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The stability analysis and control transmission of mathematical model for Ebola Virus

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Abstract: Mathematical modeling of infectious diseases has progressed dramatically over the past four decades and continues to flourish at the nexus of mathematics, epidemiology, and infectious diseases research. Now recognized as a valuable tool, mathematical models are being integrated into the public health decision-making process more than ever before. In this article, a mathematical model of Ebola virus which is named as SEIVR (susceptible, exposed, infected, vaccinated, recovered) model is considered. First, we formulate the model and present the basic properties of the proposed model. Then, basic reproductive number is obtained by using the next-generation matrix approach. Furthermore, the sensitivity analysis of R_0 is also discussed, all the endemic equilibrium points related to the disease are derived, a condition to investigate all possible equilibria of the model in terms of the basic reproduction number is obtained. In last, numerical simulation is presented with and without vaccination or control for the proposed model.

Keywords: Ebola virus, sensitivity analysis, reproduction number, formulation of model, endemic equilibrium points, local stability, global stability, numerical simulation through Matlab programming.

MSC: 30C45, 30C50, 30C55.

1. Introduction

24 July 2018 marks the end of the ninth outbreak of Ebola in the Democratic Republic of the Congo(DRC). The World Health Organization(WHO) congratulates the country and all those involved in ending the outbreak while urging them to extend this success to combatting other diseases in DRC. The recent Ebola virus (EBOV) epidemic in West Africa emerged around the end of 2013 in the prefecture of Guckdou in Guinea [1] and caused at least 11,310 deaths among 28,616 recorded cases in Guinea, Sierra Leone, and Liberia [2]. It has been argued that the West African EBOV epidemic illustrated problems in the early detection of, and rapid response to, infectious disease outbreaks of public health importance. For the past decade, researches have been conducted in laboratories to better understand the biology and potential therapies of Ebola virus (EBOV) [3]. However, field-based research in high risk populations such as impoverished villages much progress has not been accomplished. For instance, there have been outbreaks in the Democratic Republic of Congo in 2007, 2008 and in Uganda in 2007 [4].

Mathematical models have been used extensively to study the dynamics of EBOV transmission [5]. Tahir *et al.* [6] presented mathematical model for the Ebola virus. A similar mathematical model was presented recently in [7]. Another recent mathematical model on the Ebola virus was studied in [8]. Ebola virus is one of the four ebolaviruses known to cause disease in humans. It has the highest case-fatality rate of these ebolaviruses, averaging 83% since the first outbreaks in 1976, although fatality rates up to 90% have been recorded in one outbreak (200203). There have also been more outbreaks of the Ebola virus than of any other ebolavirus. In 1976 the first Ebola virus was found of the Marburg virus [9,10]. In the mean while another team found Ebola virus, from Ebola River where this river was first considered to be near to the Republic of the Congo [9–11]. A mathematical model Prevention strategy for superinfection mathematical model tuberculosis and HIV associated with AIDS was recently presented [12].

The name Ebola virus is derived from the Ebola River, a river that was at first thought to be in close proximity to the area in the Democratic Republic of Congo. The incubation period, that is, the time interval from infection with the virus symptoms is 2 to 21 days. Humans are not infectious until they develop symptoms. The family of the related virus included (1) Cuevavirus,(2) Marburgvirus, and (3) Ebolavirus. Majority of human death occurred by Ebola virus and in West Africa and it becomes epidemic in 2013 to 2015 [13]. Some cases reported out from West Africa, all infected are foreign travelers who exposed to affected regions while later they showed Ebola fever symptoms when reached to destinations [14]. In this period the virus caused near about 286,16 are suspected while 113,10 exact and confirmed deaths cases [15]. The Ebola virus spread in many countries, which start in Guinea and move across Liberia and Sierra Leone.

The Ebola virus also spreads by the human to human contacts like secretions, blood, body fluids of the infected individuals, surfaces and the materials of infected (that is) cloth and bedding. The virus causes serious acute illness and becomes fatal if the patient takes no treatment. The Ebola virus causes an acute, serious illness which is often fatal if untreated. Pathogen genome sequencing is also being used to assist with the identification of unknown infection sources and transmission chains, as pathogen genomes contain valuable information that complements contact tracing efforts. In the case of Ebola, Arias *et al.* [4] demonstrated that rapid outbreak sequencing in locally established sequencing facilities can identify transmission chains linked to sporadic cases. In addition to identifying specific transmission pathways, pathogen genome analysis can also shed light on the origins, evolution and transmission dynamics of a pathogen during an epidemic [16]. Early in the EBOV epidemic analysis such as those by Gire *et al.*[17] demonstrated that the virus entered the human population in the late 2013 and crossed from Guinea to Sierra Leone in May 2014 through the sustained human-to-human transmission.

In this article, we directed as follows: In Section 2, the model formulation has been illustrated. In Section 3, the reproductive number is derived, and its sensitivity analysis is given in the Section 4. Next endemic equilibrium points are derived and the local stability analysis is shown stable at disease free, as well as, at endemic equilibrium in Section 5. Further we derived the global stability of the model with the help of Lyapunov function at disease free, and at endemic equilibrium in Section 6. Finally, we have shown numerically result by RK_4 method and Matlab programming in Section 7 and conclude our paper in Section 8.

2. Model formulation and method

Ebola SEIVR (susceptible, exposed, infected, vaccinated, recovered) mathematical model [18,19] is defined as:

$$\left.\begin{array}{c} S^{\bullet} = \frac{1}{2}\psi - \xi S - \frac{1}{\psi}\lambda SE - \epsilon_1 SI - \epsilon_2 SI, \\ E^{\bullet} = \lambda SE - \omega EI - (\mu_1 + \mu_2)E, \\ I^{\bullet} = \omega EI + \epsilon_1 SI + \epsilon_2 SI - \frac{1}{\xi}\eta IR - (\phi_1 + \phi_2)I, \\ V^{\bullet} = (\phi_1 + \phi_2)I - \zeta V, \\ R^{\bullet} = \frac{1}{\xi}\eta I - \delta R. \end{array}\right\} \tag{1}$$

along with the following initial conditions:

$$[S(0), E(0), I(0), V(0), R(0)] \geq 0.$$

Here, S represent susceptible individuals, E shows exposed individuals, I represent infected V represent the vaccinated individuals, R represent recovered individuals, ψ and ξ represent new birth rate and death rate in susceptible individuals, ϵ_1 represent infection transmission rate from susceptible to infected individuals through wild animals infection, and ϵ_2 represent the infection transmission rate from susceptible individual to infected individuals through domestic animals, λ represent infection transmission rate from susceptible to exposed individuals, ω represent infection transmission rate from exposed individual to infected individuals, η represented the rate of recovery in recovered individuals, μ_1 and μ_2 represent natural death rate and infectious death rate in exposed individuals, ϕ_1 and ϕ_2 representing natural and infectious death rate of infected individuals respectively. We represent the total population of the model (1) as below,

$$B(t) = S + E + I + V + R,$$

which will be written as,

$$\frac{dB(t)}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dV}{dt} + \frac{dR}{dt}.$$

Using values from model (1), we get the following result

$$\frac{dB(t)}{dt} = \psi - \xi S - (\mu_1 + \mu_2)E - \eta IR - (\phi_1 + \phi_2)I + \eta I - \zeta V - \delta R. \tag{2}$$

From Equation (2), we have

$$\frac{dB(t)}{dt} \leq \psi - \xi S.$$

Clearly

$$\lim_{t\to\infty} supB \leq \frac{\psi}{\xi}. \tag{3}$$

For the study of biological purpose, the feasible and sufficient region for model (1) is denoted by $\Re$ and defined as:

$$\Re = \left\{ (S, E, I, V, R) \in R_+^5, B \leq \frac{\psi}{\xi} \right\}. \tag{4}$$

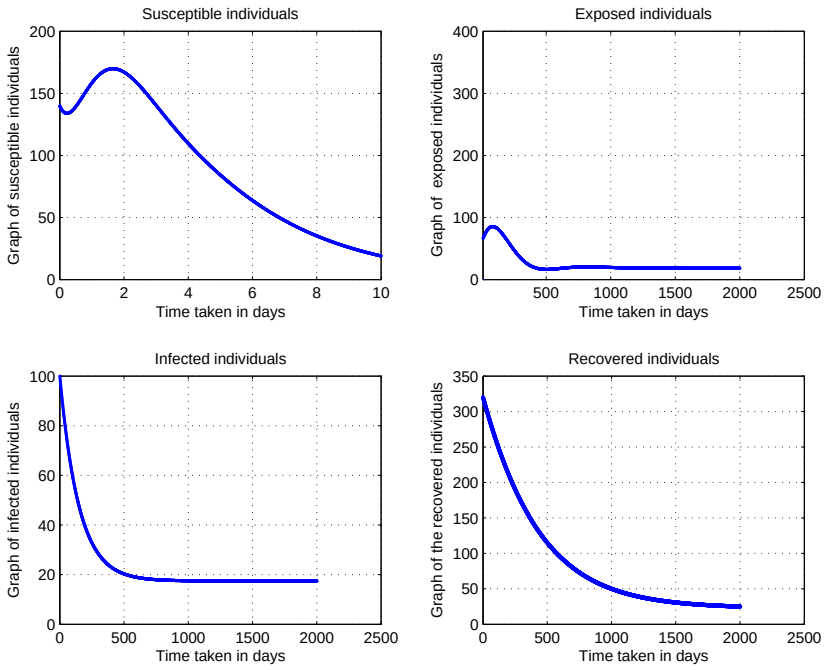

Figure 1. The plot shows the Ebola virus behavior.

3. R_0-the reproduction number of model

In many epidemiological models, the basic reproduction number is one of the key values that can predict whether the infectious disease will spread into a population or die out. The basic reproduction number is the average rate of secondary infectious cases when one infectious individual is introduced in a susceptible population. In this section we used the concept of next generation matrix method which was developed by Driessche *et al.* [20]. For this we divide the system into infectious class "F" and non infectious class "V" as under,

$$F = \begin{bmatrix} \lambda SE - \omega EI \\ \omega EI + \epsilon_1 SI + \epsilon_2 SI - \eta IR \end{bmatrix},$$

and

$$V = \begin{bmatrix} (\mu_1 + \mu_2)E \\ (\phi_1 + \phi_2)I \end{bmatrix}.$$

Now to find Jacobian $\overline{F}$ and $\overline{V}$, we processed as:

$$\overline{F} = \begin{bmatrix} \lambda S - \omega I & -\omega E \\ \omega I & \omega E + \epsilon_1 S + \epsilon_2 S - \eta R \end{bmatrix}.$$

$$\overline{V} = \begin{bmatrix} \mu_1 + \mu_2 & 0 \\ 0 & \phi_1 + \phi_2 \end{bmatrix}.$$

Therefore, the reproductive number R_0 of our model (1) is given as:

$$R_0 = \frac{(\epsilon_1 + \epsilon_2)\psi}{\xi(\phi_1 + \phi_2)}. \tag{5}$$

Table 1. Sensitivity Analysis of Chosen Parameters R_0

Parameter	Sensitivity Index	Value
New rate	S_ψ	+1.0001
Ebola treatment rate	S_ξ	+0.8087
Rate through class change	S_{ϵ_2}	-0.7687
Exposed individuals treatment	S_λ	-0.7761
Modified parameter	S_ω	+0.1245
Rate throuhg individual left class	S_ξ	-0.9011
Modified parameter	S_ψ	+0.8315

The Table 1 shows that their are two influences parameters involve on the rate of reproductive number, i.e, positive and negative. In addition, ψ, ξ, ϵ_2 and ω have positive influences while ϵ_1, λ and η have negative effect on the rate of reproductive number. From this we describe that, increasing or decreasing 10% will increase or decrease the rate of reproductive number 10%, 8.087%, 1.245% and 8.315% are given in Figures showing different images of reproductive number R_0 and 6. On the other side, we see that the parameters index by, ω, ϵ_2 and ξ describe that increase its values 10% should decrease it 10% reproductive number upto 7.687, 7.761 and 9.011 given in Figures 2, 3 and 4.

Now, to control Ebola infection, we need to focus on parameter ψ which have highest sensitivity index 1.0000, which means decreasing its value 10% will decrease the rate of reproductive number by 10% defined in [1].

4. Endemic equilibrium points with related sketch of the model

Now, we find the endemic equilibrium points which also play important role in any epidemiological model. The disease-free equilibrium points results to be locally asymptotically stable if the reproduction number(R_0) is less than unity, that is 1 while the endemic equilibrium points is locally asymptotically stable if such a number exceeds unity that is greater then 1. Following are the endemic equilibrium points of the concern model:

$$\begin{aligned} E^* &= -\frac{\xi\lambda}{}, \\ I^* &= \frac{\lambda}{\omega}S - \frac{1}{\omega}(\mu_1 + \mu_2), \\ R^* &= \frac{\eta}{\delta}(\frac{\lambda}{\omega}S - \frac{1}{\omega}(\mu_1 + \mu_2)), \end{aligned}$$

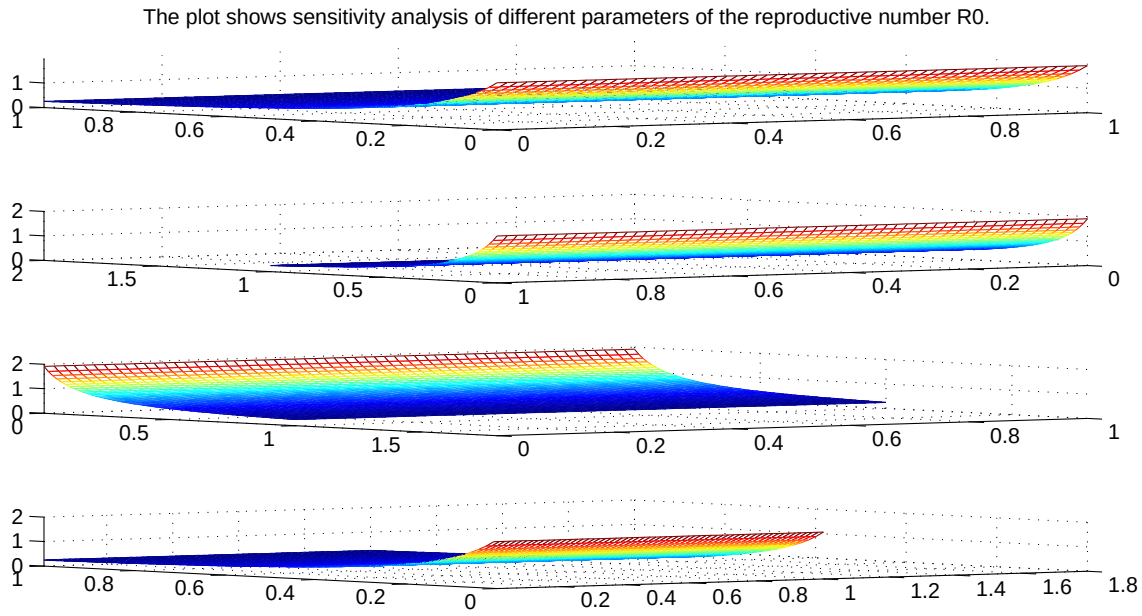

Figure 2. The plot shows sensitivity analysis of different parameters of the reproductive number R_0.

where the value of the term S^* is given below,

$$S^* = \frac{\delta\omega^2\xi^2 + \lambda\delta\omega\xi(\phi_1+\phi_2) - \lambda\delta^2(\mu_1+\mu_2)}{\lambda(\delta\omega\xi(\epsilon_1+\epsilon_2) - \lambda\xi\eta^2)}.$$

5. Local stability analysis of the proposed model

5.1. Local stability analysis at disease free equilibrium

The local stability analysis at disease free equilibrium of the model (1), are $KF_e = \{S, E, I, R\}$, which implies in disease free form as $KF_e = \{\psi/\xi, 0, 0, 0\}$. Thus, we processed by the following Jacobian matrix at KF_e:

$$K(DE_e) = \begin{bmatrix} \xi & -\lambda S & -(\epsilon_1+\epsilon_2)S^0 & 0 \\ 0 & \lambda S^0 - (\mu_1+\mu_2) & 0 & 0 \\ 0 & 0 & (\epsilon_1+\epsilon_2)S^0 - (\phi_1+\phi_2) & 0 \\ 0 & 0 & 0 & \delta\{(\epsilon_1+\epsilon_2)S^0 - (\phi_1+\phi_2)\} \end{bmatrix}. \quad (6)$$

Thus for local stability analysis of disease free equilibria, we have the following Theorem 1.

Theorem 1. *At disease free equilibrium $KF_e = \{\psi/\xi, 0, 0, 0\}$. If $R_0 < 1$, then the concern model (1) is locally asymptotically stable, while if $R_0 > 1$, the model (1) is unstable.*

Proof. We have the following eigenvalues from Jacobian matrix $J(KE_e)$:

$$\lambda_1 = -\xi, \quad (7)$$
$$\lambda_2 = \lambda S^0 - (\mu_1+\mu_2), \quad (8)$$
$$\lambda_3 = (\epsilon_1+\epsilon_2)S^0 - (\phi_1+\phi_2), \quad (9)$$
$$\lambda_4 = \delta\{(\epsilon_1+\epsilon_2)S^0 - (\phi_1+\phi_2)\}. \quad (10)$$

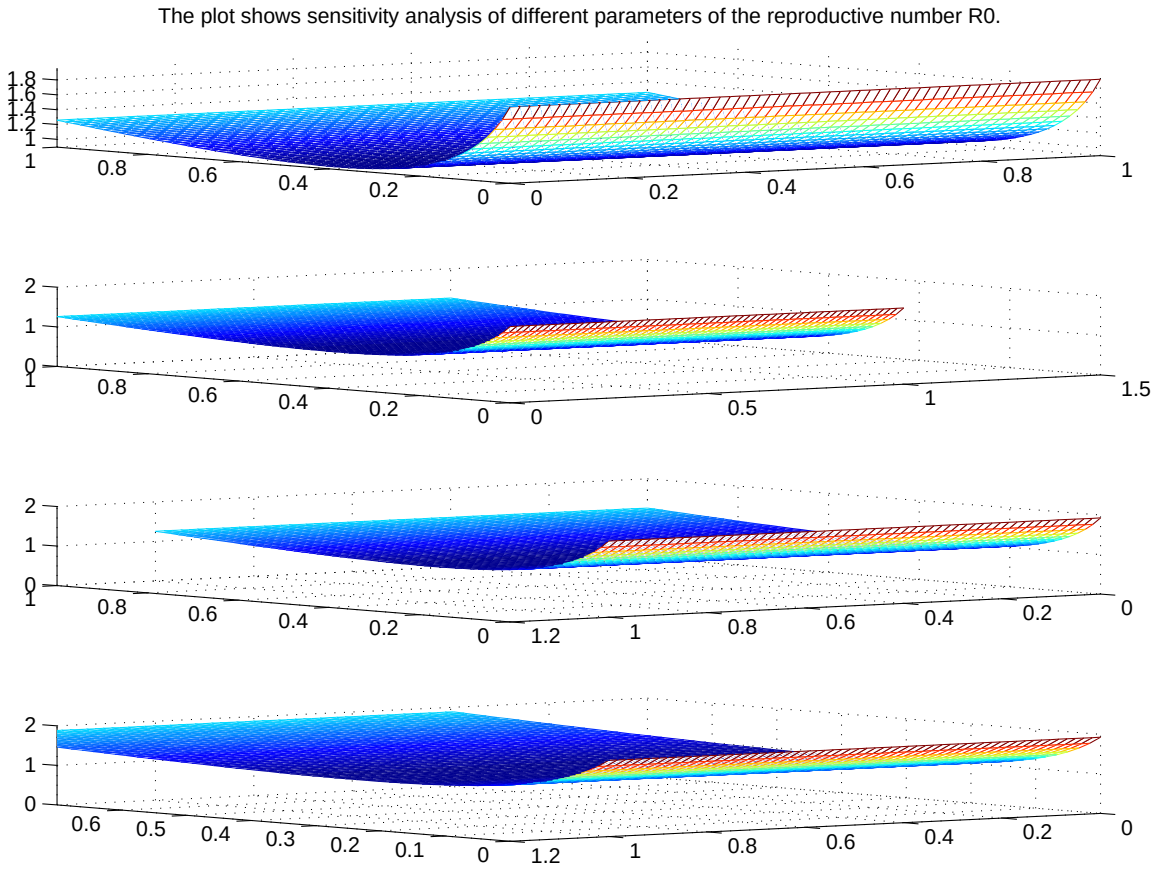

Figure 3. The plot shows sensitivity analysis of different parameters of the reproductive number R_0.

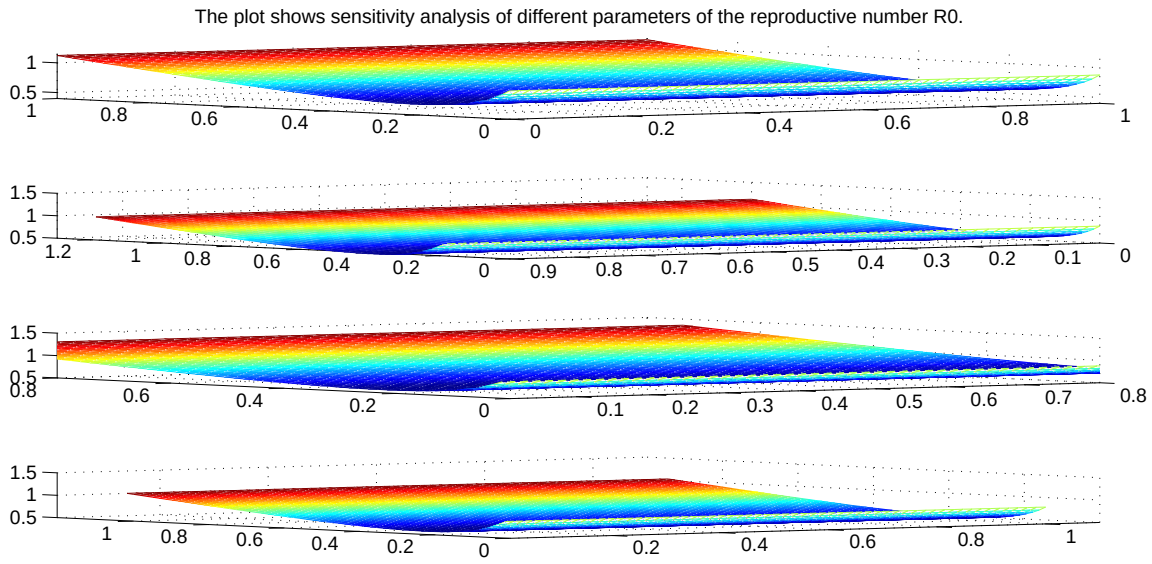

Figure 4. The plot shows sensitivity analysis of different parameters of the reproductive number R_0.

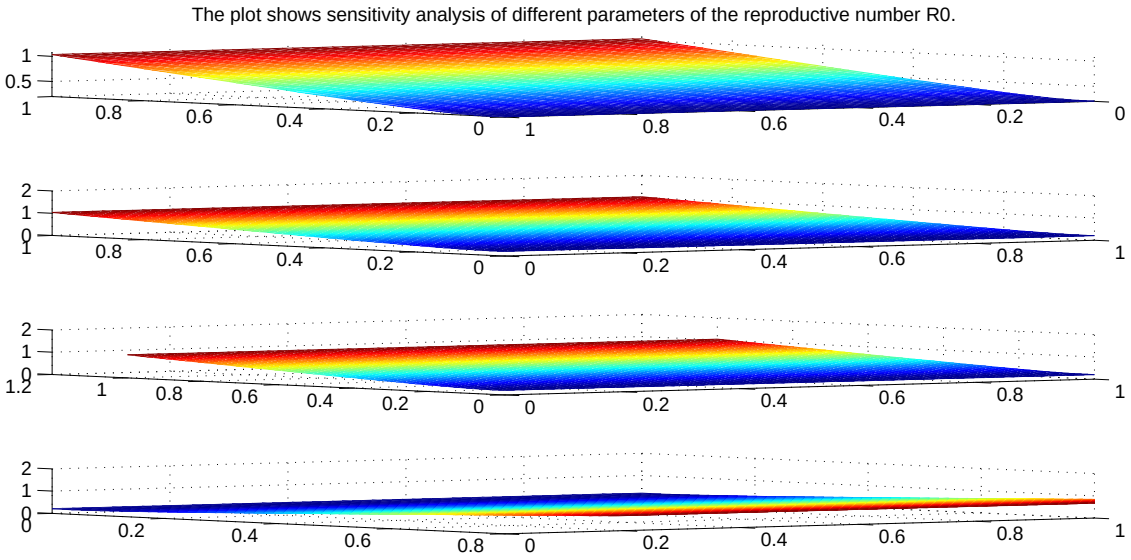

Figure 5. The plot shows sensitivity analysis of different parameters of the reproductive number R_0.

It is clear from Equation (5), $\lambda_1 = -\xi < 0$. Taking Equation (6) $\lambda_2 = -\{(\mu_1 + \mu_2) - \psi\}$. It implies that $\lambda_2 < 0$ if and only if $(\mu_1 + \mu_2) > \psi$. From Equation (7) (that is) $\lambda_3 = (\epsilon_1 + \epsilon_2)S_h^0 - (\phi_1 + \phi_2)$. So clearly $\lambda_3 = R_0 - 1$. So $\lambda_3 =< 0$ iff $R_0 < 1$. From Equation (8) $\lambda_4 = -\delta\{1 - R_0\} < 0$ if and only if $R_0 < 1$, which complete the proof. □

5.2. Local Stability Analysis At Endemic Equilibrium

For local stability analysis at endemic equilibrium, we have the following result.

Theorem 2. *Local asymptotical stability at endemic equilibrium, will hold if* $R_0 > 1$ *for model* (1) *that is, at* $KE_e = \{S^*, E^*, I^*, R^*\}$ *and unstable if* $R_0 > 1$.

Proof. For stability analysis at endemic equilibrium, consider the 4×4 matrix as:

$$KE_e = \begin{pmatrix} -\xi + \lambda E^* + K_1 & 0 & -(\epsilon_1 + \epsilon_2)S^* & 0 \\ 0 & -K_1K_2 & K_4 & 0 \\ 0 & 0 & (\omega K_4 K_1)I^* + K_1K_2K_5 & -(\eta K_1^2 K_2)I^* \\ 0 & 0 & 0 & K_6 \end{pmatrix}$$

where,

$$\begin{aligned} K_1 &= (\epsilon_1 + \epsilon_2)I^*, \\ K_2 &= \lambda S^* - \omega I^* - (\mu_1 + \mu_2), \\ K_3 &= \omega E^* + (\epsilon_1 + \epsilon_2)S^* - \eta R^* - (\phi_1 + \phi_2), \\ K_4 &= -\{\lambda(\epsilon_1 + \epsilon_2)S^* + \omega K_1\}E^*, \\ K_5 &= -\{(K_1K_2) + (\epsilon_1 + \epsilon_2)I^*(\epsilon_1 + \epsilon_2)S^*\}, \\ K_6 &= -\delta(\omega K_1 K_4 I^* + K_1K_2K_5) + \eta^2(K_1^2K_2)I^*. \end{aligned}$$

Thus for endemic equilibrium, we get

$$\lambda_1^* = -\xi + \lambda E^* + K_1, \tag{11}$$
$$\lambda_2^* = -K_1 K_2, \tag{12}$$
$$\lambda_3^* = (\omega K_4 K_1) I_e^* + K_1 K_2 K_5, \tag{13}$$
$$\lambda_4^* = K_6. \tag{14}$$

Now, from Equation (9) $\lambda_1^* = -\{\xi + \xi E^* + (\epsilon_1 + \epsilon_2) I^*\}$, so $\lambda_1 < 0$ iff $(\phi_1 + \phi_2) + \omega > (\mu_1 + \mu_2)$ and $(\phi_1 + \phi_2) + \omega > \eta^2$. Now by using Equation (10), $\lambda_2^* = -K_1 K_2 < 0$ if and only if $\lambda < 1$ and $\lambda > \omega$. Now by checking the value of λ_3^*, from Equation (11), we observed that $\lambda_3^* = (\omega K_4 K_1) I_e^* + K_1 K_2 K_5 < 0$ iff $\{\lambda(\epsilon_1 + \epsilon_2) S_h^* + K_1 \omega\} \omega E^* I^* > \omega I^* + \{(\mu_1 + \mu_2)(k_1 k_2)(\epsilon_1 + \epsilon_2)^2 - \lambda\} S^*$. By performing some calculation, we observed that $\lambda_3 < 0$. Taking Equation (12) and performing some calculation, we have $\lambda_4 < 0$ if and only if $\omega K_4 I^* + K_2 K_5 > \eta^2 K_1 K_2 I^*$. Clearly local stability analysis at endemic equilibrium is asymptotically stable for system (1) which completed the proof. □

6. Global stability analysis of the proposed model

In this section, we discuss the global stability analysis of the problem. There is a power full tool Lyapunov function, that is used for the global stability analysis, hence to check the global stability analysis of the model (1), we construct a Lyapunov function [21,22]. We have two cases: (1) global stability analysis at disease free equilibrium and (2) global stability analysis at endemic equilibrium.

6.1. Global stability analysis at disease free equilibrium

Theorem 3. *For system* (1)*, if* $R_0 \leq 1$*, then Globally asymptotically stability will hold for disease free equilibrium if* $S = S0$ *and unstable for* $R_0 > 1$.

Proof. To show global stability at disease free equilibrium of the model (1), considered the following *Lyapunov function*:

$$U(S, E, I, R) = \frac{1}{3}(S - S^0 + E - E^0 + I - I^0)^3.$$

Obviously the'above function is greater then zero at disease free equilibrium and equal to zero at $S = S^0$, and $E = I = R = 0$. Differentiating $U(S, E, I, R)$ with respect to t, we obtain the following result:

$$\frac{dU}{dt}(S, E, I, R) = (S - S^0 + E - E^0 + I - I^0)^2 \psi - \lambda S + \lambda SE - (\mu_1 + \mu_2) E - \eta IR - (\phi_1 + \phi_2) I.$$

After some simplification, we get

$$\frac{dU}{dt}(S, E, I, R) = -(S - S^0 + E - E^0 + I - I^0)(K - Q).$$

Clearly Equation (13) is less then zero if and only if $K > Q$, where

$$K = \psi E + (\mu_1 + \mu_2) E + (\eta R + (\phi_1 + \phi_2)) I,$$

and

$$Q = (1 + E)\psi.$$

Here we see that $\frac{dU}{dt}(S, E, I, R) = 0$ if and only if $S = S^0$, $E = E^0$, $I = I^0$, and $R = R^0$ while $\frac{dU}{dt}(S, E, I, R) < 0$ iff $K > Q$. Then the disease free equilibrium is globally asymptotically stable. □

6.2. Global stability analysis at endemic equilibrium

Theorem 4. *For globally asymptotically stability, if $R_0 > 1$, then the endemic equilibrium of model (1) is stable and $S = S^*$, $E = E^*$, $I = I^*$ $R = R^*$ and unstable, if $R_0 < 1$.*

Proof. For global stability analysis at endemic equilibrium, we define

$$Q(S, E, I, R) = \frac{1}{2}(S - S^*)^2 + \frac{1}{2}(I - I^*)^2,$$

we have $Q(S, E, I, R) > 0$ and it equal to zero at $S = S^*$, $E = E^*$, $I = I^*$. Differentiating $Q(S, E, I, R)$ with respect to t we get,

$$\frac{dQ}{dt}(S, E, I, R) = (S - S^* + I - I^*)(\frac{d}{dt}S + \frac{d}{dt}I),$$

Putting values from model (1) in above, we obtain

$$\frac{dQ}{dt}(S, E, I, R) = -(S - S^* + I - I^*)(\eta R + \phi_1 + \phi_2 - \omega E)I.$$

Hence we have $\frac{dQ}{dt}(S, E, I, R) = 0$ if and only if $S = S^*$, $E = E^*$, $I = I^*$ and $R = R^*$. Also $\frac{dQ}{dt}(S, E, I, R) < 0$, iff $\eta R + \phi_1 + \phi_2 > \omega E$, hence endemic equilibria is globally asymptotically stable for model (1). So the proof is completed. □

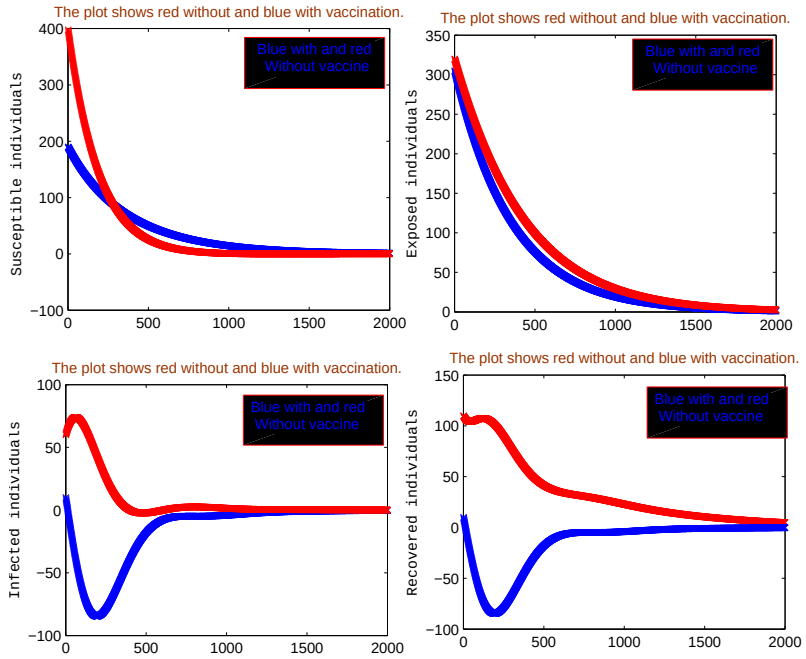

Figure 6. The plot shows red without and blue with vaccination.

7. Numerical simulation and discussion

In this section, we present numerical interpretation of the proposed model with the help of Matlab programming. Numerical results given in Figure 1 shows simple graph of the model having no vaccination used yet, Figures 2-5 represents different behaviour of the reproductive number R_0, Figure 6 with and without vaccination in population, while Figures 7 and 8 shows graph with and without vaccine and also the area infected graph if no vaccination taken.

We present the following Table 2. By using the parameters value, from Table 2, non-negative initial population sizes and from different time interval, we obtain the simulation Figure 6, which represents that there are always susceptible $S(t)$ and recovered $R(t)$ population which quickly recovered with vaccination, while the remaining individuals populations i.e. exposed $E(t)$, and infected $I(t)$ individuals respectively shown the exposed individuals recovery is very slow without vaccination, while with vaccination, a rapid effeteness occur in there health condition. Similarly if we do not provide vaccine to the infected class we see from simulation their graph is going high but vaccination rapidly cover their problem. Also the population in the model is represented by area graph. In the area graph the less area shows less infection while more cover area graph shows great infection in any time in any population.

All values taken fixed in the Table 2. In Figure 6 the simulation are presented with and without vaccination to the population, while Figure 7 and 8 shows effected area if no vaccination used.

Table 2. Values of Parameters

Notation	Description of Parameter	Value
S	Susceptible individuals population	$00-2000$
E	Exposed individuals population	$00-2000$
I	Infected individuals population	$00-2000$
R	Recovered individuals population	$00-2000$
ψ	New birth rate in susceptible individuals	0.6321
λ	Transmission rate from susceptible to exposed individuals	0.2877
ω	Transmission rate from exposed to infected individuals	0.7613
η	Transmission rate from infected to recover individuals	0.4389
ϵ_1	Individuals get wild animals infection from susceptible to infected	0.1234
ϵ_2	Individuals get domestic animals infection from susceptible to infected	0.2431
μ_1	Natural death rate of exposed individuals	0.9704
μ_2	Infectious death rate of exposed individuals	0.0432
ϕ_1	Natural death rate of infected individuals	0.2006
ϕ_2	Infectious death rate of infected individuals	0.0656
δ	Natural death rate of recover individuals	0.6704

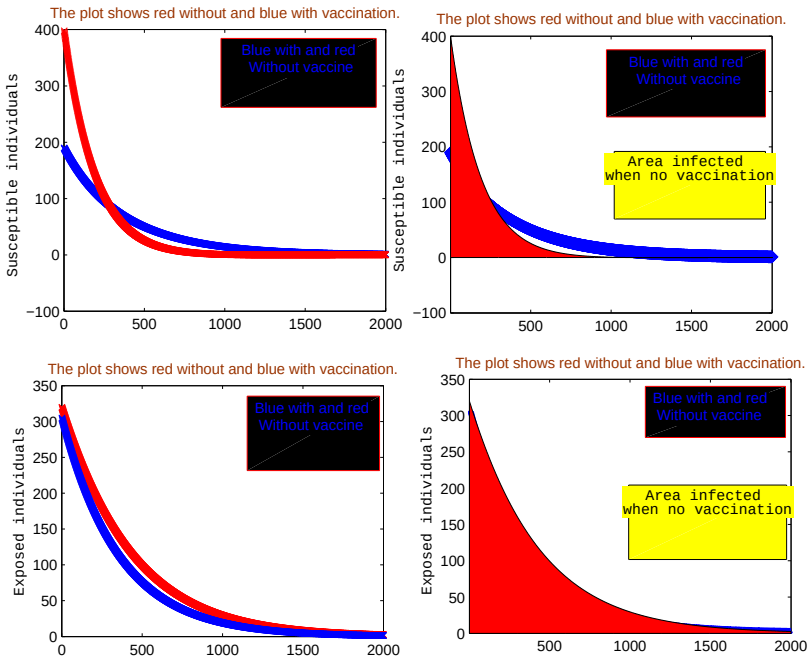

Figure 7. The plot shows red without and blue with vaccination.

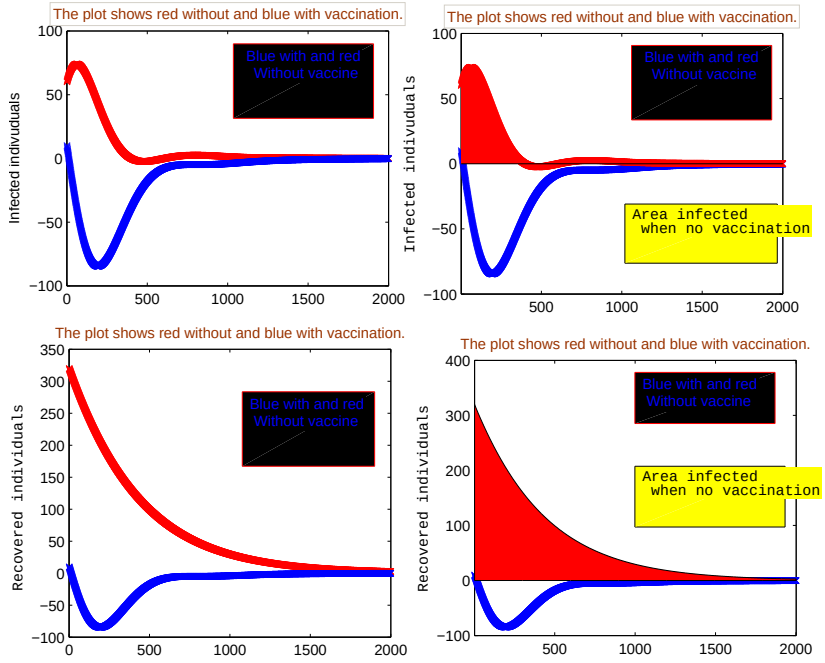

Figure 8. The plot shows red without and blue with vaccination.

8. Conclusion

A mathematical epidemic model SEIR of the Ebola virus is considered where the transmissibility agent is considered animal at any time "t" in the population. First we formulated the model according to there infectious classes, and by next-generation matrix approach, we find reproductive number (that is) R_0 with biological feasible region. Also we discussed the reproductive number sensitivity indices by showing different behavior of R_0. After we discussed endemic equilibrium points of the model. Then according to the reproductive number we discussed the local stability and global stability at disease free equilibrium and at endemic equilibrium and shown stable. The global stability at both respects is discussed with the help of Lyapunov function. Finally, we obtained numerical solution of compartmental mathematical model by the using Matlab program. Also we obtained the area involved we no vaccination taken. The figures show first column of the with and without vaccination population while the second column shows the infected population area of the model. The graph approaches represent new idea for the scientists in future. In the area graph, the less area shows less infection while more cover area graph shows great infection at any time in any population.

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