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Global dynamics of a SEIR model with varying total population size

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Abstract

A SEIR model for the transmission of an infectious disease that spreads in a population through direct contact of the hosts is studied. The force of infection is of proportionate mixing type. A threshold σ is identified which determines the outcome of the disease; if $\sigma \leqslant 1$, the infected fraction of the population disappears so the disease dies out, while if $\sigma > 1$, the infected fraction persists and a unique endemic equilibrium state is shown, under a mild restriction on the parameters, to be globally asymptotically stable in the interior of the feasible region. Two other threshold parameters σ' and $\overline{\sigma}$ are also identified; they determine the dynamics of the population sizes in the cases when the disease dies out and when it is endemic, respectively.

Keywords: Epidemic models; Endemic equilibrium; Latent period; Global stability; Compound matrices

1. Introduction

Studies of epidemic models that incorporate disease caused death and varying total population have become one of the important areas in the

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mathematical theory of epidemiology and they have largely been inspired by the works of Anderson and May (see [1,2]). Most of the research literature on these types of models assume that the disease incubation is negligible so that, once infected, each susceptible individual (in the class S) instantaneously becomes infectious (in the class I) and later recovers (in the class R) with a permanent or temporary acquired immunity. A compartmental model based on these assumptions is customarily called a SIR or SIRS model. Many diseases, however, incubate inside the hosts for a period of time before the hosts become infectious. Models that are more general than the SIR or SIRS types need to be studied to investigate the role of incubation in disease transmission. Using a compartmental approach, one may assume that a susceptible individual first goes through a latent period (and is said to become exposed or in the class E) after infection before becoming infectious. The resulting models are of SEIR or SEIRS type, respectively, depending on whether the acquired immunity is permanent or otherwise.

We assume the population has a homogeneous spatial distribution and the mixing of hosts follow the law of 'mass action'. More specifically, we assume that the local density of the total population is a constant though the total population size $N(t) = S(t) + E(t) + I(t) + R(t)$ may vary with time. Here $S(t), E(t), I(t)$ and $R(t)$ denote the sizes of the S, E, I and R classes at any time t, respectively. The per capita contact rate λ, which is the average number of effective contacts with other individual hosts per unit time, is then a constant. A fraction $I(t)/N(t)$ of these contacts is with infectious individuals and thus the average number of relevant contacts of each individual with the infectious class is $\lambda I(t)/N(t)$. The total number of new infections at a time t is given by $\lambda I(t)S(t)/N(t)$. This form of mixing term has been used in the literature under different names. Busenberg and van den Driessche [3] call it proportionate mixing, a term which they attribute to Nold [4]; Mena-Lorca and Hethcote [5] call it standard incidence; de Jong et al. [6] call it true mass-action incidence. This incidence form should not be confused with another form $\beta I(t)S(t)$ that is often called the simple mass-action incidence (it is called pseudo mass-action incidence in [6]). The recovered hosts are assumed to acquire a permanent immunity so that they will not become susceptible again. This is a technical assumption aimed to reduce the complexity of the mathematical analysis, but is nonetheless a plausible approximation in the case of many viral infections such as in measles, smallpox and rubella. The rate of removal ϵ of individuals from the exposed class is assumed to be a constant so that $1/\epsilon$ can be regarded as the mean latent period. In the limiting case, when $\epsilon \to \infty$, the latent period is negligible and a SEIR model reduces to a SIR model.

The vital dynamics include exponential natural death with rate constant d and exponential birth with rate constant b. We assume that the infectious individuals suffer a disease-caused mortality with a constant rate α. The

equation for the total population size is $N' = (b-d)N - \alpha I$. If $b = d$ and $\alpha = 0$, $N(t)$ remains a constant and can be normalized to 1. This leads to a SEIR model with constant total population and bilinear incidence rate, which is known to possess a sharp threshold $\sigma = \lambda\epsilon/(\epsilon + b)(\gamma + b)$, sometimes called the *contact number* (see [7]). If $\sigma \leqslant 1$ the disease-free equilibrium (1,0,0,0) is globally asymptotically stable, namely, the disease dies out irrespective of the initial configuration; if $\sigma > 1$, there exists a unique endemic equilibrium which is globally asymptotically stable in the interior of the feasible region and the disease persists, if it initially exists, at an endemic equilibrium state. We refer the reader to [7,8] for references on SEIRS models with constant total population and to [9] for the proof of the global stability of a unique endemic equilibrium of a SEIR model. In the general case, $N(t)$ may vary with time and the dynamical behavior of the model become more intricate; there is an interplay between the dynamics of the disease and that of the total population. This interplay has been studied in earlier SIR and SIRS models (see [1,3,5,10,11]), and is one of the primary concerns in the present paper.

Research on epidemic models of SEIR or SEIRS type with varying total populations are scarce in the literature. To the authors' knowledge, the present paper is the first that gives a rigorous treatment of the global stability of an unique endemic equilibrium for the fractions of sub-populations and the global dynamics of $(S(t), E(t), I(t), R(t))$. Several new methods are employed in the present paper to overcome mathematical difficulties that are not present in SIR models. The existence and uniqueness of the endemic equilibrium P^* is established without solving explicitly for its coordinates. This makes the verification of the Routh–Hurwitz stability condition a very technical matter. We develop in Lemma 5.1 a new criteria of linear stability using ideas from multilinear algebra. This new criteria is then used to show the local asymptotical stability of P^*. The most challenging task is the proof of the global stability of P^*. Epidemic models of this type are notorious for the fact that the method of Lyapunov functions has rarely worked for the proof of the global stability of the endemic equilibrium. In the present paper, the global stability is proved by employing the theory of monotone dynamical systems together with a stability criterion for periodic orbits of multidimensional autonomous systems due to Muldowney [12]. This approach is also used in [9] for a SEIR model with constant total population.

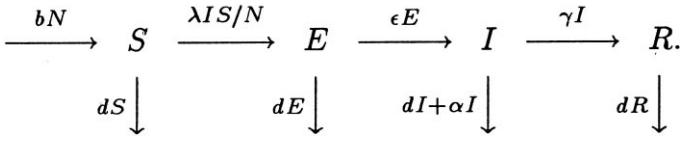
Greenhalgh [8] recently studied a class of SEIRS models that incorporate density dependence in the contact rate and natural death rate. Global stability of the disease-free equilibrium and the existence, uniqueness and local asymptotic stability of the endemic equilibrium are proved in [8]. The global stability of the endemic equilibrium, when it is unique, is unresolved in [8]. The model studied in the present paper is a special case of those considered in [8].

We prove the global stability of the unique endemic equilibrium for our model under the restriction $\alpha < \epsilon$. In addition, we present a new method for proving the local stability of the unique endemic equilibrium. Compared with the traditional approach of using Routh–Hurwitz conditions (see, for example, [8]), our method is less technical and more manageable for systems of large number of equations. Our treatment of the disease-free equilibrium and the existence and uniqueness of the endemic equilibrium is standard for models of this type. Similar methods are also used in [8]. Since our model is simpler than those in [8], we are also able to obtain a complete stability analysis for the disease-free equilibrium P_0 when $\sigma \leqslant 1$, while in [8], this analysis is done only for $\sigma < 1$.

Cook and van den Driessche [13] introduced and studied SEIRS models with two delays. Greenhalgh [8] studied Hopf bifurcations in models of SEIRS type with density dependent contact and death rates. A recent survey on SEIRS models is given in [8].

2. Model formulation

A population of size $N(t)$ is partitioned into subclasses of individuals who are susceptible, exposed (infected but not yet infectious), infectious and recovered, with sizes denoted by $S(t)$, $E(t)$, $I(t)$ and $R(t)$, respectively. The sum $E(t) + I(t)$ is the total infected population. Our assumptions on the dynamical transfer of the population are demonstrated in the diagram



The parameter $b > 0$ is the rate for natural birth and $d > 0$ that of natural death. It is assumed that all newborns are susceptible and vertical transmission can be neglected. The parameter α is the rate for disease-related death, γ is the rate for recovery and ϵ is the rate at which the exposed individuals become infective so that $1/\epsilon$ is the mean latent period. The recovered individuals are assumed to acquire permanent immunity; there is no transfer from the R class back to the S class. The force of infection is $\lambda I/N$, where λ is the effective per capita contact rate of infective individuals and the incidence rate is $\lambda IS/N$. In the limit when $\epsilon \to \infty$, or equivalently, when the mean latent period $1/\epsilon \to 0$, the SEIR model becomes a SIR model.

The following differential equations are derived based on the basic assumptions and using the transfer diagram:

$$\begin{aligned} S' &= bN - \mathrm{d}S - \lambda IS/N, \\ E' &= \lambda IS/N - (\epsilon + d)E, \\ I' &= \epsilon E - (\gamma + \alpha + d)I, \\ R' &= \gamma I - \mathrm{d}R. \end{aligned} \tag{2.1}$$

The total population size $N(t)$ can be determined by $N(t) = S(t) + E(t) + I(t) + R(t)$ or from the differential equation

$$N' = (b - d)N - \alpha I, \tag{2.2}$$

which is derived by adding the equations in (2.1). Let $s = S/N$, $e = E/N$, $i = I/N$ and $r = R/N$ denote the fractions of the classes S, E, I and R in the population, respectively. It is easy to verify that s, e, i and r satisfy the system of differential equations

$$\begin{aligned} s' &= b - bs - \lambda is + \alpha is, \\ e' &= \lambda is - (\epsilon + b)e + \alpha ie, \\ i' &= \epsilon e - (\gamma + \alpha + b)i + \alpha i^2, \\ r' &= \gamma i - br + \alpha ir, \end{aligned} \tag{2.3}$$

subject to the restriction $s + e + i + r = 1$. Note that the total population size $N(t)$ does not appear in (2.3); this is a direct result of the homogeneity of the system (2.1). Also observe that the variable r does not appear in the first three equations of (2.3). This allows us to attack (2.3) by studying the subsystem

$$\begin{aligned} s' &= b - bs - \lambda is + \alpha is, \\ e' &= \lambda is - (\epsilon + b)e + \alpha ie, \\ i' &= \epsilon e - (\gamma + \alpha + b)i + \alpha i^2, \end{aligned} \tag{2.4}$$

and determining r from $r = 1 - s - e - i$ or

$$r' = \gamma i - br + \alpha ir. \tag{2.5}$$

From biological considerations, we study (2.4) in the closed set

$$\Gamma = \{(s, e, i) \in \mathbf{R}^3_+ \mid 0 \leqslant s + e + i \leqslant 1\}, \tag{2.6}$$

where $\mathbf{R}^3_+$ denotes the non-negative cone of $\mathbf{R}^3$ including its lower dimensional faces. It can be verified that Γ is positively invariant with respect to (2.4). We denote by $\partial\Gamma$ and $\overset{\circ}{\Gamma}$ the boundary and the interior of Γ in $\mathbf{R}^3$, respectively.

The point $P_0 = (1, 0, 0) \in \Gamma$ is the *disease-free equilibrium* of (2.4) and it exists for all non-negative values of its parameters. Any equilibrium in $\overset{\circ}{\Gamma}$ corresponds to the disease being endemic and is named an *endemic equilibrium*.

In the rest of this section, we establish that (2.4) is a competitive system when $\lambda > \alpha$, a property that plays an important role in the study of the global dynamics when the disease persists. Let $x \mapsto f(x) \in \mathbf{R}^n$ be a smooth vector field defined for x in an open set $D \subset \mathbf{R}^n$. The differential equation

$$x' = f(x), \quad x \in D, \tag{2.7}$$

is said to be *competitive* in D if, for some diagonal matrix $H = \mathrm{diag}(\epsilon_1, \ldots, \epsilon_n)$ where each ϵ_i is either 1 or -1, $H(\partial f/\partial x)H$ has non-positive off-diagonal elements for all $x \in D$. If D is convex, the flow of a competitive system preserves, for $t < 0$, the partial ordering in $\mathbf{R}^n$ defined by the orthant $K = \{(x_1, \ldots, x_n) \in \mathbf{R}^n : \epsilon_i x_i \geqslant 0,\ i = 1, \ldots, n\}$ (see [14], p. 34). The concept of competitiveness used above is more general than the one in [15] in that the partial ordering is not necessarily defined by the standard orthant of $\mathbf{R}^n$. However, by a linear change of variables $y = Hx$, a competitive system as defined above can be transformed into a system that is 'competitive' in the sense of [15].

By examining its Jacobian matrix and choosing the matrix H as $H = \mathrm{diag}(-1, 1, -1)$, one can verify that, when $\lambda > \alpha$, the system (2.4) is competitive in the convex region $\overset{\circ}{\Gamma}$ with respect to the partial ordering defined by the orthant $\{(s, e, i) \in \mathbf{R}^3 : s \leqslant 0, e \geqslant 0, i \leqslant 0\}$. An important characteristic of a three-dimensional competitive system is the following Poincaré–Bendixson property.

Theorem 2.1. *Assume that $n = 3$ and D is convex. Suppose that* (2.7) *is competitive in D and that L is a non-empty compact omega limit set of* (2.7). *If L contains no equilibria, then L is a closed orbit.* (*cf.* [14], *Chapter* 3, *Theorem* 4.1)

Remark. A proof of Theorem 2.1 under the assumption that (2.7) is irreducible is given in [16]. A proof without the irreducibility requirement is first given in [15].

3. The disease-free equilibrium and its global stability

Let $\sigma = \lambda\epsilon/(\epsilon + b)(\gamma + \alpha + b)$. Following [5], σ will be called the *modified contact number*; see Section 7 for more discussion of σ. In the following result, the stability of P_0 should be understood in the sense of Lyapunov.

Theorem 3.1. *The disease-free equilibrium $P_0 = (1, 0, 0)$ of* (2.4) *is globally asymptotically stable in Γ if $\sigma \leqslant 1$; it is unstable if $\sigma > 1$, and the solutions of* (2.4) *starting sufficiently close to P_0 in Γ move away from P_0 except that those starting on the invariant s-axis approach P_0 along this axis.*

Remark. By Theorem 3.1, the disease-free equilibrium P_0 is globally stable in Γ if and only if $\sigma \leqslant 1$. An earlier result of Greenhalgh ([8], Theorem 2.3) proved the global stability of P_0 when $\sigma < 1$.

The following lemma will be used in the proof of Theorem 3.1.

Lemma 3.2. *Let $\Delta = \{(x, y) \in \mathbf{R}^2_+ \mid 0 \leqslant x + y \leqslant 1\}$ and*

$$h(x,y) = (a_1 - b_1)x + (c_1 - b_1)y + b_1.$$

Then, for any positive constants a_1, b_1 *and* c_1,

$$\max_{(x,y)\in\Delta} h(x,y) = \max\{a_1, b_1, c_1\}.$$

Proof. The affinity of h implies that its maximum in the closed set Δ is achieved at the extremal points of the boundary $\partial\Delta$. The proof is now a straightforward evaluation of h on the three vertices of the triangular set Δ. □

Proof of Theorem 3.1. Set $L = \epsilon e + (\epsilon + b)i$. Then

$$\begin{aligned} L' &= i[\lambda\epsilon s - (\epsilon + b)(\gamma + \alpha + b) + \alpha\epsilon e + \alpha(\epsilon + b)i] \\ &\leqslant i[\lambda\epsilon(1 - e - i) - (\epsilon + b)(\gamma + \alpha + b) + \alpha\epsilon e + \alpha(\epsilon + b)i] \\ &= i[h(e,i) - (\epsilon + b)(\gamma + \alpha + b)], \end{aligned} \tag{3.1}$$

where $h(e,i) = (\alpha\epsilon - \lambda\epsilon)e + (\alpha(\epsilon + b) - \lambda\epsilon)i + \lambda\epsilon$. Applying Lemma 3.2 to $h(e,i)$ leads to

$$\begin{aligned} L' &\leqslant i[\max\{\alpha\epsilon,\ \lambda\epsilon,\ \alpha(\epsilon + b)\} - (\epsilon + b)(\gamma + \alpha + b)] \\ &\leqslant 0 \quad \text{if } \sigma \leqslant 1. \end{aligned}$$

If $L' = 0$ and $i \neq 0$, then

$$\max\{\alpha\epsilon, \lambda\epsilon, \alpha(\epsilon + b)\} = (\epsilon + b)(\gamma + \alpha + b)$$

and (3.1) becomes an equality. Thus, $L' = 0$ only if either (1) $i = 0$, or (2) $\sigma = 1$ and $s + e + i = 1$. The maximum invariant set in $\{(s,e,i) \in \Gamma \mid L' = 0\}$ is the singleton $\{P_0\}$. The global stability of P_0 when $\sigma \leqslant 1$ follows from LaSalle's Invariance Principle ([17], Chapter 2, Theorem 6.4).

If $\sigma > 1$, then $L' > 0$ for s sufficiently close to 1 except when $e = i = 0$. Solutions starting sufficiently close to P_0 leave a neighborhood of P_0 except those on the invariant s-axis, on which (2.4) reduces to $s' = b - bs$ and thus $s(t) \to 1$, as $t \to \infty$. This establishes the theorem. □

Theorem 3.1 completely determines the global dynamics of (2.4) in Γ for the case $\sigma \leqslant 1$. Its epidemiological implication is that the infected fraction (the sum of the latent and the infectious fractions) of the population vanishes in time so the disease dies out. In the rest of this section, we show that the disease persists when $\sigma > 1$. We say the disease is *endemic* if the infected fraction of the population persists above a certain positive level for sufficiently large time. The endemicity of disease can be well captured and analyzed through the notion of uniform persistence. System (2.4) is said to be *uniformly persistent* (see [11,18,19]) if there exists a constant $0 < c < 1$ such that any solution $(s(t), e(t), i(t))$ with $(s(0), e(0), i(0)) \in \overset{\circ}{\Gamma}$ satisfies

$$\min\{\liminf_{t\to\infty} s(t),\quad \liminf_{t\to\infty} e(t),\quad \liminf_{t\to\infty} i(t)\} \geqslant c. \tag{3.2}$$

The disease is endemic if (2.4) is uniformly persistent. In this case, both the infective and the latent fractions persist above a certain positive level. Weaker notions of persistence have been defined and used in the literature of population dynamics (see [19]). One may choose to define endemicity of the disease using one of the weaker notions of persistence. However, as the following result shows, persistence of (2.4) in any reasonable sense is equivalent to the uniform persistence defined above.

Proposition 3.3. *System* (2.4) *is uniformly persistent in* $\overset{\circ}{\Gamma}$ *if and only if* $\sigma > 1$.

Proof. The necessity of $\sigma > 1$ follows from Theorem 3.1 and the fact that the asymptotic stability of P_0 precludes any kind of persistence. The sufficiency of the condition $\sigma > 1$ follows from a uniform persistence result, Theorem 4.3, in [20]. To demonstrate that (2.4) satisfies all the conditions of Theorem 4.3 in [20] when $\sigma > 1$, choose $X = \mathbf{R}^3$ and $E = \Gamma$. The maximal invariant set N on the boundary $\partial\Gamma$ is the singleton $\{P_0\}$ and is isolated. Thus, the hypothesis (H) of [20] holds for (2.4). The proposition is proved by observing that, in the setting of (2.4), the necessary and sufficient condition for uniform persistence in Theorem 4.3 of [20] is equivalent to P_0 being unstable. □

Remark. Theorem 3.1 and Proposition 3.3 establish the modified contact number σ as a sharp threshold parameter; if $\sigma \leqslant 1$ the disease dies out, if $\sigma > 1$ the disease remains endemic.

4. Existence and uniqueness of an endemic equilibrium P^*

Global stability of P_0 in Γ when $\sigma \leqslant 1$ precludes the existence of equilibria other than P_0; the study of endemic equilibria is restricted to the case $\sigma > 1$. We remark that $\sigma > 1$ implies $\lambda > \alpha$. This relation will be assumed throughout this and the next two sections.

The coordinates of an equilibrium $P^* = (s^*, e^*, i^*) \in \overset{\circ}{\Gamma}$ satisfy

$$\begin{aligned} b - bs - \lambda is + \alpha is &= 0, \\ \lambda is - (\epsilon + b)e + \alpha ie &= 0, \\ \epsilon e - (\gamma + \alpha + b)i + \alpha i^2 &= 0 \end{aligned} \tag{4.1}$$

and also $s^* > 0$, $e^* > 0$ and $i^* > 0$. Adding the above equations leads to

$$(b - \alpha i^*)(1 - s^* - e^* - i^*) = \gamma i^*,$$

which gives the following range of i^*

$$0 < i^* < \min\{1, b/\alpha\}. \tag{4.2}$$

Eliminating s and e from (4.1), we see that i^* satisfies

$$f(i^*) = \sigma, \tag{4.3}$$

where

$$f(i) = \left(1 - \frac{\alpha}{\epsilon + b} i\right)\left(1 - \frac{\alpha}{\gamma + \alpha + b} i\right)\left(1 + \frac{\lambda - \alpha}{b} i\right) \tag{4.4}$$

and $\sigma = \lambda\epsilon/(\epsilon + b)(\gamma + \alpha + b)$ is the modified contact number defined in Section 3. Furthermore, s^* and e^* can be uniquely determined from i^* by

$$s^* = \frac{b}{b + \lambda i^* - \alpha i^*} \quad \text{and} \quad e^* = \frac{\gamma + \alpha + b - \alpha i^*}{\epsilon} i^*, \tag{4.5}$$

respectively. Eq. (4.3) is cubic and the existence as well as the uniqueness of $i^* \in (0, b/\alpha)$ have to be established without being able to solve for i^* explicitly; this technical difficulty later forces us to develop a new method for the proof of the local stability of P^* in the next section.

The three roots of $f(i)$ are $i_1 = (\epsilon + b)/\alpha$, $i_2 = (\gamma + \alpha + b)/\alpha$ and $i_3 = -b/(\lambda - \alpha)$. They all lie outside $[0, b/\alpha]$ when $\sigma > 1$. Furthermore, $f(0) = 1$ and $f(b/\alpha) = \sigma((\alpha + \gamma)/\alpha) > \sigma$. If $b > \alpha$, then $f(1) \geqslant \sigma((\gamma + b)/b) > \sigma$. These observations lead to the conclusion that, when $\sigma > 1$, the line $y = \sigma$ has exactly one intersection $(i^*, f(i^*))$ with the graph of $f(i)$ that satisfies (4.2) (see Fig. 1). We thus have established the following result.

Theorem 4.1. *Suppose that* $\sigma > 1$. *Then* (2.4) *has a unique interior equilibrium* $P^* = (s^*, e^*, i^*)$ *and its coordinates satisfy* (4.3)–(4.5).

Remark. The results in Theorem 4.1 are also obtained in [8], Theorem 2.3 (ii) by a similar method. However, the method is more clearly illustrated in our simpler model.

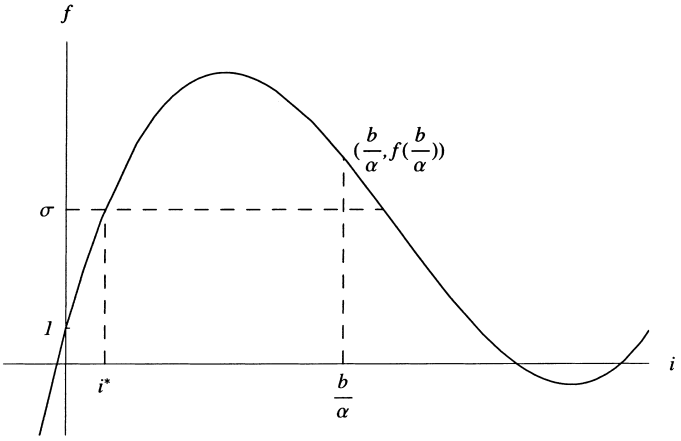


Fig. 1. The existence and uniqueness of i^* in the interval [0, b/α].

5. Local asymptotic stability of the endemic equilibrium P^*

Throughout this section, the relation $\lambda > \alpha$ is assumed since we are concerned only with the interior equilibrium P^*.

To show the asymptotic stability of the equilibrium P^*, we use the method of first approximation. The Jacobian matrix of (2.4) at a point $P = (s, e, i) \in \Gamma$ is

$$J(P) = \begin{bmatrix} -b - \lambda i + \alpha i & 0 & -\lambda s + \alpha s \\ \lambda i & -(\epsilon + b) + \alpha i & \lambda s + \alpha e \\ 0 & \epsilon & -(\gamma + \alpha + b) + 2\alpha i \end{bmatrix}. \tag{5.1}$$

We prove that the matrix $J(P^*)$ is *stable*, namely, all its eigenvalues have negative real parts. This is routinely done by verifying the Routh–Hurwitz conditions. Since the explicit coordinates of P^* are not available, verification of the inequalities in the Routh–Hurwitz conditions for $J(P^*)$ is technically very difficult. We first develop a new criteria for the stability of matrices.

Using the spectral properties of the second compound matrices (see the Appendix A), we prove the following result.

Lemma 5.1. *Let A be an $m \times m$ matrix with real entries. For A to be stable, it is necessary and sufficient that*
1. *the second compound matrix $A^{[2]}$ is stable,*
2. $(-1)^m \det(A) > 0$.

Proof. The necessity of the conditions 1. and 2. follows directly from the proposition in the Appendix A. Furthermore, by the same proposition, the stability of $A^{[2]}$ implies that at most one eigenvalue of A can have a non-negative real part. It is then simple to see that the determinant condition $(-1)^m \det(A) > 0$ preludes the case of exactly one non-negative eigenvalue. This completes the proof. □

Theorem 5.2. *If $\sigma > 1$, then* (2.4) *has a unique equilibrium P^* in $\overset{\circ}{\Gamma}$ and P^* is asymptotically stable.*

Remark. The local stability of the unique P^* is also proved for a more general model in [8], Theorem 2.3 using the Routh–Hurwitz conditions.

Proof. It remains to show that $J(P^*)$ satisfies the conditions (1) and (2) of Lemma 5.1. The second compound matrix $J^{[2]}(P)$ of the Jacobian matrix $J(P)$ is (see the Appendix A)

$$\begin{bmatrix} -2b - \lambda i - \epsilon + 2\alpha i & \lambda s + \alpha e & \lambda s - \alpha s \\ \epsilon & -2b - \lambda i - \gamma - \alpha + 3\alpha i & 0 \\ 0 & \lambda i & -2b - \epsilon - \gamma - \alpha + 3\alpha i \end{bmatrix}. \tag{5.2}$$

For $P^* = (s^*, e^*, i^*)$ and the diagonal matrix $E = \mathrm{diag}(i^*, e^*, s^*)$, the matrix $J^{[2]}(P^*)$ is similar to $EJ^{[2]}(P^*)E^{-1}$

$$\begin{bmatrix} -2b - \lambda i^* - \epsilon + 2\alpha i^* & \frac{\lambda i^* s^*}{e^*} + \alpha i^* & \lambda i^* - \alpha i^* \\ \frac{\epsilon e^*}{i^*} & -2b - \lambda i^* - \gamma - \alpha + 3\alpha i^* & 0 \\ 0 & \frac{\lambda i^* s^*}{e^*} & -2b - \epsilon - \gamma - \alpha + 3\alpha i^* \end{bmatrix}.$$

The matrix $J^{[2]}(P^*)$ is stable if and only if $EJ^{[2]}(P^*)E^{-1}$ is stable, for similarity preserves the eigenvalues. Since the diagonal elements of the matrix $EJ^{[2]}(P^*)E^{-1}$ are negative, an easy argument using Geršgorin discs shows that it is stable if it is diagonally dominant in rows. For a proof of Geršgorin's theorem, we refer the reader to [21]. Set $\mu = \max\{\cdots\}$, where

$$\begin{aligned} g_1 &= -2b - \epsilon + 2\alpha i^* + \frac{\lambda i^* s^*}{e^*}, \\ g_2 &= -2b - \lambda i^* - \gamma - \alpha + 3\alpha i^* + \frac{\epsilon e^*}{i^*}, \\ g_3 &= -2b - \epsilon - \gamma - \alpha + 3\alpha i^* + \frac{\lambda i^* s^*}{e^*}. \end{aligned} \tag{5.3}$$

Eq. (4.1) can be rewritten as

$$\begin{aligned} &\frac{b}{s^*} = b + \lambda i^* - \alpha i^*, \\ &\frac{\lambda i^* s^*}{e^*} = (\epsilon + b) - \alpha i^*, \\ &\frac{\epsilon e^*}{i^*} = \gamma + \alpha + b - \alpha i^*. \end{aligned} \tag{5.4}$$

Substituting (5.4) into (5.3) yields

$$\mu = \max\{-b + \alpha i^*,\ -b - \lambda i^* + 2\alpha i^*,\ -b - \gamma - \alpha + 2\alpha i^*\}.$$

Then, using (4.2) and the relation $\lambda < \alpha$, we have $\mu < 0$, which implies the diagonal dominance as claimed and thus verifies the first condition of Lemma 5.1.

Using (5.1) and (5.4), we have

$$\begin{aligned} \det(J(P^*)) &= \begin{vmatrix} -\frac{b}{s^*} & 0 & \frac{bs^* - b}{i^*} \\ \lambda i^* & -\frac{\lambda i^* s^*}{e^*} & \lambda s^* + \alpha e^* \\ 0 & \epsilon & -\frac{\epsilon e^*}{i^*} + \alpha i^* \end{vmatrix} \\ &= -\lambda b\epsilon(1 - s^*) + \lambda b i^* \frac{\alpha i^*}{e^*} + \frac{b\alpha\epsilon\ e^*}{s^*} \\ &= -\lambda b\epsilon(1 - s^*) + \lambda b\epsilon i^* \frac{\alpha i^*}{\epsilon e^*} + \lambda b\epsilon e^* \frac{\alpha}{\lambda s^*} \\ &\leqslant -\lambda b\epsilon(1 - s^* - i^* - e^*) < 0, \end{aligned}$$

since $\epsilon e^*/i^* = \gamma + \alpha + b - \alpha i^* > \alpha$ and $\lambda s^* = (\gamma + \alpha + b - \alpha i^*)(\epsilon + b - \alpha i^*)/\epsilon > \alpha$. This verifies the second condition of Lemma 5.1 and completes the proof. □

6. Global stability of the endemic equilibrium P^*

In this section, we establish that all solutions of (2.4) in $\overset{\circ}{\Gamma}$ converge to P^* when $\sigma > 1$, which, together with the local stability of P^*, implies that P^* is globally asymptotically stable in $\overset{\circ}{\Gamma}$. Note that the relation $\lambda > \alpha$ holds when $\sigma > 1$ and thus (2.4) is competitive from Section 2. The following strong Poincaré–Bendixson property follows from Theorem 2.1. Its proof is the same as that of Theorem 4.2 of [9] and thus is omitted.

Theorem 6.1. *Suppose that $\sigma > 1$. Then any non-empty compact omega limit set of* (2.4) *in $\overset{\circ}{\Gamma}$ is either a closed orbit or the endemic equilibrium P^*.*

In the absence of closed orbits, by Theorem 6.1, all trajectories in $\overset{\circ}{\Gamma}$ converge to P^* when $\sigma > 1$. This leads to the following result.

Corollary 6.2. *Assume that $\sigma > 1$. Then the unique endemic equilibrium P^* is globally asymptotically stable in $\overset{\circ}{\Gamma}$ if* (2.4) *has no non-constant periodic solutions.*

The key to verifying the global stability of P^* is to rule out the existence of periodic solutions. This is achieved by showing that any periodic solution to (2.4) is orbitally asymptotically stable. A periodic solution $x = p(t)$ to the autonomous system (2.7) in $\mathbf{R}^n$ with least period $\omega > 0$ and orbit $\mathcal{O} = \{p(t): 0 \leqslant t < \omega\}$ is said to be *orbitally stable* if, for each $\overline{\epsilon} > 0$, there exists $\delta > 0$ such that any solution $x(t)$, for which the distance of $x(0)$ from $\mathcal{O}$ is less than δ, remains at a distance less than $\overline{\epsilon}$ from $\mathcal{O}$ for all $t \geqslant 0$. It is *asymptotically orbitally stable with asymptotic phase* if it is orbitally stable and there exists $\overline{b} > 0$ such that, any solution, for which the distance of $x(0)$ from $\mathcal{O}$ is less than $\overline{b}$, satisfies $|x(t) - p(t-\tau)| \to 0$ as $t \to \infty$ for some τ which may depend on $x(0)$ (see [22]). The following stability criterion for periodic solutions of a general autonomous system (2.7) is given by Muldowney [12]. Let $\partial f^{[2]}/\partial x$ denote the second additive compound matrix of the Jacobian matrix $\partial f/\partial x$.

Theorem 6.3. *A sufficient condition for a periodic orbit $\mathcal{O} = \{p(t): 0 \leqslant t < \omega\}$ of* (2.7) *to be asymptotically orbitally stable with asymptotic phase is that the periodic linear system*

$$z'(t) = \frac{\partial f^{[2]}}{\partial x}(p(t))z(t), \tag{6.1}$$

is asymptotically stable.

Applying Theorem 6.3 to system (2.4) we can prove the following result.

Proposition 6.4. *Any non-constant periodic solution to* (2.4), *if one exists, is asymptotically orbitally stable with asymptotic phase provided that $\alpha \leqslant \epsilon$.*

Proof. Using the matrix $J^{[2]}(P)$ in (5.2), we can write the second compound system (6.1) for (2.4) with respect to a solution $(s(t), e(t), i(t))$ as

$$\begin{aligned} X' &= -(2b+\lambda i+\epsilon-2\alpha i)X+(\lambda s+\alpha e)Y+(\lambda s-\alpha s)Z,\\ Y' &= \epsilon X-(2b+\lambda i+\gamma+\alpha-3\alpha i)Y,\\ Z' &= \lambda i Y-(2b+\epsilon+\gamma+\alpha-3\alpha i)Z. \end{aligned} \tag{6.2}$$

Let

$$V(X,Y,Z;s,e,i)=\sup\left\{|X|,\ \frac{e}{i}\left(|Y|+\frac{\lambda-\alpha}{\lambda}|Z|\right)\right\}. \tag{6.3}$$

Suppose that the solution $(s(t), e(t), i(t))$ is periodic of least period $\omega > 0$ and that $(s(0), e(0), i(0)) \in \overset{\circ}{\Gamma}$. Then its orbit $\mathcal{O}$ is at a positive distance from the boundary $\partial\Gamma$; there exists a constant $c > 0$ such that

$$V(X,Y,Z;s,e,i) \geqslant c|(X,Y,Z)|, \tag{6.4}$$

for all $(X, Y, Z) \in \mathbf{R}^3$ and $(s, e, i) \in \mathcal{O}$. Let $(X(t), Y(t), Z(t))$ be a solution to (6.2) and $V(t) = V(X(t), Y(t), Z(t); s(t), e(t), i(t))$. The right-hand derivative of $V(t)$ exists and its calculation is described in [23]. Direct calculations lead to the following differential inequalities:

$$\begin{aligned} D_+|X(t)| \leqslant\ & -(2b+\lambda i+\epsilon-2\alpha i)|X(t)|\\ & +(\lambda s+\alpha e)|Y(t)|+(\lambda s-\alpha s)|Z(t)|\\ =\ & -(2b+\lambda i+\epsilon-2\alpha i)|X(t)|+\left(\frac{\lambda s i}{e}+\alpha i\right)\frac{e}{i}|Y(t)|\\ & +(\lambda-\alpha)\frac{si}{e}\frac{e}{i}|Z(t)|, \end{aligned} \tag{6.5}$$

and

$$D_+|Y(t)| \leqslant \epsilon|X(t)|-(2b+\lambda i+\gamma+\alpha-3\alpha i)|Y(t)|, \tag{6.6}$$

$$D_+|Z(t)| \leqslant \lambda i|Y(t)|-(2b+\epsilon+\gamma+\alpha-3\alpha i)|Z(t)|. \tag{6.7}$$

Using (6.6) and (6.7), as well as the relations $\alpha \leqslant \epsilon$ and $i < 1$, we have

$$\begin{aligned} & D_+\frac{e}{i}\left(|Y(t)|+\frac{\lambda-\alpha}{\lambda}|Z(t)|\right)\\ &= \left(\frac{e'}{e}-\frac{i'}{i}\right)\frac{e}{i}\left(|Y(t)|+\frac{\lambda-\alpha}{\lambda}|Z(t)|\right)+\frac{e}{i}D_+\left(|Y(t)|+\frac{\lambda-\alpha}{\lambda}|Z(t)|\right)\\ &\leqslant \frac{\epsilon e}{i}|X(t)|+\left(\frac{e'}{e}-\frac{i'}{i}-2b-\gamma-\alpha+2\alpha i\right)\frac{e}{i}\left(|Y(t)|+\frac{\lambda-\alpha}{\lambda}|Z(t)|\right). \end{aligned} \tag{6.8}$$

Relations (6.6) and (6.8) lead to

$$D_+V(t) \leqslant \max\{g_1(t), g_2(t)\}V(t), \tag{6.9}$$

where

$$g_1(t) = -2b - \lambda i - \epsilon + 3\alpha i + \frac{\lambda s i}{e}, \tag{6.10}$$

$$g_2(t) = \frac{e'}{e} - \frac{i'}{i} - 2b - \gamma - \alpha + 2\alpha i + \frac{\epsilon e}{i}. \tag{6.11}$$

Rewriting (2.4), we find that

$$\frac{\lambda s i}{e} + \alpha i = \frac{e'}{e} + \epsilon + b, \tag{6.12}$$

$$\frac{\epsilon e}{i} + \alpha i = \frac{i'}{i} + \gamma + \alpha + b. \tag{6.13}$$

Moreover, the periodic function $r(t) = 1 - s(t) - e(t) - i(t)$ satisfies (2.5) which leads to

$$\frac{r'}{r} = \frac{\gamma i}{r} - b + \alpha i. \tag{6.14}$$

From (6.10)–(6.14),

$$\max\{g_1(t), g_2(t)\} \leqslant \frac{e'(t)}{e(t)} + \frac{r'(t)}{r(t)} - \frac{\gamma i(t)}{r(t)},$$

and thus

$$\int_0^\omega \max\{g_1(t), g_2(t)\}\, \mathrm{d}t \leqslant \log e(t)\big|_0^\omega + \log r(t)\big|_0^\omega - \int_0^\omega \frac{\gamma i(t)}{r(t)}\, \mathrm{d}t = -\gamma C$$

by the periodicity of $e(t)$ and $r(t)$, where $C = \int_0^\omega (i(t)/r(t))\, \mathrm{d}t > 0$. This and (6.9), imply that $V(t) \to 0$ as $t \to \infty$, and in turn that $(X(t), Y(t), Z(t)) \to 0$ as $t \to \infty$ by (6.4). As a result, the second compound system (6.2) is asymptotically stable and the periodic solution $(s(t), e(t), i(t))$ is asymptotically orbitally stable with asymptotic phase by Theorem 6.3. □

Now, we are ready to prove the global stability of the endemic equilibrium P^*.

Theorem 6.5. *Suppose that $\sigma > 1$. Then the unique endemic equilibrium P^* is globally asymptotically stable in $\mathring{\Gamma}$ provided that $\alpha \leqslant \epsilon$. Moreover, P^* attracts all trajectories in Γ except those on the invariant s-axis which converge to P_0 along this axis.*

Proof. By inspecting the vector field given by (2.4), we see that all trajectories originating from the boundary $\partial\Gamma$ enter $\mathring{\Gamma}$ except those on the s-axis which converge to P_0 along this invariant axis. It remains to show that P^* attracts all points in $\mathring{\Gamma}$. Let $U \subset \mathring{\Gamma}$ be the set of points that are attracted by P^*. Then U is

an open subset of $\overset{\circ}{\Gamma}$ by the asymptotic stability of P^*. The theorem is proved if we establish that $\overset{\circ}{\Gamma} \subset U$. Assume the contrary; then the boundary ∂U of U has a non-empty intersection $\mathscr{I}$ with $\overset{\circ}{\Gamma}$. Since both U and its closure $\overline{U}$ are invariant and U is open, $\partial U = \overline{U} - U$ is also invariant. As the intersection of ∂U with the positively invariant $\overset{\circ}{\Gamma}$, $\mathscr{I}$ is positively invariant and thus $\mathscr{I}$ contains a non-empty compact omega limit set Ω. By the uniform persistence, we must have $\Omega \cap \partial\Gamma = \emptyset$. Since it contains no equilibria, by Theorem 6.1 and Proposition 6.4, Ω is a closed orbit and is asymptotically orbitally stable. We thus obtain a contradiction since Ω belongs to the alpha limit set of a trajectory in U. This completes the proof. □

Under the condition $\alpha \leqslant \epsilon$, Theorem 6.5 describes the global dynamics of (2.4) when the disease is endemic. Together with Theorem 3.1, this completely determines the global dynamics of (2.4) and establish the modified contact number σ as a sharp threshold parameter. When $\sigma \leqslant 1$, the disease dies out in the way that the infected fractions vanish, whereas when $\sigma > 1$, the disease becomes endemic so that the infected fractions approach a positive constant level. The condition $\alpha \leqslant \epsilon$ holds in both limiting cases when $\epsilon \to \infty$ or $\alpha = 0$. Theorem 6.5 thus contains, as special cases, the global stability results of SIR models with varying population in [3,5] and of SEIR models with constant population in [9].

7. The dynamics of the population sizes

We now turn to the dynamics of $(S(t), E(t), I(t), R(t))$ and $N(t) = S(t) + E(t) + I(t) + R(t)$, which are governed by systems (2.1) and (2.2). The fact that R does not appear in the first three equations in (2.1) allows us to study the equivalent system

$$\begin{aligned} S' &= bN - \mathrm{d}S - \lambda IS/N, \\ E' &= \lambda IS/N - (\epsilon + d)E, \\ I' &= \epsilon E - (\gamma + \alpha + d)I, \\ N' &= (b - d)N - \alpha I, \end{aligned} \tag{7.1}$$

in its feasible region

$$\Sigma = \{(S, E, I, N) \in \mathbf{R}^4_+ \mid 0 \leqslant S + E + I \leqslant N\}.$$

If $b < d$ and $\alpha \geqslant 0$, or if $b \leqslant d$ and $\alpha > 0$, (7.1) implies that $N(t) \to 0$ monotonically as $t \to \infty$ for all solutions with $E(0) + I(0) > 0$, namely, when the disease is initially present. If $b = d$ and $\alpha = 0$, $N(t)$ remains constant so that (7.1) reduces to a SEIR model with constant population whose dynamics have been completely determined in [9]. In the rest of this section, we assume that $b > d$ and $\alpha > 0$.

Let

$$f(x) = \left(1 - \frac{\alpha}{\epsilon + b}x\right)\left(1 - \frac{\alpha}{\gamma + \alpha + b}x\right)\left(1 + \frac{\lambda - \alpha}{b}x\right), \tag{7.2}$$

be the cubic function defined in (4.4). Set $\overline{\sigma} = f((b-d)/\alpha)$ and $\sigma' = \lambda\epsilon/(\epsilon + d)(\gamma + \alpha + d)$. Both parameters $\overline{\sigma}$ and σ' play key roles in the dynamics of the population sizes. In the limiting case when $\epsilon \to \infty$, σ' takes the form $\lambda/(\gamma + \alpha + d)$ which is called the *contact number* in the literature (it is denoted by θ in [5]), since it can be regarded as the average number of effective contacts of an infective during the death modified mean infectious period $1/(\gamma + \alpha + d)$. For the same reason, σ' will be called the contact number and $\sigma = \lambda\epsilon/(\epsilon + b)(\gamma + \alpha + b)$ the modified contact number for our SEIR model. As mentioned above, if $\alpha = 0$ and $b = d$, the total population remains a constant, and subsequently, σ' and σ are identical and agree with the contact number defined for SEIR models with constant total population, see [7,8].

Proposition 7.1. *System* (7.1) *has only one equilibrium* (0,0,0,0) *if* $\overline{\sigma} \neq \sigma$ *and has a line of equilibria* $((N^*/\sigma'), ((\gamma + d + \alpha)(b-d)/(\alpha\epsilon))N^*, ((b-d)/\alpha)N^*, N^*)$ *if* $\overline{\sigma} = \sigma$, *where* N^* *is an arbitrary positive number*.

Proof. An equilibrium (S^*, E^*, I^*, N^*) with positive entries exists if and only if the entries satisfy

$$\frac{S^*}{N^*} = \frac{(\epsilon + d)(\gamma + \alpha + d)}{\lambda\epsilon}, \quad \frac{E^*}{N^*} = \frac{\gamma + \alpha + d}{\epsilon}\frac{I^*}{N^*}, \quad \frac{I^*}{N^*} = \frac{b-d}{\alpha},$$

and

$$b - d\frac{S^*}{N^*} - \lambda\frac{I^*}{N^*}\frac{S^*}{N^*} = 0.$$

Eliminating S^*, I^* and N^* from these equations leads to the following condition

$$\frac{(\epsilon + d)(\gamma + \alpha + d)}{\lambda\epsilon}\left[d + \frac{\lambda}{\alpha}(b-d)\right] = b,$$

which is equivalent to

$$\overline{\sigma} = f\left(\frac{b-d}{\alpha}\right) = \left(1 - \frac{b-d}{\epsilon + b}\right)\left(1 - \frac{b-d}{\gamma + \alpha + b}\right)\left(1 + \frac{(\lambda - \alpha)(b-d)}{b\alpha}\right)$$
$$= \sigma,$$

completing the proof. □

Remark. In the limiting case when $\epsilon \to \infty$, the condition $\overline{\sigma} = \sigma$ reduces to the condition $\phi = 1$ in [5] with $\delta = 0$.

If the modified contact number $\sigma \leqslant 1$, then the infected fractions of the population vanish. The disease does not suppress the natural growth of the host population so that $N(t)$ and $S(t)$ grow to infinity exponentially at an exponential rate $b-d$ as $t \to \infty$. The dynamics of the infected population $(E(t), I(t))$ depend on the contact number σ', as we demonstrate in the following theorem.

Theorem 7.2. *Suppose $\sigma \leqslant 1$. Then $N(t), S(t) \to \infty$ exponentially with exponential rate $b-d$ as $t \to \infty$. In addition, $(E(t), I(t), R(t)) \to (0,0,0)$ or (∞, ∞, ∞) if $\sigma' < 1$ or $\sigma' > 1$, respectively.*

Proof. From Theorem 3.1, $\sigma \leqslant 1$ implies that $(s(t), e(t), i(t), r(t)) \to (1,0,0,0)$ exponentially as $t \to \infty$. The claim on the exponential growth of $N(t)$ follows from

$$N'(t) = [(b-d) - \alpha i(t)]N(t),$$

and the fact that $i(t) \to 0$ as $t \to \infty$ (see [22]). The behavior of $S(t)$ follows from the fact that $s(t) = S(t)/N(t) \to 1$ as $t \to \infty$, independent of the contact number σ'. To see the behavior of $E(t), I(t)$ and $R(t)$, consider the equations for E, I and R

$$\begin{bmatrix} E \\ I \\ R \end{bmatrix}' = \left(\begin{bmatrix} -(\epsilon + d) & \lambda & 0 \\ \epsilon & -(\gamma + \alpha + d) & 0 \\ 0 & \gamma & -d \end{bmatrix} + \begin{bmatrix} 0 & \lambda(s-1) & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \right) \begin{bmatrix} E \\ I \\ R \end{bmatrix}, \tag{7.3}$$

which is a perturbation of a linear system. The solutions to the principal part of (7.3) behave as claimed in the theorem, as do those for the perturbed system (7.3) since the perturbation decays exponentially as $t \to \infty$ (see [22], Chapter 3, Theorem 2.3). □

If the modified contact number $\sigma > 1$, the disease becomes endemic as the infected fraction approaches a constant level. The extent to which the disease suppresses the natural growth of the host population is determined by the ratio $\sigma/\overline{\sigma}$, as we prove in the following theorem.

Theorem 7.3. *Suppose $\sigma > 1$. Assume that $\alpha \leqslant \epsilon$. Then $(S(t), E(t), I(t), R(t), N \times (t)) \to (\infty, \infty, \infty, \infty, \infty, \infty)$ or $(0,0,0,0,0,0)$ depending on whether $\sigma < \overline{\sigma}$ or $\sigma > \overline{\sigma}$, respectively. If $\sigma = \overline{\sigma}$, the line of equilibria (S^*, E^*, I^*, N^*) described in Theorem* 7.1 *exists and is foliated with fibres of* 3*-dimensional stable manifolds.*

Proof. Since $\overline{\sigma} = f((b-d)/\alpha)$ and $\sigma = f(i^*)$, using the graph of f that is rigorously established at the end of Section 4 (see Fig. 1), we see that $\overline{\sigma} > \sigma$ (or

$\overline{\sigma} < \sigma$) is equivalent to the relation $b - d - \alpha i^* > 0$ (or $b - d - \alpha i^* < 0$). The first statement of the theorem follows from the global stability of (s^*, e^*, i^*, r^*) in Theorem 6.5 and the equation

$$N'(t) = [(b - d - \alpha i^*) - \alpha(i(t) - i^*)]N(t).$$

If $\sigma = \overline{\sigma}$, then $b - d - \alpha i(t) = -\alpha(i(t) - i^*) \to 0$ exponentially by the asymptotic stability of (s^*, e^*, i^*). This implies the convergence of the integral $\int_0^\infty (b - d - \alpha i(t))\,\mathrm{d}t$ and hence that $N(t) \to N^*$ as $t \to \infty$, where N^* depends on $N(0)$. Replacing N by N^* in the first three equations of (7.1) and scaling N^* to 1 by considering $\overline{s} = S/N$, $\overline{e} = E/N$ and $\overline{i} = I/N$, we arrive at a system that is the same as (2.4) for (s, e, i) with $\sigma > 1$. This proves the last claim of the theorem. The replacement of $N(t)$ by N^* is guaranteed by the theory of asymptotically autonomous systems (see [24,25]). □

Remark. From the proof of Theorem 7.3, we see that the relation $\overline{\sigma} > \sigma$ is equivalent to $b - d - \alpha i^* > 0$. Biologically, Theorem 7.3 implies that an endemic disease suppresses the natural growth of the host population and the extent of this suppression is directly related to i^*, the level of the infectious fraction in the population. If i^* is high compared with the natural growth rate, more specifically, if $i^* > (b - d)/\alpha$, then the disease can suppress the population growth enough to cause it to decline to zero; if i^* is sufficient low, then it can only manage to lower the exponential growth rate of the population; if $i^* = (b - d)/\alpha$, then the disease can regulate an otherwise exponentially growing population so that the total population settles to a constant level. In Fig. 2, trajectories from numerical solutions are depicted for the case $i^* = (b - d)/\alpha$.

8. Discussion

This paper has considered a SEIR model that incorporates exponential natural birth and death, as well as disease-caused death, so that the total

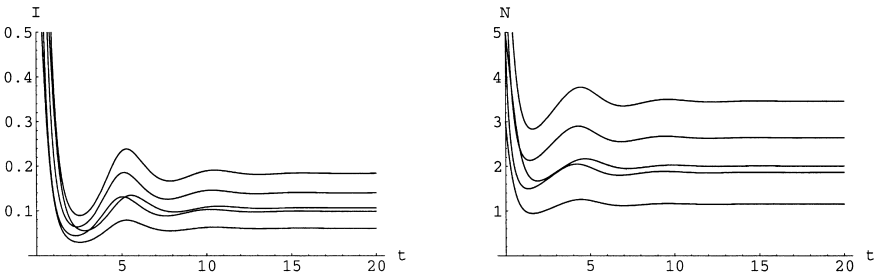


Fig. 2. A Mathematica plot showing that the solutions $I(t)$ and $N(t)$ of (7.1) converge to finite limits, when $\sigma = \overline{\sigma}$. The parameter values are $\alpha = 6$, $b = 0.5$, $\epsilon = 4$, $\gamma = 1.5$, $\lambda = 20$, $d = 0.18$, $\sigma = \overline{\sigma} = 2.22 > 1$.

population size may vary in time. The incidence rate is of the proportionate mixing type frequently used in the literature. The asymptotic behavior of this multidimensional model has been determined as a function of the basic parameters of the system.

The homogeneity of the vector field of the model suggests the way of analyzing the global dynamics; the global behavior of the derived system for the fractions (s, e, i, r) is analyzed, which in turn determines the behavior of the population sizes (S, E, I, R) and N. For epidemic models with varying total population, the endemicity of the disease can be understood as the infected population remains above a positive level in actual size or in fraction and thus needs to be clearly defined. In the present paper, the endemicity has been defined using the infected fraction of the population. For a disease with non-negligible latency, the infected fraction includes both the latent and infectious fractions. Three threshold parameters have been identified: they are the modified contact number $\sigma = \lambda\epsilon/(\epsilon + b)(\gamma + \alpha + b)$, the contact number $\sigma' = \lambda\epsilon/(\epsilon + d)(\gamma + \alpha + d)$, and the parameter $\overline{\sigma} = f((b - d)/\alpha)$. The modified contact number σ determines whether the disease can become endemic. If $\sigma \leqslant 1$, the disease dies out in the sense that the infected fraction disappears from the population. In this case, the contact number σ' determines the dynamics of the population size. Note that if $\sigma' > 1$, then the infected population grows exponentially in size but approaches zero in fraction. If $\sigma > 1$, then the disease becomes endemic. In this case, the parameter $\overline{\sigma}$ determines the extent that disease can regulate the growth of the host population. The technical condition $\alpha \leqslant \epsilon$ used in Theorem 6.4 is satisfied if the disease has a low virulence or causes a short latent period. This condition includes both limiting cases when $\alpha = 0$ and $\epsilon \to \infty$; thus the results in the present paper include as special cases the earlier results on SIR and SEIR models with constant population and SIR models with varying population and proportionate mixing term. The threshold parameters σ, σ' and $\overline{\sigma}$ generalize the relevant threshold parameters used in these earlier models.

As indicated earlier, some of the results here can be deduced from the paper of Greenhalgh [8] who considered a more general model. The global stability of the disease-free equilibrium P_0 for the fractions is proved under the condition $\sigma < 1$ in [8]; for our model, we are able to include the case when $\sigma = 1$. The existence and uniqueness of the endemic equilibrium P^* for the fractions are obtained in [8] using a similar method. The local stability for P^* is proved in [8] using Routh–Hurwitz criteria. In the present paper, the local stability of P^* is proved using a new stability criterion. Verification of our stability conditions is less technical and more manageable, especially when the number of equations is large, than the Routh–Hurwitz conditions used in [8]. Using a new method developed in [26], the global stability of P^*, which was unresolved in [8], is proved in the present paper under the restriction $\alpha \leqslant \epsilon$. The same method has also been successfully applied in [9] to a SEIR model with constant population.

It seems very hopeful that this method can be applied to a wider class of nonlinear models.

The epidemiological and demographical phenomena as well as their interaction as observed in our model is reminiscent of those discussed in earlier SIR models with similar incidence rate and varying total population (see [3,5]). For instance, no periodic solutions exist. This is proved in this paper under the restriction that $\alpha \leqslant \epsilon$. Numerical simulations carried out for system (2.4) (see Fig. 3) seem to suggest that Theorem 6.5 holds without such a restriction. Our findings seem to concur with the earlier observation that the contact rate seems

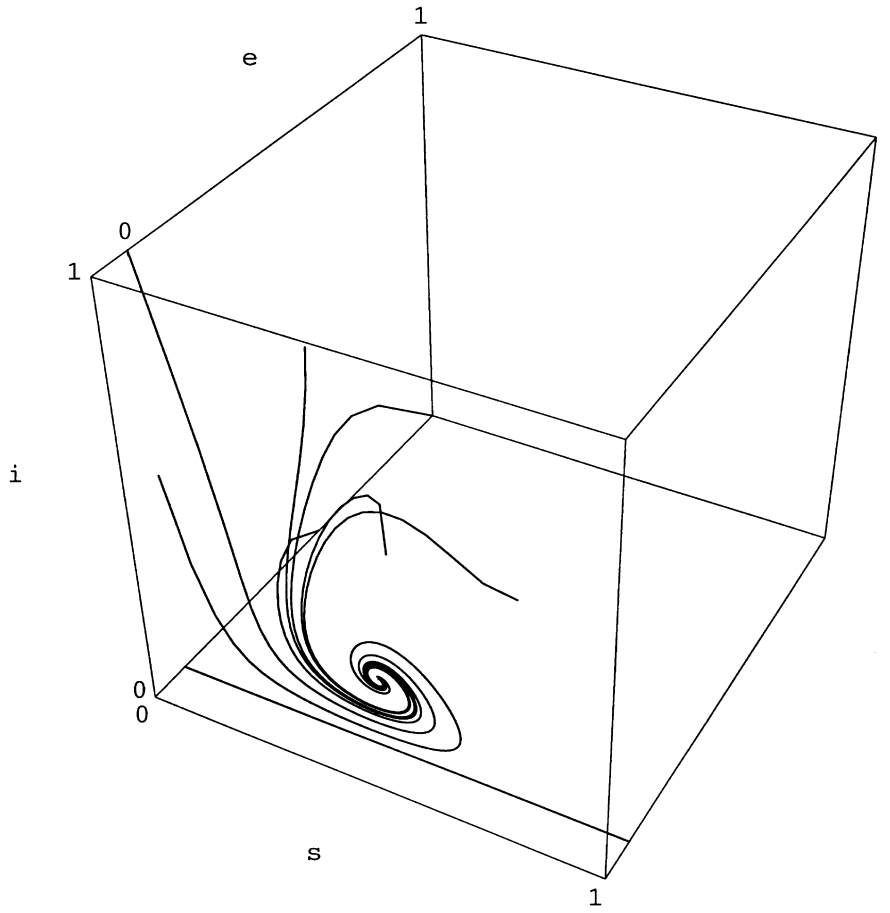


Fig. 3. A Mathematica 3D plot showing that the trajectories of (2.4) converge to the unique endemic equilibrium P^*. The parameter values are $\alpha = 6,\ b = 0.5,\ \epsilon = 4,\ \gamma = 1.5,\ \lambda = 20$. Note that $\alpha > \epsilon$ and $\sigma = 2.22 > 1$.

to be a more reliable source for more complicated dynamics such as periodic solutions to occur. For instance, periodic contact rates can lead to periodic solutions (see [27,28]) as can certain non-linear incidences (see [7,29]).

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Appendix A. Compound matrices

An $m \times m$ matrix A with real entries will be identified with the linear operator on $\mathbf{R}^m$ that it represents. Let "$\wedge$" denote the exterior product in $\mathbf{R}^m$. With respect to the canonical basis in the exterior product space $\wedge^2\mathbf{R}^m$, the *second additive compound matrix* $A^{[2]}$ of A represents a linear operator on $\wedge^2\mathbf{R}^m$ whose definition on a decomposable element $u_1 \wedge u_2$ is

$$A^{[2]}(u_1 \wedge u_2) = A(u_1) \wedge u_2 + u_1 \wedge A(u_2).$$

Definition over all of $\wedge^2\mathbf{R}^m$ is through linear extension. The entries in $A^{[2]}$ are linear relations of those in A. Let $A = (a_{ij})$. For any integer $i = 1, \ldots, \binom{m}{2}$, let $(i) = (i_1, i_2)$ be the ith member in the lexicographic ordering of integer pairs such that $1 \leqslant i_1 < i_2 \leqslant m$. Then, the entry in the ith row and the jth column of $Z = A^{[2]}$ is

$$z_{ij} = \begin{cases} a_{i_1 i_1} + a_{i_2 i_2} & \text{if } (i) = (j), \\ (-1)^{r+s} a_{i_s j_r} & \text{if exactly one entry } i_s \text{ of } (i) \text{ does not} \\ & \text{occur in } (j) \text{ and } j_r \text{ does not occur in } (i), \\ 0 & \text{if } (i) \text{ differs from } (j) \text{ in two or more entries.} \end{cases}$$

For any integer $1 \leqslant k \leqslant m$, the kth additive compound matrix $A^{[k]}$ of A is defined canonically. For discussions of compound matrices, the reader is referred to [12,30]. Pertinent to our purpose is a spectral property of $A^{[2]}$ given in the following proposition. Let $\sigma(A) = \{\lambda_i : i = 1, \ldots, m\}$ be the spectrum of A.

Proposition. *The spectrum of* $A^{[2]}$, $\sigma(A^{[2]}) = \{\lambda_{i_1} + \lambda_{i_2} : 1 \leqslant i_1 < i_2 \leqslant m\}$.

For $m = 2,3$ and 4, the second additive compound matrix $A^{[2]}$ of an $m \times m$ matrix $A = (a_{ij})$ is, respectively,

$m = 2$:
$$a_{11} + a_{22} \quad (= \mathrm{tr}(A)),$$

$m = 3$:
$$\begin{bmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{bmatrix},$$

$m = 4$:
$$\begin{bmatrix} a_{11} + a_{22} & a_{23} & a_{24} & -a_{13} & -a_{14} & 0 \\ a_{32} & a_{11} + a_{33} & a_{34} & a_{12} & 0 & -a_{14} \\ a_{42} & a_{43} & a_{11} + a_{44} & 0 & a_{12} & a_{13} \\ -a_{31} & a_{21} & 0 & a_{22} + a_{33} & a_{34} & -a_{24} \\ -a_{41} & 0 & a_{21} & a_{43} & a_{22} + a_{44} & a_{23} \\ 0 & -a_{41} & a_{31} & -a_{42} & a_{32} & a_{33} + a_{44} \end{bmatrix}.$$

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