

Research paper

Pulse vaccination in a SIR model: Global dynamics, bifurcations and seasonality[☆]

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ABSTRACT

We analyze a periodically-forced dynamical system inspired by the SIR model with impulsive vaccination. We fully characterize its dynamics according to the proportion p of vaccinated individuals and the time T between doses. If the *basic reproduction number* is less than 1 (i.e. $\mathcal{R}_p < 1$), then we obtain precise conditions for the existence and global stability of a disease-free T -periodic solution. Otherwise, if $\mathcal{R}_p > 1$, then a globally stable T -periodic solution emerges with positive coordinates.

We draw a bifurcation diagram (T, p) and we describe the associated bifurcations. We also find analytically and numerically chaotic dynamics by adding seasonality to the disease transmission rate. In a realistic context, low vaccination coverage and intense seasonality may result in unpredictable dynamics. Previous experiments have suggested chaos in periodically-forced biological impulsive models, but no analytic proof has been given.

1. Introduction

Mathematical models have proved to be a useful tool in epidemiology, providing insights into the dynamics of infectious diseases and giving insights to improve strategies to combat their spread [1,2]. In particular, the SIR model, which classifies individuals into *Susceptibles* (S), *Infectious* (I) and *Recovered* (R), has been extensively used [3,4].

A prompt response is crucial once a disease emerges in a population. A range of approaches have been explored [5–7], with vaccination being one of the most effective ways to stop the progression [8,9].

Among the vaccination strategies in SIR models, we can point out three types:

- (1) *constant vaccination*, where a fixed proportion of the population is vaccinated [10–12], as in the administration of the BCG vaccine against tuberculosis to newborns;
- (2) *pulse vaccination*, which involves vaccinating a percentage of the *Susceptible* individuals periodically [12–15], similar to mass vaccination campaigns using oral polio vaccine in areas with polio outbreaks; and
- (3) *mixed vaccination* [12], as the hepatitis B vaccination program, which starts with one shot immediately after birth, followed by subsequent shots.

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Identifying the most suitable vaccination strategy represents a challenge, requiring considerations of effectiveness and costs associated with public health policies. Although considerable literature considers the logistic growth of the susceptible population, seasonality and the effect of vaccination, the combination of them remains unexplored.

Pulse vaccination over a proportion p of the population may be a strategy to stop the progression of an infectious disease [12,15–17]. The effective strategy must be in a way that the proportion p of vaccination is at the target level needed for the disease eradication, and the time T between doses must be suitable. Finding the most appropriate pair (T, p) for specific models is an open problem.

Some authors highlight the presence of seasonal forces in epidemic models such as school holidays, climate change, and political decisions [18]. While the seasonal impact is negligible for some diseases, for others such as childhood illnesses and influenza, it may have dramatic consequences in the dynamics of the models [19]. Differential equations adjust transmission rates using periodic functions [20], making them more complex than standard models but also more realistic [21,22].

This work focuses on the application of a modified SIR model with pulse vaccination and subject to seasonality, a promising unexplored field.

State of the art

¹ An optimal design of a vaccination program requires, apart from financial and logistical considerations, subtle results in epidemiology that are currently not available in the literature. Several works focus on the study of epidemiological models with pulse vaccination.

In 2002, Lu et al. [15] explored constant and pulse vaccination strategies in an SIR model with vertical and horizontal transmission. Authors showed that the effectiveness of the vaccination strategy depended on the interval between boosters. They observed that the high susceptibility in the offspring of infected parents accelerates stabilization, emphasizing the importance of parental health. Numerical simulations supported their findings.

Wang [23] studied the periodic oscillation of seasonally forced epidemiological models with pulse vaccination. Using Mawhin's coincidence degree method, the author confirmed the existence of positive periodic solutions for these SIR models with pulse vaccination. The effectiveness of this vaccination strategy was supported by numerical simulations. For further investigation using Mawhin's degree of coincidence method, we address the reader to [24–27] for a more in-depth understanding.

Meng and Chen [16] analyzed a SIR epidemic model with vertical and horizontal disease transmission. The authors also revealed that under some conditions, the system is *permanent*. Moreover, for $\mathcal{R}_0 > 1$ (*basic reproduction number*), the system under consideration exhibits positive periodic solutions.

Using data from Thailand, authors of [28] concluded that high vaccination coverage is not enough to eradicate measles without an optimized schedule for vaccine shots.

In 2022, authors of [19] investigated a periodically-forced dynamical system inspired by the SIR model. They provided a rigorous proof of the existence of observable chaos, expressed as persistent *strange attractors* on subsets of parameters for $\mathcal{R}_0 < 1$, where \mathcal{R}_0 stands for the basic reproduction number. Their results are in line with the empirical belief that intense seasonality can induce chaotic behavior in biological systems.

In 2023, Ibrahim and Dénes [29] developed a seasonal mathematical model to study the transmission of measles, applied to real data of Pakistan. The authors found that measles can become endemic and repeat annually when $\mathcal{R}_0 > 1$. The study showed that increasing vaccination coverage and effectiveness is crucial to reducing transmission and mitigating future outbreaks.

In a recent study by Guan et al. [30], an impulsive model was applied to rubella infection data in China. The study evaluated the effectiveness of the impulse vaccination strategy in eradicating rubella, incorporating environmental and genetic factors. Their findings highlighted that pulse vaccination can successfully eradicate rubella under favorable conditions.

Achievements

The present work provides insights into the interplay between seasonality, impulsive differential equations and the presence of horseshoes in periodically-forced epidemic models. The main goals of this article are the rigorous proof of the following assertions:

- (1) in the absence of seasonality, the model goes through five scenarios when varying the parameters of the period T of the vaccine and the proportion of *Susceptible* individuals vaccinated p ;
- (2) in the absence of seasonality, we design an optimum vaccination program as a function of the period T of pulse vaccination and the proportion p of *Susceptible* individuals that need to be vaccinated to control the disease;
- (3) under the presence of seasonality in the rate transmission rate of the disease, the system may behave chaotically and exhibits topological horseshoes.

The bifurcations between the different scenarios have been identified and explored. All the results are illustrated with numerical simulations.

¹ A wide range of epidemiological models address the impact of pulse vaccination. In the section “state of the art”, we include those that best relate to our work. Readers interested in other models with different particularities can explore the references contained within our reference list.

Structure

In this paper, we analyze a modified SIR model to study the impact of the pulse vaccination strategy with and without seasonality. Section 2 provides fundamental insights into impulsive differential equations and the important concepts of periodic solutions and stability. Section 3 introduces the model (with and without vaccination and with and without seasonality) and clarifies the role of the hypotheses. Also, in this section, we present our two main results. From Section 4 to Section 7, we analyze the model from a mathematical point of view: we find the fixed points of the associated stroboscopic maps, compute the *basic reproduction number*, and evaluate the local stability of the disease-free periodic solution. From Section 8 to Section 14, we prove all items of the main results. In Section 15, we provide some numerical simulations that support the theoretical results and, finally, in Section 16, we relate our findings with others in the literature.

2. Preliminaries

For the sake of self-containedness of the paper, we present the basic definitions and notation of the theory of impulsive dynamical systems we need. We also include some fundamental results which are necessary for understanding the theory. This information can be found in [31,32] and Chapter 1 of [33].

2.1. Instantaneous impulsive differential equations

An impulsive differential equation is given by an ordinary differential equation coupled with a discrete map defining the “jump” condition. The law of evolution of the process is described by the differential equation

$$\frac{dx}{dt} = f(t, x)$$

where $t \in \mathbb{R}$, $x \in \Lambda \subset \mathbb{R}^n$ and $f : \mathbb{R} \times \Lambda \rightarrow \Lambda$ is C^1 . The instantaneous impulse at time t is defined by the jump map $J(t, x) : \mathbb{R} \times \Lambda \rightarrow \Lambda$ given by

$$(t, x) \mapsto x + J(t, x).$$

Throughout this article, we focus on the *Instantaneous impulsive equation*:

$$\begin{aligned} \frac{dx}{dt} &= f(t, x), & t \neq T_k, \\ \Delta x(T_k) &= J_k(x), & k \in \mathbb{N}_0, \\ x(0) &= x_0, \end{aligned} \tag{1}$$

where $x \in \Lambda \subset \mathbb{R}$, the impulse is fixed at the sequence $(T_k)_{k \in \mathbb{N}_0}$ such that $T_0 = 0$ and

$$\forall k \in \mathbb{N} \quad T_k < T_{k+1} \quad \text{and} \quad \lim_{k \in \mathbb{N}} T_k = +\infty.$$

The instantaneous “jump” $\Delta x(T_k) = J(T_k, x) \equiv J_k(x)$ of (1) is defined by

$$\Delta x(T_k) := \lim_{t \rightarrow T_k^+} \varphi(t, x) - \lim_{t \rightarrow T_k^-} \varphi(t, x).$$

For $k \in \mathbb{N}$ and $t \in [T_k, T_{k+1})$, $\varphi(t, x)$ is a solution of $\frac{dx}{dt} = f(t, x)$; for $t = T_k$, φ satisfies

$$\lim_{t \rightarrow T_k^+} \varphi(t, x) = \lim_{t \rightarrow T_k^-} \varphi(t, x) + J_k \left(\lim_{t \rightarrow T_k^-} \varphi(t, x) \right),$$

where $x \in \Lambda$. The next result concerns the existence of a unique solution for (1).

Proposition 1 ([32,34], Adapted). *Let the function $f : \mathbb{R} \times \Lambda \rightarrow \mathbb{R}^n$ be continuous in the sets $[T_k, T_{k+1}[\times \Lambda$, where $k \in \mathbb{N}$. For each $k \in \mathbb{N}$ and $x \in \Lambda$, suppose there exists (and is finite) the limit of $f(t, y)$ as $(t, y) \rightarrow (T_k, x)$, where $t > T_k$. Then, for each $(t_0, x_0) \in \mathbb{R} \times \Lambda$ there exist $\beta > t_0$ and a solution $\varphi :]t_0, \beta[\rightarrow \mathbb{R}^n$ of the IVP (1). If f is C^1 with respect to x in $\mathbb{R} \times \Lambda$, then the solution is unique.*

The following result imposes conditions where the solution φ may be extendable.

Proposition 2 ([32,34], Adapted). *Let the function $f : \mathbb{R} \times \Lambda \rightarrow \mathbb{R}^n$ be C^1 in the sets $[T_k, T_{k+1}[\times \Lambda$, where $k \in \mathbb{N}$. For each $k \in \mathbb{N}$ and $x \in \Lambda$, suppose there exists (and is finite) the limit of $f(t, y)$ as $(t, y) \rightarrow (T_k, x)$, where $t > T_k$. If $\varphi :]\alpha, \beta[\rightarrow \mathbb{R}^n$ is a solution of (1), then the solution is extendable to the right of β if and only if*

$$\lim_{t \rightarrow \beta^-} \varphi(t, x) = \eta,$$

and one of the following conditions holds:

- (1) $\beta \neq T_k$, for any $k \in \mathbb{N}_0$ and $\eta \in \Lambda$;

(2) $\beta = T_k$, for some $k \in \mathbb{N}_0$ and $\eta + J(T_k, \eta) \in A$.

Under the conditions of Proposition 2, for each $(t_0, x_0) \in \mathbb{R} \times A$, there exists a unique solution $\varphi(t, x_0)$ of (1) defined in \mathbb{R} [32,34] which can be written as

$$\varphi(t, x_0) = \begin{cases} x_0 + \int_{t_0}^t f(s, \varphi(s, x_0))ds + \sum_{t_0 \leq T_k < t} J(\varphi(T_k, x_0)) & \text{for } t \geq t_0, \\ x_0 + \int_{t_0}^t f(s, \varphi(s, x_0))ds + \sum_{t \leq T_k < t_0} J(\varphi(T_k, x_0)) & \text{for } t < t_0. \end{cases}$$

Definition 1. We say that $\mathcal{K} \subset (\mathbb{R}_0^+)^2$ is a *positively flow-invariant set* for (1) if for all $x \in \mathcal{K}$, the trajectory of $\varphi(t, x)$ is contained in \mathcal{K} for $t \geq 0$.

For a solution of (1) passing through $x \in \mathbb{R}^n$, the set of its accumulation points, as t goes to $+\infty$, is the *ω -limit set* of x . More formally, if \bar{A} is the topological closure of $A \subset \mathbb{R}^n$, then:

Definition 2. If $x \in \mathbb{R}^n$, then the *ω -limit* of x is

$$\omega(x) = \bigcap_{T=0}^{+\infty} \overline{\left(\bigcup_{t>T} \varphi(t, x) \right)}.$$

It is well known that $\omega(x)$ is closed and *flow-invariant*, and if the trajectory of x is contained in a compact set, then $\omega(x)$ is non-empty. If E is an invariant set of (1), we say that E is a *global attractor* if $\omega(x) \subset E$, for Lebesgue almost all points x in \mathbb{R}^n .

2.2. Periodic solutions and stability

The following definitions have been adapted from [35–37]. For $T > 0$, we say that φ is a *T-periodic solution* of (1) if and only if there exists $x_0 \in A$ such that

$$\forall t \in \mathbb{R} \quad \varphi(t, x_0) = \varphi(t + T, x_0). \tag{2}$$

We disregard constant solutions and we consider the smallest positive value T for which (2) holds. Let $x_0 \in A$ be such that $\varphi(t, x_0)$ is a *T-periodic solution* of (1). We say that $\varphi(t, x_0)$ is:

- (1) *stable* if, for any neighborhood V of x_0 , there is a neighborhood $W \subset V$ of x_0 such that for all $y_0 \in W$ and for all $k \in \mathbb{N}$ we have $\varphi(kT, y_0) \in V$;
- (2) *asymptotically stable* if, it is stable and there exists a neighborhood V of x_0 such that for any $y_0 \in V$ and $\lim_{k \rightarrow +\infty} \varphi(kT, y_0) = x_0$;
- (3) *unstable* if it is not stable.

3. Model

Similarly to the classical SIR model [38], the population in the model under consideration is divided into three subpopulations:

- *Susceptibles*: individuals that are currently not infected but can contract the disease;
- *Infectious*: individuals who are currently infected and can actively transmit the disease to a susceptible individual until their recovery;
- *Recovered*: individuals who currently can neither be infected nor infect susceptible individuals. This comprises individuals with permanent immunity because they have recovered from a recent infection or have been vaccinated.

Let $S, I,$ and R denote the proportion of individuals within the compartment of *Susceptible, Infectious,* and *Recovered* individuals (within the whole population). Susceptible individuals are those who have never had contact with the disease. Once they have contact with the disease, they become *Infectious*. They are “transferred” to the *Recovered* class if they do not die. Those who recover from the disease get lifelong immunity. We add effective pulse vaccination to a proportion $p \in [0, 1]$ of the Susceptible Individuals, providing lifelong immunity.

We propose the following nonlinear impulsive differential equation in $S, I,$ and R (depending on time $t \in \mathbb{R}_0^+$):

$$\dot{X} = F_\gamma(X) \Leftrightarrow \begin{cases} \dot{S} = S(A - S) - \beta_\gamma(t)IS \\ \dot{I} = \beta_\gamma(t)IS - (\mu + d + g)I, & t \neq nT \\ \dot{R} = gI - \mu R \\ S(nT) = (1 - p)S(nT^-) \\ I(nT) = I(nT^-) \\ R(nT) = R(nT^-) + pS(nT^-) \end{cases} \tag{3}$$

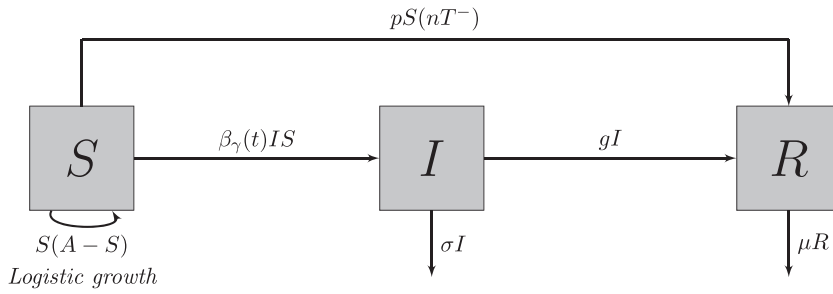


Fig. 1. Schematic diagram of model (3). Boxes represent the subpopulations S, I , and R , and arrows indicate the flow between the compartments.

where $n \in \mathbb{N}$, $p \in [0, 1]$,

$$X(t) = (S(t), I(t), R(t)),$$

$$X(t_0) := X_0 \text{ is the initial condition (in general } t_0 = 0),$$

$$\dot{X} = (\dot{S}, \dot{I}, \dot{R}) = \left(\frac{dS}{dt}, \frac{dI}{dt}, \frac{dR}{dt} \right),$$

$$\beta_\gamma(t) = \beta_0 (1 + \gamma \Psi(\omega t)),$$

$$X(nT^-) = \lim_{t \rightarrow nT^-} X(t),$$

$A \in (0, 1]$, $\gamma \geq 0$, $\omega > 0$ and Ψ is periodic. The number $S(nT^-) = \lim_{t \rightarrow nT^-} S(t)$ corresponds to the *Susceptible* individuals at the instant immediately before being vaccinated for the n th time. The model without vaccination corresponds to $p = 0$. Fig. 1 represents the dynamics of (3).

3.1. Description of the parameters

We can describe the parameters of (3) as:

A the carrying capacity of the *Susceptible* for $\beta_0 = 0$, i.e. in the absence of disease;

γ the amplitude of the seasonal variation that oscillates between $\beta_0 \left(1 + \gamma \min_{t \in [0, \tau]} \Psi(t)\right) > 0$ in the low season, and $\beta_0 \left(1 + \gamma \max_{t \in [0, \tau]} \Psi(t)\right)$ in the high season, for some $\tau > 0$;

$\Psi(\omega t)$ the effects of periodic seasonality over the time t with frequency $\omega > 0$;

β_0 the disease transmission rate in the absence of seasonality (when $\gamma = 0$). The parameter γ “measures the deformation” of the transmission rate;

μ the natural death rate of infected and recovered individuals;

d the death rate of infected individuals due to the disease;

p the proportion of the *Susceptible* individuals periodically vaccinated;

g the cure rate;

T the positive period at which a proportion p of the *Susceptible* individuals is vaccinated.

In order to simplify the notation, we denote $\mu + d$ by σ .

3.2. Hypotheses and motivation

Regarding (3), we assume:

(C1) All parameters are non-negative;

(C2) For all $t \in \mathbb{R}_0^+$, $S(t) \leq A$;

(C3) $S(t)$, $I(t)$, and $R(t)$ are proportions over the whole population. In particular, we have $S(t) + I(t) + R(t) = 1$, for all $t \in \mathbb{R}_0^+$;

(C4) For $\tau > 0$ and $\gamma > 0$, the map $\Psi : \mathbb{R} \rightarrow \mathbb{R}^+$ is τ -periodic, $\frac{1}{\tau} \int_0^\tau \beta_\gamma(t) dt > 0$ and has (at least) two nondegenerate critical points.²

From (C2) and (C3), since A is the carrying capacity of S , then $A \in (0, 1]$. The phase space of (3) is a subset of $(\mathbb{R}_0^+)^3$, endowed with the usual Euclidean distance (denoted by $dist$) and the set of parameters is described as follows (for $\varepsilon > 0$ small):

$$\Omega \subset \{(A, \beta_0, p, \sigma, g) \in (\mathbb{R}_0^+)^5\}, \quad T \in \mathbb{R}_0^+, \quad \gamma \in [0, \varepsilon] \quad \text{and} \quad \omega \in \mathbb{R}^+.$$

Remark. System (3) has been motivated by the classical SIR model [3] with the following modifications:

- We analyze the logistic growth of the *Susceptible* individuals as a result of crowding and natural competition for resources [19, 39,40] instead of assuming linear or exponential growth;
- The model includes a pulse-vaccination strategy to fight the disease where the *Susceptible* individuals are T -periodically vaccinated [12,13].

The first two equations of (3), \dot{S} and \dot{I} , are independent of R . Therefore we can reduce (3) to

$$\dot{x} = f_\gamma(x) \Leftrightarrow \begin{cases} \dot{S} = S(A - S) - \beta_\gamma(t)IS \\ \dot{I} = \beta_\gamma(t)IS - (\sigma + g)I, & t \neq nT \\ S(nT) = (1 - p)S(nT^-) \\ I(nT) = I(nT^-) \end{cases} \quad (4)$$

with $x = (S, I) \in (\mathbb{R}_0^+)^2$ and $n \in \mathbb{N}$.

3.3. No vaccination and no seasonality

If we do not consider neither vaccination nor seasonality in (4) ($\Leftrightarrow p = 0$ and $\gamma = 0$), then (4) can be rewritten as

$$\begin{cases} \dot{S} = S(A - S) - \beta_0 IS \\ \dot{I} = \beta_0 IS - (\sigma + g)I \end{cases} \quad (5)$$

and the *basic reproduction number* \mathcal{R}_0 can be explicitly computed as:

$$\mathcal{R}_0 = \lim_{\tau \rightarrow +\infty} \frac{1}{\tau} \int_0^\tau \frac{A\beta_\gamma(t)}{\sigma + g} dt \stackrel{\gamma=0}{=} \frac{A\beta_0}{\sigma + g} > 0. \quad (6)$$

The *basic reproduction number* \mathcal{R}_0 is an epidemiological measure that indicates the average number of new infections caused by a single infected individual in a completely susceptible population [10,41].

If A , β_0 , σ , and g are such that $\mathcal{R}_0 < 1$, the dynamics of (5) is quite simple: all solutions with initial condition $S_0 > 0$ converge to $(S, I) = (A, 0)$, as proved in [10]. Otherwise, if $\mathcal{R}_0 > 1$, then all solutions with initial condition $S_0, I_0 > 0$ converge to the endemic equilibrium

$$(S, I) = \left(\frac{\sigma + g}{\beta_0}, \frac{A\beta_0 - (\sigma + g)}{\beta_0^2} \right) = \left(\frac{A}{\mathcal{R}_0}, \frac{A}{\beta_0} \left(1 - \frac{1}{\mathcal{R}_0} \right) \right). \quad (7)$$

From now on, we analyze the model with pulse vaccination and no seasonality, i.e. $p \neq 0$ and $\gamma = 0$.

3.4. Pulse vaccination and no seasonality

In the absence of seasonality ($\gamma = 0$), the model (4) can be recast into the form

$$\dot{x} = f_0(x) \Leftrightarrow \begin{cases} \dot{S} = S(A - S) - \beta_0 IS \\ \dot{I} = \beta_0 IS - (\sigma + g)I, & t \neq nT \\ S(nT) = (1 - p)S(nT^-) \\ I(nT) = I(nT^-) \end{cases} \quad (8)$$

whose flow is given by

$$\varphi_0(t, (S_0, I_0)) = [S(t, (S_0, I_0)), I(t, (S_0, I_0))], \quad t \in \mathbb{R}_0^+, \quad (S_0, I_0) \in (\mathbb{R}_0^+)^2. \quad (9)$$

² This corresponds to a generic periodically-forced perturbation.

Table 1
Structure of the proof of [Theorem A](#) and the location of the items throughout the present article.

Items of Theorem A	Reference /Section
(1)	Trivial
(2)	Section 8
(3)	Section 7 and 9
(4)	Section 10
(5)	See Ref. [10]
(6)	Section 11
(7)	Section 6

We denote by S_c the epidemic critical threshold associated to [\(8\)](#):

$$S_c = \frac{\sigma + g}{\beta_0} > 0. \tag{10}$$

The meaning of S_c will be explained immediately after [Lemma 2](#). Before stating the main results, we provide two definitions adapted from [\[42\]](#):

Definition 3. System [\(8\)](#) is said to be:

- (1) *uniformly persistent* if there are constants $c_1, c_2, T_0 > 0$ such that for all solutions $(S(t), I(t))$ with initial conditions $S_0 > 0$ and $I_0 > 0$, we have

$$c_1 \leq S(t) \quad \text{and} \quad c_2 \leq I(t),$$

for all $t \geq T_0$;

- (2) *permanent* if it is uniformly persistent and bounded, that is, there are constants $c_1, c_2, C_1, C_2, T_0 > 0$ such that for all solutions $(S(t), I(t))$ with initial conditions $S_0 > 0$ and $I_0 > 0$, we have

$$c_1 \leq S(t) \leq C_1 \quad \text{and} \quad c_2 \leq I(t) \leq C_2,$$

for all $t \geq T_0$.

The main result of this article provides a complete description of the dynamics of [\(8\)](#) through a bifurcation diagram. We also exhibit an explicit expression for the basic reproduction number R_p for the system with impulsive vaccination.

Theorem A. For $\gamma = 0$ and $A > S_c$, in the bifurcation diagram $(T, p) \in (\mathbb{R}_0^+)^2$ associated to [\(8\)](#), we may define the maps $p_1, p_2 : \mathbb{R}_0^+ \rightarrow [0, 1]$ given by

$$p_1(T) = 1 - e^{-AT} \quad \text{and} \quad p_2(T) = 1 - e^{-(A-S_c)T}$$

such that:

- (1) if $p = 1$, then the ω -limit of all solutions of [\(8\)](#) is the disease-free periodic solution associated to $(S, I) = (0, 0)$;
- (2) if $p \in (p_1(T), 1)$, then the ω -limit of all solutions of [\(8\)](#) is the disease-free periodic solution associated to $(S, I) = (0, 0)$;
- (3) if $p \in (p_2(T), p_1(T))$, then the ω -limit of all solutions of [\(8\)](#) with initial condition $S_0 > 0$ is a disease-free (non-trivial) periodic solution $(S, 0)^3$;
- (4) if $p \in (0, p_2(T))$, then system [\(8\)](#) is permanent and the ω -limit of all solutions of [\(8\)](#) with initial condition $S_0, I_0 > 0$ is an endemic periodic solution (S, I) ;
- (5) if $p = 0$, then the ω -limit of all solutions of [\(8\)](#) with initial condition $S_0, I_0 > 0$ is the endemic equilibrium defined in [\(7\)](#);
- (6) The curves p_1 and p_2 correspond to saddle-node and transcritical bifurcations, respectively;
- (7) The basic reproduction number associated to [\(8\)](#) is $R_p = R_0 \left[\frac{\ln(1-p)}{AT} + 1 \right]$ and $R_p > 1$ if and only if $p < p_2$.

The scenarios [①](#)–[⑤](#) of [Theorem A](#) are represented in the bifurcation diagram (T, p) of [Figs. 2](#) and [3](#). Its proof is performed in several sections throughout the present article and its location is indicated in [Table 1](#).

3.5. Pulse vaccination and seasonality

Seasonal variations may be captured by introducing periodically-perturbed terms into a deterministic differential equation [\[14, 16\]](#). The periodically-perturbed term $\Psi(t)$ in $\beta_\gamma(t)$ may be seen as a natural periodic map over time with two global extrema

³ In [Section 9](#), we provide an explicit expression for the periodic solution $(S, 0)$; all trajectories converge to $(S, 0)$ in the topology of pointwise convergence.

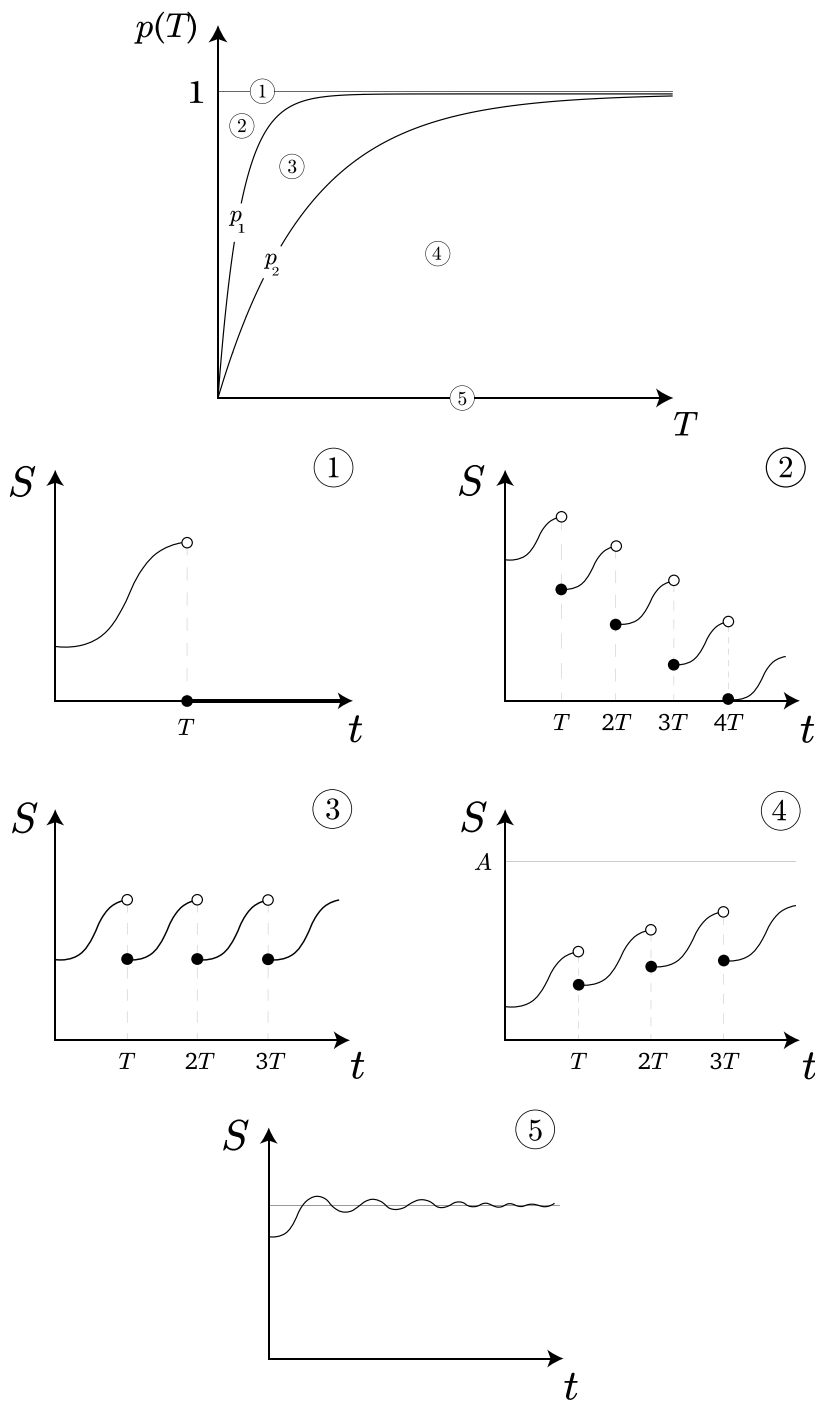


Fig. 2. Bifurcation diagram (T, p) associated to (8). In ①, all solutions converge to $(0, 0)$; in ②, all solutions converge to $(0, 0)$; in ③, all solutions with initial condition $S_0 > 0$ tend towards the disease-free (non-trivial) periodic solution $(S, 0)$; in ④, all solutions with initial condition $S_0, I_0 > 0$ tend towards the endemic periodic solution (S, T) ; in ⑤, all solutions with initial condition $S_0, I_0 > 0$ tend towards the endemic equilibrium (S, T) . Compare with the numerical simulation of these five scenarios in Fig. 9.

(governing the high and lower seasons defined by weather conditions). Corollary 1, proved in Section 13, confirms the numerical results suggested by Choisy et al. [43]: neglecting the effect of the amplitude of the seasonal transmission can lead to somewhat overoptimistic values of the optimal pulse period.

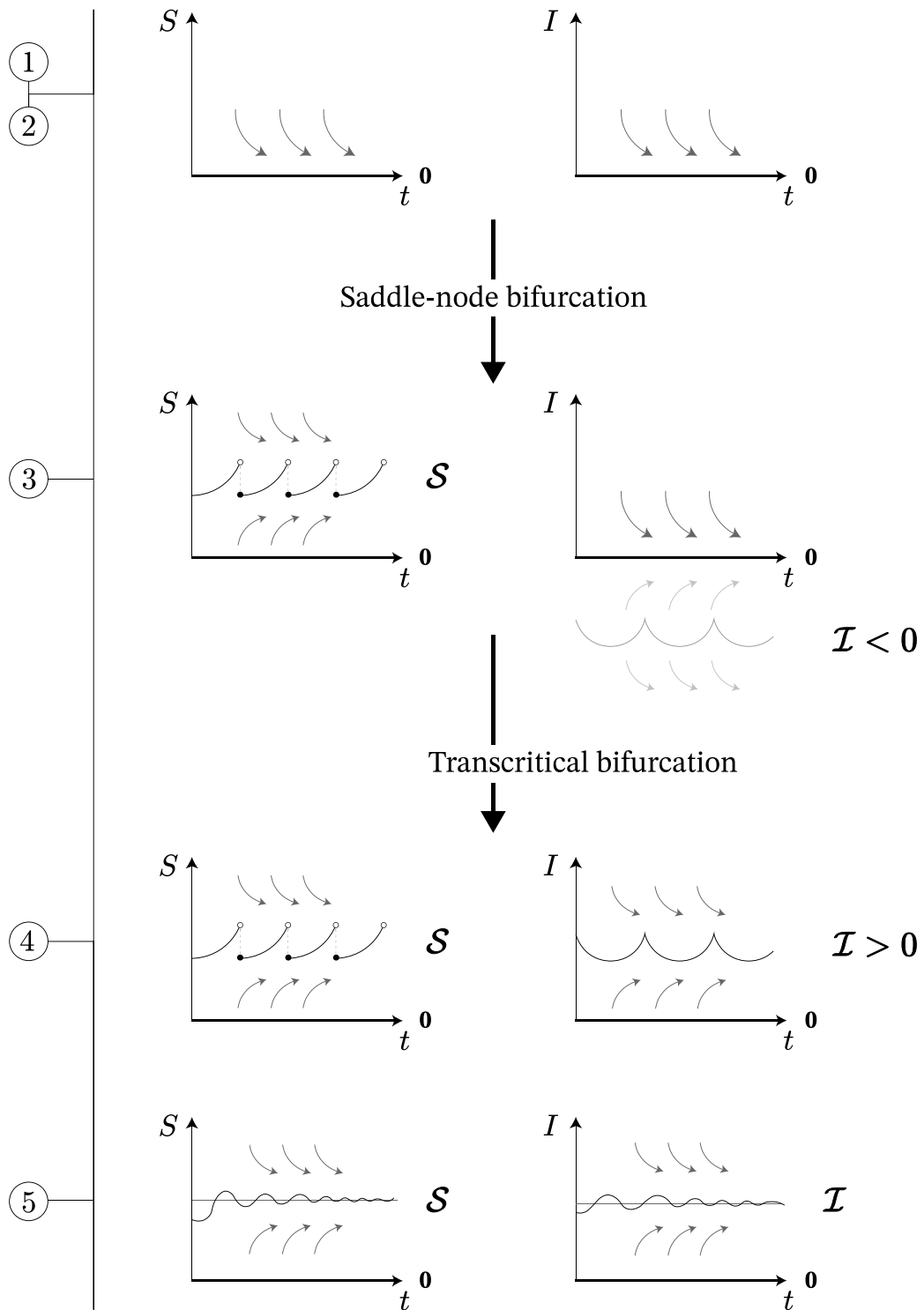


Fig. 3. Bifurcations associated to the diagram (T, p) associated to (8). From ② to ③: saddle-node associated to the solution $(0, 0)$; From ③ to ④: transcritical bifurcation associated to $(S, 0)$. For the numbering ① to ⑤, see Theorem A and Fig. 2.

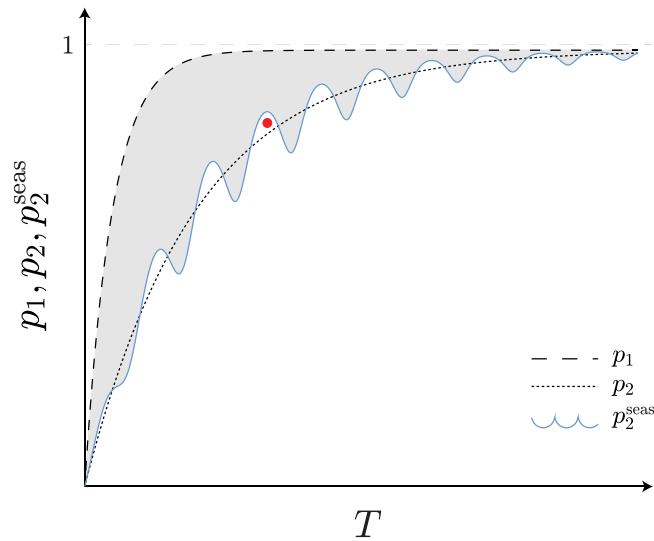


Fig. 4. Illustration of Corollary 1: the T -periodic solution $(S, 0)$ is asymptotically stable if and only if $p_2^{\text{seas}}(T) < p < p_1(T)$. The red dot is a point where $(S, 0)$ is stable (without seasonality) and unstable (with seasonality).

Corollary 1. For $\gamma, \omega > 0$ small and $A > S_c$, the T -periodic solution $(S, 0)$ is asymptotically stable if and only if $p_2^{\text{seas}}(T) < p < p_1(T)$, where

$$p_1(T) = 1 - e^{-AT} \quad \text{and} \quad p_2^{\text{seas}}(T) = 1 - e^{-\left[(A - S_c)T + \gamma \int_0^T \Psi(\omega t) S(t) dt \right]}$$

One aspect that contributes to the complexity of (4) is the existence of chaos. Let $U \subset \Omega$ such that $0 < p < p_2(T) (\Leftrightarrow \mathcal{R}_p > 1)$. Under the condition that $T = k\tau/\omega, k \in \mathbb{N}$, there is an endemic T -periodic solution for (8) (see Item (4) of Theorem A). In what follows, we assume that M is diffeomorphic to a circloid.

Definition 4. An embedding $F : M \rightarrow M$ is said to have a *horseshoe* if for some $N, n \in \mathbb{N}$, the map F^N has a uniformly hyperbolic invariant set $Y \subset M$ such that $F^N|_Y$ is topologically conjugate to the full shift on n symbols (Σ_n, σ) where σ is the usual shift operator.

A vector field possesses a suspended horseshoe if the first return map to a cross-section has a horseshoe. The existence of a horseshoe for the embedding F is equivalent to the notion of topological chaos ($\Leftrightarrow F$ has positive topological entropy).

Theorem B. For $0 < p < p_2^{\text{seas}}(T), T = k\tau/\omega, k \in \mathbb{N}$ and $\omega \in \mathbb{R}^+$, if γ is sufficiently large, then the flow of f_γ has a suspended topological horseshoe.

The shift dynamics obtained for (4) differ from that of [44], as discussed in Section 16. The proof of Theorem B is performed in Section 14.

3.6. Biological consequences

As suggested by Theorem A, the global eradication of an epidemic by means of pulse vaccination is always possible, provided the vaccination coverage is large enough.

Based on experimental data, the World Health Organization recommends that the time between successive pulses should be as short as possible. For a specific vaccination coverage $p \in (0, 1)$, there exists a pulse interval $(0, T_2)$ where $T_2 = \frac{|\ln(1-p)|}{A - S_c}$ that ensures the effective implementation of this campaign ($\Leftrightarrow \mathcal{R}_p < 1$); that is, the T -periodic administration of doses with $T \in (0, T_2)$ leads to the global eradication of the disease, and T_2 is the optimal time that determines the fastest eradication. For a specific vaccination coverage $p > 0$, if $T > T_2$ then $\mathcal{R}_p > 1$.

Adding seasonality to our model, the epidemic critical threshold S_c also depends on β_γ . Corollary 1 stresses that neglecting the effect of the amplitude of the seasonal transmission can lead to overoptimistic values of the optimal pulse period T_2 . It also stresses that the best moment for vaccination corresponds to the local minimizers of β_γ . Theorem B says that the number of Susceptible individuals and Infectious in the presence of seasonal variations could be unpredictable. The distant future is practically inaccessible and may only be described in average, in probabilistic and ergodic terms.

4. Preparatory section

In this section, we collect lemmas needed for the proof of [Theorems A, B](#) and [Corollary 1](#). We start by proving the existence of a compact set where the dynamics lies.

Lemma 1. *The region defined by*

$$\mathcal{M} = \left\{ (S, I) \in (\mathbb{R}_0^+)^2 : 0 \leq S \leq A, \quad 0 \leq S + I \leq \frac{A(\sigma + g + A)}{\sigma + g}, \quad S, I \geq 0 \right\}$$

is positively flow-invariant for [\(8\)](#).

Proof. We can easily to check that $(\mathbb{R}_0^+)^2$ is flow-invariant. Now, we show that if $(S_0, I_0) \in \mathcal{M}$, then $\varphi_0(t, (S_0, I_0))$, $t \in \mathbb{R}_0^+$, is contained in \mathcal{M} . Let us define

$$v(t) = S(t) + I(t) \geq 0$$

associated to the trajectory $\varphi_0(t, (S_0, I_0))$. Omitting the variables' dependence on t , one knows that

$$\begin{aligned} \dot{v} &= \dot{S} + \dot{I} \\ &= S(A - S) - \beta_0 IS + \beta_0 IS - (\sigma + g)I \\ &= S(A - S) - (\sigma + g)I, \end{aligned}$$

from which we deduce that

$$\begin{aligned} \dot{v} + (\sigma + g)v &= S(A - S) - (\sigma + g)I + (\sigma + g)S + (\sigma + g)I \\ &= S(A - S) + (\sigma + g)S \\ &\leq (\sigma + g + A)S. \end{aligned}$$

If $\beta_0 = p = 0$, the first component of Eq. [\(8\)](#) would represent logistic growth. Thus, its solution would have an upper bound of A (by [\(C2\)](#)). This property is also verified for $\beta_0, p \in \mathbb{R}^+$, even if p is applied periodically (note that $p \in [0, 1]$). In particular, we have

$$\dot{v} + (\sigma + g)v \leq (\sigma + g + A)A.$$

The classical differential version of the Gronwall's inequality⁴ says that for all $t \in \mathbb{R}_0^+$, we have

$$v(t) \leq v_0 e^{-(\sigma+g)t} - \frac{(\sigma + g + A)A}{(\sigma + g)} (e^{-(\sigma+g)t} - 1),$$

where $v(0) := v_0 = S(0) + I(0) \geq 0$. Taking the limit when $t \rightarrow +\infty$, we get:

$$\begin{aligned} 0 \leq \lim_{t \rightarrow +\infty} v(t) &\leq \lim_{t \rightarrow +\infty} \left[v_0 e^{-(\sigma+g)t} - \frac{(\sigma + g + A)A}{(\sigma + g)} (e^{-(\sigma+g)t} - 1) \right] \\ &= \frac{(\sigma + g + A)A}{(\sigma + g)}. \end{aligned}$$

Since $\lim_{t \rightarrow +\infty} v(t) = \lim_{t \rightarrow +\infty} (S(t) + I(t))$, the result is proved. \square

Lemma 2. *Let D be an open subinterval of \mathbb{R}_0^+ . With respect to [\(8\)](#), the following assertions hold:*

- (1) $S(t) < S_c$ for all $t \in D$ if and only if I is decreasing in D ;
- (2) $S(t) = S_c$ for all $t \in D$ if and only if I is constant;
- (3) $S(t) > S_c$ for all $t \in D$ if and only if I is increasing in D .

Proof. We just show (1); the proof of (2) and (3) follows from the same reasoning. We know that the *Infectious* is decreasing if and only if $\dot{I}(t) < 0$, for $t \in D$. Indeed,

$$\begin{aligned} \dot{I}(t) < 0 &\stackrel{(8)}{\Leftrightarrow} \beta_0 S(t)I(t) - (\sigma + g)I(t) < 0 \\ &\Leftrightarrow S(t) < \frac{\sigma + g}{\beta_0} = S_c, \end{aligned} \tag{11}$$

and the result is proved. \square

Remark. The constant S_c defined in [\(10\)](#) will be called the *epidemic critical value* and is the threshold on the number of *Susceptible* individuals that defines whether the epidemic spreads or not. When $\gamma = 0$, the most intriguing dynamical scenario is when $S(t) - S_c$ takes different signs in $D \subset \mathbb{R}_0^+$ (See [Fig. 5](#)).

⁴ If $a, b \in \mathbb{R}$ and $u : \mathbb{R}_0^+ \rightarrow \mathbb{R}_0^+$ is a C^1 map such that $u' \leq au + b$, then $u(t) \leq u(0)e^{at} + \frac{b}{a}(e^{at} - 1)$.

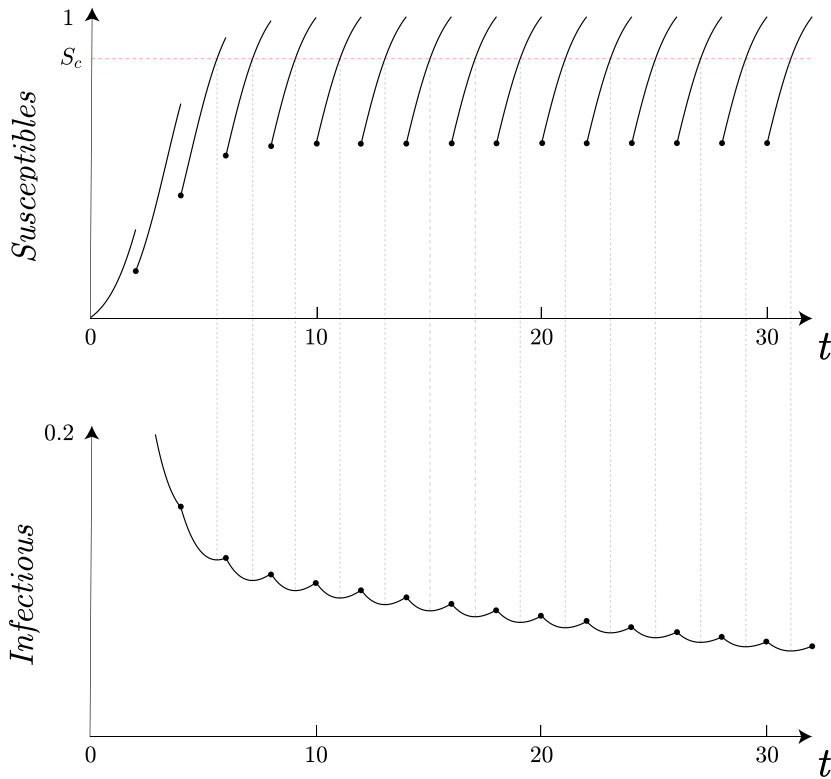


Fig. 5. Illustration of Lemma 2 for $D = \mathbb{R}^+$: evolution of S and I as $t \in \mathbb{R}^+$ evolves.

Lemma 3. *The following equivalence holds for system (8):*

$$A > S_c \Leftrightarrow \mathcal{R}_0 > 1.$$

Proof. The proof follows from:

$$A > S_c \stackrel{(10)}{\Leftrightarrow} A > \frac{\sigma + g}{\beta_0} \stackrel{(6)}{\Leftrightarrow} \mathcal{R}_0 > 1. \quad \square$$

5. Stroboscopic maps and their fixed points

In this section, we study the existence of fixed points associated with the stroboscopic maps (time T maps) of the *Susceptible* and *Infectious* individuals for system (8). We also analyze their stability in the sense of Section 2.2.

For $n \in \mathbb{N}$ and $T > 0$, using (9), define the sequences:

$$S_n = S(nT, (S_0, I_0)) \quad \text{and} \quad I_n = I(nT, (S_0, I_0)),$$

which can be seen as the stroboscopic T -maps associated to the trajectory of $(S_0, I_0) \in (\mathbb{R}_0^+)^2$.

Lemma 4.

(1) *There exists $F_I : \mathbb{R}_0^+ \rightarrow \mathbb{R}_0^+$ such that $I_{n+1} = F_I(I_n)$ and*

$$F_I(y) = y \exp \left\{ \beta_0 \int_{nT}^{(n+1)T} S(t) dt \right\} e^{-(\sigma+g)T}.$$

(2) *The fixed point of F_I is:*

(1) $y^* = 0$ if $\frac{1}{T} \int_0^T S(t) dt \neq S_c$;

(2) (any) $y^* \in \mathbb{R}_0^+$ if $\frac{1}{T} \int_0^T S(t) dt = S_c$.

Proof.

(1) From (8), for $I(t) > 0$ for all $t \in \mathbb{R}_0^+$, we have

$$\begin{aligned} \dot{I}(t) &= \beta_0 S(t)I(t) - (\sigma + g)I(t) \\ \Leftrightarrow \frac{dI(t)}{I(t)} &= [\beta_0 S(t) - (m + g)] dt \\ \Rightarrow \int_{t_0}^t \frac{dI(\tau)}{I(\tau)} &= \beta_0 \int_{t_0}^t S(\tau) d\tau - (m + g) \int_{t_0}^t d\tau \\ \Leftrightarrow \ln I(t) - \ln I(t_0) &= \beta_0 \int_{t_0}^t S(\tau) d\tau - (m + g)(t - t_0) \\ \Leftrightarrow I(t) &= I_0 \exp \left\{ \beta_0 \int_{t_0}^t S(\tau) d\tau \right\} e^{-(\sigma+g)(t-t_0)}, \end{aligned}$$

where $I(t_0) := I_0 > 0$. The sequence $I_{n+1} = I((n + 1)T, (S_0, I_0))$ is then computed as

$$\begin{aligned} I_{n+1} &:= I_n \exp \left\{ \beta_0 \int_{nT}^{(n+1)T} S(t) dt \right\} e^{-(\sigma+g)T} \\ \Leftrightarrow I_{n+1} &= F_I(I_n), \end{aligned}$$

where $F_I(y) = y \exp \left\{ \beta_0 \int_{nT}^{(n+1)T} S(t) dt \right\} e^{-(\sigma+g)T}$.

(2) The fixed points of F_I can be found by solving the equation $F_I(y^*) = y^*$ which is equivalent to

$$\begin{aligned} y^* \exp \left\{ \beta_0 \int_{nT}^{(n+1)T} S(t) dt \right\} e^{-(\sigma+g)T} &= y^* \\ \Leftrightarrow y^* \exp \left(\frac{1}{T} \int_0^T S(t) dt - S_c \right) &= y^*. \end{aligned}$$

In other words, if $\frac{1}{T} \int_0^T S(t) dt \neq S_c$, then the fixed point of F_I is given by $y^* = 0$. Otherwise any $y^* \in \mathbb{R}_0^+$ is a solution of $F_I(y^*) = y^*$. \square

If $\frac{1}{T} \int_0^T S(t) dt \neq S_c$, then the unique fixed point of F_I is $y^* = 0$. From now on, we focus the analysis on this fixed point. We remind the definition of p_1 from the statement of **Theorem A**: $p_1 \equiv p_1(T) = 1 - e^{-AT}$, $T > 0$.

Lemma 5. *If $I = 0$, then:*

(1) *There exists $F_S : \mathbb{R}_0^+ \rightarrow \mathbb{R}_0^+$ such that $S_{n+1} = F_S(S_n)$ and*

$$F_S(x) = \frac{Ax(1-p)e^{AT}}{x(e^{AT}-1)+A}.$$

(2) *The map $F_S(x)$ has two fixed points:*

$$x_1^* = A \left(1 - \frac{pe^{AT}}{e^{AT}-1} \right) \quad \text{and} \quad x_2^* = 0.$$

For $T > 0$, if $p > p_1(T)$, then $x_1^ < 0$.*

Proof.

(1) We study system (8) assuming $I = 0$, the unique fixed point of F_I if $\frac{1}{T} \int_0^T S(t) dt \neq S_c$ (cf. **Lemma 4**). The growth of S for $t_0 = nT \leq t < (n + 1)T$ is given by

$$\begin{cases} \dot{S}(t) = S(t)(A - S(t)), & t \neq nT, \\ S(nT) = (1 - p)S(nT^-). \end{cases} \tag{12}$$

Integrating (12) between pulses, and assuming that $S(t)(A - S(t)) \neq 0$, we obtain

$$\begin{aligned} \dot{S}(t) &= S(t)(A - S(t)) \\ \Leftrightarrow \int_{t_0}^t \frac{dS(\tau)}{S(\tau)(A - S(\tau))} &= \int_{t_0}^t d\tau \\ \Leftrightarrow \int_{t_0}^t \frac{dS(\tau)}{S(\tau)(A - S(\tau))} &= t - t_0 \end{aligned} \tag{13}$$

and thus we get:

$$S(t) = \frac{A S_0}{S_0 + (A - S_0) e^{-A(t-t_0)}}, \quad t \geq t_0 \geq 0, \tag{14}$$

where $S(t_0) := S_0$ and $A > S_0$. Notice that (14) holds between pulses. Bearing in mind that

$$S(nT^-) = \lim_{t \rightarrow nT^-} S(t), \quad S(nT) = (1 - p)S(nT^-), \quad n \in \mathbb{N}, \tag{15}$$

using induction over $n \in \mathbb{N}$ it is easy to show that the general expression of $S(t)$ for $nT \leq t < (n + 1)T$ is

$$S(t) = \frac{A(1 - p)S(nT^-)}{(1 - p)S(nT^-) + [A - (1 - p)S(nT^-)]e^{-A(t-nT)}}.$$

Using (15) and considering $S_n = S(nT, (S_0, I_0))$, we get

$$S(t) = \frac{A S_n}{S_n + (A - S_n) e^{-A(t-nT)}}, \tag{16}$$

where $nT \leq t < (n + 1)T$. In particular,

$$\begin{aligned} S((n + 1)T^-) &= \frac{A(1 - p)S(nT^-)}{(1 - p)S(nT^-) + [A - (1 - p)S(nT^-)]e^{-AT}} \\ \Leftrightarrow \frac{S((n + 1)T)}{1 - p} &= \frac{A(1 - p)S(nT^-)}{(1 - p)S(nT^-) + [A - (1 - p)S(nT^-)]e^{-AT}} \\ \Leftrightarrow S_{n+1} &= \frac{A S_n(1 - p)}{S_n + (A - S_n) e^{-AT}} \\ \Leftrightarrow S_{n+1} &= \frac{A S_n(1 - p)}{S_n(1 - e^{-AT}) + A e^{-AT}}. \end{aligned} \tag{17}$$

Multiplying both the numerator and the denominator of (17) by $e^{AT} > 0$, we get

$$S_{n+1} := \frac{A S_n(1 - p) e^{AT}}{S_n(e^{AT} - 1) + A} \quad \Leftrightarrow \quad S_{n+1} = F_S(S_n),$$

where

$$F_S(x) = \frac{A x(1 - p) e^{AT}}{x(e^{AT} - 1) + A}.$$

(2) The map F_S has two fixed points:

$$\begin{aligned} x_1^* &= A \left(1 - \frac{pe^{AT}}{e^{AT} - 1} \right) \\ x_2^* &= 0, \end{aligned}$$

where $x_1^* > 0$ if and only if

$$A \left(1 - \frac{pe^{AT}}{e^{AT} - 1} \right) > 0 \quad \Leftrightarrow \quad p < 1 - e^{-AT} \quad \Leftrightarrow \quad p < p_1(T). \quad \square$$

Combining Lemmas 4 and 5, we know that if $\frac{1}{T} \int_0^T S(t) dt \neq S_c$, then (F_S, F_I) has two non-negative fixed points: $(0, 0)$ and $(x_1^*, 0)$. In the flow of (8), they are denoted by $(0, 0)$ (stationary trivial equilibrium) and $(S, 0)$ (periodic non-trivial disease-free solution).

Lemma 6. For $T > 0$, the non-trivial T -periodic solution of (8) associated to $(x_1^*, 0)$ does not depend on the transmission rate β and is parametrized by

$$(S(t), 0) = \left(\frac{A [e^{AT} (1 - p) - 1]}{e^{AT} (1 - p) - 1 + pe^{A(T-(t-t_0))}}, 0 \right),$$

where $t_0 = nT \leq t < (n + 1)T$ and $n \in \mathbb{N}$.

Proof. Replacing S_0 by x_1^* in (14), we obtain

$$S(t) = \frac{A x_1^*}{x_1^* + (A - x_1^*) e^{-A(t-t_0)}} = \frac{A [e^{AT} (1 - p) - 1]}{e^{AT} (1 - p) - 1 + pe^{A(T-(t-t_0))}},$$

where $t_0 = nT \leq t < (n + 1)T$ and $n \in \mathbb{N}$. By construction, this solution is T -periodic (a consequence of Lemmas 4 and 5). \square

6. Basic reproduction number \mathcal{R}_p for (8)

For $T > 0$, following [15, Eq. (3.15)], we compute the *basic reproduction number* \mathcal{R}_p in the absence of seasonality for (8) as

$$\mathcal{R}_p(T) := \frac{\beta_0}{\sigma + g} \frac{1}{T} \int_0^T S(t) dt = \frac{1}{S_c T} \int_0^T S(t) dt, \tag{18}$$

based on the disease-free periodic solution $S(t)$ given explicitly in Lemma 6. The quantity \mathcal{R}_p (when less than 1) can be used to measure the velocity at which the disease is eradicated. For clarity, we omit the dependence of \mathcal{R}_p on $T > 0$. In the sequel, we present a series of properties of \mathcal{R}_p .

Lemma 7. *With respect to (8), the following assertions hold for $p \in [0, 1)$ and $T > 0$:*

- (1) $\mathcal{R}_p = \mathcal{R}_0 \left[\frac{\ln(1-p)}{AT} + 1 \right]$;
- (2) if $\mathcal{R}_p = 1$, then F_I has infinitely many fixed points;
- (3) for $T > 0$ and $p > 0$, we have $\frac{\partial \mathcal{R}_p}{\partial T}(T) > 0$ and $\frac{\partial \mathcal{R}_p}{\partial p}(T) < 0$;
- (4) $\lim_{T \rightarrow +\infty} \mathcal{R}_p = \mathcal{R}_0$ ⁵;
- (5) If $p = 0$, then $\mathcal{R}_p = \mathcal{R}_0$.

Proof.

(1) For the sake of simplicity, let us assume $t_0 = 0$. Then we have

$$\int_0^T S(t) dt \stackrel{6}{=} \int_0^T \frac{A [e^{AT} (1-p) - 1]}{e^{AT} (1-p) - 1 + pe^{A(T-t)}} dt = \ln(1-p) + AT.$$

Therefore,

$$\begin{aligned} \mathcal{R}_p &= \frac{\beta_0}{\sigma + g} \frac{1}{T} \int_0^T S(t) dt \\ &= \frac{\beta_0}{\sigma + g} \frac{1}{T} [\ln(1-p) + AT] \\ &\stackrel{(11)}{=} \frac{1}{S_c T} [\ln(1-p) + AT] \\ &\stackrel{(6),(11)}{=} \mathcal{R}_0 \left[\frac{\ln(1-p)}{AT} + 1 \right]. \end{aligned} \tag{19}$$

(2) Since

$$\begin{aligned} \mathcal{R}_p = 1 &\Leftrightarrow \frac{1}{T} \int_0^T S(t) dt = S_c \\ &\Leftrightarrow \exp \left\{ \beta_0 \int_{nT}^{(n+1)T} S(t) dt \right\} e^{-(\sigma+g)T} = 1, \end{aligned}$$

the result follows by observing the analytic expression of F_I in Lemma 4.

(3) From the proof of item (1), one knows that $\mathcal{R}_p = \mathcal{R}_0 \left[\frac{\ln(1-p)}{AT} + 1 \right]$. If $p > 0$, then

$$\frac{\partial \mathcal{R}_p}{\partial T}(T) = -\frac{\mathcal{R}_0 \ln(1-p)}{AT^2} > 0 \quad \text{and} \quad \frac{\partial \mathcal{R}_p}{\partial p}(T) = -\frac{\mathcal{R}_0}{AT(1-p)} < 0.$$

Items (4) and (5) are straightforward using the formula of \mathcal{R}_p of item (1). \square

The following useful result relates the stability of the impulsive periodic solution of Lemma 6 with the *basic reproduction number* \mathcal{R}_p for (8).

Lemma 8. *With respect to system (8), if $A > S_c$ ⁶ then $\mathcal{R}_p < 1 \Leftrightarrow p > p_2$.*

Proof. The proof follows from:

$$\mathcal{R}_p < 1 \Leftrightarrow \frac{1}{S_c T} [\ln(1-p) + AT] < 1 \Leftrightarrow p > p_2. \quad \square$$

⁵ This limit on T may be interpreted as the absence of vaccination.

⁶ $A > S_c \Leftrightarrow \mathcal{R}_0 > 1$, by Lemma 3.

7. Local stability of the disease-free solutions

The next result proves the local asymptotic stability of the disease-free periodic solutions of (8), (0, 0) and (S, 0), using Floquet multipliers.

Proposition 3. *With respect to (8), the following assertions hold for $T > 0$:*

- (1) *the disease-free trivial solution (0, 0) is locally asymptotically stable provided $p > p_1(T)$;*
- (2) *the disease-free periodic solution (S, 0) is locally asymptotically stable provided $p_2(T) < p < p_1(T)$.*

Proof. The stability of the disease-free periodic solution is found by analyzing the behavior of initial conditions close to it. This is achievable by computing the monodromy matrix (see [34, Chapter II, pp. 28]). If the absolute value of the eigenvalues (Floquet multipliers) of the monodromy matrix is less than one, then the periodic solution (S, 0) is asymptotically stable [34, Theorem 3.5, pp. 30].

We exhibit the computations near the T -periodic solution (S, 0). For $t \geq 0$, we set

$$\begin{aligned} S(t) &= S(t) + s(t) \\ I(t) &= I(t) + i(t) \quad \Leftrightarrow \quad I(t) = i(t), \end{aligned}$$

where $s(t)$ and $i(t)$ are small terms close to 0. We will show that they vanish when t increases. Omitting the dependence of the variables on t , from (8) and (20), one gets:

$$\begin{aligned} \frac{ds}{dt} &= \frac{dS}{dt} - \frac{dS}{dt} \\ &= S(A - S) - \beta_0 SI - [S(A - S) - \beta_0 SI] \\ &\stackrel{I=0}{=} SA - S^2 - \beta_0 SI - SA + S^2 \\ &= -SA + S^2 + A(S + s) - (S + s)^2 - \beta_0(S + s)i \\ &= -SA + S^2 + SA + sA - S^2 - 2sS - s^2 - \beta_0 Si - \beta_0 si \\ &= As - 2sS - \beta_0 Si - \beta_0 si - s^2 \\ &= (A - 2S - \beta_0 i)s - \beta_0 Si - s^2 \end{aligned}$$

and

$$\begin{aligned} \frac{di}{dt} &= \frac{dI}{dt} - \frac{dI}{dt} \\ &= \beta_0 Si - (\sigma + g)i \\ &= [\beta_0 S - (\sigma + g)]i. \end{aligned}$$

Hence, we model the evolution of (s, i) as

$$\left\{ \begin{aligned} \frac{ds}{dt} &= (A - 2S - \beta_0 i)s - \beta_0 Si \\ \frac{di}{dt} &= [\beta_0 S - (\sigma + g)]i, \\ s(nT) &= (1 - p)s(nT^-) \\ i(nT) &= i(nT^-). \end{aligned} \right. \quad t \neq nT, \tag{20}$$

Note that $S(t)$ is known explicitly (Lemma 6) and may be written as the periodic coefficient of $s(t)$ and $i(t)$. Lyapunov’s theory neglects quadratic terms to compute the stability of hyperbolic fixed points [45, §16, p. 6]. The solutions of (20) may be written as

$$\begin{pmatrix} s(t) \\ i(t) \end{pmatrix} = \Phi(t) \begin{pmatrix} s(0) \\ i(0) \end{pmatrix} \quad \text{where} \quad \Phi(t) = \begin{pmatrix} \varphi_{1,1}(t) & \varphi_{1,2}(t) \\ \varphi_{2,1}(t) & \varphi_{2,2}(t) \end{pmatrix}$$

is the fundamental matrix whose columns are the components of linearly independent solutions of (20), and $\Phi(0)$ is the identity matrix. According to Floquet theory [45, §VII.6.2, p. 146], $\Phi(t)$ satisfies

$$\begin{aligned} \frac{d\Phi}{dt} \Big|_{(s^*, i^*)} &= \mathcal{J}(t)\Phi(t) \\ &= \begin{pmatrix} A - 2S(t) - \beta_0 i^* & -\beta_0 s^* - \beta_0 S(t) \\ 0 & \beta_0 S(t) - (\sigma + g) \end{pmatrix} \Phi(t) \\ &\stackrel{(s^*, i^*) = (0,0)}{=} \begin{pmatrix} A - 2S(t) & -\beta_0 S(t) \\ 0 & \beta_0 S(t) - (\sigma + g) \end{pmatrix} \Phi(t), \end{aligned}$$

where $\mathcal{J}(t)$ is the Jacobian matrix of (20) around $(s^*, i^*) = (0, 0)$. The fundamental matrix $\Phi(t)$ can be written as

$$\Phi(t) = \begin{pmatrix} \exp \left\{ AT - 2 \int_0^T S(t) dt \right\} & \varphi_{1,2}(t) \\ 0 & \exp \left\{ \beta_0 \int_0^T S(t) dt - (\sigma + g) T \right\} \end{pmatrix}.$$

The exact form of $\varphi_{1,2}(t)$ is not necessary since it is not needed in the subsequent analysis. Since the linearization of the third and fourth equations of (20) results in

$$\begin{pmatrix} s(nT) \\ i(nT) \end{pmatrix} = \begin{pmatrix} 1 - p & 0 \\ 0 & 1 \end{pmatrix} \begin{pmatrix} s(nT^-) \\ i(nT^-) \end{pmatrix},$$

then the Floquet multipliers of $(S, 0)$ are the solutions in λ of:

$$\det \begin{pmatrix} (1 - p) \exp \left\{ AT - 2 \int_0^T S(t) dt \right\} - \lambda & \varphi_{1,2}(t) \\ 0 & \exp \left\{ \beta_0 \int_0^T S(t) dt - (\sigma + g) T \right\} - \lambda \end{pmatrix} = 0.$$

They are explicitly given by

$$\lambda_1 = (1 - p) \exp \left\{ AT - 2 \int_0^T S(t) dt \right\} > 0, \tag{21}$$

$$\lambda_2 = \exp \left\{ \beta_0 \int_0^T S(t) dt - (\sigma + g) T \right\} > 0. \tag{22}$$

Floquet multipliers associated to $(S, 0)$: From (21) we get

$$\begin{aligned} \lambda_1 &< 1 \\ \Leftrightarrow (1 - p) \exp \left\{ AT - 2 \int_0^T S(t) dt \right\} &< 1 \\ \Leftrightarrow \ln(1 - p) + AT &> 0 \\ \Leftrightarrow p &< p_1, \end{aligned}$$

and from (22) we deduce that

$$\begin{aligned} \lambda_2 &< 1 \\ \Leftrightarrow \exp \left\{ \beta_0 \int_0^T S(t) dt - (\sigma + g) T \right\} &< 1 \\ \Leftrightarrow \frac{1}{T} \int_0^T S(t) dt &< \frac{\sigma + g}{\beta_0} \stackrel{(11)}{=} S_c \\ \Leftrightarrow \frac{\beta_0}{\sigma + g} \frac{1}{T} \int_0^T S(t) dt &< 1 \\ \stackrel{(19)}{\Leftrightarrow} \mathcal{R}_p &< 1. \end{aligned}$$

Floquet multipliers associated to (0, 0):

$$\lambda_1 = (1 - p) e^{AT} \quad \text{and} \quad \lambda_2 = e^{-(\sigma+g)T}.$$

It is easy to check that $\lambda_1 < 1$ if and only if $p > p_1(T)$ and $\lambda_2 < 1$. The result is shown. \square

The maps p_1 and p_2 given in [Theorem A](#) admit inverse. Let us denote them by T_1 and T_2 , respectively, which can be explicitly given by

$$T_1(p) := \frac{|\ln(1 - p)|}{A} \quad \text{and} \quad T_2(p) := \frac{|\ln(1 - p)|}{A - S_c}, \quad \text{for } A > S_c.$$

Omitting the dependence of T_1 and T_2 on p , the following result is a consequence of [Proposition 3](#). It characterizes the stability of $(0, 0)$ and $(S, 0)$ as function of T (cf. [Fig. 2](#)).

Corollary 2. *With respect to (8), the following assertions hold for $p > 0$:*

- (1) *If $T < T_1(p)$, then there are no non-trivial periodic solutions; the trivial equilibrium $(0, 0)$ is stable;*
- (2) *If $T_1(p) < T < T_2(p)$, then the disease-free periodic solution $(S, 0)$ is stable and the trivial equilibrium $(0, 0)$ is unstable;*
- (3) *If $T > T_2(p)$, then both the periodic solution $(S, 0)$ and the trivial equilibrium $(0, 0)$ are unstable.*

8. Proof of (2) of [Theorem A](#)

For $T > 0$, if $p > p_1$, the unique compact and invariant set for the stroboscopic map (F_S, F_I) is $(0, 0)$. This is the unique candidate for the ω -limit of a trajectory of a planar differential equation (cf. [\[46\]](#)).

9. Proof of (3) of [Theorem A](#)

We prove the global stability of the disease-free periodic solution $(S, 0)$ given in [Lemma 6](#). By [Proposition 3](#), we know that if $p \in (p_2(T), p_1(T))$, then the non-trivial periodic solution $(S, 0)$ is locally asymptotically stable. Suppose that $(S(t), I(t))$, $t \in \mathbb{R}_0^+$ is a solution of [\(8\)](#) with positive initial conditions in \mathcal{M} (given in [Lemma 1](#)).

Lemma 9. *If $\mathcal{R}_p < 1$, then $\lim_{t \rightarrow +\infty} I(t) = 0$.*

Proof. According to [Lemma 5](#) we know that $x_1^* > 0$ if and only if $p < p_1$. From the first and third equations of [\(8\)](#), we see that, for any $0 < \varepsilon \ll 1$ there exists $T^\dagger \gg 1$, such that

$$S(t) < S(t) + \varepsilon, \tag{23}$$

for all $t > T^\dagger$. Substituting [\(23\)](#) into the second equation of [\(8\)](#), we obtain

$$\frac{dI(t)}{dt} \leq \beta_0 (S(t) + \varepsilon) I(t) - (\sigma + g) I(t).$$

Since $I(nT) = I(nT^-)$, for $t \in [T^\dagger + nT, T^\dagger + (n + 1)T)$, we have

$$\begin{aligned} & \frac{dI(t)}{dt} \leq [\beta_0 (S(t) + \varepsilon) - (\sigma + g)] I(t) \\ \Rightarrow & \int_{T^\dagger}^t \frac{dI(\tau)}{I(\tau)} \leq \int_{T^\dagger}^t [\beta_0 (S(\tau) + \varepsilon) - (\sigma + g)] d\tau \\ \Leftrightarrow & \ln I(t) - \ln I(T^\dagger) \leq \beta_0 \int_{T^\dagger}^t (S(\tau) + \varepsilon) d\tau - (\sigma + g) (t - T^\dagger) \\ \Leftrightarrow & I(t) \leq I(T^\dagger) \exp \left\{ \beta_0 \int_{T^\dagger}^t (S(\tau) + \varepsilon) d\tau - (\sigma + g) (t - T^\dagger) \right\}. \end{aligned} \tag{24}$$

By hypothesis, one knows that $\mathcal{R}_p < 1$. Since

$$\begin{aligned} & \mathcal{R}_p < 1 \\ \Leftrightarrow & \varepsilon > 0 \text{ suf. small } \frac{\beta_0}{(\sigma + g)} \frac{1}{(t - T^\dagger)} \int_{T^\dagger}^t (S(\tau) + \varepsilon) d\tau < 1 \\ \Leftrightarrow & \beta_0 \int_{T^\dagger}^t (S(\tau) + \varepsilon) d\tau - (\sigma + g) (t - T^\dagger) < 0, \end{aligned}$$

and $I(T^\dagger) \geq 0$, then from [\(24\)](#) we conclude that $\lim_{t \rightarrow \infty} I(t) = 0$. \square

Auxiliary variable

Since $S(t) > 0$ and $S(t) > 0$, for all $t \in \mathbb{R}_0^+$, we set:

$$x(t) = \ln \left(\frac{S(t)}{S(t)} \right) \Leftrightarrow S(t) = e^{x(t)} S(t). \tag{25}$$

Lemma 10. $x'(t) = -S(t) (e^{x(t)} - 1) - \beta_0 I(t)$

Proof. The proof follows from folklore derivative computations. Indeed,

$$\begin{aligned} x'(t) &\stackrel{I(t)=0}{=} \frac{S(t)(A - S(t)) - \beta_0 S(t)I(t)}{S(t)} - \frac{S(t)(A - S(t))}{S(t)} \\ &= A - S(t) - \beta_0 I(t) - A + S(t) \\ &\stackrel{(25)}{=} -e^{x(t)} S(t) - \beta_0 I(t) + S(t) \\ &= -S(t) (e^{x(t)} - 1) - \beta_0 I(t). \quad \square \end{aligned}$$

Lemma 9 states that, if $\mathcal{R}_p < 1$, then all solutions approach a disease free state ($I(t) = 0$). Evaluating the equality of **Lemma 10** when $I = 0$, we get: $x'(t) \leq -S(t) (e^{x(t)} - 1)$.

Lemma 11. For $T^\dagger \gg 1$ of the proof of **Lemma 9**, we have:

$$\begin{aligned} (1) \quad &\int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1} \leq - \int_{T^\dagger}^t S(\tau) d\tau \\ (2) \quad &\int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1} = - \left[(x(t) - x(T^\dagger)) - (\ln |e^{x(t)} - 1| - \ln |e^{x(T^\dagger)} - 1|) \right] \end{aligned}$$

Proof.

(1) From **Lemma 10**, since $\beta_0 I(t) \geq 0$, we may write

$$x'(t) \leq -S(t) (e^{x(t)} - 1),$$

for $t \in [T^\dagger + nT, T^\dagger + (n + 1)T)$, which implies:

$$\begin{aligned} \frac{dx(t)}{dt} &\leq -S(t) (e^{x(t)} - 1) \\ \int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1} &\leq - \int_{T^\dagger}^t S(\tau) d\tau. \end{aligned}$$

(2)

$$\begin{aligned} \int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1} &= - \int_{T^\dagger}^t \frac{e^{x(\tau)} - 1 - e^{x(\tau)}}{e^{x(\tau)} - 1} dx(\tau) \\ &= - \left[\int_{T^\dagger}^t dx(\tau) - \int_{T^\dagger}^t \frac{e^{x(\tau)}}{e^{x(\tau)} - 1} dx(\tau) \right] \\ &= - \left[(x(t) - x(T^\dagger)) - (\ln |e^{x(t)} - 1| - \ln |e^{x(T^\dagger)} - 1|) \right]. \quad \square \end{aligned}$$

For $T^\dagger \gg 1$ of the proof of **Lemma 9**, define the map $h : [T^\dagger, +\infty) \rightarrow \mathbb{R}$ as

$$h(t) = \left(e^{x(T^\dagger)} - 1 \right) \exp \left\{ - \left(x(T^\dagger) + \int_{T^\dagger}^t S(\tau) d\tau \right) \right\}.$$

Lemma 12. The following assertions hold:

- (1) $\lim_{t \rightarrow +\infty} h(t) = 0$
- (2) For all $t > T^\dagger$, we get $x(t) \leq -\ln(1 - h(t))$.

Proof.

(1) Since

$$\begin{aligned} &\lim_{t \rightarrow +\infty} \int_{T^\dagger}^t S(\tau) d\tau \\ &= \lim_{t \rightarrow +\infty} \left(-\ln(pe^{A(T-T^\dagger)} - pe^{AT} + e^{AT} - 1) + \ln(pe^{A(T-t)} + e^{AT}(1-p) - 1) \right) \end{aligned}$$

$$+A(t - T^\dagger) = +\infty,$$

then $\lim_{t \rightarrow +\infty} h(t) = 0$.

(2) Using Lemma 11, one gets

$$\begin{aligned} & \int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1} \leq - \int_{T^\dagger}^t S(\tau) d\tau \\ \stackrel{\text{Lemma 11}}{\Leftrightarrow} & - \left[(x(t) - x(T^\dagger)) - (\ln |e^{x(t)} - 1| - \ln |e^{x(T^\dagger)} - 1|) \right] \leq - \int_{T^\dagger}^t S(\tau) d\tau \\ \Leftrightarrow & \ln |e^{x(t)} - 1| \leq \ln |e^{x(T^\dagger)} - 1| + (x(t) - x(T^\dagger)) - \int_{T^\dagger}^t S(\tau) d\tau \\ \Leftrightarrow & -x(t) \geq \ln \left(1 - (e^{x(T^\dagger)} - 1) \exp \left\{ - \left(x(T^\dagger) + \int_{T^\dagger}^t S(\tau) d\tau \right) \right\} \right) \\ \Leftrightarrow & x(t) \leq -\ln(1 - h(t)). \quad \square \end{aligned}$$

On the other hand, since $\lim_{t \rightarrow \infty} I(t) = 0$, for any $0 < \varepsilon \ll 1$, there exist $T^\dagger > 0$, such that $I(t) \leq \varepsilon$ for $t > T^\dagger$. From Lemma 10 we conclude that

$$x'(t) \geq -S(t) (e^{x(t)} - 1 + \varepsilon),$$

and hence

$$\int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1 + \varepsilon} \geq - \int_{T^\dagger}^t S(\tau) d\tau. \tag{26}$$

In the next lemma, we evaluate the integral of the left hand side of (26).

Lemma 13.

$$\int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1 + \varepsilon} = - \left[(x(t) - x(T^\dagger)) - (\ln |e^{x(t)} - 1 + \varepsilon| - \ln |e^{x(T^\dagger)} - 1 + \varepsilon|) \right]$$

The proof follows the same lines as item (2) of Lemma 11. For $\varepsilon \geq 0$ and $T^\dagger \gg 1$ found in the proof of Lemma 9, define the map $h_\varepsilon : [T^\dagger, +\infty) \rightarrow \mathbb{R}$ as

$$h_\varepsilon(t) = (e^{x(T^\dagger)} - 1 + \varepsilon) \exp \left\{ - \left(x(T^\dagger) + \int_{T^\dagger}^t S(\tau) d\tau \right) \right\}.$$

Lemma 14. *The following conditions hold:*

- (1) $\lim_{t \rightarrow +\infty} h_\varepsilon(t) = 0$
- (2) *The following inequalities are equivalent:*

- (a) $\int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1 + \varepsilon} \geq - \int_{T^\dagger}^t S(\tau) d\tau$
- (b) $x(t) \geq \ln \left(\frac{1 - \varepsilon}{1 - h_\varepsilon(t)} \right)$

Proof.

- (1) The proof runs along the same lines as Lemma 11 (1st item).
- (2) The proof is a consequence of the following chain of equivalences (for $t > T^\dagger$):

$$\begin{aligned} & \int_{T^\dagger}^t \frac{dx(\tau)}{e^{x(\tau)} - 1 + \varepsilon} \geq - \int_{T^\dagger}^t S(\tau) d\tau \\ \Leftrightarrow & - \left[(x(t) - x(T^\dagger)) - (\ln |e^{x(t)} - 1 + \varepsilon| - \ln |e^{x(T^\dagger)} - 1 + \varepsilon|) \right] \geq - \int_{T^\dagger}^t S(\tau) d\tau \\ \Leftrightarrow & e^{x(t)} - 1 + \varepsilon \geq (e^{x(T^\dagger)} - 1 + \varepsilon) e^{x(t)} e^{-x(T^\dagger)} e^{-\int_{T^\dagger}^t S(\tau) d\tau} \\ \Leftrightarrow & 1 - \frac{1 - \varepsilon}{e^{x(t)}} \geq (e^{x(T^\dagger)} - 1 + \varepsilon) \exp \left\{ - \left(x(T^\dagger) + \int_{T^\dagger}^t S(\tau) d\tau \right) \right\} \\ \Leftrightarrow & e^{-x(t)} \leq \frac{1 - (e^{x(T^\dagger)} - 1 + \varepsilon) \exp \left\{ - \left(x(T^\dagger) + \int_{T^\dagger}^t S(\tau) d\tau \right) \right\}}{1 - \varepsilon} \\ \Leftrightarrow & x(t) \geq \ln \left(\frac{1 - \varepsilon}{1 - h_\varepsilon(t)} \right). \quad \square \end{aligned}$$

For a fixed $\varepsilon > 0$, combining Lemmas 12 and 14, there exists $t > T^\dagger(\varepsilon)$ such that

$$\ln\left(\frac{1-\varepsilon}{1-h_\varepsilon(t)}\right) \leq x(t) \leq -\ln(1-h(t)).$$

Since

$$\lim_{t \rightarrow +\infty} \ln\left(\frac{1-\varepsilon}{1-h_\varepsilon(t)}\right) = \ln(1-\varepsilon) \quad \text{and} \quad \lim_{t \rightarrow +\infty} -\ln(1-h(t)) = 0,$$

then, by the Squeezing Theorem, we get

$$\lim_{\varepsilon \rightarrow 0} \lim_{t \rightarrow +\infty} x(t) = \lim_{\varepsilon \rightarrow 0} \ln(1-\varepsilon) = 0.$$

Using the change of variable (25), we conclude that $S(t) \rightarrow S$ as $t \rightarrow \infty$, in the topology of pointwise convergence, i.e. $\lim_{t \rightarrow +\infty} \sup \{\text{dist}(S(t), S(t)) : t > T^\dagger\} = 0$. The proof of (3) of Theorem A is now complete.

10. Proof of (4) of Theorem A

In this section, we prove that system (8) is permanent, that is, there are positive constants $c_1, c_2, C_1, C_2, T_0 > 0$ such that for all solutions $(S(t), I(t))$ with initial conditions $S_0 > 0, I_0 > 0$, we have

$$c_1 \leq S(t) \leq C_1 \quad \text{and} \quad c_2 \leq I(t) \leq C_2,$$

for all $t \geq T_0$. Constants C_1 and C_2 come from Lemma 1. Then we may take (for instance)

$$C_1 = C_2 = \frac{A(\sigma + g + A)}{\sigma + g} > 0.$$

In the region ④ defined by $R_p > 1$, the constant c_1 comes from the time average (18)

$$1 < R_p = \frac{\beta_0}{\sigma + g} \frac{1}{T} \int_0^T S(t) dt.$$

We only need to prove that there exists a constant $c_2 > 0$ such that $I(t) \geq c_2$ for t large enough. This assumption will be proved by contradiction, i.e. we assume that for all $c_2 > 0$, we have (see Fig. 6):

- (1) $I(t) < c_2$ for all $t \geq t_1$ or
- (2) $I(t) < c_2$ at infinitely many subintervals of $[t_1, +\infty)$. Let $t^* = \inf_{t > t_1} \{I(t) < c_2\}$. There are two possibilities for such a t^* :
 - (a) $t^* = n_1 T$, for $n_1 \in \mathbb{N}$ and
 - (b) $t^* \neq n_1 T$, for $n_1 \in \mathbb{N}$.

Case (1) Assuming that $I(t) < c_2$ for all $t \geq t_1$, by including the $-\beta_0 c_2 S(t) < 0$ in (12) we get:

$$\begin{cases} \dot{S} \geq S(t)(A - S(t)) - \beta_0 c_2 S(t), & t \neq nT \\ S(t^+) \geq (1-p)S(t), & t = nT. \end{cases}$$

Now, let $z(t)$ be the solution of the following system:

$$\begin{cases} \dot{z}(t) = z(t)(A - z(t)) - \beta_0 c_2 z(t), & t \neq nT \\ z(t^+) = (1-p)z(t), & t = nT. \end{cases} \tag{27}$$

Solving (27) as in (12), for $c_2 > 0$ arbitrarily small, we conclude that it has a periodic solution given explicitly by

$$\begin{aligned} \mathcal{Z}(t) &= \frac{K}{1 + \frac{p}{K((1-p) - e^{-KT})} K e^{-K(t-nT)}} = \frac{K((1-p) - e^{-KT})}{(1-p) - e^{-KT} + p e^{-K(t-nT)}}, \\ &= \frac{K}{1 + \frac{p}{(1-p) - e^{-KT}} e^{-K(t-nT)}} \end{aligned}$$

where $nT \leq t < (n+1)T, t > t_1, n \in \mathbb{N}$ and $K := -\beta_0 c_2 + A$.

Now it is easy to conclude that $S(t) \geq z(t), S(0^+) \geq z(0^+)$, and $z(t) \rightarrow \mathcal{Z}(t)$ in the pointwise convergence topology, where \mathcal{Z} is the periodic solution of (27). Therefore, there exist $\varepsilon_1 > 0$ (arbitrarily small) and $t_2 > t_1$ such that $S(t) \geq z(t) > \mathcal{Z}(t) - \varepsilon_1$; from (8) and for $t > t_2$ we may write

$$\begin{cases} \dot{I}(t) \geq [\beta_0 (\mathcal{Z}(t) - \varepsilon_1) - (\sigma + g)] I(t), & t \neq nT \\ I(nT^+) = I(nT). \end{cases} \tag{28}$$

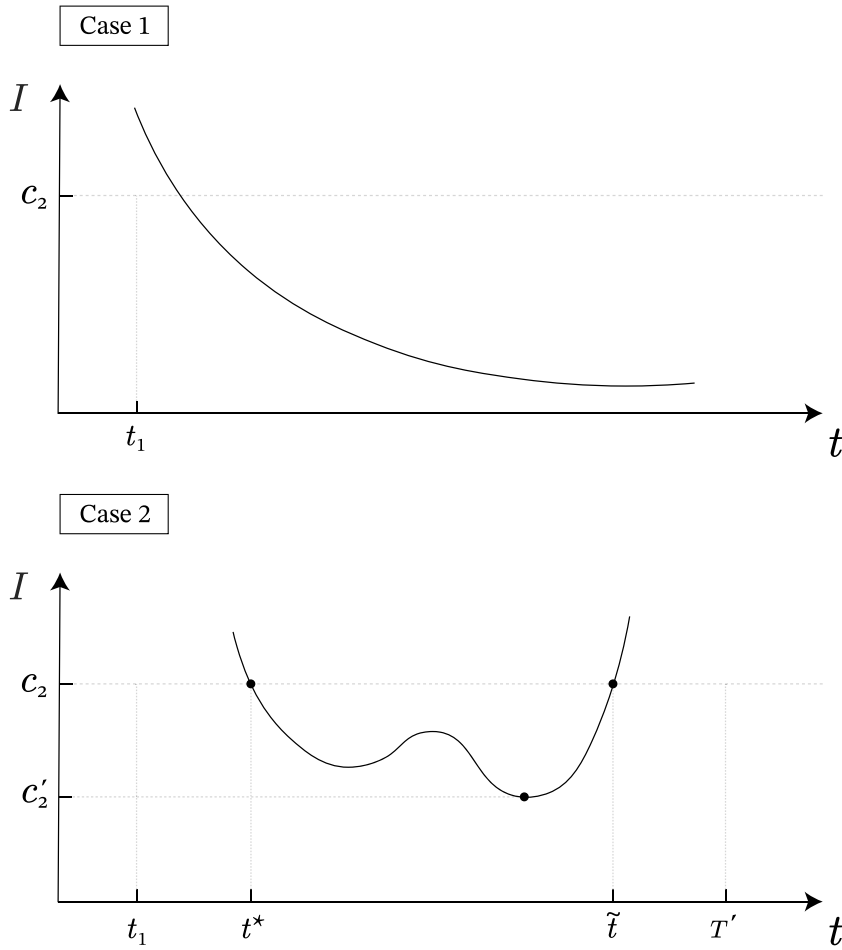


Fig. 6. Illustration of Cases 1 and (2a).

Let $N \in \mathbb{N}$ and $NT \geq t_2$. Integrating (28) between $(nT, (n+1)T]$, $n \geq N$, we have $I((n+1)T) \geq I(nT)\gamma$, where

$$\gamma = \exp \left\{ \int_{nT}^{(n+1)T} \beta_0 \mathcal{Z}(t) dt - (\beta_0 \varepsilon_1 + (\sigma + g))T \right\}.$$

Using induction over $N \in \mathbb{N}$ we get

$$I((N+n)T) \geq I(NT)\gamma^n. \tag{29}$$

Claim. If $\mathcal{R}_p > 1$, then $\gamma > 1$.

Proof. The proof follows from the chain of equivalences:

$$\begin{aligned} & \mathcal{R}_p > 1 \\ \Leftrightarrow & \stackrel{(19) \text{ and } \varepsilon_1 \geq 0}{\frac{\beta_0}{\sigma + g} \frac{1}{T} \int_{nT}^{(n+1)T} (\mathcal{Z}(t) - \varepsilon_1) dt > 1} \\ \Leftrightarrow & \exp \left\{ \int_{nT}^{(n+1)T} \beta_0 \mathcal{Z}(t) dt - (\beta_0 \varepsilon_1 + (\sigma + g))T \right\} > 1 \\ \Leftrightarrow & \gamma > 1. \quad \square \end{aligned}$$

Since $\gamma > 1$, we see that $0 < I(nT)\gamma^n \rightarrow \infty$ as $n \rightarrow \infty$, which is a contradiction by Lemma 1. So, there exist $c_2, T_0 > 0$ such that $I(t) > c_2$ for all $t \geq T_0$.

Case (2a) Let $t^* = n_1T, n_1 \in \mathbb{N}$. Then, $I(t) \geq c_2$ for $t \in [t_1, t^*]$ and $I(t^*) = c_2$ and I is decreasing. By Lemma 2, we have

$$I(t) < 0 \Leftrightarrow \beta_0 S(t) < \sigma + g \stackrel{0 < c_1 \leq S(t)}{\Rightarrow} \beta_0 c_1 < \sigma + g.$$

Hence, we can choose $n_2, n_3 \in \mathbb{N}$ such that $t^* + n_2T > t_1$ and

$$\gamma^{n_3} \exp\left\{(\beta_0 c_1 - \sigma - g)n_2T\right\} > \gamma^{n_3} \exp\left\{(\beta_0 c_1 - \sigma - g)(n_2 + 1)T\right\} > 1.$$

and define $T' = n_2T + n_3T$.

Claim. *There exists $t_2 \in (t^*, t^* + T']$ such that $I(t_2) > c_2$.*

Proof. Let us assume that this is not true, i.e. that there is no such t_2 .

Since $z(t) \rightarrow \bar{z}(t)$ as $t \rightarrow \infty$ (in the pointwise convergence topology), as above, we may write $\bar{z}(t) - \varepsilon_1 < z(t) \leq S(t)$, for $t^* + n_2T \leq t \leq t^* + T'$. From (28) and for $t^* + n_2T \leq t \leq t^* + T'$, we have (see Eq. (29))

$$I(t^* + T') \geq I(t^* + n_2T)\gamma^{n_3}.$$

From system (8), we get

$$\begin{cases} \dot{I}(t) \geq (\beta_0 c_1 - (\sigma + g)) I(t), & t \neq nT, \\ I(t^+) = I(t), & t = nT. \end{cases} \tag{30}$$

for $t \in [t^*, t^* + n_2T]$. Integrating (30) between t^* and $t^* + n_2T$, we have:

$$\begin{aligned} \int_{t^*}^{t^* + n_2T} \frac{dI(t)}{I(t)} &\geq \int_{t^*}^{t^* + n_2T} (\beta_0 c_1 - (\sigma + g)) dt \\ \Leftrightarrow I(t^* + n_2T) &\geq I(t^*) \exp\left\{(\beta_0 c_1 - (\sigma + g))n_2T\right\} \\ \Leftrightarrow I(t^* + n_2T) &\geq c_2 \exp\left\{[\beta_0 c_1 - (\sigma + g)]n_2T\right\}, \end{aligned}$$

leading to

$$I(t^* + T') \geq c_2 \exp\left\{[\beta_0 c_1 - (\sigma + g)]n_2T\right\} \gamma^{n_3} > c_2,$$

which is a contradiction, since we have assumed that no $t \in (t^*, t^* + T']$ exists such that $I(t) > c_2$. \square

Let $\tilde{t} = \inf_{t > t^*} \{I(t) > c_2\}$. Then, for $t \in (t^*, \tilde{t})$, $I(t) \leq c_2$ and $I(\tilde{t}) = c_2$, since $I(t)$ is continuous and $I(t^+) = I(t)$ when $t = nT$. For $t \in (t^*, \tilde{t})$, suppose

$$t \in (t^* + (k - 1)T, t^* + kT], \quad k \in \mathbb{N} \quad \text{and} \quad k \leq n_2 + n_3.$$

Therefore, from (30) we have

$$\begin{aligned} I(t) &\geq I(t^*) \exp\left\{(k - 1)(\beta_0 c_1 - (\sigma + g))T\right\} \exp\left\{(\beta_0 c_1 - (\sigma + g))(t - (t^* + (k - 1)T))\right\} \\ &\geq c_2 \exp\left\{k(\beta_0 c_1 - (\sigma + g))T\right\} \\ &\geq c_2 \exp\left\{(n_2 + n_3)(\beta_0 c_1 - (\sigma + g))T\right\}. \end{aligned}$$

Let $c'_2 = c_2 \exp\left\{(n_2 + n_3)(\beta_0 c_1 - (\sigma + g))T\right\} < c_2$. This lower bound does not depend neither on t^* nor \tilde{t} . Hence, we have $I(t) \geq c'_2$ for $t \in (t^*, \tilde{t})$ and then for all $t > t_1$. For $t > \tilde{t}$, the same argument can be extended to $+\infty$ since $I(\tilde{t}) \geq c_2$ and c'_2 does not depend on the interval. This is a contradiction. Hence, there exist $c_2, T_0 > 0$ such that $I(t) > c_2$ for all $t \geq T_0$. The proof of Case (2b) is entirely analogous and it is left to the reader.

The Poincaré–Bendixson theorem for impulsive planar flows [46, Theorem 3.9] says that the ω -limit of (8) is an equilibrium, a periodic solution or a union of saddles that are heteroclinically connected. The only equilibrium of (8) is the origin. In order to allow endemic T -periodic solutions for F_I , then $\frac{1}{T} \int_0^T S(t) dt = S_c$. In this case, we know that there is an implicit (positive) map $I \equiv I(t)$, where $t \in \mathbb{R}_0^+$. Since there are no more candidates for ω -limit sets, we have in the topological closure of the first quadrant the following limit sets:

$$(0, 0), (S, 0) \text{ and } (S, I).$$

Since $(0, 0)$ is repulsive (by Proposition 3), $(S, 0)$ is a saddle (attracting in the first component and repelling in the second), the solutions should converge to the endemic periodic solution (S, I) whose explicit solution is not known.

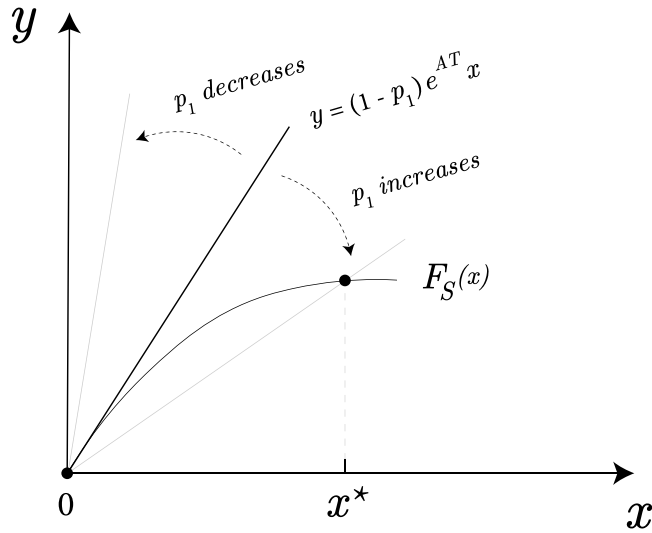


Fig. 7. Illustration of the existence of a degenerate saddle-node bifurcation at $p = p_1(T)$, for $T > 0$ fixed.

11. Proof of (6) of Theorem A

For $x \geq 0$, it is easy to check that $F'_S(x) = \frac{P(x)}{Q^2(x)}$, where

$$P(x) = A(1 - p)e^{AT}(x(e^{AT} - 1) + A) - Ax(1 - p)e^{AT}(e^{AT} - 1)$$

and

$$Q(x) = x(e^{AT} - 1) + A.$$

In particular, we have $F'_S(0) = (1 - p)e^{AT}$. Since

$$(1 - p)e^{AT} = 1 \iff p = p_1(T),$$

then we conclude that p_1 corresponds to a saddle-node bifurcation of F_S at $x = 0$. Indeed, if $p > p_1$, then the map F_S does not have fixed points besides $x = 0$; if $p < p_1$, then one extra fixed point emerges, as shown in Fig. 7. In terms of p_2 , if $p > p_2$, then the periodic solution $(S, 0)$ changes its stability because the Floquet multiplier λ_2 (cf. (22)) crosses the unit circle; this is why we have a transcritical bifurcation at $p = p_2$.

12. Proof of (7) of Theorem A

Item (7) is proved in Lemmas 7 and 8 of Section 6.

13. Proof of Corollary 1

For $\gamma, \omega > 0$ and $A > S_c$, there is a hyperbolic T -periodic solution $(S, 0)$ whose existence does not depend on β_γ . Therefore, it exists if $p < p_1(T)$ by Lemma 5. However, the region of the phase space where it is stable depends on γ . Indeed,

$$\begin{aligned} & \lambda_2 < 1 \\ \Leftrightarrow & \text{(22), adapted } \exp \left\{ \int_0^T \beta_\gamma(t)S(t) dt - (\sigma + g)T \right\} < 1 \\ \Leftrightarrow & \int_0^T \beta_0 (1 + \gamma\Psi(\omega t)) S(t) dt - (\sigma + g)T < 0 \\ \Leftrightarrow & \frac{1}{(\sigma + g)T} \int_0^T \beta_0 S(t) dt + \frac{1}{(\sigma + g)T} \int_0^T \beta_0 \gamma\Psi(\omega t) S(t) dt < 1 \\ \Leftrightarrow & \frac{\beta_0}{(\sigma + g)T} \int_0^T S(t) dt + \frac{\beta_0}{(\sigma + g)T} \int_0^T \gamma\Psi(\omega t) S(t) dt < 1 \\ \Leftrightarrow & \text{(10) } \frac{1}{S_c T} \int_0^T S(t) dt + \frac{1}{S_c T} \int_0^T \gamma\Psi(\omega t) S(t) dt < 1 \end{aligned}$$

$$\begin{aligned} \Leftrightarrow & \quad [\ln(1 - p) + AT] + \gamma \int_0^T \Psi(\omega t)S(t) dt < S_c T \\ \Leftrightarrow & \quad p > 1 - \exp \left\{ - \left[(A - S_c)T + \gamma \int_0^T \Psi(\omega t)S(t) dt \right] \right\} \\ \Leftrightarrow & \quad p > p_2^{\text{seas}}(T). \end{aligned}$$

14. Proof of Theorem B

The proof follows from the item (4) of Theorem A combined with [47,48] and Section 3.2 of [49],⁷ taking into account the following considerations:

- $\omega \in \mathbb{R}^+$ plays the role of “shear” of [49];
- the term $\beta_\gamma(t) = \beta_0(1 + \gamma\Psi(\omega t)) > 0$ may be seen as the radial “kick” of (4) that is periodic in time;
- the non-autonomous periodic forcing of (4) has nondegenerate critical points (by (C4)) – this avoids constant maps;
- the endemic periodic solution (S, I) of (4) of Theorem A is globally attracting for (8).

For $\gamma > 0$, Eq. (3) is equivalent to

$$\dot{x} = f_\gamma(x) \Leftrightarrow \begin{cases} \dot{S} = S(A - S) - \beta_\gamma(t)IS \\ \dot{I} = \beta_\gamma(t)IS - (\sigma + g)I, & t \neq nT \\ \dot{\theta} = \omega \pmod{2\pi} \\ S(nT) = (1 - p)S(nT^-) \\ I(nT) = I(nT^-) \end{cases} \quad (31)$$

whose flow lies in $(\mathbb{R}_0^+)^2 \times \mathbb{S}^1$, where $\mathbb{S}^1 \equiv \mathbb{R} \pmod{\pi/\omega}$. Since the kicks are radial, they do not affect the θ -coordinate. The following argument follows from [47,48] and Section 3.2 of [50].

Using item (4) of Theorem A applied to the amplitude component of (31), one knows that the ω -limit of Lebesgue almost all solutions of (31) is a strict subset of a two-dimensional attracting torus \mathcal{T}_0 (normally hyperbolic manifold). The torus is ergodic when T is not commensurable with ω/τ . Furthermore, for a dense set Δ of pairs $(T, \omega) \in [T_2, +\infty) \times \mathbb{R}^+$ such that $T = k\tau/\omega$, one knows that this normally hyperbolic manifold is foliated by periodic solutions, as depicted on the left-hand side of Fig. 8. In the terminology of Herman [51], the set Δ corresponds to orbits with rational rotation number.

Let Σ be a cross-section to \mathcal{T}_0 where the first return is well defined. It is easy to observe that the set $\Sigma \cap \mathcal{T}_0$ is part of a circle. If $(T, \omega) \in \Delta$ and $\gamma = 0$, there is at least one pair of periodic solutions in \mathcal{T}_0 which are heteroclinically connected, say q_1 (saddle) and q_2 (sink). The rotation number remains constant within a synchronization zone (valid for $\gamma > 0$), also known as Arnold’s tongue [52].

Since \mathcal{T}_0 is a normally hyperbolic manifold, then for $\gamma \gtrsim 0$, there is a manifold \mathcal{T}_γ diffeomorphic to \mathcal{T}_0 , which is still attracting; as γ increases further, the set $\Sigma \cap \mathcal{T}_\gamma$ becomes “non-smooth” and three generic⁸ scenarios may occur:

- (A) the circle loses its smoothness (near q_2) when a pair of multipliers of the cycle becomes complex (non-real) or one of the multipliers is negative. At the moment of bifurcation, the length of an invariant circle becomes infinite and the torus is destroyed. The transition to chaos can come either from a period-doubling bifurcation cascade or via the breakdown of a torus occurring near q_2 (route A of [48, pp. 124]);
- (B) homo or heteroclinic tangle of the dissipative saddle q_1 (route B of [48, pp. 124]);
- (C) distortion of the unstable manifold near a non-hyperbolic saddle-node; the torus becomes non-smooth (route C of [48, pp. 124]).

Following [48], the three mechanisms of torus destruction leads to a (non-hyperbolic) topological horseshoe-type map with a smooth bend. They do not cause the absorbing area to change abruptly and thus represent the bifurcation mechanism of a soft transition to chaos. The torus destruction line in the two control parameter plane (ω, γ) is characterized by a complex structure [47,48,53]. There are small regions (in terms of measure) inside the resonance wedges where chaotic trajectories are observable: they correspond to strange attractors of Hénon type and are associated with historic behavior. Other stable points with large period exist as a consequence of the Newhouse phenomena. For a better understanding of Route (B), see Fig. 8. Compare also with Figure 2.10 of [48].

15. Numerics

In this section we perform some numerical simulations to illustrate the contents of Theorems A and B. All simulations were performed via MATLAB_R2018a software.

⁷ See the comment before Theorem 2 of [49], where the authors refer impulsive differential equations.

⁸ Valid in a residual set within the set of one-parameter families $(f_\gamma)_\gamma$ periodically perturbed by maps satisfying (C4).

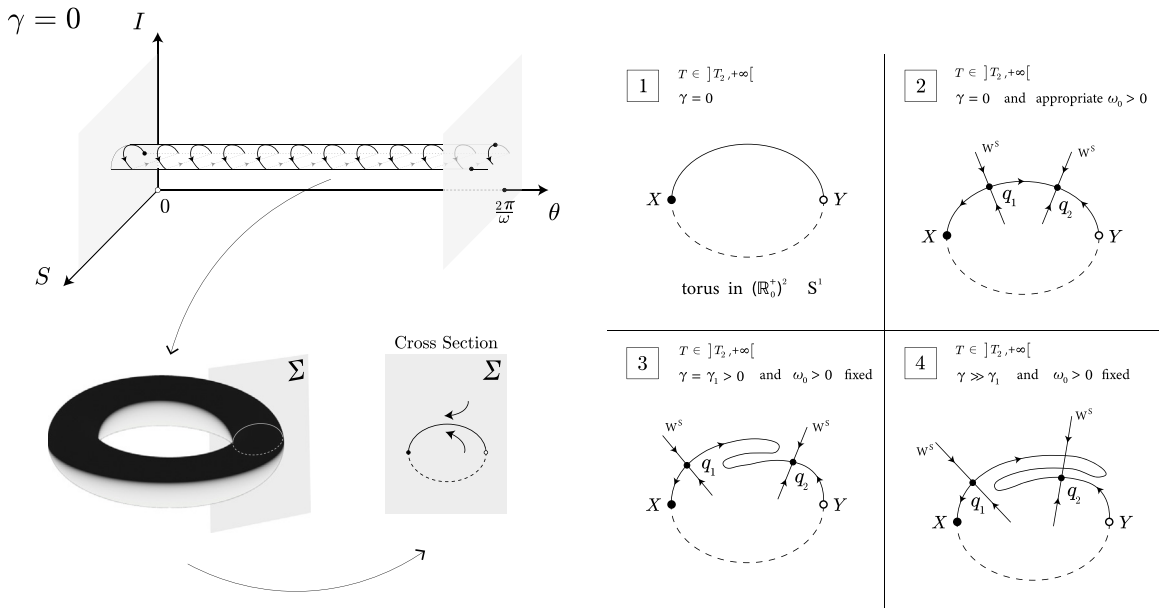


Fig. 8. Sketch for the proof of Theorem B: (1). Torus bifurcation for $\gamma = 0$. (2). For $\gamma = 0$ and an appropriate $\omega \in \mathbb{R}^+$, we observe the emergence of periodic solutions represented by q_1 and q_2 on \mathcal{T}_0 . (3 and 4). As γ increases, the intersection $\Sigma \cap \mathcal{T}_0$ starts to lose its smoothness. In (4), the unstable manifold $W^u(q_1)$ intersects the stable manifold $W^s(q_2)$, leading to the formation of homoclinic tangles that indicate the emergence of horseshoes (chaos) – compare with Figure 2.10 of [48].

Fig. 9: Numerical simulation of the five scenarios of system (8) illustrated in the bifurcation diagram (T, p) in Fig. 2 with initial condition $(S_0, I_0) = (0.5, 0.4)$ described by Theorem A. The figure shows the behavior of the Susceptible and Infectious over time for different values of p and corresponding phase space. Parameter values: $A = 1, \beta_0 = 0.9, \sigma = 0.2, g = 0.5, T = 4$ and $p \in [0, 1]$. The green line represents the disease-free non-trivial periodic solution $(S, 0)$, and the red line represents the endemic periodic solution (S, I) .

Fig. 10: Projection of the solution of (31) with initial condition $(S_0, I_0, \theta_0) = (0.5, 0.4, 0)$ for different values of γ . The parameter values are strategically chosen in order to have the dynamics of Region ③ of Fig. 2: $A = 1, \beta_0 = 0.2, \sigma = 0.05, g = 0.02, T = 1, \omega = 0.1$ and $p = 0.5$. The dynamics of S and I show that the system tends towards the disease-free periodic solution $(S, 0)$. As the amplitude of the seasonal variation γ increases, the disease-free periodic solution starts to deform.

Fig. 11: Numerical simulation of the phase space $(S, I), (S, I, \theta)$, and respective cross-section $(\theta = 2)$ for three different values of γ of (31). Parameter values used in this simulation: $A = 1, T = 4, \beta_0 = 2, \omega = 6, \sigma = 0.2, g = 0.5$ and $p = 0.4$ with initial condition $(S_0, I_0, \theta_0) = (0.4074, 0.2645, 0)$. This simulation corresponds to Region ④, where $\mathcal{R}_p > 1$. For this initial condition and $\gamma = 4.89$, there is a positive Lyapunov exponent (0.13).

16. Discussion and final remarks

In this work, we have studied a modified SIR model, introducing logistic growth to the population of the Susceptible individuals and incorporating pulse vaccination (Susceptible individuals are T -periodically vaccinated), conferring immunity to a proportion p of the Susceptible population. Additionally, we have explored the model with and without seasonality into the disease transmission rate.

Results

Regarding our first main result (Theorem A), in the absence of seasonality in the disease transmission rate ($\gamma = 0$), the analysis of the model reveals five stationary scenarios, depicted in the (T, p) bifurcation diagram of Figs. 2 and 9. The non-trivial periodic ω -limit sets have the same period as the initial pulse.

For $p = 0$ and $\mathcal{R}_0 > 1$, the outcomes align with those of [10]: the ω -limit of all solutions with initial condition $S_0, I_0 > 0$ is the endemic equilibrium. For $T > 0$ fixed and values of p between 0 and $p_2(T)$, our model is permanent. In this case, the associated basic reproduction number is greater than 1. This agrees well with the findings of [16,54,55]. Our contribution goes further in this direction — we have proved the existence of a globally stable endemic T -periodic solution.

For $p \in (p_2(T), p_1(T))$, system (8) exhibits a disease-free non-trivial periodic solution, where the curves $p = p_1(T)$ and $p = p_2(T)$ correspond to saddle-node and transcritical bifurcations, respectively.

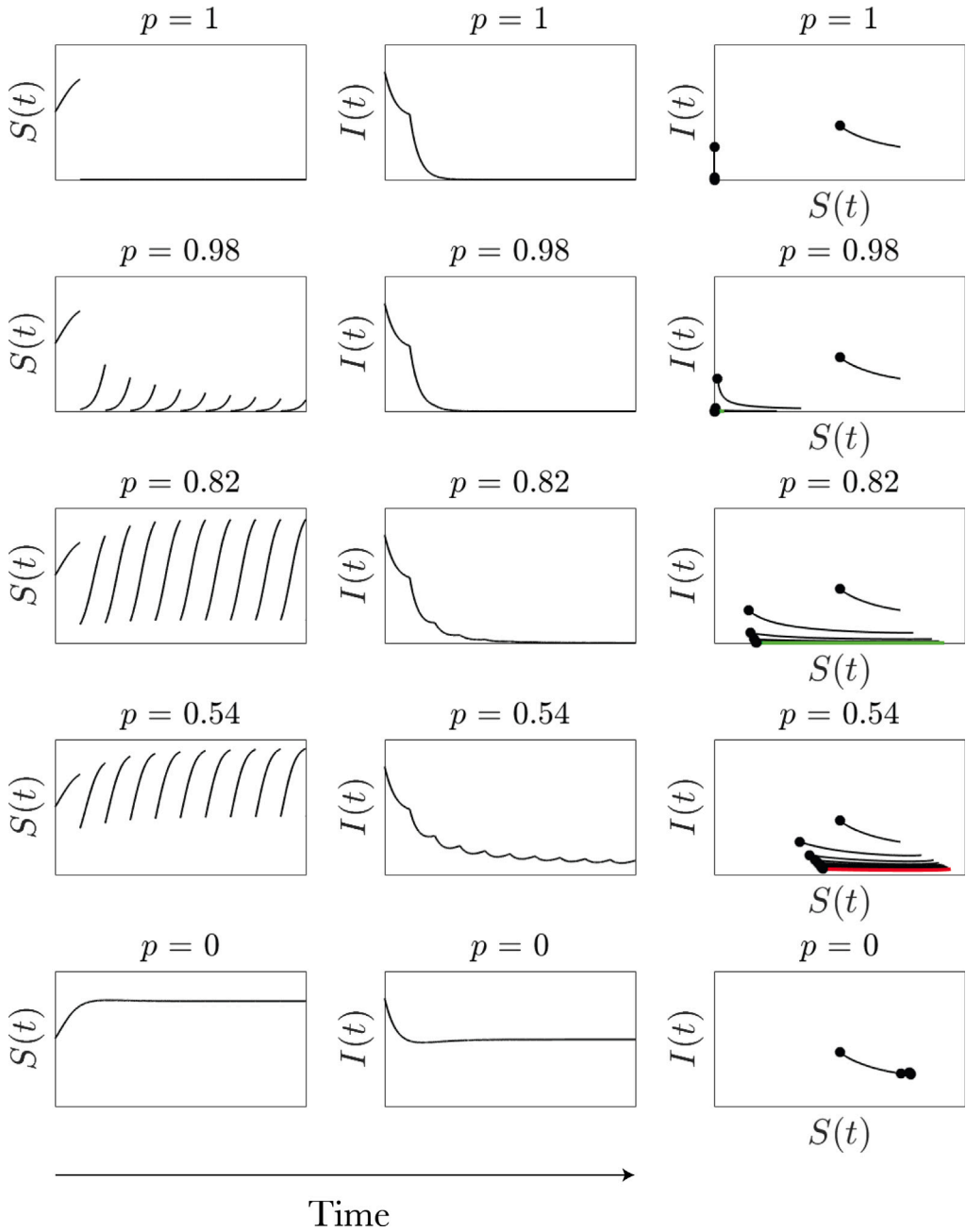


Fig. 9. Numerical simulation of the five scenarios of system (8) illustrated in the bifurcation diagram (T, p) of Fig. 2 with initial condition $(S_0, I_0) = (0.5, 0.4)$. The figure shows the behavior of the Susceptible and Infectious over time for different values of p and corresponding phase space. Parameter values: $A = 1$, $\beta_0 = 0.9$, $\sigma = 0.2$, $g = 0.5$, $T = 4$ and $p \in [0, 1]$. The green line represents the disease-free non-trivial periodic solution $(S, 0)$, and the red line represents the endemic periodic solution (S, I) .

Under the effect of seasonality in the disease transmission rate, if $p < p_2(T)$, $k\tau/\omega = T$ (within a resonance wedge), and $\gamma \gg 1$ (large), Theorem B indicates that the flow of (3) exhibits a suspended topological horseshoe. Consequently, the number of Infectious individuals persists and is more difficult to control the disease. The proof of this result follows the same lines of [48]. Our results stress that insufficient vaccination coverage combined with seasonality can generate chaotic dynamics.

We have presented numerical simulations to demonstrate that the periodic nature of pulse vaccination combined with seasonality might spread of the disease. Corollary 1 and Fig. 4 warn that neglecting the effect of the amplitude of the seasonal transmission could cause an over-optimistic approximation of the optimal pulse period [43].

Phase space (S, I)

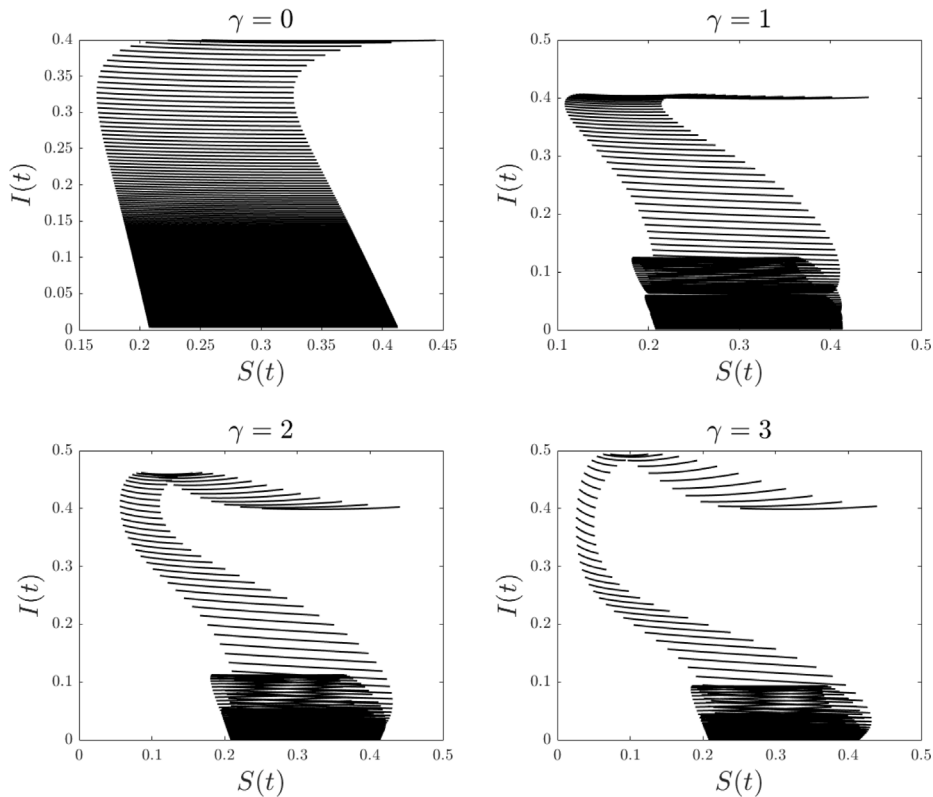


Fig. 10. Projection of the solution of (31) with initial condition $(S_0, I_0, \theta_0) = (0.5, 0.4, 0)$ for different values of γ . The parameter values are strategically chosen in order to have the dynamics of Region ③ of Fig. 2: $A = 1, \beta_0 = 0.2, \sigma = 0.05, g = 0.02, T = 1, \omega = 0.1$ and $p = 0.5$. The dynamics of S and I show that the system tends towards the disease-free periodic solution $(S, 0)$.

Literature

In a recent study of a SIR model with pulse vaccination, the author of [44] pointed out two mechanisms that lead a classical SIR model to chaotic dynamics with low vaccine coverage. The first mechanism arises from the interaction of low birth and death rates combined with a high contact rate between individuals. The second mechanism results from high birth and low contact rates.

In our paper, we have analytically proved the emergence of chaos through a different technique: modulating the contact rate of the disease through a periodic function (seasonality), system (3) may exhibit chaos under generic assumptions. Mathematically, the author of [44] proved chaos via the Zanolin’s method [56] (stretching rectangles along paths); in our paper, chaos emerges via Torus-breakdown theory [47] and [48, §2.1.4].

Pulse vs. constant vaccination

We compare the findings of [10,12] with the present work. Pulse vaccination requires a smaller percentage of people vaccinated to prevent a possible epidemic outbreak. Since pulse vaccination is periodic, it is enough to vaccinate a proportion p of the population to keep the number of *Susceptible* individuals below prescribed epidemic limits. On the other hand, constant vaccination requires a high rate of *Susceptible* individuals to be vaccinated to avoid spreading the disease.

While constant vaccination offers constant and stable protection to the population, pulse vaccination can lead to poorly planned epidemic outbreaks, especially if the number of *Susceptible* individuals exceeds epidemic limits.

Open problem

Since C^2 -hyperbolic horseshoes have zero Lebesgue measure, it is possible for a map to have a horseshoe, and at the same time, the orbit of Lebesgue-almost every point tends to a sink. Adapting the proof of [49] we conjecture that (concerning system (4)) if

Cross Section

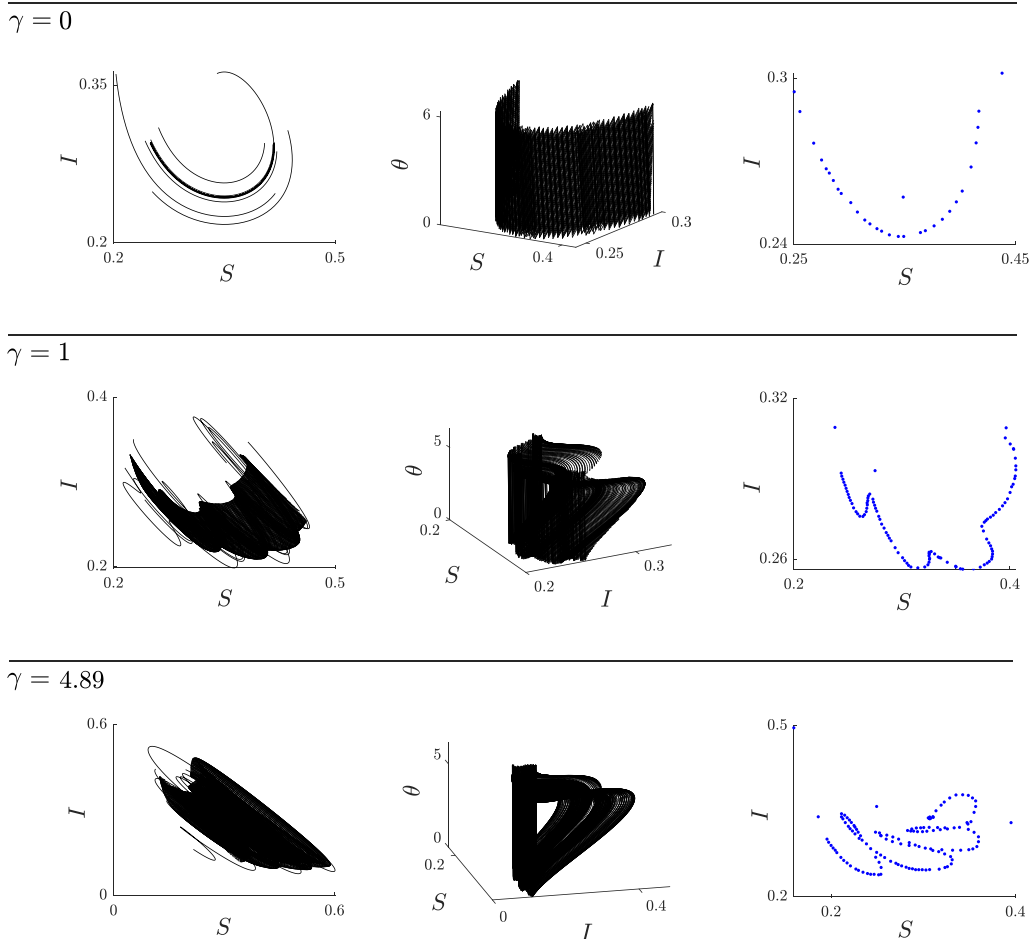


Fig. 11. Numerical simulation of the phase space (S, I) , (S, I, θ) , and respective cross-section $(\theta = 2)$ for three different values of γ of (31). Parameter values used in this simulation: $A = 1$, $T = 4$, $\beta_0 = 2$, $\omega = 6$, $\sigma = 0.2$, $g = 0.5$ and $p = 0.4$ with initial condition $(S_0, I_0, \theta_0) = (0.4074, 0.2645, 0)$. This simulation corresponds to Region ④, where $\mathcal{R}_p > 1$. The system exhibits chaos if $\gamma = 4.89$ with a positive Lyapunov exponent (0.13).

ω is large enough, then

$$\liminf_{\varepsilon \rightarrow 0^+} \frac{\text{Leb} \{ \gamma \in [0, \varepsilon] : f_\gamma \text{ exhibits a strange attractor in the sense of [49]} \}}{\varepsilon} > 0,$$

where Leb denotes the one-dimensional Lebesgue measure. Within the strange attractor, orbits jump around in a seemingly random fashion, and the future of individual orbits appears entirely unpredictable. For these chaotic attractors, however, there are laws of statistics that control the asymptotic distributions of Lebesgue almost all orbits in the basin of attraction of the attractor.

Final remarks and future work

Our study suggests that the effective strategy for controlling an infectious disease governed by (3) must be in a way that the proportion p of vaccination is at the target level needed for the disease eradication, and the time T between shots must be appropriate. The results suggest that maintaining a high proportion p of vaccination and optimizing the period T between two shots shall increase the effectiveness of the vaccination strategy. The inclusion of seasonality has dramatic impacts on the dynamics.

One of the main challenges of the World Health Organization is the global eradication of certain diseases in countries with low incomes and high birth rates. From an applied perspective, our results could be helpful for the optimal design of vaccination programs.

Further refinements of the pulse vaccination strategy need to take seasonal disease dynamics into full account. From a theoretical perspective, there is a need to develop analytical epidemiological models that incorporate information such as the natural period of the disease under analysis (resonance dynamics of [43]).

Some lines of research need to be tackled such as the dependence of vaccination efficacy over time, multiresolution modeling and the availability of medical resources [57]. Cross-immunity and delayed vaccination are important factors to be considered [58]. In addition, strategies such as preventive vaccination and the impact of the media should also be explored [59,60]. Finally, pulse vaccination modeling can be extended to include multi-group epidemic models, providing a more detailed and realistic analysis of disease dynamics in distinct subpopulations. These directions could significantly improve our understanding of the epidemiological models.

CRedit authorship contribution statement

João P.S. Maurício de Carvalho: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Alexandre A. Rodrigues:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Joao Mauricio Carvalho reports financial support was provided by Foundation for Science and Technology. Alexandre Rodrigues reports financial support was provided by the Project CEMAPRE/REM financed by Foundation for Science and Technology.

Data availability

No data was used for the research described in the article.

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