Research article

Waning immunity can drive repeated waves of infections

Desmond Z. Lai[1,*] **and Julia R. Gog**[1,2]

[1] Department of Applied Mathematics and Theoretical Physics (DAMTP), University of Cambridge, United Kingdom

[2] Joint UNIversities Pandemic and Epidemiological Research (JUNIPER) Consortium, United Kingdom

* **Correspondence:** Email: zdl21@cam.ac.uk

Abstract: In infectious disease models, it is known that mechanisms such as births, seasonality in transmission and pathogen evolution can generate oscillations in infection numbers. We show how waning immunity is also a mechanism that is sufficient on its own to enable sustained oscillations. When previously infected or vaccinated individuals lose full protective immunity, they become partially susceptible to reinfections. This partial immunity subsequently wanes over time, making individuals more susceptible to reinfections and potentially more infectious if infected. Losses of full and partial immunity lead to a surge in infections, which is the precursor of oscillations. We present a discrete-time Susceptible-Infectious-Immune-Waned-Infectious (SIRWY) model that features the waning of fully immune individuals (as a distribution of time at which individuals lose fully immunity) and the gradual loss of partial immunity (as increases in susceptibility and potential infectiousness over time). A special case of SIRWY is the discrete-time SIRS model with geometric distributions for waning and recovery. Its continuous-time analogue is the classic SIRS with exponential distributions, which does not produce sustained oscillations for any choice of parameters. We show that the discrete-time version *can* produce sustained oscillations and that the oscillatory regime disappears as discrete-time tends to continuous-time. A different special case of SIRWY is one with fixed times for waning and recovery. We show that this simpler model can also produce sustained oscillations. In conclusion, under certain feature and parameter choices relating to how exactly immunity wanes, fluctuations in infection numbers can be sustained without the need for any additional mechanisms.

Keywords: disease dynamics; bifurcation analysis; periodicity; time since infection; time since recovery

1. Introduction

Sustained oscillations in the number of infections have been observed for some infectious diseases. A classic example is the highly regular biennial pattern in measles case numbers in England and Wales from 1950 to 1967 (depicted in Fine and Clarkson, 1982 [1]), which is driven by a combination of births and seasonality of transmission related to the opening of school terms.

Mathematically, sustained oscillations can be demonstrated by relatively simple models. Such models focus on a key mechanism such as seasonality in transmission (Aron and Schwartz, 1984 [2]), births and vaccinations (Earn et al., 2000 [3]), pathogen evolution through time scale separation of within and between season dynamics (Andreasen, 2003 [4]) or in combination with seasonality forcing (Dushoff et al., 2004 [5]), changes in human social behavior (d'Onofrio and Manfredi, 2009 [6]), importations of infectious individuals (Silva and Monteiro, 2014 [7]), heterogeneity in transmission due to chronological age (Kuniya and Inaba, 2023 [8] in this special issue) and change in behavior of healthy individuals due to social pressure (Baccili and Monteiro, 2023 [9]). These mechanisms can drive a surge in infections through three main methods: (i) replenishment of susceptible individuals, (ii) replenishment of infectious individuals and (iii) variation of the transmission parameter. Sustained oscillations are possible in these models for certain choices of parameter values.

We argue that waning immunity is also a mechanism that can, by itself, produce sustained oscillations in infection numbers. For diseases with waning immunity, previous infection or vaccination only confers a temporary period of full protection against reinfection. The duration of protection ranges from a few weeks or months for the Omicron variant of SARS-CoV-2 (Burkholz et al., 2023 [10] and Bobrovitz et al., 2023 [11]), to possibly a few years for pertussis (Wendelboe et al., 2005 [12]). As fully immune individuals wane, more individuals become susceptible to infection and more infections occur. Eventually, fewer susceptibles remain and the number of infections falls. When new individuals lose full immunity, replenishment of susceptibles occurs and the cycle repeats.

Here, we present a discrete-time waning immunity model (SIRWY model) that features the aforementioned loss of full immunity as a *general* population waning distribution, where the proportion of fully immune individuals who wane can be specified on a daily basis. In this Susceptible-Infectious-Immune-Waned-Infectious (SIRWY) model, S and I represent susceptible and infectious individuals without prior immunity, R represents fully immune individuals and W and Y represent waned (susceptible) and infectious individuals with prior immunity.

A special case of SIRWY is the Susceptible-Infectious-Recovered-Susceptible (SIRS) model with geometric distributions for waning and recovery. This is the discrete-time counterpart to the continuous-time classic SIRS model with exponential distributions (Hethcote, 1976 [13]):

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \omega R - \beta S I$$
$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta S I - \gamma I \qquad\qquad (1.1)$$
$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I - \omega R.$$

In these equations, ω is the waning rate, β is the transmission rate per infectious individual and γ is the recovery rate. For all parameter values, the number of infections either tends to zero or the endemic equilibrium via exponential decay or damped oscillations (see Hethcote, 1976 [13] or a different proof

in §6 of this paper). A variation of the classic SIRS is the $SIR_1...R_nS$ model with Erlang distribution (sum of exponentials), in which individuals wait in the additional immune sub-compartments before becoming susceptible. It has been shown that sustained oscillations are possible for the $SIR_1...R_nS$ model when $n \geq 3$ (Hethcote et al., 1981 [14]). In this paper, we show that the discrete-time version of SIRS with geometric distributions can generate undamped oscillations, and the oscillatory regime disappears when discrete-time tends to continuous-time.

A different special case of SIRWY is one with homogeneous waning and recovery distributions. This model has fixed waning and recovery times, in which individuals spend exactly the same number of days fully immune and are infectious for exactly same number of days if infected. Due to this fixed delay in terms of time spent in the immune compartment, surges in susceptibles occur at fixed intervals. We show that sustained oscillations can be produced, similar to continuous-time models with delays (Hethcote et al., 1981 [14], Diekmann and Montijn, 1982 [15] and Kyrychko and Blyuss, 2005 [16]).

Besides having a general waning distribution, the full SIRWY model features another effect of waning immunity, the loss of partial immunity, which also contributes to oscillatory dynamics. After losing full immunity, individuals still have partial immunity that reduces their susceptibility to reinfection and potentially also their infectiousness if infected. However, partial immunity wanes over time and individuals become more susceptible and infectious. The accumulation of susceptibility and infectiousness in the population leads to a surge in infections. Similar to the loss of full immunity, this results in a cyclical pattern in infection numbers.

While waning immunity is a mechanism that *can* generate oscillations, it does not always do so as it depends on the choice of waning immunity effects (losses of full and partial immunity) and how exactly these effects are represented in the model (discrete/continuous time, population waning distribution, changes in susceptibility and infectiousness over time). As a general model which also encompasses different simpler sub-models, SIRWY allows an appropriate model to be chosen based on what is currently known about the infectiousness of the disease and the effects of waning immunity in the population, before scaling up to the full model when needed. Furthermore, for model implementation, discrete-time SIRWY is a system of difference equations, which makes numerical simulations easier to perform as compared with integro-differential equations in continuous-time.

The rest of this paper is structured as follows. We start by presenting the Susceptible-Infectious-Immune-Waned-Infectious (SIRWY) model in §2, before presenting a stability analysis about the disease-free and endemic equilibria in §3. We then proceed to recover two sub-models of the SIRWY model, one with fixed waning and recovery times in §4 and another with geometric distributions for waning and recovery in §5. For these sub-models, we perform bifurcation analysis, on top of the stability analyses about the disease-free and endemic equilibria. We then study the continuous-time exponential SIRS model (equation 1.1) in §6, before making the connection between this model and its discrete-time counterpart in §7 to show that the oscillatory regime disappears in continuous-time.

2. The RWY model

Since our goal is to understand how waning immunity shapes the *long term* dynamics of a disease, we consider a population comprising individuals with some degree of immunity because all individuals would have been infected at least once in the long run. Furthermore, as the focus is on waning immunity, other mechanisms that replenish susceptibles such as births are not included (similar to how
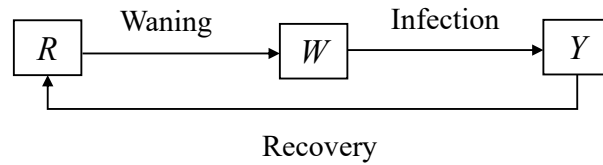
Figure 1. Immune-Waned-Infectious (RWY) model for a population with prior immunity.

the evolutionary model is constructed in Pease, 1987 [17]). Since we are also not considering pathogen evolution, this means that all properties of the disease related to infection, recovery and waning of immunity are unchanged over extended periods. Let R_t, W_t and Y_t denote the proportions of the population who are in the fully immune, waned and infectious compartments respectively on day t. Here, we always discuss in terms of *proportions* of population in each compartment rather than exact numbers. For completeness, we give the full Susceptible-Infectious-Immune-Waned-Infectious (SIRWY) model in Appendix A but our focus is on the Immune-Waned-Infectious (RWY) model (Figure 1) for the rest of the paper.
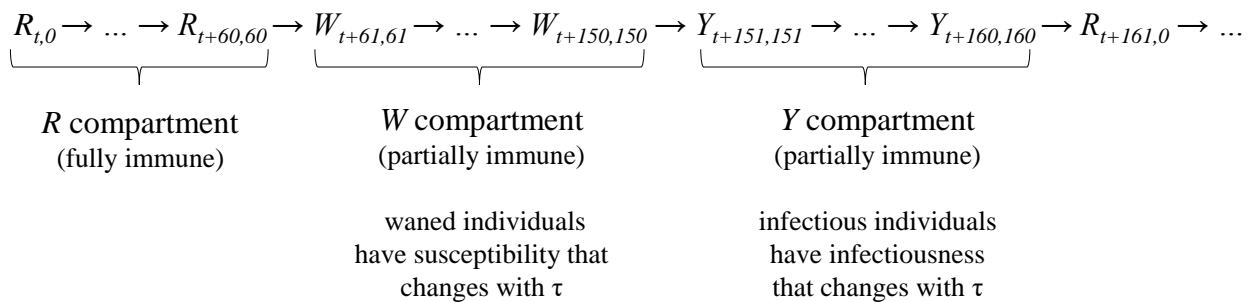
$$R_{t,0} \to \ldots \to R_{t+60,60} \to W_{t+61,61} \to \ldots \to W_{t+150,150} \to Y_{t+151,151} \to \ldots \to Y_{t+160,160} \to R_{t+161,0} \to \ldots$$

R compartment
(fully immune)

W compartment
(partially immune)

waned individuals
have susceptibility that
changes with τ

Y compartment
(partially immune)

infectious individuals
have infectiousness
that changes with τ

Figure 2. Example of how an individual moves between compartments. The second component of the subscript is the time since recovery τ, which starts from 0, increases and then resets to 0 when the individual rejoins the R compartment. Subtracting the second subscript from the first subscript gives the day of recovery t.

Each compartment in the model (R, W and Y) is further divided into sub-compartments based on time since most recent recovery τ (first introduced by Kermack and McKendrick, 1932 [18]) with respect to day t. For example, $R_{t,\tau}$ denotes the proportion of population in the immune R compartment on day t and day τ since recovery. In this notation, we can deduce the day of recovery by subtracting the second subscript from the first subscript (day $t - \tau$ for $R_{t,\tau}$).

The use of time since recovery τ allows us to track the total number of days an individual spends in the R, W and Y compartments. Time since recovery τ starts from day 0 when the individual first joins the R compartment and increases with the day as the individual progresses through the waned W and infectious Y compartments. The value of τ then resets to 0 when the individual rejoins the R compartment (see Figure 2 for an example). This formulation tracks a full RWY cycle (R→W→Y→R→ $\cdots$), not just the time spent in each compartment (Inaba, 2001 [19]).

We cover the entire immune spectrum as we have the fully immune R compartment and the waned W and infectious Y compartments with partial immunity based on each day τ since recovery. We do not have the fully susceptible S in the RWY model but it is present in the full SIRWY model. The

R compartment represents full immunity against infection, unlike that in Kermack and McKendrick (1932) [18], where R individuals are partially susceptible to infection. Partial immunity provides a reduction in susceptibility α_τ of waned W individuals and a reduction in infectiousness χ_τ of infectious Y individuals. Since the susceptibility of waned individuals depends on the time since recovery τ, there is no need to use the method of stages to partition susceptibles into a fixed number of sub-compartments $S_1, S_2, ..., S_n$ with n different susceptibilities.

Table 1. Variables, indices and parameters of the RWY model.

Symbol	Variable	Symbol	Index	Symbol	Parameter
R	immune	θ	time since infection	ϕ_θ	fraction still infectious
W	waned	$\bar\theta + 1$	max infectious period	ξ_θ	infectiousness
y	newly infected	τ	time since recovery	ζ_τ	fraction still immune
Y	infectious	$\bar\tau + 1$	max immune period	χ_τ	reduction in infectiousness
F	force of infection	$\tilde\tau$	max value of τ	α_τ	reduction in susceptibility

The RWY model, along with a summary of terms in Table 1, is described by the following system of difference equations,

$$
\begin{aligned}
R_{t+1,0} &= \sum_{\theta=0}^{\bar\theta}(\phi_\theta - \phi_{\theta+1})y_{t-\theta} \\
R_{t+1,\tau} &= \zeta_\tau R_{t-(\tau-1),0} && \text{for } \tau \in \{1, \cdots, \bar\tau\} \\[2mm]
\hline
W_{t+1,1} &= (\zeta_0 - \zeta_1)R_{t,0} \\
W_{t+1,\tau} &= W_{t,\tau-1} - y_{t+1,\tau} + (\zeta_{\tau-1} - \zeta_\tau)R_{t-(\tau-1),0} && \text{for } \tau \in \{2, \cdots, \bar\tau + 1\} \\
W_{t+1,\tau} &= W_{t,\tau-1} - y_{t+1,\tau} && \text{for } \tau \in \{\bar\tau + 2, \cdots, \tilde\tau - 1\} \\
W_{t+1,\tilde\tau} &= W_{t,\tilde\tau-1} + W_{t,\tilde\tau} - y_{t+1,\tilde\tau} \\[2mm]
\hline
F_t &= \sum_{\theta=0}^{\bar\theta} \sum_{\tau=2}^{\tilde\tau} \xi_\theta \phi_\theta y_{t-\theta,\tau}(1 - \chi_\tau) \\
y_{t+1,\tau} &= (1 - \alpha_{\tau-1})W_{t,\tau-1}F_t && \text{for } \tau \in \{2, \cdots, \tilde\tau - 1\} \\
y_{t+1,\tilde\tau} &= [(1 - \alpha_{\tilde\tau-1})W_{t,\tilde\tau-1} + (1 - \alpha_{\tilde\tau})W_{t,\tilde\tau}]F_t
\end{aligned}
\tag{2.1}
$$

and the conservation of individuals means that

$$
R_t + W_t + Y_t = 1. \tag{2.2}
$$

Each compartment (R, W and Y) can be defined in terms of their corresponding sub-compartments (see Table 2).

In the RWY model (equation 2.1), the *newly* infected y is used instead of the infectious Y because the infectious $Y_t = \sum_{\theta=0}^{\bar\theta} \phi_\theta y_{t-\theta}$ can be expressed in terms of newly infected y at time t and earlier times, with ϕ_θ representing the fraction of $y_{t-\theta}$ still infectious on day θ since infection (as similarly defined by Kermack and McKendrick, 1927 [20]). Here, $\phi_0 = 1$ because all infecteds would have just joined

Table 2. Definition of variables in the RWY model. The indices t, τ and θ refer to the time, time since recovery and time since infection respectively. The summations correspond to the max immune period ($\overline{\tau}+1$), the max value of τ ($\tilde{\tau}$) and the max infectious period ($\overline{\theta}+1$).

Symbol	Variable	Composition
R_t	immune	$R_t = \sum_{\tau=0}^{\overline{\tau}} R_{t,\tau}$ or $R_t = \sum_{\tau=0}^{\overline{\tau}} \zeta_\tau R_{t-\tau,0}$
W_t	waned	$W_t = \sum_{\tau=1}^{\tilde{\tau}} W_{t,\tau}$
Y_t	infectious	$Y_t = \sum_{\tau=2}^{\tilde{\tau}} Y_{t,\tau}$ or $Y_t = \sum_{\theta=0}^{\overline{\theta}} \phi_\theta y_{t-\theta}$
y_t	newly infected	$y_t = \sum_{\tau=2}^{\tilde{\tau}} y_{t,\tau}$

the infectious compartment the same day they became infected, which is day 0 since infection. By defining the maximum allowable infectious period to be $\overline{\theta} + 1$ days, the fraction still infectious $\phi_\theta = 0$ for $\theta \geq \overline{\theta} + 1$ and the infectious Y_t is expressed in terms of newly infecteds $y_{t-\overline{\theta}}$ to y_t .

The force of infection F_t has contributions from the fraction ϕ_θ of newly infecteds $y_{t-\theta,\tau}$ who are still infectious on day θ since infection. On day t, each of these individuals contributes a baseline infectiousness ξ_θ based on the time since infection θ, which is then reduced by a factor of χ_τ. Here, the τ in both χ_τ and $y_{t-\theta,\tau}$ refers to the number of days since recovery with respect to the day of infection $t - \theta$ and not with respect to day t. As the maximum infectious period is $\overline{\theta} + 1$ days, infectiousness $\xi_\theta = 0$ for $\theta \geq \overline{\theta} + 1$.

Individuals who recover on day $t + 1$ join the newly recovered $R_{t+1,0}$ immune compartment. Using the fraction still infectious ϕ_θ, we define the population recovery distribution $\phi_\theta - \phi_{\theta+1}$, which gives the proportion of newly infected $y_{t-\theta}$ who recover on day $\theta + 1$ since infection.

We define the immune R_t in a similar way as the infectious Y_t. Here, $R_t = \sum_{\tau=0}^{\overline{\tau}} \zeta_\tau R_{t-\tau,0}$, where ζ_τ represents the fraction of newly recovered $R_{t-\tau,0}$ still immune on day τ since recovery. Again, ζ_0 is equals to 1 because all individuals would have just joined the immune compartment the day they recover. By defining the maximum allowable immune period to be $\overline{\tau} + 1$ days, the fraction still immune $\zeta_\tau = 0$ for $\tau \geq \overline{\tau} + 1$ and the immune R_t only consists of newly recovereds $R_{t-\overline{\tau},0}$ to $R_{t,0}$. Since individuals can only remain in the immune compartment for $\overline{\tau} + 1$ days and the immune compartment is the first compartment individuals join after recovery, we can express $R_t = \sum_{\tau=0}^{\overline{\tau}} R_{t,\tau}$ in terms of its sub-compartments $R_{t,\tau}$, with the final term being $R_{t,\overline{\tau}}$ as $R_{t,\tau} = 0$ for $\tau \geq \overline{\tau} + 1$. Using the fraction still immune ζ_τ, we define the population waning distribution $\zeta_{\tau-1} - \zeta_\tau$, which gives the proportion of newly recovered $R_{t-(\tau-1),0}$ who wane on day τ since recovery.

The waned W_t and newly infected y_t can also be expressed in terms of their sub-compartments. For the waned on day t, $W_{t,\tau}$ starts from $W_{t,1}$ rather than $W_{t,0}$ because individuals are assumed to spend at least one day in the immune compartment before moving to the waned compartment. By the same reasoning, $y_{t,\tau}$ starts from $y_{t,2}$ because individuals are assumed to spend at least another day in the waned compartment. By defining a maximum value of $\tau = \tilde{\tau}$, we have $W_t = \sum_{\tau=1}^{\tilde{\tau}} W_{t,\tau}$ and $y_t = \sum_{\tau=2}^{\tilde{\tau}} y_{t,\tau}$, where the final terms $W_{t,\tilde{\tau}}$ and $y_{t,\tilde{\tau}}$ comprise all waned and newly infecteds individuals respectively with $\tau \geq \tilde{\tau}$. We may also define the infectious $Y_t = \sum_{\tau=2}^{\tilde{\tau}} Y_{t,\tau}$, in terms of its sub-compartments $Y_{t,\tau}$, where $Y_{t,\tilde{\tau}}$ comprises all infectious individuals with $\tau \geq \tilde{\tau}$.

The waning of partial immunity is a decrease in protection, which is equivalent to decreases in the value of the reduction in susceptibility α_τ and potentially also the reduction in infectiousness χ_τ as τ increases. As we are using a maximum value of $\tau = \tilde{\tau}$, the reduction in susceptibility α_τ reaches a constant value $\alpha_{\tilde{\tau}}$ for $\tau \geq \tilde{\tau}$ and the reduction in infectiousness χ_τ a constant value $\chi_{\tilde{\tau}}$ for $\tau \geq \tilde{\tau}$. Together with the population waning distribution $\zeta_{\tau-1} - \zeta_\tau$, these three features specify how the losses of full and partial immunity are modeled in the RWY model.

We conclude this section by remarking that we have intentionally made a linear approximation on how the newly infected y_t depends on the force of infection F_t (Hernandez-Ceron et al., 2013 [21], Diekmann et al., 2021 [22]). The full description is the following:

$$
\begin{aligned}
y_{t+1,\tau} &= (1 - \alpha_{\tau-1})W_{t,\tau-1}(1 - e^{-F_t}) & \text{for } \tau \in \{2, \cdots, \tilde{\tau} - 1\} \\
y_{t+1,\tilde{\tau}} &= [(1 - \alpha_{\tilde{\tau}-1})W_{t,\tilde{\tau}-1} + (1 - \alpha_{\tilde{\tau}})W_{t,\tilde{\tau}}](1 - e^{-F_t}).
\end{aligned}
\tag{2.3}
$$

This form, however, cannot be utilized easily as F_t is also a function of newly infected y_t at earlier times. As long as the force of infection F_t is not too large, we can work with the linearized equation (2.1) to perform stability analysis. Equation 2.1 also allows for efficient numerical simulations to be performed via matrix-vector multiplications.

3. Stability analysis

In this section, we derive the model equations (equation 2.1) at steady state. Letting time tend to infinity and using $*$ to denote the steady state, the immune R equations become

$$
\begin{aligned}
R_{*,0} &= y_* \\
R_{*,\tau} &= \zeta_\tau y_* & \text{for } \tau \in \{1, \cdots, \overline{\tau}\}
\end{aligned}
\tag{3.1}
$$

after substituting $\sum_{\theta=0}^{\overline{\theta}}(\phi_\theta - \phi_{\theta+1}) = 1$. Adding the equations in (3.1), the proportion of recovered individuals in the population at steady state is

$$
R_* = y_* \sum_{\tau=0}^{\overline{\tau}} \zeta_\tau,
\tag{3.2}
$$

where we have used $\zeta_0 = 1$. Performing a similar analysis on the waned equations, we get:

$$
\begin{aligned}
W_{*,1} &= (\zeta_0 - \zeta_1)y_* \\
W_{*,\tau} &= W_{*,\tau-1} - y_{*,\tau} + (\zeta_{\tau-1} - \zeta_\tau)y_* & \text{for } \tau \in \{2, \cdots, \overline{\tau} + 1\} \\
W_{*,\tau} &= W_{*,\tau-1} - y_{*,\tau} & \text{for } \tau \in \{\overline{\tau} + 2, \cdots, \tilde{\tau} - 1\} \\
W_{*,\tilde{\tau}-1} &= y_{*,\tilde{\tau}},
\end{aligned}
\tag{3.3}
$$

where we have substituted $R_{*,0} = y_*$. The waned equations can also expressed in terms of y_* and its components:

$$
\begin{aligned}
W_{*,1} &= (\zeta_0 - \zeta_1)y_* \\
W_{*,\tau} &= \sum_{k=\tau+1}^{\tilde{\tau}} y_{*,k} - \zeta_\tau y_* & \text{for } \tau \in \{2, \cdots, \overline{\tau}\} \\
W_{*,\tau} &= \sum_{k=\tau+1}^{\tilde{\tau}} y_{*,k} & \text{for } \tau \in \{\overline{\tau} + 1, \cdots, \tilde{\tau} - 1\}.
\end{aligned}
\tag{3.4}
$$

Note that in the above analysis, we do not have an expression for $W_{*,\tilde{\tau}}$ yet. The steady state equations for the force of infection and newly infectious are:

$$
\begin{aligned}
F_* &= \sum_{\theta=0}^{\bar{\theta}} \xi_\theta \phi_\theta \sum_{\tau=2}^{\tilde{\tau}} y_{*,\tau}(1 - \chi_\tau) \\
y_{*,\tau} &= (1 - \alpha_{\tau-1})W_{*,\tau-1}F_* && \text{for } \tau \in \{2, \cdots, \tilde{\tau} - 1\} \\
y_{*,\tilde{\tau}} &= [(1 - \alpha_{\tilde{\tau}-1})W_{*,\tilde{\tau}-1} + (1 - \alpha_{\tilde{\tau}})W_{*,\tilde{\tau}}]F_*.
\end{aligned}
\tag{3.5}
$$

Since $Y_t = \sum_{\theta=0}^{\bar{\theta}} \phi_\theta y_{t-\theta}$, the infectious proportion of population at steady state is

$$
Y_* = y_* \sum_{\theta=0}^{\bar{\theta}} \phi_\theta.
\tag{3.6}
$$

The steady state equations above will be used to derive the disease-free and endemic equilibria in the sub-sections that follow.

3.1. Disease-free equilibrium

We derive the disease-free equilibrium by setting the newly infected proportion of the population $y_{*,\tau}$ to be zero for all τ in the steady state equations 3.1, 3.4 and 3.5. By the conservation of individuals (equation 2.2), we find that in the disease-free equilibrium, all individuals end up in the waned $W_{*,\tilde{\tau}}$ compartment, with

$$
\begin{aligned}
y_{*,\tau} &= 0 && \text{for } \tau \in \{2, \cdots, \tilde{\tau}\} \\
W_{*,\tau} &= 0 && \text{for } \tau \in \{1, \cdots, \tilde{\tau} - 1\} \\
W_{*,\tilde{\tau}} &= 1.
\end{aligned}
\tag{3.7}
$$

We now proceed to set up a vector system of the model for linear stability analysis about the disease-free equilibrium. Let $x_{t+1} = g(x_t)$, where x_t is a vector that is formed by the newly infected $y_{t-\tilde{\tau}-\bar{\theta}}$ to y_t and the waned W_t. Here, g is a function to be specified using the model equations (2.1 and 2.2).

Linearizing about the disease-free equilibrium and performing elementary column operations on $J - \lambda I$ (where J is the Jacobian for the model equations evaluated at the disease-free equilibrium), we obtain the following characteristic equation:

$$
\lambda^{(\tilde{\tau}+\bar{\theta}+2)(\tilde{\tau}-1)-\bar{\theta}} \left[\lambda^{\bar{\theta}+1} - (1 - \alpha_{\tilde{\tau}})(1 - \chi_{\tilde{\tau}}) \sum_{k=0}^{\bar{\theta}} \xi_k \phi_k \lambda^{\bar{\theta}-k} \right] = 0.
\tag{3.8}
$$

Theorems related to the zeroes of polynomial are discussed in Appendix B. Using Theorem B.6 and Lemma B.7, we find that the disease-free equilibrium is asymptotically stable if and only if $\mathcal{R}_0 < 1$, where the modified basic reproductive ratio is defined as $\mathcal{R}_0 = (1 - \alpha_{\tilde{\tau}})(1 - \chi_{\tilde{\tau}}) \sum_{k=0}^{\bar{\theta}} \xi_k \phi_k$. Note that $\mathcal{R}_0 < 1$ is the explicit threshold for all solutions to tend towards the disease-free equilibrium. Here, $\mathcal{R}_0$ is a modified basic reproductive ratio because $\alpha_{\tilde{\tau}}$ and $\chi_{\tilde{\tau}}$ are not necessary zero, in particular, waned W individuals at $\tau = \tilde{\tau}$ may still be less susceptible to infection than susceptible S individuals.

There are two key observations. First, $\mathcal{R}_0$ depends on the reductions in infectiousness χ_τ and susceptibility α_τ only when $\tau = \tilde{\tau}$ and not earlier values of τ. This is because earlier values of τ are

transient and hence not present in the steady disease-free equilibrium. The only value of τ that has a lasting effect is when τ takes the maximum value of $\tilde{\tau}$ because τ remains at this value thereafter even as the day increases. Second, $\mathcal{R}_0$ is independent of population recovery distribution $\phi_\theta - \phi_{\theta+1}$ and population waning distribution $\zeta_{\tau-1} - \zeta_\tau$ (see equation 2.1). Again, this is because recovery takes place for a maximum allowable period of $\bar{\theta} + 1$ days (infectious period) and waning for a maximum allowable period of $\bar{\tau} + 1$ days (immune period), which are transient periods that do not continue until $\tau = \tilde{\tau}$.

3.2. Endemic equilibrium

For the endemic equilibrium, an explicit form is difficult to obtain because the steady state equations (3.1, 3.4 and 3.5) are non-linear in $y_{*,\tau}$. We can make progress in the special case where the reduction in susceptibility and infectiousness are independent of τ, namely, $\alpha_\tau = \alpha$ and $\chi_\tau = \chi$ respectively. Then, the force of infection and the newly infected (equation 3.5) can be simplified to:

$$
\begin{aligned}
F_* &= (1-\chi)y_* \sum_{\theta=0}^{\bar{\theta}} \xi_\theta \phi_\theta \\
y_{*,\tau} &= \mathcal{R}_0 y_* W_{*,\tau-1} \qquad \text{for } \tau \in \{2, \cdots, \tilde{\tau}-1\} \\
y_{*,\tilde{\tau}} &= \mathcal{R}_0 y_* (W_{*,\tilde{\tau}-1} + W_{*,\tilde{\tau}}),
\end{aligned}
\tag{3.9}
$$

where we have substituted $\mathcal{R}_0 = (1-\chi)(1-\alpha) \sum_{\theta=0}^{\bar{\theta}} \xi_\theta \phi_\theta$ defined earlier. Summing up the $y_{*,\tau}$ equations, we get $W_* = \frac{1}{\mathcal{R}_0}$. Substituting this equation along with equations 3.2 and 3.6 into $R_* + W_* + Y_* = 1$, we obtain an expression for y_*:

$$
y_* = \frac{\mathcal{R}_0 - 1}{\mathcal{R}_0 \left(\sum_{\tau=0}^{\bar{\tau}} \zeta_\tau + \sum_{\theta=0}^{\bar{\theta}} \phi_\theta \right)}.
\tag{3.10}
$$

The term in brackets is the sum of the fraction of newly recovered who remain immune and the fraction of newly infecteds who remain infectious throughout the entire immune and infectious period respectively. Clearly, y_* exists (is greater than zero) if and only if $\mathcal{R}_0$ is greater than one. Now, W_* and y_* are completely defined in terms of parameter values. Using $W_* = \sum_{\tau=1}^{\tilde{\tau}} W_{*,\tau}$, equation 3.4 and $W_* = \frac{1}{\mathcal{R}_0}$, we can express $W_{*,\tilde{\tau}}$ in terms of $y_{*,\tau}$:

$$
W_{*,\tilde{\tau}} = \frac{1}{\mathcal{R}_0} + y_* \sum_{\tau=1}^{\bar{\tau}} \zeta_\tau - \sum_{\tau=2}^{\tilde{\tau}} \sum_{j=\tau}^{\tilde{\tau}} y_{*,j}.
\tag{3.11}
$$

This equation, along with equations 3.9 and 3.3, allow us to obtain a closed form for the endemic equilibrium:

$$
\begin{aligned}
y_{*,\tau} &= \mathcal{R}_0 y_*^2 \sum_{k=0}^{\min(\tau-2,\bar{\tau})} (\zeta_k - \zeta_{k+1})(1 - \mathcal{R}_0 y_*)^{\tau-2-k} \qquad \text{for } \tau \in \{2, \cdots, \tilde{\tau}-1\} \\
y_{*,\tilde{\tau}} &= y_* \sum_{k=0}^{\bar{\tau}} (\zeta_k - \zeta_{k+1})(1 - \mathcal{R}_0 y_*)^{\tilde{\tau}-2-k} \\
W_{*,\tau} &= y_* \sum_{k=0}^{\min(\tau-1,\bar{\tau})} (\zeta_k - \zeta_{k+1})(1 - \mathcal{R}_0 y_*)^{\tau-1-k} \qquad \text{for } \tau \in \{1, \cdots, \tilde{\tau}-1\} \\
W_{*,\tilde{\tau}} &= \frac{1}{\mathcal{R}_0} + y_* \sum_{\tau=1}^{\bar{\tau}} \zeta_\tau - (\tilde{\tau}-1)y_* \sum_{k=0}^{\bar{\tau}} (\zeta_k - \zeta_{k+1})(1 - \mathcal{R}_0 y_*)^{\tilde{\tau}-2-k} \\
&\quad - \mathcal{R}_0 y_*^2 \sum_{\tau=2}^{\tilde{\tau}-1} \sum_{j=\tau}^{\tilde{\tau}-1} \sum_{k=0}^{\min(j-2,\bar{\tau})} (\zeta_k - \zeta_{k+1})(1 - \mathcal{R}_0 y_*)^{j-2-k}.
\end{aligned}
\tag{3.12}
$$

In this case, the endemic equilibrium is uniquely defined. Proceeding, we perform stability analysis for the simplest case ($\tilde{\tau} = 3$, $\overline{\tau} = 1$ and $\overline{\theta} = 1$). Here, $\mathcal{R}_0 = (1 - \chi)(1 - \alpha)(\xi_0\phi_0 + \xi_1\phi_1)$. Equation 3.10 for y_* simplifies to $y_* = \frac{\mathcal{R}_0 - 1}{\mathcal{R}_0(2 + \phi_1 + \zeta_1)}$ and the endemic equilibrium (equation 3.12) is

$$
\begin{aligned}
&y_{*,2} = \mathcal{R}_0 y_*^2 (\zeta_0 - \zeta_1) \\[4pt]
&y_{*,3} = y_* \left[(\zeta_0 - \zeta_1)(1 - \mathcal{R}_0 y_*) + \zeta_1 \right] \\[4pt]
&W_{*,1} = y_*(\zeta_0 - \zeta_1) \\[4pt]
&W_{*,2} = y_{*,3} = y_* \left[(\zeta_0 - \zeta_1)(1 - \mathcal{R}_0 y_*) + \zeta_1 \right] \\[4pt]
&W_{*,3} = \frac{1}{\mathcal{R}_0} + y_* \zeta_1 - 2y_* \left[(\zeta_0 - \zeta_1)(1 - \mathcal{R}_0 y_*) + \zeta_1 \right] - \mathcal{R}_0 y_*^2 (\zeta_0 - \zeta_1).
\end{aligned}
\tag{3.13}
$$

Performing elementary column operations on $J - \lambda I$ (where J is the Jacobian for the model equations evaluated at the endemic equilibrium), we get the following characteristic equation:

$$
\lambda^5 \left(\lambda^4 + \sum_{s=0}^{3} k_s \lambda^s \right) = 0,
\tag{3.14}
$$

where

$$
\begin{aligned}
&k_3 = (1 - \alpha)(1 - \chi)\left[-\frac{1}{\mathcal{R}_0}\sigma_0 + y_*(\sigma_0 + \sigma_1) \right] \\[4pt]
&k_2 = (1 - \alpha)(1 - \chi)\left[-\frac{1}{\mathcal{R}_0}\sigma_1 + y_*(\sigma_0 + \sigma_1) \right] \\[4pt]
&k_1 = [\phi_1 + \zeta_1(1 - \phi_1)]\mathcal{R}_0 y_* \\[4pt]
&k_0 = \zeta_1 \phi_1 \mathcal{R}_0 y_*.
\end{aligned}
\tag{3.15}
$$

Here, we have used the short-form $\sigma_\theta = \xi_\theta\phi_\theta$. Using Theorem B.6, we obtain sufficient conditions for the endemic equilibrium to be asymptotically stable. Depending on the sign of k_2 and k_3, there are four different cases to consider:

1. For $k_2 < 0$ and $k_3 > 0$, i.e., $1 + \frac{2+\phi_1+\zeta_1}{\sigma_0+\sigma_1}\sigma_0 < \mathcal{R}_0 < 1 + \frac{2+\phi_1+\zeta_1}{\sigma_0+\sigma_1}\sigma_1$,

   the condition for stability is $\mathcal{R}_0 < 1 + \frac{2\sigma_0(2+\phi_1+\zeta_1)}{(\sigma_0+\sigma_1)(\phi_1+\zeta_1)}$.

2. For $k_2 > 0$ and $k_3 < 0$, i.e., $1 + \frac{2+\phi_1+\zeta_1}{\sigma_0+\sigma_1}\sigma_1 < \mathcal{R}_0 < 1 + \frac{2+\phi_1+\zeta_1}{\sigma_0+\sigma_1}\sigma_0$,

   the condition for stability is $\mathcal{R}_0 < 1 + \frac{2\sigma_1(2+\phi_1+\zeta_1)}{(\sigma_0+\sigma_1)(\phi_1+\zeta_1)}$.

3. For $k_2 > 0$ and $k_3 > 0$, i.e., $\mathcal{R}_0 > 1 + \frac{2+\phi_1+\zeta_1}{\sigma_0+\sigma_1}\max(\sigma_0, \sigma_1)$,

   the condition for stability is $\mathcal{R}_0 < 3$.

4. For $k_2 < 0$ and $k_3 < 0$, i.e., $\mathcal{R}_0 < 1 + \frac{2+\phi_1+\zeta_1}{\sigma_0+\sigma_1}\min(\sigma_0, \sigma_1)$,

   the condition for stability is $\phi_1 + \zeta_1 < 2$.

Using Lemma B.7, the necessary conditions for the endemic equilibrium to be asymptotically stable are:

1. $\mathcal{R}_0 < 1 + \frac{2+\phi_1+\zeta_1}{\zeta_1\phi_1}$

2. $\mathcal{R}_0 < 1 + \frac{2\sigma_0(2+\phi_1+\zeta_1)}{(\sigma_0+\sigma_1)(\phi_1+\zeta_1-2\zeta_1\phi_1)}$ if $\zeta_1\phi_1 < \frac{1}{2}(\phi_1+\zeta_1)$

   $\mathcal{R}_0 > 1 + \frac{2\sigma_0(2+\phi_1+\zeta_1)}{(\sigma_0+\sigma_1)(\phi_1+\zeta_1-2\zeta_1\phi_1)}$ if $\zeta_1\phi_1 > \frac{1}{2}(\phi_1+\zeta_1)$.

Unlike the case for the disease-free equilibrium, the sufficient and necessary conditions here do not together provide equivalence conditions for asymptotic stability. As such, we do not have explicit thresholds for the solutions to be in the endemic equilibrium regime. This is similar to models with varying delays in continuous-time, in which sufficient conditions for the endemic equilibrium regime are obtained (Stech and Williams, 1981 [23]) or numerical bifurcation analysis is performed (Blyuss and Kyrychko, 2010 [24]).

4. Sub-model with fixed waning and recovery times

We consider the case where every individual follows exactly the same waning and recovery trajectory. Infected individuals join the immune compartment on day $\bar{\theta} + 1$ since infection and recovered individuals join the waned compartment on day $\bar{\tau} + 1$ since recovery. Similar to how classic SIRS is formulated, we consider the simplest model by dropping the dependence on time since infection θ and time since recovery τ, arriving at a constant value of infectiousness ξ, reduction in susceptibility α and reduction in infectiousness χ. The RWY model (equation 2.1) becomes:

$$R_{t+1} = R_t + y_{t-\bar{\theta}} - y_{t-\bar{\tau}-\bar{\theta}-1}$$

$$W_{t+1} = W_t - y_{t+1} + y_{t-\bar{\tau}-\bar{\theta}-1}$$

$$y_{t+1} = \beta W_t \sum_{\theta=0}^{\bar{\theta}} y_{t-\theta},$$

(4.1)

where $\beta = \xi(1-\chi)(1-\alpha)$. Note that the proportions of the population in the sub-compartments $y_{t+1,\tau}$ and $W_{t+1,\tau}$ are no longer needed to be considered individually as they have been summed up. In addition, from the RWY model (equation 2.1), we get $R_{t+1,0} = y_{t-\bar{\theta}}$ and $R_t = \sum_{\theta=\bar{\theta}+1}^{\bar{\theta}+\bar{\tau}+1} y_{t-\theta}$. Substituting the latter expression and $W_t = 1 - R_t - Y_t$ into the last equation of (4.1), we obtain a non-linear scalar equation:

$$y_{t+1} = \beta \sum_{\theta=0}^{\bar{\theta}} y_{t-\theta} \left(1 - \sum_{\theta=0}^{\bar{\theta}+\bar{\tau}+1} y_{t-\theta} \right). \tag{4.2}$$

We linearize about the disease-free equilibrium by setting $y_t = \epsilon\lambda^t$, where ϵ is small, to get the following characteristic equation:

$$\lambda^{\bar{\theta}+1} - \beta \sum_{s=0}^{\bar{\theta}} \lambda^s = 0. \tag{4.3}$$

By Theorem B.6 and Lemma B.7, the disease-free equilibrium is asymptotically stable if and only if $\mathcal{R}_0 < 1$, where $\mathcal{R}_0 = \beta(\bar{\theta} + 1)$. The endemic equilibrium can be found using equation (4.2) to

be $y_* = \frac{\mathcal{R}_0 - 1}{\mathcal{R}_0(\bar{\theta} + \bar{\tau} + 2)}$, which is uniquely defined. Linearizing about the endemic equilibrium, we get the following characteristic equation:

$$\lambda^{t+1} = \frac{2\bar{\theta} + \bar{\tau} + 3 - \beta(\bar{\theta} + 1)^2}{(\bar{\theta} + 1)(\bar{\theta} + \bar{\tau} + 2)} \sum_{\theta=0}^{\bar{\theta}} \lambda^{t-\theta} - \frac{\beta(\bar{\theta} + 1) - 1}{\bar{\theta} + \bar{\tau} + 2} \sum_{\theta=\bar{\theta}+1}^{\bar{\theta}+\bar{\tau}+1} \lambda^{t-\theta}. \tag{4.4}$$

Applying Theorem B.6, the sufficient conditions for asymptotic stability are:

$$1 < \mathcal{R}_0 < 3 \qquad \text{and} \qquad \bar{\theta} > \bar{\tau}. \tag{4.5}$$

The second condition requires the infectious period to be greater than the immune period for stability. This agrees with what other continuous-time models have found (Diekmann and Montijn, 1982 [15], Hethcote et al. (1981) [14] and Stech and Williams (1981) [23]): a long immune period can lead to instability of the endemic equilibrium. On the other hand, applying Lemma B.7, the necessary conditions for asymptotic stability are given by:

$$1 < \mathcal{R}_0 < \bar{\theta} + \bar{\tau} + 3. \tag{4.6}$$

Note that $\bar{\theta} + \bar{\tau} + 3$ is the minimum amount of time an individual spends going through the R ($\bar{\tau} + 1$ days), W (1 day) and Y ($\bar{\theta} + 1$ days) compartments before returning to the R compartment.

To obtain explicit thresholds, we consider two special cases. Case 1: If the immune period is one day ($\bar{\tau} = 0$), then using Theorem B.5, the endemic equilibrium is asymptotically stable if and only if equation (4.6) holds. Case 2: If the infectious period is one day ($\bar{\theta} = 0$), then we use the Determinantal Criterion directly (Theorem B.3) for $\bar{\tau} = 1, 2$ and 3 to get $1 < \mathcal{R}_0 < \frac{5}{2}$, $1 < \mathcal{R}_0 < 5 - 2\sqrt{2}$ and $1 < \mathcal{R}_0 < \frac{19 - 5\sqrt{5}}{4}$ respectively.

We perform bifurcation analysis for these two cases. For case 1 ($\bar{\tau} = 0$), at the critical value $\mathcal{R}_0 = \bar{\theta} + 3$, the endemic equilibrium loses stability. There are two sub-cases.

1. For $\bar{\theta}$ odd, setting $\bar{\theta} = 2n + 1$, the characteristic polynomial is $\lambda^{2n+3} + \frac{2n+1}{2n+2} \sum_{j=1}^{2n+2} \lambda^j + 1$. We apply Theorem (B.8) with $a = \frac{2n+1}{2n+2}$, and find that all eigenvalues lie on the unit circle. One of the eigenvalues is -1 and there are $(n + 1)$ complex conjugage pairs. A flip (also known as period doubling) and Neimark-Sacker bifurcation occur concurrently.

2. For $\bar{\theta}$ even, setting $\bar{\theta} = 2n$, the characteristic polynomial is $\lambda^{2n+2} + \frac{2n}{2n+1} \sum_{j=1}^{2n+1} \lambda^j + 1$. Again, applying Theorem (B.8) with $a = \frac{2n}{2n+1}$, all eigenvalues lie on the unit circle and there are $(n + 1)$ complex conjugate pairs. A Neimark-Sacker bifurcation occurs.

   (a) If $\bar{\theta} = 0$, the eigenvalues are $\pm i$, which leads to a $1 : 4$ (also known as period 4) resonance, for which the constants b and d in the complex normal form can be computed using equations (C.11) and (C.12) to be $\frac{9}{4} - \frac{9i}{8}$ and $-\frac{9i}{8}$ respectively.

For case 2 ($\bar{\theta} = 0$), for $\bar{\tau} = 1$ to 3, at the critical value of $\mathcal{R}_0$, we have a pair of complex conjugate eigenvalues with modulus 1, leading to a Neimark-Sacker bifurcation. There is no strong resonance for these eigenvalues found. The invariant expression d can be computed using equation (C.9) and they are all negative, which means that a stable closed invariant curve bifurcates from the fixed point for $\mathcal{R}_0$ greater than the critical value.

Our analysis above for the RWY sub-model with fixed recovery and waning times implies that there are three dynamic regimes - (i) disease-free equilibrium, (ii) endemic equilibrium and (iii) oscillatory solutions due to flip or Neimark-Sacker bifurcations.

5. Sub-model with geometric distributions

We now consider geometric distributions for both recovery and waning. At the individual level, on each day, an infectious individual has a probability γ of recovering and a recovered individual has a probability ω of joining the waned compartment. At the population level, the fraction still infectious is $\phi_\theta = (1 - \gamma)^\theta$ and the fraction still immune is $\zeta_\tau = (1 - \omega)^\tau$. The R equations can thus be expressed as:

$$R_{t+1} = \sum_{\tau=0}^{\infty} (1 - \omega)^{\tau+1} R_{t-\tau,0} + \gamma \sum_{\theta=0}^{\infty} (1 - \gamma)^\theta y_{t-\theta}. \tag{5.1}$$

Note that both the immune period and the infectious period are infinite days long. Furthermore, if we assume that the infectiousness ξ, reduction in susceptibility α and reduction in infectiousness χ are constants, then the RWY model (using equations 2.1, 2.2 and 5.1) becomes:

$$R_{t+1} = (1 - \omega)R_t + \gamma Y_t$$

$$W_{t+1} = W_t + \omega R_t - \beta W_t Y_t \tag{5.2}$$

$$Y_{t+1} = (1 - \gamma)Y_t + \beta W_t Y_t,$$

where the transmission parameter $\beta = \xi(1 - \chi)(1 - \alpha)$. Using the property of geometric distributions, we note that the mean number of days an individual stays in the infectious and immune compartments is $\frac{1}{\gamma}$ and $\frac{1}{\omega}$ respectively. Such a model with geometric distributions is often described as 'memoryless' because the probabilities that an infectious individual remains infectious and an immune individual remains immune on a particular day do not depend on the number of days they have already spent in the infectious and immune compartments respectively.

Substituting $R_t = 1 - Y_t - W_t$ and $W_t = \frac{1}{\beta Y_t}[Y_{t+1} - (1 - \gamma)Y_t]$ into the second equation of (5.2), we obtain a scalar nonlinear difference equation:

$$Y_{t+1} = \left[(1 - \omega)\frac{Y_t}{Y_{t-1}} - \beta Y_t + \omega(1 - \gamma + \beta) + \beta(1 - \gamma - \omega)Y_{t-1}\right] Y_t. \tag{5.3}$$

We linearize about the disease-free equilibrium by letting $Y_t = \epsilon \lambda^t$, where ϵ is small, and obtain the characteristic equation $\omega[\lambda - (1 - \gamma + \beta)] = 0$. The disease-free equilibrium is asymptotically stable if and only if $\mathcal{R}_0 < 1$, where $\mathcal{R}_0 = \frac{\beta}{\gamma}$.

From equation (5.3), the endemic equilibrium can be found to be $Y_* = \frac{\omega(\beta-\gamma)}{\beta(\omega+\gamma)}$, which is uniquely defined. Linearizing about the endemic equilibrium, we get the characteristic equation $\lambda^2 + p_1\lambda + p_0 = 0$, where $p_1 = -2 + \omega + \frac{\omega(\beta-\gamma)}{\omega+\gamma}$ and $p_0 = 1 - \omega + \omega(\beta - \gamma)(\frac{\omega+\gamma-1}{\omega+\gamma})$. Using the Determinantal Criterion for order 2 (equation B.2), the endemic equilibrium is asymptotically stable if and only if

$$1 < \mathcal{R}_0 < 1 + q, \tag{5.4}$$

where

$$q = \begin{cases} \frac{\gamma+\omega}{\gamma(\gamma+\omega-1)} & \text{if} \quad 0 < \omega < 1 \quad \text{and} \quad \frac{4-4\omega+\omega^2}{4-\omega} < \gamma < 1 \\ \frac{2(2-\omega)(\gamma+\omega)}{\gamma\omega(2-\gamma-\omega)} & \text{if} \quad 0 < \omega < 1 \quad \text{and} \quad 0 < \gamma < \frac{4-4\omega+\omega^2}{4-\omega}. \end{cases} \tag{5.5}$$

At the critical value $\mathcal{R}_0^* = 1 + \frac{2(2-\omega)(\gamma+\omega)}{\gamma\omega(2-\gamma-\omega)}$, the eigenvalues are -1 and $-\frac{2+(\gamma+\omega)(-3+\omega)}{-2+\gamma+\omega}$. The latter eigenvalue cannot take the value 1 or -1 under the constraints for γ and ω. Hence, the Jacobian J has a simple critical eigenvalue -1 and a flip bifurcation occurs. The invariant formula b in the normal form of a flip bifurcation (equation C.6) can be calculated using the formula (equation C.7) to be

$$b = \frac{(-2+\gamma+\omega)^3[\gamma(-1+\omega)+(-2+\omega)\omega][\gamma^2\omega+2(-2+\omega)\omega+\gamma(-4+\omega^2)]^2}{[\gamma(-4+\omega)+(-2+\omega)^2]^3(-2+\omega)^2\omega^2(\gamma+\omega)}. \tag{5.6}$$

Under the constraints for γ and ω, b is always greater than 0, which means that a stable period two cycle bifurcates from the fixed point for $\mathcal{R}_0 > \mathcal{R}_0^*$.

On the other hand, at the critical value $\mathcal{R}_0^* = 1 + \frac{\gamma+\omega}{\gamma(\gamma+\omega-1)}$, the eigenvalues are

$$\frac{-2-\gamma(-2+\omega)+2\omega-\omega^2 \pm i\sqrt{\omega[\gamma^2(4-\omega)-(-2+\omega)^2\omega+2\gamma(-2+4\omega-\omega^2)]}}{2(-1+\gamma+\omega)}, \tag{5.7}$$

which have modulus one under the constraints for γ and ω. Thus, a Neimark-Sacker bifurcation occurs at the critical value $\mathcal{R}_0^*$.

Like the RWY sub-model with fixed recovery and waning times, our analysis here for the sub-model with geometric distributions in discrete-time implies that there are three dynamic regimes - (i) the disease-free equilibrium, (ii) the endemic equilibrium and (iii) oscillatory solutions due to flip or Neimark-Sacker bifurcation.

6. Sub-model with exponential distributions

We take the limit as time step tends to zero for the discrete-time sub-model with geometric distributions (equation 5.2), to get the continuous-time RWY model with exponential distributions:

$$\frac{dR}{dt} = -\omega R + \gamma Y$$

$$\frac{dW}{dt} = \omega R - \beta WY \tag{6.1}$$

$$\frac{dY}{dt} = -\gamma Y + \beta WY,$$

where β is the transmission rate per infectious individual, ω is the waning rate and γ is the recovery rate. We present the following analysis in the same manner as earlier sections of the paper, and refer the reader to Hethcote, 1976 [13] for an alternative derivation of these results.

The disease-free equilibrium is given by: $R_* = 0$, $W_* = 1$ and $Y_* = 0$. It is asymptotically stable if and only if $\mathcal{R}_0 < 1$, where $\mathcal{R}_0 = \frac{\beta}{\gamma}$.

The endemic equilibrium is given by: $R_* = \frac{\gamma(\beta-\gamma)}{\beta(\omega+\gamma)}$, $W_* = \frac{\gamma}{\beta}$ and $Y_* = \frac{\omega(\beta-\gamma)}{\beta(\omega+\gamma)}$, which is uniquely defined. Note that the expression for the endemic equilibrium in continuous-time is the same as that in discrete-time. The eigenvalues of the Jacobian matrix are

$$\lambda_\pm = \frac{1}{2}\left[-\breve{\omega} \pm \sqrt{\breve{\omega}^2 - 4\omega\gamma(\mathcal{R}_0 - 1)}\right], \tag{6.2}$$

where $\breve{\omega} = \omega\left(\frac{\frac{\omega}{\gamma} + \mathcal{R}_0}{\frac{\omega}{\gamma} + 1}\right)$. The endemic equilibrium is asymptotically stable if and only if both eigenvalues are negative, which occurs if and only if $\mathcal{R}_0 > 1$. Hence, the RWY sub-model with exponential distributions in continuous-time only has two dynamic regimes: the disease-free equilibrium and the endemic equilibrium. No oscillatory solution is possible.

7. Bridging between discrete-time and continuous-time sub-models with geometric distributions

In this section, we seek to understand how the dynamic regimes change when we transit from discrete-time to continuous-time. The discrete-time sub-model with geometric distributions with time step h is:

$$R_{t+1} = (1 - h\omega)R_t + h\gamma Y_t$$

$$W_{t+1} = W_t + h\omega R_t - h\beta W_t Y_t \qquad (7.1)$$

$$Y_{t+1} = (1 - h\gamma)Y_t + h\beta W_t Y_t,$$

which is almost identical to the discrete-time sub-model with geometric distributions (equation 5.2), except that each of the parameters ω, β and γ is scaled by h. By taking into account this factor h in previous results for the discrete-time model with a time step of one day, we see that as before, $\mathcal{R}_0 = \frac{\beta}{\gamma}$ and the disease-free equilibrium is asymptotically stable if and only if $\mathcal{R}_0 < 1$. The endemic equilibrium also remains the same: $R_* = \frac{\gamma(\beta - \gamma)}{\beta(\omega + \gamma)}$, $W_* = \frac{\gamma}{\beta}$ and $Y_* = \frac{\omega(\beta - \gamma)}{\beta(\omega + \gamma)}$. It is asymptotically stable if and only if $1 < \mathcal{R}_0 < 1 + q_h$, where

$$q_h = \begin{cases} \frac{\gamma + \omega}{\gamma(h\gamma + h\omega - 1)} & \text{if} \quad 0 < \omega < \frac{1}{h} \quad \text{and} \quad \frac{4 - 4h\omega + h^2\omega^2}{h(4 - h\omega)} < \gamma < \frac{1}{h} \\ \frac{2(2 - h\omega)(\gamma + \omega)}{h\gamma\omega(2 - h\gamma - h\omega)} & \text{if} \quad 0 < \omega < \frac{1}{h} \quad \text{and} \quad 0 < \gamma < \frac{4 - 4h\omega + h^2\omega^2}{h(4 - h\omega)}. \end{cases} \qquad (7.2)$$

In the limit as h tends to zero, the top case in equation 7.2 collapses and q_h tends to infinity. Hence, the endemic equilibrium is asymptotically stable if and only if $\mathcal{R}_0 > 1$ and the last regime with flip/Neimark-Sacker bifurcation in discrete-time disappears (see Figure 3). The number of dynamic regimes decreases from three to two and oscillatory solutions are no longer possible.

8. Discussion

We have intentionally chosen to focus on waning immunity to improve our understanding of the underlying mechanism behind the recent waves of COVID-19 infections in this ongoing pandemic. The emergence of new variants has often been invoked as the reason causing waves of surges in case numbers. However, our work shows that the waning of prior immunity, whether that immunity is from infection or vaccine, is sufficient on its own to drive oscillations. Hence, at least in theory, pathogen evolution may be purely consequential rather the cause of these waves.

In practice, COVID-19 waves are unlikely to be driven by a single mechanism. The effects of phylodynamics (Grenfell et al., 2004 [25]) are complex and there is likely a feedback loop between waning immunity dynamics and pathogen evolution. However, we argue that for diseases with evidence of waning immunity, the loss of immunity should be considered to be a candidate for the main mechanism driving oscillatory dynamics. This is particularly true for diseases with a short temporary
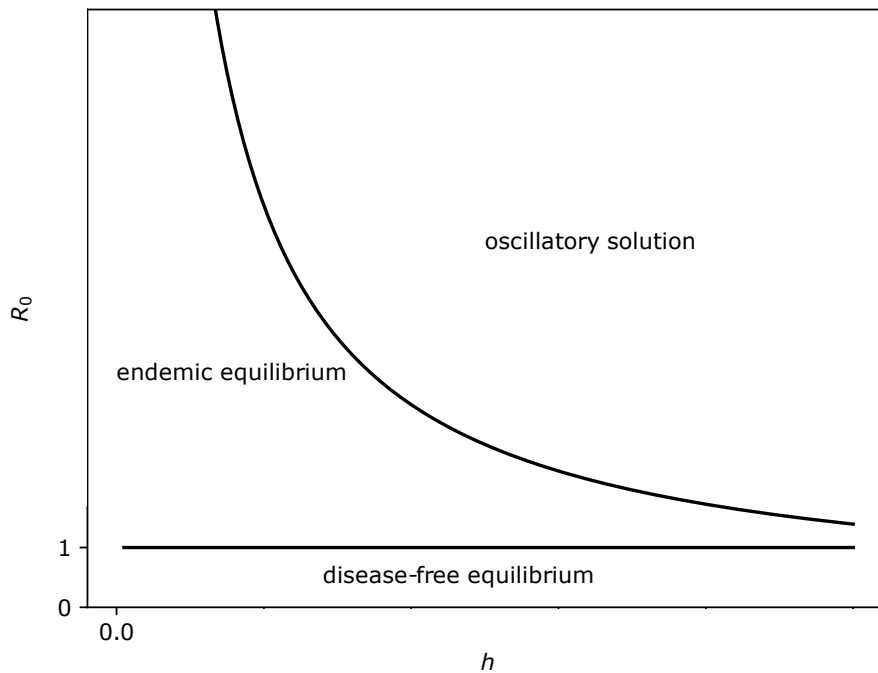
Figure 3. Schematic of the stable long term dynamic regimes for the discrete-time Immune-Waned-Infectious (RWY) model with geometric distributions. As the time step h tends to zero, this model tends to its continuous classic SIRS and the regime with sustained oscillations disappears.

period of strong protection like COVID-19, where parsimonious waning immunity models can be used to explain the key trends of observed oscillations in case numbers.

From the public health point of view, policy makers may want to know whether surges in case numbers will happen in the long term for endemic diseases with waning immunity. Surges in case numbers are particularly problematic if the disease has severe health outcomes, especially when healthcare capacity is a major constraint. Models can help by distinguishing between two model outcomes for endemic diseases – the endemic equilibrium and sustained oscillations. The former does not lead to long term surges in case numbers but the latter does. If waning immunity plays a role in sustaining oscillations, then understanding the population infection dynamics can help policy makers decide the timing of booster campaigns to reduce the size of future case surges.

Acknowledgements

Use of AI tools declaration

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

Conflict of interest

All authors declare that there is no conflict of interest.

References

1. P. E. M. Fine, J. A. Clarkson, Measles in England and Wales—-I: An Analysis of Factors Underlying Seasonal Patterns, *Int. J. Epidemiol.*, **11** (1982), 5–14. https://doi.org/10.1093/ije/11.1.5

2. J. L. Aron, I. B. Schwartz, Seasonality and Period-doubling Bifurcations in an Epidemic Model, *J. Theor. Biol.*, **110** (1984), 665–679. https://doi.org/10.1016/s0022-5193(84)80150-2

3. D. J. D. Earn, P. Rohani, B. M. Bolker, B. T. Grenfell, A Simple Model for Complex Dynamical Transitions in Epidemics, *Science*, **287** (2000), 667–670. https://doi.org/10.1126/science.287.5453.667

4. V. Andreasen, Dynamics of annual influenza A epidemics with immuno-selection, *J. Math. Biol.*, **46** (2003), 504–536. https://doi.org/10.1007/s00285-002-0186-2

5. J. Dushoff, J. B. Plotkin, S. A. Levin, D. J. D. Earn, Dynamical resonance can account for seasonality of influenza epidemics, *Proceed. Nat. Acad. Sci.*, **101** (2004), 16915–16916. https://doi.org/10.1073/pnas.0407293101

6. A. d'Onofrio, P. Manfredi, Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious diseases, *J. Theor. Biol.*, **256** (2009), 473–478. https://doi.org/10.1016/j.jtbi.2008.10.005

7. H. A. L. R. Silva, L. H. A. Monteiro, Self-sustained oscillations in epidemic models with infective immigrants, *Ecolog. Complex.*, **17** (2014), 40–45. https://doi.org/10.1016/j.ecocom.2013.08.002

8. T. Kuniya, H. Inaba, Hopf bifurcation in a chronological age-structured SIR epidemic model with age-dependent infectivity, *Math. Biosci. Eng.*, **20** (2023), 13036–13060. https://doi.org/10.3934/mbe.2023581

9. A. P. Baccili, L. H. A. Monteiro, Social Pressure from a Core Group can Cause Self-Sustained Oscillations in an Epidemic Model, *Acta Biotheor.*, **71** (2023). https://doi.org/10.1007/s10441-023-09469-3

10. S. Burkholz, M. Rubsamen, L. Blankenberg, R. T. Carback III, D. Mochly-Rosen, P. E. Harris, Analysis of well-annotated next-generation sequencing data reveals increasing cases of SARS-CoV-2 reinfection with Omicron, *Commun. Biol.*, **6** (2023). https://doi.org/10.1038/s42003-023-04687-4

11. N. Bobrovitz, H. Ware, X. Ma, Z. Li, R. Hosseini, C. Cao, et al., Protective effectiveness of previous SARS-CoV-2 infection and hybrid immunity against the omicron variant and severe disease: A systematic review and meta-regression, *Lancet Infect. Diseases*, **23** (2023), 556–567. https://doi.org/10.1016/S1473-3099(22)00801-5

12. A. M. Wendelboe, A. V. Rie, S. Salmaso, J. A. Englund, Duration of Immunity Against Pertussis After Natural Infection or Vaccination, *Pediatric Infect. Disease J.*, **24** (2005), S58–S61. https://doi.org/10.1097/01.inf.0000160914.59160.41

13. H. W. Hethcote, Qualitative Analyses of Communicable Disease Models, *Math. Biosci.*, **28** (1976), 335–356. https://doi.org/10.1016/0025-5564(76)90132-2

14. H. W. Hethcote, H. W. Stech, P. Van Den Driessche Nonlinear Oscillations in Epidemic Models, *SIAM J. Appl. Math.*, **40** (1981), 1–9. https://doi.org/10.1137/0140001

15. O. Diekmann, R. Montijn, Prelude to Hopf Bifurcation in an Epidemic Model: Analysis of a Characteristic Equation Associated with a Nonlinear Volterra Integral Equation, *J. Math. Biol.*, **14** (1982), 117–127. https://doi.org/10.1007/BF02154757

16. Y. N. Kyrychko, K. B. Blyuss, Global properties of a delayed SIR model with temporary immunity and nonlinear incidence rate, *Nonlinear Anal. Real World Appl.*, **6** (2005), 495–507. https://doi.org/10.1016/j.nonrwa.2004.10.001

17. C. M. Pease, An evolutionary epidemiological mechanism, with applications to type A influenza, *Theor. Popul. Biol.*, **31** (1987), 422–452. https://doi.org/10.1016/0040-5809(87)90014-1

18. W. O. Kermack, A. G. McKendrick, Contributions to the mathematical theory of epidemics. II. – The problem of endemicity, *Proceed. Royal Soc. London. Series A Containing Papers Math. Phys. Character*, **138** (1932), 55–83. https://doi.org/10.1098/rspa.1932.0171

19. H. Inaba, Kermack and McKendrick revisited: The variable susceptibility model for infectious diseases, *Japan J. Industr. Appl. Math.*, **18** (2001), 273–292. https://doi.org/10.1007/BF03168575

20. W. O. Kermack, A. G. McKendrick, A Contribution to the Mathematical Theory of Epidemics, in *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character*, **115** (1927), 700–721. https://doi.org/10.1098/rspa.1927.0118

21. N. Hernandez-Ceron, Z. Feng, C. Castillo-Chavez, Discrete epidemic models with arbitrary stage distributions and applications to disease control, *Bull. Math. Biol.*, **75** (2013), 1716—1746. https://doi.org/10.1007/s11538-013-9866-x

22. O. Diekmann, H. G. Othmer, R. Planqué, M. C. J. Bootsma, The discrete-time Kermack–McKendrick model: A versatile and computationally attractive framework for modeling epidemics, *PNAS*, **118** (2021). https://doi.org/10.1073/pnas.2106332118

23. H. Stech, M. Williams, Stability in a Class of Cyclic Epidemic Models with Delay, *J. Math. Biol.*, **11** (1981), 95–103. https://doi.org/10.1007/BF00275827

24. K. B. Blyuss, Y. N. Kyrychko, Stability and Bifurcations in an Epidemic Model with Varying Immunity Period, *Bull. Math. Biol.*, **72** (2010), 490–505. https://doi.org/10.1007/s11538-009-9458-y

25. B. T. Grenfell, O. G. Pybus, J. R. Gog, J. L. N. Wood, J. M. Daly, J. A. Mumford, E. C. Holmes, Unifying the epidemiological and evolutionary dynamics of pathogens, *Science*, **303** (2004), 327–332. https://doi.org/10.1126/science.1090727

26. E. I. Jury, "Inners" Approach to Some Problems of System Theory, *IEEE*, **16** (1971), 233–240. https://doi.org/10.1109/TAC.1971.1099725

27. E. I. Jury, *Inners and Stability of Dynamic Systems*, John Wiley and Sons, Inc., United States of America, 1974.

28. S. Elaydi, *An Introduction to Difference Equations*, 3rd edition, Springer-Verlag, New York, 2005. https://doi.org/10.1007/0-387-27602-5

29. E. I. Jury, *Theory and Application of the z-Transform Method*, John Wiley and Sons, Inc., United States of America, 1964.

30. P. Tomášek, Asymptotic stability of a full term linear difference equation with two parameters, *Tatra Mountains Math. Publicat.*, **63** (2015), 283–290.

31. Y. Bistritz, Zero Location with Respect to the Unit Circle of Discrete-Time Linear System Polynomials, *Proceed. IEEE*, **72** (1984), 1131–1142. https://doi.org/10.1109/PROC.1984.12993

32. V. Botta, L. F. Marques, M. Meneguette, Palindromic and perturbed polynomials: Zeros location, *Acta Math. Hungar.*, **143** (2014), 81–87. https://doi.org/10.1007/s10474-013-0382-0

33. Y. A. Kuznetsov, *Elements of Applied Bifurcation Theory*, 3rd edition, Springer, New York, 2004. https://doi.org/10.1007/978-1-4757-3978-7
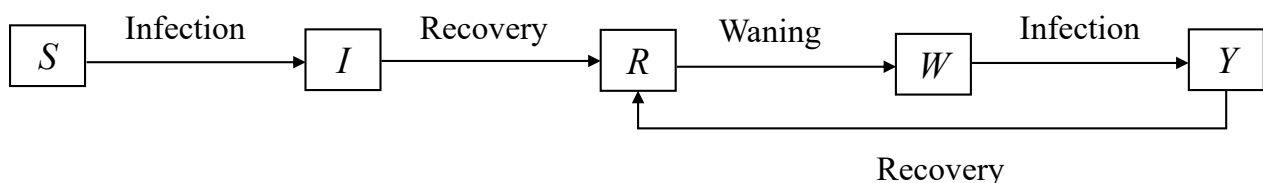
Appendix A   The SIRWY model

Figure 4. Susceptible-Infectious-Immune-Waned-Infectious (SIRWY) model.

The SIRWY model (see Figure 4) is an extension to the RWY model as it takes into account individuals in the population who do not have prior immunity to the disease. These individuals belong to either the susceptible (S) or infectious (I) compartment. The proportion of population that is infectious at time t is denoted by $I_t = \sum_{\theta=0}^{\bar{\theta}} \breve{\phi}_\theta i_{t-\theta}$, which is defined in an analogous manner as its counterpart Y_t with prior immunity. Table 3 gives a list of the variables, indices and parameters used in SIRWY model, comparing terms with/without prior immunity whenever possible. The SIRWY model is described by

Table 3. Variables, indices and parameters of the SIRWY model.

Without	With	Terms without/with prior immunity	Symbol	Other terms
$\check{f}$	f	force of infection	F	total force of infection
S	W	susceptible	R	immune
i	y	newly infected	θ	time since infection
I	Y	infectious	$\bar{\theta}+1$	max infectious period
$\check{\phi}_\theta$	ϕ_θ	fraction still infectious	τ	time since recovery
$\check{\xi}_\theta$	ξ_θ	infectiousness	$\bar{\tau}+1$	max immune period
			$\tilde{\tau}$	max value of τ
			ζ_τ	fraction still immune
			χ_τ	reduction in infectiousness
			α_τ	reduction in susceptibility

the following system of difference equations,

$$
\begin{aligned}
F_t &= \check{f}_t + f_t = \sum_{\theta=0}^{\bar{\theta}} \check{\xi}_\theta \check{\phi}_\theta i_{t-\theta} + \sum_{\theta=0}^{\bar{\theta}} \sum_{\tau=2}^{\tilde{\tau}} \xi_\theta \phi_\theta y_{t-\theta,\tau}(1 - \chi_\tau) \\
S_{t+1} &= S_t - i_{t+1} \\
i_{t+1} &= S_t F_t
\end{aligned}
$$

$$
\begin{aligned}
R_{t+1,0} &= \sum_{\theta=0}^{\bar{\theta}} (\check{\phi}_\theta - \check{\phi}_{\theta+1}) i_{t-\theta} + \sum_{\theta=0}^{\bar{\theta}} (\phi_\theta - \phi_{\theta+1}) y_{t-\theta} \\
R_{t+1,\tau} &= \zeta_\tau R_{t-(\tau-1),0} && \text{for } \tau \in \{1, \cdots, \bar{\tau}\}
\end{aligned}
$$

$$
\begin{aligned}
W_{t+1,1} &= (\zeta_0 - \zeta_1) R_{t,0} \\
W_{t+1,\tau} &= W_{t,\tau-1} - y_{t+1,\tau} + (\zeta_{\tau-1} - \zeta_\tau) R_{t-(\tau-1),0} && \text{for } \tau \in \{2, \cdots, \bar{\tau}+1\} \\
W_{t+1,\tau} &= W_{t,\tau-1} - y_{t+1,\tau} && \text{for } \tau \in \{\bar{\tau}+2, \cdots, \tilde{\tau}-1\} \\
W_{t+1,\tilde{\tau}} &= W_{t,\tilde{\tau}-1} + W_{t,\tilde{\tau}} - y_{t+1,\tilde{\tau}}
\end{aligned}
$$

$$
\begin{aligned}
y_{t+1,\tau} &= (1 - \alpha_{\tau-1}) W_{t,\tau-1} F_t && \text{for } \tau \in \{2, \cdots, \tilde{\tau}-1\} \\
y_{t+1,\tilde{\tau}} &= [(1 - \alpha_{\tilde{\tau}-1}) W_{t,\tilde{\tau}-1} + (1 - \alpha_{\tilde{\tau}}) W_{t,\tilde{\tau}}] F_t
\end{aligned}
$$

$$(\text{A.1})$$

and the conservation of individuals means that

$$
S_t + I_t + R_t + W_t + Y_t = 1. \tag{A.2}
$$

The I, R, W and Y compartments can be defined in terms of their corresponding sub-compartments (see Table 4). Similar to the RWY model, the SIRWY model is specified using the proportion of newly infecteds (i and y) rather than the proportion of infectious individuals (I and Y).

Table 4. Definition of variables in the SIRWY model. The indices t, τ and θ refer to the time, time since recovery and time since infection respectively. The summations correspond to the max infectious period ($\bar{\theta}+1$), the max immune period ($\bar{\tau}+1$) and the max value of τ ($\tilde{\tau}$).

Symbol	Variable	Composition
I_t	infectious (without prior immunity)	$I_t = \sum_{\theta=0}^{\bar{\theta}} \check{\phi}_\theta i_{t-\theta}$
R_t	immune	$R_t = \sum_{\tau=0}^{\bar{\tau}} R_{t,\tau}$ or $R_t = \sum_{\tau=0}^{\bar{\tau}} \zeta_\tau R_{t-\tau,0}$
W_t	waned	$W_t = \sum_{\tau=1}^{\tilde{\tau}} W_{t,\tau}$
Y_t	infectious (with prior immunity)	$Y_t = \sum_{\tau=2}^{\tilde{\tau}} Y_{t,\tau}$ or $Y_t = \sum_{\theta=0}^{\bar{\theta}} \phi_\theta y_{t-\theta}$
y_t	newly infected (with prior immunity)	$y_t = \sum_{\tau=2}^{\tilde{\tau}} y_{t,\tau}$

Appendix B Theorems regarding zeroes of polynomial

For a kth order difference equation, we define the following characteristic polynomial:

$$p(\lambda) = \lambda^k + p_{k-1}\lambda^{k-1} + \cdots + p_1\lambda + p_0, \tag{B.1}$$

where p_i are real numbers in our case.

Definition B.1 (Inners of a matrix). *(Jury, 1971 [26], Jury, 1974 [27] and Elaydi, 2005 [28]) The inners of a matrix $B = (b_{ij})$ are the matrix itself and all the matrices obtained by sequentially removing the first and last rows and the first and last columns. For example, other than the matrix itself, the inners of the following matrices are indicated by boxes:*

$$
\begin{array}{ccc}
\textit{3 by 3 matrix} & \textit{4 by 4 matrix} & \textit{5 by 5 matrix}
\end{array}
$$

$$
\begin{pmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & \boxed{b_{22}} & b_{23} \\ b_{31} & b_{32} & b_{33} \end{pmatrix},
\begin{pmatrix} b_{11} & b_{12} & b_{13} & b_{14} \\ b_{21} & b_{22} & b_{23} & b_{24} \\ b_{31} & b_{32} & b_{33} & b_{34} \\ b_{41} & b_{42} & b_{43} & b_{44} \end{pmatrix},
\begin{pmatrix} b_{11} & b_{12} & b_{13} & b_{14} & b_{15} \\ b_{21} & b_{22} & b_{23} & b_{24} & b_{25} \\ b_{31} & b_{32} & b_{33} & b_{34} & b_{35} \\ b_{41} & b_{42} & b_{43} & b_{44} & b_{45} \\ b_{51} & b_{52} & b_{53} & b_{54} & b_{55} \end{pmatrix}.
$$

Definition B.2 (Postive Innerwise). *(Jury, 1971 [26], Jury, 1974 [27] and Elaydi, 2005 [28]) A matrix B is positive innerwise if the determinants of all of its inners are positive.*

Theorem B.3 (Determinantal Criterion, a special case of Schur-Cohn Criterion). *(Jury, 1964 [29], Jury, 1971 [26], Jury, 1974 [27] and Elaydi, 2005 [28]) The zeroes of the characteristic polynomial (equation B.1) lie inside the unit disc if and only if the following hold:*

(i) $p(1) > 0$

(ii) $(-1)^k p(-1) > 0$

(iii) The $(k-1)$ by $(k-1)$ matrices

$$B_{k-1}^{\pm} = \begin{pmatrix} 1 & 0 & 0 & \cdots & 0 & 0 & 0 \\ p_{k-1} & 1 & 0 & \cdots & 0 & 0 & 0 \\ p_{k-2} & p_{k-1} & 1 & \cdots & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ p_4 & p_5 & p_6 & \cdots & 1 & 0 & 0 \\ p_3 & p_4 & p_5 & \cdots & p_{k-1} & 1 & 0 \\ p_2 & p_3 & p_4 & \cdots & p_{k-2} & p_{k-1} & 1 \end{pmatrix} \pm \begin{pmatrix} 0 & 0 & 0 & \cdots & 0 & 0 & p_0 \\ 0 & 0 & 0 & \cdots & 0 & p_0 & p_1 \\ 0 & 0 & 0 & \cdots & p_0 & p_1 & p_2 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & p_0 & \cdots & p_{k-6} & p_{k-5} & p_{k-4} \\ 0 & p_0 & p_1 & \cdots & p_{k-5} & p_{k-4} & p_{k-3} \\ p_0 & p_1 & p_2 & \cdots & p_{k-4} & p_{k-3} & p_{k-2} \end{pmatrix}$$

are positive innerwise.

Corollary B.4. *(Eladyi, 2005 [28]) The three criteria in the Determinantal Criterion have an explicit form for lower orders.*
For $k = 2$,

$$|p_1| < 1 + p_0 < 2. \tag{B.2}$$

For $k = 3$,

$$|p_2 + p_0| < 1 + p_1 \qquad and \qquad |p_1 - p_2 p_0| < 1 - p_0^2. \tag{B.3}$$

In the event that equation B.1 is of the following form with only 2 parameters:

$$p(\lambda) = \lambda^k + a \sum_{i=i}^{k-1} \lambda^i + b, \tag{B.4}$$

we have an explicit form for the necessary and sufficient conditions for asymptotic stability.

Theorem B.5. *(Tomášek, 2015 [30]) Let a and b be real non-zero constants and $k \geq 2$. The zeroes of the characteristic polynomial (equation B.4) lie inside the unit disc if and only if*

(i) $b > a - 1$

(ii) $b < 1$

(iii) $b > (1 - k)a - 1$.

Theorem B.6 (Sufficient condition for stability). *(Eladyi, 2005 [28]) The zeroes of the characteristic polynomial (equation B.1) lie inside the unit disc if*

$$\sum_{i=0}^{k-1} |p_i| < 1. \tag{B.5}$$

Lemma B.7 (Necessary conditions for stability). *(Bistritz, 1984 [31]) If the zeroes of the characteristic polynomial (equation B.1) lie inside the unit disc, then the following hold:*

(i) $|p_0| < 1$

(ii) $p(1) > 0$

(iii) $(-1)^k p(-1) > 0$.

Theorem B.8. *(Botta et al., 2014 [32]) The zeroes of the polynomial* $p(\lambda) = \lambda^k + a(\lambda^{k-1} + \cdots + \lambda) + 1$, $a \in \mathbb{R}$, *of degree $k > 1$ lie on the unit circle if and only if*

(i) $-\frac{2}{k-1} \le a \le 2$ *if k is even*

(ii) $-\frac{2}{k-1} \le a \le 2 + \frac{2}{k-1}$ *if k is odd.*

Appendix C Theorems regarding bifurcation analysis

We first start from the following discrete time dynamical system (map) given by

$$x \mapsto f(x, \omega), \qquad x \in \mathbb{R}^n, \qquad \omega \in \mathbb{R}^1. \tag{C.1}$$

To perform a bifurcation analysis on the fixed point x^* at the critical parameter value ω^*, we do a coordinate shift, placing the fixed point at the origin. Now, in the new coordinates, after renaming and keeping the same variable x, we assume that this system can be written in the form

$$\tilde{x} = Jx + F(x), \qquad x \in \mathbb{R}^n, \tag{C.2}$$

where J is the Jacobian matrix and $F(x)$ is a smooth function of order $O(\|x\|^2)$ with the following Taylor expansion

$$F(x) = \frac{1}{2}K(x, x) + \frac{1}{6}L(x, x, x) + O(\|x\|^4). \tag{C.3}$$

Here, $K(x, y)$ and $L(x, y, z)$ are defined by

$$K_i(x, y) = \sum_{j,k=1}^{n} \left. \frac{\partial^2 F_i(s)}{\partial s_j \partial s_k} \right|_{s=0} x_j y_k \tag{C.4}$$

and

$$L_i(x, y, z) = \sum_{j,k,l=1}^{n} \left. \frac{\partial^3 F_i(s)}{\partial s_j \partial s_k \partial s_l} \right|_{s=0} x_j y_k z_l \tag{C.5}$$

for $i = 1, \cdots, n$. This presentation and subsequent results in this section are attributed to Kuznetsov, 2004 [33].

C.1   Flip Bifurcation

Here, assume that the Jacobian J has a simple eigenvalue -1 on the unit circle. The map (equation C.2), when restricted to the center manifold, can be transformed to the normal form

$$\tilde{y} = -y + by^3 + O(y^4), \tag{C.6}$$

where b gives the direction of bifurcation of the period-two cycle and can be computed using the following invariant formula:

$$b = \frac{1}{6}\langle p, L(q, q, q) \rangle - \frac{1}{2}\langle p, K(q, (J - I)^{-1}K(q, q)) \rangle. \tag{C.7}$$

In this case, q is the real eigenvector satisfying $Jq = -q$ while p is the real adjoint eigenvector satisfying $J^T p = -p$. The eigenvectors p and q have been normalized such that $\langle p, q \rangle = 1$. Functions $K(x, y)$ and $L(x, y, z)$ are as defined in equations C.4 and C.5 respectively.

If $b > 0$, then a stable period-two cycle bifurcates from the fixed point for parameter values ω larger than the critical parameter value ω^*. When $b < 0$, then the cycle of period-two is unstable. The case of $b = 0$ means that the bifurcation is more degenerate than the normal form.

C.2   Neimark-Sacker Bifurcation

C.2.1   Absence of strong resonances

Assuming that the Jacobian J has a simple pair of eigenvalues on the unit circle $e^{\pm i\theta_0}$, where $\theta_0 \in (0, \pi)$ and these are the only eigenvalues with modulus 1. The absence of strong resonances means that $e^{ik\theta_0} \neq 1$ for $k \in \{1, 2, 3, 4\}$. Then, the map (equation C.2) can be restricted to the center manifold and transformed to the complex normal form

$$\tilde{z} = e^{i\theta_0} z \left(1 + d_1 |z|^2\right) + O(|z|^4), \tag{C.8}$$

where $d = \mathrm{Re}\, d_1$ gives the direction of the closed invariant curve and can be computed using the following invariant expression

$$d = \frac{1}{2} \mathrm{Re} \left\{ e^{-i\theta_0} [\langle p, L(q, q, \overline{q}) \rangle + 2\langle p, K(q, (I - J)^{-1} K(q, \overline{q})) \rangle + \langle p, K(\overline{q}, (e^{2i\theta_0} I - J)^{-1} K(q, q)) \rangle] \right\}. \tag{C.9}$$

Here, q is the complex eigenvector satisfying $Jq = e^{i\theta_0} q$ and $J\overline{q} = e^{-i\theta_0} \overline{q}$ while p is the complex adjoint eigenvector satisfying $J^T p = e^{-i\theta_0} p$ and $J^T \overline{p} = e^{i\theta_0} \overline{p}$. The scalar product $\langle p, q \rangle = \overline{p}^T q$ is the standard one in $\mathbb{C}^n$; p and q have been normalized such that $\langle p, q \rangle = 1$. Functions $K(x, y)$ and $L(x, y, z)$ are as defined in equations C.4 and C.5 respectively.

If $d < 0$, then a stable closed invariant curve bifurcates from the fixed point for parameter values ω larger than the critical parameter value ω^*. When $d > 0$, then the closed invariant curve is unstable. The case of $d = 0$ means that the bifurcation is more degenerate than the normal form.

C.2.2   1:4 resonance

In this case, Jacobian J has eigenvalues $e^{\pm i\frac{\pi}{2}} = \pm i$ and the map (equation C.2) can be transformed to the following complex normal form

$$\tilde{z} = iz + bz|z|^2 + d\overline{z}^3 + O(|z|^4), \tag{C.10}$$

where

$$b = \frac{1}{2} \langle p, L(q, q, \overline{q}) + 2K(q, (I - J)^{-1} K(q, \overline{q})) - K(\overline{q}, (I + J)^{-1} K(q, q)) \rangle \tag{C.11}$$

and

$$d = \frac{1}{6} \langle p, L(\overline{q}, \overline{q}, \overline{q}) - 3K(\overline{q}, (I + J)^{-1} K(\overline{q}, \overline{q})) \rangle. \tag{C.12}$$

As before, the terms K, L, p and q are as defined in the previous section where there is an absence of strong resonances.

AIMS Press