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Stability and bifurcation analysis of epidemic models with saturated incidence rates: an application to a nonmonotone incidence rate

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Abstract. We analyze local asymptotic stability of an SIRS epidemic model with a distributed delay. The incidence rate is given by a general saturated function of the number of infective individuals. Our first aim is to find a class of nonmonotone incidence rates such that a unique endemic equilibrium is always asymptotically stable. We establish a characterization for the incidence rate, which shows that nonmonotonicity with delay in the incidence rate is necessary for destabilization of the endemic equilibrium. We further elaborate the stability analysis for a specific incidence rate. Here we improve a stability condition obtained in [Y. Yang and D. Xiao, Influence of latent period and nonlinear incidence rate on the dynamics of SIRS epidemiological models, *Disc. Cont. Dynam. Sys. B* **13** (2010) 195-211], which is illustrated in a suitable parameter plane. Two-parameter plane analysis together with an application of the implicit function theorem facilitates us to obtain an exact stability condition. It is proven that as increasing a parameter, measuring saturation effect, the number of infective individuals at the endemic steady state decreases, while the equilibrium can be unstable via Hopf bifurcation. This can be interpreted as that reducing a contact rate may cause periodic oscillation of the number of infective individuals, thus disease can not be eradicated completely from the host population, though the level of the endemic equilibrium for the infective population decreases. Numerical simulations are performed to illustrate our theoretical results.

Keywords: Epidemic model, nonlinear incidence rate, linearized stability, Hopf bifurcation, delay differential equation.
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1 Introduction

In modeling of disease transmission dynamics an important ingredient is the incidence rate, describing the number of new infective individuals arising in a host population per unit of time. It is often assumed that the incidence rate is proportional to the number of infective and susceptible individuals, thus the bilinear incidence rate is frequently used [11]. The bilinear incidence rate, however, may be insufficient to consider some diseases where, for example, multiple exposures of infection are possible [10, 19, 20]. The authors in the paper [3] introduce an SIR epidemic model with a saturated incidence rate by a system of ordinary differential equations, motivated by a study of the cholera epidemic spread in Bari in 1973. In the model equation, force of infection is given by a nonlinear bounded function of the number of infective individuals, which can be interpreted as saturation or psychological effect in the disease transmission dynamics. In the same paper, the authors analyze stability and solution trajectories in the phase plane and, then, compare the final size of an epidemic to that in the model with the bilinear incidence rate.

Dynamical behavior of epidemic models with a nonlinear incidence rate has been investigated by many authors, see [10, 19, 20, 25, 28] and references therein. For SIRS and SEIRS epidemic models it is shown that periodic oscillation can appear via Hopf bifurcation, if the incidence rate increases "faster" than the bilinear incidence rate [19, 20]. It is also known that some nonlinear incidence rates do not affect the qualitative dynamics of epidemic models. In [14,15] the author considers some epidemic models with a general incidence rate in which the basic reproduction number exactly determines whether the disease free equilibrium or a unique endemic equilibrium is globally asymptotically stable. In [29] an SIRS epidemic model with a nonmonotone incidence rate is formulated. Here force of infection decreases as

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increasing the number of infective individuals for a large number of infective individuals. The authors prove that the endemic equilibrium is globally stable if it exists.

Time delays are introduced in epidemic models to capture a period in the course of infection, e.g. an incubation period of diseases [1,4,27], a constant infectious period [2] and a period of immunity [18], then, delay differential equation naturally arises in the model equations. The threshold type result in [14, 15] is successfully generalized to SIR epidemic models with distributed delay in [8, 21]. See also [12] for the same direction. The authors in [31] aim to extend the stability results in [29] for SIRS epidemic model with a discrete delay, where the saturated incidence rate can be either a monotone type [30] or a nonmonotone type [29] depending on a parameter. The authors analyze stability of an endemic equilibrium and obtain sufficient conditions such that a stable endemic equilibrium becomes unstable via Hopf bifurcation.

Though the importance of nonmonotonicity of the incidence rates is widely discussed [28, 29], qualitative description of dynamical behavior of delayed epidemic models with nonmonotone incidence rates has not been fully understood, due to the mathematical tractability. In [31] the authors obtain sufficient conditions for stability and instability of an endemic equilibrium. The instability condition seems to be difficult to interpret biologically.

In this manuscript, we first aim to detect a class of nonmonotone incidence rates such that the endemic equilibrium is always asymptotically stable. To this aim we consider an SIRS epidemic model with distributed delays. Here the incidece form is not specified, but we assume that the probability of transmission of the disease decreases as increasing the infective population. Analyzing a characteristic equation, we obtain a characterization for the nonmonotonicity such that the endemic equilibrium is always asymptotically stable. We then derive an exact stability condition for a specific incidence rate proposed in [31]. Two-parameter plane analysis together with an application of the implicit function theorem facilitates us to analyze the characteristic equation in detail. It is shown that a parameter, measuring saturation effect, is responsible for destabilization of the endemic equilibrium via Hopf bifurcation, thus the number of infective individuals would fluctuate periodically. This can be interpreted as that, if the nonmonotonicity is due to an intervention policy during an epidemic outbreak as in [28], periodic oscillation is possible by reducing the contact rate (as increasing the saturation level), though the level of the endemic equilibrium decreases.

This paper is organized as follows. In Section 2 we introduce assumptions for the incidence function to characterize crowding effect for a large number of infective individuals. Then we formulate an SIRS epidemic model with distributed delays. In Section 3, we prove unique existence of an endemic equilibrium if and only if the basic reproduction number is greater than one. We derive a characteristic equation for the endemic equilibrium. This section is divided into two subsections. In Section 3.1 we obtain a class of nonmonotone incidence rates such that an endemic equilibrium is asymptotically stable, whenever it exists. Here some known stability results are generalized. In Section 3.2, we introduce a way of using a two-parameter plane for a simplified characteristic equation to obtain instability results. In Section 4, applying the results in Section 3, we improve a stability condition obtained in [31]. With an application of the implicit function theorem we explore the two-parameter plane, introduced in Section 3.2, to find an exact stability condition. It is proven that the endemic equilibrium is destabilized via Hopf bifurcation as increasing a parameter measuring saturation effect. We perform numerical simulations to illustrate our theoretical results. In Section 5 we summarize our results comparing with recent studies. Biological interpretations are also given.

2 Model

We denote by $S(t)$ and $I(t)$ the number of susceptible individuals and the number of infective individuals at time t, respectively. Let $G : \mathbb{R}_+ \to \mathbb{R}_+$. Consider the following incidence rate:

$$\beta S(t) \int_0^h G(I(t-s))d\eta(s), \tag{2.1}$$

where the function $\eta : [0, h] \to \mathbb{R}$ is nondecreasing and has a bounded variation such that

$$\int_0^h d\eta(s) = \eta(h) - \eta(0) = 1.$$

The integral has to be understood as a Riemann-Stieltjes integral, see e.g. Section 6.2 of [24].

For the incidence function we assume that the following holds.

Assumption 2.1.

(1) $G(0) = 0$

(2) $x/G(x)$ *is monotone increasing on* $\mathbb{R}_+ \setminus \{0\}$ *with*

$$\lim_{x\to 0+} \frac{x}{G(x)} = 1. \tag{2.2}$$

The first assumption is a natural requirement as no infection occurs in absence of the infective population. In the second assumption the crowding effect in force of infection is characterized, i.e., the probability of transmission of the disease per unit of time given contact decreases as increasing the number of infective population. The condition (2.2) implies that the force of infection linearly depends on the number of infective individuals, if it is sufficiently small.

In this paper we consider the following SIRS epidemic model with a saturated incidence rate:

$$\frac{dS(t)}{dt} = B - \mu_1 S(t) - \beta S(t) \int_0^h G(I(t-s))d\eta(s) + \delta R(t), \tag{2.3a}$$

$$\frac{dI(t)}{dt} = \beta S(t) \int_0^h G(I(t-s))d\eta(s) - \mu_2 I(t), \tag{2.3b}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \mu_3 R(t), \tag{2.3c}$$

where $S(t)$, $I(t)$ and $R(t)$ denote the numbers of susceptible, infective and recovered individuals at time t, respectively. The positive constant B is the population birth rate and the positive constant μ_1 is the death rate of susceptible population. The nonnegative constant γ is the recovery rate of infective population and the nonnegative constant δ is the rate of loss of immunity to the disease. To save the number of parameters we introduce $\mu_{2,3}$ that are respectively interpreted as

$$\mu_2 = \text{death rate of infective population} + \text{recovery rate } \gamma,$$
$$\mu_3 = \text{death rate of recovered population} + \text{waning immunity rate } \delta.$$

Thus

$$\gamma < \mu_2,\ \delta < \mu_3 \tag{2.4}$$

holds. Here we note that SIRS models studied in [29–31] can be seen as special cases of (2.3) with Assumption 2.1 for the incidence function.

We denote by $C = C([-h, 0], \mathbb{R}^3)$, the Banach space of continuous functions mapping the interval $[-h, 0]$ into $\mathbb{R}^3$ equipped with the sup-norm $\|\phi\| = \sup_{\theta\in[-h,0]} |\phi(\theta)|$ for $\phi \in C$. The nonnegative cone of C is defined as $C_+ := C([-h, 0], \mathbb{R}^3_+)$. Due to the biological interpretation, we consider the initial conditions for (2.3) as follows:

$$(S, I, R)(\theta) = (\phi_1, \phi_2, \phi_3)(\theta),\ \theta \in [-h, 0],$$

where $(\phi_1, \phi_2, \phi_3) \in C_+$. Finally we assume that G is continuously differentiable on $\mathbb{R}_+ \setminus \{0\}$. Since the right hand side of equations (2.3) is locally Lipschitzian on C and a priori bound for solutions is given (see e.g. [7]), it can be shown that (2.3) has a unique positive solution defined on $(0, \infty)$ for each initial function.

3 Linearized stability analysis

We start with introducing a nondimensionalized time $\tilde{t} := \mu_2 t$. We define

$$\tilde{S}(\tilde{t}) := S\left(\frac{\tilde{t}}{\mu_2}\right),\ \tilde{I}(\tilde{t}) := I\left(\frac{\tilde{t}}{\mu_2}\right),\ \tilde{R}(\tilde{t}) := R\left(\frac{\tilde{t}}{\mu_2}\right).$$

Let

$$\tilde{B} := \frac{B}{\mu_2},\ \tilde{\beta} := \frac{\beta}{\mu_2},\ \tilde{\mu}_1 := \frac{\mu_1}{\mu_2},\ \tilde{\mu}_3 := \frac{\mu_3}{\mu_2},\ \tilde{\gamma} := \frac{\gamma}{\mu_2},\ \tilde{\delta} := \frac{\delta}{\mu_2}$$

with noting that $\tilde{h} = \mu_2 h$. Dropping the tilde we obtain

$$\frac{dS(t)}{dt} = B - \mu_1 S(t) - \beta S(t) \int_0^h G(I(t-s))d\eta(s) + \delta R(t), \tag{3.1a}$$

$$\frac{dI(t)}{dt} = \beta S(t) \int_0^h G(I(t-s))d\eta(s) - I(t), \tag{3.1b}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \mu_3 R(t). \tag{3.1c}$$

Condition (2.4) now becomes

$$\gamma < 1,\ \delta < \mu_3. \tag{3.2}$$

In the following we discuss existence of the endemic equilibrium. The basic reproduction number can be defined as

$$R_0 := \frac{\beta B}{\mu_1}. \tag{3.3}$$

For $x \in \mathbb{R}_+ \setminus \{0\}$ we define

$$H(x) := B - \frac{\mu_1 x}{\beta G(x)} - \left(1 - \frac{\gamma\delta}{\mu_3}\right) x. \tag{3.4}$$

We denote by (S^*, I^*, R^*) the endemic equilibrium, where every component is strictly positive, if it exists.

Proposition 3.1. *For* (3.1a)-(3.1c) *a unique endemic equilibrium exists if and only if* $R_0 > 1$ *holds. The second component of the endemic equilibrium,* I^**, is computed as a unique positive root of* $H(x) = 0$*. It holds that*

$$S^* = \frac{I^*}{\beta G(I^*)},\ R^* = \frac{\gamma I^*}{\mu_3}. \tag{3.5}$$

Proof. We provide a similar proof found in [7]. Consider the following equations:

$$0 = B - \mu_1 S - \beta S G(I) + \delta R, \tag{3.6a}$$
$$0 = \beta S G(I) - I, \tag{3.6b}$$
$$0 = \gamma I - \mu_3 R \tag{3.6c}$$

with $(S, I, R) = (S^*, I^*, R^*)$. Let us assume existence of the solution of (3.6). Then, from (3.6b) and (3.6c), the first component S^* and the third component R^* of the endemic equilibrium are respectively given as in (3.5). To compute I^*, by substituting (3.5) into (3.6a), we obtain the equation $H(x) = 0$. Now let us assume that $R_0 > 1$ holds. Then it holds that

$$\lim_{x \to 0+} H(x) = B - \frac{\mu_1}{\beta} = \frac{\mu_1}{\beta}(R_0 - 1) > 0.$$

On the other hand, there exists a sufficiently large constant M such that $H(M) < 0$. Using Assumption 2.1, one can see that H is strictly monotonically decreasing with respect to x. Thus there exists a unique positive root of $H(x) = 0$, which is the second component of the endemic equilibrium. Now it is obvious that if $R_0 \leq 1$ then there is no endemic equilibrium. □

Remark 3.1. *Assumption 2.1-(2) is a sufficient condition for the uniqueness of the endemic equilibrium. Without Assumption 2.1-(2) SIRS model* (3.1) *may admit multiple endemic equilibria, see [13, 19, 25].*

We introduce our main tool, so called the principle of linearized stability, see Chapter VII of [6], to analyze asymptotic stability of the endemic equilibrium.

Proposition 3.2. *The characteristic equation of* (3.1) *for the endemic equilibrium is given as*

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 - \frac{I^* G'(I^*)}{G(I^*)}\left(\lambda^2 + b_1\lambda + b_2\right)\int_0^h e^{-\lambda s} d\eta(s) = 0, \tag{3.7}$$

where

$$\begin{aligned}
a_1 &:= \mu_1 + \beta G(I^*) + 1 + \mu_3,\\
a_2 &:= \mu_3 + (\mu_3 + 1)(\mu_1 + \beta G(I^*)),\\
a_3 &:= (\mu_1 + \beta G(I^*))\mu_3 - \gamma\delta\beta G(I^*),\\
b_1 &:= \mu_1 + \mu_3,\\
b_2 &:= \mu_1\mu_3.
\end{aligned}$$

If all roots of (3.7) *have negative real parts, then the endemic equilibrium is locally exponentially stable. If, on the other hand, there exists a root with positive real part, then the endemic equilibrium is unstable.*

Proof. We define a function as

$$J(x) := \frac{xG'(x)}{G(x)},\ x \in \mathbb{R}_+ \setminus \{0\}. \tag{3.8}$$

Let us define a matrix as

$$M(\lambda) := \begin{pmatrix} -\mu_1 - \beta G(I^*) & -J(I^*)\int_0^h e^{-\lambda s} d\eta(s) & \delta \\ \beta G(I^*) & J(I^*)\int_0^h e^{-\lambda s} d\eta(s) - 1 & 0 \\ 0 & \gamma & -\mu_3 \end{pmatrix}.$$

The characteristic equation of (3.1) for the endemic equilibrium can be given as

$$\det\left(M(\lambda) - \lambda E\right) = 0,$$

where E is the identity matrix of size 3. By the straightforward calculation one obtains (3.7). The statement regarding the stability follows from Theorem 6.8 in Chapter VII of [6]. □

3.1 Stability conditions

First we consider an instantaneous infectious incidence. We assume that

$$\eta(s) = \begin{cases} 0, & s = 0, \\ 1, & s \in (0, h]. \end{cases} \tag{3.9}$$

One obtains a system of ordinary differential equations for (3.1) via

$$\int_0^h G(I(t-s))d\eta(s) = G(I(t)).$$

First we prove the following theorem:

Theorem 3.1. *Let us assume that* $R_0 > 1$ *and* (3.9) *hold. The endemic equilibrium of* (3.1) *is locally asymptotically stable.*

Proof. From (3.9) we have

$$\int_0^h e^{-\lambda s} d\eta(s) = 1.$$

For convenience, we write I instead of I^*. The characteristic equation (3.7) now becomes

$$\lambda^3 + (a_1 - J(I))\,\lambda^2 + (a_2 - J(I)b_1)\,\lambda + (a_3 - J(I)b_2) = 0, \tag{3.10}$$

where we use J defined in (3.8) in the proof of Proposition 3.2. In order to show that all roots of (3.10) have negative real parts, we now apply the Routh-Hurwitz stability criterion, see e.g. Gantmacher [9]. From (2) in Assumption 2.1 one can see that

$$\frac{d}{dx}\left(\frac{x}{G(x)}\right) = \frac{1 - J(x)}{G(x)} \geq 0,\ x \in \mathbb{R}_+ \setminus \{0\},$$

thus $J(x) \leq 1$ holds for $x \in \mathbb{R}_+ \setminus \{0\}$. Then we obtain that

$$a_1 - J(I) > 0,\ a_2 - J(I)b_1 > 0.$$

From (3.2) one has $\mu_3 - \gamma\delta > 0$. It then follows that

$$a_3 - J(I)b_2 = \mu_1\mu_3\,(1 - J(I)) + (\mu_3 - \gamma\delta)\beta G(I) > 0.$$

We have

$$\begin{aligned} &(a_1 - J(I))\,(a_2 - J(I)b_1) - (a_3 - J(I)b_2) \\ =&(a_1 - J(I) - \mu_3)\,(a_2 - J(I)b_1) + \mu_3\,(a_2 - J(I)b_1) - (a_3 - J(I)b_2). \end{aligned}$$

It is easy to see that $a_1 - J(I) - \mu_3 > 0$. We now compute that

$$\begin{aligned} &\mu_3\,(a_2 - J(I)b_1) - (a_3 - J(I)b_2) \\ =&\mu_3^2\,(1 + \mu_1 + \beta G(I) - J(I)) + \mu_3\,(\mu_1 + \beta G(I) - \mu_1 J(I)) \\ &- \mu_3\,(\mu_1 + \beta G(I)) + \gamma\delta\beta G(I) + \mu_1\mu_3 J(I) \\ =&\mu_3^2\,(1 - J(I) + \mu_1 + \beta G(I)) + \gamma\delta\beta G(I) \\ >&0. \end{aligned}$$

Hence all roots have negative real parts. By Proposition 3.2 we obtain the conclusion. □

In the following we analyze the characteristic equation (3.7) for a general η. To facilitate the analysis we introduce the following result.

Lemma 3.1. *For all* $\omega \in \mathbb{R}_+$ *it holds that*

$$\left(\int_0^h \cos(\omega s)d\eta(s)\right)^2 + \left(\int_0^h \sin(\omega s)d\eta(s)\right)^2 \leq 1. \tag{3.11}$$

Proof. Note that the two integrals exist, since the functions $\cos(\omega s)$ and $\sin(\omega s)$ are continuous. Let us fix $\omega \in \mathbb{R}_+$ arbitrarily. We define a function as

$$h(x) := \left(\int_0^h \cos^2(\omega s) d\eta(s)\right) x^2 + 2\left(\int_0^h \cos(\omega s) d\eta(s)\right) x + \int_0^h d\eta(s)$$

for $x \in \mathbb{R}$. Since it holds that

$$h(x) = \int_0^h \left(\cos(\omega s) \cdot x + 1\right)^2 d\eta(s) \geq 0,$$

the equation $h(x) = 0$ has either a single real root or no real roots. Thus

$$\left(\int_0^h \cos(\omega s) d\eta(s)\right)^2 - \int_0^h \cos^2(\omega s) d\eta(s) \int_0^h d\eta(s) \leq 0 \tag{3.12}$$

holds. Similarly one can obtain that

$$\left(\int_0^h \sin(\omega s) d\eta(s)\right)^2 - \int_0^h \sin^2(\omega s) d\eta(s) \int_0^h d\eta(s) \leq 0. \tag{3.13}$$

Then from (3.12) and (3.13) we get (3.11). □

Now we deduce a sufficient stability condition for the endemic equilibrium of (3.1).

Theorem 3.2. *Let $R_0 > 1$. If*

$$\frac{I^* G'(I^*)}{G(I^*)} \geq -1 \tag{3.14}$$

holds, then the endemic equilibrium is locally asymptotically stable.

Proof. For convenience, we write I instead of I^*. First we prove that all roots of (3.7) locate in the left half complex plane when $\gamma\delta = 0$ holds. The characteristic equation (3.7) with $\gamma\delta = 0$ is

$$0 = (\lambda + \mu_3)\left\{(\lambda + \mu_1 + \beta G(I))(\lambda + 1) - J(I)(\lambda + \mu_1)\int_0^h e^{-\lambda s} d\eta(s)\right\}, \tag{3.15}$$

where $J(I) = \frac{IG'(I)}{G(I)}$ as defined in (3.8) in the proof of Proposition 3.2. One can immediately see that (3.15) has a root $\lambda = -\mu_3$. Furthermore, if $J(I) = 0$ then (3.15) has roots $\lambda = -1,\ -(\mu_1 + \beta G(I))$. Let us assume that $J(I) \neq 0$. We suppose that

$$(\lambda + \mu_1 + \beta G(I))(\lambda + 1) - J(I)(\lambda + \mu_1)\int_0^h e^{-\lambda s} d\eta(s) = 0$$

holds for some $\lambda = \kappa + i\omega$ with $(\kappa, \omega) \in \mathbb{R}_+ \times \mathbb{R}$. Then it holds that

$$|J(I)| = \frac{|(\kappa + i\omega + \mu_1 + \beta G(I))(\kappa + i\omega + 1)|}{|(\kappa + i\omega + \mu_1)\int_0^h e^{-(\kappa + i\omega)s} d\eta(s)|}.$$

We obtain the following estimation:

$$\begin{aligned}\left|(\kappa + i\omega + \mu_1)\int_0^h e^{-(\kappa + i\omega)s} d\eta(s)\right| =& |\kappa + i\omega + \mu_1|\left|\int_0^h e^{-\kappa s}\left\{\cos(\omega s) - i\sin(\omega s)\right\} d\eta(s)\right| \\ \leq& |\kappa + i\omega + \mu_1|.\end{aligned}$$

For the last inequality, we use Lemma 3.1 as follows:

$$\begin{aligned}\left|\int_0^h e^{-\kappa s}\left\{\cos(\omega s) - i\sin(\omega s)\right\} d\eta(s)\right| \leq& \left\{\left(\int_0^h \cos(\omega s) d\eta(s)\right)^2 + \left(\int_0^h \sin(\omega s) d\eta(s)\right)^2\right\}^{\frac{1}{2}} \\ \leq& 1.\end{aligned}$$

Thus we get

$$|J(I)| \geq \frac{|\kappa + i\omega + \mu_1 + \beta G(I)|\,|\kappa + i\omega + 1|}{|\kappa + i\omega + \mu_1|} > |\kappa + i\omega + 1| > 1. \tag{3.16}$$

However, Assumption 2.1 together with (3.14) implies that

$$|J(I)| \leq 1, \tag{3.17}$$

which contradicts (3.16). Thus all roots of (3.15) are located in the left half complex plane. Hence, the endemic equilibrium is locally asymptotically stable when $\gamma\delta = 0$. Therefore the endemic equilibrium is locally asymptotically stable for sufficiently small $\gamma\delta > 0$ see, e.g. Lemma 2.8 in Chapter XI in [6]. Next we suppose that there exists either γ or δ such that there exists a purely imaginary root $\lambda = i\omega$, $\omega \in \mathbb{R}_+$. Substituting $\lambda = i\omega$ into (3.7) we obtain the following two equations:

$$-\omega^3 + a_2\omega = J(I)\left\{b_1\omega \int_0^h \cos(\omega s)d\eta(s) + \left(\omega^2 - b_2\right)\int_0^h \sin(\omega s)d\eta(s)\right\}, \tag{3.18}$$

$$-a_1\omega^2 + a_3 = J(I)\left\{b_1\omega \int_0^h \sin(\omega s)d\eta(s) - \left(\omega^2 - b_2\right)\int_0^h \cos(\omega s)d\eta(s)\right\}. \tag{3.19}$$

Using Lemma 3.1 we get

$$(-\omega^3 + a_2\omega)^2 + (-a_1\omega^2 + a_3)^2 \leq (J(I))^2\left\{b_1^2\omega^2 + (\omega^2 - b_2)^2\right\},$$

which is equivalent to

$$0 \geq \omega^6 + c_1\omega^4 + c_2\omega^2 + c_3,$$

where

$$\begin{aligned}
c_1 &:= a_1^2 - 2a_2 - (J(I))^2, \\
c_2 &:= a_2^2 - 2a_1a_3 - (J(I))^2 b_1^2 + 2(J(I))^2 b_2, \\
c_3 &:= a_3^2 - (J(I))^2 b_2^2.
\end{aligned}$$

We show a contradiction by proving positivity of the coefficients c_1, c_2 and c_3. For the presentation we write G for $G(I)$ and J for $J(I)$. We compute that

$$\begin{aligned}
c_1 =& (\mu_1 + \beta G)^2 + (1 + \mu_3)^2 - 2\mu_3 - J^2 \\
=& (\mu_1 + \beta G)^2 + \mu_3^2 + (1 - J^2) \\
>& 0
\end{aligned}$$

and that

$$\begin{aligned}
c_2 =& \mu_3^2 + (\mu_3 + 1)^2 (\mu_1 + \beta G)^2 + 2\mu_3(\mu_3 + 1)(\mu_1 + \beta G) \\
& - 2\{(\mu_1 + \beta G)^2 \mu_3 + (\mu_3 + 1)(\mu_1 + \beta G)\mu_3 - a_1\gamma\delta\beta G\} \\
& - J^2(\mu_1 + \mu_3)^2 + 2J^2\mu_1\mu_3 \\
=& \mu_3^2 + (\mu_1 + \beta G)^2\left(1 + \mu_3^2\right) + 2a_1\gamma\delta\beta G - J^2\left(\mu_1^2 + \mu_3^2\right) \\
=& \left(1 - J^2\right)\left(\mu_1^2 + \mu_3^2\right) + \mu_1^2\mu_3^2 + \left\{2\mu_1\beta G + (\beta G)^2\right\}(1 + \mu_3^2) + 2a_1\gamma\delta\beta G \\
>& 0.
\end{aligned}$$

Since one has that $\mu_3 - \gamma\delta > 0$ from (3.2) and $J \in [-1, 1]$ from (3.17), it holds that

$$\begin{aligned}
c_3 =& (a_3 + Jb_2)(a_3 - Jb_2) \\
=& \{\mu_1\mu_3(1 + J) + (\mu_3 - \gamma\delta)\beta G\}\{\mu_1\mu_3(1 - J) + (\mu_3 - \gamma\delta)\beta G\} \\
>& 0.
\end{aligned}$$

Thus we get a contradiction. Hence, all roots of (3.7) have negative real parts. □

Remark 3.2. *If G is a monotone increasing function then (3.14) follows. Thus SIRS model* (3.1) *with any monotone saturated incidence rate, which satisfies Assumption 2.1, does not admit an unstable endemic equilibrium.*

As an interesting corollary from Theorem 3.2 we introduce the following result. Since the proof is straightforward, we omit it here.

Corollary 3.1. *If*

$$\frac{d}{dx}(xG(x)) \geq 0, \ x \in \mathbb{R}_+ \tag{3.20}$$

then (3.14) follows.

One can easily see that the following nonmonotone incidence function

$$G(x) = \frac{x}{1+\alpha x^2} \tag{3.21}$$

satisfies the condition (3.20) in Corollary 3.1. Thus, with the incidence function given as in (3.21), the endemic equilibrium is asymptotically stable. The result is given in Theorem 3.2 in [31], assuming the same death rate for every compartment.

3.2 Instability analysis

In this section from [5,6] we introduce a way of using a two-parameter plane to obtain instability conditions. We simplify the characteristic equation (3.7) by assuming that

$$\mu_1 = 1,\ \gamma\delta = 0 \tag{3.22}$$

and that for some $\tau \in (0,h)$

$$\eta(s) = \begin{cases} 0, & s \in [0,\tau], \\ 1, & s \in (\tau,h]. \end{cases}$$

Note that now one has a discrete delay as

$$\int_0^h G(I(t-s))d\eta(s) = G(I(t-\tau)).$$

Via the transformation

$$\tilde{\lambda} := \lambda\tau$$

the characteristic equation (3.7) becomes

$$\frac{1}{\tau}\lambda + 1 + \beta G(I^*) - \frac{I^*G'(I^*)}{G(I^*)}e^{-\lambda} = 0, \tag{3.23}$$

where we omit the tilde for convenience.

Consider the prototype equation:

$$\frac{1}{\tau}\lambda + \nu_1 - \nu_2 e^{-\lambda} = 0 \text{ for } (\nu_1,\nu_2) \in \mathbb{R}^2. \tag{3.24}$$

In Chapter XI in [6] the location of the roots of (3.24) is studied using a two-parameter plane, normalizing the parameter τ to 1. The transcendental equation (3.24) has been revisited several times in the literature, see e.g. [5, 17, 26]. We here summarize results from [6] without normalizing the parameter τ. Let us define the intervals as

$$I_0 := (0,\pi)$$

with

$$I_k^- := ((2k-1)\pi, 2k\pi),\ I_k^+ := (2k\pi, (2k+1)\pi)$$

for $k \in \mathbb{N}_+ \setminus \{0\}$. We then define curves that are parametrized by ω in the ν_1-ν_2 parameter plane as

$$C_0 := \left\{ \left(-\frac{\omega\cos\omega}{\tau\sin\omega}, -\frac{\omega}{\tau\sin\omega} \right),\ \omega \in I_0 \right\},$$
$$C_k^{\pm} := \left\{ \left(-\frac{\omega\cos\omega}{\tau\sin\omega}, -\frac{\omega}{\tau\sin\omega} \right),\ \omega \in I_k^{\pm} \right\}$$

for $k \in \mathbb{N}_+ \setminus \{0\}$. With those ingredients the number of roots of (3.24) that are located in the right half complex plane is illustrated as in Figure 3.1. See Chapter XI in [6] for qualitative aspects of the curves such as monotonicity of the order of the curves.

If one sets

$$(\nu_1,\nu_2) = \left(1 + \beta G(I^*), \frac{I^*G'(I^*)}{G(I^*)}\right)$$

in (3.24), then (3.24) becomes the characteristic equation (3.23). Thus if one can plot the point

$$P := \left(1 + \beta G(I^*), \frac{I^*G'(I^*)}{G(I^*)}\right) \tag{3.25}$$

in the ν_1-ν_2 plane, it is possible to deduce the number of roots that have positive real parts, comparing with Figure 3.1. After specifying the incidence function G, we will elaborate (in)stability conditions in terms of parameters in Section 4.

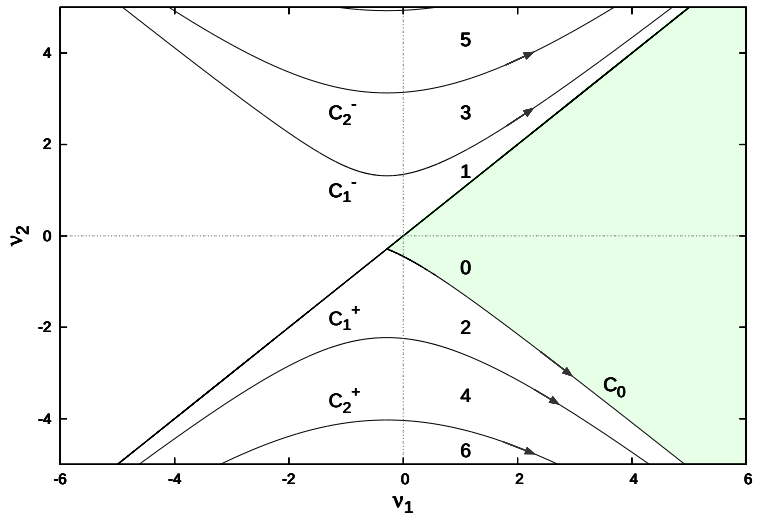


Figure 3.1: Curves and the number of roots of (3.24) that are located in the right half complex plane are illustrated in ν_1-ν_2 parameter plane. The arrows in the curves indicate the direction of increasing ω. The equation (3.24) has 0 as a root in the straight line: $\nu_2 = \nu_1$.

4 Application

We specify the incidence function G as

$$G(x) = \frac{x}{1+\alpha x^p}, \tag{4.26}$$

where $\alpha \in \mathbb{R}_+$ and $p \in \mathbb{R}_+ \setminus \{0\}$. In the context of epidemic models this incidence function was considered in the paper [31], while special cases were proposed in [3, 29]. It is easy to see that G satisfies Assumption 2.1. Let us choose α as a free parameter while we fix other parameters such that $R_0 > 1$ holds.

As in (3.4) the function characterizing the endemic equilibrium is

$$H(\alpha, x) = B - \frac{\mu_1}{\beta}(1+\alpha x^p) - \left(1 - \frac{\gamma\delta}{\mu_3}\right) x \text{ for } \alpha \in \mathbb{R}_+,\ x \in \mathbb{R}_+.$$

By Proposition 3.1 infective population at the endemic equilibrium is uniquely determined for each α as a positive root of $H(\alpha, x) = 0$. We denote it by $I^*(\alpha)$. It holds that

$$H(\alpha, I^*(\alpha)) = 0. \tag{4.27}$$

First, applying Theorem 3.2, we aim to obtain a sufficient stability condition for the endemic equilibrium. One can compute that

$$\frac{I^*(\alpha)G'(I^*(\alpha))}{G(I^*(\alpha))} = 1 - p\frac{\alpha\,(I^*(\alpha))^p}{1+\alpha\,(I^*(\alpha))^p}.$$

In order to assert that (3.14) in Theorem 3.2 holds we introduce the following elementary result.

Proposition 4.1. *For any $p \in \mathbb{R}_+ \setminus \{0\}$ it holds*

$$\frac{d}{d\alpha} I^*(\alpha) < 0,\ \frac{d}{d\alpha}\left\{\alpha\,(I^*(\alpha))^p\right\} > 0,\ \alpha \in \mathbb{R}_+.$$

It follows that

$$\lim_{\alpha\to\infty} I^*(\alpha) = 0,\ \lim_{\alpha\to\infty} \alpha\,(I^*(\alpha))^p = R_0 - 1.$$

Proof. One can compute that

$$\partial_\alpha H(\alpha, x) = -\frac{\mu_1}{\beta} x^p < 0,$$

$$\partial_x H(\alpha, x) = -\frac{\mu_1}{\beta}\alpha p x^{p-1} - \left(1 - \frac{\gamma\delta}{\mu_3}\right) < 0$$

for $\alpha \in \mathbb{R}_+$ and $x \in \mathbb{R}_+ \setminus \{0\}$. By differentiating (4.27) with respect to α one can obtain that

$$\frac{d}{d\alpha} I^*(\alpha) = -\frac{\partial_\alpha H(\alpha, I^*(\alpha))}{\partial_x H(\alpha, I^*(\alpha))} < 0 \text{ for } \alpha \in \mathbb{R}_+, \tag{4.28}$$

hence I^* is a monotone decreasing function with respect to α. Since $I^*(\alpha) > 0$ for $\alpha \in \mathbb{R}_+$, there exists $\underline{I} \geq 0$ such that $\lim_{\alpha\to\infty} I^*(\alpha) = \underline{I}$. To prove that $\underline{I} = 0$ suppose to the contrary that $\underline{I} > 0$ holds. Since it follows $\lim_{\alpha\to\infty} \alpha I^*(\alpha)^p = \infty$, one obtains

$$\lim_{\alpha\to\infty} H(\alpha, I^*(\alpha)) = -\infty,$$

which leads to a contradiction to (4.27). Therefore we get $\underline{I} = 0$, i.e.

$$\lim_{\alpha\to\infty} I^*(\alpha) = 0. \tag{4.29}$$

Next we compute that

$$\frac{d}{d\alpha}\left\{\alpha\left(I^*(\alpha)\right)^p\right\} = \left(I^*(\alpha)\right)^{p-1}\left(I^*(\alpha) + \alpha p\frac{d}{d\alpha}I^*(\alpha)\right).$$

It holds

$$\begin{aligned} I^*(\alpha) + \alpha p\frac{d}{d\alpha}I^*(\alpha) &= \frac{I^*(\alpha)\partial_x H(\alpha, I^*(\alpha)) - \alpha p\partial_\alpha H(\alpha, I^*(\alpha))}{\partial_x H(\alpha, I^*(\alpha))} \\ &= \frac{-1}{\partial_x H(\alpha, I^*(\alpha))}\left(1 - \frac{\gamma\delta}{\mu_3}\right) I^*(\alpha) \\ &> 0. \end{aligned}$$

Thus we get

$$\frac{d}{d\alpha}\left\{\alpha\left(I^*(\alpha)\right)^p\right\} > 0.$$

Since

$$1 + \alpha\left(I^*(\alpha)\right)^p = \frac{\beta}{\mu_1}\left\{B - \left(1 - \frac{\gamma\delta}{\mu_3}\right) I^*(\alpha)\right\} < \infty,\ \alpha \in \mathbb{R}_+$$

holds, $\alpha\left(I^*(\alpha)\right)^p$ is a bounded monotonically increasing function. Using (4.29) finally we obtain that $\lim_{\alpha\to\infty} \alpha\left(I^*(\alpha)\right)^p = R_0 - 1$ holds. □

Then we get the following result.

Theorem 4.1. *Let us assume that $R_0 > 1$ holds. If*

$$p \leq \frac{2}{1 - \frac{1}{R_0}} \tag{4.30}$$

holds, then the endemic equilibrium is locally asymptotically stable.

Proof. We confirm that (3.14) in Theorem 3.2 holds. By Proposition 4.1 it is easy to see that

$$1 - p\frac{\alpha\left(I^*(\alpha)\right)^p}{1 + \alpha\left(I^*(\alpha)\right)^p} > 1 - p\frac{\lim_{\alpha\to\infty}\alpha\left(I^*(\alpha)\right)^p}{1 + \lim_{\alpha\to\infty}\alpha\left(I^*(\alpha)\right)^p} = 1 - p\left(1 - \frac{1}{R_0}\right).$$

Since we have (4.30), we get

$$1 - p\frac{\alpha\left(I^*(\alpha)\right)^p}{1 + \alpha\left(I^*(\alpha)\right)^p} \geq -1, \tag{4.31}$$

thus we obtain the conclusion. □

It is easy to see that

$$\frac{2}{1 - \frac{1}{R_0}} > \lim_{R_0\to\infty}\frac{2}{1 - \frac{1}{R_0}} = 2$$

holds for $R_0 > 1$. Thus the condition $p \leq 2$ obtained in Theorem 3.2 in [31] is indeed a sufficient condition for the stability, see also a remark following Corollary 3.1 in Section 3.1. In Figure 4.2 we visualize the condition (4.30) in (R_0, p) parameter plane.

Next we aim to obtain instability results as introduced in Section 3.2. We assume that (3.22) holds.

Lemma 4.1. *It holds that*

$$1 + \beta G(I^*(\alpha)) = \frac{R_0}{1 + \alpha\left(I^*(\alpha)\right)^p}.$$

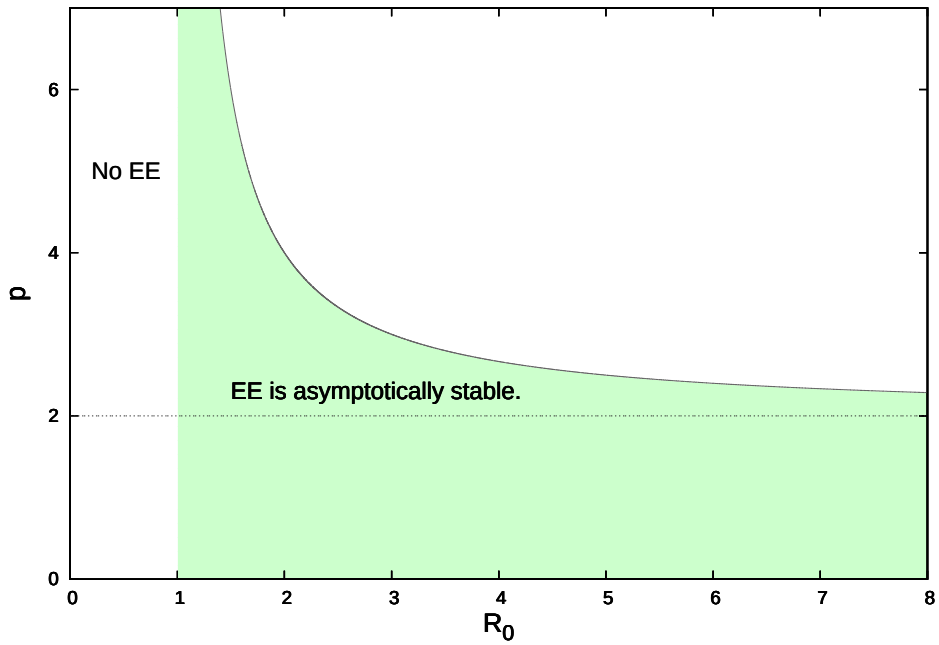


Figure 4.2: Stability region of the endemic equilibrium in (R_0, p) parameter plane. The endemic equilibrium does not exist in the region $R_0 \leq 1$ and is asymptotically stable in the colored region.

Proof. One can compute

$$1 + \beta G(I^*(\alpha)) = \frac{1 + \alpha \left(I^*(\alpha)\right)^p + \beta I^*(\alpha)}{1 + \alpha \left(I^*(\alpha)\right)^p}.$$

From (3.22) we have

$$H(\alpha, x) = B - \frac{1}{\beta}\left(1 + \alpha x^p\right) - x,$$

thus it holds

$$1 + \alpha \left(I^*(\alpha)\right)^p + \beta I^*(\alpha) = B\beta = R_0.$$

Hence we obtain the conclusion. □

In the ν_1-ν_2 parameter plane we track the parametrized path defined as

$$\Gamma := \{P(\alpha),\ \alpha \in \mathbb{R}_+\},$$

where now $P(\alpha)$ can be expressed as

$$P(\alpha) = \left(\frac{R_0}{1 + \alpha \left(I^*(\alpha)\right)^p}, 1 - p\frac{\alpha \left(I^*(\alpha)\right)^p}{1 + \alpha \left(I^*(\alpha)\right)^p}\right).$$

One can easily derive that

$$\lim_{\alpha \to 0+} P(\alpha) = P(0) = (R_0, 1). \tag{4.32}$$

Thus the endemic equilibrium is asymptotically stable for sufficiently small α, since $P(\alpha)$ is in the stability region for small α, see Figure 4.3. We now introduce the following result regarding the shape of Γ. The proof is straightforward from the expression of $P(\alpha)$, thus we omit here.

Lemma 4.2. *The parametrized path* Γ *is a straight line given as*

$$\nu_2 = \frac{p}{R_0}\nu_1 + (1 - p),\ \nu_1 \in (1, R_0].$$

Using the results in Proposition 4.1 we can compute that

$$\lim_{\alpha \to \infty} P(\alpha) = \left(1, 1 - p\left(1 - \frac{1}{R_0}\right)\right). \tag{4.33}$$

Thus the limit point lies in the straight line $\nu_1 = 1$. From Lemma 4.2 both the limit points given in (4.32) and (4.33) are connected by a straight line, see Figure 4.3. Now one can immediately see that if the point $\lim_{\alpha \to \infty} P(\alpha)$ locates

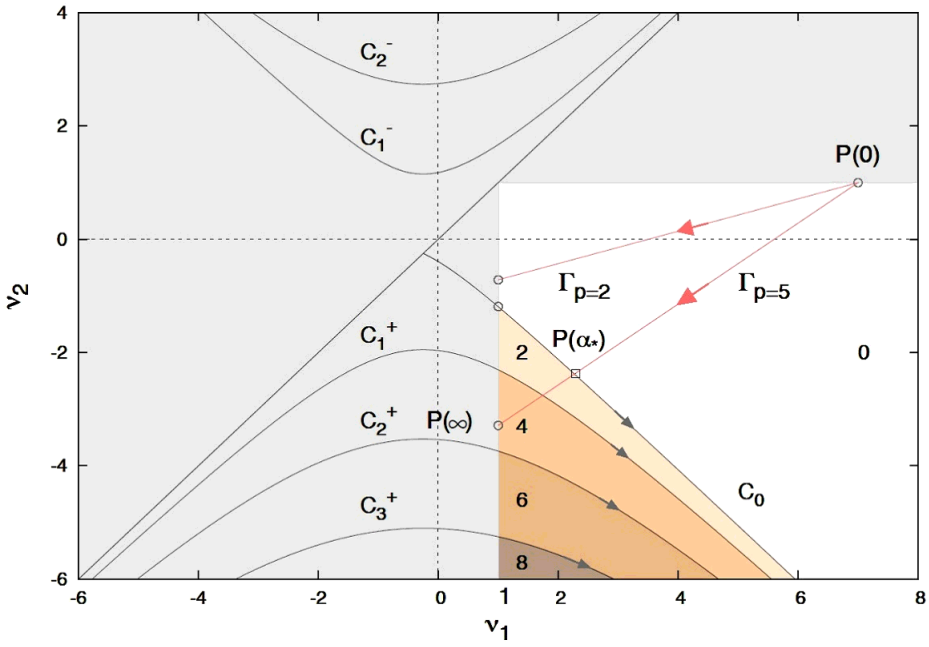


Figure 4.3: Parametrized paths Γ for $p = 2$ and $p = 5$ are illustrated in the ν_1-ν_2 parameter plane where parameters are fixed as $(R_0, \tau) = (7, 4)$. The arrows in paths Γ indicate the direction of increasing α. Both paths start from the point $P(0)$ which is located in the stability region. If $p = 2$ whole path locates at the stability region, thus the equilibrium is asymptotically stable for any α. If $p = 5$, the path intersects with the curve C_0, where the characteristic equation has purely imaginary roots, at $\alpha = \alpha_*$ and enters the instability region. The equilibrium becomes unstable for $\alpha > \alpha_*$ via Hopf bifurcation.

above the intersection of the curve C_0 and the straight line $\nu_1 = 1$, then the equilibrium is asymptotically stable for all α. To obtain the representation of the intersection we define $\omega^*(\tau)$ as a unique solution of

$$-\frac{\omega \cos \omega}{\tau \sin \omega} = 1 \text{ for } \omega \in I_0.$$

One can prove that $\omega^*(\tau) \in \left(\frac{\pi}{2}, \pi\right) \subset I_0$. The intersection of the curve C_0 and the line $\nu_1 = 1$ is given as

$$(\nu_1, \nu_2) = \left(1, \frac{1}{\cos\left(\omega^*\left(\tau\right)\right)}\right), \tag{4.34}$$

where $\cos\left(\omega^*\left(\tau\right)\right) \in (-1, 0)$. Those observations can be summarized as

Theorem 4.2. *The endemic equilibrium is asymptotically stable for any $\alpha \in \mathbb{R}_+$ if and only if*

$$p \leq \frac{1}{1 - \frac{1}{R_0}} \left(1 - \frac{1}{\cos\left(\omega^*\left(\tau\right)\right)}\right)$$

holds.

Remark 4.1. *We note that*

$$2 < 1 - \frac{1}{\cos\left(\omega^*\left(\tau\right)\right)}$$

holds. The stability condition given in (4.30) is sharpened due to the assumption (3.22) which simplifies the characteristic equation. One can also notice that the condition (4.30) is a delay-independent stability condition.

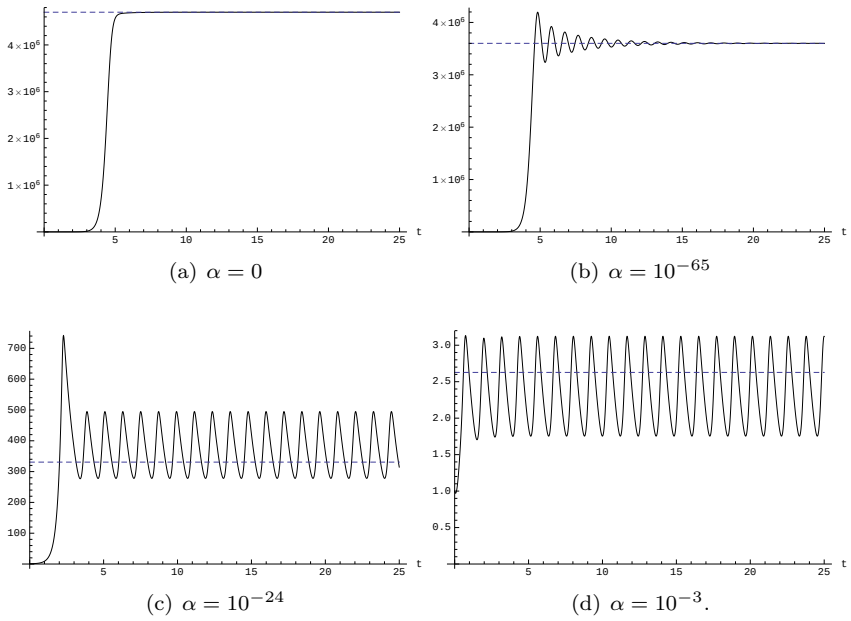
Finally we are interested in the case in which the limit point $\lim_{\alpha \to \infty} P(\alpha)$ locates below the point given in (4.34), i.e.,

$$1 - p\left(1 - \frac{1}{R_0}\right) < \frac{1}{\cos\left(\omega^*\left(\tau\right)\right)}.$$

We define α_* as a root of

$$P(\alpha) = \left(-\frac{\omega \cos \omega}{\tau \sin \omega}, -\frac{\omega}{\tau \sin \omega}\right) \text{ for } \omega \in (\omega^*(\tau), \pi) \subset I_0,$$

characterizing the intersection of the line Γ and the curve C_0. We obtain the following result for instability of the endemic equilibrium via Hopf bifurcation as increasing the parameter α.



(a) $\alpha = 0$ (b) $\alpha = 10^{-65}$

(c) $\alpha = 10^{-24}$ (d) $\alpha = 10^{-3}$.

Figure 4.4: Time development of the number of infective individuals. The dotted line denotes the level of the endemic equilibrium for the infective population. The endemic equilibrium is asymptotically stable for small α. As increasing α, the level of the endemic equilibrium decreases and, then, periodic oscillation can appear. Parameter values are fixed as $B = 5,000,000, \beta = 10^{-5}/3, p = 10$ and $\tau = 0.3$.

Theorem 4.3. *Let us assume that*

$$p > \frac{1}{1 - \frac{1}{R_0}} \left(1 - \frac{1}{\cos\left(\omega^*(\tau)\right)} \right) \tag{4.35}$$

holds. Then there exists α_* such that the endemic equilibrium is asymptotically stable for $\alpha \in (0, \alpha_*)$ and unstable for (α_*, ∞).

We close this section with illustrating a typical solution behavior for the infective compartment. In Figure 4.4 it is shown that the level of the endemic equilibrium (dotted line) decreases as increasing the parameter α, which is analytically proven in Proposition 4.1. One can also see that the endemic equilibrium is asymptotically stable for small α, while, for large α, the endemic equilibrium is unstable, as shown in Theorem 4.3. Our results suggest that, if the nonmonotone incidence rate can be interpreted as an inhibition effect due to an intervention policy [28, 29], one can successfully decrease the level of the endemic equilibrium by reducing the number of contact rate (as increasing the parameter α). The lower endemic equilibrium, however, may become unstable, which induces a periodic oscillation, thus the disease may not be eradicated completely from the host population.

5 Discussion

In this paper we analyze local asymptotic stability of an SIRS epidemic model with a general incidence rate. The class of incidence functions satisfying Assumption 2.1 includes many saturated-type functions, which have appeared in the literature, e.g. [3, 8, 29–31]. Following mathematical models proposed in [1, 4, 27] for vector-borne diseases, we consider the infectious incidence rate (2.1), which is given as a form of distributed delay. In Proposition 3.1 we prove that the model has a unique endemic equilibrium if and only if the basic reproduction number is greater than one. We then derive the characteristic equation (3.7) in Proposition 3.2 to investigate asymptotic stability of the endemic equilibrium. When the infectious incidence is given as an instantaneous term, we prove that the endemic equilibrium is asymptotically stable, see Theorem 3.1. Thus it is clearly shown that any saturated incidence rate does not trigger instability of the equilibrium of the model (2.3) when there is no time delay. If the time delay, caused by a latent period of the infection in the vector [4, 27], is incorporated into the incidence rate, we obtain a sufficient stability condition in Theorem 3.2. The condition (3.14) in Theorem 3.2 is applicable for nonmonotone type incidence functions, see Theorem 4.1 in Section 4 for an example. An important corollary from Theorem 3.2 is that the endemic equilibrium of (2.3) with any monotone saturated incidence rate is asymptotically stable, see also Remark 3.2 in Section 3.1. In Section 4 we consider a specific example (4.26), which is considered in [31], for the nonlinear incidence rate. Since, in general, the equilibrium is not given explicitly, an application of the implicit function theorem in Proposition 4.1 plays a role to proceed the analysis in this section. Applying Theorem 3.2 in Section 3.1, we first offer a sufficient stability condition in

Incidence function	Delay	Stability of the equilibrium
any type	no delay	Asymptotically stable
monotone type	with delay	Asymptotically stable
nonmonotone type	with delay	Possibly unstable

Table 1: For SIRS epidemic model (2.3) with the incidence function satisfying Assumption 2.1, a delayed nonmonotone incidence rate is a necessary modelling ingredient for destabilization of the endemic equilibrium.

terms of biological parameters in Theorem 4.1. We then elaborate the parameter plane analysis introduced in Section 3.2 to obtain instability results. Choosing the patameter α, which characterizes the level of the saturation in the incidence function, as a bifurcation parameter, we tracked the parametrized curve, given by (3.25), in the parameter plane. It is shown that increasing the parameter α can destabilize the equilibrium if p is large enough so that (4.35) holds. One can easily see that the incidence function (4.26) has a nonmonotone shape when (4.35) holds. Therefore, for the SIRS model (2.3) we have proved that nonmonotonicity with time delay in the incidence rate is a necessary modeling ingredient to destabilize the endemic equilibrium, see also Table 1. Stability properties of equilibria have been widely investigated for epidemic models with a nonlinear incidence rate that are formulated as a system of ordinary differential equations, see [10, 13–15, 19, 20, 28, 29] and references therein. In the paper [29] the authors propose a nonmonotone incidence function, namely

$$G(x) = \frac{x}{1 + \alpha x^2}, \tag{5.36}$$

which is a special form of (4.26) with $p = 2$. Nonmonotonicity is used to model inhibition effect during an outbreak of infectious diseases. In Theorem 3.4 in [29] it is proven that the endemic equilibrium is asymptotically stable, see also Theorem 3.2 in [31] for a similar result. Those stability results are generalized in Theorem 3.1 in the present manuscript as the endemic equilibrium is asymptotically stable as long as the incidence function satisfies Assumption 2.1. We here note that the same natural death rate for susceptible, infective and recovered populations is not necessary to be assumed as in those papers cited above to derive the conclusion regarding the stability.

There is a number of papers where a latent period of infection is captured by using delay differential equations [1, 4, 8, 12, 27, 30, 31]. When we take into account of waning immunity, there are some results regarding stability of the endemic equilibrium, see [7, 22, 23, 30]. In Theorem 3.2 it is clearly shown that any monotone incidence rate does not cause destabilization of the endemic equilibrium in SIRS epidemic model (2.3). Corollary 3.1, which is deduced from Theorem 3.2, gives a simple characterization for nonmonotone incidence functions that ensure the stability of the equilibrium. From Corollary 3.1 one can easily obtain the stability result in Theorem 3.2 (ii) in [31]. Figure 4.2 shows stability region of the endemic equilibrium in (R_0, p) parameter plane. One can see that the stability region is wider than the region $p \leq 2$, obtained in [31]. In the same paper [31] the authors give sufficient conditions for destabilization of the endemic equilibrium via Hopf bifurcation. It may be difficult to find a parameter set that satisfies conditions in Theorem 3.2 in [31], thus the role of the saturation effect regarding the destabilization is not clearly exposed there. Here we use a two-parameter plane analysis to clearly show that parameters α and p both have a destabilization role, which may be interpreted as that strong saturation effect cause the destabilization of the equilibrium. We also note that, since the condition (3.22) is assumed in the instability analysis, the cyclic structure of the SIRS model is not necessary for the destabilization of the endemic equilibrium.

The destabilization of the endemic equilibrium seen in Section 4 can be explained by delayed negative feedback, see e.g. [17]. Suppose that there is a large number of infective population. Recall that, if the condition (4.35) holds, the incidence function has a nonmonotone shape with respect to the number of infective individuals in the past time. Thus the infectious incidence rate decreases with a time lag. It turns out that the number of infective population decreases and, then the incidence rate increases. This cycle possibly leads to the destabilization of the equilibrium. Now the condition (3.14) in Theorem 3.2 says that the endemic equilibrium is stable if the slope of the incidence function is "mild" enough, see Figure 5.5. If the nonmonotonicity is interpreted as an intervention policy during an endemic period as in [28], our results suggest that periodic oscillation may arise by reducing the contact rate.

Though in the present paper we have focused on linearized stability analysis of SIRS epidemic model (2.3), obtaining global stability conditions may be an interesting problem. For SIRS epidemic model (2.3) with the incidence function (4.26) one may conjecture that the delay independent stability condition (4.30) in Theorem 4.1 becomes a global stability condition for the endemic equilibrium. However, even for SIRS epidemic model (2.3) with bilinear incidence rate, a "complete" global stability result is not known, see [23] and [22] where the authors attack the problem by Lyapunov functional approach and by monotone iterative method, respectively.

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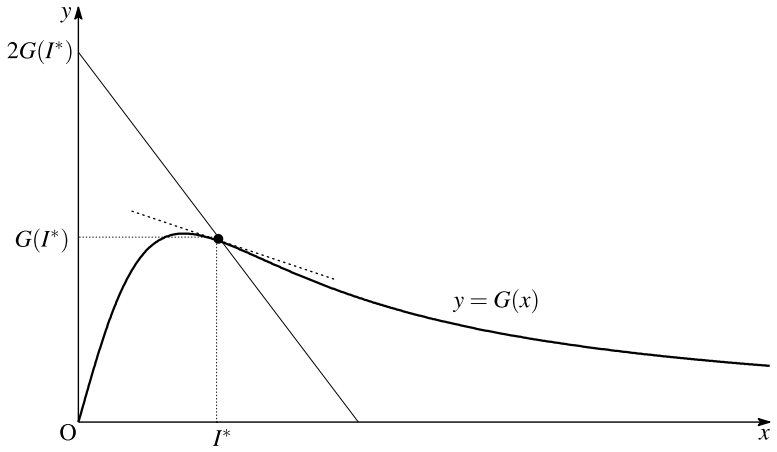

Figure 5.5: The graph of a function $y = G(x)$ that satisfies the condition (3.14) and its tangent line at $x = I^*$ are illustrated. The slope of the tangent line is large compared to the slope of the dotted straight line, where the slope is given as $-G(I^*)/I^*$.

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