



Lyapunov functions for SIR and SIRS epidemic models

Suzanne M. O'Regan^a, Thomas C. Kelly^b, Andrei Korobeinikov^{c,*}, Michael J.A. O'Callaghan^a, Alexei V. Pokrovskii^a

^a Department of Applied Mathematics, Aras na Laoi, University College Cork, Ireland

^b Department of Zoology, Ecology and Plant Science, Distillery Fields, North Mall, University College Cork, Ireland

^c MACSI, Department of Mathematics and Statistics, University of Limerick, Limerick, Ireland

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ABSTRACT

In this paper, we construct a new Lyapunov function for a variety of SIR and SIRS models in epidemiology. Global stability of the endemic equilibrium states of these systems is established.

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In 1892, Russian mathematician Lyapunov developed a method for the analysis of the stability of ordinary differential equations [1]. This method, known as the Direct Lyapunov Method, is one of the most powerful techniques for qualitative analysis of a dynamical system. The method employs an appropriate auxiliary function, called a Lyapunov function. However, the drawback of the method is that finding such a function is usually a non-trivial task.

While the intention of Lyapunov was to study the stability of motion, the direct Lyapunov method and the notion of an auxiliary function have found a wider range of application and Lyapunov functions may be used to achieve a multitude of diverse tasks. For example, this method may be applied to estimate the rate of convergence to a steady state, or the size of a basin of attraction. In addition, it has been employed in proving theorems (e.g., Hopf bifurcation theorem), etc.

The method of Lyapunov functions has been used extensively in mathematical biology [2,3]. In particular, a Lyapunov function for a SIR epidemic model was constructed by Mena-Lorca and Hethcote [4]. Korobeinikov and Wake [5] found a symmetric-in-variables Lyapunov function, which was later extended to a wider range of epidemic models, including models with a larger number of compartments [6–9] and models with non-linear functional responses [10–16]. In this paper, we construct a Lyapunov function for a variety of SIR and SIRS compartment models in epidemiology that principally differ from the above-mentioned functions.

We consider the classical Susceptible–Infected–Recovered (SIR) compartmental epidemic model [17,18]. According to the assumptions of Kermack and McKendrick [19], the population of size N is divided into three distinct classes: the susceptible population of size S , the infectious population of size I and the recovered (or removed) population of size R . When a susceptible individual acquires the disease through contact with an infectious individual, the individual moves into the infective compartment and, subsequently, as a result of recovery or isolation, into the removed class. If the recovered individuals retain their immunity permanently, then they remain in the recovered compartment. The model based on these assumptions is known as the SIR model. It is assumed that all offspring are born healthy and thus are considered members of the S compartment. Assuming that the influx of the newborn into the population is proportional to the population size

* Corresponding author.

E-mail addresses: s.m.oregan@student.ucc.ie (S.M. O'Regan), t.kelly@ucc.ie (T.C. Kelly), andrei.korobeinikov@ul.ie (A. Korobeinikov), mja.ocallaghan@ucc.ie (M.J.A. O'Callaghan), a.pokrovskii@ucc.ie (A.V. Pokrovskii).

N and that the spread of the infection occurs according to the principle of mass action, the model can be described by the system of ordinary differential equations,

$$\begin{aligned}\dot{S} &= bN - \beta \frac{SI}{N} - \omega S, \\ \dot{I} &= \beta \frac{SI}{N} - (\gamma + \omega_I + \omega)I, \\ \dot{R} &= \gamma I - \omega R.\end{aligned}\tag{1}$$

Here, a dot denotes the time derivative, b is the birth or recruitment rate, ω is the natural death rate, ω_I is the disease-associated death rate, γ is the rate of recovery and β is the incidence rate. All these coefficients are strictly positive.

This model may be extended to the case of temporary immunity, when members of the removed class may lose their immunity over time and return to the susceptible class. This extension is called the SIRS model. Furthermore, vertical transmission, that is, the transmission of disease by an infected parent to its offspring, may be incorporated into the SIR model by assuming that a fraction p of the offspring of the infectives are infected at birth and enter the infective compartment [20]. With these assumptions, and denoting the rate of the loss of immunity by α , the model may be represented by the following system of equations:

$$\begin{aligned}\dot{S} &= bN - pbl - \beta \frac{SI}{N} - \omega S + \alpha R, \\ \dot{I} &= pbl + \beta \frac{SI}{N} - (\gamma + \omega_I + \omega)I, \\ \dot{R} &= \gamma I - (\omega + \alpha)R.\end{aligned}\tag{2}$$

Under the assumption that the population size N is constant, each of these systems may be reduced to a two-dimensional system. The equation for $R(t)$ is traditionally omitted; it is easy to see that under the assumption of constant population size, this equation is decoupled from the first and the second equation of system (1). The condition $N = S + I + R = \text{constant}$ may be used to find R . However, while omitting the equation for $R(t)$ appears to be straightforward, it is clear that the equations for $S(t)$ or $I(t)$ may be omitted instead. Omitting the equation for $S(t)$, rather than for $R(t)$, has the advantage of making the resulting two-dimensional system equivalent for both of the SIR and SIRS models, including the models with vertical transmission. This two-dimensional system is given by

$$\begin{aligned}\dot{I} &= (\tilde{\beta}(N - I - R) - \sigma)I, \\ \dot{R} &= \gamma I - \tilde{\omega}R,\end{aligned}$$

where $\tilde{\beta} = \beta/N$, $\sigma = \gamma + \omega_I + \omega - pb$ and $\tilde{\omega} = \omega + \alpha$. Omitting the tildes for simplicity of notation and setting $q = \beta N - \sigma$, we can rewrite this system as

$$\begin{aligned}\dot{I} &= (q - \beta I - \beta R)I, \\ \dot{R} &= \gamma I - \omega R.\end{aligned}\tag{3}$$

Let $Q_* = (I_*, R_*)$ be the positive equilibrium state of system (3), where $I_* = \omega q / \beta(\gamma + \omega)$ and $R_* = \gamma q / \beta(\gamma + \omega)$. It is easy to see that this equilibrium exists when $R_0 = \beta N / \sigma > 1$. At equilibrium, the equalities $q = \beta(I_* + R_*)$ and $\gamma I_* - \omega R_* = 0$ hold. Substituting these expressions into system (3), we can write this system in a skew-symmetric form

$$\begin{aligned}\dot{I} &= I(\beta(I_* - I) + \beta(R_* - R)), \\ \dot{R} &= \omega(R_* - R) - \gamma(I_* - I).\end{aligned}$$

An elegant Lyapunov function may be constructed for system (3). Consider the function

$$U(I, R) = I - I_* \ln I + a(R - R_*)^2,\tag{4}$$

where $a = \beta/2\gamma > 0$. To verify that the function $U(I, R)$ is a Lyapunov function for system (3), we must show that $\dot{U}(I, R) < 0$ for $I, R > 0$ and $(I, R) \neq (I_*, R_*)$. The function (4) satisfies

$$\begin{aligned}\frac{dU(I, R)}{dt} &= \frac{\partial U}{\partial I} \cdot \frac{dI}{dt} + \frac{\partial U}{\partial R} \cdot \frac{dR}{dt} \\ &= \left(1 - \frac{I_*}{I}\right) (\beta I(I_* - I) + \beta(R_* - R)) + 2a(R - R_*)(\gamma(I - I_*) - \omega(R - R_*)) \\ &= -\beta(I - I_*)^2 - \beta(R - R_*)(I - I_*) + 2a\gamma(R - R_*)(I - I_*) - 2a\omega(R - R_*)^2 \\ &= -\beta(I - I_*)^2 - 2a\omega(R - R_*)^2.\end{aligned}$$

Clearly, $dU/dt < 0$ always holds except at the equilibrium Q_* . Furthermore, $U(I, R) \rightarrow \infty$ as $I \rightarrow 0$ or $I \rightarrow \infty$, and $U(I, R) \rightarrow \infty$ as $R \rightarrow 0$ or $R \rightarrow \infty$. Therefore, we may conclude that function (4) is a Lyapunov function for system (3) and that, by the Lyapunov asymptotic stability theorem [1], the equilibrium Q_* is globally asymptotically stable in the positive quadrant, when it exists.

However, if $R_0 < 1$, then $I_* = R_* < 0$ and no positive equilibria exist. In this case, the infection-free equilibrium $Q_0 = (0, 0)$ is the only equilibrium state. It is easy to see that $\dot{I} < 0$ and hence, $I(t) \rightarrow 0$ as $t \rightarrow \infty$. Therefore, for all $R > 0$, $\dot{R} = \gamma I - \omega R < 0$ as well. Hence, $Q_0 = (0, 0)$ is also globally asymptotically stable.

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References

- [1] A.M. Lyapunov, *The General Problem of the Stability of Motion*, Taylor and Francis, London, 1992.
- [2] B.-S. Goh, *Management and Analysis of Biological Populations*, Elsevier Science, Amsterdam, 1980.
- [3] Y. Takeuchi, *Global Dynamical Properties of Lotka–Volterra Systems*, World Scientific, Singapore, 1996.
- [4] J. Mena-Lorca, H.W. Hethcote, Dynamic models of infectious diseases as regulator of population sizes, *J. Math. Biol.* 30 (1992) 693–716.
- [5] A. Korobeinikov, G.C. Wake, Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models, *Appl. Math. Lett.* 15 (2002) 955–960.
- [6] A. Korobeinikov, Lyapunov functions and global properties for SEIR and SEIS models epidemic models, *Math. Med. Biol.* 21 (2004) 75–83.
- [7] H. Guo, M.Y. Li, Global dynamics of a staged progression model for infectious diseases, *Math. Biosci. Eng.* 3 (2006) 513–525.
- [8] A. Korobeinikov, Global properties of basic virus dynamics models, *Bull. Math. Biol.* 66 (2004) 879–883.
- [9] D. Okuonghae, A. Korobeinikov, Dynamics of tuberculosis: The effect of direct observation therapy strategy (DOTS) in Nigeria, *Math. Model. Nat. Phenom.: Epidemiol.* 2 (2007) 113–128.
- [10] P. Georgescu, Y.-H. Hsieh, Global stability for a virus dynamics model with nonlinear incidence of infection and removal, *SIAM J. Appl. Math.* 67 (2006) 337–353.
- [11] A. Korobeinikov, Lyapunov functions and global stability for SIR and SIRS epidemiological models with non-linear transmission, *Bull. Math. Biol.* 68 (2006) 615–626.
- [12] A. Korobeinikov, Global properties of infectious disease models with nonlinear incidence, *Bull. Math. Biol.* 69 (2007) 1871–1886.
- [13] A. Korobeinikov, Global asymptotic properties of virus dynamics models with dose-dependent parasite reproduction and virulence and non-linear incidence rate, *Math. Med. Biol.* 26 (2009) 225–239. doi:10.1093/imammb/dqp006.
- [14] A. Korobeinikov, P.K. Maini, A Lyapunov function and global properties for SIR and SEIR epidemiological models with nonlinear incidence, *Math. Biosci. Eng.* 1 (2004) 57–60.
- [15] A. Korobeinikov, P.K. Maini, Nonlinear incidence and stability of infectious disease models, *Math. Med. Biol.* 22 (2005) 113–128.
- [16] A. Korobeinikov, Stability of ecosystem: Global properties of a general predator–prey model, *Math. Med. Biol. Advance Access published on April 20, 2009*. doi:10.1093/imammb/dqp009.
- [17] R.M. Anderson, R.M. May, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, Oxford, 1992.
- [18] M.J. Keeling, P. Rohani, *Modeling Infectious Diseases in Humans and Animals*, Princeton University Press, Princeton, New Jersey, 2008.
- [19] W.O. Kermack, A.G. McKendrick, A contribution to the mathematical theory of epidemics, *Proc. R. Soc. Lond. Ser. A Math. Phys. Eng. Sci.* 115 (1927) 700–721.
- [20] S. Busenberg, K. Cooke, *Vertically Transmitted Diseases: Models and Dynamics*, Springer-Verlag, Berlin, 1993.