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MODELING PORCINE PSEUDORABIES WITH AGE STRUCTURE

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Abstract. Porcine pseudorabies is an acute and highly contagious viral disease caused by the pseudorabies virus. It inflicts enormous losses to the pig-breeding industry. In this paper, we propose an age-structured mathematical model. We investigate the dynamics of this model characterized by the basic reproduction number $\Re_0 = \max\{\Re_{01}, \Re_{02}\}$ by addressing the existence and global stability of equilibria. When $\Re_0 < 1$, the disease-free equilibrium is unique and globally asymptotically stable. The boundary equilibrium exists and is globally asymptotically stable under the condition $\Re_{01} < 1$ and $\Re_{02} > 1$ or $\Re_{01} > 1$ and $\Re_{02} < 1 + \epsilon$. If both $\Re_{01} > 1$ and $\Re_{02} > 1 + \epsilon$, there is a unique disease-endemic equilibrium which is globally asymptotically stable.

1. Introduction

Pseudorabies, known as Aujeszky disease (AD), is an acute and highly contagious viral disease caused by the pseudorabies virus (PRV), and inflicts high economic losses in the swine industry globally [21, 26]. PRV, a double-stranded DNA virus, has a wide range of infection and strong pathogenicity, and belongs to the natural focus disease, which can be found all over the world [14, 30, 32]. Besides pigs, primary hosts and reservoirs of PRV [22], PRV has a broad spectrum of hosts, such as dogs, sheep, cattle, bears, cats, bats, and some avian species [24]. Moreover, since an early study describing PRV's ready infection in human cells proved the PRV's zoonotic threat theoretically [27], mounting evidence has indicated that PRV has public health significance [15]. Thus, it is necessary to further study for understanding PRV's potential threat to public health security.

According to records, pseudorabies was first discovered in the United States in 1813, which was characterized by a "mad itching disease" of cattle. The sick cattle were extremely itchy and finally died [10]. At that time, the disease was mistakenly regarded as the same disease as rabies, and it was not called Aujeszky's disease until 1931 [9]. As early as 1902, the Hungarian scholar Aujeszky reported the disease for the first time [1]. Then in 1910, Schniedhofer proved that the disease was caused by the virus. Sabin and Wright confirmed that the virus was herpes virus in 1934. In the next year, Shope found that pigs played an important role in the transmission of the disease. In 1947, Liu first reported pseudorabies in cats [10]. The transmission characteristics of pseudorabies and the infection situation in different periods were

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studied in [4], and phylogenetic analysis was carried out in [2, 7, 21]. Very recently, considering the vertical transmission, [18] studied the dynamics of pseudorabies by establishing an SEIT mathematical model.

PRV infection can elicit nervous system disorders. Its clinical manifestations mainly depend on the virulence and the amount of infection, as well as the age of infected pigs. Among them, the age of infected pigs is the most important factor, and the clinical symptoms of different age groups are also different. The disease of piglets infected with PRV is the most serious. Neonatal piglets infected with PRV will cause a large number of deaths. In clinical diagnosis, the newborn piglets show normal performance on the first day. The disease begins to occur from the second day and reach the peak of death within 3 to 5 days [3, 12]. At the same time, the sick piglets show obvious neurological symptoms, such as drowsiness, singing, vomiting and diarrhea, and the temperature rises above 41°C. Once the disease occurs, the sick piglets will die within 1 to 2 days. When piglets within 15 days of age are infected with the disease, the disease is very serious and the mortality rate can reach 100%. The incidence rate of PRV in weaned piglets is about $20\% \sim 40\%$, and the mortality rate is about $10\% \sim 20\%$ [3, 12]. Piglets born more than 60 days after infection show mild symptoms, such as transient fever, mental depression and low mortality, but those piglets grow slowly and have low feed reward. Adult pigs are generally recessive infection, even if there are symptoms occur, they are also very mild and easy to recover and generally recover within $4 \sim 8$ days. At present, there is no specific drug for the disease, only to take measures such as vaccine prevention, timely isolation, elimination of sick pigs, purification of pig population and so on.

On the other hand, nowadays, although PRV has been eradicated from domesticated pigs in North America and some European countries with only sporadic outbreaks, it continues to be found throughout China [20]. Particularly, with antigenically different PRV variants have emerged since 2012 [2, 29], it becomes more and more difficult to control porcine pseudorabies with vaccination [20]. Even worse, the number of swine infected with pseudorabies has shown an upward trend, which results in severe restriction of the development of the pig industry and considerable economic losses to farmers in recent years [11].

In view of the above observations, it is urgent to develop both the theoretical and clinical method to diagnose and control PRV infection accurately and quickly. Therefore, we propose an age-structured ($S_1I_1S_2I_2R_2$) model based on the influence and transmission characteristics of PRV infection on two pig groups of different ages, and study the transmission dynamics of the disease. The outline of this paper is as following: after this introduction, we formulate the mathematical model. In Section 3, we study the existence and dynamic behavior of equilibria by a threshold value, the basic reproduction number, $\Re_0$. Finally, we draw a brief conclusion in Section 4 to accomplish this paper.

2. Model formulation

Considering that PRV infection brings different effects on piglets and adult pigs [3, 12], we divide the swine population into five classes, namely, the susceptible piglets (S_1), infected piglets (I_1), susceptible adult pigs (S_2), infected adult pigs (I_2) and recovered adult pigs(R_2), such that the total population size is $N =$

$S_1 + I_1 + S_2 + I_2 + R_2$. At first, we assume all parameters are positive and make the following hypotheses:

(i) Piglets and adult pigs are kept separately and only the symptomatic infected swines are treated. Let the recruit, newborn piglets, is a constant Λ and they are susceptible. Assume a fraction ε of newborn piglets grow up with no symptoms and progress to class S_2.
(ii) The transmission is assumed to take the form of direct contact between the infectious and the susceptible and the incidence rate is $\beta_1 S_1 I_1$ or $\beta_2 S_2 I_2$ of piglets or adult pigs, respectively.
(iii) The symptomatic infected swines are treated. After the treatment, some of piglets grow up and enter class S_2 with rate γ. After the treatment, some of adult pigs become asymptomatic and enter the recovery class R_2 with rate ν. Some recovered adult pigs with no immunity reenter class S_2 with fraction μ.
(iv) Let d_1 and α denote the natural death rate and the disease-related death rate of piglets, respectively. The adult pig is generally recessive infection, even if the adult pig gets sick, the symptoms are very mild and easy to recover. Therefore, the disease mortality of adult pigs is omitted. Here let d and δ represent the natural death rate and the killing rate of adult pigs, respectively.

For convenience, we write $d_2 = d + \delta$ to be the death rate, including the natural death rate d and the killing rate δ, of adult pigs. Considering the economic effect, mass slaughter of adult pigs leads to $d_2 \gg d_1$. Meanwhile, taking into account of the fact that the smaller the age of the piglets, the higher incidence rate and mortality rate [12], we may assume that

$$\gamma < \varepsilon \leq \alpha + \gamma, \quad d_2 \geq d_1 + \varepsilon. \tag{2.1}$$

With the above assumptions, we illustrate the model dynamics in Figure 1.

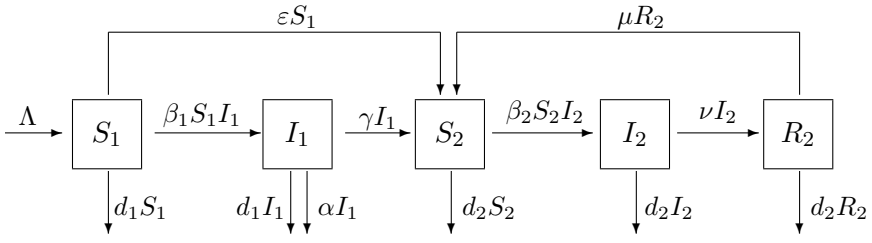

FIGURE 1. Progression of infection from the susceptible piglets (S_1) through infected piglets (I_1), susceptible adult pigs (S_2), infected adult piglets (I_2) and recovered adult pigs (R_2) for the model (2.2).

Consequently, the model we are concerned with is described by the following differential equations together with nonnegative initial conditions

$$\begin{aligned}
\frac{dS_1}{dt} &= \Lambda - \beta_1 S_1 I_1 - (d_1+\varepsilon)S_1 := F_1(S_1, I_1, S_2, I_2, R_2),\\
\frac{dI_1}{dt} &= \beta_1 S_1 I_1 - (d_1+\alpha+\gamma)I_1 := F_2(S_1, I_1, S_2, I_2, R_2),\\
\frac{dS_2}{dt} &= \varepsilon S_1 + \gamma I_1 - \beta_2 S_2 I_2 - d_2 S_2 + \mu R_2 := F_3(S_1, I_1, S_2, I_2, R_2),\\
\frac{dI_2}{dt} &= \beta_2 S_2 I_2 - (d_2+\nu)I_2 := F_4(S_1, I_1, S_2, I_2, R_2),\\
\frac{dR_2}{dt} &= \nu I_2 - (d_2+\mu)R_2 := F_5(S_1, I_1, S_2, I_2, R_2).
\end{aligned} \tag{2.2}$$

Obviously, for $i = 1,2,3,4,5$, $F_i : \mathbb{R}^5_+ \to \mathbb{R}$ are C^1 functions. Moreover, $F_1(0,0,0,0,0) = \Lambda$ and $F_i(0,0,0,0,0) = 0$ for $i = 2,3,4,5$. Hence, given an initial condition $(S_1(0), I_1(0), S_2(0), I_2(0), R_2(0)) \in \mathbb{R}^5_+$, (2.2) admits a unique solution $(S_1(t), I_1(t), S_2(t), I_2(t), R_2(t)) \in \mathbb{R}^5_+$ through $(S_1(0), I_1(0), S_2(0), I_2(0), R_2(0))$ on the existence interval.

Let $N = N_1 + N_2$ be the total number of pigs, where $N_1 = S_1 + I_1$ and $N_2 = S_2 + I_2 + R_2$ are the total number of piglets and adult pigs, respectively. Then from system (2.2), we have

$$\begin{aligned}
\frac{dN_1}{dt} &= \Lambda - d_1 N_1 - \varepsilon S_1 - (\alpha+\gamma)I_1,\\
\frac{dN_2}{dt} &= \varepsilon S_1 + \gamma I_1 - d_2 N_2,
\end{aligned} \tag{2.3}$$

and

$$\frac{dN}{dt} = \Lambda - d_1 N_1 - d_2 N_2 - \alpha I_1. \tag{2.4}$$

Taking account of $\gamma < \varepsilon \le \alpha + \gamma$ and the nonnegativity of the solution, (2.3) leads to

$$\begin{aligned}
\frac{dN_1}{dt} &\le \Lambda - (d_1+\varepsilon)N_1,\\
\frac{dN_2}{dt} &\le \varepsilon N_1 - d_2 N_2.
\end{aligned}$$

Then the above estimation gives

$$\begin{aligned}
N_1(t) &\le \frac{\Lambda}{d_1+\varepsilon} - \frac{\Lambda}{d_1+\varepsilon} e^{-(d_1+\varepsilon)t} + N_1(0)e^{-(d_1+\varepsilon)t},\\
N_2(t) &\le \frac{\varepsilon\Lambda}{d_2(d_1+\varepsilon)} - \frac{\varepsilon\Lambda}{d_2(d_1+\varepsilon)} e^{-d_2 t} + N_2(0)e^{-d_2 t}.
\end{aligned}$$

Consequently,

$$\limsup_{t\to\infty} N_1(t) \le \frac{\Lambda}{d_1+\varepsilon} \quad \text{and} \quad \limsup_{t\to\infty} N_2(t) \le \frac{\varepsilon\Lambda}{d_2(d_1+\varepsilon)},$$

that is

$$\limsup_{t\to\infty}(S_1(t)+I_1(t)) \le \frac{\Lambda}{d_1+\varepsilon}, \quad \limsup_{t\to\infty}(S_2(t)+I_2(t)+R_2(t)) \le \frac{\varepsilon\Lambda}{d_2(d_1+\varepsilon)}. \tag{2.5}$$

Consider $d_2 \gg d_1$, then the nonnegativity of the solutions of system (2.2) and (2.4) lead to

$$\frac{dN}{dt} \leq \Lambda - d_1 N.$$

In the same manner as (2.5), it yields that

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{d_1}.$$

Then

$$\limsup_{t\to\infty}(S_1(t) + I_1(t) + S_2(t) + I_2(t) + R_2(t)) \leq \frac{\Lambda}{d_1},$$

which shows that the solution $(S_1(t), I_1(t), S_2(t), I_2(t), R_2(t))$ is ultimately bounded and exists on $[0, +\infty)$. Denote

$$\Omega = \Big\{(S_1, I_1, S_2, I_2, R_2) \in \mathbb{R}^5_+ | 0 < S_1 + I_1 + S_2 + I_2 + R_2 \leq \frac{\Lambda}{d_1}\Big\}. \tag{2.6}$$

Similar to [25], one can prove that Ω is a positive invariant set with respect to system (2.2). Therefore, in order to make the model biologically meaningful, here and hereafter, we focus our investigation on the dynamical behavior of system (2.2) on Ω.

3. Dynamics of system (2.2)

In this section, we carry out detailed mathematical analysis on dynamics of system (2.2) by considering all possible nonnegative equilibria, their existence and global stability.

3.1. Dynamics of disease-free equilibrium P_0.

Let

$$\begin{aligned}
\Lambda - \beta_1 S_1 I_1 - (d_1 + \varepsilon) S_1 &= 0,\\
\beta_1 S_1 I_1 - (d_1 + \alpha + \gamma) I_1 &= 0,\\
\varepsilon S_1 + \gamma I_1 - \beta_2 S_2 I_2 - d_2 S_2 + \mu R_2 &= 0,\\
\beta_2 S_2 I_2 - (d_2 + \nu) I_2 &= 0,\\
\nu I_2 - (d_2 + \mu) R_2 &= 0.
\end{aligned} \tag{3.1}$$

Then (3.1) ensures that system (2.2) always possesses a disease-free equilibrium (DFE) P_0 unconditionally and

$$P_0 = (S_1^0, I_1^0, S_2^0, I_2^0, R_2^0) = \Big(\frac{\Lambda}{d_1 + \varepsilon}, 0, \frac{\varepsilon\Lambda}{d_2(d_1 + \varepsilon)}, 0, 0\Big).$$

Write $\Re_0$ be the basic reproduction number of system (2.2) [5, 6], according to [16, 17, 28], we have

$$\Re_0 = \max\{\Re_{01}, \Re_{02}\},$$

where

$$\Re_{01} = \frac{\beta_1 S_1^0}{d_1 + \alpha + \gamma} = \frac{\beta_1 \Lambda}{(d_1 + \varepsilon)(d_1 + \alpha + \gamma)}, \quad \Re_{02} = \frac{\beta_2 S_2^0}{d_2 + \nu} = \frac{\varepsilon\beta_2\Lambda}{d_2(d_1 + \varepsilon)(d_2 + \nu)}.$$

Theorem 3.1. *Consider system* (2.2)*, the disease-free equilibrium P_0 is globally asymptotically stable if $\Re_0 < 1$ and is unstable if $\Re_0 > 1$.*

Proof. Define a scalar function $V_0 \in C^1(\mathbb{R}^5_+, \mathbb{R})$ as $V_0 = I_1 + I_2$. Using (2.5), the derivative of V_0 along the solutions of system (2.2) is

$$\begin{aligned}\frac{dV_0}{dt} =& \frac{dI_1}{dt} + \frac{dI_2}{dt}\\ =&(\beta_1 S_1 - (d_1+\alpha+\gamma))I_1 + (\beta_2 S_2 - (d_2+\nu))I_2\\ \le&\Big(\frac{\beta_1\Lambda}{d_1+\varepsilon} - (d_1+\alpha+\gamma)\Big)I_1 + \Big(\frac{\varepsilon\beta_2\Lambda}{d_2(d_1+\varepsilon)} - (d_2+\nu)\Big)I_2\\ =&(d_1+\alpha+\gamma)(\Re_{01}-1)I_1 + (d_2+\nu)(\Re_{02}-1)I_2.\end{aligned}$$

Thus $\Re_0 = \max\{\Re_{01}, \Re_{02}\} < 1$ guarantees $\frac{dV_0}{dt} \le 0$. Moreover, $\frac{dV_0}{dt} = 0$ holds if and only if $I_1 = I_2 = 0$, which means that $\Omega = \{P_0\}$ is the largest invariant set in $\{(S_1, I_1, S_2, I_2, R_2) \in \Omega | \frac{dV_0}{dt} = 0\}$. Then the global stability of P_0 follows immediately from the LaSalle's invariance principle [23].

Owing to the expression of system (2.2), the Jacobian matrix of system (2.2) at the equilibrium P_0 is in the form

$$J(P_0) = \begin{pmatrix} -(d_1+\varepsilon) & -\beta_1 S_1^0 & 0 & 0 & 0\\ 0 & \beta_1 S_1^0 - (d_1+\alpha+\gamma) & 0 & 0 & 0\\ \varepsilon & \gamma & -d_2 & -\beta_2 S_2^0 & \mu\\ 0 & 0 & 0 & \beta_2 S_2^0 - (d_2+\nu) & 0\\ 0 & 0 & 0 & \nu & -(d_2+\mu)\end{pmatrix},$$

then the corresponding characteristic equation is

$$\begin{aligned}&(\lambda+d_2)(\lambda+d_1+\varepsilon)(\lambda+d_2+\mu)(\lambda+(d_1+\alpha+\gamma)(\Re_{01}-1))\\ &\times(\lambda+(d_2+\nu)(\Re_{02}-1)) = 0.\end{aligned} \tag{3.2}$$

Clearly, (3.2) implies that the eigenvalues of $J(P_0)$ are

$$\begin{aligned}&\lambda_1 = -d_2 < 0, \quad \lambda_2 = -(d_1+\varepsilon) < 0, \quad \lambda_3 = -(d_2+\mu) < 0,\\ &\lambda_4 = -(d_1+\alpha+\gamma)(\Re_{01}-1), \quad \lambda_5 = -(d_2+\nu)(\Re_{02}-1).\end{aligned}$$

Suppose $\Re_0 = \max\{\Re_{01}, \Re_{02}\} < 1$, which implies $\lambda_4 < 0$ and $\lambda_5 < 0$. Hence, all eigenvalues $\lambda_i s$ $(i = 1, 2, 3, 4, 5)$ are negative. Whereas, if $\Re_0 = \max\{\Re_{01}, \Re_{02}\} > 1$, then we have at least either $\lambda_4 > 0$ or $\lambda_5 > 0$. Therefore, $J(P_0)$ has at least one positive eigenvalue. As a result, the DFE P_0 is unstable. □

3.2. **Dynamics of the boundary equilibria P_{01} and P_{02}.** In addition to the DFE P_0, (3.1) indicates that system (2.2) admits other equilibria. In this subsection, we devote to studying the existence and stability of boundary equilibria P_{01} and P_{02} in the form of $(S_1^{01}, 0, S_2^{01}, I_2^{01}, R_2^{01})$ (only the adult pigs are infected) and $(S_1^{02}, I_1^{02}, S_2^{02}, 0, 0)$ (only the piglets are infected), respectively.

Theorem 3.2. *If $\Re_{02} > 1$, then system (2.2) has a boundary equilibrium P_{01} with*

$$P_{01} = \left(\frac{\Lambda}{d_1+\varepsilon}, 0, \frac{d_2+\nu}{\beta_2}, \frac{(d_2+\mu)(d_2+\nu)(\Re_{02}-1)}{\beta_2(d_2+\mu+\nu)}, \frac{\nu(d_2+\nu)(\Re_{02}-1)}{\beta_2(d_2+\mu+\nu)}\right).$$

Further, if $\Re_{01} < 1$ then P_{01} is globally asymptotically stable and if $\Re_{01} > 1$ then P_{01} is unstable.

Proof. Recall (3.1), direct computation shows that system (2.2) just possesses boundary equilibria in the forms of P_{01} and P_{02}. In the following, we start with seeking the boundary equilibrium P_{01}. Let $I_1^{01} = 0$, then

$$\begin{aligned} \Lambda - (d_1 + \varepsilon)S_1^{01} &= 0, \\ \varepsilon S_1^{01} - \beta_2 S_2^{01} I_2^{01} - d_2 S_2^{01} + \mu R_2^{01} &= 0, \\ \beta_2 S_2^{01} I_2^{01} - (d_2 + \nu) I_2^{01} &= 0, \\ \nu I_2^{01} - (d_2 + \mu) R_2^{01} &= 0. \end{aligned} \tag{3.3}$$

By (3.3), it is straightforward to find that

$$\begin{gathered} S_1^{01} = \frac{\Lambda}{d_1 + \varepsilon} > 0, \quad S_2^{01} = \frac{d_2 + \nu}{\beta_2} > 0, \\ I_2^{01} = \frac{(d_2 + \mu)(d_2 + \nu)(\Re_{02} - 1)}{\beta_2(d_2 + \mu + \nu)}, \quad R_2^{01} = \frac{\nu I_2^{01}}{d_2 + \mu}, \end{gathered}$$

$I_2^{01} > 0$ induces $R_2^{01} > 0$. Hence, $\Re_{02} > 1$ ensures both $I_2^{01} > 0$ and $R_2^{01} > 0$. Therefore, system (2.2) has a boundary equilibrium P_{01} when $\Re_{02} > 1$.

Thanks to [13, 31] and by using (3.3), system (2.2) is transformed into the following equivalent system

$$\begin{aligned} \frac{dN_1}{dt} &= -d_1(N_1 - N_1^{01}) - \varepsilon(N_1 - N_1^{01}) - (\alpha + \gamma - \varepsilon) I_1, \\ \frac{dI_1}{dt} &= \beta_1(N_1 - N_1^{01} - I_1) I_1 - (d_1 + \alpha + \gamma) I_1 + \beta_1 N_1^{01} I_1, \\ \frac{dN_2}{dt} &= \varepsilon(N_1 - N_1^{01}) - \varepsilon I_1 + \gamma I_1 - d_2(N_2 - N_2^{01}), \\ \frac{dI_2}{dt} &= \beta_2((N_2 - N_2^{01}) - (I_2 - I_2^{01}) - (R_2 - R_2^{01})) I_2, \\ \frac{dR_2}{dt} &= \nu(I_2 - I_2^{01}) - (d_2 + \mu)(R_2 - R_2^{01}), \end{aligned} \tag{3.4}$$

with $N_1 = S_1 + I_1$, $N_1^{01} = S_1^{01}$, $N_2 = S_2 + I_2 + R_2$ and $N_2^{01} = S_2^{01} + I_2^{01} + R_2^{01}$.

To obtain the global asymptotic stability of system (2.2) around P_{01}, motivated by [19], we construct a Lyapunov function as

$$V_{01} = \frac{\alpha}{\beta_1} I_1 + \frac{1}{2}(N_1 - N_1^{01} + N_2 - N_2^{01})^2 + \frac{d_1 + d_2}{2\varepsilon}(N_2 - N_2^{01})^2.$$

Then the derivative of V_{01} along the solutions of System (3.4) is

$$\begin{aligned} &\frac{dV_{01}}{dt} \\ &= \frac{\alpha}{\beta_1}\frac{dI_1}{dt} + (N_1 - N_1^{01} + N_2 - N_2^{01})\Big(\frac{dN_1}{dt} + \frac{dN_2}{dt}\Big) + \frac{d_1 + d_2}{\varepsilon}(N_2 - N_2^{01})\frac{dN_2}{dt} \\ &= (N_1 - N_1^{01} + N_2 - N_2^{01})\Big(- d_1(N_1 - N_1^{01}) - d_2(N_2 - N_2^{01}) - \alpha I_1\Big) \\ &\quad + \frac{d_1 + d_2}{\varepsilon}(N_2 - N_2^{01})\Big(\varepsilon(N_1 - N_1^{01}) - \varepsilon I_1 + \gamma I_1 - d_2(N_2 - N_2^{01})\Big) \\ &\quad + \frac{\alpha}{\beta_1}\Big(\beta_1(N_1 - N_1^{01} - I_1) I_1 - (d_1 + \alpha + \gamma) I_1 + \beta_1 N_1^{01} I_1\Big) \\ &= -d_1(N_1 - N_1^{01})^2 - d_2(N_1 - N_1^{01})(N_2 - N_2^{01}) - \alpha I_1(N_1 - N_1^{01}) \end{aligned}$$

$$
\begin{aligned}
&- d_1(N_1 - N_1^{01})(N_2 - N_2^{01}) - d_2(N_2 - N_2^{01})^2 - \alpha I_1(N_2 - N_2^{01}) \\
&+ (d_1 + d_2)(N_1 - N_1^{01})(N_2 - N_2^{01}) - (d_1 + d_2) I_1(N_2 - N_2^{01}) \\
&+ \frac{\gamma(d_1 + d_2)}{\varepsilon} I_1(N_2 - N_2^{01}) - \frac{d_2(d_1 + d_2)}{\varepsilon}(N_2 - N_2^{01})^2 - \alpha I_1^2 + \alpha I_1(N_1 - N_1^{01}) \\
&+ \frac{\alpha}{\beta_1}(\beta_1 N_1^{01} - (d_1 + \alpha + \gamma)) I_1 \\
&= -\alpha I_1^2 - d_1(N_1 - N_1^{01})^2 - \frac{d_2(\varepsilon + d_1 + d_2)}{\varepsilon}(N_2 - N_2^{01})^2 \\
&- \frac{\varepsilon(\alpha + d_1 + d_2) - \gamma(d_1 + d_2)}{\varepsilon} I_1(N_2 - N_2^{01}) - \frac{\alpha}{\beta_1}(d_1 + \alpha + \gamma - \frac{\beta_1 \Lambda}{d_1 + \varepsilon}) I_1 \\
&= -\alpha I_1^2 - d_1(N_1 - N_1^{01})^2 - \frac{d_2(\varepsilon + d_1 + d_2)}{\varepsilon}(N_2 - N_2^{01})^2 \\
&- \frac{\varepsilon(\alpha + d_1 + d_2) - \gamma(d_1 + d_2)}{\varepsilon} I_1(N_2 - N_2^{01}) - \frac{\alpha}{\beta_1}(d_1 + \alpha + \gamma)(1 - \Re_{01}) I_1 \\
&\le -\alpha I_1^2 - d_1(N_1 - N_1^{01})^2 - \frac{d_2(\varepsilon + d_1 + d_2)}{\varepsilon}(N_2 - N_2^{01})^2 \\
&- \frac{\varepsilon(\alpha + d_1 + d_2) - \gamma(d_1 + d_2)}{\varepsilon} I_1(N_2 - N_2^{01}).
\end{aligned} \tag{3.5}
$$

This last inequality is true because $\Re_{01} < 1$.

For convenience, let $\widehat{I_1} = I_1$, $\widehat{N_1} = N_1 - N_1^{01}$, $\widehat{N_2} = N_2 - N_2^{01}$ and rewrite (3.5) as

$$
\begin{aligned}
\frac{dV_{01}}{dt} &\le -\alpha \widehat{I_1}^2 - d_1 \widehat{N_1}^2 - \frac{d_2(\varepsilon + d_1 + d_2)}{\varepsilon} \widehat{N_2}^2 - \frac{\varepsilon(\alpha + d_1 + d_2) - \gamma(d_1 + d_2)}{\varepsilon} \widehat{I_1} \widehat{N_2} \\
&= -(\widehat{I_1}, \widehat{N_1}, \widehat{N_2}) A (\widehat{I_1}, \widehat{N_1}, \widehat{N_2})^T,
\end{aligned}
$$

where

$$
A = \begin{pmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{pmatrix} = \begin{pmatrix} \alpha & 0 & A_{31} \\ 0 & d_1 & 0 \\ A_{13} & 0 & A_{33} \end{pmatrix}, \tag{3.6}
$$

is a real symmetric matrix with

$$
A_{33} = \frac{d_2(\varepsilon + d_1 + d_2)}{\varepsilon} > 0, \quad A_{13} = A_{31} = \frac{\varepsilon(\alpha + d_1 + d_2) - \gamma(d_1 + d_2)}{2\varepsilon}.
$$

Next we show that A is positive definite. By (3.6),

$$
A_{11} = \alpha > 0, \quad A_{11} A_{22} - A_{21} A_{12} = \alpha d_1 > 0. \tag{3.7}
$$

It is then left to verify that $\det A > 0$. Note that

$$
\begin{aligned}
\det A =& d_1(\alpha A_{33} - A_{13}^2) \\
=& \frac{\alpha d_1 d_2(\varepsilon + d_1 + d_2)}{\varepsilon} - d_1 \Big(\frac{\varepsilon(\alpha + d_1 + d_2) - \gamma(d_1 + d_2)}{2\varepsilon} \Big)^2 \\
=& \frac{\alpha d_1 d_2(\varepsilon + d_1 + d_2)}{\varepsilon} - \frac{d_1 \varepsilon^2 (\alpha + d_1 + d_2)^2}{4\varepsilon^2} + \frac{2 d_1 \varepsilon \gamma(\alpha + d_1 + d_2)(d_1 + d_2)}{4\varepsilon^2} \\
&- \frac{d_1 \gamma^2 (d_1 + d_2)^2}{4\varepsilon^2} \\
>& \frac{d_1}{4\varepsilon^2} \left(4\varepsilon \alpha d_2(\varepsilon + d_1 + d_2) + 2\varepsilon \gamma(\alpha + d_1 + d_2)(d_1 + d_2) - 2\varepsilon^2 (\alpha + d_1 + d_2)^2 \right)
\end{aligned}
$$

$$=\frac{d_1}{2\varepsilon}\left(2\alpha d_2(\varepsilon+d_1+d_2)+\gamma(\alpha+d_1+d_2)(d_1+d_2)-\varepsilon(\alpha+d_1+d_2)^2\right).$$

Let $\xi=\min\{\varepsilon,\alpha\}$. Owing to (2.1), there holds $\varepsilon\le\min\{\alpha+\gamma,d_2-d_1\}$, then

$$\begin{aligned}\det A\ge&\frac{d_1(d_1+d_2+\xi)\left(2\alpha d_2+\gamma(d_1+d_2)-\varepsilon(\alpha+d_1+d_2)\right)}{2\varepsilon}\\ \ge&\frac{d_1(d_1+d_2+\xi)(2\alpha d_2+(\varepsilon-\alpha)(d_1+d_2)-\varepsilon(\alpha+d_1+d_2))}{2\varepsilon}\\ =&\frac{\alpha d_1(d_1+d_2+\xi)(d_2-d_1-\varepsilon)}{2\varepsilon}>0.\end{aligned}\tag{3.8}$$

Then (3.7) and (3.8) yield A is a positive definite matrix. Therefore,

$$\frac{dV_{01}}{dt}\le 0$$

and $\frac{dV_{01}}{dt}=0$ if and only if $\widehat{I_1}=0$, $\widehat{N_1}=0$ and $\widehat{N_2}=0$, that is, $S_1=S_1^{01}$, $I_1=0$, $S_2=S_2^{01}$, $I_2=I_2^{01}$ and $R_2=R_2^{01}$. Immediately, the LaSalle's invariance principle [23] ensures system (2.2) is globally asymptotically stable around P_{01}.

Next, we show the instability of system (2.2) around P_{01}. Consider the Jacobian matrix $J(P_{01})$ of system (2.2) at the equilibrium P_{01}, we have

$$J(P_{01})=\begin{pmatrix}-(d_1+\varepsilon) & -\beta_1S_1^{01} & 0 & 0 & 0\\ 0 & \beta_1S_1^{01}-(d_1+\alpha+\gamma) & 0 & 0 & 0\\ \varepsilon & \gamma & -\beta_2I_2^{01}-d_2 & -\beta_2S_2^{01} & \mu\\ 0 & 0 & \beta_2I_2^{01} & \beta_2S_2^{01}-(d_2+\nu) & 0\\ 0 & 0 & 0 & \nu & -(d_2+\mu)\end{pmatrix}.$$

We write

$$\begin{aligned}m_1=&d_2+\mu+\beta_2I_2^{01},\\ m_2=&(d_2+\mu+\nu)\beta_2I_2^{01},\\ n_1=&d_1+\varepsilon+(d_1+\alpha+\gamma)(1-\Re_{01}),\\ n_2=&(d_1+\varepsilon)(d_1+\alpha+\gamma)(1-\Re_{01}).\end{aligned}\tag{3.9}$$

Evidently, combine the positiveness of I_2^{01} with $\Re_{01}>1$, (3.9) gives

$$m_1>0,\quad m_2>0,\quad n_1<0,\quad n_2<0.\tag{3.10}$$

On the other side, the associated characteristic equation of the Jacobian matrix $J(P_{01})$ of system (2.2) at the equilibrium P_{01} is

$$(\sigma+d_2)(\sigma^2+m_1\sigma+m_2)(\sigma^2+n_1\sigma+n_2)=0.\tag{3.11}$$

We denote σ_is $(i=1,2,3,4,5)$ be the roots of (3.11). Then

$$\sigma_1=-d_2,\quad \sigma_2+\sigma_3=-m_1,\quad \sigma_2\sigma_3=m_2,\quad \sigma_4+\sigma_5=-n_1,\quad \sigma_4\sigma_5=n_2.\tag{3.12}$$

Joint (3.10) with (3.12), it follows that

$$\sigma_1<0,\quad \sigma_2<0,\quad \sigma_3<0$$

and either $\sigma_4>0$ or $\sigma_5>0$, which imply that $J(P_{01})$ has a positive eigenvalue. The proof is complete. □

Let

$$\begin{aligned}\epsilon =&\frac{((\varepsilon-\gamma)d_1+\varepsilon\alpha)(\beta_1\Lambda-(d_1+\varepsilon)(d_1+\alpha+\gamma))}{\gamma(d_1+\varepsilon)\beta_1\Lambda+(d_1+\varepsilon)(d_1+\alpha+\gamma)((\varepsilon-\gamma)d_1+\varepsilon\alpha)}\\ =&\frac{((\varepsilon-\gamma)d_1+\varepsilon\alpha)(d_1+\alpha+\gamma)(\Re_{01}-1)}{\gamma(d_1+\varepsilon)\beta_1\Lambda+(d_1+\varepsilon)(d_1+\alpha+\gamma)((\varepsilon-\gamma)d_1+\varepsilon\alpha)},\end{aligned} \tag{3.13}$$

then $\epsilon>0$ for $\Re_{01}>1$. Concerning the existence and the stability of boundary equilibrium in the form of P_{02}, we have the following result.

Theorem 3.3. *If $\Re_{01}>1$, then system (2.2) has a boundary equilibrium $P_{02}=(S_1^{02},I_1^{02},S_2^{02},0,0)$ with*

$$\begin{aligned}S_1^{02}&=\frac{d_1+\alpha+\gamma}{\beta_1},\quad I_1^{02}=\frac{(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1},\\ S_2^{02}&=\frac{\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1 d_2}.\end{aligned}$$

Further, if $\Re_{02}<1+\epsilon$, where ϵ is defined by (3.13), then P_{02} is globally asymptotically stable and if $\Re_{02}>1+\epsilon$, then P_{02} is unstable.

Proof. From (3.1), when $I_2^{02}=R_2^{02}=0$, it is straightforward to find that

$$\begin{aligned}S_1^{02}&=\frac{d_1+\alpha+\gamma}{\beta_1},\\ I_1^{02}&=\frac{(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1},\\ S_2^{02}&=\frac{\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1 d_2}.\end{aligned} \tag{3.14}$$

Immediately, $\Re_{01}>1$ ensures that S_1^{02}, I_1^{02}, S_2^{02}, given in (3.14), are all positive. Thus system (2.2) admits a boundary equilibrium P_{02} for $\Re_{01}>1$.

In the same manner as for (3.4), we turn system (2.2) into its equivalent form

$$\begin{aligned}\frac{dN_1}{dt}&=-d_1(N_1-N_1^{02})-\varepsilon((N_1-N_1^{02})-(I_1-I_1^{02}))-(\alpha+\gamma)(I_1-I_1^{02}),\\ \frac{dI_1}{dt}&=\beta_1 I_1((N_1-N_1^{02})-(I_1-I_1^{02})),\\ \frac{dN_2}{dt}&=\varepsilon((N_1-N_1^{02})-(I_1-I_1^{02}))+\gamma(I_1-I_1^{02})-d_2(N_2-N_2^{02}),\\ \frac{dI_2}{dt}&=\beta_2 I_2(N_2-N_2^{02}-I_2-R_2)+\beta_2 N_2^{02}I_2-(d_2+\nu)I_2,\\ \frac{dR_2}{dt}&=\nu I_2-(d_2+\mu)R_2.\end{aligned} \tag{3.15}$$

Here $N_1=S_1+I_1$, $N_1^{02}=S_1^{02}+I_1^{02}$, $N_2=S_2+I_2+R_2$ and $N_2^{02}=S_2^{02}$.

We define a function $V_{02}:\mathbb{R}_+^5\to\mathbb{R}$ as

$$\begin{aligned}V_{02}=&\frac{\alpha}{\beta_1}(I_1-I_1^{02}-I_1^{02}\ln\frac{I_1}{I_1^{02}})+\frac{1}{2}(N_1-N_1^{02}+N_2-N_2^{02})^2\\ &+\frac{d_1+d_2}{2\varepsilon}(N_2-N_2^{02})^2.\end{aligned} \tag{3.16}$$

Then the derivative of V_{02}, defined by (3.16), along System (3.15) is

$$\begin{aligned}\frac{dV_{02}}{dt} =&\frac{\alpha}{\beta_1}\Big(1-\frac{I_1^{02}}{I_1}\Big)\frac{dI_1}{dt}+(N_1-N_1^{02}+N_2-N_2^{02})\Big(\frac{dN_1}{dt}+\frac{dN_2}{dt}\Big)\\ &+\frac{d_1+d_2}{\varepsilon}(N_2-N_2^{02})\frac{dN_2}{dt}\\ =&-\alpha(I_1-I_1^{02})^2-d_1(N_1-N_1^{02})^2-\frac{d_2(\varepsilon+d_1+d_2)}{\varepsilon}(N_2-N_2^{02})^2\\ &-\frac{\varepsilon(\alpha+d_1+d_2)-\gamma(d_1+d_2)}{\varepsilon}(I_1-I_1^{02})(N_2-N_2^{02})\\ =&-(\widetilde{I_1},\widetilde{N_1},\widetilde{N_2})A(\widetilde{I_1},\widetilde{N_1},\widetilde{N_2})^T,\end{aligned}$$

where A is a positive definite matrix defined by (3.6) and $\widetilde{I_1}=I_1-I_1^{02}$, $\widetilde{N_1}=N_1-N_1^{02}$, $\widetilde{N_2}=N_2-N_2^{02}$.

Thus $\frac{dV_{02}}{dt}\le 0$ and $\frac{dV_{02}}{dt}=0$ is true if and only if $\widetilde{I_1}=0$, $\widetilde{N_1}=0$ and $\widetilde{N_2}=0$, which implies that $S_1=S_1^{02}$, $I_1=I_1^{02}$, $S_2=S_2^{02}$ and $I_2=R_2=0$. By the LaSalle's Invariance Principle, P_{02} is globally asymptotically stable.

Let $\theta_i s$ $(i=1,2,3,4,5)$ be the eigenvalues of the Jacobian matrix $J(P_{02})$ of system (2.2) around P_{02}, then $\theta_i s$ satisfy

$$\begin{aligned}&\begin{vmatrix}\theta+\beta_1I_1^{02}+d_1+\varepsilon & \beta_1S_1^{02} & 0 & 0 & 0\\ -\beta_1I_1^{02} & \theta+d_1+\alpha+\gamma-\beta_1S_1^{02} & 0 & 0 & 0\\ -\varepsilon & -\gamma & \theta+d_2 & \beta_2S_2^{02} & -\mu\\ 0 & 0 & 0 & \theta+d_2+\nu-\beta_2S_2^{02} & 0\\ 0 & 0 & 0 & -\nu & \theta+d_2+\mu\end{vmatrix}\\ &=(\theta+d_2)(\theta+d_2+\mu)(\theta+d_2+\nu-\beta_2S_2^{02})\\ &\quad\times\big((\theta+\beta_1I_1^{02}+d_1+\varepsilon)(\theta+d_1+\alpha+\gamma-\beta_1S_1^{02})+\beta_1S_1^{02}\beta_1I_1^{02}\big)\\ &=(\theta+d_2)(\theta+d_2+\mu)(\theta+d_2+\nu-\beta_2S_2^{02})\\ &\quad\times\big(\theta^2+(d_1+\varepsilon)\Re_{01}\theta+(d_1+\varepsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)\big)=0.\end{aligned}$$

It follows that

$$\theta_1=-d_2<0,\quad \theta_2=-(d_2+\mu)<0,\quad \theta_3=\beta_2S_2^{02}-(d_2+\nu) \tag{3.17}$$

and θ_4, θ_5 solve the equation

$$\theta^2+(d_1+\varepsilon)\Re_{01}\theta+(d_1+\varepsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)=0. \tag{3.18}$$

By Vieta's theorem, (3.18) implies

$$\theta_4+\theta_5=-(d_1+\varepsilon)\Re_{01},\quad \theta_4\theta_5=(d_1+\varepsilon)(d_1+\alpha+\gamma)(\Re_{01}-1).$$

From this and $\Re_{01}>1$, we have

$$\theta_4+\theta_5=-(d_1+\varepsilon)\Re_{01}<0,\quad \theta_4\theta_5=(d_1+\varepsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)>0,$$

which guarantee that both θ_4 and θ_5 have negative real parts.

Therefore, we show $\theta_3>0$ to obtain the instability of system (2.2) around P_{02}. Let

$$\zeta=\frac{\varepsilon(d_1+\alpha+\gamma)\Re_{01}}{\gamma(d_1+\varepsilon)(\Re_{01}-1)+\varepsilon(d_1+\alpha+\gamma)}.$$

Notice that $\varepsilon > \gamma$ and $\Re_{01} = \frac{\beta_1\Lambda}{(d_1+\varepsilon)(d_1+\alpha+\gamma)} > 1$, then we have

$$\begin{aligned}
\zeta =& \frac{\varepsilon(d_1+\alpha+\gamma)\beta_1\Lambda}{\gamma(d_1+\varepsilon)(\beta_1\Lambda-(d_1+\varepsilon)(d_1+\alpha+\gamma))+\varepsilon(d_1+\varepsilon)(d_1+\alpha+\gamma)^2}\\
=& \frac{\varepsilon(d_1+\alpha+\gamma)\beta_1\Lambda}{\gamma(d_1+\varepsilon)\beta_1\Lambda+(d_1+\varepsilon)(d_1+\alpha+\gamma)(\varepsilon(d_1+\alpha+\gamma)-\gamma(d_1+\varepsilon))}\\
=& 1+\frac{\varepsilon(d_1+\alpha+\gamma)\beta_1\Lambda}{\gamma(d_1+\varepsilon)\beta_1\Lambda+(d_1+\varepsilon)(d_1+\alpha+\gamma)((\varepsilon-\gamma)d_1+\varepsilon\alpha)}-1\\
=& 1+\frac{\varepsilon(d_1+\alpha+\gamma)\beta_1\Lambda-\gamma(d_1+\varepsilon)\beta_1\Lambda-(d_1+\varepsilon)(d_1+\alpha+\gamma)((\varepsilon-\gamma)d_1+\varepsilon\alpha)}{\gamma(d_1+\varepsilon)\beta_1\Lambda+(d_1+\varepsilon)(d_1+\alpha+\gamma)((\varepsilon-\gamma)d_1+\varepsilon\alpha)}\\
=& 1+\frac{((\varepsilon-\gamma)d_1+\varepsilon\alpha)(\beta_1\Lambda-(d_1+\varepsilon)(d_1+\alpha+\gamma))}{\gamma(d_1+\varepsilon)\beta_1\Lambda+(d_1+\varepsilon)(d_1+\alpha+\gamma)((\varepsilon-\gamma)d_1+\varepsilon\alpha)}\\
=& 1+\epsilon,
\end{aligned}$$

where ϵ is defined by (3.13). Then from (3.17), we obtain

$$\begin{aligned}
\theta_3 =& \beta_2 S_2^{02}-(d_2+\nu)\\
=& \frac{1}{\beta_1 d_2}\left(\varepsilon\beta_2(d_1+\alpha+\gamma)+\gamma\beta_2(d_1+\varepsilon)(\Re_{01}-1)-\beta_1 d_2(d_2+\nu)\right)\\
=& \frac{\beta_2}{\beta_1 d_2}\left(\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1)-\frac{\beta_1 d_2(d_2+\nu)}{\beta_2}\right)\\
=& \frac{\beta_2}{\beta_1 d_2\Re_{02}}\left(\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1)-\frac{\varepsilon(d_1+\alpha+\gamma)\Re_{01}}{\Re_{02}}\right)\\
=& \frac{\beta_2}{\beta_1 d_2\Re_{02}}\Big((\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1))\Re_{02}-\varepsilon(d_1+\alpha+\gamma)\Re_{01}\Big)\\
=& \frac{\beta_2}{\beta_1 d_2\Re_{02}}\Big(\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1)\Big)(\Re_{02}-\zeta)\\
=& \frac{\beta_2}{\beta_1 d_2\Re_{02}}\Big(\varepsilon(d_1+\alpha+\gamma)+\gamma(d_1+\varepsilon)(\Re_{01}-1)\Big)(\Re_{02}-1-\epsilon).
\end{aligned} \tag{3.19}$$

Subsequently, $\Re_{02} > 1+\epsilon$ leads to $\theta_3 > 0$. The proof is complete. □

3.3. **Dynamics of the disease-endemic equilibrium P^*.** In this subsection, we intend to study the existence and stability of the disease-endemic equilibrium $P^* = (S_1^*, I_1^*, S_2^*, I_2^*, R_2^*)$ with $S_1^* > 0$, $I_1^* > 0$, $S_2^* > 0$, $I_2^* > 0$ and $R_2^* > 0$, which implies that both the piglets and adult pigs are infected. We draw a conclusion as follows.

Theorem 3.4. *Let ϵ be given by* (3.13). *If $\Re_{01} > 1$ and $\Re_{02} > 1+\epsilon$, in addition to the DFE P_0 and the boundary equilibria P_{01} and P_{02}, system* (2.2) *also has a unique disease-endemic equilibrium*

$$\begin{aligned}
&P^*\\
&= \Big(\frac{d_1+\alpha+\gamma}{\beta_1}, \frac{(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1}, \frac{d_2+\nu}{\beta_2}, \frac{(d_2+\mu)(\varepsilon S_1^*+\gamma I_1^*-d_2 S_2^*)}{d_2(d_2+\mu+\nu)}, \frac{\nu I_2^*}{d_2+\mu}\Big).
\end{aligned}$$

Moreover, P^* is globally asymptotically stable.

Proof. In view of our obtained existence results of P_0, P_{01} and P_{02}, here we study P^*. Notice that S_1^*, I_1^*, S_2^*, I_2^* and R_2^* solve

$$\begin{aligned}
\Lambda - \beta_1 S_1^* I_1^* - (d_1+\varepsilon)S_1^* &= 0,\\
\beta_1 S_1^* I_1^* - (d_1+\alpha+\gamma)I_1^* &= 0,\\
\varepsilon S_1^* + \gamma I_1^* - \beta_2 S_2^* I_2^* - d_2 S_2^* + \mu R_2^* &= 0,\\
\beta_2 S_2^* I_2^* - (d_2+\nu)I_2^* &= 0,\\
\nu I_2^* - (d_2+\mu)R_2^* &= 0.
\end{aligned} \tag{3.20}$$

Direct calculation yields

$$\begin{gathered}
S_1^* = \frac{d_1+\alpha+\gamma}{\beta_1}, \quad I_1^* = \frac{(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1}, \quad S_2^* = \frac{d_2+\nu}{\beta_2},\\
I_2^* = \frac{(d_2+\mu)(\varepsilon S_1^* + \gamma I_1^* - d_2 S_2^*)}{d_2(d_2+\mu+\nu)}, \quad R_2^* = \frac{\nu I_2^*}{d_2+\mu}.
\end{gathered}$$

It is clear that $S_1^* > 0$, $S_2^* > 0$ and $I_1^* > 0$ for $\Re_{01} > 1$. To show the existence of P^*, we are in the position to prove $I_2^* > 0$. Moreover, the expression of I_2^* indicates that $I_2^* > 0$ is equivalent to $\varepsilon S_1^* + \gamma I_1^* - d_2 S_2^* > 0$. Similar to ((3.19), it follows that

$$\begin{aligned}
\varepsilon S_1^* + \gamma I_1^* - d_2 S_2^* =& \frac{\varepsilon(d_1+\alpha+\gamma)}{\beta_1} + \frac{\gamma(d_1+\varepsilon)(\Re_{01}-1)}{\beta_1} - \frac{d_2(d_2+\nu)}{\beta_2}\\
=& \frac{1}{\beta_1}\Big(\gamma(d_1+\varepsilon)(\Re_{01}-1) + \varepsilon(d_1+\alpha+\gamma) - \frac{\beta_1 d_2(d_2+\nu)}{\beta_2}\Big)\\
=& \frac{1}{\beta_1 \Re_{02}}\left((\varepsilon(d_1+\alpha+\gamma) + \gamma(d_1+\varepsilon)(\Re_{01}-1))\right)(\Re_{02}-1-\epsilon).
\end{aligned}$$

Therefore, $\Re_{01} > 1$ and $\Re_{02} > 1+\epsilon$ ensure $\varepsilon S_1^* + \gamma I_1^* - d_2 S_2^* > 0$ and system (2.2) has a unique disease-endemic equilibrium P^*.

In the following, we show system (2.2) is globally asymptotically stable around P^*. Here, the method of our proof is similar to that of Theorem 3.2. For completeness, we state the sketch of the remain part of the proof.

First, transform system (2.2) into the equivalent system

$$\begin{aligned}
\frac{dN_1}{dt} &= -d_1(N_1 - N_1^*) - \varepsilon((N_1 - N_1^*) - (I_1 - I_1^*)) - (\alpha+\gamma)(I_1 - I_1^*),\\
\frac{dI_1}{dt} &= \beta_1 I_1((N_1 - N_1^*) - (I_1 - I_1^*)),\\
\frac{dN_2}{dt} &= \varepsilon((N_1 - N_1^*) - (I_1 - I_1^*)) + \gamma(I_1 - I_1^*) - d_2(N_2 - N_2^*),\\
\frac{dI_2}{dt} &= \beta_2 I_2((N_2 - N_2^*) - (I_2 - I_2^*) - (R_2 - R_2^*)),\\
\frac{dR_2}{dt} &= \nu(I_2 - I_2^*) - (d_2+\mu)(R_2 - R_2^*),
\end{aligned} \tag{3.21}$$

where $N_1 - N_1^* = S_1 - S_1^* + I_1 - I_1^*$ and $N_2 - N_2^* = S_2 - S_2^* + I_2 - I_2^* + R_2 - R_2^*$.

Next, define a continuously differentiable function $V : \mathbb{R}_+^5 \to \mathbb{R}$ as

$$V = \frac{\alpha}{\beta_1}(I_1 - I_1^* - I_1^* \ln\frac{I_1}{I_1^*}) + \frac{1}{2}(N_1 - N_1^* + N_2 - N_2^*)^2 + \frac{d_1+d_2}{2\varepsilon}(N_2 - N_2^*)^2.$$

Write $\widetilde{I_1^*} = I_1 - I_1^*$, $\widetilde{N_1^*} = N_1 - N_1^*$ and $\widetilde{N_2^*} = N_2 - N_2^*$, then the derivative of V along the solution of System (3.21) is

$$\frac{dV}{dt} = -(\widetilde{I_1^*}, \widetilde{N_1^*}, \widetilde{N_2^*})A(\widetilde{I_1^*}, \widetilde{N_1^*}, \widetilde{N_2^*})^T$$

with A is a positive definite matrix given by (3.6), which means that

$$\frac{dV}{dt} \leq 0.$$

Moreover, $\frac{dV}{dt} = 0$ implies the set $\{(S_1, I_1, S_2, I_2, R_2) \in \Omega : \frac{dV}{dt} = 0\}$ is the singleton P^*. This completes the proof. □

4. Summary

In this paper, to study the infection transmission of porcine pseudorabies, we have proposed a mathematical model ($S_1I_1S_2I_2R_2$) with age structure. We have also derived conditions for the existence, global stability of disease-free equilibrium P_0, boundary equilibria P_{01} and P_{02} (only either piglets or adult pigs are infected) and disease-endemic equilibrium P^* (both piglets and adult pigs are infected).

Define a threshold $\Re_0 = \max\{\Re_{01}, \Re_{02}\}$, we have explored if $\Re_0 < 1$, P_0 is globally asymptotically stable and the disease will die out regardless its initial sizes. Conversely, the invasion is always possible if $\Re_0 > 1$ [6, 8]. If $\Re_{01} < 1$ and $\Re_{02} > 1$ or $\Re_{01} > 1$ and $\Re_{02} < 1 + \epsilon$, system (2.2) admits a globally asymptotically stable boundary equilibrium P_{01} or P_{02}, which means that either piglets or adult pigs are always disease-endemic, respectively. Meanwhile, both $\Re_{01} > 1$ and $\Re_{02} > 1 + \epsilon$, there is a unique positive equilibrium P^* of system (2.2) and P^* is globally asymptotically stable, which indicates that disease is persistent as long as P^* exists.

Furthermore, the expressions of $\Re_{01}$ and $\Re_{02}$ manifest that they are strictly increasing with respect to the contact rates β_1 and β_2. Therefore, we can draw a conclusion as that of [18], that is, isolation of infected pigs and appropriate feeding densities are beneficial to prevent and control the outbreaks of pseudorabies.

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References

[1] A. C. Aujeszky; Ueber eine new infektions krankheit bei haustieren, *Zentbl Bakt ParasitKde*, 32 (1902), 353–357.

[2] T. Q. An, J. M. Peng, Z. J. Tian, H. Y. Zhao, N. Li, Y. M. Liu, et al.; Pseudorabies virus variant in Bartha-K61-vaccinated pigs, China, 2012, *Emerg. Infect. Dis.*, 19 (2013), no. 11, 1749–1755.

[3] D. X. Cheng; Infection, transmission and epidemic of pseudorabies in pigs (in Chinese), *Fujian Animal Husbandry and Veterinary*, 7 (1999), no. 1, 34–35.

[4] B. Chen, L. Ma, J. F. Liu, J. Lin, J. Zhang, J. M. Wu, et al.; Diagnosis of massive death of newborn piglets and isolation and identification of HP-PRRSV (in Chinese), *Chinese Journal of Animal Husbandry and Veterinary Medicine*, 43 (2016), no. 2, 520–526.

[5] O. Diekmann, J. A. P. Heesterbeek, J. A. J. Metz; On the definition and the computation of the basic reproduction ratio $\Re_0$ in models for infectious diseases in heterogeneous populations, *J. Math. Biol.*, 28 (1990), no. 4, 365–382.

[6] P. Van den Driessche, J. Watmough; Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, 180 (2002), 29–48.

[7] N. Denzin, F. J. Conraths, T. C. Mettenleiter, C. M. Freuling, M. Thomas; Monitoring of pseudorabies in wild boar of Germany a spatiotemporal analysis, *Pathogens*, 9 (2020), 276.
[8] H. W. Hethcote; The mathematics of infectious diseases, *SIAM Rev.*, 42 (2000), no. 4, 599–653.
[9] R. P. Hanson; The history of pseudorabies in the United States, *J. Am. Vet. Med. Assoc.*, 124 (1954), no. 925, 259–261.
[10] X. Han, Y. Wang; Research status of pseudorabies (in Chinese), *Animal Husbandry and Veterinary Science and Technology Information*, 11 (2013), no. 1, 17–18.
[11] L. P. Huang, W. Xiao, T. Xu, H. Chen, Z. Y. Jin, Z. G. Zhang, et al.; Miniaturized paper-based smartphone biosensor for differential diagnosis of wild-type pseudorabies virus infection versus vaccination immunization, *Sens. Actuators. B. Chem.*, 327 (2021), 128893.
[12] S. Z. Jin, L. J. Sun, Q. P. Tong, Z. W. Tong; Clinical diagnosis and control of swine pseudorabies (in Chinese), *Shanghai Animal Husbandry and Veterinary Communication*, 12 (2013), no. 5, 90–91.
[13] Z. Jin; *Mathematical Modeling and Research on Dynamics of Infectious Diseases (in Chinese)*. Science Press, 2004.
[14] N. Ketusing, et al.; Evaluation of strategies for the eradication of pseudorabies virus (Aujeszky's Disease) in commercial swine farms in Chiang-Mai and Lampoon Provinces, Thailand, using a simulation disease spread model, *Transbound. Emerg. Dis.*, 61 (2014), no. 2, 169–176.
[15] H. X. Li, R. Y. Liang, Y. L. Pang, L. J. Shi, S. J. Cui, W. C. Lin; Evidence for interspecies transmission route of pseudorabies virus via virally contaminated fomites, *Vet. Microbiol.*, 251 (2020), 108912.
[16] J. P. LaSalle; *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, Siam, Philadelphia, 1976.
[17] J. Li, Z. Ma, F. Zhang; Stability analysis for an epidemic model with stage structure, *Nonlinear Anal.*, 9 (2008), no. 4, 1672–1679.
[18] Y. H. Long, Y. N. Chen; Global stability of a pseudorabies virus model with vertical transmission, *Math. Biosci. Eng.*, 17 (2020), no. 5, 5234–5249.
[19] Y. H. Long, L. Wang; Global dynamics of a delayed two-patch discrete SIR disease model, *Commun. Nonlinear Sci. Numer. Simul.*, 83 (2020), 105117.
[20] M. Q. Lu, S. Y. Qiu, L. L. Zhang, Y. Y. Sun, E. D. Bao, Y. J. Lv; Pseudorabies virus glycoprotein gE suppresses interferon-β production via CREB-binding protein degradation, *Virus. Res.*, 291 (2021), 198220.
[21] T. C. Mettenleiter; Aujeszky, Disease (Pseudorabies) virus: the virus and molecular pathogenesis state of the art, June 1999, *Vet. Res.*, 31 (2000), no. 1, 99–115.
[22] T. C. Mettenleiter; Immunobiology of pseudorabies (Aujeszky's Disease), *Vet. Immunol. Immunopathol.*, 54 (1996), 221–229.
[23] Z. E. Ma, Y. C. Zhou; *Qualitative and Stability Methods of Ordinary Differential Equations (in Chinese)*, Science Press, 2015.
[24] L. E. Pomeranz, A. E. Reynolds, C. J. Hengartner; Molecular biology of pseudorabies virus: impact on neurovirology and veterinary medicine, *Microbiol. Mol. Biol. Rev.*, 69 (2005), 462–500.
[25] F. Rao, P. Mandal, Y. Kang; Complicated endemics of an SIRS model with a generalized incidence under preventive vaccination and treatment controls, *Appl. Math. Model.*, 67 (2019), 38–61.
[26] L. Y. Sun, et al.; Control of swine pseudorabies in China: opportunities and limitations, *Vet. Microbiol.*, 183 (2016), 119–124.
[27] B. K. Tischer, N. Osterrieder; Herpesviruses-a zoonotic threat? *Vet. Microbiol.*, 140 (2010), 266–270.
[28] W. Wang, W. B. Ma, Z. Feng; Global dynamics and travelling waves for a periodic and diffusive chemostat model with two nutrients and one microorganism, *Nonlinearity*, **3**3 (2020), 4338–4380.
[29] X. Wang, C. X. Wu, X. R. Song, H. C. Chen, Z. F. Liu; Comparison of pseudorabies virus China reference strain with emerging variants reveals independent virus evolution within specific geographic regions, *Virol.*, 506 (2017), 92–98.
[30] G. Wittmann, H. Rziha; *Aujeszky's Disease (Pseudorabies) in Pigs*, Springer, Boston, MA, 1989.

[31] J. Zhu, J. Zhou, Z. G. Lin; Dynamics of a diffusive competitive model on a periodically evolving domain, *Electron. J. Differential Equations*, 2020 (2020), no. 86, pp. 1–18.
[32] J. Zimmerman, L. Karriker, A. Ramirez, K. Schwartz, G. Stevenson; *Diseases of Swine 10th Edition*, Wiley, 2019.

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