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# Eco-epidemiological Model and Optimal Control Analysis of Tomato Yellow Leaf Curl Virus Disease in Tomato Plant

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**Summary**

The purpose of this study is to analyze the impact of control strategies namely insecticide spray, roguing of a diseased tomato plant, protective netting to protect tomato plant from TYLCVD. Thus, a mathematical model for the transmission dynamics of TYLCVD that includes these control strategies is formulated and analyzed. In the case of constant control, the basic reproduction number is calculated and the existence and stability of equilibria are investigated. Besides, an optimal control model with constraints is formulated and investigated. In the non-constant control case, Pontryagin's Maximum Principle is used to deduce necessary conditions for the optimal control of the disease. It is shown that all the combined efforts of two of three strategies can significantly reduce the disease except the combination of the use of insecticide spray and rouging infected tomato plants. Relatively the other, the use of roguing diseased tomato plants and protective netting, and the use of insecticides spray, roguing diseased plants and protective netting are better decreased the disease. Moreover, the use of roguing diseased plants and protective netting has a similar effect as the use of insecticides spray, roguing diseased tomato plants, and protective netting. As resources are scarce, we recommend that policy-makers should adopt the combination of the use of roguing diseased tomato plants and protective netting as a strategy.

**KEYWORDS:**
TYLCVD, Pontryagins maximum principle, Control strategies, Optimal control of TYLCV, stability, numerical simulation.

## 1 | INTRODUCTION

Tomato (*Solanum Lycopersicum L.*) is one of the most popular and widely grown vegetables in the world. However, it is highly destructed by Tomato Yellow Leaf Curl Virus (TYLCV) disease[1]. This disease is mainly transmitted by an insect vector called whitefly *Bemisia tabaci* (*B. tabaci*) of biotype B (Gennadius) (Hemiptera: Aleyrodidae) in a circulative and persistent manner[2]. The vector is damaged tomato plant direct by feeding on phloem, excreting honeydew, and causing phytotoxic disorders[3]. With increased populations, they secrete large quantities of honeydew, which favor the growth of sooty mold on leaf surfaces and reduce the photosynthetic efficiency of the plants[4]. The honeydew also contaminates the marketable part of the plant, reducing its market value. Additionally, in severe infestations, the leaves turn yellow and drop off[2].

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[0]**Abbreviations:** TYLCVD, tomato yellow leaf curl virus disease

The first reports on TYLCV infection in tomato were from Israel and other countries in the The Middle East in the 1930s and since then the virus has further emerged[4]. In Africa, TYLC disease was first described in Sudan in 1965, but the causal agent was identified as TYLCV in 1997[5,6]. In Ethiopia, at Melka-Werner about 90% of tomato plant showed leaf curl virus symptoms with reduced size suspected to be caused by TYLCV[7], but only recently occurrence of Begomovirus associated with TYLC disease was reported for the first time from Melkasa[8].

The whitefly vector feeds on an infected host plant and acquires the virus, viral transmission can occur within hours and may continue for the life span of the vector. Acquisition and transmission thresholds were found to be between 15 and 30 min and a single *B. tabci* whitefly can accumulate 600 million TYLCV genomes[2]. From the site of inoculation, viral DNA is first mainly transported to strong sink organs, such as root and shoot apices, flowers, and fruit, and then to leaves[9]. Similar to other plant viruses, TYLCV moves in the existing host transport routes such as plasmodesmata and phloem, along with carbohydrates[29]. About 11 - 13 days after inoculation, maximum amounts of viral DNA and capsid or coat protein accumulate in the youngest tissues of shoots and roots, and 4 - 7 days later, symptoms appear for the first time[9]. As the systemic infection proceeds in the growing plant, the virus accumulates in the strongest sink tissues. The level of viral double-stranded (dsDNA) and newly generated single-stranded DNA (ssDNA), as well as CP, further increases in young organs up to several weeks post-inoculation. The infection then gradually spreads to older organs of the host, and remains strictly confined to the vascular system[29].

Mathematical modeling has been playing an important role in better understanding of epidemiological patterns by providing deeper insight into the underlying mechanisms for the spread of emerging and reemerging infectious diseases and suggesting effective control strategies[10]. Holt *et al.*[11] on their paper entitled "An epidemiological model incorporating vector population dynamics applied to African cassava mosaic virus disease" illustrated an Africa Cassava Mosaic Virus occurrence in cassava which transmitted by a cassava-specific whitefly strain, which was then sweeping through Uganda. The virus also propagates routinely from stem cuttings. The use of uninfected cutting tools and roguing of infected plants are among the control alternatives. Utilization of uninfected cutting tools would be more effective if infected cuttings were driving disease spread, whereas roguing would be more important in a largely vector-driven epidemic.

In a later paper, Holt *et al.*[12] developed epidemiological model, Susceptible - Exposed - Infective (SEI)-type epidemic for the host plant and Susceptible - Infective (SI)-type for insect vector population, that represents the incidence of TYLCV in tomato mainly relied on the immigration of vectors from alternative hosts which act as a reservoir of both the virus and vector. This is because, unlike cassava, the tomato was only an occasional host for this whitefly, and spillover from other perennials and weedy plants drove vector and virus dynamics. They considered different strategies to reduce the spread of TYLCV and studied the sensitivity analysis of their results to the parameters to explore different disease management options. In this context, the authors asked "what is the best method for disease control?" Because most of the vector lifespan occurs on other hosts, the authors adapted a model framework[13] to explain the transmission process. Because the tomato crop was a sink for whiteflies and TLCV, interventions that reduce vector immigration and survival were predicted to be most effective. The authors' identified the most effective disease control method would be to use of protective netting treated with a persistent insecticide combined with the growth of resistant varieties has the potential to decrease both *B. tabaci* immigration to the crop and to reduce virus inoculation by those insects which do reach the crop.

Alemneh *et al.*[14] proposed and examined an eco-epidemiological deterministic model for the transmission dynamics of maize streak virus (MSV) disease in maize plant. Their model depicted that increasing parameters namely the infection and predation rates made an increase of basic reproduction number which leads to the increase of the number of infected maize population. Hence the authors suggested that to intervene MSV disease, endeavors should be exerted to reduce the contact of infected maize and susceptible leafhopper. In addition, MSV infected maize should be treated using insecticide chemicals. This enabled us to bring-down the infection rate of leafhoppers and it should be administered before the reaching of leafhopper or uprooting. Moreover, infected maize should be burnt from the field.

Optimal control theory has found wide-ranging applications in biological and ecological problems[30]. Particularly, there have been various studies of epidemiological models where optimal control methods have been applied. Berhe et al.[15] formulated a deterministic model to study the effects of implementing continuous controls on the dysentery epidemic model and examined the cost-effectiveness of the optimal control measures of the disease. They took three control parameters namely treatment, sanitation, and educational campaign as a prevention strategy. As a result, they found that the disease probably eradicated by implementing continuous controls in a short period of time. However, utilizing a combination of sanitation of the environment and education campaign was found to be the most cost-effective. Okosun and Makinde[16] derived and analyzed a deterministic model for the transmission of childhood disease incorporating optimal control parameters and investigated the cost-effectiveness of the controls to identify the most effective strategy. They considered control parameters such as improvement of hygiene due

to health education campaign, improvement of treatment of the infected children, and reduction in the loss of disease immunity due to the improvement of vaccination and treatment efficacy. Thus, utilization of these control strategies has declined the disease from the community. Similar results also obtained if educational campaignss as preventive measure and treatment of infected children were used. However, as resources are scarce, the authors proposed that policymakers likely focused on optimal provision of prevention and treatment being it is cost-effective. Bokil *et, al.* [17], investigated, and analyzed optimal control of a vectored plant disease model for a crop with continuous replanting. They considered two plant-vector-virus models which take into account frequency replanting and abundance replanting strategies to study African cassava mosaic virus. They compared the two models with respect to replanting strategies through a combination of mathematical analysis, parameter sensitivity, and optimal control of the disease dynamics. They used optimal control theory to investigate the effects of roguing and insecticide to maximize the healthy plants to be harvested. The simulation results of their models suggested that various optimal control strategies were suitable for the two different replanting practices. Hugo *et, al.* [18], studied optimal control and cost-effectiveness analysis of the TYLCV disease epidemic model. Their model was extended work of [12]. The authors incorporated the time-dependent control to the tomato plants and vector populations in analyzing the cost-effectiveness of control strategies. Thus, they suggested that the use of protective netting and removal of the infected plant is the cost-effective optimal control strategy and was sufficient to combat the epidemic of tomato disease with limited resources.

The major advantage of these early models was to provide a suitable control strategy through the *Transmission threshold* criterion, which is based on the reproductive capacity of the parasite, $\mathcal{R}_0$. To wind-up, this paper is focused on optimal control strategies analysis of tomato yellow leaf curl virus disease model which is adopted from Africa Cassava Mosaic Virus disease's model [11] with the inclusion of exposed class into tomato plant population and incorporate three-time dependent controls representing the interventions.

The organization of the paper is as follows, in section 2, we presented a model consisting of ordinary differential equations that describe the transmission dynamics of tomato yellow leaf curl virus disease and the underlying assumptions. Section 3 is devoted to stability analysis of the model. Section 4 is contained by numerical simulations. Conclusions are given in section 5.

## 2 | MATHEMATICAL MODEL

The model that considered here is a small modification of the model for plant-virus transmission considered in [11]. It is a standard model of SEI type for tomato plants and SI for whiteflies *Bemisia tabaci* B - type insect vector.

The model sub-divides the total tomato plant population into the following sub-classes: Healthy or Susceptible ($S_p$), latently infected ($E_p$), and infective ($I_p$), $K$ is carrying capacity of tomatoes farm. Thus, the total population size of tomato plant is $N_p = S_p + E_p + I_p$. The total insect vector (whitefly *Bemisia tabaci* B - type) population is sub-divided with respect to tomato plant into susceptible (virus-free) ($S_v$) and infective vector ($I_v$). The latent period in the vector between the acquisition of the TYLCV and the ability to transmit the virus is roughly 30 min [19]. Thus, it is assumed to be negligible, i.e., no latent sub-class is defined for the whitefly vector in our model. The whiteflies remain infective for their lifetime. Hence, the total population size of the vector (whitefly) is $N_v = S_v + I_v$.

The net replanting rate of tomato plants is $rS_p(1 - \frac{S_p+E_p+I_p}{K})$ where $r$ is rate replanting healthy tomato. This is because, the model assumed that healthy tomatoes respond proportionately inverse to the extended intraspecific pressure for the healthy, exposed and infected tomato plants, and replanting tomato is restricted by maximum tomato plant availability and the harvesting of healthy, exposed and infected tomato plant reflects the continual turn-over of the tomato plant population. The tomato fruit is either harvested or removed at a rate $g$ or move to the exposed stage by inoculating through contact with infective whiteflies at a rate of $\beta_p$. Moreover, all stages of tomato are assumed to be harvested or removed at the same constant rate. Thus, it is assumed that the force of infection at time $t$ is given by $\beta_p I_v(t) S_p(t)$. Latently infected tomato plants propagate to the infectious stage at a rate of $a$, corresponding to a mean latent period in a tomato plant population of $\frac{1}{a}$. Here it is assumed that the infective tomato remains infected forever. Loss of tomatoes due to natural and disease-related reduction rate of $b$.

The population of whiteflies are assumed to be generated at a rate $c(S_v + I_v)\left(1 - \frac{S_v+I_v}{m(S_p+I_p)}\right)$, where $c$ is vector birth rate and $m$ is rate of vectors maximum abundance. This is because some form of density-dependent constraint is assumed to slow the net population growth rate as vector abundance approaches a maximum abundance. Besides, we assumed that the vector does not immigrate to other hosts in the case of low tomato plant abundance. The vector is either die from natural causes at a rate $e$ or propagate to the infective class by acquiring tomato yellow leaf curl virus through contacts with infected tomatoes at a rate $\beta_v I_p(t) S_v(t)$, where $\beta_v$ is the rate of virus acquisition by a susceptible vector (whitefly) during one visit to an infectious tomato

plant. The protections target mainly the following: (i) Insecticide on tomato plant population: minimize the inoculation efficiency of the vector i.e, reduce $\beta_p$; (ii) Roguing diseased tomato plants: good practice for reducing the source of primary infection, i.e., reduce $g$; (iii) Protective netting: prevent the entry of whitefly vectors, *B. tabaci*, into tomato plots, i.e., reduce $\beta_p$ and $\beta_v$. This is because the only way of controlling TYLCV is by controlling the vector[4]. Thus, the efforts made on these three intervention mechanisms enable to control of the tomato infections due to TYLCV.

Suppose that the control function $u_1(t)$ represents insecticides spray with efficacy $q$, $u_2(t)$ represents roguing diseased tomato plants, and $u_3(t)$ represents protective netting at any time $t$. Besides, controls $u_1, u_2, u_3$ are assumed to be bounded and integrable functions.

It is further assumed that the transmission of the virus by the whitefly vectors is by circulative and persistent mode[4]. Moreover, it is also assumed that infective whiteflies stay infective for life.

Based on the above assumptions the following vector - plant dynamical system is formulated:

$$
\begin{aligned}
\frac{dS_p}{dt} &= rS_p\left(1 - \frac{S_p + E_p + I_p}{K}\right) - (1 - u_3(t))\beta_p I_v S_p - gS_p \\
\frac{dE_p}{dt} &= (1 - u_3(t))\beta_p I_v S_p - (a + g)E_p \\
\frac{dI_p}{dt} &= aE_p - (g + b + u_2(t))I_p \\
\frac{dS_v}{dt} &= c(S_v + I_v)\left(1 - \frac{S_v + I_v}{m(S_p + I_p)}\right) - \beta_v I_p S_v - (qu_1(t) + e)S_v \\
\frac{dI_v}{dt} &= \beta_v I_p S_v - (qu_1(t) + e)I_v
\end{aligned}
\tag{1}
$$

with initial conditions:

$$
S_p(0) \geq 0, E_p(0) \geq 0, I_p(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0, 0 < u_i < 1, i = 1, 2, 3
\tag{2}
$$

## 3 | STABILITY ANALYSIS OF FREE DISEASE EQUILIBRIUM POINT

### 3.1 | Positivity and Boundedness of Solution

For the TYLCV transmission model (1) to be biologically meaningful, it is important to prove that all solutions with non-negative initial data will remain non-negative for all time as is presented in[20].

**Theorem 1.** Let $S_p(t) \geq 0$, $E_p(t) \geq 0$, $I_p(t) \geq 0$, $S_v(t) \geq 0$ and $I_v(t) \geq 0$. The solutions $S_p, E_p, I_p, S_v, I_v$ of the system of differential equation (1) are positive for all $t \geq 0$. Besides, the region $\Omega$ is positively invariant and all solutions starting in $\Omega$ approach, enter, or stay in $\Omega$.

By adding the first four equations in the system (1), we have that the rate at which the total population of tomato plant changes is given by

$$
\frac{dN_p}{dt} = rS_p\left(1 - \frac{S_p + E_p + I_p}{K}\right) - g(S_p + E_p + I_p) - bI_p
$$

Since $N_p = S_p + E_p + I_p$ and $S_p \leq N_p$, we have

$$
\frac{dN_p}{dt} \leq rN_p\left(1 - \frac{N_p}{K}\right) - gN_p \quad t \geq 0
$$

Thus,

$$
N_p \leq K\left(\frac{r - g}{r}\right) \quad t \geq 0
$$

Similarly, by adding the last three equations of system (1), we have that the rate at which the total population of whitefly vector changes is given by

$$
\frac{dN_v(t)}{dt} = c(S_v + I_v)\left(1 - \frac{S_v + I_v}{m(S_p + I_p)}\right) - e(S_v + I_v)
\tag{3}
$$

Since $N_v = S_v + I_v$ and $K > S_p + I_p$, then equation (3) can be written as

$$\frac{dN_v(t)}{dt} \geq cN_p\left(1 - \frac{N_v}{mK}\right) - eN_v \quad t \geq 0 \tag{4}$$

If we factorize equation (4), then we can obtain

$$\frac{dN_v(t)}{dt} \geq (c-e)N_v\left(1 - \frac{N_v}{mK\left(\frac{(c-e)}{c}\right)}\right) - eN_v \quad t \geq 0 \tag{5}$$

Thus,

$$N_v \leq mK\left(\frac{c-e}{c}\right) \quad t \geq 0$$

The region $\Omega = \Omega_p \times \Omega_v$ with

$$\Omega_p = \left\{(S_p, E_p, I_p) \in \mathcal{R}_+^3 : S_p + E_p + I_p \leq K\left(1 - \frac{g}{r}\right)\right\} \& \Omega_v = \left\{(S_v, I_v) \in \mathcal{R}_+^2 : S_v + I_v \leq mK\left(1 - \frac{e}{c}\right)\right\}$$

This implies that all solutions of tomato plants population only are confined in the feasible region $\Omega_p$ and all solutions of the whiteflies population are confined in $\Omega_v$.

Therefore, the biological feasibility, model of system (1) is studied in the following region:

$$\Omega = \left\{(S_p, E_p, I_p, S_v, I_v) \in \mathcal{R}_+^5 : N_p \leq K\left(\frac{r-g}{r}\right); N_v \leq mK\left(\frac{c-e}{c}\right)\right\}.$$

Thus $\Omega$ is positively invariant. This means, solutions of the model system with positive initial data remain positive for all time $t \geq 0$ and are bound in the region $\Omega$. Therefore, the model is mathematically and epidemiologically well-posed.

## 3.2 | Analysis of the model with constant controls

In this section, it is assumed that the control parameters are constant and determine the basic reproductive number, the steady states, and their stability.

### 3.2.1 | Local stability of the disease-free equilibrium

The disease free equilibrium of the tomato yellow leaf curl virus disease model (1) exists and is given by

$$\mathcal{E}_0 = \left(K\left(1 - \frac{g}{r}\right), 0, 0, mK\left(1 - \frac{qu_1 + e}{c}\right)\left(1 - \frac{g}{r}\right), 0\right)$$

The basic reproduction number, $\mathcal{R}_0$, is calculated by using the next generation matrix[21]. To obtain $\mathcal{R}_0$ for model (1), let the vector of disease states

$$x = \left(E_p, I_p, I_v\right)^T$$

Then the model (1) can be written as

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x)$$

where

$$\mathcal{F}(x) = \begin{pmatrix} (1-u_3)\beta_p I_v S_p \\ 0 \\ \beta_v I_p S_v \end{pmatrix} \quad \text{and} \quad \mathcal{V}(x) = \begin{pmatrix} (a+g)E_p \\ (g+b+u_2)I_p - aE_p \\ (qu_1+e)I_v \end{pmatrix}$$

Calculate the Jacobian matrix ($\mathbf{F}$ and $\mathbf{V}$) of $\mathcal{F}$ and $\mathcal{V}$ by derivating with respect to the infected classes ($E_p, I_p, I_v$) at the disease-free equilibrium point $\mathcal{E}_0$. This gives

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & \beta_p K\left(1-u_3\right)\left(1-\frac{g}{r}\right) \\ 0 & 0 & 0 \\ 0 & \beta_v mK\left(1-\frac{e+qu_1}{c}\right)\left(1-\frac{g}{r}\right) & 0 \end{pmatrix} \quad \text{and} \quad \mathbf{V} = \begin{pmatrix} a+g & 0 & 0 \\ -a & g+b+u_2 & 0 \\ 0 & 0 & qu_1+e \end{pmatrix}$$

Thus

$$\mathbf{V}^{-1} = \begin{pmatrix} \frac{1}{a+g} & 0 & 0 \\ \frac{a}{(a+g)(b+g+u_2)} & \frac{1}{b+g+u_2} & 0 \\ 0 & 0 & \frac{1}{e+qu_1} \end{pmatrix}$$

So that

$$\mathbf{FV}^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_p K(1-u_3)(r-g)}{r(e+qu_1)} \\ 0 & 0 & 0 \\ \frac{\beta_v amK(c-qu_1-e)(r-g)}{cr(a+g)(b+g+u_2)} & \frac{\beta_v mK(r-g)(c-qu_1-e)}{cr(b+g+u_2)} & 0 \end{pmatrix}$$

The basic reproduction number, $\mathcal{R}_0 = \rho(\mathbf{FV}^{-1})$, for the model (1) is

$$\mathcal{R}_0 = \sqrt{\frac{\beta_p \beta_v amK^2(r-g)^2(1-u_3)(c-qu_1-e)}{cr^2(a+g)(g+b+u_2)(qu_1+e)}} \tag{6}$$

Theorem 2 below follows from Theorem 2 of [22].

**Theorem 2.** The disease-free equilibrium (DFE) $\mathcal{E}_0$ of Eq. (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$.

The epidemiological implication of Theorem 2 is that the transmission of TYLCV can be controlled by having $\mathcal{R}_0 < 1$ if the initial total numbers of the sub-populations involved in Eq. (1) are in the basin of attraction of $\mathcal{E}_0$. To ensure that eliminating the disease is independent of the initial size of the subpopulation, the disease-free equilibrium must be globally asymptotically stable when $\mathcal{R}_0 < 1$. This is what we check next.

**Theorem 3.** The DFE $\mathcal{E}_0$ of Eq. (1) is globally asymptotically stable (GAS) for $\mathcal{R}_0 < 1$.

*Proof.* To prove the theorem, we use Kamgang-Sallet Stability Theorem in [23]. Let $X = (X_1, X_2)$ with $X_1 = (S_p, S_v) \in \mathbb{R}^2$ and $X_2 = (E_p, I_p, I_v) \in \mathbb{R}^3$. Then the system (1) can be written as

$$\dot{X}_1 = A_1(X)(X_1 - X_1^*) + A_{12}(X)X_2, \tag{7}$$

$$\dot{X}_2 = A_2(X)X_2, \tag{8}$$

where $X_1^* = \left( K\left(1 - \frac{g}{r}\right), mK\left(1 - \frac{qu_1+e}{c}\right)\left(1 - \frac{g}{r}\right) \right)$

$$A_1(X) = \begin{pmatrix} -(r-g) & 0 \\ -m\left(1 - \frac{qu_1+e}{c}\right)^2 & -(c-qu_1-e) \end{pmatrix}$$

$$A_{12}(X) = \begin{pmatrix} -\frac{rS_p}{K} & -\frac{rS_p}{K} & -(1-u_3)\beta_p S_p \\ 0 & -\beta_v S_v + \frac{c}{m}\left(\frac{S_v+I_v}{S_p+I_p}\right)^2 & c\left(1 - \frac{2S_v}{m(S_p+I_p)}\right) - \frac{2I_v}{m(S_p+I_p)} \end{pmatrix}$$

and

$$A_2(X) = \begin{pmatrix} -(a+g) & 0 & (1-u_3)\beta_p S_p \\ a & -(g+b+u_2) & 0 \\ 0 & \beta_v S_v & -(qu_1+e) \end{pmatrix}$$

We show that the five sufficient conditions of Kamgang-Sallet Theorem are satisfied as follows

1. The system (1) is a dynamical system on $\Omega$. This is proved in Theorem 1.

2. The equilibrium $X_1^*$ is GAS for the subsystem $\dot{X}_1 = A_1(X_1, 0)(X_1 - X_1^*)$. This is obvious from the structure of the involved matrix.

3. As can be seen from the elements, the matrix $A_2(X)$ is Metzler (i.e., all the off-diagonal elements are nonnegative) and irreducible for any given $X \in \Omega$.

4. There exists an upper-bound matrix $\bar{A}_2$ for the set

$$\mathcal{M} = A_2(X) : X \in \Omega.$$

More precisely,

$$A_2(X) = \begin{pmatrix} -(a+g) & 0 & (1-u_3)\beta_p K\left(1-\frac{g}{r}\right) \\ a & -(g+b+u_2) & 0 \\ 0 & \beta_v m K\left(1-\frac{g}{r}\right)\left(1-\frac{qu_1+e}{c}\right) & -(qu_1+e) \end{pmatrix}$$

is an upper-bound of $\mathcal{M}$.

5. For $\mathcal{R}_0 < 1$ in Eq. (6).

$$\alpha(\bar{A}_2(X)) = \max\{Re(\lambda) : \lambda \text{ is an eigenvalue of } \bar{A}_2\} \leq 0$$

Thus, all eigenvalues of $A$ are negative for $\mathcal{R}_0 < 1$ in Eq. 6. $\qquad \square$

Hence, by the Kamgang-Sallet Stability Theorem, the disease-free equilibrium is globally asymptotically stable for $\mathcal{R}_0 < 1$.

For any initial data, Theorem 3 implies that any solution of the system (1) converges to the DFE when $\mathcal{R}_0 < 1$. In addition, the theorem implies that the model is without backward bifurcation for $\mathcal{R}_0 < 1$. In this case, the classical approach making $\mathcal{R}_0 < 1$ to eliminate TYLCV disease from the farm is sufficient.

Next, we want to check that the system (1) has at least one endemic equilibrium (EE) point for $\mathcal{R}_0 > 1$. Let

$$\mathcal{E}^* = (S_p^*, E_p^*, I_p^*, S_v^*, I_v^*)$$

be an EE of system (1). By setting the right-hand side of (1) equal to zero, we obtain

$$
\begin{aligned}
S_p^{**} &= \frac{K}{2}\left(1-\frac{g}{r}\right) - \frac{g+b+u_2+a}{2a}I_p + \frac{M}{2} \\
E_p^{**} &= \frac{g+b+u_2}{a}I_p \\
S_v^{**} &= \frac{m(qu_1+e)(c-qu_1-e)}{c(\beta_v I_p+qu_1+e)}\left[\frac{K}{2}\left(1-\frac{g}{r}\right) + \frac{a-g-b-u_2}{2a}I_p + \frac{M}{2}\right] \\
I_v^{**} &= \frac{m(c-qu_1-e)\beta_v}{c(\beta_v I_p+qu_1+e)}\left[\frac{K}{2}\left(1-\frac{g}{r}\right) + \frac{a-g-b-u_2}{2a}I_p + \frac{M}{2}\right]I_p
\end{aligned}
\tag{9}
$$

where

$$M = \sqrt{\left[K\left(1-\frac{g}{r}\right) - \frac{g+b+u_2}{a}I_p\right]^2 - \frac{4K(a+g)(g+b+u_2)}{ar}I_p}$$

It can be shown that the equilibria of the model satisfy the following polynomial

$$f(I_p) = c_4 I_p^4 + c_3 I_p^3 + c_2 I_p^2 + c_1 I_p + c_0$$

where

$$
\begin{aligned}
&c_4 = B_3^2 - A_1 A_4, \quad c_3 = 2B_2 B_3 - A_1 A_4 - A_2 A_3, \\
&c_2 = B_2^2 + 2B_1 B_2 - A_1 A_2 - K\left(1-\frac{g}{r}\right)\left[A_4 + A_2 K\left(1-\frac{g}{r}\right)\right], \\
&c_1 = 2B_1 B_2 - (A_1 + A_2)\left[K\left(1-\frac{g}{r}\right)\right]^2, \quad c_0 = B_1^2 - 4\left[K\left(1-\frac{g}{r}\right)\right]^4, \quad A_1 = -\frac{2(g+b+u_2)}{a}K\left(1-\frac{g}{r}\right), \\
&A_2 = \left(\frac{g+b+u_2}{a}\right)^2, \quad A_3 = -\frac{K}{a}\left[\frac{2(g+b+u_2+a)}{a}\left(1-\frac{g}{r}\right) + \frac{(a+g)(g+b+u_2)}{r}\right], \quad A_4 = \frac{g+b+u_2+a}{a}, \\
&B_1 = \frac{4}{\mathcal{R}_0^2}K\left(1-\frac{g}{r}\right)\left[K\left(1-\frac{g}{r}\right) - 2\right], \quad B_3 = \frac{a^2-(g+b+u_2)^2-(g+b+u_2+a)a}{a^2} \\
&B_2 = \frac{4}{\mathcal{R}_0^2}K\left(1-\frac{g}{r}\right)\left[K\left(1-\frac{g}{r}\right)\frac{\beta_v}{qu_1+e} + \frac{2(g+b+u_2)}{a} + \frac{2(g+b+u_2+a)}{a}\right] + \frac{(a+g)(g+b+u_2)K}{ar}
\end{aligned}
$$

**Theorem 4.** When $\mathcal{R}_0 > 1$, the model (1) has at least one endemic equilibrium, which is locally asymptotically stable for $\mathcal{R}_0$ close to one.

The stability of the EE is guaranteed by using the Center Manifold Theorem in[24].

## 3.3 | Sensitivity analysis of model parameters

Sensitivity analysis assists to build confidence in the model by studying the uncertainty associated with parameters in the model. This is because many parameters in the system dynamics models characterize quantities that are very difficult or even impossible to measure accurately in the real world. It helps to comprehend the dynamics of the system under study. In general, sensitivity analysis is carried out to establish which input parameters contribute the most to output variability[25].

Now let's carried out the sensitivity analysis in order to identify the parameters that have a high impact on the basic reproductive number ($\mathcal{R}_0$).

**Definition 1.** The normalized sensitivity index of a variable, $\mathcal{R}_0$, that depends differentiably on a parameter, $p$, is defined as

$$\sigma_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \frac{p}{\mathcal{R}_0}$$

### 3.3.1 | Sensitivity Indicies of Basic Reproductive Number

Here, the sensitivity of $\mathcal{R}_0$ to every parameters of the model is drived. Hence, the sensitivity index of $\mathcal{R}_0$ with respect to $K$ and is equal to 1. It is equal to 0.5 with respect to $m$, $\beta_p$ and $\beta_v$. For the rest parameters, the following

$$\sigma_r^{\mathcal{R}_0} = -\frac{g}{g-r}, \sigma_e^{\mathcal{R}_0} = \frac{ce}{2(e+qu_1)(e-c+qu_1)}, \sigma_g^{\mathcal{R}_0} = \frac{g(2(ab+2gr)+(a+b)(g+r))}{2(a+g)(b+g)(g-r)}$$

$$\sigma_a^{\mathcal{R}_0} = \frac{g}{2(a+g)}, \sigma_c^{\mathcal{R}_0} = -\frac{e+qu_1}{2(e-c+qu_1)}, \sigma_{u_1}^{\mathcal{R}_0} = \frac{cqu_1}{2(e+qu_1)(e-c+qu_1)}$$

$$\sigma_b^{\mathcal{R}_0} = -\frac{b}{2(b+g)}, \sigma_{u_2}^{\mathcal{R}_0} = \frac{u_2}{2(u_2-1)}, \sigma_{u_3}^{\mathcal{R}_0} = \frac{u_3}{2(u_3-1)}$$

Since most of the expressions for sensitivity indices are complex, the sensitivity indices are evaluated at the baseline parameter values given in Table 2 . The sensitivity index of $\mathcal{R}_0$ with respect to $r$, for example, is:

$$\begin{aligned}
\frac{r}{\mathcal{R}_0} \frac{\partial \mathcal{R}_0}{\partial r} &= -\frac{g}{g-r} \\
&= -\frac{0.0121}{0.0121 - 0.01} \\
&= -5.7619
\end{aligned}$$

The detail sensitivity indices of $\mathcal{R}_0$, resulting from the evaluation of the eight different parameters of the model are shown below.

**TABLE 1** Sensitivity indices of $\mathcal{R}_0$

| Parameters | Prameter description | Sensitivity index |
|:---:|:---|:---:|
| $r$ | Rate of replanting of healthy tomato | + ve |
| $g$ | Rate at tomatoes fruit are harvested or removed | - ve |
| $K$ | Carrying capacity of tomatoes farm | + ve |
| $u_1$ | Optimal control due to insecticides spray | - ve |
| $u_2$ | Optimal control due to roguing diseased tomato plants | - ve |
| $u_3$ | Optimal control due to protective netting | - ve |
| $m$ | Rate of vectors maximum abundance | + ve |
| $\beta_p$ | Rate of inoculation of health tomato plant | + ve |
| $\beta_v$ | Rate of virus acquisition by susceptible vectors | + ve |
| $b$ | Loss rate of tomatoes due to infection | - ve |
| $a$ | Propagation rate from exposed plant to infected plant | + ve |

The parameters are arranged from most sensitive to least. The most sensitive parameters are the rate of replanting of healthy tomato, the rate at tomatoes fruit are harvested or removed, carrying capacity of tomatoes farm, rate of vectors maximum abundance, rate of inoculation of the health tomato plant and rate of virus acquisition by susceptible vectors and the least sensitive parameter is loss rate of tomatoes due to infection. The parameters that reduce $\mathcal{R}_0$ can be used as control parameters.

### 3.4 | Interpretation of sensitivity indices

The sensitivity indices of the basic reproductive number with respect to the main parameters are found in Table 1 . The parameters with the most important that have positive indices are $\beta_p$, $\beta_v$, $r$, $K$, $m$, $c$ and $a$ and those have negative indices are $g$, $e$ and $b$.

The results show that when the inoculation of health tomato plant rate and virus acquisition rate by susceptible vectors increase, the basic reproduction number increase as a result of TYLCV disease propagated since infected plants and infective vectors are both infectious. When the rate of replanting of healthy tomato and whitefly vector birth rate is increased, more tomato plants and whitefly vectors are exposed to TYLCV and these increase their probability of catching the virus and contribute to the disease spread. The propagation rate from exposed class to infectious class increase the number of infectious tomato plants and vectors and these contribute to the disease spread. On the other hand, tomato fruit harvested or removed and death rate of vectors are reduce the size of tomato plant population and whitefly vector population and thus limits the number of tomato plants and whitefly vectors who might be infected by TYLCV disease. Lose rate of tomatoes due to infection reduces the number of TYLCV diseases.

# 4 | ANALYSIS OF OPTIMAL CONTROL

If $\mathbf{x(t)}$ represents the tomato plant population to be protected via insecticides, cultural techniques, and virus-resistant cultivars. Insecticides reduce the number and movement of the whitefly vector, cultural techniques, such as roguing, avoidance, crop residue disposal, reduce the amount of secondary spread within a field when incidences are low at the beginning of the season when TYLCV-infected transplants are used, and virus-resistant cultivar is the best approach to reduce losses due to infection by TYLCV[31]. Thus, the aim of this study is to minimize the multiple objective cost functional $J$ considering the costs of control methods of exposed and infected tomato plants.

If $q$ is the efficacy of insecticide spray, $c_1$ and $c_2$ are cost factors due to the size of infectious tomato plants and whiteflies population, $p_1$, $p_2$ and $p_3$ represents the weight attached on the cost control methods, then the cost rate at which TYLCV disease controlled at any time $t$ can be given by:

$$f(\mathbf{x}, \mathbf{u}, t) = c_1 I_p(t) + c_2 I_v(t) + \frac{1}{2} \left[ p_1 q u_1^2(t) + p_2 u_2^2(t) + p_3 u_3^2(t) \right]$$

where, $\mathbf{x} = (I_p, I_v), \mathbf{u} = (u_1, u_2, u_3)$. Since implementation of any intervention has decreasing costs, it is customary to take a non-linear cost function. Hence, the simplest non-linear function, the quadratic, in modelling the cost of the interventions is used.

Therefore, an optimal control $\mathbf{u}^* = (u_1^*, u_2^*, u_3^*)$ are going to search such that

$$J(\mathbf{u}^*) = \min\{J(\mathbf{u}) : \mathbf{u} \in \mathcal{U}\}, \tag{10}$$

where
$\mathcal{U} = \{(u_1, u_2, u_3) \in L^1(0, T) \mid u_i \text{ is Lebesgue measurable}, 0 \leq u_i(t) \leq 1, t \in [0, T], \text{ for } i = 1, 2, 3\}$ is the set of admissible controls.

To sum - up, the optimal control problem has the following form.

$$\min_{\mathbf{u}} \ J(u_1, u_2, u_3) = \int_0^T f(\mathbf{x}, \mathbf{u}, t) \mathrm{d}t$$

**subject to** $\tag{11}$

$$\frac{\mathrm{d}\mathbf{x}}{\mathrm{d}t} = F(\mathbf{x}, \mathbf{u}, t), \quad \mathbf{x}(0) = \mathbf{x}_0, \quad \mathbf{x} \geq 0$$

$$0 \leq u_i(t) \leq 1 \quad \forall t \in [0, T], i = 1, 2, 3.$$

## 4.1 | Pontryagin's maximum principle

Since our model has no terminal constraints, it is a normal optimal control problem and hence the Hamiltonian takes the form:

$$H(\mathbf{x}, \mathbf{u}, t) = f(\mathbf{x}, \mathbf{u}, t) + \sum_{i=1}^{5} \lambda_i(t) F_i(\mathbf{x}, \mathbf{u}, t).$$

where $F_i(\mathbf{x}, \mathbf{u}, t)$ is the right hand side of the differential equations of the $i^{th}$ state variable. By using Pontryagin's Maximum Principle and the existence of results obtained for optimal control, we obtain

**Theorem 5.** There exists an optimal control $u_1^*$, $u_2^*$, $u_3^*$ and corresponding solution, $\mathbf{x} = [S_p^*, E_p^*, I_p^*, S_v^*, I_v^*]$, that minimizes $J(u_1, u_2, u_3)$ over $\Omega$. Furthermore, there exist adjoint functions $\lambda_i$, $i = 1, 2, 3, 4, 5$ such that

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= \lambda_1\left(rS_p\left(1 - \frac{S_p + E_p + I_p}{K}\right) - (1 - u_3(t))\beta_p I_v S_p - gS_p\right) \\
\frac{d\lambda_2}{dt} &= \lambda_2\left((1 - u_3(t))\beta_p I_v S_p - (a + g)E_p\right) \\
\frac{d\lambda_3}{dt} &= \lambda_3\left(aE_p - (g + b + u_2)I_p\right) \\
\frac{d\lambda_4}{dt} &= \lambda_4\left(c(S_v + I_v)\left(1 - \frac{S_v + I_v}{m(S_p + I_p)}\right) - \beta_v I_p S_v - qu_1(t)S_v - eS_v\right) \\
\frac{d\lambda_5}{dt} &= \lambda_5\left(\beta_v I_p S_v - qu_1(t)I_v - eI_v\right)
\end{aligned}
\tag{12}
$$

with transversality condition

$$
\lambda_i(T) = 0 \quad \text{for } i = 1, 2, 3, 4, 5
\tag{13}
$$

By using Pontryagin's Maximum Principle and the existence result for the optimal control (Makinde *et al.* [26]), we arrive at the following theorem.

**Theorem 6.** The optimal control $u_1^*$, $u_2^*$, $u_3^*$, that minimizes $J(u_1, u_2, u_3)$ over $\Omega$ is expressed as

$$
\begin{aligned}
u_1^* &= \min\left(1, \max\left(0, \frac{(I_v\lambda_5 + \lambda_4 S_v)q}{p_1 q}\right)\right) \\
u_2^* &= \min\left(1, \max\left(0, \frac{\lambda_3 I_p}{p_2}\right)\right) \\
u_3^* &= \min\left(1, \max\left(0, \frac{(\lambda_2 - \lambda_1)\beta_p S_p I_v}{p_3}\right)\right)
\end{aligned}
\tag{14}
$$

*Proof.* (Fleming *et al.* [27]) provides the existence of an optimal control due to the convexity of integrand with respect to $(u_1, u_2, u_3)$, a *priori* boundedness of the state solutions, and the *Lipschitz* property of the state system with respect to the state variables. Employing Pontryagins Maximum Principle, we have

$$
\frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial \mathbf{x}_i}
$$

where $\lambda_i$, $i = 1, 2, 3, 4, 5$. Therefore, the adjoint function with each state variables is calculated as follows

$$
H(\mathbf{x}, \mathbf{u}, t) = f(\mathbf{x}, \mathbf{u}, t) + \lambda_1 \frac{dS_p}{dt} + \lambda_2 \frac{dE_p}{dt} + \lambda_3 \frac{dI_p}{dt} + \lambda_4 \frac{dS_v}{dt} + \lambda_5 \frac{dI_v}{dt}
$$

Applying, the Pontryagin's maximum principle, the Hamiltonian equation can be written as:

$$
\begin{aligned}
H = {}& c_1 I_p(t) + c_2 I_v(t) + \tfrac{1}{2}(p_1 q u_1^2(t) + p_2 u_2^2(t) + p_3 u_3^2(t)) \\
&+ \lambda_1\left(rS_p\left(1 - \frac{S_p + E_p + I_p}{K}\right) - (1 - u_3(t))\beta_p I_v S_p - gS_p\right) \\
&+ \lambda_2\left((1 - u_3(t))\beta_p I_v S_p - (a + g)E_p\right) \\
&+ \lambda_3\left(aE_p - (g + b + u_2(t))I_p\right) \\
&+ \lambda_4\left(c(S_v + I_v)\left(1 - \frac{S_v + I_v}{m(S_p + I_p)}\right) - \beta_v I_p S_v - qu_1(t)S_v - eS_v\right) \\
&+ \lambda_5\left(\beta_v I_p S_v - (qu_1(t) - e)I_v\right)
\end{aligned}
$$

Considering the existence of adjoint functions $\lambda_i$, $i = 1, 2, 3, 4, 5$ satisfying

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= -\frac{\partial H}{\partial S_p} = \lambda_1\left(r\left(\frac{E_p + I_p + S_p}{K} - 1\right) - \beta_p I_v(u_3 - 1) + \frac{rS_p}{K}\right) + \beta_p I_v \lambda_2(u_3 - 1) - \lambda_4 \frac{c(S_v + I_v)^2}{m(S_p + I_p)^2} \\
\frac{d\lambda_2}{dt} &= -\frac{\partial H}{\partial E_p} = \lambda_2(a + g) - \lambda_3 a + \frac{\lambda_1 rS_p}{K} \\
\frac{d\lambda_3}{dt} &= -\frac{\partial H}{\partial I_p} = \lambda_3(b + g + u_2) - c_1 - \beta_v \lambda_5 S_v + \frac{\lambda_1 S_p r}{K} + \lambda_4\left(\beta_v S_v - \frac{c(S_v + I_v)^2}{m(S_p + I_p)^2}\right) \\
\frac{d\lambda_4}{dt} &= -\frac{\partial H}{\partial S_v} = \lambda_4\left(e - c + qu_1 + \beta_v I_p + \frac{2c(S_v + I_v)}{m(S_p + I_p)}\right) - \lambda_5 \beta_v I_p \\
\frac{d\lambda_5}{dt} &= -\frac{\partial H}{\partial I_v} = \lambda_4\left(\frac{c(I_v + S_v)}{m(Ip + S_p)} - 1\right) + \frac{c(I_v + S_v)}{m(I_p + S_p)} - c_2 + \lambda_5(e + qu_1) + \beta_p S_p(u_3 - 1)(\lambda_2 - \lambda_1)
\end{aligned}
\tag{15}
$$

with transversality condition $\lambda_i(T) = 0$, $i = 1, 2, 3, 4, 5$ for the control set $u_i$, hence

$$\frac{\partial H}{\partial \mathbf{u}_i} = 0, \quad \text{where } i = 1, 2, 3 \Rightarrow \text{the optimality condition}$$

Computed at the optimal control pair and respective corresponding states, which leads to the stated adjoint system (12) and (13),[28]. By taking into account the optimality conditions,

$$\frac{\partial H}{\partial u_1} = 0, \quad \frac{\partial H}{\partial u_2} = 0, \quad \frac{\partial H}{\partial u_3} = 0$$

and determine the values for $u_1^*$, $u_2^*$, $u_3^*$, subject to the constraints, the characterizations (14) can be obtained,

$$
\begin{aligned}
&\frac{\partial H}{\partial u_1} = p_1 q u_1 - I_v \lambda_5 q - \lambda_4 S_v q \quad \Rightarrow \quad \frac{\partial H}{\partial u_1}\Big|_{u_1 = u_1^*} = p_1 q u_1^* - I_v \lambda_5 q - \lambda_4 S_v q = 0 \\
&u_1^* = \frac{(I_v \lambda_5 + \lambda_4 S_v)q}{p_1 q} \quad \Leftrightarrow \quad u_1^* = \min\left(1, \max\left(0, \frac{(I_v \lambda_5 + \lambda_4 S_v)q}{p_1 q}\right)\right) \\
&\frac{\partial H}{\partial u_2} = p_2 u_2 - I_p \lambda_3 \quad \Rightarrow \quad \frac{\partial H}{\partial u_2}\Big|_{u_2 = u_2^*} = p_2 u_2^* - I_p \lambda_3 = 0 \\
&u_2^* = \frac{\lambda_3 I_p}{p_2} \quad \Leftrightarrow \quad u_2^* = \min\left(1, \max\left(0, \frac{\lambda_3 I_p}{p_2}\right)\right) \\
&\frac{\partial H}{\partial u_3} = p_3 u_3 + \beta_p I_v \lambda_1 S_p - \beta_p I_v \lambda_2 S_p \quad \Rightarrow \quad \frac{\partial H}{\partial u_3}\Big|_{u_3 = u_3^*} = p_3 u_3^* + \beta_p I_v \lambda_1 S_p - \beta_p I_v \lambda_2 S_p = 0 \\
&u_3^* = \frac{(\lambda_2 - \lambda_1)\beta_p S_p I_v}{p_3} \quad \Leftrightarrow \quad u_3^* = \min\left(1, \max\left(0, \frac{(\lambda_2 - \lambda_1)\beta_p S_p I_v}{p_3}\right)\right)
\end{aligned}
\tag{16}
$$

$\square$

# 5 | NUMERICAL RESULTS AND DISCUSSIONS

Simulations are carried out to determine the behavior of system (1). For this purpose, parameter values listed in Table 2 were used. Most parameter values were taken directly from[12] and the rest were estimated from the data found in[4] and[32]. The estimated parameters were calculated as follows: Since the life span of whiteflies is $20 - 50$ days, the death rate was calculated as the inverse of life span, i.e., $e = \frac{1}{(20+50)/2} = \frac{1}{35} = 0.0286 \, day^{-1}$. Depending on cultivars, tomato fruits could be made ready for harvest at about 75 to 90 $days$ after transplanting[32] and tomato fruits' harvested or removed rate, $g$, can be calculated as $g = \frac{1}{(75+90)/2} = 0.0121 \, day^{-1}$. Since the exposed period of the tomato plant is $10 - 14$ days[4], propagation rate from exposed plant to infected plant is calculated as $a = \frac{1}{(10+14)/2} = 0.0833 \, day^{-1}$.

The main objective of this study was to assess the impact of control strategies such as insecticides, protective netting, and cultural practice on the transmission of TYLCV. In order to support the analytical results, graphical representations of various strategies are visualized for determining its impact whenever the control is applied to the system[12].

## 5.1 | Optimal control effect on the model

Now it is time to look at the effect of different optimal control strategies on the propagation of TYLCV disease. It is well known that there is no single management option to control the disease. This makes management of TYLCV is challenging and costly[4]. A combination of management options is necessary to successfully manage the disease and limit losses[4]. For instance, a combination of cultural and chemical are required[31]. Therefore, the following optimal control strategies on the propagation of TYLCV disease in the tomato plant population are numerically investigated.

- ♣ Strategy I: Combination of use of insecticides spray and protective netting.

- ♣ Strategy II: Combination of use of roguing diseased tomato plants and protective netting.

- ♣ Strategy III: Combination of use of insecticides spray and roguing diseased tomato plants.

- ♣ Strategy IV: Combination of use of insecticides spray, roguing diseased tomato plants and protective netting.

The optimal control is obtained by solving the optimality system (13), (14) and (15). An iterative scheme is used for solving the optimality system. Thus, the state equations are solved with a guess for the controls over the simulated time using the

**TABLE 2** Model parameters and values used in simulation.

| Parameters | Standard Values | Reference sources |
|:---:|:---:|:---:|
| $r$ | $0.121 \ day^{-1}$ | 12 |
| $K$ | $1000 \ beds^{-1}$ | Assumed |
| $e$ | $0.0286 \ day^{-1}$ | Estimated |
| $c$ | $0.50 \ day^{-1}$ | 12 |
| $g$ | $0.01 \ day^{-1}$ | Estimated |
| $a$ | $0.075 \ day^{-1}$ | Estimated |
| $m$ | $1500 \ plant^{-1}$ | Assumed |
| $b$ | $0.003 \ day^{-1}$ | 12 |
| $q$ | $0.75$ | Assumed |
| $c_1$ | $ \$ \ 10$ | Assumed |
| $c_2$ | $ \$ \ 5$ | Assumed |
| $p_1$ | $ \$ \ 0.006$ | Assumed |
| $p_2$ | $ \$ \ 0.003$ | Assumed |
| $p_3$ | $ \$ \ 0.005$ | Assumed |
| $\beta_p$ | $0.01 \ vector^{-1} \ day^{-1}$ | 12 |
| $\beta_v$ | $0.0003 \ plant^{-1} \ day^{-1}$ | 12 |

fourth-order Runge-Kutta scheme. Because of the transversality conditions (15), the adjoint equations are solved by a backward fourth-order Runge-Kutta scheme using the current iterations solutions of the state equation. Then the controls are updated by using a convex combination of the previous controls and the value from the characterizations (16). Generally, it can be written as $u_{current} \times (1 - \alpha^k) + u_{previous} \times \alpha^k$ where $k$ is the current iteration and $0 < \alpha < 1$[30]. This process is repeated and iterations stopped if the values of the unknowns at the previous iterations are very close to the ones at the present iterations[30].

We assume that $p_1 > p_3 > p_2$. This assumption is based on the facts that the cost associated with $u_1$, $u_2$ and $u_3$ which is the cost of spray of insecticides applied five times per season and the use of roguing diseased tomato plants which mainly labor cost[31], and the cost associated with protective netting. Thus, $c_1 = 1$, $c_2 = 0.025$, $p_1 = 0.006$, $p_2 = 0.003$ and $p_3 = 0.005$ are chosen and use parameter values from Table 2 . The initial state variables are chosen as $S_p(0) = 384$, $E_p(0) = 17$, $I_p(0) = 69$, $S_v(0) = 768$ and $I_v(0) = 138$.

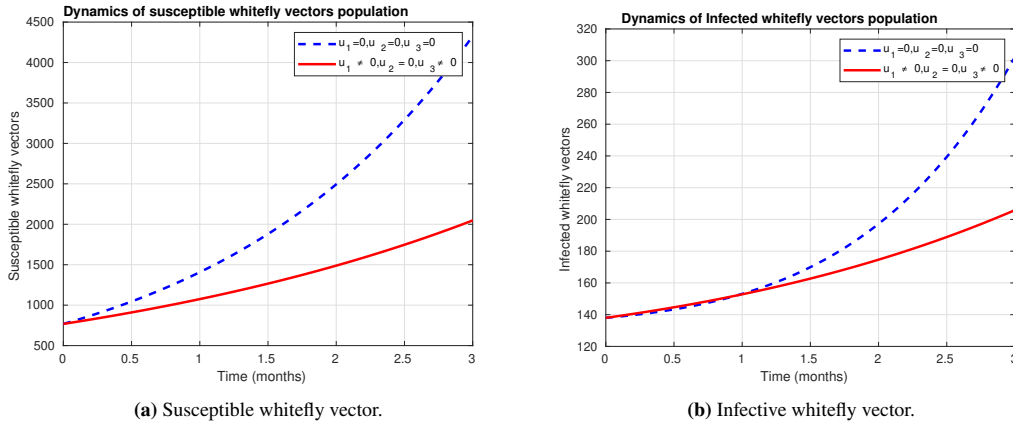# STRATEGY I: COMBINATION OF USE OF INSECTICIDES SPRAY AND PROTECTIVE NETTING

The insecticides spray control $u_1$ and protective netting control $u_3$ are used to optimize the objective function $J$ while roguing diseased tomato plant control $u_2$ is set to zero.

Figure 1 showed that the number of healthy tomato plants is increased gradually while the infected tomato plant population decreased with time in the presence of control. On the other hand without control, the number of infected tomatoes escalate while the healthy tomato plant population is reduced. This is logical because insecticides spray and protective netting help to reduce the incidence of TYLCV disease.

Figure 2 depicted that without control both susceptible and infected insect vectors increase but in the presence of control strategies the insect vector population decreases. This is because we assume that the tomato plant is the only host for the insect vector and thus suffers from lack of food. This is justifiable the protective net decreases the number of invading whiteflies into covered net and insecticide spray is effective against the TYLCV infection.

**(a)** Healthy tomato plant.

**(b)** Infected tomato plant.
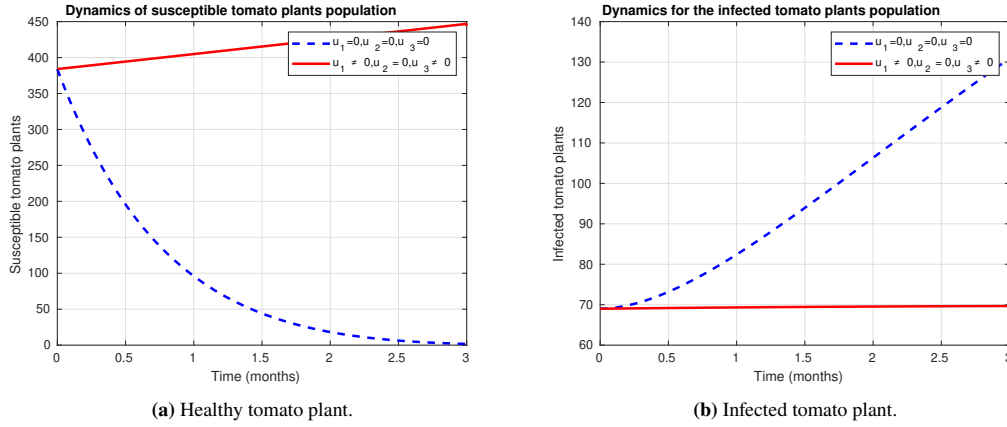
**FIGURE 1** The impact of insecticides spray and protective netting on tomato plant population.



**(a)** Susceptible whitefly vector.

**(b)** Infective whitefly vector.

**FIGURE 2** The impact of insecticides spray and protective netting on whitefly vector population.

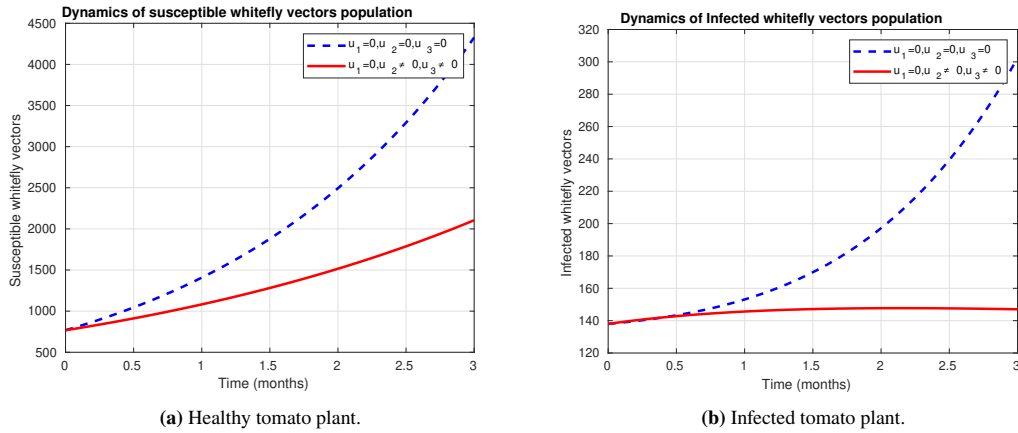## STRATEGY II: COMBINATION OF USE OF ROGUING DISEASED TOMATO PLANTS AND PROTECTIVE NETTING

The combination use of insecticides spray $u_1$ and roguing diseased tomato plants control $u_2$ strategies are used to optimize the objective function $J$ while protective netting control $u_3 = 0$ strategy is set to zero.

Figure 3 (a) illustrated that the healthy tomato plant increases with time as control is used and decreased to zero without the use of control. On the other hand 3 (b) showed that the escalation of infected tomato plants without control and deescalate infected tomato plants to some threshold with the use of control. This implies that a combination of the use of insecticide spray and roguing tomato plant control reduced tomato yellow leaf curl virus disease to some threshold.

Figure 4 depicted that the number of susceptible and infected whitefly vector population is increased in the absence of control. However, according to 4 (a) the susceptible whitefly vector increased at decreasing rate relative to uncontrolled once. As per 4 (b) the infected whitefly increase slightly but becomes stable soon in the case of control. This may be attributed to roguing infected tomato plants reduces the amount of secondary spread within a field and protective netting is reduced the number of invading whiteflies into the covered net.

**(a)** Healthy tomato plant.

**(b)** Infected tomato plant.
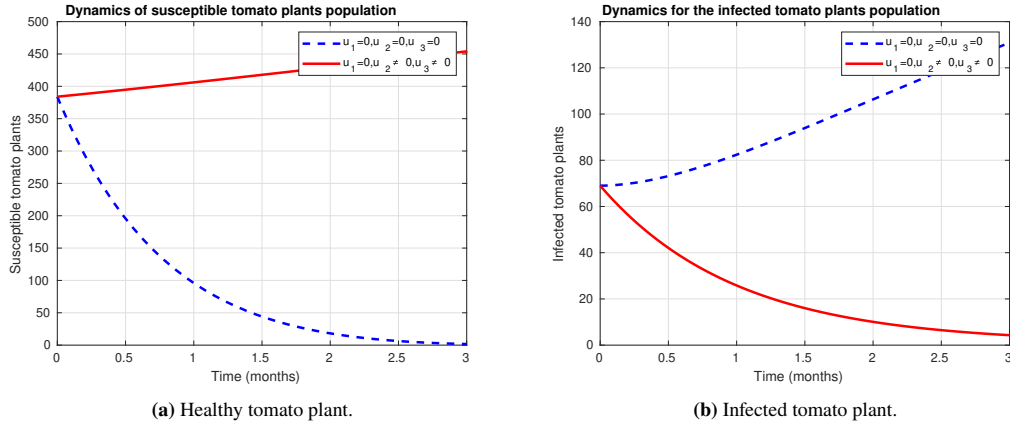
**FIGURE 3** The impact of roguing diseased tomato plants and protective netting on tomato plant population.



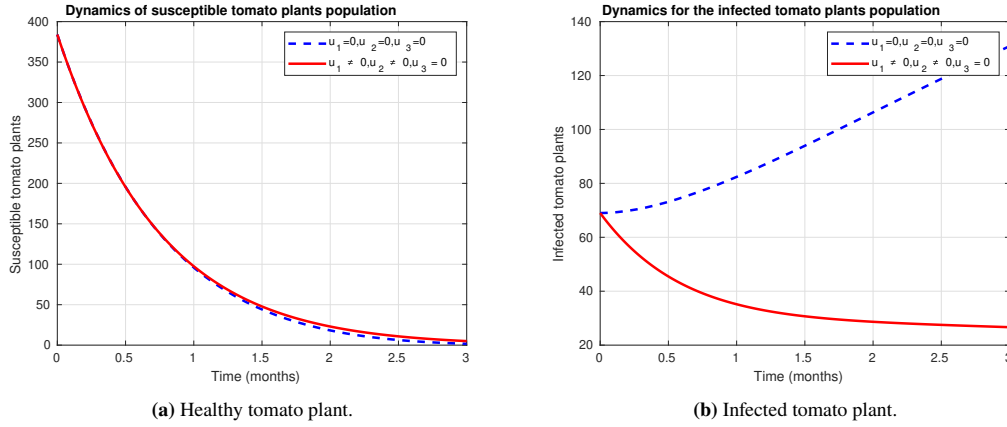**(a)** Healthy tomato plant.

**(b)** Infected tomato plant.

**FIGURE 4** The impact of roguing diseased tomato plants and protective netting.

## STRATEGY III: COMBINATION OF USE OF INSECTICIDES SPRAY AND ROGUING DISEASED TOMATO PLANTS

The objective function $J$ is optimized using insecticides spray control $u_1$ and roguing diseased tomato plants control $u_2$ while protective netting control $u_3$ is set to zero.

The results in Figure 5 (a) represented that without and with control the healthy tomato plant population decreased with time, however relative to infected tomato plant the number of healthy tomato plant increases in case of control. Figure 5 (b) is revealed that infected tomato plant increased without control gradually but decreased with time provided that control is utilized.

According to Figure 6 , the susceptible and infected whitefly vector population escalate without control whereas in the case of control whitefly transmitted viruses also increased but at a decreasing rate. This indicates to control whitefly transmitted viruses using insecticides spray and roguing diseased tomato plant is hard [33].

**(a)** Healthy tomato plant.

**(b)** Infected tomato plant.

**FIGURE 5** The impact of insecticides spray and roguing diseased tomato plant.



**(a)** Susceptible whitefly vector.

**(b)** Infective whitefly vector.
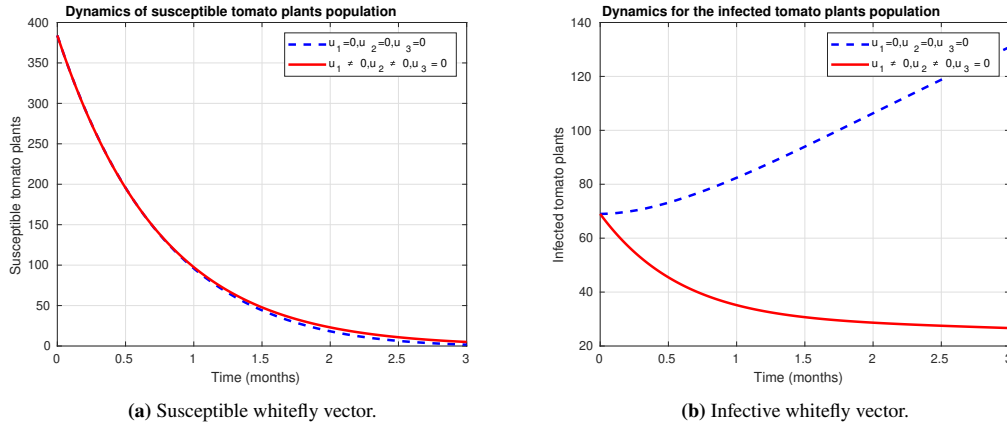
**FIGURE 6** The impact of insecticides spray and roguing diseased whitefly vector.

## STRATEGY IV: COMBINATION OF USE OF INSECTICIDES SPRAY, ROGUING DISEASED TOMATO PLANTS AND PROTECTIVE NETTING

All the three controls $u_1$, $u_2$ and $u_3$ are used to optimize the objective function $J$.

It is observed in Figure 7 (a) that the control strategies resulted in an escalation of a healthy tomato plant population in the presence of control strategies but drop-off to zero without control. Figure 7 (b) portrayed a significant decrease in the numbers of infected tomato plants in the case of control but dramatically increase in the absence of control. This implies that a combination of insecticides spray, rouging diseased tomato plan and protective netting are declined TYLCV disease.

Figure 8 (a) and 8 (b) explained that with the application of control the susceptible and infected whitefly vector decreased significantly in the case of control whereas increased in the case of uncontrolled.

**(a)** Healthy tomato plant.

**(b)** Infected tomato plant.
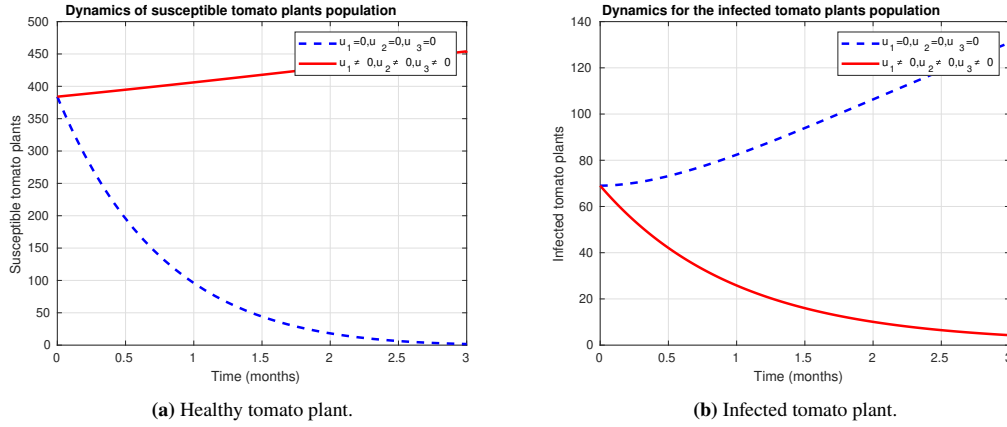
**FIGURE 7** The impact of insecticides spray, roguing diseased tomato plant and protective netting.



**(a)** Susceptible whitefly vector.

**(b)** Infective whitefly vector.
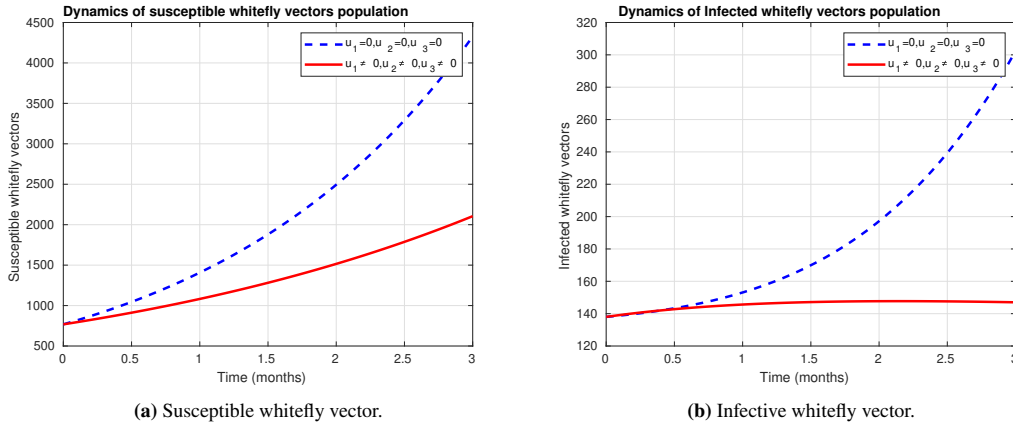
**FIGURE 8** The impact of insecticides spray, roguing diseased tomato plant and protective netting.

## 6 | CONCLUSION

In this paper, the deterministic model for the transmission of TYLCV disease that incorporates the use of insecticide spray, roguing of the diseased tomato plant and protective netting, and perform optimal control analysis of the model. The basic reproduction number was calculated and the existence and local and global stability of equilibria were analyzed. The model exhibits transcritical bifurcation at $\mathcal{R}_0 = 1$ which indicated that for $\mathcal{R}_0 < 1$, the disease-free equilibrium is stable whereas endemic equilibrium is unstable and for $\mathcal{R}_0 > 1$, the endemic equilibrium is stable whereas disease-free equilibrium is unstable. The sensitivity analysis of the basic reproduction number showed that TYLCV disease has a positive relationship with the rate of tomatoes fruit are harvested or removed, rate of inoculation of health tomato plant $\beta_v$, and rate of virus acquisition by susceptible vectors. Thus, these parameters are those that should be targeted most by policymakers to combat against the TYLCV disease. Hence, the time-varying optimal control analysis of the model was made using Pontryagin's maximum principle. From the simulation results, it can be concluded that all the combined efforts of two of three strategies of three namely insecticide spray, roguing diseased tomato plants, and protective netting can significantly reduce the disease except the combination of the use of insecticide spray and roguing infected tomato plants. Relatively the other, the use of roguing diseased tomato plants and protective netting and the use of insecticides spray, roguing diseased tomato plants and protective netting are better decreased the disease. Moreover, the use of roguing diseased tomato plants and protective netting has a similar effect as the use of insecticides

spray, roguing diseased tomato plants, and protective netting. As resources are scarce, we propose that policy-makers should adopt the combination of the use of roguing diseased tomato plants and protective netting as a strategy.

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## Conflict of interest

None of the authors have any proprietary interests or conflicts of interest related to this submission.

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