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ORIGINAL ARTICLE

A Time Since Recovery Model with Varying Rates of Loss of Immunity

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Abstract For many infectious diseases, immunity wanes over time. The majority of SIRS models assume that this loss of immunity takes place at a constant rate. We study temporary immunity within a SIRS model structure if the rate of loss of immunity can depend on the time since recovery from disease. We determine the conditions under which the endemic steady state becomes unstable and periodic oscillations set in, showing that a fairly rapid change between slow and rapid immunity loss is necessary to produce oscillations.

Keywords Epidemiology · Immunity · Time since recovery · Oscillations

1 Introduction

Compartmental models for microparasitic infectious diseases separate a population into classes depending upon the stages of infection (Anderson and May 1992). In simple compartmental models, the disease is either eradicated or reaches a stable endemic equilibrium, depending upon its basic reproductive ratio. Complex dynamics may arise due to seasonality or pathological effects. Many diseases, such as measles, mumps, rubella, chicken pox and pertussis, show periodicity in incidence due to whole array of potential causes: periodic transmission rates, host age-structure, interactions between multiple strains, complex incidence, variable population size, and disease-related death (Hethcote and Levin 1989).

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Loss of immunity creates another time delay that can destabilize dynamics. Childhood diseases like measles have essentially lifelong immunity, although the importance of "boosting" due to later exposure remains unclear (Krugman et al. 1965). Other diseases, however, show immunity that wanes either due to loss of immune memory in hosts or evolution of the disease itself (Pease 1987). Models that include multiple levels of immunity that wanes in the absence of antigen but can be boosted with re-exposure create non-constant rates of decay of immunity (Heffernan and Keeling 2009).

Temporary immunity has been addressed previously in the theoretical literature. Hethcote (1976) modeled vital disease dynamics and its endemicity in a closed population both with and without temporary immunity. Stech and Williams (1981) studied temporary immunity within a SIRS epidemic model structure and derived sufficient conditions for global stability of the endemic equilibrium. Hethcote et al. (1981) considered an epidemic model for a closed population, described by integral and integro-differential equation, to determine the effect of time delays in development of periodic oscillations. When temporary immunity was of constant duration, corresponding to a delay, they showed that locally asymptotically stable small amplitude periodic solutions exist in the appropriate parameter range. They also showed that a γ-distributed time delay can be modeled by a chain of n subclasses within the removed class as an $SIR_1R_2 \ldots R_nS$ model can support periodic solutions when $n \geq 3$. A related study (Hethcote et al. 1981) of a constant population SEIS model showed no periodicity in the absence of immunity. This model complements the model with temporary immunity by introducing the delay before rather than after the infectious period.

Cooke and van den Driessche (1996) considered an SEIRS model with two fixed delays, corresponding to latent and immune periods. Hethcote (1985) added a separate class to represent temporary acquired immunity for newborn infants in an MSEIR model (Hethcote 1985, 2000). Including a distribution of times in the infected class rather than the immune class creates substantial effects on the dynamics, particularly with regard to disease extinction (Keeling and Grenfell 1997).

White and Medley (1998) developed a model framework corresponding to continuous temporary immunity. Hosts were distributed over a continuous range of immunity with immune levels varying within the host due to waning immunity between exposures and increasing immunity during infection. Gomes et al. (2005) developed a model with a constant rate of loss of immunity, but looked at the related effects of partial immunity within the same framework.

Introducing delays in the duration of immunity by using integro-differential equations was suggested by Brauer and Castillo-Chávez (2001). Kyrychko and Blyuss (2005) studied temporary immunity and non-linear incidence rates, finding dependence of oscillatory dynamics on the immunity period τ, with the amplitude of oscillations increasing with longer duration immunity. In a more recent paper, Blyuss and Kyrychko (2010) study epidemic models with varying immunity. They model the system with delay differential equations, in which temporary immunity wanes with time. They analyze the local and global stability of the endemic steady states using Lyapunov functions and show that the endemic equilibrium is stable, and use a numerical bifurcation analysis to show that the endemic equilibrium can lose stability and give rise to stable periodic solutions.

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Other recent work considers temporary immunity in a SIRS model structure using delay differential equations (DDEs), where R and S classes are coupled using a fixed delay in the removed class (Taylor and Carr 2009). In these models, only a fraction of the recovered population returns to the susceptible class, with the rest remaining permanently immune. Using asymptotic methods they determine how oscillations depend upon the parameters, primarily the duration of the delay and the fraction of individuals who become susceptible again. With a fixed delay, they show that it is possible to obtain periodic epidemics via a Hopf bifurcation of the endemic steady state, and that longer delays lead to larger epidemics with longer periods.

The present article considers more generally the role of temporary immunity in a population. We consider a generalization of the SIR model in a closed population. Every recovered individual returns to the susceptible class at a rate that depends upon time since recovery. The model is mathematically formulated using a combination of ordinary and partial differential equations. This model is more general because the kernel function defining the rate of loss of immunity models a specific distribution of immune times instead of introducing a fixed period of temporary immunity. We show that the shape of this function determines whether or not the delays in the model can support oscillations.

2 The Mathematical Model

Our SIRS model consists of a temporarily immune class which loses immunity at rate $\rho(\tau)$ as a function of the time τ since recovery:

$$\frac{dS}{dt} = -\beta I(t)S(t) + \left(\int_0^\infty \rho(\tau)R(\tau,t)\,d\tau\right) \tag{1}$$

$$\frac{dI}{dt} = \beta I(t)S(t) - \gamma I(t) \tag{2}$$

$$\frac{\partial R}{\partial t} + \frac{\partial R}{\partial \tau} = -\rho(\tau)R(\tau,t) \tag{3}$$

$S(t)$, $I(t)$, $R(\tau,t)$ represent the susceptible, infected and recovered population, respectively, and we use the initial conditions $S(0) = S_0$, $I(0) = I_0$, $R(\tau,0) = 0$ and boundary condition $R(0,t) = \gamma I(t)$. The parameter β gives the rate of transmission of the disease and γ is the rate of recovery of the infected class. We assume constant population size and scale the total population size $N(t) = S(t) + I(t) + \int_0^\infty R(\tau,t)\,d\tau = 1$. The probability that an immune individual remains immune at time τ after recovery is $l(\tau) = e^{-\int_0^\tau \rho(s)\,ds}$. We assume that $\lim_{\tau\to\infty} l(\tau) = 0$, or equivalently that $\int_0^\infty \rho(\tau)\,d\tau = \infty$. To avoid divergence of the integrals, we assume that $\rho(\tau)$ is bounded for τ large, which is the situation for all cases considered here. The mean time to immunity is $L = \int_0^\infty l(\tau)\,d\tau$.

2.1 Steady States

This model, like a standard SIR model, has basic reproduction ratio $\mathcal{R}_0 = \frac{\beta}{\gamma}$. Equations (1)–(3) have two steady states, the disease-free steady state ($S^* = 1$, $I^* = 0$, $R^*(\tau) = 0$) and, when $\mathcal{R}_0 > 1$. an endemic steady state

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$$S^* = \frac{1}{\mathcal{R}_0} \tag{4}$$

$$I^* = \frac{\beta - \gamma}{\beta(1 + \gamma \int_0^\infty l(\tau)\, d\tau)} \tag{5}$$

$$R^*(\tau) = \gamma I^* l(\tau) \tag{6}$$

For the stability analysis of the disease-free steady state we consider perturbations around the equilibrium point of the form

$$S = 1 + x_0 e^{\lambda t}$$
$$I = y_0 e^{\lambda t}$$
$$R(\tau, t) = z_0(\tau) e^{\lambda t}$$

Substituting these into Eqs. (1)–(3) and evaluating gives

$$x_0 \lambda = \int_0^\infty \rho(\tau) z_0(\tau)\, d\tau - \beta y_0$$
$$y_0 \lambda = (\beta - \gamma) y_0$$
$$z_0(\tau) = \gamma y_0 e^{-\lambda \tau} l(\tau)$$

The corresponding characteristic equation is given by

$$\det \begin{pmatrix} -\lambda & (\gamma - \beta) - \lambda \gamma \int_0^\infty l(\tau) e^{-\lambda \tau}\, d\tau \\ 0 & (\beta - \gamma) - \lambda \end{pmatrix} = 0$$

which has the roots $\lambda = 0$ and $\lambda = (\beta - \gamma)$. Therefore the disease-free steady state is stable for $\lambda < 0$, or when $\mathcal{R}_0 < 1$.

We analyze the stability of the non-zero endemic state using linear stability analysis by perturbing around the equilibrium and assuming exponential growth of perturbations or

$$S = S^* + x_0 e^{\lambda t}$$
$$I = I^* + y_0 e^{\lambda t}$$
$$R(\tau, t) = R^*(\tau) + z_0(\tau) e^{\lambda t}$$

Substituting into Eqs. (1)–(3) and evaluating at the equilibrium gives

$$\lambda x_0 = -\beta I^* x_0 - \beta S^* y_0 + \int_0^\infty \rho(\tau) z_0(\tau)\, d\tau$$
$$\lambda y_0 = \beta I^* x_0 + \beta S^* y_0 - \gamma y_0$$

We can solve for $z(\tau) = \gamma y_0 l(\tau) e^{-\lambda \tau}$ by substituting into Eq. (3). With this substitution, the equations for x_0 and y_0 can be written as the matrix equation

$$\lambda \begin{pmatrix} x_0 \\ y_0 \end{pmatrix} = \begin{pmatrix} -\beta I^* & -\gamma + \gamma F \\ \beta I^* & 0 \end{pmatrix} \begin{pmatrix} x_0 \\ y_0 \end{pmatrix}$$

where $F = \int_0^\infty \rho(\tau) l(\tau) e^{-\lambda \tau} d\tau$. Substituting in the solution for $z(\tau)$ and integrating by parts gives the simplification $F = 1 - \lambda \int_0^\infty l(\tau) e^{-\lambda \tau} d\tau$. The matrix equation has a non-trivial solution when

$$\det \begin{pmatrix} -\beta I^* - \lambda & -\lambda \gamma \int_0^\infty l(\tau) e^{-\lambda \tau} d\tau \\ \beta I^* & -\lambda \end{pmatrix} = 0$$

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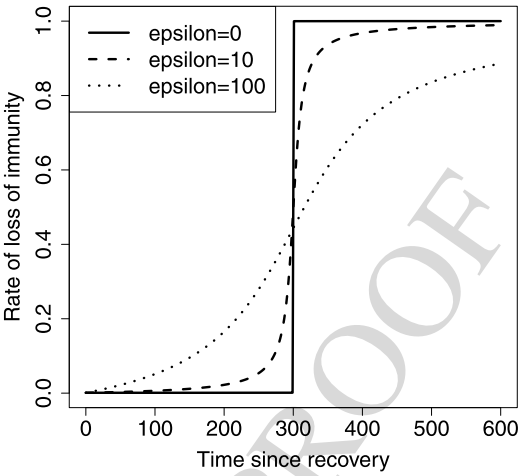

Fig. 1 Comparison of the $\rho(\tau)$ functions. $\epsilon = 0$ corresponds to the step function, while $\epsilon = 10{,}100$ corresponds to the second smoother function

This has a root when $\lambda = 0$ due to the assumption of constant population size, and other roots that solve the characteristic equation

$$\lambda + \beta I^* = -\gamma\beta I^* \left(\int_0^\infty l(\tau) e^{-\lambda\tau}\, d\tau \right) \tag{7}$$

To determine the stability of the endemic equilibrium we need to determine whether any of the solutions of the characteristic equation have positive real parts.

We break Eq. (7) into real and imaginary parts, as $\lambda = a + i\omega$ where a and ω are real. Then

$$a + \beta I^* = -\gamma\beta I^* C(\omega) \tag{8}$$

$$\omega = \gamma\beta I^* S(\omega) \tag{9}$$

where $C(\omega)$ and $S(\omega)$ are the sine and cosine transforms of $l(\tau)$ defined by

$$C(\omega) = \int_0^\infty l(\tau) e^{-a\tau} \cos(\omega\tau)\, d\tau \tag{10}$$

$$S(\omega) = \int_0^\infty l(\tau) e^{-a\tau} \sin(\omega\tau)\, d\tau \tag{11}$$

A necessary condition for Eq. (8) to have a solution with positive a is that $C(\omega) < 0$ for some ω when $a = 0$. However, this is impossible when $l(\tau)$ is everywhere concave up, or $l''(\tau) > 0$ for all τ (Tuck 2006). This shows immediately that linear instability cannot occur when $\rho(\tau)$ is constant, because $l(\tau)$ is then a declining exponential which is everywhere concave up.

Two other cases can be shown directly to fail to support oscillations. If $\rho(\tau)$ increases linearly in time according to $\rho(\tau) = \rho_1 \tau$, then

$$l(\tau) = e^{\frac{-\rho_1 \tau^2}{2}}$$

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Although this function is not everywhere concave up, with $a = 0$,

$$C(\omega) = \sqrt{\frac{\pi}{2\rho_1}} e^{\frac{\omega^2}{2\rho_1}}$$

which is everywhere positive.

The linear function

$$l(\tau) = \begin{cases} 1 - \rho_0 \tau & \text{for } \tau \le \frac{1}{\rho_1} \\ 0 & \text{for } \tau > \frac{1}{\rho_1} \end{cases}$$

cannot support oscillations. It is associated with the hazard

$$\rho(\tau) = -\frac{l'(\tau)}{l(\tau)} = \frac{\rho_1}{1 - \rho_1 \tau}$$

for $\tau \le 1/\rho_1$. Although this function increases quickly to infinity, this increase, as in the linear case, is not sufficiently fast to support oscillations.

To simplify further analysis, we use two forms for $\rho(\tau)$. One is a step function, which introduces two different rates of transfer from the R class depending on the time since recovery. Initially everyone loses immunity slowly, but this rate of loss jumps to a higher value after some fixed time. The second function is a smoothed version of the step function where the rate of loss of immunity increases gradually with time since recovery (Fig. 1).

2.2 Step Function

We assume

$$\rho(\tau) = \begin{cases} \rho_1, & 0 < \tau \le T \\ \rho_2, & T \le \tau < \infty \end{cases} \tag{12}$$

In this case, we can evaluate each of the key functions in the characteristic equation explicitly. In particular,

$$l(\tau) = \begin{cases} e^{-\rho_1 \tau} & \text{if } 0 < \tau \le T \\ e^{-\rho_1 T} e^{-\rho_2(\tau - T)} & \text{if } T \le \tau < \infty \end{cases}$$

and the mean time of immunity is

$$L = \frac{1 - e^{-\rho_1 T}}{\rho_1} + \frac{e^{-\rho_1 T}}{\rho_2}$$

which reduces to $L = T + 1/\rho_2$ if $\rho_1 = 0$. The cosine and sine transforms for $a = 0$ are

$$C(\omega) = \frac{\rho_1}{\rho_1^2 + \omega^2} + \frac{(\rho_2 - \rho_1)e^{-\rho_1 T}}{(\rho_1^2 + \omega^2)(\rho_2^2 + \omega^2)} \times \left(\left(\omega^2 - \rho_1 \rho_2\right) \cos(\omega T) + \omega(\rho_1 + \rho_2) \sin(\omega T) \right)$$

$$S(\omega) = \frac{\omega}{\rho_1^2 + \omega^2} + \frac{(\rho_2 - \rho_1)e^{-\rho_1 T}}{(\rho_1^2 + \omega^2)(\rho_2^2 + \omega^2)} \times \left(\left(\omega^2 - \rho_1 \rho_2\right) \sin(\omega T) - \omega(\rho_1 + \rho_2) \cos(\omega T) \right)$$

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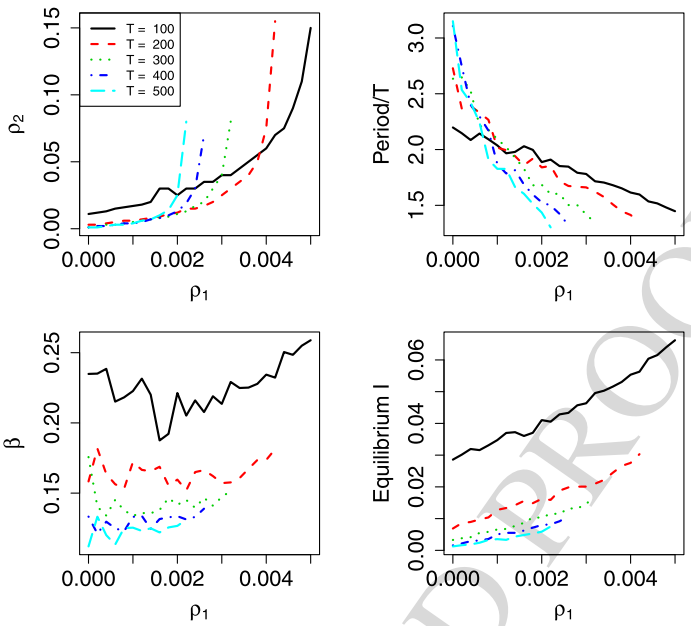

Fig. 2 Effect of the choice of ρ_1 on (**a**). The smallest value of ρ_2 above which oscillations can arise for a given value of ρ_1 with the given value of T and $\gamma = 0.1$. (**b**). The ratio of the period of the oscillation to the delay T at this critical value (**c**). The value of the contact rate β at this critical value. (**d**) The equilibrium I^* at this critical value

Because the value of $C(\omega)$ must be negative for the system to be unstable, we can see immediately that this is impossible if $\rho_2 = \rho_1$, which corresponds to the constant rate case.

If we choose a value of γ, we can numerically find the smallest value of ρ_2 which produces instability for given values of ρ_1 and T. For all values of T, there is critical value of ρ_1 above which no value of ρ_2 is sufficiently large to produce an oscillation (Fig. 2a). At onset, the oscillation has a period roughly twice that length of the time T between the change in rates of loss of immunity (Fig. 2b).

2.3 A Continuous Function

We now consider a continuous approximation of the step function,

$$\rho(\tau) = \frac{\rho_2 \tan^{-1}(\frac{T}{\epsilon}) + \frac{\pi}{2}\rho_1 + (\rho_2 - \rho_1)\tan^{-1}(\frac{\tau - T}{\epsilon})}{\frac{\pi}{2} + \tan^{-1}(\frac{T}{\epsilon})} \tag{13}$$

This function is normalized to set $\rho(0) = \rho_1$ and $\rho(\infty) = \rho_2$. The parameter ϵ gives the time scale of the change between slow and fast rates of recovery.

We choose values of the parameters for which the step function produces oscillations, and examine how ϵ affects the eigenvalues. Because a rather abrupt change in the rate is required to induce instability, we find that smoothing the curve stabilizes the interaction well before the curve becomes everywhere concave up (Fig. 3).

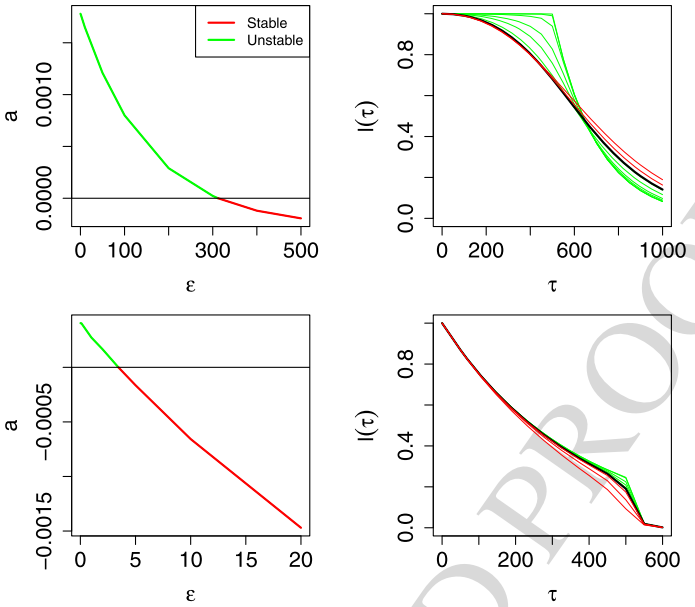

Fig. 3 Effect of the smoothing parameter ϵ on stability of the positive equilibrium. (**a**) The real part of the eigenvalue a given by Eq. (8) as a function of ϵ with $\rho_1 = 0$, $\gamma = 0.1$, $T = 500$, $\beta = 0.112$ and $\rho_2 = 0.005$ chosen to lie above the curve in Fig. 2a. (**b**) The shape of $l(\tau)$ as a function of ϵ with the parameter values in (**a**). The *black curve* shows the value $\epsilon = 312$ where stability switches. (**c**) As in (**a**) but with $\rho_1 = 0.0028$ (to correspond to loss of immunity in roughly one year), $\gamma = 0.1$, $T = 300$, $\beta = 0.141$ and $\rho_2 = 0.05$. (**d**) The shape of $l(\tau)$ with the parameter values in (**b**)

3 Discussion

This paper develops a mathematical approach for understanding the disease dynamics of a population in the presence of waning temporary immunity. The majority of SIRS models of temporary immunity have considered a constant rate of removal for the recovered individuals into the susceptible class, or loss of immunity after a fixed delay. However, temporary immunity is likely to wane at an increasing rate. Using both a step function and a smooth increasing function, we show this increase must be sufficiently large and fast for a Hopf bifurcation to occur, and confirm with numerical study. Introducing a delay in the R class can generate oscillations, but we here demonstrated that oscillations can exist in an SIRS model in the presence of a waning temporary immunity.

Alternative models consider instead a partial loss of immunity, again finding the possibility of oscillations at least in the presence of non-linear incidence (Gomes et al. 2004, 2005). Our analysis assumes that each individual switches from complete immunity to complete susceptibility, although the population will consist of a mix of these two types at any given time since recovery (Taylor and Carr 2009). It would be interesting to compare the results of the present model with one that include partial immunity at the individual level.

Our model also neglects that possibility that immune individuals can have the immunity boosted upon re-encountering the disease, a factor considered in several

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existing models through inclusion of additional internal states (Heffernan and Keeling 2009; Rouderfer et al. 1994; Glass and Grenfell 2003). This factor could be included by resetting τ, the time since recovery, to its initial value. Similarly, vaccination adds individuals to the immune class, with potentially different rates of immunity loss from those who suffered actual infections. These models could show interesting dynamics, because adding vaccination to SIRS models with variable infective and recovery rates can introduce backward bifurcations (Kribs-Zaleta and Martcheva 2002; Schenzle 1984).

For diseases like influenza which produce only temporary immunity, cocirculation of multiple disease strains is common. Models of multiple disease strains often exhibit oscillatory dynamics (Dawes and Gog 2002; Dietz 1979; Ferguson et al. 2003). The present model could also be extended to include more than one disease strain to study how waning temporary immunity affects population dynamics and the strain coexistence.

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