A Mathematical Model for Controlling the Spread of Ebola Virus Disease in Nigeria

Abdulrahman. Ndanusa, Sirajo. Abdulrahman, and Abdulrazaq. Abdulmalik

***Abstract*—**In this research work, we develop and analyse a deterministic model for controlling the spread of Ebola Virus Disease (EVD) in a population with vital dynamics (birth and death rates are not equal), incorporating quarantining of infectious individuals which is influenced by availability of isolation centres and surveillance coverage We also considered improved personal hygiene of the susceptible population influenced by public enlightenment campaign. Numerical simulations showed that improved personal hygiene and quarantining of infectious individuals are enough to control the spread of EVD, with improved personal hygiene being the more effective and efficient of the two control parameters

***Keywords*—**Effective reproduction number, Endemic, Quarantine, Vital dynamics

I. Introduction

EBOLA virus disease (EVD) (formerly known as Ebola haemorrhagic fever), named after the river in Democratic Republic of Congo (DRC, formerly Zaire) where it was initially discovered in 1976, is avirulent filovirus that is known to affect humans and primates. The virus is most commonly spread via personal contact, and it has an incubation period of two to twenty – one days. It takes approximately eight hours for the virus to replicate, and can occur several times before the onset of symptoms. "Hundreds to thousands of new virus particles are then released during periods of hours to a few days, before the cell dies." [1]. Symptoms that occur within a few days after transmission include, high fever, headache, muscle aches, stomach pain, fatigue, diarrhea sore throat, hiccups, rash, red and itchy eyes, vomiting blood, bloody diarrhea [2]. The death rate of Ebola is somewhere between 50% to 90%. Until now, there is no specific cure or vaccine for Ebola but, efforts are on-going to find a viable treatment.

The first known occurrence of Ebola was in 1976 in almost simultaneous outbreaks in the Democratic Republic of the Congo (DRC) and Sudan, each escorted by fatality rate beyond 50%. The disease then disappeared after 1979 and did not re-appear again until 1994 [3]. Ever since, outbreaks have been occurring with increasing frequency.The most horrible outbreak of Ebola to date is currently occurring in West Africa, and it's been a long affair that has infected well over 24000 and killed more than 10000 as at present. Countries affected include primarily, Guinea, Liberia and Sierra Leone. Related to this extensive outbreak Ebola has been imported into Nigeria, Mali, Senegal, Spain, UK and the USA.

The present outbreak of EVD in West Africa happens to be the most severe in recorded history; hence, the need to explore the dynamics of the disease through mathematical modeling, in order to control further outbreak of the disease in Nigeria. A great many mathematicians have developed mathematical models to better improve our understanding of the dynamics and spread of EVD in order to curb its prevalence and stem the incessant outbreaks of the virus[4] - [8].The research aims to analyze the effectiveness of quarantine and improved personal hygiene as control measures.

Abdulrahman. Ndanusa is with the Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria (corresponding author's phone: + 2348023736139; e-mail: as.ndanusa@futminna.edu.ng).

Sirajo. Abdulrahman, is with the Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria (e-mail: sirajo.abdul@futminna.edu.ng).

Abdulrazaq. Abdulmalik, was with the Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria.

II. Model Formulation

The total population (N) is divided into four (4) classes of Susceptible(S), Latent(L), Infectious(I) and Recovered(R) individuals. The model parameters are define in Table I.

TABLE I
MODEL PARAMETERS

Parameter	Description
π	recruitment rate
μ	death removal rate
β	effective contact rate with infectious individuals
τ_1	recovery rate of infected individuals due to treatment
τ_2	recovery rate of infectious individuals due to treatment
δ	death rate due to disease
γ	progression rate of infected individuals to infectious individuals
q	number of quarantined individuals
α	surveillance coverage
v	availability of isolation centres
ε	enhanced personal hygiene due to public enlightenment
ϕ	rate of public enlightenment

The corresponding mathematical equations can be described by a system of ordinary differential equations as follows:

$$\frac{dS}{dt} = \pi - \frac{\beta(1-q\alpha v)(1-\varepsilon\phi)I}{N}S - \mu S \qquad (1)$$

$$\frac{dL}{dt} = \pi - \frac{\beta(1-q\alpha v)(1-\varepsilon\phi)I}{N}S - k_1 L \quad (2)$$

$$\frac{dI}{dt} = \gamma L - k_2 I \quad (3)$$

$$\frac{dI}{dt} = \tau_1 L + \tau_2 I - \mu R \quad (4)$$

where,

$$k_1 = (\tau_1 + \gamma + \mu) \quad (5)$$

$$k_2 = (\tau_2 + \delta + \mu) \quad (6)$$

III. Analysis of Model

A. Disease–Free Equilibrium

At equilibrium, $(1)-(4)$ are set to equal zero. That is,

$$\frac{dS}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \quad (7)$$

We define $(S, L, I, R) = (S^0, L^0, I^0, R^0)$ in $(1)-(4)$. Consequently,

$$I^0 = \frac{\gamma L^0}{k_2} \quad (8)$$

$$R^0 = L^0\left[\frac{k_2\tau_1 + \gamma\tau_2}{\mu k_2}\right] \quad (9)$$

$$S^0 = \frac{\pi N^0}{\beta(1-q\alpha v)(1-\varepsilon\phi)I^0 + \pi N^0} \quad (10)$$

$$L^0 = 0 \quad (11)$$

Substituting equation (11) into $(8)-(10)$ respectively, we obtain

$$(S^0, L^0, I^0, R^0) = \left(\frac{\pi}{\mu}, 0, 0, 0\right) \quad (12)$$

Equation (12) is the Disease- FreeEquilibrium (DFE).

B. Endemic Equilibrium

We define $(S, L, I, R) = (S^{**}, L^{**}, I^{**}, R^{**})$ and set $(1)-(4)$ to equal zero respectively. Thus,

$$S^{**} = \frac{k_1 k_2 N^{**}}{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)} \quad (13)$$

$$L^{**} = \frac{\beta\gamma\pi(1-q\alpha v)(1-\varepsilon\phi) + \mu k_1 k_2 N^{**}}{\beta\gamma k_1(1-q\alpha v)(1-\varepsilon\phi)} \quad (14)$$

$$I^{**} = \frac{\beta\gamma\pi(1-q\alpha v)(1-\varepsilon\phi) + \mu k_1 k_2 N^{**}}{\beta k_1 k_2(1-q\alpha v)(1-\varepsilon\phi)} \quad (15)$$

$$R^{**} = \frac{1}{\mu}\left[\frac{(\beta\gamma\pi(1-q\alpha v)(1-\varepsilon\phi) + \mu k_1 k_2 N^{**})(\tau_1 k_2 + \tau_2\gamma)}{\beta\gamma k_1 k_2(1-q\alpha v)(1-\varepsilon\phi)}\right] \quad (16)$$

Therefore, at endemic equilibrium, $(S, L, I, R) = (S^{**}, L^{**}, I^{**}, R^{**})$, given by (13) – (16).

C. Effective Reproduction Number R_c of DFE

To derive the effective reproduction number, R_c of the DFE we employ the next generation operator technique described by [10], and which was subsequently analyzed by [11] thus:

$$R_c = \rho(K) \quad (17)$$

where $\rho(K)$ denotes the spectral radius of the next generation matrix K. The matrix K is defined by

$$K = FV^{-1} \quad (18)$$

Thus,

$$FV^{-1} = \begin{bmatrix} \frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)S^0}{k_1 k_2 N^0} & \frac{\beta(1-q\alpha v)(1-\varepsilon\phi)S^0}{k_2 N^0} \\ 0 & 0 \end{bmatrix} \quad (19)$$

and

$$\rho(FV^{-1}) = |FV^{-1} - \lambda I| = 0 \quad (20)$$

Hence,

$$R_c = \frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)S^0}{k_1 k_2 N^0} \quad (21)$$

Since $S^0 = N^0$ at DFE,

$$R_c = \frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)}{k_1 k_2} \quad (22)$$

D. Local Stability

To establish that the DFE is locally stable, we show that our effective reproduction number, $R_c < 1$. Using the Jacobian Matrix to linearize $(1)-(4)$ we have

$$J = \begin{vmatrix} -\mu & 0 & -\beta(1-q\alpha v)(1-\varepsilon\phi) & 0 \\ 0 & -k_1 & \beta(1-q\alpha v)(1-\varepsilon\phi) & 0 \\ 0 & \gamma & -k_2 & 0 \\ 0 & \tau_1 & \tau_2 & -\mu \end{vmatrix} \quad (23)$$

On reducing (23) to row-echelon form we obtain

$$\begin{vmatrix} -\mu & 0 & -\beta(1-q\alpha v)(1-\varepsilon\phi) & 0 \\ 0 & -k_1 & \beta(1-q\alpha v)(1-\varepsilon\phi) & 0 \\ 0 & 0 & \frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi) - k_1 k_2}{k_1} & 0 \\ 0 & 0 & 0 & -\mu \end{vmatrix} \quad (24)$$

The eigenvalues of (24) are found to be

$$\lambda_1 = -\mu \quad (25)$$

$$\lambda_2 = -k_1 \quad (26)$$

$$\lambda_3 = \left(\frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi) - k_1 k_2}{k_1}\right) \quad (27)$$

$$\lambda_4 = -\mu \quad (28)$$

Equations $(25)-(28)$ implies,

$$\lambda_1 < 0 \quad (29)$$

$$\lambda_2 < 0 \quad (30)$$

$$\lambda_3 < 0 \text{ iff} \left(\frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi) - k_1 k_2}{k_1}\right) < 0 \quad (31)$$

$$\lambda_4 < 0 \quad (32)$$

But,

$$[\beta\gamma(1-q\alpha v)(1-\varepsilon\phi) - k_1 k_2] < 0 \quad (33)$$

Since,

$$\beta\gamma(1-q\alpha v)(1-\varepsilon\phi) < k_1 k_2 \quad (34)$$

Dividing through (34) by $k_1 k_2$, we have

$$\frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)}{k_1 k_2} < \frac{k_1 k_2}{k_1 k_2} < 1 \quad (35)$$

From (35), we conclude that $R_c < 1$. Hence, the DFE is locally stable.

E. Global Asymptotic Stability of DFE

By employing the Lyapunov principle the DFE is globally asymptotically stable if $P < 0$ or $P' \leq 0$; where,

$$P = \gamma L + k_1 I \quad (36)$$

$$P' = \gamma L' + k_1 I' \quad (37)$$

That is,

$$P' = \gamma\left(\frac{\beta(1-q\alpha v)(1-\varepsilon\phi)}{N}S - k_1 L\right) + k_1(\gamma L - k_2 I) \quad (38)$$

$$P' = k_1 k_2 I\left(\frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)}{N k_1 k_2}S - 1\right) \quad (39)$$

Since $\frac{S}{N} \leq \frac{S^0}{N^0}$,

$$P' \leq k_1 k_2 I\left(\frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)}{N^0 k_1 k_2}S^0 - 1\right) \quad (40)$$

But at DFE $S^0 = N^0$. Thus,

$$P' \leq k_1 k_2 I\left(\frac{\beta\gamma(1-q\alpha v)(1-\varepsilon\phi)}{k_1 k_2} - 1\right) \quad (41)$$

Therefore,

$$P' \leq k_1 k_2 I(R_c - 1) \quad (42)$$

Thus, $P' = 0$ when $R_c = 1$ and $P' \leq 0$ when $R_c < 1$; thus, by Lyapunov principle, the DFE is globally asymptotically stable.

IV. Numerical Simulation

For the purpose of model validation, in order to ensure that the model is in agreement with reality, numerical simulation is undertaken using the data provided in Table I, and varying values of the control parameters, q and ε. The results are displayed in Fig. 1 – Fig. 8.

Table II
Parameter Values

Symbol	Value
π	9863 (day^{-1})
μ	0.00005479 (day^{-1})
β	0.9 (day^{-1})
τ_1	0.045 (day^{-1})
τ_2	0.15 (day^{-1})
δ	0.025 (day^{-1})
γ	0.083 (day^{-1})
α	0.75
v	0.65
ϕ	0.90

.

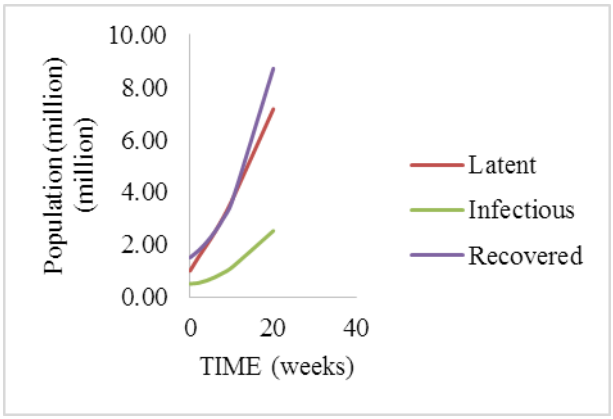

Fig.1 Latent, Infectious and Recovered classes when $q = 0, \varepsilon = 0$

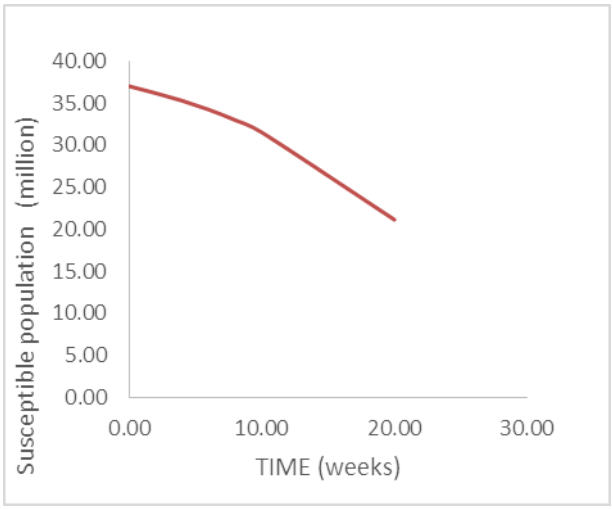

Fig. 2 Susceptible class when $q = 0, \varepsilon = 0$

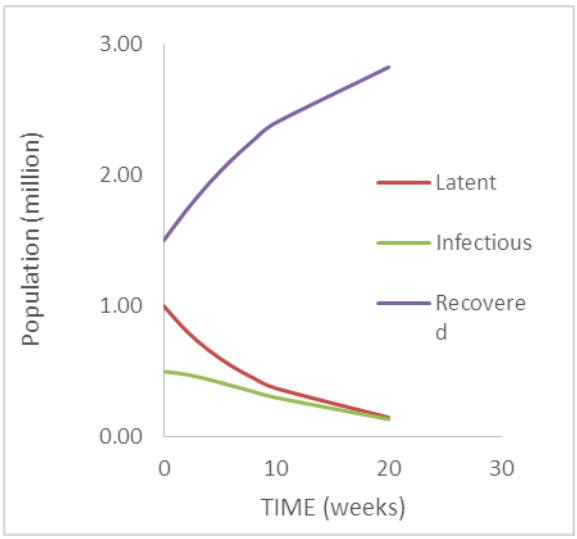

Fig. 3 Latent, Infectious and Recovered classes when $q = 1, \varepsilon = 1$

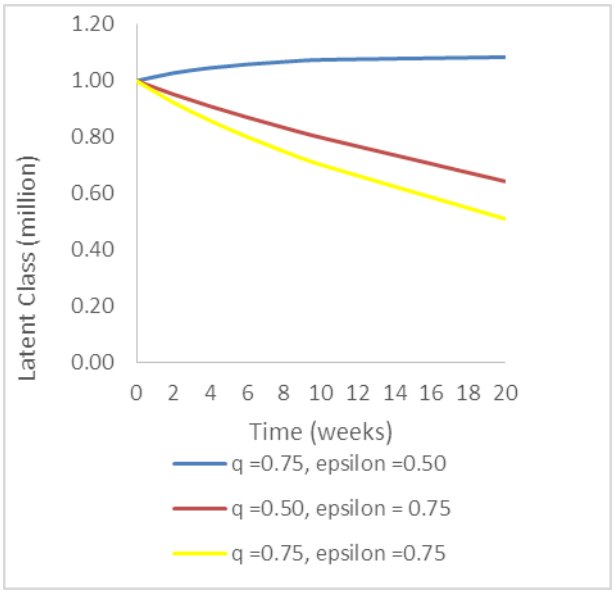

Fig. 4 Latently infected class at varying control parameters

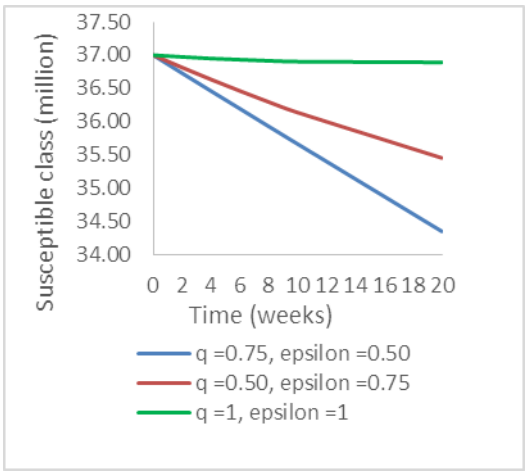

Fig. 5Susceptible class at varying control parameters

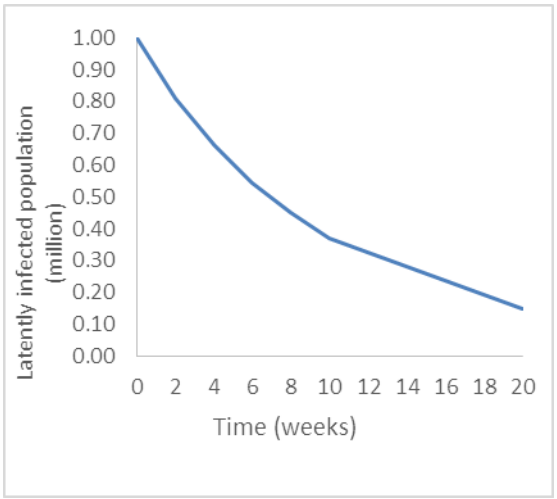

Fig. 6 Latently Infected class when $q = 1, \varepsilon = 1$

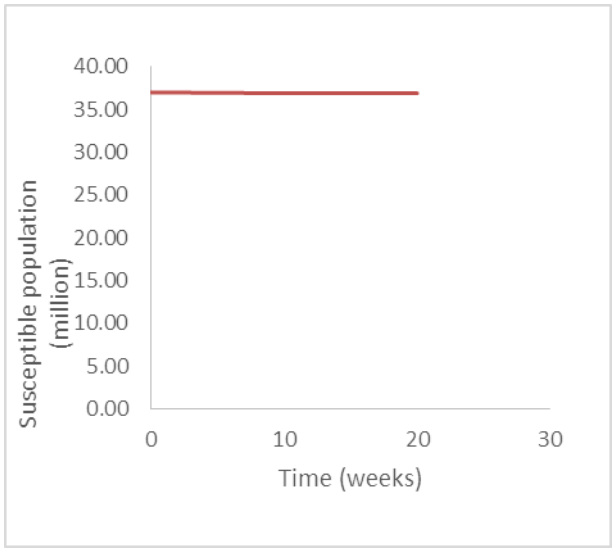

Fig. 7Susceptible class when $q = 1, \varepsilon = 1$

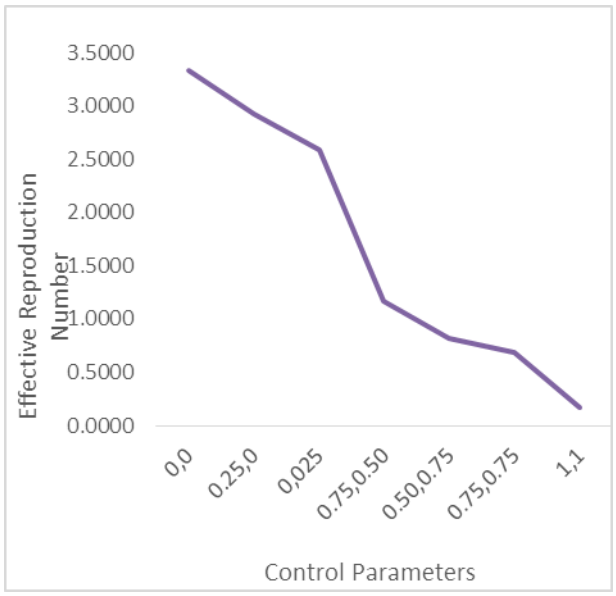

Fig. 8 Effective Reproduction number with increasing control parameters.

V. Discussion Of Results

Fig. 1 reveals the rate at which the population becomes latently infected is fast increasing when there is no control parameter in place. In Fig. 2, the susceptible population reduces at a very high rate when no control parameters are in place. Fig. 3 shows the latent, infectious and recovered classes when both control parameters are implemented at full scale (i.e. 100%). The rate of infection can be seen to have dropped drastically. In Fig. 4 the effectiveness of improved personal hygiene over quarantine is clearly exhibited. Fig. 5 exhibits a gradual drop in the rate at which susceptible individuals becomes infected. When quarantine and improved personal hygiene are implemented at full scale (i.e. 100%) in Fig. 6, the disease is put under control and dies out soon. In Fig. 7 the growth of the susceptible population is uniform when the proportion of quarantined infectious individuals and the proportion of susceptible population that improved their

personal hygiene are both 100%. Fig. 8 reveals a steady drop in the effective reproduction number of the disease which proves the effectiveness of the control parameters in place.

VI. Conclusion

Given the results obtained from the analysis of the model, we observed that a timely implementation of the control parameters would go a long way in stemming the spread of the disease in a population that has been ravaged by EVD. While this is a good thing, we must emphasize the fact that a timely identification of an outbreak remains of paramount importance in controlling the spread of the disease.

References

[1] Healthlink USA, http://www.healthlinkusa.com/101ent), March, 2015.

[2] Centre for Disease control, (http://www.cdc.gov/ncidod/ dvrd /mnpages/dispages/ebola.htm), March, 2015.

[3] Public Health England. (https://www.gov.uk/government/publications /ebola-origins- reservoirs-transmission-and-guidelines/ebola-origins-reservoirs-transmission-guidelines), March, 2015.

[4] J. Astacio, D. Briere, M. Guillen, J. Martinez, F. Rodriguez, N. Valenzuela-Campos, "Mathematical models to study the outbreaks of Ebola," *Biometrics Unit Technical Report*, Numebr BU-1365-M, Cornell University, 1996.

[5] G. Chowell, N. W. Hengartner, C. Castillo-Chavez, P. W. Fenimore, and J. M. Hyman, "The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda," *J. Theor. Biol.*, vol 229, no. 1, pp. 119-126, Jul. 2004.

[6] C. Rizkalla, F. Blanco-Silva, and S. Gruver, "Modeling the impact of Ebola and bushmeat hunting on western lowland gorillas," *EcoHealth J. Consortium*, vol 4, pp. 151-155, DOI: 10.1007/s10393-007-0096-2, Jun. 2007.

[7] Z. Yarus, "A Mathematical look at the Ebola Virus," http://home2.fvcc.edu/~dhicketh/DiffEqns/Spring2012Projects/Zach%20Yarus%20-Final%20Project/Final%20Diffy%20Q%20project.pdf, March, 2015.

[8] C. L. Althaus, "Estimating the reproduction number of Ebola virus (EBOV) during the 2014 outbreak in West Africa," http://arxiv.org/abs/1408.3505, Sep. 2014.

[9] C. L. Althaus, N. Low, E. O. Musa, F. Shuaib, S. Gsteiger, (2015) "Ebola virus disease outbreak in Nigeria: transmission dynamics and rapid control. *PeerJ PrePrints* 3:e569v3 http://dx.doi.org/10.7287/peerj.preprints.569v3, 2015.

[10] O. Diekmann, and J. A. P. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*. New York: Wiley, 2000.

[11] P. Van den Driessche, and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Math. Biosci*., vol. 180, pp. 29-48, Nov-Dec 2002.