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Mathematical modeling, analysis and Markov Chain Monte Carlo simulation of Ebola epidemics



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ABSTRACT

Ebola virus infection is a severe infectious disease with the highest case fatality rate which become the global public health treat now. What makes the disease the worst of all is no specific effective treatment available, its dynamics is not much researched and understood. In this article a new mathematical model incorporating both vaccination and quarantine to study the dynamics of Ebola epidemic has been developed and comprehensively analyzed. The existence as well as uniqueness of the solution to the model is also verified and the basic reproduction number is calculated. Besides, stability conditions are also checked and finally simulation is done using both Euler method and one of the top ten most influential algorithm known as Markov Chain Monte Carlo (MCMC) method. Different rates of vaccination to predict the effect of vaccination on the infected individual over time and that of quarantine are discussed. The results show that quarantine and vaccination are very effective ways to control Ebola epidemic. From our study it was also seen that there is less possibility of an individual for getting Ebola virus for the second time if they survived his/her first infection. Last but not least real data has been fitted to the model, showing that it can used to predict the dynamic of Ebola epidemic.

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Introduction

Ebola is a lethal virus of humans. It is a severe and often deadly illness killing between 50% and 90% of those infected with the virus [9,19,24] named after a river in the Democratic Republic of Congo (formerly Zaire) where it was first identified in 1976 with a high case fatality rate. The disease first came into the lime light in 1976 in Zaire and Sudan [5]. It is a disease of humans and other primates caused by an Ebola virus. Symptoms start two days to three weeks after contacting the virus with a fever, sore throat, muscle pain and headaches [2,3,6,15,17]. Typically, vomiting, diarrhea and rash flow, along with decreased functioning of the liver and kidneys. Around this time, the affected people may begin to bleed within the body and externally. The virus may be acquired upon contact with blood or bodily fluids of an infected people or animal. Spreading through the air has not been documented in the natural

environment. Fruit bats are believed to be a carrier and may spread the virus without being affected [7,10,14,16,20,21,23]. Once human infection occurs, the disease may spread between people, as well. Male survivors may be able to transmit the disease via semen for nearly two months. To make the diagnosis, typically other diseases with similar symptoms such as malaria, cholera and other viral hemorrhagic fevers are first excluded. To confirm the diagnosis, blood samples are tested for viral antibodies, viral RNA, or the virus itself. What makes the disease the worst of all is, no specific effective treatment available. Efforts to help those who are infected are supportive and include giving either oral rehydration therapy (slightly sweet and salty water to drink) or intravenous. As the effective measures for controlling Ebola epidemic is still lack, it needs more attention by medical staffs, epidemiologists, mathematicians and other stake holders.

Mathematical modeling is one of the most important tools in analyzing the epidemiological characteristics of infectious disease and can provide some useful insights about the dynamics of the disease. Various models has been used to study different aspects of Ebola epidemic.

Chowella et al. constructed a mathematical model for Ebola virus disease transmission (Congo 1995 and Uganda 2000) and

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fitted it to historical data in estimation of R_o [8]. Althaus presented a SEIR mathematical model and fitted the model to the reported data of infected cases and deaths for Ebola virus disease in Guinea, Sierra Leone and Liberia [1]. The latest study by A. Rachah and D.F. M Torres recommends inclusion of intervention factors like quarantine procedure in the mathematical model to treat the infected individuals and investigate the effect of vaccination on Ebola virus disease [11].

Besides, according to the World Health Organization (WHO) report 2016, an experimental Ebola vaccine and quarantine was highly protective against the deadly Ebola virus in a major trial [25].

In this work, our goal is to develop a new mathematical model to study the effect of both vaccination and quarantine on the spread of Ebola virus as per the recommendation from World Health organization(WHO). Our model differs from other mathematical models that have been used to study the Ebola epidemics [1,7,8,11,13] in that it incorporates both vaccination and quarantine interventions. In addition, our work differs in that it uses one of the top ten most influential algorithm known as Markov Chain Monte Carlo algorithm to simulate the process as the spread of Ebola virus is a random process. To the best of our knowledge, this is the first integrated simulation method used beside the Euler method for this kind of infectious disease of humans. In Euler method the parameters are regarded constant which may not be true in the practical case. To eliminate such defects we used Monte Carlo method which enable to observe the reality in a better way and see how the Ebola virus transmit in crowd more accurately. In another word, as the states(susceptible, infected, recovered/ removed, death) at time t + 1 depends only on the state at time t (that means our physical state is Markov process). Hence, Monte carlo method is more sensible way to reflect the reality.

The text is organized as follows: In this section we have provided background information about Ebola disease; in Section "M athematical model formulation and description", we developed a basic mathematical model to describe the dynamics of the Ebola virus; in Section "Model parameters", we find parameters with statistical data based on WHO; Section "Basic properties" is with the basic properties of the model; in Section "Analysis of the model" we showed the existence and uniqueness of the solution for the model, derived the basic reproduction number and proved stability conditions. The parameters in Section "Model parameters" were used to simulate the basic model in Section "Numerical simulation" using both Euler method and Monte carlo method. Finally, a conclusion and future work is presented.

Mathematical model formulation and description

A compartmental model with a constant population was used to describe the natural history and epidemiology of Ebola. Briefly, the population is divided into four compartments: Susceptible individuals (S) may become infected (I) after contact with an Ebola infected individuals who are capable of infecting others including nurses, doctors etc at hospitals and with a chance of infecting others before being recovered/removed from the disease (R) or die of Ebola and then join (D).

The susceptible population is increased by the susceptibility of individuals(rate of loss of infection acquired immunity) into the Population at the rate γ_1 . This population will be decreased if acquires infection after contact with infected non quarantined individual at the rate β_1 . As there is a proved possibility of treatment of Ebola by vaccination [4,25,26], the susceptible individuals are further decreased at the rate γ because of vaccination.

The population of infected individuals is generated by the infection of susceptible individuals at the rate β_1 . This population is

decreased by recovering from Ebola disease at the rate of α_1 and α_2 where α_1 is recovery rate of infected quarantined individual and α_2 is recovery rate of infected non quarantined individuals. This population is further decreased by death due to Ebola at a rate δ_1 and δ_2 where δ_1 is death rate of infected quarantined individual and δ_2 is death rate of infected non quarantined individuals due to Ebola. Here it is assumed that α_1 is greater than α_2 and δ_1 is less than δ_2 which is biologically reasonable.

The population of recovered infected individuals is generated by those recovered from Ebola and those individual from susceptible because of vaccination at the rate of γ and decreased by individuals that loss immunity and rejoin the susceptible group at the rate of γ_1 .

Finally, the population of individuals who deceased is generated by individuals who are killed by Ebola. The system of ordinary differential equations describing this model is given below and parameters are defined in Section "Model parameters" (see Fig. 1).

$$\frac{dS}{dt} = \gamma_1 R - \frac{\beta_1 (1 - \beta) S(I)}{N} - \gamma S \tag{2.1}$$

$$\frac{dI}{dt} = \frac{\beta_1(1-\beta)S(I)}{N} - \alpha_1\beta I - \alpha_2(1-\beta)I - \delta_1I - \delta_2I$$
 (2.2)

$$\frac{dR}{dt} = \alpha_1 \beta I + \alpha_2 (1 - \beta) I + \gamma S - \gamma_1 R \tag{2.3}$$

$$\frac{dD}{dt} = \delta_1 I + \delta_2 I \tag{2.4}$$

$$N = S + I + R + D \tag{2.5}$$

Model parameters

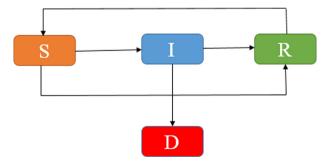


Fig. 1. Compartmental flow of a mathematical model for Ebola epidemics.

Model parameters for simulation.

Parameter	Average value
Total number of population (N)	10,000
Contact rate (β_1)	230
Rate of quarantining (β)	0.3
Rate of loss of infection acquired immunity (γ_1)	0.25
Rate from S to R(Vaccination rate) (γ)	0.15
Recovery rate, quarantined individual (α_1)	0.4
Recovery rate, non quarantined individual (α_2)	0.25
Death rate by Ebola, quarantined individual (δ_1)	0.3
Death rate by Ebola, non quarantined individual (δ_2)	0.75

(Source: World Health Organization, WHO, Ebola 2014).

Basic properties

Since the model monitors changes in the human population, all the variables and parameters are assumed to be positive for all $t\geqslant 0$.

The model is therefore be analyzed in a suitable feasible region: $D = \{S(t), I(t), R(t), D(t) \in R_+^4\}$ with initial conditions $S(0) \ge 0$, $I(0) \ge 0$, $R(0) \ge 0$ and $D(0) \ge 0$ is positively invariant for the system (2.1)–(2.4) above.

Analysis of the model

In order to retain the biological validity of the model, it is important to show that the solutions to the initial value problems exist and unique.

Existence and uniqueness theorems of first order differential equation

Theorem 5.1 (Existence Theorem). Given the general first order ordinary differential equation:

$$y' = F(x, y), y(0) = y_0$$
 (5.1)

If F(x,y) is a continuous function defined in some region $R = \{(x,y): x_0 - \delta < x < x_0 + \delta, y_0 - \epsilon < y < y_0 + \epsilon\}$ containing the point (x_0,y_0) then there exists a number δ_1 (possibly smaller than δ) so that a solution y = f(x) to (5.1) is defined for $x_0 - \delta < x < x_0 + \delta$.

Theorem 5.2 (Uniqueness Theorem). Given the general first order ordinary differential equation:

$$y' = F(x, y), y(0) = y_0$$
 (5.2)

If both F(x,y) and $\frac{\partial F(x,y)}{\partial y}$ are continuous function defined in some region $R=\{(x,y): x_0-\delta < x < x_0+\delta, y_0-\epsilon < y < y_0+\epsilon\}$ containing the point (x_0,y_0) containing the point (x_0,y_0) then there exists a number $\delta_2(\text{possibly smaller than }\delta_1)$ so that a solution y=f(x) to (5.2) is defined for $x_0-\delta_2 < x < x_0+\delta_2$.

Theorem 5.3 (Cauchy-Lipschiz Theorem (Existence and Uniqueness of the solution)). Given an initial value problem:

$$x' = f(x(t), t), x(0) = x_0$$
 (5.3)

Let $U \in R^n$ be an open set and $f: U \times [0,T] \to R^n$ be a continuous function which satisfies the Lipschitz condition: $|f(x_1,t)-f(x_2,t)| \le M|x_1-x_2| \forall (x_1,t), (x_2,t) \in U \times [0,T]$ where M is a given constant. If $x_0 \in U$ then for some positive δ there is a unique solution $x:[0,\delta] \to U$ of the initial value problem.

Theorem 5.4. The model from (2.1) to (2.4) above is continuous and satisfies Cauchy-Lipschiz Theorem (5.3).

Proof. From model (2.1)–(2.4) we have:

$$\frac{dS}{dt} = \gamma_1 R - \frac{\beta_1 (1 - \beta) S(I)}{N} - \gamma S$$

Let $\frac{dS}{dt} = F(t,S) = \gamma_1 R - \frac{\beta_1(1-\beta)S(I)}{N} - \gamma S$ and $\frac{\partial F(t,S)}{\partial S} = -\frac{\beta_1(1-\beta)(I)}{N} - \gamma S$. Here both the function F(t,S) and its partial derivatives $\frac{\partial F(t,S)}{\partial S}$ are defined and continuous at all points (t,S). Therefore by theorems (5.1) and (5.2) above there exists a unique solution in some open interval centered at t_0 for this differential equation.

Similarly, consider the second differential equation in our model (2.1)–(2.4) above.

$$\frac{dI}{dt} = \frac{\beta_1(1-\beta)S(I)}{N} - \alpha_1\beta I - \alpha_2(1-\beta)I - \delta_1I - \delta_2I \text{ and.}$$

Let $G(t,I)=\frac{dI}{dt}=\frac{\beta_1(1-\beta)S(I)}{N}-\alpha_1\beta I-\alpha_2(1-\beta)I-\delta_1I-\delta_2I$ and $\frac{\partial G(t,I)}{\partial I}=\frac{\beta_1(1-\beta)S}{N}-\alpha_1\beta I-\alpha_2(1-\beta)-\delta_1-\delta_2$. Here both the function G(t,I) and its partial derivatives $\frac{\partial G(t,I)}{\partial I}$ are defined and continuous at all points (t,I). Therefore by theorems (5.1) and (5.2) above there exists a unique solution in some open interval centered at t_0 for this differential equation.

By analogous style one can show that the remaining two differential equations in the system also satisfy these conditions.

Next let us show if the Lipschitz condition is satisfied:

Consider the first order differential equation in our model:

$$\begin{split} \frac{dS}{dt} &= \gamma_1 R - \frac{\beta_1 (1-\beta)S(I)}{N} - \gamma S \\ \text{Let } \frac{dS}{dt} &= F(S,t) = \gamma_1 R - \frac{\beta_1 (1-\beta)S(I)}{N} - \gamma S \text{ then} \end{split}$$

$$\begin{split} |F(S_1,t)-F(S_2,t)| &= \left| (\gamma_1 R - \frac{\beta_1 (1-\beta) S_1(I)}{N} - \gamma S_1) - (\gamma_1 R - \frac{\beta_1 (1-\beta) S_2(I)}{N} - \gamma S_2) \right| \\ |F(S_1,t)-F(S_2,t)| &= \left| \gamma_1 R - \frac{\beta_1 (1-\beta) S_1(I)}{N} - \gamma_1 R + \frac{\beta_1 (1-\beta) S_2(I)}{N} + \gamma S_2 \right| \\ |F(S_1,t)-F(S_2,t)| &= \left| (-1) \left[\left(\frac{\beta_1 (1-\beta)}{N} + \gamma \right) s_1 - \left(\frac{\beta_1 (1-\beta)}{N} + \gamma \right) s_2 \right] \right| \\ |F(S_1,t)-F(S_2,t)| &\leq \left(\frac{\beta_1}{N} + \gamma \right) |S_1 - S_2| \\ |F(S_1,t)-F(S_2,t)| &\leq M |S_1 - S_2| \text{ where } M = \left(\frac{\beta_1}{N} + \gamma \right) \end{split}$$

Therefore, $|F(S_1,t) - F(S_2,t)| = M|S_1 - S_2|$ in the first differential equation of our model. Once again consider the second differential equation of the model:

$$\begin{split} &\frac{dI}{dt} = \frac{\beta_1(1-\beta)S(I)}{N} - \alpha_1\beta I - \alpha_2(1-\beta)I - \delta_1I - \delta_2I \text{ and.} \\ &\text{Let } G(I,t) = \frac{\beta_1(1-\beta)S(I)}{N} - \alpha_1\beta I - \alpha_2(1-\beta)I - \delta_1I - \delta_2I \text{ then} \\ &|G(I_1,t) - G(I_2,t)| = \left| \left(\frac{\beta_1(1-\beta)S}{N} - \alpha_1\beta - \alpha_2(1-\beta) - \delta_1 - \delta_2 \right) (I_1 - I_2) \right| \\ &|G(I_1,t) - G(I_2,t)| \leqslant \left(\frac{\beta_1(1-\beta)S}{N} - \alpha_1\beta - \alpha_2(1-\beta) - \delta_1 - \delta_2 \right) |(I_1-I_2)| \\ &|G(I_1,t) - G(I_2,t)| \leqslant M^* |I_1 - I_2|, M^* = \left| \left(\frac{\beta_1(1-\beta)S}{N} - \alpha_1\beta - \alpha_2(1-\beta) - \delta_1 - \delta_2 \right) \right| \end{split}$$

Therefore, $|G(I_1,t)-G(I_2,t)| \le M^*|I_1-I_2|$ in the second differential equation of our model. In a similar way it can be shown that the remaining differential equations of the model satisfies the Lipschitz condition. Therefore we conclude that there exists a unique solutions S(t), I(t), R(t), D(t) for all t > 0.

Existence of the disease free equilibrium state, E_0

At the disease free equilibrium state we have absence of infection. Thus, all the Ebola infected classes will be zero and the entire population will comprise of only Ebola free, susceptible individuals. A disease free equilibrium state of the model above is unique and exists at the point E_0 =(S^* , I^* , I^* , I^*).

Equating the model to zero and solving we get: $E_0 = (S^*, I^*, R^*, D^*) = (N - S_0, 0, 0, S_0) \ \forall S_0 \in R \cap [0, N].$

The basic reproduction number

The basic reproduction number, R_0 of the system (2.1)–(2.4) can be obtained by using the next generation matrix method formulated in [18].

As our population is closed, let $X = (I,R)^T$ then $\frac{dX}{dt} = f(x) - \nu(x)$ where:

$$f(x) = \begin{pmatrix} \frac{\beta_1(1-\beta)SI}{N} \\ \alpha_1\beta I + \alpha_2(1-\beta)I + \gamma S \end{pmatrix}$$
 (5.4)

and

$$v(x) = \begin{pmatrix} (\alpha_1 \beta I + \alpha_2 (1 - \beta)I + \delta_1 I + \delta_2 I) \\ \gamma_1 R \end{pmatrix}$$
 (5.5)

The jacobian matrices of f(x) and v(x) evaluated at the disease free equilibrium, E_0 are:

$$Df(E_0) = F = \begin{pmatrix} \frac{\beta_1(1-\beta)(N-S_0)}{N} & 0\\ \alpha_1\beta I + \alpha_2(1-\beta) & 0 \end{pmatrix}$$
 (5.6)

and

$$D\nu(E_0) = V = \begin{pmatrix} \alpha_1\beta + \alpha_2(1-\beta) + \delta_1 + \delta_2 & 0\\ 0 & \gamma_1 \end{pmatrix} \tag{5.7}$$

The model reproduction number, denoted by R_0 is thus given by:

$$R_0 = \frac{\beta_1(1-\beta)\alpha_1(N-S_0)}{N\gamma_1(\alpha_1\beta+\alpha_2(1-\beta)+\delta_1+\delta_2)}$$

Stability conditions

Theorem 5.5. The disease free equilibrium E_0 is locally asymptotically stable for $R_0 < 1$ and unstable otherwise.

Proof. To prove the local stability of the disease free equilibrium, we used the jacobian stability method. If the Eigenvalues of (F-V) has a negative real parts then the disease free equilibrium is locally stable. Using F and V from Eqs. (5.6) and (5.7):

$$F - V = \begin{pmatrix} \frac{\beta_1(1-\beta)(N-S_0)}{N} - \alpha_1\beta - \alpha_2(1-\beta) - \delta_1 - \delta_2 & 0\\ \alpha_1\beta + \alpha_2(1-\beta) & -\gamma_1 \end{pmatrix}$$
 (5.8)

Using characteristic equation $|(F-V)-\lambda I|=0$, the following equation is obtained.

 $(\lambda+\gamma_1)(\lambda+\alpha_1\beta+\alpha_2(1-\beta)+\delta_1+\delta_2+\beta(\beta-1)\frac{(N-S_0)}{N})=0 \label{eq:lambda}$ is the eigenvalues in this case. After solving this we see that both the eigenvalues are negative for $R_0<1$. Besides, the product of the coefficient of λ^2 and the coefficient of λ is greater than the constant term for $R_0<1$. Therefore, for $R_0<1$ the disease free equilibrium is locally asymptotically stable. \qed

Theorem 5.6. For system (2.1) to (2.4), the disease free equilibrium is globally asymptotically stable if $R_0 < 1$

Proof. First let us find jacobian matrices (of order 3) evaluated at the disease free equilibrium (F') and (V'). They are given below:

$$Df(E_0) = F' = \begin{pmatrix} \frac{\beta_1(1-\beta)(N-S_0)}{N} & 0 & 0\\ \alpha_1\beta + \alpha_2(1-\beta) & 0 & 0\\ 0 & 0 & 0 \end{pmatrix}$$
 (5.9)

and

$$D\nu(E_0) = V' = \begin{pmatrix} \alpha_1 \beta + \alpha_2 (1 - \beta) + \delta_1 + \delta_2 & 0 & 0 \\ 0 & \gamma_1 & 0 \\ -\delta_1 - \delta_2 & 0 & 0 \end{pmatrix}$$
 (5.10)

To prove comparison theorem was used. The rate of change of the variables (I, R, D) of the system above can be re-written as:

$$\begin{pmatrix} \frac{dI}{dt} \\ \frac{dR}{dt} \\ \frac{dP}{dt} \end{pmatrix} = (F' - V') \begin{pmatrix} I \\ R \\ D \end{pmatrix} - \left(1 - \frac{S}{N - S_0}\right) \begin{pmatrix} \frac{\beta_1(1 - \beta)(N - S_0)}{N} & 0 & 0 \\ \alpha_1 \beta + \alpha_2(1 - \beta) & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} I \\ R \\ D \end{pmatrix}$$

where (F') and (V') are jacobian matrices (of order 3) evaluated at the disease free equilibrium. Clearly,

$$\begin{pmatrix} \frac{dI}{dt} \\ \frac{dR}{dt} \\ \frac{dD}{dt} \end{pmatrix} \leqslant (F' - V') \begin{pmatrix} I \\ R \\ D \end{pmatrix}$$
 (5.11)

Since, the eigenvalues of the matrix (F'-V') have negative real parts (this comes from the stability results in Lemma 1 in [12,18] then the system (2.1)–(2.4) is stable whenever $R_0 < 1$. So $(I,D,R) \to (0,0,0)$ and $S \to N - S_0$ as $t \to \infty$. By the comparison theorem [19,22] $(I,R,D) \to E_0$ as $t \to \infty$. Therefore, E_0 is globally asymptotically stable. \square

Numerical simulation

Numerical simulation using Euler method

Experiment 1

To approximate the solutions of the model built above, we give some simulations using the parameters values of Table 1 in Section "Model parameters" above using Euler method. The result is given below.

From Fig. 2 (above) we see that the population of susceptible individual immediately begins to drop because of the high degree of how infectious the Ebola virus is. Consequently, the population of the dead people starts rising.

Simulation using Monte Carlo method

Monte Carlo simulations are used to model the probability of different outcomes in a process that cannot easily be predicted due the intervention of random variables. As the spread of Ebola virus is a random process the Monte carlo algorithm is used to simulate the Markov Chain process of which the transfer matrix changes over time. In a Markov Chain process the physical state at time t+1 depends only on the state at time t. In other words, for random variables $\{x_t\}$, t=0,1,2,3...

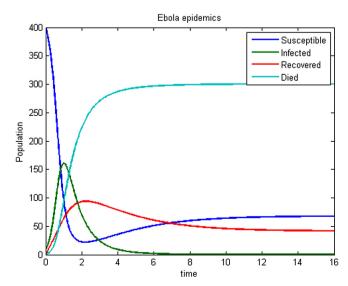


Fig. 2. Simulation result using Euler method.

$$P(X_t = j/x_0 = i_0, x_1 = i_1, \dots, x_{t-1} = i_{t-1}) = P(x_t = j/x_{t-1} = i_{t-1})$$
(6.1)

Define the state matrix as: X(t) = (S(t), I(t), R(t), D(t)) to represent the compartments in a population. Then, the initial state matrix X(0) is obtained as: $X(0) = (1 - I_0, I_0, 0, 0)$. According to Markov Chain theory the transition matrix can be given as: $p(t) = \{P(i,j)\}_{4x4}$ where P(i,j) is the transition probability from state i to state j for i, j an element of $\{1, 2, 3, 4\}$.

$$P(t) = \begin{pmatrix} \textit{Variables} & S & I & R & D \\ S & P(1,1) & P(1,2) & P(1,3) & P(1,4) \\ I & P(2,1) & P(2,2) & P(2,3) & P(2,4) \\ R & P(3,1) & P(3,2) & P(3,3) & P(3,4) \\ D & P(4,1) & P(4,2) & P(4,3) & P(4,4) \end{pmatrix} \tag{6.2}$$

Besides, P(1,4)=P(2,1)=P(3,2)=P(3,4)=P(4,1)=P(4,2)=P(4,3)=0 as there is no transition and P(1,1)+P(1,2)+P(1,3)=P(2,2)+P(2,3)+P(2,4)=P(3,1)+P(3,3)=P(4,4)=1.

Finally, the state matrix is given by:

$$X(t) = X(0) \prod_{t=1} P(t)$$

In experiment 1 above we regard the constant parameters and ignored the influence of latent period. To eliminate this defect Monte Carlo method is used. Figure below shows the Monte carlo simulation of the process under the conditions given above over time.

Experiment 2

From Fig. 3 above we clearly see that the number of Ebola infected individuals is increased and then come to decrease. At the same time the population of those who die by Ebola rises swiftly and reaches the peak showing the biological reality that Ebola is fatal. The model is more realistic to show the situation. Therefore, the medical, health departments and other stake holders should focus on this moment. Moreover, the population of the susceptible also decrease at this time as more people get infected showing that the spread of Ebola is high unless controlled.

Experiment 3

Here the experiment deals with the relation between quarantine and the population of Ebola infected individuals.

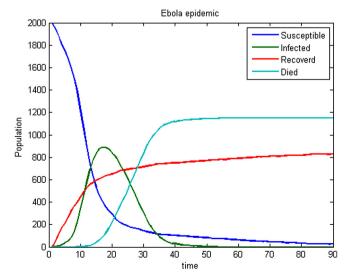


Fig. 3. Simulation result using Monte Carlo method.

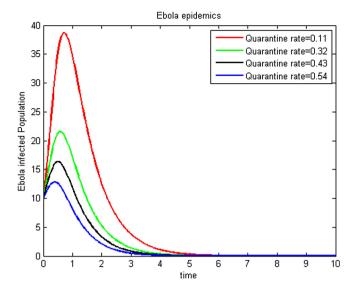


Fig. 4. Effect of rate of infected quarantine on infected population.

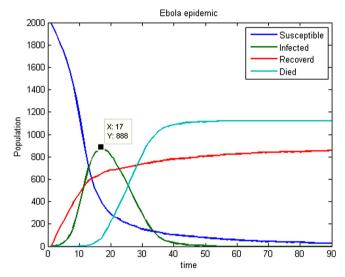


Fig. 5. Markov Chain Monte Carlo simulation when vaccination rate, $\gamma=0$ (without vaccination).

From Fig. 4 above we see the effect of rate of infected quarantine on the Ebola infected population. It is clearly seen that when the rate of infected quarantine increases, the population of Ebola infected individuals decreases.

Experiment 4

when vaccination rate, $\gamma=0$ (without vaccination) the result is given below.

Experiment 5

(Simulation result when vaccination rate is increased to $\gamma=0.3$).

Experiment 6

In this experiment the vaccination rate is more increased than the previous two experiments conducted.

Note: From Figs. 5–7(Experiment 4, 5 and 6) above, we see the effect of rate of vaccination on the Ebola infected population for $\gamma=0,\gamma=0.3$ and $\gamma=0.5$. when the rate of vaccination increases,

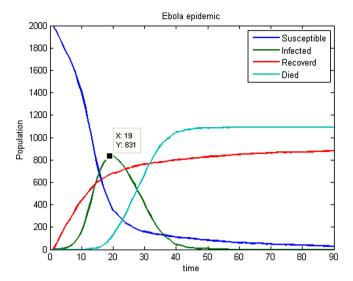


Fig. 6. Markov Chain Monte Carlo simulation when vaccination rate, $\gamma = 0.3$.

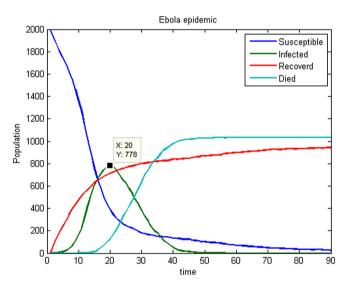


Fig. 7. Markov Chain Monte Carlo simulation when vaccination rate, $\gamma = 0.5$.

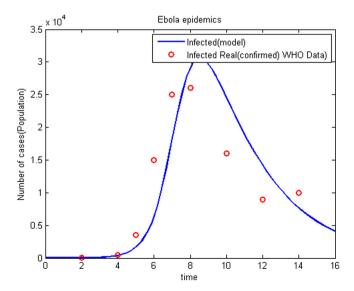


Fig. 8. Ebola Infected I(t) versus the real data of confirmed cases for 2014 Ebola outbreak occurred in Liberia.

the number of Ebola infected individuals reduces from 888 to 778. It is clearly seen that when the rate of vaccination increases, the population of Ebola infected individuals decreases.

Experiment 7 (See Fig. 8).

Conclusion

In overall, the dynamical behavior of the formulated Ebola epidemic model is investigated which plays a vital role in controlling the spread of Ebola virus. Our new model has the detail about all compartments and we found it fits well the data of confirmed cases provided by WHO for the Ebola outbreak in West Africa. The parameter values used are all the latest values. To secure more realistic approach we used two different simulations methods: Euler and Monte carlo method. As the spread of Ebola virus is a random process, Monte carlo algorithm is used to simulate the Markov Chain process of which the transfer matrix changes over time. From the point of view of our result of Markov Chain Monte carlo simulation, we claim that there is less possibility of an individual for getting Ebola virus for the second time if they survived the first infection. None of the previous researchers discovered weather a person can re catch Ebola or not if they survived the first case. Moreover, from our experimental results we also see that Ebola is really fatal and spreads swiftly Which means a regulation that reflects the reality very well is obtained and the model works better as well efficient for the Ebola outbreak in west Africa.

Once again, from our experimental results we see that though Ebola spreads swiftly, it can be controlled upon increasing vaccination. Vaccination is a very efficient method in reducing the number of Ebola infected individuals in a short period of time and increases the number of recovered individuals. Increasing rate of infected quarantine is also another efficient method to control Ebola epidemic as seen from our study. Hence, Vaccination and isolation of the Ebola patient and providing great treatment are highly the crucial measures to control the Ebola epidemics. Besides, as the cost of vaccination might be high for Ebola infected countries, we recommend an optimal control to reduce the cost and number of infected individual. Moreover, in order to prevent Ebola epidemics, through the analysis of the model the government must strictly manage the policy on Ebola and carry it out. This in turn helps for health campaigning and raising health literacy which plays a role to control the quick spread of the disease. We finally strongly believe that our study will play its own role in the current effort of controlling the Ebola outbreak in West Africa.

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