

Mathematical analysis and simulation of Ebola virus disease spread incorporating mitigation measures

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ABSTRACT

In order to understand the dynamics of the Ebola virus disease (EVD), this research developed a mathematical model that includes quarantine and public education campaigns as control measures. The model's equilibrium points are displayed and the effective basic reproduction number R_{eff} is estimated. Bifurcation theory is used for the stability analysis of endemic equilibrium state and general bifurcation theory is used to prove the existence of endemic equilibrium state. We evaluated the nature of the endemic equilibrium state of the model's equation near the disease-free equilibrium, $R_{eff} = 1$ and introduce a bifurcation parameter. The results of the centre manifold theory are used to demonstrate that there is nontrivial endemic equilibrium near the disease-free equilibrium. We demonstrated the characteristics of the endemic equilibrium state that is close to the disease-free equilibrium as well as the fact that there is a potentially unstable endemic equilibrium condition. In other words, the sickness is slowly declining and will eventually disappear, as in the case of West Africa. Finally, we simulated the model developed to study the dynamics of the diseases with varying parameters using Homotopy Perturbation Method (HPM) to validate the qualitative analysis of the model. The result confirmed the hypothesis of our research that if quarantine and public enlightenment is properly used for the mitigation of the disease, the disease outcomes will drastically reduced. The results presented in this research will be useful for public health experts to contain the Ebola disease spread most especially in Africa.

1. Introduction

Named after a river in the old Zaire (now known as the Democratic Republic of the Congo) where it was originally discovered in 1997, the Ebola virus disease (EVD) is an acute viral hemorrhagic fever that is extremely contagious (CDC, 2004). It belongs to the Filovirus family of RNA (ribonucleic acid) viruses. The Ebola virus disease spreads through personal contact with bodily fluids, tissues, or semen from infected individuals who are either dead or alive, [1] and WHO, [2]. People who have contracted the virus becomes contagious after an incubation time of 21 days [3], EVD has flu-like symptoms at first, but quickly develops into internal and external bleeding, vomiting, rash and diarrhoea. After entering the body, the virus starts attacking the liver and blood cells, which are immune system cells that ordinarily guard the body against infection. As the fever worsens, the virus attacks the liver and kidneys, two key organs, which causes major bleeding, tissue damage, shocks, respiratory arrest, and eventually death. Most affected people pass away within 10 days after contracting the disease [4], at about 50%–90% mortality WHO, [5]. In 2003, twelve Ebola

outbreaks were recorded in Sudan, Gabon, Congo and Uganda [6], as a result of two different strains of the Ebola virus (Ebola Zaire and the Ebola Sudan) that were reported in those areas. The largest outbreaks were reported in these countries, including Nigeria, Guinea, Sierra Leone, Liberia, Senegal, Mali, Spain, the United States, and the United Kingdom. There was a total of 26,724 instances of infections and 11065 fatalities throughout this time period. Mali, Senegal, Nigeria, Spain, the United Kingdom, and the United States are the six of these nations that have been deemed clear of the Ebola virus disease but have previously reported a case or cases that were imported from a nation with widespread and active transmission. [7].

The Uganda Ministry of Health in September 2022, WHO and AFRO, confirmed the breakout of EVD in Mubende District, Uganda, the strain known as confirmed was Sudan virus disease (SVD) and a fatal case. It was a case of a 24-year-old man, residing in Ngabano village, a sub-county of Madudu in the District of Mubende which was noticed that he had experienced the following symptoms like high fever, abdominal pain, diarrhoea, and vomiting of blood. In total, 142 cases were

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confirmed, 87 recovered, and 55 died (CFR: 39%). Additionally, other probable cases were 22 deaths reported which were those who died before their samples were taken (overall CFR: 47%). Also, among some healthcare workers, 19 were infected and 7 died. Up to 21 days, about 4000 contacts had been followed up, (WHO, AFRO, News). 9 Ugandan districts had been affected by the outbreak namely, Mubende, Kagadi, Kyegegwa, Masaka, Jinja, Kampala, Wakiso, Bunyangabu, and Kasanda. The Ministry of Health of Equatorial Guinea has now identified eight additional Marburg cases, increasing the total number of cases that have been identified since the viral hemorrhagic fever outbreak was notified in February 2023 to nine.

Kermack et al. [8] contributed a great deal to modelling infectious disease categories and transmission. They named their model the SIR model and this model has been extended by many researchers and applied to several infectious diseases.

The dynamics of Ebola fever disease transmission have drawn a lot of interest from researchers [9–11]. Astacio et al. [12] used the *SIR* and *SEIR* models, which simulated two Ebola outbreaks; the ones that occurred in Yambuku, Zaire, in 1976, and Kikwit, Zaire, in 1995. The per-capita mortality rate of infected people and the per-capita effective contact rate of a person catching the disease were used to estimate the dynamics of these models. The disease’s contagiousness is determined by the fundamental reproductive number R_0 . The authors claimed that Ebola is not as contagious as was previously believed, and that the outcomes of their simulations will provide researchers with knowledge that will help them reduce the number of fatalities that could follow from future epidemics. Abdulrahman et al. (2014) developed a model on the stability analysis of Disease-Free Equilibrium State for the transmission dynamics and control of Ebola and as well as Monkeypox optimal control strategies [13,14]. They obtained the basic reproduction number and analysed the disease-free equilibrium state for stability. Their finding reveal that once Ebola Fever disease is introduced into a population, the disease morbidity and mortality continue to rise, until high surveillance is put in place to quarantine and treat the infected individuals and proper burial for those that died due to the disease.

It is important to note that aside Ebola, there are other infectious disease such as Monkeypox [15,16], COVID-19 [17–19], and Tuberculosis [20,21]. However EVD have been considered in relation to other form of impact in terms of stigmatization; such as Juga et al. [22]. A mathematical model was created to explore the impact of stigmatization on the dynamics of the disease as the work’s unique examination. The approach takes into account both external stigmatization (forced on survivors by their communities) and internal stigmatization (experienced by infected individuals who witness survivors being stigmatized). The results imply that both types of stigma can raise burden by inducing concealment of infection among those who already have it and encouraging improper funeral practises for those who have already passed away. The authors contend that measures to stop stigmatization and encourage proper funerals could greatly lessen the burden of EVD. Based on the above, we develop a mathematical model that takes into consideration public campaign and awareness to reduce stigmatization which will also help in proper funeral and burial arrangements. We presented some mathematical theoretical analysis and also utilize numerical approaches to explore this process. The main reason for using Homotopy Perturbation Method (HPM) for numerical simulation is that, in contrast to other methods, it allows for greater flexibility in the selection of basis functions for the solution and does not require linear inversion operators. It also maintains a level of simplicity that makes the method easily understandable from the perspective of broader perturbation methods which shows the novelty of our work to tack the spread of Ebola virus.

2. Model formulation

The dynamics of EVD using mathematical modelling and using quarantine and public awareness as controls was developed. Susceptible $S(t)$, Latent $L(t)$, Infectious $I(t)$, Quarantined $Q(t)$, Recovered $R(t)$, and Dead $D(t)$ are the six compartments that make up the population. A total population is given in (2.1)

$$N(t) = S(t) + L(t) + I(t) + Q(t) + R(t) + D(t) \tag{2.1}$$

Individuals who have not had direct contact with the virus typically make up disease models for the Ebola virus and the individual are known to Susceptible $S(t)$. When susceptible people come into contact with infectious people, they become infected but do not immediately become contagious, therefore they enter the Latent $L(t)$ class. Once the latency period is over, these people are contagious and the infected move to the Infectious $I(t)$ class. To mitigate the EVD spread, the infected individuals are separated into Quarantine $Q(t)$ class for treatment. In the event of the treatment period, some persons in the Quarantine class recover completely and move into the Recovered $R(t)$ class. Some persons as a result of the EVD dies and move from $I(t)$ and $Q(t)$ into the Dead $D(t)$ class; however, due to unsafe burial procedures, this class exists. Individuals known as $S(t)$ are those who have not actually been exposed to the Ebola virus sickness but are nonetheless at risk of contracting it through contact with the $I(t)$ and $D(t)$ at the rate α_1 and $\alpha_2(1 - \varsigma)$, where α_1 and $\alpha_2(1 - \varsigma)$ the actual contact rate. It is inevitable that natural death would occur and this happens at rate of μ while natural birth rate occurs with β from the compartment of $S(t)$, $L(t)$ and $R(t)$. There are individuals who are within the time frame of incubation (i.e not shown symptoms) but after the 21 there is possibility of showing traits which means there high chances of showing weak immunity strength to fight back the virus, hence, they are moved to the $I(t)$ class at rate γ .

The schematic diagram is represented in Fig. 1

The model representation is given in (2.2)–(2.7) and the assumptions follows below:

In developing the model, the following assumptions holds:

1. Everyone has an identical probability of contracting an infection if they come into touch with enough contagious persons because of the homogeneous mixing of people.
2. People in $S(t)$ get infected by coming into contact with $I(t)$ and $D(t)$.
3. Since $L(t)$ only become infectious when they exhibit symptoms, they are infected but not yet contagious (i.e symptomatic).
4. The isolation of $I(t)$ to $Q(t)$ cause the spread of Ebola Fever to be very low due to treatment rate τ .
5. $\delta_2 < \delta_1$ because $Q(t)$ was managed at the rate τ .
6. Natural birth do not exist $I(t)$ and $Q(t)$ classes.
7. If persons in $Q(t)$ recover, they recover permanently due to the treatment rate τ .
8. The $D(t)$ class exist as a result of people who are infected and died or died while at the quarantine.

We define variables and parameters in Table 1:

The flow chart is described Eqs. (2.2)–(2.7):

$$\frac{dS}{dt} = \beta(S + L + R) - \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1 - \varsigma) D}{N} \right) (1 - \xi)S - \mu S \tag{2.2}$$

$$\frac{dL}{dt} = \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1 - \varsigma) D}{N} \right) (1 - \xi)S - (\gamma + \mu)L \tag{2.3}$$

$$\frac{dI}{dt} = \gamma L - (\varphi + \mu + \delta_1)I \tag{2.4}$$

$$\frac{dQ}{dt} = \varphi I - (\tau + \mu + \delta_2)Q \tag{2.5}$$

$$\frac{dR}{dt} = \tau Q - \mu R \tag{2.6}$$

$$\frac{dD}{dt} = (\mu + \delta_1)I + (\mu + \delta_2)Q - \varsigma D \tag{2.7}$$

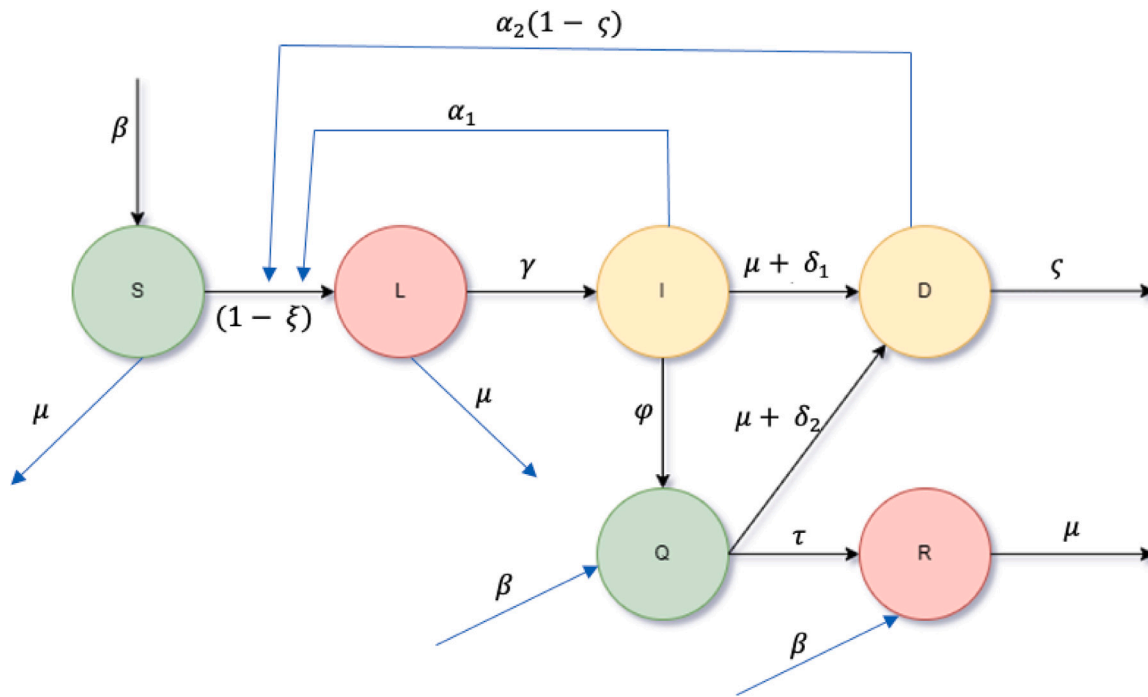


Fig. 1. Flow chart of EVD transmission and Control model.

Table 1
Definition of parameters.

Parameters	Description
β	Rate of birth
μ	Rate of death
δ_1	Disease induced death rate of $I(t)$
δ_2	Disease induced death rate of $Q(t)$
α_1	Effective contact rate between $I(t)$ and $S(t)$
$\alpha_2(1 - \xi)$	Effective contact rate between $D(t)$ and $S(t)$
γ	Progression rate from $L(t)$ to $I(t)$
φ	Rate of quarantine
τ	Treatment rate
ξ	The rate to which public campaigns effective
ζ	Rate of decontamination and burial of the deceased
$(1 - \xi)$	The percentage of those who disregarded public awareness campaigns and are nonetheless susceptible to EVD.

Where in Eq. (2.1), N is represented as

$$N(t) = S(t) + L(t) + I(t) + Q(t) + R(t)$$

Total population changes at rate $\frac{dN(t)}{dt}$, and given by Eq. (2.8)

$$\frac{dN(t)}{dt} = \beta N - \mu(S + L + R) \tag{2.8}$$

The model in Eq. (2.1) to Eq. (2.8) is epidemiological and mathematically well posed in the domain Ω with the initial conditions.

As seen in Eq. (2.9);

$$\Omega = (S, L, I, Q, R, D) \in R^6 \tag{2.9}$$

$$S \geq 0, L \geq 0, I \geq 0, Q \geq 0, R \geq 0, D \geq 0$$

$$S + L + I + Q + R \leq N$$

3. Properties of the model

Analysing model Eq. (2.1) to Eq. (2.8), we consider theorems:

Theorem 3.1. *The solutions of the model Eqs. (2.1) to (2.8) is positive \forall time $t \geq 0$, given the initial conditions are positive.*

Proof.

Using the assumptions, the initial conditions are positive, i.e., $S(0) > 0, L(0) > 0, I(0) > 0, Q(0) > 0, R(0) > 0,$ and $D(0) > 0$

We have by contradiction, the solutions of Eq. (2.1) to Eq. (2.8) are positive if it is supposed that a contradiction exists for the first time, $t_1 : S(t_1) = 0$ and

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.1}$$

for $0 < t < t_1$ or there exists $t_2 : L(t_2) = 0$, such that

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.2}$$

for $0 < t < t_2$ or there exists $t_3 : I(t_3) = 0$, such that

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.3}$$

for $0 < t < t_3$, or there exists $t_4 : Q(t_4) = 0$

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.4}$$

for $0 < t < t_4$ or there exists $t_5 : R(t_5) = 0$

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.5}$$

for $0 < t < t_5$ or there exists $t_6 : D(t_6) = 0$

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.6}$$

for $0 < t < t_6$.

Now, in the case where $S(t) = 0$, we obtain Eq. (3.7)

$$\frac{dS(t_1)}{dt} = \lim_{t \rightarrow t_1} \frac{S(t_1) - S(t)}{t_1 - t} < 0 \tag{3.7}$$

and similarly, it resulted to Eq. (3.8)

$$\frac{dL(t_2)}{dt} < 0, \quad \frac{dI(t_3)}{dt} < 0, \quad \frac{dQ(t_4)}{dt} < 0, \quad \frac{dR(t_5)}{dt} < 0, \quad \frac{dD(t_6)}{dt} < 0 \tag{3.8}$$

However, from Eqs. (2.1) to (2.6) gives,

$$\frac{dS(t_1)}{dt} = \beta(S(t_1) + L + R) - \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\zeta)D}{N} \right) (1-\xi)S(t_1) - \mu S(t_1) \tag{3.9}$$

i.e.

$$S^i(t_1) = \beta(L + R) > 0 \tag{3.10}$$

Which contradicts Eq. (3.7). Therefore, $S(t_1) \neq 0$, and S is positive for all t .

Similarly, for the remaining variables gives

$$L^i(t_2) = \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\zeta)D}{N} \right) (1-\xi)S > 0 \tag{3.11}$$

$$I^i(t_3) = \gamma L > 0 \tag{3.12}$$

$$Q^i(t_4) = \varphi I > 0 \tag{3.13}$$

$$R^i(t_5) = \tau Q > 0 \tag{3.14}$$

$$D^i = (\mu + \delta_1)I + (\mu + \delta_2)Q > 0 \tag{3.15}$$

These are contradictions with what was predicted for each of the variables, which means that $L(t_2) \neq 0$, $I(t_3) \neq 0$, $Q(t_4) \neq 0$, $R(t_5) \neq 0$, and $D(t_6) \neq 0$, hence, S, L, I, Q, R and D is non-negative for all t . From this, it evidently shows that all the solutions of Eq. (2.1) to Eq. (2.6) are in R^6 , provided that the initial conditions are positive. The feasible region is positively-invariant, hence, it is sufficient to ponder on the dynamics of Eq. (2.1) to Eq. (2.6) in the region Ω .

3.1. Existence of equilibrium states E

Let $E(S(t), L(t), I(t), R(t), Q(t), D(t))$, be the equilibrium states of the system. At equilibrium state, Each variable's rate of change is equal to zero, i.e.,

$$\frac{dS}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dQ}{dt} = \frac{dR}{dt} = \frac{dD}{dt} = 0 \tag{3.16}$$

Consider Eq. (3.17) to Eq. (3.18)

$$(S(t), L(t), I(t), Q(t), R(t), D(t)) = (r, v, w, x, y, z) \tag{3.17}$$

$$N(t) = n, \tag{3.18}$$

where $n = r + v + w + x + y$, hence, Equations ((2.1) to (2.8)) become

$$\beta(r + v + x) - \left(\frac{\alpha_1 w}{n} + \frac{\alpha_2(1-\zeta)z}{n} \right) (1-\xi)r - \mu r = 0 \tag{3.19}$$

$$\left(\frac{\alpha_1 w}{n} + \frac{\alpha_2(1-\zeta)z}{n} \right) (1-\xi)r - (\gamma + \mu)v = 0 \tag{3.20}$$

$$\gamma v - (\varphi + \mu + \delta_1)w = 0 \tag{3.21}$$

$$\varphi w - (\tau + \mu + \delta_2)x = 0 \tag{3.22}$$

$$\tau x - \mu y = 0 \tag{3.23}$$

$$(\mu + \delta_1)w + (\mu + \delta_2)x - \zeta z = 0 \tag{3.24}$$

$$\beta n - \mu(r + v + y) = 0 \tag{3.25}$$

Equations ((3.19) to (3.25)) must then be solved to get the equilibrium states.

3.2. Disease-free equilibrium E_0

At E_0 , depicts absence of infection, where the infected classes are zero. The population at this stage comprises of the susceptible class.

From Eq. (3.24) gives

$$z = \frac{(\mu + \delta_1)w + (\mu + \delta_2)x}{\zeta} \tag{3.26}$$

From Eq. (3.20) gives

$$v = \frac{(1-\xi)r}{(\gamma + \mu)n} (\alpha_1 w + \alpha_2(1-\zeta)z) \tag{3.27}$$

Substituting Eq. (3.26) in Eq. (3.27), we obtain Eq. (3.28)

$$v = \frac{(1-\xi)r}{\zeta(\gamma + \mu)n} [\alpha_1 \zeta w + \alpha_2(1-\zeta)(\mu + \delta_1)w + \alpha_2(1-\zeta)(\mu + \delta_2)x] \tag{3.28}$$

From Eq. (3.22), it resulted into Eq. (3.29)

$$x = \frac{\varphi}{(\tau + \mu + \delta_2)} w \tag{3.29}$$

Substituting Eq. (3.29) in Eq. (3.28), we obtain

$$v = \frac{(1-\xi)r}{\zeta(\gamma + \mu)n} (\alpha_1 \zeta w + \alpha_2(1-\zeta)(\mu + \delta_1)w + \alpha_2(1-\zeta)(\mu + \delta_2) \frac{\varphi}{(\tau + \mu + \delta_2)} w) \tag{3.30}$$

Substituting Eq. (3.30) in Eq. (3.21), we obtain Eq. (3.31)

$$\frac{(1-\xi)r}{\zeta(\gamma + \mu)n} \left(\alpha_1 \zeta + \alpha_2(1-\zeta)(\mu + \delta_1) + \frac{\varphi \alpha_2(1-\zeta)(\mu + \delta_2)}{(\tau + \mu + \delta_2)} \right) - (\varphi + \mu + \delta_1)w = 0 \tag{3.31}$$

as

$$w = 0$$

or

$$\frac{(1-\xi)r}{\zeta(\gamma + \mu)n} (\alpha_1 \zeta + \alpha_2(1-\zeta)(\mu + \delta_1) + \frac{\varphi \alpha_2(1-\zeta)(\mu + \delta_2)}{(\tau + \mu + \delta_2)}) - (\varphi + \mu + \delta_1) = 0 \tag{3.32}$$

From (3.32) gives

$$\frac{(1-\xi)r}{\zeta(\gamma + \mu)n} (\alpha_1 \zeta + \alpha_2(1-\zeta)(\mu + \delta_1) + \frac{\varphi \alpha_2(1-\zeta)(\mu + \delta_2)}{(\tau + \mu + \delta_2)}) = (\varphi + \mu + \delta_1) \tag{3.33}$$

Multiplying both sides by $\frac{\zeta(\gamma + \mu)n}{(1-\xi)}$ gives Eq. (3.34)

$$\frac{(\alpha_1 \zeta + \alpha_2(1-\zeta)(\mu + \delta_1) + \frac{\varphi \alpha_2(1-\zeta)(\mu + \delta_2)}{(\tau + \mu + \delta_2)})}{(\tau + \mu + \delta_2)} = \frac{\zeta(\gamma + \mu)(\varphi + \mu + \delta_1)n}{(1-\xi)} \tag{3.34}$$

From Eq. (3.34), we obtain Eq. (3.35)

$$r = \frac{\zeta(\tau + \mu + \delta_2)(\varphi + \mu + \delta_1)(\gamma + \mu)n}{\alpha_1 \zeta + \alpha_2(1-\zeta)(\mu + \delta_1) + \frac{\varphi \alpha_2(1-\zeta)(\mu + \delta_2)}{(\tau + \mu + \delta_2)}} \tag{3.35}$$

Substituting Eq. (3.31) in Eq. (3.22), and the value of x into Eq. (3.23), we obtain $x = 0$ & $y = 0$.

Substituting Eq. (3.31) and the value of x into Eq. (3.26), and Eq. (3.31) into Eq. (3.21), it gives $z = 0$ & $v = 0$. Furthermore, Substituting $y = 0$ & $v = 0$ into Eq. (3.21), we obtain Eq. (3.36)

$$r = \frac{\beta}{\mu} n \tag{3.36}$$

Consequently, the equilibrium condition that is free of disease is provided by:

$$E_0 = (r, v, w, x, y, z) = \left(\frac{\beta}{\mu} n, 0, 0, 0, 0, 0 \right) \tag{3.37}$$

3.3. Endemic equilibrium

When the disease is persistent and conditions are met, an endemic equilibrium exist.

$$E_e = (S(t), L(t), I(t), Q(t), R(t), D(t))$$

Such that

$$S(t) > 0, L(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0, D(t) > 0 \tag{3.38}$$

and

$$S(t) + L(t) + I(t) + Q(t) + R(t) + D(t) \leq N$$

Adding (3.19) and (3.20) gives

$$(\beta - \mu)r + \beta y - (\gamma + \mu)v, \tag{3.39}$$

from (3.21) gives

$$v = \frac{(\varphi + \mu + \delta_1)w}{\gamma}, \tag{3.40}$$

Considering Eq. (3.22), it gives

$$w = \frac{(\tau + \mu + \delta_2)x}{\varphi}, \tag{3.41}$$

then from Eq. (3.23) gives

$$y = \frac{\tau}{\mu}x. \tag{3.42}$$

Substituting Eq. (3.40) and Eq. (3.42) in Eq. (3.39), we obtain

$$\mu(\beta - \mu)r + \beta\tau\gamma x - \mu(\mu + \gamma - \beta)(\varphi + \mu + \delta_1)w = 0, \tag{3.43}$$

Substituting Eq. (3.41) in Eq. (3.43), we obtain

$$\mu\varphi(\beta - \mu)r + \beta\tau\gamma\varphi x - \mu(\mu + \gamma - \beta)(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)x = 0, \tag{3.44}$$

Substituting Eq. (3.35) in Eq. (3.44) gives

$$x = \frac{A}{B}. \tag{3.45}$$

Where

$$A = \mu\gamma\varphi\zeta(\beta - \mu)(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)(\gamma + \mu)n \tag{3.46}$$

$$B = ((1 - \xi)(\alpha_1\zeta + \alpha_2(1 - \zeta)\mu + \delta_1)(\tau + \mu + \delta_2) + \varphi(\mu + \delta_2))(\gamma + \mu - \beta)(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2) - \beta\gamma\varphi\tau \tag{3.47}$$

Substituting Eq. (3.45) into Eq. (3.41), gives

$$w = \frac{(\tau + \mu + \delta_2)A}{\varphi B} \tag{3.48}$$

Substituting Eq. (3.45) into Eq. (3.42), gives

$$y = \frac{\tau A}{\mu B} \tag{3.49}$$

Substituting Eq. (3.48) into Eq. (3.45), gives

$$v = \frac{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)A}{\gamma\varphi B} \tag{3.50}$$

Substituting Eqs. (3.45) and (3.48) in Eq. (3.26) gives

$$z = \frac{(\mu + \delta_1)B + (\mu + \delta_2)A}{\zeta B} \tag{3.51}$$

Hence Eqs. (3.35) and (3.45) to (3.51) give the endemic equilibrium state of the model.

$$E^e = \begin{bmatrix} r \\ v \\ w \\ x \\ y \\ z \end{bmatrix} = \begin{bmatrix} \frac{\zeta(\tau + \mu + \delta_2)(\varphi + \mu + \delta_1)(\gamma + \mu)n}{\alpha_1\zeta + \alpha_2(1 - \zeta)(\mu + \delta_1)(\tau + \mu + \delta_2) + \varphi\alpha_2(1 - \zeta)(\mu + \delta_2)} \\ \frac{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)A}{\gamma\varphi B} \\ \frac{(\tau + \mu + \delta_2)A}{\varphi B} \\ \frac{A}{B} \\ \frac{\tau A}{\mu B} \\ \frac{(\mu + \delta_1)wB + (\mu + \delta_2)A}{\zeta B} \end{bmatrix} \tag{3.52}$$

Where

$$A = \mu\gamma\varphi\zeta(\beta - \mu)(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)(\gamma + \mu)n$$

$$B = ((1 - \xi)(\alpha_1\zeta + \alpha_2(1 - \zeta)\mu + \delta_1)(\tau + \mu + \delta_2) + \varphi(\mu + \delta_2))((\gamma + \mu - \beta)\varphi + \mu + \delta_1)(\tau + \mu + \delta_2) - \beta\gamma\varphi\tau$$

The Jacobian of the model (3.18) to (3.22) is given by Eq. (3.53) (see Box I).

At disease free, (3.53) becomes

$$j(E_0) = \begin{pmatrix} \beta - \mu & \beta & -c & 0 & \beta & -d \\ 0 & \gamma - \mu & g & 0 & 0 & h \\ 0 & \gamma & j & 0 & 0 & 0 \\ 0 & 0 & \varphi & -l & 0 & 0 \\ 0 & 0 & 0 & \tau & -\mu & 0 \\ 0 & 0 & p & r & 0 & \zeta \end{pmatrix} \tag{3.54}$$

Where,

$$c = \frac{\alpha_1(1 - \xi)r}{N}, d = \frac{\alpha_2(1 - \zeta)(1 - \xi)r}{N},$$

$$g = \frac{\alpha_1(1 - \xi)r}{N}, h = \frac{\alpha_2(1 - \zeta)(1 - \xi)r}{N},$$

$$j = (\varphi - \mu - \delta_1), l = (\tau - \mu - \delta_2),$$

$$p = (\mu + \delta_1), r = (\mu + \delta_2)$$

Using reduced row echelon form gives Eq. (3.55)

$$\begin{pmatrix} \beta - \mu & \beta & -c & 0 & \beta & -d \\ 0 & \gamma - \mu & g & 0 & 0 & h \\ 0 & \gamma & \frac{g\gamma + j\gamma + j\mu}{\gamma + \mu} & 0 & 0 & \frac{\gamma h}{\gamma + \mu} \\ 0 & 0 & 0 & -l & 0 & -\frac{\varphi\gamma h}{g\gamma + j\gamma + j\mu} \\ 0 & 0 & 0 & 0 & -\mu & -\frac{\tau\varphi\gamma h}{l(g\gamma + j\gamma + j\mu)} \\ 0 & 0 & 0 & 0 & 0 & \frac{g\gamma l\zeta - \gamma hlp - \gamma hr\varphi + \gamma j l\zeta + j l\mu\zeta}{l(g\gamma + j\gamma + j\mu)} \end{pmatrix} \tag{3.55}$$

Thus, the eigenvalues are:

$$\lambda_1 = \beta - \mu < 0 \tag{3.56}$$

$$\lambda_2 = -\gamma - \mu < 0 \tag{3.57}$$

$$\lambda_3 = \frac{g\gamma + j\gamma + j\mu}{\gamma + \mu} < 0 \tag{3.58}$$

$$\lambda_4 = -(\tau - \mu - \delta_2) < 0 \tag{3.59}$$

$$\lambda_5 = -\mu < 0 \tag{3.60}$$

$$\begin{pmatrix}
 (\beta - \mu) & & & & & & \\
 -\left(\frac{\alpha_1 w}{n} + \frac{\alpha_2(1-\xi)z}{n}\right)(1-\xi) & \beta & -\frac{\alpha_1(1-\xi)r}{n} & \beta & 0 & -\frac{\alpha_2(1-\xi)(1-\xi)r}{n} & \\
 \left(\frac{\alpha_1 w}{n} + \frac{\alpha_2(1-\xi)z}{n}\right)(1-\xi) & -(\gamma + \mu) & \frac{\alpha_1(1-\xi)r}{n} & \frac{\alpha_2(1-\xi)(1-\xi)r}{n} & 0 & 0 & \\
 0 & \gamma & (\varphi - \mu - \delta_1) & 0 & 0 & 0 & \\
 0 & 0 & \varphi & -(\tau - \mu - \delta_2) & 0 & 0 & \\
 0 & 0 & 0 & \tau & -\mu & 0 & \\
 0 & 0 & (\mu + \delta_1) & (\mu + \delta_2) & 0 & -\zeta &
 \end{pmatrix} \tag{3.53}$$

Box 1.

$$\lambda_6 = \frac{g\gamma l\zeta - \gamma hlp - \varphi\gamma hr + \gamma jl\zeta + jl\mu\zeta}{l(g\gamma + j\gamma + j\mu)} < 0 \tag{3.61}$$

Therefore λ_1 is negative if $\beta < \mu$

λ_2 is negative since $\lambda_2 = -\gamma - \mu < 0$

λ_4 is negative if $\tau > \mu + \delta_2$

λ_5 is negative since $\lambda_5 = -\mu < 0$

For λ_3 to be negative, $g\gamma + j\gamma + j\mu$ must be negative and so from

$$\lambda_3 = \frac{g\gamma + j\gamma + j\mu}{\gamma + \mu} < 0 \text{ we must have } \gamma + j\gamma + j\mu < 0$$

Also, for $\lambda_6 = \frac{g\gamma l\zeta - \gamma hlp - \varphi\gamma hr + \gamma jl\zeta + jl\mu\zeta}{l(g\gamma + j\gamma + j\mu)} < 0$ to be negative, then $g\gamma l\zeta - \gamma hlp - \varphi\gamma hr + \gamma jl\zeta + jl\mu\zeta < 0$. From $-[(\tau - \mu - \delta_2)g\gamma\zeta - (\tau - \mu - \delta_2)\gamma hlp + \varphi\gamma hr + (\tau - \mu - \delta_2)\gamma j\zeta + (\tau - \mu - \delta_2)j\mu\zeta] < 0$ and $l = -(\tau - \mu - \delta_2) < 0$, it implies that $\tau > \mu + \delta_2$.

3.4. The basic reproduction number R_0 and the effective basic reproduction number, R_{eff}

Any infectious disease's capacity to spread throughout a population is one of the most significant causes for concern and as such, there is a disease-free equilibrium (DFE) in many epidemiological models, where the population continues to be healthy. Typically, these models have a threshold parameter, also known as the basic reproduction number, R_0 such that if $R_0 < 1$, then the disease free equilibrium (DFE) is locally asymptotically stable, and the disease is unable to spread among people, but if $R_0 > 1$, then the disease free equilibrium (DFE) is unstable and invasion is always possible. The basic reproduction number R_0 [23], represents the average number of secondary cases that an infected person would produce over the course of the infection period if they were introduced into a susceptible population without disease immunity in the absence of interventions to control the infection. If $R_0 < 1$, then an infected person would typically produce fewer than one newly infected person. If so, the infection might eventually go away. On the other hand, the virus may spread throughout a population if $R_0 > 1$ is more than one and each infected person (primary case) develops, on average, more than one new infection.

The approach of Diekmann et al. [24] is employed and analysed by Driessche et al. [25] known as the spectral radius. This approach is employed in finding the effective basic reproduction number, R_{eff} of the system (2.1) to (2.6) which represent the spectra radius (ρ) in using the next generation matrix, K , i.e. $R_{eff} = \rho K$, where $K = FV^{-1}$.

F and V are obtained from the Jacobian (3.53), about the disease-free equilibrium. F represents the matrix for the new infection terms and V the matrix for the transition terms. The matrices F and V are formed from the coefficient of the infected classes.

$$F = \begin{pmatrix} 0 & \frac{S}{N}\alpha_1(1-\xi) & 0 & \frac{S}{N}\alpha_2(1-\xi) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

For easy simplification, let $M = \frac{S}{N}\alpha_1(1-\xi)$ and $N = \frac{S}{N}\alpha_2(1-\xi)$ and matrix F becomes

$$F = \begin{pmatrix} 0 & M & 0 & N \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = -\begin{pmatrix} -(\gamma + \mu) & 0 & 0 & 0 \\ \gamma & (\varphi + \mu + \delta_1) & 0 & 0 \\ 0 & \varphi & -(\tau + \mu + \delta_2) & 0 \\ 0 & \mu + \delta_1 & \mu + \delta_2 & -\zeta \end{pmatrix}$$

To obtain matrix V^{-1} , the Gauss-Jordan elimination method is employed, with the applied operations, we obtain V^{-1} as seen Eq. (3.62)

$$V^{-1} = \begin{bmatrix} \frac{1}{\gamma + \mu} & 0 & 0 & 0 \\ \frac{\gamma}{(\gamma + \mu)(\varphi + \mu + \delta_1)} & \frac{1}{\varphi + \mu + \delta_1} & 0 & 0 \\ -\frac{\gamma\varphi}{(\gamma + \mu)(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)} & -\frac{\varphi}{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)} & \frac{1}{(\tau + \mu + \delta_2)} & 0 \\ \frac{\gamma((\tau + \mu + \delta_2)(\mu + \delta_1) - \varphi(\mu + \delta_2))}{(\gamma + \mu)(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)\zeta} & \frac{(\mu + \delta_1)(\tau + \mu + \delta_2) - \varphi(\mu + \delta_2)}{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)\zeta} & \frac{(\mu + \delta_2)}{(\tau + \mu + \delta_2)\zeta} & \frac{1}{\zeta} \end{bmatrix} \tag{3.62}$$

Employing the next generation matrix, we obtain Eq. (3.63)

$$FV^{-1} = \begin{bmatrix} Y_1 & Y_2 & Y_3 & Y_4 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{3.63}$$

where

$$\begin{aligned}
 Y_1 &= \frac{\gamma M_1}{(\varphi + \mu + \delta_1)} + \frac{\gamma M_2((\varphi + \mu + \delta_1)(\tau + \mu + \delta_2) - \varphi(\mu + \delta_2))}{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)\zeta} \\
 Y_2 &= M_2((\varphi + \mu + \delta_1)(\tau + \mu + \delta_2) - \frac{\varphi(\mu + \delta_2)}{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)\zeta}) + \frac{M_1}{B} \\
 Y_3 &= \frac{\gamma M_2}{(\varphi + \mu + \delta_1)\zeta} \\
 Y_4 &= \frac{M_2}{\zeta}
 \end{aligned}$$

We compute the eigenvalues to obtain the effective reproduction number R_{eff} taking the spectral radius of the matrix FV^{-1} . In doing, we

employed by Eq. (3.63), we obtain Eq. (3.64),

$$FV^{-1} = \begin{bmatrix} Y_1 & Y_2 & Y_3 & Y_4 \\ 0 & 0 - \lambda & 0 & 0 \\ 0 & 0 & 0 - \lambda & 0 \\ 0 & 0 & 0 & 0 - \lambda \end{bmatrix} \tag{3.64}$$

Eigenvalues λ_i where $i = 1, 2, 3, 4$ were obtained and are given by:

$$\lambda_1 = \frac{\gamma\alpha_1(1-\xi)}{(\varphi + \mu + \delta_1)} + \frac{\gamma\alpha_2(1-\xi)((\varphi + \mu + \delta_1)(\tau + \mu + \delta_2) - \varphi(\mu + \delta_2))}{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)\zeta}$$

λ_1 is the eigenvalue, which follows the effective basic reproduction number R_{eff} is given as:

$$R_{eff} = \frac{\gamma\alpha_1(1-\xi)(\tau + \mu + \delta_2)\zeta + \gamma\alpha_2(1-\xi)((\varphi + \mu + \delta_1)(\tau + \mu + \delta_2) - \varphi(\mu + \delta_2))}{(\varphi + \mu + \delta_1)(\tau + \mu + \delta_2)\zeta} \tag{3.65}$$

It imperative to note that Eq. (3.65) is the effective basic reproduction number.

3.5. Stability analysis of endemic equilibrium state

Bifurcation theory is used in the stability analysis of endemic equilibrium state. We consider the nature of the endemic equilibrium state of the model (3.19)–(3.25) near DFE (bifurcation point), $R_{eff} = 1$. Let α be the bifurcation parameter such that $R_{eff} < 1$ for $\alpha < 0$ and $R_{eff} > 1$ for $\alpha > 0$ such that the disease free equilibrium exists for all values of α . The results of the centre manifold theory are used to show that there is nontrivial endemic equilibrium near the disease-free equilibrium. To state these results, the partial derivatives of f with respect to x is evaluated at DFE, $\alpha = 0$. Let u and v be the left and right eigenvectors chosen such that $uv = 1$.

We Let a and b be the following:

$$a = \sum_{k,i,j=1}^n v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} \tag{3.66}$$

$$b = \sum_{k,i,j=1}^n v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha_j} \tag{3.67}$$

We then demonstrate that the nature of the endemic equilibrium near the disease-free region depends on the sign.

To begin, we make change of variables.

$$x_1 = r, \quad x_2 = v, \quad x_3 = w, \quad x_4 = x, \quad x_5 = y, \quad x_6 = z$$

Through the use of vector notation,

$$X = (x_1, x_2, x_3, x_4, x_5)^T$$

the model equations (3.19) to (3.25) now becomes,

$$\frac{dx}{dt} = (f_1, f_2, f_3, f_4, f_5)^T \tag{3.68}$$

such that:

$$\frac{dx_1}{dt} = f_1 = \beta(x_1 + x_2 + x_4) - \left(\frac{\alpha_1 x_3}{n} + \frac{\alpha_2(1-\zeta)x_6}{n}\right) (1-\xi)x_1 - \mu x \tag{3.69}$$

$$\frac{dx_2}{dt} = f_2 = \left(\frac{\alpha_1 x_3}{n} + \frac{\alpha_2(1-\zeta)x_6}{n}\right) (1-\xi)x_1 - (\gamma + \mu)x_2 \tag{3.70}$$

$$\frac{dx_3}{dt} = f_3 = \gamma x_2 - (\varphi + \mu + \delta_1)x_3 \tag{3.71}$$

$$\frac{dx_4}{dt} = f_4 = \varphi x_3 - (\tau + \mu + \delta_2)x_4 \tag{3.72}$$

$$\frac{dx_5}{dt} = f_5 = \tau x_4 - \mu x_5 \tag{3.73}$$

$$\frac{dx_6}{dt} = f_6 = (\mu + \delta_1)x_3 + (\mu + \delta_2)x_4 - \zeta x_6 \tag{3.74}$$

The Jacobian of the (3.69) to (3.74) at disease free region is obtained as in Box II.

Showing that the Jacobian of (3.69)–(3.74) at α has a right eigenvector, the following equations holds;

$$(\beta - \mu)v_1 + \beta v_2 - \frac{\alpha_1(1-\xi)x_1}{n}v_3 + \beta v_5 - \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n}v_6 = 0 \tag{3.76}$$

$$-(\gamma + \mu)v_2 + \frac{\alpha_1(1-\xi)x_1}{n}v_3 + \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n}v_6 = 0 \tag{3.77}$$

$$\gamma v_2 + (\varphi - \mu - \delta_1)v_3 = 0 \tag{3.78}$$

$$\varphi v_3 - (\tau - \mu - \delta_2)v_4 = 0 \tag{3.79}$$

$$\tau v_4 - \mu v_5 = 0 \tag{3.80}$$

$$(\mu + \delta_1)v_3 + (\mu + \delta_2)v_4 + \zeta v_6 = 0 \tag{3.81}$$

Solving (3.76) to (3.81) gives

$$v_1 = \begin{bmatrix} A_1 \\ A_2 \end{bmatrix} v_4 \tag{3.82}$$

where

$$a_1 = (\gamma + \mu)(\varphi - \mu - \delta_1)(\tau - \mu - \delta_2)n - \alpha_1\gamma(\tau - \mu - \delta_2)(1-\xi)x_1$$

$$A_1 = \frac{\alpha_1(1-\xi)x_1(\tau - \mu - \delta_2) + \varphi n \beta \tau}{\varphi n \mu (\beta - \mu)}$$

$$A_2 = \frac{\alpha_1 - \beta \alpha_2(\varphi - \mu - \delta_1)(\tau - \mu - \delta_2)(1-\zeta)(1-\xi)x_1}{\gamma \varphi \alpha_2(1-\zeta)(1-\xi)(\beta - \mu)x_1}$$

$$v_2 = \frac{(\varphi - \mu - \delta_1)(\tau - \mu - \delta_2)}{\gamma \varphi} v_4 \tag{3.83}$$

$$v_3 = \frac{(\tau - \mu - \delta_2)}{\varphi} v_4 \tag{3.84}$$

$$v_5 = \frac{\tau}{\mu} v_4 \tag{3.85}$$

$$v_6 = \frac{((\gamma + \mu)(\varphi - \mu - \delta_1)(\tau - \mu - \delta_2)n - \alpha_1\gamma(\tau - \mu - \delta_2)(1-\xi)x_1)}{\gamma \varphi \alpha_2(1-\zeta)(1-\xi)x_1} v_4 \tag{3.86}$$

and $v_4 > 0$. Also, the Jacobian of (3.69)–(3.84) at $\alpha_i = \alpha$ the left eigenvector $U = [u_1, u_2, u_3, u_4, u_5, u_6]$ (see Box III).

For easy simplification, let $p = \frac{\alpha_1(1-\xi)x_1}{n}$; $q = \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n}$; $s = (\varphi - \mu - \delta_1)$; $t = (\tau - \mu - \delta_2)$

$$[u_1, u_2, u_3, u_4, u_5, u_6] \begin{bmatrix} \beta - \mu & \beta & -p & 0 & \beta & -q \\ 0 & -(\gamma + \mu) & p & 0 & 0 & q \\ 0 & \gamma & s & 0 & 0 & 0 \\ 0 & 0 & \phi & -t & 0 & 0 \\ 0 & 0 & 0 & \tau & -\mu & 0 \\ 0 & 0 & (\mu + \beta) & (\mu + d) & 0 & \zeta \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{3.88}$$

$$[u_1, u_2, u_3, u_4, u_5, u_6] \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & -\frac{\gamma}{\gamma + \mu} & 1 & 0 & 0 & 0 \\ 0 & 0 & \frac{\phi(\gamma + \mu)}{\gamma p + \gamma s + \mu s} & 1 & 0 & 0 \\ 0 & 0 & 0 & -\frac{\tau}{\tau} & 1 & 0 \\ 0 & 0 & \frac{(\mu + \beta)(\gamma + \mu)}{\gamma p + \gamma s + \mu s} & -\frac{\mu + d}{\tau} & 0 & 1 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{3.89}$$

$$J(E_0) = - \begin{pmatrix} (\beta - \mu) & \beta & -\frac{\alpha_1(1-\xi)x_1}{n} & 0 & \beta & -\frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n} \\ 0 & -(\gamma + \mu) & \frac{\alpha_1(1-\xi)x_1}{n} & 0 & 0 & \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n} \\ 0 & \gamma & (\varphi - \mu - \delta_1) & 0 & 0 & 0 \\ 0 & 0 & \varphi & -(\tau - \mu - \delta_2) & 0 & 0 \\ 0 & 0 & 0 & \tau & -\mu & 0 \\ 0 & 0 & (\mu + \delta_1) & (\mu + \delta_2) & 0 & \zeta \end{pmatrix} \tag{3.75}$$

Box II.

$$VJ(E_0) = \begin{bmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \\ u_5 \\ u_6 \end{bmatrix} \begin{pmatrix} (\beta - \mu) & \beta & -\frac{\alpha_1(1-\xi)x_1}{n} & 0 & \beta & -\frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n} \\ 0 & -(\gamma + \mu) & \frac{\alpha_1(1-\xi)x_1}{n} & 0 & 0 & \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n} \\ 0 & \gamma & (\varphi - \mu - \delta_1) & 0 & 0 & 0 \\ 0 & 0 & \varphi & -(\tau - \mu - \delta_2) & 0 & 0 \\ 0 & 0 & 0 & \tau & -\mu & 0 \\ 0 & 0 & (\mu + \delta_1) & (\mu + \delta_2) & 0 & \zeta \end{pmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{3.87}$$

Box III.

Considering Eq. (3.89), Eq. (3.90)–Eq. (3.94) holds the following

$$u_1 = u_2 = 0 \tag{3.90}$$

$$u_3 = u_4 = u_6 > 0 \tag{3.91}$$

$$u_2 = \frac{(\gamma + \mu)}{\gamma} u_3 \tag{3.92}$$

$$u_4 = -\frac{\varphi(\gamma + \mu)}{\gamma p + \lambda s + \mu s} u_3 \tag{3.93}$$

$$u_6 = -\frac{(\varphi(\mu + \delta_2)(\gamma + \mu)t + (\mu + \delta_1)(\gamma + \mu))}{(\gamma p + \gamma s + \mu s)t} u_3 \tag{3.94}$$

Where,

$$p = \frac{\alpha_1(1-\xi)x_1}{n},$$

$$q = \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n},$$

$$s = (\varphi - \mu - \delta_1)$$

$$t = (\tau - \mu - \delta_2)$$

From the above derived results, one can interpret that if the individuals are quarantined and treated instead of taking home remedies for treatment at asymptomatic or minor symptomatic stage, the spread of the disease can be controlled to a greater extent. Moreover, effective public campaigns, decontamination and proper burial of the deceased, and isolation from infectious individuals are enough and more effective to control the spread of the disease.

3.6. Computation of a and b

The sign of a is determined by computing the partial derivatives of the system (3.69)–(3.84), associated with a.

Theorem 3. In considering Eq. (3.19)–(3.25), we defined a and b by (3.85a) and (3.85b) and assume $b \neq 0, \exists \delta > 0$ such that

- (1) if $a < 0$, there is locally asymptotically stable endemic equilibrium near the disease-free equilibrium for $0 < a < \delta$ and
- (2) if $a > 0$, there are unstable endemic equilibria near the DFE for $-\delta < a < 0$

$$a = \sum_{k,i,j=1}^n v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0, 0) \tag{3.95}$$

For the first partial derivatives, refer to the appendix Appendix A.

This same procedure was computed for the second partial derivative which will not be computed here but we computed the associated at the disease-free, the partial derivative is non-zero for a and b, refer to the appendix Appendix B

4. Sensitivity analysis

In this paper, we look at the sensitivity of the fundamental reproduction number R_0 to each of its terms, which may be calculated as

$$S_{(\cdot)'} = \frac{(\cdot)'}{R_{eff}} \times \frac{\partial R_{eff}}{\partial (\cdot)'}$$

where $(\cdot)'$ represents each parameter. This is represented in Table 2 as follows.

We provide the sensitivity index of the parameters each to R_0 . This can be seen in Fig. 2

From Fig. 2, it can be deduced that β with a sensitivity value of 0, does not have much effect R_0 within the model. With α_1 and α_2 , they seem to have a notable impact on R_0 . Higher sensitivity for α_2 (0.93) implies that small variations in α_2 lead to more significant changes in

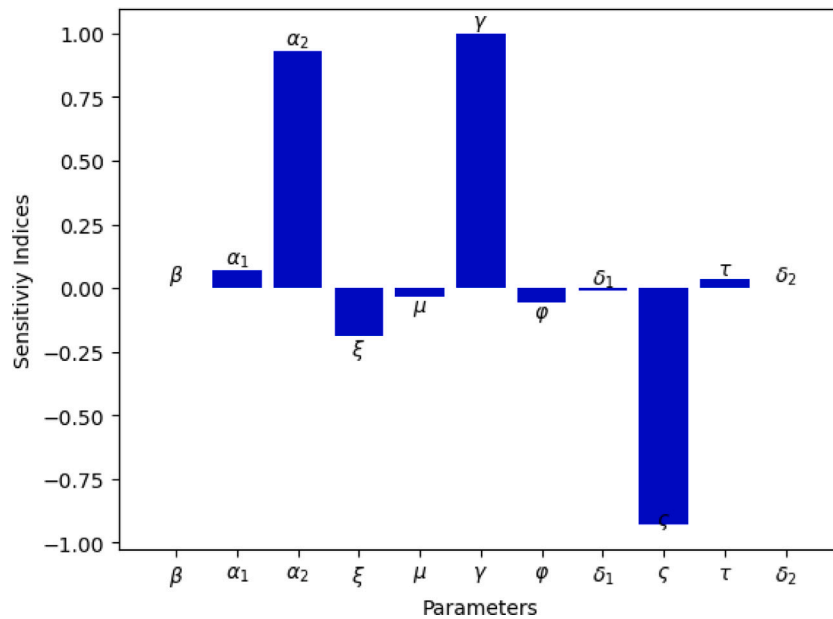


Fig. 2. R_0 Sensitivity of each parameter.

Table 2
Sensitivity analysis of the parameters.

Parameters	Sensitivity values
β	0
α_1	0.0705442476049881
α_2	0.929455752395012
μ	-0.0336194442093716
φ	-0.0565187672044668
γ	1
δ_1	-0.0138318251654430
δ_2	-0.000467961045640111
τ	0.0338937500199337
ξ	-0.190476190476190
ζ	-0.929455752395012

R_0 compared to α_1 (0.07). These parameters might represent rates of progression from one disease state to another (e.g., from exposed to infected). The following parameters $\mu, \varphi, \delta_1, \delta_2, \xi, \zeta$ all have negative sensitivity values, suggesting that changes in these parameters tend to decrease R_0 . They might represent various factors like recovery rates, interventions, or behavioural changes that reduce the disease's transmission potential. γ : With a sensitivity value of 1, γ seems to have a significant impact, directly influencing R_0 . γ often denotes the recovery rate or the reciprocal of the infectious period. In summary, parameters with higher positive sensitivity values, like α_2 and γ , have more substantial effects on increasing R_0 , while those with negative sensitivity values, like $\mu, \varphi, \delta_1, \delta_2, \xi$, and ζ , tend to decrease R_0 when altered. Parameters with sensitivity values close to 0, like β , have minimal influence on R_0 .

5. Numerical analysis

5.1. Analytical solution of the governing model via Homotopy Perturbation Method (HPM)

Ji-Haun [26] made the initial discovery of the Homotopy Perturbation Method (HPM). Numerous linear and non-linear equations can be solved analytically approximatively using the Homotopy Perturbation Method (HPM) and the series expansion method known as the homotopy perturbation method (HPM) is used to solve nonlinear partial differential equations.

5.2. Solution of the model equations

Given initial conditions

$$S(0) = S_0, \quad L(0) = L_0, \quad I(0) = I_0, \quad Q(0) = Q_0, \\ R(0) = R_0, \quad D(0) = D_0 \tag{5.1}$$

We consider the model of Eq. (5.2) - Eq. (5.7)

$$\frac{dS}{dt} = \beta(S + L + R) - \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2 (1 - \zeta) D}{N} \right) (1 - \xi) S - \mu S \tag{5.2}$$

$$\frac{dL}{dt} = \left(\frac{\alpha I}{N} + \frac{\alpha_1 (1 - \zeta) D}{N} \right) (1 - \xi) S - (\gamma + \mu) L \tag{5.3}$$

$$\frac{dI}{dt} = \gamma L - (\varphi + N + \delta_1) I \tag{5.4}$$

$$\frac{dQ}{dt} = \varphi I - (\tau + \mu + \delta_2) Q \tag{5.5}$$

$$\frac{dR}{dt} = \tau Q - \mu R \tag{5.6}$$

$$\frac{dD}{dt} = (\mu + \delta_1) I + (\mu + \delta_2) Q - \zeta D \tag{5.7}$$

From Eq. (5.1), We let,

$$S = a_0 + p a_1 + p^2 a_2 + \dots \\ L = b_0 + p b_1 + p^2 b_2 + \dots \\ I = c_0 + p c_1 + p^2 c_2 + \dots \\ Q = d_0 + p d_1 + p^2 d_2 + \dots \\ R = e_0 + p e_1 + p^2 e_2 + \dots \\ D = f_0 + p f_1 + p^2 f_2 + \dots \tag{5.8}$$

Applying HPM (5.7) using (5.8) we obtain the following equations which can be seen in Appendix C:

5.3. Visualization of results

In this section, provide the Table 3 with the parameters used for the analysis and the visualization of the results in Figs. 3–10. In the figures presented, we varied some of the parameters to understand the dynamics of the disease.

The initial conditions are fixed as follows:

$$S_0 = 439652, \quad L_0 = 238650, \quad I_0 = 201000, \quad Q_0 = 176000, \quad R_0 = 120000, \quad \text{and} \quad D_0 = 20000.$$

Table 3
Parameter values.

Parameters	Values	References
β	0.23632	[27]
α_1	3.92	[27]
α_2	3.36	[28]
μ	0.02	Assumed
φ	2.10	[28]
γ	4.10	[27]
δ_1	1.42850	Assumed
δ_2	0.00028	[29]
τ	0.30	[29]
ξ	0.16	[28]
ζ	0.2222	[29]

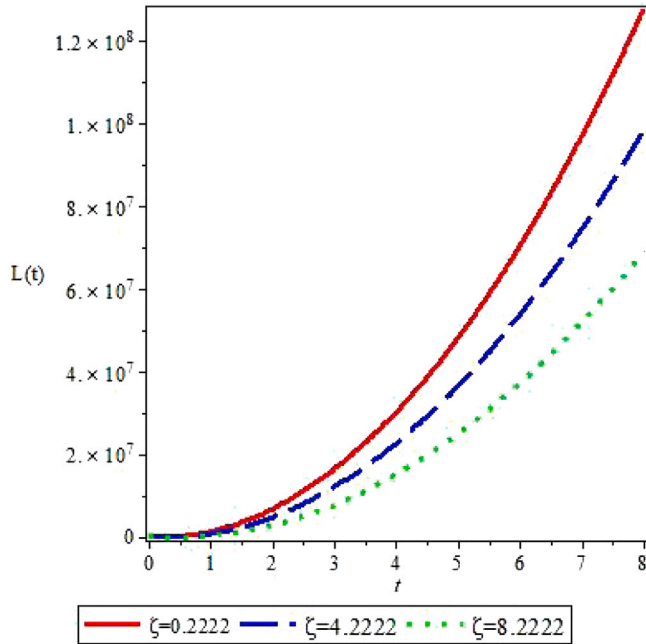


Fig. 3. Latent class against Time for varying values of ζ (t - weeks).

The Table 3 shows the values for the parameters and references for the values while other were assumed.

Visualization of the results is presented in Figs. 3–10

6. Discussion of results, conclusion and future work

6.1. Discussion of results and conclusion

To demonstrate the disease dynamics, we simulate the model we developed and varied some of the parameters in order to validate our model and also confirm our qualitative analysis results. Fig. 3 shows that when we have large decontamination in the population and they are removed from the population, it will help to decreasing the number of persons exposed to diseases hence aiding in stopping the transmission of the virus. Similarly, in Fig. 4, increasing public campaigns and making it effective will help to reduce the exposed individuals in the population and vice versa. Fig. 5 demonstrates the fact that if we have more people moved from the exposed to the infected class, there will be rapid spread in the disease in the population which makes it unsafe for the locality considered. Increasing the rate at which people are quarantine will aid to mitigate in reducing the disease’s spread as shown in Fig. 6. Fig. 7 and Fig. 8 helps to demonstrate the importance of recovery in our analysis. By varying the treatment rate shows that

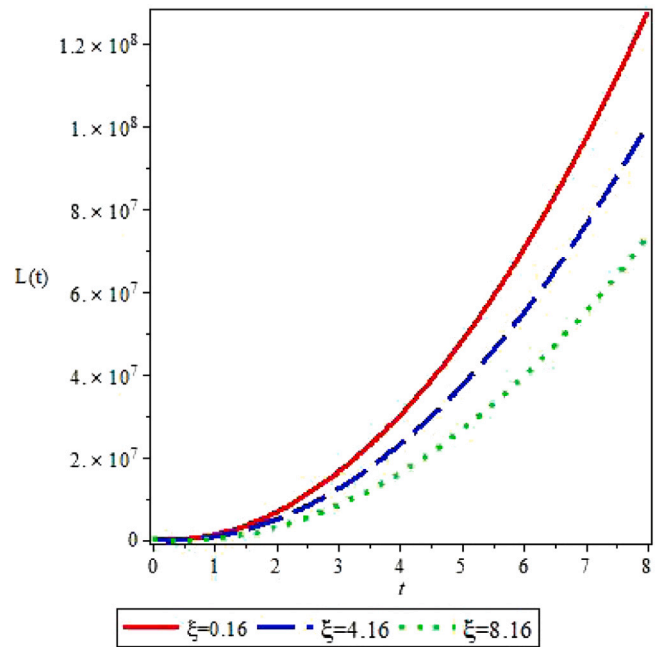


Fig. 4. Latent class against Time for varying values of ξ (t - weeks).

if it is increased, there will be increase in the number of recovery, showing the importance of prompt treatment of those infected by the virus and also having many deaths which are not properly handled will affect the way at which people will recover. Finally, Fig. 9 and Fig. 10 only shows how important and vital is birth and death rates in the susceptible class which will either make the population to grow or go into extinction.

We have been able to provide some qualitative analysis of our result and more importantly, we employed bifurcation theory to analyse the stability of the endemic equilibrium state and the general bifurcation theory is employed to demonstrate the endemic equilibrium state’s existence. We investigate how the nature of the endemic equilibrium state of the model (3.19)–(3.25) near the DFE, $R_{eff} = 1$. It is demonstrated through the use of the centre manifold theory that a nontrivial endemic equilibrium exists close to the disease-free equilibrium (bifurcation point). To state these results, the partial derivatives of f with respect to x is evaluated at disease free equilibrium (bifurcation point). We have demonstrated that if the sign of a is positive, i.e., $a > 0$, then an unstable endemic equilibrium state arises, as the sign of the partial derivatives determines the sign of a . What we have in West Africa is a situation like this, which indicates that the disease is progressively declining and will be eradicated over time. In comparing our work to [30], we provided sensitivity analysis to show how our work give a better result. We also illuminated it is not sufficient enough to create awareness and put in place public campaign but also using public campaign to reduce the sting and poking stigmatization infer on people. While [31] considered contact tracing, we encourage the use of public campaigns which is aimed at reducing stigmatization surrounding Ebola outbreak offer a more effect than contact tracing. These initiatives educate the public on the pivotal role of contact tracing in curbing the virus’s spread, fostering increased cooperation and understanding among communities. By diminishing stigma, these campaigns encourage empathy and support for individuals undergoing contact tracing, ensuring they receive necessary medical attention without fear of social repercussions. Moreover, by mitigating stigma, more individuals are likely to willingly participate in contact tracing efforts, enhancing their effectiveness in identifying and containing the virus. These campaigns also prevent discrimination,

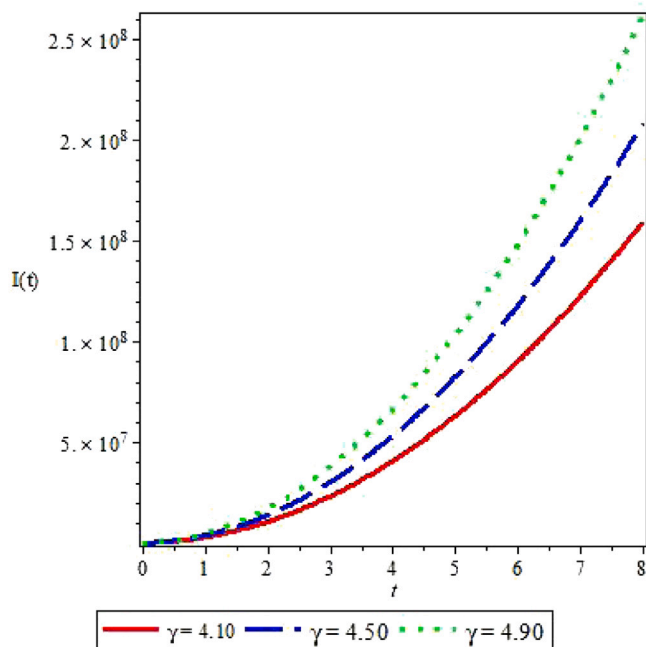


Fig. 5. Infectious class versus Time for varying values of γ (t - weeks).

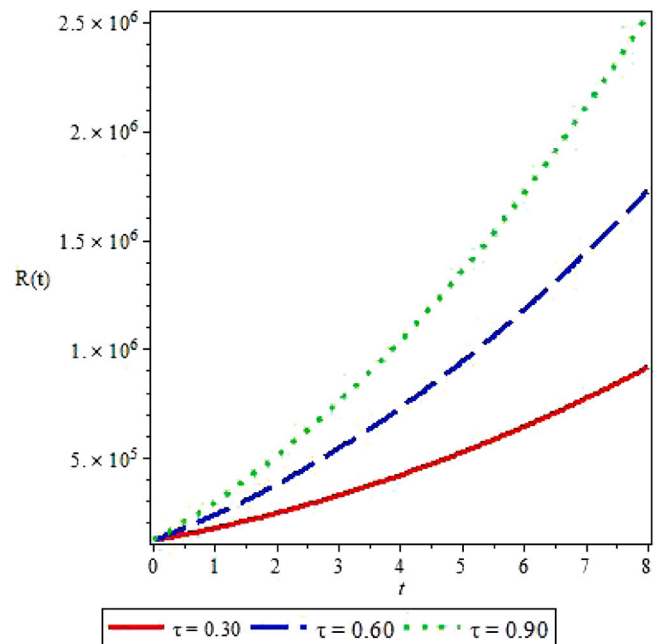


Fig. 7. Recovered class versus Time for varying values of τ (t - weeks).

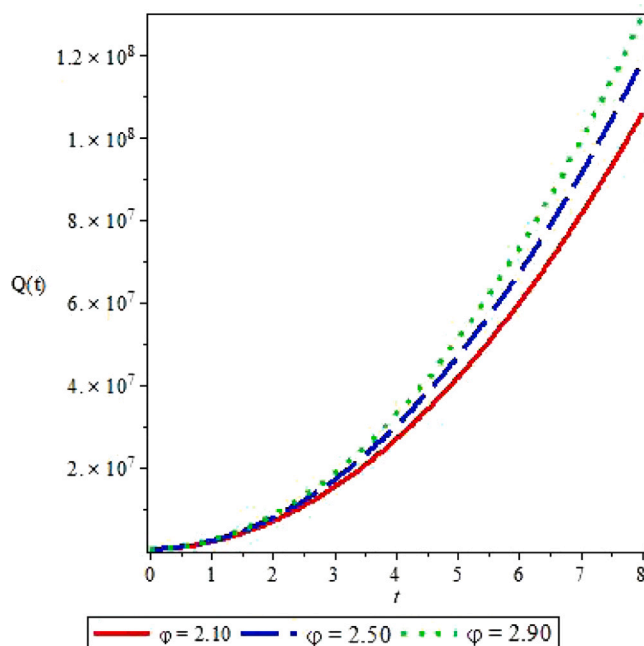


Fig. 6. Quarantined class versus Time for varying values of φ (t - weeks).

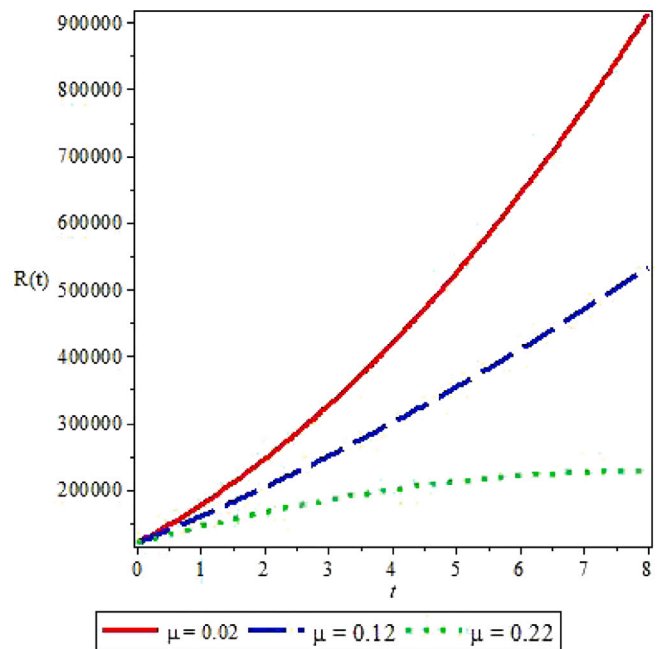


Fig. 8. Recovered class versus Time for varying values of μ (t - weeks).

promoting fair treatment and unbiased support for affected individuals or communities. Additionally, by addressing stigma, such initiatives alleviate the psychological burden on individuals and communities, supporting mental health resilience amidst an outbreak. Ultimately, these campaigns contribute not only to controlling the disease but also to fostering a more compassionate and trusting community response during a challenging public health crisis.

Conclusively, we have been able to show the importance of quarantine, treatment and public enlightenment campaign as essential preventative efforts to stop the disease's spread. This has been investigated to show the proper campaign will have a positive impact in reducing the

stigmatization which occurs at both internal and external phase of the infected individual. It is crucial to understand that stigmatization is a significant problem in society and that, if it is not well controlled, it can make it difficult for someone who has recovered from an infection to be accepted or included in society, as a result, effective campaigning is required. The result in this article will be useful for proper management of the Ebola virus disease.

6.2. Future work

In future, we do hope to expand the study to spatial modelling of the disease at small spatial scale in order to localize response but

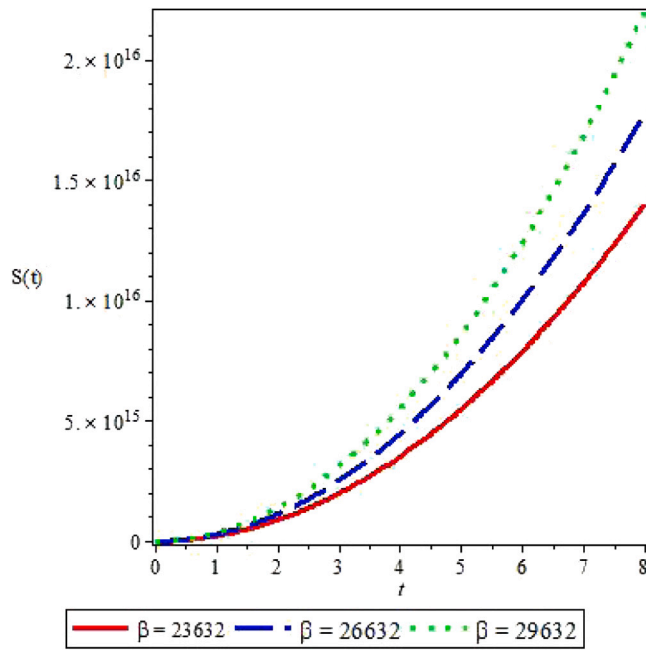


Fig. 9. Susceptible class versus Time for varying values of β (t - weeks).

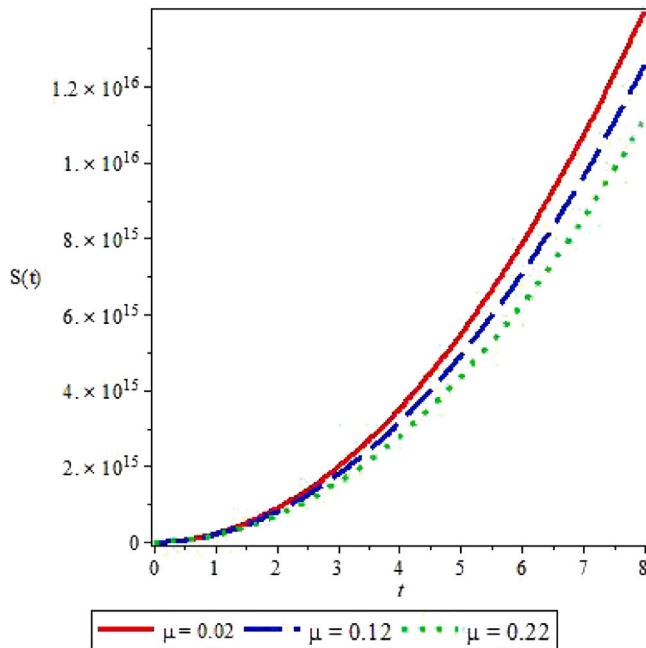


Fig. 10. Susceptible class versus Time for varying values of μ (t - weeks).

one of the challenges is getting accurate and precise data in order to develop a model that is driven by real data. This will enable us to focus on developing predictive models that can forecast the potential trajectory of the outbreak, enabling timely interventions and resource allocation to mitigate the spread of the disease. However, we tend to also investigate some pertinent determinants such as cross-border between countries using the integration of real-time data and advanced machine learning techniques to enhance the accuracy and timeliness of these models, facilitating more effective public health responses to future outbreaks of EVD.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Computation of a

$$\begin{aligned}
 f_1 &= \beta(x_1 + x_2 + x_4) - \left(\frac{\alpha_1 x_3}{n} + \frac{\alpha_2(1-\zeta)x_6}{n} \right) (1-\xi)x_1 - \mu x, \\
 \frac{\partial f_1}{\partial x_1}(0,0) &= \beta - \left(\frac{\alpha_1 x_3}{n} + \frac{\alpha_2(1-\zeta)x_6}{n} \right) (1-\xi) - \mu, \\
 \frac{\partial f_1}{\partial x_2}(0,0) &= \beta, \\
 \frac{\partial f_1}{\partial x_3}(0,0) &= -\frac{\alpha_1(1-\xi)}{n} x_1, \\
 \frac{\partial f_1}{\partial x_4}(0,0) &= \beta, \\
 \frac{\partial f_1}{\partial x_5}(0,0) &= 0, \\
 \frac{\partial f_1}{\partial x_6}(0,0) &= -\frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n}.
 \end{aligned}
 \tag{A.1}$$

$$\begin{aligned}
 f_2 &= \left(\frac{\alpha_1 x_3}{n} + \frac{\alpha_2(1-\zeta)x_6}{n} \right) (1-\xi)x_1 - (\gamma + \mu)x_2, \\
 \frac{\partial f_2}{\partial x_1}(0,0) &= \left(\frac{\alpha_1 x_3}{n} + \frac{\alpha_2(1-\zeta)x_6}{n} \right) (1-\xi), \\
 \frac{\partial f_2}{\partial x_2}(0,0) &= -(\gamma + \mu), \\
 \frac{\partial f_2}{\partial x_3}(0,0) &= \frac{\alpha_1}{n}, \\
 \frac{\partial f_2}{\partial x_4}(0,0) &= 0, \\
 \frac{\partial f_2}{\partial x_5}(0,0) &= 0, \\
 \frac{\partial f_2}{\partial x_6}(0,0) &= \frac{\alpha_2(1-\zeta)(1-\xi)x_1}{n}.
 \end{aligned}
 \tag{A.2}$$

$$\begin{aligned}
 f_3 &= \gamma x_2 - (\varphi + \mu + \delta_1)x, \\
 \frac{\partial f_3}{\partial x_1}(0,0) &= 0, \\
 \frac{\partial f_3}{\partial x_2}(0,0) &= \gamma, \\
 \frac{\partial f_3}{\partial x_3}(0,0) &= -(\varphi + \mu + \delta_1), \\
 \frac{\partial f_3}{\partial x_4}(0,0) &= 0, \\
 \frac{\partial f_3}{\partial x_5}(0,0) &= 0, \\
 \frac{\partial f_3}{\partial x_6}(0,0) &= 0.
 \end{aligned}
 \tag{A.3}$$

$$\begin{aligned}
 f_4 &= \varphi x_3 - (\tau + \mu + \delta_2)x_4 \\
 \frac{\partial f_4}{\partial x_1}(0,0) &= 0, \\
 \frac{\partial f_4}{\partial x_2}(0,0) &= 0, \\
 \frac{\partial f_4}{\partial x_3}(0,0) &= \varphi, \\
 \frac{\partial f_4}{\partial x_4}(0,0) &= -(\tau + \mu + \delta_2), \\
 \frac{\partial f_4}{\partial x_5}(0,0) &= 0,
 \end{aligned}$$

$$\frac{\partial f_4}{\partial x_6}(0, 0) = 0 \tag{A.4}$$

$$f_5 = \tau x_4 - \mu x_5,$$

$$\begin{aligned} \frac{\partial f_5}{\partial x_1}(0, 0) &= 0, \\ \frac{\partial f_5}{\partial x_2}(0, 0) &= 0, \\ \frac{\partial f_5}{\partial x_3}(0, 0) &= 0, \\ \frac{\partial f_5}{\partial x_4}(0, 0) &= \tau, \\ \frac{\partial f_5}{\partial x_5}(0, 0) &= \mu, \\ \frac{\partial f_5}{\partial x_6}(0, 0) &= 0, \end{aligned} \tag{A.5}$$

$$f_6 = (\mu + \delta_1)x_3 + (\mu + \delta_2)x_4 - \zeta x_6,$$

$$\begin{aligned} \frac{\partial f_6}{\partial x_1}(0, 0) &= 0, \\ \frac{\partial f_6}{\partial x_2}(0, 0) &= 0, \\ \frac{\partial f_6}{\partial x_3}(0, 0) &= (\mu + \delta_1), \\ \frac{\partial f_6}{\partial x_4}(0, 0) &= (\mu + \delta_2), \\ \frac{\partial f_6}{\partial x_5}(0, 0) &= 0, \\ \frac{\partial f_6}{\partial x_6}(0, 0) &= -\zeta \end{aligned} \tag{A.6}$$

Appendix B. Computation of b

$$\begin{aligned} a &= \sum_{k,i,j=1}^n v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, 0), \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_3}(0, 0) = \frac{\alpha_1(1-\xi)}{n}, \\ \frac{\partial^2 f_2}{\partial x_1 \partial x_6}(0, 0) &= \frac{\alpha_2(1-\zeta)(1-\xi)}{n}, \end{aligned} \tag{B.1}$$

Eq. (B.1) is represented further as follows

$$\begin{aligned} a &= \frac{v_1 u_1 u_3}{n} \alpha_1(1-\xi) + \frac{v_1 u_6 u_1}{n} \alpha_2(1-\zeta)(1-\xi) \\ &\quad - \left[\frac{v_1 u_3 u_1}{n} \alpha_1(1-\xi) + \frac{v_1 u_1 u_6}{n} \alpha_2(1-\zeta)(1-\xi) \right] \\ &\quad + \left[\frac{v_2 u_1 u_3}{n} \alpha_1(1-\xi) + \frac{v_2 u_1 u_6}{n} \alpha_2(1-\zeta)(1-\xi) + \frac{v_2 u_6 u_1}{n} \alpha_2(1-\zeta)(1-\xi) \right] \\ &> 0 \end{aligned} \tag{B.2}$$

We can deduce (B.3) from Eqs. (B.1) and (B.2) and represented as

$$\begin{aligned} a &= \frac{v_2 u_1 u_3}{n} \alpha_1(1-\xi) + \frac{v_2 u_1 u_6}{n} \alpha_2(1-\zeta)(1-\xi) + \frac{v_2 u_6 u_1}{n} \alpha_2(1-\zeta)(1-\xi) > 0 \\ a &= \frac{v_2 u_1 u_3}{n} \alpha_1(1-\xi) + \frac{2v_2 u_6 u_1}{n} \alpha_2(1-\zeta)(1-\xi) > 0 \end{aligned} \tag{B.3}$$

Similarly, for b , the first partial derivative was computed using Eq. (B.4). The computation of the partial derivatives contained the value for b in Eq. (B.5)

$$b = \sum_{k,i,j=1}^n v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha_j}(0, 0) \tag{B.4}$$

The procedure for a is repeated for b , the first partial derivative was computed using Eq. (B.4). The computation of the partial derivatives contained the value for b in Eq. (B.5)

$$\begin{aligned} b &= v_1 u_3 \left[\frac{(1-\xi)x_1}{n} + v_1 u_6 \frac{(1-\xi)(1-\zeta)x_6}{n} \right] \\ &\quad + v_2 u_1 \left[\frac{(1-\xi)x_3}{n} + \frac{(1-\xi)(1-\zeta)x_6}{n} \right] \\ &\quad + v_2 u_3 \left[\frac{1}{n} + v_2 u_6 \frac{(1-\xi)(1-\zeta)x_1}{n} \right] \\ &\quad - v_1 u_1 \left[\frac{(1-\xi)x_3}{n} + \frac{(1-\xi)(1-\zeta)x_6}{n} \right] > 0 \end{aligned} \tag{B.5}$$

Appendix C. Applying HPM

$$\begin{aligned} p^0 : a_0^1 &= 0 \\ p^1 : a_1^1 + \mu a_0 + \alpha_1 K e_0 + \alpha_2 K (1-\zeta)(1-\xi) f_0 a_0 - \beta (a_0 + b_0 + e_0) \\ p^2 : a_2^1 + \mu a_1 + \alpha_1 K e_1 + \alpha_2 K (1-\zeta)(1-\xi) f_0 a_1 \\ &\quad + \alpha_2 K (1-\zeta)(1-\xi) f_1 a_0 - \beta (a_1 + b_1 + e_1) \end{aligned} \tag{C.1}$$

$$\begin{aligned} p^0 : b_0^1 &= 0 \\ p^1 : b_1^1 + (\gamma + \mu) b_0 - \alpha_1 K c_0 - \alpha_2 K (1-\zeta)(1-\xi) f_0 a_0 = 0 \\ p^2 : b_2^1 + (\gamma + \mu) b_1 - \alpha_1 K c_1 - \alpha_2 K (1-\zeta)(1-\xi) f_0 a_1 \\ &\quad + \alpha_2 K (1-\zeta)(1-\xi) f_1 a_0 = 0 \end{aligned} \tag{C.2}$$

$$\begin{aligned} p^0 : c_0^1 &= 0 \\ p^1 : c_1^1 + (\varphi + \mu + \delta_1) c_0 - \gamma b_0 = 0 \\ p^2 : c_2^1 + (\varphi + \mu + \delta_1) c_1 - \gamma b_1 = 0 \end{aligned} \tag{C.3}$$

$$\begin{aligned} p^0 : d_0^1 &= 0 \\ p^1 : d_1^1 + (\tau + \mu + \delta_2) d_0 - \varphi c_0 = 0 \\ p^2 : d_2^1 + (\tau + \mu + \delta_2) d_1 - \varphi c_1 = 0 \end{aligned} \tag{C.4}$$

$$\begin{aligned} p^0 : e_0^1 &= 0 \\ p^1 : e_1^1 + \mu e_0 - \tau d_0 = 0 \\ p^2 : e_2^1 + \mu e_1 - \tau d_1 = 0 \end{aligned} \tag{C.5}$$

$$\begin{aligned} p^0 : f_0^1 &= 0 \\ p^1 : f_1^1 + (\mu + \delta_1) c_0 - (\mu + \delta_2) d_0 = 0 \\ p^2 : f_2^1 + (\mu + \delta_1) c_1 - (\mu + \delta_2) d_1 = 0 \end{aligned} \tag{C.6}$$

Solving (C.1)–(C.6) by direct integration method for p^0 using (5.1) we obtain the following:

$$\left. \begin{aligned} a_0 &= S_0 \\ b_0 &= L_0 \\ c_0 &= I_0 \\ d_0 &= Q_0 \\ e_0 &= R_0 \\ f_0 &= D_0 \end{aligned} \right\} \tag{C.7}$$

Where $S_0, L_0, I_0, Q_0, R_0,$ and D_0 are all constants initial conditions.

Substituting (C.7) into (C.1)–(C.6) and solve by direct integration method for p^1 , we obtain the following equations.

$$\left. \begin{aligned} a_1 &= (\beta (S_0 + L_0 + R_0) - \mu S_0 - \alpha_1 K I_0 - \alpha_2 K (1-\zeta)(1-\xi) D_0 S_0) t \\ b_1 &= (\alpha_1 K I_0 + \alpha_2 K (1-\zeta)(1-\xi) D_0 S_0 - (\gamma + \mu) L_0) t \\ c_1 &= (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) t \\ d_1 &= (\varphi I_0 - (\tau + \mu + \delta_2) Q_0) t \\ e_1 &= (\tau Q_0 - \mu R_0) t \\ f_1 &= ((\mu + \delta_1) I_0 + (\mu + \delta_2) Q_0) t \end{aligned} \right\} \tag{C.8}$$

Similarly, substituting (C.7) and (C.8) into (C.1)–(C.6) and solve by direct integration for p^2 , we obtain the following equations.

$$a_2 = \left(\begin{array}{l} \beta \left(\begin{array}{l} (\beta (S_0 + L_0 + R_0) - \mu S_0 - \alpha_1 K I_0) \\ -\alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 \\ + (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0) \\ - (\gamma + \mu) L_0 + (\tau Q_0 - \mu R_0) \end{array} \right) \\ -\mu (\beta (S_0 + L_0 + R_0) - \mu S_0 - \alpha_1 K I_0 - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0) - \\ \alpha_1 K (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 \left(\begin{array}{l} \beta (S_0 + L_0 + R_0) - \\ \mu S_0 - \alpha_1 K I_0 - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 \end{array} \right) \\ -\alpha_2 K (1 - \zeta) (1 - \xi) ((\mu + \delta_1) I_0 + (\mu + \delta_2) Q_0) S_0 \end{array} \right) \frac{t^2}{2} \quad (C.9)$$

$$b_2 = \left(\begin{array}{l} \alpha_2 K (1 - \zeta) (1 - \xi) D_0 \left(\begin{array}{l} \beta (S_0 + L_0 + R_0) - \\ \mu S_0 - \alpha_1 K I_0 - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 \end{array} \right) + \\ \alpha_1 K (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) \\ + \alpha_2 K (1 - \zeta) (1 - \xi) \left(\begin{array}{l} (\mu + \delta_1) I_0 \\ + (\mu + \delta_2) Q_0 \end{array} \right) S_0 \\ - (\gamma + \mu) (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 - (\gamma + \mu) L_0) \end{array} \right) \frac{t^2}{2} \quad (C.10)$$

$$c_2 = \left(\begin{array}{l} \gamma (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 - (\gamma + \mu) L_0) - \\ (\varphi + \mu + \delta_1) (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) \end{array} \right) \frac{t^2}{2} \quad (C.11)$$

$$d_2 = \left(\begin{array}{l} \varphi (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) - (\tau + \mu + \delta_2) \cdot \\ (\varphi I_0 - (\tau + \mu + \delta_2) Q_0) \end{array} \right) \frac{t^2}{2} \quad (C.12)$$

$$e_2 = (\tau (\varphi I_0 - (\tau + \mu + \delta_2) Q_0) - \mu (\tau Q_0 - \mu R_0)) \frac{t^2}{2} \quad (C.13)$$

$$f_2 = ((\mu + \delta_1) (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) + (\mu + \delta_2) (\varphi I_0 - (\tau + \mu + \delta_2) Q_0)) \frac{t^2}{2} \quad (C.14)$$

But, from (5.8) we have,

$$\left. \begin{array}{l} S = a_0 + pa_1 + p^2 a_2 + \dots \\ L = b_0 + pb_1 + p^2 b_2 + \dots \\ I = c_0 + pc_1 + p^2 c_2 + \dots \\ Q = d_0 + pd_1 + p^2 d_2 + \dots \\ R = e_0 + pe_1 + p^2 e_2 + \dots \\ D = f_0 + pf_1 + p^2 f_2 + \dots \end{array} \right\}$$

then, we let,

$$\left. \begin{array}{l} \lim_{p \rightarrow 1} S(t) = \lim_{p \rightarrow 1} (a_0 + pa_1 + p^2 a_2 + \dots) = a_0 + a_1 + a_2 + \dots \\ \lim_{p \rightarrow 1} L(t) = \lim_{p \rightarrow 1} (b_0 + pb_1 + p^2 b_2 + \dots) = b_0 + b_1 + b_2 + \dots \\ \lim_{p \rightarrow 1} I(t) = \lim_{p \rightarrow 1} (c_0 + pc_1 + p^2 c_2 + \dots) = c_0 + c_1 + c_2 + \dots \\ \lim_{p \rightarrow 1} Q(t) = \lim_{p \rightarrow 1} (d_0 + pd_1 + p^2 d_2 + \dots) = d_0 + d_1 + d_2 + \dots \\ \lim_{p \rightarrow 1} R(t) = \lim_{p \rightarrow 1} (e_0 + pe_1 + p^2 e_2 + \dots) = e_0 + e_1 + e_2 + \dots \\ \lim_{p \rightarrow 1} D(t) = \lim_{p \rightarrow 1} (f_0 + pf_1 + p^2 f_2 + \dots) = f_0 + f_1 + f_2 + \dots \end{array} \right\} \quad (C.15)$$

This implies that,

$$S(t) = \lim_{p \rightarrow 1} S(t) = \lim_{p \rightarrow 1} (a_0 + pa_1 + p^2 a_2 + \dots) = a_0 + a_1 + a_2 + \dots$$

$$\left. \begin{array}{l} S(t) = S_0 + (\beta (S_0 + L_0 + R_0) - \mu S_0 - \alpha_1 K I_0 - \\ -\alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0) t \\ \left(\begin{array}{l} \beta \left(\begin{array}{l} (\beta (S_0 + L_0 + R_0) - \mu S_0 - \alpha_1 K I_0) \\ -\alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 \\ + (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0) \\ - (\gamma + \mu) L_0 + (\tau Q_0 - \mu R_0) \end{array} \right) \\ -\mu (\beta (S_0 + L_0 + R_0) - \mu S_0 - \alpha_1 K I_0 - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0) - \\ \alpha_1 K (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 \left(\begin{array}{l} \beta (S_0 + L_0 + R_0) - \\ \mu S_0 - \alpha_1 K I_0 - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 \end{array} \right) \\ -\alpha_2 K (1 - \zeta) (1 - \xi) ((\mu + \delta_1) I_0 + (\mu + \delta_2) Q_0) S_0 \end{array} \right) \frac{t^2}{2} \end{array} \right\} \quad (C.16)$$

$$L(t) = \lim_{p \rightarrow 1} L(t) = \lim_{p \rightarrow 1} (b_0 + pb_1 + p^2 b_2 + \dots) = b_0 + b_1 + b_2 + \dots$$

$$\left. \begin{array}{l} L(t) = L_0 + (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 - \\ - (\gamma + \mu) L_0) t + \\ \left(\begin{array}{l} \alpha_2 K (1 - \zeta) (1 - \xi) D_0 \left(\begin{array}{l} \beta (S_0 + L_0 + R_0) - \\ \mu S_0 - \alpha_1 K I_0 - \\ \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 \end{array} \right) + \\ \alpha_1 K (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) \\ + \alpha_2 K (1 - \zeta) (1 - \xi) \left(\begin{array}{l} (\mu + \delta_1) I_0 \\ + (\mu + \delta_2) Q_0 \end{array} \right) S_0 \\ - (\gamma + \mu) (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 - (\gamma + \mu) L_0) \end{array} \right) \frac{t^2}{2} \end{array} \right\} \quad (C.17)$$

$$I(t) = \lim_{p \rightarrow 1} I(t) = \lim_{p \rightarrow 1} (c_0 + pc_1 + p^2 c_2 + \dots) = c_0 + c_1 + c_2 + \dots$$

$$\left. \begin{array}{l} I(t) = I_0 + (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) t + \\ \left(\begin{array}{l} \gamma (\alpha_1 K I_0 + \alpha_2 K (1 - \zeta) (1 - \xi) D_0 S_0 - (\gamma + \mu) L_0) - \\ (\varphi + \mu + \delta_1) (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) \end{array} \right) \frac{t^2}{2} \end{array} \right\} \quad (C.18)$$

$$Q(t) = \lim_{p \rightarrow 1} Q(t) = \lim_{p \rightarrow 1} (d_0 + pd_1 + p^2 d_2 + \dots) = d_0 + d_1 + d_2 + \dots$$

$$\left. \begin{array}{l} Q(t) = Q_0 + (\varphi I_0 - (\tau + \mu + \delta_2) Q_0) t + \\ \left(\begin{array}{l} \varphi (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) - (\tau + \mu + \delta_2) \cdot \\ (\varphi I_0 - (\tau + \mu + \delta_2) Q_0) \end{array} \right) \frac{t^2}{2} \end{array} \right\} \quad (C.19)$$

$$R(t) = \lim_{p \rightarrow 1} R(t) = \lim_{p \rightarrow 1} (e_0 + pe_1 + p^2 e_2 + \dots) = e_0 + e_1 + e_2 + \dots$$

$$\left. \begin{array}{l} R(t) = R_0 + (\tau Q_0 - \mu R_0) t + \\ (\tau (\varphi I_0 - (\tau + \mu + \delta_2) Q_0) - \mu (\tau Q_0 - \mu R_0)) \frac{t^2}{2} \end{array} \right\} \quad (C.20)$$

$$D(t) = \lim_{p \rightarrow 1} D(t) = \lim_{p \rightarrow 1} (f_0 + pf_1 + p^2 f_2 + \dots) = f_0 + f_1 + f_2 + \dots$$

$$\left. \begin{array}{l} D(t) = D_0 + ((\mu + \delta_1) I_0 + (\mu + \delta_2) Q_0) t + \\ ((\mu + \delta_1) (\gamma L_0 - (\varphi + \mu + \delta_1) I_0) + \\ (\mu + \delta_2) (\varphi I_0 - (\tau + \mu + \delta_2) Q_0)) \frac{t^2}{2} \end{array} \right\} \quad (C.21)$$

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