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GLOBAL DYNAMICS OF AN SEIR EPIDEMIC MODEL WITH VERTICAL TRANSMISSION*

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Abstract. We study a population model for an infectious disease that spreads in the host population through both horizontal and vertical transmission. The total host population is assumed to have constant density and the incidence term is of the bilinear mass-action form. We prove that the global dynamics are completely determined by the basic reproduction number $R_0(p, q)$, where p and q are fractions of infected newborns from the exposed and infectious classes, respectively. If $R_0(p, q) \leq 1$, the disease-free equilibrium is globally stable and the disease always dies out. If $R_0(p, q) > 1$, a unique endemic equilibrium exists and is globally stable in the interior of the feasible region, and the disease persists at an endemic equilibrium state if it initially exists. The contribution of the vertical transmission to the basic reproduction number is also analyzed.

Key words. epidemic models, vertical transmission, endemic equilibrium, latent period, global stability, compound matrices

AMS subject classifications. Primary, 92D30; Secondary, 34D20

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1. Introduction.

Many infectious diseases in nature transmit through both horizontal and vertical modes. These include such human diseases as rubella, herpes simplex, hepatitis B, Chagas' disease, and, most notorious, AIDS [2, 3]. For human and animal diseases, horizontal transmission typically occurs through direct or indirect physical contact with infectious hosts, or through disease vectors such as mosquitos, ticks, or other biting insects. Vertical transmission can be accomplished through transplacental transfer of disease agents. Among insects or plants, vertical transmission is often through eggs or seeds. Busenberg and Cooke [3] discussed a variety of diseases that transmit both vertically and horizontally, and gave a comprehensive survey of the formulation and the mathematical analysis of compartmental models that also incorporate vertical transmission. In a standard S-I-R compartmental model, the vertical transmission can be incorporated by assuming that a fraction q of the offspring from the infectious (I) class are infectious at birth, and hence a birth flux, qbI, enters the I class, and the remaining birth, $b - qbI$, enters the susceptible (S) class. Here b is the natural birth rate of the host population, which is assumed to have a constant density 1. SIR models assume the disease has no latent period, so that infected hosts instantaneously become infectious. For many human diseases such as hepatitis B, Chagas' disease, and AIDS, the infected hosts stay in a latent period before becoming infectious [1, 10]. The latent hosts form an additional exposed (E) class. To incorporate vertical transmission in an SEIR model, it is plausible to assume

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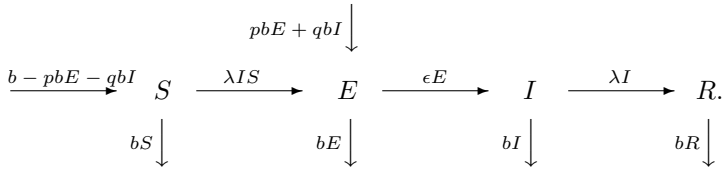
that a fraction of the offsprings of infected hosts (both E and I) are infected at birth and, like adult infected hosts, will stay latent before becoming infectious, and hence the infected birth flux will enter the E class. In this paper, we study an SEIR model in which vertical transmission is incorporated based on the above assumption. The transfer diagram is depicted in the following figure.



The total host population is partitioned into susceptibles, exposed (in the latent period), infectious, and recovered, with the densities respectively denoted by $S(t)$, $E(t)$, $I(t)$, and $R(t)$. The natural birth rate and death rate are assumed to be identical and denoted by b. The disease is assumed not to inflict death on the infected hosts so that the total population density is constant; $S(t) + E(t) + I(t) + R(t) = 1$. The horizontal transmission is assumed to take the form of direct contact between infectious and susceptible hosts. The incidence term λIS is of the bilinear mass-action form. For the vertical transmission, we assume that a fraction p and a fraction q of the offspring from the exposed and the infectious classes, respectively, are born into the exposed class E. Consequently, the birth flux into the exposed class is given by $pbE + qbI$ and the birth flux into the susceptible class is given by $b - pbE - qbI$. Naturally, $0 \le p \le 1$ and $0 \le q \le 1$. Rubella is among the diseases for which our model is a good approximation [2]. There is no recovery in Chagas' disease; this can be approximated in our model by taking the limit $\gamma \to 0$. Herpes simplex tends to relapse after recovery; one needs to modify the permanent immunity assumption in our model and consider an SEIRS model by allowing recovered hosts to return to the S class. In the case of AIDS, one needs to modify the constant population assumption and incorporate disease-related death to the infectives.

The transfer diagram leads to the following system of differential equations:

$$\begin{aligned} S' &= b - \lambda IS - pbE - qbI - bS, \\ E' &= \lambda IS + pbE + qbI - (\epsilon + b)E, \\ I' &= \epsilon E - (\gamma + b)I, \\ R' &= \gamma I - bR. \end{aligned} \tag{1.1}$$

We study (1.1) in the following feasible region:

$$\Sigma = \{\, (S, E, I, R) \in \mathbf{R}^4_+ \ : \ S + E + I + R = 1 \,\}.$$

It can be verified that Σ is positively invariant for (1.1). The parameter $\epsilon > 0$ is the rate at which the exposed individuals become infectious, and $\gamma \ge 0$ is the rate at which the infectious individuals recover. Therefore $1/\epsilon$ is the mean latent period and $1/\gamma$ is the mean infectious period. In the limiting case when $\epsilon \to \infty$, the latent period is negligible, and the model (1.1) reduces to an SIR model with bilinear incidence and no vertical transmission [3]. When $\gamma = 0$, there is no recovery from the disease, and (1.1) reduces to an SEI model. When $p = q = 0$, (1.1) becomes a classical SEIR model with no vertical transmission, and the vertical transmission is also lost if the birth rate $b = 0$, in which case (1.1) is an SEIR model with no birth and death. See [9, 10, 11, 19] for surveys of SEIR models and their limiting cases.

We derive a basic reproduction number $R_0(p,q)$ and prove that it completely determines the global dynamics of (1.1); if $R_0(p,q) \le 1$, the disease-free equilibrium $(1,0,0,0)$ is globally asymptotically stable and the disease always dies out, whereas if $R_0(p,q) > 1$, a unique endemic equilibrium P^* is globally asymptotically stable in the interior of the feasible region so that the disease persists at the endemic equilibrium level if it is initially present. When $p = q = 0$, vertical transmission is not present in the model; $R_0(p,q)$ reduces to the basic reproduction number R_0 for an SEIR model with no vertical transmission [1]; this threshold parameter is also denoted by σ and called the contact number in the literature [10, 19]. When $p, q > 0$, we give, in section 5, a detailed analysis of $R_0(p,q)$ and identify the relation between $R_0(p,q)$ and R_0, as well as the contribution from the vertical transmission.

A nontrivial part of the model analysis is to establish the global stability of the unique endemic equilibrium P^*. For earlier SIR and SIRS models, the global stability is proved by reducing the model to a two-dimensional system and applying the classical Poincaré–Bendixson theorem. Periodic orbits are ruled out using the Dulac criteria or a condition of Busenberg and van den Driessche [4]. The addition of the exposed class in an SEIR model introduces an additional equation to the SIR model, and hence makes the global stability analysis highly nontrivial. Li and Muldowney [18] and Li et al. [15] studied SEIR models with no vertical transmission. The models in [15, 18] can be reduced to a three-dimensional competitive system. The global stability of a unique P^* was proved using a Poincaré–Bendixson theorem for competitive systems in $\mathbf{R}^3$ established in Hirsch [12] and Smith [24]. The nonexistence of periodic solutions for these models was proved using a stability condition of Muldowney [21] for periodic solutions in higher dimensions. Our system (1.1) does not appear to reduce to any system that is known to possess a Poincaré–Bendixson property, so the method used in [15] and [18] does not apply.

In our main result, Theorem 2.3, we prove that the endemic equilibrium P^* is unique and globally stable whenever it exists. Our proof utilizes a general approach to the global stability problem developed in Li and Muldowney [17] and Smith [25]. The key step in the proof of Theorem 2.3 is the construction of a suitable Lyapunov function for the second compound (linear) system (3.2). The Lyapunov function used in section 4 of [17] does not work for our model (1.1), and a different Lyapunov function is constructed. We give a brief outline of the mathematical framework in section 3. The proof of Theorem 2.3 is given in section 4. In section 5, we give a detailed discussion of the basic reproduction number $R_0(p,q)$.

2. Statement of results. Using the relation $R(t) = 1 - S(t) - E(t) - I(t)$ we may reduce (1.1) to the following equivalent system:

$$\begin{aligned} S' &= b - \lambda IS - pbE - qbI - bS, \\ E' &= \lambda IS + pbE + qbI - (\epsilon + b)E, \\ I' &= \epsilon E - (\gamma + b)I \end{aligned} \tag{2.1}$$

on the closed, positively invariant set

$$\Gamma = \{\, (S,E,I) \in \mathbf{R}^3_+ \ : \ S + E + I \le 1 \,\}. \tag{2.2}$$

Denote the interior of Γ by $\overset{\circ}{\Gamma}$.

System (2.1) has two possible equilibria in Γ: the disease-free equilibrium $P_0 = (1,0,0)$ and an endemic equilibrium $P^* = (S^*, E^*, I^*) \in \overset{\circ}{\Gamma}$ with $S^* = 1/R_0(p,q)$,

where

$$R_0(p,q) = \frac{\lambda\epsilon}{(b+\epsilon)(b+\gamma) - bp(b+\gamma) - bq\epsilon}. \tag{2.3}$$

It is easy to verify that $R_0(p,q) > 0$ for $0 \le p, q \le 1$. Note that P^* exists in Γ and is unique if and only if $R_0(p,q) > 1$.

THEOREM 2.1. (a) *If $R_0(p,q) \le 1$, then P_0 is the only equilibrium and it is globally stable in Γ.* (b) *If $R_0(p,q) > 1$, then P_0 is unstable and there exists a unique endemic equilibrium P^*. Furthermore, all solutions starting in Γ and sufficiently close to P_0 move away from P_0 if $R_0(p,q) > 1$.*

Proof. Set

$$L = \epsilon E + (\epsilon + b - pb)I.$$

Then

$$\begin{aligned} L' &= \lambda\epsilon IS - [(b+\gamma)(b+\epsilon-pb) - qb\epsilon]\, I \\ &= (b+\gamma)\,[(b+\epsilon-pb) - qb\epsilon]\,[R_0(p,q)\,S - 1]\, I \\ &\le 0 \qquad \text{if} \qquad R_0(p,q) \le 1. \end{aligned}$$

Furthermore, $L' = 0$ if and only if $I = 0$ or $R_0(p,q) = 1$ and $S = 1$. Therefore the largest compact invariant set in $\{(S,E,I) \in \Gamma \ : \ L' = 0\}$ is the singleton $\{P_0\}$. LaSalle's invariance principle [14] then implies that P_0 is globally stable in Γ. This proves claim (a). Claim (b) follows from the fact that $L' > 0$ if $I > 0$ and $S > 1/R_0(p,q)$. □

If $R_0(p,q) > 1$, then the disease-free equilibrium is unstable by Theorem 2.1. Moreover, the behavior of the local dynamics near P_0 as described in Theorem 2.1 implies that system (2.1) is uniformly persistent in Γ; namely, there exists constant $c > 0$ such that

$$\begin{gathered} \liminf_{t\to\infty} S(t) > c, \quad \liminf_{t\to\infty} E(t) > c, \quad \liminf_{t\to\infty} I(t) > c, \\ \text{and} \qquad \liminf_{t\to\infty} [\,1 - S(t) - E(t) - I(t)\,] > c, \end{gathered} \tag{2.4}$$

provided $(S(0), E(0), I(0)) \in \Gamma$ [5, 8, 26]. Here, c is independent of initial data in Γ. This can be proved by applying a uniform persistence result in [8] and using a similar argument as in the proof of Proposition 3.3 of [15].

PROPOSITION 2.2. *If $R_0(p,q) > 1$, the system (2.1) is uniformly persistent.*

The uniform persistence of (2.1) in the bounded set Γ is equivalent to the existence of a compact $K \subset \overset{\circ}{\Gamma}$ that is absorbing for (2.1), namely, each compact set $K_0 \subset \overset{\circ}{\Gamma}$ satisfies $x(t, K_0) \subset K$ for sufficiently large t, where $x(t, x_0)$ denotes the solution of (2.1) such that $x(0, x_0) = x_0$ [5, 26]. We state our main result in the following theorem. Its proof will be given in section 4.

THEOREM 2.3. *Assume that $R_0(p,q) > 1$. Then the unique endemic equilibrium P^* is globally stable in $\overset{\circ}{\Gamma}$.*

Theorems 2.1 and 2.3 completely determine the global dynamics of system (2.1) and hence of our original model (1.1). They establish $R_0(p,q)$ as a sharp threshold parameter. If $R_0(p,q) \le 1$ the disease dies out, whereas when $R_0(p,q) > 1$ the disease persists at an endemic equilibrium level if it initially exists. The parameter

$R_0(p,q)$ can be interpreted as the basic reproduction number since, at the endemic equilibrium, $R_0(p,q)S^* = 1$ [1, p. 17]. If $p = q = 0$, then $R_0(0,0)$ gives the basic reproduction number R_0 [1] or the contact number σ [10, 19] for the SEIR or SEIRS models with only horizontal transmission; see [9] for a recent survey on these models. In the limiting case when $\epsilon \to \infty$, $R_0(p,q)$ gives a threshold parameter R_0 in [3, p. 63] for an SIR model.

Theorem 2.3 contains a global stability result in [18] for the bilinear incidence, where no vertical transmission is assumed ($p = q = 0$). The proof in [18] utilizes the property that the model in [18] can be reduced to a three-dimensional competitive system and thus satisfies a Poincaré–Bendixson theorem [12] and [24]. System (2.1) does not appear to reduce to such a system when $p, q > 0$. Since our model contains SIR models as special cases ($\epsilon \to \infty$), Theorem 2.3 generalizes some earlier results on SIR models with vertical transmission and a constant population; see [3] and the references therein.

3. A geometric approach to global-stability problems. In this section we briefly outline a general mathematical framework for proving global stability, which will be used in section 4 to prove Theorem 2.3. The framework is developed in the papers of Smith [25] and Li and Muldowney [16, 17]. The presentation here follows that in [17].

Let $x \mapsto f(x) \in \mathbf{R}^n$ be a C^1 function for x in an open set $D \subset \mathbf{R}^n$. Consider the differential equation

$$x' = f(x). \tag{3.1}$$

Denote by $x(t, x_0)$ the solution to (3.1) such that $x(0, x_0) = x_0$. We make the following two assumptions:

(H_1) There exists a compact absorbing set $K \subset D$.

(H_2) Equation (3.1) has a unique equilibrium $\overline{x}$ in D.

The equilibrium $\overline{x}$ is said to be *globally stable* in D if it is locally stable and all trajectories in D converge to $\overline{x}$. The following global-stability problem is formulated in [17].

Global-stability problem. Under the assumptions (H_1) and (H_2), find conditions on the vector field f such that the local stability of $\overline{x}$ implies its global stability in D.

The assumptions (H_1) and (H_2) are satisfied if $\overline{x}$ is globally stable in D. For $n \geq 2$, a *Bendixson criterion* is a condition satisfied by f which precludes the existence of nonconstant periodic solutions of (3.1). A Bendixson criterion is said to be *robust under C^1 local perturbations of f at $x_1 \in D$* if, for sufficiently small $\epsilon > 0$ and neighborhood U of x_1, it is also satisfied by $g \in C^1(D \to \mathbf{R}^n)$ such that the support $(f - g) \subset U$ and $|f - g|_{C^1} < \epsilon$, where

$$|f - g|_{C^1} = \sup \left\{ |f(x) - g(x)| + \left| \frac{\partial f}{\partial x}(x) - \frac{\partial g}{\partial x}(x) \right| : \ x \in D \right\}.$$

Such g will be called *local ϵ-perturbations of f at x_1*. It is easy to see that the classical Bendixson's condition $f(x) < 0$ for $n = 2$ is robust under C^1 local perturbations of f at each $x_1 \in \mathbf{R}^2$. Bendixson criterion for higher dimensional systems that are C^1 robust are discussed in [16, 17, 25].

A point $x_0 \in D$ is *wandering* for (3.1) if there exists a neighborhood U of x_0 and $T > 0$ such that $U \cap x(t, U)$ is empty for all $t > T$. Thus, for example, all equilibria and limit points are nonwandering. The following is a version of the local C^1 closing lemma of Pugh [22, 23] as stated in [13].

LEMMA 3.1. *Let $f \in C^1(D \to \mathbf{R}^n)$. Suppose that x_0 is a nonwandering point of* (3.1) *and that $f(x_0) \neq 0$. Also assume that the positive semiorbit of x_0 has compact closure. Then, for each neighborhood U of x_0 and $\epsilon > 0$, there exists a C^1 local ϵ-perturbation g of f at x_0 such that*

(1) $\operatorname{supp}(f - g) \subset U$, *and*

(2) *the perturbed system $x' = g(x)$ has a nonconstant periodic solution whose trajectory passes through x_0.*

The following general global-stability principle is established in [17].

THEOREM 3.2. *Suppose that assumptions* (H_1) *and* (H_2) *hold. Assume that* (3.1) *satisfies a Bendixson criterion that is robust under C^1 local perturbations of f at all nonequilibrium nonwandering points for* (3.1). *Then $\overline{x}$ is globally stable in D provided it is stable.*

The main idea of the proof in [17] for Theorem 3.2 is as follows: Suppose that system (3.1) satisfies a Bendixson criterion. Then it does not have any nonconstant periodic solutions. Moreover, the robustness assumption on the Bendixson criterion implies that all nearby differential equations have no nonconstant periodic solutions. Thus by Lemma 3.1, all nonwandering points of (3.1) in D must be equilibria. In particular, each omega limit point in D must be an equilibrium. Therefore $\omega(x_0) = \{\overline{x}\}$ for all $x_0 \in D$ since $\overline{x}$ is the only equilibrium in D.

A method of deriving a Bendixson criterion in $\mathbf{R}^n$ is developed in [16]. The idea is to show that the second compound equation

$$z'(t) = \frac{\partial f}{\partial x}^{[2]}(x(t, x_0))\, z(t), \tag{3.2}$$

with respect to a solution $x(t, x_0) \subset D$ to (3.1), is uniformly asymptotically stable, and the exponential decay rate of all solutions to (3.2) is uniform for x_0 in each compact subset of D. Here $\frac{\partial f}{\partial x}^{[2]}$ is the second additive compound matrix of the Jacobian matrix $\frac{\partial f}{\partial x}$; see the appendix. It is an $\binom{n}{2} \times \binom{n}{2}$ matrix, and thus (3.2) is a linear system of dimension $\binom{n}{2}$. If D is simply connected, the above-mentioned stability property of (3.2) implies the exponential decay of the surface area of any compact two-dimensional surface in D, which in turn precludes the existence of any invariant simple closed rectifiable curve in D, including periodic orbits. The required uniform asymptotic stability of the linear system (3.2) can be proved by constructing a suitable Lyapunov function.

Let $x \mapsto P(x)$ be an $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function that is C^1 for $x \in D$. Assume that $P^{-1}(x)$ exists and is continuous for $x \in K$, the compact absorbing set. Set

$$B = P_f P^{-1} + P \frac{\partial f}{\partial x}^{[2]} P^{-1}, \tag{3.3}$$

where the matrix P_f is obtained by replacing each entry p_{ij} of P by its derivative in the direction of f, p_{ij_f}. Let $|z|$ be a suitable vector norm for $z \in \mathbf{R}^N$, $N = \binom{n}{2}$, and let $\mu(B)$ be the *Lozinskiĭ measure* of B with respect to the induced matrix norm $|\cdot|$ in $\mathbf{R}^N$, defined by

$$\mu(B) = \lim_{h \to 0^+} \frac{|I + hB| - 1}{h};$$

see [6, p. 50]. Define a quantity $\overline{q}_2$ as

$$\overline{q}_2 = \limsup_{t\to\infty} \sup_{x_0\in K} \frac{1}{t}\int_0^t \mu(B(x(s,x_0)))\,ds. \tag{3.4}$$

It is shown in [17] that if D is simply connected and $\overline{q}_2 < 0$, the function $|P(x)z|$ is a Lyapunov function for (3.2), and hence (3.1) has no orbit that gives rise to an invariant simple closed rectifiable curve, such as periodic orbits, homoclinic orbits, and heteroclinic cycles. Hence $\overline{q}_2 < 0$ is a Bendixson criterion for (3.1) in D. Moreover, it is robust under C^1 local perturbations of f near any nonequilibrium point that is nonwandering. In particular, the following global-stability result is proved in Theorem 3.5 of [17].

THEOREM 3.3. *Assume that D is simply connected and that the assumptions* (H_1), (H_2) *hold. Then the unique equilibrium $\overline{x}$ of* (3.1) *is globally stable in D if* $\overline{q}_2 < 0$.

We remark that, under the assumptions of Theorem 3.3, the condition $\overline{q}_2 < 0$ also implies the local stability of $\overline{x}$, since, assuming the contrary, $\overline{x}$ is both the alpha and the omega limit point of a homoclinic orbit that is ruled out by the condition $\overline{q}_2 < 0$.

4. Proof of Theorem 2.3. We now apply the theory outlined in the preceding section, in particular Theorem 3.3, to prove Theorem 2.3. From the discussion in section 2, we know that system (2.1) satisfies the assumptions (H_1), (H_2). Let $x = (S, E, I)$ and $f(x)$ denote the vector field of (2.1). The Jacobian matrix $J = \frac{\partial f}{\partial x}$ associated with a general solution $x(t)$ of (2.1) is

$$J = \begin{bmatrix} -\lambda I - b & -pb & -\lambda S - qb \\ \lambda I & pb - b - \epsilon & \lambda S + qb \\ 0 & \epsilon & -b - \gamma \end{bmatrix},$$

and its second additive compound matrix $J^{[2]}$ is, from the appendix,

$$J^{[2]} = \begin{bmatrix} -\lambda I - \epsilon - 2b + pb & \lambda S + qb & \lambda S + qb \\ \epsilon & -\lambda I - \gamma - 2b & -pb \\ 0 & \lambda I & -\epsilon - \gamma - 2b + pb \end{bmatrix}. \tag{4.1}$$

Set the function $P(x) = P(S, E, I)$ as

$$P(S,E,I) = \begin{bmatrix} a_1 & 0 & 0 \\ 0 & (1-a_2)\dfrac{E}{I} & 0 \\ 0 & a_2\dfrac{E}{I} & \dfrac{E}{I} \end{bmatrix}, \tag{4.2}$$

where

$$a_2 = \begin{cases} 0 & \text{if} \quad \epsilon \geq pb, \\ 1 - \dfrac{\epsilon}{pb} & \text{if} \quad \epsilon < pb, \end{cases} \tag{4.3}$$

and $1 < a_1 < 1 + \lambda c^2/(\lambda + b)$. Here c is the uniform persistence constant in (2.4). Then

$$P_f P^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \dfrac{E'}{E} - \dfrac{I'}{I} & 0 \\ 0 & 0 & \dfrac{E'}{E} - \dfrac{I'}{I} \end{bmatrix}$$

and the matrix $B = P_f P^{-1} + P J^{[2]} P^{-1}$ in (3.3) can be written in block form:

$$B = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix},$$

where $B_{11} = -\lambda I - \epsilon - 2b + pb$,

$$B_{12} = a_1 \left[(\lambda S + qb)\frac{I}{E}, \quad (\lambda S + qb)\frac{I}{E} \right], \qquad B_{21} = \frac{1}{a_1} \begin{bmatrix} (1 - a_2)\dfrac{\epsilon E}{I} \\ a_2 \dfrac{\epsilon E}{I}, \end{bmatrix},$$

$$B_{22} = \begin{bmatrix} \dfrac{E'}{E} - \dfrac{I'}{I} - \lambda I - \gamma - 2b + a_2 pb & -(1 - a_2)pb \\ \lambda I + \dfrac{a_2\,[\epsilon - (1 - a_2)pb]}{1 - a_2} & \dfrac{E'}{E} - \dfrac{I'}{I} - \epsilon - \gamma - 2b + (1 - a_2)pb \end{bmatrix}$$

$$= \begin{bmatrix} \dfrac{E'}{E} - \dfrac{I'}{I} - \lambda I - \gamma - 2b + a_2 pb & -(1 - a_2)pb \\ \lambda I & \dfrac{E'}{E} - \dfrac{I'}{I} - \epsilon - \gamma - 2b + (1 - a_2)pb \end{bmatrix},$$

since $a_2[\epsilon - (1 - a_2)pb] = 0$ by (4.3). Let $z = (u, v, w)$ denote the vectors in $\mathbf{R}^3 \cong \mathbf{R}^{\binom{3}{2}}$; we select a norm in $\mathbf{R}^3$ as

$$|(u, v, w)| = \max\{|u|,\ |v| + |w|\}$$

and let μ denote the Lozinskiĭ measure with respect to this norm. As described in [20], we have the estimate

$$\mu(B) \le \sup\{g_1,\ g_2\}, \tag{4.4}$$

where

$$g_1 = \mu_1(B_{11}) + |B_{12}|,$$
$$g_2 = |B_{21}| + \mu_1(B_{22}),$$

$|B_{12}|,\ |B_{21}|$ are matrix norms with respect to the l_1 vector norm, and μ_1 denotes the Lozinskiĭ measure with respect to the l_1 norm; also see [6, 16, 17]. More specifically, $\mu_1(B_{11}) = -\lambda I - \epsilon - 2b + pb$, $|B_{12}| = a_1(\lambda S + qb)I/E$, $|B_{21}| = \epsilon E/(a_1 I)$. To calculate $\mu_1(B_{22})$, add the absolute value of the off-diagonal elements to the diagonal one in each column of B_{22} and then take the maximum of two sums; see [6, p. 50]. We thus obtain

$$\mu_1(B_{22}) = \frac{E'}{E} - \frac{I'}{I} - \gamma - 2b + \max\{\, a_2 pb, \quad (1 - a_2)pb - \epsilon + (1 - a_2)pb\}$$
$$\le \frac{E'}{E} - \frac{I'}{I} - \gamma - 2b + pb,$$

since $0 \le a_2 < 1$ and $(1-a_2)pb - \epsilon \le 0$ from (4.3). Therefore

$$g_1 = -\lambda I - \epsilon - 2b + pb + a_1(\lambda S + qb)\frac{I}{E}, \tag{4.5}$$

$$g_2 \le \frac{E'}{E} - \frac{I'}{I} - \gamma - 2b + pb + \frac{1}{a_1}\frac{\epsilon E}{I}. \tag{4.6}$$

Rewriting (2.1), we have

$$\frac{E'}{E} + b + \epsilon - pb = (\lambda S + qb)\frac{I}{E}, \tag{4.7}$$

$$\frac{I'}{I} + b + \gamma = \frac{\epsilon E}{I}. \tag{4.8}$$

The uniform persistence constant c in (2.4) can be adjusted so that there exists $T > 0$ independent of $x(0) \in K$, the compact absorbing set, such that

$$I(t) \ge c \qquad \text{and} \qquad E(t) \ge c \qquad \text{for} \quad t > T. \tag{4.9}$$

Substituting (4.7) into (4.5) and (4.8) into (4.6) and using (4.9) and our choice of a_1, we obtain, for $t > T$,

$$\begin{aligned} g_1 &= -\lambda I - b + \frac{E'}{E} + (a_1 - 1)(\lambda S + qb)\frac{I}{E} \\ &\le \frac{E'}{E} - \lambda c - b + (a_1 - 1)\frac{\lambda + b}{c} \le \frac{E'}{E} - b \end{aligned} \tag{4.10}$$

and

$$\begin{aligned} g_2 &\le \frac{E'}{E} - b + pb + \left(\frac{1}{a_1} - 1\right)\frac{\epsilon E}{I} \\ &\le \frac{E'}{E} - b + pb + \frac{(1-a_1)\epsilon c}{a_1} \le \frac{E'}{E} - \frac{(a_1 - 1)\epsilon c}{a_1}, \end{aligned} \tag{4.11}$$

since $0 \le p \le 1$. Therefore $\mu(B) \le E'/E - \bar{b}$ for $t > T$ by (4.4), (4.10), and (4.11), where $\bar{b} = \min\{b,\ (a_1 - 1)\epsilon c/a_1\} > 0$. Along each solution $x(t, x_0)$ to (2.1) such that $x_0 \in K$ and for $t > T$, we thus have

$$\frac{1}{t}\int_0^t \mu(B)\,ds \le \frac{1}{t}\int_0^T \mu(B)\,ds + \frac{1}{t}\log\frac{E(t)}{E(T)} - \bar{b}\,\frac{t-T}{t},$$

which implies $\bar{q}_2 \le -\bar{b}/2 < 0$ from (3.4), proving Theorem 2.3.

Remark. In [17], as an example, the same approach as in the preceding proof was used to prove the global stability of a unique endemic equilibrium of an SEIRS model without vertical transmission. Our choice of the matrix function $P(x)$, and hence the Lyapunov function for the second compound system (4.2), differs from that in [17] in an essential way; when $p = q = 0$, our $P(x)$ does not reduce to the one in [17], and the construction in [17] does not extend trivially to our model.

5. The basic reproductive number. To get a better understanding of the basic reproduction number $R_0(p,q)$ in (2.3), we rewrite it using Taylor expansion as

$$R_0(p,q) = R_0\,(1 + R + R^2 + \cdots), \tag{5.1}$$

where

$$R_0 = \frac{\lambda\epsilon}{(b+\gamma)(b+\epsilon)} \tag{5.2}$$

and

$$R = R_p + R_q = \frac{pb}{(b+\epsilon)} + \frac{qb\epsilon}{(b+\epsilon)(b+\gamma)}.$$

The R_0 in (5.2) is the basic reproduction number for the horizontal transmission; it represents the number of secondary infections contributed through horizontal infection by a single infectious host during the infective period in an entirely susceptible population [1]. Rewrite $R_0\,R_p$ as

$$R_0 \cdot R_p = \lambda \cdot \frac{1}{b+\gamma} \cdot \frac{bp}{b+\epsilon} \cdot \frac{\epsilon}{b+\epsilon}.$$

This expression can be deciphered as follows. Over the mean infectious period $\frac{1}{b+\gamma}$, a single infective produces $\lambda \cdot \frac{1}{b+\gamma}$ latent hosts through direct contact. Each of these new latent hosts gives $\frac{bp}{b+\epsilon}$ latent offspring during the mean latent period, a fraction $\frac{\epsilon}{b+\epsilon}$ of which survives latency and becomes infectious. Similarly, rewrite $R_0 R_q$ as

$$R_0 \cdot R_q = \lambda \cdot \frac{1}{b+\gamma} \cdot \frac{\epsilon}{(b+\epsilon)} \cdot \frac{bq}{b+\gamma} \cdot \frac{\epsilon}{(b+\epsilon)},$$

which can be interpreted as follows. Over the mean infectious period, a single infective produces $\lambda\frac{1}{b+\gamma}$ latent hosts through direct contact, a fraction $\frac{\epsilon}{b+\epsilon}$ of which survives latency and becomes infectious. Each of these new infectious hosts gives $\frac{bq}{b+\gamma}$ latent offspring during the mean infectious period, a fraction $\frac{\epsilon}{b+\epsilon}$ of which survives latency and becomes infectious. Therefore, $R_0\,R = R_0\,(R_p + R_q)$ represents the total contribution to the infective class made by the first generation offspring of our original infective. Similarly, $R_0\,R^2$ and the higher order terms in (5.1) represent the contribution through vertical transmission in the second generation and so on. If $p = q = 0$, no secondary infections come from vertical transmission, $R_0(p,q)$ simply gives the standard basic reproduction number R_0 for the horizontal transmission.

6. Summary. In this paper, we have studied an SEIR model for the dynamics of an infectious disease. The infection can be through a direct horizontal transmission and a vertical transmission where a fraction p of the newborns from the exposed class and a fraction q of the newborns from the infectious class are assumed to be infected at birth. In comparison to some SIR models that incorporate vertical transmission, we assume that the disease possesses a nonnegligible latent period and infected individuals will stay in the latent period before they become infectious. The natural birth and death rates are assumed to be equal and disease-related death is negligible so that the total population is a constant. The incidence term is of the bilinear mass-action form and the immunity is assumed to be permanent.

A threshold parameter $R_0(p,q)$ is identified which completely determines the global dynamics of our model. If $R_0(p,q) \leq 1$, the disease-free equilibrium is globally stable in the feasible region and the disease always dies out, whereas if $R_0(p,q) > 1$, a unique endemic equilibrium is globally stable in the interior of the feasible region, and the disease persists at a constant endemic level if it initially presents. Our result generalizes a global stability result in [18] in the case of no vertical transmission and some earlier global stability results on SIR models with vertical transmission. Our proof of the global stability of the endemic equilibrium when $R_0(p,q) > 1$ utilizes a general approach established in [17], and relies on the construction of a new Lyapunov function for the second compound (linear) system (3.2).

Appendix. The second additive compound matrix. Let A be a linear operator on $\mathbf{R}^n$ and also denote its matrix representation with respect to the standard basis of $\mathbf{R}^n$. Let $\wedge^2\mathbf{R}^n$ denote the exterior product of $\mathbf{R}^n$. A induces canonically a linear operator $A^{[2]}$ on $\wedge^2\mathbf{R}^n$: for $u_1, u_2 \in \mathbf{R}^n$, define

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

and extend the definition over $\wedge^2\mathbf{R}^n$ by linearity. The matrix representation of $A^{[2]}$ with respect to the canonical basis in $\wedge^2\mathbf{R}^n$ is called the *second additive compound matrix* of A. This is an $\binom{n}{2} \times \binom{n}{2}$ matrix and satisfies the property $(A+B)^{[2]} = A^{[2]} + B^{[2]}$. In the special case when $n = 2$, we have $A^{[2]}_{2\times 2} = \mathrm{tr}A$. In general, each entry of $A^{[2]}$ is a linear expression of those of A. For instance, when $n = 3$, the second additive compound matrix of $A = (a_{ij})$ is

$$A^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix}.$$

For detailed discussions of compound matrices and their properties we refer the reader to [7, 21]. A comprehensive survey on compound matrices and their relations to differential equations is given in [21].

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