MODELLING THE DYNAMICS OF AVIAN INFLUENZA WITH NONLINEAR RECOVERY RATE AND PSYCHOLOGICAL EFFECT*

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Abstract In this paper, a SI-SEIR type avian influenza epidemic model with psychological effect, nonlinear recovery rate and saturation inhibition effect is formulated to study the transmission and control of avian influenza virus. By setting the basic reproductive number as the threshold parameter and constructing Lyapunov function, Dulac function and using the Li-Muldowney's geometry approach, we prove the local and global stability of disease-free equilibria and endemic equilibrium. Theoretical analysis are carried out to show the role of the saturation inhibition effect, psychological effect and effective medical resources in this model, and numerical simulations are also given to verify the results.

Keywords Avian influenza epidemic model, psychological effect, nonlinear recovery rate, nonlinear incidence rate, globally asymptotically stable.

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1. Introduction

Avian influenza refers to the disease which caused by infection with avian (bird) influenza (flu) Type A viruses. These viruses occur naturally among wild aquatic birds worldwide and can infect domestic poultry, other birds and animal species. Avian flu viruses do not normally infect humans. However, sporadic human infections with avian flu viruses have occurred [7].

Avian influenza A viruses are designated as highly pathogenic avian influenza (HPAI) or low pathogenic avian influenza (LPAI). The other three subtypes AH9, AH5 and AH7 can simultaneously infect humans and birds. Viruses (H1-H9) found in poultry and wild birds worldwide belong to the same category (LPAI). Rarely, sporadic cases of human infection with H9N2 leading to mild upper respiratory disease have been reported. Reports from more than 15 countries indicate that the H5N1 viruses can infect humans, most of which directly cause severe pneumonia, with a mortality rate close to 60%. The human infected H7 viruses are not

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common, but it has also been documented that direct contact with infected birds can infect H7 viruses, especially during outbreaks of H7 viruses in poultry. LPAI (H7N2, H7N3 and H7N7) viruses would result in mild-to-moderate diseases, while HPAI(H7N3,H7N7) virus infection can result in mild-to-moderate or even lethal diseases. Human cases of H7N9 virus infection were first detected in 2013, including death and severe respiratory illnesses [8].

In recent years, avian influenza has been rampant, and its mutation rate is very fast, which has a non-negligible impact on human life, health as well as social economy. In view of this, according to the epidemiological characteristics of avian influenza, researchers have established a large number of mathematical models to study, and combined with its dynamical behavior, remarkable achievements have been made in the prevention and control of avian influenza [1, 19, 23]. Iwami et al. [18] proposed an Ordinary Differential Equation (ODE) model in 2007 to describe the dynamics of avian influenza between human and avian populations. Then, many different mathematical models were designed for the H5N1 avian influenza virus [2, 12, 30, 33]. A series of analysis has also been carried out on the epidemiology of influenza A H7N9 influenza in recent years [5, 21, 27, 36]. However, most of them have not considered the latent state of avian influenza virus in human body, and it does exist according to reported cases, so it is very necessary to take it into account in models.

Human behavior and social reaction have great significance to the spread of infectious disease [4, 13–15], since it is a key factor in disease control efforts. Wang et al. [34] found that 70% urban respondents who participated in the survey said that since the first case of H7N9 were detected in China in March 2013, their visit to live poultry market have been relatively reduced. In addition, the research of Wu et al. [35] also suggested that people experience changes in protective behaviors such as reducing access to live poultry markets and buying live poultry in the context of continuous H7N9 outbreaks. Although human behavior and social responses during the transmission of infectious diseases are often reported, few systematic studies have been conducted on their effects. It must be admitted that it is difficult to combine social behavioral responses with human behavior and social responses in mathematical models.

As we know, incidence rate of a disease is one of crucial factors in the transmission of disease. It is worth noting that in almost all models related to avian influenza, the incidence between susceptible birds and infected birds (susceptible humans and infected birds) has taken in form of bilinear interaction, which is constantly increasing unbounded, whereas as survey [37] revealed that if the case was reported by the media, people would go to live poultry market less, incidence of human will decline. In order to better explain the practical significance of infectious disease model, the non-monotonic incidence rate is used here which was proposed by Liu et al. [22] perfectly describes this phenomenon caused by psychological effects(also see [20, 21, 38]); Similarly, with the increasing number of infected birds, poultry farmers will be highly vigilant and take the corresponding protective measures to make the poultry incidence rate reach saturation, which can be described by the saturation incidence function used in [6, 31].

There is one thing in common in the classical avian influenza model, that is, the recovery rate is always assumed to be constant, which is equivalent to the default public medical resources are always sufficient, and it is obviously unreasonable by the following two points. First of all, hospital resources (such as doctors, medicines,

beds) are limited for the public; Secondly, according to the cases reported by the CDC [7], there are some similarities between human infection with avian influenza virus and common influenza virus in terms of infection time and early clinical manifestations, then some of the available hospital resources have already occupied. Proportion between the number of beds and the population, ie., the number of hospital beds available per 10,000 people, is widely used by health planners to estimate public resource availability [17]. Abdelrazec et al. [3] established a dengue propagation dynamics model which considered a recovery rate function restricted by hospital bed-population proportion and the number of infected. They proved that the model has oscillations and backward bifurcation due to the limited resources. At this time, controlling the basic regeneration number $R_0 < 1$ is not enough to ensure the eradication of the disease. Therefore, the recovery rate function which is defined by [32] will be introduced in this paper, further learning the impact of the limited availability of existing medical resources on the spread of avian influenza virus.

The rest of this paper is outlined as follows: Section 2 constructs an avian influenza epidemic model based on the above discussion, and gives a general explanation of parameters; In Section 3, the avian-only sub-model and its detailed mathematical analysis are given; Section 4 presents the whole mathematical analysis of the model in details; Section 5 carries out the numerical simulation to the full avian influenza infectious disease model; Finally, conclusions and discussions are also given in Section 6.

2. Model discription

In this section, we assume that the avian influenza virus does not spread from person to person and mutate. The avian population is classified into two subclasses: susceptible $S_a(t)$ and infected $I_a(t)$, respectively. The human population is classified into four subclasses: susceptible $S_h(t)$, exposed $E_h(t)$, infected $I_h(t)$ and recovered human $R_h(t)$, respectively. Then we have the model (2.1) as follows:

$$\begin{cases} \dfrac{dS_a(t)}{dt} = m_a S_a(1 - \dfrac{S_a}{K_a}) - \dfrac{\beta_a I_a S_a}{1 + \alpha I_a}, \\[2mm] \dfrac{dI_a(t)}{dt} = \dfrac{\beta_a I_a S_a}{1 + \alpha I_a} - (\mu_a + \delta_a)I_a, \\[2mm] \dfrac{dS_h(t)}{dt} = \Lambda_h - \mu_h S_h - \dfrac{\beta_h I_a S_h}{1 + c I_h^2}, \\[2mm] \dfrac{dE_h(t)}{dt} = \dfrac{\beta_h I_a S_h}{1 + c I_h^2} - (\mu_h + \theta_h)E_h, \\[2mm] \dfrac{dI_h(t)}{dt} = \theta_h E_h - (\mu_h + \delta_h)I_h - (\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b + I_h})I_h, \\[2mm] \dfrac{dR_h(t)}{dt} = (\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b + I_h})I_h - \mu_h R_h, \end{cases} \tag{2.1}$$

with the following assumptions:

(1) The net growth rate function of susceptible poultry is subject to Logistic growth $g(S_a) = m_a S_a(1 - \dfrac{S_a}{K_a}) : R_+ \to R$ is continuous, where m_a and K_a are the

intrinsic growth rate and the maximum environmental capacity of poultry, respectively. $R = (-\infty, \infty)$ and $R_+ = (0, \infty)$;

(2) All new population (poultry) are subordinate to susceptible populations (poultry), and new population recruitment rate is represented by Λ_h;

(3) The avian influenza virus is transmitted only between infected poultry and susceptible populations;

(4) Infected poultry can't recover and maintain the disease state until death. Infected population can recover and gain permanent immunity;

(5) The incidence rate between susceptible poultry (or population) and infected poultry is $\dfrac{\beta_a I_a S_a}{1 + \alpha I_a}$ $\left(or \ \dfrac{\beta_h I_a S_h}{1 + c I_h^2} \right)$;

(6) The recovery rate is $u(b, I_h) = \mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b + I_h}$, where μ_1 is the highest per capita recovery rate, μ_0 is the minimum per capita recovery rate due to lack of clinical resources, b is the ratio between the number of hospital bed and the population.

Thus, we obtain the above model, where β_a is the transmission rate from infective avian to susceptible avian, μ_a is the natural death rate of the avian population, δ_a is the disease-related death rate of the infected avian, β_h is the transmission rate from the infective avian to the susceptible human, μ_h is the natural death rate of the human population, δ_h is the disease-related death rate of the infected human. The parameters α and c are constants measuring the inhibition effect and psychological effect, respectively.

We consider system (2.1) with initial conditions

$$S_a(0) \geq 0, I_a(0) \geq 0, S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0. \qquad (2.2)$$

The parameters of system (2.1) are shown in Table 1.

Table 1. List of parameters

parameter	implication	measured value	reference
m_a	Intrinsic growth rate of poultry	$5 * 10^{-3}$	[23]
K_a	Maximum environmental capacity of poultry	$5 * 10^4$	[23]
β_a	The rate of disease transmission between infected and susceptible birds	⌣	⌣
α	Inhibitory effect coefficient	⌣	⌣
μ_a	Natural mortality in poultry	$(1/5 - 1/10)(year^{-1})$	[26]
δ_a	Poultry disease-related mortality	$4 * 10^{-4}$	[23]
Λ_h	Human recruitment and birth rates	30	[23]
β_h	The rate of disease transmission between infected poultry and susceptible human	$5 * 10^{-9}$	⌣
μ_h	The natural mortality of population	$1/70(year^{-1})$	⌣
δ_h	Human disease-related mortality	0.077	[36]
θ_h	The covert rate of disease from latent to infected state	$1/7(day^{-1})$	CDC [7]
μ_0	The lowest rate of recovery in humans	$(0.067 \sim 0.100)$	[36]
μ_1	The highest recovery rate in humans	$(\mu_0 \sim 10)$	[32]
b	Hospital beds-population ratio	$(0, 20)$	[32]
c	Psychological effect coefficient	⌣	⌣

Theorem 2.1. *The set* $D = \{(S_a, I_a, S_h, E_h, I_h, R_h) \in R_+^6 \ | S_a + I_a \leq \dfrac{\phi}{\mu_a}, S_h + E_h + I_h + R_h \leq \dfrac{\Lambda_h}{\mu_h}\}$ *is a positively invariant and attracting region of system* (2.1), *where* $\phi = \dfrac{K_a(\mu_a + m_a)^2}{4m_a}$.

Proof. Setting $N_a = S_a + I_a$ and adding the first two equations of system (2.1),

we have

$$
\begin{aligned}
\frac{dN_a}{dt} &= m_a S_a (1 - \frac{S_a}{K_a}) - (\mu_a + \delta_a) I_a \\
&= m_a S_a (1 - \frac{S_a}{K_a}) - \mu_a (S_a + I_a) + \mu_a S_a - \delta_a I_a \\
&\leq m_a S_a (1 - \frac{S_a}{K_a}) - \mu_a N_a + \mu_a S_a \\
&\leq \frac{K_a(\mu_a + m_a)^2}{4m_a} - \mu_a N_a.
\end{aligned}
$$

Then, we obtain

$$
0 \leq N_a(t) \leq \frac{\phi}{\mu_a}(1 - e^{-\mu_a t}) + N_a(0)e^{-\mu_a t},
$$

where $\phi = \dfrac{K_a(\mu_a + m_a)^2}{4m_a}$. Taking $t \to +\infty$, we can get $N_a(t) \to \dfrac{\phi}{\mu_a}$.

Similarly, setting $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$, we have

$$
\frac{dN_h(t)}{dt} = \Lambda_h - \mu_h N_h(t) - \delta_h I_h(t) \leq \Lambda_h - \mu_h N_h(t),
$$

then it follows

$$
0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}) + N_h(0)e^{-\mu_h t}.
$$

It is clear that $N_h \to \dfrac{\Lambda_h}{\mu_h}$ as $t \to +\infty$. Moreover, if $N_a > \dfrac{\phi}{\mu_a}$, $N_h > \dfrac{\Lambda_h}{\mu_h}$, then

$$
\frac{dN_a}{dt} \leq \phi - \mu_a N_a < 0,
$$

and

$$
\frac{dN_h}{dt} \leq \Lambda - \mu_h N_h < 0.
$$

Therefore, D is positively invariant. $\qquad\square$

Next, we will calculate the basic reproduction number of system (2.1).

System (2.1) has two disease-free equilibria $A(0,0,\frac{\Lambda_h}{\mu_h},0,0,0)$ and $B(K_a,0,\frac{\Lambda_h}{\mu_h},0,0,0)$. Using the method of the next generation matrix in [10, 11], system (2.1) can be rewritten as

$$
\frac{dX}{dt} = M - N,
$$

where

$$
X = \begin{pmatrix} I_a(t) \\ E_h(t) \\ I_h(t) \\ S_a(t) \\ S_h(t) \\ R_h(t) \end{pmatrix}, \quad
M = \begin{pmatrix} \frac{\beta_a I_a S_a}{1 + \alpha I_a} \\ \frac{\beta_h I_a S_h}{1 + c I_h^2} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad
N = \begin{pmatrix} (\mu_a + \delta_a) I_a \\ (\mu_h + \theta_h) E_h \\ -\theta_h E_h + (\mu_h + \delta_h) I_h + [\mu_0 + b\frac{(\mu_1 - \mu_0)}{b + I_h}] I_h \\ -m_a S_a(1 - \frac{S_a}{K_a}) + \frac{\beta_a I_a S_a}{1 + \alpha I_a} \\ -\Lambda_h + \mu_h S_h + \frac{\beta_h I_a S_h}{1 + c I_h^2} \\ -[\mu_0 + b\frac{(\mu_1 - \mu_0)}{b + I_h}] I_h + \mu_h R_h \end{pmatrix}.
$$

Let

$$M_1 = (\frac{\beta_a I_a S_a}{1 + \alpha I_a}, \frac{\beta_h I_a S_h}{1 + cI_h^2}, 0)^T,$$

$$N_1 = ((\mu_a + \delta_a)I_a, (\mu_h + \theta_h)E_h, -\theta_h E_h + (\mu_h + \delta_h)I_h + \mu(b, I_h)I_h)^T.$$

Then, we have

$$F = \begin{pmatrix} \beta_a K_a & 0 & 0 \\ \frac{\beta_h \Lambda_h}{\mu_h} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \; V = \begin{pmatrix} \mu_a + \delta_a & 0 & 0 \\ 0 & \mu_h + \theta_h & 0 \\ 0 & -\theta_h & \mu_h + \delta_h + \mu_1 \end{pmatrix}, \; FV^{-1} = \begin{pmatrix} \frac{\beta_a K_a}{\mu_a + \delta_a} & 0 & 0 \\ \frac{\beta_a \Lambda_h}{\mu_h(\mu_a + \delta_a)} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Then the basic reproduction number of system (2.1) is

$$R_0 = \rho(FV^{-1}) = \frac{\beta_a K_a}{\mu_a + \delta_a}.$$

3.   Analysis of Avian-only sub-model

We first discuss the dynamic behavior of the following avian sub-model:

$$\begin{cases} \frac{dS_a(t)}{dt} = m_a S_a(1 - \frac{S_a}{K_a}) - \frac{\beta_a I_a S_a}{1 + \alpha I_a}, \\ \frac{dI_a(t)}{dt} = \frac{\beta_a I_a S_a}{1 + \alpha I_a} - (\mu_a + \delta_a)I_a. \end{cases} \tag{3.1}$$

Clearly, $D_1 = \{(S_a, I_a) \in R_+^2 \mid S_a + I_a \leq \frac{\phi}{\mu_a}\}$ is positively invariant.

3.1. Existence of equilibria in system (3.1)

Obviously, $A_a(0,0)$ and $B_a(K_a, 0)$ are two disease-free equilibria of system (3.1). Now we consider the existence of endemic equilibrium $C_a(S_a^*, I_a^*)$. Then S_a^* and I_a^* satisfy

$$\begin{cases} m_a S_a(1 - \frac{S_a}{K_a}) - \frac{\beta_a I_a S_a}{1 + \alpha I_a} = 0, \\ \frac{\beta_a I_a S_a}{1 + \alpha I_a} - (\mu_a + \delta_a)I_a = 0. \end{cases} \tag{3.2}$$

From (3.2), we can derive

$$S_a^* = \frac{(\mu_a + \delta_a)(1 + \alpha I_a^*)}{\beta_a}.$$

Substituting S_a^* into (3.2), we obtain

$$A(I_a^*)^2 + BI_a^* + C = 0, \tag{3.3}$$

where

$$A = \alpha^2 m_a(\mu_a + \delta_a) > 0,$$
$$B = K_a \beta_a^2 + \alpha m_a(\mu_a + \delta_a) + \alpha m_a(\mu_a + \delta_a)(1 - R_0),$$
$$C = m_a(\mu_a + \delta_a)(1 - R_0).$$

(1) If $R_0 = 1$, then $C = 0$ and $B > 0$. Equation (3.3) is reduced to $A(I_a^*)^2 + BI_a^* = 0$. So, we have $I_a^* = 0$ or $I_a^* = \frac{-B}{A} < 0$, i.e., equation (3.3) has no positive root.

(2) If $R_0 > 1$, then $C < 0$ and $\Delta = B^2 - 4AC > 0$. Thus, equation (3.3) has two distinct real roots. According to the Vieta theorem, we have

$$I_a^* = \frac{-B + \sqrt{\Delta}}{2A}.$$

(3) If $R_0 < 1$, then $A > 0, B > 0$ and $C > 0$. It follows from Descartes' rule of signs that equation (3.3) does not have a positive root.

From the above discussions, we know that system (3.1) exists a unique endemic equilibrium $C_a(S_a^*, I_a^*)$ when $R_0 > 1$.

3.2. Local stability of equilibria in system (3.1)

Theorem 3.1. *The disease-free equilibrium $A_a(0,0)$ of system (3.1) is always unstable; if $R_0 \leq 1$, the disease-free equilibrium $B_a(K_a, 0)$ of system (3.1) is locally asymptotically stable; if $R_0 > 1$, then $B_a(K_a, 0)$ is unstable, and the endemic equilibrium $C_a(S_a^*, I_a^*)$ of system (3.1) is locally asymptotically stable.*

Proof. The Jacobian matrix of system (3.1) is

$$J_a = \begin{pmatrix} m_a - \frac{2m_a S_a}{K_a} - \frac{\beta_a I_a}{1 + \alpha I_a} & -\frac{\beta_a S_a}{(1 + \alpha I_a)^2} \\ \frac{\beta_a I_a}{1 + \alpha I_a} & \frac{\beta_a S_a}{(1 + \alpha I_a)^2} - (\mu_a + \delta_a) \end{pmatrix}.$$

Then the corresponding characteristic equation is

$$(\lambda - m_a + \frac{2m_a S_a}{K_a} + \frac{\beta_a I_a}{(1 + \alpha I_a)})(\lambda - \frac{\beta_a S_a}{(1 + \alpha I_a)^2} + \mu_a + \delta_a) + \frac{\beta_a^2 S_a I_a}{(1 + \alpha I_a)^3} = 0. \quad (3.4)$$

(1) If $(S_a, I_a) = (0, 0)$, then equation (3.4) becomes

$$(\lambda - m_a)(\lambda + \mu_a + \delta_a) = 0.$$

Clearly, $\lambda_1 = m_a > 0$ and $\lambda_2 = -\mu_a - \delta_a < 0$. Hence, the disease-free equilibrium A_a is unstable.

(2) If $(S_a, I_a) = (K_a, 0)$, then we get

$$(\lambda + m_a)(\lambda + (\mu_a + \delta_a)(1 - R_0)) = 0.$$

So, $\lambda_1 = -m_a < 0$ and $\lambda_2 = (\mu_a + \delta_a)(R_0 - 1)$.

(2a) If $R_0 < 1$, then $B_a(K_a, 0)$ is locally asymptotically stable. Obviously, B_a is unstable when $R_0 > 1$.

(2b) If $R_0 = 1$, then $B_a(K_a, 0)$ is a saddle node bifurcation point which is locally asymptotically stable.

Actually, let $S_a = S_1 + K_a$ and $I_a = I_1$. System (3.1) is transformed into

$$\begin{cases} \frac{dS_1}{dt} = \frac{-(K_a + S_1)[m_a S_1(1 + \alpha I_1) + K_a \beta_a I_1]}{K_a(1 + \alpha I_1)}, \\ \frac{dI_1}{dt} = \frac{\beta_a I_1(K_a + S_1) - I_1(1 + \alpha I_1)(\mu_a + \delta_a)}{1 + \alpha I_1}. \end{cases}$$

Using $S_1 = x - \frac{k\beta y}{m_a}$, $I_1 = y$, $t = -\frac{\tau}{m_a}$, the above system can be rewritten as

$$\begin{cases} \frac{dx}{d\tau} = x + g_1(x, y), \\ \frac{dy}{d\tau} = g_2(x, y), \end{cases}$$

where

$$g_1(x, y) = \frac{1}{m_a^3(\alpha y K_a + K_a)}[y\beta_a K_a^2 m_a(\delta_a - \beta_a K_a + \mu_a - x\beta_a + \alpha y(\mu_a + \delta_a + y\beta_a))$$
$$- y\beta_a K_a m_a^2(\alpha y(K_a + 2x) + x) + y^2\beta_a^3 K_a^3 + x^2 m_a^3(\alpha y + 1)],$$
$$g_2(x, y) = \frac{y[\beta_a K_a(y\beta_a - m_a) + m_a((\alpha y + 1)(\mu_a + \delta_a) - x\beta_a)]}{(\alpha y + 1)m_a}.$$

Suppose $x = \phi(y)$ is the solution of $x + g_1(x, y) = 0$. Substituting x into $g_2(x, y)$ and expanding its Taylor series, we get

$$q_2(y) \triangleq \frac{-K_a\beta(\alpha m_a + \beta_a)}{m_a}y^2 + \frac{-2K_a\beta_a[-m_a^3\alpha^2 - 2m_a^2\alpha\beta + K_a\beta_a^3 + m_a\alpha\beta_a(\mu_a + \delta_a)]}{m_a^3}y^3 + o(y^3).$$

According to [29, p147–p152], we can get that $B_a(K_a, 0)$ is a saddle-node point when $R_0 = 1$. Further, it is locally asymptotically stable.
(3) If $R_0 > 1$ and $(S_a, I_a) = (S_a^*, I_a^*)$, then equation (3.4) becomes

$$\lambda^2 + p\lambda + q = 0,$$

where

$$p = \frac{2m_a S_a^*}{K_a} + \frac{\beta_a I_a^*}{1 + \alpha I_a^*} - m_a - \frac{\beta_a S_a^*}{(1 + \alpha I_a^*)^2} + \mu_a + \delta_a,$$

$$q = (\frac{2m_a S_a^*}{K_a} + \frac{\beta_a I_a^*}{1 + \alpha I_a^*} - m_a)(\mu_a + \delta_a - \frac{\beta_a S_a^*}{(1 + \alpha I_a^*)^2}) + \frac{\beta_a^2 S_a^* I_a^*}{(1 + \alpha I_a^*)^3}.$$

The first equation of (3.2) can be deduced to

$$m_a = \frac{\beta_a I_a^*}{1 + \alpha I_a^*} + \frac{m_a S_a^*}{K_a}.$$

Since

$$S_a^* = \frac{(\mu_a + \delta_a)(1 + \alpha I_a^*)}{\beta_a},$$

we have

$$p = \frac{2m_a S_a^*}{K_a} + \frac{\beta_a I_a^*}{1 + \alpha I_a^*} - m_a - \frac{\beta_a S_a^*}{(1 + \alpha I_a^*)^2} + \mu_a + \delta_a$$
$$= \frac{m_a S_a^*}{K_a} - \frac{\mu_a + \delta_a}{1 + \alpha I_a^*} + \mu_a + \delta_a > 0,$$
$$q = (\frac{2m_a S_a^*}{K_a} + \frac{\beta_a I_a^*}{1 + \alpha I_a^*} - m_a)(\mu_a + \delta_a - \frac{\beta_a S_a^*}{(1 + \alpha I_a^*)^2}) + \frac{\beta_a^2 S_a^* I_a^*}{(1 + \alpha I_a^*)^3}$$
$$= \frac{2m_a S_a^*(\mu_a + \delta_a)}{K_a} - \frac{2m_a\beta_a(S_a^*)^2}{K_a(1 + \alpha I_a^*)^2} + \frac{\beta_a I_a^*(\mu_a + \delta_a)}{1 + \alpha I_a^*} - m_a(\mu_a + \delta_a) + \frac{m_a\beta_a S_a^*}{(1 + \alpha I_a^*)^2}$$

$$= \frac{2m_a S_a^*(\mu_a+\delta_a)}{K_a} - \frac{2m_a S_a^*(\mu_a+\delta_a)}{K_a(1+\alpha I_a^*)} + \frac{\beta_a I_a^*(\mu_a+\delta_a)}{1+\alpha I_a^*} - m_a(\mu_a+\delta_a) + \frac{m_a(\mu_a+\delta_a)}{1+\alpha I_a^*}$$

$$= \frac{1}{K_a(1+\alpha I_a^*)}[2\alpha m_a S_a^* I_a^*(\mu_a+\delta_a) + \beta_a K_a I_a^*(\mu_a+\delta_a) - \alpha K_a m_a(\mu_a+\delta_a) I_a^*]$$

$$= \frac{(\mu_a+\delta_a) I_a^*}{K_a(1+\alpha I_a^*)}(2\alpha m_a S_a^* + \beta_a K_a - \alpha K_a m_a)$$

$$= \frac{(\mu_a+\delta_a) I_a^*}{K_a(1+\alpha I_a^*)}(2\alpha m_a S_a^* + \beta_a K_a - \frac{\alpha K_a \beta_a I_a^*}{1+\alpha I_a^*} - \alpha m_a S_a^*)$$

$$= \frac{(\mu_a+\delta_a) I_a^*}{K_a(1+\alpha I_a^*)}[\alpha m_a S_a^* + \beta_a K_a(1 - \frac{\alpha I_a^*}{1+\alpha I_a^*})] > 0.$$

Then we conclude that all eigenvalues have strictly negative real parts. Therefore, the endemic equilibrium C_a is locally asymptotically stable when $R_0 > 1$. □

3.3. Global stability of equilibria in system (3.1)

Theorem 3.2. *If $R_0 \leq 1$, then the disease-free equilibrium $B_a(K_a, 0)$ of system* (3.1) *is globally asymptotically stable.*

Proof. Define
$$V = f(S_a) + I_a,$$
where $f(S_a) = K_a(\frac{S_a}{K_a} - 1 - \ln\frac{S_a}{K_a})$.

Obviously, $f(S_a) \geq 0$ and $f(S_a) = 0$ if and only if $S_a = K_a$. Then we get

$$\frac{dV}{dt}|_{(3)} = S_a'(t)(1 - \frac{K_a}{S_a}) + I_a'(t)$$

$$= [m_a S_a(1 - \frac{S_a}{K_a}) - \frac{\beta_a I_a S_a}{1+\alpha I_a}](1 - \frac{K_a}{S_a}) + \frac{\beta_a I_a S_a}{1+\alpha I_a} - (\mu_a+\delta_a) I_a$$

$$= [m_a(1 - \frac{S_a}{K_a}) - \frac{\beta_a I_a S_a}{1+\alpha I_a}](S_a - K_a) + \frac{\beta_a I_a S_a}{1+\alpha I_a} - (\mu_a+\delta_a) I_a$$

$$= -\frac{m_a}{K_a}(S_a - K_a)^2 - \frac{\beta_a I_a}{1+\alpha I_a}(S_a - K_a) + \frac{\beta_a I_a S_a}{1+\alpha I_a} - (\mu_a+\delta_a) I_a$$

$$= -\frac{m_a}{K_a}(S_a - K_a)^2 + \frac{\beta_a K_a I_a}{1+\alpha I_a} - (\mu_a+\delta_a) I_a$$

$$= -\frac{m_a}{K_a}(S_a - K_a)^2 + (\mu_a+\delta_a)(\frac{R_0}{1+\alpha I_a} - 1) I_a.$$

So, $\frac{dV}{dt}|_{(3)} \leq 0$ if $R_0 \leq 1$. Moreover, $\frac{dV}{dt}|_{(3)} = 0$ if and only if $S_a = K_a, I_a = 0$. We can also get $\{(S_a, I_a) \in \text{int}D_1)|\frac{dV}{dt}|_{(3)} = 0\} = \{(S_a, I_a)|S_a = K_a, I_a = 0\} = \{B_a\}$. According to LaSalle's invariant principle, the disease-free equilibrium $B_a = (K_a, 0)$ is globally asymptotically stable if $R_0 \leq 1$. □

Theorem 3.3. *If $R_0 > 1$, then the endemic equilibrium $C_a(S_a^*, I_a^*)$ of system* (3.1) *is globally asymptotically stable.*

Proof. Define the Dulac function $B = \frac{1}{S_a I_a}$. By simple calculation, we obtain

$$BP = \frac{m_a}{I_a}(1 - \frac{S_a}{K_a}) - \frac{\beta_a}{1+\alpha I_a}, \quad BQ = \frac{\beta_a}{1+\alpha I_a} - \frac{\mu_a+\delta_a}{S_a},$$

$$\frac{\partial(BP)}{\partial S_a} = -\frac{m_a}{K_a I_a}, \qquad \frac{\partial(BQ)}{\partial I_a} = -\frac{\alpha\beta_a}{(1+\alpha I_a)^2}.$$

Thus

$$\frac{\partial(BP)}{\partial S_a} + \frac{\partial(BQ)}{\partial I_a} = -\frac{m_a}{K_a I_a} - \frac{\alpha\beta_a}{(1+\alpha I_a)^2} < 0.$$

By using Bendixon Dulac criterion, we know that system (3.1) can't have a closed orbit in D_1, namely, the endemic equilibrium $C_a(S_a^*, I_a^*)$ is globally asymptotically stable. $\qquad\square$

4. Analysis of full influenza epidemic model

Since the first five equations of system (2.1) are independent of R_h, we only need to consider the following system:

$$\begin{cases} \dfrac{dS_a(t)}{dt} = m_a S_a(1 - \dfrac{S_a}{K_a}) - \dfrac{\beta_a I_a S_a}{1+\alpha I_a}, \\[2mm] \dfrac{dI_a(t)}{dt} = \dfrac{\beta_a I_a S_a}{1+\alpha I_a} - (\mu_a + \delta_a)I_a, \\[2mm] \dfrac{dS_h(t)}{dt} = \Lambda_h - \mu_h S_h - \dfrac{\beta_h I_a S_h}{1+cI_h^2}, \\[2mm] \dfrac{dE_h(t)}{dt} = \dfrac{\beta_h I_a S_h}{1+cI_h^2} - (\mu_h + \theta_h)E_h, \\[2mm] \dfrac{dI_h(t)}{dt} = \theta_h E_h - (\mu_h + \delta_h)I_h - (\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b+I_h})I_h. \end{cases} \qquad (4.1)$$

Obviously, the set $D_2 = \{(S_a, I_a, S_h, E_h, I_h) \in R_+^5 | S_a + E_a \le \frac{\phi}{\mu_a}, S_h + E_h + I_h \le \frac{\Lambda_h}{\mu_h}\}$ is positively invariant.

4.1. Existence of equilibria in system (4.1)

Theorem 4.1. *System* (4.1) *always has two disease-free equilibria* $A_{ah}(0, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ *and* $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$. *If* $R_0 > 1$, *then system* (4.1) *has a unique endemic equilibrium* $C_{ah}(S_a^*, I_a^*, S_h^*, E_h^*, I_h^*)$.

Proof. We know that S_a^*, I_a^*, S_h^*, E_h^* and I_h^* satisfy

$$\begin{cases} m_a S_a(1 - \dfrac{S_a}{K_a}) - \dfrac{\beta_a I_a S_a}{1+\alpha I_a} = 0, \\[2mm] \dfrac{\beta_a I_a S_a}{1+\alpha I_a} - (\mu_a + \delta_a)I_a = 0, \\[2mm] \Lambda_h - \mu_h S_h - \dfrac{\beta_h I_a S_h}{1+cI_h^2} = 0, \\[2mm] \dfrac{\beta_h I_a S_h}{1+cI_h^2} - (\mu_h + \theta_h)E_h = 0, \\[2mm] \theta_h E_h - (\mu_h + \delta_h)I_h - (\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b+I_h})I_h = 0. \end{cases} \qquad (4.2)$$

Then we have

$$S_h = \frac{\Lambda_h - (\mu_h + \theta_h)E_h}{\mu_h}, \qquad E_h = \frac{(\mu_h + \mu_0 + \delta_h)I_h^2 + b(\mu_h + \delta_h + \mu_1)I_h}{\theta_h(b+I_h)}. \qquad (4.3)$$

Substituting (4.3) into the third equation of (4.2), we obtain

$$f(I_h) = m_4 I_h^4 + m_3 I_h^3 + m_2 I_h^2 + m_1 I_h + m_0 = 0, \tag{4.4}$$

where

$$m_4 = -c\mu_h(\mu_h + \theta_h)(\mu_0 + \mu_h + \delta_h) < 0,$$
$$m_3 = -bc\mu_h(\mu_h + \theta_h)(\mu_h + \delta_h + \mu_1) < 0,$$
$$m_2 = -(\mu_h + \theta_h)(\mu_0 + \mu_h + \delta_h)(\beta_h I_a^* + \mu_h) < 0,$$
$$m_1 = \beta_h \theta_h \Lambda_h I_a^* - b(\mu_h + \theta_h)(\mu_h + \delta_h + \mu_1)(\beta_h I_a^* + \mu_h),$$
$$m_0 = b\beta_h \theta_h \Lambda_h I_a^* > 0.$$

It is clear that $f(0) = m_0 > 0$ and $f(+\infty) < 0$. Applying the interval-value theorem of continuous functions, (4.4) has at least one positive root. Suppose that (4.4) has four real roots $I_{h1}, I_{h2}, I_{h3}, I_{h4}$. By the Vieta theorem, we have

$$I_{h1}I_{h2}I_{h3}I_{h4} = \frac{m_0}{m_4} < 0, \tag{4.5}$$

$$I_{h1} + I_{h2} + I_{h3} + I_{h4} = -\frac{m_3}{m_4} < 0, \tag{4.6}$$

$$I_{h1}I_{h2} + I_{h1}I_{h3} + I_{h1}I_{h4} + I_{h2}I_{h3} + I_{h3}I_{h4} + I_{h2}I_{h4} = \frac{m_2}{m_4} > 0. \tag{4.7}$$

From (4.5) and (4.6), we can deduce that there are one negative root and three positive roots (or one positive root and three negative roots). Assume that (4.4) has a negative root I_{h1} and three positive roots I_{h2}, I_{h3}, I_{h4}. It follows from (4.6) that

$$I_{h1} + I_{h2} < 0, I_{h1} + I_{h3} < 0, I_{h1} + I_{h4} < 0.$$

Then, we have

$$I_{h1}I_{h2} + I_{h1}I_{h3} + I_{h1}I_{h4} + I_{h2}I_{h3} + I_{h3}I_{h4} + I_{h2}I_{h4}$$
$$= (I_{h1} + I_{h3})I_{h2} + (I_{h1} + I_{h4})I_{h3} + (I_{h1} + I_{h2})I_{h4} < 0,$$

which contradicts with (4.7). This yields that (4.4) has a unique positive real root I_h^*. Thus, system (4.1) has a unique endemic equilibrium $C_{ah}(S_a^*, I_a^*, S_h^*, E_h^*, I_h^*)$ when $R_0 > 1$. □

4.2. Local stability of equilibria in system (4.1)

Theorem 4.2. *The disease-free equilibrium $A_{ah}(0, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ of system* (4.1) *is always unstable; If $R_0 \leq 1$, then the disease-free equilibrium $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ of system* (4.1) *is locally asymptotically stable; If $R_0 > 1$, then the equilibrium $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ is unstable, and the endemic equilibrium $C_{ah}(S_a^*, I_a^*, S_h^*, E_h^*, I_h^*)$ of system* (4.1) *is locally asymptotically stable.*

Proof. The Jacobian matrix of system (4.1) is

$$
J = \begin{pmatrix}
J_1 & -\dfrac{\beta_a S_a}{(1+\alpha I_a)^2} & 0 & 0 & 0 \\[2mm]
\dfrac{\beta_a I_a}{1+\alpha I_a} & J_2 & 0 & 0 & 0 \\[2mm]
0 & -\dfrac{\beta_h S_h}{1+cI_h^2} & J_3 & 0 & J_4 \\[2mm]
0 & \dfrac{\beta_h S_h}{1+cI_h^2} & \dfrac{\beta_h I_a}{1+cI_h^2} & -(\mu_h+\theta_h) & -J_4 \\[2mm]
0 & 0 & 0 & \theta_h & J_5
\end{pmatrix},
$$

where

$$
J_1 = m_a - \frac{2m_a S_a}{K_a} - \frac{\beta_a I_a}{1+\alpha I_a}, \; J_2 = \frac{\beta_a S_a}{(1+\alpha I_a)^2} - (\mu_a + \delta_a),
$$

$$
J_3 = -\mu_h - \frac{\beta_h I_a}{1+cI_h^2}, \; J_4 = \frac{2c\beta_h I_a I_h S_h}{(1+cI_h^2)^2}, \; J_5 = -\mu_h - \delta_h - \mu_0 - \frac{b^2(\mu_1-\mu_0)}{(b+I_h)^2}.
$$

(1) When $(S_a, I_a, S_h, E_h, I_h) = (0, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$, we know that the characteristic equation corresponding to $J(A_{ah})$ always has a positive root $\lambda = m_a$. Hence, A_{ah} is always unstable.

(2) When $R_0 > 1$ and $(S_a, I_a, S_h, E_h, I_h) = (K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$, we get

$$
(\lambda + m_a)(\lambda + (\mu_a + \delta_a)(1 - R_0))(\lambda + \mu_h)(\lambda + \mu_h + \theta_h)(\lambda + \mu_h + \delta_h + \mu_1) = 0.
$$

If $R_0 < 1$, then all the eigenvalues are negative. Hence $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ is locally asymptotically stable when $R_0 < 1$. And $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ is unstable if $R_0 > 1$.

(3) When $R_0 > 1$ and $(S_a, I_a, S_h, E_h, I_h) = (S_a^*, I_a^*, S_h^*, E_h^*, I_h^*)$, we obtain

$$
(\lambda^2 + p\lambda + q)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0, \tag{4.8}
$$

where

$$
p = \frac{2m_a S_a^*}{K_a} + \frac{\beta_a I_a^*}{1+\alpha I_a^*} - m_a - \frac{\beta_a S_a^*}{(1+\alpha I_a^*)^2} + \mu_a + \delta_a > 0,
$$

$$
q = \left(\frac{2m_a S_a^*}{K_a} + \frac{\beta_a I_a^*}{1+\alpha I_a^*} - m_a\right)\left(\mu_a + \delta_a - \frac{\beta_a S_a^*}{(1+\alpha I_a^*)^2}\right) + \frac{\beta_a^2 S_a^* I_a^*}{(1+\alpha I_a^*)^3} > 0,
$$

$$
a_1 = 3\mu_h + \theta_h + \delta_h + \mu_0 + \frac{b^2(\mu_1-\mu_0)}{(b+I_h^*)^2} + \frac{\beta_h I_a^*}{1+c(I_h^*)^2} > 0,
$$

$$
a_2 = (\mu_h+\theta_h)\left[\mu_h+\delta_h+\mu_0+\frac{b^2(\mu_1-\mu_0)}{(b+I_h^*)^2}\right] + \frac{2c\beta_h\theta_h I_a^* I_h^* S_h^*}{(1+c(I_h^*)^2)^2} + \left(\mu_h+\frac{\beta_h I_a^*}{1+c(I_h^*)^2}\right)*
$$

$$
\left[2\mu_h + \theta_h + \delta_h + \mu_0 + \frac{b^2(\mu_1-\mu_0)}{(b+I_h^*)^2}\right] > 0,
$$

$$
a_3 = \left(\mu_h+\frac{\beta_h I_a^*}{1+c(I_h^*)^2}\right)(\mu_h+\theta_h)\left[\mu_h+\delta_h+\mu_0+\frac{b^2(\mu_1-\mu_0)}{(b+I_h^*)^2}\right] + \mu_h\frac{2c\beta_h\theta_h I_a^* I_h^* S_h^*}{(1+c(I_h^*)^2)^2} > 0.
$$

By some calculations, we can get $a_2 a_1 - a_3 > 0$. Then by using the Routh-Hurwitz criterion, we obtain that the endemic equilibrium C_{ah} is locally asymptotically stable. □

Remark 4.1. If $R_0 = 1$, then $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ of system (4.1) is a saddle-node point which is locally asymptotically stable.

4.3. Global stability of equilibria in system (4.1)

We first use the method in [9] to prove the global stability of $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$. Rewrite system (4.1) as

$$\begin{cases} \frac{dX}{dt} = F(X, Z), \\ \frac{dZ}{dt} = G(X, Z). \end{cases} \tag{4.9}$$

The disease-free equilibrium of system (4.9) is $Q_0 = (X^0, 0)$. It is globally asymptotically stable if and only if the following conditions are satisfied.

(H_1) when $\frac{dX}{dt} = F(X, 0)$, X^0 is globally asymptotically stable.

(H_2) $G(X, Z) = BZ - \hat{G}(X, Z)$, where $B = D_Z G(X^0, 0)$ is M matrix (the non-diagonal elements are non-negative); $\forall (X, Z) \in \Gamma$, $\hat{G}(X, Z) \geq 0$, where Γ is the constant attraction region of system (4.9).

Theorem 4.3. If $R_0 < 1$, then $B_{ah}(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0)$ of system (4.1) is globally asymptotically stable.

Proof. Let $X = (S_a, S_h)$, $Z = (I_a, E_h, I_h)$. System (4.1) can be rewritten as

$$\frac{dX}{dt} = F(X, Z),$$

$$\frac{dZ}{dt} = G(X, Z),$$

where

$$F(X, Z) = \begin{pmatrix} m_a S_a (1 - \frac{S_a}{K_a}) - \frac{\beta_a I_a S_a}{1 + \alpha I_a} \\ \Lambda_h - \mu_h S_h - \frac{\beta_h I_a S_h}{1 + c I_h^2} \end{pmatrix},$$

$$G(X, Z) = \begin{pmatrix} \frac{\beta_a I_a S_a}{1 + \alpha I_a} - (\mu_a + \delta_a) I_a \\ \frac{\beta_h I_a S_h}{1 + c I_h^2} - (\mu_h + \theta_h) E_h \\ \theta_h E_h - (\mu_h + \delta_h) I_h - (\mu_0 + \frac{b(\mu_1 - \mu_0)}{b + I_h}) I_h \end{pmatrix}.$$

Clearly, $G(X, 0) = 0$ and the disease-free equilibrium of system (4.1) is $P_0(X^0, 0)$ with $X^0 = (K_a, \frac{\Lambda_h}{\mu_h})$. Since

$$F(X, 0) = \begin{pmatrix} m_a S_a (1 - \frac{S_a}{K_a}) \\ \Lambda_h - \mu_h S_h \end{pmatrix},$$

we know that

$$\lim_{t \to \infty} S_a(t) = K_a, \quad \lim_{t \to \infty} S_h(t) = \frac{\Lambda_h}{\mu_h}.$$

Thus, $X^0 = (K_a, \frac{\Lambda_h}{\mu_h})$ is globally asymptotically stable. Then condition (H_1) is satisfied.

Let

$$B = \begin{pmatrix} -\mu_a - \delta_a + \beta_a K_a & 0 & 0 \\ \beta_h(1 + I_h)\frac{\Lambda_h}{\mu_h} & -\mu_h - \theta_h & 0 \\ 0 & \theta_h & -\mu_h - \delta_h \end{pmatrix}.$$

Then $G(X, Z) = BZ - \hat{G}(X, Z)$, where

$$\hat{G}(X, Z) = \begin{pmatrix} \beta_a I_a(K_a - \frac{S_a}{1 + \alpha I_a}) \\ \beta_h I_a(1 + I_h)(\frac{\Lambda_h}{\mu_h} - \frac{S_h}{(1 + I_h)(1 + cI_h^2)}) \\ \mu_0 + \frac{b(\mu_1 - \mu_0)}{b + I_h} \end{pmatrix}.$$

Clearly, B is M matrix and $\hat{G}(X, Z) \geq 0$, $\forall(X, Z) \in D_2$. So, condition (H_2) is satisfied. Thus, B_{ah} of system (4.1) is globally asymptotically stable when $R_0 < 1$.

$\square$

According to the above discussions, we can reduce system (4.1) to

$$\begin{cases} \dfrac{dS_h(t)}{dt} = \Lambda_h - \mu_h S_h - \dfrac{\beta_h I_a^* S_h}{1 + cI_h^2}, \\ \dfrac{dE_h(t)}{dt} = \dfrac{\beta_h I_a^* S_h}{1 + cI_h^2} - (\mu_h + \theta_h)E_h, \\ \dfrac{dI_h(t)}{dt} = \theta_h E_h - (\mu_h + \delta_h)I_h - (\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b + I_h})I_h. \end{cases} \quad (4.10)$$

We shall use Li-Muldowney's geometry method [24] to study the global stability of $C'_{ah}(S_h^*, E_h^*, I_h^*)$ in system (4.10) . Let $|\cdot|$ denote a vector norm in R^n and also denote the induced matrix norm in $R^{n \times n}$, the space of all $n \times n$ matrices. For matrix A in $R^{n \times n}$, the Lozinskǐ measure or the logarithmic norm of A with respect to $|\cdot|$ (see [25]) is

$$\mu(A) = \lim_{h \to 0^+} \frac{|I + hA| - 1}{h}.$$

Let $y(t)$ be a solution of linear differential equation

$$\dot{y}(t) = A(t)y(t),$$

where $A(t)$ is $m \times m$ matrix-valued continuous function. Then, we have

$$|y(t)| \leq |y(t_0)|e^{\int_{t_0}^t \mu(A(t))dt}, \quad \text{for } t \geq t_0.$$

Let B be an $n \times n$ matrix. The second additive compound matrix of B, denoted by $B^{[2]}$, is an $\binom{n}{2} \times \binom{n}{2}$ matrix. For instance, if $B = (b_{ij})$ is a 3×3 matrix, then

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

Consider the following autonomous system

$$\dot{x} = f(x), \tag{4.11}$$

where $f : \Omega \to R^n$, $\Omega \subset R^n$ is an open set and simply connected and $f \in C^1(\Omega)$. Let $x(t, x_0)$ be the solution of system (4.11) such that $x(0, x_0) = x_0$. Let x^* be an equilibrium of system (4.11), i.e., $f(x^*) = 0$. A set K is said to be absorbing in Ω for system (4.11) if $x(t, K_1) \subset K$ for each compact set $K_1 \subset \Omega$ and sufficiently large t. Assume the following assumptions hold:

(H_3) System (4.11) has a unique equilibrium point x^* in Ω.

(H_4) System (4.11) has a compact absorbing set $K \subset \Omega$.

Let $Q : \Omega \mapsto Q(x)$ be an $\binom{n}{2} \times \binom{n}{2}$ matrix-valued functions with its inverse $Q^{-1}(x)$. Let μ be a Lozinskǐ measure on $R^{N \times N}$, where $N = \binom{n}{2}$. Define

$$\bar{q}_2 = \limsup_{t \to \infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^1 \mu(X(x(s, x_0))) ds,$$

where

$$X = Q_f Q^{-1} + Q J^{[2]} Q^{-1},$$

and the matrix Q_f is obtained by replacing each entry q_{ij} of Q by its derivative in the direction of f, $(q_{ij})f$, and $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J of system (4.11) . The following lemma in Li and Muldowney [24] will be used here.

Lemma 4.1 ([24]). *Assume that Ω is simply connected and assumptions (H_3) and (H_4) hold. Then, the unique equilibrium x^* of system* (4.11) *is globally asymptotically stable in Ω if there exist a function Q and a Lozinskǐ measure μ such that $\bar{q}_2 < 0$.*

From the above statement, we now state our main result.

Theorem 4.4. *If $R_0 > 1$ and* (4.13) *hold, then the unique endemic equilibrium of system* (4.10) *is globally asymptotically stable in D_2.*

Proof.   The Jacobian matrix of system (4.10) is

$$J_p = \begin{pmatrix} J_{P1} & 0 & \dfrac{2c\beta_h I_a^* I_h S_h}{(1 + cI_h^2)^2} \\ \dfrac{\beta_h I_a^*}{1 + cI_h^2} & J_{P2} & -\dfrac{2c\beta_h I_a^* I_h S_h}{(1 + cI_h^2)^2} \\ 0 & \theta_h & J_{P3} \end{pmatrix}, \tag{4.12}$$

where $J_{P1} = -\mu_h - \dfrac{\beta_h I_a^*}{1 + cI_h^2}$, $J_{P2} = -(\mu_h + \theta_h)$, $J_{P3} = -d - \dfrac{b^2(\mu_1 - \mu_0)}{(b + I_h)^2}$, $d = \mu_h + \delta_h + \mu_0$.

By Theorems 4 and 5, we have

(1) If $R_0 > 1$, then system (4.1) has a unique endemic equilibrium which is locally asymptotically stable in D_2. Assumption (H_3) holds.

(2) If $R_0 > 1$, then A_{ah} and B_{ah} are unstable. The instability of $A_{ah} \in \partial D_2$ and $B_{ah} \in \partial D_2$ imply the uniform persistence [16], i.e., there exists a constant $m > 0$ such that

$$\liminf_{t \to \infty} x(t) \geq m, \quad \text{where}\ \ x = S_a, I_a, S_h, E_h, I_h.$$

Due to D_2 is bounded, there must exist a compact set in the interior of D_2 which is absorbing for system (4.10). So, (H_3) is verified.

From system (4.10), we get

$$J^{[2]} = \begin{pmatrix} J_{P1} + J_{P2} & -\dfrac{2c\beta_h I_a^* I_h S_h}{(1+cI_h^2)^2} & -\dfrac{2c\beta_h I_a^* I_h S_h}{(1+cI_h^2)^2} \\ \theta_h & J_{P1} + J_{P3} & 0 \\ 0 & \dfrac{\beta_h I_a^*}{1+cI_h^2} & J_{P2} + J_{P3} \end{pmatrix}.$$

Let $P(S_h, E_h, I_h) = \text{diag}(1, \frac{E_h}{I_h}, \frac{E_h}{I_h})$. Then, $P_f P^{-1} = \text{diag}(0, \frac{\dot{E}_h}{E_h} - \frac{\dot{I}_h}{I_h}, \frac{\dot{E}_h}{E_h} - \frac{\dot{I}_h}{I_h})$ and $B = P_f P^{-1} + P J^{[2]} P^{-1}$, where

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

$$B_{11} = -\mu_h - \frac{\beta_h I_a^*}{1+cI_h^2} - (\mu_h + \theta_h), \, B_{21} = (\frac{E_h}{I_h}\theta_h, 0)^{\mathrm{T}},$$

$$B_{12} = (-\frac{I_h}{E_h}\frac{2c\beta_h I_a^* I_h S_h}{(1+cI_h^2)^2}, -\frac{I_h}{E_h}\frac{2c\beta_h I_a^* I_h S_h}{(1+cI_h^2)^2}),$$

$$B_{22} = \begin{pmatrix} -\mu_h - \dfrac{\beta_h I_a^*}{1+cI_h^2} - d - \dfrac{b^2(\mu_1-\mu_0)}{(b+I_h)^2} + \dfrac{\dot{E}_h}{E_h} - \dfrac{\dot{I}_h}{I_h} & 0 \\ \dfrac{\beta_h I_a^*}{1+cI_h^2} & -d - \dfrac{b^2(\mu_1-\mu_0)}{(b+I_h)^2} - (\mu_h + \theta_h) + \dfrac{\dot{E}_h}{E_h} - \dfrac{\dot{I}_h}{I_h} \end{pmatrix}.$$

Take any vector $(S_h, E_h, I_h) \in R^3 \simeq R^{\binom{n}{2}}$. We choose a norm $|(S_h, E_h, I_h)| = \max\{|S_h|, |E_h| + |I_h|\}$ in R^3. Let $\mu(\cdot)$ be the Lozinskĭ measure with this vector norm, which can be obtained according to [28], then

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

where $|B_{12}|, |B_{21}|$ are the matrix norms with respect to l_1 vector form

$$B_{11} = -2\mu_h - \frac{\beta_h I_a^*}{1+cI_h^2} - \theta_h, \; B_{12} = -\frac{I_h}{E_h}\frac{2c\beta_h I_a^* I_h S_h}{(1+cI_h^2)^2}.$$

Then we have

$$g_1 = -\frac{\beta_h I_a^*}{1+cI_h^2} - (2\mu_h + \theta_h) - \frac{I_h}{E_h}\frac{2c\beta_h I_a^* I_h S_h}{(1+cI_h^2)^2}.$$

Calculations show that $B_{21} = \frac{E_h}{I_h}\theta_h$ and

$$B_{22} = \max\{-\mu_h - d - \frac{b^2(\mu_1-\mu_0)}{(b+I_h)^2} + \frac{\dot{E}_h}{E_h} - \frac{\dot{I}_h}{I_h}, -\mu_h - \theta_h - d - \frac{b^2(\mu_1-\mu_0)}{(b+I_h)^2} + \frac{\dot{E}_h}{E_h} - \frac{\dot{I}_h}{I_h}\}$$

$$= -\mu_h - d - \frac{b^2(\mu_1 - \mu_0)}{(b+I_h)^2} + \frac{\dot{E}_h}{E_h} - \frac{\dot{I}_h}{I_h}.$$

Hence, we have

$$g_2 = -\mu_h - d - \frac{b^2(\mu_1 - \mu_0)}{(b + I_h)^2} + \frac{\dot{E_h}}{E_h} - \frac{\dot{I_h}}{I_h} + \frac{E_h}{I_h}\theta_h.$$

From the second and third equations of system (4.10), we obtain

$$\frac{\dot{E_h}}{E_h} = \frac{\beta_h I_a^*}{1 + cI_h^2}\frac{S_h}{E_h} - \mu_h - \theta_h,$$

$$\frac{\dot{I_h}}{I_h} = \theta_h\frac{E_h}{I_h} - d - \frac{b(\mu_1 - \mu_0)}{b + I_h}.$$

Then

$$g_1 = \frac{\dot{E_h}}{E_h} - \mu_h - \frac{\beta_h I_a^*}{1 + cI_h^2} - \frac{\beta_h I_a^*}{1 + cI_h^2}\frac{S_h}{E_h} - \frac{I_h}{E_h}\frac{2c\beta_h I_a^* I_h S_h}{(1 + cI_h^2)^2},$$

$$g_2 = \frac{\dot{E_h}}{E_h} - \mu_h + \frac{b(\mu_1 - \mu_0)I_h}{(b + I_h)^2} \leq \frac{\dot{E_h}}{E_h} - \mu_h + \frac{(\mu_1 - \mu_0)I_h}{b + I_h},$$

$$\mu_B \leq \frac{\dot{E_h}}{E_h} - \mu_h + \frac{(\mu_1 - \mu_0)I_h}{b + I_h}.$$

Since $I_h \leq \frac{\Lambda_h}{\mu_h}$, it is easy to see that if

$$b > \frac{(\mu_1 - \mu_0 - \mu_h)\Lambda_h}{\mu_h^2} \tag{4.13}$$

holds, then

$$\mu_B \leq \frac{\dot{E_h}}{E_h} - \sigma,$$

where

$$\sigma = \mu_h - \frac{(\mu_1 - \mu_0)\Lambda_h}{b\mu_h + \Lambda_h}.$$

Along each solution (S_h, E_h, I_h) of system (4.10), we have

$$\frac{1}{t}\int_0^t \mu(B)ds = \frac{1}{t}\int_0^{t_1} \mu(B)ds + \frac{1}{t}\int_{t_1}^t \mu(B)ds$$

$$\leq \frac{1}{t}\int_0^{t_1} \mu(B)ds + \frac{1}{t}\ln\frac{E_h(t)}{E_h(t_1)} - \frac{\sigma(t - t_1)}{t}.$$

This means that

$$\bar{q}_2 = \limsup_{t \to \infty}\sup_{x \in D_2}\frac{1}{t}\int_0^t \mu(B(x(s, x_0)))ds \leq -\frac{\sigma}{2} < 0.$$

According to the analysis above-mentioned, if $R_0 > 1$ and (4.13) hold, $C'_{ah}(S_h^*, E_h^*, I_h^*)$ of system (4.10) is globally asymptotically stable.   □

Based on the above-mentioned analysis, then we can obtain the following theorem.

Theorem 4.5. *The disease-free equilibrium $A(0, 0, \frac{\Lambda_h}{\mu_h}, 0, 0, 0)$ of system (2.1) is always unstable; the disease-free equilibrium $B(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0, 0)$ of system (2.1) is globally asymptotically stable if $R_0 < 1$; the equilibrium $B(K_a, 0, \frac{\Lambda_h}{\mu_h}, 0, 0, 0)$ is unstable if $R_0 > 1$; the endemic equilibrium $C(S_a^*, I_a^*, S_h^*, E_h^*, I_h^*, R_h^*)$ of system (2.1) is globally asymptotically stable if $R_0 > 1$ and (4.13) holds.*

Remark 4.2. Note that, the global stability of the endemic equilibrium $C(S_a^*, I_a^*, S_h^*, E_h^*, I_h^*, R_h^*)$ of system (2.1) is obtained under the particular condition (4.13) which may be limited by the analytical method. Numerical simulation shows that the equilibrium $C(S_a^*, I_a^*, S_h^*, E_h^*, I_h^*, R_h^*)$ of system (2.1) is globally asymptotically stable when condition (4.13) is not satisfied (see Figure 1(b)).
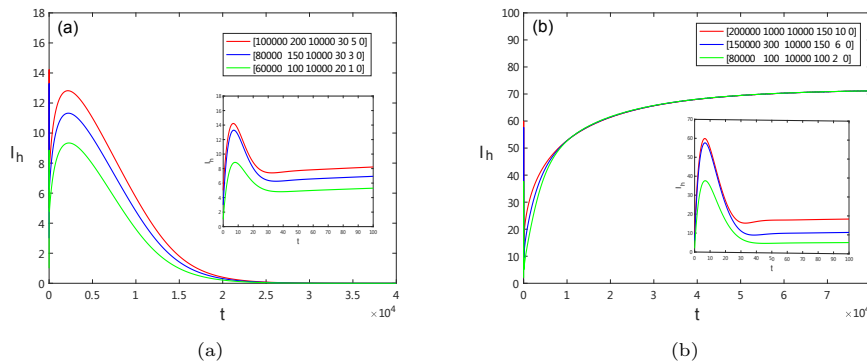

Figure 1. (*a*): $I_h(t)$ converges to the disease-free equilibrium when $\beta_a = 8 \times 10^{-9}(R_0 < 1)$, (*b*): $I_h(t)$ is asymptotically stable and converges to the endemic equilibrium when $\beta_a = 3.5 \times 10^{-8}(R_0 > 1)$.

5. Numerical simulations

In this section, we give some numerical simulations to illustrate the effect of the basic reproduction number R_0, inhibitory effect coefficient α, the psychological effect coefficient c, and hospital beds-population ratio b during disease transmission. We fixed the following parameter values in [23]: $m_a = 5 \times 10^{-3}, K_a = 5 \times 10^4, \mu_a = 3.4246 \times 10^{-4}, \delta_a = 4 \times 10^{-4}, \Lambda_h = 30, \mu_h = 3.91 \times 10^{-5}, \theta_h = \frac{1}{7}, \delta_h = 0.077, \mu_0 = 0.067, \beta_h = 8 \times 10^{-7}$. Let $R_0 = 1$, we have $\beta_a^* = 1.48 \times 10^{-8}$.

Example 5.1. Choose $\alpha = 0.001, c = 0.01, b = 0.05, \mu_1 = 0.1$ and vary $\beta_a^* = \{8 \times 10^{-9}, 3.5 \times 10^{-8}\}$. In Figure 1(a), we keep $\beta_a^* = 8 \times 10^{-9}$ and change $\{S_a(0), I_a(0), S_h(0), E_h(0), I_h(0), R_h(0)\} = \{[100000, 200, 10000, 30, 5, 0], [80000, 150, 10000, 30, 3, 0],$ and $[60000, 100, 10000, 20, 1, 0]\}$, respectively. In Figure 1(b), we take $\beta_a^* = 3.5 \times 10^{-8}$ and vary $\{S_a(0), I_a(0), S_h(0), E_h(0), I_h(0), R_h(0)\} = \{[200000, 1000, 10000, 150, 10, 0], [150000, 300, 10000, 150, 6, 0],$ and $[80000, 100, 10000, 100, 2, 0]\}$, respectively. When $\beta_a < \beta_a^*$, i.e., $R_0 < 1$, $I_h(t)$ converges to the disease-free equilibrium which is locally asymptotically stable (see Figure 1(a)). When $\beta_a > \beta_a^*$, i.e., $R_0 > 1$, $I_h(t)$ converges to the endemic equilibrium which is locally asymptotically stable (see Figure 1(b)). The figures illustrate that the increasing of the rate of disease transmission will cause the number of infected human

to increase.

Example 5.2. Set $\mu_1 = 0.1, \beta_h = 8 \times 10^{-7}$ and vary $\beta_a = \{8 \times 10^{-9}, 3.5 \times 10^{-8}\}, \{\alpha, c, b\} = \{(0,0,0), (0.001, 0.001, 0.001)\}$. In Figure 2(a), the initial value is [100000, 200, 1000, 30, 5, 0] and $\beta_a = 8 \times 10^{-9} < \beta_a^*$, i.e., $R_0 < 1$. In Figure 2(b), $\{S_a(0), I_a(0), S_h(0), E_h(0), I_h(0), R_h(0)\} = [80000, 100, 10000, 100, 2, 0]$ and $\beta_a = 3.5 \times 10^{-8} > \beta_a^*$, i.e., $R_0 > 1$. When the inhibitory effect α, psychological effect c, the ratio of hospital-beds population b are increased together, it will cause the number of infected people to decrease. (see Figure 2).
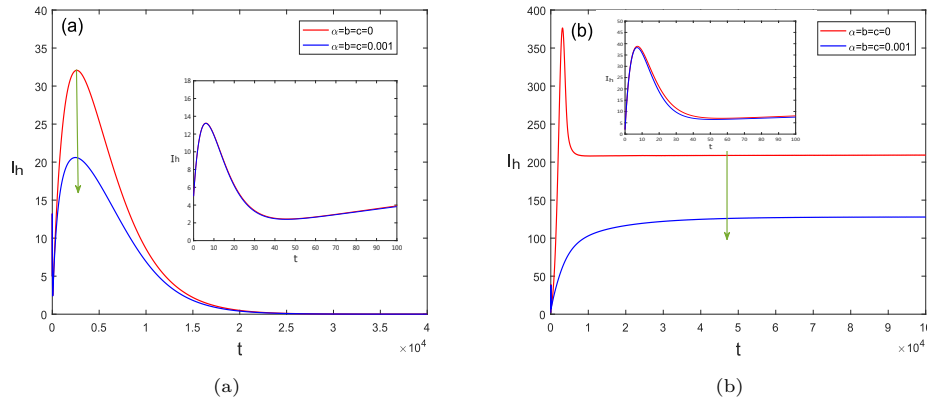

(a)   (b)

Figure 2. The change curve of I_h. (*a*): Initial value is [100000, 200, 1000, 30, 5, 0], $\beta_a = 8 \times 10^{-9}(R_0 < 1)$; (*b*): Initial value is [80000, 100, 10000, 100, 2, 0], $\beta_a = 3.5 \times 10^{-9}(R_0 > 1)$.

Example 5.3. Let $\beta_h = 8 \times 10^{-7}, \alpha = 0.001, c = 0.01$ and vary $b = \{1, 5, 10\}$. In Figure 3(a), $\mu_1 = 0.1, \beta_a = 8 \times 10^{-9} < \beta_a^*, R_0 < 1$. In Figure 3(b), $\mu_1 = 0.25, \beta_a = 1.6 \times 10^{-8} > \beta_a^*, R_0 > 1$. Figure 3 shows that the number of infected individuals I_h decreases when the ratio of hospital beds-population b increases.
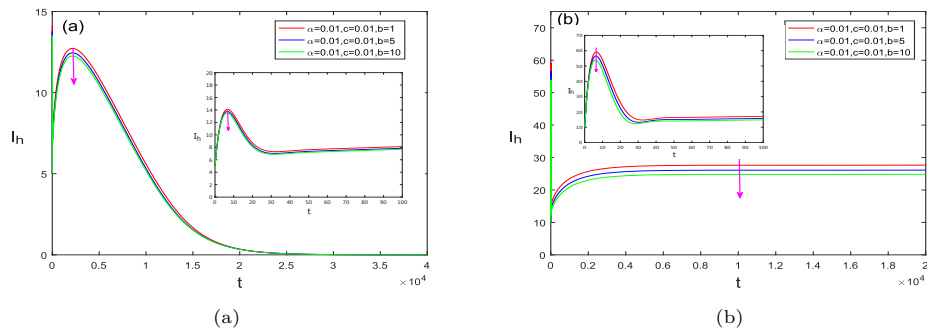

(a)   (b)

Figure 3. The change curve of I_h. (*a*): Initial value is [100000, 200, 10000, 30, 5, 0], $\mu_1 = 0.1$, $\beta_a = 8 \times 10^{-9}(R_0 < 1)$; (*b*): Initial value is [200000, 1000, 10000, 150, 10, 0], $\mu_1 = 0.25$, $\beta_a = 1.6 \times 10^{-8}(R_0 > 1)$.

Example 5.4. Set $\beta_h = 8 \times 10^{-7}, \alpha = 0.001, b = 0.05, \mu_1 = 0.1$ and vary $c = \{0.001, 0.005, 0.015\}$. In Figure 4(a), $\beta_a = 8 \times 10^{-9} < \beta_a^*, R_0 < 1$. In Figure 4(b), $\beta_a = 3.5 \times 10^{-8} > \beta_a^*, R_0 > 1$. Figure 4 shows that the number of infected people I_h decreases when the coefficient of psychological effect c increases.
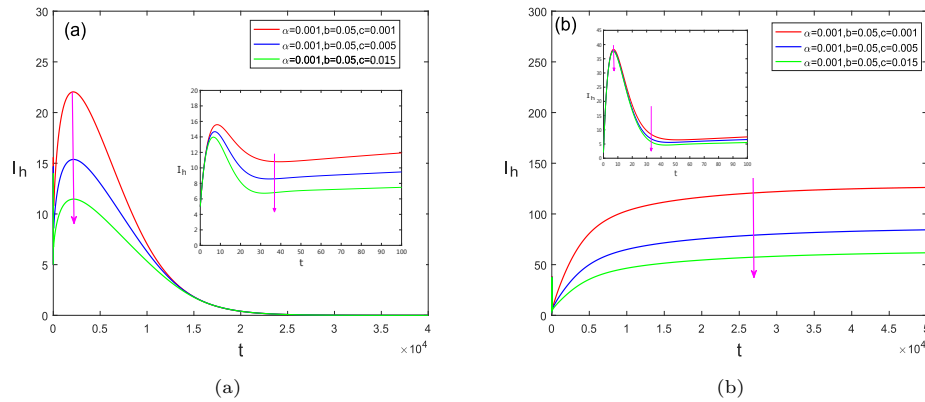

(a)   (b)

Figure 4. The change curve of I_h. (a): Initial value is [100000, 200, 10000, 30, 5, 0],$\beta_a = 8 \times 10^{-9}(R_0 < 1)$; ($b$): Initial value is [80000, 100, 10000, 100, 2, 0], $\beta_a = 3.5 \times 10^{-8}(R_0 > 1)$.

Example 5.5. Choose $\beta_h = 8 \times 10^{-7}, c = 0.001, b = 0.05, \mu_1 = 0.1$ and vary $\alpha = \{0.001, 0.005, 0.01\}$. In Figure 5(a), $\beta_a = 8 \times 10^{-9} < \beta_a^*, R_0 < 1$. In Figure 5(b), $\beta_a = 3.5 \times 10^{-8} > \beta_a^*, R_0 > 1$. Figure 5 indicates that the infected people I_h decreases when the inhibitory effect α increases.
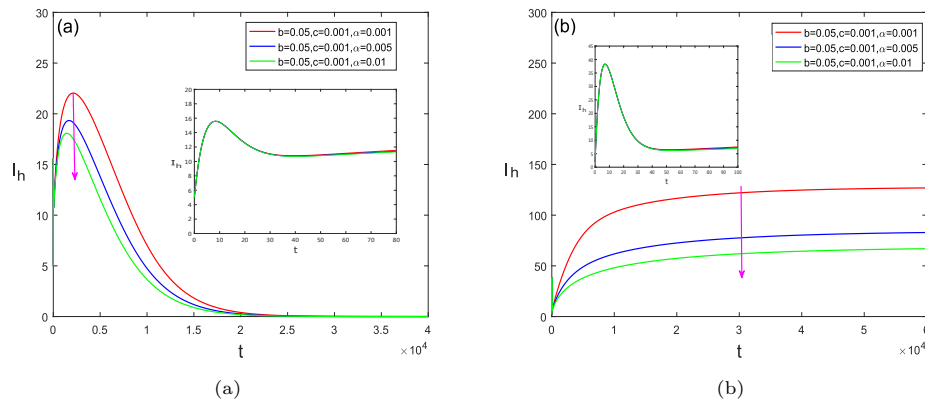

(a)   (b)

Figure 5. The change curve of I_h. (a): Initial value is [100000, 200, 10000, 30, 5, 0], $\beta_a = 8 \times 10^{-9}(R_0 < 1)$; ($b$): Initial value is [80000, 100, 10000, 100, 2, 0], $\beta_a = 3.5 \times 10^{-8} > \beta_a^*(R_0 > 1)$.

6. Discussions and conclusions

In order to investigate the influence of saturation inhibition, psychological effect and medical resources on the spread of avian influenza, we proposed an SI-SEIR avian influenza epidemic model with nonlinear incidence rate and nonlinear recovery rate functions and presented the detailed analysis.

Firstly, the result of the avian sub-model (3.1) is given. It shows that the disease-free equilibrium A_a is always unstable. Moreover, when the basic reproduction number $R_0 < 1$ ($R_0 > 1$), the disease-free equilibrium B_a (the endemic equilibrium C_a) is globally asymptotically stable in D_1;

Secondly, the dynamic behavior of the whole avian influenza model is analyzed. From the theoretical analysis, the disease-free equilibrium A_{ah} is unstable; when the basic reproduction number $R_0 < 1$, the disease-free equilibrium B_{ah} is globally asymptotically stable in D_2 (see Figure 1(a)), i.e., the disease will be eradicted; when $R_0 > 1$ and (4.13) holds, the endemic equilibrium C is globally asymptotically stable in D_2 (see Figure 1(b)), that is to say, the avia inluenza will persist for a long time;

Finally, numerical simulations (Figures 1-5) are also given and some conclusions can be drawn as follows.

(1) The saturation effect inhibition coefficient α, the psychological effect coefficient c, and the bed-to-population ratio b do not change the stability of system (2.1).

(2) When the inhibitory effect α, the ratio of hospital beds-population b and the psychological effect c increase together or seperately, the peak value of infected human will drop, and then the final size of the number of infected individual is relatively reduced (see Figures 2-5).

In order to better embody the epidemiology of avian flu, it will be very interesting to induce the periodic incidence rate and the periodic recovery rate in avian influenza epidemic model, we leave it in the future work.

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