Tahir et al., *Cogent Biology* (2019), 5: 1619219
https://doi.org/10.1080/23312025.2019.1619219

cogent biology

CELL, MOLECULAR & DEVELOPMENTAL BIOLOGY | RESEARCH ARTICLE

Modeling and stability analysis of epidemic expansion disease Ebola virus with implications prevention in population

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Received: 18 July 2018
Accepted: 05 May 2019
First Published: 3 June 2019

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Reviewing editor:
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Abstract: In this subsection, we presented a mathematical model of Ebola virus (EBOV) proposed by susceptible exposed infected recovered (SEIR) model. In our model, the population is affected by animals. EBOV is an infectious agent causing haemorraghic fever, a severe infectious disease characterised by high fever and bleeding, in humans and some monkeys. Here, we assessed the transmissibility associated with the infection stages of EBOV that generated an epidemic model. In order to do this, in the first step, we formulate the model, and the basic properties of the proposed model are presented. The basic reproductive number is obtained by using the next generation matrix approach. Then, all the endemic equilibrium points related to the disease are derived. We also find the conditions to investigate all possible equilibria of the model in terms of the basic reproduction number (local and global stability). In last, numerical simulation is presented with and without vaccination or control for the proposed model.

Subjects: Biology; Epidemiology; Applied Mathematics

Keywords: Ebola virus; R0 reproduction number; formulation of model; endemic equilibrium points; stability analysis; numerical simulation

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PUBLIC INTEREST STATEMENT

Viruses are considered as a big threat all over the world. All viruses are harmful but now a days Ebola virus is baneful disease either in urban and some parts of rural areas, which become epidemic slowly. Here, we assessed Ebola SEIR mathematical model, and the transmissibility agent of the virus is animals, which spread chronic disease both in human and monkey. This study will help the people to care about animals because no corner of the world is without animals. Ebola virus is so harmful and transmissible disease even if an individual gets infection from Ebola virus, it will spread to other individual or family member. Thus, to avoid from Ebola virus and for good environment of human health, this study provides a range of safety in life to treat the animals in a safety way.

1. Introduction

The recent Ebola virus (EBOV) epidemic in West Africa emerged around the end of 2013 in the prefecture of Guckdou in Guinea (Baize et al., 2014) and caused at least 11,310 deaths among 28,616 recorded cases in Guinea, Sierra Leone, and Liberia (World Health Organization, 2016c). 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. Various reasons may explain the slow initial response to the West African EBOV epidemic, including poor public health infrastructure and local unfamiliarity with EBOV disease, as well as a lack of preparedness by the international community. Because efforts to control the epidemic could not rely on vaccination or effective antiviral drugs, the outbreak response focused on standard medical practices (e.g. case identification and isolation), as well as community practices (e.g. sanitary funeral practices) (Blackwood & Childs, 2016).

Mathematical models have been used extensively to study the dynamics of EBOV transmission e.g. superspreading events (Lau et al., 2017), also M.Tahir. et al. also presented mathematical model for EBOV (Tahir, Inayat Ali Shah, Zaman, & Muhammad, 2018). EBOV is one of the four EBOVs known to cause disease in humans. It has the highest case-fatality rate of these EBOVs, averaging 83% since the first outbreaks in 1976, although fatality rates up to 90% have been recorded in one outbreak (200,203). There have also been more outbreaks of EBOV than of any other EBOV. In 1976, the first EBOV was found of Marburg virus (Bowen et al., 1977; Pattyn, Jacob, van der Groen, Piot, & Courteille, 1977). In the mean while, other team introduced EBOV (Bowen et al., 1977; Kuhn et al., 2010; Pattyn et al., 1977). 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 Democratic Republic of Congo. To avoid from the confusion they renamed in 2010 that Ebola virus.

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 include (1) Cuevavirus,(2) Marburgvirus, and (3) Ebolavirus. Majority of human death occurred by EBOV, and in West Africa, it becomes epidemic in 2013 to 2015 (World Health Organisation, 2016a). Some cases reported out from West Africa reveal that all infected are foreign travellers who exposed to affected regions while later they showed Ebola fever symptoms when reached to destinations (Khan, Islam, Arif, & Ul Haq, 2013). In this period, the virus caused nearly about 28,616 are suspected while exactly 11,310 confirmed deaths cases (Reardan, 2014; Bausch & Schwarz, 2016b). The EBOV spread in many countries, which started in Guinea and moved across Liberia and Sierra Leone. The EBOV also spreads by human to human contacts such as secretions, blood, body fluids of the infected individuals, surfaces, and the materials of infected like cloth and bedding.

The virus causes serious acute illness and becomes fatal if the patient takes no treatment. The EBOV 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 EBOV, Arias et al. (2016) 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 analyses can also shed light on the origins, evolution and transmission dynamics of a pathogen during an epidemic (Holmes, Dudas, Rambaut, & Andersen, 2016). Early in the EBOV epidemic, analyses such as those by Gire et al. (2014) demonstrated that the virus entered the human population in late 2013 and crossed from Guinea to Sierra Leone in May 2014 through sustained human-to-human transmission.

In this article, we processed as fallows: First, the disease according to their infection has been formulated, then the key value reproductive number is derived. After that endemic equilibrium points are obtained. Then, the local stability analysis is shown stable at disease free, as well as, at

endemic equilibrium. Further, we derived the global stability of the model with the help of Lyapunov function has been discussed at disease free, and at endemic equilibrium. Finally, we show numerically the result with and without vaccination or control which supports the model.

2. Model formulation and method

In this subsection, we presented Ebola SEIR mathematical model (Anderson & May, 1991; Keeling & Rohani, 2007). In this model, virus is transmitted from wild and domestic animals. The proposed model is defined in the following four classes: first class is about susceptible individuals and represented by S_h whose are not still infected but have a chance to be suffer in the said disease in any time t . Second class is about E_e that is, exposed individuals showing no symptoms of virus. The third class is about infected individuals and represented by I_e who infected by EBOV in any time t where the fourth class is the class of R_e that is, the people recovered from EBOV. Then the differential equations of the mathematical SEIR model of the EBOV are represented as:

$$\frac{dS_h}{dt} = \psi - \xi S_h - \lambda S_h E_e - \epsilon_1 S_h I_e - \epsilon_2 S_h I_e,$$

$$\frac{dE_e}{dt} = \lambda S_h E_e - \omega E_e I_e - (\mu_1 + \mu_2) E_e,$$

$$\frac{dI_e}{dt} = \omega E_e I_e + \epsilon_1 S_h I_e + \epsilon_2 S_h I_e - \eta I_e R_e - (\phi_1 + \phi_2) I_e, \tag{1}$$

$$\frac{dR_e}{dt} = \eta I_e - \delta R_e.$$

With the initial conditions,

$$S_h(0) \geq 0, E_e(0) \geq 0, I_e(0) \geq, R_e(0) \geq 0.$$

Certain assumptions have been used in model (1), which are classified as, S_h represents susceptible individuals, E_e shows exposed individuals, I_e represents infected individuals, R_e represents recovered individuals, ψ represents new birth rate inter in the susceptible individuals, ξ shows natural death rate of susceptible individuals, ϵ_1 shows transmission rate from susceptible to infected individuals through wild animals, ϵ_2 represents the transmission rate from susceptible to infected individuals through domestic animals, λ shows the transmission rate from susceptible to exposed individuals, ω represents transmission rate from exposed to infected individuals, η represents the rate of infected to recovered individuals, μ_1 and μ_2 show natural death rate and infectious death rate of exposed individuals, ϕ_1 and ϕ_2 represent natural and infectious death rate of infected individuals, respectively.

The total population of the model is represented by,

$$B(t) = S_h + E_e + I_e + R_e.$$

Which will be written as,

$$\frac{dB(t)}{dt} = \frac{dS_h}{dt} + \frac{dE_e}{dt} + \frac{dI_e}{dt} + \frac{dR_e}{dt}.$$

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

$$\frac{dB(t)}{dt} = \psi - \xi S_h - (\mu_1 + \mu_2) E_e - \eta I_e R_e - (\phi_1 + \phi_2) I_e + \eta I_e - \delta R_e. \tag{2}$$

While from Equation (2) we write that,

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

Clearly

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

Now, the feasible region, which is sufficient for the study of the model (1), that is, R is., $\Re$ as.

$$\Re = \{(S_h, E_e, I_e, R_e) \in R_+^5, B \leq \frac{\psi}{\xi}\}.$$

3. R_0 the reproduction number of model

Reproductive number is considered as one of the important key values in many epidemiological work represented by R_0, which predicts that where the infectious disease will be spread into a population class or not. We define the basic reproduction number as it shows average rate of a secondary infectious cases when one of the infectious individuals introduced in a susceptible population (Keeling & Rohani, 2007). 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., (Anderson & May, 1991). For this, we divide the system as follows,

$$F = \begin{bmatrix} \lambda S_h E_e - \omega E_e I_e \\ \omega E_e I_e + \epsilon_1 S_h I_e + \epsilon_2 S_h I_e - \eta I_e R_e \end{bmatrix}.$$

We have also,

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

Now to find *jacobian* $\overline{F}$ and $\overline{V}$ we processed as.

$$\overline{F} = \begin{bmatrix} \lambda S_h - \omega I_e & -\omega E_e \\ \omega I_e & \omega E_e + \epsilon_1 S_h + \epsilon_2 S_h - \eta R_e \end{bmatrix}.$$

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

So R_0 (Reproductive number) of our model (1) is given below,

$$R_0 = \frac{(\epsilon_1 + \epsilon_2)\psi}{\xi(\phi_1 + \phi_2)}. \tag{3}$$

4. Endemic equilibrium points of model

In this section, we find the endemic equilibrium points, which also play important role in any epidemiological model. Following are the endemic equilibrium points of the concerned model.

$$E_e^* = -\frac{\xi\lambda}{},$$

$$I_e^* = \frac{\lambda}{\omega} S_h - \frac{1}{\omega}(\mu_1 + \mu_2),$$

$$R_e^* = \frac{\eta}{\delta}\left(\frac{\lambda}{\omega} S_h - \frac{1}{\omega}(\mu_1 + \mu_2)\right).$$

Where the value of the term S_h^* is given below,

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

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5. Local stability analysis of proposed model

In this area of the model, we work on the model local stability analysis. The local stability analysis needs to be found on disease-free equilibrium and endemic equilibrium.

6. Local stability analysis at disease-free equilibrium

Now the local stability analysis at disease-free equilibrium of the model (1), is$KF_e = \{S_h, E_e, I_e, R_e\}$, 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, under,

$$K(DE_e) = \begin{bmatrix} \xi & -\lambda S_h & -(\epsilon_1+\epsilon_2)S_h^0 & 0 \\ 0 & \lambda S_h^0 - (\mu_1+\mu_2) & 0 & 0 \\ 0 & 0 & (\epsilon_1+\epsilon_2)S_h^0 - (\phi_1+\phi_2) & 0 \\ 0 & 0 & 0 & \delta\{(\epsilon_1+\epsilon_2)S_h^0 - (\phi_1+\phi_2)\} \end{bmatrix}. \tag{4}$$

Thus, for disease-free equilibria at local stability analysis, we have the following theorem.

Theorem 8.1. *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* $R_0>1$ *we said the model is unstable.*

Proof: We have the following eigenvalues from Jacobian matrix $J(KE_e)$ in Equation (4),

$$\lambda_1 = -\xi, \tag{5}$$

$$\lambda_2 = \lambda S_h^0 - (\mu_1+\mu_2), \tag{6}$$

$$\lambda_3 = (\epsilon_1+\epsilon_2)S_h^0 - (\phi_1+\phi_2), \tag{7}$$

$$\lambda_4 = \delta\{(\epsilon_1+\epsilon_2)S_h^0 - (\phi_1+\phi_2)\}. \tag{8}$$

Now, we discuss the local stability analysis at disease-free equilibrium as it is clear from Equation (5), that is $\lambda_1 = -\xi<0$. Taking Equation (6) $\lambda_2 = -\{(\mu_1+\mu_2) - \psi\}.$, it implies that $\lambda_2<0$ if and only if $(\mu_1+\mu_2)>\psi$ which is clear from Equation (7), that is, $\lambda_3 = (\epsilon_1+\epsilon_2)S_h^0 - (\phi_1+\phi_2)$. So it clearly shows $\lambda_3 = R_0 - 1$. So $\lambda_3 = <0$ iff $R_0<1$. From Equation (8) $\lambda_4 = -\delta\{1-R_0\}<0$ iff $R_0<1$ which completes the proof. Hence, we say that local stability analysis at disease-free equilibrium of the model (1) is asymptotically stable.

7. Local stability analysis at endemic equilibrium

Now, in this subsection of the article, we find stability analysis of the model (1) at endemic equilibrium. For local stability analysis at endemic equilibrium, we have the following result.

Theorem 9.1. *Local asymptotical stability at endemic equilibrium, will hold if* $R_0>1$ *for model* (1) *that is, at* $KE_e = \{S_h^*, E_e^*, I_e^*, R_e^*\}$ *and unstable if* $R_0>1$.

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

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

The above terms are specified below,

$$K_1 = (\epsilon_1+\epsilon_2)I_e^*,$$

$$K_2 = \lambda S_h^* - \omega I_e^* - (\mu_1 + \mu_2),$$

$$K_3 = \omega E_e^* + (\epsilon_1 + \epsilon_2)S_h^* - \eta R_e^* - (\phi_1 + \phi_2),$$

$$K_4 = -\{\lambda(\epsilon_1 + \epsilon_2)S_h^* + \omega K_1\}E_e^*,$$

$$K_5 = -\{(K_1K_2) + (\epsilon_1 + \epsilon_2)I_e^*(\epsilon_1 + \epsilon_2)S_h^*\},$$

$$K_6 = -\delta(\omega K_1K_4I_e^* + K_1K_2K_5) + \eta^2(K_1^2K_2)I_e^*.$$

Thus for endemic equilibrium, we get,

$$\lambda_1^* = -\xi + \lambda E^* + K_1, \tag{9}$$

$$\lambda_2^* = -K_1\,K_2, \tag{10}$$

$$\lambda_3^* = (\omega K_4K_1)I_e^* + K_1K_2K_5, \tag{11}$$

$$\lambda_4^* = K_6. \tag{12}$$

Now, we discuss the endemic equilibrium points, 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 using Equation (10) $\lambda_2^* = -K_1K_2<0$ if and only if $\lambda<1$ and $\lambda>\omega$. Now, we check the value of λ_3^*, from Equation (11) we observed that $\lambda_3^* = (\omega K_4K_1)I_e^* + K_1K_2K_5<0$ iff $\{\lambda(\epsilon_1 + \epsilon_2)S_h^* + K_1\omega\}\omega E_e^*I_e^*>\omega I_e^* + \{(\mu_1 + \mu_2)(k_1k_2)(\epsilon_1 + \epsilon_2)^2 - \lambda\}S_h^*$ by performing some calculation we observed that $\lambda_3<0$. Taking Equation (12) and performing some calculation then $\lambda_4<0$ if and only if $\omega K_4I^* + K_2K_5>\eta^2K_1K_2I_e^*$. Clearly, local stability analysis at endemic equilibrium is asymptotically stable for system (1), which completed the proof.

8. Global stability analysis of the proposed model

In this section, we discuss the global stability analysis of the problem because the global stability analysis is considered one of the fundamental. There is a power full tool Lyapunov function is used for the global stability analysis of the model. To check the global stability analysis of our model (1), we construct a Lyapunov function, which used many authors also in their models (Reardan, 2014; Van Den Driessche & Watmough, 2002). For the global stability analysis of model (1) at disease-free equilibrium, considered the following Lyapunov function. Now we have the following known stability results which are stated below,

9. Global stability analysis at disease free equilibrium

To prove global stability analysis at disease-free equilibria, we construct Lyapunov function for which the following result states as under,

Theorem 11.1. *For system* (1) *if* $R_0 \leq 1$, *then Globally asymptotically stability, well hold for disease-free equilibrium if* $S_h = S_h0$ *and unstable for* $R_0>1$.

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

$$U(S_h, E_e, I_e, R_e) = \frac{1}{3}(S_h - S_h^0 + E_e - E_e^0 + I_e - I_e^0)^3.$$

Obviously, the 'above function is greater then zero at disease-free equilibrium and equal to zero at $S_h = S_h^0$, and $E_e = I_e = R_e = 0$. Differentiating $U(S_h, E_e, I_e, R_e)$ with respect to t, we obtain the following result using model (1),

$$\frac{dU}{dt}(S_h, E_e, I_e, R_e) = (S_h - S_h^0 + E_e - E_e^0 + I_e - I_e^0)^2$$

$$(\psi - \lambda S_h + \lambda S_h E_e - (\mu_1 + \mu_2)E_e - \eta I_e R_e - (\phi_1 + \phi_2)I_e.$$

After some simplification we get,

$$\frac{dU}{dt}(S_e, E_e, I_e, R_e) = -(S_h - S_h^0 + E_e - E_e^0 + I_e - I_e^0)(K - Q).$$

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

$$K = \psi E_e + (\mu_1 + \mu_2)E_e + (\eta R_e + (\phi_1 + \phi_2))I_e.$$

And

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

Here, we see that $\frac{dU}{dt}(S_h, E_e, I_e, R_e) = 0$ if and only if $S_h = S_h^0$, $E_e = E_e^0$, $I_e = I_e^0$, and $R_e = R_e^0$ while $\frac{dU}{dt}(S_h, E_e, I_e, R_e)<0$ iff $K>Q$. Then the disease-free equilibrium is globally asymptotically stable.

10. Global stability analysis at endemic equilibrium

In this subsection, we construct the following *Lyapunov* function to verify the endemic equilibrium, as

Theorem 12.1. *For globally asymptotically stability for* $R_0>1$, *then the endemic equilibrium of model* (1) *is stable, if* $S_h = S_h^*$, $E_e = E_e^*$, $I_e = I_e^*$ $R_e = R_e^*$ *and unstable, if* $R_0<1$.

Proof: For global stability analysis at endemic equilibrium, we define the following *Lyapunov* for model (1),

$$Q(S_h, E_e, I_e, R_e) = \frac{1}{2}(S_h - S_h^*)^2 + \frac{1}{2}(I_e - I - e^*)^2.$$

the define function $Q(S_h, E_e, I_e, R_e)>0$ and it is equal to zero at $S_h = S_h^*$, $E_e = E_e^*$, $I_e = I_e^*$ Differentiating $Q(S_h, E_e, I_e, R_e)$ with respect to 't' we get,

$$\frac{dQ}{dt}(S_h, E_e, I_e, R_e) = (S_h - S_h^* + I_e - I_e^*)(\frac{d}{dt}S_h + \frac{d}{dt}I_e),$$

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

$$\frac{dQ}{dt}(S_e, E_e, I_e, R_e) = -(S_h - S_h^* + I_e - I_e^*)(\eta R_e + \phi_1 + \phi_2 - \omega E)I_e,$$

Hence we have $\frac{dQ}{dt}(S_h, E_e, I_e, R_e) = 0$ if and only if $S_h = S_h^*$ and $E_e = E_e^*$ and $I_e = I_e^*$, $R_e = R_e^*$ also $\frac{dQ}{dt}(S_h, E_e, I_e, R_e)<0$, iff $\eta R_e + \phi_1 + \phi_2 > \omega E_e$ from above it is clear that endemic equilibria is globally asymptotically stable for model (1). So the proof is completed.

11. Numerical simulation and discussion

Work in this subsection contains numerical interpretation of the proposed model by the use of Runge-kutta method, that is, $RK-4$. We use different parameters. Its values and numerical result are stated in Figures 1–4. In this section, the numerical simulations of the proposed model (1) are presented for the verification of analytical results. The numerical results are obtained by using the Runge-Kutta method of order four. The parameter values used in the simulation are given in Table 1,

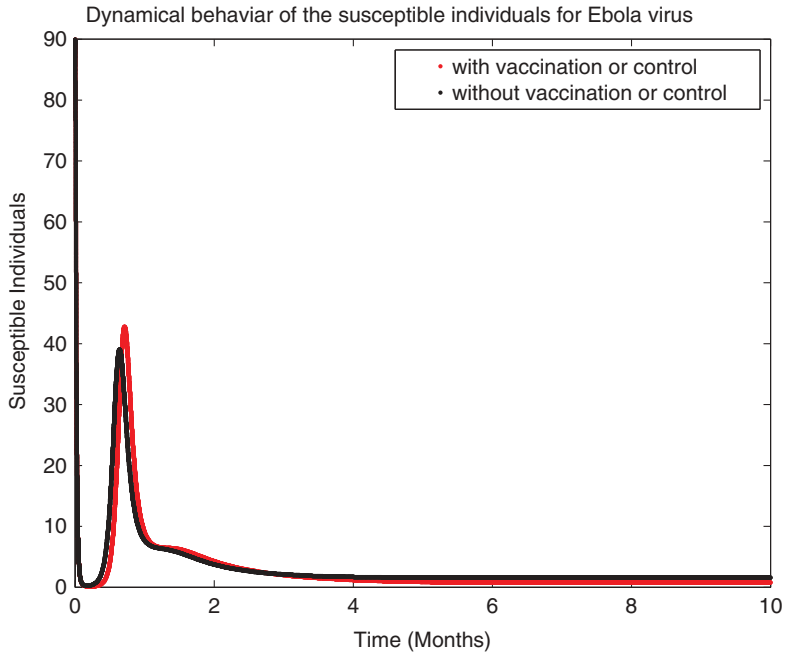

Figure 1. The plot shows the Ebola virus behaviour.

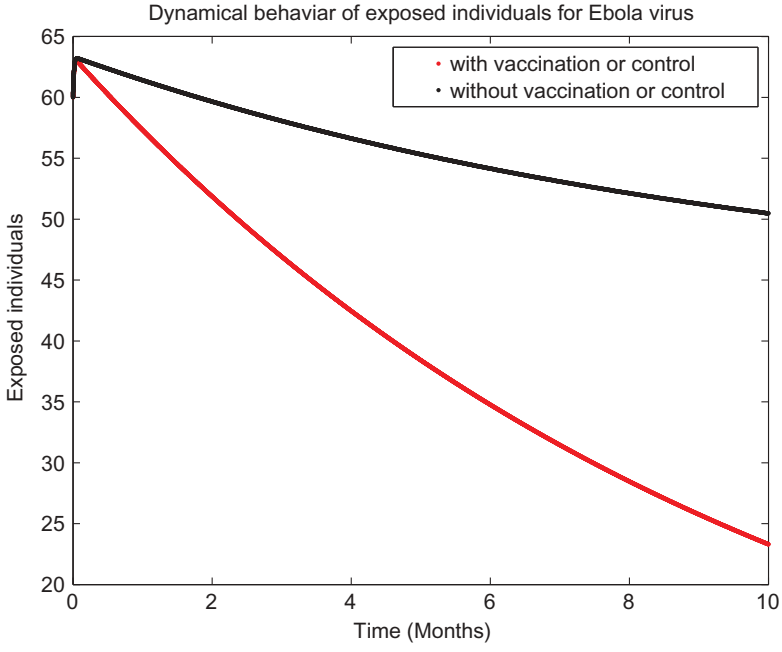

Figure 2. The plot shows the Ebola virus behaviour.

which are biologically feasible. By using the parameter values, non-negative initial population sizes and from the time interval, we obtain the simulation (Figures 1–4), which represents that there are always susceptible S(t) and recovered R(t) population which quickly recovered with vaccination, while the remaining individuals i.e., exposed E(t), and infected I(t) individuals, respectively, show that exposed also need vaccination, while without vaccination exposed class getting health but very slowly, while vaccine provides a rapid health recovery. Similarly, if we do not provide vaccine to the infected class, we see from the graph, simulation goes high but vaccination rapidly covers their

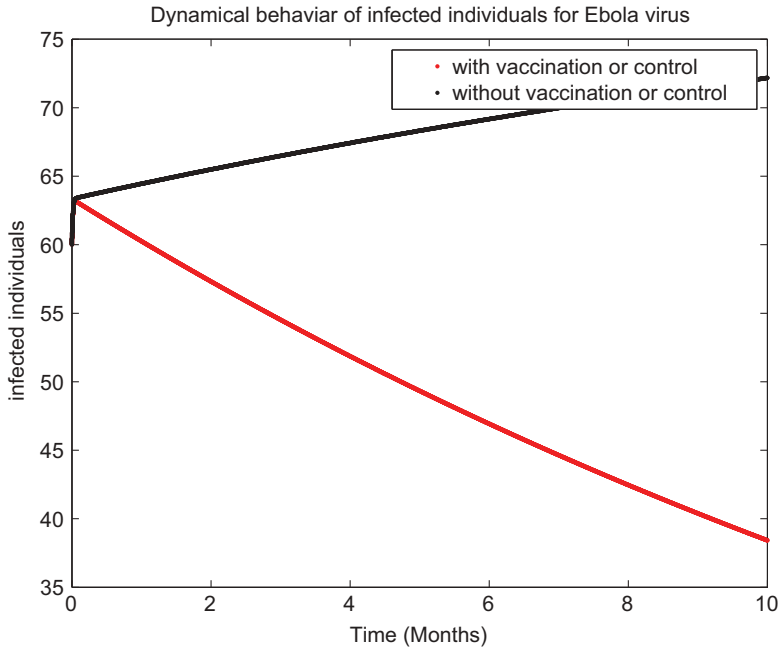

Figure 3. The plot shows the Ebola virus behaviour.

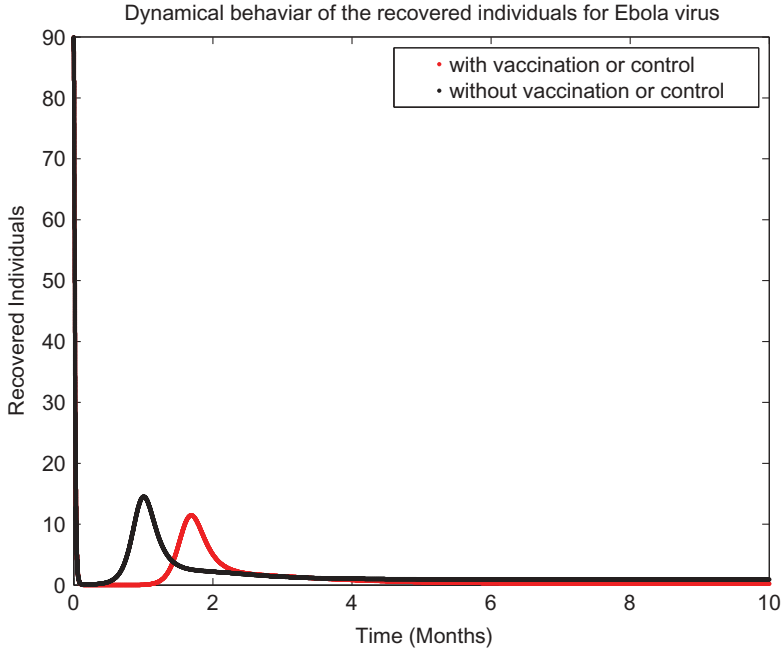

Figure 4. The plot shows the Ebola virus behaviour.

problem. All values taken are fixed in the table above. In Figures 1–4, the simulation is presented with vaccination to the population (Khan et al., 2013).

12. Conclusion of the proposed model

A compartmental (SEIR) mathematical epidemic model of the EBOV is considered. In our proposed model, the transmissibility agent is considered animal at any time t in the population. First, we formulated the model, and obtained key number reproductive number, that is, R0. After that, we discussed endemic equilibrium points of the model. Then, according to the reproductive number, we

Table 1. Description of parameter and its values

Notation	Description of Parameter	Value
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

discussed the local stability and global stability at disease-free equilibrium and at endemic equilibrium and shown stable. Finally, we obtained numerical solution of compartmental mathematical model by the use of Runge-Kutta method RK4 tool and presented the results from Figures 1–4 with vaccination which support the model.

Conflict of Interest
There is no conflict of interest regarding this paper.

Authors contribution
All authors equally contributed this paper.

Acknowledgements
All authors read and approved the final version.

Funding
The authors received no direct funding for this research.

Competing Interests
The authors declares no competing interests.

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Citation information
Cite this article as: Modeling and stability analysis of epidemic expansion disease Ebola virus with implications prevention in population, Muhammad Tahir, Nousheen Anwar, Syed Inayat Ali Shah & Tahir Khan, *Cogent Biology* (2019), 5: 1619219.

References

Anderson, R. M., & May, R. M. (1991). *Infectious diseases of humans: Dyn and Cont*. Oxford, UK: Oxford University Press.

Arias, A., Watson, S. J., Asogun, D., Tobin, E. A., Lu, J., Phan, M. V. T., ... Cotten, M. (2016). Rapid outbreak sequencing of Ebola virus in Sierra Leone identifies transmission chains linked to sporadic cases. *Virus Evolution*, *2*, vew016.

Baize, S., Pannetier, D., Pharm, D., Oestereich, L., Rieger, T., & Koivogui, L. (2014). Emergence of Zaire Ebola virus disease in Guinea. *The New England Journal of Medicine*, *371*, 1418–1425. doi:10.1056/NEJMoa1410490

Bausch, D. G., & Schwarz, L. (2016b). *Outbreak of Ebola virus disease in Guinea: Where ecology meets economy*. Retrieved from https://doi.org/10.1371/journal.pntd.0003056

Blackwood, J. C., & Childs, L. M. (2016). The role of interconnectivity in control of an Ebola epidemic. *Scientific Reports*, *6*, 29262. doi:10.1038/srep29262

Bowen, E. T. W., Lloyd, G., Harris, W. J., Platt, G. S., Baskerville, A., & Vella, E. E. (1977). Viral haemorrhagic fever in southern Sudan and northern Zaire. Preliminary studies on the aetiological agent. *Lancet*, *309*(8011), 5713. PMID 65662. doi:10.1016/s0140-6736(77)92001-3

Gire, S. K., Goba, A., Andersen, K. G., Sealfon, R. S. G., Park, D. J., Kanneh, L., ... Sabeti, P. C. (2014). Genomic surveillance elucidates Ebola virus origin and transmission during the 2014 outbreak. *Science*, *345*, 1369–1372. doi:10.1126/science.1259657

Holmes, E. C., Dudas, G., Rambaut, A., & Andersen, K. G. (2016). The evolution of Ebola virus: Insights from the 20132016 epidemic. *Nature*, *538*, 193200.

Keeling, M., & Rohani, P. (2007). *Modelling infectious diseases in humans and animals*. Princeton, NJ: Princeton University Press.
Khan, M. A., Islam, S., Arif, M., & Ul Haq, Z. (2013). Transmission model of hepatitis B virus with the migration effect. *Hindawi Publishing Corporation BioMed Research International*, *150681*, 10.
Kuhn, J. H., Becker, S., Ebihara, H., Geisbert, T. W., Johnson, K. M., Kawaoka, Y., … Jahrling, P. B. (2010). Proposal for a revised taxonomy of the family filoviridae: Classification, names of taxa and viruses, and virus abbreviations. *Archives of Virology*, *155*(12), 2083103. PMC 3074192?Freely accessible. PMID 21046175.. doi:10.1007/s00705-010-0814-x
Lau, M. S. Y., Dalziel, B. D., Funk, S., McClelland, A., Tiffany, A., Riley, S., … Grenfell, B. T. (2017). Spatial and temporal dynamics of superspreading events in the 2014–2015 West Africa Ebola epidemic. *Proceedings of the National Academy of Sciences*, *114*, 2337–2342. doi:10.1073/pnas.1614595114
Pattyn, S., Jacob, W., van der Groen, G., Piot, P., & Courteille, G. (1977). Isolation of Marburg-like virus from a case of haemorrhagic fever in Zaire. *Lancet*, *309*(8011), 5734. PMID 65663. doi:10.1016/s0140-6736(77)92002-5
Reardan, S. (2014). The first nine months of the epidemic and projection, Ebola virus disease in west Africa. archive of Ebola Response Team. *New England Journal of Medicine*, *511*(75.11), 520.
Tahir, M., Inayat Ali Shah, S., Zaman, G., & Muhammad, S. (2018). Ebola virus epidemic disease its modeling and stability analysis required abstain strategies. *Cogent Biology*, *4*(1), 1488511. doi:10.1080/23312025.2018.1488511
Van Den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, *180*, 29–48. doi:10.1016/S0025-5564(02)00108-6
World Health Organisation. (2016a). *Ebola data and statistics*.
World Health Organization. (2016c). *Ebola situation report*. Retrieved from apps.who.int/iris/bitstream/10665/208883/1/ebolasitrep-10Jun2016-eng.pdf

Cogent Biology **(ISSN: 2331-2025) is published by Cogent OA, part of Taylor & Francis Group.**