MATHEMATICAL MODEL FOR EBOLA VIRUS TRANSMISSION DYNAMICS WITH QUARANTINE COMPARTMENT

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Abstract
We formulated a mathematical model for the dynamics of Ebola virus using a set of ordinary differential equations. We obtained both the disease free and disease endemic equilibria and analysed them for stability. Key to our analysis is the determination of the threshold, Effective Reproductive number (R_c)*. We found that the disease free equilibrium state is asymptotically stable when* $R_c \leq 1$ *, Further, we obtained the conditions for endemic equilibrium state of the model and some numerical results*

Keywords: Stability, Equilibrium State, Quarantine, Exposed, Endemic

Introduction
The first identified case of Ebola was on 26 August 1976, in Yambuku, a small rural village in Mongala District in northern Democratic Republic of the Congo (DCR) then known as Zaire Hewlett (2007). The first victim, and the index case for the disease, was village school headmaster Mabalo Lokela, who had toured an area near the Central African Republic border along the Ebola river between 12–22 August. On 8 September he died of what would become known as the Ebola virus. Subsequently a number of other cases were reported, almost all centered on the Yambuku mission hospital or having close contact with another case. 318 cases and 280 deaths (a 88% fatality rate) occurred in the DRC (King, 2008). The disease is endemic in some West African countries including, Sierra Loene, Liberia, Giunea (WHO, 2014).

Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals. In Africa, infection has been documented through the handling of infected chimpanzees, gorillas, fruit bats, monkeys, forest antelope and porcupines found ill or dead or in the rainforest WHO (2014).
Ebola then spreads in the community through human-to-human transmission, with infection resulting from direct contact (through broken skin or mucous membranes) with the blood, secretions, organs or other bodily fluids of infected people, and indirect contact with environments contaminated with such fluids. Burial ceremonies in which mourners have direct contact with the body of the deceased person can also play a role in the transmission of Ebola. There is evidence that live Ebola virus can be isolated in semenal fliud of convalescent men for 82 days after onset of symptoms (WHO, 2014) hence a pointer to sexsual transmission.

Ebola virus disease (EVD) is a severe acute viral illness often characterized by the sudden onset of fever, intense weakness, muscle pain, headache and sore throat. This is followed by vomiting, diarrhoea, rash, impaired kidney and liver function, and in some cases, both internal

and external bleeding. The incubation period, that is, the time interval from infection with the virus to onset of symptoms, is 2 to 21 days WHO (2014) .
Ebola virus infections can be diagnosed definitively in a laboratory through several types of tests: antibody-capture enzyme-linked immunosorbent assay (ELISA), antigen detection tests, serum neutralization test, reverse transcriptase polymerase chain reaction (RT-PCR) assa, electron microscopy, virus isolation by cell culture, etc. No licensed vaccine for EVD is available. Several vaccines are being tested, but none are available for clinical use, and no specific treatment is available. New drug therapies are being evaluated.

Mathematical model has been an important tool in analyzing the spread and control of infectious diseases. The first of such models was credited to Daniel Bernoulli in 1760, the aim of his model was to evaluate the impact of variolation or inoculation on healthy people with smallpox virus Hethcote (2000). This was followed by Hamer (1906) model on measles, Rose (1911) model on malaria, and the famous Kermack and Mckendrick (1927) SIR model for the transmission of disease in a closed population. The past century has witnessed rapid development of mathematical models to understand the dynamics of infectious diseases.

Abdon &Emile (2014), constructed a model describing the spread of the deadly disease called Ebola hemorrhagic fever. The model was first constructed using the classical derivative and then converted to the generalized version using the beta derivative. He studied in detail the endemic equilibrium points and provided the Eigen values associated using the Jacobian method.

The study showed that, for small portion of infected individuals, the whole country could die out in a very short period of time in case there is no good prevention. Zach (2012) represented the transmission of Ebola virus using a modified Susceptible – Infected – Recovered (SIR) disease model. The model had four compartments, $S(t)$, $I(t)$, $R(t)$, and $D(t)$ representing the Susceptible, Infective, Recovered and the Deceased compartment. The parameters of the model are a the rate of infection, b the rate of recovery, c the rate of Susceptibility, e the rate of death.

Similarly other recent models Gomes (2014), Fisman (2014), Althaus(2014) centered on the estimation of basic reproduction number, a key threshold in disease control but lack other further mathematical analysis

Materials and Methods

Model Formulation

We represented the dynamics of Ebola virus using a set of differential equation. We divided the human population into 5 compartments as shown below:
In formulating our model we made the following assumptions; The infection is from, Infectious and Quarantine individual to Susceptible. The transmissibility from Quarantine Class is weighted using η. The model assumed that individuals move both from only Infectious and Quarantine to Removed class upon recovery from the virus, while Exposed individual move to Infectious class. The researcher assumed that recovered individual remain immune therefore do not go back to Susceptible class.

$$\frac{dS}{dt} = a - \frac{\beta(I + \eta Q)S}{N} - \mu S \qquad (1)$$

The equation (1) above for susceptible population is generated *via* recruitment of humans by birth or immigration into the community at a constant rate a. This population is decreased following infection, which can be acquired *via* contact with member(s) of exposed, infectious or quarantine population at a rate β and by natural death at the rate μ. η is a weight factor for infection from quarantine to susceptible population, this becomes necessary as movement is restricted in the quarantine center.

$$\frac{dE}{dt} = \frac{\beta(I + \eta Q)S}{N} - (\mu + \sigma)E \qquad (2)$$

The equation (2) above for exposed population is generated when there is effective contact between the susceptible and any member of, infectious or quarantine population and the susceptible population. The population is decreased by natural death at the rate μ, and by progression rate to the infectious class at the rate σ

$$\frac{dI}{dt} = \sigma E - (\alpha + \gamma_1 + \mu + \delta)I \qquad (3)$$

The equation (3) above for Infected population is generated as the exposed class develops symptoms and become infectious at the rate σ , the population is decreased by natural death, death due to infection, recovery and migration to quarantine center at the rate $\mu, \delta, \gamma_1,$ and γ_2 respectively.

$$\frac{dQ}{dt} = \alpha I - (\mu + \delta + \gamma_2)Q \qquad (4)$$

The equation (4) above for quarantine population is generated as members of the exposed class migrate to quarantine center at the rate α and is decreased by natural death, death due to infection and as members of the infectious population recover from the virus as a result of treatment at the rate $\mu, \delta, \gamma_2,$ respectively

$$\frac{dR}{dt} = \gamma_1 I + \gamma_2 Q - \mu R \qquad (5)$$

The equation (5) above is generated via recovery from infectious and quarantine population at the rate γ_1, and γ_2 and is reducedby natural death μ.

Thus the dynamics of the ebola virus can be represented by the following set of ordinary differential equation:

$$\frac{dS}{dt} = a - \frac{\beta(I+\eta Q)S}{N} - \mu S$$
$$\frac{dE}{dt} = \frac{\beta(I+\eta Q)S}{N} - (\mu+\sigma)E$$
$$\frac{dI}{dt} = \sigma E - (\alpha+\gamma_1+\mu+\delta)I \quad (6)$$
$$\frac{dQ}{dt} = \alpha I - (\mu+\delta+\gamma_2)Q$$
$$\frac{dR}{dt} = \gamma_1 I + \gamma_2 Q - \mu R$$

Where $S(t), E(t), I(t), Q(t), R(t)$ denote the number of Susceptible, Exposed, Infectious, Quarantine, and Removed population at time t respectively. And

$N(t) = S(t) + E(t) + I(t) + Q(t) + R(t)$

Such that

$$\frac{dN}{dt} = a - \mu N - \delta(I+Q) \quad (7)$$

Parameters of the Model Equation are:

a – recruitment rate which includes birth and immigration

β – the effective contact rate

η – weight for the assumed reduction in transmissibility of Quarantine class

μ – natural mortality rate

σ – progression rate from Exposed to Infectious class

δ – death rate due to Infection

γ_1 – recovery rate from Infectious class

γ_2 – recovery rate from Quarantine class due to treatment

α – the rate at which infected are being quarantined

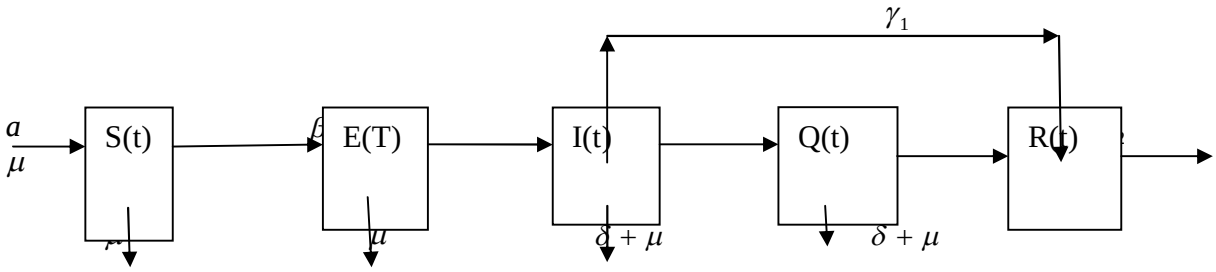


Figure 1: Schematic diagram of the model

Model Analysis

In this section, the basic dynamical features of the model (1) to (5) will be explored. We claim the following:

Lemma 1 *The closed set*

$$D = \left\{ (S,E,I,Q,R) \in \Re_+^5 : S + E + I + Q + R \le \frac{a}{\mu} \right\}$$

is positively invariant and attracting with respect to the basic model equations (1) to (5).

Proof

Adding all the equations of (6) gives

$$\frac{dN}{dt} = a - \mu N - \delta(I + Q) \qquad (7)$$

Since $\frac{dN}{dt} < a - \mu N$, It follows that $\frac{dN}{dt} < 0$ if, $N(t) > \frac{a}{\mu}$

Thus a standard comparison theorem, Lakshmikantham (1989) can be used to show that $N(t) < N(0)e^{-\mu(t)} + \frac{a}{\mu}\left[1 - e^{-\mu(t)}\right]$. In particular, $N(t) \le \frac{a}{\mu}$ if $N(0) \le \frac{a}{\mu}$. Thus, D is positively invariant. Further, if $N(t) > \frac{a}{\mu}$, then either the solution enters D in finite time or $N(t)$ approaches $\frac{a}{\mu}$, and the infected variables I, Q approaches zero. Hence , D is attracting i.e (all solutions $\Re_+^5$ eventually enters D). Thus in D, the basic model (6) is well posed epidemiologically and mathematically, Hethcote (2000). Hence, it is sufficient to study the dynamics of the model equations in D

Existence and Stability of Equilibrium State

The system has two possible equilibria in D where,

$$D = \left\{ (S,E,I,Q,R) \in \Re_+^5 : S + E + I + Q + R \le N \right\}$$

Disease free Equilibrium State

At equilibrium state, the rate of change of the state variables is zero. The disease free equilibrium of our model equations (6) gives

$$E_0 = \left(\frac{a}{\mu}, 0, 0, 0, 0 \right) \qquad (8)$$

The stability of this disease free equilibrium given by equation (8) will be analyzed via the basic reproductive number

The Effective Reproductive Number (R_c)

One of the most important concerns about any infectious disease is its ability to invade a population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of the disease. These models usually have a threshold parameter, known as the basic reproductive number R_c such that when $R_c \le 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $R_c > 1$,then the DFE is unstable and invasion is always possible see Hethcote (1978).

We define the basic reproductive number R_c as the number of secondary infections that one infective individual would create over the duration of the infectious period provided that everyone else is susceptible. Our model is suited for a heterogeneous population in which the vital and epidemiological parameter for an individual may depend on such factors as the stage of the disease, spatial position, etc. however, we assume that the population can be broken into homogeneous subpopulation or compartment such that individual in a given compartment are indistinguishable f rom one another.

We use the next generation matrix approach as described by Driessch and Watmough (2005) to derive our Basic Reproductive Number R_0. Numerous other articles Dietz (1993), Roberts (2003), and Diekmann (1990), are devoted to the calculation of basic reproductive number R_c for different models of various diseases.

Here, the basic reproductive number R_c is the spectral radius (dominant eigenvalue) of the product matrix
FV^{-I}, i.e $R_c = \rho(FV^{-I})$
Our model has three Infective compartments namely the, Exposed E, Infectious I and Quarantine Q compartments. It follows that the matrices F and V for the new infective terms and remaining transfer terms respectively are given below. Where the entries of F and V are partial derivatives of $f_i(x)$ and $v_i(x)$. For our model, F and V are given below.

$$F = \begin{bmatrix} 0 & \frac{\beta}{N} & \frac{\eta\beta}{N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \tag{9}$$

$$V = \begin{bmatrix} -(\mu+\delta) & 0 & \frac{\eta\beta}{N} \\ 0 & -(\alpha+\gamma_1+\mu+\delta) & 0 \\ 0 & \alpha & -(\mu+\gamma_2+\delta) \end{bmatrix} \tag{10}$$

$$R_0 = \frac{\beta\sigma}{(\mu+\delta)(\alpha+\gamma_1+\mu+\delta)(\mu+\gamma+\delta)} \tag{11}$$

Lemma 2. The disease free equilibrium is locally asymptotically stable if
$R_c < 1$

Proof
We prove lemma 2 by showing that the Jacobian matrix of the system (6) have positive eigen values at the disease free equilibrium
The Jacobian Matrix of our model equations (6) is given by

$$J=\begin{bmatrix} \frac{-\beta((I+\eta Q)+\mu)}{N} & 0 & \frac{-\beta S}{N} & \frac{-\eta\beta S}{N} & 0 \\ \frac{\beta(I+\eta Q)}{N} & -(\mu+\delta) & \frac{\beta S}{N} & \frac{\eta\beta S}{N} & 0 \\ 0 & \sigma & -(\alpha+\gamma_1+\mu+\delta) & 0 & 0 \\ 0 & 0 & \alpha & -(\mu+\delta+\gamma_2) & 0 \\ 0 & O & \gamma_1 & \gamma_2 & -\mu \end{bmatrix} \quad (12)$$

At disease free equilibrium the Jacobian matrix becomes

$$J=\begin{bmatrix} -\mu & 0 & \frac{-\beta S^*}{N} & \frac{-\eta\beta S^*}{N} & 0 \\ 0 & -(\mu+\delta) & \frac{\beta S^*}{N} & \frac{\eta\beta S^*}{N} & 0 \\ 0 & \sigma & -(\alpha+\gamma_1+\mu+\delta) & 0 & 0 \\ 0 & 0 & \alpha & -(\mu+\delta+\gamma_2) & 0 \\ 0 & O & \gamma_1 & \gamma_2 & -\mu \end{bmatrix} \quad (13)$$

The characteristic equation of equation (13) is given by

$$J=\begin{bmatrix} -(\mu+\lambda) & 0 & \frac{-\beta S^*}{N} & \frac{-\eta\beta S^*}{N} & 0 \\ 0 & -((\mu+\delta)+\lambda) & \frac{\beta S^*}{N} & \frac{\eta\beta S^*}{N} & 0 \\ 0 & \sigma & -(\alpha+\gamma_1+\mu+\delta+\lambda) & 0 & 0 \\ 0 & 0 & \alpha & -(\mu+\delta+\gamma_2+\lambda) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -(\mu+\lambda) \end{bmatrix} \quad (14)$$

The eigenvalues of equation (14) is given by

$\lambda_1 = -\mu$

$\lambda_2 = -(\mu+\delta)$

$\lambda_3 = -(\alpha+\gamma_1+\mu+\delta)$

$\lambda_4 = -(\mu+\delta+\gamma_2)$

$\lambda_5 = -\mu$

From our evaluations we found that $\lambda_3 < 0$, when $R_c < 1$ where

$$R_0 = \frac{\beta\sigma}{\mu(\mu+\delta)^2(\alpha+\gamma+\mu+\delta)}$$

For local stability of disease free equilibrium, Routh-Hurwitz criteria requires that all eigenvalues have negative real part. Since all the eigenvalues of equation (14) have negative real part, the disease free equilibrium is locally asymptotically stable

Lemma 3. The disease free equilibrium is globally asymptotically stable (GAS)

Proof

Consider the Lyapunov function

$$F = A_3A_2E + A_1A_3I + A_1A_2Q$$

where $A_1 = (\mu + \sigma)$

$A_2 = (\alpha + \gamma_1 + \mu + \delta)$

$A_3 = (\mu + \delta + \gamma_2)$

The Lyapunov derivative is given by (where a dot represents differentiation with respect to time, t) is given by:

$$\dot{F} = A_3 A_2 \dot{E} + A_1 A_3 \dot{I} + A_1 A_2 \dot{Q}$$

$$= A_3 A_2 \left[\left(\frac{\beta I + E + \eta Q}{N} \right) S - A_1 E \right] + A_1 A_3 \left[\sigma E - A_2 I \right] + A_1 A_2 \left[\alpha I - A_3 Q \right]$$

$$= \left[\frac{A_3 A_2 \beta I S}{N} + \frac{A_3 A_2 E S}{N} + \frac{A_3 A_2 \eta Q S}{N} - A_3 A_2 A_1 E \right] + \left[A_1 A_3 \sigma E - A_1 A_3 A_2 I \right] + \left[A_1 A_2 \alpha I - A_1 A_2 A_3 Q \right]$$

$$= \left[\frac{A_3 A_2 S}{N} - A_3 A_2 A_1 + A_1 A_3 \sigma \right] E + \left[\frac{A_3 A_2 \beta S}{N} - A_1 A_3 A_2 + A_1 A_2 \alpha \right] I + \left[\frac{A_3 A_2 \eta S}{N} - A_1 A_2 A_3 \right] Q$$

$$= \frac{A_3 A_2 S}{N} \left\{ (1 - A_3 A_2 A_1 + A_1 A_3 \sigma) E + (\beta - A_1 A_3 A_2 + A_1 A_2 \alpha) I + (\eta - A_1 A_2 A_3) Q \right\}$$

Since

$S \le S^* \le \frac{A_3 A_2}{N} \left\{ (1 - A_3 A_2 A_1 + A_1 A_3 \sigma) R_c E + (\beta - A_1 A_3 A_2 + A_1 A_2 \alpha) R_c I + (\eta - A_1 A_2 A_3) R_c Q \right\}$ Thus, $\dot{F} \le 0$ if $R_0 \le 1$, with $\dot{F} = 0$ if and only if $E = I = Q$. Further, the largest compact invariant set in is the singleton $\{E_0\}$. It follows from Lasalle invariant principle (chapter 2) ,theorem 6.4 of Lasalle (1976) that every solution to the model equations (6) with invariant condition in D converges to the DFE E_0 as $t \to \infty$ that is $E(t), I(t), Q(t) \to (0,0,0)$ as $t \to \infty$. Substituting $E = I = Q = 0$ into equations (2), (3) and (4) gives $S(t) \to S^*$ as $t \to \infty$. Thus, $[S(t), E(t), I(t), Q(t), R(t)] \to [0,0,0]$ as $t \to \infty$ for $R_0 \le 1$, so that the E_0 is GAS in D if $R_0 \le 1$

Existence and Stability of Endemic Equilibrium State

In order to find the endemic equilibrium of the our model equations given by equations (6) i.e. equilibria where at least one of the infected components of the model is non zero, the following steps are taken. We let $E_1 = (S^*, E^*, I^*, Q^*, R^*)$ represent any arbitrary endemic equilibrium of our model equations (6). Further, let

$$\pi = \frac{\beta(I + \eta Q)}{N} \tag{15}$$

Be the force of infection at steady state. Substituting (15) into equations (6) and solving at steady state, we have

$$S^* = \frac{a}{(\pi - \mu)} \tag{16}$$

$$E^* = \frac{a\pi}{(\pi - \mu)(\mu + \delta)} \tag{17}$$

$$I^* = \frac{a\pi\sigma}{(\pi - \mu)(\mu + \delta)(\alpha + \gamma_1 + \mu + \delta)} \tag{18}$$

$$Q^* = \frac{a\pi\sigma\alpha}{(\pi-\mu)(\mu+\delta)(\alpha+\gamma_1+\mu+\delta)(\mu+\delta+\gamma_2)} \quad (19)$$

$$R^* = \frac{a\pi\sigma\gamma_1(\pi-\mu)(\mu+\delta)(\mu+\delta+\gamma_2)A+\gamma_2\alpha a\pi(\pi-\mu)(\mu+\delta)A}{(\pi-\mu)(\mu+\delta)(\mu+\delta+\gamma_2)A^2} \quad (20)$$

where

$$A=(\alpha+\gamma_1+\mu+\delta) \quad (21)$$

Substituting equations (15) to (19) into equation (14) gives

$$\pi = \frac{\beta\left(\frac{a\pi\sigma}{(\pi-\mu)(\mu+\delta)A}+\frac{a\pi}{(\pi-\mu)(\mu+\delta)}+\eta\frac{a\pi\sigma\alpha}{(\pi-\mu)(\mu+\delta)(\mu+\delta+\gamma_2)A}\right)}{N} \quad (22)$$

$$\pi=\beta\left(\frac{a\pi\sigma(\mu+\delta+\gamma_2)+a\pi(\mu+\delta+\gamma_2)A+\eta a\pi\sigma\alpha}{(\pi-\mu)(\mu+\delta)(\mu+\delta+\gamma_2)AN}\right) \quad (23)$$

$$\pi(\pi-\mu)(\mu+\delta)(\mu+\delta+\gamma_2)AN=\beta a\pi\left(\sigma(\mu+\delta+\gamma_2)+(\mu+\delta+\gamma_2)A+\eta\sigma\alpha\right) \quad (24)$$

$$\pi(\pi-\mu)(\mu+\delta)(\mu+\delta+\gamma_2)AN-\beta a\pi\left(\sigma(\mu+\delta+\gamma_2)+(\mu+\delta+\gamma_2)A+\eta\sigma\alpha\right)=0 \quad (25)$$

$$\pi^2-\pi\mu(\mu+\delta)(\mu+\delta+\gamma_2)AN-\beta a\pi\left(\sigma(\mu+\delta+\gamma_2)+(\mu+\delta+\gamma_2)A+\eta\sigma\alpha\right)=0 \quad (26)$$

$$\pi^2-\pi\left\{\mu(\mu+\delta)(\mu+\delta+\gamma_2)AN+\beta a\left(\sigma(\mu+\delta+\gamma_2)+(\mu+\delta+\gamma_2)A+\eta\sigma\alpha\right)\right\}=0 \quad (27)$$

For

$$a_0=1$$

$$b_0=\mu(\mu+\delta)(\mu+\delta+\gamma_2)AN$$

$$c_0=\beta a\left(\sigma(\mu+\delta+\gamma_2)+(\mu+\delta+\gamma_2)A+\eta\sigma\alpha\right)$$

Equation (27) becomes

$$a_0\pi^2-b_0\pi+c_0=0 \quad (28)$$

It can be shown that the non-zero equilibria of the model (6) satisfy the following quadratic (28). Solving the quadratic equation (28) for π and substituting into equations (16) to (20) gives the explicit values of the endemic equilibrium $E_1=\left(S^*,E^*,I,^*Q^*,R^*\right)$. Clearly, the coefficient a_0 of (28), is always positive, and c_0 is positive (negative) if R_c is less than (greater than) unity, respectively. Thus, the following result is established.

Theorem 1 The Ebola model (6) has:

(i) a unique endemic equilibrium if ; $c_0<0\Leftrightarrow R_c>1$

(ii) a unique endemic equilibrium if $b_0<0,$ and $c_0=0$ or $b_0^2-4a_0c_0=0$

(iii) two endemic equilibria if $c_0<0$, $b_0<0,$ and $b_0^2-4a_0c_0>0$

Result and Discussion

Variables and Parameters Values

Table 1: Parameter values and initial conditions

Parameters	Values
a	0.3
β	0.016
η	0.001
μ	0.2
σ	8
δ	0.6
γ_1	0.3
γ_2	0.4
α	2
S	1000
E	0
I	10
Q	0
R	0

Note: The values in table 1 above are hypothetical but however, are in line with some reported cases

Numerical Simulation

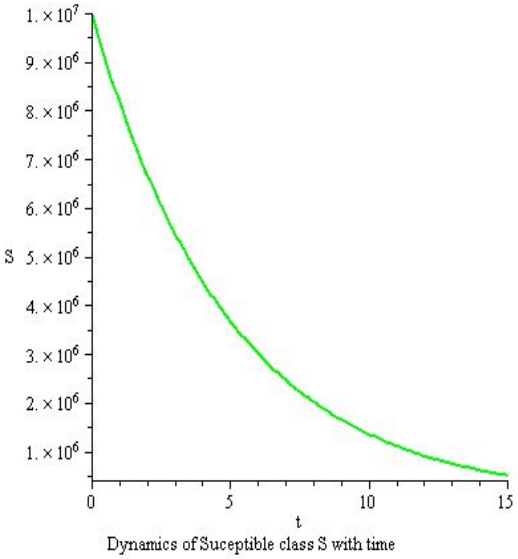


Figure 1:

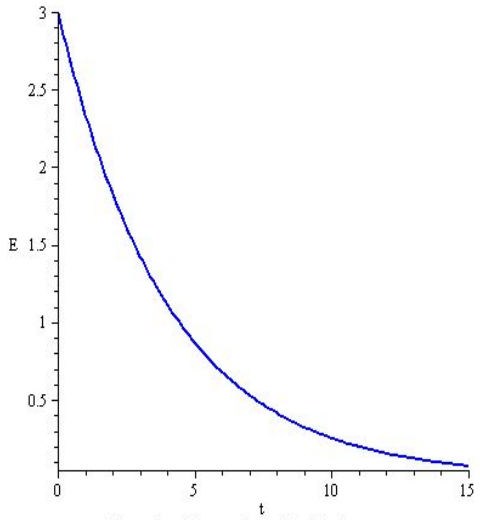


Figure 2:

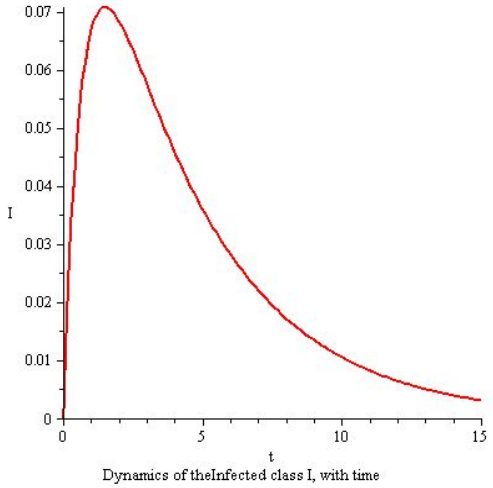
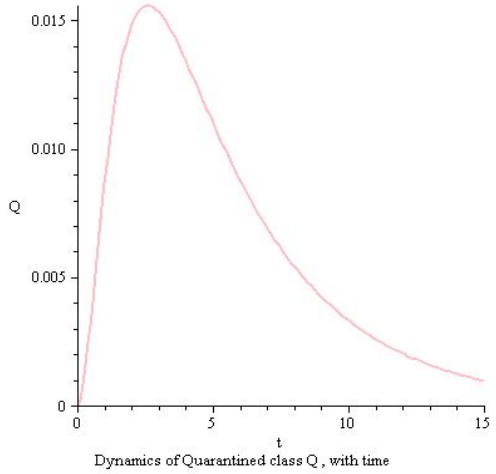


Figure 3:



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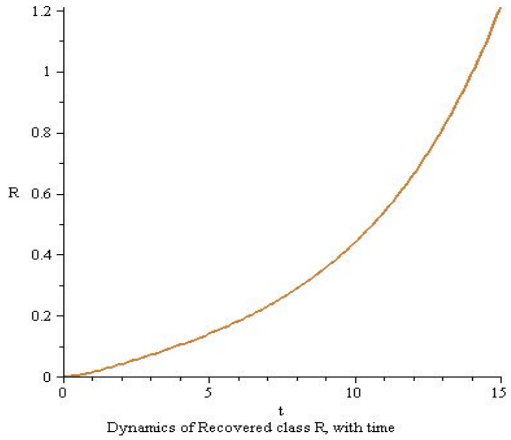

Figure 5:

The parameter values for the graph are as shown in table 1 above.

Discussion

Figures 1 to 5 are numerical simulation of the Ebola virus model given by equations (1) to (5), using the original system variables with parameter values and initial condition as given in table 1 the simulations were conducted using the Runge-Kuta method (rkf45) embedded in Maple 13. The rkf45 method is a fourth-order method, meaning that the local truncation error is on the order of $0(h^5)$, while the total accumulated error is order $0(h^4)$. Figure 1 shows that the susceptible population is on the decrease, this is expected since infection is decreasing parameter of the population. Figure 2 shows that the Exposed class tends to zero within small time, this is in line with epidemiology and our assumption since members either progress to the Infected class or are out of the population due to natural death. Figure 3 shows that the Infected class first increases, then begins to decline as a result of quarantine, recovery, or death due to infection. Figure 4 shows that the quarantine class has similar behavior with the Infected class. Figure 5 shows that the Recovered population is increasing. The figures 1 to 5 confirms the asymptotic stability of the disease free equilibrium.

Conclusion and Recommendation

Conclusion

In this paper a deterministic mathematical model for the dynamics of Ebola Virus was formulated. The model incorporated a Quarantine compartment, and the rate of transmission from the quarantined to the susceptible was weight as a result of restriction of movement imposed on the quarantined territory. We first showed that our model is epidemiologically and mathematically well posed. Further, we obtained both the disease and endemic equilibria and analyzed them for stability. It was established that the disease free equilibrium is both locally and globally stable.

Recommendation

The numerical results show that the susceptible population approaches zero within a short period of time in the absence of control. We therefore recommend high rate of quarantining and tretment which will help stablise the population.The model should be investigated for possibility of backward bifurcation as indicated by case (iii) of theorem 1. We recommend the

inclusion of the vector population to give a comprehensive understanding of the transmission dynamics.

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