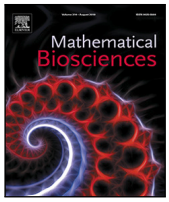
Mathematical Biosciences 364 (2023) 109059

Contents lists available at ScienceDirect

Mathematical Biosciences

journal homepage: www.elsevier.com/locate/mbs



Original research article

Global dynamics of a compartmental model to assess the effect of transmission from deceased

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ARTICLE INFO

Keywords:
Transmission from deceased
Vaccination
Compartmental model
Global dynamics

ABSTRACT

During several epidemics, transmission from deceased people significantly contributed to disease spread, but mathematical analysis of this transmission has not been seen in the literature numerously. Transmission of Ebola during traditional burials was the most well-known example; however, there are several other diseases, such as hepatitis, plague or Nipah virus, that can potentially be transmitted from disease victims. This is especially true in the case of serious epidemics when healthcare is overwhelmed and the operative capacity of the health sector is diminished, such as seen during the COVID-19 pandemic. We present a compartmental model for the spread of a disease with an imperfect vaccine available, also considering transmission from deceased infected in general. The global dynamics of the system are completely described by constructing appropriate Lyapunov functions. To support our analytical results, we perform numerical simulations to assess the importance of transmission from the deceased, considering the data collected from three infectious diseases, Ebola virus disease, COVID-19, and Nipah fever.

1. Introduction

Several infectious diseases caused by pathogenic microorganisms (e.g. bacteria, viruses, parasites, or fungi) can be spread directly or indirectly, from person to person, however, apart from infection from infectious individuals, also corpses of those deceased due to a given epidemic may pose a risk of transmission, especially under special circumstances like natural disasters [1], an overwhelmed health care system [2], or due to traditional funerary practices [3].

Various examples can be mentioned for this phenomenon. Deaths from diseases such as plague, cholera, typhoid fever, tuberculosis, anthrax, smallpox, and influenza pose a substantial risk to health though most agents do not survive long in the human body after death [4]. Contracting dead bodies from tuberculosis, blood-borne viruses (e.g. hepatitis B and C and HIV), and gastrointestinal infections (e.g. cholera, *E. coli*, hepatitis A, rotavirus diarrhea, salmonellosis, shigellosis, and typhoid/paratyphoid fevers) with persons who are involved in close contact with the dead – such as health care workers, military personnel, rescue workers, volunteers, and others – may be exposed to chronic infectious hazards [1,5].

Ebola virus disease (EVD or Ebola) is a severe illness in humans that is found primarily in the African continent. EVD can be transmitted between humans through contact with blood, secretions, organs, or other bodily fluids of infected or dead humans or animals, and has become especially known for the role of traditional burials in disease transmission. Some of the early symptoms of this deadly disease are fever, exhaustion, aches and pains, and loss of appetite. People who exhibit Ebola symptoms should seek medical attention right once, and treatment options include hospital-provided medications and oral or intravenous fluids [6–8]. Influenza remains active in the environment for only one day and HIV remains active in dead bodies kept at 2 °C between 6–15 days, therefore corpses can transmit disease and cause death if not handled safely [4]. Recently the world has seen a devastating COVID-19 pandemic and due to the massive transmission of the virus, infection in humans has led to an unexpected situation in global health. Infected individuals with this disease experience mild, moderate, or severe clinical symptoms. Fever, fatigue, dry cough, shortness of breath, etc. are some of its most typical symptoms [9]. It can also be transmitted via human contact or aerial droplets [10]. After postmortem and forensic tests of the corpse, it was observed that the SARS-CoV-2 persists in the human body months after death and should be infectious for weeks (see e.g. [11,12]). Nipah virus (NiV) is a zoonotic virus that was first identified when a cluster of patients associated with pig farming in Peninsular Malaysia in late September 1998. Close contact with a person who has been infected with the NiV, direct contact with infected animals, such as bats or pigs, or their body fluids (such as their blood, urine, or saliva), eating food products that

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https://doi.org/10.1016/j.mbs.2023.109059
Received 23 March 2023; Received in revised form 31 May 2023; Accepted 1 August 2023
Available online 22 August 2023

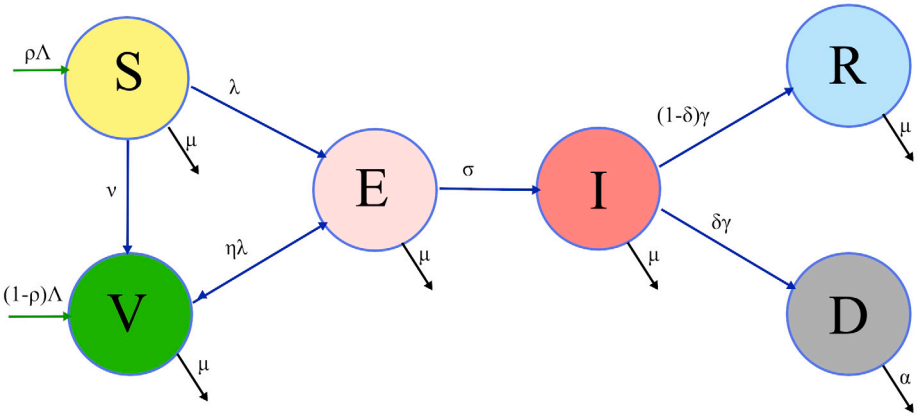


Fig. 1. Transmission diagram. Blue arrows indicate the transition from one compartment to another, and green arrows and black arrows indicate new entries and released for death respectively.

have been contaminated by the body fluids of infected animals (such as palm sap or fruit contaminated by an infected bat) is the transmission route of this virus. Sporadical outbreaks of NiV have been seen in South and Southeast Asia [13,14].

Though not strictly the phenomenon studied in our work, it is interesting to mention that during the 14th-century plague pandemic, also known as the 'Black Death' was not only spread from dead bodies but also was used as biological warfare. Namely, the Mongol army hurled plague-infected cadavers into the besieged Crimean city of Caffa. This disease was transmitted to the sieged inhabitants and fleeing survivors from that area, spreading plague from Caffa to the Mediterranean Basin [15].

Numerous mathematical models are available in literature where a compartment for the deceased can be found, but few of them considered infection transmitted from corpses (see for instance [6,16–20]). However, several studies considered pathological phenomena, and review articles on transmission from corpses can be found [21–29]. For this reason, we are interested to study the transmission of pathogens from the deceased in general. This paper is organized in accordance with the following: formulation of our model and description of the parameters are presented in Section 2. In Section 3, model analysis and in Section 4 the basic reproduction number, equilibrium points, and their stability are presented. Numerical simulations are discussed in Section 5. Finally, the overall discussion is presented in Section 6 as a conclusion.

2. Model formulation

To develop our model, we first divide the total actively-mixing human population (e.g. for EVD, total human population excluding the Ebola-deceased individuals) population, denoted by $N(t)$ at time t, into the following compartments: susceptibles ($S(t)$), vaccinated ($V(t)$), exposed (newly-infected but not infectious) individuals ($E(t)$), infectious individuals with clinical symptoms of the disease ($I(t)$) and recovered ($R(t)$). Hence,

$$N(t) = S(t) + V(t) + E(t) + I(t) + R(t).$$

There is an additional compartment D introduced for deceased humans who passed away due to virus infection and have not been buried yet.

We denote the birth and natural death rates by Λ and μ, respectively. A fraction ρ (with $0 < \rho < 1$) of newborns not vaccinated after birth enter the susceptible compartment, while the remaining fraction enters the vaccinated compartment. The force of infection is given by $\lambda(t) = (\beta_1 I(t) + \beta_2 D(t))$, where the parameter β_1 represents the effective contact rate of susceptible individuals to get an infection from infected individuals and β_2 is the effective unprotected contact rate of susceptible individuals, who become infected from dead bodies. Since vaccines are not fully efficient enough for a disease so we consider vaccine efficiency, which is modeled by introducing the parameter $\eta \in [0, 1]$. Infected individuals progress from the exposed to the infectious compartment at rate σ, and further, they leave the visibly infected compartment at rate γ (i.e. the average duration of the latent period is $1/\sigma$ days and that of the infectious period is $1/\gamma$ days). Disease-induced death affects individuals in the infected compartment. A fraction $0 < \delta < 1$ of those leaving the infectious compartment will die due to the infection and arrive in the D class, while the remaining fraction recovers and moves to the recovered compartment R. Infected corpses are buried at the rate α, i.e. the average time until the burial equals $1/\alpha$ days. Besides vaccination right after birth, we also consider υ as the vaccination rate of adults and with that, susceptible individuals are transferred to the vaccinated compartment. The transmission diagram of our model is shown in Fig. 1. A complete description of the model parameters is summarized in Table 1. With the above notations and assumptions, our model takes the form

$$\begin{aligned}
S'(t) &= \rho\Lambda - (\beta_1 I(t) + \beta_2 D(t))S(t) - \upsilon S(t) - \mu S(t),\\
V'(t) &= (1-\rho)\Lambda - \eta(\beta_1 I(t) + \beta_2 D(t))V(t) + \upsilon S(t) - \mu V(t),\\
E'(t) &= (\beta_1 I(t) + \beta_2 D(t))(S(t) + \eta V(t)) - (\sigma + \mu)E(t),\\
I'(t) &= \sigma E(t) - (\gamma + \mu)I(t),\\
R'(t) &= (1-\delta)\gamma I(t) - \mu R(t),\\
D'(t) &= \delta\gamma I(t) - \alpha D(t).
\end{aligned} \tag{1}$$

The following initial conditions are associated with the system (1): $S(0) > 0, V(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, D(0) \geq 0$.

Model (1) has a similar structure to some earlier models, especially for Ebola [19,22,30]. However, those papers do not have compartments for vaccinated individuals. On the other hand, some of these papers include compartments for hospitalized, which we did not include in our model, concentrating on the role of transmission from deceased. We note that system (1) is similar to the model studied in [18], where the compartment of low-risk susceptibles corresponds to our vaccinated compartment. Apart from the main difference, the presence of vaccination of susceptible individuals (i.e. movement from the S class to the V class, which term clearly cannot be present in [18] due

Table 1
Description of parameters of model (1).

Parameters	Description
Λ	Recruitment rate
ρ	Fraction of unvaccinated at birth
μ	Natural death rate
β_1	Transmission rate from infectious
β_2	Transmission rate from deceased
η	Vaccination efficiency
$1/\sigma$	Incubation period
$1/\gamma$	Length of infectious period
$1/\alpha$	Average time until burial
δ	Fraction of lethal cases
υ	Adult vaccination rate

to the different meaning of the corresponding compartments), another important difference is that we use mass action incidence, which allows us to prove global asymptotic stability of the endemic equilibrium without additional conditions. Note that with the total population being close to constant, applying mass action incidence (with appropriately modified constants) does not significantly differ from using standard incidence.

To obtain our analytical results described in Sections 3 and 4, for technical reasons we will omit vaccination of adults, hence, we study the reduced system

$$\begin{aligned}
S'(t) &= \rho\Lambda - (\beta_1 I(t) + \beta_2 D(t))S(t) - \mu S(t),\\
V'(t) &= (1-\rho)\Lambda - \eta(\beta_1 I(t) + \beta_2 D(t))V(t) - \mu V(t),\\
E'(t) &= (\beta_1 I(t) + \beta_2 D(t))(S(t) + \eta V(t)) - (\sigma+\mu)E(t),\\
I'(t) &= \sigma E(t) - (\gamma+\mu)I(t),\\
R'(t) &= (1-\delta)\gamma I(t) - \mu R(t),\\
D'(t) &= \delta\gamma I(t) - \alpha D(t)
\end{aligned} \tag{2}$$

with the initial conditions $S(0) \geq 0, V(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, D(0) \geq 0$. We note that the assumption of omitting vaccination of older individuals is not merely technical: in the case of several childhood diseases, vaccination almost entirely takes place within a short time after birth, and vaccination of older individuals is negligible.

3. Basic properties

3.1. Positivity and boundedness of solutions

For system (2), it is necessary to prove that all the state variables are non-negative and all the solutions of the system with positive initial conditions have a positive invariant solution. Thus we have the following lemma.

Lemma 1. *All solutions of system (2) with non-negative initial conditions will enter the invariant region* $\phi = \{S, V, E, I, R, D \in \mathbb{R}^6_+ : 0 < N \leq \Lambda/\mu\}$.

Proof. It can be easily proved that all existing solutions starting from non-negative initial conditions remain non-negative for all time $t > 0$. We already know that the total human population of individuals is $N = S + V + E + I + R$. Then we have

$$N'(t) = S'(t) + V'(t) + E'(t) + I'(t) + R'(t) = \Lambda - \mu N(t) - \delta\gamma I(t),$$

from which

$$N'(t) \leq \Lambda - \mu N(t)$$

follows. If the initial value of the total population $N(0) = N_0$, then we obtain that

$$N(t) \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N_0\right)e^{-\mu t}.$$

So $N(t) \leq \frac{\Lambda}{\mu}$ as $t > 0$ and this implies $I(t) \leq \frac{\Lambda}{\mu}$. Now we can write the sixth equation of (2) as

$$D'(t) \leq \delta\gamma\frac{\Lambda}{\mu} - \alpha D(t),$$

calculating in a similar fashion to the above, we can see that $D(t) \leq \frac{\Lambda\delta\gamma}{\alpha\mu}$ as $t > 0$. Hence the region is positively invariant and attracts all solutions of the equations of the system. □

3.2. Derivation of the basic reproduction number

To calculate the basic reproduction number $\mathscr{R}_0$ of (2), we follow the general approach established in [31,32]. In model (2), the infectious states are E, I and D.

The model (2) has a unique disease-free equilibrium, given by

$$E_0 = \left(\tfrac{\rho\Lambda}{\mu}, \tfrac{(1-\rho)\Lambda}{\mu}, 0, 0, 0, 0\right).$$

Substituting the corresponding coordinates of the disease-free equilibrium E_0, we compute the Jacobian F as

$$F = \begin{bmatrix} 0 & \beta_1\left(\frac{\rho\Lambda}{\mu} + \eta\frac{(1-\rho)\Lambda}{\mu}\right) & \beta_2\left(\frac{\rho\Lambda}{\mu} + \eta\frac{(1-\rho)\Lambda}{\mu}\right)\\ 0 & 0 & 0\\ 0 & 0 & 0 \end{bmatrix}$$

and the Jacobian V given by

$$V = \begin{bmatrix} \mu+\sigma & 0 & 0\\ -\sigma & \gamma+\mu & 0\\ 0 & -\gamma\delta & \alpha \end{bmatrix},$$

According to [31,32], the basic reproduction number $\mathscr{R}_0$ is the spectral radius of FV^{-1}, hence we obtain

$$\rho(FV^{-1}) = \mathscr{R}_0 = \frac{(\alpha\beta_1 + \beta_2\gamma\delta)\Lambda(\eta(1-\rho)+\rho)\sigma}{\alpha\mu(\gamma+\mu)(\mu+\sigma)}.$$

4. Existence of equilibria and stability analysis

4.1. Existence of endemic equilibrium

To determine the existence of endemic equilibria, we let the right-hand sides of all equations in (2) to be equal to zero. Solving the last three equations we get $E = \frac{I(\gamma+\mu)}{\sigma}$, $R = \frac{I\gamma(1-\delta)}{\mu}$ and $D = \frac{I\gamma\delta}{\alpha}$. Substituting these values in the first three equations, the system becomes

$$\begin{aligned}
S\left(I\left(\beta_1 + \frac{\beta_2\gamma\delta}{\alpha}\right) + \mu\right) &= \Lambda\rho,\\
IV\left(\beta_1 + \frac{\beta_2\gamma\delta}{\alpha}\right)\eta + V\mu &= \Lambda(1-\rho),\\
\frac{(\gamma+\mu)(\mu+\sigma)}{\sigma} &= \left(\beta_1 + \frac{\beta_2\gamma\delta}{\alpha}\right)(S + \eta V).
\end{aligned} \tag{3}$$

Solving the first two equations of (3) for S and V in terms of I, we get $S = \frac{\alpha\Lambda\rho}{I(\alpha\beta_1+\beta_2\gamma\delta)+\alpha\mu}$ and $V = \frac{\alpha\Lambda(1-\rho)}{I(\alpha\beta_1+\beta_2\gamma\delta)\eta+\alpha\mu}$. Substituting these values in the third equation of (3) we get the quadratic equation

$$aI^2 + bI + c = 0,$$

where

$$\begin{aligned}
a &= (\gamma+\mu)(\mu+\sigma)(\alpha\beta_1 + \beta_2\gamma\delta)^2\eta,\\
b &= (\alpha\beta_1 + \beta_2\gamma\delta)\left(\alpha\mu(1+\eta)(\gamma+\mu)(\mu+\sigma) - \eta\Lambda\sigma(\alpha\beta_1 + \beta_2\gamma\delta)\right),\\
&= (\alpha\beta_1 + \beta_2\gamma\delta)\left(\alpha\mu(1+\eta)(\gamma+\mu)(\mu+\sigma) - \Lambda\sigma(\alpha\beta_1 + \beta_2\gamma\delta)(\eta+\rho-\eta\rho)\right.\\
&\quad \left. + \Lambda\sigma(\rho-\eta\rho)(\alpha\beta_1 + \beta_2\gamma\delta)\right),\\
&= (\alpha\beta_1 + \beta_2\gamma\delta)(\alpha\mu\eta(\gamma+\mu)(\mu+\sigma) + \alpha\mu(\gamma+\mu)(\mu+\sigma)(1-\mathscr{R}_0)\\
&\quad + \Lambda\sigma\rho(1-\eta)(\alpha\beta_1 + \beta_2\gamma\delta)),\\
c &= -\alpha\mu(\alpha\beta_1 + \beta_2\gamma\delta)\Lambda\sigma(\eta(1-\rho)+\rho) + \alpha^2\mu^2(\gamma+\mu)(\mu+\sigma),\\
&= \alpha^2\mu^2(\gamma+\mu)(\mu+\sigma)(1-\mathscr{R}_0).
\end{aligned}$$

Clearly, $c < 0$ holds if and only if $\mathscr{R}_0 > 1$. As $a > 0$ independently of the parameters, using Vieta's formulas, we obtain that for $\mathscr{R}_0 \geq 1$, there is exactly one positive solution of the quadratic equation, while if $\mathscr{R}_0 < 1$, there is no positive solution. Therefore, there is no endemic equilibrium if $\mathscr{R}_0 < 1$ and there exists a unique endemic equilibrium if $\mathscr{R}_0 \geq 1$.

4.2. Local stability of the equilibria

Theorem 2. *The disease-free equilibrium* $E_0\left(\frac{\rho\Lambda}{\mu}, \frac{(1-\rho)\Lambda}{\mu}, 0, 0, 0, 0\right)$ *is locally asymptotically stable if* $\mathscr{R}_0 < 1$*, while* E_0 *is unstable if the inequality is altered.*

Proof. The Jacobian of system (2) evaluated in disease-free equilibrium takes the form

$$\mathscr{J}(E_0) = \begin{bmatrix} -\mu & 0 & 0 & -\frac{\beta_1\Lambda\rho}{\mu} & 0 & -\frac{\beta_2\Lambda\rho}{\mu} \\ 0 & -\mu & 0 & -\frac{\beta_1\eta\Lambda(1-\rho)}{\mu} & 0 & -\frac{\beta_2\eta\Lambda(1-\rho)}{\mu} \\ 0 & 0 & -\mu-\sigma & \frac{\beta_1\Lambda(\eta+\rho-\eta\rho)}{\mu} & 0 & \frac{\beta_2\Lambda(\eta+\rho-\eta\rho)}{\mu} \\ 0 & 0 & \sigma & -\gamma-\mu & 0 & 0 \\ 0 & 0 & 0 & \gamma-\gamma\delta & -\mu & 0 \\ 0 & 0 & 0 & \gamma\delta & 0 & -\alpha \end{bmatrix},$$

The system is locally asymptotically stable if all the eigenvalues of $\mathscr{J}(E_0)$ have a negative real part. The characteristic equation of $\mathscr{J}(E_0)$ is

$$\Phi(\lambda) := (\lambda+\mu)^3(\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3) = 0,$$

where

$$C_1 = \alpha+\gamma+2\mu+\sigma > 0,$$

$$C_2 = (\gamma+\mu)(\mu+\sigma)(1-\mathscr{R}_0) + \alpha\sigma + \alpha\gamma + 2\alpha\mu + \frac{\beta_2\Lambda\gamma\delta(\eta(1-\rho)+\rho)}{\alpha\mu},$$

$$C_3 = (\alpha\gamma\mu + \alpha\mu^2 + \alpha\gamma\sigma + \alpha\mu\sigma)(1-\mathscr{R}_0)$$

Here C_2 and C_3 will be positive if $\mathscr{R}_0 < 1$, Furthermore,

$$\begin{aligned} C_1C_2 - C_3 &= (\alpha+\gamma+\mu+\sigma)\left[(\gamma+\mu)(\mu+\sigma)(1-\mathscr{R}_0) + \alpha\sigma + \alpha\gamma \right. \\ &\quad \left. + 2\alpha\mu + \tfrac{\beta_2(\eta\Lambda(1-\rho)+\Lambda\rho)}{\alpha\mu}\right] \\ &\quad - \left[(\alpha\gamma\mu + \alpha\mu^2 + \alpha\gamma\sigma + \alpha\mu\sigma)(1-\mathscr{R}_0)\right] \\ &= \left[(\alpha+\gamma+\mu+\sigma)(\gamma+\mu)(\mu+\sigma)\right. \\ &\quad \left. - (\alpha\gamma\mu + \alpha\mu^2 + \alpha\gamma\sigma + \alpha\mu\sigma)\right](1-\mathscr{R}_0) \\ &\quad + (\alpha+\gamma+\mu+\sigma)\left[\alpha\sigma + \alpha\gamma + 2\alpha\mu + \tfrac{\beta_2(\eta\Lambda(1-\rho)+\Lambda\rho)}{\alpha\mu}\right] \\ &= \left[\gamma^2\mu + 2\gamma\mu^2 + \mu^3 + \gamma^2\sigma + 3\gamma\mu\sigma \right. \\ &\quad \left. + 2\mu^2\sigma + \gamma\sigma^2 + \mu\sigma^2\right](1-\mathscr{R}_0) \\ &\quad + (\alpha+\gamma+\mu+\sigma)\left[\alpha\sigma + \alpha\gamma + 2\alpha\mu + \tfrac{\beta_2(\eta\Lambda(1-\rho)+\Lambda\rho)}{\alpha\mu}\right] \end{aligned}$$

Again $C_1C_2 - C_3 > 0$ if $\mathscr{R}_0 < 1$. Thus, the Routh–Hurwitz criteria are satisfied if $\mathscr{R}_0 < 1$ and in this case, all the eigenvalues of the characteristic equation have negative real parts. Hence, E_0 is stable and is unstable if $\mathscr{R}_0 > 1$. This completes our proof. □

4.3. Global stability of the equilibria

In this subsection, we will show the global asymptotic stability of one of the two equilibria, depending on the basic reproduction number. First, we need the following auxiliary result.

Lemma 3. *For the limit superior of* $S(t)$ *and* $V(t)$*, the inequalities*

$$S^\infty \leq \frac{\rho\Lambda}{\mu} \quad \text{and} \quad V^\infty \leq \frac{(1-\rho)\Lambda}{\mu}$$

hold.

Proof. According to the fluctuation lemma (see e.g. [33]), there exists a sequence $\{t_n\}$ such that $t_n \to \infty$ we have $S(t_n) \to S^\infty$, and $S'(t_n) \to 0$ as $n \to \infty$. Thus, we can say

$$S'(t_n) = \rho\Lambda - (\beta_1 I(t_n) + \beta_2 D(t_n))S(t_n) - \mu S(t_n) \leq \rho\Lambda - \mu S(t_n),$$

which implies

$$0 \leq \rho\Lambda - \mu S^\infty$$

and from this

$$S^\infty \leq \frac{\rho\Lambda}{\mu}.$$

The other inequality can be shown in an analogous way. □

Theorem 4. *The disease-free equilibrium* $E_0\left(\frac{\rho\Lambda}{\mu}, \frac{(1-\rho)\Lambda}{\mu}, 0, 0, 0, 0\right)$ *is globally asymptotically stable in* $\Gamma := \{(S, V, E, I, R, D) \in \mathbb{R}^6_+\}$ *if* $\mathscr{R}_0 < 1$.

Proof. From the calculation of the basic reproduction number for our model, we have the matrices $\mathscr{F}, \mathscr{V}, F$, and V associated with system (2). One can easily calculate

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu+\sigma} & 0 & 0 \\ \frac{\sigma}{(\gamma+\mu)(\mu+\sigma)} & \frac{1}{\gamma+\mu} & 0 \\ \frac{\gamma\delta\sigma}{\alpha(\gamma+\mu)(\mu+\sigma)} & \frac{\gamma\delta}{\alpha(\gamma+\mu)} & \frac{1}{\alpha} \end{bmatrix}.$$

Following [34, Theorem 2.1] and using the notations therein, we have the disease compartments $x = (E, I, D)$ and the disease-free compartments $y = (S, V, R)$. Then we define the function $\phi(S, V, E, I, R, D)$ appearing in [34, Theorem 2.1] in the form

$$\begin{aligned} \phi(S, V, E, I, R, D)^T &= (F-V)x - \mathscr{F}(x, y) + \mathscr{V}(x, y) \\ &= \left(\frac{(\beta_1 I + \beta_2 D)(\eta\Lambda(1-\rho) + \Lambda\rho - \mu S - \eta\mu V)}{\mu}, 0, 0\right). \end{aligned}$$

The function ϕ will be positive if and only if $S + \eta V < \frac{\Lambda\rho}{\mu} + \frac{\Lambda\eta(1-\rho)}{\mu}$ holds. However, from Lemma 3 we know that for any $\epsilon > 0$ there exists a T large enough such that $S < \frac{\Lambda\rho}{\mu} + \epsilon$ and $V < \frac{\Lambda(1-\rho)}{\mu} + \epsilon$ for all $t > T$. Therefore, the function ϕ will be positive for t large enough. Therefore, each condition mentioned in the above theorem is satisfied, and $\phi > 0$ for $t > T$. Let $\omega^T \geq 0$ be the left eigenvector of the nonnegative matrix $V^{-1}F$ corresponding to $\mathscr{R}_0$. It follows from the theorem that if $\phi \geq 0, F \geq 0, V^{-1} \geq 0$ and $\mathscr{R}_0 \leq 1$ then the function $\omega^T V^{-1}x$, where x stands for the infectious compartments, is a Lyapunov function for the model (2). Hence the theorem is proved using LaSalle's invariance principle [35]. □

Theorem 5. *The endemic equilibrium* $E^* := \left(S^*, V^*, E^*, I^*, R^*, D^*\right)$ *is globally asymptotically stable in* $\Gamma := \{(S(t), V(t), E(t), I(t), R(t), D(t)) \in \mathbb{R}^6_+\}$ *if* $\mathscr{R}_0 > 1$.

Proof. Let us define the Lyapunov function $V(t)$ as

$$\begin{aligned} V(t) &= \frac{S^*}{E^*}\left(\frac{S}{S^*} - 1 - \ln\frac{S}{S^*}\right) + \frac{V^*}{E^*}\left(\frac{V}{V^*} - 1 - \ln\frac{V}{V^*}\right) \\ &\quad + \left(\frac{E}{E^*} - 1 - \ln\frac{E}{E^*}\right) \\ &\quad + \frac{(\beta_1 I^* + \beta_2 D^*)(S^* + \eta V^*)I^*}{\sigma(E^*)^2}\left(\frac{I}{I^*} - 1 - \ln\frac{I}{I^*}\right) \\ &\quad + \frac{\beta_2(S^* + \eta V^*)(D^*)^2}{\delta\gamma I^* E^*}\left(\frac{D}{D^*} - 1 - \ln\frac{D}{D^*}\right) \end{aligned}$$

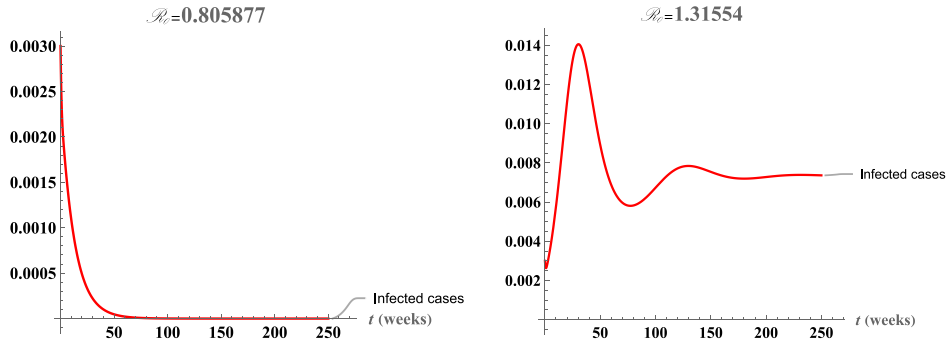
Consider the function $g : \mathbb{R} \to \mathbb{R}$ defined as $g(x) = 1 - x + \ln x$, here $x > 0$ leads to $g(x) \leq 0$, while if $x = 1$ then $g(x) = 0$. So for any $x > 0$ we get $x - 1 \geq \ln x$. The derivative of the Lyapunov function along solutions of system (2) can be calculated as

$$\begin{aligned} V'(t) &= \tfrac{S^*}{E^*}\tfrac{1}{S^*}\left(1 - \tfrac{S^*}{S}\right)S' + \tfrac{V^*}{E^*}\tfrac{1}{V^*}\left(1 - \tfrac{V^*}{V}\right)V' + \tfrac{1}{E^*}\left(1 - \tfrac{E^*}{E}\right)E' \\ &\quad + \tfrac{(\beta_1 I^* + \beta_2 D^*)(S^* + \eta V^*)I^*}{\sigma(E^*)^2}\tfrac{1}{I^*}\left(1 - \tfrac{I^*}{I}\right)I' \end{aligned}$$

$$
\begin{aligned}
&+\frac{\beta_2(S^*+\eta V^*)(D^*)^2}{\delta\gamma I^* E^*}\frac{1}{D^*}\left(1-\frac{D^*}{D}\right)D'\\
&=\frac{S^*}{E^*}\frac{1}{S^*}\left(1-\frac{S^*}{S}\right)\left((\beta_1 I^*+\beta_2 D^*)S^*+\mu S^*-(\beta_1 I+\beta_2 D)S-\mu S\right)\\
&\quad+\frac{V^*}{E^*}\frac{1}{V^*}\left(1-\frac{V^*}{V}\right)(\eta(\beta_1 I^*+\beta_2 D^*)V^*+\mu V^*\\
&\quad-\eta(\beta_1 I+\beta_2 D)V-\mu V)\\
&\quad+\frac{1}{E^*}\left(1-\frac{E^*}{E}\right)\left((\beta_1 I+\beta_2 D)(S+\eta V)-\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)E}{E^*}\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)I^*}{\sigma(E^*)^2}\frac{1}{I^*}\left(1-\frac{I^*}{I}\right)\left(\sigma E-\frac{\sigma E^* I}{I^*}\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)(D^*)^2}{\delta\gamma I^* E^*}\frac{1}{D^*}\left(1-\frac{D^*}{D}\right)\left(\delta\gamma I-\frac{\delta\gamma I^* D}{D^*}\right)\\
&=\frac{S^*}{E^*}\frac{1}{S^*}\left(1-\frac{S^*}{S}\right)\left(\beta_1 I^* S^*\left(1-\frac{IS}{I^*S^*}\right)\right.\\
&\quad\left.+\beta_2 D^* S^*\left(1-\frac{DS}{D^*S^*}\right)+\mu S^*\left(1-\frac{S}{S^*}\right)\right)\\
&\quad+\frac{V^*}{E^*}\frac{1}{V^*}\left(1-\frac{V^*}{V}\right)\left(\eta\beta_1 I^* V^*\left(1-\frac{IV}{I^*V^*}\right)+\eta\beta_2 D^* V^*\left(1-\frac{DV}{D^*V^*}\right)\right.\\
&\quad\left.+\mu V^*\left(1-\frac{V}{V^*}\right)\right)\\
&\quad+\frac{1}{E^*}\left(1-\frac{E^*}{E}\right)\left(\beta_1 I^* S^*\left(\frac{IS}{I^*S^*}-\frac{E}{E^*}\right)+\beta_2 D^* S^*\left(\frac{DS}{D^*S^*}-\frac{E}{E^*}\right)\right)\\
&\quad+\frac{1}{E^*}\left(1-\frac{E^*}{E}\right)\left(\eta\beta_1 I^* V^*\left(\frac{IV}{I^*V^*}-\frac{E}{E^*}\right)\right.\\
&\quad\left.+\eta\beta_2 D^* V^*\left(\frac{DV}{D^*V^*}-\frac{E}{E^*}\right)\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)I^*}{\sigma(E^*)^2}\frac{1}{I^*}\left(1-\frac{I^*}{I}\right)\sigma E^*\left(\frac{E}{E^*}-\frac{I}{I^*}\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)(D^*)^2}{\delta\gamma I^* E^*}\frac{1}{D^*}\left(1-\frac{D^*}{D}\right)\delta\gamma I^*\left(\frac{I}{I^*}-\frac{D}{D^*}\right)\\
&\le\frac{\beta_1 I^* S^*}{E^*}\left(1-\frac{S^*}{S}-\frac{IS}{I^*S^*}+\frac{I}{I^*}\right)+\frac{\beta_2 D^* S^*}{E^*}\left(1-\frac{S^*}{S}-\frac{DS}{D^*S^*}+\frac{D}{D^*}\right)\\
&\quad+\frac{\eta\beta_1 I^* V^*}{E^*}\left(1-\frac{V^*}{V}-\frac{IV}{I^*V^*}+\frac{I}{I^*}\right)\\
&\quad+\frac{\eta\beta_2 D^* V^*}{E^*}\left(1-\frac{V^*}{V}-\frac{DV}{D^*V^*}+\frac{D}{D^*}\right)\\
&\quad+\frac{\beta_1 I^* S^*}{E^*}\left(\frac{IS}{I^*S^*}-\frac{E^*IS}{EI^*S^*}-\frac{E}{E^*}+1\right)\\
&\quad+\frac{\beta_2 D^* S^*}{E^*}\left(\frac{DS}{D^*S^*}-\frac{E^*DS}{ED^*S^*}-\frac{E}{E^*}+1\right)\\
&\quad+\frac{\eta\beta_1 I^* V^*}{E^*}\left(\frac{IV}{I^*V^*}-\frac{E^*IV}{EI^*V^*}-\frac{E}{E^*}+1\right)\\
&\quad+\frac{\eta\beta_2 D^* V^*}{E^*}\left(\frac{DV}{D^*V^*}-\frac{E^*DV}{ED^*V^*}-\frac{E}{E^*}+1\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)}{E^*}\left(\frac{E}{E^*}-\frac{I^*E}{IE^*}-\frac{I}{I^*}+1\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)D^*}{E^*}\left(\frac{I}{I^*}-\frac{D^*I}{DI^*}-\frac{D}{D^*}+1\right)\\
&\le\frac{\beta_1 I^* S^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-1-\frac{S^*}{S}+\ln\frac{S}{S^*}+1-\frac{IS}{I^*S^*}+\ln\frac{IS}{I^*S^*}+1\right)\\
&\quad+\frac{\beta_2 D^* S^*}{E^*}\left(\frac{D}{D^*}-\ln\frac{D}{D^*}-1-\frac{S^*}{S}\right.\\
&\quad\left.+\ln\frac{S}{S^*}+1-\frac{DS}{D^*S^*}+\ln\frac{DS}{D^*S^*}+1\right)\\
&\quad+\frac{\eta\beta_1 I^* V^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-1-\frac{V^*}{V}+\ln\frac{V}{V^*}\right.\\
&\quad\left.+1-\frac{IV}{I^*V^*}+\ln\frac{IV}{I^*V^*}+1\right)\\
&\quad+\frac{\eta\beta_2 D^* V^*}{E^*}\left(\frac{D}{D^*}-\ln\frac{D}{D^*}-1-\frac{V^*}{V}+\ln\frac{V}{V^*}\right.\\
&\quad\left.+1-\frac{DV}{D^*V^*}+\ln\frac{DV}{D^*S^*}+1\right)\\
&\quad+\frac{\beta_1 I^* S^*}{E^*}\left(\frac{IS}{I^*S^*}-\ln\frac{IS}{I^*S^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\beta_2 D^* S^*}{E^*}\left(\frac{DS}{D^*S^*}-\ln\frac{DS}{D^*S^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\eta\beta_1 I^* V^*}{E^*}\left(\frac{IV}{I^*V^*}-\ln\frac{IV}{I^*V^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\eta\beta_2 D^* V^*}{E^*}\left(\frac{DV}{D^*V^*}-\ln\frac{DV}{D^*V^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)}{E^*}\left(\frac{E}{E^*}-\ln\frac{E}{E^*}-\frac{I}{I^*}+\ln\frac{I}{I^*}\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)D^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{D}{D^*}+\ln\frac{D}{D^*}\right)\\
&=\frac{\beta_1 I^* S^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\beta_2 D^* S^*}{E^*}\left(\frac{D}{D^*}-\ln\frac{D}{D^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\eta\beta_1 I^* V^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\eta\beta_2 D^* V^*}{E^*}\left(\frac{D}{D^*}-\ln\frac{D}{D^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)}{E^*}\left(\frac{E}{E^*}-\ln\frac{E}{E^*}-\frac{I}{I^*}+\ln\frac{I}{I^*}\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)D^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{D}{D^*}+\ln\frac{D}{D^*}\right)\\
&=\frac{\beta_1 I^*(S^*+\eta V^*)}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\beta_2 D^*(S^*+\eta V^*)}{E^*}\left(\frac{D}{D^*}-\ln\frac{D}{D^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)}{E^*}\left(\frac{E}{E^*}-\ln\frac{E}{E^*}-\frac{I}{I^*}+\ln\frac{I}{I^*}\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)D^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{D}{D^*}+\ln\frac{D}{D^*}\right)\\
&=\frac{\beta_1 I^*(S^*+\eta V^*)}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{\beta_2 D^*(S^*+\eta V^*)}{E^*}\left(-\frac{E}{E^*}+\ln\frac{E}{E^*}\right)\\
&\quad+\frac{(\beta_1 I^*+\beta_2 D^*)(S^*+\eta V^*)}{E^*}\left(\frac{E}{E^*}-\ln\frac{E}{E^*}-\frac{I}{I^*}+\ln\frac{I}{I^*}\right)\\
&\quad+\frac{\beta_2(S^*+\eta V^*)D^*}{E^*}\left(\frac{I}{I^*}-\ln\frac{I}{I^*}\right)\\
&=0.
\end{aligned}
$$

From the previous calculation, it is clear that $V'(t) \le 0$. Furthermore, the equality $V'(t) = 0$ holds only if $S = S^*, V = V^*, E = E^*, I = I^*$, and $D = D^*$. Thus, the endemic equilibrium E^*, is the only positive invariant set to the system (2) contained entirely in $\Gamma := \{(S(t), V(t), E(t), I(t), R(t), D(t)) \in \mathbb{R}^6_+\}$. Therefore, it follows from LaSalle's invariance principle [35] that every solution of system (2) with initial conditions in Γ converges to the endemic equilibrium point, E^*, as $t \to \infty$. Hence, the positive equilibrium is globally asymptotically stable if $\mathscr{R}_0 > 1$. □

To support our analytical results, we present some numerical simulations showing the two possible scenarios concerning the global dynamics of system (2). We chose baseline parameter values realistic for Ebola and shown in Table 2 with the exception of Λ and μ, which are chosen to have the value $\Lambda = \mu = 0.0388$ as, for better visibility of the results, we decided to scale the population to 1 in this simulation. With these parameter values, we obtain $\mathscr{R}_0 = 1.31554$, corresponding to the disease becoming endemic and the endemic equilibrium being globally asymptotically stable. Changing the values of the two transmission rates to $\beta_1 = 0.273$ and $\beta_2 = 1.226$, the basic reproduction number is decreased to 0.805877, hence, in this case, the disease dies out and the disease-free equilibrium is globally asymptotically stable. The case of the disease dying out is shown in Fig. 2(a), while the situation of the disease becoming endemic in the population is depicted in Fig. 2(b).



(a) Global asymptotic stability of the disease-free equilibrium for $\mathscr{R}_0 < 1$.

(b) Global asymptotic stability of the endemic equilibrium for $\mathscr{R}_0 > 1$.

Fig. 2. Global dynamics of model (2) for different values of $\mathscr{R}_0$.

Table 2
Parameters value of the model (2).

Parameters	Ebola and Ref.	COVID-19 and Ref.	Nipah and Ref.
Λ	11 826/week [36]	352/day [36]	19.57/day [36]
μ	0.00054/week [36]	0.000036/day [36]	0.00004145/day [36]
β_1	2.375×10^{-8}/week [37]	1.14×10^{-8}/day [38]	5.93×10^{-7}/day [39,40]
β_2	9.71×10^{-8}/week [16,19]	2×10^{-9}/day [Assumed]	5.08×10^{-7}/day [20,40]
η	–	0.03 [41]	–
$1/\sigma$	1.498 weeks [30,42]	5.2 days [43]	8.34 days [44]
$1/\gamma$	0.175 weeks [45]	10 days [46]	11.1 days [39]
$1/\alpha$	0.762 weeks [47]	3 days [Assumed]	1.71 days [Assumed]
δ	0.69 [30,48]	0.02 [49]	0.76 [20]

5. Assessing the effects of transmission by deceased on disease spread

In this section, we perform numerical simulations to assess the effects of disease transmission via contact with the corpses of those deceased due to an infectious disease. Using the baseline values for the available parameter values as listed in Table 2, we utilize the Latin Hypercube Sampling method to find the parameter values which provide the best fit to the data. This is a computational technique used in statistics to estimate the simultaneous variation of various model parameters to construct a representative sample set of n-tuples of parameters (n is the number of parameters fitted) taking values from given ranges. The estimated values of the fitted parameters of the model are given in Table 2. Although, as mentioned in the introduction, there is a risk of becoming infected this way with several infectious diseases, however, the situation might be very different in various cases. Hence, in our simulations, we will consider three infectious diseases with significantly different concerns regarding transmission from the deceased. The three diseases studied in this section are Ebola virus disease, COVID-19, and Nipah fever.

The role of transmission from the deceased is widely known in the case of Ebola as during the large outbreak in 2014 outbreak, several articles in the news reported about the traditional funeral ceremonies which included touching and kissing the deceased. For this reason, we chose this disease as our first example, considering a baseline scenario similar to the first weeks of 2014–16 epidemic in Liberia, Sierra Leone, and Guinea. This situation corresponds to a case where transmission from the deceased highly contributes to the number of new infections, while the total number of infected is moderate with respect to the total population. Furthermore, as there was no vaccine available at the time of the epidemic, we exclude vaccination from our model in this case and use model (2) for the simulation. Fig. 3 shows the solution of model (2) with parameters given in Table 2 applied to Ebola data of the first 38 weeks of the 2015 epidemic in Guinea, Liberia, and Sierra Leone. Fig. 4 shows the expected result of changes in parameter values connected to transmission from the deceased. Namely, we consider a

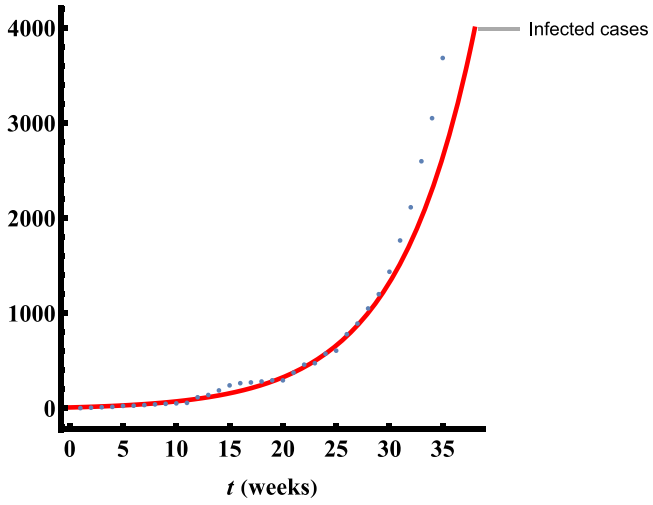
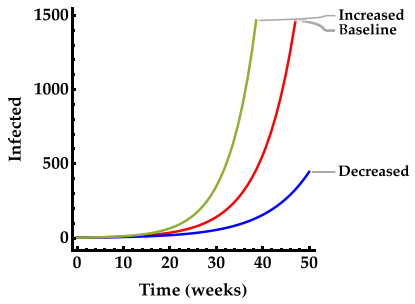
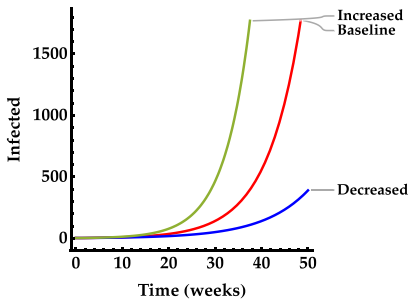


Fig. 3. Model (2) applied to Ebola data of the first 38 weeks of the 2015 epidemic in Guinea, Liberia, and Sierra Leone.

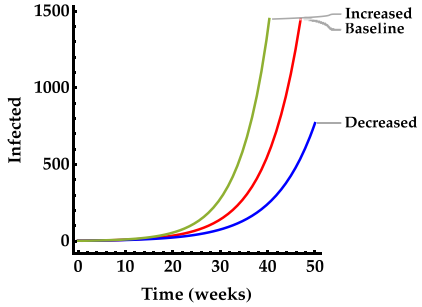
change in the transmission rate β_2, corresponding to a reduction of contacts with deceased and decreasing the probability of transmission by increasing hygiene, then a change in the average time until burial, $1/\alpha$, and finally, a parallel change of the two parameters. The three panels of the figure compare the baseline situation to an increase and a decrease of the parameters. The results suggest that the transmission rate from the deceased is a very important parameter in view of the number of infected: an increase of this value can result in much higher numbers of cases, while a successful introduction of an intervention measure affecting this parameter can be very useful in reducing disease burden (see Fig. 4(a)). Fig. 4(b) suggests that although less impactful than β_2, the time until the burial is also an important parameter, and encouraging fast and safe burials might save plenty of people from infection.



(a) Baseline $\beta_2 = 8.82 \times 10^{-8}$. Increased $\beta_2 = 9.82 \times 10^{-8}$. Decreased $\beta_2 = 7.82 \times 10^{-8}$.



(b) Baseline $\alpha = 1.31$. Increased $\alpha = 1.11$. Decreased $\alpha = 1.51$.



(c) Baseline $\beta_2 = 8.82 \times 10^{-8}, \alpha = 1.31$. Increased $\beta_2 = 8.92 \times 10^{-8}, \alpha = 1.21$. Decreased $\beta_2 = 8.72 \times 10^{-8}, \alpha = 1.41$.

Fig. 4. Number of Ebola virus infected cases for changing β_2 and α along with their parallel application.

Another recent epidemic where transmission from the deceased is possible is the COVID-19 pandemic [50,51], for this disease, we take the situation in Hungary in spring 2021, after the introduction of vaccination as an example. So we have used model (1) for the simulation (see Fig. 5). Although various reports have confirmed this way of transmission, it is much less important than in the case of Ebola. One can identify two main possibilities for disease transmission from deceased: one is for those who lived in the same household with the deceased individual, and another way is in health care facilities. The latter is especially crucial in case of a large epidemic when the health system is overwhelmed and it is difficult to handle the corpses. Such situations arose in several countries during the COVID pandemic [26,52,53]. Unlike Ebola, in this case, it is not the high transmission rate, but the high number of infections and victims that provide a risk of the occurrence of an elevated number of infections due to contact with the infected deceased. Accordingly, as our simulations shown in Fig. 6 suggest, in comparison with the total number of infected, infections via contact with victims of the epidemic are relatively small. However, due to the magnitude of the pandemic, even in this case, several cases and deaths can be spared if proper attention is paid to avoiding direct contact with victims of the epidemic.

Our third example is Nipah fever, a highly lethal emerging disease that appears in the WHO Blueprint list of epidemic threats needing urgent R&D action [28]. Drinking raw date palm sap, contaminated by Virus from urine or saliva of *Pteropus* fruit bats, is one of the main transmission routes to humans in Bangladesh, however, it was reported that corpse-to-human transmission is also an important way of disease spread due to caregivers being exposed to bodily secretions of infected deceased during ritual bathing of the corps and traditional burial practices [28]. In comparison with the other two diseases, the number of infections is much lower, however, according to various studies (see [20,28,40]), transmission from deceased might significantly contribute to new infections, like Ebola, so we have used model

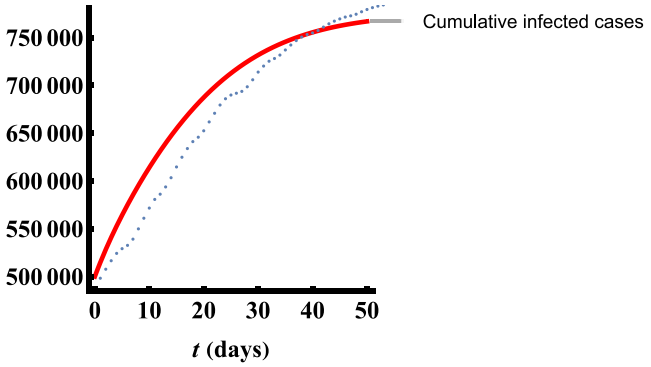
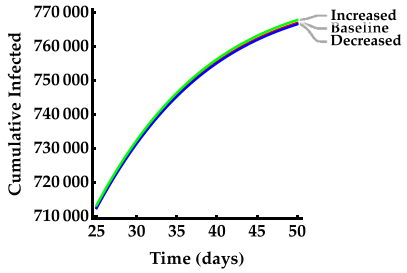


Fig. 5. Model (1) applied to COVID-19 data from spring 2021 in Hungary.

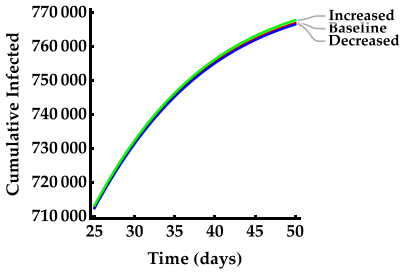
(2) for simulation. As there is no certified vaccine for NiV, we chose the Siliguri outbreak in early 2001 as an example, a solution approximating data of this epidemic [54] is shown in Fig. 7. Although less significant than in the case of Ebola, again we can see important changes in the number of infections. Even a small increase in the value of both parameters can increase the number of infected individuals significantly in comparison with the total number of infected (see Fig. 8).

5.1. PRCC analysis

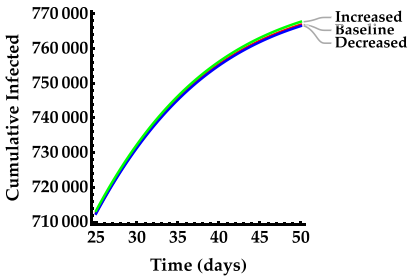
To compare the effects of varying the values of deceased-related parameters with those of other parameters, we performed PRCC analysis, a statistical measure used to determine the strength and direction of the linear relationship between two variables, while controlling for the effects of other variables. The results for all three epidemics are shown in Fig. 9. One can see that the PRCC values are in accordance



(a) Baseline $\beta_2 = 2.08 \times 10^{-9}$. Increased $\beta_2 = 5.28 \times 10^{-9}$. Decreased $\beta_2 = 0$.



(b) Baseline $\alpha = 0.333$. Increased $\alpha = 0.5$. Decreased $\alpha = 12$.



(c) Baseline $\beta_2 = 2.08 \times 10^{-10}, \alpha = 0.333$. Increased $\beta_2 = 5.2 \times 10^{-10}, \alpha = 0.5$. Decreased $\beta_2 = 0, \alpha = 12$.

Fig. 6. Number of COVID-19 cumulative infected cases for changing β_2 and α along with their parallel application.

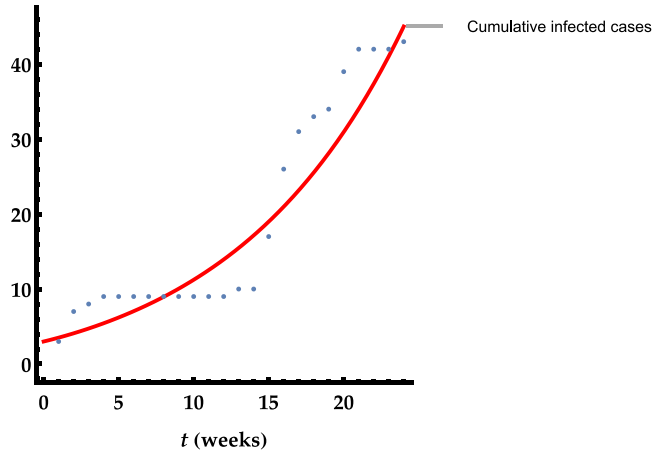


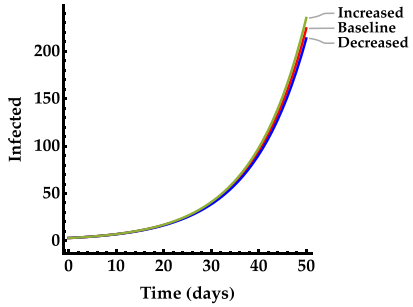
Fig. 7. Model (2) applied to the 2001 Nipah outbreak in Siliguri, India.

with the results shown in the simulations of the previous subsection. The values show that among the three diseases, it is Ebola for which transmission from deceased contributes the most to the number of infections, while for COVID-19, a very mild effect is shown. The same holds for the average time of burial. As for parameters unrelated to transmission from deceased, we see that transmission from infected has the highest impact, while the length of infectious period is shown to be less important for Ebola, while significant for the other two diseases. Most certainly, the vaccination rate has a very high negative effect on the number of infections in the case of COVID-19.

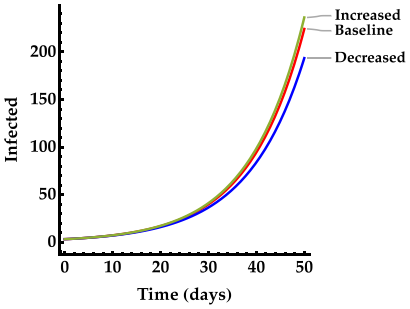
6. Conclusions

We established a compartmental model to assess the importance of transmission due to contact with victims of an epidemic, a phenomenon known to occur in many infectious diseases, including Ebola haemorrhagic fever, COVID-19, and plague. The model also includes vaccination, one of the most important tools to protect ourselves from infection. In our work, vaccination is assumed to be imperfect, i.e., those who have received the vaccine can still become infected, however, with a lower probability. We first performed theoretical analysis for a special case of the model, namely, when vaccination takes place after birth and vaccination of adults is neglected. After determining some basic properties of the model and calculating the basic reproduction number, we applied a result by van den Driessche and Shuai to show global asymptotic stability of the disease-free equilibrium in the case $\mathscr{R}_0 < 1$, while constructing an appropriate Lyapunov function allowed us to prove the same for the endemic equilibrium in case $\mathscr{R}_0 > 1$.

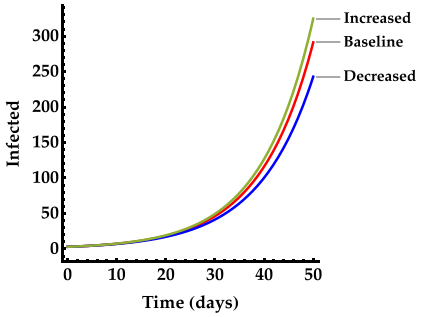
Following the analytical results, we performed numerical simulations to estimate the disease burden due to infection via contact with deceased individuals. To do so, we selected three recent epidemics with different characteristics. The three diseases chosen were Ebola, COVID-19, and Nipah fever. Both Ebola and Nipah fever are known for an important contribution of infections by deceased to the total number of cases. This phenomenon is less typical for COVID-19. For this latter disease, it is the immense number of cases that might result in a significant number of infections caused by contact with deceased infected. On the other hand, up to now, the world has not experienced Ebola or Nipah outbreaks of the scale of the COVID pandemic. The numerical results are in accordance with the known characteristics of the diseases and show that in the case of Ebola and Nipah, where traditional funeral ceremonies contribute to transmission from deceased, this way of spread might result in a significant increase of the number of infected. People should keep away from contact with the bodies of people who have died from Ebola and avoid funeral or burial practices that involve touching the body of someone who is suspected or confirmed to have had Ebola disease. At the same time, the simulations suggest that for such epidemics, a very efficient way to reduce the epidemic spread is to diminish this way of transmission as much as possible. On the contrary, generalizing the results of our simulations regarding the COVID-19 epidemic, we may conclude that if corpses are handled in a safe and adequate way and contact of susceptibles



(a) Baseline $\beta_2 = 0.00000051$. Increased $\beta_2 = 0.00000057$. Decreased $\beta_2 = 0.00000044$.

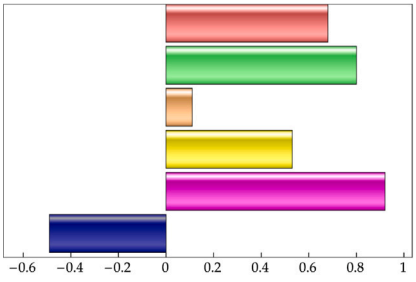


(b) Baseline $\alpha = 0.583336$. Increased $\alpha = 0.5$. Decreased $\alpha = 1$.

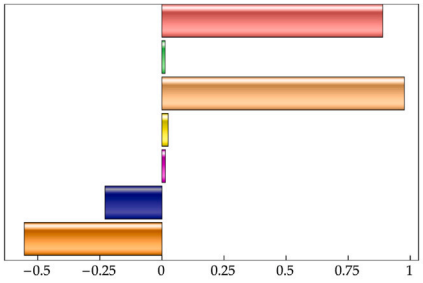


(c) Baseline $\beta_2 = 0.00000051, \alpha = 0.583336$. Increased $\beta_2 = 0.00000057, \alpha = 0.5$. Decreased $\beta_2 = 0.00000044, \alpha = 1$.

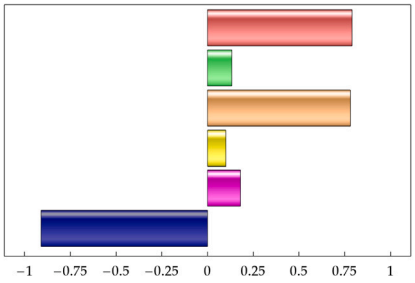
Fig. 8. Number of Nipah virus infected cases for changing β_2 and α along with their parallel application.



(a) PRCC for Ebola.



(b) PRCC for COVID-19.



(c) PRCC for Nipah.

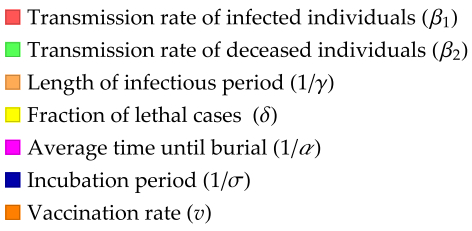


Fig. 9. Partial Rank Correlation Coefficients (PRCC).

with them is reduced, then even in the case of a large-scale epidemic, one may more or less eliminate the contribution of deceased to disease spread. Furthermore, guidelines from WHO and CDC for an epidemic are to be followed to eradicate the disease.

Our work certainly has its limitations. First of all, we decided to create a general model which might not include some characteristics of a special disease. However, this allowed us to obtain analytical results on the global dynamics of the system which might not be possible in case of a very complex model taking into account all specialties of the given disease. Establishing and studying such more realistic models can be considered a future work.

Declaration of competing interest

None.

Data availability

Data will be made available on request.

Acknowledgments

This research was supported by project TKP2021-NVA-09, implemented with the support provided by the Ministry for Innovation and Technology of Hungary from the National Research, Development and Innovation Fund, financed under the TKP2021-NVA funding scheme. S.B. was supported by Stipendium Hungaricum scholarship and Ministry of Education, Government of Bangladesh. A.D. was supported by the National Laboratory for Health Security, RRF-2.3.1-21-2022-00006 and by the projects no. 129877 and no. 125119 of the National Research, Development and Innovation Office of Hungary, financed under the KKP_19 and SNN_17 funding schemes, respectively.

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