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Mathematical Modeling of Ebola Virus Disease Dynamics Incorporating Vital Dynamics, Contact Tracing and Quarantining

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Abstract: This thesis extends the standard SEIR epidemiology model of Ebola virus to include both Human and Monkey population. Nine (9) compartments were considered, namely: (S_H), susceptible Human (E_H), individual that are suspected to have had contact with infected human and monkey (I_H), infected Human (R_H), Recovered Human (Q_H), Quarantine Human and (D_H), Dead Human. For Monkey (S_M), Susceptible Monkey, (I_M), infected Monkey and (D_M), Dead Monkey. We mathematically modeled the natural growth, the interactions between these two populations. The disease-free equilibrium (DFE) and endemic equilibrium (EE) were established. We obtained the basic reproduction number, which can be used to control the transmission dynamics of the disease and thus, established the conditions for local and global stability of the disease free- equilibrium thus, using Routh- Hurwitz criterion and Castillo-Chavez approach respectively. The result of the analysis of the stability of the disease-free equilibrium state that Ebola can totally be eradicated if effort is made to ensure that the rate of recovery infected individuals with Ebola virus and the rate of natural death must have a lower bound. Numerical analysis for the model has done and demonstrated that in the case of patients with Ebola virus, Ebola Virus Disease will be eradicated if effort is intensified in bringing down the transmission rate of Ebola Virus.
Keywords: Vital Dynamics, Contact Tracing and Quarantine

Introduction

Ebola disease is a severe, often fatal illness in humans. The virus is transmitted to people from animals and then spreads in the human population through human -to-human transmission. The average Ebola case fatality rate is around 50%. Early supportive care with rehydration, symptomatic treatment improves survival. Ebola is coursed by infection with a virus of family filoviridae, genus Ebolavirus. There are five identified Ebola virus species, four of which are known to cause disease in humans: Ebola virus (Zaire eblolavirus); Sudan virus (Sudan eblolavius); Tai forest virus (Tai forest ebolavirus, formerly Cotedhh'ivore ebolavirus); and Bundibugyo virus (bundibugyo ebolavirus). The fifth, Reston virus (Restonbolavirus), has caused disease in nonhuman primates, but not in humans (WHO, 2018). A number of different viruses cause viral hemorrhagic fever. Some illness from these infections, such as Lassa fever, dengue, or yellow fever, may be encountered in West Africa and can easily be confused with Ebola virus because symptoms are similar. For example, because Lassa fever is endemic in West Africa and accepted drug treatment (Ribavirin) exists, it is important to differentiate this from Ebola virus (CDC, 2014). Based on evidence and the known transmission cycles of other similar viruses, researchers believe that Ebola virus is animal borne. Bats are the most likely reservoir although the exact species is unknown. Transmission occurs via direct or indirect contact with body fluids from Ebola virus infected persons or animals. Potentially infectious body fluids include blood, respiratory secretions, urine, feces, vomit, saliva, sweat, breast milk, semen, and vaginal secretions. The transmission risk from semen after recovery is uncertain. However, seminal fluid is an immunologically protected site, meaning that antibodies might not have access to virus present in semen. Hence, it is recommended men use condoms for three months after the Ebola virus is no longer detectable in the patient's blood (CDC, 2014). Ebola virus outbreaks have occurred, most notably in parts of Central Africa. However, the largest and most devastating outbreak of EVD is the 2014 epidemic in three West African countries (Guinea, Liberia and Sierra Leone).The first outbreak in West Africa occurred in Guinea in March, 2014. The outbreak was widely spread in Liberia (its capital city Monrovia and other metropolitan cities) and Sierra Leone. The disease also spread to Nigeria by an airline passenger who arrived from Liberia. It spread to Senegal by a student from Guinea who arrived by land transportation. This spread was not limited to Africa alone; it elected a Western European country (Madrid, Spain) and the United States of America (Dallas, Texas; New York City). However, outside the 3 West African countries, there was little to no local transmission, with the only local transmission happening in Nigeria which was quickly contained (Adefisan, 2018). In July 2014, Nigeria experienced an outbreak of Ebola virus disease following the introduction of the disease by an ill Liberian Traveler. The epidemiological profile of the outbreak that majorly affected two States in the country in terms of person, place and time characteristics of the cases identified is hereby described. Using field investigation technique, all confirmed and probable cases were identified, line-listed and analyzed using Microsoft Excel 2007 by persons, time and place. Results indicated that, a total of 20 confirmed and probable cases; 16 in Lagos (including the index case from Liberia) and 4 in Port Harcourt were identified. The mean age was 39.5 ± 12.4 years with over 40% within the age group 30-39 years. The most frequent exposure type was direct physical contact in 70% of all cases and 73% among health care workers. The total case-fatality was 40%; higher among healthcare workers (46%) compared with non-healthcare workers (22%). The epidemic curve initially shows a typical common source outbreak, followed by a propagated pattern. Investigation revealed the size and spread of the outbreak and provided information on the characteristics of persons, time and place. Enhanced surveillance measures, including contact tracing and follow- up proved very useful in early case detection and containment of the outbreak (Musa et al., 2019). There is currently no licensed vaccine available for

Ebola. Several vaccines have been tested, but at this time, not are available for clinical use. At the moment, treatment for Ebola is limited to intensive supportive care and includes: balancing the patience fluids and electrolytes, maintaining their oxygen status and blood pressure and treating the patient for any complicating infections (Nichols, 2019).

Mathematical Formulation

In this paper, we divide the total human population at time (t), denoted by $N_H(t)$ into the following sub-populations of susceptible individuals at time t, $S_H(t)$. those exposed to Ebola virus $E_H(t)$, individuals with Ebola symptom $I_H(t)$, quarantine individual at time t , $Q_H(t)$, individual recovered from Ebola at time t, $R_H(t)$, and those individual who died due to Ebola disease So that $N_H = S_H + E_H + I_H + Q_H + R_H + D_H$ .The total host (monkey) population at time t, denoted by $N_M(t)$, is subdivided into susceptible monkey $S_M(t)$, infected monkey at time t, $I_M(t)$, those monkey that died due to infection with Ebola virus at time t, $D_M(t)$, so that $N_M = S_M + I_M + D_H$. The Human and Monkey recruitment rates are denoted by $\wedge_H$ and $\wedge_M$ respectively. The susceptible human population is increased by migration of quarantine human at the rate ρ. This population further reduced due to the transfer of newly infected individual into exposed human population due to natural death at the rate μ_H,

The population is further decreased due to natural death and due to migration of exposed individual to the infected human population at the rate γ so that the population of infected human I_H increased due to transfer of exposed individual at the rate γ . This population decreased due to natural death at the rate μ_H and due to transfer of infected individual to Quarantine population at the rate μ the population is further decreased by the transfer of death individual to Deceased compartment at the rate τ_H .The population of Quarantine human Q_H increased due to transfer of infected individual at the rate μ . This population decreased due to natural death at the rate μ_H and due to transfer of Quarantine individual to recovered population at the rate φ. The population is further decreased by the transfer of Quarantine individual to Deceased compartment at the rate σ. The population of recovered individual R_H increased due to transfer of Quarantine individual at the rate φ . This population decreased due to natural death at the rate μ_H. the population of deceased increased due to transfer of infected individual at the rate τ_H and also increased due to transfer of Quarantine individual who died at the rate τ_H .The susceptible monkey population is increased due to the recruitment of newly susceptible monkey at the rate $\wedge_M$. The population is further reduced due natural death at the rate μ_M. the population of infected monkey increased due to migration of susceptible monkey. The population reduced due to natural death at the rate μ_M and due to transfer of infected monkey to death monkey compartment at the rate τ_M. The population of death monkey increased due to progression of infected monkey which died at the rate τ_M.

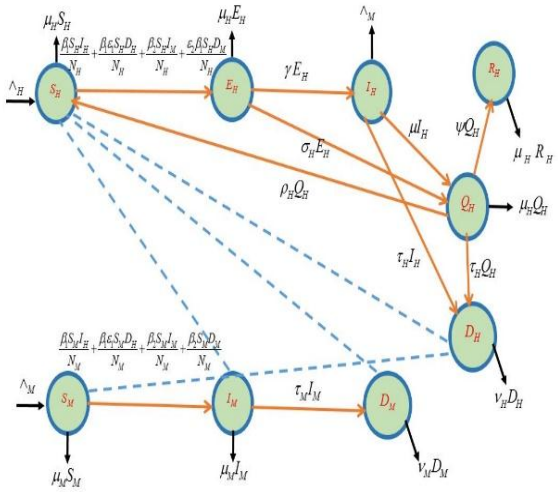

Figure 2.1: Schematic diagram for the model with vital dynamics, contact tracing and Quarantining

Model equations

$$\frac{dS_H}{dt} = \wedge_H + \rho Q_H - \left(\frac{\beta_1 S_H I_H}{N_H} + \frac{\varepsilon_1\beta_1 S_H D_H}{N_H} + \frac{\beta_2 S_H I_M}{N_H} + \frac{\varepsilon_2\beta_1 S_H D_M}{N_H}\right) - \mu_H S_H$$

$$\frac{dE_H}{dt} = \frac{\beta_1 S_H I_H}{N_H} + \frac{\varepsilon_1\beta_1 S_H D_H}{N_H} + \frac{\beta_2 S_H I_M}{N_H} + \frac{\varepsilon_2\beta_1 S_H D_M}{N_H} - \mu_H E_H - \gamma E_H - \sigma_H E_H$$

$$\frac{dI_H}{dt} = \gamma E_H - \mu_H I_H - \mu I_H - \tau_H I_H$$

$$\frac{dQ_H}{dt} = \mu I_H + \sigma_H E_H - \mu_H Q_H - \varphi Q_H - \tau_H Q_H - \rho Q_H$$

$$\frac{dR_H}{dt} = \varphi Q_H - \mu_H R_H$$

$$\frac{dD_H}{dt} = \tau_H I_H + \tau_H Q_H - \nu_H D_H$$

$$\frac{dS_M}{dt} = \wedge_M - \left(\frac{\beta_1 S_M I_H}{N_M} + \frac{\varepsilon_1\beta_1 S_M D_H}{N_M} + \frac{\beta_2 S_M I_M}{N_M} + \frac{\varepsilon_2\beta_1 S_M D_M}{N_M}\right) - \mu_M S_M$$

$$\frac{dI_M}{dt} = \frac{\beta_1 S_M I_H}{N_M} + \frac{\varepsilon_1\beta_1 S_M D_H}{N_M} + \frac{\beta_2 S_M I_M}{N_M} + \frac{\varepsilon_2\beta_2 S_M D_M}{N_M} - \mu_M I_M - \tau_M I_M$$

$$\frac{dD_M}{dt} = \tau_M I_M - \nu_M D_M$$

Where the total populations and initial conditions are:

$$\left.\begin{aligned} N_H &= S_H + E_H + I_H + Q_H + D_H + R_H \\ N_M &= S_M + I_M + D_M \end{aligned}\right\}$$

$$\left.\begin{aligned} S_H(0) = S_{H0}, E_H(0) = E_{H0}, I_H(0) = I_{H0}, Q_H(0) = Q_{H0}, D_H(0) = D_{H0}, R_H(0) = R_{H0} \\ S_M(0) = S_{M0}, I_M(0) = I_{M0}, D_M(0) = D_{M0} \end{aligned}\right\}$$

Table 1. Variables and parameter for the model with vital dynamics, contact tracing and Quarantine

Variables	*Description*
S_M	Susceptible monkey population at time t
S_H	Susceptible Human population at time t
E_H	Individual that are suspected to have had contact with infectious Monkey or Humans
I_M	Infected monkey population at time t
I_H	Infected human population at time t
Q_H	Quarantine human at time t
D_H	Death human at time t
D_M	Death Monkey at time t
$\wedge_H$	Recruitment rate into the susceptible human population
$\wedge_M$	Recruitment rate into the susceptible Monkey population
ε	Deceased transmission rate
μ_H	Natural mortality rate in human population
μ	Progression rate from infectious human to quarantine
μ_M	Natural mortality rate in monkey
γ	Progression rate from quarantine to recovered human
σ_H	Progression rate from quarantine to death human
τ_H	Progression rate from infectious to death human
ρ	Progression rate from infectious to susceptible human
τ_M	Progression rate from infectious to death Monkey
ν_H	the rate of proper burial
ν_M	the rate of successful cremate

Model Analysis

Positivity of solutions

***Theorem1*:** Let the initial solution set $\left(S_{H0} > 0, E_{H0} > 0, I_{H0} > 0, Q_{H0} > 0, R_{H0} > 0, D_{H0} > 0, S_{M0} > 0, I_{M0} > 0, D_{M0} > 0\right)$ Then, the solution set $\left(S_H, E_H, I_H, Q_H, R_H, D_H, S_M, I_M, D_M\right)$ is positive for all time, $t > 0$.

Invariant region

Let $\left(S_H + E_H + I_H + Q_H + R_H + D_H\right)$ and $N_M = S_M + I_M + D_M$ be the solution of the model equations (1) for Human and monkey population with the initial conditions and biological feasible region given by

$$\Omega_1 = \left\{(S_H, E_H, I_H, Q_H, R_H, D_H) \in \Re_+^6 : N_H \le \frac{\wedge_H}{\mu_H}\right\}$$

and $\Omega_2 = \left\{(S_M, I_M, D_M) \in \Re_+^3 : N_M \le \frac{\wedge_M}{\mu_M}\right\}$. From (1), the invariant region for the solution of all the populations is $\Omega = \Omega_1 \bigcup \Omega_2 \subset \Re_+^6 \times \Re_+^3 \subset \Re_+^9$

Thus, the region Ω is positively invariant. The model (1) can be considered epidemiologically and mathematically well-posed in the region.

Disease free and the endemic equilibrium

setting

$$\frac{dS_H}{dt} = \frac{dE_H}{dt} = \frac{dI_H}{dt} = \frac{dQ_H}{dt} = \frac{dR_H}{dt} = \frac{dD_H}{dt} = \frac{dS_M}{dt} = \frac{dI_M}{dt} = \frac{dD_M}{dt} = 0$$

the disease free and endemic equilibrium state (DFE) and (EES) denoted by E_0 **and** E_1 of system (1) is given by and

$$\left.\begin{aligned}
S_H^* &= \frac{N_H(\Lambda_H + \rho Q^*_H)}{\beta_1 I^*_H + \varepsilon_1\beta_1 D^*_H + \beta_2 I^*_M + \varepsilon_2\beta_1 D^*_M + \mu_H N_H} \\
E_H^* &= \frac{\beta_1 S^*_H I^*_H + \varepsilon_1\beta_1 S^*_H D^*_H + \beta_2 S^*_H I^*_M + \varepsilon_2\beta_1 S^*_H D^*_M}{(\mu_H + \gamma + \sigma_H)N_H} \\
I_H^* &= \frac{\gamma E^*_H}{(\mu_H + \mu + \tau_H)} \\
Q_H^* &= \frac{\mu I^*_H + \sigma_H E^*_H}{(\mu_H + \varphi + \tau_H + \rho)} \\
R_H^* &= \frac{\varphi Q^*_H}{\mu_H} \\
D_H^* &= \frac{\tau_H I^*_H + \tau_H Q^*_H}{\nu_H} \\
S_M^* &= \frac{N_M \Lambda_M}{\left(\beta_1 I^*_H + \varepsilon_1\beta_1 D^*_H + \beta_2 I^*_M + \varepsilon_2\beta_1 D^*_M\right) + \mu_M} \\
I_M^* &= \frac{\beta_1 S^*_M I^*_H + \varepsilon_1\beta_1 S^*_M D^*_H + \varepsilon_2\beta_2 S^*_M D^*_M}{N_H(\mu_M + \tau_M) - \beta_2 S^*_M} \\
D_M^* &= \frac{\tau_M I^*_M}{\nu_M}
\end{aligned}\right\}$$

respectively.

Basic Reproduction Number

The basic reproduction number is denoted by R_0. It is an important parameter that is used to study the behavior of epidemiological models. It is defined as the average number of secondary infections infected by an infective individual during an infective period provided that all members of the population are susceptible. It is an important threshold parameter that determines whether or not, an infection will spread through a given population.

We apply the next generation matrix technique by (Diekmann, Heesterbeek & Metz, 1990) to obtain the basic reproduction number, R_0 by considering the infected compartments of the system (1). Let F_i be the rate of appearance of new infection in the i compartment and V_i be the rate of transfer of individuals out of i, given the disease free equilibrium, then R_O is the spectral radius (largest Eigenvalues) of the next generation matrix denoted by $G = FV^{-1}$

$$R_0 = \rho FV^{-1}$$

$$R_0 = \frac{\gamma\beta_1}{a_1 a_2} + \frac{\varepsilon_1\beta_1(\gamma\mu\tau_H + \gamma\mu a_3)}{a_1 a_2 a_3} + \frac{\beta_2(\gamma\mu\tau_H + \gamma\tau_H a_3 + \sigma_H\tau_H a_2)}{a_1 a_2 a_3 a_4}$$

Local stability of the disease free equilibrium

Theorem 2: The disease free equilibrium point, E_O is locally asymptotically stable if $R_O < 1$ and unstable if $R_o > 1$.

Proof: Let

$$\left.\begin{aligned}
F_1 &= \wedge_H - \left(\frac{\beta_1 S_H I_H}{N_H} + \frac{\varepsilon_1\beta_1 S_H D_H}{N_H} + \frac{\beta_2 S_H I_M}{N_H} + \frac{\varepsilon_2\beta_1 S_H D_M}{N_H}\right) - \mu_H S_H + \rho Q_H \\
F_2 &= \frac{\beta_1 S_H I_H}{N_H} + \frac{\varepsilon_1\beta_1 S_H D_H}{N_H} + \frac{\beta_2 S_H I_M}{N_H} + \frac{\varepsilon_2\beta_1 S_H D_M}{N_H} - \mu_H E_H - \gamma E_H - \sigma_H E_H \\
F_3 &= \gamma E_H - \mu_H I_H - \mu I_H - \tau_H I_H \\
F_4 &= \mu I_H - \mu_H Q_H - \varphi Q_H - \tau_H Q_H - \rho Q_H + \sigma_H E_H \\
F_5 &= \varphi Q_H - \mu_H R_H \\
F_6 &= \tau_H I_H + \tau_H Q_H - V_H D_H \\
F_7 &= \wedge_M - \frac{\beta_1 S_M I_H}{N_M} + \frac{\varepsilon_1\beta_1 S_M D_H}{N_M} + \frac{\beta_2 S_M I_M}{N_M} + \frac{\varepsilon_2\beta_1 S_M D_M}{N_M} - \mu_M S_M \\
F_8 &= \frac{\beta_1 S_M I_H}{N_M} + \frac{\varepsilon_1\beta_1 S_M D_H}{N_M} + \frac{\beta_2 S_M I_M}{N_M} + \frac{\varepsilon_2\beta_2 S_M D_M}{N_M} - \mu_M I_M - \tau_M I_M \\
F_9 &= \tau_M I_M - V_M D_M
\end{aligned}\right\} \quad (2)$$

Evaluating the Jacobean matrix $J(E_0)$ of the system (2), we have

$$J(E_O) = \begin{bmatrix}
J_{11} & 0 & J_{13} & J_{14} & 0 & J_{16} & 0 & J_{18} & J_{19} \\
J_{21} & J_{22} & J_{23} & 0 & 0 & J_{26} & 0 & J_{28} & J_{29} \\
0 & J_{32} & J_{33} & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & J_{42} & J_{43} & J_{44} & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & J_{54} & J_{55} & 0 & 0 & 0 & 0 \\
0 & 0 & J_{63} & J_{64} & 0 & J_{66} & 0 & 0 & 0 \\
0 & 0 & J_{73} & 0 & 0 & J_{76} & J_{77} & J_{78} & J_{79} \\
0 & 0 & J_{83} & 0 & 0 & J_{86} & J_{87} & J_{88} & J_{89} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & J_{98} & J_{99}
\end{bmatrix} \quad (3)$$

where

$$J_{11}=-\frac{\beta_1}{N_H}(I_H+\varepsilon_1 D_H+\varepsilon_2 D_M)-\frac{\beta_2 I_M}{N_H}-\mu_H, J_{13}=\frac{-\beta_1 S_H}{N_H}, J_{14}=\rho, J_{16}=\frac{-\varepsilon_1\beta_1 S_H}{N_H}, J_{99}=-\nu_M$$

$$J_{18}=\frac{-\beta_2 S_H}{N_H}, J_{19}=\frac{-\varepsilon_2\beta_1 S_H}{N_H}, J_{21}=\frac{\beta_1}{N_H}(I_H+\varepsilon_1 D_H+\varepsilon_2 D_M)+\frac{\beta_2 I_M}{N_H}, J_{22}=-\mu-\gamma-\sigma_{,H}$$

$$J_{23}=\frac{\beta_1 S_H}{N_H}, J_{26}=\frac{\varepsilon_1\beta_1 S_H}{N_H}, J_{28}=\frac{\beta_2 S_H}{N_H}, J_{29}=\frac{\varepsilon_2\beta_1 S_H}{N_H} J_{32}=\gamma, J_{83}=\frac{\beta_1 S_M}{N_M}, J_{86}=\frac{\varepsilon_1\beta_1 S_M}{N_M},$$

$$J_{87}=\frac{\beta_1}{N_H}(I_H+\varepsilon_1 D_H+\varepsilon_2 D_M)+\frac{\beta_2 I_M}{N_H}, J_{88}=\frac{\beta_2 S_M}{N_M}-\mu_M-\tau_M, J_{89}=\frac{\varepsilon_2\beta_1 S_M}{N_M}, J_{98}=\tau_M,$$

Evaluating (3) at disease free we have

$$J(E_0)=\begin{bmatrix}
-\mu_H & 0 & \beta_1 & \rho & 0 & -\varepsilon_1\beta_1 & 0 & -\beta_2 & -\varepsilon_2\beta_1 \\
0 & -(\mu+\gamma+\sigma_H) & \beta_1 & 0 & 0 & \varepsilon_1\beta_1 & 0 & \beta_2 & \varepsilon_2\beta_1 \\
0 & \gamma & -(\mu_H+\mu+\tau_H) & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \sigma & \mu & -(\mu_H+\varphi+\tau_H+\rho) & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \varphi & -\mu_H & 0 & 0 & 0 & 0 \\
0 & 0 & \tau_H & 0 & 0 & -\nu_H & 0 & 0 & 0 \\
0 & 0 & -\beta_1 & 0 & 0 & -\varepsilon_1\beta_1 & -\mu_M & -\beta_2 & -\varepsilon_2\beta_1 \\
0 & 0 & \beta_1 & 0 & 0 & \varepsilon_1\beta_1 & 0 & \beta_2-(\mu_M+\tau_M) & \varepsilon_2\beta_1 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau_M & -\nu_M
\end{bmatrix}$$

(4)

We need to show that all eigenvalues of (CDC, 2014) are negative. We observe that the first, fifth and sixth columns contain only the diagonal terms which form the first three eigenvalues;

$$\lambda_1=-\mu_H \quad , \quad \lambda_1=-\mu_H, \quad \lambda_1=-\mu_M$$

From (4) the other six eigenvalues can be obtained from sub-matrix, $J_1(E_0)$ as

$$\left|J_1(E_0)-\lambda I\right|=\begin{vmatrix}
-(\mu_H+\gamma)-\lambda & \beta_1 & 0 & \varepsilon_1\beta_1 & \beta_2 & \varepsilon_2\beta_1 \\
\gamma & -(\mu_H+\mu+\tau_H)-\lambda & 0 & 0 & 0 & 0 \\
\sigma & \mu & -(\mu_H+\varphi+\tau_H+\rho)-\lambda & 0 & 0 & 0 \\
0 & \tau_H & \tau_H & -\nu_H-\lambda & 0 & 0 \\
0 & \beta_1 & 0 & \varepsilon_1\beta_1 & -[\beta_2(\mu_M+\tau_M)]-\lambda & \varepsilon_2\beta_1 \\
0 & 0 & 0 & 0 & \tau_M & -\nu_M-\lambda
\end{vmatrix}=0$$

(5)

The eigenvalues of the matrix $J_1(E_0)$ (5) are the roots of the characteristic equation

$$A_6\lambda^6+A_5\lambda^5+A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_0$$

(6)

Where:

$$A_6=1$$

$$A_5=(a_1+a_2+a_3+a_4+a_5+a_6)$$

$$A_4=(a_6(a_1+a_2+a_3+a_4+a_5)+a_3(a_1+a_2)-\gamma\beta_1-\tau_M b_2+a_1a_2+a_4(a_1+a_2+a_3)+a_5(a_1+a_2+a_3+a_4))$$

$$A_3=(a_6(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2+a_4(a_1+a_2+a_3)+a_5(a_1+a_2+a_3+a_4)+a_4(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2)+a_5(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2+a_4(a_1+a_2+a_3)-a_3(\gamma\beta_1-a_1a_2)-\tau_M b_2(a_1+a_2+a_3+a_4+a_5)-\gamma\beta_1\beta_2-\sigma\tau_H b_1-\gamma\tau_H b_1+\tau_M b_2 a_5)$$

$$A_2=(\tau_H(\gamma a_1b_1+\gamma\alpha_2b_1)-(\sigma\tau_H b_1+\gamma\tau_H b_1)(a_1+a_2+a_3)-a_5(a_3(\gamma\beta_1-a_1a_2)-a_4(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2)+\sigma\tau_H b_1+\sigma\tau_H b_1)-a_6(a_3(\gamma\beta_1-a_1a_2)-a_5(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2+a_4(a_1+a_2+a_3)-a_4(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2)+\gamma\beta_1\beta_2+\sigma\tau_H b_1+\sigma\tau_H b_1)-\tau_M(b_2a^2{}_5+\gamma\beta_1\beta_2)+\tau_H(\sigma a_1b_1-\gamma\mu b_1+\sigma a_3b_1-b_1(\sigma\beta_2\tau_H+\gamma\beta_2\tau_H)+\beta_1(\gamma\beta_2a_1+\gamma\beta_2a_2)-\tau_M b_2(a_3(a_1+a_2)-\gamma\beta_1+\sigma$$

$$A_1=(b_1a_4(\sigma\beta_2\tau_H+\gamma\beta_2\tau_H)+\tau_H(\gamma\beta_2a_1+\gamma\beta_2a_2)+\tau_H(\sigma\beta_2a_1-\gamma\mu\beta_2+\sigma\beta_2a_3))-a_6(a_5(a_3(\gamma\beta_1-a_1a_2)-a_4(a_3(a_1+a_2)-\gamma\beta_1+a_1a_2+\sigma\tau_H b_1+\gamma\tau_H b_1)+(\sigma\tau_H b_1+\gamma\tau_H b_1)(a_1+a_2+a_3)-\tau_H(\gamma a_1b_1+\gamma a_1b_1)-\tau_H(\sigma a_1b_1-\gamma\mu b_1+\sigma a_3b_1)+b_1(\sigma\beta_2\tau_H+\gamma\beta_1\tau_H)-\beta_1(\gamma\beta_2a_2+\gamma\beta_2a_2)+a_3a_4(\gamma\beta_1-a_1a_2)+\gamma\beta_1\beta_2(a_1+a_2+a_3+a_4)-\tau_M(b_1(\sigma\tau_H b_2+\gamma\tau_H b_2)-a_5(b_2a_5\gamma\beta_1b_2)+\beta_1$$

$$A_0=(\tau_M(a_5(b_1(\sigma\tau_H b_2+\gamma\tau_H b_2)-a_5(b_2a^2{}_5+\gamma\beta_1b_2)+\beta_1(\gamma\beta_2b_2-a_1b_1)-\gamma a_2b_2))-b_1(\tau_H(\sigma(\beta_2b_2-a_1b_2)+\gamma\mu b_2-\sigma a_3b_2)-a_4(\sigma\tau_H b_2+\gamma\tau_H b_2)+\tau_M(\gamma(\beta_2b_2-a_1b_2)-\gamma a_2b_2))+\beta_1(a_2(\gamma(\beta_2b_2-a_1b_2)-\gamma a_2b_2)+\gamma(\alpha_1(\beta_2b_2-a_1b_2)-\gamma\beta_1b_2+\beta_2b_2a_5)))-a_6(a_5((\gamma\tau_H b_1+\gamma\tau_H b_1)(a_1+a_2+a_3)-\tau_H(\gamma a_1b_1+\gamma a_2b_1)-\tau_H(\sigma a_1b_1-\gamma\mu b_1\sigma a_3b_1)+a_3a_4(\gamma\beta_1-a_1a_2))+\beta_1(a_2(\gamma\beta_2a_1))$$

We employ the Routh – Hurwitz criterion, which states that all roots of the polynomial (6) have negative real parts if and only if the coefficients A_i are positive and matrices $H_i>0$, for $i=0,1,2,3,4,5,6.$

From (6) we observe that $A_1>0, A_2>0, A_3>0, A_4>0, A_5>0, A_6>0]$

Also the Hurwitz matrices for the polynomial (6) are found to be positive. That is,

$$H=\begin{bmatrix}
A_1 & A_3 & 0 & 0 & 0 & 0 \\
1 & A_2 & A_4 & 0 & 0 & 0 \\
0 & A_1 & A_3 & A_5 & 0 & 0 \\
0 & 1 & A_2 & A_4 & A_0 & 0 \\
0 & 0 & A_1 & A_3 & A_5 & 0 \\
0 & 0 & 1 & A_2 & A_4 & A_0
\end{bmatrix}$$

(7)

H is called the Hurwitz matrix. The principal minors are:

$$H_1=A_1>0$$

$$H_2=\begin{vmatrix} A_1 & A_3 \\ 1 & A_2 \end{vmatrix}=A_1A_2-A_3>0$$

$$H_3=\begin{vmatrix} A_1 & A_3 & 0 \\ 1 & A_2 & A_4 \\ 0 & A_1 & A_3 \end{vmatrix}=A_1(A_2A_3-A_1A_4)-A_3{}^2>0$$

$$H_4=\begin{vmatrix} A_1 & A_3 & 0 & 0 \\ 1 & A_2 & A_4 & 0 \\ 0 & A_1 & A_3 & A_5 \\ 0 & 1 & A_2 & A_4 \end{vmatrix} A_1A_2A_3A_4 - A^2{}_2A_5 - A^2{}_1A^2{}_4 + A_1A_4A_5 - A^2{}_3A_1 + A_3A_2A_5 > 0$$

$$H_5=\begin{vmatrix} A_1 & A_3 & A_5 & 0 & 0 \\ 1 & A_2 & A_4 & A_0 & 0 \\ 0 & A_1 & A_3 & A_5 & 0 \\ 0 & 1 & A_2 & A_4 & A_0 \\ 0 & 0 & A_1 & A_3 & A_5 \end{vmatrix}$$

$$= A_1A_2A_3A_4A_5 + A^2{}_1A_5A_2A_0 + A_0A_2A^2{}_1A_5 + A^2{}_1A_0A_3A_4 + A_3A_2A^2{}_5 + A^2{}_5A_1A_3 + A_0A_3A^2{}_3$$
$$> A_5A^3{}_3 + A^3{}_5 + A_0A_3A_1A_5 + A_0A_5A_1A_3 + A_1A^2{}_2A^2{}_5 + A^2{}_1A_5A^2{}_4 + A_1A_0A_2A^2{}_3 + A^3{}_1A^2{}_0$$

$$H_6=\begin{vmatrix} A_1 & A_3 & A_5 & 0 & 0 & 0 \\ 1 & A_2 & A_4 & A_0 & 0 & 0 \\ 0 & A_1 & A_3 & A_5 & 0 & 0 \\ 0 & 1 & A_2 & A_4 & A_0 & 0 \\ 0 & 0 & A_1 & A_3 & A_5 & 0 \\ 0 & 0 & 1 & A_2 & A_4 & A_0 \end{vmatrix}$$

$$= A_1A_0A_5A_2A_3A_4 - A_1A_0A_2A_5 - A_0A^2{}_1A^2{}_4 + A^2{}_1A^2{}_0A_2 + A_1A_0A_4A_5 - A^2{}_0A_1A_3 - A^2{}_0A_1A_5A_2A_3 + A^2{}_0A^2{}_1A_5A_4$$
$$- A_0A_5A^2{}_3A_4 + A_0A_2A_5 + A_0A_5A_1A_4 - A_0A^2{}_5 + A_0A^3{}_3 - A_0A_3A_1A_5 + A_0A_5A_1A_3 > 0$$

.

Therefore, all the eigenvalues of the Jacobian matrix J(E0) have negative real parts when $R_0<1$ and the disease-free equilibrium point is locally asymptotically stable.

Global Stability of the Disease Free Equilibrium

We employ the method of (Castillo-Chavez, Feng & Huang, 2002) for analyzing global stability of DFE. The GAS is achieved if the two conditions below are met. The model equations (1) are rewritten in the form:

$$\left.\begin{aligned} \frac{dX}{dt} &= F(X,Z) \\ \frac{dZ}{dt} &= G(X,Z);\quad G(X,0)=0 \end{aligned}\right] \tag{9}$$

Where $X\in\mathbb{R}^2$ denote the number of uninfected individuals and $Z\in\mathbb{R}^7$ denote the number of infected individuals. The DFE of the model below

$$E_0=(S^*_H, E^*_H, I^*_H, Q^*_H, R^*_H, D^*_H, S^*_M, I^*_M, D^*_M)=\left(\frac{\Lambda_H}{\mu_H},0,0,0,0,0,\frac{\Lambda_M}{\mu_M},0,0\right)$$

Condition (9) may be met to guaranteed global asymptotic stability

(H_1): For $\frac{dX}{dt}=K(X,0)$, X^* is globally asymptotic stable

(H_2): For $G(X,Z)=AZ-\hat{G}(X,Z),\hat{G}(X,Z)\geq 0\forall(X,Z)\in\Pi$

where $A=D_ZG(X^*,0)$ is an M

Where $A=D_vG(X,0)$ is an M matrix (The off diagonal elements are non-negative) and Π is the biological feasible region. If the two conditions given above are satisfied by the model equations, them the following theorem holds.

Theorem 2: If system (1) satisfies condition (6), then the fixed point $E_0=\left(\frac{\Lambda_H}{\mu_H},0,0,0,0,0,\frac{\Lambda_M}{\mu_M},0,0\right)$ is a globally asymptotically stable equilibrium of the system (1) provided that $R_0<1$ and the conditions (H_1) and (H_2) are satisfied:

Proof:

Consider $\frac{dX}{dt}=F(X,0)=\begin{pmatrix}\wedge_H-\mu_HS_H\\ \wedge_M-\mu_MS_M\end{pmatrix}$

Therefore, $X^*=\left[\frac{\Lambda_H}{\Lambda_H},\frac{\Lambda_M}{\Lambda_M}\right]$ is globally asymptotically stable and

$$G(X,Z)=AZ-\hat{G}(X,Z)$$

(10)

$$A=\begin{bmatrix} -(\mu_H+\gamma+\sigma) & \frac{\beta_1S_H}{N_H} & 0 & 0 & \frac{\varepsilon_1\beta_1S_H}{N_H} & \frac{\beta_2S_H}{N_H} & \frac{\varepsilon_2\beta_1S_H}{N_H} \\ \gamma & -(\mu_H+\mu+\tau_H) & 0 & 0 & 0 & 0 & 0 \\ \sigma & \mu & -(\mu_H+\varphi+\tau_H+\rho) & 0 & 0 & 0 & 0 \\ 0 & 0 & \varphi & -\mu_H & 0 & 0 & 0 \\ 0 & \tau_H & \tau_H & 0 & -\nu_H & 0 & 0 \\ 0 & \frac{\beta_1S_M}{N_M} & 0 & 0 & \frac{\varepsilon_1\beta_1S_M}{N_M} & \frac{\left[\beta_2-(\mu_M+\tau_M)\right]S_M}{N_M} & \frac{\varepsilon_2\beta_2S_M}{N_M} \\ 0 & 0 & 0 & 0 & 0 & \tau_M & -\nu_M \end{bmatrix}$$

(11)

$$\hat{G}(X,Z)=\begin{bmatrix} \frac{\beta_1S_HI_H}{N_H}+\frac{\varepsilon_1\beta_1S_HD_H}{N_H}+\frac{\beta_2S_HI_M}{N_H}+\frac{\varepsilon_2\beta_1S_HD_M}{N_H}-\mu_HE_H-\gamma E_H-\sigma E_H \\ \gamma E_H-\mu_HI_H-\mu I_H-\tau_HI_H \\ \mu I_H+\sigma E_H-\mu_HQ_H-\varphi Q_H-\tau_HQ_H-\rho Q_H \\ \varphi Q_H-\mu_HR_H \\ \tau_HI_H+\tau_HQ_H-\nu_HD_H \\ \frac{\beta_1S_MI_H}{N_M}+\frac{\varepsilon_1\beta_1S_MD_H}{N_M}+\frac{\beta_2S_MI_M}{N_M}+\frac{\varepsilon_2\beta_2S_MD_M}{N_M}-\mu_MI_M-\tau_MI_M \\ \tau_MI_M-\nu_MD_M \end{bmatrix}$$

(12)

Evaluating (10) using (11) and and (12) we have

$$G(X,Z) = AZ - \hat{G}(X,Z) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (13)$$

i.e. $G(X,Z) = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}^T$. This shows that $G(X,Z) = 0$. Hence, the model is globally asymptotically stable.

Numerical Simulation

In this section, we carry out some numerical simulations using ode function from MATLABR2016a.

Parameter/Variable	Values	Reference
μ_H	4.65753×10^{-5} day^{-1}	(Madubueze, Kimbir & Aboiyar, 2018)
μ_M	0.033652	Assumed
τ_H	0.489	Assumed
τ_M	0.0846	Assumed
ρ	0.047619 day^{-1}	(River, Lofgren, Marathe, Eubank & Lewis, 2014)
φ	0.0314862 day^{-1}	(River, Lofgren, Marathe, Eubank & Lewis, 2014)
$\wedge_H$	422 day^{-1}	(Madubueze, Kimbir & Aboiyar, 2018)
σ_H	0.00005	(Madubueze, Kimbir & Aboiyar, 2018)
ε_1	0.160	(River, Lofgren, Marathe, Eubank & Lewis, 2014)
ε_2	0.35	Assumed
γ	0.06	(Madubueze, Kimbir & Aboiyar, 2018)
μ	0.160	(River, Lofgren, Marathe, Eubank & Lewis, 2014)
ν_M	0.63	(Agness, 2018)
β_1	0.35	Assumed
$S_H(0)$	750 day^{-1}	Assumed
$E_H(0)$	200	Assumed
$I_H(0)$	10	Assumed
$Q_H(0)$	150	Assumed
$R_H(0)$	6	Assumed
$I_M(0)$	2	Assumed
$D_M(0)$	15	Assumed
$S_M(0)$	150	Assumed
$N_H(0)$	1126	Assumed
$N_M(0)$	1715	Assumed

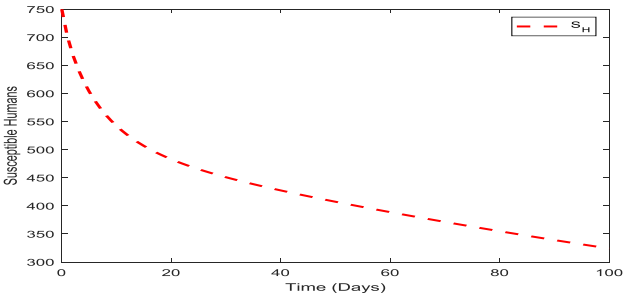

Figure.1 Numerical solution of susceptible humans.

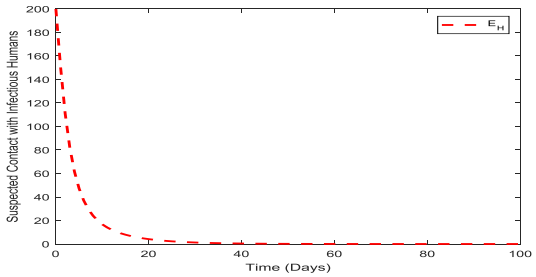

Figure. 2 Numerical solution of suspected contact with infectious humans.

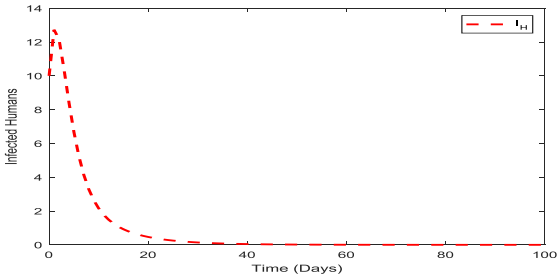

Figure 3 Numerical solution of infected humans.

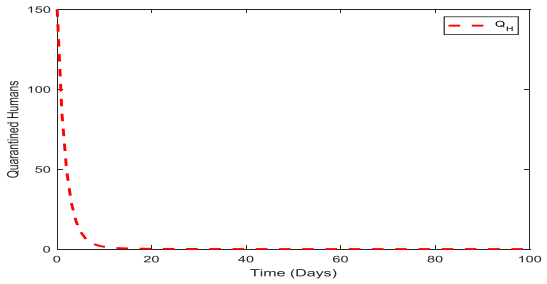

Figure 4 Numerical solution of quarantined humans

From Figure (1) to Figure (4) shows that population of susceptible humans (S_H), Exposed humans (E_H), infected humans (I_H), and Quarantine humans (Q_H) reduces significantly in the presence of Ebola Disease. This simply means in the presence of Ebola disease the population will drastically reduce due to infections.

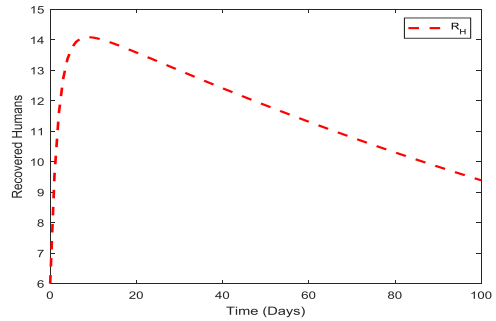

Figure 5.Numerical solution of recovered humans.

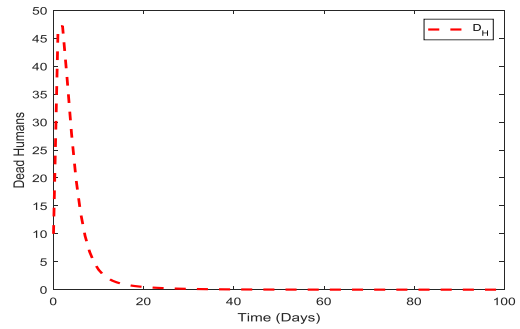

Figure 6. Numerical solution of dead humans.

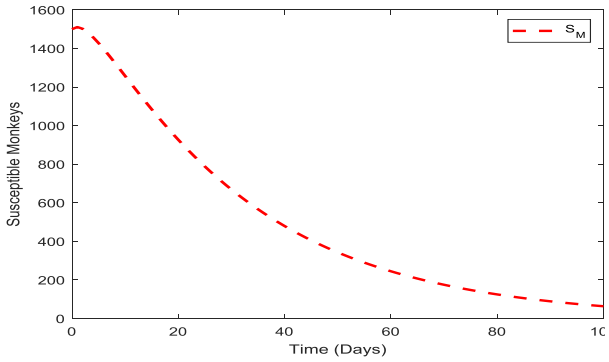

Figure 7.Numerical solution of susceptible monkeys.

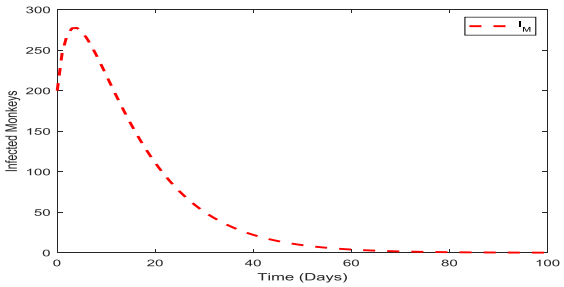

Figure 8 Numerical solution of infected monkeys.

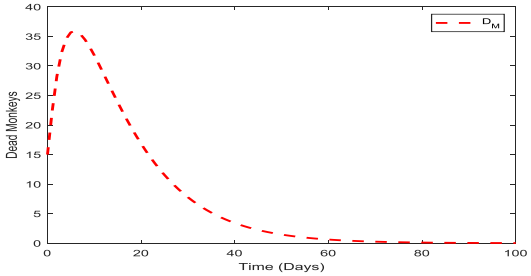

Figure 9. Numerical solution of dead monkeys.

Figure (5) shows that population of recovered humans grows rapidly and then decreases with time, Figure (6) shows that the population of dead humans as a result of Ebola disease increases rapidly and decreases to asymptotic level where they remain constant, In Figure (7), it shows that population of susceptible monkeys (S_M) reduces significantly as a result of infections. In Figure (8) and (9), the population of infected (I_M) and dead (D_M) monkeys begins to rise and later decreases to asymptotic level where they remain constant.

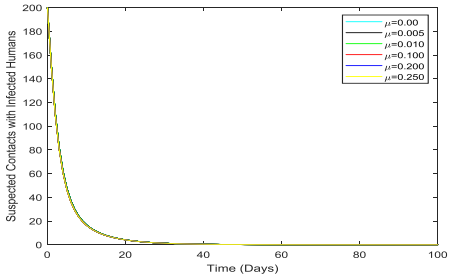

Figure 10. Effects of quarantine rate of infected humans on suspected contact with Humans.

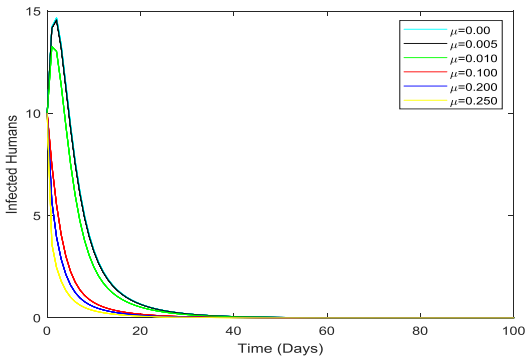

Figure 11: Effect of quarantine rate of infected humans on infested humans

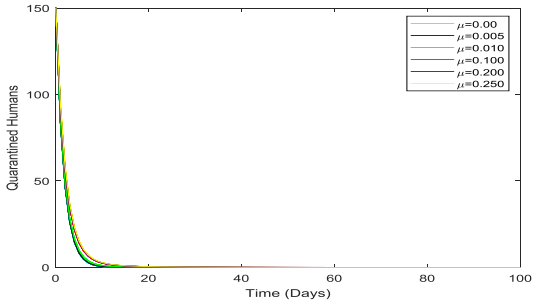

Figure 12. Effect of quarantine rate of infected humans on quarantined humans.

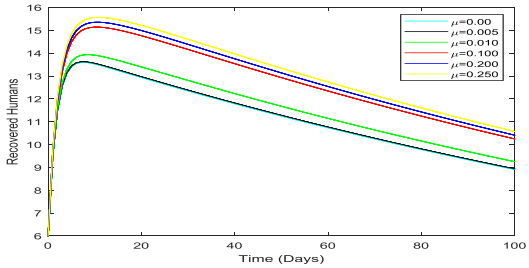

Figure 13. Effects of quarantine rate of infected humans on recovered humans.

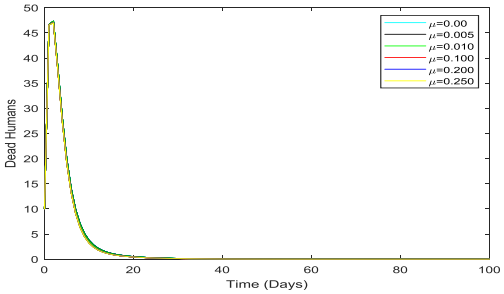

Figure 14: Effects of quarantine rate of infected humans on dead humans.

Figure (10) – (14) shows the effect of progression rate from infectious human to quarantine on susceptible humans $\left(S_H\right)$, infected humans $\left(I_H\right)$, and quarantine humans $\left(Q_H\right)$. It is observed that the populations in these classes continue to decrease as progression rate is increased from 0.00 – 0.250. Figure (4.13) shows the effect of progression rate from infectious human to quarantine on recovered humans $\left(R_H\right)$ which increases the population of the class as progression rate is increased from 0.00 – 0.250. Also, in Figure (14), it can be observed that increase in progression rate from infectious human to quarantine decreases the dead human's $\left(D_H\right)$ population significantly reduced as progression rate increased from 0.00 – 0.250.

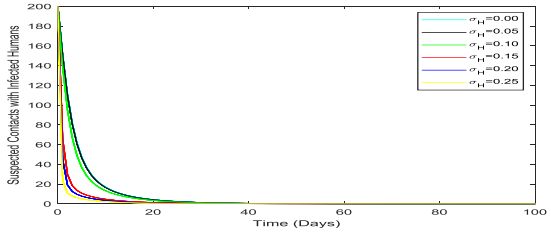

Figure 15: Effects of quarantine rate of suspected contact with infected humans on the suspected contact with infected humans

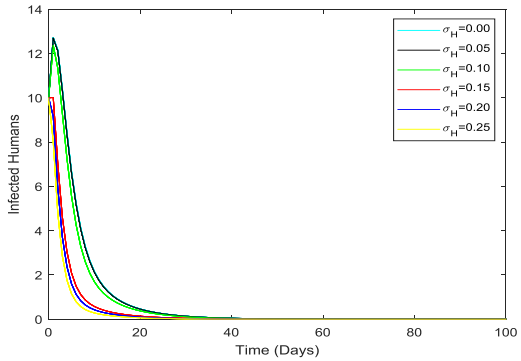

Figure 16: Effects of quarantine rate of suspected contacts with infected humans on infected humans

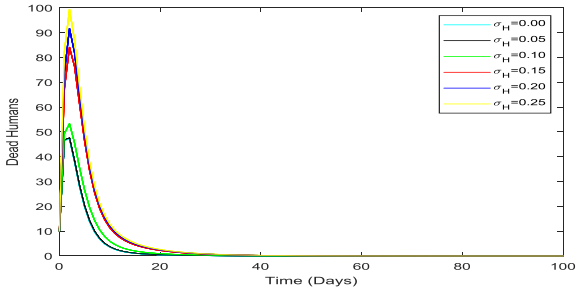

Figure 17: Effects of quarantine rate of suspected contacts with infected humans on dead humans

Figure (15) – (17) shows the effect of progression rate from infectious human to quarantine on susceptible monkeys $\left(S_M\right)$ and infected monkeys $\left(I_M\right)$, it is observed that increase in quarantine rate decreases the population of the class in the present of the diseases. Figure (17) shows the effects of quarantine rate on dead monkeys $\left(D_M\right)$. it can be observed that increase in quarantine rate decreases the dead monkeys population significantly as quarantine rate increases from 0.00 – 0.250.

Ebola virus disease could be eradicated when vital dynamics, contact tracing and quarantine are concurrently put in place as measures of reducing the spread of the disease.

This is affirmed by the work of (Madubueze, Kimbir & Aboiyar, 2018) which suggest that Ebola Virus Disease (EVD) could be eliminated faster when contact tracing and quarantine measures are implemented together.

Conclusion

We modified (Durojaye & Ajie, 2017) Mathematical model for dynamics transmission of Ebola virus disease by adding E_H individual that are suspected to have had contact with infected Human or Monkey, quarantined, and Death human compartment and we split the Monkey compartment into susceptible, infected and Death Monkey compartments. Stability analysis was carried out using Routh-Hurwitz criterion for the local stability while Castillo-chavez conditions were applied to obtain the global stability. The disease free equilibrium points were obtained and our results shows that the equilibrium point of the system is locally asymptotically stable if $R_0 < 1.$ The result of the numerical experiment carried out

indicates that quarantine measures of both E_H individual, infected humans and monkeys can significantly eradicate Ebola Virus from the society

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