GLOBAL DYNAMICS OF A TWO-STRAIN DISEASE MODEL WITH LATENCY AND SATURATING INCIDENCE RATE

Dedicated to Professor H.I. Freedman's 70th birthday

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ABSTRACT. This paper deals with a vector-borne disease model containing latency and nonlinear incidence rates. Global dynamics of the model is completely determined by suitable Lyapunov functionals. If the basic reproduction number is less than one, then disease dies out, but if the number is larger than one, we found that one or both of the strains become endemic. A unique co-endemic equilibrium appears when both the boundary equilibria exist but are unstable, and this in contrast to the situation when mass action incidence is adopted in which co-persistence is impossible and competition exclusion is generic. It is also found that the persistence of a strain not only depends on the respective reproduction number but also depends on the combined parameters and a strain may disappear even though the strain specific reproduction number is larger than one. The higher saturation level of one strain may result in emerge or extinction of the other strain in some situations.

1 Introduction Cooke \cite{7} presented a mathematical model to describe the dynamics of a communicable disease through a vector population based on the scenario below. Human beings are divided into three classes: the susceptible class with its population denoted by $S(t)$, the infective class with its population denoted by $I(t)$, and the removed class with its population denoted by $R(t)$. The susceptible vectors are infected by infectious human individuals. It was assumed in \cite{7} that it takes an infected vector $\tau$ time units to become infectious. Being infectious, the vectors then infect susceptible human individuals. Denote the populations of susceptible and infectious vectors at time $t$ by $V_S(t)$ and $V_I(t)$ respectively. As a vector population is usually quite large,
it was also simply assumed in [7] that $V_I(t)$ is proportional to $I(t - \tau)$. Let $k$ be the proportionality, that is, $V_I(t) = kI(t - \tau)$. If the mass action infection mechanism is adopted, that is, $\beta V_I(t)S(t)$, then the force of infection for human beings at time $t$ is then given by

$$\beta V_I(t)S(t) = \beta kI(t - \tau)S(t) = \beta I(t - \tau)S(t),$$

where $\beta = \hat{\beta}k$, with $k$ possibly depending on the length of latency $\tau$. This leads to the following model of delay differential equation

$$\begin{cases}
\dot{S}(t) = \Lambda - \mu S(t) - \beta S(t)I(t - \tau), \\
\dot{I}(t) = \beta S(t)I(t - \tau) - (\mu + \gamma)I(t), \\
\dot{R}(t) = \gamma I(t) - \mu R(t),
\end{cases}$$

where $R$ is the population of the removed class. For more biological explanations and the mathematical results for this model; see [3, 14, 18].

The mass action law for infection is the simplest mechanism since it assumes a linear incidence rate $g(V_I) = V_I$. Motivated by Capasso [5], many authors (see, e.g., [6, 11, 22]) have used a saturating incidence rate defined by

$$h(V_I) = \frac{\beta V_I}{1 + \sigma V_I},$$

where $\sigma > 0$ determines the saturation levels when the infectious population is large. When $\sigma = 0$, this reduces to the mass action incidence rate. Thus the saturated incidence rate is a generalization of mass action incidence rate and is more reasonable as it reflects the crowding effect of the infective individuals. Using the this saturation incidence function and under the proportion assumption suggested in [7], the model (1.2) is generalized to the following model

$$\begin{cases}
\dot{S}(t) = \Lambda - \mu S(t) - \frac{\beta S(t)I(t - \tau)}{1 + \sigma I(t - \tau)}, \\
\dot{I}(t) = \frac{\beta S(t)I(t - \tau)}{1 + \sigma I(t - \tau)} - (\mu + \gamma)I(t), \\
\dot{R}(t) = \gamma I(t) - \mu R(t),
\end{cases}$$

where $\sigma = \alpha k$. In [19], Xu and Ma analyzed the stability of this SIR epidemic model. In particular, when the basic reproduction number is larger than 1, they
showed that the endemic equilibrium is globally asymptotically stable under certain extra conditions. In a recent work, by using a Lyapunov functional, McCluskey [16] was able to prove the global stability of endemic equilibrium without the extra conditions.

It is well known that mutation of a pathogen is common and causes serious problems in treating the resulting disease. Thus, one often needs to deal with more than one strain. Hence, the study of disease dynamics with multiple strains is an important research topic. An example is the influenza in 2009: in addition to the seasonal influenza, the H1N1 influenza also emerged and became pandemic. Co-infection of vector-borne diseases (e.g. malaria, dengue, leptospirosis) is also a possible phenomenon, although it is not that frequent [20] at the present. The management strategy with two vector-borne diseases or two strains of one vector-borne disease is a challenging task. Indeed, the dynamics of a single vector-borne disease with multiple strains has attracted many researchers, and yet, not too many studies on this have been documented [2, 4, 17]. For instance, the dynamics of acute infection of human malaria parasites such as Plasmodium falciparum and Plasmodium vivax are not fully elucidated [1, 15]. It is widely agreed that mathematical modeling is an effective tool for developing strategies to control possible outbreaks of diseases. In this paper, we present a mathematical model to describe the dynamics of a vector-borne disease with two strains along the lines of [7, 16, 19]. By analyzing this two-strain model with time delays and saturating incidence rates, we hope to shed some light on how the interaction of the two strains affect the disease dynamics.

The rest of this paper is organized as follows. In Section 2, we formulate a two-strain disease model based on those in [7, 16, 19]. Equilibria and the basic reproduction number of the model are discussed in Section 3. Section 4 deals with global stability of the equilibria. Section 5 provides some numeric simulation results which agree with the theoretical results in Section 4. Finally, Section 6 offers some concluding remarks and discussion.

2 A two-strain disease model The model we present here is a straightforward modification of (1.4) by incorporating another strain of the disease. To proceed, we denote by $S$, $I_1$ and $I_2$ the sub-populations of susceptible class, infective classes with strain 1 and strain 2, respectively. Let $R$ still be the population of the removed class. Following the discussion in the introduction, the vector’s sub-populations can be omitted from the equations by including delays in the infectious classes of human beings. Based on (1.4), the dynamics of such a vector-borne disease with two strains, assuming a saturating incidence
rate, is thus governed by the following system of delay differential equations:

\[
\begin{aligned}
    \dot{S} &= \Lambda - \mu S - \frac{\beta_1 SI_{1}}{1 + \alpha_1 I_{1_t}} - \frac{\beta_2 SI_{2}}{1 + \alpha_2 I_{2_t}}, \\
    \dot{I}_1 &= \frac{\beta_1 SI_{1}}{1 + \alpha_1 I_{1_t}} - (\mu_1 + \gamma_1)I_1, \\
    \dot{I}_2 &= \frac{\beta_2 SI_{2}}{1 + \alpha_2 I_{2_t}} - (\mu_2 + \gamma_2)I_2, \\
    \dot{R} &= \gamma_1 I_1 + \gamma_2 I_2 - \mu R.
\end{aligned}
\]  

(2.1)

Here, the parameters in the model are summarized in Table 1. Also, to avoid excessive brackets and notational ambiguity, we use the following conventions: \(I_{1_t}(t) = I_1(t - \tau_1), I_{2_t}(t) = I_2(t - \tau_2).\)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\Lambda)</td>
<td>Recruitment of individuals</td>
</tr>
<tr>
<td>(1/\mu)</td>
<td>Life expectancy</td>
</tr>
<tr>
<td>(\beta_1)</td>
<td>Transmission coefficient of susceptible individuals to strain 1</td>
</tr>
<tr>
<td>(\beta_2)</td>
<td>Transmission coefficient of susceptible individuals to strain 2</td>
</tr>
<tr>
<td>(1/\gamma_1)</td>
<td>Average infected period of strain 1</td>
</tr>
<tr>
<td>(1/\gamma_2)</td>
<td>Average infected period of strain 2</td>
</tr>
<tr>
<td>(\mu_1)</td>
<td>Combination of infection induced death rate and natural death rate of strain 1</td>
</tr>
<tr>
<td>(\mu_2)</td>
<td>Combination of infection induced death rate and natural death rate of strain 2</td>
</tr>
</tbody>
</table>

TABLE 1: Description of variables and parameters of model (2.1).

For biological reasons, it is natural to pose the following conditions on initial values of the unknowns:

\[
S(0) > 0 \quad \text{and} \quad I_i(\theta) = \phi_i(\theta) \geq 0 \quad \text{for} \quad \theta \in [-\tau_i, 0], \quad i = 1, 2,
\]  

(2.2)

where

\[
\phi_i \in C([-\tau_i, 0], \mathbb{R}_+).
\]  

(2.3)
3 Basic properties of the model

Applying the standard argument, one can easily show that for initial functions satisfying (2.2)–(2.3), the system (2.1) has a unique solution with all components being non-negative. Adding all equations of (2.1), the total population $N = S + I_1 + I_2 + R$ satisfies

$$\dot{N} \leq \Lambda - \mu N.$$ 

Here, we have used the reasonable assumption

$$\mu \leq \min \{\mu_1, \mu_2\},$$

meaning the mortality rates for infectious individuals are not less than that of the susceptible individuals. By comparison theorem, it follows that

$$\limsup_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu}.$$ 

This suggests confining ourselves to the biologically feasible region

$$\Gamma = \left\{ (S, I_1, I_2, R) : S, I_1, I_2, R \geq 0, S + I_1 + I_2 + R \leq \frac{\Lambda}{\mu} \right\}.$$ 

Since $R$ is decoupled in (2.1), it is sufficient and reasonable to analyze the following reduced dimensional system

$$\begin{cases}
\dot{S} = \Lambda - \mu S - \frac{\beta_1 SI_1}{1 + \alpha_1 I_1} - \frac{\beta_2 SI_2}{1 + \alpha_2 I_2}, \\
\dot{I}_1 = \frac{\beta_1 SI_1}{1 + \alpha_1 I_1} - (\mu_1 + \gamma_1)I_1, \\
\dot{I}_2 = \frac{\beta_2 SI_2}{1 + \alpha_2 I_2} - (\mu_2 + \gamma_2)I_2.
\end{cases}$$

(3.2)

Corresponding to (3.1), we only need to consider for (3.2) the following set

$$\Gamma_1 = \left\{ (S, I_1, I_2) : S, I_1, I_2 \geq 0, S + I_1 + I_2 \leq \frac{\Lambda}{\mu} \right\},$$

(3.3)

which is obviously positively invariant for (3.2).
3.1 Steady states The system has a disease-free equilibrium (DFE) $E_0$ given by

$$E_0 = (S_0, 0, 0) \text{ with } S_0 = \frac{\Lambda}{\mu}.$$ 

There are two possible single-infection equilibria, $E_1$ and $E_2$, given by

$$E_1 = (\bar{S}, \bar{I}_1, 0) \quad \text{and} \quad E_2 = (\bar{S}, 0, \bar{I}_2),$$

where

$$\bar{S} = \frac{1}{\beta_1} (\mu_1 + \gamma_1)(1 + \alpha_1 \bar{I}_1), \quad \bar{I}_1 = \frac{\mu}{\alpha_1 \mu + \beta_1} (\mathcal{R}_i - 1),$$

and

$$\bar{S} = \frac{1}{\beta_2} (\mu_2 + \gamma_2)(1 + \alpha_2 \bar{I}_2), \quad \bar{I}_2 = \frac{\mu}{\alpha_2 \mu + \beta_2} (\mathcal{R}_2 - 1),$$

where

$$\mathcal{R}_i = \frac{S_0 \beta_i}{\mu_i + \gamma_i}, \quad i = 1, 2.$$ 

Clearly, for $i = 1, 2$, $E_i$ is biologically meaningful if and only if $\mathcal{R}_i > 1$.

Other than the two single-infection equilibria, there is a possible co-existence equilibrium (all components are positive) $E^* = (S^*, \bar{I}_1^*, \bar{I}_2^*)$, where

$$\bar{I}_i^* = \frac{1}{\alpha_i} \left( \frac{\beta_i S^*}{\mu_i + \gamma_i} - 1 \right), \quad i = 1, 2,$$

and

$$S^* = \frac{\alpha_1 \alpha_2 \Lambda + \alpha_1 (\mu_2 + \gamma_2) + \alpha_2 (\mu_1 + \gamma_1)}{\alpha_1 \alpha_2 \mu + \beta_1 \alpha_2 + \beta_2 \alpha_1}.$$ 

It is readily seen that the co-existence equilibrium is biologically meaningful if and only if

$$\frac{\beta_i S^*}{\mu_i + \gamma_i} > 1, \quad i = 1, 2.$$ 

The following theorem shows that the existence of both boundary equilibria $E_1$ and $E_2$ is a prerequisite for the existence of the co-existence equilibrium $E^*$.

**Theorem 3.1.** Let $\mathcal{R}_m = \min \{\mathcal{R}_1, \mathcal{R}_2\}$. If $E^*$ exists, then $\mathcal{R}_m > 1$. 

Proof. Let \( E^* \) exists. Then both \( I_1^* \) and \( I_2^* \) are positive. Therefore,

\[
\Lambda = \mu S^* + \frac{\beta_1 S^* I_1^*}{1 + \alpha_1 I_1^*} + \frac{\beta_2 S^* I_2^*}{1 + \alpha_2 I_2^*} > \mu S^*
\]

which implies that \( S_0 = \Lambda/\mu > S^* \). Thus, if (3.4) holds, then

\[
\frac{\beta_i S_0}{\mu_i + \gamma_i} > 1, \quad i = 1, 2,
\]

which implies \( R_i > 1, \ i = 1, 2 \) and thus, \( R_m > 1 \). This completes the proof. \( \square \)

By the proof and similar argument, we actually have the following observations:

(O1) If \( R_1 > 1 \), then \( S_0 > S^* \);
(O2) If \( R_2 > 1 \), then \( S_0 > S^* \);
(O3) If \( R_m > 1 \), then \( S_0 > S^* \).

In Section 4, we will further explore sufficient conditions for the existence of the co-existence equilibrium \( E^* \).

3.2 Basic reproduction number  The basic reproduction number for the model, denoted by \( R_0 \), plays an important role in determining the disease persistence. The number \( R_0 \) is defined as “the expected number of secondary cases produced, in a completely susceptible population, by a typical infected individual” (see, e.g., [8]). For a single strain infectious disease model, it is usually not hard to compute \( R_0 \); however, for a multi-strain model the task becomes harder. Following [21], we will use next-generation matrix to compute \( R_0 \). The non-negative matrix \( F \) and the non-singular M-matrix \( V \), known as new-infection and transition matrices respectively for the system (3.2), are given by

\[
F = \begin{pmatrix}
\frac{\Delta \beta_1}{\mu} & 0 \\
0 & \frac{\Delta \beta_2}{\mu}
\end{pmatrix}, \quad V = \begin{pmatrix}
\mu_1 + \gamma_1 & 0 \\
0 & \mu_2 + \gamma_2
\end{pmatrix}.
\]

It follows that

\[
FV^{-1} = \begin{pmatrix}
\frac{\Delta \beta_1}{\mu(\mu_1 + \gamma_1)} & 0 \\
0 & \frac{\Delta \beta_2}{\mu(\mu_2 + \gamma_2)}
\end{pmatrix} = \begin{pmatrix}
R_1 & 0 \\
0 & R_2
\end{pmatrix}.
\]

The basic reproduction number is then given by the spectrum radius of \( FV^{-1} \), that is

\[
R_0 = \rho \left( FV^{-1} \right) = \max \{ R_1, R_2 \}.
\]
By Theorem 2 in [21], we have the following theorem relating the stability/instability of $E_0$ to the value of $\mathcal{R}_0$.

**Theorem 3.2.** $E_0$ is asymptotically stable, if $\mathcal{R}_0 < 1$; and it becomes unstable if $\mathcal{R}_0 > 1$.

In this connection we note that, if $\mathcal{R}_0 < 1$, both the single-infection equilibria $E_1$ and $E_2$ do not exist.

### 4 Global stability analysis

In this section, we discuss global stability of each of the equilibria. To this end, we apply Lyapunov functionals similar to those recently used by [10, 12, 16]. Such Lyapunov functionals take advantages of the properties of the function

$$g(x) = x - 1 - \ln(x),$$

which is positive in $\mathbb{R}_+$ except at $x = 1$ where it vanishes. For convenience of notations in constructing Lyapunov functionals, we will also make use of the following two functions:

$$f_i(x) = \frac{x}{1 + \alpha_i x}, \quad i = 1, 2.$$

In the rest of this section, we show that each of the equilibria exhibits global stability under some threshold conditions. We begin with the DFE $E_0$.

**Theorem 4.1.** When $\mathcal{R}_0 < 1$, $E_0$ is indeed globally asymptotically stable.

**Proof.** Consider the Lyapunov functional

$$V(S, I_1, I_2) = S_0 g \left( \frac{S}{S_0} \right) + I_1 + \beta_1 S_0 \int_{-\tau_1}^{0} I_1(t + \theta) \, d\theta + I_2 + \beta_2 S_0 \int_{-\tau_2}^{0} I_2(t + \theta) \, d\theta.$$

Obviously, $V$ is non-negative in the positive cone $\Omega = \mathbb{R}_+ \times C([-\tau_1, 0], \mathbb{R}_+) \times C([-\tau_2, 0], \mathbb{R}_+)$ and attains zero at $E_0$. We need to show that $\dot{V}$ is negative definite. Differentiating $V$ along the trajectories of (3.2), we obtain

$$\dot{V} = \left(1 - \frac{S_0}{S} \right) \dot{S} + \dot{I}_1 + \beta_1 S_0 (I_1 - I_{\tau_1}) + \dot{I}_2 + \beta_2 S_0 (I_2 - I_{\tau_2})$$
Therefore, $\dot{V} \leq 0$ if $\mathcal{R}_e < 1$ with equality holding only at $E_0$. By Theorem 5.3.1 of [9], the solutions approach $\mathcal{M}$, the largest invariant subset of $\{dV/dt = 0\}$. Since $dV/dt$ is zero only at $E_0$, $\mathcal{M} = \{E_0\}$ is a singleton set. Thus, the equilibrium $E_0$ is globally attractive if $\mathcal{R}_e < 1$. Combining this fact with Theorem 3.2, we conclude that the DFE is indeed globally asymptotically stable if $\mathcal{R}_e < 1$.

**Theorem 4.2.** If the single-infection equilibrium $E_1$ exists (i.e. $\mathcal{R}_e > 1$), but $E_2$ does not exist (i.e., $\mathcal{R}_e \leq 1$), then $E_1$ is globally attractive.

**Proof.** Consider the Lyapunov functional
\[ V = \frac{1}{\beta_1 f_1(I_1)} V_S + \frac{I_1}{\beta_1 S f_1(I_1)} V_I + \nabla I_1 + \frac{1}{\beta_1 S f_1(I_1)} I_2 + \frac{\beta_2}{\beta_1 f_1(I_1)} \int_{-\tau_2}^0 I_2(t + \theta) d\theta. \]

where

\[ V_S = g \left( \frac{S}{S} \right), \quad V_I = g \left( \frac{I_1}{I_1} \right), \quad \nabla I_1 = \int_0^{\tau_1} g \left( \frac{I_1(t - s)}{I_1} \right) ds. \]

By the properties of \( g \) function, it is easy to see that the Lyapunov functional \( V \) is non-negative for non-negative variables and attains zero at \( E_1 \), i.e., \( V \) is positive definite. We need to show \( \dot{V} \) is negative definite. Differentiating \( V \) along the solution of (3.2), we obtain

\[ \dot{V} = \frac{1}{\beta_1 f_1(I_1)} \dot{V}_S + \frac{I_1}{\beta_1 S f_1(I_1)} \dot{V}_I + \nabla \dot{I}_1 + \frac{1}{\beta_1 S f_1(I_1)} \dot{I}_2 + \frac{\beta_2}{\beta_1 f_1(I_1)} (I_2 - I_{\tau_2}). \]

For the first derivative on the right hand side, we further calculate it as

\[ \dot{V}_S = \frac{1}{S} \left( 1 - \frac{\dot{S}}{S} \right) \dot{S} \]
\[ = \frac{1}{S} \left( 1 - \frac{\dot{S}}{S} \right) \left( \Lambda - \mu S - \beta_1 S f_1(I_{\tau_1}) - \beta_2 S f_2(I_{\tau_2}) \right) \]
\[ = \frac{1}{S} \left( 1 - \frac{\dot{S}}{S} \right) \left( \mu \dot{S} + \beta_1 S f_1(I_{\tau_1}) - \mu S - \beta_1 S f_1(I_{\tau_1}) - \beta_2 S f_2(I_{\tau_2}) \right) \]
\[ = \frac{1}{S} \left( 1 - \frac{\dot{S}}{S} \right) \left\{ \mu (\dot{S} - S) + \beta_1 (S f_1(I_{\tau_1}) - S f_1(I_{\tau_1})) - \beta_2 S f_2(I_{\tau_2}) \right\} \]
\[ = - \frac{\mu}{SS} (S - S)^2 + \beta_1 f_1(I_{\tau_1}) \left( 1 - \frac{\dot{S}}{S} \right) \left( 1 - \frac{S f_1(I_{\tau_1})}{\dot{S} f_1(I_{\tau_1})} \right) \]
\[ - \frac{S \beta_2}{S} \left( 1 - \frac{\dot{S}}{S} \right) f_2(I_{\tau_2}). \]

Let

\[ x = \frac{S}{S}, \quad y = \frac{I_1}{I_1}, \quad z = \frac{I_{\tau_2}}{I_1}, \]

and

\[ F_1(z) = \frac{f_1(I_{\tau_1})}{f_1(I_1)} = \frac{f_1(I_{\tau_1})}{f_1(I_1)}. \]
Then we can write
\[
\dot{V}_S = -\frac{\mu}{SS} (S - S)^2 + \beta_1 f_1(I_1) \left( 1 - \frac{1}{x} - x F_1(z) + F_1(z) \right) - x \beta_2 \left( 1 - \frac{1}{x} \right) f_2(I_{\tau_2}).
\]

Similarly, we calculate \( \dot{V}_{I_1} \) as
\[
\dot{V}_{I_1} = \frac{1}{I_1} \left( 1 - \frac{I_1}{I_1} \right) I_1
\]
\[
= \frac{1}{I_1} \left( 1 - \frac{I_1}{I_1} \right) \left( \beta_1 S f_1(I_1) \frac{S f_1(I_{\tau_1})}{f_1(I_1)} - (\mu_1 + \gamma_1) I_1 \right)
\]
\[
= \frac{1}{I_1} \left( 1 - \frac{I_1}{I_1} \right) \left( \beta_1 S f_1(I_1) \frac{S f_1(I_{\tau_1})}{f_1(I_1)} - (\mu_1 + \gamma_1) I_1 \frac{I_1}{I_1} \right).
\]

Using the equations relating to the equilibrium \( E_1 \), it follows that
\[
\dot{V}_{I_1} = \frac{1}{I_1} \left( 1 - \frac{I_1}{I_1} \right) \beta_1 S f_1(I_1) \left( \frac{S f_1(I_{\tau_1})}{f_1(I_1)} - \frac{I_1}{I_1} \right)
\]
\[
= \frac{1}{I_1} \left( 1 - \frac{I_1}{I_1} \right) \beta_1 S f_1(I_1) \left( x F_1(z) - y \right)
\]
\[
= \frac{1}{I_1} \beta_1 S f_1(I_1) \left( x F_1(z) - y - \frac{x F_1(z)}{y} + 1 \right).
\]

Now calculating the derivative of \( \nabla_{I_1} \), we obtain
\[
\dot{V}_{I_1} = \frac{d}{dt} \int_{0}^{\tau_1} g \left( \frac{I_1(t - s)}{I_1} \right) ds = \frac{d}{dt} \int_{t - \tau_1}^{t} g \left( \frac{I_1(s)}{I_1} \right) ds
\]
\[
= g \left( \frac{I_1(t)}{I_1} \right) - g \left( \frac{I_1(t - \tau_1)}{I_1} \right)
\]
\[
= g(y) - g(z)
\]
\[
= y - z + \ln z - \ln y.
\]

Combining the above calculation, we obtain
\[
\dot{V} = -\frac{\mu}{\beta_1 f_1(I_1)} \left( \frac{S - \bar{S}}{SS} \right)^2 + \left( 1 - \frac{1}{x} - x F_1(z) + F_1(z) \right)
\]
\[-\frac{x\beta_2}{\beta_1} \left( 1 - \frac{1}{x} \right) \frac{f_2(I_2)}{f_1(I_1)} + \left( x F_1(z) - y - \frac{xF_1(z)}{y} + 1 \right) + (y - z + \ln z - \ln y) + \frac{1}{\beta_1 S f_1(I_1)} \left( \frac{\beta_2 S I_2}{1 + \alpha_2 I_2} - (\mu_2 + \gamma_2) I_2 \right) + \frac{\beta_2}{\beta_1 f_1(I_1)} (I_2 - I_{\tau_2}). \]

Adding and subtracting among the first five terms except the third one, and grouping the resulting terms in a square bracket, we get

\[
\dot{V} = \left[ -\frac{\mu}{\beta_1 f_1(I_1)} \frac{(S - \bar{S})^2}{SS} + \left( 1 - \frac{1}{x} - \ln x \right) + \left( 1 - \frac{xF_1(z)}{y} + \ln \left( \frac{xF_1(z)}{y} \right) \right) \right] + (F_1(z) - z + \ln z - \ln F_1(z)) - \frac{x\beta_2}{\beta_1} \left( 1 - \frac{1}{x} \right) \frac{f_2(I_{\tau_2})}{f_1(I_1)} + \frac{1}{\beta_1 S f_1(I_1)} \left( \beta_2 S f_2(I_{\tau_2}) - (\mu_2 + \gamma_2) I_2 \right) + \frac{\beta_2}{\beta_1 f_1(I_1)} (I_2 - I_{\tau_2}).
\]

(4.1)

Obviously, each of the first three terms in the square bracket is non-positive. For the fourth term in square bracket, we further calculate it as

\[
(4.2) \quad F_1(z) - z + \ln z - \ln F_1(z) = (F_1(z) - z) \left[ 1 - \frac{\ln F_1(z) - \ln z}{F_1(z) - z} \right]
= (F_1(z) - z) \left[ 1 - \frac{1}{\xi} \right],
\]

where, by the Lagrange’s intermediate value theorem, \( \xi \) is a number between \( z \) and \( F_1(z) \). Note that \( F_1(z) \) is an increasing and concave down function.
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satisfying $F_1(1) = 1$. Thus, for $z < 1$, $z < F_1(z) < 1$ implying $\xi < 1$ and hence $(\eta_1 - z) \left[ 1 - 1/\xi \right] < 0$. Similarly, for $z > 1$, we have $z > F_1(z) > 1$ and hence $\xi > 1$, also leading to $(\eta_1 - z) \left[ 1 - 1/\xi \right] < 0$. Therefore,

(4.3) \quad F_1(z) - z + \ln z - \ln F_1(z) < 0

for all $z > 0$ except at $z = 1$ where it vanishes.

Therefore, the square bracket term is negative except for $S = \bar{S}, x = y = z = 1$, at which the square bracket becomes zero.

Next, we estimate the remaining parts on the right hand side of (4.1) as

$$
\frac{\eta_2}{\eta_1} \left( 1 - \frac{1}{x} \right) \frac{f_2(I_2)}{f_1(I_1)} + \frac{1}{\eta_1 f_1(I_1)}
\times \left( \eta_2 f_2(I_2) - (\mu_2 + \gamma_2) I_2 \right) + \frac{\beta_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)
\leq \frac{\beta_2}{\eta_1 f_1(I_1)} I_\tau_2 + \frac{(\mu_2 + \gamma_2)}{\eta_1 f_1(I_1)} I_2 + \frac{\beta_2 I_2}{\eta_1 f_1(I_1)} (I_2 - I_\tau_2)

Therefore, $\dot{V} \leq 0$ with equality holding only at $E_1$. By (9), all positive solutions approach $\mathcal{M}$, the largest invariant subset of in the set $\{ dV/dt = 0 \}$.

Since $dV/dt$ is zero only at $E_1$, $\mathcal{M} = \{ E_1 \}$ is a singleton set. Thus, the equilibrium $E_1$ is globally attractive. □
By symmetry, we can prove the following theorem parallel to Theorem 4.2 in a similar fashion.

**Theorem 4.3.** Assume that $E_2$ exists (i.e., $R_2 > 0$), but $E_1$ does not exist (i.e., $R_1 \leq 1$). Then $E_2$ is globally attractive.

**Proof.** In this case, we consider the following Lyapunov functional

$$V = \frac{1}{\beta_2 f_2(I_2)} V_S + \frac{I_1}{\beta_2 S f_2(I_2)} V_I_1 + \frac{I_2}{\beta_2 S f_2(I_2)} V_I_2$$

$$+ \hat{V}_{I_2} + \frac{\beta_1}{\beta_2 f_2(I_2)} \int_{\tau_1}^{0} I_1(t + \theta) d\theta,$$

where

$$V_S = g \left( \frac{S}{S} \right), \quad V_I_2 = g \left( \frac{I_2}{I_2} \right), \quad \hat{V}_{I_2} = \int_{0}^{\tau_2} g \left( \frac{I_2(t - s)}{I_2} \right) ds.$$

The rest of the proof is similar to that of Theorem 4.2 is omitted here. □

Next, we analyze the global stability of the co-existence equilibrium $E^*$ under the assumption that $E^*$ exists.

**Theorem 4.4.** Assume that (3.4) holds so that $E^*$ exists. Then $E^*$ is globally attractive.

**Proof.** Assume that the endemic equilibrium $E^* = (S^*, I_1^*, I_2^*)$ exists. To determine its stability we construct the following Lyapunov functional

$$V = \frac{1}{\beta_1 f_1(I_1^*)} V_S + \frac{I_1^*}{\beta_1 S^* f_1(I_1^*)} V_I_1 + \frac{I_2^*}{\beta_1 S^* f_1(I_1^*)} V_I_2 + V_{I_1}^* + \frac{\beta_2 f_2(I_2^*)}{\beta_1 f_1(I_1^*)} V_{I_2}^*,$$

where $V_S, V_I_1, V_I_2, V_{I_1}^*$ and $V_{I_2}^*$ are defined as

$$V_S = g \left( \frac{S}{S^*} \right), \quad V_I_1 = g \left( \frac{I_1}{I_1^*} \right), \quad V_I_2 = g \left( \frac{I_2}{I_2^*} \right),$$

$$V_{I_1}^* = \int_{0}^{\tau_1} g \left( \frac{I_1(t - s)}{I_1^*} \right) ds, \quad V_{I_2}^* = \int_{0}^{\tau_2} g \left( \frac{I_2(t - s)}{I_2^*} \right) ds.$$

Obviously, $V$ is non-negative in $\Omega$ and attains zero at $E^*$. Following the approach as in Theorem 4.2 with the following modification on function $F$ as
\[ u = \frac{S}{S'}, \quad y_i = \frac{I_i}{I_i'}, \quad z_i = \frac{I_i}{I_i'}, \quad i = 1, 2, \]

and

\[ F_i(z_i) = \frac{f_i(I'_i z_i)}{f_i(I'_i)} = \frac{f_i(I_z)}{f_i(I'_z)}, \quad i = 1, 2, \]

we can find the derivative of \( V \) along the trajectories of (3.2) and obtain

\[
\dot{V} = -\frac{\mu}{\beta_1 SS' f_1(I'_1)} (S - S')^2 \\
+ \left( 1 - \frac{1}{u} - u F_1(z_1) + F_1(z_1) \right) \\
+ \frac{\beta_2 f_2(I'_2)}{\beta_1 f_1(I'_1)} \left( 1 - \frac{1}{x} - u F_2(z_2) + F_2(z_2) \right) \\
+ \left( u F_1(z_1) - y_1 - \frac{u F_1(z_1)}{y_1} + 1 \right) \\
+ \frac{\beta_2 f_2(I'_2)}{\beta_1 f_1(I'_1)} \left( u F_2(z_2) - y_2 - \frac{u F_2(z_2)}{y_2} + 1 \right) \\
+ (y_1 - z_1 + \ln z_1 - \ln y_1) \\
+ \frac{\beta_2 f_2(I'_2)}{\beta_1 f_1(I'_1)} (y_2 - z_2 + \ln z_2 - \ln y_2).
\]

Notice the first term is non-positive. Cancelling and rearranging the like terms, we have

\[
\dot{V} \leq \left( 2 - \frac{1}{u} + F_1(z_1) - \frac{u F_1(z_1)}{y_1} - z_1 + \ln(z_1) - \ln(y_1) \right) \\
+ \frac{\beta_2 f_2(I'_2)}{\beta_1 f_1(I'_1)} \left( 2 - \frac{1}{u} + F_2(z_2) - \frac{u F_2(z_2)}{y_2} - z_2 \right) \\
+ \ln z_2 - \ln y_2 \\
= \left[ \left( 1 - \frac{1}{u} + \frac{1}{u} \right) + \left( 1 - \frac{u F_1(z_1)}{y_1} + \ln \frac{u F_1(z_1)}{y_1} \right) \right. \\
\left. + (F_1(z_1) - z_1 + \ln z_1 - \ln F_1(z_1)) \right]
\]
From the properties of the function $g(u)$ and (4.3) that $\dot{V} \leq 0$ in $\Omega$ with equality holding only at $E^*$. Again by [9], every positive solution approaches $E^*$, that is, the co-endemic equilibrium $E^*$ is globally attractive, completing the proof.

We have established the global attractivity of $E^*$ whenever it exists. Although (3.4) gives explicit conditions for $E^*$ to exist (in terms of $S^*$), it is interesting, from the viewpoint of dynamical systems, to see how the existence is related to the stability of the two boundary equilibria $E_1$ and $E_2$. Theorem 3.1 gives a necessary condition for $E^*$ to exist, under which, both $E_1$ and $E_2$ exist. The following theorem shows $E^*$ can exist only when both $E_1$ and $E_2$ are unstable.

**Theorem 4.5.** Assume $\mathcal{R}_m > 1$ and let

$$\mathcal{R}_1 = \frac{\beta_1 \bar{S}}{\mu_1 + \gamma_1} \quad \text{and} \quad \mathcal{R}_2 = \frac{\beta_2 \bar{S}}{\mu_2 + \gamma_2}.$$ 

Then,

(i) $E_1$ is globally attractive if $\mathcal{R}_2 < 1$, and it is unstable if $\mathcal{R}_2 > 1$;
(ii) $E_2$ is globally attractive if $\mathcal{R}_1 < 1$, and it is unstable if $\mathcal{R}_1 > 1$.

**Proof.** The Jacobian matrix of (3.2) at $E_1$ is

$$J(E_1) = \begin{pmatrix}
-\mu - \frac{\beta_1 I_1}{1 + \alpha_1 I_1} & \frac{\beta_1 \bar{S}}{(1 + \alpha_1 I_1)^2} & -\beta_2 \bar{S} \\
\frac{\beta_1 I_1}{1 + \alpha_1 I_1} & \frac{\beta_1 \bar{S}}{(1 + \alpha_1 I_1)^2} - \left(\mu_1 + \gamma_1\right) & 0 \\
0 & 0 & \beta_2 \bar{S} - \left(\mu_2 + \gamma_2\right)
\end{pmatrix}.$$
It is easy to see that $J(E_1)$ has an eigenvalue

$$\lambda = \beta_2 \bar{S} - (\mu_2 + \gamma_2) = (\mu_2 + \gamma_2) \left( \frac{\beta_2 \bar{S}}{\mu_2 + \gamma_2} - 1 \right) = (\mu_2 + \gamma_2)(R_2 - 1).$$

Thus, if $R_2 > 1$, then $\lambda > 0$, and hence, $E_1$ is unstable, having a one dimensional unstable manifold pointing to the interior of $\Omega$. However, if $R_2 < 1$, then from the third line from the end in equation (4.4), we know that the Lyapunov functional $V$ in Theorem 4.2 still works, ensuring the global attractivity of $E_1$ and hence proving (i). By symmetry, (ii) also holds, and the proof is completed. □

The above theorem makes one conjecture that $R_m = \min \{ R_1, R_2 \} > 1$ (equivalent to instability of both $E_1$ and $E_2$) is indeed equivalent to (3.4), and hence, is necessary and sufficient for $E^*$ to exist. We confirm this conjecture below. Firstly, we rewrite (3.4) as

$$\frac{\mu_i + \gamma_i}{\beta_i} < S^*, \quad i = 1, 2.$$ 

Note that $\bar{S}$ and $\bar{S}$ can be rewritten as

$$\bar{S} = \frac{\mu_2 + \gamma_2 + \alpha_2 \Lambda}{\alpha_2 \mu + \beta_2}, \quad S = \frac{\mu_1 + \gamma_1 + \alpha_1 \Lambda}{\alpha_1 \mu + \beta_1}.$$

Thus, we can rewrite $S^*$ as

$$S^* = \frac{\alpha_1 \alpha_2 \Lambda + \alpha_1 (\mu_2 + \gamma_2) + \alpha_2 (\mu_1 + \gamma_1)}{\alpha_1 \alpha_2 + \beta_1 \alpha_2 + \beta_2 \alpha_1} = \frac{\alpha_1 (\alpha_2 \mu + \beta_2 + \gamma_2) + \alpha_2 (\mu_1 + \gamma_1)}{\alpha_1 (\alpha_2 \mu + \beta_2) + \beta_1 \alpha_2} = \bar{S} + (\mu_1 + \gamma_1) x = h(x),$$

where

$$x = \frac{\alpha_2}{\alpha_1 (\alpha_2 \mu + \beta_2)}.$$

Note that $h(0) = \bar{S}$, $h(\infty) = \frac{\mu_1 + \gamma_1}{\beta_1}$. It is easy to show that $h(x)$ is decreasing if $R_1 > 1$ and increasing if $R_1 < 1$. Therefore,

$$\frac{\mu_1 + \gamma_1}{\beta_1} = h(\infty) < h(x) = S^* < h(0) = \bar{S} \quad \text{when} \quad R_1 > 1;$$

$$\frac{\mu_1 + \gamma_1}{\beta_1} = h(\infty) > h(x) = S^* > h(0) = \bar{S} \quad \text{when} \quad R_1 < 1.$$
Similarly, or symmetrically, we can show that

\[
\frac{\mu_2 + \gamma_2}{\beta_2} < S^* < \bar{S} \quad \text{when} \quad \mathcal{R}_2 > 1;
\]

\[
\frac{\mu_2 + \gamma_2}{\beta_2} > S^* > \bar{S} \quad \text{when} \quad \mathcal{R}_2 < 1.
\]

The equivalence of (3.4) and \( \mathcal{R}_m > 1 \) follows from (4.7) and (4.9)–(4.10).

5 Numerical simulations
In this section, we simulate the model for some parameter values. The parameters are chosen so that it can illustrate some vital aspects of the model which have also been confirmed by analytical conclusions. In Figure 1, we see that both strains may co-persist when the contact rates are relatively high, but saturation levels are low. Keeping \( \alpha_1 = 0 \) for strain 1 makes strain 1 the dominant strain and causes strain 2 to become extinct (Figure 2). On the other hand, if we raise \( \alpha_1 \) to a significant level, then strain 1 dies out and strain 2 persists and this is shown in Figure 3. If both \( \alpha_1 \) and \( \alpha_2 \) are kept zero then the co-persistence equilibrium \( E^* \) does not exist and one of the strains must die out (as is shown in Figure 4; in this case strain 2 dies out). Finally, both strains can be made extinct with lower contact rates and higher saturation levels that can be seen in Figure 5.

\[
\begin{align*}
\text{FIGURE 1: Both strains are endemic. Parameter values are} & \quad \beta_1 = 0.0001, \quad \beta_2 = 0.00012, \quad \gamma_1 = 0.007, \quad \gamma_2 = 0.009, \quad \mu = 0.02, \quad \mu_1 = 0.02, \quad \mu_2 = 0.02, \\
& \quad \alpha_1 = 0.001, \quad \alpha_2 = 0.002, \quad \Lambda = 100, \quad \text{giving} \; \mathcal{R}_1 = 37.04, \; \mathcal{R}_2 = 34.29, \; \mathcal{R}_1 = 10.07, \; \mathcal{R}_2 = 6.49. 
\end{align*}
\]
FIGURE 2: Strain 1 is endemic and strain 2 goes to extinction. Parameter values are $\beta_1 = 0.0001$, $\beta_2 = 0.00012$, $\gamma_1 = 0.007$, $\gamma_2 = 0.015$, $\mu = 0.02$, $\mu_1 = 0.02$, $\mu_2 = 0.02$, $\alpha_1 = 0.00$, $\alpha_2 = 0.002$, and $\Lambda = 200$, giving $R_1 = 37.04$, $R_2 = 34.29$, $R_1 = 10.07$, $R_2 = 0.93$.

FIGURE 3: Strain 2 is endemic and strain 1 becomes extinct. Parameter values are $\beta_1 = 0.0001$, $\beta_2 = 0.00012$, $\gamma_1 = 0.045$, $\gamma_2 = 0.015$, $\mu = 0.02$, $\mu_1 = 0.02$, $\mu_2 = 0.02$, $\alpha_1 = 0.2$, $\alpha_2 = 0.002$, and $\Lambda = 200$, giving $R_1 = 15.38$, $R_2 = 34.29$, $R_1 = 0.93$, $R_2 = 32.76$. 
FIGURE 4: Abandoning saturating incidence in strain 1: $\alpha_1 = 0.0$. Parameter values are $\beta_1 = 0.001$, $\beta_2 = 0.0012$, $\gamma_1 = 0.07$, $\gamma_2 = 0.09$, $\mu = 0.02$, $\mu_1 = 0.03$, $\mu_2 = 0.04$, $\alpha_2 = 0.0$, and $\Lambda = 100$, giving $\mathcal{R}_1 = 50.0$, $\mathcal{R}_2 = 11.76$, $\mathcal{R}_1 = 4.25$, $\mathcal{R}_2 = 0.23$.

FIGURE 5: Disease free state. Parameter values are $\beta_1 = 0.00001$, $\beta_2 = 0.000012$, $\gamma_1 = 0.007$, $\gamma_2 = 0.009$, $\mu = 0.02$, $\mu_1 = 0.02$, $\mu_2 = 0.02$, $\alpha_1 = 0.2$, $\alpha_2 = 0.1$, and $\Lambda = 50$, giving $\mathcal{R}_1 = 0.25$, $\mathcal{R}_2 = 0.06$, $\mathcal{R}_1 = 0.27$, $\mathcal{R}_2 = 0.06$. 
6 Conclusion and discussion  Adopting the assumption in [7] that the population of infectious vectors at time $t$ is proportional to the population of the infectious hosts, and making use of the saturating incidence rate functions, instead of mass action or standard incidence functions, we we have formulated a mathematical model to describe the dynamics of a vector-borne disease with two-strains and with latency delays. For this model, the global dynamics is completely determined by selecting suitable Lyapunov functionals. Roughly speaking, the analytical results obtained shows that the model supports equilibrium dynamics, in the sense one of the equilibria is globally attractive. More precisely, we have shown that if the basic reproduction number $R_0$ is less than one, the disease dies out from the population; however, if $R_0 > 1$, then the disease will persist and one or both of the strains become endemic: depending the model parameter values, either one of the two boundary (one-strain) equilibrium or the co-persistence equilibrium is globally attractive. Unlike the single strain model [16], the condition $R_0 > 1$ does not ensure the existence of the co-persistence equilibrium. This equilibrium exists if both boundary equilibria exist and are unstable (featured by $R_m > 1$). The persistence of a strain not only depends on the respective reproduction number but also depends on the combined parameter $R_i$, $i = 1, 2$. That is why a strain may die out even though the strain specific reproduction number is larger than one.

The impact of the saturation levels, characterized by $\alpha_i$ ($i = 1, 2$), can be easily seen from the dependence of $R_i$ ($i = 1, 2$) on $\alpha_i$ ($i = 1, 2$). For instance, $R_1$ increases in $\alpha_2$ if $\Lambda > \mu(\mu_2 + \gamma_2)$ and decreases if $\Lambda < \mu(\mu_2 + \gamma_2)$. A higher saturation level for strain 2, $\alpha_2$, may lead strain 1 to become endemic by raising $R_1$, if $\Lambda > \mu(\mu_2 + \gamma_2)$. However, this saturation level of strain 2 may also cause strain 1 to be extinct if $\Lambda < \mu(\mu_2 + \gamma_2)$. That is, if the inflow ($\Lambda$) is sufficiently large, then the saturation level for strain 2 has a positive impact on the infection of strain 1, but if the inflow is not large enough then this level has a negative impact on the infection of strain 1. In analogy to the impact of $\alpha_2$ on strain 1, the saturation level of strain 1 ($\alpha_1$) also has similar effects on the infection of strain 2. If both $\alpha_1$ and $\alpha_2$ become zero then the co-persistence equilibrium does not exists and one or both of the strains must die out. In such a case, competition exclusion is generic when $R_0 > 1$. Thus, the adoption of saturated incidence rate functions does lead to an essential difference in disease dynamics.

The existence of equilibria and the value of reproduction numbers do not depend explicitly on the latency delays. However, the proportional constant $\beta$ in (1.1) depends, in general, on the latency delay $\tau$, hence, do does the combined parameter $\beta$. Typically, $\beta$ and $\beta$ are decreasing in $\tau$. Moving to the two strain model (2.1), the parameters $\beta_1$ and $\beta_2$ should be decreasing in $\tau$. Therefore, the latency $\tau$ affects the calculated parameters $R_i$ and $R_i$ ($i = 1, 2$) in such an implicit way.
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A TWO-STRAIN DISEASE MODEL

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