+b}\right)(aI + dC)S(1-L) + (1+p)(L+I) - (1+p)(S+L+I+C) - (1+p)I - (1+p)L^2 - \frac{e(1+p)LN(1-L)}{1+b} - \frac{f(1+p)LI(1-L)}{1+b} - \frac{aL(1-I)}{(f+p)(g+f+b)} + \frac{aI}{f+p} - \frac{aI^2}{f+p} - \frac{aeNI(1-I)}{(f+p)(g+f+b)} - \frac{afI(1-I)}{(f+p)(g+f+b)} - \frac{dg(1-C)}{(h+p)(h+b)} + \frac{dC}{h+p} - \frac{dC^2}{h+p} - \frac{deNC(1-C)}{(h+p)(h+b)} - \frac{dfIC(1-C)}{(h+p)(h+b)} - (1+p)C - b(1+p)R(1-R) - \frac{e(1+p)N(1-R)}{h} - \frac{f(1+p)IR(1-R)}{h} \quad (4.5)$$

Further simplification of (4.5) leads to

$$\frac{d\Psi}{dt} = -\frac{(1+p)[\{(aI+dC)S + L(eN+fI)\}(S+I+C+R) + \{(1+b)(S+L^2+C)\}]}{1+b} - \frac{\{aI^2(h+p) + dC^2(f+p)\}}{(f+p)(h+p)} - \frac{a\{L + I(eN+f)\}(S+L+C+R)}{(f+p)(g+f+b)} - \frac{d\{g + C(eN+fI)\}(S+L+I+R)}{(h+p)(h+b)} - \frac{(1+p)\{eN + R(bh+fI)\}(S+L+I+C)}{h} + \frac{\{a-(f+p)(1+p)\}I}{f+p} + \frac{\{d-(h+p)(1+p)\}I}{h+p}$$

We can see clearly that $\frac{d\Psi}{dt} < 0$ when

$$a < (f+p)(1+p), d < (h+p)(1+p). \quad (4.6)$$

Simple deduction from(3.6) shows that$R_0 < 1$whenever (4.6) is satisfied. In fact, for $(L, I, C, R) = (0,0,0,0)$, $\frac{d\Psi}{dt} \leq 0$ and (L, I, C, R) is the largest positively invariance subset in the interior of Φ and by LaSalle's invariant principle [10], $(L, I, C, R) \to (0,0,0,0)$ as $t \to \infty$ while $S \to 1$ on the boundary of Φ. Thus, the disease free state is globally stable if $R_0 < 1$.

4 Numerical Simulations

The numerical solution is obtained by using MATLAB's ode$15s$, a variable order Runge-Kutta method with a relative and absolute tolerance of 10^{-9}. The parameters used for the simulations as defined in Table Table 3 are $b = 1.20, a = 3.76, d = 1.28, e = 0.15, f = 0.20, g = 0.067, h = 0.36, p = 0.80$. At time $t = 0$ we have the following initial conditions in the proportions; $S = 0.99, L = 0.01, I = 0, C = 0, R = 0, N = 1$. This is a situation where the entire vulnerable human population is exposed to a small fraction of infected humans. The program was run in MATLABandMAPLEwith different sets of initial conditionsand the qualitative form of the steady state solutions in all cases were the same, although the system gets to a steady state faster as the initial fraction of infected humans increases.In Figure $2a$, the proportion of susceptible human population drops as

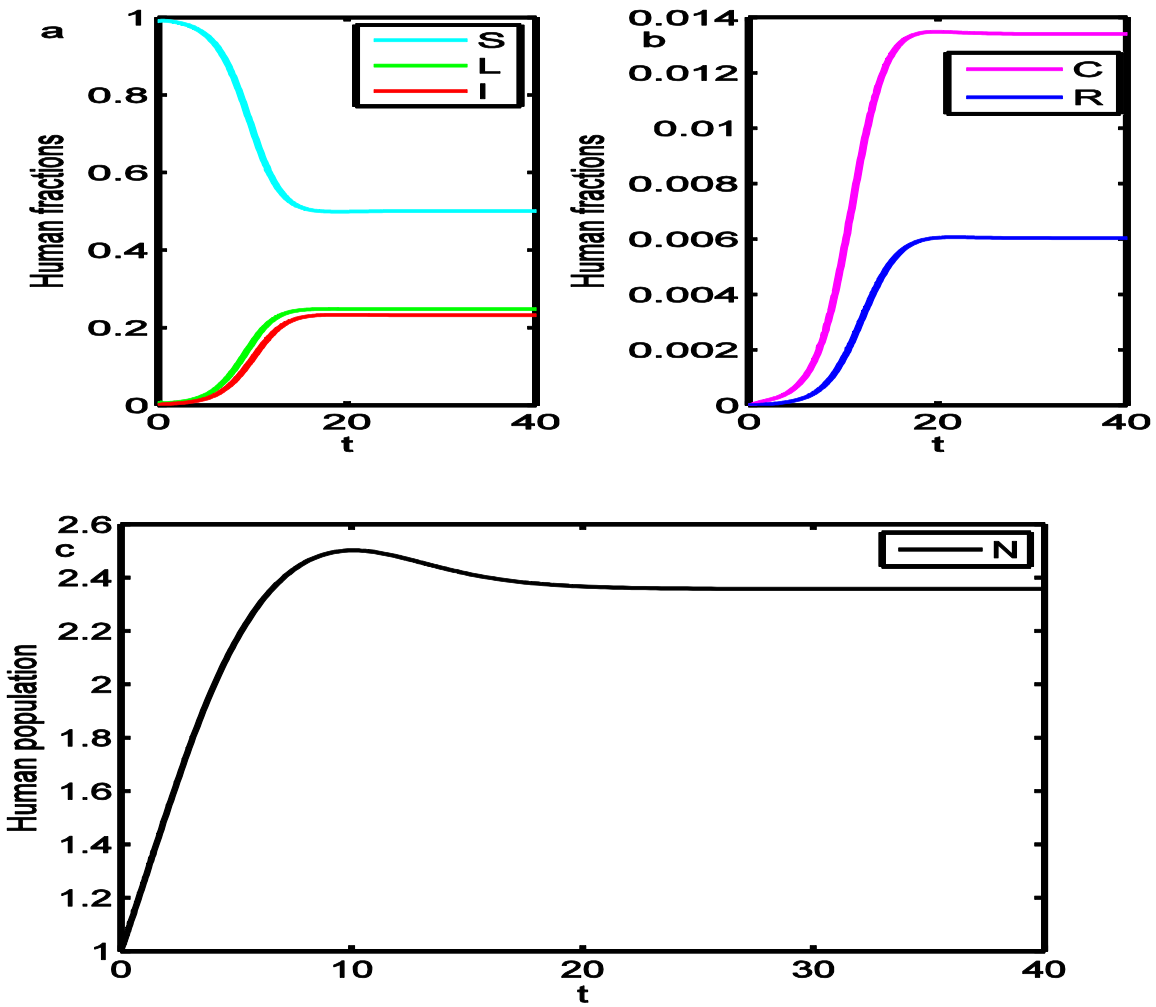


Figure 2: Results showing the effect of small amount of infected human on evolution of Ebola epidemic in a disease free society, where $t = 1$, represents approximately 5 days in real time. The initial conditions used are$S = 0.99, L = 0.01, I = 0, C = 0, R = 0, N = 1$and the parameter values are given in Table 3.

more people contact the disease while in Figure 2b, more of the recovered humans merely assume a convalescent state whereas the entire population is gradually increasing in Figure 2d. In other to study the impact of convalescent humans on the evolution of the disease we consider a situation where there is small amount of convalescent humans in the absence of infected and infectious individuals in figure 3. The quality of the solutions is not significantly different from that in Figure 2. However, the initial amount of convalescent individuals drops sharply in Figure 2a and later grows to a steady state. Consequently, the fraction of recovered humans peaks in a short time scale and drops for a period and later increases to a steady. This is an expected behaviour as proportion of the convalescent humans acquire full recovery in a short while whereas the rest generate infection.

Figure 4 represents a scenario where the

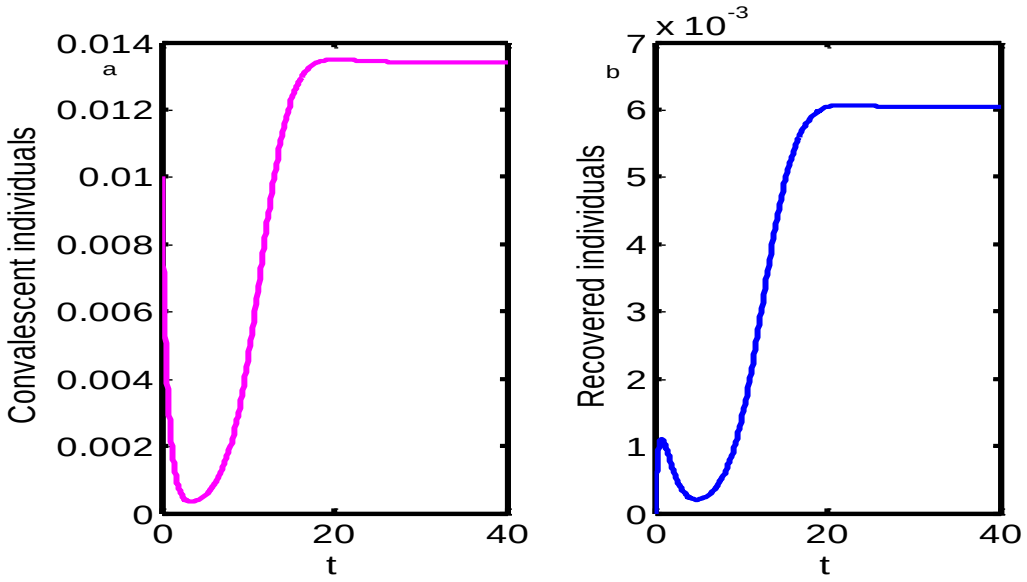
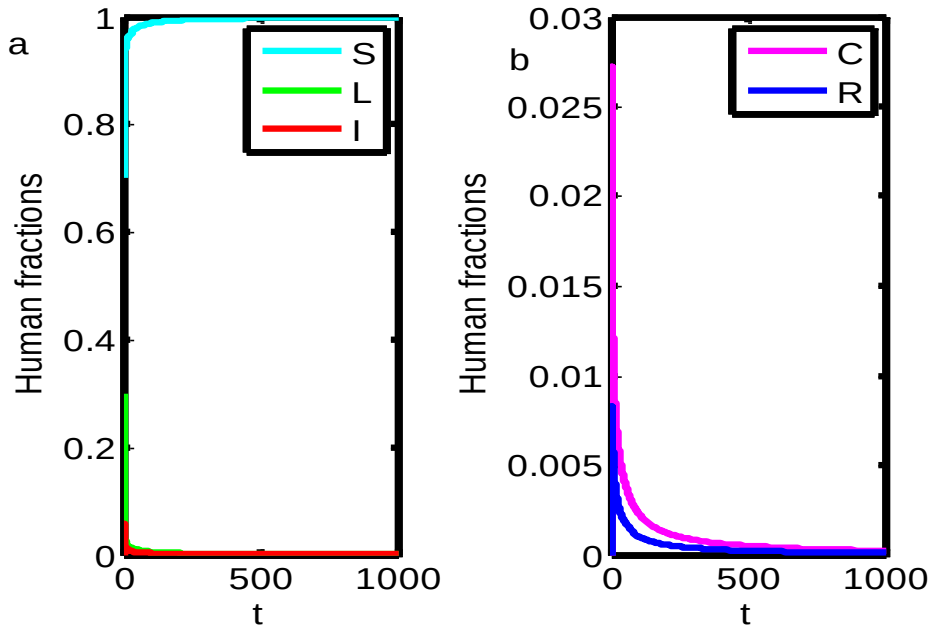


Figure 3: Results showing the effect of small amount of convalescent human on evolution of Ebola epidemic in a disease free society. The initial conditions used are $S = 0.99, L = 0, I = 0, C = 0.01, R = 0,\ N = 1$ and the parameter values are the same as those in Figure 2.

disease could be eliminated by carefully choosing the values of some sensitive parameters connected with the infection rate of susceptible humans by symptomatic individuals, the rate of removal from disease death trap and the infection rate of susceptible humans by convalescent individuals. The entire population grows to a steady based on the carrying capacity of the environment as the disease classes recover. The effects of different values



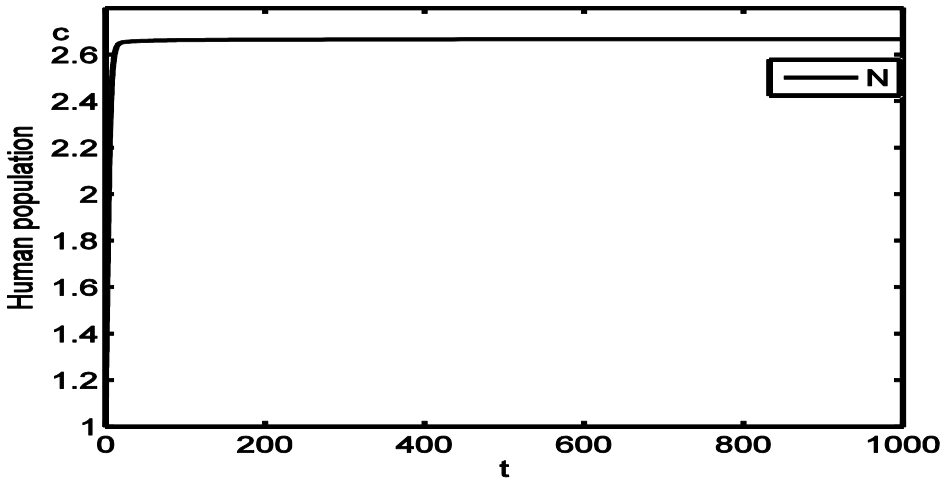


Figure 4: Results showing the disease profile on the entire population when the control parameter, $R_0 < 1$. The initial conditions and parameter values used for the simulations are the same as those in Figure 2 except that $a = 1.799, d = 2.087$ and $= 0.9$.

of a_0 on the various fractions of the human population are demonstrated in Figure $5a, b, c, d$. We investigate each of the human sub-populations as a_0 varies from 0.00001 to 0.2 and the results show there is a unique steady state for each human compartment irrespective of the value of a_0. Figure 6

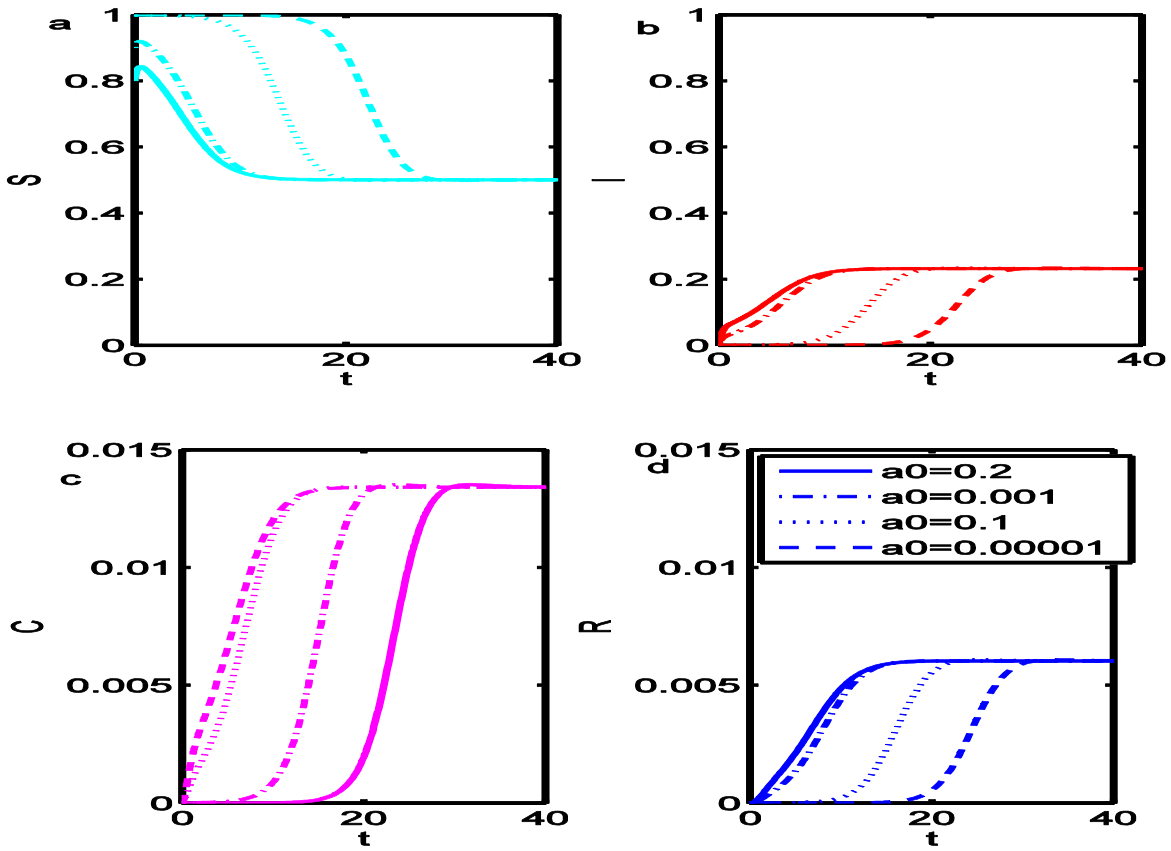


Figure 5: Results showing the effect of introducing different amount of infected humans on the different compartments. The parameter values used for the simulations are the same as those in Figure 2 except that for the

initial conditions, we have used $S = 1 - a_0$, $L = a_0$, $I = 0, C = 0, R = 0, N = 1$ with different values of a_0, as shown in the graphs.

shows the relationship between the basic reproduction number and the disease profile as it affects the population.In order to demonstrate the impact of the basic reproduction number on the dynamics of the system, we plot the steady states of the various compartments against the basic reproduction number (R_0), in which we show a disease free state when R_0 is less than unity and for $R_0 > 1$the disease invades the population.The values of R_0 were obtained by $a$and $d$and $R_0 = 1.00$correspondsto $a = 2.76$ and $d = 1.28$ .Figure 7 is a bifurcation diagramshowing a switch froma disease free state to an endemic state. The result is obtained by drawing the steady state of

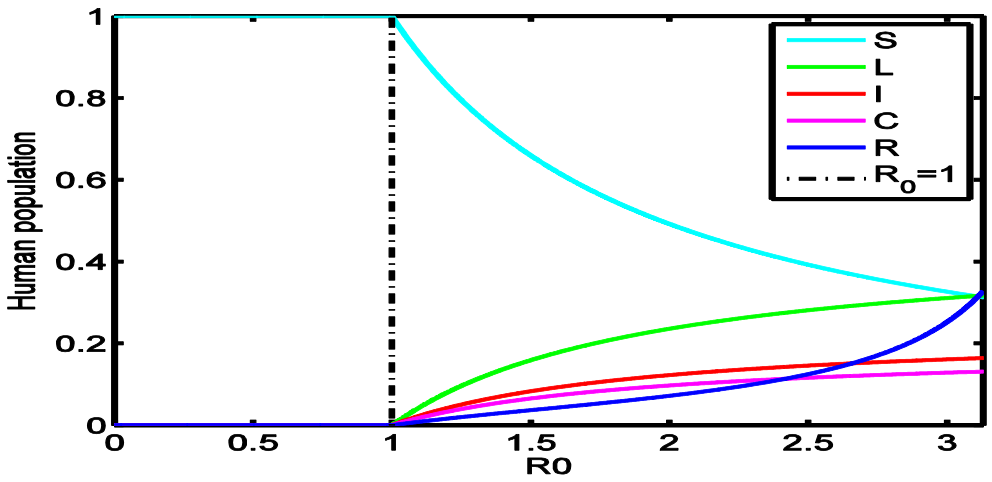


Figure 6: Results showing the disease free state when $R_0 < 1$ and the disease persistent state for $R_0 > 1$ by varying the value of R_0 from 0 to 3.1. The parameter values used to obtain these results are given in Table 3. We used the parameters, $a$and $d$to change R_0 where $R_0 = 1.00$ corresponds to $a = 2.76$ and $= 1.28$.

covalescent individuals against different values of R_0. The plot shows a transcritical bifurcation in the vicinity of $R_0 = 1$, as is expected from the analysis. Although some uncertainty still surrounds our quest on whether or not the diseases invades at $R_0 = 1$, the disease free state is stable for values of $R_0 < 1$, but becomes unstable when $R_0 > 1$ whereas, the disease persistent state becomes stable as expected.

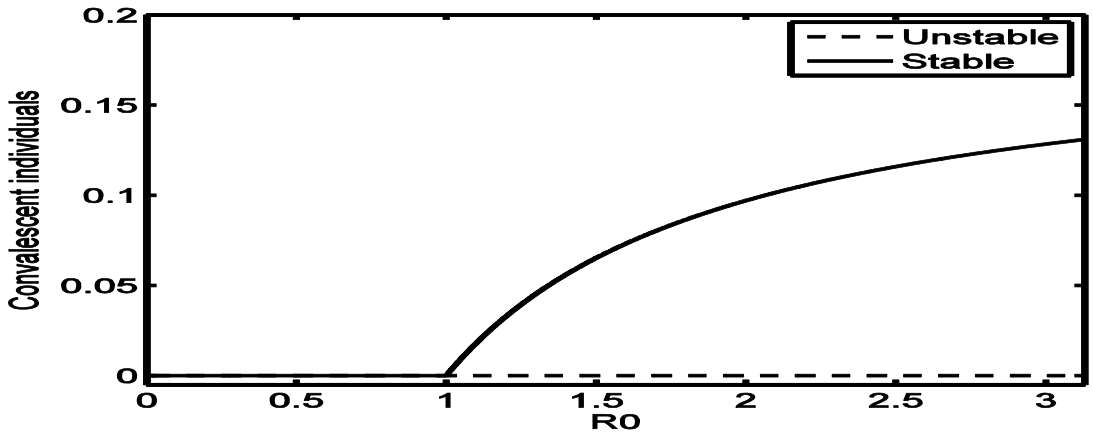


Figure 7: Basicreproduction number (R_0) bifurcation diagram. The curve shows a transcritical bifurcation obtained by drawing the steady states of convalescent individuals against different values of R_0 ranging from 0 to 3.1. Parameter values are the same as those in Figure 6.

5 Discussion and conclusion

Our model describes a typical situation of Ebola epidemic as shown by the value of$R_0 = 2.00$ using the data in Table 3. This value falls within the estimates given in [13, 9].

The numerical solution (Figure 3) shows that about 20% of the entire population will be engulfed by the disease within a period of three months. Although only a small proportion of the population seem to recover from the disease, more of this number only assume a convalescent state instead of full recovery, which portends great danger of unsuspected epidemic due to the findings of [7].

The economic cost of the disease to households and government is enormous since the sick would require medical attention in the hospital resulting in a huge loss of man-hours. In order to bring a disease under control in a population of varying size, we need to reduce the Basic reproduction number,R_0, below a threshold value with increasing time.

This is demonstrated by the numerical solution in Figure 4a,b.From the results of our analyses in section 3.3. It is worth noting that reducing a or$d$only cannot drag R_0 into extinction but can only cause it to fall below the threshold value of 1 depending on the values of the model parameters. However, significant disease control strategies targeting infection rates of infectious humans, *a*, and convalescent individuals, *d*,could potentially be moreeffective since these would drastically reduceR_0 to zero. This could be done through rigorous quarantine and tracking of allindividuals who may have come into contact with infected patients in order to prevent them from infectiousness and further contact with susceptible humans. In the other hand, a careful preventive and risk control management of the convalescent period could help reduce the infection rate of convalescent individuals and facilitate their full recovery.

The analyses suggest that the disease free state is locally and globally asymptotically stableWhen$R_0 < 1$, and unstable for$R_0 > 1$. This is further confirmed by the numerical solution. The contribution of convalescent individuals in the transmission of the disease cannot lead to the eradication of kidnapping but could only help in the management and control of the crime.

References

1. Allen, L. J. S. (2007). An Introduction to Mathematical Biology, Prentice Hall, Upper Saddle River, N. J.
2. Bekoe, C. (2015). The SIR model and the 2014 Ebola virus disease outbreak in Guinea, Liberia and Sierra Leone. International Journal of Applied Sciences, 6(2), pp. 11-24.
3. Berge, T., Lubuma, J. M. S., Moremedi, G. M., Moris, M., Kondera-shava, R. (2017). A simple mathematical model for Ebola in Africa. Journal of Biological Dynamics, 11(1), 42-47. DOI:10.1080/17513758.2016.1229817
4. Casillas, A. M., Nyamathi, A. M., Sosa, A., Wilder, C. L., Sands, H. (2003). A current review of Ebola virus: Pathogenesis, clinical presentation, and diagnostic assessment. Biological Research for Nursing, 4(4).
5. Centres for Disease Control and Prevention. Ebola (Ebola virus disease). Question and Answers on Transmission: online. Available at http://www.cdc.gov/vhf/ebola/transmission/quas.html
6. Chowell, G., Hengartner, N. W., Castillo-Chavez, C., Fenimore, P.W., Hyman,J. M. (2004). The basic reproductive number of Ebola and the effects of public health measures: The cases of Congo and Uganda, J. Theor. Biol. 229 (2004), pp. 119–126.
7. Chughatai, A. A., Bones, M., Macintyre, C. R. (2016). Persistence of Ebola Virus in Various Body Fluids During Convalescence: Evidence and implications for Disease Transmission and Control, Epidemol Infect. 144 (8): 1652-1660. Online 2016 Jan. 25. doi: 10.1017/50950268816000054
8. Fisman, D., Khoo, E., Tuite, A. (2014). Early epidemic dynamics of the Western African 2014 Ebola outbreak: Estimates derived with a simple two Parameter model, PLOS Curr. Outbreaks. Online 2014 Sept 8. doi:10.1371/currents.outbreaks.89c0d3783f36958d96ebbae97348d571.
9. Ivorra, B., Ngom, D., Ramos, A. M. (2015). A mathematical model to predict the risk of human diseases spread between countries-validation and application to the 2014–2015 ebola virus disease epidemic, Bull. Math. Biol. 77, pp. 1668–1704. doi:10.1007/s11538-015-0100-x
10. LaSalle,J. P. (1968). Stability theory for ordinary differential equations, J. Differential Equations 4: 57-65.

11. Legrand, J., Grais, R. F., Boelle, P. Y., Valleron, A.J., Flahault, A. (2007). Understanding the dynamics of Ebola epidemics, Epidemiol. Infect. 135, 610–621.
12. Mate, S. E., Kugelman, J. R., Nyeswa, T. G., (2015). Molecular Evidence of Sexual Transmission of Ebola Virus. N Engl J Med., 372: 2448-2454.
13. Ndanguza, D., Tchuenche, J. M., Haario, H. (2013). Statistical data analysis of the 1995 Ebola outbreakIn the Democratic Republic of Congo, Afrika Mat. 24, pp. 55–68.
14. Varkey, J. B., Shanta, J. G., Crozier, I., Craft, C. S., Lyon, G. M., Mehta, A. K. (2015). Persistence of Ebola Virus in Ocular Fluid During Convalescence. N Engl J Med., 372: 2423-2427
15. WHO (2016): Interim Guidance, Clinical care for Survivors of Ebola virus disease. Available athttp://www.who.int/csr/resources/publications/ebola/guidance-survivors/en/.