

Leveraging Multi-Component Control Strategies for Marburg Virus Suppression: A Mathematical Modeling Approach

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ABSTRACT

Marburg virus disease is a severe and often fatal hemorrhagic fever that poses a significant threat to global health. Its high transmissibility, incubation period, and nonspecific symptoms hinder early detection and effective containment. Limited public awareness, delayed diagnosis, and restricted access to reliable health information further heighten the risk of widespread outbreaks.

This study develops a mathematical model for Marburg virus transmission using a compartmental SEIR (Susceptible, Exposed, Infectious, Recovered) framework. The inclusion of an Exposed compartment captures the latent period before symptom onset, improving the accuracy of disease progression modeling. Key parameters, such as the basic reproduction number R_0 , are estimated to evaluate the virus's potential for sustained transmission. Sensitivity and stability analyses identify the most critical factors influencing outbreak dynamics and thresholds for containment.

To guide intervention efforts, optimal control strategies such as public education, early diagnosis and treatment, case isolation, and vector control are incorporated into the model. Numerical simulations and cost-effectiveness assessments demonstrate that combined strategies can reduce infection rates and lower R_0 below the epidemic threshold.

Despite data limitations and implementation challenges, this model offers valuable insights for public health decision-making, emphasizing the need for integrated and timely responses to prevent future Marburg virus outbreaks.

Keywords- Marburg virus, SEIR model, Basic reproduction number, Sensitivity analysis and Infectious disease control.

I. INTRODUCTION

Marburg virus disease (MVD) is a severe hemorrhagic fever caused by Marburg virus (MARV), a member of the *Filoviridae* family closely related to Ebola virus [1, 2]. First identified in 1967 during simultaneous outbreaks in Marburg and Frankfurt, Germany, and Belgrade, Serbia, MVD is a highly fatal zoonotic disease, with case fatality rates reaching up to 88%, depending on clinical care and outbreak conditions [3, 4].

The natural reservoir of MARV is the Egyptian fruit bat (*Rousettus aegyptiacus*), which can transmit the virus to humans through direct contact with bats or their excreta [5–8]. Human infection also occurs via

prolonged exposure to bat-inhabited caves and mines, contact with infected animals, or consumption of contaminated food products [4]. Secondary human-to-human transmission occurs through contact with blood, bodily fluids, or contaminated materials from infected individuals or animals. Healthcare workers are especially vulnerable due to exposure to contaminated medical instruments or unprotected contact with patients [9–12].

Transmission occurs mainly through contact with infected blood or secretions via broken skin or mucous membranes. Infectious fluids include saliva, urine, sweat, semen, feces, vomit, and breast milk. The virus can persist in immune-privileged sites such as the eyes and testes of survivors, and in placental tissues and breast milk of pregnant individuals [4,19]. Traditional

burial practices involving contact with deceased infected bodies also contribute to virus spread.

Clinically, MVD has an incubation period of 2 to 21 days, followed by sudden onset of fever, headache, muscle pain, and malaise. The disease progresses through three phases, including an initial flu-like phase; an early organ phase with respiratory symptoms, rash, and neurological signs; and a late organ phase featuring hemorrhagic symptoms, multi-organ failure, and frequently death [3, 9]. Survivors often endure prolonged fatigue and neurological complications.

The basic reproduction number, R_0 , is a critical epidemiological parameter indicating the average number of secondary infections caused by one infected individual in a fully susceptible population. For Marburg virus, R_0 quantifies contagiousness and outbreak potential. Values above 1 suggest sustained transmission and epidemic risk, whereas values below 1 indicate likely outbreak decline. Containment efforts focus on reducing R_0 below 1 through quarantine, vaccination, or public health interventions [13]. Understanding R_0 informs intervention and preparedness strategies during outbreaks.

Optimal control of MVD involves both non-pharmaceutical and pharmaceutical measures. Non-pharmaceutical interventions (NPIs), such as social distancing, quarantine, and travel restrictions, are essential when vaccines or antiviral treatments are unavailable. These measures reduce transmission and allow health systems to respond effectively. Pharmaceutical interventions include supportive care, including hydration, oxygen therapy, and symptom relief, as well as experimental antivirals like remdesivir and favipiravir, alongside monoclonal antibody therapies under development [14, 15]. Vaccine development faces challenges including efficacy and distribution logistics, necessitating continued research.

In areas where vectors such as mosquitoes, ticks, or rodents contribute to transmission, vector control measures, such as insecticide-treated nets, indoor residual spraying, and environmental management, are crucial for reducing vector populations and interrupting transmission. Behavioral interventions, including risk communication, community engagement, and public awareness campaigns, foster trust, compliance, and timely healthcare seeking. Adapting these strategies to local cultural contexts improves their effectiveness and sustainability.

Since the initial outbreaks linked to African green monkeys imported from Uganda, sporadic cases and outbreaks have mainly occurred in sub-Saharan Africa, including Angola, Democratic Republic of Congo, Uganda, Kenya, South Africa, Zimbabwe, Guinea, and Ghana [4, 16, 17]. Surveillance, rapid diagnosis, infection control, and public health preparedness remain vital to minimizing MVD's impact. Marburg virus disease is a rare but highly lethal zoonosis with epidemic potential. The absence of approved

treatments or vaccines and the virus's complex transmission dynamics require a multifaceted approach combining epidemiological insight, effective control measures, and community participation. Ongoing research and global collaboration are essential for improving outbreak responses, developing therapies, and achieving long-term disease control.

II. METHODS

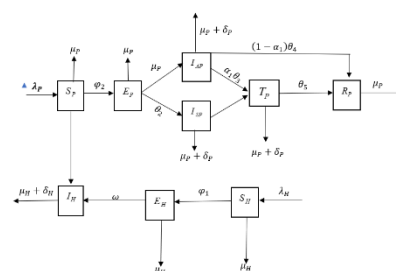
Compartmental Models

Mathematical models are essential for understanding the spread of infectious diseases. Compartmental models like the SIR (Susceptible-Infectious-Recovered) and SEIR (Susceptible-Exposed-Infectious-Recovered) models divide the population into compartments based on disease status and simulate transitions between them.

The SIR model assumes that individuals immediately become infectious upon exposure. It is well-suited for diseases like Marburg virus disease (MVD), which lacks a significant latent period. In this model, susceptible individuals become infected and later recover with immunity. Diekmann et al. emphasized the role of the basic reproduction number R_0 in the SIR model and its value in understanding immediate infectiousness and controlling rapid outbreaks [10]. Lipsitch et al. applied SIR models to influenza, offering insights into rapid transmission and intervention strategies that are also relevant to MVD [11].

The SEIR model extends the SIR framework by including an Exposed (E) compartment to account for the incubation period. Individuals move from the Exposed phase to the Infectious phase before recovery, making this model more appropriate for diseases with a latent phase. Li et al. demonstrated the usefulness of SEIR in modeling COVID-19, highlighting the importance of capturing incubation periods for better intervention assessment [15]. Lewnard et al. also used the SEIR model to analyze Ebola transmission in Liberia, providing insights into how latency influences outbreak dynamics; findings applicable to Marburg virus as well [16, 20].

These compartmental models inform preparedness, guide interventions, and help estimate key parameters such as R_0 contributing to effective outbreak response strategies.



Model of Equations

Considering the above schematic diagram, we obtain the following system of differential equations:

$$\frac{dS_P}{dt} = \lambda_p - (\varphi_2 + \mu_p)S_p$$

(1)

$$\frac{dE_P}{dt} = \varphi_2 S_p - (\theta_1 + \theta_2 + \mu_p)E_p$$

(2)

$$\frac{dI_{AP}}{dt} = \theta_1 E_p - (\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)I_{AP}$$

(3)

$$\frac{dI_{SP}}{dt} = \theta_2 E_p - (\mu_p + \delta_p + \alpha_2)I_{SP}$$

(4)

$$\frac{dT_P}{dt} = \alpha_1\theta_3 I_{AP} + \alpha_2 I_{SP} - (\mu_p + \delta_p + \theta_5)T_P$$

(5)

$$\frac{dR_P}{dt} = \theta_5 T_P + (1 - \alpha_1)\theta_4 I_{AP} - \mu_p R_P$$

(6)

$$\frac{dS_H}{dt} = \lambda_H - (\varphi_1 + \mu_H)S_H$$

(7)

$$\frac{dE_H}{dt} = \varphi_1 S_H - (\omega + \mu_H)E_H$$

(8)

$$\frac{dI_H}{dt} = \omega E_H - (\mu_H + \delta_H)I_H$$

(9)

Where $\varphi_1 = \beta_H I_H$, and $\varphi_2 = \beta_{P1} I_H + \beta_{P2} (I_{AP} + I_{SP})$

With initial condition $S_P(0) = S_{P0}$, $E_P(0) =$

E_{P0} , $I_{AP}(0) = I_{AP0}$, $I_{SP}(0) = I_{SP0}$, $T_P(0) = T_{P0}$,

$R_P(0) = R_{P0}$, $S_H(0) = S_{H0}$, $E_H(0) = E_{H0}$, $I_H(0) = I_{H0}$.

Table 1: Variables Description

Variables	Description
S_P	The susceptible human population
E_P	The exposed human population
I_{AP}	Asymptomatic infected human population
I_{SP}	Symptomatic infection human population
T_P	Treatment population
R_P	Recovered human population
S_H	The susceptible host population
E_H	The exposed host population
I_H	The infected host population
θ_1	Transition rate of exposed to asymptomatic
θ_2	Transition rate of exposed to symptomatic
θ_3	Transition rate of I_{AP} to T_P
θ_4	Transition rate of I_{AP} to β_P
θ_5	Recovery rate of T_P
α_1	Transition rate of asymptomatic to treatment
α_2	Transition rate of symptomatic to treatment
λ_p	Recruitment rate for human population
λ_H	Recruitment rate for host population

δ_P	Disease induced death rate for human population
δ_H	Disease induced death rate for host
μ_p	Natural death rate for human population
μ_H	Natural death rate for human population
φ_1	Force of infection for Host population
φ_2	Force of infection for Human population
ω	The rate at which exposed E_H to I_H
α_2	The rate at which symptomatic human get treated
β_H	Contact rate of Host to Host
β_{P1}	Contact rate of Host to Human population
β_{P2}	Contact rate of Human-to-Human population
I_H	Probability of MARV transmission by infected host

Let $D = \{(S_P, E_P, I_{AP}, I_{SP}, T_P, R_P, S_H, E_H, I_H) \in \mathbb{R}_+^9 :$

$N_P \leq N_P^{max}, N_H \leq N_H^{max}\}$, where N_P is the total population of human, $N_P = S_P + E_P + I_{AP} + I_{SP} + T_P + R_P$ and N_H is the total population size of host, $N_H = S_H + E_H + I_H$.

The disease-free equilibrium for the Marburg Virus Disease Model is given by:

$$E_0 = \left(\frac{\lambda_p}{\mu_p}, 0, 0, 0, 0, 0, \frac{\lambda_H}{\mu_H}, 0, 0 \right). \quad (10)$$

Positivity of Solution

Since model (1) to (9) describes the dynamics of both human and vector populations, all parameters are nonnegative. Therefore, it is necessary to demonstrate that all state variables remain non-negative for all $t \geq 0$.

Theorem 1: Assume the initial conditions satisfy, $S_P(0) \geq 0, E_P(0) \geq 0, I_{AP}(0) \geq 0, I_{SP}(0) \geq 0, T_P(0) \geq 0, R_P(0) \geq 0, S_H(0) \geq 0, E_H(0) \geq 0, I_H(0) \geq 0$ with $(S_P(0), E_P(0), \dots, I_H(0)) \in D$. Then solution set $\{S_P(t), E_P(t), I_{AP}(t), I_{SP}(t), T_P(t), R_P(t), S_H(t), E_H(t), I_H(t)\}$ remains non-negative for all $t \geq 0$.

Proof: To show $S_P(t) > 0$, we first remove the nonnegative term λ_p from equation (1), giving $\frac{dS_P}{dt} \geq -(\varphi_2 + \mu_p)S_p$, this leads to the linear equation $\frac{dS_P}{dt} + (\varphi_2 + \mu_p)S_p = 0$, and then solving by integrating factor method, $S_P(t) = S_{P0} e^{-\int (\varphi_2 + \mu_p) dt}$, $S_{P0} \geq 0$. Since the exponential is always positive, it follows that $S_P(t) > 0, \forall t \geq 0$.

We proceed recursively using this approach and apply it to equations (2) through (9), each ensuring that the corresponding state variable remains positive for all $t \geq 0$.

→ Equation (2):

$$E_P(t) = E_{P0} e^{-\int (\theta_1 + \theta_2 + \mu_p) dt}, \quad E_P(t) > 0, \forall t \geq 0. \quad (11)$$

→ Equation (3):

$$\begin{aligned}
 I_{AP}(t) &= I_{AP0} e^{-\int(\mu_p + \delta_p + (1-\alpha_1)\theta_4 + \alpha_1\theta_3)dt}, \\
 I_{AP}(t) &> 0, \forall t \geq 0. \quad (12) \\
 \rightarrow \text{Equation (4):} \\
 I_{SP}(t) &= I_{SP0} e^{-\int(\mu_p + \delta_p + \alpha_2)dt}, \quad I_{SP}(t) > 0, \forall t \geq 0. \quad (13) \\
 \rightarrow \text{Equation (5):} \\
 T_p(t) &= T_{p0} e^{-\int(\mu_p + \delta_p + \theta_5)dt}, \quad T_p(t) > 0, \forall t \geq 0. \quad (14) \\
 \rightarrow \text{Equation (6):} \\
 R_p(t) &= R_{p0} e^{-\int \mu_p dt}, \quad R_p(t) > 0, \forall t \geq 0. \quad (15) \\
 \rightarrow \text{Equation (7):} \\
 S_H(t) &= S_{H0} e^{-\int(\varphi_1 + \mu_H)dt}, \quad S_H(t) > 0, \forall t \geq 0. \quad (14) \\
 \rightarrow \text{Equation (8):} \\
 E_H(t) &= E_{H0} e^{-\int(\omega + \mu_H)dt}, \quad E_H(t) > 0, \forall t \geq 0. \quad (15) \\
 \rightarrow \text{Equation (9):} \\
 I_H(t) &= I_{H0} e^{-\int(\mu_H + \delta_H)dt}, \quad I_H(t) > 0, \forall t \geq 0. \quad (16)
 \end{aligned}$$

These results show that the solution set $\{S_p, E_p, I_{AP}, I_{SP}, T_p, R_p, S_H, E_H, I_H\}$ of the system of equation (1) – (9) is positive for all $t \geq 0$.

Boundedness of solution

The differential equation for *human population* $N_p = S_p + E_p + I_{AP} + I_{SP} + T_p + R_p$ is given by

$$\frac{dN_p}{dt} = \frac{dS_p}{dt} + \frac{dE_p}{dt} + \frac{dI_{AP}}{dt} + \frac{dI_{SP}}{dt} + \frac{dT_p}{dt} + \frac{dR_p}{dt} \quad (17)$$

Including all compartments, equation (17) becomes

$$\begin{aligned}
 \frac{dN_p}{dt} &= \lambda_p - \varphi_2 S_p - \mu_p S_p + \varphi_2 S_p - \theta_1 E_p - \theta_2 E_p - \\
 &\mu_p E_p + \theta_1 E_p - \mu_p I_{AP} + \delta_p I_{AP} - \theta_4 I_{AP} + \alpha_1 \theta_4 I_{AP} - \\
 &\alpha_1 \theta_3 I_{AP} + \theta_2 E_p - \mu_p I_{SP} - \delta_p I_{SP} - \alpha_2 I_{SP} + \alpha_1 \theta_3 I_A + \\
 &\alpha_2 I_S - \mu_p T_p - \delta_p T_p - \theta_5 T_p + \theta_5 T_p + \theta_4 I_A - \alpha_1 \theta_4 I_A - \\
 &\mu_p R.
 \end{aligned}$$

Upon simplifying by canceling internal transition terms, we obtain:

$$\frac{dN_p}{dt} = \lambda_p - \mu_p (S_p + E_p + I_{AP} + I_{SP} + T_p + R_p) - \delta_p (I_{AP} + I_{SP} + T_p) \quad (17)$$

This expression shows that the total primate population increases by the recruitment rate λ_p , and decreases due to natural deaths (μ_p) from all compartments and disease-induced deaths (δ_p) from infected and treated individuals. In summary, $\frac{dN_p}{dt} = \lambda_p - \mu_p (\text{total population}) - \delta (\text{infected} + \text{treated})$. Taking $\delta_p = 0$, $\frac{dN_p}{dt} = \lambda_p - \mu_p (S_p + E_p + I_{AP} + I_{SP} + T_p + R_p) = \lambda_p - \mu_p N_p$, solving this using integrating factor method, we obtained, $N_p(t) = \frac{\lambda_p}{\mu_p} (1 - e^{-\mu_p t}) + N_0 e^{-\mu_p t}$. Hence $N_p(t) = \frac{\lambda_p}{\mu_p}$, at $t \rightarrow \infty$.

The differential equation for *host population* $N_H = S_H + E_H + I_H$ is given by

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} \quad (18)$$

Then, $\frac{dN_H}{dt} = \lambda_H - \varphi_1 S_H - \mu_H S_H + \varphi_1 S_H - \omega E_H - \mu_H E_H + \omega E_H - \mu_H I_H - \delta_H I_H \lambda_H - \mu_H (S_H + E_H + I_H) - \delta_H I_H$. Taking, $\delta_H = 0$, we have, $N_H(t) = \lambda_H - \mu_H (S_H + E_H + I_H) = \lambda_H - \mu_H N_H$, which has the solution $\frac{\lambda_H}{\mu_H} (1 - e^{-\mu_H t}) + N_0 e^{-\mu_H t}$. Therefore $N_H(t) = \frac{\lambda_H}{\mu_H}$ at $t \rightarrow \infty$.

Hence, all feasible solutions of the model (1) – (9), enter the region

$$\begin{aligned}
 \Omega = \{ &(S_p, E_p, I_{AP}, I_{SP}, T_p, R_p, S_H, E_H, I_H) \in \mathbb{R}_+^9 \\
 &: S_p + E_p + I_{AP} + I_{SP} + T_p + R_p \\
 &+ S_H + E_H + I_H \leq N, N \leq \frac{\lambda}{\mu} \}.
 \end{aligned}$$

This implies that all the state variables remain non-negative and bounded for all $t \geq 0$. Therefore, the region Ω is positively invariant, and the model (1) – (9) is epidemiologically meaningful and well-posed in the domain D . Hence, the population remains bounded over time.

Control Reproduction Number (R_n)

The control reproduction number R_n quantifies the expected number of secondary infections in the presence of control measures (e.g. treatment, vector control, or immunity).

It is derived using the next generation matrix method: $R_n = \rho(FV^{-1})$, where F is the matrix of new infection terms, V is the matrix of transitions between compartments and ρ denotes the spectral radius (dominant eigenvalue).

Following Diekmann and Heesterbeek (2000), both natural and disease-induced deaths are included in V for simplicity and to ensure it is invertible: In this model: $R_n^2 = R_{p0} \times R_{H0}$, where R_{p0} is the reproduction number for the human population, and R_{H0} is the reproduction number for the host population [18].

The subsystem below is three dimensional, hence transmission and transition matrices are also in three dimensions. To derive the control reproduction number, we separate the transmission events from all other transitions in the model. The infected compartments in the human population are governed by the following system of differential equations:

By separating the transmission event from other events, we get:

$$\begin{aligned}
 \frac{dE_p}{dt} &= \varphi_2 S_p - (\theta_1 + \theta_2 + \mu_p) E_p \\
 \frac{dI_{AP}}{dt} &= \theta_1 E_p - (\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3) I_{AP} \\
 \frac{dI_{SP}}{dt} &= \theta_2 E_p - (\mu_p + \delta_p + \alpha_2) I_{SP}
 \end{aligned}$$

$$\frac{dT_P}{dt} = \alpha_1 \theta_3 I_{AP} + \alpha_2 I_{SP} - (\mu_p + \delta_p + \theta_5) T_P$$

Here, the force of infection for the human population is given by: $\varphi_2 = \beta_{P1} I_H + \beta_{P2} (I_{AP} + I_{SP})$.

Then, the rate of appearance of new infections in each infected compartment is given as

$$f = \begin{bmatrix} T_1 \\ T_2 \\ T_1 \\ T_2 \end{bmatrix} = \begin{bmatrix} \varphi_2 S_p \\ 0 \\ 0 \\ 0 \end{bmatrix}; \quad \text{and} \quad \text{the non-infectious}$$

transitions between infected compartment is

$$v = \begin{bmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{bmatrix} = \begin{bmatrix} (\theta_1 + \theta_2 + \mu_p) E_p \\ -\theta_1 E_p + (\mu_p + \delta_p + (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3) I_{AP} \\ -\theta_2 E_p + (\mu_p + \delta_p + \alpha_2) I_{SP} \\ -\alpha_1 \theta_3 I_{AP} - \alpha_2 I_{SP} + (\mu_p + \delta_p + \theta_5) T_P \end{bmatrix}$$

Computing the Jacobian of f and v give F and V respectively. Therefore, we have,

$$F = \begin{bmatrix} 0 & \beta_{P2} S_p & \beta_{P2} S_p & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} a & 0 & 0 & 0 \\ -\theta_1 & b & 0 & 0 \\ -\theta_2 & 0 & c & 0 \\ 0 & -\alpha_1 \theta_3 & -\alpha_2 & d \end{bmatrix}, \text{ where } a = (\theta_1 + \theta_2 + \mu_p),$$

$$b = (\mu_p + \delta_p + (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3); c = (\mu_p + \delta_p + \alpha_2); d = (\mu_p + \delta_p + \theta_5).$$

$$F = \begin{bmatrix} 0 & \beta_{P2} S_p & \beta_{P2} S_p & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V^{-1} = \begin{bmatrix} \frac{1}{a} & 0 & 0 & 0 \\ \frac{\theta_1}{ab} & \frac{1}{b} & 0 & 0 \\ -\frac{\theta_2}{ac} & 0 & \frac{1}{c} & 0 \\ \frac{\alpha_1 \theta_3 \theta_1 c + \alpha_2 \theta_2 b}{abcd} & \frac{\alpha_1 \theta_3}{bd} & \frac{\alpha_2}{cd} & \frac{1}{d} \end{bmatrix}$$

$$\text{Hence, } FV^{-1} = \begin{bmatrix} \frac{\beta_{P2} \theta_1 S_p}{ab} + \frac{\beta_{P2} \theta_1 S_p \theta_2}{ac} & \frac{\beta_{P2} S_p}{b} & \frac{\beta_{P2} S_p}{c} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Therefore, we have the reproduction number for human class to be,

$$R_{P0} = \frac{\beta_{P2} \lambda_P}{\mu_P (\theta_1 + \theta_2 + \mu_P)} \left(\frac{\theta_1}{(\mu_p + \delta_p + (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3)} + \frac{\theta_2}{(\mu_p + \delta_p + \alpha_2)} \right) \quad (19)$$

The infected classes for the reservoir population are: E_H and I_H . The models are given by:

$$\frac{dE_H}{dt} = \varphi_1 S_H - (\omega + \mu_H) E_H \text{ and } \frac{dI_H}{dt} = \omega E_H - (\mu_H + \delta_H) I_H.$$

$$\text{Then, } f = \begin{bmatrix} X_1 \\ X_2 \end{bmatrix} = \begin{bmatrix} \varphi_1 S_H \\ 0 \end{bmatrix} \quad \text{and} \quad v = \begin{bmatrix} Y_1 \\ Y_2 \end{bmatrix} = \begin{bmatrix} (\omega + \mu_H) E_H \\ -\omega E_H + (\mu_H + \delta_H) I_H \end{bmatrix},$$

$$\text{so } F = \begin{bmatrix} 0 & \beta_H S_H \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} (\omega + \mu_H) & 0 \\ -\omega & (\mu_H + \delta_H) \end{bmatrix} \quad \text{and}$$

$$FV^{-1} = \begin{bmatrix} \frac{\omega \beta_H \lambda_H}{(\omega + \mu_H)(\mu_H + \delta_H) \mu_H} & \frac{\beta_H \lambda_H}{(\mu_H + \delta_H) \mu_H} \\ 0 & 0 \end{bmatrix}.$$

$$\text{Therefore, } R_{H0} = \frac{\omega \beta_H \lambda_H}{(\omega + \mu_H)(\mu_H + \delta_H) \mu_H}.$$

$$\text{Hence, } R_n^2 = R_{PO} \times R_{H0} =$$

$$\frac{\beta_{P2} \lambda_P}{\mu_P (\theta_1 + \theta_2 + \mu_P)} \left(\frac{\theta_1}{(\mu_p + \delta_p + (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3)} + \frac{\theta_2}{(\mu_p + \delta_p + \alpha_2)} \right) \frac{\omega \beta_H \lambda_H}{(\omega + \mu_H)(\mu_H + \delta_H) \mu_H}.$$

Local and Global Stability

Theorem 2: Let E_0 denotes the disease-free equilibrium of the Marburg virus transmission model given by (1) – (9), then, the disease-free equilibrium, E_0 is locally asymptotically stable whenever the basis reproduction number $R_0 < 1$.

Proof: To assess the local asymptotic stability of the disease-free equilibrium E_0 , we linearize the nonlinear system around E_0 and evaluate the Jacobian matrix $J(E_0)$. The model equations are:

$$\begin{aligned} F_1 &= \lambda_p - (\varphi_2 + \mu_p) S_p \\ F_2 &= \varphi_2 S_p - (\theta_1 + \theta_2 + \mu_p) E_p \\ F_3 &= \theta_1 E_p - (\mu_p + \delta_p + (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3) I_{AP} \\ F_4 &= \theta_2 E_p - (\mu_p + \delta_p + \alpha_2) I_{SP} \\ F_5 &= \alpha_1 \theta_3 I_{AP} + \alpha_2 I_{SP} - (\mu_p + \delta_p + \theta_5) T_P \\ F_6 &= \theta_5 T_P + (1 - \alpha_1) \theta_4 I_{AP} - \mu_P R_p \\ F_7 &= \lambda_H - (\varphi_1 + \mu_H) S_H \\ F_8 &= \varphi_1 S_H - (\omega + \mu_H) E_H \\ F_9 &= \omega E_H - (\mu_H + \delta_H) I_H \end{aligned}$$

At steady state, the Jacobian $F_1, F_2, F_3, F_4, F_5, F_6, F_7, F_8, F_9$ with respect to $S_p, E_p, I_{AP}, I_{SP}, T_p, R_p, S_H, E_H, I_H$ evaluated to the endemic equilibrium point is given by the matrix below:

$$J = \begin{bmatrix} -\mu_P & 0 & -\beta_{P2} S_p & -\beta_{P2} S_p & 0 & 0 & 0 & 0 & \beta_{P1} S_p \\ 0 & -a & \beta_{P2} S_p & \beta_{P2} S_p & 0 & 0 & 0 & 0 & \beta_{P1} S_p \\ 0 & \theta_1 & -b & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \theta_2 & 0 & -c & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 \theta_3 & \alpha_2 & -d & 0 & 0 & 0 & 0 \\ 0 & 0 & (1 - \alpha_1) \theta_4 & 0 & \theta_5 & -\mu_P & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_H & 0 & -\beta_H S_H \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\omega + \mu_H) & \beta_H S_H \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega & -(\mu_H + \delta_H) \end{bmatrix}$$

$$\text{Since } |J(E_0) - \lambda I| = 0$$

$$\begin{bmatrix} -\mu_p - \lambda & 0 & -\beta_{P2}S_p & -\beta_{P2}S_p & 0 & 0 & 0 & 0 & -\beta_{P1}S_p \\ 0 & -a - \lambda & \beta_{P2}S_p & \beta_{P2}S_p & 0 & 0 & 0 & 0 & \beta_{P1}S_p \\ 0 & \theta_1 & -b - \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \theta_2 & 0 & -c - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_1\theta_3 & \alpha_2 & -d - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & (1 - \alpha_1)\theta_4 & 0 & \theta_5 & -\mu_p - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_H - \lambda & 0 & -\beta_H S_H \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\omega + \mu_H) - \lambda & \beta_H S_H \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega & -(\mu_H + \delta_H) - \lambda \end{bmatrix} = 0.$$

The eigenvalue analysis of the computed Jacobian matrix reveals that all eigenvalues possess negative real parts given as,

$$\lambda_1 = -\mu_p, \lambda_2 = -\mu_H, \lambda_3 = -\mu_H, \lambda_4 = -(\mu_p + \delta_p + \theta_5), \lambda_5 = -(\theta_1 + \theta_2 + \mu_p),$$

$$\lambda_6 = \frac{-ba + \theta_1\beta_{P2}S_p}{a}, \lambda_7 = -\frac{-cab + c\theta_1\beta_{P2}S_p + b\theta_2\beta_{P2}S_p}{\theta_1\beta_{P2}S_p - ba},$$

$$\lambda_8 = -(\omega + \mu_H),$$

$$\text{and } \lambda_9 = \frac{-\mu_H\omega - \mu_H^2 - \delta_H\omega - \delta_H\mu_H + \omega\beta_H S_H}{\omega + \mu_H}, \text{ where } a =$$

$$(\theta_1 + \theta_2 + \mu_p); b = (\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3); c = (\mu_p + \delta_p + \alpha_2); d = (\mu_p + \delta_p + \theta_5).$$

This shows that E_0 is locally asymptotically stable whenever the basis reproduction number $R_0 < 1$.

Lemma 1: The disease-free equilibrium of both the human and reservoir populations is asymptotically stable when $R_{p0} < 1$ and $R_{H0} < 1$. The eigenvalues derived in

Theorem 2 can be expressed in factored form as follows:

$$\lambda_9 = \frac{-\mu_H\omega - \mu_H^2 - \delta_H\omega - \delta_H\mu_H + \omega\beta_H S_H}{\omega + \mu_H}$$

$$\lambda_7 = -\frac{-cab + c\theta_1\beta_{P2}S_p + b\theta_2\beta_{P2}S_p}{\theta_1\beta_{P2}S_p - ba},$$

Simplifying this, we obtained:

$$\lambda_9 = -(\mu_H + \delta_H) \left(1 - \frac{\omega\beta_H S_H}{(\omega + \mu_H)(\mu_H + \delta_H)} \right) \leq -(\mu_H + \delta_H)(1 - R_{H0}). \quad (20)$$

$$\lambda_7 = -\mu_p(\theta_1 + \theta_2 + \mu_p) \left(1 - \right.$$

$$\left. \frac{\beta_{P2}\lambda_p}{\mu_p(\theta_1 + \theta_2 + \mu_p)} \left(\frac{\theta_1}{(\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)} + \frac{\theta_2}{(\mu_p + \delta_p + \alpha_2)} \right) \right) \leq$$

$$\mu_p(\theta_1 + \theta_2 + \mu_p)(1 - R_{p0}). \quad (21)$$

The above inequalities (20) and (21) hold and remain negative (stable) if and only if $R_{p0} < 1$ and $R_{H0} < 1$.

Theorem 3: The disease-free equilibrium is locally asymptotically stable when $R_n < 1$.

Proof: The result follows from the *Lemma* above. Since $R_{H0} < 1$ and $R_{p0} < 1$ holds. Then, $R_{p0}R_{H0} < 1 \Rightarrow R_n^2 = R_{p0}R_{H0} < 1, \therefore R_n < 1$ holds.

Theorem 4: The disease-free equilibrium E_0 of the model (1) – (9) is globally asymptotically stable whenever the threshold parameter M^0 satisfies $M^0 > 1$.

Proof: To demonstrate the global stability of the equilibrium E^0 , we define the following Lyapunov function based on the established approach:

$$M(S_p, E_p, I_{AP}, I_{SP}, T_p, R_p, S_H, E_H, I_H) = (S_p - S_p^0 - S_p^0 \log \frac{S_p}{S_p^0}) + (E_p - E_p^0 - E_p^0 \log \frac{E_p}{E_p^0}) + (I_{AP} - I_{AP}^0 - I_{AP}^0 \log \frac{I_{AP}}{I_{AP}^0}) + (I_{SP} - I_{SP}^0 - I_{SP}^0 \log \frac{I_{SP}}{I_{SP}^0}) + (R_p - R_p^0 - R_p^0 \log \frac{R_p}{R_p^0}) + (S_H - S_H^0 - S_H^0 \log \frac{S_H}{S_H^0}) + (E_H - E_H^0 - E_H^0 \log \frac{E_H}{E_H^0}) + (I_H - I_H^0 - I_H^0 \log \frac{I_H}{I_H^0}). \quad (22)$$

By directly differentiating M along the solution trajectory of system (1) – (9), we obtain:

$$\begin{aligned} \frac{dM}{dt} = & \left(1 - \frac{S_p^0}{S_p}\right) \lambda_p + \left(1 - \frac{E_p^0}{E_p}\right) \varphi_2 S_p + \left(1 - \frac{I_{AP}^0}{I_{AP}}\right) \theta_1 E_p + \\ & \left(1 - \frac{I_{SP}^0}{I_{SP}}\right) \theta_1 E_p + \left(1 - \frac{T_p^0}{T_p}\right) (\alpha_1 \theta_3 I_{AP} + \alpha_2 I_{SP}) + \\ & \left(1 - \frac{R_p^0}{R_p}\right) (\theta_3 T_p + (1 - \alpha_1) \theta_4 I_{AP}) + \left(1 - \frac{S_H^0}{S_H}\right) \lambda_H + \\ & \left(1 - \frac{E_p^0}{E_p}\right) \varphi_1 S_p + \left(1 - \frac{I_H^0}{I_H}\right) \omega E_H + \frac{(S_p - S_p^0)^2}{S_p} (\varphi_2 + \mu_p) + \\ & \frac{(E_p - E_p^0)^2}{E_p} (\theta_1 + \theta_1 + \mu_p) + \frac{(I_{AP} - I_{AP}^0)^2}{I_{AP}} (\mu_p + \delta_p + \\ & (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3) + \frac{(I_{SP} - I_{SP}^0)^2}{I_{SP}} (\mu_p + \delta_p + \alpha_2) + \\ & \frac{(T_p - T_p^0)^2}{T_p} (\mu_p + \delta_p + \theta_5) + \frac{(R_H - R_H^0)^2}{R_H} (\mu_H) + \\ & \frac{(S_H - S_H^0)^2}{S_H} (\varphi_1 + \mu_H) + \frac{(E_H - E_H^0)^2}{E_H} (\omega + \mu_H) + \\ & \frac{(I_H - I_H^0)^2}{I_H} (\mu_H + \delta_H). \end{aligned}$$

Let $\frac{dM}{dt} = S - E$, then

$$\begin{aligned} S = & \left(1 - \frac{S_p^0}{S_p}\right) \lambda_p + \left(1 - \frac{E_p^0}{E_p}\right) \varphi_2 S_p + \left(1 - \frac{I_{AP}^0}{I_{AP}}\right) \theta_1 E_p + \\ & \left(1 - \frac{I_{SP}^0}{I_{SP}}\right) \theta_1 E_p + \left(1 - \frac{T_p^0}{T_p}\right) (\alpha_1 \theta_3 I_{AP} + \alpha_2 I_{SP}) + \\ & \left(1 - \frac{R_p^0}{R_p}\right) (\theta_3 T_p + (1 - \alpha_1) \theta_4 I_{AP}) + \left(1 - \frac{S_H^0}{S_H}\right) \lambda_H + \\ & \left(1 - \frac{E_p^0}{E_p}\right) \varphi_1 S_p + \left(1 - \frac{I_H^0}{I_H}\right) \omega E_H. \end{aligned}$$

Similarly,

$$\begin{aligned} E = & \frac{(S_p - S_p^0)^2}{S_p} (\varphi_2 + \mu_p) + \frac{(E_p - E_p^0)^2}{E_p} (\theta_1 + \theta_1 + \mu_p) + \\ & \frac{(I_{AP} - I_{AP}^0)^2}{I_{AP}} (\mu_p + \delta_p + (1 - \alpha_1) \theta_4 + \alpha_1 \theta_3) + \\ & \frac{(I_{SP} - I_{SP}^0)^2}{I_{SP}} (\mu_p + \delta_p + \alpha_2) + \frac{(T_p - T_p^0)^2}{T_p} (\mu_p + \delta_p + \theta_5) + \\ & \frac{(R_H - R_H^0)^2}{R_H} (\mu_H) + \frac{(S_H - S_H^0)^2}{S_H} (\varphi_1 + \mu_H) + \frac{(E_H - E_H^0)^2}{E_H} (\omega + \\ & \mu_H) + \frac{(I_H - I_H^0)^2}{I_H} (\mu_H + \delta_H). \end{aligned}$$

If $S < E$, then $\frac{dM}{dt}$ becomes negative definite along the solution trajectory of the system. Consequently, $\frac{dM}{dt} \leq 0$ holds only at the disease-free equilibrium E_0 , which implies that the system is globally asymptotically stable at the Marburg Virus disease-free equilibrium.

Existence of the Endemic Equilibrium Points

The endemic equilibrium points are defined as $(S_P^*(t), 0, 0, 0, S_H^*(t), 0, 0)$ satisfying $S_P' = E_P' = I_{AP}' = I_{SP}' = T_P' = R_H' = S_H' = E_H' = I_H' = 0$, by (1) – (9) to 0, we have

$$\begin{aligned} \lambda_p - (\varphi_2 + \mu_p)S_P^* &= 0 \\ \varphi_2 S_P^* - (\theta_1 + \theta_2 + \mu_p)E_P^* &= 0 \\ \theta_1 E_P^* - (\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)I_{AP}^* &= 0 \\ \theta_2 E_P^* - (\mu_p + \delta_p + \alpha_2)I_{SP}^* &= 0 \\ \alpha_1\theta_3 I_{AP}^* + \alpha_2 I_{SP}^* - (\mu_p + \delta_p + \theta_5)T_P^* &= 0 \end{aligned} \quad (23)$$

$$\theta_5 T_P^* + (1 - \alpha_1)\theta_4 I_{AP}^* - \mu_p R_P^* = 0$$

$$\lambda_H - (\varphi_1 + \mu_H)S_H^* = 0$$

$$\varphi_1 S_H^* - (\omega + \mu_H)E_H^* = 0$$

$$\omega E_H^* - (\mu_H + \delta_H)I_H^* = 0.$$

Simplifying these equations, we have

$$\begin{aligned} S_P^* &= \frac{\lambda_p}{(\varphi_2 + \mu_p)}, \\ E_P^* &= \frac{\varphi_2 \lambda_p}{(\theta_1 + \theta_2 + \mu_p)(\varphi_2 + \mu_p)}, \\ I_{AP}^* &= \frac{\theta_1 \varphi_2 \lambda_p}{(\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)(\theta_1 + \theta_2 + \mu_p)(\varphi_2 + \mu_p)}, \\ I_{SP}^* &= \frac{\theta_2 \varphi_2 \lambda_p}{(\mu_p + \delta_p + \alpha_2)(\theta_1 + \theta_2 + \mu_p)(\varphi_2 + \mu_p)}, \\ T_P^* &= \frac{\varphi_2 \lambda_p}{(\mu_p + \delta_p + \theta_5)(\theta_1 + \theta_2 + \mu_p)(\varphi_2 + \mu_p)} \left(\frac{\alpha_1 \theta_3 \theta_1}{(\mu_p + \delta_p + \theta_5)(\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)} + \frac{\alpha_2 \theta_2}{(\mu_p + \delta_p + \alpha_2)} \right) + \\ R_P^* &= \frac{\varphi_2 \lambda_p}{\mu_p(\theta_1 + \theta_2 + \mu_p)(\varphi_2 + \mu_p)} \left[\frac{\theta_5}{(\mu_p + \delta_p + \theta_5)} \left(\frac{\alpha_1 \theta_3 \theta_1}{(\mu_p + \delta_p + \theta_5)(\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)} + \frac{\alpha_2 \theta_2}{(\mu_p + \delta_p + \alpha_2)} \right) + \frac{(1 - \alpha_1)\theta_4 \theta_1}{(\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)} \right] \end{aligned} \quad (24)$$

$$S_H^* = \frac{\lambda_H}{(\varphi_1 + \mu_H)}.$$

$$E_H^* = \frac{\varphi_1 \lambda_H}{(\omega + \mu_H)(\varphi_1 + \mu_H)}.$$

$$I_H^* = \frac{\omega \varphi_1 \lambda_H}{(\mu_H + \delta_H)(\omega + \mu_H)(\varphi_1 + \mu_H)}.$$

Using the established approach, the model is endemic and disease-free E^0 (which existed only when $R_n > 1$) equilibrium points are established.

Sensitivity analysis of the Marburg Virus Disease model

We analyzed the reproduction number $R_n = \sqrt{R_{P_0} R_{H_0}}$ to examine how variations in specific parameters influence its value when increased or decreased.

The *normalized forward* sensitivity index of a variable R , which depends differentiability on a parameter on a parameter U , is defined as:

$$X_U^R = \frac{\partial R}{\partial U} \cdot \frac{U}{R}$$

We now compute the sensitivity indices of the basic reproduction number R_n with respect to model parameters.

The normalized forward sensitivity index for β_{P_2} is given by:

$$X_{\beta_{P_2}}^{R_n} = \frac{\partial R_n}{\partial \beta_{P_2}} \cdot \frac{\beta_{P_2}}{R_n}$$

Where, $R^2 = \frac{\beta_{P_2} \lambda_P}{\mu_P(\theta_1 + \theta_2 + \mu_P)} \left(\frac{\theta_1}{(\mu_p + \delta_p + (1 - \alpha_1)\theta_4 + \alpha_1\theta_3)} + \frac{\theta_2}{(\mu_p + \delta_p + \alpha_2)} \right) \frac{\omega \beta_H \lambda_H}{(\omega + \mu_H)(\mu_H + \delta_H)\mu_H}$, and $\frac{\partial R_n}{\partial \beta_{P_2}} = \frac{1}{2\beta_{P_2}} R_n$. Therefore $X_{\beta_{P_2}}^{R_n} = \frac{\partial R_n}{\partial \beta_{P_2}} \cdot \frac{\beta_{P_2}}{R_n} = \frac{1}{2\beta_{P_2}} R_n \cdot \frac{\beta_{P_2}}{R_n}$. Hence $X_{\beta_{P_2}}^{R_n} = +\frac{1}{2}$.

The sensitivity indices of all other parameters in R_n are computed using the same procedure described above. By systematically applying this method, we obtained the sensitivity indices of each parameter, which are presented in the table below:

Table 1: Numerical values of sensitivity indices for model parameters in R_n .

Parameter	Sign of sensitivity index (S.I.)	Value of S.I.	Parameter value
β_{P_2}	+	+0.5	0.04
β_H	+	+0.5	0.005
λ_P	+	+0.5	1.2
λ_H	+	+0.5	0.70
μ_H	-	-0.50311	0.0003465
μ_F	-	-1	0.00261026
δ_H	-	-0.0200378	0.01710615
δ_P	-	-	0.4
		0.476512224	

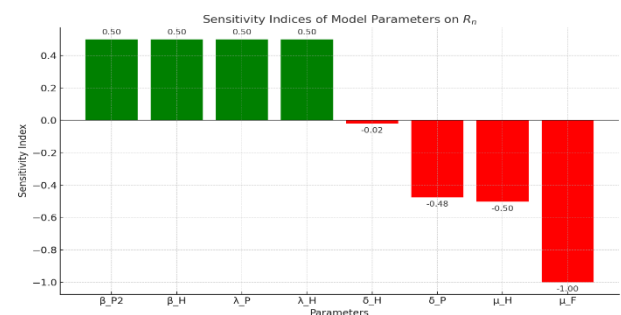


Figure 1: Graphical representation of sensitivity parameters

Table 2: Values for Parameters of the Model

Parameter	θ_1	θ_2	θ_4	θ_5	α_1	λ_P	λ_H	δ_P	δ_H	μ_P	μ_H	ω	β_H	β_{P_1}	β_{P_2}
Value	0.1	0.3	0.8	0.9	0.5	1.2	0.7	0.4	0.01710615	0.0003465	0.0003465	0.00261026	0.005	0.04	0.04

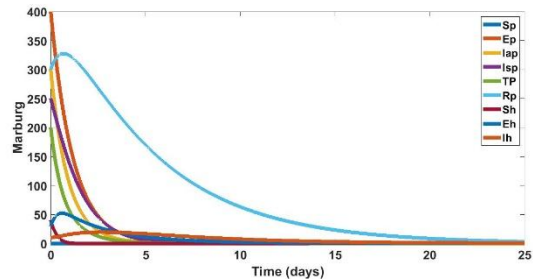


Figure 2: Model-Based Solutions for Marburg Virus Disease (MVD)

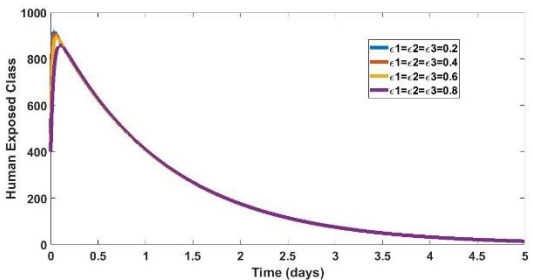


Figure 2: Optimal Control Strategies on Exposed human class

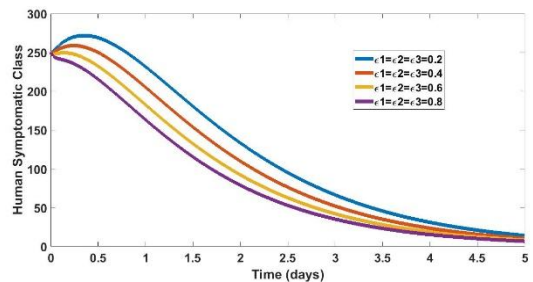


Figure 3: Optimal Control Strategies on Exposed symptomatic class

III. DISCUSSION

From our analysis, a slight initial increase in the exposed human population may occur as awareness and education campaigns are rolled out. This rise likely results from more individuals recognizing their exposure or seeking testing, leading to earlier identification before symptoms appear. As individuals are identified and isolated after contact with the virus, the exposed class may temporarily grow. However, the subsequent decline in exposed cases suggests that isolation measures are effective in curbing further transmission.

An increase in the symptomatic human population can be attributed to improved public awareness and early detection efforts. As individuals become more informed about the symptoms, diagnosis and reporting increase. This facilitates timely treatment, resulting in more individuals progressing through the symptomatic phase with reduced severity. The concentration of cases in this phase also reflects the success of isolation strategies in preventing secondary transmission.

Public education appears to contribute to a rise in the asymptomatic population. Behavior changes, such as improved hygiene and adherence to preventive measures, may lead to milder infections or asymptomatic cases. Early treatment and medical care reduce the severity of illness, enabling a greater proportion of individuals to recover without developing severe symptoms.

The reduction in overall infections and fatalities can be linked to effective public awareness, early detection, and prompt treatment. As individuals adopt protective behaviors and infected persons are quickly identified and isolated, the chain of transmission is disrupted. These strategies not only lower disease burden but also ease pressure on healthcare systems.

The overall increase in the recovered population indicates that these combined interventions; public awareness, early detection, and case isolation are effective in promoting recovery and reducing the spread of Marburg Virus Disease. These findings highlight the importance of sustained control efforts to improve public health outcomes in affected communities.

Table 3: Compartments of MVD Model

Compartment	Values
S_P	550
E_P	400
I_{AP}	300
I_{SP}	250
T_P	200
R_P	300
S_H	40
E_H	30
I_H	10

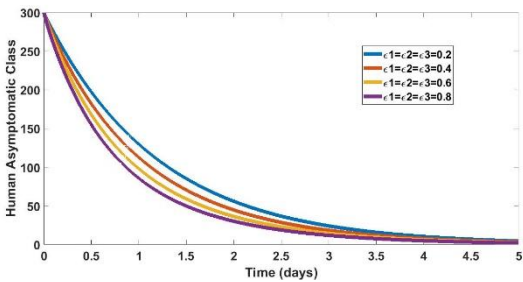


Figure 4: Optimal Control Strategies on Asymptomatic human class

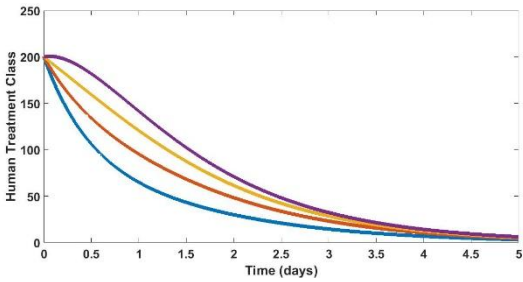


Figure 5: Optimal Control Strategies on Treated human class

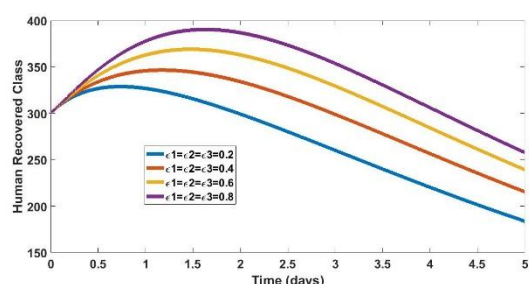


Figure 6: Optimal Control Strategies on Recovered human class

IV. CONCLUSION

The implementation of optimal control strategies such as public awareness, early detection, and isolation profoundly impacts the dynamics of Marburg virus transmission. The initial rise in exposed and symptomatic individuals can be understood as a temporary effect driven by increased awareness and education campaigns, which promote more prompt detection and reporting of cases. This early increase is followed by a decline, reflecting the effectiveness of timely isolation and treatment measures in interrupting transmission chains.

Moreover, the growth in the asymptomatic population suggests a shift toward milder disease presentations, likely resulting from early interventions and widespread adoption of preventive behaviors encouraged by public education. These behavioral changes contribute to reduced transmission and improve overall outcomes.

The steady increase in recovered individuals further highlights the success of these control measures, indicating not only a reduction in new infections but also enhanced recovery rates. Collectively, these results emphasize that a coordinated and multifaceted approach focused on raising awareness, enabling early diagnosis, and ensuring isolation is essential to control the spread and severity of Marburg virus disease.

Sustained investment in public health infrastructure and community education remains critical for effectively managing infectious disease outbreaks and mitigating their impact on affected populations.

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