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Global dynamics and sliding motion in A(H7N9) epidemic models with limited resources and Filippov control

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ABSTRACT

This paper investigates the effects of different control measures on the dynamics of A(H7N9) virus transmission from poultry to human. An SI-SIR model with nonlinear incidence and recovery rates is formulated to evaluate the combined impacts of government intervention strategies and available hospital resources. The global stability of the disease-free and endemic equilibria is obtained by constructing Lyapunov and Dulac functions. Furthermore, based on the threshold policy, the SI-SIR model is extended to a novel three-dimensional Filippov one to represent the control measures being triggered once the total number of infected poultry and human reaches the tolerant level I_c. Model solutions are able to approach either one real equilibrium or the pseudo-equilibrium, depending on the tolerant threshold. Our results suggest that in order to diminish the outbreak of A(H7N9) virus or lead the number of infections to an expected level, it requires not only adequate hospital resources and certain government interventions, but also a good threshold policy.
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1. Introduction

In March 2013, the first case of human infection by avian influenza A(H7N9) virus was reported in mainland China [12]. According to Chinese Center for Disease Control and Prevention (China CDC), by May 31st, 2018, there had been 1400 laboratory-confirmed human cases, of which included 562 deaths. This particular virus is low pathogenic that typically causes no clinical features in infected poultry, but is highly pathogenic for human [10]. High fever, cough, sputum and respiratory failure are the most common clinical features of patients [7]. In addition, the overall case fatality rate of H7N9 infections is about 40.14%, which has raised attention from both domestic and overseas. Therefore, it is necessary to explore effective control measures in preventing the spread of A(H7N9) virus.

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Direct and indirect contact with infected poultry may be the most risky factor for human [5], thus the government should take appropriate interventions to reduce exposure. The Chinese government released official avian influenza A(H7N9) data immediately on its websites, official news agency, and newspaper [6], so that the public could understand the progress of A(H7N9) outbreak and avoid unnecessary contact. Some municipal governments, like Shanghai, Nanjing, and Guangzhou, implemented strategies including disinfection, market closures and culling of poultry [25,26], where the last two measures were proved to be more effective in controlling A(H7N9) infections [8]. In order to explore the impact of government interventions on the disease transmission dynamics, various types of functions have been proposed [11, 14,24]. In particular, the nonlinear function $\frac{kI^lS}{1+\alpha I^h}$ [18] and the negative exponential function βe^{-mI} [16] have been widely used and developed as incidence rates to characterize the reduction in contact due to government interventions.

Medical treatment is another important method to control and reduce the spread of A(H7N9) virus, which is typically taken as a constant recovery rate in previous avian influenza models [2,9,13,15,22]. However, the limited hospital resources, such as doctors, drugs, medical environment, and hospital beds, can restrict recovery rates. Recently, many researchers have begun working on the dynamic models incorporating the impact of hospital resource limitation [11,17,20]. Hospital bed-population ratio, the number of available hospital beds per 10,000 population, has been used by health planner as a measure of available resources for public [21]. Shan and Zhu [19] defined the nonlinear recovery rate which was reflected by the hospital bed-population ratio and the number of infections. They verified that there were complex dynamical behaviors of the epidemic model due to the variation of the number of hospital beds. Abdelrazec et al. [1] introduced this recovery rate in the transmission dynamics of dengue fever and the study could be helpful in planning of sensible resource allocation for controlling dengue fever.

However, at the emerging stage of the disease, the total number of infected poultry and human is at a relatively lower level, thus the disease is considered to be controllable. In this case, the public is not aware of the disease, and several control measures are usually not implemented by the authorities. When the disease further spreads, the total number exceeds a certain tolerant threshold level (denoted by I_c), and we have to take immediate and effective actions to suppress further progression of the epidemic. This type of control strategy is called a threshold policy and Filippov models have been widely used to describe it [17,23,24]. Chong and Smith formulated the avian-only Filippov model with two thresholds to evaluate the control strategies of culling of birds [3]. In addition, they also proposed a three-dimensional Filippov model for human population to determine quarantine [4].

The purpose of this paper is to derive mathematical models to investigate the effectiveness of different control measures in A(H7N9) transmission. The paper is organized as follows. The classical SI-SIR model is formulated with nonlinear incidence and recovery rates to characterize government interventions and available hospital resources in Section 2. In Section 3, based on the threshold policy, we further extend the work to a novel three-dimensional Filippov model with culling of infected poultry and improving hospital resources as control measures. Moreover, the global dynamics, the sliding motion and numerical simulations of each model are performed accordingly. Conclusion and discussion for further work are given in the final section.

2. The SI-SIR avian influenza model

2.1. Model formulation

For this section, a mathematical model is established to investigate the combined impacts of government interventions and available hospital resources on the spread of A(H7N9) virus. Based on the fact that there is no obvious evidence of sustained human to human transmission, we only concentrate on poultry to

human transmission. The poultry population is divided into two groups: susceptible (S_p) and infected (I_p), respectively, and the human population is divided into three groups: susceptible (S_h), infected (I_h), and recovered (R_h). In order to formulate the corresponding model, we give the following assumptions.

Assumption 1. The susceptible poultry population is subject to the logistic growth

$$r_p S_p \left(1 - \frac{S_p}{K_p}\right),$$

where r_p and K_p are the intrinsic growth rate and maximal carrying capacity of poultry.

Assumption 2. The incidence rate function $f(I_h)$ is chosen as follows, to reflect the impact of government interventions, such as media, market closures and other policies,

$$f(I_h) = \frac{\beta_h}{1 + aI_h + cI_h^2},$$

where β_h is the transmission coefficient, a and c are nonnegative constants. If $a = 0$, $c = 0$, $f(I_h) = \beta_h$, we do not take the impact of government interventions into account. Otherwise, $f(I_h)$ can describe the phenomenon that the infection force may decrease with respect to the infected human as government interventions are implemented.

Assumption 3. We consider the impact of available hospital resources on the recovery rate with a nonlinear function

$$\mu(b, I_h) = \mu_0 + b\frac{(\mu_1 - \mu_0)}{b + I_h},$$

where μ_1 is the maximum per capita recovery rate due to the sufficient health care resources and few infected individuals, μ_0 is the minimum per capita recovery rate due to the basic clinical resources, b is the hospital bed-population ratio which is a nonnegative constant.

Based on the above assumptions, we formulate the SI-SIR avian influenza model as follows:

$$\begin{cases} \dfrac{dS_p}{dt} = r_p S_p \left(1 - \dfrac{S_p}{K_p}\right) - \beta_p S_p I_p, \\ \dfrac{dI_p}{dt} = \beta_p S_p I_p - (\mu_p + \delta_p) I_p, \\ \dfrac{dS_h}{dt} = \Lambda - S_h I_p f(I_h) - \mu_h S_h, \\ \dfrac{dI_h}{dt} = S_h I_p f(I_h) - (\mu_h + \delta_h) I_h - \left(\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b + I_h}\right) I_h, \\ \dfrac{dR_h}{dt} = \left(\mu_0 + b\dfrac{(\mu_1 - \mu_0)}{b + I_h}\right) I_h - \mu_h R_h, \end{cases} \tag{1}$$

where the detailed descriptions of system parameters and their estimated values are listed in Table 1. Since the variable R_h can be decoupled from the first four equations of system (1), we can reduce system (1) to the following form

Table 1
Description of parameters in system (1).

Parameter	Definition	Value (per day)	Ref.
r_p	Intrinsic growth rate of poultry	0.0476	Assumed
K_p	Maximal carrying capacity of the poultry	150000	Assumed
β_p	Transmission rate from infected poultry to susceptible poultry	–	–
μ_p	Natural death rate of poultry (chicken)	$1/(2*365)$	[9]
δ_p	Disease induced death rate of poultry	4.5×10^{-4}	Assumed
Λ	New recruitment and newborn of human	20	Assumed
β_h	Transmission rate from infected poultry to susceptible human	2×10^{-9}	Assumed
μ_h	Natural death rate of human	$1/(75*365)$	China NBS
δ_h	Disease induced death rate of human	0.3445	[15]
μ_0	Minimum recovery rate of human	(0.067–0.100)	[22]
μ_1	Maximum recovery rate of human	$(\mu_0, 10)$	[19]
b	Hospital bed-population ratio	$(0, 20)$	[19]
a	Government intervention effect parameters	–	–
c	Government intervention effect parameters	–	–

$$\begin{cases} \dfrac{dS_p}{dt} = r_p S_p\left(1-\dfrac{S_p}{K_p}\right) - \beta_p S_p I_p, \\ \dfrac{dI_p}{dt} = \beta_p S_p I_p - (\mu_p+\delta_p) I_p, \\ \dfrac{dS_h}{dt} = \Lambda - S_h I_p f(I_h) - \mu_h S_h, \\ \dfrac{dI_h}{dt} = S_h I_p f(I_h) - (\mu_h+\delta_h) I_h - \left(\mu_0 + b\dfrac{(\mu_1-\mu_0)}{b+I_h}\right) I_h. \end{cases} \tag{2}$$

For the convenience of analysis, we study the dynamic system (2) of A(H7N9) virus in the rest part of Section 2.

2.2. Existence of equilibria

In this subsection, we verify the existence of equilibria of system (2) in set

$$\Gamma = \left\{ (S_p, I_p, S_h, I_h) \in R_+^4 : S_h + I_h \le \frac{\Lambda}{\mu_h} \right\},$$

which is the positively invariant attracting set of system (2). Simple calculation yields that system (2) always has two disease-free equilibria $E_1\left(0, 0, \frac{\Lambda}{\mu_h}, 0\right)$ and $E_2\left(K_p, 0, \frac{\Lambda}{\mu_h}, 0\right)$ for all parameter values. Using the next generation matrix, we deduce the basic reproduction number R_0 of system (2):

$$\begin{aligned} R_0 &= \rho\left(\begin{bmatrix} \beta_p K_p & 0 \\ f(0)\frac{\Lambda}{\mu_h} & 0 \end{bmatrix}\begin{bmatrix} \mu_p+\delta_p & 0 \\ 0 & \mu_h+\delta_h+\mu_1 \end{bmatrix}^{-1}\right) \\ &= \frac{\beta_p K_p}{\mu_p+\delta_p}, \end{aligned}$$

where ρ is the spectral radius of a matrix.

Further, we discuss the endemic equilibrium denoted by $E^*(S_p^*, I_p^*, S_h^*, I_h^*)$. Simple calculation yields

$$S_p^* = \frac{\mu_p+\delta_p}{\beta_p},\ I_p^* = \frac{r_p}{\beta_p R_0}(R_0-1).$$

Obviously, if $R_0 > 1$, I_p^* is positive. The coordinate S_h^* of E^* must satisfy

$$S_h^* = \frac{\Lambda - (\mu_h + \delta_h)I_h^* - \left(\mu_0 + b\frac{(\mu_1 - \mu_0)}{b + I_h^*}\right)I_h^*}{\mu_h}. \tag{3}$$

Substitute (3) into the last equation of system (2), and let $dI_h/dt = 0$. Therefore, the coordinate I_h^* of E^* is the positive root of the polynomial equation

$$g(I_h) = n_4 I_h^4 + n_3 I_h^3 + n_2 I_h^2 + n_1 I_h + n_0 = 0, \tag{4}$$

where

$$\begin{aligned}
n_4 &= -cd_0,\\
n_3 &= -bcd_1 - ad_0,\\
n_2 &= -\frac{\beta_h}{\mu_h} d_0 I_p^* - d_0 - abd_1,\\
n_1 &= -b\frac{\beta_h}{\mu_h} d_1 I_p^* - bd_1 + \Lambda\frac{\beta_h}{\mu_h} I_p^*,\\
n_0 &= \Lambda b\frac{\beta_h}{\mu_h} I_p^*,\\
d_0 &= \mu_h + \delta_h + \mu_0,\\
d_1 &= \mu_h + \delta_h + \mu_1.
\end{aligned}$$

To completely examine the existence of the endemic equilibrium E^*, we consider four possible cases.

Case 1. If $a = 0$, $c = 0$, it's clear that equation (4) has a simple positive root $I_h = \frac{-n_1 - \sqrt{n_1^2 - 4n_2 n_0}}{2n_2}$.

Case 2. If $a \neq 0$, $c = 0$, it yields

$$g(0) = n_0 > 0,\ g(+\infty) < 0.$$

From the Intermediate Value Theorem, we know equation (4) has at least one positive root. Then, we consider all the situations.

(i) Equation (4) has three real roots. The Vieta Theorem shows

$$I_{h_1} I_{h_2} I_{h_3} = -\frac{n_0}{n_3} > 0, \tag{5}$$

$$I_{h_1} + I_{h_2} + I_{h_3} = -\frac{n_2}{n_3} < 0. \tag{6}$$

It follows from (5) that there is one positive root with two negative roots, or three positive roots. (6) indicates that at least one negative root should exist. Hence, equation (4) has a unique positive root.

(ii) There is a positive real root and a pair of complex roots, denoted by x, $y + zi$, and $y - zi$, where x, y, z are positive numbers. We have

$$\begin{aligned}
g(I_h) &= (I_h - x)[I_h - (y + zi)][I_h - (y - zi)]\\
&= I_h^3 - (x + 2y)I_h^2 + (y^2 + z^2 + 2xy)I_h - x(y^2 + z^2) = 0.
\end{aligned}$$

Compared with equation (4), it satisfies $-(x + 2y) < 0$, which contradicts with $n_2/n_3 > 0$.

Case 3. If $a = 0$, $c \neq 0$, calculate the first and second derivatives of $g(I_h)$ respectively,

$$\dot{g}(I_h) = 4n_4 I_h^3 + 3n_3 I_h^2 + 2n_2 I_h + n_1,$$
$$\ddot{g}(I_h) = 12n_4 I_h^2 + 6n_3 I_h + 2n_2.$$

For $I_h \geq 0$, $\ddot{g}(I_h) < 0$, which means $\dot{g}(I_h)$ is monotonically decreasing.

(i) If $n_1 > 0$, similar to the analysis of Case 2, $\dot{g}(I_h) = 0$ has a unique positive root denoted by $\widetilde{I}_h$. We get

$$\begin{cases} 0 < \dot{g}(I_h) \leq n_1, & 0 \leq I_h < \widetilde{I}_h, \\ \dot{g}(I_h) \leq 0, & \widetilde{I}_h \leq I_h. \end{cases}$$

Thus, when $0 \leq I_h < \widetilde{I}_h$, $g(I_h)$ is increasing; and it is decreasing for $\widetilde{I}_h \leq I_h$. Obviously, equation (4) has a simple positive root.

(ii) If $n_1 < 0$, $\dot{g}(I_h)$ is always negative for $I_h \geq 0$, i.e., $g(I_h)$ is decreasing, and there is only a positive root of equation (4).

Case 4. If $a \neq 0$, $c \neq 0$, similarly, equation (4) has a unique positive root.

From the above analysis, it can be concluded that equation (4) has a unique positive root denoted by I_h^* for all cases, which implies that system (2) only has one endemic equilibrium $E^*(S_p^*, I_p^*, S_h^*, I_h^*)$. Consequently, we get the following proposition about the existence of all equilibria of system (2).

Proposition 2.1. *Two disease-free equilibria* $E_1\left(0, 0, \frac{\Lambda}{\mu_h}, 0\right)$ *and* $E_2\left(K_p, 0, \frac{\Lambda}{\mu_h}, 0\right)$ *always exist in system (2) and a unique endemic equilibrium* $E^*(S_p^*, I_p^*, S_h^*, I_h^*)$ *exists if* $R_0 > 1$.

2.3. The dynamical behavior of the poultry-only subsystem

In order to discuss the dynamical behavior of system (2), we initially learn the poultry-only subsystem in $\Omega = \{(S_p, I_p) \in R_+^2 : S_p \geq 0,\ I_p \geq 0\}$,

$$\begin{cases} \dfrac{dS_p}{dt} = r_p S_p \left(1 - \dfrac{S_p}{K_p}\right) - \beta_p S_p I_p \equiv P_1(S_p, I_p), \\ \dfrac{dI_p}{dt} = \beta_p S_p I_p - (\mu_p + \delta_p) I_p \equiv Q_1(S_p, I_p), \end{cases} \tag{7}$$

which is given by the first two equations of system (2). By virtue of Proposition 2.1, two disease-free equilibria denoted by $P_1(0, 0)$ and $P_2(K_p, 0)$ always exist in subsystem (7), and a unique endemic equilibrium denoted by $P^*(S_p^*, I_p^*)$ exists if $R_0 > 1$.

Then, we give the following lemma to show the local asymptotical stability of equilibria P_1, P_2 and P^*.

Lemma 2.1. *The disease-free equilibrium* $P_1(0, 0)$ *is always unstable. Furthermore, (i) if* $R_0 < 1$, *the disease-free equilibrium* $P_2(K_p, 0)$ *is locally asymptotically stable; (ii) if* $R_0 > 1$, *the endemic equilibrium* $P^*(S_p^*, I_p^*)$ *is locally asymptotically stable.*

Proof. The Jacobian matrix of subsystem (7) is

$$\mathbf{J}_p(S_p, I_p) = \begin{bmatrix} r_p - 2r_p \frac{S_p}{K_p} - \beta_p I_p & -\beta_p S_p \\ \beta_p I_p & \beta_p S_p - (\mu_p + \delta_p) \end{bmatrix}.$$

(i) $\mathbf{J}_p(P_1)$ has a positive eigenvalue $\lambda = r_p$, thus the disease-free equilibrium P_1 is always unstable.
(ii) All eigenvalues of $\mathbf{J}_p(P_2)$ are $\lambda_1 = -r_p < 0$ and $\lambda_2 = (\mu_p + \delta_p)(R_0 - 1)$. It is shown that, if $R_0 < 1$, the disease-free equilibrium P_2 is locally asymptotically stable, otherwise P_2 is unstable.
(iii) If $R_0 > 1$, the endemic equilibrium P^* exists. The characteristic equation at P^* can be rewritten as

$$w_1(\lambda) = \lambda^2 + \gamma_1\lambda + \gamma_0 = 0 \tag{8}$$

with

$$\gamma_1 = \frac{r_p}{K_p}S_p^*, \qquad \gamma_0 = \beta_p^2 S_p^* I_p^*.$$

Since γ_1, γ_0 are positive, all eigenvalues of $\mathbf{J}_p(P^*)$ have negative real parts. Hence, the endemic equilibrium P^* is locally asymptotically stable. □

Moreover, the global dynamical behavior of subsystem (7) is shown as follows.

Theorem 2.1. *If $R_0 < 1$, the disease-free equilibrium P_2 is globally asymptotically stable in Ω; if $R_0 > 1$ the endemic equilibrium P^* is globally asymptotically stable in Ω.*

Proof. If $R_0 < 1$, we construct Lyapunov function

$$V_1 = S_p - K_p - K_p ln\frac{S_p}{K_p} + I_p.$$

It is not difficult to calculate the derivative V_1 along subsystem (7) as

$$\begin{aligned} V_1'|_{(7)} &= \dot{S}_p\left(1 - \frac{K_p}{S_p}\right) + \dot{I}_p \\ &= -\frac{r_p}{K_p}(K_p - S_p)^2 + (\mu_p + \delta_p)I_p(R_0 - 1) \\ &\le 0. \end{aligned}$$

Then, we obtain the set $M_1 = \{(S_p, I_p) \in R_2^+ \mid V_1' = 0\} = \{P_2\}$. In light of the LaSalle Invariance Principle, all solutions of subsystem (7) approach the largest positively invariant subset of the set M_1. Thus, if $R_0 < 1$, the disease-free equilibrium P_2 is globally asymptotically stable in Ω.

If $R_0 > 1$, we exclude the existence of closed orbits of subsystem (7) by constructing the suitable Dulac function as $B_1(S_p, I_p) = 1/(S_p I_p)$. Direct calculation yields

$$\frac{\partial(B_1P_1)}{\partial S_p} + \frac{\partial(B_1Q_1)}{\partial I_p} = -\frac{r_p}{K_p I_p} < 0.$$

It follows that there is no closed orbit lying entirely in region Ω, which means the endemic equilibrium P^* is globally asymptotically stable in Ω. □

2.4. The dynamical behavior of system (2)

Now, we discuss the dynamical behavior of system (2). To this end, we first show the local asymptotic stability of equilibria E_1, E_2, and E^*.

Lemma 2.2. *The disease-free equilibrium* $E_1\left(0,0,\frac{\Lambda}{\mu_h},0\right)$ *is always unstable. Furthermore,* (*i*) *if* $R_0<1$, *the disease-free equilibrium* $E_2\left(K_p,0,\frac{\Lambda}{\mu_h},0\right)$ *is locally asymptotically stable;* (*ii*) *if* $R_0>1$, *the endemic equilibrium* $E^*(S_p^*,I_p^*,S_h^*,I_h^*)$ *is locally asymptotically stable.*

Proof. The Jacobian matrix of system (2) is

$$\mathbf{J}(S_p,I_p,S_h,I_h)=\begin{bmatrix} r_p-2r_p\frac{S_p}{K_p}-\beta_pI_p & -\beta_pS_p & 0 & 0\\ \beta_pI_p & \beta_pS_p-(\mu_p+\delta_p) & 0 & 0\\ 0 & -S_hf(I_h) & -I_pf(I_h)-\mu_h & -S_hI_p\dot{f}(I_h)\\ 0 & S_hf(I_h) & I_pf(I_h) & S_hI_p\dot{f}(I_h)-d_0-\frac{b^2(\mu_1-\mu_0)}{(b+I_h)^2} \end{bmatrix}.$$

(i) $\mathbf{J}(E_1)$ always has a positive eigenvalue $\lambda=r_p$, thus the disease-free equilibrium E_1 is unstable.
(ii) One of the eigenvalues of $\mathbf{J}(E_2)$ is $\lambda=(\mu_p+\delta_p)(R_0-1)$, and the others are negative. It follows that, if $R_0<1$, the disease-free equilibrium E_2 is locally asymptotically stable, otherwise E_2 is unstable.
(iii) If $R_0>1$, the endemic equilibrium E^* exists. The characteristic equation at E^* is given by

$$w_2(\lambda)=\left(\lambda^2+\gamma_1\lambda+\gamma_0\right)\left(\lambda^2+\eta_1\lambda+\eta_0\right)=0, \tag{9}$$

where γ_1 and γ_2 are defined in (8) and

$$\eta_1=f(I_h^*)I_p^*+\mu_h-\dot{f}(I_h^*)S_h^*I_p^*+d_0+\frac{b^2(\mu_1-\mu_0)}{(b+I_h^*)^2},$$
$$\eta_0=-\mu_h\dot{f}(I_h^*)S_h^*I_p^*+(f(I_h^*)I_p^*+\mu_h)\left(d_0+\frac{b^2(\mu_1-\mu_0)}{(b+I_h^*)^2}\right).$$

Since γ_1, γ_0, η_1, and η_0 are positive, all eigenvalues of $\mathbf{J}(E^*)$ have negative real parts. Therefore, the endemic equilibrium E^* is locally asymptotically stable. □

Further, we give the following theorem about the global dynamics of system (2).

Theorem 2.2. *If* $R_0<1$, *the disease-free equilibrium* E_2 *is globally asymptotically stable in* Γ; *if* $R_0>1$, *the endemic equilibrium* E^* *is globally asymptotically stable in* Γ.

Proof. If $R_0<1$, Theorem 2.1 shows that the disease-free equilibrium $P_2(K_p,0)$ is globally asymptotically stable in subsystem (7). It is easy to know that system (2) can be reduced to

$$\begin{cases}\dfrac{dS_h}{dt}=\Lambda-\mu_hS_h,\\ \dfrac{dI_h}{dt}=-(\mu_h+\delta_h)I_h-\left(\mu_0+b\dfrac{(\mu_1-\mu_0)}{b+I_h}\right)I_h.\end{cases} \tag{10}$$

Construct Lyapunov function

$$V_2=\frac{1}{2}\left(S_h-\frac{\Lambda}{\mu_h}\right)^2+I_h.$$

Calculating the derivative V_2 along system (10), we have

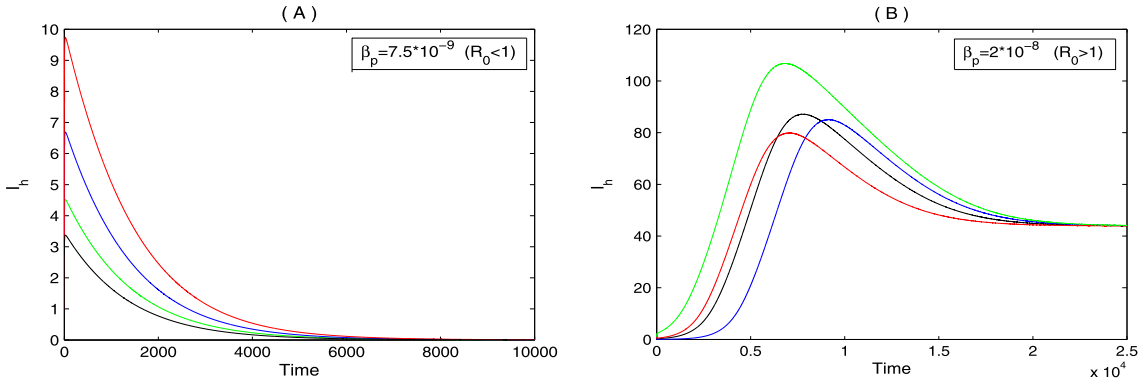


Fig. 1. The plot of $I_h(t)$ with different initial states. (A) If $\beta_p < \beta_p^*(R_0 < 1)$, the solution $I_h(t)$ converges to the disease-free state; (B) If $\beta_p > \beta_p^*(R_0 > 1)$, the solution $I_h(t)$ is globally asymptotically stable and converges to the endemic state value. (For interpretation of the colors in the figures, the reader is referred to the web version of this article.)

$$\begin{aligned}\dot{V}_2|_{(10)} &= -\mu_h\left(S_h - \frac{\Lambda}{\mu_h}\right)^2 - (\mu_h+\delta_h)I_h - \left(\mu_0 + b\frac{(\mu_1-\mu_0)}{b+I_h}\right)I_h \\ &\le -\mu_h\left(S_h - \frac{\Lambda}{\mu_h}\right)^2 - d_0 I_h \\ &\le 0.\end{aligned}$$

Since the set $M_2 = \{(S_h, I_h) \in R_+^2 \mid \dot{V}_2 = 0\} = \left\{(S_h, I_h) \in R_+^2 \mid S_h = \frac{\Lambda}{\mu_h}, I_h = 0\right\}$, according to the LaSalle Invariance Principle, it is concluded that the disease-free equilibrium E_2 is globally asymptotically stable in Γ.

If $R_0 > 1$, system (2) can also be simplified to

$$\begin{cases} \dfrac{dS_h}{dt} = \Lambda - f(I_h)S_h I_p^* - \mu_h S_h \equiv P_2(S_h, I_h), \\ \dfrac{dI_h}{dt} = f(I_h)S_h I_p^* - (\mu_h+\delta_h)I_h - \left(\mu_0 + b\dfrac{(\mu_1-\mu_0)}{b+I_h}\right)I_h \equiv Q_2(S_h, I_h). \end{cases} \tag{11}$$

By the Bendixson-Dulac Theorem, we construct a continuous function for system (11) as $B_2(S_h, I_h) = 1$, and get

$$\frac{\partial(B_2P_2)}{\partial S_h} + \frac{\partial(B_2Q_2)}{\partial I_h} = -f(I_h)I_p^* - \mu_h + \dot{f}(I_h)S_h I_p^* - d_0 - \frac{b^2(\mu_1-\mu_0)}{(b+I_h)^2} < 0.$$

It implies that there is no closed orbit lying entirely in the region $\left\{(S_h, I_h) \in R_+^2 | S_h + I_h \le \frac{\Lambda}{\mu_h}\right\}$. Thus, the endemic equilibrium E^* is globally asymptotically stable in Γ. □

2.5. Numerical simulations

In this part, numerical simulations are presented to verify the theoretical results and the specific parameters used are shown in Table 1. Take $a = 0.005$, $b = 0.5$, $c = 0.0005$, $\mu_0 = 0.091$, $\mu_1 = 0.5$. When the basic reproduction number $R_0 = \beta_p K_p/(\mu_p + \delta_p) = 1$, the threshold value $\beta_p^* = 1.21 * 10^{-8}$. For $\beta_p < \beta_p^*$, the solution $I_h(t)$ converges to the disease-free state E_2 and the disease goes extinct ultimately (see Fig. 1 (A)). For $\beta_p > \beta_p^*$, the solution $I_h(t)$ converges to the endemic state E^* and the disease persists (see Fig. 1 (B)).

Next, in order to investigate the effect of government interventions, we show trajectories of $I_h(t)$ under different parameters values. In Fig. 2 (A), take $(S_p(0), I_p(0), S_h(0), I_h(0), R_h(0)) = (310000, 500, 136000, 1, 0)$

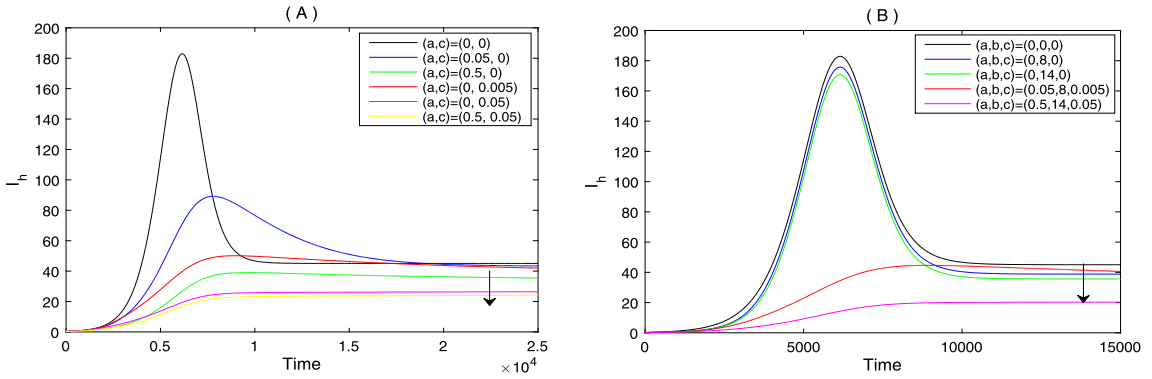


Fig. 2. (A) Impact of government interventions on infected human population ($I_h(t)$) size with increasing parameters a and c values, when $\beta_p = 2 * 10^{-8}$ ($R_0 = 1.648 > 1$); (B) Impact of available hospital resources on infected human population ($I_h(t)$) size with increasing parameter b value, when $\beta_p = 2 * 10^{-8}$ ($R_0 = 1.648 > 1$).

and $\beta_p = 2*10^{-8}$ ($R_0 = 1.648 > 1$). At first, we consider that government interventions are unavailable and hospital resources are at basic level, parameters $(a, b, c) = (0, 0, 0)$ (see the black line). Then for comparison, some appropriate interventions by government are introduced, thus parameters a and c increase from 0 and parameter b remains. The blue and green lines indicate that the outbreak of disease significantly decreases as parameter a increases. Parameter c seems to have a stronger effect on reducing the number of infected human reflected by the red and pink lines. The endemic state declines sharply when both parameters a and c rise (see the yellow line). In addition, it is worth pointing out that the peak time is also be postponed as the parameters a and c increase.

Finally, we discuss the influence of available hospital resources on the control of A(H7N9) virus in Fig. 2 (B). Consider the initial states and conditions are unchanged (see the black line). In the beginning, there are no government interventions, and the changes of parameter b indicate that of hospital resources. Comparing the blue and green lines, we know that higher value of parameter b can lead to the lower peak of the disease outbreak. Furthermore, the red and pink lines represent the conditions for some appropriate strategies implemented by government. With all parameters a, b, and c increasing at the same time, there is a huge impact on the number of infected human, which can reduce not only the outbreak peak, but also the endemic disease state.

3. The SI-SIR avian influenza model with threshold policy

In this section, based on the idea of the threshold policy, we further extend the SI-SIR model into a Filippov one which incorporates culling of infected poultry and improving hospital resources as control measures. Assume that if the total number of infected poultry and human is less than the tolerant threshold, the epidemic situation is controllable and no actions are taken. However, in order to reduce the disease outbreak, effective measures have to be implemented immediately when the total number exceeds the threshold. Then, we have the following non-smooth dynamic system

$$\begin{cases} \dfrac{dS_p}{dt} = \Pi - \beta_p S_p I_p - \mu_p S_p, \\ \dfrac{dI_p}{dt} = \beta_p S_p I_p - (\mu_p + \delta_p) I_p - \varepsilon \mu_c I_p, \\ \dfrac{dS_h}{dt} = \Lambda - \beta_h S_h I_p - \mu_h S_h, \\ \dfrac{dI_h}{dt} = \beta_h S_h I_p - (\mu_h + \delta_h + \mu_0) I_h - \varepsilon \mu_b I_h, \\ \dfrac{dR_h}{dt} = \mu_0 I_h + \varepsilon \mu_b I_h - \mu_h R_h, \end{cases} \tag{12}$$

Table 2
Description of parameters in system (12).

Parameter	Definition	Value (per day)	Ref.
Π	Poultry recruitment rate	2060/365	[4]
β_p	Transmission rate from infected poultry to susceptible poultry	0.001	Assumed
μ_p	Natural death rate of poultry (chicken)	$1/(2*365)$	[9]
δ_p	Disease induced death rate of poultry	0.05	Assumed
μ_c	Culling rate of infected poultry	0.2	Assumed
Λ	Human recruitment rate	1000/365	[4]
β_h	Transmission rate from infected poultry to susceptible human	6.7×10^{-7}	Assumed
μ_h	Natural death rate of human	$1/(75*365)$	China NBS
δ_h	Disease induced death rate of human	0.3445	[15]
μ_0	Recovery rate of human due to the basic clinical resources	0.067	[22]
μ_b	Recovery rate of human due to improving medical resources	0.1	Assumed

with

$$\varepsilon=\begin{cases}0, & I_p+I_h-I_c<0,\\ 1, & I_p+I_h-I_c>0,\end{cases} \tag{13}$$

where I_c is a critical level of the total number of infected poultry and human, μ_c is the culling rate of infected poultry, and μ_b is the recovery rate of human due to improving hospital resources. Detailed descriptions of other system parameters and their estimated values are listed in Table 2.

Without loss of generality, we assume that $\mu_c>\mu_b$ and S_h+I_h is a constant Λ/μ_h. Thus, system (12) can become

$$\begin{cases}\dfrac{dS_p}{dt}=\Pi-\beta_pS_pI_p-\mu_pS_p,\\ \dfrac{dI_p}{dt}=\beta_pS_pI_p-(\mu_p+\delta_p)I_p-\varepsilon\mu_cI_p,\\ \dfrac{dI_h}{dt}=\beta_h\left(\dfrac{\Lambda}{\mu_h}-I_h\right)I_p-(\mu_h+\delta_h+\mu_0)I_h-\varepsilon\mu_bI_h.\end{cases} \tag{14}$$

For convenience of description, we further define $\sigma(Z)=I_p+I_h-I_c$ with vector $Z=(S_p,I_p,I_h)^T\in R^3_+$,

$$f_1(Z)=\begin{pmatrix}\Pi-\beta_pS_pI_p-\mu_pS_p\\ \beta_pS_pI_p-(\mu_p+\delta_p)I_p\\ \beta_h\left(\frac{\Lambda}{\mu_h}-I_h\right)I_p-(\mu_h+\delta_h+\mu_0)I_h\end{pmatrix},$$

$$f_2(Z)=\begin{pmatrix}\Pi-\beta_pS_pI_p-\mu_pS_p\\ \beta_pS_pI_p-(\mu_p+\delta_p)I_p-\mu_cI_p\\ \beta_h\left(\frac{\Lambda}{\mu_h}-I_h\right)I_p-(\mu_h+\delta_h+\mu_0)I_h-\mu_bI_h\end{pmatrix}.$$

Then system (14) with (13) can be rewritten as

$$\dot{Z}=\begin{cases}f_1(Z), & Z\in G_1,\\ f_2(Z), & Z\in G_2,\end{cases} \tag{15}$$

where $G_1=\{Z\in R^3_+|\sigma(Z)<0\}$, $G_2=\{Z\in R^3_+|\sigma(Z)>0\}$. Moreover, the manifold G_s is described as $G_s=\{Z\in R^3_+|\sigma(Z)=0\}$, and the normal vector perpendicular to G_s is denoted as $n=(0,1,1)^T$.

The following definitions of system (15) are necessary for our arguments next.

Definition 3.1. A point Z^* is called a real equilibrium of system (15) if $f_1(Z^*) = 0$ with $\sigma(Z^*) < 0$, or $f_2(Z^*) = 0$ with $\sigma(Z^*) > 0$.

Definition 3.2. A point Z^* is called a virtual equilibrium of system (15) if $f_1(Z^*) = 0$ with $\sigma(Z^*) > 0$, or $f_2(Z^*) = 0$ with $\sigma(Z^*) < 0$.

Definition 3.3. S is the sliding domain, if $\langle n, f_1\rangle > 0$ and $\langle n, f_2\rangle < 0$ on $S \subset G_s$.

Definition 3.4. A point Z^* is called a pseudo-equilibrium, if it is an equilibrium of the sliding mode of system (15).

3.1. Analysis in region G_1

The dynamics of system (15) in region G_1 are described by

$$\begin{pmatrix} S_p' \\ I_p' \\ I_h' \end{pmatrix} = \begin{pmatrix} \Pi - \beta_p S_p I_p - \mu_p S_p \\ \beta_p S_p I_p - (\mu_p + \delta_p) I_p \\ \beta_h \left(\frac{\Lambda}{\mu_h} - I_h\right) I_p - (\mu_h + \delta_h + \mu_0) I_h \end{pmatrix}. \tag{16}$$

For subsystem (16), it has the disease-free equilibrium $E_1^0 = \left(\frac{\Pi}{\mu_p}, 0, 0\right)$ and the endemic equilibrium $E_1^* = (S_{p1}^*, I_{p1}^*, I_{h1}^*)$, where

$$\begin{aligned} S_{p1}^* &= \frac{\mu_p + \delta_p}{\beta_p}, \\ I_{p1}^* &= \frac{\mu_p}{\beta_p}(R_{01} - 1), \\ I_{h1}^* &= \frac{\beta_h \Lambda I_{p1}^*}{\mu_h(\beta_h I_{p1}^* + \mu_h + \delta_h + \mu_0)}, \\ R_{01} &= \frac{\beta_p \Pi}{\mu_p(\mu_p + \delta_p)}. \end{aligned}$$

Next, we give the following theorem about the local asymptotic stability of equilibria E_1^0 and E_1^*.

Theorem 3.1. (i) *The disease-free equilibrium E_1^0 is locally asymptotically stable if $R_{01} < 1$; (ii) the endemic equilibrium E_1^* exists and is locally asymptotically stable if $R_{01} > 1$.*

Proof. The Jacobian matrix of subsystem (16) is

$$\mathbf{J}_1(S_p, I_p, I_h) = \begin{pmatrix} -\beta_p I_p - \mu_p & -\beta_p S_p & 0 \\ \beta_p I_p & \beta_p S_p - (\mu_p + \delta_p) & 0 \\ 0 & \beta_h \left(\frac{\Lambda}{\mu_h} - I_h\right) & -\beta_h I_p - (\mu_h + \delta_h + \mu_0) \end{pmatrix}.$$

(i) The characteristic equation at E_1^0 is

$$(\lambda + \mu_p)[\lambda - (\mu_p + \delta_p)(R_{01} - 1)][\lambda + (\mu_h + \delta_h + \mu_0)] = 0,$$

which implies that

$$\lambda_1 = -\mu_p < 0,$$
$$\lambda_2 = (\mu_p + \delta_p)(R_{01} - 1) < 0, \quad \text{if } R_{01} < 1,$$
$$\lambda_3 = -(\mu_h + \delta_h + \mu_0) < 0.$$

Thus, if $R_{01} < 1$, all eigenvalues of $\mathbf{J}(E_1^0)$ are negative. It is shown that E_1^0 is locally asymptotically stable.

(*ii*) If $R_{01} > 1$, the endemic equilibrium E_1^* exists. The characteristic equation at E_1^* is

$$\lambda^3 + \alpha_1\lambda^2 + \alpha_2\lambda + \alpha_3 = 0,$$

where

$$\alpha_1 = \beta_p I_{p1}^* + \mu_p + \beta_h I_{p1}^* + \mu_h + \delta_h + \mu_0 > 0,$$
$$\alpha_2 = (\beta_p I_{p1}^* + \mu_p)(\beta_h I_{p1}^* + \mu_h + \delta_h + \mu_0) + \beta_p^2 S_{p1}^* I_{p1}^* > 0,$$
$$\alpha_3 = \beta_p^2 S_{p1}^* I_{p1}^* (\beta_h I_{p1}^* + \mu_h + \delta_h + \mu_0) > 0.$$

Furthermore, we can get

$$\alpha_1\alpha_2 - \alpha_3 = \alpha_1(\beta_p I_{p1}^* + \mu_p)(\beta_h I_{p1}^* + \mu_h + \delta_h + \mu_0) + (\beta_p I_{p1}^* + \mu_p)\beta_p^2 S_{p1}^* I_{p1}^* > 0,$$

which indicates that all eigenvalues of $\mathbf{J}(E_1^*)$ have negative real parts by the Routh-Hurwitz Criterion. Hence, the local asymptotic stability of E_1^* is proved. □

3.2. Analysis in region G_2

In region G_2, the dynamics of system (15) are given by

$$\begin{pmatrix} S_p' \\ I_p' \\ I_h' \end{pmatrix} = \begin{pmatrix} \Pi - \beta_p S_p I_p - \mu_p S_p \\ \beta_p S_p I_p - (\mu_p + \delta_p) I_p - \mu_c I_p \\ \beta_h \left(\frac{\Lambda}{\mu_h} - I_h\right) I_p - (\mu_h + \delta_h + \mu_0) I_h - \mu_b I_h \end{pmatrix}. \tag{17}$$

In subsystem (17), there are two equilibria: the disease-free equilibrium $E_2^0 = \left(\frac{\Pi}{\mu_p}, 0, 0\right)$ and a unique endemic equilibrium $E_2^* = (S_{p2}^*, I_{p2}^*, I_{h2}^*)$, where

$$S_{p2}^* = \frac{\mu_p + \delta_p + \mu_c}{\beta_p},$$
$$I_{p2}^* = \frac{\mu_p}{\beta_p}(R_{02} - 1),$$
$$I_{h2}^* = \frac{\beta_h \Lambda I_{p2}^*}{\mu_h(\beta_h I_{p2}^* + \mu_h + \delta_h + \mu_0 + \mu_b)},$$
$$R_{02} = \frac{\beta_p \Pi}{\mu_p(\mu_p + \delta_p + \mu_c)}.$$

It should be noted that

$$R_{01} = \frac{\beta_p \Pi}{\mu_p(\mu_p + \delta_p)} > \frac{\beta_p \Pi}{\mu_p(\mu_p + \delta_p + \mu_c)} = R_{02},$$

$$
\begin{aligned}
S_{p1}^* &= \frac{\mu_p+\delta_p}{\beta_p} < \frac{\mu_p+\delta_p+\mu_c}{\beta_p} = S_{p2}^*,\\
I_{p1}^* &= \frac{\mu_p}{\beta_p}(R_{01}-1) > \frac{\mu_p}{\beta_p}(R_{02}-1) = I_{p2}^*,\\
I_{h1}^* &= \frac{\beta_h\Lambda I_{p1}^*}{\mu_h(\beta_h I_{p1}^*+\mu_h+\delta_h+\mu_0)}\\
&> \frac{\beta_h\Lambda I_{p2}^*}{\mu_h(\beta_h I_{p2}^*+\mu_h+\delta_h+\mu_0)}\\
&> \frac{\beta_h\Lambda I_{p2}^*}{\mu_h(\beta_h I_{p2}^*+\mu_h+\delta_h+\mu_0+\mu_b)} = I_{h2}^*.
\end{aligned}
$$

Further, the local asymptotic stability of equilibria E_2^0 and E_2^* is shown as follows.

Theorem 3.2. (i) *The disease-free equilibrium E_2^0 is locally asymptotically stable if $R_{02}<1$; (ii) the endemic equilibrium E_2^* exists and is locally asymptotically stable if $R_{02}>1$.*

Proof. The Jacobian matrix of subsystem (17) is

$$
\mathbf{J}_2(S_p,I_p,I_h)=\begin{pmatrix}
-\beta_p I_p-\mu_p & -\beta_p S_p & 0\\
\beta_p I_p & \beta_p S_p-(\mu_p+\delta_p+\mu_c) & 0\\
0 & \beta_h\left(\frac{\Lambda}{\mu_h}-I_h\right) & \beta_h I_p-(\mu_h+\delta_h+\mu_0+\mu_b)
\end{pmatrix}.
$$

Similar to the proof of Theorem 3.1, we know if $R_{02}<1$, all eigenvalues of $\mathbf{J}(E_2^0)$ are negative, thus the local asymptotic stability of E_2^0 is concluded. Furthermore, all eigenvalues of $\mathbf{J}(E_2^*)$ have negative real parts if $R_{02}>1$, which means that E_2^* is locally asymptotically stable. □

3.3. Existence of sliding mode and its dynamics

In this subsection, in order to investigate the dynamics of sliding mode of system (15), we initially verify its existence. Note $I_p+I_h=I_c$ on G_s, and calculate

$$
\begin{aligned}
\langle n,f_1\rangle &= \left\langle \begin{pmatrix}0\\1\\1\end{pmatrix}, \begin{pmatrix}\Pi-\beta_p S_p I_p-\mu_p S_p\\ \beta_p S_p I_p-(\mu_p+\delta_p)I_p\\ \beta_h(\frac{\Lambda}{\mu_h}-I_h)I_p-(\mu_h+\delta_h+\mu_0)I_h\end{pmatrix}\right\rangle\\
&= \beta_p S_p I_p-(\mu_p+\delta_p)I_p+\beta_h\left(\frac{\Lambda}{\mu_h}-I_c+I_p\right)I_p-(\mu_h+\delta_h+\mu_0)(I_c-I_p)\\
&= \beta_p S_p I_p+h_1(I_p),
\end{aligned}
$$

where

$$
h_1(I_p)=\beta_h I_p^2+\left(\beta_h\left(\frac{\Lambda}{\mu_h}-I_c\right)+(\mu_h+\delta_h+\mu_0)-(\mu_p+\delta_p)\right)I_p-(\mu_h+\delta_h+\mu_0)I_c,
$$

and

$$\begin{aligned}\langle n, f_2\rangle &= \left\langle \begin{pmatrix} 0 \\ 1 \\ 1 \end{pmatrix}, \begin{pmatrix} \Pi - \beta_p S_p I_p - \mu_p S_p \\ \beta_p S_p I_p - (\mu_p + \delta_p + \mu_c) I_p \\ \beta_h (\frac{\Lambda}{\mu_h} - I_h) I_p - (\mu_h + \delta_h + \mu_0 + \mu_b) I_h \end{pmatrix} \right\rangle \\ &= \beta_p S_p I_p - (\mu_p + \delta_p + \mu_c) I_p + \beta_h \left(\frac{\Lambda}{\mu_h} - I_c + I_p \right) I_p - (\mu_h + \delta_h + \mu_0 + \mu_b)(I_c - I_p) \\ &= \beta_p S_p I_p + h_2(I_p),\end{aligned}$$

where

$$\begin{aligned}h_2(I_p) &= \beta_h I_p^2 + \left(\beta_h \left(\frac{\Lambda}{\mu_h} - I_c \right) + (\mu_h + \delta_h + \mu_0 + \mu_b) - (\mu_p + \delta_p + \mu_c) \right) I_p - (\mu_h + \delta_h + \mu_0 + \mu_b) I_c \\ &= h_1(I_p) - \mu_c I_p - \mu_b (I_c - I_p) \\ &< h_1(I_p).\end{aligned}$$

From $\langle n, f_1\rangle > 0$ and $\langle n, f_2\rangle < 0$, we have

$$\begin{aligned}\langle n, f_1\rangle > 0, \quad \text{if } S_p > H_1(I_p) = -\frac{h_1(I_p)}{\beta_p I_p}, \\ \langle n, f_1\rangle < 0, \quad \text{if } S_p < H_2(I_p) = -\frac{h_2(I_p)}{\beta_p I_p},\end{aligned}$$

and

$$H_2 = H_1 + \frac{\mu_b (I_c - I_p) + \mu_c I_p}{\beta_p I_p} > H_1.$$

In fact, $h_1(0) < 0$ and there is only one positive root of $h_1(I_p) = 0$. If $(\beta_h \Lambda)/\mu_h < \mu_p + \delta_p$ (i.e., $h_1(I_c) < 0$), it can guarantee that $h_1(I_p)$ is negative on the interval $(0, I_c)$, so is $h_2(I_p)$. Thus, the sliding domain $S \subset G_s$ is defined as follows

$$S \triangleq \{(S_p, I_p, I_h) \in G_s : H_1(I_p) < S_p < H_2(I_p), I_p + I_h = I_c\}.$$

Moreover, we apply Utkin equivalent control method to obtain sliding mode equations in the region S. From $\sigma(I_p, I_h) = I_p + I_h - I_c = 0$ and $d\sigma/dt = 0$,

$$\begin{aligned}\frac{d\sigma}{dt} &= \frac{\partial \sigma}{\partial I_p}\frac{dI_p}{dt} + \frac{\partial \sigma}{\partial I_h}\frac{dI_h}{dt} \\ &= \beta_p S_p I_p - (\mu_p + \delta_p) I_p - \varepsilon \mu_c I_p + \beta_h \left(\frac{\Lambda}{\mu_h} - I_h \right) I_p - (\mu_h + \delta_h + \mu_0) I_h - \varepsilon \mu_b I_h \\ &= 0. \end{aligned} \tag{18}$$

Solving equation (18) for ε yields that

$$\begin{aligned}\varepsilon(I_p) &= \frac{\beta_p S_p I_p - (\mu_p + \delta_p) I_p + \beta_h \left(\frac{\Lambda}{\mu_h} - I_h \right) I_p - (\mu_h + \delta_h + \mu_0) I_h}{\mu_c I_p + \mu_b I_h} \\ &= \frac{\beta_p S_p I_p - (\mu_p + \delta_p) I_p + \beta_h \left(\frac{\Lambda}{\mu_h} - I_c \right) I_p + \beta_h I_p^2 - (\mu_h + \delta_h + \mu_0) I_c + (\mu_h + \delta_h + \mu_0) I_p}{\mu_b I_c + (\mu_c - \mu_b) I_p}. \end{aligned} \tag{19}$$

Substitute equation (19) into system (14), the dynamics of sliding mode in S can be given by the following differential equations

$$\begin{cases} \dfrac{dS_p}{dt} = \Pi - \beta_p S_p I_p - \mu_p S_p, \\ \dfrac{dI_p}{dt} = \beta_p S_p I_p - (\mu_p + \delta_p) I_p - \varepsilon(I_p)\mu_c I_p \\ \qquad = \dfrac{I_p[(\beta_p \mu_b S_p - (\mu_p + \delta_p)\mu_b + (\mu_h + \delta_h + \mu_0)\mu_c](I_c - I_p) - \mu_c \beta_h \left(\frac{\Lambda}{\mu_h} - I_c + I_p\right) I_p^2}{\mu_b I_c + (\mu_c - \mu_b) I_p}, \\ \dfrac{dI_h}{dt} = -\dfrac{dI_p}{dt}. \end{cases} \tag{20}$$

Next, we give the following proposition about the existence of equilibrium of sliding mode (20).

Proposition 3.1. *The existence of a unique positive pseudo-equilibrium* $E_s^* = \left(\frac{\Pi}{\beta_p I_{ps}^* + \mu_p}, I_{ps}^*, I_c - I_{ps}^*\right)$ *of sliding mode (20) is assured, if* $\mu_c/\mu_b > (\mu_p + \delta_p)/(\mu_h + \delta_h)$ *and* $E_s^* \in S$.

Proof. Set $dS_p/dt = 0$ and $dI_p/dt = 0$. It follows that

$$S_p = \frac{\Pi}{\beta_p I_p + \mu_p}, \tag{21}$$

and

$$[\mu_b \beta_p S_p - (\mu_p + \delta_p)\mu_b + (\mu_h + \delta_h + \mu_0)\mu_c](I_c - I_p) - \mu_c \beta_h \left(\frac{\Lambda}{\mu_h} - I_c + I_p\right) I_p = 0. \tag{22}$$

After submitting equation (21) into (22), we obtain the following equation of I_p

$$\gamma_1 I_p^3 + \gamma_2 I_p^2 + \gamma_3 I_p + \gamma_4 = 0, \tag{23}$$

where

$$\begin{aligned} \gamma_1 =& -\mu_c \beta_h \beta_p, \\ \gamma_2 =& \mu_b(\mu_p + \delta_p)\beta_p - \mu_c(\mu_h + \delta_h + \mu_0)\beta_p - \mu_c \beta_h \beta_p \left(\frac{\Lambda}{\mu_h} - I_c\right) - \mu_c \beta_h \mu_p, \\ \gamma_3 =& -\mu_b \beta_p \Pi - \mu_b(\mu_p + \delta_p)\beta_p I_c + \mu_b(\mu_p + \delta_p)\mu_p - \mu_c(\mu_h + \delta_h + \mu_0)\mu_p \\ & + \mu_c(\mu_h + \delta_h + \mu_0)\beta_p I_c - \mu_c \beta_h \mu_p \frac{\Lambda}{\mu_h} + \mu_c \beta_h \mu_p I_c, \\ \gamma_4 =& \beta_p \mu_b \Pi I_c - \mu_b(\mu_p + \delta_p)\mu_p I_c + \mu_c(\mu_h + \delta_h + \mu_0)\mu_p I_c. \end{aligned}$$

If $\mu_c/\mu_b > (\mu_p + \delta_p)/(\mu_h + \delta_h)$, we have $\gamma_1 < 0$, $\gamma_2 < 0$, and $\gamma_4 > 0$. Similar to the proof of Proposition 2.1, it can be concluded that equation (23) has a unique positive root denoted by I_{ps}^*. From (21) and $I_p + I_h = I_c$,

$$E_s^* = (S_{ps}^*, I_{ps}^*, I_{hs}^*) = \left(\frac{\Pi}{\beta_p I_{ps}^* + \mu_p}, I_{ps}^*, I_c - I_{ps}^*\right).$$

Furthermore, if $E_s^* \in S \subset G_s$ holds, E_s^* is a unique pseudo-equilibrium. □

Then, we consider the local asymptotical stability of E_s^* and give the following theorem.

Theorem 3.3. *E_s^* is locally asymptotically stable on the sliding domain S.*

Proof. The Jacobian matrix of the first two equations of system (20) is

$$\mathbf{J}_s(S_{ps}^*, I_{ps}^*) = \begin{pmatrix} J_{11} & J_{12} \\ J_{21} & J_{22} \end{pmatrix}, \tag{24}$$

where

$$\begin{aligned} J_{11} &= \frac{\partial \dot{S}_p}{\partial S_p}(S_{ps}^*, I_{ps}^*) = -\beta_p I_{ps}^* - \mu_p < 0, \\ J_{12} &= \frac{\partial \dot{S}_p}{\partial I_p}(S_{ps}^*, I_{ps}^*) = -\beta_p S_{ps}^* < 0, \\ J_{21} &= \frac{\partial \dot{I}_p}{\partial S_p}(S_{ps}^*, I_{ps}^*) = \frac{\beta_p \mu_b I_{ps}^*(I_c - I_{ps}^*)}{\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*} > 0, \end{aligned}$$

and

$$\begin{aligned} J_{22} = \frac{\partial \dot{I}_p}{\partial I_p}(S_{ps}^*, I_{ps}^*) =& \frac{I_{ps}^*(-\beta_p \mu_b S_{ps}^* + \mu_b(\mu_p + \delta_p) - \mu_c(\mu_h + \delta_h + \mu_0))}{\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*} \\ &+ \frac{I_{ps}^* \left(-\mu_c \beta_h \frac{\Lambda}{\mu_h} + \mu_c \beta_h (I_c - I_{ps}^*) - \mu_c \beta_h I_{ps}^* \right)}{\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*} \\ =& - \frac{\mu_c \beta_h \left(\frac{\Lambda}{\mu_h} - I_c + I_{ps}^* \right) I_{ps}^{*2}}{(\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*)(I_c - I_{ps}^*)} \\ &- \frac{\mu_c \beta_h \left(\frac{\Lambda}{\mu_h} - I_c \right) I_{ps}^* + 2\mu_c \beta_h I_{ps}^{*2}}{\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*} < 0. \end{aligned}$$

Thus, we have

$$\begin{aligned} J_{11} \cdot J_{22} - J_{12} \cdot J_{21} =& \frac{\mu_c \beta_h (\beta_p I_{ps}^* + \mu_p) \left(\frac{\Lambda}{\mu_h} - I_c + I_{ps}^* \right) I_{ps}^{*2}}{(\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*)(I_c - I_{ps}^*)} + \frac{\beta_p^2 \mu_b S_{ps}^* I_{ps}^* (I_c - I_{ps}^*)}{\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*} \\ &+ \frac{\mu_c \beta_h (\beta_p I_{ps}^* + \mu_p) \left(\frac{\Lambda}{\mu_h} - I_c \right) I_{ps}^* + 2\mu_c \beta_h (\beta_p I_{ps}^* + \mu_p) I_{ps}^{*2}}{\mu_b I_c + (\mu_c - \mu_b) I_{ps}^*} > 0. \end{aligned}$$

Since $J_{11} + J_{22} < 0$ and $J_{11} \cdot J_{22} - J_{12} \cdot J_{21} > 0$, all eigenvalues of (24) have negative real parts. It is shown that E_s^* is locally asymptotically stable. □

3.4. Dynamical behavior of system (15)

We address the dynamical behavior of system (15) in this subsection. Note that for different choices of the threshold level I_c, system (15) can exhibit various possible dynamics. From the relationships among I_{p1}^*, I_{h1}^*, I_{p2}^* and I_{h2}^*, which are the key factors in determining the dynamics of system (15), we give following

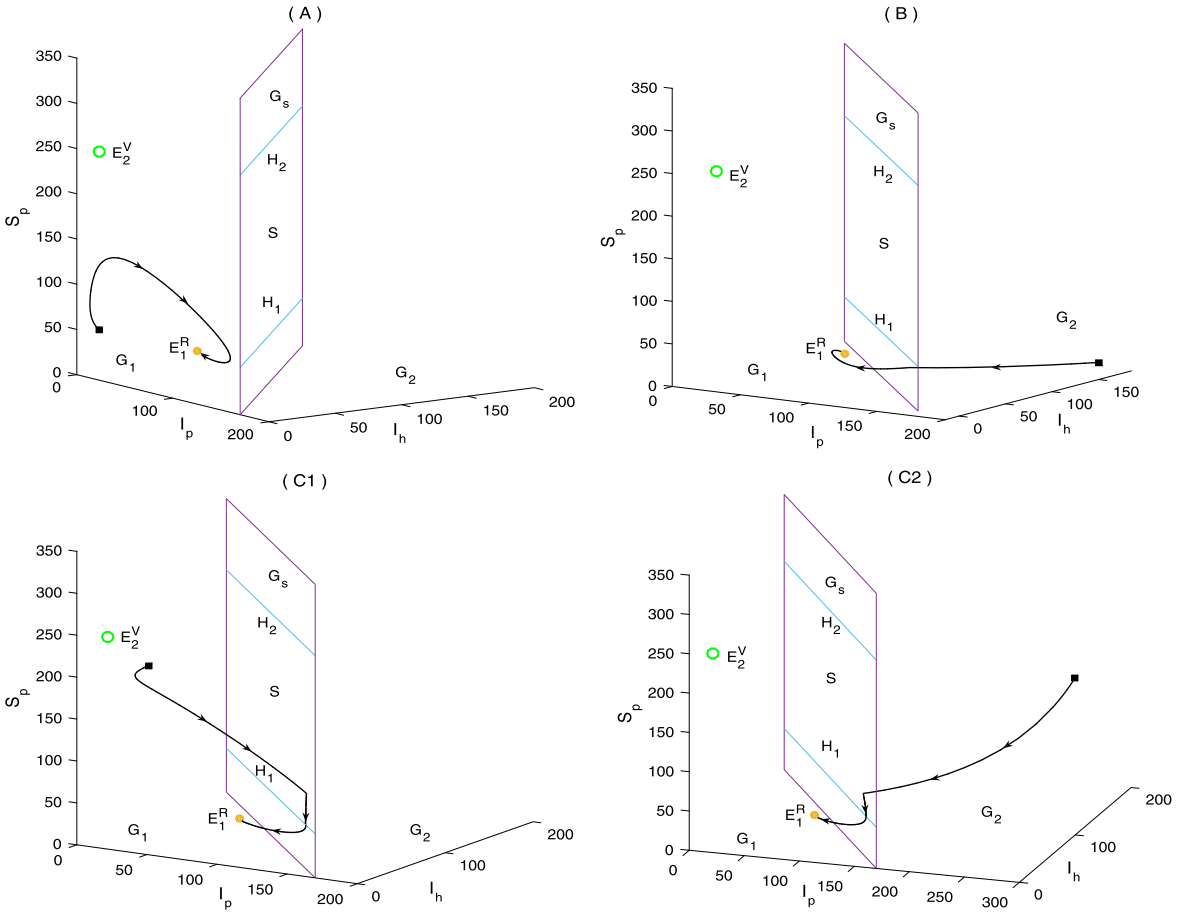


Fig. 3. $E_1^R \in G_1$ achieves stability with $I_c = 170$.

three cases. In order to further demonstrate the theoretical results, some numerical simulations are carried out. The values of all parameters used are given in Table 2.

Case 1: E_1^* is a real equilibrium and E_2^* is a virtual equilibrium.

E_1^* is a real equilibrium and E_2^* is a virtual equilibrium, if the following condition is satisfied

$$I_{p1}^* + I_{h1}^* < I_c \quad \text{and} \quad I_{p2}^* + I_{h2}^* < I_c. \tag{25}$$

In this case, both E_1^* and E_2^* are present in the same side of the manifold (i.e., G_1), and there is no equilibrium located in region G_2. Then, we denote the real equilibrium E_1^* as E_1^R and the virtual equilibrium E_2^* as E_2^V. If (25) is satisfied, we prove that E_1^R can achieve stability.

Theorem 3.4. *If condition (25) is satisfied, E_1^R is stable.*

Proof. This condition implies that region G_1 has only one equilibrium E_1^R, and according to Theorem 3.1, E_1^R is stable. □

Fig. 3 depicts the Case 1 with $I_c = 170$, and the possible trajectories of this figure are as follows:

(a) a trajectory initiating from region G_1 will converge to E_1^R as $t \to +\infty$ without hitting or crossing through the manifold G_s;

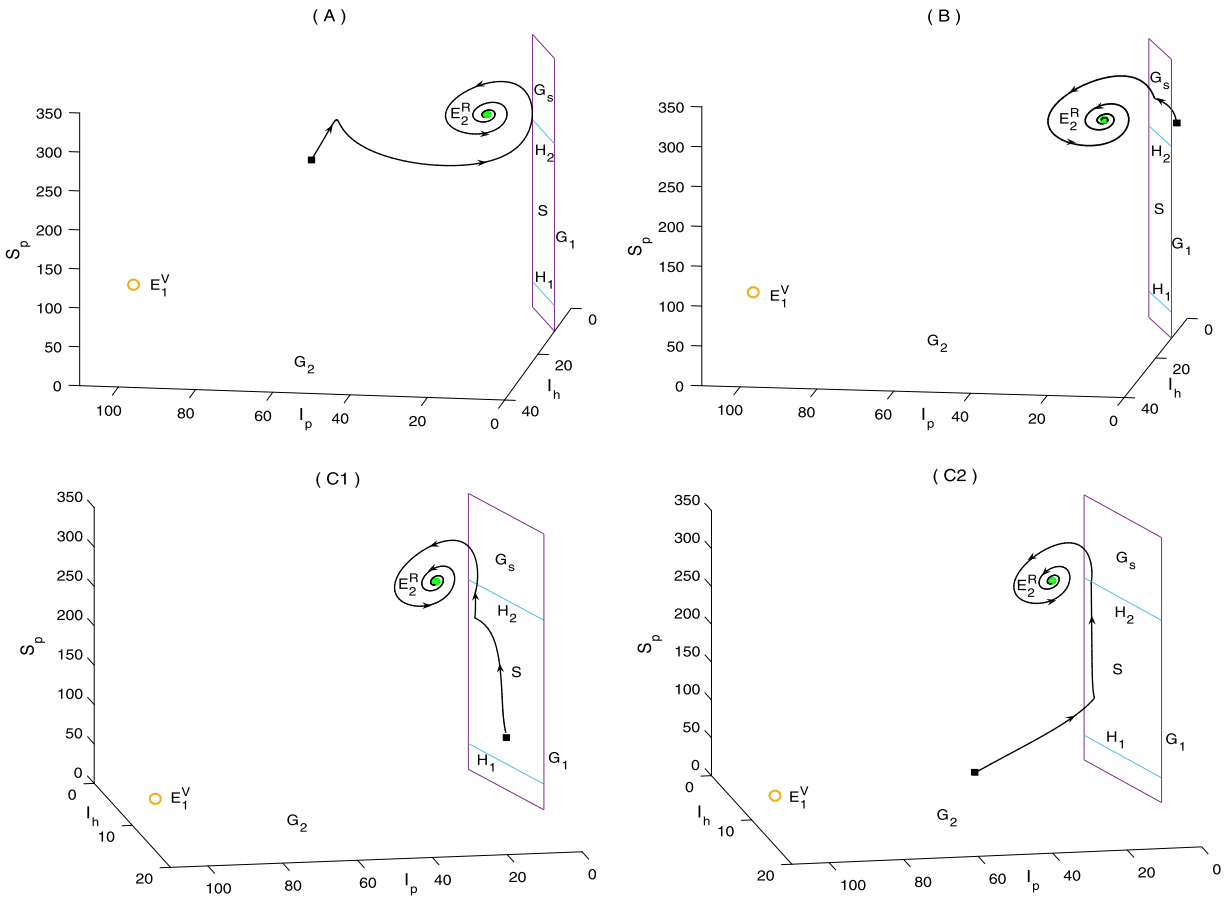


Fig. 4. $E_2^R \in G_2$ achieves stability with $I_c = 10$.

(b) a trajectory with initial point located in region G_2 will cross the manifold G_s, then enter region G_1, and finally converge to E_1^R;

(c) a trajectory that starts in region G_1 or G_2 will hit and slide to the boundary of the sliding domain $S \subset G_S$ before moving towards E_1^R.

Case 2: E_2^* is a real equilibrium and E_1^* is a virtual equilibrium.

If E_1^* and E_2^* can meet the following requirement

$$I_{p1}^* + I_{h1}^* > I_c \quad \text{and} \quad I_{p2}^* + I_{h2}^* > I_c, \tag{26}$$

E_1^* is a virtual equilibrium and E_2^* is a real equilibrium. In this case, both E_1^* and E_2^* are located in the same region G_2, and no equilibrium is lying in region G_1. Then, let us denote the virtual equilibrium E_1^* as E_1^V and the real equilibrium E_2^* as E_2^R. If (26) is satisfied, we show that E_2^R can achieve stability.

Theorem 3.5. *If the condition (26) is satisfied, E_2^R is stable.*

Proof. It is noted that only an equilibrium E_2^R can be found in region G_2, and we have proved that E_2^R is stable in Theorem 3.2. □

Fig. 4 illustrates the result of Theorem 3.5, and the possible trajectories in the Case 2 with $I_c = 10$ are as follows:

(a) a trajectory that begins in region G_2 will converge to E_2^R directly as $t \to +\infty$;
(b) a trajectory with initial point located in region G_1 will pass through the manifold G_s from G_1 to G_2, then move towards E_2^R;
(c) a trajectory with initial condition in region G_1 or G_2 will hit and slide up on the sliding domain $S \subset G_S$ before converging to E_2^R.

Case 3: Both E_1^* and E_2^* are virtual equilibria.

If the following condition is satisfied

$$I_{p1}^* + I_{h1}^* > I_c \quad \text{and} \quad I_{p2}^* + I_{h2}^* < I_c, \tag{27}$$

both E_1^* and E_2^* are virtual equilibria. In this case, it is not difficult to find that E_1^* and E_2^* belong to region G_2 and G_1, respectively. If $E_s^* \in S \subset G_s$, it is a pseudo-equilibrium. We denote these two virtual equilibrium E_1^* as E_1^V and E_2^* as E_2^V. If (27) is satisfied, we prove that E_s^* is stable when it exists.

Theorem 3.6. *If the condition (27) is satisfied, $E_s^* \in S \subset G_s$ is stable when it exists.*

Proof. There is no equilibrium can be found on either side of the manifold G_s. The fact that E_s^* is stable has been proven in Theorem 3.3, and it does not change this character whatever I_c takes between $I_{p1}^* + I_{h1}^*$ and $I_{p2}^* + I_{h2}^*$. □

For this case, we find the possible trajectories from Fig. 5 with $I_c = 80$, which are as follows:

(a) a trajectory with initial condition in region G_1 or G_2 will hit and slid down on $S \subset G_s$ before converging to E_s^*;
(b) a trajectory will pass through the manifold G_s towards G_2 (G_1) from G_1 (G_2). Then it will hit G_s again and move towards E_s^*.

4. Conclusion and discussion

In this paper, we have proposed mathematical models to evaluate the effects of different control strategies on the transmission dynamics of A(H7N9) virus from poultry to human. By applying qualitative theory for continuous and non-smooth systems, we have analyzed the global dynamics and sliding mode dynamics of the models.

In system (2), nonlinear incidence rate $f(I_h)$ and recovery rate $\mu(b, I_h)$ have been introduced to characterize the combined impacts of government interventions and available hospital resources respectively. We have obtained the basic reproduction number R_0, which is a crucial threshold for determining the prevalence of A(H7N9) virus, and addressed the stability of equilibria. Theoretical analysis has shown that the disease-free equilibrium $E_2\left(K_p, 0, \frac{\Lambda}{\mu_h}, 0\right)$ is globally asymptotically stable if $R_0 < 1$, whilst the endemic equilibrium $E^*(S_p^*, I_p^*, S_h^*, I_h^*)$ is globally asymptotically stable if $R_0 > 1$, which means the avian influenza A(H7N9) is persistent (see Fig. 1). Furthermore, although the combined impacts do not cause the A(H7N9) virus to become extinct, the numerical results have revealed that they do effectively delay the peak time and rapidly reduce the outbreak magnitude (see Fig. 2).

It is further considered that typically, the government only carries out the corresponding intervention strategies when the number of infected cases reaches a critical level. Note that the number of infections is a key indicator of reference in implementing control strategies. Therefore, we have extended our work to a novel Filippov model to represent the control measures (i.e., culling of infected poultry and improving hospital resources) being triggered once the total number of infected poultry and human reaches the tolerant level I_c. Based on the discussion of dynamic system (14), we have demonstrated that solutions could approach

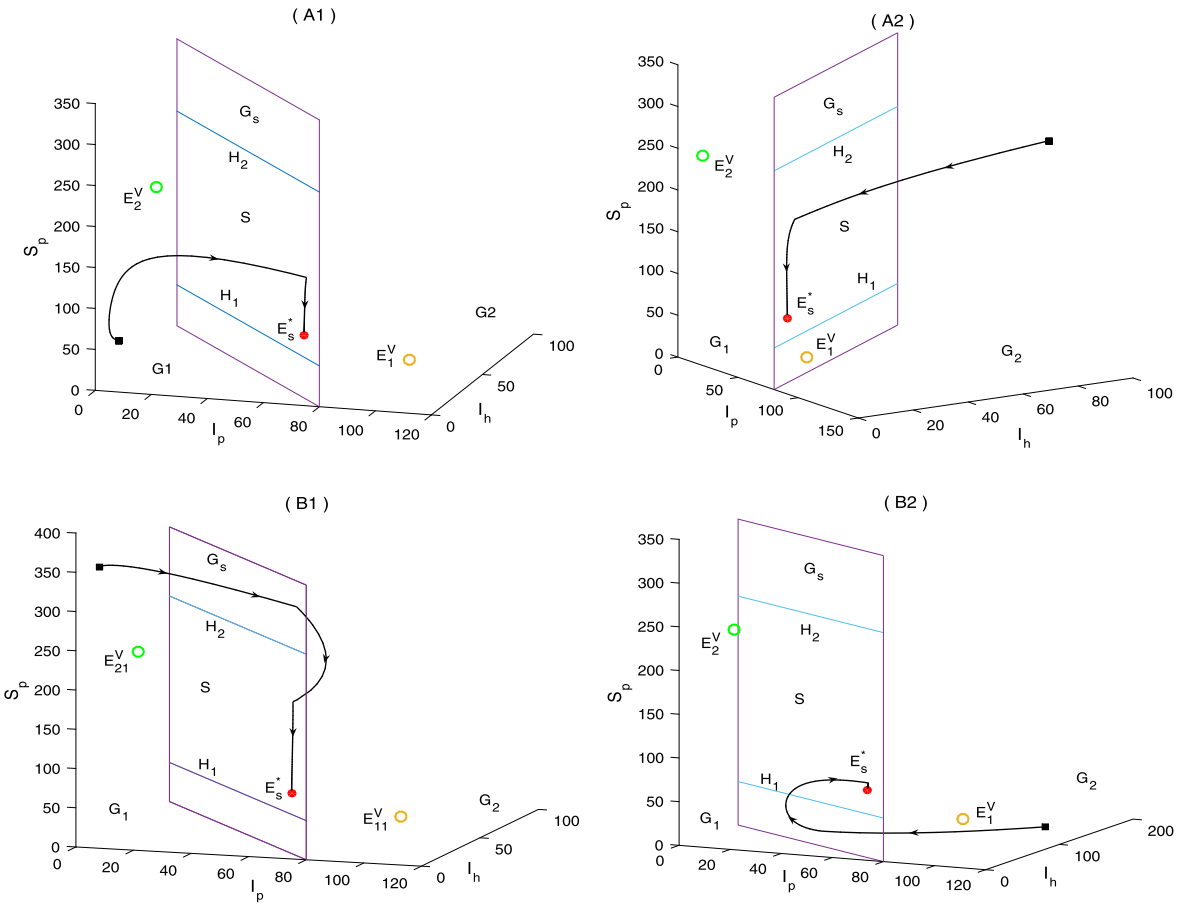


Fig. 5. $E_s^* \in S$ achieves stability with $I_c = 80$.

either one real equilibrium (i.e., E_1^R or E_2^R) or the pseudo-equilibrium (i.e., E_s^*), depending on the threshold level. In particular, Fig. 5 has indicated that the total number can reduce to a previously desired level, when the tolerant threshold is chosen properly, which means that A(H7N9) virus is controllable. Hence, an appropriate threshold policy is crucial for us to trigger the interventions and combat the avian influenza A(H7N9) outbreak.

It is worth mentioning that not only adequate hospital resources and certain government interventions (such as culling of infected poultry and market closure, etc.), but also a good threshold policy is required to suppress the outbreak of A(H7N9) virus or stabilize the infections to an expected level. The results presented in this paper are practical for the control of A(H7N9) virus transmission.

However, our work is just a preliminary exploration of the impacts of some control measures on the A(H7N9) virus transmission, and can be improved in many ways. We have considered the resources occupied by infections with H7N9 virus in system (2). Since the early symptoms of flu are similar to avian influenza, those patients would also put pressure on limited hospital resources, which requires us to further modify the nonlinear recovery rate. In addition, it is interesting and challenging to investigate the global stability of system (14), and we will pay attention to this issue as our future study.

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