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Mathematical Model of Potato Virus Y Disease Spread with Optimal Control

Shambel Tadesse Degefa[a], **Oluwole Daniel Makinde**[b] **and Tamirat Temesgen Dufera**[a]

[a] *Department of Applied Mathematics, Adama Science and Technology University, Adama, Ethiopia*
[b] *Faculty of Military Science, Stellenbosch University, Stellenbosch, South Africa*

E-mail(*corresp.*): nanashambel2012@gmail.com

E-mail: makinded@gmail.com

E-mail: tamirat.temesgen@astu.edu.et

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Abstract. Potato virus Y (PVY) is an aphid-borne plant virus that causes substantial yield losses in potato production. Control measures of the viral infection are both limited and expensive. A proper use of mixed-cropping strategy can reduce the spread of PVY. In this paper, we formulate and analyze a mathematical model of PVY spread in a mixed-cropping system. Then, we extend the model to an optimal control problem by considering use of mineral oil, insecticide and farmer's level of field inspection for infected plants. The analytic results show that the basic reproduction number $\Re_0$, a threshold parameter that decides properties of the dynamics. The disease free equilibrium is stable if $\Re_0 < 1$ and unstable when $\Re_0 > 1$. It is found that $\Re_0$, and hence, the disease dynamics is highly sensitive to the representative parameters of density the non-host plant and its quality in attracting vectors. The model exhibits forward bifurcation at $\Re_0 = 1$. The study of optimal control problem suggests that mixed-cropping combined with either mineral oil or insecticide is the best to control the disease. Furthermore, simulation results show that mixed-cropping can be used as an alternative strategy and can reduce the need of mineral oil or insecticide.

Keywords: PVY, mixed-cropping, mathematical model, optimal control.

AMS Subject Classification: 34C60; 92D30; 37M05; 97M10.

1 Introduction

Potato (Solanum tuberosum L.) is the most important food crop ranking third in production [22]. It is a highly recommended food security and cash crop in Sub-Saharan Africa [2,11,25]. In recent years, seed certification and advances in crop protection have made it possible to increase potato yield and production. Nonetheless, problems with viral diseases such as Potato virus Y (PVY) are still prevalent, especially in developing countries, and can cause huge losses of the annual harvest [3, 23, 27]. PVY belongs to the family Potyviradae genus Potyvirus and infection in potato causes seed degeneration and yield reduction [11, 13, 21].

Transmission by aphid vectors is the most important means of PVY spread in potatoes crop. It can be vectored by several species of colonizing and non-colonizing aphids in a non-persistent manner, which means, a brief superficial probes of few seconds is sufficient for acquisition or inoculation. Aphids usually cease to be infective within 4–8 hours after acquisition [6, 7, 10, 12, 16, 21].

Control measures of PVY are both limited and expensive. Use of certified seed is the best to avoid field infection. The virus can also be managed by controlling its aphid vectors by insecticides and mineral oils [19]. However, these chemical requires repeated applications and their success strongly depends on the environmental conditions, density and species of the aphid vectors involved. Aphids can also be controlled by cultural methods such as mulching, border-cropping and mixed-cropping [10, 12, 21]. These strategies are based on manipulation of the search and feeding behaviors of aphids and the transmission mechanism of the virus. Aphids use visual and chemical cues to locate the host habitat. After landing on a plant, recognition as a host (or rejection) follows only after brief superficial probes of few seconds which is sufficient for acquisition or inoculation [5, 16, 21].

Mixed-cropping is an agricultural practice of planting the virus-host (potato in our case) with a non-host crop (sometimes called barrier crop) plants. The efficiency of mixed-cropping strategy is determined by the quality of the barrier plant in attracting vectors. In order to achieve the desired goal, the barrier plant species should be selected so that it is appealing to vectors and taller than the potato crop. It was reported that maize, sorghum, sunflower, vetch and oat can be used [10, 12]. The barrier crop reduces the spread of PVY in potatoes crop by obstructing the transmission processes in many ways; by attracting vectors, acting as a physical barrier between the host and vectors and acting as a vector-trap and virus-sink crop, i.e., the virus is lost from an infective vector when it feeds on the plant [12].

Currently, modeling plant diseases dynamics has received considerable attention. Models of vector-born plant diseases have been constructed and studied in an attempt to identify important factors which affects the spread, for example [4, 14, 15, 26, 28]. However, the protective nature of mixed-cropping system against a host plant infection with non-persistently transmitted vector-borne plant viral diseases has been rarely described and studied through mathematical models. We are not familiar with models of PVY or other non-persistent viruses which consider mixed-cropping practice as a control strategy

except the work in [9]. In this work, the impact of mixed-cropping system on the disease dynamics was expressed by its effect on the basic reproduction number $\Re_0$. However, the study did not consider other possible ways such as vector-trap effects in during the acquisition process. Also, the efficiencies of mixed-cropping and chemical controls are not investigated and compared by extending the model to an optimal control problem. It is important to study the potential of mixed-cropping strategy in controlling the disease and its significance in reducing the need for chemical controls.

As a result, in this paper, we propose a model of PVY disease dynamics in a mixed-cropping system by describing possible way by which a non-host plant can reduce the virus spread in the host plants. Then, we extend the model to an optimal control problem by considering insecticide and mineral oil, and the farmers rate of field inspection for roguing infected plants as controls. The paper is organized as follows: Section 2 is devoted to model construction. Analysis of the formulated model is performed in Section 3. An optimal control problem is studied in Section 4. In Section 5, numerical simulations are demonstrated. Finally, discussion and conclusions are given in Section 6.

2 Model description

The proposed model of PVY disease spread consists of two plant species; the host potato and a non-host crop plants. Being transmitted by the aphid vectors, the viral infection affects only the potato but not the non-host plant. Therefore, in the model, there are three populations: the potato plant N_p, the non-host plant Q, and the aphid vector N_v. We assume that the non-host plant Q is attractive to vectors, planted earlier, taller and has longer maturation period than the host. It is distributed uniformly in the field and constant during the potatoes crop growing period.

There are three, respectively two compartments of the host plant and vector population. These are; susceptible host H, latent (not infectious) host L, and infected (infectious) I host; and susceptible (virus-free) vector X and infective (carrier) vector Y. Accordingly, the total number of host plant and vector populations are given by $N_p(t) = H(t)+L(t)+I(t)$ and $N_v(t) = X(t)+Y(t)$ at any time t, respectively. Susceptible host plants are recruited into the crop field by plantation rate r in the available space constrained by abundance of the host crop and the maximum carrying capacity of the crop field $K, K > 0$. That is, $r(K-H-L-I)$ is a replenishment rate of susceptible plant. The host plants, irrespective of their status, are subject to natural death rate d. Susceptible vectors are recruited either by birth (in the crop field) or by immigration rate Λ. Vectors are not affected by the viral infection but subject to natural death or emigration rate m.

A susceptible host plant can get infected and move to latent class only when inoculated with the virus by infective vectors. Latent (exposed) plants can progress to the infected class with rate γ. Infected plants neither die of nor recover from the disease but subject to roguing rate g. A susceptible vector can acquire the virus and becomes infective only when it feeds/probes on infected plants. Infective vectors will lose the virus and become susceptible

both naturally at rate $a\tau$ and when feeding/probing on Q [7, 12].

The protective nature of Q against the vector-mediated infection of host plants is represented in the inoculation and acquisition processes. Because, in a mixed-cropping situation, Q affects the acquisition and inoculation processes in several ways including; (i) by attracting vector, (ii) acting as a virus-sink and/or vector-trap, and (iii) acting as a physical/chemical barrier between the host crop and the aphid vectors [6, 7, 10, 12].

We assume that the host plant infection is a vector-dependent saturating incidence, $\frac{a_1\beta_1 HY}{1+(\alpha_1+a_3q_1Q)Y}$. Because an infective vector will lose the virus naturally after few probes on the healthy host and the Q plants [7, 10, 12]. The acquisition incidence is assumed to follow a saturation functional response, $\frac{a_2\beta_2 IX}{1+\alpha_2 I+a_3q_2QX}$. The parameter $\alpha_1(\alpha_2)$ denotes the effect of sustained feeding on the infectivity of Y (acquisition efficiency of X), respectively [6, 7]. Similarly, the terms a_3q_1QY and a_3q_2QX denote the loss of virus from Y due to feeding on Q and the interference of Q on X during acquisition, respectively or the vector-trap effect of Q. Finally, the virus-sink process from Y due to feeding on Q is given by a vector-dependent saturating response $\frac{a_3\beta_3 QY}{1+\alpha_3 Y}$. Based on the above assumptions and from the schematic diagram Figure 1, the transmission dynamics of PVY disease is given by the following system of ordinary differential equations:

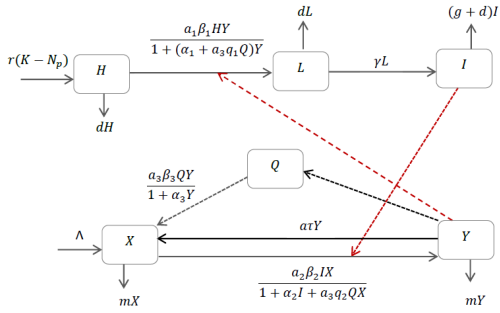


Figure 1. Schematic diagram of the formulated PVY disease model in a mixed-cropping system comprised of host and non-host crop plants.

$$\begin{cases} \dfrac{dH}{dt} = r\big(K-H-L-I\big) - \dfrac{a_1\beta_1 HY}{1+(\alpha_1+a_3q_1Q)Y} - dH, \\ \dfrac{dL}{dt} = \dfrac{a_1\beta_1 HY}{1+(\alpha_1+a_3q_1Q)Y} - (\gamma+d)L, \\ \dfrac{dI}{dt} = \gamma L - (g+d)I, \\ \dfrac{dX}{dt} = \Lambda + a\tau Y - \dfrac{a_2\beta_2 IX}{1+\alpha_2 I+a_3q_2QX} + \dfrac{a_3\beta_3 QY}{1+\alpha_3 Y} - mX, \\ \dfrac{dY}{dt} = \dfrac{a_2\beta_2 IX}{1+\alpha_2 I+a_3q_2QX} - \dfrac{a_3\beta_3 QY}{1+\alpha_3 Y} - (m+a\tau)Y, \end{cases} \tag{2.1}$$

with the initial conditions

$$H(0) > 0, L(0) \geq 0, I(0) \geq 0, X(0) \geq 0, Y(0) \geq 0. \tag{2.2}$$

Note that an equation for the dynamics of Q is lacking in (2.1) because of the assumption that Q is constant, i.e., $\frac{dQ}{dt} = 0$. The model (2.1) is different from the one in [9] in that; (i) it considers the possible vector-trap effect of Q which is incorporated in the acquisition functional response, (ii) the virus-sink action of Q is assumed a saturation response due to the infective vectors, (iii) it is assumed that, only a proportion $a,\ 0 < a \leq 1$ of infected vectors will be fasting and can hold the virus for a maximum (average) time $1/\tau$ and, finally, (iv) the host plant recruitment rate function allows the basic reproduction number $\Re_0$ to depend on the parameter γ which is more realistic.

The parameters $a_i,\ 0 < a_i < 1,\ i = 1, 2, 3$ implicitly denote the effect of habitat complexity induced by Q on vector-host contact, and subsequently, the acquisition/inoculation rates. The biological/ecological descriptions of parameters and their values are taken from [9] and organized in the Table 1. The parameters $a, \alpha_3,\ q_1, q_2$ are assign assumed values and $d = 1/120 = 0.0083$ (FAO, 2009).

3 Model analysis

First, we can reduce the model system (2.1) into a four dimensional subsystem. Adding the last two equations and solving the resulting differential equation $\frac{dN_v}{dt} = \Lambda - mN_v$. Which gives $N_v(t) = \Lambda/m - (\Lambda/m - N_v(0))e^{-mt}$ and $N_v(t) \longrightarrow \Lambda/m$ as $t \longrightarrow \infty$, where $N_v = X + Y$. Therefore, we can replace X by $\Lambda/m - Y$ and study the dynamics of the resulting reduced subsystem:

$$\begin{cases} \dfrac{dH}{dt} = r\big(K - H - L - I\big) - \dfrac{a_1\beta_1 HY}{1 + b_1 Y} - dH, \\ \dfrac{dL}{dt} = \dfrac{a_1\beta_1 HY}{1 + b_1 Y} - (\gamma + d)L, \\ \dfrac{dI}{dt} = \gamma L - (g + d)I, \\ \dfrac{dY}{dt} = \dfrac{a_2\beta_2 I}{b_2 + \alpha_2 I - cY}\big(\dfrac{\Lambda}{m} - Y\big) - \dfrac{a_3\beta_3 QY}{1 + \alpha_3 Y} - zY, \end{cases} \tag{3.1}$$

where

$$b_1 = \alpha_1 + a_3 q_1 Q,\ b_2 = 1 + c\Lambda/m,\ \ c = a_3 q_2 Q,\ z = m + a\tau.$$

3.1 Boundedness and non-negativity of solutions

Since we are studing biological populations, it is important to show the non-negativity and boundedness of the system.

Theorem 1. *If the initial conditions of the system (3.1) are in the region*

$$\Omega = \Big\{(H, L, I, Y) \in \mathcal{R}_+^4 :\ 0 < H, L, I \leq K^*,\ 0 \leq Y \leq \frac{\Lambda}{m}\ ,\ K^* = \frac{rK}{r + d}\Big\},$$

Table 1. Biological/ecological descriptions of the model parameters and their illustrative values to be used for numerical simulations.

Param.	Biological description and unit	Value	Range
β_1	virus inoculation rate. $(vector \times\ day)^{-1}$	0.015	0.0014-0.0176
β_2	virus acquisition rate. $(plant \times\ day)^{-1}$	0.015	0.0014-0.0176
β_3	the rate of virus losses from Y on feeding (or probing) the Q plant. $(vector \times\ day)^{-1}$	0.05	0.01-0.1
a_1	efficiency of vectors to visit H. *constant*	0.2	0.2-0.5
a_2	efficiency of vectors to visit I. *constant*	0.2	0.2-0.5
a_3	proportion (or preference) of vectors visiting Q. *constant*	0.5	0.5-0.8
a	proportion of fasting aphids. *constant*	0.01	0.01-0.05
α_1	saturation effect due to Y, i.e. loss of infectivity on feeding H. $(vector \times\ day)^{-1}$	0.2	0.22-1
α_2	effect of saturation due to the time spent by X feeding on I. $(plant \times\ day)^{-1}$	0.1	0.01-1
α_3	effect of saturation due to scarcity of Q or rejection of vectors after landing. $(vector \times\ day)^{-1}$	0.1	0.01-0.2
q_1	time spent by Y searching for H due to the interference of (or feeding on) Q, and hence loss of infectivity. $(vector \times\ day)^{-1}$	0.002	0.0005-0.005
q_2	time spent by X searching for I due to the interference of (or feeding on) Q. $(vector \times\ day)^{-1}$	0.001	0.0005-0.005
Λ	recruitment rate of vectors. $vector \times (day)^{-1}$	5	0.1-10
r	recruitment rate of host plants. $(day)^{-1}$	0.01	
d	natural death rate of host crop plant. $(day)^{-1}$	0.0083	
g	roguing rate of infected plant. $(day)^{-1}$	0.05	0.0883-0.05
γ	progress rate of L to I. $(day)^{-1}$	0.071	0.0083-0.17
K	maximum carrying capacity of the field. *plant*	500	50-1000
τ	inverse of infectivity period of vectors. $(day)^{-1}$	8	6-12
m	emigration and/or natural death rate. $(day)^{-1}$	0.2	0.025-0.5
Q	total number of a non-virus host plants in the potatoes crop field. *constant*	200	100-300

then all solutions of the system equations will enter and remain in Ω. Also, any solution with initial conditions in Ω is non-negative.

Proof. Note that $N_p'(t) \le rK - (r+d)N_p(t)$, where $'$ denotes the time derivative $\frac{d}{dt}$. Solving the differential inequality for $N_p(t)$ and taking the limit $\lim_{t\longrightarrow\infty} N_p(t) \le K^*$ and also $Y \le \Lambda/m$ showing the boundedness of the system (3.1) in Ω. Also, any solution H, L, I, Y which initial values in Ω satisfies:

$$H'(t) \ge -H\big(d + \frac{\beta_1\Lambda}{m + b_1\Lambda}\big), \quad L'(t) \ge -(\gamma+d)L,\ I'(t) \ge -(g+d)I,$$
$$Y'(t) \ge -f(Y)Y \ge -f(\Lambda/m)Y,$$

where $f(Y) = (z + a_3\beta_3 Q + \alpha_3 zY)/(1+\alpha_3 Y)$ is a decreasing function of Y, i.e., $f(Y) \le f(\Lambda/m)$ which means $Y'(t) \ge -f(\Lambda/m)Y$. From the above differential inequalities, we can obtain $H(t)$, $L(t)$, $I(t)$ and $Y(t)$ are non-negative for all $t \ge 0$. We conclude that the model system (3.1) is epidemiologically meaningful and mathematically well-posed in Ω. □

3.2 The disease-free equilibrium (E_0) and basic reproduction number ($\Re_0$)

Clearly, system (3.1) always has a disease-free equilibrium given by $E_0 = (K^*, 0, 0, 0)$. We compute the basic reproduction number $\Re_0$ using the next generation matrix method [24]. The rate at which new infections are created is determined by the matrix $\mathcal{F}$, and the rates of transfer into and out of the class of infected states are represented by the matrix $\mathcal{V}$; these are given by

$$\mathcal{F} = \begin{pmatrix} \dfrac{a_1\beta_1 HY}{1+b_1Y} \\ 0 \\ 0 \end{pmatrix}, \ \mathcal{V} = \begin{pmatrix} (\gamma+d)L \\ -\gamma L+(g+d)I \\ \dfrac{a_3\beta_3 QY}{1+\alpha_3 Y} + zY - \dfrac{a_2\beta_2 I(\frac{\Lambda}{m}-Y)}{b_2+\alpha_2 I - cY} \end{pmatrix}.$$

From the next generation matrix $\mathcal{F}\mathcal{V}^{-1}$, we get the average value of secondary cases defined as the basic reproduction number $\Re_0$ which is given by:

$$\Re_0 = \frac{\gamma a_1 a_2 \beta_1 \beta_2 K^* \Lambda}{m(d+g)(d+\gamma)D_0 b_2}, \quad D_0 = z + a_3\beta_3 Q. \tag{3.2}$$

Remark 1. Note that without mixed-cropping system (i.e. $Q = 0$), the basic reproduction number is:

$$\Re_{01} := \frac{\gamma\beta_1\beta_2 K^*\Lambda}{m(d+g)(d+\gamma)z} > \frac{\gamma a_1 a_2\beta_1\beta_2 K^*\Lambda}{m(d+g)(d+\gamma)D_0 b_2} = \Re_0.$$

3.3 Local stability of E_0

In this subsection we study the local stability of the disease-free equilibrium point E_0.

Theorem 2. *The disease-free equilibrium, E_0 of (3.1) is locally asymptotically stable if $\Re_0 < 1$; it is unstable if $\Re_0 > 1$.*

Proof. Here, we apply the Routh-Hurwitz criteria to study the local stability of E_0. The Jacobian of (3.1) at E_0 is:

$$J(E_0) = \begin{pmatrix} -(r+d) & -r & -r & -a_1\beta_1 K^* \\ 0 & -(\gamma+d) & 0 & a_1\beta_1 K^* \\ 0 & \gamma & -(g+d) & 0 \\ 0 & 0 & \dfrac{a_2\beta_2\Lambda}{mb_2} & -D_0 \end{pmatrix},$$

where D_0 is given in (3.2). The characteristic equation is evaluated as:

$$(\lambda + r + d)\big(\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3\big) = 0,$$

where

$$p_1 = \gamma + 2d + g + D_0 > 0, \ p_2 = (\gamma + 2d + g)D_0 + (\gamma + d)(g + d) > 0,$$
$$p_3 = (\gamma + d)(g + d)D_0(1 - \Re_0).$$

It is clear that one of the eigenvalues, $\lambda = -(r+d)$ is negative. The remaining eigenvalues are given by solutions of the cubic equation $\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3 = 0$. If $\Re_0 < 1$, then $p_3 > 0$ and $p_3(p_1p_2 - p_3) > 0$. Hence, E_0 is locally asymptotically stable. Otherwise, if $\Re_0 > 1$, then $p_3 < 0$ and E_0 becomes unstable. □

3.4 Global stability of E_0

Here, we establish the global stability of E_0 via Lyapunov functions. For this, we define a Lyapunov function V as

$$V = m_1\Big(H - K^* - K^* \log\frac{H}{K^*} + L + \frac{(\gamma+d)I}{\gamma}\Big) + m_2 Y,$$

where m_1 and m_2 are positive constants to be determined latter. We get the following result:

Theorem 3. *Assume $\Re_0 < 1$ and $D_0b_1 \geq \alpha_3a_3\beta_3Q$. Then the disease-free equilibrium is globally asymptotically stable.*

Proof. The time derivative of V along the solutions of system (3.1) is

$$\begin{aligned} V' =& m_1\big[(\frac{H-K^*}{H})H' + L' + \frac{(\gamma+d)I'}{\gamma}\big] + m_2Y' \leq rm_1(\frac{H-K^*}{H})(K^*-H-L-I) \\ &+ \Big(m_1\frac{a_1\beta_1K^*}{1+b_1Y} - m_2\frac{D_0+\alpha_3zY}{1+\alpha_3Y}\Big)Y + \Big(m_2\frac{a_2\beta_2(\frac{\Lambda}{m}-Y)}{b_2+\alpha_2I-cY} - m_1q\Big)I, \\ \leq& \Big(m_1\frac{a_1\beta_1K^*}{1+b_1Y} - m_2\frac{D_0+\alpha_3zY}{1+\alpha_3Y}\Big)Y + \Big(m_2\frac{a_2\beta_2\Lambda}{mb_2} - m_1q\Big)I, \end{aligned}$$

where $q = (\gamma+d)(g+d)/\gamma$. Because $K^* \geq H+L+I$ and $f(Y) = \frac{a_2\beta_2(\frac{\Lambda}{m}-Y)}{b_2+\alpha_2I-cY}$ is decreasing with respect to Y. Let $m_2 = m_1\frac{a_1\beta_1K^*}{D_0}$, $m_1 = \frac{1}{q}$ and $D_0b_1 \geq \alpha_3a_3\beta_3Q$. Then $V' \leq \Big(\frac{\gamma a_1a_2\beta_1\beta_2K^*\Lambda}{mb_2D_0(g+d)(\gamma+d)} - 1\Big)I = (\Re_0 - 1)I$. Therefore, $V'(t) < 0$ when $\Re_0 < 1$ and $V'(t) = 0$ only at E_0. Hence, the conclusion follows by Lyapunov stability theorem [20]. □

3.5 Bifurcation analysis

Now we perform bifurcation analysis of the model (3.1) near $\Re_0 = 1$ and the disease-free equilibrium E_0. Investigating the direction of bifurcation is important to determine conditions for stability of E_0 and/or existence of endemic equilibrium E^*. We follow the technique of Castillo-Chavez and Song [8]. Let β_1 be the bifurcation parameter, $\boldsymbol{X} = (x_1, x_2, x_3, x_4) = (H, L, I, Y)$, $\boldsymbol{G} = (f_1, f_2, f_3, f_4) = \big(H', L', I', Y'\big)$. Then the system (3.1) can be re-written as:

$$\frac{d\boldsymbol{X}}{dt} = \boldsymbol{G}(\mathbf{X}, \beta_1^*); \quad \boldsymbol{G} : R^4 \times R^+ \longrightarrow R^4, \quad \boldsymbol{G} \in C^2(R^4 \times R^+), \tag{3.3}$$

where β_1^* is a value of β_1 at which $\Re_0 = 1$. Let E_0 be the equilibrium point of the system, that is $\boldsymbol{G}(E_0, \beta_1^*) = 0$ for all β_1^* is the value of β_1 such that $\Re_0 = 1$,

i.e., $\beta_1^* = \frac{mb_2D_0(\gamma+d)(g+d)}{\gamma a_1 a_2 \beta_2 K^* \Lambda}$. Let $A = J(E_0, \beta_1^*)$ be the Jacobian matrix of (3.3) evaluated at E_0 and β_1^*. We need to show the following conditions hold: [**A1**] A has a simple zero eigenvalue and all the other eigenvalues has negative real parts. [**A2**] A has a non-negative right eigenvector $\mathbf{w}$ and a left eigenvectors $\mathbf{v}$ each corresponding to the zero eigenvalue.

Theorem 4. *The system (3.1) exhibits forward bifurcation near $\Re_0 = 1$ and the disease free equilibrium E_0 provided that $D_0 b_1 \geq \alpha_3 a_3 \beta_3 Q$.*

Proof. The Jacobian at (E_0, β_1^*) is:

$$J(E_0, \beta_1^*) = \begin{pmatrix} -(r+d) & -r & -r & -a_1\beta_1^* K^* \\ 0 & -(\gamma+d) & 0 & a_1\beta_1^* K^* \\ 0 & \gamma & -(g+d) & 0 \\ 0 & 0 & \frac{a_2\beta_2\Lambda}{mb_2} & -D_0 \end{pmatrix}.$$

We found that two of the eigenvalues corresponding characteristic polynomial as $\lambda_1 = -(r+d), \lambda_2 = 0$ and the other two are roots of

$$\lambda^2 + (\gamma + 2d + g + D_0)\lambda + (\gamma+d)(g+d) + (\gamma + 2d + g)D_0 = 0.$$

It is clear that three eigenvalues have negative real parts and one eigenvalue is zero and simple. Let $\mathbf{w} = (w_1, w_2, w_3, w_4)$, $\mathbf{v} = (v_1, v_2, v_3, v_4)$ be the right and left eigenvector corresponding to $J(E_0, \beta_1^*)$, respectively. That is, the following matrix product hold:

$$[J(E_0, \beta_1^*)] \cdot [\mathbf{w}]^T = 0, \ [J(E_0, \beta_1^*)]^T \cdot [\mathbf{v}]^T = 0,$$

where A^T denotes the transpose of a matrix A. From the above matrix product we get:

$$w_1 = -\frac{a_1\beta_1^* K^*}{r+d}\left(1 + \frac{r(\gamma+d+g)}{(\gamma+d)(g+d)}\right)w_4, \ w_2 = \frac{a_1\beta_1^* K^* w_4}{\gamma+d}, \ w_3 = \frac{\gamma a_1 \beta_1^* K^* w_4}{(g+d)(\gamma+d)},$$

$$v_1 = 0, \ v_2 = \frac{\gamma a_2 \beta_2 \Lambda v_4}{mb_2(g+d)(\gamma+d)}, v_3 = \frac{a_2\beta_2\Lambda v_4}{mb_2(g+d)}, \ w_4 = w_4 > 0, \ v_4 = v_4 > 0$$

To complete the proof, we need to show that $a < 0$ and $b > 0$, where a and b be defined as follows:

$$a = \sum_{k,j,i=1}^{4} v_k w_j w_i \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, \beta_1^*) \ , \quad b = \sum_{k,j=1}^{4} v_k w_j \frac{\partial^2 f_k}{\partial x_j \partial \beta_1^*}(E_0, \beta_1^*). \tag{3.4}$$

Since $v_1 = 0$ we need only compute the second order partial derivatives of f_2, f_3 and f_4 with respect to x_i and $\beta_1^*, i = 1, ..., 4$ evaluated at (E_0, β_1^*) by equation (3.4) and taking $v_4 = w_4 = 1$, gives:

$$a = -\left[\frac{a_1^2\beta_1^{*2}K^*}{r+d}\left(1 + \frac{r(\gamma+d+g)}{(\gamma+d)(g+d)}\right) + \frac{\gamma a_1 a_2 \beta_1^* \beta_2 K^*}{(g+d)(\gamma+d)b_2^2}\left(1 + \frac{2\alpha_2\gamma a_1 \beta_1^* K^* \Lambda}{m(g+d)(\gamma+d)}\right)\right.$$
$$\left. + 2[D_0 b_1 - \alpha_3 a_3 \beta_3 Q]\right] < -2[D_0 b_1 - \alpha_3 a_3 \beta_3 Q], \ b = v_2 w_2 K^* = \frac{D_0}{\gamma+d} > 0.$$

Therefore, $a < 0$ when $D_0 b_1 \geq \alpha_3 a_3 \beta_3 Q$. We conclude that the system (3.1) exhibits forward bifurcation near $\Re_0 = 1$ since $a < 0$ and $b > 0$ [8]. The implication of the occurrence of forward bifurcation means that the endemic equilibrium can not exist if $\Re_0$ is less that one. □

3.6 Endemic equilibrium (E^*)

An endemic equilibrium E^* of the system (3.1) is a steady state solution when the disease persists. That is, $E^* = (H^*, L^*, I^*, Y^*)$ where $H^* \neq 0,\ L^* \neq 0,\ I^* \neq 0$ and $Y^* \neq 0$. It is obtained by setting the right-hand sides of (3.1) to zero. Therefore, H^*, L^* and Y^* are obtained as:

$$\begin{aligned} L^* =& \frac{(g+d)I^*}{\gamma},\ H^* = K^* - wI^*,\ Y^* = \frac{I^*}{c_0K^* - c_1I^*}, \quad \text{with} \\ w =& \frac{\gamma(r+d+g)+(r+d)(g+d)}{\gamma(r+d)},\ c_0 = \frac{\gamma a_1\beta_1}{(\gamma+d)(g+d)},\ c_1 = c_0w + b_1. \end{aligned}$$

And I, ignoring the star sign, is a feasible root of the cubic polynomial:

$$P(I) = A_3I^3 + 3A_2I^2 + 3A_1I + A_0 = 0, \tag{3.5}$$

where

$$\begin{aligned} A_0 =& b_2(c_0K^*)^2D_0(1-\Re_0),\ A_1 = \frac{c_0K^*}{3}\big\{[\alpha_3zb_2 + c_0K^*a_2\beta_2] \\ &+ D_0\big[\alpha_2c_0K^* - c + b_2(c_1-\alpha_3)\Re_0 + 2b_2c_1(\Re_0-1)\big]\big\} \\ A_2 =& \frac{-1}{3}\big\{c_0K^*\big[\alpha_2(D_0c_1-\alpha_3z) + a_2\beta_2(2c_1-\alpha_3)\big] + \alpha_3z(b_2c_1+c) \\ &+ D_0c_1\big[\alpha_2c_0K^* - c + b_2c_1(\Re_0-1) + 2b_2(c_1-\alpha_3)\Re_0\big]\big\} \\ A_3 =& c_1\big[(c_1-\alpha_3)\big(a_2\beta_2\big(1 + c_1\frac{\Lambda}{m}\big) + \alpha_2z\big) + c_1\alpha_2a_3\beta_3Q\big]. \end{aligned}$$

For $\Re_0 > 1,\ c_1 \geq \alpha_3$ and $\Lambda(c_1-\alpha_3)\Re_0 \geq m$ then $A_0 < 0,\ A_1 > 0,\ A_2 < 0$ and $A_3 > 0$. Thus, by Descartes rule of sign changes, the Equation (3.5) has one, two or three real positive roots. On the other hand, let ψ be a primitive cube root of unity. Using Cardano's method of solving cubic equations, a general solution to (3.5) can be given by:

$$\begin{aligned} I =& \frac{-A_2}{A_3} + \psi^k\left(\frac{-Q + \sqrt{Q^2+4P^3}}{2}\right)^{1/3} + \psi^k\left(\frac{-Q - \sqrt{Q^2+4P^3}}{2}\right)^{1/3}, \\ P =& \frac{A_1A_3 - A_2^2}{A_3^2},\ Q = \frac{2A_2^3 - 3A_1A_2A_3 + A_0A_3^2}{A_3^3},\ k = 0,1,2. \end{aligned}$$

3.7 Local stability of E^* and non-existence of Hopf bifurcation

In this subsection, we study the local stability of the endemic equilibrium by using the Routh-Hurwitz criteria [20]. Also, we will establish the non-existence of periodic solutions near E^* of the system (3.1). We obtain the following result:

Theorem 5. *Let $D_0b_1 \geq \alpha_3a_3\beta_3Q$ an endemic equilibrium E^* exists. Then E^* is locally asymptotically stable. Furthermore, the system (3.1) does not exhibit a Hopf bifurcation near E^*.*

Proof. The Jacobian at E^* is given by:

$$J(E^*) = \begin{pmatrix} -A_{11} & -r & -r & -A_{14} \\ A_{21} & -A_{22} & 0 & A_{14} \\ 0 & \gamma & -A_{33} & 0 \\ 0 & 0 & A_{43} & -A_{44} \end{pmatrix},$$

where

$$A_{11} = r + d + A_{21},\ A_{21} = \frac{a_1\beta_1 Y^*}{1 + b_1 Y^*},\ A_{14} = \frac{a_1\beta_1 H^*}{(1 + b_1 Y^*)^2},$$
$$A_{22} = \gamma + d,\ A_{33} = g + d,\ A_{43} = \frac{a_2\beta_2(\frac{\Lambda}{m} - Y^*)(b_2 - cY^*)}{(b_2 + \alpha_2 I^* - cY^*)^2},$$
$$A_{44} = z + \frac{a_3\beta_3 Q}{(1 + \alpha_3 Y^*)^2} + \frac{a_2\beta_2 I^*(1 + \alpha_2 I^*)}{(b_2 + \alpha_2 I^* - cY^*)^2}.$$

The corresponding characteristic equation is

$$P(\lambda) = \lambda^4 + l_1\lambda^3 + l_2\lambda^2 + l_3\lambda + l_4 = 0,$$

with

$$l_1 = A_{11} + A_{22} + A_{33} + A_{44} > 0,$$
$$l_2 = A_{11}A_{22} + A_{33}A_{44} + (A_{11} + A_{22})(A_{33} + A_{44}) + rA_{21} > 0,$$
$$l_3 = A_{11}A_{22}(A_{33}+A_{44}) + (A_{11}+A_{22})A_{33}A_{44}+rA_{21}(\gamma+A_{33}+A_{44})-\gamma A_{14}A_{43},$$
$$l_4 = (A_{11}A_{22} + rA_{21})A_{33}A_{44} + \gamma rA_{21}A_{44} - (r + d)\gamma A_{14}A_{43}.$$

The corresponding Hurwitz determinants are given by: $M_1 = l_1 > 0$,

$$\begin{aligned} M_2 =& l_1l_2 - l_3 = A_{11}A_{22}(A_{11} + A_{22}) + A_{33}A_{44}(A_{33} + A_{44}) \\ &+ rA_{21}(A_{11} + d) + \gamma A_{14}A_{43} + l_1(A_{11} + A_{22})(A_{33} + A_{44}) > 0, \\ M_3 =& l_3(l_1l_2 - l_3) - l_1^2l_4 = l_1(A_{11} + A_{22})\big[A_{11}A_{22}A_{33}^2 \\ &+ rA_{21}[A_{33}^2 + A_{33}A_{44} + A_{44}^2]\big] + l_3\big[A_{11}A_{22}(A_{11} + A_{22}) \\ &+ A_{33}A_{44}(A_{33} + A_{44}) + rA_{11}A_{21} + \gamma A_{14}A_{43}\big],\ M_4 = l_4M_3. \end{aligned}$$

Using the equilibrium equation, we obtain:

$$\gamma A_{14}A_{34} = \frac{A_{22}A_{33}(D_0 + \alpha_3 zY)(b_2 - cY)}{(1 + b_1Y)(1 + \alpha_3 Y)(b_2 + \alpha_2 I - cY)},\ A_{44} > z + \frac{a_3\beta_3 Q}{(1 + \alpha_3 Y^*)^2},$$
$$\implies A_{22}A_{33}A_{44} - \gamma A_{14}A_{43} > \frac{A_{22}A_{33}(D_0b_1 - \alpha_3 a_3\beta_3 Q)Y}{(1 + b_1Y)(1 + \alpha_3 Y)^2(b_2 + \alpha_2 I - cY)}.$$

Therefore, if $D_0b_1 \geq \alpha_3 a_3\beta_3 Q$ then $l_3 > 0,\ l_4 > 0, M_3 > 0$ and $M_4 > 0$. By the Routh-Hurwitz criteria, we conclude that the endemic equilibrium E^* is locally asymptotically stable. Furthermore, since $M_3 > 0$, there is no Hopf bifurcation near E^* [20]. □

3.8 Global stability of E^*

In order to discuss the global stability of the endemic equilibrium E^*, we follow the procedure used in Theorem 3 with a Lyapunov function V defined as:

$$V = \big(H - H^* - H^* \log \frac{H}{H^*}\big) + \big(L - L^* - L^* \log \frac{L}{L^*}\big) + \big(I - I^* - I^* \log \frac{I}{I^*}\big) + \big(Y - Y^* - Y^* \log \frac{Y}{Y^*}\big).$$

The time derivative of V in the direction of system Equation (3.1) is:

$$\begin{aligned}
V' =& \frac{dV}{dt} = \big(\frac{H - H^*}{H}\big)H' + \big(\frac{L - L^*}{L}\big)L' + \big(\frac{I - I^*}{I}\big)I' + \big(\frac{Y - Y^*}{Y}\big)Y' \\
=& r\big(K - N_p\big) + \frac{a_1\beta_1 H^* Y}{1 + b_1 Y} + dH^* + (\gamma + d)L^* + (g + d)I^* + \frac{a_2\beta_2 I(\frac{\Lambda}{m} - Y)}{b_2 + \alpha_2 I - cY} \\
&+ \big(\frac{a_3\beta_3 Q}{1 + \alpha_3 Y} + z\big)Y^* - \frac{rH^*(K - N_p)}{H} - dH - \frac{a_1\beta_1 HYL^*}{(1 + b_1 Y)L} - dL - (g + d)I \\
&- \gamma\frac{LI^*}{I} - \frac{a_2\beta_2 I(\frac{\Lambda}{m} - Y)Y^*}{(b_2 + \alpha_2 I - cY)Y} - \big(\frac{a_3\beta_3 Q}{1 + \alpha_3 Y} + z\big)Y = M - N, \\
M =& r(K - N_p) + \frac{a_1\beta_1 H^* Y}{1 + b_1 Y} + dH^* + (\gamma + d)L^* + (g + d)I^* \\
&+ \frac{a_2\beta_2 I(\Lambda/m - Y)}{b_2 + \alpha_2 I - cY} + \Big(\frac{a_3\beta_3 Q}{1 + \alpha_3 Y} + z\Big)Y^*, \\
N =& \frac{H^*}{H} r(K - N_p) + dH + \frac{a_1\beta_1 HYL^*}{(1 + b_1 Y)L} + dL + (g + d)I + \gamma\frac{LI^*}{I} \\
&+ \frac{a_2\beta_2 I(\frac{\Lambda}{m} - Y)Y^*}{(b_2 + \alpha_2 I - cY)Y} + \Big(\frac{a_3\beta_3 Q}{1 + \alpha_3 Y} + z\Big)Y.
\end{aligned}$$

Clearly, $V' < 0$ provided that $M < N$ and $V' = 0$ only at E^*. Thus, by Lyapunov stability theorem, the endemic equilibrium E^* is globally asymptotically stable if $M < N$ [20].

3.9 Sensitivity of the basic reproduction number $\Re_0$

It is important to determine the influence of parameters on the disease dynamics and to evaluate the efficiency of a control strategy used. For these, we evaluate the sensitivity of $\Re_0$ with respect to the involved parameters. According to [20], the sensitivity of $\Re_0$ to the parameter p is defined as

$$\Upsilon_p^{\Re_0} := \frac{\partial \Re_0}{\partial p} \times \frac{p}{\Re_0}.$$

Using this formula and parameters values in Table 1, the sensitivity indices of parameters are evaluated and organized in the Table 2. The sensitivity index of a parameter is interpreted as follows: A parameter with positive (negative) sensitivity index will increase (decrease) the disease spread when it increases (increases), respectively. From the table, we see that parameters related to the mixed-cropping strategy Q, a_3, β_3 and q_2 are are the most influential in reducing the rate of disease spread.

Table 2. Sensitivity indices of parameters.

Parameters	Sensitivity indices	Parameter	Sensitivity indices
$\beta_1,\ \beta_2,\ a_1,\ a_2,\ K^*$	+1.000	q_2	-0.909
Λ	+0.091	g	-0.858
γ	+0.007	d	-0.247
$Q,\ a_3$	-1.884	m	-0.092
β_3	-0.975	$a,\ \tau$	-0.016

4 Extension into optimal control model

In this section, we extend the model system (2.1) to an optimal control problem by incorporating the use three additional controls; mineral oil u_1, insecticide u_2, and farmer's inspection of the crop field u_3. The objectives of this section are to investigate the advantage of combining mixed-cropping with chemical controls and also to compare the efficiency of mixed-cropping with that of insecticide and/or mineral oil in reducing the disease spread. An other objective is to illustrate the advantages of mixed-cropping strategy in reducing the need for chemical controls, u_1 and u_2.

Spraying mineral oil reduces the probability of acquisition and inoculation by preventing vectors from probing on the host crop. Treating the host plant with mineral oil also increases the death or emigration rate of vectors. Insecticide reduces vector population by increasing the death rate. Farmer's inspection of the crop field aims to increase the identification and timely removal of infected (virus source) plants, that is, to decrease the number of infected plants. Incorporating these controls, the model system (2.1) becomes:

$$\begin{cases} \dfrac{dH}{dt} = r\big(K - H - L - I\big) - \dfrac{(1-u_1)a_1\beta_1 HY}{1+(\alpha_1 + a_3q_1Q)Y} - dH, \\ \dfrac{dL}{dt} = \dfrac{(1-u_1)a_1\beta_1 HY}{1+(\alpha_1 + a_3q_1Q)Y} - (\gamma + d)L, \\ \dfrac{dI}{dt} = \gamma L - [g(1+u_3) + d]I, \\ \dfrac{dX}{dt} = \Lambda + a\tau Y - \dfrac{(1-u_1)a_2\beta_2 IX}{1+\alpha_2 I + a_3q_2QX} + \dfrac{a_3\beta_3 QY}{1+\alpha_3 Y} - (eu_1 + u_2 + m)X, \\ \dfrac{dY}{dt} = \dfrac{(1-u_1)a_2\beta_2 IX}{1+\alpha_2 I + a_3q_2QX} - \dfrac{a_3\beta_3 QY}{1+\alpha_3 Y} - (eu_1 + u_2 + a\tau + m)Y, \end{cases} \tag{4.1}$$

subject to the initial conditions (2.2). The factor $e,\ 0 < e < 1$ in (4.1) denotes the death/emigration of vectors caused by mineral oil.

We will apply optimal control theory [1] to determine the optimal level of efforts that would be needed to minimize the disease prevalence and the costs of applying controls. For this, we need to define an objective functional $\mathcal{J}$ which is to minimize the number of latent L and infected I host plants, susceptible X and infected Y vectors, and the costs associated with the controls u_1, u_2, u_3 over a finite interval of time $[0, T]$. By choosing quadratic cost functions of the

controls, we define the objective functional as

$$\mathcal{J} = \min_{u_1,u_2,u_3} \int_0^T \big[m_1(L+I) + m_2(X+Y) + \frac{1}{2}\sum_{i=1}^{3} n_i u_i^2\big]dt, \tag{4.2}$$

subject to the constraint (4.1) with initial conditions (2.2). The set of admissible controls is a subset of bounded Lebesgue measurable functions $\mathcal{U} \subset (\mathcal{L}^\infty[0,T])^3$, $(u_1,u_2,u_3) \in \mathcal{U}$ where

$$0 \le u_i(t) \le \phi \le 1,\ 0 \le t \le T,\ \phi = \max\{u_{imax}\}, i = 1,2,3.$$

The quantities m_1, m_2 denote the weight constants of the exposed and infected plants, and vector population, respectively. The constants n_1, n_2, n_3 denote the weight for u_1, u_2, u_3, respectively. The terms $\frac{1}{2}n_1u_1^2$, $\frac{1}{2}n_2u_2^2$ and $\frac{1}{2}n_3u_3^2$ are the costs of applying the controls. The weights of state variables are assigned based on their relative importance while those of controls are assigned in relation to their cost implications. We thus seek an optimal control $(u_1^*,\ u_2^*, u_3^*)$ such that:

$$\mathcal{J}(u_1^*, u_2^*, u_3^*) = min\{\mathcal{J}(u_1,u_2,u_3) : (u_1,u_2,u_3) \in \mathcal{U}\}.$$

For the optimal control problem (4.1)–(4.2), we know that the state system is bounded and satisfies Lipschitz property with respect to the state variables. Also, the integrand in $\mathcal{J}$ is convex with respect to both the control and state variables. Therefore, the existence of an optimal control pair is a consequence of the standard results of optimal control theory [1,4,17,18].

4.1 The Hamiltonian and optimality system

Applying Pontryagin's Maximum Principle (PMP), the necessary conditions that an optimal pair $(u^*,x^*), u^*{=}(u_1^*,u_2^*,u_3^*)$, $x^*{=}(H^{u^*}, L^{u^*}, I^{u^*}, X^{u^*}, Y^{u^*})$ must satisfy can be obtained by converting the problem of minimization of the objective functional coupled with the state variables into a problem of minimizing point-wise a Hamiltonian, $\mathcal{H}$, with respect to the controls (u_1,u_2,u_3) [1,18]. We define the Hamiltonian as:

$$\mathcal{H} = m_1(L{+}I){+}m_2(X{+}Y){+}\frac{1}{2}\sum_{i=1}^{3} n_i u_i^2{+}\lambda_1\frac{dH}{dt}{+}\lambda_2\frac{dL}{dt}{+}\lambda_3\frac{dI}{dt}{+}\lambda_4\frac{dX}{dt}{+}\lambda_5\frac{dY}{dt},$$

where $\lambda_i,\ i = 1,\ldots,5$ are the adjoint or co-state variables. By the PMP we have the following result:

Theorem 6 [Proposition]. *If the optimal pair (u_1^*,u_2^*,u_3^*) minimizes $\mathcal{J}(u_1,u_2,u_3)$ over $\mathcal{U}$, then there exist adjoint functions $\lambda_i,\ i = 1,\ldots,5$ which satisfies:*

i. the adjoint system

$$\frac{d\lambda_1}{dt} = \frac{-\partial\mathcal{H}}{\partial H} = \lambda_1(r+d) + (\lambda_1-\lambda_2)\frac{(1-u_1)a_1\beta_1 Y}{1+(\alpha_1+a_3q_1Q)Y},$$
$$\frac{d\lambda_2}{dt} = \frac{-\partial\mathcal{H}}{\partial L} = -m_1 + \lambda_1 r + \lambda_2(\gamma+d) - \lambda_3\gamma,$$

$$\begin{aligned}
\frac{d\lambda_3}{dt} = \frac{-\partial \mathcal{H}}{\partial I} =& -m_1 + \lambda_1 r + \lambda_3(g + d + gu_3) \\
&+ \frac{(\lambda_4 - \lambda_5)(1-u_1)a_2\beta_2 X(1 + a_3q_2QX)}{(1 + \alpha_2 I + a_3q_2QX)^2}, \\
\frac{d\lambda_4}{dt} = \frac{-\partial \mathcal{H}}{\partial X} =& -m_2 + (\lambda_4 - \lambda_5)\frac{(1-u_1)a_2\beta_2 I(1+\alpha_2 I)}{(1 + \alpha_2 I + a_3q_2QX)^2} \\
&+ \lambda_4(eu_1 + u_2 + m), \\
\frac{d\lambda_5}{dt} = \frac{-\partial \mathcal{H}}{\partial Y} =& -m_2 + (\lambda_1 - \lambda_2)\frac{(1-u_1)a_1\beta_1 H}{(1 + (\alpha_1 + a_3q_1Q)Y)^2} \\
&+ \frac{(\lambda_5 - \lambda_4)a_3\beta_3 Q}{(1+\alpha_3 Y)^2} - \lambda_4 a\tau + \lambda_5(eu_1 + u_2 + z),
\end{aligned}$$

ii. the control system

$$\frac{dH}{dt} = \frac{d\mathcal{H}}{d\lambda_1},\ \frac{dL}{dt} = \frac{d\mathcal{H}}{d\lambda_2},\ \frac{dI}{dt} = \frac{d\mathcal{H}}{d\lambda_3},\ \frac{dX}{dt} = \frac{d\mathcal{H}}{d\lambda_4},\ \frac{dY}{dt} = \frac{d\mathcal{H}}{d\lambda_5},$$

iii. the transversality conditions

$$\lambda_i(T) = 0,\ i = 1, \ldots, 5, \quad \textit{and}$$

iv. the optimality condition,

$$\begin{aligned}
&\mathcal{H}\big(H^{u^*}(t), L^{u^*}(t), I^{u^*}(t), X^{u^*}(t), Y^{u^*}(t), u_1^*(t), u_2^*(t), u_3^*(t), \lambda_i^*(t)\big) \\
&= \min_{\mathcal{U}} \mathcal{H}\big(H^*(t), L^*(t), I^*(t), X^*(t), Y^*(t), u_1(t), u_2(t), u_3(t), \lambda_i^*(t)\big), \\
&i = 1, \ldots, 5,\ \textit{for } t \textit{ almost everywhere in } [0, T].
\end{aligned}$$

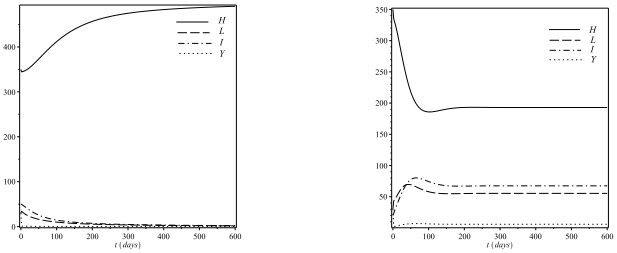
The optimality condition $\frac{\partial \mathcal{H}}{\partial u_i} = 0,\ i = 1, 2, 3$ gives the optimal control pair (u_1^*, u_2^*, u_3^*), where

$$\begin{aligned}
u_1^* =& \min\Big\{1, \max\Big\{0\ ,\ \frac{1}{n_1}\Big(\frac{(\lambda_2 - \lambda_1)a_1\beta_1 HY}{1 + (\alpha_1 + a_3q_1Q)Y} + \frac{(\lambda_5 - \lambda_4)a_2\beta_2 IX}{1 + \alpha_2 I + a_3q_2QX} \\
&+ e(\lambda_4 X + \lambda_5 Y)\Big)\Big\}\Big\}, \\
u_2^* =& \min\Big\{1, \max\Big\{0\ ,\ \frac{\lambda_4 X + \lambda_5 Y}{n_2}\Big\}\Big\},\ u_3^* = \min\Big\{1, \max\Big\{0\ ,\ \frac{\lambda_3 gI}{n_3}\Big\}\Big\}.
\end{aligned}$$

5 Numerical simulations

In this section, we present numerical simulations to support the analytic results of the study. We use the set of parameters values given in the Table 1. Unless and otherwise mentioned, the following parameters are used in the simulations: $r = 0.01,\ K = 915,\ Q = 200,\ d = 0.0083,\ \gamma = 0.071,\ a = g = m = 0.05,\ \beta_1 = \beta_2 = 0.015,\ \beta_3 = 0.05,\ \tau = 8,\ \Lambda = 10,\ \alpha_1 = 0.2,\ \alpha_2 = 0.01, q_1 = 0.002, q_2 = 0.001$ and $\alpha_3 = 0.1$, unless and otherwise given specifically.

If we let $Q = 200,\ \Lambda = 5,\ \beta_1 = \beta_2 = 0.0175,\ \beta_3 = 0.08,\ a_1 = a_2 = 0.5,\ a_3 = 0.4$, then $\Re_0 = 0.954 < 1$. The simulation result of the model is

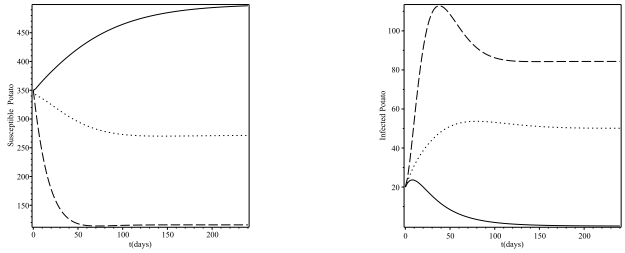


(a) (b)

Figure 2. Simulation of system (3.1) showing the time evolution of healthy $H(t)$, latent $L(t)$, infected $I(t)$ hosts and infected vectors $Y(t)$ when (a) $\Re_0 < 1$ and (b) $\Re_0 > 1$. The plots show that the disease will die out if $\Re_0 < 1$ (a) and can persist if $\Re_0 > 1$ (b).

given by Figure 2(a) which shows the latent host (L), infected host (I) and infective vector (Y) populations tend to zero as time increases, whereas, the healthy host plant population (H) tend towards K^*. This implies the global asymptotic stability of the disease-free equilibrium and confirms the analytic finding.

On the other hand, when $Q = 100, \Lambda = 5, \beta_1 = \beta_2 = 0.015,\ \beta_3 = 0.05,\ a_1 = a_2 = 0.5,\ a_3 = 0.3$, we get $\Re_0 = 4.595$ and the dynamics is illustrated by Figure 2(b) which shows the persistence of disease in the host population, i.e., the stability of endemic equilibrium.



(a) (b)

Figure 3. Plots showing effects of density of the barrier plant Q and its associated parameters on the dynamics of healthy $H(t)$ and infected $I(t)$ plant populations. The **solid** curves are when $Q = 200,\ a_1 = 0.3,\ a_2 = 0.3, a_3 = 0.5, \beta_3 = 0.05$, the dotted are when $Q = 100, a_1 = a_2 = 0.5, a_3 = 0.4, \beta_3 = 0.03$, and the dashed are when $Q = 0, a_1 = a_2 = 1$.

The impact of parameters related to the mixed-cropping system a_1, a_2, a_3, Q is illustrated on the evolution of healthy and infected plants Figure 3(a) and 3(b).

6 Discussion and conclusions

In this paper, we have investigated the dynamical properties of a model for PVY spread in a mixed-cropping system comprised of two crops; the host (potato) and a non-host crop. Then, we extend the model to an optimal control problem

by considering the use of mineral oil u_1, insecticide u_2 and farmer's level of field inspection u_3 and investigated their impact on the disease spread.

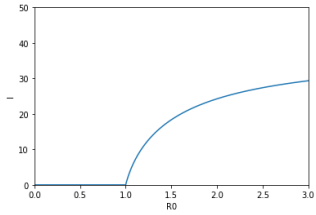
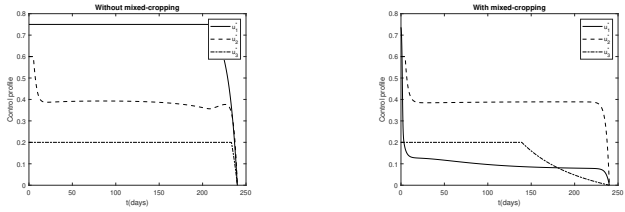


Figure 4. Forward bifurcation diagram of the endemic equilibrium near $\Re_0 = 1$ and the disease-free point E_0. It means, the endemic equilibrium exists if and only if $\Re_0 > 1$. We have used the Equation (3.5) to plot the graph by writing the coefficients A_i in terms of $\Re_0$ as much as possible. The bifurcation parameter can be any of the parameters involved in $\Re_0$. The implication of the occurrence of forward bifurcation means that the disease can be eradicated if $\Re_0$ is less that one.

The analytic results of the model show that the basic reproduction number $\Re_0$ is a threshold parameter that decides properties of the disease dynamics. The model exhibits forward bifurcation near $\Re_0 = 1$ and the disease-free equilibrium E_0 (see Figure 4). This means, the endemic equilibrium E^* can exist only when $\Re_0 > 1$. It is shown that E_0 is globally asymptotically stable if $\Re_0 < 1$ and $b_1 D_0 \geq \alpha_3 a_3 \beta_3 Q$ (Theorem 3) and demonstrated by numerical simulation, Figure 2(a). The epidemiological implication is that the disease will eventually be eliminated when $\Re_0 < 1$. However, the disease can persist in the host plant population if $\Re_0 > 1$ as shown in Figure 2(b).

From the derivation of the basic reproduction number $\Re_0$ and sensitivity analysis results, we see that $\Re_0$ is significantly influenced in a mixed-cropping system. The density (number) of the mixed-cropped plants (Q), the quality of Q in attracting vectors a_3, the probability of virus removal β_3 and its vector-trap factor q_2 are crucial in reducing the value of $\Re_0$, and hence, to control the disease. We present the graphs in Figures 3(a) and 3(b) to demonstrate how significant the values of these parameters affect the disease dynamics.



(a) (b)

Figure 5. Plots of the optimal controls u_1^*, u_2^* and u_3^* as functions of time without (a) and with (b) mixed-cropping strategy. The graphs are results of the parameters values $m_1 = 100,\ m_2 = 80,\ n_1 = n_2 = 10,000,\ n_3 = 1000$.

The optimal control profiles of the model without and with mixed-cropping

system is shown in Figures 5(a) and 5(b), respectively. These graphs depict the time and amount of mineral oil u_1, insecticide u_2 needed and the need of field observation u_3. The graphs show that mixed-cropping strategy can reduce the need for chemical controls u_1 and u_2.

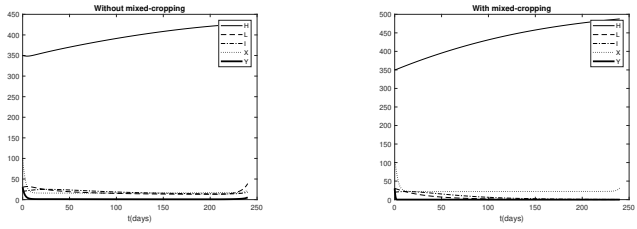


Figure 6. Simulations of the model (4.1) (a) without mixed-cropping, i.e. $Q = 0, a_1 = a_2 = 1$, and (b) with mixed-cropping system for $Q = 200$. The graphs show the disease can persist in the host population in case (a) but dies out in case (b).

The impact of these controls on the dynamical behaviors of the host and vector populations without and with mixed-cropping is demonstrated in Figures 6(a) and 6(b), respectively. Use of mineral oil, insecticide and roguing without mixed-cropping may not sufficient to eradicate the disease as shown in Figure 6(a), however, the disease can be successfully controlled when these measures are integrated with mixed-cropping, see Figure 6(b).

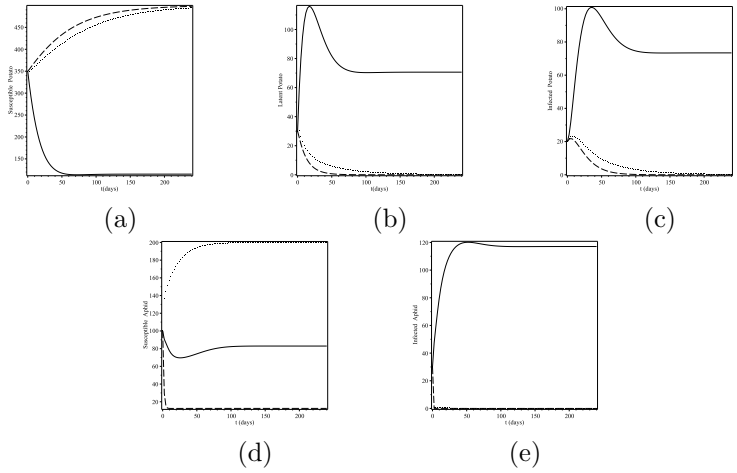


Figure 7. Simulations showing the time evolution of the host and vector populations under different situations; when mixed-cropping, mineral oil u_1 and insecticide u_2 are used (dash), mixed-cropping only, i.e. $u_1 = u_2 = 0$ (dot), and when no measures except u_3 are taken, i.e. $Q = u_1 = u_2 = 0,\ a_1 = a_2 = 1$ (solid). The parameters values used are $\beta_3 = 0.02, \Lambda = 10, a_1 = a_2 = 0.3, a_3 = 0.6, u_3 \neq 0$.

We can conclude that (i) mixed-cropping strategy alone can produce equivalent result in controlling the disease as that of using mineral oil and insecticide (see Figures 7(a)–7(e)), and (ii) the integration of mixed-cropping with either

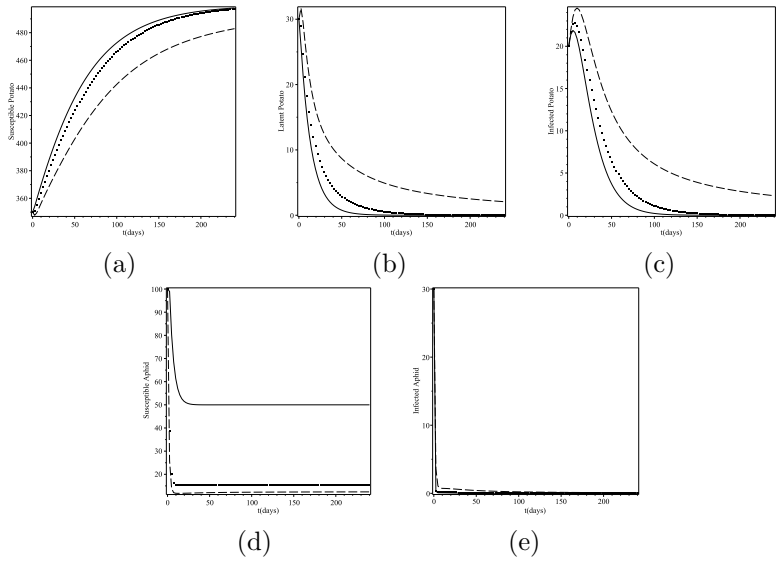


Figure 8. The host and vector population dynamics under different situations where $u_3 \neq 0$. When mixed-cropping combined with mineral oil is used (solid), mixed-cropping with insecticide (dotted), and mineral oil combined with insecticide but without mixed-cropping, i.e., $Q = 0$ (dashed).

mineral oil or insecticide is the best to manage the disease as shown in Figures 8(a)–8(e). We believe that our study can contribute further knowledge in the search for affordable and non-chemical control methods of non-persistently transmitted vector-borne plant viral diseases.

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