Research Article

Avian Influenza A (H7N9) Model Based on Poultry Transport Network in China

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In order to analyze the spread of avian influenza A (H7N9), we construct an avian influenza transmission model from poultry (including poultry farm, backyard poultry farm, live-poultry wholesale market, and wet market) to human according to poultry transport network. We obtain the threshold value for the prevalence of avian influenza A (H7N9) and also give the existence and number of the boundary equilibria and endemic equilibria in different conditions. We can see that poultry transport network plays an important role in controlling avian influenza A (H7N9). Finally, numerical simulations are presented to illustrate the effects of poultry in different places on avian influenza. In order to reduce human infections in China, our results suggest that closing the retail live-poultry market or preventing the poultry of backyard poultry farm into the live-poultry market is feasible in a suitable condition.

1. Introduction

Avian influenza A (H7N9) is a subtype of influenza viruses that have been detected in birds in the past. Until 2013 outbreak in China, no human infections with H7N9 viruses had ever been reported. But from March 31 to August 31, 2013, 134 cases had been reported in mainland China, resulting in 45 deaths [1]. However, the virus came back in November 2013 again. Afterwards the disease came back in November every year. In fact, the second outbreak occurred from November 2013 to May 2014. The third outbreak occurred from November 2014 to June 2015. The fourth outbreak occurred from November 2015 to June 2016. And the fifth outbreak occurred from September 2016 to May 2017 (NHFPC [1]). The disease causes a high death rate. In China, from March 2013 to May 2017, H7N9 has resulted in 1263 human cases including 459 deaths with a death rate of nearly 37%. In China, from September 2016 to May 2017, provinces with human cases are shown as Figure 1. H7N9 virus does not induce clinical signs in poultry and is classified as a low pathogenicity avian influenza virus (LPAIV) [2]. However, the virus can infect humans and most of the reported cases of human H7N9 infection have resulted in severe respiratory illness [3].

Jones et al. [4] 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. Pantin-Jackwood et al. [3] 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, and it is therefore conceivable that passerine birds may serve as vectors for transmission of H7N9 virus to domestic poultry [4]. Zhang et al. [5] concluded that migrant birds are the original infection source. Many authors investigated the epidemic model which describes the transmission of avian influenza among birds and humans [8–15]. Liu et al. [16] constructed 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 analyzed their dynamical behavior. Lin et al. [17] developed three different SIRS models to fit the observed human cases between March 2013 and July 2015 in China and found that environmental
transmission via viral shedding of infected chickens had contributed to the spread of H7N9 human cases in China. Chen and Wen [18] took into account gene mutation in poultry. Guo et al. [19] proposed and analyzed an SE-SEIS avian-human influenza model. Mu and Yang [20] analyzed an SI-SEIR avian-human influenza model with latent period and nonlinear recovery rate. Gourley et al. [21] analyzed the patchy model for the spatiotemporal distribution of a migratory bird species. Bourouiba et al. [22] 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. In China, in 2013, to control the outbreak, 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. 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 [6, 7]. How is avian influenza A (H7N9) transmitted from live-poultry to human in China? In order to reveal the fact, the global network model of avian influenza A (H7N9) is constructed based on poultry transport network. The relationship between the global system and subsystem is analyzed. The corresponding risk indices are obtained. We study the impact of subsystems on the risk index of the global system. When the disease occurs, it can provide theoretical guidance for the global and local transport of poultry.

In this paper, we construct an avian influenza A (H7N9) transmission model from live poultry (including poultry farm, backyard poultry farm, live-poultry wholesale market, and wet market) to human for the heterogenous environments which affect the spread of H7N9. The remaining part of this paper is organized as follows: in Section 2, we first establish the model based on poultry transport network. We derive the threshold value of the model. In Sections 3 and 4, we discuss the different boundary and endemic equilibrium in the different thresholds. Section 5 gives the
effect of different transmission rate on H7N9 by numerical simulation. Finally, concluding remarks are made in Section 6.

2. Model Based on Poultry Transport Network

The avian population is classified into poultry farm, backyard poultry farm, live-poultry wholesale market, and wet market (the retail live-poultry market). According to the present situation in China, the backyard poultry feeding is regarded as a large node, which is considered to be connected with all other nodes (except poultry farm) in network. The relationship diagram of poultry transport and contacts between human and poultry are described in Figure 2. Let \( N_{ta}(t), N_{pa}(t), \) and \( N_{ma}(t) \) be the total number of poultry in \( i \)th poultry farm, \( j \)th live-poultry wholesale market, and \( k \)th wet market at time \( t \), respectively, where \( N_{ta}(t), N_{pa}(t), \) and \( N_{ma}(t) \) are classified into two subclasses: susceptible and infective, denoted by \( S_{ta}(t) \) and \( I_{ta}(t) \), \( S_{pa}(t) \) and \( I_{pa}(t) \), and \( S_{ma}(t) \) and \( I_{ma}(t) \), respectively. Suppose there are \( L \) poultry farms, \( M \) live-poultry wholesales, and \( K \) wet markets, namely, \( i = 1, \ldots, L; j = 1, \ldots, M; k = 1, \ldots, K \). And they are independent of each other. Let \( N_{h}(t) \) be the total number of human at time \( t \). The human population is classified into three subclasses: susceptible, infective, and recovered, denoted by \( S_{h}(t), I_{h}(t), \) and \( R_{h}(t) \), respectively. All new recruitments of human population and avian population are susceptible. 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 avian and a susceptible human. 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. We neglect death rates of the poultry individuals during the transport process. The detailed description of dynamical transmission of H7N9 avian influenza is described in the following flowchart (Figure 3).

The corresponding dynamical model can be seen in the following equation:

\[
\begin{align*}
\frac{dS_{ta}(t)}{dt} &= A_{t} - \beta_{ta} S_{ta} I_{ta} - d_{t} S_{ta} - \sum_{j} a_{ij} S_{ta}, \\
\frac{dI_{ta}(t)}{dt} &= \beta_{ta} S_{ta} I_{ta} - d_{t} I_{ta} - \alpha_{t} I_{ta} - \sum_{j} a_{ij} I_{ta}, \quad i = 1, \ldots, L, \\
\frac{dS_{ba}(t)}{dt} &= A_{b} - \beta_{ba} S_{ba} I_{ba} - d_{b} S_{ba} - \sum_{j} l_{j} S_{ba} - \sum_{k} c_{k} S_{ba}, \\
\frac{dI_{ba}(t)}{dt} &= \beta_{ba} S_{ba} I_{ba} - d_{b} I_{ba} - a_{b} I_{ba} - \sum_{j} l_{j} I_{ba} - \sum_{k} c_{k} I_{ba}, \\
\frac{dS_{pa}(t)}{dt} &= \sum_{i} a_{ij} S_{pa} + l_{j} S_{pa} - \beta_{pa} S_{pa} I_{pa} - d_{p} S_{pa} - \sum_{k} b_{jk} S_{pa}, \quad j = 1, \ldots, M, \\
\frac{dI_{pa}(t)}{dt} &= \sum_{j} a_{ij} I_{pa} + l_{j} I_{pa} + \beta_{pa} S_{pa} I_{pa} - d_{p} I_{pa} - \alpha_{p} I_{pa} - \sum_{k} b_{jk} I_{pa}, \\
\frac{dS_{ma}(t)}{dt} &= \sum_{j} b_{jk} S_{pa} + c_{k} S_{ba} - \beta_{ma} S_{ma} I_{ma} - d_{m} S_{ma}, \\
\frac{dI_{ma}(t)}{dt} &= \sum_{j} b_{jk} I_{pa} + c_{k} I_{ba} + \beta_{ma} S_{ma} I_{ma} - d_{m} I_{ma} - \alpha_{m} I_{ma}, \quad k = 1, \ldots, K, \\
\frac{dS_{h}(t)}{dt} &= A_{h} - \sum_{k} \beta_{kh} S_{ma} I_{ma} - \beta_{h} S_{h} I_{ba} - d_{h} S_{h}, \\
\frac{dI_{h}(t)}{dt} &= \sum_{k} \beta_{kh} S_{ma} I_{ma} + \beta_{h} S_{h} I_{ba} - d_{h} I_{h} - \alpha_{h} I_{h} - \gamma_{h} I_{h}, \\
\frac{dR_{h}(t)}{dt} &= \gamma_{h} I_{h} - d_{h} R_{h}.
\end{align*}
\]
The interpretations of parameters of system (1) are described in Table 1. The parameters in system (1) are all nonnegative constants.

The variation of the number of poultry in \(i\)th poultry farm \(N_{fa}(t)\) is

\[
\frac{dN_{fa}(t)}{dt} = A_{i} - d_{i}N_{fa} - \alpha_{i}I_{fa} - \sum_{j} a_{ij}N_{fa}^{j},
\]

(2)

and thus,

\[
N_{fa}(t) \leq \frac{A_{i}}{d_{i} + \sum_{j}a_{ij}} = W_{fa}.\]

(3)

Similarly, the variation of the number of poultry in backyard poultry farm \(N_{ba}(t)\) is

\[
\frac{dN_{ba}(t)}{dt} = A_{b} - d_{b}N_{ba} - \alpha_{b}I_{ba} - \sum_{j} l_{j}N_{ba} - \sum_{k} c_{k}N_{ba},
\]

(4)

and thus,

\[
N_{ba}(t) \leq \frac{A_{b}}{d_{b} + \sum_{j}l_{j} + \sum_{k}c_{k}} = W_{ba}.
\]

(5)

The variation of the number of poultry in \(j\)th live-poultry wholesale market \(N_{pa}(t)\) is
The natural death rate (including slaughter) of the avian in different places
The disease-related death rate of the infected avian in different places
The transmission rate from infective avian to susceptible avian in different places
The transport rate of individuals from jth live-poultry wholesale market to kth wet market
The transport rate of individuals from backyard poultry farm to jth live-poultry wholesale market
The transport rate of individuals from backyard poultry farm to kth wet market
The variation of the number of human
The recovery rate of the human

and thus,

\[
\frac{dN_{pa}^j(t)}{dt} = \sum_i a_{ij}N_{fa}^i + J_jN_{ba} - d_pN_{pa}^j - a_p^jI_{pa} - \sum_k b_{jk}N_{pa}^j,
\]

(6)

and thus,

\[
N_{pa}^j(t) \leq \frac{\sum_i a_{ij}N_{fa}^i + J_jN_{ba}}{d_p + \sum_k b_{jk}} \leq \frac{\sum_i \left(\left(\frac{a_{ij}N_{fa}^i}{d_p + \sum_k b_{jk}}\right) + \left(\frac{J_jN_{ba}}{d_p + \sum_k b_{jk}}\right)\right)\left(\frac{d_p + \sum_k b_{jk}}{d_p + \sum_k b_{jk}}\right)\left(\frac{d_p + \sum_k b_{jk}}{d_p + \sum_k b_{jk}}\right)}{d_m} = W_{pa}^j.
\]

(7)

The variation of the number of poultry in kth wet market

\[
\frac{dN_{ma}^k(t)}{dt} = \sum_j b_{jk}N_{pa}^j + c_kN_{ba} - d_m^kN_{ma}^k - a_m^kN_{ma}^k.
\]

(8)

The variation of the number of human

\[
\frac{dN_h(t)}{dt} = A_h - d_hN_h - a_hI_h,
\]

(10)

and thus,

\[
N_h(t) \leq \frac{A_h}{d_h}.
\]

(11)

For convenience, we denote the positive solution

\[
(S_{fa}^1, ..., S_{fa}^i, I_{fa}^1, ..., I_{fa}^i, I_{ba}^1, S_{ba}^1, ..., S_{ba}^i, I_{ba}^1, ..., I_{pa}^i, S_{ma}^1, ..., S_{ma}^i, I_{ma}^1, ..., I_{ma}^i, S_{ha}^1, ..., S_{ha}^i, I_{ha}^1, ..., I_{ha}^i).
\]

Let \( G \) be the positive solution of system (1) by \( (S, I) \). Then \( G \) is a positively invariant for system (1).
System (12) has the unique positive equilibrium\( S^0 = (S_{10}, S_{0}, S_{p0}, S_{m0}, S_{0})\), where \( S_{10} = A_l/(d_t + \sum a_{ij})\), \( S_{ba} = A_l/(d_b + \sum j + \sum \kappa c_k)\), \( S_{pa} = (\sum a_{ij} S_{10} + l S_{ba})/(d_p + \sum \beta_{ijk} S_{10} + \sum \kappa S_{ba})/d_m\), and \( S_{h} = A_b/d_h\). Thus, \( E_0 = (S_{10}, 0, S_{ba}, 0, S_{pa}, 0, S_{m0}, 0, S_{0})\) is the disease-free equilibrium of system (1).

According to the concepts of the next generation matrix and reproduction number presented in [23, 24], we define

\[
F = \begin{pmatrix}
F_{11} & 0 & 0 & 0 \\
0 & F_{22} & 0 & 0 \\
0 & 0 & F_{33} & 0 \\
0 & 0 & 0 & F_{44}
\end{pmatrix},
\]

\[
V = \begin{pmatrix}
V_{11} & 0 & 0 & 0 \\
0 & V_{22} & 0 & 0 \\
0 & 0 & V_{33} & 0 \\
0 & 0 & 0 & V_{44}
\end{pmatrix},
\]

where

\[
F_{11} = \begin{pmatrix}
\beta_1 S_{10} & 0 & \cdots & 0 \\
0 & \beta_2 S_{10} & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & \beta_{44} S_{10}
\end{pmatrix},
\]

\[
F_{22} = \begin{pmatrix}
\beta_p S_{pa} & 0 & \cdots & 0 \\
0 & \beta_p S_{pa} & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & \beta_p S_{pa}
\end{pmatrix},
\]

\[
F_{33} = \begin{pmatrix}
\beta_m S_{p0} & 0 & \cdots & 0 \\
0 & \beta_m S_{p0} & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & \beta_m S_{p0}
\end{pmatrix},
\]

\[
F_{44} = \begin{pmatrix}
\beta_m S_{m0} & 0 & \cdots & 0 \\
0 & \beta_m S_{m0} & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & \beta_m S_{m0}
\end{pmatrix},
\]

\[
V_{11} = \begin{pmatrix}
d_t + \alpha_t + \sum a_{ij} & 0 & \cdots & 0 \\
0 & d_t + \alpha_t + \sum a_{ij} & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & d_t + \alpha_t + \sum a_{ij}
\end{pmatrix},
\]

\[
V_{22} = \begin{pmatrix}
d_p + \alpha_p + \sum j + \sum \kappa c_k & 0 & \cdots & 0 \\
0 & d_p + \alpha_p + \sum j + \sum \kappa c_k & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & d_p + \alpha_p + \sum j + \sum \kappa c_k
\end{pmatrix},
\]

\[
V_{33} = \begin{pmatrix}
d_h + \alpha_h + \sum h & 0 & \cdots & 0 \\
0 & d_h + \alpha_h + \sum h & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & d_h + \alpha_h + \sum h
\end{pmatrix},
\]

\[
V_{44} = \begin{pmatrix}
d_m + \alpha_m & 0 & \cdots & 0 \\
0 & d_m + \alpha_m & \cdots & 0 \\
\vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & d_m + \alpha_m
\end{pmatrix}.
\]

(13)

Set \( R_0 = \rho(FV^{-1}) \), where \( \rho \) represents the spectral radius of the matrix. Then, \( R_0 \) is called the reproduction number for system (1), where

\[
R_{i0} = \frac{\beta_i S_{10}}{d_t + \alpha_t + \sum a_{ij}},
\]

\[
R_{j0} = \frac{\beta_p S_{pa}}{d_p + \alpha_p + \sum j + \sum \kappa c_k},
\]

\[
R_{m0} = \frac{\beta_m S_{m0}}{d_m + \alpha_m},
\]

\[
R_{bo} = \frac{\beta_b S_{bo}}{d_b + \alpha_b + \sum j + \sum \kappa c_k}.
\]

(14)

If \( R_0 < 1 \), then system (1) has the disease-free equilibrium \( E_0 \), and \( E_0 \) is locally asymptotically stable.

**Remark 1.** If we do not consider backyard poultry farm, then system (1) becomes

\[
\frac{dS_{10}(t)}{dt} = A_l - \beta_1 S_{10} I_{1a} - d_t S_{10} - \sum a_{ij} S_{10},
\]

\[
\frac{dI_{1a}(t)}{dt} = \beta_1 S_{10} I_{1a} - d_t I_{1a} - \alpha_i I_{1a} - \sum a_{ij} I_{1a}, \quad i = 1, \ldots, L,
\]

\[
\frac{dS_{pa}(t)}{dt} = \sum a_{ij} S_{pa} - \beta_p S_{pa} I_{pa} - d_p S_{pa} - \sum b_{jk} S_{pa},
\]

\[
\frac{dI_{pa}(t)}{dt} = \sum a_{ij} I_{pa} + \beta_p S_{pa} I_{pa} - d_p I_{pa} - \alpha_p I_{pa} - \sum b_{jk} I_{pa}, \quad j = 1, \ldots, M,
\]

\[
\frac{dS_{m0}(t)}{dt} = \sum b_{jk} S_{m0} - \beta_m S_{m0} I_{m0} - d_m S_{m0},
\]

\[
\frac{dI_{m0}(t)}{dt} = \sum b_{jk} I_{m0} + \beta_m S_{m0} I_{m0} - d_m I_{m0} - \alpha_m I_{m0}, \quad k = 1, \ldots, K,
\]

\[
\frac{dS_{h}(t)}{dt} = A_h - \sum b_{kh} S_{h} - d_h S_{h},
\]

\[
\frac{dI_{h}(t)}{dt} = \sum b_{kh} I_{h} - d_h I_{h} - \alpha_h I_{h} - \gamma_h I_{h},
\]

\[
\frac{dR_{b}(t)}{dt} = \gamma_h I_{h} - d_h R_{b}.
\]

(16)

A similar analysis is available for the above system.
3. Analysis of Subsystems of System (1)

Consider the poultry of the poultry farm subsystem, given by the first two equations of system (1), as follows:

\[
\begin{align*}
\frac{dS_{ia}(t)}{dt} &= \alpha_i S_{ia} R_{ia} - \beta_i S_{ia} S_{ia} - \sum_j a_{ij} I_{ja} S_{ia}, \\
\frac{dI_{ia}(t)}{dt} &= \beta_i S_{ia} I_{ia} - \alpha_i I_{ia} - \sum_j a_{ij} I_{ia}.
\end{align*}
\tag{17}
\]

Let the right-hand side of system (17) equals to zero; when \( I_{ia} \neq 0 \), we obtain

\[
S_{ia}^* = \frac{d_i + \alpha_i + \sum_j a_{ij}}{\beta_i},
\]

\[
I_{ia}^* = \frac{A_i^{*} \beta_i - (d_i + \sum_j a_{ij})(d_i + \alpha_i + \sum_j a_{ij})}{\beta_i(d_i + \alpha_i + \sum_j a_{ij})}.
\tag{18}
\]

If \( R_{i0}^* > 1 \), system (17) has the positive equilibrium \((S_{ia}^*, I_{ia}^*)\). If \( R_{i0}^* < 1 \), system (17) has only the disease-free equilibrium \((S_{ia}, 0)\).

Remark 2.

(1) If \( \min \{R_{i0}^j\} > 1 \), then each farm has the positive equilibrium.

(2) If \( \max \{R_{i0}^j\} > 1 \), then some of the poultry farms have the positive equilibrium, and the others have only the disease-free equilibrium.

Consider the poultry of the backyard poultry farm subsystem, given by the third and fourth equations of system (1), as follows:

\[
\begin{align*}
\frac{dS_{ba}(t)}{dt} &= A_b - \beta_b S_{ba} I_{ba} - d_b S_{ba} - \sum j l_j S_{ba} - \sum k c_k S_{ba}, \\
\frac{dI_{ba}(t)}{dt} &= \beta_b S_{ba} I_{ba} - \alpha_b I_{ba} - \sum j l_j I_{ba} - \sum k c_k I_{ba}.
\end{align*}
\tag{19}
\]

Let the right-hand side of system (19) equals to zero; when \( I_{ba} \neq 0 \), we obtain

\[
S_{ba}^* = \frac{d_b + \alpha_b + \sum_j l_j + \sum k c_k}{\beta_b},
\]

\[
I_{ba}^* = \frac{A_b - (d_b + \sum_j l_j + \sum k c_k)S_{ba}}{\beta_b S_{ba}}.
\tag{20}
\]

If \( R_{i0} > 1 \), system (19) has the positive equilibrium \((S_{ba}^*, I_{ba}^*)\). If \( R_{i0} < 1 \), system (19) has only the disease-free equilibrium \((S_{ba}, 0)\).

Consider the poultry of the live-poultry wholesale market subsystem, given by the fifth and sixth equations of system (1), as follows:

\[
\begin{align*}
\frac{dS_{pa}(t)}{dt} &= \sum_i a_{ij} S_{ia} + l_j S_{ba} - \beta_p S_{pa} I_{pa} - d_p S_{pa} - \sum k b_k S_{pa}, \\
\frac{dI_{pa}(t)}{dt} &= \sum_i a_{ij} I_{ia} + l_j I_{ba} + \beta_p S_{pa} I_{pa} - d_p I_{pa} - \sum k b_k I_{pa}.
\end{align*}
\tag{21}
\]

Let the right-hand side of system (21) equals to zero; when \( I_{pa} \neq 0 \), we can divide it into two cases.

If \( I_{pa} = 0 \) and \( I_{ba} = 0 \), then we have

\[
S_{pa}^* = \frac{d_p + \alpha_p + \sum_k b_k}{\beta_p},
\]

\[
I_{pa}^* = \frac{\sum a_{ij} S_{i0} + l_j S_{ba} - (d_p + \sum k b_k)S_{pa}}{d_p + \alpha_p + \sum_k b_k - \beta_p S_{pa}}.
\tag{22}
\]

If \( R_{pa}^j > 1 \), then system (21) has the positive equilibrium \((S_{pa}^*, I_{pa}^*)\).

If \( I_{pa} \neq 0 \) or \( I_{ba} \neq 0 \), then we obtain

\[
I_{pa}^* = \frac{\sum a_{ij} I_{i0} + l_j I_{ba}}{d_p + \alpha_p + \sum_k b_k - \beta_p S_{pa}}.
\tag{23}
\]

where

\[
b_1 = -\beta_p \left( d_p + \sum_k b_k \right) < 0,
\]

\[
b_2 = \beta_p \left( \sum a_{ij} S_{i0} + l_j S_{ba} \right) + \beta_p \left( \sum a_{ij} I_{i0} + l_j I_{ba} \right) + \left( d_p + \sum_k b_k \right) \left( d_p + \alpha_p + \sum_k b_k \right) > 0,
\]

\[
b_3 = -\left( d_p + \alpha_p + \sum_k b_k \right) \left( \sum a_{ij} S_{i0} + l_j S_{ba} \right) < 0.
\tag{24}
\]

Because \( b_2 - 4b_1b_3 > 0 \), the solutions of the above equation are

\[
S_{pa1}^* = \frac{-b_2 + \sqrt{b_2^2 - 4b_1b_3}}{2b_1} > 0,
\]

\[
S_{pa2}^* = \frac{-b_2 - \sqrt{b_2^2 - 4b_1b_3}}{2b_1} > 0.
\tag{25}
\]

If \( R_{p0}^j = (d_p + \alpha_p + \sum_k b_k / (\beta_p S_{pa2})) > 1 \), system (21) has two positive equilibria \((S_{pa1}^*, I_{pa1}^*)\) and \((S_{pa2}^*, I_{pa2}^*)\). If \( R_{p0}^j < 1 \) and \( R_{p0}^j = (d_p + \alpha_p + \sum_k b_k / (\beta_p S_{pa2})) > 1 \), system (21) has one positive equilibrium \((S_{pa1}^*, I_{pa1}^*)\). If \( R_{p0}^j < 1 \), system (21) has no positive equilibrium.

Consider the poultry of the wet market (the retail live-poultry market) subsystem, given by the seventh and eighth equations of system (1), as follows:
Let the right-hand side of system (26) equals to zero, when $I_{pa}^k \neq 0$, we can divide it into two cases. If $I_{pa} = 0$ and $I_{ba} = 0$, then we have

$$S_{m}^{k*} = \frac{d_m + \alpha_m}{\beta_m},$$

$$I_{m}^{k*} = \frac{\sum b_{jk} S_{pa}^{j*} + c_k S_{ba}^{j*} - d_m S_{m}^{k*}}{d_m + \alpha_m}.$$

If $R_{ma}^k > 1$, then system (26) has the positive equilibrium $(S_{m}^{k*}, I_{m}^{k*})$. If $I_{pa} \neq 0$ or $I_{ba} \neq 0$, then we have

$$I_{ma}^{k*} = \frac{\sum b_{jk} I_{pa}^{j*} + c_k I_{ba}^{j*}}{d_m + \alpha_m - \beta_m S_{ma}^{k*}}.$$  

$$g_1 S_{ma}^{k*} + g_2 S_{ma}^{k*} + g_3 = 0.$$

where

$$g_1 = -d_m \beta_m < 0,$$

$$g_2 = \beta_m \left( \sum b_{jk} S_{pa}^{j*} + c_k S_{ba}^{j*} \right) + \beta_m \left( \sum b_{jk} I_{pa}^{j*} + c_k I_{ba}^{j*} \right) + d_m (d_m + \alpha_m) > 0,$$

$$g_3 = -(d_m + \alpha_m) \left( \sum b_{jk} S_{pa}^{j*} + c_k S_{ba}^{j*} \right) < 0.$$

Because $g_2^2 - 4g_1g_3 > 0$, the solutions of the above equation are

$$S_{ma1}^{k*} = \frac{-g_2 + \sqrt{g_2^2 - 4g_1g_3}}{2g_1} > 0,$$

$$S_{ma2}^{k*} = \frac{-g_2 + \sqrt{g_2^2 - 4g_1g_3}}{2g_1} > 0.$$

If $R_{ma0}^k = \left( (d_m + \alpha_m)/(\beta_m S_{ma}^{k*}) \right) > 1$, system (26) has two positive equilibria $(S_{ma1}^{k*}, I_{ma1}^{k*})$ and $(S_{ma2}^{k*}, I_{ma2}^{k*})$. If $R_{ma0}^k < 1$ and $R_{ma0}^{k1} = \left( (d_m + \alpha_m)/(\beta_m S_{ma}^{k*}) \right) > 1$, system (26) has one positive equilibrium $(S_{ma1}^{k*}, I_{ma1}^{k*})$. If $R_{ma0}^k < 1$, system (26) has no positive equilibrium.

Consider the human subsystem, given by the last three equations of system (1), as follows:

$$\frac{dS_{h}}{dt} = A_h - \sum \beta_{kh} S_{h} I_{ma}^k - \beta_h S_{h} b_{h} - d_h S_{h},$$

$$\frac{dI_{h}}{dt} = \sum \beta_{kh} S_{h} I_{ma}^k + \beta_h S_{h} b_{h} - d_h I_{h} - \alpha_h I_{h} - \gamma_h I_{h},$$

$$\frac{dR_{h}}{dt} = \gamma_h I_{h} - d_h R_{h}.$$

(31)

Since the first two equations of system (31) are independent of the variable $R_{h}$, we only need to analyze the first two equations of system (31). Let the right-hand side of system (31) equals to zero, when $I_{h} \neq 0$, if $I_{ma}^k \neq 0$ or $I_{ba} \neq 0$, then we have

$$S_{h}^* = \frac{A_h}{\sum \beta_{kh} K_{ma}^k + \beta_h b_{h} + d_h},$$

$$I_{h}^* = \left( \sum \beta_{kh} K_{ma}^k + \beta_h b_{h} \right) S_{h}^*/d_h + \alpha_h + \gamma_h.$$

(32)

4. Analysis of the Full System (1)

We analyze the following equivalent system:

$$\frac{dS_{ma}^j}{dt} = A_{ma}^j - \beta_j S_{ma}^j - d_j S_{ma}^j - \sum a_j S_{ma}^j,$$

$$\frac{dI_{ma}^j}{dt} = \beta_j S_{ma}^j - d_j I_{ma}^j - \alpha_j I_{ma}^j - \sum a_j I_{ma}^j,$$

$$\frac{dR_{ma}^j}{dt} = A_h - \beta_j S_{ma}^j - d_j R_{ma}^j - \alpha_j R_{ma}^j - \sum a_j R_{ma}^j,$$

(33)
For the sake of discussion, without loss of generality, we assume that a node has at least one link with the nodes in the next layer. So we have the following cases.

Case 1. If \( R_0 = \max_{1 \leq i \leq L, 1 \leq j \leq M, 1 \leq k \leq K} \left\{ R_{ij}^{(1)}, R_{ij}^{(2)}, R_{ij}^{(3)}, R_{ij}^{(4)} \right\} < 1 \), system (33) has only the disease-free equilibrium \( E_0 = \left( \frac{S_0}{L}, \frac{0}{L}, \frac{S_0}{M}, \frac{0}{M}, \frac{0}{K}, \frac{0}{K}, \frac{0}{K}, \frac{S_0}{K} \right) \). Namely, when all poultry has no avian influenza, human will not be infected with avian influenza.

Case 2. If \( \max_{1 \leq i \leq L, 1 \leq j \leq M} \left\{ R_{ij}^{(1)}, R_{ij}^{(2)} \right\} < 1 \), \( R_{0i} < 1 \), and \( \min_{1 \leq k \leq K} \left\{ R_{ik}^{(3)} \right\} > 1 \), system (33) has the boundary equilibrium

\[
E^c = \left( \frac{S_0}{L}, \frac{0}{L}, \frac{S_0}{M}, \frac{0}{M}, \frac{0}{K}, \frac{0}{K}, \frac{0}{K}, \frac{S_0}{K} \right).
\]

This shows that avian influenza A (H7N9) virus is most likely transmitted from the retail live-poultry market to humans when poultry has no disease in other types of farms.

Case 3. If \( \max_{1 \leq i \leq L} \left\{ R_{i0}^{(1)} \right\} < 1 \), \( R_{00} < 1 \), and \( \min_{1 \leq k \leq K} \left\{ R_{k0}^{(3)} \right\} > 1 \), system (33) has the boundary equilibrium as described next. If \( \min_{1 \leq k \leq K} \left\{ R_{k0}^{(3)} \right\} > 1 \) and \( \max_{1 \leq k \leq K} \left\{ R_{k0}^{(4)} \right\} < 1 \), system (33) has one boundary equilibrium:

\[
E^c = \left( \frac{S_0}{L}, \frac{0}{L}, \frac{S_0}{M}, \frac{0}{M}, \frac{0}{K}, \frac{0}{K}, \frac{0}{K}, \frac{S_0}{K} \right).
\]

This shows that avian influenza A (H7N9) virus is most likely transmitted from the secondary wholesale market to the retail live-poultry market and then to humans [6, 7]. And there may be two boundary equilibria.

Case 4. If \( \max_{1 \leq i \leq L} \left\{ R_{i0}^{(1)} \right\} < 1 \) and \( R_{00} > 1 \), system (33) has the boundary equilibrium as described next. If \( \min_{1 \leq k \leq M} \left\{ R_{k0}^{(1)} \right\} > 1 \), \( \max_{1 \leq k \leq M} \left\{ R_{k0}^{(1)} \right\} < 1 \), and \( \max_{1 \leq k \leq K} \left\{ R_{k0}^{(3)} \right\} < 1 \), system (33) has one boundary equilibrium:

\[
E^c = \left( \frac{S_0}{L}, \frac{0}{L}, \frac{S_0}{M}, \frac{0}{M}, \frac{0}{K}, \frac{0}{K}, \frac{0}{K}, \frac{S_0}{K} \right).
\]
If \( \min_{1 \leq k \leq K} R_{p0}^j > 1 \) and \( \max_{1 \leq k \leq K} R_{p0}^j < 1 \), and \( \min_{1 \leq k \leq K} R_{k0}^j > 1 \), system (33) has two boundary equilibria:

\[
E^1 = \left( \frac{S_{fa}^*, S_{ab}^*, S_{ba}^*, S_{pa}^*, S_{pp}^*, \chi_{p0}^*, \chi_{p1}^*, \chi_{p2}^*, \chi_{p3}^*, \chi_{pm}^*, \chi_{ph}^*, \chi_{pmn}^*, \chi_{phm}}{\mu_{fa*M}^*, \mu_{pa*M}^*, \mu_{mp*M}^*, \mu_{pp*M}^*, \mu_{pm*M}^*, \mu_{ph*M}^*, \mu_{pmn*M}^*, \mu_{phm*M}^*} \right).
\]

If \( \min_{1 \leq j \leq M^1} R_{p0}^j > 1 \), \( \max_{1 \leq j \leq M^1} R_{p0}^j < 1 \), and \( \min_{1 \leq j \leq M^1} R_{k0}^j > 1 \), system (33) has two boundary equilibria:

\[
E^2 = \left( \frac{S_{fa}^*, S_{ab}^*, S_{ba}^*, S_{pa}^*, S_{pp}^*, \chi_{p0}^*, \chi_{p1}^*, \chi_{p2}^*, \chi_{p3}^*, \chi_{pm}^*, \chi_{ph}^*, \chi_{pmn}^*, \chi_{phm}}{\mu_{fa*M}^*, \mu_{pa*M}^*, \mu_{mp*M}^*, \mu_{pp*M}^*, \mu_{pm*M}^*, \mu_{ph*M}^*, \mu_{pmn*M}^*, \mu_{phm*M}^*} \right).
\]

If \( \min_{1 \leq j \leq M^1} R_{p0}^j > 1 \) and \( \min_{1 \leq j \leq M^1} R_{k0}^j > 1 \), system (33) has four boundary equilibria

\[
E^3 = \left( \frac{S_{fa}^*, S_{ab}^*, S_{ba}^*, S_{pa}^*, S_{pp}^*, \chi_{p0}^*, \chi_{p1}^*, \chi_{p2}^*, \chi_{p3}^*, \chi_{pm}^*, \chi_{ph}^*, \chi_{pmn}^*, \chi_{phm}}{\mu_{fa*M}^*, \mu_{pa*M}^*, \mu_{mp*M}^*, \mu_{pp*M}^*, \mu_{pm*M}^*, \mu_{ph*M}^*, \mu_{pmn*M}^*, \mu_{phm*M}^*} \right).
\]

When the poultry of poultry farms has avian influenza, and the poultry of backyard poultry farm has no avian influenza, we can obtain four cases. In four cases, human is most likely transmitted from the poultry farm to the secondary wholesale market, then to the retail live-poultry market, and finally to humans.

**Case 6.** If \( \min_{1 \leq j \leq L} R_{b0}^j > 1 \) and \( R_{b0} > 1 \), system (33) has the positive equilibrium as described next.

If \( \min_{1 \leq j \leq M^1} R_{b0}^j > 1 \), \( \max_{1 \leq j \leq M^1} R_{b0}^j < 1 \), \( \min_{1 \leq k \leq K^j} R_{k0}^j > 1 \), and \( \max_{1 \leq k \leq K^j} R_{k0}^j < 1 \), system (33) has one positive equilibrium:

\[
E^* = \left( \frac{S_{fa}^*, S_{ab}^*, S_{ba}^*, S_{pa}^*, S_{pp}^*, \chi_{p0}^*, \chi_{p1}^*, \chi_{p2}^*, \chi_{p3}^*, \chi_{pm}^*, \chi_{ph}^*, \chi_{pmn}^*, \chi_{phm}}{\mu_{fa*M}^*, \mu_{pa*M}^*, \mu_{mp*M}^*, \mu_{pp*M}^*, \mu_{pm*M}^*, \mu_{ph*M}^*, \mu_{pmn*M}^*, \mu_{phm*M}^*} \right).
\]
situation, it can be calculated and analyzed by a similar method. Hence, we omit them here.

5. Numerical Simulations

In this section, we first use $L = 3$, $M = 2$, and $K = 3$ submodel to simulate. The course of the infected human is typically 1–4 weeks, and we assume that it is 2.5 weeks on average. Thus, the recovery rate of the infected human is $\beta_h = 1.6/month$. The disease-related death rate of the infected human is $a_h = 0.37$. The disease-induced death rates of poultry are assumed to be $\alpha_{t,p,m} = 4 \times 10^{-3}$ and $\alpha_p = 5 \times 10^{-4}$. We assume that human can survive 70 years, and the poultry can survive 2 months in the farm, 1 week in wholesale market, 1 month in wet market, and 8 months in backyard farm, respectively. These rates also referred to removal due to slaughter. Hence, these rates referred to removal due to slaughtering and the natural death. We take the parameter values as $d_t = 1.19 \times 10^{-1}/month$, $d_f = 0.8/month$, $d_p = d_m = 1/month$, and $d_h = 0.125/month$, respectively.

We estimate that the number of susceptible poultry population is between $10^2$ and $10^3$, the number of infective poultry population is between 0 and 1000 in farm, the number of susceptible poultry population is between 102 and 103, the number of infective poultry population is between 0 and 500 in live-poultry wholesale market, the number of susceptible poultry population is between 102 and 103, the number of infective poultry population is between 0 and 100 in wet market, and the number of susceptible human population is between $10^2$ and $10^3$ in the region. So, we choose the initial values as $(S_{la}(0), I_{la}(0), S_{lp}(0), I_{lp}(0), S_{lm}(0), I_{lm}(0), S_{lr}(0), I_{lr}(0)) = (5 \times 10^2, 1000, 4.9 \times 10^2, 900, 4.5 \times 10^2, 800), (S_{ma}(0), I_{ma}(0), S_{mp}(0), I_{mp}(0)) = (7 \times 10^4, 200, 5 \times 10^4, 100), (S_{mh}(0), I_{mh}(0), S_{mh}(0), I_{mh}(0)) = (10^3, 50, 10^3, 50), (S_{hr}(0), I_{hr}(0)) = (10^4, 100),$ and $(S_h(0), I_h(0), R_h(0)) = (10^2, 0, 0)$.

The difficulty in parameter estimations is that there is no scientifically or officially reported data of live-poultry transportation in China. The values of $a_{ij}, b_{jk}, l_j, j$, and $c_k$ used in simulations may be estimated based on living habits of people of regions, the density of human population, and so on. Now, we assume that the transport rates of the backyard poultry are the same to each node, namely, $l_j = 0.1$ and $c_k = 0.1, j = 1, 2, 3$. Let $a_{11} = 0.03, a_{12} = 0.04, a_{21} = 0.03, a_{22} = 0.04, a_{31} = 0.05, a_{32} = 0.02$ and $b_{11} = 0.03, b_{12} = 0.03, b_{21} = 0.04, b_{22} = 0.05, b_{31} = 0.02$, and $b_{32} = 0.03$. We assumed the replenishment rate to be 2 months which is the mean lifetime of farm poultry. Let $A_{k}^I = 2.5 \times 10^2, A_{k}^L = 2.45 \times 10^3, A_{t}^I = 2.25 \times 10^7, A_h = 833$, and $A_b = 1000$, respectively.

The transmission rates from the infective human in $k$th wet market to the susceptible human are $\beta_{h_{sk}} = 1.18 \times 10^{-9}$, $k = 1, 2, 3$. The transmission rate from the infective human in backyard farm to the susceptible human is $\beta_{h_{sb}} = 1.66 \times 10^{-8}$.

The transmission rates from infective poultry to susceptible poultry in different places are $\beta_{p_{la}} = 2.78 \times 10^{-8}, \beta_{p_{mb}} = 4.69 \times 10^{-8}, \beta_{p_{lm}} = 2.88 \times 10^{-8}$, and $\beta_{m_{lm}} = 1.88 \times 10^{-8}$, respectively. Then, $R_0 = 0.9784 < 1$. Solution $I_h(t)$ is asymptotically stable and converges to the disease-free equilibrium in Figure 4.

The transmission rates from infective poultry to susceptible poultry in different places are $\beta_{p_{la}} = 4.79 \times 10^{-4}, \beta_{p_{mb}} = 2.88 \times 10^{-8}$, and $\beta_{m_{lm}} = 1.88 \times 10^{-8}$, respectively. These parameters are fixed. $\beta_f$ is varied. Let $\beta_f = 3.18 \times 10^{-8}$, $\beta_f = 4.18 \times 10^{-8}$, and $\beta_f = 5.18 \times 10^{-8}$. From Figure 5(a), we can see that the beginning is almost the same, but the later is different. Therefore, the transmission rate $\beta_f$ has a small impact in the earliest stages but has an important impact on the late disease.

The transmission rates from infective poultry to susceptible poultry in different places are $\beta_{p_{la}} = 3.18 \times 10^{-8}, \beta_{p_{mb}} = 4.79 \times 10^{-4}, \beta_{p_{lm}} = 1.88 \times 10^{-8}$, and $\beta_{m_{lm}} = 1.88 \times 10^{-8}$, respectively. These parameters are fixed. $\beta_b$ is varied. Let $\beta_b = 4.79 \times 10^{-4}, \beta_b = 5.79 \times 10^{-4}$, and $\beta_b = 7.79 \times 10^{-4}$. This only affects the number of infected humans, whereas it has no effect on the arrival time of the peak (Figure 5(b)). Therefore, preventing poultry of backyard poultry farm into the live-poultry market is feasible in a suitable condition.

The transmission rates from infective poultry to susceptible poultry in different places are $\beta_{p_{la}} = 3.18 \times 10^{-8}, \beta_{p_{mb}} = 4.79 \times 10^{-4}, \beta_{p_{lm}} = 1.88 \times 10^{-8}$, and $\beta_{m_{lm}} = 1.88 \times 10^{-8}$, respectively. These parameters are fixed. $\beta_m$ is varied. Let $\beta_m = 2.88 \times 10^{-8}, \beta_m = 6.88 \times 10^{-8}$, and $\beta_m = 9.88 \times 10^{-8}$. The bigger the $\beta_m$ the more the human infected (Figure 5(c)). The secondary wholesale market plays an amplifier role.

The transmission rates from infective poultry to susceptible poultry in different places are $\beta_{p_{la}} = 3.18 \times 10^{-8}, \beta_{p_{mb}} = 4.79 \times 10^{-4}, \beta_{p_{lm}} = 1.88 \times 10^{-8}$, and $\beta_{m_{lm}} = 1.88 \times 10^{-8}$, respectively. These parameters are fixed. $\beta_m$ is varied. Let $\beta_m = 1.88 \times 10^{-8}, \beta_m = 6.88 \times 10^{-8}$, and $\beta_m = 9.88 \times 10^{-8}$. The impact is relatively small (Figure 5(d)).

6. Conclusion

In this paper, we construct the avian influenza transmission model from poultry (including poultry farm, backyard poultry farm, live-poultry wholesale market, and wet market) to human. We obtain the threshold value for the prevalence of avian influenza and the number of the boundary equilibria and endemic equilibria in different conditions. Numerical simulations show the effects of different transmission rates of different layer on the infected human. And, we can obtain the following cases:

(1) The poultry of poultry farm, backyard poultry farm, and poultry wholesale market have no avian influenza, but there is a possible outbreak of avian influenza in wet market (the retail live-poultry market), and avian influenza A (H7N9) virus is most likely transmitted from the retail live-poultry market to humans.

(2) The poultry of poultry farm and backyard poultry farm has no avian influenza, but there is a possible outbreak of avian influenza in poultry wholesale market, and then avian influenza A (H7N9) virus is...
Figure 4: Solution $I_h(t)$ is asymptotically stable and converges to the disease-free state value.

Figure 5: The plots display the changes of $I_h(t)$ with $\beta_{l,h,p,m}$ varying.
most likely transmitted from the poultry wholesale market to the retail live-poultry market and finally to humans.

(3) The poultry of poultry farm has avian influenza, and the poultry of backyard poultry farm has no avian influenza, but there is a possible outbreak of avian influenza in poultry farm, and then avian influenza A (H7N9) virus is most likely transmitted from poultry market to poultry wholesale market, then to the retail live-poultry market, and finally to humans.

(4) The poultry of poultry farm has no avian influenza, and the poultry of backyard poultry farm has avian influenza, but there is a possible outbreak of avian influenza in backyard poultry farm, and then avian influenza A (H7N9) virus is most likely transmitted from backyard poultry farm to poultry wholesale market, then to the retail live-poultry market, and finally to humans, or direct transmission from backyard poultry to humans.

(5) The poultry of poultry farm and backyard poultry farm has avian influenza, but there is a possible outbreak of avian influenza in poultry farm and backyard poultry farm, and then avian influenza A (H7N9) virus is most likely transmitted from poultry farm and backyard poultry farm to poultry wholesale market, then to the retail live-poultry market, and finally to humans, or direct transmission from backyard poultry to humans.

Hence, the poultry of some nodes on network has avian influenza, and then all edges connected to the node should be cut off. It has a great inhibitory on preventing the spread of disease. So, the network of poultry transportation plays an important role in controlling avian influenza A (H7N9). Moreover, we find that there may have been avian influenza A (H7N9) among humans when there is avian influenza A (H7N9) in the retail live-poultry market, so closing the live-poultry market can reduce the spread of disease to humans at a certain time. In addition, we find that there may have been avian influenza A (H7N9) among humans when there is avian influenza A (H7N9) in the backyard poultry farm. But the spread of backyard poultry to human is quit complex. It can be either direct infection or indirect infection. In China, there are many backyard poultry, so there are still some difficulties in the prevention and control of avian influenza A (H7N9).

Data Availability

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

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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