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Mathematical Modelling of Transmission Dynamics of Marburg Virus with Effective Quarantine Approach

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Marburg virus disease (MVD) is a severe infection with an extremely high fatality rate, spread through direct and indirect contact. Recently, there was an outbreak of the deadly MVD disease. We studied the transmission dynamics of MVD through the SEQIR model and determined the basic reproduction number, the local stability, and the global stability of the disease-free equilibrium. This study aims to mathematically model the transmission dynamics of MVD using an effective Quarantine Approach. Building upon existing research, the model introduces a quarantine compartment and analyses the disease-free equilibrium.

The results indicate that quarantining exposed individuals effectively curb the transmission of MVD and prevents secondary infections. Additionally, the introduction of treatment in the model ensures the survival of patients. However, it is emphasized that further research is essential to develop specific treatments for MVD, as preventive measures alone may not be sufficient to control the disease's spread.

Keywords: Marburg Virus, Quarantining, Local Stability, Global Stability, Bifurcation.

1. Introduction

Marburg virus disease (MVD), formerly known as Marburg Haemorrhagic fever, is a severe, often fatal illness in humans. It causes severe viral haemorrhagic fever in humans [1]. The virus, Marburg, is the carrier of Marburg virus disease (MVD), a disease with a case fatality ratio between 24-88%, but it can be much lower with good patient care [1]- [2].

The first reported case of Marburg virus (MARV) was in August 1967, when laboratory workers in Marburg and Frankfurt, Germany, and Belgrade, Yugoslavia (now Serbia), were infected with a previously unknown infectious agent [1], [3]- [4]. The source of infection was traced back to African green monkeys (Chlorocebus aethiops) that had been imported from Uganda and were shipped to all three locations. The primary infections ironically occurred when the monkeys were necropsied to obtain kidney cells to culture poliomyelitis vaccine strains. In the remarkable period of fewer than three months, the etiologic agent was isolated, characterized, and identified by the joint effort of scientists in Marburg and Hamburg [5] and was later confirmed by Kunz and colleagues [6] and Kissling and colleagues [7]. An outbreak of MVD poses a serious public health threat because of its severe and often fatal nature [2]. A cumulative number of 108 individuals (50 from the Ashanti region, 48 from the Savannah region, and 10 from the Western region) were identified as contacts of the two cases, all of whom were under self-quarantine and daily monitoring for 21 days. On 20 July, all contacts completed their follow-up period. These contacts included healthcare workers and immediate family members of the cases. One contact reported some symptoms, but a collected blood sample tested negative on 7 July. All the other contacts were reported to be in good health during the follow-up period [2]. As of the year 2012, 452 cases and 368 documented deaths were recorded due to MVD [3]. However, there is reason to assume that the actual numbers might be higher.

Initial MVD patients are believed to contract the virus via exposure to an infected animal: either a reservoir host (several bat species) or a spill-over host such as NHPs, as described in the first MVD outbreak [8]- [9]. Following transmission to humans, the virus's spread between individuals results from direct contact with blood or other body fluids (saliva, sweat, stool, urine, tears, and breast milk) from infected patients. People remain

infectious as long as their blood contains the virus [1].

Supportive care, including rehydration with oral or intravenous fluids and treatment of specific symptoms, improves survival. There is no proven treatment available for MVD. However, a range of potential treatments, including blood products, immune therapies, and drug therapies, are being evaluated [1]- [2]. The focus of this study is to mathematically model the transmission dynamics of Marburg Virus Disease (MVD) with an effective Quarantine Approach.

2. Mathematical Formulation

In this paper, we divided the total population into five compartments. $S(t)$ represents the number of susceptible individuals at a time t. $E(t)$ symbolizes the number of exposed individuals at a time t. The exposed category refers to the incubation period where all of the individuals have been infected but are not yet infectious [10].

The Quarantine compartment $Q(t)$ contains individuals who are being observed to see if they would show symptoms of the disease. Those who show symptoms return to the susceptible class. symbolizes the number of infectious individuals at a time t and $R(t)$ represents the number of recovered (or removed) individuals at a time t.

The recruitment into the susceptible population $(S-compartment)$ is due to birth or migration at the rate Λ and reduced by natural death μS. β is the contact rate, ω is the transmission rate from exposed to infectious, δ is induced death rate, γ is the recovery rate and μ is the natural death rate in all the compartments. where $\beta > 0$. As the susceptible human population encounter infectious humans either by direct or indirect contact, they proceed to the exposed compartment, E. In this phase of incubation, the exposed humans become infectious (for those who show clear signs and symptoms) and enter the infected compartment I, at a rate ω, while those exposed humans who are asymptomatic are isolated by contact tracing for a close watch within the Quarantine compartment Q, at a rate φ. After a close watch and laboratory diagnosis of the quarantine population, those that are without the MARV return to the susceptible population at a rate τ, while those who become infectious are moved to the infected compartment I. The infected individuals, I remain infected and infectious for some time before recovering by treatment or reduced to natural death μI and casualty δI.

Those who recover from the MVD by way of treatment enter into the recovered compartment, R at a rate, γ and, the population of the recovered is normally reduced to natural death, μR. The natural dynamics of Marburg Virus Disease (MVD) is well represented in the model transmission diagram in Figure 1, with the description of the variables and parameters given in Table 1. The model is illustrated below

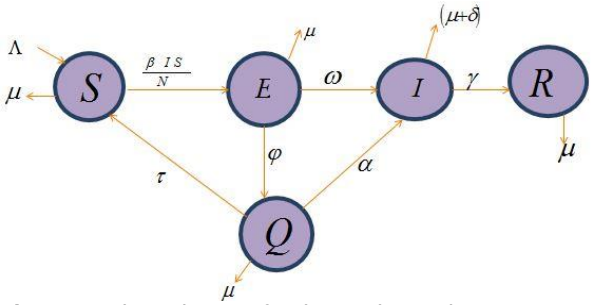

Figure 1: Flow Diagram for the Marburg Virus Diseases

2.1 Model Equations

$$\left.\begin{aligned} \frac{dS}{dt} &= \Lambda + \tau Q - (\mu +)S \\ \frac{dE}{dt} &= -(\mu + \varphi + \omega)E \\ \frac{dQ}{dt} &= \varphi E - (\mu + \alpha + \tau)Q \\ \frac{dI}{dt} &= \omega E + \alpha Q - (\mu + \delta + \gamma)I \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned}\right\} \quad (1)$$

With initial conditions;

$$S(t_0) = S_0, E(t_0) = E_0, Q(t_0) = Q_0, I(t_0) = I_0, R(t_0) = R_0$$

3. Model Analysis

In this section, we obtain the following results which guarantee that the MVD governed by the system (1) is epidemiologically and mathematically well-posed in a feasible Region given by:

$$\left\{\Omega = (S, E, Q, I, R) = \mathrm{R}_+^5 / S > 0, E > 0, Q > 0, I > 0, R > 0, N \le \frac{\Lambda}{\mu}\right\}$$

Table 1: Variables and Parameter Description

Variable	Description
$S(t)$	The susceptible human population at a time t
$E(t)$	Exposed human population at a time t
$I(t)$	The infected human population at a time t
$R(t)$	The recovered human population at a time t
$Q(t)$	The quarantined human population at a time t
Λ	Recruitment rate for the human population
μ	Natural death rate
δ	Disease-induced death rate
β	Probability of MARV transmission by human-to-human
ω	The rate at which exposed humans get infected
φ	The rate at which exposed humans get quarantined
τ	The rate at which exposed humans moved back to susceptible populations
α	The rate at which quarantine humans get infected
γ	The recovery rate from the MVD

3.1 Disease-Free and Endemic Equilibrium Points of the MVD Model

We find the equilibrium points by setting the right-hand side of the system (1) to zero as follows:

$$\left.\begin{aligned} &\Lambda+\tau Q-\left(\mu+\frac{\beta I}{N}\right)S=0\\ &\frac{\beta IS}{N}-(\mu+\varphi+\omega)E=0\\ &\varphi E-(\mu+\alpha+\tau)Q=0\\ &\omega E+\alpha Q-(\mu+\delta+\gamma)I=0\\ &\gamma I-\mu R=0 \end{aligned}\right\} \quad (2)$$

When there is no disease in the population $E(t)=Q(t)=I(t)=R(t)=E_b(t)=I_b(t)=0$ the disease-free equilibrium (DFE) point of the MDV model (1) exists and is given by,

$$E_0=(S_0,E_0,Q_0,I_0,R_0)=\left(\frac{\Lambda}{\mu},0,0,0,0\right)$$

To compute the endemic equilibrium point (EEP), we define

$\theta_1=,\theta_2=\mu+\varphi+\omega,\ \theta_3=\mu+\alpha+\tau,\ \theta_4=\mu+\delta+\gamma$

The system of equations (2) can be solved in terms of $\theta_1,\theta_2,\theta_3$ and θ_4 to obtain the EEP,

$$E_p=(S,\ E,\ Q,\ I,\ R)$$

where,

$$S=\frac{\theta_2\theta_3\ \Lambda}{\theta_2\theta_3(\mu+\theta_1)-\tau\theta_1\varphi}$$

$$Q=\frac{\theta_1\ \Lambda\varphi}{\theta_2\theta_3(\mu+\theta_1)-\tau\theta_1\varphi}$$

$$R=\frac{\theta_1\ \Lambda\gamma(\omega\theta_3+\alpha\varphi)}{\theta_4\theta_3\theta_2(\mu^2+\mu\theta_1)-\mu\tau\varphi\theta_4\theta_1}$$

3.2 Basic Reproduction Number

The basic reproduction number, R_0 of the MVD model for the system of equations (1) is the average number of secondary infections created by an infectious individual introduced during the period of infectiousness into the susceptible population. It can be calculated as $R_0=\eta\left(FV^{-1}\right)$ where.

$$F=\begin{pmatrix}0&0&\beta\\0&0&0\\0&0&0\end{pmatrix} \text{ and } V=\begin{pmatrix}\theta_2&0&0\\-\varphi&\theta_3&0\\-\omega&-\alpha&\theta_4\end{pmatrix}$$

and $$F\square V^{-1}=\begin{pmatrix}\frac{\beta(\alpha\varphi+\omega\theta_3)}{\theta_2\theta_3\theta_4}&\frac{\beta\alpha}{\theta_3\theta_4}&\frac{\beta}{\theta_4}\\0&0&0\\0&0&0\end{pmatrix}$$

Hence

$$R_0=\frac{\beta(\alpha\varphi+\omega\theta_3)}{\theta_2\theta_3\theta_4} \quad (3)$$

3.3 Local Stability of the Disease-Free Equilibrium of the MVD Model

Proposition 1: The disease-free equilibrium E_0 point of the MVD model is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$. This proposition is achieved by $\left|J(E_0)-\lambda I\right|=0$

$$\left|J(E_0)-\lambda I\right|=\begin{vmatrix}-(\lambda+\mu) & 0 & \tau & -\beta & 0\\ 0 & -(\lambda+\theta_2) & 0 & \beta & 0\\ 0 & \varphi & -(\lambda+\theta_3) & 0 & 0\\ 0 & \omega & \alpha & -(\lambda+\theta_4) & 0\\ 0 & 0 & 0 & \gamma & -(\lambda+\mu)\end{vmatrix}=0 \quad (4)$$

Reducing (4) we have

$$\begin{vmatrix}-(\lambda+\theta_2) & 0 & \beta\\ \varphi & -(\lambda+\theta_3) & 0\\ \omega & \alpha & -(\lambda+\theta_4)\end{vmatrix} \quad (5)$$

$$\lambda_1=\lambda_5=-\mu$$

Therefore, the characteristics of equation (5) are.

$$A_4\lambda^3+A_3\lambda^2+A_2\lambda+A_1=0 \quad (6)$$

where

$$A_4=1,\ A_3=(\theta_2+\theta_3+\theta_4),\ A_2=\begin{pmatrix}\theta_4(\theta_2+\theta_3)\\ -\beta\omega+\theta_2\theta_3\end{pmatrix},$$

$$A_1=\left(\beta\omega\theta_2-\alpha\beta\varphi-\beta\omega(\theta_2+\theta_3)+\theta_2\theta_3\theta_4\right)$$

We apply the Routh-Hurwitz criterion on (6) which states that all roots of the polynomial have a negative real part if and only if the coefficients $A_{i=5}$ are positive and the determinant of the matrices $H_1>0$ for $i=1,2,3,4,5$. Thus

$$H=\begin{bmatrix}A_1 & A_3 & A_5\\ 1 & A_2 & A_4\\ 0 & A_1 & A_3\end{bmatrix}>0$$

Therefore,

$H_1=A_1$

$H_1>0$

$$H_2=\begin{bmatrix}A_1 & A_3\\ 1 & A_2\end{bmatrix}$$

$A_1A_2>A_3$

$H_2>0$

$$H_3=\begin{bmatrix}A_1 & A_3 & 0\\ 1 & A_2 & A_4\\ 0 & A_1 & A_3\end{bmatrix}$$

$-A_4A_1^2+A_2A_1A_3-A_3^2>0$

$H_3>0$

3.4 Global Stability of the disease-free equilibrium point

For global stability of the DFE, the technique used by Castilo-Chavez et al. [11] was employed. The model is rewritten as follows:

$$\left.\begin{aligned}\frac{\mathrm{d}X}{\mathrm{d}t}&=K(X,Z)\\ \frac{\mathrm{d}Z}{\mathrm{d}t}&=G(X,Z),\ G(X,0)=0\end{aligned}\right\} \quad (7)$$

Where, $X\in\mathrm{R}^2$ and $X=\{S,R\}$ denotes the number of uninfected individuals, and $Z\in\mathrm{R}^3$ where, $Z=\{E,Q,1\}$ denotes the number of infected individuals. $E^0=\left\{\frac{\Lambda}{\mu},0,0,0,0\right\}$ denotes the disease-free equilibrium point of this system, where,

$$x^*=\left\{\frac{\Lambda}{\mu}\right\} \quad (8)$$

Condition (7) may be met to guarantee global asymptotic stability

(H_1): For $\frac{\mathrm{d}x}{\mathrm{d}t}=k(x,0),x^*$ is globally asymptotic stable.

(H_2): For $G(X,Z)=AZ-\hat{G}(X,Z),\hat{G}(X,Z)\geq 0$ $\forall(x,z)\in\Gamma$ where, $A=D^G\left(x^*,0\right)$ is an m matrix and Γ is the region where the model has biological meaning.

Theorem 1. If the system (1) satisfies condition (7), then the fixed point $E^0=\left\{\frac{\Lambda}{\mu},0,0,0,0\right\}$ is a globally asymptotically stable equilibrium of the system (1) provided that $R_o<1$ and the conditions (H_1) and (H_2) are satisfied:

Proof

Consider $K(X,0)=[\Lambda-\mu S]$ and $G(X,Z)$ and

$$G(X,Z)=AZ-\hat{G}(X,Z) \quad (9)$$

And

$$Z=\begin{bmatrix}E\\ Q\\ I\end{bmatrix} \quad (10)$$

$$G(X,Z)=\begin{bmatrix}\frac{\beta IS}{N}-(\mu+\varphi+\omega)E\\ \varphi E-(\mu+\alpha+\tau)Q\\ \omega E-(\mu+\delta+\gamma)I\end{bmatrix} \quad (11)$$

Given

$$\hat{G}(X,Z)=AZ-G(X,Z) \quad (12)$$

Substituting equations (9), (10), and (11) into (12) we have

$$G(\hat{X},Z)=\begin{bmatrix} -(\mu+\varphi+\omega) & 0 & \frac{\beta S}{N} \\ \varphi & -(\mu+\alpha+\tau) & 0 \\ \omega & \alpha & -(\mu+\delta+\gamma) \end{bmatrix}\begin{bmatrix} E \\ Q \\ I \end{bmatrix}-\begin{bmatrix} \frac{\beta IS}{N}-(\mu+\varphi+\omega)E \\ \varphi E-(\mu+\alpha+\tau)Q \\ \omega E-(\mu+\delta+\gamma)I \end{bmatrix}=\begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} \quad (13)$$

Since all the conditions are satisfied, $\hat{G}(X,Z)=0$, the DFE, E^0 is globally asymptotically stable.

3.5 Stability Analysis Using Bifurcation Analysis

The centre manifold theory can be used to analyse the stability nearer the DFE (E^0) and $R_0=1$. Therefore, from $R_0=\frac{\beta_\Lambda(\alpha\varphi+\omega\theta_3)}{N\mu\theta_2\theta_3\theta_4}$, let $\beta=\bar{\omega}$ be the bifurcation parameter and $R_0=1$ be the bifurcation point and making $\bar{\omega}$ subject of the formula we have

$$\bar{\omega}=\frac{\omega_\Lambda(\alpha\varphi+\omega\theta_3)}{\Lambda(\alpha\varphi+\bar{\omega}\theta_3)}.$$

Also, let

$$x_1=S\ \ x_2=E\ \ x_3=Q\ \ x_4=I\ \ x_5=R \quad (14)$$

substituting (14) into (1) we have

$$\left.\begin{aligned} \dot{x}_1 &= f_1=\Lambda+\tau x_3-\left(\mu+\frac{\bar{\omega}x_4}{N}\right)x_1 \\ \dot{x}_2 &= f_2=\frac{\bar{\omega}x_4x_1}{N}-(\mu+\varphi+\omega)x_2 \\ \dot{x}_3 &= f_3=\varphi x_2-(\mu+\alpha+\tau)x_3 \\ \dot{x}_4 &= f_4=\omega x_2+\alpha x_3-(\mu+\delta+\gamma)x_4 \\ \dot{x}_5 &= \gamma x_4-\mu x_5 \end{aligned}\right\} \quad (15)$$

Differentiating (15) at disease free equilibrium we have

$$\begin{bmatrix} -\mu & 0 & \tau & -\bar{\omega} & 0 \\ 0 & -\theta_2 & 0 & \bar{\omega} & 0 \\ 0 & \varphi & -\theta_3 & 0 & 0 \\ 0 & \omega & \alpha & -\theta_4 & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{bmatrix} \quad (16)$$

Evaluating (16) for right and left eigenvalues we have

$$\left.\begin{aligned} W_1 &= \left(\frac{\tau\varphi}{\mu\theta_3}-\frac{\varpi}{\theta_4}\left(\omega+\frac{\alpha\gamma}{\theta_3}\right)\right)w_2 \\ W_2 &= w_2\ \ free \\ W_3 &= \frac{\gamma}{\theta_3}w_2 \\ W_4 &= \frac{1}{\theta_4}\left(\omega+\frac{\alpha\gamma}{\theta_3}\right)w_2 \\ W_5 &= \frac{1}{\mu\theta_4}\left(\omega+\frac{\alpha\gamma}{\theta_3}\right)w_2 \end{aligned}\right\} \quad (17)$$

$$\left.\begin{aligned} v_1 &= 0 \\ v_2 &= v_2\ \ free \\ v_3 &= \frac{\alpha\gamma}{\theta_3\theta_4}v_2 \\ v_4 &= \frac{\alpha\gamma}{\theta_3\theta_4}v_2 \\ v_5 &= 0 \end{aligned}\right\} \quad (18)$$

After computing the right and left eigenvalues we have to establish the conditions for the existence of backward bifurcation by determining the sign of **a** and **b** as indicated in the theorem below.

Theorem 2. Consider the following general system of ordinary differential equations with a parameter θ.

$$\frac{dx}{dt}=f(x,\theta),\ f:\mathbb{R}^n\times\mathbb{R}\to\mathbb{R}^n \text{ and } f\in\mathbb{R}^2(\mathbb{R}^n\times\mathbb{R}) \quad (19)$$

where θ is an equilibrium point of the system, that is, $f(0,\theta)\equiv 0\forall\theta$ and

i. $A=D_xf(0,0)=\left[\frac{\partial f_i}{\partial x_i}(0,0)\right]$ is the linearization matrix of the system around the equilibrium with θ evaluated at 0.

ii. Zero is a simple eigenvalue of A and all other eigenvalue A have negative real parts.

iii. Matrix A has a right eigenvector w and a left eigenvector v corresponding to the zero eigenvalues.

Let f_k be the Kth component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \quad (20)$$

and

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \theta}(0,0) \quad (21)$$

The local dynamics of system (15) around equilibrium are totally governed by the signs of a and b. thus, the following conditions:

i. $a > 0,\ b > 0,$ when $\theta < 0,$ with $|\theta| << 1,\ 0$ is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \theta << 1,$ the equilibrium is unstable and there exists a negative and locally asymptotically stable equilibrium.

ii. $a < 0,\ b < 0$ when $\theta < 0$ with $|\theta| << 1,\ 0$ is unstable; when $0 < \theta << 1,$ the equilibrium is locally asymptotically stable, and there exists a positive unstable equilibrium.

iii. $a > 0,\ b < 0,$ when $\theta < 0,$ with $0 < \theta << 1,$ is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \theta << 1,\ 0$ is stable, and a positive unstable equilibrium appears.

iv. $a < 0,\ b > 0,$ when θ 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 $\theta = 0$: [12].

Computation of a and b

$V_1 = V_5 = 0$ or $k = 1,5;$ we can only consider $k = 2,3,4.$ The only partial derivatives different from zero.

Evaluating equation (15) we have

$$\frac{\partial^2 f_2}{\partial x_4 \partial x_1} = \frac{\bar{\omega}}{\mu} \quad (22)$$

Substituting equation (17), (18) and (22) into (20) we have

$$a = v_2 \omega_2 \frac{\bar{\omega}\mu}{\theta_4 \theta_2}\left(\omega + \frac{\alpha\varphi}{\theta_3}\right){\omega_2}^2 > 0.$$

Evaluating equation (15) we have

$$\frac{\partial^2 f_2}{\partial x_4 \partial \varpi} = \frac{x_1}{N} = \frac{\Lambda}{\mu} \times \frac{\mu}{\Lambda} = 1 \quad (23)$$

Substituting equations (17), (18) and (23) into (21) we have, $b = v_2 \omega_2 \bar{\omega} > 0$.

Since $a > 0$ and $b > 0,$ the first condition in theorem 2 holds. Thus, the Marburg model exhibits a backward bifurcation.

4. Numerical Simulation

There is a shortage of data about the MVD outbreak. We performed numerical simulations and estimated the parameters based on statistics from [1], [3]- [10]. A typical little town in Angola with 253 inhabitants would have experienced a massive Marburg epidemic. We include a

population size of susceptible humans, $S(t) = 253$ with 2 initial infected individuals in our simulation [10]. As shown in the following figures, we examine the dynamics of the spread across intervals of days.

Table 2: Parameter values

Variable/Parameter	Value	Reference
$S(0)$	3	[11], [13]
$E(0)$		Estimated
$Q(0)$		Estimated
$I(0)$		[11]
$R(0)$		Estimated
Λ	40	[11]
μ	15	Estimated
δ	90	[13]
β	18	Estimated
ω	15	Estimated
τ	10	Estimated
φ	85	Estimated
α	90	Estimated
γ	25	Estimated

Figure 2 shows a decline of susceptible population within a very short time to zero.

When the susceptible human is exposed as shown in Figure 3, there is a swift rise in the population of the exposed group within a short time. It also shows a swift decline with the effect of quarantining, the further transmission of human-to-human transmission is averted.

There will be the need to quarantine more humans as a result of the exposure of susceptible humans. The exposed class group is controlled limiting an increase in the spread of the infection due to quarantining as seen in Figure 4.

Figure 5 illustrates a rapid increase in the infected human population and within a short time, there is a drastic decline due to the effects of quarantining.

In figure 7 we discover that if no treatment $(\gamma = 0)$ is administered, there would be no recovery at all. More treatment induced will salvage more lives from the infection We see from figure 8 that the introduction of the quarantine compartment reduces the exposure of the susceptible population to the infection. Therefore, quarantining helps in the reduction of any secondary infection or even the spread of the infection because there will be no contact between the infected class and the susceptible, reducing the exposure significantly.

With little or no quarantining, we can say that the number of infected humans increases rapidly. We can conveniently say that the quarantine compartment has an effect in reducing the number of infected humans, thereby showing the significance of having a quarantine compartment. See Figure 9.

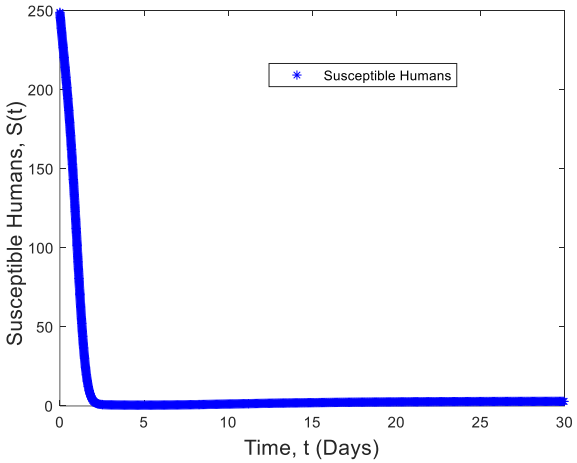

Figure 2: Diagram showing the dynamics of Susceptible Humans

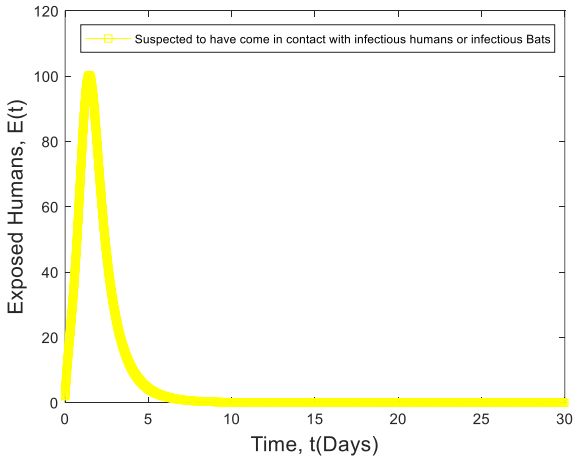

Figure 3: Diagram showing the dynamics of Exposed Humans

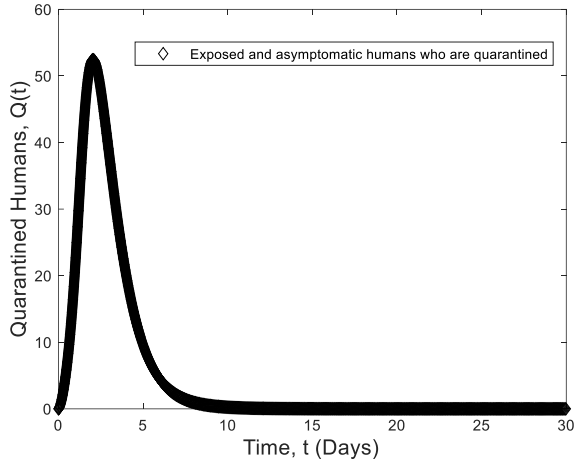

Figure 4: Diagram showing the dynamics of Quarantined Humans

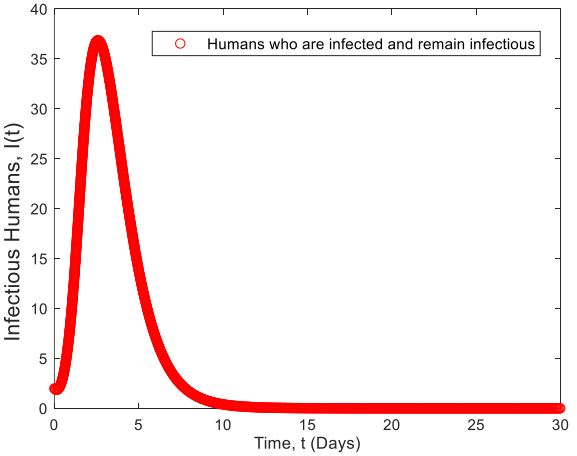

Figure 5: Diagram showing the dynamics of Infectious Humans

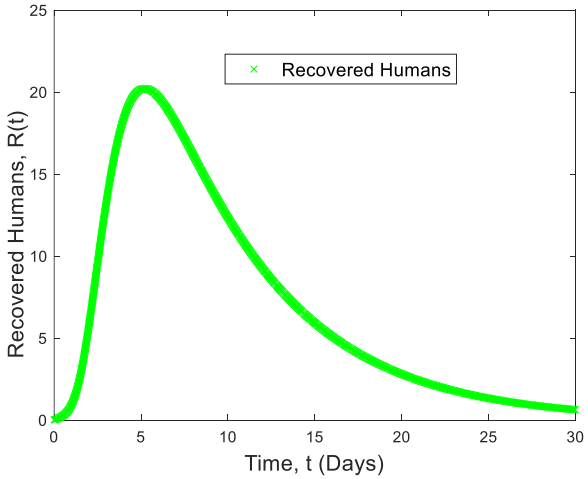

Figure 6: Diagram showing the dynamics of Recovered Humans

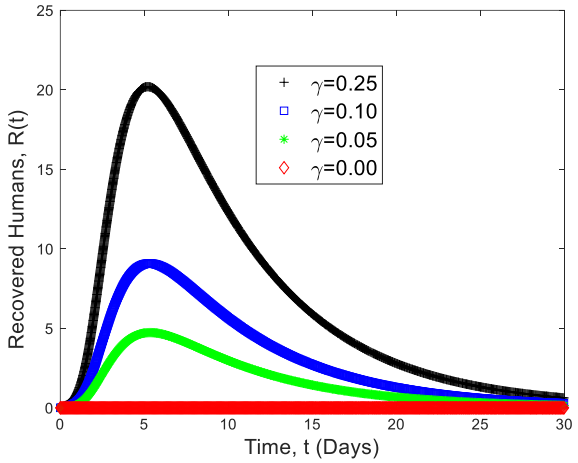

Figure 7: Showing the effect of Treatment Therapy on Recovered Humans

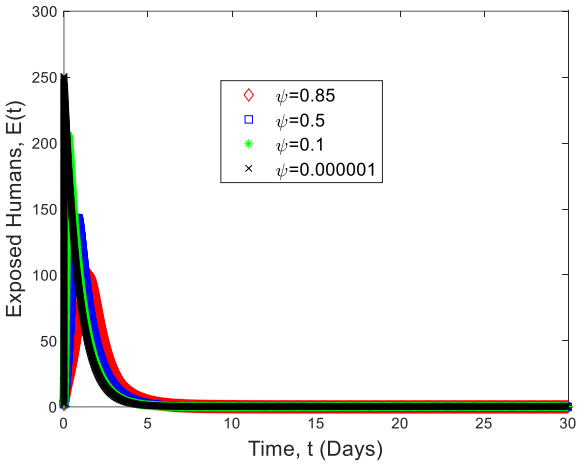

Figure 8: Showing the effect of Effective Quarantine on Exposed Humans

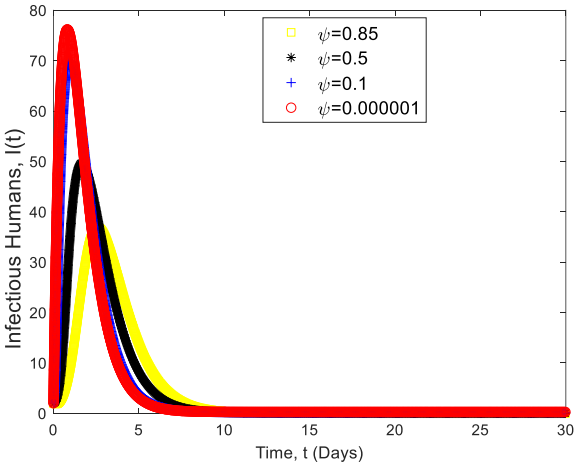

Figure 9: Showing the effect of Effective Quarantine on Infectious Humans

5. Conclusion

We have extended the work of [10] by adding a quarantine compartment. Analytic studies have been carried out and a disease-free equilibrium obtained. From the results we see that the model is locally stable at $R_0 < 1$ and for the global stability [11] technique was employed.

We have seen from this research that quarantining exposed humans is effective in curbing the transmission of MVD and preventing any form of secondary infection. It is also noteworthy to state that in the absence of any form of treatment at the moment, this model has shown that when treatment is introduced the survival of humans from MVD is ascertained. It is important to state here that even though preventive measures can be taken to reduce the spread of the disease, more research should be carried out to produce treatment solutions for the disease.

Conflict of interest

The authors declare no conflict of interest.

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