# Complex bifurcation structures in SIRWS and SIRWJS models of pertussis with asymmetric partition of immunity period

## Abstract of Ph.D. dissertation

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# 1 Introduction

Pertussis is a communicable disease caused by the bacterium Bordetella pertussis. The facts about this infection discussed in [1–5] reveal that it is a global health issue that deserves great attention. This infection confers non-permanent immunity upon recovery, and immune boosting is possible upon re-exposure. To examine the behavior of this infection, the SIRWS and SIRWJS models, which incorporate waning-boosting dynamics in their formulation are applied. We find in [6] a very general framework of waning-boosting dynamics.

A notable assumption in previous investigations of the SIRWS model [7–11] is that immune waning rates are the same for individuals who move from the recovered compartment to the waning compartment ($R \to W$) and for those who transition to the susceptible compartment ($W \to S$). We refer to this as the symmetric partition of the immune period. The same assumption is made in the study of the SIRWJS model [9].

In this work, we propose a new, potentially asymmetric partitioning of the total immune period and investigate its effect on the dynamics of the SIRWS and SIRWJS models. Our investigations of the resulting systems include analytical computation of the formulae for equilibria, stability analysis of equilibria, and numerical continuation techniques to construct bifurcation diagrams. We find stability switches and Hopf bifurcations from steady states forming multiple endemic bubbles, and saddle–node bifurcations of periodic orbits. As these models are used actively in the study of pertussis, we believe that our results contribute to a better understanding of the dynamics of the transmission of the disease.

The dissertation is based on the following two scientific papers of the author:

- Richmond Opoku-Sarkodie, Ferenc A. Bartha, Mónika Polner, Gergely Röst, Dynamics of an SIRWS model with waning of immunity and varying immune boosting period, Journal of Biological Dynamics, 16 (1) (2022) 596-618, [mtmt: 33047203].

- Richmond Opoku-Sarkodie, Ferenc A. Bartha, Mónika Polner, Gergely Röst, Bifurcation analysis of waning-boosting epidemiological models with repeat infections and varying immunity periods, Journal of Mathematics and Computers in Simulation, 281 (2024) 624-643, [mtmt: 34425145].

## 2 SIRWS model with waning of immunity and varying immune boosting period

The susceptible-infectious-recovered-waned-susceptible (SIRWS) model is an extension of the susceptible-infectious-recovered-susceptible (SIRS) model to include an intermediate compartment $W$ that holds individuals with waning immunity. This model separates the population of immune individuals into two categories according to the level of immunity.

Figure 1 shows the flow diagram of the SIRWS model that incorporates our asymmetric partition of the immunity period. In this transition diagram, recovered individuals in $R$ have full immunity, and may lose it by the chain of transitions $R \to W \to S$. Members of $W$ have varying levels of immunity and boosting of immunity is possible upon re-exposure. The frequency of that re-exposure is regulated by the boosting force $\nu$. Also, hosts that undergo boosting are not infectious, as in [6, 7, 10, 12], as opposed to [9].

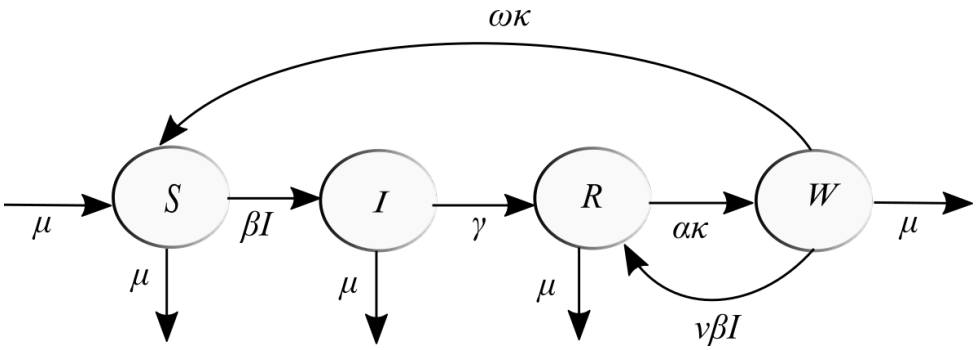



Figure 1: Flow diagram of the SIRWS system (1).

The dynamics of the SIRWS model is governed by the following system of ordinary differential equations

$$\frac{dS}{dt} = -\beta IS + \omega\kappa W + \mu(1-S), \tag{1a}$$
$$\frac{dI}{dt} = \beta IS - \gamma I - \mu I, \tag{1b}$$
$$\frac{dR}{dt} = \gamma I - \alpha\kappa R + \nu\beta IW - \mu R, \tag{1c}$$
$$\frac{dW}{dt} = \alpha\kappa R - \omega\kappa W - \nu\beta IW - \mu W, \tag{1d}$$

with parameters summarized in Table 1.

| | | |
|---|---|---|
| $\beta$ | $> 0$ | transmission rate |
| $\mu$ | $> 0$ | birth and death rate |
| $\gamma$ | $> 0$ | recovery rate from primary infection |
| $\kappa$ | $> 0$ | immune decay rate |
| $\alpha^{-1}$ | $\in (0,1)$ | relative size of the first immune protection period from $R \longrightarrow W$ |
| $\omega^{-1}$ | $(1-\alpha^{-1}) \in (0,1)$ | relative size of the second immune protection period from $W \longrightarrow S$ |
| $\nu$ | $> 0$ | boosting force |

Table 1: Description of parameters of the SIRWS system.

The population is normalized to 1, and the basic reproduction number of our system, which we compute using the next-generation matrix method [13] is $\mathcal{R}_0 = \frac{\beta}{\gamma+\mu}$.

The symmetric partition of the immunity period means that the expected duration in $R$ is $(2\kappa)^{-1}$ and in $W$ is $(2\kappa)^{-1}$, thus the average duration of immune protection is

$$\frac{1}{2\kappa} + \frac{1}{2\kappa} = \frac{1}{\kappa},$$

where $\kappa$ is the immune decay rate. We relax this symmetry by introducing two new parameters $\alpha > 1$ and $\omega > 1$ setting the average time spent in $R$ and $W$ to $(\alpha\kappa)^{-1}$ and $(\omega\kappa)^{-1}$, respectively. Hence,

$$\frac{1}{\alpha\kappa} + \frac{1}{\omega\kappa} = \frac{1}{\kappa} \qquad \text{that is} \qquad \omega = \frac{\alpha}{\alpha - 1}. \tag{2}$$

We use the relation $W(t) = 1 - S(t) - I(t) - R(t)$ to get the reduced system

$$\frac{dS}{dt} = -\beta IS + \omega\kappa W + \mu(1-S), \tag{3a}$$
$$\frac{dI}{dt} = \beta IS - \gamma I - \mu I, \tag{3b}$$
$$\frac{dR}{dt} = \gamma I - \alpha\kappa R + \nu\beta IW - \mu R. \tag{3c}$$

### 2.1 Equilibria and stability

The disease free equilibrium (DFE) of our system is $(1, 0, 0)$. There are two other equilibria, which we represent by $\mathbf{EE}_+ = (S^*, I^*_+, R^*_+)$ and $\mathbf{EE}_- = (S^*, I^*_-, R^*_-)$. Their corresponding formulae are

$$S^* = \tfrac{\gamma+\mu}{\beta}, \tag{4}$$

$$I^*_\pm = \frac{\pm\sqrt{(c_1\nu + c_2)^2 + c_3\nu} + (c_1\nu - c_2)}{2\beta\nu(\gamma+\mu)}, \tag{5}$$

$$R^*_\pm = \frac{\gamma+\mu+\omega\kappa}{2\beta\omega\kappa}\left[\left(2c_0 - \frac{1}{\gamma+\mu}\right)c_1 + \frac{1}{\nu(\gamma+\mu)}\left(c_2 \mp \sqrt{(c_1\nu+c_2)^2 + c_3\nu}\right)\right]. \tag{6}$$

where

$$c_0 = \frac{1}{\gamma+\mu+\omega\kappa}\cdot\left(1 + \frac{\omega\kappa}{\mu}\right)$$
$$c_1 = \mu(\beta - (\gamma+\mu)).$$
$$c_2 = (\gamma+\mu)(\alpha\kappa+\omega\kappa) + \mu(\gamma+\mu) + \alpha\omega\kappa^2$$
$$c_3 = 4\gamma(\beta - (\gamma+\mu))\alpha\omega\kappa^2$$

With regard to our epidemiological setting

$$(S(t), I(t), R(t)) \in \mathcal{D} := \left\{(s,i,r) \in \mathbb{R}^3_{\geq 0} \mid 0 \leq s+i+r \leq 1\right\},$$

only $\mathbf{EE}_+$ may be admissible and therefore is the endemic equilibrium.

Our analysis showed that the DFE is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. The type of bifurcation associated with the change in dynamics when $\mathcal{R}_0 = 1$ is established in the following Theorem.

**Theorem 2.1.** *For all the model parameters of the system (1), a transcritical bifurcation of forward type occurs at* $\mathcal{R}_0 = 1$.

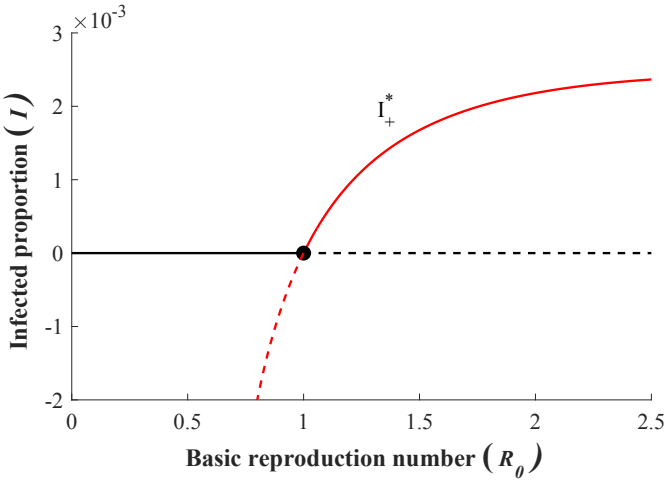



Figure 2: Transcritical bifurcation of forward type and the appearance of the LAS endemic equilibrium $\mathbf{EE}_+$ at $\mathcal{R}_0 = 1$.

## 2.2 The Routh-Hurwitz criterion for $\mathbf{EE}_+$

To ascertain the stability of the endemic equilibrium $\mathbf{EE}_+$ given that $\mathcal{R}_0 > 1$ holds, we apply the Routh-Hurwitz (RH) criterion [14, 15] to our characteristic polynomial

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$\begin{aligned}
a_0 &= 1,\\
a_1 &= \beta I_+^*(1+\nu) + (\alpha\kappa + \omega\kappa + 2\mu),\\
a_2 &= \beta I_+^*[(\alpha\kappa + \omega\kappa + 2\mu) + \gamma + \beta\nu I_+^* + \mu\nu] + (\omega\kappa + \mu)(\alpha\kappa + \mu),\\
a_3 &= \beta I_+^*[(\omega\kappa + \mu)(\alpha\kappa + \mu) + (\gamma + \mu)\beta\nu I_+^* + \gamma(\alpha\kappa + \omega\kappa + \mu) +\\
&\quad \omega\kappa\beta\nu(1 - S^* - I_+^* - R_+^*)].
\end{aligned} \tag{7}$$

Primarily, the criterion states that $\mathbf{EE}_+$ is locally asymtotically stable iff the following inequalities are satisfied

$$a_i > 0, \quad \text{for } i = 0, 1, 2, 3, \text{ and}$$
$$a_1 a_2 > a_3.$$

The positivity of $a_0, \ldots, a_3$ is readily established. Hence we define the function

$$y_\nu(\alpha) = a_1 a_2 - a_3, \tag{8}$$

and analyze signs changes for $\alpha > 1$ and $\nu > 0$.

### 2.3 Transformation of $y_\nu(\alpha)$

Noticing the symmetry in the formulae in (5) and (7) with respect to $\alpha$ and $\omega$, we further make the substitution

$$\eta = \kappa(\alpha + \omega) = \kappa(\alpha\omega) = \kappa\frac{\alpha^2}{\alpha - 1}, \tag{9}$$

with $\eta \in [4\kappa, \infty)$. With the help of this substitution, we obtain a different version of (8) in terms of $\eta$

$$y_\nu(\alpha) \equiv y_\nu(\eta) = \hat{a}_1 \hat{a}_2 - \hat{a}_3,$$

where

$$\begin{aligned}
\hat{a}_1 &= \hat{I}(1 + \nu) + (\eta + 2\mu), \\
\hat{a}_2 &= \hat{I}[(\eta + \mu) + (\gamma + \mu) + \mu\nu + \nu\hat{I}] + \mu(\eta + \mu) + \kappa\eta, \\
\hat{a}_3 &= \hat{I}[2\nu\hat{I}(\gamma + \mu) - \nu\mu(\beta - (\gamma + \mu)) + (\gamma + \mu)(\mu + \eta) + \kappa\eta],
\end{aligned}$$

with $\hat{I} = \beta I_+^*$.

A significant feature of $y_\nu(\alpha)$ made known by the substitution (9) is that there is a bijection $(1, 2) \ni \alpha \mapsto \alpha' \in (2, \infty)$ such that $y_\nu(\alpha) = y_\nu(\alpha')$. In particular, local extrema at $\alpha \neq 2$ appear in pairs.

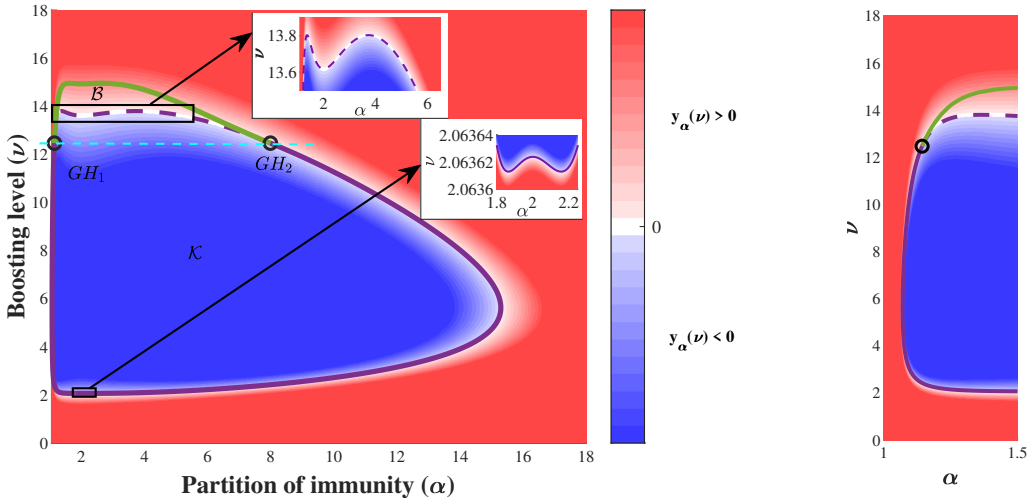
Furthermore, using the chain rule, we obtain that

$$\frac{\partial y_\nu}{\partial \alpha} = \frac{\partial y_\nu}{\partial \eta} \cdot \frac{\mathrm{d}\eta}{\mathrm{d}\alpha} = \frac{\partial y_\nu}{\partial \eta} \cdot \frac{\kappa\alpha(\alpha-2)}{(\alpha-1)^2}.$$

Clearly, $\alpha = 2$ (that is $\eta = 4\kappa$) is a critical point of $y_\nu$ for all immune boosting parameters $\nu$. We recall that $\alpha = 2$ represents the symmetric partition of the immunity period used in previous investigations of the SIRWS model. Other values of $\alpha \neq 2$, represent our proposed asymmetric partition of the immunity period, and it produces significantly different qualitative behavior in the SIRWS model.

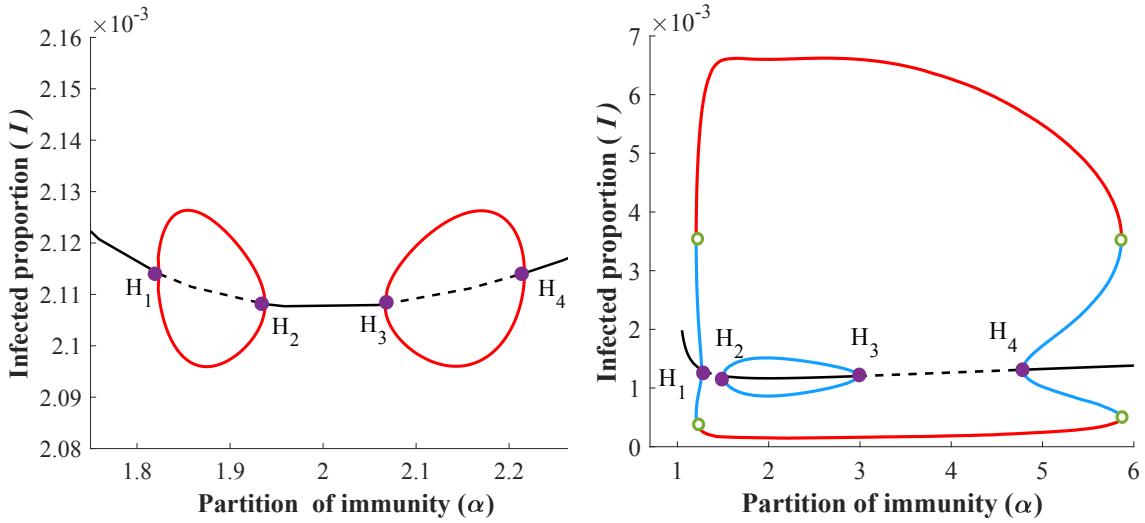
### 2.4 Numerical simulations

For the numerical investigations, we use the following parameters $\gamma = 17$, $\kappa = 1/10$, $\mu = 1/80$, and $\beta = 260$, taken from [7], where the authors studied natural immune boosting in pertussis dynamics. We construct a heatmap of (8), see Figure 3, where the red and blue regions indicate where $\mathbf{EE}_+$ is stable and unstable respectively. As already shown earlier the local extrema occur in pairs, we observe this phenomenon from the two inserts highlighted in Figure 3. Furthermore, it also reveals double bubbles of instability for certain boosting rates, *e.g.* $\nu = 2.06362$ and $\nu = 13.7$, see Figure 4 for their corresponding bifurcation diagrams, which are constructed using `MatCont` [16].




(a) (b)

Figure 3: Heatmap of the RH stability criterion (8) and bistability region. Purple curve represents $y_\nu(\alpha) = 0$. Points on the thick purple curve are supercritical Hopf bifurcation points and points on the broken purple curve are subcritical Hopf bifurcation points.




(a) (b)

Figure 4: Bifurcation diagram w.r.t $\alpha$ with $\nu = 2.06362$ (4a), and $\nu = 13.7$ (4b). We clearly see the *double endemic bubbles*, and a small bubble appearing inside the region of stable oscillations. The concept of the endemic bubble is found in [17].

# 3 The SIRWJS model: a compartmental model with waning and boosting, where secondary exposure can make the host infective

In the SIRWJS model, boosting of immunity occurs through a secondary infectious state, labelled $J$. According to the flow diagram of this model displayed in Figure 5, the primary force of infection is $\beta(I+\xi J)$. The path from $W$ to $R$ results in a boosting of the individual's immunity level. On the other hand, in the absence of re-exposure to the disease causing pathogen, hosts eventually lose their immunity modelled as a transition from $W$ back to the $S$ compartment where they are fully susceptible again to the infection.

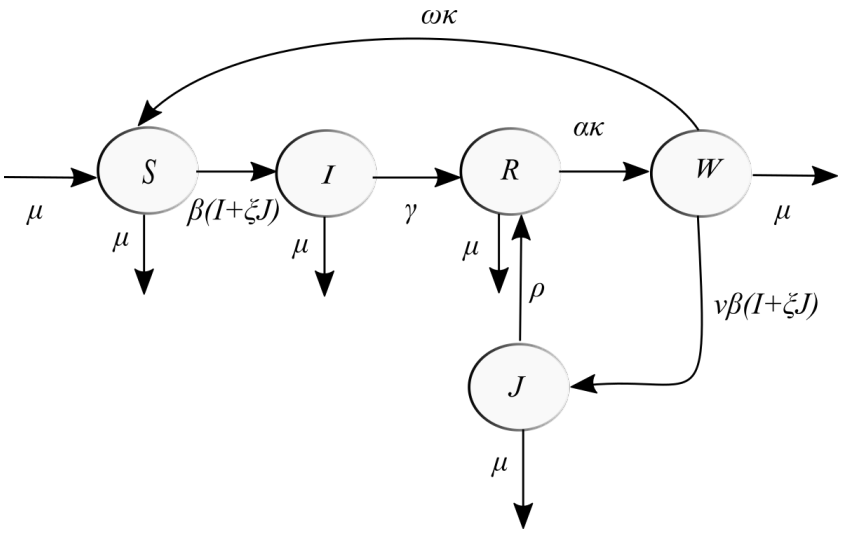



Figure 5: Flow diagram for the SIRWS system with secondary infections

The governing system of ordinary differential equations describing the dynamics presented in Figure 5

$$\frac{dS}{dt} = -\beta(I+\xi J)S + \omega\kappa W + \mu(1-S) \tag{10a}$$

$$\frac{dI}{dt} = \beta(I+\xi J)S - \gamma I - \mu I \tag{10b}$$

$$\frac{dR}{dt} = \gamma I - \alpha\kappa R + \rho J - \mu R \tag{10c}$$

$$\frac{dW}{dt} = \alpha\kappa R - \omega\kappa W - \nu\beta(I+\xi J)W - \mu W \tag{10d}$$

$$\frac{dJ}{dt} = \nu\beta(I+\xi J)W - \mu J - \rho J, \tag{10e}$$

with descriptions and assumptions on the system parameters are summarized in Table 1 and Table 2.

| | | |
|---|---|---|
| $\xi$ | $\geq 0$ | relative infectivity of secondary infections w.r.t. primary |
| $\rho$ | $> 0$ | recovery rate from secondary infection |

Table 2: Parameters of the SIRWJS system.

The substitution

$$R = 1 - S - I - W - J \tag{11}$$

reduces system (10) to

$$\frac{dS}{dt} = -\beta(I + \xi J)S + \omega\kappa W + \mu(1 - S) \tag{12a}$$
$$\frac{dI}{dt} = \beta(I + \xi J)S - \gamma I - \mu I \tag{12b}$$
$$\frac{dW}{dt} = \alpha\kappa(1 - S - I - W - J) - \omega\kappa W - \nu\beta(I + \xi J)W - \mu W \tag{12c}$$
$$\frac{dJ}{dt} = \nu\beta(I + \xi J)W - \mu J - \rho J \tag{12d}$$

Note that the feasible region for our epidemiological setting

$$(S(t), I(t), W(t), J(t)) \in \mathcal{D} := \left\{(s, i, w, j) \in \mathbb{R}^4_{\geq 0} \mid 0 \leq s + i + w + j \leq 1\right\}$$

is forward invariant and the the basic reproduction number $\mathcal{R}_0 = \frac{\beta}{\gamma+\mu}$. We again relax this restrictive assumption of symmetric partition of the immunity period such that the total period of immune protection is

$$\frac{1}{\alpha\kappa} + \frac{1}{\omega\kappa} = \frac{1}{\kappa}, \tag{13}$$

under the assumption of $\alpha + \omega = \alpha\omega$.

### 3.1 Equilibria and stability analysis

The disease free equilibrium (DFE) of (12) is $(S^*, I^*, W^*, J^*) = (1, 0, 0, 0)$, and formulae for the non-trivial equilibria is

$$S^* = \frac{\gamma+\mu}{\beta} - \frac{\nu\xi(\gamma+\mu)}{\rho+\mu}W^*, \tag{14}$$

$$I^* = \mu\left(\frac{1}{\gamma+\mu} - \frac{1}{\beta}\right) + \left(\frac{\omega\kappa}{\gamma+\mu} + \frac{\mu\nu\xi}{\rho+\mu}\right)W^*, \tag{15}$$

$$J^* = \frac{\nu\beta I^* W^*}{\rho+\mu-\nu\beta\xi W^*}. \tag{16}$$

Note that $\rho+\mu-\nu\beta\xi W^* \neq 0$, and $W^*$ is the solution of the quadratic equation

$$f(W^*) := A(W^*)^2 + BW^* + C = 0, \tag{17}$$

with

$$\begin{aligned} A &= \nu\beta^2\Big[-\nu\xi^2(\gamma+\mu)Q_0 + \xi Q_1 + \big(\alpha\kappa(\rho+\mu) - \nu\xi\mu(\gamma+\mu) - \eta\kappa(\rho+\mu)\big)Q2\Big], \\ B &= \beta(\rho+\mu)\Big[\big(\nu\xi(\gamma+\mu) - \nu\xi(\beta-\gamma-\mu)\big)Q_0 - Q_1 - \nu\mu(\beta-\gamma-\mu)Q_2\Big], \\ C &= (\beta-\gamma-\mu)(\rho+\mu)^2 Q_0, \end{aligned} \tag{18}$$

where

$$\begin{aligned} \eta &:= \alpha+\omega = \alpha\omega, \\ Q_0 &:= \alpha\kappa\gamma, \\ Q_1 &:= \Big[(\gamma+\mu)(\eta\kappa+\mu) + \eta\kappa^2\Big](\rho+\mu), \\ Q_2 &:= \alpha\kappa+\rho+\mu. \end{aligned} \tag{19}$$

For $\mathcal{R}_0 > 1$, there is an endemic equilibrium (EE) given by the root

$$W^* \equiv W^*_- = \frac{-B-\sqrt{B^2-4AC}}{2A}. \tag{20}$$

Naturally, non-negative equilibria exist when $\mathcal{R}_0 > 1$. However, our investigations revealed the existence of other non-negative equilibria occurring in instances when $\mathcal{R}_0 < 1$.

**Theorem 3.1.** *Let*

$$\Theta = \nu\xi(\gamma + \mu)Q_0 - Q_1, \tag{21}$$

*with $Q_0, Q_1$ defined in (19). If $\Theta > 0$, then there is a $0 < \tilde{\beta} < \gamma + \mu$ such that, besides the DFE, there are two other epidemiologically relevant equilibria for $\beta \in (\tilde{\beta}, \gamma + \mu)$ and only the DFE for $\beta < \tilde{\beta}$. On the other hand, if $\Theta \leq 0$, then the only epidemiologically relevant equilibrium is the DFE for $\mathcal{R}_0 < 1$.*

The sign of $\Theta$ plays a significant role in determining the type of bifurcation that occurs when $\mathcal{R}_0 = 1$. This result is formally stated in Theorem 3.2.

**Theorem 3.2.** *If $\Theta > 0$, then a transcritical bifurcation of backward type occurs at $\mathcal{R}_0 = 1$, and when $\Theta < 0$, then a transcritical bifurcation of forward type occurs at $\mathcal{R}_0 = 1$.*

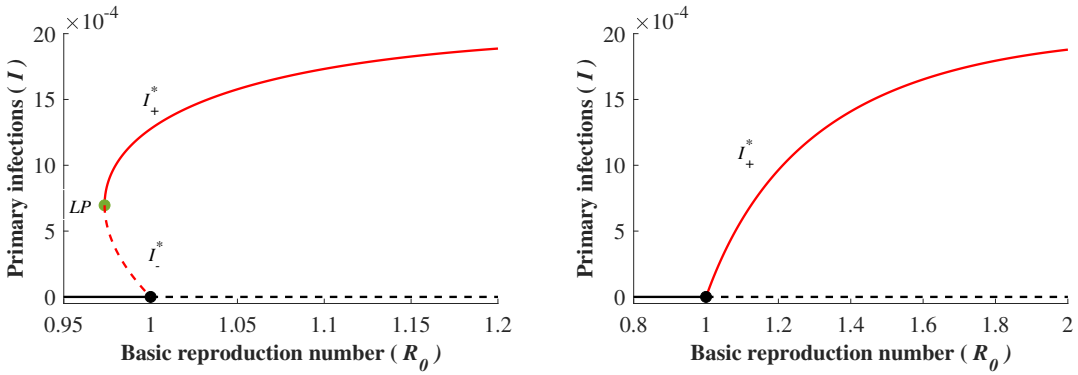



Figure 6: Backward bifurcation (left) and forward bifurcation (right) at $\mathcal{R}_0 = 1$. Stable branches are marked with continuous and unstable branches with dashed lines. The parameters used for both cases are $\rho = 17, \kappa = 0.1, \gamma = 17, \mu = 0.0125, \nu = 150, \alpha = 200$. The relative infectivity in the backward case is $\xi = 0.9$ and in the forward case $\xi = 0.001$. The saddle-node bifurcation point is marked with LP on the equilibria branch.

## 3.2 The Routh-Hurwitz criterion for EE

For the stability of the endemic equilibrium, we obtain the characteristic polynomial

$$\det(\mathbf{J} - \lambda I) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0 \tag{22}$$

with $a_4 = \det(\mathbf{J})$. Here, $\mathbf{J}$ is the Jacobian of our system evaluated at EE and expressions for the coefficients of (22) are in [18].

By applying the Routh-Hurwitz stability criterion, given that $\mathcal{R}_0 > 1$ holds, we found that the

EE is locally asymptotically stable if and only if the coefficients of the characteristic polynomial (22) satisfy

(i) $a_i > 0$ for $i = 1, 2, 3, 4$,

(ii) $a_1 a_2 > a_3$, and

(iii) $a_1 a_2 a_3 > a_1^2 a_4 + a_3^2$.

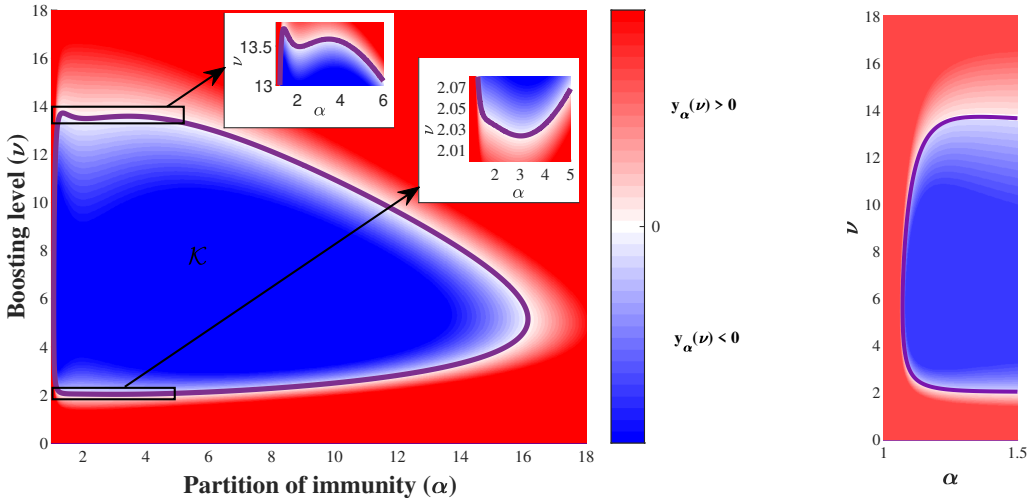
We establish the positivity of the coefficients that is condition *(i)*, details of which are outlined in [18]. For *(iii)*, we define

$$y_\nu(\alpha, \xi) = a_1 a_2 a_3 - (a_1^2 a_4 + a_3^2), \tag{23}$$

such that all conditions of the criterion are satisfied if and only if $y_\nu(\alpha, \xi) > 0$. We note here that condition *(ii)* can be derived from the other two conditions.

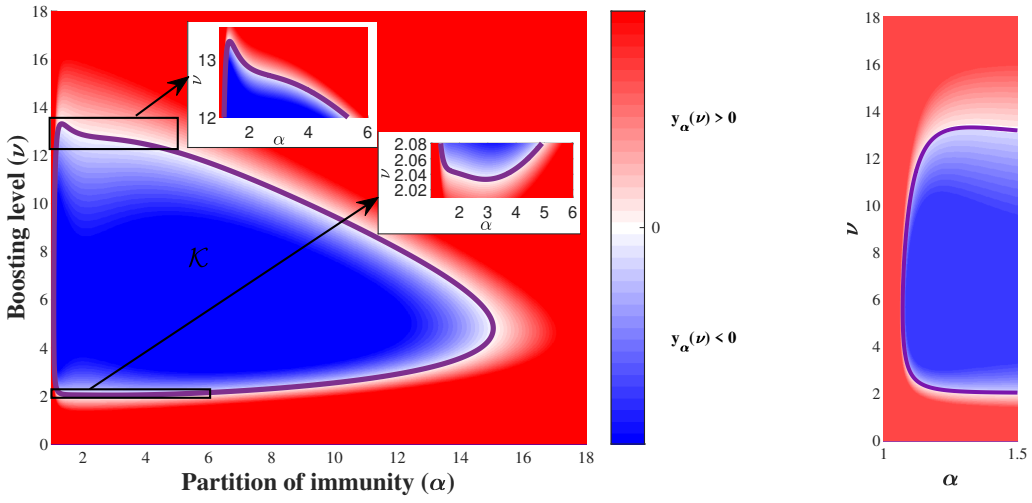
### 3.3 Numerical simulations

Using the same parameters as before, we construct heatmaps to study the sign of $y_\alpha(\nu, \xi)$, given by (23). Here, $\xi \in (0, 1)$ is taken from [9]. For $\xi = 0$, we readily experience changes in the dynamics w.r.t Figure 3. The instability set, marked as $\mathcal{K}$ is somewhat similar but the regular shape resulting in simultaneous appearance of double-bubbles of instability is lost, see Figure 7. The region around $\nu \approx 2.06$ displays much simpler behaviour and for $\nu \approx 13.5$, we still see bubbles, though without the symmetry they possess. The numerical investigations also revealed that as $\xi$ grows, the curve $y_\nu(\alpha, \xi) = 0$ deforms so that these double-bubbles disappear, see Figure 8. In addition, the region $\mathcal{K}$ shrinks and disappears, hence it results in the increase of local asymptotic stability region of the EE.




(a) (b)

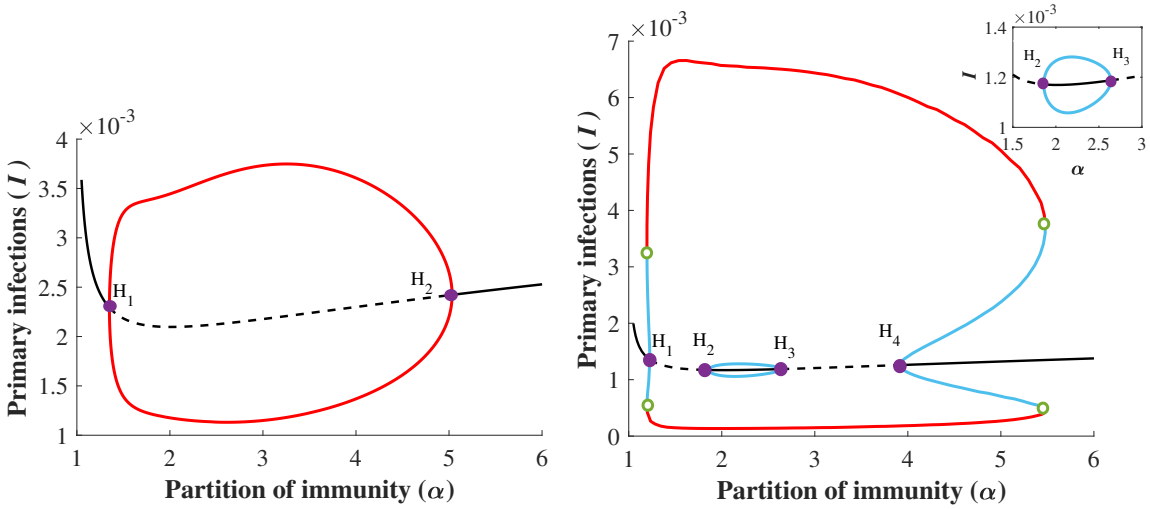
Figure 7: Heatmap of the RH criterion (23) for $\xi = 0$. Purple curve represents $y_\nu(\alpha) = 0$.




(a) (b)

Figure 8: Heatmap of the RH criterion (23) for $\xi = 10^{-4}$. Purple curve represents $y_\nu(\alpha) = 0$.

Depicted in Figure 9 are one-parameter bifurcation diagrams showing the bubble for small and large $\nu$ respectively.




(a) (b)

Figure 9: One-parameter bifurcation diagram with $\xi = 10^{-5}$ for (a) $\nu = 2.07$ and (b) $\nu = 13.45$

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