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Complete global stability for an SIR epidemic model with delay — Distributed or discrete

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ABSTRACT

SIR models with distributed delay and with discrete delay are studied. The global dynamics are fully determined for $\mathcal{R}_0 > 1$ by using a Lyapunov functional. For each model it is shown that the endemic equilibrium is globally asymptotically stable whenever it exists.

1. Introduction

In modelling the transmission of an infectious disease, a common model structure involves dividing the population into susceptible, infectious and recovered individuals. If the immunity that is obtained upon recovery is permanent, then one gets an SIR model. In this paper we consider SIR models with mass action incidence and constant recruitment. We now review some works on related models.

In [6], an SIR model using ordinary differential equations was given. That model assumed that the infectivity of individuals was constant over the duration of infection. The model exhibits threshold behaviour where the disease dies out if a key parameter $\mathcal{R}_0 \leq 1$ and the disease limits to an endemic equilibrium if $\mathcal{R}_0 > 1$. In [4], a complete analysis of the global dynamics of an ordinary differential equation model with multiple infectious stages was presented, showing the same threshold behaviour.

In [7], an SIR model with a discrete delay was analyzed. The delay was used to model the fact that an individual may not be infectious until some time after becoming infected. This system also exhibits threshold behaviour. The disease dies out for $\mathcal{R}_0 \leq 1$, whereas it is permanent (survives) for $\mathcal{R}_0 > 1$. Additionally, it was shown that if $\mathcal{R}_0 > 1$ and the delay is small enough, then the endemic equilibrium is globally asymptotically stable. The global stability for larger delay remained an open problem. A solution to that problem is given in Section 5 of this paper, where it is shown that the endemic equilibrium is globally asymptotically stable whenever $\mathcal{R}_0 > 1$.

An SIR model with distributed delay was studied in [1,8,10]. The distributed delay allows infectivity to be a function of the duration since infection, up to some maximum duration. Once again, the model displays threshold dynamics. For $\mathcal{R}_0 \leq 1$, the disease dies out. It was shown in [8] that the disease is permanent for $\mathcal{R}_0 > 1$. In [1,10], conditions are found on the model parameters that guarantee that the endemic equilibrium is globally asymptotically stable for $\mathcal{R}_0 > 1$. These conditions would typically hold for small delays but not larger delays. The global stability when these conditions fail remained an open problem. A solution to that problem is given in Section 4 of this paper, where it is shown that the endemic equilibrium is globally asymptotically stable whenever $\mathcal{R}_0 > 1$.

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The global attractivity proofs in this paper use Lyapunov functionals that are similar in nature to the one used in [9] in which the global dynamics are resolved for an SEIR model with infinite delay. In each case, the Lyapunov functional includes an integral over the prior history.

In this paper, an SIR model with distributed delay is given in Section 2. In Section 3, some results from the literature relating to earlier work on this model are given. Section 4 contains a proof of the global asymptotic stability of the endemic equilibrium for $\mathcal{R}_0 > 1$. In Section 5, an SIR model with discrete delay is presented and the endemic equilibrium is shown to be globally asymptotically stable for $\mathcal{R}_0 > 1$.

2. SIR model with distributed delay

A population is divided into three classes: susceptible, infectious and recovered, denoted by s, i and r, respectively. All recruitment is into the susceptible class at a constant rate b. The death rates for the classes are μ_1, μ_2 and μ_3, respectively. The average duration of infectiousness is $1/\lambda$. Infectiousness is assumed to vary over time from the initial time of infection until a duration h has passed; this variation is described by the function f which accounts for the relative infectivity, as well as the survival of individuals infected at earlier times until time t. The mass action coefficient is β and is chosen so that $\int_{\tau=0}^{h} f(\tau)\mathrm{d}\tau = 1$. (It is assumed that f takes on non-zero values arbitrarily close to h, so that the interval of integration is not artificially extended by concluding with an interval for which the integrand is automatically zero.)

The model is given in [10] by the equations

$$\begin{aligned} \frac{\mathrm{d}s(t)}{\mathrm{d}t} &= b - \beta s(t)\int_{\tau=0}^{h} f(\tau)i(t-\tau)\mathrm{d}\tau - \mu_1 s(t) \\ \frac{\mathrm{d}i(t)}{\mathrm{d}t} &= \beta s(t)\int_{\tau=0}^{h} f(\tau)i(t-\tau)\mathrm{d}\tau - (\mu_2+\lambda)i(t) \end{aligned} \tag{2.1}$$

and

$$\frac{\mathrm{d}r(t)}{\mathrm{d}t} = \lambda i(t) - \mu_3 r(t).$$

Since the variable r does not appear in the equations for $\frac{\mathrm{d}s}{\mathrm{d}t}$ and $\frac{\mathrm{d}i}{\mathrm{d}t}$, it is sufficient to analyze the behaviour of solutions to (2.1).

The initial condition for (2.1) is

$$s(0) \in \mathbb{R}_{\geq 0} \quad \text{and} \quad i_0 \in \mathcal{C}([-h, 0], \mathbb{R}_{\geq 0}).$$

Thus, the number of infectives is determined from time $-h$, allowing (2.1) to describe how the variables change for all $t \geq 0$.

3. Previous results

The basic reproduction number [3] for the model is

$$\mathcal{R}_0 = \frac{\beta b}{\mu_1(\mu_2+\lambda)}.$$

For all parameter values, the disease-free equilibrium is given by

$$E_0 = \left(\frac{b}{\mu_1}, 0\right).$$

If $\mathcal{R}_0 \leq 1$, then E_0 is the only equilibrium. If $\mathcal{R}_0 > 1$, then there is also a unique endemic equilibrium

$$E_* = (s^*, i^*) = \left(\frac{\mu_2+\lambda}{\beta}, \frac{b-\mu_1 s^*}{\beta s^*}\right).$$

By noting that $s' > 0$ for $s = 0$ and $i \geq 0$, that $i' \geq -(\mu_2+\lambda)i$ for $s, i \geq 0$ and that $(s+i)' = b - \mu_1 s - (\mu_2+\lambda)i$, one may obtain the following result [8].

Proposition 3.1. *The region*

$$\Omega = \left\{(s, i) \in \mathbb{R}^2_{\geq 0} : s + i \leq \frac{b}{\bar{\mu}}\right\},$$

where $\bar{\mu} = \min\{\mu_1, \mu_2+\lambda\}$, *is positively invariant and is attracting.*

We now state without proof some key results from [2,8,10], which provide the setting in which the main results of this paper (Sections 4 and 5) are to be enjoyed.