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Stability of epidemic models with waning immunity

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Abstract. SIRS type epidemiological model has a fundamental form to study the role of temporal immunity of recovered individuals in disease transmission dynamics and several variant models have been considered in the last century, but up to now dynamical aspects of the model are not fully elucidated. We here look over previous studies concerning qualitative analysis for a family of SIRS type epidemiological models. To this aim we construct a general model in a form of delay equation, a coupled system of a renewal equation and delay differential equations, structuring infected population by infection-age (time elapsed since infection). We re-examine the structure of equilibria and stability of the disease free equilibrium. We then introduce slightly improved stability conditions for an endemic equilibrium. Specifying modelling ingredients we derive two special cases where the model can be represented as a system of ordinary and delay differential equations that have appeared in the literature. For those models we have a powerful tool, namely Lyapunov function, to study global stability of the endemic equilibrium. Relating epidemic models are also discussed.

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§1. Introduction

In the last century with the help of mathematical models extensive studies have been performed for understanding dynamical aspects of transmission of diseases. The mathematical models are typically represented by a system of differential equations forming a dynamical system [18]. The pioneering work in the field of mathematical epidemiology is the paper [43] written by Kermack and McKendrick in 1927. The authors proposed a mathematical model to study a short course of an epidemic in a closed population. Subsequently, in [44] the same authors assume that recovered individuals obtain susceptibility after infection and may re-infect with the disease as the immunity level

decreases. There an endemic equilibrium (a constant positive solution) exists in the population, since recovered population is continuously recruited to susceptible population due to the temporal immunity, differently from the model considered in [43]. The authors consider individual heterogeneity of susceptible, infective and recovered populations in the model and the heterogeneity leads a complex structure in the model. See also [7, 41] where Kermack and McKendrick models are re-formulated in a modern way by a system of partial differential equations [41] and by a scalar delay equation [7].

There are now many variant models called SIRS type epidemic model, but the typical one can be found in [2]. We refer models as SIRS type if individuals in the model change the status as Susceptible→Infective→Recovered→Susceptible. In [2] Anderson and May formulate a mathematical model to fit date of mice population dynamics observed in experimental laboratories. It would be informative for readers to introduce a model in [2]. Let $S(t), I(t)$ and $R(t)$ respectively denote the number of susceptible, infected and recovered population at time t. The population dynamics of each compartment is described by the nonlinear ordinary differential equations:

$$\frac{dS(t)}{dt} = B - \mu S(t) - \beta S(t)I(t) + \delta R(t), \tag{1.1a}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - (\mu + \eta + \gamma)\, I(t), \tag{1.1b}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - (\mu + \delta)\, R(t). \tag{1.1c}$$

Here δ denotes the rate of immunity loss. We refer main text for interpretations of other parameters, see also Figure 2.1 for the model diagram. Qualitative properties of (1.1) and its variant models are investigated in many papers. In [60] (local) stability analysis is performed for several SIRS type epidemic models including (1.1). A positive (an endemic) equilibrium of (1.1) is known to be locally asymptotically stable in [2, 60], however, as far as we know, the global stability of the equilibrium was not analytically shown until that Chen founds a nice Lyapunov function in [2]. In [82] an SIRS type model was formulated by delay differential equations to consider an extension of an SIR model in [72], where recovered individuals do not obtain susceptibility again due to permanent immunity after an infectious period. Recently, McCluskey proves that an endemic equilibrium of the SIR model in [72] is globally asymptotically asymptotically stable [57]. We noticed that it is not straightforward to extend the proof in [57] designed for the SIR model to the SIRS model considered in [82] and some of us puzzled over this problem. We here recapitulate the situation regarding the global stability analysis of the endemic equilibrium of SIRS epidemic models, which will be described in Section 4.

Loss of immunity is considered to be one of the sources causing recurrence

of infectious disease dynamics observed in many epidemics. The authors in [35] explore which mechanisms in a form of SIRS model lead destabilisation of the endemic equilibrium, analysing location of roots of characteristic equations. The authors found that if the immunity period is assumed to be constant then the destabilisation is possible through Hopf bifurcation. A similar model is considered in [50], where the authors obtain a sufficient condition for stability of the endemic equilibrium and then numerically show instability of the endemic equilibrium, see also [6] as a continuation of the work by the same authors. Diekmann and Montijn [23] considered a cyclic type epidemic model by formulating Volterra integral equation, assuming that infected individuals immediately obtain susceptibility to the disease upon recovery (without having the immunity). Assuming that the infectious period of all infected individuals is constant, the authors show that destabilisation of the endemic equilibrium is possible through Hopf bifurcation, whereas the fixed infectious period is not responsible for destabilisation in a framework of SIRS model [35]. How can we obtain a general view for those destabilisation mechanisms?

Structured population model is an ideal framework to consider thousands of epidemiological models. Since in general population consists of heterogeneous individuals, to describe population dynamics behavior at the individual level such as reproduction process is an essential modelling ingredient [61]. Putting assumptions on the individual behavior, e.g. infectious process and recovery process, one usually obtains a rather simple model studied previously in the literature. Analyzing mathematical properties of structured population models, it is also expected to detect mechanisms at the individual level that affect population dynamics. Traditionally, structured population models are formulated by a hyperbolic type partial differential equations [76]. See also [12, 59, 56, 81, 74, 41] and references therein for the use of structured population models in mathematical epidemiology.

In the spirit of Lotka's renewal equation, Diekmann and his collaborators formulated structured population models in terms of Volterra type integral equations, see [21, 22, 32] for detail and references therein. Principle of linearised stability has been recently proven by the perturbation theory of the adjoint semigroups [16, 14]. See applications in [15] for a consumer-resource model and [1] for a cell population dynamical model with quiescent cells.

With a certain distance to the theory of structured population models, delay differential equation is also one of the fields that have rapidly grew in the last century [34, 20]. Delay differential equation is a powerful modelling tool for biological processes and population dynamics [69, 49]. Some mathematical tools designed in the analysis of delay differential equations are recently recognised to be efficient in the analysis of structured population models with a slight modification. For the proof of the linearised stability, the tool used in delay equations [20], so called the suns-stars calculus, are equally efficient to

treat Volterra functional equations describing structured population dynamics [16, 14]. In the paper [56] the authors find a Lyapunov functional, which was formally considered in [57] to analyse an epidemic model by delay differential equations, perfectly works for infection-age-structured epidemic model. In [12], to consider an SIRS model by a system of partial differential equations, the authors reuse Lyapunov functional considered in [66] for an SIRS model by delay differential equations (which is not mentioned in the paper). Of course, both structured population models and delay differential equations form infinite dimensional dynamical systems where many mathematical theories are now available [33, 70].

At this point we would like to once wrap up the state-of-the-arts of the qualitative analysis of SIRS epidemiological model, though the progress is relatively slow compared to SIR epidemic models. In this note one can find that the taste of SIR and SIRS models are rather different in the view of a structured population model. We wish to shed the light on difficulty in the analysis of a class of SIRS type epidemic models. One of the reasons is that if we assume temporal immunity in the model then the characteristic equation suddenly becomes too complicated to draw a concrete conclusion regarding stability of the endemic equilibrium, see Section 3. Furthermore, on the global stability analysis, the approach by Lyapunov functional seems not to be the almighty tool for this kind of cyclic epidemic models. Alternative interpretation of the incidence rate proposed in [72, 9] for a vector-borne disease model, namely $\beta S(t)I(t-\tau)$, is a fruit made when writing this paper. We wish that this note guides readers to explore a possible future direction of the research in this field: stability analysis of epidemic models with waning immunity.

The remainder of the paper is as follows. In Section 2, to set a stage, we formulate a mathematical model with waning immunity as a system of a renewal equation and delay differential equations. This model can be considered as a reformulation of the SIRS model considered in [12] by a system of partial differential equations. We re-examine the structure of equilibria and stability of the disease free equilibrium. In Section 3 we derive the characteristic equation to study local asymptotic stability of equilibria. We provide a new sufficient condition for local stability of an endemic equilibrium, which improves a result obtained in [12]. We further elaborate a special case such that the endemic equilibrium is always locally asymptotically stable. In Section 3.2, as an interlude, we draw a connection between our model and one considered in [23]. The characteristic equation studied in [23] can be derived from our characteristic equation as a special case. In Section 4 we specify modelling ingredients so that the model equation can be expressed as a system of ordinary differential equations and delay differential equations. For those models we present global asymptotic stability results. We then show that a discrete delay model has an equivalent expression as an SEIRS type epidemic model in Section 5. In

Section 6 we discuss other related epidemiological models to close this note.

§2. Epidemic model with waning immunity

To begin the main part of the paper we formulate an epidemic model by *delay equations,* writing down firstly "history" of infected individuals to determine the current population state. The formulation leads to a coupled system of a renewal equation (Volterra integral equation) and delay differential equations. The biological assumption, which we make here, is same as in [12], where the authors slightly extends the model in [56] by assuming that recovered individuals do not have the permanent immunity, thus one may re-infect with the disease.

Consider a closed population with neither immigration nor emigration. Infected individuals are structured by age since infection, which is the time elapsed since the last infection took place. For each infection the clock starts from zero and we do not consider multiple strains in the population. We always refer to age since infection as infection-age and denote it by a. Let us denote by $b(t)$ the incidence rate at time t, which is the number of newly infected individuals per unit of time at time t. We write $\mathcal{F}(a)$ for a probability, for an infected individual, to be in the infectious state until his or her infection-age becomes a. The natural interpretation requires that $\mathcal{F}$ is a decreasing function with $\mathcal{F}(0) = 1$. Since infected individuals of infection-age a at time t experienced the incidence at time $t - a$,

$$a \mapsto b(t-a)\mathcal{F}(a)$$

gives the density function of infected individuals with respect to infection-age at time t. To obtain the population size consisting of infected individuals we integrate the density function (with respect to infection-age) as

$$I(t) := \int_0^\infty b(t-a)\mathcal{F}(a)da = \text{infective population at time } t. \tag{2.1}$$

Infectivity of an infected individual is assumed to depend on the infection-age as in [43], see also [7, 74, 41]. Denote by $\beta(a)$ the age-specific transmission coefficient of infected individuals whose infection-age is a. The force of infection at time t is all contribution from each infected individual towards susceptible population, thus it is given as

$$\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da.$$

We arrive at the renewal equation

$$b(t) = S(t)\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da,$$

where $S(t)$ denotes the number of susceptible individuals at time t.

We shall add more information for the probability $\mathcal{F}$. Assume that infected individuals leave the infectious class due to either the mortality or recovery from the infection. The mortality rate could be decomposed into the natural mortality rate μ and the disease-related mortality rate $\eta(a)$, which is assumed to depend on individual's infection-age. Let $\gamma(a)$ be the recovery rate of infected individuals whose infection-age is a. With those rates one can express the probability

$$\mathcal{F}(a) = e^{-\int_0^a (\gamma(s)+\eta(s))ds - \mu a}. \tag{2.2}$$

Often the dynamics of infected individuals is formulated by the first order partial differential equation with a nonlocal boundary condition, see also [12, 59, 56, 81, 74]. Denote by $i(t,a)$ the density of infected individuals at time t with respect to infection-age a. Then the removing process from the infective class is described as

$$\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right) i(t,a) = -\left(\mu + \gamma(a) + \eta(a)\right) i(t,a), \ a > 0,$$

supplemented by the boundary condition:

$$i(t,0) = S(t) \int_0^\infty \beta(a) i(t,a) da.$$

We denote by $R(t)$ the recovered population at time t. It is assumed that recovered individuals get susceptibility to the disease again at a rate δ. Then

$$\frac{dR(t)}{dt} = \int_0^\infty \gamma(a) b(t-a) \mathcal{F}(a) da - (\mu + \delta) R(t).$$

Note that the first term in the right hand side is the number of newly recovered individuals per unit of time at time t. Considering a demographic process we arrive at the following model:

$$\frac{dS(t)}{dt} = B - \mu S(t) - S(t) \int_0^\infty \beta(a) b(t-a) \mathcal{F}(a) da + \delta R(t), \tag{2.3a}$$

$$b(t) = S(t) \int_0^\infty \beta(a) b(t-a) \mathcal{F}(a) da, \tag{2.3b}$$

$$\frac{dR(t)}{dt} = \int_0^\infty \gamma(a) b(t-a) \mathcal{F}(a) da - (\mu + \delta) R(t). \tag{2.3c}$$

See also Figure 2.1 for a schematic representation of the model equation and Table 1 for the parameter description. One may skip to Section 4 to see how this system (2.3) is related to the system of ordinary differential equations introduced as in (1.1).

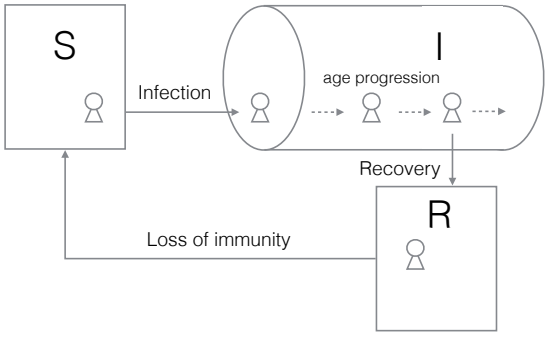

Figure 2.1: Transition of individual's state without a demographic process

In [7] the authors proposed the formulation of epidemic models in terms of force of infection as a primary unknown dynamical variable. This can be done by introducing a notation Λ by

$$\Lambda(t) := \int_0^\infty \beta(a) b(t-a) \mathcal{F}(a) da.$$

Then one has $b(t) = S(t)\Lambda(t)$. Substituting this into the definition we get a nonlinear renewal equation for the force of infection $\Lambda(t)$ as

$$\Lambda(t) = \int_0^\infty \beta(a) S(t-a) \Lambda(t-a) \mathcal{F}(a) da.$$

Thus one can easily switch to the formulation in terms of force of infection. We here stick to (2.3).

2.1. Analytical setting

For $\rho > 0$ we denote by $L^1_\rho(\mathbb{R}_-; \mathbb{R})$ the space consists of all equivalence classes of measurable functions $\phi : \mathbb{R}_- \to \mathbb{R}$ such that the weighted integral with respect to the function $a \mapsto e^{-\rho a},\ a \in \mathbb{R}_+$ is finite i.e.,

$$\|\phi\|_{L^1_\rho} = \int_0^\infty e^{-\rho a} \left|\phi(-a)\right| da < \infty.$$

We define

$$X_+ := \mathbb{R}_+ \times L^1_\rho(\mathbb{R}_-; \mathbb{R}_+) \times \mathbb{R}_+.$$

System (2.3) is supplemented by the initial conditions:

$$(S(0), b(\theta), R(0)) = (s, \phi(\theta), r)\,,\ \theta \in \mathbb{R}_- \tag{2.4}$$

with $(s, \phi, r) \in X_+$, where X_+ is the biologically relevant space. We now make

Table 1: Description of symbols and model ingredients

Symbols	Description
$S(t)$	susceptible population at time t
$b(t)$	newly infected individuals per unit of time at time t
$R(t)$	recovered population at time t
$N(t)$	total population at time t
a	infection-age (the time elapsed since the last infection)
$\beta(a)$	age-specific transmission coefficient
$\mathcal{F}(a)$	probability that infected individuals do not recover until infection-age a
$\gamma(a)$	age-specific recovery rate
$\eta(a)$	age-specific disease-related mortality rate
μ	natural mortality rate
B	population birth rate
δ	waning immunity rate

Assumption 2.1. *For (2.3) it holds*

- $\beta, \gamma, \eta \in L^\infty(\mathbb{R}_+; \mathbb{R}_+)$.
- $B, \mu > 0$ *and* $\delta \geq 0$.

Let us denote by $N(t)$ the total population at time t consisting of susceptible, infected and recovered populations at time t, i.e.,

$$N(t) = S(t) + I(t) + R(t).$$

At the initial time $t = 0$ one can compute the total population as

$$N(0) = s + \int_0^\infty \phi(-a)\mathcal{F}(a)da + r.$$

For (2.3) we specify ρ as $\rho = \frac{1}{2}\mu$. From the expression of $\mathcal{F}$ in (2.2) one can see that

$$\int_0^\infty \phi(-a)\mathcal{F}(a)da = \int_0^\infty \phi(-a)e^{-\rho a}e^{\rho a}\mathcal{F}(a)da \leq \|\phi\|_{L^1_\rho},$$

thus $N(0)$ is bounded above. Similarly, it can be shown that the right hand side of (2.3) is well defined in X_+.

We introduce a standard notation from the theory of functional differential equations [34]

$$b_t : \mathbb{R}_- \to \mathbb{R},$$

defined via the relation $b_t(\theta) = b(t+\theta),\ \theta \in \mathbb{R}_-$. Let $F : X_+ \to \mathbb{R}^3$ with

$$F(s,\phi,r) := \begin{pmatrix} B - \mu s - s\int_0^\infty \beta(a)\phi(-a)\mathcal{F}(a)da + \delta r \\ s\int_0^\infty \beta(a)\phi(-a)\mathcal{F}(a)da \\ \int_0^\infty \gamma(a)\phi(-a)\mathcal{F}(a)da - (\mu+\delta)r \end{pmatrix}. \tag{2.5}$$

Then (2.3) can be written as an abstract form:

$$\begin{pmatrix} \frac{d}{dt}S(t) \\ b(t) \\ \frac{d}{dt}R(t) \end{pmatrix} = F(S(t), b_t, R(t)),$$

For the existence and uniqueness of the solution, similar proofs used in [81, 24] are applicable. First, one can show that F satisfies the Lipschitz condition on each bounded subset of X_+, from which the local existence of solutions of (2.3) follows by the standard contraction argument. One can also show that $S(t), b(t), R(t) \geq 0$ as long as the solution exists. Subsequently, a priori bound for the solution can be given, see also Proposition 2.1 below. The boundedness ensures the standard continuation argument to show that (2.3) has a unique positive solution defined on $(0,\infty)$.

To show that the total population is indeed bounded we let

$$\mathsf{W} := \left\{ (S,\varphi,R) \in X_+ : S + \int_0^\infty \varphi(-a)\mathcal{F}(a)da + R \leq \max\left\{ \frac{B}{\mu}, N(0) \right\} \right\}.$$

Proposition 2.1. *It holds that*

$$(S(t), b_t, R(t)) \in \mathsf{W},\ t > 0.$$

Proof. First we show that

$$N(t) = S(t) + \int_0^\infty b_t(-a)\mathcal{F}(a)da + R(t) \leq \max\left\{ \frac{B}{\mu}, N(0) \right\}. \tag{2.6}$$

Since one has $\int_0^\infty b(t-a)\mathcal{F}(a)da = \int_{-\infty}^t b(s)\mathcal{F}(t-s)ds$, it follows

$$\begin{aligned} &\frac{d}{dt}\int_0^\infty b(t-a)\mathcal{F}(a)da \\ =&b(t) - \int_0^\infty (\mu + \gamma(a) + \eta(a))\, b(t-a)\mathcal{F}(a)da \\ =&S(t)\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da - \int_0^\infty (\mu+\gamma(a)+\eta(a))\, b(t-a)\mathcal{F}(a)da. \end{aligned} \tag{2.7}$$

Then

$$\frac{d}{dt}N(t) = B - \mu N(t) - \int_0^\infty \eta(a)b(t-a)\mathcal{F}(a)da \le B - \mu N(t).$$

From the standard comparison theorem we obtain (2.6). To show that, for each t, b_t is an element of $L^1(\mathbb{R}_-;\mathbb{R}_+)$ we verify that $\int_0^\infty b_t(-a)e^{-\rho a}da < \infty$. Fixing t we compute

$$\begin{aligned}\int_0^\infty b_t(-a)e^{-\rho a}da &= \int_0^t b(t-a)e^{-\rho a}da + \int_0^\infty \phi(-a)e^{-\rho(t+a)}da \\ &= \int_0^t b(t-a)e^{-\rho a}da + \|\phi\|_{L^1_\rho} e^{-\rho t}.\end{aligned}$$

Since we have (2.6) one can see that

$$S(t) \le L,\ \int_0^\infty b(t-a)\mathcal{F}(a)da \le L,$$

where $L := \max\left\{\frac{B}{\mu}, N(0)\right\}$. Then from (2.3b) one can obtain an estimation: $b(t) \le \|\beta\|_{L^\infty} L^2$. Therefore we get $\int_0^\infty b_t(-a)e^{-\rho a}da < \infty$. □

2.2. The basic reproduction number and existence of an endemic equilibrium

To proceed the analysis we reduce the number of parameters. Define

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

Let

$$\tilde{B} := \frac{B}{\mu},\ \tilde{\delta} := \frac{\delta}{\mu},\ \tilde{\beta}(a) := \frac{\beta(\frac{a}{\mu})}{\mu},\ \tilde{\gamma}(a) := \frac{\gamma(\frac{a}{\mu})}{\mu},\ \tilde{\eta}(a) := \frac{\eta(\frac{a}{\mu})}{\mu}.$$

Dropping the tilde we obtain

$$\frac{dS(t)}{dt} = B - S(t) - S(t)\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da + \delta R(t), \tag{2.8a}$$

$$b(t) = S(t)\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da, \tag{2.8b}$$

$$\frac{dR(t)}{dt} = \int_0^\infty \gamma(a)b(t-a)\mathcal{F}(a)da - (1+\delta)R(t), \tag{2.8c}$$

where $\mathcal{F}(a) = e^{-a-\int_0^a(\eta(s)+\gamma(s))ds}$. In the rest of the paper we consider (2.8).

The basic reproduction number is the most important quantity in mathematical epidemiology, which denotes the expected numbers of secondary infective individuals produced by a typical infective individual during an entire infectious period, in a completely susceptible population. In general the basic reproduction number is given as the dominant eigenvalue of a positive linear operator [19]. See also [42] for more general definition that is applicable for population growth in time-heterogeneous environment. For (2.8) it is computed as

$$R_0 := B\int_0^\infty \beta(a)\mathcal{F}(a)da. \tag{2.9}$$

We write $\hat{b}$ for the element of $L^1_\rho(\mathbb{R}_-;\mathbb{R}_+)$ satisfying $b(\theta) = b$ for all $\theta \in \mathbb{R}_-$ (except for a set of measure zero). For (2.8) one can see that there exists the disease free equilibrium expressed as

$$(B, \hat{0}, 0) \in X_+.$$

Let us denote by $(S, \hat{b}, R) \in X_+$ the endemic equilibrium with $(S, b, R) \in \mathrm{int}\mathbb{R}^3_+$. In order to find the endemic equilibrium we consider the following equations:

$$0 = B - S - Sb\int_0^\infty \beta(a)\mathcal{F}(a)da + \delta R, \tag{2.10a}$$

$$b = Sb\int_0^\infty \beta(a)\mathcal{F}(a)da, \tag{2.10b}$$

$$0 = b\int_0^\infty \gamma(a)\mathcal{F}(a)da - (1+\delta)\,R \tag{2.10c}$$

with $(S, b, R) \in \mathrm{int}\mathbb{R}^3_+$.

Proposition 2.2. *For* (2.8) *a unique endemic equilibrium exists if and only if* $R_0 > 1$ *holds. Components of the endemic equilibrium are identified as*

$$S = \frac{1}{\int_0^\infty \beta(a)\mathcal{F}(a)da}, \tag{2.11a}$$

$$b = \frac{1+\delta}{1+\delta\int_0^\infty (1+\eta(a))\,\mathcal{F}(a)da}\left(B - \frac{1}{\int_0^\infty \beta(a)\mathcal{F}(a)da}\right), \tag{2.11b}$$

$$R = \frac{\int_0^\infty \gamma(a)\mathcal{F}(a)da}{1+\delta\int_0^\infty (1+\eta(a))\,\mathcal{F}(a)da}\left(B - \frac{1}{\int_0^\infty \beta(a)\mathcal{F}(a)da}\right). \tag{2.11c}$$

Proof. Assume existence of the solution of (2.10). First, from (2.10b), the first component is given as in (2.11a). From (2.10c) one obtains

$$R = \frac{1}{1+\delta} b \int_0^\infty \gamma(a)\mathcal{F}(a)da. \tag{2.12}$$

From (2.10b) and (2.12) the first equation (2.10a) is

$$0 = B - S - b + \frac{\delta}{1+\delta} b \int_0^\infty \gamma(a)\mathcal{F}(a)da.$$

Using the integration by parts, we get

$$\begin{aligned} 1 - \frac{\delta}{1+\delta}\int_0^\infty \gamma(a)\mathcal{F}(a)da &= 1 - \frac{\delta}{1+\delta}\left(1 - \int_0^\infty (1+\eta(a))\,\mathcal{F}(a)da\right) \\ &= \frac{1+\delta\int_0^\infty (1+\eta(a))\,\mathcal{F}(a)da}{1+\delta}. \end{aligned}$$

Therefore (2.11b) follows. Finally (2.11c) follows from (2.12) with (2.11b). Then one can easily see that $R_0 > 1$ if and only if $(S, b, R) \in \mathrm{int}\mathbb{R}^3_+$. □

§3. The characteristic equation and linearised stability analysis

We compute the characteristic equation to analyse stability of equilibria of (2.8) by investigating the location of roots of the characteristic equation. We here apply the principle of linearised stability recently established in [16, 14], by the theory of perturbed adjoint semigroups, for systems consisting of Volterra functional equations and delay differential equations.

Proposition 3.1. *For an equilibrium* $(S, \hat{b}, R) \in X_+$ *of* (2.8) *the characteristic equation is given as*

$$\begin{aligned} 0 =& (1+\delta+\lambda)\left\{-(1+J+\lambda) + (1+\lambda)\,S\int_0^\infty \beta(a)e^{-\lambda a}\mathcal{F}(a)da\right\} \\ &+ \delta J\int_0^\infty \gamma(a)e^{-\lambda a}\mathcal{F}(a)da, \end{aligned} \tag{3.1}$$

where

$$J := b\int_0^\infty \beta(a)\mathcal{F}(a)da. \tag{3.2}$$

If all roots of (3.1) *have negative real parts, then the equilibrium is exponentially stable. If, on the other hand, there exists a root with positive real part, then the equilibrium is unstable.*

Proof. To derive the characteristic equation we linearise (2.8) about an equilibrium, see Section 5 in [14]. Redefining F in (2.5) with $\mu = 1$, we compute the Frechét derivative of $F : X_+ \to \mathbb{R}^3$ evaluated at an equilibrium $(S, \hat{b}, R) \in X_+$:

$$DF(S,\hat{b},R)\begin{pmatrix} s \\ \phi \\ r \end{pmatrix} = \begin{pmatrix} -(1+J)\,s - S\int_0^\infty \beta(a)\phi(-a)\mathcal{F}(a)da + \delta r \\ Js + S\int_0^\infty \beta(a)\phi(-a)\mathcal{F}(a)da \\ \int_0^\infty \gamma(a)\phi(-a)\mathcal{F}(a)da - (1+\delta)\,r \end{pmatrix},$$

where J is defined as in (3.2). Note that $DF(S, \hat{b}, R) : X_+ \to \mathbb{R}^3$. The characteristic equation is given as

$$\det\left(M(\lambda) - \begin{pmatrix} \lambda & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & \lambda \end{pmatrix}\right) = 0,$$

with

$$M(\lambda) := \begin{pmatrix} -(1+J) & -S\int_0^\infty \beta(a)e^{-\lambda a}\mathcal{F}(a)da & \delta \\ J & S\int_0^\infty \beta(a)e^{-\lambda a}\mathcal{F}(a)da & 0 \\ 0 & \int_0^\infty \gamma(a)e^{-\lambda a}\mathcal{F}(a)da & -(1+\delta) \end{pmatrix}.$$

By the straightforward calculation one obtains (3.1). The statement regarding the stability follows combining Theorems 3.15 and 4.7 in [14], see again Section 5 in the same paper. □

3.1. Stability of the disease free equilibrium

First we show that the basic reproduction number is the threshold parameter for (in)stability of the disease free equilibrium. Indeed the disease free equilibrium is globally asymptotically stable if $R_0 < 1$ as shown in [12].

Theorem 3.2. *Let us assume that $R_0 < 1$ holds then the disease free equilibrium is globally asymptotically stable. If $R_0 > 1$ holds then it is unstable.*

Proof. For the disease free equilibrium $(S, \hat{b}, R) = (B, \hat{0}, 0)$ the characteristic equation (3.1) is written as

$$g(\lambda) = 1, \tag{3.3}$$

where we define

$$g(\lambda) := B\int_0^\infty \beta(a)e^{-\lambda a}\mathcal{F}(a)da.$$

One can see that g is a monotonically decreasing function with respect to $\lambda \in \mathbb{R}$ and that $g(0) = R_0$. Thus there exists a negative real root if $R_0 < 1$ whereas

there exists a positive real root if $R_0 > 1$. Let us show that there is no root with positive real part when $R_0 < 1$. For $R_0 < 1$ suppose that $\lambda = \kappa + i\omega$, where $(\kappa, \omega) \in \mathbb{R}^2$ with $\kappa > 0$ solves the equation (3.3). We then obtain

$$B \int_0^\infty \beta(a) e^{-\kappa a} \cos(\omega a) \mathcal{F}(a) da = 1.$$

One can easily estimate the left hand side to get

$$\left| B \int_0^\infty \beta(a) e^{-\kappa a} \cos(\omega a) \mathcal{F}(a) da \right| < B \int_0^\infty \beta(a) \mathcal{F}(a) da = R_0 < 1,$$

which leads a contradiction. Thus there exists no root with positive real part. Consequently the disease free equilibrium is asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$. We now prove global attractivity for $R_0 < 1$. Since $\limsup_{t\to\infty} N(t) \le B$ holds, see also the proof in Proposition 2.1, without loss of generality, we can assume that $S(t) \le B$ for $t > 0$. Thus we have

$$b(t) \le B \int_0^\infty \beta(a) b(t-a) \mathcal{F}(a) da, \ t > 0.$$

Let us denote $\limsup_{t\to\infty} b(t) = \overline{b}$. Using Fatou's lemma we get $\overline{b} \le R_0 \overline{b}$. Since b is nonnegative, we can conclude that $\lim_{t\to\infty} b(t) = 0$. Then one can easily see that $\lim_{t\to\infty} (S(t), R(t)) = (B, 0)$. Hence we obtain the conclusion. □

3.2. Stability of the endemic equilibrium

In this section we consider stability of the endemic equilibrium. For $\delta = 0$ the characteristic equation is

$$1 + J + \lambda = (1 + \lambda) S \int_0^\infty \beta(a) e^{-\lambda a} \mathcal{F}(a) da. \tag{3.4}$$

The characteristic equation (3.4) was studied in detail in [17], see Exercise 3.10. We can state the following result, see [56, 17, 7] for the proof.

Lemma 3.3. *Let us assume that $R_0 > 1$ holds. If $\delta = 0$ then the endemic equilibrium is asymptotically stable.*

For $\delta > 0$ the authors in [12] presented a sufficient condition for local stability of the endemic equilibrium. We quote the result from Theorem 4.10 in [12] as

Theorem 3.4. *If $\delta \le 1$ and $\eta(a) = 0$ for $a \in \mathbb{R}_+$ then the endemic equilibrium is asymptotically stable.*

With a slight modification of the proof of Theorem 4.10 in [12], we obtain a better condition for stability of the endemic equilibrium. To formulate the condition we define a set:

$$\begin{aligned}\mathsf{D} :=& \{\delta \in \mathbb{R}_+ : \delta \in [0,1]\} \\ &\cup \left\{\delta \in \mathbb{R}_+ : \delta > 1 \text{ such that } R_0 > 1 + \frac{2(\delta-1)(1+\delta)(1+\delta A)}{1+2\delta}\right\}\end{aligned}$$

for a fixed $R_0 > 1$, where

$$A := \int_0^\infty (1+\eta(a))\,\mathcal{F}(a)da.$$

Theorem 3.5. *Let us assume that $R_0 > 1$ holds. If $\delta \in \mathsf{D}$ then the endemic equilibrium is asymptotically stable.*

Proof. From Lemma 3.3, the endemic equilibrium is locally asymptotically stable for sufficiently small $\delta > 0$ due to the continuation of the roots with respect to parameters, see, e.g. Lemma 2.8 in Chapter XI in [20]. Suppose that there exists $\delta \in \mathbb{R}_+$ such that there exists a purely imaginary root $\lambda = i\omega$, $\omega \in \mathbb{R}_+$ of (3.1). We can rewrite (3.1) as

$$1 + J + \lambda = (1+\lambda)\,S\int_0^\infty \beta(a)e^{-\lambda a}\mathcal{F}(a)da + \delta J\frac{\int_0^\infty \gamma(a)e^{-\lambda a}\mathcal{F}(a)da}{1+\delta+\lambda}, \tag{3.5}$$

as one can easily see that $\lambda \neq -1-\delta$. We substitute $\lambda = i\omega$ into (3.5). As in [12] one can find the estimation:

$$\left|S\int_0^\infty \beta(a)e^{-i\omega a}\mathcal{F}(a)da\right| \leq 1,\ \left|\int_0^\infty \gamma(a)e^{-i\omega a}\mathcal{F}(a)da\right| \leq 1. \tag{3.6}$$

Then it holds

$$|1+J+i\omega| \leq |1+i\omega| + \frac{\delta J}{|1+\delta+i\omega|}.$$

Taking square of both sides, we get

$$(1+J)^2 + \omega^2 \leq \left(1+\omega^2\right) + \frac{(\delta J)^2}{(1+\delta)^2+\omega^2} + 2\delta J\left\{\frac{1+\omega^2}{(1+\delta)^2+\omega^2}\right\}^{\frac{1}{2}},$$

which can be simplified as $J + 2 \leq l(\delta,\omega)$, where

$$l(\delta,\omega) := \frac{\delta^2 J}{(1+\delta)^2+\omega^2} + 2\delta\left\{\frac{1+\omega^2}{(1+\delta)^2+\omega^2}\right\}^{\frac{1}{2}}.$$

Now our aim is, to lead a contradiction, to find a set of δ such that $l(\delta,\omega) < J+2$. Now $l(\delta,\omega)$ can be estimated as

$$l(\delta,\omega) \leq \left(\frac{\delta}{1+\delta}\right)^2 J + 2\delta. \tag{3.7}$$

First assume that $\delta \leq 1$ holds. Then

$$l(\delta,\omega) \leq \frac{1}{4}J + 2 < J + 2,$$

which is a contradiction. We here note that, from (3.2) with (2.10b), J is written as

$$J = \frac{1+\delta}{1+\delta A}\left(R_0 - 1\right), \tag{3.8}$$

thus $R_0 > 1$ implies $J > 0$. Next assume that $\delta > 1$ and $\delta \in \mathsf{D}$. To show that $l(\delta,\omega) < J+2$ it is sufficient to show

$$2\left(\delta - 1\right) < J\left\{1 - \left(\frac{\delta}{1+\delta}\right)^2\right\} \tag{3.9}$$

using the estimation (3.7). From (3.8) one can see that

$$R_0 > 1 + \frac{2\left(\delta-1\right)\left(1+\delta\right)\left(1+\delta A\right)}{1+2\delta} \iff J > 2\frac{\left(\delta-1\right)\left(1+\delta\right)^2}{1+2\delta}.$$

Thus (3.9) holds. Consequently, if $\delta \in \mathsf{D}$ there is no purely imaginary root solving the characteristic equation (3.5). □

It is apparent that the stability result in Theorem 3.4 is deduced as a corollary of Theorem 3.5. We also note that, in Theorem 3.5, the disease-related mortality rate is not assumed to be zero, differently from the paper [12]. In Figure 3.1 we illustrate the set D in (R_0, δ) parameter plane, where the endemic equilibrium is asymptotically stable. On the other hand, combining Proposition 2.2 together with Theorem 3.2, it is shown that at $R_0 = 1$ the disease free equilibrium loses its stability and then the endemic equilibrium emerges. It can be shown that the endemic equilibrium is asymptotically stable if R_0 is slightly above one (the exchange of stability), see e.g. [41].

3.2.1. Constant recovery rate and disease-related mortality rate

We here elaborate a special case that both the recovery rate and the disease-related mortality rate are respectively given as constants. We put

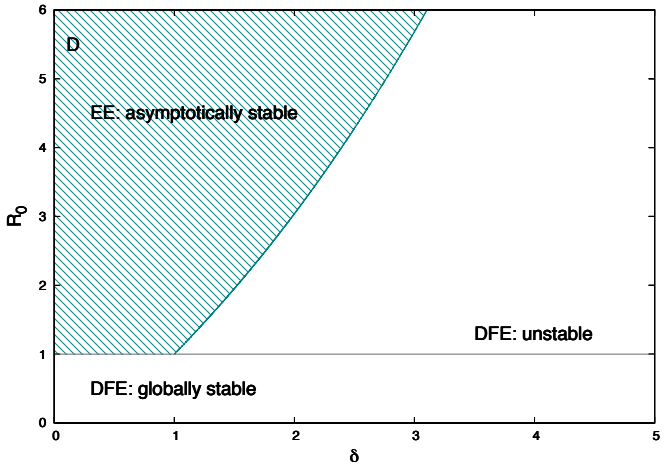

Figure 3.1: The stability region of the endemic equilibrium (EE) in (δ, R_0)-parameter plane. The disease free equilibrium (DFE) is globally asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.

Assumption 3.1.

$$\gamma(a) = \gamma,\ \eta(a) = \eta,\ a \in \mathbb{R}_+. \tag{3.10}$$

One now has $\mathcal{F}(a) = e^{-(1+\eta+\gamma)a}$. It can be shown that the endemic equilibrium is stable for any β.

Theorem 3.6. *Let $R_0 > 1$ holds. Then the endemic equilibrium is asymptotically stable.*

Proof. We compute

$$\int_0^\infty \gamma(a) e^{-\lambda a} \mathcal{F}(a) da = \frac{\gamma}{1+\eta+\gamma+\lambda}.$$

For convenience we define

$$k(a) := S\beta(a)\mathcal{F}(a).$$

Then the characteristic equation (3.1) is expressed as

$$\begin{aligned} &(1+J+\lambda)(1+\delta+\lambda)(1+\eta+\gamma+\lambda) - \delta\gamma J \\ &= (1+\lambda)(1+\delta+\lambda)(1+\eta+\gamma+\lambda)\int_0^\infty k(a)e^{-\lambda a} da. \end{aligned} \tag{3.11}$$

From Lemma 3.3 there exists no root with a positive real part for $\delta = 0$. Let us assume that there exists $\delta > 0$ such that (3.11) has a purely imaginary root, $\lambda = i\omega,\ \omega \in \mathbb{R}_+$. Separating real part and imaginary part of (3.11) with $\lambda = i\omega$, one obtains

$$\begin{aligned}
&l_1 - l_2\omega^2 \\
&= \left(m_1 - m_2\omega^2\right) \int_0^\infty k(a)\cos(\omega a)\,da + \left(m_3\omega - \omega^3\right) \int_0^\infty k(a)\sin(\omega a)\,da, \\
&\quad -\omega^3 + l_3\omega \\
&= -\left(m_1 - m_2\omega^2\right) \int_0^\infty k(a)\sin(\omega a)\,da + \left(m_3\omega - \omega^3\right) \int_0^\infty k(a)\cos(\omega a)\,da,
\end{aligned}$$

where we can specify

$$\begin{aligned}
l_1 &:= (1+J)(1+\delta)(1+\eta+\gamma) - \delta\gamma J, \\
l_2 &:= (1+J) + (1+\delta) + (1+\eta+\gamma), \\
l_3 &:= (1+J)(1+\delta) + (1+\delta)(1+\eta+\gamma) + (1+\eta+\gamma)(1+J), \\
m_1 &:= (1+\eta+\gamma)(1+\delta), \\
m_2 &:= 1 + (1+\eta+\gamma) + (1+\delta), \\
m_3 &:= (1+\eta+\gamma)(1+\delta) + (1+\eta+\gamma) + (1+\delta).
\end{aligned}$$

Since

$$\left(\int_0^\infty k(a)\cos(\omega a)\,da\right)^2 + \left(\int_0^\infty k(a)\sin(\omega a)\,da\right)^2 \le 1$$

(see also (3.6) in the proof of Theorem 3.5), we get the following inequality

$$\left(l_1 - l_2\omega^2\right)^2 + \left(-\omega^3 + l_3\omega\right)^2 \le \left(m_1 - m_2\omega^2\right)^2 + \left(m_3\omega - \omega^3\right)^2,$$

which is equivalent to

$$c_1\omega^4 + c_2\omega^2 + c_3 \le 0, \tag{3.12}$$

where $c_j,\ j \in \{1,2,3\}$ are defined as

$$\begin{aligned}
c_1 &:= l_2^2 - 2l_3 - m_2^2 + 2m_3, \\
c_2 &:= -2l_1l_2 + l_3^2 + 2m_1m_2 - m_3^2, \\
c_3 &:= l_1^2 - m_1^2.
\end{aligned}$$

We now show that $c_j > 0,\ j \in \{1,2,3\}$. To facilitate the computations we introduce constants

$$q_1 := 1 + \delta,\ q_2 := 1 + \eta + \gamma.$$

We use the following relations:

$$\begin{aligned} l_1 &= m_1 + J\left(m_1 - \delta\gamma\right), \\ l_2 &= m_2 + J, \\ l_3 &= m_3 + J\left(q_1 + q_2\right). \end{aligned}$$

Then we obtain

$$\begin{aligned} c_1 &= J\left(2m_2 + J\right) - 2J\left(q_1 + q_2\right) \\ &= J^2 + 2J\left\{m_2 - \left(q_1 + q_2\right)\right\} \\ &= J^2 + 2J, \end{aligned}$$

thus $c_1 > 0$ follows. We next show the positivity of c_2. One can see that

$$l_3^2 - m_3^2 = J\left\{J\left(q_1 + q_2\right)^2 + 2\left(q_1 + q_2\right)\left(q_1 q_2 + q_1 + q_2\right)\right\}$$

and that

$$\begin{aligned} &- l_1 l_2 + m_1 m_2 \\ =&J\left\{\left(\delta\gamma - m_1\right)J - m_1 m_2 - m_1 + \delta\gamma m_2\right\} \\ =&J\left\{\left(\delta\gamma - q_1 q_2\right)J - q_1 q_2\left(1 + q_1 + q_2\right) - q_1 q_2 + \delta\gamma\left(1 + q_1 + q_2\right)\right\}. \end{aligned}$$

Therefore it follows

$$\begin{aligned} c_2 =&J^2\left\{\left(q_1 + q_2\right)^2 + 2\left(\delta\gamma - q_1 q_2\right)\right\} \\ &+ 2J\left\{\left(q_1 + q_2\right)\left(q_1 q_2 + q_1 + q_2\right) - q_1 q_2\left(1 + q_1 + q_2\right) - q_1 q_2\right. \\ &\left.+\delta\gamma\left(1 + q_1 + q_2\right)\right\}. \end{aligned}$$

Straightforward calculations show that

$$\left(q_1 + q_2\right)^2 + 2\left(\delta\gamma - q_1 q_2\right) = q_1^2 + q_2^2 + 2\delta\gamma > 0$$

and that

$$\begin{aligned} &\left(q_1 + q_2\right)\left(q_1 q_2 + q_1 + q_2\right) - q_1 q_2\left(1 + q_1 + q_2\right) - q_1 q_2 + \delta\gamma\left(1 + q_1 + q_2\right) \\ =&\left(q_1 + q_2\right)^2 - 2q_1 q_2 + \delta\gamma\left(1 + q_1 + q_2\right) \\ =&q_1^2 + q_2^2 + \delta\gamma\left(1 + q_1 + q_2\right) \\ >&0. \end{aligned}$$

Finally we compute c_3. Since $m_1 > \delta\gamma$, one can see that $l_1 > m_1$, thus c_3 is also positive. Therefore, we get $c_j > 0$ for $j \in \{1, 2, 3\}$, which leads a contradiction to (3.12). Thus there is no possibility of having a purely imaginary roots and all roots locate in the left half complex plane. Hence the conclusion holds. □

3.3. SIS epidemic model

It is meaningful to discuss a special case that δ tends to infinity, i.e., infected individuals immediately obtain the susceptibility to the disease upon the recovery. One can see that the second component of the endemic equilibrium is an increasing function, with respect to δ, that approaches to

$$\frac{1}{\int_0^\infty (1+\eta(a))\,\mathcal{F}(a)da}\left(B - \frac{1}{\int_0^\infty \beta(a)\mathcal{F}(a)da}\right),$$

as $\delta \to \infty$: the incidence (at the equilibrium) increases with respect to δ. This could be reasonably explained as that the period of recovery, when individuals are fully protected from the disease, decreases to zero and thus they likely to infect again, obtaining the susceptibility. For this scenario the model is called SIS type and is reformulated as

$$\frac{d}{dt}S(t) = B - S(t) - S(t)\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da + \int_0^\infty \gamma(a)b(t-a)\mathcal{F}(a)da,$$
$$b(t) = S(t)\int_0^\infty \beta(a)b(t-a)\mathcal{F}(a)da.$$

For simplicity let us assume that $\eta(a) = 0$ for $a \in \mathbb{R}_+$. The characteristic equation for the endemic equilibrium is easily computed as

$$1 - S\int_0^\infty \beta(a)e^{-\lambda a}\mathcal{F}(a)da + b\int_0^\infty \beta(a)\mathcal{F}(a)da\int_0^\infty e^{-\lambda a}\mathcal{F}(a)da = 0.$$

In the paper [23] the equation is studied with assuming that $\mathcal{F}$ is a step function. The authors formally show instability of the endemic equilibrium through Hopf bifutcation, see also [61]. From the results, we expect that destabilisation of the endemic equilibrium of (2.8) is also possible. Two integral terms in the characteristic equation is a source of difficulty on the analysis of stability. Considering a special function $\mathcal{F}$ such as a step function, one can solve the integral. In Section 3.2.1 we consider the special form $\mathcal{F}(a) = e^{-(1+\eta+\gamma)a}$ and obtain the local stability result, which shows it is necessary to consider other forms for $\mathcal{F}$ in order to catch destabilisation.

Indeed if $\mathcal{F}$ and β are step functions with different size of supports, as pointed out in the paper [23], we numerically observe the occurrence of periodic solution, see Figure 3.2. Note that, in Figure 3.2, τ is the maximum attainable age while in [23] the corresponding parameter is the maximum duration of immunity, thus interpretations of the parameter are slightly different.

§4. Global stability of the endemic equilibrium

In the previous section, with Assumption 3.1, we show that the endemic equilibrium is locally asymptotically stable if it exists i.e., $R_0 > 1$ holds. We here

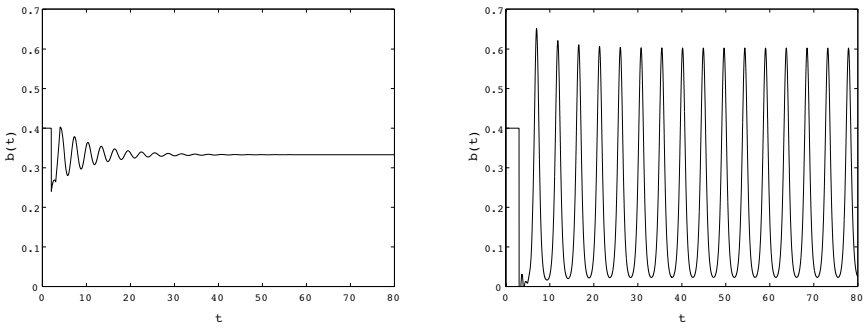

Figure 3.2: Examples of time evolution of the incidence rate $b(t)$ with different supports $[0, 1]$ and $[0, \tau]$ for step functions β and $\mathcal{F}$, respectively. (Left) if $\tau = 2$ the endemic equilibrium is stable. (Right) if $\tau = 3$ the endemic equilibrium is unstable and periodic solution exists.

continue to keep the same assumption and consider the global stability of the endemic equilibrium. To facilitate the analysis, we introduce two special cases that (2.8) can be expressed as a system of ordinary differential equations and a system of delay differential equations.

4.1. Ordinary differential equations

First, let us consider the case $\beta(a) \equiv \beta > 0$ for $a \in \mathbb{R}_+$. By the definition (2.1), one can see

$$b(t) = \beta S(t) \int_0^\infty b(t-a)\mathcal{F}(a)da = \beta S(t)I(t).$$

Similar to the equality (2.7), it holds that

$$\begin{aligned} \frac{dI(t)}{dt} =& b(t) - (1+\eta+\gamma)I(t) \\ =& \beta S(t)I(t) - (1+\eta+\gamma)I(t). \end{aligned} \tag{4.1}$$

We get the following system of the ordinary differential equations:

$$\frac{dS(t)}{dt} = B - S(t) - \beta S(t)I(t) + \delta R(t), \tag{4.2a}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - (1+\eta+\gamma)I(t), \tag{4.2b}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - (1+\delta)R(t). \tag{4.2c}$$

One can choose the same initial conditions for S and R as in (2.4). To ensure positivity of the solution we assume that $I(0) = I_0 > 0$.

SIRS epidemic model (4.2) is introduced in [2] to describe the experiments of disease dynamics on mice population. The authors fit the data of mice infected with diseases caused by a bacterium and virus. In the paper the threshold theorem is mentioned: the disease-free equilibrium is asymptotically stable if the basic reproduction number is less than 1 and an endemic equilibrium exists and it is asymptotically stable if the basic reproduction number is greater than 1. Local stability of the endemic equilibrium is considered in Section 3 in [60]. Later, Chen [11] proves the global asymptotic stability of the endemic equilibrium of the model (4.2).

Theorem 4.1. *Let us assume that $R_0 > 1$. Then the endemic equilibrium of system* (4.2) *is globally asymptotically stable.*

For the case $\eta = 0$, Korobeinikov and Wake [47] applied the direct Lyapunov method. Their proof is based on an idea that the SIRS model (4.2) can be rewritten in an SIR model by changing variables S (into $S + \frac{\delta B}{\beta\mu}$) on a limit system. Later, Vargas [75] offered non-uniqueness construction methods of Lyapunov functions for SIRS and SIS epidemic models for the case $\eta \geq 0$. (see also [25])

4.2. Delay differential equations

Now we make

Assumption 4.1. *For $h \in \mathbb{R}_+ \setminus \{0\}$, $\beta \in L^\infty(\mathbb{R}_+; \mathbb{R}_+)$ satisfies*

1. *β is nondecreasing,*
2. *$\beta(0) = 0$ and β is continuous from the right on the open interval $(0, h)$, i.e., $\beta(a) = \beta(a+)$ at every point $a \in (0, h)$,*
3. *$\beta(a) \equiv \beta > 0$ for $a \in [h, \infty)$.*

Then one can see

$$\int_0^a d\beta(\tau) = \beta(a), \ a \in \mathbb{R}_+, \tag{4.3}$$

thus

$$\int_0^\infty d\beta(\tau) = \int_0^h d\beta(\tau) = \beta(h),$$

where the integral has to be understood as a Riemann-Stieltjes integral (see e.g. Chapter 1 of [20] and Chapter 6 of [67]). Hence β is a function of normalised

bounded variations on $\mathbb{R}_+$. Notice that β is not necessary to be continuous on $\mathbb{R}_+$. In [12] β is assumed to be a uniformly continuous function. Therefore we are in a different situation from that paper.

We first express b using S and I.

Lemma 4.2. *It holds that*

$$b(t) = S(t) \int_0^h \mathcal{F}(\tau) I(t-\tau)\, d\beta(\tau). \tag{4.4}$$

Proof. By the equality (4.3), we have

$$\begin{aligned}
&\int_0^\infty b(t-a)\mathcal{F}(a)\beta(a)da \\
=&\int_0^h b(t-a)\mathcal{F}(a)\int_0^a d\beta(\tau)da + \int_h^\infty b(t-a)\mathcal{F}(a)\int_0^h d\beta(\tau)da.
\end{aligned}$$

By changing the order of the integrals, it holds that

$$\int_0^h \int_0^a b(t-a)\mathcal{F}(a)d\beta(\tau)da = \int_0^h \int_\tau^h b(t-a)\mathcal{F}(a)dad\beta(\tau).$$

This yields

$$\begin{aligned}
&\int_0^h \int_\tau^h b(t-a)\mathcal{F}(a)dad\beta(\tau) + \int_0^h \int_h^\infty b(t-a)\mathcal{F}(a)dad\beta(\tau) \\
=&\int_0^h \int_\tau^\infty b(t-a)\mathcal{F}(a)dad\beta(\tau).
\end{aligned}$$

Letting $a = s + \tau$, we have

$$\int_\tau^\infty b(t-a)\mathcal{F}(a)da = \int_0^\infty b(t-s-\tau)\mathcal{F}(s+\tau)ds.$$

Thus

$$\int_0^\infty b(t-a)\mathcal{F}(a)\beta(a)da = \int_0^h \int_0^\infty b(t-s-\tau)\mathcal{F}(s+\tau)dsd\beta(\tau).$$

Since Assumption 3.1 yields $\mathcal{F}(s+\tau) = e^{-(1+\eta+\gamma)(s+\tau)}$, we have $\mathcal{F}(s+\tau) = \mathcal{F}(\tau)\mathcal{F}(s)$. It follows from the definition (2.1) that

$$\begin{aligned}
b(t) =& S(t) \int_0^h \mathcal{F}(\tau) \int_0^\infty b(t-s-\tau)\mathcal{F}(s)dsd\beta(\tau) \\
=& S(t) \int_0^h \mathcal{F}(\tau) I(t-\tau) d\beta(\tau).
\end{aligned}$$

This completes the proof. □

Substituting (4.4) into system (2.3), we get the following system of delay differential equations:

$$\frac{dS(t)}{dt} = B - S(t) - S(t)\int_0^h \mathcal{F}(\tau)I(t-\tau)\, d\beta(\tau) + \delta R(t), \tag{4.5a}$$

$$\frac{dI(t)}{dt} = S(t)\int_0^h \mathcal{F}(\tau)I(t-\tau)d\beta(\tau) - (1+\eta+\gamma)I(t), \tag{4.5b}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - (1+\delta)R(t). \tag{4.5c}$$

This SIRS model (4.5) is considered in [82]. One can also see that (4.5) becomes the SIR epidemic model in [72] if $\delta = 0$. In those papers epidemic models are constructed to describe dynamics of a vector-borne disease, see also [9]. In those papers the incidence rate

$$S(t)\int_0^h \mathcal{F}(\tau)I(t-\tau)d\beta(\tau) \tag{4.6}$$

appears via a quasi-steady-state-assumption on the number of infected mosquitoes, see again [72]. This incidence form is widely appeared in the literature [3, 4, 5, 57, 66, 73, 82]. If we further assume

$$\beta(a) = \begin{cases} 0, & a \in [0, \hat{\tau}), \\ \beta, & a \in [\hat{\tau}, \infty) \end{cases} \tag{4.7}$$

for some $\hat{\tau} \leq h$ then we get a incidence rate by discrete delay:

$$S(t)\int_0^h \mathcal{F}(\tau)I(t-\tau)d\beta(\tau) = \beta\mathcal{F}(\hat{\tau})S(t)I(t-\hat{\tau}).$$

In Figure 4.1 we illustrate graph trajectories of β such that the incidence rate (4.6) is expressed by a discrete-delay and a distributed-delay, respectively.

Denote by $C([-h,0],\mathbb{R})$, the Banach space of continuous functions mapping the interval $[-h,0]$ into $\mathbb{R}$ equipped with the sup-norm $\|\psi\| = \sup_{\theta\in[-h,0]}|\psi(\theta)|$ for $\psi \in C([-h,0],\mathbb{R})$. We choose $\mathbb{R}_+ \times C([-h,0],\mathbb{R}_+) \times \mathbb{R}_+$ as the state space of (4.5). For I we choose the initial condition as

$$I(\theta) = \varphi(\theta),\ \theta \in [-h,0]$$

with $\varphi \in C([-h,0],\mathbb{R}_+)$ and $\|\varphi\| > 0$. Let us denote S_e, I_e and R_e as the first, second and third element of the endemic equilibrium, respectively. In [28] we obtain a result for the global stability:

Theorem 4.3. *Let us assume that $R_0 > 1$. If*

$$S_e - \delta R_e \geq 0, \tag{4.8}$$

then the endemic equilibrium of system (4.5) *is globally asymptotically stable.*

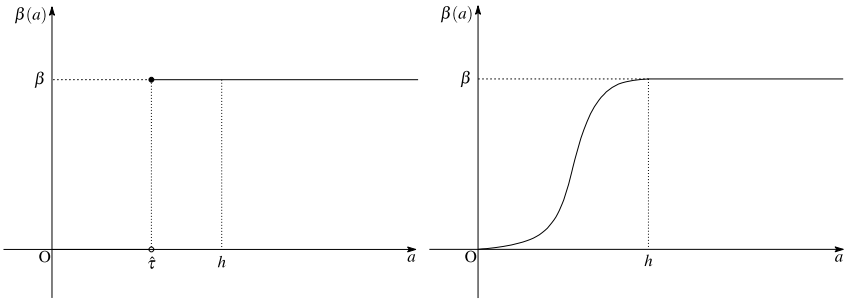

Figure 4.1: Curves of age-dependent function β. (Left) The incidence rate (4.6) is expressed by a discrete-delay. (Right) The incidence rate (4.6) is expressed by a distributed-delay.

The condition (4.8) is firstly introduced in [66] with assuming no disease-induced mortality rate ($\eta = 0$). One immediately obtains the following result proven in the celebrated paper [57] by McCluskey.

Corollary 4.4. *Let us assume that $R_0 > 1$. If $\delta = 0$ then the endemic equilibrium of system* (4.5) *is globally asymptotically stable.*

For the case $\delta = 0$, the global stability of the endemic equilibrium has been analysed in [4, 5, 3, 72]. With respect to delay, limited results are obtained: if the time delay is small enough then the endemic equilibrium is globally asymptotically stable. Their proofs are based on construction of Lyapunov functionals. This problem is recently revisited by McCluskey [57] with a novel Lyapunov functional and it is proven that the endemic equilibrium is *always* globally asymptotically stable. For a complete presentation we would like to show the proof of Theorem 4.3:

Proof. We define

$$G = \{(s, \varphi, r) \in \mathbb{R}_+ \times C([-h, 0], \mathbb{R}_+) \times \mathbb{R}_+ : s \geq 0,\ \|\varphi\| > 0,\ r \geq 0\}.$$

To construct Lyapunov functions, we introduce the following Volterra-type function:

$$g(x) := x - 1 - \ln x,\ x \in \mathbb{R}_+ \setminus \{0\}. \tag{4.9}$$

One can see that $x = 1$ is a strict global minimum with $g(1) = 0$. For $\psi = (s, \varphi, r) \in G$, let us define the following functional:

$$U_1(\psi) := S_e g\left(\frac{s}{S_e}\right) + I_e g\left(\frac{\varphi(0)}{I_e}\right) + S_e I_e \int_0^h \mathcal{F}(\tau) \int_{-\tau}^0 g\left(\frac{\varphi(u)}{I_e}\right) du d\beta(\tau).$$

We now show that U defined as

$$\begin{aligned} U(\psi) :=& U_1(\psi) + \frac{\delta}{\gamma S_e}\frac{(r-R_e)^2}{2} \\ &+ \frac{\delta\gamma}{\eta(2+\delta)S_e}\frac{\left[\{s+\varphi(0)+r-(S_e+I_e+R_e)\}+\frac{\eta}{\gamma}(r-R_e)\right]^2}{2} \end{aligned}$$

for $\eta > 0$ and

$$U(\psi) := U_1(\psi) + \frac{\delta}{\gamma S_e}\frac{(r-R_e)^2}{2} + \frac{\delta}{4S_e}\frac{\{s+\varphi(0)+r-(S_e+I_e+R_e)\}^2}{2}$$

for $\eta = 0$ is a Lyapunov functional. It suffices to show that

$$\frac{d}{dt}U(S(t), I_t, R(t)) \le 0, \tag{4.10}$$

where $(S(t), I_t, R(t))$ is a solution of (4.5) and a solution segment I_t is defined by $I_t(\theta) = I(t+\theta)$ for $\theta \in [-h, 0]$. See also the same notation introduced in Section 2.1. In the following we drop e from the notations S_e, I_e and R_e. In the proof of Theorem 4.1 in [57], the time derivative of the functional U_1 along the solution can be computed as follows.

$$\begin{aligned} \frac{dU_1}{dt} =& -\frac{(S(t)-S)^2}{S(t)} + \delta R\left(1-\frac{S}{S(t)}\right)\left(\frac{R(t)}{R}-1\right) \\ &- SI\int_0^h \mathcal{F}(\tau)\left\{g\left(\frac{S}{S(t)}\right) + g\left(\frac{S(t)I(t-\tau)}{SI(t)}\right)\right\}d\beta(\tau). \end{aligned} \tag{4.11}$$

Noting that $I(t) = N(t) - S(t) - R(t)$, we get

$$\begin{aligned} &\frac{1}{2}\frac{d}{dt}\left\{(R(t)-R)^2\right\} \\ =&(R(t)-R)\left\{\gamma I(t) - (1+\delta)R(t)\right\} \\ =&(R(t)-R)\left\{\gamma(N(t)-N) - \gamma(S(t)-S) - (1+\gamma+\delta)(R(t)-R)\right\}. \end{aligned} \tag{4.12}$$

For the case $\eta > 0$, we have $\frac{dN(t)}{dt} = B - N(t) - \eta I(t)$, which implies

$$\begin{aligned} \frac{dN(t)}{dt} + \frac{\eta}{\gamma}\frac{dR(t)}{dt} =& B - N(t) - \eta I(t) + \frac{\eta}{\gamma}(\gamma I(t) - (1+\delta)R(t)) \\ =& B - N(t) - \frac{\eta(1+\delta)}{\gamma}R(t). \end{aligned}$$

It follows that

$$\begin{aligned}
&\frac{1}{2}\frac{d}{dt}\{(N(t)-N)+\frac{\eta}{\gamma}(R(t)-R)\}^2\\
=&\left\{(N(t)-N)+\frac{\eta}{\gamma}(R(t)-R)\right\}\left(\frac{dN(t)}{dt}+\frac{\eta}{\gamma}\frac{dR(t)}{dt}\right)\\
=&\left\{(N(t)-N)+\frac{\eta}{\gamma}(R(t)-R)\right\}\left\{B-N(t)-\frac{\eta(1+\delta)}{\gamma}R(t)\right\}.
\end{aligned}$$

Since at the equilibrium $B-N-\frac{\eta(1+\delta)}{\gamma}R=0$ holds, we derive

$$\begin{aligned}
&\frac{1}{2}\frac{d}{dt}\{(N(t)-N)+\frac{\eta}{\gamma}(R(t)-R)\}^2\\
=&\left\{(N(t)-N)+\frac{\eta}{\gamma}(R(t)-R)\right\}\left\{-(N(t)-N)-\frac{\eta(1+\delta)}{\gamma}(R(t)-R)\right\}\\
=&-(N(t)-N)^2-\frac{\eta(2+\delta)}{\gamma}(N(t)-N)(R(t)-R)
\end{aligned}$$

(4.13)

$$-\frac{\eta^2(1+\delta)}{\gamma^2}(R(t)-R)^2.$$

Combining (4.11), (4.12) and (4.13), we obtain

$$\begin{aligned}
&\frac{dU}{dt}\\
=&-\frac{(S(t)-S)^2}{S(t)}+\delta\left(1-\frac{S}{S(t)}\right)(R(t)-R)+\frac{\delta}{S}(R(t)-R)(N(t)-N)\\
&-\delta(R(t)-R)\left(\frac{S(t)}{S}-1\right)-\frac{\delta(1+\gamma+\delta)}{\gamma S}(R(t)-R)^2\\
&-\frac{\delta\gamma}{\eta(2+\delta)S}(N(t)-N)^2-\frac{\delta}{S}(N(t)-N)(R(t)-R)\\
&-\frac{\delta\eta(1+\delta)}{\gamma(2+\delta)S}(R(t)-R)^2-\int_0^h\mathcal{F}(\tau)\left\{g\left(\frac{S}{S(t)}\right)+g\left(\frac{S(t)I(t-\tau)}{SI(t)}\right)\right\}d\beta(\tau).
\end{aligned}$$

By the following equality

$$\begin{aligned}
&\left(1-\frac{S}{S(t)}\right)(R(t)-R)-(R(t)-R)\left(\frac{S(t)}{S}-1\right)\\
=&-(R(t)-R)\left(\frac{S}{S(t)}-2+\frac{S(t)}{S}\right)\\
=&-(R(t)-R)\left(\frac{S(t)^2-2SS(t)+S^2}{SS(t)}\right)\\
=&-(R(t)-R)\frac{(S(t)-S)^2}{SS(t)},
\end{aligned}$$

we get

$$\begin{aligned}\frac{dU}{dt} =& -(S+\delta(R(t)-R))\frac{(S(t)-S)^2}{SS(t)} - \frac{\delta\gamma}{\eta(2+\delta)S}(N(t)-N)^2 \\ & - \left\{\frac{\delta(1+\gamma+\delta)}{\gamma S} + \frac{\delta\eta(1+\delta)}{\gamma(2+\delta)S}\right\}(R(t)-R)^2 \\ & - \int_0^h \mathcal{F}(\tau)\left\{g\left(\frac{S}{S(t)}\right) + g\left(\frac{S(t)I(t-\tau)}{SI(t)}\right)\right\} d\beta(\tau).\end{aligned}$$

Since the condition (4.8) implies

$$S+\delta(R(t)-R) \geq S-\delta R \geq 0, \tag{4.14}$$

we obtain

$$\begin{aligned}\frac{dU}{dt} \leq& -(S-\delta R)\frac{(S(t)-S)^2}{SS(t)} - \frac{\delta\gamma}{\eta(2+\delta)S}(N(t)-N)^2 \\ & - \left\{\frac{\delta(1+\gamma+\delta)}{\gamma S} + \frac{\delta\eta(1+\delta)}{\gamma(2+\delta)S}\right\}(R(t)-R)^2 \\ & - \int_0^h \mathcal{F}(\tau)\left\{g\left(\frac{S}{S(t)}\right) + g\left(\frac{S(t)I(t-\tau)}{SI(t)}\right)\right\} d\beta(\tau).\end{aligned} \tag{4.15}$$

For the case $\eta = 0$, we have $\frac{dN(t)}{dt} = B - N(t)$, thus

$$\frac{1}{2}\frac{d}{dt}(N(t)-N)^2 = (N(t)-N)\frac{dN(t)}{dt} = (N(t)-N)(B-N(t)).$$

Since at the equilibrium $N = B$ holds, one can get

$$\frac{1}{2}\frac{d}{dt}(N(t)-N)^2 = -(N(t)-N)^2. \tag{4.16}$$

Combining (4.11), (4.12) and (4.16), we obtain

$$\begin{aligned}\frac{dU}{dt} =& -S\frac{(S(t)-S)^2}{SS(t)} + \delta\left(1-\frac{S}{S(t)}\right)(R(t)-R) - \delta(R(t)-R)\left(\frac{S(t)}{S}-1\right) \\ & - \frac{\delta}{S}(R(t)-R)^2 + \frac{\delta}{S}(R(t)-R)(N(t)-N) - \frac{\delta}{4S}(N(t)-N)^2 \\ & - \beta SI\int_0^h \mathcal{F}(\tau)\left\{g\left(\frac{S}{S(t)}\right) + g\left(\frac{S(t)I(t-\tau)}{SI(t)}\right)\right\} d\beta(\tau) \\ & - \frac{\delta}{\gamma S}(1+\delta)(R(t)-R)^2.\end{aligned}$$

It follows from the inequality (4.14) and

$$\begin{aligned}&-(R(t)-R)^2+(R(t)-R)(N(t)-N)-\frac{1}{4}(N(t)-N)^2\\ =&-\left\{(R(t)-R)-\frac{1}{2}(N(t)-N)\right\}^2,\end{aligned}$$

that

$$\begin{aligned}\frac{dU}{dt}\leq&-(S-\delta R)\frac{(S(t)-S)^2}{SS(t)}-\frac{\delta}{S}\left\{(R(t)-R)-\frac{1}{2}(N(t)-N)\right\}^2\\ &-\int_0^h\mathcal{F}(\tau)\left\{g\left(\frac{S}{S(t)}\right)+g\left(\frac{S(t)I(t-\tau)}{SI(t)}\right)\right\}d\beta(\tau)\\ &-\frac{\delta}{\gamma S}(1+\delta)(R(t)-R)^2. \qquad (4.17)\end{aligned}$$

From (4.15) and (4.17), for both cases $\eta>0$ and $\eta=0$, we obtain (4.10) for all $t>0$. We define a set

$$\overline{G}:=\{\psi\in G:U(\psi)\leq U(S(0),I_0,R(0))\}.$$

One can see that $\overline{G}$ is closed and positively invariant. Thus the closure of $\overline{G}$ is itself and $\overline{G}$ contains $(S(t),I_t,R(t))$ for all $t>0$. Since U is continuous on $\overline{G}$, U is a Lyapunov functional on G. We define the set

$$E=\left\{\psi\in\overline{G}:\frac{dU}{dt}(\psi)=0\right\}.$$

We get

$$E=\left\{\psi=(s,\varphi,r)\in\overline{G}\Big|s=S,\ r=R,\ \frac{s\varphi(-\tau)}{S\varphi(0)}=1\right\}.$$

Let M be the largest subset in E that is invariant with respect to (4.5). By LaSalle invariance principle ([49, Corollary 5.2]), the solution tends to M. We can prove that M consists only of the infected equilibrium. Thus every solution tends to the endemic equilibrium, that is, the endemic equilibrium is globally attractive. Since the endemic equilibrium is asymptotically stable, the endemic equilibrium is globally asymptotically stable. □

We solve (4.8) with respect to δ to get an explicit global stability condition in terms of δ for a fixed R_0, see [66, 28] for the proof.

Proposition 4.5. *The condition (4.8) is equivalent to*

$$\begin{aligned}\delta\in\mathbb{R}_+ \text{ for } R_0\in\left(1,1+\frac{1+\eta}{\gamma}\right],\\ \delta\in\left[0,\hat{\delta}(R_0)\right] \text{ for } R_0\in\left(1+\frac{1+\eta}{\gamma},\infty\right),\end{aligned}$$

where

$$\hat{\delta}(R_0) := \frac{1}{\frac{R_0}{1+\frac{1+\eta}{\gamma}} - 1}.$$

In [28] it is shown that the idea of the Lyapunov functional can be successfully applied for an SIRS model with a nonlinear incidence rate, if the incidence function has a monotone property, see also [27, 39, 58, 46, 45] for analyses of SIR models. Another type of stability conditions are given in [29] constructing a different Lyapunov functional.

As in [63] we can obtain a different condition for the global stability by the monotone iterative method, which amounts to find convergence sequences for eventual upper and lower bounds of the solutions [62, 63]. It is now convenient to assume $\eta = 0$ holds, i.e., no disease-induced mortality rate. Then one has

$$\lim_{t\to\infty}(S(t) + I(t) + R(t)) = \lim_{t\to\infty} N(t) = B,$$

thus we get the following limit system:

$$\frac{dI(t)}{dt} = (B - I(t) - R(t)) \int_0^h \mathcal{F}(\tau) I(t-\tau) d\beta(\tau) - (1+\gamma) I(t), \tag{4.18a}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - (1+\delta) R(t). \tag{4.18b}$$

We have the following theorem from [63, Corollary 1.1]. For readers we adopt the proof used in [63] for (4.18).

Theorem 4.6. *Let us assume that $R_0 > 1$. If $\eta = 0$ and*

$$\delta > \max\{0, \gamma - 1\}, \tag{4.19}$$

then the endemic equilibrium of system (4.18) is globally asymptotically stable.

Proof. For a function $f : \mathbb{R}_+ \to \mathbb{R}$, we write

$$\overline{f} = \limsup_{t\to+\infty} f(t), \quad \underline{f} = \liminf_{t\to+\infty} f(t).$$

We show that the condition (4.19) implies $\underline{I} = \overline{I}$. From the fluctuation lemma (see [77, Lemma 4.2] and [38, Lemma 4.2]), there exists a sequence $\{t_n\}_{n=1}^{\infty}$ such that $t_n \to \infty$ as $n \to \infty$ and

$$\lim_{n\to\infty} I(t_n) = \overline{I}.$$

Since $0 \le B - I(t_n) - R(t_n)$ and $\liminf_{n\to+\infty} R(t_n) \ge \underline{R}$, we get $0 \le B - \overline{I} - \underline{R}$. Similarly, $B - \underline{I} - \overline{R} \ge 0$ holds. We thus have

$$B - \overline{I} - \underline{R} \ge 0 \text{ and } B - \underline{I} - \overline{R} \ge 0. \tag{4.20}$$

Considering similar sequences for $\overline{R}$ we obtain

$$0 \le K\left(B - \overline{I} - \underline{R}\right)\overline{I} - (1+\gamma)\,\overline{I}, \tag{4.21}$$

$$0 \le \gamma\overline{I} - (1+\delta)\,\overline{R} \tag{4.22}$$

and similarly

$$0 \ge K\left(B - \underline{I} - \overline{R}\right)\underline{I} - (1+\gamma)\,\underline{I}, \tag{4.23}$$

$$0 \ge \gamma\underline{I} - (1+\delta)\,\underline{R}, \tag{4.24}$$

where

$$K := \int_0^\infty \mathcal{F}(\tau)d\beta(\tau).$$

From (4.21) and (4.24) one can obtain that

$$0 \le B - \overline{I} - \underline{R} - \frac{1+\gamma}{K} \le B - \overline{I} - \frac{\gamma}{1+\delta}\underline{I} - \frac{1+\gamma}{K}.$$

It also holds

$$0 \ge B - \underline{I} - \overline{R} - \frac{1+\gamma}{K} \ge B - \underline{I} - \frac{\gamma}{1+\delta}\overline{I} - \frac{1+\gamma}{K}.$$

Note that $R_0 > 1 \leftrightarrow B - \frac{1+\gamma}{K} > 0$. We consequently get

$$\overline{I} - \underline{I} \le \frac{\gamma}{1+\delta}\left(\overline{I} - \underline{I}\right). \tag{4.25}$$

Since the condition (4.19) is equivalent to $\frac{\gamma}{1+\delta} < 1$, the inequality (4.25) shows $\underline{I} = \overline{I}$. Since it holds that

$$\frac{\gamma}{1+\delta}\underline{I} \le \underline{R} \le \overline{R} \le \frac{\gamma}{1+\delta}\overline{I},$$

we obtain $\underline{R} = \overline{R}$. Hence the endemic equilibrium of (4.18) is globally attractive. Since the endemic equilibrium is asymptotically stable, the endemic equilibrium is globally asymptotically stable. □

For the case $\eta = 0$, from Theorems 4.3 and 4.6, using Proposition 4.5, we can illustrate parameter regions of global stability of the endemic equilibrium in (R_0, δ) plane, see Figure 4.2 for $\gamma > 1$. At first glance, the global stability conditions in Theorems 4.3 and 4.6 do not cover the whole parameter plane where $R_0 > 1$.

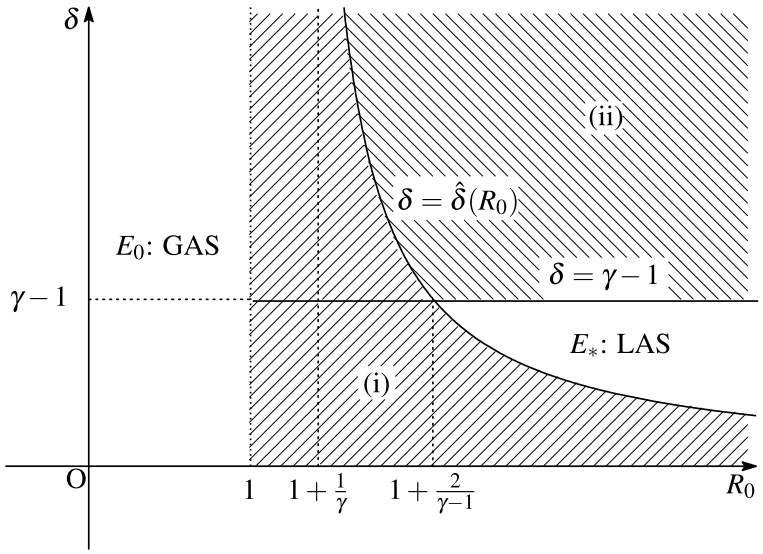

Figure 4.2: Global stability regions of the endemic equilibrium (E_*) when $\gamma > 1$. The global stability conditions in Theorems 4.3 and 4.6 are illustrated as a set of regions (i) and (ii), respectively. GAS = "globally asymptotically stable" and LAS = "locally asymptotically stable".

§5. On the discrete delay model

In Section 4.2 one sees that the incidence rate can be given as a discrete delay:

$$S(t)\int_0^h \mathcal{F}(a)I(t-a)d\beta(a) = \beta S(t)I(t-\hat{\tau})\mathcal{F}(\hat{\tau})$$

if we assume (4.7). For this scenario it would be natural to decompose the infectious compartment into two classes as

$$I(t) = \hat{E}(t) + \hat{I}(t),$$

where

$$\hat{E}(t) := \int_0^{\hat{\tau}} b(t-a)\mathcal{F}(a)da,\ \hat{I}(t) := \int_{\hat{\tau}}^{\infty} b(t-a)\mathcal{F}(a)da.$$

With (4.7) the interpretations of those compartments are clear: only the infected individuals in the compartment $\hat{I}$ transmit the disease. Now we aim to formulate the equivalent model to (4.5) in terms of $\hat{E}$ and $\hat{I}$. First let us introduce a connection between I and $\hat{I}$.

Lemma 5.1. *We have*

$$\hat{I}(t) = I(t-\hat{\tau})\mathcal{F}(\hat{\tau}).$$

Proof. One can see that

$$\hat{I}(t) = \int_{\hat{\tau}}^{\infty} b(t-a)\mathcal{F}(a)da = \int_{0}^{\infty} b(t-\hat{\tau}-s)\mathcal{F}(\hat{\tau}+s)ds.$$

Since $\mathcal{F}(\hat{\tau}+s) = \mathcal{F}(\hat{\tau})\mathcal{F}(s)$ from Assumption 3.1, we get

$$\hat{I}(t) = \int_{0}^{\infty} b(t-\hat{\tau}-s)\mathcal{F}(s)ds\mathcal{F}(\hat{\tau}) = I(t-\hat{\tau})\mathcal{F}(\tau).$$

□

Therefore one can see that $\beta S(t)I(t-\hat{\tau})\mathcal{F}(\hat{\tau}) = \beta S(t)\hat{I}(t)$ and that (2.8) is finally expressed as the following SEIRS model:

$$\frac{d}{dt}S(t) = B - S(t) - \beta S(t)\hat{I}(t) + \delta R(t), \tag{5.1a}$$

$$\frac{d}{dt}\hat{E}(t) = \beta S(t)\hat{I}(t) - \beta S(t-\hat{\tau})\hat{I}(t-\hat{\tau})\mathcal{F}(\hat{\tau}) - (1+\eta+\gamma)\,\hat{E}(t), \tag{5.1b}$$

$$\frac{d}{dt}\hat{I}(t) = \beta S(t-\hat{\tau})\hat{I}(t-\hat{\tau})\mathcal{F}(\hat{\tau}) - (1+\eta+\gamma)\,\hat{I}(t), \tag{5.1c}$$

$$\frac{d}{dt}R(t) = \gamma\left(\hat{I}(t) + \hat{E}(t)\right) - (1+\delta)\,R(t). \tag{5.1d}$$

The specification (4.7) implies that infected individuals obtain infectivity after infection-age is $\hat{\tau}$, thus discrete delay appears in (5.1). Similar models are considered in [10, 40, 80] assuming that latent individuals stays a fixed period in the compartment. In [40] the authors consider the case $\delta = 0$ and obtain the global stability of the endemic equilibrium. In [80] uniform persistent of the solution is shown if $R_0 > 1$ for the case $\delta > 0$, but the global stability of the endemic equilibrium is not discussed.

Since β defined in (4.7) can be seen as a special case of the one considered in Section 4.2, global stability results in the same section automatically hold for (5.1). Again a natural question arises: is the endemic equilibrium *always* globally asymptotically stable for (5.1)?

SEIRS models with a constant transition rate from E to I studied in [52, 37], formulated by a system of ordinary differential equations, are closely related to the model (5.1). For those models the global stability of the endemic equilibrium had been an open problem for a long time. In the paper [52] partial results were obtained by a geometric approach-a generalisation of the Bendixon-Dulac criteria [53]. Recently, a paper [13] has been published, where the authors improve the proof used in [52] and show that the endemic equilibrium of the SEIRS epidemic model is indeed globally asymptotically stable. The geometric approach is, however, not directly applicable to the infinite-dimensional dynamical systems such as (5.1).

§6. Other epidemic models with waning immunity

Finally we here briefly discuss other epidemic models taking into account waning immunity to close the paper.

6.1. Multi-group epidemic model

Epidemic models, in which the heterogeneous population is subdivided into several homogeneous groups due to the heterogeneity (e.g., sex, age, position, etc.) of each individual, are called multi-group models. In 1976, the paper [51] studied the global stability of a multi-group SIS epidemic model for the spread of gonorrhea. For multi-group SIR epidemic models, the local stability of equilibria had been studied by several authors (see e.g., [36]), however, the global stability of the endemic equilibrium had been an open problem for a long time. In 2006, the paper [30] proved the global asymptotic stability of a multi-group SIR epidemic model for $R_0 > 1$ by using a novel Lyapunov functional method based on the graph theory. After that, their method has been applied to various multi-group models by many authors (see e.g., [31] for a multi-group SEIR model, [54] for a SIR-type model with distributed time delays, [71] for an SIR model with nonlinear incidence, [48] for a discretized age-structured SIR model, etc).

Recently, the global stability of a multi-group SIRS epidemic model is considered in [64]. In their result, the global asymptotic stability of the endemic equilibrium is guaranteed if the basic reproduction number is greater than one and the similar condition with that in Theorem 4.3 holds. Although their proof is partly based on the idea of construction of a Laplacian matrix as in the graph-theoretic approach of [30], Muroya et al. showed that the graph-theoretic approach can be essentially reduced to the usual calculation of the derivative of Lyapunov functional together with the Volterra-type function (4.9). This way of calculation has been applied to various multi-group models by the same authors (see e.g., [64] for a multi-group SIR epidemic models with patch structure). In [65], the monotone iterative method of [63] is extended to a multi-group SIRS model.

6.2. Epidemic models with nonlinear incidence rate

The incidence rate, which characterises a new infection per unit of time, is modified by a class of nonlinear incidence rates. One of the pioneering works is achieved in [8] introducing a saturation level for the force of the infection, motivated by cholera epidemic spread in Bari in 1973. Observing that the number of contacts of susceptible individuals to infective individuals may decrease

when there is a large number of infected individuals, they consider psychological effects and model the nonlinear incidence rate, namely $\beta SG(I)$ where G is a saturated function.

For SIRS and SEIRS epidemic models periodic oscillation appears through Hopf bifurcation, if the incidence rate increases "faster" than the bilinear incidence rate [55]. On the other hand, it is shown that a class of nonlinear incidence rates do not change the qualitative dynamics of epidemic models. For example, in [78] the authors consider an SIRS epidemic model with a nonmonotone incidence rate, namely

$$\beta S(t)\frac{I(t)}{1+\alpha I^2(t)}, \tag{6.1}$$

and prove that the endemic equilibrium is globally stable if it exists. Comparing with the Theorem 4.1 one can see that the nonlinearity in the incidence rate do not affect the qualitative dynamics. SIRS model in [78] is extended by introducing delay in the nonlinear incidence rate [79, 26]. In [26] we explore a class of nonmonotone incidence rates such that the endemic equilibrium is always asymptotically stable. Here it is shown that nonmonotonicity with time delay in the incidence rate is a necessary ingredient for destabilisation of the endemic equilibrium. In that paper the global stability of the endemic equilibrium is not discussed. We expect that the endemic equilibrium is globally asymptotically stable, when the local stability condition does not depend on the delay. However, the treatment of the nonmonotonicity in the global stability analysis, e.g. construction of a Lyapunov function, seems to be a challenging problem.

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