RESEARCH ARTICLE

Dynamics of An SIR Model with Isolation and Pulse Vaccinated Susceptible Population for Delay Acquiring Immunity

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Abstract

Vaccination is one of the most cost-effective and simplest interventions for protecting against infectious-disease epidemics. The COVID-19 (coronavirus disease 2019) outbreak in China demonstrated that physical protection and social isolation are critical to controlling an epidemic in the absence of vaccines or antiviral drugs. Considering isolation and pulse vaccination of the susceptible population subject to a delay in acquiring immunity, we propose a susceptible-infectious-recovered (SIR) model incorporating these features. Using the theory of impulsive differential equations and comparison theorems, we prove that the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) is globally asymptotically stable if $H < 0$, and that the disease tends to become endemic if $H > 0$. The expression of H and the numerical simulations indicate that isolation and the delay in acquiring immunity of the vaccinated susceptible population play important roles in achieving the infection-free state, and that the maximum enrolling amount from the exterior region also affects disease elimination.

Keywords: SIR model, isolation, pulse vaccination, delay in acquiring immunity, infection-free periodic solution, endemic disease.

1 Introduction

Vaccines represent one of the most cost-effective and simple interventions to protect humans against epidemics. Vaccination brings mortality- and morbidity-related benefits by preventing infectious diseases; these include financial benefits from avoiding hospitalization, preventing long-term disability, and increasing productivity [1]. Skrzat-Klapaczyńska et al. [2] found that, within a cocoon vaccination strategy, healthcare workers developed anti-SARS-CoV-2 S-RBD antibodies as early as 14 days after the second vaccine dose. Shulgin et al. [3] pioneered

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the study of pulse vaccination strategy in the SIR epidemic model. Subsequently, many authors [4–7] investigated delayed SIR, network-based SIR, and state-dependent SIR epidemic models with pulse vaccination. In particular, Jiao et al. [4] studied impulsive vaccination together with restricting infected individuals from boarding transports. In order to understand the effect of impulsive vaccination on disease spread, Jiao et al. [8] established an SEIRS (susceptible-exposed-infectious-recovered-susceptible) system with two delays and pulse vaccination

$$\begin{cases} \left.\begin{aligned} \frac{dS(t)}{dt} &= \mu(1-S(t)) - \beta S(t)I(t) + \gamma R(t), \\ \frac{dE(t)}{dt} &= \beta S(t)I(t) - \beta e^{-\mu\omega_1} S(t-\omega_1)I(t-\omega_1) \\ &\quad -\mu E(t), \\ \frac{dI(t)}{dt} &= \beta e^{-\mu\omega_1} S(t-\omega_1)I(t-\omega_1) \\ &\quad -\beta e^{-\mu(\omega_1+\omega_2)} S(t-\omega_1-\omega_2)I(t-\omega_1-\omega_2) \\ &\quad -\mu I(t), \\ \frac{dR(t)}{dt} &= \beta e^{-\mu(\omega_1+\omega_2)} S(t-\omega_1-\omega_2)I(t-\omega_1 \\ &\quad -\omega_2) - (\mu+\gamma)R(t), \end{aligned}\right\} t \neq n\tau, \\ \left.\begin{aligned} S(t^+) &= (1-\theta)S(t), \\ E(t^+) &= E(t), \\ I(t^+) &= I(t), \\ R(t^+) &= R(t) + \theta S(t), \end{aligned}\right\} t = n\tau, n = 1, 2, \cdots, \end{cases} \tag{1.1}$$

where $S(t), E(t), I(t), R(t)$ denote the numbers of susceptible, exposed, infectious, and recovered individuals, respectively. All coefficients are assumed to be positive constants. β is the average number of effective contacts of an infectious individual per unit time; the birth rate and the death rate μ are assumed to be the same constant for all hosts; ω_1 is the latent period of the disease and ω_2 is the length of the infectious period; the term $\beta e^{-\mu(\omega_1+\omega_2)} S(t-\omega_1-\omega_2)I(t-\omega_1-\omega_2)$ reflects the fact that the individuals who have recovered from infection are still alive after the infectious period ω_2; θ $(0<\theta<1)$ is the proportion of susceptibles that are successfully vaccinated, called the pulse vaccination rate; γ is the rate at which recovered hosts return to the susceptible class, so that the influx of susceptibles from the recovered hosts is $\gamma R(t)$; and τ is the interpulse time, i.e., the time between two consecutive pulse vaccinations. The initial conditions for (1.1) are

$$\begin{aligned} &(\varphi_1(\zeta), \varphi_2(\zeta), \varphi_3(\zeta), \varphi_4(\zeta)) \in C_+ \\ &= C([-(\omega_1+\omega_2), 0], R_+^4), \varphi_i(0) > 0, i = 1, 2, 3, 4. \end{aligned} \tag{1.2}$$

However, they did not consider the delay in acquiring immunity of the vaccinated susceptible population.

Motivated by the fact that physical protection and social isolation are critical to controlling the COVID-19 epidemic in China in the absence of vaccines or antiviral drugs, Jiao et al. [9] presented an SEIR (susceptible–exposed–infectious–recovered) model with infectivity in the incubation period and homestead-isolation of the susceptible.

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \beta(1-\theta_1)S(t)[I(t) + \theta_2 E(t)] - \mu S(t), \\ \frac{dE(t)}{dt} = \beta(1-\theta_1)S(t)[I(t) + \theta_2 E(t)] - (\delta+\mu)E(t), \\ \frac{dI(t)}{dt} = \delta E(t) - (\gamma+\sigma+\mu)I(t), \\ \frac{dR(t)}{dt} = (\gamma + \theta_3\sigma)I(t) - \mu R(t), \end{cases} \tag{1.3}$$

where $S(t)$ represents the number of the susceptible population at time t. $E(t)$ represents the number of the exposed population at time t. $I(t)$ represents the number of the infected population at time t. $R(t)$ represents the number of the recovered population at time t. $\Lambda > 0$ represents the enrolling rate. $\beta > 0$ represents the infective rate from S to E. $0 < \theta_1 < 1$ represents the homestead-isolation rate of the susceptible. $0 < \theta_2 < 1$ represents the infective effect of the exposed in incubation period. $\mu > 0$ represents the natural death rate. $\delta > 0$ represents the transition rate from E to I. $\gamma > 0$ represents the transition rate from I to R. $\sigma > 0$ represents hospitalized rate of I for the disease. $\theta_3 > 0$ represents the recurring rate of I, and $\delta > \theta_2(\gamma+\sigma+\mu)$.

They proved that the infection-free equilibrium is locally and globally asymptotically stable when the basic reproduction number satisfies $\mathcal{R}_0 < 1$, and that the positive equilibrium is locally and globally asymptotically stable when $\mathcal{R}_0 > 1$.

Furthermore, the fear effect, first modelled in predator–prey interactions by Wang et al. [10] as a reduction factor $\frac{1}{1+ky}$ on the prey birth rate, has been introduced into a stochastic delay predator–prey

model with diffusion for prey [11]

$$\begin{cases} dx_1(t) = [x_1(t)(\frac{r_1}{1+ky(t)} - a_{11}x_1(t) - a_{13}y(t)) \\ \quad +D(x_2(t) - x_1(t))]dt + \frac{\sigma_1 x_1(t)}{1+ky(t)}dW_1(t), \\ dx_2(t) = [x_2(t)(\frac{r_2}{1+ky(t)} - a_{22}x_1(t) - a_{23}y(t)) \\ \quad +D(x_1(t) - x_2(t))]dt + \frac{\sigma_2 x_2(t)}{1+ky(t)}dW_2(t), \\ dy(t) = [y(t)(-r_3 + a_{31}x_1(t-\tau) - a_{32}x_2(t-\tau) \\ \quad -a_{33}y(t))]dt + \sigma_3 y(t)dW_3(t), \end{cases} \tag{1.4}$$

where $x_i(t)$ denotes the density of the prey species in patch i $(i = 1, 2)$ at time t, $y(t)$ denotes the total predator population of both patches, r_i is the intrinsic growth rate of the prey in patch i, k is the level of fear, a_{ij} are interaction coefficients, D is the diffusion (dispersal) rate between the two patches, σ_i are the intensities of the white noises, $W_i(t)$ are independent standard Brownian motions, $\frac{r_i}{1+ky(t)}$ is the fear factor function, which represents the cost of anti-predator defense of the prey due to the fear induced by the predator, and τ is a delay caused by pregnancy. The system is subject to the initial condition

$$x_1(t) = \phi_1(t), x_2(t) = \phi_2(t),$$

$$y(t) = \phi_3(t), (\phi_1(t), \phi_2(t), \phi_3(t)) \in C([-\tau, 0], R^3).$$

Note that the symbols in the cited models (1.1), (1.3) and (1.4) are local to each model and should not be confused with those of system (2.1) below.

2 The model

Motivated by the above discussion, in particular by the fear effect used in [10, 11], we construct an SIR model with isolation and pulse vaccination of the susceptible population, taking into account the delay in acquiring immunity

$$\begin{cases} \left.\begin{array}{l} \frac{dS(t)}{dt} = \mu + \frac{\lambda_{\max}}{1+\delta I(t)} - \frac{\beta S(t)(1-\theta)I(t)}{1+\xi(1-\theta)I(t)} \\ \quad -\mu S(t), \\ \frac{dI(t)}{dt} = \frac{\beta S(t)(1-\theta)I(t)}{1+\xi(1-\theta)I(t)} - (\sigma+\mu)I(t), \\ \frac{dR(t)}{dt} = \sigma I(t) - \mu R(t), \end{array}\right\} t \in (n\tau, (n+1)\tau], \\ \left.\begin{array}{l} \Delta S(t) = -\mu_1 e^{-\mu l\tau} S(t - l\tau), \\ \Delta I(t) = 0, \\ \Delta R(t) = \mu_1 e^{-\mu l\tau} S(t - l\tau), \end{array}\right\} t = (n+1)\tau, \end{cases} \tag{2.5}$$

where $S(t)$ represents the number of the susceptible population at time t. $I(t)$ represents the number of the infected population at time t. $R(t)$ represents the number of the recovered population at time t. $\mu > 0$ represents the birth and death rate of the population (assumed equal). $\lambda_{\max} > 0$ represents the maximum enrolling amount from the exterior region for residents on $(n\tau, (n+1)\tau]$.

In addition, the enrolling amount of the susceptible population from the exterior region is described by the function $\frac{\lambda_{\max}}{1+\delta I(t)}$, which decreases as the fear intensity $\delta > 0$ and the density of the infective population increase, reflecting the reduced willingness of individuals to enter the region during an outbreak. $\beta > 0$ represents the infective rate from S to I on $(n\tau, (n+1)\tau]$. The saturated incidence rate $\frac{\beta(1-\theta)I(t)}{1+\xi(1-\theta)I(t)}$ is introduced into system (2.1), where $\xi > 0$ is the saturation (inhibition) coefficient, $\beta(1-\theta)I(t)$ measures the infection force of the disease and $\frac{1}{1+\xi(1-\theta)I(t)}$ measures the inhibition effect from the behavioral change of the susceptible individuals when the number of infective individuals increases or from the crowding effect of the infective individuals, and $0 < \theta < 1$ represents the isolation rate of the infectious population on $(n\tau, (n+1)\tau]$. $\sigma > 0$ represents the transition rate from I to R on $(n\tau, (n+1)\tau]$. $0 < \mu_1 < 1$ represents the vaccinated proportion of the susceptible population at time $t = (n-l)\tau$ $(0 < l < 1)$, and $l\tau$ is the delay for the vaccinated individuals to acquire immunity. During the time interval $((n+1-l)\tau, (n+1)\tau]$, a vaccinated individual survives natural death with probability $0 < e^{-\mu l\tau} < 1$, that is to say, at the end of this interval the proportion $\mu_1 S(t - l\tau)$ of vaccinated susceptibles that have survived and successfully acquired immunity is moved to R and becomes $\mu_1 e^{-\mu l\tau} S(t - l\tau)$. $\tau > 0$ represents the vaccinating period of the susceptible population.

Let $\phi(t) = (\phi_1(t), \phi_2(t), \phi_3(t))$ be a continuous function on $[-l\tau, 0]$. In this paper, we assume that all solutions of equation (2.1) satisfy the initial conditions as follows:

$$(S(t), I(t), R(t)) = (\phi_1(t), \phi_2(t), \phi_3(t)) > 0,$$

$$t \in [-l\tau, 0], (S(0^+), I(0^+), R(0^+)) = (S_0^+, I_0^+, R_0^+).$$

3 Main results

Since $R(t)$ does not appear in the first two equations of system (2.1), we only discuss the following subsystem

for $(S(t), I(t))$.

$$\left\{\begin{array}{l} \left.\begin{array}{l} \frac{dS(t)}{dt} = \mu + \frac{\lambda_{\max}}{1+\delta I(t)} - \frac{\beta S(t)(1-\theta)I(t)}{1+\xi(1-\theta)I(t)} \\ \quad -\mu S(t), \\ \frac{dI(t)}{dt} = \frac{\beta S(t)(1-\theta)I(t)}{1+\xi(1-\theta)I(t)} - (\sigma+\mu)I(t), \end{array}\right\} t \in (n\tau, (n+1)\tau], \\ \left.\begin{array}{l} \Delta S(t) = -\mu_1 e^{-\mu l\tau} S(t-l\tau), \\ \Delta I(t) = 0, \end{array}\right\} t = n\tau, \end{array}\right. \tag{3.6}$$

Lemma 3.1. For any $\varepsilon > 0$, each solution $(S(t), I(t), R(t))$ of system (2.1) satisfies $S(t) \leq \frac{\mu+\lambda_{\max}}{\mu} + \varepsilon$, $I(t) \leq \frac{\mu+\lambda_{\max}}{\mu} + \varepsilon$, $R(t) \leq \frac{\mu+\lambda_{\max}}{\mu} + \varepsilon$ for all t large enough.

Proof. For $\frac{d(S(t)+I(t)+R(t))}{dt} = \mu + \frac{\lambda_{\max}}{1+\delta I(t)} - \mu(S(t)+I(t)+R(t)) \leq \mu + \lambda_{\max} - \mu(S(t)+I(t)+R(t))$, and $\Delta(S(t)+I(t)+R(t)) = 0$, a standard comparison argument gives $\limsup_{t\to\infty}(S(t)+I(t)+R(t)) \leq \frac{\mu+\lambda_{\max}}{\mu}$, which completes the proof.

If $I(t) = 0$, we obtain the subsystem of system (3.1) as

$$\left\{\begin{array}{l} \frac{dS(t)}{dt} = \mu + \lambda_{\max} - \mu S(t), t \in (n\tau, (n+1)\tau] \\ \Delta S(t) = -\mu_1 e^{-\mu l\tau} S(t-l\tau), t = n\tau, \end{array}\right. \tag{3.7}$$

Integrating system (3.2) between pulse, we gain

$$\begin{array}{rl} S(t) = & \frac{\mu+\lambda_{\max}}{\mu} - (\frac{\mu+\lambda_{\max}}{\mu} - S(n\tau^+))e^{-\mu(t-n\tau)}, \\ & t \in (n\tau, (n+1)\tau]. \end{array} \tag{3.8}$$

Then,

$$\begin{array}{rl} S(t-l\tau) = & \frac{\mu+\lambda_{\max}}{\mu} - (\frac{\mu+\lambda_{\max}}{\mu} - S(n\tau^+))e^{-\mu(t-(n+l)\tau)}, \\ & t \in ((n+l)\tau, (n+1+l)\tau]. \end{array} \tag{3.9}$$

Considering the pulse vaccination effect, we can obtain the stroboscopic map of system (3.2).

$$\begin{array}{l} S((n+1)\tau^+) = \frac{\mu+\lambda_{\max}}{\mu}[1-\mu_1 e^{-\mu l\tau} - (1-\mu_1)e^{-\mu\tau}] \\ \quad +(1-\mu_1)e^{-\mu\tau} S(n\tau^+) \triangleq F(S(n\tau^+)). \end{array} \tag{3.10}$$

The unique fixed point of (3.5) is gotten as $P(S^*)$ with

$$S^* = \frac{(\mu+\lambda_{\max})[1-\mu_1 e^{-\mu l\tau} - (1-\mu_1)e^{-\mu\tau}]}{\mu[1-(1-\mu_1)e^{-\mu\tau}]} > 0. \tag{3.11}$$

Because $\frac{\partial F(S(n\tau^+))}{\partial S(n\tau^+)} = (1-\mu_1)e^{-\mu\tau} < 1$, we obtain the following two lemmas, similar to those in [4].

Lemma 3.2. The unique positive fixed point $P(S^*)$ of system (3.5) is globally asymptotically stable.

Lemma 3.3. The periodic solution $\widetilde{S(t)}$ of system (3.1) is globally asymptotically stable, where $\widetilde{S(t)}$ is defined as

$$\begin{array}{rl} \widetilde{S(t)} = & \frac{\mu+\lambda_{\max}}{\mu} - (\frac{\mu+\lambda_{\max}}{\mu} - S^*)e^{-\mu(t-n\tau)}, \\ & t \in (n\tau, (n+1)\tau]. \end{array} \tag{3.12}$$

where S^* is determined by (3.6).

For convenience, define

$$M = \frac{\mu+\lambda_{\max}}{\mu},$$

and

$$\begin{array}{rl} H = & \frac{\beta(1-\theta)(\mu+\lambda_{\max})}{\mu}\tau \\ & -\frac{\beta(1-\theta)(\frac{\mu+\lambda_{\max}}{\mu}-S^*)(1-e^{-\mu\tau})}{\mu} - (\sigma+\mu)\tau. \end{array} \tag{3.13}$$

Theorem 3.1. If $H < 0$ holds, the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) is globally asymptotically stable.

Proof. Defining $s(t) = S(t) - \widetilde{S(t)}, i(t) = I(t)$, we obtain the following linearized system of system (3.1) about the periodic solution $(\widetilde{S(t)}, 0)$

$$\begin{pmatrix} \frac{ds(t)}{dt} \\ \frac{di(t)}{dt} \end{pmatrix} = \begin{pmatrix} -\mu & -\delta\lambda_{\max} - \beta(1-\theta)\widetilde{S(t)} \\ 0 & \beta(1-\theta)\widetilde{S(t)} - (\sigma+\mu) \end{pmatrix} \begin{pmatrix} s(t) \\ i(t) \end{pmatrix}.$$

It is easy to obtain the fundamental solution matrix

$$\Phi(t) = \begin{pmatrix} e^{-\mu t} & *_1 \\ 0 & e^{\int_0^t(\beta(1-\theta)\widetilde{S(s)} - (\sigma+\mu))ds} \end{pmatrix}.$$

There is no need to calculate the exact form of $*_1$ as it is not required in the analysis that follows. The linearization of the impulsive conditions (the third and fourth equations) of system (3.1) is

$$s(n\tau^+) = s(n\tau) - \mu_1 e^{-\mu l\tau} s(n\tau - l\tau), \qquad i(n\tau^+) = i(n\tau).$$

Restricting to the s-component (i.e., $i = 0$), we have $s(t) = s((n-1)\tau^+)e^{-\mu(t-(n-1)\tau)}$ on $((n-1)\tau, n\tau]$, hence $s(n\tau^+) = (1-\mu_1)e^{-\mu\tau} s((n-1)\tau^+)$. Therefore, the stability of the infection-free periodic solution $(\widetilde{S(t)}, 0)$ is determined by the Floquet multipliers $\lambda_1 = (1-\mu_1)e^{-\mu\tau} < 1$, and $\lambda_2 = e^{\int_0^\tau(\beta(1-\theta)\widetilde{S(s)} - (\sigma+\mu))ds} = e^H$.

According to condition $H < 0$, we have $\mid \lambda_2 \mid < 1$, then $(\widetilde{S(t)}, 0)$ is locally stable.

Choose an $\varepsilon > 0$ small enough such that $\rho = \exp(\int_0^\tau (\beta(1-\theta)(\widetilde{S(t)}+\varepsilon) - (\sigma+\mu))dt) < 1$. From the first equation of system (3.1), we notice that $\frac{dS(t)}{dt} \leq \mu + \lambda_{\max} - \mu S(t)$, then

$$\begin{cases} \dfrac{dS(t)}{dt} \leq \mu + \lambda_{\max} - \mu S(t), t \neq n\tau, \\ \Delta S(t) = -\mu_1 e^{-\mu l\tau} S(t-l\tau), t = n\tau. \end{cases} \tag{3.14}$$

and its comparative impulsive differential equation is

$$\begin{cases} \dfrac{dS_1(t)}{dt} = \mu + \lambda_{\max} - \mu S_1(t), t \neq n\tau, \\ \Delta S_1(t) = -\mu_1 e^{-\mu l\tau} S_1(t-l\tau), t = n\tau. \end{cases} \tag{3.15}$$

From Lemma 3.3 and the comparison theorem of impulsive equations [12], we have $S(t) \leq S_1(t)$ and $S_1(t) \to \widetilde{S_1(t)} = \widetilde{S(t)}$ as $t \to \infty$. Then

$$S(t) \leq S_1(t) \leq \widetilde{S(t)} + \varepsilon, \tag{3.16}$$

for all t large enough. For convenience, we may assume (3.11) holds for all $t \geq 0$. From system (3.1) and (3.11), we get

$$\begin{cases} \dfrac{dI(t)}{dt} \leq [\beta(1-\theta)(\widetilde{S(t)}+\varepsilon) - (\sigma+\mu)]I(t), t \neq n\tau, \\ I(n\tau^+) = I(n\tau), t = n\tau. \end{cases} \tag{3.17}$$

So $I((n+1)\tau) \leq I(n\tau^+) e^{\int_{n\tau}^{(n+1)\tau} [\beta(1-\theta)(\widetilde{S(t)}+\varepsilon) - (\sigma+\mu)]dt}$. Hence, $I(n\tau) \leq I(0^+)\rho^n$ and $I(n\tau) \to 0$ as $n \to \infty$. Therefore, $I(t) \to 0$ as $t \to \infty$.

Next, we prove that $S(t) \to \widetilde{S(t)}$ as $t \to \infty$. For $\varepsilon > 0$ small enough, there must exist a $t_0 > 0$ such that $0 < I(t) < \varepsilon$ for all $t \geq t_0$, without loss of generality, we may assume that $0 < I(t) < \varepsilon$ for all $t \geq 0$. Then, we have

$$\begin{aligned} &\mu + \frac{\lambda_{\max}}{1+\delta\varepsilon} - (\mu + \beta(1-\theta)\varepsilon)S(t) \\ &\leq \tfrac{dS(t)}{dt} \\ &\leq \mu + \lambda_{\max} - \mu S(t). \end{aligned} \tag{3.18}$$

Then, we have $z_1(t) \leq S(t) \leq z_2(t)$ and $z_1(t) \to \widetilde{z_1(t)}, z_2(t) \to \widetilde{S(t)}$ as $t \to \infty$, where $z_1(t)$ and $z_2(t)$ are the solutions of

$$\begin{cases} \dfrac{dz_1(t)}{dt} = \mu + \dfrac{\lambda_{\max}}{1+\delta\varepsilon} - (\mu + \beta(1-\theta)\varepsilon)z_1(t), t \neq n\tau, \\ \Delta z_1(t) = -\mu_1 e^{-\mu l\tau} z_1(t-l\tau), t = n\tau, \\ z_1(0^+) = S(0^+), \end{cases} \tag{3.19}$$

and

$$\begin{cases} \dfrac{dz_2(t)}{dt} = \mu + \lambda_{\max} - \mu z_2(t), t \neq n\tau, \\ \Delta z_2(t) = -\mu_1 e^{-\mu l\tau} z_2(t-l\tau), t = n\tau, \\ z_2(0^+) = S(0^+), \end{cases} \tag{3.20}$$

respectively. Let $a_\varepsilon = \mu + \frac{\lambda_{\max}}{1+\delta\varepsilon}$ and $\kappa_\varepsilon = \mu + \beta(1-\theta)\varepsilon$. For $n\tau < t \leq (n+1)\tau$, the globally asymptotically stable periodic solution of the first system is $\widetilde{z_1(t)} = \frac{a_\varepsilon}{\kappa_\varepsilon} - \left(\frac{a_\varepsilon}{\kappa_\varepsilon} - z_1^*\right) e^{-\kappa_\varepsilon(t-n\tau)}$ with $z_1^* = \frac{a_\varepsilon\left[1-e^{-\kappa_\varepsilon\tau}-\mu_1 e^{-\mu l\tau}\left(1-e^{-\kappa_\varepsilon(1-l)\tau}\right)\right]}{\kappa_\varepsilon\left[1-e^{-\kappa_\varepsilon\tau}+\mu_1 e^{-\mu l\tau}e^{-\kappa_\varepsilon(1-l)\tau}\right]}$, while the periodic solution of the second system is $\widetilde{S(t)}$ (the second system coincides with (3.10)). Therefore, for any $\varepsilon_1 > 0$, there exists $t_1 > 0$ such that, for all $t > t_1$,

$$\widetilde{z_1(t)} - \varepsilon_1 < S(t) < \widetilde{S(t)} + \varepsilon_1$$

Letting $\varepsilon \to 0$ (so that $\kappa_\varepsilon \to \mu$, $a_\varepsilon \to \mu + \lambda_{\max}$ and $\widetilde{z_1(t)} \to \widetilde{S(t)}$), we have $\widetilde{S(t)} - \varepsilon_1 < S(t) < \widetilde{S(t)} + \varepsilon_1$ for t large enough, which implies $S(t) \to \widetilde{S(t)}$ as $t \to \infty$. This completes the proof.

Theorem 3.2. If $H > 0$ holds, then the disease in system (3.1) tends to be endemic, i.e., the infective population persists.

Proof. From Lemma 3.1, we have proved that

$$S(t) \leq \frac{\mu + \lambda_{\max}}{\mu}, \qquad I(t) \leq \frac{\mu + \lambda_{\max}}{\mu}$$

for all sufficiently large t. Hence, without loss of generality, we may assume that these bounds hold for all $t \geq 0$ (up to an arbitrarily small $\varepsilon > 0$, cf. Lemma 3.1).

From system (3.1), we have

$$I(t) > I(0^+)e^{-(\sigma+\mu)t}$$

for all sufficiently large t. Thus, it suffices to find $m_1 > 0$ such that $I(t) \geq m_1$ for all sufficiently large t. Suppose, for contradiction, that no such m_1 exists;

then for any $m_2 > 0$ sufficiently small, $I(t) < m_2$ holds for all $t \geq 0$.

Let

$$\kappa_2 = \frac{\beta(1-\theta)m_2}{1+\xi(1-\theta)m_2} + \mu. \tag{3.21}$$

By the condition $H > 0$, we can choose $m_2 > 0$ and $\varepsilon_3 > 0$ sufficiently small such that

$$\begin{aligned}\rho_1 = &\frac{\beta(1-\theta)\left(\mu + \dfrac{\lambda_{\max}}{1+\delta m_2}\right)}{\kappa_2\left[1+\xi(1-\theta)m_2\right]}\tau \\ &- \frac{\beta(1-\theta)\left(\dfrac{\mu + \dfrac{\lambda_{\max}}{1+\delta m_2}}{\kappa_2} - S_2^*\right)(1-e^{-\kappa_2\tau})}{\kappa_2\left[1+\xi(1-\theta)m_2\right]} \\ &- \frac{\beta(1-\theta)\varepsilon_3}{1+\xi(1-\theta)m_2}\tau - (\sigma+\mu)\tau > 0.\end{aligned} \tag{3.22}$$

where S_2^* is defined in (3.25). Then,

$$\begin{cases} \dfrac{dS(t)}{dt} \geq \mu + \dfrac{\lambda_{\max}}{1+\delta m_2} - [\dfrac{\beta(1-\theta)m_2}{1+\xi(1-\theta)m_2} + \mu]S(t), \\ \qquad t \neq n\tau, \\ \Delta S(t) = -\mu_1 e^{-\mu l\tau} S(t-l\tau), t = n\tau. \end{cases} \tag{3.23}$$

Similar to Lemmas 3.2 and 3.3 (and using the comparison theorem [12]), we have $S(t) \geq S_2(t)$, and $S_2(t) \to \overline{S_2(t)}, t \to \infty$, where $S_2(t)$ is the solution of

$$\begin{cases} \dfrac{dS_2(t)}{dt} = \mu + \dfrac{\lambda_{\max}}{1+\delta m_2} - [\dfrac{\beta(1-\theta)m_2}{1+\xi(1-\theta)m_2} + \mu]S_2(t), \\ \qquad t \neq n\tau, \\ \Delta S_2(t) = -\mu_1 e^{-\mu l\tau} S_2(t-l\tau), t = n\tau. \end{cases} \tag{3.24}$$

Since the slope $e^{-\kappa_2\tau}\left(1-\mu_1 e^{(\kappa_2-\mu)l\tau}\right)$ of the corresponding stroboscopic map lies in $(0,1)$ for m_2 small enough, its unique periodic solution $\overline{S_2(t)}$ is globally asymptotically stable (as in Lemmas 3.2 and 3.3), where $\overline{S_2(t)} = \frac{\mu+\frac{\lambda_{\max}}{1+\delta m_2}}{\kappa_2} - \left(\frac{\mu+\frac{\lambda_{\max}}{1+\delta m_2}}{\kappa_2} - S_2^*\right)e^{-\kappa_2(t-n\tau)}$ for $t \in (n\tau, (n+1)\tau]$, with κ_2 given in (3.21) and

$$S_2^* = \frac{\left(\mu+\dfrac{\lambda_{\max}}{1+\delta m_2}\right)\left[1-e^{-\kappa_2\tau}-\mu_1 e^{-\mu l\tau}\left(1-e^{-\kappa_2(1-l)\tau}\right)\right]}{\kappa_2\left[1-e^{-\kappa_2\tau}+\mu_1 e^{-\mu l\tau}e^{-\kappa_2(1-l)\tau}\right]}. \tag{3.25}$$

Therefore, for any $\varepsilon_3 > 0$ there exists $T_1 > 0$ such that

$$S(t) \geq S_2(t) \geq \overline{S_2(t)} - \varepsilon_3.$$

Then,

$$\frac{dI(t)}{dt} \geq [\frac{\beta(1-\theta)(\overline{S_2(t)}-\varepsilon_3)}{1+\xi(1-\theta)m_2} - (\sigma+\mu)]I(t), \tag{3.26}$$

for $t \geq T_1$. Let $N_1 \in \mathbb{N}$ and $N_1\tau > T_1$, integrating (3.26) on $(n\tau, (n+1)\tau], n \geq N_1$, we have

$$\begin{aligned} &I((n+1)\tau) \\ &\geq I(n\tau^+)\exp\left\{\int_{n\tau}^{(n+1)\tau}\left[\frac{\beta(1-\theta)(\overline{S_2(t)}-\varepsilon_3)}{1+\xi(1-\theta)m_2} - (\sigma+\mu)\right]dt\right\} \\ &= I(n\tau^+)e^{\rho_1}. \end{aligned}$$

then $I((N_1+k)\tau) \geq I(N_1\tau^+)e^{k\rho_1} \to \infty$, as $k \to \infty$, which contradicts the boundedness of $I(t)$. Hence $I(t) < m_2$ cannot hold for all $t \geq 0$, i.e., there exists a $t_1 > 0$ such that $I(t_1) \geq m_2$.

From the first equation of system (3.1), we can have $\frac{dS(t)}{dt} \geq \mu + \frac{\lambda_{\max}}{1+\delta M} - (\frac{\beta(1-\theta)M}{1+\xi(1-\theta)M} + \mu)S(t)$. Then,

$$\begin{cases} \dfrac{dS(t)}{dt} \geq \mu + \dfrac{\lambda_{\max}}{1+\delta M} - (\dfrac{\beta(1-\theta)M}{1+\xi(1-\theta)M} + \mu)S(t), \\ \qquad t \neq n\tau, \\ \Delta S(t) = -\mu_1 e^{-\mu l\tau} S(t-l\tau),\ t = n\tau. \end{cases} \tag{3.27}$$

By Lemmas 3.2, 3.3 and the comparison theorem [12], we obtain $S(t) \geq S_3(t)$ and $S_3(t) \to \widetilde{S_3(t)},\ t \to \infty$, and $\widetilde{S_3(t)}$ is the periodic solution of

$$\begin{cases} \dfrac{dS_3(t)}{dt} = \mu + \dfrac{\lambda_{\max}}{1+\delta M} - [\dfrac{\beta(1-\theta)M}{1+\xi(1-\theta)M} + \mu]S_3(t), \\ \qquad t \neq n\tau, \\ \Delta S_3(t) = -\mu_1 e^{-\mu l\tau} S_3(t-l\tau),\ t = n\tau, \end{cases} \tag{3.28}$$

where

$$\kappa_3 = \frac{\beta(1-\theta)M}{1+\xi(1-\theta)M} + \mu. \tag{3.29}$$

Then

$$\begin{aligned}\widetilde{S_3(t)} = &\frac{\mu + \dfrac{\lambda_{\max}}{1+\delta M}}{\kappa_3} \\ &- \left(\frac{\mu + \dfrac{\lambda_{\max}}{1+\delta M}}{\kappa_3} - S_3^*\right)e^{-\kappa_3(t-n\tau)},\end{aligned} \tag{3.30}$$

for $t \in (n\tau, (n+1)\tau)$, is globally asymptotically stable, where

$$S_3^* = \frac{\left(\mu + \dfrac{\lambda_{\max}}{1+\delta M}\right)\left[1-e^{-\kappa_3\tau}-\mu_1 e^{-\mu l\tau}\left(1-e^{-\kappa_3(1-l)\tau}\right)\right]}{\kappa_3\left[1-e^{-\kappa_3\tau}+\mu_1 e^{-\mu l\tau}e^{-\kappa_3(1-l)\tau}\right]}. \tag{3.31}$$

For any sufficiently small $\varepsilon_4 > 0$, we obtain

$$S_3(t) > \widetilde{S_3(t)} - \varepsilon_4. \tag{3.32}$$

From the comparison theorem for impulsive differential equations [12], we have

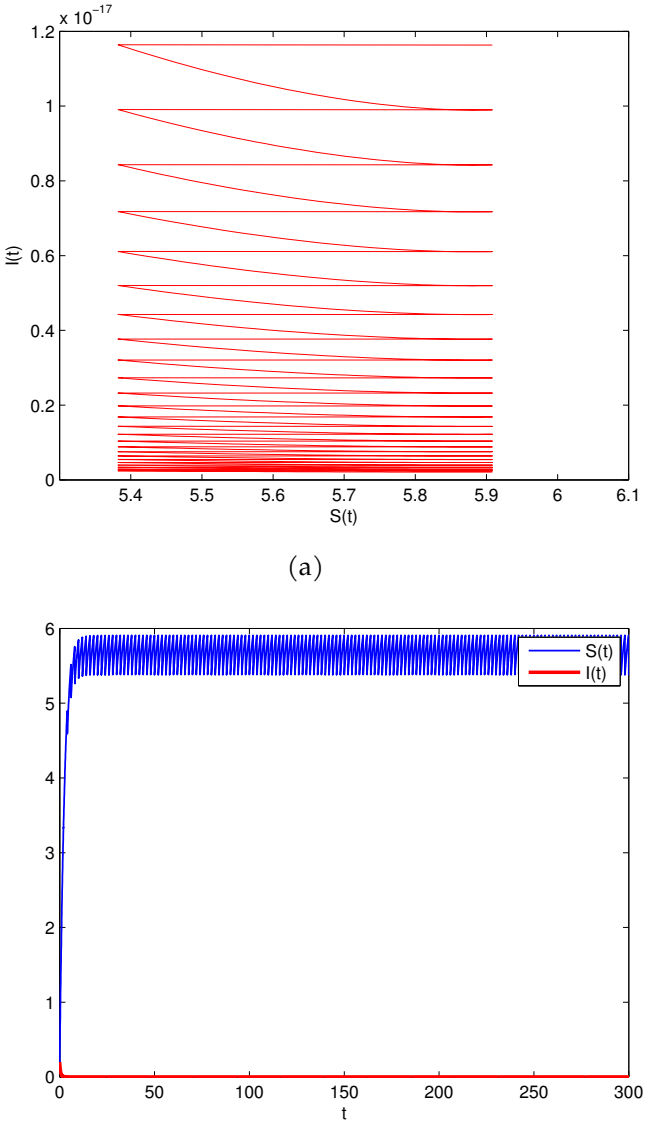
$$S(t) > S_3(t) > \widetilde{S_3(t)} - \varepsilon_4 \geq S_3^* - \varepsilon_4 =: m_3, \tag{3.33}$$

where we used that $\widetilde{S_3(t)}$ increases from S_3^* (its value right after a pulse) on each interval, and $0 < \varepsilon_4 < S_3^*$. Therefore, $S(t) > m_3 > 0$. This completes the proof.

4 Numerical simulations

First, take the parameter values given in the caption of Figure 1 ($\theta = 0.763,\ l = 0.01$); then $H = -0.213 < 0$, and the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) is globally asymptotically stable (see Figure 1). Next, let $l = 0.7$ with all other parameters unchanged; then $H = 0.0407 > 0$, and system (3.1) tends to be endemic (see Figure 2). From the numerical analysis of Figures 1 and 2, we conjecture that there is a threshold l^*: when $l < l^*$, the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) is globally asymptotically stable, and when $l > l^*$, system (3.1) tends to be endemic. This indicates that the delay in acquiring immunity of the pulse-vaccinated susceptible population plays an important role in system (2.1).

Finally, let $\theta = 0.8$ and $l = 0.7$ with all other parameters unchanged; then $H = -0.7056 < 0$, and the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) is globally asymptotically stable (see Figure 3). From the numerical analysis of Figures 2 and 3, we conjecture that there is a threshold θ^*: when $\theta > \theta^*$, the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) is globally asymptotically stable, and when $\theta < \theta^*$, system (3.1) tends to be endemic. This also indicates that isolation plays an important role in system (2.1).



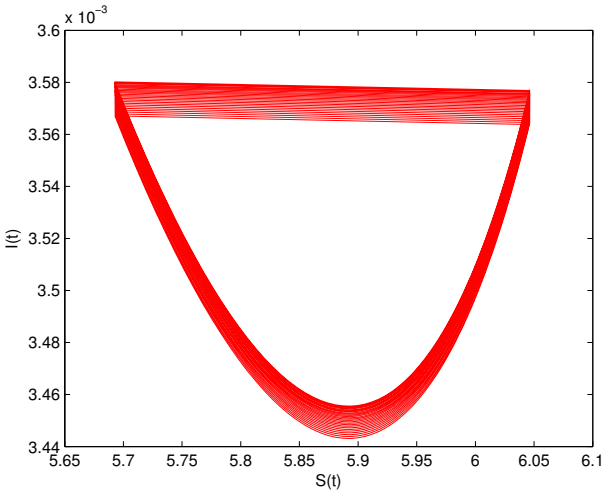
(a)

(b)

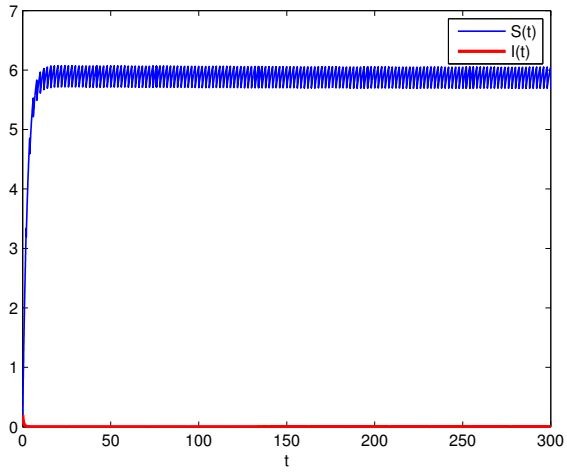
Figure 1. Globally asymptotically stable infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) for $\theta = 0.763$ and $l = 0.01$ ($H = -0.213 < 0$), with $S(0^+) = 0.2,\ I(0^+) = 0.2,\ \mu = 0.58,\ \mu_1 = 0.1,\ \lambda_{\max} = 2,\ \delta = 0.5,\ \beta = 1,\ \xi = 2,\ \sigma = 1.5,\ \tau = 2$. (a) Phase diagram of $S(t)$ and $I(t)$; (b) time series of $S(t)$ and $I(t)$.

5 Conclusion

In this work, we have presented an SIR model with isolation and pulse vaccination of the susceptible population, taking into account the delay in acquiring immunity. The expression of H in (3.13) shows that isolation (θ) and the immunity delay ($l\tau$) play important roles in achieving the infection-free state of system (3.1), and that the maximum enrolling amount $\lambda_{\max}$ from the exterior region also affects disease elimination. If $H < 0$ holds, we prove that the infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system

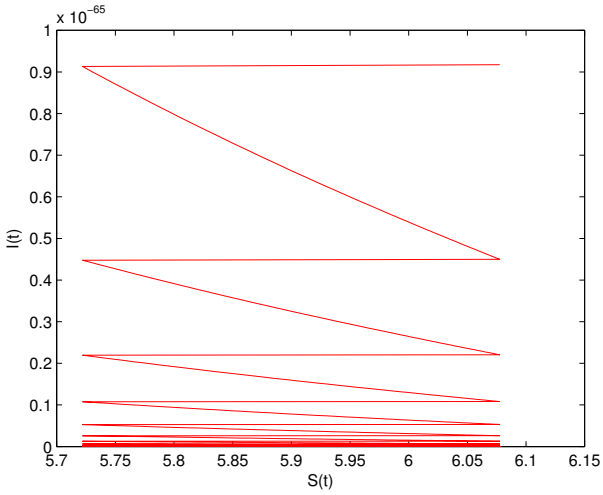


(a)

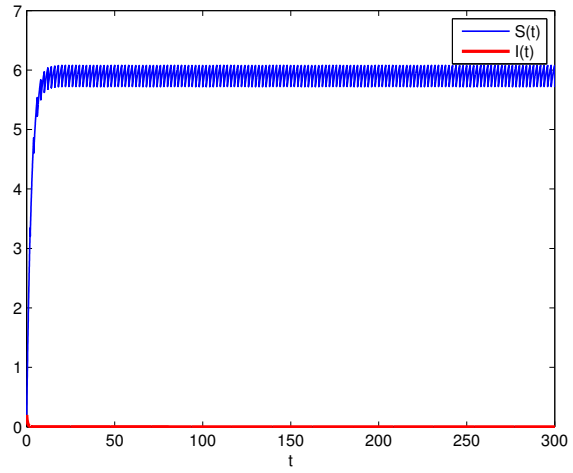


(b)

Figure 2. Endemic dynamics of system (3.1) for $\theta = 0.763$ and $l = 0.7$ $(H = 0.0407 > 0)$, with $S(0^+) = 0.2,\ I(0^+) = 0.2,\ \mu = 0.58,\ \mu_1 = 0.1,\ \lambda_{\max} = 2,\ \delta = 0.5,\ \beta = 1,\ \xi = 2,\ \sigma = 1.5,\ \tau = 2$. (a) Phase diagram of $S(t)$ and $I(t)$; (b) time series of $S(t)$ and $I(t)$.



(a)



(b)

Figure 3. Infection-free periodic solution $(\widetilde{S(t)}, 0)$ of system (3.1) for $\theta = 0.8$ and $l = 0.7$ $(H = -0.7056 < 0)$, with $S(0^+) = 0.2,\ I(0^+) = 0.2,\ \mu = 0.58,\ \mu_1 = 0.1,\ \lambda_{\max} = 2,\ \delta = 0.5,\ \beta = 1,\ \xi = 2,\ \sigma = 1.5,\ \tau = 2$. (a) Phase diagram of $S(t)$ and $I(t)$; (b) time series of $S(t)$ and $I(t)$.

(3.1) is globally asymptotically stable. If $H > 0$ holds, we also prove that the disease in system (3.1) tends to be endemic. The numerical examples further suggest thresholds l^* and θ^* (see Section 4), whose existence and explicit characterization remain to be established analytically.

Data Availability Statement

Data will be made available on request.

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Conflicts of Interest

Jianjun Jiao served as an Editor-in-Chief of the *Journal of Mathematics and Interdisciplinary Applications* at the time of manuscript submission. To ensure the integrity of the peer-review process, Jianjun Jiao was not involved in the editorial handling, peer review, or decision-making process for this manuscript, which was handled independently by another editor. The

remaining authors declare no conflicts of interest.

AI Use Statement

The authors declare that no generative AI was used in the preparation of this manuscript.

Ethical Approval and Consent to Participate

Not applicable.

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