Stability of the Endemic Equilibrium in Epidemic Models with Subpopulations

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ABSTRACT

For two models of infectious diseases, thresholds are identified, and it is proved that above the threshold there is a unique endemic equilibrium which is locally asymptotically stable. Both models are for diseases for which infection confers immunity, and both have the population divided into subpopulations. One model is a system of ordinary differential equations and includes immunization. The other is a system of integrodifferential equations and includes class-age infectivity.

1. INTRODUCTION

The spread of an infectious disease in a population depends not only on the character of the disease, but also on the structure of the population. Often there are some individuals who have many interactions with others in the population while others have only a few interactions. For example, school children have many more opportunities to contract and spread diseases such as measles, mumps, rubella, and chickenpox than most adults have. People with many different sexual partners have more opportunities to infect and be infected with gonorrhea than people who have fewer sexual partners [17]. Consequently, realistic analysis of disease transmission in a heterogeneous

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population often requires the model to include subpopulations, each of which is homogeneous in the sense that individuals in it have similar contact rates with other subpopulations and have similar recovery rates.

For epidemic models with one population, it often possible to prove global asymptotic stability. It is common for epidemic models of endemic infectious diseases (those which can persist in a population) to identify a threshold condition so that below the threshold the disease dies out, and above the threshold the disease remains endemic. Since models with complexities such as delays can have periodic solutions while the corresponding ordinary differential equation models have globally asymptotically stable equilibrium points [13–15], one might expect a more complex behavior for models with subpopulations. Thus it is important to carefully analyze epidemic models with subpopulations to determine whether they behave like the one-population models or behave in a new way. After establishing the existence and uniqueness of equilibrium points, it is essential to analyze their stability, since a periodic solution cannot arise by Hopf bifurcation if the equilibrium point is always locally asymptotically stable.

As epidemic models become more complex and subpopulations are incorporated, the thresholds are less obvious and it becomes more difficult or impossible to prove global stability. It is usually easier to prove global asymptotic stability below the threshold where the disease dies out, since in this case there is only one relevant equilibrium point. Above the threshold it may only be possible to prove that there is a unique endemic equilibrium point which is locally asymptotically stable. In order to be epidemiologically relevant, an endemic equilibrium should be at least locally asymptotically stable, i.e., if the disease levels are driven away from the endemic equilibrium by a small perturbation, then they should stay in a small neighborhood and finally converge to it. See [11] for an introduction to the terminology, notation, and basic types of models for infectious diseases, and [15] for a survey of mathematical results for epidemic models.

The notation most often used for epidemic models is $S(t)$, $E(t)$, $I(t)$, and $R(t)$ for the fractions of the population that are susceptible, exposed (latent), infectious, and removed by immunity, isolation, or death, respectively. Models are commonly referred to by a sequence of letters which indicate the flow of individuals between the compartments; for example, in an $SEIRS$ model, people are first susceptible, then exposed, then infectious, then removed, and finally susceptible again after they have lost their temporary immunity.

We now briefly describe the previous results on deterministic epidemic models with n subpopulations. Rushton and Mautner [28] obtained solutions for the simple SI epidemic model with n subpopulations. Models of SIS type with n subpopulations were analyzed by Lajmanovich and Yorke [22], who identified the stability modulus of a matrix as the threshold quantity and proved global asymptotic stability both below and above the threshold. These

results have been extended by Nold [26] and have been applied to gonorrhea [17]. Lajmanovich and Yorke's results have been extended to models with periodic contact rate by Aronsson and Mellander [1], have been extended to include spatial spread by Nallaswamy and Shukla [25], have been generalized to models with continuous stratification of risk by Thieme [32], and have been obtained for general ordinary-differential-equation models by Hirsch [18].

There are fewer mathematical results for epidemic models involving n subpopulations for diseases with immunity. Hethcote [12] analyzed the global behavior of solutions of an *SIR* model with n subpopulations, but without vital dynamics (births and deaths). For an *SIRS* model with n subpopulations and with immunization which has vital dynamics or temporary immunity or both, Hethcote [12] identified the stability modulus of a matrix as the threshold quantity. He also proved global asymptotic stability below the threshold and proved the existence of a unique endemic equilibrium above the threshold. He did not prove local asymptotic stability of the endemic equilibrium.

For an *SEIRS* model with n subpopulations, Thieme [30] showed global asymptotic stability if the latent and removed periods are sufficiently short. He also showed that immigration and emigration do not affect the global asymptotic stability. Thieme [31] identified the threshold quantity for an *SEIRS* model with n subpopulations and with vital dynamics. He also proved that above the threshold, there is a unique endemic equilibrium, which is locally asymptotically stable.

A model of *SIRS* type with vital dynamics and with n subpopulations corresponding to age groups has been analyzed by Tudor [33], who proved global asymptotic stability below the threshold and the existence of an endemic equilibrium above the threshold. Dietz and Schenzle [6] and Schenzle [29] consider *SIR* models with n age groups.

In this paper we analyze two models for infectious diseases with immunity involving n subpopulations. First a constructive theorem for the existence and uniqueness of a positive fixed point is proved. Next we briefly describe an *SIRS* immunization model and show that an equilibrium point must satisfy an equation in fixed-point form. Then the fixed-point theorem is applied to this immunization model, and the endemic equilibrium is shown to be locally asymptotically stable. The second model which we formulate is an *SIRS* model with class-age-dependent infectivity and with n subpopulations. The fixed point theorem is used to show existence of a unique endemic equilibrium point, and then the local asymptotic stability is obtained. In this second model the infectivity of an infected person is a function of age in the infected class; this generality allows a latent period with zero infectivity followed by an infectious period with an infectivity which first increases to a maximum and then decreases to zero. Although the second model includes

the first model without immunization as a special case, the proofs for the first model are included, since they are illuminating and are much easier.

The global stability of the endemic equilibrium points for the two models is still an open problem. Because the solutions approach the equilibrium point exponentially, it was possible to prove global asymptotic stability of the endemic equilibrium for the *SIS* model of Lajmanovich and Yorke [22]. Since the rate of approach was fastest for the component with the largest relative distance from the equilibrium point, they were able to use a supremum norm as a Liapunov function to show global asymptotic stability. Since solutions of *SIRS* models with temporary immunity or births and deaths usually spiral into the endemic equilibrium point [11], the method of Lajmanovich and Yorke does not work for these models. Indeed, global asymptotic stability of the endemic equilibrium point has not even been proved for some simple models, e.g. for the standard one-population *SEIRS* model involving ordinary differential equations [15,16].

2. A FIXED POINT THEOREM

Here we prove a theorem for the existence and uniqueness of a positive fixed point of a multivariable function. This theorem is used in Sections 4 and 6 to establish the existence of unique endemic equilibrium points.

Although the existence part of the theorem follows from Theorem 4.11 of Krasnoselskii [21] for fixed points of operators on cones in Banach spaces, we present an easy and constructive proof for maps on R^n. The existence proof procedure is similar to one in Nold [26] and uses results for irreducible, nonnegative matrices in a manner similar to Lajmanovich and Yorke [22]. Uniqueness is proved using a Krasnoselskii sublinearity trick as in Thieme [31]. Although our theorem is not original, since other analyses of population biology models have used similar special cases of the Krasnoselskii theorems, we believe that it is worthwhile to give a precise statement of this special theorem and to give an elementary proof. The M-matrix theory in Berman and Plemmons [2] gives many conditions equivalent to the condition in the theorem on the spectral radius ρ; these conditions may be easier to verify for a particular matrix [27].

An $n \times n$ matrix $[\alpha_{ij}]$ is *irreducible* if for any nonempty subset S of $\{1,\ldots,n\}$ with a nonempty complement S', there exist i in S and j in S' such that $\alpha_{ij} \neq 0$. The directed graph associated with a matrix $[\alpha_{ij}]$ has a directed edge from node i to node j if $\alpha_{ij} \neq 0$. A matrix is irreducible iff the directed graph is strongly connected, i.e., for any ordered pair (i, j) there is a path from node i to node j. The epidemiologic interpretation of an irreducible transmission matrix is that every pair of subpopulations is joined by an infectious path so that the presence of infectious people in the first subpopulation causes infection in the second subpopulation. If the transmission matrix $[\alpha_{ij}]$ is reducible, and α_{ij} and α_{ji} are either both positive or both

zero, then the strongly connected components have no epidemiological interactions and hence can be considered separately. A function $F(x)$ from $R_+^n = [0,\infty)^n$ into itself is called *strictly sublinear* if for fixed x in $(0,\infty)^n$ and fixed r in $(0,1)$, there exists an $\epsilon > 0$ such that $F(rx) \geqslant (1+\epsilon)rF(x)$. Strict sublinearity means that the graph of $F(x)$ with $F(0) = 0$ is strictly concave when one endpoint is fixed at $(0, F(0))$, i.e., $F((1-r)0 + rx) \geqslant (1+\epsilon)[(1-r)F(0) + rF(x)]$. Here $\geqslant$ denotes the pointwise ordering in R^n, i.e., the ordering generated by the cone R_+^n.

THEOREM 2.1

Let $F(x)$ be a continuous, monotone nondecreasing, strictly sublinear, bounded function which maps the nonnegative orthant $R_+^n = [0,\infty)^n$ into itself. Let $F(0) = 0$ and $F'(0)$ exist and be irreducible. Then $F(x)$ does not have a nontrivial fixed point on the boundary of R_+^n. Moreover, $F(x)$ has a positive fixed point iff $\rho(F'(0)) > 1$. If there is a positive fixed point, then it is unique.

Proof. Since $F'(0)$ is irreducible and nonnegative, the Perron-Frobenius theory [2,34] implies that it has a real, simple eigenvalue equal to its spectral radius $\rho(F'(0))$, the corresponding eigenvector v is positive, any nonnegative eigenvector is a multiple of v, and the spectral radius is equal to the stability modulus $s(F'(0))$ (i.e., the maximum real part of the eigenvalues).

Suppose F had a nonzero fixed point Y on the boundary of R_+^n. Let S be the set of indices i where $Y_i = 0$, and S' be the set of indices where $Y_i \neq 0$. Thus $F_i(Y) = Y_i = 0$ for i in S, and also $F_i(rY) = 0$ for $0 \leqslant r \leqslant 1$, since F is nondecreasing. Using Taylor's theorem, we find $0 = F_i(rY) = (F'(0)Y)_i r +$ (higher order terms), so that $(F'(0)Y)_i = \sum_{j=1}^n \alpha_{ij} Y_j = 0$ for i in S, where $F'(0) = [\alpha_{ij}]$. Hence $\alpha_{ij} = 0$ for i in S and j in S' with nonempty S and S', which contradicts the irreducibility of $F'(0)$.

Let $f(x) = F(x) - x$, and define $M = \{x \in R_+^n \mid f_i(x) \geqslant 0 \text{ for } i = 1,\ldots,n\}$. The set M is compact, since $F(x)$ is bounded and $f(x)$ is continuous. Let E_i be the maximum x_i for all x in M.

We now show that E is a fixed point of F. Suppose E is not in M; then $f_i(E) < 0$ for some i. The definition of E implies that there is an x in M such that $x_i = E_i$ and $x_j < E_j$ for some $j \neq i$. Since F is monotone nondecreasing, $0 \leqslant f_i(x) \leqslant f_i(E)$ which is a contradiction. Suppose $f_i(E) > 0$ for some i. Then there is some x in M with $x_i > E_i$, because f_i is continuous and $f_j(x)$ is a monotone nondecreasing function of x_i for $j \neq i$. But this contradicts the definitions of E_i, so that $f(E) = 0$ and E is a fixed point of F.

If $B = f'(0)$, then $s(B) = s(F'(0)) - 1$, so B has a real simple eigenvalue $\mu = s(B)$ with a positive eigenvector v. Taylor's theorem implies $f(\delta v) = \delta\mu v$ + (higher-order terms). If $\mu = \rho(F'(0)) - 1 > 0$, then $f(\delta v) > 0$ for sufficiently small positive δ. Since δv is positive and in M, the fixed point E is positive.

Assume that there is a positive fixed point E. Using strict sublinearity and Taylor's theorem, we obtain $rF(E/2) \leqslant F(rE/2) = F'(0)rE/2 +$ (higher-order terms) for all r in $[0,1]$. Dividing by r, we find $F(E/2) \leqslant F'(0)E/2 +$ (higher-order terms), so that $F(E/2) \leqslant F'(0)E/2$ for $r \neq 0$. Now using strict sublinearity with $r = \frac{1}{2}$, we obtain $F'(0)E/2 \geqslant F(E/2) \geqslant (1+\epsilon)F(E)/2 = (1+\epsilon)E/2$, so that $F'(0)E \geqslant (1+\epsilon)E$, which implies that $\rho(F'(0)) \geqslant 1$.

The uniqueness proof uses a Krasnoselskii sublinearity trick [21, Theorem 6.3]. Let E_1 and E_2 be two different positive fixed points of F, and let r be the maximum number such that $E_1 \geqslant rE_2$ and $E_2 \geqslant rE_1$, so that $r \in (0,1)$. Using strict sublinearity, we find $E_1 = F(E_1) \geqslant F(rE_2) \geqslant (1+\epsilon_2)rF(E_2) = (1+\epsilon_2)rE_2$ and similarly $E_2 \geqslant (1+\epsilon_1)rE_1$ for some positive ϵ_1 and ϵ_2. This contradicts the maximality of r.

We remark that if x^0 is in the interior of R_+^n, then the iterates $x^{k+1} = F(x^k)$ converge to the positive equilibrium point if $\rho(F'(0)) > 1$ and converge to the origin if $\rho(F'(0)) \leqslant 1$. See [21, Section 6.1.6].

3. AN *SIRS* MODEL WITH IMMUNIZATION

A model for the spread of an infectious disease in a heterogeneous population with n homogeneous subpopulations was analyzed by Hethcote [12]. Immunization of newborns and of susceptibles of all ages was incorporated. A threshold condition was found in terms of the stability modulus $s(A)$, which is the maximum of the real parts of the eigenvalues of a matrix A depending on the parameters in the model. It was shown that below the threshold $[s(A) \leqslant 0]$ the disease dies out, and above the threshold $[s(A) > 0]$ there is a unique endemic equilibrium point. The stability properties of the endemic equilibrium point remained open.

The terminology and detailed assumptions in the model are given in [12], so they are not repeated here. Briefly, N_i are constant subpopulation sizes, λ_{ij} are contact rates, γ_i are removal rates, μ_i are death rates (= birth rates), ρ_i are rates of loss of temporary immunity, φ_i are immunized fractions of newborns, and θ_i are immunization rates in subpopulations.

Since $S_i(t) + I_i(t) + R_i(t) = 1$, the differential equations in [12] can be reformulated in terms of I_i and R_i as follows:

$$I_i'(t) = (1 - I_i - R_i) \sum_{j=1}^{n} \beta_{ij} I_j - (\gamma_i + \mu_i) I_i$$

$$R_i'(t) = \gamma_i I_i + \varphi_i \mu_i + \theta_i (1 - I_i - R_i) - (\mu_i + \rho_i) R_i, \tag{3.1}$$

and

$$I_i(0) = I_{i0} \geqslant 0, \qquad R_i(0) = R_{i0} \geqslant 0, \tag{3.2}$$

with $I_{i0} + R_{i0} \leqslant 1$ for $i = 1,2,\ldots,n$ and

$$\beta_{ij} = \lambda_{ij} N_j / N_i. \tag{3.3}$$

An equilibrium point is a solution of the simultaneous nonlinear equations obtained by setting the right-hand sides of the equations (3.1) equal to zero. The system (3.1) has the trivial equilibrium with $I_i = 0$, $R_i = (\varphi_i \mu_i + \theta_i)/(\mu_i + \rho_i + \theta_i)$ corresponding to the trivial equilibrium in the formulation of [12]. In order to prove the existence and uniqueness of an endemic equilibrium point, we reformulate the nonlinear equations as a fixed-point equation.

Solving the second equilibrium-point equation for R_i and substituting into the first equation, we obtain after some algebraic manipulation that

$$I_i = \left[1 - \frac{\mu_i + \rho_i + \gamma_i}{\mu_i(1-\varphi_i) + \rho_i} I_i\right] \sum_{j=1}^{n} \frac{\beta_{ij}[\mu_i(1-\varphi_i) + \rho_i]}{(\gamma_i + \mu_i)(\mu_i + \rho_i + \theta_i)} I_j. \tag{3.4}$$

We write this as

$$I_i = (1 - \eta_i I_i) \sum_{j=1}^{n} \alpha_{ij} I_j = G_i(I), \tag{3.5}$$

where η_i and α_{ij} are the coefficients in (3.4). Since $\partial G_i / \partial I_i$ is not necessarily nonnegative, G is not monotone; we cannot apply the fixed-point theorem in Section 2 to this formulation.

If we rearrange (3.5) so that the I_i term is isolated on one side, then we obtain

$$I_i = \frac{\sum_{j=1}^{n} \alpha_{ij} I_j}{1 + \eta_i \sum_{j=1}^{n} \alpha_{ij} I_j} = F_i(I). \tag{3.6}$$

Thus the equilibrium points are fixed points of F given by

$$I = F(I), \tag{3.7}$$

and this is the formulation that we use to prove existence and uniqueness of an endemic equilibrium point.

Assume that $\mu_i + \rho_i > 0$ for every i, so that there is inflow into the susceptible class due to either births or loss of temporary immunity. Assume $\varphi_i < 1$, so that not all newborns are immunized. Assume that $\gamma_i + \mu_i > 0$ for every i, so that there is some outflow from the infectious class. Assume

$\varphi_i\mu_i + \gamma_i + \theta_i > 0$, so that there is some inflow into the removed class. Observe that $F'(0) = [\alpha_{ij}]$, which is a nonnegative matrix. As in [12], we assume that $[\beta_{ij}]$ or equivalently $[\lambda_{ij}]$ in (3.3) is an irreducible matrix, i.e., all of the subpopulations are connected by some path. Then $[\alpha_{ij}]$ is also an irreducible matrix. These conditions guarantee that no solution remains on the boundary of the positively invariant polyhedron for a finite time unless there are no infectives in any group [12, Lemma 4.2]. Thus the only fixed point of (3.7) with a zero component is the origin. The epidemiological interpretation is that if there is infectivity in any subpopulation of a strongly connected population, then immediately there is infectivity in every subpopulation; moreover, the flows between classes guarantee that no class contains everyone in a subpopulation.

4. EXISTENCE, UNIQUENESS, AND STABILITY OF THE ENDEMIC EQUILIBRIUM POINT FOR THE IMMUNIZATION MODEL

Theorem 2.1 is now applied to the fixed-point formulation (3.7) to prove existence and uniqueness of the endemic equilibrium point. In this case $F(I)$ is infinitely differentiable and is monotone nondecreasing. The function $F(I)$ is strictly sublinear with

$$\epsilon = \min_i \frac{(1-r)\eta_i \sum_{j=1}^{n} \alpha_{ij} I_j}{1 + r\eta_i \sum_{j=1}^{n} \alpha_{ij} I_j} \tag{4.1}$$

and is bounded, since each $F_i(I)$ is bounded above by $1/\eta_i$.

Thus Theorem 2.1 implies that the immunization model in Section 3 has a unique endemic equilibrium point iff the spectral radius of the matrix $[\alpha_{ij}]$ is greater than one. This result was obtained in [12], but the proof there relied on the similarity of the quadratic equations (3.4) to those in Lajmanovich and Yorke [22]. Note from [26] that the spectral radius of $[\alpha_{ij}]$ is greater than one iff the stability modulus of the matrix A is positive, where

$$A = \left[\beta_{ij} \frac{\mu_i(1-\varphi_i) + \rho_i}{\mu_i + \rho_i + \theta_i}\right] - \left[(\gamma_i + \mu_i)\delta_{ij}\right] \tag{4.2}$$

corresponds to [12] to the linearization around the die-out equilibrium point. The advantage of the general fixed point approach used here is that it applies not only to the *SIS* model of Lajmanovich and Yorke and to the immunization model here, but also to other epidemic models as illustrated in Section 6.

We now prove that if there is an endemic equilibrium point of the immunization model, then it is locally asymptotically stable. The proof uses a Krasnoselskii sublinearity trick as in Thieme [31]. If $u' = f(u)$ is an $n \times n$

system of differential equations and $E \in \mathbb{R}^n$ is an equilibrium point, then the local asymptotic stability of E is usually shown by proving that the linearized equation $w' = f'(E)w$ has no solutions of the form $w(t) = We^{zt}$ with $W \in \mathbb{C}^n$, $z \in \mathbb{C}$, $\operatorname{Re} z \geqslant 0$, i.e., $zW = f'(E)W$ with $W \in \mathbb{C}^n \setminus \{0\}$, $z \in \mathbb{C}$ implies $\operatorname{Re} z < 0$. Proceeding in this way, we consider the equations

$$\begin{aligned} zU_i &= \left(1 - I_i^* - R_i^*\right) \sum_{j=1}^{n} \beta_{ij} U_j \\ &\quad - (\gamma_i + \mu_i) U_i - (U_i + V_i) \sum_{j=1}^{n} \beta_{ij} I_j^*, \\ zV_i &= \gamma_i U_i - \theta_i (U_i + V_i) - (\mu_i + \rho_i) V_i \end{aligned} \tag{4.3}$$

for $i = 1, \ldots, n$, with $U_i, V_i, z \in \mathbb{C}$ and I_i^*, R_i^* being the coordinates of the endemic equilibrium. We suppose that $\operatorname{Re} z \geqslant 0$ and try to obtain a contradiction.

Solving the second equation in (4.3) for V_i and inserting the results into the first yields after some rearrangements that

$$\left[1 + \eta_i(z)\right] U_i = (HU)_i \tag{4.4}$$

with

$$\eta_i(z) = (\gamma_i + \mu_i)^{-1} \left\{ z + \sum_{j=1}^{n} \beta_{ij} I_j^* \left[\frac{z + \gamma_i + \mu_i + \rho_i}{z + \mu_i + \rho_i + \theta_i} \right] \right\} \tag{4.5}$$

and the matrix $H = (h_{ij})$, where

$$h_{ij} = (\gamma_i + \mu_i)^{-1} \beta_{ij} \left(1 - I_i^* - R_i^*\right). \tag{4.6}$$

Note that H is a nonnegative matrix and that $I^* = (I_1^*, \ldots, I_n^*)$ satisfies $I^* = HI^*$. Taking absolute values in (4.4), we obtain

$$\left[1 + \eta(z)\right] |U| \leqslant H|U| \tag{4.7}$$

with $|U| = (|U_1|, \ldots, |U_n|)$ and

$$\eta(z) = \inf\{\operatorname{Re} \eta_i(z),\ i = 1, \ldots, n\}. \tag{4.8}$$

An elementary calculation shows that if $\operatorname{Re} z \geqslant 0$, then $\operatorname{Re} \eta_i(z) > 0$ for all i, so that $\eta(z) > 0$. We now use Krasnoselskii's trick in another version [21, Theorem 2.11]. Let r be the minimum number such that $|U| \leqslant rI^*$. Since I^* is a strictly positive vector, $r < \infty$. Now by (4.7), $[1 + \eta(z)]|U| \leqslant H|U| \leqslant rHI^* = rI^*$. Since $\eta(z) > 0$ if $\operatorname{Re} z \geqslant 0$, we obtain a contradiction to the minimality of r. Thus $\operatorname{Re} z < 0$.

5. AN *SIRS* MODEL WITH CLASS-AGE-DEPENDENT INFECTIVITY

Epidemic models can be formulated with an infectivity which depends on the age in the infected class. For example, the infectivity can be zero, positive, and zero as the person ages through the latent, infectious, and immune stages, respectively. Alternatively, before they move to the removed class, infected individuals may have an infectivity which is zero and then positive as the person ages through the latent and infectious stages. The total infectivity of the infected people is the integral over all class ages of the infectivity at each age times the probability density of infected persons with class age a. Models with class-age formulations have been considered by several authors [3–5, 7–10, 19, 30–32]. Models with chronological age, which is different than class age, have also been considered [6, 19, 29, 33]. The model of Gripenberg [9] includes a time-invariant chronological-age structure and a permanent-immunity stage in the infected class. The *SIRS* model considered here is unusual in that it incorporates a chronological-age structure through birth and death rates and it has n subpopulations.

The *SIRS* model with n subpopulations considered here is similar to the model in Section 3 except that it has a class-age structure for the infected class. As usual S_j and R_j denote the fraction of susceptible and removed individuals in subpopulation j. Here I_j does not denote the infectious fraction, but is the fraction of infected (both latent and infectious) individuals in subpopulation j. The representation of $I_j(t)$ is

$$I_j(t) = \int_0^\infty x_j(t,a)\, da \tag{5.1}$$

with $x_j(t,a)$ denoting the probability density of infected individuals with class age a (the lapse of time since infection).

The normalized infective impact $J_j(t)$ on subpopulation j at time t is given by

$$J_j(t) = \sum_{k=1}^{n} \int_0^\infty \beta_{jk}(a)\, x_k(t,a)\, da \tag{5.2}$$

where $\beta_{jk}(a)$ denotes N_k/N_j times the infective impact on a susceptible individual in subpopulation j by an infected individual in subpopulation k with class age a [cf. Equation (3.3)]. Note that the dependence of the infection rates β_{jk} on the class age a allows incorporation of latent periods in various ways.

The different equations for the susceptible subpopulation are

$$S_j'(t) = -S_j J_j(t) + \mu_j(I_j + R_j) + \rho_j R_j, \tag{5.3}$$

where μ_j is the birth and mortality rate in subpopulation j, and ρ_j is the rate of return from the class of removed to the class of susceptible individuals. The equations for the probability densities of the infected subpopulations are

$$(\partial_t + \partial_a) x_j(t,a) = -\left[\mu_j + \gamma_j(a)\right] x_j(t,a),$$
$$x_j(t,0) = S_j(t) J_j(t) \tag{5.4}$$

for $t, a > 0$, $t \neq a$, $a \neq a_j \leqslant \infty$. Here $\gamma_j(a)$ is the class-age-dependent removal rate, a_j is the maximum class age at removal, and $\gamma_j(a) = 0$ for $a > a_j$. The removed subpopulations satisfy

$$R_j'(t) = \int_0^\infty \gamma_j(a) x_j(t,a)\, da - (\mu_j + \rho_j) R_j(t). \tag{5.5}$$

Equations (5.1)–(5.5) together with initial conditions constitute the *SIRS* model with class-age structure. Note that $S_j(t)$, $I_j(t)$, and $R_j(t)$ are the fractions of the subpopulations in the susceptible, infected and removed classes, so that they add up to 1 for all time.

All parameters in Equations (5.1)–(5.5) are nonnegative. As in Section 3, we assume that $\mu_j + \rho_j > 0$ so that there is some inflow into the susceptible class. We further suppose that γ_j is continuous on $[0, a_j)$ and $\gamma_j \equiv 0$ on (a_j, ∞). In order that no individual may stay infected forever, we assume $\mu_j > 0$ or $\int_0^\infty \gamma_j(a)\, da = \infty$. Note that

$$h_j(a) = \exp\left(-\mu_j a - \int_0^a \gamma_j(s)\, ds \right) \tag{5.6}$$

is the probability of still being infected at time a after infection began, and $[\mu_j + \gamma_j(a)] h_j(a)$ is the rate at which infected individuals leave the infected class at time a after infection began. Our assumption implies that $h_j(a) \to 0$ as $a \to \infty$, and

$$\int_0^\infty \left[\mu_j + \gamma_j(a)\right] h_j(a)\, da = 1, \tag{5.7}$$

so that the rate above is actually a probability density. If we want to model diseases with finite periods of infectedness, we choose $a_j < \infty$ and $\int_0^{a_j} \gamma_j(a)\, da = \infty$ with $\gamma_j(a) = 0$ for $a > a_j$. The condition $\int_0^\infty \gamma_j(a)\, da < \infty$ makes sense in diseases with lifelong carriers.

If $a_j = \infty$, we must impose additional technical assumptions on γ_j: (a) $\mu_j > 0$ or $\liminf a\gamma_j(a) > 1$ as $a \to \infty$, and (b) $\gamma_j(a) h_j(a)$ is monotone nonincreasing for large $a > 0$. Note that $\gamma_j(a) h_j(a)$ gives the rate at which a just-infected individual will be removed at time a after infection began. If γ_j

is absolutely continuous, (b) is equivalent to $\gamma_j'(a) \leqslant \gamma_j(a)[\mu_j + \gamma_j(a)]$ for large a. Assumption (a) implies that $ah_j(a) \to 0$ for $a \to \infty$ and

$$\int_0^\infty h_j(a)\, da < \infty.$$

Furthermore, the average class age H_j at leaving the infected class is finite, namely

$$H_j = \int_0^\infty a\left[\mu_j + \gamma_j(a)\right] h_j(a)\, da = \int_0^\infty h_j(a)\, da < \infty.$$

Assumption (b) guarantees that $\gamma_j(a)h_j(a) \to 0$ for $a \to \infty$, because $\int_0^\infty \gamma_j(a)h_j(a)\, da \leqslant 1$. Further

$$\gamma_j(t+a)\, h_j(t+a) \leqslant \text{const}\, \gamma_j(a)\, h_j(a)$$

for $t, a \geqslant 0$ and t large. These implications of (a) and (b) are trivially satisfied if $a_j < \infty$.

We assume that $\beta_{jk}(a) \geqslant 0$ and β_{jk} is continuous on $[0, a_k)$ and (a_k, ∞). We further assume that β_{jk} is bounded on $[0, \infty)$. In order for the population to be epidemiologically connected, i.e. for the disease to spread over all subpopulations regardless of the one in which it first breaks out, we assume that the matrix $\left(\int_0^\infty \beta_{jk}(a)\, da\right)$ is irreducible. If both $\beta_{ij}(a)$ and $\gamma_j(a)$ are constants, then the model here reduces to the model in Section 3 without immunization.

6. EXISTENCE, UNIQUENESS, AND STABILITY OF THE ENDEMIC EQUILIBRIUM FOR THE CLASS-AGE MODEL

The model in Section 5 is

$$\begin{aligned}
(\partial_t + \partial_a)\, x_j(t,a) &= -\left[\mu_j + \gamma_j(a)\right] x_j(t,a), \\
x_j(t,0) &= \left[1 - I_j(t) - R_j(t)\right] J_j(t) \\
&\text{for} \quad t, a > 0, \quad t \neq a, \quad a \neq a_j > 0, \qquad (6.1) \\
R_j'(t) &= \int_0^\infty \gamma_j(a)\, x_j(t,a)\, da - (\mu_j + \rho_j)\, R_j(t), \\
I_j(t) &= \int_0^\infty x_j(t,a)\, da, \\
J_j(t) &= \sum_{k=1}^n \int_0^\infty \beta_{jk}(a)\, x_k(t,a)\, da,
\end{aligned}$$

together with initial conditions satisfying $x_j(0,a) \geqslant 0$, $I_j(0) = \int_0^\infty x_j(0,a)\, da < \infty$, $R_j(0) \geqslant 0$, and $I_j(0) + R_j(0) < 1$. Theorems for the existence, unique-

ness, and stability of the endemic equilibrium for model (6.1) are given at the end of this section.

For equilibrium (i.e., time-independent) solutions, (6.1) takes the form

$$\begin{aligned} I_j^* &= \int_0^\infty x_j^*(a)\,da \\ x_j^{*\prime}(a) &= -\left[\mu_j + \gamma_j(a)\right] x_j^*(a) \\ x_j^*(0) &= \left(1 - I_j^* - R_j^*\right) J_j^* \\ (\mu_j + \rho_j)\, R_j^* &= \int_0^\infty \gamma_j(a)\, x_j^*(a)\,da \\ J_j^* &= \sum_{k=1}^n \int_0^\infty \beta_{jk}(a)\, x_k^*(a)\,da. \end{aligned} \tag{6.2}$$

We now obtain a fixed-point equation for $U_j = x_j^*(0)$.

Integrating the x_j^*-equations, we obtain

$$x_j^*(a) = h_j(a)\, U_j \tag{6.3}$$

with h_j in (5.6) and

$$I_j^* = \hat{h}_j(0)\, U_j, \tag{6.4}$$

$$R_j^* = (\mu_j + \rho_j)^{-1} \widehat{\gamma_j h_j}(0)\, U_j, \tag{6.5}$$

$$J_j^* = \sum_{k=1}^n \widehat{\beta_{jk} h_k}(0)\, U_k, \tag{6.6}$$

where $\hat{h}$ denotes the Laplace transform of h. Thus

$$U_j = \left(1 - \eta_j U_j\right) \sum_{k=1}^n \alpha_{jk} U_k \tag{6.7}$$

with

$$\eta_j = \hat{h}_j(0) + (\mu_j + \rho_j)^{-1} \widehat{\gamma_j h_j}(0), \tag{6.8}$$

$$\alpha_{jk} = \widehat{\beta_{jk} h_k}(0). \tag{6.9}$$

The irreducibility of $[\hat{\beta}_{jk}(0)]$ implies the irreducibility of $[\alpha_{jk}]$.

The formulation (6.7) is similar to (3.5) and can be converted as in Section 3 to

$$U_j = F_j(U) = \frac{\sum_{k=1}^n \alpha_{jk} U_k}{1 + \eta_j \sum_{k=1}^n \alpha_{jk} U_k}. \tag{6.10}$$

Thus the endemic equilibrium is a fixed point of F given by

$$U = F(U). \tag{6.11}$$

Theorem 2.1 can be applied as in Section 4 to obtain the lemma below.

LEMMA 6.1

Let the earlier assumptions be satisfied.

(a) *If (spectral radius of* $[\alpha_{jk}]) \leqslant 1$, *then* $U = (0,\ldots,0)$ *is the only nonnegative solution of* (6.7).

(b) *If (spectral radius of* $[\alpha_{jk}]) > 1$, *then there exists a unique positive solution of* (6.7) *which is different from* $(0,\ldots,0)$. *Moreover,* $U_j > 0$ *and* $\eta_j U_j < 1$ *for* $j = 1,\ldots,n$.

In terms of the original model, we have

THEOREM 6.2

Let the assumptions in Section 5 be satisfied.

(a) *If (spectral radius of* $[\alpha_{jk}]) \leqslant 1$, *then* $x_j^*(a) = 0$, $I_j^* = 0$, $R_j^* = 0$ *is the unique nonnegative equilibrium solution of the epidemic model* (6.1).

(b) *If (spectral radius of* $[\alpha_{ij}]) > 1$, *then there exists a unique positive endemic equilibrium solution of* (6.1) *which is different from the trivial equilibrium in (a). Moreover,* $I_j^* + R_j^* < 1$ *for* $j = 1,\ldots,n$.

Remark. It follows from (6.7) that there exists a vector $U > 0$ with

$$U_j = \left(1 - I_j^* - R_j^*\right) \sum_{k=1}^{n} \widehat{\beta_{jk} h_k}(0)\, U_k .$$

The following theorem on the stability of the endemic equilibrium is proved in the next section.

THEOREM 6.3

Let the assumptions in Section 5 be satisfied. The positive endemic equilibrium solution of (6.1) *(provided it exists) is locally asymptotically stable if*

$$\mu_j + \rho_j \left\{ \int_0^\infty (\cos ar)\left[\rho_j + 2\mu_j + \gamma_j(a)\right] \times \exp\left(-\mu_j a - \int_0^a \gamma_j(s)\, ds \right) da \right\} \geqslant 0 \tag{6.12}$$

for $r \in \mathbb{R}$, $j = 1,\ldots,n$.

The technical condition (6.12) is satisfied for epidemiologically reasonable choices of $\gamma_j(a)$, since we assumed earlier that $\mu_j + \rho_j > 0$. For example, (6.12) is satisfied if $\gamma_j(a)$ is a constant for every j. Condition (6.12) is also satisfied if immunity is permanent ($\rho_j = 0$). A wider class of examples is obtained by noting that

$$\int_0^\infty (\cos ra)\,\varphi(a)\,da \geqslant 0 \qquad \text{for} \quad r \in \mathbb{R}$$

if $\varphi(a)$ is monotone nonincreasing, convex, and such that $\varphi(\infty) = 0$. If γ_j is differentiable and γ_j' is absolutely continuous on $[0, a_j)$, then (6.12) reduces to the conditions

$$\gamma_j'(a) \leqslant \left[\rho_j + 2\mu_j + \gamma_j(a)\right]\left[\mu_j + \gamma_j(a)\right],$$

$$\gamma_j''(a) - \gamma_j'(a)\left[4\mu_j + \rho_j + 3\gamma_j(a)\right] + \left[\rho_j + 2\mu_j + \gamma_j(a)\right]\left[\mu_j + \gamma_j(a)\right]^2 \geqslant 0.$$

These conditions are satisfied if the average infectious periods $1/\gamma_j(a)$ are nondecreasing functions of age a and $\gamma_j''(a) \geqslant 0$.

Even if the sufficient conditions (6.12) are not satisfied, the positive endemic equilibrium solution is probably still locally asymptotically stable, since waiting times in the removed class have negative exponential distributions and this form of waiting time has not led to instability and Hopf bifurcation in previous models [13,15]. Note that the assumptions of Theorem 6.3 do not contain any conditions for the infectivity rates $\beta_{jk}(a)$ beyond the assumptions in section 5.

7. PROOF OF THEOREM 6.3

The nontrivial equilibrium in Theorem 6.2 (provided that it exists) is called locally asymptotically stable if for any $\epsilon > 0$ there exists a $\delta > 0$ such that if

$$\int_0^\infty \left[1 + \gamma_j(a)\right]\left|x_j(0,a) - x_j^*(a)\right|da + \left|R_j(0) - R_j^*\right| \leqslant \delta$$

for $j = 1, \ldots, n$, then

$$\int_0^\infty \left[1 + \gamma_j(a)\right]\left|x_j(t,a) - x_j^*(a)\right|da + \left|R_j(t) - R_j^*\right|$$

remains less than ϵ and also approaches 0 as $t \to \infty$ for $j = 1, \ldots, n$.

In order to find conditions for local asymptotic stability to hold we translate the equilibrium to the origin by letting

$$\begin{aligned} x_j(t,a) &= x_j^*(a) + y_j(t,a), \\ I_j(t) &= I_j^* + u_j(t), \\ R_j(t) &= R_j^* + v_j(t), \\ J_j(t) &= J_j^* + w_j(t). \end{aligned} \tag{7.1}$$

Then

$$\begin{aligned} (\partial_t + \partial_a)\, y_j(t,a) &= -\left[\mu_j + \gamma_j(a)\right] y_j(t,a) \\ y_j(t,0) &= -\left[u_j(t) + v_j(t)\right]\left[J_j^* + w_j(t)\right] + \left(1 - I_j^* - R_j^*\right) w_j(t), \\ v_j'(t) &= \int_0^\infty \gamma_j(a)\, y_j(t,a)\, da - (\mu_j + \rho_j)\, v_j(t) \\ u_j(t) &= \int_0^\infty y_j(t,a)\, da \\ w_j(t) &= \sum_{k=1}^{n} \int_0^\infty \beta_{jk}(a)\, y_k(t,a)\, da \end{aligned} \tag{7.2}$$

together with initial conditions $y_j(t,0)$ and $v_j(0)$.

We now transform the problem (7.2) into an integral equation for $z_j(t) = y_j(t,0)$. Recall that the convolution of two functions $u, v : [0,\infty) \to \mathbb{R}$ is defined by

$$(u * v)(t) = \int_0^t u(t-s)\, v(s)\, ds.$$

From (7.2) we obtain

$$y_j(t,a) = \begin{cases} h_j(a)\, z_j(t-a) & \text{for} \quad t > a, \\ \dfrac{h_j(a)}{h_j(a-t)}\, y_j(0, a-t) & \text{for} \quad t < a, \end{cases} \tag{7.3}$$

where h_j is given by (5.6).

We now separate u_j, v_j, and w_j into two parts which depend on $z_j(t) = y_j(t,0)$ and on $y_j(0,a)$ and $v_j(0)$. Thus

$$u_j = h_j * z_j + \mathring{u}_j, \tag{7.4}$$

with

$$\mathring{u}_j(t) = \int_0^\infty \frac{h_j(a+t)}{h_j(a)} y_j(0,a)\, da. \tag{7.5}$$

Furthermore,

$$v_j' = (\gamma_j h_j) * z_j + \tilde{v}_j - (\mu_j + \rho_j) v_j$$

with

$$\tilde{v}_j(t) = \int_0^\infty \gamma_j(a+t) \frac{h_j(a+t)}{h_j(a)} y_j(0,a)\, da,$$

so that

$$v_j(t) = (\gamma_j h_j) * \tilde{h}_j * z_j + \mathring{v}_j \tag{7.6}$$

with

$$\tilde{h}_j(a) = e^{-(\mu_j + \rho_j)a}, \tag{7.7}$$

$$\mathring{v}_j(t) = v_j(0)\tilde{h}_j(t) + (\tilde{h}_j * \tilde{v}_j)(t). \tag{7.8}$$

Moreover,

$$w_j = \sum_{k=1}^n (\beta_{jk} h_k) * z_k + \mathring{w}_j \tag{7.9}$$

with

$$\mathring{w}_j(t) = \sum_{k=1}^n \int_0^\infty \beta_{jk}(a+t) \frac{h_k(a+t)}{h_k(a)} y_k(0,a)\, da. \tag{7.10}$$

Fitting (7.4)–(7.10) into the second equation of (7.2), we obtain the integral equation

$$\begin{aligned} z_j = &-(c_j * z_j + \mathring{z}_j)\left(J_j^* + \sum_{k=1}^n b_{jk} * z_k + \mathring{w}_j\right) \\ &+ \left(1 - I_j^* - R_j^*\right)\left(\sum_{k=1}^n b_{jk} * z_k + \mathring{w}_j\right) \end{aligned} \tag{7.11}$$

with

$$\begin{aligned} c_j &= h_j + (\gamma_j h_j) * \tilde{h}_j, \\ b_{jk} &= \beta_{jk} h_k, \\ \mathring{z}_j &= \mathring{u}_j + \mathring{v}_j. \end{aligned} \tag{7.12}$$

Using the definitions above and $h_j(a+t)/h_j(a) \to 0$ as $t \to \infty$, and the additional assumptions (a) and (b) if $a_j = \infty$, we obtain the lemma below.

LEMMA 7.1

(a) $$\left|\mathring{z}_j(t)\right| + \left|\mathring{w}_j(t)\right| \leqslant \text{const}\left(\sum_{k=1}^{n} \int_0^\infty [1+\gamma_k(a)] \left| y_k(0,a) \right| da + \left| v_j(0) \right| \right)$$

(b) $$\left|\mathring{z}_j(t)\right| + \left|\mathring{w}_j(t)\right| \to 0 \qquad \text{for} \quad t \to \infty$$

if $\int_0^\infty [1+\gamma_k(a)]|y_k(0,a)|\,da < \infty$.

Under our assumptions, local asymptotic stability of the nontrivial equilibrium of the epidemic model follows using Lemma 7.1 if Statement 7.2 below is true. Statement 7.2 is obtained by a local-stability analysis of the integral equation (7.11).

STATEMENT 7.2

For every $\epsilon > 0$ *there exists* $\delta > 0$ *such that the following holds: If*

$$\left|\mathring{z}_j(t)\right| + \left|\mathring{w}_j(t)\right| \leqslant \delta \qquad \text{for} \quad j = 1, \ldots, n, \quad t \geqslant 0$$

and

$$\left|\mathring{z}_j(t)\right| + \left|\mathring{w}_j(t)\right| \to 0 \qquad \text{for} \quad t \to \infty,$$

then $|z_j(t)| \leqslant \epsilon$ *for* $t \geqslant 0$ *and* $z_j(t) \to 0$ *for* $t \to \infty$, $j = 1, \ldots, n$.

A closer look at the integral equation (7.11) reveals that Statement 7.2 holds if the integral kernel of the linearization $A(s) = [a_{jk}(s)]$ where

$$a_{jk}(s) = J_j^* c_j(s)\,\delta_{jk} - \left(1 - I_j^* - R_j^*\right) b_{jk}(s) \tag{7.13}$$

has an integrable resolvent kernel. It is well known from a result of Paley and Wiener that this is the case iff

$$\det(E + \hat{A}(z)) \neq 0 \qquad \text{for} \quad z \in \mathbb{C}, \quad \operatorname{Re} z \geqslant 0, \tag{7.14}$$

with E denoting the unit matrix; see Londen [23] or Miller [24]. We now obtain a sufficient condition for (7.14) to hold.

LEMMA 7.3

Let the integral kernel A *be given by* $A(s) = [a_{jk}(s)]$ *with*

$$a_{jk}(s) = \delta_{jk} g_j(s) - f_{jk}(s)$$

and non-negative integrable functions g_j, f_{jk}. *Further let* $(\hat{f}_{jk}(0))$ *be a nonnegative irreducible matrix with eigenvalue* 1 *and a corresponding eigenvector* U *satisfying* $U_j > 0$ *for* $j = 1, \ldots, n$. *Then* $\det(E + \hat{A}(z)) \neq 0$ *for* $z \in \mathbb{C}$, $\operatorname{Re} z \geqslant 0$, *provided* $\operatorname{Re} \hat{g}_j(z) \geqslant 0$ *for all pure imaginary* $z \in \mathbb{C}$, $j = 1, \ldots, n$.

The assumptions of Lemma 7.3 are satisfied for a_{jk} in (7.13) by the assumptions above and the remark to Theorem 6.2.

Proof of Lemma 7.3. In the first step we show that if $z \in \mathbb{C}$, $\operatorname{Re} z \geqslant 0$, and $\det(E + \hat{A}(z)) = 0$, then $\operatorname{Re} \hat{g}_j(z) < 0$ for some $j \in \{1, \ldots, n\}$. If $\det(E + \hat{A}(z)) \neq 0$, then there exists some $V \in \mathbb{R}^m$, $V \neq 0$, such that

$$V_j\left[1 + \hat{g}_j(z)\right] = \sum_{k=1}^{n} \hat{f}_{jk}(z) V_k.$$

Hence

$$\left|1 + \hat{g}_j(z)\right| |V_j| \leqslant \sum_{k=1}^{n} \left|\hat{f}_{jk}(z)\right| |V_k|.$$

On the other hand,

$$0 < U_j = \sum_{k=1}^{n} \hat{f}_{jk}(0) U_k$$

with some vector U. Choose $\tau > 0$ such that $U \geqslant \tau |V|$, where $|V| := (|V_1|, \ldots, |V_n|)$ and $U_j = \tau |V_j|$ for some $j \in \{1, \ldots, n\}$.

Now $\hat{f}_{jk}(0) \geqslant |\hat{f}_{jk}(z)|$ for $\operatorname{Re} z \geqslant 0$. For each j irreducibility of $[\hat{f}_{jk}(0)]$ implies that $\hat{f}_{jk}(0) > 0$ for some k, so that for this k, $\hat{f}_{jk}(0) > |\hat{f}_{jk}(z)|$ for $\operatorname{Re} z \geqslant 0$, $z \neq 0$. Thus

$$U_j = \sum_{k=1}^{n} \hat{f}_{jk}(0) U_k \geqslant [1 + \delta(z)] \sum_{k=1}^{n} \left|\hat{f}_{jk}(z)\right| U_k,$$

where $\delta(z) > 0$ if $\operatorname{Re} z \geqslant 0$, $z \neq 0$. Actually, by the Riemann-Lebesque lemma $\delta(z)$ can be chosen in such a way that

$$\inf\{\delta(z) : |z| \geqslant \epsilon, \operatorname{Re} z \geqslant 0\} > 0 \tag{7.15}$$

for any $\epsilon > 0$. Next

$$\begin{aligned} U_j &\geqslant [1 + \delta(z)] \sum_{k=1}^{n} \left|\hat{f}_{jk}(z)\right| \tau |V_k| \\ &\geqslant [1 + \delta(z)] \left|1 + \hat{g}_j(z)\right| \tau |V_j| \end{aligned}$$

Since $U_j = \tau|V_j|$, then

$$[1+\delta(z)]|1+\hat{g}_j(z)| \leqslant 1.$$

This implies that

$$\operatorname{Re}\hat{g}_j(z) \leqslant \frac{-\delta(z)}{1+\delta(z)} < 0 \tag{7.16}$$

for some $j \in \{1,\dots,n\}$. Note that δ does not depend on the functions g_i. This proves the first step.

The claim of the lemma follows from the second step below. Let $g_j^\xi(s) = (1-\xi)e^{-s} + \xi g_j(s)$ for $0 \leqslant \xi \leqslant 1$, and let A^ξ be the matrix with g replaced by g^ξ. We define

$$\xi_0 = \inf\{\xi \in [0,1], \det(E + \hat{A}^\xi(z)) = 0 \text{ for some } z \in \mathbb{C}, \operatorname{Re} z \geqslant 0\}.$$

If $\det(E+\hat{A}(z)) = 0$ for some $z \in \mathbb{C}$, $\operatorname{Re} z \geqslant 0$, then $\xi_0 \leqslant 1$. If $\operatorname{Re} z = 0$, then the lemma follows immediately from the first step. So we may assume $\xi_0 < 1$ by Rouche's theorem. We claim that $\xi_0 > 0$. Now

$$\widehat{g^\xi}(z) = (1-\xi)\frac{1}{z+1} + \xi\hat{g}(z);$$

hence

$$\operatorname{Re}\widehat{g^\xi}(z) = (1-\xi)\frac{x+1}{(x+1)^2+y^2} + \xi \operatorname{Re}\hat{g}(z).$$

Applying the first step to A^ξ instead of A, we obtain from (7.15), (7.16) and the Riemann-Lebesque lemma that $\det(E + \widehat{A^\xi}(z)) \neq 0$ for $\operatorname{Re} z \geqslant 0$ if $\xi > 0$ is sufficiently small. Thus $\xi_0 > 0$. Again applying Rouche's theorem and the Riemann-Lebesque lemma, we obtain $\det(E + \widehat{A^{\xi_0}}(z)) = 0$ for some $z \in \mathbb{C}$, $\operatorname{Re} z = 0$. Applying (7.16) to $g_j^{\xi_0}$ yields

$$\operatorname{Re}\widehat{g_j^{\xi_0}}(z) = (1-\xi_0)\frac{1}{1+y^2} + \xi_0 \operatorname{Re}\widehat{g_j}(z) < 0$$

for some j with $\operatorname{Re} z = 0$. This proves lemma 7.3.

Guided by Lemma 7.3, we now consider what $\operatorname{Re}\widehat{g_j}(z) \geqslant 0$ for $\operatorname{Re} z = 0$, $j = 1,\dots,n$, means for our original epidemic problem. By (7.12) and (7.13) it is equivalent to

$$\operatorname{Re}\widehat{c_j}(z) = \operatorname{Re}\left(\widehat{h_j}(z) + \frac{1}{\rho_j+\mu_j+z}\widehat{\gamma_j h_j}(z)\right) \geqslant 0$$

for $\operatorname{Re} z = 0$, $j = 1, \ldots, n$. Let us drop the subscript j for convenience. Using the definition of h_j in (5.6) and integrating by parts we obtain

$$\widehat{\gamma h}(z) = 1 - (z + \mu)\hat{h}(z).$$

Hence $\operatorname{Re} \hat{c}(ir) \geqslant 0$, $r \in \mathbb{R}$, iff

$$(\rho + \mu)[\rho \operatorname{Re} \hat{h}(ir) + 1] \geqslant -r\rho \operatorname{Im} \hat{h}(ir).$$

Since $-r \operatorname{Im} \hat{h}(ir) = 1 + \operatorname{Re} \hat{h}'(ir)$, we obtain after some calculations $\operatorname{Re} \hat{c}(ir) \geqslant 0$, $r \in \mathbb{R}$, iff

$$\mu + \rho \int_0^\infty (\cos rs)[(\rho + \mu) h(s) - h'(s)]\, ds \geqslant 0.$$

This result together with (5.6) yields Theorem 6.3.

With the transformation in this section it is possible to transform (6.1) to an integral equation for $\psi_j(t) = x_j(t, 0)$, namely

$$\psi_j = \left(1 - c_j * \psi_j + \dot{\psi}_j\right)\left(\sum_{k=1}^{m} b_{jk} * \psi_k + \tilde{\psi}_j\right). \tag{7.17}$$

Similar equations have been dealt with by Gripenberg [7–9], and for a special case by Diekmann and Montijn [4] and by Diekmann and van Gils [5] for $m = 1$, i.e. for homogeneous populations.

A local-asymptotic-stability result can be derived for (7.17) by the same techniques we used in this section. Again we obtain

$$\operatorname{Re} \widehat{c_j}(z) \geqslant 0 \qquad \text{for} \quad z \in \mathbb{C}, \quad \operatorname{Re} z = 0, \quad j = 1, \ldots, n$$

as a sufficient condition. We avoided that approach in order to avoid the retranslation from what stability means for (7.17) into what it means for (6.1).

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