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Nonlinear dynamics of avian influenza epidemic models☆

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ABSTRACT

Avian influenza is a zoonotic disease caused by the transmission of the avian influenza A virus, such as H5N1 and H7N9, from birds to humans. The avian influenza A H5N1 virus has caused more than 500 human infections worldwide with nearly a 60% death rate since it was first reported in Hong Kong in 1997. The four outbreaks of the avian influenza A H7N9 in China from March 2013 to June 2016 have resulted in 580 human cases including 202 deaths with a death rate of nearly 35%. In this paper, we construct two avian influenza bird-to-human transmission models with different growth laws of the avian population, one with logistic growth and the other with Allee effect, and analyze their dynamical behavior. We obtain a threshold value for the prevalence of avian influenza and investigate the local or global asymptotical stability of each equilibrium of these systems by using linear analysis technique or combining Liapunov function method and LaSalle's invariance principle, respectively. Moreover, we give necessary and sufficient conditions for the occurrence of periodic solutions in the avian influenza system with Allee effect of the avian population. Numerical simulations are also presented to illustrate the theoretical results.

"Dedicated to our friend Dr. Dingbian Qian, Professor in the School of Mathematical Sciences at Soochow University, Suzhou, Jiangsu Province, China, who was critically infected by the H7N9 avian influenza virus in April 2013, fearfully stayed in the intensive care unit for more than two months, and miraculously recovered."

1. Introduction

Influenza A viruses are divided into subtypes based on two proteins on the surface of the virus: hemagglutinin (HA) and neuraminidase (NA). For example, the avian influenza A virus designation of H7N9 identifies it as having HA of the H7 subtype and NA of the N9 subtype (CDC [8]). Avian influenza A H7 viruses are a group of influenza viruses that normally circulate among birds. H7 influenza infections in humans are uncommon, 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 [9] and OIE [59]). Although some H7 viruses (e.g. H7N2, H7N3 and H7N7) have occasionally been found to infect humans, H7N9 had previously been isolated only in birds, with outbreaks reported in the Netherlands, Japan, and the United States. Until the 2013 outbreak in China, no human infections with H7N9 viruses had ever been reported (CIDRAP [9] and OIE [59]).

Differing from the highly pathogenic avian influenza virus H5N1, the H7N9 virus does not induce clinical signs in poultry and is classified as a low pathogenicity avian influenza virus (LPAIV) [46]. However, the virus can infect humans and most of the reported cases of human H7N9 infection have resulted in severe respiratory illness [39]. From March 31 to August 31, 2013, 134 cases had been reported in mainland China, resulting in 45 deaths (NHFPC [45]), an unusually high rate for a new infection and high death rate. Genetic characterization of H7N9 shows that the virus resulted from the recombination of genes between several parent viruses noted in poultry and wild birds in Asia [37]. Evidence suggests that the gene that codes for HA has its origin in ducks and the gene that codes for NA has its origin with ducks and probably also wild birds. The HA genes were circulating in the East Asian flyway in both wild birds and ducks, while the NA genes were introduced from European lineages and transferred to ducks in China by wild birds through migration along the East Asian flyway [40]. There is very little information on the H7N9 virus in wild birds to access their potential as source of domestic poultry and human infection. The mode of H7N9 virus transmission between avian species remains unknown, but various wild birds have been im-

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plicated as a source of transmission. Jones et al. [31] showed that society finches, zebra finches, sparrows, and parakeets are susceptible to H7N9 virus and shed virus into water. Jones et al. [32] further demonstrated that interspecies transmission of H7N9 virus occurs readily between society finches and bobwhite quail but only sporadically between finches and chickens, and transmission occurs through shared water. Since the experimental data of Pantin-Jackwood et al. [46] showed that quail and chickens are susceptible to infection, shed large amounts of virus, and are likely important in the spread of the virus to humans, it is therefore conceivable that passerine birds may serve as vectors for transmission of H7N9 virus to domestic poultry [32]. Data 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 [4,11] . To control the outbreak, from late April to early June in 2013, local authorities of the provinces and municipalities, such as Jiangsu, Shanghai, and Zhejiang, temporarily closed the retail live-poultry markets which proved to be an effective control measure. There were no reported cases in the summer and fall 2013. However, the virus came back in November 2013 and again in November 2014 and November 2015. In fact, the second outbreak (from November 2013 to May 2014), the third outbreak (from November 2014 to June 2015), and the fourth outbreak (from November 2015 to June 2016) caused 130 human cases with 35 deaths, 216 confirmed human cases with 99 deaths, and 110 confirmed human cases with 44 deaths, respectively (NHFPC [45]).

Mathematical modeling has become an important tool in analyzing the epidemiological characteristics of infectious diseases and can provide useful control measures [3,36] . In 2007, Iwami et al. [28] 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 aspects of avian influenza transmitted by the H5N1 virus. Lucchetti et al. [43] developed an ODE model to describe the transmission dynamics of the avian influenza A virus from birds to humans and used the model to fit the human cases reported by the WHO. Iwami et al. [29] investigated relations between the evolution of virulence and the effectiveness of pandemic control measures after the emergence of mutant avian influenza. Jung et al. [33] extended the study of Iwami et al. [28] for the prevention of the pandemic influenza to evaluate the time-dependent optimal prevention policies, which were associated with elimination policy and quarantine policy, considering its execution cost. Iwami et al. [30] designed and analyzed a deterministic patch-structured model in heterogeneous areas (with or without vaccination) illustrating transmission of vaccine-sensitive and vaccine-resistant strains during a vaccination program. Gumel [24] incorporated the dynamics of both wild and domestic birds and the isolation of individuals with symptoms of both the avian and mutant strains. Ma and Wang [44] formulated a discrete-time model with reproductive and overwintering periods to assess the impact of avian influenza transmission in poultry. Bourouiba et al. [5] investigated the role of migratory birds in the spread of H5N1 avian influenza among birds by considering a system of delay differential equations for the numbers of birds on patches, where the delays represent the flight times between patches. See also Gourley et al. [22]. Tuncer and Martcheva [52] constructed several bird-to-human transmission models to investigate the mechanisms for the seasonality in avian influenza H5N1 transmission. Wang and Wu [55] constructed a periodic systems of delay differential equations modeling the spread of avian influenza by migratory birds between the refuge ground and the summer breeding site. Chong and Smith [12] proposed two Filippov models with threshold policy to determine culling of infected birds and quarantine.

Considering the fact that the domesticated birds are probably the important infectious source for human population, Iwami et al. [28] assumed that the avian populations are subject to the rule of constant growth. But the possibility that migrant birds are viewed as the original infection source is the largest [62]). Migratory hosts may transmit pathogens to new areas, leading to the exposure and potential infection of new host species [1]. Resident hosts, immunologically naive to these novel pathogens, may subsequently act as local amplifiers. For example, the global spread of West Nile Virus is considered to be greatly facilitated by migratory birds introducing the virus to other wildlife and humans in many parts of the world [47]. It is well-known that the logistic growth, where the rate of reproduction is proportional to both the existing population and the amount of available resources and increases quickly at first and then more slowly as the population approaches its carrying capacity, is more reasonable than the constant growth for the wildlife birds, including migratory and resident birds. Allee effect, a phenomenon in which the reproduction rate of a population decreases when its density drops below a certain critical level, was firstly observed by Allee [2] about aggregation and associated cooperative and social characteristics among members of a species in animal populations. The phenomenon in biology is called strong Allee effect, which is particularly relevant to endangered species and small or invasive populations. Habitat destruction, spread of alien species, overharvest, pollution (including siltation), and disease (caused by either alien or native pathogens) are responsible for endangering species [57]. The study of Serrano et al. [49] on Allee effect in colonial birds demonstrates that Allee effect, that is positive density dependence, appears to be the cause of the evolution of dispersal behavior. Skagen and Yackel [50] observed that population density of small bird populations is correlated positively with both per capita fecundity and population growth rate due to the Allee effect.

It has been reported that some wild species, such as the African wild dog *Lycaon pictus* [6] and the island fox *Urocyon littoralis* [13], suffer from both disease and an Allee effect. Diseases can drive populations to low densities as a result of Allee effect, in particular for diseases having reservoirs or affecting populations that are at small pre-epidemic sizes [18] or for native island species exposed to new pathogens [56]. In wild populations of Serins (*Serinus serinus*), Senar and Conroy [48] reported that avian pox infections were very virulent and survival rates of infected birds were half that of uninfected ones. Recently, great attention has been paid to the theoretical modeling and analysis of the joint interplay of infectious disease and Allee effects (see [20,21,26,27,34,35,51] , and the references cited therein). On one hand, it has been observed that recurrent infectious disease outbreaks tend to enhance the deleterious role of Allee effects within diseases capable of inducing reductions in host fitness [35]. On the other hand, sustained oscillations can occur induced by Allee effects via bifurcations [7,26,35,51] .

In this paper we construct two simplified avian-human epidemic models according to different growth rates of the avian population, namely, with avian population being subject to logistic growth and Allee effect. 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/removed, 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:

(1) The net growth rate of the susceptible avian population is described by the function $g(S_a)$, where $g(\cdot) : \mathbb{R}_+ \to \mathbb{R}$ is continuous, $\mathbb{R} = (-\infty, \infty), \mathbb{R}_+ = [0, \infty)$;
(2) All new recruitments and newborns of the human population are susceptible, the rate is denoted by Π_h;

(3) The avian influenza virus is not contagious from an infective human to a susceptible human. It is only contagious from an infective avian to a susceptible human;
(4) An infected avian keeps in the state of disease and cannot recover, but an infected human can recover and the recovered human has permanent immunity;
(5) The incidence rate between the susceptible avian and the infective avian is bilinear. The incidence rate between the susceptible human and the infective avian is also bilinear.

Based on the above assumptions, we have the following SI-SIR avian influenza model:

$$\begin{cases} \dfrac{dS_a}{dt} = g(S_a) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a + \delta_a) I_a \\ \dfrac{dS_h}{dt} = \Pi_h - \beta_h S_h I_a - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_h S_h I_a - (\mu_h + \delta_h + \gamma) I_h \\ \dfrac{dR_h}{dt} = \gamma I_h - \mu_h R_h, \end{cases} \tag{1}$$

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; γ is the recovery rate of the infective human. If the susceptible avian population is subject to the logistic growth, then

$$g(S_a) = r_a S_a \left(1 - \frac{S_a}{K_a}\right), \tag{2}$$

where r_a and K_a are the intrinsic growth rate and maximal carrying capacity of the avian population, respectively. If the susceptible avian population is subject to Allee effect, then

$$g(S_a) = r_a S_a \left(1 - \frac{S_a}{M_a}\right)\left(\frac{S_a}{m_a} - 1\right), \tag{3}$$

where r_a, M_a, and $m_a (m_a < M_a)$ are the intrinsic growth rate, the maximal carrying capacity and the critical carrying capacity of the avian population, respectively. We assume that all parameters are positive.

We will analyze the global asymptotical stability of these systems and compare the sizes of the basic reproduction numbers for both cases. The paper is organized as follows. The global analysis of avian-human epidemic models in which the avian population is subject to the rule of logistic growth law and Allee effect is discussed in Sections 2 and 3, respectively, where the human population is always subject to the rule of constant growth. In Section 4, we compare the sizes of two basic reproduction numbers and provide numerical simulations of the model for both cases. A brief discussion about the biological interpretations and conclusions is given in the last section.

2. Model (1) with logistic growth for avian population

2.1. The model

If the net growth rate of the avian population is subject to the logistic growth law in system (1), then we obtain the following SI-SIR model:

$$\begin{cases} \dfrac{dS_a}{dt} = r_a S_a \left(1 - \dfrac{S_a}{K_a}\right) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a + \delta_a) I_a \\ \dfrac{dS_h}{dt} = \Pi_h - \beta_h I_a S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_h I_a S_h - (\mu_h + \delta_h + \gamma) I_h \\ \dfrac{dR_h}{dt} = \gamma I_h - \mu_h R_h, \end{cases} \tag{4}$$

where r_a (K_a) is the intrinsic growth rate (the maximal carrying capacity) of the avian population, the assumptions and the meanings of the other parameters are the same as in (1). System (4) has a unique solution satisfying initial conditions in $\mathbb{R}^5_+$ which is the positively invariant set for system (4).

We can deduce two disease-free equilibria given by $A(0, 0, S_h^*, 0, 0)$ and $B(K_a, 0, S_h^*, 0, 0)$ from system (4), where $S_h^* = \frac{\Pi_h}{\mu_h}$.

Following the definition and computation procedure in Diekmann et al. [19] and van den Driessche and Watmough [54], we can rewrite system (4) as follows:

$$\frac{dX}{dt} = \mathscr{F} - \mathscr{V}$$

where,

$$X(t) = \begin{pmatrix} I_a(t) \\ I_h(t) \\ S_a(t) \\ S_h(t) \\ R_h(t) \end{pmatrix}, \quad \mathscr{F} = \begin{pmatrix} \beta_a I_a S_a \\ \beta_h I_a S_h \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

$$\mathscr{V} = \begin{pmatrix} (\mu_a + \delta_a) I_a \\ (\mu_h + \delta_h + \gamma) I_h \\ \beta_a I_a S_a - \beta_a S_a \left(1 - \dfrac{S_a}{K_a}\right) \\ \mu_h S_h + \beta_h I_a S_h - \Pi_h \\ \mu_h R_h - \gamma I_h \end{pmatrix},$$

then,

$$F = \begin{pmatrix} \beta_a K_a & 0 \\ \beta_h S_h^* & 0 \end{pmatrix}, \; V = \begin{pmatrix} \mu_a + \delta_a & 0 \\ 0 & \mu_h + \delta_h + \gamma \end{pmatrix},$$

$$FV^{-1} = \begin{pmatrix} \dfrac{\beta_a K_a}{\mu_a + \delta_a} & 0 \\ \dfrac{\beta_h S_h^*}{\mu_a + \delta_a} & 0 \end{pmatrix}.$$

Hence, we derive the basic reproduction number as follows

$$\mathcal{R}_{0,1} = \frac{K_a \beta_a}{\mu_a + \delta_a}.$$

If $\mathcal{R}_{0,1} > 1$, we can also derive a unique endemic equilibrium given by $C(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$, where

$$S_a^{**} = \frac{\mu_a + \delta_a}{\beta_a}, \quad I_a^{**} = \frac{r_a(\mu_a + \delta_a)}{K_a \beta_a^2}(\mathcal{R}_{0,1} - 1),$$

$$S_h^{**} = \frac{\Pi_h}{\beta_h I_a^{**} + \mu_h}, \quad I_h^{**} = \frac{\beta_h I_a^{**} S_h^{**}}{\mu_h + \delta_h + \gamma}, \quad R_h^{**} = \frac{\gamma I_h^{**}}{\mu_h}.$$

Before analyzing the dynamical behavior of the full model (4), we study the dynamical behavior of the avian-only subsystem.

2.2. Analysis of the avian-only subsystem

Consider the avian-only subsystem, given by the first two equations of system (4), as follows:

$$\begin{cases} \dfrac{dS_a}{dt} = r_a S_a\left(1-\dfrac{S_a}{K_a}\right) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a+\delta_a) I_a. \end{cases} \tag{5}$$

It should be noted that the above avian system is independent of the human system. Clearly, $\mathbb{R}^2_+$ is the positively invariant attracting set of subsystem (5). Next we will discuss the dynamical behavior of solutions to subsystem (5) in $\mathbb{R}^2_+$.

2.2.1. Local stability of the avian-only subsystem (5)

The avian-only subsystem (5) always has two disease-free equilibria given by $A_a(0, 0)$ and $B_a(K_a, 0)$. If $\mathcal{R}_{0,1} > 1$, the system also has a unique endemic equilibrium given by $C_a(S_a^{**}, I_a^{**})$.

Lemma 2.1. *(i) The disease-free equilibrium A_a is always unstable; (ii) If $\frac{\mu_a+\delta_a}{\beta_a} \geq K_a$ (i.e., $\mathcal{R}_{0,1} \leq 1$), then the disease-free equilibrium B_a is locally asymptotically stable for positive trajectories; (iii) If $0 < \frac{\mu_a+\delta_a}{\beta_a} < K_a$ (i.e., $\mathcal{R}_{0,1} > 1$), then the disease-free equilibrium B_a is unstable but the endemic equilibrium C_a is locally asymptotically stable.*

Proof. The characteristic equation of the Jacobian matrix at an arbitrary equilibrium (S_a, I_a) is

$$\left(\lambda - \left(r_a - \frac{2r_a}{K_a}S_a - \beta_a I_a\right)\right)(\lambda - (\beta_a S_a - \mu_a - \delta_a)) + \beta_a^2 S_a I_a = 0.$$

(i) If $(S_a, I_a) = (0, 0)$, the eigenvalues are $\lambda_1 = r_a > 0$, $\lambda_2 = -(\mu_a+\delta_a) < 0$. Hence, the equilibrium A_a is always unstable.

(ii) If $\mathcal{R}_{0,1} < 1$ and $(S_a, I_a) = (K_a, 0)$, the eigenvalues are $\lambda_1 = -r_a < 0$, $\lambda_2 = (\mu_a+\delta_a)(\mathcal{R}_{0,1}-1) < 0$. Hence, the equilibrium B_a is locally asymptotically stable.

(iii) If $\mathcal{R}_{0,1} > 1$ and $(S_a, I_a) = (K_a, 0)$, the eigenvalues are $\lambda_1 = -r_a < 0$, $\lambda_2 = (\mu_a+\delta_a)(\mathcal{R}_{0,1}-1) > 0$. Hence, the equilibrium B_a is unstable; If $\mathcal{R}_{0,1} > 1$ and $(S_a, I_a) = (S_a^{**}, I_a^{**})$, the above characteristic equation becomes

$$\lambda^2 + \frac{r_a}{K_a}S_a^{**}\lambda + \beta_a^2 S_a^{**} I_a^{**} = 0.$$

Since $S_a^{**} > 0$ and $I_a^{**} > 0$ if $\mathcal{R}_{0,1} > 1$, all eigenvalues have negative real parts. Hence, the equilibrium C_a is locally asymptotically stable. □

Remark 2.2. If $\mathcal{R}_{0,1} = 1$, then the endemic equilibrium C_a coincides with the disease-free equilibrium B_a which is a saddle-node and is locally asymptotically stable for positive trajectories.

2.2.2. Global stability of the avian-only subsystem (5)

Lemma 2.3. *(i) If $\frac{\mu_a+\delta_a}{\beta_a} \geq K_a$ (i.e., $\mathcal{R}_{0,1} \leq 1$), then the disease-free equilibrium B_a is globally asymptotically stable for positive trajectories; (ii) If $0 < \frac{\mu_a+\delta_a}{\beta_a} < K_a$ (i.e., $\mathcal{R}_{0,1} > 1$), then the endemic equilibrium C_a is globally asymptotically stable.*

Proof. (i) If $\mathcal{R}_{0,1} \leq 1$, we choose a Liapunov function as follows

$$V_1 = S_a - K_a - K_a \ln\frac{S_a}{K_a} + I_a.$$

Then we have

$$\begin{aligned} \left.\frac{dV_1}{dt}\right|_{(5)} &= (S_a - K_a)\left(r_a - \frac{r_a S_a}{K_a} - \beta_a I_a\right) + \beta_a S_a I_a - (\mu_a+\delta_a) I_a \\ &= \frac{-r_a(S_a-K_a)^2}{K_a} - \beta_a I_a (S_a - K_a) + \beta_a S_a I_a - (\mu_a+\delta_a) I_a \\ &= \frac{-r_a(S_a-K_a)^2}{K_a} + \beta_a K_a I_a - (\mu_a+\delta_a) I_a \\ &= \frac{-r_a(S_a-K_a)^2}{K_a} + I_a(\mu_a+\delta_a)(\mathcal{R}_{0,1}-1) \leq 0. \end{aligned}$$

Since $\{(S_a, I_a) \in \mathbb{R}^2_+ : \frac{dV_1}{dt} = 0\} = \{(S_a, I_a) \in \mathbb{R}^2_+ : S_a = K_a, I_a = 0\} = \{B_a\}$, according to LaSalle's invariance principle (Hale [25]), the equilibrium B_a is globally asymptotically stable for positive trajectories.

(ii) If $\mathcal{R}_{0,1} > 1$, we choose a Liapunov function

$$V_2 = \left(S_a - S_a^{**} - S_a^{**}\ln\frac{S_a}{S_a^{**}}\right) + \left(I_a - I_a^{**} - I_a^{**}\ln\frac{I_a}{I_a^{**}}\right).$$

Then we obtain

$$\begin{aligned} \left.\frac{dV_2}{dt}\right|_{(5)} &= (S_a - S_a^{**})\left(r_a\left(1-\frac{S_a}{K_a}\right) - \beta_a I_a\right) \\ &\quad + (I_a - I_a^{**})(\beta_a S_a - \mu_a - \delta_a) \\ &= (S_a - S_a^{**})\left(\frac{r_a S_a^{**}}{K_a} + \beta_a I_a^{**} - \frac{r_a S_a}{K_a} - \beta_a I_a\right) \\ &\quad + \beta_a (I_a - I_a^{**})(S_a - S_a^{**}) \\ &= -\frac{r_a}{K_a}(S_a - S_a^{**})^2 \leq 0. \end{aligned}$$

It follows that $\hat{D} = \{(S_a, I_a) \in \operatorname{int} R^2_+ : \frac{dV_2}{dt} = 0\} = \{(S_a, I_a) : S_a = S_a^{**}, I_a \geq 0\}$. If $\hat{D}$ is an invariant set of subsystem (5), then $I_a = I_a^{**}$ by the first equation of subsystem (5). Hence $D_2 = \{C_a\}$. LaSalle's invariance principle implies that the equilibrium C_a is globally asymptotically stable. □

2.3. Analysis of the full system

Since the first four equations of system (4) are independent of the variable R_h, we only need to analyze the dynamical behavior of the following equivalent system

$$\begin{cases} \dfrac{dS_a}{dt} = r_a S_a\left(1-\dfrac{S_a}{K_a}\right) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a+\delta_a) I_a \\ \dfrac{dS_h}{dt} = \Pi_h - \beta_h I_a S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_h I_a S_h - (\mu_h+\delta_h+\gamma) I_h. \end{cases} \tag{6}$$

Clearly, $\mathbb{R}^4_+$ is a positively invariant attracting set. We discuss the dynamical behavior of system (6) in the positively invariant set $\mathbb{R}^4_+$.

2.3.1. Local stability of the full system (6)

System (6) always has two disease-free equilibria given by $A_{ah}(0, 0, S_h^*, 0)$ and $B_{ah}(K_a, 0, S_h^*, 0)$; if $R_{0,1} > 1$, then system (6) also has a unique endemic equilibrium given by $C_{ah}(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$.

Lemma 2.4. *(i) The disease-free equilibrium A_{ah} is always unstable; (ii) If $\frac{\mu_a+\delta_a}{\beta_a} \geq K_a$ (i.e., $\mathcal{R}_{0,1} \leq 1$), then the disease-free equilibrium B_{ah} is locally asymptotically stable for positive trajectories; (iii) If $\frac{\mu_a+\delta_a}{\beta_a} < K_a$ (i.e., $\mathcal{R}_{0,1} > 1$), then the disease-free equilibrium B_{ah} is unstable and the endemic equilibrium C_{ah} is locally asymptotically stable.*

Proof. The characteristic equation of the Jacobian matrix at an arbitrary equilibrium (S_a, I_a, S_h, I_h) takes the form

$$(\lambda+\beta_h I_a+\mu_h)(\lambda+\mu_h+\delta_h+\gamma)\times\left(\left(\lambda-\left(r_a-\frac{2r_a}{K_a}S_a-\beta_a I_a\right)\right)\times(\lambda-(\beta_a S_a-\mu_a-\delta_a))+\beta_a^2 S_a I_a\right)=0.$$

(i) If $(S_a, I_a, S_h, I_h)=(0,0,S_h^*,0)$, the eigenvalues are $\lambda_1=r_a>0$, $\lambda_2=-(\mu_a+\delta_a)$, $\lambda_3=-\mu_h$, $\lambda_4=-(\mu_h+\delta_h+\gamma)$. Hence, A_{ah} is always unstable.

(ii) If $\mathcal{R}_{0,1}<1$ and $(S_a, I_a, S_h, I_h)=(K_a,0,S_h^*,0)$, the eigenvalues are $\lambda_1=-r_a<0$, $\lambda_2=(\mu_a+\delta_a)(\mathcal{R}_{0,1}-1)<0$, $\lambda_3=-\mu_h<0$, $\lambda_4=-(\mu_h+\delta_h+\gamma)<0$. Hence, the equilibrium B_{ah} is locally asymptotically stable.

(iii) If $\mathcal{R}_{0,1}>1$ and $(S_a, I_a, S_h, I_h)=(K_a,0,S_h^*,0)$, the eigenvalues are $\lambda_1=-r_a<0$, $\lambda_2=(\mu_a+\delta_a)(\mathcal{R}_{0,1}-1)>0$, $\lambda_3=-\mu_h<0$, $\lambda_4=-(\mu_h+\delta_h+\gamma)<0$. Hence, the equilibrium B_{ah} is unstable; If $\mathcal{R}_{0,1}>1$ and $(S_a, I_a, S_h, I_h)=(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$, the characteristic equation of the Jacobian matrix at the endemic equilibrium C_{ah} is

$$\left(\lambda^2+\frac{r_a}{K_a}S_a^{**}\lambda+{\beta_a}^2 S_a^{**}I_a^{**}\right)(\lambda+\beta_h I_a^{**}+\mu_h)\times(\lambda+\mu_h+\delta_h+\gamma)=0.$$

Since $S_a^{**}>0, I_a^{**}>0$ if $\mathcal{R}_{0,1}>1$, all eigenvalues have negative real parts. Hence, the endemic equilibrium C_{ah} is locally asymptotically stable.

□

Remark 2.5. If $\mathcal{R}_{0,1}=1$, then the equilibrium C_{ah} coincides with the equilibrium B_{ah} which is a saddle-node and is locally asymptotically stable for positive trajectories.

2.3.2. Global stability of the full system (6)

Theorem 2.6. *(i) If $\frac{\mu_a+\delta_a}{\beta_a}\geq K_a$ (i.e. $\mathcal{R}_{0,1}\leq 1$), then the disease-free equilibrium B_{ah} of the full system (6) is globally asymptotically stable; (ii) If $0<\frac{\mu_a+\delta_a}{\beta_a}<K_a$ (i.e., $\mathcal{R}_{0,1}>1$), then the endemic equilibrium C_{ah} of the full system (6) is globally asymptotically stable.*

Proof. (i) According to Lemma 2.3, the disease-free equilibrium B_a of the avian-only subsystem (5) is globally asymptotically stable if $\mathcal{R}_{0,1}\leq 1$. To prove the global stability of B_{ah}, we only need to consider system (6) with the avian components already at the disease-free steady state, given by

$$\begin{cases}\dfrac{dS_h}{dt}=\Pi_h-\mu_h S_h\\ \dfrac{dI_h}{dt}=-(\mu_h+\delta_h+\gamma)I_h.\end{cases}\tag{7}$$

Clearly, we can obtain that $S_h\to S_h^*$, $I_h\to 0$ if $t\to\infty$. Hence, the disease-free equilibrium B_{ah} is globally asymptotically stable.

(ii) Similarly, by Lemma 2.3, the endemic equilibrium C_a of avian-only subsystem (5) is globally asymptotically stable if $\mathcal{R}_{0,1}>1$. To prove the global stability of the equilibrium C_{ah}, we only need to consider system (6) with the avian components already at the endemic steady state, given by

$$\begin{cases}\dfrac{dS_h}{dt}=\Pi_h-\beta_h I_a^{**}S_h-\mu_h S_h\\ \dfrac{dI_h}{dt}=\beta_h I_a^{**}S_h-(\mu_h+\delta_h+\gamma)I_h.\end{cases}\tag{8}$$

We can easily deduce that subsystem (8) has a unique positive equilibrium (S_h^{**}, I_h^{**}) which is locally asymptotically stable.

To prove the global stability of the positive equilibrium (S_h^{**}, I_h^{**}) of subsystem (8), we choose a Lyapunov function as follows

$$V=S_h^{**}\left(\frac{S_h}{S_h^{**}}-\ln\frac{S_h}{S_h^{**}}\right)+I_h^{**}\left(\frac{I_h}{I_h^{**}}-\ln\frac{I_h}{I_h^{**}}\right),$$

then,

$$\left.\frac{dV}{dt}\right|_{(8)}=\frac{dS_h}{dt}-\frac{S_h^{**}}{S_h}\frac{dS_h}{dt}+\frac{dI_h}{dt}-\frac{I_h^{**}}{I_h}\frac{dI_h}{dt}.$$

Using the relationships that (at endemic state) $\Pi_h=\beta_h I_a^{**}S_h^{**}+\mu_h S_h^{**}$ and $\mu_h+\delta_h+\gamma=\frac{\beta_h I_a^{**}S_h^{**}}{I_h^{**}}$, we obtain

$$\begin{aligned}\frac{dS_h}{dt}-\frac{S_h^{**}}{S_h}\frac{dS_h}{dt}&=(\Pi_h-\beta_h I_a^{**}S_h-\mu_h S_h)-\frac{S_h^{**}}{S_h}(\Pi_h-\beta_h I_a^{**}S_h-\mu_h S_h)\\&=(\beta_h I_a^{**}S_h^{**}+\mu_h S_h^{**}-\beta_h I_a^{**}S_h-\mu_h S_h)-\frac{S_h^{**}}{S_h}(\beta_h I_a^{**}S_h^{**}+\mu_h S_h^{**}-\beta_h I_a^{**}S_h-\mu_h S_h)\\&=\mu_h S_h^{**}\left(2-\frac{S_h^{**}}{S_h}-\frac{S_h}{S_h^{**}}\right)+2\beta_h I_a^{**}S_h^{**}-\beta_h I_a^{**}S_h-\beta_h I_a^{**}\frac{(S_h^{**})^2}{S_h}\end{aligned}$$

and

$$\begin{aligned}\frac{dI_h}{dt}-\frac{I_h^{**}}{I_h}\frac{dI_h}{dt}&=(\beta_h I_a^{**}S_h-(\mu_h+\delta_h+\gamma)I_h)-\frac{I_h^{**}}{I_h}(\beta_h I_a^{**}S_h-(\mu_h+\delta_h+\gamma)I_h)\\&=\left(\beta_h I_a^{**}S_h-\beta_h I_a^{**}S_h^{**}\frac{I_h}{I_h^{**}}\right)-\frac{I_h^{**}}{I_h}\left(\beta_h I_a^{**}S_h-\beta_h I_a^{**}S_h^{**}\frac{I_h}{I_h^{**}}\right)\\&=\beta_h I_a^{**}S_h+\beta_h I_a^{**}S_h^{**}-\beta_h I_a^{**}S_h^{**}\frac{I_h}{I_h^{**}}-\beta_h I_a^{**}S_h\frac{I_h^{**}}{I_h}.\end{aligned}$$

Therefore, we have

$$\left.\frac{dV}{dt}\right|_{(8)}=\mu_h S_h^{**}\left(2-\frac{S_h^{**}}{S_h}-\frac{S_h}{S_h^{**}}\right)+\beta_h I_a^{**}S_h^{**}\left(3-\frac{S_h^{**}}{S_h}-\frac{I_h}{I_h^{**}}-\frac{S_h}{S_h^{**}}\frac{I_h^{**}}{I_h}\right).$$

Since the arithmetic mean exceeds the geometric mean, we have

$$2-\frac{S_h^{**}}{S_h}-\frac{S_h}{S_h^{**}}\leq 0,$$

$$3-\frac{S_h^{**}}{S_h}-\frac{I_h}{I_h^{**}}-\frac{S_h}{S_h^{**}}\frac{I_h^{**}}{I_h}\leq 0.$$

Hence, $\frac{dV}{dt}|_{(8)}\leq 0$. Due to $\tilde{D}=\{(S_h,I_h)\in \operatorname{int}\mathbb{R}^2_+ : \frac{dV}{dt}=0\}=\{(S_h^{**},I_h^{**})\}$, by the LaSalle's invariance principle, it follows that $S_h\to S_h^{**}$ and $I_h\to I_h^{**}$ if $t\to\infty$. Therefore, the endemic equilibrium C_{ah} of the full system (6) is globally asymptotically stable. □

Now we can state our results for the original SI-SIR model (4) with logistic growth for the avian population.

Corollary 2.7. *(i) The disease-free equilibrium A of model (4) with logistic avian growth is always unstable; (ii) If $\frac{\mu_a+\delta_a}{\beta_a}\geq K_a$ (i.e., $\mathcal{R}_{0,1}\leq 1$), then the disease-free equilibrium B of model (4) is globally asymptotically stable for positive trajectories; (iii) If $0<\frac{\mu_a+\delta_a}{\beta_a}<K_a$ (i.e.,*

$\mathcal{R}_{0,1} > 1$), then the disease-free equilibrium B of model (4) is unstable but the endemic equilibrium C of model (4) is globally asymptotically stable.

Remark 2.8. If the susceptible avian population is subject to constant growth, that is, $g(S_a) = \Pi_a - \mu_a S_a$, where Π_a is the recruit rate of new recruitments and newborns and μ_a is the mortality rate of the avian population, then we can obtain analogous results and the dynamics are very much similar to that of system (4) with logistic avian growth.

3. Model (1) with Allee effect for avian population

3.1. The model

If the avian population is subject to Allee effect in system (1), then we have the following SI-SIR model:

$$\begin{cases} \dfrac{dS_a}{dt} = r_a S_a\left(1 - \dfrac{S_a}{M_a}\right)\left(\dfrac{S_a}{m_a} - 1\right) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a + \delta_a) I_a \\ \dfrac{dS_h}{dt} = \Pi_h - \beta_h I_a S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_h I_a S_h - (\mu_h + \delta_h + \gamma) I_h \\ \dfrac{dR_h}{dt} = \gamma I_h - \mu_h R_h, \end{cases} \tag{9}$$

where r_a, M_a and m_a ($m_a < M_a$) are the intrinsic growth rate, the maximal carrying capacity and the critical carrying capacity of the avian population, respectively, other assumptions and the meanings of other parameters remain unchanged. System (9) has a unique solution satisfying the initial conditions in $\mathbb{R}^5_+$ which is a positively invariant set.

Define the basic reproduction number by

$$\mathcal{R}_{0,2} = \frac{\beta_a (M_a + m_a)(\mu_a + \delta_a)}{(\mu_a + \delta_a)^2 + M_a m_a \beta_a^2}.$$

We can deduce three disease-free equilibria given by $H_1(0, 0, S_h^*, 0, 0)$, $H_2(m_a, 0, S_h^*, 0, 0)$, and $H_3(M_a, 0, S_h^*, 0, 0)$, where $S_h^* = \frac{\Pi_h}{\mu_h}$. If $\mathcal{R}_{0,2} > 1$, we can also derive a unique endemic equilibrium given by $H_4(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$, where

$$S_a^{**} = \frac{\mu_a + \delta_a}{\beta_a},\ I_a^{**} = \frac{r_a}{\beta_a}\frac{\beta_a^2 M_a m_a + (\mu_a + \delta_a)^2}{M_a m_a \beta_a^2}(\mathcal{R}_{0,2} - 1),$$
$$S_h^{**} = \frac{\Pi_h}{\beta_h I_a^{**} + \mu_h},\ I_h^{**} = \frac{\beta_h I_a^{**} S_h^{**}}{\mu_h + \delta_h + \gamma},\ R_h^{**} = \frac{\gamma I_h^{**}}{\mu_h}.$$

Comparing the relationship between $\mathcal{R}_{0,2}$ and 1, we have the following results:

(i) $\mathcal{R}_{0,2} < 1 \Leftrightarrow \frac{\mu_a+\delta_a}{\beta_a} < m_a$ or $\frac{\mu_a+\delta_a}{\beta_a} > M_a$;
(ii) $\mathcal{R}_{0,2} > 1 \Leftrightarrow m_a < \frac{\mu_a+\delta_a}{\beta_a} < M_a$;
(iii) $\mathcal{R}_{0,2} = 1 \Leftrightarrow \frac{\mu_a+\delta_a}{\beta_a} = m_a$ or $\frac{\mu_a+\delta_a}{\beta_a} = M_a$.

Before analyzing the dynamical behavior of the full model (9) with Allee effect, once again we first study the dynamical behavior of the avian-only subsystem.

3.2. Analysis of the avian-only subsystem

Consider the following avian-only subsystem:

$$\begin{cases} \dfrac{dS_a}{dt} = r_a S_a\left(1 - \dfrac{S_a}{M_a}\right)\left(\dfrac{S_a}{m_a} - 1\right) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a + \delta_a) I_a. \end{cases} \tag{10}$$

Similarly, $\mathbb{R}^2_+$ is a positively invariant set of the subsystem (10). First we discuss its dynamical behavior in $\mathbb{R}^2_+$.

3.2.1. Local stability of the avian-only subsystem (10)

The avian-only subsystem (10) always has three disease-free equilibria given by $O(0, 0)$, $A(m_a, 0)$ and $B(M_a, 0)$; if $\mathcal{R}_{0,2} > 1$, then the subsystem also has a unique endemic equilibrium given by $E(S_a^{**}, I_a^{**})$.

Lemma 3.1. *(i) The disease-free equilibrium O is always locally asymptotically stable but the disease-free equilibrium A is always unstable; (ii) The disease-free equilibrium B is unstable if $0 < \frac{\mu_a+\delta_a}{\beta_a} < M_a$ but locally asymptotically stable if $\frac{\mu_a+\delta_a}{\beta_a} \geq M_a$ for positive trajectories; (iii) The endemic equilibrium E is unstable if $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$ but locally asymptotically stable if $\frac{M_a+m_a}{2} \leq \frac{\mu_a+\delta_a}{\beta_a} < M_a$.*

Proof. The characteristic equation of the Jacobian matrix of an arbitrary equilibrium (S_a, I_a) is

$$\left[\lambda - \left(r_a\left(\frac{-3S_a^2 + 2(M_a + m_a)S_a}{M_a m_a} - 1\right) - \beta_a I_a\right)\right]$$
$$[\lambda - (\beta_a S_a - \mu_a - \delta_a)] + \beta_a^2 S_a I_a = 0.$$

(i) If $(S_a, I_a) = (0, 0)$, the eigenvalues are $\lambda_1 = -r_a < 0$, $\lambda_2 = -(\mu_a + \delta_a) < 0$. Hence, the disease-free equilibrium O is always locally asymptotically stable; If $(S_a, I_a) = (m_a, 0)$, the eigenvalues are $\lambda_1 = \frac{(M_a - m_a) r_a}{M_a} > 0$, $\lambda_2 = \beta_a(m_a - \frac{\mu_a+\delta_a}{\beta_a})$. Hence, the equilibrium A is always unstable;

(ii) If $(S_a, I_a) = (M_a, 0)$, the eigenvalues are $\lambda_1 = \frac{(m_a - M_a) r_a}{m_a} < 0$, $\lambda_2 = \beta_a(M_a - \frac{\mu_a+\delta_a}{\beta_a})$. If $0 < \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then $\lambda_2 > 0$. Hence, the equilibrium B is unstable; If $\frac{\mu_a+\delta_a}{\beta_a} > M_a$, then $\lambda_2 < 0$. Hence, the equilibrium B is locally asymptotically stable;

(iii) If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < M_a$ and $(S_a, I_a) = (S_a^{**}, I_a^{**})$, the above characteristic equation becomes

$$\lambda^2 + a\lambda + b = 0, \tag{11}$$

where

$$a = -\frac{r_a}{M_a m_a}\frac{2(\mu_a + \delta_a)}{\beta_a}\left(\frac{M_a + m_a}{2} - \frac{\mu_a + \delta_a}{\beta_a}\right),$$
$$b = \frac{r_a(\mu_a + \delta_a)(\beta_a^2 M_a m_a + (\mu_a + \delta_a)^2)}{M_a m_a {\beta_a}^2}(\mathcal{R}_{0,2} - 1).$$

Clearly, if $\frac{M_a+m_a}{2} < \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then $a > 0$ and $b > 0$. Thus all eigenvalues have negative real parts and the endemic equilibrium E is locally asymptotically stable; if $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$, then $a < 0$ and $b > 0$. Hence all the eigenvalues have positive real parts and the endemic equilibrium E is unstable. If $\frac{\mu_a+\delta_a}{\beta_a} = \frac{M_a+m_a}{2}$, the characteristic Eq. (11) has purely imaginary eigenvalues $\pm i\omega$, where $\omega = \sqrt{\beta_a^2 S_a^{**} I_a^{**}} > 0$. In this case, the endemic equilibrium E is a center or a fine focus.

Next, we shall study the type of the equilibrium E if $\frac{\mu_a+\delta_a}{\beta_a} = \frac{M_a+m_a}{2}$. Making a transformation

$$S = S_a - S_a^{**},\quad I = I_a - I_a^{**}$$

System (10) can be turned into

$$\begin{cases} \dfrac{dS}{dt} = r_a(S+S_a^{**})\Big(1-\dfrac{S+S_a^{**}}{M_a}\Big)\Big(\dfrac{S+S_a^{**}}{m_a}-1\Big) \\ \qquad\quad -\beta_a(S+S_a^{**})(I+I_a^{**}) \\ \dfrac{dI}{dt} = \beta_a(S+S_a^{**})(I+I_a^{**})-(\mu_a+\delta_a)(I+I_a^{**}). \end{cases} \tag{12}$$

Simplifying system (12), it becomes

$$\begin{cases} \dfrac{dS}{dt} = -\beta_a S_a^{**} I - \dfrac{r_a S_a^{**}}{M_a m_a}S^2 - \beta_a IS - \dfrac{r_a}{M_a m_a}S^3 \\ \dfrac{dI}{dt} = \beta_a I_a^{**} S + \beta_a IS. \end{cases} \tag{13}$$

Let $x = S$, $y = \sqrt{\frac{S_a^{**}}{I_a^{**}}}I$. System (13) can be written as

$$\begin{cases} \dfrac{dx}{dt} = -\sqrt{\beta_a^2 S_a^{**} I_a^{**}}\,y - \dfrac{r_a S_a^{**}}{M_a m_a}x^2 - \dfrac{\beta_a}{\sqrt{\frac{S_a^{**}}{I_a^{**}}}}xy - \dfrac{r_a}{M_a m_a}x^3 \\ \dfrac{dy}{dt} = \sqrt{\beta_a^2 S_a^{**} I_a^{**}}\,x + \beta_a xy. \end{cases} \tag{14}$$

According to the Hopf bifurcation formula in Guckenheimer and Holmes [23] in two-dimensional systems

$$\frac{dx}{dt} = -\omega y + f(x,y),\quad \frac{dy}{dt} = \omega x + g(x,y),$$

the singular point (0, 0) of system (14) is a stable fine focus of order one for $c < 0$, where

$$c = \frac{f_{xxx}+f_{xyy}+g_{xxy}+g_{yyy}}{16} + \frac{f_{xy}(f_{xx}+f_{yy}) - g_{xy}(g_{xx}+g_{yy}) - f_{xx}g_{xx} + f_{yy}g_{yy}}{16\omega},$$

in which the partial derivatives are all evaluated at (0, 0) as follows: $f_{xxx} = \frac{-6r_a}{M_a m_a}$, $f_{xyy} = 0$, $g_{xxy} = 0$, $g_{yyy} = 0$, $f_{xx} = \frac{-2r_a S_a^{**}}{M_a m_a}$, $f_{xy} = \frac{-\beta_a}{\sqrt{\frac{S_a^{**}}{I_a^{**}}}}$, $f_{yy} = 0$, $g_{xx} = 0$, $g_{xy} = \beta_a$, $g_{yy} = 0$. Then $c = \frac{-r_a}{4M_a m_a} < 0$. Thus, the trivial equilibrium (0, 0) of system (14) is a stable fine focus of order one. Hence, the endemic equilibrium E of subsystem (10) is a stable fine focus of order one for $\frac{\mu_a+\delta_a}{\beta_a} = \frac{M_a+m_a}{2}$.

In summary, the endemic equilibrium E of the avian-only subsystem (10) is locally asymptotically stable if $\frac{M_a+m_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$ and unstable if $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$. □

Remark 3.2. If $\frac{\mu_a+\delta_a}{\beta_a} = m_a$, then the equilibrium E coincides with the equilibrium A, which is a saddle-node and the disease-free equilibrium A is unstable for positive trajectories; If $\frac{\mu_a+\delta_a}{\beta_a} = M_a$, then the equilibrium E coincides with the equilibrium B, which is a saddle-node and the disease-free equilibrium B is locally asymptotically stable for positive trajectories.

In order to discuss the existence and uniqueness of limit cycles in the avian-only subsystem (10), we introduce a lemma.

Lemma 3.3. *([15, 16]) Let $f(x)$ and $g(x)$ be continuously differentiable functions on an open interval (r_1, r_2) and $\psi(y)$ be a continuously differentiable function on $\mathbb{R}$. Consider the Liénard system*

$$\begin{cases} \dfrac{dx}{dt} = \psi(y) - \displaystyle\int_{x_0}^{x} f(u)du, \\ \dfrac{dy}{dt} = -g(x) \end{cases} \tag{15}$$

and assume that

(i) $\frac{d\psi(y)}{dy} > 0$;

(ii) *there is a unique $x_0 \in (r_1, r_2)$ such that $(x-x_0)g(x-x_0) > 0$ for $x \neq x_0$ and $g(x_0) = 0$;*

(iii) $f(x_0)\frac{d}{dx}\big(\frac{f(x)}{g(x)}\big) < 0$ *for $x \neq x_0$.*

Then system (15) has at most one limit cycle, and if it exists, it is hyperbolic.

Theorem 3.4. *If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$, then the avian-only subsystem (10) has a unique limit cycle which is hyperbolic.*

Proof. In order to apply Lemma 3.3, we make a transformation

$$S_a = x,\ I_a = e^y,\ dt = -x^{-1}d\tau.$$

System (10) can be written as

$$\begin{cases} \dfrac{dx}{d\tau} = \beta_a e^y - r_a\Big(1-\dfrac{x}{M_a}\Big)\Big(\dfrac{x}{m_a}-1\Big) & \equiv \psi(y) - F(x) \\ \dfrac{dy}{d\tau} = \dfrac{(\mu_a+\delta_a)}{x} - \beta_a & \equiv -g(x), \end{cases} \tag{16}$$

where $\psi(y) = \beta_a e^y$, $F(x) = r_a(1-\frac{x}{M_a})(\frac{x}{m_a}-1)$, and $g(x) = \beta_a - \frac{(\mu_a+\delta_a)}{x}$.

Set $r_1 = m_a$, $r_2 = \frac{M_a+m_a}{2}$. We check the three conditions of Lemma 3.3:

(i) $\frac{d\psi(y)}{dy} = \beta_a e^y > 0$.

(ii) $\frac{dg(x)}{dx} = \frac{\mu_a+\delta_a}{x^2} > 0$. We choose $x_0 = \frac{\mu_a+\delta_a}{\beta_a} \in (r_1, r_2)$, where x_0 satisfies that $g(x_0) = 0$. Hence, $(x-x_0)g(x-x_0) > 0$ for $x \neq x_0$.

(iii) $f(x) = \frac{dF(x)}{dx} = \frac{-2r_a}{M_a m_a}(x - \frac{M_a+m_a}{2})$, $f(x_0) = \frac{-2r_a}{M_a m_a}(x_0 - \frac{M_a+m_a}{2}) > 0$, $\frac{f(x)}{g(x)} = \frac{-r_a x(2x-(M_a+m_a))}{M_a m_a(\beta_a x-(\mu_a+\delta_a))}$, $\frac{d}{dx}(\frac{f(x)}{g(x)}) = \frac{h(x)}{M_a m_a(\beta_a x-(\mu_a+\delta_a))^2}$, where $h(x) = r_a(-2\beta_a x^2 + 4(\mu_a+\delta_a)x - (\mu_a+\delta_a)(M_a+m_a))$, $\frac{d}{dx}(h(x)) = -4\beta_a r_a(x - \frac{\mu_a+\delta_a}{\beta_a})$. When $\widetilde{x} = \frac{\mu_a+\delta_a}{\beta_a} = x_0$, $h'(\widetilde{x}) = 0$, $h'(x) > 0$ for $m_a < x < \widetilde{x}$ and $h'(x) < 0$ for $\widetilde{x} < x < \frac{m_a+M_a}{2}$. $h(\widetilde{x}) = 2(\mu_a+\delta_a)r_a(\frac{\mu_a+\delta_a}{\beta_a} - \frac{M_a+m_a}{2}) < 0$. Hence, we have $h(x) < h(\widetilde{x}) < 0$, $f(x_0)\frac{d}{dx}(\frac{f(x)}{g(x)}) < 0$ for $m_a < x < \frac{M_a+m_a}{2}$ and $x \neq x_0$.

Thus, system (16) satisfies the three conditions of Lemma 3.3. So the avian-only subsystem (10) has at most one limit cycle, and it is hyperbolic.

Next, we prove the existence of a limit cycle of subsystem (10). We choose β_a as a perturbed parameter. The equation $\frac{\mu_a+\delta_a}{\beta_a} = \frac{M_a+m_a}{2}$ implies that $\beta_a = \frac{2(\mu_a+\delta_a)}{M_a+m_a}$. Set $\mu = \beta_a - \frac{2(\mu_a+\delta_a)}{M_a+m_a}$, where $|\mu| \ll 1$. According to Lemma 3.1, we have the following results:

If $\mu < 0$ (i.e., $\frac{\mu_a+\delta_a}{\beta_a} > \frac{M_a+m_a}{2}$), then the endemic equilibrium E is locally asymptotically stable; If $\mu = 0$ (i.e., $\frac{\mu_a+\delta_a}{\beta_a} = \frac{M_a+m_a}{2}$), then the endemic equilibrium E is a stable fine focus of order one; If $\mu > 0$ (i.e., $\frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$), then the endemic equilibrium E is an unstable focus.

By the results in Zhang and Feng [60] (p.207), there exists at least one stable limit cycle in the neighborhood of the endemic equilibrium E of system (10) for sufficient small $\mu > 0$. Thus, system (10) has a unique limit cycle which is hyperbolic for $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$. □

3.2.2. Global stability of the avian-only subsystem (10)

In order to study global stability of these equilibria, we need to analyze the critical point at infinity of the avian-only subsystem (10).

Making a Poincaré transformation

$$S_a = \frac{1}{z},\ I_a = \frac{u}{z} \quad \text{or} \quad z = \frac{1}{S_a},\ u = \frac{I_a}{S_a}$$

and let $d\tau = \frac{dt}{z^2}$. Then system (10) can be written as

$$\begin{cases} \dfrac{du}{d\tau} = \dfrac{r_a}{M_a m_a}u + \left(\beta_a - \dfrac{r_a(M_a+m_a)}{M_a m_a}\right)uz \\ \qquad + \beta_a u^2 z + (r_a - \mu_a - \delta_a)uz^2 \\ \dfrac{dz}{d\tau} = \dfrac{r_a}{M_a m_a}z - \dfrac{r_a(M_a+m_a)}{M_a m_a}z^2 + \beta_a u z^2 + r_a z^3. \end{cases} \tag{17}$$

It is clear to see that there is a unique equilibrium $C(0, 0)$ on the u-axis. The eigenvalues of the Jacobian matrix of the equilibrium $C(0, 0)$ of system (17) are $\lambda_1 = \lambda_2 = \frac{r_a}{M_a m_a}$. Hence, the equilibrium $C(0, 0)$ is an unstable node.

Making another Poincaré transformation

$$S_a = \frac{v}{z},\ I_a = \frac{1}{z} \quad \text{or} \quad z = \frac{1}{I_a},\ v = \frac{S_a}{I_a}$$

and letting $d\tau = \frac{dt}{z^2}$. Then system (10) is transformed into

$$\begin{cases} \dfrac{dv}{d\tau} = -\beta_a vz + (\mu_a + \delta_a)vz^2 - \beta_a v^2 z + r_a v\left(z - \dfrac{v}{M_a}\right)\left(\dfrac{v}{m_a} - z\right) \\ \dfrac{dz}{d\tau} = -\beta_a vz^2 + (\mu_a + \delta_a)z^3. \end{cases} \tag{18}$$

Let $z = 0$. Then system (18) has an equilibrium $D(0, 0)$ which is a higher order singular point. The geometric property of the higher order singular point $D(0, 0)$ of system (18) is decided by the following system:

$$\begin{cases} \dfrac{dv}{d\tau} = -\beta_a vz \\ \dfrac{dz}{d\tau} = -\beta_a vz^2 + (\mu_a + \delta_a)z^3. \end{cases} \tag{19}$$

Making a time transformation

$$d\tau = d\tau_1/z.$$

Then system (19) becomes

$$\begin{cases} \dfrac{dv}{d\tau_1} = -\beta_a v \\ \dfrac{dz}{d\tau_1} = -\beta_a vz + (\mu_a + \delta_a)z^2. \end{cases} \tag{20}$$

System (20) has a unique equilibrium $(0, 0)$ which is a higher order singular point with one of the eigenvalues being zero. By the results in Zhang et al. [61], the equilibrium $(0, 0)$ is a saddle-node.

Thus, we have the following results: system (10) has two critical points at infinity given by $C(0, 0)$ and $D(0, 0)$, where $C(0, 0)$ corresponds with the critical point at infinity of the S_a-axis and is an unstable node, and $D(0, 0)$ corresponds with the critical point at infinity of the I_a-axis and is a saddle-node.

Hence, we can always divide the region $\mathbb{R}^2_+$ into sub-regions D_1 and D_2 as follows:

(i) If $0 < \frac{\mu_a+\delta_a}{\beta_a} \le m_a$, then the sub-region D_1 is surrounded by the saddle-node separatrix BD, curve DO, and curve OB; the sub-region D_2 is surrounded by the saddle-node separatrix BD, curve CD, and curve CB.

(ii) If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{m_a+M_a}{2}$, then the sub-region D_1 is surrounded by the saddle-node separatrix DA, curve DO, and curve AO; the sub-region D_2 is surrounded by the saddle-node separatrix DA, curve AC, and curve CD.

(iii) If $\frac{m_a+M_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then the sub-region D_1 is surrounded by the saddle-node separatrix DA, curve DO, and curve AO; the sub-region D_2 is surrounded by the saddle-node separatrix DA, curve AC, and curve CD.

(iv) If $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$, then the sub-region D_1 is surrounded by the saddle-node separatrix DA, curve DO, and curve AO; the sub-region D_2 is surrounded by the saddle-node separatrix DA, curve AC, and curve CD.

The global dynamics of the avian-only subsystem (10) can be summarized in the following theorem.

Theorem 3.5. *(i) The disease-free equilibrium O of the avian-only subsystem (10) is always globally asymptotically stable in D_1; (ii) If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{m_a+M_a}{2}$, then there is a limit cycle in the neighborhood of the endemic equilibrium E of the avian-only subsystem (10) which is globally asymptotically stable in D_2; (iii) If $\frac{m_a+M_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then the endemic equilibrium E of the avian-only subsystem (10) is globally asymptotically stable in D_2; (iv) If $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$, then the disease-free equilibrium B of the avian-only subsystem (10) is globally asymptotically stable in D_2.*

Proof. Lemma 3.1 implies that the disease-free equilibrium O is always locally asymptotically stable, the endemic equilibrium E is locally asymptotically stable for $\frac{m_a+M_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$, and the disease-free equilibrium B is locally asymptotically stable for $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$.

(i) If $(S_a, I_a) \in D_1$, it should be noted that subsystem (10) has no endemic equilibrium in the interior of D_1 and the S_a- and I_a-axes are positively invariant, so there is no limit cycle in D_1. Hence, the disease-free equilibrium O is globally asymptotically stable in D_1 (see Fig. 1(a)).

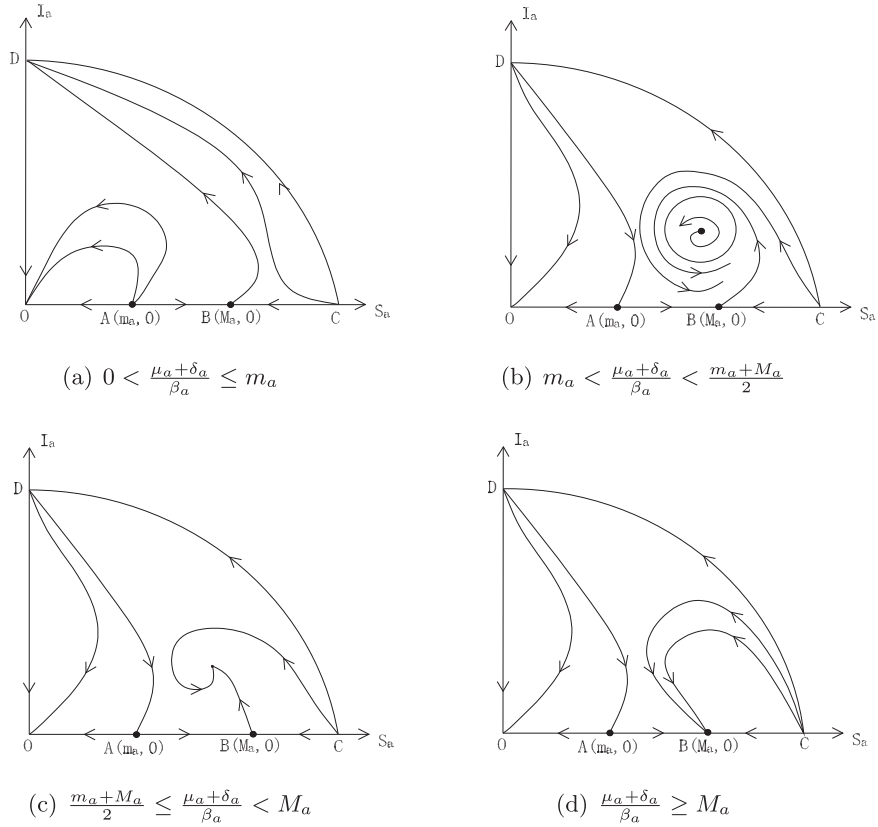
(ii) If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$ and $(S_a, I_a) \in D_2$, by Lemma 3.1 and Theorem 3.4, subsystem (10) has a unique limit cycle which is hyperbolic in D_2 and the endemic equilibrium E is an unstable focus, thus we can deduce that the limit cycle is internally stable (semistable from inside); according to Lemma 3.1, the equilibrium B is an unstable node, the infinite point C is an unstable node, the infinite point D is a saddle-node, and the saddle-node separatrix DA is a curve from the point D to the point A, thus solutions starting from the exterior of the limit cycle are tending to the limit cycle, that is, the limit cycle is externally stable (semistable from outside). Hence, the limit cycle is globally asymptotically stable in D_2 (see Fig. 1(b)).

(iii) If $\frac{m_a+M_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$ and $(S_a, I_a) \in D_2$, by Theorem 3.4, we know that there is no limit cycle in the neighborhood of the endemic equilibrium E in the D_2. On the other hand, according to Lemma 3.1, the endemic equilibrium E is a stable focus, the equilibrium B is an unstable saddle, the infinite point C is an unstable node, the infinite point D is a saddle-node, and the saddle-node separatrix DA is a curve from the point D to the point A, thus solutions starting from the region D_2 are tending to the equilibrium E. Hence, the endemic equilibrium E is globally asymptotically stable in D_2 (see Fig. 1(c)).

(iv) If $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$ and $(S_a, I_a) \in D_2$, it should be noted that there is no endemic equilibrium in the region D_2, so there is no limit cycle. By Lemma 3.1, the equilibrium B is a stable node, the infinite point C is an unstable node, the infinite point D is a saddle-node, and the saddle-node separatrix DA is a curve from the point D to the point A, thus all solutions starting from the region D_2 are tending to the disease-free equilibrium B. Hence, the disease-free equilibrium B is globally asymptotically stable in D_2 (see Fig. 1(d)). □

3.3. Analysis of the full system

Since the first four equations of system (9) are independent of the variable R_h, similarly we only analyze the dynamical behavior of the following equivalent system



(a) $0 < \frac{\mu_a+\delta_a}{\beta_a} \le m_a$ (b) $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{m_a+M_a}{2}$

(c) $\frac{m_a+M_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$ (d) $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$

Fig. 1. The plots are the global phase portraits of the avian-only subsystem (10) with respect to $\frac{\mu_a+\delta_a}{\beta_a}$. (a) $0 < \frac{\mu_a+\delta_a}{\beta_a} \le m_a$; (b) $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{m_a+M_a}{2}$; (c) $\frac{m_a+M_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$; (d) $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$.

$$\begin{cases} \dfrac{dS_a}{dt} = r_a S_a\left(1-\dfrac{S_a}{M_a}\right)\left(\dfrac{S_a}{m_a}-1\right) - \beta_a S_a I_a \\ \dfrac{dI_a}{dt} = \beta_a S_a I_a - (\mu_a+\delta_a) I_a \\ \dfrac{dS_h}{dt} = \Pi_h - \beta_h I_a S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_h I_a S_h - (\mu_h+\delta_h+\gamma) I_h. \end{cases} \tag{21}$$

We discuss the dynamical behavior of system (21) in its positively invariant set $\mathbb{R}^4_+$.

3.3.1. Local stability of the full system (21)

System (21) has three equilibria given by $O_{ah}(0,0,S_h^*,0)$, $A_{ah}(m_a,0,S_h^*,0)$, and $B_{ah}(M_a,0,S_h^*,0)$. If $\mathcal{R}_{0,2} > 1$, system (21) also has a unique endemic equilibrium given by $E_{ah}(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$.

Lemma 3.6. *(i) The disease-free equilibrium O_{ah} is always locally asymptotically stable and the disease-free equilibrium A_{ah} is always unstable; (ii) If $\frac{\mu_a+\delta_a}{\beta_a} < M_a$, then the disease-free equilibrium B_{ah} is unstable; if $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$, then the disease-free equilibrium B_{ah} is locally asymptotically stable; (iii) If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$, then the endemic equilibrium E_{ah} is unstable; if $\frac{M_a+m_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then the endemic equilibrium E_{ah} is locally asymptotically stable.*

Proof. The characteristic equation of the Jacobian matrix of an arbitrary equilibrium (S_a, I_a, S_h, I_h) of system (21) is given by

$$\left[\left(\lambda - \frac{r_a(-3S_a^2+2(M_a+m_a)S_a - M_a m_a)}{M_a m_a} - \beta_a I_a\right)\right.$$
$$\left.(\lambda - \beta_a S_a + \mu_a + \delta_a) + \beta_a^2 S_a I_a\right]$$
$$(\lambda + \beta_h I_a + \mu_h)(\lambda + \mu_h + \delta_h + \gamma) = 0.$$

(i) If $(S_a, I_a, S_h, I_h) = (0,0,S_h^*,0)$, the eigenvalues are $\lambda_1 = -r_a$, $\lambda_2 = -(\mu_a+\delta_a)$, $\lambda_3 = -\mu_h$, $\lambda_4 = -(\mu_h+\delta_h+\gamma)$. Obviously, these eigenvalues are negative. Hence, the disease-free equilibrium O_{ah} is always locally asymptotically stable; If $(S_a, I_a, S_h, I_h) = (m_a, 0, S_h^*, 0)$, the eigenvalues are

$$\lambda_1 = \frac{(M_a-m_a)r_a}{M_a} > 0,\ \lambda_2 = \beta_a\left(m_a - \frac{\mu_a+\delta_a}{\beta_a}\right),$$
$$\lambda_3 = -\mu_h, \lambda_4 = -(\mu_h+\delta_h+\gamma).$$

Since one of the eigenvalues is positive, the disease-free equilibrium A_{ah} is always unstable.

(ii) If $(S_a, I_a, S_h, I_h) = (M_a, 0, S_h^*, 0)$, the eigenvalues are

$$\lambda_1 = \frac{(m_a-M_a)r_a}{m_a},\ \lambda_2 = \beta_a\left(M_a - \frac{\mu_a+\delta_a}{\beta_a}\right),$$
$$\lambda_3 = -\mu_h, \lambda_4 = -(\mu_h+\delta_h+\gamma).$$

Obviously, if $\frac{\mu_a+\delta_a}{\beta_a} > M_a$, all the above eigenvalues are negative, the disease-free equilibrium B_{ah} is locally asymptotically stable; If $0 < \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then $\lambda_2 > 0$. Hence the disease-free equilibrium B_{ah} is unstable.

(iii) If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < M_a$ and $(S_a, I_a, S_h, I_h) = (S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$, the above characteristic equation becomes

$$(\lambda+\mu_h+\delta_h+\gamma)(\lambda+\beta_h I_a^{**}+\mu_h)(\lambda^2+a\lambda+b) = 0, \tag{22}$$

where the meanings of a and b are the same as in the characteristic equation (11). Since the characteristic equation (22) has at least

two negative eigenvalues $\lambda = -(\mu_h + \delta_h + \gamma), \lambda = -(\beta_h I_a^{**} + \mu_h)$, the local stability of the endemic equilibrium E_{ah} of system (21) is decided by the equation $\lambda^2 + a\lambda + b = 0$. Lemma 3.1 implies that if $\frac{M_a+m_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$, then the endemic equilibrium E_{ah} is locally asymptotically stable; if $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$, then the endemic equilibrium E_{ah} is unstable. □

Remark 3.7. If $\frac{\mu_a+\delta_a}{\beta_a} = M_a$, then the equilibrium E_{ah} coincides with the equilibrium B_{ah}, which is a saddle-node and is locally asymptotically stable for positive trajectories.

3.3.2. Global stability of the full system (21)

Set $E_1 = \{(S_a, I_a, S_h, I_h) : (S_a, I_a) \in D_1, S_h \ge 0, I_h \ge 0\}$ and $E_2 = \{(S_a, I_a, S_h, I_h) : (S_a, I_a) \in D_2, S_h \ge 0, I_h \ge 0\}$, where D_1 and D_2 are defined in Theorem 3.5.

Theorem 3.8. *(i) The disease-free equilibrium O_{ah} of system (21) is always globally asymptotically stable in E_1; (ii) If $\frac{M_a+m_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$, only the endemic equilibrium $E_{ah}(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})$ of system (21) is globally asymptotically stable in E_2; (iii) If $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$, only the disease-free equilibrium $B_{ah}(M_a, 0, S_h^*, 0)$ of system (21) is globally asymptotically stable in the region E_2.*

Proof. (i) If $(S_a, I_a, S_h, I_h) \in E_1$, then $(S_a, I_a) \in D_1$. According to Theorem 3.5, the disease-free equilibrium O of the avian-only subsystem (10) is always globally asymptotically stable in the region D_1. To prove the global stability of the disease-free equilibrium O_{ah}, we only need to consider the system (21) with the avian components already at the disease-free steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = -(\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{23}$$

Obviously, $I_h \to 0$, $S_h \to S_h^*$ if $t \to \infty$. Hence, the equilibrium O_{ah} is always globally asymptotically stable in the region E_1.

(ii) If $\frac{M_a+m_a}{2} \le \frac{\mu_a+\delta_a}{\beta_a} < M_a$ and $(S_a, I_a, S_h, I_h) \in E_2$, then $(S_a, I_a) \in D_2$. According to Theorem 3.5, the endemic equilibrium E of the subsystem (10) is globally asymptotically stable in the region D_2. To prove the global stability of the endemic equilibrium E_{ah}, we consider system (21) with the avian components already at the endemic steady state given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \beta_a I_a^{**} S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_a I_a^{**} S_h - (\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{24}$$

According to the proof of Theorem 2.6(ii), $S_h \to S_h^{**}$, $I_h \to I_h^{**}$ if $t \to \infty$. Hence, the endemic equilibrium E_{ah} is globally asymptotically stable in the region E_2.

(iii) If $\frac{\mu_a+\delta_a}{\beta_a} \ge M_a$ and $(S_a, I_a, S_h, I_h) \in E_2$, then $(S_a, I_a) \in D_2$. By Theorem 3.5, the disease-free equilibrium B of the subsystem (10) is globally asymptotically stable in the region D_2. To prove the global stability of the disease-free equilibrium B_{ah}, we consider system (21) with the avian components already at the disease-free steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = -(\mu_h + \delta_h + \gamma) I_h. \end{cases}$$

Obviously, $I_h \to 0$, $S_h \to S_h^*$ if $t \to \infty$. Hence, the disease-free equilibrium B_{ah} is globally asymptotically stable in the region E_2. □

Lemma 3.9. *The full system (21) has a unique periodic solution if and only if the subsystem (10) has a unique limit cycle.*

Proof. At first, we prove the sufficient condition. According to Theorem 3.4, the subsystem (10) has a unique limit cycle. Let the ω-periodic solution $(\tilde{S}_a(t), \tilde{I}_a(t))$ be the unique limit cycle of the subsystem (10). We will prove that the third equation of system (21) has a unique ω-periodic solution $\tilde{S}_h(t)$.

Any solution of the third equation of system (21) can be represented by

$$S_h(t) = e^{-\beta_h \int_{t_0}^t I_a(s)ds - \mu_h t}\left[e^{\mu_h t_0} S_h(t_0) + \Pi_h \int_{t_0}^t e^{\beta_h \int_{t_0}^s I_a(u)du} e^{\mu_h s} ds\right],$$

where $S_h(t_0)$ is the initial value of $S_h(t)$. Thus, all solutions of the third equation of system (21) on the three-dimensional cylinder

$$\Gamma \times \mathbb{R}^+ \times \mathbb{R}^+ = \Gamma \times [0, \infty) \times [0, \infty)$$

are denoted as

$$S_h(t) = e^{-\beta_h \int_{t_0}^t \tilde{I}_a(s)ds - \mu_h t}\left[e^{\mu_h t_0} S_h(t_0) + \Pi_h \int_{t_0}^t e_h^{\beta} \int_{t_0}^s \tilde{I}_a(u)du e^{\mu_h s} ds\right], \tag{25}$$

where $\Gamma = \{(\tilde{S}_a(t), \tilde{I}_a(t)) : t \in [0, \omega]\}$, $R^+ = [0, \infty)$.

In (25), we have

$$\begin{aligned} S_h(t+\omega) &= e^{-\beta_h \int_{t_0}^{t+\omega} \tilde{I}_a(s)ds - \mu_h (t+\omega)}\left[e^{\mu_h t_0} S_h(t_0)\right. \\ &\quad \left. + \Pi_h \int_{t_0}^{t+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds\right] \\ &= e^{-\beta_h \int_{t_0}^{t_0+\omega} \tilde{I}_a(s)ds} e^{-\beta_h \int_{t_0+\omega}^{t+\omega} \tilde{I}_a(s)ds} e^{-\mu_h (t+\omega)}\left[e^{\mu_h t_0} S_h(t_0)\right. \\ &\quad + \Pi_h \int_{t_0}^{t_0+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds \\ &\quad \left. + \Pi_h \int_{t_0+\omega}^{t+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds\right] \\ &= e^{-\beta_h \int_{t_0+\omega}^{t+\omega} \tilde{I}_a(s)ds} e^{-\mu_h t}\{e^{-\beta_h \int_{t_0}^{t_0+\omega} \tilde{I}_a(s)ds} e^{-\mu_h \omega}\left[e^{\mu_h t_0} S_h(t_0)\right. \\ &\quad \left. + \Pi_h \int_{t_0}^{t_0+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds\right] \\ &\quad + \Pi_h \int_{t_0+\omega}^{t+\omega} e^{\beta_h (-\int_{t_0}^{t_0+\omega} + \int_{t_0}^s) \tilde{I}_a(u)du} e^{\mu_h (s-\omega)} ds\} \\ &= e^{-\beta_h \int_{t_0}^t \tilde{I}_a(s)ds} e^{-\mu_h t}\left\{e^{-\beta_h \int_0^{\omega} \tilde{I}_a(s)ds} e^{-\mu_h \omega}\left[e^{\mu_h t_0} S_h(t_0)\right.\right. \\ &\quad \left. + \Pi_h \int_{t_0}^{t_0+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds\right] \\ &\quad \left. + \Pi_h \int_{t_0}^t e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds\right\}. \end{aligned}$$

If

$$e^{-\beta_h \int_0^{\omega} \tilde{I}_a(s)ds} e^{-\mu_h \omega}\left[e^{\mu_h t_0} S_h(t_0) + \Pi_h \int_{t_0}^{t_0+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds\right] = e^{\mu_h t_0} S_h(t_0),$$

i.e., if

$$S_h^*(t_0) = \frac{\Pi_h \int_{t_0}^{t_0+\omega} e^{\beta_h \int_{t_0}^s \tilde{I}_a(u)du} e^{\mu_h s} ds}{e^{\mu_h t_0}[e^{\beta_h \int_0^{\omega} \tilde{I}_a(s)ds} e^{\mu_h \omega} - 1]},$$

then

$$\tilde{S}_h(t) = e^{-\beta_h \int_{t_0}^{t} \tilde{I}_a(s)ds - \mu_h t}\left[e^{\mu_h t_0} S_h^*(t_0) + \Pi_h \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} \tilde{I}_a(u)du} e^{\mu_h s} ds\right] \tag{26}$$

is a unique periodic solution of the third equation of system (21).

Similarly,

$$\tilde{I}_h(t) = e^{-(\mu_h+\delta_h+\gamma)t}\left[e^{(\mu_h+\delta_h+\gamma)t_0} I_h^*(t_0) + \beta_h \int_{t_0}^{t} e^{(\mu_h+\delta_h+\gamma)s} \tilde{I}_a(s)\tilde{S}_h(s)ds\right] \tag{27}$$

is a unique periodic solution of the fourth equation of system (21), where

$$I_h^*(t_0) = \frac{\beta_h \int_{t_0}^{t_0+\omega} e^{(\mu_h+\delta_h+\gamma)s} \tilde{I}_a(s)\tilde{S}_h(s)ds}{e^{(\mu_h+\delta_h+\gamma)t_0}[e^{(\mu_h+\delta_h+\gamma)\omega} - 1]}.$$

Hence, $(\tilde{S}_a(t), \tilde{I}_a(t), \tilde{S}_h(t), \tilde{I}_h(t))$ is a unique periodic solution of system (21).

We now prove the necessary condition. If system (21) has a unique periodic solution, then the subsystem (10) must have at least one periodic solution. Suppose that the subsystem (10) has two periodic solutions $(\tilde{S}_a(t), \tilde{I}_a(t))$ and $(\phi_1(t), \phi_2(t))$. Then $(\tilde{S}_a(t), \tilde{I}_a(t), \tilde{S}_h(t), \tilde{I}_h(t))$ and $(\phi_1(t), \phi_2(t), \phi_3(t), \phi_4(t))$ are periodic solutions of system (21), where

$$\phi_3(t) = e^{-\beta_h \int_{t_0}^{t} \phi_2(s)ds - \mu_h t}\left(e^{\mu_h t_0}\phi_3^*(t_0) + \Pi_h \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} \phi_2(u)du} e^{\mu_h s} ds\right)$$

and

$$\phi_4(t) = e^{-(\mu_h+\delta_h+\gamma)t}\left(e^{(\mu_h+\delta_h+\gamma)t_0}\phi_4^*(t_0) + \beta_h \int_{t_0}^{t} e^{(\mu_h+\delta_h+\gamma)s}\phi_2(s)\phi_3(s)ds\right)$$

with $\phi_3^*(t_0) = \frac{\Pi_h \int_{t_0}^{t_0+\omega} e^{\beta_h \int_{t_0}^{s} \phi_2(u)du} e^{\mu_h s} ds}{e^{\mu_h t_0}[e^{\beta_h \int_0^{\omega} \phi_2(s)ds} e^{\mu_h \omega} - 1]}$ and $\phi_4^*(t_0) = \frac{\beta_h \int_{t_0}^{t_0+\omega} e^{(\mu_h+\delta_h+\gamma)s}\phi_2(s)\phi_3(s)ds}{e^{(\mu_h+\delta_h+\gamma)t_0}[e^{(\mu_h+\delta_h+\gamma)\omega} - 1]}$. This is a contradiction. Therefore, the subsystem (10) has a unique limit cycle. □

Theorem 3.10. *If $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$, then the unique periodic solution $(\tilde{S}_a(t), \tilde{I}_a(t), \tilde{S}_h(t), \tilde{I}_h(t))$ of the full system (21) is globally asymptotically stable if and only if the unique limit cycle $(\tilde{S}_a(t), \tilde{I}_a(t))$ of the subsystem (10) is globally asymptotically stable.*

Proof. The necessary condition is obvious. We only prove the sufficient condition.

By Theorem 3.5, the unique limit cycle Γ of the subsystem (10) is globally asymptotically stable in D_2 if $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{M_a+m_a}{2}$. For any solution $(S_a(t), I_a(t))$, by the results in Coppel [17] (p. 82) or Coddington and Levinson [14] (p. 323), we have

$$\lim_{t\to\infty}\left|S_a(t) - \tilde{S}_a(t+c)\right| = 0, \quad \lim_{t\to\infty}\left|I_a(t) - \tilde{I}_a(t+c)\right| = 0,$$

where c is some constant depending on $(S_a(t), I_a(t))$.

Next, we prove that $\lim_{t\to\infty}\left|S_h(t) - \tilde{S}_h(t+c)\right| = 0$. Since

$$\left|S_h(t) - \tilde{S}_h(t)\right| = e^{-\beta_h \int_{t_0}^{t} \tilde{I}_a(s)ds} e^{-\mu_h(t-t_0)}\left|S_h(t_0) - S_h^*(t_0)\right|,$$

we have

$$\lim_{t\to\infty}\left|S_h(t) - \tilde{S}_h(t)\right| = 0.$$

Since $\lim_{t\to\infty}\left|I_a(t) - \tilde{I}_a(t+c)\right| = 0$, $\forall \varepsilon > 0$, there exists a $T_1 > 0$ such that if $t > T_1$, then

$$\tilde{I}_a(t+c) - \varepsilon < I_a(t) < \tilde{I}_a(t+c) + \varepsilon. \tag{28}$$

We construct the following equations:

$$\frac{dS_h^-}{dt} = \Pi_h - \beta_h(\tilde{I}_a(t+c) + \varepsilon)S_h - \mu_h S_h, \tag{29}$$

$$\frac{dS_h^+}{dt} = \Pi_h - \beta_h(\tilde{I}_a(t+c) - \varepsilon)S_h - \mu_h S_h. \tag{30}$$

The Eq. (29) has a unique periodic solution $\tilde{S}_h^-(t)$ and

$$\lim_{t\to\infty}\left|S_h^-(t) - \tilde{S}_h^-(t)\right| = 0;$$

The Eq. (30) has a unique periodic solution $\tilde{S}_h^+(t)$ and

$$\lim_{t\to\infty}\left|S_h^+(t) - \tilde{S}_h^+(t)\right| = 0,$$

where,

$$\tilde{S}_h^-(t) = e^{-\beta_h \int_{t_0}^{t} (\tilde{I}_a(s+c)+\varepsilon)ds - \mu_h t}\left[e^{\mu_h t_0} S_h^*(t_0) + \Pi_h \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} [\tilde{I}_a(u+c)+\varepsilon]du} e^{\mu_h s} ds\right],$$

$$\tilde{S}_h^+(t) = e^{-\beta_h \int_{t_0}^{t} [\tilde{I}_a(s+c)-\varepsilon]ds - \mu_h t}\left[e^{\mu_h t_0} S_h^*(t_0) + \Pi_h \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} [\tilde{I}_a(u+c)-\varepsilon]du} e^{\mu_h s} ds\right],$$

$\tilde{S}_h^-(t)$ is defined on the three-dimensional cylinder $\Gamma^- \times \mathbb{R}^+ \times \mathbb{R}^+ = \Gamma^- \times [0,\infty) \times [0,\infty)$ and $\tilde{S}_h^+(t)$ is defined on the three-dimensional cylinder $\Gamma^+ \times \mathbb{R}^+ \times \mathbb{R}^+ = \Gamma^+ \times [0,\infty) \times [0,\infty)$, where $\Gamma^- = \{(\tilde{S}_a(t), \tilde{I}_a(t) + \varepsilon) : t \in [0,\omega]\}$ and $\Gamma^+ = \{(\tilde{S}_a(t), \tilde{I}_a(t) - \varepsilon) : t \in [0,\omega]\}$, $\mathbb{R}^+ = [0,\infty)$.

By the third equation of system (21) and the comparison theorem of ordinary differential equations, we have

$$S_h^-(t) < S_h(t) < S_h^+(t) \tag{31}$$

for $t > T_1$.

Next, we prove that

$$\lim_{t\to\infty}\left|\tilde{S}_h^-(t) - \tilde{S}_h(t+c)\right| = 0, \lim_{t\to\infty}\left|\tilde{S}_h^+(t) - \tilde{S}_h(t+c)\right| = 0.$$

Since

$$\frac{dS_h(t+c)}{dt} = \Pi_h - \beta_h I_a(t+c)S_h(t+c) - \mu_h S_h(t+c),$$

we have,

$$\tilde{S}_h(t+c) = e^{-\beta_h \int_{t_0}^{t} \tilde{I}_a(s+c)ds - \mu_h t}\left[e^{\mu_h t_0} S_h^*(t_0+c) + \Pi_h \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} \tilde{I}_a(u+c)du} e^{\mu_h s} ds\right].$$

Thus,

$$\begin{aligned}\tilde{S}_h^-(t) - \tilde{S}_h(t+c) =& e^{-\beta_h \int_{t_0}^{t} \tilde{I}_a(s+c)ds - \mu_h(t-t_0)}[e^{-\beta_h \varepsilon(t-t_0)} S_h^{-*}(t_0) - S_h^*(t_0+c)] \\ &+ e^{-\beta_h \int_{t_0}^{t} (\tilde{I}_a(s+c)+\varepsilon)ds - \mu_h t} \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} \tilde{I}_a(u+c)du} e^{\mu_h s} \\ &\times [e^{\beta_h \varepsilon(s-t_0)} - 1]ds,\end{aligned}$$

$$\begin{aligned}\tilde{S}_h^+(t) - \tilde{S}_h(t+c) =& e^{-\beta_h \int_{t_0}^{t} \tilde{I}_a(s+c)ds - \mu_h(t-t_0)}[e^{-\beta_h \varepsilon(t-t_0)} S_h^{-*}(t_0) - S_h^*(t_0+c)] \\ &+ e^{-\beta_h \int_{t_0}^{t} (\tilde{I}_a(s+c)-\varepsilon)ds - \mu_h t} \int_{t_0}^{t} e^{\beta_h \int_{t_0}^{s} \tilde{I}_a(u+c)du} e^{\mu_h s} \\ &\times [e^{-\beta_h \varepsilon(s-t_0)} - 1]ds.\end{aligned}$$

If ε is small enough, then

$$\lim_{t\to\infty}\left|\tilde{S}_h^-(t)-\tilde{S}_h(t+c)\right|=0,\quad \lim_{t\to\infty}\left|\tilde{S}_h^+(t)-\tilde{S}_h(t+c)\right|=0.$$

By (31), we have

$$S_h^-(t)-\tilde{S}_h(t+c)<S_h(t)-\tilde{S}_h(t+c)<S_h^+(t)-\tilde{S}_h(t+c).$$

Since

$$\left|S_h^-(t)-\tilde{S}_h(t+c)\right|\le\left|S_h^-(t)-\tilde{S}_h^-(t)\right|+\left|\tilde{S}_h^-(t)-\tilde{S}_h(t+c)\right|,$$

$$\left|S_h^+(t)-\tilde{S}_h(t+c)\right|\le\left|S_h^+(t)-\tilde{S}_h^+(t)\right|+\left|\tilde{S}_h^+(t)-\tilde{S}_h(t+c)\right|,$$

we have

$$\lim_{t\to\infty}\left|S_h^-(t)-\tilde{S}_h(t+c)\right|=0,\ \lim_{t\to\infty}\left|S_h^+(t)-\tilde{S}_h(t+c)\right|=0.$$

Hence,

$$\lim_{t\to\infty}\left|S_h(t)-\tilde{S}_h(t+c)\right|=0.$$

Similarly, we can prove $\lim_{t\to\infty}\left|I_h(t)-\tilde{I}_h(t+c)\right|=0$. □

Let $F_i=\{(S_a,I_a,S_h,I_h,R_h)|(S_a,I_a)\in D_i,S_h\ge 0,I_h\ge 0,R_h\ge 0\}$ with $i=1,2$. Finally, we have the following results on the global dynamics of the original system (9) with Allee effect for the avain population.

Corollary 3.11. *(i) The disease-free equilibrium $H_1(0,0,S_h^*,0,0)$ of model (9) avian Allee effect is always globally asymptotically stable in F_1; (ii) if $\frac{\mu_a+\delta_a}{\beta_a}\ge M_a$, then the disease-free equilibrium $H_3(M_a,0,S_h^*,0,0)$ of model (9) avian Allee effect is globally asymptotically stable in F_2; (iii) if $\frac{M_a+m_a}{2}\le\frac{\mu_a+\delta_a}{\beta_a}<M_a$, then the unique endemic equilibrium $H_4(S_a^{**},I_a^{**},S_h^{**},I_h^{**},R_h^{**})$ of model (9) avian Allee effect is globally asymptotically stable in F_2; (iv) if $m_a<\frac{\mu_a+\delta_a}{\beta_a}<\frac{M_a+m_a}{2}$, then there is a unique periodic solution of model (9) avian Allee effect at the neighborhood of the endemic equilibrium $H_4(S_a^{**},I_a^{**},S_h^{**},I_h^{**},R_h^{**})$ which is globally asymptotically stable in F_2.*

4. Numerical simulations

4.1. Comparison and numerical simulations of the basic reproduction numbers

By the results in Sections 2 and 3, we know that the basic reproduction numbers of systems (4) and (9) are $\mathcal{R}_{0,1}=\frac{K_a\beta_a}{\mu_a+\delta_a}$ and $\mathcal{R}_{0,2}=\frac{\beta_a(M_a+m_a)(\mu_a+\delta_a)}{(\mu_a+\delta_a)^2+M_am_a\beta_a^2}$, respectively. Now we keep the maximal carrying capacity in systems (4) and (9) identical (i.e., $K_a=M_a$), then we can easily obtain that

$$\begin{aligned}\mathcal{R}_{0,2}-\mathcal{R}_{0,1}&=\frac{\beta_a(M_a+m_a)(\mu_a+\delta_a)}{(\mu_a+\delta_a)^2+M_am_a\beta_a^2}-\frac{\beta_aK_a}{\mu_a+\delta_a}\\&=\frac{\beta_a(M_a+m_a)(\mu_a+\delta_a)}{(\mu_a+\delta_a)^2+M_am_a\beta_a^2}-\frac{\beta_aM_a}{\mu_a+\delta_a}\\&=\frac{\beta_am_a(\mu_a+\delta_a+\beta_aM_a)\beta_a(\frac{\mu_a+\delta_a}{\beta_a}-M_a)}{(\mu_a+\delta_a)[(\mu_a+\delta_a)^2+M_am_a\beta_a^2]}.\end{aligned}$$

Thus, we have the following results:

(i) If $K_a=M_a$ and $\frac{\mu_a+\delta_a}{\beta_a}\le m_a$, then $\mathcal{R}_{0,1}>1\ge\mathcal{R}_{0,2}$;
(ii) If $K_a=M_a$ and $m_a<\frac{\mu_a+\delta_a}{\beta_a}<M_a$, then $\mathcal{R}_{0,1}>\mathcal{R}_{0,2}>1$;
(iii) If $K_a=M_a$ and $\frac{\mu_a+\delta_a}{\beta_a}=M_a$, then $\mathcal{R}_{0,1}=\mathcal{R}_{0,2}=1$;
(iv) If $K_a=M_a$ and $\frac{\mu_a+\delta_a}{\beta_a}>M_a$, then $\mathcal{R}_{0,1}<\mathcal{R}_{0,2}<1$.

In order to substantiate the above results, we present some numerical simulations as follows. First we fix some parameters. We assume human and the wild avian can survive 70 years and 8 years, respectively. Hence the natural death rates of human and wild avian population are $\mu_h=3.91*10^{-5}$ and $\mu_a=3.4246*10^{-4}$ per day, respectively. We also assume that the disease-related death rates of the infected avian and the infective human population are $\delta_a=4*10^{-4}$ and $\delta_h=0.3445$ per day, respectively; the intrinsic growth rate of the avian population is $r_a=5*10^{-3}$; the maximal and critical carrying capacities of the avian population are $K_a=M_a=50,000$ and $m_a=800$, respectively. The numerical simulations of the basic reproduction numbers of both systems are given in Fig. 2.

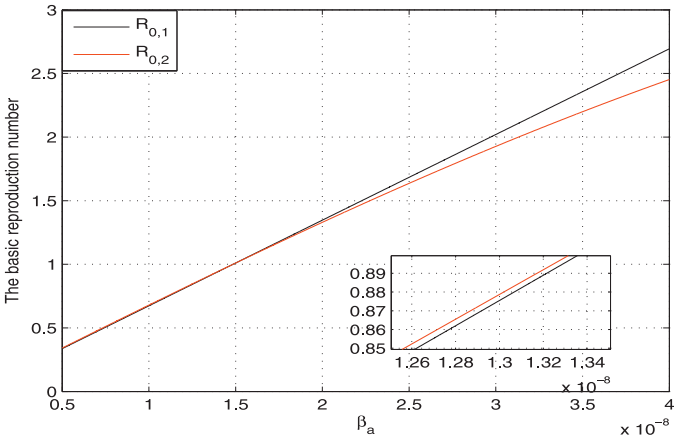


Fig. 2. The plot shows the changes of $\mathcal{R}_{0,1}$ and $\mathcal{R}_{0,2}$ with respect to β_a.

From Fig. 2, we know that if $\beta_a<1*10^{-8}$, then $\mathcal{R}_{0,1}<\mathcal{R}_{0,2}<1$; if $\beta_a>2*10^{-8}$, then $\mathcal{R}_{0,1}>\mathcal{R}_{0,2}>1$; if $\beta_a=1.48492*10^{-8}$, then $\mathcal{R}_{0,1}=\mathcal{R}_{0,2}=1$. These results support the theoretical conclusions.

4.2. Numerical simulations of the models

Noted that the expression $\frac{\mu_a+\delta_a}{\beta_a}$ is a key quantity. The relationship between $\frac{\mu_a+\delta_a}{\beta_a}$ and K_a or m_a, M_a determines whether the avian influenza disappears or not. When μ_a,δ_a, m_a and M_a are fixed, then β_a is a key parameter. In this subsection, we investigate the influence of parameter β_a on the number of infected humans by performing some numerical simulations. Besides the fixed parameters in the above subsection, we further assume that the recovery rate of infectious human individuals is 0.1 per day, so $\gamma=0.1$. In general, avian influenza mainly outbreaks in a specific location. We estimate that the number of susceptible avian population is between 100,000 and 1,000,000, the number of infective avian population is between 0 and 100, and the number of susceptible human population is between 100,000 and 1,000,000 in the region. So we choose the initial values as $(S_a(0),I_a(0),S_h(0),I_h(0),R_h(0))=(100,000,100,100,000,1,0)$.

Firstly, we study the influence of parameter β_a on the number of infective individuals of model (4) with logistic avian growth. When parameters K_a, μ_a, and δ_a are fixed, the threshold value $\beta_a^*=1.48492\times10^{-8}$ such that $\mathcal{R}_{0,1}=1$. If $\beta_a\le\beta_a^*$, the disease disappears and the solution $I_h(t)$ is asymptotically stable and converges to the disease-free state value (see Fig. 3(A)); If $\beta_a>\beta_a^*$, the endemic disease is prevalent, the solution $I_h(t)$ is asymptotically stable and converges to the endemic state value (see Fig. 3(B)). Furthermore, we can also observe that the peak value of $I_h(t)$ increases with β_a increasing from Fig. 3.

Secondly, we investigate the influence of parameter β_a on the number of infective individuals of model (9) with avian Allee effect. Recall that $\mathcal{R}_{0,2}=1\Leftrightarrow\frac{\mu_a+\delta_a}{\beta_a}=m_a$ or $\frac{\mu_a+\delta_a}{\beta_a}=M_a$. Then for fixed parameters μ_a, δ_a, m_a and M_a, the threshold value $\beta_a^*=1.48492\times10^{-8}$ or 9.28075×10^{-7} such that $\mathcal{R}_{0,2}=1$. According to Corollary 3.11, for the above parameter and initial values, if $\beta_a\ge9.28075\times10^{-7}$, the disease disappears and the solu-

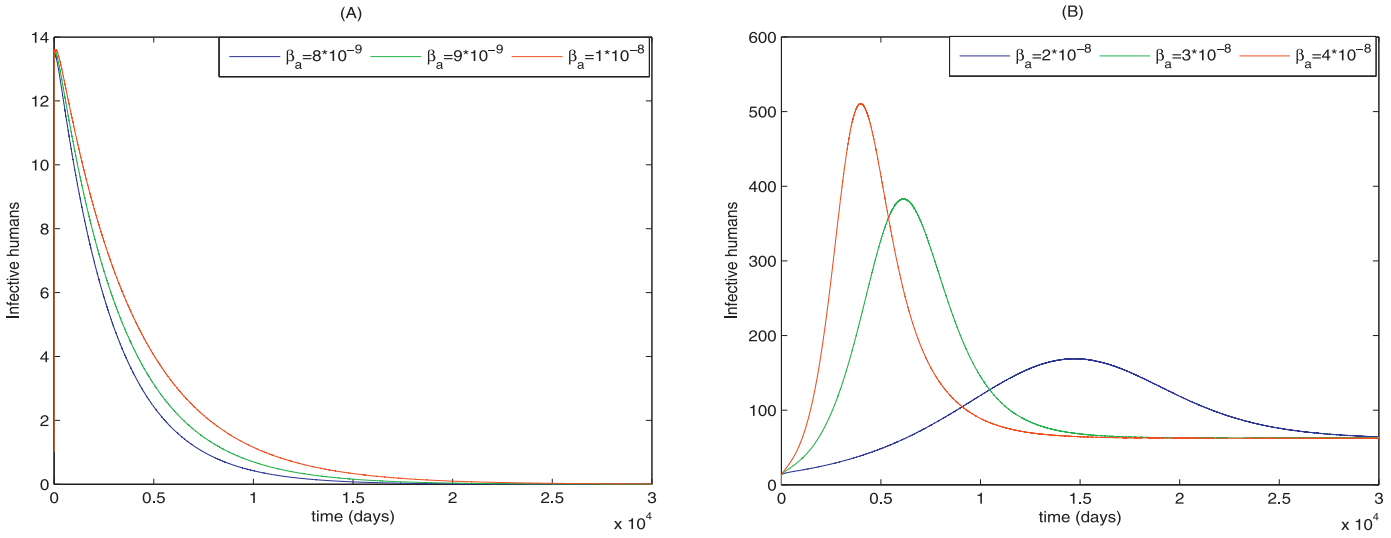


Fig. 3. The plots display the changes of $I_h(t)$ with β_a varying where $\beta_h = 6 \times 10^{-9}$. (A) Solutions $I_h(t)$ are asymptotically stable and converge to the disease-free state value; (B) Solutions $I_h(t)$ are asymptotically stable and converges to the endemic state value.

tion $I_h(t)$ is asymptotically stable in the region F_2 (see Fig. 4(A)); if $2.9231 \times 10^{-8} \leq \beta_a < 9.28075 \times 10^{-7}$, the endemic disease is prevalent, the solution $I_h(t)$ is asymptotically stable in the region F_2 (see Fig. 4(B)).

Thirdly, we simulate the periodic solutions of model (9) with avian Allee effect. Parameters $\mu_a, \delta_a, \mu_h, \gamma, M_a, m_a$ and r_a are chosen as before. Other parameters and initial values are selected as follows: $\Pi_h = 30$, $\beta_h = 6 \times 10^{-8}$, $\delta_h = 0.3445$, $(S_a(0), I_a(0), S_h(0), I_h(0), R_h(0)) = (1,000,000, 2000, 100,000, 30, 5)$. When $\beta_a = 2.57 \times 10^{-7}$ or $\beta_a = 2.58 \times 10^{-7}$, then $\frac{\mu_a+\delta_a}{\beta_a}$ is between m_a and $\frac{M_a+m_a}{2}$, which satisfies the condition of Corollary 3.11. Hence, there is a unique periodic solution of system (9) in the neighborhood of the endemic equilibrium which is globally asymptotically stable in F_2 (see Fig. 4(C)).

Finally, we examine the influence of parameter β_h on the number of infective individuals of model (4) with logistic avian growth and model (9) with avian Allee effect. When the birds are at endemic state, we can observe that the human population is also at endemic state even if bird-to-human contact rate (β_h) is reduced by 99% (see Fig. 5). Furthermore, we can also observe that the peak value of $I_h(t)$ and the endemic state value of these systems increase when β_h is increasing (see Fig. 5).

5. Discussion

It is believed that the H7N9 was transferred to ducks in China by wild birds through migration along the East Asian flyway [40]. Experimental data [46] showed that it is conceivable that passerine birds may serve as vectors for transmission of H7N9 virus to domestic poultry [32], which in turn transmitted the virus to humans through live-poultry markets [4,11], . After the first outbreak in the spring of 2013, the H7N9 avian influenza resurged in China from November 2013 to May 2014, from November 2014 to June 2015, and from November 2015 to June 2016 (WHO [58]). The data strongly indicate that it is becoming seasonal and persistent like the H5N1 avian influenza. Tuncer and Martcheva [52] used periodic contact/incidence rates to model the seasonality in H5N1 avian influenza transmission. Since the live-poultry markets are open all year around, the contact/incidence rates are more likely to be constant in this case. Cross-sectional surveys conducted in China after the outbreaks of the avian influenza A H7N9 viruses show a high degree of awareness of human avian influenza in both urban and rural populations, a higher level of proper hygienic practice among urban residents, and in particular a dramatically reduced number of visits to live markets in urban population after the H7N9 outbreak in 2013. Taking into account the psychological effect toward avian influenza in the human population, we [41] proposed a bird-to-human transmission model in which the avian population exhibits saturation effect. However, our study shows that the saturation effect within avian population and the psychological effect in human population cannot change the stability of equilibria but can affect the number of infected humans if the disease is prevalent, so there is no periodic solutions. In Liu et al. [42], we also took account of the incubation periods of avian influenza A virus, constructed 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, and obtained global asymptotical stability of equilibria of the system. Once again the time delays in such models do not induce oscillations. Chen et al. [10] argued that the lack of understanding of the virus ecology in birds has resulted in the persistent circulating of H7N9 in China. Since the H7N9 virus does not induce clinical signs in poultry and is classified as a low pathogenicity avian influenza virus [46], we believe that the population dynamics of avian species contribute significantly to the persistence and potential periodicity of the virus in avian as well as human populations. Note that it has been observed [49] the growth of some avian populations exhibit Allee effect due to habitat destruction, spread of alien species, pollution, and diseases.

In this paper, to study the transmission dynamics of avian influenza from birds to humans we constructed ordinary differential equation models with two different growth laws for the avian population: (i) logistic growth and (ii) Allee effect. We obtained a threshold value for the prevalence of avian influenza and discussed the local or global asymptotical stability of each equilibrium of these systems. Our results indicate that the asymptotic dynamics of the model with logistic growth for the avian population are completely determined by the basic reproduction number: the disease-free equilibrium exists and is locally asymptotically stable if the basic reproduction number is less than the unity; the disease-free equilibrium becomes unstable and the endemic equilibrium exists and is locally asymptotically stable if the basic

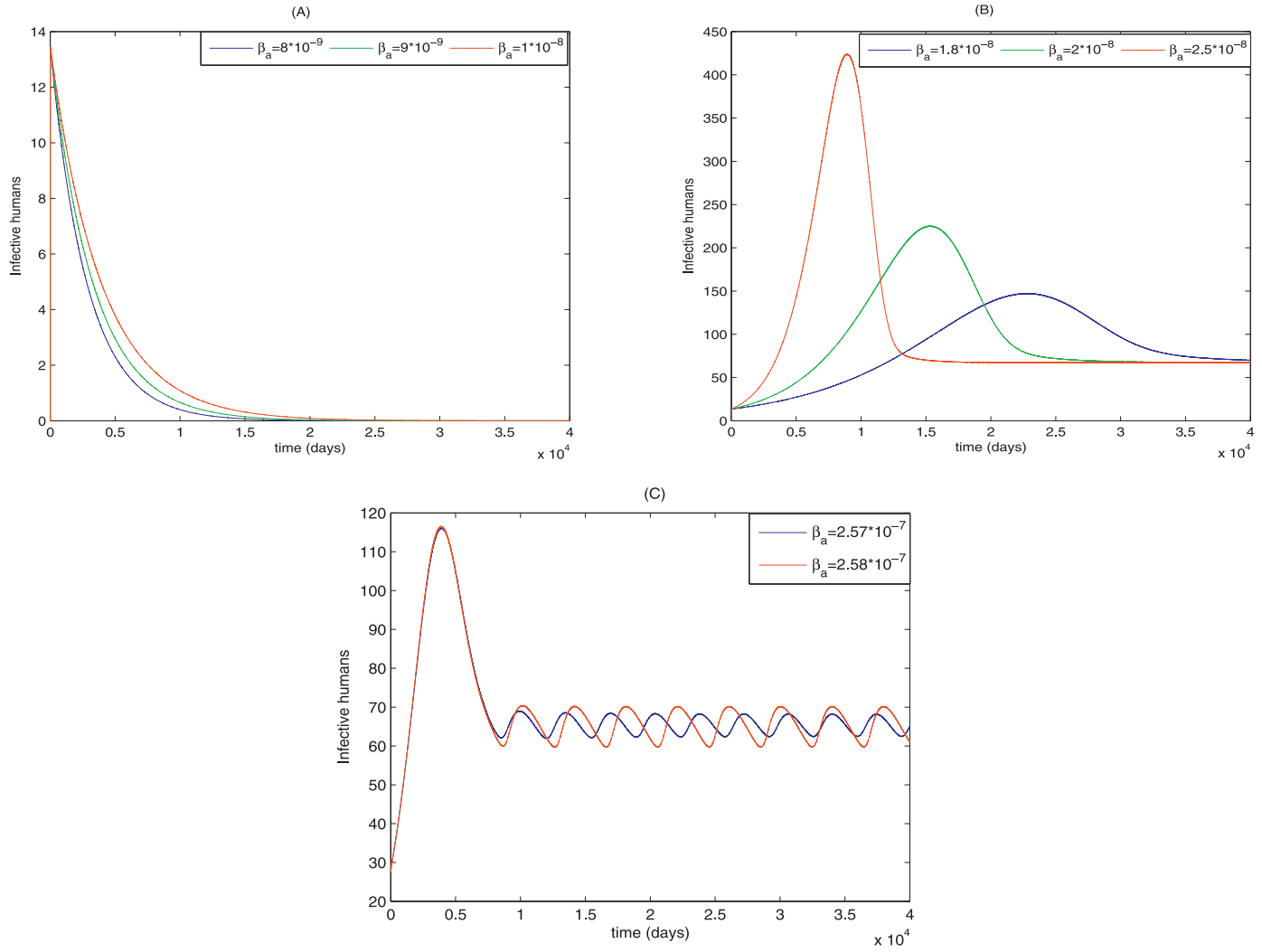


Fig. 4. The plots bring to light the changes of $I_h(t)$ with β_a varying. (A) $I_h(t)$ is asymptotically stable in the region F_2 and converges to the disease-free state value where the disease-free equilibrium is $(50,000, 0, 767263.43, 0, 0)$; (B) $I_h(t)$ is asymptotically stable in the region F_2 and converges to the endemic state value; (C) The periodic solution $I_h(t)$ is asymptotically stable in F_2.

reproduction number is greater than the unity. Global asymptotic stability of these equilibria were also established by using Liapunov function method and LaSalle's invariance principle. For the model with Allee effect for the avian population, beside stability results it was shown that periodic solutions exists via Hopf bifurcations. Global stability of the periodic solutions was also considered.

Recall that for the system (4) with logistic avian growth, the basic reproduction number was given as follows

$$\mathcal{R}_{0,1} = \frac{K_a \beta_a}{\mu_a + \delta_a}. \tag{32}$$

There were two disease-free equilibria given by $A(0, 0, S_h^*, 0, 0)$ and $B(K_a, 0, S_h^*, 0, 0)$, where $S_h^* = \frac{\Pi_h}{\mu_h}$. If $\mathcal{R}_{0,1} > 1$, and a unique endemic equilibrium given by $C(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$, where

$$S_a^{**} = \frac{\mu_a + \delta_a}{\beta_a},\ I_a^{**} = \frac{r_a(\mu_a + \delta_a)}{K_a \beta_a^2}(\mathcal{R}_{0,1} - 1), \tag{33}$$

$$S_h^{**} = \frac{\Pi_h}{\beta_h I_a^{**} + \mu_h},\ I_h^{**} = \frac{\beta_h I_a^{**} S_h^{**}}{\mu_h + \delta_h + \gamma},\ R_h^{**} = \frac{\gamma I_h^{**}}{\mu_h}. \tag{34}$$

We only consider the biologically meaningful equilibria $B(K_a, 0, S_h^*, 0, 0)$ and $C(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$, the results about system (4) with logistic avian growth can be summarized in the following chart (BRN=basic reproduction number).

For the system (9) with Allee effect in the avian population, the basic reproduction number is given by

$$\mathcal{R}_{0,2} = \frac{\beta_a (M_a + m_a)(\mu_a + \delta_a)}{(\mu_a + \delta_a)^2 + M_a m_a \beta_a^2}. \tag{35}$$

There are three disease-free equilibria given by $H_1(0, 0, S_h^*, 0, 0)$, $H_2(m_a, 0, S_h^*, 0, 0)$, and $H_3(M_a, 0, S_h^*, 0, 0)$, where $S_h^* = \frac{\Pi_h}{\mu_h}$, and if $\mathcal{R}_{0,2} > 1$, there is also a unique endemic equilibrium given by $H_4(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$, where

$$S_a^{**} = \frac{\mu_a + \delta_a}{\beta_a},\ I_a^{**} = \frac{r_a}{\beta_a} \frac{\beta_a^2 M_a m_a + (\mu_a + \delta_a)^2}{M_a m_a \beta_a^2}(\mathcal{R}_{0,2} - 1), \tag{36}$$

$$S_h^{**} = \frac{\Pi_h}{\beta_h I_a^{**} + \mu_h},\ I_h^{**} = \frac{\beta_h I_a^{**} S_h^{**}}{\mu_h + \delta_h + \gamma},\ R_h^{**} = \frac{\gamma I_h^{**}}{\mu_h}. \tag{37}$$

Similarly, considering only the biologically meaningful equilibria we can summarize the results about system (9) with Allee effect in the avian population in the following chart (GSPS=globally stable periodic solution).

Through the analysis, we found that if the maximal carrying capacity of the avian population of each system is the same (i.e., $K_a = M_a$) and $m_a < \frac{\mu_a + \delta_a}{\beta_a} < M_a$, then $\mathcal{R}_{0,1} > \mathcal{R}_{0,2} > 1$, which indicates that the transmission speed of the avian influenza virus of

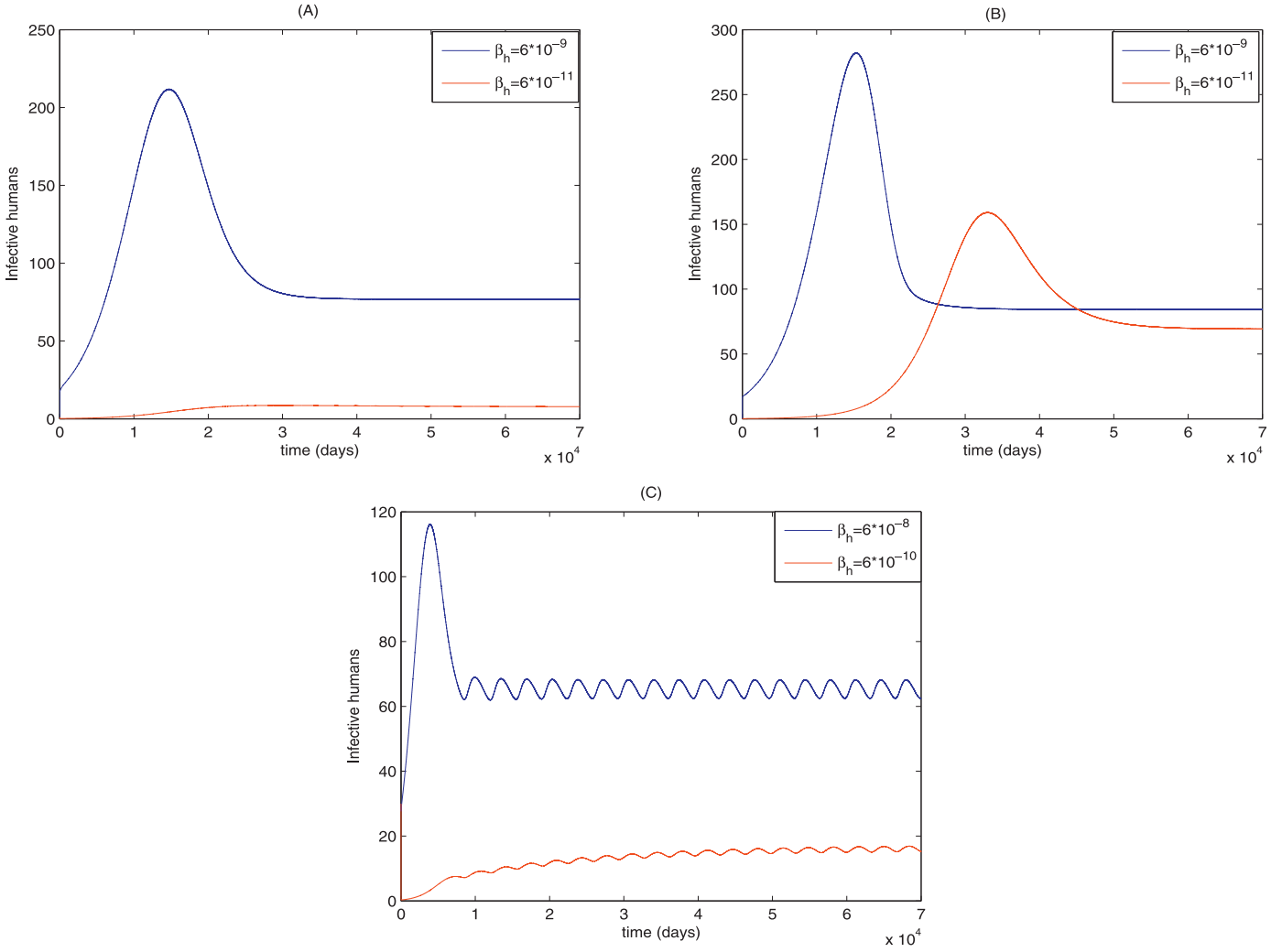


Fig. 5. The plots reveal the changes of $I_h(t)$ with β_h varying. (A) $I_h(t)$ of system (4) with avian logistic growth is asymptotically stable and converges to the endemic state value; (B) $I_h(t)$ of system (9) with avian Allee effect converges to the endemic state value; (C) The periodic solution $I_h(t)$ of system (9) with avian Allee effect is asymptotically stable.

system (4) (with logistic growth) is greater than system (9) (with Allee effect) and the endemic disease of the two systems is prevalent; if the maximal carrying capacity of each system is the same and $\frac{\mu_a+\delta_a}{\beta_a} \leq m_a$, then $\mathcal{R}_{0,1} > 1 \geq \mathcal{R}_{0,2}$, which indicates that the endemic disease of system (4) is prevalent but the endemic disease of system (9) disappears; if the maximal carrying capacity of each system is the same and $\frac{\mu_a+\delta_a}{\beta_a} > M_a$, then $\mathcal{R}_{0,1} < \mathcal{R}_{0,2} < 1$, which indicates that the endemic disease of both systems disappears. Therefore, we can make the quantity $\frac{\mu_a+\delta_a}{\beta_a}$ greater than the maximal carrying capacity of the avian population to control the disease by reducing β_a (transmission rate from infective avian to susceptible avian) or increasing μ_a (natural death rate of the avian population) and δ_a (disease-related death rate of the infected avian). The effective methods will be to reduce the transmission between the susceptible and infective avian populations and isolating or culling the infective birds if necessary.

For the system (4) with logistic avian growth, from Table 1 we can see that if $\frac{\mu_a+\delta_a}{\beta_a} > K_a$ so that $\mathcal{R}_{0,1} < 1$, then the disease-free equilibrium $B(K_a, 0, S_h^*, 0, 0)$ is globally stable; if $\frac{\mu_a+\delta_a}{\beta_a} < K_a$ so that $\mathcal{R}_{0,1} > 1$, then the disease-free equilibrium $B(K_a, 0, S_h^*, 0, 0)$ becomes unstable and the endemic equilibrium $C(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$ exists and is globally stable. For the system (9) with Allee effect in the avian population, the dynamics are more interesting.

Table 1
Stability chart for system (4) with logistic avian growth.

Conditions	BRN	$B(K_a, 0, S_h^*, 0, 0)$	$C(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$
$\frac{\mu_a+\delta_a}{\beta_a} > K_a$	$\mathcal{R}_{0,1} < 1$	Globally stable	Does not exist
$\frac{\mu_a+\delta_a}{\beta_a} < K_a$	$\mathcal{R}_{0,1} > 1$	Unstable	Globally stable

If $M_a < \frac{\mu_a+\delta_a}{\beta_a}$ (where M_a is the maximal carrying capacity of the avian population) so that $\mathcal{R}_{0,2} < 1$, then the disease-free equilibrium $H_2(m_a, 0, S_h^*, 0, 0)$ with less avian density (where m_a is the critical carry capacity of the avian population, $m_a < M_a$) is unstable and the disease-free equilibrium $H_3(M_a, 0, S_h^*, 0, 0)$ with more avian density is globally stable; if β_a increases or $\mu_a + \delta_a$ increases such that $\frac{m_a+M_a}{2} < \frac{\mu_a+\delta_a}{\beta_a} < M_a$ so $\mathcal{R}_{0,2} > 1$, then the disease-free equilibrium $H_3(M_a, 0, S_h^*, 0, 0)$ with more avian density becomes unstable and an endemic equilibrium $H_4(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$ exists and is globally stable; if $m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{m_a+M_a}{2}$ so $\mathcal{R}_{0,2} > 1$ remains hold, then the endemic equilibrium $H_4(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$ becomes unstable and there is a globally stable periodic orbit bifurcated from it; if, further, $\frac{\mu_a+\delta_a}{\beta_a} < m_a$ so that $\mathcal{R}_{0,2} < 1$, both disease-free equilibria $H_2(m_a, 0, S_h^*, 0, 0)$ and $H_3(M_a, 0, S_h^*, 0, 0)$ exist and the disease die out. We have provided references to sup-

Table 2
Stability chart for system (9) with Allee effect in the avian population.

Conditions	BRN	$H_2(m_a, 0, S_h^*, 0, 0)$	$H_4(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**}, R_h^{**})$	$H_3(M_a, 0, S_h^*, 0, 0)$
$M_a < \frac{\mu_a+\delta_a}{\beta_a}$	$\mathcal{R}_{0,2} < 1$	Unstable	Does not exists	Globally stable
$\frac{m_a+M_a}{2} < \frac{\mu_a+\delta_a}{\beta_a} < M_a$	$\mathcal{R}_{0,2} > 1$	Unstable	Globally stable	Unstable
$m_a < \frac{\mu_a+\delta_a}{\beta_a} < \frac{m_a+M_a}{2}$	$\mathcal{R}_{0,2} > 1$	Unstable	Unstable (GSPS)	Unstable
$\frac{\mu_a+\delta_a}{\beta_a} < m_a$	$\mathcal{R}_{0,2} < 1$	Unstable	Does not exist	Unstable

port the observation that the H7N9 avian virus has been transmitted from wild birds to domestic poultry and then to humans and pointed out some potential avian species that are believed to be responsible for the cross-species transmission. Though we are not able to obtain data on specific avian species and apply our models and conclusions directly, we believe that our results on the existence and stability of periodic solutions in the model with Allee effect for the avian population may be useful in understanding the seasonal/periodic outbreaks of the H7N9 avian influenza.

From the expressions of the basic reproduction numbers $\mathcal{R}_{0,1}$ and $\mathcal{R}_{0,2}$ defined in (32) and (35), respectively, and the existence and stability conditions listed in Tables 1 and 2, it seems that the parameters involving human population do not appear and the overall disease could be controlled if it can be controlled in birds. Theoretically it is true: if there is no disease among birds then there is no outbreaks in humans since there is no human-to-human transmission yet. However, H7N9 is classified as a low pathogenicity avian influenza virus and causes no symptoms and mortality in birds. Controlling the disease in the avian population is very difficult and the basic reproduction numbers do not provide effective control measures for the human population. Notice that β_h (the transmission rate from infective avian to susceptible human) appears in the expressions (34) and (37) for the steady state values of I_h^{**}, the number of infective human individuals. In fact, it should be understood that $\beta_h = c_h p_h$, where c_h is the contact rate between a susceptible human and an infective bird and p_h is the probability of transmitting the virus per contact. Thus, to prevent spread of the avian influenza virus from birds to humans, we suggest to reduce contacting poultry and to take extra protection when contacting is necessary. If either $c_h = 0$ or $p_h = 0$, then $I_h^{**} = 0$ and there is no outbreaks in humans. This also explains that in the spring of 2013, when the poultry markets in Jiangsu, Shanghai, and Zhejiang were temporarily closed, the outbreak was controlled soon.

Our study also indicates that if birds are at endemic state, then the human population is also at endemic state even if the bird-to-human contact rate (β_h) is reduced by 99% (see Fig. 5). Furthermore, we can see that the peak value of $I_h(t)$ and the endemic state value of these systems increase when β_h is increasing (see Fig. 5). Our models results may not accurately describe all situations, but they can explain most of situations because perfect prevention (i.e. 100% reduction of β_h) is unlikely to happen in reality.

Note that asymptotic dynamics of avian influenza models consisted of bird and human populations, in particular global stability in such models, have been studied by other researchers, see for example [28] and [24]. Constant growth was assumed for the avian population in these studies. Compared to their models and results, our main contributions are as follows: First, we assumed that the growth rate of the avian population follows either the logistic law or the Allee effect, which is more general than the constant growth rate. Secondly, we not only obtained global stability of the disease-free and endemic equilibria but also established the global stability of the periodic solutions generated via Hopf bifurcations. To the best of our knowledge, there are very few results on the global stability of periodic solutions for epidemic models. Thus, our techniques could be useful to study the existence and global stability of periodic solutions in similar ecological and epidemiological models.

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 [5,22,43,52]. 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 are considering such a model, estimating model parameters, and trying to simulate the datasets on reported human H7N9 cases from China. The results will be reported somewhere else in the future.

Since the H7N9 virus is classified as a low pathogenicity avian influenza virus (LPAIV) [46], we ignored the recovery class of birds in our models. The model of Vaidya and Wahl [53] predicts that birds infected by avian influenza virus lose their immunity in approximately 4 weeks, it would be interesting to take account of the recovery class of birds in future models of avian influenza.

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Appendix A

In this section, we prove the global stability of the full system (4) with logistic avian growth and the full system (9) with avian Allee effect by using LaSalle's invariance principle.

A.1. Boundedness of solutions

For system (4) with logistic avian growth, we have the following result.

Lemma A.1. *All solutions of system (4) with initial values in $\mathbb{R}^5_+$ are bounded.*

Proof. Define a function $\eta = S_a + I_a + S_h + I_h + R_h$, then for each ν: $0 < \nu < \min\{\mu_a, \mu_h\}$, the following inequality holds:

$$\frac{d\eta}{dt} + \nu\eta \le \frac{K_a(r_a+\nu)^2}{4r_a} + \Pi_h = \phi.$$

Applying the theory of differential inequalities ([38]), we obtain that

$$0 < \eta(S_a, I_a, S_h, I_h, R_h)(t) < \frac{\phi}{\nu}(1 - e^{-\nu t}) + \eta(S_a(0), I_a(0), S_h(0), I_h(0), R_h(0))e^{-\nu t},$$

and for $t \to \infty$ we have $0 < \eta < \frac{\phi}{\nu}$.

For $\epsilon = 1$, there exists $t_0 > 0$, if $t > t_0$ then $(S_a + I_a + S_h + I_h + R_h)(t) < \frac{\phi}{\nu} + 1$. Furthermore, $(S_a + I_a + S_h + I_h + R_h)(t)$ is continuous on the interval $[0, t_0]$, so $(S_a + I_a + S_h + I_h + R_h)(t)$ has a maximum value A^* on the interval $[0, t_0]$. Choose $M = \max\{A^*, \frac{\phi}{\nu} + 1\}$, then $(S_a + I_a + S_h + I_h + R_h)(t) \le M$. Hence all the solutions of system (4) with initial values in $\mathbb{R}^5_+$ are confined in the region $D = \{(S_a, I_a, S_h, I_h, R_h) \in \mathbb{R}^5_+ : S_a + I_a + S_h + I_h + R_h \le M\}$. □

Similarly, for system (9) with avian Alle effect, we have the following result.

Lemma A.2. *All solutions of system (9) with initial values in* $\mathbb{R}^5_+$ *are uniformly bounded in the region* $F = \{(S_a, I_a, S_h, I_h, R_h) \in \mathbb{R}^5_+ : S_a + I_a + S_h + I_h + R_h \le \max\{A_0, \frac{\rho}{\omega} + 1\}\}$, *where* $\omega : 0 < \omega < \min\{r_a, \mu_a + \delta_a, \mu_h\}$, $\rho = \frac{4r_a(M_a+m_a)^3}{27M_a m_a} + \Pi_h$, A_0 *is the maximum value of* $(S_a + I_a + S_h + I_h + R_h)(t)$ *on interval* $[0, t_1]$.

Proof. The proof is similar to that of Lemma A.1, we omit it. □

A.2. Another proof of Theorem 2.6

Proof. (i) According to Lemma 2.3, the disease-free equilibrium B_a of system (5) is globally asymptotically stable if $\mathcal{R}_{0,1} \le 1$ which implies that $S_a \to K_a$ and $I_h \to 0$ if $t \to \infty$. Hence, we analyze the global stability of B_{ah} only at the region $D_{01} = \{(S_a, I_a, S_h, I_h) | S_a = K_a, I_a = 0, S_a + I_a + S_h + I_h \le M\}$. Consider system (6) with the avian components already at the disease-free steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = -(\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{38}$$

Choose a Liapunov function as follows

$$V_{21} = S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*} + I_h,$$

then,

$$\begin{aligned} \left.\frac{dV_{21}}{dt}\right|_{(38)} &= \frac{S_h - S_h^*}{S_h}(\Pi_h - \mu_h S_h) - (\mu_h + \delta_h + \gamma) I_h \\ &= -\frac{\mu_h}{S_h}(S_h - S_h^*)^2 - (\mu_h + \delta_h + \gamma) I_h \le 0. \end{aligned}$$

Since $D_{01} = \{(S_a, I_a, S_h, I_h) | S_a = K_a, I_a = 0, S_a + I_a + S_h + I_h \le M : \frac{dV_{21}}{dt} = 0\} = \{(S_a, I_a, S_h, I_h) : S_a = K_a, I_a = 0, S_h = S_h^*, I_h = 0\} = \{B_{ah}\}$, according to LaSalle's invariance principle (Hale [25]), the equilibrium B_{ah} is globally asymptotically stable for positive trajectories.

(ii) Similarly, by Lemma 2.3, the endemic equilibrium C_a of system (5) is globally asymptotically stable if $\mathcal{R}_{0,1} > 1$ which shows that $S_a \to S_a^{**}$ and $I_a \to I_a^{**}$ if $t \to \infty$. We consider the global stability of C_{ah} only at the region $D_{02} = \{(S_a, I_a, S_h, I_h) | S_a = S_a^{**}, I_a = I_a^{**}, S_a + I_a + S_h + I_h \le M\}$. Consider system (6) with the avian components already at the endemic steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \beta_h I_a^{**} S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_h I_a^{**} S_h - (\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{39}$$

Choose the following Liapunov function

$$V_{22} = S_h^{**}\left(\frac{S_h}{S_h^{**}} - \ln \frac{S_h}{S_h^{**}}\right) + I_h^{**}\left(\frac{I_h}{I_h^{**}} - \ln \frac{I_h}{I_h^{**}}\right),$$

According to the proof of Theorem 2.6(ii), we have $\left.\frac{dV_{22}}{dt}\right|_{(39)} \le 0$. Due to $D_{02} = \{(S_a, I_a, S_h, I_h) | S_a = S_a^{**}, I_a = I_a^{**}, S_a + I_a + S_h + I_h \le M : \frac{dV_{22}}{dt} = 0\} = \{(S_a^{**}, I_a^{**}, S_h^{**}, I_h^{**})\} = \{C_{ah}\}$, by the LaSalle's invariance principle, the endemic equilibrium C_{ah} is globally asymptotically stable. □

A.3. Another proof of Theorem 3.8

Set $E_1 = \{(S_a, I_a, S_h, I_h) : (S_a, I_a) \in D_1, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\}\}$ and $E_2 = \{(S_a, I_a, S_h, I_h) : (S_a, I_a) \in D_2, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\}\}$, where D_1 and D_2 are defined in Theorem 3.5.

Proof. (i) If $(S_a, I_a, S_h, I_h) \in E_1$, then $(S_a, I_a) \in D_1$. According to Theorem 3.5, the disease-free equilibrium O of the avian-only subsystem (10) is always globally asymptotically stable in the region D_1 which implies that $S_a \to 0$ and $I_a \to 0$ if $t \to \infty$. So we only consider the global stability of O_{ah} only at the region $E_{12} = \{(S_a, I_a, S_h, I_h) | S_a = 0, I_a = 0, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\}\}$. Now consider system (21) with the avian components already at the disease-free steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = -(\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{40}$$

Choose a Liapunov function as follows

$$V_{31} = S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*} + I_h,$$

According to the proof of Theorem 2.6(i), we have $E_{12} = \{(S_a, I_a, S_h, I_h) | S_a = K_a, I_a = 0, S_a + I_a + S_h + I_h \le \max\{\frac{\phi}{\nu} + 1, A_0\} : \frac{dV_{31}}{dt} = 0\} = \{(S_a, I_a, S_h, I_h) : S_a = 0, I_a = 0, S_h = S_h^*, I_h = 0\} = \{O_{ah}\}$, LaSalle's invariance principle (Hale [25]) implies that the equilibrium O_{ah} is globally asymptotically stable for positive trajectories in the region E_1.

(ii) If $\frac{M_a + m_a}{2} \le \frac{\mu_a + \delta_a}{\beta_a} < M_a$ and $(S_a, I_a, S_h, I_h) \in E_2$, then $(S_a, I_a) \in D_2$. According to Theorem 3.5, the disease-free equilibrium E of the subsystem (10) is always globally asymptotically stable in the region D_2 which shows that $S_a \to S_a^{**}$ and $I_h \to I_a^{**}$ if $t \to \infty$. Thus we only need to analyze the global stability of E_{ah} only at the region $E_{22} = \{(S_a, I_a, S_h, I_h) | S_a = S_a^{**}, I_a = I_a^{**}, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\}\}$. Once again consider system (21) with the avian components already at the disease-free steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \beta_a I_a^{**} S_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = \beta_a I_a^{**} S_h - (\mu_h + \delta_h + \gamma) I_h. \end{cases} \tag{41}$$

Choose the following Liapunov function

$$V_{32} = S_h^{**}\left(\frac{S_h}{S_h^{**}} - \ln \frac{S_h}{S_h^{**}}\right) + I_h^{**}\left(\frac{I_h}{I_h^{**}} - \ln \frac{I_h}{I_h^{**}}\right),$$

According to the proof of Theorem 2.6(ii), we have $E_{22} = \{(S_a, I_a, S_h, I_h) | S_a = S_a^{**}, I_a = I_a^{**}, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\} : \frac{dV_{32}}{dt} = 0\} = \{(S_a, I_a, S_h, I_h) : S_a = S_a^{**}, I_a = I_a^{**}, S_h = S_h^{**}, I_h = I_h^{**}\} = \{E_{ah}\}$, LaSalle's invariance principle then implies that the equilibrium E_{ah} is globally asymptotically stable for positive trajectories in the region E_2.

(iii) If $\frac{\mu_a + \delta_a}{\beta_a} \ge M_a$ and $(S_a, I_a, S_h, I_h) \in E_2$, then $(S_a, I_a) \in D_2$. By Theorem 3.5, the disease-free equilibrium B of the subsystem (10) is globally asymptotically stable in the region D_2 which illustrates that $S_a \to M_a$ and $I_h \to 0$ if $t \to \infty$. Similarly we only need to study the global stability of B_{ah} only at the region $E_{22} = \{(S_a, I_a, S_h, I_h) | S_a = M_a, I_a = 0, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\}\}$. To do so we consider system (21) with the avian components already at the disease-free steady state, given by

$$\begin{cases} \dfrac{dS_h}{dt} = \Pi_h - \mu_h S_h \\ \dfrac{dI_h}{dt} = -(\mu_h + \delta_h + \gamma) I_h. \end{cases}$$

Choose a Liapunov function as follows

$$V_{33} = S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*} + I_h,$$

Proceeding with the proof process of (i), we have $E_{22} = \{(S_a, I_a, S_h, I_h) | S_a = M_a, I_a = 0, S_a + I_a + S_h + I_h \le \max\{A_0, \frac{\rho}{\omega} + 1\} :$

$\frac{dV_{33}}{dt} = 0\} = \{(S_a, I_a, S_h, I_h) : S_a = M_a, I_a = 0, S_h = S_h^*, I_h = 0\} = \{B_{ah}\}$, by LaSalle's invariance principle we claim that the equilibrium B_{ah} is globally asymptotically stable for positive trajectories in the region E_2. □

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