Southeast-Asian J. of Sciences Vol. 5, No. 1 (2017) pp. 32-46

MODELING PORCINE REPRODUCTIVE AND RESPIRATORY SYNDROME INCORPORATING DECAYING INFECTIOUSNESS AND DELAYED INFECTION INCIDENCES

Puttida Matkhao[1], **Yongwimon Lenbury**[1*]
Chontita Rattanakul[1] and **Nattawan Chuchalerm**[1]

[1] *Dept. of Mathematics, Faculty of Science, Mahidol Univ.*
Centre of Excellence in Mathematics, CHE,
Bangkok 10400, Thailand
e-mail: puttida_m@outlook.co.th; scylb@yahoo.com;
Chontita.rat@mahidol.ac.th; nattawanc13@gmail.com

Abstract

There are many epidemic diseases in the swine population such as swine fever disease, foot and mouth disease and Aujesky's disease. One of the most important diseases in the swine industry is porcine reproductive and respiratory syndrome (PRRS). Mathematical models of the disease are needed to study its behavior and discover effective and control strategies. Various research works on this subject have not been able find out how to control the disease effectively. In this work, we study a mathematical model of PRRS infection with a time-delay in the infection process, incorporating infectiousness decay. We carry out a stability analysis to discover the effect of time delay on the dynamic behavior of the model. Our work is expected to form a basis for further investigation, building upon our basic model, to test the potential effectiveness of employing various intervention strategies for disease containment, such as vaccination and isolation.

Key words: porcine reproductive and respiratory syndrome, time-delay, stability, decaying infectiousness.
*Corresponding author.

1. Introduction

Porcine Reproductive and Respiratory Syndrome (PRRS) is one of the most devastating infectious diseases in the swine industry. PRRS was found in the United States in 1987 and the causative virus was classified as Arterivirus in the Netherlands in 1991.

PRRS is caused by positive-stranded RNA or porcine reproductive and respiratory syndrome virus (PRRSV) which is responsible for reproductive failure in sows resulting in infertility, abortions and stillbirths. The virus has a particular relevance for macrophages (white blood cells) in the lungs where it reduces the immune response. Apart from severe reproductive failure in sows, other important manifestations of the disease are respiratory symptoms and increased pre-weaning mortality in suckling pigs and a mild flu-like symptoms in grower-finisher pigs [1-3].

Several researchers have investigated the disease and how the infection spread and the factors that play crucial roles in its symptoms [4-8] but have not yet been able to discover efficient control strategies. Specifically, the delay in time before the susceptible pigs are effectively infected by the infective population has not been taken into account, neither is the fact that infectiousness decays with time even though this has been reported by some investigators [8]. Therefore, we construct here a structured model for the spread of PRRSV that incorporates both the time delay as well as the decline of infectiousness with time.

2. Model system

In this work, we propose a model of PRRS infection with a time-delay in the infection incidences, incorporating infectiousness decay in the following form:

$$\begin{aligned}
\frac{d}{dt}S(t) &= b_{ss}S(t) + b_{is}I(t) - \beta e^{-\mu\tau}S(t-\tau)G(t-\tau) - d_sS(t) + C \\
\frac{d}{dt}I(t) &= \beta e^{-\mu\tau}S(t-\tau)G(t-\tau) + b_{ii}I(t) - d_iI(t) \qquad (1) \\
\frac{d}{dt}G(t) &= \alpha I(t) - \gamma G(t)
\end{aligned}$$

where $S(t)$ is the number of swine susceptible to the disease at time t, $I(t)$ is the number of infected swine at time t, $G(t)$ represents decaying infectiousness

of infected swine at time t, $b_{ss}S(t)$ is the birth rate of susceptible swine from susceptible sows, $b_{is}I(t)$ is the birth rate of susceptible swine from infected sows, $b_{ii}I(t)$ is the birth rate of infected swine from infected sows, $d_sS(t)$ is the natural death rate of susceptible swine, $d_iI(t)$ is the natural death rate of infected swine, β is the transmission rate constant, C represents a constant flow of susceptible swine into the whole population per unit time and the factor $\beta e^{-\mu\tau}S(t-\tau)G(t-\tau)$ represents the infection rate in which we incorporate the effect of the time-delay with $e^{-\mu\tau}$ being the probability that a swine survives from the time $t-\tau$ to time t. Considering PRRS data reported by Charpin *et al.*[8], we see that the number of new infections per infected swine decreases exponentially as time passes, after a delay of a few days before infection reaches a peak. So, $e^{\gamma(t-\tau)}$ represents the rate at which susceptible swine at time t is infected by a swine infected at time τ per infected swine. To obtain the total rate of infection at time t due to all swine infected at time τ, we multiply $e^{\gamma(t-\tau)}$ by $I(t)d\tau$ and integrate from 0 to t. We denote $\alpha\int_0^t e^{-\gamma(t-\tau)}I(\tau)d\tau$ by G and differentiate G with respect to t to obtain the third equation in (1).

For convenience, we rewrite the system (1) into the form:

$$\begin{aligned} \tfrac{dS}{dt} &= -\omega S + bI - \beta e^{-\mu\tau}S_\tau G_\tau + C \\ \tfrac{dI}{dt} &= \beta e^{\mu\tau}S_\tau G_\tau - \varphi I \\ \tfrac{dG}{dt} &= \alpha I - \gamma G \end{aligned} \tag{2}$$

where $S = S(t)$, $I = I(t)$, $G = G(t)$, $S_\tau = S(t-\tau)$, $G_\tau = G(t-\tau)$, $\omega = d_s - b_{ss}$, $b = b_{is}$ and $\varphi = d_i - b_{ii}$.

3. Boundedness of solutions

The boundedness of solutions to the delayed model system (2) can be determined using the comparison theorem and can be stated as in Theorem 3.1.

Theorem 3.1 *There exists an $M > 0$ such that for any solution $(S(t), I(t), G(t))$ of system (2) with positive initial values, $S(t) \leq M, I(t) \leq M$ and $G(t) \leq M$ for all $t > t_0$, for some $t_0 \geq 0$, provided $\omega > 0$ and $\varphi - b > \alpha$.*

Proof. We define

$$w(t) = S(t) + I(t) + G(t)$$

Finding the derivative of w with respect to t, we obtain

$$\begin{aligned}
\tfrac{dw}{dt} &= \tfrac{dS}{dt} + \tfrac{dI}{dt} + \tfrac{dG}{dt} \\
&= (-\omega S + BI - \beta e^{-\mu\tau} S_\tau G_\tau + C) + (\beta e^{-\mu\tau} S_\tau G_\tau - \varphi I) + (\alpha I - \gamma G) \\
&= C - [\omega S - bI + \varphi I - \alpha I + \gamma G] \\
&= C - [\omega S + (\varphi - b - \alpha) I + \gamma G] \\
\tfrac{dw}{dt} &\leq C - hw(t);
\end{aligned}$$

where $h = \min\{\omega, \varphi - b - \alpha, \gamma\}$.

Next, we consider the following comparison equation:

$$\frac{dw_1}{dt} = C - hw_1(t).$$

Then,

$$w_1(t) = \frac{C}{h} + w_1(0)e^{-ht}.$$

By the Comparison Test, we obtain

$$w(t) \leq w_1(t) = \frac{C}{h} + w_1(0)e^{-ht} \leq \frac{C}{h} + w_1(0)e^{-ht_0} \equiv M, \text{ if } t_0 \leq t.$$

Since $w(t) = S(t) + I(t) + G(t)$, $S(t), I(t)$, and $G(t)$ are also bounded. That is, there exists an $M > 0$ such that $S(t) \leq M$, $I(t) \leq M$ and $G(t) \leq M$ for all $t \geq t_0$, for some $t_0 \geq 0$, and thus each solution of system (2) with positive initial values is uniformly ultimately bounded. □

4. Stability

In this section, we analyze the model system in terms of its stability near the equilibrium points of (2). By setting $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dG}{dt} = 0$, we obtain 2 equilibrium points for the system (2) as follows.

1. Disease-free equilibrium

$$E_0 = (S_0, I_o, G_0) = \Big(\frac{C}{\omega}, 0, 0\Big)$$

in which S_0 is positive if

$$\omega > 0 \qquad (3).$$

2. Endemic equilibrium

$$E^* = (S^*, I^*, G^*) = \Big(\frac{\gamma\varphi}{\alpha\beta e^{-\mu\tau}}, \frac{\gamma\varphi\omega - C\alpha\beta e^{-\mu\tau}}{\alpha\beta e^{-\mu\tau}(b-\varphi)}, \frac{\alpha}{\gamma}I^*\Big)$$

which is positive, making ohysical sense, if

(i) $\omega > 0,\ \varphi - b > 1$ and $C\alpha\beta e^{-\mu\tau} > \gamma\varphi\omega$, or

(ii) $\omega < 0$ and $\varphi - b > \alpha$.

This means that if (i) holds then there could be co-existence of both equilibriums. However, if (ii) holds, there will only be the endemic equilibrium.

The stability of the disease free equilibrium is given in the following theorem.

Theorem 4.1 *If (3) holds and*

$$\gamma\varphi > \frac{C\alpha\beta}{\omega}, \tag{4}$$

then the disease-free equilibrium E_0 is locally asymptotically stable for all $\tau \geq 0$.

Proof. Local stability of the solution E_0 may be determined by considering the behavior of the linearized system of (2). If we define $x(t) = S(t) - \frac{C}{\omega}, y(t) = I(t)$ and $z(t) = G(t)$, then we obtain:

$$\begin{aligned} \tfrac{d}{dt}x(t) &= -\omega x(t) + by(t) - \tfrac{C}{\omega}\beta e^{-\mu\tau} z(t-\tau) \\ \tfrac{d}{dt}y(t) &= -\varphi y(t) + \tfrac{C}{\omega}\beta e^{-\mu\tau} z(t-\tau) \\ \tfrac{d}{dt}z(t) &= \alpha y(t) - \gamma z(t) \end{aligned} \tag{5}$$

and the characteristic equation of the Jacobian matrix of the system (2) is then:

$$(\lambda+\omega)\Big[\lambda^2 + (\gamma+\varphi)\lambda + \gamma\varphi - \frac{C\alpha\beta}{\omega}e^{-(\lambda+\mu)\tau}\Big] = 0 \tag{6}$$

Clearly, the equation (6) has a solution $\lambda_1 = -\omega$, which is negative since (3) holds. Next, we consider the factor

$$\lambda^2 + (\gamma+\varphi)\lambda + \gamma\varphi - \frac{C\alpha\beta}{\omega}e^{-(\lambda+\mu)\tau} \tag{7}$$

For $\tau = 0$, we have

$$\lambda^2 + (\gamma+\varphi)\lambda + \gamma\varphi - \frac{C\alpha\beta}{\omega} \tag{8}$$

Since $\gamma\varphi > \frac{C\alpha\beta}{\omega}$, from the Routh-Hurwitz criteria, we can conclude that all roots of (8) have negative real parts. So, all solutions of (6) have negative real parts for $\tau = 0$.

Next, for $\tau > 0$, we suppose that $\mathrm{Re}(\lambda)$ of (7) can be positive for some $\tau > 0$, then there must be a value of $\tau > 0$ such that $\mathrm{Re}(\lambda) = 0$. Let $\lambda = \eta i$

where η is real. Substituting $\lambda = \eta i$ into (7) and equating coefficients of like terms to zero, we have

$$-\eta^2 + \gamma\varphi = \frac{C\alpha\beta}{\omega} e^{-\mu\tau} cos\ \eta\tau \tag{9}$$

and

$$(\gamma + \varphi)\eta = -\frac{C\alpha\beta}{\omega} e^{-\mu\tau} sin\ \eta\tau \tag{10}$$

Squaring both sides of equations (9) and (10) and adding, we obtain

$$\eta^4 + (\gamma^2 + \varphi^2)\eta^2 - \frac{C^2\alpha^2\beta^2}{\omega^2} e^{-2\mu\tau} + \gamma^2\varphi^2 = 0.$$

Letting $\theta = \eta^2$, we have

$$\theta^2 + (\gamma^2 + \varphi^2)\theta - \frac{C^2\alpha^2\beta^2}{\omega^2} e^{-2\mu\tau} + \gamma^2\varphi^2 = 0 \tag{11}$$

Since (4) holds, we have

$$\gamma^2\varphi^2 > \frac{C^2\alpha^2\beta^2}{\omega^2} > \frac{C^2\alpha^2\beta^2}{\omega^2} e^{-2\mu\tau}.$$

Thus,

$$\gamma^2\varphi^2 = \frac{C^2\alpha^2\beta^2}{\omega^2} e^{-2\mu\tau} > 0$$

Hence, by the Routh-Hurwitz criteria, equation (11) has no positive solutions which means there is no value of $\tau > 0$ such that $\mathrm{Re}(\lambda) = 0$, which means that λ remains negative for $\tau \geq 0$. Therefore, the disease-free equilibrium is locally asymptotically stable for all $\tau \geq 0$. □

The next theorem involves the stability of the endemic equilibrium E^*.

Theorem 4.2 *If*

(i) $\omega > 0, \varphi - b > \alpha, C\alpha\beta e^{-\mu\tau} > \gamma\varphi\omega$ *and* $\frac{C\alpha\beta}{\gamma^2(\varphi - b)} > 1$, *or*

(ii) $\omega < 0, \varphi - b > \alpha, \gamma + \varphi + \omega > 0$ *and* $\frac{C\alpha\beta}{\gamma^2(\varphi - b)} > 1$,

then the endemic equilibrium E^* *is locally asymptotically stable for* $\tau = 0$.

Proof. we define $x(t) = S(t) - S^*$, $y(t) = I(t) - I^*$ and $z(t) = G(t) - G^*$ which leads us to

$$\begin{aligned}
\tfrac{d}{dt}x(t) &= -\omega x(t) - \beta e^{-\mu\tau} G^* x(t-\tau) + by(t) - \beta e^{-\mu\tau} S^* z(t-\tau) \\
\tfrac{d}{dt}y(t) &= \beta e^{-\mu\tau} G^* x(t-\tau) - \varphi y(t) + \beta e^{-\mu\tau} S^* z(t-\tau) \\
\tfrac{d}{dt}z(t) &= \alpha y(t) - \gamma z(t)
\end{aligned} \tag{12}$$

and the corresponding characteristic equation is given by

$$\lambda^3 + a_1\lambda^2 + a_2\lambda^2 e^{-\mu\tau} + a_3\lambda + a_4\lambda e^{-\mu\tau} + a_5 e^{-\mu\tau} + \gamma\varphi\omega = 0 \qquad (13)$$

where

$$\begin{aligned} a_1 &= \gamma + \varphi + \omega, \\ a_2 &= \tfrac{\gamma\varphi\omega - C\alpha\beta e^{-\mu\tau}}{\gamma(b-\varphi)}, \\ a_3 &= \gamma\varphi + \gamma\omega + \varphi\omega, \\ a_4 &= \tfrac{\gamma\varphi\omega - C\alpha\beta e^{-\mu\tau}}{b-\varphi} - \tfrac{\gamma\varphi\omega - C\alpha\beta e^{-\mu\tau}}{\gamma} - \gamma\varphi \\ \text{and } a_5 &= C\alpha\beta e^{-\mu\tau} - 2\gamma\varphi\omega. \end{aligned}$$

For $\tau = 0$, we have

$$\lambda^3 + \Big[\gamma+\varphi+\omega+\frac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)}\Big]\lambda^2 + \Big[\frac{\gamma\varphi\omega - C\alpha\beta}{b-\varphi} + \frac{C\alpha\beta}{\gamma} + \gamma\omega\Big]\lambda + C\alpha\beta - \gamma\varphi\omega = 0 \qquad (14)$$

In the case (i), since $C\alpha\beta > C\alpha\beta e^{-\mu\tau} > \gamma\varphi\omega$ and $\frac{C\alpha\beta}{\gamma^2(\varphi-b)} > 1$, applying the Routh-Hurwitz criteria, we have

$$\gamma + \varphi + \omega + \frac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)} > 0,$$

$$C\alpha\beta - \gamma\varphi\omega > 0$$

and

$$\begin{aligned} &\Big[\gamma + \varphi + \omega + \tfrac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)}\Big]\Big[\tfrac{\gamma\varphi\omega - C\alpha\beta}{b-\varphi} + \tfrac{C\alpha\beta}{\gamma} + \gamma\omega\Big] \\ &= \tfrac{1}{\gamma(\varphi-b)}\Big[C\alpha\beta(\varphi-b) + \gamma(C\alpha\beta - \gamma b\omega)\Big]\Big[\gamma + \varphi + \omega + \tfrac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)}\Big] \\ &\geq \tfrac{1}{\gamma(\varphi-b)} C\alpha\beta(\varphi-b)\Big[\gamma + \varphi + \omega + \tfrac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)}\Big] \geq C\alpha\beta - \gamma\varphi\omega. \end{aligned}$$

So, all solution of (14) have negative real parts for $\tau = 0$.

In the case (ii), we have $\omega < 0, \varphi - b > \alpha, \gamma + \varphi + \omega > 0$ and $\frac{C\alpha\beta}{\gamma^2(\varphi-b)} > 1$. From the Routh-Hurwitz criteria, we obtain

$\gamma + \varphi + \omega + \frac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)} > 0,$

$C\alpha\beta - \gamma\varphi\omega > 0$ and

$$\begin{aligned} &[\gamma + \varphi + \omega + \tfrac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)}][\tfrac{\gamma\varphi\omega - C\alpha\beta}{b-\varphi} + \tfrac{C\alpha\beta}{\gamma} + \gamma\omega] \\ &\geq \tfrac{1}{\gamma(\varphi-b)} C\alpha\beta(\varphi-b)[\gamma + \varphi + \omega + \tfrac{\gamma\varphi\omega - C\alpha\beta}{\gamma(b-\varphi)}] \geq C\alpha\beta - \gamma\varphi\omega. \end{aligned}$$

Thus, all solutions of (14) have negative real parts for $\tau = 0$. Therefore, in cases (i) and (ii), all solutions of (13) have negative real parts for $\tau = 0$ such

that the endemic equilibrium is locally asymptotically stable for this completes the proof of Theorem 4.2. □

Next, we consider the case where $\tau > 0$. We know that all solutions of (13) have negative real parts for $\tau = 0$. So, we suppose that $\mathrm{Re}(\lambda)$ of Eq. (13) can be positive for some $\tau > 0$. Then there must be a value of $\tau > 0$ such that $\mathrm{Re}(\lambda) = 0$. So, we assume $\lambda = \eta i$ where η is real. Substituting $\lambda = \eta i$ into equation (13), we have

$$-a_1\eta^2 + \gamma\varphi\omega = a_2\eta^2 cos\ \eta\tau - a_4\eta sin\ \eta\tau - a_5 cos\ \eta\tau \tag{15}$$

and

$$-\eta^3 + a_3\eta = -a_2\eta^2 sin\ \eta\tau - a_4\eta cos\ \eta\tau + a_5 sin\ \eta\tau \tag{16}$$

Squaring both sides of equations (15) and (16) and adding, we obtain

$$\eta^6 + (a_1^2 - a_2^2 - 2a_3)\eta^4 + (a_3^2 - a_4^2 - 2a_1\gamma\varphi\omega + 2a_2a_5)\eta^2 + \gamma^2\varphi^2\omega^2 - a_5^2 = 0 \tag{17}$$

Letting $\theta = \eta^2$, we are led to the following equation in θ :

$$P(\theta) = \theta^3 + A_1\theta^2 + A_2\theta + A_3 = 0 \tag{18}$$

where

$$\begin{aligned} A_1 &= a_1^2 - a_2^2 - 2a_3, \\ A_2 &= a_3^2 - a_2^2 - 2a_1\gamma\varphi\omega + 2a_2a_5, \\ A_3 &= \gamma^2\omega^2 - a_5^2 \end{aligned} \tag{19}$$

We need the following Lemmas.

Lemma 4.1 *Let $\tau > 0$. Suppose that the equation (18) has no positive roots. Then, all solutions of equation (13) have negative real parts.*

Proof. We refer the readers to [11] for the proof of this lemma. □

Lemma 4.2 *Let $\tau > 0$ and $A_3 > 0$.*

(i) If $A_2 > 0$ and there exists the real number $\theta_1 > 0$ such that $P(\theta_1) < 0$, then equation (18) has a positive root.

(ii) If $A_1^2 - 3A_2 < 0$, then equation (18) has no positive roots.

Proof. We refer the readers to [11] for the proof of this lemma. □

From, Lemma 4.1 and Lemma 4.2 (ii), we conclude that there is no a value of $\tau > 0$ such that $\mathrm{Re}(\lambda) = 0$. So, all roots of Eq. (13) have negative real parts. Hence, we obtain Theorem 4.3.

Theorem 4.3 *Suppose that the endemic equilibrium E^* exists and the conditions*

$$\frac{C\alpha\beta}{\gamma^2(\varphi - b)} > 1, \tag{20}$$

and

$$A_1^2 - 3A_2 < 0 \tag{21}$$

are satisfied. Then, the endemic equilibrium E^* is locally asymptotically stable for $\tau \geq 0$.

Proof. For $\tau = 0$, by Theorem 4.2, all solutions of (13) have negative real parts. By Lemma 4.1 and Lemma 4.2 (ii), we conclude that all roots of (13) have negative real parts for $\tau \geq 0$. Therefore, the endemic equilibrium E^* is locally asymptotically stable for $\tau \geq 0$.

5. Numerical simulations

In this section, we carry out numerical simulations of the model system to show the dynamic behavior of delayed model system (1) by using DDE23 in MATLAB which is in agreement of our theoretical predictions.

In Figure 1, we show a computer simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $C = 0.4, b_{ss} = 0.3, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.6, \alpha = 0.1, \beta = 0.4, \gamma = 0.1$ and $\mu = 0.120808$. For $\tau = 1$, we have $\omega = 0.3 > 0, \varphi - b_{is} - \alpha = 0.3 > 0, \gamma\varphi - \frac{C\alpha\beta}{\omega} = 0.00666667 > 0$ satisfying the conditions in Theorem 4.1. So, the disease free equilibrium point $E_0(\frac{4}{3}, 0, 0)$ is locally asymptotically stable, as seen in this figure where the time series of S converges to 1.33, and that of I vanishes to 0, while the solution trajectory seen projected onto the phase planes tends to the equilibrium point E_0 as time passes.

In Figure 2, we show a computer simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $C = 1, b_{ss} = 0.33 b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.6, \alpha = 0.1, \beta = 0.4, \gamma = 0.1$ and $\mu = 0.120808$.

For $\tau = 1$, we have $\varphi - b_{is} - \alpha = 0.3 > 0$, $C\alpha\beta e^{-\mu\tau} - \gamma\varphi\omega = 0.0174481 > 0$, $\omega = 0.3 > 0$, $A_1 = 0.121735 > 0, A_2 = 0.005169 > 0$, $A_3 = 3.23695 \times 10^{-4} > 0$ and $A_1^2 - 3A_2 = -6.8747986e - 04 < 0$ satisfying the conditions in Theorem 4.3. So, E^* is locally asymptotically stable for $\tau \geq 0$, as seen in Figure 2 where the time series of all state variables converge to their respective equilibrium values, while the solution trajectory seen projected onto the phase planes tends to the equilibrium point E^* as time passes.

Now, we have shown that, for equation (18) to have no positive solutions for $\tau \geq 0$, we need

$$A_1^2 - 3A_2 < 0$$

in (i). In this case there is no $\tau > 0$ such that $\lambda(\tau) = 0$, which means the equilibrium point is stable for all $\tau \geq 0$. However, if (i) does not hold, it does not mean that the equilibrium point must be unstable for all $\tau > 0$. It simply means that the equilibrium point may not be stable for all $\tau > 0$. That is, there can be a $\tau_0 > 0$ such that $\lambda(\tau_0) > 0$, but $\lambda(\tau) < 0$ for some $\tau < \tau_0$. This is what happens in Figure 3 where, even though $A_1^2 - 3A_2 > 0$ here, the equilibrium point is still stable for this value of τ that we used because τ is still less than τ_0.

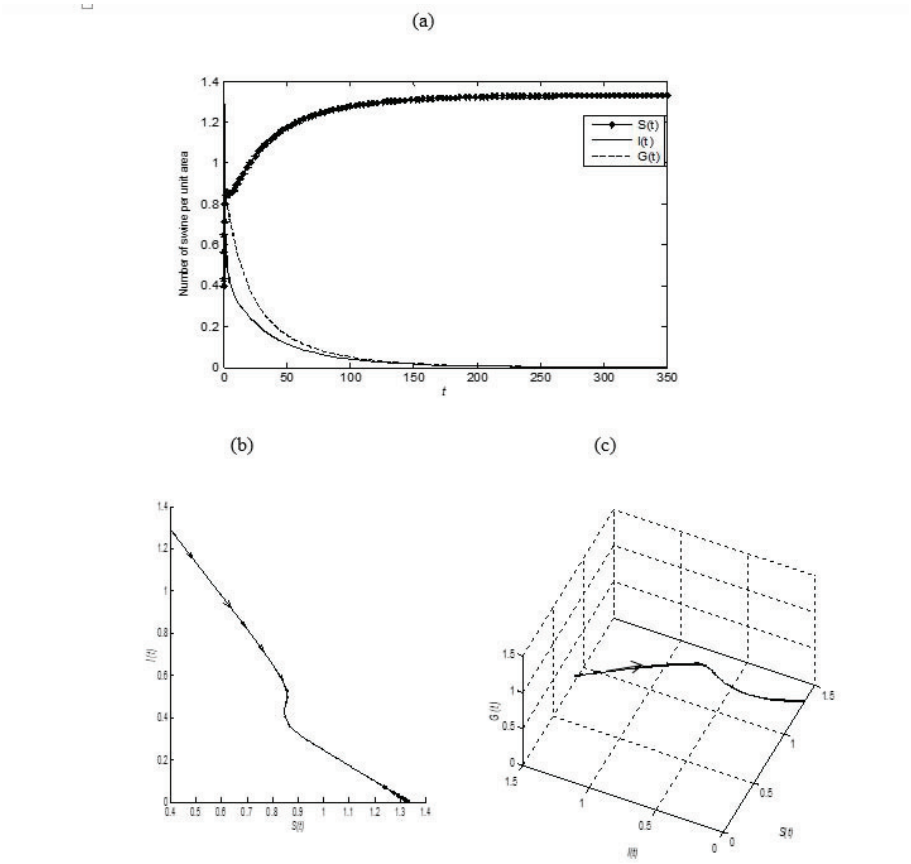

Figure 1: Numrical simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $C = 0.4, b_{ss} = 0.3, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.6, \alpha = 0.1, \beta = 0.4, \gamma = 0.1$, $\mu = 0.120808$. and $\tau = 1$. (a) Time series of $S()t$, $I(t)$ and $G(t)$. (b) Solution trajectory projected onto the (S, I) plane. (c) Three dimensional phase portrait of $S()t$, $I(t)$ and $G(t)$. The equilibrium point E_0 is stable here.

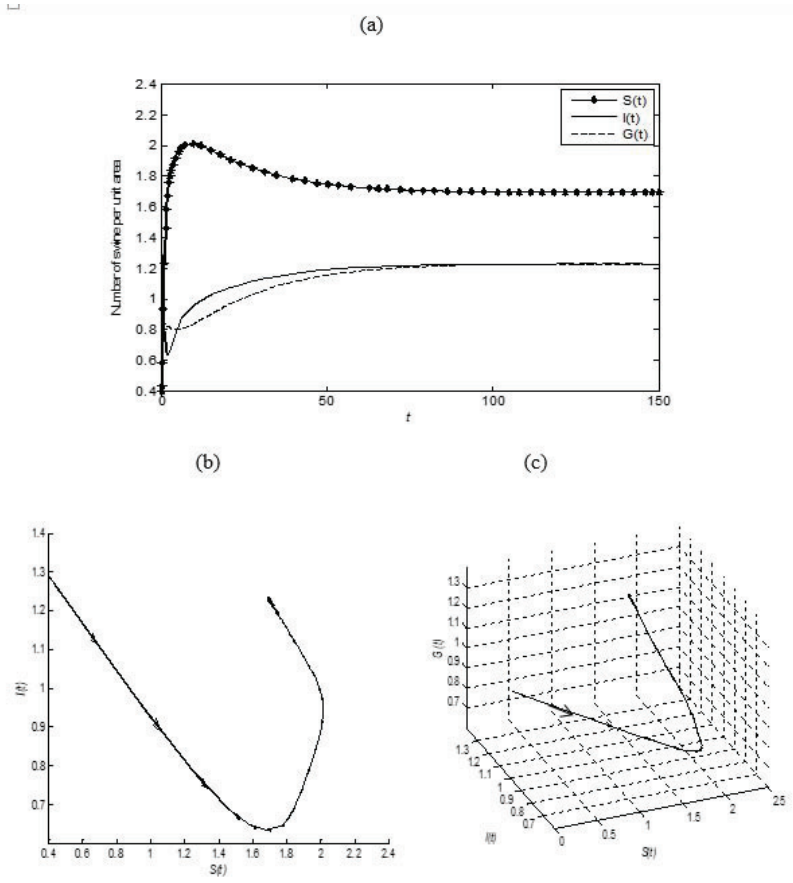

Figure 2: Numrical simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $C = 1, b_{ss} = 0.33, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.6, \alpha = 0.1, \beta = 0.4, \gamma = 0.1$, $\mu = 0.120808$ and $\tau = 1$. (a) Time series of $S()t$, $I(t)$ and $G(t)$. (b) Solution trajectory projected onto the (S, I) plane. (c) Three dimensional phase portrait of $S()t$, $I(t)$ and $G(t)$. The equilibrium point E^* is stable here.

In Figure 3, we show a computer simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $C = 1, b_{ss} = 0.3, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.1, \alpha = 0.1, \beta = 0.4, \gamma = 0.1$ and $\mu = 0.1208008$.

For $\tau = 1$, we have $A_1^2 - 3A_2 = 4.17978259$ which is non-negative. However, E^* is seen in this figure to be stable still for $\tau = 1$.

Since the condition $A_1^2 - 3A_2 < 0$ is not satisfied, we expect that the E^*

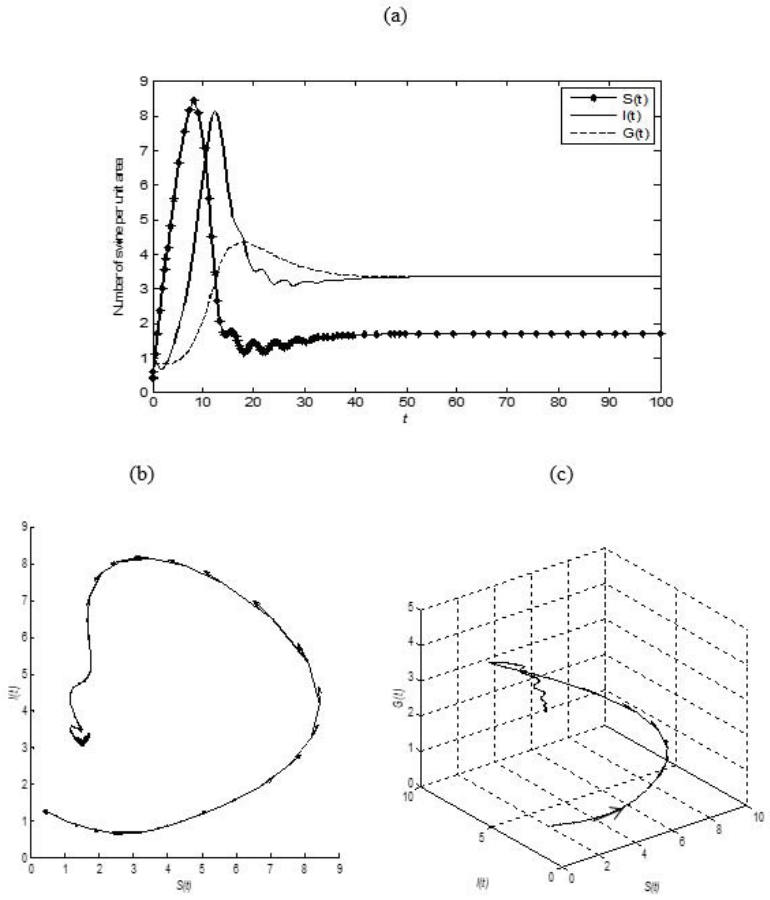

Figure 3: Numrical simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $C = 1, b_{ss} = 0.3, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.1, \alpha = 0.1, \beta = 0.4, \gamma = 0.1, \mu = 0.1208008$ and $\tau = 1$. (a) Time series of $S()t$, $I(t)$ and $G(t)$. (b) Solution trajectory projected onto the (S, I) plane. (c) Three dimensional phase portrait of $S()t$, $I(t)$ and $G(t)$. The equilibrium point E^* is still stable here.

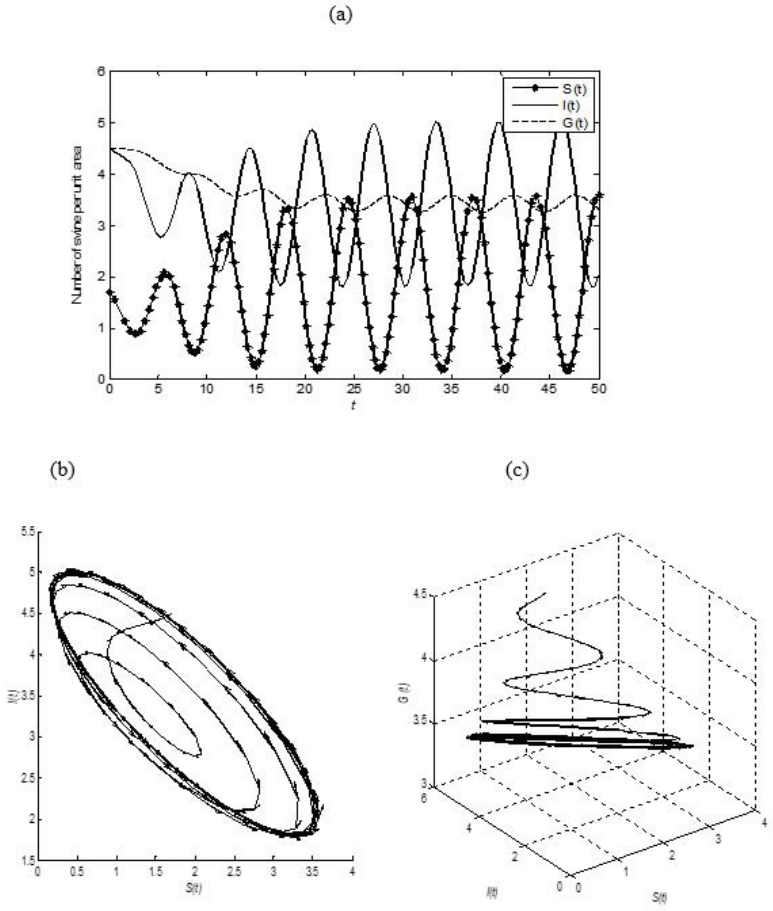

Figure 4: Numrical simulation of the model system (2) subject to initial values $S(0) = 0.4, I(0) = 1.29, G(0) = 0.82$ with $S(0) = 1.7, I(0) = 4, 5, G(0) = 4.5, C = 1, b_{ss} = 0.3, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.1, \alpha = 0.1, \beta = 0.4, \gamma = 0.1, \mu = 0.120808$ and $\tau = 1.6$. (a) Time series of $S()t$, $I(t)$ and $G(t)$. (b) Solution trajectory projected onto the (S, I) plane. (c) Three dimensional phase portrait of $S()t$, $I(t)$ and $G(t)$. The equilibrium point E_* is unstable and the solution trajectory is seen here to tend to a limit cycle.

cannot be stable for all $\tau > 0$. We therefore increase τ until it passes the critical value τ_0 at which point we expect E^* to become unstable. This is shown in Figure 4, where $S(0) = 1.7, I(0) = 4,5, G(0) = 4.5, C = 1, b_{ss} = 0.3, b_{is} = 0.2, b_{ii} = 0.3, d_i = 0.9, d_s = 0.1, \alpha = 0.1, \beta = 0.4, \gamma = 0.1$ and $\mu = 0.120808$. Here $\tau = 1.6$, large enough for E^* to become unstable and bifurcate into a limit cycle, giving rise to a periodic solution as seen in Figure 4.

6. Discussion and conclusion

In this work, we study and investigate the behavior of the solution to a model of the PRRS infection with time delay incorporating infectiousness decay. We proved, that for the disease-free equilibrium E_0 to be locally asymptotically stable for all $\tau \geq 0$, we need $\gamma\varphi > \frac{C\alpha\beta}{\omega}$ and $d_s - b_{ss} = \omega > 0$ which means that φ must be big enough, that is the death rate of infected swine is sufficiently bigger than its birth rate, or the infection constant γ is large enough so that infectiousness decreases fast enough. And for the endemic equilibrium E^* to be locally asymptotically stable for $\tau \geq 0$, we need $\frac{C\alpha\beta}{\gamma^2(\varphi - b)} > 1$ and $A_1^2 - 3A_2 < 0$ which means that the constant flow of swine C must be large enough.

Moreover, we observe that the delay in infection incidences τ is the critical parameter which delineates different dynamical behavior in the model system. In the report of Charpin et al. [8], a delay of up to 10 days is observed before infectiousness reaches a peak and then decreases in an exponential fashion. Such delay can play a crucial role in whether the disease is endemic or be controllable.

Another parameter that plays a crucial role in differentiating dynamical behavior that could be exhibited by the system is γ appearing in the exponential factor in the expression $G(t)$, which actually controls how fast the infectiousness decays with time. The bigger γ is, the faster the infected swine become ineffective in infecting the susceptible population. When γ large enough so that $\gamma > \frac{C\alpha\beta}{\omega\varphi}$ such that inequality (4) is satisfied, the population can be disease free, under suitable conditions on other parametric values. We in fact observe that the time series of $G(t)$ in Figure 1 shows an exponentially decreasing structure that is close to the data reported by Charpin et al. [6]. Theoretically, this suggests that, in such a situation, the spread of PRRS infection may be put under control if appropriate measures are taken, such as making sure that the specific death rate of infective swine is higher than the specific birth rate if the same thing holds for the susceptible swine population. In Figure 2, on the other hand, $G(t)$ does not decrease very sharply, so that the disease is endemic in this case.

Such insights gained from model construction and analysis can be extremely valuable in deciding on the most suitable intervention strategies.

Acknowledgement We would like to thank the Department of Mathematics, Faculty of Science, Mahidol University, the Center of Excellence in Mathematics, and the Science Achievement Scholarship of Thailand for its financial support.

References

[1] K. Dietze, J. Pinto, S. Wainwright, C. Hamilton and S. Khomenko, *Porcine reproductive and respiratory syndrome (PRRS) virulence jumps and persistent circulation,* intelligence information intervention, No.5(2011), 1-8.

[2] J.J.M. Meulenberg, M.M. Hulst, E.J. de Meijer, et al., *Lelystad virus, the causative agent of porcine epidemic abortion and respiratory syndrome (PEARS), is related to LDV and EAV,* Virology, 192 (1993), 62-72.

[3] OIE. (June 2008), *Report of the OIE ad hoc group on porcine reproductive and respiratory syndrome,* Retrieved from http://www.oie.int/ fileadmin/ Home/ eng/Our scienti?c expertise/docs/pdf/PRRS guide web bulletin.pdf

[4] G. Nodelijk, M.C.M de Jong, L.A.M.G. van Leengoed, et al., *A quantitative assessment of the effectiveness of PRRSV vaccination in pigs under experimental conditions,* Vaccine, 19 (2001), 3636-3644.

[5] A. Pitkin, J. Deen, D. Dee, *Use of a production region model to assess the airbourn spread of porcine reproductive and respiratory syndrome virus,* Vet. Micro., 136 (2009), 1-7.

[6] C.M. Evans, G.F. Medley, S.J. Creasey, L.E. Green, *A stochastic mathematical model of the within-herd transmission dynamics of porcine reproductive and respiratory syndrome virus (PRRSV): Fade-out and persistence,* Pre. Vet. Med., 93 (2010), 248-257

[7] A. Wilson, L.G. Pantoja, *Using mathematical models to gain insight into host pathogen interaction in mammals: porcine reproductive and respiratory syndrome,* Nova Science Publishers, (2010), 109-131.

[8] C. Charpin, S. Mahe, A. Keranflech, et al., *Infectiousness of pigs infected by the porcine reproductive and respiratory syndrome virus (PRRSV) is time-dependent,* Vet. Res., 43, 69 (2012). doi: 10.1186/1297-9716-43-69.

[9] O. Plaat,"Ordinary Differential Equations, Holden-day", Inc., California (1971).

[10] Y. Kuang, "Delay Differential Equations from Applications", Academic Press,Inc., San Diego CA, (1993).

[11] R.V. Culshaw, S. Ruan, *A delay-differential equation model of HIV infection of CD4+ T-cells,* Math. Biosci., 165 (2000), 27-39.

[12] R. Ouncharoen, S. Daengkongkho, T. Dumrongpokaphan and Y. Lenbury, *Delay SIR Model with Nonlinear Incident Rate and Varying Total Population,* Inter. J. of Mathematical Models and Computer in Simulation, **4** (2013), 369-378.