

GLOBAL ASYMPTOTIC STABILITY OF A DELAYED PLANT DISEASE MODEL

YUMING CHEN AND CHONGWU ZHENG

ABSTRACT. In this paper, we consider the following system of delayed differential equations,

$$\begin{cases} S'(t) = \sigma\phi - \beta S(t)I(t - \tau) - \eta S(t), \\ I'(t) = \sigma(1 - \phi) + \beta S(t)I(t - \tau) - (\eta + \omega)I(t), \end{cases}$$

which can be used to model plant diseases. Here $\phi \in (0, 1]$, $\tau \geq 0$, and all other parameters are positive. The case where $\phi = 1$ is well studied and there is a threshold dynamics. The system always has a disease-free equilibrium, which is globally asymptotically stable if the basic reproduction number $R_0 \triangleq \beta\sigma/\eta(\eta + \omega) \leq 1$ and is unstable if $R_0 > 1$; when $R_0 > 1$, the system also has a unique endemic equilibrium, which is globally asymptotically stable. In this paper, we study the case where $\phi \in (0, 1)$. It turns out that the system only has an endemic equilibrium, which is globally asymptotically stable. The local stability is established by the linearization method while the global attractivity is obtained by the Lyapunov functional approach. The theoretical results are illustrated with numerical simulations.

1. INTRODUCTION

Plant diseases can decrease the economic, aesthetic, and biological value of many types of plants. Examples of plant diseases caused by plant viruses can be found in the journal, *Plant Disease*. As a result, integrated management concepts have been developed to combat various plagues suffered by crops. Integrated management strategies combine available host resistance with cultural, chemical and biological control measures. For example, a cultural control strategy including replanting, and/or removing (roguing) diseased plants is a widely accepted treatment for plant epidemics.

Mathematical models of plant-virus disease epidemics are often built to provide a detailed exposition on how to describe, analyze, and predict epidemics of plant diseases. To name a few, see [7, 14, 16, 18, 19] and the references therein.

Based on the biological background in [2, 19], a simple model for plant diseases with a continuous cultural strategy can be built as follows. The plant population is divided into three compartments, susceptible plants (S), infected plants (I), and removed plants (R). Removal occurs by death or by sanitation. The removed plants are assumed not to be infected again, so we need to focus only on S and I . We make the following assumptions.

- a) New plants enter the system at the rate σ with a proportion ϕ being susceptible and $(1 - \phi)$ being infected.
- b) The incidence rate is proportional to SI and the transmission rate is β .
- c) Plants are removed from the system at the rate η , in other words, $1/\eta$ is either the harvest time or the end of their reproductive lifetime.

Received by the editors 23 January 2020; revised 22 February 2020; accepted 22 February, 2020; published online 24 February 2020.

2000 *Mathematics Subject Classification*. Primary 34K20; Secondary 92D30.

Key words and phrases. Delay, equilibrium, stability, plant disease.

The first author was supported in part by NSERC of Canada.

d) The rouging (removing infected plants from the system) rate is ω .

Figure 1 schematically sketches the transmission of a plant disease. The above assumptions lead to the

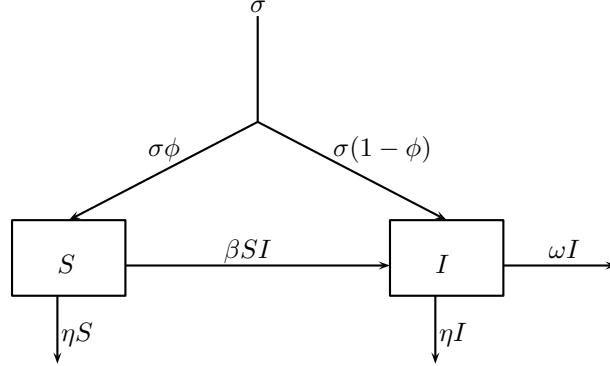


FIGURE 1. Schematic diagram for the transmission of a plant disease

following SI model for plant diseases,

$$\begin{cases} \frac{dS(t)}{dt} = \sigma\phi - \beta S(t)I(t) - \eta S(t), \\ \frac{dI(t)}{dt} = \sigma(1 - \phi) + \beta S(t)I(t) - (\eta + \omega)I(t). \end{cases} \quad (1.1)$$

Here the removal is continual, which is common for plant diseases. For human epidemics, the removal can be impulsive at some certain time instants. For example, see De la Sen et al. [4].

However, in disease transmission models, time delay is an important quantity for many epidemiological mechanisms, but this is not reflected in (1.1). We refer to van den Driessche [20] for a brief review of delay differential equations arising from disease modeling. As in Cooke [3], we incorporate a constant delay into (1.1) to obtain the following system of delayed differential equations,

$$\begin{cases} \frac{dS(t)}{dt} = \sigma\phi - \beta S(t)I(t - \tau) - \eta S(t), \\ \frac{dI(t)}{dt} = \sigma(1 - \phi) + \beta S(t)I(t - \tau) - (\eta + \omega)I(t). \end{cases} \quad (1.2)$$

Here τ is the average latent period of the plant disease, that is, the average time for a susceptible plant from getting infected to becoming infectious. An alternative approach, perhaps more realistic, is to incorporate a distributed delay as follows,

$$\begin{cases} \frac{dS(t)}{dt} = \sigma\phi - \beta S(t) \int_0^\tau I(t - s) dk(s) - \eta S(t), \\ \frac{dI(t)}{dt} = \sigma(1 - \phi) + \beta S(t) \int_0^\tau I(t - s) dk(s) - (\eta + \omega)I(t), \end{cases} \quad (1.3)$$

where $k : [0, \tau] \rightarrow \mathbb{R}$ is nondecreasing and has bounded variation such that $\int_0^\tau dk(s) = k(\tau) - k(0) = 1$. The discussion for (1.3) is quite similar to that for (1.2). We deal with (1.2) in the sequel and in Section 4 we will mention the necessary modification for a general system including (1.3).

Let $C = C([- \tau, 0], \mathbb{R}^2)$ be the Banach space of continuous functions from $[- \tau, 0]$ to \mathbb{R}^2 equipped with the usual supremum norm $\| \cdot \|$. The initial condition of (1.2) is given as

$$S(t) = \varphi_1(\theta) \text{ and } I(t) = \varphi_2(\theta) \text{ for } \theta \in [- \tau, 0], \quad (1.4)$$

where $\varphi = (\varphi_1, \varphi_2) \in C$ such that $\varphi_i(\theta) \geq 0$ for $\theta \in [-\tau, 0]$ and $i = 1, 2$. If $(S(t), I(t))$ is a solution of (1.2), then we have

$$S(t) = S(0)e^{-\int_0^t [\beta I(s-\tau) + \eta] ds} + \phi\sigma \int_0^t e^{-\int_u^t [\beta I(s-\tau) + \eta] ds} du \quad (1.5)$$

and

$$I(t) = e^{-(\eta+\omega)t} I(0) + \int_0^t e^{-(\eta+\omega)(t-u)} [(1-\phi)\sigma + \beta S(u)I(u-\tau)] du \quad (1.6)$$

for $t \geq 0$. It follows that given an initial condition (1.4), we can use the step-by-step method to find $S(t)$ first by (1.5) and then $I(t)$ by (1.6). One can also easily see that any solution of (1.2) with initial condition (1.4) satisfies $S(t) > 0$ and $I(t) > 0$ for $t > 0$. Without loss of generality, we need to consider only positive solutions of (1.2). Sometimes, the solution of (1.2) with the initial condition (1.4) is also denoted by $(S(t; \varphi), I(t; \varphi))$. As usual, for a solution $(S(t), I(t))$ of (1.2) and $t > 0$, we define $(S_t, I_t) \in C$ by $S_t(\theta) = S(t + \theta)$ and $I_t(\theta) = I(t + \theta)$ for $\theta \in [-\tau, 0]$. For more information on delay differential equations, we refer to Hale and Verduyn Lunel [8] or Diekmann et al. [5].

The case where $\phi = 1$ of (1.2) is the standard delayed SI or SIR model. These standard models with discrete delays and distributed delays have been well studied. To name a few, see [1, 11, 12, 17]. The dynamics of (1.2) with $\phi = 1$ is quite simple, which is a threshold dynamics. Precisely, the system always has a disease free equilibrium $E_0 = (\frac{\sigma}{\eta}, 0)$, which is globally asymptotically stable if $R_0 \triangleq \beta\sigma/\eta(\eta + \omega) \leq 1$ and is unstable if $R_0 > 1$; when $R_0 > 1$, it also has an endemic equilibrium

$$E^* = \left(\frac{\eta + \omega}{\beta}, \frac{\beta\sigma - \eta(\eta + \omega)}{\beta(\eta + \omega)} \right),$$

which is globally asymptotically stable. R_0 is called the basic reproduction number of (1.2).

Though (1.2) with $\phi = 1$ and its modifications have been extensively studied, system (1.2) with $\phi \in (0, 1)$ has not been studied yet. One reason is that the epidemics considered in the literature are mainly human diseases. In such situations, in order to lower the economic cost, infected persons usually will be quarantined or hospitalized. Therefore, it is reasonable to assume that $\phi = 1$. But, for plant diseases, it is necessary and important to study the dynamics of (1.2) with $\phi \in (0, 1)$, which is the goal of this paper. For example, African small holders replant cassava and sweet potato with cuttings only from previous crop while commercial sweet potato growers in China use disease-free material from *in vitro* propagation programmes [6]. The first corresponds to $\phi \in (0, 1)$ and the latter corresponds to $\phi = 1$ if we use (1.2) to model them, respectively.

The remainder of the paper is organized as follows. We first study the existence of equilibria and their local stability in Section 2. We then show that the system is globally asymptotically stable in Section 3 by the Lyapunov functional approach. The paper concludes with general remarks. It is mentioned that the approach here can be applied to deal with generalized versions of (1.2) with nonlinear incidence functions and distributed delays.

2. EXISTENCE OF EQUILIBRIA AND THEIR LOCAL STABILITY

In this section, we study the local dynamics of (1.2) with $\phi \in (0, 1)$. We first consider the existence of equilibria.

Proposition 2.1. Suppose $\phi \in (0, 1)$. Then (1.2) only has an endemic equilibrium $E_\phi^* = (S_\phi^*, I_\phi^*)$, where

$$\begin{aligned} S_\phi^* &= \frac{\beta\sigma + \eta(\eta + \omega) - \sqrt{\Delta_\phi}}{2\beta\eta}, \\ I_\phi^* &= \frac{\sigma - \eta S_\phi^*}{\eta + \omega}, \\ \Delta_\phi &= [\eta(\eta + \omega) + \beta\sigma]^2 - 4\phi\beta\sigma\eta(\eta + \omega). \end{aligned} \quad (2.1)$$

Proof. Let (S, I) be an equilibrium of (1.2). Then we have

$$\sigma\phi - \beta SI - \eta S = 0 \quad (2.2)$$

and

$$\sigma(1 - \phi) + \beta SI - (\eta + \omega)I = 0. \quad (2.3)$$

Adding (2.2) and (2.3) yields $\sigma - \eta S - (\eta + \omega)I = 0$, or $I = \frac{\sigma - \eta S}{\eta + \omega}$. Then substituting it into (2.2), we obtain

$$G(S) = \frac{\beta\eta}{\eta + \omega} S^2 - \left(\frac{\beta\sigma}{\eta + \omega} + \eta \right) S + \phi\sigma = 0.$$

Note that $G(0) = \phi\sigma > 0$ and $G(\frac{\sigma}{\eta}) = -(1 - \phi)\sigma < 0$. It follows that $G(S) = 0$ has two positive solutions, one less than $\frac{\sigma}{\eta}$ and the other larger than $\frac{\sigma}{\eta}$. From $I = (\sigma - \eta S)/(\eta + \omega) \geq 0$, we know that $S \leq \sigma/\eta$. Hence (1.2) has only one equilibrium, which is positive and is given by (2.1). This completes the proof. \square

Proposition 2.1 tells us that (1.2) has no disease-free equilibrium, which is due to the recruitment of infected plants. Therefore, the basic reproduction number cannot be defined. In the following, we investigate the local stability of E_ϕ^* . E_ϕ^* is *locally stable* if for any $\varepsilon > 0$ there exists $\delta > 0$ such that

$$\|(S_t(\cdot; \varphi), I_t(\cdot; \varphi)) - E_\phi^*\| \leq \varepsilon \quad \text{for } t \geq 0 \text{ and } \|\varphi - E_\phi^*\| \leq \delta;$$

otherwise, it is *unstable*. The local stability of E_ϕ^* is obtained by the technique of linearization. Precisely, if all roots of the characteristic equation associated with the linearized system about E_ϕ^* have negative real parts then E_ϕ^* is locally exponentially stable and hence stable; if at least one of the roots has positive real part then E_ϕ^* is unstable. E_ϕ^* is locally exponentially stable if there exist positive constants m , α , and δ such that

$$\|(S_t(\cdot; \varphi), I_t(\cdot; \varphi)) - E_\phi^*\| \leq m e^{-\alpha t} \|\varphi - E_\phi^*\|$$

for all $t \geq 0$ and $\|\varphi - E_\phi^*\| \leq \delta$.

To prove the local stability of E_ϕ^* , it is worthy to note that $\Delta_\phi > [\eta(\eta + \omega) + \beta\sigma]^2 - 4\phi\beta\sigma\eta(\eta + \omega) = [\beta\sigma - \eta(\eta + \omega)]^2$ as $\phi \in (0, 1)$.

Theorem 2.1. *Suppose $\phi \in (0, 1)$. Then the equilibrium E_ϕ^* of (1.2) is locally exponentially stable.*

Proof. Linearize (1.2) around E_ϕ^* to obtain

$$\begin{cases} \frac{dS(t)}{dt} = -(\beta I_\phi^* + \eta)S(t) - \beta S_\phi^* I(t - \tau), \\ \frac{dI(t)}{dt} = \beta I_\phi^* S(t) + \beta S_\phi^* I(t - \tau) - (\eta + \omega)I(t). \end{cases}$$

The corresponding characteristic equation is

$$\det \begin{pmatrix} \lambda + \beta I_\phi^* + \eta & \beta S_\phi^* e^{-\lambda\tau} \\ -\beta I_\phi^* & \lambda + \eta + \omega - \beta S_\phi^* e^{-\lambda\tau} \end{pmatrix} = 0,$$

or

$$\lambda^2 + \lambda(2\eta + \omega + \beta I_\phi^*) - \beta S_\phi^* e^{-\lambda\tau}(\lambda + \eta) + (\beta I_\phi^* + \eta)(\eta + \omega) = 0. \quad (2.4)$$

To finish the proof, we only need to show that all roots of (2.4) have negative real parts.

First, suppose that $\tau = 0$. Then (2.4) reduces to

$$\lambda^2 + \lambda(2\eta + \omega + \beta I_\phi^* - \beta S_\phi^*) + [(\beta I_\phi^* + \eta)(\eta + \omega) - \beta S_\phi^* \eta] = 0.$$

With (2.1), we get

$$\lambda^2 + P_\phi \lambda + \sqrt{\Delta_\phi} = 0,$$

where

$$P_\phi = \frac{2\eta^3 + 3\eta^2\omega + \eta\omega^2 - \beta\sigma\omega + (2\eta + \omega)\sqrt{\Delta_\phi}}{2\eta(\eta + \omega)}.$$

If $\beta\sigma \geq \eta(\eta + \omega)$, then $\sqrt{\Delta_\phi} > \beta\sigma - \eta(\eta + \omega)$ and hence

$$P_\phi \geq \frac{2\eta^3 + 3\eta^3\omega + \eta\omega^2 - \beta\sigma\omega + (2\eta + \omega)[\beta\sigma - \eta(\eta + \omega)]}{2\eta(\eta + \omega)} = \frac{\beta\sigma}{\eta + \omega} > 0;$$

if $\beta\sigma < \eta(\eta + \omega)$, then

$$\begin{aligned} P_\phi &> \frac{2\eta^3 + 3\eta^2\omega + \eta\omega^2 - \omega\eta(\eta + \omega) + (2\eta + \omega)\sqrt{\Delta_\phi}}{2\eta(\eta + \omega)} \\ &= \frac{2\eta^3 + 2\eta^2\omega + (2\eta + \omega)\sqrt{\Delta_\phi}}{2\eta(\eta + \omega)} \\ &> 0. \end{aligned}$$

Therefore, if $\tau = 0$ then all roots of (2.4) have negative real parts.

Next, we claim that (2.4) has no roots on the imaginary axis. By way of contradiction, suppose that (2.4) has a root $i\xi$ with $\xi \in \mathbb{R}$ for some $\tau > 0$. Then substitute it into (2.4) and separate the real and imaginary parts to obtain

$$(\beta I_\phi^* + \eta)(\eta + \omega) - \xi^2 = \beta S_\phi^*(\xi \sin \xi\tau + \eta \cos \xi\tau)$$

and

$$(2\eta + \omega + \beta I_\phi^*)\xi = \beta S_\phi^*(\xi \cos \xi\tau - \eta \sin \xi\tau).$$

It follows that

$$\xi^4 + [(2\eta + \omega + \beta I_\phi^*)^2 - 2(\beta I_\phi^* + \eta)(\eta + \omega) - (\beta S_\phi^*)^2]\xi^2 + [(\beta I_\phi^* + \eta)^2(\eta + \omega)^2 - (\beta S_\phi^* \eta)^2] = 0. \quad (2.5)$$

Note that

$$\begin{aligned} &(\beta I_\phi^* + \eta)^2(\eta + \omega)^2 - (\beta S_\phi^* \eta)^2 \\ &= [(\beta I_\phi^* + \eta)(\eta + \omega) + \beta S_\phi^* \eta][(\beta I_\phi^* + \eta)(\eta + \omega) - \beta S_\phi^* \eta] \\ &= [(\beta I_\phi^* + \eta)(\eta + \omega) + \beta S_\phi^* \eta]\sqrt{\Delta_\phi} \\ &> 0 \end{aligned}$$

and

$$\begin{aligned}
& (2\eta + \omega + \beta I_\phi^*)^2 - 2(\beta I_\phi^* + \eta)(\eta + \omega) - (\beta S_\phi^*)^2 \\
&= [(\eta + \omega) + (\eta + \beta I_\phi^*)]^2 - 2(\beta I_\phi^* + \eta)(\eta + \omega) - (\beta S_\phi^*)^2 \\
&= (\eta + \omega)^2 + (\eta + \beta I_\phi^*)^2 - (\beta S_\phi^*)^2 \\
&= (\eta + \beta I_\phi^*)^2 + (\eta + \omega + \beta S_\phi^*)(\eta + \omega - \beta S_\phi^*) \\
&= (\eta + \beta I_\phi^*)^2 + (\eta + \omega + \beta S_\phi^*) \frac{\eta(\eta + \omega) - \beta\sigma + \sqrt{\Delta_\phi}}{2\eta} \\
&> 0 \quad (\text{as } \sqrt{\Delta_\phi} > |\eta(\eta + \omega) - \beta\sigma|).
\end{aligned}$$

It follows that (2.5) cannot hold and this proves the claim.

Note that the roots of (2.4) depend continuously on τ and that all roots of (2.4) with non-negative real parts are uniformly bounded (which is easy to see). This, combined with the claim and the result that all roots of (2.4) have negative real parts for the case where $\tau = 0$, tells us that all roots of (2.4) have negative real parts for any $\tau \geq 0$. Therefore, we have completed the proof. \square

3. GLOBAL ASYMPTOTIC STABILITY OF THE ENDEMIC EQUILIBRIUM

In the previous section, we have shown that (1.2) has a unique endemic equilibrium E_ϕ^* which is locally stable. Indeed, E_ϕ^* is also *globally asymptotically stable*, that is, E_ϕ^* is locally stable and globally attractive. E_ϕ^* is globally attractive if $\lim_{t \rightarrow \infty} (S_t, I_t) = E_\phi^*$ for every solution $(S(t), I(t))$ of (1.2).

To prove the global attractivity, we first establish the permanence of (1.2).

Definition 3.1 ([9]). System (1.2) is said to be *permanent* if there are positive constants ν_i and M_i ($i = 1, 2$) such that

$$\nu_1 \leq \liminf_{t \rightarrow \infty} S(t) \leq \limsup_{t \rightarrow \infty} S(t) \leq M_1$$

and

$$\nu_2 \leq \liminf_{t \rightarrow \infty} I(t) \leq \limsup_{t \rightarrow \infty} I(t) \leq M_2$$

hold for any solution of (1.2) with the initial condition (1.4). Here ν_i and M_i ($i = 1, 2$) are independent of (1.4).

Proposition 3.1. Suppose $\phi \in (0, 1)$. Then (1.2) is permanent. In fact, for every solution $(S(t), I(t))$, we have

$$\frac{\sigma\phi\eta}{\beta\sigma + \eta^2} \leq \liminf_{t \rightarrow \infty} S(t) \leq \limsup_{t \rightarrow \infty} S(t) \leq \frac{\sigma}{\eta}, \quad (3.1)$$

$$\frac{\sigma(1-\phi)}{\eta + \omega} \leq \liminf_{t \rightarrow \infty} I(t) \leq \limsup_{t \rightarrow \infty} I(t) \leq \frac{\sigma}{\eta}. \quad (3.2)$$

Proof. Let $(S(t), I(t))$ be any solution of (1.2). Then

$$\frac{d(S(t) + I(t))}{dt} = \sigma - \eta S(t) - (\eta + \omega)I(t) \leq \sigma - \eta(S(t) + I(t))$$

and hence by the comparison principle (see, for example, [10]) we get

$$\begin{aligned}
S(t) + I(t) &\leq \left[\frac{\sigma(e^{\eta t} - 1)}{\eta} + S(0) + I(0) \right] e^{-\eta t} \\
&= \frac{\sigma}{\eta} + \frac{\eta(S(0) + I(0)) - \sigma}{\eta} e^{-\eta t}.
\end{aligned}$$

It follows that

$$\limsup_{t \rightarrow \infty} (S(t) + I(t)) \leq \frac{\sigma}{\eta},$$

which implies that

$$\limsup_{t \rightarrow \infty} S(t) \leq \frac{\sigma}{\eta} \quad \text{and} \quad \limsup_{t \rightarrow \infty} I(t) \leq \frac{\sigma}{\eta}. \quad (3.3)$$

In particular, for any $\varepsilon > 0$, there exists a $T > 0$ such that

$$I(t) \leq \frac{\sigma}{\eta} + \varepsilon \quad \text{for all } t \geq T.$$

Then, for $t \geq T + \tau$,

$$\frac{dS(t)}{dt} \geq \phi\sigma - \left[\beta \left(\frac{\sigma}{\eta} + \varepsilon \right) + \eta \right] S(t),$$

which immediately yields from the comparison principle that

$$\liminf_{t \rightarrow \infty} S(t) \geq \frac{\phi\sigma}{\beta \left(\frac{\sigma}{\eta} + \varepsilon \right) + \eta}.$$

As ε is arbitrary, we have

$$\liminf_{t \rightarrow \infty} S(t) \geq \frac{\phi\sigma\eta}{\beta\sigma + \eta^2}. \quad (3.4)$$

Finally, note that

$$\frac{dI(t)}{dt} \geq (1 - \phi)\sigma - (\eta + \omega)I(t) \quad \text{for } t \geq 0.$$

Again, by the comparison principle we get

$$\liminf_{t \rightarrow \infty} I(t) \geq \frac{(1 - \phi)\sigma}{\eta + \omega}. \quad (3.5)$$

It follows from (3.3)–(3.5) immediately that (1.2) is permanent and both (3.1) and (3.2) hold. This completes the proof. \square

Now, we are ready to prove the main result of this section.

Theorem 3.2. *Suppose $\phi \in (0, 1)$. Then the equilibrium E_ϕ^* of (1.2) is globally asymptotically stable.*

Proof. We have shown in Theorem 2.1 that E_ϕ^* is locally stable and hence it suffices to show that E_ϕ^* is globally attractive. The proof is completed by using the Lyapunov functional approach and is parallel to that of Theorem 4.1 of McCluskey [13] (but we use the notation in Hale and Verduyn Lunel [8]). To construct the Lyapunov functional, we need the function $g(x) = x - 1 - \ln x$ for $x > 0$. It is well-known that $g(x) \geq 0$ and $g(x) = 0$ if and only if $x = 1$.

Let

$$D = \left\{ \varphi = (\varphi_1, \varphi_2) \in C \left| \begin{array}{l} \varphi_1(\theta) \in \left[\frac{\sigma\phi\eta}{\beta\sigma + \eta^2}, \frac{\sigma}{\eta} \right] \text{ and} \\ \varphi_2(\theta) \in \left[\frac{(1-\phi)\sigma}{\eta + \omega}, \frac{\sigma}{\eta} \right] \text{ for } \theta \in [-\tau, 0] \end{array} \right. \right\}.$$

It is not difficult to see that D is an invariant set of (1.2). Moreover, by Proposition 3.1, D is attractive. Therefore, to study the global attractivity of E_ϕ^* , we only need to consider solutions starting in D . We define $V : D \rightarrow \mathbb{R}$ by

$$V(\varphi) = \frac{1}{\beta I_\phi^*} V_S(\varphi_1) + \frac{1}{\beta S_\phi^*} V_I(\varphi_2) + V_+(\varphi_2) \quad \text{for } \varphi = (\varphi_1, \varphi_2) \in D,$$

where

$$V_S(\varphi_1) = g\left(\frac{\varphi_1(0)}{S_\phi^*}\right), \quad V_I(\varphi_2) = g\left(\frac{\varphi_2(0)}{I_\phi^*}\right) \quad \text{and} \quad V_+(\varphi_2) = \int_0^\tau g\left(\frac{\varphi_2(-s)}{I_\phi^*}\right) ds.$$

To find the derivative of V along solutions of (1.2), the following relationships between S_ϕ^* and I_ϕ^* will be helpful,

$$\phi\sigma = \beta S_\phi^* I_\phi^* + \eta S_\phi^*, \quad (3.6)$$

$$(\eta + \omega)I_\phi^* = (1 - \phi)\sigma + \beta S_\phi^* I_\phi^*. \quad (3.7)$$

For clarity, we first calculate the derivatives of V_S , V_I , and V_+ along solutions of (1.2) one by one.

Firstly,

$$\begin{aligned} \dot{V}_S(\varphi_1) &= \limsup_{h \rightarrow 0^+} \frac{1}{h} [V_S(S_h) - V_S(\varphi_1)] \\ &= \left. \frac{dV_S(S_h)}{dh} \right|_{h=0} \\ &= \frac{1}{S_\phi^*} \left(1 - \frac{S_\phi^*}{S(0)} \right) \frac{dS(0)}{dt} \\ &= \frac{1}{S_\phi^*} \left(1 - \frac{S_\phi^*}{S(0)} \right) (\phi\sigma - \beta S(0)I(-\tau) - \eta S(0)) \\ &= \frac{1}{S_\phi^*} \left(1 - \frac{S_\phi^*}{\varphi_1(0)} \right) (\phi\sigma - \beta\varphi_1(0)\varphi_2(-\tau) - \eta\varphi_1(0)). \end{aligned}$$

Using (3.6) to replace $\phi\sigma$ gives us

$$\begin{aligned} \dot{V}_S(\varphi_1) &= \frac{1}{S_\phi^*} \left(1 - \frac{S_\phi^*}{\varphi_1(0)} \right) \{ \eta[S_\phi^* - \varphi_1(0)] + \beta[S_\phi^* I_\phi^* - \varphi_1(0)\varphi_2(-\tau)] \} \\ &= -\eta \frac{(\varphi_1(0) - S_\phi^*)^2}{\varphi_1(0)S_\phi^*} + \beta I_\phi^* \left(1 - \frac{S_\phi^*}{\varphi_1(0)} \right) \left(1 - \frac{\varphi_1(0)}{S_\phi^*} \frac{\varphi_2(-\tau)}{I_\phi^*} \right). \end{aligned}$$

For simplicity of notation, let

$$x = \frac{\varphi_1(0)}{S_\phi^*}, \quad y = \frac{\varphi_2(0)}{I_\phi^*}, \quad \text{and} \quad z = \frac{\varphi_2(-\tau)}{I_\phi^*}.$$

Then we can get

$$\dot{V}_S(\varphi_1) = -\eta \frac{(\varphi_1(0) - S_\phi^*)^2}{\varphi_1(0)S_\phi^*} + \beta I_\phi^* \left(1 - xz - \frac{1}{x} + z \right). \quad (3.8)$$

Secondly,

$$\begin{aligned} \dot{V}_I(\varphi_2) &= \frac{1}{I_\phi^*} \left(1 - \frac{I_\phi^*}{\varphi_2(0)} \right) [(1 - \phi)\sigma + \beta\varphi_1(0)\varphi_2(-\tau) - (\eta + \omega)\varphi_2(0)] \\ &= \frac{1}{I_\phi^*} \left(1 - \frac{I_\phi^*}{\varphi_2(0)} \right) \left[(1 - \phi)\sigma + \beta\varphi_1(0)\varphi_2(-\tau) - (\eta + \omega)I_\phi^* \frac{\varphi_2(0)}{I_\phi^*} \right]. \end{aligned}$$

With the help of (3.7), we get

$$\dot{V}_I(\varphi_2) = \frac{1}{I_\phi^*} \left(1 - \frac{I_\phi^*}{\varphi_2(0)} \right) \left[(1 - \phi)\sigma \left(1 - \frac{\varphi_2(0)}{I_\phi^*} \right) \right. \quad (3.9)$$

$$\begin{aligned} &\left. + \beta S_\phi^* I_\phi^* \left(\frac{\varphi_1(0)}{S_\phi^*} \frac{\varphi_2(-\tau)}{I_\phi^*} - \frac{\varphi_2(0)}{I_\phi^*} \right) \right] \\ &= -(1 - \phi)\sigma \frac{(\varphi_2(0) - I_\phi^*)^2}{\varphi_2(0)(I_\phi^*)^2} + \beta S_\phi^* \left(1 - y - \frac{xz}{y} + xz \right). \quad (3.10) \end{aligned}$$

Finally,

$$\begin{aligned}
\dot{V}_+(\varphi_2) &= \frac{d}{dt} \int_0^\tau g\left(\frac{I(t-s)}{I_\phi^*}\right) ds \Big|_{t=0} \\
&= \int_0^\tau \frac{d}{dt} g\left(\frac{I(t-s)}{I_\phi^*}\right) \Big|_{t=0} ds \\
&= \int_0^\tau -\frac{d}{ds} g\left(\frac{I(-s)}{I_\phi^*}\right) ds \\
&= g\left(\frac{I(0)}{I_\phi^*}\right) - g\left(\frac{I(-\tau)}{I_\phi^*}\right) \\
&= g(y) - g(z) \\
&= y - z + \ln z - \ln y.
\end{aligned} \tag{3.11}$$

Now, we obtain from (3.8), (3.10), and (3.11) that

$$\begin{aligned}
\dot{V}(\varphi) &= -\frac{\eta}{\beta I_\phi^* S_\phi^*} \frac{(\varphi_1(0) - S_\phi^*)^2}{\varphi_1(0)} - \frac{(1-\phi)\sigma}{\beta S_\phi^* (I_\phi^*)^2} \frac{(\varphi_2(0) - I_\phi^*)^2}{\varphi_2(0)} \\
&\quad + 2 - \frac{1}{x} - \frac{xz}{y} + \ln z - \ln y \\
&= -\frac{\eta}{\beta I_\phi^* S_\phi^*} \frac{(\varphi_1(0) - S_\phi^*)^2}{\varphi_1(0)} - \frac{(1-\phi)\sigma}{\beta S_\phi^* (I_\phi^*)^2} \frac{(\varphi_2(0) - I_\phi^*)^2}{\varphi_2(0)} \\
&\quad + \left(1 - \frac{1}{x} - \ln x\right) + \left(1 - \frac{xz}{y} + \ln \frac{xz}{y}\right) \\
&= -\frac{\eta}{\beta I_\phi^* S_\phi^*} \frac{(\varphi_1(0) - S_\phi^*)^2}{\varphi_1(0)} - \frac{(1-\phi)\sigma}{\beta S_\phi^* (I_\phi^*)^2} \frac{(\varphi_2(0) - I_\phi^*)^2}{\varphi_2(0)} \\
&\quad - g\left(\frac{1}{x}\right) - g\left(\frac{xz}{y}\right).
\end{aligned}$$

Then $\dot{V}(\varphi) \leq 0$ and $K = \{\varphi \in \overline{D} \mid \dot{V}(\varphi) = 0\} = \{\varphi = (\varphi_1, \varphi_2) \in C \mid \varphi_1(0) = S_\phi^*, \varphi_2(0) = I_\phi^*\}$. Obviously, the largest set in K that is invariant with respect to (1.2) is the singleton $\{E_\phi^*\}$. By Theorem 3.1 in Chapter 5 of Hale and Verduyn Lunel [8], every solution converges to E_ϕ^* . This completes the proof. \square

4. CONCLUDING REMARKS

In this paper, we considered a plant disease model (1.2) with $\phi \in (0, 1)$. Unlike the case where $\phi = 1$, there is no threshold dynamics. The model has a unique endemic equilibrium E_ϕ^* , which is always globally asymptotically stable. Figure 2 and Figure 3 illustrate this result for the cases $\beta\sigma < \eta(\eta + \omega)$ and $\beta\sigma > \eta(\eta + \omega)$, respectively. In both cases, $\tau = 4$, $\beta = 0.0064$, $\eta = 0.002$, $\phi = 0.75$. But for Figure 2, $\sigma = 0.0015$ and $\omega = 0.003$; while for Figure 3, $\sigma = 0.015$ and $\omega = 0.03$. These parameter values are reasonable for cassava (Figure 2) and sweet potatoes (Figure 3), respectively. We refer to van den Bosch *et al* [19] for more detail.

The global asymptotic stability implies that the disease cannot be eradicated. This is realistic since, in practice, it is almost impossible to guarantee that all replanted plants are healthy. This result looks quite different from that for the case where $\phi = 1$. However, when ϕ is very close to 1, the result is

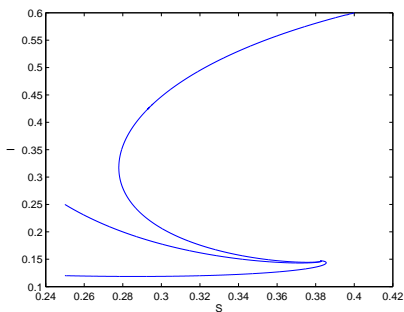


FIGURE 2. When $\sigma = 0.0015$, $\phi = 0.75$, $\beta = 0.0064$, $\eta = 0.002$, $\omega = 0.003$ and $\tau = 4$, the equilibrium $E_\phi^* \approx (0.3826, 0.1470)$ of (1.2) is globally asymptotically stable.

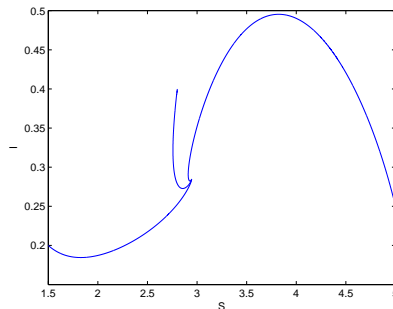


FIGURE 3. When $\sigma = 0.015$, $\phi = 0.75$, $\beta = 0.0064$, $\eta = 0.002$, $\omega = 0.03$ and $\tau = 4$, the equilibrium $E_\phi^* \approx (2.9428, 0.2848)$ of (1.2) is globally asymptotically stable.

consistent with that for the case where $\phi = 1$. In fact,

$$I_\phi^* = \frac{\beta\sigma - \eta(\eta + \omega) + \sqrt{\Delta_\phi}}{2\beta(\eta + \omega)}.$$

If $\beta\sigma = \eta(\eta + \omega)$ then

$$I_\phi^* = \frac{\sqrt{(1 - \phi)\beta\sigma\eta(\eta + \omega)}}{\beta(\eta + \omega)} = \frac{\eta\sqrt{1 - \phi}}{\beta}.$$

Suppose that $\beta\sigma \neq \eta(\eta + \omega)$. If $\phi \approx 1$, then

$$\sqrt{\Delta_\phi} \approx |\beta\sigma - \eta(\eta + \omega)| \left[1 + \frac{2(1 - \phi)\beta\sigma\eta(\eta + \omega)}{[\beta\sigma - \eta(\eta + \omega)]^2} \right] = |\beta\sigma - \eta(\eta + \omega)| + \frac{2(1 - \phi)\beta\sigma\eta(\eta + \omega)}{|\beta\sigma - \eta(\eta + \omega)|}$$

and hence

$$I_\phi^* \approx \frac{(1 - \phi)\sigma\eta}{\eta(\eta + \omega) - \beta\sigma} \quad \text{if } \beta\sigma < \eta(\eta + \omega)$$

while

$$I_\phi^* \approx \frac{(\beta\sigma - \eta(\eta + \omega))^2 + (1 - \phi)\sigma\eta(\eta + \omega)}{(\eta + \omega)[\beta\sigma - \eta(\eta + \omega)]} \quad \text{if } \beta\sigma > \eta(\eta + \omega),$$

which are quite close to the disease levels in the case where $\phi = 1$. The discussion also tells us that in order to develop a good control strategy, we should make $\beta\sigma < \eta(\eta + \omega)$. In this case, though the disease cannot be eradicated, the disease level can be very small and hence it cannot reach the economic injury level [15] (the lowest population density that will cause economic damage or the amount of injury which will justify the cost of using controls).

We mention that we can use the same Lyapunov functional to show the global attractivity of the endemic equilibrium of the following generalization of (1.2),

$$\begin{cases} \frac{dS(t)}{dt} = \phi\sigma - \beta S(t)f(I(t - \tau)) - \eta S(t), \\ \frac{dI(t)}{dt} = \sigma(1 - \phi) + \beta S(t)f(I(t - \tau)) - (\eta + \omega)I(t), \end{cases} \quad (4.1)$$

provided that an equilibrium (which is of course an endemic equilibrium) is guaranteed by the incidence function f . For example, $f(x) = \frac{1}{1 + \alpha x}$ in McCluskey [13]. Here, we considered the case where $f(x) = x$ for the simple reason that we could obtain the local stability of the endemic equilibrium. For a general

incidence function f , it may not be easy to analyze the local stability. Moreover, consider modifications of (4.1) with a distributed delay,

$$\begin{cases} \frac{dS(t)}{dt} = \phi\sigma - \beta S(t) \int_0^\tau f(I(t-s))dk(s) - \eta S(t), \\ \frac{dI(t)}{dt} = \sigma(1-\phi) + \beta S(t) \int_0^\tau f(I(t-s))dk(s) - (\eta + \omega)I(t), \end{cases}$$

where $k : [0, \tau] \rightarrow \mathbb{R}$ is nondecreasing and has bounded variation such that $\int_0^\tau dk(s) = k(\tau) - k(0) = 1$. Same results can be obtained by replacing $V_+(\varphi_2)$ with $\hat{V}_+(\varphi_2)$, where

$$\hat{V}_+(\varphi_2) = \int_0^\tau \alpha(s)g\left(\frac{\varphi_2(-s)}{I_\phi^*}\right) ds$$

and $\alpha(h) = \int_h^\tau dk(s)$ for $h \in [0, \tau]$. We refer to McCluskey [12] for some guidance.

REFERENCES

- [1] E. Beretta and Y. Takeuchi, *Global stability of an SIR epidemic model with times*, J. Math. Biol. **33**(1995) 250–260.
- [2] M.S. Chan and M.J. Jeger, *An analytical model of plant virus disease dynamics with roguing and replanting*, J. Appl. Ecol. **31**(1994) 413–427.
- [3] K.L. Cooke, *Stability analysis for a vector disease model*, Rocky Mount. J. Math. **7**(1979) 253–263.
- [4] M. De la Sen, Ravi P. Agarwal, A. Ibeas and S. Alonso-Quesada, *On a generalized time-varying SEIR epidemic model with mixed point and distributed time-varying delays and combined regular and impulsive vaccination controls*, Advances in Differential Equations **2010** (2010), Article ID 281612, 42 pages.
- [5] O. Diekmann, S.A. van Gils, S.M. Verduyn Lunel and H.-O. Walthert, *Delay Equations: Functional-, Complex-, and Nonlinear Analysis*, Springer-Verlag, New York, 1995.
- [6] G. Feng, G. Yifu and Z. Pinbo, *Production and development of virus-free sweet potato in China*, Crop Prot. **19**(2000) 105–111.
- [7] S. Fishman and R. Marcus, *A model for spread of plant disease with periodic removals*, J. Math. Biol. **21**(1984) 149–158.
- [8] J. Hale and S.M. Verduyn Lunel, *Introduction to Functional Differential Equations*, Springer-Verlag, New York, 1993.
- [9] Y. Kuang, *Delay Differential Equations with Applications in Population Dynamics*, Academic Press, San Diego, 1993.
- [10] V. Lakshmikantham and S. Leela, *Differential and Integral Inequalities: Theory and Applications, Vol. I: Ordinary Differential Equations*, Academic Press, New York-London, 1969.
- [11] W. Ma, Y. Takeuchi, T. Hara and E. Beretta, *Permanence of an SIR epidemic model with distributed time delays*, Tohoku Math. J. **54**(2002) 581–591.
- [12] C.C. McCluskey, *Complete global stability for an SIR epidemic model with delay—Distributed or discrete*, Nonlinear Anal. Real World Appl. **11**(2010) 55–59.
- [13] C.C. McCluskey, *Global stability for an SIR epidemic model with delay and nonlinear incidence*, Nonlinear Anal. Real World Appl. **11**(2010) 3106–3109.
- [14] S. Soubeyrand, L. Held, M. Höhle and I. Sache, *Modelling the spread in space and time of an airborne plant disease*, J. Roy. Statist. Soc. Ser. C **57**(2008) 253–272.
- [15] V.M. Stern, R.F. Smith, R. van den Bosch and K.S. Hagen, *The integrated control concept*, Hilgardia, **29**(1959) 81–101.
- [16] N. Stollenwerk and K.M. Briggs, *Master equation solution of a plant disease model*, Phys. Lett. A **274**(2000) 84–91.
- [17] Y. Takeuchi, W. Ma and E. Beretta, *Global asymptotic properties of a delay SIR epidemic model with finite incubation times*, Nonlinear Anal., **42**(2000) 931–947.
- [18] H.R. Thieme and J.A.P. Heesterbeek, *How to estimate the efficacy of periodic control of an infectious plant disease*, Math. Biosci. **93**(1989) 15–29.
- [19] F. van den Bosch, M.J. Jeger and C.A. Gilligan, *Disease control and its selection for damaging plant virus strains in vegetatively propagated staple food crops; a theoretical assessment*, Proc. R. Soc. B **274**(2007) 11–18.
- [20] P. van den Driessche, *Some epidemiological models with delays*, in *Differential Equations and Applications to Biology and to Industry*, pp. 507–520, World Sci. Publ., River Edge, NJ, 1996.

CORRESPONDING AUTHOR. DEPARTMENT OF MATHEMATICS, WILFRID LAURIER UNIVERSITY, WATERLOO, ON N2L 3C5,
CANADA

E-mail address: ychen@wlu.ca

DEPARTMENT OF APPLIED MATHEMATICS, YUNCHENG UNIVERSITY, YUNCHENG, SHANXI 044000, P.R. CHINA

E-mail address: chongyang1894@163.com