Research Article

Threshold Dynamics and Competitive Exclusion in a Virus Infection Model with General Incidence Function and Density-Dependent Diffusion

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In this paper, we investigate single-strain and multistrain viral infection models with general incidence function and density-dependent diffusion subject to the homogeneous Neumann boundary conditions. For the single-strain viral infection model, by using the linearization method and constructing appropriate Lyapunov functionals, we obtain that the global threshold dynamics of the model is determined by the reproductive numbers for viral infection \( R_0 \). For the multistrain viral infection model, we have discussed the competitive exclusion problem. If the reproduction number \( R_i \) for strain \( i \) is maximal and larger than one, the steady state \( E_i \) corresponding to the strain \( i \) is globally stable. Thus, competitive exclusion happens and all other strains die out except strain \( i \). Meanwhile, we can prove that the single-strain and multistrain viral infection models are well posed. Furthermore, numerical simulations are also carried out to illustrate the theoretical results, which is seldom seen in the relevant known literatures.

1. Introduction

Nowadays, more and more people in the world are dying of various diseases such as AIDS, avian influenza, cholera, Ebola, and Zika virus. To explore the mechanisms of these diseases, scientists have proposed plenty of mathematical models describing the transmission of disease such as the infectious disease model (SI, SIR, SEI, and SIS) and the within-host virus model (HBV, HCV, HIV, Ebola, and Zika). Since Nowak et al. [1, 2] has established the basic within-host virus dynamics model, more and more scholars have devoted to investigate the within-host virus dynamics model and obtained so many significant dynamical results [3–16].

Assuming that the motion of the virus belongs to the Fickian diffusion [17], Wang et al. [5] have firstly investigated a delayed diffusive hepatitis B virus model. Here, we have to mention that the equation with Fickian diffusion implies that random walkers in the system have no interactions. In fact, random walkers in the system may have interactions and the density-dependent diffusion happens [17] and we may establish the equation with density-dependent diffusion. So, parabolic or elliptic systems with density-dependent diffusion have been widely studied by many researchers [18–21]. More recently, Wang et al. have investigated a virus infection model with density-dependent diffusion and Holling II type [6] or Beddington–DeAngelis type incidence function [7] subjected to the homogeneous Neumann boundary conditions. And, they have obtained the significant results of threshold dynamics and competitive exclusion.

It is well known that there are many factors which affect the dynamics of within-host models. One crucial component is incidence function, which can be classified into many different types, such as bilinear type [2], Holling II type [22], Beddington–DeAngelis type [23], and Crowley–Martin type.
Many researchers have concentrated on these within-host models or epidemic models and have obtained some interesting results such as global threshold dynamics [8, 9], delay-induced Hopf bifurcation [10, 11], competitive exclusion [6, 7], traveling wave [12, 13], and pattern formation [14–16]. Recently, Korobeinikov [25] studied an ordinary differential equation model with general incidence function and dose-dependent parasite reproduction and virulence. Huang et al. [26] investigated a viral infection model involving time delay and general incidence function. McCluskey and Yang [27] considered a diffusive viral infection model with general incidence function and time delay. Hattaf and Yousfi [28] studied a diffusive HBV model with two delays and general incidence function. Miao et al. [29] considered the global stability of a diffusive humoral immunity virus infection model with time delay and general incidence function and so on.

Based on the aforementioned works, we firstly propose a single-strain viral infection model with density-dependent diffusion and general incidence function as follows:

\[
\begin{align*}
\frac{\partial u(x,t)}{\partial t} &= \nabla \cdot (D_u(u)\nabla u) + \lambda - du(x,t) - gu((x,t), v(x,t)), \\
\frac{\partial w(x,t)}{\partial t} &= \nabla \cdot (D_w(w)\nabla w) + g(u(x,t), v(x,t)) - \delta w(x,t), \\
\frac{\partial v(x,t)}{\partial t} &= \nabla \cdot (D_v(v)\nabla v) + pv(x,t) - cv(x,t),
\end{align*}
\]

(1)

where \(D_u(u) = u^{m_i}, D_w(w) = w^{m_i}, \) and \(D_v(v) = v^{m_i}\) are the density-dependent diffusion coefficients for \(m_i > 0, i = 1, 2, 3; u(x,t)\) represents the densities of uninfected cells; \(w(x,t)\) represents the densities of infected cells; \(v(x,t)\) represents the densities of free virus at position \(x\) and at time \(t\), respectively; constant \(\lambda\) represents the production rate of the uninfected cells; the death rates of infected cells, infected cells, and free virus are represented by the parameters \(d, \delta,\) and \(c,\) respectively; the infected cells produce the free virions at a rate \(pw;\) further, we assume that the incidence function in this paper is a general incidence function given by \(g(u,v),\) which represents the contacts between target cells and viruses; and the general incidence function \(g(u,v)\) satisfies the following conditions [27].

\[(H_1)\ g: \mathbb{R}_+^2 \to \mathbb{R}_+ \text{ is differentiable; there exists } \eta > 0 \text{ such that } g(u,v) \leq \eta uv \text{ for all } u,v \geq 0; \ g(u,v) > 0 \text{ for all } u,v > 0; \ g(u,0) = g(0,v) = 0 \text{ for all } u,v \geq 0; \ (\partial g(u,v)/\partial u) > 0 \text{ for all } u \geq 0 \text{ and } v > 0; \ (\partial g(u,v)/\partial v) \geq 0 \text{ for all } u,v \geq 0; \ v(\partial g(u,v)/\partial v)g(u,v) \leq 0 \text{ for all } u,v \geq 0.
\]

These assumptions are biologically motivated, see [27] in detail. Moreover, for \(a,b,c,\beta > 0,\) we choose the incidence functions such as \(g(u,v) = (\beta uv/(1 + bv)) [22], g(u,v) = (\beta uv/(1 + au + bv)) [23],\) and \(g(u,v) = (\beta uv/(1 + au + bv + cv)) [24],\) which satisfy \((H_1)\) clearly.

Model (1) we considered in this paper satisfies the following Neumann boundary conditions:

\[
\begin{align*}
D_u(u)x = 0, \\
D_w(w)x = 0, \\
D_v(v)x = 0,
\end{align*}
\]

on \(\partial \Omega \times (0, +\infty),\)

and initial conditions

\[
\begin{align*}
u(x, 0) &= \phi_1(x) \geq 0, \\
w(x, 0) &= \phi_2(x) \geq 0, \\
v(x, 0) &= \phi_3(x) \geq 0, \\
x &\in \bar{\Omega}.
\end{align*}
\]

Here, we give out a bounded domain \(\Omega\) in \(\mathbb{R}^n\) and its boundary \(\partial \Omega\) is smooth, and the outward normal derivative on \(\partial \Omega\) is denoted by \((\partial/\partial n)\). The nonnegative functions \(\phi_i(x,0) (i = 1, 2, 3)\) are Hölder continuous and satisfy \((\partial \phi_i/\partial n) = 0 (i = 1, 2, 3)\) on \(\partial \Omega\).

For (1), we can obtain its steady-state system,

\[
\begin{align*}
-\nabla \cdot (D_u(u)\nabla u) &= \lambda - du(x,t) - g(u(x,t), v(x,t)) = f_1(u,v,w), \\
-\nabla \cdot (D_w(w)\nabla w) &= g(u(x,t), v(x,t)) - \delta w(x,t) = f_2(u,v,w), \\
-\nabla \cdot (D_v(v)\nabla v) &= pv(x,t) - cv(x,t) = f_3(u,v,w),
\end{align*}
\]

(4)

which satisfies boundary conditions (2). We denote

\[
f(u,v,w) = (f_1(u,v,w), f_2(u,v,w), f_3(u,v,w)),
\]

(5)

and let \(f_i(u,v,w) (i = 1, 2, 3)\) satisfy the following conditions.

\((H_2)\) The following relations

\[
\begin{align*}
y_1u^{m_1} + \frac{\partial f_1}{\partial u} &\geq 0, \\
y_2w^{m_2} + \frac{\partial f_2}{\partial w} &\geq 0, \\
y_3v^{m_3} + \frac{\partial f_3}{\partial v} &\geq 0,
\end{align*}
\]

(6)
hold for constants $\gamma_i \geq 0$ ($i = 1, 2, 3$).

Since Gause has discussed the principles that govern species competitive exclusion [30], competitive exclusion has been considered in such settings as within-host models, ecological models, and epidemiological models. Here, we have to point out that Bremermann and Thieme [31] have given out the first rigorous proof of the competitive exclusion principle in the epidemiological model setting. For the multistrain SIR model, they have proved that the strain with the largest reproduction number persists in the population, while the remaining strains die out. This principle was also used in within-host models and has been studied by many scholars [32–37]. For instance, De Leenheer and Pilyugin [32] proved the global competitive exclusion in an ODE version with a multistrain virus. Feng and Velasco-Hernández [33] discussed competitive exclusion in a vector-host model for the dengue fever. Iggidr et al. [34] analyzed globally a new malaria within-host model through a competitive exclusion principle. Wang et al. investigated the competitive exclusion in the virus infection model with density-dependent diffusion and two types of incidence functions [6, 7]. Duan et al. [36] considered the competitive exclusion in the diffusive virus infection model with age of infection and general incidence function, and so on. So, in this paper, we further investigate the competitive exclusion problem for the multistrain virus infection model. Setting $i = 1, 2, \ldots, n$, we can obtain the following multistrain viral infection model with density-dependent diffusion and general incidence function:

$$
\begin{align*}
\frac{d\mathbf{u}(x, t)}{dt} &= \nabla \cdot (D_{\mathbf{u}}(\mathbf{u})\nabla \mathbf{u}) + \lambda - d\mathbf{u}(x, t) - \sum_{i=1}^{n} g_i(\mathbf{u}(x, t), v_i(x, t)), \\
\frac{d\mathbf{w}_i(x, t)}{dt} &= \nabla \cdot (D_{\mathbf{w}_i}(\mathbf{w}_i)\nabla \mathbf{w}_i) + g_i(\mathbf{u}(x, t), v_i(x, t)) - \delta_i \mathbf{w}_i(x, t), \\
\frac{d\mathbf{v}_i(x, t)}{dt} &= \nabla \cdot (D_{\mathbf{v}_i}(\mathbf{v}_i)\nabla \mathbf{v}_i) + p_i \mathbf{w}_i(x, t) - c_i \mathbf{v}_i(x, t), \quad i = 1, 2, \ldots, n.
\end{align*}
$$

Here, we have to stress that the Neumann boundary conditions, initial conditions, and steady-state system of Model (7) are described in Section 3. However, to the best of our knowledge, there are no results on the above models (1) and (7) such as global threshold dynamics and competitive exclusion. So, in this paper, we shall focus on the global threshold dynamics for the single-strain viral infection model (1) and competitive exclusion for the multi-strain viral infection model (7). It is worthwhile to mention that the incidence function discussed in [6, 7] is a special incidence function. But, the opposite case is more difficult and complicated. Through constructing an appropriate Lyapunov functional, Lemma 2, and mathematic analysis skills, we overcome the difficulties successfully and obtain the global threshold dynamics for the single-strain viral infection model (1) and competitive exclusion for the multi-strain viral infection model (7).

The rest of this paper is organized as follows: in Section 2, for the single-strain viral infection model (1), we shall prove the existence, uniqueness, and boundedness of solutions to problems (1)–(4) and consider the global threshold dynamics of the model through constructing appropriate Lyapunov functionals, which is determined by the reproductive numbers for viral infection $\mathcal{R}_0$. In Section 3, for the multistrain viral infection model (7), we also prove the existence, uniqueness, and boundedness of solutions to problems (7) and (42) and discuss the competitive exclusion problem. If the reproduction number $\mathcal{R}_i$ for strain $i$ is maximal and larger than one, the steady state $E_i$ corresponding to the strain $i$ is globally stable. Thus, competitive exclusion happens and all other strains die out except strain $i$. In Section 4, we illustrate our results with numerical simulations, which support and extend the theoretical results. The paper ends with a conclusion.

2. Single-Strain Viral Infection Model

In this section, we shall prove firstly that the single-strain viral infection model (1) is well posed by the method used in [18]. Then, we continue to discuss the existence of equilibria for model (1). Finally, through constructing appropriate Lyapunov functionals, we investigate the global threshold dynamics of model (1), which is determined by the reproductive numbers for viral infection $\mathcal{R}_0$.

2.1. Well Posedness. In this section, we give out some definitions and results first, which can help us to prove that the solutions to problems (1)–(4) exist and are unique and bounded. Similar to the definition method in [6, 7], we can define that two pairs of functions $\bar{\mathbf{U}} = (\bar{\mathbf{u}}, \bar{\mathbf{v}})$, $\breve{\mathbf{U}} = (\breve{\mathbf{u}}, \breve{\mathbf{v}}, \breve{\nu})$ in $C(\Omega \times [0, T]) \cap C^{1,2}(\Omega \times [0, T])$ and $\bar{\mathbf{U}}_i = (\bar{\mathbf{u}}_i, \bar{\mathbf{v}}_i, \bar{\nu}_i)$, $\breve{\mathbf{U}}_i = (\breve{\mathbf{u}}_i, \breve{\mathbf{v}}_i, \breve{\nu}_i)$ in $C(\Omega \times [0, T]) \cap C^{1,2}(\Omega \times [0, T])$ are called coupled upper and lower solutions to system (1)–(3) and to system (4), respectively. Then, we can easily conclude that each pair of coupled upper and lower solutions $\bar{\mathbf{U}}_i, \breve{\mathbf{U}}_i$ of problems (1)–(3) is ones of (4). In what follows, we shall search a pair of constant solutions of (4), which is called upper and lower solutions of (4).
Lemma 1. For constant $l_1 > 0$ and $L_1 > 0$ and any sufficiently large constant $L_2 > 0$ and small constant $l_j > 0$ ($j = 2, 3$), the coupled upper and lower solutions to (4) is a constant pair $L = (L_1, L_2, L_3)$ and $l = (l_1, l_2, l_3)$.

Proof. Obviously, the constant pair $L = (L_1, L_2, L_3)$ and $l = (l_1, l_2, l_3)$ satisfy the homogeneous Neumann boundary conditions (2). Further, by means of condition $(H_1)$, we can confirm that the differential inequalities in (4) can also be satisfied by the constant pair $L$ and $l$ if

$$\lambda - dL_1 - g(L_1, l_3) \leq \lambda - dL_1 \leq 0,$$

$$g(L_1, L_2) - \delta L_2 \leq \eta L_1 L_3 - \delta L_2 \leq 0,$$

$$pL_2 - cL_3 \leq 0;$$

$$\lambda - d_1 - g(l_1, L_3) \geq \lambda - dL_1 - \eta l_1 L_3 = \lambda - (d + \eta L_3) l_1 \geq 0,$$

$$g(l_1, l_3) - \delta l_2 \geq g_0(l_1, l_3) l_3 - \delta l_2 \geq 0,$$

$$p l_2 - c l_3 \geq 0,$$

and then, the above inequalities can be fulfilled for constants $l_1$ and $L_1$, sufficiently large $L_j$, and small $l_j$ ($j = 2, 3$) if

$$\frac{\lambda}{d} \leq L_1,$$

$$\frac{\lambda \eta L_3}{d \delta} \leq L_2 \leq \frac{c L_3}{p},$$

$$l_1 \leq \frac{\lambda}{d + \eta L_3},$$

$$l_2 \leq \frac{g_0(l_1, l_3) l_3}{\delta},$$

$$l_3 \leq \frac{p l_2}{c}.$$  (8)

According to the results of Lemma 1 and method in [6, 7], we can conclude that problems (1)–(4) is well-posed similarly. That is, the solutions to problems (1)–(4) exist and are unique and bounded.  

2.2. Equilibria. To describe that a single infected cell generates the average number of newly infected cells when the disease has just entered the body, we define the following basic reproduction number:

$$R_0 = \frac{p}{c \delta} \frac{\partial g((\lambda/d), 0)}{\partial \nu},$$  (10)

which is independent of spatial variety. Obviously, $E_0 = ((\lambda/d), 0, 0)$ is always an infection-free equilibrium of model (1). Similar to the analysis and discussion in [27], together with the basic reproduction number $R_0$, it is not hard to see that model (1) has only a positive equilibrium $E^* = (u^*, w^*, v^*)$ if $R_0 > 1$. So, we have the results on the equilibria of model (1) as follows.

Theorem 1. The infection-free equilibrium $E_0 = ((\lambda/d), 0, 0)$ of model (1) always exists. If $R_0 > 1$, model (1) has only an infection equilibrium $E^* = (u^*, w^*, v^*)$.

2.3. Stability Properties of Model (1)

2.3.1. The Local Stability of Model (1). In this section, we shall investigate the local stability of model (1). Under the homogeneous Neumann boundary conditions, assume that

$$0 = \nu_0 < \nu_1 < \cdots < \nu_q < \cdots$$

be the eigenvalues of $-\Delta$ on $\Omega$ and for $q = 0, 1, 2, \ldots$, and $E(\nu_q)$ be the eigenfunctions space corresponding to $\nu_q$ in $C^1(\Omega)$. Suppose that $E(\nu_q)$ has an orthonormal basis $\{\phi_{qm}; m = 1, 2, \ldots, dim E(\nu_q)\}$, $X = [C^1(\Omega)]^3$, and $X_{qm} = \{c \phi_{qm}; c \in \mathbb{R}^3\}$. Thus, we obtain

$$X = \bigoplus_{q=1}^{\infty} X_q,$$

$$X_q = \bigoplus_{m=1}^{dim E(\nu_q)} X_{qm}.$$  (11)

Linearizing system (1) at the equilibrium $(\bar{u}, \bar{w}, \bar{v})$ is as follows:

$$\frac{\partial u(x, t)}{\partial t} = D_u(u) \Delta u - d u(x, t) - \frac{\partial g(\bar{u}, \bar{v})}{\partial u} u(x, t) - \frac{\partial g(\bar{u}, \bar{v})}{\partial v} v(x, t),$$

$$\frac{\partial w(x, t)}{\partial t} = D_w(w) \Delta w + \frac{\partial g(\bar{u}, \bar{v})}{\partial u} u(x, t) + \frac{\partial g(\bar{u}, \bar{v})}{\partial v} v(x, t) - \delta w(x, t),$$

$$\frac{\partial v(x, t)}{\partial t} = D_v(v) \Delta v + p w(x, t) - c v(x, t).$$  (12)
Note that, under the linearization, $X_q$ is an invariant for each $q = 0, 1, 2, \ldots$. We let the expression of solution be $Q(x, t) = e^{\delta t} \psi(x)$, where $\psi(x) \in X_q$ satisfies $-\Delta \psi = \nu_q \psi$. Thus, the characteristic equation (13) is changed into

$\left( \mu + D_u (\overline{u}) \nu_q + d + \frac{\partial g(u, \overline{v})}{\partial u} \right)$

$\cdot \left( \mu + D_w (\overline{w}) \nu_q + \delta \right) \left( \mu + D_v (\overline{v}) \nu_q + c - p \frac{\partial g(u, \overline{v})}{\partial v} \right)$

$+ p \frac{\partial g(u, \overline{v})}{\partial u} = 0.$

(13)

When the equilibrium of model (1) is $E_0 = ((\lambda/d), 0, 0)$, the characteristic equation (13) is changed into

$\left( \mu + D_u \left( \frac{\lambda}{d} \right) \nu_q + d \right) \left( \mu + \delta (\mu + c) - p \frac{\partial g((\lambda/d), 0)}{\partial v} \right) = 0.$

(14)

Obviously, equation (14) has a negative real root $\sigma = -D_u (\lambda/d) \nu_q - d$. We now discuss the transcendental equation as follows:

$\left( \mu + \delta \right) (\mu + c) - p \frac{\partial g((\lambda/d), 0)}{\partial v} = 0.$

(15)

Set

$H(\mu, q) = (\mu + \delta) (\mu + c) - p \frac{\partial g((\lambda/d), 0)}{\partial v}$

(16)

Case 1. $\mathcal{R}_0 < 1$. By direct computation, we have

$H(0, q) = c \delta - p \frac{\partial g((\lambda/d), 0)}{\partial v} = c \delta (1 - \mathcal{R}_0) > 0,$

(17)

and for $\mu \geq 0$,

$\frac{\partial H(\mu, q)}{\partial \mu} = 2 \mu + \delta + c > 0.$

(18)

So, there is only a negative real root for equation (14).

Assume that there be a complex root $\rho + i \omega$ with $\rho \geq 0$, then we can conclude that

$\left| \frac{\mu}{\delta} + 1 \right| > 1,$

$\left| \frac{\mu}{C} + 1 \right| > 1,$

(19)

which implies

$\left| \frac{\mu}{\delta} + 1 \right| \left| \frac{\mu}{C} + 1 \right| > \mathcal{R}_0 = \frac{\partial g((\lambda/d), 0)}{\partial v}.$

(20)

This contradicts to equation (15). Then, all roots of equation (14) have no positive real parts yet. Thus, the infection-free equilibrium $E_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$.

Case 2. $\mathcal{R}_0 > 1$. Obviously, we need to only consider the space $X_q$ corresponding to $\nu_q = 0$ when $q = 0$. By direct computation, we get

$H(0, 0) = c \delta (1 - \mathcal{R}_0) > 0,$

(21)

$\lim_{\mu \to \infty} H(\mu, 0) = +\infty.$

This implies that the equation $H(\mu, 0) = 0$ must hold for some $\mu_0 > 0$. Thus, there exists at least one positive root of equation (14). So, the infection-free equilibrium $E_0$ is not stable for $\mathcal{R}_0 > 1$. Then, we have the following results.

Theorem 2. The infection-free equilibrium $E_0$ of model (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$. However, $E_0$ is unstable if $\mathcal{R}_0 > 1$.

When the equilibrium of model (1) is $E^* = (u^*, w^*, v^*)$, the characteristic equation (13) is changed into

$\mu^3 + A \mu^2 + B \mu + C = 0,$

(22)

where

$A = D_u (u^*) \nu_q + D_w (w^*) \nu_q + D_v (v^*) \nu_q + d + \delta + c + \frac{\partial g(u^*, v^*)}{\partial u},$

$B = \left( D_u (u^*) \nu_q + d + \frac{\partial g(u^*, v^*)}{\partial u} \right) \left( D_w (w^*) \nu_q + D_v (v^*) \nu_q + \delta + c \right) + \left( D_w (w^*) \nu_q + d \right) \left( D_v (v^*) \nu_q + c - p \frac{\partial g(u^*, v^*)}{\partial v} \right) - p \frac{\partial g(u^*, v^*)}{\partial v},$

$C = \left( D_u (u^*) \nu_q + d + \frac{\partial g(u^*, v^*)}{\partial u} \right) \left( D_w (w^*) \nu_q + \delta \right) \left( D_v (v^*) \nu_q + c - p \frac{\partial g(u^*, v^*)}{\partial v} \right) + p \frac{\partial g(u^*, v^*)}{\partial u} \frac{\partial g(u^*, v^*)}{\partial v}.$

(23)

It is easy to see that $A > 0$. From the condition $(H_1)$, together with $g(u^*, v^*) = \delta w^* + \phi w^* = cv^*$, we get

$p \frac{\partial g(u^*, v^*)}{\partial v} \leq \frac{\partial g(u^*, v^*)}{\partial v} = \frac{p \delta w^*}{(pw^*/c)} = c \delta,$

(24)
which yields \( B > 0 \) and \( C > 0 \). By direct computation, we have that

\[
AB - C = \left[ D_u(u^*)v_q + D_w(w^*)v_y + D_v(v^*)v_q + d + \frac{\partial g(u^*, v^*)}{\partial u} \right] \left[ D_u(u^*)v_q + d + \frac{\partial g(u^*, v^*)}{\partial u} \right] \\
\cdot \left[ D_w(w^*)v_q + D_v(v^*)v_q + \delta + c \right] + \left[ D_u(u^*)v_q + d \right] \left[ c + D_w(w^*)v_q + D_v(v^*)v_q + \delta \right] \\
+ c \frac{\partial g(u^*, v^*)}{\partial u} \left[ D_w(w^*)v_q + D_v(v^*)v_q + c \right] + \frac{\partial g(u^*, v^*)}{\partial u} \left[ c\delta - p\frac{\partial g(u^*, v^*)}{\partial v} \right] > 0.
\]

So, we can see that all eigenvalues of (22) have no positive real parts by the Routh–Hurwitz criterion. Therefore, \( E^* \) is locally asymptotically stable for \( R_0 > 1 \). Thus, we obtain the following results.

**Theorem 3.** The infection equilibrium \( E^* \) of model (1) is locally asymptotically stable if \( R_0 > 1 \).

2.3.2. The Global Stability of Model (1). In this section, by constructing appropriate Lyapunov functionals, we continue to investigate the global stability of model (1). To this end, we need the following lemma.

**Lemma 2.** Under homogeneous Neumann boundary condition, for a density-dependent diffusion problem, we can obtain the following result:

\[
\int_{\Omega} \frac{1}{g(u)} \nabla \cdot (D(u)\nabla u) \, dx = \int_{\Omega} D(u)g'(u)\|\nabla u\|^2 \, dx,
\]

where \( \Omega \) is a bounded domain in \( \mathbb{R}^n \) and its boundary \( \partial \Omega \) is smooth.

**Proof.** According to the homogeneous Neumann boundary condition, we get

\[
\int_{\Omega} \frac{1}{\alpha g(u)} \nabla \cdot (D(u)\nabla u) \, dx = \int_{\Omega} \frac{1}{\alpha g(u)} D(u)g'(u)\|\nabla u\|^2 \, dx + \int_{\Omega} \frac{1}{\alpha g(u)} D(u)\Delta u \, dx
\]

\[
= \int_{\Omega} \frac{1}{\alpha g(u)} D(u)g'(u)\|\nabla u\|^2 \, dx + \int_{\Omega} \frac{1}{\alpha g(u)} D(u)\frac{\partial u}{\partial \nu} \, ds - \int_{\Omega} D(u)\left( \nabla \left( \frac{1}{g(u)} \right) \cdot \nabla u \right) \, dx
\]

\[
- \int_{\Omega} \frac{1}{\alpha g(u)} D(u)\|\nabla u\|^2 \, dx = \int_{\Omega} \frac{D(u)g'(u)\|\nabla u\|^2}{g^2(u)} \, dx.
\]

This completes the proof of Lemma 2. \( \square \)

**Remark 1.** Choosing \( g(u) = 1, u, \) and \( (1/u) \) in Lemma 2, respectively, we can obtain that

(i) \( \int_{\Omega} \nabla \cdot (D(u)\nabla u) \, dx = 0; \)

(ii) \( \int_{\Omega} (1/u)\nabla \cdot (D(u)\nabla u) \, dx = \int_{\Omega} (D(u)\|\nabla u\|^2/u^2) \, dx; \)

(iii) \( \int_{\Omega} u\nabla \cdot (D(u)\nabla u) \, dx = -\int_{\Omega} D(u)\|\nabla u\|^2 \, dx; \)

which implies that Lemma 2 generalized the results in [6, 7].

Firstly, we discuss the global asymptotic stability of the infection-free equilibrium \( E_0 \) of model (3).

**Theorem 4.** If \( R_0 \leq 1 \), then the infection-free equilibrium \( E_0 \) of model (3) is globally asymptotically stable.

**Proof.** Define the following Lyapunov functional:

\[
V(t) = \int_{\Omega} \left[ w(x, t) + \frac{\delta}{p} v(x, t) \right] \, dx.
\]
Through calculating the derivative of $V(t)$ about time, we can get

$$\frac{dV(t)}{dt} = \int_{\Omega} \left[ \frac{\partial w}{\partial t} + \delta \frac{\partial v}{\partial t} \right] dx$$

$$= \int_{\Omega} \left[ \nabla \cdot (D(w)\nabla w) + g(u(x,t),v(x,t)) - \delta w(x,t) + \frac{\delta}{p} \cdot (D(v)\nabla v) + \delta w(x,t) \cdot \frac{c\delta}{p} v(x,t) \right] dx$$

$$= \int_{\Omega} g(u(x,t),v(x,t)) \frac{c\delta}{p} v(x,t) \right] dx + \int_{\Omega} \nabla \cdot (D(w)\nabla w) dx + \frac{\delta}{p} \int_{\Omega} \nabla \cdot (D(v)\nabla v) dx.$$ (29)

Note that $\frac{\partial u}{\partial t} \leq \lambda - du$, then we have $\lim_{t \to \infty} u(x,t) \leq (\lambda/d)$, which means that all omega limit points satisfy $u \leq (\lambda/d)$. So, we need to only consider solutions for which $u \leq (\lambda/d)$. Since $(\partial g/\partial u) \geq 0$, we can conclude the inequality $g(u,v) \leq g((\lambda/d),v)$. Under the condition $(H_1)$, together with the fact that $(g((\lambda/d),v)/v)$ is decreasing in $v$, we can obtain

$$g(u(x,t),v(x,t)) - \frac{c\delta}{p} v(x,t) = \frac{c\delta}{p} v \left( \frac{p}{c\delta} \frac{g(u,v)}{v} - 1 \right)$$

$$\leq \frac{c\delta}{p} v \left( \frac{p}{\delta} \frac{g((\lambda/d),v)}{v} - 1 \right) \leq \frac{c\delta}{p} v \left( \lim_{v \to \infty} \frac{p}{c\delta} \frac{g((\lambda/d),v)}{v} - 1 \right)$$

$$= \frac{c\delta}{p} v \left( \frac{p}{\delta} \frac{\partial g((\lambda/d),0)}{\partial v} - 1 \right) = \frac{c\delta}{p} v(\mathcal{R}_0 - 1).$$ (30)

Meanwhile, from Remark 1, we get

$$\int_{\Omega} \nabla \cdot (D(w)\nabla w) dx = 0 = \frac{\delta}{p} \int_{\Omega} \nabla \cdot (D(v)\nabla v) dx.$$ (31)

Thus, we obtain

$$\frac{dV(t)}{dt} \leq \frac{c\delta}{p} (\mathcal{R}_0 - 1) \int_{\Omega} v dx.$$ (32)

So, we have that $(dV(t)/dt) \leq 0$ when $\mathcal{R}_0 \leq 1$. Further, it is not hard to believe that $(dV(t)/dt) \leq 0$ leads to either $v = 0$ or $\mathcal{R}_0 = 1$ and $u = (\lambda/d)$. It is easy to confirm that the singleton $E_0$ is just the largest compact invariant set in $\{u,w,v\} (dV(t)/dt) \leq 0$. When $\mathcal{R}_0 \leq 1$, we can know that the infection-free equilibrium $E_0$ of model (3) is globally asymptotically stable by the LaSalle’s invariance principle. The proof of Theorem 4 is complete.

Next, we investigate the global asymptotic stability problem of the infection equilibrium $E^*$ of model (3). To prove the following theorem, we need the following hypothesis:

$$(H_3) \left( \frac{v}{\nu} - \frac{g(u,v)}{g(u,v)} \right) \left( \frac{g(u,v)}{g(u,v)} - 1 \right) \geq 0, \quad \text{for all } u, v > 0.$$ (33)

**Theorem 5.** Suppose $(H_3)$ is satisfied. If $\mathcal{R}_0 > 1$, then the infection equilibrium $E^*$ of model (3) is globally asymptotically stable.

**Proof.** Let $H(z) = z - 1 - \ln z$, $z > 0$, and define

$$V(t) = \int_{\Omega} \left\{ u - u^* - \int_{u^*}^{u} \frac{g(u,v)}{g(s,v)} ds \right\} + \frac{\delta}{p} \int_{\Omega} H \left( \frac{v}{\nu} \right) dx.$$ (34)

which is called a Lyapunov functional. Obviously, 0 is the minimum value of function $H(z)$ at $z = 1$. This leads to $H(z) \geq 0$. Meanwhile, 0 is also the global minimum of function $G: u \to u - u^* - \int_{u^*}^{u} (g(u,v)/g(s,v)) ds$ at $u = u^*$, which implies $G(u) \geq 0$ for any $x > 0$. Therefore, for all $x \in \Omega$ and $t \geq 0, V(t) \geq 0$ with equality holding if and only if $u(x,t)/u^* = (w/w^*) = (v(x,t)/v^*) = 1$.

Through calculating the derivative of $V(t)$ about time, we can get
\[
\frac{dV(t)}{dt} = \int_\Omega \left( 1 - \frac{g(u^*, v^*)}{g(u, v^*)} \right) \frac{\partial u}{\partial t} + \left( 1 - \frac{w^*}{w} \right) \frac{\partial w}{\partial t} + \frac{\delta}{p} \left( 1 - \frac{\nu^*}{\nu} \right) \frac{\partial \nu}{\partial t} \right] dx \\
= \int_\Omega \left( 1 - \frac{g(u^*, v^*)}{g(u, v^*)} \right) [\nabla \cdot (D(u)\nabla u) + \lambda - du(x, t) - g(u(x, t), v(x, t))] \\
+ \left( 1 - \frac{w^*}{w} \right) [\nabla \cdot (D(w)\nabla w) + g(u(x, t), v(x, t)) - \delta w(x, t)] + \frac{\delta}{p} \left( 1 - \frac{\nu^*}{\nu} \right) [\nabla \cdot (D(v)\nabla v) + pw(x, t) - cv(x, t)] \right] dx.
\]

(35)

Note that \( \lambda = du^* + g(u^*, v^*), \) \( \delta w^* = g(u^*, v^*), \) and \( cv^* = pw^* \). So, we have

\[
\frac{dV(t)}{dt} = \int_\Omega \left( 1 - \frac{g(u^*, v^*)}{g(u, v^*)} \right) [\nabla \cdot (D(u)\nabla u) + du^* - du(x, t) + g(u^*, v^*) - g(u(x, t), v(x, t))] \\
+ \left( 1 - \frac{w^*}{w} \right) [\nabla \cdot (D(w)\nabla w) + g(u(x, t), v(x, t)) - \frac{w(x, t)}{w^*} g(u^*, v^*)] + \frac{\delta}{p} \left( 1 - \frac{\nu^*}{\nu} \right) [\nabla \cdot (D(v)\nabla v)] \\
+ p \left( w(x, t) - \frac{\nu(x, t)}{\nu^*} w^* \right) \right] dx
\]

(36)
From the hypothesis \((H_1)\), we can know that \(g(u, v')\) is increasing in \(u\) strictly. Hence, we have
\[
\left(1 - \frac{u}{u^*}\right)\left(1 - \frac{g(u, v')}{g(u, v^*)}\right) \leq 0, \tag{37}
\]
and \((1 - (u/u^*)) (1 - (u'/(u^*)^2))\) equals 0 only if \(u = u^*\). Based on the assumption \((H_3)\), we can conclude that
\[
\frac{dV(t)}{dt} \leq -g(u^*, v') \int_\Omega \left[ H\left(\frac{g(u, v')}{g(u, v^*)}\right) + H\left(\frac{w(x,t)v^*}{v(x,t)w^*}\right) \right] + \int_\Omega \left(1 - \frac{w^*}{w}\right) \nabla \cdot (D(u)\nabla u)dx + \int_\Omega \left(1 - \frac{v^*}{v}\right) \nabla \cdot (D(v)\nabla v)dx. \tag{38}
\]

From Lemma 2 and Remark 1, it is easy to conclude that
\[
\frac{dV(t)}{dt} \leq 0. \tag{39}
\]

Hence, \(E^*\) is stable and \((dV(t)/dt) = 0\) iff \(u = u^*\), \(w = w^*\), and \(v = v^*\). It is easy to confirm that the singleton \(\{u^*, w^*, v^*\}\) is just the largest compact invariant set in \(\{(u, w, v) | (dV(t)/dt) = 0\}\). According to the LaSalle’s invariance principle, we can know that the infection equilibrium \(E^*\) of model (3) is globally asymptotically stable when \(\mathcal{R}_0 > 1\). This completes the proof of Theorem 5. \(\square\)

3. Multistrain Viral Infection Model

In this section, we shall begin to study the multi-strain viral infection model (7) with Neumann boundary conditions:
\[
\begin{align*}
D_u(u) \frac{\partial u}{\partial n} &= 0, \\
D_w(w_i) \frac{\partial w_i}{\partial n} &= 0, \\
D_v(v_i) \frac{\partial v_i}{\partial n} &= 0, \\
\text{on } \partial \Omega \times (0, +\infty),
\end{align*}
\tag{40}
\]
and initial conditions
\[
\begin{align*}
u(x, 0) &= \phi_i(x) \geq 0, \\
\omega_i(x, 0) &= \phi_{i\omega}(x) \geq 0, \\
\nu_i(x, 0) &= \phi_{i\nu}(x) \geq 0, \\
x &\in \Omega.
\end{align*}
\tag{41}
\]

Suppose \(\Omega\) is a bounded domain in \(\mathbb{R}^n\) and its boundary \(\partial \Omega\) is smooth, then the outward normal derivative on \(\partial \Omega\) is denoted by \(\nabla\). The nonnegative functions \(\phi_i(x, 0)\) and \((g(u(x,t), v(x,t))/g(u(x,t), v^*))\) lies between 1 and \((v(x,t)/v^*)\). Thus, by the fact that \(H(z)\) is a monotone function on each side of 1, we can obtain that \(H(g(u(x,t), v(x,t))/g(u(x,t), v^*)) \leq H(v(x,t)/v^*)\).

Therefore,
\[
\phi_{r,i}(x, 0) \ (r = 2, 3, i = 1, 2, \ldots, n) \text{ are H"older continuous and satisfy } (\partial \phi_{r,i}/\partial \nu) = 0 \text{ and } (\partial \phi_{r,i}/\partial v) = 0 \text{ on } \partial \Omega, \text{ respectively. Moreover, we can get the steady-state system of model (7) as follows:}
\[
\begin{align*}
-\nabla \cdot (D_u(u)\nabla u) &= \lambda - du(x,t) - \sum_{i=1}^{n} g_i(u(x,t), v_i(x,t)), \\
-\nabla \cdot (D_w(w_i)\nabla w_i) &= g_i(u(x,t), v_i(x,t)) - \delta_i w_i(x,t), \\
-\nabla \cdot (D_v(v_i)\nabla v_i) &= p_i w_i(x,t) - c_i v_i(x,t), \quad i = 1, 2, \ldots, n,
\end{align*}
\tag{42}
\]
where \(D_u(u) = u^{m_1}, D_w(w_i) = w_i^{m_2},\) and \(D_v(v_i) = v_i^{m_3}\) are the density-dependent diffusion coefficients for \(m_1 > 0\) and \(m_{r,i} > 0, r = 2, 3, i = 1, 2, \ldots, n\).

3.1. Well Posedness. In this section, we give out some definitions and results first, which can help us to prove that the solutions to problems (7) and (42) exist and are unique and bounded. Similar to the definition method in [6, 7], we can define that two pairs of functions \(\bar{U} = (\bar{u}, \bar{v_i}, \bar{v}), \bar{U} = (\bar{u}, \bar{v_i}, \bar{v}), \) \((\bar{\Omega} \times [0, T]) \cap C^{1,2} (\Omega \times [0, T])\) and \(\bar{U} = (\bar{u}, \bar{v_i}, \bar{v}), \bar{U} = (\bar{u}, \bar{v_i}, \bar{v}), \) \((\bar{\Omega} \times [0, T]) \cap C^{1,2} (\Omega \times [0, T])\) are called coupled upper and lower solutions to system (7) and to system (42), respectively. Then, we can easily conclude that each pair of coupled upper and lower solutions \(\bar{U}_s, \bar{U}_s\) to problems (7) is also ones of (42). In what follows, we shall search a pair of constant solutions of (42), which is called upper and lower solutions of (42).

Lemma 3. For constant \(l_i > 0\) and \(L_i > 0\) and any sufficiently large constant \(L_{2j} > 0\) and \(L_{2j} > 0\) and small constant \(l_{2j} > 0\) and \(l_{2j} > 0, i = 1, 2, \ldots, n\), the coupled upper and lower solutions to (42) are a constant pair \(L = (L_1, L_{2j}, L_{2j})\) and \(l = (l_1, l_{2j}, l_{2j})\).
Proof. Obviously, the constant pair $L = (L_1, L_{2,j}, L_{3,j})$ and $I = (l_1, l_{2,j}, l_{3,j})$ satisfy the homogeneous Neumann boundary conditions (40). Further, by means of the condition ($H_1$), we can confirm that the differential inequalities in (42) can also be satisfied by a constant pair $L$ and $I$ if

$$\begin{align*}
\lambda - dL_1 - \sum_{i=1}^{n} g_i(L_1, L_{3,i}) &\leq \lambda - dL_1 \leq 0, \\
g_i(L_1, L_{3,i}) - \delta L_{2,j} &\leq \eta L_1 L_{3,i} - \delta L_{2,j} \leq 0, \\
p_i L_{2,j} - c_i L_{3,i} &\leq 0; \\
\lambda - dL_1 - \sum_{i=1}^{n} g_i(l_1, L_{3,i}) &\geq \lambda - dL_1 \\
- \sum_{i=1}^{n} \eta_i l_{3,i} &\leq - \sum_{i=1}^{n} \eta_i L_{3,i} \geq 0, \\
g_i(l_1, L_{3,i}) - \delta L_{2,j} &\geq g_i(l_1, L_{3,i}) - \delta L_{2,j} \geq 0, \\
p_i l_{2,j} - c_i L_{3,i} &\geq 0.
\end{align*}$$

(43)

Then, the above inequalities can be fulfilled for constants $l_1$ and $L_1$, sufficiently large $L_{2,j}$ and $L_{3,i}$, and small $l_{2,j}$ and $l_{3,i}$ ($i = 1, 2, \ldots, n$) if

$$\begin{align*}
\frac{\lambda - dL_1}{d} &\leq L_1, \\
\frac{\lambda - dL_1}{d} &\leq \frac{L_{2,j}}{c_i} \leq \frac{c_i L_{3,i}}{p}, \\
l_1 &\leq \frac{\lambda}{d + \sum_{i=1}^{n} \eta_i L_{3,i}}, \\
g_i(l_1, L_{3,i}) - \delta L_{2,j} &\geq \frac{g_i(l_1, L_{3,i}) - \delta L_{2,j}}{\delta_i}, \\
l_{3,i} &\leq \frac{p_i l_{2,j}}{c_i}.
\end{align*}$$

(44)

To obtain the upper solution of (42), we only need to take $L_1 = (\lambda/d)$ and sufficiently large $L_{2,j}$ and $L_{3,i}$ satisfying $(\lambda \eta_i L_{3,i}/d \delta_i) \leq L_{2,j} \leq (c_i L_{3,i}/p)$. Meanwhile, choosing sufficiently small $l_{3,i}$, $l_{2,j} = (c_i L_{3,i}/p)$, and $l_1$ satisfies $(c_i / p) \leq g_i(l_1, L_{3,i})$ and $l_1 \leq (\lambda/d + \sum_{i=1}^{n} \eta_i)$, and we can also obtain the lower solution of (42). This means that the coupled upper and lower solutions of (42) are a constant pair $L$ and $I$. This completes the proof of Lemma 3.

According to the results of Lemma 3 and method in [6, 7], we can conclude that problems (7) and (42) are well-posed similarly. That is, the solutions to problems (7) and (42) exist and are unique and bounded. □

3.2. Competitive Exclusion. To describe a single-strain $i$-infected cell which generates the average number of newly infected cells when the disease has just entered the body, we define

$$R_i = \frac{p_i v_i}{c_i \delta_i \min \left\{ g_i(u(s, s'), v_i(s)) - \frac{\partial g_i(u(s, s'), v_i(s))}{\partial v_i} \right\}},$$

(45)

which is called the basic reproduction number of strain $i$ in model (7). Obviously, $E_0^n = ((\lambda/d), 0, 0)$ is always an infection-free steady state of system (7), where $0$ is an $n$-dimensional vector of zeros. Similar to the analysis and discussion in [27], together with the basic reproduction number $R_i$, it is not hard to see that for each strain $i$, model (7) has only a corresponding single-strain steady state as follows:

$$E_i = (u_i, 0, \ldots, 0, \omega^*_i, 0, \ldots, 0, \psi^*_i, 0, \ldots, 0).$$

(46)

iff $R_i > 1$ and it satisfies

$$\begin{align*}
\lambda - du(s, t) - g_i(u(s, t), v_i(s, t)) = 0, \\
g_i(u(s, t), v_i(s, t)) - \delta_i u_i(s, t) = 0, \\
p_i u_i(s, t) - c_i v_i(s, t) = 0.
\end{align*}$$

(47)

Here, we have to point out that the nonzero components $\omega^*_i$ is in position $i + 1$ and $\psi^*_i$ are in position $n + i + 1$.

Now, we begin to investigate the competitive exclusion problem of model (7). From the results in [36], to discuss the competitive exclusion problem, we only need to discuss the globally asymptotically stability of steady state $E_i$ of strain one. Next, we shall verify that the globally asymptotically stability of the infected steady state $E_i$ of strain one by constructing an appropriate Lyapunov functional. Here, we also assume that for $i = 1, 2, \ldots, n$, $g_i(u, v_i)$ satisfies the condition ($H_1$) and $\eta_k < \eta_1, k = 2, \ldots, n$. Moreover, we need the following condition:

$$\begin{align*}
(H_4) \eta_k p_k / c_k \delta_k &\leq \frac{d}{\lambda} \frac{p_1}{c_1 \delta_1} \min \left\{ g_i(u(s, s'), v_i(s)) - \frac{\partial g_i(u(s, s'), v_i(s))}{\partial v_i} \right\}.
\end{align*}$$

(48)

Theorem 6. Suppose ($H_3$) and ($H_4$) are satisfied. For $k = 2, \ldots, n$, let $R_1 > 1$ and $R_k > R_k$ hold. Then, the single-strain steady state $E_i$ of model (7) is globally asymptotically stable.

Proof. Let $H(z) = z - 1 - \ln z, z > 0$ and define

$$V(t) = \int_{\alpha}^{\beta} \left\{ u - u_i^* - \int_{u_i^*}^{u(x)} \frac{g_i(u^*, v_i^*)}{g_i(s, v_i^*)} ds + w_i^* H\left( \frac{w_i}{w_i^*} \right) + \frac{\delta_i v_i^*}{P_i} H\left( \frac{v_i}{v_i^*} \right) + \sum_{k=2}^{n} \frac{\delta_k v_k}{P_k} \right\} dx.$$  

(49)
$H(z) \geq 0$. Meanwhile, the $0$ is also the global minimum of 
function $G: u \rightarrow u - u_t^* - \int_{\Omega} (g_1(u_t^*, v_1^*)/g_1(s, v_1^*)) ds$ at 
$u = u_t^*$, which implies $G(u) \geq 0$ for any $x > 0$. Therefore, 
$V(t) \geq 0$ with equality holding if and only 
$(u(x,t)/u_t^*) = (w_1(x,t)/w_t^*) = (v_1(x,t)/v_1^*) = 1$ and $w_k = v_k = 0$ ($k = 2, 
\ldots , n$) for all $x \in \Omega$ and $t \geq 0$.

Through calculating the derivative of $V(t)$ about time, we get

$$
\frac{dV(t)}{dt} = \int_{\Omega} \left\{ \left(1 - \frac{g_1(u_t^*, v_1^*)}{g_1(u, v_t^*)}\right) \frac{\partial u}{\partial t} + \left(1 - \frac{w_t^*}{w_1}\right) \frac{\partial w_1}{\partial t} + \frac{\delta_1}{p_1} \left(1 - \frac{v_1^*}{v_1}\right) \frac{\partial v_1}{\partial t} + \sum_{k=2}^{n} \left( \frac{\partial w_k}{\partial t} + \frac{\delta_k}{p_k} \frac{\partial v_k}{\partial t} \right) \right\} dx
$$

$$
= \int_{\Omega} \left\{ \left(1 - \frac{g_1(u_t^*, v_1^*)}{g_1(u, v_t^*)}\right) [\nabla \cdot (D(u)\nabla u) + \lambda - d u (x, t) - g_1(u(x,t), v_1(x,t))] \right.
$$

$$
+ \left(1 - \frac{w_t^*}{w_1}\right) [\nabla \cdot (D(w_1)\nabla w_1) + g_1(u(x,t), v_1(x,t)) - \delta_1 w_1(x,t)]
$$

$$
+ \frac{\delta_1}{p_1} \left(1 - \frac{v_1^*}{v_1}\right) [\nabla \cdot (D(v_1)\nabla v_1) + p_1 w_1(x,t) - c_1 v_1(x,t)] - \left(1 - \frac{g_1(u_t^*, v_1^*)}{g_1(u, v_t^*)}\right) \sum_{k=2}^{n} g_k(u(x,t), v_k(x,t))
$$

$$
+ \sum_{k=2}^{n} \left( \frac{\delta_k}{p_k} [\nabla \cdot (D(w_k)\nabla w_k) + g_k(u(x,t), v_k(x,t)) - \delta_k w_k(x,t)] \right) \sum_{k=2}^{n} \frac{\delta_k}{p_k} [\nabla \cdot (D(v_k)\nabla v_k) + p_k w_k(x,t) - c_k v_k(x,t)] \right\} dx.
$$

(50)
Note that $\lambda = du^*_t + g_1(u^*_t, v^*_t)$, $\delta_i w^*_t = g_1(u^*_t, v^*_t)$, and $c_1 v^*_t = p_1 w^*_t$. So, we have

$$\frac{dV(t)}{dt} = \int_\Omega \left\{ \left( 1 - \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} \right) \left( \nabla \cdot (D(u)\nabla u) + du^*_t - du(x, t) + g_1(u^*_t, v^*_t) - g_1(u(x, t), v_1(x, t)) \right) \right. $n \left. + \left( 1 - \frac{\omega^*_t}{\omega_1} \right) \left( \nabla \cdot (D(w_1)\nabla w_1) + g_1(u(x, t), v_1(x, t)) - \frac{\omega_1(x, t)}{\omega^*_t} g_1(u^*_t, v^*_t) \right) \right. $n \left. + \left. \frac{\delta_i}{p_i} \left( 1 - \frac{v^*_t}{v_1} \right) \left( \nabla \cdot (D(v_1)\nabla v_1) + p_i \left( \omega_1(x, t) - \frac{v_1(x, t)}{v^*_t} \omega^*_t \right) \right) \right. $n \left. + \sum_{k=2}^n \left[ \nabla \cdot (D(w_k)\nabla w_k) + \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} g_k(u(x, t), v_k(x, t)) - \delta_k w_k(x, t) \right] \right. $n \left. + \sum_{k=2}^n \frac{\delta_k}{p_k} \left[ \nabla \cdot (D(v_k)\nabla v_k) + p_k w_k(x, t) - c_k v_k(x, t) \right] \right\} dx $n \left. - \int_\Omega \left( 1 - \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} \right) \nabla \cdot (D(u)\nabla u) dx + \int_\Omega \left( 1 - \frac{\omega^*_t}{\omega_1} \right) \nabla \cdot (D(w_1)\nabla w_1) dx \right. $n \left. + \frac{\delta_i}{p_i} \int_\Omega \left( 1 - \frac{v^*_t}{v_1} \right) \nabla \cdot (D(v_1)\nabla v_1) dx + \sum_{k=2}^n \int_\Omega \nabla \cdot (D(w_k)\nabla w_k) dx + \sum_{k=2}^n \frac{\delta_k}{p_k} \int_\Omega \nabla \cdot (D(v_k)\nabla v_k) dx \right. $n \left. = \int_\Omega \left\{ du^*_t \left( 1 - \frac{u}{u^*_t} \right) \left( 1 - \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} \right) + g_1(u^*_t, v^*_t) \left[ \left( 1 - \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} \right) \left( 1 - \frac{g_1(u(x, t), v_1(x, t))}{g_1(u^*_t, v^*_t)} \right) \right. $n \left. - \left. \frac{\omega_1(x, t)}{\omega^*_t} g_1(u(x, t), v_1(x, t)) \right) + H\left( \frac{g_1(u(x, t), v_1(x, t))}{g_1(u^*_t, v^*_t)} \right) \right. $n \left. - \frac{\omega_1(x, t)}{\omega^*_t} g_1(u(x, t), v_1(x, t)) \right) - \left. \frac{\omega_1(x, t)}{\omega^*_t} g_1(u^*_t, v^*_t) \omega(x, t) \right) - \left. \frac{\omega_1(x, t)}{\omega^*_t} g_1(u^*_t, v^*_t) \right] \right. $n \left. \right\} dx + \sum_{k=2}^n \int_\Omega \left[ \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} g_k(u(x, t), v_k(x, t)) - \frac{c_k}{p_k} v_k(x, t) \right] dx \right. $n \left. + \int_\Omega \left( 1 - \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} \right) \nabla \cdot (D(u)\nabla u) dx + \int_\Omega \left( 1 - \frac{\omega^*_t}{\omega_1} \right) \nabla \cdot (D(w_1)\nabla w_1) dx + \frac{\delta_i}{p_i} \int_\Omega \left( 1 - \frac{v^*_t}{v_1} \right) \nabla \cdot (D(v_1)\nabla v_1) dx \right. $n \left. + \sum_{k=2}^n \int_\Omega \nabla \cdot (D(w_k)\nabla w_k) dx + \sum_{k=2}^n \frac{\delta_k}{p_k} \int_\Omega \nabla \cdot (D(v_k)\nabla v_k) dx \right. $n \left. = \int_\Omega \left\{ du^*_t \left( 1 - \frac{u}{u^*_t} \right) \left( 1 - \frac{g_1(u^*_t, v^*_t)}{g_1(u, v^*_t)} \right) + g_1(u^*_t, v^*_t) \left[ H\left( \frac{g_1(u(x, t), v_1(x, t))}{g_1(u^*_t, v^*_t)} \right) \right. $n \left. - \frac{\omega_1(x, t)}{\omega^*_t} g_1(u(x, t), v_1(x, t)) \right) - \left. \frac{\omega_1(x, t)}{\omega^*_t} g_1(u^*_t, v^*_t) \omega(x, t) \right) - \left. \frac{\omega_1(x, t)}{\omega^*_t} g_1(u^*_t, v^*_t) \right] \right. $n \left. \right\} dx. \right.$
From the hypothesis \((H_1)\), we can know the function \(g_1(u, v^*_1)\) is strictly increasing in \(u\). Hence, we have
\[
\left(1 - \frac{u}{u^*_1}\right)\left(1 - \frac{g_1(u, v^*_1)}{g_1(u, v_1)}\right) \leq 0,
\] with equality only if \(u = u^*_1\). Based on the assumption \((H_3)\), we can conclude that \(g_1(u(x, t), v_1(x, t))/g_1(u(x, t), v^*_1)\) lies between 1 and \((v_1(x, t)/v^*_1)\). Thus, by the fact that \(H(z)\) is a monotone on each side of 1, we can obtain that
\[
H(g_1(u(x, t), v_1(x, t))/g_1(u(x, t), v^*_1)) \leq H(v_1(x, t)/v^*_1).
\] Meanwhile, for \(k = 2, \ldots, n\), we have
\[
g_1(u^*_1, v^*_1)g_k(u(x, t), v_k(x, t)) - \frac{c_k \delta_k g_k(x, t)}{p_k} v_k(x, t) = \frac{c_k \delta_k g_k(u^*_1, v^*_1)}{p_k g_k(u, v_1)} v_k(g_k(u, v_k) - g_1(u, v^*_1)) v_k(\lambda/0) / \partial v_k - \frac{g_1(u, v^*_1)}{v^*_1} \frac{\partial g_1(u, v^*_1)}{\partial v_1} \mathcal{R}_1 = \frac{c_k \delta_k g_k(u^*_1, v^*_1)}{p_k g_k(u, v_1)} v_k(\alpha_k \mathcal{R}_k - \alpha_i \mathcal{R}_i),
\] where \(\alpha_k = (g_k(u, v_k)/v_k(\partial g_k(\lambda/0))/\partial v_k)\) and \(\alpha_i = (g_1(u, v^*_1)/v^*_1(\partial g_1(\lambda/0))/\partial v_1)\).

Under the condition \((H_1)\) and \((H_4)\), we have
\[
\frac{\alpha_1}{\alpha_k} = \frac{g_1(u, v^*_1) / v^*_1 / \partial g_1(\lambda/0))/\partial v_1}{g_k(u, v_k) / v_k / \partial g_k(\lambda/0))/\partial v_k} \geq \frac{\partial g_1(u, v^*_1)/\partial v_1 / \partial g_k(u, v_k) / \partial v_k}{\eta_k(\lambda/d)} \geq \frac{p_k}{c_k \delta_k} \frac{c_k \delta_k g_k(u^*_1, v^*_1)}{p_k g_k(u, v_1)} v_k = \mathcal{R}_k / \mathcal{R}_1,
\] which yields that
\[
\sum_{k=1}^{n} \int_{\Omega} \left[ \frac{g_1(u^*_1, v^*_1)}{g_1(u, v^*_1)} g_k(u(x, t), v_k(x, t)) - \frac{c_k \delta_k v_k(x, t)}{p_k} \right] dx \leq 0.
\] Therefore, we obtain
\[
\frac{dV(t)}{dt} \leq - g_1(u^*_1, v^*_1) \int_{\Omega} \left[ H(g_1(u^*_1, v^*_1)) + H(w_1(x, t)v^*_1/v_1(x, t))w_1(x, t) + H(g_1(u^*_1, v^*_1)w_1(x, t)) \right] dx
\]}

\[
+ \int_{\Omega} \left(1 - \frac{g_1(u^*_1, v^*_1)}{g_1(u, v^*_1)}\right) \nabla \cdot (D(u)\nabla u) dx + \int_{\Omega} \left(1 - \frac{w^*_1}{w_1}\right) \nabla \cdot (D(w_1)\nabla w) dx
\]

\[
+ \frac{\delta_1}{\nu_1} \int_{\Omega} \left(1 - \frac{v^*_1}{v_1}\right) \nabla \cdot (D(v_1)\nabla v_1) dx + \sum_{k=2}^{n} \int_{\Omega} \nabla \cdot (D(w_k)\nabla w_k) dx + \sum_{k=2}^{n} \frac{\delta_k}{\nu_k} \int_{\Omega} \nabla \cdot (D(v_k)\nabla v_k) dx.
\] It follows from Lemma 2 and Remark 1 that
\[
\frac{dV(t)}{dt} \leq 0.
\] Hence, \(E_1\) is stable, and \((dV(t))/dt = 0\) if \(u = u^*_1\), \(w = w^*_k, v = v^*_1\), and \(w_k = 0 = v_k\) for \(k = 2, \ldots, n\). It is easy to confirm that the singleton \([E_1]\) is just the largest compact invariant set in \((u, w, v) \cap ((dV(t))/dt = 0)\). When \(\mathcal{R}_i > 1\) and \(\mathcal{R}_k > \mathcal{R}_k\) hold for \(k = 2, \ldots, n\), we can know that the infection steady state \(E_i\) of model (7) is globally asymptotically stable by the LaSalle’s invariance principle. The proof of Theorem 6 is complete. □

**Theorem 7.** Assume \(\mathcal{R}_i < 1\) for \(i = 1, \ldots, n\). Then, the steady state \(E_i^*\) of model (7) is globally asymptotically stable.
Proof. Define
\[ V(t) = \int_{\Omega} \sum_{i=1}^{n} \left[ \omega_i(x,t) + \frac{\delta_i}{\rho_i} \nu_i(x,t) \right] \, dx, \]
which is called the Lyapunov functional. The rest of the proof is similar to Theorem 4. So, we omit it. This completes the proof of Theorem 7.

\[ \square \]

Remark 2. From Theorem 7, when the basic reproductive number of each strain is less than 1, we can see that all viral strains die out.

\[ \begin{aligned}
\frac{\partial u(x,t)}{\partial t} &= \nabla \cdot (D_u(u)\nabla u) + \lambda - du(x,t) - \frac{\beta u(x,t)v(x,t)}{1 + au(x,t) + bv(x,t) + abu(x,t)v(x,t)}, \\
\frac{\partial w(x,t)}{\partial t} &= \nabla \cdot (D_w(w)\nabla w) + \frac{\beta u(x,t)v(x,t)}{1 + au(x,t) + bv(x,t) + abu(x,t)v(x,t)} - \delta w(x,t), \\
\frac{\partial v(x,t)}{\partial t} &= \nabla \cdot (D_v(v)\nabla v) + pw(x,t) - cv(x,t),
\end{aligned} \]

with Neumann boundary conditions (2) and initial conditions (3). From (10), together with direct computation, we get the basic reproduction number \( R_0 = (p\beta \delta (d + a\lambda)) \)

\[ \begin{aligned}
\text{is a unique positive solution of the following quadratic equation:} \\
pabd + (p\beta + pbd - pabd - ac\delta)u - pbd - c\delta = 0. 
\end{aligned} \]

\[ (61) \]

It is not hard to verify that if \( R_0 > 1 \), then \( \lambda - du^* > 0 \). So, \( E^* = (u^*, w^*, v^*) \) if \( R_0 > 1 \) is a unique interior equilibrium of system (59).

For simplicity of the numerical illustration, we consider only one spatial dimension with spatial domain \( x \in \Omega = [0, 6] \). The numerical simulations are observed for a time duration of 500 or 1000 days. Here, we have to point out that some parameter values are taken from the literature [24]. Then, we set choose the parameter values as \( m_1 = m_2 = m_3 = 2, \lambda = 10, d = 0.09, \beta = 0.0005, \delta = 0.4, c = 2.4, p = 300, a = 0.5, \) and \( b = 0.4 \). Through direct computation, we obtain \( R_0 = 0.3070 < 1 \) and \( E_0 = (111.1111, 0, 0) \). From Theorem 4, we can know that infection-free equilibrium \( E_0 \) of model (59) is globally asymptotically stable, see Figure 1. Further, we choose the parameter values as \( m_1 = m_2 = m_3 = 2, \lambda = 10, d = 0.09, \beta = 0.0025, \delta = 0.4, c = 2.4, p = 300, a = 0.0015, \) and \( b = 0.4 \). Through direct computation, we obtain \( R_0 = 74.4048 > 1 \) and \( E^* = (104.9054, 1.3963, 174.5354) \). From Theorem 5, we can know that the infection equilibrium \( E^* \) of model (59) is globally asymptotically stable, see Figure 2.

Secondly, we perform the numerical simulation for competitive exclusion. Taking the incidence function as \( g_i(u(x,t), v(x,t)) = (\beta_i u(x,t) v_i(x,t)) / (1 + a_i u(x,t) + b_i v_i(x,t) + a_i b_i u(x,t) v_i(x,t)) \) and \( n = 3 \), we can change model (7) into the following form:

4. Numerical Calculations and Simulations

In this section, we perform some numerical simulations that illustrate and supplement the analytic results given in the previous sections. In what follows, we perform the numerical simulations from the two aspects of global threshold dynamics and competitive exclusion, respectively.

Firstly, we carry out the numerical simulation for global threshold dynamics. Taking the incidence function as \( g(u(x,t), v(x,t)) = (\beta u(x,t)v(x,t)) / (1 + au(x,t) + bv(x,t) + abu(x,t)v(x,t)) \), we can change model (1) into the following form:

\[ (60) \]

\[ \frac{du}{dt} = pabd + ac\delta - p\beta - pbd + \sqrt{(pabd + ac\delta - p\beta - pbd) + 2abcd} \]

\[ = \frac{pabd + ac\delta - p\beta - pbd + \sqrt{(pabd + ac\delta - p\beta - pbd)^2 + 4abcd (pabd + ac\delta)}}{2pabd}. \]
Taking $m_1 = m_2 = m_3 = 2$, $\lambda = 10$, $d = 0.09$, $\beta = 0.0025$, $\delta = 0.4$, $c = 2.4$, $p = 300$, $a = 0.0015$, and $b = 0.4$, we have $R_0 = 74.4048 > 1$ and the infection equilibrium $E^* = (104.9054, 1.3963, 174.5354)$ of model (59) is globally asymptotically stable.

With Neumann boundary conditions (40) and initial conditions (41). By $R_i = (p_i/c_i)\partial g_i(\lambda/d, 0)/\partial v_i$, together with direct computation, we can get the basic reproduction number for viral strain $i$ for model (62) as $R_i = (p_i/\beta_i)/(c_i/(\lambda + a_i \lambda))$. Meanwhile, we can obtain the equilibria $E^* = (\lambda/d, 0, 0)$, where $0$ is a 3-dimensional vector of zeros, and $E_i = (u_i^*, 0, w_i^*, 0, v_i^*, 0)$ for each strain $i (i = 1, 2, 3)$, where $u_i^* = ((\lambda - du^*)/\delta_i)$, $v_i^* = ((p_i/\beta_i) - du^*)/c_i \delta_i$, and

$$w_i^* = \frac{\lambda p_i a_i b_i + a_i c_i \delta_i - p_i \beta_i - d p_i b_i}{2 d p_i a_i b_i} + \sqrt{\left(\frac{\lambda p_i a_i b_i + a_i c_i \delta_i - p_i \beta_i - d p_i b_i}{2 d p_i a_i b_i}\right)^2 + 4 d p_i a_i b_i (p_i b_i + c_i \delta_i).}$$

Choosing the parameter values as $m_1 = m_2 = m_3 = 2$, $\lambda = 10$, and $d = 0.09$ and $\beta_1 = 0.0025$, $\delta_1 = 0.4$, $c_1 = 2.4$, $p_1 = 300$, $a_1 = 0.0015$, and $b_1 = 0.4$ for strain 1; $\beta_2 = 0.0015$, $\delta_2 = 0.6$, $c_2 = 2.4$, $p_2 = 250$, $a_2 = 0.5$, and $b_2 = 0.2$ for strain 2; $\beta_3 = 0.0002$, $\delta_3 = 0.2$, $c_3 = 3.2$, $p_3 = 320$, $a_3 = 0.8$, and $b_3 = 0.6$ for strain 3, we can obtain $R_1 = 74.4048 > 1$. 

![Figure 2: Taking $m_1 = m_2 = m_3 = 2$, $\lambda = 10$, $d = 0.09$, $\beta = 0.0025$, $\delta = 0.4$, $c = 2.4$, $p = 300$, $a = 0.0015$, and $b = 0.4$, we have $R_0 = 74.4048 > 1$ and the infection equilibrium $E^* = (104.9054, 1.3963, 174.5354)$ of model (59) is globally asymptotically stable.](image)
Figure 3: Taking $m_1 = m_2 = m_3 = 2$, $\lambda = 10$, and $d = 0.09$ and $\beta_1 = 0.0025$, $\delta_1 = 0.4$, $c_1 = 2.4$, $p_1 = 300$, $a_1 = 0.0015$, and $b_1 = 0.4$ for strain 1; $\beta_2 = 0.0015$, $\delta_2 = 0.6$, $c_2 = 2.4$, $p_2 = 250$, $a_2 = 0.5$, and $b_2 = 0.2$ for strain 2; $\beta_3 = 0.0002$, $\delta_3 = 0.2$, $c_3 = 3.2$, $p_3 = 320$, $a_3 = 0.8$, and $b_3 = 0.6$ for strain 3, we have $\mathcal{R}_0 = 74.4048 < 1$, $\mathcal{R}_2 = 0.5147 < 1$, $\mathcal{R}_3 = 0.1357 < 1$, and the infection equilibrium $E^1 = (104.9054, 1.3963, 0, 0, 174.5354, 0, 0)$ of model (62) is globally asymptotically stable.
Figure 4: Taking $m_1 = m_2 = m_3 = 2, \lambda = 10$, and $d = 0.09$ and $\beta_1 = 0.0025, \delta_1 = 0.4, c_1 = 2.4, p_1 = 300, a_1 = 0.0015$, and $b_1 = 0.1$ for strain 1; $\beta_2 = 0.0015, \delta_2 = 0.6, c_2 = 2.4, p_2 = 250, a_2 = 0.25$, and $b_2 = 0.2$ for strain 2; $\beta_3 = 0.0002, \delta_3 = 0.2, c_3 = 3.2, p_3 = 320, a_3 = 0.092$, and $b_3 = 0.6$ for strain 3, we have $R_0 = 74.4048 > 1$, $R_2 = 1.0055 > 1$, $R_3 = 1.0204 > 1$, and the infection equilibrium $E_1 = (89.5396, 4.8536, 0, 0, 606.6994, 0, 0)$ of model (62) is globally asymptotically stable.
Figure 5: Taking $m_1 = m_2 = m_3 = 2$, $\lambda = 10$, and $d = 0.09$ and $\beta_1 = 0.0005$, $\delta_1 = 0.4$, $c_1 = 2.4$, $p_1 = 300$, $a_1 = 0.5$, and $b_1 = 0.4$ for strain 1; $\beta_2 = 0.0015$, $\delta_2 = 0.6$, $c_2 = 2.4$, $p_2 = 250$, $a_2 = 0.5$, and $b_2 = 0.2$ for strain 2; $\beta_3 = 0.0002$, $\delta_3 = 0.2$, $c_3 = 3.2$, $p_3 = 320$, $a_3 = 0.8$, and $b_3 = 0.6$ for strain 3, we have $R_1 = 0.3070 < 1$, $R_2 = 0.5147 < 1$, $R_3 = 0.1357 < 1$, and the infection-free equilibrium $E_0 = (111.1111, 0, 0, 0, 0, 0, 0, 0)$ of model (62) is globally asymptotically stable.
which yields that \((a_1/a_2) \times R_1 = 0.9003 > 0.5147 = R_2\) and \((a_1/a_3) \times R_1 = 0.9003 > 1.357 = R_3\). From Theorem 6, we can know that the infection equilibrium \(E^1\) of model (62) is globally asymptotically stable, which implies that strain one persists, while strains 2 and 3 die out. These facts are numerically confirmed in Figure 3.

Further, we take another parameter value as \(\beta_1 = 0.0025, \delta_1 = 0.4, c_1 = 2.4, p_1 = 300, a_1 = 0.0015, \) and \(b_1 = 0.1\) for strain 1; \(\beta_2 = 0.0015, \delta_2 = 0.6, c_2 = 2.4, p_2 = 250, a_2 = 0.25, \) and \(b_2 = 0.2\) for strain 2; \(\beta_3 = 0.0002, \delta_3 = 0.2, c_3 = 3.2, p_3 = 320, a_3 = 0.092, \) and \(b_3 = 0.6\) for strain 3. By direct computation again, we can also obtain \(R_1 = 74.4048 > 1, R_2 = 1.0055 > 1, R_3 = 1.0204 > 1, E^1 = (89.5396, 4.8536, 0, 0, 606.6994, 0, 0), \) and

\[
\begin{align*}
\alpha_4 &= \frac{1 + a_4 u(x,t) + b_4 y(x,t) + a_4 b_4 u(x,t) y(x,t)}{1 + a_4 u(x,t) + b_4 y(x,t) + a_4 b_4 u(x,t) y(x,t)} \\
&\geq \frac{1}{1 + a_4 (\lambda/d) + b_4 y(x,t) + a_4 b_4 (\lambda/d) y(x,t)} = 0.0139, \quad k = 2, 3,
\end{align*}
\]

which yields that \((a_1/a_2) \times R_1 = 1.0342 > 1.0055 = R_2\) and \((a_1/a_3) \times R_1 = 1.0342 > 1.0204 = R_3\). From Theorem 6, we can know that the infection equilibrium \(E^1\) of model (62) is globally asymptotically stable, which implies that strain one persists, while strains 2 and 3 die out. These facts are numerically confirmed in Figure 4.

Next, we take the parameter values as \(m_1 = m_2 = m_3 = 2, \lambda = 10, \) and \(d = 0.09\) and \(\beta_1 = 0.0005, \delta_1 = 0.4, c_1 = 2.4, p_1 = 300, a_1 = 0.5, \) and \(b_1 = 0.4\) for strain 1; \(\beta_2 = 0.0015, \delta_2 = 0.6, c_2 = 2.4, p_2 = 250, a_2 = 0.5, \) and \(b_2 = 0.2\) for strain 2; \(\beta_3 = 0.0002, \delta_3 = 0.2, c_3 = 3.2, p_3 = 320, a_3 = 0.8, \) and \(b_3 = 0.6\) for strain 3. By direct computation, we can get \(R_1 = 0.3070 < 1, R_2 = 0.5147 < 1, R_3 = 1.357 < 1, \) and \(E^1 = (111.111, 0, 0, 0, 0, 0, 0, 0)\). From Theorem 7, we can know that infection-free equilibrium \(E^1\) of model (62) is globally asymptotically stable, which implies that all viral strains die out. These facts are numerically confirmed in Figure 5.

5. Conclusion

In this paper, we investigate the global threshold dynamics in single-strain viral infection models (1) and competitive exclusion in multistrain viral infection models (7) under the homogeneous Neumann boundary conditions.

For model (1), by constructing a Lyapunov functional, we obtain that the infection-free equilibrium \(E_0\) of model (1) is globally asymptotically stable if the basic reproduction number \(R_0 < 1\), and the infection equilibrium \(E^*\) of model (1) is globally asymptotically stable if the basic reproduction number \(R_0 > 1\). These facts are numerically confirmed in Figures 1 and 2, respectively. For model (7), by constructing a Lyapunov functional, we also obtain that the steady state \(E_1\) of model (7) is globally asymptotically stable if \(R_1 > 1\) and \(R_i > R_k\) for \(k = 2, \ldots, n\), which implies that strain one persists, while strains \(k = 2, \ldots, n\) die out. These facts are numerically confirmed in Figures 3 and 4. Meanwhile, the steady state \(E^*_1\) of model (7) is globally asymptotically stable if \(R_i < 1\) for \(i = 1, \ldots, n\), which implies that all viral strains die out. These facts are numerically confirmed in Figure 5. Here, we have to point out that the incidence function in the present paper is general and performed the numerical simulation for global threshold dynamics and competitive exclusion. So, our results in the present paper are new and can also be applied to other reaction-diffusion systems. Of course, we hope that our work could be instructive to study the dynamics of the viral infection model.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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