e-ISSN: 2564-7954 CUSJE 20(02): 129-145 (2023) Research Article

Çankaya University

Journal of Science and Engineering

https://dergipark.org.tr/cankujse

CUJSE

On the Dynamics of Ebola Virus Disease (EVD) with the Impact of Vaccination and Isolation on the Containment of its Spread: A Mathematical Modelling Approach

Oludapo Omotola Olubanwo[1], Deborah Oluwatobi Daniel[2*], Timothy Ayobami Adegboye[1], Abosede Nimotali Adeniran[1]

[1] Department of Mathematical Science, Olabisi Onabanjo University, Nigeria
[2] Department of Mathematics and Computer Science, Southwestern University, Nigeria

Keywords

Ebola Virus Disease, Transmission Dynamics, Vaccination, Isolation, Infectious Disease, Mathematical Modelling.

Abstract

Ebola is a highly contagious and fatal viral disease that has sparked widespread panic and devastation and thus affected global health, the economy, and social dynamics. Hence, a model is formulated to examine the impact of isolation and vaccination on curbing the transmission dynamics of Ebola Virus Disease (EVD). The model's epidemiological viability in a given region was established. Numerical simulations were conducted using MATLAB to examine the effect of vaccination and isolation on curtailing the spread of the Ebola virus disease. The impact of the parameters used in the model on the basic reproduction number and the estimation of the sensitivity of the parameters were also carried out. It is observed that if the rate of symptomatic infected individuals being isolated and vaccinated is high enough, this would reduce the infection rate. Hence, the isolation of infected individuals and efficacious vaccination with a zero-wane-off vaccine will help a great deal in curtailing the spread of the Ebola virus disease.

1. Introduction

Ebola virus has the potential to induce a viral hemorrhagic fever, commonly referred to as Ebola virus disease (EVD) or Ebola hemorrhagic fever (EHF), in humans and other primates. Both outbreaks can be attributed to the same source, namely the Ebola virus. The Ebola virus disease, a highly uncommon yet fatal condition, is attributed to an infection caused by any of the various species of the Ebola virus. The onset of symptoms typically occurs within a span of two to three weeks following the initial infection, whereby individuals commonly manifest indications such as fever, sore throat, muscular discomfort, and headaches. Subsequently, a cutaneous eruption frequently manifests, followed by symptoms of queasiness, gastrointestinal distress, and a progressive decline in hepatic and renal performance. At this stage, individuals can experience both external and internal bleeding. The illness exhibits a significantly elevated fatality rate, ranging from 25 to 90 percent, with an approximate mean of 50 percent, indicating a substantial likelihood of death among affected individuals. This medical condition, commonly associated with hypotension caused by fluid depletion, may present itself between six to sixteen days following the onset of symptoms. Various bodily fluids, including blood, saliva, sweat, faeces, vomit, breast milk, and semen, can serve as carriers of infection. Additionally, inanimate objects such as needles and syringes can become contaminated, as well as fruits and vegetables [1-5]; Research has demonstrated there is a potential correlation between breastmilk consumption and a potential reduction in the infant's immune system. Their actions have the potential to facilitate the transmission of the contagious pathogen. Numerous studies have provided evidence indicating that the viral presence can persist in seminal fluid for approximately 40 days after the initial infection [6]. According to [7] and [8], it is strongly recommended that individuals who have survived Ebola refrain from participating in breastfeeding and engaging in sexual activity without the use

* Corresponding Author: oludeboradaniel@gmail.com
Received: July 14, 2023, Accepted: October 11, 2023

of protection. This is because their actions have the potential to transmit the pathogen to others. According to a study, it has been suggested that Ebola can be transmitted by various arthropods, including bats and rats [9].

Numerous disease pandemics have inflicted severe devastation upon their respective regions. Comprehending the patterns of disease transmission and the methods employed for its control is imperative for the effective management and mitigation of epidemics. The utilisation of mathematical modelling has emerged as a valuable instrument in comprehending intricate disease mechanisms, as evidenced by the works of researchers [10-13]. Scientists can conduct experiments on alternative scenarios and treatments, potentially exerting an influence on public health policy.

The study of mathematical modelling in relation to Ebola Virus Disease (EVD) has attracted significant interest in recent years [14-20]. These studies have provided insights into the epidemiology of the disease [14,17,19-20], its patterns of transmission [14,15,18-20], and potential therapeutic interventions [15,16,17,19].

Several researchers have employed the SIR and SEIR models [19-21] in an attempt to predict the transmission dynamics of Ebola. The research paper proposes the use of vaccination as a preventive measure against the disease and employs the SEIR model to assess the transmission dynamics and control strategies of the illness. Various vaccination rates are employed to assess the potential extent of disease transmission. After determining the minimum vaccination threshold necessary to impede the transmission of the illness, researchers may proceed to examine the impact of the vaccine on the proportion of individuals susceptible to the disease, capable of transmitting it to others, exposed to the disease, and successfully treated [22] demonstrated the application of mathematical modelling in evaluating the effects of fundamental public health interventions on disease transmission and gaining an understanding of the potential occurrence of a severe epidemic caused by Ebola virus disease (EVD). During the discourse on the dissemination and management of viral hemorrhagic fevers within the framework of the greatly diverse economic landscape of African nations, the scholars also deliberated on the imperative requirement to gather comprehensive epidemiological data in a timely manner throughout the progression of an ongoing epidemic. Additionally, they emphasised the necessity of conducting supplementary investigations to assess the efficacy of interventions, as well as the development of extensive modelling studies on a large scale. During the meeting, the aforementioned issues were discussed.

The deterministic SEIR model of the 2014 Ebola epidemic in West Africa was understudied [21]. The proposed model elucidates the intricate interplay between populations that are susceptible to a particular disease and those that have already been infected. This model takes into consideration the impact of hospitalisation on the spread of the disease as well as the transmission of the illness through contact with individuals who have recently passed away. The model's parameters were refined by utilising current estimates of infected and deceased patients in each nation, with data sourced from the World Health Organisation (WHO). The model was subsequently evaluated using the established parameters. Proposed indicators were put forth to ascertain the nations that are in dire need of additional funding to effectively curb the proliferation of the disease. The factors under consideration encompass the fundamental reproductive number in relation to hospitalisation and appropriate burial rates, as well as local and global sensitivity assessments of the affected population.

In [23], a study was carried out that examined and compared two distinct mathematical models employed for elucidating the transmission dynamics of the Ebola virus in West Africa. The researchers conducted a comparative analysis of the two models in order to enhance their accuracy in predicting and mitigating the transmission of the infection. Specifically, the researchers examined the scenario in which the outcomes derived from both models exhibit congruence. [19] used the SEIR (Susceptible-Exposed-Infectious-Recovered) model to examine Ebola transmission dynamics. They also suggested ways to control this infectious sickness. The research was focused on susceptible, infected, and recovered people. The researchers examined many methods for managing the 2014 Ebola pandemic using WHO statistics. The steps were taken to reduce viral transmission and test vaccination effectiveness.

It is observed that most existing Ebola virus disease models exclude the combined use of vaccination and isolation as measures against Ebola. Furthermore, existing models that incorporate vaccination as a control measure fail to adequately emphasise the declining effectiveness of vaccination over time. This study is focused on enhancing comprehension of the impacts of these measures in order to acquire valuable recommendations for preventive and therapeutic measures against the Ebola virus disease. Hence, we examine the transmission

dynamics of Ebola Virus Disease (EVD) with prevention and control measures as well as the possibility of the vaccination waning off.

2. Model Formulation

Based on the following assumptions, we build a deterministic compartmental mathematical model for EVD that includes vaccination, isolation, and therapeutic treatment as control strategies:

(1) There is continuous disease-induced immunity.

(2) We suppose a crucial dynamic exists. In fact, several Ebola epidemics have lasted longer than two years (for example, the Western Africa outbreak). As a result, throughout this rather lengthy period, there may be new births or an influx of susceptible persons from other/neighboring regions, as well as natural deaths.

(3) Homogeneous mixing of members of the population being studied.

The model is composed of eight compartments namely

$S(t)$ – Susceptible persons at a time.

$E(t)$ – Exposed persons at a time.

$A(t)$ – Asymptomatic infectious persons at a time i.e people who are infected but not infectious yet.

$I(t)$ – Symptomatic infectious persons at a time i.e individuals who are aware.

$Q(t)$ – Quarantined or isolated infectious persons at a time.

$V(t)$ – Vaccinated persons at a time.

$D(t)$ - Death

R(t) – Recovered persons at a time.

Figure 1 carefully shows the schematic illustration of the model. The susceptible class is increased by the constant rate Λ of births or immigration, rate of vaccination ξ and lost immunity of the recovered individuals, which is then decreases due to natural mortality rates μ, interactions with infectious people $\Omega(t)$, and those going for vaccination $v\phi$. Susceptible individuals transfer to compartment E after being exposed to infection at a rate $\Omega(t)$. They either die naturally or become asymptomatic infected (unaware infectious) at the rate γ. The rate at which the asymptomatic infected becomes symptomatic infected (aware infectious) is ε. The proportion of A and I are quarantined at a rate η and α respectively. After treatment they are recovered at the rate of ρ. The induced death rate for the symptomatic infected and quarantined person is δ. The vaccinated are populated by the susceptible person being vaccinated at the rate $v\phi$ and reduce by the rate of wane off of vaccination.

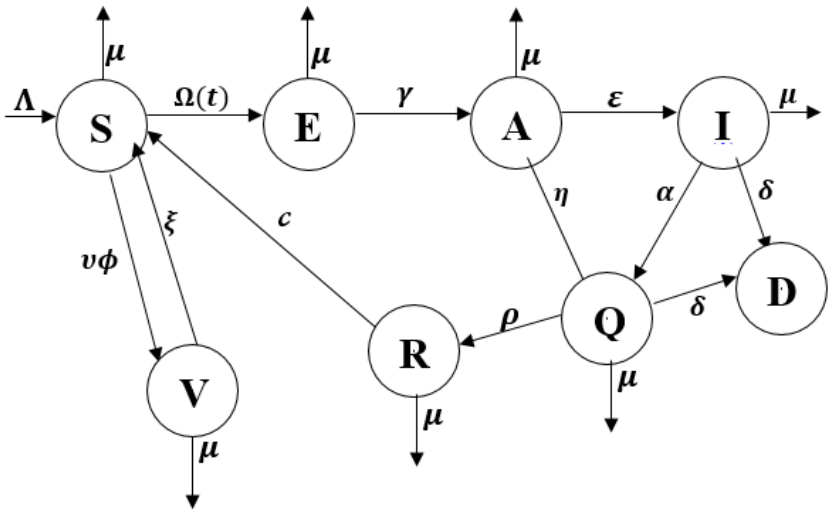


Figure 1. Schematic Illustration of the Model

2.1. Model Formulation

$$\frac{dS}{dt} = \Lambda - \Omega(t) - v\phi S + \xi V + cR - \mu S \quad (1)$$
$$\frac{dE}{dt} = \Omega(t) - (\gamma + \mu)E \quad (2)$$
$$\frac{dA}{dt} = \gamma E - \varepsilon A - \eta A - \mu A \quad (3)$$
$$\frac{dI}{dt} = \varepsilon A - (\mu + \alpha + \delta)I \quad (4)$$
$$\frac{dQ}{dt} = \eta A + \alpha I - (\mu + \rho + \delta)Q \quad (5)$$
$$\frac{dV}{dt} = v\phi S - \xi V - \mu V \quad (6)$$
$$\frac{dR}{dt} = \rho Q - (c + \mu)R \quad (7)$$
$$\frac{dD}{dt} = \delta I + \delta Q \quad (8)$$
$$\Omega(t) = c_1 SE + c_2 SA + c_3 SI$$

where

Λ = birth rate unto the susceptible
ϕ= proportion of susceptible individual vaccinated
v = vaccination rate
η = rate of recruitment into the quarantine class
c_1 = contact rate between susceptible and exposed
c_2 = contact rate between susceptible and asymptomatic infectious individuals
c_3 = contact rate between susceptible and symptomatic infected
ξ = rate of vaccine wane
c = loss of immunity
ρ= recovery rate of the quarantined
μ = natural death rate
α = rate of infected isolated
δ = disease induced death rate
γ = incubation rate of the disease
ε = rate at which symptomatic infected become symptomatic

2.2. Disease-Free Equilibrium E_0

Since no disease is in the system, there is no illness and no need for recovery.
Hence,

$$\boldsymbol{E_0} = \left(\frac{\Lambda}{V\phi+\mu}, 0,0,0,0,\frac{v\phi S_0}{(\xi+\mu)}, 0,0\right) \quad (9)$$

2.3. Basic Reproduction Number R_0

The basic reproduction number predicts how long-term disease stays in the body. It controls how quickly a disease spends in the system. Using next generation method, the basic reproduction number denoted by R_0 is obtained by taking the largest (dominant) eigenvalue of

$$\rho(R_0) = FV^{-1} \quad (10)$$

The reproduction number $R_0 = \rho(FV^{-1})$

$$R_0 = R_E + R_A + R_I \quad (11)$$
$$R_0 = \frac{c_1 S_0}{\gamma+\mu} + \frac{c_2 S_0 \gamma}{(\gamma+\mu)(\eta+\varepsilon+\mu)} + \frac{c_3 S_0 \varepsilon\gamma}{(\eta+\varepsilon+\mu)(\alpha+\delta+\mu)(\gamma+\mu)} \quad (12)$$

Where

R_E is the contribution of the exposed individuals

R_A is the contribution of asymptomatic infectious individuals
R_I is the contribution of the symptomatic infectious individuals

2.4. Endemic Equilibrium

In epidemiology, the term "endemic" is used to describe the state of an illness within a particular population or geographical area. Specifically, it refers to the continuous presence of the virus at a baseline level, without the introduction of additional infections. To find the endemic equilibrium, equate equations (1) – (7) to zero. Hence the endemic equilibrium is

$$S^* = \frac{\Lambda+\beta_4 E}{(C_1+\beta_1+\beta_2)E+\beta_3} \quad (13)$$

$$A^* = \frac{\gamma E}{(\varepsilon+\eta+\mu)} \quad (14)$$

$$I^* = \frac{\varepsilon\gamma E}{(\alpha+\delta+\mu)(\varepsilon+\eta+\mu)} \quad (15)$$

$$Q^* = \frac{\gamma(\eta(\alpha+\delta+\mu)\alpha\varepsilon)E}{(\varepsilon+\eta+\mu)(\mu+\rho+\delta)} \quad (16)$$

$$V^* = \frac{V\phi}{(\xi+\mu)}\left(\frac{\Lambda+\beta_4 E}{(C_1+\beta_1+\beta_2)E\beta_3}\right) \quad (17)$$

$$R^* = \frac{\gamma\rho(\eta(\alpha+\delta+\mu)+\alpha E)}{(\eta+\rho+\delta)(C+\mu)(\varepsilon+\eta+\mu)} \quad (18)$$

2.5. Stability Analysis of the Model

This section covers the stability analysis of the model ranging from local stability to global stability.

2.5.1. Local Stability of the Disease-Free Equilibrium

Theorem 1: The disease-free equilibrium (DFE) of the system is locally asymptomatically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

To get the local stability of E_0, the following variational matrix is solved corresponding to the equilibrium E_0
The Jacobian Matrix at DFE is given as

$$J(S,E,A,I,Q,V,R) = \begin{pmatrix} -(v\phi+\mu) & -C_1S_0 & -C_2S_0 & C_3S_0 & 0 & \xi & C \\ 0 & -(\gamma+\mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\mu+\xi+\eta) & 0 & 0 & 0 & 0 \\ 0 & \xi & -(\alpha+\delta+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta & \alpha & -(\alpha+\rho+\delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\xi+\mu) & 0 \end{pmatrix} \quad (19)$$

The eigenvalues $-(c+\mu), -(V\phi+\mu), -(\gamma+\mu), and -(\mu+\rho+\delta)$.

The remaining eigenvalues are obtained from the following matrix

$$J(S,E,A,I,Q,V,R) = \begin{bmatrix} -(\mu+\xi+\eta) & 0 \\ \eta & (\alpha+\delta+\mu) \end{bmatrix} \quad (20)$$

$$\lambda^2 + (\mu+\xi+\eta+\alpha+\delta+\mu)\lambda + (\mu+\xi+\eta)(\alpha+\delta+\mu) = 0$$

$$\lambda_{1,2} = -\frac{\mu+\xi+\eta+\alpha+\delta+\mu \pm \sqrt{(\mu+\xi+\eta+\alpha+\delta+\mu)^2 - 4(\mu+\xi+\eta)(\alpha+\delta+\mu)}}{2}$$

$$\lambda^2 + a\lambda + b = 0 \quad (21)$$

If this is satisfied, it implies that all the roots are negative hence, the DFE is locally asymptomatically stable but otherwise unstable.

2.5.2. Global Stability of the Disease-Free Equilibrium

Theorem 2: The Ebola model at DFE is globally asymptomatic stable if $R_0 \leq 1$

Proof

The proof is based on using the Lyapunov function. Using the idea of Onitilo and Daniel (2022), the Lyapunov function needs to satisfy the model of the infected class $E = A = I = Q = 0$ to be asymptomatically stable

$$\text{Let } L = b_1E + b_2A + b_3I + b_4Q \tag{22}$$

The derivative of equation (22) is

$$\frac{\partial L}{\partial t} = b_1\frac{dE}{dt} + b_2\frac{dA}{dt} + b_3\frac{dI}{dt}b_4\frac{dQ}{dt} \tag{23}$$

$$\frac{dL}{dt} \leq b_1\big((c_1SE + c_2SA + c_3SI) - (\gamma + \mu)E\big) + (b_2\varepsilon - b_2(\varepsilon + \eta + \mu)A + (b_4\alpha - b_3(\alpha + \delta + \mu)I - b_4(\mu + \rho + \delta)Q \tag{24}$$

At DFE $\mathcal{E}_0, E_0, A_0, I_0, Q_0 = 0$

$$\frac{dL}{dt} \leq (b_2\gamma - b_1(\gamma + \mu)E + \big(b_3\varepsilon - b_2(\varepsilon + \eta + \mu)\big)A + b_4\eta A + \big(b_4\alpha - b_3(\alpha + \delta + \mu)\big)I - b_4(\mu + \rho + \delta)Q \tag{25}$$

equating the coefficient of E in equation (25) and applying the multiplication property of equality i.e.

If $pr = qs\ the\ p = q\ as\ well\ as\ r = s$

$$b_2\gamma = b_1(\gamma + \mu)$$

$$b_2 = \gamma + \mu \quad \text{and } b_1 = \gamma \tag{26}$$

Also equating the coefficient of I

$$b_4\alpha = b_3(\alpha + \delta + \mu)$$

$$b_4 = \alpha + \delta + \mu \qquad and \qquad b_3 = \alpha \tag{27}$$

substituting for $b_1, b_2, b_3$and b_4 in equation (25) and further simplification gives

$$\frac{dL}{dt} \leq \left(-\left(\frac{c_1S_0}{(\gamma+\mu)} + \frac{c_2S_0}{(\gamma+\mu)(\eta+\varepsilon+\mu)} + \frac{c_3\varepsilon\gamma S_0}{(\eta+\varepsilon+\mu)(\alpha+\delta+\mu)(\gamma+\mu)}\right) + \alpha(\varepsilon+\eta) + (\delta+\mu)\eta - (\gamma+\mu)(\varepsilon+\eta+\mu)(R_0-1)\right)A \tag{28}$$

Therefore, $\frac{dL}{dt} = 0$ if and only if $A = 0$ and $\frac{dL}{at} < 0$ if $R_0 < 1$. Hence the largest compact invariant set in $\Omega = \left\{S, E, A, I, Q, R \in \mathbb{R}: \frac{dL}{dt}\right\}$ Is the singleton set $\{\mathcal{E}_0\}$. from Lasalle's invariant principle. It can be concluded that $R_0 < 1$ implies $\{\mathcal{E}_0\}$ is asymptomatically stable in Ω.

2.6. Sensitivity Analysis

To determine how to most effectively lower illness-related mortality and morbidity, epidemiologists must understand the relative significance of the many elements involved for the transmission and prevalence of the disease. Sensitivity analysis is performed to investigate how sensitive parameter changes are to the spread and incidence of the illness. As a parameter value for changes, sensitivity indices are used to quantify the relative change in a state variable. The ratio of the relative change in the parameter determines the normalised forward sensitivity index of a variable to the parameter. The sensitivity index may be constructed using partial derivatives in the case when the variable is a differential function of the parameter as shown below: The normalised forward sensitivity index of a variable that depends differently on a parameter ξ is defined as

$$\pi_\xi^\eta = \frac{\partial\eta}{\partial\xi} \times \frac{\xi}{\eta}$$

Where ξ represent all the basic parameter. The sensitivity of the parameters is computed using maple and presented in Table 1.

3. Results and Discussion

A numerical simulation of the Ebola virus disease is analysed using the baseline values given in Table 1. The numerical simulations were done over a period of 150 days using MATLAB, and the results are shown in Figures 2–10 to evaluate the efficacy of vaccination and the isolation of infectious individuals. Also, the effect of waning

vaccination on the population is examined. The sensitivity index of the parameters used in the model was calculated and presented in Table 1 to check the most contributing parameters to the basic reproduction number.

Table 1. Model parameters and values used in the simulation.

Parameters	Values	Source	Sensitivity Index
Λ	136	[24]	1
ε	0.1	[25]	0.01839
c_1	2.568e-7	Assumed	0.904
c_2	1.417e-7	[26]	0.09593
c_3	1.1102e-9	Assumed	0.00007368
ξ	0 – 1	Control parameter	-
δ	0.8	[25]	-0.0000578
η	0.4	[25]	-0.0738
μ	0.02	[25]	-0.17036
v	0 – 1	Control parameter	-0.5
α	0 – 1	Control parameter	0.00001445
ϕ	0.1	[25]	-0.5
ρ	1/15.88	[25]	-
c	0.0314862	[25]	-
γ	0.1	[25]	-0.73733

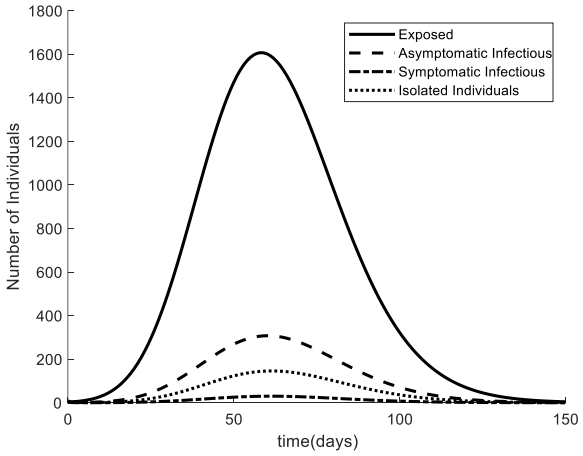


Figure 2. Simulation of Exposed, Asymptomatic Infectious, Symptomatic Infectious and Isolated Individuals in response to the Ebola Virus Diseases

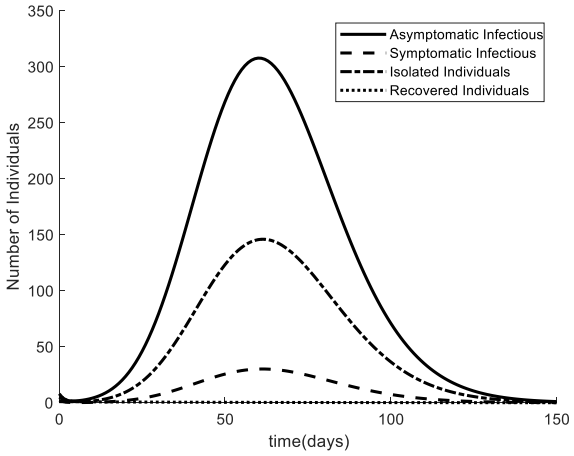


Figure 3. Simulation of Asymptomatic Infectious, Symptomatic Infectious, Isolated Individuals and Recovered Individuals in Response to the Ebola Virus Diseases

Figure 2 illustrates the simulation plot of Exposed, Asymptomatic Infectious, Symptomatic infectious, and Isolated Individuals in response to the Ebola Virus disease (EVB) in the absence of vaccination. It is observed that the number of individuals exposed is much greater than the number of asymptomatic, symptomatic, and isolated individuals. The peak is achieved in 60 days, and after that, it is seen to decline. This shows that there would be a high number of people exposed if vaccination was not in place; hence, the contact rate would sooner increase.

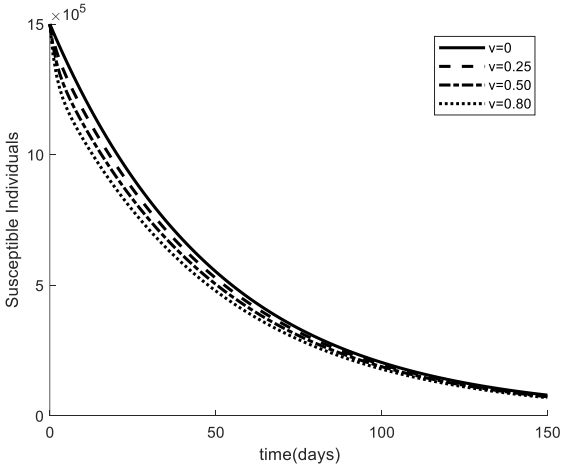
Figure 3 shows the simulation plot of the Asymptomatic Infectious, Symptomatic Infectious, Isolated individuals, and Recovered Individuals in response to the Ebola Virus disease in the absence of vaccination. It is revealed that the number of asymptomatic infectious individuals is much higher than the number of symptomatic infectious individuals and isolated individuals. Recovered individuals are seen to be the smallest number in that compartment. Hence, sooner or later, the human population will go extinct.

Figures 2 and 3 show the need to curtail the spread of Ebola Virus Disease (EVB); hence, the effectiveness of vaccination and isolation of symptomatic infectious individuals is examined. Also, the sensitivity of the parameter of the basic reproduction R_0 is assessed to know the impact of the parameters on R_0.

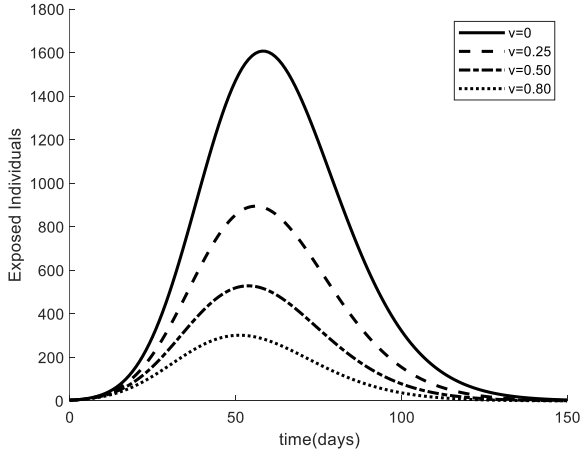
3.1. Effect of Vaccination

Figure 4a shows the simulation plot showing the impact of vaccination on susceptible individuals at the vaccination rate $v = 0, 0.25, 0.5, 0.8$. It is observed that as the rate of vaccination increases, the number of individuals leaving the compartment for vaccination increases. Hence, there is a reduction in the number of individuals in the compartment.

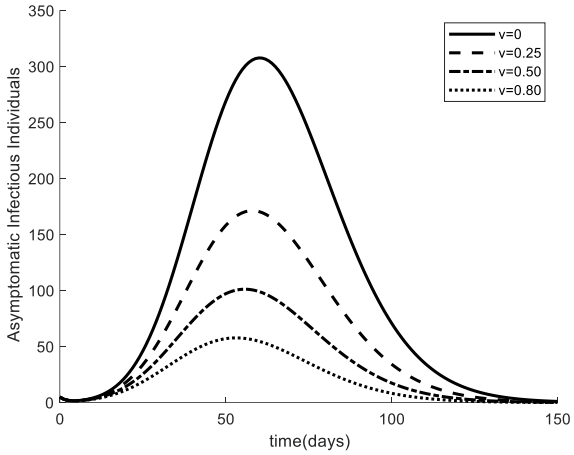
Figures 4b, 4c, 4d, 4e, and 4g display the simulation plot showing the impact of vaccination on exposed, asymptomatic infectious, symptomatic infectious, isolated, and recovered individuals at the vaccination rate $v = 0, 0.25, 0.5, 0.8$. It is seen that the number of individuals in these compartments reduces, which is a result of an increase in the rate at which individuals are vaccinated. This shows that if individuals are vaccinated, the contact rate between susceptible and exposed, susceptible and asymptomatic infectious individuals, and susceptible and symptomatic infectious individuals will drastically reduce, i.e., the infection rates will reduce. Since the infection is reduced, the number of individuals in the infection compartment reduces, and hence, the number of recoveries also reduces since there are fewer individuals infected, they are hospitalised or isolated, and then they recover after treatment. However, an increase in the rate of vaccination increases the number of vaccinated individuals, as displayed in Figure 4f. Figure 4f illustrates the impact of vaccination on the vaccinated individuals at the vaccination rate. Hence, Figure 4 demonstrates the effectiveness of vaccination in curbing the spread of EVB.



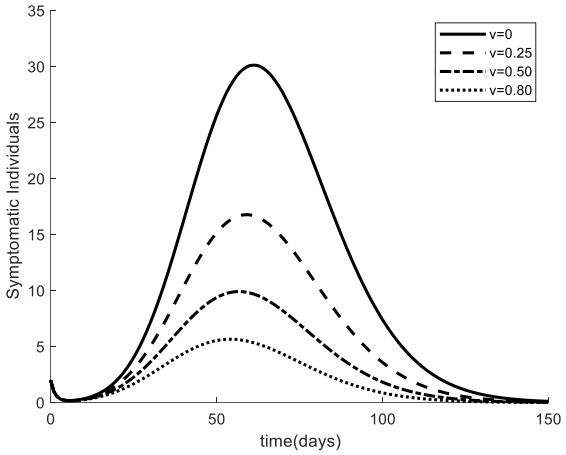
(a)



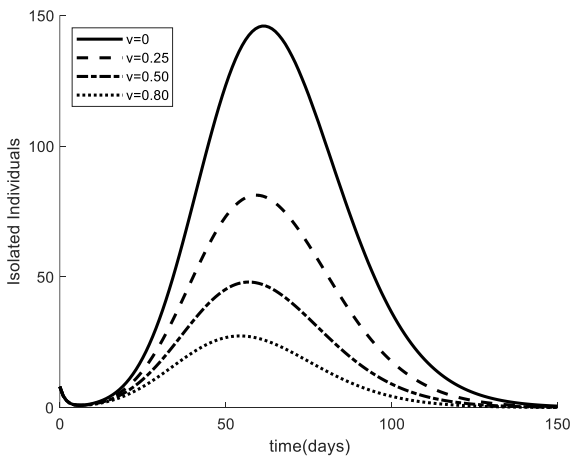
(b)



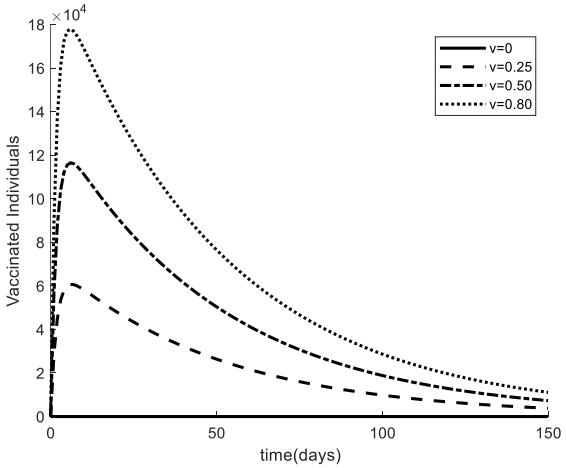
(c)



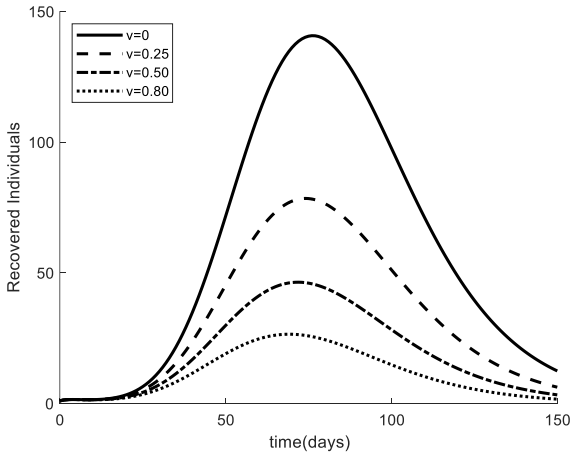
(d)



(e)



(f)



(g)

Figure 4. Simulation showing the impact of vaccination on (a) susceptible individuals (b) exposed individuals (c) asymptomatic infectious individuals (d) symptomatic infectious (e) isolated individuals (f) vaccinated individuals (g) recovered individuals.

3.2. Effect of Isolation on the Spread of the Disease

Figure 5a depicts the Simulation of the effects of isolation on the symptomatic infectious individuals at the isolation rate $\alpha = 0, 0.25, 0.5, 0.8$. It is observed that with an increase in the rate at which symptomatic infectious individuals are isolated, the number of symptomatic individuals in the compartment reduces drastically.

However, there is an increase in the number of individuals isolated and recovered, as observed in Figures 5b and 5c, respectively. Figure 5b shows the simulation effects of isolation on the isolated individuals, while Figure 5c shows the simulation effects of isolation on the recovered individuals.

In essence, increasing the isolation rate makes individuals who are symptomatically infected more likely to be isolated and treated appropriately. After their duration of treatment, they move to the recovered compartment, thereby populating the number of recovered individuals in the compartment. This is also seen as an effective way of curbing the spread of EVB.

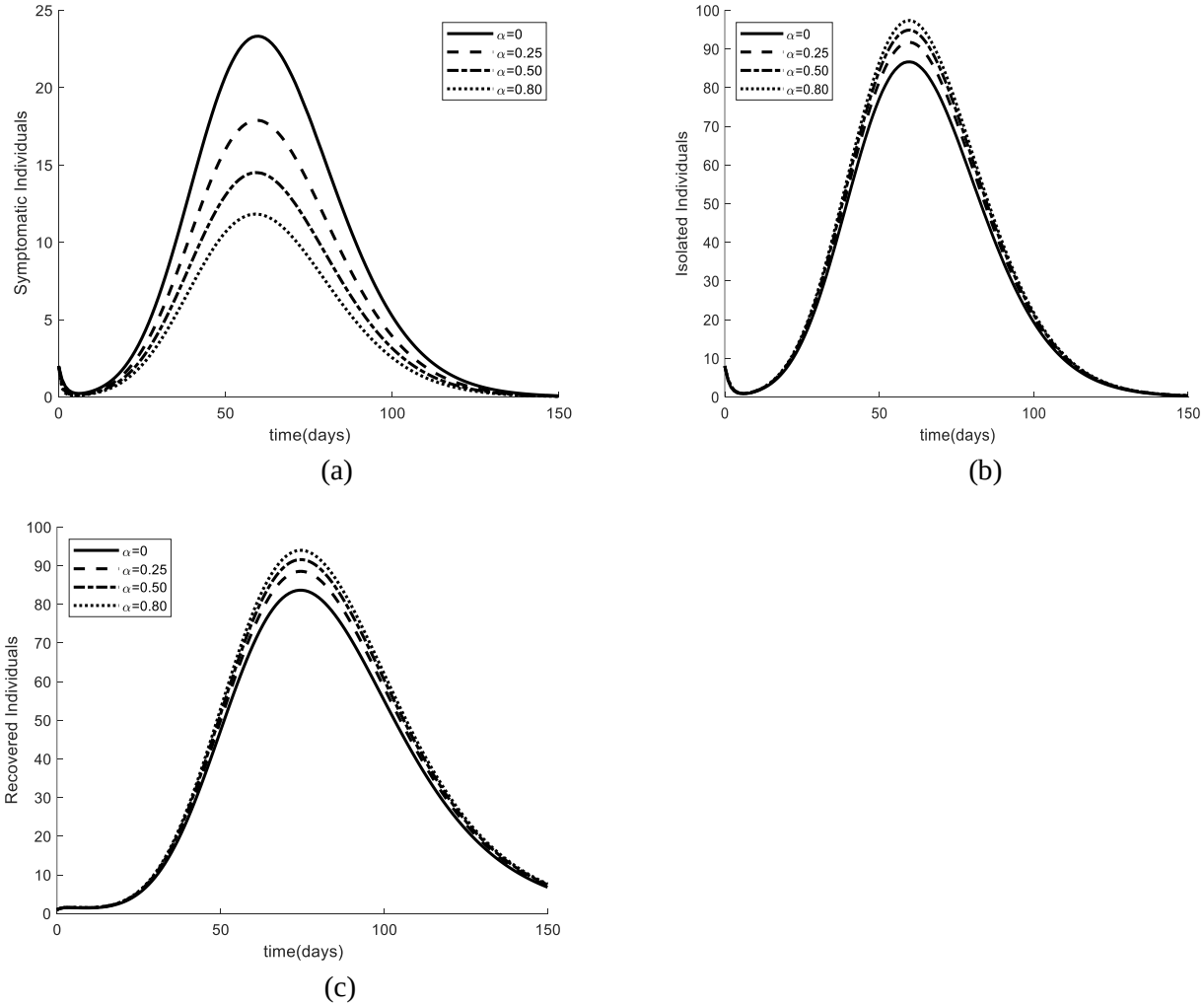


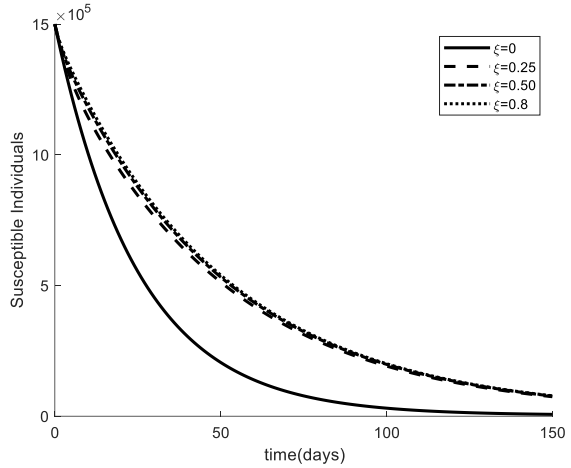
Figure 5. Simulation of the effects of isolation on the (a) symptomatic infectious (b) isolated (c) recovered individuals.

3.3. Effect of Waning of Vaccination

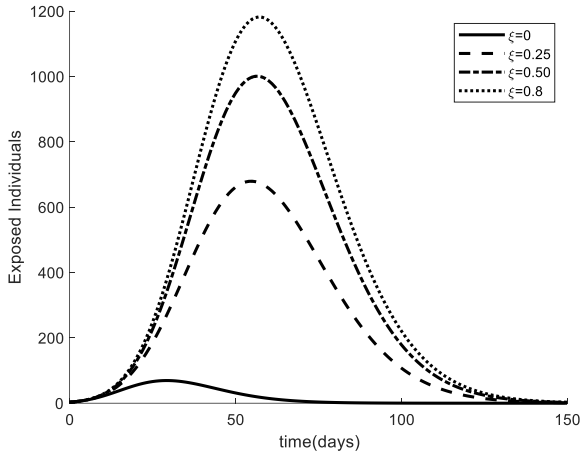
The simulation of the effects of the waning vaccine on susceptible individuals, exposed individuals, asymptomatic infectious individuals, symptomatic infectious individuals, isolated individuals, and recovered individuals is displayed respectively in Figures 6a, 6b, 6c, 6d, 6e, and 6g at the waning rate $\xi = 0, 0.25, 0.5, 0.8$. It is observed that the increase in the rate at which the vaccine is administered increases the number of people in this compartment. The implication is that the infection rate continues to increase due to an increase in contact

between the susceptible and the exposed, susceptible and the asymptomatic infected, and susceptible and symptomatic infected individuals, which poses a great threat to human health.

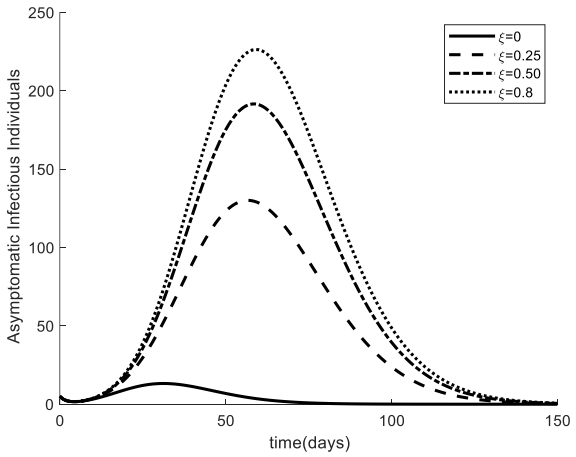
Figure 6f shows the simulation of the effects of the wane vaccine on the vaccinated individuals at the waning rate $\xi = 0, 0.25, 0.5, 0.8$. It is observed that an increase in the rate of vaccination reduces the number of individuals vaccinated. When the vaccines wane, they move back into the susceptible population and can get infected in contact with exposed individuals, asymptomatic individuals, or symptomatic individuals. Although the disease is managed as long as individuals who get infected are isolated and treated, the cycle keeps continuing, which can be avoided if the vaccine provided does not wear off.



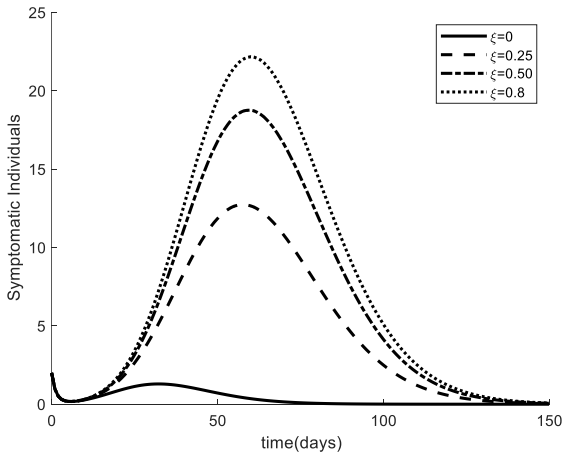
(a)



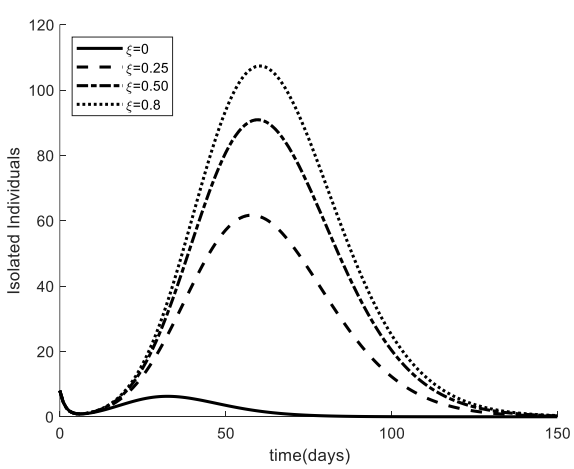
(b)



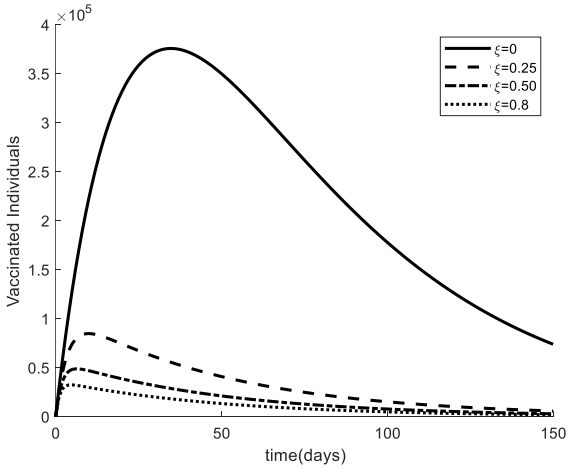
(c)



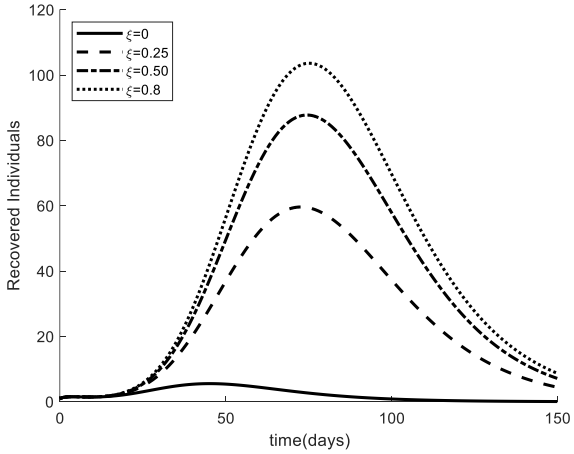
(d)



(e)



(f)



(g)

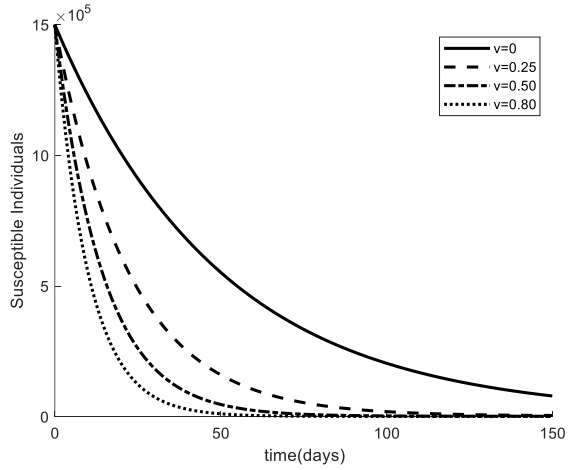
Figure 6. Simulation of the effects of the wane vaccine on the (a) susceptible (b) exposed (c) asymptomatic infectious (d) symptomatic infectious (e) isolated (f) vaccinated (g) recovered individuals

3.4. Effectiveness of Vaccination in the Absence of Wane Vaccine

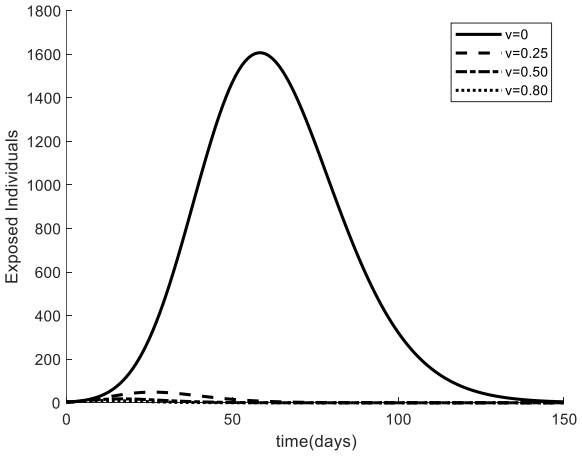
Figures 7a, 7b, 7c, 7d, 7e, 7f, and 7g, respectively, show the effectiveness of vaccination in the absence of waning for susceptible individuals, exposed individuals, asymptomatic individuals, symptomatic individuals, isolated individuals, vaccinated individuals, and recovered individuals at the vaccination rate $v = 0, 0.25, 0.5, 0.8$. It is observed in Figures 7a, 7b, 7c, 7d, 7e, and 7g that in the absence of the wane vaccine, the number of individuals in these compartments is reduced. In essence, as the number of vaccinations increases in the absence of wane vaccines, the number of individuals in the susceptible class, exposed class, asymptomatic class, symptomatic class, isolated class, and recovered class reduces greatly. The effectiveness is seen in its reduction as compared to when the vaccine is not introduced to the system.

Figure 7f displays the simulation of the effectiveness of vaccination in the absence of waning for the isolated individuals. It is seen that the number of individuals vaccinated keeps increasing. When there are no vaccines introduced into the system, no one is vaccinated. As vaccines are introduced into the system, the number of vaccinated people increases, and as the rate of vaccination increases, the number of vaccinated individuals is also seen to increase.

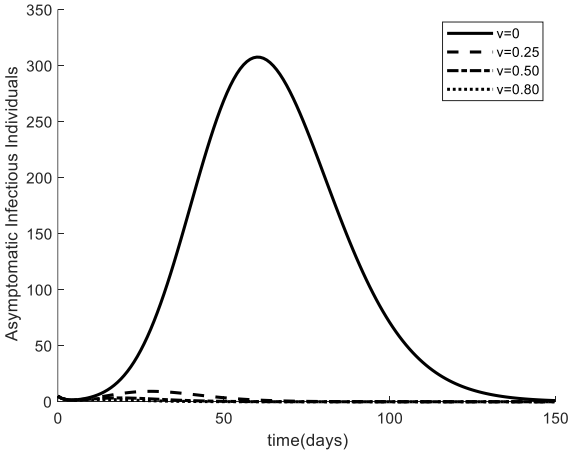
Hence, in order to curb the spread of EVB, the vaccine provided should be the one that does not wane.


×10^5
Susceptible Individuals
time(days)
v=0
v=0.25
v=0.50
v=0.80

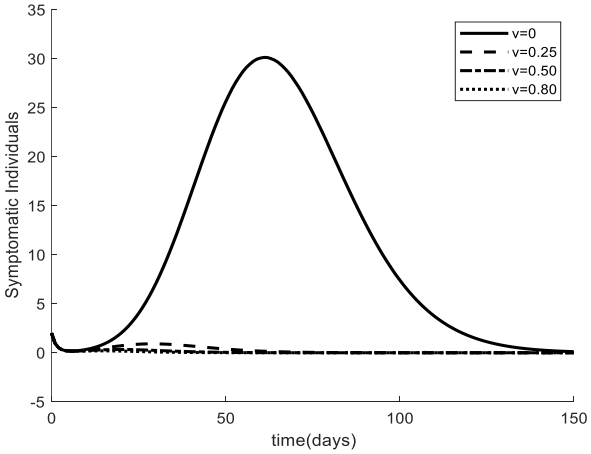
(a)


Exposed Individuals
time(days)
v=0
v=0.25
v=0.50
v=0.80

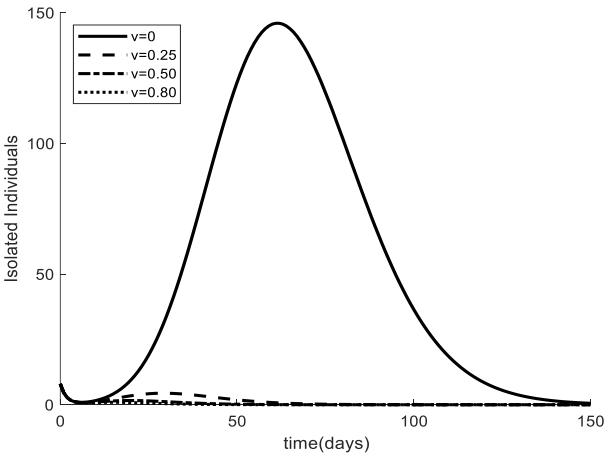
(b)


Asymptomatic Infectious Individuals
time(days)
v=0
v=0.25
v=0.50
v=0.80

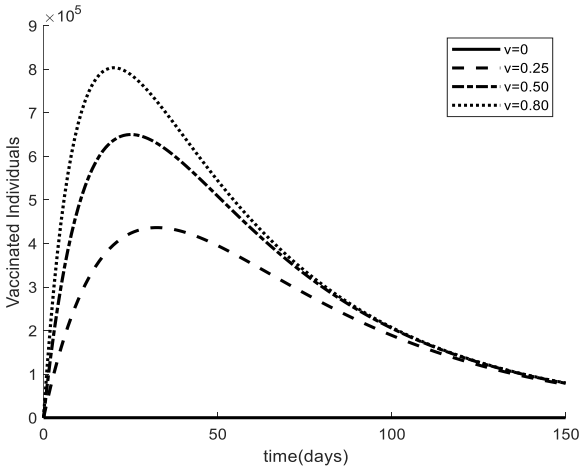
(c)


Symptomatic Individuals
time(days)
v=0
v=0.25
v=0.50
v=0.80

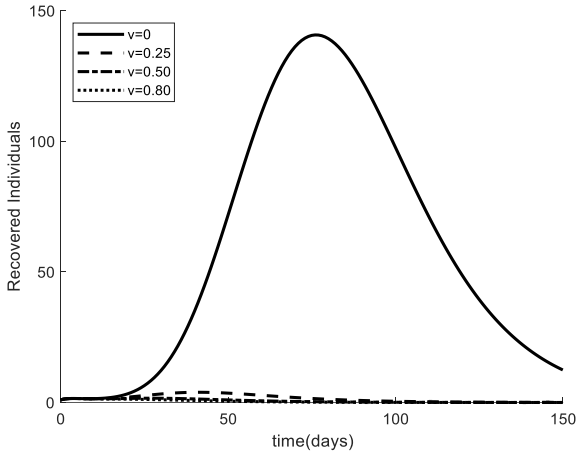
(d)


Isolated Individuals
time(days)
v=0
v=0.25
v=0.50
v=0.80

(e)


×10^5
Vaccinated Individuals
time(days)
v=0
v=0.25
v=0.50
v=0.80

(f)



(g)

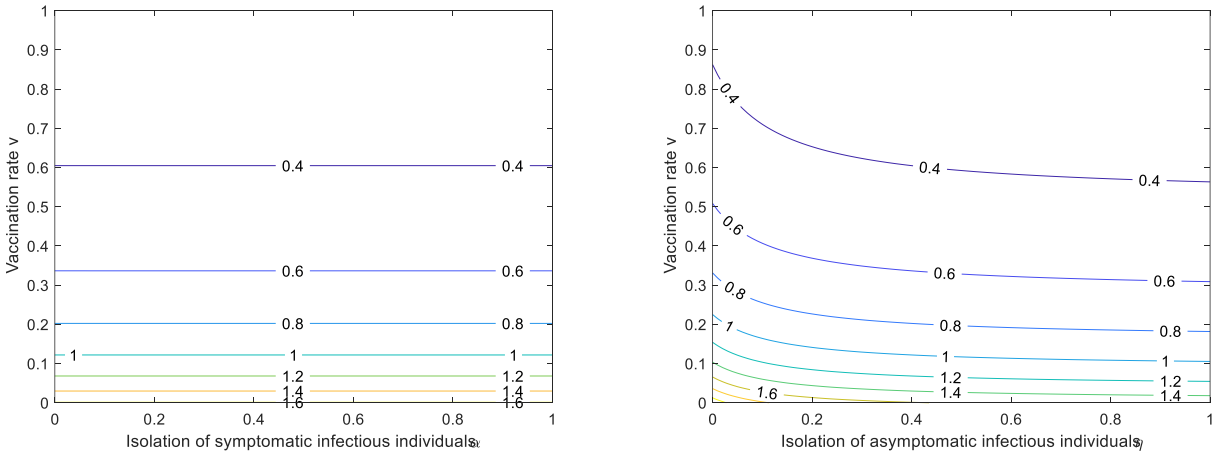
Figure 7. Simulation on the effectiveness of vaccination in the absence of waning for (a) susceptible (b) exposed (c) asymptomatic (d) symptomatic (e) isolated (f) vaccinated (g) recovered individuals.

3.5. Basic Reproduction Number and Sensitivity Index

Figure 8a displays the contour plot of the impact of the rate of vaccination v and isolation of symptomatic infectious individuals α on the basic reproduction number R_0. It is shown that for the combined measure (i.e. vaccination and isolation of symptomatic infectious individuals) to be effective, the vaccination rate should be between 20% and 60%. At these points, the basic reproduction number $R_0 < 1$. If otherwise less than 0.2, the basic reproduction number $R_0 > 1$. The contour plot shows the impact of v and η on R_0 is demonstrated in Figure 8b. It is revealed that for the combined measure (i.e. vaccination and isolation of asymptomatic infectious individuals) to be effective, the vaccination rate should be between 32% and 85%. At these points, the basic reproduction number $R_0 < 1$. If otherwise less than 0.32, the basic reproduction number $R_0 > 1$.

Figure 9 shows the plot of the basic reproduction number R_0 against vaccination v. It is observed that the basic reproduction number can be reduced from 1.6 (in the absence of vaccination) to 0.25 if the effectiveness of vaccination can be increased to 1 i.e. 100%.

The sensitivity analysis of the parameters in the model is displayed in Table 1 and Figure 10. The essence is to study how sensitive a model is to changes in the value of the parameters of the model and to changes in the structure of the model. From Table 1, the sensitivity index for $\Lambda = +1$, which implies that an increase in Λ will bring about an increase of the same proportion in R_0. Similarly, a decrease in Λ will cause a decrease in R_0. Hence, Λ is directly proportional to R_0. Likewise for c_1, c_2, c_3, α and α are all directly proportional to R_0 as observed in Figure 10. Also, the sensitivity index for $\gamma = -0.73733$, which implies that an increase in μ will bring about a decrease of the same proportion in R_0. Similarly, an increase in γ will cause a decrease in R_0. Hence, γ is inversely proportional to R_0. The same for v, ϕ, δ, η and μ are all inversely proportional to R_0 as observed in Figure 10. As observed from Figure 10, the order of magnitude from the largest to the lowest is $\Lambda, c_1, \gamma, v, \phi, c_2, \eta, \varepsilon, c_3, \delta, \alpha$ and α. α is the less sensitive parameter.



(a) (b)

Figure 8. Contour plot showing the impact of v and (a) α (b) η on R_0

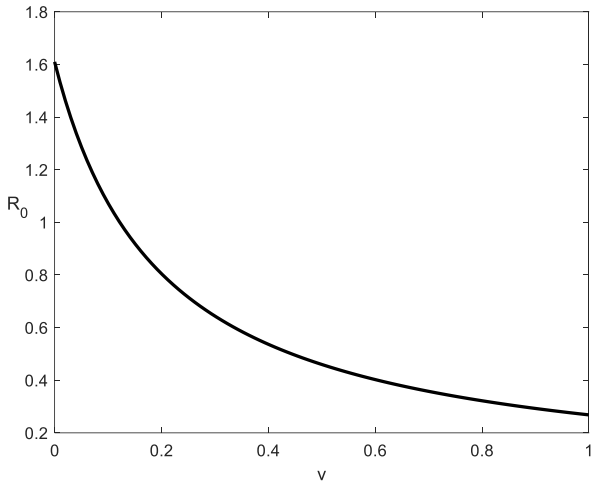


Figure 9. Plot of R_0 against

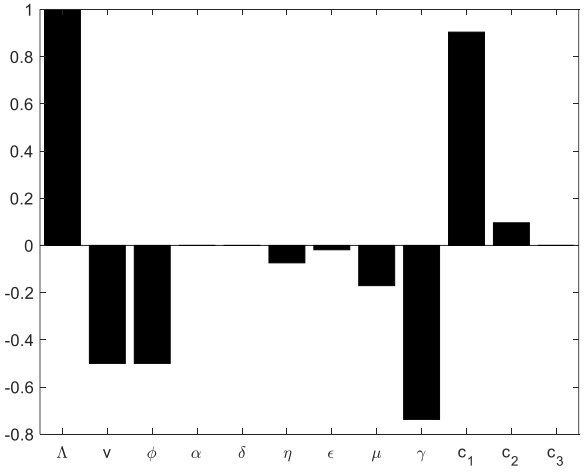


Figure 10. Sensitivity Index of the Basic Reproduction Number for each Parameters

4. Conclusion

This research investigated a deterministic model of the Ebola Virus Disease (EVD) transmission dynamics made up of systems of ordinary differential equations. The model includes susceptible, exposed, asymptomatic, and symptomatic people as well as isolated, vaccinated, and recovered individuals. It was demonstrated that the area exists in which the model is epidemiologically viable. The model is globally asymptotically stable when the reproduction number $R_0 < 1$, which implies that ebola disease will eventually be eliminated from the population. But, unstable when $R_0 > 1$, which implies that ebola would continue to be prevalent among us if control measures are neglected. The effectiveness of isolation and vaccination in preventing the spread of the Ebola virus illness was further investigated via the use of numerical simulation. The effect of the model's parameters on the fundamental reproduction number as well as an assessment of the parameters' sensitivity was also done. According to the study's findings, if the rate of isolated symptomatic infected people and vaccination can be high enough, this would reduce the rate of contact between the susceptible and the exposed people, the rate of contact between the susceptible and asymptomatic people, and the rate of contact between the susceptible and symptomatic people, thereby reducing the infection rate. Consequently, there is a reduction in the number of individuals exposed and infected; hence, there is a reduction in the number of individuals going for treatment. This greatly impacts the transmission dynamics of the Ebola virus disease; the peak of infections is rapidly flattened. Hence, the combined effect (effective vaccination with a zero-wane-off vaccine and isolation of symptomatic individuals) produced a greater result, which, when implemented, would reduce infection rates and thereby lead to the eradication of Ebola Virus Disease (EVD) over time. This study recommends the isolation of infected individuals and efficacious vaccination with a zero-wane-off vaccine, which will help a great deal in curtailing the spread of the Ebola virus disease.

Declaration of Competing Interest

No conflict of interest was declared by the authors.

Authorship Contribution Statement

Oludapo Olubanwo: Writing, Reviewing, and Supervision
Deborah Daniel: Editing, Analysis & Interpretation of Results and Manuscript Preparation
Timothy Adegboye: Methodology, Writing, Reviewing and Editing
Abosede Adeniran: Methodology, Writing, Reviewing and Editing

References

[1] World Health Organization. Frequently asked questions on Ebola virus disease, 2016.

[2] World Health Organization, WHO strategic response plan: West Africa Ebola Outbreak ISBN: ISBN 978 92 4 150869 8 www.who.int, 2015. 0

[3] World Health Organization, October 2014.

[4] Centers for Disease Control and Prevention. Questions and Answers, 2014.

[5] Centers for Disease Control and Prevention. Ebola virus disease, 2015.

[6] Q. Ahman, O. David, A. Chukwuma, N. Ugochukwu and M. Ezike, "Transmission dynamics of Ebola virus disease with vaccine, condom use, quarantine, isolation and treatment drug," *African Journal of Infectious Diseases,* vol. 15, pp. 10-23, 2020.

[7] D. Bausch, J. Towner, S. Dowell, F. Kaducu, M. Lukwiya, A. Sanchez, S. Nichol, T. Ksiazek and P. Rollin, "Assessment of the Risk of Ebola Virus Transmission from Bodily Fluids and Fomites," *The Journal of Infectious Diseases,* vol. 196, pp. S142-S147, 2007.

[8] W. Ahmad and M. Abbas, "Effect of quarantine on transmission dynamics of Ebola virus epidemic: a mathematical analysis," *The European Physical Journal Plus,* vol. 136, 2021.

[9] N. Sullivan, Z.-Y. Yang and G. Nabel, "Ebola Virus Pathogenesis: Implications for Vaccines and Therapies," *Journal of Virology,* vol. 77, pp. 9733-9737, 2003.

[10] A. Pandey, K. Atkins, J. Medlock, N. Wenzel, J. Townsend, J. Childs, T. Nyenswah, M. Ndeffo-Mbah and A. Galvani, "Strategies for containing Ebola in West Africa," *Science,* vol. 346, pp. 991-995, 2014.

[11] D. O. Daniel, "Mathematical Model for the Transmission of Covid-19 with Nonlinear Forces of Infection and the Need for Prevention Measure in Nigeria," *Journal of Infectious Diseases and Epidemiology,* vol. 6, 2020.

[12] S. Onitilo, M. Usman, D. Daniel, T. Odule and S. Sanusi, "Modelling the Transmission Dynamics of Cholera Disease with the Impact of Control Strategies in Nigeria," *Cankaya University Journal of Science and Engineering,* vol. 20, p. 35–52, 2023.

[13] S. Onitilo, M. Usman, D. Daniel, O. Odetunde, Z. Ogunwobi, F. Hammed, O. Olubanwo, A. Ajani, A. Sanusi and A. Haruna, "Mathematical Modelling of the Transmission Mechanism of Plamodium Falciparum," *Natural Science and Advanced Technology Education,* vol. 31, pp. 435-457, 2022.

[14] F. Agusto, "Mathematical model of Ebola transmission dynamics with relapse and reinfection," *Mathematical Biosciences,* vol. 283, pp. 48-59, 2017.

[15] C. Althaus, N. Low, E. Musa, F. Shuaib and S. Gsteiger, "Ebola virus disease outbreak in Nigeria: Transmission dynamics and rapid control," *Epidemics,* vol. 11, pp. 80-84, 2015.

[16] N. Lasisi, N. Akinwande, R. Olayiwola, and A. Cole, "Mathematical Model for Ebola Virus Infection in Human with Effectiveness of Drug Usage," *Journal of Applied Sciences and Environmental Management,* vol. 22, p. 1089, 2018.

[17] F. Fasina, A. Shittu, D. Lazarus, O. Tomori, L. Simonsen, C. Viboud, and G. Chowell, "Transmission dynamics and control of Ebola virus disease outbreak in Nigeria, July to September 2014," *Eurosurveillance,* vol. 19, 2014.

[18] H. Lee, and H. Nishiura, "Sexual transmission and the probability of an end of the Ebola virus disease epidemic," *Journal of Theoretical Biology,* vol. 471, pp. 1-12, 2019.

[19] A. Rachah, "A mathematical model with isolation for the dynamics of Ebola virus," *Journal of Physics: Conference Series,* vol. 1132, p. 012058, 2018.

[20] A. Osemwinyen, "Mathematical Modelling of the Transmission Dynamics of Ebola Virus," *Applied and Computational Mathematics,* vol. 4, p. 313, 2015.

[21] P. Diaz, P. Constantine, K. Kalmbach and E. Jones, " In A Modified SEIR Model for the Spread of Ebola in Western Africa and Metrics for Resource Allocation," *Applied Mathematics and Computation*, vol. 1, 2016.

[22] H. Nishiura and G. Chowell, "Early transmission dynamics of Ebola virus disease (EVD), West Africa, March to August 2014," *Eurosurveillance,* vol. 19, 2014.

[23] A. Rachah and D. Torres, "Mathematical Modelling, Simulation, and Optimal Control of the 2014 Ebola Outbreak in West Africa," *Discrete Dynamics in Nature and Society,* vol. 2015, pp. 1-9, 2015.

[24] M. Safi and A. Gumel, "Mathematical analysis of a disease transmission model with quarantine, isolation and an imperfect vaccine," *Computers & amp; Mathematics with Applications,* vol. 61, pp. 3044-3070, 2011.

[25] S. Edward, E. Luseko, D. Ndidi and E. Simanjilo, "Mathematical Modelling of the Transmission Dynamics of Ebola Virus Disease with Control Strategies,"*International Journal of Sciences: Basic and Applied Research (IJSBAR),* vol. 33, p. 112–130, 2017.

[26] R. Abdulrahman and E. H. Al-Allaf, "Interactive Modelling of lost Landmarks – Qishla of Mosul A Case Study," *Diyala Journal of Engineering Sciences,* vol. 43, pp. 72-86, 2016.