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On Some Compartmental Models for Ebola Disease

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Abstract

In this paper, we consider an epidemic model of Ebola disease which is deadly in its transmission.

Local stability analysis of the model equilibria was investigated. We computed the basic reproduction number R_0 using the next generation method. The threshold parameter R_0 was found to be dependent on several hosts of model parameters in determining the stability of an invading epidemic into the population. We have numerically described the model trajectories using Matlab.

KEYWORDS: Basic Reproduction number, Ebola virus, Next-generation matrix, Local stability analysis.

1. INTRODUCTION

Ebola, one of the deadly diseases for humans, emanates from the family of the virus filoviridae [1]. There are five recognized subtype of the Ebola virus [9]. Four of these strains; Ebola-Ta, Ebola-Bundibugyo, Ebola-Zaire and Ebola-Sudan have all caused disease in humans. The only stain that is known of ever causing disease in non-human primates is Ebola-Reston. The virus can be spread when a direct contact is made with blood or body fluids of infected persons, objects that have been contaminated with the virus and non-human primates [4]. The history of Ebola traces back to 1976, in Sudan and Zaire where it was first found [13]. Ebola has high mortality rate, and ever since its outbreak in west Africa , the streams of the infection has poured forth into all parts of west Africa; Gabon, Liberia, Guinea, Uganda, Democratic Republic of Congo , Nigeria etc [15]. The rapid instreaming of the infection into the neighboring countries of West Africa has been alarming and a major public health concern for World Health Organization (WHO) [2].

(WHO, [5]), reported approximately 4700 cases of Ebola deaths in the last quarter of 2014. This is just an epitome of the reported death cases emanating from Ebola virus infection in that year. Evidently, the 2014 Ebola virus outbreak in West Africa is recognized as the largest outbreak of the genus Ebolavirus to date [14]. Infection from the virus may produce headache, loss of appetite, body weakness, muscle ache, fever, sore throat and fatigue at the early stages

of the infection [11]. The development of the virus takes approximately 1 to 2 weeks, in which the infective shows no symptoms of a disease. The last stage of the infection is characterized by profuse vomiting, diarrhea, chest pain, severe blood lost and coagulation abnormalities. Eventually, the infection leads to liver and kidney failure as well as internal bleeding which usually results in death due to the blood loss [7]. Recently, Ebola vaccine has been developed by researchers and its trial stages of testing has proved to be hundred percent effective. However, recovery from Ebola infection depends on the person's immune system and probably good health care for the infected person [23].

Ever since its outbreak, related works of the Ebola virus disease have been proposed and studied by modelers, and the analysis of these models has been of great importance to researchers and health bodies at large and has provided insight to these bodies in the area of vaccine development and control intervention strategy [6]. Amira and Delffin [8], studied a simple mathematical model that describes the 2014 Ebola outbreak in Liberia. They used numerical simulations and available data provided by the World Health Organization (WHO) to validate their model. They again developed a new model including vaccination of individuals, and discuss different cases of vaccination in order to predict the effect of vaccination on the infected individual over time. Optimal control was then applied to study the impact of vaccination on the spread of the Ebola virus. Althaus [3], studied EVD epidemic using SEIR model and the model was then fitted to the most recent data of deaths cases provided in Guinea, Sierra Leone and Liberia. Arreola et al., [12], analyzed an Ebola deterministic and stochastic model of SIR compartments and used it to study the effect of quarantine on the infective. Nishiura and chowell [17], clarified and illustrated different hypothetical concepts of the quality of being infectious in order to compare the infectiousness of several diseases which are spread through contacts, including EVD. Pandey et al. [16], introduces a stochastic model of Ebola and used the general community, hospital and funerals as their case study, calibrated to incidence data from Liberia. Lekone and Finkenstadt [20], formulated a discrete time SEIR stochastic model for infectiou disease and used it to estimate parameters from mortality time series for 1995 Ebola outbreak of Democratic republic of Congo. Weitz and Dushoff [21], examined an important component of Ebola dynamics by studying the transmission of Ebola from the dead to the living. Atangana and Gonfo [18], constructed a model that describes the dynamics of the spread of Ebola hemorrhagic fever using the methods of classical and beta derivative.

Chowell et al [19]., estimated an important threshold parameter called the basic reproduction ratio (R_0) by using data from the outbreak of 1995 Congo and 2000 Uganda Ebola for his model. Ebihara et al [24].,provided a better tool for comprehending the important processes in pathogenesis and provides a better way for evaluating prophylactic and post exposure intervention for testing in non-human primate by using mouse-adapted Ebola virus for their lethal Syrian monster model of Ebola hemorrhagic fever. McElroy [25], provided a better insight of Ebola hemorrhagic fever path-physiology and a starting point for researching new potential targets for therapeutic interventions by using a series of multiplex assays to measure

the concentrations of 55 biomarkers. Pyankov et al [22]., describe the evaluation of an Ebola virus vaccine candidate based on kunjin replicon virus –like particles (KUN VLPs) encoding EBOV glycoprotein with a D637L mutation (GP/D637L) in non-human primates.

In this research article, using the mathematical formulation of Ebola disease dynamics by Khan et al. [10], we carry out the local qualitative stability analysis of the model which was not considered in their paper. The dynamics of the Ebola model is shown in figure 1.

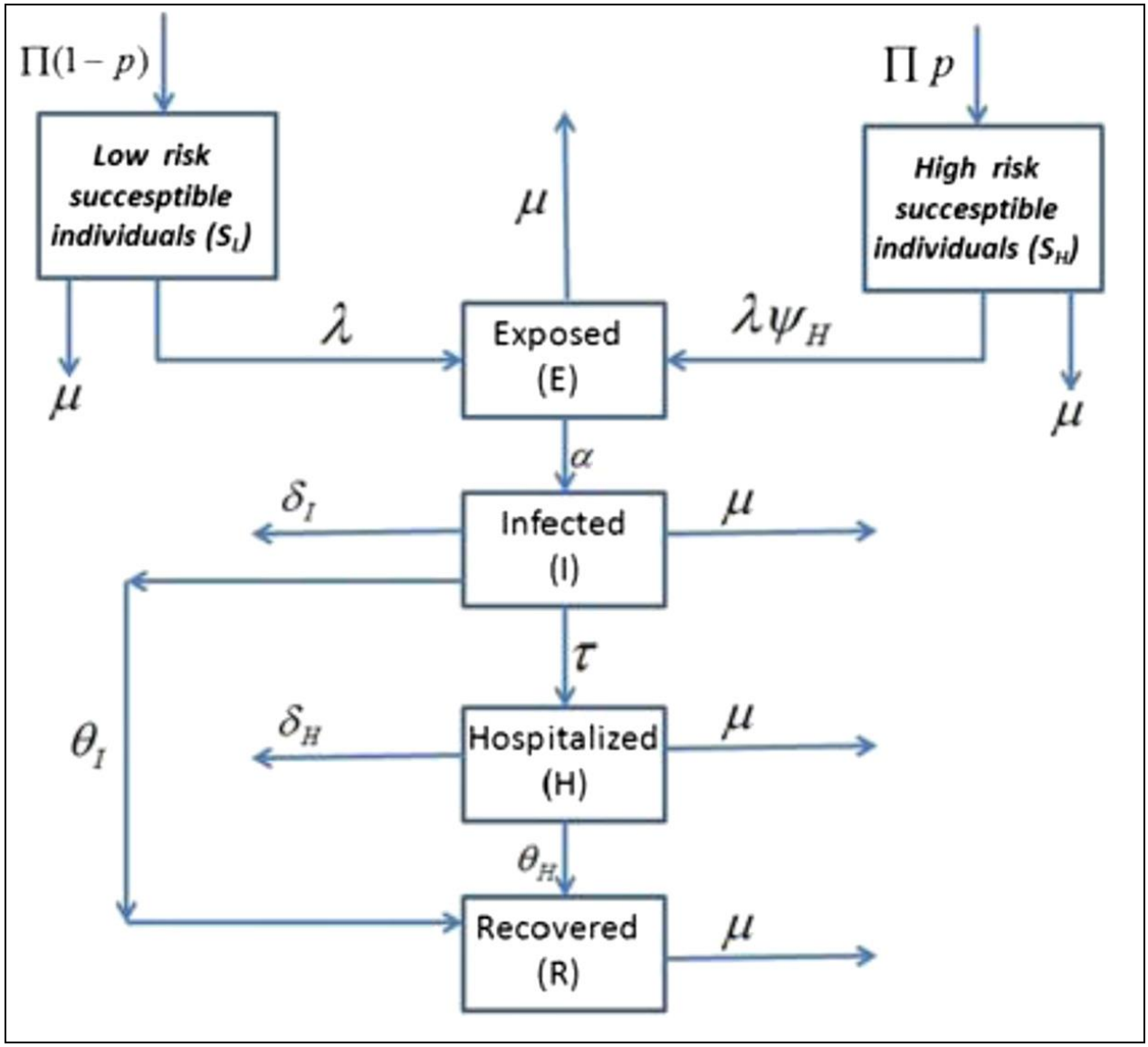

Figure 1: Shows the flowchart of the dynamics of the Ebola model by Khan et al. [10].

2. The Model formulation

The dynamics of the model as described by Khan et al. [10] is given by the system of the differential equations:

$$\frac{dS_L}{dt} = \pi(1-p) - \frac{\beta I S_L}{N} - \frac{n\beta H S_L}{N} - \mu S_L \quad (1)$$

$$\frac{dS_H}{dt} = \pi p - \frac{\beta I \psi_H S_H}{N} - \frac{nBH\psi_H S_H}{N} - \mu S_H \quad (2)$$

$$\frac{dE}{dt} = \frac{\beta I S_L}{N} + \frac{n\beta H S_L}{N} + \frac{\beta I \psi_H S_H}{N} + \frac{n\beta H \psi_H S_H}{N} - (\alpha+\mu)E \quad (3)$$

$$\frac{dI}{dt} = \alpha E - (\tau + \theta_I + \delta_I + \mu)I \quad (4)$$

$$\frac{dH}{dt} = \tau I - (\theta_H + \delta_H + \mu)H \quad (5)$$

$$\frac{dR}{dt} = \theta_I I + \theta_H H - \mu R \quad (6)$$

The total population, N for the model is given by

$$N(t) = S_L(t) + S_H(t) + E(t) + I(t) + H(t) + R(t) \quad (7)$$

where $S_L(0) = S_{L0} \geq 0$, $S_H(0) = S_{H0} \geq 0$, $E(0) = E_0 \geq 0$,
$I(0) = I_0 \geq 0$, $H(0) = H_0 \geq 0$, $R(0) = R_0 \geq 0$

From equation (7), let $R(t) = N(t) - S_L(t) - S_H(t) - E(t) - I(t) - H(t)$.

Therefore the equivalent system of differential equations becomes

$$\frac{dS_L}{dt} = \pi(1-p) - \frac{\beta I S_L}{N} - \frac{n\beta H S_L}{N} - \mu S_L \quad (8)$$

$$\frac{dS_H}{dt} = \pi p - \frac{\beta I \psi_H S_H}{N} - \frac{nBH\psi_H S_H}{N} - \mu S_H \quad (9)$$

$$\frac{dE}{dt} = \frac{\beta I S_L}{N} + \frac{n\beta H S_L}{N} + \frac{\beta I \psi_H S_H}{N} + \frac{n\beta H \psi_H S_H}{N} - (\alpha+\mu)E \quad (10)$$

$$\frac{dI}{dt} = \alpha E - (\tau + \theta_I + \delta_I + \mu)I \quad (11)$$

$$\frac{dH}{dt} = \tau I - (\theta_H + \delta_H + \mu)H \quad (12)$$

With $R(t) = N(t) - S_L(t) - S_H(t) - E(t) - I(t) - H(t)$.

2.1 Model Equilibria

The equilibrium points of the model are the disease –free equilibrium point

$E_0 = (S_{L0}, S_{H0}, E_0, I_0 H_0,) = \left(\frac{\pi(1-p)}{\mu}, \frac{\pi p}{\mu}, 0,0,0\right)$ and the endemic equilibrium point

$$EE = \left(\frac{N\pi(1-p)}{(\beta I + n\beta H + N\mu)}, \frac{N\pi p}{(\beta I\psi_H + n\beta H\psi_H + N\mu)}, \frac{(\tau+\theta_I+\delta_I+\mu)I}{\alpha}, \frac{\alpha E}{(\tau+\theta_I+\delta_I+\mu)}, \frac{\tau I}{(\theta_H+\delta_H+\mu)}\right)$$

2.2 The Basic reproduction Ratio

In a population of only susceptible individuals, the basic reproduction ratio is defined as the infection from a single infected individual introduced into the population. Our model calculation would be based on the approach of Diekmann and Heesterbeek ([26], [27]).

The infective compartments of the model are

$$\frac{dE}{dt} = \frac{\beta I S_L}{N} + \frac{n\beta H S_L}{N} + \frac{\beta I\psi_H S_H}{N} + \frac{n\beta H\psi_H S_H}{N} - (\alpha + \mu)E \quad (13)$$

$$\frac{dI}{dt} = \alpha E - (\tau + \theta_I + \delta_I + \mu)I \quad (14)$$

$$\frac{dH}{dt} = \tau I - (\theta_H + \delta_H + \mu)H \quad (15)$$

Hence $\mathcal{F} = \begin{bmatrix} \frac{\beta I S_L}{N} + \frac{n\beta H S_L}{N} + \frac{\beta I\psi_H S_H}{N} + \frac{n\beta H\psi_H S_H}{N} \\ 0 \\ 0 \end{bmatrix}$ and $\mathcal{V} = \begin{bmatrix} (\alpha + \mu)E \\ -\alpha E + (\tau + \theta_I + \delta_I + \mu)I \\ -\tau I + (\theta_H + \delta_H + \mu)H \end{bmatrix}$.

Evaluating the Jacobian of F and V at $(S_{L0}, S_{H0}, E_0, I_0 H_0,) = \left(\frac{\pi(1-p)}{\mu}, \frac{\pi p}{\mu}, 0,0,0\right)$ gives

$$F = \begin{bmatrix} 0 & \frac{\beta\pi(1-p)+\beta\pi p\psi_H}{\mu N} & \frac{n\beta\pi(1-p)+n\beta\pi p\psi_H}{\mu N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and}$$

$$V = \begin{bmatrix} (\alpha + \mu) & 0 & 0 \\ -\alpha & (\tau + \theta_I + \delta_I + \mu) & 0 \\ 0 & -\tau & (\theta_H + \delta_H + \mu) \end{bmatrix}$$

But since $N = S_{L0} + S_{H0} = \frac{\pi}{\mu}$ at $I_0 = 0,\ H_0 = 0,\ E_0 = 0,$ F becomes

$$F = \begin{bmatrix} 0 & \beta(1-p)+\beta p\psi_H & n\beta(1-p)+n\beta p\psi_H \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

But $V^{-1} = \begin{bmatrix} \frac{1}{(\alpha+\mu)} & 0 & 0 \\ \frac{\alpha}{(\alpha+\mu)(\tau+\theta_I+\delta_I+\mu)} & \frac{1}{(\tau+\theta_I+\delta_I+\mu)} & 0 \\ \frac{\alpha\tau}{(\alpha+\mu)(\tau+\theta_I+\delta_I+\mu(\theta_H+\delta_H+\mu))} & \frac{\tau}{(\tau+\theta_I+\delta_I+\mu)(\theta_H+\delta_H+\mu)} & \frac{1}{(\theta_H+\delta_H+\mu)} \end{bmatrix}$

$$FV^{-1} = \begin{bmatrix} \frac{\alpha\beta(1-p)+\alpha\beta p\psi_H}{(\alpha+\mu)(\tau+\theta_I+\delta_I+\mu)} + \frac{\alpha\tau n\beta(1-p)+\alpha n\tau\beta p\psi_H}{(\alpha+\mu)(\tau+\theta_I+\delta_I+\mu)(\theta_H+\delta_H+\mu)} & k_1 & k_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

where $k_1 = \frac{\beta(1-p)+\beta p\psi_H}{(\tau+\theta_I+\delta_I+\mu)} + \frac{n\tau\beta(1-p)+n\tau\beta p\psi_H}{(\tau+\theta_I+\delta_I+\mu)(\theta_H+\delta_H+\mu)}$

$$k_2 = \frac{n\beta(1-p)+n\beta p\psi_H}{(\theta_H+\delta_H+\mu)}$$

According to Diekmann and Heesterbeek [27], the reproduction ratio is given by $R_0 = \rho(FV^{-1})$ where ρ is the spectral radius of the next generation matrix (FV^{-1}).
Hence

$$R_0 = \frac{\alpha\beta(1-p)+\alpha\beta p\psi_H}{(\alpha+\mu)(\tau+\theta_I+\delta_I+\mu)} + \frac{\alpha\tau n\beta(1-p)+n\beta\tau p\psi_H}{(\alpha+\mu)(\tau+\theta_I+\delta_I+\mu)(\theta_H+\delta_H+\mu)}$$

3. Local stability of the Disease- free Equilibrium (DFE)

The model is analyzed for its stability at the disease-free equilibrium where $E_0 = I_0 = H_0 = 0$ and the steady state has $E_0 = (S_{L0}, S_{H0}, E_0, I_0, H_0,) = \left(\frac{\pi(1-p)}{\mu}, \frac{\pi p}{\mu}, 0,0,0\right)$.

The local stability is deduced from the system (8-12) evaluated at E_0.

The Jacobian matrix of the system (8-12) is given by

$$\begin{bmatrix} \left(-\frac{\beta I}{N}-\frac{n\beta H}{N}-\mu\right) & -\frac{n\beta S_L}{N} & 0 & \frac{-\beta S_L}{N} & \frac{-n\beta S_L}{N} \\ 0 & \left(\frac{-\beta I\psi_H}{N}-\frac{n\beta H\psi_H}{N}-\mu\right) & 0 & -\frac{\beta\psi_H S_H}{N} & \frac{-n\beta\psi_H S_H}{N} \\ \left(\frac{\beta I}{N}+\frac{n\beta H}{N}\right) & \left(\frac{\beta I\psi_H}{N}+\frac{n\beta H\psi_H}{N}\right) & -(\alpha+\mu) & \left(\frac{\beta S_L}{N}+\frac{\beta\psi_H S_H}{N}\right) & \left(\frac{n\beta S_L}{N}+\frac{n\beta\psi_H S_H}{N}\right) \\ 0 & 0 & \alpha & -(\tau+\theta_I+\delta_I+\mu) & 0 \\ 0 & 0 & 0 & \tau & -(\theta_H+\delta_H+\mu) \end{bmatrix}$$

Evaluating the Jacobian matrix at the point $E_0=\left(\frac{\pi(1-p)}{\mu},\frac{\pi p}{\mu},0,0,0\right)$ gives

$$J(E_0) = \begin{bmatrix} -\mu & -n\beta(1-\rho) & 0 & -\beta(1-\rho) & -n\beta(1-p) \\ 0 & -\mu & 0 & -\beta\psi_H p & -n\beta\psi_H p \\ 0 & 0 & -(\alpha+\mu) & (\beta(1-p)+\beta\psi_H p) & (n\beta(1-p)+n\beta\pi\psi_H p) \\ 0 & 0 & \alpha & -(\tau+\theta_I+\delta_I+\mu) & 0 \\ 0 & 0 & 0 & \tau & -(\theta_H+\delta_H+\mu) \end{bmatrix}$$

We now determine the eigenvalues of $J(E_0)$.

$$\det\ |J(E_0)-\lambda I| =$$

$$\begin{vmatrix} -\mu-\lambda & -n\beta(1-\rho) & 0 & -\beta(1-\rho) & -n\beta(1-p) \\ 0 & -\mu-\lambda & 0 & -\beta\psi_H p & -n\beta\psi_H p \\ 0 & 0 & -(\alpha+\mu)-\lambda & (\beta(1-p)+\beta\psi_H p) & (n\beta(1-p)+n\beta\pi\psi_H p) \\ 0 & 0 & \alpha & -(\tau+\theta_I+\delta_I+\mu)-\lambda & 0 \\ 0 & 0 & 0 & \tau & -(\theta_H+\delta_H+\mu)-\lambda \end{vmatrix}$$

Clearly $\lambda_1=-\mu$ and $\lambda_2=-\mu$ are negative eigenvalues from the first 2×2 block matrix.

Hence, the remaining three eigenvalues can obtained from the 3×3 determinant matrix below.

$$\begin{vmatrix} -(\alpha+\mu)-\lambda & (\beta(1-p)+\beta\psi_H p) & (n\beta(1-p)+n\beta\pi\psi_H p) \\ \alpha & -(\tau+\theta_I+\delta_I+\mu)-\lambda & 0 \\ 0 & \tau & -(\theta_H+\delta_H+\mu)-\lambda \end{vmatrix}$$

We obtain the characteristics equation

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \qquad (16)$$

where

$a_0 = 1$

$a_1 = \big((\tau + \theta_I + \delta_I + \mu) + (\theta_H + \delta_H + \mu) + (\alpha + \mu)\big)$

$a_2 = \big((\alpha + \mu)(\tau + \theta_I + \delta_I + \mu) + (\alpha + \mu)(\theta_H + \delta_H + \mu)$

$+ (\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H + \mu) - \alpha(\beta(1-p) + \beta\psi_H p)\big)$

$a_3 = \big((\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H + \mu) - \alpha(\beta(1-p) + \beta\psi_H p)(\theta_H + \delta_H + \mu)$

$- \alpha\tau(n\beta(1-p) + n\beta\pi\psi_H p)\big)$

By Routh–Hurwitz stability criterion, for the characteristic equation of the cubic polynomial (16) to have a negative real part then $a_0 > 0,\ a_1a_2 > a_3 > 0$. Hence the disease-free equilibrium is locally asymptotically stable if these conditions hold, otherwise unstable.

3.1 Local stability of the Endemic Equilibrium (EE)

At the endemic equilibrium, the system of the equations (8-12) gives the steady state,

$$(S_L{}^*, S_H{}^*, E^*, I^*, H^*) = \left(\frac{N\pi(1-p)}{(\beta I + n\beta H + N\mu)}, \frac{N\pi p}{(\beta I\psi_H + n\beta H\psi_H + N\mu)}, \frac{(\tau+\theta_I+\delta_I+\mu)I}{\alpha}, \frac{\alpha E}{(\tau+\theta_I+\delta_I+\mu)}, \frac{\tau I}{(\theta_H+\delta_H+\mu)}\right)$$

Therefore the Jacobain matrix evaluated the at the endemic equilibrium is given by

$$\begin{bmatrix} \frac{-\beta k_4}{N} - \frac{n\beta k_5}{N} - \mu & \frac{-n\beta k_1}{N} & 0 & \frac{-\beta k_1}{N} & \frac{-n\beta k_1}{N} \\ 0 & \frac{-\beta\psi_H k_4}{N} - \frac{n\beta\psi_H k_5}{N} - \mu & 0 & \frac{-\beta\psi_H k_2}{N} & \frac{-n\beta\psi_H k_2}{N} \\ \frac{\beta k_4}{N} + \frac{n\beta k_5}{N} & \frac{\beta\psi_H k_4}{N} + \frac{n\beta\psi_H k_5}{N} & -(\alpha+\mu) & \frac{\beta k_1}{N} + \frac{\beta\psi_H k_2}{N} & \frac{n\beta k_1}{N} + \frac{n\beta\psi_H k_2}{N} \\ 0 & 0 & 0 & -(\tau+\theta_I+\delta_I+\mu) & 0 \\ 0 & 0 & 0 & \tau & -(\theta_H+\delta_H+\mu) \end{bmatrix}$$

Where $k_1 = \frac{N\pi(1-p)}{(\beta I + n\beta H + N\mu)}$, $k_2 = \frac{N\pi p}{(\beta I\psi_H + n\beta H\psi_H + N\mu)}$, $k_3 = \frac{(\tau+\theta_I+\delta_I+\mu)I}{\alpha}$, $k_4 = \frac{\alpha E}{(\tau+\theta_I+\delta_I+\mu)}$, $k_{5=} \frac{\tau I}{(\theta_H+\delta_H+\mu)}$

Hence $\det|J(EE) - \lambda I|$ gives

$$\begin{vmatrix} \frac{-\beta k_4}{N} - \frac{n\beta k_5}{N} - \mu - \lambda & \frac{-n\beta k_1}{N} & 0 & \frac{-\beta k_1}{N} & \frac{-n\beta k_1}{N} \\ 0 & \frac{-\beta\psi_H k_4}{N} - \frac{n\beta\psi_H k_5}{N} - \mu - \lambda & 0 & \frac{-\beta\psi_H k_2}{N} & \frac{-n\beta\psi_H k_2}{N} \\ \frac{\beta k_4}{N} + \frac{n\beta k_5}{N} & \frac{\beta\psi_H k_4}{N} + \frac{n\beta\psi_H k_5}{N} & -(\alpha+\mu) - \lambda & \frac{\beta k_1}{N} + \frac{\beta\psi_H k_2}{N} & \frac{n\beta k_1}{N} + \frac{n\beta\psi_H k_2}{N} \\ 0 & 0 & 0 & -(\tau+\theta_I+\delta_I+\mu) - \lambda & 0 \\ 0 & 0 & 0 & \tau & -(\theta_H+\delta_H+\mu) - \lambda \end{vmatrix}$$

The determinant matrix satisfy the characteristics equation

$$P(\lambda) = a_0\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0 \qquad (17)$$

where

$a_0 = 1$

$$a_1 = \left(\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right) + (\alpha+\mu) + (\tau+\theta_I+\delta_I+\mu) + (\theta_H+\delta_H+\mu) + \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\right)$$

$$\begin{aligned} a_2 = \Bigg(&\left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right) + \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\alpha+\mu) \\ &+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\tau+\theta_I+\delta_I+\mu) \\ &+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\theta_H+\delta_H+\mu) + \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha+\mu) \\ &+ \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\tau+\theta_I+\delta_I+\mu) \\ &+ \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\theta_H+\delta_H+\mu) - (\alpha+\mu)(\tau+\theta_I+\delta_I+\mu) \\ &- (\alpha+\mu)(\theta_H+\delta_H+\mu) - (\tau+\theta_I+\delta_I+\mu)(\theta_H+\delta_H+\mu)\Bigg) \end{aligned}$$

$$a_3 = \Bigg(-\left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)$$
$$- \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\tau + \theta_I + \delta_I + \mu)$$
$$- \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\theta_H + \delta_H + \mu)$$
$$- \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)$$
$$- \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)(\theta_H + \delta_H + \mu)$$
$$- \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H + \mu)$$
$$- (\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H + \mu)$$
$$+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)$$
$$+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\alpha + \mu)(\theta_H + \delta_H + \mu)$$
$$+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H + \mu)\Bigg)$$

$$a_4 = \Bigg(\left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)$$
$$+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)(\theta_H + \delta_H + \mu)$$
$$+ \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H$$
$$+ \mu) + \left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)(\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H + \mu)\Bigg)$$

$$a_5 = \Bigg(\left(\frac{\beta k_4}{N} + \frac{n\beta k_5}{N} + \mu\right)\left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)(\tau + \theta_I + \delta_I + \mu)(\theta_H + \delta_H$$
$$+ \mu)$$
$$+ \left(\frac{\beta\psi_H K_4}{N} + \frac{n\beta\psi_H K_5}{N} + \mu\right)(\alpha + \mu)(\tau + \quad \theta_I + \delta_I + \mu)(\theta_H + \delta_H$$
$$+ \mu)\Bigg)$$

By Routh–Hurwitz stability criterion [28]: if $a_i > 0,\ i = 0,1,2,3,4,5,\ a_1.a_2.a_3 > {a_3}^2 + {a_1}^2.a_4$ and
$(a_1.a_4 - a_5)(a_1.a_2.a_3 - {a_3}^2 - {a_1}^2.a_4) > a_5(a_1.a_2 - a_3)^2 + a_1.{a_5}^2$, then the endemic equilibrium is locally asymptotically stable, otherwise unstable.

4. Simulation Results

The simulated results bring insight to the dynamics of the model. Since the reproduction number depends on hosts of parameters in determining the stability of an invading epidemic into the population, we have thus described the effect of these parameters on the equilibrium level of the exposed, infected, hospitalized population.

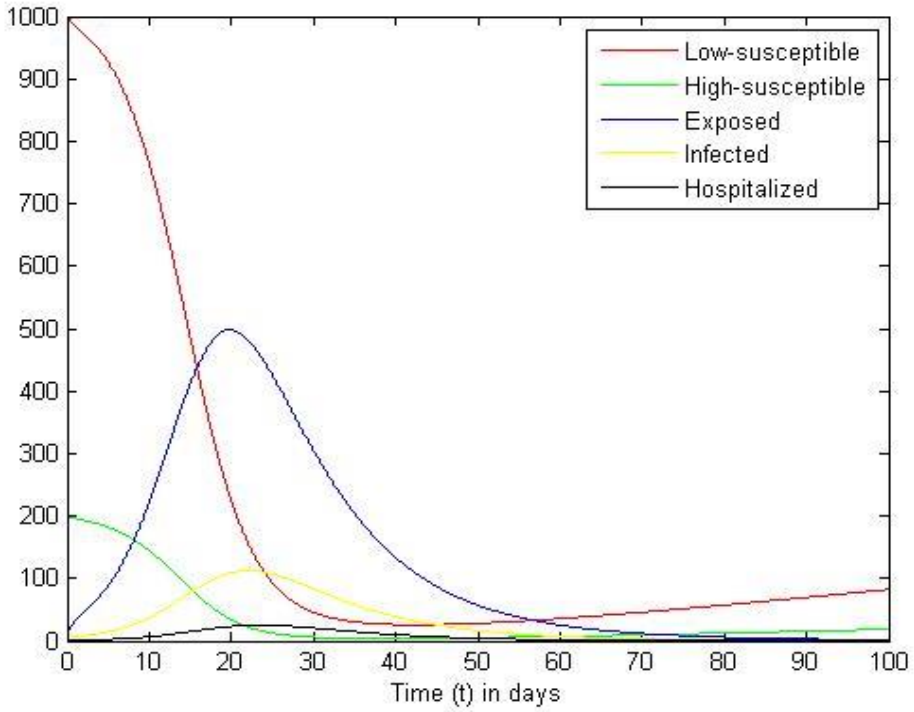

Figure 1 Plot showing the dynamics of the model using $\psi_H = 1.2$, $\delta_I = 0.1$, $\delta_H = 0.5$, $\theta_I = 0.16$, $\theta_H = 0.2$, $\alpha = 0.1$, $\beta = 2.0$, $\pi = 1.7$, $\tau = 0.15$, $\rho = 0.2$, $\mu = 0.00004$

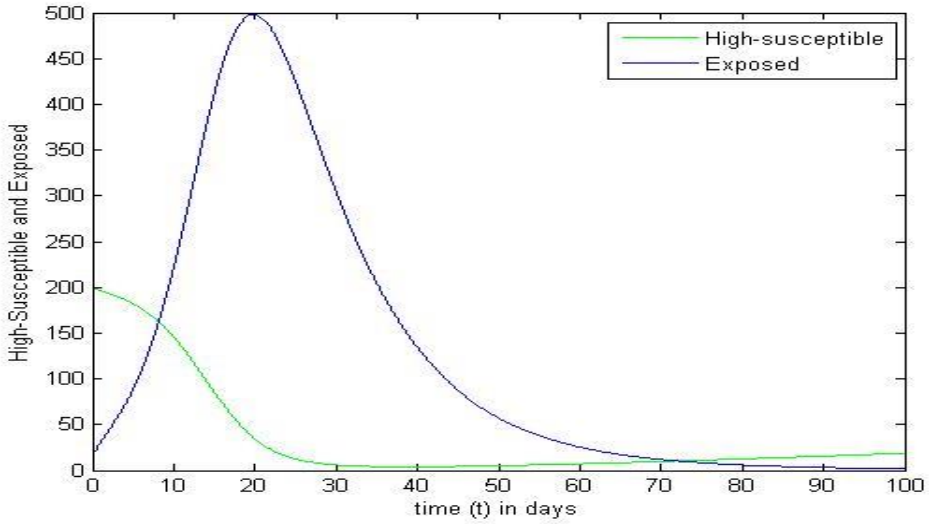

Figure 2 Plot showing the dynamics of High-Susceptible and Exposed.

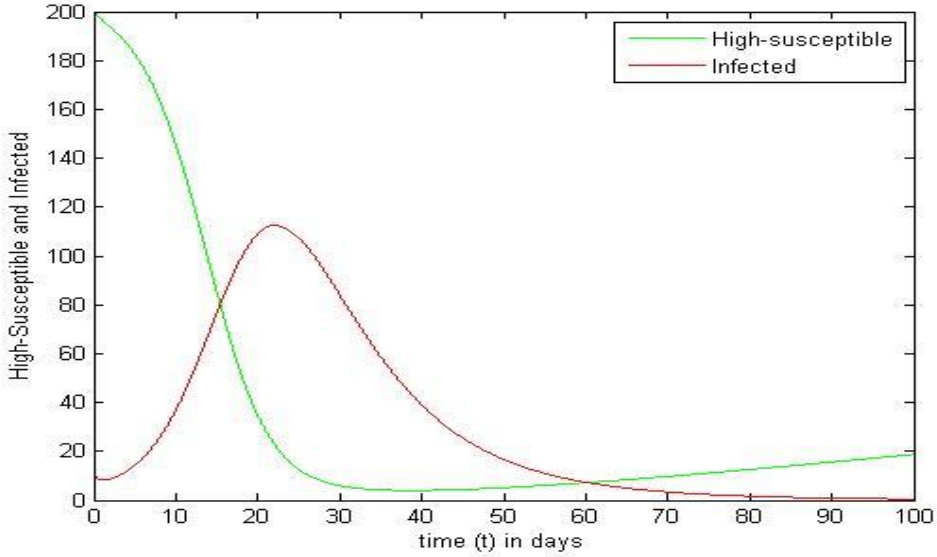

Figure 3 Plot showing the dynamics of High-Susceptible and Infected.

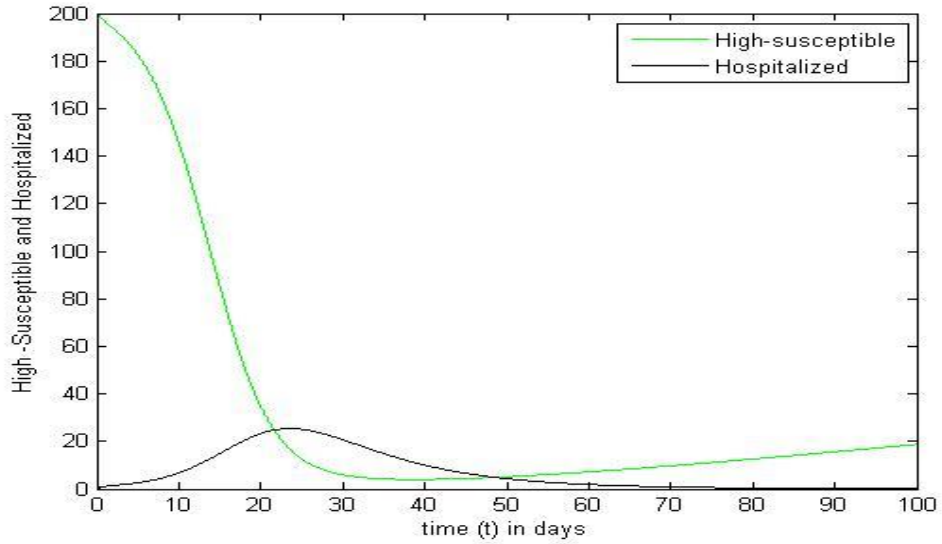

Figure 4 Plot showing the dynamics of High-Susceptible and Hospitalized.

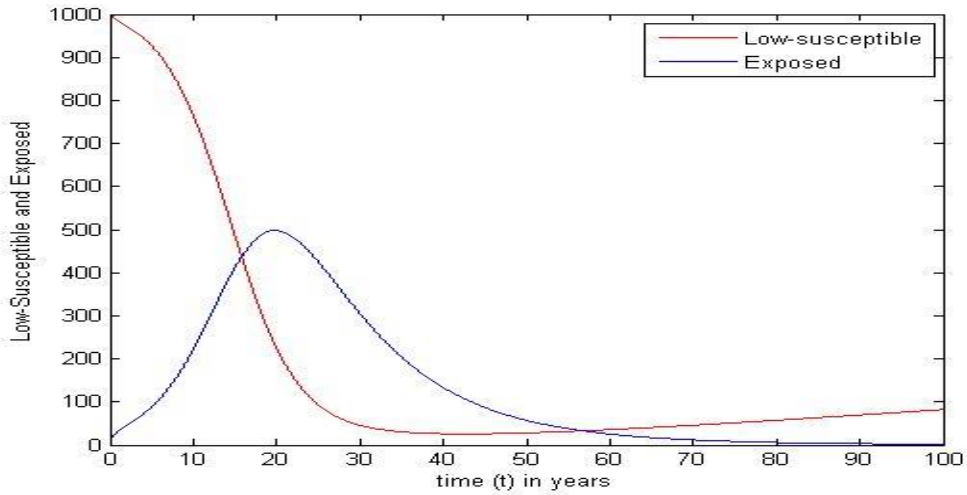

Figure 5 Plot showing the dynamics of Low-Susceptible and Exposed.

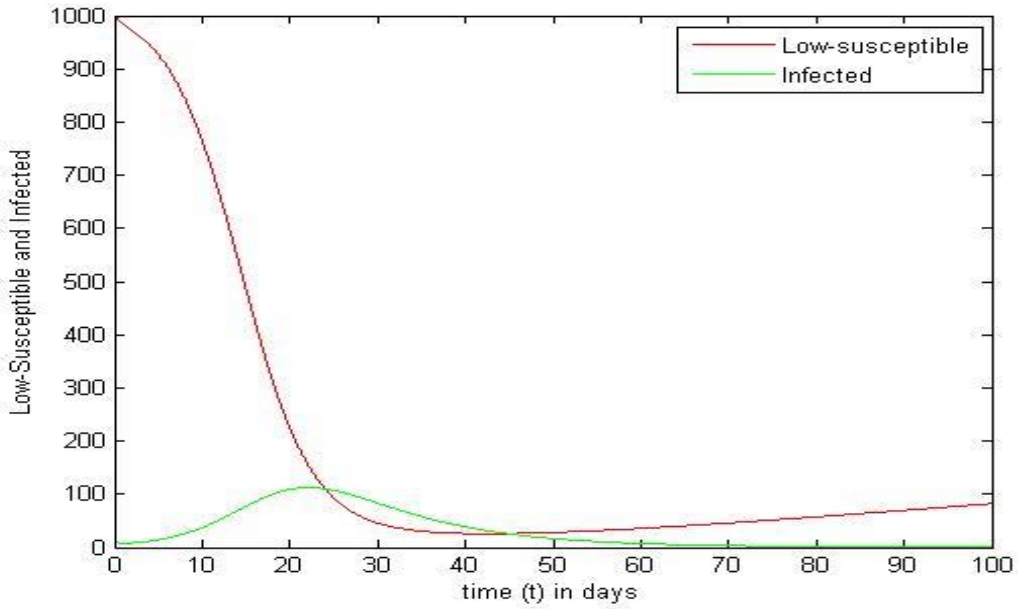

Figure 6 Plot showing the dynamics of Low-Susceptible and Infected.

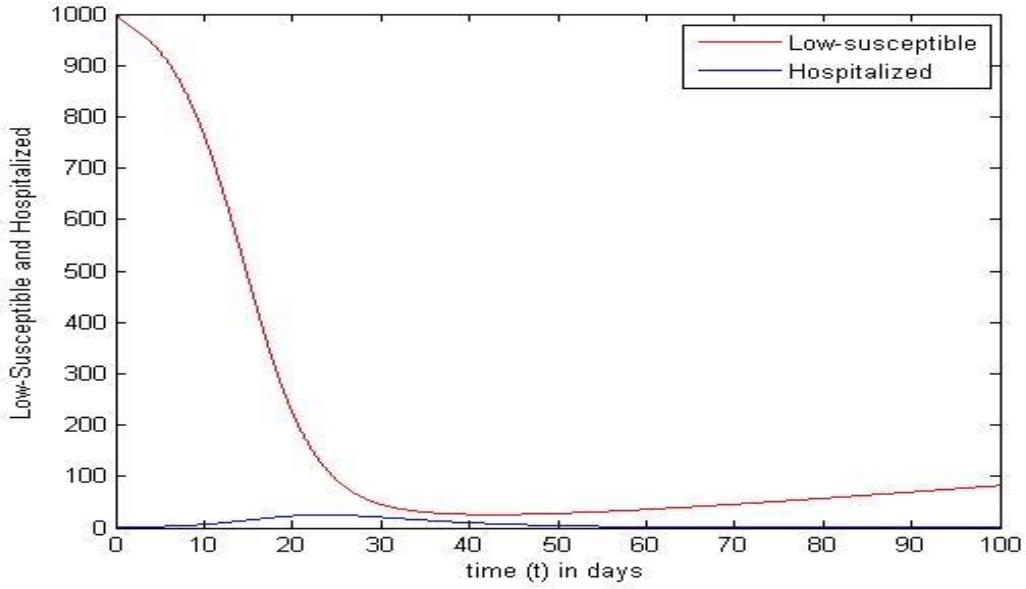

Figure 7 Plot showing the dynamics of Low-Susceptible and Hospitalized.

4.1 Conclusion

In this article, we have analytically studied the qualitative analysis of one of the deadly diseases that affect the human population and mostly in some part of the Africa Continent. Our main purpose of the study was to understand the local stability analysis of the Ebola model that was proposed by Khan et al. [10]. We have numerically simulated the model

dynamics using Matlab. In our future studies of Ebola disease modeling, we will employ the idea of fractional calculus due to the property of memory effect in fractional order derivatives. It will also be interesting to model this disease using stochastic differential equations.

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