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Mathematical Modeling and Optimal Control of Ebola Virus Disease (EVD)

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Authors' contributions

This work was carried out in collaboration between both authors. Author AS performed the optimal control analysis, wrote the protocol and wrote the first draft of the manuscript. Author JL designed the mathematical model, managed the analyses of the study and managed the literature searches. Both authors read and approved the final manuscript.

Article Information

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ABSTRACT

In this paper, a nonlinear mathematical model is developed and analyzed to study the dynamics of Ebola virus (EVD) and the effects of some control strategies. The model validity is investigated and was found to be locally asymptotically stable when the basic reproduction number $(\Re_{0} < 1)$ and

unstable otherwise. Pontryagin's maximum principle is applied to obtain the optimality conditions. Numerical simulation was carried out and the results obtained indicate that a combination of all three control parameters is highly effective in containing the spread of the virus.

Keywords: Ebola virus; contact tracing; personal protective equipment; optimal control.

1. INTRODUCTION

Ebola virus disease (EVD), named after the Ebola River in Democratic Republic of the Congo

(formerly Zaire) is acknowledged to be greatly infectious disease with a high mortality rate [1]. The virus has a number of different strains, formerly known as Ebola hemorrhagic Fever [2]. But some Ebola patients did not present hemorrhage, thus, it is now referred to as Ebola virus disease [3]. There have been a number of cases over the years, the virus originated from Zaire in 1976 [4]. Since then up to 2008, the fatality rate of EVD victims was about 79% [5]. The continuing epidemic of EVD is affecting countries in Central and Western Africa. The first case of Ebola virus was in 1976 in northern Zaire, currently the Democratic Republic of Congo (DRC) [6].

The epidemic resulted to many cases of about 350 out of which more than two third losses their lives due to infection [5]. Regrettably, hospital personnel that attended to the patients were eventually exposed to the virus that leads to the loss of life. The outbreak was examined to determine the cause of the infection, though the patient bought, cooked, and ate bush meat (antelopes) hence, it could be the source of the infection [7]. Subsequently, outbreaks of Ebola virus do occur from time to time in Africa over the past 40 years. The highest fatality rate of the 19 out of 20 outbreak excluding the one in 2014 was (66.3%) though it may varies depending on the outbreak [8]. The human-to-human transmission of the virus occurs mostly through contact with body fluids [9]. Health workers and family of the diseased person are mostly vulnerable to the infection due to insufficient or lack of personal protective equipment (PPE) [10]. African traditional funeral practices that involve washing, touching and kissing the body is what compounded the problem. There are currently no strong pointers regarding the source of the virus but fruit bats of the Pteropodidae family are considered the natural host of the Ebola virus. It is also thought to transmit through monkeys, gorillas, and chimpanzees. However, the initial human-to-human transmission must occur through contact with the body fluids of an infected patient. Thus, Ebola is not airborne, as is the case with influenza, nor is it food or waterborne, as is the case with other diarrheal diseases (cholera, dysentery, or typhoid).

Further, the Ebola virus does not infect other individuals during the incubation period (the period between the initial infection and the onset of symptoms), which for the Ebola virus can be 2-21 days. As a result, if a person who has been in contact with an Ebola patient, or a patient suspected of Ebola presented fever, immediate quarantine, treatment, and management by the hospital can halt the spread of the outbreak. However, even after recovery, the virus may still be found in the body fluids of the patient for an extended period. In a report, the Ebola virus was detected in the semen of the patient three months after recovery [7]. Thus, it is important that the patient only can be released from isolation after confirming that the Ebola virus is no longer present. Although there are currently no established treatments or vaccines of Ebola available worldwide, symptomatic therapy and strict quarantine are adequate to prevent transmission of an Ebola.

Some research findings reveal that incubation period of EVD is between 2 and 21 days [11]. During these period, the virus infects body cells, duplicates and spurts out of the infected cells, creating EBOV glycoproteins that attach to the inside of blood vessels, rendering the blood vessels to be more penetrable. The increased penetrability causes the blood vessels to leak out blood [11]. The virus also attack the host's natural defense system, by infecting immune cells, a channel through which it is transported to other body parts and organs, such as the liver, kidney and brain. The disease causes these organs to fail, eventually, leading to death of the infected human [11].

Mathematical modeling of diseases has help immensely in combating the spread of infectious including EVD. [4] Propose a disease mathematical model on the spread of Ebola virus to estimate the basic reproduction number in the absence of control interventions. Further, they introduce some control parameters of education, contact tracing and quarantine. [5] formulated a mathematical model of Ebola virus transmission in order to predict the epidemic trends and to evaluate intervention measures efficacy. [6] presented a mathematical model of Ebola virus that divides the population of interest into individuals in the community and those in the health care setting. The model incorporates notable crucial features associated with disease transmission, such as the interaction between members of the community and their health-care settings, the role of Ebola-deceased individuals, and traditional belief systems and customs. The results of their study shows that, in the absence of public health interventions, the 2014 EBOV outbreaks would have had a much higher public health burden in all the countries involved. Other models on the dynamics of the disease include the works of [7,8]. Therefore, the purpose of this paper, is to apply some control parameters by extending the work of [9] using Pontryagin's Maximum Principle in order to contain the spread of the virus.

The remaining part of the paper is organized as follows. In section 2, we describe the formulation of the model using system of ordinary differential equations. In section 3, some basic properties of the model are presented. In section 4, optimal control analysis in order to contain the spread of the virus was discussed in section 5 numerical simulation and conclusion of the paper was presented in section 6.

2. MODEL FORMULATION

The proposed Ebola model subdivides the total human population represented by N(t) into five mutually exclusive compartments. The susceptible population is divided into two subclasses of low-risk susceptible individuals represented by $S_1(t)$ and high-risk susceptible individuals represented by $S_2(t)$. Exposed individuals E(t), infected individuals I(t), and recovered individuals R(t) thus,

$$N(t) = S_{1}(t) + S_{2}(t) + E(t) + I(t) + R(t)$$
(1)

The first susceptible population includes those individuals that are at high risk of contracting the virus $S_{2}(t)$ from the infected individuals [10, 12]. These categories include the people involved in burial processes, healthcare workers and relatives of the infected individuals. The rest of the susceptible population $S_1(t)$ is considered to be at a low risk of acquiring the virus. The susceptible humans are recruited through birth and immigration at a constant rate π . All newly recruited individuals are assumed to be susceptible as the virus is not transmitted through vertical transmission. The parameter auis assumed to be the fraction of recruited individuals who are at a high risk of acquiring the infection. While the remaining $(1 - \tau)$ are those with low risk of acquiring the disease. The lowrisk vulnerable population acquires infection at a rate λ while the high-risk population acquires infection at a rate ${}_{\mathcal{E}\!\lambda}$ as $\mathcal E$ is the modification parameter for the class. All the five classes are reduced through natural death μ and infected class has additional mortality rate due to infection δ .

The population of exposed class increases after the two susceptible populations of (S_1, S_2) acquires infection from the infected individuals at a rate $\lambda_{\mathcal{L}}\mathcal{E}\lambda$ respectively. The class is reduced

due to the manifestation of the symptoms of the disease at a rate α . Infected class is increase when the exposed individuals manifest the symptoms of the disease at a rate α . The class is reduced through natural death and disease induced death (μ , δ) respectively. The class is also reduced when the infected individuals either died or recovered from the infection at a rate φ . From the model formulations and assumptions above with the schematic diagram below the system of ordinary differential equations are presented in equation (2). Fig. 1 below shows a schematic diagram of the model utilized in this work.



Fig. 1. The Schematic diagram of the model

$$\frac{dS_1}{dt} = \pi (1 - \tau) - \beta S_1 I - \mu S_1$$

$$\frac{dS_2}{dt} = \pi \tau - \beta \varepsilon S_2 I - \mu S_2$$

$$\frac{dE}{dt} = \beta (S_1 + \varepsilon S_2) I - (\mu + \alpha) E$$

$$\frac{dI}{dt} = \alpha E - (\varphi + \mu + \delta) I$$

$$\frac{dR}{dt} = \varphi I - \mu R$$
(2)

3. BASIC PROPERTIES OF THE MODEL

3.1 Invariant Region

The equation 2 above monitors' human populations hence, it is necessary to consider that the associated population sizes can never be negative. The equation 2 should be considered in a feasible region where such property (non-negative) is conserved. Thus, in this section we discuss the invariant region. Recall that invariant region indicates the nonnegativity of the total size of a given population.

Definition: A set M is invariant if and only if for all $x \in M$, $\phi(x,t) \in M$ for all t. A set is positively (negatively) invariant if for all $x \in M$, $\phi(x,t) \in M$ for all t > 0 (t < 0) [13,14, 15].

From equation (2) we have that,

$$N = S_1 + S_2 + E + I + R \tag{3}$$

This formulation considered human population, in the absence of the (EVD), it implies that $\delta = 0$ and thus,

$$\frac{dN}{dt} \le \pi - \mu N - \delta I \le \pi - \mu N$$

Equation (2) has solutions which is contained in the feasible region,

 $\Omega = \Omega_h$

To show this, the approach of [16] will be followed to establish the invariant region as follows:

Let $(S_1, S_2, E, I, R) \in {5 \atop +}$ be any solutions of the system with nonnegative initial conditions as.

$$\frac{dN}{dt} \le \pi - \mu N \tag{4}$$

Separating the variables we have,

$$\frac{dN}{\pi - \mu N} \le dt$$

Taking the integral of both sides, thus,

$$\int \frac{dN}{\pi - \mu N} \leq \int dt$$
$$-\frac{1}{\mu} \ln(\pi - \mu N) \leq t + c$$
$$\ln(\pi - \mu N) \geq -\mu(t + c)$$

Taking the exponential of both sides it gives,

$$\left(\pi - \mu N\right) \ge A e^{-\mu t} \tag{5}$$

Where A is a constant, by applying the initial condition N(t) = N(0)

Thus,

 $A = \pi - \mu N(0)$

Substituting the above expression into equation (5) will give:

$$\left(\pi - \mu N(\mathbf{t})\right) \ge \left(\pi - \mu N(\mathbf{0})\right) e^{-\mu t} \tag{6}$$

By making N (t) the subject in (6) we obtain:

$$N(t) \le \frac{\pi}{\mu} - \left[\frac{\pi - \mu N(0)}{\mu}\right] e^{-\mu t}$$
(7)

From equation (7), as $t \to \infty, 0 \le N(t) \le \frac{\pi}{\mu}$,

therefore, the feasible solution set of the equation (2) is part of the region:

$$\Omega = \left\{ \left(S_1, S_2, E, I, R \right) \in {}^{5}_{+} : S_1 + S_2 + E + I + R \le \frac{\pi}{\mu} \right\}$$
(8)

In this case, whenever $N \leq \frac{\pi}{\mu}$, every solution with the initial condition in \int_{+}^{5} remains in that region for t > 0. Thus, the region Ω is positively invariant with respect to equation (2) and the model is epidemiologically meaningful in the domain Ω . Hence, it is sufficient to study the dynamics of the model in Ω [17].

3.2 Computation of the Basic Reproduction Number

The basic reproduction number \Re_0 is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population. The linear stability of the disease can be established using the next generation operator method [18,19] on the equation (2). The matrix F and V for the new infection terms and the remaining transfer terms are individually given by,

$$F = \begin{pmatrix} 0 & \beta(S_1 + \varepsilon S_2) \\ 0 & 0 \end{pmatrix}, V = \begin{pmatrix} \mu + \alpha & 0 \\ -\alpha & \varphi + \mu + \delta \end{pmatrix}$$

At disease free-equilibrium point,

$$F = \begin{pmatrix} 0 & \frac{\beta\pi(1-\tau)}{\mu} + \frac{\beta\varepsilon\pi\tau}{\mu} \\ 0 & 0 \end{pmatrix}$$

And inverse of V is given by,

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu + \alpha} & 0\\ \frac{\alpha}{(\mu + \alpha)(\varphi + \mu + \delta)} & \frac{1}{(\varphi + \mu + \delta)} \end{pmatrix}$$

It follows that the basic reproduction number \Re_0 of the equation (2) is given by,

$$\Re_{0} = \frac{\beta \pi \alpha \left(1 + \varepsilon \tau - \tau\right)}{\mu (\mu + \alpha) (\varphi + \mu + \delta)} \tag{9}$$

Moreover, with theorem 2 by [18], the following result is obtained. The DFE of the equation (2) is locally asymptotically stable (LAS) if $\mathfrak{R}_0 < 1$ and unstable if $\mathfrak{R}_0 > 1$ [18].

4. OPTIMAL CONTROL ANALYSIS

Optimal control is a procedure of establishing control and state trajectories for a dynamic scheme over a period of time in order to minimize a performance index [20,21]. The problem needs a performance index or cost functional J[x(t), u(t)], a set of state variables $x(t) \in X$, a set of control variables $u(t) \in U$ in a time t with $t_0 \le t \le t_f$. The main aim is to find a piecewise continuous control u(t) and the related state variables x(t) to minimize a given objective functional [21].

Three controls measures of (protective materials) u_1 used for people involved in burial processes, healthcare workers and relative of the infected individuals. Contact tracing u_2 for individuals that has contact with infected individuals. Lastly,

 u_2 for treating infected individuals with (EVD)

are considered in order to contain the spread of the virus in a community. The three time dependent control parameters are used to extend the model (2) in order to achieve the eradication of the virus in finite time in a community.

The higher risk vulnerable individuals that include the medical personnel and those involve in the burial processes of the victims of infection. These categories of individuals are protected from infection by using personal protective equipment at a rate $\pi \tau (1-u_1)$ where $u_1 (0 \le u_1 \le 1)$ is the control efforts for using personal protective equipment (PPE). Individuals infected with the virus moved to the exposed class at a rate where $(1 - u_2)(\beta S_1 + \beta \varepsilon S_2)$ $u_2 (0 \le u_2 \le 1)$ represent control effort for contact tracing. While individuals that developed symptoms of infection will immediately be isolated and start receiving treatment at a rate $(1-u_3)\alpha$ where $u_3(0 \le u_3 \le 1)$ represent the control effort for treatment. Thus, putting the above formulations and assumptions together gives the following (EVD) model in the form of ordinary differential equations below,

$$\frac{dS_1}{dt} = \pi (1-\tau) - \beta S_1 I - \mu S_1$$

$$\frac{dS_2}{dT} = (1-u_1) \pi \tau - \beta \varepsilon S_2 I - \mu S_2$$

$$\frac{dE}{dt} = (1-u_2) (\beta S_1 + \beta \varepsilon S_2) I - \mu E - (1-u_3) \alpha E$$

$$\frac{dI}{dt} = (1-u_3) \alpha E - (\varphi u_3 + \mu + \delta) I$$

$$\frac{dR}{dt} = \varphi u_3 I - \mu R$$
(10)

When the control is time dependent the disease free equilibrium no longer exists. Hence, we apply the Pontryagin's Maximum Principle to determine the conditions under which eradication of the disease can be achieved in finite time. We seek to minimize the number of higher risk susceptible individuals, exposed and infective individuals and the cost of applying protective equipment, contact tracing and treatment controls. The objective functional that we consider is given by,

$$J = \min_{u_1, u_2, u_3} \int \begin{pmatrix} A_1 S_2 + A_2 E + A_3 I \\ + B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 \end{pmatrix} dt$$
(11)

Subject to the model equation (10),

Where A_1S_2 represents the weight of high-risk susceptible individuals and A_2E, A_3I are the cost related with a number E of exposed individuals and I of infected individuals. The time T, is the period of the intervention. The term $B_1 u_1^2$ is the cost associated with personal protective equipment, $B_2 u_2^2$ is the cost of contact tracing and $B_3 u_3^2$ the cost of treatment. The coefficients $A_1, A_2, A_3, B_1, B_2, B_3$ are positive weights to balance the factors. Linear functions was chosen for the cost on higher-risk individuals A_1S_2 and cost of infection $A_2 E$, $A_3 I$ and quadratic forms for the cost on the controls $B_1u_1^2$, $B_2u_2^2$, $B_3u_3^2$ similar to what is found in the literature [22,23]. With the given objective function $J(u_1, u_2, u_3)$ the aim is to minimize the populations of higher-risk, exposed and infected individuals at the same time minimizing the controls costs of $(u_1(t), u_2(t), u_3(t))$. Hence, an optimal control (u_1^*, u_2^*, u_3^*) is obtained such that,

$$J\left(u_{1}^{*}, u_{2}^{*}, u_{3}^{*}\right) = \min_{u_{1}, u_{2}, u_{3}} \begin{cases} J\left(u_{1}, u_{2}, u_{3}\right) \\ |u_{1}, u_{2}, u_{3} \in U \end{cases}$$
(12)

Where the control set,

$$U = \left\{ \left(u_1^*, u_2^*, u_3^*\right) \middle| u_i : [0, 1] \text{ measurable } i = 1, 2, 3 \right\}$$

4.1 Pontryagin's Maximum Principle

The necessary conditions that an optimal control must satisfy come from the Pontryagin's Maximum Principle (Pontryagin [24]). This principle converts (10)–(11) into a problem of minimizing pointwise a Hamiltonian H, with respect to (u_1, u_2, u_3) . By optimal control theory,

let $\lambda_i(t)$ be adjoint variables with i = 1, ..., 5.

$$H = (A_{1}S_{2} + A_{2}E + A_{3}I + B_{1}u_{1}^{2}, B_{2}u_{2}^{2}, B_{3}u_{3}^{2}) + \lambda_{1} \{\pi(1-\tau) - \beta S_{1}I - \mu S_{1}\} + \lambda_{2} \{(1-u_{1})\pi\tau - \beta \varepsilon S_{2}I - \mu S_{2}\} + \lambda_{3} \{(1-u_{2})(\beta S_{1} + \beta \varepsilon S_{2})I - \mu E - (1-u_{3})\alpha E\} + \lambda_{4} \{(1-u_{3})\alpha E - (\varphi u_{3} + \mu + \delta)I\} + \lambda_{5} \{\varphi u_{3}I - \mu R\}$$
(13)

For the optimal control triple (u_1^*, u_2^*, u_3^*) that minimizes $J(u_1, u_2, u_3)$ over U there exist adjoint variables λ_i for i = 1, 2, ..., 5 satisfying adjoint system, transversality conditions and stationary values [24] as follows,

$$-\frac{d\lambda_{1}}{dt} = (\lambda_{1} - \lambda_{3})\beta I + \lambda_{3}\beta Iu_{2}$$

$$-\frac{d\lambda_{2}}{dt} = -A_{1} + (\lambda_{1} - \lambda_{2})\mu + (\lambda_{2} - \lambda_{3})\beta\varepsilon I + \lambda_{3}\beta u_{2}\varepsilon I$$

$$-\frac{d\lambda_{3}}{dt} = -A_{2} + (\lambda_{3} - \lambda_{4})(u_{3} - 1) + \lambda_{3}\mu$$

$$-\frac{d\lambda_{4}}{dt} = -A_{3} + (\lambda_{4} - \lambda_{5})\varphi u_{3} + (\lambda_{2} - \lambda_{3})\varepsilon S_{2}\beta + (\lambda_{1} - \lambda_{3})S_{1}\beta + \lambda_{3}u_{2}(S_{1} + \varepsilon S_{2})\beta$$

$$-\frac{d\lambda_{5}}{dt} = \lambda_{5}\mu$$
(14)

Hence, the transversality conditions is given by,

$$\lambda_i = 0 \text{ for } i = 1,...,5$$
 (15)

Using the transversality conditions $\lambda_i = 0$ for i = 1, ..., 5 where $0 < u_i < 1$ for i = 1, 2, 3, gives,

$$0 = \frac{dH}{dt} = 2B_{1}u_{1} - \lambda_{2}\pi\tau$$

$$0 = \frac{dH}{dt} = 2B_{2}u_{2} - \lambda_{3}(\varepsilon S_{2} + S_{1})\beta I$$

$$0 = \frac{dH}{dt} = 2B_{3}u_{3} + \lambda_{3}\alpha E - (\alpha E + \varphi I)\lambda_{4} + \varphi I\lambda_{5}$$
(16)

At this stage, the control u_1^*, u_2^*, u_3^* satisfy the optimality conditions,

$$u_{1}^{*} = \max\left\{0, \min\left(1, \frac{\lambda_{2}\pi\tau}{2B_{1}}\right)\right\}$$

$$u_{2}^{*} = \max\left\{0, \min\left(1, \frac{\lambda_{3}\left(\varepsilon S_{2} + S_{1}\right)\beta I}{2B_{2}}\right)\right\}$$

$$u_{3}^{*} = \max\left\{0, \min\left(1, \frac{\left(\lambda_{3} - \lambda_{4}\right)\alpha E + \left(\lambda_{4} - \lambda_{5}\right)\varphi I}{2B_{3}}\right)\right\}\right\}$$
(17)

Putting the bounds [0, 1] on the controls, it can be established that,

$$u_{1}^{*} = \begin{cases} 0 & \varsigma_{1}^{*} \leq 0 \\ \varsigma_{1}^{*} & if & 0 < \varsigma_{1}^{*} < 1, u_{2}^{*} \\ 1 & \varsigma_{1}^{*} \geq 1 \end{cases}$$
$$= \begin{cases} 0 & \varsigma_{2}^{*} \leq 0 \\ \varsigma_{2}^{*} & if & 0 < \varsigma_{2}^{*} < 1, u_{3}^{*} \\ 1 & \varsigma_{2}^{*} \geq 1 \end{cases}$$
$$= \begin{cases} 0 & \varsigma_{3}^{*} \leq 0 \\ \varsigma_{3}^{*} & if & 0 < \varsigma_{3}^{*} < 1 \\ 1 & \varsigma_{3}^{*} \geq 1 \end{cases}$$

Where,

$$\varsigma_{1}^{*} = \frac{\lambda_{2}\pi\tau}{2B_{2}}$$

$$\varsigma_{2}^{*} = \frac{\lambda_{3}\left(\varepsilon S_{2} + S_{1}\right)\beta I}{2B_{2}}$$

$$\varsigma_{3}^{*} = \frac{\left(\lambda_{3} - \lambda_{4}\right)\alpha E + \left(\lambda_{4} - \lambda_{5}\right)\varphi I}{2B_{3}}$$

$$(18)$$

Taking the second derivative of equation (16) gives,

$$\frac{\partial^2 H}{\partial u_1} = 2B_1, \ \frac{\partial^2 H}{\partial u_2} = 2B_2, \ \frac{\partial^2 H}{\partial u_3} = 2B_3 > 0$$

Thus, the problem is associated with minimization i.e. reducing the populations of higher-risk, exposed and infected individuals and cost of control since the second derivative of (16) is greater than zero [11].

5. NUMERICAL SIMULATION

In this section, the effect of the optimal control strategies on the spread of EVD is studied numerically. Using an iterative approach, the optimality system containing five ordinary differential equations from the state and adjoint equations attached with the three control The characterizations are solved. state differential equations with initial estimates for controls and the state are solved using fourth order Runge-Kutta scheme. Using the result of state and the given final time values, the ad joint system are solved using the fourth order Runge-Kutta scheme. The state and the adjoints system are used to update the three control strategies using the characterizations given by (17). The process is repeated in order to arrive at a desired result when the current state, adjoint, and control values converge sufficiently [25]. The influence of the following optimal control strategies on the spread of the disease in a population is examined numerically. Using two controls at a time while setting the other one to zero and finally considering the three controls at the same time. Range of parameters used in this section is on the interval of [0, 1].

- {Strategy} 1: Combination of use of personal protective equipment u_1 and contact tracing u_2 .
- {Strategy} 2: Combination of use of contact tracing u_2 and treatment u_2 .
- {Strategy} 3: Combination of use of personal protective equipment u_1 , contact tracing u_2 and treatment u_3 .

The following hypothetical weight factors are chosen as $(A_1 = 10, A_2 = A_3 = 5, B_1 = B_2 = B_3 = 6)$, with the parameter values in Table 1. The initial state variables are chosen as $(S_1 = 2,000,000, S_2 = 15,000, E = 0, I = 0, R = 0)$. In order to illustrate the effect of different optimal

In order to illustrate the effect of different optimal strategies the study considered a disease free population i.e. a point where all infective classes are assumed to be zero.

Parameter	Description	Value	Reference
π	Recruitment rate	400	[11]
μ	Natural death rate	0.00004	[11]
au	Fraction of susceptible at high risk of infection	0.30450	[11]
λ	Rate of acquiring infection by the low-risk susceptible	0.20000	Assumed
ε	Modification parameter	0.21000	[25]
δ	Death due to infection	0.51100	[6]
α	Rate of movement from exposed class to infected class	0.52390	[11]
arphi	Rate of recovery	0.53660	[25]

Table 1. Describing the parameters used in the model

5.1 Optimal Use of Personal Protective Equipment u_1 and Contact Tracing

 u_2

The personal protective equipment like (masks, gloves, goggles, and gowns etc.) u_1 and contact tracing u_2 are considered to enhance the objective function J as the control treatment u_3 is set to zero. The results obtained indicate a substantial variance in the high-risk population (s_2) with optimal control strategy contrary to (s_2) without control as found in [1] in their study on the impacts of interventions on an epidemic of

Ebola in Sierra Leone and Liberia. Increase use of PPE will help in eliminating the possibility of post-mortem infection from hospitalized patients due to inappropriate funereal practices. Fig. 2, indicates that the population of high-risk and exposed individuals are reduced due to the control interventions. The population of exposed are reduce as a results of contact tracing as we can see in Fig. 2b.

5.2 Optimal Use of Contact Tracing u_2 and Treatment u_3

The contact tracing u_2 and treatment u_3 are considered to enhance the objective function J as the control personal protective equipment u_1 .

is set to zero. The results obtained indicate a substantial variance in the exposed individuals E with optimal control strategy contrary to E without control as found in [2] in their study on the strategies for containing Ebola in West Africa. Fig. 3, indicates that the population of exposed and infected individuals are reduced due to the control interventions.



(a) High-risk individuals

(b) Exposed individuals

Fig. 2. Plots the effect of optimal use of personal protective equipment and contact tracing. (a) Shows the effect on high-risk individuals, (b). Shows the effect on exposed individuals

5.3 Optimal Use of Personal Protective Equipment u_1 , Contact Tracing u_2 and Treatment u_3

All the three control strategies (u_1, u_2, u_3) are used to optimize the objective function J. It was observed in Fig. 4 that there exist significant difference in the (S_2, E, I) with optimal control strategy as compared to (S_2, E, I) without control. In Fig. 4, it was observed that all the three classes are reduced due to the use of time dependent controls this is similar to what is found in [3] in their study on assessing the effectiveness of containment strategies using stochastic model of Ebola.



(a) Exposed individuals







(a) High-risk individuals



(b) Exposed individuals



(c) Infected individuals

Fig. 4. Plots the effect of optimal use of personal protective equipment, contact tracing and finally treatment on the spread of the virus. (a) Shows the effect on high-risk individuals, (b) Shows the effect on exposed individuals, (c) Shows the effect on the infected individuals

6. CONCLUSION

In this paper, a nonlinear mathematical model is developed and analyzed to study the dynamics of Ebola virus (EVD) and the effects of some control strategies. The model was found to be epidemiologically well posed after investigating its validity. Basic reproduction number was obtained and the model was found to be locally asymptotically stable when \Re <1 and unstable

otherwise. Conditions for optimal control were obtain, the problem is associated with minimization i.e. reducing the infected classes and class of high-risk susceptible individuals and cost of control as the second derivative of (16) is greater than zero. Numerical simulation results revealed that combination containing all the three control parameters of protective materials (u,),

contact tracing (u_2) and treatment (u_3) is the best in controlling the spread of Ebola virus disease.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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