

Modeling the Dynamics of the Marburg Virus Disease

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ABSTRACT

This work models the infection dynamics of the Marburg Virus Disease using the SEIQR model. It is a five-compartment model. We investigated the non-negativity of solutions, invariant region and boundedness, equilibrium points, basic reproduction number, sensitivity, equilibrium points' stability, and bifurcation of the model. For all time $t \geq 0$ and positive initial conditions, we observed that the model has non-negative solutions. The invariant region of the model is established, revealing the population size is bounded. The sensitivity analysis shows that the contact rate, the probability of effective contact and the rate at which exposed individuals become infectious have positive sensitivity indices, while the quarantine rates for the exposed and infectious classes, the death rates, and the recovery rates for the exposed and infectious classes have negative sensitivity indices. We established that the non-negativity of the residual vector $\bar{M}(X, Z)$, resulting from the difference between the rate of change of infected individuals and the Metzler matrix, stops the existence of several endemic equilibria for $R_0 < 1$. Thus, no critical value $R_c < 1$ exists, and there is an extension of local stability of the disease-free equilibrium to global stability on the entire domain. The bifurcation analysis reveals a forward bifurcation and the bifurcation plot confirms that for effective management and eradication the disease, the basic reproduction number R_0 must satisfy $R_0 < 1$, where R_c is a critical value. The mathematical analysis also reveals that the disease-free equilibrium is globally asymptotically stable. The effects of some sensitive parameters on the infectious class are shown via numerical simulations. The numerical simulations were done using the Maple Programming Language, and results were given for a period of 150 days. The model simulation suggests that investments in faster diagnostics and isolation facilities yield a direct return in the form of a smaller, shorter epidemic, and to eradicate the disease quickly, interventions must focus on driving the probability of effective contact down.

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INTRODUCTION

Marburg virus disease (MVD) is a viral hemorrhagic fever caused by a zoonotic pathogen known as Marburg virus. MV affects both human and non-human primates. It is primarily transmitted through prolonged exposure or via contact with infected non-human.

The presence MVD was first acknowledged in 1967 after two Marburg and Germany's Frankfurt outbreaks, as well as in Belgrade. These initial outbreaks were reported to have been linked to research involving green monkeys in African imported from Uganda. Since then, outbreaks and cases of MVD have been reported Angola, Equatorial Guinea, Democratic Republic of the Congo, Ghana, Uganda, Guinea, South Africa, Kenya, and Tanzania. After the initial crossover of the virus to humans, human-to-human transmission may occur with the virus entering the body via mucous membranes or broken skin, through any of the following pathways:

- Direct contact with bodily fluids (blood, urine, saliva, semen, sweat, feces, vomit) of an infected individual who is sick with or has died from Marburg disease;
- Direct contact with contaminated objects and surfaces;
- Direct body fluids contact including semen of recovered patients. Just like the Ebola virus, it is

assumed that Marburg can also persist in body fluids (such as semen) of recovered individuals (WHO, 2025).

Healthcare workers are particularly at risk and have often been infected while caring for patients infected with the virus, especially when appropriate infection prevention and control measures are not rigorously followed. Transmission resulting from the use of contaminated needles or accidental needle-stick injuries is linked to more severe illness, faster clinical decline, and potentially increased mortality. Traditional burial practices that involve physical contact also play a role the transmission of the virus. Individuals are not infectious before the onset of symptoms but remain capable of transmitting the virus for as long as it is present in their blood. Mathematical modeling has been powerful tool in analyzing the dynamics of infectious diseases and population dynamics. Tsetimi, Ossaiugbo and Atonuje (2022) studied the optimal control strategy of the pneumonia infection using the Pontryagin's Maximum Principle on a constructed SEIR deterministic model. Their result shows that combining prevention, vaccination and treatment is the optimal control strategy in the management of the pneumonia infection. Marcus, Ighomaro and Sarduana (2024) extended

mathematical modeling to study dynamics of communal violence using the PAB deterministic model of nonlinear ordinary differential equations where they divided the community into the Peaceful class, the Aggressive class and the Brutal class. They considered the Obiaruku community in Delta State of Nigeria as a case study. Their result showed that under some prescribed conditions, the risk level of communal violence can be reduced to the lowest.

One common approach in modeling MVD dynamics is the use of compartmental models, which partition the population into discrete health states. Washachi, Amoka and Orapine (2024) employed a SEQIR (Susceptible–Exposed–Quarantined–Infectious–Recovered) model to explore the impact of quarantine on transmission dynamics. By this, the stability of the disease-free equilibrium was guaranteed. In order to characterize the latent period, Oladele Durowoju and Adeniyi (2025) formulated a SEIR. Control strategies were formulated and shown that combined efforts reduce the reproduction number.

Beyond deterministic compartment models, Qian, Edmunds, Bausch, *et al.* (2023) employed a branching process approach to model Marburg virus outbreaks by leveraging data from the first 15 recorded outbreaks up to 2022. Their stochastic framework used Approximate Bayesian Computation (ABC) to approximate important parameters like the reproduction numbers, which were found to vary across outbreaks but generally remain low (between 0.5 and 1.2). The branching process model also enabled simulation of different vaccination strategies, prophylactic (mass or targeted) and reactive (ring vaccination), demonstrating how early vaccination can significantly increase the probability of controlling outbreaks within one year compared with no vaccination.

A systematic review by McCabe, *et al.* (2024) highlights that, despite several modeling efforts, mathematical studies focused on MVD remain relatively sparse. Only a single mathematical transmission model was explicitly identified prior to their review, indicating a significant gap in the literature given the public health implications of Marburg virus outbreaks. The review also underscores the limited availability of reliable epidemiological parameter estimates, which constrains model validation and reduces confidence in predictive performance. It calls for improved surveillance data and more extensive modeling research to strengthen epidemic response planning.

To this end, it is important to further improve modelling research to strengthen epidemic response planning. Considering the above literatures, and incorporating some

realistic assumptions, we employ the SEIQR model to clearly reveal and analyse the dynamics of MVD.

Model Formulation

The SEIQR model for MVD divides the human population into: Susceptible class, $S(t)$, exposed class, $E(t)$, infectious class, $I(t)$, quarantined class, $Q(t)$, and recovered class, $R(t)$. All five compartments have been indicated at time t . The susceptible compartment consists individuals that are within the force of infection domain and lacks immunity to the infection. Exposed class comprises of individuals that are infected by the virus and are not yet infectious as the infection is still within the incubation period. Infected individuals who have left the incubation period and can now infect others are grouped under the infectious compartment. The quarantined compartment refers to all infected individuals, symptomatic or asymptomatic, who have been separated from other individuals in the study population in order to prevent further spread, and foster treatment and recovery. Individuals that recovers from the infection are labeled $R(t)$.

Susceptible individuals become exposed at the rate χ , also known as the force of infection. The individual enters into the exposed compartment and remains there according to the incubation period. At the expiration of the incubation period, exposed individuals who have not encountered natural death and have not recovered from the infection, become infectious thereby moving to the infectious class at rate δ . Due to observation and contact tracing, exposed individuals and infectious individuals are quarantined at the rates ω and φ respectively. Individuals in the exposed, infectious and quarantined compartments recover from the infection at rates ξ_1, ξ_2, ξ_3 respectively. We have assumed that exposed individuals, infectious individuals and quarantined individuals have different recovery rates due to the incubation period, environmental exposure and availability of effective treatment.

All compartments experience natural death at rate μ , while death due to the disease occurs in the infectious compartments at rate η . We assumed that the rate of disease-induced death is the same for the infectious and the quarantined individuals, but does not apply to the exposed individuals. Since recovery from MVD does not confer permanent immunity, the model indicates that individuals in the recovered compartment become susceptible again at the rate θ . The flow diagram is given as figure 1 while the model is shown in equation (1).

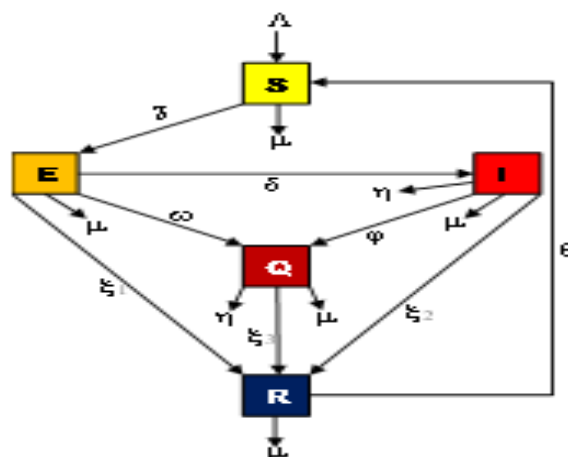


Figure 1: Schematic diagram

The Model Equations

$$\left. \begin{aligned} \frac{dS(t)}{dt} &= \Lambda + \theta R(t) - (\mu + \chi)S(t), \\ \frac{dE(t)}{dt} &= \chi S(t) - (\mu + \delta + \omega + \xi_1)E(t), \\ \frac{dI(t)}{dt} &= \delta E(t) - (\mu + \eta + \phi + \xi_2)I(t), \\ \frac{dQ(t)}{dt} &= \omega E(t) + \phi I(t) - (\mu + \eta + \xi_3)Q(t), \\ \frac{dR(t)}{dt} &= \xi_1 E(t) + \xi_2 I(t) + \xi_3 Q(t) - (\mu + \theta)R(t), \end{aligned} \right\} \quad (1)$$

Subject to the nonnegative initial conditions $S(0) = S_0$, $E(0) = E_0$, $I(0) = I_0$, $Q(0) = Q_0$, $R(0) = R_0$, where $\chi = \frac{\kappa c I}{N}$.

Table 1: Parameters Descriptions and Values

Parameter	Description	Value	Source
Λ	Recruitment rate.	0.45 day ⁻¹	Ajelli and Merler (2012)
μ	Natural death rate	0.000045 day ⁻¹	Addai, Adeniji, et. al. (2012)
η	Disease-induced death rate	0.085 day ⁻¹	Amoah-Mensah, et. al. (2024)
θ	Loss of immunity rate	0.005 day ⁻¹	Assumed
δ	Infectious transition rate	0.18 day ⁻¹	WHO (2025) and Amoah-Mensah, et. al. (2024)
ω	Quarantine rate for exposed	0.05 day ⁻¹	Assumed
ϕ	Quarantine rate for infectious	0.35 day ⁻¹	Amoah-Mensah, et. al. (2024)
ξ_1	Recovery rate for exposed	0.01 day ⁻¹	Assumed
ξ_2	Recovery rate for infectious	0.08 day ⁻¹	Amoah-Mensah, et. al. (2024)
ξ_3	Recovery rate for quarantine	0.09 day ⁻¹	Amoah-Mensah, et. al. (2024)
κ	Effective transmission probability	0.045	Assumed
c	Contact rate	0.70 day ⁻¹	Assumed

Analyses of the MVD Model

Here, we investigate the MVD model to ascertain its biological relevance. Here, we consider the non-negativity of solutions, invariant region and boundedness, equilibrium points, basic reproduction number, sensitivity analysis and stability analysis.

Non-Negativity and Invariant Region of Model Solutions

Theorem 1:

Let

$$\Omega = \left\{ (S, E, I, Q, R) \in \mathbb{R}^{+5} : 0 \leq S + E + I + Q + R = N \leq \frac{\Lambda}{\mu} \right\}$$

be a closed set. Then the MVD model (1) is positively invariant.

Proof: Considering the total population,

$$N(t) = S(t) + E(t) + I(t) + Q(t) + R(t),$$

it is easy to see that

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dQ(t)}{dt} + \frac{dR(t)}{dt} \leq \Lambda - \mu N(t).$$

This implies

$$N(t) \leq (1 - e^{-\mu t}) \frac{\Lambda}{\mu} + N(0)e^{-\mu t}.$$

Furthermore,

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Thus, Ω is the invariant region of the MVD model. This completes the proof.

Theorem 2:

Let

Where:

$$S_e = -\frac{(T_2 \Lambda ((\xi_1 + T_4) T_3 + \omega (\xi_3 + T_4)) T_2 + ((\xi_2 + T_4) T_3 + \phi (\xi_3 + T_4)) \delta) T_1}{(-(-T_3 T_4 T_1 + (\mu T_4 + \xi_1 (\theta + \mu)) T_3 + (\mu T_4 + \xi_3 (\theta + \mu)) \omega) T_1 T_2^2 + (((-c \kappa - \mu) T_4 - \xi_2 (\theta + \mu)) T_3 - (\mu T_4 + \xi_3 (\theta + \mu)) \phi) T_1 + c \theta \kappa (\xi_1 T_3 + \xi_3 \omega)) \delta T_2 + c \theta \delta^2 \kappa (\xi_2 T_3 + \xi_3 \phi))},$$

$$\{(S, E, I, Q, R) \in \mathbb{R}^{+5} : S(0) > 0, E(0) > 0, I(0) > 0, Q(0) > 0, R(0) > 0\}.$$

Then the solution set $(S(t), E(t), I(t), Q(t), R(t))$ of model given in equation 1 is positive for all $t \geq 0$.

Proof: Considering the first equation of the formulated model (1), we have

$$\frac{dS(t)}{dt} = \Lambda + \theta R(t) - (\mu + \chi)S(t) \geq -(\mu + \chi)S(t).$$

This implies $S(t) \geq S_0 e^{-(\mu + \chi)t} > 0$ for all $t \geq 0$. In a similar manner, it can also be shown that $E(t) > 0, I(t) > 0, Q(t) > 0, R(t) > 0$ for all $t \geq 0$. This completes the proof.

Model Equilibrium Points

Here, we determine the Marburg-free equilibrium (MFE) $\mathbb{E}_0$ and Marburg -endemic equilibrium (MEE) $\mathbb{E}_1$. To this end, we set the derivatives in equation (1) to zero to obtain:

$$\left. \begin{aligned} \Lambda + \theta R - (\mu + \chi)S &= 0, \\ \chi S - (\mu + \delta + \omega + \xi_1)E &= 0, \\ \delta E - (\mu + \eta + \phi + \xi_2)I &= 0, \\ \omega E + \phi I - (\mu + \eta + \xi_3)Q &= 0, \\ \xi_1 E + \xi_2 I + \xi_3 Q - (\mu + \theta)R &= 0, \\ E = I = Q &= 0. \end{aligned} \right\} \quad (2)$$

With the understanding that at MFEE $I = Q = R = 0$, we solve (s) to obtain the MFE as

$$\mathbb{E}_0 = (S, E, I, Q, R) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right). \quad (3)$$

To obtain the MEE, we make the notations

$$T_1 = \mu + \delta + \omega + \xi_1, \quad T_2 = \mu + \eta + \phi + \xi_2, \quad T_3 = \mu + \eta + \xi_3, \quad T_4 = \mu + \theta$$

and then solve (2) for S, E, I, Q, and R to obtain the MEE as

$$\mathbb{E}_e = (S_e, E_e, I_e, Q_e, R_e), \quad (4)$$

$$\begin{aligned}
E_e &= -(\Lambda T_2 T_3 T_4 (c\delta\kappa - T_1 T_2)) / (-(-T_3 T_4 T_1 + (\theta\xi_1 + \mu(\xi_1 + T_4))T_3 + (\theta\xi_3 + \mu(\xi_3 + T_4))\omega)T_1 T_2^2 + (((-\xi_2\theta + (-\xi_2 - T_4)\mu - cT_4\kappa)T_3 - (\theta\xi_3 + \mu(\xi_3 + T_4))\phi)T_1 + c\theta\kappa(\xi_1 T_3 + \xi_3\omega))\delta T_2 + c\theta\delta^2\kappa(\xi_2 T_3 + \xi_3\phi)), \\
I_e &= -(\Lambda T_2 T_3 T_4 (c\delta\kappa - T_1 T_2)) / (-(-T_3 T_4 T_1 + (\theta\xi_1 + \mu(\xi_1 + T_4))T_3 + (\theta\xi_3 + \mu(\xi_3 + T_4))\omega)T_1 T_2^2 + (((-\xi_2\theta + (-\xi_2 - T_4)\mu - cT_4\kappa)T_3 - (\theta\xi_3 + \mu(\xi_3 + T_4))\phi)T_1 + c\theta\kappa(\xi_1 T_3 + \xi_3\omega))\delta T_2 + c\theta\delta^2\kappa(\xi_2 T_3 + \xi_3\phi)), \\
Q_e &= -(\Lambda T_4 (c\delta\kappa - T_1 T_2) (T_2\omega + \delta\phi)) / (-(-T_3 T_4 T_1 + (\theta\xi_1 + \mu(\xi_1 + T_4))T_3 + (\theta\xi_3 + \mu(\xi_3 + T_4))\omega)T_1 T_2^2 + (((-\xi_2\theta + (-\xi_2 - T_4)\mu - cT_4\kappa)T_3 - (\theta\xi_3 + \mu(\xi_3 + T_4))\phi)T_1 + c\theta\kappa(\xi_1 T_3 + \xi_3\omega))\delta T_2 + c\theta\delta^2\kappa(\xi_2 T_3 + \xi_3\phi)), \\
R_e &= -((c\delta\kappa - T_1 T_2)\Lambda((\xi_1 T_3 + \xi_3\omega)T_2 + \delta(\xi_2 T_3 + \xi_3\phi))) / (-(-T_3 T_4 T_1 + (\theta\xi_1 + \mu(\xi_1 + T_4))T_3 + (\mu T_4 + \xi_3(\theta + \mu))\omega)T_1 T_2^2 + (((-\xi_2\theta + (-\xi_2 - T_4)\mu - cT_4\kappa)T_3 - (\mu T_4 + \xi_3(\theta + \mu))\phi)T_1 + c\theta\kappa(\xi_1 T_3 + \xi_3\omega))\delta T_2 + c\theta\delta^2\kappa(\xi_2 T_3 + \xi_3\phi)),
\end{aligned}$$

Basic Reproduction Number

Here, we will obtain an expression for basic reproduction number (R_0), the average number of secondary MVD infections resulting from one infectious individual in a completely susceptible population. To this end, we employ the next generation matrix approach (Tsetimi, Ossaiugbo & Atonuje, 2022a; Ossaiugbo & Okposo, 2021; Tsetimi, Ossaiugbo & Atonuje, 2022b; Ossaiugbo & Atonuje, 2022c; Marcus, Augustine, Jonathan & Ighomaro, 2024; Marcus, Augustine, Jonathan, 2024). By the aforementioned approach, we consider the set (E, I, Q) consisting of the MVD infected compartments consider the model $X' = \mathcal{F} - \mathcal{V}$, where

$$\mathcal{F} = \begin{pmatrix} \chi S \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} T_1 E \\ -\delta E + T_2 I \\ -\omega E - \phi I + T_3 Q \end{pmatrix}.$$

Now, the Jacobian matrices evaluated at the MFE are:

$$F = \begin{pmatrix} 0 & \kappa c & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} T_1 & 0 & 0 \\ -\delta & T_2 & 0 \\ -\omega & -\phi & T_3 \end{pmatrix}.$$

Thus,

$$FV^{-1} = \begin{pmatrix} \frac{\kappa c \delta}{T_1 T_2} & \frac{\kappa c}{T_2} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The eigenvalues are:

$$\lambda_1 = \frac{\kappa c \delta}{T_1 T_2}, \lambda_2 = 0, \lambda_3 = 0.$$

Thus, the basic reproduction number of the MVD model, which is the spectral radius of the matrix FV^{-1} , is given as

$$R_0 = \frac{\kappa c \delta}{T_1 T_2} \quad (5)$$

Evaluating equation (6) and table 1, we obtain $R_0 = 0.0459$. R_0 shows that when an infectious individual is brought into a susceptible population, individuals catch infection at rate κc and by the end of the incubation period, proceed to class I at rate δ . Total time spent is $\frac{1}{T_1 T_2}$.

Sensitivity Analysis

To this end, we now study the sensitivity indices of all the parameters of the expression of the basic reproduction number given in equation (6). By this analysis, we clearly present which parameters are positively sensitive, which parameters are negatively sensitive. We employ the approach used by (Kizito & Tumwiine, 2018). The sensitivity index of a parameter v of R_0 is defined as:

$$\zeta_v^{R_0} = \frac{\partial R_0}{\partial v} \times \frac{v}{R_0}. \quad (6)$$

Thus, we obtained the following sensitivity indices:

$$\zeta_{\kappa}^{R_0} = 1 > 0, \quad \zeta_c^{R_0} = 1 > 0, \quad \zeta_{\delta}^{R_0} = \frac{\mu + \omega + \xi_1}{T_1} > 0, \quad \zeta_{\omega}^{R_0} =$$

$$-\frac{\omega}{T_1} < 0, \quad \zeta_{\xi_1}^{R_0} = -\frac{\xi_1}{T_1} < 0,$$

$$\zeta_{\mu}^{R_0} = -\frac{(T_1 + T_2)\mu}{T_1 T_2} < 0, \quad \zeta_{\eta}^{R_0} = -\frac{\eta}{T_2} < 0, \quad \zeta_{\phi}^{R_0} = -\frac{\phi}{T_2} <$$

$$0, \quad \zeta_{\xi_2}^{R_0} = -\frac{\xi_2}{T_2} < 0.$$

Therefore, the parameters with positive sensitivity indices are κ , c and δ . Parameters with negative sensitivity indices include ω , μ , η , ϕ and ξ_2 . Increasing the values of the parameters with positive indices while keeping fixed the values of the parameters with negative indices, results in an increment in the value of R_0 . Lowering their values decreases the value of R_0 . However, increasing the values of the parameters with negative sensitivity indices, results in a reduction of R_0 .

Stability Analysis

Knowing the stability of an equilibrium point helps to modify the essential parameters in order to achieve the desired initial compartmental sizes, which in turn shall produce the desired compartmental sizes over time. The stabilities of the MFE are verified using the following theorems.

Local Stability of the DFE

Theorem 3

The DFE given in equation (3) is locally asymptotically stable if $R_c < R_0 < 1$. It is unstable if $R_0 > 1$. R_c is a critical value.

Proof:

Consider $J(\mathbb{E}_0)$, the Jacobian matrix, evaluated at MFE.

$$J(\mathbb{E}_0) = \begin{pmatrix} -\mu & 0 & -\kappa c & 0 & \theta \\ 0 & -\mu - \delta - \omega - \xi_1 & \kappa c & 0 & 0 \\ 0 & \delta & -\mu - \eta - \phi - \xi_2 & 0 & 0 \\ 0 & \omega & \phi & -\mu - \eta - \xi_3 & 0 \\ 0 & \xi_1 & \xi_2 & \xi_3 & -\mu - \theta \end{pmatrix} \quad (7)$$

$$\text{Trace}(J(\mathbb{E}_0)) = -(5\mu + \delta + \omega + \xi_1 + 2\eta + \phi + \xi_2 + \xi_3 + \theta) < 0.$$

$$\text{Det}(J(\mathbb{E}_0)) = (\mu + \theta)(\mu + \eta + \xi_3)\mu(-\mu^2 + (-\xi_2 - \eta - \phi - \delta - \omega - \xi_1)\mu + (\kappa c - \xi_2 - \eta - \phi)\delta - (\omega + \xi_1)(\xi_2 + \eta + \phi)).$$

For locally asymptotic stability, we require that $\text{Det}(J(\mathbb{E}_0)) > 0$ and $\text{Trace}(J(\mathbb{E}_0)) < 0$. The trace of $J(\mathbb{E}_0)$ satisfies this condition. From equation (6), we obtain

$$\delta = \frac{R_0(\xi_1 + \mu + \omega)(\mu + \eta + \phi + \xi_2)}{(-\mu - \eta - \phi - \xi_2)R_0 + \kappa c},$$

so that substitution into the expression for $\text{Det}(J(\mathbb{E}_0))$ yields

$$\text{Det}(J(\mathbb{E}_0)) = \frac{(\mu + \eta + \xi_3)\kappa c(R_0 - 1)(\xi_1 + \mu + \omega)(\mu + \eta + \phi + \xi_2)(\mu + \theta)\mu}{\kappa c - (\mu + \eta + \phi + \xi_2)R_0}.$$

Now from the condition $\text{Det}(J(\mathbb{E}_0)) > 0$, we solved for R_0 and obtained $R_0 < 1$ iff $\kappa c - (\mu + \eta + \phi + \xi_2)R_0 < 0$. Therefore, MFE is locally asymptotically stable if $\frac{\kappa c}{(\mu + \eta + \phi + \xi_2)} < R_0 < 1$. To this end, we have established that under the condition $\frac{\kappa c}{(\mu + \eta + \phi + \xi_2)} < R_0 < 1$ (using table 1, $0.0612 < R_0 < 1$), all solutions of the MVD model, whose initials conditions are within the pull of the MFE, are attracted to $\mathbb{E}_0$. ■

Global Stability of the DFE

In order to investigate the global stability of the DFE, we use the method employed by Ossaiugbo and Okposo (2021). In this approach, we rewrite the MVD model in the form

$$\left. \begin{aligned} \frac{dX}{dt} &= L(X, Z) \\ \frac{dZ}{dt} &= M(X, Z), \quad M(X, 0) = 0 \end{aligned} \right\} \quad (8)$$

Where (X, Z) denotes the state variables, $X = (S, R) \in \mathbb{R}^2$ denotes the components of uninfected individuals, $Z = (E, I, Q) \in \mathbb{R}^3$ denotes the components of infected individuals, and the DFE is denoted by $\mathbb{E}_0 = (X^*, 0)$ with $X^* = (\frac{\Lambda}{\mu}, 0)$. The global stability of the DFE is guaranteed by the following two conditions, whenever $R_0 \leq 1$:

C1: For $\frac{dX}{dt} = L(X, 0)$, X^* is globally asymptotically stable.

C2: $\frac{dZ}{dt} = D_Z M(X^*, 0)Z - \hat{M}(X, Z)$, where $\hat{M}(X, Z) \geq 0$ for all $(X, Z) \in \Omega$.

$D_Z M(X^*, 0)$ is a Metzler matrix, that is any matrix which satisfies $A = (a_{ij}) \geq 0 \quad \forall \quad i \neq j$ and $X, Z \in \Omega$, the invariant region.

Theorem 4

The DFE $\mathbb{E}_0 = (X^*, 0)$ is globally asymptotically stable if $R_0 \leq 1$ and conditions C1 and C2 are satisfied.

Proof: Observe that

$$\frac{dX}{dt} = L(X, Z) = \begin{pmatrix} \Lambda + \theta R - (\mu + \chi)S \\ \xi_1 E + \xi_2 I + \xi_3 Q - (\mu + \theta)R \end{pmatrix} \quad (9)$$

$$\frac{dZ}{dt} = M(X, Z) = \begin{pmatrix} \chi S - (\mu + \delta + \omega + \xi_1)E \\ \delta E - (\mu + \eta + \phi + \xi_2)I \\ \omega E + \phi I - (\mu + \eta + \xi_3)Q \end{pmatrix} \quad (10)$$

Thus, let us consider the system

$$\left. \frac{dX}{dt} \right|_{Z=0} = L(X, 0) = \begin{pmatrix} \Lambda + \theta R - \mu S \\ -(\mu + \theta)R \end{pmatrix} \quad (11)$$

From equation (12), we can extract the ordinary differential equation:

$$\frac{dS}{dt} = \Lambda + \theta R(t) - \mu S \leq \Lambda - \mu S \quad (12)$$

Equation reveals that $S(t) \leq \frac{\Lambda}{\mu} + (S(0) - \frac{\Lambda}{\mu})e^{-\mu t}$. Observe that as $t \rightarrow \infty$, $S \rightarrow \frac{\Lambda}{\mu}$. Thus, $X^* = (\frac{\Lambda}{\mu}, 0)$ is a globally asymptotically stable equilibrium point for $\left. \frac{dX}{dt} \right|_{Z=0} = L(X, 0)$.

To this end, let us consider the matrix $D_Z M(X^*, 0)Z$.

$$D_Z M(X^*, 0) = \begin{pmatrix} -\mu - \delta - \omega - \xi_1 & \kappa c & 0 \\ \delta & -\mu - \eta - \phi - \xi_2 & 0 \\ \omega & \phi & -\mu - \eta - \xi_3 \end{pmatrix}.$$

$$\therefore D_Z M(X^*, 0)Z = \begin{pmatrix} (-\mu - \delta - \omega - \xi_1)E + \kappa c I \\ \delta E + (-\mu - \eta - \phi - \xi_2)I \\ \omega E + \phi I + (-\mu - \eta - \xi_3)Q \end{pmatrix}.$$

From condition C2 and equation (11), we now obtain

$$\hat{M}(X, Z) = D_Z M(X^*, 0)Z - M(X, Z) = \begin{pmatrix} \kappa c I (E + I + Q + R) \\ N \\ 0 \\ 0 \end{pmatrix}.$$

Since $D_Z M(X^*, 0)$ is a Metzler matrix and $\hat{M}(X, Z) \geq 0$ for all $(X, Z) \in \Omega$, it follows that condition C2 is also satisfied. Therefore, the DFE is globally asymptotically stable whenever $R_0 \leq 1$. ■

Note: We emphasize here that the non-negativity of $\hat{M}(X, Z)$ stops the existence of several endemic equilibria for $R_0 < 1$. Thus, no critical value $R_c < 1$ exists, and there is an extension of local stability to global stability on the domain.

Bifurcation Analysis

We now study the bifurcation pattern of the MVD model (1) using the center manifold theorem as implemented by

(Ossaiugbo & Atonuje, 2022c; Marcus, Augustine & Jonathan, 2024).

Theorem 5

(Castillo-Chavez & Song, 2004): Consider the following general system of ODEs with a parameter ϕ :

$$\frac{dx}{dt} = f(x, \phi), \quad f: \mathbb{R}^n \times \mathbb{R} \rightarrow \mathbb{R}^n, \quad f \in C^2(\mathbb{R}^n \times \mathbb{R}) \quad (13)$$

where 0 is an equilibrium point of the system, that is, $f(0, \phi) \equiv 0$ for all ϕ . Consider the following:

A1. The linearization matrix $\mathcal{A} = D_x f(0, 0) = \left(\frac{\partial f_i}{\partial x_j}(0, 0) \right)$ of (13) around 0 with ϕ calculated at 0. Zero is a simple eigenvalue of $\mathcal{A}$, and other eigenvalues have negative real parts.

A2. The matrix $\mathcal{A}$ takes a nonnegative right eigenvector w , and also takes a left eigenvector v both matching with the zero eigenvalue.

Consider f_k the k th element of f and

$$\left. \begin{aligned} a &= \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, 0) \\ b &= \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0, 0) \end{aligned} \right\} \quad (14)$$

The local dynamics of (14) around 0 are entirely determined by the signs of a and b :

- $a > 0, b > 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable, and there exists a negative and locally asymptotically stable equilibrium.
- $a < 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;
- $a > 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears;
- $a < 0, b > 0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if $a > 0$ and $b > 0$, then a backward bifurcation occurs at $\phi = 0$.

Proof: Given the MVD model (1), we set $x_1 = S, x_2 = E, x_3 = I, x_4 = Q, x_5 = R$. i.e.

$$\left. \begin{aligned} x_1' &= \Lambda + \theta x_5 - (\mu + \chi)x_1, \\ x_2' &= \chi x_1 - (\mu + \delta + \omega + \xi_1)x_2, \\ x_3' &= \delta x_2 - (\mu + \eta + \phi + \xi_2)x_3, \\ x_4' &= \omega x_2 + \phi x_3 - (\mu + \eta + \xi_3)x_4, \\ x_5' &= \xi_1 x_2 + \xi_2 x_3 + \xi_3 x_4 - (\mu + \theta)x_5, \\ \chi &= \frac{\kappa c I}{x_1 + x_2 + x_3 + x_4 + x_5}. \end{aligned} \right\} \quad (15)$$

Setting $R_0 = 1$ in equation (6) and using c (contact rate) as the bifurcation parameter, we obtain

$$c^* = \frac{(\mu + \delta + \omega + \xi_1)(\mu + \eta + \phi + \xi_2)}{\kappa \delta}.$$

The Jacobian matrix around the MFE ($x_1 = \frac{\Lambda}{\mu}, x_2 = 0, x_3 = 0, x_4 = 0, x_5 = 0$), is:

$$\mathcal{A} = \begin{pmatrix} -\mu & 0 & -\kappa c^* & 0 & \theta \\ 0 & -\mu - \delta - \omega - \xi_1 & \kappa c^* & 0 & 0 \\ 0 & \delta & -\mu - \eta - \phi - \xi_2 & 0 & 0 \\ 0 & \omega & \phi & -\mu - \eta - \xi_3 & 0 \\ 0 & \xi_1 & \xi_2 & \xi_3 & -\mu - \theta \end{pmatrix} \quad (16)$$

The Eigenvalues are:

$$\lambda_1 = 0, \lambda_2 = -\mu, \lambda_3 = -\mu - \theta, \lambda_4 = -\mu - \eta - \xi_3, \lambda_5 = -\xi_1 - \xi_2 - \delta - \eta - 2\mu - \omega - \phi.$$

Hence 0 is a simple eigenvalue of $\mathcal{A} = D_x f(0,0)$. Let $w = (w_1, w_2, w_3, w_4, w_5)^T$ be a right eigenvector for the system $\mathcal{A}w = 0$, expanded as

$$\left. \begin{aligned} -\kappa c^* w_3 - \mu w_1 + \theta w_5 &= 0 \\ (-\mu - \delta - \omega - \xi_1) w_2 + \kappa c^* w_3 &= 0 \\ \delta w_2 + (-\mu - \eta - \phi - \xi_2) w_3 &= 0 \\ \omega w_2 + \phi w_3 + (-\mu - \eta - \xi_3) w_4 &= 0 \\ \xi_1 w_2 + \xi_2 w_3 + \xi_3 w_4 + (-\mu - \theta) w_5 &= 0 \end{aligned} \right\} \quad (17)$$

Solving the system, we obtain the following solution

$$w_3 > 0, w_2 = \frac{(\mu + \eta + \phi + \xi_2)}{\delta} w_3 > 0, w_4 = \frac{w_3(\omega(\mu + \eta + \phi + \xi_2) + \phi\delta)}{\delta(\mu + \eta + \xi_3)} > 0, w_5 = \frac{(\xi_1(\mu + \eta + \phi + \xi_2)(\mu + \eta + \xi_3) + (\omega\eta + \omega\mu + (\delta + \omega)(\phi + \xi_2))\xi_3 + \delta\xi_2(\mu + \eta))w_3}{\delta(\mu + \eta + \xi_3)(\mu + \theta)} > 0,$$

$$w_1 = -(w_3(((\delta(c\kappa - \xi_2) - (\xi_1 + \omega)\xi_3 - (\xi_2 + 2\eta + \phi)\xi_1)\mu + ((c\kappa - \xi_2 - \phi)\delta - \omega(\xi_2 + \eta + \phi) - (\xi_2 + \eta + \phi)\xi_1)\xi_3 + (\delta(c\kappa - \xi_2) - (\xi_2 + \eta + \phi)\xi_1)\eta - \xi_1\mu^2)\theta + c\mu\delta\kappa(\mu + \eta + \xi_3)))/(\delta(\mu + \eta + \xi_3)(\mu + \theta)\mu).$$

The negativity of the component w_1 is negligible since the vector w corresponds to the zero eigenvalue and the component w_1 corresponds the state variable S . Right eigenvectors of the Jacobian matrix provide information about the direction and rates of change of the system's components (state variables) at equilibrium. A zero eigenvalue or an eigenvalue with a small real part (close to zero) implies that the system will tend toward stability over time.

Let $v = (v_1, v_2, v_3, v_4, v_5)$ be a left eigenvector for the system $v\mathcal{A} = 0$, expanded as

$$\left. \begin{aligned} -\mu v_1 &= 0 \\ v_2(-\mu - \delta - \omega - \xi_1) + v_3\delta + v_4\omega + v_5\xi_1 &= 0 \\ -m_1 v_1 + m_2 v_2 - v_3(\mu + \eta + \phi + \xi_2) + v_4\phi + v_5\xi_2 &= 0 \\ v_4(-\mu - \eta - \xi_3) + v_5\xi_3 &= 0 \\ (v_1 - v_5)\theta - v_5\mu &= 0 \end{aligned} \right\} \quad (18)$$

Where

$$m_1 = \frac{(\mu + \delta + \omega + \xi_1)(\mu + \eta + \phi + \xi_2)}{\delta}, m_2 = \frac{(\mu + \delta + \omega + \xi_1)(\mu + \eta + \phi + \xi_2)}{\delta}.$$

Solving the system (19), we obtain the following solution

$$v_1 = v_4 = v_5 = 0, v_3 > 0, v_2 = \frac{v_3\delta}{\mu + \delta + \omega + \xi_1}.$$

From equation (15) we obtain the a and b as

$$a = -m_3(1 + m_4 + m_5 + m_6)w_3^2 < 0, b = \frac{\kappa\delta}{\mu + \delta + \omega + \xi_1}w_3v_3 > 0,$$

Where

$$m_3 = 2\mu v_3(\mu + \eta + \phi + \xi_2)/\Lambda,$$

$$m_4 = (\xi_1(\mu + \eta + \phi + \xi_2)(\mu + \eta + \xi_3) + (\omega\eta + \omega\mu + (\delta + \omega)(\phi + \xi_2))\xi_3 + \delta\xi_2(\mu + \eta))/\delta(\mu + \eta + \xi_3)(\mu + \theta),$$

$$m_5 = \frac{(\omega(\mu + \eta + \phi + \xi_2) + \phi\delta)}{\delta(\mu + \eta + \xi_3)},$$

$$m_6 = \frac{(\mu + \eta + \phi + \xi_2)}{\delta}.$$

Therefore, since $a < 0$ and $b > 0$, when $R_0 < 1$, MFE is locally asymptotically stable. Any small introduction of infection dies out. When $R_0 > 1$, MFE becomes unstable. Thus, below the threshold, the disease cannot conquer the population. For $R_0 > 1$, a unique positive endemic equilibrium emerges from the MFE. Thus once R_0 exceeds unity, the disease persists at a low but stable level in the population. It follows that there is no backward bifurcation. Lowering the value of R_0 below unity is enough to eliminate the disease. Figure 2 shows the forward bifurcation plot.

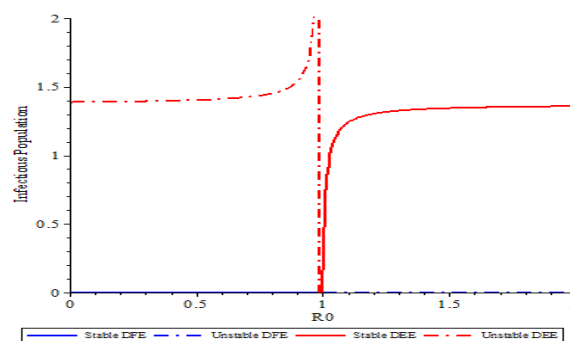


Figure 2: Forward Bifurcation plot

Numerical Simulations

Using the dsolve and odeplot functions in maple programming software, together with the values of the parameter in table 1, and with the initial data: $S(0) = 400$,

$E(0) = 40$, $I(0) = 0$, $Q(0) = 50$ and $R(0) = 20$, we obtained the following simulations.

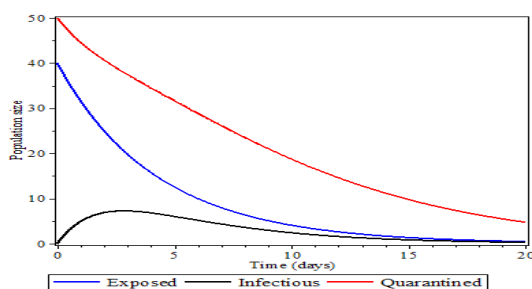


Figure 3: Exposed, Infectious and Quarantined

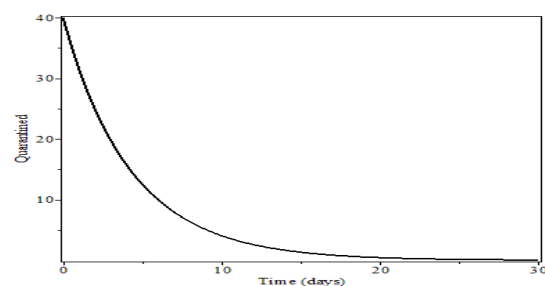


Figure 4a: Exposed class (first 30 days)

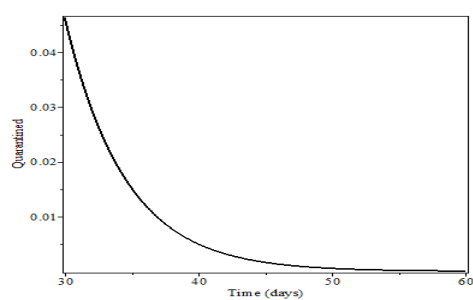


Figure 4b: Exposed class (days 30-60)

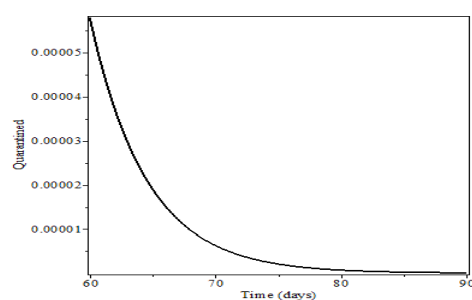


Figure 4c: Exposed class (days 60-90)

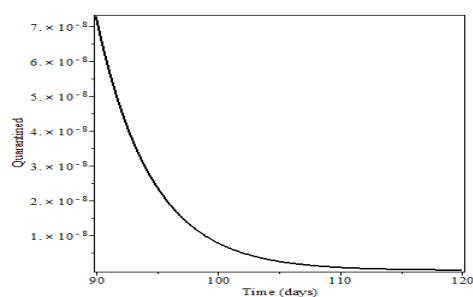


Figure 4d: Exposed class (days 90-120)

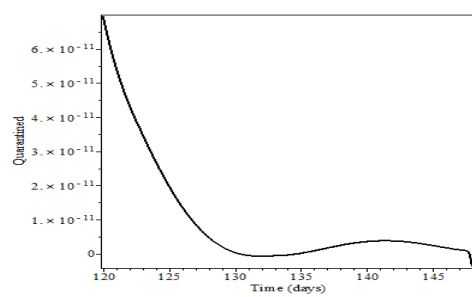


Figure 4e: Exposed class (days 120-150)

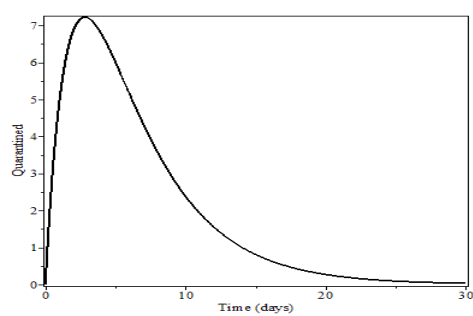


Figure 5a: Infectious class (first 30 days)

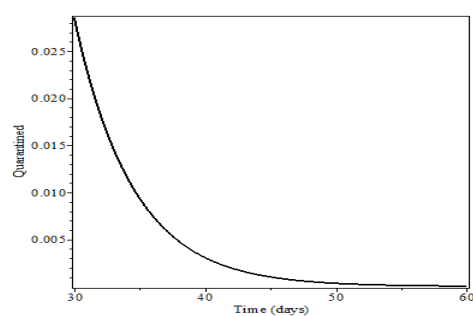


Figure 5b: Infectious class (days 30-60)

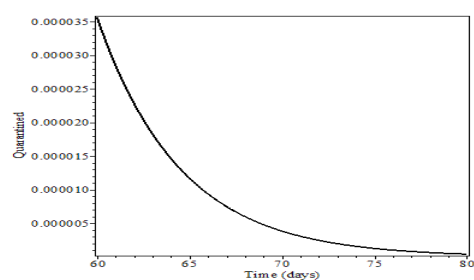


Figure 5c: Infectious class (days 60-80)

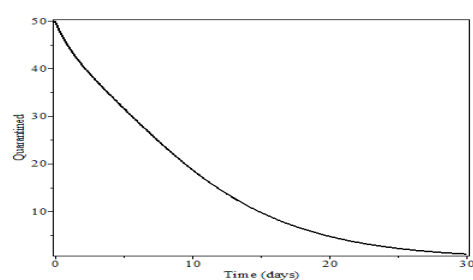


Figure 6a: Quarantined class (first 30 days)

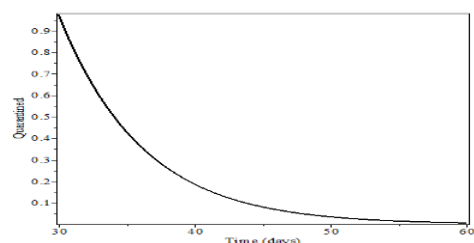


Figure 6b: Quarantined class (days 30-60)

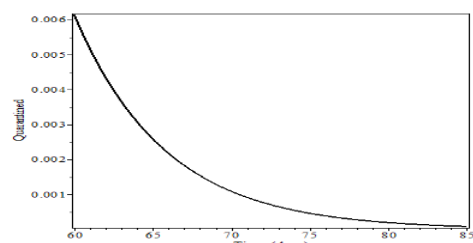


Figure 6c: Quarantined class (days 60-85)

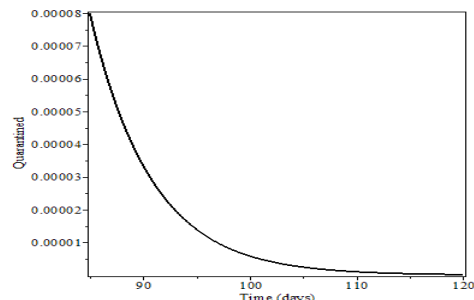
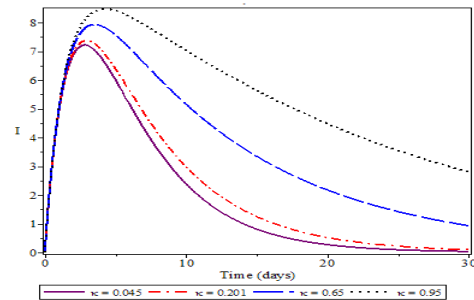
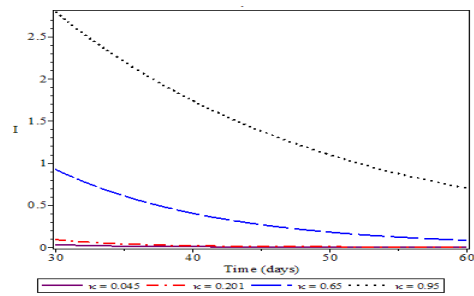
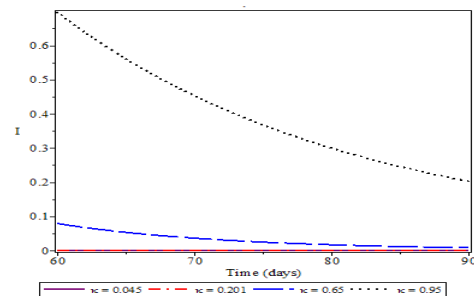
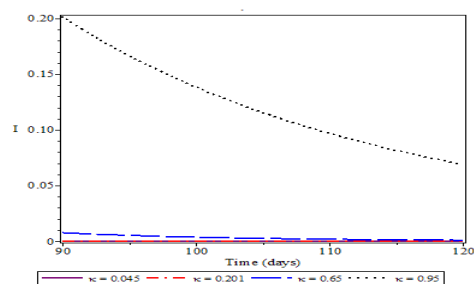
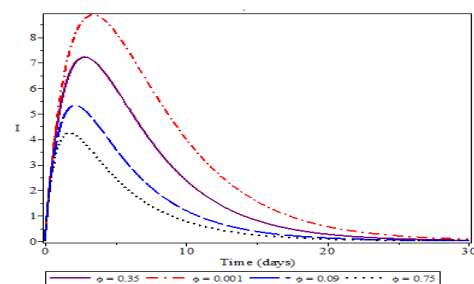
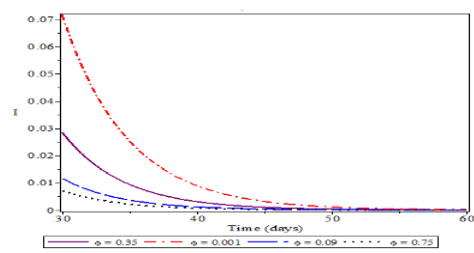
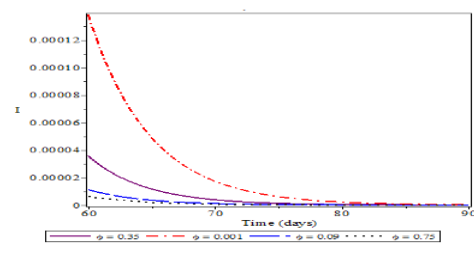


Figure 6d: Quarantined class (days 85-120)

Figure 7a: Effect of κ on class I (first 30 days)Figure 7b: Effect of κ on class I (days 30-60)Figure 7c: Effect of κ on class I (days 60-90)Figure 7d: Effect of κ on class I (days 90-120)Figure 8a: Effect of ϕ on class I (first 30 days)Figure 8b: Effect of ϕ on class I (days 30-60)Figure 8c: Effect of ϕ on class I (days 60-90)

Discussion of Simulations and Conclusion

Figure 3 shows that the exposed population starts at a high initial value, indicating substantial early exposure following disease introduction. Then decreases time, revealing the fast advancement to infectious compartment. We observe that the infectious population remains low during the time interval considered, showing that the basic reproduction number is well suppressed and that by public health intervention, there is a quick detection and quarantine. The quarantined population shows a sharp early peak followed by rapid decay, revealing fast isolation of suspected individuals and followed by recovery or release of quarantined individuals. The fast decline implies quarantine is effective but temporary, preventing onward transmission.

Figures 4a to 4e decompose the exposed class over successive time windows to reveal both macro and micro level behavior. Figure 4a shows the early phase of the exposed class. Here,

the exposed population declines very steeply. This period corresponds to high initial exposure, rapid progression out of the exposed compartment, and strong early response, such as quarantine. Figure 4b shows the intermediate phase of the exposed class. The decline continues but at a slower rate. Most exposed individuals have already transitioned, and residual exposure is minimal, suggesting reduced community transmission, and near elimination of new exposure events. Figure 4c reveals the advanced phase of the exposed class where the exposed population reaches very small magnitudes. The disease is now largely under control, and transmission chains are nearly exhausted. Figure 4d reveals the late phase of the exposed class, where the curve approaches zero asymptotically, no resurgence is observed, and long-term stability of the disease-free state is confirmed. Figure 4e shows the tail-end dynamics of the exposed class where very small oscillations appear near zero. These represent

numerical effects of the solver, minor fluctuations due to parameter sensitivity, and the oscillations do not grow, indicating system stability and absence of re-infection waves. Figure 5a shows the first 30 days class where the population of infectious individuals rises sharply at the beginning, reaching a small but noticeable peak within the first few days. This initial increase represents transition of individuals from exposed to infectious class, and early-stage transmission before full-scale intervention effects dominate. This is followed shortly by a rapid monotonic decline, revealing fast isolation, reduction of effective contact and that the effective reproduction number is below unity. Figure 5b shows the infectious class continues to decrease exponentially for days 30 to 60, showing prevalence of a very low order. This suggests absence of secondary outbreaks and system stability. For days 60 to 80 as shown in figure 5c, we see that the infectious population reduces to order 10^{-5} . No resurgence. Now there is effective elimination of the infection from the population. Thus, confirming that the MFE is a global attractor.

All four graphs in figures 6a to 6d show a movement toward total eradication; the endemic equilibrium reduces to zero. Stability is confirmed in figure 6c. In figure 6d, the value is zero, effectively, hence meaningless, biologically.

Figure 7a shows that the number of infectious individuals spikes rapidly, peaking around day 3-4, and then drops sharply. At the very peak, the curves are clustered, suggesting that the initial explosion of cases is driven by the initial conditions. However, as the curve starts to slope downwards, a gap begins to form. The dotted line ($\kappa = 0.95$) stays higher than the solid line ($\kappa = 0.045$). Thus, even with effective interventions in place, a higher transmission probability buffers the decline, keeping more active cases in the system. In figure 7b, the y-axis scale drops to a maximum of 2.8, thus the separation between the curves becomes very distinct. The curves order themselves perfectly according to the parameter value. Therefore, as time passes, the “force of infection” (which is a function of κ) fights against the decay. A high κ creates a “fatter tail” for the epidemic curve, meaning the disease takes much longer to burn out. At day 60 as shown in figure 7c, for $\kappa = 0.95$, the infection level is about seven times that of $\kappa = 0.045$. Hence, the simulation reveals that the disease duration is stretched due to high probability of effective contact. Figure 7d further reveals this trend, confirming that in the presence of the mathematical noise, higher values of κ results in stronger infection. Thus, for effective eradication of the disease, κ should be reduced.

We observe a sudden initial growth in all cases shown in figure 8a, thereafter a decline followed. The peak heights vary according to the value of ϕ . For $\phi = 0.001$, the highest peak is at 9 on the y-axis, while for $\phi = 0.75$, the lowest peak is at 4.2. Thus, secondary infections are prevented by quickly taking away infectious individuals from the overall population. Figure 8b shows a wider gap between the scenarios, even as the different cases decay. Here, $\phi = 0.001$ gives significantly higher graph than the rest, while $\phi = 0.75$ gives a much lower result. We observe here that given an efficient quarantine; the peak infection is not only lowered but a fast decline is guaranteed. Figure 8c confirms that this substantial difference in the results is maintained even at low levels. Here, nearly the double of the prevalence obtained at fast quarantine is obtained at slow quarantine. Thus, poor isolation increases the risk of resurgence. The model suggests that investments in faster diagnostics and isolation facilities ($\phi \rightarrow 1$) yield a direct return in the form of a smaller, shorter epidemic.

To this end, we have established that no critical value $R_c < 1$ exists for the SEIQR model and there is an extension of local stability of the disease-free equilibrium to global stability on the entire domain. The bifurcation analysis resulted in a forward bifurcation and the bifurcation plot, as shown in figure 2, confirmed that for effective management and eradication the disease, R_0 must satisfy $R_0 < 1$. The sensitivity analysis shows that the contact rate, the probability of effective contact and the rate at which exposed individuals become infectious have positive sensitivity indices, while the quarantine rates for the exposed and infectious classes, the rates of natural and disease-induced deaths, and the recovery rates for the exposed and infectious classes have negative sensitivity indices. The effects of some sensitive parameters on the infectious class have been shown via simulations.

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