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Mathematical Modeling of the Transmission Dynamics of Ebola Virus Disease with control Strategies

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Abstract

In this paper we develop a deterministic compartmental mathematical model for the spread of the Ebola virus disease (EVD) in the community. Our model incorporates education campaigns, quarantine, safe burial and therapeutic treatment as control strategies to bring the disease to an end. We derived the effective reproductive number for the model, and proved the stability of equilibrium points. We performed numerical simulations of the model and our results show that all control measures under consideration have an effect of decreasing severity of the epidemic when constantly administered. Furthermore, we found that reducing the number of contacts with infectious individuals in the general population is the most effective intervention method for mitigating an EVD epidemic.

Keywords: Ebola virus; therapeutic treatment; reproductive number; Education campaign; Safe burial.

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1. Introduction

The Ebola virus was first identified in 1976 near the Ebola River infecting at least 280 people, and there were several outbreaks of Ebola virus disease (EVD) over the years. However, none of those were as serious as the current outbreak in West Africa, which started in March 2014 and is affecting the whole world. Multiple species have been identified, but the present outbreak was caused by the Zaire species [1]. In December of 2013, an outbreak of Ebola Virus Disease began in the West African country of Guinea and later spread to neighbouring countries Liberia and Sierra Leone. By November 4th, 2014, the outbreak had reached 13,268 cases, 27 of which had spread to neighbouring countries and overseas in Senegal, Nigeria, Mali, Spain and the US [2].

Ebola Virus Disease (known simply as Ebola) is caused by the epizootic Ebola Virus, which is thought to be found in mammals of the family Pteropodidae (aka fruit bats) [2]. Ebola virus belongs to the family of Filoviruses, characterised by filamentous particles. Its particles have a uniform diameter of 80 nm with length up to 14000 nm [3]. Ebola is a highly pathogenic virus, and the mortality of EVD is about 50–90%. Patients who are infected by Ebola virus may have the symptoms of headaches, vomiting, diarrhoea and so on [4]. However, its natural reservoirs have not been well identified until now [5]. The main route of infection for EVD is direct contact with the patients' bodily fluids, including blood, sweat, vomit, excrement, urine, saliva, or semen and so on [3]. The incubation period of EVD is about 2 - 21 days and the patients in the incubation period are not infectious [6].

Transmission of the virus can occur from bats to other mammals, usually chimpanzees, gorillas and baboons [7]. The current outbreak in West Africa was started from a 2-year-old boy who was infected by a bat, and then Ebola has spread through human-to-human transmission via direct contact with bodily fluids of infected people, and with surfaces and materials contaminated with these fluids. That is why health care workers have frequently been infected while treating patients with suspected or confirmed EVD. It has not been proved that Ebola can spread among humans via airborne transmission, although Ebola goes airborne from pigs to monkeys [1].

Transmission from infected to susceptible humans occurs through direct contact with the saliva, mucus, vomit, faeces, sweat, tears, breast milk, urine and semen of an infected individual. Since direct contact of bodily fluids is necessary, the points of entry of the virus include the nose, mouth, open wounds, eyes, abrasions and cuts [7]. Symptoms include fever, sore throat, muscle pains and headaches, followed by vomiting, diarrhoea, rashes and decreased function of the kidney and liver, then internal and external bleeding. The risk of death from the disease is around 50%, increasing as the disease progresses to the bleeding stage [8]. This is also the stage at which infected individuals become infectious. Diagnosis of Ebola can be difficult, because Ebola is frequently misdiagnosed as typhoid and malaria. Currently there is no treatment of Ebola [9]. Based on the actual situations, it was found that absence of effective control measures was the main cause for Ebola outbreak. Moreover, severe shortage of medical resources [10, 11] and traditional funerals [12] may result in the persistence of EVD.

Epidemiologists build rings around the virus to stop the spread of Ebola, which starts with the circle of people in direct contact with the patient. All the people in the circle are asked about their own circle of close contacts.

With close observation and clear education, such as monitoring the symptoms and avoiding crowded public spaces among others, these rings are usually sufficient to stop the spread of EVD [13]. Isolation is absolutely necessary to bring an end to the spread of Ebola. However, it is not easy to decide whether to quarantine a person or not a according to Sankarankutty and Mekaru [14].

The principal aim of modelling infectious diseases is to be able to make judicious decisions in the application of control interventions of the infection to eliminate and ideally to eradicate it from the human population. Simulations and modelling can optimize control efforts such that limited resources are targeted to achieve the highest impact [15].

In this paper we aim to model the role of education campaigns, hospitalization, therapeutic treatment and safe burial as control strategies in the transmission dynamics of Ebola. The rest of our paper is organized as follows; second section will base on formulation of the model, the third section will be on model analysis, the fourth on Simulation and discussions and lastly we will wind up our work by conclusion and recommendations.

2. Model Formulation

In this paper we develop a deterministic compartmental mathematical model for EVD that captures education campaigns, safe burial, hospitalization and therapeutic treatment as control strategies based on following assumptions:

(1) Deceased human individuals can still infect during funerals. This assumption is motivated by the African practices (e.g. washing of deceased individuals) during burial ceremonies.

(2) There is a permanent disease-induced immunity. This assumption is motivated by the fact that it has never been reported that an individual caught the EVD for the second time.

(3) We assume that there is a vital dynamics. Indeed, some of the Ebola outbreaks have lasted more than two years (for instance the Western Africa outbreak). Thus, during this relatively long period of time, there might be new births or inflow of susceptible individuals from other/surrounded places as well as natural deaths, which allow a demographic process to take place, as studied in [16,17].

(4) Homogeneous mixing of members of the population under consideration.

Based on the above mentioned assumptions and motivated by the works of [18, 19, 20, 21, 22, 23], we develop a new deterministic model as follows:

Our model is composed of eight compartments namely $S(t), E(t), I_u(t), I_e, H(t), F(t), B(t)$ and $R(t)$; $S(t)$ is the number of susceptible individuals at time t, $E(t)$ is the number of latent individuals at time t, who are infected but not infectious yet, or individuals with symptoms but misdiagnosed by a doctor, $I_u(t)$ denotes the class of unaware infectious individuals at time t, $I_e(t)$ denotes the class of aware infectious individuals at time t, $H(t)$ is the number of individuals who are hospitalized or isolated infectious individuals at time t, $F(t)$ is the

number of cases who are dead but not yet buried at time t, $B(t)$ is the number of cases who are dead and already buried at time t and $R(t)$ is the group of recovered individuals at time t; the total active population i.e individual who are alive, $N(t)$ is given by:$N(t) = S(t) + E(t) + I_u(t) + I_e(t) + H(t) + R(t)$ which is in fact not constant because we have assumed the vital dynamics and so the existence of births and deaths.

Susceptible class is increased by birth or immigration at a constant rate Λ and decrease either by natural mortality rate μ or interact with infectious individuals at a force of infection $\lambda = \omega_u I_u + \omega_e I_e + \omega_f F + \omega_h H$ where $\omega_u, \omega_e, \omega_f$ and ω_h are the contacts rates for unaware, aware, dead body and hospitalized individuals respectively. After contamination, susceptibles move to compartment, E. A fraction f of Individuals in class E move to aware infectious class at the rate θ while the compliment $(1-f)$ move to unaware infectious class at the rate α. A fraction l of unaware individuals die due to EVD at the rate ϕ and the compliment $(1-l)$ of I_u progress either to Hospitalized or Recovery classes. A fraction δ_1 of $(1-l)$ move to recovery class at the rate η_3 while the compliment $(1-\delta_1)$ of $(1-l)$ are hospitalized at the rate γ_2. A fraction p of I_e are recovered at the rate η_1 while the compliment $(1-p)$ are hospitalized at the rate γ_1. A fraction q of H are safely buried at the rate τ_2 while the compliment $(1-q)$ are recovered at the rate η_2.The dead bodies, F are unsafely buried at the rate τ_1.

Detailed description of parameters is shown in Table 1 while the compartmental flow diagram of the model is shown by Figure 1.

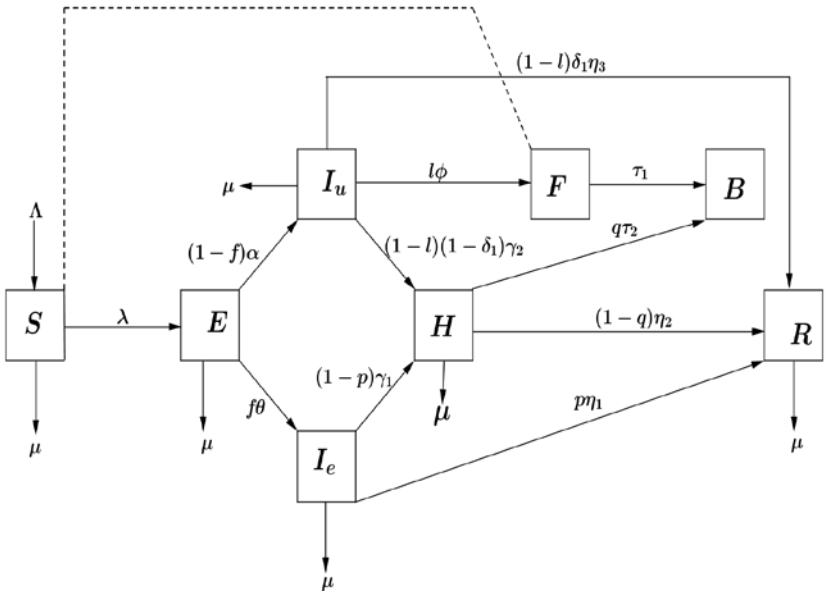

Figure 1: When susceptible people (S) are infected, they progress to the exposed, but not infectious, state (E). From there, they become infectious (I) to the susceptible population. An infected person whether I_u or I_e either enters a medical facility (H), or does not. If they do not enter a medical facility, they may recover (R) or die. When I_u die, they may have a traditional funeral (F), where others can be infected, or 'unsafe' burial. People in a medical facility are more likely to recover and if they die they have a safe burial. The dynamics are described by differential equations (1).

Table 1: Parameters and their description

Parameter	Value	Description	Source
Λ	10^6people year^{-1}	Recruitment rate	[24]
α	0.1/day	Rate at which Latent individuals progress to unaware infectious individuals	[25]
f	0.7	Fraction of Latent individuals who progress to aware infectious individuals	Estimated
θ	0.1/day	Rate at which Latent individuals progress to aware infectious individuals	[25]
p	0.3	Fraction of aware individuals who are recovered	Estimated
η_1	0.05/day	recovery rate for aware infectious individuals	[25]
η_2	$\frac{1}{15.88}$/day	recovery rate for Hospitalized individuals	[25]
η_3	$\frac{1}{25}$/day	recovery rate for unaware infectious individuals	Estimated
ϕ	0.33	Disease induced death rate of unaware individuals	Estimated
γ_1	0.2	Rate at which aware individuals are hospitalized	Estimated
γ_2	0.4	Rate at which unaware individuals are hospitalized	Estimated
τ_1	1/4.50 day	Duration of Traditional Funeral (unsafe burial rate)	[26]
τ_2	$\frac{1}{6.26\ weeks}$	Safe burial rate	[26]
q	0.7	Fraction of Hospitalized individuals who are buried safely	Estimated
ω_e	0.12 people weeks^{-1}	contact rate with aware infectious individuals	[26]
ω_u	0.1102 people weeks^{-1}	contact rate with unaware infectious individuals	[26]
ω_h	0.08 people weeks^{-1}	contact rate with hospitalized infectious individuals	[27]
ω_f	0.111 people weeks^{-1}	contact rate with infectious dead body	[27]
δ_1	0.8	Fraction of unaware individuals who are recovered	Estimated
l	0.8	Fraction of unaware individuals who die due to EVD	Estimated
μ	0.02 $year^{-1}$	Per capita natural mortality rate	[25]

2.1 Model Equations

From the description of the dynamics of Ebola and with the aid of the compartmental diagram in Figure 1, the following set of non-linear ordinary differential equations can be derived:

$$\frac{dS}{dt} = \Lambda - \left(\omega_e I_e + \omega_u I_u + \omega_f F + \omega_h H\right)S - \mu S \quad (1)$$

$$\frac{dE}{dt} = \left(\omega_e I_e + \omega_u I_u + \omega_f F + \omega_h H\right)S - a_1 E \quad (2)$$

$$\frac{dI_u}{dt} = a_2 E - a_3 I_u \quad (3)$$

$$\frac{dI_e}{dt} = a_4 E - a_5 I_e \quad (4)$$

$$\frac{dH}{dt} = a_6 I_e + a_7 I_u - a_8 H \quad (5)$$

$$\frac{dF}{dt} = a_9 I_u - \tau_1 F \quad (6)$$

$$\frac{dR}{dt} = a_{10} I_u + a_{11} I_e + a_{12} H - \mu R \quad (7)$$

where

$$a_1 = \mu + (1-f)\alpha + f\theta$$
$$a_2 = (1-f)\alpha$$
$$a_3 = \mu + (1-l)(1-\delta_1)\gamma_2 + l\phi + (1-l)\delta_1\eta_3$$
$$a_4 = f\theta$$
$$a_5 = \mu + p\eta_1 + (1-p)\gamma_1$$
$$a_6 = (1-p)\gamma_1$$
$$a_7 = (1-l)(1-\delta_1)\gamma_2$$
$$a_8 = \mu + (1-q)\eta_2 + \tau_2 q$$
$$a_9 = l\phi$$
$$a_{10} = (1-l)\delta_1\eta_3$$
$$a_{11} = p\eta_1$$
$$a_{12} = (1-q)\eta_2$$

It must be noted that a variable B does not appear in the system so we neglect equation with variable B,that is $\frac{dB}{dt} = \tau_1 F + q\tau_2 H$ is useless in our system. With this in mind we derived 7 equations above instead of 8 equations expected.

2.2 Basic Properties of the Mode

Since the model monitors human population, we need to show that all the state variables remain non-negative for all times. It is easy to show that the state variables of the model remain non-negative for all non-negative initial conditions.

Consider the biological feasible region

$D = \{(S, E, I_e, I_u, H, F, R) \in \mathbb{R}_+^7 : S + E + I_e + I_u + H + R \leq N\}$.

L***emma 1.*** *The closed region D is positively invariant and attracting.*

Proof. Adding equations (1-7) gives the rate of change of the total population.

$$\frac{dN}{dt} = \Lambda - \mu N \tag{8}$$

Thus, the total active human population N is bounded by $\frac{\Lambda}{\mu}$, so that $\frac{dN}{dt} = 0$ whenever $N(t) = \frac{\Lambda}{\mu}$.

It can be shown that

$$N(t) = \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) e^{-\mu t} \tag{9}$$

In particular $N(t) = \frac{\Lambda}{\mu}$, if $N(0) = \frac{\Lambda}{\mu}$,

Hence, the region is positively invariant and attracts all solution in$\mathbb{R}_+^7$.

3. Model Analysis

The model system (1-7) is analyzed qualitatively to get insights into its dynamical features which give better understanding of the impact control strategies on the transmission dynamics of Ebola virus.

3.1 Equilibria

Disease Free Equilibrium (DFE), E_0

The disease free equilibrium of the model system (1-7) is obtained by setting the right hand side of system (1-7) equal to zero, and in the absence of infection: $I_u = I_e = F = H = 0$ thus we get:

$$0 = \Lambda - \left(\omega_e I_e + \omega_u I_u + \omega_f F + \omega_h H\right) S - \mu S \tag{10}$$
$$0 = \left(\omega_e I_e + \omega_u I_u + \omega_f F + \omega_h H\right) S - a_1 E \tag{11}$$
$$0 = a_2 E - a_3 I_u \tag{12}$$
$$0 = a_4 E - a_5 I_e \tag{13}$$
$$0 = a_6 I_e + a_7 I_u - a_8 H \tag{14}$$
$$0 = a_9 I_u - \tau_1 F \tag{15}$$
$$0 = a_{10} I_u + a_{11} I_e + a_{12} H - \mu R \tag{16}$$

Solving system (10-16), a disease-free equilibrium of the system (1-7) is obtained as

$$E_0 = (S^0, E^0, I_u^0, I_e{}^0, F^0, H^0, R^0) = (\tfrac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0). \tag{17}$$

An endemic equilibrium

In the presence of infection, that is : $I_u \neq I_e \neq F \neq H \neq 0$ the model system (10-16) has a non-trivial equilibrium point, E_1 called the endemic equilibrium point which is given by

$E_1 = (S^*, E^*, I_u{}^*, I_e{}^*, H^*, F^*, R^*)$ that satisfies $S^*, E^*, I_u{}^*, I_e{}^*, H^*, F^*, R^* > 0$.

From the equilibrium equations we can show that E_1 exists with

$$S^* = \frac{\Lambda}{\mu R_e}$$

For E_1 to exist in the feasible region D, the necessary and sufficient condition is that:

$0 < S^* < \frac{\Lambda}{\mu}$ or equivalent $\frac{\Lambda}{\mu S^*} \geq 1$ (18)

Define

$$R_e = \frac{\Lambda}{\mu S^*}$$

$$S^* = \frac{\Lambda}{\mu R_e}$$

Theorem 1

(a) *The model system* (1-7) *always has a disease-free equilibrium* $E_0(\frac{\Lambda}{\mu}, 0,0,0,0,0,0)$

(b) *If* $R_e < 1$, *there is no endemic equilibrium for model* (1-7).

(c) *If* $R_e > 1$, *there exits a unique endemic equilibrium* $E_1(S^*, E^*, I_u{}^*, I_e{}^*, H^*, F^*, R^*)$ *given by* the *equation(*19).

$$\boldsymbol{S^* = \frac{\Lambda}{\mu R_e}}$$

$$\boldsymbol{E^* = \frac{\lambda^*}{a_1} = \left(\frac{\Lambda(R_e - 1)}{a_1 R_e}\right)}$$

$$\boldsymbol{I_u^* = \frac{a_2}{a_3} E^* = \frac{a_2}{a_3}\left(\frac{\Lambda(R_e - 1)}{a_1 R_e}\right)}$$

$$\boldsymbol{I_e^* = \frac{a_4}{a_5} E^* = \frac{a_4}{a_5}\left(\frac{\Lambda(R_e - 1)}{a_1 R_e}\right)}$$

$$H^* = \frac{a_4}{a_5}E^* = \left(\frac{a_4 a_6}{a_5} + \frac{a_7 a_2}{a_3}\right)\left(\frac{\Lambda(R_e - 1)}{a_1 R_e}\right) \tag{19}$$

$$F = \frac{a_2 a_9}{a_3 \tau_1}\left(\frac{\Lambda(R_e - 1)}{a_1 R_e}\right)$$

$$R^* = \frac{1}{\mu}\left(\frac{a_{10} a_2}{a_3} + \frac{a_{11} a_4}{a_5} + a_{12}\left(\frac{a_4 a_6}{a_5} + \frac{a_7 a_2}{a_3}\right)\right)\left(\frac{\Lambda(R_e - 1)}{a_1 R_e}\right)$$

If $R_e > 1$ then $S^*, E^*, I_u{}^*, I_e{}^*, H^*, F^*, B^*, R^* > 0$ and endemic equilibrium exist. Then R_e is a threshold parameter that determines the number of equilibria. We will show in Section (3.2) that R_e is the basic reproduction number.

Proposition 1. If $R_e < 1$ then E_0 is the only equilibrium of the system (1-7); if $R_e > 1$, then there are two equilibria, disease free equilibrium, E_0 and a unique endemic equilibrium, E_1.

3.2 The Reproduction Number, R_0

The basic reproduction number denoted by R_0 is the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness [27].The basic reproduction number is an important non-dimensional quantity in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. Thus, whether a disease becomes persistent or dies out in a community depends on the value of the reproduction number, R_0. Furthermore, stability of equilibria can be analyzed usingR_0; if $R_0 < 1$ it means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when $R_0 > 1$, every infectious individual will cause more than one secondary infection and hence the disease will invade the population. A large number of R_0 may indicate the possibility of a major epidemic. For the case of a model with a single infected class, R_0 is simply the product of the infection rate and the mean duration of the infection.

In this paper, the reproductive number accounts for the average number of new Ebola cases generated by a single Ebola infected individual (either from unaware infectious individuals, aware infectious individuals, Hospitalized or dead body from traditional Funeral) introduced into a wholly susceptible population.

Due to complicated epidemics in our model, we compute the reproduction number, R_e using the next generation operator approach by [28]. This method is described as follows:

Assume there are *n* compartments so that the first *m* compartments correspond to infected individuals.

Let $\mathcal{F}_i(x)$ be the rate of appearance of new infections in compartment i. The disease transmission model consists of the system of equations

$$x'_i = f_i(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x)$$

where

$$\mathcal{V}_i(x) = \mathcal{V}_i^-(x) - \mathcal{V}_i^+{}_i(x)$$

One other important property is to obtain the disease free point x_0 . We then compute the matrices F and V which are $m \times m$ matrices, where m represent the infected classes, defined by $F = \frac{\partial \mathcal{F}_i(x_0)}{\partial x_j}$ and $V = \frac{\partial \mathcal{V}_i(x_0)}{\partial x_j}$ with $1 \le i, j \le m$ and F is nonnegative and V is non-singular M -matrix (a matrix with inverse, belonging to the class of positive matrices). Since F is nonnegative and V is nonsingular, then V^{-1} is nonnegative and also FV^{-1} is nonnegative. We then compute the matrix FV^{-1}, defined as the next generation matrix [28].The basic reproduction number (reproduction ratio) R_0 is then defined as $R_0 = \rho(FV^{-1})$ where $\rho(A)$ is the spectral radius of matrix A,(or the maximum modulus of the eigenvalues of A). By using the method described above, we establish local stability of the basic model using the basic reproduction number (R_0) as follows,

From the system Eq. (1-7) we define $\mathcal{F}_i$ and $\mathcal{V}_i$ as

$$\mathcal{F}_i = \begin{bmatrix} \lambda S \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \mathcal{V}_i = \begin{bmatrix} a_1 E \\ -(a_2 E - a_3 I_u) \\ -(a_4 E - a_5 I_e) \\ -(a_6 I_e + a_7 I_u - a_8 H) \\ -(a_9 I_u - \tau_1 F) \end{bmatrix}$$

We differentiate $\mathcal{F}_i$ with respect to S, E, I_u, I_e, F, R to get

$$F = \begin{bmatrix} 0 & \omega_e S^0 & \omega_e S^0 & \omega_h S^0 & \omega_f S^0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

We differentiate $\mathcal{V}_i$ with respect to S, E, I_u, I_e, F, R to get

$$V = \begin{bmatrix} a_1 & 0 & 0 & 0 & 0 \\ -a_2 & a_3 & 0 & 0 & 0 \\ -a_4 & 0 & a_5 & 0 & 0 \\ 0 & -a_7 & -a_6 & a_8 & 0 \\ 0 & -a_9 & 0 & 0 & \tau_1 \end{bmatrix}$$

We find the inverse of V and get

$$V^{-1} = \begin{bmatrix} \frac{1}{a_1} & 0 & 0 & 0 & 0 \\ \frac{a_2}{a_1 a_3} & \frac{1}{a_3} & 0 & 0 & 0 \\ \frac{a_4}{a_1 a_5} & 0 & \frac{1}{a_5} & 0 & 0 \\ \frac{a_7 a_2 a_5 + a_6 a_4 a_3}{a_1 a_3 a_8 a_5} & \frac{a_7}{a_3 a_8} & \frac{a_6}{a_8 a_5} & \frac{1}{a_8} & 0 \\ \frac{a_9\, a_2}{a_1 a_3 \tau_1} & \frac{a_9}{a_3 \tau_1} & 0 & 0 & \frac{1}{\tau_1} \end{bmatrix}$$

$$FV^{-1} = \left[\frac{\partial \mathcal{F}_i(x_0)}{\partial x_j}\right]\left[\frac{\partial \mathcal{V}_i(x_0)}{\partial x_j}\right]^{-1}$$

$$FV^{-1} = \begin{bmatrix} 0 & \omega_e S^0 & \omega_e S^0 & \omega_h S^0 & \omega_f S^0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{a_1} & 0 & 0 & 0 & 0 \\ \frac{a_2}{a_1 a_3} & \frac{1}{a_3} & 0 & 0 & 0 \\ \frac{a_4}{a_1 a_5} & 0 & \frac{1}{a_5} & 0 & 0 \\ \frac{a_7 a_2 a_5 + a_6 a_4 a_3}{a_1 a_3 a_8 a_5} & \frac{a_7}{a_3 a_8} & \frac{a_6}{a_8 a_5} & \frac{1}{a_8} & 0 \\ \frac{a_9\, a_2}{a_1 a_3 \tau_1} & \frac{a_9}{a_3 \tau_1} & 0 & 0 & \frac{1}{\tau_1} \end{bmatrix}$$

$$= \begin{bmatrix} R_{eu} + R_{ee} + R_{eh} + R_{ef} & \frac{\omega_e S^0}{a_3} + \frac{a_7 \omega_h S^0}{a_3 a_8} + \frac{a_9 \omega_f S^0}{a_3 \tau_1} & \left(\frac{\omega_e S^0}{a_5} + \frac{a_6 \omega_h S^0}{a_8 a_5}\right) & \frac{\omega_h S^0}{a_8} & \frac{\omega_f S^0}{\tau_1} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

where

$$R_{ee} = \frac{a_4 S^0 \omega_e}{a_1 a_5}, R_{eu} = \frac{a_2 S^0 \omega_u}{a_1 a_3}, R_{eh} = \frac{(a_7 a_2 a_5 + a_3 a_4 a_6) S^0 \omega_h}{a_3 a_1 a_5 a_8}, R_{ef} = \frac{a_2 a_9 S^0 \omega_f}{a_1 a_3 \tau_1}$$

The eigenvalues, λ of FV^{-1} can be computed from the characteristic equation:

$$|FV^{-1} - \lambda I| = 0$$

Direct computation gives:

$\lambda_1 = R_{eu} + R_{ee} + R_{eh} + R_{ef}$ and $\lambda_{2,3,4,5} = 0$

Obviously, λ_1 is the dominant eigenvalue and becomes equal to R_e of the model.

Therefore the effective reproduction number is given by

$$R_e = R_{eu} + R_{ee} + R_{eh} + R_{ef}$$
$$= \frac{a_4 S^0 \omega_e}{a_1 a_5} + \frac{a_2 S^0 \omega_u}{a_1 a_3} + \frac{(a_7 a_2 a_5 + a_3 a_4 a_6) S^0 \omega_h}{a_3 a_1 a_5 a_8} + \frac{a_2 a_9 S^0 \omega_f}{a_1 a_3 \tau_1}$$

where

R_{eu}, the contribution of unaware infectious individuals I_u;

R_{ee}, the contribution of aware infectious individuals I_e;

R_{eh}, the contribution of hospitalized infectious individuals H;

R_{ef}, the contribution resulting from manipulation of infected corpses F;

It is worth mentioning that in the absence of control strategies, we have $\theta = f = p = \gamma_1 = \gamma_2 = \eta_1 = \eta_2 = q = \tau_2 = 0$ so the effective reproduction number reduces to the basic reproduction number

$R_0 = R_{eh} + R_{ef}$.

$$R_0 = \frac{(a_7 a_2 a_5 + a_3 a_4 a_6) S^0 \omega_h}{a_3 a_1 a_5 a_8} + \frac{a_2 a_9 S^0 \omega_f}{a_1 a_3 \tau_1}$$

3.3 Local Stability of Disease-Free Equilibrium point (DFE)

We show that, the variation matrix $J(E_0)$ of model system (1-7) has negative trace and positive determinant. The partial differentiation of (1-7) with respect to S, E, I_u, I_e, F, R at the disease free e quilibrium gives:

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\omega_u S^0 & -\omega_e S^0 & -\omega_h S^0 & -\omega_f S^0 & 0 \\ 0 & -a_1 & \omega_u S^0 & \omega_e S^0 & \omega_h S^0 & \omega_f S^0 & 0 \\ 0 & a_2 & -a_3 & 0 & 0 & 0 & 0 \\ 0 & a_4 & 0 & -a_5 & 0 & 0 & 0 \\ 0 & 0 & a_7 & a_6 & -a_8 & 0 & 0 \\ 0 & 0 & a_9 & 0 & 0 & -\tau & 0 \\ 0 & 0 & a_{10} & a_{11} & a_{12} & 0 & -\mu \end{bmatrix}$$

We have the following stability result that shows R_e is a sharp threshold.

Proposition 2.

E_0 is locally asymptotically stable if $R_e < 1$ and is unstable if $R_e > 1$.

Proof

We want to show, when $R_e < 1$, that the Routh-Hurwitz conditions hold, namely,

$tr(J(E_0)) < 0$ and $det(J(E_0)) > 0$.

We have

$$trace\big(J(E_0)\big) = -2\mu - a_1 - a_3 - a_5 - a_8 - \tau_1 < 0$$

$$det\big(J(E_0)\big) = \mu^2[(a_3a_4\omega_e S^0 a_8\tau_1 + (a_3a_4a_6\omega_h + a_7a_2a_5)\tau_1\omega_h S^0 + a_2a_5a_8\tau_1 S^0\omega_u + a_9a_2a_5a_8 S^0\omega_f - a_3a_1a_5a_8\tau_1]$$

$$= a_3a_1a_5a_8\tau_1\mu^2\left(\left(\frac{a_4S^0\omega_e}{a_1a_5} + \frac{a_2S^0\omega_u}{a_1a_3} + \frac{(a_7a_2a_5 + a_3a_4a_6)S^0\omega_h}{a_3a_1a_5a_8} + \frac{a_2a_9S^0\omega_f}{a_1a_3\tau_1}\right) - 1\right)$$

$$= a_3a_1a_5a_8\tau_1\mu^2(R_e - 1)$$

where

$$R_e = \frac{a_4S^0\omega_e}{a_1a_5} + \frac{a_2S^0\omega_u}{a_1a_3} + \frac{(a_7a_2a_5 + a_3a_4a_6)S^0\omega_h}{a_3a_1a_5a_8} + \frac{a_2a_9S^0\omega_f}{a_1a_3\tau_1}$$

$$det\big(J(E_0)\big) = a_3a_1a_5a_8\tau_1\mu^2(R_e - 1)$$

Hence $det\big(J(E_0)\big) > 0$ under the condition that $R_e > 1$.

Hence the disease free E_0 is locally asymptotically unstable and so the disease prevails.

4. Simulation and Discussions

The main objective of this study was to model the transmission dynamics of Ebola virus disease with control strategies which included public health education campaigns, safe burial, hospitalization and therapeutic treatment. In order to support the analytical results, numerical results were presented with the aid of MATLAB programming language, we present graphical representations showing the variations in parameters with respect to effective reproduction number. In order to perform simulations, baseline values of parameters from Table 1 presented were used and unavailable data were assumed.

Figure 2 shows that, with the increase of infectious aware population there is a simultaneous increase of Ebola outbreak. Figure 3 shows that, with the increase of exposed population there is an increase of Ebola outbreak. It can be noticed from Figures 2 and 3 that there is a slight difference in severerity of the outbreak, that is in Figure 2 there is more infected people as compared to Figure 3, since the infected individuals in Figure 2 are infectious so they are likely to transmit the disease rapidly.

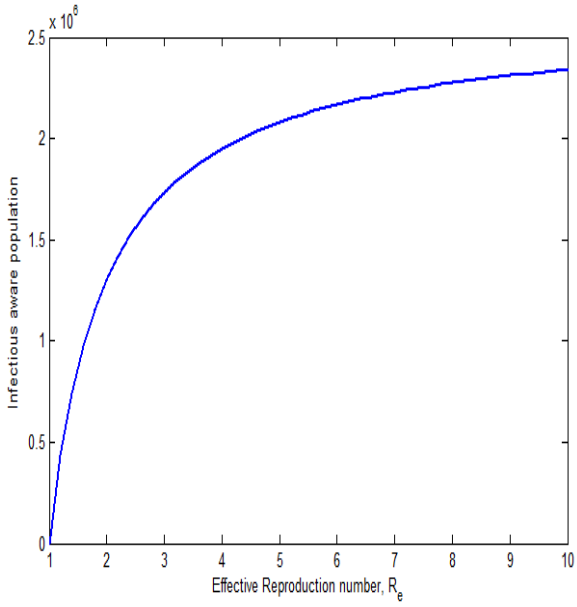

Figure 2: Variation of R_e with respect to Infectious aware population.

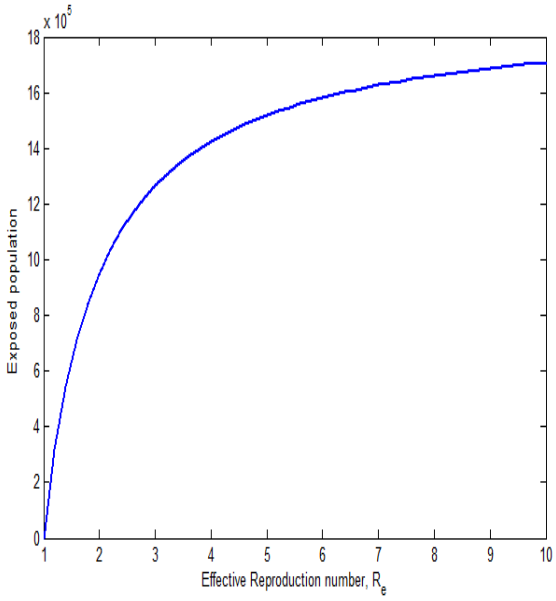

Figure 3: Variation of R_e with respect to Infectious aware population.

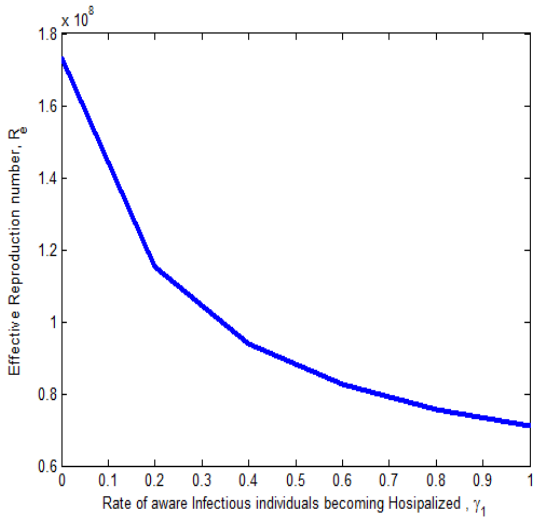

Figure 4: Variation of γ_1 with respect to effective reproduction number, R_e.

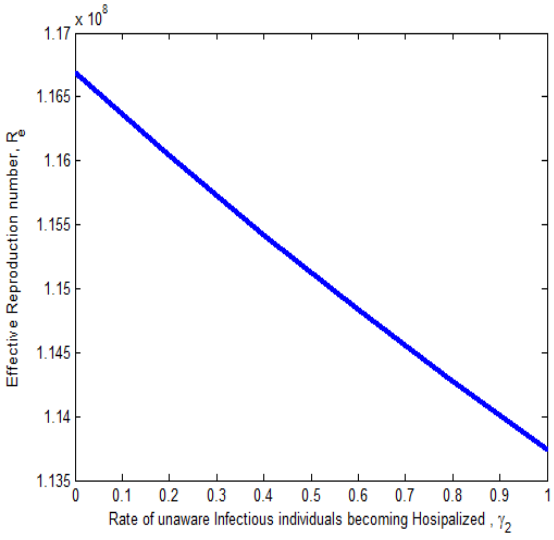

Figure 5: Variation of γ_2 with respect to effective reproduction number, R_e.

It is clear from Figure 4 that the more aware infectious individuals are hospitalized, the less the EVD epidemic. This is attributed by the fact that hospitalized people do not contact with usual healthier people until they recover. It can be seen from Figure 5 that there is a sharp decrease in EVD with an increase in hospitalization rate for unaware infectious individuals, this might be due the fact that unaware infectious individuals tend to transmit the disease more than those who are aware. This means that if we are at all to mitigate EVD outbreak then more effort must be put on quarantine of the infected population since quarantine minimize the spread of the disease.

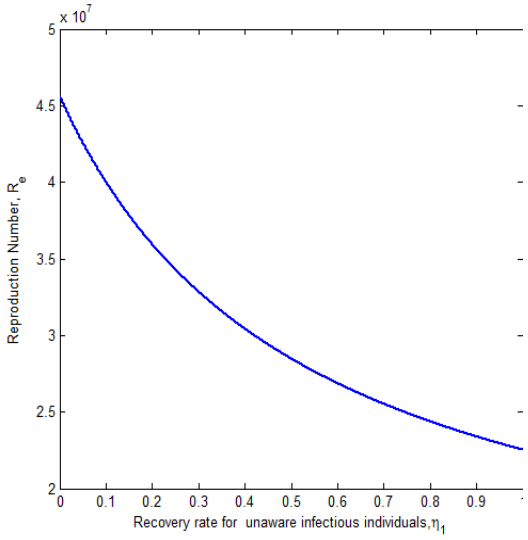

Figure 6: Effects of treatment, n_1 on effective reproduction number, R_e.

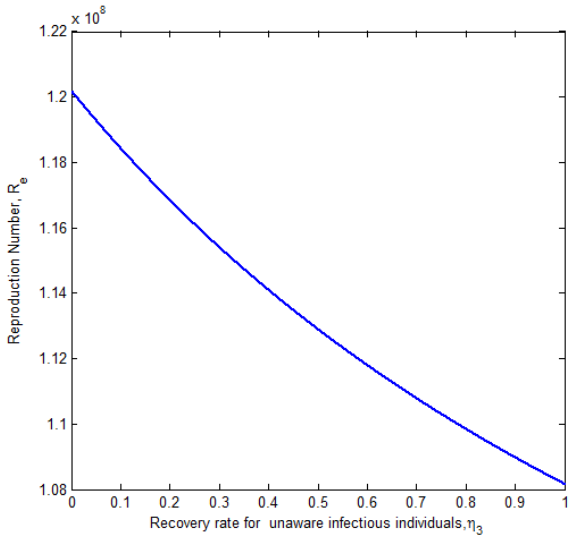

Figure 7: Effects of treatment, n_3 effective reproduction number, R_e.

It can be seen from Figure 6 that the EVD decreases with an increase of treatment rate of unaware infectious individuals. This result suggests the significance of therapeutic treatment on the infectious individuals. Sick people should seek medical treatment immediately after being identified in the community otherwise they may end up spreading the disease or else die. It can be seen from Figure 7 that the EVD decreases with an increase of recovery rate n_3 of unaware infectious individuals. This type of recovery might be as a result of pharmaceutical treatment or natural recovery.

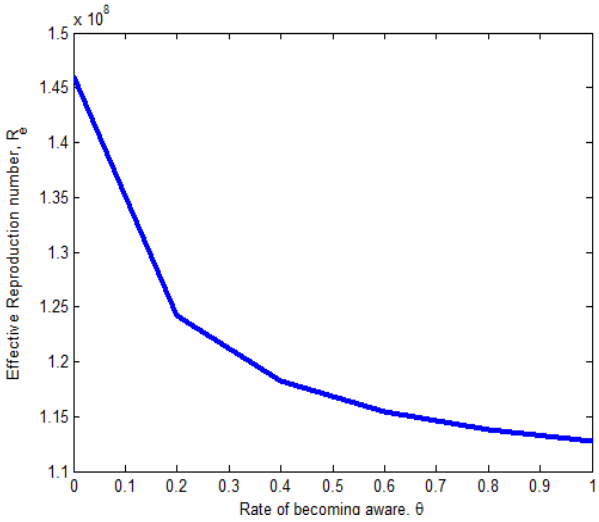

Figure 8: Variation of θ with respect to effective respect to reproduction number, R_e.

It is evident from Figure 8 that the raising rate of EVD awareness to infectious individuals minimizes the transmission dynamics of EVD. This is because aware individuals avoid more spreading through direct contact with the susceptible individuals, they get immediate medical attention and may have safe burial.

Figure 9 is a contour that depicts how the variation of recovery rates for aware, η_1 and unaware, η_3 individuals affects the reproduction number. It can be visualized that an increase in η_1 or η_3 causes a decrease in reproduction number. Since recovery of individuals tend to minimize the disease dynamics, more effort should be done on treating infected patients. It must be pointed out from the contour that less effort is needed to treat aware infectious individuals as compared to unaware ones.

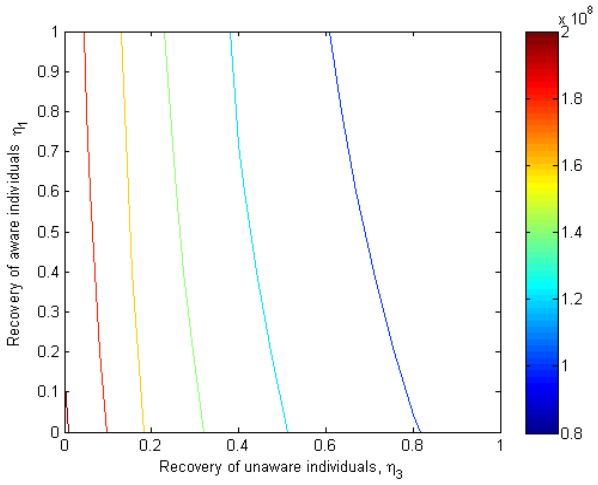

Figure 9: Variation effective reproduction number, R_e with respect to η_1 and η_3 .

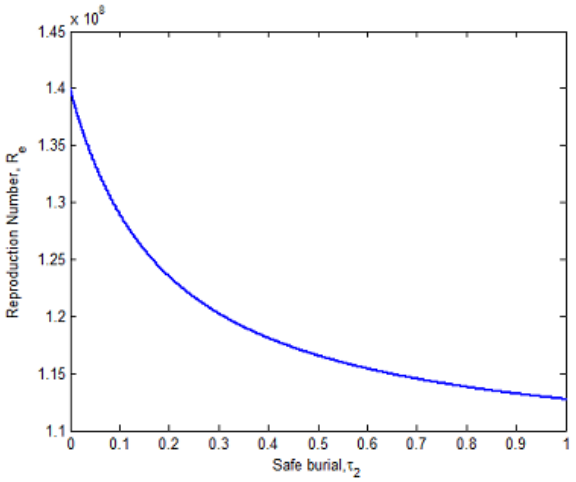

Figure 10: Variation effective reproduction number, R_e with respect to safe burial rate, τ_2 .

It can be observed from Figure 10 that an increase in safe burial practices tends to diminish the spread of EBV outbreak in the community. Thus there is a need for the community to stop traditional funerals especially in the affected regions.

5. Conclusion and Recommendations

In this paper, a deterministic mathematical model which incorporates person-to-person and person-to-corpse contact rate was presented and analyzed. Important mathematical features of the model such as the threshold for the epidemic, steady states, positivity and boundedness of solutions as well as the region of biological significance were determined. The model has a unique endemic equilibrium for $R_e > 1$.The disease free equilibrium point also exists and is unstable when this disease threshold is greater than unity.

Our findings suggest that, it is beneficial to minimize contact with Ebola patients, avoid touching dead body, encourage hospitalization of Ebola patients, safe burial practices, more training should be given to medical staff to specially handle Ebola virus disease and maximizing EBV awareness programs to the population at large. The study furthermore, recommends that there should be more international co-operation to prevent cross-border transmission of the disease. As has been studied by [29,30] , it must be pointed out that even though therapeutic treatment of both aware and unaware EBV patients is imperative to halt the transmission of this epidemic, however this strategy alone would have been insufficient to stop this epidemic from spreading through a population. This calls for a need of a combination of several control strategies if we are at all to eradicate this

epidemic.

We acknowledge the fact that this work may have shortfalls as follows. The model could be improved by incorporating the role of environment and bush meat in the transmission dynamics of EBV. The model has not taken into account the impact of hypothetical vaccine, which could show how vaccination could help to combat EVD, sensitivity analysis was not carried out in this work and no optimal control and cost effectiveness of the control measures were considered in this model which could perhaps yield more appealing results. However our great attempt in this work has laid a strong cornerstone to fill these gaps because it has improved our understanding of Ebola Transmission dynamics.

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