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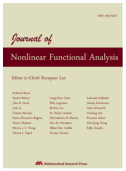

ANALYSIS OF AN SIR EPIDEMIC MODEL WITH SATURATION INCIDENCE RATE IN AN ENVIRONMENTALLY-DRIVEN INFECTIOUS DISEASE

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Abstract. In this paper, we construct and analyze a model for the linking of within- and between-host with saturation incidence rate. It is postulated that the infection process depends on the size of the infective inoculum that susceptible hosts may acquire by interacting with a contaminated environment. Since the dynamical processes associated with the within- and between-host occur on different time scales, the dynamical behaviors of the system can be analyzed by using a singular perturbation argument, which allows us to decouple the full system by separating the fast- and slow-systems. For the fast system, it is shown that the infection-free equilibrium U_0 is locally asymptotically stable if $R_{v0} < 1$, whereas U_0 is unstable if $R_{v0} > 1$, and a unique interior equilibrium $U^*(E)$ exists and it is locally asymptotically stable. For the slow system, under the condition that the fast system has a stable interior equilibrium $U^*(E)$ (i.e., $R_{v0} > 1$), there exists at least one endemic equilibrium W^* if $R_h > 1$. We also get the sufficient condition for its local stability of the endemic equilibrium W^*. At last, some simple discussion is presented at the end of the article.

Keywords. Epidemic model; Saturation incidence rate; Equilibrium; Basic reproduction number.

1. Introduction

There are two key processes in the host-parasite interaction. One is the epidemiological process associated with the disease transmission, and the other is the immunological process

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at the individual host level. For viral dynamic models, it is needed to consider the within-host dynamics independent of the interaction at the population level. For epidemiological models, it is a need to consider the interaction between susceptible and infected hosts without an explicit link to the viral dynamics within the hosts. There are some questions that can only be studied by using models that explicitly link the two processes, including : (i) How does the within-host dynamics influence the transmission of a pathogen from individual to individual? (ii) What is the effect of population dynamics of disease transmission on the viral dynamics at the individual level? (iii) Will the model predictions in terms of the virulence and basic reproduction number of the pathogen be altered if the two process are dynamically linked [7]? In [8], the authors used a nested model to evaluate the direction of natural selection at the within- and between-host levels.

In this paper, we will propose a framework that explicitly links the epidemiological and immunological dynamics with saturation incidence rate. This model is used to analyze the dynamics of parasite *Toxoplasma gondii* and their host, which is an obligate intracellular parasite that can infect all warm-blooded vertebrates, including mammals and birds. The parasite reproduces sexually in felines. For example, if a cat becomes infected, it will shed oocysts, which will then contaminate the environment. These oocysts can be ingested by mammals and birds which then become infected with the parasite. Some authors suggested that eating another organism that is infected may cause secondary infection [7, 13].

Compartment models have been used to analyze the dynamics of infectious diseases for a long time since the poinnering work of Kermack and McKendrick in 1927. And this method is still widely used, such as in [1-12]. In general, some kinds of diseases spreading by bacteria, such as encephalitis, gonorrhea, rehabilitation have no immunity. Thus, it can be infected again, and it is suitable to describe by SI model [11]. However, other diseases such as flu, measles, and chickenpox, rehabilitation of the original virus may bring forth immunity. That is to say, in a short period of time, the host won't be infected again, and it is suitable for SIR model to describe [3, 9, 10, 16, 17].

The organization of this paper is as follows: In section 2, a mathematical model for coupling within-host and between-host dynamics with saturation incidence rate is constructed. The existence and stability of equilibria of the fast system and slow system are investigated in sections 3 and 4, respectively. In the last section, we comment the results.

2. Formulation of the model

In [4], the authors proposed a novel approach to study the interplay between within- and between-host competition. Z. Feng et al, presented a new model that allows the two dynamic processes to explicitly depend on each other [6]; Z. Feng and Jorge proposed and discussed an *SI* mathematical model for coupling within-host and between-host dynamics with bilinear incidence rate in [7].

The between-host dynamics is governed by the *SI* system

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= \mu N - \lambda ES - \mu S, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= \lambda ES - \mu I. \end{cases} \tag{2.1}$$

The variables S and I represent the numbers of susceptible and infectious individuals at time t, respectively. The parameter μ is the per-capita host natural mortality rate, which is assumed to be the same as the per-capital birth rate, so that the total population size $N = S + I$ remains constant for all time t. The parameter λ is the per-capita infection rate of hosts in a contaminated environment, and E stands for the level of environment contamination at time t, or the concentration of oocysts per unit area or volume of a region being considered ($0 \leq E \leq 1$). This level of contamination depends on the number of infected hosts (I) and the average parasite load (V) within the host as described by the following equation

$$\frac{\mathrm{d}E}{\mathrm{d}t} = \theta IV(1-E) - \gamma E, \tag{2.2}$$

where θ is the rate of contamination and γ is the clearance rate.

For the within-host dynamics, the sub-system is

$$\begin{cases} \dfrac{\mathrm{d}T}{\mathrm{d}t} &= \Lambda - kVT - mT, \\ \dfrac{\mathrm{d}T^*}{\mathrm{d}t} &= kVT - (m+d)T^*, \\ \dfrac{\mathrm{d}V}{\mathrm{d}t} &= g(E) + pT^* - cV. \end{cases} \tag{2.3}$$

Here, the variables T, T^* and V represent the density of healthy cells, infected cells, and parasite load, respectively. The parameter k denotes the per-capita infection rate of cells; m and d are the per-capita natural mortality and infection-induced mortality rates of cell, respectively; p is the parasite reproduction rate by an infected cell; and c is the within-host mortality rate of parasites. The function $g(E)$ represents the rate at which an average host is inoculated.

In order to describe the reality more accurately, in this article, the research population (N) will be divided into three subclasses: susceptible (S), infectious(I) and recovered (R), respectively. Motivated by [6-10], we will construct the following mathematical model for an environmentally-driven infectious disease with saturation incidence rate.

The between-host dynamics is governed by the SIR system

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= \mu N - \frac{\lambda ES}{1+\beta S} - \mu S, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\lambda ES}{1+\beta S} - (\mu+\alpha)I, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I - \mu R, \\ \dfrac{\mathrm{d}E}{\mathrm{d}t} &= \theta IV(1-E) - \gamma E, \\ N &= S+I+R. \end{cases} \tag{2.4}$$

For the within-host dynamics, the subsystem will be described by

$$\begin{cases} \dfrac{\mathrm{d}H}{\mathrm{d}t} &= \Lambda - kVH - mH, \\ \dfrac{\mathrm{d}P}{\mathrm{d}t} &= kVH - (m+d)P - \beta P, \\ \dfrac{\mathrm{d}Q}{\mathrm{d}t} &= \beta P - mQ, \\ \dfrac{\mathrm{d}V}{\mathrm{d}t} &= g(E) + pP - cV. \end{cases} \tag{2.5}$$

Here, the variables H, P, Q and V represent the density of healthy cells, infected cells, recovered cells, and parasite load, respectively. The parameter β is the transversion rate from infected cell to recovered cell, and the other parameters are similar to those in [7].

The function $g(E)$ indicates the fact that if the environmental contamination is high, then the inoculum is high as well. For biological consideration, the function $g(E)$ should have the following properties:

$$g(E) \geq 0, \quad g(0) = 0, \quad g^{'}(E) > 0. \tag{2.6}$$

In [7], $g(E) = aE$, and a is a positive constant. Here we choose the function $g(E)$ with the following nonlinear type

$$g(E) = \frac{aE}{1+E^2}, \tag{2.7}$$

where a is a positive constant.

An important biological feature of this coupled system (2.4)-(2.5) is that the within-host dynamics occurs on a faster time scale than the dynamics of the between-host and the environment. The multiple time-scale allows us to study the mathematical properties of the model by analyzing the fast- and slow-systems determined by the two time scales.

3. Analysis of the fast system (2.5)

3.1 Existence of equilibriums

The subsystem (2.5) for the within-host dynamics can be considered as the fast system in which the variable E will be treated as a constant. For simple case of $g(E) = 0$, the system (2.5) always has an infection-free equilibrium $U_0 = (H_0, P_0, Q_0, V_0)$, where $H_0 = \frac{\Lambda}{m}$, $P_0 = 0$, $Q_0 = 0$, $V_0 = 0$. However, if $g(E) > 0$, the system (2.5) has no infection-free equilibrium.

Let $R_v(E)$ be the within-host reproduction number [15], which is a function of the environmental contamination level. Denote $R_{v^0} = R_v(0)$, then, it is easy to derive that

$$R_{v^0} = \frac{H_0 kp}{c(m+d+\beta)}.$$

Next, we will consider the case of $g(E) > 0$.

Let $U^*(E) = (H^*(E), P^*(E), Q^*(E), V^*(E))$ be a nontrivial equilibrium, then by simple calculation, we will get

$$\begin{aligned} P^*(E) &= \tfrac{\Lambda - mH^*}{m+d+\beta} = \tfrac{m}{m+d+\beta}(H_0 - H^*(E)) > 0, \\ V^*(E) &= \tfrac{1}{c}(g(E) + pH^*) > 0, \\ Q^*(E) &= \tfrac{\beta}{m} P^*(E) > 0, \end{aligned}$$

and $H^*(E)$ is a root of the following quadratic equation

$$H^2 - a_1 H + a_2 = 0, \tag{3.1}$$

where

$$\begin{aligned} a_1 &= \tfrac{m+d+\beta}{pm} g(E) + H_0(1 + \tfrac{1}{R_{v0}}) > 0, \\ a_2 &= \tfrac{1}{R_{v0}} H_0^2 > 0. \end{aligned}$$

The discriminant of equation (3.1) is given by

$$\begin{aligned} a_1^2 - 4a_2 &= [\tfrac{m+d+\beta}{pm} g(E) + H_0(1 + \tfrac{1}{R_{v0}})]^2 - 4\tfrac{H_0^2}{R_{v0}} \\ &\geq H_0^2(1 + \tfrac{1}{R_{v0}})^2 - 4\tfrac{H_0^2}{R_{v0}} \\ &= H_0^2(1 - \tfrac{1}{R_{v0}})^2 \geq 0. \end{aligned}$$

Thus, equation (3.1) has two positive real solutions given by $H_\pm^*(E) = \frac{a_1 \pm \sqrt{a_1^2 - 4a_2}}{2}$. Note that

$$\begin{aligned} a_1'(E) &= \tfrac{m+d+\beta}{pm} g'(E) > 0, \\ (H_\pm^*)'(E) &= \tfrac{1}{2} a_1'(E)(1 \pm \tfrac{a_1}{\sqrt{a_1^2 - 4a_2}}). \end{aligned}$$

Since $a_2 > 0$ and $a_1'(E) > 0$, we obtain that

$$(H_+^*)'(E) > 0, \quad (H_-^*)'(E) < 0. \tag{3.2}$$

In addition, we have

$$H_+^*(0) = \begin{cases} H_0 \; R_{v0} \geq 1, \\ \frac{H_0}{R_{v0}} \; R_{v0} < 1, \end{cases} \tag{3.3}$$

$$H_-^*(0) = \begin{cases} \frac{H_0}{R_{v0}} \; R_{v0} > 1, \\ H_0 \; R_{v0} \leq 1. \end{cases} \tag{3.4}$$

From $H_+^*(0) \geq H_0$ and $(H_+^*)'(E) > 0$, we know that $H_+^*(E) > H_0$ for all $E > 0$. But $H_+^*(E) \leq H_0$, and thus, $H_+^*(E)$ does not exist.

From equations (3.2) and (3.4) we know that $U^*(E) = (H_-^*(E), P^*(E), Q^*(E), V^*(E))$ exists when $R_{v^0} > 1$, and it coincides with U_0 when $R_{v^0} \leq 1$.

Motivated by the existence condition for $U^*(E)$, we will arrive at the definition of $R_v(E)$ as

$$R_v(E) = \frac{H_0}{H_-^*(E)} R_{v^0}. \tag{3.5}$$

Then, according to the above discussion, we can obtain the following result.

Theorem 3.1. *(i) If $g(E) = 0$, the fast system (2.5) always has an infection-free equilibrium $U_0 = (H_0, P_0, Q_0, V_0)$.*

(ii) If $g(E) > 0$, the fast system (2.5) has a unique nontrivial equilibrium $U^*(E)$ if $R_{v^0} > 1$.

3.2 Stability of the equilibriums

The Jacobian matrix at U_0 is given by

$$J(U_0) = \begin{bmatrix} -m & 0 & 0 & \frac{-k\Lambda}{m} \\ 0 & -(m+d+\beta) & 0 & \frac{k\Lambda}{m} \\ 0 & \beta & -m & 0 \\ 0 & p & 0 & -c \end{bmatrix}.$$

One of the eigenvalues of the above matrix is $X_1 = -m < 0$, and the other three eigenvalues are determined by the following equation

$$X^3 + b_1 X^2 + b_2 X + b_3 = 0, \tag{3.6}$$

where

$$\begin{aligned} b_1 &= 2m + d + \beta + c > 0, \\ b_2 &= mc + (m+c)(m+d+\beta) - p\tfrac{k\Lambda}{m}, \\ b_3 &= mc(m+d+\beta) - \tfrac{k\Lambda}{p}. \end{aligned}$$

It is easy to show that $b_2 > 0$, $b_3 > 0$, $b_1 b_2 - b_3 > 0$, if $R_{v^0} < 1$. Thus, by the Routh-Hurwitz criterion, all the roots of the equation (3.6) have negative real parts. Hence, U_0 is locally asymptotically stable. However, we will get $b_3 < 0$ if $R_{v^0} > 1$, then one of the roots of the equation (3.6) has positive real parts, which means that U_0 is unstable.

The Jacobian matrix at $U^*(E)$ is given by

$$J(U^*(E)) = \begin{bmatrix} -\frac{\Lambda}{H_-^*(E)} & 0 & 0 & -kH_-^*(E) \\ \frac{\Lambda}{H_-^*(E)} - m & -(m+d+\beta) & 0 & kH_-^*(E) \\ 0 & \beta & -m & 0 \\ 0 & p & 0 & -c \end{bmatrix},$$

and the corresponding characteristic equation is

$$X^4 + c_1X^3 + c_2X^2 + c_3X + c_4 = 0, \tag{3.7}$$

where

$$\begin{aligned} c_1 &= c + 2m + d + \beta + \tfrac{\Lambda}{H_-^*(E)}, \\ c_2 &= mc + (m+d+\beta)(c+m) + \tfrac{(c+2m+d+\beta)\Lambda}{H_-^*(E)}, \\ c_3 &= mc(m+d+\beta + \tfrac{\Lambda}{H_-^*(E)}) + \tfrac{(c+m)(m+d+\beta)\Lambda}{H_-^*(E)}, \\ c_4 &= mc(m+d+\beta)\tfrac{\Lambda}{H_-^*(E)} - pmk\Lambda. \end{aligned}$$

It is easy to show that $c_1 > 0$, $c_2 > 0$, $c_3 > 0$; and from $R_v(E) > R_{v^0}$, we will further get $c_4 = mkp\Lambda(\frac{R_v(E)}{R_{v^0}} - 1) > 0$.

Note that

$$\begin{aligned} |H_2| &= c_1c_2 - c_3 > 0, \\ |H_3| &= c_1c_2c_3 - c_3^2 - c_1^2c_4 = c_3(c_1c_2 - c_3) - c_1^2c_4 > 0, \\ |H_4| &= c_4[c_3(c_1c_2 - c_3) - c_1^2c_4] > 0. \end{aligned}$$

By the Routh-Hurwitz criterion, we will obtain the local stability of $U^*(E)$. Thus, we will arrive at the following result.

Theorem 3.2. *(i) U_0 is locally asymptotically stable if $R_{v^0} < 1$; and it is unstable if $R_{v^0} > 1$.*

(ii) For all $E > 0$, $U^*(E)$ is always locally asymptotically stable whenever it exists.

4. Analysis of the slow system (2.4)

4.1. Existence of the equilibrium point

System (2.4) is considered to be slow system. It is assumed that $R_v(E) > 1$ holds, so that the fast system (2.5)is at the stable nontrivial equilibrium $U^*(E) = (H^*(E), P^*(E), Q^*(E), V^*(E))$ with

$$\begin{aligned} H^*(E) &= \tfrac{H_0}{R_v(E)}, \\ P^*(E) &= \tfrac{\Lambda}{m+d+\beta}(1-\tfrac{1}{R_v(E)}), \\ Q^*(E) &= \tfrac{\beta\Lambda}{m(m+d+\beta)}(1-\tfrac{1}{R_v(E)}), \\ V^*(E) &= \tfrac{1}{c}[g(E)+\tfrac{p\Lambda}{m+d+\beta}(1-\tfrac{1}{R_v(E)})], \\ R_v(E) &= \tfrac{2H_0R_{v0}}{a_1-\sqrt{a_1^2-4a_2}}. \end{aligned}$$

Since the total number of the host population $N = S+I+R$ remains constant for all time, we only need to consider the following three-dimensional subsystem of system (2.4)

$$\begin{cases} \dfrac{\mathrm{d}I}{\mathrm{d}t} &= \lambda E(N-I-R)-\mu I-\alpha I, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I-\mu R, \\ \dfrac{\mathrm{d}E}{\mathrm{d}t} &= \theta I V^*(E)(1-E)-\gamma E. \end{cases} \tag{4.1}$$

Notice that the dynamic linkage for the between- and within-host systems is through the environmental contamination variable E. The dependence of E on the within-host dynamics is through the term θVI. We will consider two cases.

Case 1. The fast subsystems (2.5) and the slow subsystem (2.4) will be decoupled if V is a constant, in which case we can obtain an isolated reproduction number R_{h^0} for the between-host system (2.4). Without loss of generality, we assume that $V = 1$, then it is easy to obtain

$$R_{h^0} = \frac{\theta\lambda N}{\gamma(\mu+\alpha)}.$$

Denote $W_0 = (I_0, R_0, E_0)$ as an interior equilibrium of the slow system (4.1), where

$$I_0 = \frac{\mu}{\alpha}R_0 > 0, \quad E_0 = \frac{\theta I_0}{\theta I_0+\gamma} > 0, \quad R_0 = \frac{\alpha(\theta\lambda N-\gamma(\mu+\alpha))}{\theta(\mu+\alpha)(\mu+\lambda)} > 0.$$

Obviously, the interior equilibrium W_0 exists when $V = 1$ and $R_{h^0} > 1$.

Case 2. When the fast subsystems (2.5) and the slow subsystem (2.4) are coupled near the equilibrium $U^*(E)$, the fourth equation in system (2.4) will be replaced by the corresponding third equation in system (4.1).

Let $W^* = (I^*, R^*, E^*)$ be an interior equilibrium of the slow system (4.1), where

$$I^* = \frac{\lambda E^* N}{\lambda E^*(1+\frac{\alpha}{\mu})+\mu+\alpha} > 0, \quad R^* = \frac{\alpha}{\mu} I^* > 0,$$

and E^* satisfies the following equation

$$\frac{1-E^*}{c}\left[g(E^*)+\frac{p\Lambda}{m+d+\beta}(1-\frac{1}{R_v(E^*)})\right] = \frac{rE^*(1+\frac{\alpha}{\mu})}{\theta N}+\frac{1}{R_{h^0}}. \tag{4.2}$$

Let $F(E)$ and $G(E)$ be the functions of the left and right sides of the equation (4.2), i.e.,

$$F(E) = \frac{1-E}{c}\left[g(E)+\frac{p\Lambda}{m+d+\beta}(1-\frac{1}{R_v(E)})\right],$$

$$G(E) = \frac{rE(1+\frac{\alpha}{\mu})}{\theta N}+\frac{1}{R_{h^0}}.$$

Then, E^* is a solution of the equation $F(E) = G(E)$ within $(0,1)$. Simple calculation implies that

$$\begin{aligned}
G(0) &= \tfrac{1}{R_{h^0}} > 0,\\
G(1) &= \tfrac{\gamma}{\theta N}(1+\tfrac{\alpha}{\mu})+\tfrac{1}{R_{h^0}} > 0,\\
G'(E) &= \tfrac{\gamma}{\theta N}(1+\tfrac{\alpha}{\mu}) > 0,\\
F(0) &= \tfrac{pmT_0}{c(m+d+\beta)}(1-\tfrac{1}{R_{v^0}}) > 0,\\
F(1) &= 0.
\end{aligned}$$

It is easy to see that there exists at least one solution $E^* \in (0,1)$ if $F(0) > G(0)$, which is equivalent to

$$\frac{pmT_0}{c(m+d+\beta)}(1-\frac{1}{R_{v^0}})R_{h^0} > 1. \tag{4.3}$$

By equation (4.3), we can define the basic reproduction number of the slow system (4.1), when it is coupled with the fast system, as

$$R_h = \frac{pmT_0}{c(m+d+\beta)}(1-\frac{1}{R_{v^0}})R_{h^0}.$$

We can summarize the the above discussion as following result.

Theorem 4.1. *(i) When $V = 1$, system (4.1) has a unique nontrivial equilibrium W_0 if $R_{h^0} \geq 1$.*

(ii) If $V = V(E)$ and $R_{v^0} > 1$, then the system (4.1) has at least one endemic equilibrium $W^* = (I^*, R^*, E^*)$ provided that $R_h > 1$.

4.2. Stability of the equilibrium point

The Jacobian matrix of the slow system (4.1) at equilibrium $W^* = (I^*, R^*, E^*)$ is given by

$$J(W^*) = \begin{bmatrix} -\lambda E^* - \mu - \alpha & -\lambda E^* & \lambda(N - I^* - R^*) \\ \alpha & -\mu & 0 \\ \theta V^*(E^*)(1-E^*) & 0 & \theta I^*(V^*(E^*))'(1-E^*) - \theta I^* V^*(E^*) - \gamma \end{bmatrix}.$$

By use of the formula of $F(E)$ and $G(E)$, it will become

$$J(W^*) = \begin{bmatrix} -\mu F(E^*)R_{h^0} - \alpha & -\mu(F(E^*)R_{h^0} - 1) & \frac{\lambda N}{R_{h^0} G(E^*)} \\ \alpha & -\mu & 0 \\ \theta G(E^*) & 0 & -\frac{\gamma}{F(E^*)}(F(E^*) - E^* F'(E^*)) \end{bmatrix},$$

where

$$\begin{aligned} V^*(E) &= \tfrac{F(E)}{1-E}, \\ \theta I^* &= \tfrac{\gamma E^*}{F(E^*)}, \\ \lambda E^* &= \tfrac{\mu\lambda\theta N}{\gamma(\alpha+\mu)}[F(E^*) - \tfrac{1}{R_{h^0}}] = \mu[F(E^*)R_{h^0} - 1]. \end{aligned}$$

Then the corresponding characteristic equation of the above matrix will be

$$X^3 + d_1 X^2 + d_2 X + d_3 = 0, \tag{4.4}$$

where

$$\begin{aligned} d_1 &= \frac{\gamma}{F}(F - E^* F') + \mu + \alpha + \mu F R_{h^0}, \\ d_2 &= \gamma\mu(F - E^* F')(R_{h^0} + \tfrac{1}{F}) + \mu F R_{h^0}(\mu + \alpha) - \gamma(\mu + \alpha), \\ d_3 &= \mu\gamma(\mu + \alpha)[(F - E^* F')R_{h^0} - 1]. \end{aligned}$$

Note that $R_v(E) > R_{v^0} > 1$, $F(E) > 0$ and $F'(E^*) < 0$. Thus, we get $d_1 > 0$ and $d_1 d_2 - d_3 > 0$. If we further have $d_3 > 0$, then by the Routh-Hurwitz criterion, we will obtain the local stability of W^*.

Let $R^*_{h^0} = \frac{1}{F(E^*) - E^* F'(E^*)}$, then we get the following result.

Theorem 4.2. *The endemic equilibrium W^* of the system (4.1) is locally asymptotically stable if $R_{h^0} > R^*_{h^0}$.*

5. Conclusions

In this work, we develop and analyze a model that couples explicitly the between- and within-host systems with saturation incidence rate. This coupled system provided new insights into the effect of each process on the other.

Theorems 3.1, 3.2, 4.1 and 4.2 suggest that the existence and stability of equilibriums of the fast and slow systems are determined by four reproduction numbers: R_{v^0}, R_h, R_{h^0} and $R^*_{h^0}$, respectively. The results can be summarized as follows:

- ♠: For the fast system (2.5), the infection-free equilibrium U_0 is locally asymptotically stable if $R_{v^0} < 1$. If $R_{v^0} > 1$, U_0 is unstable and a unique interior equilibrium $U^*(E)$ exists and it is locally asymptotically stable.
- ♠: For the slow system (4.1), under the condition that the fast system has the stable interior equilibrium $U^*(E)$ (when $R_{v^0} > 1$), there exists one endemic equilibrium W^* if $R_h > 1$.
- ♠: The endemic equilibrium W^* of the slow system (4.1) is locally asymptotically stable if $R_{h^0} > R^*_{h^0}$.

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