Hindawi
Complexity
Volume 2019, Article ID 4161287, 15 pages
https://doi.org/10.1155/2019/4161287

Research Article

Dynamics Analysis of an Avian Influenza A (H7N9) Epidemic Model with Vaccination and Seasonality

Juping Zhang[1,2] **Yun Li**,[3] **Zhen Jin**[1,2] **and Huaiping Zhu**[4]

[1] *Complex Systems Research Center, Shanxi University, Taiyuan, Shanxi 030006, China*
[2] *Shanxi Key Laboratory of Mathematical Techniques and Big Data Analysis on Disease Control and Prevention, Shanxi University, Taiyuan, Shanxi 030006, China*
[3] *School of Science, North University of China, Taiyuan, Shanxi 030051, China*
[4] *LAMPS and Department of Mathematics and Statistics, York University, Toronto, ON, Canada M3J 1P3*

Correspondence should be addressed to Juping Zhang; zhangjuping@sxu.edu.cn and Zhen Jin; jinzhn@263.net

Received 12 December 2018; Accepted 12 February 2019; Published 17 March 2019

Academic Editor: Xiaopeng Zhao

H7N9 virus in the environment plays a role in the dynamics of avian influenza A (H7N9). A nationwide poultry vaccination with H7N9 vaccine program was implemented in China in October of 2017. To analyze the effect of vaccination and environmental virus on the development of avian influenza A (H7N9), we establish an avian influenza A (H7N9) transmission model with vaccination and seasonality among human, birds, and poultry. The basic reproduction number for the prevalence of avian influenza is obtained. The global stability of the disease-free equilibrium and the existence of positive periodic solution are proved by the comparison theorem and the asymptotic autonomous system theorem. Finally, we use numerical simulations to demonstrate the theoretical results. Simulation results indicate that the risk of H7N9 infection is higher in colder environment. Vaccinating poultry can significantly reduce human infection.

1. Introduction

In general, the avian influenza virus does not infect human. However, in 1997, Hong Kong reported for the first time 18 cases of human infection with avian influenza A (H5N1), of which 6 cases died. It caused widespread concern worldwide [1–3]. Further, H5N1, H7N4, H7N7, H7N9, H9N2, and other avian influenza viruses with pathogenicity have great potential threat to human. Especially, the virus subtype H7N9 is mainly transmitted through the respiratory tract, infected poultry and their secretions, excreta, and water contaminated by the virus. In February 2013, 3 people were firstly infected, and by May 31, 132 cases were found, including 37 deaths, and the mortality rate even reached 30%. These cases are distributed in some provinces such as Shanghai, Jiangsu, Zhejiang, Anhui, Fujian, and so on [4–7]. At present, infected humans of avian influenza A (H7N9) are still sporadic, and it has not yet found the ability that the virus can spread among humans. Sporadic infections almost contact with poultry mainly in farms, live-poultry markets, wet markets, and other regions [8–12].

Zhang et al. [13] analyzed the source of infection of avian influenza A (H7N9). According to theirs analysis, the most probable transmission route of avian influenza A (H7N9) is that migratory birds carry the virus and transmit it to local birds through physical space transitions, which then transmit the virus to poultry and to human with direct or indirect contact with poultry. Vaidya and Wahl [14], considering the seasonal bird migration, studied the relationship between the time-varying environment-temperature and the decline of the avian influenza A (H7N9) virus in the environment. Xiao et al. [12] proposed and analyzed a mathematical model to mimic its transmission dynamics to assess the transmission potential of the novel avian influenza A (H7N9) virus. Xing et al. [15] fitted the dynamic model with the actual data and found that the main reason for the recurrence of avian influenza A (H7N9) in winter was temperature cycling. Che et al. [16] studied the model of highly pathogenic avian influenza with saturated contact rate. Li et al. [17] established an avian influenza A (H7N9) dynamics model with different groups in specific environment and studied the transmission of H7N9 avian influenza in the epidemic-prone areas, which reflected the impact of farms, live-poultry markets, and wet markets.

Note that birds and poultry are the natural storage hosts of avian influenza virus. The direct contact transmission between birds and poultry is very little and the cross infection between them happens through H7N9 virus carried in coexistence environment. Exposed and infected birds and poultry can shed virus into the environment due to secretions and excreta. The virus can survive for several weeks, or even months in the feces or contaminated environment under suitable conditions. So, environment transmission is indispensable during the process of the spread of influenza virus. The most easily infectious source of human infection is poultry with the virus, and the main routes of transmission are poultry-human and environment-human transmissions. In addition, the high-risk humans infected with avian influenza virus are mainly concentrated on humans who are often in contact with poultry, including slaughter, breeding, processing, trafficking in poultry, and low immunity groups. In order to reduce the production of sick poultry and the concentration of viruses in the environment, vaccinating poultry and disinfecting the environment are used.

The organization of this paper is as follows. In Section 2, we have constructed an avian influenza A (H7N9) epidemic model with vaccination and seasonality to study the spread of avian influenza A (H7N9). In Sections 3 and 4, the threshold value R_0 is obtained. By using the comparison theorem, and the asymptotic autonomous system theorem and so on, it is found that there exists a globally asymptotically stable disease-free equilibrium when $R_0 < 1$ and at least a positive periodic solution when $R_0 > 1$, respectively. The numerical simulations used to illustrate the theoretical results and some conclusions are included in Sections 5 and 6.

2. Formulation of the Model

We combine birds, poultry, and viruses in the environment with human to establish a mathematical model of the spread of avian influenza A (H7N9). Their total numbers of birds, poultry, and human at any time t are denoted by $N_b(t)$, $N_a(t)$, and $N_h(t)$, respectively. The concentration of viruses in the environment is denoted by $W(t)$, and the average number of viruses that causes a H7N9 individual case is called an infectious unit (IU) [13, 18]. The infectivity of virus in the environment to birds, poultry, or humans is more affected by the temperature, and we will consider it is periodic. The infectivities among birds, poultry, and human-poultry are related to themselves, which are less affected by the temperature, so periodicities are not considered. It is assumed that there is no transmission between humans and humans. Therefore, human individuals are infected by two ways: poultry-human and environment-human transmissions. The human population is classified into four subclasses: ordinary susceptible, high-risk susceptible, infected, and recovered, denoted by $S_{h1}(t), S_{h2}(t), I_h(t)$, and $R_h(t)$, respectively. $S_b(t)$ and $I_b(t)$ denote the number of susceptible and infective individuals in the bird population. $S_a(t), V_a(t)$, and $I_a(t)$ denote the number of susceptible, vaccinated, and infective individuals in the poultry population, respectively. Transmission process of avian influenza A (H7N9) virus among these populations is described in Figure 1.

The dynamic model of avian influenza A (H7N9) is described as the following ordinary differential equations:

$$\begin{aligned}
\frac{dS_b(t)}{dt} &= A_b - \beta_b S_b I_b - \beta_{wb}(t) S_b W - d_b S_b, \\
\frac{dI_b(t)}{dt} &= \beta_b S_b I_b + \beta_{wb}(t) S_b W - d_b I_b - \alpha_b I_b, \\
\frac{dS_a(t)}{dt} &= A_a - \beta_a S_a I_a - \beta_{wa}(t) S_a W - d_a S_a - p_a S_a \\
&\quad + \eta_a V_a, \\
\frac{dV_a(t)}{dt} &= p_a S_a - \sigma_1 \beta_a V_a I_a - \sigma_2 \beta_{wa}(t) V_a W - d_a V_a \\
&\quad - \eta_a V_a, \\
\frac{dI_a(t)}{dt} &= \beta_a S_a I_a + \beta_{wa}(t) S_a W + \sigma_1 \beta_a V_a I_a \\
&\quad + \sigma_2 \beta_{wa}(t) V_a W - d_a I_a - \alpha_a I_a, \\
\frac{dW(t)}{dt} &= q_b I_b + q_a I_a - d_w W - \delta_w W, \\
\frac{dS_{h1}(t)}{dt} &= A_h - \beta_{h1} S_{h1} I_a - \beta_{wh1}(t) S_{h1} W - d_h S_{h1} \\
&\quad - \xi_h S_{h1}, \\
\frac{dS_{h2}(t)}{dt} &= \xi_h S_{h1} - \beta_{h2} S_{h2} I_a - \beta_{wh2}(t) S_{h2} W - d_h S_{h2}, \\
\frac{dI_h(t)}{dt} &= \beta_{h1} S_{h1} I_a + \beta_{wh1}(t) S_{h1} W + \beta_{h2} S_{h2} I_a \\
&\quad + \beta_{wh2}(t) S_{h2} W - d_h I_h - \alpha_h I_h - \gamma_h I_h, \\
\frac{dR_h(t)}{dt} &= \gamma_h I_h - d_h R_h.
\end{aligned} \tag{1}$$

The interpretations of the variables and parameters are shown in Table 1. All variables and parameters are nonnegative.

According to [15, 19, 20], the relationships between environmental transmission rates and temperature can be obtained

$$\beta_{wb}(t) = f_{wb}(T) = \begin{cases} 0, & T > 30^\circ\mathrm{C}, \\ \beta_{wb0}(30 - T) e^{-g_1 T - g_2}, & T \le 30^\circ\mathrm{C}. \end{cases} \tag{2}$$

$$\beta_{wa}(t) = f_{wa}(T) = \begin{cases} 0, & T > 30^\circ\mathrm{C}, \\ \beta_{wa0}(30 - T) e^{-g_3 T - g_4}, & T \le 30^\circ\mathrm{C}. \end{cases} \tag{3}$$

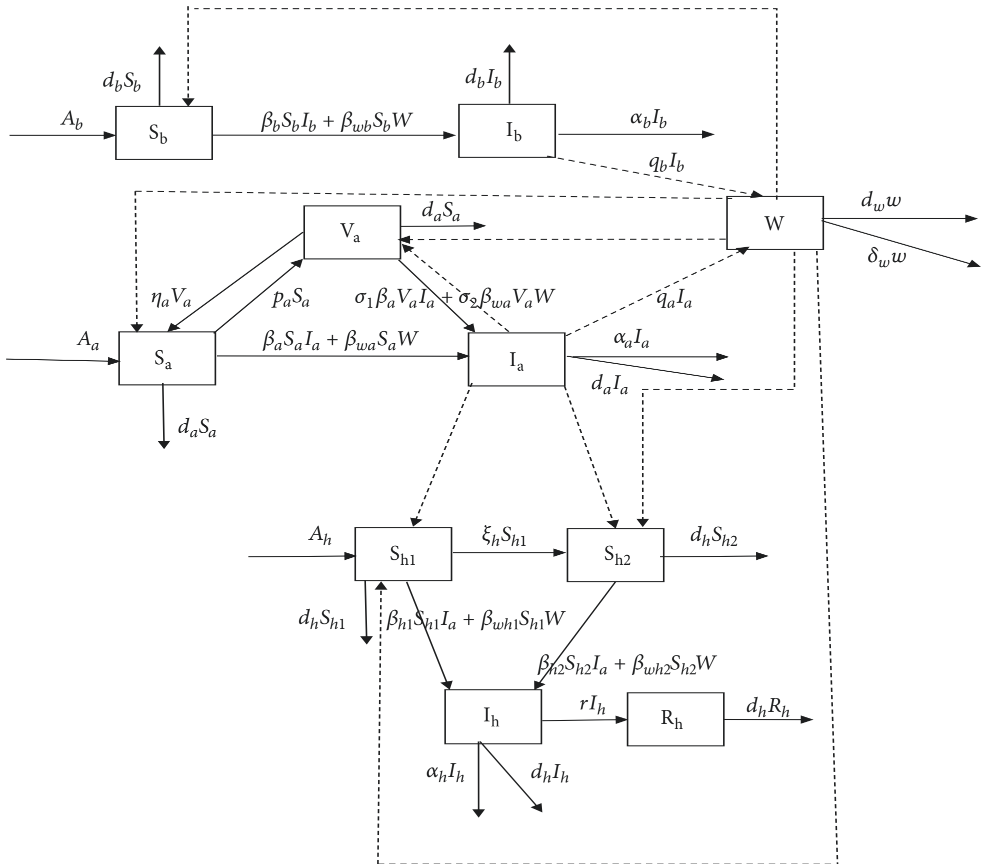

Figure 1: Flowchart of avian influenza A (H7N9) transmission.

$$\beta_{wh1}(t) = f_{wh1}(T) = \begin{cases} 0, & T > 30^\circ\text{C}, \\ \beta_{wh10}(30 - T)\, e^{-g_5 T - g_6}, & T \le 30^\circ\text{C}, \end{cases} \tag{4}$$

and

$$\beta_{wh2}(t) = f_{wh2}(T) = \begin{cases} 0, & T > 30^\circ\text{C}, \\ \beta_{wh20}(30 - T)\, e^{-g_7 T - g_8}, & T \le 30^\circ\text{C}, \end{cases} \tag{5}$$

where $T = T_0(1 + \phi_1 \sin(\omega t + \phi_2))$ is the temperature, T_0 is the average temperature, ω is a period, ϕ_1 is the amplitude, and ϕ_2 is the phase difference.

We model the implementation of intervention strategies (initiated at time $t^\star$) by the following piecewise functions β_{h1} and β_{h2} [12]:

$$\beta_{h1}(t) = \begin{cases} \beta_{h10}, & t \le t^\star, \\ \beta_{h10} e^{-k_1(t - t^\star)}, & t > t^\star, \end{cases} \tag{6}$$

and

$$\beta_{h2}(t) = \begin{cases} \beta_{h20}, & t \le t^\star, \\ \beta_{h20} e^{-k_2(t - t^\star)}, & t > t^\star. \end{cases} \tag{7}$$

From system (1), these can find that

$$\frac{dN_b}{dt} = A_b - d_b N_b - \alpha_b I_b \le A_b - d_b N_b. \tag{8}$$

$$\frac{dN_a}{dt} = A_a - d_a N_a - \alpha_a I_a \le A_a - d_a N_a. \tag{9}$$

$$\frac{dN_h}{dt} = A_h - d_a N_h - \alpha_h I_h \le A_h - d_h N_h. \tag{10}$$

Then, from (8), it follows that

$$N_b(t) \le \frac{A_b}{d_b} + \left(N_b(0) - \frac{A_b}{d_b} \right) e^{-d_b t}, \tag{11}$$

and $e^{-d_b t} \longrightarrow 0$ as $t \longrightarrow +\infty$, so $\lim_{t \longrightarrow +\infty} N_b(t) \le A_b/d_b$.

In the same way, from (9) and (10), it can be obtained that $\lim_{t \longrightarrow +\infty} N_a(t) \le A_a/d_a$, $\lim_{t \longrightarrow +\infty} N_h(t) \le A_h/d_h$. The feasible region of system (1) is

$$\Omega = \left\{ (S_b, I_b, S_a, V_a, I_a, W, S_{h1}, S_{h2}, I_h, R_h) \in R_+^{10} : N_b \le \frac{A_b}{d_b},\ N_a \le \frac{A_a}{d_a},\ N_h \le \frac{A_h}{d_h},\ W \le \frac{q_b A_b/d_b + q_a A_a/d_a}{d_w + \delta_w} \right\}. \tag{12}$$

Table 1: Description of parameters and variables.

Parameters	Interpretation	Value(week)	Resource
A_b	The birth number of birds	$1 * 10^9/520$	[15]
d_b	The natural mortality rate of birds	1/10/52	[13]
α_b	The mortality rate of birds caused by H7N9	0.08	Assuming
β_b	$I_b(t)$-to-$S_b(t)$transmission rate	Parameter	
$\beta_{wb}(t)$	$W(t)$-to-$S_b(t)$transmission rate	Parameter	
β_{wb0}		Parameter	
g_1		0.0313	[15]
g_2		3.0624	[15]
A_a	The birth number of poultry	$1.0438 * 10^9/8$	[13]
d_a	The natural mortality rate of poultry	1/8	[13]
p_a	The vaccination rate of poultry	$[0, 1]$	Assuming
η_a	The vaccine failure rate of poultry	$[0, 1]$	Assuming
σ_1	Probability of $I_a(t)$-to-$V_a(t)$	0.12	Assuming
σ_2	Probability of $W(t)$-to-$V_a(t)$	0.08	Assuming
α_a	The mortality rate of poultry caused by H7N9	Parameter	
β_a	$I_a(t)$-to-$S_a(t)$transmission rate	Parameter	
$\beta_{wa}(t)$	$W(t)$-to-$S_a(t)$transmission rate	Parameter	
β_{wa0}		Parameter	
g_3		0.0313	[15]
g_4		3.0624	[15]
q_b	The discharging concentration of H7N9 virus by $I_b(t)$	5	[15]
q_a	The discharging concentration of H7N9 virus by $I_a(t)$	5	[15]
d_w	The natural mortality rate of virus	0.7	Assuming
δ_w	The effective disinfection rate	Parameter	
A_h	The birth number of human	$2.269741 * 10^8/70/52$	[13]
d_h	The natural death rate of human	1/70/52	[13]
ξ_h	The proportion of $S_{h1}(t)$-to-$S_{h2}(t)$	0.0071	Assuming
γ_h	The recovery rate of human	2	[15]
α_h	The mortality rate of human caused by H7N9	0.36	[13]
β_{h1}	$I_a(t)$-to-$S_{h1}(t)$ transmission rate	Parameter	
$\beta_{wh1}(t)$	$W(t)$-to-$S_{h1}(t)$ transmission rate	Parameter	
β_{h10}	The baseline transmission rate from poultry to ordinary humans	Parameter	
β_{wh10}		Parameter	
g_5		0.0313	[15]
g_6		3.0624	[15]
k_1	Intervention intensity to $S_{h1}(t)$	Parameter	
t^*	The initiated time for implementation of measures	Parameter	
β_{h2}	$I_a(t)$-to-$S_{h2}(t)$ transmission rate	Parameter	
$\beta_{wh2}(t)$	$W(t)$-to-$S_{h2}(t)$ transmission rate	Parameter	
β_{h20}	The baseline transmission rate from poultry to high-risk humans	Parameter	
β_{wh20}		Parameter	
g_7		0.0313	[15]
g_8		3.0624	[15]
k_2	Intervention intensity to $S_{h2}(t)$	Parameter	

3. The Basic Reproduction Number

The last equation is independent of the first nine equations of system (1); we can only consider the following subsystem of system (1):

$$\begin{aligned}
\frac{dS_b(t)}{dt} &= A_b - \beta_b S_b I_b - \beta_{wb}(t) S_b W - d_b S_b, \\
\frac{dI_b(t)}{dt} &= \beta_b S_b I_b + \beta_{wb}(t) S_b W - d_b I_b - \alpha_b I_b, \\
\frac{dS_a(t)}{dt} &= A_a - \beta_a S_a I_a - \beta_{wa}(t) S_a W - d_a S_a - p_a S_a \\
&\quad + \eta_a V_a, \\
\frac{dV_a(t)}{dt} &= p_a S_a - \sigma_1 \beta_a V_a I_a - \sigma_2 \beta_{wa}(t) V_a W - d_a V_a \\
&\quad - \eta_a V_a, \\
\frac{dI_a(t)}{dt} &= \beta_a S_a I_a + \beta_{wa}(t) S_a W + \sigma_1 \beta_a V_a I_a \\
&\quad + \sigma_2 \beta_{wa}(t) V_a W - d_a I_a - \alpha_a I_a, \\
\frac{dW(t)}{dt} &= q_b I_b + q_a I_a - d_w W - \delta_w W, \\
\frac{dS_{h1}(t)}{dt} &= A_h - \beta_{h1} S_{h1} I_a - \beta_{wh1}(t) S_{h1} W - d_h S_{h1} \\
&\quad - \xi_h S_{h1}, \\
\frac{dS_{h2}(t)}{dt} &= \xi_h S_{h1} - \beta_{h2} S_{h2} I_a - \beta_{wh2}(t) S_{h2} W - d_h S_{h2}, \\
\frac{dI_h(t)}{dt} &= \beta_{h1} S_{h1} I_a + \beta_{wh1}(t) S_{h1} W + \beta_{h2} S_{h2} I_a \\
&\quad + \beta_{wh2}(t) S_{h2} W - d_h I_h - \alpha_h I_h - \gamma_h I_h.
\end{aligned} \tag{13}$$

It is easy to see that system (13) always has the disease-free equilibrium $E^0 = (S_b^0, 0, S_a^0, V_a^0, 0, 0, S_{h1}^0, S_{h2}^0, 0)$, where

$$\begin{aligned}
S_b^0 &= \frac{A_b}{d_b}, \\
S_a^0 &= \frac{A_a(d_a + \eta_a)}{d_a(d_a + p_a + \eta_a)}, \\
V_a^0 &= \frac{p_a S_a^0}{d_a + \eta_a}, \\
S_{h1}^0 &= \frac{A_h}{d_h + \xi_h}, \\
S_{h2}^0 &= \frac{\xi_h S_{h1}^0}{d_h}.
\end{aligned} \tag{14}$$

From system (13), we know that human dynamic equations do not affect the basic reproduction number. But for the convenience of dynamic analysis, we add human dynamic equations when calculating the basic reproduction number. According to the method of Wang and Zhao [21], we can obtain

$$\mathscr{F}(t) = \begin{pmatrix}
\beta_b S_b I_b + \beta_{wb}(t) S_b W \\
\beta_a S_a I_a + \beta_{wa}(t) S_a W + \sigma_1 \beta_a V_a I_a + \sigma_2 \beta_{wa}(t) V_a W \\
0 \\
\beta_{h1} S_{h1} I_a + \beta_{wh1}(t) S_{h1} W + \beta_{h2} S_{h2} I_a + \beta_{wh2}(t) S_{h2} W \\
0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}, \tag{15}$$

and

$$\mathscr{V}(t) = \begin{pmatrix}
\alpha_b I_b + d_b I_b \\
\alpha_a I_a + d_a I_a \\
\delta_w W + d_w W - q_b I_b + q_a I_a \\
\alpha_h I_h + d_h I_h + \gamma_h I_h \\
\beta_b S_b I_b + \beta_{wb}(t) S_b W + d_b S_b - A_b \\
\beta_a S_a I_a + \beta_{wa}(t) S_a W + d_a S_a + p_a S_a - \eta_a V_a - A_a \\
\sigma_1 \beta_a V_a I_a + \sigma_2 \beta_{wa}(t) V_a W + d_a V_a + \eta_a V_a - p_a S_a \\
\beta_{h1} S_{h1} I_a + \beta_{wh1}(t) S_{h1} W + d_h S_{h1} + \xi_h S_{h1} - A_h \\
\beta_{h2} S_{h2} I_a + \beta_{wh2}(t) S_{h2} W + d_h S_{h2} - \xi_h S_{h1}
\end{pmatrix}. \tag{16}$$

Then

$$F_{(E^0)}(t) = \begin{pmatrix}
\beta_b S_b^0 & 0 & \beta_{wb}(t) S_b^0 & 0 \\
0 & \beta_a S_a^0 + \sigma_1 \beta_a V_a^0 & \beta_{wa}(t) S_a^0 + \sigma_2 \beta_{wa}(t) V_a^0 & 0 \\
0 & 0 & 0 & 0 \\
0 & \beta_{h1} S_{h1}^0 + \beta_{h2} S_{h2}^0 & \beta_{wh1}(t) S_{h1}^0 + \beta_{wh2}(t) S_{h2}^0 & 0
\end{pmatrix}, \tag{17}$$

and

$$V_{(E^0)} = \begin{pmatrix}
\alpha_b + d_b & 0 & 0 & 0 \\
0 & d_a + \alpha_a & 0 & 0 \\
-q_b & -q_a & \delta_w + d_w & 0 \\
0 & 0 & 0 & d_h + \alpha_h + \gamma_h
\end{pmatrix}. \tag{18}$$

Assume $Y(t,s), t \geq s$ is the evolution operator of the linear ω-periodic system

$$\frac{dy}{dt} = -V_{(E^0)} y. \tag{19}$$

That is, for each $s \in R$, the 4×4 matrix $Y(t,s)$ satisfies

$$\frac{dY(t,s)}{dt} = -V_{(E^0)} Y(t,s), \quad \forall t \geq s, \ Y(s,s) = I, \tag{20}$$

where I is the 4×4 identity matrix. Thus, the monodromy matrix $\Phi_{-V_{(E^0)}}$ of (19) is equal to $Y(t,0), t \geq 0$.

In view of the periodic environment, we assume that $\varphi(s)$, ω-periodic in s, is the initial distribution of infectious individuals. Then $F_{(E^0)}(s)\varphi(s)$ is the rate of new infections produced by the infected individuals who were introduced at time s. Given $t \geq s$, then $Y(t,s)F_{(E^0)}(s)\varphi(s)$ gives the distribution of those infected individuals who were newly infected at time s and remain in the infected compartments at time t.

Let $\mathscr{C}_\omega$ be the ordered Banach space of all ω-periodic functions from R to R^4, which is equipped with the maximum norm $\| \bullet \|$ and the positive cone $\mathscr{C}_\omega^+ = \{\varphi \in \mathscr{C}_\omega : \varphi(t) \geq 0, \forall t \in R\}$. Then we can define a linear operator $L: \mathscr{C}_\omega \longrightarrow \mathscr{C}_\omega$ by $(L\varphi)(t) = \int_0^{+\infty} Y(t, t-\zeta)F_{(E^0)}(t-\zeta)\varphi(t-\zeta)d\zeta$. From Wang and Zhao [21], we call L the next infection operator and define the basic reproduction number as $R_0 = \rho(L)$, where ρ is the spectral radius of L.

Theorem 1. *For system (13), if $R_0 < 1$, the disease-free equilibrium E^0 is locally asymptotically stable; if $R_0 > 1$, it is unstable.*

4. The Stability of the Disease-Free Equilibrium and Existence of Periodic Solutions

4.1. The Stability of the Disease-Free Equilibrium

Theorem 2. *For system (13), if $R_0 < 1$, the disease-free equilibrium E^0 is globally asymptotically stable.*

Proof. Let $(S_b, I_b, S_a, V_a, I_a, W, S_{h1}, S_{h2}, I_h)$ be a nonnegative solution of system (13). To complete the proof, it is sufficient to show that this nonnegative solution tends to the disease-free equilibrium E^0 as $t \longrightarrow +\infty$.

The first, third, fourth, seventh, and eighth equations of system (13) with $V_a \leq N_a - S_a$ give the respective inequalities

$$\begin{aligned} \frac{dS_b(t)}{dt} &\leq A_b - d_b S_b, \\ \frac{dS_a(t)}{dt} &\leq A_a + \eta_a N_a - d_a S_a - p_a S_a - \eta_a S_a, \\ \frac{dV_a(t)}{dt} &\leq p_a N_a - p_a V_a - d_a V_a - \eta_a V_a, \\ \frac{dS_{h1}(t)}{dt} &\leq A_h - d_h S_{h1} - \xi_h S_{h1}, \\ \frac{dS_{h2}(t)}{dt} &\leq \xi_h S_{h1} - d_h S_{h2}. \end{aligned} \tag{21}$$

Hence, for any $\varepsilon > 0$, it exists $t_\varepsilon > 0$; when $t \geq t_\varepsilon$, we have

$$\begin{aligned} S_b &\leq S_b^0 + \varepsilon, \\ S_a &\leq S_a^0 + \varepsilon, \\ V_a &\leq V_a^0 + \varepsilon, \\ S_{h1} &\leq S_{h1}^0 + \varepsilon, \\ S_{h2} &\leq S_{h2}^0 + \varepsilon. \end{aligned} \tag{22}$$

The second, fifth, sixth, and ninth equations of system (13), with $S_b \leq S_b^0 + \varepsilon$, $S_a \leq S_a^0 + \varepsilon$, $V_a \leq V_a^0 + \varepsilon$, $S_{h1} \leq S_{h1}^0 + \varepsilon$, and $S_{h2} \leq S_{h2}^0 + \varepsilon$, give the respective inequalities

$$\begin{aligned} \frac{dI_b(t)}{dt} &\leq \beta_b \left(S_b^0 + \varepsilon\right) I_b + \beta_{wb}(t) \left(S_b^0 + \varepsilon\right) W - d_b I_b \\ &\quad - \alpha_b I_b, \\ \frac{dI_a(t)}{dt} &\leq \left(\beta_a I_a + \beta_{wa}(t) W\right) \left(S_a^0 + \varepsilon\right) \\ &\quad + \left(\sigma_1 \beta_a I_a + \sigma_2 \beta_{wa}(t) W\right) \left(V_a^0 + \varepsilon\right) \\ &\quad - d_a I_a - \alpha_a I_a, \\ \frac{dW(t)}{dt} &= q_b I_b + q_a I_a - d_w W - \delta_w W, \\ \frac{dI_h(t)}{dt} &\leq \left(\beta_{h1} I_a + \beta_{wh1}(t) W\right) \left(S_{h1}^0 + \varepsilon\right) \\ &\quad + \left(\beta_{h2} I_a + \beta_{wh2}(t) W\right) \left(S_{h2}^0 + \varepsilon\right) - d_h I_h \\ &\quad - \alpha_h I_h - \gamma_h I_h. \end{aligned} \tag{23}$$

We consider the auxiliary system of system (23), where the coefficient matrix is

$$M_\varepsilon = \begin{pmatrix} \beta_b \varepsilon & 0 & \beta_{wb}(t)\varepsilon & 0 \\ 0 & \beta_a \varepsilon + \sigma_1 \beta_a \varepsilon & \beta_{wa}(t)\varepsilon + \sigma_2 \beta_{wa}(t)\varepsilon & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta_{h1}\varepsilon + \beta_{h2}\varepsilon & \beta_{wh1}(t)\varepsilon + \beta_{wh2}(t)\varepsilon & 0 \end{pmatrix}. \tag{24}$$

Clearly, if $R_0 < 1$, it is known from Theorem 2.2 in [21] that $\rho(\Phi_{F-V}(\omega)) < 1$. We can choose $\varepsilon > 0$ small enough giving $\rho(\Phi_{F-V+M_\varepsilon}(\omega)) < 1$. It can be concluded from Lemma 2.1 in Zhang and Zhao [22] that there exists a positive, ω-periodic function $\overline{f}(t) = (\overline{I_b}(t), \overline{I_a}(t), \overline{W}(t), \overline{I_h}(t))$ such that $\widehat{f}(t) = e^{\Theta t}\overline{f}(t)$ is a solution of the auxiliary system, where $\Theta = (1/\omega)\ln(\rho(\Phi_{F-V+M_\varepsilon}(\omega)))$. Here, $\rho(\Phi_{F-V+M_\varepsilon}(\omega) < 1 \Longrightarrow \Theta < 0$, which implies $\widehat{f}(t) \longrightarrow 0$ as $t \longrightarrow +\infty$. Therefore, the zero solution of the auxiliary system is globally asymptotically stable. For any nonnegative initial value, there is a sufficiently large M. Applying the comparison theorem [23], it can be obtained that $f(t) \leq M\widehat{f}(t), \forall t > 0$. Therefore, we obtain $I_b(t) \longrightarrow 0, I_a(t) \longrightarrow 0, W(t) \longrightarrow 0, I_h(t) \longrightarrow 0$ as $t \longrightarrow +\infty$. By the theory of asymptotic autonomous systems [24], we get $S_b(t) \longrightarrow S_b^0, I_b(t) \longrightarrow 0, S_a(t) \longrightarrow S_a^0, V_a(t) \longrightarrow V_a^0, I_a(t) \longrightarrow 0, W(t) \longrightarrow 0, S_{h1}(t) \longrightarrow S_{h1}^0, S_{h2}(t) \longrightarrow S_{h2}^0, I_h(t) \longrightarrow 0$ as $t \longrightarrow +\infty$. Hence, if $R_0 < 1$, the disease-free equilibrium E^0 is globally asymptotically stable. □

4.2. The Existence of Positive Periodic Solutions. Define

$$\begin{aligned} X &:= \Omega, \\ X_0 &:= \{(S_b, I_b, S_a, V_a, I_a, W, S_{h1}, S_{h2}, I_h) \in X : I_b(t) \\ &\quad > 0,\ I_a(t) > 0,\ W(t) > 0,\ I_h(t) > 0\}, \\ \partial X_0 &:= X \backslash X_0. \end{aligned} \tag{25}$$

Let $P : X \longrightarrow X$ be the Poincaré map associated with system (13); that is, $P(z_0) = u(\omega, z_0), \forall z_0 \in X$, where ω is the period. $u(t, z_0)$ is the unique solution of system (13) with $u(0, z_0) = z_0 = (S_b(0), I_b(0), S_a(0), V_a(0), I_a(0), W(0), S_{h1}(0), S_{h2}(0), I_h(0))$. It is easy to see that $P^n(z_0) = u(n\omega, z_0), \forall n \geq 0$.

Lemma 3. *For system (13), if $R_0 > 1$, then there exists a $\mu > 0$ such that, for any $z_0 = (S_b(0), I_b(0), S_a(0), V_a(0), I_a(0), W(0), S_{h1}(0), S_{h2}(0), I_h(0)) \in X_0$ with $\|z_0 - E^0\| \leq \mu$, we have $\lim_{n\longrightarrow+\infty} \sup d[P^n(z_0), E^0] \geq \mu$.*

Proof. Since $R_0 > 1$, Theorem 1 implies that E^0 is unstable; i.e., $\rho(\Phi_{F-V}(\omega)) > 1$. We can choose $\varepsilon_1 > 0$ small enough such that $\rho(\Phi_{F-V-M_{\varepsilon_1}}(\omega)) > 1$, where

$$M_{\varepsilon_1} = \begin{pmatrix} \beta_b \varepsilon_1 & 0 & \beta_{wb}(t)\varepsilon_1 & 0 \\ 0 & \beta_a\varepsilon_1 + \sigma_1\beta_a\varepsilon_1 & \beta_{wa}(t)\varepsilon_1 + \sigma_2\beta_{wa}(t)\varepsilon_1 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta_{h1}\varepsilon_1 + \beta_{h2}\varepsilon_1 & \beta_{wh1}(t)\varepsilon_1 + \beta_{wh2}(t)\varepsilon_1 & 0 \end{pmatrix}. \tag{26}$$

To the contrary, if possible suppose that the limit $\lim_{n\longrightarrow+\infty} \sup d[P^n(z_0), E^0] < \mu$ for some $z_0 \in X_0$. Without loss of generality, we assume that $d[P^n(z_0), E^0] < \mu, \forall n \geq 0$. By the continuity of the solution with respect to the initial value, it follows that

$$\left\| u(t, P^n(z_0)) - u(t, E^0) \right\| < \varepsilon_1, \quad \forall n \geq 0,\ \forall t \in [0, \omega]. \tag{27}$$

From the periodicity of the system, for $\varepsilon_1 > 0$, there exists t_{ε_1} such that, for all $t > t_{\varepsilon_1}$, there holds

$$\begin{aligned} S_b &\geq S_b^0 - \varepsilon_1, \\ S_a &\geq S_a^0 - \varepsilon_1, \\ V_a &\geq V_a^0 - \varepsilon_1, \\ S_{h1} &\geq S_{h1}^0 - \varepsilon_1, \\ S_{h2} &\geq S_{h2}^0 - \varepsilon_1. \end{aligned} \tag{28}$$

Then

$$\begin{aligned} \frac{dI_b(t)}{dt} &\geq \beta_b (S_b^0 - \varepsilon_1) I_b + \beta_{wb}(t) (S_b^0 - \varepsilon_1) W \\ &\quad - d_b I_b - \alpha_b I_b, \\ \frac{dI_a(t)}{dt} &\geq (\beta_a I_a + \beta_{wa}(t) W) (S_a^0 - \varepsilon_1) \\ &\quad + (\sigma_1 \beta_a I_a + \sigma_2 \beta_{wa}(t)) W) (V_a^0 - \varepsilon_1) \\ &\quad - d_a I_a - \alpha_a I_a, \\ \frac{dW(t)}{dt} &= q_b I_b + q_a I_a - d_w W - \delta_w W, \\ \frac{dI_h(t)}{dt} &\geq (\beta_{h1} I_a + \beta_{wh1}(t) W) (S_{h1}^0 - \varepsilon_1) \\ &\quad + (\beta_{h2} I_a + \beta_{wh2}(t) W) (S_{h2}^0 - \varepsilon_1) \\ &\quad - d_h I_h - \alpha_h I_h - \gamma_h I_h. \end{aligned} \tag{29}$$

Now, consider the auxiliary system of (29); it can be concluded from Lemma 2.1 in Zhang and Zhao [22] that there exists a positive, ω-periodic function $\overline{f}(t) = (\overline{I_b}(t), \overline{I_a}(t), \overline{W}(t), \overline{I_h}(t))$ such that $\widehat{f}(t) = e^{\Theta_1 t}\overline{f}(t)$ is a solution of the auxiliary system, where $\Theta_1 = (1/\omega)\ln(\rho(\Phi_{F-V-M_{\varepsilon_1}}(\omega)))$. Here, $\rho(\Phi_{F-V-M_{\varepsilon_1}}(\omega)) > 1 \Longrightarrow \Theta_1 > 0$, which implies that, for nonnegative integer n, $\widehat{f}(n\omega) \longrightarrow +\infty$ as $n \longrightarrow +\infty$. For any nonnegative initial value, there is a sufficiently small $m > 0$. Applying the comparison theorem [23], it can be obtained that $f(t) \geq m\widehat{f}(t), \forall t > 0$. Thus, we obtain $I_b(t) \longrightarrow +\infty, I_a(t) \longrightarrow +\infty, W(t) \longrightarrow +\infty, I_h(t) \longrightarrow +\infty$ as $t \longrightarrow +\infty$, which is a contradiction. This completes the proof. □

Lemma 4. *The following equation is established:*

$$\begin{aligned} M_\partial &:= \{z_0 \in \partial X_0 : P^n(z_0) \in \partial X_0,\ \forall n \geq 0\} \\ &= \left\{ (S_b^0, 0, S_a^0, V_a^0, 0, 0, S_{h1}^0, S_{h2}^0, 0) \in X : S_b^0 \geq 0,\ S_a^0 \right. \\ &\quad \left. \geq 0,\ V_a^0 \geq 0,\ S_{h1}^0 \geq 0,\ S_{h2}^0 \geq 0 \right\}. \end{aligned} \tag{30}$$

Proof. It is easy to know that

$$\begin{aligned} &\left\{ (S_b^0, 0, S_a^0, V_a^0, 0, 0, S_{h1}^0, S_{h2}^0, 0) \right\} \\ &\quad \subseteq \{z_0 \in \partial X_0 : P^n(z_0) \in \partial X_0,\ \forall n \geq 0\}. \end{aligned} \tag{31}$$

Proof by contradiction can be used to prove

$$\begin{aligned} &\left\{ (S_b^0, 0, S_a^0, V_a^0, 0, 0, S_{h1}^0, S_{h2}^0, 0) \right\} \\ &\quad \supseteq \{z_0 \in \partial X_0 : P^n(z_0) \in \partial X_0,\ \forall n \geq 0\}. \end{aligned} \tag{32}$$

If possible suppose that

$$\begin{aligned} z_0 &= (S_b(0), I_b(0), S_a(0), V_a(0), I_a(0), W(0), S_{h1}(0), \\ &\quad S_{h2}(0), I_h(0)) \in \{(z_0 \in \partial X_0 : P^n(z_0) \in \partial X_0,\ \forall n \\ &\quad \geq 0)\} \setminus \left\{ (S_b^0, 0, S_a^0, V_a^0, 0, 0, S_{h1}^0, S_{h2}^0, 0) \right\}. \end{aligned} \tag{33}$$

Without loss of generality, we assume that $I_b(n\omega) > 0$. By solving the general solution of system (13), we can derive

$I_b(t) > 0, I_a(t) > 0, W(t) > 0, I_h(t) > 0$. Meaning, $(S_b(t), I_b(t), S_a(t), V_a(t), I_a(t), W(t), S_{h1}(t), S_{h2}(t), I_h(t)) \notin \partial X_0$, which contradicts with $(S_b(0), I_b(0), S_a(0), V_a(0), I_a(0), W(0), S_{h1}(0), S_{h2}(0), I_h(0)) \in \partial X_0$. Therefore, the equation is established. □

Theorem 5. *If $R_0 > 1$, then there exists $\varepsilon^* > 0$ such that any solution $(S_b(t), I_b(t), S_a(t), V_a(t), I_a(t), W(t), S_{h1}(t), S_{h2}(t), I_h(t))$ of system (13) with initial value $z_0 = (S_b(0), I_b(0), S_a(0), V_a(0), I_a(0), W(0), S_{h1}(0), S_{h2}(0), I_h(0)) \in X_0$ satisfies*

$$\begin{aligned} &\lim_{t\longrightarrow+\infty} \inf I_b(t) \geq \varepsilon^*, \\ &\lim_{t\longrightarrow+\infty} \inf I_a(t) \geq \varepsilon^*, \\ &\lim_{t\longrightarrow+\infty} \inf W(t) \geq \varepsilon^*, \\ &\lim_{t\longrightarrow+\infty} \inf I_h(t) \geq \varepsilon^*, \end{aligned} \tag{34}$$

and system (13) admits at least one positive periodic solution.

Proof. It is now proved that $\{P^n\}_{n\geq 0}$ is uniformly persistent with respect to $(X_0, \partial X_0)$. For any $z_0 \in X_0$, from the first equation of system (13), it follows that

$$\begin{aligned} S_b(t) &= e^{-\int_0^t (\beta_b I_b(\bar{s}) + \beta_{wb}(\bar{s}) W(\bar{s}) + d_b) d\bar{s}} \Big[S_b(0) \\ &\quad + A_b \Big(\int_0^t e^{\int_0^{\bar{s}_1} (\beta_b I_b(\bar{s}) + \beta_{wb}(\bar{s}) W(\bar{s}) + d_b) d\bar{s}} d\widetilde{s_1} \Big) \Big]. \end{aligned} \tag{35}$$

Then, $S_b(t) > 0, \forall t > 0$. Similarly, $S_a(t) > 0, V_a(t) > 0, S_{h1}(t) > 0, S_{h2}(t) > 0, \forall t > 0$. As generalized to nonautonomous systems [25], the irreducibility of the cooperative matrix $\widetilde{M}(t)$ implies that $I_b(t) > 0, I_a(t) > 0, W(t) > 0, I_h(t) > 0, \forall t > 0$, where

$$\widetilde{M}(t) = \begin{pmatrix} \beta_b S_b - d_b - \alpha_b & 0 & \beta_{wb}(t) S_b & 0 \\ 0 & \beta_a S_a + \sigma_1 \beta_a V_a & \beta_{wa}(t) S_a + \sigma_2 \beta_{wa}(t) V_a & 0 \\ q_b & q_a & -d_w - \delta_w & 0 \\ 0 & \beta_{h1} S_{h1} + \beta_{h2} S_{h2} & \beta_{wh1}(t) S_{h1} + \beta_{wh2}(t) S_{h2} & -d_h - \alpha_h - \gamma_h \end{pmatrix}. \tag{36}$$

Thus, both X and X_0 are positively invariant. Clearly, ∂X_0 is relatively closed in X.

The disease-free equilibrium E^0 of system (13) is globally asymptotically stable. Lemmas 3 and 4 imply that E^0 is a unique fixed point of P in M_∂. Moreover, E^0 is an isolated invariant set in X, and $W^s(E^0) \bigcap X_0 = \phi$. Note that every orbit in M_∂ approaches E^0 and E^0 is acyclic in M_∂. By [26], it follows that $\{P^n\}_{n\geq 0}$ is uniformly persistent with respect to $(X_0, \partial X_0)$ and the solutions of system (13) are uniformly persistent with respect to $(X_0, \partial X_0)$; i.e., if $R_0 > 1$, there exists $\varepsilon^* > 0$ such that any solution $(S_b(t), I_b(t), S_a(t), V_a(t), I_a(t), W(t), S_{h1}(t), S_{h2}(t), I_h(t))$ of system (13) with initial value $z_0 = (S_b(0), I_b(0), S_a(0), V_a(0), I_a(0), W(0), S_{h1}(0), S_{h2}(0), I_h(0)) \in X_0$ satisfies

$$\begin{aligned} &\lim_{t\longrightarrow+\infty} \inf I_b(t) \geq \varepsilon^*, \\ &\lim_{t\longrightarrow+\infty} \inf I_a(t) \geq \varepsilon^*, \\ &\lim_{t\longrightarrow+\infty} \inf W(t) \geq \varepsilon^*, \\ &\lim_{t\longrightarrow+\infty} \inf I_h(t) \geq \varepsilon^*. \end{aligned} \tag{37}$$

Furthermore, P has a fixed point $(S_b^*(0), I_b^*(0), S_a^*(0), V_a^*(0), I_a^*(0), W^*(0), S_{h1}^*(0), S_{h2}^*(0), I_h^*(0)) \in X_0$. Further, there exists some $\bar{t} \in [0, \omega]$ such that $S_b^*(\bar{t}) > 0$. If it is not the case, $S_b^*(\bar{t}) \equiv 0$. Then, due to the periodicity of $S_b^*(t)$, we have $S_b^*(t) \equiv 0$, for all $t \geq 0$. From the first equation of system (13), we get $0 = A_b > 0$, which is a contradiction. Thus $\forall t \in [\bar{t}, \bar{t} + \omega]$, we obtain

$$\begin{aligned} S_b^*(t) &= e^{-\int_{\bar{t}}^t (\beta_b I_b^*(\bar{s}) + \beta_{wb}(\bar{s}) W^*(\bar{s}) + d_b) d\bar{s}} \Big[S_b^*(\bar{t}) \\ &\quad + A_b \Big(\int_{\bar{t}}^t e^{\int_{\bar{t}}^{\bar{s}_1} (\beta_b I_b^*(\bar{s}) + \beta_{wb}(\bar{s}) W^*(\bar{s}) + d_b) d\bar{s}} d\widetilde{s_1} \Big) \Big] > 0. \end{aligned} \tag{38}$$

The periodicity of $S_b^*(t)$ implies that $S_b^*(t) > 0, \forall t \geq 0$. Similarly, $I_b^*(t) > 0, S_a^*(t) > 0, V_a^*(t) > 0, I_a^*(t) > 0, W^*(t) > 0, S_{h1}^*(t) > 0, S_{h2}^*(t) > 0, I_h^*(t) > 0$. Therefore, $(S_b^*(t), I_b^*(t), S_a^*(t), V_a^*(t), I_a^*(t), W^*(t), S_{h1}^*(t), S_{h2}^*(t), I_h^*(t))$ is a positive ω-periodic solution of system (13). □

5. Numerical Simulations

In this section, based on the initial value of

$$\begin{aligned} z_0^1 &= (999999900, 100, 1043798853, 1043, 104, 100, 226960414, 13618, 11, 57), \\ z_0^2 &= (999999500, 500, 1043788518, 10438, 1044, 300, 226942176, 31776, 57, 91), \\ z_0^3 &= (999999000, 1000, 1043765555, 31314, 3131, 500, 226451720, 522040, 113, 227), \end{aligned} \tag{39}$$

the temperature of the Yangtze River Delta, i.e., $T = 20.8286[1 - 0.6273\sin((\pi/26)t + 19.9451)]$, and other parameter values from Table 1 [12, 13, 15, 19, 20] are taken as some examples to simulate the stability of the disease-free equilibrium and the existence of positive periodic solutions of system (1), and the time-series diagram is given. At last, when parameters take different values, the time-variation diagrams of I_h are given.

Example 1. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-11}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $p_a = 0.87, \eta_a = 0.018 * 7$, $\beta_a = 85/100 * 3.0904 * 10^{-10}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\alpha_a = 0.18, \delta_w = 0.8 * 7$, $\beta_{h10} = 85/100 * 9.0876 * 10^{-16}$, $\beta_{h20} = 60/100 * 1.5954 * 10^{-15}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $\beta_{wh20} = 60/100 * 1.5954 * 10^{-15}$, $k_1 = 0.35, k_2 = 0.48$, $t^* = 7$. Figure 2 shows the time-variation diagram of system (1) state variables. It is found that if $R_0 < 1$, the curve tends to be stable with time, and the disease-free equilibrium E^0 is globally asymptotically stable.

Example 2. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $p_a = 0.87, \eta_a = 0.018 * 7$, $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\alpha_a = 0.18$, $\delta_w = 0.018 * 7$, $\beta_{h10} = 85/100 * 9.0876 * 10^{-16}$, $\beta_{h20} = 60/100 * 1.5954 * 10^{-15}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $\beta_{wh20} = 60/100 * 1.5954 * 10^{-15}$, $k_1 = 0.35, k_2 = 0.48$, $t^* = 7$. As shown in Figure 3, it is found that if $R_0 > 1$, the state variables of system (1) change periodically with time.

Example 3. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $p_a = 0.87, \eta_a = 0.018 * 7$, $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\alpha_a = 0.18$, $\delta_w = 0.018 * 7$, $\beta_{h10} = 85/100 * 9.0876 * 10^{-16}$, $\beta_{h20} = 60/100 * 1.5954 * 10^{-15}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $\beta_{wh20} = 60/100 * 1.5954 * 10^{-15}$, $k_1 = 0.35, k_2 = 0.48$, $t^* = 7$ and let $T = 25^\circ\mathrm{C}, 15^\circ\mathrm{C}, 5^\circ\mathrm{C}$. Figure 4 shows the curve-trend diagram of I_h with time. It is found that the lower the temperature, the larger I_h.

Example 4. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $p_a = 0.87, \eta_a = 0.018 * 7$, $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\alpha_a = 0.18$, $\delta_w = 0.8 * 7$, $\beta_{h20} = 60/100 * 1.5954 * 10^{-15}$, $\beta_{wh20} = 60/100 * 1.5954 * 10^{-15}$, $k_2 = 0.48$, $t^* = 7$ and let $k_1 = 0.35$, $\beta_{h10} = 85/100 * 3.0904 * 10^{-14}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-15}$; $\beta_{h10} = 85/100 * 3.0904 * 10^{-15}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-15}$; $\beta_{h10} = 85/100 * 3.0904 * 10^{-14}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$ or $\beta_{h10} = 85/100 * 3.0904 * 10^{-16}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $k_1 = 0.007, 0.085, 0.35$. Figure 5 shows the curve-trend diagram of I_h with time. It is found that I_h will increase with the increase of β_{h1} or β_{wh1} over a period of time.

Example 5. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $p_a = 0.87, \eta_a = 0.018 * 7$, $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\alpha_a = 0.18$, $\delta_w = 0.8 * 7$, $\beta_{h10} = 85/100 * 3.0904 * 10^{-16}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $k_1 = 0.35$, $t^* = 7$ and let $k_2 = 0.48$, $\beta_{h20} = 85/100 * 3.0904 * 10^{-12}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-16}$; $\beta_{h20} = 85/100 * 3.0904 * 10^{-12}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-15}$; $\beta_{h20} = 85/100 * 3.0904 * 10^{-15}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-15}$ or $\beta_{h20} = 85/100 * 3.0904 * 10^{-15}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-15}$, $k_2 = 0.008, 0.09, 0.48$. Figure 6 shows the curve-trend diagram of I_h with time. It is found that I_h will increase with the increase of β_{h2} or β_{wh2} over a period of time.

Example 6. Take parameters $p_a = 0.87, \eta_a = 0.018 * 7$, $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\alpha_a = 0.18, \delta_w = 0.8 * 7$, $\beta_{h10} = 85/100 * 3.0904 * 10^{-16}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $k_1 = 0.15, k_2 = 0.28$, $t^* = 7$, $\beta_{h20} = 85/100 * 3.0904 * 10^{-15}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-15}$ and let $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$; $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-15}$; $\beta_b = 85/100 * 1.09546 * 10^{-8}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$. Figure 7 shows the curve-trend diagram of I_h with time. It is found that I_h will increase with the increase of β_b or β_{wb} over a period of time.

Example 7. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $p_a = 0.87, \eta_a = 0.018 * 7$, $\alpha_a = 0.18, \delta_w = 0.8 * 7$, $\beta_{h10} = 85/100 * 3.0904 * 10^{-16}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $k_1 = 0.35, k_2 = 0.48$, $t^* = 7$, $\beta_{h20} = 85/100 * 3.0904 * 10^{-15}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-15}$ and let $\beta_a = 85/100 * 3.0904 * 10^{-5}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$; $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$; $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-9}$. Figure 8 shows the curve-trend diagram of I_h with time. It is found that I_h will increase with the increase of β_a or β_{wa}.

Example 8. Take parameters $\beta_b = 85/100 * 1.09546 * 10^{-10}$, $\beta_{wb0} = 85/100 * 1.09546 * 10^{-10}$, $\beta_a = 85/100 * 3.0904 * 10^{-9}$, $\beta_{wa0} = 85/100 * 1.3579 * 10^{-10}$, $\beta_{h10} = 85/100 * 3.0904 * 10^{-16}$, $\beta_{wh10} = 85/100 * 9.0876 * 10^{-16}$, $k_1 = 0.35, k_2 = 0.48$, $t^* = 7$, $\beta_{h20} = 85/100 * 3.0904 * 10^{-15}$, $\beta_{wh20} = 85/100 * 9.0876 * 10^{-15}$ and let p_a, η_a, α_a, and δ_w change each other. Figure 9 shows the curve-trend diagram of I_h with time. It is found that I_h will increase with the increase of η_a or the decrease of p_a, α_a, δ_w.

6. Discussion

Avian influenza is the potential threat to human health. The exposure of infected poultry is a key factor for human infection with avian influenza A (H7N9) virus. Avian influenza A (H7N9) virus mainly spreads from poultry to human through infected poultry and its secretions, excreta, and virus-contaminated water. Most humans contact with poultry mainly in farms, live-poultry markets, wet markets, and other areas. In this paper, a $SI - SVI - W - SSIR$ dynamic model of avian influenza A (H7N9) is established by combining birds,

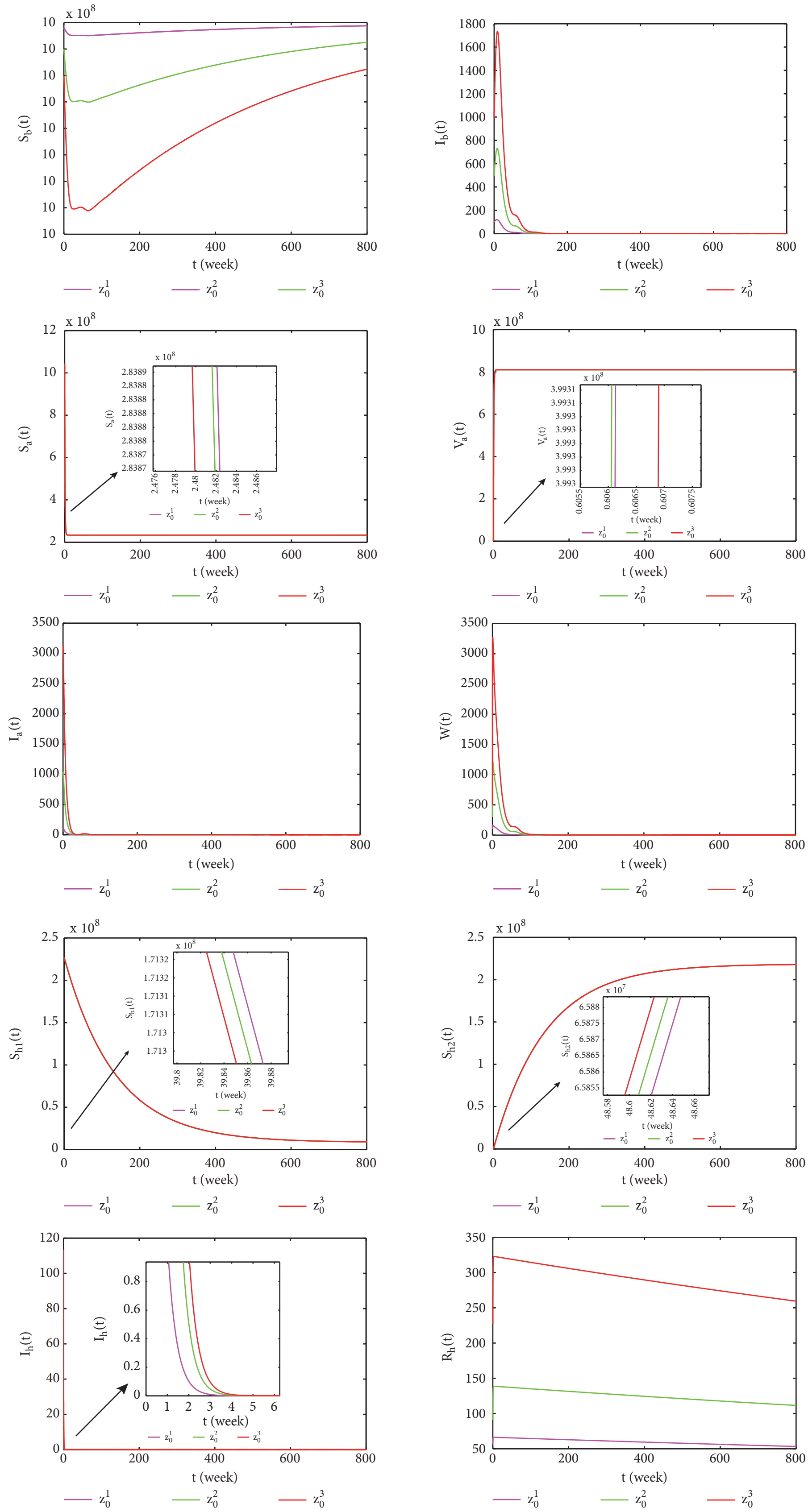

FIGURE 2: If $R_0 = 0.8575 < 1$, the time-variation diagram of system (1) state variables.

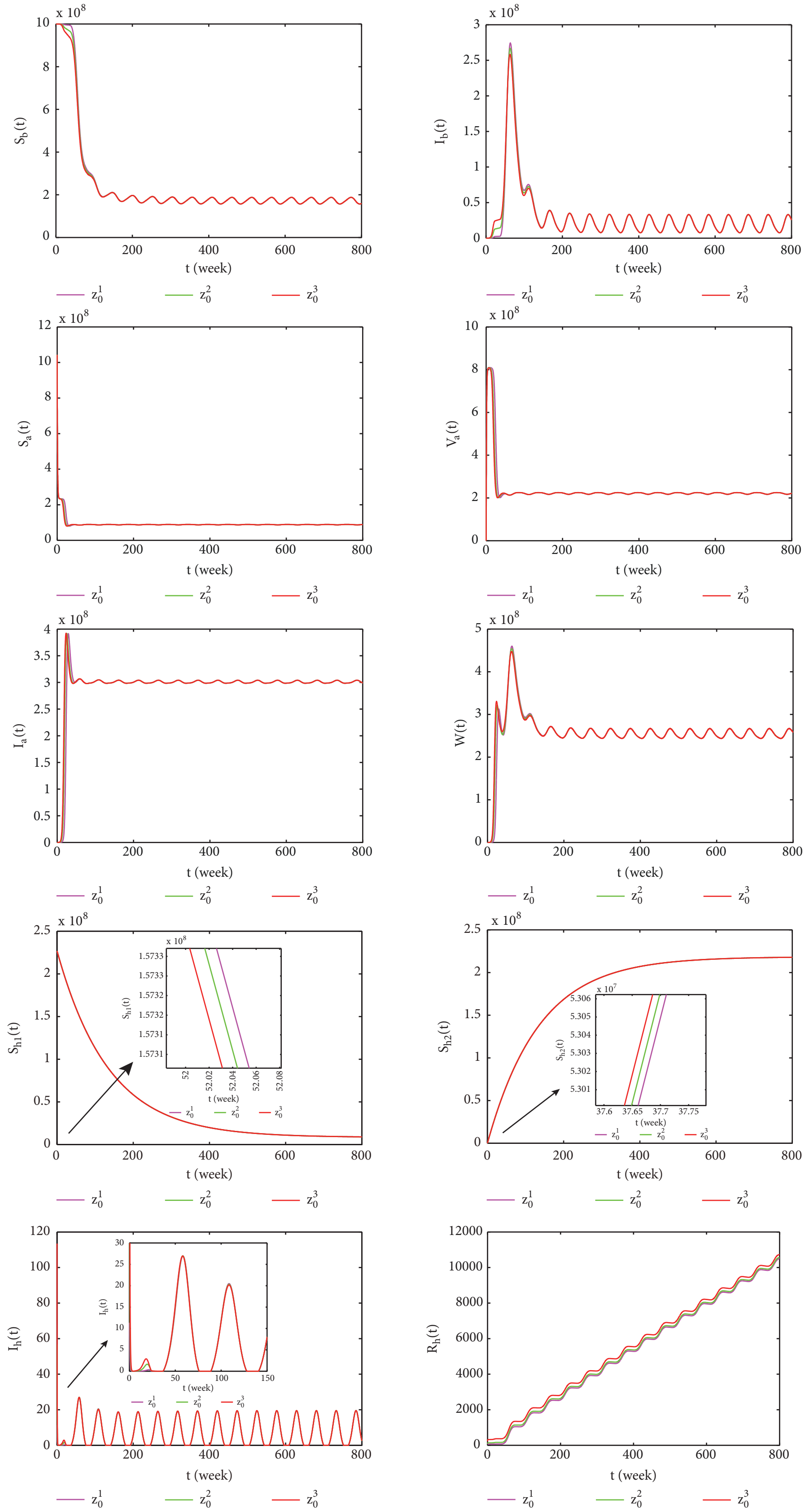

FIGURE 3: If $R_0 = 2.92 > 1$, the time-variation diagram of system (1) state variables.

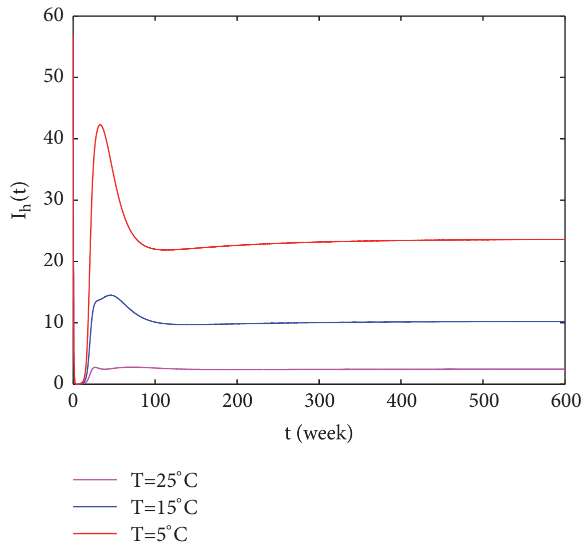

Figure 4: The curve-trend diagram of I_h with time, when T takes different values.

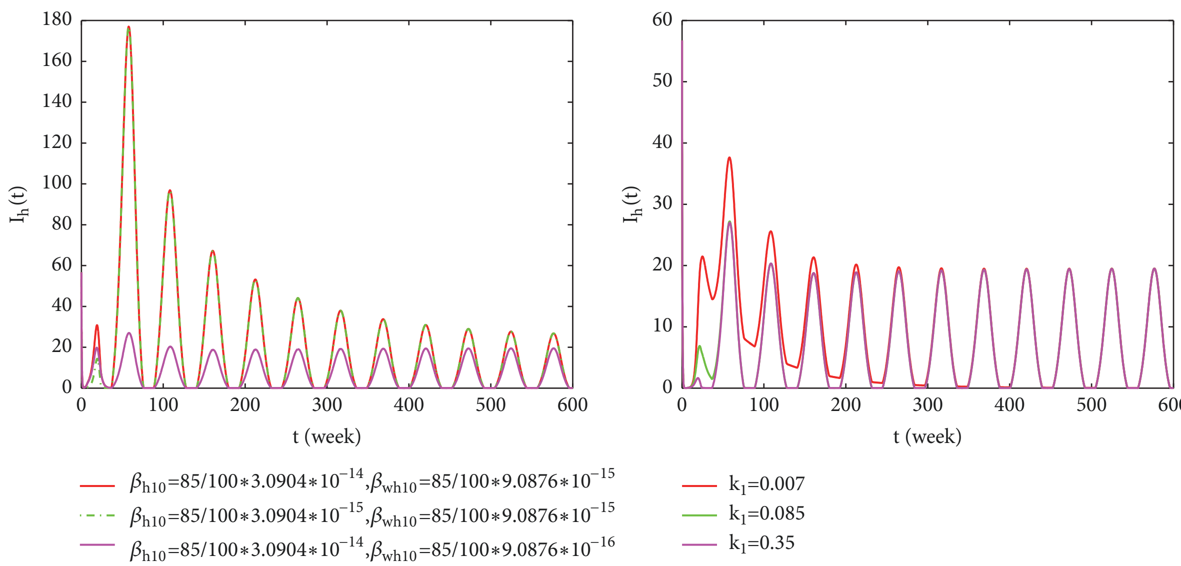

Figure 5: The curve-trend diagram of I_h with time, when β_{h1} or β_{wh1} takes different values.

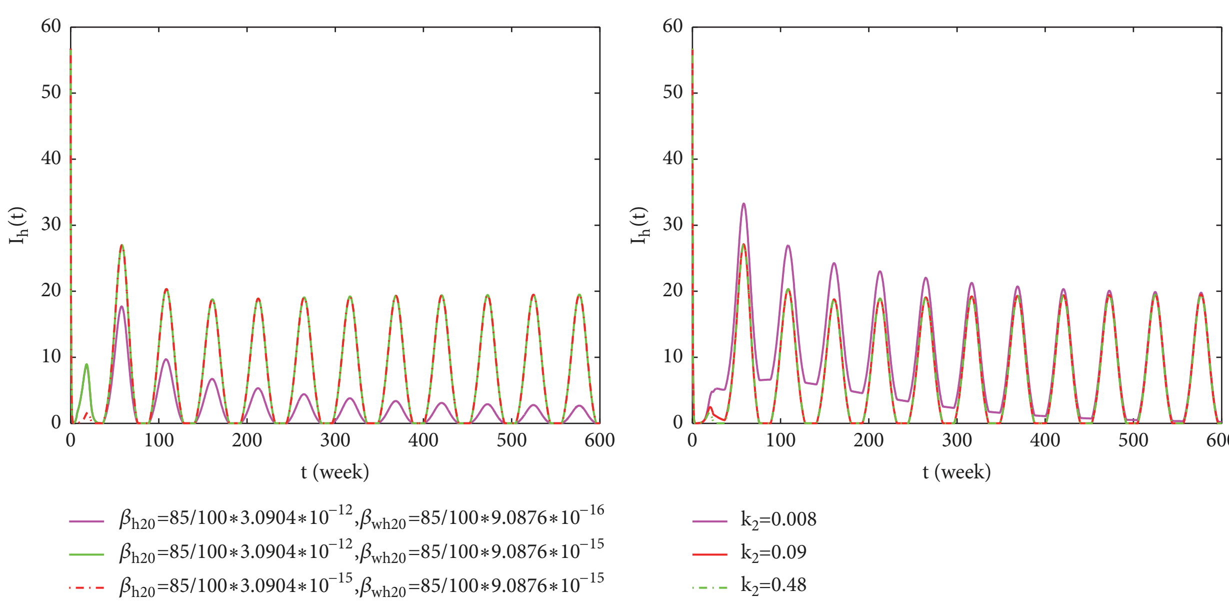

Figure 6: The curve-trend diagram of I_h with time, when β_{h2} or β_{wh2} takes different values.

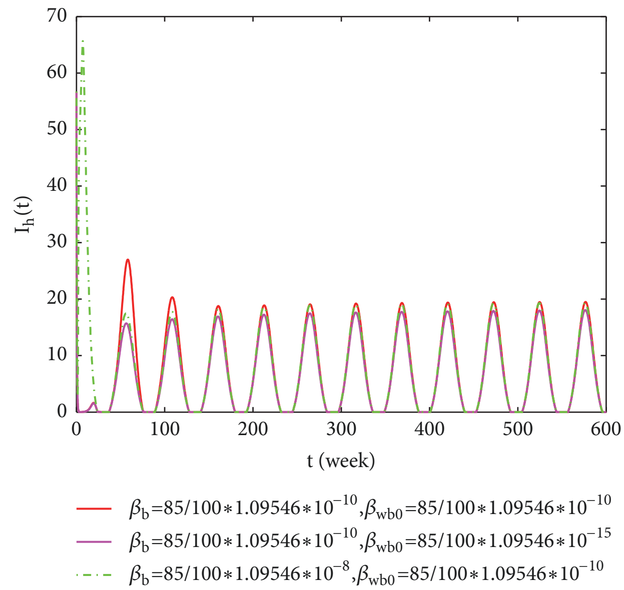

FIGURE 7: The curve-trend diagram of I_h with time, when β_b or β_{wb} takes different values.

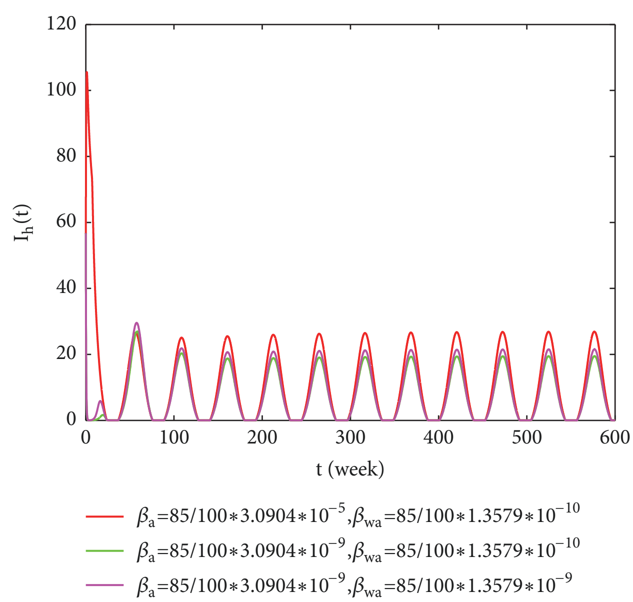

FIGURE 8: The curve-trend diagram of I_h with time, when β_a or β_{wa} takes different values.

poultry and human. We get the basic reproduction number R_0; it is the threshold which is endemic or not. If $R_0 < 1$, there is only the disease-free equilibrium E^0, and it is globally asymptotically stable, which implies that the disease dies out. If $R_0 > 1$, there is at least one positive periodic solution, that is, the disease will spread. Our numerical simulation suggests that raising α_a by killing infected poultry, reducing β_a, β_{h1}, and β_{h2} by closing farms, live-poultry markets, and wet markets, reducing β_{wb}, β_{wa}, β_{wh1}, and β_{wh2} by adjusting the temperature of the living environment, or increasing δ_w and p_a by routine disinfection in areas prone to outbreak and vaccination of poultry can reduce I_h, so as to control the occurrence and development of diseases. These can provide some measures of the intensity of interventions for public-health management on avian influenza A (H7N9) during high-risk period. In addition, our simulation shows that the temperature of the environment has a great effect on the prevalence of epidemic. Therefore, at suitable temperature for disease outbreak, human should not be in contact with birds and live poultry as much as possible.

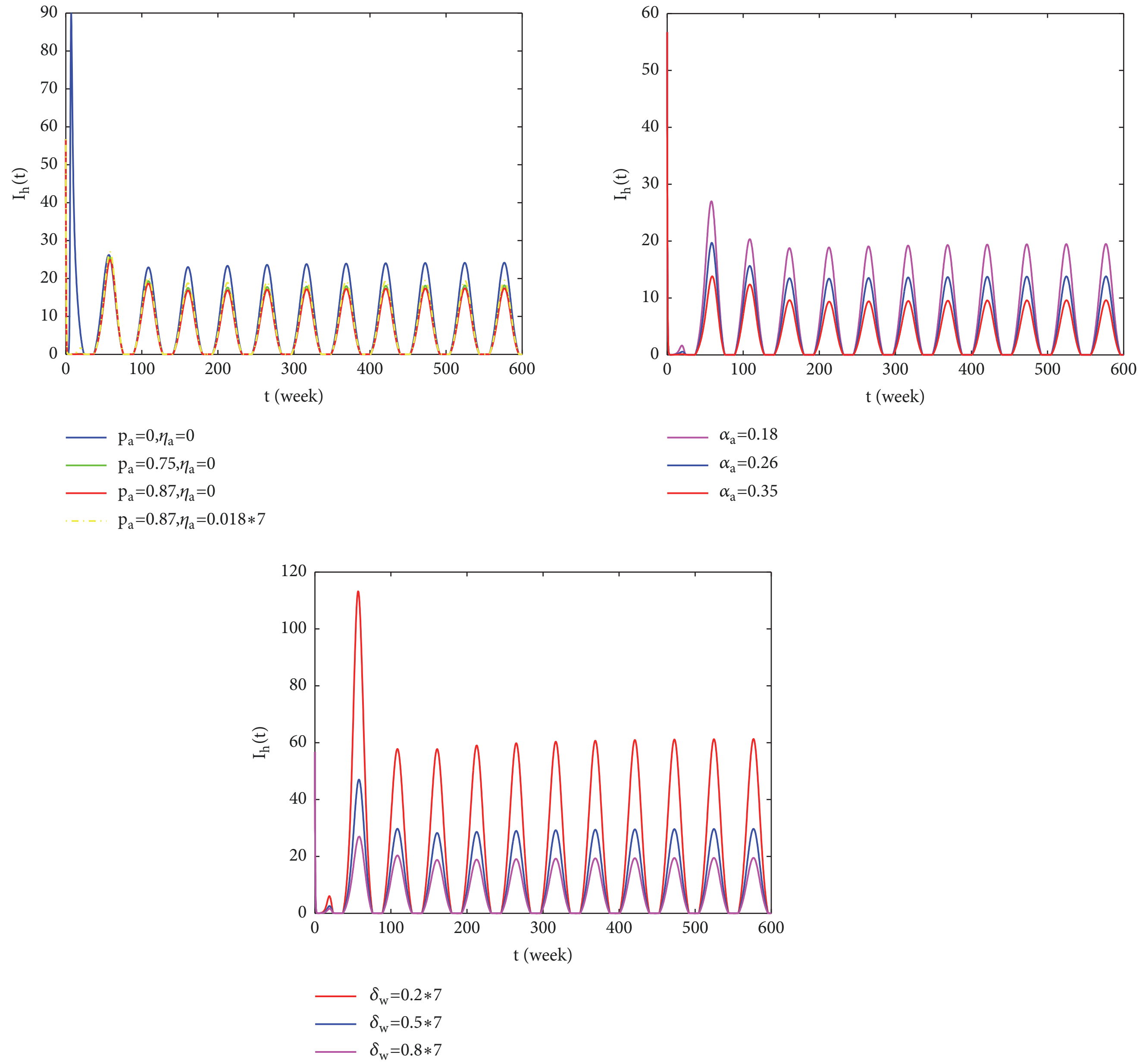

FIGURE 9: The curve-trend diagram of I_h with time, when parameters take different values.

Data Availability

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

Conflicts of Interest

The authors declare no competing financial interests.

Acknowledgments

This work is supported by the National Sciences Foundation of China (no. 61873154, no. 11314091, and no. 11331009) and the Natural Sciences Foundation of Shanxi Province (no. 201801D121008 and no. 201801D121026).

References

[1] D. J. Alexander, "A review of avian influenza in different bird species," *Veterinary Microbiology*, vol. 74, no. 1-2, pp. 3–13, 2000.

[2] E. C. J. Claas, A. D. M. E. Osterhaus, R. van Beek et al., "Human influenza A H5N1 virus related to a highly pathogenic avian influenza virus," *The Lancet*, vol. 351, no. 9101, pp. 472–477, 1998.

[3] WHO, *Cumulative Number of Confirmed Human Cases of Avian Influenza A(H5N1) Reported to WHO*, World Health Organization, 2017, http://www.who.int/influenza/human_animal_interface/H5N1_cumulative_table_archives/en/.

[4] N. H. Gao, Z. H. Lu, B. Cao et al., "Clinical findings in 111 cases of influenza A (H7N9) virus infection," *The New England Journal of Medicine*, vol. 368, no. 24, pp. 2277–2285, 2013.

[5] T. T.-Y. Lam, J. Wang, and Y. Shen, "The genesis and source of the H7N9 influenza viruses causing human infections in China," *Nature*, vol. 502, no. 7470, pp. 241–244, 2013.

[6] WHO, *Number of confirmed human cases of avian influenza A (H7N9) reported to World Health Organization*, World Health Organization, 2013, http://www.who.int/influenza/human_animal_interface/influenza_h7n9/08_ReportWebH7N9Number.pdf.

[7] R. Gao, B. Cao, and Y. Hu, "Human infection with a novel avian-origin influenza A (H7N9) virus," *The New England Journal of Medicine*, vol. 368, no. 20, pp. 1888–1897, 2013.

[8] T. Watanabe, M. Kiso, S. Fukuyama et al., "Characterization of H7N9 influenza A viruses isolated from humans," *Nature*, vol. 501, no. 7468, pp. 551–555, 2013.

[9] C.-J. Bao, L.-B. Cui, M.-H. Zhou, L. Hong, G. F. Gao, and H. Wang, "Live-animal markets and influenza A (H7N9) virus infection," *The New England Journal of Medicine*, vol. 368, no. 24, pp. 2337–2339, 2013.

[10] D. M. Rao, A. Chernyakhovsky, and V. Rao, "Modeling and analysis of global epidemiology of avian influenza," *Environmental Modeling and Software*, vol. 24, no. 1, pp. 124–134, 2009.

[11] M.-T. Li, Z. Jin, G.-Q. Sun, and J. Zhang, "Modeling direct and indirect disease transmission using multi-group model," *Journal of Mathematical Analysis and Applications*, vol. 446, no. 2, pp. 1292–1309, 2017.

[12] Y. Xiao, X. Sun, S. Tang, and J. Wu, "Transmission potential of the novel avian influenza A(H7N9) infection in mainland China," *Journal of Theoretical Biology*, vol. 352, pp. 1–5, 2014.

[13] J. Zhang, Z. Jin, G.-Q. Sun, X.-D. Sun, Y.-M. Wang, and B. Huang, "Determination of original infection source of H7N9 avian influenza by dynamical model," *Scientific Reports*, vol. 4, article 4846, no. 6183, 2014.

[14] N. K. Vaidya and L. M. Wahl, "Avian influenza dynamics under periodic environmental conditions," *SIAM Journal on Applied Mathematics*, vol. 75, no. 2, pp. 443–467, 2015.

[15] Y. Xing, L. Song, G.-Q. Sun, Z. Jin, and J. Zhang, "Assessing reappearance factors of H7N9 avian influenza in China," *Applied Mathematics and Computation*, vol. 309, pp. 192–204, 2017.

[16] S. Che, Y. Xue, and L. Ma, "The stability of highly pathogenic avian influenza epidemic model with saturated contact rate," *Applied Mathematics*, vol. 05, no. 21, pp. 3365–3371, 2014.

[17] Y. Li, P. Qin, and J. Zhang, "Dynamics analysis of avian influenza A(H7N9) epidemic model," *Discrete Dynamics in Nature and Society*, Art. ID 7485310, 12 pages, 2018.

[18] N. JonzEn, "Rapid advance of spring arrival dates in long-distance migratory birds," *Science*, vol. 312, no. 5782, pp. 1959–1961, 2006.

[19] A. C. Lowen, S. Mubareka, J. Steel, and P. Palese, "Influenza virus transmission is dependent on relative humidity and temperature," *PLoS Pathogens*, vol. 3, no. 10, p. e151, 2007.

[20] A. C. Lowen, J. Steel, S. Mubareka, and P. Palese, "High temperature (30°C) blocks aerosol but not contact transmission of influenza virus," *Journal of Virology*, vol. 82, no. 11, pp. 5650–5652, 2008.

[21] W. Wang and X. Zhao, "Threshold dynamics for compartmental epidemic models in periodic environments," *Journal of Dynamics and Differential Equations*, vol. 20, no. 3, pp. 699–717, 2008.

[22] F. Zhang and X. Zhao, "A periodic epidemic model in a patchy environment," *Journal of Mathematical Analysis and Applications*, vol. 325, no. 1, pp. 496–516, 2007.

[23] H. L. Smith and P. Waltman, *The Theory of the Chemostat*, Cambridge University Press, 1995.

[24] H. R. Thieme, "Convergence results and a Poincaré-Bendixson trichotomy for asymptotically autonomous differential equations," *Journal of Mathematical Biology*, vol. 30, no. 7, pp. 755–763, 1992.

[25] H. Smith, "Monotone dynamical systems: an introduction to the theory of competitive and cooperative systems," *Ams Ebooks Program*, vol. 41, no. 05, p. 174, 1995.

[26] X. Q. Zhao, *Dynamical Systems in Population Biology*, Springer, New York, NY, USA, 2003.