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On a bi-virus epidemic model with partial and waning immunity ⋆

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Abstract: We propose a deterministic compartmental model to study the impact of partial and waning immunity on the spread of two competitive epidemic diseases, hereafter termed viruses. Building on a standard bi-virus SIS model, we introduce additional compartments to account for individuals who recovered from each virus, and tunable parameters to capture the level of virus-specific and cross protection acquired after recovery from a specific virus, and the rate at which such immunity could wane. We formalise the model as a system of nonlinear ordinary differential equations, which is amenable to analytical treatment, and we focus our analysis on two specialisations of the model. First, in the absence of waning immunity, we establish a global convergence result showing that, above the epidemic threshold, only the "fittest" virus becomes endemic. Second, in the absence of cross-immunity, we demonstrate instead that long-lasting co-existence of the two viruses may emerge, depending on the model parameters.

Keywords: Compartmental model; Competitive epidemics; Nonlinear dynamics; Stability

1. INTRODUCTION

The development and analysis of mathematical models has allowed the systems and control community to gain analytical insight into the spreading of epidemic diseases (Mei et al., 2017; Paré et al., 2020; Zino and Cao, 2021). While most of the models deal with a single disease (referred to as a virus) spreading in the population, in the last decade, the study of competitive viruses in a population has become increasingly popular and relevant (Castillo-Chavez et al., 1999; Prakash et al., 2012; Darabi Sahneh and Scoglio, 2014; Liu et al., 2019). These models have enabled the study of the spread of viruses with multiple strains (e.g. coronaviruses) or the simultaneous spread of mutually exclusive viruses, such as orthopoxviruses, including smallpox and monkeypox (Townsend et al., 2013).

The majority of the literature on multi-virus models focuses on two simple compartmental paradigms: the susceptible–infected–susceptible (SIS) (Prakash et al., 2012; Ye and Anderson, 2023) and susceptible–infected–recovered (SIR) models (Zhang et al., 2022), which rely on the simplifying assumption that, after recovery from either of the two viruses, individuals become either immediately susceptible again to both diseases and permanently immune, respectively. However, for most real-world epidemic diseases, recovery may yield *partial immunity*, which may further *wane over time*, as is observed for COVID-19 (Goldberg et al., 2022). Moreover, recovery from a disease may provide cross immunity against other strains or similar viruses, such as for orthopoxviruses (Townsend et al., 2013). Despite their importance, these aspects have been typically investigated via numerical simulations (Poletto et al., 2015; Burbano Lombana et al., 2022), and instead have been neglected in analytically-tractable multi-virus models, limiting their real-world applicability.

To fill in this gap, we propose a novel bi-virus compartmental model. Our model, built upon the standard bi-virus SIS model (Ye and Anderson, 2023), incorporates two additional compartments to account for individuals who have recovered from the two viruses. We formalise the model as a system of ordinary differential equations (ODEs), which capture the evolution of the fraction of population belonging to each compartment. The system is regulated by a set of tunable parameters, which model several key real-world features of immunity. In particular, we assume that immunity may have a partial effect, that is, a recovered individual has a decreased but nonzero probability to be again infected with the virus. In this regard, we also differentiate between *virus-specific immunity* and *cross immunity*, i.e. the different levels of protections that are gained due to infection with the same virus or with a different competing virus, respectively. Finally, we account for *waning immunity*, whereby the (partial) protection gained after recovery vanishes with an exponential rate.

Besides formalising the model and ensuring that it is well-defined, our main contribution is the analysis of two specialisations, which allow us to shed light into some key aspects of immunity. First, we consider a scenario in which immunity after recovery does not wane, but provides only partial protection against contagion. We

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analytically characterise the asymptotic behaviour above the epidemic threshold, proving almost global convergence to an endemic equilibrium in which only the "fittest" virus is present. Second, we consider a scenario in which recovery from a virus does not provide cross immunity. Interestingly, the analysis of this system shows that, depending on the infectiousness of the two viruses and on the rate at which immunity wanes, long-lasting co-existence of the two viruses can emerge. Such a co-existence, which is observed in the real life and reported in the clinical literature (Balmer and Tanner, 2011), cannot be predicted with the simpler bi-virus SIS model in well-mixed populations (Prakash et al., 2012). Thus, our model provides a powerful tool to gain analytical insight into the impact of immunity and its features on competitive epidemic disease.

2. BI-VIRUS SIRIS MODEL

We propose a novel deterministic compartmental model, termed the *bi-virus SIRIS model*, to describe two competitive viruses spreading in a well-mixed population of unit mass. Specifically, we extend a bi-virus SIS model to account for immunity after recovery and incorporate the related features discussed in the Introduction.

Before presenting our model, we briefly recall the standard bi-virus SIS model (Prakash et al., 2012). In this model, it is assumed that the unit-mass population is split into three different compartments: susceptible (S), infected with virus 1 (I_1), and with virus 2 (I_2). The two viruses are assumed to be competitive, so that co-infection is not possible. The fraction of the population that is susceptible at (continuous) time $t \geq 0$ is denoted by $w(t) \in [0,1]$. For $i \in \{1,2\}$, we let $x_i(t) \in [0,1]$ be the fraction of the population infected with virus i. Since $w(t) = 1 - x_1(t) - x_2(t)$, the dynamics of the bi-virus SIS model can be captured by the following planar system of two ODEs:

$$\dot{x}_1 = -\mu_1 x_1 + \lambda_1(1 - x_1 - x_2)x_1 \tag{1a}$$
$$\dot{x}_2 = -\mu_2 x_2 + \lambda_2(1 - x_1 - x_2)x_2, \tag{1b}$$

where the first term in each equation represents infected individuals who recover from virus i with rate $\mu_i > 0$, becoming again susceptible to both viruses, and the second term captures new infections, which occur with contagion rate $\lambda_i > 0$ when susceptible individuals (the fraction being $1 - x_1 - x_2$) come into contact with individuals infected with virus i (the fraction being x_i). In (Prakash et al., 2012), (1) is analysed, and the main results can be summarised in the following survival-of-the-fittest result.

Proposition 1. If $\lambda_i/\mu_i < 1$ for both viruses $i \in \{1,2\}$, then (1) converges to the disease-free equilibrium $(\bar{x}_1, \bar{x}_2) = \mathbf{0}$. Otherwise, it converges to a unique endemic equilibrium $(\bar{x}_1, \bar{x}_2)$, where $\bar{x}_i > 0$ and $\bar{x}_j = 0$ for $i, j \in \{1,2\}$, $i \neq j$, satisfying $\lambda_i/\mu_i > \lambda_j/\mu_j$.

To capture extra features arising from immunity, we propose to include two additional compartments to the model, accounting for individuals who recovered from virus 1 and 2 (R_1 and R_2, respectively). Consequently, two variables are added to the system, $y_1(t) \in [0,1]$ and $y_2(t) \in [0,1]$, accounting for the fraction of the population recovered from virus 1 and 2, respectively, and thus being (partially) immune. Noting that $w(t) = 1 - x_1(t) - x_2(t) - y_1(t) - y_2(t)$, we can reduce the state space of the system to the four

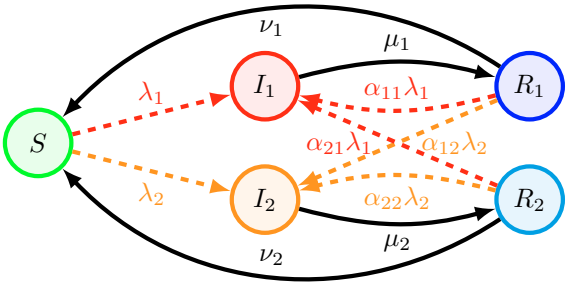


Fig. 1. Schematic of the bi-virus SIRIS model. Black solid arrows denote spontaneous transitions; red (orange) dashed arrows are transitions driven by interactions with individuals infected with virus 1 (2).

dimensional vector $(x_i(t), x_j(t), y_i(t), y_j(t)) \in \mathcal{D}$, where $\mathcal{D} := \{(x_i, x_j, y_i, y_j) \in [0,1]^4 : x_1 + x_2 + y_1 + y_2 \leq 1\}$. We propose the following disease spreading dynamics:

$$\begin{aligned}\dot{x}_1 =& -\mu_1 x_1 + \lambda_1[1 - x_1 - x_2 - (1-\alpha_{11})y_1 \\ & - (1-\alpha_{21})y_2]x_1 \end{aligned} \tag{2a}$$
$$\begin{aligned}\dot{x}_2 =& -\mu_2 x_2 + \lambda_2[1 - x_1 - x_2 - (1-\alpha_{22})y_2 \\ & - (1-\alpha_{12})y_1]x_2 \end{aligned} \tag{2b}$$
$$\dot{y}_1 = \mu_1 x_1 - \nu_1 y_1 - \alpha_{11}\lambda_1 y_1 x_1 - \alpha_{12}\lambda_2 y_1 x_2 \tag{2c}$$
$$\dot{y}_2 = \mu_2 x_2 - \nu_2 y_2 - \alpha_{21}\lambda_1 y_2 x_1 - \alpha_{22}\lambda_2 y_2 x_2. \tag{2d}$$

The first term in the four equations accounts for individuals who recover from virus i and, similar to an SIR model, transition from the I_i compartment to R_i, acquiring (partial) immunity against the two viruses. However, immunity due to recovery from infection with virus i wanes at a rate $\nu_i \in \mathbb{R}_+$, and the individual becomes susceptible again, as captured by the second term in (2c) and (2d). For each virus $i \in \{1,2\}$, we introduce two parameters $\alpha_{ii} \in [0,1]$ and $\alpha_{ij} \in [0,1]$ with $j \neq i$, which captures virus-specific and cross immunity due to recovery from virus i, respectively. Such parameters affect the contagion rate of the corresponding viruses as multiplicative factors: smaller values of such parameters denote higher levels of immunity. Specifically, while susceptible individuals become infected with virus i after an interaction with an infected individual with contagion rate $\lambda_i \in \mathbb{R}_+$ as in (1), those that are (partially) immune due to virus-specific immunity (y_i) and cross immunity (y_j) reduce their contagion rate to $\alpha_{ii}\lambda_i$ and $\alpha_{ji}\lambda_i$, respectively. The model is illustrated in Fig. 1. We introduce some terminology.

Definition 2. We define the *healthy manifold* as $\mathcal{H} := \{(x_1, x_2, y_1, y_2) \in \mathcal{D} : x_1 = x_2 = 0\}$, and the *disease-free equilibrium (DFE)* as $(\bar{x}_1, \bar{x}_2, \bar{y}_1, \bar{y}_2) = \mathbf{0}$. Given a fixed point $(\bar{x}_i, \bar{x}_j, \bar{y}_i, \bar{y}_j) \in \mathcal{D}$ of (2), we say that it is an *endemic equilibrium* if $\bar{x}_1 + \bar{x}_2 > 0$. Specifically, we classify an endemic equilibrium as a *boundary equilibrium* if $\bar{x}_1 > 0$ and $\bar{x}_2 = 0$ or $\bar{x}_1 = 0$ and $\bar{x}_2 > 0$, and a *co-existence equilibrium* if both $\bar{x}_1 > 0$ and $\bar{x}_2 > 0$.

We show that the model is well-defined on its domain , and we prove some basic properties of the healthy manifold.

Proposition 3. The domain $\mathcal{D}$ and the healthy manifold $\mathcal{H}$ are positively invariant for (2). Moreover, if $(x_1(0), x_2(0), y_1(0), y_2(0)) \in \mathcal{H}$ and $\nu_1, \nu_2 > 0$, then $\lim_{t\to\infty}(x_1(t), x_2(t), y_1(t), y_2(t)) = \mathbf{0}$.

Proof. The domain $\mathcal{D}$ is compact and convex and the vector field in (2) is Lipschitz-continuous. Hence, Nagumo's

Theorem can be applied (Blanchini, 1999). We are left with checking the direction of the vector field at the boundary of the domain. We observe that, if $x_i = 0$, then $\dot{x}_i = 0$, and if $y_i = 0$ then $\dot{y}_i = \mu_i x_i \geq 0$. Finally, for $x_1+x_2+y_1+y_2 = 1$, we observe that the field either is zero or it points towards the interior, as $\dot{x}_1 + \dot{x}_2 + \dot{y}_1 + \dot{y}_2 = -\nu_1 y_1 - \nu_2 y_2 \leq 0$. This implies that the domain $\mathcal{D}$ is invariant. Positive invariance of the healthy manifold can be easily checked, since $\dot{x}_1 = \dot{x}_2 = 0$ for all $(x_1, x_2, y_1, y_2) \in \mathcal{H}$. Finally, given $(x_1(0), x_2(0), y_1(0), y_2(0)) = (0, 0, y_1^0, y_2^0) \in \mathcal{H}$, (2) can be solved analytically, yielding $(x_1(t), x_2(t), y_1(t), y_2(t)) = (0, 0, y_1^0 e^{-\nu_1 t}, y_2^0 e^{-\nu_2 t}) \to \mathbf{0}$. □

Proposition 4. If $\nu_1 = \nu_2 = 0$, then either (2) converges to $\mathcal{H}$ or $\lim_{t\to\infty} w(t) = 0$.

Proof. Let us assume that the system does not converge to $\mathcal{H}$. Hence, since (2) is Lipschitz-continuous, there exist constants $\varepsilon > 0$ and $\eta > 0$ such that $x_1(t)+x_2(t) \geq \varepsilon$ for all $t \in \mathcal{T}$, which is the union of infinitely many nonvanishing intervals of length at least η. Observe from (2) that $\dot{w}(t) = -\lambda_1 x_1 w - \lambda_2 x_2 w$ for all $t \geq 0$, thus $\dot{w}(t) \leq -\min\{\lambda_1, \lambda_2\}\varepsilon w$ for all $t \in \mathcal{T}$, and, in general, $\dot{w}(t) \leq 0$, for all $t \notin T$. Hence, since $\mathcal{T}$ has infinite measure, we have that $\lim_{t\to\infty} w(t) \leq \exp\{-\min\{\lambda_1, \lambda_2\}\varepsilon|\mathcal{T}| = 0$. □

The general model in (2) is characterised by ten parameters and four independent nonlinear ODEs. Such a complexity makes its general tractability a nontrivial problem. In the rest of the paper, we focus on two specific implementations of the model that allow us to shed light on some specific aspects of the bi-virus epidemic process. First, we discuss a formulation of the model that allows one to understand how partial immunity impacts the course of the epidemic. Second, we unveil the role of waning immunity on the long-term co-existence of the two viruses.

3. PARTIAL IMMUNITY

We study partial immunity by making two simplifications: we neglect waning immunity and assume that, after recovery from either of the two viruses, an individual acquires the same level of partial immunity against both viruses.

Assumption 5. Let $\nu_1 = \nu_2 = 0$, $\alpha_{21} = \alpha_{11}$, $\alpha_{12} = \alpha_{22}$.

Intuitively, since virus-specific immunity and cross immunity are assumed to have the same effect, there is no need to define two distinct variables for recovered compartments R_1 and R_2. Indeed, if we define $y(t) := y_1(t)+y_2(t)$, under Assumption 5, the dynamics in (2) reduces to

$$\dot{x}_1 = -\mu_1 x_1 + \lambda_1[1 - x_1 - x_2 - (1 - \alpha_{11})y]x_1 \quad (3a)$$
$$\dot{x}_2 = -\mu_2 x_2 + \lambda_2[1 - x_1 - x_2 - (1 - \alpha_{22})y]x_2 \quad (3b)$$
$$\dot{y} = \mu_1 x_1 + \mu_2 x_2 - \alpha_{11}\lambda_1 x_1 y - \alpha_{22}\lambda_2 x_2 y, \quad (3c)$$

with $w(t) = 1 - x_1(t) - x_2(t) - y(t)$. This model is fully determined by six parameters: λ_1, λ_2, μ_1, μ_2, α_{11}, and α_{22}. We will show, using Proposition 4, that all endemic equilibria of (3) coincide with those of a bi-virus SIS model, with a suitable change of parameters. Then, we fully characterise the asymptotic behaviour of (3).

Lemma 6. Under Assumption 5, a state $(\bar{x}_1, \bar{x}_2, \bar{y})$ with $\bar{x}_1 + \bar{x}_2 > 0$ is an equilibrium of (3) if and only if $\bar{x}_1 + \bar{x}_2 + \bar{y} = 1$ and $(\bar{x}_1, \bar{x}_2)$ is an equilibrium of (1) with recovery rates μ_1 and μ_2, and infection rates $\alpha_{11}\lambda_1$ and $\alpha_{22}\lambda_2$.

Proof. Proposition 4 applies under Assumption 5, and with the definition of $y = y_1 + y_2$, it is evident from the proposition that for (3), there holds $w(t) \to 0$, with $\bar{x}_1 + \bar{x}_2 + \bar{y} = 1$ at the equilibrium. At an equilibrium point of (3), this equality and the first two equations yield

$$-\mu_1\bar{x}_1 + \alpha_{11}\lambda_1(1 - \bar{x}_1 - \bar{x}_2)\bar{x}_1 = 0 \quad (4a)$$
$$-\mu_2\bar{x}_2 + \alpha_{22}\lambda_2(1 - \bar{x}_1 - \bar{x}_2)\bar{x}_2 = 0, \quad (4b)$$

These are precisely the equilibrium conditions for the bi-virus SIS model in (1) with recovery rates μ_1 and μ_2, and infection rates $\alpha_{11}\lambda_1$ and $\alpha_{22}\lambda_2$. The converse is easily shown. □

Note that the above lemma makes no claim about the relation between the stability properties of equilibria of (3) and the corresponding ones for the bi-virus SIS model. The equation sets are even of different dimension.

However, a straightforward consequence of Lemma 6 yields a relation, in the first instance for local stability. For this result, we choose to exclude the nongeneric case $\alpha_{11}\lambda_1/\mu_1 = \alpha_{22}\lambda_2/\mu_2$, which gives rise to a continuum of co-existence equilibria. The local stability result using further argument extends to a global result.

Theorem 7. The bi-virus SIRIS model under Assumption 5, with $\alpha_{11}\lambda_1/\mu_1 \neq \alpha_{22}\lambda_2/\mu_2$, admits at most two endemic equilibria, i.e. the boundary equilibria:

$$\left(\frac{\alpha_{11}\lambda_1 - \mu_1}{\alpha_{11}\lambda_1}, 0, \frac{\mu_1}{\alpha_{11}\lambda_1}\right), \left(0, \frac{\alpha_{22}\lambda_2 - \mu_2}{\alpha_{22}\lambda_2}, \frac{\mu_2}{\alpha_{22}\lambda_2}\right), \quad (5)$$

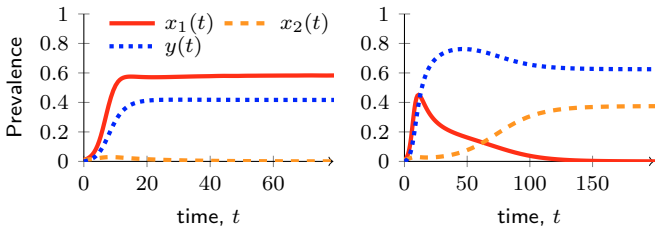
which exist iff $\alpha_{11}\lambda_1 > \mu_1$ and $\alpha_{22}\lambda_2 > \mu_2$, respectively. The first equilibrium is locally asymptotically stable iff $\lambda_1\alpha_{11}/\mu_1 > \lambda_2\alpha_{22}/\mu_2$; the second equilibrium is locally asymptotically stable iff $\lambda_1\alpha_{11}/\mu_1 < \lambda_2\alpha_{22}/\mu_2$. In each case, the locally asymptotically stable boundary equilibrium is globally stable for all initial conditions in the interior of $\{(x_1, x_2, y) \in [0, 1]^3 : x_1 + x_2 + y \leq 1\}$.

Before proving the theorem, we remark that the stability conditions are actually the same as those applying to the associated bi-virus SIS model (see Proposition 1 and Lemma 6). Also, generically precisely one of the boundary equilibria is stable, so that in the whole region of interest, there is only one attractive equilibrium. Unsurprisingly, global or almost global stability is then to be expected, as asserted in the latter part of the theorem statement.

Proof. That the only nonzero solutions of (3) are those given is easy to verify using Lemma 6 and the fact that the only boundary solutions of (4) are $(\frac{\alpha_{11}\lambda_1-\mu_1}{\alpha_{11}\lambda_1}, 0)$ and $(0, \frac{\alpha_{22}\lambda_2-\mu_2}{\alpha_{22}\lambda_2})$. The lemma also ensures that $\bar{y} = 1-\bar{x}_1-\bar{x}_2$, as evidenced in the coordinates of the two equilibria.

We focus initially on the local stability properties of the first endemic equilibrium point. The eigenvalues of the Jacobian matrix of (3) evaluated at that equilibrium are $-\lambda_1(1 - \frac{\mu_1}{\alpha_{11}\lambda_1})$, $-\alpha_{11}\lambda_1(1 - \frac{\mu_1}{\alpha_{11}\lambda_1})$, and $-\mu_2 + \alpha_{22}\lambda_2(\frac{\mu_1}{\alpha_{11}\lambda_1})$. The first two are always negative under the existence conditions of the equilibrium, i.e. $\lambda_{11}\alpha_{11} > \mu_1$. The third one is negative iff $\lambda_1\alpha_{11}/\mu_1 > \lambda_2\alpha_{22}/\mu_2$. The same argument applied to the second equilibrium yields the second condition.

Now to establish the global stability claim, we first observe a further connection between the equations in (3) and



(a) $\alpha_{11} = 0.6$ (b) $\alpha_{11} = 0.3$

Fig. 2. Simulations of the model in Section 3 with $\lambda_1 = 0.8$, $\lambda_2 = 0.4$, $\mu_1 = \mu_2 = 0.2$, and $\alpha_{22} = 0.8$.

the associated bi-virus SIS model. The former set using $w(t) = 1 - x_1(t) - x_2(t) - y(t)$ can be rewritten as

$$\dot{x}_1 = -\mu_1 x_1 + \alpha_{11}\lambda_1(1 - x_1 - x_2)x_1 + w\lambda_1 x_1 \quad (6a)$$
$$\dot{x}_2 = -\mu_2 x_2 + \alpha_{22}\lambda_2(1 - x_1 - x_2)x_2 + w\lambda_2 x_2 \quad (6b)$$
$$\dot{w} = -(\lambda_1 x_1 + \lambda_2 x_2)w. \quad (6c)$$

Stability properties are the same as those of (3), *mutatis mutandis*. The first two equations coincide with those of (1) with infection rates re-scaled by the parameters α_{11} and α_{22}, aside from the additive terms $w\lambda_i x_i$. As shown in Proposition 4, these additive terms decay asymptotically to 0, provided that the trajectories do not tend to $\mathcal{H}$. Convergence to $\mathcal{H}$ is precluded however, by the requirement stated in the theorem hypothesis that when a boundary equilibrium exists, $\alpha_{ii}\lambda_i > \mu_i$ for one or both of $i \in \{1, 2\}$; this means that the corresponding $\dot{x}_i$ in (6) are positive when $x_1 + x_2$ is sufficiently small, i.e. is close enough to $\mathcal{H}$. It follows that for t exceeding some sufficiently large T, the additional additive terms will be arbitrarily small. Intuitively, one would then expect the x_1 and x_2 components of trajectories of (3) or of (6) and trajectories of the standard bi-virus SIS equations to look very similar, at least after a transient phase. This idea can be rigorously formulated. As established in (Krasovskii, 1963, see Theorem 20.1) and (Sastry, 2013, see problem 5.16) for the rigorous formalisation, the x_1 and x_2 components of the trajectories of (6) must mimic the limiting behaviour involving convergence to an equilibrium of the bi-virus SIS equations. Any initial condition in the region of attraction of an exponentially stable equilibrium of the latter will enjoy the same property for (6) —here being crucial that $\mathcal{D}$ is bounded. The behaviour of the bi-virus SIS equations for the single population case has been analysed in Prakash et al. (2012): given the inequality condition in the hypothesis, a winner-take-all or survival-of-the fittest convergence property follows (the local stability properties are in other words global for initial conditions in the interior of $\mathcal{D}$); chaos or limit cycles cannot occur. The fact that the standard bi-virus SIS equations give rise to trajectories converging exponentially fast to the equilibrium is crucial in assuring the same property for the equations (3). □

Theorem 7 does not deal with stability of the DFE. We omit treatment entirely, since this case is not straightforward. The reason is that the DFE is not an isolated equilibrium but lies on a continuum of equilibria for which $\bar{x}_1 = 0, \bar{x}_2 = 0$ but $\bar{y}$ is free, as inspection of (3) reveals, and the Jacobian at the DFE has a zero eigenvalue.

The simulations in Fig. 2 illustrate our findings. We consider two different scenarios, both with $\alpha_{ii}\lambda_i > \mu_i$ for $i \in \{1, 2\}$, i.e. in which both the boundary endemic equilibria exist. In Fig. 2a, $\alpha_{11}\lambda_1/\mu_1 > \alpha_{22}\lambda_2/\mu_2$. Consistent with our analytical predictions, we observe convergence to the boundary equilibrium where only virus 1 is present. The opposite behaviour is recorded in Fig. 2b, where we have decreased α_{11} so that $\alpha_{11}\lambda_1/\mu_1 < \alpha_{22}\lambda_2/\mu_2$. Finally, we observe that the epidemic spreading may display a non-trivial transient behaviour: in Fig. 2b we observe an initial surge in virus 1, even though it is eventually eradicated and virus 2 slowly emerges as dominant. This observation suggests that further studies should be performed toward shedding light into the transient behaviour of this model.

Partial immunity impacts the behaviour of the system by changing the epidemic threshold and potentially adding further features to its transient behaviour. Moreover, the notion of "fittest" virus also changes, as it now additionally depends on the parameters α_{ii}, as highlighted in Theorem 7. However, it does not affect the long-term emergent behaviour of the system: if any of the two viruses is above the threshold, then only the "fittest" one will survive.

4. WANING VIRUS-SPECIFIC IMMUNITY

We study a specialisation of the model to understand the effect of waning and virus-specific immunity by assuming that after recovery from any of the two viruses, an individual acquires full waning immunity only against that virus, while no immunity is acquired against the other virus.

Assumption 8. Let $\alpha_{11} = \alpha_{22} = 0$, $\alpha_{12} = \alpha_{21} = 1$, $\nu_1 > 0$, and $\nu_2 > 0$.

Under Assumption 8, (2) reduces to

$$\dot{x}_1 = -\mu_1 x_1 + \lambda_1(1 - x_1 - x_2 - y_1)x_1 \quad (7a)$$
$$\dot{x}_2 = -\mu_2 x_2 + \lambda_2(1 - x_1 - x_2 - y_2)x_2 \quad (7b)$$
$$\dot{y}_1 = \mu_1 x_1 - \nu_1 y_1 - \lambda_2 x_2 y_1 \quad (7c)$$
$$\dot{y}_2 = \mu_2 x_2 - \nu_2 y_2 - \lambda_1 x_1 y_2, \quad (7d)$$

which is fully determined by six positive scalar parameters: λ_1, λ_2, μ_1, μ_2, ν_1, and ν_2.

We now present the main result of this section, which establishes necessary and sufficient conditions for global exponential stability of the DFE for (7), and necessary and sufficient conditions for the existence and local exponential stability (and instability) of the boundary equilibria.

Theorem 9. Consider the bi-virus SIRIS model under Assumption 8 with $\lambda_i > 0$ and $\mu_i > 0$, for $i \in \{1, 2\}$. Then, the following hold true:

(1) The DFE is globally exponentially stable iff $\lambda_i/\mu_i < 1$ for both $i \in \{1, 2\}$.
(2) For $i \in \{1, 2\}$, a boundary equilibrium, $\mathbf{b_i}$, exists iff $\lambda_i/\mu_i > 1$. Moreover, $\mathbf{b_i}$ has the form

$$\bar{x}_i = \frac{\nu_i(\lambda_i - \mu_i)}{\lambda_i(\mu_i + \nu_i)}, \quad \bar{y}_i = \frac{\mu_i(\lambda_i - \mu_i)}{\lambda_i(\mu_i + \nu_i)}, \quad \bar{x}_j = \bar{y}_j = 0, \quad (8)$$

and is the unique boundary equilibrium with $\bar{x}_i > 0$.
(3) If a boundary equilibrium exists, then the DFE is unstable.
(4) For $i \in \{1, 2\}$, the equilibrium $\mathbf{b_i}$ is locally exponentially stable iff

$$\frac{\lambda_i/\mu_i}{\lambda_j/\mu_j} > \frac{\lambda_i + \nu_i}{\mu_i + \nu_i}, \quad j \neq i. \quad (9)$$

If the inequality sign is reversed, then $\mathbf{b_i}$ is unstable.

Proof. *(1)* First, we prove local stability. We compute the Jacobian of (7) evaluated at the origin, which reads

$$J(0,0,0,0) = \begin{bmatrix} -\mu_1+\lambda_1 & 0 & 0 & 0 \\ 0 & -\mu_2+\lambda_2 & 0 & 0 \\ \mu_1 & 0 & -\nu_1 & 0 \\ 0 & \mu_2 & 0 & -\nu_2 \end{bmatrix}. \quad (10)$$

Under Assumption 8, it follows that the DFE is locally exponentially stable iff $\lambda_i/\mu_i < 1$ for all $i \in \{1,2\}$. Global exponential stability is obtained by observing that, under these conditions, for $i \in \{1,2\}$, we can bound $\dot{x}_i \leq (\lambda_i - \mu_i)x_i$, which guarantees that $x_i(t) \leq x_i(0)e^{-(\mu_i-\lambda_i)t} \to 0$ exponentially fast, for $i \in \{1,2\}$. Then, for $i,j \in \{1,2\}$ and $i \neq j$, there holds $\dot{y}_i = -\nu_i y_i + \mu_i x_i - \lambda_j x_j y_i = -\nu_i y_i + \omega_i(t)$, with $\omega_i(t)$ being an input signal that decays to zero exponentially fast. Evidently, $\lim_{t\to\infty} y_i(t) = 0$ exponentially fast for all $i \in \{1,2\}$, which yields the claim.

(2) Let $(\bar{x}_1, \bar{x}_2, \bar{y}_1, \bar{y}_2)$ denote an equilibrium of (7), not including the DFE. Without loss of generality, we consider boundary equilibria for which $\bar{x}_1 = 0$, and hence $\bar{x}_2 > 0$. First of all, we notice that (7c) implies that an equilibrium with $\bar{x}_1 = 0$ must have $\bar{y}_1 = 0$. Based on (2b), it follows that if an endemic boundary equilibrium exists with $\bar{x}_2 > 0$ and $\bar{x}_1 = 0$, then necessarily $\mu_2 < \lambda_2$. In the following, we assume this condition holds as we look for an equilibrium point of (7) of the form $(0, \bar{x}_2, 0, \bar{y}_2)$, obtaining the following equilibrium equations:

$$0 = -\mu_2 x_2 + \lambda_2(1 - \bar{x}_2 - \bar{y}_2)\bar{x}_2, \quad (11a)$$
$$0 = \mu_2 x_2 - \nu_2 \bar{y}_2. \quad (11b)$$

The first and second equations yield $\mu_2 = \lambda_2(1 - \bar{x}_2 - \bar{y}_2)$ and $\bar{x}_2 = \bar{y}_2\nu_2/\mu_2$, respectively. Substituting the second expression into the first and rearranging for $\bar{y}_2$ yields $\bar{y}_2 = \frac{\mu_2(\lambda_2-\mu_2)}{\lambda_2(\mu_2+\nu_2)}$, which always belongs to the domain $(0,1)$, because $\nu_2 > 0$ and $\lambda_2 > \mu_2$ by hypothesis. It follows that $\bar{x}_2 = \frac{\nu_2(\lambda_2-\mu_2)}{\lambda_2(\mu_2+\nu_2)}$, and this also belongs to $(0,1)$. We therefore know that an equilibrium $\mathbf{b_2}$ exists, and it is the unique equilibrium on the boundary $\bar{x}_1 = 0$ with $\bar{x}_2 > 0$.

(3) Observe that $\mathbf{b_i}$ exists iff $\lambda_i/\mu_i > 1$. This implies that one of the eigenvalues of (10) is positive.

(4) Without loss of generality, we focus on $\mathbf{b_2}$. One can compute the Jacobian at the equilibrium $(0, \bar{x}_2, 0, \bar{y}_2)$, which has the block triangular form $J(0, \bar{x}_2, 0, \bar{y}_2) = \begin{bmatrix} J_{11} & 0 \\ J_{21} & J_{22} \end{bmatrix}$. The first block is in fact the scalar $J_{11} = -\mu_1 + \lambda_1(1-\bar{x}_2)$. The second, 3-dimensional block, is re-arranged using a permutation and the identity $-\mu_2 + \lambda_2(1 - \bar{x}_2 - \bar{y}_2) = 0$ from (11a) into $\begin{bmatrix} -\lambda_2\bar{x}_2 - \nu_1 & 0 & 0 \\ 0 & -\lambda_2\bar{x}_2 & -\lambda_2\bar{x}_2 \\ 0 & \mu_2 & -\nu_2 \end{bmatrix}$, which is Hurwitz. Thus, stability of $(0, \bar{x}_2, 0, \bar{y}_2)$ depends solely on the sign of $J_{11} = -\mu_1 + \lambda_1(1-\bar{x}_2)$. Observe that $1 - \bar{x}_2 = \frac{\mu_2}{\lambda_2}\frac{\lambda_2+\nu_2}{\mu_2+\nu_2}$. Thus, $J_{11} < 0$ iff $\frac{\lambda_2/\mu_2}{\lambda_1/\mu_1} > \frac{\lambda_2+\nu_2}{\mu_2+\nu_2}$. If the inequality is reversed, then the Jacobian has a positive eigenvalue, and thus $\mathbf{b_2}$ is unstable. By symmetry, it follows that there exists an endemic boundary equilibrium $\mathbf{b_1} = (\bar{x}_1, 0, \bar{y}_1, 0)$ iff $\lambda_1 > \mu_1$. This equilibrium is the unique boundary equilibrium with $\bar{x}_1 > 0$ and is locally exponentially stable iff $\frac{\lambda_1/\mu_1}{\lambda_2/\mu_2} > \frac{\lambda_1+\nu_1}{\mu_1+\nu_1}$, yielding (9). □

Remark 10. For boundary equilibrium $\mathbf{b_i}$ to be locally stable, three conditions should be satisfied. Similar to the standard bi-virus SIS model (Proposition 1), virus i should be above the epidemic threshold ($\lambda_i/\mu_i > 1$) and it should be the fittest virus ($\lambda_i/\mu_i > \lambda_j/\mu_j$). However, these two conditions are not sufficient in the presence of *waning immunity*. In fact, (9) posits a further condition, namely that virus i should be sufficiently fitter than virus j to be able to compensate for the protection gained after recovery. Interestingly, the left-hand side of such a condition is monotonically decreasing in ν_i. Thus, as $\nu_i \to 0$, condition (9) translates into requiring virus j to be below the epidemic threshold $\lambda_j < \mu_j$, whereas, as $\nu_i \to \infty$, (9) coincides with just requiring virus i to be the fittest virus. Hence, decreasing the rate at which virus-specific immunity is lost for virus i makes it harder for virus i to dominate and kill off virus j, while increasing such a rate can help a "fittest" virus i to dominate virus j.

We conclude with some observations that flow from Theorem 9 and help formalise the intuition in Remark 10.

Corollary 11. Adopt the hypotheses of Theorem 9, with $\mathbf{b_i}$ denoting the boundary equilibrium in (8). Then:

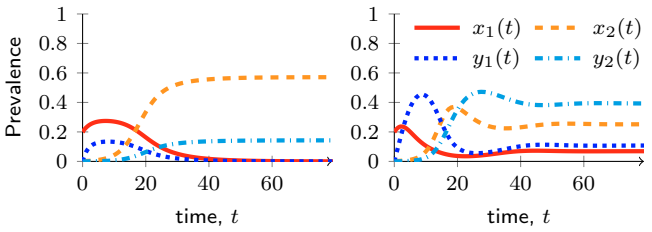
(1) If
$$\frac{\mu_2+\nu_2}{\lambda_2+\nu_2} < \frac{\lambda_1/\mu_1}{\lambda_2/\mu_2} < \frac{\lambda_1+\nu_1}{\mu_1+\nu_1}, \quad (12)$$
then $\mathbf{b_1}$ and $\mathbf{b_2}$ and the DFE are all unstable.
(2) It is not possible for both $\mathbf{b_1}$ and $\mathbf{b_2}$ to be simultaneously locally stable.
(3) If $\lambda_i/\mu_i > \lambda_j/\mu_j > 1$, for $i \neq j \in \{1,2\}$, then the DFE and $\mathbf{b_j}$ are both unstable. Under this condition, $\mathbf{b_i}$ is locally asymptotically stable if
$$\nu_i > \nu_i^* = \frac{\mu_i\lambda_i(\lambda_j-\mu_j)}{\lambda_i\mu_j - \lambda_j\mu_i}, \quad (13)$$
and unstable if $\nu_i < \nu_i^*$.

Proof. *(1)* The two inequalities in (12) are obtained by applying to the two equilibria the negations of the stability conditions of Item 4 in Theorem 9 for $i \in \{1,2\}$.

(2) The left-hand side of the condition in (9) satisfies $(\lambda_i+\nu_i)/(\mu_i+\nu_i) \in (1, \frac{\lambda_i}{\mu_i}]$. Hence, a necessary condition for $\mathbf{b_i}$ to be locally exponentially stable is that $\lambda_i/\mu_i > \lambda_j/\mu_j$, with $j \neq i$. As a consequence, it is not possible for both endemic boundary equilibria to be locally stable, since (9) cannot be satisfied simultaneously for $i \in \{1,2\}$.

(3) From the proof of Item 1, we obtain that $\lambda_i/\mu_i > \lambda_j/\mu_j$ implies $\mathbf{b_j}$ is unstable. Since $\lambda_j/\mu_j > 1$ by hypothesis, the DFE is also unstable according to Theorem 9. The inequality of (13) is obtained by rearranging (9) for ν_i. □

The simulations in Fig. 3 illustrate our findings. We consider a scenario in which $\lambda_2/\mu_2 > \lambda_1/\mu_1 > 1$. Hence, among the three equilibria of the DFE, $\mathbf{b_1}$, and $\mathbf{b_2}$, we have that either i) $\mathbf{b_2}$ is the only locally stable equilibrium, or ii) none of these three equilibria are stable. The condition in (13) yields the threshold $\nu_2^* = 0.3$. In Fig. 3a, $\nu_2 > \nu_2^*$ and thus $\mathbf{b_2}$ is locally stable. Simulations suggest that the equilibrium might be almost globally stable. In Fig. 3b, the rate ν_i is decreased below the threshold and $\mathbf{b_2}$ becomes unstable, as predicted by Theorem 9. In this scenario, the system converges to a *co-existence* equilibrium, where



(a) $\nu_1 = \nu_2 = 0.8$ (b) $\nu_1 = \nu_2 = 0.08$

Fig. 3. Simulations of the model in Section 4 with $\lambda_1 = 0.7$, $\lambda_1 = 0.7$, $\mu_1 = 0.4$, and $\mu_2 = 0.2$.

$\lim_{t\to\infty} x_1(t) > 0$ and $\lim_{t\to\infty} x_2(t) > 0$. Indeed, our simulations suggest that this co-existence equilibrium is asymptotically stable for all initial conditions in the interior of $\mathcal{D}$, and simulations with other parameter values giving rise to unstable $\mathbf{b_1}$ and $\mathbf{b_2}$ also appear to have a co-existence equilibrium that is globally asymptotically stable. We would like to point out that Theorem 9 and Corollary 11 do not deal with co-existence equilibria, but they do guarantee that, for a range of the modelling parameters, the system will not converge to the boundary for any interior initial condition in the interior. That is, neither virus dominates the other, nor go extinct. The partial immunity model studied Section 3 does not have co-existence equilibria except for a nongeneric parameter condition, and this is the same for the bi-virus SIS model that has been extensively studied Ye and Anderson (2023); Prakash et al. (2012). However, the above simulations suggest that co-existence equilibria can occur for the model treated in this section, even with generic parameters. A key focus of future work will be to identify parameter values for co-existence equilibria to occur and determine how many.

5. CONCLUSION

We proposed a compartmental model for the spread of two competitive viruses in a population. Our model, formalised as a system of ODEs, incorporates key real-world features of immunity gained after recovery, such as its partial and waning effect in preventing contagion, with a distinction between virus-specific and cross immunity. The analysis of two specialisations of our model allowed us to shed light on some aspects of the impact of immunity on the course of competing epidemic diseases. For the standard bi-virus SIS model, it is known that either both viruses are extinguished or only the "fittest" virus survives (Prakash et al., 2012). In our SIRIS model, such a scenario can still be observed in many cases, but immunity plays a role in determining which virus is the "fittest". Moreover, when immunity is slow to wane, a third outcome is possible, whereby both viruses remain present in the population.

These preliminary findings depict a wider range of possible behaviours that can be unlocked by the presence and the characteristics of immunity, paving the way for several lines of future research. First, while our results establish some local stability properties and global convergence results, an exhaustive analysis of the asymptotic behaviour of the two specialised models is still missing. In particular, for the waning virus-specific immunity model, the presence of a co-existence equilibrium, which is suggested by our simulations, still requires analytical verification. Afterwards, a treatment of the general model should be performed. Second, our simulations suggest that the epidemic prevalence may display nontrivial oscillations. Efforts should be placed toward analysing the transient behaviour of the system. Finally, we have studied our SIRIS model in the simple scenario of a fully-mixed population. Following the work done for the simpler bi-virus SIS and SIR models (Ye and Anderson, 2023; Zhang et al., 2022), we could embed our model on a network and study its impact on the dynamics of the two competitive viruses.

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