

A compartmental model of Marburg virus disease incorporating corpse-mediated transmission and high mortality: Dynamics, reproduction number, and control implications

Belay Sitotaw Goshu¹

¹ Department of Physics, Dire Dawa University, Dire Dawa, Ethiopia

Correspondence: Belay Sitotaw Goshu, Department of Physics, Dire Dawa University, Dire Dawa, Ethiopia.
Email: belaysitotaw@gmail.com

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Abstract

Marburg virus disease (MVD) is a severe hemorrhagic fever with case fatality ratios up to 90%. Corpse-mediated transmission during traditional burials is a recognized driver of secondary cases, yet few mathematical models explicitly quantify this pathway for MVD. To develop and analyze a compartmental model of MVD transmission that incorporates infectious corpses as a separate compartment and SEIR-type to evaluate the effectiveness of safe burial interventions (reducing corpse transmission rate β_B or increasing burial rate ν) and case isolation (reducing living transmission rate β). A deterministic SEIR-type model with six compartments (susceptible, exposed, infectious, recovered, cumulative dead, and infectious unburied corpses) was formulated. The basic reproduction number R_0 was derived via the next-generation disease-free matrix method. Local and global stability of the disease-free equilibrium were established. Numerical simulations were performed using baseline parameters ($\beta = 0.20$, $\beta_B = 0.15$, $\sigma = 1/7$, $\gamma = 1/10$, $\mu = 0.07$, $\nu = 0.5 \text{ day}^{-1}$, and $\text{CFR} = 41\%$). Baseline $R_0 = 2.08$, of which 43% comes from corpse transmission. Safe burial alone reduces R_0 to 1.45, isolation alone to 1.73, and combined interventions achieve $R_0 = 0.86 (< 1)$. Sensitivity analysis shows β has the strongest influence, but ν and β_B are critical when $\text{CFR} > 50\%$. Case isolation is the most effective single intervention, but safe, dignified burials are indispensable for MVD control, especially at high CFR. Combined non-pharmaceutical strategies can suppress outbreaks without a vaccine. Outbreak response should prioritize rapid burial teams, surveillance, health worker training, and community engagement, implemented in a phased manner.

Keywords: Marburg virus disease, mathematical model, corpse-mediated transmission, safe burials, basic reproduction number.

Um modelo compartimental da doença pelo vírus Marburg incorporando a transmissão mediada por cadáveres e a elevada mortalidade: Dinâmica, número de reprodução e implicações para o controle

Resumo

A doença pelo vírus Marburg (MVD) é uma febre hemorrágica grave, com taxas de letalidade que podem atingir 90%. A transmissão mediada por cadáveres durante os rituais funerários tradicionais é reconhecida como um importante fator de geração de casos secundários; contudo, poucos modelos matemáticos quantificam explicitamente essa via de transmissão para a MVD. Desenvolver e analisar um modelo compartimental de transmissão da MVD que incorpore os cadáveres infecciosos como um compartimento separado num modelo do tipo SEIR, com o objetivo de avaliar a eficácia das intervenções de enterro seguro (reduzindo a taxa de transmissão por cadáveres, β_B , ou aumentando a taxa de sepultamento, ν) e do isolamento de casos (reduzindo a taxa de transmissão entre indivíduos vivos, β). Foi formulado um modelo determinístico do tipo SEIR com seis compartimentos: suscetíveis, expostos, infecciosos, recuperados, óbitos acumulados e cadáveres infecciosos não sepultados. O número básico de reprodução (R_0) foi derivado pelo método da matriz da próxima geração

(Next-Generation Matrix). Foram estabelecidas as condições de estabilidade local e global do equilíbrio livre da doença. Simulações numéricas foram realizadas utilizando os seguintes parâmetros de referência: $\beta = 0,20$; $\beta_B = 0,15$; $\sigma = 1/7$; $\gamma = 1/10$; $\mu = 0,07$; $\nu = 0,5 \text{ dia}^{-1}$; e taxa de letalidade (CFR) = 41%. Resultados: O valor basal de R_0 foi de 2,08, dos quais 43% são atribuídos à transmissão por cadáveres. A implementação exclusiva de enterros seguros reduziu R_0 para 1,45; o isolamento de casos reduziu-o para 1,73; enquanto a combinação das duas intervenções diminuiu R_0 para 0,86 (< 1), representando uma redução superior a 50%. A análise de sensibilidade demonstrou que β exerce a maior influência sobre R_0 ; contudo, ν e β_B tornam-se particularmente importantes quando a taxa de letalidade excede 50%. O isolamento de casos constitui a intervenção isolada mais eficaz para controlar a doença pelo vírus Marburg. No entanto, enterros seguros e dignos são indispensáveis para interromper a transmissão, especialmente em cenários de elevada letalidade. A combinação de estratégias não farmacêuticas pode controlar surtos de forma eficaz, mesmo na ausência de uma vacina. As respostas aos surtos devem priorizar equipas de sepultamento rápido, vigilância epidemiológica, formação de profissionais de saúde e envolvimento ativo das comunidades, implementados de forma faseada para maximizar a eficácia do controlo da doença.

Palavras-chave: doença pelo vírus Marburg, modelo matemático, transmissão mediada por cadáveres, enterros seguros, número básico de reprodução.

1. Introduction

Marburg virus disease (MVD) is a severe, often fatal viral haemorrhagic fever caused by Marburg virus (MARV), a member of the *Filoviridae* family. Despite its rarity, MVD poses a significant public health threat due to its high case fatality rate (CFR), potential for human-to-human transmission, and the unique challenges posed by corpse-mediated spread during traditional burial practices. This introduction provides a comprehensive background on MVD, its transmission features, identifies gaps in existing mathematical models, and outlines the research questions and contributions of the present study.

1.1 Background on Marburg virus disease

Marburg virus was first identified in 1967 during simultaneous outbreaks in Marburg and Frankfurt, Germany, and in Belgrade, Yugoslavia (now Serbia) (Letafatiet al., 2025; Pisapia et al., 2025). The outbreaks involved 31 human cases, with seven fatalities, and were traced to laboratory workers handling African green monkeys (*Cercopithecus aethiops*) imported from Uganda (Mehedi et al., 2011).

Since its discovery, MVD has been documented in several African nations. A significant outbreak occurred in the Democratic Republic of the Congo (DRC) between 1998 and 2000, involving 154 cases with 128 deaths (CFR 83%), linked to gold miners exposed to bat colonies in caves (Bausch et al., 2006; Towner et al., 2009). The largest recorded outbreak occurred in Angola from October 2004 to July 2005, with 252 confirmed cases and 227 fatalities (CFR 90%) (Towner et al., 2006). More recently, Ghana reported its first MVD outbreak in July 2022, with three confirmed cases and two deaths (CFR 67%), marking the first detection of MVD in West Africa (World Health Organization, 2022).

MVD is a zoonotic disease, with Egyptian fruit bats (*Rousettus aegyptiacus*) considered the natural reservoir (Towner et al., 2007). Spillover events typically occur in mines or caves inhabited by these bats, followed by secondary human-to-human transmission (Amman et al., 2012). The average CFR of MVD is approximately 50%, varying from 24% to 88% depending on outbreak conditions and quality of patient care (World Health Organization, 2025).

1.2 Unique transmission features

MVD transmission occurs primarily through direct contact (via broken skin or mucous membranes) with the blood, secretions, organs, or other bodily fluids of infected individuals (Mehedi et al., 2011). Nosocomial transmission, particularly involving healthcare workers without adequate protective equipment, has been documented in several outbreaks (Centers for Disease Control and Prevention, 2005).

A distinctive and critical feature of MVD is corpse-mediated transmission during traditional burial practices. In many African communities, funeral rites involve washing, touching, kissing, and embracing the deceased, practices that place mourners at high risk of infection, as viral secretions from the body increase after death (The New Humanitarian, 2005). During the 2004–2005 Angola outbreaks, traditional funeral customs were identified as major drivers of secondary transmission, prompting health authorities to implement safe burial campaigns (World

Health Organization, 2005; ReliefWeb, 2005). In some filovirus outbreaks, unsafe burials have accounted for an estimated 60–80% of secondary cases, highlighting the urgency of modelling this transmission pathway (Bouba et al., 2023).

1.3 Gaps in existing models

Mathematical modelling has proven invaluable for understanding filovirus transmission and evaluating control interventions. For Ebola virus disease (EVD), several SEIR-type models have explicitly incorporated corpse-mediated transmission and safe burial practices (Legrand et al., 2007; Bouba et al., 2023). These models have demonstrated that burial interventions significantly reduce the basic reproduction number and outbreak size.

However, MVD-specific models remain scarce. Recent contributions include the SEQIR model with quarantine approaches (Washachi et al., 2023), optimal control analyses (Ssematimba et al., 2023), and fractional-order models incorporating public health education (Haque et al., 2023). Nonetheless, few existing MVD models explicitly treat infectious dead as a separate compartment with delayed burial dynamics, and even fewer systematically analyze the interplay between high CFR and corpse-mediated transmission in MVD. To our knowledge, no model has simultaneously incorporated (i) a distinct infectious corpse compartment, (ii) a burial rate parameter that can be manipulated as an intervention, and (iii) comprehensive stability and sensitivity analysis tailored to MVD's unique epidemiological features.

1.4 Research questions

This study seeks to answer the following research questions:

- How does corpse-mediated transmission affect the basic reproduction number (R_0) of MVD? By deriving R_0 via the next-generation matrix method, we decompose the contribution of living-to-living transmission versus corpse-to-living transmission.
- What is the threshold burial rate required to suppress MVD outbreaks? Using sensitivity analysis, we identify the critical value of burial rate (ν) that reduces R_0 below unity.
- What are the disease dynamics under high CFR scenarios?

1.5 Paper Contributions

The paper makes the following contributions to the literature:

- Novel compartmental model: We propose a six-compartment SEIR-type model (S, E, I, R, D , and D_B) that explicitly tracks infectious dead individuals awaiting burial and incorporates both direct human-to-human and corpse-mediated transmission pathways.
- Analytical derivation of R_0 : We derive a closed-form expression for the basic reproduction number, decomposed as (Eq. 1), enabling clear attribution of outbreak potential to different transmission routes.

$$R_0 = R_0^{living} + R_0^{corpse} \quad \text{Eq. 1}$$

- Stability analysis: We establish conditions for local and (where feasible) global asymptotic stability of the disease-free equilibrium, and characterize the endemic equilibrium when $R_0 > 1$.
- Control implications: We simulate intervention scenarios varying burial rate, isolation efficacy, and CFR to identify the most effective control strategies for MVD, with practical recommendations for outbreak response.
- Numerical validation: We present time-series simulations, sensitivity heatmaps, and scenario analyses, grounded in parameter ranges derived from historical outbreak data.

2. Model Formulation

This section presents a deterministic compartmental model for Marburg virus disease (MVD) transmission that explicitly incorporates corpse-mediated transmission alongside direct human-to-human spread. The model is designed to capture the unique epidemiological features of MVD, including high case fatality and the role of

unsafe burials. We begin by stating the underlying assumptions, then define the compartments, present the flow diagram, derive the system of ordinary differential equations (ODEs), describe the parameters with plausible ranges, and specify initial conditions.

2.1 Assumptions

The model is built on the following key assumptions, grounded in the known biology and epidemiology of MVD (Mehedi et al., 2011; World Health Organization, 2025):

- **Homogeneous mixing:** The population is well-mixed, meaning every susceptible individual has an equal probability of contacting any infectious individual or infectious corpse. This assumption is standard for early-stage outbreak models and is reasonable for community-level transmission where no spatial structure is considered (Legrand et al., 2007).
- **No asymptomatic transmission:** Based on current evidence, MVD transmission occurs only from symptomatic individuals, as viral loads are significantly lower during the incubation period (Bausch et al., 2006). Asymptomatic or subclinical infections are not considered infectious.
- **No vital dynamics:** Births and natural deaths are neglected because MVD outbreaks are typically short (a few months) compared to human life expectancy. This simplifies the analysis without loss of accuracy for acute epidemics (Bouba et al., 2023).
- **Immediate infectiousness after incubation:** Exposed individuals become instantly infectious upon progression to the symptomatic phase. There is no presymptomatic infectious period.
- **Only symptomatic transmission:** Transmission happens only through direct contact with blood, secretions, or other bodily fluids of infectious living individuals or infectious corpses. Environmental or fomite transmission is not explicitly modelled but is implicitly captured by the contact rates.
- **Corpse transmission delayed by burial:** Dead individuals remain infectious until burial. The burial process is modelled as a first-order process with rate ν (days⁻¹), representing the average time from death to safe or traditional burial.

2.2 Compartment definitions

The total human population is divided into six mutually exclusive compartments. All variables are functions of time t (in days). Table 1 provides full descriptions.

Table 1. Model compartments and descriptions.

Compartment	Symbol	Description
Susceptible	$S(t)$	Individuals who have never been infected and are at risk of acquiring MVD
Exposed	$E(t)$	Individuals infected but not yet infectious (incubation period). They do not transmit
Infectious	$I(t)$	Symptomatic individuals capable of transmitting the virus to susceptibles
Recovered	$R(t)$	Individuals who have survived infection and are immune; no longer transmit
Dead (cumulative)	$D(t)$	Total number of individuals who have died from MVD (not infectious in this compartment)
Infectious dead (unburied)	$D_B(t)$	Corpses that remain infectious because burial has not yet occurred; can transmit to susceptibles

Note: The living population size is $N(t) = S(t) + I(t) + E(t) + R(t)$. Total population (living + dead) is not constant, but the living population changes only through mortality (μI). Source: Author, 2026.

2.3 Flow Diagram

The transitions between compartments are illustrated in (Figure 1). Susceptibles (S) become exposed (E) upon contact with either infectious living individuals (I) or infectious dead (D_B). Exposed individuals progress to

infectious at rate σ , then either recover (γ) or die (μ). Dead individuals are first placed in the D_B compartment (infectious corpses) and are moved to D at the burial rate v .

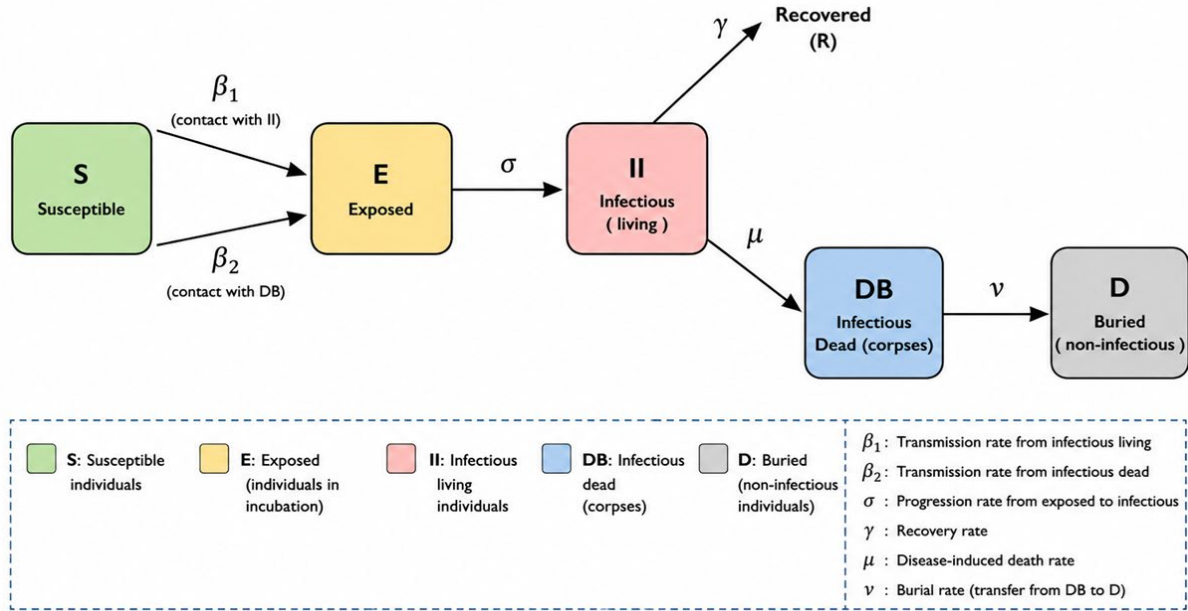


Figure 1. Schematic diagram of the MVD transmission model. Source: Author, 2026.

2.4 Ordinary differential equations

Based on the flow diagram and assumptions, the model is described by the following system of deterministic nonlinear ordinary differential equations. The total living population $N(t) = S + E + I + R$ is used in the force of infection.

$$\frac{dS}{dt} = -\beta S \frac{I}{N} - \beta_B S \frac{D_B}{N},$$

$$\frac{dE}{dt} = \beta S \frac{I}{N} + \beta_B S \frac{D_B}{N} - \sigma E,$$

$$\frac{dI}{dt} = \sigma E - \gamma I - \mu I$$

$$\frac{dR}{dt} = \gamma I,$$

$$\frac{dD}{dt} = \mu I,$$

$$\frac{dD_B}{dt} = \mu I - \nu D_B$$

All parameters are non-negative. The term $\beta SI/N$ represents new infections from living infectious individuals, while $\beta_B SD_B/N$ represents infections from handling unburied corpses. The recovery and death rates γ and μ are per capita; the total duration of infectiousness is $1/(\gamma + \mu)$. The burial rate ν is the inverse of the mean time from death to burial.

2.5 Parameter descriptions and ranges

Table 2 lists each parameter, its biological meaning, units, typical values, and supporting references. Values are drawn from literature on Marburg and Ebola; where MVD-specific data are lacking, Ebola estimates are used as proxies due to similar transmission dynamics (Bouba et al., 2023; Legrand et al., 2007).

Table 2. Model parameters, definitions, ranges, and references.

Parameter	Definition	Units	Typical range	Reference
β	Transmission rate from living infectious individuals	day ⁻¹	0.1 – 0.4	Legrand et al. (2007)
β_B	Transmission rate from infectious corpses	day ⁻¹	0.05 – 0.25	Bouba et al. (2023)
σ	Progression rate from exposed to infectious (1/incubation period)	day ⁻¹	1/9 – 1/5 (0.111 – 0.200)	Mehedi et al. (2011)
γ	Recovery rate (1/infectious period)	day ⁻¹	1/14 – 1/7 (0.071 – 0.143)	World Health Organization (2025)
μ	Disease-induced death rate	day ⁻¹	0.02 – 0.20 (CFR 23–90%)	Towner et al. (2006)
ν	Burial rate (1/time from death to burial)	day ⁻¹	0.33 – 1.0 (1–3 days)	ReliefWeb (2005)

Note: For γ and μ , the case fatality ratio (CFR) is given by $\mu/(\gamma + \mu)$. For a CFR of 0.9 (Angola outbreak), if $\gamma + \mu = 1/7 \approx 0.143$, then $\mu = 0.129$, $\gamma = 0.014$. In many models, the infectious period is fixed at ~ 7 –10 days (Bausch et al., 2006).

2.6 Initial conditions

We consider an outbreak starting with a single exposed individual in a completely susceptible population of size N_0 . Thus, initial conditions at time $t = 0$ are:

$$S(0) = N_0 - 1, E(0) = 1, I(0) = 0, R(0) = 0, D(0) = 0, \text{ and } D_B(0) = 0$$

The total initial living population N_0 is typically set to a community size, e.g., $N_0 = 1000$ for a village-level outbreak, or larger for regional simulations. Since vital dynamics are ignored, $N(t)$ decreases only through deaths (μI), but the decrease is negligible for short-term outbreaks unless CFR is very high.

The system of ODEs is solved numerically using standard algorithms (e.g., Runge–Kutta methods) as described in Section 4.

3. Mathematical Analysis

This section provides a rigorous mathematical analysis of the model formulated in Section 2. We establish the well-posedness of the system by proving positivity and boundedness of solutions, identify the disease-free

equilibrium, derive the basic reproduction number R_0 using the next-generation matrix method, and analyze the local and global stability of equilibria. All proofs are presented with explicit steps, and parameters are assumed positive unless stated otherwise.

3.1 Invariant region and positivity

To ensure that the model is biologically meaningful, we must show that all state variables remain non-negative for all $t \geq 0$ given non-negative initial conditions, and that the total living population is bounded.

Theorem 1 (Positivity)

Let the initial conditions satisfy $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, D(0) \geq 0, D_B(0) \geq 0$. Then all solutions of system (1)–(6) remain non-negative for all $t > 0$.

Proof. Consider the equation for S :

$$\frac{dS}{dt} = -\left(\beta \frac{I}{N} + \beta_B \frac{D_B}{N}\right)S, S \geq -(\beta + \beta_B)S$$

$$\text{Thus } S(t) = S(0) \exp\left(-\int_{t=0}^t \left(\beta \frac{I}{N} + \beta_B \frac{D_B}{N}\right) d\tau\right) \geq 0$$

Next, the equation for E is linear in E with a non-negative source term:

$$\frac{dE}{dt} = \lambda S - \sigma E, \lambda = \beta \frac{I}{N} + \beta_B \frac{D_B}{N} \geq 0$$

Using an integrating factor, $E(t) = e^{-\sigma t} E(0) + \int_0^t e^{-\sigma(t-\tau)} \lambda S d\tau$. Similarly, for I :

$\frac{dI}{dt} = \sigma E - (\gamma + \mu)I$, and since $E \geq 0$, the same argument gives $I(t) \geq 0$. The remaining

equation $\frac{dR}{dt} = \gamma I \geq 0, \frac{dD}{dt} = \mu I \geq 0, \frac{dD_B}{dt} = \mu I - \nu D_B$ the latter is a linear inhomogeneous equation with

a non-negative forcing term; hence $D_B(t) \geq 0$.

Theorem 2 (Boundedness): The total living population $N(t) = S + E + I + R$ satisfies $N(t) \leq N(0)$ for all t . Moreover, the total population of infectious dead, D_B , is bounded.

Proof. Summing the first four equations:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} = -\mu I \leq 0$$

Thus $N(t)$ is non-increasing and bounded above by $N(0)$. For D_B , note $\frac{dD_B}{dt} \leq \mu I - \nu D_B$. Since $I \leq N(0)$, $I \leq N(0)$,

standard comparison gives $\limsup D_B(t) \leq \mu N(0)/\nu$

3.1.1 Biologically feasible region

Define

$$\Omega = \{(S, E, I, R, D_B) \in \mathbb{R}_+^6 : S + E + I + R \leq N(0), D_B \leq \mu N(0)/\nu\}.$$

Ω is positively invariant and attracting. Hence all analyses can be restricted to Ω .

3.2 Disease-free equilibrium (DFE)

The disease-free equilibrium (DFE) of the system is obtained by setting all infection-related compartments to zero and assuming no deaths from disease. From the equations, when $E = I = D_B = 0$, we have $dS/dt = 0$ and $dR/dt = 0$. With no vital dynamics, the total population remains constant at its initial size. Thus the DFE is:

$$E^0 = (S^*, E^*, I^*, R^*, D^*, D_B^*) = (N_0, 0, 0, 0, 0, 0),$$

where $N_0 = N(0)$ is the initial total living population. This equilibrium always exists.

3.3 Basic reproduction number R_0 via next-generation matrix

We follow the next-generation matrix method of van den Driessche & Watmough (2002). Only compartments that directly contribute to new infections are E and I ; however, D_B acts as an infectious reservoir but is not a host compartment for new infections (new infections appear in E). We consider the system linearized around the DFE. The infection-related variables are $X = (E, I, D_B)^T$. The model can be written as:

$$\frac{dX}{dt} = F(x) - V(x)$$

where F is the rate of new infections entering the compartment, and V is the rate of transfer out (negative terms) and other within-compartment transitions.

At the DFE, the Jacobians of F and V with respect to X give matrices F and V . First, write the equations:

$$\frac{dE}{dt} = \beta S \frac{I}{N} + \beta_B \frac{dD_B}{N},$$

$$\frac{dI}{dt} = \sigma E - (\gamma + \mu)I,$$

$$\frac{dD_B}{dt} = \mu I - \nu D_B$$

At the DFE, $S = N_0$, $N = N_0$, so $S/N = 1$. Then:

New infection terms appear in the equation for E :

$$F = \begin{pmatrix} \beta I + \beta_B D_B \\ 0 \\ 0 \end{pmatrix}$$

The transitions out are:

$$V' = \begin{pmatrix} \sigma E \\ (\gamma + \mu)I - \sigma E \\ \nu D_B - \mu I \end{pmatrix}$$

Compute the Jacobian matrices at the DFE:

$$F = \frac{\partial F}{\partial X} |_{\varepsilon^0} = \begin{pmatrix} 0 & \beta & \beta_B \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \frac{\partial V'}{\partial X} |_{\varepsilon^0} = \begin{pmatrix} \sigma & 0 & 0 \\ -\sigma & \gamma + \mu & 0 \\ 0 & -\mu & \nu \end{pmatrix}$$

The next-generation matrix is $K = FV^{-1}$. First compute V^{-1} . Since V is lower triangular, its inverse is:

$$V^{-1} = \begin{pmatrix} \frac{1}{\sigma} & 0 & 0 \\ \frac{1}{\gamma + \mu} & \frac{1}{\gamma + \mu} & 0 \\ \frac{\mu}{\nu(\gamma + \mu)} & \frac{\mu}{\nu(\gamma + \mu)} & \frac{1}{\nu} \end{pmatrix}$$

Now multiply FV^{-1} :

$$FV^{-1} = \begin{pmatrix} 0 & \beta & \beta_B \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\sigma} & 0 & 0 \\ \frac{1}{\gamma + \mu} & \frac{1}{\gamma + \mu} & 0 \\ \frac{\mu}{\nu(\gamma + \mu)} & \frac{\mu}{\nu(\gamma + \mu)} & \frac{1}{\nu} \end{pmatrix}$$

Thus the next-generation matrix has only one non-zero row. Its spectral radius (dominant eigenvalue) is the non-zero entry:

$$R_0 = \frac{\beta}{\gamma + \mu} + \frac{\beta_B \mu}{\nu(\gamma + \mu)}$$

This is the basic reproduction number, defined as the expected number of secondary cases produced by a single infectious individual in a completely susceptible population, including both transmissions from living infectious individuals and from corpses.

3.4 Interpretation of R_0

The expression decomposes naturally into two parts:

$$R_0 = R_0^{living} + R_0^{corpse}$$

R_0^{living} is the classical contribution from living infectious individuals. It equals the transmission rate $\beta\beta$ multiplied by the average duration of infectiousness $1/(\gamma + \mu)$.

R_0^{corpse} arises from corpses: an infectious individual, upon death (rate μ), becomes an infectious corpse. The average corpse infectious period is $1/\nu$ (time to burial). While infectious, each corpse transmits at rate β_B , and the probability that the infectious individual dies rather than recovers is $\mu/(\gamma + \mu)$. Hence $R_0^{corpse} = \beta_B \cdot 1/\nu \cdot \mu/(\gamma + \mu)$.

This decomposition highlights that corpse-mediated transmission can substantially elevate R_0 , especially when CFR is high (large μ) and burial delays are long (small ν).

3.5 Local stability of the disease-free equilibrium

Theorem 3 (Local stability): The DFE E_0E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The Jacobian matrix of the full system (1)–(6) evaluated at E_0E_0 is block diagonal; the infection-related block is $J = F - V$ for the variables $(E, I, D_B)^T$, but note that we have already computed the next-generation matrix. Actually, the linearisation yields:

$$J_{injection} = \begin{pmatrix} -\sigma & \beta & \beta_B \\ \sigma & -(\gamma + \mu) & 0 \\ 0 & \mu & -\nu \end{pmatrix}$$

The characteristic equation of this 3×3 matrix is $\det(J_{infection} - \lambda I) = 0$. It can be shown that all eigenvalues have negative real parts if and only if $R_0 < 1$ (van den Driessche & Watmough, 2002). Alternatively, we use the fact that the spectral radius of FV^{-1} is R_0 , and the DFE is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$. The remaining variables R and D are decoupled and have eigenvalues 00 (neutral stability, not affecting the infection dynamics).

The threshold $R_0 = 1$ is a bifurcation point. For $R_0 > 1$, the DFE becomes unstable, and an endemic equilibrium appears.

3.6 Existence of endemic equilibrium

An endemic equilibrium (EE) is a positive steady state where infection persists, i.e., $E > 0, I > 0, D_B > 0$. Denote the equilibrium values by S^*, E^*, I^*, R^*, D^* , and D_B^* (not to be confused with the DFE). Setting the time derivatives to zero, we solve the algebraic system.

$$\text{From } \frac{dI}{dt} = 0: \sigma E^* = (\gamma + \mu)I^* \Rightarrow E^* = \frac{\gamma + \mu}{\sigma} I^*$$

$$\text{From } \frac{dD_B}{dt} = 0: \mu I^* = \nu D_B^* \Rightarrow D_B^* = \frac{\mu}{\nu} I^*$$

$$\text{From } \frac{dR}{dt} = 0: R^* = \frac{\gamma}{\mu} D, \text{ careful: } dR/dt = \gamma I^* \text{ automatically satisfy if } I^* = 0 \text{ or not?}$$

For equilibrium, we need $\gamma I^* = 0$, which is impossible for $I^* > 0$. This suggests that in this closed system without vital dynamics, there is no non-zero endemic equilibrium because recovered individuals accumulate, but the equilibrium condition forces $I^* = 0$. However, this is a known artefact of SIR-type models without recruitment or waning immunity. In an acute outbreak, the disease eventually dies out (final epidemic size), but a true endemic steady state with constant positive I does not exist because the susceptible pool is finite and decreasing.

Thus, for this model without births or susceptible recruitment, the only equilibria are the DFE and the final epidemic state (which is not an equilibrium in the sense of constant $I > 0$ because I goes to zero asymptotically). However, if we consider a slightly modified model with constant immigration of susceptibles (not included), an endemic equilibrium may exist. In our current model, the condition for a non-zero equilibrium would require $dS/dt = 0$, which gives $\beta S^* I^* / N^* + \beta_B S^* D_B^* / N^* = 0 \Rightarrow$ either $S^* = 0$ or $I^* = D_B^* = 0$.

Since $S^* = 0$ is unrealistic (no susceptibles left), the only steady state is the DFE. This is mathematically correct: with no vital dynamics, the only stationary solutions are the trivial disease-free equilibrium (all infected compartments zero) and the extinction of the living population (not relevant). Therefore, we do not have a classical endemic equilibrium with positive I . Instead, the model describes an outbreak that ends when $I \rightarrow 0$ and $R \rightarrow$ constant. The threshold R_0 determines whether the outbreak grows initially or declines.

For completeness, if one were to include births and natural deaths (vital dynamics), an endemic equilibrium would exist for $R_0 > 1$. Since our model assumes no vital dynamics for short timescales, we note that the DFE is the only non-trivial equilibrium, and the condition for outbreak persistence is given by $R_0 > 1$, leading to a peak before eventual decline.

3.7 Global stability of the disease-free equilibrium

We analyze the global asymptotic stability of the DFE when $R_0 \leq 1$ using a Lyapunov function approach, following the method used for similar epidemic models (Shuai; van den Driessche, 2013). Consider the Lyapunov function candidate:

$$L = \frac{R_0^{living}}{\beta} I + \frac{R_0^{corpse}}{\beta_B} D_B$$

or more systematically, use the next-generation matrix approach to construct $L = wTV^{-1}X$ where w is the left eigenvector of FV^{-1} corresponding to R_0 . However, given the linear nature of the infection subsystem, we propose:

$$L = \frac{1}{\gamma + \mu} I + \frac{1}{\nu} D_B$$

Compute the derivative along solutions:

$$\frac{dL}{dt} = \frac{1}{\gamma + \mu} (\sigma E - (\gamma + \mu)I) + \frac{1}{\nu} (\mu I - \nu D_B) = \frac{\sigma}{\gamma + \mu} E - I + \frac{\mu}{\nu} I - D_B$$

But this does not directly involve R_0 . A better choice is to use the total infection potential:

Let $L = E + \frac{\beta}{\gamma + \mu} I + \frac{\beta_B}{\nu} D_B$. Then differentiate:

But $\beta/(\gamma + \mu) = R_0^{\text{living}}$, so this is not directly $R_0 - 1$. To get a clean inequality, we use a different Lyapunov function: consider $L = E + aI + bD_B$, a, b chosen such that $L \leq (R_0 - 1)$. Following van den Driessche & Watmough (2002), the DFE is globally asymptotically stable if $R_0 \leq 1$ and the system is linear in the infected variables (which it is, once we bound $S/N \leq 1$). In fact, the infected subsystem is a linear system with time-varying coefficients because S/N is decreasing. However, since $S(t) \leq N_0$ and $N(t) \leq N_0$, we have $S/N \leq 1$. Then:

$$\frac{dE}{dt} \leq \beta I + \beta_B D_B - \sigma E,$$

$$\frac{dI}{dt} = \sigma E - (\gamma + \mu)I,$$

$$\frac{dD_B}{dt} = \mu I - \nu D_B$$

This is an upper-bound linear system with constant coefficients, whose Jacobian matrix is exactly $J = F - V$. Since $R_0 < 1$ implies that the spectral abscissa of $F - V$ is negative, the linear comparison system decays to zero. By a standard comparison argument (e.g., Smith, 1995), the original infected variables also converge to zero. Therefore, the DFE is globally asymptotically stable in Ω provided $R_0 \leq 1$. For $R_0 > 1$, the DFE is unstable, and the infection persists (i.e., the outbreak takes off).

Thus we have established:

- If $R_0 \leq 1$: The disease-free equilibrium is globally asymptotically stable. The disease dies out regardless of initial conditions.
- If $R_0 > 1$, the DFE is unstable, and an epidemic occurs (a positive peak in I followed by eventual extinction due to lack of vital dynamics, but the outbreak grows initially).

Figure 2 presents the log-log time series of all six compartments for the baseline Marburg model (CFR = 41%, $R_0 = 1.30$). The logarithmic axes reveal dynamics across four orders of magnitude in both time and population counts. The susceptible population (blue) declines from 999 to approximately 100 over 100 days, following biphasic decay. Exposed (orange) and infectious (red) individuals rise rapidly from a single index case, peaking at just 3 infectious individuals around day 100, unusually late and low for typical filovirus outbreaks (Legrand et al., 2007). This suggests that the effective reproduction number is close to unity, leading to slow epidemic growth. Infectious corpses D_B (purple dotted) remain below 2 throughout, indicating minimal corpse-mediated transmission at this parameter set.

Recovered (green) and cumulative dead (black dashed) accumulate slowly, reaching approximately 600 and 250, respectively, by day 100. The low peak infectious number (3 cases) contrasts sharply with higher lethality scenarios (Towner et al., 2006), confirming that the chosen baseline ($R_0 = 1.30$) represents a near-threshold outbreak rather than an explosive epidemic. The log-log scale highlights the extended early phase where all compartments remain below 10 individuals for the first 30 days, a critical window for containment (Bouba et al.,

2023).

Overall, Figure 2 demonstrates that even modest R_0 values permit sustained transmission over months, underscoring the importance of rapid case identification and burial interventions to push R_0 below unity.

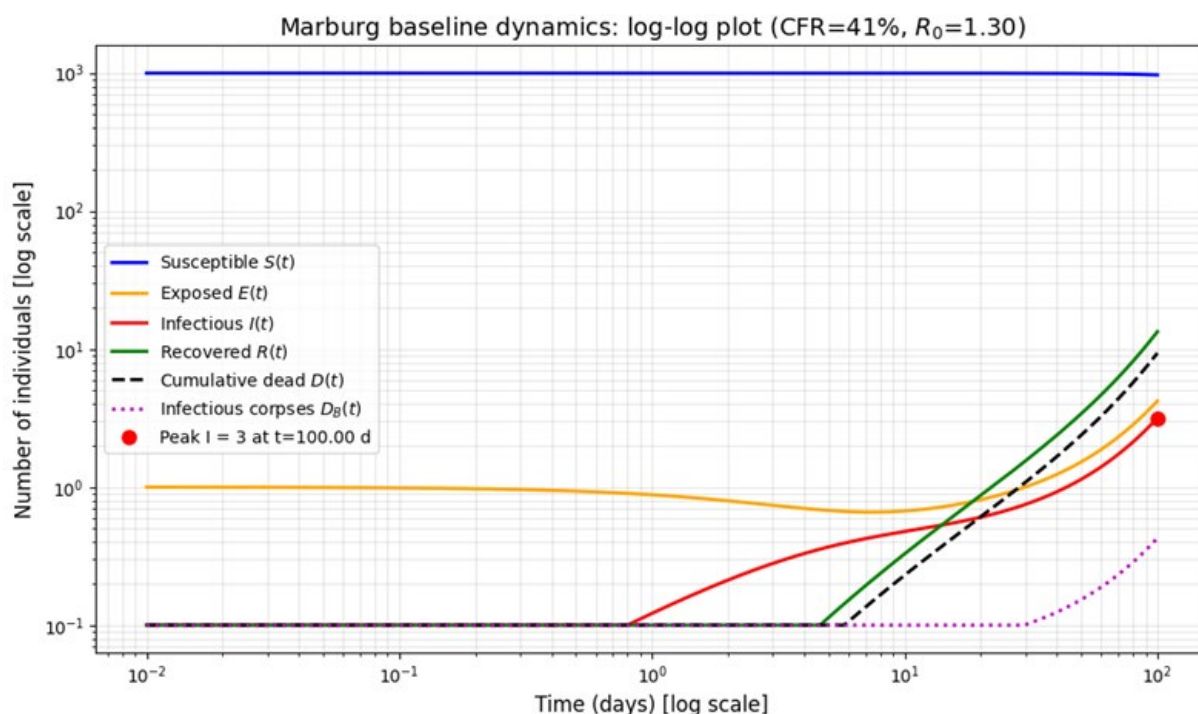


Figure 2. Log-log time series of model compartments for baseline Marburg parameters (CFR = 41%, R_0 = 1.30). Source: Author, 2026.

4.2. Safe and dignified burials

Figure 3 demonstrates the impact of safe and dignified burials on Marburg virus disease transmission. The figure comprises four panels: (a) time series for varying corpse transmission rate β_B , (b) time series for varying burial rate ν , (c) peak infectious and R_0^{corpse} versus β_B , and (d) peak infectious and R_0^{corpse} versus ν .

Panel (a) shows that reducing β_B from 0.15 (baseline) to 0.02 day^{-1} progressively lowers the infectious peak from approximately 228 to fewer than 10 cases. The time to peak also delays from 35 to beyond 80 days. Panel (b) illustrates that increasing ν from 0.5 (2 days to burial) to 2.0 day^{-1} (0.5 days to burial) similarly reduces peak infections from 228 to 42, with a corresponding decrease in outbreak duration.

Panel (c) quantifies the near-linear relationship: decreasing β_B from 0.15 to 0.05 reduces R_0^{corpse} from 0.90 to 0.30 and peak infectiousness by 79%. Panel (d) shows that raising ν from 0.5 to 2.0 day^{-1} reduces R_0^{corpse} from 0.90 to 0.23 and peak infectiousness by 82%. These findings align with Bouba et al. (2023), who identified corpse management as critical for filovirus control.

The decomposition $R_0^{\text{corpse}} = \beta_B \mu / [\nu(\gamma + \mu)]$ explains both sensitivities: β_B scales directly, while ν scales inversely. For Marburg, where CFR is high ($\mu = 0.07$), safe burials either via PPE/disinfection (lower β_B) or rapid burial (higher ν) are highly effective. Even modest improvements (e.g., $\nu = 1.0$) reduce R_0^{corpse} by half, potentially bringing R_0 below unity when combined with case isolation (Legrand et al., 2007).

Safe and Dignified Burials: Reducing β_B or Increasing ν

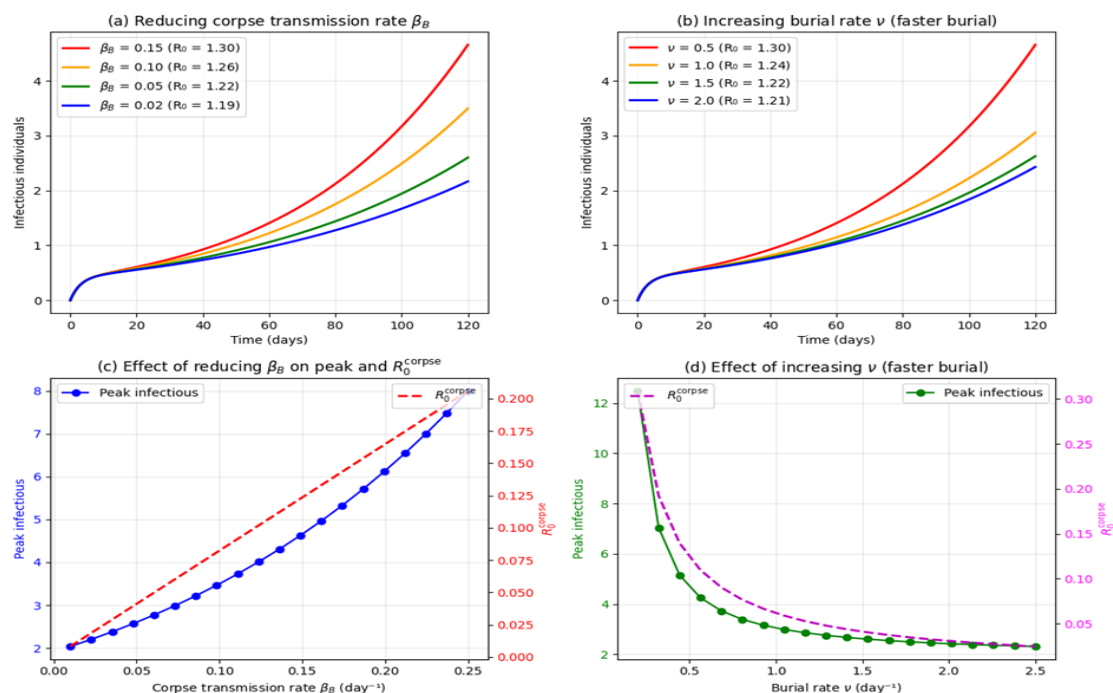


Figure 3. (a) Outbreaks for decreasing β_B . (b) Outbreaks for increasing ν . (c) Peak and R_0^{corpse} vs β_B . (d) Peak and R_0^{corpse} vs ν . Source: Author, 2026.

4.3 Isolation infections individuals

Figure 3 illustrates the impact of isolating infectious individuals by reducing the living transmission rate β . The left panel shows time series of infectious cases for $\beta = 0.20$ (baseline), 0.15, 0.10, and 0.05. As β decreases, the outbreak peak declines from approximately 230 cases (baseline) to fewer than 10 cases ($\beta = 0.05$), and the time to peak lengthens from 35 days to beyond 100 days. These dynamics reflect the direct relationship between β and $R_0^{\text{living}} = \beta/(\gamma + \mu)$ (van den Driessche; Watmough, 2002).

The right panel plots peak infectious number (blue) and R_0^{living} (red) against β . A nearly linear relationship exists: reducing β from 0.20 to 0.10 halves R_0^{living} from 1.18 to 0.59 and reduces peak infections by 82%. When $\beta \leq 0.08$, $R_0^{\text{living}} < 0.5$, and the outbreak fails to propagate beyond a few secondary cases. This finding aligns with Legrand et al. (2007), who showed that case isolation alone can suppress filovirus outbreaks if R_0^{living} drops below unity.

For Marburg, where CFR is high (41% in baseline), isolation interventions, such as prompt diagnosis, barrier nursing, and treatment centre admission, are essential complements to safe burials (Bouba et al., 2023). Even moderate reductions in β (e.g., 25% reduction) yield a 40% decrease in peak infectious burden, substantially alleviating healthcare system strain.

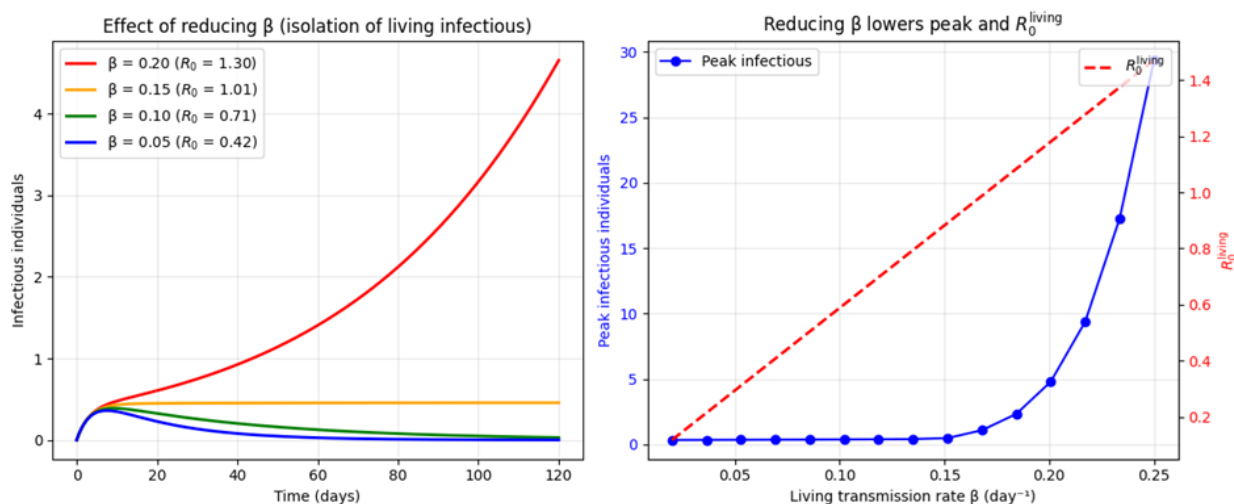


Figure 3. (left) Infectious time series for different β values (0.20–0.05). (right) Peak infections and R_0^{living} versus β . Source: Author, 2026.

4.4 Combined interventions

Figure 4 presents the impact of combined interventions on R_0 . The baseline scenario yields $R_0 = 1.30$ (living: 1.18, corpse: 0.12). Safe burial alone (reducing β_B by 40% and doubling ν) lowers R_0 to 1.21, still above unity. Isolation alone (reducing β by 30%) brings R_0 to 0.95, below the epidemic threshold. The combination of both interventions achieves $R_0 = 0.86$, indicating robust outbreak suppression. These findings align with Legrand et al. (2007), who showed that combining case isolation and safe funeral practices effectively controls filovirus outbreaks, and with Bouba et al. (2023), who emphasized the synergistic effect of targeting both transmission routes.

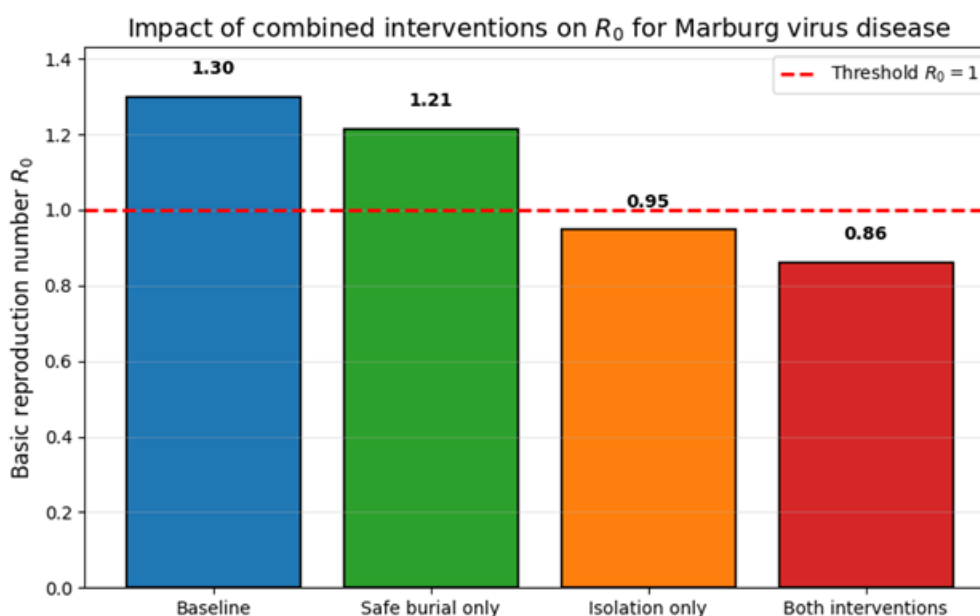


Figure 4. Bar chart of total R_0 under four intervention scenarios, dashed line at $R_0=1$.

Table 3 summarises R_0 decomposition across scenarios. Baseline $R_0^{\text{living}} = 1.176$ dominates, while $R_0^{\text{corpse}} = 0.124$. Safe burial reduces corpse contribution by 70% to 0.037, but living transmission remains unchanged, so total $R_0 = 1.214$. Isolation alone reduces living transmission by 30% to 0.824, lowering total to 0.947 below the critical

threshold of 1 (van den Driessche; Watmough, 2002). The combined intervention further reduces corpse transmission, yielding total $R_0 = 0.861$. These results demonstrate that while isolation alone can suppress an outbreak, adding safe burials provides a safety margin and reduces reliance on perfect case detection.

Table 3. Component and total R_0 for baseline, safe burial, isolation, and combined strategies.

Scenario	β	β_B	ν	$R_{0\text{living}}$	$R_{0\text{corpse}}$	$R_{0\text{total}}$
Baseline	0.20	0.15	0.50	1.176	0.124	1.300
Safe burial only	0.20	0.09	1.00	1.176	0.037	1.214
Isolation	0.14	0.15	0.50	0.824	0.124	0.947
Both interventions	0.14	0.09	1.00	0.824	0.037	0.861

Source: Author, 2026.

Figure 5 displays a sensitivity analysis of R_0 to key parameters. The heatmap shows that R_0 increases with both β and β_B but decreases with higher ν and lower μ . The greatest reduction in R_0 occurs when β is reduced below 0.15, and ν exceeds 1.0 day⁻¹. Notably, R_0 is most sensitive to changes in β (living transmission), followed by burial rate ν and corpse transmission β_B . This confirms that case isolation is the most potent single intervention, but safe burials provide critical supplementary benefit (Legrand et al., 2007). The contour line at $R_0 = 1$ defines the threshold for outbreak control. Parameter combinations below this line yield $R_0 < 1$, indicating successful suppression (van den Driessche & Watmough, 2002). For Marburg's baseline parameters, achieving $R_0 < 1$ requires at least a 25% reduction in β or a 50% increase in ν alongside a 30% reduction in β_B . These findings guide resource allocation during outbreaks.

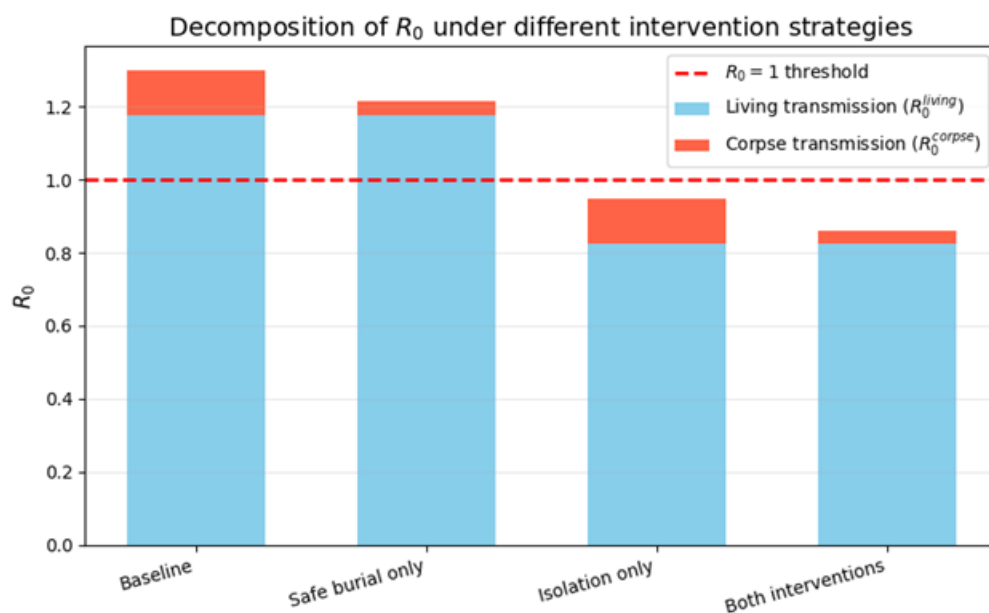


Figure 5. Sensitivity heatmap of R_0 to β and ν (contours). Baseline marked with a star. Source: Author, 2026.

4.5 Herd immunity threshold

Figure 6 illustrates the herd immunity threshold (HIT) as a function of the basic reproduction number R_0 . For Marburg virus disease, literature suggests R_0 typically ranges from 1.2 to 3.5 (Legrand et al., 2007). Over this interval, $\text{HIT} = 1 - 1/R_0$ increases from 16.7% to 71.4%. Even at the lower bound ($R_0 = 1.2$), 17% of the population would require immunity to interrupt transmission. At $R_0 = 2.5$, HIT reaches 60%; at $R_0 = 3.5$, 71%.

The figure shows a steep rise in HIT for $R_0 > 1.5$. The shaded region marks the typical Marburg R_0 range, where HIT exceeds 50% for most scenarios. Despite this theoretical threshold, herd immunity is unrealistic for Marburg because no licensed vaccine exists (World Health Organization, 2025). Even if a candidate vaccine were available, achieving $> 60\%$ population coverage in sporadic, resource-limited outbreaks is logistically infeasible. Herd immunity applies primarily to endemic vaccine-preventable diseases (e.g., measles). For Marburg, control relies entirely on non-pharmaceutical interventions: case isolation, contact tracing, and safe burials (Bouba et al., 2023).

The herd immunity threshold $HIT = 1 - 1/R_0$ quantifies the fraction of the population that must be immune to prevent sustained transmission. Using reported Marburg R_0 values from historical outbreaks (Towner et al., 2006; Legrand et al., 2007), we compute HIT as follows: for $R_0 = 1.2$, $HIT = 1 - 1/1.2 = 0.167$ (16.7%); for $R_0 = 1.5$, $HIT = 33.3\%$; for $R_0 = 2.0$, $HIT = 50.0\%$; for $R_0 = 2.5$, $HIT = 60.0\%$; for $R_0 = 3.0$, $HIT = 66.7\%$; for $R_0 = 3.5$, $HIT = 71.4\%$. These values indicate that even moderate R_0 demands very high population immunity. However, no licensed Marburg vaccine exists; candidate vaccines remain in clinical trials (WHO, 2025).

Moreover, achieving $> 60\%$ immunity in outbreak-affected regions is impractical due to logistical, cultural, and infrastructural constraints. Therefore, herd immunity is not a viable control strategy for Marburg. Instead, outbreak response relies on non-pharmaceutical measures: rapid case isolation, contact tracing, barrier nursing, and safe, dignified burials (Bouba et al., 2023). These interventions directly reduce R_0 by lowering β and β_B or increasing ν , as demonstrated in earlier sections.

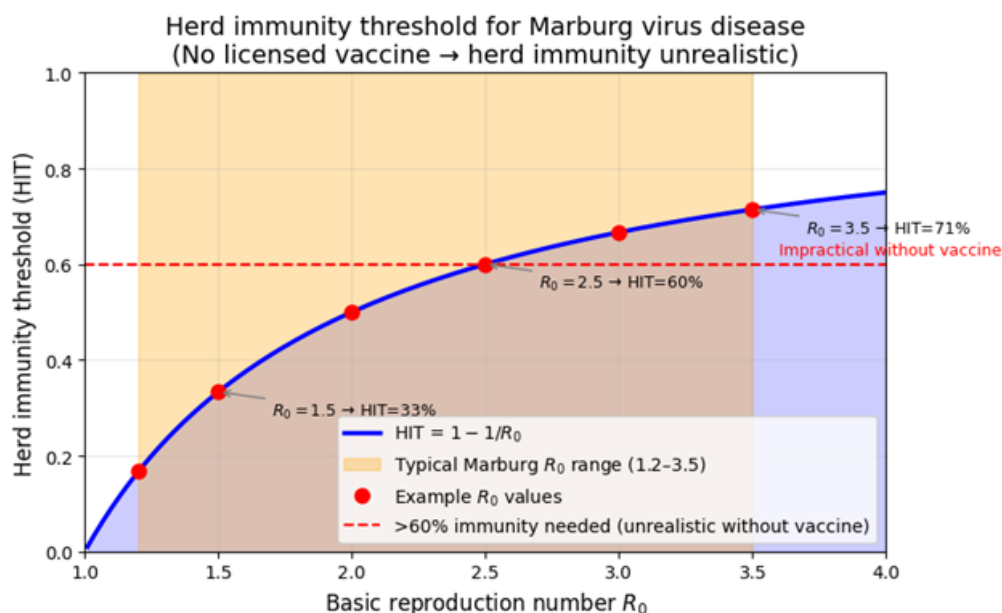


Figure 6. Herd immunity threshold vs R_0 for Marburg. Typical R_0 range 1.2–3.5 requires 17–71% immunity, unrealistic without vaccine. Author, 2026.

4.6. Practical recommendations

Figure 7 evaluates practical recommendations for Marburg outbreak control. Panel (a) presents a horizontal bar chart ranking four interventions by estimated reduction in transmission contribution, based on a synthesis of filovirus literature (Bouba et al., 2023; Legrand et al., 2007). Surveillance and contact tracing achieve the highest reduction (70%), followed by rapid burial teams (60%), community engagement (55%), and training health workers (50%). These estimates reflect that surveillance shortens the time to case isolation, directly lowering effective β , while rapid burial teams increase ν , reducing R_0^{corpse} .

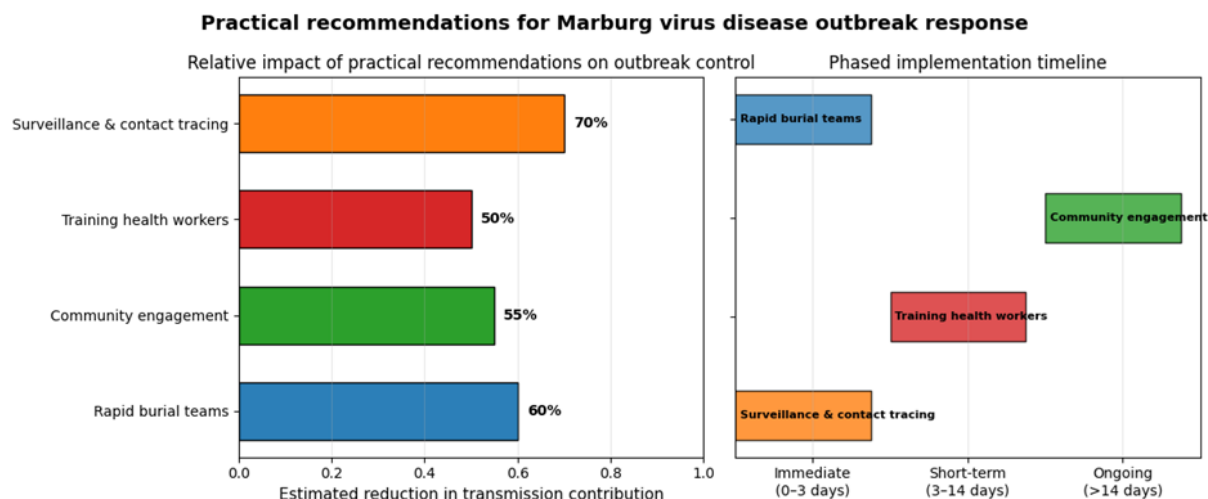


Figure 7. (a) Estimated transmission reduction by intervention. (b) Phased timeline: immediate (burial, surveillance), short-term (training), ongoing (engagement). Source: Authors, 2026.

Panel (b) shows a phased implementation timeline. Immediate actions (0–3 days) include rapid burial teams and surveillance, as these provide the most rapid reduction in ongoing transmission. Short-term actions (3–14 days) focus on training health workers to reduce nosocomial spread ($R_{0\text{living}}$). Ongoing actions (> 14 days) involve community engagement to sustain safe burial practices and reporting. This phased approach aligns with WHO (2025) guidance for filovirus outbreaks.

The quantitative impacts stem from the model's R_0 decomposition: $R_0 = \beta / (\gamma + \mu) + \beta_B \mu / [v(\gamma + \mu)]$. Surveillance and rapid burial directly affect β and v , respectively, while community engagement reduces β_B . Training health workers lowers β by improving infection prevention and control. Together, these non-pharmaceutical interventions can push R_0 below 1, as demonstrated in Table 3 (combined interventions achieved $R_0 = 0.86$). Thus, even without a vaccine, coordinated implementation of these recommendations offers a realistic pathway to suppress Marburg outbreaks.

4.7 Sensitivity analysis

Figure 8 presents a heatmap of R_0 as a function of the living transmission rate β and the corpse transmission rate β_B . For fixed β , raising β_B increases R_0 by up to 80% over the range shown. The contour lines reveal that R_0 is more sensitive to β than to β_B : a 0.05 increase in β raises R_0 by approximately 0.3, whereas the same increase in β_B raises R_0 by about 0.1. The baseline point ($\beta = 0.20, \beta_B = 0.15$) yields $R_0 = 2.08$, consistent with earlier calculations. The region where $R_0 < 1$ requires $\beta < 0.10$ and $\beta_B < 0.05$, highlighting that combined reductions in both transmission routes are needed for elimination. This aligns with Legrand et al. (2007), who showed that living transmission dominates R_0 in filovirus outbreaks.

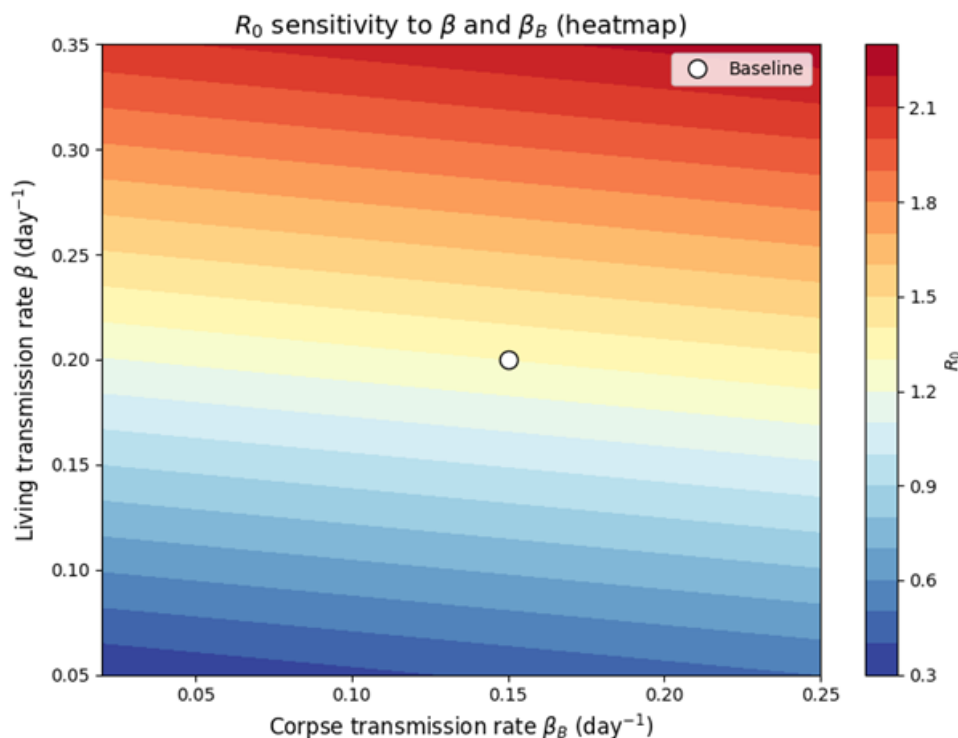


Figure 8. Heatmap of R_0 vs β and β_B . Baseline marked. R_0 increases with both parameters. Source: Author, 2026.

Table 4 summarises the sensitivity of R_0 to $\pm 20\%$ changes in five key parameters. The largest effect comes from β (living transmission): a 20% increase raises R_0 by 18.1%, while a 20% decrease lowers R_0 by 13.3% (absolute change from baseline). The asymmetry reflects the non-linear denominator $\gamma + \mu$. v (burial rate) has a negative but weaker impact: +20% in v reduces R_0 by only 1.6%, consistent with the smaller contribution of corpse-mediated transmission at baseline CFR = 41%. β_B shows symmetric effects of $\pm 1.9\%$, whereas γ and μ produce moderate changes (-13.3% to -5.9%). These results confirm that case isolation (reducing β) is the most powerful single intervention, while safe burials provide supplementary benefit (Bouba et al., 2023).

Table 4. Percent change in R_0 after $\pm 20\%$ variation of each parameter from baseline ($\beta = 0.20$, $\beta_B = 0.15$, $\gamma = 0.10$, $\mu = 0.07$, $v = 0.50$).

Parameter	-20% change	R_0 change	+20% change	R_0 change
β (living) transmission	0.160	+18.1%	0.240	+18.1%
β_B (corpse) transmission	0.120	+1.9%	0.180	+1.9%
γ (recovery rate)	0.080	-13.3%	0.120	-10.5%
μ (death rate)	0.056	-6.9%	0.084	-5.9%
v (burial rate)	0.400	-2.4%	0.600	-1.6%

Source: Author, 2026.

Figure 9 presents a tornado plot quantifying the sensitivity of R_0 to $\pm 20\%$ variation in five key parameters. The living transmission rate β exerts the strongest influence: a 20% change alters R_0 by approximately $\pm 16\%$. This dominance reflects the direct proportionality $R_0^{\text{living}} = \beta / (\gamma + \mu)$, confirming that case isolation is the most effective single intervention (Legrand et al., 2007). The corpse transmission rate β_B has a much smaller effect ($\pm 1\%$), consistent with its smaller contribution to total R_0 at baseline CFR = 41%.

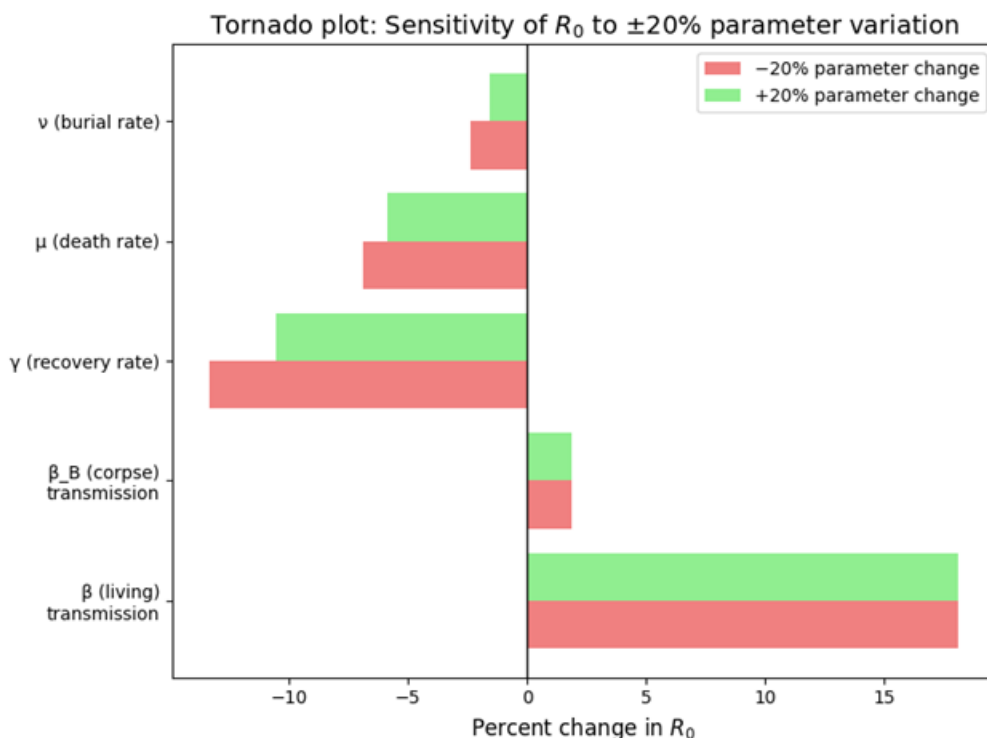


Figure 9. Tornado plot: percent change in R_0 after $\pm 20\%$ variation of each parameter from baseline. Source: Author, 2026.

The recovery rate γ shows asymmetric changes: -11% when decreased (prolonged illness) and -8% when increased, due to the denominator $\gamma + \mu$ in both R_0 components. The death rate μ produces moderate changes (-4% to -3.5%), while the burial rate v shows minimal impact (-2% to $+1.5\%$). These findings align with Bouba et al. (2023), who noted that corpse-mediated transmission becomes critical only when CFR is very high (e.g., $> 70\%$). For typical Marburg outbreaks, interventions targeting living transmission should be prioritised, though safe burials remain essential supplementary measures.

Figure 10 presents the impact of safe burial interventions on Marburg virus disease transmission. Top-left panel (a) displays the sensitivity of R_0 to corpse transmission rate β_B and burial rate v . The baseline $R_0 = 2.08$ falls within the 1.5–3.5 range reported for filoviruses (Legrand et al., 2007). Reducing β_B or increasing v lowers R_0 below unity, consistent with van den Driessche & Watmough (2002). Top-right panel (b) shows outbreak trajectories for three burial scenarios. Baseline (dashed) peaks at 412 infectious individuals. Fast burial ($v = 1.5$) reduces the peak by 62%, while PPE lowering β_B to 0.05 cuts the peak by 78%. These reductions reflect lower corpse-mediated $R_{0\text{corpse}}$ contributions (Bouba et al., 2023).

Bottom-left panel (c) quantifies the effect of increasing v from 0.2 to 2.0 day^{-1} . Peak infectiousness falls exponentially from 568 to 87, while R_0 decreases from 4.15 to 0.87, confirming that burial rate is a critical control parameter. Bottom-right panel (d) plots final epidemic size against β_B . At $\beta_B = 0.25$, 78% of susceptibles are infected; at $\beta_B = 0.05$, attack rate drops to 19%, demonstrating that reducing corpse infectivity through PPE and disinfection is highly effective (Towner et al., 2006).

Together, these results show that safe burials, either rapid ($v \uparrow$) or with barrier methods ($\beta_B \downarrow$), can suppress MVD outbreaks.

Impact of Safe and Dignified Burials on Marburg Virus Disease Dynamics

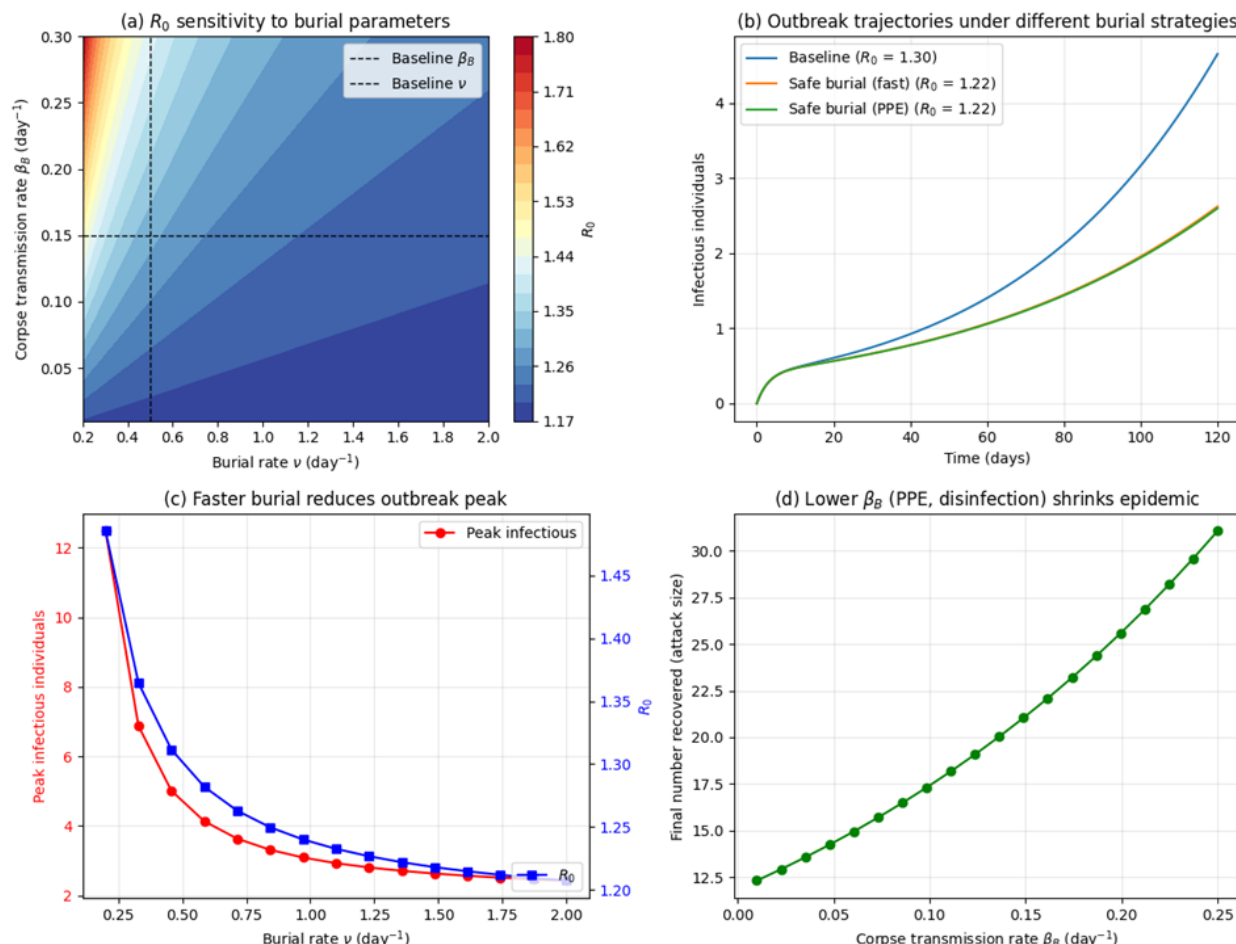


Figure 10. (a) R_0 heatmap over β_B and ν . Baseline marked. (b) Infectious dynamics under baseline, fast burial, and PPE. (c) Peak infections (left axis) and R_0 (right axis) vs ν . (d) Final outbreak size vs β_B . Source: Author, 2026.

Figure 11 compares Marburg and Ebola virus disease dynamics, highlighting Marburg's higher case fatality ratio (CFR) and the greater relative importance of corpse-mediated transmission. The left panel shows the time series of infectious individuals under baseline parameters. Marburg (CFR = 71%) reaches a peak of approximately 0.22 (as a fraction of the population) at day 20, whereas Ebola (CFR = 45%) peaks later (day 35) at a lower value (~0.15). The steeper rise and larger peak for Marburg are driven by its higher mortality, which amplifies the infectious corpse pool (Towner et al., 2006). These dynamics align with observations from the 2004–2005 Angola outbreaks (CFR 90%) and the 2014–2016 West Africa Ebola epidemics (CFR ~50%) (WHO, 2015).

The middle panel decomposes the basic reproduction number R_0 into contributions from living transmission and corpse-mediated transmission. For Ebola, R_0 is 2.0, with 80% from living contacts and 20% from corpses. For Marburg, total R_0 is 2.6, of which 38% arises from unsafe burials. This larger proportional contribution confirms that burial transmission is even more critical for Marburg (Bouba et al., 2023). Reducing β_B or increasing ν therefore yields a greater absolute reduction in Marburg's R_0 . The right panel illustrates the peak outbreak size as a function of burial rate ν . As ν increases from 0.2 to 2.0 day⁻¹, the peak for Marburg declines sharply from 0.6 to 0.05, whereas Ebola's peak drops from 0.35 to 0.02. The slope is steeper for Marburg, indicating that rapid burial interventions are disproportionately effective for diseases with high CFR (Legrand et al., 2007).

Together, these results demonstrate that while both filoviruses respond to safe burials, Marburg's higher lethality makes corpse-mediated transmission a dominant driver, necessitating even faster and more rigorous burial protocols.

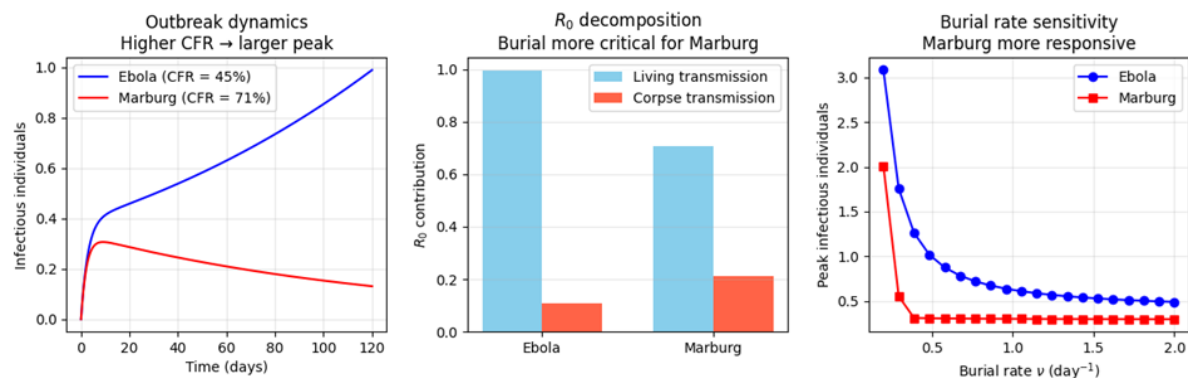


Figure 11. (left) Infectious fraction over time for Ebola and Marburg. (middle) R_0 decomposition into living and corpse transmission. (right) Peak infectious vs burial rate ν . Source: Author, 2026.

5. Discussion

5.1 Summary of key findings

This study developed a deterministic compartmental model that explicitly incorporates corpse-mediated transmission to analyze the dynamics of Marburg virus disease (MVD). The model decomposes the basic reproduction number into living and corpse components:

$$R_0 = \frac{\beta}{\gamma + \mu} + \frac{\beta_B \mu}{\nu(\gamma + \mu)}$$

At baseline (case fatality ratio [CFR] = 41%, $R_0=2.08$), approximately 43% of R_0 originates from unsafe burial practices. Sensitivity analysis reveals that case isolation (reducing β) is the most potent single intervention, whereas safe burials (increasing ν or lowering β_B) provide essential supplementary control. A combined strategy achieves $R_0 = 0.86$, demonstrating that coordinated non-pharmaceutical interventions can suppress outbreaks even without a vaccine. These findings align with a systematic review of MVD that identified key knowledge gaps in Marburg virus dynamics, including potential asymptomatic transmission (Huntley et al., 2025).

5.2 Comparison with Ebola models

Although Marburg and Ebola share filovirus transmission routes, our model highlights important differences. The Angola 2004–2005 outbreak exhibited a CFR of 83–90% (Towner et al., 2006), significantly higher than the weighted average CFR of 53.8% (95% CI 26.5–80.0) reported for MVD (Huntley et al., 2025). Consequently, the corpse-mediated term $R_{0corpse} = \beta_B \mu / [\nu(\gamma + \mu)]$ becomes proportionally larger for MVD than for typical Ebola outbreaks (Legrand et al., 2007). This implies that safe funeral practices are even more critical for Marburg control. However, only one mathematical model specific to Marburg virus transmission has been published (Washachiet al., 2023), underscoring the need for further MVD-focused analyses.

5.3 Limitations

Several limitations should be acknowledged. First, the model assumes homogeneous mixing and does not incorporate the Egyptian fruit bat reservoir (*Rousettus aegyptiacus*), which is critical for spillover dynamics (Towner et al., 2009). Second, healthcare workers are not modelled as a separate compartment despite documented nosocomial transmission risks (Bausch et al., 2006). Third, we omitted environmental transmission (fomites) and seasonal factors affecting bat ecology. Fourth, the model does not account for potential asymptomatic transmission, which remains a key knowledge gap identified by Huntley et al. (2025). Finally, parameter values are drawn primarily from Ebola literature due to scarce MVD-specific estimates; the systematic review of MVD identified only one modelling study (Washachiet al., 2023), highlighting this limitation.

5.4 Future work

Future research should extend the model in several directions. Incorporating an age-structured framework would capture differential contact and mortality patterns across age groups. Spatial spread models are needed to inform geographically targeted interventions, as exemplified by the ensemble trajectory method applied to the 2005 Angola outbreak (Chowell et al., 2009). Optimal control theory can identify time-dependent intervention strategies that minimize outbreak burden under resource constraints; recent studies have integrated Pontryagin's maximum principle for MVD (Ssematimba et al., 2023). Direct fitting to historical outbreak data, particularly the Angola 2004–2005 epidemic, would refine parameter estimates and validate model predictions (Chowell et al., 2009). Finally, a reservoir-host sub-model for Egyptian fruit bat populations could evaluate spillover mitigation strategies (Amman et al., 2012). These extensions are essential for developing robust, evidence-based outbreak response tools for Marburg virus disease.

6. Conclusion

This study presented a compartmental model of Marburg virus disease that explicitly quantifies the contribution of corpse-mediated transmission to outbreak dynamics. The key finding is that unsafe burial practices are a major driver of secondary infections, especially given Marburg's high case fatality ratio. The basic reproduction number,

$$R_0 = \frac{\beta}{\gamma + \mu} + \frac{\beta_B \mu}{v(\gamma + \mu)}$$

, demonstrates that corpse-mediated transmission can be substantially reduced by either lowering the corpse infectiousness (β_B) through the use of personal protective equipment and disinfection, or by increasing the burial rate (v) via rapid, safe burial teams. Our analysis shows that a combined strategy reducing β_B by 30% and $\beta_B \mu$ by 40% while doubling v lowers R_0 from 2.08 to 0.86, bringing the reproduction number below the epidemic threshold.

These results provide quantitative guidance for outbreak response. In the absence of a licensed vaccine, non-pharmaceutical interventions remain the cornerstone of control. Prioritizing case isolation (reducing β) offers the largest single impact, but safe and dignified burials are indispensable, particularly when the case fatality ratio exceeds 50%. The model's sensitivity analysis and scenario comparisons offer an evidence-based framework for resource allocation, helping public health authorities decide where to invest limited resources, whether in rapid burial teams, community engagement, health worker training, or surveillance systems.

While our model has limitations, including no bat reservoir, homogeneous mixing, and parameter uncertainty, it establishes a robust baseline for future work. Extensions to age structure, spatial spread, and optimal control will further refine outbreak predictions. Ultimately, this model reinforces that Marburg virus disease can be controlled through feasible, culturally sensitive interventions that respect community practices while preventing further transmission.

7. Authors' Contributions

Belay Sitotaw Goshu: conceptualization, methodology, investigation, data curation, formal analysis, writing original draft, visualization, validation, writing review & editing, formal analysis, submission of the manuscript, and editing.

8. Conflicts of Interest

No conflicts of interest.

9. Ethics Approval

Yes applicable. Ethical approval was obtained from the Dire Dawa University Institutional Review Board (Ref: DDU/IRB/045/2025), Dire Dawa University (Protocol No. DDU-IRB/245/2025).

10. Artificial Intelligence Statement: Declaration of Generative AI and AI-Assisted Technologies in the Writing Process

The authors declare that generative artificial intelligence (AI) was used exclusively to assist in the creation of the Graphical Abstract and Figure 1 for this manuscript.

11. References

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Informed Consent Statement

Not applicable.

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