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Research article

On a two-strain epidemic model involving delay equations

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Abstract: We propose an epidemiological model for the interaction of either two viruses or viral strains with cross-immunity, where the individuals infected by the first virus cannot be infected by the second one, and without cross-immunity, where a secondary infection can occur. The model incorporates distributed recovery and death rates and consists of integro-differential equations governing the dynamics of susceptible, infectious, recovered, and dead compartments. Assuming that the recovery and death rates are uniformly distributed in time throughout the duration of the diseases, we can simplify the model to a conventional ordinary differential equation (ODE) model. Another limiting case arises if the recovery and death rates are approximated by the delta-function, thereby resulting in a new point-wise delay model that incorporates two time delays corresponding to the durations of the diseases. We establish the positiveness of solutions for the distributed delay models and determine the basic reproduction number and an estimate for the final size of the epidemic for the delay model. According to the results of the numerical simulations, both strains can coexist in the population if the disease transmission rates for them are close to each other. If the difference between them is sufficiently large, then one of the strains dominates and eliminates the other one.

Keywords: epidemic model; distributed recovery and death rates; disease duration; time delay

1. Introduction

Mathematical modeling plays a crucial role in comprehending the transmission dynamics of infectious diseases, predicting the future trajectory of outbreaks, and assessing strategies for epidemic control [1]. Its significance has been underscored by successive epidemics of viral

infections, such as HIV since the 1980s [2, 3], the SARS epidemic in 2002–2003 [4, 5], H5N1 influenza in 2005 [6, 7], H1N1 in 2009 [8, 9], Ebola in 2014 [10, 11], and the recent COVID-19 pandemic [12, 13].

Following the classical SIR (susceptible-infected-recovered) model developed by Kermack and McKendrick after the Spanish flu epidemic [14], several notable advancements have been made in epidemiological modeling. These advancements include the development of multi-compartment models [15–19], models incorporating either time-varying or non-linear disease transmission rates [20, 21], multi-patch models [22, 23], and multi-group models that consider the effect of disease and population heterogeneity [24]. Additionally, there are epidemic models that incorporate vaccinations and other control measures [25–30]. Moreover, spatio-temporal models have been employed to describe the random movement of individuals and the spatial distribution of infections [31, 32]. In [33], the authors investigated a model that integrated asymptomatic and dead-infective subpopulations, along with vaccination, antiviral treatment controls, and impulsive culling actions. Reference [34] is devoted to multiple pathogen interactions in disease transmission with a susceptible-infected-susceptible (SIS) model for each pathogen. For a comprehensive review of the literature, monographs [35, 36] and review articles in [37, 38] are available.

Time delays related to infection latency, disease duration and immunity waning directly influence disease transmission dynamics, response strategies, and the overall control of infectious outbreaks [39–42]. Despite advancements, limited research has been conducted on the impact of time delays in recovery and death rates within epidemiological models. In [43], the authors introduced a compartmental epidemiological model that incorporated distributed recovery and death rates. Under certain assumptions, this model can be reduced to the conventional SIR model. However, in most cases, the dynamics of the epidemic progression in this new model exhibit notable distinctions. To determine the distributed recovery and death rates, the authors evaluated COVID-19 data. By validating the model with epidemiological data from various countries, it demonstrated an improved agreement with the data compared to the SIR model. Furthermore, the authors estimated the time-dependent disease transmission rate. In [44], the authors utilized the average disease duration as the delay parameter to introduce a time delay in the recovery and death rates. The distributed model was discussed, and a delay model was derived. Epidemic characterization of the delay model was obtained. A numerical comparison was conducted among the distributed model, delay model, and conventional SIR model. Additionally, a method to estimate the disease duration using real disease incidence data was presented. The authors validated the delay model using epidemiological data from the COVID-19 epidemic and discussed the main outcomes and epidemiological implications of the proposed model. Moreover, apart from [44], the model proposed in [45] incorporated the infectivity period and disease duration instead of the incubation period, and did not include mandatory quarantine. This model considered the presence of exposed individuals and incorporated two time delays, namely the infectivity and disease duration.

In this study, we investigate epidemic models of either two viruses or viral strains. In the first model, we consider the presence of two viral strains, assuming that individuals recovered from either strain developed immunity to both strains. In the second model, we explore the scenario where individuals who have recovered from the first strain can be infected by the second strain,

and similarly, individuals who have recovered from the second strain can be infected by the first strain. Here, we use the aforementioned modeling approach, based on distributed and point-wise delays in recovery and death rates.

The paper is structured as follows. In the first part, we introduce the model with distributed recovery and death rates in the presence of cross-immunity. We thoroughly examine the positiveness of solutions. Next, we reduce the distributed model to a conventional ODE model. Subsequently, we derive the delay model from the distributed model. Additionally, we determine the characteristics of epidemic progression for the delay model. Moreover, this structure is followed for the model without cross-immunity. The main outcomes of the proposed models and their epidemiological implications are discussed in the concluding section.

2. Model with cross-immunity

The recovery and death rates of infected individuals are influenced by the time elapsed since the onset of the disease. In this section, we will develop a model that takes the number of newly infected individuals and their time-dependent recovery and death rates into account [45]. We will examine various characteristics of this model and demonstrate that the conventional SIR model can be derived from the model under certain assumptions [43–45].

2.1. Model formulation

The following compartments of the total population N are considered:

- $S(t)$ - susceptible individuals. They are not infected and can contract the disease.
- $I_1(t)$ - infected of the first viral strain. They are infectious and can transmit disease.
- $I_2(t)$ - infected of the second viral strain. They are infectious and can transmit disease.
- $R_1(t)$ - recovered from the first viral strain. They have recovered from the infection and are no longer infected. They are immune to both strains.
- $R_2(t)$ - recovered from the second viral strain. They have recovered from the infection and are no longer infected. They are immune to both strains.
- $D_1(t)$ - deceased from the first viral strain.
- $D_2(t)$ - deceased from the second viral strain.

We will use the following parameters and functions:

- The transmission rate of the first viral strain is denoted as β_1, while the transmission rate of the second viral strain is denoted as β_2.
- As a function of time since infection η, the recovery rate of individuals infected with the first viral strain is represented by $r_1(\eta)$. Similarly, as a function of time since infection η, the recovery rate of individuals infected with the second viral strain, is represented by $r_2(\eta)$.
- As a function of time since infection η, the mortality rate of the first viral strain is denoted as $d_1(\eta)$. Likewise, the mortality rate of the second viral strain is represented by $d_2(\eta)$.
- The number of newly infected individuals by the first viral strain and the second viral strain, denoted as $J_1(t)$ and $J_2(t)$, respectively, is determined by the relations $J_1(t) = (\beta_1 S I_1)/N$ and $J_2(t) = (\beta_2 S I_2)/N$, respectively.

- The total number of individuals who have recovered from the first viral strain and the second viral strain at time t is represented by $\int_0^t r_1(t-\eta)J_1(\eta)d\eta$ and $\int_0^t r_2(t-\eta)J_2(\eta)d\eta$, respectively.

- The total number of individuals who have died from the first viral strain and the second viral strain at time t is represented by $\int_0^t d_1(t-\eta)J_1(\eta)d\eta$ and $\int_0^t d_2(t-\eta)J_2(\eta)d\eta$, respectively.

The two-strain model with distributed recovery and death rates has the following form:

$$\frac{dS}{dt} = -J_1(t) - J_2(t), \tag{2.1}$$

$$\frac{dI_1}{dt} = J_1(t) - \int_0^t r_1(t-\eta)J_1(\eta)d\eta - \int_0^t d_1(t-\eta)J_1(\eta)d\eta, \tag{2.2}$$

$$\frac{dI_2}{dt} = J_2(t) - \int_0^t r_2(t-\eta)J_2(\eta)d\eta - \int_0^t d_2(t-\eta)J_2(\eta)d\eta, \tag{2.3}$$

$$\frac{dR_1}{dt} = \int_0^t r_1(t-\eta)J_1(\eta)d\eta, \tag{2.4}$$

$$\frac{dR_2}{dt} = \int_0^t r_2(t-\eta)J_2(\eta)d\eta, \tag{2.5}$$

$$\frac{dD_1}{dt} = \int_0^t d_1(t-\eta)J_1(\eta)d\eta, \tag{2.6}$$

$$\frac{dD_2}{dt} = \int_0^t d_2(t-\eta)J_2(\eta)d\eta. \tag{2.7}$$

The system is subject to the following initial condition:

$$S(0) = S_0 > 0, \;\; I_1(0) = I_{1,0} > 0, \;\; I_2(0) = I_{2,0} > 0, \;\; R_1(0) = R_2(0) = D_1(0) = D_2(0) = 0, \tag{2.8}$$

where $I_{1,0}$ and $I_{2,0}$ are sufficiently small as compared to N. We suppose that the total population is constant :

$$N = S(t) + I_1(t) + I_2(t) + R_1(t) + R_2(t) + D_1(t) + D_2(t). \tag{2.9}$$

2.2. Positiveness of solutions

We establish the positiveness of the solution to this problem on a time interval $t \in [0, T)$, where $T \in (0, +\infty)$. We assume that the recovery and death rates satisfy the following inequalities:

$$\int_\eta^{t_0} (r_1(t-\eta) + d_1(t-\eta))d\eta \leq 1, \tag{2.10}$$

$$\int_\eta^{t_0} (r_2(t-\eta) + d_2(t-\eta))d\eta \leq 1, \tag{2.11}$$

for any $\eta \geq 0$ and $t_0 > \eta$. These assumptions dictate that the number of recoveries and deaths on a given time interval cannot exceed the whole population.

Lemma 1. *If conditions (2.10) and (2.11) are satisfied, then any solution of systems (2.1)–(2.7) with the initial condition (2.8) satisfies the inequality* $0 \leq X \leq N$, *where*

$$X \in \{S(t), I_1(t), I_2(t), R_1(t), R_2(t), D_1(t), D_2(t)\}.$$

Proof. From Eq (2.1), we observe that if there exists a time $t_* > 0$ such that $S(t_*) = 0$, then $\frac{dS}{dt}|_{t=t_*} = 0$. This implies that $S(t) \geq 0$ for $t > 0$. By examining Eqs (2.4)–(2.7), we can deduce that $R_1(t)$, $R_2(t)$, $D_1(t)$, and $D_2(t)$ also remain positive for all values of $t \geq 0$. At $t = 0$, we have $J_1(0) = \beta_1 S_0 I_{1,0}/N > 0$ and $J_2(0) = \beta_2 S_0 I_{2,0}/N > 0$. Let $t_0 > 0$ be such that $J_1(t)$ and $J_2(t)$ remain non-negative for $0 \leq t < t_0$. Next, we have the following:

$$I_1(t_0) = \int_0^{t_0} J_1(t)dt - R_1(t_0) - D_1(t_0),$$
$$I_2(t_0) = \int_0^{t_0} J_2(t)dt - R_2(t_0) - D_2(t_0).$$

Integrating Eqs (2.4)–(2.7) with respect to t from 0 to t_0, and taking into account the initial conditions given in (2.8), we obtain the following:

$$R_1(t_0) + D_1(t_0) = \int_0^{t_0} \Big(\int_0^t (r_1(t-\eta) + d_1(t-\eta))J_1(\eta)d\eta \Big)dt,$$
$$R_2(t_0) + D_2(t_0) = \int_0^{t_0} \Big(\int_0^t (r_2(t-\eta) + d_2(t-\eta))J_2(\eta)d\eta \Big)dt.$$

Changing the order of integration and taking the inequalities (2.10) and (2.11) into account, we discover the following:

$$R_1(t_0) + D_1(t_0) = \int_0^{t_0} \Big(\int_\eta^{t_0} (r_1(t-\eta) + d_1(t-\eta))dt \Big)J_1(\eta)d\eta \leq \int_0^{t_0} J_1(\eta)d\eta, \tag{2.12}$$
$$R_2(t_0) + D_2(t_0) = \int_0^{t_0} \Big(\int_\eta^{t_0} (r_2(t-\eta) + d_2(t-\eta))dt \Big)J_2(\eta)d\eta \leq \int_0^{t_0} J_2(\eta)d\eta. \tag{2.13}$$

It follows from inequalities (2.12) and (2.13) that $I_1(t_0)$ and $I_2(t_0)$ are non-negative. Consequently, $J_1(t_0)$ and $J_2(t_0)$ are also non-negative. This implies that $I_1(t)$, $I_2(t)$, $J_1(t)$, and $J_2(t)$ remain non-negative for all $t \geq 0$. Furthermore, we have the relation (2.9). Hence, any solution to the system lies within the range of 0 and N, there by ensuring the non-negativity of the solution.

2.3. Reduction to the ODE model

In this section, we show how models (2.1)–(2.7) can be reduced to the conventional ODE model. We consider the recovery and death rates in the following form:

$$r_1(t) = \begin{cases} r_{1,0}\,, & 0 \leq t < \tau_1 \\ 0\,, & t > \tau_1 \end{cases}, \quad d_1(t) = \begin{cases} d_{1,0}\,, & 0 \leq t < \tau_1 \\ 0\,, & t > \tau_1 \end{cases}, \tag{2.14}$$

$$r_2(t) = \begin{cases} r_{2,0}\,, & 0 \leq t < \tau_2 \\ 0\,, & t > \tau_2 \end{cases}, \quad d_2(t) = \begin{cases} d_{2,0}\,, & 0 \leq t < \tau_2 \\ 0\,, & t > \tau_2 \end{cases}. \tag{2.15}$$

The disease durations τ_1 and τ_2 and the parameters $r_{1,0}$, $r_{2,0}$, $d_{1,0}$ and $d_{2,0}$ are positive constants. By substituting these functions into Eqs (2.4)–(2.7), we obtain the following expressions:

$$\frac{dR_1}{dt} = r_{1,0} \int_{t-\tau_1}^t J_1(\eta)d\eta, \quad \frac{dD_1}{dt} = d_{1,0} \int_{t-\tau_1}^t J_1(\eta)d\eta, \tag{2.16}$$

$$\frac{dR_2}{dt} = r_{2,0}\int_{t-\tau_2}^{t} J_2(\eta)d\eta, \quad \frac{dD_2}{dt} = d_{2,0}\int_{t-\tau_2}^{t} J_2(\eta)d\eta. \tag{2.17}$$

By integrating Eq (2.16) from $t-\tau_1$ to t and Eq (2.17) from $t-\tau_2$ to t, we obtain the following:

$$R_1(t) - R_1(t-\tau_1) = r_{1,0}\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds, \tag{2.18}$$

$$D_1(t) - D_1(t-\tau_1) = d_{1,0}\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds, \tag{2.19}$$

$$R_2(t) - R_2(t-\tau_2) = r_{2,0}\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta\Big)ds, \tag{2.20}$$

$$D_2(t) - D_2(t-\tau_2) = d_{2,0}\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta\Big)ds. \tag{2.21}$$

Therefore,

$$I_1(t) = \int_{t-\tau_1}^{t} J_1(\eta)d\eta - (R_1(t) - R_1(t-\tau_1)) - (D_1(t) - D_1(t-\tau_1)), \tag{2.22}$$

$$I_2(t) = \int_{t-\tau_2}^{t} J_2(\eta)d\eta - (R_2(t) - R_2(t-\tau_1)) - (D_2(t) - D_2(t-\tau_1)). \tag{2.23}$$

We note that $(R_1(t)-R_1(t-\tau_1))$ and $(D_1(t)-D_1(t-\tau_1))$ correspond to the number of individuals who have recovered and died from the first viral strain during the time interval $(t-\tau_1, t)$, respectively. Similarly, $(R_2(t)-R_2(t-\tau_2))$ and $(D_2(t)-D_2(t-\tau_2))$ represent the number of individuals who have recovered and died from the second viral strain during the time interval $(t-\tau_2, t)$, respectively. From Eqs (2.18)–(2.21), we have the following:

$$I_1(t) = \int_{t-\tau_1}^{t} J_1(\eta)d\eta - (r_{1,0}+d_{1,0})\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds, \tag{2.24}$$

$$I_2(t) = \int_{t-\tau_2}^{t} J_2(\eta)d\eta - (r_{2,0}+d_{2,0})\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta\Big)ds. \tag{2.25}$$

Now, from (2.22)–(2.25),

$$\begin{aligned}
\frac{dI_1}{dt} &= \beta_1\frac{S}{N}I_1 - (r_{1,0}+d_{1,0})\int_{t-\tau_1}^{t} J_1(\eta)d\eta \\
&= \beta_1\frac{S}{N}I_1 - (r_{1,0}+d_{1,0})\Big[I_1(t) + (r_{1,0}+d_{1,0})\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds\Big], \\
\frac{dI_2}{dt} &= \beta_2\frac{S}{N}I_2 - (r_{2,0}+d_{2,0})\int_{t-\tau_2}^{t} J_2(\eta)d\eta \\
&= \beta_2\frac{S}{N}I_2 - (r_{2,0}+d_{2,0})\Big[I_2(t) + (r_{2,0}+d_{2,0})\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta\Big)ds\Big].
\end{aligned}$$

Under the assumption that $(r_{1,0}+d_{1,0})$ and $(r_{2,0}+d_{2,0})$ are sufficiently small, we can disregard the terms involving $(r_{1,0}+d_{1,0})^2$ and $(r_{2,0}+d_{2,0})^2$. Consequently, we obtain the following:

$$\frac{dI_1}{dt} \simeq \beta_1\frac{S}{N}I_1 - (r_{1,0}+d_{1,0})I_1,$$

$$\frac{dI_2}{dt} \simeq \beta_2 \frac{S}{N} I_2 - (r_{2,0} + d_{2,0}) I_2.$$

In this case, the system represented by Eqs (2.1)–(2.7) is simplified to the following conventional ODE model:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta_1 \frac{S}{N} I_1 - \beta_2 \frac{S}{N} I_2, \\
\frac{dI_1}{dt} &= \beta_1 \frac{S}{N} I_1 - (r_{1,0} + d_{1,0}) I_1, \\
\frac{dI_2}{dt} &= \beta_2 \frac{S}{N} I_2 - (r_{2,0} + d_{2,0}) I_2, \\
\frac{dR_1}{dt} &= r_{1,0} I_1, \quad \frac{dR_2}{dt} = r_{2,0} I_2, \\
\frac{dD_1}{dt} &= d_{1,0} I_1, \quad \frac{dD_2}{dt} = d_{2,0} I_2.
\end{aligned}$$

Therefore, assuming that the recovery and death rates have uniform distributions as given in Eqs (2.14) and (2.15), and that they are sufficiently small, we can reduce models (2.1)–(2.7) to the ODE model. However, it is important to note that, in general, these assumptions may not hold, and we can consider different distributions for recovery and death rates.

3. Delay model

3.1. Derivation of the delay model

Let us recall that the duration of the first disease is denoted by τ_1, and the duration of the second disease by τ_2. Additionally, suppose that individuals $J_1(t-\tau_1)$ and $J_2(t-\tau_2)$ infected at time $t-\tau_1$ and $t-\tau_2$, respectively, either recover or die at time t with certain probabilities. This assumption corresponds to the following choices for the functions $r_1(t-\eta)$, $r_2(t-\eta)$, $d_1(t-\eta)$, and $d_2(t-\eta)$:

$$r_1(t-\tau_1) = r_{1,0}\delta(t-\eta-\tau_1), \;\; r_2(t-\tau_2) = r_{2,0}\delta(t-\eta-\tau_2),$$

$$d_1(t-\tau_1) = d_{1,0}\delta(t-\eta-\tau_1), \;\; d_2(t-\tau_2) = d_{2,0}\delta(t-\eta-\tau_2),$$

where $r_{1,0}$, $r_{2,0}$, $d_{1,0}$, $d_{2,0}$ are constants, $r_{1,0} + d_{1,0} = 1$ and $r_{2,0} + d_{2,0} = 1$, and δ is the Dirac delta-function. Then,

$$\begin{aligned}
\frac{dR_1}{dt} &= \int_0^t r_1(t-\eta) J_1(\eta) d\eta = r_{1,0} \int_0^t \delta(t-\eta-\tau_1) J_1(\eta) d\eta = r_{1,0} J_1(t-\tau_1), \\
\frac{dR_2}{dt} &= \int_0^t r_2(t-\eta) J_2(\eta) d\eta = r_{2,0} \int_0^t \delta(t-\eta-\tau_2) J_2(\eta) d\eta = r_{2,0} J_2(t-\tau_2), \\
\frac{dD_1}{dt} &= \int_0^t d_1(t-\eta) J_1(\eta) d\eta = d_{1,0} \int_0^t \delta(t-\eta-\tau_1) J_1(\eta) d\eta = d_{1,0} J_1(t-\tau_1), \\
\frac{dD_2}{dt} &= \int_0^t d_2(t-\eta) J_2(\eta) d\eta = d_{2,0} \int_0^t \delta(t-\eta-\tau_2) J_2(\eta) d\eta = d_{2,0} J_2(t-\tau_2).
\end{aligned}$$

Therefore, the systems (2.1)–(2.7) are reduced to the following delay model:

$$\frac{dS}{dt} = -J_1(t) - J_2(t), \tag{3.1}$$

$$\frac{dI_i}{dt} = J_i(t) - J_i(t-\tau_i), \qquad i = 1, 2, \tag{3.2}$$

$$\frac{dR_i}{dt} = r_{i,0}J_i(t-\tau_i), \qquad i = 1, 2, \tag{3.3}$$

$$\frac{dD_i}{dt} = d_{i,0}J_i(t-\tau_i), \qquad i = 1, 2 \tag{3.4}$$

with $J_1(t) = J_2(t) = 0$ for $t < 0$.

3.2. Basic reproduction number

Let us consider Eq (3.2) for the infected compartments:

$$\frac{dI_i}{dt} = \frac{\beta_i S(t) I_i(t)}{N} - \frac{\beta_i S(t-\tau_i) I_i(t-\tau_i)}{N}, \quad i = 1, 2. \tag{3.5}$$

To determine the basic reproduction numbers $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$. We set $S(t) = S(t-\tau_1) = S(t-\tau_2) = S_0$ since the number of susceptible individuals is close to its initial condition in the beginning of the epidemic. Then, according to (3.5), we have

$$\frac{dI_i}{dt} = \frac{\beta_i S_0}{N}(I_i(t) - I_i(t-\tau_i)), \quad i = 1, 2.$$

Substituting $I_i(t) = I_{0i}e^{\lambda_i t}$, $i = 1, 2$, we have for $i = 1, 2$

$$\lambda_i = \frac{\beta_i S_0}{N}(1 - e^{-\lambda_i \tau_i}). \tag{3.6}$$

Let

$$G_i(\lambda_i) = \frac{\beta_i S_0}{N}(1 - e^{-\lambda_i \tau_i}).$$

It is evident that Eq (3.6) have solutions (i.e., $\lambda_1 = \lambda_2 = 0$) and a non-zero solution, the sign of which is determined by $G_i'(0)$. Denote

$$\mathcal{R}_{0i} = G_i^{'}(0) = \frac{\beta_i S_0 \tau_i}{N}.$$

Let $\mathcal{R}_{01} > 1$ and $\mathcal{R}_{02} > 1$. This means that $G_1^{'}(0) > 1$ and $G_2^{'}(0) > 1$. We see that $G_1(\lambda_1)$ and $G_2(\lambda_2)$ are increasing functions of λ_1 and λ_2, respectively, with $G_1(\lambda_1) \to \frac{\beta_1 S_0}{N}$ as $\lambda_1 \to \infty$ and $G_2(\lambda_2) \to \frac{\beta_2 S_0}{N}$ as $\lambda_2 \to \infty$. This implies that Eq (3.6) has positive solutions, (i.e., there exist $\lambda_1^* > 0$ and $\lambda_2^* > 0$ such that $G_i(\lambda_i^*) = \lambda_i^*$, $i = 1, 2$).

If $G_i^{'}(0) < 1$, then

$$G_i{}'(\lambda_i) = \frac{\beta_i S_0 \tau_i}{N} e^{-\lambda_i \tau_i} < 1,$$

for all $\lambda_1 \geq 0$ and $\lambda_2 \geq 0$. Hence, there is no positive solution to the equation $G_i(\lambda_i) = \lambda_i$, $i = 1, 2$. Consequently, the basic reproduction numbers can be expressed as follows:

$$\mathcal{R}_{0i} = \frac{\beta_i S_0 \tau_i}{N}, \quad i = 1, 2.$$

3.3. Final size of the epidemic

Next, we aim to estimate the final size of the susceptible compartment $S_\infty = \lim_{t\to\infty} S(t)$. Integrating (3.2), we obtain the following:

$$I_1(t) = \int_{t-\tau_1}^{t} J_1(\eta)d\eta,$$
$$I_2(t) = \int_{t-\tau_2}^{t} J_2(\eta)d\eta.$$

From (3.1), we obtain the following:

$$\frac{dS(t)}{dt} = -\beta_1\frac{S(t)}{N}\int_{t-\tau_1}^{t} J_1(\eta)d\eta - \beta_2\frac{S(t)}{N}\int_{t-\tau_2}^{t} J_2(\eta)d\eta.$$

Then, integrating from 0 to ∞, we obtain the following:

$$\ln\left(\frac{S_0}{S_\infty}\right) = \frac{\beta_1}{N}\int_0^\infty\left(\int_{t-\tau_1}^{t} J_1(\eta)d\eta\right)dt + \frac{\beta_2}{N}\int_0^\infty\left(\int_{t-\tau_2}^{t} J_2(\eta)d\eta\right)dt.$$

Changing the order of integration, we obtain the following:

$$\begin{aligned}\ln\left(\frac{S_0}{S_\infty}\right) &= \frac{\beta_1}{N}\left[\int_{-\tau_1}^{0}\left(\int_0^{\eta+\tau_1} J_1(\eta)dt\right)d\eta + \int_0^\infty\left(\int_\eta^{\eta+\tau_1} J_1(\eta)dt\right)d\eta\right]\\ &+ \frac{\beta_2}{N}\left[\int_{-\tau_2}^{0}\left(\int_0^{\eta+\tau_2} J_2(\eta)dt\right)d\eta + \int_0^\infty\left(\int_\eta^{\eta+\tau_2} J_2(\eta)dt\right)d\eta\right].\\ &= \frac{\beta_1}{N}\left[\int_{-\tau_1}^{0}(\eta+\tau_1)J_1(\eta)d\eta + \int_0^\infty \tau_1 J_1(\eta)d\eta\right] + \frac{\beta_2}{N}\left[\int_{-\tau_2}^{0}(\eta+\tau_2)J_2(\eta)d\eta\right.\\ &+ \left.\int_0^\infty \tau_2 J_2(\eta)d\eta\right].\end{aligned}$$

Now, integrating (3.1) from 0 to ∞, we obtain the following:

$$\int_0^\infty (J_1(\eta) + J_2(\eta))d\eta = S_0 - S_\infty.$$

Since $J_1(t) = 0$ for all $t \in [-\tau_1, 0]$ and $J_2(t) = 0$ for all $t \in [-\tau_2, 0]$, then

$$\begin{aligned}\ln\left(\frac{S_0}{S_\infty}\right) &= \frac{\beta_1}{N}\int_0^\infty \tau_1 J_1(\eta)d\eta + \frac{\beta_2}{N}\int_0^\infty \tau_2 J_2(\eta)d\eta\\ &\le \max\left(\frac{\beta_1\tau_1 S_0}{N}, \frac{\beta_2\tau_2 S_0}{N}\right)\frac{1}{S_0}\int_0^\infty (J_1(\eta) + J_2(\eta))d\eta.\end{aligned}$$

Thus, we obtain an upper bound for the final size:

$$\ln\left(\frac{S_0}{S_\infty}\right) \le \max(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right).$$

In the same way, we obtain a lower bound for the final size:

$$\ln\left(\frac{S_0}{S_\infty}\right) \geq \min(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right)$$

Hence, we obtain a two-sided estimate for S_∞:

$$\min(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right) \leq \ln\left(\frac{S_0}{S_\infty}\right) \leq \max(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right). \tag{3.7}$$

Let us recall that in the case of a single strain, there is an equation for S_∞ instead of the inequalities.

4. Numerical simulation

For the delay models (3.1)–(3.4), we compare the maximum of infected by each virus. We chose the following values for the parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $\beta_2 = 0.2$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

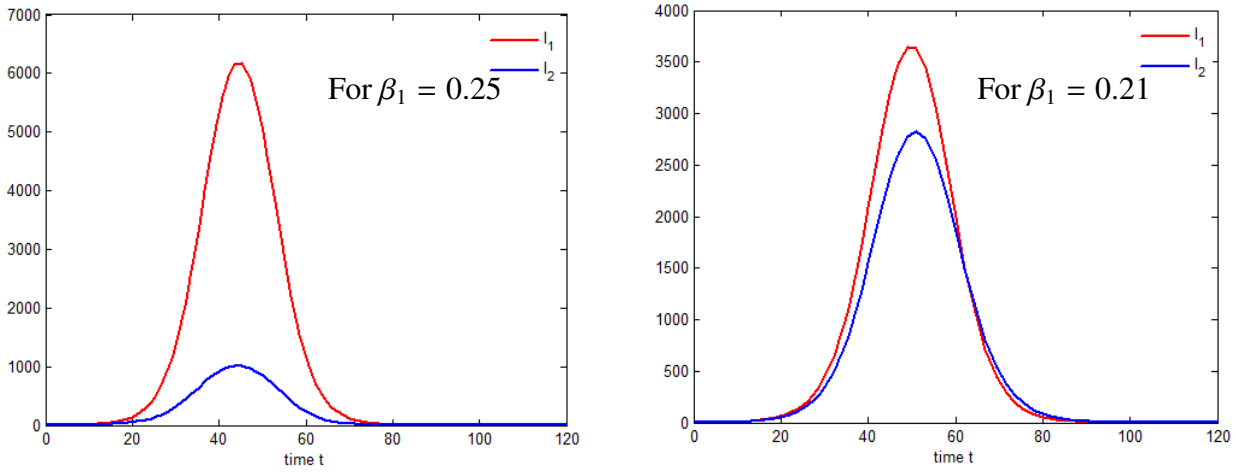

Figure 1. The number of infected individuals by each strain for $\mathcal{R}_{01} > \mathcal{R}_{02}$ (left), $\mathcal{R}_{01} < \mathcal{R}_{02}$ (right). The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $\beta_2 = 0.2$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

Figure 1 shows the number of infected individuals in time for each strain. In the first case, when $\mathcal{R}_{01} > \mathcal{R}_{02}$, the maximum of infected individuals is higher for the first strain. In the other case, when $\mathcal{R}_{01} < \mathcal{R}_{02}$, we obtain the same result. Therefore, the domination of the strains is not uniquely determined by the basic reproduction numbers but rather by the individual parameters.

Next, we choose the following values for the parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$, and vary the values of β_1 and β_2 in the interval $[0.1, 0.5]$. Figures 2–4 display the dependence of the maximal number of infected I_{1m} and I_{2m}, the time to the maximal number of infected for the first and the second virus and the total number of infected of the two virus of the models (3.1)–(3.4) on β_1, β_2.

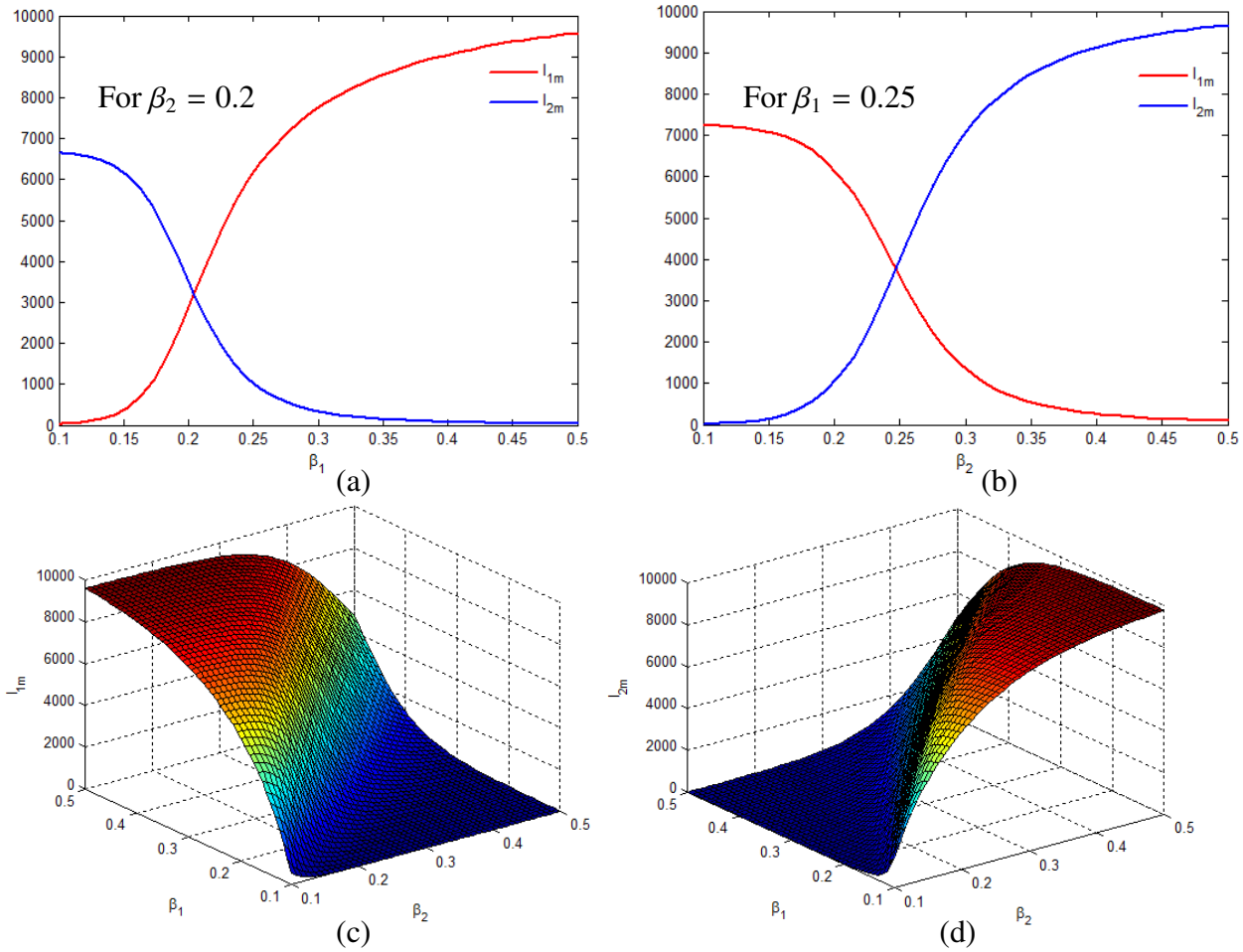

Figure 2. Dependence of the maximal number of infected individuals by each strain on : (a) β_1 for $\beta_2 = 0.2$ (b) β_2 for $\beta_1 = 0.25$. Dependence of the maximal number of infected individuals on β_1, β_2 : (c) the first strain (d) the second strain. The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

Figure 2(a) shows when the transmission rate, β_2, is fixed and the evolution of the epidemic peak is examined in relation to variations in β_1. As β_1 increases, the epidemic peak corresponding to the first virus also increases; for the second virus, the epidemic peak gradually decreases, and the number of individuals infected by the second virus decreases.

In other words, when comparing the impact of the first and second virus by modifying the transmission rate of the first virus, while keeping the transmission rate of the second epidemic constant, we observe that the first virus becomes more prevalent and reaches a higher epidemic peak as the transmission rate increases. On the other hand, the second virus is gradually eliminated, and its epidemic peak decreases as the transmission rate of the first virus increases.

It is interesting to note that in Figure 2(b), when β_1 is fixed and β_2 varies, we observe the opposite phenomenon. As β_2 increases, the epidemic peak corresponding to the second virus also increases; while for the first virus, the epidemic peak gradually decreases. The last two graphs (c) and (d) present the variation of the epidemic peak for both viruses in relation to β_1 and β_2. These

observations underscore the importance of the transmission rate in the spread of epidemics and how different values of β_1 and β_2 can influence the evolution of different epidemics.

The relationship between the timing of the epidemic peak and β_1 and β_2 can be complex. For $\beta_1 > \beta_2$, if the two viruses infect the same number of individuals, then the first strain reaches the maximum faster than the other. Additionally, if the difference between β_1 and β_2 is large, then we can expect that the difference between t_{1m} and t_{2m} is also large, though this is not the case here. If $\beta_1 > \beta_2$, the first strain infects more individuals to reach the maximum though it is faster, and the second strain infects less individuals to reach the maximum, though it is slower. Therefore, if β_1 is very large compared to β_2, this does not imply that t_{1m} will be very large compared to t_{2m}. As a result, the difference between the transmission rates does not strongly influence the peak time. Thus, t_{1m} is either equal or very close to t_{2m} (see Figure 3).

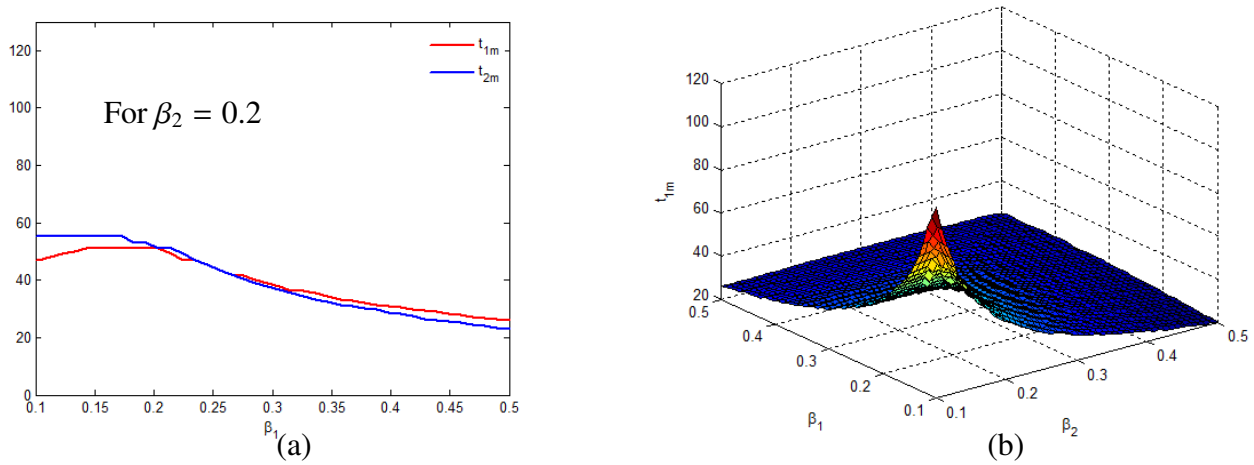

Figure 3. (a) Dependence of the time to reach the maximal number of infected by each strain on β_1 for $\beta_2 = 0.2$. (b) Dependence of the time to reach the maximal number of infected on β_1, β_2 by the first strain. The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

In terms of the dependence on β_1 and β_2 for the number of individuals affected by the first and second viruses, we can observe a similar phenomenon as seen in the epidemic peak (see Figure 4). As β_1 increases, the number of individuals affected by the first virus tends to increase. This indicates that a higher transmission rate for the first virus leads to a larger population being infected by it compared to a larger population affected by the first virus. On the other hand, as β_1 increases, the number of individuals affected by the second virus gradually decreases. This suggests that the second virus experiences a decline in its transmission and a lower number of individuals become infected by it.

Similarly, when β_2 is varied while keeping β_1 constant, higher values of β_2 can lead to an increase in the number of individuals affected by the second virus; alternatively, the number of individuals affected by the first virus may decrease.

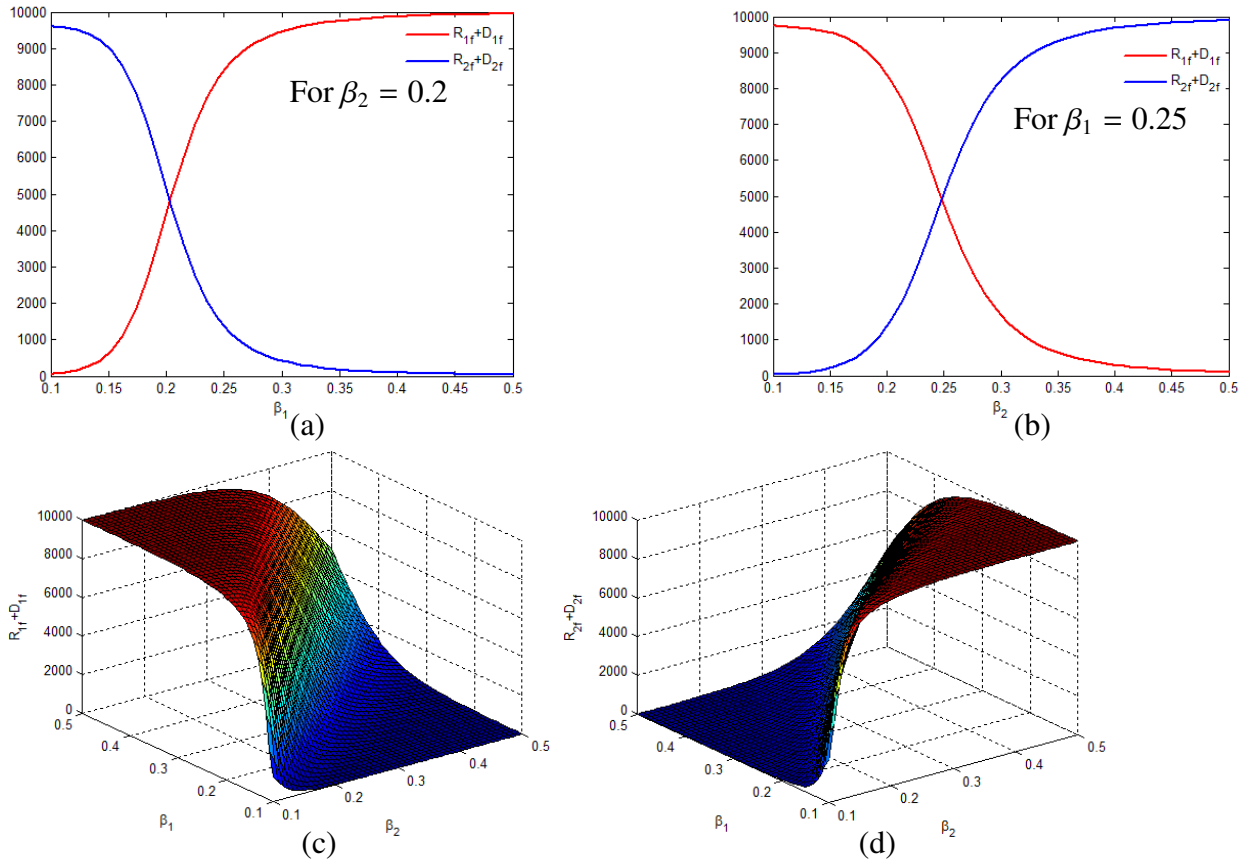

Figure 4. Dependence of the total number of infected by each strain on : (a) β_1 for $\beta_2 = 0.2$ (b) β_2 for $\beta_1 = 0.25$. Dependence of the total number of infected on β_1, β_2 by : (c) the first strain (d) the second strain. The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

Finally, we estimate the inequality terms of (3.7). Let

$$S_l = \min(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right), \quad S_m = \ln\left(\frac{S_0}{S_\infty}\right), \quad S_u = \max(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right).$$

In Figure 5, we can see that the graph of S_m is very close to the graph of S_u. This result suggests the following conjecture:

$$\ln\left(\frac{S_0}{S_\infty}\right) \simeq \max(\mathcal{R}_{01}, \mathcal{R}_{02})\left(1 - \frac{S_\infty}{S_0}\right).$$

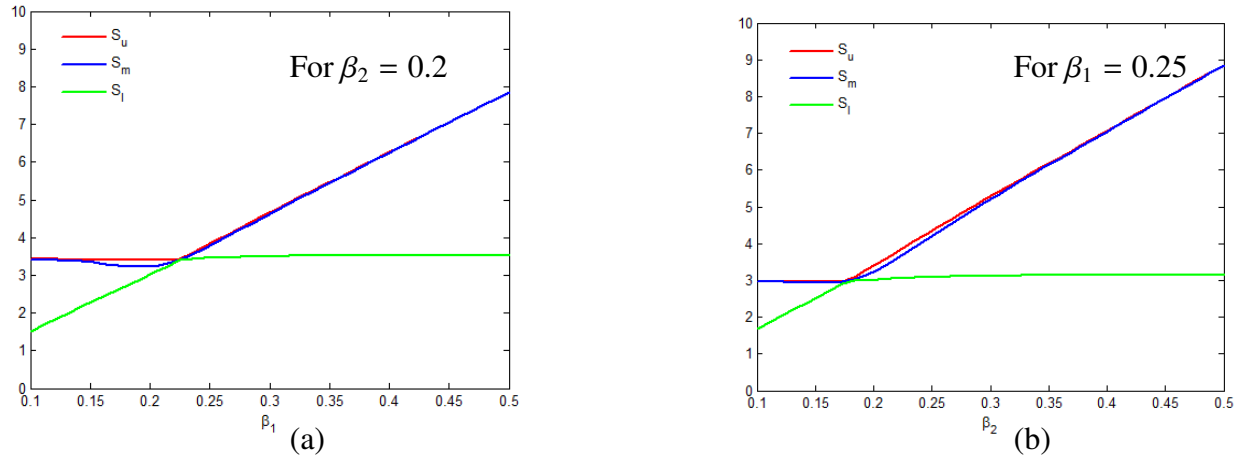

Figure 5. Simulation of inequality terms (3.7) as a function of : (a) β_1 for $\beta_2 = 0.2$ (b) β_2 for $\beta_1 = 0.25$. The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

5. Model without cross-immunity

In this section, we study a similar model assuming that individuals who have recovered from the first viral strain can be infected by the second viral strain, and individuals who have recovered from the second viral strain can be infected by the first viral strain.

5.1. Model formulation

Let $J_{12}(t) = (\epsilon_1\beta_2 I_2 R_1)/N$ represent the number of individuals who have previously recovered from the first viral strain and are newly infected with the second viral strain. Similarly, $J_{21}(t) = (\epsilon_2\beta_1 I_1 R_2)/N$ represents the number of individuals who have previously recovered from the second viral strain and are newly infected with the first viral strain, where ϵ_1 and ϵ_2 are constants that indicate the level of cross-immunity. For $\epsilon_1 = \epsilon_2 = 0$, there is complete cross-immunity. For $\epsilon_1 = \epsilon_2 = 1$, there is the absence of cross-immunity. In this modified system, we introduce additional variables to account for individuals who have recovered from one strain and are subsequently infected with the other strain. Let us define the following variables:

- $I_{12}(t)$ - infected with the first viral strain initially and later infected with the second viral strain.
- $I_{21}(t)$ - infected with the second viral strain initially and later infected with the first viral strain.
- $R_{12}(t)$ - recovered from the first viral strain initially and later infected with the second viral strain, becoming immune to both strains.
- $R_{21}(t)$ - recovered from the second viral strain initially and later infected with the first viral strain, becoming immune to both strains.
- $D_{12}(t)$ - recovered from the first viral strain initially and later die due to the second viral strain.
- $D_{21}(t)$ - recovered from the second viral strain initially and later die due to the first viral strain.

The individuals that are recovered and infected at time $t-\eta$ with the first viral strain and the second viral strain are represented by $J_{21}(t-\eta)$ and $J_{12}(t-\eta)$, respectively. With these additional variables,

the system can be expressed as follows, using the same notations as in the second section :

$$\frac{dS}{dt} = -J_1(t) - J_2(t), \tag{5.1}$$

$$\frac{dI_1}{dt} = J_1(t) - \int_0^t r_1(t-\eta)J_1(\eta)d\eta - \int_0^t d_1(t-\eta)J_1(\eta)d\eta, \tag{5.2}$$

$$\frac{dI_2}{dt} = J_2(t) - \int_0^t r_2(t-\eta)J_2(\eta)d\eta - \int_0^t d_2(t-\eta)J_2(\eta)d\eta, \tag{5.3}$$

$$\frac{dR_1}{dt} = \int_0^t r_1(t-\eta)J_1(\eta)d\eta - J_{12}(t), \tag{5.4}$$

$$\frac{dR_2}{dt} = \int_0^t r_2(t-\eta)J_2(\eta)d\eta - J_{21}(t), \tag{5.5}$$

$$\frac{dI_{12}}{dt} = J_{12}(t) - \int_0^t r_2(t-\eta)J_{12}(\eta)d\eta - \int_0^t d_2(t-\eta)J_{12}(\eta)d\eta, \tag{5.6}$$

$$\frac{dI_{21}}{dt} = J_{21}(t) - \int_0^t r_1(t-\eta)J_{21}(\eta)d\eta - \int_0^t d_1(t-\eta)J_{21}(\eta)d\eta, \tag{5.7}$$

$$\frac{dR_{12}}{dt} = \int_0^t r_2(t-\eta)J_{12}(\eta)d\eta, \tag{5.8}$$

$$\frac{dR_{21}}{dt} = \int_0^t r_1(t-\eta)J_{21}(\eta)d\eta, \tag{5.9}$$

$$\frac{dD_1}{dt} = \int_0^t d_1(t-\eta)J_1(\eta)d\eta, \tag{5.10}$$

$$\frac{dD_2}{dt} = \int_0^t d_2(t-\eta)J_2(\eta)d\eta, \tag{5.11}$$

$$\frac{dD_{12}}{dt} = \int_0^t d_2(t-\eta)J_{12}(\eta)d\eta, \tag{5.12}$$

$$\frac{dD_{21}}{dt} = \int_0^t d_1(t-\eta)J_{21}(\eta)d\eta. \tag{5.13}$$

The system is subject to the following initial condition:

$$\begin{aligned} &S(0) = S_0 > 0, \;\; I_1(0) = I_{1,0} > 0, \;\; I_2(0) = I_{2,0} > 0, \;\; R_1(0) = R_2(0) = I_{12}(0) = I_{21}(0) = R_{12}(0) \\ &= R_{21}(0) = D_1(0) = D_2(0) = D_{12}(0) = D_{21}(0) = 0, \end{aligned} \tag{5.14}$$

where $I_{1,0}$ and $I_{2,0}$ are sufficiently small as compared to N. We assume that the total population is constant :

$$\begin{aligned} N =& S(t) + I_1(t) + I_2(t) + R_1(t) + R_2(t) + I_{12}(t) + I_{21}(t) + R_{12}(t) + R_{21}(t) + D_1(t) + D_2(t) \\ &+ D_{12}(t) + D_{21}(t). \end{aligned} \tag{5.15}$$

5.2. Positiveness of solutions

We prove the positivity of the solution to this problem on a time interval $[0, T)$ where $T \in (0, +\infty)$. We make the assumption that the recovery and death rates satisfy the inequalities (2.10) and (2.11).

Lemma 2. *If conditions (2.10) and (2.11) are satisfied, then any solution of systems (5.1)–(5.13) with the initial condition (5.14) satisfies the inequality* $0 \le X \le N$, *where*

$$X \in \{S(t), I_1(t), I_2(t), R_1(t), R_2(t), I_{12}(t), I_{21}(t), R_{12}(t), R_{21}(t), D_1(t), D_2(t), D_{12}(t), D_{21}(t)\}.$$

Proof. From Eq (5.1), we can observe that if there exists a $t_* > 0$ such that $S(t_*) = 0$, then $\frac{dS}{dt}\Big|_{t=t_*} = 0$. This implies that $S(t) \ge 0$ for $t > 0$. Furthermore, based on Eqs (5.4), (5.5), and (5.8)–(5.13), we can conclude that $R_1(t)$, $R_2(t)$, $R_{12}(t)$, $R_{21}(t)$, $D_1(t)$, $D_2(t)$, $D_{12}(t)$, and $D_{21}(t)$ also remain positive for all values of t. At $t = 0$, we have $J_1(0) = \beta_1 S_0 I_{1,0}/N > 0$, $J_2(0) = \beta_2 S_0 I_{2,0}/N > 0$ and $J_{12}(0) = J_{21}(0) = 0$. Suppose $t_0 > 0$ be such that $J_1(t)$, $J_2(t)$, $J_{12}(t)$, $J_{21}(t)$ remain non-negative for $0 \le t < t_0$. Next, we have the following:

$$\begin{aligned} I_1(t_0) &= \int_0^{t_0} J_1(t)dt - R_1(t_0) - D_1(t_0) - \int_0^{t_0} J_{12}(t)dt, \\ I_2(t_0) &= \int_0^{t_0} J_2(t)dt - R_2(t_0) - D_2(t_0) - \int_0^{t_0} J_{21}(t)dt. \end{aligned}$$

By integrating (5.4), (5.5), (5.10) and (5.11) with respect to t from 0 to t_0, and using the initial conditions given in (5.14), obtain the following:

$$\begin{aligned} R_1(t_0) + D_1(t_0) &= \int_0^{t_0} \Big(\int_0^t (r_1(t-\eta) + d_1(t-\eta))J_1(\eta)d\eta \Big)dt - \int_0^{t_0} J_{12}(t)dt, \\ R_2(t_0) + D_2(t_0) &= \int_0^{t_0} \Big(\int_0^t (r_2(t-\eta) + d_2(t-\eta))J_2(\eta)d\eta \Big)dt - \int_0^{t_0} J_{21}(t)dt. \end{aligned}$$

Changing the order of integration and using the inequalities (2.10) and (2.11), we observe the following:

$$\begin{aligned} R_1(t_0) + D_1(t_0) &= \int_0^{t_0} \Big(\int_\eta^{t_0} (r_1(t-\eta) + d_1(t-\eta))dt \Big)J_1(\eta)d\eta - \int_0^{t_0} J_{12}(t)dt \\ &\le \int_0^{t_0} J_1(\eta)d\eta - \int_0^{t_0} J_{12}(t)dt, \qquad (5.16) \\ R_2(t_0) + D_2(t_0) &= \int_0^{t_0} \Big(\int_\eta^{t_0} (r_2(t-\eta) + d_2(t-\eta))dt \Big)J_2(\eta)d\eta - \int_0^{t_0} J_{21}(t)dt \\ &\le \int_0^{t_0} J_2(\eta)d\eta - \int_0^{t_0} J_{21}(t)dt. \qquad (5.17) \end{aligned}$$

It follows from the inequalities (5.16) and (5.17) that $I_1(t_0)$ and $I_2(t_0)$ are both non-negative. Consequently, $J_1(t_0)$, $J_2(t_0)$, $J_{12}(t_0)$ and $J_{21}(t_0)$ are also non-negative. Thus, we have shown that $I_1(t)$, $I_2(t)$, $J_1(t)$, $J_2(t)$, $J_{12}(t)$ and $J_{21}(t)$ remain non-negative for all $t \ge 0$. On the other hand, we have the following:

$$\begin{aligned} I_{12}(t_0) &= \int_0^{t_0} J_{12}(t)dt - R_{12}(t_0) - D_{12}(t_0), \\ I_{21}(t_0) &= \int_0^{t_0} J_{21}(t)dt - R_{21}(t_0) - D_{21}(t_0). \end{aligned}$$

By integrating (5.8), (5.9), (5.12) and (5.13) with respect to t from 0 to t_0 and using the initial conditions (5.14), we obtain the following:

$$R_{12}(t_0) + D_{12}(t_0) = \int_0^{t_0} \Big(\int_0^t (r_2(t-\eta) + d_2(t-\eta)) J_{12}(\eta) d\eta \Big) dt,$$
$$R_{21}(t_0) + D_{21}(t_0) = \int_0^{t_0} \Big(\int_0^t (r_1(t-\eta) + d_1(t-\eta)) J_{21}(\eta) d\eta \Big) dt.$$

Changing the order of integration and using inequalities (2.10) and (2.11), we observe the following:

$$R_{12}(t_0) + D_{12}(t_0) = \int_0^{t_0} \Big(\int_\eta^{t_0} (r_2(t-\eta) + d_2(t-\eta)) dt \Big) J_{12}(\eta) d\eta \leq \int_0^{t_0} J_{12}(\eta) d\eta, \tag{5.18}$$
$$R_{21}(t_0) + D_{21}(t_0) = \int_0^{t_0} \Big(\int_\eta^{t_0} (r_1(t-\eta) + d_1(t-\eta)) dt \Big) J_{21}(\eta) d\eta \leq \int_0^{t_0} J_{21}(\eta) d\eta. \tag{5.19}$$

It follows from the inequalities (5.18) and (5.19) that $I_{12}(t_0)$ and $I_{21}(t_0)$ are both non-negative. Consequently, $I_{12}(t)$ and $I_{21}(t)$ remain non-negative for all $t \geq 0$. Furthermore, we have (5.15). Thus, any solution of system (5.1)–(5.13) lies between 0 and N, thereby ensuring the non-negativity of the solution. □

5.3. Reduction to the ODE model

In this section, we demonstrate that models (5.1)–(5.13) can be reduced to a conventional ODE model under certain assumptions. Let us assume that the recovery and death rates are given by the following functions :

$$r_1(t) = \begin{cases} r_{1,0}\,, & 0 \leq t < \tau_1 \\ 0\,, & t > \tau_1 \end{cases}, \quad d_1(t) = \begin{cases} d_{1,0}\,, & 0 \leq t < \tau_1 \\ 0\,, & t > \tau_1 \end{cases}, \tag{5.20}$$

$$r_2(t) = \begin{cases} r_{2,0}\,, & 0 \leq t < \tau_2 \\ 0\,, & t > \tau_2 \end{cases}, \quad d_2(t) = \begin{cases} d_{2,0}\,, & 0 \leq t < \tau_2 \\ 0\,, & t > \tau_2 \end{cases}, \tag{5.21}$$

The disease durations $\tau_1, \tau_2 > 0$ and the parameters $r_{1,0}$, $r_{2,0}$, $d_{1,0}$ and $d_{2,0}$ are positive constants. By substituting these functions in (5.4), (5.5) and (5.8)–(5.13), we obtain the following:

$$\frac{dR_1}{dt} = r_{1,0} \int_{t-\tau_1}^t J_1(\eta) d\eta - J_{12}(t), \quad \frac{dD_1}{dt} = d_{1,0} \int_{t-\tau_1}^t J_1(\eta) d\eta, \tag{5.22}$$
$$\frac{dR_2}{dt} = r_{2,0} \int_{t-\tau_2}^t J_2(\eta) d\eta - J_{21}(t), \quad \frac{dD_2}{dt} = d_{2,0} \int_{t-\tau_2}^t J_2(\eta) d\eta, \tag{5.23}$$
$$\frac{dR_{12}}{dt} = r_{2,0} \int_{t-\tau_2}^t J_{12}(\eta) d\eta, \quad \frac{dD_{12}}{dt} = d_{2,0} \int_{t-\tau_2}^t J_{12}(\eta) d\eta, \tag{5.24}$$
$$\frac{dR_{21}}{dt} = r_{1,0} \int_{t-\tau_1}^t J_{21}(\eta) d\eta, \quad \frac{dD_{21}}{dt} = d_{1,0} \int_{t-\tau_1}^t J_{21}(\eta) d\eta. \tag{5.25}$$

By integrating Eqs (5.22) and (5.25) from $t-\tau_1$ to t, and Eqs (5.23) and (5.24) from $t-\tau_2$ to t, we obtain the following:

$$R_1(t) - R_1(t-\tau_1) = r_{1,0}\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds - \int_{t-\tau_1}^{t} J_{12}(\eta)d\eta, \tag{5.26}$$

$$D_1(t) - D_1(t-\tau_1) = d_{1,0}\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds, \tag{5.27}$$

$$R_2(t) - R_2(t-\tau_2) = r_{2,0}\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta\Big)ds - \int_{t-\tau_2}^{t} J_{21}(\eta)d\eta, \tag{5.28}$$

$$D_2(t) - D_2(t-\tau_2) = d_{2,0}\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta\Big)ds, \tag{5.29}$$

$$R_{21}(t) - R_{21}(t-\tau_1) = r_{1,0}\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_{21}(\eta)d\eta\Big)ds, \tag{5.30}$$

$$D_{21}(t) - D_{21}(t-\tau_1) = d_{1,0}\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_{21}(\eta)d\eta\Big)ds, \tag{5.31}$$

$$R_{12}(t) - R_{12}(t-\tau_2) = r_{2,0}\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_{12}(\eta)d\eta\Big)ds, \tag{5.32}$$

$$D_{12}(t) - D_{12}(t-\tau_2) = d_{2,0}\int_{t-\tau_2}^{t}\Big(\int_{s-\tau_2}^{s} J_{12}(\eta)d\eta\Big)ds. \tag{5.33}$$

Thus,

$$I_1(t) = \int_{t-\tau_1}^{t} J_1(\eta)d\eta - (R_1(t) - R_1(t-\tau_1)) + \int_{t-\tau_1}^{t} J_{12}(\eta)d\eta - (D_1(t) - D_1(t-\tau_1)), \tag{5.34}$$

$$I_2(t) = \int_{t-\tau_2}^{t} J_2(\eta)d\eta - (R_2(t) - R_2(t-\tau_2)) + \int_{t-\tau_2}^{t} J_{21}(\eta)d\eta - (D_2(t) - D_2(t-\tau_2)), \tag{5.35}$$

$$I_{21}(t) = \int_{t-\tau_1}^{t} J_{21}(\eta)d\eta - (R_{21}(t) - R_{21}(t-\tau_1)) - (D_{21}(t) - D_{21}(t-\tau_1)), \tag{5.36}$$

$$I_{12}(t) = \int_{t-\tau_2}^{t} J_{12}(\eta)d\eta - (R_{12}(t) - R_{12}(t-\tau_2)) - (D_{12}(t) - D_{12}(t-\tau_2)), \tag{5.37}$$

where $(R_1(t) - R_1(t-\tau_1))$ and $(D_1(t) - D_1(t-\tau_1))$ represent the number of individuals who have recovered and died from the first viral strain during the time interval $(t-\tau_1, t)$, respectively. Similarly, $(R_2(t) - R_2(t-\tau_2))$ and $(D_2(t) - D_2(t-\tau_2))$ represent the number of individuals who have recovered and died from the second viral strain during the time interval $(t-\tau_2, t)$, respectively. Moreover, the expressions $(R_{21}(t) - R_{21}(t-\tau_1))$, $(R_{12}(t) - R_{12}(t-\tau_2))$, $(D_{21}(t) - D_{21}(t-\tau_1))$, and $(D_{12}(t) - D_{12}(t-\tau_2))$ characterize the difference in the number of recovered and died individuals for two viral strains at different time intervals. Hence, from (5.26)–(5.33), we have the following:

$$I_1(t) = \int_{t-\tau_1}^{t} J_1(\eta)d\eta - (r_{1,0} + d_{1,0})\int_{t-\tau_1}^{t}\Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta\Big)ds, \tag{5.38}$$

$$I_2(t) = \int_{t-\tau_2}^{t} J_2(\eta)d\eta - (r_{2,0} + d_{2,0}) \int_{t-\tau_2}^{t} \Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta \Big) ds, \tag{5.39}$$

$$I_{21}(t) = \int_{t-\tau_1}^{t} J_{21}(\eta)d\eta - (r_{1,0} + d_{1,0}) \int_{t-\tau_1}^{t} \Big(\int_{s-\tau_1}^{s} J_{21}(\eta)d\eta \Big) ds, \tag{5.40}$$

$$I_{12}(t) = \int_{t-\tau_2}^{t} J_{12}(\eta)d\eta - (r_{2,0} + d_{2,0}) \int_{t-\tau_2}^{t} \Big(\int_{s-\tau_2}^{s} J_{12}(\eta)d\eta \Big) ds. \tag{5.41}$$

Now, from (5.38)–(5.41),

$$\begin{aligned} \frac{dI_1}{dt} &= \beta_1 \frac{S}{N} I_1 - (r_{1,0} + d_{1,0}) \int_{t-\tau_1}^{t} J_1(\eta)d\eta \\ &= \beta_1 \frac{S}{N} I_1 - (r_{1,0} + d_{1,0}) \Big[I_1(t) + (r_{1,0} + d_{1,0}) \int_{t-\tau_1}^{t} \Big(\int_{s-\tau_1}^{s} J_1(\eta)d\eta \Big) ds \Big], \end{aligned} \tag{5.42}$$

$$\begin{aligned} \frac{dI_2}{dt} &= \beta_2 \frac{S}{N} I_2 - (r_{2,0} + d_{2,0}) \int_{t-\tau_2}^{t} J_2(\eta)d\eta \\ &= \beta_2 \frac{S}{N} I_2 - (r_{2,0} + d_{2,0}) \Big[I_2(t) + (r_{2,0} + d_{2,0}) \int_{t-\tau_2}^{t} \Big(\int_{s-\tau_2}^{s} J_2(\eta)d\eta \Big) ds \Big], \end{aligned} \tag{5.43}$$

$$\begin{aligned} \frac{dI_{21}}{dt} &= \epsilon_2 \beta_1 \frac{R_2}{N} I_1 - (r_{1,0} + d_{1,0}) \int_{t-\tau_1}^{t} J_{21}(\eta)d\eta \\ &= \epsilon_2 \beta_1 \frac{R_2}{N} I_1 - (r_{1,0} + d_{1,0}) \Big[I_{21}(t) + (r_{1,0} + d_{1,0}) \int_{t-\tau_1}^{t} \Big(\int_{s-\tau_1}^{s} J_{21}(\eta)d\eta \Big) ds \Big], \end{aligned} \tag{5.44}$$

$$\begin{aligned} \frac{dI_{12}}{dt} &= \epsilon_1 \beta_2 \frac{R_1}{N} I_2 - (r_{2,0} + d_{2,0}) \int_{t-\tau_2}^{t} J_2(\eta)d\eta \\ &= \epsilon_1 \beta_2 \frac{R_1}{N} I_2 - (r_{2,0} + d_{2,0}) \Big[I_{12}(t) + (r_{2,0} + d_{2,0}) \int_{t-\tau_2}^{t} \Big(\int_{s-\tau_2}^{s} J_{12}(\eta)d\eta \Big) ds \Big]. \end{aligned} \tag{5.45}$$

Assuming that $(r_{1,0} + d_{1,0})$ and $(r_{2,0} + d_{2,0})$ are small enough, we neglect the terms involving $(r_{1,0} + d_{1,0})^2$ and $(r_{2,0} + d_{2,0})^2$. Hence, we obtain the following:

$$\frac{dI_1}{dt} \simeq \beta_1 \frac{S}{N} I_1 - (r_{1,0} + d_{1,0}) I_1, \tag{5.46}$$

$$\frac{dI_2}{dt} \simeq \beta_2 \frac{S}{N} I_2 - (r_{2,0} + d_{2,0}) I_2, \tag{5.47}$$

$$\frac{dI_{21}}{dt} \simeq \epsilon_2 \beta_1 \frac{R_2}{N} I_1 - (r_{1,0} + d_{1,0}) I_{21}, \tag{5.48}$$

$$\frac{dI_{12}}{dt} \simeq \epsilon_1 \beta_2 \frac{R_1}{N} I_2 - (r_{2,0} + d_{2,0}) I_{12}. \tag{5.49}$$

In this case, systems (5.1)–(5.13) is reduced to the following conventional ODE model:

$$\begin{aligned} \frac{dS}{dt} &= -\beta_1 \frac{S}{N} I_1 - \beta_2 \frac{S}{N} I_2, \\ \frac{dI_1}{dt} &= \beta_1 \frac{S}{N} I_1 - (r_{1,0} + d_{1,0}) I_1, \end{aligned}$$

$$\frac{dI_2}{dt} = \beta_2 \frac{S}{N} I_2 - (r_{2,0} + d_{2,0}) I_2,$$
$$\frac{dR_1}{dt} = r_{1,0} I_1 - \epsilon_1 \beta_2 \frac{R_1}{N} I_2,$$
$$\frac{dR_2}{dt} = r_{2,0} I_2 - \epsilon_2 \beta_1 \frac{R_2}{N} I_1,$$
$$\frac{dI_{21}}{dt} = \epsilon_2 \beta_1 \frac{R_2}{N} I_1 - (r_{1,0} + d_{1,0}) I_{21},$$
$$\frac{dI_{12}}{dt} = \epsilon_1 \beta_2 \frac{R_1}{N} I_2 - (r_{2,0} + d_{2,0}) I_{12},$$
$$\frac{dR_{21}}{dt} = r_{1,0} I_{21}, \quad \frac{dR_{12}}{dt} = r_{2,0} I_{12},$$
$$\frac{dD_1}{dt} = d_{1,0} I_1, \quad \frac{dD_2}{dt} = d_{2,0} I_2, \quad \frac{dD_{21}}{dt} = d_{1,0} I_{21}, \quad \frac{dD_{12}}{dt} = d_{2,0} I_{12}.$$

Therefore, if we assume that the recovery and death rates described in Eqs (5.20) and (5.21) follow uniform distributions and are sufficiently small, we can reduce the model given by Eqs (5.1)–(5.13) to the ODE model. However, in general, these assumptions may not hold. Another approximation of recovery and death rate distributions is considered in the next section.

6. Delay model

Let us recall that the duration of the first and the second disease are denoted by τ_1 and τ_2, respectively. Furthermore, suppose that individuals $J_1(t-\tau_1)$ and $J_{21}(t-\tau_1)$ infected at time $t-\tau_1$ and individuals $J_2(t-\tau_2)$ and $J_{12}(t-\tau_2)$ infected at time $t-\tau_2$, either recover or die at time t with certain probabilities. This assumption corresponds to the following choice of the functions $r_1(t-\eta)$, $r_2(t-\eta)$, $d_1(t-\eta)$ and $d_2(t-\eta)$:

$$r_1(t-\tau_1) = r_{1,0}\delta(t-\eta-\tau_1), \;\; r_2(t-\tau_2) = r_{2,0}\delta(t-\eta-\tau_2),$$
$$d_1(t-\tau_1) = d_{1,0}\delta(t-\eta-\tau_1), \;\; d_2(t-\tau_2) = d_{2,0}\delta(t-\eta-\tau_2),$$

where $r_{1,0}$, $r_{2,0}$, $d_{1,0}$, $d_{2,0}$ are constants, $r_{1,0} + d_{1,0} = 1$ and $r_{2,0} + d_{2,0} = 1$, and δ is the Dirac delta-function. Then

$$\frac{dR_1}{dt} = \int_0^t r_1(t-\eta)J_1(\eta)d\eta - J_{12}(t) = r_{1,0}\int_0^t \delta(t-\eta-\tau_1)J_1(\eta)d\eta - J_{12}(t) = r_{1,0}J_1(t-\tau_1) - J_{12}(t),$$
$$\frac{dR_2}{dt} = \int_0^t r_2(t-\eta)J_2(\eta)d\eta - J_{21}(t) = r_{2,0}\int_0^t \delta(t-\eta-\tau_2)J_2(\eta)d\eta - J_{21}(t) = r_{2,0}J_2(t-\tau_2) - J_{21}(t),$$
$$\frac{dR_{12}}{dt} = \int_0^t r_2(t-\eta)J_{12}(\eta)d\eta = r_{2,0}\int_0^t \delta(t-\eta-\tau_2)J_{12}(\eta)d\eta = r_{2,0}J_{12}(t-\tau_2),$$
$$\frac{dR_{21}}{dt} = \int_0^t r_1(t-\eta)J_{21}(\eta)d\eta = r_{1,0}\int_0^t \delta(t-\eta-\tau_1)J_{21}(\eta)d\eta = r_{1,0}J_{21}(t-\tau_1),$$
$$\frac{dD_1}{dt} = \int_0^t d_1(t-\eta)J_1(\eta)d\eta = d_{1,0}\int_0^t \delta(t-\eta-\tau_1)J_1(\eta)d\eta = d_{1,0}J_1(t-\tau_1),$$
$$\frac{dD_2}{dt} = \int_0^t d_2(t-\eta)J_2(\eta)d\eta = d_{2,0}\int_0^t \delta(t-\eta-\tau_2)J_2(\eta)d\eta = d_{2,0}J_2(t-\tau_2),$$

$$\frac{dD_{12}}{dt} = \int_0^t d_2(t-\eta)J_{12}(\eta)d\eta = d_{2,0}\int_0^t \delta(t-\eta-\tau_2)J_{12}(\eta)d\eta = d_{2,0}J_{12}(t-\tau_2),$$
$$\frac{dD_{21}}{dt} = \int_0^t d_1(t-\eta)J_{21}(\eta)d\eta = d_{1,0}\int_0^t \delta(t-\eta-\tau_1)J_{21}(\eta)d\eta = d_{1,0}J_{21}(t-\tau_1).$$

Therefore, systems (5.1)–(5.13) is reduced to the following delay model:

$$\frac{dS}{dt} = -J_1(t) - J_2(t), \tag{6.1}$$
$$\frac{dI_i}{dt} = J_i(t) - J_i(t-\tau_i), \qquad i = 1,2, \tag{6.2}$$
$$\frac{dR_i}{dt} = r_{i,0}J_i(t-\tau_i) - J_{ij}(t), \qquad i,j = 1,2, j \neq i, \tag{6.3}$$
$$\frac{dI_{ij}}{dt} = J_{ij}(t) - J_{ij}(t-\tau_j), \qquad i,j = 1,2, j \neq i, \tag{6.4}$$
$$\frac{dR_{ij}}{dt} = r_{j,0}J_{ij}(t-\tau_j), \qquad i,j = 1,2, j \neq i, \tag{6.5}$$
$$\frac{dD_i}{dt} = d_{i,0}J_i(t-\tau_i), \qquad i = 1,2, \tag{6.6}$$
$$\frac{dD_{ij}}{dt} = d_{j,0}J_{ij}(t-\tau_j), \qquad i,j = 1,2, j \neq i \tag{6.7}$$

with $J_1(t) = J_2(t) = J_{12}(t) = J_{21}(t) = 0$ for $t < 0$.

Using the arguments similar to those in Subsections 3.2 and 3.3, it can be demonstrated that the basic reproduction numbers can be expressed as follows:

$$\mathcal{R}_{0i} = \frac{\beta_i S_0 \tau_i}{N}, \quad i = 1,2.$$

Additionally, we obtain a two-sided estimate for S_∞:

$$\min(\mathcal{R}_{01},\mathcal{R}_{02})\left(1-\frac{S_\infty}{S_0}\right) \leq \ln\left(\frac{S_0}{S_\infty}\right) \leq \max(\mathcal{R}_{01},\mathcal{R}_{02})\left(1-\frac{S_\infty}{S_0}\right).$$

Using the Figure 5, we obtain the following:

$$\ln\left(\frac{S_0}{S_\infty}\right) \simeq \max(\mathcal{R}_{01},\mathcal{R}_{02})\left(1-\frac{S_\infty}{S_0}\right).$$

7. Numerical simulation

For the delay models (6.1)–(6.7), we compare the maximum number of infected individuals and the timing of this peak, as well as the total number of infected individuals caused by each virus. We choose the following values for the parameters: population size $N = 10^4$, initial infections $I_{1,0} = I_{2,0} = 1$, delays $\tau_1 = 15.7$ and $\tau_2 = 17.7$, and transmission rates $\beta_1 = 0.25$, $\beta_2 = 0.2$ and the constants $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$. Additionally, we set $\epsilon_1 = 0.8$ and $\epsilon_2 = 1$.

In the first infection, where $\beta_1 > \beta_2$, Figure 6 shows that the maximal number of infected individuals during the first strain is higher than the maximal number of infected individuals observed during the second strain. In the second infection, it is worth noting that the maximum value of I_{21} is lower than the maximum value of I_{12} where, in this case, the maximum depends on the parameters $\beta_1, \beta_2, \epsilon_1, \epsilon_2, r_{1,0}$ and $r_{2,0}$.

The maximum number of infected individuals behaves similarly to a single strain scenario, where the maximum is determined by the strain with the larger transmission rate (i.e, $(I_1 + I_{21})_m > (I_2 + I_{12})_m$). Furthermore, with regards to the timing of the maximum, the difference between the transmission rates does not strongly influence on the peak time. Thus, t_{1m} is very close to t_{2m}.

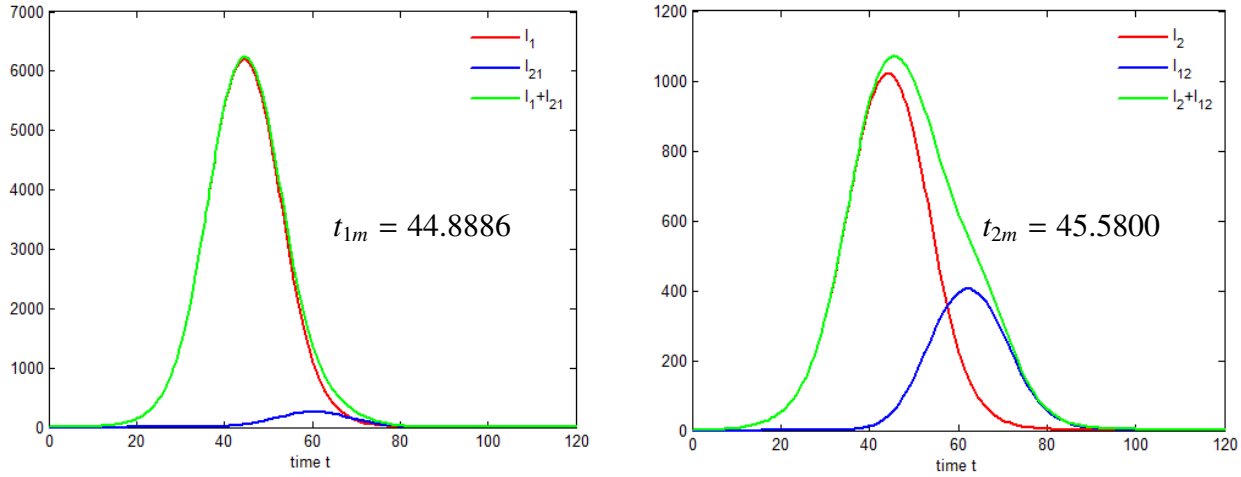

Figure 6. The number of infected individuals by the first strain (left) and the second strain (right). The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $\beta_1 = 0.25$, $\beta_2 = 0.2$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

Next, let us consider another set of parameters - $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$, $\tau_2 = 17.7$ - and vary the values of β_1 and β_2 within the range of $[0.1, 0.5]$. During the first infection with a fixed transmission rate β_2 ($\beta_2 < \beta_1$), increasing the transmission rate β_1 causes the total number of individuals infected by the first strain ($R_{1f} + D_{1f}$) to approach the entire population N, while the total number of individuals infected by the second strain ($R_{2f} + D_{2f}$) diminishes significantly, nearly reaching zero. Conversely, with a fixed transmission rate β_1 ($\beta_1 < \beta_2$), by increasing the transmission rate β_2, the total number of individuals infected by the second strain ($R_{2f} + D_{2f}$) approaches the entire population N, while the total number of individuals infected by the first strain ($R_{1f} + D_{1f}$) decreases significantly, nearly reaching zero. Notably, 70% of those infected by the first strain and 60% of those infected by the second strain have recovered.

In the second infection, by increasing the transmission rate β_1 ($\beta_2 < \beta_1$), the total number of individuals infected by the second strain ($R_{12f} + D_{12f}$) approaches 70% of the total population N, while the total number of individuals infected by the first strain ($R_{21f} + D_{21f}$) approaches a very small value close to zero. On the other hand, increasing the transmission rate β_2 ($\beta_1 < \beta_2$) causes the total number of individuals infected by the first strain ($R_{21f} + D_{21f}$) to approach 60% of the total population N, while the total number of individuals infected by the second strain ($R_{12f} + D_{12f}$)

approaches a very small value close to zero.

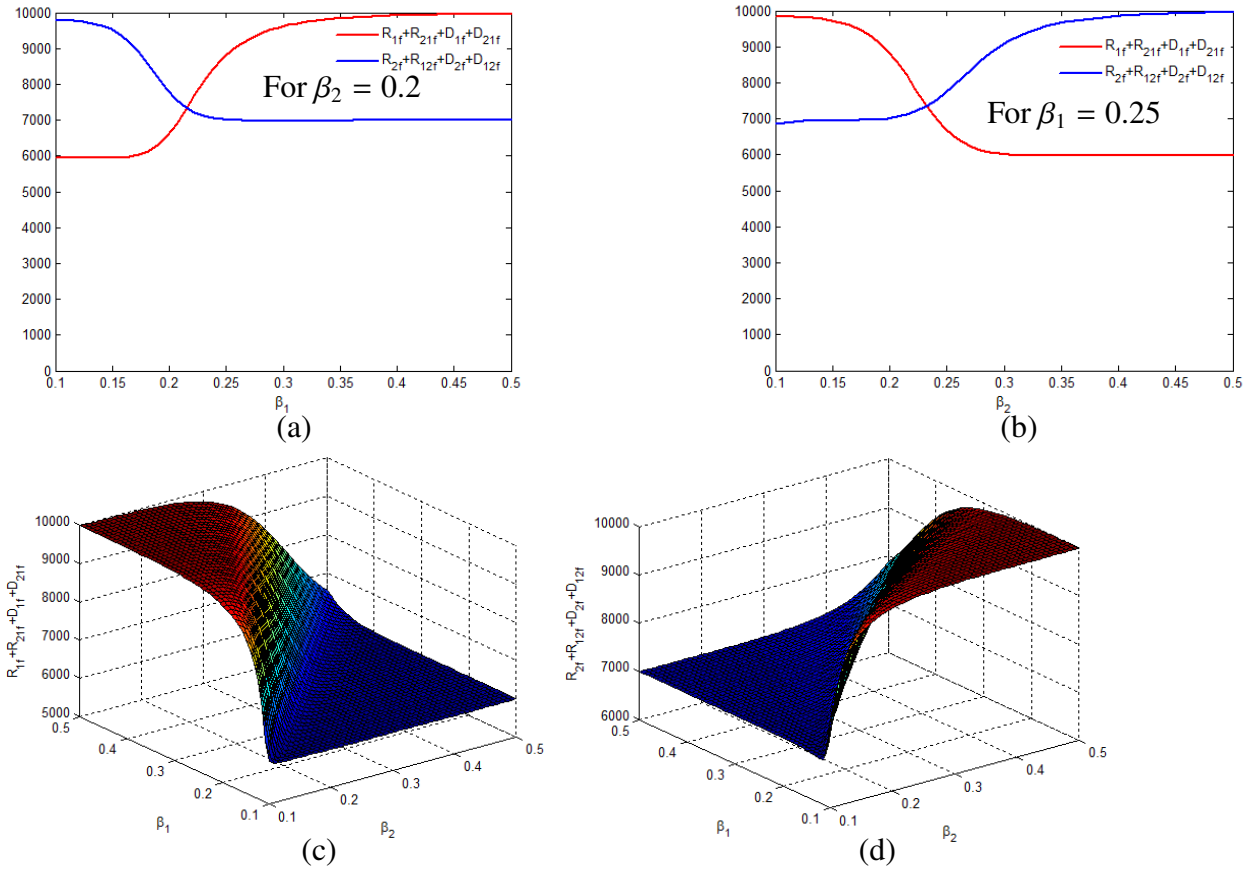

Figure 7. Dependence of the total number of infected by each strain on : (a) β_1 for $\beta_2 = 0.2$ (b) β_2 for $\beta_1 = 0.25$. Dependence of the total number of infected on β_1, β_2 by : (c) the first strain (d) the second strain. The values of parameters: $N = 10^4$, $I_{1,0} = I_{2,0} = 1$, $r_{1,0} = 0.7$, $r_{2,0} = 0.6$, $d_{1,0} = 0.3$, $d_{2,0} = 0.4$, $\tau_1 = 15.7$ and $\tau_2 = 17.7$.

Consequently, if β_1 is increased, then the number of individuals affected by the first virus ($R_{1f} + D_{1f} + R_{21f} + D_{21f}$) also increases. This indicates that a higher transmission rate for the first virus leads to a larger proportion of the population being infected by it.

Conversely, with an increase in β_1, the number of individuals affected by the second virus ($R_{2f} + D_{2f} + R_{12f} + D_{12f}$) gradually decreases. This suggests that the transmission of the second virus diminishes, thereby resulting in a lower number of individuals becoming infected by it.

Similarly, when β_2 is changed while keeping β_1 constant, increasing β_2 can lead to a higher number of individuals being affected by the second virus, while the number of individuals affected by the first virus may decrease. Note that the total number of infected individuals exceeds the total population because there are individuals who have been infected by both strain.

8. Discussion

In this paper we introduced two epidemiological models, each of which described the dynamics of the dual viral strain SIRD model with distributed recovery and death rates to track the impact of multiple strains variants during an epidemic. In the first model, some individuals may be infected by the first strain and others by the second strain. Furthermore, individuals who have recovered from either strain develop immunity to both strains. In the second model, we explored the scenario where individuals who have recovered from the first strain can be infected by the second strain, and similarly, individuals who have recovered from the second strain can be infected by the first strain.

The proposed distributed delay models, represented by integro-differential equations, offer a more accurate description of epidemic dynamics. However, these models are relatively complex and require knowledge of distributed recovery and death rates, which may not be readily available in the literature. To compensate this disadvantage of the distributed models, we derived two simplified models from each model to address different limiting cases. In the first case, assuming that the recovery and death rates are uniformly distributed, we obtained a conventional compartmental ODE model. In the second case, considering the recovery and death rate distributions as delta-function, we obtained a novel delay model that has not been previously explored in the epidemiological literature. As mentioned in [43–45], since distributed recovery and death rates are commonly described by a Gamma-distribution, approximating them with a delta-function can provide a more accurate representation, especially in capturing the dynamics during the initial phase of infection. The point-wise delay models, based on the duration of the disease and known recovery and death rates, provide a simple and biologically meaningful representation of epidemics.

For these delay model we determined the reproduction numbers for each strain $\mathcal{R}_{0j}, \quad j = 1, 2$. We employed these delay models to perform a numerical comparison of the maximum number of infected individuals,the peak timing and the total number of infected cases by each virus. This analysis was conducted by utilizing the final size of the recovered and deceased individuals. The transmission rates of the diseases played a crucial role in determining these outcomes, as they significantly influenced the dynamics of the epidemics being studied.

We compared the two models with and without cross-immunity. The maximum number of infected individuals in the model without cross-immunity was higher than in the other model because there were individuals who had been infected with both viruses. The timing of the maximum was either equal or very close between the two models. In the model without cross-immunity, the total number of infected individuals surpassed the total population because some individuals could be infected by both strains simultaneously. As a consequence, the number of infected individuals would be higher compared to the total number of infected individuals in the other model (with cross-immunity). As result, cross-immunity between the two viruses can significantly reduce their global spread. Individuals already immunized against one of the viruses would decrease the total number of cases of diseases caused by these pathogens. If a large part of the population acquires this cross-immunity, it would limit the spread of viruses and minimize their impact on public health. In the absence of cross-immunity, the spread of both viruses would accelerate, potentially leading to serious co-infections and increasing the burden on healthcare

systems. Therefore, it is crucial to understand and promote cross-immunity to better prevent and control these viral diseases.

Comparing our results with the data, we used the example of Omicron and Delta variants in COVID-19, where Omicron exhibited a higher transmission rate. In [46], the authors showed that the duration of encounters played a crucial role in modeling and significantly impacted disease progression and the competition between variants, where the faster spreading strain dominated over the slower strain. Reference [12] reports that Omicron rapidly spread across France and displayed higher fitness compared to Delta. Additionally, reference [13] estimated that the total number of individuals infected by Omicron exceeds the total number of those infected by Delta. All of these works confirm our result that the rate of transmission is crucial in which strain dominates the other. In general, the strain with a higher transmission rate (Omicron) tends to have a higher maximum number of infected individuals and a higher total number of infected individuals compared to the other strain (Delta).

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Conflict of interest

The authors declare there is no conflict of interest.

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