Mathematical Modelling of Ebola Epidemic and the Impact of Prevention and Control

Jean de Dieu NIYIGENA (jniyigena@aims.ac.tz)
AIMS141501014

African Institute for Mathematical Sciences (AIMS)

Supervised by: Dr. Angelina M Lutambi
Ifakara Health Institute, Tanzania
Co-supervised by: Dr. Martial L Ndeffo
Yale School of Public Health, USA

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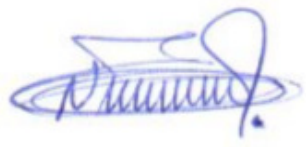
Abstract

Ebola has emerged as a threat to public health in Africa after a major outbreak occurred in West Africa in 2014. Efforts to prevent further transmission are underway in Africa and the world at large. Mathematical models are key tools in deriving new knowledge and guiding decisions before and after the occurrence of diseases. In this project, we develop and simulate mathematical models of Ebola to investigate the dynamics and effects of various prevention and control strategies, and investigate the minimum effort needed for a targeted type host to prevent the Ebola disease for informed decisions. We revisited an ordinary differential equation model of Ebola epidemic and extend it to incorporate community, hospitals, and funeral components, necessary categorization for the Ebola transmission dynamics and control. The models were analysed and numerically simulated to investigate effects of prevention and control. Control of this disease should be immediate, as many individuals would die within a few days if nothing is done. Hospital closure, efficient treatment, and vaccination of healthcare workers have a greater impact and potentially eradicate Ebola from the community. Therefore, results suggest that, the effect of control measures are dependent on the type of intervention and group of people that it targets. Although the results presented in this report are based on the assumptions of the models developed, results are useful to consider when designing interventions. This work has highlighted the importance of mathematical models in the generating essential knowledge for evidence based decisions and for designing better ways of controlling Ebola.
Keywords: Ebola, Reproduction number, Simulation, Intervention, Control.

Declaration

I, the undersigned, hereby declare that the work contained in this research project is my original work, and that any work done by others or by myself previously has been acknowledged and referenced accordingly.



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1. Introduction

1.1 Background

Ebola is a viral disease which is highly infectious and can be transmitted from one person to another through several ways. It is the most spread epidemic[1] of the Ebola Virus Disease (EVD). The EVD started in the Democratic Republic of Congo (Zaire) and Sudan in the year 1976 (CDC, 2015). In the Democratic Republic of Congo (DRC), the Ebola outbreak occurred between August and November 1976, where 318 Ebola cases were reported and among them, 280 infected people died, making the mortality rate approximately 88% (Camacho et al., 2014). In Sudan, Ebola occurred in the Southern part of the country between June and November 1976. During this period, 284 cases were reported with a mortality rate of about 53% (WHO, 1978). Although the origin of the Ebola virus is unknown, little evidence has started to emerge indicating that forest animals such as fruit bats, chimpanzees, gorillas, monkeys, forest antelope and porcupines are hosts of the Ebola virus (Legrand et al., 2007).

Recent statistics show that the burden of Ebola is high in West African countries (CDC, 2015). On July 2015, the World Health Organization (WHO) reported a total of 16,874 Ebola cases, with Sierra Leone and Guinea leading in transmission. During the 2014 outbreak, 3,745 and 13,129 occurred in Guinea and Sierra Leone respectively with 2,490 and 3,933 deaths occurring in the same countries. Liberia registered a total of 10,666 cases of which, 4,806 died during the same period. The epidemic spread to other African countries such as Nigeria (20 cases), Senegal (1 case), and Mali (8 cases) during the same period. Ebola transmission has spread to other non-African countries: One Ebola case occurred in Spain and in the United Kingdom, and four cases occurred in United States (CDC, 2015).

The Ebola virus disease is transmitted via direct contact (through broken skin or mucous membranes) with the blood, organs, secretions or other bodily fluids (stool, urine, saliva, semen) of infected people. Surfaces and materials such as beddings, and clothing contaminated with body fluids of infected people, local burial customs where dead bodies are washed before burial can also contribute to transmission. Men who have recovered from Ebola are said to transmit the virus (up to 7 weeks after recovery) to their partner through semen. Breastfeeding is also a risk factor[2] as babies may acquire the virus through breast milk of infected mothers (CDC, 2015).

Symptoms of Ebola are divided into two stages: The first stage often includes fever, headache, sore throat, fatigue, and muscle pain. This stage can often be mistaken for other diseases such as malaria or typhoid. The second stage includes more severe symptoms such as vomiting, diarrhoea, rash and in some cases, internal and external bleeding. Recovery is more likely to occur from the first stage, with much higher death rates in the second stage (indeed, in some outbreaks all second-stage patients die). Ebola is unlikely to be transmitted during the incubation period and the transmissibility is likely to be higher as the disease progresses. An Ebola patient can take from 2 weeks to 2 months to recover from the time she/he gets the symptoms (CDC, 2015).

Although there are currently no vaccines or drugs for Ebola, several prevention and control measures are possible and they have been implemented in several countries. Prevention strategies targeted at reducing contact between people at risk and those infected have been invested by governments and the

[1] A disease is epidemic if it spreads rapidly to a large number of people in a given population of a particular region within a short period of time (Newman, 2002).

[2] Ebola also appears in the breast milk of infected women after recovery, and the time taken for it to disappear is not known (CDC, 2015).

world at large. These include wearing appropriate personal protective equipments (PPE), practice proper infection control, isolating patients with Ebola from other patients, and avoiding direct or unprotected contact with the bodies of people who have died from Ebola. In addition, health officials are notified if a direct contact with the blood or other body fluids of the Ebola infected individual is made (CDC, 2015).

1.2 Literature Review

Over the years, studies have considered mathematical models on Ebola epidemics. In (Legrand et al., 2007), the authors developed a stochastic compartment model of the Ebola outbreaks in the Democratic Republic of Congo (DRC) in 1995, and Uganda in 2000. The model was further expanded (Chowell et al., 2004) to include infections that occur in the community, hospital, and after death from the bodies not yet buried, awaiting a traditional funerals.

In (Bashar et al., 2014), authors analysed the 2014 Ebola epidemic in West Africa countries focusing on Guinea, Sierra Leone, and Liberia using an SEIR model. Using networks models, authors found that the Ebola outbreak was less likely to become an endemic disease. A modelling work by Camacho et al. (2014) also indicates that the transmission of the disease decreased before the closure of the hospital (Yambuku Mission hospital). Analysis based on empirical data from DRC showed that changes of the host behaviour resulted into a significant reduction of the Ebola outbreak.

Other studies (Khan et al., 2015) have used deterministic ordinary differential equations to model transmission of the Ebola epidemic. One of the interesting results of their work was to subdivide the susceptible compartment into high and low risk classes. The high risk classes included the health care workers and providers such as front-line workers, people related to the infected individuals visiting hospitals, and people involved in burial processes. The other categories of people were considered in low risk susceptible classes. In their analysis, the next generation matrix approach was used to calculate the basic reproduction number. The basic reproduction number was found to be made up of two terms reflecting on the infectiousness associated on community and hospitals transmission.

Mathematical models play an important role in assessing the effect of prevention and control strategies. Mathematical models of Ebola outbreaks can help to derive conditions by projecting the suitable impact of proposed interventions before implementation. This in turn, provides policy makers, the media, healthcare personnel, and the public with appropriate, quantifiable evidence to support decision making (Rivers et al., 2014).

Simulation studies of the Ebola epidemic are used to provide better understanding of the method and suggest prevention, control strategies to guide policy development and improve control efforts. Models are also used to derive conditions that can guide decisions. Stability analysis of the equilibrium points for example, is a basic concept in mathematical modelling (Koya and Mamo, 2015).

The concept of the basic reproduction number [3] is an important parameter in epidemiology that gives an indication of how much efforts are needed to control epidemics. If a prevention, intervention, or control strategy such as vaccination, social distancing, treatment is aimed at all host individuals regardless of their epidemiological status, the value of the basic reproduction number is a measure of the strength of the control required to prevent outbreaks from occurring in a given population (Shuai et al., 2013).

[3] The basic reproduction number is the average number of secondary infections generated by one infected individual of Ebola in a population where every individual is susceptible. It determines whether an infectious disease dies out or spreads in the population.

Mathematical models have also been used in determining better ways of optimizing intervention for control of Ebola. In (Salaam-Blyther, 2014), the author discusses three main effective interventions including quarantine, vaccination, and delivery. Although there exists no cure or vaccines, this model suggests that, to prevent the spread of Ebola, quarantine was found to be an efficient control strategy. In the events of outbreaks, the study suggests proper channels and rapid medicine delivery options through the use of efficient algorithm that solves the delivery problem.

1.3 Objectives of the Study

The general objective of this project is to develop a mathematical model of Ebola to gain a broader understanding of the natural dynamics and its interaction with different prevention and control strategies for informed decisions. The specific objectives are as follows:

- Develop a mathematical model that captures the natural dynamics of Ebola.
- Investigate the potential impact of several interventions such as early or late change of behaviour, the closure of the hospitals at different dates, vaccine availability to healthcare workers and the efficacy of the treatment to the Ebola outbreak.
- Investigate the contribution of locations such as community, hospitals, and funeral places to the spread of Ebola using the type reproduction numbers.
- Investigate the impact of treatment availability for Ebola patients in the hospitals.

1.4 Outline

In the following chapters, we develop and analyse an Ebola basic model using differential equations. In addition, we derive the basic reproduction number, compute the disease-free equilibrium point and perform stability analysis. Furthermore, extend the basic model in order to include location dependent components where transmission is thought to occur. Numerical simulations are performed to study the effect of various preventions, interventions, and control strategies for the basic model, and the general dynamics for the extended model. Finally, discussions, conclusion and some future directions are covered in the last Chapter.

2. Model

2.1 Model Formulation

We formulate a compartmental differential equation model of the Ebola epidemic. The total human population N at any time t is sub-divided into seven compartments of use: Susceptible S, exposed E, infected in the community I, infected at hospital H, infected during funerals F, recovered R, and buried compartment B. The size N of the population is constant. It is thus given by;

$$N = S(t) + E(t) + I(t) + H(t) + F(t) + R(t) + B(t). \tag{2.1.1}$$

Susceptible are individuals with the potential to get initiated into the Ebola epidemic. Once susceptible individuals contract the Ebola virus, they enter the compartment of exposed individuals E at a rate ε. Individuals in the exposed compartment then undergo an incubation, lasting a period of $1/\alpha$ days before progressing to the compartment of the infected in the community I. We assume that individuals in the exposed compartment are asymptomatic and non infectious. Some infectious individuals (θ_1) may be hospitalized to join the compartment H at the rate $\gamma_H\theta_1$. Untreated patients in the infectious group I may experience one of the two outcomes: Patients may die (δ_1), but have a chance of infecting others during the funeral ceremonies. Such infectious patients are grouped in the funeral compartment F at the rate $\delta_1(1-\theta_1)\gamma_D$ or may recover at the rate $\gamma_I\,(1-\theta_1)\,(1-\delta_1)$ and join the recovery compartment R. Upon burial, individuals join the compartment B of buried Ebola victims at the rate γ_F. With successful treatment, the Ebola victims recover to join the compartment R of recovered individuals. A given proportion (δ_2) of hospitalized patients may die to join the funeral compartment F at the rate $\gamma_{DH}\delta_2$ but they may infect others through contact. Upon successful treatment, hospitalized individuals $(1-\delta_2)$ join the recovery compartment R at the rate $\gamma_{IH}\,(1-\delta_2)$.

We assume that the susceptibles become infected in three different ways: Person to person transmission occurring at community level, at the rate $\beta_I(t)$; transmission occurring in hospitals at the rate $\beta_H(t)$ and transmission from the dead (not yet buried individual) at the rate $\beta_F(t)$.

The Ebola transmission rate ε is a composition of transmission rates $\beta_I(t), \beta_H(t)$ and $\beta_F(t)$, defined as follows.

$$\begin{cases} \beta_I(t) = \beta_I\left(1-\sigma_I\left(t,q_I,\tau_I\right)\right), \\ \beta_H(t) = \beta_H\left(1-\sigma_H\left(t,q_H,\tau_H\right)\right), \\ \beta_F(t) = \beta_F\left(1-\sigma_F\left(t,q_F,\tau_F\right)\right), \end{cases} \tag{2.1.2}$$

where

$$\sigma_i\left(t,q_i,\tau_i\right) = \frac{1}{1+e^{-q_i(t-\tau_i)}}, \quad i = I, H \text{ or } F.$$

τ_i gives the percentage of intervention effect for i infectious compartment. $q_i > 0$ determines the rate of decay from two successive transmission rates in i infectious compartment (Ndanguza et al., 2013).

Other assumptions made in this work include the following. We assume homogeneous mixing among population under study; that is, the probability of one individual from any compartment to interact with another is the same. We ignore the number of births as well as the number of individuals who die due to the natural mortality; because Ebola does not take a long time (10 days minimum) to die or recover

from it (Legrand et al., 2007). Also, the movement (migration and immigration) of individuals from one place to another is ignored because it has been shown to have no significant contribution to the results (Koya and Mamo, 2015). We further assume that the transmission rates are constants in the part of analysis of this project.

A schematic diagram showing the transmission of Ebola is as shown in Figure 2.1.

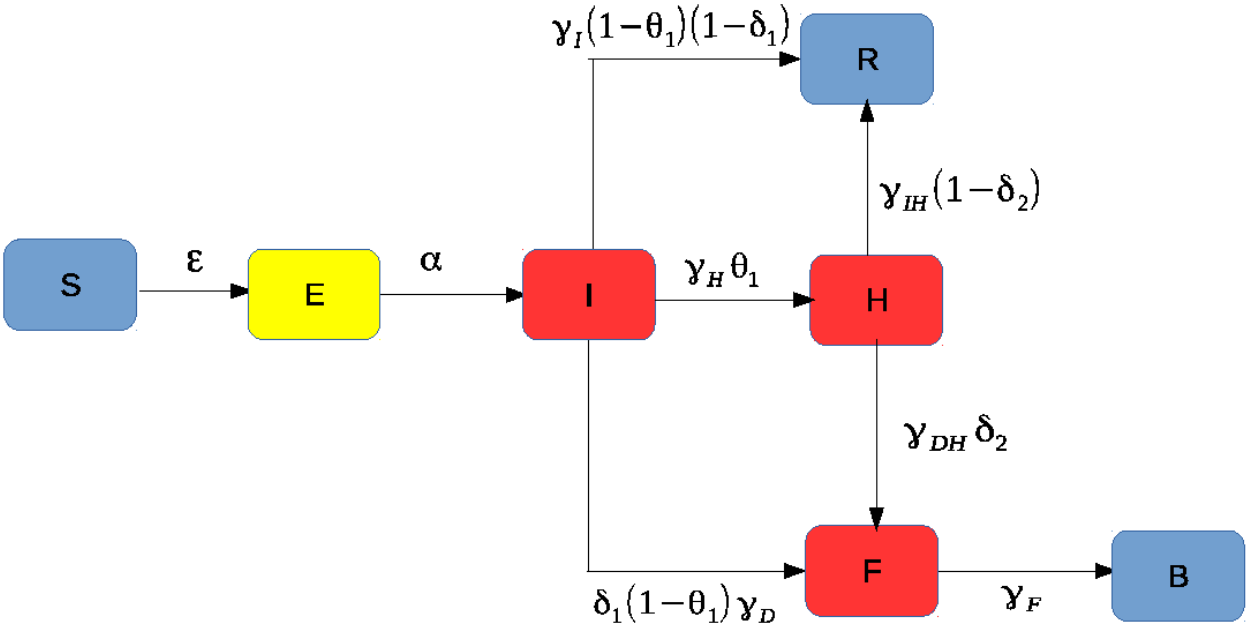

Figure 2.1: A schematic diagram of Ebola transmission. Arrows indicate the possible transitions and the parameters that govern them.

The dynamic of the Ebola disease is represented by the following system of ordinary differential equations:

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = -\left(\beta_I SI + \beta_H SH + \beta_F SF\right)\frac{1}{N}, \\ \frac{\mathrm{d}E}{\mathrm{d}t} = \left(\beta_I SI + \beta_H SH + \beta_F SF\right)\frac{1}{N} - \alpha E, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \alpha E - \left[\gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right) + \gamma_H\theta_1 + \delta_1\left(1-\theta_1\right)\gamma_D\right] I, \\ \frac{\mathrm{d}H}{\mathrm{d}t} = \gamma_H\theta_1 I - \left[\gamma_{DH}\delta_2 + \gamma_{IH}\left(1-\delta_2\right)\right] H, \\ \frac{\mathrm{d}F}{\mathrm{d}t} = \gamma_{DH}\delta_2 H + \delta_1\left(1-\theta_1\right)\gamma_D I - \gamma_F F, \\ \frac{\mathrm{d}R}{\mathrm{d}t} = \gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right) I + \gamma_{IH}\left(1-\delta_2\right) H, \\ \frac{\mathrm{d}B}{\mathrm{d}t} = \gamma_F F. \end{cases} \tag{2.1.3}$$

From the model system (2.1.3), we deduce that $\frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}E}{\mathrm{d}t} + \frac{\mathrm{d}I}{\mathrm{d}t} + \frac{\mathrm{d}H}{\mathrm{d}t} + \frac{\mathrm{d}F}{\mathrm{d}t} + \frac{\mathrm{d}R}{\mathrm{d}t} + \frac{\mathrm{d}B}{\mathrm{d}t} = 0$, which confirms that the size of the population under study is constant.

We assume that all the model parameters are non negative and the initial condition of the model system (2.1.3) are given by; $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $H(0) \geq 0$, $F(0) \geq 0$, $R(0) \geq 0$, $B(0) \geq 0$. The definitions and descriptions of parameters in system (2.1.3) are presented in Table 2.1.

We now normalize the system (2.1.3) as follows. We let;

$$\frac{S}{N} = s, \quad \frac{I}{N} = i, \quad \frac{E}{N} = e, \quad \frac{H}{N} = h, \quad \frac{F}{N} = f, \quad \frac{R}{N} = r, \quad \frac{B}{N} = b,$$

so that the normalized form of system (2.1.3) is;

$$\begin{cases} \frac{\mathrm{d}s}{\mathrm{d}t} = -\left(\beta_I i + \beta_H h + \beta_F f\right) s, \\ \frac{\mathrm{d}e}{\mathrm{d}t} = \left(\beta_I i + \beta_H h + \beta_F f\right) s - \alpha e, \\ \frac{\mathrm{d}i}{\mathrm{d}t} = \alpha e - \left[\gamma_I \left(1-\theta_1\right)\left(1-\delta_1\right) + \gamma_H \theta_1 + \delta_1 \left(1-\theta_1\right)\gamma_D\right] i, \\ \frac{\mathrm{d}h}{\mathrm{d}t} = \gamma_H \theta_1 i - \left[\gamma_{DH}\delta_2 + \gamma_{IH}\left(1-\delta_2\right)\right] h, \\ \frac{\mathrm{d}f}{\mathrm{d}t} = \gamma_{DH}\delta_2 h + \delta_1\left(1-\theta_1\right)\gamma_D i - \gamma_F f, \\ \frac{\mathrm{d}r}{\mathrm{d}t} = \gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right) i + \gamma_{IH}\left(1-\delta_2\right) h, \\ \frac{\mathrm{d}b}{\mathrm{d}t} = \gamma_F f. \end{cases} \tag{2.1.4}$$

Table 2.1: Description of model parameters

Description	Symbol	Units
Transmission rate in the community	β_I	per day
Transmission rate at the hospital	β_H	per day
Transmission rate during funerals	β_F	per day
Average incubation period	$\frac{1}{\alpha}$	per day
Mean duration from symptom onset to hospitalization	$\frac{1}{\gamma_H}$	days
Mean duration from hospitalization to death	$\frac{1}{\gamma_{DH}}$	days
Mean duration of the infectious period for survivors	$\frac{1}{\gamma_I}$	days
Mean duration from hospitalization to end of infectiousness for survivors	$\frac{1}{\gamma_{IH}}$	days
Mean duration of the infectious in a community to death	$\frac{1}{\gamma_D}$	days
Mean duration from death to burial	$\frac{1}{\gamma_F}$	days
Probability of joining the hospital for an infected population	θ_1	unit-less
Probability of dying for infected population in the community	δ_1	unit-less
Proportion of hospitalized individuals who die from the disease	δ_2	unit-less

- θ_1 is included in this model in order to identify the proportion of hospitalized infectious individuals (θ%) (Camacho et al., 2014).

$$\theta_1 = \frac{\theta\left[\gamma_I\left(1-\delta_1\right) + \gamma_D\delta_1\right]}{\theta\left[\gamma_I\left(1-\delta_1\right) + \gamma_D\delta_1\right] + \left(1-\theta\right)\gamma_H}. \tag{2.1.5}$$

- Similarly, δ_1 and δ_2 combined represent the overall case fatality ratio δ (Camacho et al., 2014).

$$\begin{cases} \delta_1 &= \frac{\delta\gamma_I}{\delta\gamma_I+(1-\delta)\gamma_D}, \\ \delta_2 &= \frac{\delta\gamma_{IH}}{\delta\gamma_{IH}+(1-\delta)\gamma_{DH}}. \end{cases} \tag{2.1.6}$$

- γ_{DH} and γ_{IH} are the inverse of the mean time from hospitalization to death and recovery respectively (Camacho et al., 2014).

$$\begin{cases} \gamma_{DH} &= \frac{1}{\frac{1}{\gamma_D}-\frac{1}{\gamma_H}} = \frac{\gamma_D\gamma_H}{\gamma_H-\gamma_D}, \\ \gamma_{IH} &= \frac{1}{\frac{1}{\gamma_I}-\frac{1}{\gamma_H}} = \frac{\gamma_I\gamma_H}{\gamma_H-\gamma_I}. \end{cases} \tag{2.1.7}$$

Next, we determine the basic reproduction number of the model system (2.1.4).

2.2 Basic Reproduction Number

The basic reproduction number denoted by R_0, is the average number of secondary infections produced when one infected individual is introduced into a host population where everyone is susceptible. It determines how much work has to be done to control the epidemic (Heffernan et al., 2005).

If $R_0 < 1$, then on average, an infected individual produces less than one new infected individual over his or her infectious period. This shows that the infection will die out. One can say that the infection can be controlled. On the other hand, if $R_0 > 1$, then each infected individual generates, on average, more than one new infected individual. This indicates that the disease spreads in the population (van den Driessche and Watmough, 2002). However, $R_0 = 1$ indicates that one infected individual infects exactly one individual. This implies that the disease prevalence will remain constant in the population (persist endemically) (Holme and Masuda, 2015).

Generally, the computation of model reproduction number as described in (van den Driessche and Watmough, 2002) is given as follows. Let $X = (X_i, i = 1, 2, ..., n)$ denote the number or proportion of individuals in the i^{th} compartment. Also, let $\mathcal{F}_i(X)$ be the rate of appearance of new infections in compartment i, and $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$ be the rate of transfer of individuals into $(\mathcal{V}_i^+)$ and out $(\mathcal{V}_i^-)$ of compartment i. It is assumed that each function $\left(\mathcal{F}_i(X), \mathcal{V}_i^+, \mathcal{V}_i^-\right)$ is continuously differentiable at least twice with respect to each variable (Heffernan et al., 2005). The difference $\mathcal{F}_i(X) - \mathcal{V}_i(X)$ gives the rate of change in X. The model reproduction number computed at the disease free equilibrium is the spectral radius of the next generation matrix FV^{-1}.

Using the procedure described above, we now calculate the reproduction number for the Ebola model system (2.1.4). We first distinguish new infections from all other changes in the population. The infected compartments under consideration include the Exposed, Infected in the community, Infected in hospitals and the Infected at funerals. We thus consider the following equations from our model system

(2.1.4).

$$\begin{cases} \dfrac{\mathrm{d}e}{\mathrm{d}t} = (\beta_I i + \beta_H h + \beta_F f)\, s - \alpha e, \\ \dfrac{\mathrm{d}i}{\mathrm{d}t} = \alpha e - [\gamma_I\,(1-\theta_1)\,(1-\delta_1) + \gamma_H\theta_1 + \delta_1\,(1-\theta_1)\,\gamma_D]\, i, \\ \dfrac{\mathrm{d}h}{\mathrm{d}t} = \gamma_H\theta_1 i - [\gamma_{DH}\delta_2 + \gamma_{IH}\,(1-\delta_2)]\, h, \\ \dfrac{\mathrm{d}f}{\mathrm{d}t} = \gamma_{DH}\delta_2 h + \delta_1\,(1-\theta_1)\,\gamma_D i - \gamma_F f. \end{cases} \tag{2.2.1}$$

From the sub-system (2.2.1), $\mathcal{F}$ and $\mathcal{V}$ are given respectively as:

$$\mathcal{F} = \begin{pmatrix} (\beta_I i + \beta_H h + \beta_F f)\, s \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} \alpha e \\ -\alpha e + y i \\ -\gamma_H\theta_1 i + [\gamma_{DH}\delta_2 + \gamma_{IH}\,(1-\delta_2)]\, h \\ -\gamma_{DH}\delta_2 h - \delta_1\,(1-\theta_1)\,\gamma_D i + \gamma_F f \end{pmatrix},$$

where $y = \gamma_I\,(1-\theta_1)\,(1-\delta_1) + \gamma_H\theta_1 + \delta_1\,(1-\theta_1)\,\gamma_D$.

Given E_0 as the disease free equilibrium, we calculate the non-negative matrix F and the non-singular matrix V as follows. $F = \left[\frac{\partial \mathcal{F}_i(E_0)}{\partial X_j}\right]$ and $V = \left[\frac{\partial \mathcal{V}_i(E_0)}{\partial X_j}\right]$. i and j run from 1 to 4 denote the number of infected classes (Heffernan et al., 2005).

We therefore have;

$$F = \begin{pmatrix} 0 & \beta_I & \beta_H & \beta_F \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad \text{and} \quad V = \begin{pmatrix} \alpha & 0 & 0 & 0 \\ -\alpha & z & 0 & 0 \\ 0 & -\gamma_H\theta_1 & \delta_2\gamma_{DH} + (1-\delta_2)\gamma_{IH} & 0 \\ 0 & -\delta_1\gamma_D(1-\theta_1) & -\delta_2\gamma_{DH} & \gamma_F \end{pmatrix},$$

where $z = \gamma_H\theta_1 + \gamma_I(1-\delta_1)(1-\theta_1) + \delta_1\gamma_D(1-\theta_1)$.

The next generation matrix FV^{-1} is given by;

$$FV^{-1} = \begin{pmatrix} \kappa_1 & \kappa_2 & \kappa_3 & \kappa_4 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{2.2.2}$$

where

$$\kappa_1 = \frac{\beta_I}{\delta_1\gamma_D(1-\theta_1) + \gamma_I(1-\delta_1)(1-\theta_1) + \gamma_H\theta_1} + \frac{\gamma_H\theta_1}{(\delta_1\gamma_D(1-\theta_1) + \gamma_I(1-\delta_1)(1-\theta_1) + \gamma_H\theta_1)(\delta_2\gamma_{DH} + (1-\delta_2)\gamma_{IH})}\beta_H + \frac{\left(\frac{\delta_1\gamma_D(1-\theta_1)}{\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1} + \frac{\delta_2\gamma_{DH}\gamma_H\theta_1}{(\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1)(\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH})}\right)\beta_F}{\gamma_F}.$$

$$\kappa_2 = \frac{\left(\frac{\delta_1\gamma_D(1-\theta_1)}{\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1} + \frac{\delta_2\gamma_{DH}\gamma_H\theta_1}{(\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1)(\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH})}\right)\beta_F}{\gamma_F} + \frac{\gamma_H\theta_1}{(\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1)(\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH})}\beta_H + \frac{\beta_I}{\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1},$$

$$\kappa_3 = \frac{\beta_F\delta_2\gamma_{DH}}{(\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH})\gamma_F} + \frac{\beta_H}{\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH}},$$

$$\kappa_4 = \frac{\beta_F}{\gamma_F}.$$

The basic reproduction number R_0 is the dominant eigenvalue of the next generation matrix, i.e. $\rho\left(FV^{-1}\right)$. Since the matrix in (2.2.2) is an upper triangle matrix, the set of eigenvalues is the set of elements in main diagonal. The model R_0 is thus κ_1; that is,

$$R_0 = \frac{\beta_I}{\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1} + \frac{\gamma_H\theta_1}{(\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1)(\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH})}\beta_H + \frac{\left(\frac{\delta_1\gamma_D(1-\theta_1)}{\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1} + \frac{\delta_2\gamma_{DH}\gamma_H\theta_1}{(\delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1)(\delta_2\gamma_{DH}+(1-\delta_2)\gamma_{IH})}\right)\beta_F}{\gamma_F} \quad (2.2.3)$$

If we let $\Phi = \delta_1\gamma_D(1-\theta_1)+\gamma_I(1-\delta_1)(1-\theta_1)+\gamma_H\theta_1$, and $\Theta = \gamma_{DH}\delta_2+\gamma_{IH}(1-\delta_2)$ throughout the report, then the model R_0 can be expressed as follows.

$$\begin{aligned} R_0 &= \frac{\beta_I}{\Phi} + \frac{\gamma_H\theta_1}{\Theta\Phi}\beta_H + \frac{\frac{\gamma_{DH}\delta_2\gamma_H\theta_1}{\Theta} + \gamma_D(1-\theta_1)\delta_1}{\Phi\gamma_F}\beta_F, \\ R_0 &= \frac{\beta_I}{\Phi} + \frac{\gamma_H\theta_1}{\Theta\Phi}\beta_H + \frac{\delta\beta_F}{\gamma_F}, \\ R_0 &= R_{0I} + R_{0H} + R_{0F}. \end{aligned} \quad (2.2.4)$$

We observe that the basic reproduction number of the Ebola disease is the sum of the reproduction numbers of the three infectious compartments in the community, hospital and funerals. This means that the Ebola intervention strategy should focus on reducing transmission in communities, hospitals and in funerals.

2.3 Model Disease Free Equilibrium Point

In this work, we shall only focus on the model disease-free equilibrium point (E_0) because Ebola is an epidemic disease (not endemic). It is obtained by solving system (2.1.3) when the right hand side is equal to zero. Relying on the assumption that the infected individuals do not exist before the onset (the community is free of the disease at the beginning), the population in all compartments are zero except for the susceptible compartment S; that is, $E = 0$, $I = 0$, $H = 0$, $F = 0$. It is clear that in the

absence of the disease, there are no recoveries and burials, therefore $R = 0$ and $B = 0$. Since $N = S$ in the absence of the disease, the disease free equilibrium point for model system (2.1.3) is therefore given by: $E_0 = (N, 0, 0, 0, 0, 0, 0)$. For the normalized system (2.1.4), we have $E_0 = (1, 0, 0, 0, 0, 0, 0)$. In the next section, we analyse the stability of E_0.

2.4 Stability Analysis of the Disease Free Equilibrium Point

In this section, we analyse the stability of the disease free equilibrium point (E_0). We evaluate the Jacobian matrix of the model system (2.1.4) at E_0. Stability of E_0 is then determined based on the sign of the eigenvalues of the corresponding Jacobian matrix (Marc R. Roussel, 2005). The point E_0 is said to be stable if all the eigenvalues of the Jacobian matrix at E_0 have negative real parts and unstable if at least one of the eigenvalues has a positive real part. The Jacobian matrix for the system (2.1.4) is given by;

$$J = \begin{pmatrix} -A & 0 & -\beta_I s & -\beta_H s & -\beta_F s & 0 & 0 \\ A & -\alpha & \beta_I s & \beta_H s & \beta_F s & 0 & 0 \\ 0 & \alpha & -[\gamma_I(1-\delta_1)(1-\theta_1) + \gamma_H\theta_1 + \delta_1\gamma_D(1-\theta_1)] & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_H\theta_1 & -[\delta_2\gamma_{DH} + (1-\delta_2)\gamma_{IH}] & 0 & 0 & 0 \\ 0 & 0 & \delta_1(1-\theta_1)\gamma_D & \delta_2\gamma_{DH} & -\gamma_F & 0 & 0 \\ 0 & 0 & \gamma_I(1-\delta_1)(1-\theta_1) & (1-\delta_2)\gamma_{IH} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma_F & 0 & 0 \end{pmatrix}, \tag{2.4.1}$$

where $A = (\beta_I i + \beta_H h + \beta_F f)$.

At E_0, the Jacobian matrix becomes;

$$J(E_0) = \begin{pmatrix} 0 & 0 & -\beta_I & -\beta_H & -\beta_F & 0 & 0 \\ 0 & -\alpha & \beta_I & \beta_H & \beta_F & 0 & 0 \\ 0 & \alpha & -[\gamma_I(1-\delta_1)(1-\theta_1) + \gamma_H\theta_1 + \delta_1\gamma_D(1-\theta_1)] & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_H\theta_1 & -[\delta_2\gamma_{DH} + (1-\delta_2)\gamma_{IH}] & 0 & 0 & 0 \\ 0 & 0 & \delta_1(1-\theta_1)\gamma_D & \delta_2\gamma_{DH} & -\gamma_F & 0 & 0 \\ 0 & 0 & \gamma_I(1-\delta_1)(1-\theta_1) & (1-\delta_2)\gamma_{IH} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma_F & 0 & 0 \end{pmatrix}. \tag{2.4.2}$$

The eigenvalues of the Jacobian matrix in (2.4.2) are calculated as follows;

$$\begin{aligned} det\,(J(E_0) - \lambda I) &= 0, \\ -\lambda^3 B &= 0, \end{aligned} \tag{2.4.3}$$

where

$$B = \begin{vmatrix} -\alpha - \lambda & \beta_I & \beta_H & \beta_F \\ \alpha & -[\gamma_I(1-\delta_1)(1-\theta_1) + \gamma_H\theta_1 + \delta_1\gamma_D(1-\theta_1)] - \lambda & 0 & 0 \\ 0 & \gamma_H\theta_1 & -[\delta_2\gamma_{DH} + (1-\delta_2)\gamma_{IH}] - \lambda & 0 \\ 0 & \delta_1(1-\theta_1)\gamma_D & \delta_2\gamma_{DH} & -\gamma_F - \lambda \end{vmatrix}, \tag{2.4.4}$$

I is 7×7 unity matrix and λ represents eigenvalues (λ_i where $i = 1, 2, \ldots, 7$).

Next, consider B as a matrix and not as determinant and remove λ's on the main diagonal. Using the echelon method, we make B a lower triangular matrix. This manipulation only affects the first row of B. The other rows remain constant. Without loss of generality, we find three eigenvalues from the last three rows on the main diagonal. The fourth eigenvalue is obtained based on the following proposition.

2.4.1 Proposition If B is a square matrix with eigenvalues λ_i $i = 1, 2, \ldots, n$, then the trace of the matrix B is equal to the sum of all eigenvalues (Yuan et al., 2003).

The fourth eigenvalue is thus $-\alpha$. The solutions of the characteristic equation in (2.4.3) can be read by inspection; so that we have the following eigenvalues,

$$\begin{aligned}
\lambda_1 &= \lambda_2 = \lambda_3 = 0, \\
\lambda_4 &= -\alpha, \\
\lambda_5 &= -\left[\gamma_I(1-\delta_1)(1-\theta_1) + \gamma_H\theta_1 + \delta_1\gamma_D(1-\theta_1)\right], \\
\lambda_6 &= -\left[\delta_2\gamma_{DH} + (1-\delta_2)\gamma_{IH}\right], \\
\lambda_7 &= -\gamma_F.
\end{aligned}$$

Since δ_1, θ_1, and δ_2 are in $(0, 1)$, then, $(1-\delta_1)$, $(1-\delta_2)$ and $(1-\theta_1)$ must also be non negative. This confirms that the eigenvalues λ_5 and λ_6 are negative always. Therefore, λ_4, λ_5, λ_6 and λ_7 are real and negative. We also observe that eigenvalues λ_1, λ_2 and λ_3 are all zero. Based on the results of our calculation, we cannot conclude whether E_0 is stable or not.

3. Extended Ebola Model

In this chapter, we extend the formulated Ebola model in Chapter 2 (see Figure 2.1). We use the calculated type reproduction number from the extended model to establish the minimum effort needed to control the Ebola disease.

The deterministic Ebola model discussed in Chapter 2 (see Figure 2.1) by sub-dividing the susceptible population compartment S (into S_C, S_H and S_F) based on the different population settings. S_H refers to the susceptible population in the hospital environment. They contract the Ebola virus from hospitalized individuals H. This compartment is mainly composed of the healthcare workers in hospital. Upon infection at the hospital, these individuals are allowed to join the infected compartment I_H (infected at the hospital environment). The susceptible population at funerals S_F get infected upon physical contact with the dead during funeral ceremonies. Upon infection, they are termed as infected at funerals I_F. Lastly, we have the susceptible individuals in the community S_C. When individuals contract the Ebola virus away from hospitals and during burial ceremonies, they are hereby assumed to have been infected in the community. We then allow them to join compartment of those infected in the community I_C. The infected populations may experience one of three outcomes: They may seek treatment from the hospital, recover or die of the Ebola disease. The hospitalised Ebola patients may recover upon successful treatment or simply die of Ebola. The dead individuals F remain infectious until they are buried B. The parameters α_H and α_C represent the rate of movement from S_H to S_C and from S_C to S_F compartments respectively. Similarly, the parameters ω_F and ω_C represent the rate at which people move from S_F to S_C and from S_C to S_H respectively. The infected population compartment I is subsequently sub-divided into three compartments, I_C, I_H and I_F.

Other model assumptions employed in our extended model include the following: We assume only homogeneous mixing among the susceptible population. We ignore the exposed compartment while considering the incubation period. We assume also that, the asymptomatic infected individuals return to the community upon infection from other places (in hospitals and funerals). In addition, we assume a constant progression rate $\gamma_H\theta_1$ from different newly infected compartments to the hospital compartment. We then assume that, those who do not attend hospitals join the recovery compartment R at the same rate $\gamma_I(1-\theta_1)(1-\delta_1)$. We assume further that the mortality rate $\delta_1(1-\theta_1)\gamma_D$ is the same for all newly infected individuals. We further assume a constant mean duration from disease onset to hospitalization $\frac{1}{\gamma_H}$. Moreover, we assume that the average time from onset of infection to survival $\frac{1}{\gamma_I}$ is the same for all newly infected compartments. We finally assume that the average time from onset to death $\frac{1}{\gamma_D}$ is the same for all newly infected compartments.

The schematic representation of the above model descriptions is as shown in Figure 3.1.

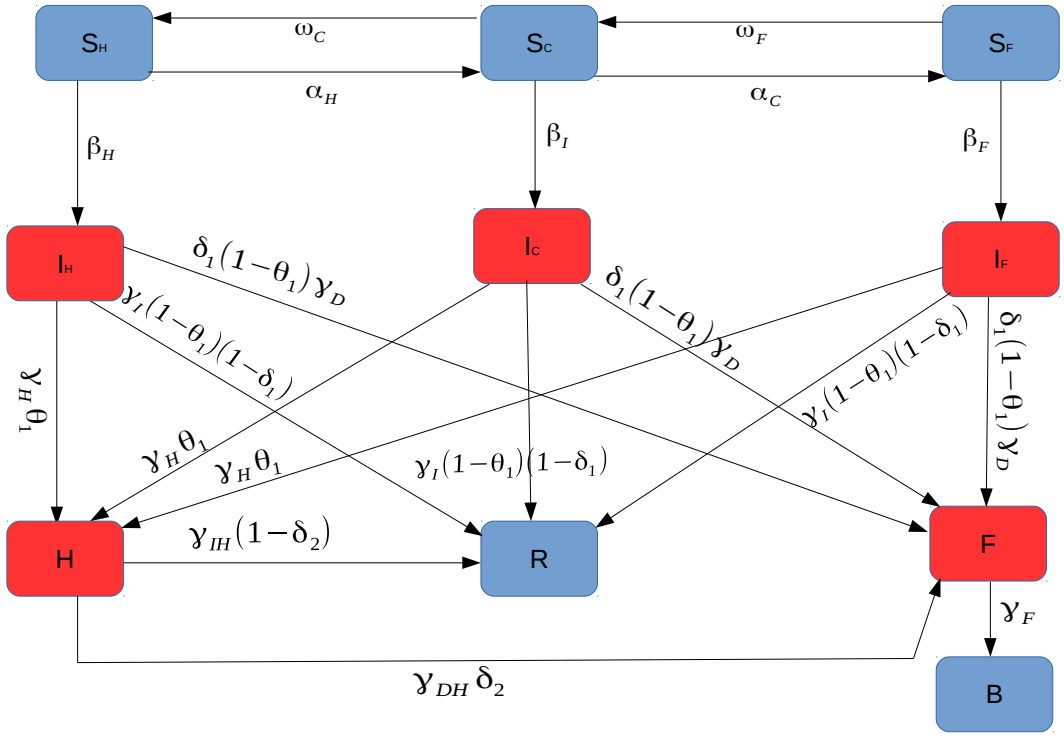

Figure 3.1: Schematic representation of the extended Ebola model.

The normalized system of ordinary differential equations representing the dynamics from the extended model is as follows:

$$
\begin{cases}
\dfrac{\mathrm{d}s_H}{\mathrm{d}t} = -\beta_H s_H h - s_H \alpha_H + s_C \omega_C, \\
\dfrac{\mathrm{d}s_C}{\mathrm{d}t} = -\beta_I s_C \left(i_H + i_C + i_F\right) - s_C \left(\alpha_C + \omega_C\right) + s_F \omega_F + s_H \alpha_H, \\
\dfrac{\mathrm{d}s_F}{\mathrm{d}t} = -\beta_F s_F f - s_F \omega_F + s_C \alpha_C, \\
\dfrac{\mathrm{d}i_H}{\mathrm{d}t} = \beta_H s_H h - \left[\gamma_H \theta_1 + \gamma_I \left(1-\theta_1\right)\left(1-\delta_1\right) + \delta_1 \left(1-\theta_1\right)\gamma_D\right] i_H, \\
\dfrac{\mathrm{d}i_C}{\mathrm{d}t} = \beta_I s_C \left(i_H + i_C + i_F\right) - \left[\gamma_H \theta_1 + \gamma_I \left(1-\theta_1\right)\left(1-\delta_1\right) + \delta_1 \left(1-\theta_1\right)\gamma_D\right] i_C, \\
\dfrac{\mathrm{d}i_F}{\mathrm{d}t} = \beta_F s_F f - \left[\gamma_H \theta_1 + \gamma_I \left(1-\theta_1\right)\left(1-\delta_1\right) + \delta_1 \left(1-\theta_1\right)\gamma_D\right] i_F, \\
\dfrac{\mathrm{d}h}{\mathrm{d}t} = \gamma_H \theta_1 \left(i_H + i_I + i_F\right) - \left[\gamma_{DH}\delta_2 + \gamma_{IH}\left(1-\delta_2\right)\right] h, \\
\dfrac{\mathrm{d}f}{\mathrm{d}t} = \gamma_{DH}\delta_2 h + \delta_1 \left(1-\theta_1\right)\gamma_D \left(i_H + i_C + i_F\right) - \gamma_F f, \\
\dfrac{\mathrm{d}r}{\mathrm{d}t} = \gamma_I \left(1-\theta_1\right)\left(1-\delta_1\right)\left(i_H + i_C + i_F\right) + \gamma_{IH}\left(1-\delta_2\right) h, \\
\dfrac{\mathrm{d}b}{\mathrm{d}t} = \gamma_F f,
\end{cases}
\tag{3.0.1}
$$

where $s_H = \frac{S_H}{N}$, $s_C = \frac{S_C}{N}$, $s_F = \frac{S_F}{N}$, $i_H = \frac{I_H}{N}$, $i_C = \frac{I_C}{N}$, $i_F = \frac{I_F}{N}$, $h = \frac{H}{N}$, $f = \frac{F}{N}$, $r = \frac{R}{N}$ and $b = \frac{B}{N}$.

In this extended model, we sub-divide the population into epidemiologically different infected groups based on their characteristics; that is, the population is assumed to mix heterogeneously. To point out the control effort needed, we target a particular host type rather than the population as a whole. When a single type of the population is targeted, we refer to the quantity T, known as *the type reproduction number* instead of R_0. We now need to focus on three infected compartments i_H, i_C and i_F. We shall now establish the type reproduction number in the following section.

3.1 Type Reproduction Number

3.1.1 Definition: The type reproduction number, T is the average number of cases in individuals of type i, caused by one infected individual of type i, in an entirely susceptible population, either directly or through chains of infection passing through any sequence of the other types (Heesterbeek and Roberts, 2007).

The type reproduction number is computed based on the following formula;

$$T = e^t K \left(I - (I - P) K\right)^{-1} e, \tag{3.1.1}$$

where I is the identity matrix, e is the unit vector, e^t is the matrix transpose of e, P is the projection matrix (that is, for type i, $P_{ii} = 1$ and $P_{mn} = 0$ for all m, n different from i) and K is the next generation matrix.

To calculate the type reproduction number, we begin by calculating the next generation matrix K from the extended model. We then categorise and subdivide the extended model system (3.0.1) into susceptibles, infectious and non-infectious compartments as follows;

The sub-system of the susceptible compartments only, is given by;

$$\begin{cases} \dfrac{\mathrm{d}s_H}{\mathrm{d}t} = -\beta_H s_H h - s_H \alpha_H + s_C \omega_C, \\ \dfrac{\mathrm{d}s_C}{\mathrm{d}t} = -\beta_I s_C \left(i_H + i_C + i_F\right) - s_C \left(\alpha_C + \omega_C\right) + s_F \omega_F + s_H \alpha_H, \\ \dfrac{\mathrm{d}s_F}{\mathrm{d}t} = -\beta_F s_F f - s_F \omega_F + s_C \alpha_C. \end{cases} \tag{3.1.2}$$

Similarly, the sub-system of the infectious compartments is given by;

$$\begin{cases} \dfrac{\mathrm{d}i_H}{\mathrm{d}t} = \beta_H s_H h - \left[\gamma_H \theta_1 + \gamma_I \left(1 - \theta_1\right)\left(1 - \delta_1\right) + \delta_1 \left(1 - \theta_1\right) \gamma_D\right] i_H, \\ \dfrac{\mathrm{d}i_C}{\mathrm{d}t} = \beta_I s_C \left(i_H + i_C + i_F\right) - \left[\gamma_H \theta_1 + \gamma_I \left(1 - \theta_1\right)\left(1 - \delta_1\right) + \delta_1 \left(1 - \theta_1\right) \gamma_D\right] i_C, \\ \dfrac{\mathrm{d}i_F}{\mathrm{d}t} = \beta_F s_F f - \left[\gamma_H \theta_1 + \gamma_I \left(1 - \theta_1\right)\left(1 - \delta_1\right) + \delta_1 \left(1 - \theta_1\right) \gamma_D\right] i_F, \\ \dfrac{\mathrm{d}h}{\mathrm{d}t} = \gamma_H \theta_1 \left(i_H + i_I + i_F\right) - \left[\gamma_{DH} \delta_2 + \gamma_{IH} \left(1 - \delta_2\right)\right] h, \\ \dfrac{\mathrm{d}f}{\mathrm{d}t} = \gamma_{DH} \delta_2 h + \delta_1 \left(1 - \theta_1\right) \gamma_D \left(i_H + i_C + i_F\right) - \gamma_F f. \end{cases} \tag{3.1.3}$$

The last sub-system is that of non-infectious compartments and is given by;

$$\begin{cases} \dfrac{\mathrm{d}r}{\mathrm{d}t} = \gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right)\left(i_H+i_C+i_F\right)+\gamma_{IH}\left(1-\delta_2\right)h, \\ \dfrac{\mathrm{d}b}{\mathrm{d}t} = \gamma_F f. \end{cases} \tag{3.1.4}$$

We adopt the same procedure in (van den Driessche and Watmough, 2002) to calculate the next generation matrix K, of the extended model. Assuming the infected compartments are i_H, i_C, i_F, h and f, we have;

$$\mathcal{F} = \begin{pmatrix} \beta_H s_H h \\ \beta_I s_C\left(i_H+i_C+i_F\right) \\ \beta_F s_F f \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} \left[\gamma_H\theta_1+\gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right)+\delta_1\left(1-\theta_1\right)\gamma_D\right]i_H \\ \left[\gamma_H\theta_1+\gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right)+\delta_1\left(1-\theta_1\right)\gamma_D\right]i_C \\ \left[\gamma_H\theta_1+\gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right)+\delta_1\left(1-\theta_1\right)\gamma_D\right]i_F \\ -\gamma_H\theta_1\left(i_H+i_C+i_F\right)+\left[\gamma_{DH}\delta_2+\gamma_{IH}\left(1-\delta_2\right)\right]h \\ -\gamma_{DH}\delta_2 H-\delta_1\left(1-\theta_1\right)\gamma_D\left(i_H+i_C+i_F\right)+\gamma_F f \end{pmatrix}.$$

The matrices F and V are respectively given by;

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_H s_H & 0 \\ \beta_I s_I & \beta_I s_I & \beta_I s_I & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_F s_F \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} A & 0 & 0 & 0 & 0 \\ 0 & A & 0 & 0 & 0 \\ 0 & 0 & A & 0 & 0 \\ -\gamma_D\theta_1 & -\gamma_D\theta_1 & -\gamma_D\theta_1 & B & 0 \\ C & C & C & -\gamma_{DH}\delta_2 & \gamma_F \end{pmatrix},$$

where

$$\begin{aligned} A &= \gamma_H\theta_1+\gamma_I\left(1-\theta_1\right)\left(1-\delta_1\right)+\delta_1\left(1-\theta_1\right)\gamma_D, \\ B &= \gamma_{DH}\delta_2+\gamma_{IH}\left(1-\delta_2\right), \\ C &= -\delta_1(1-\theta_1)\gamma_D. \end{aligned}$$

So that, the next generation matrix K becomes;

$$K = \begin{pmatrix} \tau_1 & \tau_1 & \tau_1 & \tau_2 & 0 \\ \tau_3 & \tau_3 & \tau_3 & 0 & 0 \\ \tau_4 & \tau_4 & \tau_4 & \tau_5 & \frac{s_F\beta_F}{\gamma_F} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$\begin{aligned} \tau_1 &= \frac{s_H\beta_H\gamma_D\theta_1}{\Phi\Theta}, \\ \tau_2 &= \frac{s_H\beta_H}{\Theta}, \\ \tau_3 &= \frac{s_I\beta_I}{\Phi}, \\ \tau_4 &= \frac{\left(\frac{\delta_1\gamma_D(1-\theta_1)}{\Phi}+\frac{\delta_2\gamma_D\gamma_{DH}\theta_1}{\Phi\Theta}\right)s_F\beta_F}{\gamma_F}, \\ \tau_5 &= \frac{s_F\beta_F\delta_2\gamma_{DH}}{\Theta\gamma_F}. \end{aligned}$$

Next, we calculate the type reproduction number based on three cases as follows.

Case 1: Consider I_H, the infected population at hospitals to be type 1. In this case, the calculated type reproduction number is given by;

$$T_H = e_H^t K \left(I - (I - P_H) K\right)^{-1} e_H, \tag{3.1.5}$$

where e_H is the first unit vector; which means, the vector for which the first element is equal to one and all other elements are equal to zero, e_H^t is the matrix transpose of e_H, P_H is the projection matrix (that is $P_{H_{11}} = 1$ and $P_{H\,mn} = 0$ for all m, n different from one).

We have,

$$I = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{pmatrix}, \quad P_H = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad e_H = \begin{pmatrix} 1 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

The type reproduction number from equation (3.1.5) is thus;

$$T_H = \frac{s_H \beta_H \gamma_D \gamma_F \theta_1}{\chi},$$

where

$$\begin{aligned} \chi = {} & s_C \beta_I \gamma_F \left(\gamma_{IH} \left(-1 + \delta_2\right) - \gamma_{DH} \delta_2\right) \\ & - \gamma_F \left(\gamma_{IH} \left(-1 + \delta_2\right) - \gamma_{DH} \delta_2\right) \left(\gamma_I \left(-1 + \delta_1\right) \left(-1 + \theta_1\right) - \gamma_D \delta_1 \left(-1 + \theta_1\right) + \gamma_H \theta_1\right) \\ & - s_F \beta_F \gamma_D \left(\gamma_{IH} \delta_1 \left(-1 + \delta_2\right) \left(-1 + \theta_1\right) + \gamma_{DH} \delta_2 \left(-\left(\delta_1 \left(-1 + \theta_1\right)\right) + \theta_1\right)\right). \end{aligned}$$

$\left(1 - \frac{1}{T_H}\right)$ shows the minimum proportion of the population who visit hospitals that need to be vaccinated to prevent the Ebola epidemic.

Case 2: Consider I_C, the infected population in the community, as type 2. In this case

$$T_C = e_C^t K \left(I - (I - P_C) K\right)^{-1} e_C, \tag{3.1.6}$$

where e_C is the second unit vector, e_C^t is the matrix transpose of e_C, P_C is the projection matrix. We now have;

$$P_C = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad e_C = \begin{pmatrix} 0 \\ 1 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

By computing the type reproduction number from equation (3.1.6) we get;

$$T_C = \frac{s_C \beta_I \gamma_F \left(-\left(\gamma_{IH} \left(-1 + \delta_2\right)\right) + \gamma_{DH} \delta_2\right)}{\Psi},$$

where

$$\begin{aligned}\Psi = \gamma_F \left(\left(\gamma_I \left(-1 + \delta_1 \right) - \gamma_D \delta_1 \right) \left(\gamma_{IH} \left(-1 + \delta_2 \right) - \gamma_{DH} \delta_2 \right) \right. \\ \left. - \left(s_H \beta_H \gamma_D + \left(\gamma_H + \gamma_I \left(-1 + \delta_1 \right) - \gamma_D \delta_1 \right) \left(\gamma_{IH} \left(-1 + \delta_2 \right) - \gamma_{DH} \delta_2 \right) \right) \theta_1 \right) \\ - s_F \beta_F \gamma_D \left(\gamma_{IH} \delta_1 \left(-1 + \delta_2 \right) \left(-1 + \theta_1 \right) + \gamma_{DH} \delta_2 \left(- \left(\delta_1 \left(-1 + \theta_1 \right) \right) + \theta_1 \right) \right).\end{aligned}$$

$\left(1 - \frac{1}{T_C}\right)$ is the minimum proportion that need to be vaccinated to prevent the disease in the community.

Case 3: We finally consider I_F, the infected population during funerals, as type 3. In this case

$$T_F = e_F^t K \left(I - \left(I - P_F \right) K \right)^{-1} e_F, \tag{3.1.7}$$

where e_F is the third unit vector, and e_F^t is the matrix transpose of e_F.

$$P_F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad e_F = \begin{pmatrix} 0 \\ 0 \\ 1 \\ 0 \\ 0 \end{pmatrix}.$$

Using the formula in equation (3.1.7), we get the following;

$$T_F = \frac{s_F \beta_F \gamma_D \left(\gamma_{IH} \delta_1 \left(-1 + \delta_2 \right) \left(-1 + \theta_1 \right) + \gamma_{DH} \delta_2 \left(- \left(\delta_1 \left(-1 + \theta_1 \right) \right) + \theta_1 \right) \right)}{\Gamma},$$

where

$$\begin{aligned}\Gamma = \gamma_F \left(\left(s_C \beta_I + \gamma_I \left(-1 + \delta_1 \right) - \gamma_D \delta_1 \right) \left(\gamma_{IH} \left(-1 + \delta_2 \right) - \gamma_{DH} \delta_2 \right) \right. \\ \left. - \left(s_H \beta_H \gamma_D + \left(\gamma_H + \gamma_I \left(-1 + \delta_1 \right) - \gamma_D \delta_1 \right) \left(\gamma_{IH} \left(-1 + \delta_2 \right) - \gamma_{DH} \delta_2 \right) \right) \theta_1 \right).\end{aligned}$$

$\left(1 - \frac{1}{T_F}\right)$ is therefore the minimum proportion of the population who attend funerals that need to be vaccinated to prevent the Ebola disease in funeral.

In conclusion, we observe that we need to vaccinate a proportion greater than $\sum_i \left(1 - \frac{1}{T_i}\right)$, $i = H, C, F$, of the whole population to contain the Ebola epidemics.

4. Numerical Simulations and Results

This chapter presents numerical results of the two models developed in this project.

4.1 Numerical Simulations

The models described in chapters 2 and 3 were numerically simulated to generate results presented in the following sections. Simulations of the two models were carried out using Scilab. In the basic model, simulations were performed to examine the response of the model parameters to the general dynamics of the models and to investigate the effect of various intervention and control strategies. Similar to the basic model, the extended model was simulated with the purpose of identifying the effect of splitting the population into three components such as; community level, hospital, and funeral places necessary for informing targeted control.

Parameter values used in the numerical simulations were derived from literature and are summarized in Table 4.1. Least square method was used to estimate other parameters. For the remaining parameters, their values were derived from other parameters in Table 4.1.

Table 4.1: Parameter definition and their corresponding estimates.

Parameter	Description	Estimates	Source
β_I	Transmission rate in the community	0.10 per day	(Camacho et al., 2014)
β_H	Transmission rate in hospital	3.24 per day	(Camacho et al., 2014)
β_F	Transmission rate during traditional burial	0.78 per day	(Camacho et al., 2014)
$\frac{1}{\alpha}$	Incubation period	5.99 days	(Camacho et al., 2014)
$\frac{1}{\gamma_H}$	Mean time from onset to hospitalization	3.00 days	(Camacho et al., 2014)
$\frac{1}{\gamma_F}$	Mean time from death to burial	0.99 days	(Camacho et al., 2014)
$\frac{1}{\gamma_I}$	Mean time from onset to recovery	10.00 days	(Camacho et al., 2014)
$\frac{1}{\gamma_D}$	Mean time from onset to death	13.31 days	(Camacho et al., 2014)
θ	Proportion of cases hospitalized	0.21	(Camacho et al., 2014)
δ	Case-fatality ratio	0.88	(Camacho et al., 2014)
α_H	Movement rate from S_H to S_C	2.45 per day	Estimated
α_C	Movement rate from S_C to S_F	3.23 per day	Estimated
ω_F	Movement rate from S_F to S_C	2.9 per day	Estimated
ω_C	Movement rate from S_C to S_H	2.2 per day	Estimated

The values of γ_{DH} and γ_{IH} which represent the inverse of the mean time from hospitalization to death and recovery respectively, were obtained from equation (2.1.7) as follows:

$$\begin{cases} \gamma_{DH} &= \frac{1}{\frac{1}{\gamma_D}-\frac{1}{\gamma_H}} = \frac{1}{7.49-3.00} = 0.22271715, \\ \gamma_{IH} &= \frac{1}{\frac{1}{\gamma_I}-\frac{1}{\gamma_H}} = \frac{1}{10.00-3.00} = 0.14285714. \end{cases}$$

Similarly, equation (2.1.6) was used to obtain values for δ_1 and δ_2 as follows:

$$\begin{cases} \delta_1 &= \frac{\delta\gamma_I}{\delta\gamma_I+(1-\delta)\gamma_D} = \frac{0.88\times\frac{1}{10}}{0.88\times\frac{1}{10}+\frac{1-0.88}{7.49}} = 0.84598009, \\ \delta_2 &= \frac{\delta\gamma_{IH}}{\delta\gamma_{IH}+(1-\delta)\gamma_{DH}} = \frac{0.88\times 0.14285714}{0.88\times 0.14285714+(1-0.88)\times 0.22271715} = 0.8224678574. \end{cases}$$

Furthermore, the value for θ_1 was computed from equation (2.1.5) as follows:

$$\begin{aligned} \theta_1 &= \frac{\theta\left[\gamma_I\left(1-\delta_1\right)+\gamma_D\delta_1\right]}{\theta\left[\gamma_I\left(1-\delta_1\right)+\gamma_D\delta_1\right]+\left(1-\theta\right)\gamma_H}, \\ &= \frac{0.21\times\left[\frac{1}{10}\times(1-0.84598009)+\frac{0.84598009}{7.49}\right]}{0.21\times\left[\frac{1}{10}\times(1-0.84598009)+\frac{0.84598009}{7.49}\right]+(1-0.21)\times\frac{1}{3}}, \\ &= 0.092851222. \end{aligned}$$

4.2 Results of the Basic Model

Theoretical results show that $R_0 < 1$ is an indicator of a disease free equilibrium. As expected, results in Figure 4.1 show that the infected groups decrease with time and approach zero after the passage few days.

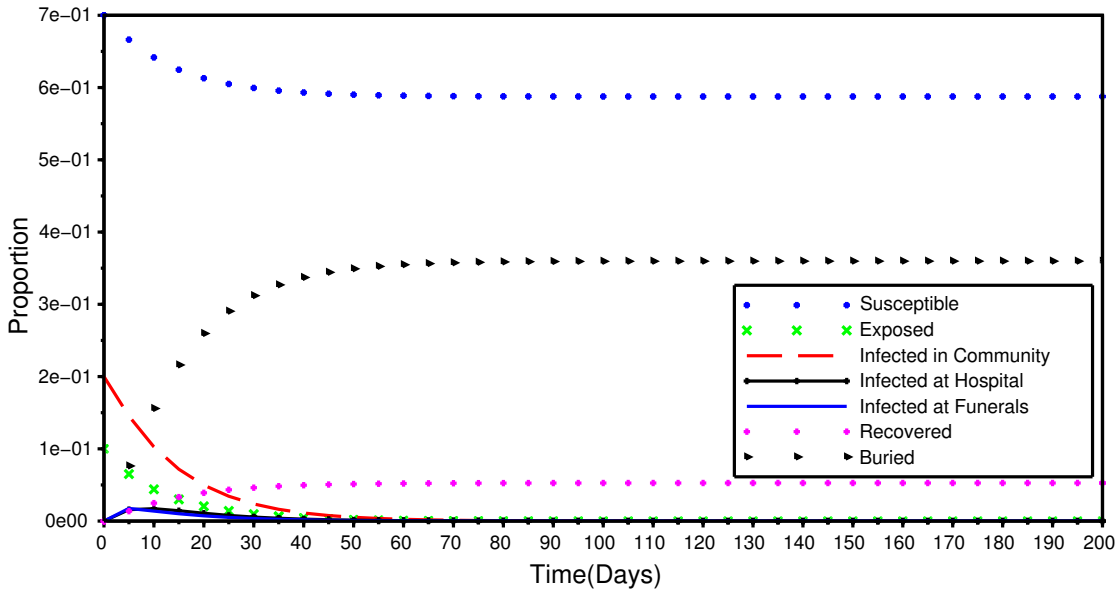

Figure 4.1: Curves representing the simulation study of the basic model when $R_0 < 1$ (when parameters are set to be $\beta_I = 0.10$; $\beta_H = 0.014$; $\beta_F = 0.08$; $\alpha = 0.17$; $\gamma_H = 0.33$; $\gamma_{DH} = 0.21$; $\gamma_I = 0.1$; $\gamma_{IH} = 0.14$; $\gamma_D = 0.13$; $\gamma_F = 1.01$; $\theta_1 = 0.09$; $\delta_1 = 0.84$ and $\delta_2 = 0.82$).

We observe that the susceptible proportion slowly decreases and stabilizes after a short time. The first point where all curves become stable indicates the equilibrium point of the system. In addition, if

there is no new infected individual in the population (at disease free equilibrium, DFE), then the total population size is the same as the susceptible population and is constant while the size of the other compartments are zero.

In Figure (4.2) we present results when $R_0 = 4.61$ (R_0 greater than 1). The susceptible group decreases due to the spread of the disease until the equilibrium point is reached. The size of the buried group grows and converges asymptotically. Moreover, more infected individuals recover due to the successful treatment. In addition, the size of infected individuals in hospitals and in the funerals show a slower increase but also decrease to zero after a certain period. However, the size of the exposed and infected in the community group start to decrease and approach zero.

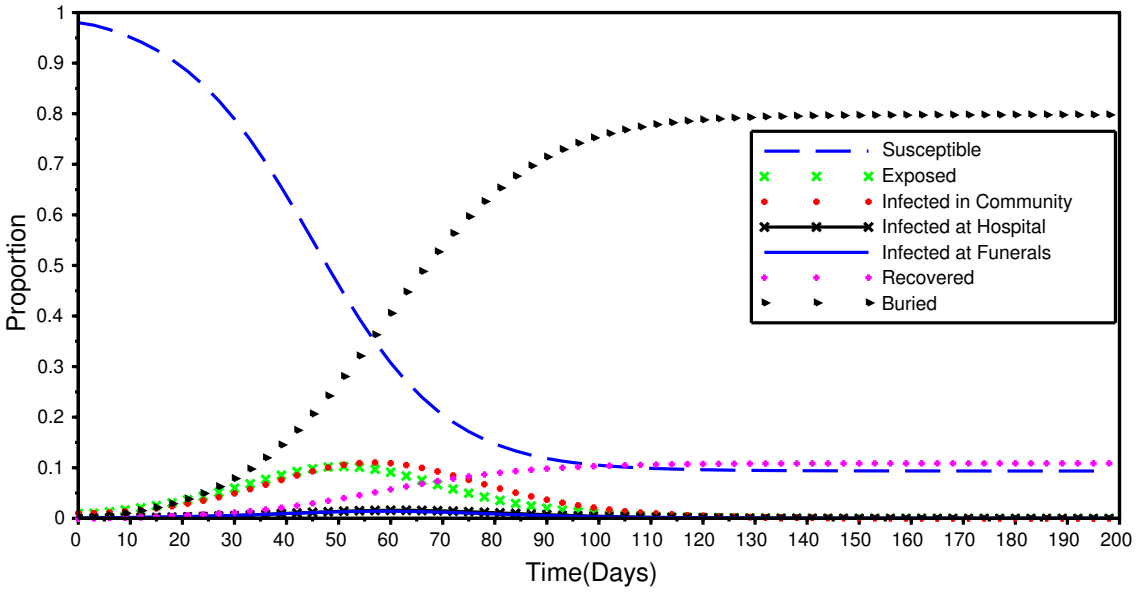

Figure 4.2: Curves representing the simulation study of the basic model when $R_0 = 4.61$. All parameters are the same as parameters in Figure 4.1, except the transmission rates $\beta_H = 3.24$ and $\beta_F = 0.78$, representing the transmission rate at hospitals and during funerals respectively.

4.2.1 Effects of behaviour change on the Ebola epidemic

Behaviour change is related to information given to the population to protect themselves through hand-washing, and minimizing contact with infectious individuals. In this section, we consider the behaviour change on day 2 and day 12 from the onset of the disease as the behaviour change happened earlier and later respectively. Figure 4.3 shows the expected differences when the behaviour change comes earlier or later in the epidemic. We only simulate the infected compartments to observe the effect of change in behaviour among various populations under study.

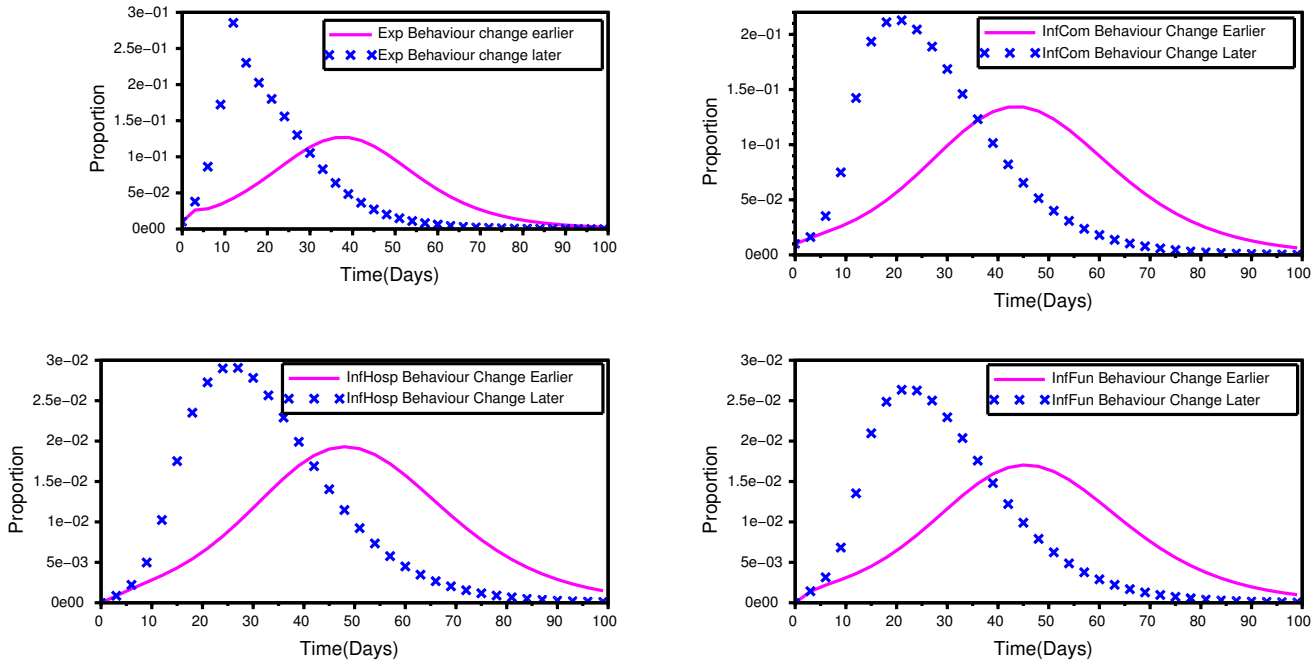

Figure 4.3: Effect of changing behaviour (earlier or later) on the Ebola population dynamics. Exposed (Exp), Infected in the community (InfCom), Infected at hospital (InfHosp) and Infected during funerals (InfFun) compartment. Parameters used are presented in Table 4.1.

From Figure 4.3, we observe different dynamics when behaviour change is applied either early or late. The maximum proportion in each case is higher when behaviour change is applied later than earlier. This indicates that when behaviour change is delayed, more people will be infected than when it occurs early. Also, when the behaviour change is applied earlier, the infected compartments (Exposed, Infected in the community, Infected at hospitals and Infected during funerals) are normally distributed. However, infected compartments are right skewed when the change of behaviour happens later; that is, from the onset of the disease, many people get the Ebola virus in a short time. Once the behaviour change is applied, the disease dies out quickly (in a short period of time). But the infected humans decrease and approach zero as time goes to infinity.

4.2.2 Effects of hospital closure on Ebola dynamics

We assume that the hospitals were closed from the 30^{th} to 60^{th} and from the 85^{th} to 100^{th} day from the onset of the disease. The infectious classes show the up and down oscillations (Figure 4.4).

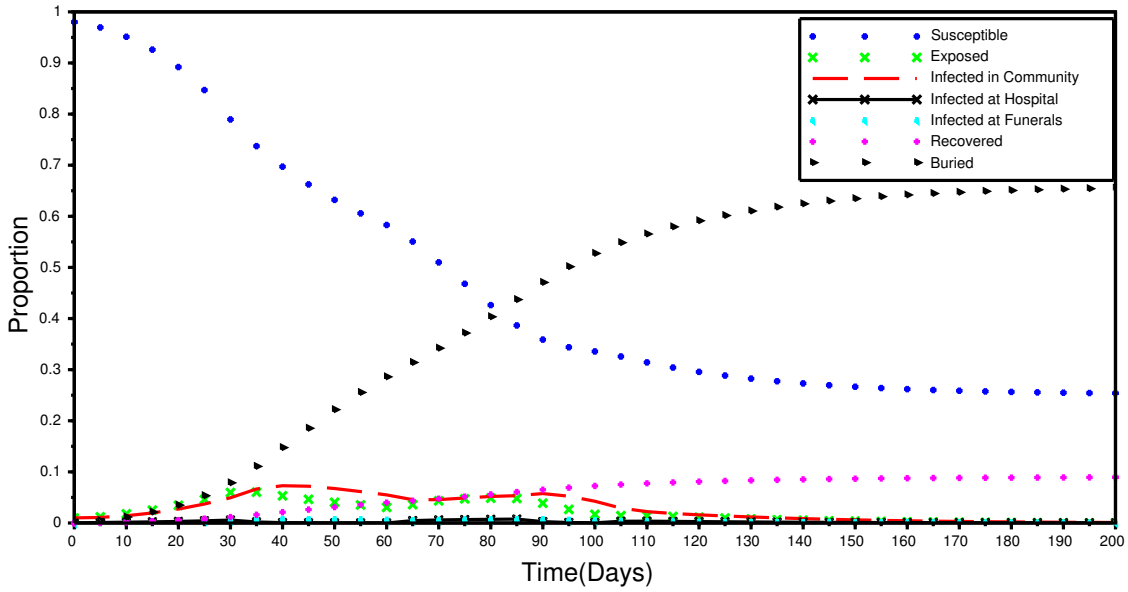

Figure 4.4: Effects of hospital closure on population dynamics. The rate at which people join the hospital, $\gamma_H = 0$ when hospitals are closed and $\gamma_H = 0.33$ when open. Other parameters are as defined in Table 4.1.

We observe that when the hospitals are closed, the proportion of infectious individuals decreases. On the contrary, when the hospitals are open, the proportion of infectious individuals increases because of the high transmission rate of hospitalized individuals.

4.2.3 Effects of treatment on Ebola dynamics

Although there exist no treatments currently, several drugs such as ZMapp, TKM-Ebola are now under development. When the treatment is available in hospitals, the probability of dying from the Ebola reduces, thus increasing survival chances of infected individuals. We simulated the effect of treatment by decreasing the probability of dying in the hospitals (δ_2) from 0.22 to 0.022 and that of the hospital fatality rate per day, γ_{DH} from 0.82 to 0.082.

Figure 4.5 shows that treatment has a significant impact on the lives of patients (Hayden and Reardon, 2014). The effect of treating individuals in hospitals has implications outside hospitals because the burden of the disease is reduced not only for those in hospitals, but also those exposed, infected in communities, and even those in funerals.

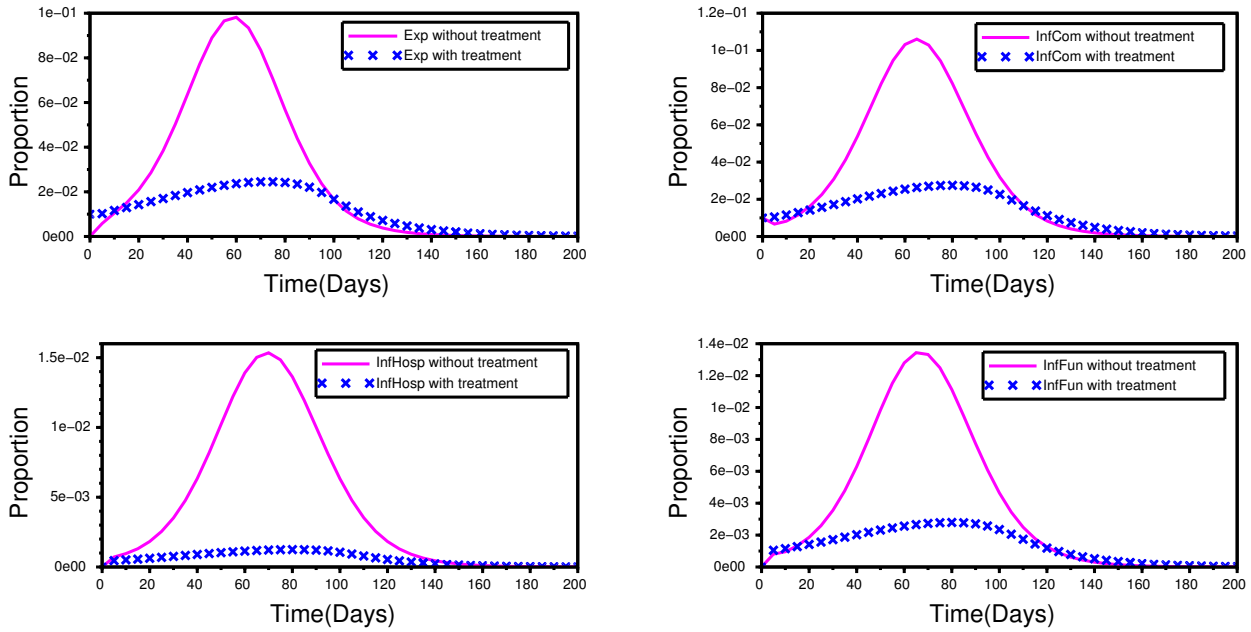

Figure 4.5: Effects of treatment of Ebola. The probability of dying in hospitals, $\delta_2 = 0.022$ and the hospital fatality rate per day $\gamma_{DH} = 0.082$. Other parameters are defined in Table 4.1.

4.2.4 Effects of treatment efficacy on Ebola dynamics

In this subsection, we simulated the treatment efficacy by changing the values of δ_2 and γ_{DH}. When δ_2 is reduced from 0.22 to 0.022 and γ_{DH} reduced from 0.82 to 0.082, treatment efficacy becomes 37% and when both δ_2 and γ_{DH} are set to zero, then treatment efficacy becomes 53%.These two scenarios were compared with simulations performed when all parameter values shown in Table 4.1 were unchanged (i.e. zero treatment efficacy on Ebola patients).

Results indicate that when there is no treatment efficacy, the infected population sizes are higher (Figure 4.6). The sizes of the infected population decrease when the treatment efficacy increases from 0% to 37%. These sizes reduce further when the treatment efficacy increases to 53%.

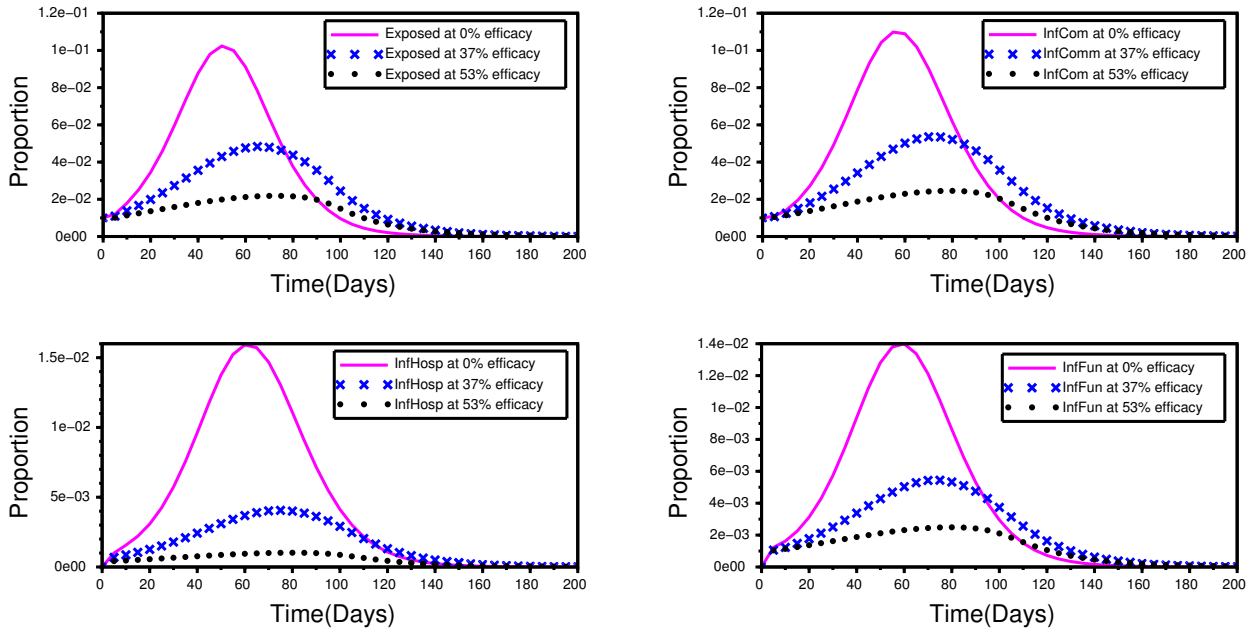

Figure 4.6: Effects of the treatment efficacy. The parameters are defined in Table 4.1.

4.2.5 Effects of healthcare workers vaccination on Ebola dynamics

Ebola vaccines are being developed at biotechnology companies, Profectus pharmaceuticals in Baltimore, Maryland and under programmes sponsored by the US government. The WHO is also investigating the use of serum from people who have had Ebola and recovered; because their blood contains a high concentration of antibodies against the Ebola virus (Hayden and Reardon, 2014). To investigate the effect of vaccination, we simulated this scenario with an assumption that in hospitals, only healthcare workers were susceptible to Ebola and that vaccines protect susceptible individuals from getting infections. In this case, the hospital transmission rate is assumed to be zero. Results indicate that in the absence of vaccine, the infected population in the general population is higher than when the vaccine is available (Figure 4.7).

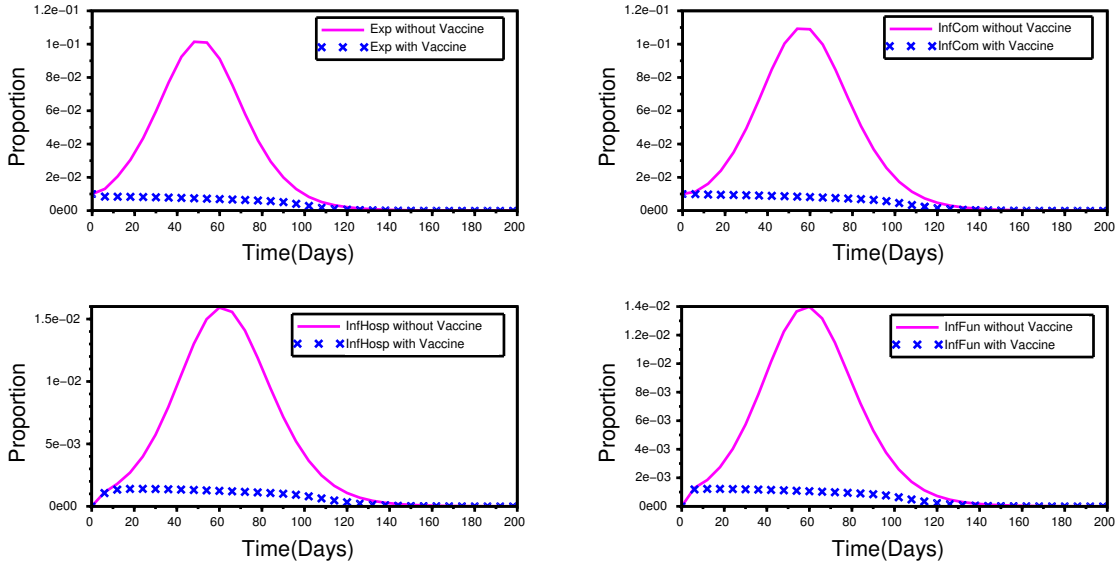

Figure 4.7: Effects of healthcare workers vaccination. The parameters used are shown in Table 4.1.

4.3 Results of the Extended Model

In this section, we present numerical simulation results of the extended model to study the dynamics of the disease when parameters are varied. Figure 4.8 shows the population dynamics when parameters are similar to those used in Figure 4.1. In this case, the disease free equilibrium is reached within a few days. This extended model behaves similarly to the original model when R_0 is less than one.

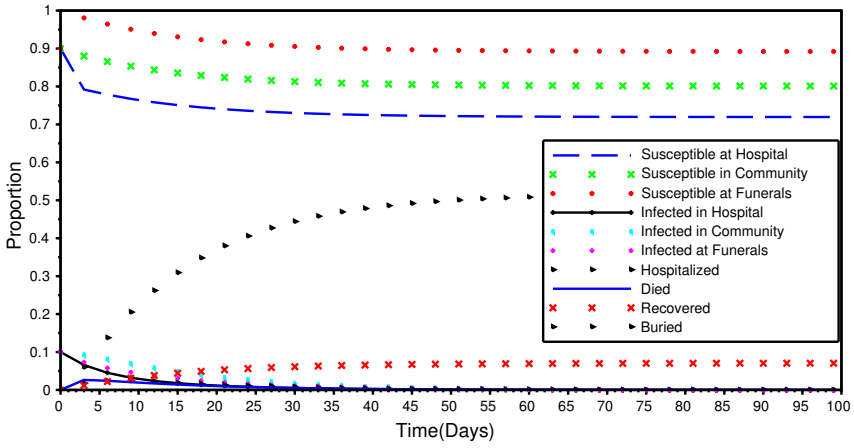

Figure 4.8: Numerical results of the extended model when parameters are set to be $\beta_I = 0.10$; $\beta_H = 0.014$; $\beta_F = 0.08$; $\alpha = 0.17$; $\gamma_H = 0.33$; $\gamma_{DH} = 0.21$; $\gamma_I = 0.1$; $\gamma_{IH} = 0.14$; $\gamma_D = 0.13$; $\gamma_F = 1.01$; $\theta_1 = 0.09$; $\delta_1 = 0.84$ and $\delta_2 = 0.82$. Other parameters are defined in Table 4.1

On the other hand, the infectious groups increase with time to a peak before decreasing towards zero

(Figure 4.9). The proportion of susceptible individuals decrease and become stable at the equilibrium point. We also observe that individuals recover as the disease continue spreading in the population. However, after the peak of infected individuals in the hospitals is reached, the recovery becomes stable. During this period, majority of infected individuals in all of the three components of community, hospitals, and funeral decreases and clears from the population. However, after a period of about 75 days of the Ebola epidemic, the disease clears from the population.

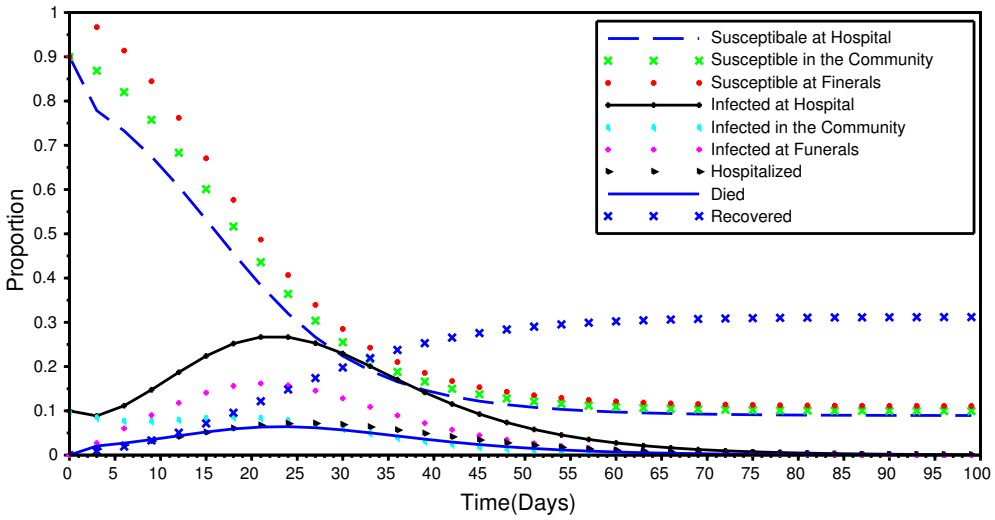

Figure 4.9: Simulation of the extended model. Parameter values similar to those used in Figure 4.2.

When compared to Figure 4.2, results from the Figure 4.9 show that the equilibrium point can occur earlier than when the basic model is used (the equilibrium point occurred on about the 120^{th} day).

5. Discussion and Conclusion

In this project, we revisited the model of Camacho et al. (2014) for Ebola outbreak in Democratic Republic of Congo (1995). We then extended this model and divided the susceptible population into three components related to community, hospital, and funeral settings where transmission occurs. The models were analysed and numerically simulated to investigate various aspects related to prevention and control.

The analysis presented in this project shows that the basic reproduction number, R_0, is the sum of R_0 for the three infectious compartments. Using the estimated parameters published in (Camacho et al., 2014), we have calculated R_0. The basic reproduction number, R_0 of the basic model is similar to that presented by (Legrand et al., 2007). The only difference appears in the flow compartment of the model where we have considered the buried individuals in compartment B. Numerical simulations confirmed that when $R_0 < 1$ the disease dies out from the population and spreads whenever $R_0 > 1$.

Behaviour change resulting from an epidemic can help slow down or even stop the Ebola transmission. Moreover, cultural perception in response to the disease enable people to change behaviour in ways that will help to control the disease outbreak. Local information campaign to change funeral practices and other behaviours seem to play an important role in decreasing the Ebola prevalence in Liberia (Funk et al., 2014). Adequate financial support and manpower can greatly help to defeat Ebola. Furthermore, improving support services like providing enough information and mobilization of the population about the disease dangers has helped to eradicate the disease in certain regions of the world. In these particular regions, the major control efforts focus on cultural, religious and social events. These events are targeted owing to the large gatherings (Darius Mans, 2015). Without including social, cultural and behavioural responses to the Ebola epidemic, the models may overestimate outbreak size (Funk et al., 2014). The reduction of infected humans is due to death or recovery. This indicates that the disease can be controlled through behaviour change. The reduction may occur when people of a certain community know how Ebola is transmitted, leading to the fight against the disease. In addition, churches, traditional healers and other secret societies need to support and help people to change their behaviours.

During this simulation, we assume that the Ebola treatments exist, and are only available at hospitals. This implies that the patients who are under treatment at hospitals are more likely to survive than to die from the Ebola disease (treatments reduce the probability of dying from the disease). If the treatment exists, it could have benefit beyond saving the lives of the patients, and the hospitals might produce survivors instead of dead bodies (Hayden and Reardon, 2014). After simulating the model for different efficacy values, we observed that as long as the treatment is efficient, the infected compartment sizes decrease. We expect that the recovered cases increase in size but the size of dead population reduces due to the effect of treatment.

Findings indicated that, if the vaccines were available for healthcare workers alone, a positive effect would be obtained on preventing further transmission. Vaccination of healthcare workers makes them immune; that is, they cannot be infected by any Ebola patients so that no further transmission from patients to healthcare workers can occur. In such case, the spread of the disease in the community is reduced.

Numerical simulations also suggested that, using the extended model by splitting susceptible compartment leads to disease clearance from the population within a short time. This has been observed based on the time at which the Disease-Free Equilibrium (DFE) point occurred. Our findings also showed that the DFE point occurs within a short period from the onset of the disease in the population in the

extended model compared to the time in the basic model. In addition, the type reproduction number T, of the extend model determine the minimum number of individuals to be vaccinated in order to prevent the disease. We observed that the minimum proportion of the population need to be $\left(1-\frac{1}{T_H}\right)$, $\left(1-\frac{1}{T_C}\right)$ and $\left(1-\frac{1}{T_F}\right)$ at hospital, in the community and during funerals respectively; where T_H, T_C, and T_F are type reproduction numbers in the respective types.

Even if there is currently no treatment or vaccines for Ebola, our extended model indicates that, at the treatment and vaccination period, the disease would be much shorter and evaluated quicker. This would contribute much in saving peoples' lives. Isolation is also one of the interventions currently applied in West Africa. However, we have not looked at isolation as an intervention to control the Ebola outbreak in this project. Several studies (Khan et al., 2015; Mamo and Koya, 2015; Legrand et al., 2007) have recommended isolation as an intervention.

The dynamics of the results in the two scenarios show that, Ebola is an epidemic disease. Control of this disease should be immediate as many individuals would die within a few days. This work has highlighted the importance of mathematical models in the deriving conditions and knowledge for Ebola prevention and control. This work and the results generated from the numerical simulations are essential for decision making and for designing better ways of controlling Ebola.

As part of future work, we suggest that simulations of intervention scenarios targeting hospitals, community, and funerals be performed to identify target groups for effective control. For further improvement of the model, we recommend the analytical analysis of the impact of intervention strategies for the basic model. We also recommend the analysis of the extended model, including the calculation of the threshold parameter R_0. Furthermore, as an extension of this work, time-dependent strategies should be developed and advised to public health authorities in order to control the disease.

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