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ORIGINAL PAPER

On avian influenza epidemic models with time delay

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Abstract After the outbreak of the first avian influenza A virus (H5N1) in Hong Kong in 1997, another avian influenza A virus (H7N9) crossed the species barrier in mainland China in 2013 and 2014 and caused more than 400 human cases with a death rate of nearly 40 %. In this paper, we take account of the incubation periods of avian influenza A virus and construct a bird-to-human transmission model with different time delays in the avian and human populations combining the survival probability of the infective avian and human populations at the latent time. By analyzing the dynamical behavior of the model, we obtain a threshold value for the prevalence of avian influenza and investigate local and global asymptotical stability of equilibria of the system.

Keywords Avian influenza · Incubation period · Time delay · Basic reproduction number · Local and global asymptotical stability

Introduction

Influenza is a viral infection that affects mainly the nose, throat, bronchi and, occasionally, lungs. Infection usually lasts for about a week and is characterized by sudden onset of high fever, aching muscles, headache and severe malaise, non-productive cough, sore throat and rhinitis (WHO 2015). Avian influenza is an infectious disease of avian caused by type A strains of the influenza virus. Avian influenza viruses with all 16 haemagglutinin (H1–H16) and all 9 neuraminidase (N1–N9) influenza A subtypes in the majority of possible combinations have been isolated from avian species (Alexander 2007).

The avian influenza A H7N9 virus has HA of the H7 subtype and NA of the N9 subtype (CDC 2014; WHO 2014) and is a subgroup among this larger group of H7 viruses. Avian influenza A H7 viruses are a group of influenza viruses that normally circulate among birds, but have been confirmed world-wide in people who have direct contact with infected birds. Most infections have been mild involving only conjunctivitis and mild upper respiratory symptoms (CIDRAP 2013; WOAH 2013). H7N9 had previously been isolated only in birds and no human infections with H7N9 viruses had ever been reported until the 2013 outbreak in mainland China (CIDRAP 2013; WOAH 2013). Most of the reported cases of human infection have resulted in severe respiratory illness (Li et al. 2014) with an unusually high rate for a new infection and high death rate (NHFPC 2015). Data (Bao et al. 2013; Chen et al. 2013) indicate that the novel avian influenza A H7N9 virus was most likely transmitted from the secondary wholesale market to the retail live-poultry market and then to humans.

Mathematical modeling has become an important tool in analyzing the epidemiological characteristics of infectious diseases and can provide useful control measures (Anderson and May 1991; Keeling and Rohani 2008). Iwami et al. (2007) proposed ordinary differential equation (ODE) models to characterize the dynamical behavior of avian influenza between human and avian populations. Since then various models have been used to study different

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aspects of avian influenza transmitted by the H5N1 virus (see Lucchetti et al. 2009; Iwami et al. 2009a, b; Jung et al. 2009; Gumel 2009; Agusto 2013; Ma and Wang 2010; Bourouiba et al. 2011; Gourley et al. 2010; Tuncer and Martcheva 2013). Chong et al. (2014) investigated the effect of half-saturated incidence on the transmission dynamics of avian influenza. Liu et al. (2015), we constructed avian influenza bird-to-human transmission models with different growth laws of the avian population (logistic growth and with Allee effect) and analyzed their dynamical behavior completely. We obtained a threshold value for the prevalence of avian influenza and investigate the local or global asymptotical stability of each equilibrium of these systems by linear analysis or combining Liapunov function method and LaSalles invariance principle, respectively. Moreover, we gave necessary and sufficient conditions for the occurrence of periodic solutions in the avian influenza system with Allee effect of the avian population.

It should be noted that avian influenza virus has an incubation period in both avian and human populations which is the time between infection and symptom onset. Current data for A(H7N9) infection indicate an incubation period ranging from 2 to 8 days, with an average of 5 days (Gao et al 2013). An omission in the above models is the incubation periods of the avian influenza in both avian and human populations. Time delays caused by latent periods in hosts are used to model the mechanisms in the disease dynamics (Cooke and Van Den Driessche 1996; Beretta et al. 2001; Beretta and Takeuchi 1995; Ruan et al. 2008, etc.). Samanta (2010) modified the mathematical model of avian influenza transmission dynamics among birds and humans by introducing time-dependent parameters and distributed time delay due to the intracellular delay between initial infection of a cell and the release of new virus particles on the basis of Iwami et al. (2007) and established some sufficient conditions on the permanence and extinction of the disease and global asymptotic stability of the model. Gourley et al. (2010) formulated a patch model with delay to describe the temporal evolution of the migratory birds, which play a very important role in spreading the avian influenza H5N1 virus globally, within each stopover and to provided some qualitative analysis of the long-term dynamics of such a model. Bourouiba et al. (2011) proposed delayed avian influenza models to investigate the role of migratory birds in the spread of H5N1 avian influenza focusing on the interaction of a migratory bird species with nonmigratory poultry.

Generally speaking, the rate of change of the infectives at time t depends not only on the number at the previous moment $t-\tau$, but also on the probability which the infective individuals survived natural death (with the death rate μ), where τ is the finite time delay representing the incubation period. Inspired by Iwami et al. (2007) and Beretta et al. (2001), we model the incubation periods in both avian and human populations by introducing different time delays and combining the survival probability of the infectives. This is the first time that the incubation periods in both avian and human populations are explicitly modeled. The goal of this article is to study the effect of the time delays on the transmission dynamics of the avian influenza.

The paper is organized as follows. In Sect. 2, we take explicit account of the incubation periods of avian influenza within both the avian and human populations and propose a delayed SI-SIR model. The local and global analyses of the disease-free equilibrium and the endemic equilibrium are presented in Sects. 3 and 4 on basis of the parameter τ_a, respectively. In Sect. 5, we simulate the number of infected human population. A brief discussion about the biological interpretation and conclusion is given in Sect. 6.

The delayed avian influenza model

We always assume that the avian influenza virus does not spread from person to person and mutate. The avian population is classified into two subclasses: susceptible and infective, denoted by $S_a(t)$ and $I_a(t)$, respectively, and the human population is classified into three subclasses: susceptible, infective and recovered, denoted by $S_h(t)$, $I_h(t)$, and $R_h(t)$, respectively. In order to construct the corresponding model, we make the following assumptions:

(a) All new recruitments and newborns of the avian population (the human population) are susceptible, the rate is denoted by Π_a (Π_h respectively).

(b) The avian influenza virus is not contagious from an infected human to a susceptible human. It is only contagious from an infected avian to a susceptible human.

(c) The incidence rate between the susceptible avian and the infective avian depends not only on their numbers at the previous moment $t-\tau_a$, but also on the probability which the infective avian population survived natural death (with the death rate μ_a); similarly, the incidence rate between the susceptible human and the infective avian depends not only on their numbers at the previous moment $t-\tau_h$ but also on the probability which the infective human population survived natural death (with the death rate μ_h). Here $\tau_a \geq 0$ ($\tau_h \geq 0$) is a time delay describing the latency of avian influenza virus on avian population (human population); The incidence rate

between the susceptible human and the infective avian is bilinear.

(d) An infected avian remains in the state of disease and cannot recover, but an infected human can recover and the recovered human has permanent immunity.

Based on the above assumptions, we have the following avian influenza model with incubation periods:

$$\begin{cases} \dfrac{\mathrm{d}S_a}{\mathrm{d}t} = \Pi_a - \mu_a S_a - \beta_a S_a I_a, \\ \dfrac{\mathrm{d}I_a}{\mathrm{d}t} = \beta_a \mathrm{e}^{-\mu_a \tau_a} S_a(t-\tau_a) I_a(t-\tau_a) - (\mu_a + \delta_a) I_a(t), \\ \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = \Pi_h - \mu_h S_h - \beta_h S_h I_a, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = \beta_h \mathrm{e}^{-\mu_h \tau_h} S_h(t-\tau_h) I_a(t-\tau_h) - (\mu_h + \delta_h + \gamma) I_h, \\ \dfrac{\mathrm{d}R_h}{\mathrm{d}t} = \gamma I_h - \mu_h R_h, \end{cases} \tag{1}$$

where β_a (β_h) is the contact rate between the susceptible avian and the infective avian (between the susceptible human and the infective avian); μ_a (μ_h) is the natural death rate of the avian population (the human population); δ_a (δ_h) is the disease-related death rate of the infected avian (the infected human); γ is the recovery rate of the infective human. The time τ_a (τ_h) is the latent period of avian influenza virus on avian population (human population), $S_a(t-\tau_a)$ ($I_a(t-\tau_a)$) represents the numbers of the susceptible (infectious) avian population at time $t-\tau_a$, $S_h(t-\tau_h)$ ($I_a(t-\tau_h)$) represents the numbers of the susceptible (infectious) human population at time $t-\tau_h$ and $\mathrm{e}^{-\mu_a\tau_a}$ ($\mathrm{e}^{-\mu_a\tau_h}$) is the probability which the infected avian (human) survives to time t (with the death rate μ_a, μ_h, respectively), all other parameters are positive.

The initial conditions for system (1) take the form of

$$\begin{cases} S_a(\theta) = \varphi_1(\theta), I_a(\theta) = \varphi_2(\theta), S_h(\theta) = \varphi_3(\theta), I_h(0) = \varphi_4, R_h(0) = \varphi_5, \\ \varphi_i(\theta) \geq 0, \theta \in [-\tau, 0],\ \varphi_i(0) \geq 0, i = 1,2,3;\ \varphi_4 \geq 0, \varphi_5 \geq 0, \end{cases} \tag{2}$$

where $\Phi = (\varphi_1(\theta), \varphi_2(\theta), \varphi_3(\theta), \varphi_4, \varphi_5) \in C([-\tau, 0], \mathbb{R}^5_+)$, the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^5_+$, $\tau = \max\{\tau_a, \tau_h\}$, $\mathbb{R}^5_+ = \{(S_a, I_a, S_h, I_h, R_h) : S_a \geq 0, I_a \geq 0, S_h \geq 0, I_h \geq 0, R_h \geq 0\}$.

It is well known by the fundamental theory of functional differential equations Hale (1977) that system (1) has a unique solution $(S_a, I_a, S_h, I_h, R_h)$ satisfying the initial conditions (2). It is easy to show that all solutions of system (1) with initial conditions (2) are defined on $[0, +\infty)$ and remain positive for all $t \geq 0$.

Define the basic reproduction number by

$$\mathcal{R}_0 = \frac{\Pi_a \beta_a \mathrm{e}^{-\mu_a \tau_a}}{\mu_a(\mu_a + \delta_a)}.$$

We can deduce a unique disease-free equilibrium given by $A(S_a^*, 0, S_h^*, 0, 0)$ from system (1), where

$$S_a^* = \frac{\Pi_a}{\mu_a}, \quad S_h^* = \frac{\Pi_h}{\mu_h}.$$

If $\mathcal{R}_0 > 1$, we can also derive a unique endemic equilibrium given by $B(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$, where

$$S_a^{**} = \frac{(\mu_a + \delta_a)\mathrm{e}^{\mu_a \tau_a}}{\beta_a}, \quad I_a^{**} = \frac{\mu_a}{\beta_a}(\mathcal{R}_0 - 1), \quad S_h^{**} = \frac{\Pi_h}{\beta_h I_a^{**} + \mu_h},$$
$$I_h^{**} = \frac{\beta_h \mathrm{e}^{-\mu_h \tau_h} I_a^{**} S_h^{**}}{\mu_h + \delta_h + \gamma}, \quad R_h^{**} = \frac{\gamma I_h^{**}}{\mu_h}.$$

Remark 2.1 From the expression of $\mathcal{R}_0$ we have

$\mathcal{R}_0 \leq 1 \Leftrightarrow \tau_a \geq \tau_a^*$, $\mathcal{R}_0 > 1 \Leftrightarrow 0 \leq \tau_a < \tau_a^*$, where

$$\tau_a^* = \frac{1}{\mu_a} \ln \frac{\Pi_a \beta_a}{\mu_a(\mu_a + \delta_a)}.$$

It should be noted that the first four equations of system (1) are independent of the fifth equation, so we only need to consider the following subsystem:

$$\begin{cases} \dfrac{\mathrm{d}S_a}{\mathrm{d}t} = \Pi_a - \mu_a S_a - \beta_a S_a I_a, \\ \dfrac{\mathrm{d}I_a}{\mathrm{d}t} = \beta_a \mathrm{e}^{-\mu_a \tau_a} S_a(t-\tau_a) I_a(t-\tau_a) - (\mu_a + \delta_a) I_a(t), \\ \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = \Pi_h - \mu_h S_h - \beta_h S_h I_a, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = \beta_h \mathrm{e}^{-\mu_h \tau_h} S_h(t-\tau_h) I_a(t-\tau_h) - (\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{3}$$

Obviously, system (3) always has a unique disease-free equilibrium $A_1(S_a^*, 0, S_h^*, 0)$ and also has a unique endemic equilibrium $B_1(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$ if $0 \leq \tau_a < \tau_a^*$.

Next, we will study the property of these equilibria.

Local stability of the equilibria

In order to study the local stability of the equilibria, we first recall some known results on the distribution of roots for some transcendental equations.

Lemma 3.1 (Cooke and Grossman 1982) *Let $f(\lambda, \tau) = \lambda + A + B\mathrm{e}^{-\lambda\tau_a}$, where A, B, τ_a are real numbers and $\tau_a \geq 0$. Then, as τ_a varies, the sum of the multiplicities of zeros of f in the open right half-plane can change only if a zero appears on or crosses the imaginary axis.*

Lemma 3.2 (Ruan and Wei 2003) *Let $f(\lambda, \tau) = \lambda^2 + A(\tau_a)\lambda + B(\tau_a)\lambda\mathrm{e}^{-\lambda\tau_a} + C(\tau_a) + D(\tau_a)\mathrm{e}^{-\lambda\tau_a}$, where $A(\tau_a)$, $B(\tau_a)$, $C(\tau_a)$, $D(\tau_a)$, τ_a are real-valued functions and $\tau_a \geq 0$. Then, as τ_a varies, the sum of the multiplicities of*

zeros of f in the open right half-plane can change only if a zero appears on or crosses the imaginary axis.

Theorem 3.3 (1) *The disease-free equilibrium* $A_1(S_a^*, 0, S_h^*, 0)$ *of system* (3) *is locally asymptotically stable if* $\tau_a \geq \tau_a^*$ *and unstable if* $0 \leq \tau_a < \tau_a^*$; (2) *the endemic equilibrium* $B_1(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$ *of system* (3) *is locally asymptotically stable if* $0 \leq \tau_a < \tau_a^*$.

Proof (1) The matrix form of the linearized system of (3) around the disease-free equilibrium $A_1(S_a^*, 0, S_h^*, 0)$ takes the form

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \mathcal{A}_1 X(t) + \mathcal{A}_2 X(t-\tau_a) + \mathcal{A}_3 X(t-\tau_h), \tag{4}$$

where

$$X(t) = \begin{pmatrix} S_a(t) \\ I_a(t) \\ S_h(t) \\ I_h(t) \end{pmatrix}, \quad \mathcal{A}_1 = \begin{pmatrix} -\mu_a & -\beta_a S_a^* & 0 & 0 \\ 0 & -(\mu_a+\delta_a) & 0 & 0 \\ 0 & -\beta_h S_h^* & -\mu_h & 0 \\ 0 & 0 & 0 & -(\mu_h+\delta_h+\gamma) \end{pmatrix},$$

$$\mathcal{A}_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & \beta_a \mathrm{e}^{-\mu_a \tau_a} S_a^* & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad \mathcal{A}_3 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta_h \mathrm{e}^{-\mu_h \tau_h} S_h^* & 0 & 0 \end{pmatrix}.$$

The characteristic equation of the linearized system (4) is

$$(\lambda+\mu_h+\delta_h+\gamma)(\lambda+\mu_h)(\lambda+\mu_a) \\ (\lambda+\mu_a+\delta_a-\beta_a \mathrm{e}^{-\mu_a\tau_a} S_a^* \mathrm{e}^{-\lambda\tau_a}) = 0. \tag{5}$$

It should be noted that the characteristic equation (5) always has three negative eigenvalues given by $\lambda_1 = -(\mu_h+\delta_h+\gamma) < 0$, $\lambda_2 = -\mu_h < 0$, $\lambda_3 = -\mu_a < 0$, then the real parts of other eigenvalues of (5) are decided by the following equation:

$$f(\lambda) = \lambda + \mu_a + \delta_a - \beta_a S_a^* \mathrm{e}^{-(\mu_a+\lambda)\tau_a} = 0. \tag{6}$$

Clearly, if $\tau_a = 0$, then $\mathcal{R}_0 = \mathcal{R}_0^* = \frac{\beta_a S_a^*}{\mu_a+\delta_a}$, the eigenvalue of Eq. (6) is $\lambda = (\mu_a+\delta_a)(\mathcal{R}_0^* - 1) < 0$.

We consider two cases.

Case I $\tau_a > \tau_a^*$ (i.e., $\mathcal{R}_0 < 1$). Suppose $\lambda = i\omega(\omega > 0)$ is a root of (6), separating the real and imaginary parts, we have the following:

$$\begin{cases} \mu_a + \delta_a = (\mu_a+\delta_a)\cos(\omega\tau_a), \\ \omega = -(\mu_a+\delta_a)\sin(\omega\tau_a). \end{cases}$$

Squaring and adding both equations, we have $(\mu_a+\delta_a)^2 + \omega^2 = (\mu_a+\delta_a)^2$, which reduces to $\omega = 0$. It implies that all the eigenvalues have negative real part according to Lemma 3.1, hence the disease-free equilibrium is locally asymptotically stable.

Case II $0 \leq \tau_a < \tau_a^*$ (i.e., $\mathcal{R}_0 > 1$). Obviously, $f(0) = \mu_a + \delta_a - \beta_a S_a^* \mathrm{e}^{-\mu_a\tau_a} = (\mu_a+\delta_a)(1-\mathcal{R}_0) < 0$, and $f(\lambda) \to +\infty$ as $\lambda \to +\infty$, then there must exist a $\lambda_0 > 0$ such that $f(\lambda_0) = 0$. Hence, the characteristic equation (6) has at least one root with positive real part. Therefore, A_1 is unstable.

(2) If $0 \leq \tau_a < \tau_a^*$ (i.e., $\mathcal{R}_0 > 1$), the endemic equilibrium $B_1(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$ exists. The matrix form of linearized system of (3) around the endemic equilibrium B_1 is

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \mathcal{B}_1 X(t) + \mathcal{B}_2 X(t-\tau_a) + \mathcal{B}_3 X(t-\tau_h) \tag{7}$$

with

$$X(t)=\begin{pmatrix} S_a(t)\\ I_a(t)\\ S_h(t)\\ I_h(t)\end{pmatrix},\quad \mathcal{B}_1=\begin{pmatrix} -(\beta_a I_a^{**}+\mu_a) & -\beta_a S_a^{**} & 0 & 0\\ 0 & -(\mu_a+\delta_a) & 0 & 0\\ 0 & -\beta_h S_h^{**} & -(\beta_h I_a^{**}+\mu_h) & 0\\ 0 & 0 & 0 & -(\mu_h+\delta_h+\gamma)\end{pmatrix},$$

$$\mathcal{B}_2=\begin{pmatrix} 0 & 0 & 0 & 0\\ \beta_a e^{-\mu_a\tau_a} I_a^{**} & \beta_a e^{-\mu_a\tau_a} S_a^{**} & 0 & 0\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\end{pmatrix},\quad \mathcal{B}_3=\begin{pmatrix} 0 & 0 & 0 & 0\\ 0 & & 0 & 0\\ 0 & 0 & 0 & 0\\ 0 & \beta_h e^{-\mu_h\tau_h} S_h^{**} & \beta_h e^{-\mu_h\tau_h} I_a^{**} & 0\end{pmatrix}.$$

The characteristic equation of (7) is

$$(\lambda+\mu_h+\delta_h+\gamma)(\lambda+\beta_h I_a^{**}+\mu_h)(\lambda^2+A\lambda+B\lambda e^{-\lambda\tau_a} + C+De^{-\lambda\tau_a})=0 \tag{8}$$

where $A(\tau_a)=\mu_a+\delta_a+\mu_a\mathcal{R}_0$, $B=-(\mu_a+\delta_a)$, $C(\tau_a)=(\mu_a+\delta_a)\mu_a\mathcal{R}_0$, $D=-\mu_a(\mu_a+\delta_a)$.

Since the characteristic equation (8) always has two negative eigenvalues $\lambda=-(\mu_h+\delta_h+\gamma)<0$, $\lambda=-(\beta_h I_a^{**}+\mu_h)<0$, the real parts of other eigenvalues are decided by the equation

$$\lambda^2+A(\tau_a)\lambda+B\lambda e^{-\lambda\tau_a}+C(\tau_a)+De^{-\lambda\tau_a}=0. \tag{9}$$

If $\tau_a=0$, Eq. (9) reduces to

$$\lambda^2+\mu_a\mathcal{R}_0\lambda+\mu_a(\mu_a+\delta_a)(\mathcal{R}_0-1)=0, \tag{10}$$

where $\mathcal{R}_0=\mathcal{R}_0^*=\frac{\beta_a S_a^*}{\mu_a+\delta_a}$, which is independent of the parameter τ_a. Obviously, all the eigenvalues of system (10) have negative real parts if $\mathcal{R}_0>1$.

If $\tau_a>0$, let $\lambda=i\omega, \omega>0$, be a root of (9), separating real and imaginary parts, we have the following:

$$\begin{cases} -\omega^2+C=-B\omega\sin(\omega\tau_a)-D\cos(\omega\tau_a),\\ A\omega=-B\omega\cos(\omega\tau_a)+D\sin(\omega\tau_a).\end{cases}$$

Squaring and adding both equations we have

$$\omega^4+Q_1\omega^2+Q_2=0, \tag{11}$$

where $Q_1=A^2-2C-B^2=\mu_a^2\mathcal{R}_0^2>0, Q_2=C^2-D^2=\mu_a^2(\mu_a+\delta_a)^2(\mathcal{R}_0^2-1)$. If $\mathcal{R}_0>1$, then $Q_2>0$, hence the Eq. (11) has no positive root, which implies that all eigenvalues of Eq. (10) have negative real parts according to Lemma 3.2. Hence, the endemic equilibrium B_1 is locally asymptotically stable for any $\tau_a\in[0,\tau_a^*)$.□

Remark 3.4 If $\tau_a=\tau_a^*$ (i.e., $\mathcal{R}_0=1$), the endemic equilibrium B_1 coincides with the disease-free equilibrium A_1 which is a saddle-node and is locally asymptotically stable for positive trajectories.

Global stability of the equilibria

In order to prove the global stability of the equilibria, we first consider the avian-only subsystem:

$$\begin{cases} \dfrac{dS_a}{dt}=\Pi_a-\mu_a S_a-\beta_a S_a I_a,\\ \dfrac{dI_a}{dt}=\beta_a e^{-\mu_a\tau_a} S_a(t-\tau_a)I_a(t-\tau_a)-(\mu_a+\delta_a)I_a(t).\end{cases} \tag{12}$$

It should be noted that the avian-only system (12) is independent of the full system (1). System (12) always has a unique disease-free disease $A_a(S_a^*,0)$ and a unique endemic equilibrium $B_a(S_a^{**},I_a^{**})$ if $0\le\tau_a<\tau_a^*$. Similarly, we can derive that the disease-free disease A_a is locally asymptotically stable if $\tau_a\ge\tau_a^*$ and the endemic equilibrium B_a is locally asymptotically stable if $0\le\tau_a<\tau_a^*$. In fact, the avian-only system (12) has been studied by Huang et al. (2010) and we recall one of their results here.

Lemma 4.1 (Huang et al. 2010) (1) *If* $\tau_a \geq \tau_a^*$, *then the disease-free equilibrium* A_a *is globally asymptotically stable;* (2) *if* $0 \leq \tau_a < \tau_a^*$, *then the endemic equilibrium* B_a *is globally asymptotically stable.*

Now we apply Lemma 4.1 to prove the global stability of the disease-free equilibrium A_1 and the endemic equilibrium B_1 of system (3).

Theorem 4.2 (1) *If* $\tau_a \geq \tau_a^*$, *then the disease-free equilibrium* $A_1(S_a^*, 0, S_h^*, 0)$ *of system* (3) *is globally asymptotically stable;* (2) *if* $0 \leq \tau_a < \tau_a^*$, *then the endemic equilibrium* $B_1(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$ *of system* (3) *is globally asymptotically stable.*

Proof

1. According to Lemma 4.1, the disease-free equilibrium A_a of system (12) is globally asymptotically stable if $\tau_a \geq \tau_a^*$. To prove the global stability of the equilibrium A_1 of system (3), we only need to consider system (3) with the avian components already at the endemic steady state, given by

$$\begin{cases} \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = \Pi_h - \mu_h S_h, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = -(\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{13}$$

   It is clear that $S_h \to S_h^*$ and $I_h \to 0$ when $t \to \infty$. Hence, the disease-free equilibrium A_1 of system (3) is globally asymptotically stable.
2. Similarly, by Lemma 4.1, the endemic equilibrium B_a of system (12) is globally asymptotically stable if $0 \leq \tau_a < \tau_a^*$. To prove the global stability of the equilibrium B_1 of system (3), we once again consider system (3) with the avian components already at the endemic steady state, given by

$$\begin{cases} \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = \Pi_h - \beta_h I_a^{**} S_h - \mu_h S_h, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = \beta_h \mathrm{e}^{-\mu_h \tau_h} I_a^{**} S_h(t - \tau_h) - (\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{14}$$

   By the first equation of system (14), we can easily obtain that $S_h \to S_h^{**}$ when $t \to \infty$. Then, $S_h(t - \tau_h) \to S_h^{**}$, hence $I_h \to I_h^{**}$ when $t \to \infty$ from the second equation of system (14). Hence, the endemic equilibrium B_1 of system (3) is globally asymptotically stable. □

Corollary 4.3

1. *If* $\tau_a \geq \tau_a^*$, *then the disease-free equilibrium* $A(S_a^*, 0, S_h^*, 0, 0)$ *of system* (1) *is globally asymptotically stable*;
2. If $0 \leq \tau_a < \tau_a^*$, *then the endemic equilibrium* $B(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$ *of system* (1) *is globally asymptotically stable.*

Remark 4.4 The parameter τ_a (the time delay describing the latent period in the avian population) is also a threshold value that determines whether the avian influenza disappears or not. If $\tau_a \geq \tau_a^*$, the avian influenza disappears, but if $0 \leq \tau_a < \tau_a^*$, the avian influenza exists and becomes an endemic disease.

Remark 4.5 The parameter τ_h (the time delay describing the latent period in the human population) does not influence the stability of the equilibria.

Numerical simulations

In this section, we investigate the influence of the time delays on the number of infected humans by performing some numerical simulations.

We choose parameters $\Pi_a = 350$, $\mu_a = 0.01$, $\beta_a = 7 * 10^{-6}$, $\delta_a = 0.05$, $\Pi_h = 100$, $\beta_h = 8 \times 10^{-7}$, $\mu_h = 3.91 \times 10^{-3}$, $\delta_h = 0.3$, $\gamma = 0.01$. The initial values are fixed at $(S_a(0), I_a(0), S_h(0), I_h(0), R_h(0)) = (20{,}000, 1111, 20{,}000, 0, 0)$. With these parameters, the threshold value of time delay τ_a is $\tau_a^* \approx 141$.

If $\tau_a < \tau_a^*$, the endemic disease is prevalent, the solution $I_h(t)$ is asymptotically stable and converges to the endemic state value. By comparison, when we choose time delays as $\tau_a = 6$, $\tau_h = 4$ and $\tau_a = 0$, $\tau_h = 0$, respectively, we observe that the time delay indeed decreases the number of infected humans slightly (see Fig. 1). When the time delay

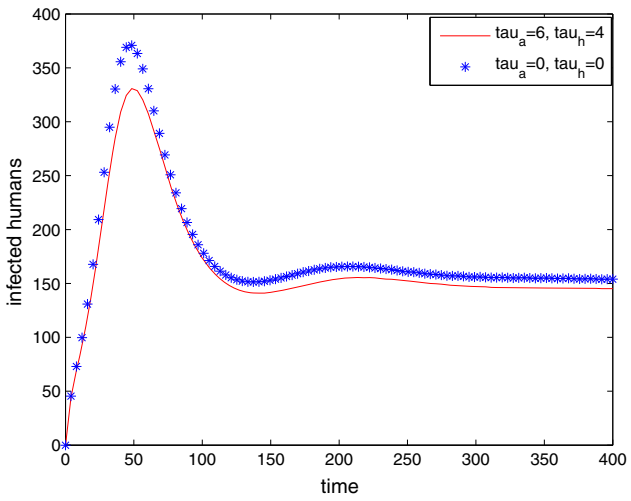

Fig. 1 The plot of $I_h(t)$ with or without time delay when τ_a is less than τ_a^*. The solution $I_h(t)$ is asymptotically stable and converges to the endemic state value

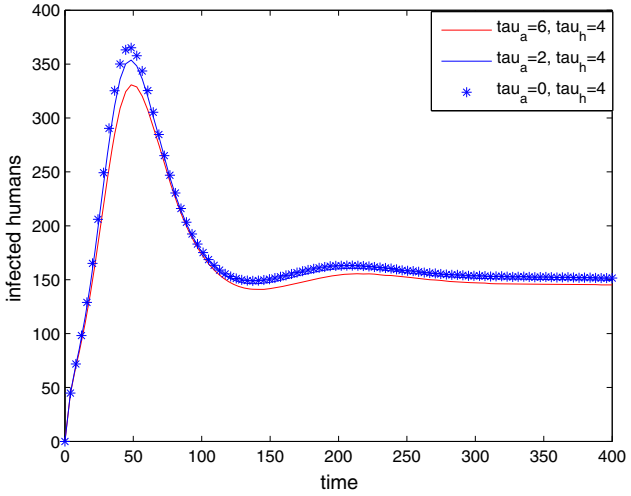

Fig. 2 The plots reveal that different values of the time delay τ_a influence the change of $I_h(t)$, where τ_a is less than τ_a^* and $\tau_h = 4$. The solution $I_h(t)$ is asymptotically stable and converges to the endemic state value

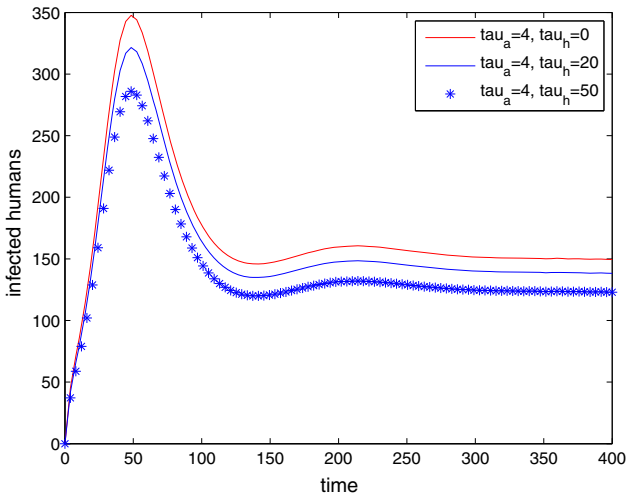

Fig. 3 The plots exhibit that different values of the time delay τ_h influence the changes of $I_h(t)$, where $\tau_a = 4 < \tau_a^*$. The solution $I_h(t)$ is asymptotically stable and converges to the endemic state value

τ_h is fixed and the time delay τ_a takes different values, we can see that the number of infected humans decreases when the time delay τ_a increases (see Fig. 2). When the time delay τ_a is fixed and the time delay τ_h takes different values, we observe that the number of infected humans also decreases when the time delay τ_h increases (see Fig. 3).

If $\tau_a > \tau_a^*$, the endemic disease disappears. For example, when the time delay τ_a is fixed at 142, we can see that the number of infected humans decreases with time delay τ_h increasing and the solution $I_h(t)$ is asymptotically stable and converges to the disease-free state value (see Fig. 4).

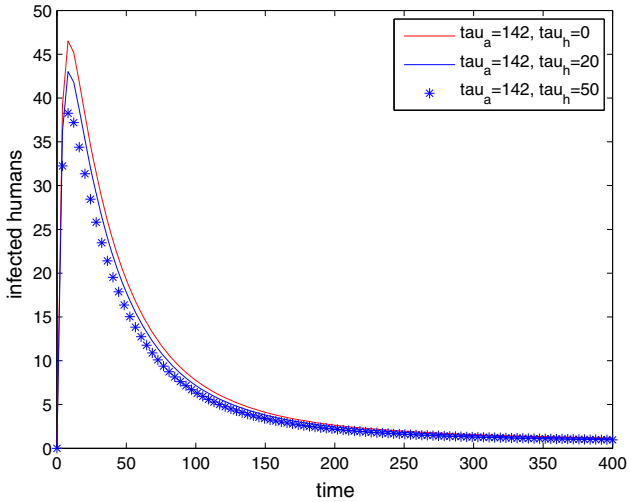

Fig. 4 The plots indicate that different values of the time delay τ_h influence the changes of $I_h(t)$ with $\tau_a = 142 > \tau_a^*$. The solution $I_h(t)$ is asymptotically stable and converges to the disease-free state value

Discussion

In this paper, to study the transmission dynamics of avian influenza from birds to humans we considered the incubation periods of avian influenza within both the human and the avian populations and constructed a delay differential equation model with different time delays. We obtained a threshold value for the prevalence of avian influenza and discussed the local and global asymptotical stability of each equilibrium of the delay system. Our results indicate that the asymptotic dynamics of the model are completely determined by the threshold value τ_a^* (or the basic reproduction number): the disease-free equilibrium exists and is locally asymptotically stable if $\tau_a \geq \tau_a^*$ (or the basic reproduction number is no less than the unity); the disease-free equilibrium becomes unstable and the endemic equilibrium exists and is locally asymptotically stable if $0 \leq \tau_a < \tau_a^*$ (or the basic reproduction number is greater than the unity). In other words, the avian influenza disappears if $\tau_a \geq \tau_a^*$, but is prevalent and becomes endemic disease if $0 \leq \tau_a < \tau_a^*$. Furthermore, we proved the globally asymptotic stability of the positive steady state for all delay values τ_a as long as $\tau_a < \tau_a^*$ (i.e., the reproduction number is greater than one). Our numerical simulations (see Figs. 1, 2, 3) confirmed the result as well. Since the incubation period of avian influenza virus in avian population is much shorter than the threshold value, our theory result may explain the present avian influenza's prevalence.

Our results demonstrate that transmission dynamics of the avian influenza is completely determined by the incubation period in birds. It is interesting to note that the incubation period of the avian virus in human population does not affect the stability of the equilibria and thus the

transmission and outbreak of the disease, since infected humans do not spread the virus any further. However, the theoretical analyses and numerical simulations indicate that prolong both the incubation periods within the human and avian populations could reduce the numbers of the infected human and may help to control the disease.

The roles of wild birds and domestic birds in the transmission of the H5N1 avian influenza are different and mathematical models have been proposed to include both types of birds (Bourouiba et al. 2011; Gourley et al. 2010; Lucchetti et al. 2009; Tuncer and Martcheva 2013). It will be very interesting to include both wild birds and domestic birds in modeling the bird-to-human transmission of the H7N9 avian influenza; we leave this for future consideration.

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