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ANALYSIS OF A VECTOR PREFERENCE MODEL FOR POTATO VIRUS Y TRANSMISSION

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Abstract. Potato virus Y (PVY) is one of the most common widespread vector-borne transmission diseases through aphids. In recent years, biologists have focused on the effect of vector preference to the spread of PVY. In this paper, according to transmission mechanism of PVY, a mathematical model of a vector-borne disease including preference behavior and vertical transmission of vector is formulated. The basic reproduction number R_0 is calculated by using the next generation matrix method. The existence of a backward bifurcation presents a further sub-threshold condition below R_0 for the spread of the disease by theoretical and numerical analysis. Numerical simulations suggest that vector preference plays an important role in the spread of PVY.

Keywords: potato virus Y; vector preference; basic reproduction number; backward bifurcation.

2020 AMS Subject Classification: 92D30, 34C23.

1. INTRODUCTION

The spread of plant virus diseases relies on vector transmission, of which nearly 80% are transmitted by specific vectors [1-3], such as aphids, whiteflies, leafhoppers, and thrips. These viruses cause drastic reductions in crop yield, especially for tomato, cassava crops, and potato [4]. Potato (Solanum tuberosum) is the third most important staple food crop in the world

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consumed by approximately 1.3 billion people [5]. One of the most devastating diseases of potato is potato virus Y (PVY, genus Potyvirus, family Potyviridae), transmitted by the aphid [6] which causes quality and yield of potatoes in commercial production [7,8]. PVY infection is a major issue in potatoes because PVY spreads easily and quickly and results in yield losses up to 85% [9]. PVY is transmitted by aphid to potato in a nonpersistent manner. Acquisition and inoculation periods are brief (only a few minutes) [6]. Primary symptoms of PVY are mottling or yellowing of leaflets, necrosis, leaf dropping or sometimes premature death [10].

Aphid is the most efficient vector of PVY [11,12]. Like other insects, aphids exhibit directed responses to external stimuli, known as preference behavior. It is a complex process for an aphid to accept or reject a potato which governed by visual, tactile, and chemical cues [13,14]. From landing the potato leaf surface a few seconds to several minutes, an aphid receives stimulatory cues communicating whether to continue feeding [15]. If the potato provides appropriate cues, the aphid settles and feeds deeper [16]. It is reported that aphids can often detect and respond to the phenotypic differences between healthy and infected potatoes, and then exhibit different landing and feeding preferences [17]. Phenotypic preference of aphid can directly influence the transmission rate of PVY.

Recent research on mathematical models of PVY has been mainly focused on the effects of transmission efficiency, vector behavior, and vector activity [18-20], in which these models have assumed that vector transmission occurs essentially at random. Research indicates that, after being infected by PVY virus, the host plant can alter the expression of related genes within its body, thereby causing changes in the feeding behavior and preferences of the aphids. The aphid exhibits different preferences for infected and uninfected host plants. It is essential to incorporate vector preference behavior into the model construction because vectors with strong host selection contribute to the spread of plant virus diseases [21-23]. In spite of research regarding the effect of vector preference on the spread of PVY is relatively rare, there have been some research works recently focused on the impact of vector preference on the transmission of malaria, West Nile virus, and barley yellow dwarf virus (see [24-27]). Several studies suggest that infected humans may be more attractive to mosquitoes than healthy humans, which shows that vector preference plays an important role in controlling the spread of malaria [24,25]. Marini et

al. [26] affirm that vector feeding preferences can have important consequences for the pathogen invasion, the probability to start an epidemic, and the influence of West Nile virus transmission rates. Mcelhany et al. [27] indicate that the effect of vector preference for healthy or infected hosts on the spread of barley yellow dwarf virus. The results show that vector preference for infected plants influence the probability of disease spread, which depends on the frequency of diseased plants in the population.

The epidemiology of plant viruses diseases is very sensitive to the vector preference of the insect vectors [28-30]. However, the simultaneous effects of vector preference and vertical transmission in potatoes on PVY transmission have never been studied before by using mathematical models. In this paper, our main purpose is to investigate how vector preference and vertical transmission influence the transmission of PVY.

2. Model Formulation

Following the idea of [24,31], we propose a mathematical model (2.1) that includes landing and feeding preferences of vector. The proposed model includes healthy, incubation (infected but not infectious) and infected host potatoes, and non-viruliferous and viruliferous aphids. Let $H(t)$, $L(t)$, and $I(t)$ denote the density of the three stages of host potatoes and $X(t)$, $Y(t)$ denote the two stages for the aphids, where the time t is measured on a daily basis. Hence, the total host potatoes and aphid population are given as

$$N_p(t) = H(t) + L(t) + I(t),\ N_v(t) = X(t) + Y(t).$$

PVY virus is carried by the vector aphid in a nonpersistent manner. After being inoculated with PVY, aphids are considered as infective aphid. When aphids carrying the virus come into contact with healthy potatoes, they transmit the virus to the potatoes, resulting in potato infection after an incubation period. Following the idea of [32,33], the mathematical model we will establish does not take into account the virus population, but it can exist in the system through infective aphids.

In the absence of disease, susceptible potatoes grow logistically towards the environmental carrying capacity, denoted as K, at a rate of r. Therefore, the vital dynamics of the potatoes can

be described by the equation

$$rH\left(1-\frac{H+L+I}{K}\right).$$

Aphids show preferences in their interactions with potatoes [17]. Inspired by the idea of [34], we define v to be the degree to which aphids prefer to land on infected potatoes, then aphids can be biased either in favour of ($v>1$), or against ($v<1$), landing on infected potatoes. Similarly, we define c to be the degree to which aphids prefer to land on incubation period potatoes, aphids can then be biased either in favour of ($c>1$), or against ($c<1$), landing on incubation period potatoes. Hence, the probability that aphid (healthy or infected) lands on a host potato (healthy, incubation, or infected) is $\frac{H}{H+cL+vI}$, $\frac{cL}{H+cL+vI}$, or $\frac{vI}{H+cL+vI}$ (see Fig. 1).

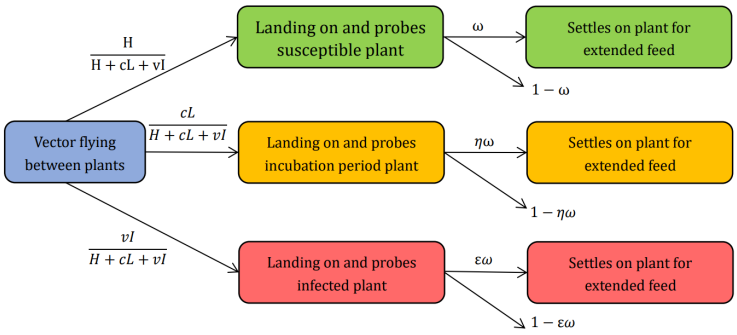


FIGURE 1. Schematic showing how the movement, landing, and feeding behaviours of aphids are modelled.

It is considered that there is vertical transmission of the infection among aphids. Let b be the proportion of aphids get infected due to birth on infectious potatoes. Thus the infection occurs due to birth from infectious potatoes can be given as following forms:

$$bX(t)\frac{vI(t)}{H+cL(t)+vI(t)}.$$

It is noted that a aphid probes a potato directly after landing, but then chooses between settling for an extended feed or immediately moving off to probe a different potato. The probability of feeding potentially depends on the state of the potato (healthy, incubation, or infected). We define ω is the probability that aphids settle to feed on healthy potatoes, η is the probability that aphids settle to feed on incubation period potatoes and ε is the probability that aphids settle to feed on infected potatoes, where $\omega\leq 1, \varepsilon\omega\leq 1, \eta\omega\leq 1$. Hence, the probability that aphid

settles for extended feed on a host potato (healthy, incubation, or infected) is ω, $\eta\omega$, or $\varepsilon\omega$ (see Fig. 1). And the probability that aphid probes a host potato (healthy, incubation, or infected) but moves elsewhere is $1-\omega$, $1-\eta\omega$, or $1-\varepsilon\omega$ (see Fig. 1).

Based on the idea of [34], in the presence of vector preference, the overall rate at which healthy potatoes are inoculated is taken as following forms:

$$\frac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} \doteq \Phi\times\omega\times Y\times\frac{H}{\omega\Gamma(H+\eta cL+\varepsilon vI)},$$

where

$$\Phi=\frac{H+cL+vI}{\omega\Gamma(H+\eta cL+\varepsilon vI)},$$

represents the average number of potatoes visited by each aphid per unit of time, and

$$\frac{H}{\omega\Gamma(H+\eta cL+\varepsilon vI)}$$

is the probability that a single visit by a aphid is made to a susceptible potato.

Similarly, the overall rate at which susceptible aphids acquire infection should be taken as forms:

$$\frac{\varepsilon XvI(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} \doteq \Phi\times\varepsilon\omega\times X\times\frac{vI}{\omega\Gamma(H+\eta cL+\varepsilon vI)},$$

where $\varepsilon\omega$ is the probability that a aphid acquires the virus from an infected potato, and

$$\frac{vI}{\omega\Gamma(H+\eta cL+\varepsilon vI)},$$

is the probability that an individual visit by a aphid is made to an infected potato.

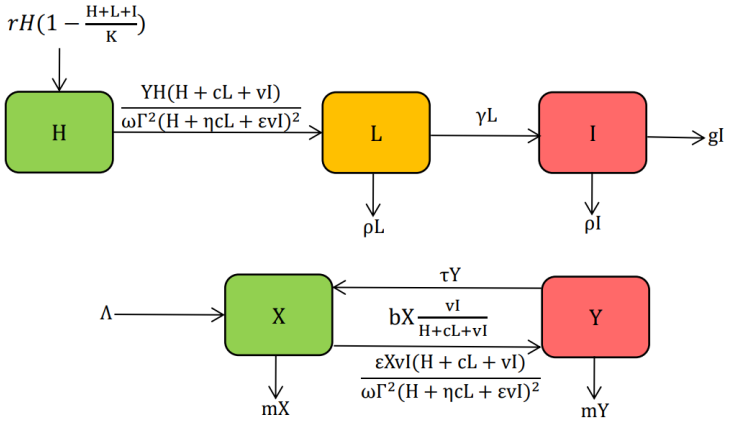


FIGURE 2. Schematic diagram of the formulated PVY disease dynamic model.

Taking into account the above considerations and from model structure diagrams (Fig. 1 and Fig. 2), the PVY transmission dynamics will be governed by the following system of differential equations:

$$
\begin{cases}
\dfrac{dH}{dt} = rH(1-\dfrac{H+L+I}{K}) - \dfrac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2}, \\
\dfrac{dL}{dt} = \dfrac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} - \gamma L - \rho L, \\
\dfrac{dI}{dt} = \gamma L - \rho I - gI, \\
\dfrac{dX}{dt} = \Lambda - bX\dfrac{vI}{H+cL+vI} + \tau Y - \dfrac{\varepsilon XvI(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} - mX, \\
\dfrac{dY}{dt} = bX\dfrac{vI}{H+cL+vI} + \dfrac{\varepsilon XvI(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} - (\tau+m)Y,
\end{cases} \tag{2.1}
$$

where the description of all parameters of system (2.1) can be seen in Table 1.

The initial condition of system (2.1) is given as

$$H(0) \geq 0, L(0) \geq 0, I(0) \geq 0, X(0) \geq 0, Y(0) \geq 0.$$

3. Main Results

3.1. Boundedness and Non-negativity of Solutions. The non-linear system (2.1) will be qualitatively analysed boundedness and non-negativity of solutions. Adding the last two equations of system (2.1), we have

$$\frac{dN_v(t)}{dt} = \Lambda - mN_v(t).$$

Obviously, we have $\lim\limits_{t\to\infty} N_v(t) = \dfrac{\Lambda}{m}$, which gives the asymptotic relation $X = \dfrac{\Lambda}{m} - Y$. Therefore, we only need to study the following limit system of (2.1):

$$
\begin{cases}
\dfrac{dH}{dt} = rH(1-\dfrac{H+L+I}{K}) - \dfrac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} \triangleq f_1(H,L,I,Y), \\
\dfrac{dL}{dt} = \dfrac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} - \gamma L - \rho L \triangleq f_2(H,L,I,Y), \\
\dfrac{dI}{dt} = \gamma L - \rho I - gI \triangleq f_3(H,L,I,Y), \\
\dfrac{dY}{dt} = (b\dfrac{vI}{H+cL+vI} + \dfrac{\varepsilon vI(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2})(\dfrac{\Lambda}{m} - Y) - (\tau+m)Y \triangleq f_4(H,L,I,Y).
\end{cases} \tag{3.1}
$$

TABLE 1. Description of the variables and parameters for model (2.1).

Variable	Description
H	Density of susceptible and healthy potatoes
L	Density of incubation (infected but not infectious) period potatoes
I	Density of infected potatoes
X	Density of non-viruliferous aphids
Y	Density of viruliferous aphids
Parameter	**Description**
r	Net recruitment rate of healthy potatoes through plantation
K	Maximum carrying capacity of the field for the potatoes
ρ	Natural mortality rate in potatoes
γ	Intrinsic incubation rate in potatoes
g	Potatoes disease-induced death rate
Λ	Recruitment rate of aphids by birth in the crop field or by immigration
b	Proportion of aphids get infected due to birth on infectious potatoes
τ	Recovery rate of infectious aphids to be susceptible
m	Natural mortality rate in aphids
v	Bias of aphid to land on infected potatoes
c	Bias of aphid to land on incubation period potatoes
ω	Probability that aphid settles to feed on a susceptible potato
η	Bias of a aphid to settle to feed on an incubation period potato
ε	Bias of a aphid to settle to feed on an infected potato
Γ	Average time spent feeding on a potato when aphid chooses to settle for extended feed

Denote $R^4_+ \doteq \{(H,L,I,Y) \in R^4 : H,L,I,Y \geq 0\}$, and let $\gamma(t) = (H(t),L(t),I(t),Y(t))$ denote the solution orbit of system (3.1). Then we have the following lemmas.

Lemma 3.1. *The sets R^4_+ and its interior $\mathring{R}^4_+$ are the positively invariant of system (3.1).*

Proof. In order to prove the positively invariant of set R_+^4 and $\mathring{R}_+^4$, we only show that

$$(H_0,L_0,I_0,Y_0)\in R_+^4 \Longrightarrow \gamma(t)\in R_+^4 \text{ and } (H_0,L_0,I_0,Y_0)\in \mathring{R}_+^4 \Longrightarrow \gamma(t)\in \mathring{R}_+^4, \tag{3.2}$$

for all $t\geq 0$.

Indeed, if $\gamma(t)$ is initiated from H-axis with $H_0>0$, $L_0=I_0=Y_0=0$, then $f_2(H,0,0,0)=0$, $f_3(H,0,0,0)=0$ and $f_4(H,0,0,0)=0$ imply $L(t)=0$, $I(t)=0$ and $Y(t)=0$ for all $t>0$, and $\gamma(t)$ remains on the H-axis. Similarly, If $\gamma(t)$ is initiated from L-axis with $L_0>0$, $H_0=I_0=Y_0=0$, then $f_1(0,L,0,0)=0$, $f_3(0,L,0,0)>0$, and $f_4(0,L,0,0)=0$ imply $H(t)=0$, $I(t)>0$ and $Y(t)=0$ for all $t>0$ and $\gamma(t)$ enters into L-I plane. If $\gamma(t)$ is initiated from I-axis with $I_0>0$, $H_0=L_0=Y_0=0$, then $f_1(0,0,I,0)=0$, $f_2(0,0,I,0)=0$ and $f_4(0,0,I,0)>0$ imply $H(t)=0$, $L(t)=0$ and $Y(t)>0$ for all $t>0$, and $\gamma(t)$ enters into I-Y plane. If $\gamma(t)$ is initiated from Y-axis with $Y_0>0$, $H_0=L_0=I_0=0$, then $f_1(0,0,0,Y)=0$, $f_2(0,0,0,Y)=0$ and $f_3(0,0,0,Y)=0$ imply $H(t)=0$, $L(t)=0$ and $I(t)=0$ for all $t>0$, and $\gamma(t)$ remains on the Y-axis.

If $\gamma(t)$ is initiated from H-L plane with $H_0>0, L_0>0$, $I_0=Y_0=0$, then $I(t)>0$ and $Y(t)=0$ for all $t>0$, and $\gamma(t)$ enters into H-L-I plane. Similarly, if $\gamma(t)$ is initiated from H-I plane with $H_0>0, I_0>0$, $L_0=Y_0=0$, then $L(t)>0$ and $Y(t)>0$ for all $t>0$, and $\gamma(t)$ enters into $\mathring{R}_+^4$. If $\gamma(t)$ is initiated from H-Y plane with $H_0>0, Y_0>0$, $L_0=I_0=0$, then $L(t)>0$ and $I(t)=0$ for all $t>0$, and $\gamma(t)$ enters into H-L-Y plane. If $\gamma(t)$ is initiated from L-I plane with $L_0>0, I_0>0$, $H_0=Y_0=0$, then $H(t)=0$ and $Y(t)>0$ for all $t>0$, and $\gamma(t)$ enters into L-I-Y plane. If $\gamma(t)$ is initiated from L-Y plane with $L_0>0, Y_0>0$, $H_0=I_0=0$, then $H(t)=0$ and $I(t)>0$ for all $t>0$, and $\gamma(t)$ enters into L-I-Y plane. If $\gamma(t)$ is initiated from I-Y plane with $I_0>0, Y_0>0$, $H_0=L_0=0$, then $H(t)=0$ and $L(t)=0$ for all $t>0$, and $\gamma(t)$ remains on the I-Y plane.

Similarly, if $\gamma(t)$ is initiated from H-L-I, H-L-Y, H-I-Y and L-I-Y planes, then we have $f_1(0,L,I,Y)=0$, $f_2(H,0,I,Y)>0$, $f_3(H,L,0,Y)>0$ and $f_4(H,L,I,0)>0$, respectively, which corresponds $H(t)=0$, $L(t)>0$, $I(t)>0$ and $Y(t)>0$ in sequence for all $t>0$, and $\gamma(t)$ enters into L-I-Y plane.

Moreover, based on the uniqueness of solution of system (3.1), when $(H_0,L_0,I_0,Y_0)\in \mathring{R}_+^4$, $\gamma(t)$ will not touch the four axes in finite time, so $\gamma(t)$ remains in the inside of $\mathring{R}_+^4$. Thus, (3.2) is verified. □

Lemma 3.2. *All solutions of system (3.1) with the initial values from R_+^4 are uniformly bounded.*

Proof. Let $N_p(t) = H(t) + L(t) + I(t)$. The derivative of $N_p(t)$ along system (3.1) is

$$\begin{aligned} \frac{dN_p(t)}{dt} &= \frac{dH}{dt} + \frac{dL}{dt} + \frac{dI}{dt} \\ &= rH(1 - \frac{N_p(t)}{K}) - \rho(L+I) - gI \\ &\leq rH(1 - \frac{N_p(t)}{K}) \\ &\leq r\frac{N_p(t)}{K}(K - N_p). \end{aligned}$$

Then we have $\limsup_{t\to\infty} N_p(t) \leq K$. In addition, We know $\lim_{t\to\infty} N_v(t) = \frac{\Lambda}{m}$. Therefore, all solutions of system (3.1) with the initial values from R_+^4 are uniformly bounded. □

By Lemmas 3.1 and 3.2, we have following result.

Lemma 3.3. *If the initial conditions $(H(0), L(0), I(0), Y(0))^T$ of system (3.1) are in the region*

$$\Omega = \left\{ (H,\, L,\, I,\, Y) \in R_+^4 \mid 0 \leq H + L + I \leq K, 0 \leq Y \leq \frac{\Lambda}{m} \right\}, \tag{3.3}$$

then all solutions $(H(t), L(t), I(t), Y(t))^T$ will enter and remain in Ω.

Based on Lemma 3.3 , we can conclude that it is enough to analyze the dynamic properties of system (3.1) in Ω, which will be presented in the following subsections.

3.2. The Basic Reproduction Number.

In this section, we will calculate the basic reproduction number of system (3.1). It is easy to see that system (3.1) always has a disease-free equilibrium (the absence of infection, that is, $L = I = Y = 0$), $E_0 = (K, 0, 0, 0)$. Let $\mathscr{X} = (L, I, Y, H)^T$. Then model (3.1) can be written as

$$\frac{d\mathscr{X}}{dt} = \mathscr{F}(\mathscr{X}) - \mathscr{V}(\mathscr{X}),$$

where

$$\mathscr{F}(\mathscr{X}) = \begin{pmatrix} \dfrac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} \\ 0 \\ \left(b\dfrac{vI}{H+cL+vI} + \dfrac{\varepsilon vI(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2}\right)(\dfrac{\Lambda}{m}-Y) \\ 0 \end{pmatrix},$$

$$\mathscr{V}(\mathscr{X}) = \begin{pmatrix} \gamma L+\rho L \\ -\gamma L+\rho I+gI \\ (\tau+m)Y \\ -rH(1-\dfrac{H+L+I}{K})+\dfrac{YH(H+cL+vI)}{\omega\Gamma^2(H+\eta cL+\varepsilon vI)^2} \end{pmatrix}.$$

We can get

$$F = \begin{pmatrix} 0 & 0 & \dfrac{1}{\omega\Gamma^2} \\ 0 & 0 & 0 \\ 0 & \dfrac{\Lambda}{m}(\dfrac{vb}{K}+\dfrac{\varepsilon v}{\omega\Gamma^2 K}) & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \gamma+\rho & 0 & 0 \\ -\gamma & \rho+g & 0 \\ 0 & 0 & \tau+m \end{pmatrix}.$$

The next generation matrix for model (3.1) is described as

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \dfrac{1}{\omega\Gamma^2(\tau+m)} \\ 0 & 0 & 0 \\ \dfrac{\Lambda\gamma}{m(\gamma+\rho)(\rho+g)}(\dfrac{bv}{K}+\dfrac{\varepsilon v}{\omega\Gamma^2 K}) & \dfrac{\Lambda}{m(\rho+g)}(\dfrac{bv}{K}+\dfrac{\varepsilon v}{\omega\Gamma^2 K}) & 0 \end{pmatrix}.$$

It then follows that the spectral radius of matrix $r(FV^{-1}) = \sqrt{\frac{\Lambda v\gamma(b\Gamma^2\omega+\varepsilon)}{K\omega^2\Gamma^4 m(\tau+m)(\gamma+\rho)(\rho+g)}}$. According to Theorem 2 in [35], the basic reproduction number of model (3.1) is

$$R_0 = \sqrt{\frac{\Lambda v\gamma(b\Gamma^2\omega+\varepsilon)}{K\omega^2\Gamma^4 m(\tau+m)(\gamma+\rho)(\rho+g)}}. \tag{3.4}$$

Theorem 3.1. *The disease-free equilibrium E_0 is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.*

Proof. Evaluating the Jacobian matrix of system (3.1) at E_0:

$$J(E_0) = \begin{pmatrix} -r & -r & -r & -\frac{1}{\omega\Gamma^2} \\ 0 & -(\gamma+\rho) & 0 & \frac{1}{\omega\Gamma^2} \\ 0 & \gamma & -(\rho+g) & 0 \\ 0 & 0 & \frac{\Lambda}{m}(\frac{bv}{K}+\frac{v\varepsilon}{K\omega\Gamma^2}) & -(\tau+m) \end{pmatrix}.$$

Thus the characteristic equation gives

$$(\lambda+r)(\lambda^3+b_1\lambda^2+b_2\lambda+b_3)=0, \tag{3.5}$$

where

$$\begin{aligned} b_1 &= \tau+m+\rho+g+\gamma+\rho>0, \\ b_2 &= (\rho+g)(\tau+m)+(\gamma+\rho)(\tau+m)+(\gamma+\rho)(\rho+g)>0, \\ b_3 &= (\gamma+\rho)(\rho+g)(\tau+m)-\frac{\gamma\Lambda}{\omega\Gamma^2 m}\left(\frac{bv}{K}+\frac{v\varepsilon}{K\omega\Gamma^2}\right) \\ &= \frac{v\gamma\Lambda(b\omega\Gamma^2+\varepsilon)}{K\omega^2\Gamma^4 m R_0^2}-\frac{v\gamma\Lambda}{K\omega\Gamma^2 m}\left(b+\frac{\varepsilon}{\omega\Gamma^2}\right) \\ &= \frac{v\gamma\Lambda(b\omega\Gamma^2+\varepsilon)}{K\omega^2\Gamma^4 m}\left(\frac{1}{R_0^2}-1\right). \end{aligned}$$

According to the Routh-Hurwitz criterion, we obtain that E_0 is locally asymptotically stable provided that $R_0<1$; whereas if $R_0>1$, equation (3.5) has a positive eigenvalue. Hence E_0 becomes unstable. □

Remark 3.1. Theorem 3.1 illustrates that the disease may be go to the state of extinction if $R_0<1$; otherwise, the disease will be endemic if $R_0>1$.

3.3. Bifurcation Analysis. In this section we investigate the center manifold near the criticality (at E_0 and $R_0=1$) by using the approach developed in [35-37], which is based on the general center manifold theory [38]. The approach establishes that the normal form representing the dynamics of the system on the center manifold is given by

$$\dot{u} = Au^2+B\mu u+\mathscr{O}(3), \tag{3.6}$$

where

$$A = \frac{v}{2} D_{xx} f(x_0, 0) w^2 = \frac{1}{2} \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(x_0, 0), \tag{3.7}$$

$$B = v D_{x\xi} f(x_0, 0) w = \sum_{k,i}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \xi}(x_0, 0). \tag{3.8}$$

Note that, in (3.6), μ denotes a bifurcation parameter such that $R_0 < 1$ for $\mu < 0$ and $R_0 > 1$ for $\mu > 0$, the notation $\mathscr{O}(3)$ is used to denote terms of third order and higher in u and μ. In (3.7) and (3.8), the term ξ denotes a bifurcation parameter to be chosen, f_i denote the right-hand side of system (3.1), x denotes the state vector, x_0 denotes the disease-free equilibrium E_0 and v and w denote the left and right eigenvectors, respectively, corresponding to the null eigenvalue of the Jacobian matrix of system (3.1) evaluated at the criticality. Now we take b as a bifurcation parameter. Solving for b from $R_0 = 1$, we get

$$b = \bar{b} = \frac{K\omega^2\Gamma^4 m(\gamma+\rho)(\tau+m)(\rho+g) - \Lambda v\gamma\varepsilon}{\Lambda v\gamma\omega\Gamma^2}.$$

In view of Theorem 4 in [35], we know that a local stability analysis of (3.7) shows that $B > 0$. Therefore, the nature of the bifurcation at $b = \bar{b}$ is given by the sign of coefficient (3.7). By [35-37], we can conclude that, if $A > 0$, then system (3.1) exhibits a backward bifurcation at $R_0 = 1$. If $A < 0$, then the system exhibits a forward bifurcation at $R_0 = 1$.

Denote

(3.9)

$$\begin{aligned}\Theta &= \frac{\Lambda v\gamma(r\gamma + (r+\rho+\gamma)(\rho+g))\left(\bar{b}\omega\Gamma^2+\varepsilon\right)}{K\omega^2\Gamma^4 mr(g+\rho)^2(\gamma+\rho)} + \frac{\Lambda cv\gamma\left(-\bar{b}\omega\Gamma^2+\varepsilon(1-2\eta)\right)}{K\omega^2\Gamma^4 m(\rho+g)(\gamma+\rho)} \\ &+ \frac{\Lambda v^2\gamma^2\left(-\bar{b}\omega\Gamma^2+\varepsilon(1-2\varepsilon)\right)}{K\omega^2\Gamma^4 m(\gamma+\rho)(g+\rho)^2} + (m+\tau)\left(c(1-2\eta) + \frac{v\gamma(1-2\varepsilon)}{g+\rho}\right) - \frac{v\gamma(\bar{b}\omega\Gamma^2+\varepsilon)}{\omega\Gamma^2(g+\rho)}.\end{aligned}$$

Theorem 3.2. *If $\Theta > 0$, then system (3.1) exhibits a backward bifurcation at $R_0 = 1$. If the reversed inequality holds, then the system exhibits a forward bifurcation at $R_0 = 1$.*

Proof. Let us begin by observing that the matrix

$$J(E_0,\bar{b}) = \begin{pmatrix} -r & -r & -r & -\frac{1}{\omega\Gamma^2} \\ 0 & -(\gamma+\rho) & 0 & \frac{1}{\omega\Gamma^2} \\ 0 & \gamma & -(\rho+g) & 0 \\ 0 & 0 & \frac{\omega\Gamma^2(\gamma+\rho)(\tau+m)(\rho+g)}{\gamma} & -(\tau+m) \end{pmatrix},$$

admits a simple zero eigenvalue and the other eigenvalues are real and negative. Hence, when $b=\bar{b}$ (or, equivalently, when $R_0=1$), the disease-free equilibrium E_0 is a non-hyperbolic equilibrium. Denote by $v=(v_1,v_2,v_3,v_4)$, and $w=(w_1,w_2,w_3,w_4)^T$, a left and a right eigenvector associated with the zero eigenvalue, respectively, such that $v\cdot w=1$. We get:

$$v_1=0,\ \ v_2=\omega\Gamma^2(\tau+m)v_4,\ \ v_3=\frac{\omega\Gamma^2(\gamma+\rho)(\tau+m)}{\gamma}v_4,\ \ v_4=v_4.$$

$$w_1=-\frac{r\gamma+(r+\gamma+\rho)(\rho+g)}{r\omega\Gamma^2(\gamma+\rho)(\rho+g)}w_4,\ \ w_2=\frac{1}{\omega\Gamma^2(\gamma+\rho)}w_4,\ \ w_3=\frac{\gamma}{\omega\Gamma^2(\gamma+\rho)(\rho+g)}w_4,\ \ w_4=w_4.$$

We can choose w_4 and v_4 satisfy

$$w_4\cdot v_4=\left[\frac{(\tau+m)(\gamma+g+2\rho)}{(\gamma+\rho)(\rho+g)}+1\right]^{-1}>0,$$

such that $v\cdot w=1$.

Taking into account of system (3.1) and considering only the nonzero components of the left eigenvector v, it follows that

$$\begin{aligned} A &= v_2\left[w_2w_4\frac{\partial^2 f_2}{\partial L\partial Y}(x_0,\bar{b})+w_3w_4\frac{\partial^2 f_2}{\partial I\partial Y}(x_0,\bar{b})\right] \\ &+ v_4\left[w_1w_3\frac{\partial^2 f_4}{\partial H\partial I}(x_0,\bar{b})+w_2w_3\frac{\partial^2 f_4}{\partial L\partial I}(x_0,\bar{b})+w_3w_4\frac{\partial^2 f_4}{\partial I\partial Y}(x_0,\bar{b})\right] \\ &+ \frac{v_4}{2}w_3w_3\frac{\partial^2 f_4}{\partial I\partial I}(x_0,\bar{b}). \end{aligned}$$

Now it can be checked that

$$\frac{\partial^2 f_2}{\partial L\partial Y}(x_0,\bar{b})=\frac{c(1-2\eta)}{K\omega\Gamma^2},\frac{\partial^2 f_2}{\partial I\partial Y}(x_0,\bar{b})=\frac{v(1-2\varepsilon)}{K\omega\Gamma^2},$$

and

$$\frac{\partial^2 f_4}{\partial H\partial I}(x_0,\bar{b})=-\frac{\Lambda v}{mK^2}(\bar{b}+\frac{\varepsilon}{\omega\Gamma^2}),\frac{\partial^2 f_4}{\partial L\partial I}(x_0,\bar{b})=\frac{\Lambda cv}{mK^2}[-\bar{b}+\frac{\varepsilon(1-2\eta)}{\omega\Gamma^2}],$$

and

$$\frac{\partial^2 f_4}{\partial I \partial I}(x_0,\bar{b}) = \frac{2\Lambda v^2}{mK^2}[-\bar{b} + \frac{\varepsilon(1-2\varepsilon)}{\omega\Gamma^2}], \frac{\partial^2 f_4}{\partial I \partial Y}(x_0,\bar{b}) = -\frac{v}{K}(\bar{b} + \frac{\varepsilon}{\omega\Gamma^2}).$$

Thus, we have

$$A = \frac{v_4 w_4^2}{\omega K \Gamma^2(\gamma+\rho)}\Theta,$$

where Θ is defined in (3.9). Therefore, system (3.1) exhibits backward or forward bifurcation at $R_0 = 1$ according to the sign of Θ.

□

4. NUMERICAL SIMULATION

In this section, we first provide results from numerical simulations of model (2.1) that demonstrate and support our theoretical results, and then study the influence of the landing and feeding preferences on the PVY spread. We need to estimate the model parameters in order to carry out the numerical simulations. The values of parameters of model (2.1) are given in Table 2.

Fig. 3 shows a backward bifurcation occurs at $b = \bar{b}$. There exists two threshold values of b, namely b^{crit} and $\bar{b}$. We also observe that the disease-free equilibrium E_0 is the only equilibrium for system (2.1) for $b < b^{crit}$; a couple of endemic equilibria coexist with the disease-free equilibrium E_0 for $b^{crit} < b < \bar{b}$; system (2.1) exhibits the disease-free equilibrium and the larger endemic equilibrium for $b > \bar{b}$; a backward bifurcation occurs for $b = \bar{b}$.

Next, in order to show the effects of vector landing preferences v, c and vector feeding preferences η, ε on the total infective population. On the t-axis, the time index, ranges from 0 to 1200, we fix the different panels refer to different values of $v = c$ and $\varepsilon = \eta$ that assume respectively the values of (0.5, 1, 1.5, 4), respectively. The numerical results (see Fig. 4) show that there is high infected value with vector landing preferences ($v = c = 4$) and vector feeding preferences ($\varepsilon = \eta = 4$); however, there are low infected values with vector landing preferences ($v = c = 0.5$ and $v = c = 1$) and vector feeding preferences ($\varepsilon = \eta = 0.5$ and $\varepsilon = \eta = 1$). It shows that vector landing and feeding preferences play a key role in PVY transmission in potatoes.

From Fig. 5, we can observe that the basic reproduction number R_0 is independent on the values of the landing and feeding preferences of aphids on incubation potato plants c and η. However, the final disease incidence $\frac{L_\infty + I_\infty}{H_\infty + L_\infty + I_\infty}$ decreases as c or η increases. Because the

TABLE 2. Parameters to be used in the system (2.1) and numerical simulations.

Parameter	Baseline values	Unit	Reference
r	7.3613	day^{-1}	Assumed
K	108.6110	$plant$	[31,39]
ρ	3.7361	day^{-1}	[34]
γ	0.1590	day^{-1}	[40]
g	2.1806	day^{-1}	[34]
Λ	4.1329	$vector \times day^{-1}$	[39]
b	1.5014	day^{-1}	[19]
τ	6.3354	day^{-1}	[41]
m	0.1731	day^{-1}	[31]
v	0.0895	-	Assumed
c	3.9271	-	Assumed
ω	0.1023	-	Assumed
η	0.5755	-	Assumed
ε	0.7181	-	Assumed
Γ	0.1509	day	Assumed

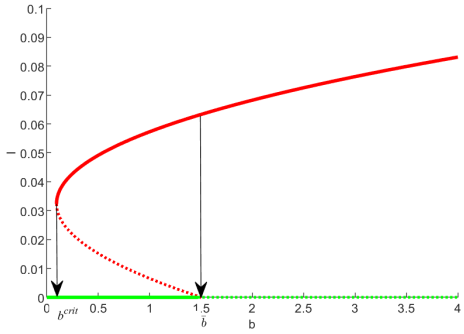
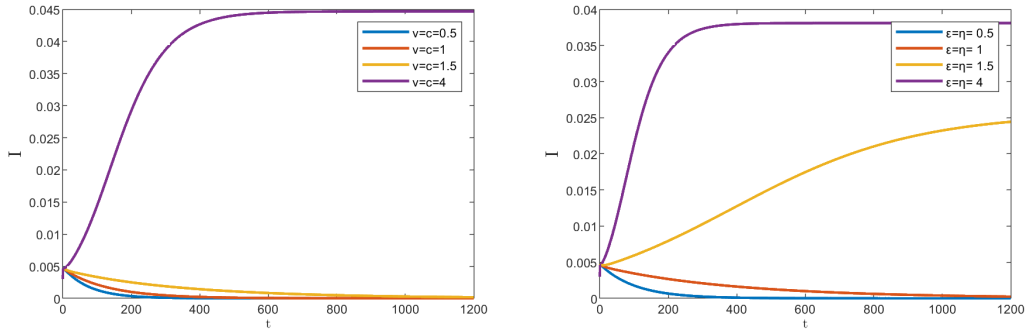
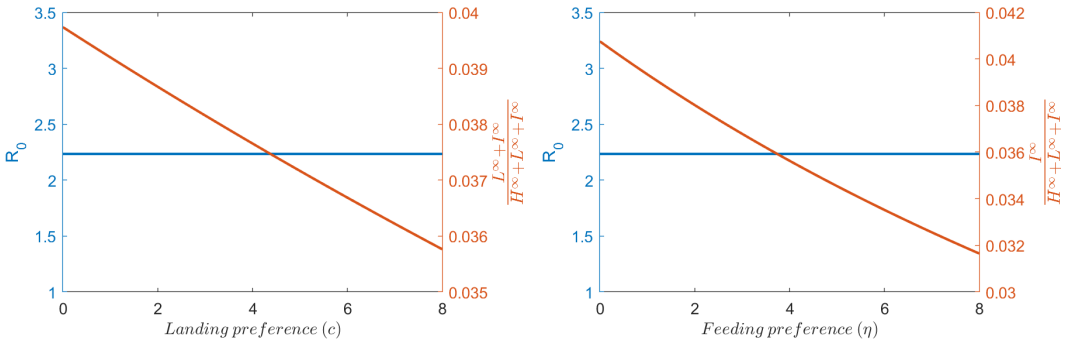


FIGURE 3. Backward bifurcation diagram in the plan (b, I). In this diagram, solid lines indicate stability and the dashed lines indicate instability.



(a) (b)

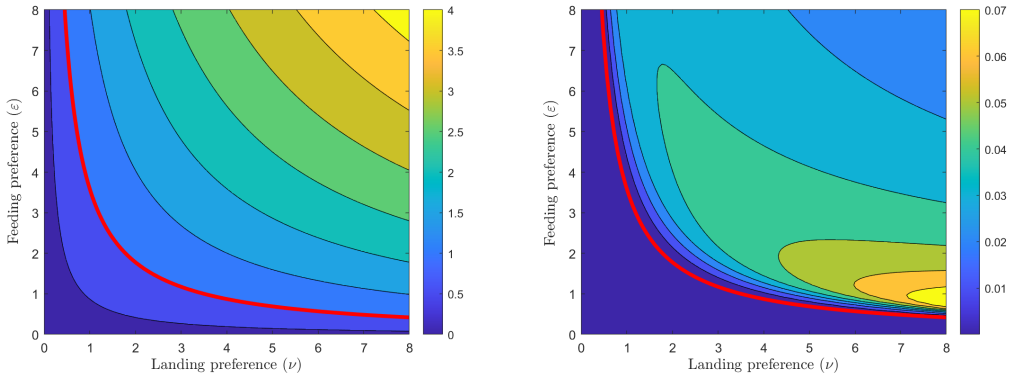
FIGURE 4. The plots of time series: influence of the vector preferences on the infected host population. (a) $v = c = 0.5,\ v = c = 1,\ v = c = 1.5,\ v = c = 4$; (b) $\varepsilon = \eta = 0.5,\ \varepsilon = \eta = 1,\ \varepsilon = \eta = 1.5,\ \varepsilon = \eta = 4$.



(a) (b)

FIGURE 5. Responses of the basic reproduction number (blue) and equilibrium incidence (red) to changes in the vector preference parameters: (a) landing preference c; (b) feeding preference η.

incubation potato plants do not spread the virus, so vector preference behavior to incubation host will not affect the basic reproductive number, but they will have an impact on the final disease incidence.



(a) (b)

FIGURE 6. The responses of the basic reproduction number are simultaneously altered as both landing preference v and feeding preference ε. Pairs of parameters for which $R_0 = 1$ are again marked with a red curve. (a) Isocline of the basic reproduction number; (b) Isocline of the final disease incidence.

The effect of landing and feeding preferences to infected host v and ε on the basic reproduction number and final disease incidence is shown in Fig. 6. It is observed in Fig. 6(a) (left panel) that the effect of vector preference on R_0 which is given in equation (3.4). The red line indicates the threshold value where $R_0 = 1$, and the different colors indicate increasing values of R_0 from 0 to 4, with 0.5 steps (blue to yellow). As vector landing and feeding preferences on infected host increase, R_0 increases. The dark blue area indicates the parameter region where the pathogen is always driven to extinction. Fig. 6(b) (right panel) shows how final disease incidence is influenced by landing and feeding preferences on infected host. From Fig. 6(b), we can see that, when landing preference v is very low, the final disease incidence is monotonically increasing with feeding preference ε increasing (see Fig. 7a); whereas when $v > 1.8$, the final disease incidence is initially monotonically increasing to peak value, and then monotonically decreasing with ε increasing (see Fig. 7b). Similar phenomena are observed regarding parameter v as well. The influence of vector behavior on infection incidence can lead to the change in the final disease scale trend. Indeed, when landing preference v is large and the aphid has a strong feeding preference ε for infected potatoes, the probability of viruliferous aphids feeding on infected potato plants is becoming large. This leads to reduce the final disease incidence.

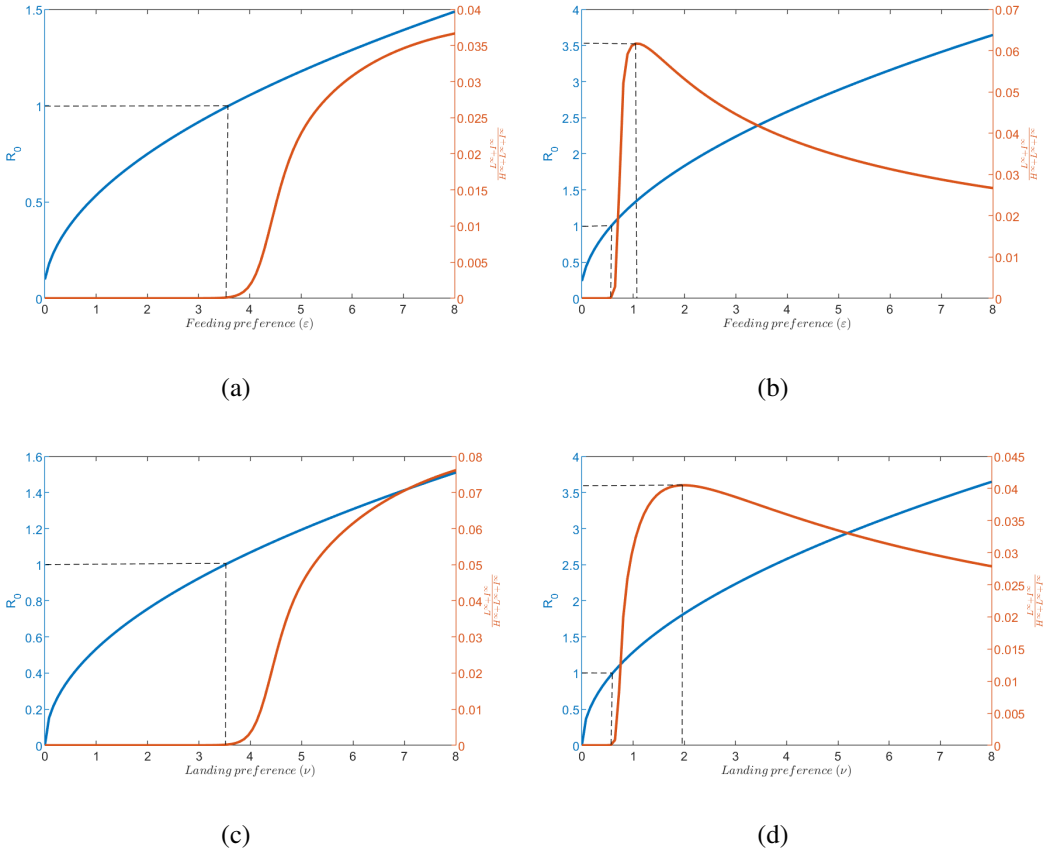


FIGURE 7. Responses of the basic reproduction number (blue) and final disease incidence (red) to changes in the vector preference of aphids on infected potato plants: (a) feeding preference ε, fix $v = 1$; (b) feeding preference ε, fix $v = 6$; (c) landing preference v, fix $\varepsilon = 1$; (d) landing preference v, fix $\varepsilon = 6$.

5. CONCLUSION

In this paper, we propose and analyze a vector-borne epidemic model for PVY by incorporating preference behavior and vertical transmission of vector. We compute the basic reproduction number and discuss the local stability of the disease-free equilibrium. When we choose appropriate parameters, there exists a backward bifurcation occurs at $b = \bar{b}$. If $b > \bar{b}$, then there is a unique endemic equilibrium and the disease is uniformly persistent, whereas if $b < \bar{b}$, there may be two endemic equilibria, and the endemic equilibrium can coexist with the disease-free equilibrium. This illustrates that $b < \bar{b}$ (i.e. $R_0 < 1$) cannot ensure the eradication of the disease, and decreasing b below the sub-threshold $b = b^{crit}$ would be a propositional control strategy.

If $b^{crit} < b < \bar{b}$, only when the numbers of infected cases are small enough, it is a sufficient condition to eliminate PVY. The numerical results indicate that the landing and feeding preferences of aphids on infected potato plants will lead to a more efficient transmission of PVY virus. However, the final infection incidence of PVY may not necessarily increase with an increase of the preference parameter (v or ε). Further, the number simulation also shows that R_0 is independent of the landing and feeding preferences of aphids on incubation potato plants, but the final infection incidence decreases with an increase of the preference parameter (c or η).

Our investigations suggest that the landing and feeding preferences of aphids on infected potato plants play an important role in the spread of PVY, and phenotypic preference of aphid for infected population may increase the risk of PVY transmission. Vertical transmission dramatically affect the disease transmission dynamics. The result strongly suggests and supports the previous observations.

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

REFERENCES

[1] P. Andret-Link, M. Fuchs, Transmission specificity of plant viruses by vectors, J. Plant. Pathol. 87 (2005), 153–165. http://www.jstor.org/stable/41998234.

[2] T. Hohn, Plant virus transmission from the insect point of view, Proc. Natl. Acad. Sci. U.S.A. 104 (2007), 17905–17906. https://doi.org/10.1073/pnas.0709178104.

[3] A.G. Power, Insect transmission of plant viruses: a constraint on virus variability, Curr. Opin. Plant Biol. 3 (2000), 336–340. https://doi.org/10.1016/s1369-5266(00)00090-x.

[4] S.E. Seal, F. vandenBosch, M.J. Jeger, Factors influencing begomovirus evolution and their increasing global significance: implications for sustainable control, Critic. Rev. Plant Sci. 25 (2006), 23–46. https://doi.org/10.1080/07352680500365257.

[5] D. Eriksson, U. Carlson-Nilsson, R. Ortíz, et al. Overview and breeding strategies of table potato production in sweden and the Fennoscandian region, Potato Res. 59 (2016), 279–294. https://doi.org/10.1007/s11540-016-9328-6.

[6] C. Kerlan, B. Moury, Potato virus Y, in: Encyclopedia of Virology, Elsevier, 2008: pp. 287–296. https://doi.org/10.1016/B978-012374410-4.00737-8.

[7] J.A. De Bokx, H. Huttinga, Potato virus Y, Descriptions of plant viruses, No. 242. Commonw Mycol Inst/Assoc Appl Biol, Kew, (1981).

[8] D.E. Van der Zaag, Yield reduction in relation to virus infection, in: J.A. De Bokx, J.P.H. Van der Want (Eds.), Viruses of potatoes and seed-potato production, Pudoc (Centre for Agricultural Publishing and Documentation), Wageningen, pp. 146-150, (1987).

[9] J.L. Whitworth, P. Nolte, C. McIntosh, et al. Effect of potato virus Y on yield of three potato cultivars grown under different nitrogen levels, Plant Dis. 90 (2006), 73–76. https://doi.org/10.1094/pd-90-0073.

[10] J.A. De Bokx, Potato virus Y, in: W.J. Hooker (Eds.), Compendium of potato diseases, American Phytopathological Society, St. Paul, pp. 70-71, (1986).

[11] W.J. Hooker (ed). Compendium of potato diseases, American Phytopathological Society, St. Paul, Minnesota, (1981).

[12] P.G.M. Piron, New aphid vectors of potato virus yn, J. Plant. Pathol. 92 (1986), 223-229.

[13] F.A. Klingauf, Host plant feeding and acceptance, in: A.K. Minks, P. Harrewijn (Eds.), Aphids: their biology, natural enemies, and control, Elsevier, Amsterdam, pp. 209–224, (1987).

[14] F.A. Klingauf, Feeding, adaptation and excretion, in: A.K. Minks, P. Harrewijn (Eds.), Aphids: their biology, natural enemies, and control, Elsevier, Amsterdam, pp. 225–253, (1987).

[15] F.A. Klingauf, Die fr*ü*hphase in der wirtswahl von bl*ä*ttlausen, Meded. Fac. Land-bouwwet. Gent. 40 (1975), 351-364.

[16] D.G. Pollard, Aphid penetration of plant tissues, in: K.F. Harris, K. Maramorosch (Eds.), Aphids as virus vectors, Academic Press, New York, pp. 105–118, (1977).

[17] N.A. Bosque-Pérez, S.D. Eigenbrode, The influence of virus-induced changes in plants on aphid vectors: Insights from luteovirus pathosystems, Virus Res. 159 (2011), 201–205. https://doi.org/10.1016/j.virusres.2011.04.020.

[18] A. Galimberti, A. Alyokhin, H. Qu, J. Rose, Simulation modelling of potato virus Y spread in relation to initial inoculum and vector activity, J. Integr. Agric. 19 (2020), 376–388. https://doi.org/10.1016/s2095-3119(19)62656-0.

[19] D.S. Degefa, O.D. Makinde, D.T. Temesgen, Modeling potato virus Y disease dynamics in a mixed-cropping system, Int. J. Model. Simul. 42 (2021), 370–387. https://doi.org/10.1080/02286203.2021.1919818.

[20] S.T. Degefa, O.D. Makinde, T.T. Dufera, Mathematical model of potato virus Y disease spread with optimal control, Math. Model. Anal. 27 (2022), 408–428. https://doi.org/10.3846/mma.2022.15077.

[21] J. Moore, Parasites and the behavior of animals, Oxford University Press, New York, 2002.

[22] T. Lefèvre, F. Thomas, Behind the scene, something else is pulling the strings: Emphasizing parasitic manipulation in vector-borne diseases, Infect. Genetics Evol. 8 (2008), 504–519. https://doi.org/10.1016/j.meegid.2007.05.008.

[23] M.J. Lehane, The biology of blood-sucking in insects, Cambridge University Press, Cambridge, 2005.

[24] F. Chamchod, N.F. Britton, Analysis of a vector-bias model on malaria transmission, Bull. Math. Biol. 73 (2010), 639–657. https://doi.org/10.1007/s11538-010-9545-0.

[25] J.G. Kingsolver, Mosquito host choice and the epidemiology of Malaria, Amer. Naturalist. 130 (1987), 811–827. https://doi.org/10.1086/284749.

[26] G. Marini, R. Rosá, A. Pugliese, H. Heesterbeek, Exploring vector-borne infection ecology in multi-host communities: A case study of West Nile virus, J. Theor. Biol. 415 (2017), 58–69. https://doi.org/10.1016/j.jtbi.2016.12.009.

[27] P. McElhany, L.A. Real, A.G. Power, Vector preference and disease dynamics: a study of barley yellow dwarf virus, Ecology. 76 (1995), 444–457. https://doi.org/10.2307/1941203.

[28] K. Clay, The effect of fungi on the interaction between host plants and their herbivores, Can. J. Plant Pathol. 9 (1987), 380–388. https://doi.org/10.1080/07060668709501871.

[29] M.E. Irwin, J.M. Thresh, Epidemiology of barley yellow dwarf: A study in ecological complexity, Annu. Rev. Phytopathol. 28 (1990), 393–424. https://doi.org/10.1146/annurev.py.28.090190.002141.

[30] R.E.F. Matthews, Plant virology, Elsevier, Amsterdam, 2012.

[31] M.J. Jeger, F. Van Den Bosch, L.V. Madden, et al. A model for analysing plant-virus transmission characteristics and epidemic development, Math. Med. Biol. 15 (1998), 1–18. https://doi.org/10.1093/imammb/15.1.1.

[32] R.D. Davidson, A.J. Houser, K. Sather, et al. Controlling PVY in seed: what works and what does not, Amer. J. Potato Res. 90 (2013), 28–32. https://doi.org/10.1007/s12230-012-9290-z.

[33] J.L. Whitworth, P.B. Hamm, C.S. McIntosh, Effect of potato virus Y on yield of a clonal selection of russet norkotah, Amer. J. Pot Res. 87 (2010), 310–314. https://doi.org/10.1007/s12230-010-9134-7.

[34] N.J. Cunniffe, N.P. Taylor, F.M. Hamelin, et al. Epidemiological and ecological consequences of virus manipulation of host and vector in plant virus transmission, PLoS Comput. Biol. 17 (2021), e1009759. https://doi.org/10.1371/journal.pcbi.1009759.

[35] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Mathematical Biosciences. 180 (2002), 29–48. https://doi.org/10.1016/s0025-5564(02)00108-6.

[36] C. Castillo-Chavez, B. Song, Dynamical models of tuberculosis and their applications, Math. Biosci. Eng. 1 (2004), 361–404. https://doi.org/10.3934/mbe.2004.1.361.

[37] J. Dushoff, W. Huang, C. Castillo-Chavez, Backwards bifurcations and catastrophe in simple models of fatal diseases, J. Math. Biol. 36 (1998), 227–248.

[38] J. Guckenheimer, P. Holmes, Nonlinear oscillations, dynamical systems, and bifurcations of vector fields, Springer-Verlag, New York, 1983.

[39] J.M. Hily, A. García, A. Moreno, et al. The relationship between host lifespan and pathogen reservoir potential: an analysis in the system arabidopsis thaliana-Cucumber mosaic virus, PLoS Pathog. 10 (2014), e1004492. https://doi.org/10.1371/journal.ppat.1004492.

[40] K.S. Choi, F. del Toro, F. Tenllado, et al. A model to explain temperature dependent systemic infection of potato plants by potato virus Y, Plant Pathol. J. 33 (2017), 206–211. https://doi.org/10.5423/ppj.nt.06.2016.0144.

[41] R.H.E. Bradley, Loss of virus from the stylets of aphids, Virology. 8 (1959), 308–318. https://doi.org/10.1016/0042-6822(59)90032-7.