Stability analysis of epidemic models of Ebola hemorrhagic fever with non-linear transmission

Emile Franc Doungmo Goufo*, Morgan Kamga Pene, Stella Mugisha
Department of Mathematical Sciences, University of South Africa, Florida Sciences Campus, 003 South Africa.

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Abstract

Some Epidemic models with fractional derivatives were proved to be well-defined, well-posed and more accurate (Brockmann et al. [D. Brockmann, L. Hufnagel, Phys. Review Lett., 98 (2007), 17–27]; Doungmo Goufo et al. [E. F. Doungmo Goufo, R. Maritz, J. Munganga, Adv. Diff. Equ., 2014 (2014), 9 pages]; Pooseh et al. [S. Pooshe, H. S. Rodrigues, D. F. M. Torres, In: Numerical Analysis and Applied Mathematics, ICNAAM, American Institute of Physics, Melville, (2011), 739–742]), compared to models with the conventional derivative. In this paper, an Ebola epidemic model with non linear transmission is analyzed. The model is expressed with the conventional time derivative with a new parameter included, which happens to be fractional. We proved that the model is well-defined, well-posed. Moreover, conditions for boundedness and dissipativity of the trajectories are established. Exploiting the generalized Routh-Hurwitz Criteria, existence and stability analysis of equilibrium points for Ebola model are performed to show that they are strongly dependent on the non-linear transmission. In particular, conditions for existence and stability of a unique endemic equilibrium to the Ebola system are given. Finally, numerical simulations are provided for particular expressions of the non-linear transmission (with parameters $\kappa = 0.01$, $\kappa = 1$ and $p = 2$). The obtained simulations are in concordance with the usual threshold behavior. The results obtained here are significant for the fight and prevention against Ebola haemorrhagic fever that has so far exterminated hundreds of families and is still infecting many people in West-Africa. ©2016 All rights reserved.

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1. Introduction

Due to the complexity of new outbreaks of diseases happening around the world, the development and
application of new approaches in mathematical epidemiology has exploded recently. Many authors have paid special attention to the modeling of real world phenomena in a broader outlook like for instance, the inclusion of the concept of fractional order derivatives or simply adding new parameters in the process. It happened that some of such modellings are more reliable and provide better predictions compared to models with conventional (integer order) derivative \[11\] \[20\] \[31\] \[40\]. A concrete proof was given in \[40\] with the fact that some epidemic models based on variation with conventional derivative were unable to reproduce the statistical data collected in a real outbreak of some disease with enough degree of accuracy. Other examples are provided in \[30\] \[37\] \[39\] with the application of half-order derivatives and integrals, which, compared to classical models, are proved to be more useful and reliable for the formulation of certain electrochemical problems. For more examples the reader can refer to the works \[4\] \[6\] \[7\] \[10\] \[15\] \[20\] \[31\] \[42\] that have successfully generalized, in various ways, classical derivatives to derivatives of fractional order.

In the domain of mathematical epidemiology, Doungmo Goufo et al. \[20\] provided several interesting and useful properties of Kermack-McKendrick epidemic model with non linear incidence and fractional order derivative. Recall that Kermack-McKendrick epidemic model is considered as the basis from which many other multi-compartmental models were developed. The results obtained therein sustain the legitimation of epidemic models with fractional order derivative and may help analyze more complex models in the field.

Accordingly, The outbreak of Ebola haemorrhagic fever is currently occurring in West African countries and has infected around 28637 people, killed more than 11315 people so far around the world, and these numbers are still rising. Not only the West African region is affected as clearly shown in Fig. \[1\] there is no known and yet confirmed cure for the disease and since the true and real dynamic of the virus is not yet apprehended totally, it is reasonable to applied recent developed concepts to the disease in order to establish a broader outlook on the real nature of this killing disease that has become a nightmare for all the nations. More justifications and motivations are provided in Section \[2.2\] here below.

![Figure 1: Number of Ebola cases and deaths per country](http://apps.who.int/iris/bitstream/10665/147112/1/roadmapsitrep7Jan2016eng.pdf)

2. Some important notes

2.1. Ebola haemorrhagic fever and non-linear transmission

Ebola haemorrhagic fever is caused by Ebolavirus, a virus from the family of filoviridea. The genus Ebolavirus counts itself among three members of the Filoviridae family (filovirus), together with the genus Marburgvirus and the genus Cuevavirus. Three distinct species of the Genus Ebolavirus, namely Bundibugyoebolavirus (BDBV), Zaire ebolavirus (EBOV), Sudan ebolavirus (SUDV) are believed to be largely
responsible for the Ebola outbreaks in Africa in general and the actual 2014 fatal outbreak occurring in West Africa. Ebola virus is an unusual but fatal virus that, when spreading throughout the body, damages the immune system and organs. Ultimately, it causes levels of blood-clotting cells to drop [8]. This causes uncontrollable bleeding inside and outside the body [28] to yield a severe hemorrhagic disease characterized by initial fever and malaise followed by shock, gastrointestinal bleeding symptoms, to end by multi-organ system failure.

In Africa, the transmission of ebola virus is believed to be non-linear and happen in various ways. Most of the infections that occur in living beings are possible by the handling of infected fruit bats, macaques, baboons, vervets, monkeys, chimpanzees, gorillas, forest antelope and porcupines, sometime found dead or sick in the scrubland or forest. Ebola virus is then transmitted from one person to another through human-to-human, human-to-animal or human-to-fruit birelations. The usual infection results from direct contact (through broken skin or mucous membranes) with the blood, secretions, organs or other bodily fluids of infected people. Transmission of Ebola disease also occurs due to indirect contact with environments contaminated with such fluids [22, 23, 32, 44] or during burial ceremonies in which mourners have direct contact with the body of a deceased person.

The literature concerning Ebola’s cure, vaccine, species variety and dynamics is still limited and far from being complete. Therefore, it is urgently necessary to conduct various research and explore new methods and techniques. This will help to better understand the outbreak process and educate people about the real dynamic of Ebola virus, its transmission’s mode and ways to avoid or reduce its spreading. Fig. 2 graphically shows the various and most common modes of transmission used by ebola virus to infect human beings and Fig. 3 shows some basic prevention of the spread of Ebola virus.

2.2. Conventional derivative with new parameter: Justification, motivation

Today, it is widely known that the Newtonian concept of derivative can no longer satisfy all the complexity of the natural occurrences. A couple of complex phenomena and features happening in some areas of sciences or engineering are still (partially) unexplained by the traditional existing methods and remain open problems. Usually in mathematical modeling of a natural phenomenon that changes, the evolution is described by a family of time-parameter operators, that map an initial given state of the system to all subsequent states that takes the system during the evolution. A widely devotion has been predominantly offered to way of looking at that evolution in which time’s change is described as transitions from one state to another. Hence, this is how the theory of semigroups was developed [21, 38], providing the mathematicians with very interesting tools to investigate and analyze resulting mathematical models. However, most of the phenomena that scientists try to analyze and describe mathematically are complex and very hard to handle. Some of them, like depolymerization, rock fractures and fragmentation processes are difficult to analyze [45] and often involve evolution of two intertwined quantities: the number of particles and the distribution of mass among the particles in the ensemble. Then, though linear, they display non-linear features such as phase transition (called “shattering”) causing the appearance of a “dust” of “zero-size” particles with nonzero mass.

Figure 2: Ebola virus transmission modes

Another example is the groundwater flowing within a leaky aquifer. Recall that an aquifer is an underground layer of water-bearing permeable rock or unconsolidated materials (gravel, sand, or silt) from which groundwater can be extracted using a water well. Then, how do we explain accurately the observed movement of water within the leaky aquifer? As an attempt to answer this question, Hantush [24, 25] proposed an equation with the same name and his model has since been used by many hydro-geologists around the world. However, it is necessary to note that the model does not take into account all the non-usual details surrounding the movement of water through a leaky geological formation. Indeed, due to the deformation of some aquifers, the Hantush equation is not able to account for the effect of the changes in the mathematical formulation. Hence, all those non-usual features are beyond the usual models’ resolutions and need other techniques and methods of modeling with more parameters involved.

Furthermore, time’s evolution and changes occurring in some systems do not happen on the same manner after a fixed or constant interval of time and do not follow the same routine as one would expect. For instance, a huge variation can occur in a fraction of second causing a major change that may affect the whole system’s state forever. Indeed, it has turned out recently that many phenomena in different fields, including sciences, engineering and technology can be described very successfully by the models using fractional order differential equations [9, 11, 14, 15, 17, 18, 20, 27, 31, 41]. Hence, differential equations with fractional derivative have become a useful tool for describing nonlinear phenomena that are involved in many branches of chemistry, engineering, biology, ecology and numerous domains of applied sciences. Many mathematical models, including those in acoustic dissipation, mathematical epidemiology, continuous time random walk, biomedical engineering, fractional signal and image processing, control theory, Levy statistics, fractional phase-locked loops, fractional Brownian, porous media, fractional filters motion and nonlocal phenomena have proved to provide a better description of the phenomenon under investigation than models with the conventional integer-order derivative [11, 20, 31, 40].

One of the attempts to enhance mathematical models was to introduce the concept of derivative with fractional order. There exists a very large literature on different definitions of fractional derivatives. The most popular are the Riemann–Liouville and the Caputo derivatives respectively defined as

\[
D_x^\alpha (f(x)) = \frac{1}{\Gamma(n-\alpha)} \left( \frac{d}{dx} \right)^n \int_0^x (x-t)^{n-\alpha-1} f(t) \, dt,
\]

\(n - 1 < \alpha \leq n\) and \(D_x^\alpha (f(x)) = \frac{1}{\Gamma(n-\alpha)} \int_0^x (x-t)^{n-\alpha-1} \left( \frac{d}{dt} \right)^n f(t) \, dt,\)

\(n - 1 < \alpha \leq n.\)
Each of them presents some advantages and disadvantages \cite{17, 39, 42}. Not all of them satisfy the common properties of the standard concept of derivative, and therefore, there are some limitations that will not allow them to adequately describe real-world problems and phenomena. For instance, the Riemann–Liouville derivative of a constant is not zero while Caputo’s derivative of a constant is zero but demands higher conditions of regularity for differentiability.

To compute the fractional derivative of a function in the Caputo sense, we must first calculate its derivative.

Caputo derivatives are defined only for differentiable functions while functions that have no first order derivative might have fractional derivatives of all orders less than one in the Riemann–Liouville sense.

Guy Jumarie (2005 and 2006) proposed a simple alternative definition to the Riemann–Liouville derivative, the modified one shown above.

New fractional derivatives with no singular kernel were recently proposed by many authors including Caputo et al. in \cite{13}, Doungmo Goufo \cite{19}, and a version with non-local and non-singular kernel was introduced by Atangana and Baleanu \cite{5}. However, Caputo fractional derivative \cite{12}, for instance, remains the one mostly used for modelling real-world problems in the field \cite{9, 11, 17, 18, 20}. However, this derivative exhibits some limitations like not obeying the traditional chain rule; which chain rule represents one of the key elements of the match asymptotic method \cite{4, 6, 29, 43}. Recall that the match asymptotic method has never been used to solve any kind of fractional differential equations because of the nature and properties of fractional derivatives. Hence, the conformable derivative was proposed \cite{1, 30}. This derivative is theoretically very easier to handle and obeys the chain rule. But it also exhibits a huge failure that is expressed by the fact that the derivative of any differentiable function at the point zero is zero. This does not make any sense in a physical point of view.

Accordingly, a modified new version, the $\beta$–derivative was proposed in order to skirt the noticed weakness. The main aim of this new derivative was, first of all, to perform a wider analysis on the well-known match asymptotic method \cite{4, 6, 29, 43} and later extend and describe the boundary layers problems within new parameters. Note that the $\beta$–derivative is not considered here as a fractional derivative in the same sense as Riemann–Liouville or Caputo fractional derivative. It is the conventional derivative with a new (fractional) parameter and as such, has been proven to have many applications in applied sciences \cite{4, 6} and mathematical epidemiology \cite{3}. Our goal is to pursue the investigation in the same momentum. It is defined as:

**Definition 2.1.** Let $g$ be a function, such that, $g : [a, \infty) \rightarrow \mathbb{R}$ then, the $\beta$–derivative of $g$ is defined as:

$$A_0^\beta D_t^\beta g(t) = \begin{cases} 
\lim_{\varepsilon \to 0} \frac{g(t+\varepsilon(t+1)^{1-\beta})-g(t)}{\varepsilon} & \text{for all } t \geq 0, \ 0 < \beta \leq 1 \\
g(t) & \text{for all } t \geq 0, \ \beta = 0, 
\end{cases} \quad (2.3)$$

where $\Gamma$ is the gamma-function

$$\Gamma(\zeta) = \int_0^\infty t^{\zeta-1}e^{-t}dt.$$ 

If the above limit exists then $g$ is said to be $\beta$–differentiable.

Note that for $\beta = 1$, we have $A_0^\beta D_t^\beta g(t) = A_0^1 g(t)$. Moreover, unlike other derivatives with fractional parameters, the $\beta$–derivative of a function can be locally defined at a certain point, the same way like the first order derivative. For a general order, let us say $m\beta$, the $m\beta$–derivative of $g$ is defined as

$$A_0^m D_t^{m\beta} g(t) = A_0^\beta D_t^\beta \left(A_0^{(m-1)\beta} g(t)\right) \quad \text{for all } t \geq 0, \ m \in \mathbb{N}, \ 0 < \beta \leq 1 \quad (2.4)$$
Notice that the $m\beta$-derivative of a given function provides information about the previous $n-1$-derivatives of the same function. For instance we have
\[
\mathcal{A}_t D^2_\beta g(t) = \mathcal{A}_t D^\beta \left( \mathcal{A}_t D^\beta g(t) \right) = \left( t + \frac{1}{\Gamma(\beta)} \right)^{1-\beta} \left[ \left( t + \frac{1}{\Gamma(\beta)} \right)^{-\beta} g' + \left( t + \frac{1}{\Gamma(\beta)} \right)^{1-\beta} g'' \right].
\] (2.5)

This gives the $\beta$-derivative a unique property of memory, that is not provided by any other derivative. It is also easy to verify that for $\beta = 1$, we recover the second derivative of $g$. For more properties and details on this new derivative, the readers can consult the reference [4, 6].

**Theorem 2.2** ([4, 6], Theorem 2.1). Assume that a given function $g : [a, \infty) \to \mathbb{R}$ is $\beta-$differentiable at a given point $t_0 \geq a$, $\beta \in (0, 1]$, then, $g$ is also continuous at $t_0$

**Theorem 2.3** ([4, 6], Theorem 2.2). Assume that $f$ is $\beta-$differentiable on an open interval $(a, b)$ then

1. If $\mathcal{A}_t D^\beta f(t) < 0$ for all $t \in (a, b)$ then, $f$ is decreasing on $(a, b)$;
2. If $\mathcal{A}_t D^\beta f(t) > 0$ for all $t \in (a, b)$ then, $f$ is increasing on $(a, b)$;
3. If $\mathcal{A}_t D^\beta f(t) = 0$ for all $t \in (a, b)$ then, $f$ is constant on $(a, b)$.

**Theorem 2.4** ([4, 6], Theorem 2.3). Assume that, $g \neq 0$ and $f$ are two functions $\beta-$differentiable with $\beta \in (0, 1]$ then, the following relations can be satisfied

1. $\mathcal{A}_t D^\beta_\alpha (af(x) + bg(t)) = a \mathcal{A}_t D^\beta_\alpha (f(t)) + b \mathcal{A}_t D^\beta_\alpha (g(t))$ for all $a$ and $b$ real number;
2. $\mathcal{A}_t D^\beta_\alpha (c) = 0$ for $c$ any given constant;
3. $\mathcal{A}_t D^\beta_\alpha (f(t)g(t)) = g(t) \mathcal{A}_t D^\beta_\alpha (f(t)) + f(t) \mathcal{A}_t D^\beta_\alpha (g(t))$;
4. $\mathcal{A}_t D^\beta_\alpha \left( \frac{f(t)}{g(t)} \right) = \frac{g(t) \mathcal{A}_t D^\beta_\alpha (f(t)) - f(t) \mathcal{A}_t D^\beta_\alpha (g(t))}{g^2(t)}$.

**Theorem 2.5** ([4, 6], Theorem 2.4). Let $f : [a, \infty) \to \mathbb{R}$ be a function differentiable and also $\beta-$differentiable and let $g$ be a function defined in the range of $f$, also differentiable, then we have the following rule
\[
\mathcal{A}_t D^\beta_\alpha (gof(x)) = \left( t + \frac{1}{\Gamma(\beta)} \right)^{1-\beta} f'(t)g'(f(t)).
\] (2.6)

3. Model formulation with a new parameter

As mentioned here above, the aim of this article is to propose new approaches, extend classical models to models with the new derivative and investigate them with various and different techniques in order to establish broader outlooks on the real phenomena they describe. So let us consider a region with a constant overall population $N(t)$ at a given time $t$, with $N(0)$ noted $N_0$. The population $N(t)$ is divided into four compartments, namely $S(t)$ the number representing individuals susceptible to catch Ebola, $I(t)$ the number of individuals infected with Ebola, $R(t)$ the number representing people that recover from Ebola and $M(t)$ the number of individuals that are believed to have become immunized after Ebola infection and recovery. We assume that all recruitment, occurring at a constant rate $\Lambda$, is into the class of susceptible to catch the Ebola fever and that every infected person becomes automatically infectious. Some people of the total population are considered to die due to a non-disease related death at a rate constant $\mu$, so that thus $\frac{1}{\mu}$ can be taken as the average lifetime. In addition, Ebola virus kills infectious people at a rate constant $d$. We consider the usual non-linear mass balance incidence expressed as $\kappa Sg(I)$ to indicate successful transmission of Ebola virus due to non-linear contacts dynamics in the populations by infectious. Here, the function $g$ characterizing the nonlinearity is assumed to be at least $C^3(0, N_0]$ with $g(0) = 0$ and $g(I) > 0$ for
0 < I ≤ N_0 and κ is some rate constant. After receiving an effective test treatment or due to personal and yet unknown biological factors, Ebola infectious individuals can spontaneously recover from the disease with a rate constant τ, entering the recovered (immunized) class. Since research about the real dynamics and transmission mode of Ebola virus is still ongoing, we assume that a fraction γR of recovered people γ ≤ 1, after receiving a treatment reduces their risk to get infected again and are believed to be immunized. Thus, a fraction (1 − γ)R of recovered people go back to susceptible class with a rate constant δ. The transfer diagram describing the above dynamics for Ebola fever is given in Fig. 1 and expressed by the system

\[
\begin{align*}
\frac{dA}{dt} &= \Lambda - \kappa S(t)g(I(t)) + (1 - \gamma)\delta R(t) - \mu S(t) \\
\frac{dI}{dt} &= \kappa S(t)g(I(t)) - (\mu + d + \tau)I(t) \\
\frac{dR}{dt} &= \tau I(t) - (\mu + \gamma)R(t) - (1 - \gamma)\delta R(t) \\
\frac{dM}{dt} &= \gamma R(t) - \mu M(t),
\end{align*}
\]

(3.1)

with initial conditions

\[
S(0) = S_0, \quad I(0) = I_0, \quad R(0) = R_0, \quad M(0) = M_0,
\]

(3.2)

where

\[
\frac{\partial^\beta}{\partial t^\beta} (f(t)) = \lim_{\varepsilon \to 0} \frac{f\left(t + \varepsilon \left(t + \frac{1}{\Gamma(\beta)}\right)^{1-\beta}\right) - f(t)}{\varepsilon}
\]

for all t ≥ 0 and 0 < β ≤ 1.

Figure 4: Transfer diagram for the dynamics of Ebola fever transmission in West-Africa

4. Mathematical analysis

In this section, the model (3.1)-(3.2) is analyzed in order to prove its well posedness, study the conditions for the existence of disease free and endemic non-trivial equilibria, provide an expression for the basic reproduction ratio and threshold conditions for asymptotic stability of equilibria.

4.1. Positivity of solutions

**Proposition 4.1.** There exists a unique solution for the initial value problem given (3.1)-(3.2). Furthermore, if the initial condition (3.2) is non-negative then the corresponding solution (S(t), I(t), R(t), M(t)) of the Ebola model (3.1) is non-negative for all t > 0.

**Proof.** The proof of the first part follows from Remark 3.2 supported by Theorem 3.1 in [33]. For the second part, we show the positively invariance of the nonegative orthant \( R^4_+ = \{(S, I, R, M) \in R^4 : S \geq 0, I \geq 0, R \geq 0, M \geq 0\} \). Then, we can investigate the direction of the vector field
Making use of the same arguments as in [20, Property ii] together with Theorem 2.3, we conclude the proof.

4.2. Boundedness and dissipativity of the trajectories

From the above model (3.1), if we add all the equations, we obtain from \( N(t) = S(t) + I(t) + R(t) + M(t) \) and Theorem 2.4 that

\[
D_t^\beta \Lambda = \Lambda - \mu N(t) - dI(t).
\]

Then, this yields \( D_t^\beta N(t) \leq \Lambda - \mu N(t) \). Therefore, making use of the previous section, we have proven the following Proposition.

**Proposition 4.2.**

\[
\lim_{t \to +\infty} N(t) \leq \frac{\Lambda}{\mu}.
\]

Furthermore, we have the following invariance property: If \( N(0) \leq \frac{\Lambda}{\mu} \), then \( N(t) \leq \frac{\Lambda}{\mu} \) for all \( t \geq 0 \).

In particular, the region

\[
\Psi_\varepsilon = \left\{ (S; I; R; M) \in R^4_+; \ N(t) \leq \frac{\Lambda}{\mu} + \varepsilon \right\}
\]

is a compact forward and positively-invariant set for the system (3.1) with non-negative initial conditions in \( R^4_+ \) and that is absorbing for \( \varepsilon > 0 \).

Thus, we will restrict our analysis to this region \( \Psi_\varepsilon \) for \( \varepsilon > 0 \).

4.3. Existence and stability analysis of equilibrium points

We can consider the systems

\[
\begin{align*}
\frac{\partial}{\partial t} D_t^\beta S(t) &= \Lambda - \kappa S(t) g(I(t)) + (1 - \gamma) \delta R(t) - \mu S(t) \\
\frac{\partial}{\partial t} D_t^\beta I(t) &= \kappa S(t) g(I(t)) - (\mu + d + \tau) I(t) \\
\frac{\partial}{\partial t} D_t^\beta R(t) &= \tau I(t) - (\mu + \gamma) R(t) - (1 - \gamma) \delta R(t)
\end{align*}
\]

and

\[
\frac{\partial}{\partial t} D_t^\beta N(t) = \Lambda - \mu N(t) - dI(t).
\]

To obtain the equilibrium points of the system (4.3)-(4.4), let us put...
The solutions of this system are $X^e = (\frac{\Delta}{\mu}, 0, 0)$ and $X^c = (S^e, I^e, R^e, N^e)$, where

$$S^e = \frac{(\mu + d + \tau) I^e}{\kappa g(I^e)}$$

$$R^e = \frac{\tau I^e}{\mu + \gamma + (1 - \gamma) \delta}$$

$$N^e = \frac{\Delta - d I^e}{\mu}$$

and $I^e$ satisfying the equation:

$$\frac{g(I)}{I} \left[ 1 - \left( \frac{(\mu + d + \tau)(\mu + \gamma + (1 - \gamma)\delta) - (1 - \gamma)\delta \tau}{\Lambda(\mu + \gamma + (1 - \gamma)\delta)} \right) I \right] = \mu(\mu + d + \tau) $$

(4.6)

4.3.1. Existence and stability of the disease-free equilibrium (DFE)

$X^o$ is the DFE and to analyze its stability for the system (4.3)-(4.4), we study the eigenvalues of the Jacobian matrix evaluated at that equilibrium point. Thus, evaluated at $X^o$, the Jacobian obtained from the linearized system (4.3), (4.4) is given by:

$$J(X^o) = Df(X^o) = \begin{pmatrix}
-\mu & -\kappa \Delta g'(0) & (1 - \gamma)\delta & 0 \\
0 & \kappa \Delta g'(0) - (\mu + d + \tau) & 0 & 0 \\
0 & \tau & -(\mu + \gamma) - (1 - \gamma)\delta & 0 \\
0 & -d & 0 & -\mu
\end{pmatrix}$$

(4.7)

**Theorem 4.3.** Taking into Consideration the non linear incidence function $g$. defined above, the disease free equilibrium of the Ebola disease system (4.3)-(4.4) always exists and is asymptotically stable if

$$\frac{\kappa \Delta g'(0)}{\mu(\mu + d + \tau)} < 1.$$  

**Proof.** The existence of $X^o$ is obvious. Following the same approach as [20, 35] we know that asymptotical stability the DFE (equilibrium point) $X^o$ for the model (4.3)-(4.4) is guaranted if and only if all the four eigenvalues, say $\lambda_{1,2,3,4}$ of $J(X^o)$ lie outside the closed angular sector

$$\alpha \frac{\pi}{2} \geq |\text{arg}\lambda_i|, \quad \text{for } i = 1, 2, 3, 4.$$  

Hence, it is enough to show that

$$\alpha \frac{\pi}{2} < |\text{arg}\lambda_i|$$

(4.8)

for all $i = 1, 2, 3, 4$. Making use of the characteristic matrix

$$\Delta_J(\lambda) = \begin{pmatrix}
\mu + \lambda & \kappa \Delta g'(0) & -(1 - \gamma)\delta & 0 \\
0 & -\kappa \Delta g'(0) + (\mu + d + \tau) + \lambda & 0 & 0 \\
0 & -\tau & \mu + \gamma + (1 - \gamma)\delta + \lambda & 0 \\
0 & d & 0 & \mu + \lambda
\end{pmatrix}$$

(4.9)

and the characteristic equation $(\mu + \lambda)^2(\mu + \gamma - (1 + \gamma)\delta + \lambda)(-\kappa \Delta g'(0) + (\mu + d + \tau) + \lambda) = 0$, we obtain the eigenvalues
\[ \lambda_{1,2} = -\mu \]
\[ \lambda_3 = -(\mu + \gamma - (1 - \gamma)\delta) \]
\[ \lambda_4 = \kappa \frac{\Lambda}{g'}(0) - (\mu + d + \tau). \]

\( \lambda_4 \) satisfies the constraint (4.3) if \( \frac{\kappa \Lambda g'(0)}{\mu} < \mu + d + \tau \) and since \( \lambda_{1,2,3} \) obviously satisfy the constraint, the proof is complete. \( \square \)

For the Ebola model (4.3)-(4.4), we usually refer the quantity
\[ R_0 = \frac{\kappa \Lambda g'(0)}{\mu (\mu + d + \tau)} \]  
(4.10)
to as the basic reproduction number and is defined to be the number of secondary Ebola cases that one case will produce in a completely Ebola disease susceptible population. In the biological points of view, Theorem 4.3 insinuates that Ebola epidemic disease will die out if \( R_0 < 1 \).

### 4.3.2. Existence and stability of the endemic equilibrium

As in [4, 6, 34], we can put (4.6) in the form
\[ \frac{1}{\vartheta} = \frac{\mu (\mu + d + \tau)}{\Lambda \kappa} = \frac{g(I)}{I} \left( 1 - \frac{I}{\Theta} \right) = h(I), \]  
(4.11)
where \( \Theta = \frac{\Lambda (\mu + \gamma + (1 - \gamma)\delta)}{(\mu + d + \tau)(\mu + \gamma + (1 - \gamma)\delta) - (1 - \gamma)\delta \vartheta} \). Then, the number of solutions in terms of \( I \) of equation (4.10) is dependent on the non linear incidence function \( g(I) \), especially, \( \lim_{I \to 0} \frac{g(I)}{I} \equiv h(0) \) and the sign of \( h'(I) \). Moreover, \( \Theta \) is the maximum possible value that can take \( I^c \) and in the classical mass action incidence, where \( g(I) = I \), the quantity \( \vartheta = \frac{\Lambda \kappa}{\mu (\mu + d + \tau)} \) is viewed as the contact reproduction number. As shown in [20, 26], if we denote by \( \vartheta^* \) the unique value of \( \vartheta \) verifying (4.11) when \( I \) reaches a unique maximum value \( I_m \) in \((0, \Theta)\), then conditions of existence of the endemic equilibrium \( X^e \) are given in the following theorem:

**Theorem 4.4.** The Ebola model (4.3)-(4.4)

1. has no endemic equilibrium point if \( h(0) \leq \frac{1}{\vartheta} \) and \( h'(I) < 0 \) for all \( I \in (0, \Theta) \)
2. has no endemic equilibrium point if \( h(0) = 0 \), \( h''(I) < 0 \) on \((0, \Theta)\) and \( \vartheta < \vartheta^* \)
3. has 1 endemic equilibrium point if \( h(0) > \frac{1}{\vartheta} \) and \( h'(I) < 0 \) for all \( I \in (0, \Theta) \)
4. has 1 endemic equilibrium point if \( h(0) = 0 \), \( h''(I) < 0 \) on \((0, \Theta)\) and \( \vartheta > \vartheta^* \),
   where \( I_1^e \in (0, I_m) \) and \( I_2^e \in (I_m, \Theta) \).

Considering the expression of \( R_0 \) given in (4.10), knowing that \( g'(0) \sim \lim_{I \to 0} \frac{g(I) - g(0)}{I - 0} \equiv h(0) \) and that \( h(I) \) is positive for \( I \in (0, \Theta) \), with \( h(\Theta) = 0 \), then, item 3 of Theorem 4.4 together with (4.11) yield the following lemma.

**Corollary 4.5.** The Ebola model (4.3)-(4.4) has a unique endemic equilibrium if \( R_0 > 1 \) and \( h'(I) < 0 \) for \( I \in (0, \Theta) \).

Next, conditions for the stability of \( X^e \) is studied from the linearized system of (4.3)-(4.4) around the endemic equilibrium \( X^e = (S^e, I^e, R^e, N^e) \). The following Jacobian matrix is obtained:

\[
J(X^e) = \begin{pmatrix}
-\kappa g(I^e) - \mu & -\kappa S^e g'(I^e) & (1 - \gamma)\delta & 0 \\
\kappa g(I^e) & \kappa S^e g'(I^e) - (\mu + d + \tau) & 0 & 0 \\
0 & \tau & -(\mu + \gamma) - (1 - \gamma)\delta & 0 \\
0 & -d & 0 & -\mu \\
\end{pmatrix}. \]  
(4.12)
To analyse the eigenvalues $\lambda_i$, $i = 1, 2, 3, 4$, we develop the characteristic equation

$$
\begin{vmatrix}
\kappa g(I^e) + \mu + \lambda & \kappa S^e g'(I^e) & -(1 - \gamma)\delta & 0 \\
-\kappa g(I^e) & -\kappa S^e g'(I^e) + (\mu + d + \tau) & 0 & 0 \\
0 & -\tau & (\mu + \gamma) + (1 - \gamma)\delta + \lambda & 0 \\
0 & d & 0 & \mu + \lambda \\
\end{vmatrix} = 0,
$$

(4.13)

which yields

$$(\mu + \lambda)(\lambda^3 + K_1\lambda^2 + K_2\lambda + K_3) = 0,$$

(4.14)

where

$$
K_1 = \kappa g(I^e) + 2\mu + (1 - \gamma)\delta + (\mu + d + \tau) \left(1 - \frac{I^e g'(I^e)}{g(I^e)}\right)
$$

$$
K_2 = \kappa(\mu + d + \tau)g'(I^e)I^e + (\mu + \gamma + (1 - \gamma)\delta)(\kappa g(I^e) + 2\mu) + (\kappa g(I^e) + 2\mu + \gamma + (1 - \gamma)\delta)(\mu + d + \tau) \left(1 - \frac{I^e g'(I^e)}{g(I^e)}\right)
$$

$$
K_3 = \kappa(\mu + d + \tau)(\mu + \gamma + (1 - \gamma)\delta)g'(I^e)I^e - \kappa g(I^e)\gamma(1 - \gamma)\delta + (\kappa g(I^e) + \mu)(\mu + \gamma + (1 - \gamma)\delta)(\mu + d + \tau) \left(1 - \frac{I^e g'(I^e)}{g(I^e)}\right).
$$

(4.15)

We see that the coefficients $K_1$, $K_2$, and $K_3$ are dependent on the nonlinear incidence $g(I)$, hence, since $\lambda = -\mu$ is already an eigenvalue which is non-positive, the stability of the endemic equilibrium $X^e$ is fully determined by analyzing the roots of

$$
P(\lambda) = \lambda^3 + K_1\lambda^2 + K_2\lambda + K_3 = 0
$$

given in (4.14). Let us denote by $\Delta_P$ the discriminant of the polynomial $P(\lambda)$ then, making use of the Routh-Hurwitz Criteria generalized in [2], we state the following Corollary:

**Corollary 4.6.** The positive endemic equilibrium $X^e$ of the Ebola model (4.3)-(4.4) is asymptotically stable if one of the following conditions is satisfied:

1. $K_1 \geq 0$, $K_2 \geq 0$, $K_3 > 0$, $\Delta_P < 0$, and $0 < \beta \leq \frac{2}{3}$.
2. $K_1 < 0$, $K_2 < 0$, $\Delta_P < 0$, and $\frac{2}{3} < \beta \leq 1$.
3. $K_1 > 0$, $K_3 > 0$, $K_1K_2 > K_3$, and $\Delta_P > 0$.

5. Numerical simulations

Let us consider the nonlinear incidence function $g(I) = \frac{Ip}{r + qI}$, $p, q > 0$, $r > 0$. We restrict ourselves to the case $r = 0$, to have $g(I) = Ip$. We use the implementation code of the predictor-corrector PECE method of Adams-Bashforth-Moulton type described in [16] to perform numerical simulations for the Ebola model (4.3)-(4.4). We will consider different values for $\beta$ in order to appreciate the accuracy of the method employed in this article. The table below presents the description and estimated values of the evolved parameters.

The approximation for solutions $S(t), I(t), R(t)$ and $N(t)$ are presented in Figs. [5-6], respectively. In each case two different values of $\beta$, namely $\beta = 0.93$ and 1 are considered. It appears that numerical results show that the Ebola model (4.3)-(4.4), using the new $\beta$-derivative, exhibits the traditional threshold behaviour.

In Fig. [5] we have considered for the non-linear incidence, the transmission coefficient $k = 0.01$ and $p = 2$. Then trajectory of the Ebola model (4.3)-(4.4) converges to the disease-free equilibrium, which is
<table>
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<th>Parameters' symbols</th>
<th>Description</th>
<th>Estimation and range$^b$</th>
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<tr>
<td>$\Lambda$</td>
<td>Recruitment rate by susceptible people in the region</td>
<td>55 (day)$^{-1}$</td>
</tr>
<tr>
<td>$\kappa$</td>
<td>Transmission coefficient</td>
<td>Not constant</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>Proportion of recovered individuals that become immunized</td>
<td>0.04</td>
</tr>
<tr>
<td>$\delta$</td>
<td>Rate at which recovered people go back to susceptible class</td>
<td>0.06</td>
</tr>
<tr>
<td>$\mu$</td>
<td>Non-Ebola-disease related death rate</td>
<td>0.01</td>
</tr>
<tr>
<td>$d$</td>
<td>Ebola related death rate</td>
<td>0.7</td>
</tr>
<tr>
<td>$\tau$</td>
<td>Recovery rate from Ebola</td>
<td>0.1</td>
</tr>
<tr>
<td>$p$</td>
<td>Symbolizing the non-linear incidence</td>
<td>2</td>
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</tbody>
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http://apps.who.int/iris/bitstream/10665/147112/1/roadmapsitrep7Jan2016eng.pdf

...approximatelly at (5500, 0, 0, 5500) with the above given parameters. We also note that the behavior of the system remains similar for close values of the derivative parameter $\beta$.

In Fig. 6 we have taken the transmission coefficient $\kappa = 0.01$ and $p = 2$. Making use of the involved parameter in the table above, the dynamics shows that there exists one positive endemic equilibrium point, approximately at (11.11, 7.29, 6.78, 4989.70) satisfying the condition 3 of Theorem 4.4. Again a similar behavior of the model appears for close values of $\beta$.

![Figure 5: The dynamics of Ebola model](image-url)
6. Conclusion

We have intensively analyzed an Ebola epidemic model with non-linear transmission and have shown that this model, which is itself relatively new in the literature, is well-defined, well-posed. In addition to provide conditions for boundedness and dissipativity of the trajectories for the Ebola model, we also studied existence and stability of equilibrium points to show that they are dependent on the non-linear incidence included in the established expression of the basic reproduction $R_0$. One of the main results here is reflected by conditions for existence and stability of a unique endemic equilibrium point for the Ebola model. Numerical simulations performed for some particular expressions of the non-linear transmission, with coefficients $\kappa = 0.01$, $\kappa = 1$ and power $p = 2$, agree with the obtained results and satisfy the traditional threshold behavior. The work performed in this paper is pertinent since it generalized the preceding ones with the inclusion of a general expression of the incidence together with a new derivative that extends the conventional one. This is useful and might happen to be capital in the ongoing fight and future prevention again the Ebola virus that has recently shaken the whole world and killed dozens of people in West-Africa.

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References

