Dynamic behaviour of a discrete-time SIR model with information dependent vaccine uptake

Journal:	*Journal of Difference Equations and Applications*
Manuscript ID:	GDEA-2014-0239.R2
Manuscript Type:	Original Article
Date Submitted by the Author:	n/a
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Keywords:	discrete deterministic SIR models, rational opposition to vaccination, global stability, persistence, oscillations
2010 Mathematics Subject Classification:	92D30, 34K20, 34K25

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Abstract

This paper proposes and analyzes a discrete-time deterministic SIR model with information dependent immunization behaviour, where vaccination coverage at birth during any period of time is a general phenomenological function of the risk of infection that is perceived at the beginning of the period. Results on existence of equilibria, their local stability, and system persistence are proved. Then, by considering the noteworthy subcase of a piecewise linear "prevalence-dependent" coverage function, the local stability of the endemic state is proved and conditions for its global asymptotic stability are given. Some insight on both Neimarck-Sacher and period-doubling bifurcations are provided. Overall we show that prevalence-dependent coverage is an essentially stabilising force. However period-doubling bifurcations are possible though under stressed parameter constellations.*Key words*: discrete deterministic SIR models, global stability, oscillations, persistence, rational opposition to vaccination.

1 Introduction

In the mathematical models traditionally used in the epidemiology of transmissible infections (see e.g. the classical Anderson and May 1991 book [2]) individual being virtually play no role: they are just passive actors of the disease's scene. However, there is nowadays an endlessly increasing list of examples unequivocally documenting that this passive role of humans can be considered at best an approximation, perhaps usable for modeling infections in traditional communities, but not anymore valid in modern - highly interconnected and information seeking – societies [4], [5], [10].

The most well- known examples in this list range from the worldwide diffusion of panic following the SARS outbreak in the Far-East in 2003, yielding a dramatic fall in travels and tourism in those areas, to the schizophrenic reactions to the 2009 H1N1 pandemic alert, to the many episodes of opposition to vaccines, one of the single innovations that mostly contributed to increase in life-expectancy, well-being, and living standards of human societies [5].

Consequently, a main news in the landscape of recent research in the epidemiology of transmissible infections has been represented by the full acknowledgement of this increasingly important role played, in modern societies, by individual human behavior, for the transmission dynamics and control of infectious diseases. This has brought the need to look for new models and explanations by importing into mathematical epidemiology the typical approaches from behavioral sciences. This cross-fertilization aims to set out the feedbacks between epidemiological inputs, the related information flows, the agents' elaboration of this information, and the ensuing responses to the epidemiological signals [4], [5], [16].

In this fast growing literature on "behavioral epidemiology" [16] a prominent area of investigation has focused on the modeling of immunization choices in relation to vaccine preventable infections of childhood, such as measles, mumps, rubella and pertussis, in regimes of voluntary immunization [5]. Much of this literature has been stimulated by two major episodes of vaccine opposition, i.e. the opposition to the whole-cell pertussis vaccine, and the MMR-autism scare following the evidence in Wakefield et al paper [24], published in 1998 on the Lancet and later retracted by the journal by suggesting the possibility of a causal link between MMR immunization and autistic spectrum disorders.

A range of alternative mathematical modeling approaches have been proposed to represent the complex interplay between choices related to childhood immunization and the ensuing transmission and control of infections. These approaches are reviewed in [5], to which the reader can refer for a broader overview. A wide sample of these approaches is reported in [16]. Among these modeling strategies a simple one allowing substantial mathematical deepening is the one developed by d'Onofrio et al [7], [8] that proposed a generalized family of information-dependent, deterministic, SIR (Susceptible-Infective-Removed) continuous-time models, where the vaccination coverage at birth is taken to be a general phenomenological function of the perceived risk related to the disease, taken in its turn as a function of the information available on the infection.

In this research line no papers have however been devoted, to the best of our knowledge, to the investigation of the dynamic properties of discrete-time epidemiological models incorporating vaccinating behavior.

It is the aim of this paper therefore to fill this gap by proposing and analyzing a

discrete-time deterministic SIR model with information dependent vaccination coverage at birth. The model reformulates in a discrete setting the unlagged version of the continuous-time model proposed in [7], and assumes that the vaccination coverage at birth at any time is a general function of the risk of infection that is perceived at the beginning of the period.
Our results provide a fairly general characterization of the system dynamics and also a systematic comparison with the results provided in the benchmark paper by Allen [7]. As for the general case we are able to provide conditions for the existence of equilibria, their local stability, and in particular system persistence. The latter is a fairly important property from the epidemiological viewpoint related to the substantive epidemiological question of whether the infection will "persists" or not in the population over the long-term irrespective of whether solutions converge (e.g. globally) to a steady state or oscillate wildly around it. Then, we move to consider the noteworthy subcase of a piecewise linear "prevalence-dependent" coverage function, where the actual infective prevalence is the determinant of the perceived risk of infection used by families to decide whether to vaccinate or not their children. For this case we have been able to provide conditions for the global asymptotic stability of the endemic state, and the onset of bifurcations.
The paper is organized as follows. In Section 2 we critically review previous results about the dynamics of discrete SIR models. In Section 3 we present our model, and report some main features in Section 4. Section 5 focuses on the local stability of the endemic state and on system persistence. Section 6 considers the subcase of a "prevalence-dependent" coverage function, for which the local stability of the endemic state is proved and conditions for its global asymptotic stability are given. Numerical illustrations are postponed to Section 7. Concluding remarks follow.

2 Discrete SIR models with constant population

Though the first discrete models for the transmission dynamics of infection date back to long ago (e.g. [11]) the first work systematically focusing on the dynamic properties of deterministic discrete SIR (and SI, SIS) models is (to the best of our knowledge) the paper by Allen in 1994 [1]. Denoting by $S(t)$, $I(t)$ and $R(t)$ respectively the susceptible, infected and removed fractions at time $t \geq 0$ and setting the time step Δt equal to 1, the discrete epidemic SIR model introduced in [1] is:

$$\begin{aligned} S(t+1) &= S(t) - \beta S(t) I(t) \\ I(t+1) &= I(t)(1-\nu) + \beta S(t) I(t) \\ R(t+1) &= R(t) + \nu I(t) \end{aligned} \tag{1}$$

where $\beta > 0$ and $\nu > 0$ represent the transmission and the removal rate respectively, and the initial conditions $S(0) > 0$, $I(0) > 0$ and $R(0) \geq 0$ satisfy $S(0) + I(0) + R(0) = 1$. Solutions of the model are positive for all t if and only if $\max\{\beta, \nu\} \leq 1$. The latter condition means that, in order to have nontrivial epidemiological behavior, the (unit) time step must be smaller of both the two model relevant time scales, i.e., according to the geometric distribution, the average time spacing between two consecutive infective contacts $(1/\beta)$ and the average duration of the infectious period $(1/\nu)$.
The reproduction number at time t is $\mathcal{R}(t) = \beta/\nu S(t)$. If $\mathcal{R}(0) \leq 1$ there is no

epidemics and the infection goes extinct; if $\mathcal{R}(0) > 1$ there is an epidemics but periodic behavior is impossible. In particular for $S(0) = 1$, i.e. a wholly susceptible population, we have the *basic reproduction number* (BRN) $\mathcal{R}_0 = \beta/\nu$. Considering also births and deaths at the same rate $\mu > 0$ in order to ensure a constant population size in model (1), Allen introduced the SIR model with vital dynamics:

$$\begin{array}{l} S(t+1) = (1-\mu) S(t) + \mu - \beta S(t) I(t) \\ I(t+1) = (1-\nu-\mu) I(t) + \beta S(t) I(t) \\ R(t+1) = (1-\mu) R(t) + \nu I(t) \end{array} \tag{2}$$

where the third equation can be discarded for the analysis. Solutions are non-negative for all initial conditions if and only if

$$\text{i)} \;\; \mu + \nu \leq 1; \qquad \text{ii)} \;\; \beta \leq (1+\sqrt{\mu})^2 . \tag{3}$$

In this case the basic reproduction number is $\mathcal{R}_0 = \beta/(\mu+\nu)$. If $\mathcal{R}_0 \leq 1$ there is only the infection free equilibrium, $(1,0)$ which is globally asymptotically stable; for $\mathcal{R}_0 > 1$ the author showed there is the unique endemic equilibrium state EE defined by:

$$S_E = \frac{\mu+\nu}{\beta} = \frac{1}{\mathcal{R}_0}; \qquad I_E = \frac{\mu}{\mu+\nu}\left(\frac{\mathcal{R}_0 - 1}{\mathcal{R}_0}\right) \tag{4}$$

that is locally asymptotically stable and no cycles of period two exist. For μ and β "sufficiently small" numerical simulations showed the global stability of the endemic equilibrium but no analytical results were proven. When conditions (3) are not fulfilled complex dynamics are possible: the author showed that for $\mu = 0.95$, $\nu = 0.05$, $S_0 = 0.99$, $I_0 = 0.01$ for $\beta = 3.5$ the fraction of infectives follows a 2-periodic orbit and more periodic behaviors for larger values of β. In [3], where model (2) is extended to the case of a more general nonlinear force of infection $\lambda(t)$, the local stability of the endemic equilibrium (for $\mathcal{R}_0 > 1$) of model (2) was obtained under the assumption $\beta < 1 - \mu$ (compare with (3ii)). We now briefly detail the (local) stability behavior of (S_E, I_E) for $1 - \mu \leq \beta < \left(1+\sqrt{\mu}\right)^2$. By applying the Jury criterion to the Jacobian matrix at the endemic equilibrium (4)

$$J_E = \begin{pmatrix} 1 - \mu\mathcal{R}_0 & -\mu - \nu \\ \mu(\mathcal{R}_0 - 1) & 1 \end{pmatrix}$$

we conclude that Neimark-Sacker bifurcations are excluded (as $\det J_E < 1$) and the local stability at the endemic state (S_E, I_E) follows under the assumption

$$1 < \mathcal{R}_0 < \frac{4 - \mu(\mu+\nu)}{\mu(2 - (\mu+\nu))}. \tag{5}$$

As $\mathcal{R}_0 = \beta/(\mu+\nu)$, the right condition in (5) is verified for all μ and ν such that the r.h.s. is greater than the ii) positivity condition in (3), that is if and only if

$$(\mu+\nu)\frac{4 - \mu(\mu+\nu)}{\mu(2 - (\mu+\nu))} > (1+\sqrt{\mu})^2 \tag{6}$$

equivalent to

$$\mu\nu^2 + \left(\mu^2 - 2\mu^{3/2} - \mu - 4\right)\nu - \mu\left(2\mu^{3/2} - \mu - 4\mu^{1/2} + 2\right) < 0.$$

Since the associated discriminant

$$\Delta = \left(\mu^2 - 2\mu^{3/2} - \mu - 4\right)^2 + 4\mu\left(2\mu^{3/2} - \mu - 4\mu^{1/2} + 2\right)$$

is positive, and

$$\nu_2 := \frac{4 - \mu^2 + 2\mu^{3/2} + \mu + \sqrt{\Delta}}{2\mu} > 1 - \mu$$

the local stability of (S_E, I_E) is ensured for all pairs (μ, ν) such that

$$\nu_1 := \frac{4 - \mu^2 + 2\mu^{3/2} + \mu - \sqrt{\Delta}}{2\mu} < \nu < 1 - \mu. \tag{7}$$

Remark 1 *A sufficient (though not necessary) condition for the local stability of the endemic state EE is that $I_E < S_E$. In fact:*

$$I_E < S_E \quad \Leftrightarrow \quad \frac{\mu}{\mu+\nu}\left(\frac{\mathcal{R}_0 - 1}{\mathcal{R}_0}\right) < \frac{1}{\mathcal{R}_0} \quad \Leftrightarrow \quad \mathcal{R}_0 < \frac{2\mu + \nu}{\mu}.$$

The conclusion follows by comparing this last inequality with (5):

$$\frac{4 - \mu(\mu+\nu)}{\mu(2 - (\mu+\nu))} - \frac{2\mu + \nu}{\mu} = \frac{(\mu+\nu)^2 + 4 - 4\mu - 2\nu}{\mu(2 - (\mu+\nu))} > 0.$$

Previous results on the local stability imply the existence of a "small" region, characterized by $0 < \nu \leq \nu_1$, of the set $\{(\mu, \nu) : \mu > 0, \nu > 0, \mu + \nu \leq 1\}$ in correspondence of the local stability of the EE does not hold and therefore more complicated behaviors are possible. For instance, if $\mu = 0.81$ and $\nu = 0.05$ (with $I_E = 0.67858$) one finds

$$\beta^{BIF} = \mu + \nu + \frac{2(2-\mu)(\mu+\nu)}{\mu(2 - (\mu+\nu))} = 3.07659 \leq (1 + \sqrt{\mu})^2 = 3.61.$$

Figure 1 illustrates the ensuing cascade of bifurcations. We must however emphasize the lack of epidemiological significance of the considered parameter constellation, where infection duration is 16-fold longer than humans lifespan, possibly causing the model to approach the SI form.

Figure 1. Role of the transmission parameter β in triggering period-doublig bifurcation in Allen's model [1] under Allen parameter constellation ($\mu = 0.81$, $\nu = 0.05$).

We conclude by observing that the literature on discrete SIR models presents several other different approaches. Some global results on the stability of the endemic equilibrium were obtained recently in [15] (see also [12]) for a SIR model with non constant population. Further results appeared in the literature have dealt with discrete SIR models obtained via different discretization methods of the continuous ones and analyzed with various approaches [9], [13], [14], [18], [19], [20], [21] and [22].

3 A discrete SIR model with information-dependent vaccination

Motivated by the recent research work on behavioral epidemiology models ([7], [16]), we consider the following family of discrete SIR models for a non-fatal disease in a constant homogeneously mixing population, with state-dependent vaccination coverage at birth:

$$\begin{aligned} S(t+1) &= (1-\mu)S(t) + \mu(1-p(M)) - \beta S(t)I(t) \\ I(t+1) &= (1-(\mu+\nu))I(t) + \beta S(t)I(t) \\ R(t+1) &= (1-\mu)R(t) + \nu I(t) + \mu p(M) \end{aligned} \tag{8}$$

S,I are as before, whereas the removed fraction R now includes not only those who are permanently immune due to recovery from infection but also those who are immune through vaccination.

Model (8) represents a discrete version of the one introduced in [7] in the unlagged case. It can be derived directly, by relating flows of demographic and epidemiological events over the time interval $(t, t+\Delta t)$ to the numbers in the various states at time t and finally setting $\Delta t = 1$. This choice does not cause a loss of generality given that Δt would everywhere be embedded into the system parameters.

All the parameters are assumed positive: $\mu \in (0,1)$ denotes the birth and death rates (assumed identical to keep the total population size constant), $\nu \in (0,1)$ the rate of recovery from infection, $\beta \in (0, +\infty)$ the constant transmission rate. The *vaccination coverage function* p describes, assuming a 100% effective vaccine, the actual vaccination coverage at birth as a function of the state of the information variable M, which in turn summarizes the available information about the perceived risk of infection that is used by families to decide on whether to vaccinate or not their children. M is assumed to be a differentiable function g of S and I, with $\partial g/\partial I > 0$ and with $g(S,0) = 0$ for all S. Thus, $M(t) = g(S(t), I(t))$ is bounded when S and I are, taking its values in the interval $\mathcal{I} = [0, M^{\sup})$, where $M^{\sup} = \sup_{S,I} g(S,I)$.

Analogously to [7], we define p as:

$$p(M) = p_0 + p_1(M) \qquad M \in \mathcal{I} \tag{9}$$

where:

i) p_0 is a constant in $(0,1)$;

ii) $0 \le p_1(M) \le 1 - p_0$ for all $M \in \mathcal{I}$;

iii) $p_1(0) = 0$;

iv) p_1 is continuous and differentiable, except at a finite number of points, with $p_1' > 0$.

This formulation amounts to assuming that the vaccination coverage is the sum of a constant component, related to families which vaccinate their children independently of the information about the state of the infection, and of an information dependent one, which is increasing in M. This implies that when the perceived risk related to infection increases some families previously not

vaccinating will react by increasing the probability of immunizing their children, thereby increasing the overall vaccine uptake of the population.
Discarding the variable R because its dynamics follow trivially from that one of S, I, system (8) reduces to:

$$\begin{array}{l} S(t+1) = (1-\mu)\, S(t) + \mu\,(1 - p_0 - p_1(g(S(t), I(t)))) - \beta S(t)\, I(t) \\ I(t+1) = (1-(\mu+\nu))\, I(t) + \beta S(t)\, I(t) \end{array} \tag{10}$$

on the compact set $\Omega = \{(S, I) :\ S \geq 0,\ I \geq 0,\ S + I \leq 1\}$. We shall assume $S(0) > 0$ and $I(0) > 0$.

Remark 2 *Model (10) is intermediate with respect two limit cases.*
The first one is for $1 - p_0 - p_1(g(S(t), I(t)) \equiv 0$ *corresponding to* full coverage: *all newborns are vaccinated and it is reasonable that in the long run both susceptibles and infected fractions tend to zero. This is can be easily proved by observing that in this case the sequence* $\{S(t)\}$ *is positive (for* $\beta \leq 1 - \mu$*) and strictly decreasing and a positive limit is incompatible with the first equation in (10). As* $S(t) \to 0^+$ *when* $t \to +\infty$*, assuming* $\mu + \nu < 1$*, with a similar reasoning we get* $I(t) \to 0^+$*.*
The second limit case is for $1 - p_0 - p_1(g(S(t), I(t)) \equiv 1$*, corresponding to the case of* no vaccination*, namely model (2).*

4 Some general properties

In this section we state some general properties of model (10), i.e. properties that are valid for any possible form of the functions p_1 or g. First we supply a necessary and sufficient condition for the invariance of the state space. This conditions ensures that trajectories originating from epidemiologically meaningful initial conditions remain meaningful at any subsequent times. Then we investigate the existence of equilibria. Here we show that the steady state structure is the one typical of simple epidemiological models: the model always has the disease free equilibrium (DFE), and moreover, when an appropriate threshold condition is met, an endemic equilibrium (EE) also appears. The relation between the threshold parameter and the global asymptotic stability of the DFE is also stated.
By setting $p_1(M^{\sup}) = \sup_{S,I \in \Omega} p_1(g(S, I))$, we denote with

$$(1-p)^{\inf} := 1 - p_0 - p_1(M^{\sup}) \tag{11}$$

the minimum fraction of newborn individuals that are not vaccinated. By i) and ii) it follows $0 \leq (1-p)^{\inf} \leq 1$.
Our first proposition, following the same approach of [1], provides conditions for the invariance of the state space Ω .

Proposition 3 *For every initial conditions* $S(0) \geq 0$ *and* $I(0) \geq 0$ *all the solutions of (10) are in* Ω *for all* $t \geq 0$ *if and only if the following conditions hold:*

i) $\mu + \nu \leq 1$;

ii) $\beta \leq 1 - \mu + 2\mu(1-p)^{\inf} + 2\sqrt{\left(1 - \mu\left(1 - (1-p)^{\inf}\right)\right)\mu(1-p)^{\inf}} := \beta^*$ (12)

Proof. By the second equation in (10) it is evident that for each $S(t) \geq 0$ and $I(t) \geq 0$ we have $I(t+1) \geq 0$ if and only if (12i) holds. Now, set $\varepsilon = S(0)$, then $I(0) \leq 1-\varepsilon$. Since $(1-p)^{\inf} \geq 0$, we obtain:

$$S(1) \geq \varepsilon(1-\mu-\beta(1-\varepsilon)) + \mu(1-p)^{\inf} = f(\varepsilon). \tag{13}$$

We show now that, under (12ii), the convex quadratic function f has global minimum $f(\varepsilon^*) \geq 0$ for every $\varepsilon \in [0,1]$, then $S(1) \geq 0$. In fact, the unique global minimum point of f on $[0,1]$ is

$$\varepsilon^* = \max\left\{0, \frac{\mu+\beta-1}{2\beta}\right\}.$$

Notice that $\varepsilon^* \leq 1/2$. If $\mu+\beta-1 \leq 0$ then $\varepsilon^* = 0$ and $f(\varepsilon^*) = \mu(1-p)^{\inf} \geq 0$. If $\mu+\beta-1 > 0$, the corresponding minimum value

$$f(\varepsilon^*) = \frac{4\beta\mu(1-p)^{\inf} - (\beta+\mu-1)^2}{4\beta}$$

is non negative if and only if

$$\beta^2 - 2\left(1-\mu+2\mu(1-p)^{\inf}\right)\beta + (1-\mu)^2 \leq 0 \tag{14}$$

equivalent to ii) in (12). Summing up, if $\beta \in (0, \beta^*]$, then $f(\varepsilon^*) \geq 0$ and from (13) it follows $S(1) \geq 0$ for each $S(0) \in [0,1]$. Therefore, nonnegativity follows by induction. The necessity part immediately follows by observing that, as stated in the previous section, p_1 is strictly increasing and g is increasing with respect to I, so $p_1(g(S,I)) \leq p_1(g(S,1-S)) \leq p_1(M^{\sup})$.
To conclude, observe that, if $S(t)+I(t) \leq 1$, then:

$$\begin{aligned} S(t+1)+I(t+1) &= (S(t)+I(t))(1-\mu)+\mu(1-p_0-p_1(M(t)))-\nu I(t) \leq \\ &\leq (1-\mu)+\mu(1-p_0-p_1(M(t))) \leq 1 \end{aligned}$$

and the statement follows by an induction argument. ■

Remark 4 *The upper bound for β in (12ii)) is an increasing function of $(1-p)^{\inf}$, belonging to the interval $\left[1-\mu, \left(1+\sqrt{\mu}\right)^2\right]$, then $\beta \in (0,4)$. Notice furthermore that if $p_1(M(t)) \equiv 0$ (that is the case of information-independent vaccination) then*

$$\beta \leq 1+\mu-2\mu p_0 + 2\sqrt{(1-\mu p_0)(1-p_0)\mu}.$$

In words, considering vaccination (be this information-related or not) has the effect of reducing the range of the values of the contact rate that ensure the model to be epidemiologically meaningful.

The next result shows that the size of the unvaccinated population is asymptotically bounded above by $1-p_0$.

Proposition 5 *The following subset of Ω*

$$\widetilde{\Omega} = \{(S,I):\ S \geq 0,\ I \geq 0,\ S+I \leq 1-p_0\} \tag{15}$$

is an asymptotically attractive set.

Proof. Defining $\sigma(t) = S(t) + I(t)$, we obtain:

$$\begin{aligned} \sigma(t+1) &= (1-\mu)\sigma(t) + \mu(1-p_0) - \mu p_1(M(t)) - I(t)\nu \leq \\ &\leq (1-\mu)\sigma(t) + \mu(1-p_0) \end{aligned} \tag{16}$$

This implies $\sigma(t) \leq (1-\mu)^t (\sigma(0) - (1-p_0)) + 1 - p_0$ and then asymptotically:

$$S + I \leq 1 - p_0 \tag{17}$$

■

Remark 6 *Referring to the previous proof, if $\sigma(0) \leq 1 - p_0$ then by (16) it follows $\sigma(t) \leq 1 - p_0$ for all $t \geq 1$. In this case $\widetilde{\Omega}$ is invariant.*

Our next results deal with the existence of steady states. It is trivial to show that model (10) always has the disease free equilibrium $DFE = (1-p_0, 0)$. A local stability analysis shows that the DFE is unstable if $(1-p_0)R_0 > 1$ where $R_0 = \beta/(\mu+\nu)$. Note that the quantity $(1-p_0)R_0$ represents the number of secondary infections that would be caused by a single infective individual in a homogeneously mixing population where a fraction p_0 is immune by vaccination and every one else is susceptible. We call this quantity the "residual reproduction number" under immunization with constant coverage p_0. The next results gives a global property of the DFE.

Proposition 7 *If $(1-p_0)R_0 \leq 1$ the DFE of model (10) is globally asymptotically stable.*

Proof. If $(1-p_0)R_0 \leq 1$, a direct inspection of the second equation in (10) shows that DFE is the unique equilibrium of the system. Furthermore, by exploiting (17), for t sufficiently large:

$$\begin{aligned} I(t+1) &\leq I(t)(1-(\mu+\nu)) + \beta I(t)(1-p_0-I(t)) \leq \\ &\leq I(t) + I(t)(\beta(1-p_0) - (\mu+\nu)) \ . \end{aligned}$$

Thus if $(1-p_0)R_0 \leq 1$ then $I(t+1) \leq I(t)$, that is $\{I(t)\}$ is a non negative definitively decreasing sequence, converging to the unique fixed point 0. Consequently $S(t)$ converges to $1-p_0$, i.e. the DFE is globally asymptotically stable.

■

Remark 8 *The previous theorem shows that, as stated above, the model has the equilibrium structure of simple epidemiological models: a DFE which always exists, and an endemic equilibrium which appears only when an appropriate threshold condition is met. The threshold parameter (the residual reproduction number) includes only the behavior-independent component of vaccination p_0. In other words the presence of a behavioral component does not affect the existence of equilibria, as also noted in [7].*

Remark 9 *Obviously, for most infectious diseases it is unlikely that the condition $(1-p_0)R_0 \leq 1$ is fulfilled on the large scale (it might clearly be at small-medium scales where adhesion to vaccination is large). Indeed, it is equivalent to require that the fraction p_0 of parents who steadily (i.e. regardless of rumors)*

vaccinate their children is in excess of the critical vaccination threshold ([2]) $p_c > 1 - 1/R_0$.
As far as p_0 is concerned, we expect that in the real world circumstances, particularly under voluntary vaccination, p_0 might be an increasing function of the long-term perceived risk from the disease. Therefore, other things (e.g. risk of serious disease given infection, risk of vaccine side effects) being equal we expect that p_0 might be an increasing function of R_0. It might be interesting to validate this hypthesis against experimental data.

When $(1 - p_0)R_0 > 1$ it is possible to show that there is a unique endemic equilibrium, as stated in the next proposition.

Proposition 10 *If $(1 - p_0) R_0 > 1$ model (10) admits a unique endemic equilibrium $EE = (S_e, I_e)$.*

Proof. The unique non zero solution of $I(t+1) = I(t)$ is:

$$S_e = \frac{\mu + \nu}{\beta} = \frac{1}{R_0} . \tag{18}$$

We set $\widehat{p}_1(I) = p_1(g(R_0^{-1}, I))$, and prove that there exists a unique solution I_e of the equation $S(t+1) = S(t)$ on the interval $(0, 1 - S_e)$:

$$1 - p_0 - \frac{1}{R_0} - \frac{\mu + \nu}{\mu} I = \widehat{p}_1(I). \tag{19}$$

Indeed, by assumptions on p_1 and g, the function $f_2(I) = \widehat{p}_1(I)$ is strictly increasing, whereas

$$f_1(I) = 1 - p_0 - \frac{1}{R_0} - \frac{\mu + \nu}{\mu} I$$

is strictly decreasing. Condition $(1 - p_0) R_0 > 1$ is equivalent to state that

$$f_1(0) = 1 - p_0 - \frac{1}{R_0} > 0 = f_2(0).$$

Hence, by continuity, f_1 and f_2 intersect at a single point I_e of $(0, 1 - S_e)$ because

$$f_1(1 - S_e) = -p_0 - \frac{\nu}{\mu}\left(1 - \frac{1}{R_0}\right) < 0 < f_2(1 - S_e).$$

■

As pointed out in the continuous-time framework in [7], the following estimate holds

$$I_E^{\infty} < I_e < I_E^{o} \tag{20}$$

where $I_E^o := ((1 - p_0) R_0 - 1) \frac{\mu}{\beta}$ and $I_E^{\infty} := \left(R_0 p^{\inf} - 1\right) \frac{\mu}{\beta}$. Note that I_E^{∞} and I_E^o are equal to the infectious fraction obtainable in a SIR model with constant vaccination rate at birth equal to $p_0 + p_1(M^{\sup})$ and p_0 respectively.

5 Stability analysis of the endemic equilibrium

In this section we study some properties of the endemic equilibrium $EE = (S_e, I_e)$, assuming p_1 and g are differentiable: we obtain conditions for the local asymptotic stability and persistence.

5.1 Local asymptotic stability

The following general result holds:

Proposition 11 *Let*

$$A := \mu + \beta I_e + \mu p_1' \left(g \left(S_e, I_e \right) \right) \frac{\partial g}{\partial S} \left(S_e, I_e \right). \tag{21}$$

If $(1 - p_0)R_0 > 1$ *and*

$$\frac{1}{\mu \beta I_e} \left(2A - \beta^2 I_e S_e - 4 \right) < p_1' \left(g \left(S_e, I_e \right) \right) \frac{\partial g}{\partial I} \left(S_e, I_e \right) < \frac{1}{\mu \beta I_e} \left(A - \beta^2 I_e S_e \right) \tag{22}$$

then provided (12) holds the unique endemic state EE of system (10) is locally asymptotically stable (LAS).

Proof. Since $(1 - p_0)R_0 > 1$, by Proposition 10, there exists a unique endemic equilibrium $EE = (S_e, I_e)$ for system (10). The Jacobian matrix $J_e = J(S_e, I_e)$ at EE is:

$$J_e = \begin{pmatrix} 1 - A & -\beta S_e - \mu p_1' \left(g \left(S_e, I_e \right) \right) \frac{\partial g}{\partial I} \left(S_e, I_e \right) \\ \beta I_e & 1 \end{pmatrix}. \tag{23}$$

We apply the Jury stability criterion, that is we prove that

$$\begin{aligned} 1 - \mathrm{tr} J_e + \det J_e &> 0 \\ \det J_e &< 1 \\ 1 + \mathrm{tr} J_e + \det J_e &> 0. \end{aligned} \tag{24}$$

Since

$$\begin{aligned} \mathrm{tr} J_e &= 2 - A \\ \det J_e &= 1 - A + \beta I_e \left(\beta S_e + \mu p_1' \left(g \left(S_e, I_e \right) \right) \frac{\partial g}{\partial I} \left(S_e, I_e \right) \right) \end{aligned} \tag{25}$$

by $p_1' > 0$ and $\partial g / \partial I > 0$ the first condition in (24) is always satisfied, signaling that can not exists an eigenvalue equal to 1. Furthermore, the right inequality in (22) implies

$$\det J_e < 1 - A + \beta I_e \left(\beta S_e + \frac{1}{\beta I_e} \left(A - \beta^2 I_e S_e \right) \right) = 1$$

whereas the left side in (22) gives

$$\begin{aligned} 1 + \det J_e + \mathrm{tr} J_e &= 2 \left(2 - A \right) + \beta I_e \left(\beta S_e + \mu p_1' \left(g \left(S_e, I_e \right) \right) \frac{\partial g}{\partial I} \left(S_e, I_e \right) \right) > \\ &> 2 \left(2 - A \right) + \beta I_e \left(\beta S_e + \frac{1}{\beta I_e} \left(2A - \beta^2 I_e S_e - 4 \right) \right) = \\ &= 0. \end{aligned}$$

■

Some remarks on the previous proposition are appropriate.
Since, by assumption, $p_1' > 0$ and $\partial g/\partial I > 0$, condition (22) makes sense if and only if $\beta^2 I_e S_e < A < 4$ i.e. if and only if

$$-1 - \frac{\beta I_e}{\mu}(1 - \beta S_e) < p_1'(g(S_e, I_e))\frac{\partial g}{\partial S}(S_e, I_e) < -1 - \frac{\beta I_e}{\mu}\left(1 - \frac{4}{\beta I_e}\right) \quad (26)$$

where

$$-1 - \frac{\beta I_e}{\mu}(1 - \beta S_e) < 0; \qquad -1 - \frac{\beta I_e}{\mu}\left(1 - \frac{4}{\beta I_e}\right) > 0. \quad (27)$$

The first inequality in (27) is a direct consequence of condition 12i). The second one is equivalent to condition $\beta I_e + \mu < 4$. This last follows from the upper estimation in (20) $I_e < I_E^o$ that rewrites as

$$\beta I_e + \mu < \beta \cdot \frac{\mu}{\mu + \nu}(1 - p_0)$$

and Remark 4 where it is shown that $\beta < 4$. In words (26) means that a necessary condition for the local stability of EE is that the "sensitivity" of the information-dependent component of coverage to changes in the proportions of susceptibles at the equilibrium is bounded. With respect to the LAS condition of EE in [7] the left relation in (26) is more restrictive.
By (27), it follows that, depending on the sign of $\partial g/\partial S$, one of the two conditions in (26) is satisfied. In particular, if $\partial g/\partial S = 0$ both inequalities in (26) are fulfilled and condition (22) reduces to:

$$\frac{(2-\mu)(\beta I_e - 2)}{\mu \beta I_e} - \frac{\nu}{\mu} < p_1'(g(S_e, I_e))\frac{\partial g}{\partial I}(S_e, I_e) < \frac{1}{\beta I_e} + \frac{1 - \mu - \nu}{\mu}. \quad (28)$$

The role played by the coverage function p_1 and by the "information" function g in the local stability condition (22) is twofold: on one side they act explicitly via their derivatives, on the other hand they act implicitly via the value of I_e as it is evident from (19). The same reasoning applies to conditions for bifurcations. These facts will be illustrated in the example of Section 6.

Remark 12 *With respect to (10), if we set $F(S, I) = S(t+1) - S(t)$, that is*

$$F(S, I) = \mu(1 - p_0 - p_1(g(S, I)) - \mu S - \beta S I$$

under the assumption $(1 - p_0) R_0 > 1$, we have $F(S_e, I_e) = 0$. As

$$\frac{\partial F}{\partial S}(S, I) = -\mu p_1'(g(S, I))\frac{\partial g}{\partial S}(S, I) - \mu - \beta I = -A$$

$$\frac{\partial F}{\partial I}(S, I) = -\mu p_1'(g(S, I))\frac{\partial g}{\partial I}(S, I) - \beta S$$

from the hypotheses $p_1' > 0$ and $\partial g/\partial I > 0$ it follows $\partial F/\partial I < 0$. Hence, by the implicit function theorem, it follows the existence and the uniqueness of a continuously differentiable function $h : (S_e - \delta, S_e + \delta) \to \mathbb{R}$, $\delta > 0$, such that $h(S_e) = I_e$ and

$$h'(S_e) = -\frac{A}{\mu p_1'(g(S, I))\frac{\partial g}{\partial I}(S_e, I_e) + \beta S_e}. \quad (29)$$

Comparing (29) with the right term in (22), we conclude the previous stability condition is equivalent to say that $h'(S_e) < -\beta I_e$*, in particular* I_e *is decreasing with respect to* S_e*.*

In this general framework we limit to give a sufficient condition to exclude two-periodic cycles.

Theorem 13 *Assuming (12), if*

$$p_1'(g(S(t), I(t))) \frac{\partial g}{\partial S}(S(t), I(t)) < \frac{2-\mu-\beta}{\mu} \tag{30}$$

for every $t \geq 0$*, system (10) does not admit any nontrivial cycle of period* 2*.*

Proof. To prove the theorem we use a result of McCluskey and Muldowney (Corollary 1(a) in [17]), by showing that matrix

$$\mathbf{I}_2 + J(S,I) = \begin{pmatrix} 2-\mu-\beta I - \mu p_1'(g(S,I)) \frac{\partial g}{\partial S}(S,I) & -\beta S - \mu p_1'(g(S,I)) \frac{\partial g}{\partial I}(S,I) \\ \beta I & 2-(\mu+\nu)+\beta S \end{pmatrix}$$

is definite positive on Ω, where $\mathbf{I}_2$ is the identity matrix of order 2 and $J(S,I)$ is the Jacobian of system (10). The main diagonal elements are positive by conditions (30) and i) in (12); the positivity of the determinant of $\mathbf{I}_2 + J(S,I)$, follows then by the positivity of $p_1'(g(S,I)) \frac{\partial g}{\partial I}(S,I)$. ■

5.2 Persistence

An important characterization of the long term endemic behavior is the study of the possibility that the endemic equilibrium EE may be globally asymptotically stable (GAS). Unfortunately we have not been able to demonstrate the GAS of the endemic state in the general case, though we will prove it for a special but important subcase in the next section.
We have, however, been able to prove a persistence result ([23]). More precisely our next result shows that if the residual BRN $(1-p_0)R_0$ is "sufficiently" greater than one then system (10) is persistent in the sense that it is possible to show the existence of a positive lower bound such that the asymptotic infective fraction lies always above it.

Proposition 14 *If* $(1-p_0)R_0 > 1$*, then:*

1. *there exists* $\xi \in (0, 1-p_0)$ *such that*

$$\liminf_{t\to+\infty} S(t) + I(t) \geq \xi;$$

2. *if* $(1-p_0)\gamma R_0 > 1$*, where*

$$\gamma := \frac{\mu(1-p)^{\inf}}{(\mu+\nu)(1-p_0)} \in (0,1)$$

it holds:

$$\liminf_{t\to+\infty} I(t) \geq \xi - S_e > 0.$$

Proof. By setting $\sigma(t) = S(t) + I(t)$ and summing the two equation in (10) gives

$$\begin{aligned} \sigma(t+1) &\geq (1-\mu-\nu)\,\sigma(t) + \mu\,(1 - p_0 - p_1(g(S(t), I(t))) \geq \\ &\geq (1-\mu-\nu)\,\sigma(t) + \mu\,(1-p)^{\inf} \end{aligned}$$

where the constant $(1-p)^{\inf}$ is defined in (11). The equation

$$y(t+1) = (1-\mu-\nu)\,y(t) + \mu\,(1-p)^{\inf}$$

admits the unique equilibrium $y^* = \mu\,(1-p)^{\inf}/(\mu+\nu)$ in $(0, 1-p_0)$ that is GAS. Therefore, by setting $\xi = y^*$, the first statement follows.
Since $(1-p_0)\,\gamma R_0 > 1$ there exists S_e and

$$\xi - S_e = \frac{\mu+v}{\beta}\left((1-p_0)\,R_0\frac{\mu\,(1-p)^{\inf}}{(\mu+\nu)\,(1-p_0)} - 1\right) > 0$$

therefore the linear system

$$\begin{cases} S + I = \xi \\ \beta S = \mu + \nu \end{cases}$$

admits the unique positive solution $(S_e, \xi - S_e)$. We have

$$\begin{aligned} I(t+1) - I(t) &= (\beta S(t) - (\mu+\nu))\,I(t) \geq \\ &\geq (\beta\,(S(t) + I(t) - (\xi - S_e)) + \beta\,(\xi - S_e - I(t)))\,I(t) \end{aligned}$$

hence $\xi - S_e \geq I(t)$ would imply $I(t+1) \geq I(t)$ asymptotically, a contradiction. We conclude that

$$\liminf_{t\to+\infty} I(t) \geq \xi - S_e > 0.$$

■

Remark 15 *The persistency property is a fundamental one on the epidemiological standpoint. Indeed, a major substantive epidemiological question is whether the infection will "persists" or not in the population over the long-term. This question bypasses the issue of stability i.e. whether solutions asymptotically get closer and closer to the endemic state or they oscillate about it (also with quite large amplitude) around it. In this perspective the proved persistency property represents a fairly general characterization of the dynamics of the system.*

6 A noteworthy case: piece-wise linear prevalence dependence.

We now look in more detail at the functional specification $g(S, I) = kI$, $k > 0$ that corresponds to the so called "pure prevalence-dependent" case, which is the most commonly investigated in the literature ([7]). In this case the public (e.g. parents of children eligible for vaccination) only react to changes in the current prevalence of infection, taken as a measure of the perceived risk of infection. Assuming further $p_1(x) = \min\{cx, 1-p_0\}$, $c > 0$, we obtain

$$p_1(g(S, I)) = \widehat{p}_1(I) = \min\{ckI, 1-p_0\}, \qquad I \in [0, 1) \tag{31}$$

and

$$(1-p)^{\inf} = \begin{cases} 1-(p_0+ck) & 0<p_0+ck<1 \\ 0 & p_0+ck\geq 1 \end{cases}. \tag{32}$$

System (10) writes as

$$\begin{aligned} S(t+1) &= (1-\mu)S(t) + \mu(1-p_0-\min\{ckI(t), 1-p_0\}) - \beta S(t)I(t) \\ I(t+1) &= (1-(\mu+\nu))I(t) + \beta S(t)I(t) \end{aligned} \tag{33}$$

Notice that if $0<p_0+ck<1$ then $p_1(g(S,I)) = ckI$, whereas when $p_0+ck\geq 1$ we know by Remark 2 that cannot eventually be $ckI(t) > 1-p_0$. In particular, when $(1-p_0)R_0 < 1$ the sequence $\{I(t)\}$ is strictly decreasing and then there exists $t_0 > 0$ such that for every $t \geq t_0$ we have $ckI(t) < 1-p_0$. For $(1-p_0)R_0 > 1$ the scenario can be more complicated, as illustrated in the sequel.

The invariance conditions (12) become:

$$\text{i)}\ \ \mu+\nu\leq 1; \qquad \text{ii)}\ \ \beta\leq\beta^* = \begin{cases} f(\mu,p_0,ck) & 0<p_0+ck<1 \\ 1-\mu & p_0+ck\geq 1 \end{cases} \tag{34}$$

where we have set for convenience

$$f(\mu,p_0,ck) = 1+\mu+2\left(\sqrt{(1-\mu p_0-\mu ck)(1-p_0-ck)\mu} - \mu(p_0+ck)\right). \tag{35}$$

As f is a positive, bounded and strictly decreasing function of both p_0 and ck, its maximum value (≤ 4) occurs for $p_0 = ck = 0$, corresponding to the case of no vaccination (see the general discussion in Remark 4).

Under the assumption $(1-p_0)R_0 > 1$, the infective fraction at endemic state can be computed explicitly from equation (19):

$$I_e^{(ckI)} = ((1-p_0)R_0 - 1)\left(\frac{\beta}{\mu} + ckR_0\right)^{-1} = \frac{I_E^o}{1+ck\frac{\mu}{\mu+\nu}}. \tag{36}$$

As expected, $I_e^{(ckI)}$ is a decreasing function of ck which tunes the reactivity of vaccination coverage to the perceived risk of infection. The following theorem shows the conditions under which the endemic state is LAS, by suggesting that prevalence-dependent vaccinating behaviour is an essentially stabilizing force.

Theorem 16 *The endemic equilibrium EE is LAS for every value of the parameters satisfying (34) if:*

i) $p_0+ck\geq 1$, $\forall\mu,\nu,\beta$;

ii) $0<p_0+ck<1$, $\forall\beta$ *and* $\forall\mu$ *and* ν *such that (7) is satisfied.*

Proof. We start by observing that $p_1\circ g$ is differentiable at (S_e,I_e). Since $\partial g/\partial S = 0$ and $p_1'(g(S_e,I_e))\frac{\partial g}{\partial I}(S_e,I_e) = ck$, the local stability condition (28) becomes:

$$\frac{(2-\mu)(\beta I_e-2)}{\mu\beta I_e} - \frac{\nu}{\mu} < ck < \frac{1}{\beta I_e} + \frac{1-\mu-\nu}{\mu}. \tag{37}$$

We set, without loss of generality, $k=1$. The right condition in (37) can be rewritten as

$$c - \frac{1-\mu-\nu}{\mu} < \frac{1}{\beta I_e} \tag{38}$$

then, it is always satisfied for $(0<)\ c \leq (1-\mu-\nu)/\mu$, in particular for

$$\frac{1-\mu-\nu}{\mu} \geq 1 \quad \Leftrightarrow \quad 1-2\mu-\nu \geq 0. \tag{39}$$

If $c > (1-\mu-\nu)/\mu$, with $1-2\mu-\nu<0$, by (36), with simple manipulations, we obtain the following equivalent version of (38):

$$\beta < \left(1+\mu+\nu+\frac{1}{\mu c+\mu+\nu-1}\right)\frac{1}{1-p_0} := g(\mu,\nu,p_0,c). \tag{40}$$

We first observe that (40) is satisfied for $ck \geq 1-p_0$, because in this case $\beta \leq 1-\mu$. Hence, we just proceed for $0<ck<1-p_0$.
Notice now that g is a strictly increasing and convex function of p_0, with $g \to +\infty$ for $p_0 \to 1^-$. This latter implies, as $\beta \leq 4$, that (40) holds true when p_0 belongs to a suitable left neighborhood of 1. We conclude our proof about (38), by showing that $g(\mu,\nu,p_0,c) > f(\mu,p_0,c)$ under the remaining admissible values of the parameters, where f was defined in (35). To this aim, since, as previously observed, $f(\mu,p_0,c)$ is decreasing with respect to p_0, by the continuity of f and g, we have to show that $g(\mu,\nu,0,c) > f(\mu,0,c)$ for all admissible values of μ, ν and c. This inequality is surely satisfied if $f(\mu,0,c) \leq 1+\mu+\nu$, equivalent to $c \geq \frac{4\mu-\nu^2}{4\mu(1+\mu+\nu)}$ (always true for $4\mu-\nu^2 \leq 0$).
Summing up, we have to solve the following system:

$$\begin{cases} \mu>0, \nu>0 \\ \max\left\{\frac{1-\nu}{2}, \frac{\nu^2}{4}\right\} < \mu \leq 1-\nu \\ \frac{1-\mu-\nu}{\mu} < c < \frac{4\mu-\nu^2}{4\mu(1+\mu+\nu)} \\ \nu+\frac{1}{\mu c+\mu+\nu-1} > 2\sqrt{(1-\mu c)(1-c)\mu}-2\mu c \end{cases}$$

Set

$$h_1(c) := \nu+\frac{1}{\mu c+\mu+\nu-1}; \qquad h_2(c) := 2\sqrt{(1-\mu c)(1-c)\mu}-2\mu c$$

and observe that h_1 and h_2 are both strictly decreasing functions of c:

$$\begin{aligned} h_1'(c) &= -\frac{\mu}{(\mu c+\mu+\nu-1)^2} < 0 \\ h_2'(c) &= -2\mu\left(\frac{1+\mu-2\mu c}{2\sqrt{(1-\mu c)(1-c)\mu}}+1\right) < 0. \end{aligned}$$

Then, we conclude our proof by showing that (intended as limits)

$$h_1\left(\frac{4\mu-\nu^2}{4\mu(1+\mu+\nu)}\right) > h_2\left(\frac{1-\mu-\nu}{\mu}\right).$$

With simple computations we obtain:

$$\begin{aligned} h_1\left(\frac{4\mu-\nu^2}{4\mu(1+\mu+\nu)}\right) &= \nu+\frac{4(1+\mu+\nu)}{4\mu-\nu^2+4\left((\mu+\nu)^2-1\right)}; \\ h_2\left(\frac{1-\mu-\nu}{\mu}\right) &= 2\left(\sqrt{(\mu+\nu)(2\mu+\nu-1)}-(1-\mu-\nu)\right). \end{aligned}$$

As

$$\frac{\partial}{\partial\mu}h_1\left(\frac{4\mu-\nu^2}{4\mu\left(1+\mu+\nu\right)}\right)=-\frac{4\left(4\mu^2+8\mu\nu+8\mu+5\nu^2+12\nu+8\right)}{\left(4\mu^2+8\mu\nu+4\mu+3\nu^2-4\right)^2}<0$$

the minimum value of $h_1\left(\frac{4\mu-\nu^2}{4\mu\left(1+\mu+\nu\right)}\right)$ is attained for $\nu=1-\mu$:

$$h_1\left(\frac{4\mu-\nu^2}{4\mu\left(1+\mu+\nu\right)}\right)=1-\mu+\frac{8}{4\mu-\left(1-\mu\right)^2}>2.$$

Furthermore, as

$$\frac{\partial}{\partial\mu}h_2\left(\frac{1-\mu-\nu}{\mu}\right)=1+\frac{4\mu+3\nu-1}{2\sqrt{\left(\mu+\nu\right)\left(2\mu+\nu-1\right)}}>0$$

the function $h_2\left(\frac{1-\mu-\nu}{\mu}\right)$ attains its maximum value for $\nu=1-\mu$:

$$h_2\left(\frac{1-\mu-\nu}{\mu}\right)=2\sqrt{\mu}<2.$$

Consider now the left condition in (37). We first observe that

$$\beta I_e-2\leq 0\quad\Leftrightarrow\quad\beta\leq\frac{\left(2+\mu\right)\left(\mu+\nu\right)+2\mu c}{\mu\left(1-p_0\right)}.$$

Therefore, if $c>1-p_0$ the r.h.s. is greater than 2 but $\beta\leq 1-\mu$ and the left condition in (37) is satisfied. Assume $c<1-p_0$. The left condition in (37) is equivalent to

$$\beta<\left(\left(\mu+\nu\right)+\frac{2\left(2-\mu\right)\left(\mu+\nu+\mu c\right)}{\left(2-\mu-\nu-\mu c\right)\mu}\right)\frac{1}{1-p_0}\tag{41}$$

without any particular restriction on the parameters. As the r.h.s. of (41) increases with p_0 we have to compare this term with the previously considered function f for $p_0=0$. If we set:

$$\begin{aligned}h_3\left(c\right)&=\nu+\frac{2\left(2-\mu\right)\left(\mu+\nu+\mu c\right)}{\left(2-\mu-\nu-\mu c\right)\mu}\\h_4\left(c\right)&=1+2\left(\sqrt{\left(1-\mu c\right)\left(1-c\right)\mu}-\mu c\right)\end{aligned}$$

we can note that

$$h_3'\left(c\right)=\frac{4\left(2-\mu\right)}{\left(\mu+\nu+c\mu-2\right)^2}>0$$

then h_3 is strictly increasing, while h_4 (a translation of h_2) is strictly decreasing. Therefore, in order $h_3\left(c\right)>h_4\left(c\right)$ for all admissible values of μ, ν and c it must be $h_3\left(0\right)>h_4\left(0\right)$. But this condition coincides with (6) and this concludes the proof. ■

Remark 17 *Comparing the previous result with the corresponding one about Allen's model in Section 2, and taking into account Remark 4, we conclude that prevalence-dependent vaccinating behavior is a stabilizing force for discrete time SIR models. This can be further detailed by introducing some sufficient conditions for the local stability of EE. As an example, observe that if* $I_e \leq S_e$, *then*

$$\beta I_e = (\mu + \nu)\frac{I_e}{S_e} < 2$$

and the left condition in (37) is satisfied too, so EE is LAS. Finally, we note that, thanks to Theorem 13, there are no two-periodic cycles when $\beta < 2 - \mu$.

We now prove a result which provides a simple and epidemiologically interpretable condition for the global stability of the endemic state, by following a recent approach presented in [15].

Theorem 18 *If* p_1 *is given by (31) and*

$$1 + \frac{\nu + \mu ck}{\mu} < (1 - p_0) R_0 < 1 + \min\left\{\frac{1}{\mu + \nu}, \frac{\mu}{\nu + \mu ck}\right\} \tag{42}$$

$$ck < \min\left\{1 - p_0, 1 - \frac{\nu}{\mu}\right\} \tag{43}$$

the EE of (33) is GAS.

Proof. System (33) thanks to (43) simplifies in

$$\begin{array}{l} \sigma(t+1) = (1-\mu)\sigma(t) + \mu(1-p_0) - (\nu + \mu ck) I(t) \\ I(t+1) = I(t)(1 - (\mu + \nu)) + \beta I(t)(\sigma(t) - I(t)) \end{array} \tag{44}$$

where we have set $\sigma(t) = S(t) + I(t)$. Therefore for all $t > 0$:

$$(1 - \mu - \nu - \mu ck)\sigma(t) + \mu(1 - p_0) \leq \sigma(t+1) \leq (1-\mu)\sigma(t) + \mu(1-p_0). \tag{45}$$

It is a simple task to verify that the affine equations

$$X(t+1) = (1-\mu)X(t) + \mu(1-p_0) \tag{46}$$

$$Y(t+1) = (1 - \mu - \nu - \mu ck) Y(t) + \mu(1 - p_0) \tag{47}$$

have respectively the globally stable equilibria

$$\alpha_1^u = 1 - p_0; \qquad \alpha_1^l = \frac{\mu(1-p_0)}{\mu(1+ck) + \nu}. \tag{48}$$

Therefore, by (45) for any $\varepsilon > 0$ there exists $T_1 > 0$ such that

$$\boldsymbol{\sigma}_1^l = \alpha_1^l - \varepsilon \leq \boldsymbol{\sigma}(t) \leq \alpha_1^u + \varepsilon = \boldsymbol{\sigma}_1^u \tag{49}$$

for $t > T_1$. Substituting (49) in the second equation of (44), we obtain

$$I(t)\left(1 - (\mu + \nu) + \beta \boldsymbol{\sigma}_1^l\right) - \beta I(t)^2 \leq I(t+1) \leq I(t)\left(1 - (\mu + \nu) + \beta \boldsymbol{\sigma}_1^u\right) - \beta I(t)^2. \tag{50}$$

The following equation corresponding to the second equality in (50):

$$X(t+1) = X(t)\left(1 - (\mu + \nu) + \beta \boldsymbol{\sigma}_1^u\right) - \beta X(t)^2 \tag{51}$$

by the change of variable $X(t) = (1-(\mu+\nu)+\beta\boldsymbol{\sigma}_1^u)\,x(t)/\beta$ is equivalent to the logistic one

$$x(t+1) = r_x x(t)(1-x(t)) \tag{52}$$

where $r_x = 1-(\mu+\nu)+\beta\boldsymbol{\sigma}_1^u$. It is well known that if $r_x \in (1,3)$ then equation (52) has the unique positive equilibrium $(1-\frac{1}{r_x})$ globally asymptotically stable on $(0,1)$. As

$$r_x = 1-(\mu+\nu)+\beta(1-p_0+\varepsilon) = 1+(\mu+\nu)((1-p_0)R_0-1)+\beta\varepsilon$$

and $(1-p_0)R_0 > 1$ we get $r_x > 1$. On the other hand, by (34), $\mu+\nu<1$ and $0<\widetilde{p}_0<1$, the upper bound in (42) gives $r_x \le 2+\beta\varepsilon$.
Operating in the same way with the equation corresponding to the first equality in (50)

$$Y(t+1) = Y(t)\left(1-(\mu+\nu)+\beta\boldsymbol{\sigma}_1^l\right) - \beta Y(t)^2 \tag{53}$$

we get

$$y(t+1) = r_y y(t)(1-y(t)), \qquad r_y = 1-(\mu+\nu)+\beta\boldsymbol{\sigma}_1^l. \tag{54}$$

Since

$$r_y = 1+\frac{\mu+\nu}{\mu(1+ck)+\nu}\left((1-p_0)R_0-1+ck+\frac{\nu}{\mu}\right)-\beta\varepsilon$$

by the lower bound in (42) and the arbitrariness of ε, it follows $r_y > 1$. Furthermore, by the upper bound in (42) we find:

$$r_y \le 1-\mu-\nu+\frac{\mu(1+\mu+\nu)}{\mu(1+ck)+\nu}-\beta\varepsilon < 3.$$

Then, we conclude that equations (51) and (53) admit respectively the positive equilibria $X_E = \boldsymbol{\sigma}_1^u - \frac{1}{R_0}$ and $Y_E = \boldsymbol{\sigma}_1^l - \frac{1}{R_0}$ that are globally asymptotically stable. This implies the existence of $T_{1I} \ge T_{1\boldsymbol{\sigma}}$ such that

$$\boldsymbol{\sigma}_1^l - \frac{1}{R_0} - \varepsilon = I_1^l \le I(t) \le I_1^u = \boldsymbol{\sigma}_1^u - \frac{1}{R_0} + \varepsilon \tag{55}$$

for $t \ge T_{1I}$. Substituting (55) into system (44), we get

$$\begin{aligned} \boldsymbol{\sigma}(t+1) &\le (1-\mu)\boldsymbol{\sigma}(t)+\mu(1-p_0)-(\nu+\mu ck)I_1^l \\ \boldsymbol{\sigma}(t+1) &\ge (1-\mu)\boldsymbol{\sigma}(t)+\mu(1-p_0)-(\nu+\mu ck)I_1^u. \end{aligned} \tag{56}$$

Operating as before, there exists a positive integer $T_{2\boldsymbol{\sigma}} > T_{1I}$ such that

$$\frac{\mu(1-p_0)-(\nu+\mu ck)I_1^u}{\mu}-\varepsilon = \boldsymbol{\sigma}_2^l \le \boldsymbol{\sigma}(t) \le \boldsymbol{\sigma}_2^u = \frac{\mu(1-p_0)-(\nu+\mu ck)I_1^l}{\mu}+\varepsilon \tag{57}$$

for all $t \ge T_{2\boldsymbol{\sigma}}$. Notice that:

$$\sigma_1^u - \sigma_2^u = \frac{\nu+\mu ck}{R_0(\mu(1+ck)+\nu)}\left((1-p_0)R_0-\frac{\mu(1+ck)+\nu}{\mu}\right)+\frac{\nu+\mu ck}{\mu}\varepsilon > 0$$

and by (42)

$$\sigma_2^l - \sigma_1^l = \frac{(\nu+\mu ck)^2}{R_0}\frac{\frac{\mu(1+ck)+\nu}{\nu+ck\mu}-(1-p_0)R_0}{\mu(\mu(1+ck)+\nu)}-\frac{\nu+\mu ck}{\mu}\varepsilon > 0.$$

Substituting (58) into the second equation of (44), we find:

$$\begin{aligned} I(t+1) &\leq I(t)(1-(\mu+\nu)+\beta\boldsymbol{\sigma}_2^u)-\beta I(t)^2 \\ I(t+1) &\geq I(t)\left(1-(\mu+\nu)+\beta\boldsymbol{\sigma}_2^l\right)-\beta I(t)^2. \end{aligned} \tag{58}$$

Operating as before, there exists $T_{2I} \geq T_{2\boldsymbol{\sigma}}$ such that $I_2^l \leq I(t) \leq I_2^u$ where

$$I_2^u = \boldsymbol{\sigma}_2^u - \frac{1}{R_0} + \varepsilon; \qquad I_2^l = \boldsymbol{\sigma}_2^l - \frac{1}{R_0} - \varepsilon.$$

Substituting the expressions of $\boldsymbol{\sigma}_2^l$ and $\boldsymbol{\sigma}_2^u$, we get

$$\begin{aligned} I_2^u &= -\frac{\nu+\mu ck}{\mu} I_1^l + \frac{(1-p_0)R_0-1}{R_0} + 2\varepsilon \\ I_2^l &= -\frac{\nu+\mu ck}{\mu} I_1^u + \frac{(1-p_0)R_0-1}{R_0} - 2\varepsilon. \end{aligned}$$

These last equations hold when $t > T_{2I}$ and $I_2^l \leq I(t) \leq I_2^u$. By induction, there exist sequences $\{T_{k\boldsymbol{\sigma}}\}$, $\{T_{kI}\}$, $\{\boldsymbol{\sigma}_k^u\}$, $\{\boldsymbol{\sigma}_k^l\}$, $\{I_k^l\}$ and $\{I_k^u\}$ such that $I_k^l \leq I(t) \leq I_k^u$ for all $t > T_{kI}$ and

$$\begin{aligned} I_{k+1}^u &= -\frac{\nu+\mu ck}{\mu} I_k^l + \frac{(1-p_0)R_0-1}{R_0} + 2\varepsilon \\ I_{k+1}^l &= -\frac{\nu+\mu ck}{\mu} I_k^u + \frac{(1-p_0)R_0-1}{R_0} - 2\varepsilon. \end{aligned}$$

The last system has the positive equilibrium

$$\begin{aligned} I_E^u &= \frac{((1-p_0)R_0-1)\mu}{R_0(\mu+\nu+\mu ck)} + \frac{2\mu}{\mu-\nu-\mu ck}\varepsilon \\ I_E^l &= \frac{((1-p_0)R_0-1)\mu}{R_0(\mu+\nu+\mu ck)} - \frac{2\mu}{\mu-\nu-\mu ck}\varepsilon \end{aligned}$$

If λ_1 and λ_2 denote the eigenvalues of the coefficient matrix

$$\begin{pmatrix} 0 & -\frac{\nu+\mu ck}{\mu} \\ -\frac{\nu+\mu ck}{\mu} & 0 \end{pmatrix}$$

we have $|\lambda_1| = |\lambda_2| = \frac{1}{\mu}(\nu + ck\mu) < 1$ by assumption. Therefore, the equilibrium $\left(I_E^l, I_E^u\right)$ is GAS. Notice that

$$\lim_{\varepsilon\to 0^+} I_E^l(\varepsilon) = \lim_{\varepsilon\to 0^+} I_E^u(\varepsilon) = \frac{((1-p_0)R_0-1)\mu}{R_0(\mu+\nu+\mu ck)}.$$

Since $I_k^l \leq I(t) \leq I_k^u$ we obtain

$$\lim_{t\to+\infty} I(t) = \frac{((1-p_0)R_0-1)\mu}{R_0(\mu+\nu+\mu ck)}.$$

Similarly, the sequences $\{\boldsymbol{\sigma}_k^u\}$ and $\left\{\boldsymbol{\sigma}_k^l\right\}$ satisfy the affine system

$$\begin{aligned} \boldsymbol{\sigma}_{k+1}^l &= -\frac{\nu+\mu ck}{\mu}\boldsymbol{\sigma}_k^u + \frac{\mu(1-p_0)-(\nu+\mu ck)\left(-\frac{1}{R_0}+\varepsilon\right)}{\mu} - \varepsilon \\ \boldsymbol{\sigma}_{k+1}^u &= -\frac{\nu+\mu ck}{\mu}\boldsymbol{\sigma}_k^l + \frac{\mu(1-p_0)-(\nu+\mu ck)\left(-\frac{1}{R_0}+\varepsilon\right)}{\mu} + \varepsilon \end{aligned}$$

Operating as before, it is easy to verify that

$$\lim_{\varepsilon\to 0^+}\lim_{k\to+\infty}\boldsymbol{\sigma}_k^u=\lim_{\varepsilon\to 0^+}\lim_{k\to+\infty}\boldsymbol{\sigma}_k^l=\frac{\mu\left(1-p_0\right)R_0+\left(\nu+\mu ck\right)}{R_0\left(\nu+\mu ck+\mu\right)}=\lim_{t\to+\infty}\sigma\left(t\right)$$

and then:

$$\lim_{t\to+\infty}S\left(t\right)=\frac{\mu\left(1-p_0\right)R_0+\left(\nu+\mu ck\right)}{R_0\left(\nu+\mu ck+\mu\right)}-\frac{\left(\left(1-p_0\right)R_0-1\right)\mu}{R_0\left(\mu+\nu+\mu ck\right)}=\frac{1}{R_0}$$

that is the endemic equilibrium is GAS. ■

Remark 19 *When they are non-empty, the conditions (42)-(43) essentially state that, provided the residual reproduction number is appropriately bounded, the endemic state will be GAS if the responsiveness of the prevalence dependent component of vaccine uptake to changes in prevalence is sufficiently moderate. In simple words the endemic state will be GAS if, provided the infection is sufficiently controlled by the baseline vaccination coverage p_0, individual reactions to changing perceived risk of infection are not too violent. This appears to be epidemiologically meaningful. Unfortunately we have not been able to prove that the result holds for any value of the residual BRN ensuring the infection persistence.*

7 Illustrations and simulation results

In this section we illustrate by numerical examples some of the previous results on the piecewise linear prevalence-dependent case and supply some further considerations.

We start by observing that the convergence to EE in model (33) can assume very different patterns in dependence on the size of ck (see Figure 2). It is to be noted that, for sufficiently large values of the behavioral response parameter ck, there might be an initial, transient, phase where the "roof" of 100% vaccine uptake can be achieved. However this phase will eventually end and the system will unavoidably enter its long term stable regime, with (oscillatory) convergence to the endemic state.

Figure 2. Time pattern of convergence to the endemic equilibrium of the susceptible and infected fractions in system (33) when $ck+p_0>1$.

For what concerns bifurcations, by Theorem 16 we know that in the admissible region of parameters flip bifurcations are possible (see Figure 3). To check for the possibility of a flip bifurcation we have to solve (for $k=1$) the equation $1+\det J_e+\mathrm{tr}J_e=0$, that is:

$$4-2\left(\mu+\beta I_e\right)+\beta I_e\left(\mu+\nu+\mu c\right)=0$$

from which we obtain the flip bifurcation value (note that the denominator is always positive)

$$\beta^{FLIP}=\frac{\left(\mu+\nu\right)\left(4-\mu^2-\nu\mu\right)+c\mu\left(4-\mu\left(\mu+\nu+2\right)\right)}{\left(1-p_0\right)\mu\left(\left(2-\mu-\nu\right)-c\mu\right)}.$$

A straightforward computation shows that β^{FLIP} is strictly increasing with respect to both c and p_0. As an illustration, by setting (as for the Allen model) $\mu = 0.81$, $\nu = 0.05$, and $p_0 = 0.02 = c$, with $\beta^* = 3.48$ we found $\beta^{FLIP} = 3.215$. The bifurcation diagram of Figure 3 illustrates this case. It should be noted that, consistent with Theorem 16, it suffices to take $c = 0.06$, the others parameters fixed, in order that β^{FLIP} not longer belongs to the positivity region.

Figure 3. Bifurcation diagram for system (33) when $\mu = 0.81$, $\nu = 0.05$, $p_0 = 0.02 = c$, $\beta^{FLIP} = 3.215$ and $\beta^* = 3.48$.

Differently from the Allen model, Neimark-Sacker bifurcations can appear, though only outside of the positivity set. Therefore any result in this last case depends on the initial conditions, thus losing generality, and could correspond to no epidemiologically meaningful parameter constellations (see Figure 4). For the sake of the mathematical completeness, we nonetheless report the condition for Neimark-Sacker bifurcations. These are clear and therefore highlight the underlying mechanisms; moreover, our simulations suggest that they can live at least in appropriate time windows.
In order to have a Neimark-Sacker bifurcation of the endemic state EE, it must be in the notation of Section 5.1 (always for $k = 1$):

$$\mu + \beta I_e = \beta I_e \left(\beta S_e + \mu c\right) \tag{59}$$

with

$$\beta I_e \left(\mu + \nu + \mu c\right) < 4. \tag{60}$$

It is immediate to verify that (59) and (60) respectively imply $\det J_e = 1$ and $\left(\mathrm{tr} J_e\right)^2 < 4$ (that is $0 < \mu + \beta I_e < 4$). By the proof of Theorem 16, we know that (59) is equivalent to (for $\mu + \nu + \mu c - 1 > 1$)

$$\beta = \left(1 + \mu + \nu + \frac{1}{\mu c + \mu + \nu - 1}\right) \frac{1}{1 - p_0} \tag{61}$$

that is not compatible with the admissible value of the parameters stated in (34).
Moreover, since

$$\lambda = \frac{1}{2}\left(2 - (\mu + \beta I_e) + i\sqrt{(\mu + \beta I_e)\left(4 - (\mu + \beta I_e)\right)}\right)$$

and

$$\begin{aligned}
\lambda^2 &= \frac{1}{4}\left(2A^2 - 8A + 4 + 2i\left(2 - A\right)\sqrt{A\left(4 - A\right)}\right) \\
\lambda^3 &= \frac{1}{2}\left(\left(2 - A\right)\left(\left(2 - A\right)^2 - 3\right) + i\left(\left(2 - A\right)^2 - 1\right)\sqrt{A\left(4 - A\right)}\right) \\
\lambda^4 &= \frac{1}{2}\left(2 + \left(2 - A\right)^4 - 4\left(2 - A\right)^2 - i\left(2 - A\right)\left(2 - \left(2 - A\right)^2\right)\sqrt{A\left(4 - A\right)}\right)
\end{aligned}$$

one easily verify that $\lambda^i \neq 1$ for $i = 1, 2, 3, 4$.
Furthermore, since

$$\left|\lambda\left(\beta\right)\right| = \sqrt{1 - \mu\frac{\mu c + \left(1 - p_0\right)\beta}{\mu + \nu + \mu c} + \mu\left(\left(1 - p_0\right)\beta - \left(\mu + \nu\right)\right)}$$

we obtain:

$$\frac{d}{d\beta}\left|\lambda\left(\beta\right)\right| = \frac{\mu\left(1-p_0\right)\left(\mu+\nu+\mu c-1\right)}{2\left(\mu+\nu+c\mu\right)\left|\lambda\left(\beta\right)\right|} > 0.$$

If we fix μ, ν and p_0 starting from (61) we can express the bifurcation value β^{bif} of the constant as a function of c. For example, if we choose the parameter constellation:

$$\mu = 0.01; \qquad \nu = 0.02; \qquad p_0 = 0.75$$

we have $\beta^{bif}\left(c\right) = 4.12 + \frac{4}{0.01c - 0.97}$. In the instability region the behavior nearby the bifurcating value β^{bif} is represented by stable oscillations. Figure 4 reports the shape of one closed invariant curve arising for a value of c very close to the bifurcation threshold.

Figure 4. A stable closed invariant curve emerging from the Neimark-Sacker bifurcation of the endemic state in model (33) for a value of the bifurcation parameter β very close to the bifurcation threshold. Top-left panel: time trend of the susceptible fraction; top-right: time trend of the infective fraction; bottom-left: phase-plane behaviour in the (S, I) plane. Notice that β does not respect its upper bound and yet the dynamics takes place in the feasible region.

8 Discussion

Behavioral epidemiology of transmissible infections is a fast developing new discipline aiming to include human behavior in models of infection spread. This paper has made an attempt to fill a gap in the behavioral epidemiology literature by investigating the dynamic properties of a deterministic discrete time model for a SIR infection with vaccine coverage specified by a phenomenological function of the currently perceived risk of infection. Besides some general characterizations on existence of equilibria, their local stability and system persistence, we have focused on the noteworthy subcase where the perception of infection risk by families of children eligible for vaccination is prevalence-dependent. In this case we have been able to supply a fairly general mathematical characterization of system properties, including conditions for the global stability of the endemic state. In particular the conditions for the global stability of the endemic state are neat and state that the GAS will hold provided the infection is sufficiently well controlled by the behavior-independent component of vaccination coverage, if individual reactions to changing perceived risk of infection are not too violent. This is a clear and epidemiologically meaningful result.

Future work will consider: (a) more general functional forms for the prevalence-dependent component of the vaccination function; (b) we will consider, following the lines of [7], [8] and [6], that both there may exist a delay in the information (due to the fact that the process of notification of cases sometime can be quite slow, and also the fact that laboratory analyses are not instantaneous) as well as another phenomenon of epidemiological relevance, i.e. that the agents may take into account their memory of the past epidemics; (c) alternative specifications of the underlying discrete-time mechanism linking current and past values of state variables, e.g. in the sense of [13], [14], [18].

Acknowledgments

We thank Francesca Centrone for her helpful comments, and two anonymous referees whose precious comments and suggestions have substantially improved our work. Anyway, usual disclaimers apply.

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9 Appendix

Proposition 20 *If (31) holds, then the solutions of (10) are in* Ω *for all* $t > 0$ *in the following cases:*

1. *if* $\beta + \mu \leq 1$ *for all* $ck > 0$*;*
2. *if* $\beta + \mu > 1$ *for*

$$0 < ck < 1 - p_0 - \frac{(1 - \beta - \mu)^2}{4\beta\mu}. \tag{62}$$

Proof. If $\beta + \mu \leq 1$ then condition ii) in (12) is fullfilled as $p^{\inf} \geq 0$.
If $\beta + \mu > 1$ then positivity is compatible only with the case $0 < ck < 1 - p_0$ and $p^{\inf} = 1 - p_0 - ck$. So, condition ii) in (12) becomes:

$$\beta \leq 1 - \mu + 2\mu\left(1 - p_0 - ck\right) + 2\sqrt{\left(1 - \mu p_0 - ck\mu\right)\left(1 - p_0 - ck\right)\mu}$$

equivalent to

$$2\mu\left(ck-(1-p_0)\right)-1+\beta+\mu\leq 2\sqrt{\left(1-\mu p_0-ck\mu\right)\mu\left(1-p_0-ck\right)}. \tag{63}$$

Hence, if (the l.h.t. is non positive, i.e.)

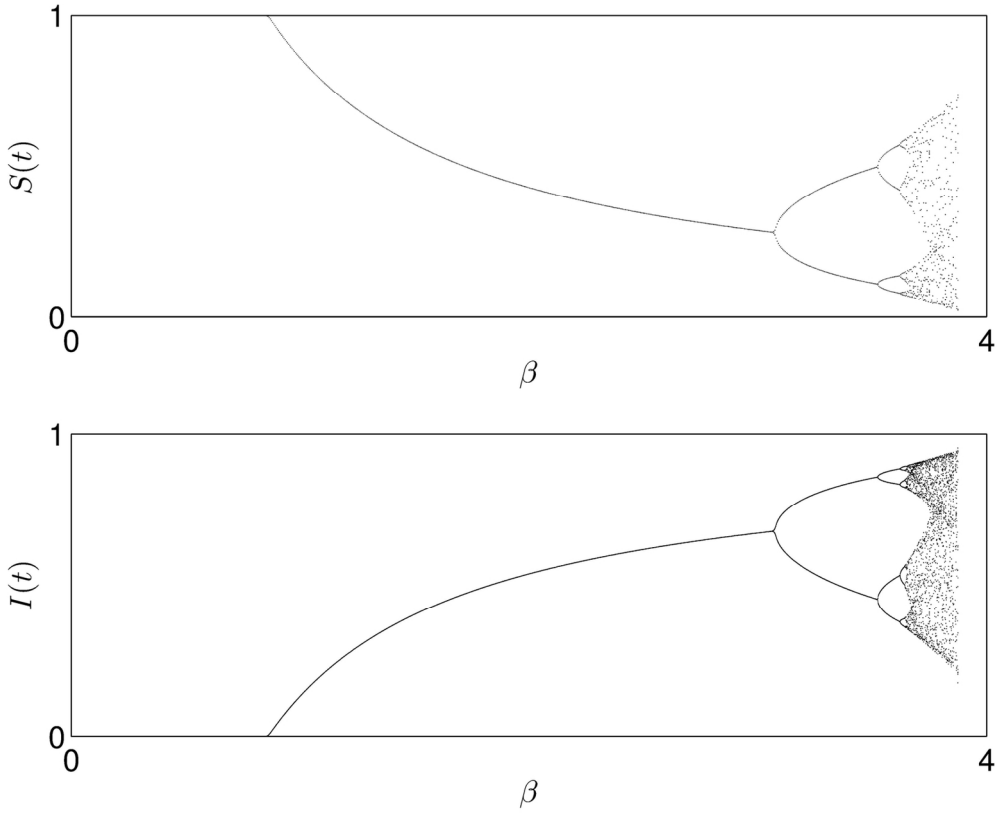
$$0<ck\leq 1-p_0+\frac{1-\mu-\beta}{2\mu} \tag{64}$$

then (63) is fulfilled. Otherwise, inequality (63) is equivalent to

$$\begin{cases} 1-p_0+\frac{1-\mu-\beta}{2\mu}<ck<1-p_0 \\ 0<ck\leq 1-p_0-\frac{(1-\beta-\mu)^2}{4\beta\mu} \end{cases} \tag{65}$$

and conclusion 2. follows by $\frac{1-\mu-\beta}{2\mu}<-\frac{(1-\beta-\mu)^2}{4\beta\mu}$ when $1-\mu-\beta<0$.

■



Role of the transmission parameter β in triggering period-doublig bifurcation in Allen's model [1] under Allen parameter constellation (μ=0.81, v=0.05).
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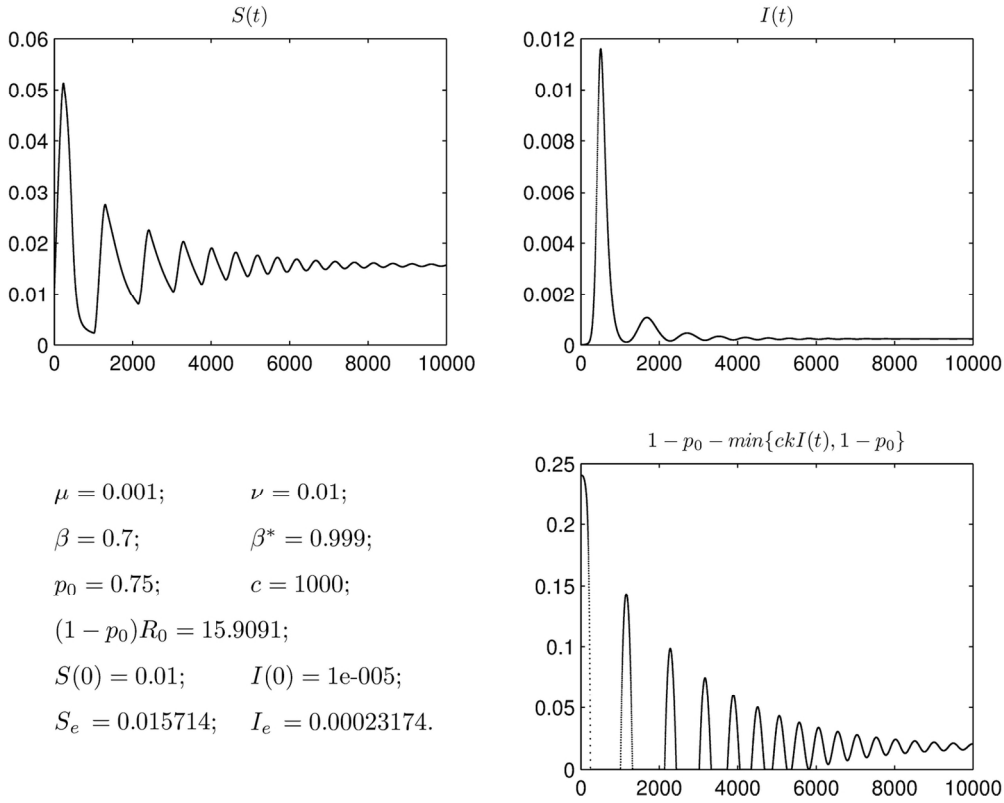

Time pattern of convergence to the endemic equilibrium of the susceptible and infected fractions in system (27) when $ck+p_0>1$.
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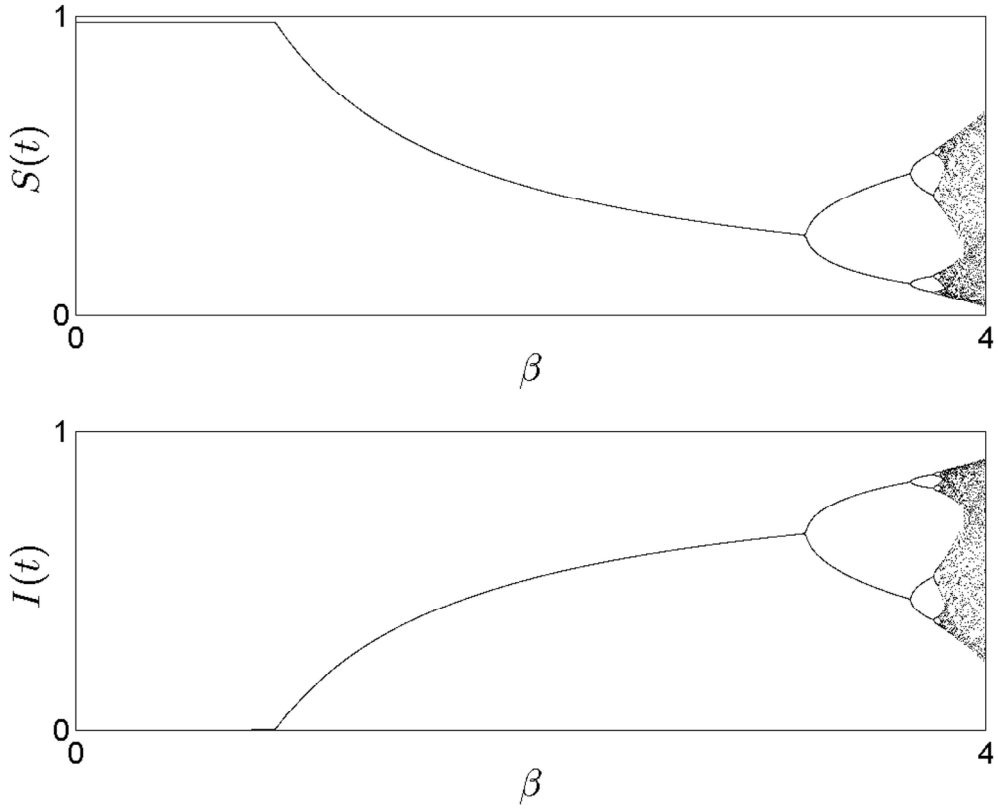
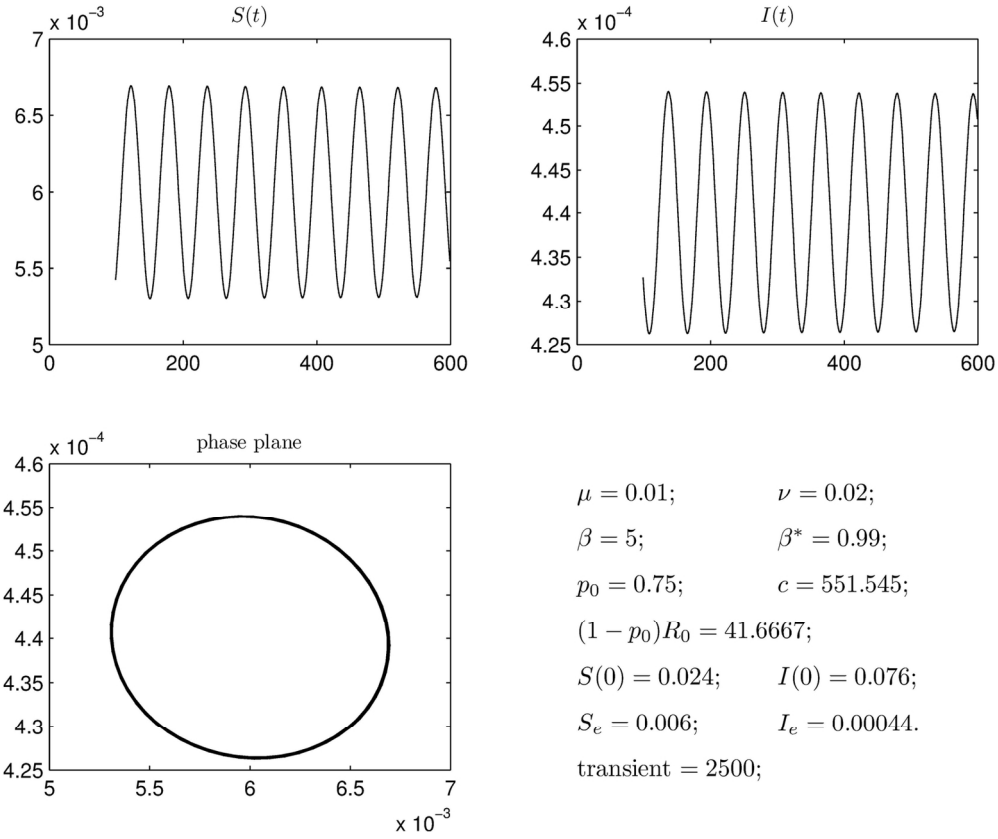


Bifurcation diagram for system (32) when μ=0.81, v=0.05, p_0=0.02=c, β^{FLIP}=3.215 and β^{∗}=3.48.
142x117mm (300 x 300 DPI)



$\mu = 0.01;$ $\nu = 0.02;$

$\beta = 5;$ $\beta^* = 0.99;$

$p_0 = 0.75;$ $c = 551.545;$

$(1 - p_0)R_0 = 41.6667;$

$S(0) = 0.024;$ $I(0) = 0.076;$

$S_e = 0.006;$ $I_e = 0.00044.$

$\text{transient} = 2500;$

A stable closed invariant curve emerging from the Neimark-Sacker bifurcation of the endemic state in model (32) for a value of the bifurcation parameter β very close to the bifurcation threshold. Top-left panel: time trend of the susceptible fraction; top-right: time trend of the infective fraction; bottom-left: phase-plane behaviour in the (S,I) plane. Notice that β does not respect its upper bound and yet the dynamics takes place in the feasible region.
142x118mm (300 x 300 DPI)