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Research Article

Ecoepidemiological Model and Optimal Control Analysis of Tomato Yellow Leaf Curl Virus Disease in Tomato Plant

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The purpose of this study is to analyze the impact of control strategies, namely, insecticide spray, roguing of a diseased tomato plant, and protective netting to protect tomato plant from tomato yellow leaf curl virus disease (TYLCVD). For this, we formulate and analyze a simple deterministic model for the transmission dynamics of TYLCVD that incorporates these control strategies. We initially take into account the constant control case, we calculate the basic reproduction number, and we investigate the existence and stability of the disease-free and endemic equilibria. We use the Kamgang-Sallet stability to ensure that the disease-free equilibrium point is globally asymptotically stable when the reproduction number $\mathscr{R}_0$ is less than one. This indicates that TYLCVD dies out independent of the initial size of the tomato population. For $\mathscr{R}_0 < 1$, the disease-free equilibrium becomes unstable, and the endemic equilibrium is globally asymptotically stable. This indicates that TYLCVD propagates. In the nonconstant control case, we use Pontryagin's maximum principle to derive the necessary conditions for the optimal control of the disease. Our findings show that all the combined efforts of two out of three strategies can significantly reduce the power of infectivity of the disease except the combination of the use of insecticide spray and rouging infected tomato plants. Besides our numerical simulations show, the implementation of the combination of roguing diseased plants and protective netting has a similar effect in minimizing or eliminating TYLCV as the use of all strategies. Hence, as resources are always in scarce, we recommend policymakers to adapt the combination of the use of roguing diseased tomato plants and protective netting to eradicate the disease.

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, 2]. 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 [3]. The vector damages tomato plant directly by feeding on phloem, excreting honeydew, and causing phytotoxic disorders [4]. With increased populations, they secrete large quantities of honeydew. The vector favors the growth of sooty mold on leaf surfaces and reduces the photosynthetic efficiency of the plants [5]. The honeydew also contaminates the marketable part of the plant, reducing its market value. Additionally, in severe infestations, the leaves turn yellow and are dropped off from its leaves [3].

The first report on TYLCV infection in tomato was from Israel and other countries in the Middle East in the 1930s. And since then, the virus has further emerged [5]. In Africa, The TYLC disease was first seen in Sudan in 1965; but the causal agent was identified as TYLCV in 1997 [6, 7]. In Ethiopia, at Melka-Werner, about 90% of tomato plant showed leaf curl virus symptoms with reduced size, suspected to be caused by TYLCV [8]. However, only recently occurrence of *Begomovirus* associated with the TYLC disease was reported for the first time from Melkasa [9].

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. tabaci* whitefly can accumulate 600 million TYLCV genomes [3]. From the site of inoculation, viral DNA is first mainly transported to strongly sink organs, such as root and shoot apices, flowers, and fruit, and then moves to leaves [10]. Similarly to other plant viruses, TYLCV moves in the existing host transport routes such as plasmodesmata and phloem, along with carbohydrates [11]. About 11-13 days after inoculation, maximum amount of viral DNA and capsid or coat protein (CP) accumulates in the youngest tissues of shoots and roots. Four to seven days later, unique symptoms appear for the first time [10]. As the systemic infection proceeds in the growing plant, the virus is accumulated in the strongest sink tissues. The level of viral double-stranded DNA (dsDNA) and newly generated single-stranded DNA (ssDNA), as well as CP, further increases in young organs up to several weeks postinoculation. The infection then gradually spreads to older organs of the host and remains strictly confined to the vascular system [11].

Mathematical modeling has been playing an important role in better understanding the epidemiological patterns. It provides deeper insight into the underlying mechanisms for the spread of emerging and reemerging infectious diseases and the suggested effective control strategies [12]. Holt et al. [13] on their paper entitled "An epidemiological model incorporating vector population dynamics applied to African Cassava Mosaic Virus Disease" illustrated the African cassava mosaic virus occurrence in cassava which is transmitted by a cassava-specific whitefly strain, which was later seen in 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 the infected cuttings transmit disease, whereas roguing would be more important in a largely vector-driven epidemic.

In a later paper, Holt et al. [14] developed an epidemiological model, a susceptible-exposed-infective- (SEI-) type epidemic model for the host plant and a susceptible-infective- (SI-) type for the insect vector population. It represents the incidence of TYLCV in tomato plant, mainly relying on the immigration of vectors from alternative hosts which act as a reservoir of both the virus and vector. This is because, unlike the cassava, the tomato was only an occasional host for this whitefly and is spilledover from other perennials and weedy plants driven by 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 the best method to control the disease is?" Because most of the vector lifespan occurs on other hosts. The authors adapted a model framework [15] to explain the transmission process of the disease. Since the tomato crop was a sink for whiteflies and TLCV, interventions that reduce vector immigration and their survival were predicted to be most effective.

Alemneh et al. [16] proposed and examined an ecoepidemiological deterministic model for the transmission dynamics of maize streak virus (MSV) disease in the maize plant. Their model depicted that increased parameters namely the infection and predation rates made an increment of the basic reproduction number that leads to the increment of the number of infected maize population. Hence, the authors suggested that to intervene in MSV disease, endeavors should be exerted to reduce the contact of infected maize and susceptible leafhopper. In addition to this, 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 reach of the leafhopper or uprooting it. Moreover, infected maize should be burnt from the field.

Nowadays, many sophisticated metaheuristics have been initiated to solve the most complex problems by transforming them into problems of optimization. For instance, Farman et al. [17] used an evolutionary Pade approximation (EPA) scheme for the treatment of nonlinear epidemiological hepatitis-B model, instead of numerical methods. Thus, they showed that as compared to a nonstandard finite difference discretization (NSFD) numerical scheme, EPA scheme is more reliable and significant in approximations of state variables that are highly accurate to the governing equations. Naik et al. [18] proposed and examined a nonlinear fractional order SEIR model for the transmission of HIV epidemics. For such nonlinear fractional-order model, they inspired the application of the homotopy analysis method (HAM) to solve highly nonlinear fractional-order problems, describing biological phenomena. They also gave a research direction to use another approximate solution technique such as fractional-order derivatives with Alangana-Gomez, Atangana-Baleanu, Caputo-Fabrizio, and fractal-fractional, to get a nonlinear fractional-order SEIR epidemic model.

Optimal control theory has found wide-range of the applications in biological and ecological problems [19]. Particularly, there have been various studies of epidemiological models where optimal control methods have been applied. Berhe et al. [20] formulated a deterministic model to study the effects of implementing continuous controls on the dysentery epidemic model. It 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 preventive strategy. As a result, they found that the disease can probably be eradicated by implementing continuous controls in a short period of time. However, utilizing a combination of sanitation of the environment and educational campaign was found to be the most cost-effective control strategy. Okosun and Makinde [21] 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 educational 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 are also obtained if educational campaigns as preventive measure and treatment of infected children were used. However, as resources are scarced, the authors proposed that policymakers likely should focus on optimal provision of prevention and treatment being cost-effective. Bokil et al. [22] investigated and analyzed optimal control of a vectored plant disease model for crops with continuous replanting. They considered two plant-vector-virus models which take into account frequency replanting and abundance replanting strategies to study the 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 dynamism. 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 different replanting practices. Hugo et al. [23] studied optimal control and cost-effectiveness analysis of the TYLCV disease epidemic model. Their model was an extended work of [14]. The authors incorporated the time-dependent control to the tomato plants and vector populations in analyzing the cost-effectiveness of the control strategies. Thus, they suggested that the use of the combined protective netting and removal of the infected plant is the cost-effective optimal control strategy. It was sufficient to combat the epidemic of the tomato disease with limited resources. A preprint of our paper has previously been published [24].

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, R_0. To wind up, this paper is focused on optimal control strategies analysis of the tomato yellow leaf curl virus disease model, which is adopted from the African cassava mosaic virus disease's model [13] 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 the stability analysis of the model. Section 4 is contained by numerical simulations. Our conclusions are given in Section 5.

2. Mathematical Model

The model that is considered here is a small modification of the model for plant-virus transmission considered in [13]. It is a standard model of SEI type for tomato plants and SI for whiteflies *Bemisia tabaci* B-type insect vector.

The model subdivides the total tomato plant population into the following subclasses: healthy or susceptible (S_p), latently infected (E_p) and infective (I_p), K is the carrying capacity of the tomato 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 subdivided in 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 [25]. Thus, it is assumed to be negligible, i.e., no latent subclass was 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 $rS_p(1 - S_p + E_p + I_p/K)$ where r is the rate replanting healthy tomato. This is because, the model assumed that healthy tomatoes responded proportionately inverse to the extended intraspecific pressure for the healthy, exposed, and infected tomato plants. Replanting tomato is restricted by maximum tomato plant availability and the harvesting of healthy, exposed, and infected tomato plant reflects the continual turnover of the tomato plant population. The tomato fruit is either harvested or removed at a rate of g or move to the exposed stage by inoculating through contact with infective whiteflies at a rate of β_p. Moreover, all stages of tomatoes 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 $1/a$. Here, it is assumed that the infective tomatoes remain infected forever. Loss of tomatoes due to nature- and disease-related reduction is rated as b.

The population of whiteflies is assumed to be generated at a rate of $c(S_v + I_v)(1 - S_v + I_v/m(S_p + I_p))$, where c is the vector birth rate and m is the rate of vector maximum abundance. Besides, we assumed that the vector does not immigrate to other hosts in the case of low tomato plant abundance. The vector dies either from natural causes at a rate of e or propagates to the infective class by acquiring tomato yellow leaf curl virus through contacts with infected tomatoes at a rate of $\beta_v I_p(t) S_v(t)$, where β_v is the rate of virus acquisition by a susceptible vector (whitefly) during a single visit to an infectious tomato plant. The protections target mainly the following: (i) insecticide on tomato plant population: minimizes the inoculation efficiency of the vector, i.e., it reduces to β_p; (ii) roguing diseased tomato plants: good practice for reducing the source of primary infection, i.e., reduced to g; (iii) protective netting, prevent the entry of whitefly vectors, *B. tabaci*, into tomato plots, i.e., reduced to β_p and β_v. This is because the only way of controlling TYLCV is by controlling the vector [5]. Thus, the efforts made on these three intervention mechanisms enable to control the tomato infections due to TYLCV.

Suppose that the control function like $u_1(t)$ represents insecticide spray with efficacy q, $u_2(t)$ represents roguing diseased tomato plants, and $u_3(t)$ represents protective netting at any time t. Besides, controlling u_1, u_2, and 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

[5]. 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

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

3. Stability Analysis of Free Disease Equilibrium Point

In this section, we study the positivity and boundedness of the solution and the stability of the disease-free equilibrium of model system 1. As the variables in model system 1 represent tomato and whitefly population densities, positivity indicates survival of the population, and boundedness may be interpreted as a natural restriction to growth as a result of limited resources. The stability of the disease-free equilibrium point tells us TYLCV disease dies out from the population.

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 nonnegative initial data will remain nonnegative for all the time as is presented in [26].

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 Ω is positively invariant and all solutions starting in Ω approach, enter, or stay in Ω.*

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

$$\frac{dN_p}{dt} = rS_p\left(1-\frac{S_p+E_p+I_p}{K}\right)-g(S_p+E_p+I_p)-bI_p. \tag{3}$$

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

$$\frac{dN_p}{dt}\leq rN_p\left(1-\frac{N_p}{K}\right)-gN_p,\quad t\geq 0. \tag{4}$$

Thus,

$$N_p\leq K\left(\frac{r-g}{r}\right),\quad t\geq 0. \tag{5}$$

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

$$\frac{dN_v(t)}{dt} = c(S_v+I_v)\left(1-\frac{S_v+I_v}{m(S_p+I_p)}\right)-e(S_v+I_v). \tag{6}$$

Since $N_v = S_v+I_v$ and $K>S_p+I_p$, then Equation (6) can be written as

$$\frac{dN_v(t)}{dt}\geq cN_p\left(1-\frac{N_v}{mK}\right)-eN_v, t\geq 0. \tag{7}$$

If we factorize Equation (7), then we can obtain

$$\frac{dN_v(t)}{dt}\geq (c-e)N_v\left(1-\frac{N_v}{mK((c-e)/c)}\right)-eN_v, t\geq 0. \tag{8}$$

Thus,

$$N_v\leq mK\left(\frac{c-e}{c}\right), t\geq 0. \tag{9}$$

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

$$\begin{aligned}
\Omega_p &= \left\{(S_p,E_p,I_p)\in\mathcal{R}_+^3 : S_p+E_p+I_p\leq K\left(1-\frac{g}{r}\right)\right\},\\
\Omega_v &= \left\{(S_v,I_v)\in\mathcal{R}_+^2 : S_v+I_v\leq mK\left(1-\frac{e}{c}\right)\right\}.
\end{aligned} \tag{10}$$

This implies that all solutions of tomato plants population are only confined in the feasible region Ω_p, and all solutions of the whiteflies population are confined in Ω_v.

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

$$\Omega=\left\{(S_p,E_p,I_p,S_v,I_v)\in R_+^5 : N_p\leq K\left(\frac{r-g}{r}\right); N_v\leq mK\left(\frac{c-e}{c}\right)\right\}. \tag{11}$$

Thus, Ω is positively invariant. This means that solutions of the model system with positive initial data remain positive for all time $t\geq 0$ and are bound in the region Ω. 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 and Global Stability. The disease-free equilibrium of the tomato yellow leaf curl virus disease model (1) exists and is given by

$$E_0 = \left(K\left(1-\frac{g}{r}\right), 0, 0, mK\left(1-\frac{qu_1+e}{c}\right)\left(1-\frac{g}{r}\right), 0\right). \quad (12)$$

The basic reproduction number, $\mathscr{R}_0$, is calculated by using the next-generation matrix [27]. To obtain $\mathscr{R}_0$ for model (1), let the vector of the disease states

$$x = \left(E_p, I_p, I_v\right)^T. \quad (13)$$

Then, the model (1) can be written as

$$\frac{\mathrm{dx}}{\mathrm{dt}} = F(x) - V(x). \quad (14)$$

where

$$F(x) = \begin{pmatrix} (1-u_3)\beta_p I_v S_p \\ 0 \\ \beta_v I_p S_v \end{pmatrix}, \qquad V(x) = \begin{pmatrix} (a+g)E_p \\ (g+b+u_2)I_p - aE_p \\ (qu_1+e)I_v \end{pmatrix}. \quad (15)$$

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

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & \beta_p K(1-u_3)\left(1-\frac{g}{r}\right) \\ 0 & 0 & 0 \\ 0 & \beta_v mK\left(1-\frac{e+qu_1}{c}\right)\left(1-\frac{g}{r}\right) & 0 \end{pmatrix},$$
$$V = \begin{pmatrix} a+g & 0 & 0 \\ -a & g+b+u_2 & 0 \\ 0 & 0 & qu_1+e \end{pmatrix}. \quad (16)$$

Thus

$$\mathbf{V}^{-1} = \begin{pmatrix} \frac{1}{a+g} & 0 & 0 \\ \frac{a}{(a+g)(b+g+u_2)} & \frac{1}{b+g+u_2} & 0 \\ 0 & 0 & \frac{1}{e+qu_1} \end{pmatrix}, \quad (17)$$

So that

$$\mathbf{FV}^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_p K(1-u_3)(r-g)}{r(e+qu_1)} \\ 0 & 0 & 0 \\ \frac{\beta_v amK(c-qu_1-e)(r-g)}{cr(a+g)(b+g+u_2)} & \frac{\beta_v mK(r-g)(c-qu_1-e)}{cr(b+g+u_2)} & 0 \end{pmatrix}. \quad (18)$$

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

$$\mathscr{R}_0 = \sqrt{\frac{\beta_p\beta_v amK^2(r-g)^2(1-u_3)(c-qu_1-e)}{cr^2(a+g)(g+b+u_2)(qu_1+e)}}. \quad (19)$$

Theorem 2 below follows from Theorem 2 of [28].

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

The epidemiological implication of Theorem 2 is that the transmission of TYLCV can be controlled by having $\mathscr{R}_0 < 1$ if the initial total numbers of the subpopulations involved in Equation (1) are in the basin of attraction of $\mathscr{E}_0$. 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 $\mathscr{R}_0 < 1$. This is what we are checking next.

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

Proof. To prove the theorem, we use the Kamgang-Sallet stability theorem in [29]. 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

$$\begin{aligned} \dot{X}_1 &= A_1(X)(X_1 - X_1^*) + A_{12}(X)X_2, \\ \dot{X}_2 &= A_2(X)X_2, \end{aligned} \tag{20}$$

where

$$\begin{aligned} X_1^* &= \left(K\left(1-\frac{g}{r}\right), mK\left(1-\frac{qu_1+e}{c}\right)\left(1-\frac{g}{r}\right)\right), \\ A_1(X) &= \begin{pmatrix} -(r-g) & 0 \\ -m\left(1-\frac{qu_1+e}{c}\right)^2 & -(c-qu_1-e) \end{pmatrix}, \\ A_{12}(X) &= \begin{pmatrix} -\frac{rS_p}{K} & -\frac{rS_p}{K} & -(1-u_3)\beta_p S_p \\ 0 & -\beta_v S_v + \frac{c}{m}\left(\frac{S_v+I_v}{S_p+I_p}\right)^2 & c\left(1-\frac{2S_v}{m(S_p+I_p)}\right) - \frac{2I_v}{m(S_p+I_p)} \end{pmatrix}, \\ A_2(X) &= \begin{pmatrix} -(a+g) & 0 & (1-u_3)\beta_p S_p \\ a & -(g+b+u_2) & 0 \\ 0 & \beta_v S_v & -(qu_1+e) \end{pmatrix}. \end{aligned} \tag{21}$$

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

(1) The system (1) is a dynamical system on Ω. 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

$$\mathcal{M} = A_2(X) \colon X \in \Omega. \tag{22}$$

More precisely,

$$A_2(X) = \begin{pmatrix} -(a+g) & 0 & (1-u_3)\beta_p K\left(1-\frac{g}{r}\right) \\ a & -(g+b+u_2) & 0 \\ 0 & \beta_v mK\left(1-\frac{g}{r}\right)\left(1-\frac{qu_1+e}{c}\right) & -(qu_1+e) \end{pmatrix} \tag{23}$$

is an upper bound of M.

(5) For $R_0 < 1$ in Equation (19),

$$\alpha\left(\bar{A}_2(X)\right) = \max\left\{\mathrm{Re}\,(\lambda) \colon \lambda \text{ is an eigenvalue of } \bar{A}_2\right\} \le 0. \tag{24}$$

Thus, all eigenvalues of A are negative for $\mathcal{R}_0 < 1$ in Equation (19). □

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

$$\mathcal{E}^* = \left(S_p^*, E_p^*, I_p^*, S_v^*, I_v^*\right) \tag{25}$$

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

$$\begin{aligned} S_p^{**} &= \frac{K}{2}\left(1-\frac{g}{r}\right) - \frac{g+b+u_2+a}{2a}I_p + \frac{M}{2}, \\ E_p^{**} &= \frac{g+b+u_2}{a}I_p, \\ S_v^{**} &= \frac{m(qu_1+e)(c-qu_1-e)}{c\left(\beta_v I_p + qu_1 + e\right)}\left[\frac{K}{2}\left(1-\frac{g}{r}\right) + \frac{a-g-b-u_2}{2a}I_p + \frac{M}{2}\right], \\ I_v^{**} &= \frac{m(c-qu_1-e)\beta_v}{c\left(\beta_v I_p + qu_1 + e\right)}\left[\frac{K}{2}\left(1-\frac{g}{r}\right) + \frac{a-g-b-u_2}{2a}I_p + \frac{M}{2}\right]I_p, \end{aligned} \tag{26}$$

where

$$M = \sqrt{\left[K\left(1-\frac{g}{r}\right) - \frac{g+b+u_2}{a}I_p\right]^2 - \frac{4K(a+g)(g+b+u_2)}{ar}I_p}. \tag{27}$$

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

$$f\left(I_p\right) = c_4 I_p^4 + c_3 I_p^3 + c_2 I_p^2 + c_1 I_p + c_0, \tag{28}$$

where

$$c_4 = B_3^2 - A_1 A_4,\ c_3 = 2B_2 B_3 - A_1 A_4 - A_2 A_3,$$

$$c_2 = B_2^2 + 2B_1B_2 - A_1A_2 - K\left(1-\frac{g}{r}\right)\left[A_4 + A_2K\left(1-\frac{g}{r}\right)\right],$$
$$c_1 = 2B_1B_2 - (A_1+A_2)\left[K\left(1-\frac{g}{r}\right)\right]^2, c_0 = B_1^2 - 4\left[K\left(1-\frac{g}{r}\right)\right]^4, A_1 = -\frac{2(g+b+u_2)}{a}K\left(1-\frac{g}{r}\right),$$
$$A_2 = \left(\frac{g+b+u_2}{a}\right)^2, A_3 = -\frac{K}{a}\left[\frac{2(g+b+u_2+a)}{a}\left(1-\frac{g}{r}\right) + \frac{(a+g)(g+b+u_2)}{r}\right], A_4 = \frac{g+b+u_2+a}{a},$$
$$B_1 = \frac{4}{R_0^2}K\left(1-\frac{g}{r}\right)\left[K\left(1-\frac{g}{r}\right)-2\right], B_3 = \frac{a^2-(g+b+u_2)^2-(g+b+u_2+a)a}{a^2},$$
$$B_2 = \frac{4}{R_0^2}K\left(1-\frac{g}{r}\right)\left[K\left(1-\frac{g}{r}\right)\frac{\beta_v}{qu_1+e} + \frac{2(g+b+u_2)}{a} + \frac{2(g+b+u_2+a)}{a}\right] + \frac{(a+g)(g+b+u_2)K}{ar}. \quad (29)$$

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

The stability of the EE is guaranteed by using the center manifold theorem in [30].

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 dynamic 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 most to output variability [31].

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

Definition 5. The normalized sensitivity index of a variable, $\mathscr{R}_0$, that depends differentiably on a parameter, p, is defined as

$$\sigma_p^{R_0} = \frac{\partial \mathscr{R}_0}{\partial p}\frac{p}{\mathscr{R}_0}. \quad (30)$$

3.3.1. Sensitivity Indices of Basic Reproductive Number. Here, the sensitivity of $\mathscr{R}_0$ to every parameter of the model is derived. Hence, the sensitivity index of $\mathscr{R}_0$ with respect to K and is equal to 1. It is equal to 0.5 with respect to m, β_p, and β_v. The rest of the parameters are the following:

$$\sigma_r^{R_0} = -\frac{g}{g-r}, \sigma_e^{R_0} = \frac{ce}{2(e+qu_1)(e-c+qu_1)}, \sigma_g^{R_0} = \frac{g(2(ab+2gr)+(a+b)(g+r)}{2(a+g)(b+g)(g-r)},$$
$$\sigma_a^{R_0} = \frac{g}{2(a+g)}, \sigma_c^{R_0} = -\frac{e+qu_1}{2(e-c+qu_1)}, \sigma_{u_1}^{R_0} = \frac{cqu_1}{2(e+qu_1)(e-c+qu_1)},$$
$$\sigma_b^{R_0} = -\frac{b}{2(b+g)}, \sigma_{u_2}^{R_0} = \frac{u_2}{2(u_2-1)}, \sigma_{u_3}^{R_0} = \frac{u_3}{2(u_3-1)}. \quad (31)$$

Since most of the expressions for sensitivity indices are complex, the sensitivity indices are evaluated at the baseline parameter values given in Table 1. The sensitivity index of R_0 with respect to r, for example, is as follows:

$$\frac{r}{\mathscr{R}_0}\frac{\partial \mathscr{R}_0}{\partial r} = -\frac{g}{g-r} = -\frac{0.0121}{0.0121-0.01} = -5.7619. \quad (32)$$

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

The parameters are arranged from most sensitive to least. The most sensitive parameters are the rate of replanting of healthy tomato, the rate at which tomato fruits are either harvested or removed, the carrying capacity of tomato farm, the rate of vectors' maximum abundance, the rate of inoculation of the healthy tomato plant, and the rate of virus acquisition by susceptible vectors. The least sensitive parameter is the loss rate of tomatoes due to infection. The parameters that reduce 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 2. The parameters with the most important that have positive indices are β_p, β_v, r, K, m, c, and a, and those with negative indices are g, e, and b. The results show that when the inoculation of the healthy tomato plant rate and virus acquisition rate by susceptible vectors increases, the basic reproduction number increases as a result of TYLCV disease propagated since the infected plants and infective vectors are both infectious. When the rate of replanting of a 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 spread of the disease. The propagation rate from the exposed class to the infectious class increases the number of infectious tomato plants and vectors. These contribute to the disease spread. On the other hand, tomato fruit harvested or removed and death rate of vectors are reduced to the size of tomato plant population and whitefly vector population.

4. Analysis of Optimal Control

Let $x(t)$ represent 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 (roguing, avoidance, crop residue disposal) reduce the amount of secondary spread within a tomatoes' field. Virus-resistant cultivars reduce the loosed ones, due to infections by

Table 1: Model parameters and values used in the simulation.

Parameters	Standard values	Reference sources
r	0.121 day^{-1}	[14]
K	1000	Assumed
e	0.0286 day^{-1}	Estimated
c	0.50 day^{-1}	[14]
g	0.01 day^{-1}	Estimated
a	0.075 day^{-1}	Estimated
m	1500 plant^{-1}	Assumed
b	0.003 day^{-1}	[14]
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
β_p	0.01 vector^{-1}day^{-1}	[14]
β_v	0.0003 plant^{-1}day^{-1}	[14]

TYLCV [32]. 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 the infectious tomato plants and whiteflies population; p_1, p_2, and p_3 represent the weight attached on the cost control methods, and then the cost rate at which TYLCV disease is controlled at any time t can be given by the following:

$$f(x,u,t) = c_1 I_p(t) + c_2 I_v(t) + \frac{1}{2}\left[p_1 q u_1^2(t) + p_2 u_2^2(t) + p_3 u_3^2(t)\right], \tag{33}$$

where $x = (I_p, I_v)$, $u = (u_1, u_2, u_3)$. Since the implementation of any intervention has decreased costs, it is customary to take a nonlinear cost function. Hence, the simplest nonlinear function (the quadratic) in modeling the cost of the interventions is used.

Therefore, an optimal control $u^* = (u_1^*, u_2^*, u_3^*)$ searches the following:

$$J(u^*) = \min\{J(u) : u \in \mathcal{U}\}, \tag{34}$$

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

To sum up, the optimal control problem has the following form:

$$\begin{aligned} &\min_u J(u_1,u_2,u_3) = \int_0^T f(x,\mathbf{u},t)\mathrm{dt}, \\ &\text{subject to } \frac{\mathrm{dx}}{\mathrm{dt}} = F(x,\mathbf{u},t), x(0) = x_0, x \ge 0, \\ &0 \le u_i(t) \le 1 \forall t \in [0,T], i = 1,2,3. \end{aligned} \tag{35}$$

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 following form:

$$H(x,\mathbf{u},t) = f(x,\mathbf{u},t) + \sum_{i=1}^{5} \lambda_i(t) F_i(x,\mathbf{u},t), \tag{36}$$

where $F_i(x,\mathbf{u},t)$ 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:

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

$$\begin{aligned} \frac{d\lambda_1}{dt} &= \lambda_1\left(rS_p\left(1 - \frac{S_p + E_p + I_p}{K}\right) - (1 - u_3(t))\beta_p I_v S_p - gS_p\right), \\ \frac{d\lambda_2}{dt} &= \lambda_2\left((1 - u_3(t))\beta_p I_v S_p - (a+g)E_p\right), \\ \frac{d\lambda_3}{dt} &= \lambda_3\left(aE_p - (g + b + u_2)I_p\right), \\ \frac{d\lambda_4}{dt} &= \lambda_4\left(c(S_v + I_v)\left(1 - \frac{S_v + I_v}{m(S_p + I_p)}\right) - \beta_v I_p S_v - qu_1(t)S_v - eS_v\right), \\ \frac{d\lambda_5}{dt} &= \lambda_5\left(\beta_v I_p S_v - qu_1(t)I_v - eI_v\right), \end{aligned} \tag{37}$$

with transversality condition

$$\lambda_i(T) = 0 \text{ for } i = 1,2,3,4,5. \tag{38}$$

By using Pontryagin's maximum principle and the existence result for the optimal control (Makinde et al. [33]), we arrive at the following theorem:

Table 2: Sensitivity indices of $\mathscr{R}_0$.

Parameters	Parameter description	Sensitivity index
r	Rate of replanting of healthy tomato	+ve
g	Rates at tomato fruits are harvested or removed	-ve
K	Carrying capacity of tomato farm	+ve
u_1	Optimal control due to insecticide 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
β_p	Rate of inoculation of healthy tomato plant	+ve
β_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 optimal control, u_1^*, u_2^*, u_3^*, that minimizes $J(u_1, u_2, u_3)$ over Ω is expressed as

$$\begin{aligned} u_1^* &= \min\left(1, \max\left(0, \frac{(I_v\lambda_5 + \lambda_4 S_v)q}{p_1 q}\right)\right), \\ u_2^* &= \min\left(1, \max\left(0, \frac{\lambda_3 I_p}{p_2}\right)\right), \\ u_3^* &= \min\left(1, \max\left(0, \frac{(\lambda_2 - \lambda_1)\beta_p S_p I_v}{p_3}\right)\right). \end{aligned} \tag{39}$$

Proof (Fleming et al. [34]). Provide the existence of 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 Pontryagin's maximum principle, we have

$$\frac{\mathrm{d}\lambda_i}{\mathrm{dt}} = -\frac{\partial H}{\partial x_i}, \tag{40}$$

where λ_i, $i = 1, 2, 3, 4, 5$. Therefore, the adjoint function with each state variable is calculated as follows:

$$\begin{aligned} H(x, \mathbf{u}, t) = f(x, \mathbf{u}, t) &+ \lambda_1 \frac{dS_p}{\mathrm{dt}} + \lambda_2 \frac{dE_p}{\mathrm{dt}} + \lambda_3 \frac{dI_p}{\mathrm{dt}} \\ &+ \lambda_4 \frac{dS_v}{\mathrm{dt}} + \lambda_5 \frac{dI_v}{\mathrm{dt}}. \end{aligned} \tag{41}$$

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

$$\begin{aligned} H = {} & c_1 I_p(t) + c_2 I_v(t) + \frac{1}{2}\left(p_1 q u_1^2(t) + p_2 u_2^2(t) + p_3 u_3^2(t)\right) \\ & + \lambda_1\left(rS_p\left(1 - \frac{S_p + E_p + I_p}{K}\right) - (1 - u_3(t))\beta_p I_v S_p - gS_p\right) \\ & + \lambda_2\left((1 - u_3(t))\beta_p I_v S_p - (a + g)E_p\right) \\ & + \lambda_3\left(aE_p - (g + b + u_2(t))I_p\right) \\ & + \lambda_4\left(c(S_v + I_v)\left(1 - \frac{S_v + I_v}{m(S_p + I_p)}\right) - \beta_v I_p S_v - qu_1(t)S_v - eS_v\right) \\ & + \lambda_5\left(\beta_v I_p S_v - (qu_1(t) - e)I_v\right). \end{aligned} \tag{42}$$

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

$$\begin{aligned} &\frac{d\lambda_1}{\mathrm{dt}} = -\frac{\partial H}{\partial S_p} = \lambda_1\left(r\left(\frac{E_p + I_p + S_p}{K} - 1\right) - \beta_p I_v(u_3 - 1) + \frac{rS_p}{K}\right) + \beta_p I_v\lambda_2(u_3 - 1) - \lambda_4\frac{c(S_v + I_v)^2}{m(S_p + I_p)^2}, \\ &\frac{d\lambda_2}{\mathrm{dt}} = -\frac{\partial H}{\partial E_p} = \lambda_2(a + g) - \lambda_3 a + \frac{\lambda_1 rS_p}{K}, \\ &\frac{d\lambda_3}{\mathrm{dt}} = -\frac{\partial H}{\partial I_p} = \lambda_3(b + g + u_2) - c_1 - \beta_v\lambda_5 S_v + \frac{\lambda_1 S_p r}{K} + \lambda_4\left(\beta_v S_v - \frac{c(S_v + I_v)^2}{m(S_p + I_p)^2}\right), \\ &\frac{d\lambda_4}{\mathrm{dt}} = -\frac{\partial H}{\partial S_v} = \lambda_4\left(e - c + qu_1 + \beta_v I_p + \frac{2c(S_v + I_v)}{m(S_p + I_p)}\right) - \lambda_5\beta_v I_p, \\ &\frac{d\lambda_5}{\mathrm{dt}} = -\frac{\partial H}{\partial I_v} = \lambda_4\left(\frac{c(I_v + S_v)}{m(Ip + S_p)} - 1\right) + \frac{c(I_v + S_v)}{m(I_p + S_p)} - c_2 + \lambda_5(e + qu_1) + \beta_p S_p(u_3 - 1)(\lambda_2 - \lambda_1). \end{aligned} \tag{43}$$

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

$$\frac{\partial H}{\partial u_i} = 0, \text{ where } i = 1, 2, 3 \Rightarrow \text{the optimality condition}, \tag{44}$$

computed at the optimal control pair and respective corresponding states, which leads to the stated adjoint systems (37) and (38), [35]. By taking into account the optimality conditions,

$$\frac{\partial H}{\partial u_1} = 0, \frac{\partial H}{\partial u_2} = 0, \frac{\partial H}{\partial u_3} = 0, \tag{45}$$

and to determine the values for u_1^*, u_2^*, u_3^*, subject to the constraints, the characterizations (39) can be obtained,

$$\begin{aligned}
&\frac{\partial H}{\partial u_1} = p_1 q u_1 - I_v \lambda_5 q - \lambda_4 S_v q \Rightarrow \left.\frac{\partial H}{\partial u_1}\right|_{u_1 = u_1^*} = p_1 q u_1^* - I_v \lambda_5 q - \lambda_4 S_v q = 0,\\
&u_1^* = \frac{(I_v \lambda_5 + \lambda_4 S_v) q}{p_1 q} \Leftrightarrow u_1^* = \min\left(1, \max\left(0, \frac{(I_v \lambda_5 + \lambda_4 S_v) q}{p_1 q}\right)\right),\\
&\frac{\partial H}{\partial u_2} = p_2 u_2 - I_p \lambda_3 \Rightarrow \left.\frac{\partial H}{\partial u_2}\right|_{u_2 = u_2^*} = p_2 u_2^* - I_p \lambda_3 = 0,\\
&u_2^* = \frac{\lambda_3 I_p}{p_2} \Leftrightarrow u_2^* = \min\left(1, \max\left(0, \frac{\lambda_3 I_p}{p_2}\right)\right),\\
&\frac{\partial H}{\partial u_3} = p_3 u_3 + \beta_p I_v \lambda_1 S_p - \beta_p I_v \lambda_2 S_p \Rightarrow \left.\frac{\partial H}{\partial u_3}\right|_{u_3 = u_3^*} = p_3 u_3^* + \beta_p I_v \lambda_1 S_p - \beta_p I_v \lambda_2 S_p = 0,\\
&u_3^* = \frac{(\lambda_2 - \lambda_1)\beta_p S_p I_v}{p_3} \Leftrightarrow u_3^* = \min\left(1, \max\left(0, \frac{(\lambda_2 - \lambda_1)\beta_p S_p I_v}{p_3}\right)\right).
\end{aligned} \tag{46}$$

□

5. Numerical Simulation

Simulations are carried out to determine the behavior of the system (1). For this purpose, the parameter values listed in Table 1 were used. Most parameter values were taken directly from [14], and the rest were estimated from the data found in [5, 36]. The estimated parameters were calculated as follows. Since the life span of whiteflies is 20-50 days, the death rate was calculated as per the inverse of the average life span, i.e., $e = 1/((20 + 50)/2) = 1/35 = 0.0286\text{day}^{-1}$. Depending on cultivars, tomato fruits could be made ready to harvest at about 75 to 90 days after transplanting [36], and tomato fruits' are harvested or removed at the rate of g, which can be calculated as $g = 1/(75 + 90)/2 = 0.0121\text{day}^{-1}$. Since the exposed period of the tomato plant is 10-14 days [5], the propagation rate from exposed plant to infected plant is calculated as per $a = 1/(10 + 14)/2 = 0.0833\text{day}^{-1}$.

The main objective of this study is to examine the impact of control strategies such as insecticides, cultural practices, and virus-resistant cultivars on the transmission of TYLCV. In order to support the analytical results, graphical representations of various strategies are visualized to determine their impact whenever the controls are applied to the system [14].

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 the management of TYLCV challenging and costly [5]. A combination of management options is necessary to successfully manage the disease and limit the losses of tomato fruits' production [5]. For instance, a combination of cultural and chemical uses is required [32]. Therefore, the following optimal control strategies on the propagation of TYLCV disease in the tomato plant population are numerically investigated below.

(i) Strategy I: combination of the use of insecticide spray and virus-resistant cultivars

(ii) Strategy II: combination of the use of roguing diseased tomato plants and virus-resistant cultivars

(iii) Strategy III: combination of the use of insecticide spray and roguing diseased tomato plants

(iv) Strategy IV: combination of the use of insecticide spray, roguing diseased tomato plants, and virus-resistant cultivars

The optimal control is obtained by solving the optimality system (38), (39), and (43). An iterative scheme is used for solving the optimality system. Thus, the state equations are solved with an assumption for the controls over the simulated time using the fourth-order Runge-Kutta scheme. Because of the transversality conditions (43), the adjoint equations are solved by a backward fourth-order Runge-Kutta scheme using the current iteration solutions of the state equation. Then, the controls are updated by using a convex combination of the previous controls and the value taken from the characterizations (46). Generally, it can be written as $u_{\text{current}} \times (1 - \alpha^k) + u_{\text{previous}} \times \alpha^k$ where k is the current iteration and $0 < \alpha < 1$ [19]. This process is repeated, and the iterations are also stopped if the values of the unknown variables in the previous iterations are very close in contrast to the iterations [19].

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 spraying insecticides is applied five times per season; and the use of roguing diseased tomato plants mainly labors cost [32] and the cost associated with the 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 used as parameter values in Table 1. The initial state variables are chosen as $S_p(0) = 384$, $E_p(0) = 17$, $I_p(0) = 69$, $S_v(0) = 768$, and $I_v(0) = 138$.

5.1.1. Strategy I: Combination of the Use of Insecticide Spray and Virus-Resistant Cultivars. The insecticide spray control u_1 and virus-resistant cultivars control u_3 are used to optimize the objective function J while roguing diseased tomato plant control strategy u_2 is set to zero.

Figure 1 shows that the number of healthy tomato plants is increased gradually while the infected tomato plant population decreased with time in the presence of the control. On the other hand, without control, the number of infected tomatoes escalates while the healthy tomato plant population is reduced. This is logical, because insecticide spray and virus-resistant cultivars 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 is decreased. This is because we assume that the tomato plant is the only host for the insect vector and thus suffers from lack of food.

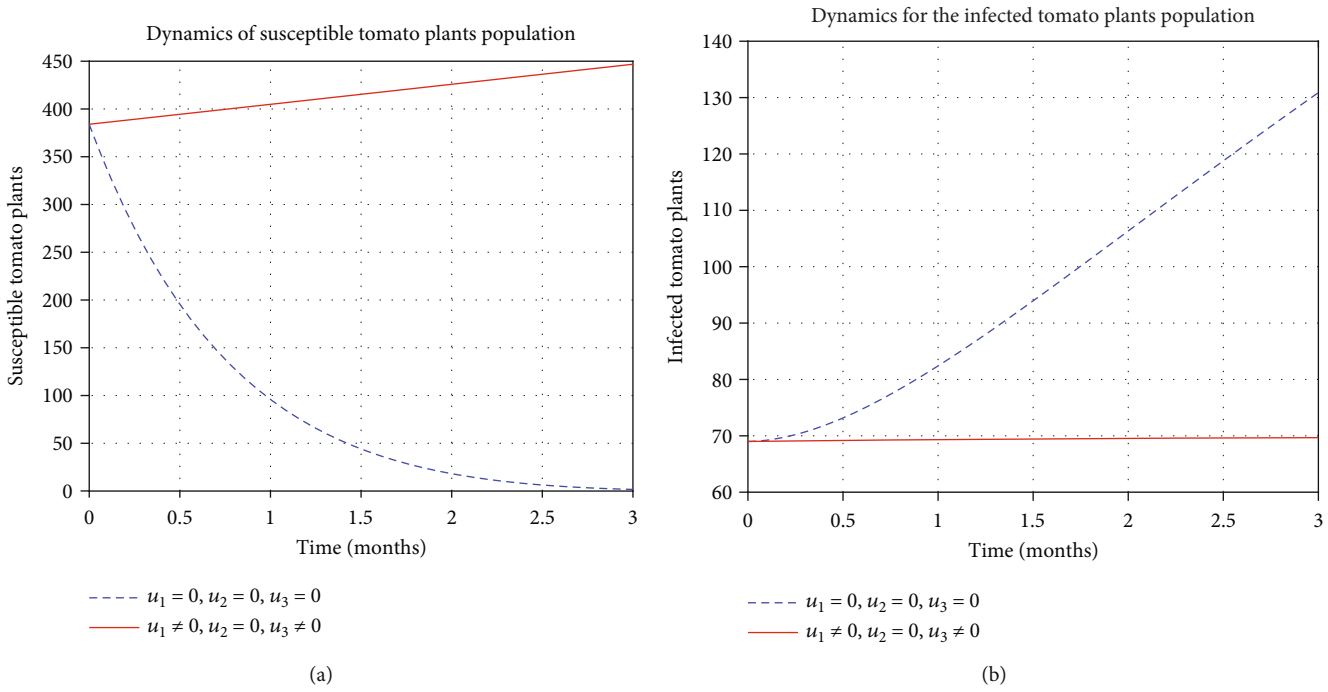

Figure 1: The impact of insecticide spray and virus-resistant cultivars on (a) healthy tomato plant and (b) infected tomato plant.

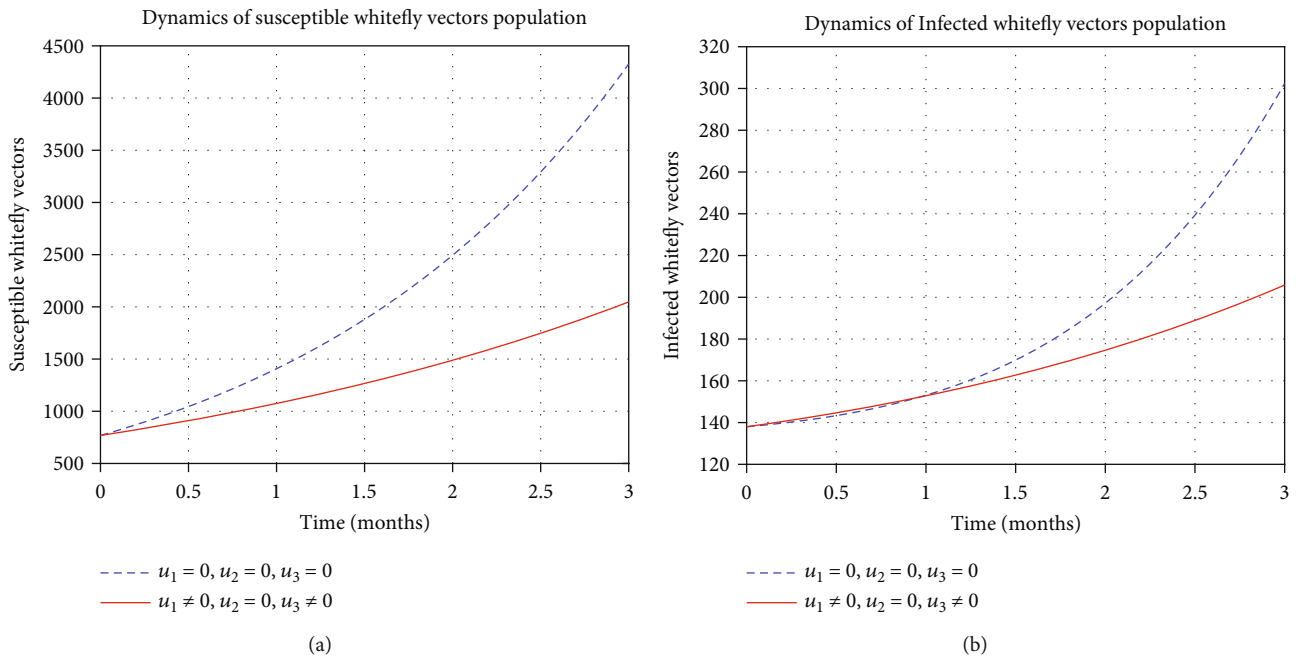

Figure 2: The impact of insecticide spray and virus-resistant cultivars on (a) susceptible whitefly vector and (b) infective whitefly vector.

This is justifiable because the virus-resistant cultivars resisted not to be infected, and insecticide spray is effective against the TYLCV infection.

5.1.2. Strategy II: Combination of the Use of Roguing Diseased Tomato Plants and Virus-Resistant Cultivars. The combination of the use of roguing diseased tomato plants u_2 and virus-resistant cultivar control u_3 strategies is used to optimize the objective function J, while the insecticide spray control strategy u_1 is set to zero.

Figure 3(a) illustrates that the healthy tomato plant increases with time as the control is used and decreased to zero without the use of control. On the other hand, Figure 3(b) shows the escalation of infected tomato plants

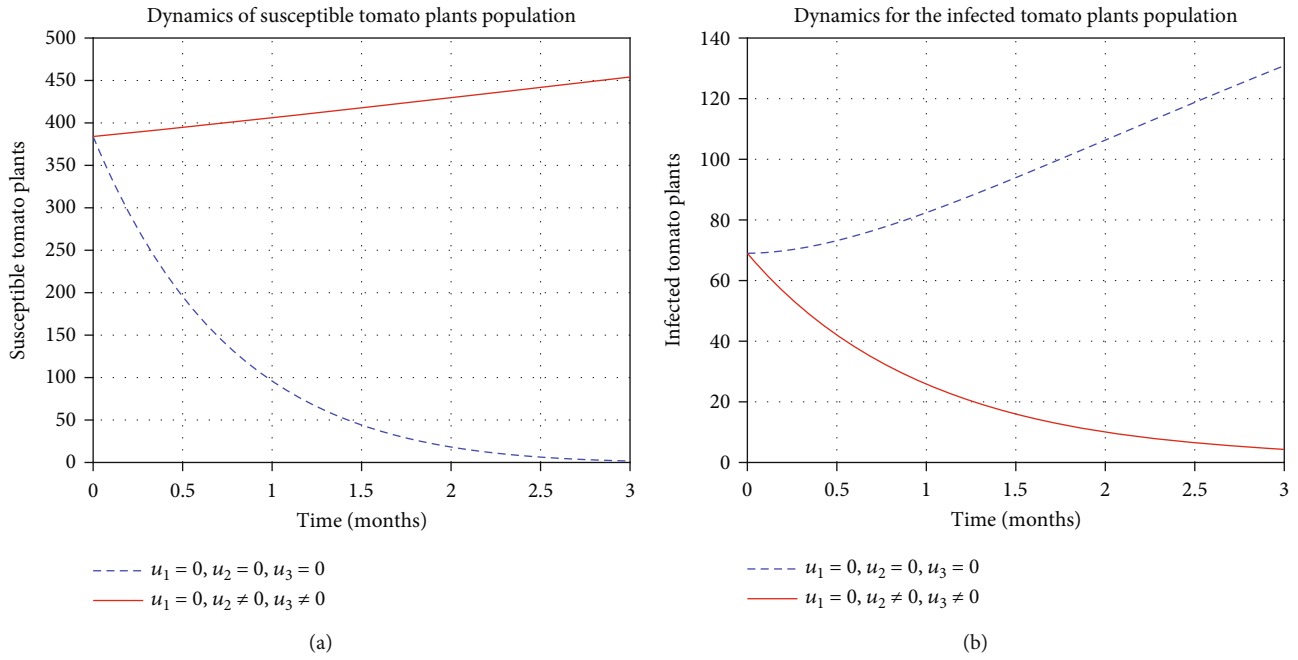

Figure 3: The impact of roguing diseased tomato plants and virus-resistant cultivars on (a) healthy tomato plant and (b) infected tomato plant.

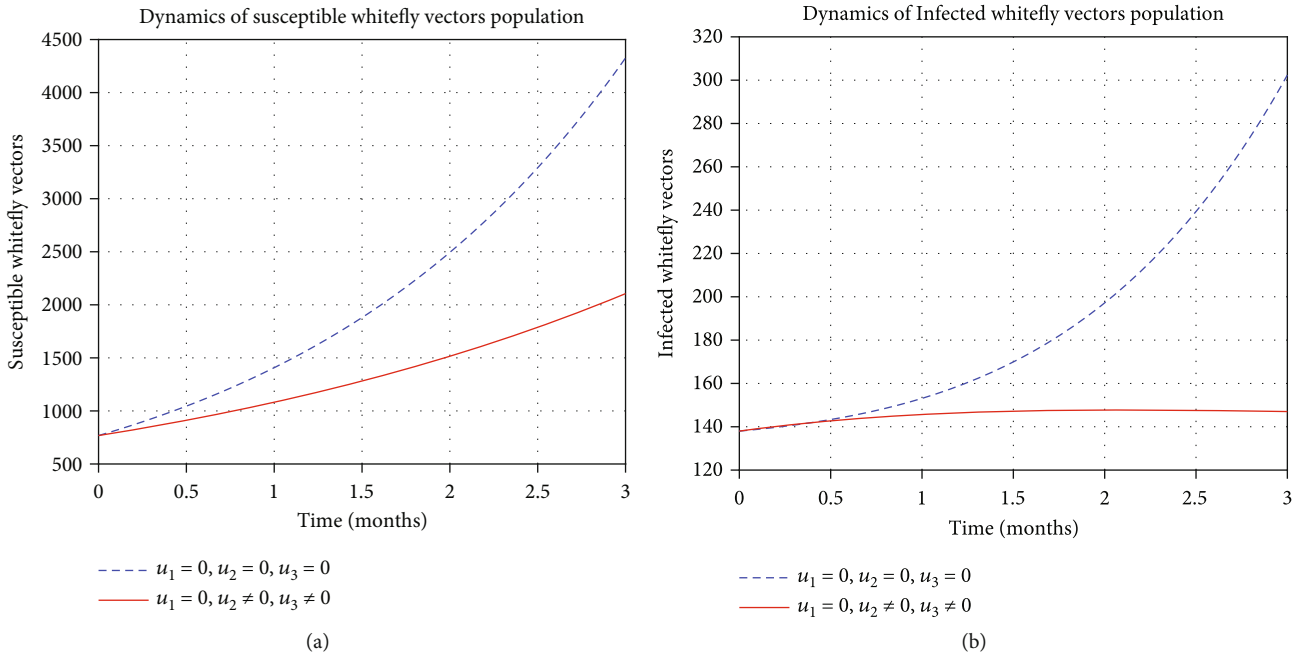

Figure 4: The impact of roguing diseased tomato plants and virus-resistant cultivars on (a) susceptible whitefly vector and (b) infective whitefly vector.

without control and the deescalation of 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 the tomato yellow leaf curl virus disease to some threshold.

Figure 4 depicts that the number of susceptible and infected whitefly vector population is increased in the absence of control. However, according to Figures 4(a) and 4(b), both the susceptible and infective whitefly vectors increased. Even though the infected whitefly vectors increase slightly in the case of control, this may be attributed to roguing infected tomato plants, decreasing the amount of secondary spread of the disease within a farm when incidences of the disease are low. However, if there are higher rates of infection (>10%), roguing may not be successful. In this study, for the simulation purpose, the number

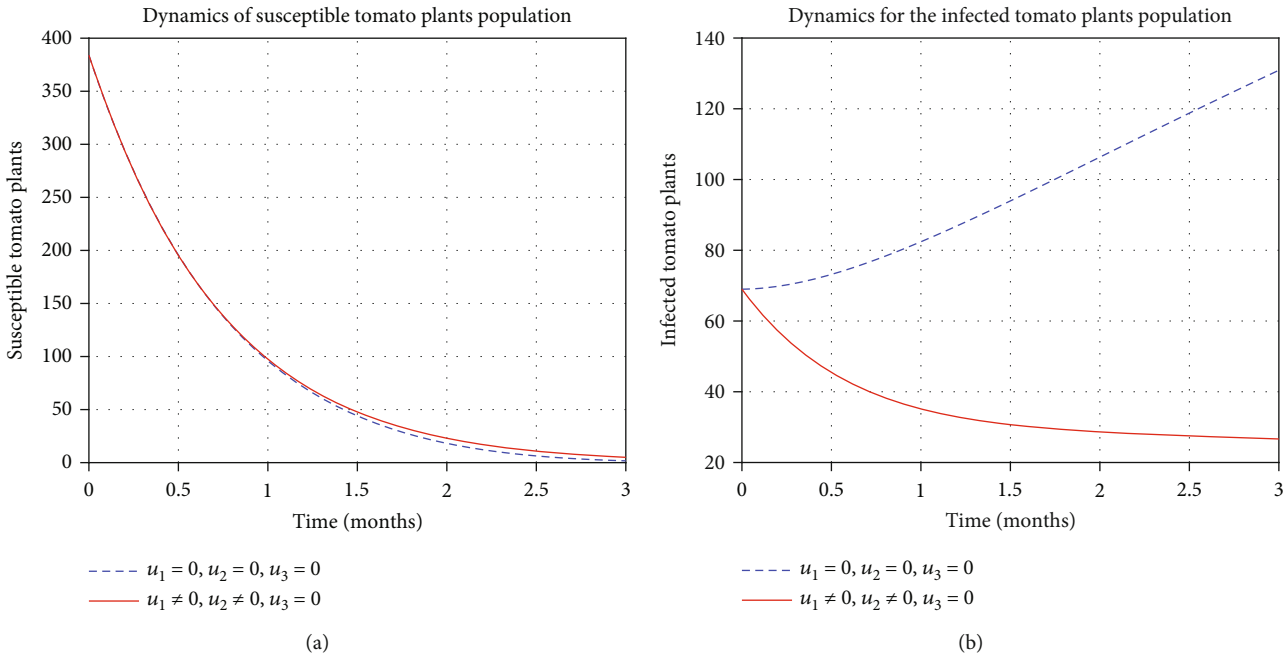

Figure 5: The impact of insecticide spray and roguing diseased tomato plant on (a) healthy tomato plant and (b) infected tomato plant.

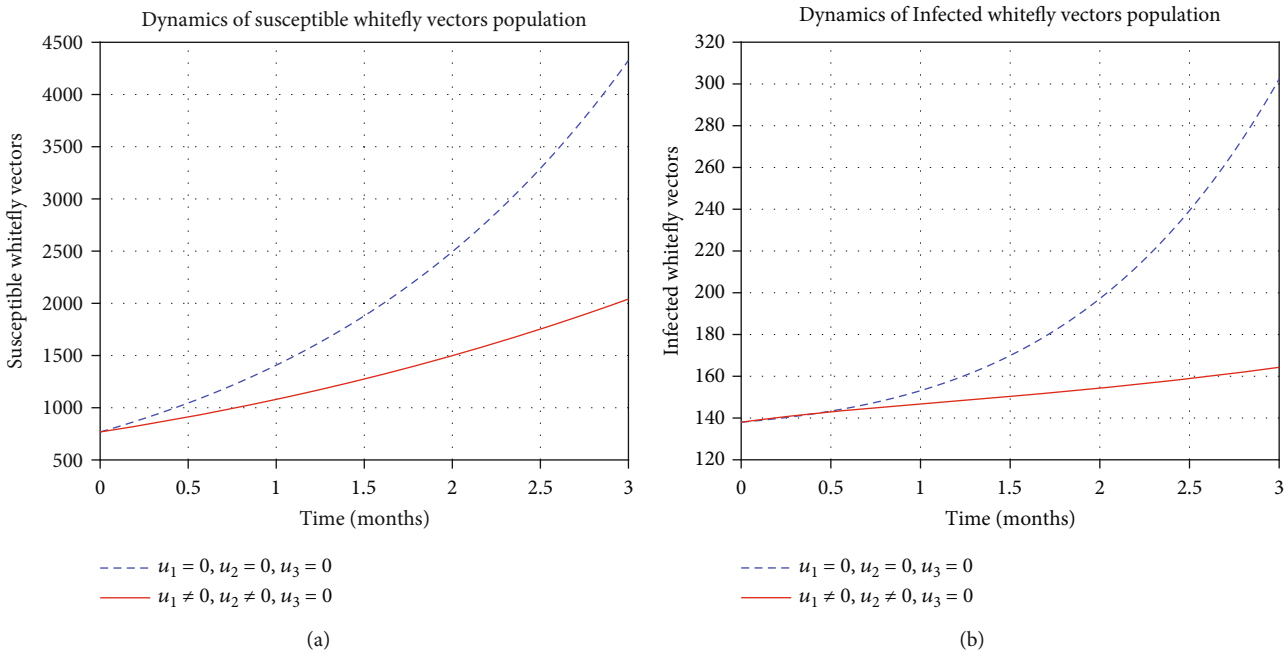

Figure 6: The impact of insecticide spray and roguing diseased tomato plant on (a) susceptible whitefly vector and (b) infective whitefly vector.

of infected tomato plants greater than 17% is considered. Besides, virus-resistant cultivars reduced losses that occurred due to the infection of TYLCV but do not act against whiteflies.

5.1.3. Strategy III: Combination of Use of Insecticide Spray and Roguing Diseased Tomato Plants. The objective function J is optimized using insecticide spray control u_1 and roguing diseased tomato plants control u_2 while virus-resistant cultivars control u_3 is set to zero.

The results in Figure 5(a) represented that without the healthy tomato plant, the population decreased with time. Figure 5(b) reveals that the infected tomato plant gradually increased without control. It is decreased with time, providing that the control is utilized. This implies that the use of insecticide spray against the spread of tomato yellow leaf

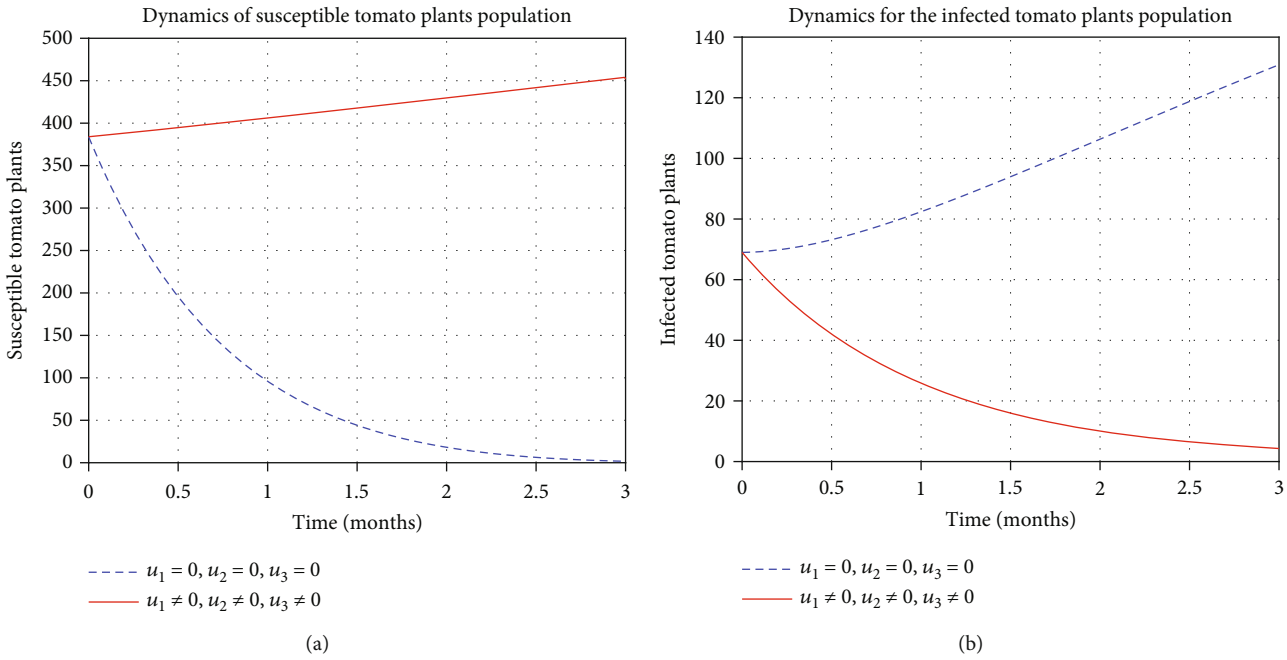

Figure 7: The impact of insecticide spray, roguing diseased tomato plant, and virus-resistance cultivars on (a) healthy tomato plant and (b) infected tomato plant.

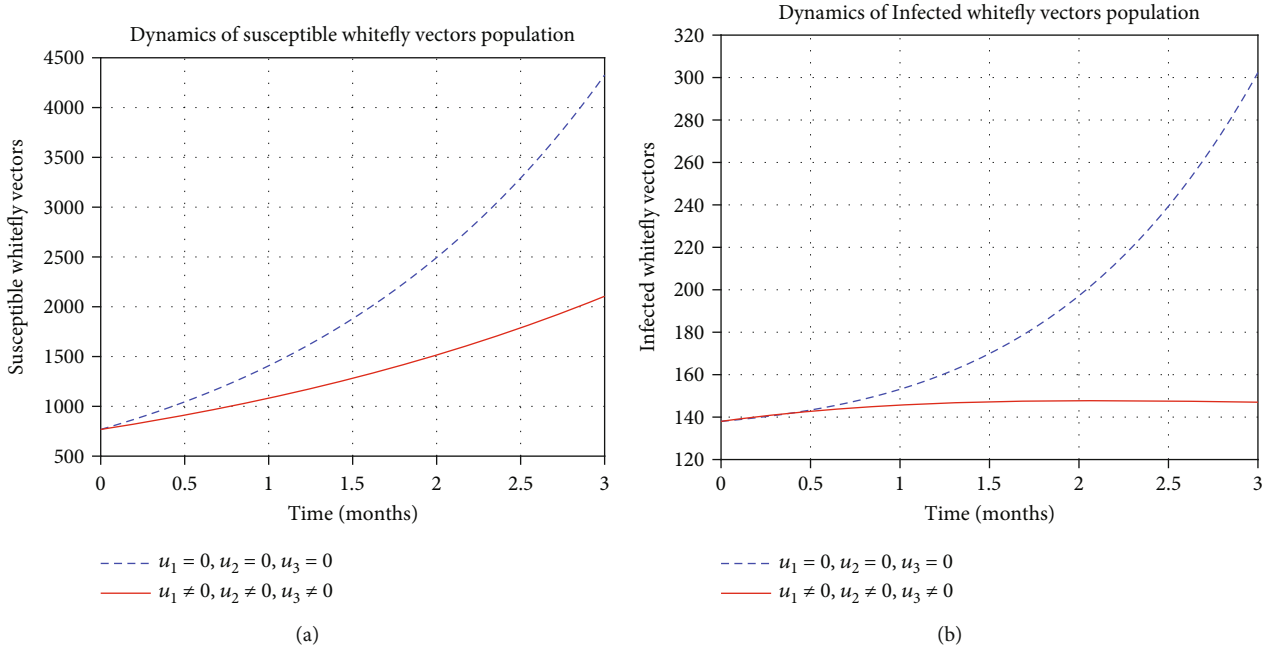

Figure 8: The impact of insecticide spray, roguing diseased tomato plant, and virus-resistant cultivars on (a) susceptible whitefly vector and (b) infective whitefly vector.

curl virus may be less efficient as it is used to hinder the foundation of the vector *B. tabaci* within the crop to protect plants from direct devastation.

According to Figure 6, the susceptible and infected whitefly vector populations escalate without control, whereas in the case of control, virus-transmitting whitefly also increased, but at a decreasing rate. This indicates that controlling virus-transmitting whitefly vectors using insecticide spray and roguing diseased tomato plant is hard [37].

5.1.4. Strategy IV: Combination of the Use of Insecticide Spray, Roguing Diseased Tomato Plants, and Virus-Resistant Cultivars. 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. It dropped off to zero without control. Figure 7(b) portrays a significant decrease in the numbers of infected tomato plants in the case of control. It is dramatically increased in the absence of control. This implies that a combination of insecticide spray, rouging diseased tomato plant, and virus-resistant cultivars declined TYLCV disease.

Figures 8(a) and 8(b) explain that with the application of control, the susceptible and infected whitefly vectors are decreased significantly in the case of control usage, whereas it is increased in the case of no control.

6. Conclusion

In this paper, a simple deterministic model for the transmission of TYLCV disease that incorporates the strategies of the use of insecticide spray, roguing the diseased tomato plants, and virus-resistance cultivars is used. It performs optimal control analysis of the model. The basic reproduction number is calculated, and the existence of local and global stability of equilibria is analyzed. We initially take into account the constant control case, we calculate the basic reproduction number, and we investigate the existence and stability of the disease-free and endemic equilibria. We use the Kamgang-Sallet stability to ensure that the disease-free equilibrium point is globally asymptotically stable when the reproduction number R_0 is less than one. This indicates that TYLCVD dies out independent of the initial size of the tomato population. For $R_0 < 1$, the disease-free equilibrium becomes unstable, and the endemic equilibrium is globally asymptotically stable. This indicates that TYLCVD spreads. The sensitivity analysis of the basic reproduction number shows that TYLCV disease has a positive relationship with the rate of harvested or removed tomato fruits, inoculation of healthy tomato plants, and virus acquisition by susceptible vectors. Thus, these parameters are those that have to be targeted mostly by policymakers to combat the TYLCV disease. Hence, the optimal control analysis of the model is made, using Pontryagin's maximum principle. From the simulation results, it can be concluded that all the combined efforts of the two out of the three strategies are listed as insecticide spray, rouging diseased tomato plants, and virus resistance cultivars. These can significantly reduce the disease, except the strategic combination of the use of insecticide spray and rouging infected tomato plants. Since the strategic combination of the use of roguing diseased tomato plants and virus-resistance cultivars has a similar effect as the strategic combination of the use of all strategies, we recommend that policymakers ought to adopt the combination of the use of the combination of roguing diseased tomato plants and virus-resistance cultivars as a strategy.

Data Availability

The parameter values and initial values to support this study are from the literature and assumptions.

Conflicts of Interest

The authors have no conflicts of interest to declare.

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