Available online at www.sciencedirect.com
SCIENCE DIRECT®
Nonlinear Analysis: Real World Applications 6 (2005) 495–507
Nonlinear Analysis
Real World Applications
www.elsevier.com/locate/na

# Global properties of a delayed SIR model with temporary immunity and nonlinear incidence rate

Yuliya N. Kyrychko[a,*], Konstantin B. Blyuss[b]

[a] *Department of Engineering Mathematics, University of Bristol, Queen's Building, University Walk, Bristol BS8 1TR, UK*
[b] *Department of Mathematical Sciences, University of Exeter, Laver Building, North Park Road, Exeter EX4 4QE, UK*

Received 18 March 2004; accepted 27 October 2004

**Abstract**

We derive and study a time-delayed SIR model with a general incidence rate. The time delay represents temporary immunity period, i.e. time from recovery to becoming susceptible again. Both trivial and endemic equilibria are found, and their stability is investigated. Using Lyapunov functional approach, the global stability of an endemic equilibrium is shown. Numerical simulations support our analytical conclusions and illustrate possible behaviour scenarios of the model.


*Keywords:* SIR model; Temporal delay; Equilibria; Global stability; Numerical simulations

## 1. Introduction

In recent years there has been made a significant progress in understanding different scenarios for disease transmissions and behaviour of epidemics. Many models in the literature represent dynamics of diseases by systems of ordinary differential equations without delay. However, inclusion of temporal delays in such models makes them more realistic by allowing to describe the effects of disease latency or immunity [1–4].

One of the main issues in the study of behaviour of epidemics is the analysis of steady states of the model and their stability. Generally, a model contains a disease-free equilibrium

* Corresponding author. Tel.: +44 117 33 17369; fax: +44 117 925 1154.
*E-mail address:* Y.Kyrychko@bristol.ac.uk (Y.N. Kyrychko).

1468-1218/$ - see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.nonrwa.2004.10.001

and one or more endemic equilibria. The stability of a disease-free steady state as well as the existence of other non-trivial equilibria can be determined using the so-called basic reproduction ratio, which quantifies how many secondary infections appear from a single infected put in a population of susceptibles [5]. When the basic reproduction number is less that unity, the disease-free equilibrium is locally asymptotically stable, and, therefore, the disease dies out after some period of time. Similarly, when the endemic equilibrium is a global attractor, epidemiologically this means that the disease will prevail and persist in a population.

It has been suggested by several authors that the disease transmission process may have a nonlinear incidence rate. This allows one to include behavioural changes and prevent unbounded contact rates [8–10]. A particular example of such an incidence rate is given by $\alpha I^s/(1+\beta I^k)$, with $s, k, \alpha, \beta > 0$. Another type of a nonlinear incidence rate, $\alpha I^s S^k$, with $\alpha, k, s > 0$ or $k, s$ near 1, represents saturation or multiple exposures before infection.

In this paper we derive a model which includes a general nonlinear incidence rate and a temporary immunity from a disease. This means, that after recovery an individual has a temporary immunity against a disease, and, therefore, it moves into the susceptible class after some period of time. This can be observed in the case of influenza, when after recovery there is a long (but not lifelong) immunity to the same strain of the disease but no immunity against other strains. Other cases of temporary immunity include *Chlamydia trachomatis* with very short temporal immunity and very high rates of reinfection; *Salmonella* infection with partial immunity; non-plague yersiniosis where the actual time of the immunity is unclear; respiratory syncytial virus after which immunity is incomplete and short-lived.

The temporary immunity is incorporated in our model by introducing the term $I(t-\tau)\mathrm{e}^{-\mu\tau}$, where $\tau$ is the length of immunity period. This term reflects the fact that an individual has survived from natural death in a recovery pool before becoming susceptible again. We analyse existence and linear stability of the infection-free and endemic equilibria. Using the basic reproduction ratio $\mathscr{R}_0$ we deduce that when the infection-free steady state is linearly asymptotically stable the model has no other equilibria. Moreover, we prove that under the condition that $\mathscr{R}_0 < 1$ the infection-free equilibrium is globally asymptotically stable, which means that after some period of time the disease will die out. After the infection-free equilibrium becomes unstable there appears a non-trivial steady state. We study linear stability of this state and also prove its global stability assuming incidence rate to be linear. Furthermore, numerical simulations are carried out to illustrate possible behaviour of solutions for different values of the immunity time $\tau$. In particular, for some values of $\tau$ a sustainable oscillatory dynamics can be observed.

## 2. Derivation of the model

A delayed SIR model which incorporates temporary immunity and a general nonlinear incidence rate has the following form:

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu - \mu S - \varphi f(I(t))S(t) + \gamma I(t-\tau)\mathrm{e}^{-\mu\tau},$$

$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} = \varphi f(I(t))S(t) - (\mu+\gamma)I(t),$$

$$\frac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma I(t) - \gamma I(t-\tau)\mathrm{e}^{-\mu\tau} - \mu R(t), \tag{1}$$

where it is assumed that

(i) there is a nonlinear incidence rate which is governed by the function $f(I)$;
(ii) there is a temporary immunity period of the fixed length $\tau$, after which recovered infectives revert to the susceptible class;
(iii) there are no disease-caused deaths.

The function $f(I)$ is assumed to have the following properties [10]:

- $f(0) = 0$,
- $f'(I) > 0$,
- $f''(I) < 0$,
- $\lim_{I\to\infty} f(I) = c < \infty$.

Parameters in the system are as follows: $\mu$ is a natural death rate; $\gamma$ is a recovery rate, i.e. rate with which individuals move from the infected class to the recovered, and $\varphi$ is a recruitment rate from susceptibles to the infected class. Under the assumption that birth and death rates are the same, the total population $N(t)$ evolves according to $\mathrm{d}N/\mathrm{d}t = \mu(1 - N(t))$, and $N(t) \to 1$ as $t \to \infty$. The first two equations in system (1) do not depend on the third equation, and therefore this equation can be omitted without loss of generality. Hence, system (1) can be rewritten as

$$\begin{aligned}\frac{\mathrm{d}S(t)}{\mathrm{d}t} &= \mu - \mu S - \varphi f(I(t))S(t) + \gamma I(t-\tau)\mathrm{e}^{-\mu\tau},\\ \frac{\mathrm{d}I(t)}{\mathrm{d}t} &= \varphi f(I(t))S(t) - (\mu+\gamma)I(t).\end{aligned} \tag{2}$$

## 3. Positivity of solutions

Model (1) describes a human population, and, therefore, it is very important to prove that all quantities (susceptibles, infectives and recovered) will be positive for all time. In other words, we want to prove that all solutions of system (2) with positive initial data will remain positive for all times $t > 0$. The idea of the proof was introduced by Li and Kuang [7].

**Theorem 1.** *Let the initial data be* $S(0) = S_0 > 0$ *and* $I(s) = I_0(s) \geqslant 0$ *for all* $s \in [-\tau; 0)$ *with* $I_0(0) > 0$. *Then solutions* $S(t)$ *and* $I(t)$ *of system* (2) *are positive for all* $t > 0$.

**Proof.** To see this, we assume for contradiction that there exists the first time $t_1$ such that $I(t_1)S(t_1)=0$. Assume that $I(t_1)=0$. Then $S(t) \geqslant 0$ for all $t \in [0; t_1]$. Noticing that $f(0)=0$

and the quantity $f(I(t))$ always has a factor of $I(t)$ in it, one can define

$$A = \min_{0 \leqslant t \leqslant t_1} \left\{ \varphi \frac{f(I(t))S(t)}{I(t)} - \mu - \gamma \right\}.$$

Then, for $t \in [0; t_1]$, $\mathrm{d}I(t)/\mathrm{d}t \geqslant AI(t)$. Therefore, $I(t_1) \geqslant I(0)\mathrm{e}^{At_1} > 0$, which is a contradiction. Thus, $I(t) > 0$ for all $t > 0$. By the same argument it can be proved that $S(t)$ is positive. Suppose not. Let $t_1$ be the first time when again $S(t)I(t) = 0$. Assume that $S(t_1) = 0$. Then $I(t) \geqslant 0$ for all $t \in [0; t_1]$. Then, from the first equation of system (2) we have

$$\left.\frac{\mathrm{d}S(t)}{\mathrm{d}t}\right|_{t=t_1} = \underbrace{\mu}_{>0} - \underbrace{\mu S(t_1)}_{=0} - \underbrace{\varphi f(I(t_1))S(t_1)}_{=0} + \underbrace{\gamma I(t_1 - \tau)\mathrm{e}^{-\mu\tau}}_{\geqslant 0} > 0.$$

Since $S(0) > 0$, for $S(t_1) = 0$ we must have $\mathrm{d}S(t)/\mathrm{d}t|_{t=t_1} \leqslant 0$, which is a contradiction.

Next, we show the positivity of $R(t)$. Equation for $R(t)$ from system (1) can be readily solved to give

$$R(t) = \gamma \int_{t-\tau}^{t} \mathrm{e}^{-\mu(t-s)} I(s)\,\mathrm{d}s.$$

Since it was established that $I(t)$ is positive for all $t > 0$, therefore, $R(t)$ is also positive for all $t > 0$. This completes the proof. □

In this section we have proved that all solutions of system (2) will remain positive for all time, i.e. $S(t) > 0$, $I(t) > 0$ and $R(t) > 0$ for all $t > 0$.

## 4. Infection-free steady state and its stability

Now we analyse system (2) by finding its equilibria and studying their linear stability. Steady states of system (2) satisfy the following system of equations:

$$\begin{cases} \mu - \mu S - \varphi f(I)S + \gamma I\mathrm{e}^{-\mu\tau} = 0, \\ \varphi f(I)S - (\mu + \gamma)I = 0. \end{cases} \tag{3}$$

It is easy to check that system (3) has the disease-free equilibrium $E_0 = (1, 0)$ and one more non-zero steady state for certain parameter values. We start with analysing the behaviour of the original system (2) near $E_0$. The eigenvalues of the linearisation of system (2) near the steady state $E_0$ are $\lambda_1 = -\mu$ and $\lambda_2 = \varphi f'(0) - \mu - \gamma$. All parameters of the model are assumed to be positive and from the properties of the function $f(I)$ it follows that $f'(I) > 0$. Therefore, for $\lambda_1$, $\lambda_2$ to be negative, i.e. for a disease-free equilibrium to be locally asymptotically stable, the following condition has to be required:

$$\varphi f'(0) < \mu + \gamma. \tag{4}$$

As long as condition (4) holds, the disease-free steady state of system (2) stays locally asymptotically stable and no other equilibrium is feasible, as we will show later. Let us

define the *basic reproduction number* of the infection as

$$\mathscr{R}_0 = \frac{\varphi f'(0)}{\mu + \gamma}.$$

Using $\mathscr{R}_0$ we can state the following lemma indicating the stability of $E_0 = (1, 0)$.

**Lemma 1.** *The uninfected equilibrium $E_0$ is locally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$.*

## 5. Global stability of the trivial steady state

We have seen that the equilibrium point $(1, 0)$ is unstable when $\mathscr{R}_0 > 1$. In this section we shall prove the global stability of this steady state under the condition $\mathscr{R}_0 < 1$. Returning to system (2) we make the following transformation:

$$\hat{S} = 1 - S, \quad \text{and} \quad \hat{I} = I.$$

With this transformation, system (2) becomes

$$\begin{aligned} \frac{\mathrm{d}\hat{S}(t)}{\mathrm{d}t} &= -\mu\hat{S} + \varphi f(\hat{I}(t))(1 - \hat{S}(t)) + \gamma\hat{I}(t-\tau)\mathrm{e}^{-\mu\tau}, \\ \frac{\mathrm{d}\hat{I}(t)}{\mathrm{d}t} &= \varphi f(\hat{I}(t))(1 - \hat{S}(t)) - (\mu + \gamma)\hat{I}(t). \end{aligned} \tag{5}$$

Now, this system has an equilibrium $(0, 0)$ and proving global stability of this point means proving global stability of $E_0 = (1, 0)$ for system (2).

From $f(0) = 0$ and the concavity of $f$, we can conclude that for all $I > 0$

$$\varphi f(I) < (\mu + \gamma)I. \tag{6}$$

Using the above-mentioned argument, the equation for $\hat{I}$ can be rewritten as follows:

$$\frac{\mathrm{d}\hat{I}}{\mathrm{d}t} = \varphi f(\hat{I}(t))(1 - \hat{S}(t)) - (\mu + \gamma)\hat{I}(t) \leqslant \varphi f(\hat{I}(t)) - (\mu + \gamma)\hat{I}.$$

Now, with the help of (6) we obtain

$$\frac{\mathrm{d}\hat{I}}{\mathrm{d}t} \leqslant \varphi f(\hat{I}(t)) - (\mu + \gamma)\hat{I} < 0,$$

i.e. there exists a positive constant $c$ such that

$$\frac{\mathrm{d}\hat{I}}{\mathrm{d}t} \leqslant -c. \tag{7}$$

The solutions of this differential inequality are bounded above by the solutions of the corresponding differential equation. Therefore, we obtain the following result:

$$\hat{I} \leqslant I(0)\mathrm{e}^{-ct}$$

and as $t \to \infty$ it follows that $\hat{I} \to 0$, and so does the original $I(t)$.

Now, we need to prove that $\hat{S}(t) \to 0$ as $t \to \infty$. In order to do it we rewrite the equation for $\hat{S}(t)$ as

$$\begin{aligned}\frac{\mathrm{d}\hat{S}(t)}{\mathrm{d}t} &= -\mu\hat{S} + \varphi f(\hat{I}(t))(1-\hat{S}(t)) + \gamma\hat{I}(t-\tau)\mathrm{e}^{-\mu\tau} \\ &\leqslant -\mu\hat{S} + (\mu+\gamma)\hat{I} + \gamma\hat{I}(t-\tau)\mathrm{e}^{-\mu\tau} \\ &\leqslant -\mu\hat{S} + \left[\mu + \gamma(1+\mathrm{e}^{(c-\mu)\tau})\right] I(0)\mathrm{e}^{-ct}. \end{aligned} \tag{8}$$

From the solution of the corresponding differential equation

$$\hat{S}(t) = c_1\mathrm{e}^{-\mu t} + \frac{\mu+\gamma(1+\mathrm{e}^{(c-\mu)\tau})}{\mu-c} I(0)\mathrm{e}^{-ct}$$

with $c_1$ being a constant determined by initial conditions, it can be seen that the solutions of (8) are bounded above by the exponentially decaying function as $t \to \infty$. Thus, we have proved that the solution $E_0 = (1, 0)$ is globally asymptotically stable.

## 6. Existence of a non-trivial equilibrium and its stability analysis

From the previous section it is follows that when the trivial steady state $E_0$ of system (2) is locally asymptotically stable, then non-trivial, or endemic equilibrium is not feasible. This situation is controlled by condition (4). When condition (4) violates, besides the disease-free equilibrium, system (2) has a non-trivial equilibrium $E^*_\tau = (S^*_\tau, I^*_\tau)$. System (2) is a nonlinear delayed system, so it is very complicated task to find an explicit expression for $E^*_\tau = (S^*_\tau, I^*_\tau)$. Therefore, rather than look for an explicit form of it, we shall prove that an endemic equilibrium exists, and then perform its stability analysis.

From the second equation of system (3) it follows that

$$S = \frac{(\mu+\gamma)I}{\varphi f(I)}.$$

After substituting this expression into the first equation of system (3), we obtain the following equation for $I$:

$$H(I) = \mu - \frac{\mu(\mu+\gamma)I}{\varphi f(I)} - (\mu+\gamma)I + \gamma I\mathrm{e}^{-\mu\tau} = 0.$$

It can be easily seen that the function $H(I)$ is negative for large positive $I$. Next, we determine the sign of its derivative:

$$H'(I) = -\mu(\mu+\gamma)\frac{f(I) - If'(I)}{\varphi f^2(I)} - (\mu+\gamma) + \gamma\mathrm{e}^{-\mu\tau}.$$

From the properties of the function $f(I)$, in particular, from $f(0) = 0$ and $f''(I) < 0$ it follows that $f(I) - If'(I) > 0$, and consequently, $H'(I) < 0$ for all $I > 0$. Therefore, for a positive root of $H(I) = 0$ to exist, $H(I)$ has to satisfy $H(0) > 0$, i.e.

$$H(0) = \mu\left[1 - \frac{\mu+\gamma}{\varphi f'(0)}\right] = \mu\left[1 - \frac{1}{\mathscr{R}_0}\right].$$

Hence, one needs to require $\mathscr{R}_0 > 1$ to ensure the existence of an endemic equilibrium. Therefore, we have proved the existence and uniqueness of the endemic equilibrium $E_\tau^* = (S_\tau^*, I_\tau^*)$ for system (2). This result is summarized below:

**Theorem 2.** *Assume that all conditions imposed on the function $f(I)$ hold. Then, is $\mathscr{R}_0 > 1$ then system* (2) *has a unique equilibrium $E_\tau^*$.*

The linearisation matrix of system (2) near the steady state $E_\tau^* = (S_\tau^*, I_\tau^*)$ has the following characteristic equation:

$$\begin{aligned}\Delta_1(\lambda) = \lambda^2 + (2\mu + \varphi f(I_\tau^*) - \varphi f'(I_\tau^*)S_\tau^* + \gamma)\lambda - \mu\varphi f'(I_\tau^*)S_\tau^* + \mu^2 + \mu\gamma \\ + \varphi f(I_\tau^*)\gamma + \varphi\mu f(I_\tau^*) - \gamma\varphi f(I_\tau^*)\mathrm{e}^{-\tau(\mu+\lambda)}.\end{aligned} \tag{9}$$

First, we look at the situation when there is no temporal immunity from a disease, i.e. $\tau = 0$. With $\tau = 0$, system (3) reduces to

$$\begin{cases} \mu - \mu S_0^* - \varphi f(I_0^*)S_0^* + \gamma I_0^* = 0, \\ \varphi f(I_0^*)S_0^* - (\mu + \gamma)I_0^* = 0. \end{cases}$$

Let the solution of this system be $E_0^* = (S_0^*, I_0^*)$. From Eq. (9) with $\tau = 0$ it follows, that this steady state is locally asymptotically stable if the following condition holds:

$$\mu + \gamma + \varphi f(I_0^*) > \varphi f'(I_0^*)S_0^*. \tag{10}$$

Returning to Eq. (9), we choose $\tau > 0$, and introduce the following notation:

$$A_\tau = 2\mu + \varphi f(I_\tau^*) - \varphi f'(I_\tau^*)S_\tau^* + \gamma$$

and

$$B_\tau = -\mu\varphi f'(I_\tau^*)S^* + \mu^2 + \mu\gamma + \varphi f(I_\tau^*)\gamma + \varphi\mu f(I_\tau^*).$$

With this notation Eq. (9) becomes

$$\lambda^2 + A_\tau\lambda + B_\tau - \gamma\varphi f(I_\tau^*)\mathrm{e}^{-\tau(\lambda+\mu)} = 0. \tag{11}$$

Suppose, $\lambda = \mathrm{i}v$ with $v > 0$ is a root of Eq. (11) for some $\tau$. Then, after substituting this into (11) we obtain

$$-v^2 + A_\tau \mathrm{i}v + B_\tau - \gamma\varphi f(I_\tau^*)\mathrm{e}^{-\tau(\lambda+\mu)} = 0.$$

Then, after separating it into real and imaginary parts, we obtain

$$\begin{cases} -v^2 + B_\tau = \gamma\varphi f(I_\tau^*)\mathrm{e}^{-\tau\mu}\cos\,\tau v, \\ A_\tau v = -\gamma\varphi f(I_\tau^*)\mathrm{e}^{-\tau\mu}\sin\,\tau v. \end{cases}$$

Thus, upon squaring and adding the last two equations, it gives us the following equation:

$$v^4 + (A_\tau^2 - 2B_\tau)v^2 + B_\tau^2 - \gamma^2\varphi^2 f^2(I_\tau^*)\mathrm{e}^{-2\tau\mu} = 0. \tag{12}$$

**Theorem 3.** *Suppose that the conditions*

$$A_\tau^2 > 2B_\tau, \quad \text{and} \quad B_\tau > \gamma^2\varphi^2 f^2(I_\tau^*)\mathrm{e}^{-2\tau\mu} \tag{13}$$

*hold for all* $\tau \geqslant 0$. *Then the infected steady state* $E_\tau^* = (S_\tau^*, I_\tau^*)$ *of system* (2) *is locally asymptotically stable.*

In terms of the parameters of system (2), the two conditions in (13) can be explicitly written as

$$\mu(\mu + 2\varphi f(I_\tau^*)) + (\gamma+\mu)^2 + \varphi^2(f'(I_\tau^*)S_\tau^* - f(I_\tau^*))^2 > 2\varphi f'(I_\tau^*)S_\tau^*(\gamma+\mu)$$

and

$$\begin{aligned}&[\mu^2+\mu\gamma]^2 + [\mu\varphi f'(I_\tau^*)S_\tau^* + \gamma\varphi f(I_\tau^*)]^2 + \varphi^2\gamma^2 f^2(I_\tau^*)(1-\mathrm{e}^{-2\mu\tau})\\ &\quad + 2\mu^2\varphi f(I_\tau^*)[2\gamma - \varphi f(I_\tau^*)S_\tau^*] + 2\mu\varphi\gamma[\varphi f^2(I_\tau^*) - \mu f'(I_\tau^*)S_\tau^*]\\ &\quad + 2\mu^3\varphi[f(I_\tau^*) - f'(I_\tau^*)S_\tau^*] + 2\varphi^2\gamma\mu f^2(I_\tau^*) > 0.\end{aligned}$$

We have proved that when an endemic steady state is feasible, then under condition (13) it is locally asymptotically stable.

## 7. Global stability of the endemic steady state

Choosing a linear incidence rate $f(I) = I$, we return to system (1) and center it at the endemic equilibrium $E_\tau^* = (S_\tau^*, I_\tau^*, R_\tau^*)$ by introducing new variables as

$$u_1 = S - S_\tau^*, \quad u_2 = I - I_\tau^* \quad \text{and} \quad u_3 = R - R_\tau^*.$$

After substituting these variables, system (1) can be rewritten in the following form:

$$\begin{aligned}\frac{\mathrm{d}u_1}{\mathrm{d}t} &= -\mu u_1 - \varphi S u_2 - \varphi u_1 I_\tau^* + \gamma u_2(t-\tau)\mathrm{e}^{-\mu\tau},\\ \frac{\mathrm{d}u_2}{\mathrm{d}t} &= \varphi S u_2 + \varphi u_1 I_\tau^* - (\mu+\gamma)u_2,\\ \frac{\mathrm{d}u_3}{\mathrm{d}t} &= \gamma u_2 - \gamma u_2(t-\tau)\mathrm{e}^{-\mu\tau} - \mu u_3.\end{aligned} \tag{14}$$

Now, proving that a trivial solution of system (14) is globally asymptotically stable, will immediately prove the fact that the endemic equilibrium $E_\tau^*$ of system (1) is globally asymptotically stable. We shall employ Lyapunov functional technique to prove it (see, for example [1]). Before embarking on the analysis, we prove the following lemma which will be used in our further calculations.

**Lemma 2.** *Let the initial data for system* (1) *be* $S(0) = S_0 > 0$, $I(s) = I_0(s) \geqslant 0$ *for all* $s \in [-\tau, 0)$ *with* $I_0(0) > 0$ *and* $R(0) = R_0 > 0$. *Then* $S(t) \leqslant \max\{1, S_0 + I_0 + R_0\} = M$ *for all* $t > 0$.

**Proof.** From Section 2 we know that $N(t) = S(t) + I(t) + R(t)$ is a monotone function and $N(t) \to 1$ as $t \to \infty$. Suppose that $N(0) \leqslant 1$. Then, $N(t) \leqslant 1$ for all $t > 0$. From positivity

of solutions of system (1) it follows that $S(t) \leqslant 1$ for all $t > 0$. On the contrary, if $N(0) > 1$ then $N(t) < N(0)$ and, hence, $S(t) < N(0)$ for all $t > 0$. The proof is complete. □

Now, let us introduce the following functional:

$$V(u) = \tfrac{1}{2}w(u_1 + u_2)^2 + \tfrac{1}{2}(u_2^2 + u_3^2),$$

where $w > 0$ is an arbitrary real constant. The derivative of $V$ is

$$\begin{aligned} V'(u) = {} & u_3[\gamma u_2 - \gamma u_2(t-\tau)\mathrm{e}^{-\mu\tau} - \mu u_3] + w[-\mu u_1 - \varphi S u_2 - \varphi u_1 I_\tau^* \\ & + \gamma u_2(t-\tau)\mathrm{e}^{-\mu\tau} + \varphi S u_2 + \varphi u_1 I_\tau^* - (\mu+\gamma)u_2](u_1+u_2) \\ & + u_2(\varphi S u_2 + \varphi u_1 I_\tau^* - (\mu+\gamma)u_2) \end{aligned}$$

or, equivalently,

$$\begin{aligned} V'(u) \leqslant {} & -\mu u_3^2 - w\mu u_1^2 - w\mu u_2^2 - w\mu u_2^2 + u_1u_2[-w(\mu+\gamma) - w\mu + \varphi I_\tau^*] \\ & + \gamma u_2 u_3 + \varphi S u_2^2 - \gamma u_3 u_2(t-\tau)\mathrm{e}^{-\mu\tau} + w\gamma u_1 u_2(t-\tau)\mathrm{e}^{-\mu\tau} \\ & + w\gamma u_2 u_2(t-\tau)\mathrm{e}^{-\mu\tau} - (\mu+\gamma)u_2^2. \end{aligned}$$

Choosing $w$ as follows:

$$w = \frac{\varphi I_\tau^*}{2\mu+\gamma}$$

and applying Cauchy–Schwartz inequality to all $u_i u_j$-type terms, we arrive at the following expression:

$$\begin{aligned} V'(u) \leqslant {} & -\mu u_3^2 - w\mu u_1^2 - [w\mu - \varphi M + (\mu+\gamma) + w\gamma]u_2^2 + \frac{\gamma}{2}(u_2^2+u_3^2) \\ & + \frac{\gamma}{2}u_3^2\mathrm{e}^{-\mu\tau} + \frac{\gamma}{2}u_2^2(t-\tau)\mathrm{e}^{-\mu\tau} + \frac{w\gamma}{2}u_1^2\mathrm{e}^{-\mu\tau} + \frac{w\gamma}{2}u_2^2(t-\tau)\mathrm{e}^{-\mu\tau} \\ & + \frac{w\gamma}{2}u_2^2\mathrm{e}^{-\mu\gamma} + \frac{w\gamma}{2}u_2^2(t-\tau)\mathrm{e}^{-\mu\tau}. \end{aligned}$$

Arranging similar terms in the last inequality gives

$$\begin{aligned} V'(u) \leqslant {} & -\left(\mu - \frac{\gamma}{2}\right)u_3^2 - w\mu u_1^2 - \left[(w+1)(\mu+\gamma) - \varphi M - \frac{\gamma}{2}\right]u_2^2 + \frac{w\gamma}{2}u_1^2\mathrm{e}^{-\mu\tau} \\ & + \frac{\gamma}{2}u_3^2\mathrm{e}^{-\mu\tau} + \gamma\left(w+\frac{1}{2}\right)u_2^2(t-\tau)\mathrm{e}^{-\mu\tau}. \end{aligned} \tag{15}$$

Assuming that $\mu > \gamma$ and $\mu + \gamma/2 + \varphi I_\tau^* - (\varphi I_\tau^* \mu/(2\mu+\gamma)) - \varphi M > 0$ for sufficiently large time, e.g., $t > t_1 + \tau$, leads us to the following:

$$\begin{aligned} V'(u) \leqslant {} & -w\mu u_1^2 - \left[(w+1)(\mu+\gamma) - \varphi M - \frac{\gamma}{2}\right]u_2^2 - \left(\mu - \frac{\gamma}{2}\right)u_3^2 + \frac{w\gamma}{2}u_1^2\mathrm{e}^{-\mu\tau} \\ & + \gamma\left(w+\frac{1}{2}\right)u_2^2(t-\tau)\mathrm{e}^{-\mu\tau} + \frac{\gamma}{2}u_3^2\mathrm{e}^{-\mu\tau}. \end{aligned} \tag{16}$$

We choose Lyapunov functional to be of the form:

$$U(u_t) = V(u) + \gamma\left(w + \frac{1}{2}\right)\mathrm{e}^{-\mu\tau}\int_{t-\tau}^{t} u_2^2(\theta)\,\mathrm{d}\theta$$

and, hence,

$$U'(u_t) = V'(u) + \gamma(w+\tfrac{1}{2})\mathrm{e}^{-\mu\tau}u_2^2(t) - \gamma(w+\tfrac{1}{2})\mathrm{e}^{-\mu\tau}u_2^2(t-\tau).$$

Substituting inequality for $V'(u)$, we get

$$\begin{aligned} U'(u_t) \leqslant & -\left(\mu - \frac{\gamma}{2}\right)u_3^2 - w\mu u_1^2 - \left[(w+1)(\mu+\gamma) - \varphi M - \frac{\gamma}{2}\right]u_2^2 + \frac{w\gamma}{2}u_1^2\mathrm{e}^{-\mu\tau} \\ & + \gamma\left(w+\frac{1}{2}\right)u_2^2(t-\tau)\mathrm{e}^{-\mu\tau} + \frac{\gamma}{2}u_3^2\mathrm{e}^{-\mu\tau} + \gamma\left(w+\frac{1}{2}\right)\mathrm{e}^{-\mu\tau}u_2^2 \\ & - \gamma\left(w+\frac{1}{2}\right)\mathrm{e}^{-\mu\tau}u_2^2(t-\tau). \end{aligned} \tag{17}$$

Therefore,

$$\begin{aligned} U'(u_t) \leqslant & -\left(\mu - \frac{\gamma}{2} - \frac{\gamma}{2}\mathrm{e}^{-\mu\tau}\right)u_3^2 - \left(w\mu - \frac{w\gamma}{2}\mathrm{e}^{-\mu\tau}\right)u_1^2 \\ & - \left[(w+1)(\mu+\gamma) - \varphi M - \frac{\gamma}{2} - \gamma\left(w+\frac{1}{2}\right)\mathrm{e}^{-\mu\tau}\right]u_2^2. \end{aligned} \tag{18}$$

The last expression is negative definite provided that

$$\tau > \max\left\{\frac{1}{\mu}\log\left[\frac{\gamma}{2\mu-\gamma}\right], \frac{1}{\mu}\log\left[\frac{\gamma}{2\mu}\right], \frac{1}{\mu}\log\left[\frac{\gamma(w+1/2)}{(w+1)(\mu+\gamma)-\varphi M-\gamma/2}\right]\right\}.$$

A direct application of the Lyapunov–LaSalle type theorem (Theorem 2.5.3 of Kuang [6, p. 30]) shows that $\lim_{t\to\infty} u_i(t) = 0$, $i = 1, 2, 3$. We have proved the following theorem.

**Theorem 4.** *Let the initial conditions for system* (1) *be* $S(0) = S_0 > 0$, $I(s) = I_0(s) \geqslant 0$, $s \in [-\tau, 0)$ *with* $I_0(0) > 0$ *and* $R(0) = R_0 > 0$. *Assume further that the parameters of system* (1) *satisfy*

$$\mu > \gamma, \quad \mu + \frac{\gamma}{2} + \varphi I^* > \frac{\varphi I^*\mu}{2\mu+\gamma} + \varphi M, \quad \textit{and} \quad \varphi f'(0) > \mu + \gamma,$$

*where* $M = S_0 + I_0 + R_0$. *Then, for any immunity time* $\tau$ *satisfying*

$$\tau > \max\left\{\frac{1}{\mu}\log\left[\frac{\gamma}{2\mu-\gamma}\right], \frac{1}{\mu}\log\left[\frac{\gamma}{2\mu}\right], \frac{1}{\mu}\log\left[\frac{\gamma(w+1/2)}{(w+1)(\mu+\gamma)-\varphi M-\gamma/2}\right]\right\},$$

*the endemic equilibrium* $E^*$ *is globally asymptotically stable.*

## 8. Numerical simulations

In this section we study model (1) numerically. All simulations were performed using the Delay Differential Equations (DDE) suite in Matlab [11].

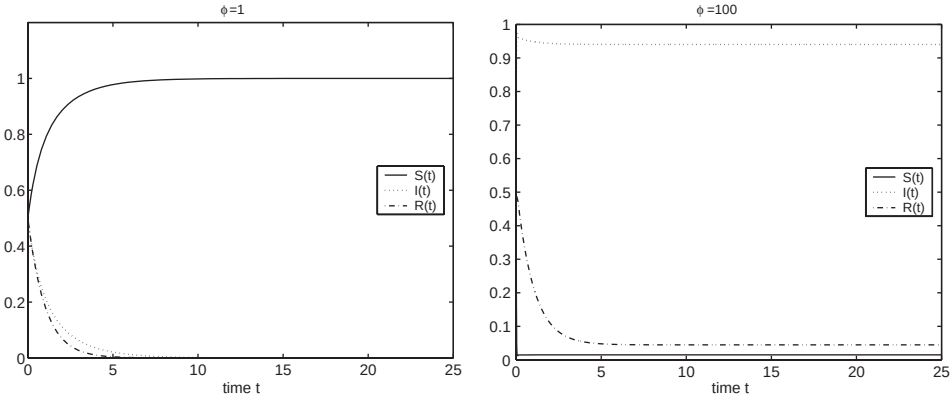


Fig. 1. A solution of model (1) with $f(I) = I$, $S(s) = I(s) = R(s) = 0.5$, $s \in [-\tau, 0]$. Parameter values are $\mu = 1$, $\gamma = 0.5$, $\tau = 0.1$, and $\varphi$ varies from 1 to 100.

We begin by considering a case of small time delay $\tau$. Fig. 1 represents two different possibilities which can be realised in this situation. For sufficiently small disease transmission rate $\varphi$ (such that $\mathscr{R}_0 < 1$) the disease dies out of the population, and the solutions approach the globally stable disease-free steady state $E_0$. On the other hand, for larger $\varphi$ (i.e. $\mathscr{R}_0 > 1$) the endemic equilibrium $E^*_\tau$ is feasible and stable while $E_0$ loses its stability.

In the remainder of this section we concentrate on the case when $f(I) = I/(1 + I)$ and $\mathscr{R}_0 > 1$. In this case, the endemic equilibrium is given explicitly by

$$E^*_\tau = (S^*_\tau, I^*_\tau, R^*_\tau) = \begin{cases} S^*_\tau = \dfrac{(\mu+\gamma)(2\mu+\gamma-\gamma e^{-\mu\tau})}{\mu(\mu+\varphi+\gamma)-\gamma\varphi(1-e^{-\mu\tau})}, \\ I^*_\tau = \dfrac{\mu(\varphi-\mu-\gamma)}{\mu(\mu+\varphi+\gamma)-\gamma\varphi(1-e^{-\mu\tau})}, \\ R^*_\tau = \dfrac{\gamma(\varphi-\mu-\gamma)(1-e^{-\mu\tau})}{\mu(\mu+\varphi+\gamma)-\gamma\varphi(1-e^{-\mu\tau})}. \end{cases}$$

As $\tau$ increases, this steady state can undergo Hopf bifurcation and give rise to a stable periodic solution. In the beginning ($\tau = 1$), the amplitude of these oscillations is very small, and, therefore, this periodic orbit is hardly distinguishable from the steady state $E^*_\tau$ itself. As it is shown in Fig. 2, for larger delays the amplitude of the oscillations increases. For sufficiently large $\tau$, stability of $E^*_\tau$ is regained, and initial oscillations around $E^*_\tau$ are quickly damped. In the case of linear incidence rate, dynamics of solutions is qualitatively the same as in Fig. 2.

## 9. Conclusions

This paper has been concerned with modelling of a disease dynamics with temporary immunity period, which is an important feature of many diseases. Previous efforts on incorporating delays in epidemic models have been mainly concentrated on inclusion of latency

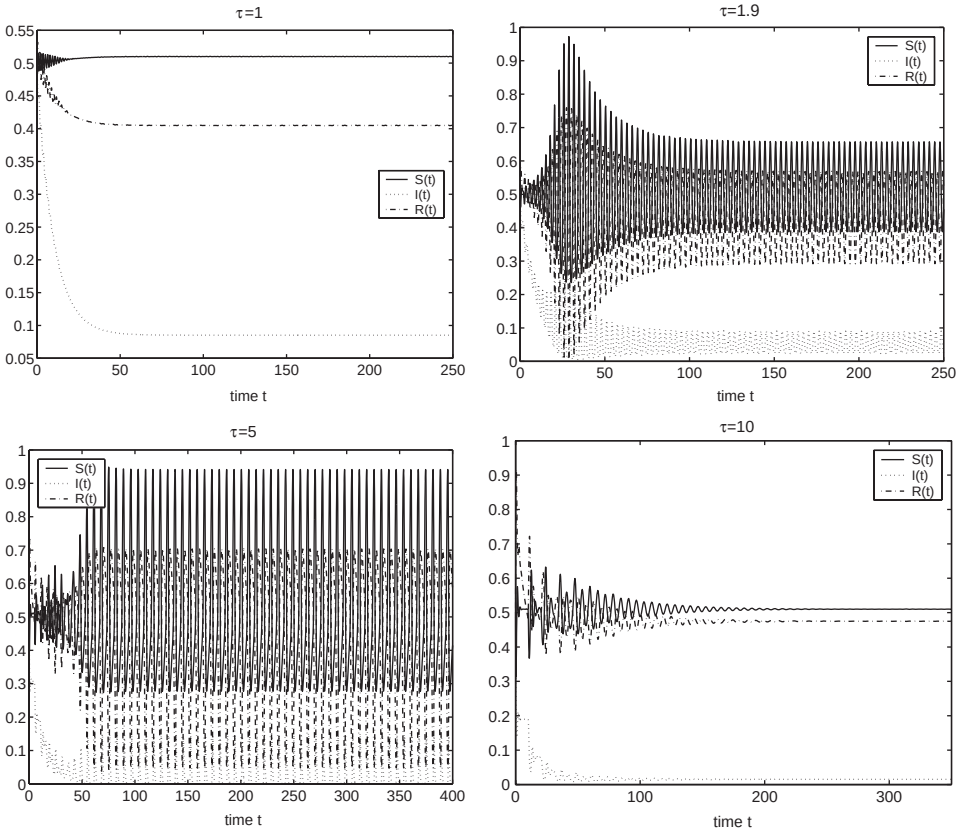


Fig. 2. A solution of model (1) with $f(I) = I/(1 + I)$, $S(s) = I(s) = R(s) = 0.5$, $s \in [-\tau, 0]$. Parameter values are $\mu = 0.1$, $\gamma = 5$, $\varphi = 10$, and $\tau$ varies from 1 to 10.

periods (this assumes that the force of infection at a present time is determined by the number of infectives in the past). However, it is epidemiologically reasonable to allow individuals to have immunity for some time after they recover from infection.

We have analytically studied model (1) with a general (possibly, nonlinear) incidence rate as far as possible, and restricted ourselves to the case of $f(I) = I$ and $f(I) = I/(1 + I)$ later to enable further analytic progress and for the purposes of numerical simulations. When $\mathscr{R}_0 < 1$, the disease-free steady state $E_0$ is globally asymptotically stable, and no other equilibria exist. When $\mathscr{R}_0 > 1$, the steady state $E_0$ loses its stability, and an endemic equilibrium $E_\tau^*$ appears. Using Lyapunov functional technique, we have been able to show that under certain restrictions on the parameter values and the delay time, this equilibrium is globally asymptotically stable.

To further investigate our model we resorted to numerical simulations. They show that for $\mathscr{R}_0 > 1$ and a small immunity time $\tau$, the solutions are represented by small amplitude oscillations near the steady state $E_\tau^*$. As the immunity period $\tau$ increases, the amplitude of

these oscillations increases correspondingly. Further increase of $\tau$ returns the oscillatory dynamics to the globally attractive steady-state form. This shows the dependence of a long-term dynamics of solutions on the immunity period $\tau$. A realistic extension of this work is to assume that the immunity time may depend on the particular characteristics of individuals (such as age, general state of health, loss of immunity due to waning of vaccine, etc.), and this is currently a work in progress.

## Acknowledgements

The authors would like to thank the Referee for careful reading of the manuscript and making useful comments which helped improve the presentation of the paper.

## References

[1] E. Beretta, Y. Kuang, Modeling and analysis of a marine bacteriophage infection with latency period, Nonlinear Anal. 2 (2001) 35–74.
[2] E. Beretta, Y. Takeuchi, Global stability of an SIR epidemic model with time delays, J. Math. Biol. 33 (1995) 250–260.
[3] E. Beretta, Y. Takeuchi, Convergence results in SIR epidemic models with varying population sizes, Nonlinear Anal. 28 (1997) 1909–1921.
[4] K.L. Cooke, Stability analysis for a vector disease model, Rocky Mount. J. Math. 9 (1979) 253–263.
[5] O. Diekmann, J.A.P. Heesterbeek, Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation, Wiley, New York, 2000.
[6] Y. Kuang, Delay Differential Equations with Applications in Population Biology, Academic Press, New York, 1993.
[7] B. Li, Y. Kuang, Simple food chain in a chemostat with distinct removal rates, J. Math. Anal. Appl. 242 (2000) 75–92.
[8] W. Liu, H.W. Hethcote, S.A. Levin, Dynamical behaviour of epidemiological models with nonlinear incidence rate, J. Math. Biol. 25 (1987) 359–380.
[9] W. Liu, S.A. Levin, Y. Iwasa, Influence of nonlinear incidence rates upon behaviour of SIRS epidemiological models, J. Math. Biol. 23 (1986) 187–204.
[10] S.M. Moghadas, A.B. Gumel, Global stability of a two-stage epidemic model with generalized non-linear incidence rate, Math. Comput. Simulation 60 (2002) 107–118.
[11] L.F. Shampine, S. Thompson, Solving DDEs in MATLAB, Appl. Numer. Math. 37 (2001) 441–458.