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# An advanced version of a conformable mathematical model of Ebola virus disease in Africa



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**Abstract** An SIR-type (Susceptible-Infected-Recovered) model for the study of the spread of Ebola Virus Disease (EVD) is developed, by using conformable derivatives. Every possible way of transmission of the disease is incorporated (direct or indirect), such as funeral practices, consumption of contaminated bush meat and the environmental contamination etc. We have added an extremely important term to the model which have very high physical significance i.e., the possibility of the birth of an infected individual and the migration of an infected individual to the existing population. Well-posedness of the proposed problem has been shown by using a well-known theorem. The situations for the disease to be died out or sustain, have been discussed in the details. We found that the only disease-free situation is, the absence of flow of Ebola virus disease from the environment. We also have observed that by adopting a few strategies, such as isolation of infected individuals and careful burial of deceased bodies, the spread of EVD can be controlled. Memory effects for each case (disease-free and endemic states) are discovered (by using Khalil's conformable transform) and plotted to make future predictions more accurately. Graphs are clearly elaborating that the problem is stable for both the equilibria states i.e., endemic state and disease-free state.

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## 1. Introduction

The more deadly outbreak of Ebola Virus Disease (EVD) since 1976, took place in West Africa in 2014, that destroyed a large proportion of the population. The statistics of clinical cases and death cases was about more than 16,000 people and 70% of the population respectively. In almost all the cases,

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the main source to initiate the disease was “animal”, in such a way that when a man hunted for food, his contact took place with infected animals (such as monkey, ape, chimpanzees and fruit bats etc.). The observation as mentioned above enabled us to state that indirect contact could be one of the reasons for the spread of the disease [1]. Poor hygiene and sanitary conditions are also one of the reasons for the spread of the Ebola virus in Africa. It was observed in 2015 [2], when a non-negligible amount of Ebola Virus was found active for up to 50 days on different surfaces like glass and plastic etc. In Africa (particularly the region effected by EVD outbreaks), the majority of the population harvest forest fruits for food, hunts bats and monkeys and live close to the rain-forests [3,4]. Their traditional values are very high that even in the presence of a contagious disease, they do not avoid to shake hand and kissing. Moreover, in their death ceremonies, they wash and properly dressed up their deceased without the fear of germ/disease transfer. They also share the dresses of their deceased relatives. Huge gatherings on funerals from all close villages are also the cause of the quick spread of Ebola Virus.

On studying the paragraph as mentioned earlier, we reach the following points;

Persistence and recurrence of EVD in Africa is due to

- Consumption of contaminated bush meat
- The funeral ceremonies
- Environmental pollutant

in the presence of both transmissions

- Human-human by body fluid like blood, sweat, saliva, vomit, breast milk and urine etc (direct transmission)
- Environment-human-environment by objects like contaminated clothes etc (indirect transmission)

By keeping all the points mentioned above, about African practices, we propose a Conformable SIRDP model where P is denoting the compartment of the environment.

In the existing literature, we found models related to the spread of EVD in the human population through only direct contact in [5–14]. We can find classical types of models in almost all studies such as SI [15], SIR [8,10], SEIR [5,7], SEIRD [11] and SEIRHD [6,13].

In this century, fractional calculus emerged as one of the most important parts of many research fields like neural networks, biomathematics, physics etc. [16–39]. In [40], Kumar et al., presented his studies of fractional calculus on modified kahwara equation, with a non-singular kernel. Caputo et al., resolved many difficulties regarding singular kernel by proposing a non-singular operator with exponential kernel function [41]. Furthermore, the properties for the exponential kernel were defined and presented by Losada et al., in [42]. In [43], Atangana et al., used Mittag Leffler function and gave a new definition for fractional-order derivative. To see different types of operators for fractional derivatives and their importance for real-life occurring, one can search [44–48].

Jumarie presented few basic derivative formulae for fractional calculus in [49]. He proposed Modified R-L fractional derivative in [50]. After that, few conflicts were raised regarding Jumarie formulae in [51–53]. So, in response to those conflicts, Khalil et al. in [54], provided a new definition of fractional derivatives that is users friendly. So, We have opted

the above definition, to study the current model related to the spread of the Ebola Virus.

Throughout the literature review, we did not find the factor  $a_2$ . But it has a lot of importance in the regard that it can affect the total number of population and obviously the change in the statistics affect the final results. We have dig out the hidden phenomena that there might be a possibility that a fetal can catch the infection from his mother in the uterus and it can directly be added to the infected population, by birth. Similarly, there is a chance of migration of infected person/persons to the particular population. So, it is also a source of addition to the population of infected individuals. In addition, we are the first to propose a conformable model for Ebola Virus Disease that provides memory effects which have a lot of importance in understanding any physical phenomena. We have also proposed a few control strategies for the spread of the Ebola virus.

The organization of the rest of the article is as follows. The development of the model is given in Section 2. Well-posedness of the problem is proved in Section 3 along with the findings of equilibria points and basic reproduction number. Discussion on the main findings are given in Sections 4, and Section 5 contains concluding remarks.

## 2. Formulation of the model

To investigate the spread, persistence and recurrence of Ebola Virus Disease (EVD) outbreaks in Africa, few following assumptions could be made;

- One of the causes of the spread of EVD infection is deceased human individuals. As the deceased bodies can transmit the infection during their burial ceremonies.
- The infection can be added to the environment through urine and stool of infected/ deceased individuals.
- Infection can be cached not only through direct contact but also by indirect contacts, such as through contaminated environment and surfaces.
- Provision of EVD in the environment due to the consumption of contaminated bush meat.
- Existence of permanent disease-induced immunity.
- EVD outbreaks lasted for two or more years in Africa, so during this long time-period, the new addition in the population in the form of new births and migration, as well as the deaths (natural or due to disease) takes place. So, we can call it a demographic process.

A mathematical model can be developed based on the above, mentioned assumptions.

$$\frac{dF(t)}{dt} = a_1 - (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - \sigma F(t), \quad (1)$$

$$\begin{aligned} \frac{dL(t)}{dt} = & a_2 + (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) \\ & - (\sigma + \eta + \gamma)L(t), \end{aligned} \quad (2)$$

$$\frac{dQ(t)}{dt} = \gamma L(t) - \sigma Q(t), \quad (3)$$

$$\frac{dW(t)}{dt} = (\sigma + \eta)L(t) - bW(t), \quad (4)$$

$$\frac{dG(t)}{dt} = \mu + \xi L(t) + \alpha W(t) - \delta G(t). \tag{5}$$

In the model, Eqs. (1)–(5),  $F, L, Q, W, G$  are the variables used to represent the susceptible population, infected individuals, recovered humans, deceased population and environment respectively. The population of susceptible individuals is increasing at a constant rate  $a_1$ , by migrant people and new births. The contact rate of a susceptible individual with an infectious one ( $\beta_1$ ), with a deceased individual ( $\beta_2$ ) and with the contaminated environment ( $\lambda$ ) might acquire infection.  $\sigma$  is the natural death rate whereas the infectious human have an additional death rate due to the disease ( $\eta$ ). They get to recover at rate  $\gamma$ . Birth of an infected neonatal or migration of an infected person may increase the infected population, shown by a constant rate  $a_2$ . Rate of deceased human burial is  $b$ .  $\mu$  is the constant rate at which the EVD is contaminating the environment by all means such as wildlife, fruit bats etc. Moreover,  $\xi$  and  $\alpha$  are the rates at which the infectious and deceased individuals shed the environment respectively.

The details, about the above-used parameters (for time  $t$ ), are in the following in Table 1.

Khalil et al. in [54], proposed a new definition of fractional derivatives as following;

**Definition.** Let

$$g : (0, \infty) \rightarrow \mathbb{R},$$

then the conformable derivative of  $g$  (with order  $\zeta$ ) is defined as following,

$$B_\zeta(g)(t) = \lim_{\epsilon \rightarrow 0} \frac{g(t + \epsilon t^{1-\zeta}) - g(t)}{\epsilon}, \quad \forall t > 0, \quad \zeta \in (0, 1]. \tag{6}$$

The above definition, also satisfy few properties (mentioned in [54]). One of those properties is given below,

If  $g$  is differentiable then,

$$B_\zeta(g)(t) = t^{1-\zeta} \frac{dg}{dt}. \tag{7}$$

Let's re-develop the model (1)-(5), by using the above-mentioned Khalilzadeh's conformable derivative [54], as follows

$$B_\zeta(F)(t) = a_1 - (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - \sigma F(t), \tag{8}$$

$$B_\zeta(L)(t) = a_2 + (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - (\sigma + \eta + \gamma)L(t), \tag{9}$$

$$B_\zeta(Q)(t) = \gamma L(t) - \sigma Q(t), \tag{10}$$

$$B_\zeta(W)(t) = (\sigma + \eta)L(t) - bW(t), \tag{11}$$

$$B_\zeta(G)(t) = \mu + \xi L(t) + \alpha W(t) - \delta G(t). \tag{12}$$

In the above model, Eqs. (8)-(12),  $B_\zeta$  is the operator, symbolizing the conformable derivative of the function, with  $\zeta$  ( $\zeta \in (0, 1]$ ) as the order of the derivative.

Now, using Eq. (7), Eqs. (8)-(12) can be transformed as follows,

$$t^{1-\zeta}(F)'(t) = a_1 - (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - \sigma F(t), \tag{13}$$

$$t^{1-\zeta}(L)'(t) = a_2 + (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - (\sigma + \eta + \gamma)L(t), \tag{14}$$

$$t^{1-\zeta}(Q)'(t) = \gamma L(t) - \sigma Q(t), \tag{15}$$

$$t^{1-\zeta}(W)'(t) = (\sigma + \eta)L(t) - bW(t), \tag{16}$$

$$t^{1-\zeta}(G)'(t) = \mu + \xi L(t) + \alpha W(t) - \delta G(t). \tag{17}$$

On simplifying the above, we get the final form of the system as follows;

$$(F)'(t) = t^{\zeta-1}(a_1 - (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - \sigma F(t)), \tag{18}$$

$$(L)'(t) = t^{\zeta-1}(a_2 + (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - (\sigma + \eta + \gamma)L(t)), \tag{19}$$

$$(Q)'(t) = t^{\zeta-1}(\gamma L(t) - \sigma Q(t)), \tag{20}$$

$$(W)'(t) = t^{\zeta-1}((\sigma + \eta)L(t) - bW(t)), \tag{21}$$

$$(G)'(t) = t^{\zeta-1}(\mu + \xi L(t) + \alpha W(t) - \delta G(t)). \tag{22}$$

Initial conditions attached to the above system (18)-(22), are

$$F(0) = F_0,$$

$$L(0) = L_0,$$

$$Q(0) = Q_0,$$

$$W(0) = W_0,$$

**Table 1** Details and description of variables and parameters..

Parameters	Values	Description
$F$	–	Susceptible (human)
$L$	–	Infectious (human)
$Q$	–	Infected deceased (human)
$W$	–	Recovered (human)
$G$	–	Ebola virus pathogens in the environment
$a_1$	Variable [assumed]	The rate of recruitment of susceptible (human)
$a_2$	Variable [assumed]	The rate of recruitment of infected (human)
$b$	(0,1) [14]	Burial rate of deceased (human)
$\beta_1$	Variable [7,13,14]	Rate of contact (effective) of infectious human
$\beta_2$	Variable [8,14]	Rate of contact (effective) of deceased (human)
$\lambda$	Variable [assumed]	Rate of contact (effective) of Ebola virus
$\sigma$	(0,1) [11]	Rate of natural deaths of human
$\eta$	[0.4-0.9] [5,7,8]	Rate of deaths of human individuals due to infection
$\mu$	(0,1) [assumed]	Rate of recruitment of EVD in the environment
$\xi$	(0,∞) [assumed]	Rate of shedding of infectious human
$\alpha$	(0,∞) [assumed]	Rate of shedding of deceased human

$$G(0) = G_0.$$

The conservation law is obtained by adding the first three equations of the above system (18)-(22),

$$\frac{dZ(t)}{dt} = t^{1-\zeta}(a_1 + a_2 - \sigma Z - \eta L), \tag{23}$$

where,  $Z = F + L + Q$ , is the sum of total alive/active population. Moreover, the burial rate should always less than or equal to the total death rate to cope up with real-world phenomena.

For the structural view of the model see Fig. 1.

### 3. Well-posedness and equilibria

Firstly, we will prove the well-posedness of the model and then equilibria points will be found in this section.

#### 3.1. Well-posedness

We will prove it in three steps below.

**Proposition 3.1.1.** *Suppose that model (18)-(22), has a global solution corresponding to non-negative initial conditions,*

then the solution is non-negative for all time.

**Proof.** Let's assume that

$$0 \leq F(0), 0 \leq L(0), 0 \leq Q(0), 0 \leq W(0), 0 \leq G(0).$$

We can write the first equation of the model (18)-(22), as follows;

$$\frac{dF(t)}{dt} = t^{\zeta-1}(a_1 - C(t)F(t)), \tag{24}$$

where,

$$C(t) = \beta_1 L(t) + \beta_2 W(t) + \lambda G(t) - \sigma.$$

Eq. (24) is a first-order linear equation in F. So, its solution has the following form;

$$F(t) = F(0)e^{\int_0^t -s^{\zeta-1}C(s)ds} + e^{\int_0^t -s^{\zeta-1}C(s)ds} \left( \int_0^t a_1 u^{\zeta-1} \left( e^{\int_0^u w^{\zeta-1}A(w)dw} \right) du \right) \geq 0.$$

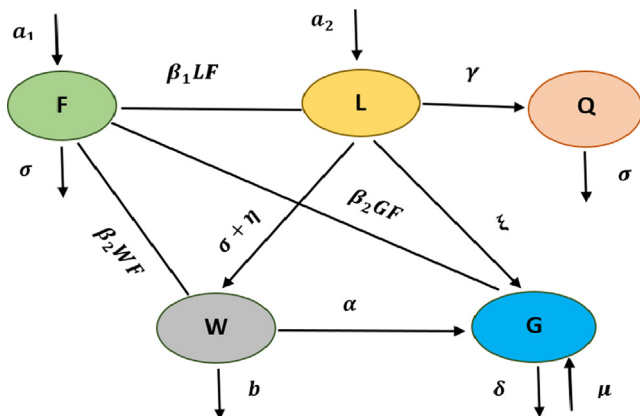


Fig. 1 Flow diagram.

Which implies  $F(t) \geq 0, \forall t \geq 0$ .

As the remaining variables are non-negative so, we can write the subsystem as follows

$$\frac{dL(t)}{dt} = t^{\zeta-1}(a_2 + (\beta_1 L(t) + \beta_2 W(t) + \lambda G(t))F(t) - (\sigma + \eta + \gamma)L(t)), \tag{25}$$

$$\frac{dQ(t)}{dt} = t^{\zeta-1}(\gamma L(t) - \sigma Q(t)), \tag{26}$$

$$\frac{dW(t)}{dt} = t^{\zeta-1}((\sigma + \eta)L(t) - bW(t)), \tag{27}$$

$$\frac{dG(t)}{dt} = t^{\zeta-1}(\mu + \zeta L(t) + \alpha W(t) - \delta G(t)). \tag{28}$$

Which can be written in the matrix form as follows;

$$\frac{dE(t)}{dt} = t^{\zeta-1} \mathcal{M} Y(t) + H(t), \tag{29}$$

where,

$$\mathcal{M} = \begin{bmatrix} \beta_1 F(t) - (\sigma + \eta + \gamma) & 0 & \beta_2 F(t) & \lambda F(t) \\ \gamma & -\sigma & 0 & 0 \\ \sigma + \eta & 0 & -b & 0 \\ \zeta & 0 & \alpha & -\delta \end{bmatrix},$$

$$Y(t) = \begin{bmatrix} L(t) \\ Q(t) \\ W(t) \\ G(t) \end{bmatrix}, \quad H(t) = \begin{bmatrix} a_2 \\ 0 \\ 0 \\ \mu \end{bmatrix},$$

The above matrix  $\mathcal{M}$ , is a Metzler matrix (a matrix with non-negative off-diagonal entries) where non-negativity of F has already been established. So, Eq. (29) is a monotone system. Thus,  $\mathcal{R}_+^4$  is invariant under the flow of system (29). Hence the proposition is proved.

To guarantee the boundedness of the solution of system (18)-(22), the following proposition can be stated along with its proof.

**Proposition 3.1.2.** *Suppose that the initial conditions for the model (18)-(22), satisfy the following*

$$Z(0) \leq Z_m, W(0) \leq W_m, G(0) \leq G_m,$$

where,

$$Z_m = \frac{a_1 + a_2}{\sigma}, W_m = \frac{(\sigma + \eta)(a_1 + a_2)}{b\sigma},$$

$$G_m = \frac{\sigma b \mu + b \zeta (a_1 + a_2) + \alpha (\sigma + \eta)(a_1 + a_2)}{b \delta \sigma}.$$

Furthermore, whenever the solution exists on an interval  $I_1$ , it satisfies the following bounds;

$$Z(t) \leq Z_m, W(t) \leq W_m, G(t) \leq G_m.$$

**Proof.** Since,  $L(t) \geq 0$ , we can write Eq. (23) as follows

$$\frac{dZ(t)}{dt} \leq t^{1-\zeta}(a_1 + a_2 - \sigma Z) \tag{30}$$

Gronwall's inequality yields following

$$\frac{dZ(t)}{dt} \leq \frac{a_1 + a_2}{\sigma} + \left( Z(0) - \frac{a_1 + a_2}{\sigma} \right) e^{-\frac{t\sigma}{\zeta}}. \tag{31}$$

Implies

$$Z(t) \leq Z_m, \text{ if } Z(0) \leq Z_m.$$

Consequently,  $L(t) \leq Z_m$ . Using it in Eq. (21) implies the following

$$\frac{dW(t)}{dt} \leq t^{\zeta-1}((\sigma + \eta)Z_m - bW(t)),$$

Now, Gronwall's inequality yields the following

$$W(t) \leq W_m, \text{ whenever } W(0) \leq W_m.$$

Following the same process for  $G(t)$  we get,

$$G(t) \leq G_m, \text{ whenever } G(0) \leq G_m.$$

Hence, boundedness is proved.

To ensure the well-posedness of the problem, let's combine the propositions 3.1.1 and 3.1.2 together (with the trivial existence and uniqueness of a solution for the system (18)-(22)), and state the following theorem.

**Theorem 3.1.3.** *Model (18)-(22), is a dynamical system on the following compact set*

$$\mathcal{H} = \left\{ (F(t), L(t), Q(t), W(t), G(t)) \in \mathbb{R}_+^4; Z(t) \leq \frac{a_1 + a_2}{\sigma}, \right.$$

$$W(t) \leq \frac{(\sigma + \eta)(a_1 + a_2)}{b\sigma},$$

$$G(t) \leq \frac{\sigma b\mu + b\zeta(a_1 + a_2) + \alpha(\sigma + \eta)(a_1 + a_2)}{b\delta\sigma}$$

### 3.2. Equilibria

This subsection is dedicated to the investigation of the existence of equilibria points of the model (18)-(22). The induction of the parameter  $\mu$  as a constant flow of EVD from the environment has a significant part. So, we can state that there would be only chance of disease-free equilibrium point in the absence of  $\mu$ , i.e.,

$$\psi_0 = (F, 0, 0, 0, 0) = \left( \frac{a_1}{\sigma}, 0, 0, 0, 0 \right). \tag{32}$$

And there will be no disease-free condition if  $\mu$  is positive. In this case, we can find the endemic equilibrium point as follows.

Let us take  $\mathfrak{E} = (\mathcal{F}, \mathcal{L}, \mathcal{Q}, \mathcal{W}, \mathcal{G})$ , be an equilibrium point i.e.

$$a_1 - (\beta_1 \mathcal{L} + \beta_2 \mathcal{W} + \lambda \mathcal{G}) \mathcal{F} - \sigma \mathcal{F} = 0, \tag{33}$$

$$a_2 + (\beta_1 \mathcal{L} + \beta_2 \mathcal{W} + \lambda \mathcal{G}) \mathcal{F} - (\sigma + \eta + \gamma) \mathcal{L} = 0, \tag{34}$$

$$\gamma \mathcal{L} - \sigma \mathcal{Q} = 0, \tag{35}$$

$$(\sigma + \eta) \mathcal{L} - b \mathcal{W} = 0, \tag{36}$$

$$\mu + \zeta \mathcal{L} + \alpha \mathcal{W} - \delta \mathcal{G} = 0. \tag{37}$$

On solving the above system (33)-(37), we get the following endemic equilibria points,

$$\mathcal{F} = \frac{(a_1 + a_2) - (\sigma + \eta + \gamma) \mathcal{L}}{\sigma}, \tag{38}$$

$$\mathcal{Q} = \frac{\gamma \mathcal{L}}{\sigma}, \tag{39}$$

$$\mathcal{W} = \frac{(\sigma + \eta) \mathcal{L}}{b}, \tag{40}$$

$$\mathcal{G} = \frac{b\mu + (b\zeta + \alpha\eta + \alpha\sigma) \mathcal{L}}{b\delta}. \tag{41}$$

### 3.3. Basic reproduction number

To obtain basic reproduction number  $\mathbb{R}_0$ , we follow the working steps of [55], and get the following;

$$\mathbb{X} = \begin{bmatrix} -\frac{\beta_1 a_1}{\sigma} & 0 & -\frac{\beta_2 a_1}{\sigma} & -\frac{\lambda a_1}{\sigma} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \tag{42}$$

and

$$\mathbb{Y} = \begin{bmatrix} \sigma + \eta + \gamma & 0 & 0 & 0 \\ -\gamma & \sigma & 0 & 0 \\ -\sigma - \eta & 0 & b & 0 \\ -\zeta & 0 & -\alpha & \delta \end{bmatrix}, \tag{43}$$

where  $\mathbb{X}$  and  $\mathbb{Y}$  are transmissions and transition matrices respectively. Now, the matrix  $\mathbb{X}\mathbb{Y}^{-1}$ , can be found as follows;

$$\mathbb{X}\mathbb{Y}^{-1} = \begin{bmatrix} \frac{\beta_1 a_1}{(\sigma + \eta + \gamma)\sigma} + \frac{\beta_2 a_1(\sigma + \eta)}{(\sigma + \eta + \gamma)\sigma b} + \frac{\lambda a_1(\alpha\eta + \alpha\sigma + b\zeta)}{(\sigma + \eta + \gamma)\sigma b \delta} & 0 & \frac{\beta_2 a_1}{\sigma b} + \frac{\lambda a_1 \alpha}{\sigma b \delta} & \frac{\lambda a_1}{\sigma \delta} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{44}$$

Finally, we have basic reproduction number as follows;

$$\mathbb{R}_0 = \frac{\beta_1 a_1}{(\sigma + \eta + \gamma)\sigma} + \frac{\beta_2 a_1(\sigma + \eta)}{(\sigma + \eta + \gamma)\sigma b} + \frac{\lambda a_1(\alpha\eta + \alpha\sigma + b\zeta)}{(\sigma + \eta + \gamma)\sigma b \delta}. \tag{45}$$

## 4. Results and discussion on the solution of the model

The system of differential equations (18)-(22), has been solved numerically to get approximate solutions. The solution is painted with the help of graphs that are presented below. These figures are drawn to show the memory effects for different values of  $\zeta$ . Indeed, the use of fractional order discovers the hidden phenomena because of the memory effects, that can't be seen in the mathematical models with  $\zeta = 1$ . The beauty of the fractional order is that the solution of the fractional model (18)-(22), tends to the solution of the classical model (1)-(5) [1], as the value of  $\zeta$  tends to 1.

Figs. 2-6 are drawn to show the results for disease-free equilibrium points when  $\mu = 0$ . Other values of parameters are  $a_1 = 10, a_2 = 3, \sigma = 0.5, \eta = 0.05, \gamma = 0.06, b = 0.8, \zeta = 0.04, \alpha = 0.04, \delta = 0.03, \lambda = 0.01, \beta_1 = 0.006; \beta_2 = 0.012$ .

Figs. 7-11 are presenting the results for endemic equilibrium points in the absence of a contaminated environment i.e.,  $\mu = 0$  and with values of others parameters as  $a_1 = 10,$



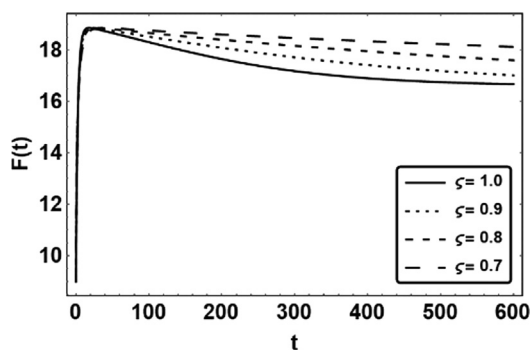


Fig. 2 Plotting of different values of  $\zeta$ , (for susceptible individuals).

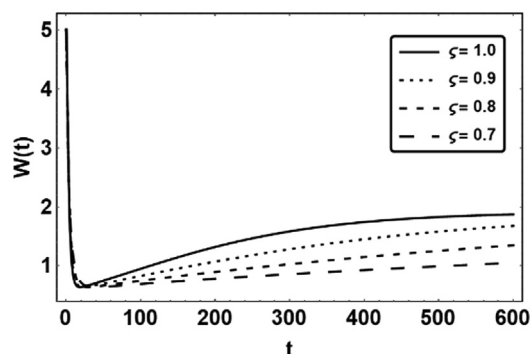


Fig. 5 Plotting of different values of  $\zeta$ , (for deceased individuals).

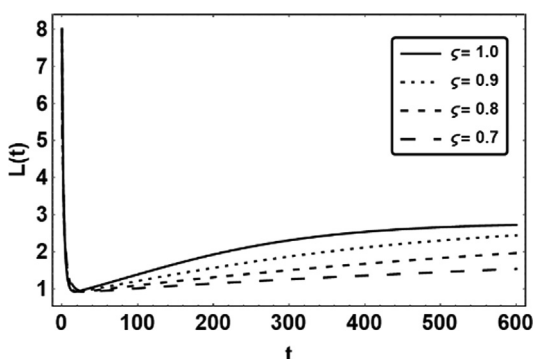


Fig. 3 Plotting of different values of  $\zeta$ , (for infected individuals).

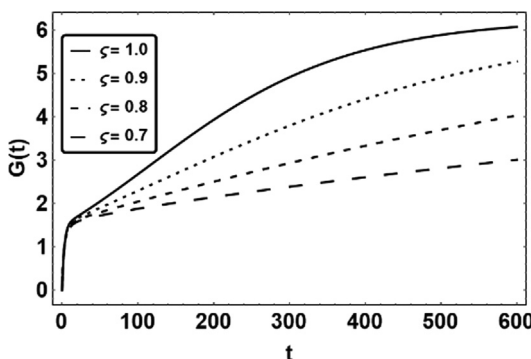


Fig. 6 Plotting of different values of  $\zeta$ , (for virus pathogens in the environment).

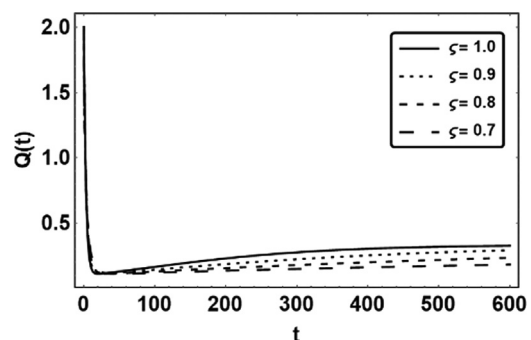


Fig. 4 Plotting of different values of  $\zeta$ , (for recovered individuals).

$a_2 = 3, \sigma = 0.02, \eta = 0.9, \gamma = 0.06, b = 0.8, \xi = 0.04, \alpha = 0.04, \delta = 0.03, \lambda = 0.01, \beta_1 = 0.006, \beta_2 = 0.012.$

We can observe in Fig. 12, that the recruitment of EVD in the environment has a significant impact on the contamination of the environment.

There are few control strategies which could be adopted to minimize the virus and its effects on a community, like;

- Isolation of infected individuals (i.e.  $\beta_1 = 0$ ).
- Careful burial of deceased bodies (i.e.  $\beta_2 = 0$ )

The effect of above-mentioned strategies (on all compartments) has been shown in the following Figs. 13-17.

### 5. Conclusion

A conformable mathematical model for the spread of Ebola Virus Disease has been proposed and presented. A system of conformable differential equations is developed to govern the problem and a well-known theorem has been used to ensure the well-posedness of it. We have concluded that to minimize the spread of EVD, isolation of infected individuals and careful burial of deceased bodies should be adopted. In addition, the absence of flow of EVD from environment can play a major part to make the population a disease-free one. Graphs have been plotted, to show the behaviors of the solutions of all subclasses, for fractional values of derivatives between 0 and 1. These variation memories for the solutions have not been revealed yet. So, the use of conformable derivatives is more important in order to understand any physical phenomena in depth.

### Declaration of Competing Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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