Research Article

Modeling Transmission Dynamics of *Streptococcus suis* with Stage Structure and Sensitivity Analysis

Chaojian Shen,^{1,2} Mingtao Li,³ Wei Zhang,¹ Ying Yi,⁴ Youming Wang,²
Qiang Hou,³ Baoxu Huang,² and Chengping Lu¹

¹ Key Lab of Animal Bacteriology of Ministry of Agriculture, College of Veterinary Medicine, Nanjing Agricultural University, Nanjing, Jiangsu 210095, China

² China Animal Health and Epidemiology Center, Qingdao, Shandong 266032, China

³ Department of Mathematics, North University of China, Taiyuan, Shan'xi 030051, China

⁴ Qingdao Municipal Center for Disease Control and Prevention, Qingdao, Shandong 266032, China

Correspondence should be addressed to Chengping Lu; lucp@njau.edu.cn

Received 7 December 2013; Revised 29 January 2014; Accepted 29 January 2014; Published 16 March 2014

Academic Editor: Delfim F. M. Torres

Copyright © 2014 Chaojian Shen et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Streptococcosis is one of the major infectious and contagious bacterial diseases for swine farm in southern China. The influence of various control measures on the outbreaks and transmission of *S. suis* is not currently known. In this study, in order to explore effective control and prevention measures we studied a deterministic dynamic model with stage structure for *S. suis*. The basic reproduction number $\mathcal{R}_0$ is identified and global dynamics are completely determined by $\mathcal{R}_0$. It shows that if $\mathcal{R}_0 < 1$, the disease-free equilibrium is globally stable and the disease dies out, whereas if $\mathcal{R}_0 > 1$, there is a unique endemic equilibrium which is globally stable and thus the disease persists in the population. The model simulations well agree with new clinical cases and the basic reproduction number of this model is about 1.1333. Some sensitivity analyses of $\mathcal{R}_0$ in terms of the model parameters are given. Our study demonstrates that combination of vaccination and disinfection of the environment are the useful control strategy for *S. suis*.

1. Introduction

Streptococcosis is a zoonosis caused by various pathogenic strains of *Streptococcus suis* (*S. suis*). The disease is primarily associated with *S. suis* capsular type 2 in pigs and humans, commonly manifested clinically as porcine septic streptococcosis and porcine lymphadenopathy with abscess formation [1]. The most common route of infection in pigs is through the respiratory tract, alimentary tract, and damaged skin. Pigs are the most important source of infection, as the pathogen is transmitted mainly via fomites, such as dust in the air or other insect vectors [2–7]. Porcine streptococcosis may cause death in animals and humans [8, 9]. Up to now, at least 832 human cases of porcine streptococcosis have been reported, with a mortality rate of approximately 10% [5, 10]. Despite a worldwide distribution, porcine streptococcosis is found primarily in Asia, with China and Southeast Asia most severely affected [11]. Porcine streptococcosis not only is

responsible for serious public health problems, but also poses severe economic losses in the swine industry.

Streptococcosis is widely distributed in China [12]; especially southern China is the most severely affected. In 1963, the disease began spreading in parts of Guangxi and then to most provinces and municipalities in southern China, including Guangdong, Sichuan, and Fujian. In the 1980s, the disease grew in severity, with many areas experiencing fulminating or endemic outbreaks. Since the 1990s, a rapid increase of pig production has led to an increasing number of reports of *S. suis* infection and outbreaks of the disease [13–17]. In southern China, *S. suis* infection has become one of the region's most important zoonoses.

Most streptococcosis studies are currently focused on the description of its etiology and epidemiology, while the studies on the mechanism of transmission and the risk factors influencing its spread have not been found. In this paper,

we constructed dynamical models to discuss the transmission dynamics of the *S. suis* and study the influence of two control measures, as well as the pathogenic *S. suis* shed into the environment from streptococcosis in a porcine herd in southern China.

The paper is organized as follows. In Section 2, we present and interpret the dynamical model that describes the *S. suis* transmission and give the basic reproduction number of the model and some mathematical analysis. And in Section 3, some numerical simulations are showed. Section 4 gives a brief discussion about main results.

2. Mathematical Modeling and Analysis

2.1. Model Formulation. A self-breeding and self-raising swine farm in southern China with good disease diagnosis often done by professional laboratories was employed for this study. There were no introductions of live pigs and the *S. suis* vaccine was not used on the farm. According to the epidemiological characteristics of *S. suis*, the population of this swine farm was divided into five subpopulations compartments: susceptible piglet compartment $S_j(t)$ (which is less than 2 months old), susceptible adult pig compartment $S_a(t)$ (including fattening pigs and sows), recessive infected compartment $I_r(t)$, recovered compartment $R(t)$, and quarantined clinical compartment $Q(t)$ at time t . Let $W(t)$ denote the quantity of the pathogenic bacteria in the environment at time t .

There are some assumptions on the dynamical transmission of *S. suis* in this swine farm. (1) The number of pigs supplemented (the number of newborn piglets) is considered to be approximately constant because the swine farm is a self-breeding and self-raising one. (2) Antibiotics are used not only for treatment in the pig raising process, but also for disease prevention. (3) The recessive infected and quarantined clinical cases have the same recovery rate after antibiotic treatment, and both recover to the recovered compartment. (4) The infection rate is different between adults and piglets, and the infection rate of adults is less than that of piglets. The dynamical transmission of *S. suis* in swine farm is demonstrated in the flowchart.

From Figure 1 we can know that the new infection occurring with piglet and adult pigs is given by $\beta S_j I_r + \beta_1 S_j W$ and $\sigma(\beta S_a I_r + \beta_1 S_a W)$, respectively, where β and β_1 describe the transmission rate from recessive infected pig and bacteria in the environment to the susceptible piglet. σ is the ratio of the transmission rate with susceptible adult pig to the transmission rate with susceptible junior pig. The recruitment rate of individuals into this swine farm is given by A ; within the piglet and adult pig group, the removed rates are μ_1 and μ . m represents the rate from susceptible junior pig to susceptible adult pig and l describes transfer rate from recessive infected pig to quarantined clinical pig. α denotes the removed rate for *S. suis* and r and γ represent the recovery rate from recessive infected compartment and quarantined clinical compartment to recovered compartment, respectively. In the environment compartment, the bacteria shedding rate from the recessive infected pig is k ,

FIGURE 1: Transmission diagram on the dynamical transmission of *S. suis*.

the decaying rate of bacteria is δ , the disinfection number of the environment is n , and the effective disinfection rate of every time is τ . From the previous assumptions, the model is a system of ordinary differential equations:

$$\begin{aligned} \frac{dS_j}{dt} &= A - (\mu_1 + m) S_j - (\beta S_j I_r + \beta_1 S_j W), \\ \frac{dS_a}{dt} &= m S_j - \sigma(\beta S_a I_r + \beta_1 S_a W) - \mu S_a, \\ \frac{dI_r}{dt} &= \sigma(\beta S_a I_r + \beta_1 S_a W) + \beta S_j I_r + \beta_1 S_j W - (r + l + \mu) I_r, \\ \frac{dQ}{dt} &= l I_r - (\mu + \alpha + \gamma) Q, \\ \frac{dR}{dt} &= r I_r + \gamma Q - \mu R, \\ \frac{dW}{dt} &= k I_r - (\delta + n\tau) W. \end{aligned} \quad (1)$$

All parameters are assumed to be nonnegative in system (1). System (1) always has a disease-free equilibrium $E_0 = (S_j^0, S_a^0, 0, 0, 0, 0)$, where

$$S_j^0 = \frac{A}{m + \mu_1}, \quad S_a^0 = \frac{mA}{\mu(m + \mu_1)}. \quad (2)$$

Lemma 1. Every forward solution $(S_j(t), S_a(t), I_r(t), R(t), Q(t), W(t))$ of system (1) eventually enters

$$\begin{aligned} \Gamma = & \left\{ (S_j, S_a, I_r, R, Q, W) \in \mathbb{R}_+^6 \mid \right. \\ & S_j, S_a, I_r, R, Q, W \geq 0, 0 \leq S_j \leq \frac{A}{m + \mu_1}, \\ & 0 \leq S_a + I_r + R + Q \leq \frac{mA}{\mu(m + \mu_1)}, \\ & \left. 0 \leq W \leq \frac{mA k}{\mu(m + \mu_1)(\delta + n\tau)} \right\}, \end{aligned} \quad (3)$$

and Γ is a positively invariant set for (1).

Proof. Let $N(t)$ be the total number of pigs. So from system (1) we can see that

$$\begin{aligned}\frac{dS_j}{dt} &= A_j - (\mu_1 + m)S_j - (\beta S_j I_r + \beta_1 S_j W) \\ &\leq A_j - (\mu_1 + m)S_j, \\ \frac{dN}{dt} &= mS_j(t) - \mu(S_a + I_r + R + Q) - \alpha Q \leq mS_j(t) - \mu N, \\ \frac{dW}{dt} &= kI_r - (\delta + n\tau)W.\end{aligned}\quad (4)$$

Thus, $dS_j/dt \leq 0$ if $S_j \geq A/(m + \mu_1)$, $dN/dt \leq 0$ if $N \geq mA/\mu(m + \mu_1)$, and $dW/dt \leq 0$ if $W \geq mA/\mu(m + \mu_1)(\delta + n\tau)$, which implies that Γ is positively invariant with respect to system (1). $\square$

Now we introduce the basic reproduction number $\mathcal{R}_0$ for system (1), according to the definition of [18]. We order the infected variables first by disease state; we only need the vector $x = (I_r, W)$ for system (1). Consider the following system:

$$\begin{aligned}\frac{dI_r}{dt} &= \sigma(\beta S_a I_r + \beta_1 S_a W) + \beta S_j I_r + \beta_1 S_j W \\ &\quad - (r + l + \mu)I_r, \\ \frac{dW}{dt} &= kI_r - (\delta + n\tau)W.\end{aligned}\quad (5)$$

Follow the recipe from van den Driessche and Watmough [18] to obtain

$$\begin{aligned}\mathcal{F} &= \begin{pmatrix} \sigma(\beta S_a I_r + \beta_1 S_a W) + \beta S_j I_r + \beta_1 S_j W \\ 0 \end{pmatrix}, \\ \mathcal{V} &= \begin{pmatrix} (r + l + \mu)I_r \\ -kI_r + (\delta + n\tau)W \end{pmatrix}.\end{aligned}\quad (6)$$

Calculate $\mathcal{F}$ and $\mathcal{V}$ for derivatives about $x = (I_r, W)$ and generate them into the disease-free equilibrium $E_0 = (S_j^0, S_a^0, 0, 0, 0, 0)$:

$$\begin{aligned}F &= \begin{pmatrix} \beta S_j^0 + \sigma\beta S_a^0 & \beta_1 S_j^0 + \sigma\beta_1 S_a^0 \\ 0 & 0 \end{pmatrix}, \\ V &= \begin{pmatrix} r + l + \mu & 0 \\ -k & n\tau + \delta \end{pmatrix}.\end{aligned}\quad (7)$$

So the basic reproduction number R_0 for system (1) is

$$\mathcal{R}_0 = \rho(FV^{-1}) = \frac{S_j^0 + \sigma S_a^0}{r + l + \mu} \left(\beta + \frac{\beta_1 k}{\delta + n\tau} \right). \quad (8)$$

The endemic equilibrium $E^* = (S_j^*, S_a^*, I_r^*, R^*, Q^*, W^*)$ of system (1) is determined by equations

$$\begin{aligned}A - (\mu_1 + m)S_j^* - (\beta S_j^* I_r^* + \beta_1 S_j^* W^*) &= 0, \\ mS_j^* - \sigma(\beta S_a^* I_r^* + \beta_1 S_a^* W^*) - \mu S_a^* &= 0, \\ \sigma(\beta S_a^* I_r^* + \beta_1 S_a^* W^*) \\ &\quad + \beta S_j^* I_r^* + \beta_1 S_j^* W^* - (r + l + \mu)I_r^* = 0, \\ lI_r^* - (\mu + \alpha + \gamma)Q^* &= 0, \\ rI_r^* + \gamma Q^* - \mu R^* &= 0, \\ kI_r^* - (\delta + n\tau)W^* &= 0.\end{aligned}\quad (9)$$

We can obtain

$$\begin{aligned}W^* &= \frac{k}{\delta + n\tau} I_r^*, \\ R^* &= \frac{l\gamma + r(\mu + \alpha + \gamma)}{\mu(\mu + \alpha + \gamma)} I_r^*, \\ Q^* &= \frac{l}{\mu + \alpha + \gamma} I_r^*, \\ S_j^* &= \frac{A}{m + \mu + (\beta + \beta_1 k/(\delta + n\tau))I_r^*}, \\ S_a^* &= \frac{mS_j^*}{\mu + (\beta + \beta_1 k/(\delta + n\tau))I_r^*}, \\ \sigma S_a^* + S_j^* &= \frac{r + l + \mu}{\beta + \beta_1 k/\delta} = \frac{\sigma S_a^0 + S_j^0}{\mathcal{R}_0}.\end{aligned}\quad (10)$$

Direct calculation for $0 < I_r^* < mA/\mu(m + \mu_1)$ shows

$$\frac{dS_j^*}{dI_r^*} < 0, \quad \frac{dS_a^*}{dI_r^*} < 0. \quad (11)$$

Now we define the following equation:

$$f(I_r^*) = \sigma S_a^*(I_r^*) + S_j^*(I_r^*) - \frac{\sigma S_a^0 + S_j^0}{\mathcal{R}_0}. \quad (12)$$

From system (11), we can find that the function $f(I_r^*)$ is also monotonically decreasing for $0 < I_r^* < mA/\mu(m + \mu_1)$. As the function $f(0) = \sigma S_a^0 + S_j^0 - (\sigma S_a^0 + S_j^0)/\mathcal{R}_0 > 0$ and $f(mA/\mu(m + \mu_1)) < 0$ when $\mathcal{R}_0 > 1$, system (1) has a unique endemic equilibrium $E^* = (S_j^*, S_a^*, I_r^*, R^*, Q^*, W^*)$.

2.2. Global Stability of the Equilibrium. Because the fourth and fifth equations are independent of other equations for system (1), we only need to consider the following system:

$$\begin{aligned}\frac{dS_j}{dt} &= A - (\mu_1 + m)S_j - (\beta S_j I_r + \beta_1 S_j W), \\ \frac{dS_a}{dt} &= mS_j - \sigma(\beta S_a I_r + \beta_1 S_a W) - \mu S_a, \\ \frac{dI_r}{dt} &= \sigma(\beta S_a I_r + \beta_1 S_a W) + \beta S_j I_r + \beta_1 S_j W - (r + l + \mu)I_r, \\ \frac{dW}{dt} &= kI_r - (\delta + n\tau)W.\end{aligned}\quad (13)$$

From previous analysis, we know that system (13) also has a disease-free equilibrium $P_0 = (S_j^0, S_a^0, 0, 0)$, $S_j^0 = A/(\mu + \mu_1)$, and $S_a^0 = mA/(\mu(\mu + \mu_1))$ when $\mathcal{R}_0 < 1$ and a unique endemic equilibrium $P^* = (S_j^*, S_a^*, I_r^*, W^*)$ when $\mathcal{R}_0 > 1$. In the following, we will prove the global stability of the disease-free equilibrium and endemic equilibrium of system (13) by using a Lyapunov function. The Lyapunov function is a powerful tool for the stability analysis of autonomous differential system; it has been used for some epidemiological models with constant inflow and bilinear incidences or nonlinear incidences [19–23].

For the disease-free equilibrium, we have the following conclusion.

Theorem 2. *The disease-free equilibrium P_0 of system (13) is globally asymptotically stable when $\mathcal{R}_0 < 1$.*

Proof. For the disease-free equilibrium P_0 , define the Lyapunov function

$$\begin{aligned}L_1 &= S_j - S_j^0 - S_j^0 \ln \frac{S_j}{S_j^0} + S_a - S_a^0 \\ &\quad - S_a^0 \ln \frac{S_a}{S_a^0} + I_r + \frac{\beta_1}{\delta + n\tau} (\sigma S_a^0 + S_j^0) W.\end{aligned}\quad (14)$$

Then the derivative of L_1 along solutions of system (13) is

$$\begin{aligned}\frac{dL_1}{dt} &= \left(1 - \frac{S_j^0}{S_j}\right) S_j' + \left(1 - \frac{S_a^0}{S_a}\right) S_a' + I_r' + \frac{\beta_1}{\delta + n\tau} (\sigma S_a^0 + S_j^0) W' \\ &= \left(1 - \frac{S_j^0}{S_j}\right) (A - (\mu_1 + m)S_j - (\beta S_j I_r + \beta_1 S_j W)) \\ &\quad + \left(1 - \frac{S_a^0}{S_a}\right) (mS_j - \mu S_a - \sigma(\beta S_a I_r + \beta_1 S_a W)) \\ &\quad + \sigma(\beta S_a I_r + \beta_1 S_a W) + \beta S_j I_r + \beta_1 S_j W - (r + l + \mu)I_r \\ &\quad + \frac{\beta_1}{\delta + n\tau} (\sigma S_a^0 + S_j^0) (kI_r - (\delta + n\tau)W)\end{aligned}$$

$$\begin{aligned}&= \left(1 - \frac{S_j^0}{S_j}\right) (A - (\mu_1 + m)S_j) + \left(1 - \frac{S_a^0}{S_a}\right) (mS_j - \mu S_a) \\ &\quad + \beta S_j^0 I_r + \sigma \beta S_a^0 I_r + \frac{\beta_1}{\delta + n\tau} (\sigma S_a^0 + S_j^0) kI_r - (r + l + \mu)I_r \\ &= (\mu_1 + m)S_j^* \left(2 - \frac{S_j}{S_j^0} - \frac{S_j^0}{S_j}\right) + mS_j^0 \left(1 + \frac{S_j}{S_j^0} - \frac{S_a}{S_a^0} - \frac{S_a^0 S_j}{S_a S_j^0}\right) \\ &\quad + (\sigma S_a^0 + S_j^0) \left(\beta + \frac{\beta_1 k}{\delta + n\tau}\right) I_r - (r + l + \mu)I_r \\ &= \mu_1 S_j^0 \left(2 - \frac{S_j}{S_j^0} - \frac{S_j^0}{S_j}\right) + mS_j^0 \left(3 - \frac{S_j^0}{S_j} - \frac{S_a}{S_a^0} - \frac{S_a^0 S_j}{S_a S_j^0}\right) \\ &\quad + (r + l + \mu)(\mathcal{R}_0 - 1)I_r.\end{aligned}\quad (15)$$

Therefore, when $\mathcal{R}_0 < 1$, $dL_1/dt < 0$ and the equality $dL_1/dt = 0$ holds if and only if $S_j = S_j^0$, $S_a = S_a^0$, and $I_r = 0$. Thus, the disease-free equilibrium P_0 of system (13) is globally asymptotically stable by LaSalle's Invariance Principle [24]. This completes the proof. $\square$

Next, we will also prove global stability of the endemic equilibrium of system (13) by using a Lyapunov function. It is important for us to understand the extinction and persistence of the disease.

Theorem 3. *The unique endemic equilibrium P^* of system (13) is globally asymptotically stable when $\mathcal{R}_0 > 1$.*

Proof. System (1) can be transformed into the following form:

$$\begin{aligned}\frac{dS_j}{dt} &= S_j \left(\frac{A}{S_j^*} \left(\frac{S_j^*}{S_j} - 1 \right) - \left(\beta I_r^* \left(\frac{I_r}{I_r^*} - 1 \right) + \beta_1 W^* \left(\frac{W}{W^*} - 1 \right) \right) \right), \\ \frac{dS_a}{dt} &= S_a \left(\frac{mS_j^*}{S_a^*} \left(\frac{S_a^* S_j}{S_a S_j^*} - 1 \right) - \sigma \beta I_r^* \left(\frac{I_r}{I_r^*} - 1 \right) - \sigma \beta_1 W^* \left(\frac{W}{W^*} - 1 \right) \right), \\ \frac{dI_r}{dt} &= I_r \left(\sigma \beta S_a^* \left(\frac{S_a}{S_a^*} - 1 \right) + \beta S_j^* \left(\frac{S_j}{S_j^*} - 1 \right) + \sigma \frac{\beta_1 S_a^* W^*}{I_r^*} \right. \\ &\quad \times \left. \left(\frac{S_a W I_r^*}{S_a^* W^* I_r} - 1 \right) + \frac{\beta_1 S_j^* W^*}{I_r^*} \left(\frac{S_j W I_r^*}{S_j^* W^* I_r} - 1 \right) \right), \\ \frac{dW}{dt} &= \frac{kW I_r^*}{W^*} \left(\frac{I_r W^*}{I_r^* W} - 1 \right).\end{aligned}\quad (16)$$

Define the Lyapunov function

$$L_2 = S_j - S_j^* - S_j^* \ln \frac{S_j}{S_j^*} + S_a - S_a^* - S_a^* \ln \frac{S_a}{S_a^*} + I_r - I_r^* - I_r^* \ln \frac{I_r}{I_r^*} + \frac{\beta_1 W^*}{k I_r^*} (\sigma S_a^* + S_j^*) \left(W - W^* - W^* \ln \frac{W}{W^*} \right). \quad (17)$$

Then the derivative of L along solutions of system (16) is

$$\begin{aligned} \frac{dL_2}{dt} &= \left(1 - \frac{S_j^*}{S_j}\right) S_j' + \left(1 - \frac{S_a^*}{S_a}\right) S_a' + \left(1 - \frac{I_r^*}{I_r}\right) I_r' \\ &\quad + \frac{\beta_1 W^*}{k I_r^*} (\sigma S_a^* + S_j^*) \left(1 - \frac{W^*}{W}\right) W' \\ &= (S_j - S_j^*) \\ &\quad \times \left(\frac{A}{S_j^*} \left(\frac{S_j^*}{S_j} - 1 \right) - \left(\beta I_r^* \left(\frac{I_r}{I_r^*} - 1 \right) + \beta_1 W^* \left(\frac{W}{W^*} - 1 \right) \right) \right) \\ &\quad + (S_a - S_a^*) \left(\frac{m S_j^*}{S_a^*} \left(\frac{S_a^* S_j}{S_a S_j^*} - 1 \right) - \sigma \beta I_r^* \left(\frac{I_r}{I_r^*} - 1 \right) - \sigma \beta_1 W^* \left(\frac{W}{W^*} - 1 \right) \right) \\ &\quad + (I_r - I_r^*) \left(\sigma \beta S_a^* \left(\frac{S_a}{S_a^*} - 1 \right) + \beta S_j^* \left(\frac{S_j}{S_j^*} - 1 \right) + \sigma \frac{\beta_1 S_a^* W^*}{I_r^*} \left(\frac{S_a W I_r^*}{S_a^* W^* I_r} - 1 \right) + \frac{\beta_1 S_j^* W^*}{I_r^*} \left(\frac{S_j W I_r^*}{S_j^* W^* I_r} - 1 \right) \right) \\ &\quad + \beta_1 (\sigma S_a^* + S_j^*) (W - W^*) \left(\frac{I_r W^*}{I_r^* W} - 1 \right) = B + C, \end{aligned} \quad (18)$$

where

$$\begin{aligned} B &= A (S_j - S_j^*) \left(\frac{1}{S_j} - \frac{1}{S_j^*} \right) + m (S_a - S_a^*) \left(\frac{S_j}{S_a} - \frac{S_j^*}{S_a^*} \right) \\ &= A \left(2 - \frac{S_j^*}{S_j} - \frac{S_j}{S_j^*} \right) + m S_j^* \left(1 + \frac{S_j}{S_j^*} - \frac{S_a}{S_a^*} - \frac{S_j S_a^*}{S_j^* S_a} \right), \end{aligned}$$

$$\begin{aligned} C &= -\beta_1 (S_j - S_j^*) (W - W^*) + \beta_1 (I_r - I_r^*) \\ &\quad \times \left(\frac{S_j W}{I_r} - \frac{S_j^* W^*}{I_r^*} \right) - \sigma \beta_1 (S_a - S_a^*) (W - W^*) \\ &\quad + \sigma \beta_1 (I_r - I_r^*) \left(\frac{S_a W}{I_r} - \frac{S_a^* W^*}{I_r^*} \right) + \frac{\beta_1 W^*}{I_r^*} \\ &\quad \times (\sigma S_a^* + S_j^*) (W - W^*) \left(\frac{I}{W} - \frac{I_r^*}{W^*} \right) \\ &= \sigma \beta_1 S_a^* W^* \left(1 + \frac{S_a}{S_a^*} - \frac{I_r W^*}{I_r^* W} - \frac{S_a I_r^* W}{S_a^* I_r W^*} \right) \\ &\quad + \beta_1 S_j^* W^* \left(1 + \frac{S_j}{S_j^*} - \frac{I_r W^*}{I_r^* W} - \frac{S_j I_r^* W}{S_j^* I_r W^*} \right). \end{aligned} \quad (19)$$

For the endemic equilibrium P^* , we have the following equation:

$$\begin{aligned} A &= \mu_1 S_j^* + \mu S_a^* + \beta S_j^* I_r^* + \beta_1 S_j^* W^* \\ &\quad + \sigma (\beta S_a^* I_r^* + \beta_1 S_a^* W^*), \\ m S_j^* &= \sigma (\beta S_a^* I_r^* + \beta_1 S_a^* W^*) + \mu S_a^*. \end{aligned} \quad (20)$$

Hence, we can obtain

$$\begin{aligned} \frac{dL_2}{dt} &= B + C \\ &= (\mu S_a^* + \sigma \beta S_a^* I_r^*) \left(3 - \frac{S_a}{S_a^*} - \frac{S_j^*}{S_j} - \frac{S_j S_a^*}{S_j^* S_a} \right) \\ &\quad + (\mu_1 S_j^* + \beta S_j^* I_r^*) \left(2 - \frac{S_j^*}{S_j} - \frac{S_j}{S_j^*} \right) \\ &\quad + \beta_1 S_j^* W^* \left(3 - \frac{S_j^*}{S_j} - \frac{I_r W^*}{I_r^* W} - \frac{S_j I_r^* W}{S_j^* I_r W^*} \right) \\ &\quad + \sigma \beta_1 S_a^* W^* \left(4 - \frac{S_j^*}{S_j} - \frac{S_j S_a^*}{S_j^* S_a} - \frac{I W^*}{I^* W} - \frac{S_a I^* W}{S_a^* I W^*} \right) \\ &\leq 0. \end{aligned} \quad (21)$$

The equality $dL_2/dt = 0$ holds only for $S_j = S_j^*$, $S_a = S_a^*$, and $I_r/I_r^* = W/W^*$. By LaSalle's Invariance Principle [24], the endemic equilibrium P^* is globally asymptotically stable. This completes the proof. $\square$

So far all our analyses focus on the mathematical models and their dynamic behavior, such as the basic reproduction number and the global stability of the disease-free equilibrium and endemic equilibrium. In the next section we will present some numerical simulations about the actual data and give some sensitivity analyses of the basic production number $\mathcal{R}_0$ on parameters.

TABLE 1: Swine data and incidence of *S. suis* for the past three years from a swine farm in southern China.

Year	Quarter	Inventory	New birth	Sales and death	<i>S. suis</i> cases	Removed for <i>S. suis</i>	Disinfection times
2011	1	4057	3196	2873 + 197	43	16 + 27 = 43	13
	2	4093	3210	2885 + 196	37	15 + 22 = 37	13
	3	4122	3217	2892 + 193	41	16 + 25 = 41	13
	4	4087	3230	2905 + 198	39	13 + 26 = 39	13
2012	1	4193	3245	2917 + 186	42	14 + 28 = 42	13
	2	4186	3267	2930 + 189	47	17 + 30 = 47	13
	3	4170	3328	2954 + 199	51	19 + 32 = 51	13
	4	4213	3317	2978 + 202	46	17 + 29 = 46	13
2013	1	4239	3400	2993 + 214	55	20 + 35 = 55	13
	2	4317	3337	3004 + 210	57	18 + 39 = 57	13

3. Numerical Simulations and Sensitivity Analysis

The number of new births, sales, and deaths for live pigs, inventory at the beginning of the quarter, quarantined clinical cases, and deaths from *S. suis* and disinfection numbers from the past three years are recorded in Table 1.

3.1. Parameter Estimation. In order to carry out the numerical simulations, we need to estimate the model parameters. Some parameter values can be calculated by using the data in Table 1, some parameter values need to be assumed, and other parameter values need the parameter estimation. For the recruitment rate of individuals into this swine farm A , which is the average of new birth, the value is $A \approx (3196 + 3210 + 3217 + 3230 + 3245 + 3267 + 3328 + 3317 + 3400 + 3337)/10 \approx 3275$. For the transfer rate from susceptible piglet to susceptible adult pig m , we know that susceptible piglet can survive in piglet compartment for about two months, and the unit time of this model is a quarter, so the transfer rate is $m = 3/2 = 1.5$. With the removed rate of this swine farm, we assume the removed rates of susceptible piglet compartment and susceptible adult pig compartment are the same, which is the average of the proportion for sales and death to the breeding stock of this swine farm, which is $\mu_1 = \mu \approx 0.75$. For the removed rate for quarantined clinical *S. suis*, we can calculate $\alpha = 1$, so the recovered rate from quarantined clinical compartment to recovered compartment is $\gamma = 0$. Due to the fact that antibiotics are used for treatment in the pig raising process and disease prevention and we do not know the transfer rate from recessive infected compartment to recovered compartment, we assume that $r = 0.75$ and $l = 0.25$. We can obtain the disinfection time of the environment from the data in Table 1, which is $n = 13$. With the effective disinfection rate of every time τ , we assume $\tau = 0.6$. According to the survival of *S. suis* in the environment mentioned previously, combined with the local climate characteristics, and assuming that the survival time of *S. suis* is 10 days under natural conditions, we obtain $\delta = 9$. For other parameters σ , β , β_1 , and k , we need some parameter estimation. Hence, the parameter values are listed in Table 2.

For the initial values, we also need the fitting data. $Q(0) = 43$ can be directly obtained, but $I_r(0) = 130$ and $R(0) = 80$

TABLE 2: Parameter values of system (1) (Unit Quarter⁻¹).

Parameter	Value	Source
A	3275	Calculated
μ_1	0.75	Calculated
μ	0.75	Calculated
m	1.5	Calculated
r	0.75	Assumed
l	0.25	Assumed
γ	0	Calculated
α	1	Calculated
k	30	Fitting
δ	9	Assumed
n	13	Calculated
τ	0.6	Assumed
σ	0.4	Fitting
β	3.65×10^{-4}	Fitting
β_1	2.195×10^{-4}	Fitting

are assumed. Since the rate of piglet births is assumed to be continuous, we assume that the initial value of S_j is the final size with the disease-free state. So S_j satisfies the following equation: $dS_j/dt = A - (m + \mu_1)S_j$, with its final size given by $S_j(\infty) = A/(m + \mu_1) \approx 1450$; therefore $S_j(0) \approx 1450$. Hence, we can also obtain $S_a(0) \approx 4057 - S_j(0) - I_r(0) - Q(0) - R(0) \approx 2350$. For the initial value of $W(t)$, we use the data fitting to obtain $W(0) = 750$.

3.2. Numerical Simulations. Using system (1), we simulate newly quarantined clinical cases in this swine farm from the first quarter of 2011 to the second quarter of 2013. The numerical simulation of newly quarantined clinical cases is shown in Figure 2(a), and Figure 2(b) shows the cumulative quarantined clinical cases, which indicate that our model provides a good match to the reported data. According to the predictions of the model (Figure 2(a)), if no further effective prevention and control measures are taken, the disease will not vanish and the clinical cases of streptococcosis will increase during the period after 2013. With the simulated parameter values, we obtain that the estimated value for

FIGURE 2: (a) The comparison between the reported clinical cases and the simulation of new clinical cases from system (1). (b) *S. suis* model fitting for the cumulative clinical cases.

the basic reproduction number of this swine farm is $\mathcal{R}_0 \approx 1.1333$, which predicts that *S. suis* will persist in this farm.

In an epidemic model, the basic reproduction number $\mathcal{R}_0$ is calculated and shown to be a threshold for the dynamics of the disease. The main purpose is to control the disease by making the basic reproduction number $\mathcal{R}_0$ less than 1, so we must know how the basic reproduction number depends on the model parameter values. In the following result, we will show some sensitivity analyses of the basic production number $\mathcal{R}_0$.

As shown in Figure 3(a), in order to make $\mathcal{R}_0 < 1$, with all other parameters held constant, when the disinfection times reach 18, the basic reproduction number is still larger than 1. In the real world, it is very difficult to disinfect this whole farm 18 times per quarter for costs. Figure 3(b) shows that the effective disinfection rate must reach 1 which can make $\mathcal{R}_0 < 1$, when other parameters remain constant. Figures 3(c) and 3(d) illustrate that $\mathcal{R}_0$ increases with the proportion parameter σ and the recruitment rate A , when $A < 2900$ or $\sigma < 0.3$, which can make $\mathcal{R}_0 < 1$. However, in practice it is also very difficult to reach these two measures.

Finally, the influence of different parameters on the incidence of disease was studied based on the sensitivity analysis of the basic reproduction number as mentioned above.

From the two figures of Figure 4, we can find that it is very difficult to control the disease only by changing parameters n and τ , because the final size of the new clinical cases is larger than 25 in 2019. In order to effectively control the disease, it is necessary to use other control measures for this swine farm. So in the next section, we will give the dynamic model with vaccination for *S. suis*.

3.3. The Analysis of the Dynamic Model with Vaccination. Since disinfection cannot effectively control the disease, it

is necessary to use other measures to effectively control the disease, such as vaccination. In the real world, breeding pigs and fattening pigs are vaccinated. So we assume that vaccinated pigs are susceptible adult pigs and their removed rate is the same as the adult susceptible ones. For the susceptible adult pig, v is the vaccination rate and λ is the losing vaccination rate. Therefore, system (1) can be transformed into the following system after the vaccination measure is applied:

$$\begin{aligned}
 \frac{dS_j}{dt} &= A - (\mu_1 + m)S_j - (\beta S_j I_r + \beta_1 S_j W), \\
 \frac{dS_a}{dt} &= mS_j - \sigma(\beta S_a I_r + \beta_1 S_a W) - (\mu + v)S_a + \lambda V, \\
 \frac{dI_r}{dt} &= \sigma(\beta S_a I_r + \beta_1 S_a W) + \beta S_j I_r + \beta_1 S_j W - (r + l + \mu)I_r, \\
 \frac{dQ}{dt} &= lI_r - (\mu + \alpha + \gamma)Q, \\
 \frac{dR}{dt} &= rI_r + \gamma Q - \mu R, \\
 \frac{dW}{dt} &= kI_r - (\delta + n\tau)W, \\
 \frac{dV}{dt} &= vS_a - (\lambda + \mu)V.
 \end{aligned} \tag{22}$$

Define the basic reproduction number of system (22) by [18]. We have

$$\mathcal{R}_0^v = \rho(FV^{-1}) = \frac{S_j^1 + \sigma S_a^1}{r + l + \mu} \left(\beta + \frac{\beta_1 k}{\delta + n\tau} \right), \tag{23}$$

FIGURE 3: (a) $\mathcal{R}_0$ in terms of n . (b) $\mathcal{R}_0$ in terms of τ . (c) $\mathcal{R}_0$ in terms of σ . (d) $\mathcal{R}_0$ in terms of A . Other parameters are the same as in Figure 2.

where

$$S_j^1 = \frac{A}{m + \mu_1}, \quad S_a^1 = \frac{mA}{\mu(m + \mu_1)} \times \frac{\lambda + \mu}{\lambda + \mu + \nu}. \quad (24)$$

Generally, the immune validity duration is about 4-5 months, taking the averaging 4.5 months, so we can calculate the losing vaccination rate to be $\lambda = 3/4.5 = 0.75$. In the following result, we will show sensitivity analysis of the basic production number $\mathcal{R}_0^v$ of system (22) and give the variations of new clinical cases of system (22) for different values of the vaccination rate ν since the second quarter of 2013.

Figure 5(a) shows that increasing ν will decrease the basic reproduction number $\mathcal{R}_0^v$. So as to make the basic reproduction number of system (22) less than 1, the vaccination rate of the susceptible adult pig only reaches about $\nu = 0.6$. The second figure of Figure 5 shows the variations of new clinical cases of system (22) for different values of the vaccination rate ν since the second quarter of 2013; we know that when the vaccination rate reaches $\nu = 0.6$, the final size of the

new clinical cases is less than 10 in the future; it means that *S. suis* infection of this farm can be effectively controlled within 3-4 years. Hence, vaccination is a very important factor for the *S. suis* transmission; it plays an important role in the persistence of *S. suis* in this swine farm. Compared with Figures 4(b) and 5(b), we conclude that combination of vaccination and disinfection for *S. suis* are more effective than only the disinfection for the environment.

4. Conclusion and Discussion

In southern China, *S. suis* has not only become a serious public health problem, but also caused severe economic losses in the swine production. So both the government and the swine industry have been seeking forceful methods to reduce the outbreaks and the spread of *S. suis*. However, no people have studied the transmission mechanism of this disease, and there are also no given effective control measures for the disease. In this paper, we established a deterministic dynamic

FIGURE 4: The variations of new clinical cases of system (1) for different values of n and τ since the second quarter of 2013. (a) New clinical cases with different values of τ with system (1). (b) New clinical cases with different values of n with system (1).

FIGURE 5: (a) $\mathcal{R}_0^\nu$ in terms of ν . (b) New clinical cases with different values of ν with system (22). Other parameter values are showed in Table 2.

transmission model with stage structure for *S. suis*. It is found that the model has two nonnegative equilibria, the disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium exists without any condition whereas the endemic equilibrium exists provided $\mathcal{R}_0 > 1$. Through the analysis of the model it has been found that the global asymptotic behavior of system (1) is completely determined by the size of the basic reproduction number $\mathcal{R}_0$; that is, the disease-free equilibrium is globally asymptotically stable if

$\mathcal{R}_0 < 1$ while an endemic equilibrium exists uniquely and is globally asymptotically stable if $\mathcal{R}_0 > 1$.

The model simulations (see Figure 2(a)) agreed with the new clinical cases, and we gave an estimate of the basic reproduction number $\mathcal{R}_0$ to be about 1.1333, which implies that *S. suis* of this farm cannot be controlled with the current strategies. The trend of future *S. suis* incidence on the farm was predicted using system (1) and it was found that if the current control measures remained unchanged, the number

of *S. suis* cases would increase. By some sensitivity analyses of the basic reproduction number $\mathcal{R}_0$ on parameters, we find that disinfection is the effective control measure with *S. suis*, but it can not eradicate it for this farm. From Section 3.3 we know that vaccination is a very important factor for *S. suis*; it plays an important role in the persistence of *S. suis*. Hence, we conclude that combination of disinfection and vaccination control measures will become more reasonable and effective for *S. suis* in this swine farm.

However, there are some limitations and shortcomings in our model. Firstly, while *S. suis* in the environment as defined in the study is a quantity value, it is only an assumption, without actual laboratory data. Secondly, for the lack of the cost of vaccination and elimination for *S. suis*, we can only give some macrocontrol measures for *S. suis* of this farm; specific control measures cannot be given and the optimal control strategy cannot also be taken into account. So we need to continue research in the future.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Acknowledgment

This project was funded by the National Key Technologies R D Program of China (2004BA519A60, 2006BAK02A03-1).

References

- [1] M. Gottschalk, R. Higgins, and S. Quessy, "Dilemma of the virulence of *Streptococcus suis* strains," *Journal of Clinical Microbiology*, vol. 37, no. 12, pp. 4202–4203, 1999.
- [2] F. Berthelot-Hérault, M. Gottschalk, A. Labbé, R. Cariolet, and M. Kobisch, "Experimental airborne transmission of *Streptococcus suis* capsular type 2 in pigs," *Veterinary Microbiology*, vol. 82, no. 1, pp. 69–80, 2001.
- [3] I. D. Robertson, D. K. Blackmore, D. J. Hampson, and Z. F. Fu, "A longitudinal study of natural infection of piglets with *Streptococcus suis* types 1 and 2," *Epidemiology & Infection*, vol. 107, no. 1, pp. 119–126, 1991.
- [4] S. A. Dee, A. R. Carlson, N. L. Winkelman, and M. M. Corey, "Effect of management practices on the *Streptococcus suis* carrier rate in nursery swine," *Journal of the American Veterinary Medical Association*, vol. 203, no. 2, pp. 295–299, 1993.
- [5] Z.-R. Lun, Q.-P. Wang, X.-G. Chen, A.-X. Li, and X.-Q. Zhu, "*Streptococcus suis*: an emerging zoonotic pathogen," *The Lancet Infectious Diseases*, vol. 7, no. 3, pp. 201–209, 2007.
- [6] J. J. Staats, I. Feder, O. Okwumabua, and M. M. Chengappa, "*Streptococcus suis*: past and present," *Veterinary Research Communications*, vol. 21, no. 6, pp. 381–407, 1997.
- [7] A. T. R. Costa, F. C. F. Lobato, V. L. V. Abreu, R. A. Assis, R. Reis, and F. A. Uzal, "Serotyping and evaluation of the virulence in mice of *Streptococcus suis* strains isolated from diseased pigs," *Revista do Instituto de Medicina Tropical de Sao Paulo*, vol. 47, no. 2, pp. 113–115, 2005.
- [8] A. C. F. Hui, K. C. Ng, P. Y. Tong et al., "Bacterial meningitis in Hong Kong: 10-Years' experience," *Clinical Neurology and Neurosurgery*, vol. 107, no. 5, pp. 366–370, 2005.
- [9] R. Kay, A. F. Cheng, and C. Y. Tse, "*Streptococcus suis* infection in Hong Kong," *Quarterly Journal of Medicine*, vol. 88, no. 1, pp. 39–47, 1995.
- [10] H. F. L. Wertheim, H. D. T. Nghia, W. Taylor, and C. Schultz, "*Streptococcus suis*: an emerging human pathogen," *Clinical Infectious Diseases*, vol. 48, no. 5, pp. 617–625, 2009.
- [11] S. D. E. Geffner, M. R. Moreno, A. M. S. Campillo, S. F. J. Pardo, G. A. Gómez, and M. D. Martínez-Lozano, "*Streptococcus suis* meningitis," *Anales de Medicina Interna*, vol. 18, no. 6, pp. 317–318, 2001.
- [12] C.-P. Zhang, Y.-B. Ning, Z.-Q. Zhang et al., "Prevalence of *Streptococcus suis* isolated from clinically healthy sows in China," *Agricultural Sciences in China*, vol. 8, no. 5, pp. 638–642, 2009.
- [13] K. C. Wang, Z.-I. Xiong, Y.-M. Shang et al., "Detection of the *Streptococcus suis* of clinical healthy pigs in Chongqing," *Chinese Journal of Animal and Veterinary Sciences*, vol. 41, pp. 594–599, 2010.
- [14] L. Luo, X. Wang, Z.-G. Cui et al., "Isolation of *Streptococcus suis* type 2 from healthy pigs in Ziyang district of Sichuan province and analysis of their molecular characteristics," *Chinese Journal of Zoonoses*, vol. 25, no. 9, pp. 842–845, 2009.
- [15] C. L. Li, W.-L. Yu, A.-G. Jia et al., "Carrier rate of *Streptococcus suis* in palatine tonsils of slaughter pigs in south China," *Chinese Journal of Preventive Veterinary Medicine*, vol. 30, pp. 343–348, 2008.
- [16] Y. Xiong, F.-Y. Qin, Y. Bai et al., "Identification of type 2 of swine *Streptococcus suis* from piglets by PCR in Guangxi," *Guangxi Agricultural Sciences*, vol. 37, pp. 449–451, 2006.
- [17] L. X. Lu, K.-W. He, Y.-X. Ni et al., "Isolation and identification of virulent *Streptococcus suis* type 2 from tonsillar specimens of slaughtered healthy pigs," *Chinese Journal of Zoonoses*, vol. 24, no. 4, pp. 379–383, 2008.
- [18] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, no. 1-2, pp. 29–48, 2002.
- [19] M. T. Li, G. Q. Sun, J. Zhang, and Z. Jin, "Global dynamic behavior of a multi-group cholera model with indirect transmission," *Discrete Dynamics in Nature and Society*, vol. 2013, Article ID 703826, 11 pages, 2013.
- [20] J. Li, Y. Yang, and Y. Zhou, "Global stability of an epidemic model with latent stage and vaccination," *Nonlinear Analysis: Real World Applications*, vol. 12, no. 4, pp. 2163–2173, 2011.
- [21] H. Guo, M. Y. Li, and Z. Shuai, "Global stability of the endemic equilibrium of multigroup SIR epidemic models," *Canadian Applied Mathematics Quarterly*, vol. 14, no. 3, pp. 259–284, 2006.
- [22] H. Guo, M. Y. Li, and Z. Shuai, "A graph-theoretic approach to the method of global Lyapunov functions," *Proceedings of the American Mathematical Society*, vol. 136, no. 8, pp. 2793–2802, 2008.
- [23] R. Sun and J. Shi, "Global stability of multigroup epidemic model with group mixing and nonlinear incidence rates," *Applied Mathematics and Computation*, vol. 218, no. 2, pp. 280–286, 2011.
- [24] J. P. LaSalle, *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, Pa, USA, 1976.