Theoretical Population Biology 113 (2017) 47–55

Contents lists available at ScienceDirect

Theoretical Population Biology

journal homepage: www.elsevier.com/locate/tpb

Equivalent probability density moments determine equivalent epidemics in an SIRS model with temporary immunity

Thomas W. Carr *

Department of Mathematics, Southern Methodist University, Dallas, TX 75275-0156, United States

ARTICLE INFO

Article history:
Received 13 June 2016
Available online 1 December 2016

Keywords:
Integro differential equations
Delay differential equations
Epidemiology
Temporary immunity

ABSTRACT

In an SIRS compartment model for a disease we consider the effect of different probability distributions for remaining immune. We show that to first approximation the first three moments of the corresponding probability densities are sufficient to well describe oscillatory solutions corresponding to recurrent epidemics. Specifically, increasing the fraction who lose immunity, increasing the mean immunity time, and decreasing the heterogeneity of the population all favor the onset of epidemics and increase their severity. We consider six different distributions, some symmetric about their mean and some asymmetric, and show that by tuning their parameters such that they have equivalent moments that they all exhibit equivalent dynamical behavior.

1. Introduction

A common model for the spread of disease in a population is the SIR model, where the population is divided into susceptible, infectious and recovered compartments/classes. Susceptible individuals become infectious via contact, infectious individuals recover, and recovered individuals maintain immunity (Anderson and May, 1991). The simplest version of the SIR model employs ordinary differential equations (ODEs), because they are a simple modeling tool and are relatively easy to analyze. However, a fundamental assumption of ODEs is that processes are governed by exponential distributions, e.g., the probability of dying due to natural causes at time t after birth is $1-\exp(-\mu t)$, where μ is the death rate. For a birth or death process, an exponential probability may be reasonably appropriate. In contrast, the recovery process may, as a first approximation, be better described by a step distribution where all individuals are infectious for the same fixed time and then all recover. More generally, the time spent in a particular compartment, sometimes referred to as the sojourn time, could be an arbitrary function of time.

In this paper we consider the effect of choosing different probability distributions modeling temporary immunity. That is, upon recovery, individuals leave the infectious class, enter the recovered class, then leave the recovered class either by natural death or due to losing their immunity. When they lose their immunity they reenter the susceptible class. Temporary immunity occurs in diseases such as influenza, cholera, pertussis and malaria. The mechanism may be due either to waning of an individual's immune response, or the disease itself mutating to present a new challenge to the individual's immune system. In Taylor and Carr (2009) we considered the case where everyone was immune for the same time τ such that the SIRS model (recovered individuals become re-susceptible) was a system of delay differential equations (DDEs). More recently, in Carr (2016) we showed that an SIRS model with an arbitrary probability for remaining immune, modeled by integro-differential equations (IDEs), may be approximated by DDEs. We used the case of a linearly decreasing probability as an example in order to carry out a complete bifurcation analysis to predict the amplitude of periodic oscillations that correspond to recurrent epidemics of the disease. In this paper, we consider five different probability distributions (arctan, algebraic, logistic, piecewise linear, shifted exponential) for remaining immune and compare them to the linear case that serves as the reference. We show that by appropriately tuning the parameters of each distribution, we can obtain effectively equivalent results for how the amplitude of oscillations depends on the fraction that become re-susceptible (P_s). More specifically, we tune the zeroth, first and second moments of the different density functions (M_0, M_1 and M_2) such that they generate equal amplitude (and period) epidemics. These results will be demonstrated by numerical simulations of the IDEs.

The population is divided into three compartments representing fractions of a normalized population of size 1: susceptible ($S(t)$), infectious ($I(t)$), and recovered individuals ($R(t)$). We assume that infectious individuals recover with constant rate γ. Once

* Fax: +1 214 768 2355.
E-mail address: tcarr@smu.edu.

http://dx.doi.org/10.1016/j.tpb.2016.11.001
0040-5809/

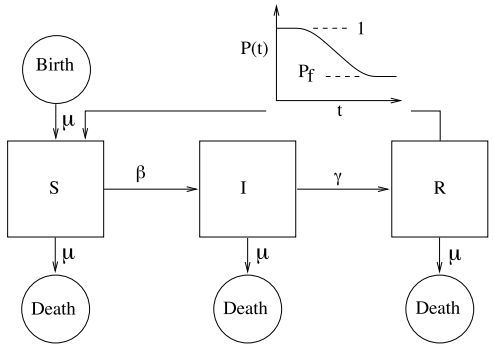

Fig. 1. Flow diagram illustrating SIRS model with an arbitrary probability for temporary immunity.

in the recovered class, they may leave either by natural death with rate μ, or due to losing their immunity. Let $\bar{P}(t)$ be the probability of maintaining immunity; that is, it is the probability that an individual who recovered at $t = 0$ is still recovered and immune at time t ($\bar{P}(0) = 1$ and $\bar{P}(t)$ is decreasing). The SIRS equations are then (Carr, 2016)

$$\begin{aligned}
\frac{dS(t)}{dt} &= \mu[1 - S(t)] - \beta S(t)I(t) \\
&\quad - \int_0^\infty \gamma I(t-r)e^{-\mu r}\frac{d\bar{P}(r)}{dr}dr, \\
\frac{dI(t)}{dt} &= \beta S(t)I(t) - (\mu+\gamma)I(t), \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t) + \int_0^\infty \gamma I(t-r)e^{-\mu r}\frac{d\bar{P}(r)}{dr}dr, \qquad (1)
\end{aligned}$$

where the birth rate in the $S(t)$ class is assumed to be equal to the death rate μ, and transition from the $S(t)$ class to the $I(t)$ class is governed by the mass-action term with transmission rate β. Individuals lose immunity, leave the $R(t)$ class and re-enter the $S(t)$ class (becoming re-susceptible) via the integral terms. The effect of the integral is to sum all previously infectious individuals who have entered the $R(t)$ class up to the present time, decremented by the probability that an individual has not died a natural death, and decremented by the probability that an individual is still immune. Note that the time derivative of $P(t)$ is negative indicating a decreasing probability of remaining in the class; the implicit negative sign means that the integral in the $R(t)$ class represents a individuals exiting the $R(t)$ class, whereas they are entering the $S(t)$ class. The model is illustrated by the flow diagram in Fig. 1. Because the equations for $S(t)$ and $I(t)$ do not depend on $R(t)$, it suffices to consider only those two equations, with $R(t) = 1 - [S(t) + I(t)]$ for a normalized population size.

Introductory descriptions of IDE models for diseases can be found in Brauer and Castillo-Chávez (2001) and Breda et al. (2012). One of the very earliest disease models by Kermack and McKendrick (1927) in 1927 used arbitrary probabilities for the sojourn times between classes. Cooke and Yorke (1973) considered an arbitrary density of recovery times before individuals return directly to the susceptible class. Busenberg and Cooke (1980) used a latency time before becoming infectious instead of introducing an exposed class. Hethcote and Tudor (1980) allowed for densities in both the latency time and the recovery time. Hethcote et al. (1981) showed that both IDE and DDE models require feedback into the susceptible class to support persistent oscillations. Stech and Williams (1981) developed a method to analyze the global stability of the steady states in an SIRS type model with temporary immunity and the case of no natural death ($\mu = 0$). Diekmann and Montijn (1982) and Chow et al. (1985) considered a fixed period of temporary immunity in combination with a generalized probability for the time of infectiousness; they determined conditions for a Hopf bifurcation to periodic oscillations corresponding to recurrent epidemics in the population. Brauer (1990) showed that in a disease with universal fatality the form of the density for disease-related death can change the stability of the endemic state.

Keeling and Grenfell (1997, 1998) examined general distributions for the infectious time. They showed that measles data are more consistent with a density with small variation than with the exponential density. Further, this difference is particularly important when computing the statistics from stochastic simulations; in particular, a density with wide variance is more susceptible to stochastic fadeout and thus underestimates the persistence time of a disease. The results of Stech and Williams (1981) were generalized by Thieme and van den Driessche (1999) for the case of $\mu \neq 0$, while also consider various forms for modeling the recovering process; a very general result of note is that with temporary immunity the endemic state is globally stable if the immunity time is shorter than the recovery time. Feng and Thieme (2000a,b) also generalized the infection process by allowing for both age structure and densities in the infectious class. In Feng and Thieme (2000a) their results are generally related to existence and global stability of solutions. In Feng and Thieme (2000b) they extend their previous work to obtain results on the stability of the endemic state and conditions for a Hopf bifurcation to periodic solutions. Of particular note is that, similar to our work here, they characterize their results in terms of the mean and variance of the density function.

In van den Driessche and Zou (2007) and van den Driessche et al. (2007) van den Driessche et al. consider an IDE model for relapse where recovered individuals return to the infectious class. Wang et al. (2012, 2014) continued the study of relapse phenomena modeled with IDEs, while also considering the effect of heterogeneous population. In both sets of studies relapse to the infectious class did not result in oscillatory solutions.

Lloyd (2001a,b) summarized cases and conditions where ODE models are appropriate and contrasted these to cases where arbitrary densities must be used. As a specific example, they considered the gamma density, which lends itself to detailed analysis because the IDEs reduce to a multistage chain of ODEs for the compartment in question. This approach dates back at least as far as Hethcote et al. (1981), who considered an $SIR_1R_2 \ldots R_n - S$ model for temporary immunity and a delay of the return of individuals to the susceptible class. Thus, rather than solving a delayed system, they explored a system with multiple recovered classes and found that $n \geq 3$ has qualitatively the same dynamics as the delayed system. More recently, Camitz and Svensson (2009) also considered the gamma density in a numerical study and noted how the details of the tails of the density can affect the final results.

As mentioned, an important result of our work is that we show how the stability, amplitude and period of oscillations depend on the first three moments of the probability density for immunity. Hethcote and Tudor (1980) found that the threshold quantity for the disease to be endemic depends on the mean of the probability for remaining infectious. As mentioned above, Feng and Thieme (2000b) considered the effect of the mean and variance of the density function corresponding to individuals remaining infectious. Then in Feng et al. (2007) Feng et al. consider generalized probabilities for remaining in an exposed and infectious and obtain stability results for the steady states in terms of the mean and variance. Arino and van den Driessche (2006), Blyuss and Kyrychko (2010) and Yuan and Bélair (2014) all found that the mean of the density is important in linear stability results and in defining the basic reproduction number. Beyond that, they focus on the simpler cases of exponential, Dirac-δ or gamma densities, where Arino et al. performed a numerical investigation, Blyuss et al. obtained stability results via Lyapunov