LYAPUNOV FUNCTIONS AND GLOBAL STABILITY FOR SIR, SIRS AND SIS EPIDEMIOLOGICAL MODELS

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Abstract. Lyapunov functions for classical SIR, SIRS and SIS epidemiological models are introduced. Global stability of the endemic equilibrium states of the models is thereby established.

Key words: Direct Lyapunov method, Lyapunov functions, epidemiological models, endemic equilibrium state, global stability.

1. Introduction

Establishing global properties of a dynamical system is generally a nontrivial problem. The most successful approach to the problem is the direct Lyapunov method [1]. However, the method requires an auxiliary function with specific properties, a Lyapunov function, which is not easy to find. In this article we introduce a family of Lyapunov functions for three-compartment epidemiological models, which appear to be also useful for more sophisticated models.

We have to note that the global stability of SIR, SIRS and SIS models which are to be considered in this paper has been already established by applying the classical Poincaré-Bendixson theorem, or by combination of that with the direct Lyapunov method applied on a limited area of the phase space [2], [3], [4], [5]. Periodic orbits are ruled out using the Dulac criteria or a condition of Busenberg and van den Driessche [6]. The direct Lyapunov method enables us to obtain the result straightforwardly. Furthermore, apart from the stability verification, the direct Lyapunov method provides insight into other properties of the system; for example, it allows us to find and compare the rates of convergence toward an equilibrium state for different models and under different conditions.

2. SIR and SIRS Models

Following the classical assumption [5], [7], [8], we divide the entire population of size $N$ into subpopulations of epidemiological significance: the susceptible, the infective and the removed compartments with sizes $S$, $I$ and $R$ respectively, that is $N = S + I + R$. After infection an individual moves from the susceptibles compartment into the infectives compartment and then into the removed compartment as a result of recovery, isolation or death caused by the disease. We assume that recovery implies permanent or temporary acquired immunity; in the latter case there is a return of the removed individuals into the susceptibles compartment. A model based on these assumptions is known as a SIR (acquired immunity is permanent) or a SIRS (acquired immunity is temporary) model [5], [7], [8]. The transfer diagram of the model is in Fig 1.

![Figure 1. Transfer diagram of the SIRS model.](image)

We assume that the population size $N$ is constant, that is deaths are balanced by births. The births are proportional to the population size $N$ with a birth rate $\gamma$. All disease-associated deaths are from the $R$ compartment. The susceptibles and the infectives may also die from causes not connected with the disease with the rate $\sigma \geq 0$. 
An infection can be transmitted through contacts between the infectives and the susceptibles (horizontal transmission) and, for some diseases, from an infective parent to an unborn or newly born offspring (vertical transmission). The horizontal transmission is assumed to occur according to the mass action incidence $\beta \frac{SI}{N}$. Vertical transmission can be incorporated into a model by assuming that a fraction $p$ of the offspring from the infectives are infected at birth and hence a part of birth flux, $p\gamma I$, enters the infective compartment while the remaining births, $\gamma N - p\gamma I$, come to the susceptibles compartment [5] (see Fig. 1).

If an average life expectancy of the susceptibles, an average infective period and an average period of immunity are $1/\sigma$, $1/\delta$ and $1/\alpha$ respectively, then the differential equations are

\[
\dot{S} = (\gamma + \alpha)N - \beta \frac{SI}{N} - (\alpha + p\gamma)I - (\alpha + \sigma)S, \\
\dot{I} = \beta \frac{SI}{N} - (\delta + \sigma - p\gamma)I.
\]

If immunity is permanent, then the average period of immunity $1/\alpha$ is infinite and $\alpha = 0$, so that the SIRS model reduces to the SIR model. We do not need an equation for the removed class $R$, since $N = S + I + R = \text{constant}$.

Many authors postulate $\sigma = \gamma$ to ensure that deaths exactly balance births. However for the majority of etiologically persistent diseases, such as measles, chickenpox and pertussis, the class of susceptibles is composed from mainly younger people, for whom the rate of natural mortality does not necessarily coincide with that of the population as whole. In developed countries, due to comparatively low child mortality, the natural susceptibles mortality rate $\sigma$ is considerably lower than $\gamma$ and can be neglected whereas for developing countries, where child mortality is commonly high, $\sigma$ may exceed $\gamma$. Therefore there is no reason to limit ourselves by the particular case $\sigma = \gamma$. Furthermore, we may assume that the vaccination of the susceptibles is proportional to the susceptible population [5, p. 37]. Then the rate $\sigma$ is the sum of the death rate of susceptibles and of the vaccination rate. In this case $\sigma$ is also not necessarily equal to $\gamma$.

The system (1) has two equilibria: an infection-free equilibrium $E_0 = (S_0, I_0)$, with

\[
S_0 = \left(\frac{\alpha + \gamma}{\alpha + \sigma}\right) N, \quad I_0 = 0,
\]

and an endemic equilibrium $E^* = (S^*, I^*)$, where

\[
S^* = \left(\frac{\alpha + \gamma}{\alpha + \sigma}\right) \frac{N}{R_0}, \quad I^* = \frac{\alpha + \gamma}{\alpha + \delta + \sigma} \left(1 - \frac{1}{R_0}\right) N.
\]

The parameter

\[
R_0 = \frac{\beta(\alpha + \gamma)}{(\alpha + \sigma)(\delta + \sigma - p\gamma)}
\]

is often called the basic reproduction number. The condition $R_0 > 1$ ensures existence of the positive endemic equilibrium state $E^*$. We assume that this condition holds.

For $\alpha, p \neq 0$ the positive quadrant $\mathbb{R}_+^2$ of the $SI$ plane is not an invariant set of the system (1). Indeed, at $S = 0$ we have $\dot{S} < 0$ for all $I > \frac{\alpha + \gamma}{\alpha + p\gamma}N$, and hence the boundary $S = 0$ is penetrable from $\mathbb{R}_+^2$. This deficiency is avoided by the substitution $(S, I) \rightarrow (P, I)$ where $P = S + \frac{\alpha + \gamma}{\beta} N$. In the new variables we have

\[
\dot{P} = \gamma N - \beta \frac{PI}{N} - \sigma P, \quad \dot{I} = \beta \frac{PI}{N} - \delta I,
\]
where \( \tilde{\gamma} = \gamma + \alpha + (\alpha + \sigma)(\alpha + p) \)/\( \beta \), \( \tilde{\delta} = \alpha + \delta + \sigma \) and \( \tilde{\sigma} = \alpha + \sigma \). The phase space of the system (2) is the positive quadrant \( \mathbb{R}^2_+ \) of the PI plane. The system (2) obtained by the shift of the system (1) along the \( S \) axis inherits the global properties of the system (1) and vice versa. When \( \alpha, p = 0 \) (that is for the SIR model with no vertical transmission) the system (2) coincides with (1). In the new variables the endemic equilibrium state \( E^* \) has coordinates

\[
P^* = \frac{\tilde{\gamma}}{\tilde{\delta}} R_0, \quad I^* = \frac{\tilde{\gamma}}{\tilde{\delta}} \left(1 - \frac{1}{R_0}\right) N,
\]

and \( R_0 = \frac{\beta}{\tilde{\delta}} \). It follows from (2) that

\[
(3) \quad \beta \frac{P^* I^*}{N} = \tilde{\gamma} N - \tilde{\sigma} P^* = \tilde{\delta} I^*.
\]

Global properties of the system (2), and hence the system (1) are given by the following Theorem.

**Theorem 1.** The endemic equilibrium state \( E^* \) of the system (2) (and hence that of the system (1)) is globally stable.

![Figure 2. Level curves of the Lyapunov function \( V(P, I) \).](image)

**Proof:** A Lyapunov function

\[
V(P, I) = P^* \left( \frac{P}{P^*} - \ln \frac{P}{P^*} \right) + I^* \left( \frac{I}{I^*} - \ln \frac{I}{I^*} \right)
\]

is defined and continuous for all \( P, I > 0 \) (see Fig. 2) and satisfies

\[
\frac{\partial V}{\partial P} = 1 - \frac{P^*}{P} \quad \text{and} \quad \frac{\partial V}{\partial I} = 1 - \frac{I^*}{I}.
\]

It is easy to see that the endemic equilibrium state \( E^* = (P^*, I^*) \) is the only extremum and the global minimum of the function \( V(P, I) \) in \( \mathbb{R}^2_+ \). In the case of
the system (2), using (3), the function \( V(P, I) \) satisfies
\[
V(P, I) = \gamma N - \beta \frac{PI}{N} - \delta P - \gamma N \frac{P^*}{P} + \beta \frac{P^*}{N} I + \delta P^*
+ \beta \frac{PI}{N} - \delta I - \beta \frac{P^*}{N} + \delta I^*
\]
\[
= \gamma N \left( 1 - \frac{P^*}{P} - \frac{P}{P^*} + 1 \right)
+ \frac{\delta}{\beta} \left( \frac{P}{P^*} + 1 + \frac{P}{P^*} - 1 \right)
\]
\[
= -\gamma N \frac{P^*}{P} \left( 1 - \frac{P}{P^*} \right)^2 \leq 0 \quad \text{for all } P, I \geq 0.
\]

The equality \( \dot{V}(P, I) = 0 \) holds only on the straight line \( P = P^* \). Since the endemic equilibrium state \( E^* \) is the only invariant set of the system (2) on the straight line \( P = P^* \), by the asymptotic stability theorem (see [9, p. 28] or [10, p. 58]) the equilibrium \( E^* \) is globally asymptotically stable. The theorem is proven. \( \square \)

Remark 1. Many authors assume that a “feasible region”
\[
\tilde{\Sigma} = \{(S, I) \in \mathbb{R}^2 | S, I \geq 0; S + I \leq N\}
\]
is the phase space of the system and consider stability in this region only; in this case the term “global stability” implies “asymptotic stability in \( \Sigma \).” However if \( \sigma \neq \gamma \) the “feasible region” does not coincide with a stable invariant set of the system
\[
\Sigma = \{(S, I) \in \mathbb{R}^2 | S, I \geq 0; S + I \leq \frac{\alpha + \gamma}{\alpha + \sigma} N\}.
\]

Furthermore, since the Lyapunov function employed here is global by its nature allowing to prove stability of the system in the whole positive quadrant \( \mathbb{R}_+^2 \), we see no reason to limit ourselves by a region of that.

Remark 2. It follows from the Theorem that when the positive endemic equilibrium state \( E^* \) exists (i.e. when \( R_0 > 1 \)), the infection-free equilibrium \( E_0 \) is an unstable point. Since the \( S \) axis is a stable subspace of this, it is a saddle point.

Also the case \( R_0 \leq 1 \) (when there is no positive endemic equilibrium state) is not particularly interesting, it is easy to prove that in this case the infection-free equilibrium \( E_0 \) is globally stable. It suffices to observe that in this case the derivative of a Lyapunov function
\[
L(P, I) = P_0 \left( \frac{P}{P_0} - \ln \frac{P}{P_0} \right) + I
\]
satisfies
\[
\dot{L}(P, I) = -\gamma \frac{P_0}{P} N \left( 1 - \frac{P}{P_0} \right)^2 - \delta (1 - R_0) I \leq 0 \quad \text{for all } P, I \geq 0.
\]

3. SIS Model

Some infections (e.g. gonorrhoea) do not give rise to acquired immunity in the host. In this case an individual who has recovered from the infection will again be susceptible immediately after recovery. Hence there is no \( R \) class and the population is composed from the susceptibles and the infectives only, i.e. \( N = S + I \). The corresponding model is known as a SIS model [5], [7], [8] which can be regarded as the limiting case of the SIRS model when the average period of immunity \( 1/\alpha \to 0 \).
\[
\begin{align*}
S & \xrightarrow{\gamma (N-p)} S \\
S & \xrightarrow{\beta SI/N} I \\
I & \xrightarrow{\delta I} I \\
I & \xrightarrow{\sigma I} S
\end{align*}
\]

**Figure 3.** Transfer diagram of the SIS model.

Let us consider the SIS model with vertical transmission (see Fig. 3). The differential equations are

\[
\begin{align*}
\dot{S} &= \gamma N - \beta \frac{SI}{N} - p\gamma I + \delta I - \sigma S, \\
\dot{I} &= \beta \frac{SI}{N} - (\delta + \sigma + \epsilon - p\gamma) I,
\end{align*}
\]

where \( \delta \) is the rate of recovery, \( \sigma \) and \( \epsilon \) are the rates of natural and disease-associated mortality and other parameters are the same as for the SIRS system (1). We again assume that the population size \( N \) is constant.

The system has two equilibria: an infection-free equilibrium state \( E_0 = (\frac{\gamma}{N}, 0) \), and an endemic equilibrium state \( E^* = (S^*, I^*) \), with

\[
S^* = \frac{\gamma N}{\sigma + \epsilon} \quad \text{and} \quad I^* = \frac{\gamma}{\sigma + \epsilon} \left( 1 - \frac{1}{R_0} \right) N,
\]

where \( R_0 = \frac{\beta \gamma}{(\delta + \sigma + \epsilon - p\gamma)} \). The positive endemic equilibrium exists if \( R_0 > 1 \). It is easy to see that

\[
\beta \frac{S^* I^*}{N} = \gamma N + (\delta - p\gamma)I^* - \sigma S^* = (\delta + \sigma + \epsilon - p\gamma)I^*.
\]

After a small alteration, the Lyapunov function (4) can be applied to the system (5).

**Theorem 2.** The endemic equilibrium state \( E^* = (S^*, I^*) \) of the system (5) is globally stable.

**Proof.** A Lyapunov function

\[
U(S, I) = S^* \left( \frac{S}{S^*} - \ln \frac{S}{S^*} \right) + \frac{\sigma + \epsilon}{\delta + \sigma + \epsilon - p\gamma} I^* \left( \frac{I}{I^*} - \ln \frac{I}{I^*} \right)
\]

is a modification of the function (4). The endemic equilibrium state \( E^* = (S^*, I^*) \) is the only extremum and the global minimum of the function \( U(S, I) \) in \( \mathbb{R}_+^2 \). In the case of the system (5), using (6), the derivative of the function satisfies

\[
\dot{U}(S, I) = \gamma N - \beta \frac{SI}{N} + (\delta - p\gamma)I - \sigma S
\]

\[
= -\gamma N \frac{S^*}{S} + \beta \frac{S^*}{N} - (\delta - p\gamma) \frac{S^*}{S} I + \sigma S^*
\]

\[
+ \frac{\sigma + \epsilon}{\delta + \sigma + \epsilon - p\gamma} \left( \beta \frac{SI}{N} - \beta \frac{I^* S}{N} \right) - (\sigma + \epsilon) (I - I^*)
\]

\[
= \gamma N \left( 2 - \frac{S}{S^*} - \frac{S^*}{S} \right) + (\delta - p\gamma) I \left( 2 - \frac{S}{S^*} - \frac{S^*}{S} \right)
\]

\[
= - (\gamma N + (\delta - p\gamma)I) \frac{S^*}{S} \left( 1 - \frac{S^*}{S} \right)^2.
\]

That is \( \dot{U}(S, I) \leq 0 \) for all \( S, I \geq 0 \) ensured by \( \delta - p\gamma \geq 0 \). Since \( U(S, I) = 0 \) holds only for \( S = S^* \) and the endemic equilibrium state \( E^* \) is the only invariant set of
the system on the line $S = S^*$, by the asymptotic stability theorem [9], [10] the
equilibrium state $E^*$ is globally asymptotically stable.

Although the case $\delta - p \gamma < 0$ is hardly biologically feasible, the theorem holds
in this case as well. In this case an approach used in the previous sections, that
is the shift of the system to the right, can be applied. After the substitution
$(S, I) \to (P, I)$, where $P = S - \frac{\delta - p \gamma}{\beta} N$, we have

\begin{equation}
\dot{P} = \gamma N - \beta \frac{PI}{N} - \sigma P, \quad \dot{I} = \beta \frac{PI}{N} - (\sigma + \epsilon) I, \tag{7}
\end{equation}

where $\dot{\gamma} = \gamma + \sigma(p \gamma - \delta)/\beta > 0$. The endemic equilibrium state of the system (7) is given by

$$
P^* = \frac{\sigma + \epsilon}{\beta} N, \quad I^* = \frac{\beta \gamma - \sigma(\sigma + \epsilon)}{\beta(\sigma + \epsilon)} N. $$

The Lyapunov function (4) introduced in the previous section can be straightforwardly applied to the system (7). The derivative of the function satisfies

$$
\dot{V}(P, I) = \dot{\gamma} N - \beta \frac{PI}{N} - \sigma P - \gamma N \frac{P^*}{P} + \beta \frac{P^*}{N} I + \sigma P^*
$$

$$
+ \beta \frac{PI}{N} - (\sigma + \epsilon) I - \beta \frac{I^*}{N} P + (\sigma + \epsilon) I^*
$$

$$
= \dot{\gamma} N \left( 2 - \frac{P}{P^*} - \frac{P^*}{P} \right)
$$

$$
= -\dot{\gamma} N \frac{P}{P} \left( 1 - \frac{P}{P^*} \right)^2 \leq 0 \quad \text{for all } P, I \geq 0.
$$

Hence in the case $\delta - p \gamma < 0$ the endemic equilibrium state of the system (7) and consequently that of the system (5) is globally asymptotically stable as well. The theorem is proven. \hfill \square

In conclusion we have to note that a Lyapunov function with a term $I/I^* -
\ln(I/I^*)$ was applied to a particular case of a SIR model with no vertical trans-
mission by J. Mena-Lorca and H. Hethcote [4]. Unfortunately they did not extend
the method to the more general SIR and SIRS models covered in this paper. Also
they did not attempt to use a Lyapunov function which is symmetric with respect
to both $S$ and $I$ variables, as is introduced in this paper and which is simpler,
considerably more efficient, and appeals aesthetically.

The Lyapunov function of the type (4), applied here to the epidemiological mod-
els, is also extremely useful for Lotka-Volterra predator-prey systems [11], [12].
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