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Global asymptotic stability in a pseudorabies virus model with age structure

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ABSTRACT

Pseudorabies is a highly contagious disease caused by pseudorabies virus (PRV) or suid herpesvirus 1 (SuHV1), causing significant economic losses to the swine industry in countries where the disease exists. In this paper, we formulate an age structure model of pseudorabies virus that takes into account disease-related mortality and vertical transmission. We find a threshold to determine the stability and existence of the disease. We show that there is always a globally asymptotically stable boundary equilibrium if and only if $\Re_{02} < 1 + \theta$, which means that the disease always exists in piglets and will die out in adult pigs. When $\Re_{02} > 1 + \theta$, the boundary equilibrium is unstable and there exists a unique disease-endemic equilibrium, which is globally asymptotically stable. We give detailed proofs of our theoretical results and numerical examples. Brief concluding remarks are also provided.

1. Introduction

Porcine pseudorabies is an acute infectious disease induced by porcine pseudorabies virus (PRV), also known as Aujeszky's disease (AD), that causes substantial economic losses to the swine industry in countries, where the disease is present and causes a large number of deaths of piglets under two weeks of age (Ketusing et al., 2014; Wittmann & Rziha, 1989; Zimmerman, Karriker, Ramirez et al., 2019; Müller, 2011). Members of the family suidae are the only natural hosts of PRV, although the virus can infect many other mammals, including ruminants, carnivores and rodents (Müller et al., 2011; Pensaert & Kluge, 1989). In pig farms, PRV is transmitted mainly through detoxification of infected pigs to healthy pigs, while the virus is transmitted mainly through nasal secretions, with milk and semen also possible routes of transmission.

The severity of clinical symptoms and mortality rate from PRV infection decreased with age. Newborn piglets unprotected by maternal antibodies develop nervous signs after infection, such as chirping, lethargy, hoarseness, circular movement and swimming posture of limbs, etc., and usually die within 1 ~ 2 days (Cheng, 1999; Nes, 2001). The incidence of PRV in weaned piglets is about 20% ~ 40% and the mortality rate is about 10% ~ 20% (Jin, Sun, Tong et al., 2020). Sows infected with PRV often become sterile and sometimes remain infertile after multiple mating and pregnant sows infected after abortion, weak fetus, stillbirth or mummy fetus, which is mainly stillbirth. Boar infected with PRV, resulting in testicular swelling and atrophy, loss

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of reproductive ability. There is currently no effective treatment for the disease other than early prevention, such as the use of swine serum antibodies.

According to reports, the disease is widespread in pig farms in China, causing huge economic losses to farmers and endangering the healthy development of pig industry. Since the early 1980s, the disease had spread nearly globally. In 1813, Dr. Hildreth in the United States was recorded in a notebook detailing a case of "mad itch" in one of his client's cows which included the cow's rubbing its head, twisting its neck muscles, scratching itself. The cow died a painful death within 12 ~ 14 h of the onset of clinical symptoms (Mayr & Claes, 2010). It was first reported in Hungary by Aladar Aujeszky in 1902 (Aujeszky, 1902), and was known as Aujeszky's disease in 1931 (Hanson, 1954). In 1943, Ray, McNutt and Packer in Iowa described two outbreaks in piglets, with mortality rates reaching 52% and 60% (Mayr & Claes, 2010), respectively. In 1947, Liu first reported pseudorabies in cats (Han & Wang, 2013). In the existing references, most of the researches on porcine pseudorabies are medical, animal husbandry and animal medicine, there are few references about the mathematical models of pseudorabies. In recent years, the health effects of PRV on pigs have become increasingly serious, especially in the actual pig raising process in China (Wu & Ye, 2018). The reference (Nes, 2001) used a stochastic SIR model to estimate a typical infectious pig (R_{ind}) using data from the field and from experiments, which showed that PRV would be eradicated from the Netherlands if the average infection rate of a typical infectious pig (R_{ind}) was lower than that of another pig. The transmission characteristics of pseudorabies were studied in the work of (Ketusing et al., 2014), and the infection situation in different periods was analyzed in (Denzin et al., 2020; Mettenleiter, 1999; Zhai et al., 2019). The reference (Long & Chen, 2020), which considers a class of porcine pseudorabies model with disease-related death and vertical transmission of SEIT, and the reference (Long & Chen, 2021), which considers a swine pseudorabies $S_1I_1S_2I_2R_2S_2$ model with age-structure and disease-related death but vertical propagation is not considered.

In this paper, some improvements are made to the model studied in (Long & Chen, 2021), and the effect of vertical transmission on the dynamic behavior of the disease is discussed. We first give model descriptions in Section 2. We show that the existence and uniqueness of boundary equilibrium and positive equilibrium in Section 3. The local asymptotic stability of equilibria is solved by linearized system and the global asymptotic stability of equilibria is discussed by constructing the Lyapunov function (Long & Wang, 2020) with a method different from (Long & Chen, 2021) in Section 4. We also give some numerical examples to confirm our results in Section 5. Finally, a brief summary is given in Section 6.

2. Model formulation

We establish $S_1I_1S_2I_2R_2S_2$ model of susceptible piglets (S_1), diseased piglets (I_1), susceptible adult pigs (S_2), diseased adult pigs (I_2), recovered adult pigs (R_2), and susceptible adult pigs (S_2) with age structure which classified pigs according to their physiological ages, so as to better reflect individual physiological characteristics and differences affecting disease transmission. The total number of pigs is $N{=}S + I + R$, where $S{=}S_1 + S_2$, $I{=}I_1 + I_2$ and $R{=}R_2$. We let

$$\beta U(N(t))\frac{S(t)}{N(t)}I(t)$$

denote the number of new members infected by all diseased pigs in unit time at time t, where β is the probability of infection per contact, $U(N(t))$ is the number of times a diseased pig contacts other pigs in unit time, also known as the contact rate, which is usually depends on the total number of pigs $N(t)$ and $S(t)/N(t)$ is the proportion of susceptible pigs in the total members. In fact, the scale of the pig farm is limited. We might as well assume that the contact rate is proportional to the total number of pigs, that is, $U(N(t)) = kN(t)$. Under the assumption and after we merge k into the probability of infection per contact and still write it as β, the number of new infected pigs produced in unit time at time t becomes

$$\beta kN(t)\frac{S(t)}{N(t)}I(t) = \beta S(t)I(t).$$

Considering that piglets and adult pigs are raised separately, horizontal transmission is thought to be a form of direct contact between pigs. Hence, the incidence rates of piglets and adult pigs are $\beta_1S_1I_1$ and $\beta_2S_2I_2$, which are form of bilinear incidence rates, where β_1 and β_2 are the infection coefficients of piglets and adult pigs, respectively.

The associations among the five pig groups are illustrated in Fig. 1.

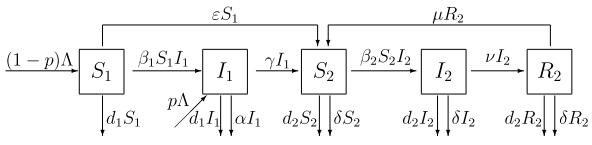

Fig. 1. Progression of infection from the susceptible piglets (S_1) through infected piglets (I_1), susceptible adult pigs (S_2), infected adult piglets (I_2) and recovered adult pigs (R_2) for model (3).

Here Λ is the number of the newborn piglets per unit of time and p is the vertical propagation rate. $p\Lambda$ and $(1-p)\Lambda$ represent the number of newborn diseased piglets and newborn susceptible piglets, respectively. ϵ is the probability that piglets grow up and enter the class S_2. Pigs with symptoms need to be treated appropriately. After treatment, some of the individual symptoms disappeared. Diseased piglets I_1 grow into susceptible adult pigs S_2 with the probability of ν, and diseased adult pigs I_2 enter recovered adult pigs R_2 with the rate of γ. Some recovered adult pigs lose immunity and re-enter S_2 with a probability of μ. In particular, adult pigs are generally recessive infections, and the symptoms of diseased adult pigs are very mild and easy to recover (Sun, 2020). Therefore, the disease-related death rate of adult pigs is not considered in this model. Let d_i, $i = 1, 2$, be the natural death rates of piglets and adult pigs, respectively; α be the disease-related death rate of piglets and δ be the killing rate of adult pigs. Neonatal piglets infected with porcine pseudorabies virus (PRV) cause a large number of deaths, the younger the age of piglets, the higher the morbidity and mortality (Jin et al., 2020), we might as well assume that

$$\gamma < \epsilon \leq \alpha + \gamma. \tag{1}$$

On the other hand, considering the economic benefits, mass slaughter of mature pigs results in $d_2 + \delta \gg d_1$. Then we might as well assume that

$$d_2 + \delta \geq d_1 + \epsilon. \tag{2}$$

Without loss of generality, all of the above parameters are non-negative.

Based on Fig. 1 and the above assumptions, the model we are concerned with is described by the following differential equations with nonnegative initial conditions

$$\begin{cases} \dfrac{dS_1}{dt} = (1-p)\Lambda - \beta_1 S_1 I_1 - (d_1 + \epsilon) S_1, \\ \dfrac{dI_1}{dt} = p\Lambda + \beta_1 S_1 I_1 - (d_1 + \alpha + \gamma) I_1, \\ \dfrac{dS_2}{dt} = \epsilon S_1 + \gamma I_1 - \beta_2 S_2 I_2 - (d_2 + \delta) S_2 + \mu R_2, \\ \dfrac{dI_2}{dt} = \beta_2 S_2 I_2 - (d_2 + \delta + \nu) I_2, \\ \dfrac{dR_2}{dt} = \nu I_2 - (d_2 + \delta + \mu) R_2. \end{cases} \tag{3}$$

Write $N_1 = S_1 + I_1$ and $N_2 = S_2 + I_2 + R_2$. Then $N = N_1 + N_2$ is the total number of pigs. Model (3) can be transformed into the following equivalent model

$$\begin{cases} \dfrac{dI_1}{dt} = p\Lambda + \beta_1 I_1 (N_1 - I_1) - (d_1 + \alpha + \gamma) I_1 := G_1(I_1, N_1, I_2, R_2, N_2), \\ \dfrac{dN_1}{dt} = \Lambda - (d_1 + \epsilon) N_1 - (\alpha + \gamma - \epsilon) I_1 := G_2(I_1, N_1, I_2, R_2, N_2), \\ \dfrac{dI_2}{dt} = \beta_2 I_2 (N_2 - I_2 - R_2) - (d_2 + \delta + \nu) I_2 := G_3(I_1, N_1, I_2, R_2, N_2), \\ \dfrac{dR_2}{dt} = \nu I_2 - (d_2 + \delta + \mu) R_2 := G_4(I_1, N_1, I_2, R_2, N_2), \\ \dfrac{dN_2}{dt} = \epsilon N_1 - (\epsilon - \gamma) I_1 - (d_2 + \delta) N_2 := G_5(I_1, N_1, I_2, R_2, N_2). \end{cases} \tag{4}$$

Here, S_1 and S_2 are replaced by $N_1 - I_1$ and $N_2 - I_2 - R_2$, respectively. Obviously, $G_i : \mathbb{R}^5_+ \to \mathbb{R}$ are C^1 functions for $i = 1, 2, 3, 4, 5$. Moreover, $G_1(0, 0, 0, 0, 0) = p\Lambda$, $G_2(0, 0, 0, 0, 0) = \Lambda$ and $G_i(0, 0, 0, 0, 0) = 0$ for $i = 3, 4, 5$. Hence, we give the initial condition:

$$(I_1(0), N_1(0), I_2(0), R_2(0), N_2(0)) \in \mathbb{R}^5_+.$$

Model (4) admits a unique solution $(I_1(t), N_1(t), I_2(t), R_2(t), N_2(t)) \in \mathbb{R}^5_+$ through ($I_1(0)$, $N_1(0)$, $I_2(0)$, $R_2(0)$, $N_2(0)$) on the existence interval.

Since $d_2 + \delta \gg d_1$, summing up the five equations in model (4), we have

$$\frac{dN}{dt} = \Lambda - d_1 N_1 - (d_2 + \delta) N_2 - \alpha I_1 \leq \Lambda - d_1 N,$$

which leads to

$$N(t) \leq \frac{\Lambda}{d_1} - \frac{\Lambda}{d_1} e^{-d_1 t} + N(0) e^{-d_1 t}.$$

It follows that

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

Moreover, as in (Long & Chen, 2021), we have

$$\begin{aligned} N_1(t) &\leq A - A e^{-(d_1+\epsilon)t} + N_1(0) e^{-(d_1+\epsilon)t}, \\ N_2(t) &\leq B - B e^{-d_2 t} + N_2(0) e^{-d_2 t}, \end{aligned}$$

and hence,

$$\limsup_{t \to \infty} N_1(t) \leq A \quad \text{and} \quad \limsup_{t \to \infty} N_2(t) \leq B,$$

where A and B are defined by

$$A := \frac{\Lambda}{d_1 + \epsilon}, \quad B := \frac{\epsilon \Lambda}{d_2 (d_1 + \epsilon)}. \tag{5}$$

So we denote the positive invariant set

$$D = \left\{ (I_1, N_1, I_2, R_2, N_2), 0 \leq I_1 \leq N_1 \leq A, 0 \leq I_2 + R_2 \leq N_2 \leq B, 0 < N_1 + N_2 \leq \frac{\Lambda}{d_1} \right\}.$$

3. The existence of equilibria in model (4)

According to (LaSalle, 1976; Li et al., 2008; Long & Chen, 2021; Van Den Driessche, 2002; Wang et al., 2020), the basic reproduction number of model (4) is

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

where

$$\Re_{01} = \frac{\beta_1 \Lambda}{(d_1 + \epsilon)(d_1 + \alpha + \gamma)}, \quad \Re_{02} = \frac{\epsilon \beta_2 \Lambda}{(d_1 + \epsilon)(d_2 + \delta)(d_2 + \delta + \nu)}. \tag{6}$$

Let the right-hand side of model (4) be equal to zero. Then equilibria of (4) satisfy the following equations

$$\begin{cases} p\Lambda + \beta_1 I_1 (N_1 - I_1) - (d_1 + \alpha + \gamma) I_1 = 0, \\ \Lambda - (d_1 + \epsilon) N_1 - (\alpha + \gamma - \epsilon) I_1 = 0, \\ \beta_2 I_2 (N_2 - I_2 - R_2) - (d_2 + \delta + \nu) I_2 = 0, \\ \nu I_2 - (d_2 + \delta + \mu) R_2 = 0, \\ \epsilon N_1 - (\epsilon - \gamma) I_1 - (d_2 + \delta) N_2 = 0. \end{cases} \tag{7}$$

In (Long & Chen, 2021), the authors studied the critical situation of $p = 0$ and found that model (4) has a disease-free equilibrium, two boundary equilibria and a disease-endemic equilibrium, and gave the global dynamic analysis as follows.

Theorem 3.1. (Theorem 3.1-3.4 in Long and Chen (2021))
Let

$$\varepsilon := \frac{((\epsilon - \gamma) d_1 + \epsilon \alpha)(d_1 + \alpha + \gamma)(\Re_{01} - 1)}{\gamma (d_1 + \epsilon) \beta_1 \Lambda + (d_1 + \epsilon)(d_1 + \alpha + \gamma)((\epsilon - \gamma) d_1 + \epsilon \alpha)} > 0.$$

(1) *The disease-free equilibrium P_0 is globally asymptotically stable if $\Re_0 < 1$ and is unstable if $\Re_0 > 1$;*
(2) *If $\Re_{02} > 1$, then model (4) has a boundary equilibrium P_{01}. Further, if $\Re_{01} < 1$, then P_{01} is globally asymptotically stable and if $\Re_{01} > 1$, then P_{01} is unstable;*

(3) *If $\Re_{01}>1$, then model (4) has another boundary equilibrium P_{02}. Further, if $\Re_{02}<1+\varepsilon$, then P_{02} is globally asymptotically stable and if $\Re_{02}>1+\varepsilon$, then P_{02} is unstable*;
(4) *If $\Re_{01}>1$ and $\Re_{02}>1+\varepsilon$, in addition to the disease-free equilibrium P_0 and the boundary equilibria P_{01} and P_{02}, model (4) also has a unique disease-endemic equilibrium P^*, which is globally asymptotically stable.*

In this paper, we will discuss the dynamic behavior of model (4) for $p\in(0,1)$. Let

$$I:=\frac{(d_1+\epsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)+\sqrt{\Delta}}{2\beta_1(d_1+\alpha+\gamma)} \quad \text{and} \quad \theta:=\frac{\beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha]I}{(d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu)}, \tag{8}$$

where

$$\Delta=\sqrt{[(d_1+\epsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)]^2+4p\beta_1\Lambda(d_1+\epsilon)(d_1+\alpha+\gamma)}.$$

We establish two lemmas for the existence of equilibria for model (4) as follows.

Lemma 3.2. *Model (4) has no disease-free equilibria, and there is always a boundary equilibrium $E_1=(I_1^1,N_1^1,0,0,N_2^1)$, where*

$$I_1^1=\frac{(d_1+\epsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)+\sqrt{\Delta}}{2\beta_1(d_1+\alpha+\gamma)},\ N_1^1=\frac{\Lambda-(\alpha+\gamma-\epsilon)I_1^1}{d_1+\epsilon},\ N_2^1=\frac{\epsilon(N_1^1-I_1^1)+\gamma I_1^1}{d_2+\delta}.$$

Proof. Set $I_2^1=R_2^1=0$. The solution of (7) satisfies

$$\begin{cases} p\Lambda+\beta_1 I_1^1(N_1^1-I_1^1)-(d_1+\alpha+\gamma)I_1^1=0,\\ \Lambda-(d_1+\epsilon)N_1^1-(\alpha+\gamma-\epsilon)I_1^1=0,\\ \epsilon N_1^1-(\epsilon-\gamma)I_1^1-(d_2+\delta)N_2^1=0. \end{cases} \tag{9}$$

From the first and second equations of (9), we obtain

$$N_1^1=\frac{\beta_1(I_1^1)^2+(d_1+\alpha+\gamma)I_1^1-p\Lambda}{\beta_1 I_1^1} \quad \text{and} \quad N_1^1=\frac{\Lambda-(\alpha+\gamma-\epsilon)I_1^1}{d_1+\epsilon}\le A, \tag{10}$$

respectively, which follows that

$$\frac{(d_1+\alpha+\gamma)I_1^1-p\Lambda}{\beta_1 I_1^1}=\frac{\Lambda-(d_1+\alpha+\gamma)I_1^1}{d_1+\epsilon},$$

or equivalently,

$$g(I_1^1):=\beta_1(d_1+\alpha+\gamma)(I_1^1)^2-(d_1+\epsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)I_1^1-p\Lambda(d_1+\epsilon)=0, \tag{11}$$

whose discriminant is

$$\Delta=[(d_1+\epsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)]^2+4p\beta_1\Lambda(d_1+\epsilon)(d_1+\alpha+\gamma)>0.$$

It is obvious that equation (11) has a unique positive solution

$$I_1^1=\frac{(d_1+\epsilon)(d_1+\alpha+\gamma)(\Re_{01}-1)+\sqrt{\Delta}}{2\beta_1(d_1+\alpha+\gamma)}. \tag{12}$$

Since

$$g\left(\frac{\Lambda}{d_1+\alpha+\gamma}\right)=(1-p)(d_1+\epsilon)\Lambda>0 \quad \text{for} \quad p\in(0,1),$$

we have

$$I_1^1\in\left(0,\frac{\Lambda}{d_1+\alpha+\gamma}\right)\subset(0,A].$$

Hence, by (10), we obtain

$$N_1^1 = \frac{\Lambda - (d_1 + \alpha + \gamma)I_1^1}{d_1 + \epsilon} + I_1^1 \in (0, A]. \tag{13}$$

Further, it follows from the third equation of (9) that

$$N_2^1 = \frac{\epsilon(N_1^1 - I_1^1) + \gamma I_1^1}{d_2 + \delta} \in (0, B],$$

since

$$N_2^1 \le \frac{\epsilon N_1^1}{d_2 + \delta} \le \frac{\epsilon A}{d_2 + \delta} = \frac{\epsilon \Lambda}{(d_1 + \epsilon)(d_2 + \delta)} \le B.$$

This ends the proof of Lemma 3.2.

Lemma 3.3. *Suppose $\Re_{02} > 1 + \theta$. Model (4) admits a unique disease-endemic equilibrium $E^* = (I_1^*, N_1^*, I_2^*, R_2^*, N_2^*)$, where*

$$I_1^* = \frac{(d_1 + \epsilon)(d_1 + \alpha + \gamma)(\Re_{01} - 1) + \sqrt{\Delta}}{2\beta_1(d_1 + \alpha + \gamma)},\ N_1^* = \frac{\Lambda - (\alpha + \gamma - \epsilon)I_1^*}{d_1 + \epsilon},$$

$$I_2^* = \frac{(d_2 + \delta + \mu)(d_2 + \delta + \nu)[\Re_{02} - (1 + \theta)]}{\beta_2(d_2 + \delta + \mu + \nu)},\ R_2^* = \frac{\nu I_2^*}{d_2 + \delta + \mu},\ N_2^* = \frac{\epsilon N_1^* - (\epsilon - \gamma)I_1^*}{d_2 + \delta}.$$

Proof. The disease-endemic equilibrium E^* satisfies

$$\begin{cases} p\Lambda + \beta_1 I_1^*(N_1^* - I_1^*) - (d_1 + \alpha + \gamma)I_1^* = 0, \\ \Lambda - (d_1 + \epsilon)N_1^* - (\alpha + \gamma - \epsilon)I_1^* = 0, \\ \beta_2 I_2^*(N_2^* - I_2^* - R_2^*) - (d_2 + \delta + \nu)I_2^* = 0, \\ \nu I_2^* - (d_2 + \delta + \mu)R_2^* = 0, \\ \epsilon N_1^* - (\epsilon - \gamma)I_1^* - (d_2 + \delta)N_2^* = 0. \end{cases} \tag{14}$$

Following from a similar procedure done in Lemma 3.2, we can calculate directly that

$$I_1^* = \frac{(d_1 + \epsilon)(d_1 + \alpha + \gamma)(\Re_{01} - 1) + \sqrt{\Delta}}{2\beta_1(d_1 + \alpha + \gamma)} \in (0, A],\ N_1^* = \frac{\Lambda - (\alpha + \gamma - \epsilon)I_1^*}{d_1 + \epsilon} \in (0, A],$$

and

$$N_2^* = \frac{\epsilon N_1^* - (\epsilon - \gamma)I_1^*}{d_2 + \delta} \in (0, B]. \tag{15}$$

From the fourth equation of (14), we get

$$R_2^* = \frac{\nu I_2^*}{d_2 + \delta + \mu}. \tag{16}$$

Substituting (16) into the third equation of (14) then yields

$$N_2^* = \frac{d_2 + \delta + \nu}{\beta_2} + \frac{(d_2 + \delta + \mu + \nu)I_2^*}{d_2 + \delta + \mu}, \tag{17}$$

which, by (15), leads to

$$\frac{d_2 + \delta + \nu}{\beta_2} + \frac{(d_2 + \delta + \mu + \nu)I_2^*}{d_2 + \delta + \mu} = \frac{\epsilon N_1^* - (\epsilon - \gamma)I_1^*}{d_2 + \delta}. \tag{18}$$

Thus, we have

$$
\begin{aligned}
I_2^* &= \frac{d_2+\delta+\mu}{d_2+\delta+\mu+\nu}\cdot\frac{\beta_2\left[\epsilon N_1^* - (\epsilon-\gamma)I_1^*\right] - (d_2+\delta)(d_2+\delta+\nu)}{\beta_2(d_2+\delta)} \\
&= \frac{d_2+\delta+\mu}{d_2+\delta+\mu+\nu}\cdot\frac{\beta_2\left[\epsilon\Lambda - \epsilon(d_1+\alpha+\gamma)I_1^* + \gamma(d_1+\epsilon)I_1^*\right] - (d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu)}{\beta_2(d_1+\epsilon)(d_2+\delta)} \\
&= \frac{d_2+\delta+\mu}{d_2+\delta+\mu+\nu}\cdot\frac{\epsilon\beta_2\Lambda - (d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu) - \beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha]I_1^*}{\beta_2(d_1+\epsilon)(d_2+\delta)} \\
&= \frac{d_2+\delta+\mu}{d_2+\delta+\mu+\nu}\cdot\frac{(d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu)(\Re_{02}-1) - \beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha)\,]I_1^*}{\beta_2(d_1+\epsilon)(d_2+\delta)} \\
&= \frac{(d_2+\delta+\mu)(d_2+\delta+\nu)}{\beta_2(d_2+\delta+\mu+\nu)}\left(\Re_{02} - 1 - \frac{\beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha)\,]I_1^*}{(d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu)}\right) \\
&= \frac{(d_2+\delta+\mu)(d_2+\delta+\nu)[\Re_{02}-(1+\theta)]}{\beta_2(d_2+\delta+\mu+\nu)},
\end{aligned}
$$

and hence $I_2^* > 0$ since $\Re_{02} > 1+\theta$. Further, it follows from (17) that $I_2^* \le N_2^* \le B$. Then, by (16), we have $R_2^* \in (0, B]$ for $\Re_{02} > 1+\theta$.

To sum up, when $\Re_{02} > 1+\theta$, model (4) admits a unique disease-endemic equilibrium E^* and thus we complete the proof of Lemma 3.3.

4. Dynamics of equilibria E_1 and E^*

In this section, we study the stability of the boundary equilibrium $E_1 = (I_1^1, N_1^1, 0, 0, N_2^1)$ and the disease-endemic equilibrium $E^* = (I_1^*, N_1^*, I_2^*, R_2^*, N_2^*)$. We have the following results.

Theorem 4.1. *When $\Re_{02} < 1+\theta$, the boundary equilibrium E_1 is globally asymptotically stable, while when $\Re_{02} > 1+\theta$, E_1 is unstable.*

Proof. We first discuss the stability of E_1. The characteristic equation of the linearized system of model (4) at E_1 is

$$
H(\lambda) := \det(\lambda I - J(E_1)) = (\lambda + d_2 + \delta)(\lambda + d_2 + \delta + \mu)(\lambda + d_2 + \delta + \nu - \beta_2 N_2^1)(\lambda^2 + a_1\lambda + a_2) = 0,
$$

where

$$
\begin{aligned}
a_1 &= d_1 + \epsilon + \beta_1 I_1^1 + d_1 + \alpha + \gamma - \beta_1(N_1^1 - I_1^1), \\
a_2 &= \beta_1 I_1^1(d_1+\alpha+\gamma) + (d_1+\epsilon)\Big(d_1+\alpha+\gamma-\beta_1(N_1^1-I_1^1)\Big).
\end{aligned}
$$

From the first equation of (9), we have

$$
\Big(d_1+\alpha+\gamma-\beta_1(N_1^1-I_1^1)\Big)I_1^1 = p\Lambda > 0. \tag{19}
$$

which implies that $a_1 > 0$ and $a_2 > 0$.

Let $\lambda_i (i = 1, 2, 3, 4, 5)$ be the eigenvalues of $J(E_1)$, which satisfy

$$
\lambda_1 = -(d_2+\delta) < 0, \quad \lambda_2 = -(d_2+\delta+\mu) < 0, \quad \lambda_3 = \beta_2 N_2^1 - (d_2+\delta+\nu) \tag{20}
$$

and

$$
\lambda_4 + \lambda_5 = -a_1 < 0, \quad \lambda_4 \cdot \lambda_5 = a_2 > 0. \tag{21}
$$

(21) implies that $\lambda_4 < 0$ and $\lambda_5 < 0$. Moreover, it can be calculated directly that

$$\begin{aligned}
\lambda_3 &= \beta_2 N_2^1 - (d_2+\delta+\nu)\\
&= \frac{\epsilon\beta_2\Lambda - \beta_2[\epsilon(d_1+\alpha+\gamma)-\gamma(d_1+\epsilon)]I_1^1}{(d_2+\delta)(d_1+\epsilon)} - (d_2+\delta+\nu)\\
&= \frac{\epsilon\beta_2\Lambda - (d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu) - \beta_2[\epsilon(d_1+\alpha+\gamma)-\gamma(d_1+\epsilon)]I_1^1}{(d_1+\epsilon)(d_2+\delta)}\\
&= \frac{(d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu)(\Re_{02}-1) - \beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha]I_1^1}{(d_1+\epsilon)(d_2+\delta)}\\
&= (d_2+\delta+\nu)(\Re_{02}-1) - \frac{\beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha]I_1^1}{(d_1+\epsilon)(d_2+\delta)}\\
&= (d_2+\delta+\nu)\left(\Re_{02} - 1 - \frac{\beta_2[(\epsilon-\gamma)d_1+\epsilon\alpha]I_1^1}{(d_1+\epsilon)(d_2+\delta)(d_2+\delta+\nu)}\right)\\
&= (d_2+\delta+\nu)[\Re_{02}-(1+\theta)].
\end{aligned} \tag{22}$$

Hence, we have $\lambda_3 < 0$ for $\Re_{02} < 1+\theta$. By combining (20), (21) and (22), all eigenvalues $\lambda_i (i = 1, 2, 3, 4, 5)$ are negative. Consequently, E_1 is locally asymptotically stable on D. Whereas, if $\Re_{02} > 1+\theta$, then $\lambda_3 > 0$, which means that E_1 is unstable.

We then discuss the attractivity of the boundary equilibrium E_1. Due to the first two equations of model (4) do not contain I_2, R_2 and N_2, we can consider the subsystem of (4) with respect to I_1 and N_1, that is

$$\begin{cases}
\dfrac{dI_1}{dt} = p\Lambda + \beta_1 I_1(N_1 - I_1) - (d_1+\alpha+\gamma)I_1,\\
\dfrac{dN_1}{dt} = \Lambda - (d_1+\epsilon)N_1 - (\alpha+\gamma-\epsilon)I_1.
\end{cases} \tag{23}$$

The second equation of (23) is transformed into an integral equation

$$N_1(t) = A\left(1 - e^{-(d_1+\epsilon)t}\right) + N_1(0)e^{-(d_1+\epsilon)t} - (\alpha+\gamma-\epsilon)\int_0^t e^{-(d_1+\epsilon)(t-s)}I_1(s)ds.$$

By substituting the above equation into the first equation of (23), the following integro-differential equation can be obtained

$$\begin{aligned}
I_1'(t) = p\Lambda + \beta_1 I_1(t)\Big[&A\left(1 - e^{-(d_1+\epsilon)t}\right) + N_1(0)e^{-(d_1+\epsilon)t}\\
&- (\alpha+\gamma-\epsilon)\int_0^t e^{-(d_1+\epsilon)(t-s)}I_1(s)ds \quad - I_1(t)\Big] - (d_1+\alpha+\gamma)I_1(t).
\end{aligned} \tag{24}$$

Let $I_1(t) = I_1^1 e^{y(t)}$. Then we have

$$I_1'(t) = I_1^1 e^{y(t)} y'(t),$$

and

$$\begin{aligned}
y'(t) = \frac{I_1'(t)}{I_1(t)} = \frac{p\Lambda}{I_1^1}e^{-y(t)} + \beta_1\Bigg[&A\left(1 - e^{-(d_1+\epsilon)t}\right) + N_1(0)e^{-(d_1+\epsilon)t} - I_1^1 e^{y(t)}\\
&-(\alpha+\gamma-\epsilon)I_1^1\int_0^t e^{-(d_1+\epsilon)(t-s)}e^{y(s)}ds\Bigg] - \left(d_1+\alpha+\gamma\right).
\end{aligned} \tag{25}$$

We next prove that $y(t) \to 0$ as $t \to \infty$. To this purpose, we further define

$$g(y) = e^y - 1 \quad \text{and} \quad a(s) = \begin{cases} 0, & s \le 0, \\ \beta_1 I_1^1 \left[1 + (\alpha + \gamma - \epsilon) \int_0^s e^{-(d_1+\epsilon)u} du\right], & s > 0. \end{cases}$$

Obviously, when $s = 0$, $\beta_1 I_1^1$ is the jump point of $a(s)$. When $s > 0$,

$$a'(s) = \beta_1 I_1^1 (\alpha + \gamma - \epsilon) e^{-(d_1+\epsilon)s},$$

and

$$\int_0^t g[y(t-s)]da(s) = \beta_1 I_1^1 e^{y(t)} - \beta_1 I_1^1 + \beta_1 I_1^1 (\alpha + \gamma - \epsilon) \int_0^t e^{y(t-s)} e^{-(d_1+\epsilon)s} ds - \frac{\beta_1 I_1^1 (\alpha + \gamma - \epsilon)}{d_1 + \epsilon} \left(1 - e^{-(d_1+\epsilon)t}\right).$$

On the other hand, we denote

$$h(y) = \frac{p\Lambda}{I_1^1}(1 - e^{-y}),$$

and

$$\begin{aligned} f(t) &= -\beta_1 I_1^1 + \frac{\beta_1}{d_1 + \epsilon}\left[1 - e^{-(d_1+\epsilon)t}\right]\left[\Lambda - (\alpha + \gamma - \epsilon) I_1^1\right] \\ &\quad + \beta_1 N_1(0) e^{-(d_1+\epsilon)t} - (d_1 + \alpha + \gamma) + \frac{p\Lambda}{I_1^1} \\ &= \beta_1 e^{-(d_1+\epsilon)t}\left[N_1(0) - \frac{\Lambda - (\alpha + \gamma - \epsilon) I_1^1}{d_1 + \epsilon}\right] \\ &\quad + \left[\frac{p\Lambda}{I_1^1} - \beta_1 I_1^1 + \frac{\beta_1 \Lambda - \beta_1 (\alpha + \gamma - \epsilon) I_1^1}{d_1 + \epsilon} - (d_1 + \alpha + \gamma)\right], \\ &= \beta_1 e^{-(d_1+\epsilon)t}\left[N_1(0) - \frac{\Lambda - (\alpha + \gamma - \epsilon) I_1^1}{d_1 + \epsilon}\right] \\ &\quad + \left[\frac{p\Lambda}{I_1^1} + \beta_1 A - \frac{\beta_1 (d_1 + \alpha + \gamma) I_1^1}{d_1 + \epsilon} - (d_1 + \alpha + \gamma)\right]. \end{aligned}$$

From (13), we have

$$\frac{p\Lambda}{I_1^1} + \beta_1 A - \frac{\beta_1 (d_1 + \alpha + \gamma) I_1^1}{d_1 + \epsilon} - (d_1 + \alpha + \gamma) = 0, \tag{26}$$

which leads to

$$f(t) = \beta_1 e^{-(d_1+\epsilon)t}\left[N_1(0) - \frac{\Lambda - (\alpha + \gamma - \epsilon) I_1^1}{d_1 + \epsilon}\right]. \tag{27}$$

With the help of above preparations, the differential integral equation (25) can be reduced to

$$
\begin{aligned}
y'(t) &= \frac{p\Lambda}{I_1^1}e^{-y(t)} + \beta_1\left[A\left(1-e^{-(d_1+\epsilon)t}\right) + N_1(0)e^{-(d_1+\epsilon)t} - I_1^1 e^{y(t)}\right. \\
&\quad \left. - (\alpha+\gamma-\epsilon)I_1^1\int_0^t e^{-(d_1+\epsilon)(t-s)}e^{y(s)}ds\right] - (d_1+\alpha+\gamma) \\
&= -\left[\beta_1 I_1^1 e^{y(t)} - \beta_1 I_1^1 + \beta_1 I_1^1(\alpha+\gamma-\epsilon)\int_0^t e^{y(t-s)}e^{-(d_1+\epsilon)s}ds\right. \\
&\quad \left. - \frac{\beta_1 I_1^1(\alpha+\gamma-\epsilon)}{d_1+\epsilon}\left(1-e^{-(d_1+\epsilon)t}\right)\right] - \frac{p\Lambda}{I_1^1}(1-e^{-y(t)}) \\
&\quad + \beta_1 e^{-(d_1+\epsilon)t}\left[N_1(0) - \frac{\Lambda-(\alpha+\gamma-\epsilon)I_1^1}{d_1+\epsilon}\right] \\
&\quad + \left[\frac{p\Lambda}{I_1^1} + \beta_1 A - \frac{\beta_1(d_1+\alpha+\gamma)I_1^1}{d_1+\epsilon} - (d_1+\alpha+\gamma)\right] \\
&= -\int_0^t g[y(t-s)]da(s) - h(y) + f(t).
\end{aligned} \tag{28}
$$

As in (Ma et al., 2004), we have $a(s)$ is strong positive type. It is clear that $g(y)$ is a continuous function and $\lim_{|y|\to\infty}\int_0^y g(y)dy = \infty$, then $h'(y)$ is also a continuous function. Moreover,

$$
g(y)h(y) = \frac{p\Lambda}{I_1^1}(e^y-1)(1-e^{-y}) = \frac{p\Lambda}{I_1^1}(e^y+e^{-y}-2) \geq 0, \quad \text{for all} \quad y\in\mathbb{R}^n, \tag{29}
$$

$f(t), f'(t) \in L^2(0,+\infty)$. According to (Gripenberg et al., 2013)(page 566, Theorem 18.2.3) and (Ma et al., 2004)(page 131), every bounded solution $y(t)$ of equation (28) satisfies

$$\lim_{t\to\infty} g(y(t)) = 0,$$

and hence we have

$$\lim_{t\to\infty} y(t) = 0.$$

Thus, the equilibrium I_1^1 of (24) is globally attractive. Moreover, equation (23) is equivalent to equation (24), so the equilibrium (I_1^1, N_1^1) of (23) is globally attractive. Combined with the locally asymptotically stable of (I_1^1, N_1^1), we obtain that (I_1^1, N_1^1) of (23) is globally asymptotically stable. Hence, we have

$$\lim_{t\to\infty} N_1(t) = N_1^1, \quad \lim_{t\to\infty} I_1(t) = I_1^1.$$

Similarly, the last equation of model (4) can be transformed into an integral equation

$$N_2(t) = N_2(0)e^{-(d_2+\delta)t} + \epsilon\int_0^t e^{-(d_2+\delta)(t-s)}N_1(s)ds - (\epsilon-\gamma)\int_0^t e^{-(d_2+\delta)(t-s)}I_1(s)ds.$$

Then we have

$$\lim_{t\to\infty} N_2(t) = \lim_{t\to\infty}\frac{\epsilon N_1(t) - (\epsilon-\gamma)I_1(t)}{d_2+\delta} = \frac{\epsilon N_1^1 - (\epsilon-\gamma)I_1^1}{d_2+\delta} = N_2^1.$$

Further, when $I_2(0)=0$ and $R_2(0)=0$, it is obvious that $I_2(t)=R_2(t)=0$ for any $t>0$. As a whole, it can be concluded that the boundary equilibrium E_1 is globally attractive.

Combined with the local stability and global attractivity of E_1, we conclude that E_1 is globally asymptotically stable for $\Re_{02}<1+\theta$, while it is unstable for $\Re_{02}>1+\theta$. The proof is complete.

Theorem 4.2. *When $\Re_{02}>1+\theta$, the unique disease-endemic equilibrium E^* is globally asymptotically stable*.**Proof**. We first prove that the local asymptotically stable of disease-endemic equilibrium E^*. The characteristic equation of the linearized system of model (4) at E^* is

$$L(\tau):=\det(\tau I-J(P^*))=(\tau+d_2+\delta)(\tau^2+b_1\tau+b_2)(\tau^2+c_1\tau+c_2)=0,$$

where

$$\begin{aligned} b_1 &= d_2+\delta+\mu+\beta_2 I_2^*>0,\\ b_2 &= (d_2+\delta+\mu+\nu)\beta_2 I_2^*>0,\\ c_1 &= d_1+\epsilon+\beta_1 I_1^*+d_1+\alpha+\gamma-\beta_1(N_1^*-I_1^*),\\ c_2 &= (d_1+\epsilon)\big(d_1+\alpha+\gamma-\beta_1(N_1^*-I_1^*)\big)+(d_1+\alpha+\gamma)\beta_1 I_1^*. \end{aligned}$$

From the first equation of (14), we have

$$d_1+\alpha+\gamma-\beta_1(N_1^*-I_1^*)=\frac{p\Lambda}{I_1^*}>0, \tag{30}$$

which implies that $c_1>0$ and $c_2>0$, and hence

$$\tau_1=-(d_2+\delta)<0,\quad \tau_2+\tau_3=-b_1<0,\quad \tau_2\cdot\tau_3=b_2>0, \tag{31}$$

and

$$\tau_4+\tau_5=-c_1<0,\quad \tau_4\cdot\tau_5=c_2>0. \tag{32}$$

Clearly, (31) and (32) ensure all eigenvalues $\tau_i(i=1,2,3,4,5)$ are negative, which implies that the disease-endemic equilibrium E^* is local asymptotically stable.

Next, we prove the disease-endemic equilibrium E^* is globally asymptotically stable. Since the first two equations of (14) do not contain I_2, R_2 and N_2, we consider the sub-equation with respect to I_1 and N_1, that is

$$\begin{cases} p\Lambda=-\beta_1 I_1^*(N_1^*-I_1^*)+(d_1+\alpha+\gamma)I_1^*,\\ \Lambda=(d_1+\epsilon)N_1^*+(\alpha+\gamma-\epsilon)I_1^*. \end{cases} \tag{33}$$

Substituting (33) into (23) yields

$$\begin{cases} \dfrac{dI_1}{dt}=\beta_1 I_1\big[(N_1-N_1^*)-(I_1-I_1^*)\big]-\big[(d_1+\alpha+\gamma)-\beta_1(N_1^*-I_1^*)\big](I_1-I_1^*),\\ \dfrac{dN_1}{dt}=-(d_1+\epsilon)(N_1-N_1^*)-(\alpha+\gamma-\epsilon)(I_1-I_1^*). \end{cases} \tag{34}$$

We then show that (I_1^*,N_1^*) of (34) is globally asymptotically stable. To this end, we construct the Lyapunov function

$$V(I_1^*,N_1^*)=(\alpha+\gamma-\epsilon)\left(I_1-I_1^*-I_1^*\ln\frac{I_1}{I_1^*}\right)+\frac{\beta_1}{2}(N_1-N_1^*)^2.$$

Then the derivative of $V(I_1^*,N_1^*)$ along the trajectory of (34) is

$$\begin{aligned}\frac{dV}{dt} &= (\alpha+\gamma-\epsilon)\left(1-\frac{I_1^*}{I_1}\right)\frac{dI_1}{dt}+\beta_1(N_1-N_1^*)\frac{dN_1}{dt}\\ &= \beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)\left[(N_1-N_1^*)-(I_1-I_1^*)\right]\\ &\quad -\frac{\alpha+\gamma-\epsilon}{I_1}\left[(d_1+\alpha+\gamma)-\beta_1(N_1^*-I_1^*)\right](I_1-I_1^*)^2\\ &\quad -\beta_1(d_1+\epsilon)(N_1-N_1^*)^2-\beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)(N_1-N_1^*)\\ &= \beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)(N_1-N_1^*)-\beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)^2\\ &\quad -\frac{\alpha+\gamma-\epsilon}{I_1}\left[(d_1+\alpha+\gamma)-\beta_1(N_1^*-I_1^*)\right](I_1-I_1^*)^2\\ &\quad -\beta_1(d_1+\epsilon)(N_1-N_1^*)^2-\beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)(N_1-N_1^*)\\ &= -\beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)^2-\beta_1(d_1+\epsilon)(N_1-N_1^*)^2\\ &\quad -\frac{\alpha+\gamma-\epsilon}{I_1}\left[(d_1+\alpha+\gamma)-\beta_1(N_1^*-I_1^*)\right](I_1-I_1^*)^2.\end{aligned}$$

From (1) and (30), we have $\alpha+\gamma-\epsilon\geq 0$ and $d_1+\alpha+\gamma-\beta_1(N_1^*-I_1^*)=\frac{p\Lambda}{I_1^*}>0$. Then,

$$\begin{aligned}\frac{dV}{dt} &= -\beta_1(\alpha+\gamma-\epsilon)(I_1-I_1^*)^2-\beta_1(d_1+\epsilon)(N_1-N_1^*)^2-\frac{(\alpha+\gamma-\epsilon)p\Lambda}{I_1I_1^*}(I_1-I_1^*)^2\\ &= -(\alpha+\gamma-\epsilon)\left(\beta_1+\frac{p\Lambda}{I_1I_1^*}\right)(I_1-I_1^*)^2-\beta_1(d_1+\epsilon)(N_1-N_1^*)^2\leq 0,\end{aligned}$$

and $dV/dt=0$ if and only if $I_1=I_1^*$ and $N_1=N_1^*$. It's easy to see that the largest invariant subset of $dV/dt=0$ is (I_1^*,N_1^*). Then according to the LaSalle's invariance principle (Ma & Zhou, 2015), $(I_1^*,N_1^*)\in\mathbb{R}_+^2$ is globally asymptotically stable. Hence, we have

$$\lim_{t\to\infty}I_1(t)=I_1^*,\quad \lim_{t\to\infty}N_1(t)=N_1^*.$$

Note that the last equation of model (4) can be transformed into an integral equation

$$N_2(t)=N_2(0)e^{-(d_2+\delta)t}+\epsilon\int_0^t e^{-(d_2+\delta)(t-s)}N_1(s)ds-(\epsilon-\gamma)\int_0^t e^{-(d_2+\delta)(t-s)}I_1(s)ds,$$

which implies that

$$\lim_{t\to\infty}N_2(t)=\lim_{t\to\infty}\frac{\epsilon N_1(t)-(\epsilon-\gamma)I_1(t)}{d_2+\delta}=\frac{\epsilon N_1^*-(\epsilon-\gamma)I_1^*}{d_2+\delta}=N_2^*.$$

Finally, we prove that $\lim_{t\to\infty}I_2(t)=I_2^*$ and $\lim_{t\to\infty}R_2(t)=R_2^*$. In fact, from (18), we have

$$\frac{d_2+\delta+\nu}{\beta_2}+\frac{(d_2+\delta+\mu+\nu)I_2(t)}{d_2+\delta+\mu}=\frac{\epsilon N_1(t)-(\epsilon-\gamma)I_1(t)}{d_2+\delta}. \tag{35}$$

Taking the limit on both sides of (35) yields

$$\lim_{t\to\infty}\left[\frac{d_2+\delta+\nu}{\beta_2}+\frac{(d_2+\delta+\mu+\nu)I_2(t)}{d_2+\delta+\mu}\right]=\frac{\epsilon N_1^*-(\epsilon-\gamma)I_1^*}{d_2+\delta}=\frac{d_2+\delta+\nu}{\beta_2}+\frac{(d_2+\delta+\mu+\nu)I_2^*}{d_2+\delta+\mu},$$

which implies that

$$\lim_{t\to\infty}I_2(t)=I_2^*.$$

Similarly, from the penultimate equation of model (4), we get

$$R_2(t) = R_2(0)e^{-(d_2+\delta+\mu)t} + \nu \int_0^t e^{-(d_2+\delta+\mu)(t-s)} I_2(s)ds.$$

Then we have

$$\lim_{t\to\infty} R_2(t) = \lim_{t\to\infty} \frac{\nu I_2(t)}{d_2+\delta+\mu} = \frac{\nu I_2^*}{d_2+\delta+\mu} = R_2^*.$$

Therefore, it can be concluded that the diseases-endemic equilibrium E^* is globally attractive. Together with the local asymptotic stability of E^*, we prove that E^* is globally asymptotically stable for $\Re_{02} > 1+\theta$. Thus we complete the proof of Theorem 4.2.

5. Numerical simulation

Literatures (Jin et al., 2020; Song et al., 2020) indicate that the mortality rate of pseudorabies is between 10% ~ 50%. However, for other parameters, due to the different scale of pig farms and the different probability of positive pseudorabies in pigs in different regions, it is difficult to count their range. We can only analyze and estimate the values of these parameters according to existing data and actual conditions. In this section, we properly select the range of parameters, make them meet our threshold conditions, and we provide examples below to confirm the validity of our results theoretically.

Example 5.1.

$$\begin{aligned} &\Lambda = 50, \quad \beta_1 = 0.09, \quad \beta_2 = 0.05, \quad d_1 = 0.04, \quad d_2 = 0.03, \quad \alpha = 0.35, \\ &\gamma = 0.011, \quad \epsilon = 0.33, \quad \nu = 0.34, \quad p = 0.16, \quad \delta = 0.55, \quad \mu = 0.04. \end{aligned} \tag{36}$$

From (6) and (8), we have $\Re_{02} \approx 4.1787$ and $\theta \approx 3.9386$, which satisfies $\Re_{02} < 1+\theta$. We find that no matter what the initial value is, the solution of model (4) will eventually tend to the boundary equilibrium E_1, which implies that E_1 is globally asymptotically stable. We choose four different initial values to verify our conclusion, and the initial values are as follows:

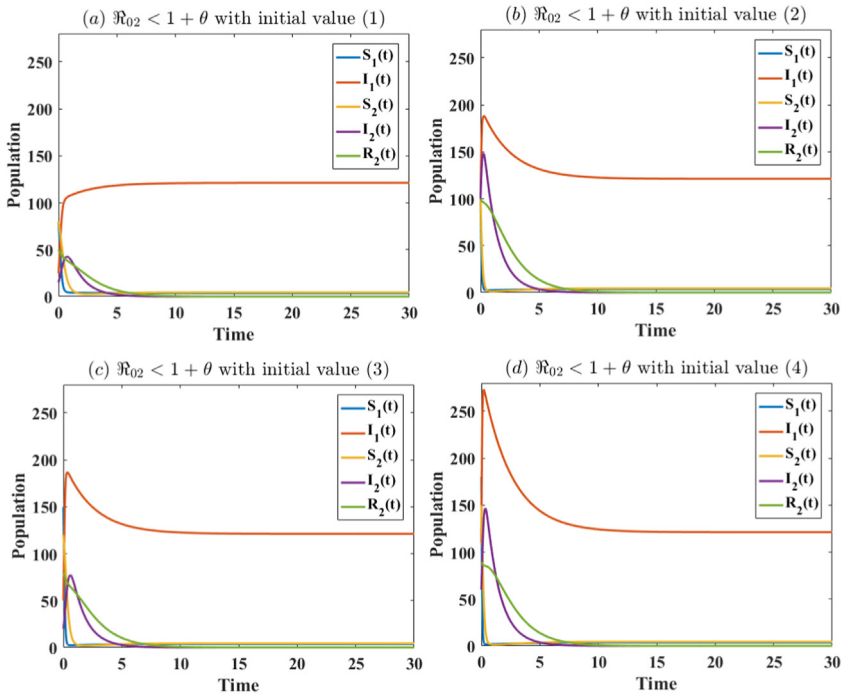

Fig. 2. The blue, red, orange, purple and green curves show the number of susceptible piglets $S_1(t)$, infected piglets $I_1(t)$, susceptible adult pigs $S_2(t)$, infected adult pigs $I_2(t)$ and recovered adult pigs $R_2(t)$, respectively. With parameters given in (36), we have $\Re_{02} \approx 4.1787 < 1+\theta$, we obtain that E_1 is globally asymptotically stable.

$$
\begin{aligned}
&(1)\ (S_1(0), I_1(0), S_2(0), I_2(0), R_2(0)) = (80, 25, 80, 15, 50);\\
&(2)\ (S_1(0), I_1(0), S_2(0), I_2(0), R_2(0)) = (100, 100, 100, 100, 100);\\
&(3)\ (S_1(0), I_1(0), S_2(0), I_2(0), R_2(0)) = (150, 50, 120, 20, 80);\\
&(4)\ (S_1(0), I_1(0), S_2(0), I_2(0), R_2(0)) = (180, 110, 150, 60, 90),
\end{aligned}
$$

as shown in Fig. 2 (a)-(d), respectively.

Example 5.2. Given parameters

$$
\begin{aligned}
&\Lambda = 50, \quad \beta_1 = 0.009, \quad \beta_2 = 0.05, \quad d_1 = 0.04, \quad d_2 = 0.03, \quad \alpha = 0.45,\\
&\gamma = 0.12, \quad \epsilon = 0.42, \quad \nu = 0.34, \quad p = 0.16, \quad \delta = 0.65, \quad \mu = 0.04.
\end{aligned} \tag{37}
$$

From (6) and (8), we have $\Re_{02} \approx 3.2910$ and $\theta \approx 1.4353$, which satisfies $\Re_{02} > 1 + \theta$. We find that no matter what the initial value is, the solution of model (4) will eventually tend to the disease-endemic equilibrium E^*, which implies that E^* is globally asymptotically stable. We choose four different initial values that are the same as Example 1 to verify our conclusion, see Fig. 3 (a)-(d), respectively.

Example 5.3. Given parameters

$$
\begin{aligned}
&\Lambda = 50, \quad \beta_2 = 0.0141, \quad d_1 = 0.004, \quad d_2 = 0.13, \quad \alpha = 0.34,\\
&\gamma = 0.1051, \quad \epsilon = 0.33, \quad \nu = 0.34, \quad p = 0.16, \quad \delta = 0.55, \quad \mu = 0.04.
\end{aligned} \tag{38}
$$

Parameter β_1 is increased from 0.001 to 0.005 in an arithmetic sequence with a tolerance of 0.001. With parameters given in (38), when $\beta_1 = 0.003$, we have $\Re_{01} = 1$. This illustrates the threshold $\Re_{01}$ increase strictly with respect to the contact rate β_1. When $\Re_{01} > 1$, the population of I_1 increases rapidly in a short time and will eventually approach positive constant. When $\Re_{01} < 1$, the population of I_1 gradually decreases and approach positive constant, that is, no matter what value $\Re_{01}$ takes, the disease always exists in piglets, as shown in the left figure (a) in Fig. 4.

Example 5.4. Given parameters

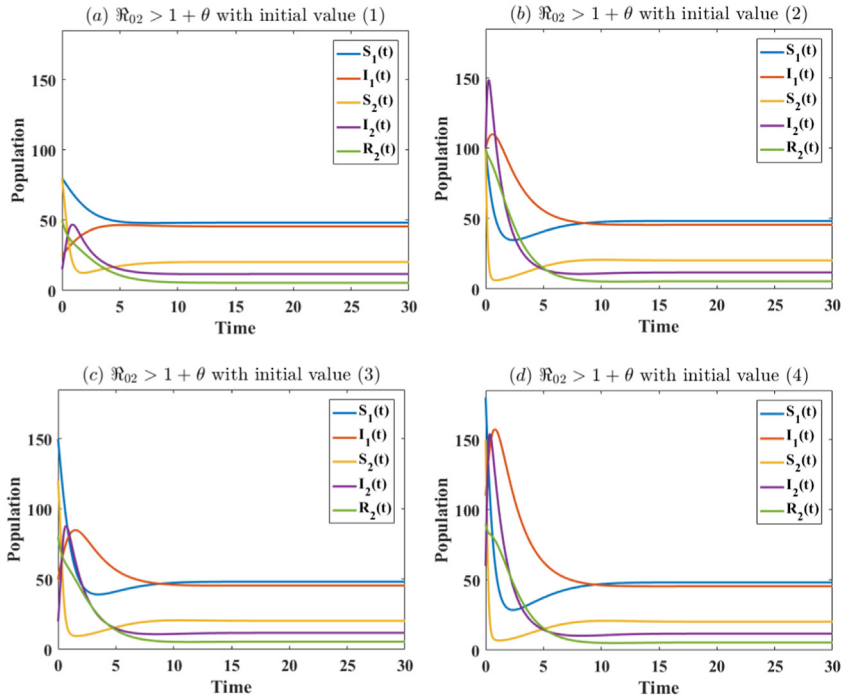

Fig. 3. The blue, red, orange, purple and green curves show the number of susceptible piglets $S_1(t)$, infected piglets $I_1(t)$, susceptible adult pigs $S_2(t)$, infected adult pigs $I_2(t)$ and recovered adult pigs $R_2(t)$, respectively. With parameters given in (37), the threshold is $\Re_{02} \approx 3.2910 > 1 + \theta$, we obtain that E^* is globally asymptotically stable.

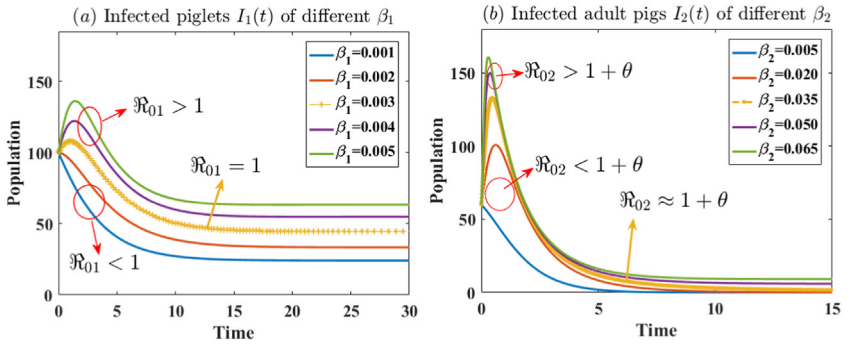

Fig. 4. The orange curve in (a) shows the threshold $\Re_{01} = 1$ and $\Re_{01}$ increases strictly with respect to the contact rate β_1. When $\Re_{01} > 1$, population of I_1 will eventually approach positive constant. When $\Re_{01} < 1$, the population of I_1 gradually decreases and approach positive constant, that is, no matter what value $\Re_{01}$ takes, the disease always exists in piglets, as shown in the left figure. The orange curve in (b) shows the threshold $\Re_{02} \approx 1 + \theta$, and $\Re_{02}$ increases strictly with respect to the contact rate β_2. When $\Re_{02} > 1 + \theta$, the population of I_2 will eventually approach positive constant. When $\Re_{02} < 1 + \theta$, the population of I_2 gradually decreases and finally approach zero, as shown in the right figure.

$$\begin{aligned} \Lambda &= 60, \quad \beta_1 = 0.09, \quad d_1 = 0.04, \quad d_2 = 0.13, \quad \alpha = 0.45, \quad \gamma = 0.22086, \\ \epsilon &= 0.32, \quad \nu = 0.35, \quad p = 0.16, \quad \delta = 0.55, \quad \mu = 0.04. \end{aligned} \tag{39}$$

Parameter β_2 is increased from 0.005 to 0.065. With parameters given in (39), when $\beta_2 = 0.035$, we have $\Re_{02} \approx 2.6651$ and $\theta \approx 1.6651$, then $\Re_{02} \approx 1 + \theta$. This illustrates the threshold $\Re_{02}$ increases strictly with respect to the contact rate β_2. When $\Re_{02} > 1 + \theta$, the population of I_2 increases rapidly in a short time and will eventually approach positive constant. When $\Re_{01} < 1 + \theta$, the population of I_2 gradually decreases and finally approach zero. That is, when $\Re_{02} > 1 + \theta$, the positive equilibrium E^* exists; when $\Re_{02} < 1 + \theta$, only the boundary equilibrium E_1 exists, as shown in the right figure (b) in Fig. 4.

6. Concluding remarks

In this paper, we built an age structure model of $S_1I_1S_2I_2R_2S_2$ with vertical transmission. The existence conditions of boundary equilibrium and disease-endemic equilibrium of model (4) are obtained. The analysis and numerical simulation show that there is no disease-free equilibrium of model (4) with vertical transmission, and there is always a boundary equilibrium, which is globally asymptotically stable when $\Re_{02} < 1 + \theta$. When $\Re_{02} > 1 + \theta$, the boundary equilibrium is unstable, and the model (4) admits a unique disease-endemic equilibrium, which is globally asymptotically stable. This also shows that the existence and stability of equilibria are independent of $\Re_{01}$.

The above numerical simulations show that the disease always exists in piglets, which will cause great economic losses to the pig industry and is not conducive to the development of China's pig industry. In addition, the expressions for $\Re_{01}$ (or $\Re_{02}$) show that $\Re_{01}$ (or $\Re_{02}$) increases strictly with respect to the contact rate β_1 (or β_2), i.e., the greater the value of β_1 (or β_2), the greater the value of $\Re_{01}$ (or $\Re_{02}$). Therefore, isolation of infected pigs, proper feeding density and vaccination would help prevent and control the outbreak of pseudorabies in pigs.

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Declaration of competing interest

All authors have no conflicts of interest. On behalf of all authors, the corresponding author states that there is no conflict of interest.

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