Computational Modelling and Optimal Control of Ebola Virus Disease with non-Linear Incidence Rate

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Computational Modelling and Optimal Control of Ebola Virus Disease with non-Linear Incidence Rate

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Abstract. The 2014 Ebola outbreak in West Africa has exposed the need to connect modellers and those with relevant data as pivotal to better understanding of how the disease spreads and quantifying the effects of possible interventions. In this paper, we model and analyse the Ebola virus disease with non-linear incidence rate. The epidemic model created is used to describe how the Ebola virus could potentially evolve in a population. We perform an uncertainty analysis of the basic reproductive number R_0 to quantify its sensitivity to other disease-related parameters. We also analyse the sensitivity of the final epidemic size to the time control interventions (education, vaccination, quarantine and safe handling) and provide the cost effective combination of the interventions.

1. Introduction

The 2014 Ebola virus outbreak is the largest and most complex ever to occur since the disease's first appearance in 1976 via simultaneous outbreaks in Nzara, Sudan and in Yambuku, Democratic Republic of Congo. The latter occurred in a village next to Ebola River, from where the disease took its name. The Ebola virus is a zoonotic filovirus which erupts occasionally and has caused at least 14 confirmed outbreaks in Africa between 1976 and 2006 [13]. The exact reservoir of Ebola viruses is still unknown, but researchers believe the most likely natural hosts are fruit bats [7]. Ebola causes haemorrhagic fever and death in humans after about ten days, and people in contact with infectives can be infected. The virus can be spread from one person to others through direct contact with blood or body fluids (urine, saliva, sweat, faeces, vomit, breast milk, and semen) of a person who is sick with or has died from Ebola and objects (like needles and syringes) that have been contaminated with body fluids [3].

The typical trajectory of the disease begins with an average incubation period of one to two weeks. Patients mostly present with fever, asthenia, diarrhoea, abdominal pain, headache, arthralgia, myalgia, sore throat, dysphagia, and conjunctivitis [19]. One week after the onset of symptoms a rash often appears followed by haemorrhagic complications, leading to death after an average of ten days in 50–90% of infections. The virus can lurk in the body for more than a week before it begins a cascading meltdown of the immune system, blood vessels and vital organs [18]. Recovery usually occurs in two weeks to two months after the onset of symptoms. Most individuals acquire infection after direct

contact with blood, bodily secretions and tissues of infected ill or dead humans and non-human primates [8, 15]. There is evidence that individuals (health-care workers, relatives) may become infected following contacts with patients' body fluids or direct contact with patients during a visit at the hospital or participation in traditional burial ceremonies [11]. Ebola is not transmissible if someone is asymptomatic or once someone has recovered from it. However, the virus has been found in semen for up to three months [7]. Vaccine and therapeutic strategies have been under development since around 2003 [17, 6].

There is as yet no proven treatment for Ebola virus disease (EVD). However, a range of potential treatments including blood products, immune therapies and drug therapies are currently being investigated. No licensed vaccines are available yet, but two potential vaccines are undergoing human safety tests (WHO). The recent outbreak has had more cases and deaths than all previous ones combined. More than 20 000 people had been infected with Ebola, as of 28 December 2014, as the recent outbreak continued to spread globally. Specific statistics were as follows: Liberia (8018 cases, 3423 deaths), Sierra Leone (9446 cases, 2758 deaths), Guinea (2707 cases, 1709 deaths), Nigeria (20 cases, 8 deaths), Mali (8 cases, 6 deaths), United States (4 cases, 1 death), Senegal (1 case, 0 deaths) and Spain (1 case, 0 deaths) [3].

The authors in [2] showed, using SIR and SEIR models, that it was possible to simulate Ebola outbreaks. They considered the 1976 outbreak in Yambuku, DRC (then Zaire) and the 1995 outbreak in Kikwit, DRC. Chowell et al 2004 modelled the course of the Congo 1995 and Uganda 2000 Ebola outbreaks via an SEIR (susceptible-exposed-infectious-removed) epidemic model that included a smooth transition in the transmission rate after control interventions are put in place. The control measures implemented during these two outbreaks (including education and contact tracing followed by quarantine) reduced the final epidemic size by a factor of 2 relative to the final size with a 2-week delay in their implementation.

Previously published Ebola Haemorrhagic Fever (EHF) data from two epidemics was analysed using a stochastic compartmental model which incorporated the explicit settings of the transmission in the community, in the hospital and during burial ceremonies [13]. The goal was to better understand and to provide insight into where control interventions could be targeted in the future. The infectious phase was subdivided into three stages to account for transmission in the community, in the hospital (including isolation wards), and after death during traditional burial. Various epidemic scenarios were simulated to explore the impact of control interventions on a potential epidemic. Increasing hospitalization rate, for both epidemics, reduced the predicted epidemic size.

Yarus [21] analysed an epidemic model consisting of susceptible-infected-recovered-dead created from first order differential equations to describe how the Ebola virus could potentially ravage a population. Rivers et al 2014 used existing data from Liberia and Sierra Leone to parameterize a mathematical model of Ebola and used this model to forecast the progression of the epidemic, as well as the efficacy of several interventions, including increased contact tracing, improved infection control practices, the use of a hypothetical pharmaceutical intervention to improve survival in hospitalized patients. The Ebola virus epidemic was described using an SEIR model to estimate the reproduction number in the West Africa outbreak [1]. The maximum likelihood estimates of the basic reproduction number are 1.51 for Guinea, 2.53 for Sierra Leone and 1.59 for Liberia.

A discrete, stochastic SEIR model was combined with a two-scale community network model to demonstrate that the different regional trends may be explained by different community mixing rates [12]. The spatio-temporal epidemiological modeler (STEM) developers implemented a preliminary epidemiological model, for the three African countries mostly affected by the Ebola epidemic, based on 8 compartments and 8 differential equations. The model includes the usual susceptible, exposed,

infectious states along with 5 additional compartments: death (but not buried), buried, clinical isolation, and burial. The usual recovered or removed compartment is represented by asymptomatic but still infectious individuals [9].

The paper is organized as follows. In Section 2, we present a compartmental deterministic model consisting of ordinary differential equations describing the transmission dynamics of the Ebola virus disease given the underlying assumptions and also provide the basic properties of the model as well as investigate the existence and stability of equilibria. Section 3 is devoted to optimal control of the disease making use of Pontryagin's maximum principle. In Section 4, we present and discuss the numerical simulations results. Cost effectiveness analysis is the subject of Section 5 and our conclusions are in Section 6.

2. Model Formulation

The population, $N(t)$, is sub-divided into the following non-overlapping but exhaustive compartments: the susceptible, $S(t)$; the exposed, $E(t)$; the infected, $I(t)$; individuals on treatment, $T(t)$, the recovered, $R(t)$ and those who die due to the disease $D(t)$. We assume that individuals are recruited into the population being susceptible, through birth and immigration, at a constant rate Q. The disease is transmitted to the susceptible by the infected, those on treatment and the deceased corpses at rates β_1, β_2 and β_3 respectively, $\beta_1 > \beta_3 > \beta_2$ due to the effect of treatment and that the virus in a corpse is not as deadly. Individuals in the exposed class progress to the infected class at a rate σ, the infected migrate to the treatment class at a rate γ and the treated recover at rate b. Individuals in all compartments die due to natural causes at a rate μ while the infected and the treated may also die due to the Ebola virus at rates m_1 and m_2 respectively, $m_1 > m_2$. The recovered individuals progress to the susceptible class at a rate α while the deceased are removed from the system through safe burial at a rate ρ. We denote by u_1 the control measure due to quarantine of the exposed and infected individuals as well as safe handling of the dead while u_2 represents the control parameter due to efficacy of the treatment drug used for Ebola virus victims.

The resulting ***state system*** of first order differential equations is the following:

$$\frac{dS}{dt} = Q - (1-u_1)\lambda S + \alpha R - \mu S$$

$$\frac{dE}{dt} = (1-u_1)\lambda S - (\sigma + \mu)E$$

$$\frac{dI}{dt} = \sigma E - (\gamma + m_1 + \mu)I$$

$$\frac{dT}{dt} = \gamma I - (1+u_2)bT - m_2 T - \mu T$$

$$\frac{dR}{dt} = (1+u_2)bT - (\alpha + \mu)R$$

$$\frac{dD}{dt} = m_1 I + m_2 T - \rho D$$

where $\lambda = \frac{\beta_1 I + \beta_2 T + \beta_3 D}{a + I + T + D}$ is the force of infection.

2.1. Basic Model Properties

The model represents the dynamics of a human population. Hence, all its parameters and initial conditions are assumed to be non-negative. Similarly, for the model to make sense, all its solutions must be non-negative. Thus, we claim the following:

Theorem: The solutions $S(t)$, $E(t)$, $I(t)$, $T(t)$, $R(t)$ and $D(t)$ of (1) with non-negative initial conditions exist for all $t > 0$, and are unique. Furthermore, the solutions are non-negative and are bounded for all $t > 0$.

Proof: From the first equation of the ***state system***,

$$\frac{dS}{dt} \geq -(\lambda + \mu)S$$

Assuming $S(0) \geq 0$ and that all other parameters are non-negative, we have

$S(t) \geq S(0)exp\left(-\int_0^t (\lambda + \mu)dv\right) \geq 0$ for all $t > 0$.

Similarly, $E(t)$, $I(t)$, $T(t)$, $R(t)$ and $D(t)$ are non-negative for $t > 0$.

Now, the total population, $N(t)$ satisfies

$$Q - (\mu + \rho)N \leq \frac{dN}{dt} = Q - \mu N - \rho D \leq Q - \mu N$$

This implies that

$$\frac{Q}{\mu + \rho} \leq N(t) \leq \frac{Q}{\mu}$$

□

The set

$$\Gamma = \left\{(S, E, I, T, R, D) \in \mathbb{R}_+^6 : S + E + I + T + R + D = N \leq \frac{Q}{\mu}\right\}$$

is positively invariant and attracting with respect to the model. The model is thus well-posed epidemiologically and mathematically, hence we can study the qualitative properties of the system.

2.2. Analysis of the Model

The model has an Ebola-free equilibrium (EFE) given by

$$\xi^0 = (S^0, E^0, I^0, T^0, R^0, D^0) = \left(\frac{Q}{\mu}, 0, 0, 0, 0, 0\right)$$

The transmissibility of an infection can be quantified by its basic reproductive number R_0, defined as the mean number of secondary infections seeded by a typical infective into a completely susceptible

(naive) host population [5]. Thus, to determine the impact of introducing an infectious individual in a population of completely susceptible individuals, we proceed by computing the basic reproductive number of the system. This is the spectral radius of the next generation matrix $K = F\,V^{-1}$, where F and V respectively represent the transmission (at the EFE) and transition matrices and are given by

$$F = \begin{bmatrix} 0 & f_1 & f_2 & f_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}; V = \begin{bmatrix} \sigma+\mu & 0 & 0 & 0 \\ -\sigma & \gamma+m_1+\mu & 0 & 0 \\ 0 & -\gamma & v & 0 \\ 0 & -m_1 & -m_2 & \rho \end{bmatrix}$$

where $f_i = \frac{(1-u_1)\beta_i Q}{a\mu}$, $i = 1,2,3$ and $v = (1+u_2)b + \mu + m_2$. F describes the production of new infections whereas V describes the changes in states. It follows that, at the EFE, the basic reproduction number is

$$R_0 = \frac{Q(1-u_1)[\rho\beta_1\big((1+u_2)b+\mu+m_2\big) + \gamma(\rho\beta_2 - m_2\beta_3)]}{a\mu\rho(\sigma+\mu)(\gamma+m_1+\mu)\big((1+u_2)b+\mu+m_2\big)}$$

Lemma: The Ebola free equilibrium, ξ^0, of the model represented by the ***state system*** is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.

The Jacobian matrix J of the **state system** at ξ^0 is

$$J(\xi^0) = \begin{bmatrix} -\mu & 0 & -f_1 & -f_2 & \alpha & -f_3 \\ 0 & -y & f_1 & f_2 & 0 & f_3 \\ 0 & \sigma & -u & 0 & 0 & 0 \\ 0 & 0 & \gamma & -v & 0 & 0 \\ 0 & 0 & 0 & w & -x & 0 \\ 0 & 0 & m_1 & m_2 & 0 & -\rho \end{bmatrix}$$

where $y = \sigma + \mu$, $u = \delta + m + \mu$, $w = (1-u_2)b$ and $x = \alpha + \mu$. The eigenvalues are

$$\Lambda_1 = 0 = \Lambda_2, \quad \Lambda_3 = -\mu < 0, \qquad \Lambda_4 = -\rho < 0,$$
$$\Lambda_5 = -\frac{1}{2}\left(\sigma + 2\mu + \gamma + m_1 + \sqrt{\frac{4\sigma(1-u_1)\beta_1 Q + a\mu(\sigma+2\mu+\gamma+m_1)^2}{a\mu}}\right) < 0$$
$$\Lambda_6 = -\frac{1}{2}\left(\sigma + 2\mu + \gamma + m_1 - \sqrt{\frac{4\sigma(1-u_1)\beta_1 Q + a\mu(\sigma+2\mu+\gamma+m_1)^2}{a\mu}}\right)$$

$\Lambda_6 < 0$ only if $\sigma + 2\mu + \gamma + m_1 - \sqrt{\frac{4\sigma(1-u_1)\beta_1 Q + a\mu(\sigma+2\mu+\gamma+m_1)^2}{a\mu}} > 0$

implying

$$0 > 4\sigma(1-u_1)\beta_1 Q$$

only if $R_0 < 1$. Thus, ξ^0 is LAS.

The state system has an endemic equilibrium $\xi^* = (S^*, E^*, I^*, T^*, R^*)$ described by

$$S^* = \frac{(\delta+\mu)E^*}{(1-u_1)\lambda^*}; I^* = \frac{\delta E^*}{\delta+m+\mu}; T^* = \frac{\gamma\delta E^*}{(\delta+m+\mu)[(1-u_2)b+\mu]}; R^* = \frac{(1-u_2)b\gamma\delta E^*}{(\alpha+\mu)(\delta+m+\mu)[(1-u_2)b+\mu]}$$

where λ^* represents the endemic force of infection.

2.3. Sensitivity Analysis

The following partial derivatives of the reproduction number with respect to its parameters

$$\frac{\partial R_0}{\partial \beta_1} = \frac{(1-u_1)Q}{a\mu(\delta+\mu)}; \frac{\partial R_0}{\partial Q} = \frac{(1-u_1)\beta_1}{a\mu(\delta+\mu)}; \frac{\partial R_0}{\partial \delta} = -\frac{\beta_1(1-u_1)Q}{a\mu(\delta+\mu)^2}; \frac{\partial R_0}{\partial \mu} = -\frac{\beta_1(1-u_1)Q(\delta+2\mu)}{a\mu^2(\delta+\mu)^2}$$

show that increasing (decreasing) β_1 or Q may increase (decrease) R_0 whilst increasing (decreasing) δ or μ may decrease (increase) R_0. To determine the relative importance of each parameter we make use of the normalized forward sensitivity index defined as follows:

Definition: The normalized forward sensitivity index of a variable U, that depends differentiably on a parameter p, is defined as:

$$\zeta_p^U = \frac{\partial U}{\partial p} \times \frac{p}{U}$$

We evaluate the sensitivity indices using the baseline parameter values given in Table 2.

Table 1: Sensitivity indices

Parameter	Index
β_1	1
Q	1
δ	-5.188755252
μ	-1.44800465

It turns out that the reproduction number, R_0, is most sensitive to the rate of progression of the exposed to the infected class, δ. In particular, a 1 % decrease (increase) in δ results in a 5.2 % increase (decrease) in R_0.

3. Optimal Control

To investigate the optimal level of efforts that would be needed to control the disease, we wish to minimize the number of exposed, infected and dead individuals, that is the classes E, I and D, and the cost of applying the controls u_1 and u_2 over a finite time interval $[0, T_f]$. We achieve this by defining an objective functional, J, by choosing a quadratic cost on the controls

$$J = \int_0^{T_f} (kE + nI + pD + b_1u_1^2 + b_2u_2^2)dt$$

where k, n, p, b_1 and b_2 are positive weights. The weights of state variables are assigned depending on their relative importance while those of controls are assigned relative to their cost implications.

With the given objective functional, $J(u_1, u_2)$, our goal is to minimize the number of the exposed E and the infectious I while minimizing the cost of the controls $u_1(\mathrm{t})$ and $u_2(\mathrm{t})$. We thus seek an optimal control pair (u_1^*, u_2^*) such that

$$J(u_1^*, u_2^*) = \min\{J(u_1, u_2): (u_1, u_2) \in \mathcal{U}\}$$

where

$$\mathcal{U} = \{(u_1, u_2): u_i \text{ measurable with } 0 \leq u_i \leq w_i < 1, \mathrm{i} = 1, 2\ ;\ \mathrm{t} \in [0, T_f]\}$$

is the control set where w_i represents the maximum coverage for control u_i.

The necessary conditions that an optimal pair $(\boldsymbol{u}^*, \boldsymbol{x}^*)$, where $\boldsymbol{u}^* = (u_1^*, u_2^*)$ and $\boldsymbol{x}^* = (S^*, E^*, I^*, T^*, R^*, D^*)$, must satisfy come from Pontryagin's Maximum Principle [16]. We use this principle to convert the problem of minimization of the objective functional coupled with the state variables into a problem of minimizing point-wise a Hamiltonian, H, with respect to the controls u_1 and u_2.

$$H = kE + nI + pD + b_1 u_1^2 + b_2 u_2^2 + \lambda_S \cdot \frac{dS}{dt} + \lambda_E \cdot \frac{dE}{dt} + \lambda_I \cdot \frac{dI}{dt} + \lambda_T \cdot \frac{dT}{dt} + \lambda_R \cdot \frac{dR}{dt} + \lambda_D \cdot \frac{dD}{dt}$$

where λ_S, λ_E, λ_I, λ_T, λ_R and λ_D are adjoint or co-state variables. By applying Pontryagin's Maximum Principle and the existence result for the optimal control [10], we obtain the following:

Proposition: *For the optimal control pair* (u_1^*, u_2^*) *that minimizes* $J(u_1, u_2)$ *over* $\mathcal{U}$*, there exist adjoint variables* λ_S, λ_E, λ_I, λ_T, λ_R *and* λ_D *satisfying the following:*

(i) Adjoint System

$$\frac{d\lambda_S}{dt} = (1 - u_1)\left(\frac{\beta_1 I + \beta_2 T + \beta_3 D}{a + I + T + D}\right)(\lambda_S - \lambda_E) + \mu\lambda_S$$
$$\frac{d\lambda_E}{dt} = -k + \sigma(\lambda_E - \lambda_I) + \mu\lambda_E$$
$$\frac{d\lambda_I}{dt} = -n + B_1(\lambda_S - \lambda_E) + \gamma(\lambda_I - \lambda_T) + m_1(\lambda_I - \lambda_D) + \mu\lambda_I$$
$$\frac{d\lambda_T}{dt} = B_2(\lambda_S - \lambda_E) + (1 + u_2)b(\lambda_T - \lambda_R) + m_2(\lambda_T - \lambda_D) + \mu\lambda_T$$
$$\frac{d\lambda_R}{dt} = \alpha(\lambda_R - \lambda_S) + \mu\lambda_R$$
$$\frac{d\lambda_D}{dt} = -p + B_3(\lambda_S - \lambda_E) + \rho\lambda_D$$

where $B_i = \frac{(1-u_1)S\{(a+I+T+D)\beta_i - (\beta_1 I + \beta_2 T + \beta_3 D)\}}{(a+I+T+D)^2}$, $i = 1, 2, 3$.

(ii) Transversality Conditions

$$\lambda_S(T_f) = \lambda_E(T_f) = \lambda_I(T_f) = \lambda_T(T_f) = \lambda_R(T_f) = \lambda_D(T_f) = 0$$

(iii) Stationary Values

$$u_1^* = min\left\{w_1, max\left\{0, \frac{(\beta_1 I + \beta_2 T + \beta_3 D)S(\lambda_E - \lambda_S)}{2(a + I + T + D)b_1}\right\}\right\}$$

$$u_2^* = min\left\{w_2, max\left\{0, \frac{bT(\lambda_R - \lambda_T)}{2b_2}\right\}\right\}$$

4. Numerical Simulations

We study numerically an optimal transmission parameter control for the Ebola virus model. The optimal control set is obtained by solving the associated optimality system which consists of ***state*** and ***adjoint*** systems. An iterative fourth order Runge-Kutta scheme is used for solving the optimality system. We start to solve the state equations with a guess for the controls over the simulated time. The state equations are solved using a forward method with given initial conditions for the state variables. The corresponding adjoint system is solved using a backward scheme with the ***transversality conditions***. The controls are updated by using a convex combination of the previous controls and the ***stationary values***. This process is repeated and iterations stopped if the values of the unknowns at the previous iterations are very close to the ones at the present iterations [14].

Table 2: Model parameters

Parameter	Description	Value
Q	Recruitment rate	10
β_2	Contact rate between the susceptible and the treated	0.112195104
β_1	Contact rate between the susceptible and the infected	0.224390208
β_3	Contact rate between the susceptible and the dead	0.168292656
γ	Migration rate for infected to treatment	0.12195120
α	Progression rate for recovered to susceptible	0.023
b	Recovery rate for the treated	0.03560976
m_1	Ebola-induced mortality rate for the infected	0.708
m_2	Ebola-induced mortality rate for the treated	0.508
μ	Natural death rate	0.0863414496
σ	Progression rate for exposed to infected	0.1063830
ρ	Removal rate for the dead	0.5

For the simulations, we choose the model parameter values as in Table 1. All the values except the recruitment rate Q, which is assumed, are sourced from [9]. We assume that the weight factor b_1, associated with control u_1, is lower than b_2, which is associated with the control u_2. This assumption is based on the fact that it probably costs more to treat an infected person. Moreover, the cost associated with treatment will also include the cost of medical examinations, any prescribed drugs and hospitalization, thus making it costlier. We make use of the following objective functional parameter values: $n = 75$, $k = 150$, $p = 50$, $b_1 = 50$, $b_2 = 120$. For illustrative purposes we make use of $w_1 = 0.9 = w_2$ and the initial state conditions $S(0) = 100$, $E(0) = 30$, $I(0) = 2$, $T(0) = 1$, $R(0) = 0$ and $D(0) = 0$.

We discuss results for the case when deploying both the quarantine and treatment interventions. Numerical results show that there is a marked reduction from the onset in the number of the exposed when employing interventions, Fig. 1. Interventions result in a gradual decline in the infected class, Fig. 2. The declines in the treated and recovered classes, Fig. 3 and Fig. 4, are not marked. This may be due to the fact that fewer people are being exposed to the infection.

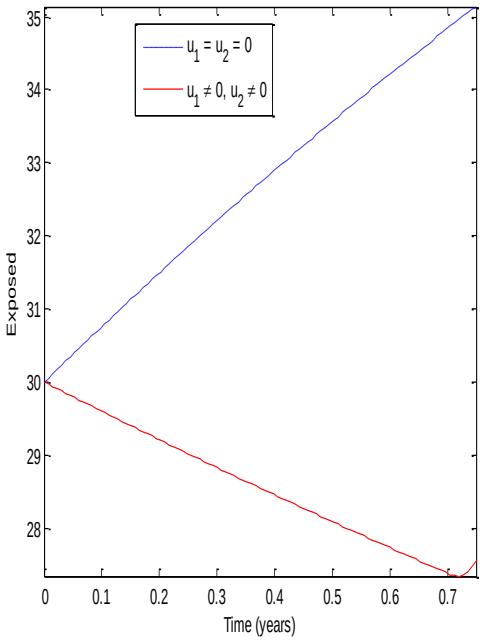

Fig. 1: Exposed

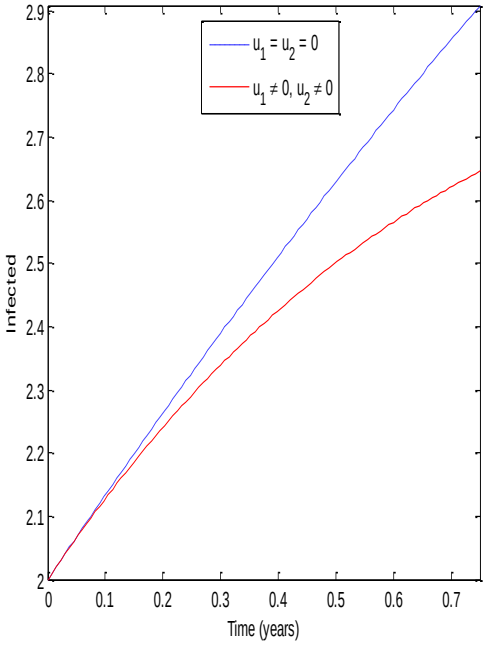

Fig. 2: Infected

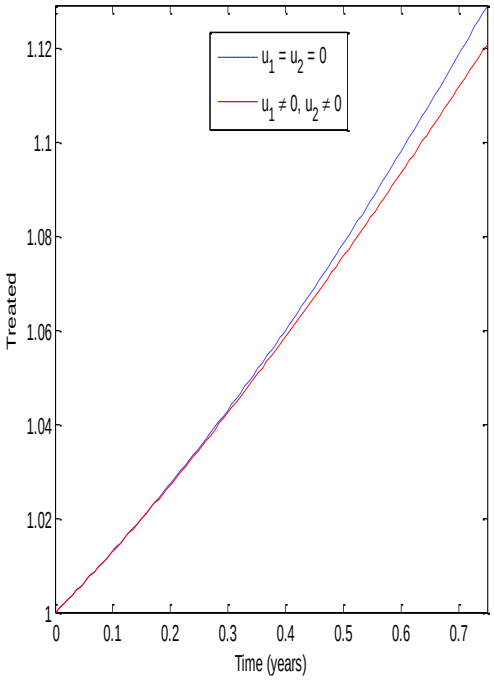

Fig. 3: Treated

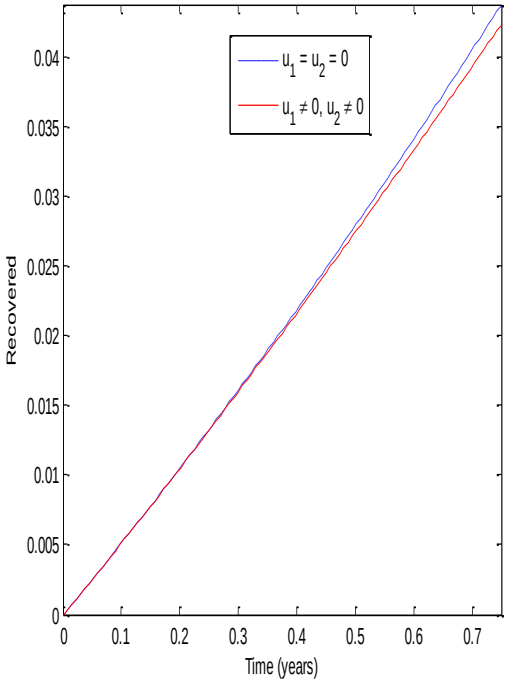

Fig. 4: Recovered

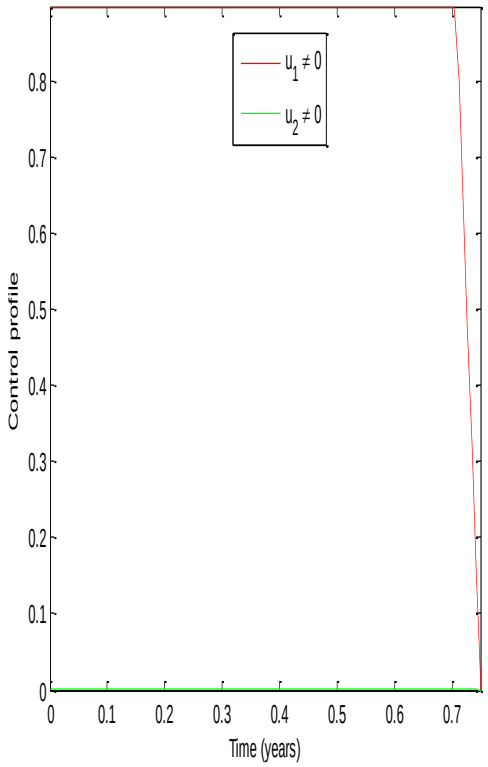

Fig. 5: Control Profiles

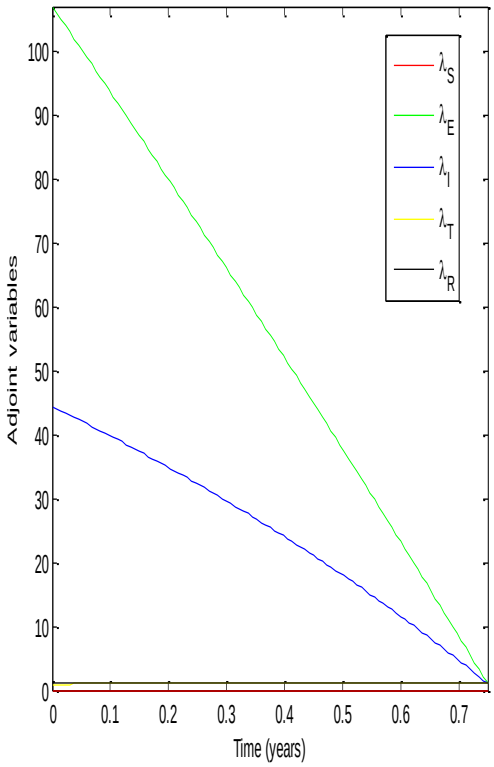

Fig. 6: Adjoint Variables

Control profiles, Fig. 5, show that maximum effort needs to be put in deploying quarantine measures (for about 8 months if the intervention period is 9 months) to ensure that the exposed and infected do not interact with the rest of the population. The treatment control can then be minimal. Shadow prices, Fig. 6, show that the exposed and the infected are the costliest.

It is of utmost importance to determine the cost effective interventions so that funds may be appropriately spent given the tight budgets national economies operate on.

5. Cost Effectiveness

A critical goal of public health programmes is the realization of positive impact for a given population. This impact is usually a result of a combination of various inputs and it is often not due to a single intervention. Cost effective analysis is one economic evaluation tool used to measure the costs and consequences of alternative programmes. The measures are then compared to determine how the greatest health benefits can be generated. We use indicators related to a change in health status such as the estimated number of fatalities or infections averted to measure impact. The strategy which realises the most positive impact is identified by use of incremental cost effectiveness ratios (ICERs) defined by

$$ICER = \frac{Difference\ in\ costs\ between\ strategies}{Difference\ in\ health\ effects\ between\ strategies}$$

We consider as health effects the cases averted in the E and I classes for the following three strategies: A – quarantine measures; B – treatment measures; C – both controls employed. The

strategies are ranked from the least effective by considering health effects and then compared pairwise using ICERs.

Table 3. Cases averted and associated costs

Strategy	Cases Averted	Costs
B	137	11 831
A	403	753.80
C	404	753.84

The ICER between B and A is given by $\frac{753.80-11\,831}{403-137} = -41.64$. It costs 41.64 less for each additional case averted from B to A, so B is excluded.

Next, we calculate the ICER between A and C, which is 0.04. Hence, it costs 0.04 more for each additional case averted as we switch from A to C. So, we exclude C and come to that strategy A is the most cost effective.

6. Conclusion

Shadow prices show that the cost and impact of the exposed is very high, followed by the infected. This may result in negative effects on the population. The results suggest that quarantining of the exposed and the infected is the most effective strategy. However, the marginal additional cost per extra case averted when switching from strategy A to C justifies the inclusion of treatment measures. Therefore, budgetary provision needs to be made to include the treatment of the Ebola virus victims. Control programs that follow these strategies can effectively reduce the incidence and endemicity of the Ebola virus disease.

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