Modeling Earth Systems and Environment (2024) 10:6309–6322
https://doi.org/10.1007/s40808-024-02124-x

ORIGINAL ARTICLE

Mathematical modeling of Ebola using delay differential equations

Ali Raza[1] · Nauman Ahmed[2,5] · Muhammad Rafiq[3,5] · Ali Akgül[4,5] · Alicia Cordero[6] · Juan R. Torregrosa[6]

Received: 10 June 2024 / Accepted: 31 July 2024 / Published online: 28 August 2024

Abstract

Nonlinear delay differential equations (NDDEs) are essential in mathematical epidemiology, computational mathematics, sciences, etc. In this research paper, we have presented a delayed mathematical model of the Ebola virus to analyze its transmission dynamics in the human population. The delayed Ebola model is based on the four human compartments susceptible, exposed, infected, and recovered (SEIR). A time-delayed technique is used to slow down the dynamics of the host population. Two significant stages are analyzed in the said model: Ebola-free equilibrium (EFE) and Ebola-existing equilibrium (EEE). Also, the reproduction number of a model with the sensitivity of parameters is studied. Furthermore, the local asymptotical stability (LAS) and global asymptotical stability (GAS) around the two stages are studied rigorously using the Jacobian matrix Routh–Hurwitz criterion strategies for stability and Lyapunov function stability. The delay effect has been observed in the model in inverse relation of susceptible and infected humans (it means the increase of delay tactics that the susceptibility of humans increases and the infectivity of humans decreases eventually approaches zero which means that Ebola has been controlled into the population). For the numerical results, the Euler method is designed for the system of delay differential equations (DDEs) to verify the results with an analytical model analysis.

Keywords Ebola disease · Delay differential equations (DDE's) · Reproduction number and sensitivity analysis · Lyapunov function · Stability analysis · Numerical results

Introduction

The Ebola virus was found in the year 1976. When two successive cases of deadly hemorrhagic fever occurred in several parts of middle Africa. The first case happened in the Republic of Congo (previously Zaire) in a village near the Ebola River, which gave the name to the infection. The second case occurred in what is currently South Sudan. Symptoms appear 2–20 days after interaction with the infection, with an average of 8–10 days. Rhoubari et al. (2018) developed the epizootic SEIR model for transmitting the Ebola virus in bats. They proved the model is epidemiologically and mathematically well-stated by showing the solutions' presence, positivity, and boundness. Also analyzed the equations and developed appropriate Lyapunov functional to check the stability of equilibrium points. Berge et al. (2017) described how the Ebola virus was transmitted in society from the infected wild animal meat and funeral practices in Africa. Ivorra et al. (2015) presented a mathematical model for the spread of Ebola virus disease between the regions in 2014–2015. Chretien et al. (2015) reviewed 66 models of the Ebola virus and proposed steps to use mathematical

Nauman Ahmed, Muhammad Rafiq, Ali Akgül, Alicia Cordero, and Juan R. Torregrosa contributed equally to this work.

✉ Juan R. Torregrosa
jrtorre@mat.upv.es

[1] Department of Mathematics, Faculty of Physical Sciences, The University of Chenab, 50700 Gujrat, Pakistan

[2] Department of Mathematics and Statistics, The University of Lahore, 54590 Lahore, Pakistan

[3] Department of Mathematics, Faculty of Science and Technology, University of Central Punjab, 54000 Lahore, Pakistan

[4] Art and Science Faculty, Department of Mathematics, Siirt University, Siirt 56100, Turkey

[5] Department of Computer Science and Mathematics, Lebanese American University, 1102-2801 Beirut, Lebanon

[6] Multidisciplinary Institute of Mathematics, Universitat Politècnica de València, València 46022, Spain

modeling to analyze the disease. Chen (2015) proposed a mathematical model for the Ebola virus, which is based on the SIR model, and explained that the Ebola virus is very severe. The virus is transferred to humans through infected animal contact and then spreads from person to person.

Nazir et al. (2020) proposed the advanced SIR model for spreading the Ebola virus using the derivative. They explained every possible way of spreading the Ebola virus and how the virus spreads between individuals. It also gave measures of how the Ebola virus can be controlled. Chowell and Nishimura (2014) reviewed transmission elements and control of Ebola infection disease and epidemiological information from past Ebola outbreaks. Osemwinyen and Diakhaby (2015) analyzed two mathematical Ebola Zaire virus transmission models. Also described, a recovered person becomes infected again and dies at a specific rate and developed a quarantine model which describes the impacts of isolating. Moreover, a system of differential equations was developed, and a linearized stability approach was used to solve the equations. Wester (2015) evaluated that the Ebola virus affects the immune system during the infection period and used numerical modeling to investigate and analyze the immune system in the presence of the Ebola virus. Rachah (2018) researched that Ebola infection is one of the most destructive micro-organisms for people and presented an SEIR mathematical model of the Ebola virus disease. Salem and Smith (2016) examined a mathematical model of Ebola infection, utilizing awareness investigation to decide effective targets. Mathematical models provide a ground to search out real-world problems. Banton et al. (2010) designed a mathematical model for the vaccine for the Ebola virus and understanding how the immune system works.

More noteworthy comprehension can be accomplished through numerical models. Almuqrin et al. (2021) presented a Fractional model of Ebola infection in the populace of bats in the format of the Atangana–Baleanu fractional derivative. Proof of the answer's presence, uniqueness, and stability for the fractional numerical model was introduced. A mathematical methodology is utilized to calculate the solution of the expressed model, and the outcomes are addressed graphically. Dokuyucu and Dutta (2020) reviewed the model of the Ebola virus, which was quickly spreading in specific areas of Africa and was revised by using the fractional derivative without a singular kernel proposed by Caputo and Fabrizio. The model was rearranged, getting improved results from the model. In the main stage, the Ebola infection model was enlarged to the Caputo–Fabrizio fractional derivative. Later, the presence and uniqueness of the solution were derived for the fractional Ebola model through a fixed-point hypothesis. Then, mathematical results were acquired from the model by utilizing Atangana and Owolabi's new mathematical methodology through the Adam–Basford strategy for the Caputo–Fabrizio partial derivative. Mathematical advancement introduced for different results of fractional order derivatives. Tahir et al. (2019) prepared a SEIVR mathematical model of the Ebola virus, its spread, and control.

The reproduction number was calculated using the next-generation method, sensitivity analysis, and equilibrium points related to the disease were derived. The numerical interpretation presented with and without vaccination to control the disease. Abdalla et al. (2022) reviewed a mathematical model of the Ebola disease systematic way. Their work aims to identify the research gaps in the Ebola virus disease to motivate future research. Deepa et al. (2015) prepared a mathematical transmission model of the Ebola disease. The transmission model gives a better understanding of the spread of disease. Wild animals are a major carrier of the Ebola disease virus. Latha et al. (2018) discussed a fractional order model of Ebola disease using a delayed immune system with a time-delayed system. The Laplace transformation was used to obtain stability conditions. In Abah et al. (2024) the authors created a mathematical model including awareness initiatives and quarantines as control strategies. In Ren and Xu (2024) the authors developed an Ebola virus disease (EVD) model that describes the moment of incubation utilizing four propagation forms and an estimated delay. In Nisar et al. (2024) the authors created a unique individual combination technique that employed a nonlinear fractionally ordered Ebola virus to study the dynamical transmission through different compartments. In Nash et al. (2024) the authors examined the models and parameters used to describe the development, natural history, severity, and risk factors of Ebola virus disease (EVD) propagation. In Lolika et al. (2024) the authors suggested that the approach includes treatment as a control approach, public health campaigns, measures to prevent disease, and all essential biological aspects. In Xu and Farman (2023) the authors created a fractional-order Ebola virus model using a constant proportional Caputo (CPC) operator to examine the dynamical transmission of the illness as it is affected by vaccination, learning, early detection, cleanliness laws, isolation, and mobility restrictions. Mathematical epidemiology is a study field in which a mathematical and statistical model is used to predict the infectious disease is present or not in the host population. Mathematical techniques are used to analyze data to make predictions about the dynamics of disease. To simulate the spread of infectious diseases, mathematical epidemiological models are used. The main aim of mathematical epidemiology is to inform about public health and to help control and prevent infectious disease outbreaks. Delay differential equations (DDEs) are differential equations where the derivative of differential equations not only depends on their present value but also on values from the past times. DDEs are the rate of change in the history of the infectious disease. Delayed differential equations are more complex than ordinary differential equations and are more difficult to analyze. DDEs have applications in mathematics,

physics, chemistry, and biology. Lyapunov Function is a scalar function used to determine the stability of the dynamics of the disease. It is used to determine the global stability of the dynamic system. Lyapunov Functions are widely used in the control system and the analysis of the system's stability and for the convergence of the dynamical system (Table 1).

Model formulation

For evaluating the spread of the Ebola infection, an Ebola delayed epidemic model is presented in Rafiq et al. (2020). We distribute the total population $N(t)$ into four compartments: $S(t)$, $E(t)$, $I(t)$, and $R(t)$. Here $S(t)$, $E(t)$, $I(t)$ and $R(t)$ depict susceptible, exposed, infected and recovered humans. The values of the parameters used in the underlying model are shown in Table 2.

The system of differential equations of the Ebola delayed epidemic model presented in Fig. 1 is described as follows:

$$\begin{aligned}\frac{dS(t)}{dt} =&\lambda - \mu S(t) - (\beta_1 + \beta_4 + \beta_6)S(t-\tau)W(t)\\ &- (\beta_5 + \beta_7)S(t-\tau)I(t-\tau)e^{-\mu\tau},\end{aligned} \tag{1}$$

$$\begin{aligned}\frac{dE(t)}{dt} =&(\beta_1 + \beta_4 + \beta_6)S(t-\tau)W(t) -\\ &\beta_2 E(t-\tau)I(t-\tau)e^{-\mu\tau} - (\mu_{1+}\mu_2)E(t),\end{aligned} \tag{2}$$

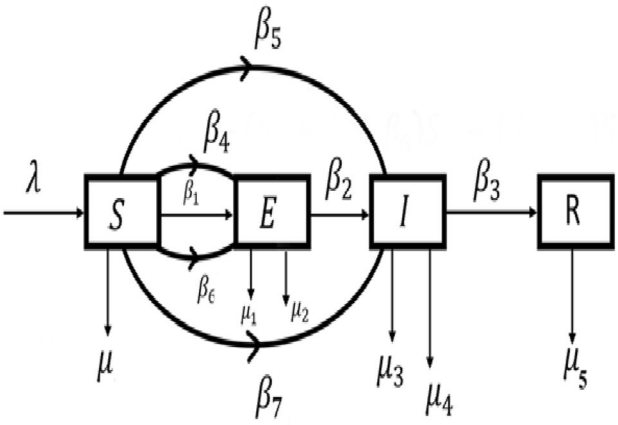

Fig. 1 Flow chart of the Ebola epidemic

$$\begin{aligned}\frac{dI(t)}{dt} =&\beta_2 I(t-\tau)W(t) + (\beta_5 + \beta_7)S(t-\tau)I(t-\tau)e^{-\mu\tau}\\ &- (\beta_3 + \mu_3 + \mu_4)I(t),\end{aligned} \tag{3}$$

$$\frac{dR(t)}{dt} =\beta_3 I(t) - \mu_5 R(t), \tag{4}$$

where $W(t) = E(t-\tau)e^{-\mu\tau}$, and with non-negative initial conditions $S(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, and $R(0) \geq 0$.

Table 1 Parameter values used in the underlying model

Notation	Descriptions	Values
λ	Newborn rate to human population	0.06321 (EFE), 2.06321 (EEE)
μ	The natural death rate of susceptible persons	0.9704
β_1	The infection rate of susceptible to exposed persons	0.2877
β_2	Infection rate from exposure to infected persons	0.7613
β_3	Infection rate from infected to recovered persons	0.4389
β_4	Infection ratio of susceptible wild animals to exposed persons	0.1234
β_5	Infection ratio of susceptible wild animals to exposed persons	0.2431
β_6	Infection ratio of susceptible wild animals to exposed persons	0.4000
β_7	Infection ratio of susceptible wild animals to exposed persons	0.3000
μ_1	The natural death rate of exposed persons	0.0432
μ_2	The sickness-induced death rate of exposed persons	0.2006
μ_3	The natural death rate of infected humans	0.0656
μ_4	The sickness-induced death rate of infected persons	0.9764
μ_5	The natural death rate of recovered persons	0.6704
R_0	Reproduction number	$0.2167 < 1$ (EFE), $7.0735 > 1$ (EEE)

Reproduction number

The parameter R_0 is used to measure the spread rate of any disease. The disease will exist in the population if the infected individual is present in the host population. R_0 is the ratio of new diseases coming from the original infection, and determines the behavior of the illness. There will be no disease in the human population if $R_0 < 1$ and disease will exist if $R_0 > 1$.

In what follows, F and V are the transmission and transition matrices, respectively

$$F = \begin{bmatrix} (\beta_1+\beta_4+\beta_6)Se^{-\mu\tau} & 0 \\ 0 & (\beta_5+\beta_7)Se^{-\mu\tau} \end{bmatrix},$$

$$V = \begin{bmatrix} \mu_{1+}\mu_2 & 0 \\ 0 & \beta_3+\mu_3+\mu_4 \end{bmatrix}.$$

They are calculated by using next generation matrix method (see Table 2 for the parameters involved). We use them to calculate the basic reproductive number of an infection. It is the expected number of cases directly generated by one case in a population where all individuals are susceptible to infection and whether disease dies out or persist in population.

Then,

$$FV^{-1} = \begin{bmatrix} \frac{(\beta_1+\beta_4+\beta_6)Se^{-\mu\tau}}{\mu_{1+}\mu_2} & 0 \\ 0 & \frac{(\beta_5+\beta_7)Se^{-\mu\tau}}{\beta_3+\mu_3+\mu_4} \end{bmatrix},$$

$$FV^{-1} = \begin{bmatrix} \frac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_{1+}\mu_2)} & 0 \\ 0 & \frac{(\beta_5+\beta_7)\lambda e^{-\mu\tau}}{(\beta_3+\mu_3+\mu_4)\mu} \end{bmatrix} = A.$$

For calculating the eigenvalues, we solve

$$|A - \lambda_1 I| = 0,$$

that is,

$$\begin{vmatrix} \frac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_{1+}\mu_2)} - \lambda_1 & 0 \\ 0 & \frac{(\beta_5+\beta_7)\lambda e^{-\mu\tau}}{(\beta_3+\mu_3+\mu_4)\mu} - \lambda_1 \end{vmatrix} = 0.$$

By expanding the determinant, the largest eigenvalue is

$$\lambda_1 = \frac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_1+\mu_2)}.$$

Hence, $R_0 = \frac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_1+\mu_2)}$.

Ebola free equilibrium

For Ebola Free Equilibrium (EFE), S, E, I, and R are constants, then equations (1), (2), (3), and (4) can be written as follows.

From (1),

$$\lambda - \mu S - (\beta_1+\beta_4+\beta_6)SEe^{-\mu\tau} - (\beta_5+\beta_7)SIe^{-\mu\tau} = 0,$$

$$\lambda - \mu S - (\beta_1+\beta_4+\beta_6)S(0)e^{-\mu\tau} - (\beta_5+\beta_7)S(0)e^{-\mu\tau} = 0,$$

$$\lambda - \mu S = 0,$$

$$S = \frac{\lambda}{\mu}.$$

From (2),

$$(\beta_1+\beta_4+\beta_6)SEe^{-\mu\tau} - \beta_2 EIe^{-\mu\tau} - (\mu_{1+}\mu_2)E = 0,$$

$$(\beta_1+\beta_4+\beta_6)S(0)e^{-\mu\tau} - \beta_2(0)(0)e^{-\mu\tau} - (\mu_{1+}\mu_2)0 = 0,$$

$$0 = 0.$$

From (3),

$$\beta_2 EIe^{-\mu\tau} + (\beta_5+\beta_7)SIe^{-\mu\tau} - (\beta_3+\mu_3+\mu_4)I = 0,$$

$$\beta_2(0)(0)e^{-\mu\tau} + (\beta_5+\beta_7)S(0)e^{-\mu\tau} - (\beta_3+\mu_3+\mu_4)(0) = 0,$$

$$0 = 0.$$

From (4), $\beta_3 I - \mu_5 R = 0$, and therefore

$$\beta_3(0) - \mu_5(0) = 0,$$

$$0 = 0.$$

As $S \neq 0$, and $E = I = R = 0$,

$$S = \frac{\lambda}{\mu},\ E = 0,\ I = 0\ R = 0,$$

so

$$F_0 = (S^0, E^0, I^0, R^0) = \left(\frac{\lambda}{\mu}, 0, 0, 0\right).$$

Ebola existing equilibrium

For Ebola Existing Equilibrium (EEE), S, E, I, and R are constants and their derivative is always zero. Equations (1), (2), (3), and (4) can be written as follows.

From (2), we get

$$E\left[\left(\beta_1+\beta_4+\beta_6\right)Se^{-\mu\tau}-\beta_2Ie^{-\mu\tau}-\left(\mu_{1+}\mu_2\right)\right]=0.$$

As $E\neq 0$, so $\left(\beta_1+\beta_4+\beta_6\right)Se^{-\mu\tau}-\beta_2Ie^{-\mu\tau}-\left(\mu_{1+}\mu_2\right)=0$, and

$$I=\frac{\left(\beta_1+\beta_4+\beta_6\right)S}{\beta_2}-\frac{\mu_{1+}\mu_2}{\beta_2e^{-\mu\tau}}.$$

Now, from (3)

$$I\left[\beta_2Ee^{-\mu\tau}+\left(\beta_5+\beta_7\right)Se^{-\mu\tau}-\left(\beta_3+\mu_3+\mu_4\right)\right]=0.$$

As$I\neq 0$,so$\beta_2Ee^{-\mu\tau}+\left(\beta_5+\beta_7\right)Se^{-\mu\tau}-\left(\beta_3+\mu_3+\mu_4\right)=0$, and

$$E=\frac{\beta_3+\mu_3+\mu_4}{\beta_2e^{-\mu\tau}}-\frac{\left(\beta_5+\beta_7\right)S}{\beta_2}.$$

By replacing the value of I and E in (1), we get

$$\lambda-\mu S-\left(\beta_1+\beta_4+\beta_6\right)S\left[\frac{\beta_3+\mu_3+\mu_4}{\beta_2e^{-\mu\tau}}-\frac{\left(\beta_5+\beta_7\right)S}{\beta_2}\right]e^{-\mu\tau}-\left(\beta_5+\beta_7\right)S\left[\frac{\left(\beta_1+\beta_4+\beta_6\right)S}{\beta_2}-\frac{\mu_{1+}\mu_2}{\beta_2e^{-\mu\tau}}\right]e^{-\mu\tau}=0,$$

that is,

$$\lambda-\mu S-\frac{\left(\beta_1+\beta_4+\beta_6\right)\left(\beta_3+\mu_3+\mu_4\right)S}{\beta_2}+\frac{\left(\beta_5+\beta_7\right)\left(\mu_{1+}\mu_2\right)S}{\beta_2}=0,$$

$$\mu S+\frac{\left(\beta_1+\beta_4+\beta_6\right)\left(\beta_3+\mu_3+\mu_4\right)S}{\beta_2}-\frac{\left(\beta_5+\beta_7\right)\left(\mu_{1+}\mu_2\right)S}{\beta_2}=\lambda,$$

and therefore,

$$S^*=\frac{\lambda}{\mu+\frac{1}{\beta_2}\left[\left(\beta_1+\beta_4+\beta_6\right)\left(\beta_3+\mu_3+\mu_4\right)-\left(\beta_5+\beta_7\right)\left(\mu_{1+}\mu_2\right)\right]}.$$

Let $\pi=\frac{1}{\beta_2}\left[\left(\beta_1+\beta_4+\beta_6\right)\left(\beta_3+\mu_3+\mu_4\right)-\left(\beta_5+\beta_7\right)\left(\mu_{1+}\mu_2\right)\right]$. So,

$$S^*=\frac{\lambda}{\mu+\pi},$$

and then,

$$E^*=\frac{\beta_3+\mu_3+\mu_4}{\beta_2e^{-\mu\tau}}-\frac{\left(\beta_5+\beta_7\right)\lambda}{\beta_2(\mu+\pi)},\ \text{or}$$

$$E^*=\frac{\left(\beta_3+\mu_3+\mu_4\right)(\mu+\pi)-\lambda\left(\beta_5+\beta_7\right)e^{-\mu\tau}}{\beta_2(\mu+\pi)e^{-\mu\tau}},$$

and

$$I^*=\frac{\lambda\left(\beta_1+\beta_4+\beta_6\right)}{\beta_2(\mu+\pi)}-\frac{\mu_{1+}\mu_2}{\beta_2e^{-\mu\tau}},\ \text{or}$$

$$I^*=\frac{\lambda\left(\beta_1+\beta_4+\beta_6\right)e^{-\mu\tau}-\left(\mu_{1+}\mu_2\right)(\mu+\pi)}{\beta_2(\mu+\pi)e^{-\mu\tau}}.$$

From (4),

$$\beta_3I-\mu_5R=0,$$

so

$$R=\frac{\beta_3I}{\mu_5},$$

and

$$R^*=\frac{\lambda\beta_3\left(\beta_1+\beta_4+\beta_6\right)e^{-\mu\tau}-\beta_3\left(\mu_{1+}\mu_2\right)(\mu+\pi)}{\mu_5\beta_2(\mu+\pi)e^{-\mu\tau}}.$$

Therefore, the Endemic point is

$$F_1=\left(S^1,E^1,I^1,R^1\right)=\left(\frac{\lambda}{\mu+\pi},\frac{\left(\beta_3+\mu_3+\mu_4\right)(\mu+\pi)-\lambda\left(\beta_5+\beta_7\right)e^{-\mu\tau}}{\beta_2(\mu+\pi)e^{-\mu\tau}},\frac{\lambda\left(\beta_1+\beta_4+\beta_6\right)e^{-\mu\tau}-\left(\mu_{1+}\mu_2\right)(\mu+\pi)}{\beta_2(\mu+\pi)e^{-\mu\tau}},\frac{\lambda\beta_3\left(\beta_1+\beta_4+\beta_6\right)e^{-\mu\tau}-\beta_3\left(\mu_{1+}\mu_2\right)(\mu+\pi)}{\mu_5\beta_2(\mu+\pi)e^{-\mu\tau}}\right),$$

where $\pi=\frac{1}{\beta_2}\left[\left(\beta_1+\beta_4+\beta_6\right)\left(\beta_3+\mu_3+\mu_4\right)-\left(\beta_5+\beta_7\right)\left(\mu_{1+}\mu_2\right)\right]$.

Positivity and boundness of the model

Now, we analyze the positivity and boundness of the Ebola delayed epidemic model. To get this aim, $S(t)$, $E(t)$, $I(t)$, $R(t)$ must be non-negative. Then, the results remain positive and bounded for any $t\geq 0$, $\tau\leq t$ in a feasible region,

$$\bar{t}=\sup\{t>0:\ S(t)\geq 0,\ E(t)\geq 0,\ I(t)\geq 0,\ R(t)\geq 0\}.$$

Theorem 1 *Solutions* $S,\ E,\ I,\ R \in \mathbb{R}^4_+$ *of the system* (1)–(4) *are positive at any time* $t \geq 0$ *with given non-negative initial conditions.*

Proof Let us suppose that $S(0) \geq 0$. Equation (1) leads to

$$\begin{aligned}\frac{dS}{dt} =&\lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \left(\beta_5+\beta_7\right)SIe^{-\mu\tau}\\ =&\lambda - \left[\mu + \left(\beta_1 + \beta_4 + \beta_6\right)Ee^{-\mu\tau} + \left(\beta_5+\beta_7\right)Ie^{-\mu\tau}\right]S.\end{aligned}$$

Let us define $f(t) = \left(\beta_1 + \beta_4 + \beta_6\right)Ee^{-\mu\tau} + \left(\beta_5+\beta_7\right)Ie^{-\mu\tau}$. By multiplying both sides of the previous expression by $e^{(\mu t+\int_0^t f(t)dt)} > 0$,

$$\frac{d}{dt}\left[Se^{(\mu t+\int_0^t f(t)dt)}\right] = \lambda e^{(\mu t+\int_0^t f(t)dt)}.$$

Now, integrating at both sides from $t = 0$ to $t = \bar{t}$, we obtain

$$S(\bar{t})e^{(\mu\bar{t}+\int_0^{\bar{t}} f(t)dt)} - S(0) = \lambda\int_0^{\bar{t}} e^{(\mu y+\int_0^y f(x)dxdy)}.$$

By multiplying both sides by $e^{(-\mu\bar{t}-\int_0^{\bar{t}} f(t)dt)} > 0$, we get

$$\begin{aligned}S(\bar{t}) =&S(0)e^{(-\mu\bar{t}-\int_0^{\bar{t}} f(t)dt)}\\ &+ \lambda e^{(-\mu\bar{t}-\int_0^{\bar{t}} f(t)dt)}\int_0^{\bar{t}} e^{(\mu y+\int_0^y f(x)dx)dy)}.\end{aligned}$$

As $S(0) \geq 0$, the sum of the positive terms S is positive. Also,

$$\begin{aligned}\left.\frac{dS}{dt}\right|_{S=0} &= \lambda \geq 0,\\ \left.\frac{dE}{dt}\right|_{E=0} &= 0 \geq 0,\\ \left.\frac{dI}{dt}\right|_{I=0} &= 0 \geq 0,\\ \left.\frac{dR}{dt}\right|_{R=0} &= \beta_3 I \geq 0,\end{aligned}$$

that is the desired result.

Theorem 2 *Solutions* $(S,\ E,\ I,\ R) \in \mathbb{R}^4_+$ *of system* (1)–(4) *are all bounded.*

Proof The total amount of population is denoted by Z, $Z(t) = S(t) + E(t) + I(t) + R(t)$. By differentiating $Z(t)$, we get that

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

Using (1)–(4), we get

$$\begin{aligned}\frac{dZ}{dt} =&\lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \left(\beta_5+\beta_7\right)SIe^{-\mu\tau}\\ &+ \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \beta_2 EIe^{-\mu\tau} - (\mu_{1+}\mu_2)E\\ &+ \beta_2 EIe^{-\mu\tau}\\ &+ \left(\beta_5 + \beta_7\right)SIe^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right)I\\ &+ \beta_3 I - \mu_5 R,\end{aligned}$$

so,

$$\frac{\mathrm{dZ}}{\mathrm{dt}} = \lambda - \mu S - (\mu_3 + \mu_4)I - (\mu_{1+}\mu_2)E - \mu_5 R.$$

let us assume, for any initial setting, $Z(0) = S(0) + E(0) + I(0) + R(0)$, $Z(t) \leq \frac{\lambda}{\mu}$, $\forall t \geq 0$. This implies that

$$\frac{dZ}{dt} = \lambda - \mu S.$$

The Grown Wall's inequality says

$$Z(t) \leq \frac{\lambda}{\mu} + \left(Z(0) + \frac{\lambda}{\mu}\right)e^{-\mu t},$$

and hence

$$Z(t) \leq \frac{\lambda}{\mu},\ \forall t \geq 0,$$

whenever $Z(0) \leq \frac{\lambda}{\mu}$.

It is clear that,

$$\lim_{t\to+\infty} \sup Z(t) \leq \frac{\lambda}{\mu}.$$

Then, $Z(t)$ and all other variables S, E, I, and R of the model (1)–(4) are all bounded. Therefore, SEIR models (1)–(4) lie in a biologically feasible region.

Methodology

In this section, for the theoretical analysis of delay differential equations (DDEs) of the Ebola virus model, We begin by identifying the equilibrium points of the system and then use the Routh–Hurwitz Criteria to determine their local stability through the linearization of the system and analysis of the Jacobian matrix. For global stability, we construct appropriate Lyapunov functions and apply the Lyapunov stability theorem to show global asymptotic stability. Additionally, we perform a sensitivity analysis by varying key model parameters to assess their impact on the stability of the equilibria.

Local stability

In this section, we are going to study the stability of the *The Ebola-Free Equilibrium*.

Theorem 3 *The Ebola-Free Equilibrium* $F_0 = \left(\frac{\lambda}{\mu}, 0, 0, 0\right)$ *of model* (1)–(4) *is locally asymptotically stable* (*LAS*) *whenever* $R_0 < 1$ *and is unstable for* $R_0 > 1$.

Proof

$$\frac{dS}{dt} = \lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \left(\beta_5+\beta_7\right)SIe^{-\mu\tau},$$

$$\frac{dE}{dt} = \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \beta_2 EIe^{-\mu\tau} - \left(\mu_{1+}\mu_2\right)E,$$

$$\frac{dI}{dt} = \beta_2 EIe^{-\mu\tau} + \left(\beta_5 + \beta_7\right)SIe^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right)I,$$

$$\frac{dR}{dt} = \beta_3 I - \mu_5 R.$$

The Jacobian matrix of the system is

$$J(F_0) = \begin{bmatrix} -\mu & -\frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} & -\frac{\lambda}{\mu}\left(\beta_5+\beta_7\right)e^{-\mu\tau} & 0 \\ 0 & \frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} - \left(\mu_{1+}\mu_2\right) & 0 & 0 \\ 0 & 0 & \frac{\lambda}{\mu}\left(\beta_5 + \beta_7\right)e^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right) & 0 \\ 0 & 0 & \beta_3 & -\mu_5 \end{bmatrix}.$$

Now $\left|J\left(F_0\right) - \Lambda I\right| = 0.$

$$\begin{vmatrix} -\mu - \Lambda & -\frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} & -\frac{\lambda}{\mu}\left(\beta_5+\beta_7\right)e^{-\mu\tau} & 0 \\ 0 & \frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} - \left(\mu_{1+}\mu_2\right) - \Lambda & 0 & 0 \\ 0 & 0 & \frac{\lambda}{\mu}\left(\beta_5 + \beta_7\right)e^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right) - \Lambda & 0 \\ 0 & 0 & \beta_3 & -\mu_5 - \Lambda \end{vmatrix} = 0.$$

Expanding column 1, we get

$$(-\mu - \Lambda)\begin{vmatrix} \frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} - \left(\mu_{1+}\mu_2\right) - \Lambda & 0 & 0 \\ 0 & \frac{\lambda}{\mu}\left(\beta_5 + \beta_7\right)e^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right) - \Lambda & 0 \\ 0 & \beta_3 & -\mu_5 - \Lambda \end{vmatrix} = 0.$$

Now the determinant is lower triangular, so we get

$$(-\mu - \Lambda)\left[\frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} - \left(\mu_{1+}\mu_2\right) - \Lambda\right]\left[\frac{\lambda}{\mu}\left(\beta_5 + \beta_7\right)e^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right) - \Lambda\right]\left(-\mu_5 - \Lambda\right) = 0.$$

Therefore,

$$-\mu - \Lambda = 0, \tag{5}$$

or

$$\frac{\lambda}{\mu}\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau} - \left(\mu_{1+}\mu_2\right) - \Lambda = 0, \tag{6}$$

or

$$\frac{\lambda}{\mu}\left(\beta_5 + \beta_7\right)e^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right) - \Lambda = 0, \tag{7}$$

or

$$-\mu_5 - \Lambda = 0. \tag{8}$$

From (5), we have $\Lambda_1 = -\mu$. Then $\Lambda_1 < 0$ because $\mu > 0$.

From (7), we get $\Lambda_3 = \frac{\lambda}{\mu}(\beta_5 + \beta_7)e^{-\mu\tau} - (\beta_3 + \mu_3 + \mu_4)$, so $\Lambda_3 < 0$ since

$$\frac{\lambda}{\mu}(\beta_5 + \beta_7)e^{-\mu\tau} < (\beta_3 + \mu_3 + \mu_4).$$

From (8), we deduce $\Lambda_4 = -\mu_5$, so $\Lambda_4 < 0$ because $\mu_5 > 0$.

Finally, from Eq. (6), $\lambda_2 = \frac{\lambda}{\mu}(\beta_1 + \beta_4 + \beta_6)e^{-\mu\tau} - \left(\mu_{1+}\mu_2\right)$, that is,

$$\Lambda_2 = \frac{\lambda}{\mu}\frac{(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu_{1+}\mu_2}(\mu_{1+}\mu_2) - (\mu_{1+}\mu_2).$$

Then,

$$\Lambda_2 = R_0(\mu_{1+}\mu_2) - (\mu_{1+}\mu_2),$$

since $R_0 = \dfrac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_{1+}\mu_2)}$. Therefore,

$\Lambda_2 < 0$ if and only if $R_0 - 1 < 0$ or $R_0 < 1$.

Now, if $R_0 < 1$ then all eigenvalues are negative, therefore Ebola Free Equilibrium point $F_0(S^0, E^0, I^0, R^0) = \left(\frac{\lambda}{\mu}, 0, 0, 0\right)$ of the system (1)–(4) is LAS. There is no Ebola virus in the host population.

Moreover, if $R_0 > 1$, then $\dfrac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_{1+}\mu_2)} > 1$, that is,

$$\frac{\lambda}{\mu}(\beta_1+\beta_4+\beta_6)e^{-\mu\tau} > \mu_{1+}\mu_2 \;\Rightarrow\; \frac{\lambda}{\mu}(\beta_1+\beta_4+\beta_6)e^{-\mu\tau} - (\mu_{1+}\mu_2) > 0.$$

Then from (6) we get $\Lambda_2 > 0$ and hence F_0 is unstable if $R_0 > 1$.

Theorem 4 *The Ebola-Existing Equilibrium (EEE-F_1) of the model (1)–(4) is locally asymptotically stable (LAS) whenever $R_0 < 1$ and is unstable for $R_0 > 1$.*

Proof The Jacobian matrix of the system (1)–(4) at endemic equilibrium point is presented as

$$J(F_1) = \begin{bmatrix} -E_1 & -E_4 & -E_6 & 0 \\ E_2 & & -E_7 & 0 \\ -E_3 & E_5 & & 0 \\ 0 & 0 & \beta_3 & -\mu_5 \end{bmatrix}$$

and

$$\left|J(F_1) - \Lambda I\right| = \begin{vmatrix} -E_1-\Lambda & -E_4 & -E_6 & 0 \\ E_2 & -\Lambda & -E_7 & 0 \\ -E_3 & E_5 & -\Lambda & 0 \\ 0 & 0 & \beta_3 & -\mu_5-\Lambda \end{vmatrix} = 0.$$

Then,

$$\mu_5 + \Lambda = 0,\ \Lambda_1 = -\mu_5 < 0,\ \text{ or } \begin{vmatrix} -E_1-\Lambda & -E_4 & -E_6 \\ E_2 & -\Lambda & -E_7 \\ -E_3 & E_5 & -\Lambda \end{vmatrix} = 0.$$

Therefore,

$$-E_1 - \Lambda(\Lambda^2 + E_5E_7) + E_4(-\Lambda E_2 - E_3E_7) - E_6(E_2E_5 - \Lambda E_3) = 0,$$

$$\Lambda^3 + E_1\Lambda^2 + (E_5E_7 + E_2E_4 - E_3E_6)\Lambda + E_3E_7E_4 + E_2E_5E_6 = 0.$$

Then, according to Routh–Hurwitz,

$$\Lambda^3 + a_2\Lambda^2 + a_1\Lambda + a_0 = 0,$$

where

$$a_2 = E_1,$$
$$a_1 = E_5E_7 + E_2E_4 - E_3E_6,$$
$$a_0 = E_3E_7E_4 + E_2E_5E_6,$$

being a_2,a_1 and a_0 all positive and $a_2a_1 > a_0$.

So, according to Routh–Hurwitz, system is stable at F_1.

$$E_1 = -\frac{1}{\beta_2}\left[\mu\beta_2 + (\beta_1+\beta_4+\beta_6)(\beta_3+\mu_3+\mu_4)e^{-\mu\tau} + (\beta_5+\beta_7)(\mu_{1+}\mu_2)e^{-\mu\tau}\right],$$

$$E_2 = -\frac{1}{\beta_2(\pi+\mu)}\left[-(\beta_1+\beta_4+\beta_6)(\beta_3+\mu_3 + \mu_4)(\pi+\mu)e^{-\mu\tau} + \lambda(\beta_5+\beta_7)(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}\right],$$

$$E_3 = -\frac{1}{\beta_2(\pi+\mu)}\left[-\lambda(\beta_5+\beta_7)(\beta_1+\beta_4+\beta_6)e^{-\mu\tau} + (\beta_5+\beta_7)(\mu_{1+}\mu_2)e^{-\mu\tau}\right],$$

$$E_4 = \frac{\lambda}{\mu+\pi}(\beta_1+\beta_4+\beta_6)e^{-\mu\tau},$$

$$E_5 = \frac{\lambda}{\mu+\pi}(\beta_1+\beta_4+\beta_6)e^{-\mu\tau} - (\mu_{1+}\mu_2),$$

$$E_6 = \frac{\lambda}{\mu+\pi}(\beta_5+\beta_7)e^{-\mu\tau},$$

$$E_7 = \frac{\lambda}{\mu+\pi}(\beta_5+\beta_7)e^{-\mu\tau} - (\beta_3+\mu_3+\mu_4).$$

Sensitivity analysis of parameters

Each parameter in the model has an essential character in the disease dynamics. Sensitivity analysis of each parameter of reproduction is given by

$$P_\lambda = \frac{\partial R_0}{\partial\lambda}\frac{\lambda}{R_0} = \frac{\lambda}{R_0}\frac{\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}}{\mu(\mu_1+\mu_2)} = \lambda > 0,$$

$$P_{\beta_1} = \frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} = \frac{\beta_1}{\beta_1+\beta_4+\beta_6} > 0,$$

$$P_{\beta_4} = \frac{\partial R_0}{\partial \beta_4} \times \frac{\beta_4}{R_0} = \frac{\beta_4}{\beta_1+\beta_4+\beta_6} > 0,$$

$$P_{\beta_6} = \frac{\partial R_0}{\partial \beta_6} \times \frac{\beta_6}{R_0} = \frac{\beta_6}{\beta_1+\beta_4+\beta_6} > 0,$$

$$P_{\mu} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} = \frac{\lambda(\beta_1+\beta_4+\beta_6)(-\mu^{-2}e^{-\mu\tau} - \tau\mu^{-1}e^{-\mu\tau})}{\mu_{1+}\mu_2} < 0,$$

$$P_{\mu_1} = \frac{\partial R_0}{\partial \mu_1} \times \frac{\mu_1}{R_0} = \frac{-\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}\mu_1^{-2}}{\mu} < 0,$$

$$P_{\mu_2} = \frac{\partial R_0}{\partial \mu_2} \times \frac{\mu_2}{R_0} = \frac{-\lambda(\beta_1+\beta_4+\beta_6)e^{-\mu\tau}\mu_2^{-2}}{\mu} < 0,$$

$$P_{\tau} = \frac{\partial R_0}{\partial \tau} \times \frac{\tau}{R_0} = \frac{-\lambda\left(\beta_1+\beta_4+\beta_6\right)e^{-\mu\tau}}{\left(\mu_{1+}\mu_2\right)} < 0.$$

Hence the sensitive parameters are λ, β_1, β_4, β_6 and μ, μ_1, μ_2, τ are not sensitive parameters.

Global stability

Theorem 5 *The Ebola-free equilibrium* $\left(\frac{\lambda}{\mu}, 0, 0, 0\right)$ *of the* (1)–(4) *is globally asymptotically stable* (*GAS*) *if* $R_0 > 1$ *and is unstable for* $R_0 > 1$.

Proof Consider a Volterra-type Lyapunov function $U : \Omega \to \mathbb{R}$ as given by

$$U = S - S^0 \ln S + E + I.$$

Taking the time derivative of U

$$\dot{U} = \left(1 - \frac{S^0}{S}\right)\dot{S} + \dot{E} + \dot{I}.$$

Then,

$$\begin{aligned}\dot{U} =& \left(1 - \frac{S^0}{S}\right)\left[\lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau}\right.\\ &\left.-\left(\beta_5 + \beta_7\right)SIe^{-\mu\tau}\right]\\ &+ \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \beta_2 EIe^{-\mu\tau}\\ &- \left(\mu_{1+}\mu_2\right)E + \beta_2 EIe^{-\mu\tau}\\ &+ \left(\beta_5 + \beta_7\right)SIe^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right)I,\end{aligned}$$

$$\begin{aligned}\dot{U} =& \lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} - \left(\beta_5+\beta_7\right)SIe^{-\mu\tau}\\ &- \frac{S^0}{S}[\lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau}\\ &- \left(\beta_5+\beta_7\right)SIe^{-\mu\tau}] + \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau}\\ &- \beta_2 EIe^{-\mu\tau} - \left(\mu_{1+}\mu_2\right)E + \beta_2 EIe^{-\mu\tau}\\ &+ \left(\beta_5 + \beta_7\right)SIe^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right)I,\end{aligned}$$

$$\begin{aligned}\dot{U} =& \lambda - \mu S - \frac{S^0}{S}[\lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau}\\ &- \left(\beta_5+\beta_7\right)SIe^{-\mu\tau}] - \left(\mu_{1+}\mu_2\right)E - \left(\beta_3 + \mu_3 + \mu_4\right)I,\end{aligned}$$

$$\begin{aligned}\dot{U} =& \lambda - \mu S - \frac{S^0}{S}\lambda + \frac{S^0}{S}\mu S + \frac{S^0}{S}\left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau}\\ &+ \frac{S^0}{S}\left(\beta_5+\beta_7\right)SIe^{-\mu\tau} - \left(\mu_{1+}\mu_2\right)E - \left(\beta_3 + \mu_3 + \mu_4\right)I.\end{aligned}$$

By putting $\lambda = \mu S^0$ in the above equation,

$$\begin{aligned}\dot{U} =& \mu S^0 - \mu S - \frac{\mu S^0 S^0}{S} + \frac{\mu S^0 S}{S}\\ &+ \left[S^0\left(\beta_5+\beta_7\right)e^{-\mu\tau} - \left(\beta_3 + \mu_3 + \mu_4\right)\right]I\\ &+ S^0\left(\beta_1 + \beta_4 + \beta_6\right)e^{-\mu\tau}E - \left(\mu_{1+}\mu_2\right)E,\end{aligned}$$

$$\begin{aligned}\dot{U} =& -\frac{\mu}{S}\left(S^{0^2} + S^2 - 2S^0 S\right) + \left[\frac{\lambda}{\mu}\left(\beta_5+\beta_7\right)e^{-\mu\tau}\right.\\ &\left.-\left(\beta_3 + \mu_3 + \mu_4\right)\right]I + \left(\mu_{1+}\mu_2\right)\left(R_0 - 1\right)E,\end{aligned}$$

$$\begin{aligned}\dot{U} =& -\frac{\mu}{S}\left(S - S^0\right)^2 + \left[\frac{\lambda}{\mu}\left(\beta_5+\beta_7\right)e^{-\mu\tau}\right.\\ &\left.-\left(\beta_3 + \mu_3 + \mu_4\right)\right]I + \left(\mu_{1+}\mu_2\right)\left(R_0 - 1\right)E.\end{aligned}$$

Since $\frac{\lambda}{\mu}(\beta_5+\beta_7)e^{-\mu\tau} < (\beta_3 + \mu_3 + \mu_4)$, it follows that $\dot{U}$ *leq*0 for $R_0 < 1$. Moreover, if $R_0 < 1$ then $\dot{U}$=0. That is equivalent to

$$S = S^0,\ E = 0,\ I = 0,\ \text{ whereas }\ \dot{U} = 0,$$
$$\forall (S, E, I, R) \neq (S^0, 0, 0, 0).$$

Theorem 6 *For $R_0 > 1$, the system (1)–(4) is globally asymptotically stable at $F_1 = (S^1, E^1, I^1, R^1)$.*

Proof Consider the Lyapunov function $V : \Omega \to \mathbb{R}$ defined as

$$V = K_1[S - S^1 \ln S] + K_2[E - E^1 \ln E] + K_3[I - I1 \ln I],$$

where K_1, K_2, and K_3 are positive constants to be chosen later.

$$\dot{V} = K_1\left(1 - \frac{S^1}{S}\right)\dot{S} + K_2\left(1 - \frac{E^1}{E}\right)\dot{E} + K_3\left(1 - \frac{I^1}{I}\right)\dot{I}.$$

Then,

$$\begin{aligned}
\dot{V} =& K_1\left(1 - \frac{S^1}{S}\right)\left[\lambda - \mu S - \left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau}\right. \\
&\left. -\left(\beta_5 + \beta_7\right)SIe^{-\mu\tau}\right] \\
&+ K_2\left(1 - \frac{E^1}{E}\right)\left(\beta_1 + \beta_4 + \beta_6\right)SEe^{-\mu\tau} \\
&- \beta_2 EIe^{-\mu\tau} - (\mu_1 + \mu_2)E \\
&+ K_3\left(1 - \frac{I^1}{I}\right)\beta_2 EIe^{-\mu\tau} + \left(\beta_5 + \beta_7\right)SIe^{-\mu\tau} \\
&- \left(\beta_3 + \mu_3 + \mu_4\right)I \\
=& K_1\left(S - S^1\right)\left[\frac{\lambda}{S} - \mu\right. \\
&\left. -\left(\beta_1 + \beta_4 + \beta_6\right)Ee^{-\mu\tau} - \left(\beta_5 + \beta_7\right)Ie^{-\mu\tau}\right] \\
&+ K_2\left(E - E^1\right)[\left(\beta_1 + \beta_4 + \beta_6\right)Se^{-\mu\tau} - \beta_2 Ie^{-\mu\tau} \\
&- \left(\mu_{1+}\mu_2\right)] \\
&+ K_3\left(I - I^1\right)[\beta_2 Ee^{-\mu\tau} + \left(\beta_5 + \beta_7\right)Se^{-\mu\tau} \\
&- \left(\beta_3 + \mu_3 + \mu_4\right)].
\end{aligned}$$

Since $F_1 = \left(S^1, E^1, I^1, R^1\right)$, so from the system (1)–(4),

$$\frac{dS^1}{dt} = \frac{dE^1}{dt} = \frac{dI^1}{dt} = 0,$$

being

$$\mu = \frac{\lambda}{S_1} - \left(\beta_1 + \beta_4 + \beta_6\right)E^1 e^{-\mu\tau} - \left(\beta_5 + \beta_7\right)I^1 e^{-\mu\tau},$$

$$\mu_1 + \mu_2 = [\left(\beta_1 + \beta_4 + \beta_6\right)S^1 e^{-\mu\tau} - \beta_2 I^1 e^{-\mu\tau}],$$

$$\beta_3 + \mu_3 + \mu_4 = [\beta_2 E^1 e^{-\mu\tau} + \left(\beta_5 + \beta_7\right)S^1 e^{-\mu\tau}].$$

So,

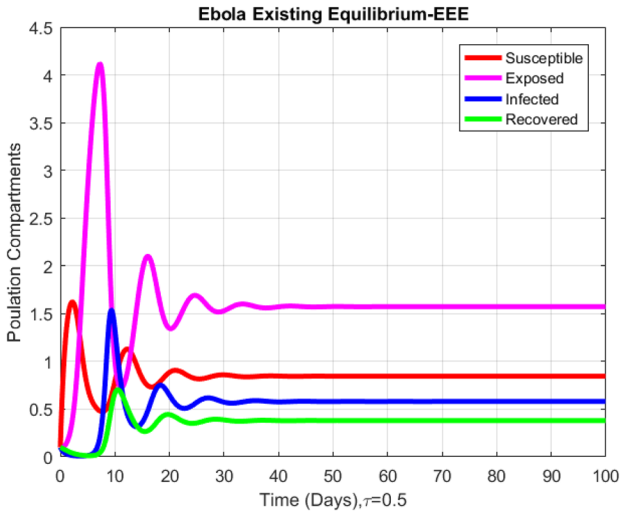

Fig. 2 Time delay graph of the system (1)–(4) at Ebola Existing Equilibrium (EEE) $F_1 = \left(S^1, E^1, I^1, R^1\right)$ the behavior of each compartment at any time t with delay time $\tau = 0.5$

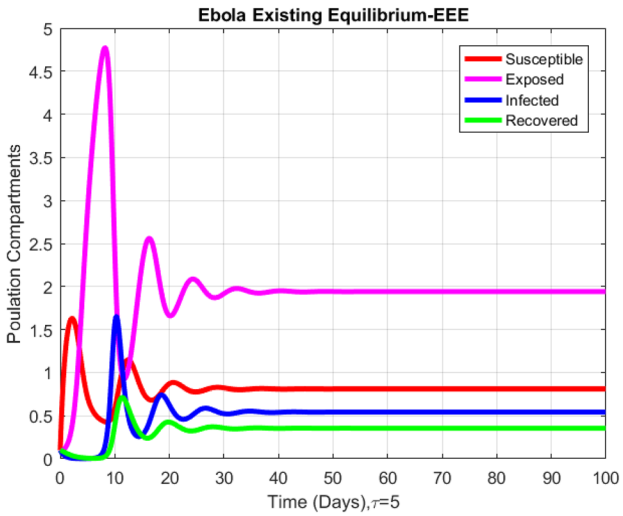

Fig. 3 Graph Shows the Time delay graph of the system (1)–(4) at Ebola Existing Equilibrium (EEE) $F_1 = \left(S^1, E^1, I^1, R^1\right)$ the behavior of each compartment at any time t with delay time $\tau = 5$

$$
\begin{aligned}
\dot{V} =& K_1\left(S-S^1\right)\left[\frac{\lambda}{S}-\frac{\lambda}{S_1}+\left(\beta_1+\beta_4+\beta_6\right)E^1e^{-\mu\tau}\right.\\
&+\left(\beta_5+\beta_7\right)I^1e^{-\mu\tau}\\
&\left.-\left(\beta_1+\beta_4+\beta_6\right)Ee^{-\mu\tau}-\left(\beta_5+\beta_7\right)Ie^{-\mu\tau}\right]\\
&+K_2\left(E-E^1\right)[\left(\beta_1+\beta_4+\beta_6\right)Se^{-\mu\tau}-\beta_2Ie^{-\mu\tau}\\
&-[\left(\beta_1+\beta_4+\beta_6\right)S^1e^{-\mu\tau}-\beta_2I^1e^{-\mu\tau}]\\
&+K_3\left(I-I^1\right)[\beta_2Ee^{-\mu\tau}+\left(\beta_5+\beta_7\right)Se^{-\mu\tau}-\beta_2E^1e^{-\mu\tau}\\
&-\left(\beta_5+\beta_7\right)S^1e^{-\mu\tau}]\\
=& K_1\left(S-S^1\right)\frac{\lambda}{S}-\frac{\lambda}{S_1}+K_1[\left(S-S^1\right)\left[\left(\beta_1+\beta_4\right.\right.\\
&\left.+\beta_6\right)E^1e^{-\mu\tau}+\left(\beta_5+\beta_7\right)I^1e^{-\mu\tau}\\
&\left.-\left(\beta_1+\beta_4+\beta_6\right)Ee^{-\mu\tau}-\left(\beta_5+\beta_7\right)Ie^{-\mu\tau}\right]\\
&+K_2\left(E-E^1\right)[\left(\beta_1+\beta_4+\beta_6\right)Se^{-\mu\tau}-\beta_2Ie^{-\mu\tau}]\\
&-[\left(\beta_1+\beta_4+\beta_6\right)S^1e^{-\mu\tau}-\beta_2I^1e^{-\mu\tau}]\\
&+K_3\left(I-I^1\right)[\beta_2Ee^{-\mu\tau}+\left(\beta_5+\beta_7\right)Se^{-\mu\tau}\\
&-\beta_2E^1e^{-\mu\tau}-\left(\beta_5+\beta_7\right)S^1e^{-\mu\tau}]\\
=& K_1\left(S-S^1\right)\frac{\lambda\left(S^1-S\right)}{SS_1}+K_1\left(S-S^1\right)\left[\{\left(\beta_1+\beta_4\right.\right.\\
&\left.+\beta_6\right)(E^1-E)e^{-\mu\tau}+\left(\beta_5+\beta_7\right)\left(I^1-I\right)\}e^{-\mu\tau}]\\
&+K_2\left(E-E^1\right)\left[\left(\beta_1+\beta_4+\beta_6\right)\left(S-S^1\right)e^{-\mu\tau}+\beta_2Ie^{-\mu\tau}\right]\\
&+K_3\left(I-I^1\right)[\beta_2\left(E-E^1\right)e^{-\mu\tau}\\
&+\left(\beta_5+\beta_7\right)\left(S-S^1\right)e^{-\mu\tau}]\\
=& -\lambda K_1\frac{\left(S-S^1\right)^2}{SS_1}+K_2\left(S-S^1\right)\left(\beta_1+\beta_4\right.\\
&\left.+\beta_6\right)\beta_2\left(E-E^1\right)e^{-\mu\tau}\\
&-K_1\left(S-S^1\right)\left[\left(\beta_1+\beta_4+\beta_6\right)(E-E^1)e^{-\mu\tau}+K_3\left(\beta_5+\beta_7\right)\left(S-S^1\right)\left(I-I^1\right)e^{-\mu\tau}\right.\\
&-K_1\left(\beta_5+\beta_7\right)\left(S-S^1\right)\left(I-I^1\right)e^{-\mu\tau}\\
&+K_3\beta_2\left(E-E^1\right)\left(I-I^1\right)e^{-\mu\tau}-K_2\beta_2\left(E-E^1\right)\left(I-I^1\right)e^{-\mu\tau}\\
=& -\lambda K_1\frac{\left(S-S^1\right)^2}{SS_1}+\left(K_2-K_1\right)\left(S-S^1\right)\left(\beta_1+\beta_4\right.\\
&\left.+\beta_6\right)\beta_2\left(E-E^1\right)e^{-\mu\tau}\\
&+\left(K_3-K_1\right)\left(\beta_5+\beta_7\right)\left(S-S^1\right)\left(I-I^1\right)e^{-\mu\tau}\\
&+\left(K_3-K_2\right)\left(E-E^1\right)\beta_2\left(I-I^1\right)e^{-\mu\tau}.
\end{aligned}
$$

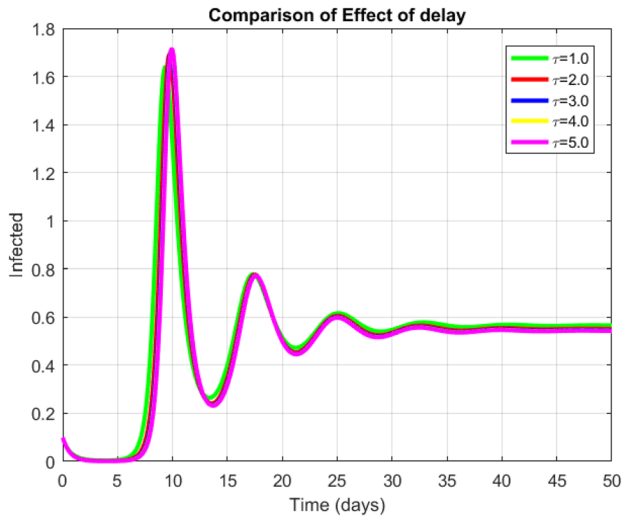

Fig. 4 The graph shows the effect of delay on the infected class of the model at different values of τ

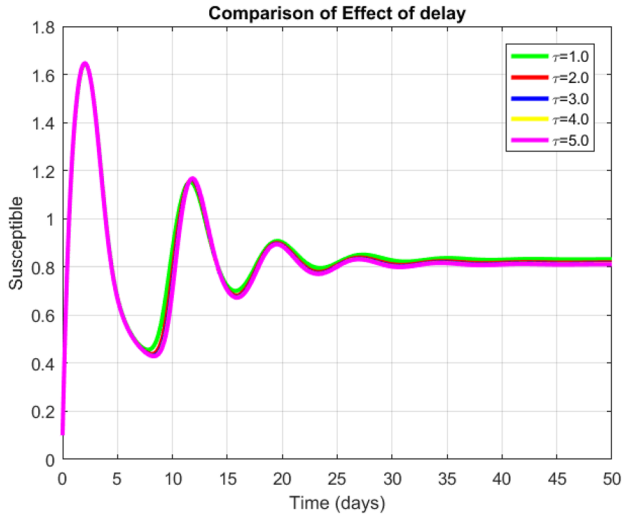

Fig. 5 The graph shows the effect of τ at different values on the susceptible class of the model to varying values of τ

For $K_1 = K_2 = K_3 = 1$, we have

$$\dot{V} = -\lambda K_1 \frac{\left(S - S^1\right)^2}{SS_1} + 0 + 0 + 0 \leq 0.$$

Thus, V is indeed a Lyapunov function. F_1 is globally asymptotically stable.

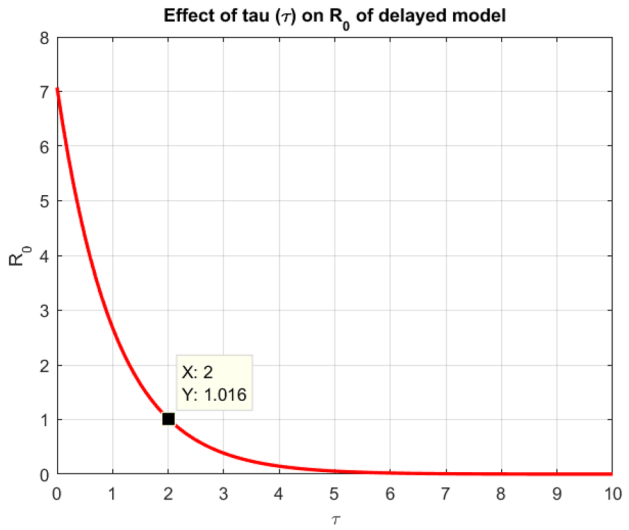

Fig. 6 The graph shows the comparison of delay terms τ and R_0. At different values of τ (days are increasing), R_0 is decreasing. At $\tau = 2$, $R_{0=}1.016$. As time passes, the disease will vanish from the host population

Results

This section uses MATLAB to present parameter values from the table, the numerical simulations, and the Ebola delayed epidemic model analysis.

At the given particular data of the model, the system's nonlinear delay differential equations (DDE's) behaviour is plotted in two states. Figures 2 and 3 exhibit the behaviour of each subpopulation (Susceptible, Exposed, Infected, and Recovered) for Ebola-free equilibrium and Ebola-existing equilibrium at any time t respectively. Also, the effect of delay is observed during the simulations for the different values of delay like 0.1, 0.5, and 5. When we increase the delay then the Ebola-existing equilibrium converges to Ebola-free equilibrium gradually as shown in Fig. 4, 5 and 6. In Fig. 4, we plotted the behaviour of infected subpopulations, so the infectivity reduces for the increase of delay practises meanwhile the susceptibility increases as shown in Fig. 5. Figure 6 shows the comparison behaviour of the reproduction number and the effect of the delay. It means that on which value of delay the disease may switch from the existing state to a disease-free state with the condition of reproduction numbers as claimed by the stability theorems around steady states of the model.

Conclusion

The mathematical modeling of the Ebola virus with delay differential equations has been studied rigorously. The fundamental properties of the delay model verified like positivity and

Table 2 Parameter values used in the underlying model

Notation	Descriptions	Values
λ	Newborn rate to human population	0.06321 (EFE), 2.06321 (EEE)
μ	The natural death rate of susceptible humans	0.9704
β_1	The infection rate of susceptible to exposed humans	0.2877
β_2	Infection rate from exposure to infected humans	0.7613
β_3	Infection rate from infected to recovered humans	0.4389
β_4	The infection rate of wild animals from susceptible to exposed humans	0.1234
β_5	The infection rate of wild animals from susceptible to infected humans	0.2431
β_6	The infection rate of domesticated animals from susceptible to exposed humans	0.4000
β_7	The infection rate of domesticated animals from susceptible to infected humans	0.3000
μ_1	The natural death rate of exposed humans	0.0432
μ_2	The disease-induced death rate of exposed humans	0.2006
μ_3	The natural death rate of infected humans	0.0656
μ_4	The disease-induced death rate of infected humans	0.9764
μ_5	The natural death rate of recovered humans	0.6704
R_0	Reproduction Number	$0.2167 < 1$ (EFE), $7.0735 > 1$ (EEE)

boundedness within in feasible region of the model. The two steady states of the delay model analyzed like Ebola-free equilibrium and Ebola present equilibrium. After that, by using the next-generation method, we have calculated the reproduction number. We have also calculated the sensitivity of the parameters of the reproduction number to analyze which parameter is more sensitive or less sensitive. We have analyzed Local stability at Ebola Free Equilibrium, and the Ebola Existing Equilibrium point is calculated by developing a Jacobian matrix. Using the Lyapunov function, we analyzed global stability at both equilibrium points. We have carefully studied the delayed factor's effects on reproduction in each compartment of our model. Also, the effect of delay has been observed for different values that susceptibility increases and infectivity decreases. Moreover, the graphical illustration gave support to the theoretical analysis of the Ebola delayed model. In the end, delay differential equations in modeling the Ebola virus have proven to be a powerful tool in simulating real-world scenarios, demonstrating that delayed responses in infection and intervention processes must be considered for more accurate and reliable disease management.

Acknowledgements The authors would like to thank the anonymous reviewers for their useful comments and suggestions.

Author contributions Ali Raza: Conceptualization. Nauman Ahamed: writing the original draft. Muhammad Rafiq: software. Ali Akgül: review. Alicia Cordero: analysis. Juan R. Torregrosa: editing final version of the paper.

Funding Open Access funding provided thanks to the CRUE-CSIC agreement with Springer Nature. No funding available.

Data availability All the data used to generate this manuscript appear in the text. No materials have been used.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethics and consent All the authors approve the ethics and consent to participate in this paper. All the authors consent for publication.

References

Abah RT, Zhiri AB, Oshinubi K, Adeniji A (2024) Mathematical analysis and simulation of ebola virus disease spread incorporating

mitigation measures. Franklin Open 6:100066. https://doi.org/10.1016/j.fraope.2023.100066. (**ISSN 2773-1863**)

Abdalla SJM, Chirove F, Govinder KS (2022) A systematic review of mathematical models of the ebola virus disease. Int J Model Simul 42(5):814–830. https://doi.org/10.1080/02286203.2021.1983745

Almuqrin M, Goswami P, Sharma S, Khan I, Dubey R, Khan A (2021) Fractional model of ebola virus in population of bats in frame of Atangana–Baleanu fractional derivative. Results Phys. 26:104295. https://doi.org/10.1016/j.rinp.2021.104295

Banton S, Roth Z, Pavlovic M (2010) Mathematical modeling of ebola virus dynamics as a step towards rational vaccine design. In: Herold KE, Vossoughi J, Bentley WE (eds) 26th southern biomedical engineering conference. Springer, Berlin Heidelberg, pp 196–200

Berge T, Lubuma JMS, Moremedi G, Morris N, Shava RK (2017) A simple mathematical model for Ebola in Africa. J Biol Dyn 11:42–74. https://doi.org/10.1080/17513758.2016.1229817

Chen W (2015) A mathematical model of ebola virus based on sir model. In: 2015 International conference on industrial informatics—computing technology, intelligent technology, industrial information integration, pp. 213–216 . https://doi.org/10.1109/ICIICII.2015.135

Chowell G, Nishimura H (2014) Transmission dynamics and control of Ebola virus disease (evd): a review. BMC Med 12(1):1–17. https://doi.org/10.1186/s12916-014-0196-0

Chretien JP, Riley S, George DB (2015) Mathematical modeling of the West Africa ebola epidemic. eLife 15:01–15. https://doi.org/10.7554/eLife.091864e09186

Deepa O, Nallamalli S, Naik LNS, Teja GVS (2015) Mathematical model for transmission of ebola. Proc Comput Sci 48:741–745. https://doi.org/10.1016/j.procs.2015.04.210

Dokuyucu MA, Dutta H (2020) A fractional order model for ebola virus with the new caputo fractional derivative without singular kernel. Chaos Solitons Fract. 134:109717. https://doi.org/10.1016/j.chaos.2020.109717

Ivorra B, Ngom D, Ramos MA (2015) Be-codes: a mathematical model to predict the risk of human diseases spread between countries-validation and application to the 2014–2015 Ebola virus disease epidemic. Bull Math Biol 77:1668–1704. https://doi.org/10.1007/s11538-015-0100-x

Latha VP, Rihan FA, Rakkiyappan R, Velmurugan G (2018) A fractional-order model for ebola virus infection with delayed immune response on heterogeneous complex networks. J Comput Appl Math 339:134–146. https://doi.org/10.1016/j.cam.2017.11.032

Lolika PO, Helikumi M, Jomah SAS, Bakhet MYA, Galla KC, Kheiralla AH (2024) Global stability analysis of a fractional-order ebola epidemic model with control strategies. J Adv Math Comput Sci 39(2):20–51. https://doi.org/10.9734/jamcs/2024/v39i21866

Nash RK, Bhatia S, Morgenstern C, Doohan P, Jorgensen D, et al. (2024) Ebola virus disease mathematical models and epidemiological parameters: a systematic review and meta-analysis. Preprint at https://doi.org/10.1101/2024.03.20.24304571

Nazir A, Ahmed N, Khan U, Mohyud-Din ST, Nisar K, Khan I (2020) An advanced version of a conformable mathematical model of Ebola virus disease in Africa. Alex Eng J 59(5):3261–3268. https://doi.org/10.1016/j.aej.2020.08.050

Nisar KS, Farman M, Jamil K, Akgul A, Jamil S (2024) Computational and stability analysis of ebola virus epidemic model with piecewise hybrid fractional operator. PLoS ONE 19(4):e0298620. https://doi.org/10.1371/journal.pone.0298620

Osemwinyen AC, Diakhaby A (2015) Mathematical modeling of the transmission dynamics of ebola virus. Appl Comput Math 4(4):313–320. https://doi.org/10.11648/j.acm.20150404.19

Rachah A (2018) Analysis, simulation and optimal control of an seir model for ebola virus with demographic effects. Comm. Fac. Sci. Univ. Ank Stat. 67(1):179–197. https://doi.org/10.48550/arXiv.1705.01079

Rafiq M, Ahmad W, Abbas M, Baleanu D (2020) A reliable and competitive mathematical analysis of ebola epidemic model. Adv. Differ. Equ. 540:01–19. https://doi.org/10.1186/s13662-020-02994-2

Ren H, Xu R (2024) Prevention and control of ebola virus transmission: mathematical modelling and data fitting. J Math Biol 89:25. https://doi.org/10.1007/s00285-024-02122-8

Rhoubari ZE, Besbassi H, Hattaf K, Yousfi N (2018) Mathematical modeling of Ebola virus disease in bat population. Discret Dyn Nat Soc 01–07:2018. https://doi.org/10.1155/2018/5104524

Salem D, Smith RJ (2016) A mathematical model of ebola virus disease: using sensitivity analysis to determine effective intervention targets. In: Proceedings of the summer computer simulation conference, SCSC '16, San Diego, CA, USA. Society for Computer Simulation International

Tahir M, Zaman G, Shah SIA, Muhammad S, Hussain SA, Ishaq M (2019) The stability analysis and control transmission of mathematical model for ebola virus. Open J Math Anal 3(2):91–102. https://doi.org/10.30538/psrp-oma2019.0042

Wester T (2015) Analysis and simulation of a mathematical model of ebola virus dynamics in vivo. SIAM 8:236–256. https://doi.org/10.1137/15s013855

Xu C, Farman M (2023) Dynamical transmission and mathematical analysis of ebola virus using a constant proportional operator with a power law kernel. Fract Fractional. https://doi.org/10.3390/fractalfract7100706