

Optimal Control of Mathematical Modelling for Ebola Virus Population Dynamics in the Presence of Vaccination

^{1,3}Andrawus, J., ^{2,3}Abdulrahman, S., ³ Singh, R.V.K. and ⁴Manga, S.S.

¹Department of Mathematics,
Federal University Dutse,
Jigawa State, Nigeria

²Department of Mathematics,
Federal University Birnin Kebbi,
Kebbi State, Nigeria

³Department of Mathematics
Kebbi State University of Science and Technology Aliero,
Kebbi State, Nigeria

⁴Department of Biological Sciences,
Kebbi State University of Science and Technology Aliero,
Kebbi State, Nigeria

Abstract

Ebola virus is a severe often fatal illness in human, which is known to be very dangerous and highly infectious disease that seized many lives in west African countries. In this paper, a mathematical model for the population dynamics of Ebola virus diseases incorporating bats compartment, recovery due to immune response and vaccination was constructed. Pontryagin's maximum principle has been applied on the model to determine the necessary conditions for the optimal control of the Ebola virus in the presence of vaccination and fruit bats population, the optimality of most of the controls have been analyzed to use a small resources available in other to maximize the performance of the controls. The numerical simulation shows that with small resources if 0.1 percent of the people in a society can be vaccinated daily, Ebola can be mitigated in the environment.

Keywords: Ebola Virus, Mathematical Model, Optimal Control, Population Dynamics

INTRODUCTION

Ebola virus is a severe often fatal illness in human, a virological taxon species of the genus Ebola virus, a distinctive member of the ribonucleic acid virus family. Ebola virus disease is extremely infectious and deadly disease that seized many lives and create huge economic burden in the affected West African Countries. The virus was named after a river in DRC (Democratic Republic of Congo) and it was believed that the genesis was discovered in the year 1976 which also affect humans and primates (Murphy *et al.*, 1998; WHO, 2008; Abdulrahman *et al.*, 2015).

*Author for Correspondence

Ebola virus is one of the most infectious diseases which claimed many lives in West Africa (Richards *et al.*, 2006; Stahelin, 2014 and Towner *et al.*, 2006). The virus has caused so many deaths in West Africa particularly, in DRC (Formenty *et al.*, 1999; Baron *et al.*, 1983 and Martini and Siegert, 1971). From 1976 to date not less than fifty thousand confirmed cases of Ebola virus cases have been witnessed in West African Countries. Of these, more than 66% died as a result of the virus (WHO, 2021).

Following the history of Ebola virus in the human population through animals to human transmission, human to human transmission by direct contact with body fluids/secretions of infected persons is identified to be the principal mode of transmission (WHO, 2019; LMH, 2015 and WHO, 2014). The incubation period of Ebola virus disease (EVD) varies from 2 to 21 days with an observed average of 8 to 10 days. First symptoms are the sudden onset of fever fatigue, muscle pain, headache and sore throat. This is followed by vomiting, diarrhea, rash, symptoms of impaired kidney and liver function, and in some cases, both internal and external bleeding (e.g. oozing from the gums, blood in the stools) (WHO,2021 and PAH & WHO, 2014). There is no risk of transmission during the incubation period. However, despite considerable efforts, it remains unclear how the Ebola virus disease (EVD) is maintained and transmitted in nature and how the index case (first patient) is infected (Berge *et al.*, 2017).

Bats are majorly recognized as a reservoir of Ebola virus (Rhoubari *et al.*, 2018; Leroy *et al.*,2005; Pourrut *et al.*,2009;Hayman, 2016). Leroy *et al.*(2005) suggest that, consumption of contaminated fruits by bodily fluids of infected bats is one of the modes of transmission (Rhoubari *et al.*, 2018). Many years ago, many mathematical models have been proposed and developed to describe the dynamics of EVD (Collins, 2015; Christie *et al.*, 2015; Espinoza *et al.*, 2015; Kalu *et al.*, 2016;Lekone and Finkenstadt, 2006; Rachah and Torres, 2017).

However, these models have primarily treated human to human transmission. In addition, they do not take into account bats compartment, natural recovery due to immune response and vaccinated humans. For these mathematical and biological reasons, a global deterministic model for Ebola is proposed. This paper addressed only optimal control strategy on the formulated model.

1. Model formulation and basic properties

A mathematical modelling for the transmission dynamics of EVD by incorporating vaccine was developed. The model is subdivided into nine (9) compartments and eleven (11) state variables, namely: Susceptible humans (S_h); Latently infected humans (L_h); Infectious humans (I_h); Isolated humans (J_h); Remove individuals due to permanent recovery from infection(R_h); Ebola-induced death Dead bodies (humans) before burial (D_h); Virus in the environment (V); Non carrier bats (N_b); carrier bats (C_b); Total population of humans (T_h) and Total population of bats (T_b). The S_h compartment represents the at-risk humans that are prone to the disease. This compartment is generated from daily recruitment due to the birth and immigration given by Λ_h . They acquired infection and moved to L_h compartment through effective contact with humans in the I_h , D_h , V compartment and carrier bats C_b compartment given by the term

$$\lambda_h = \frac{\beta_1(1-\varepsilon)(I_h + \eta D_h)}{T_h} + \frac{\beta_2(1-\varepsilon)V}{T_h} + \frac{\beta_3(1-\varepsilon)C_b}{T_h} \quad (1)$$

The parameter β_1, β_2 and β_3 are the effective contact rate (human), effective contact rate of virus in the environment to human and effective contact rate of carrier bats to human respectively. η is the modification parameter associated with reduced contact with the dead body (human) compare to infectious humans; and the term $(1 - \varepsilon)$ reflect the impact of personal hygiene which is enhanced by public enlightenment campaign on Ebola transmission; $0 < \varepsilon < 1$. The compartment decrease due to vaccinated individuals at rate ρ .

The L_h compartment represents individuals that have been infected with the virus but have not yet developed clinical symptoms of Ebola and thus, cannot infect others. The compartment is generated from effective contact between S_h with I_h, D_h, V and C_b given by (1). They decrease at the rate γ due to natural recovery. They reduce at rate σ which is the progression rate. The I_h compartment represents the symptomatic individuals that are infected as well as infectious. The population is generated at rate σ from the L_h compartment due to development of clinical symptoms of Ebola by members of the L_h compartment. They diminished at rates φ and δ_1 due to isolation and disease induced dead rate respectively. The J_h compartment represents the humans who have developed clinical symptoms and have been isolated to be given treatment. The compartment is generated from I_h at the rate σ and φ due to isolation. The compartment decreases at the rates τ and δ_2 due to treatment and disease-induced death rates respectively.

The R_h compartment represents humans that recovered from the disease and it is assumed possesses permanent immunity against the disease. The compartment is generated from L_h, J_h and S_h at the rates γ, τ and ρ due to natural recovery, recovery due to treatment and vaccination rates respectively. The D_h compartment represents the dead bodies of those that die due to the disease from both J_h and I_h compartments. The class diminishes at the rate ϕ due to proper burial and the natural death of human occurs in all the human classes (except D_h) at the rate μ_h . The V compartment represents the virus in the environment. This compartment is generated due to the virus shade by I_h, D_h and C_b in the environment denoted by α_1, α_2 and α_3 respectively. The class decreases due to natural death of virus ξ .

The N_b compartment represents the Non carrier bats. This compartment is generated from daily recruitment given by Λ_b . They acquired infection and moved to C_b compartment through effective contact with carrier bats in the C_b compartment, given by the term

$$\lambda_b = \frac{\beta_4 C_b}{T_b} + \frac{\beta_5 V}{T_b} \quad (2)$$

where β_4 and β_5 effective contact rate bats to bats and effective contact rate of virus in the environment to bats respectively. The C_b compartment represents the Carrier bats which are capable of transmitting the disease to humans and among themselves. The compartment is generated from effective contact between N_b with C_b given by (2) as explained earlier. Both the bats compartments diminish at the rate δ_b and μ_b death of bats due to hunting and bats' natural death rate in both the bats' classes. Considering

all these assumptions and definition together gives the following equation for the rate of change of susceptible human with respect to time.

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h - \lambda_h S_h - (\rho + \mu_h) S_h \\
 \frac{dL_h}{dt} &= \lambda_h S_h - (\sigma + \gamma + q + \mu_h) L_h \\
 \frac{dQ_h}{dt} &= q L_h - (\sigma + \mu_h) Q_h \\
 \frac{dI_h}{dt} &= \sigma L_h - (\varphi + \delta_1 + \mu_h) I_h \\
 \frac{dJ_h}{dt} &= \sigma Q_h + \varphi I_h - (\tau + \delta_2 + \mu_h) J_h \\
 \frac{dR_h}{dt} &= \gamma L_h + \rho S_h + \tau J_h - \mu_h R_h \\
 \frac{dD_h}{dt} &= (\delta_1 + \mu_h) I_h + (\delta_2 + \mu_h) J_h - \phi D_h \\
 \frac{dN_b}{dt} &= \Lambda_b - \lambda_b N_b - (\delta_b + \mu_b) N_b \\
 \frac{dC_b}{dt} &= \lambda_b N_b - (\delta_b + \mu_b) C_b
 \end{aligned} \tag{3}$$

with $T_h = S_h + L_h + Q_h + I_h + J_h + R_h + D_h$ and $T_b = N_b + C_b$

Optimal Control

In this section Pontryagin's maximum principle have been applied on the model to determine the necessary conditions for optimal control of the EBV in the presence of vaccination and fruit bats population. A time dependent controls has been incorporated into the system (3) to determine the optimal strategy for controlling the disease. Hence we have

$$\begin{aligned}
 \dot{S}_h &= \Lambda_h - \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{T_h} S_h - (\rho + \mu_h + u_2) S_h \\
 \dot{L}_h &= \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{T_h} S_h - (\sigma + \gamma + \mu_h + u_3) L_h \\
 \dot{I}_h &= \sigma L_h - (\varphi + \delta_1 + \mu_h) I_h \\
 \dot{J}_h &= \varphi I_h - (\tau + \delta_2 + \mu_h + u_4) J_h \\
 \dot{R}_h &= (\gamma + u_3) L_h + (\rho + u_2) S_h + (\tau + u_4) J_h - \mu_h R_h \\
 \dot{D}_h &= (\delta_1 + \mu_h) I_h + (\delta_2 + \mu_h) J_h - \phi D_h \\
 \dot{V} &= \alpha_1 I_h + \alpha_2 D_h + \alpha_3 C_b - \xi V \\
 \dot{N}_b &= \Lambda_b - \frac{(1-u_5)(\beta_4 C_b + \beta_5 V)}{T_b} N_b - (\delta_b + \mu_b) N_b \\
 \dot{C}_b &= \frac{(1-u_5)(\beta_4 C_b + \beta_5 V)}{T_b} N_b - (\delta_b + \mu_b) C_b
 \end{aligned} \tag{4}$$

For this we consider the objective functional to be minimized as

$$G(u_1, u_2, u_3, u_4, u_5) = \int_0^{t_f} [z_1 I_h + z_2 J_h + z_3 D_h + z_4 C_b + Au_1^2 + Bu_2^2 + Cu_3^2 + Du_4^2 + Eu_5^2] dt \quad (5)$$

The optimal control functions $u_1(t)$, $u_2(t)$, $u_3(t)$, $u_4(t)$ and $u_5(t)$ are bounded, Lebesgue integrable functions, since control parameters are usually between zero and one. The control $u_1(t)$ and $u_5(t)$ signify the effort in preventing Ebola virus disease in humans and bats population respectively, while $(1 - u_1(t))$ and $(1 - u_5(t))$ signify the false effort in preventing Ebola virus disease in humans and bats population respectively. The control on vaccination of susceptible humans $u_2(t)$ satisfies $0 \leq u_2 \leq g_3$ where g_3 is the vaccine efficacy used on susceptible humans. The control on natural recovery rate of latently infected humans $u_3(t)$ satisfies $0 \leq u_3 \leq g_4$, where g_4 is the efficacy of immunity booster and the control on the treatment of isolated humans $u_4(t)$ satisfies $0 \leq u_4 \leq g_5$, where g_5 is the drug efficacy use for treatment of isolated humans. Our control problem involves a situation in which the number of EBV infected humans, bats and the cost of applying prevention and treatment controls $u_1(t)$, $u_2(t)$, $u_3(t)$, $u_4(t)$ and $u_5(t)$ are minimized subject to the system (5).

t_f is the final time and coefficient $z_1, z_2, z_3, z_4, A, B, C, D, E$ are the balancing cost factors due to scales and importance of the nine parts of the objective function. We seek to find an optimal control $u_1^*, u_2^*, u_3^*, u_4^*$ and u_5^* such that

$$G(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*) = \min \{G(u_1, u_2, u_3, u_4, u_5) | u_1, u_2, u_3, u_4, u_5 \in \Omega\} \quad (6)$$

where $\Omega = \{(u_1, u_2, u_3, u_4, u_5) \text{ such that } u_1, u_2, u_3, u_4, u_5 \text{ are measurable with } 0 \leq u_1 \leq 1, 0 \leq u_5 \leq 1, 0 \leq u_2 \leq g_3, 0 \leq u_3 \leq g_4, \text{ and } 0 \leq u_4 \leq g_5, \text{ for } t \in [0, t_f]\}$ is the control set.

The necessary condition that an optimal control solution must satisfy come from the Pontryagin *et. al.* (1986) maximum principle. Pontryagin *et. al.* (1986) converts (4) and (5) into a problem of minimizing point wise a Hamiltonian H with respect to u_1, u_2, u_3, u_4 and u_5 .

$$\begin{aligned} H = & z_1 I_h + z_2 J_h + z_3 D_h + z_4 C_b + Au_1^2 + Bu_2^2 + Cu_3^2 + Du_4^2 + Eu_5^2 \\ & + \lambda_1 \left\{ \Lambda_h - \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{T_h} S_h - (\rho + \mu_h + u_2) S_h \right\} \\ & + \lambda_2 \left\{ \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{T_h} S_h - (\sigma + \gamma + \mu_h + u_3) L_h \right\} \\ & + \lambda_3 \{ \sigma L_h - (\varphi + \delta_1 + \mu_h) I_h \} + \lambda_4 \{ \varphi I_h - (\tau + \delta_2 + \mu_h + u_4) J_h \} \\ & + \lambda_5 \{ (\gamma + u_3) L_h + (\rho + u_2) S_h + (\tau + u_4) J_h - \mu_h R_h \} + \lambda_6 \{ (\delta_1 + \mu_h) I_h + (\delta_2 + \mu_h) J_h - \phi D_h \} \\ & + \lambda_7 \{ \alpha_1 I_h + \alpha_2 D_h + \alpha_3 C_b - \xi V \} + \lambda_8 \left\{ \Lambda_b - \frac{(1-u_5)(\beta_4 C_b + \beta_5 V)}{T_b} N_b - (\delta_b + \mu_b) N_b \right\} \\ & + \lambda_9 \left\{ \frac{(1-u_5)(\beta_4 C_b + \beta_5 V)}{T_b} - (\delta_b + \mu_b) C_b \right\} \end{aligned} \quad (7)$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8$ and λ_9 are the adjoin variables or co-state variables. The system of equations is found by taking the appropriate partial derivatives of the Hamiltonian (7) with respect to the associated state variable.

Theorem 1: Given optimal controls $u_1^*, u_2^*, u_3^*, u_4^*, u_5^*$ and solutions $S_h, L_h, I_h, J_h, R_h, D_h, V, N_b, C_b$ of the corresponding state system (4) and (5) that minimize $G(u_1, u_2, u_3, u_4, u_5)$ over Ω . Then there exists adjoint variable $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8$ and λ_9 satisfying.

$$\frac{d\lambda_i}{dt} = -\frac{\delta H}{\delta j} \tag{8}$$

where $j = S_h, L_h, I_h, J_h, R_h, D_h, V, N_b, C_b$, $i = 1, 2, 3, \dots, 9$ and with transversality condition

$$\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = \lambda_4(t_f) = \lambda_5(t_f) = \lambda_6(t_f) = \lambda_7(t_f) = \lambda_8(t_f) = \lambda_9(t_f) = 0 \tag{9}$$

and

$$\begin{aligned} u_1^* &= \min \left\{ 1, \max \left(0, \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{2AT_h} (\lambda_2 - \lambda_1) \right) \right\} \\ u_2^* &= \min \left\{ 1, \max \left(0, \frac{(\lambda_6 - \lambda_1)S_h}{2B} \right) \right\} \\ u_3^* &= \min \left\{ 1, \max \left(0, \frac{(\lambda_6 - \lambda_2)L_h}{2C} \right) \right\} \\ u_4^* &= \min \left\{ 1, \max \left(0, \frac{(\lambda_6 - \lambda_5)J_h}{2D} \right) \right\} \\ u_5^* &= \min \left\{ 1, \max \left(0, \frac{(\beta_4 C_b + \beta_5 V)(\lambda_9 - \lambda_8)N_b}{2ET_b} \right) \right\} \end{aligned} \tag{10}$$

Proof: Corollary 4.1 of Feming and Rishel (1975) gives the existence of an optimal control due to the convexity of the integrand of G with respect to u_1, u_2, u_3, u_4 and u_5 a prior boundedness of the state solutions and the Lipschitz property of the state system with respect to the state variables. The differential equation governing the adjoint variables are obtained by differentiation of the Hamiltonian function, evaluated at the optimal control. Then the adjoint equations can be written as

$$\begin{aligned} \frac{d\lambda_1}{dt} = -\frac{\delta H}{\delta S_h} &= \lambda_1(\rho + \mu_h + u_2) - \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{T_h} (\lambda_2 - \lambda_1) \\ &+ \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)}{T_h} (\lambda_2 - \lambda_1) - \lambda_2(\rho + u_2) \\ &- \frac{(\beta_4 C_b + \beta_5 V)N_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2} \end{aligned} \tag{11}$$

$$\begin{aligned} \frac{d\lambda_2}{dt} = -\frac{\delta H}{\delta L_h} &= \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h^2} \\ &+ \lambda_2(q + \gamma + \sigma + \mu_h + u_3) - \lambda_6(\gamma + u_3) + \frac{(\beta_4 C_b + \beta_5 V)N_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2} \end{aligned} \tag{12}$$

$$\frac{d\lambda_3}{dt} = -\frac{\delta H}{\delta I_h} = -z_1 + \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h^2} - \frac{\beta_1 S_h(1-u_1)(1-\varepsilon)(\lambda_2 - \lambda_1)}{T_h} + \lambda_4(\varphi + \delta_1 + \mu_h) - \lambda_7(\delta_1 + \mu_h) \quad (13)$$

$$+ \frac{(\beta_4 C_b + \beta_5 V)N_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2}$$

$$\frac{d\lambda_4}{dt} = -\frac{\delta H}{\delta J_h} = -z_2 + \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h^2} + \lambda_5(\tau + \delta_2 + \mu_h + u_4) - \lambda_6(\tau + u_4) - \lambda_7(\delta_2 + \mu_h) + \frac{(\beta_4 C_b + \beta_5 V)N_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2} \quad (14)$$

$$\frac{d\lambda_5}{dt} = -\frac{\delta H}{\delta R_h} = -z_3 + \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h^2} - \frac{\beta_1 S_h(1-u_1)\eta(1-\varepsilon)(\lambda_2 - \lambda_1)}{T_h} + \frac{(\beta_4 C_b + \beta_5 V)N_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2} \quad (15)$$

$$\frac{d\lambda_6}{dt} = -\frac{\delta H}{\delta D_h} = \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h^2} + \frac{\beta_3(1-\varepsilon)C_b N_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2} + \lambda_6 \mu_h \quad (16)$$

$$\frac{d\lambda_7}{dt} = -\frac{\delta H}{\delta V} = \frac{(1-u_1)(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h^2} + \frac{\beta_2(1-\varepsilon)C_b N_b(1-u_5)(\lambda_8 - \lambda_7)}{T_b^2} - \frac{\beta_5 C_b(1-u_5)(\lambda_9 - \lambda_8)}{T_b^2} + \lambda_7 \xi \quad (17)$$

$$\frac{d\lambda_8}{dt} = -\frac{\delta H}{\delta N_b} = -\frac{(\beta_4 C_b + \beta_5 V)(1-u_5)(\lambda_9 - \lambda_8)}{T_b} + \lambda_8(\delta_b + \mu_b) \quad (18)$$

$$\frac{d\lambda_9}{dt} = -\frac{\delta H}{\delta C_b} = -z_4 - \frac{(\beta_4 C_b + \beta_5 V)(1-u_5)(\lambda_9 - \lambda_8)}{T_b} + \lambda_9(\delta_b + \mu_h) \quad (19)$$

Solving for $u_1^*, u_2^*, u_3^*, u_4^*$ and u_5^* subject to the constraints, the characterization (11)-(19) be solved

$$0 = \frac{\delta H}{\delta u_1} = 2Au_1^* - \frac{(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{T_h} \quad (20)$$

$$0 = \frac{\delta H}{\delta u_2} = 2Bu_2^* - (\lambda_6 - \lambda_1)S_h \quad (21)$$

$$0 = \frac{\delta H}{\delta u_3} = 2Cu_3^* - (\lambda_6 - \lambda_2)L_h \quad (22)$$

$$0 = \frac{\delta H}{\delta u_4} = 2Du_4^* - (\lambda_6 - \lambda_5)J_h \quad (23)$$

$$0 = \frac{\delta H}{\delta u_5} = 2Eu_5^* - \frac{(\beta_4 C_b + \beta_5 V)N_b(\lambda_9 - \lambda_8)}{T_b} \quad (24)$$

Solving for $u_1^*, u_2^*, u_3^*, u_4^*$ and u_5^* from (20)-(24) we obtain

$$u_1^* = \frac{(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{2AT_h} \quad (25)$$

$$u_2^* = \frac{(\lambda_6 - \lambda_1)S_h}{2B} \quad (26)$$

$$u_3^* = \frac{(\lambda_6 - \lambda_2)L_h}{2C} \quad (27)$$

$$u_4^* = \frac{(\lambda_6 - \lambda_5)J_h}{2D} \quad (28)$$

$$u_5^* = \frac{(\beta_4 C_b + \beta_5 V)(\lambda_9 - \lambda_8)N_b}{2ET_b} \quad (29)$$

by standard control arguments involving the bounds on the controls, we conclude

$$u_i^* = \begin{cases} 0 & \text{if } \xi_i^* \leq 0 \\ \xi_i^* & \text{if } \xi_i^* < 1 \\ 1 & \text{if } \xi_i^* \geq 1 \end{cases} \quad (30)$$

for $i = 1, 2, 3, 4, 5$ and where

$$\xi_1^* = \frac{(\beta_1(1-\varepsilon)(I_h + \eta D_h) + \beta_2(1-\varepsilon)V + \beta_3(1-\varepsilon)C_b)S_h(\lambda_2 - \lambda_1)}{2AT_h} \quad (31)$$

$$\xi_2^* = \frac{(\lambda_6 - \lambda_1)S_h}{2B} \quad (32)$$

$$\xi_3^* = \frac{(\lambda_6 - \lambda_2)L_h}{2C} \quad (33)$$

$$\xi_4^* = \frac{(\lambda_6 - \lambda_5)J_h}{2D} \quad (34)$$

$$\xi_5^* = \frac{(\beta_4 C_b + \beta_5 V)(\lambda_9 - \lambda_8)N_b}{2ET_b} \quad (35)$$

Hence the prove.

Table 1. Baseline numerical values for the parameters of system (3)

Parameter	Value(per day)	Source(s)
$\beta_1, \beta_2, \beta_3$	0.9, 0.7, 0.8	Estimated from Abdulrahman (2016)
β_4, β_5	0.5, 0.6	Estimated from Berge <i>et al.</i> (2018)
δ_1, δ_2	0.04227, 0.027855	Safi and Gumel, 2011; Leung <i>et al.</i> , 2004; Chowell <i>et al.</i> (2004)
δ_b	0.00014	Berge <i>et al.</i> (2018)
γ	0.03521	Safi and Gumel (2011)
φ	(0,1)	Control parameter
σ	0.1	Gumel <i>et al.</i> (2014)
τ	(0,1)	Control parameter
η	0.25	Berge <i>et al.</i> (2018)
ε	(0,1)	Control parameter
$\alpha_1, \alpha_2, \alpha_3$	0.11, 0.21, 0.25	Assumed
ρ	(0,1)	Control parameter
q	(0,1)	Control parameter
ϕ	(0,1)	Control parameter

Λ_h	136	Safi and Gumel (2011)
Λ_b	10	Berge <i>et al.</i> ,2018
μ_h	0.0000351	Safi and Gumel (2011)
μ_b	0.0011	Berge <i>et al.</i> (2018)

Numerical Simulations

In this section, simulations are performed under maple software in order to numerically illustrate the impact of sensitive parameters on the long run dynamics of EVD. We will simulate our model with most of the baseline parameters drawn from Table 1

Optimal Control Plotting

To determine the optimality of the model there is a need to simulate model (4) in other to minimize the cost of eradicating EBV in a society.

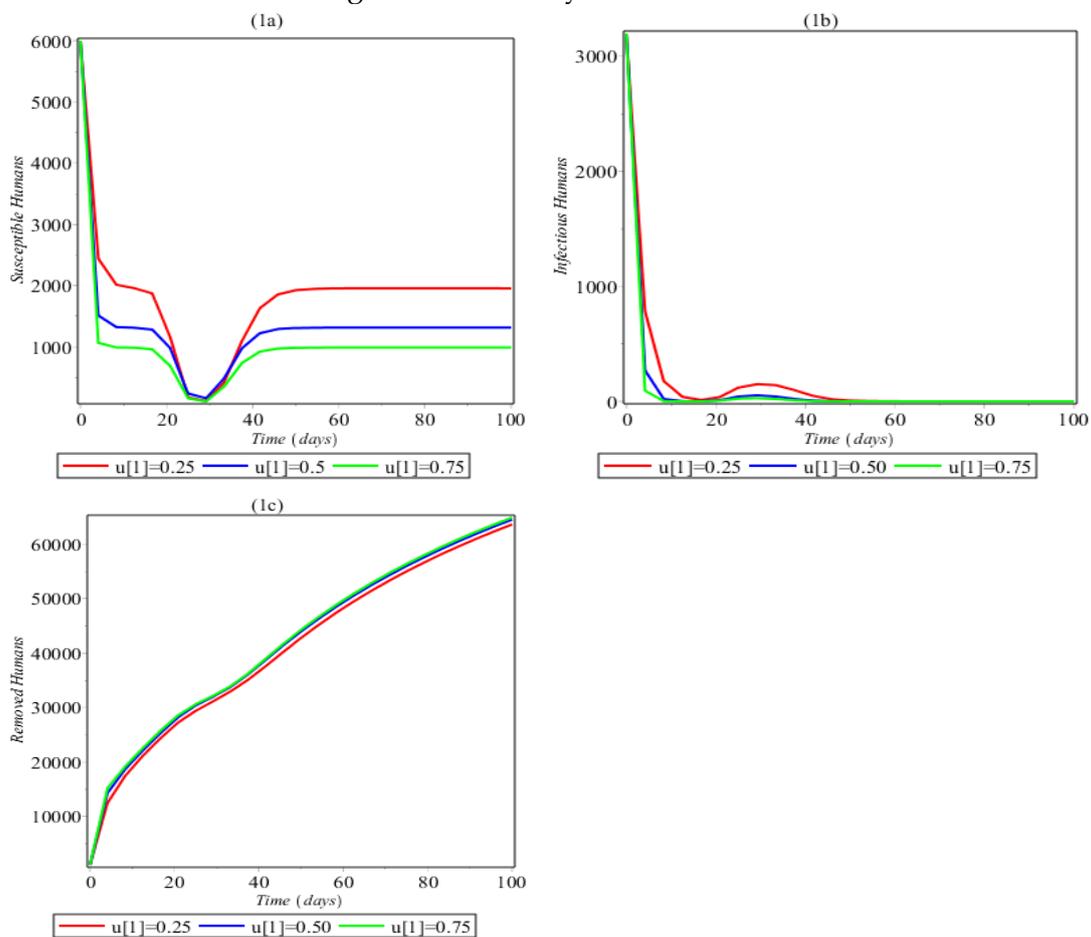


Figure 1a, Figure 1b and Figure 1c are comparison of the effectiveness of the effort in preventing EBV in humans. Variables and parameters used are as in table 1.

Fig 1a shows the Susceptible humans varying the rate of effort in preventing EBV, the susceptible reduces to a stable value and Fig 1b is showing the Infectious humans varying the rate of effort in preventing EBV the Infectious humans reduces drastically while Fig 1c is showing the Removed humans varying the rate of effort in preventing EBV the Removed humans Increases drastically.

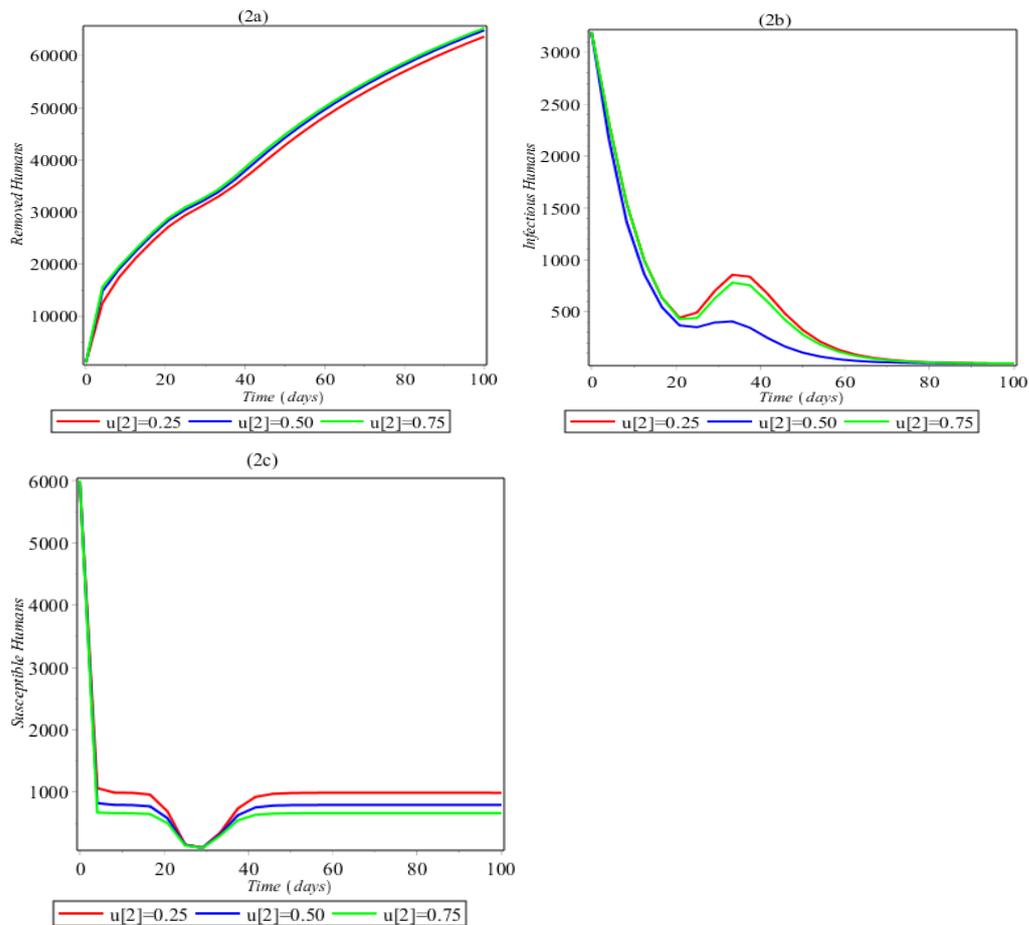


Figure 2a, Figure 2b and Figure 2c are comparison the effectiveness of the efficacy of vaccine in preventing EBV in humans. Parameters used are as in table 1.

Fig 2c is showing the Susceptible humans varying the rate of efficacy of vaccine in preventing EBV the susceptible reduces to a stable value and Fig 2b is showing the Infectious humans varying the rate of efficacy of vaccine in preventing EBV, the Infectious humans reduces drastically while Fig 2a is showing the Removed humans varying the rate of efficacy of vaccine in preventing EBV the Removed humans Increases drastically.

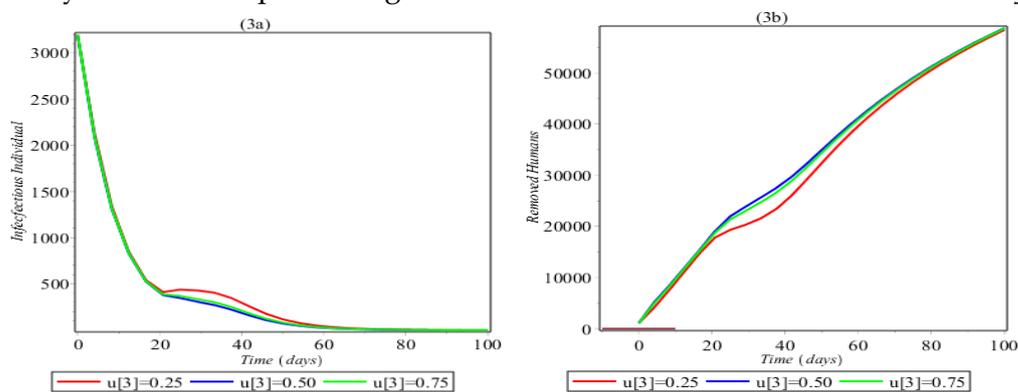


Figure 3a and Fig 3b are comparison of effectiveness of the efficacy of Immune buster in preventing EBV in humans. Parameters used are as in Table 1.

Fig 3a is showing the Infectious humans varying the rate of efficacy of Immune buster in preventing EBV, the Infectious humans reduces as the Immune buster increases and Fig 3b is showing the Removed humans varying the rate of efficacy of Immune buster in preventing EBV the Removed humans Increases drastically.

DISCUSSION

Many EVD mathematical models were developed and analyzed to explain human to human transmission and animals to human transmission, but very few considered vaccination and accessed its impact on the control of the disease. Also, very few considered bat to bat transmission. In this paper, we concentrate mainly on optimal control strategy (which implies efficacy of vaccination, efficacy of Immune buster and effort of preventing transmission) with a target that implementing vaccine does not lead to hundred percent vaccinations.

A qualitative optimal control analysis of model (3) was performed. In this gaze, the main results obtain are point out as follows. The result shows that applying optimal control helps in eradicating Ebola virus in the society especially when the optimal control is applied to vaccine and the effort of controlling the transmission. On the other hand, numerically, we have shown that: (1) the disease can be trash out if optimal control can be applied on vaccine. (2) the number of infected individuals decreases when the optimal control is applied on the effort of reducing transmission of Ebola virus.

CONCLUSION

The above-mentioned theoretical and numerical studies suggest that vaccination alone cannot drive EVD to death but it can reduce it to the minimum. It is recommended to develop a similar model taking into account age-structure which will also help in controlling the disease, though this will leads to a large number of differential equations, which can be more realistic but less mathematical tractable.

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