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MODELLING THE IMPACT OF BEMISIA TABACI IN DYNAMICS OF TOMATO YELLOW LEAF CURL VIRUS

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Abstract. Mathematical model has been developed and analysed for the interaction between tomato yellow leaf curl virus (TYLCV) and tomato plants under the influence of Bemisia tabaci. Positivity and boundedness of the solution has been checked to ensure our model being well posed and then we computed the basic reproductive number R_0 using the next generation matrix method. Also, both local and global stability analysis at the disease free equilibrium points of the model has been done. By constructing suitable Lyapunov functional and using LaSalle's invariance principle, global stability of endemic disease equilibrium was obtained. The results show that the disease free equilibrium point (DFE) will be both locally and globally asymptotically stable if $R_0 < 1$ but unstable if $R_0 > 1$ and the endemic equilibrium point (EE) will be globally asymptotically stable if $R_0 > 1$ and unstable if $R_0 < 1$. Finally some numerical simulations were done to validate our theoretical outcomes, and the epidemiological implications of the key outcomes were briefly discussed in the last section.

1. Introduction

Tomato yellow leaf curl virus (TYLCV) is the name given to complex virus species that causes disease known as tomato yellow leaf curl disease (TYLCD). Tomato yellow leaf curl virus (TYLCV) is a major threat to the tomato crop in many tropical and sub-tropical regions[12]. TYLCV is transmitted by the insect vector Bemisia tabaci (whitefly). The virus accounts for huge losses in quantity and quality of tomatoes if unchecked. Incidences as high as 100% with undesirable consequences of crop failure have been recorded[15, 5]. The virus was detected first in Israel around 1930, and currently it affects about 30 countries around the world that grow tomatoes. Symptoms of TYLCV that exhibited by tomato plants is reduction in leaf size, leaf curling upward, severe stunting and distortion associated with interveinal chlorosis, observed mainly on the upper portion of plants[16, 13].The disease is among the major virus diseases that causes low yields of tomatoes in Tanzania, especially, in farmer's fields. Disease occurrence of about 100% had been reported in some regions in Tanzania mainland[9, 14].

Key words and phrases. TYLCD; TYLCV; Bemisia tabaci; Basic reproductive number, Stability analysis.

2. Formulation of the TYLCD model

Plant population has been divided into three categories in SEI type structure, reflecting the disease status: susceptible (healthy), exposed (latent infection) and infectious. Similarly we have defined categories for the vector population in SI type structure as susceptible (non infectious) and infective which links the epidemiology of disease with the population dynamics of the vector. In SEI, the total tomato population N_T was subdivided into three sub-populations; tomato plants that are susceptible to infection with TYCLV S_T, those exposed to TYCLV E_T and infectious tomato plants I_T. That is $N_T = S_T+E_T+I_T$. The total whitefly vector population N_V is sub-divided into two sub-populations; susceptible whitefly vector population S_V and infectious whitefly vector population I_V. That is $N_V = S_V + I_V$. The transmission dynamics of TYLCV is summarised in the compartmental diagram in Figure 2.1.

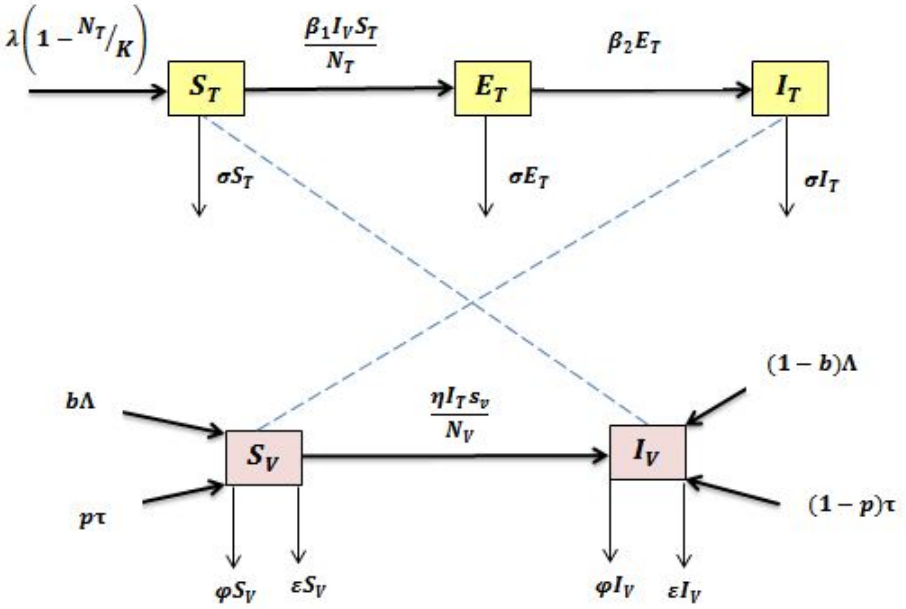

Figure 1. Compartmental diagram for the transmission dynamics of TYLCV

We assume that healthy tomato plants are planted/replanted at the rate λ . Plants either die naturally at a rate σ or move to the exposed class after acquiring TYLCV through the contact with infectious whitefly vector at a rate β_1. The exposed tomato plants are either die naturally at rate σ or move to the infectious class at rate β_2. Whitefly vectors are recruited by two different ways that is by birth and immigration. Whitefly vectors born at rate of Λ and immigration at rate of τ. Probability that whitefly vector born susceptible is b while probability of being born infected will be $1-b$. Probability that whitefly vector immigrating susceptible is p while probability of immigrating infected will be $1-p$. They either die naturally at a rate φ or emigrate at rate ε. They move to the infectious class after acquiring TYLCV from the infected tomato plants at a rate η.

The model assumes that a year-round vegetable production system is used. Replanting took place at a rate which exactly balanced those plants removed. This means that we maintain a host population at a constant size by balancing the birth and death (or replanting and mortality) rates. Farmers plant only healthy varieties of tomato in a garden of carrying capacity K, no death of tomato plants before harvesting and the whitefly vectors are assumed to remain infectious once they acquire the virus. The variables and the parameters are summarised in Tables 2.1 and 2.2.

TABLE 1. Model Variables

Variable	Description
S_T	Susceptible tomato plants
E_T	Exposed tomato plants
I_T	Infectious plants
N_T	Total plant population
S_V	Susceptible whitefly
I_V	Infectious whitefly
N_V	Total whitefly population

Table 2: Model Parameters

Parameter	**Description**
λ	Planting/replanting rate of tomato
Λ	Total whitefly population birth rate
ε	Total whitefly population immigration rate
b	Probability of susceptible offspring being born
p	Probability of susceptible whitefly vector immigrate
τ	Total whitefly population emigration rate
φ	Natural death rate of tomato
σ	Natural death rate of whitefly vector
β_1	Rate of tomato plants to move to exposed class
β_2	Rate of exposed tomato to move to infectious class
η	Rate of susceptible whitefly to move to the infectious class
K	Garden carrying capacity

From the compartmental diagram in Figure 2.1 and basing on assumptions and descriptions made we derive the following equations:

Tomato plants equations

$$
\begin{aligned}
\frac{dS_T}{dt} &= \lambda(1-\frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T,\\
\frac{dE_T}{dt} &= \frac{\beta_1 I_V S_T}{N_T} - \beta_2 E_T - \sigma E_T,\\
\frac{dI_T}{dt} &= \beta_2 E_T - \sigma I_T.
\end{aligned} \tag{1}
$$

Vector equations

$$
\begin{aligned}
\frac{dS_V}{dt} &= \Lambda + p\tau - \frac{\eta I_T S_V}{N_V} - \varphi S_V - \varepsilon S_V,\\
\frac{dI_V}{dt} &= (1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V.
\end{aligned} \tag{2}
$$

3. Basic properties of the model

3.1. **Positivity.** In order the TYLCD model to be meaningful epidemiologically, we must show that all state variables are non-negative for all times $t \geq 0$. We find solution of each equation in systems (1) and (2) in their patches for testing positivity.

Lemma 3.1. *Let the initial data be* $\{(S_T(0), E_T(0), I_T(0), S_V(0), I_V(0)) > 0\} \in \Omega$; *then solution set* $\{S_T(t), E_T(t), I_T(t), S_V(t), I_V(t)\}$ *of the model system is positive* $\forall t > 0$.

Proof. From the first equation of the tomato plant population of the model system (1),

$$\frac{dS_T}{dt} = \lambda(1-\frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T.$$

$$\frac{dS_T}{dt} \geq -(\frac{\beta_1 I_V}{N_T} - \sigma) S_T(t). \tag{3}$$

By separating variables and integrating both sides of the equation (3) above we obtain,

$S_T(t) \geq S_T(0) e^{-(\frac{\beta_1 I_V}{N_T} - \sigma)t} \geq 0$. Therefore $S_T(t) > 0$.

Following similar procedure we can compute all variables $E_T(t), I_T(t), S_V(t)$ and $I_V(t)$ and establish that $E_T(t), I_T(t), S_V(t)$ and $I_V(t) > 0$.

Therefore the solution set $\{S_T(t), E_T(t), I_T(t), S_V(t), I_V(t)\}$ of the model is positive $\forall t > 0$.

3.2. **Invariant region.** Since the system is modelling tomato plants and whitefly vector populations, we assumed that the state variables and parameters of the model are completely non-negative $t \geq 0$. The TYLCD transmission model has two compartments where each population is treated separately.

Lemma 3.2. *All forward solutions of the of the TYLCD model in* R^5_+ *enter the invariant region* $\Omega = \Omega_T + \Omega_V$ *where* $\Omega_T = \{(S_T, E_T, I_T) \in: (S_T + E_T + I_T = N_T)\}$, $\Omega_V = \{(S_V, I_V) \in: (S_V + I_V = N_V)\}$, *and* Ω *is the invariant region of the whole system.*

Proof. Let N_T the total population of the Tomato plants $N_T = S_T + E_T + I_T$. Addition

of equations in system (1) yield $\frac{N_T}{dt} = \lambda(1 - \frac{N_T}{K}) - \sigma N_T$. By separating variables in this equation, applying an integrating factor technique we compute the differential equation and obtain,

$$N_T = \frac{\lambda + K\lambda}{K} + Ce^{-(\frac{\lambda}{K}+\sigma)t}.$$

$$N_T = \frac{\lambda + K\lambda}{K} + (N_T(0) - \frac{\lambda + K\lambda}{K})e^{-(\frac{\lambda}{K}+\sigma)t}.$$

when t $\longrightarrow \infty$ $N_T(t) \longrightarrow \frac{\lambda+K\lambda}{K}$, for $t = 0$, $N_T(t) = N_T(0)$ which implies that $N_T(t) \geq 0$. Let N_T be the total population of the whitefly vector, then $N_V = S_T + I_V$. Addition of equations in system (2) yield $\frac{N_V}{dt} = 2(b\Lambda + p\tau - 1) - (\varphi + \varepsilon)$. By separating variables in this equation, applying an integrating factor technique we compute the differential equation and obtain,

$$N_V = \frac{2(b\Lambda + p\tau - 1)}{\varphi + \varepsilon} + Ce^{-(\varphi+\varepsilon)t}.$$

$$N_V = \frac{2(b\Lambda + p\tau - 1)}{\varphi + \varepsilon} + (N_V(0) - \frac{2(b\Lambda + p\tau - 1)}{\varphi + \varepsilon})e^{-(\varphi+\varepsilon)t}, \quad where \quad (\varphi + \varepsilon) \neq 0.$$

when $t \longrightarrow \infty$ $N_V(t) \longrightarrow \frac{2(b\Lambda+p\tau-1)}{\varphi+\varepsilon}$, for $t = 0$, $N_V(t) = N_V(0)$ which implies that $N_V(t) \geq 0$.

Therefore since $N_T(t) > 0$ and $N_V(t) > 0$, hence the set $\{(S_T, E_T, I_T) \in R^3_+; ((S_V, I_V) \in R^2_+)\}$ is a positive invariant set in Ω.

4. Model analysis

4.1. **Disease free equilibrium point D^0.** At disease free, $\frac{dE_T}{dt} = \frac{dI_T}{dt} = \frac{dI_V}{dt} = 0$,
$\frac{dS_T}{dt} = \lambda(1 - \frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T, \lambda(1 - \frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T = 0.$
But since at disease free, $I_V = 0$, $S_T = N_T$ therefore $\lambda(1 - \frac{S_T}{K}) - \sigma S_T = 0$. Making S_T the subject, we obtain $S_T = \frac{\lambda K}{\sigma K + \lambda}$. Considering following equations, $\frac{dS_V}{dt} = \Lambda + p\tau - \frac{\eta I_T S_V}{N_V} - \varphi S_V - \varepsilon S_V$, $\frac{dI_V}{dt} = (1 - b)\Lambda + (1 - p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V$.

$$\Lambda + p\tau - \frac{\eta I_T S_V}{N_V} - \varphi S_V - \varepsilon S_V = 0, \tag{4}$$

$$(1 - b)\Lambda + (1 - p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V = 0. \tag{5}$$

Addition of these equations (4) and (5), results $b\Lambda + p\tau - \varphi S_V - \varepsilon S_V + (1 - b)\Lambda + (1 - p)\tau - \varphi I_V - \varepsilon I_V = 0$. But $I_V = 0$.
$b\Lambda + p\tau - \varphi S_V - \varepsilon S_V + (1 - b)\Lambda + (1 - p)\tau = 0$, $S_V = \frac{b\Lambda+p\tau}{\varphi+\varepsilon}$.
Therefore the D^0 of the model system is given by $(\frac{\lambda K}{\sigma K+\lambda}, 0, 0, \frac{b\Lambda+p\tau}{\varphi+\varepsilon}, 0)$.

4.2. **Endemic equilibrium point D^*.** In the presence of TYLCD, $E_T \neq 0, I_T \neq 0, I_V \neq 0$ our model has an equilibrium point called endemic equilibrium point denoted $D^* = (S_T^*, E_T^*, I_T^*, S_V^*, I_V^*) \neq 0$. D^* is the steady state solution where TYLCD persist in the population of tomato plants. For the existence of D^*, the elements must satisfy; $S_T^* > 0, E_T^* > 0, I_T^* > 0, S_V^* > 0, I_V^* > 0$. We find the endemic equilibrium point by setting the right side of the model system equations (1) and (2) equal to zero. Thus;
$I_T^* = \frac{\beta_2\sigma(b\Lambda+p\tau)-(\varphi+\varepsilon)}{\beta_2\eta\lambda(1-\frac{N_T^*}{K})-(\beta_2\sigma)}$,
$S_T^* = \frac{\lambda}{\sigma}(1-\frac{N_T^*}{K}) - \frac{I_T^*}{\beta_2}(\beta_2+\sigma)$,
$E_T^* = \frac{\sigma I^* T}{\beta_2}$,
$S_T^* = \frac{(b\Lambda+p\tau)N_V^*}{\eta I_T^*+(\varphi+\varepsilon)N_V^*}$,
$I_V^* = \frac{N_V^*(I_T^*+(\varphi+\varepsilon))((1-b)\Lambda+(1-p)\tau)+ITNV(b\Lambda+p\tau)}{(\varphi+\varepsilon)(\eta I_T^*+(\varphi+\varepsilon)N_V^*)}$.
For a positive endemic equilibrium point, the conditions $\frac{\lambda}{\sigma}(1-\frac{N_T^*}{K}) > \frac{I_T^*}{\beta_2}(\beta_2+\sigma), \beta_2\sigma(b\Lambda + p\tau) > (\varphi+\varepsilon)$ and $\beta_2\eta\lambda(1-\frac{N_T^*}{K}) > (\beta_2+\sigma)$ must hold.

4.3. **The basic reproduction number R_0.** The basic reproduction number (R_0) is the expected number of secondary cases produced by a single infection into a completely susceptible population[1]. [3] defined R_0 as a spectral radius ($\rho(FV^{-1})$) of the next generation matrix. This is a parameter which usually tells if the epidemiologic might persist or die out within a purely susceptible population. The epidemiological criterion of R_0 is that if $Ro > 1$ then the disease free equilibrium point is unstable and can invade the population and persist for long time [17]. There are several methods for calculating R_0 but in this study the next generation operator method as proposed by [17] is used.
The disease transmission model comprised of the system of equations $X_j^* = F_j(x) - V_j(x)$, where $F_j(x)$ is for new infection and $V_j(x)$ is for remaining transfer terms. To obtain the matrix F and V (Jacobian matrices), we then find derivatives of vectors $F_j(x)$ and $V_j(x)$ respectively.

From the model equation,

$$F_j(x_0) = \begin{pmatrix} \frac{\beta_1 I_V S_T}{N_T} \\ 0 \\ (1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} \end{pmatrix}.$$

The Jacobian matrix at D^0 is

$$F = \begin{pmatrix} 0 & 0 & \beta_1 \\ 0 & 0 & 0 \\ 0 & \eta & 0 \end{pmatrix}.$$

From the model equation,

$$V_j(x)= \begin{pmatrix} \beta_2 E_T + \sigma E_T \\ \sigma I_T - \beta_2 E_T \\ \varphi I_V + \varepsilon I_V \end{pmatrix}.$$

The Jacobian matrix at D^0 is

$$V = \begin{pmatrix} \beta_2+\sigma & 0 & 0 \\ -\beta_2 & \sigma & 0 \\ 0 & 0 & \varphi+\varepsilon \end{pmatrix}, \text{ and } V^{-1}= \begin{pmatrix} \frac{1}{\beta_2+\sigma} & 0 & 0 \\ \frac{\beta_2}{\beta_2+\sigma} & \frac{1}{\sigma} & 0 \\ 0 & 0 & \frac{1}{\varphi+\varepsilon} \end{pmatrix}.$$

Multiplying F and V^{-1} yields a next generation matrix FV^{-1} as shown below,

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_2}{\varphi+\varepsilon} \\ 0 & 0 & 0 \\ \frac{\beta_2\eta}{\beta_2+\sigma} & \frac{\eta}{\sigma} & 0 \end{pmatrix}.$$

Computing the maximum eigen value of the next generation matrix we get $R_0 = \sqrt{\frac{\beta_1\beta_2\eta}{(\varphi+\varepsilon)(\beta_2+\sigma)}}$. R_0 is a threshold parameter that indicates the average number of infected vectors and infected hosts caused by a cross-infection of one tomato plant host or one whitefly vector when the other population consist of only susceptible [10]. The square root arises from the fact that dual generations are necessary for transmission of TYLCD to take place, i.e. From an infectious tomato plant to a susceptible whitefly vector and then from an infectious whitefly vector to susceptible tomato plant(host) [10].

4.4. **Stability analysis.**

4.4.1. *Local stability of the disease free equilibrium point.* We examine the stability of the disease free equilibrium point $D^0 = (\frac{\lambda K}{\sigma K+\lambda}, 0, 0, \frac{b\Lambda+p\tau}{\varphi+\varepsilon}, 0)$ by employing the method used by Kinene et al. (2015).

Lemma 4.1. *The disease free equilibrium D^0 is locally asymptotically stable if $R_0 < 1$ but unstable if $R_0 > 1$, where R_0 is the basic reproduction number.*

Proof. We linearize the model system (1) and (2) by computing its Jacobian matrix J_{D^0}. The Jacobian matrix is computed at disease free equilibrium point by differentiating each equation in the system with respect to the state variables S_T, E_T, I_T, S_V, and I_V. Therefore the Jacobian matrix is

$$J_{D^0} = \begin{pmatrix} -\frac{\beta_1 I_V}{N_T} - \sigma & 0 & 0 & 0 & -\beta_1 \\ 0 & -\beta_2 - \sigma & 0 & 0 & \beta_1 \\ 0 & \beta_2 & -\sigma & 0 & 0 \\ 0 & 0 & -\eta & -(\frac{\eta}{S_V} + \varphi + \varepsilon) & 0 \\ 0 & 0 & \eta & 0 & -\varphi - \varepsilon \end{pmatrix}. \tag{6}$$

It is clear from equation (6) that the first and second eigen values are $\lambda_1 = -\sigma$ and $\lambda_2 = -(\frac{\eta(\varphi+\varepsilon)}{b\Lambda+p\tau} + \varphi + \varepsilon)$. This can be reduced to a (3×3) matrix

$$J_{D^0} = \begin{pmatrix} -\beta_2 - \sigma & 0 & \beta_1 \\ \beta_2 & -\sigma & 0 \\ 0 & \eta & -\varphi - \varepsilon \end{pmatrix}. \tag{7}$$

Its characteristics polynomial is
$(\varepsilon+\mu+2\sigma+\beta_2)\lambda^2 + (\sigma^2 + (2\mu+2\varepsilon+\beta_2)\sigma + \beta_2(\varepsilon+\mu))\lambda + (\varepsilon+\mu)\sigma^2 + (\varepsilon+\mu)\beta_2\sigma - \beta_1\beta_2\eta +$
$(\varepsilon+\mu+2\sigma+\beta_2)\lambda^2 + (\sigma^2 + (2\mu+2\varepsilon+\beta_2)\sigma + \beta_2(\varepsilon+\mu))\lambda + (\varepsilon+\mu)\sigma^2 + (\varepsilon+\mu)\beta_2\sigma - \beta_1\beta_2\eta.$
$a_1 = \varepsilon+\mu+2\sigma+\beta_2, \quad a_2 = (\sigma^2+(2\mu+2\varepsilon+\beta_2)\sigma+\beta_2(\varepsilon+\mu)), \quad a_3 = (\varepsilon+\mu)\sigma^2+(\varepsilon+\mu)\beta_2\sigma-\beta_1\beta_2\eta.$
Consider $(\varepsilon+\mu+2\sigma+\beta_2) > 0$. Since all model parameters are positive, it is obvious that a_1 is positive $(a_1 > 0)$.
Consider $(\sigma^2 + (2\mu+2\varepsilon+\beta_2)\sigma + \beta_2(\varepsilon+\mu)) > 0$. From the point that all model parameters are positive, it is obvious that a_2 is positive $(a_2 > 0)$.
In order that a_3 being positive, $(\varepsilon+\mu)\sigma^2 + (\varepsilon+\mu)\beta_2\sigma - \beta_1\beta_2\eta > 0$.
$(\varepsilon+\mu)\sigma^2 + (\varepsilon+\mu)\beta_2\sigma > \beta_1\beta_2\eta, \quad 1 > \frac{\beta_1\beta_2\eta}{(\varepsilon+\mu)\sigma^2+(\varepsilon+\mu)\beta_2\sigma}, \quad 1 > \dfrac{R_0^2}{\sigma}.$
$a_1a_2-a_3 = (\varepsilon+\mu+2\sigma+\beta_2)*(\sigma^2+(2\mu+2\varepsilon+\beta_2)\sigma+\beta_2(\varepsilon+\mu))-((\varepsilon+\mu)\sigma^2+(\varepsilon+\mu)\beta_2\sigma-\beta_1\beta_2\eta).$
After simplification yields $a_1a_2 - a_3 > 0$.
By Routh-Hurwitz criteria all eigenvalues have negative real parts if $R_0 < 1$ thus making the disease free equilibrium locally asymptotically stable.

4.4.2. *Global stability of the disease free equilibrium point.* We examine global stability of the disease free equilibrium using the theorem proposed by [2] similar as in [10]. Thus we rewrite our model and mention two conditions, if satisfied will guarantee global asymptotic stability of the disease free equilibrium.

$$F(X,Z) = \begin{cases} \dfrac{dX}{dt} & = F(X,Z), \\ \dfrac{dZ}{dt} & = G(X,Z), \quad G(X,Z) = 0, \end{cases} \tag{8}$$

where $X = (S_T, S_V) \in R^2$ denote susceptible populations and $Z = (E_T, I_T, I_V) \in R^3$ denote infected populations. $D^0 = (X^*, 0)$ represent the disease free equilibrium of this system. The conditions (i) and (ii) below guarantee global asymptotic stability:

(i): for $\dfrac{dX}{dt} = F(X,0)$, X^* is global asymptotically stable.

(ii): $G(X,Z) = D_Z G(X^*,Z)Z - \hat{G}(X,Z), \quad \hat{G}(X,Z) \geq 0$ for, $(X,Z) \in \Omega$,

where $D_Z G(X^*,Z)$ is Metzler Matrix (the off diagonal elements are non negative) and also the Jacobian of $G(X,Z)$ taken in (E_T, I_T, I_V) and evaluated at $(X^*,0) = (\frac{\lambda K}{\sigma K+\lambda}, \frac{b\Lambda+p\tau}{\varphi+\varepsilon}, 0, 0, 0)$.
If the system satisfies the above conditions, then according to [2] the following theorem holds.

Lemma 4.2. *The equilibrium point $D^0 = (X^*, 0)$ of the system (8) is globally asymptotically stable if $R_0 \leq 1$ and the conditions (i) and (ii) are satisfied.*

Proof. We now start our proof by describing new variables and breaking the system into subsystems. $X = (S_T, S_V)$ and $Z = (E_T, I_T, I_V)$. From equation (8) we have two vector valued functions G(X.Z) and F(X,Z) given by:

$$F(X,Z) = \begin{cases} \lambda(1 - \frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T, \\ b\Lambda + p\tau - \frac{\eta I_T S_V}{N_V} - \varphi S_V - \varepsilon S_V, \end{cases}$$

$$G(X,Z) = \begin{cases} \frac{\beta_1 I_V S_T}{N_T} - \beta_2 E_T - \sigma E_T, \\ \beta_2 E_T - \sigma I_T, \\ (1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V. \end{cases}$$

For condition (i) we considering the reduced system $\dfrac{dX}{dt} = F(X, 0)$.

$$F(X,Z) = \begin{cases} \dfrac{dS_T}{dt} = \lambda(1 - \frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T, \\ \dfrac{dS_V}{dt} = b\Lambda + p\tau - \frac{\eta I_T S_V}{N_V} - \varphi S_V - \varepsilon S_V. \end{cases} \tag{9}$$

$X^* = (\frac{\lambda K}{\sigma K + \lambda}, \frac{b\Lambda + p\tau}{\varphi + \varepsilon})$ is globally asymptotically stable equilibrium. To prove this, we find the solution of $\dfrac{dX}{dt} = F(X, 0)$. When we find solution of the first equation from (9) yields $S_T = \frac{\lambda K}{\sigma K + \lambda} + (S_T(0) - \frac{\lambda K}{\sigma K + \lambda}) e^{-(\frac{\sigma K + \lambda}{\lambda K})t}$ that approaches $\frac{\lambda K}{\sigma K + \lambda}$ if $t \longrightarrow \infty$. Similarly solving the second equation of (9) yields $S_V = \frac{b\Lambda + p\tau}{\varphi + \varepsilon} + (S_V(0) - \frac{b\Lambda + p\tau}{\varphi + \varepsilon})\ e^{-(\frac{b\Lambda + p\tau}{\varphi + \varepsilon})t}$ that approaches $\frac{b\Lambda + p\tau}{\varphi + \varepsilon}$ if $t \longrightarrow \infty$ regardless of the initial condition. Thus, is globally asymptotically stable.

Meanwhile for condition (ii) we compute $G(X,Z) = D_Z G(X^*, Z)Z - \hat{G}(X,Z)$, and then prove that $\hat{G}(X,Z) \geq 0, for \quad (X,Z) \in \Omega$.

$$G(X,Z) = \begin{pmatrix} \frac{\beta_1 I_V S_T}{N_T} - \beta_2 E_T - \sigma E_T \\ \beta_2 E_T - \sigma I_T \\ (1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V \end{pmatrix}.$$

At disease free equilibrium point,

$$D_Z G(X^*, 0) = \begin{pmatrix} -(\beta_1 + \sigma) & 0 & \beta_1 \\ \beta_2 & -\sigma & 0 \\ 0 & \eta & -(\varphi + \varepsilon) \end{pmatrix},\ D_Z G(X^*, 0)Z = \begin{pmatrix} (\beta_1 + \sigma)E_T + \beta_1 I_V \\ \beta_2 E_T - \sigma I_T \\ \eta I_T - (\varphi + \varepsilon) I_V \end{pmatrix}.$$

By applying the formula $\hat{G}(X,Z) = D_Z G(X^*, Z)Z - G(X,Z)$,

$$\hat{G}(X,Z) = \begin{pmatrix} -(\beta_1+\sigma)E_T + \beta_1 I_V \\ \beta_2 E_T - \sigma I_T \\ \eta I_T - (\varphi+\varepsilon)I_V \end{pmatrix} - \begin{pmatrix} \frac{\beta_1 I_V S_T}{N_T} - \beta_2 E_T - \sigma E_T \\ \beta_2 E_T - \sigma I_T \\ (1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V \end{pmatrix}.$$

$$\hat{G}(X,Z) = \begin{pmatrix} \beta_1 I_V(1-\frac{S_T}{N_T}) \\ 0 \\ \eta I_T - ((1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V}) \end{pmatrix}.$$

Since $(1-\frac{S_T}{N_T}) \geq 0$ then $\beta_1 I_V(1-\frac{S_T}{N_T}) \geq 0$, meanwhile for $\eta I_T \geq ((1-b)\Lambda+(1-p)\tau+\frac{\eta I_T S_V}{N_V})$ then $\eta I_T-((1-b)\Lambda+(1-p)\tau+\frac{\eta I_T S_V}{N_V}) \geq 0$. Therefore $\hat{G}(X,Z) \geq 0$ and hence this completes the proof of lemma 4.2.

4.4.3. *Global stability of endemic equilibrium point.* Global stability of the endemic equilibrium point D^* is analyzed by constructing a suitable Lyapunov function. We prove for global stability of endemic equilibrium point following the approach used by [8] and other several epidemiological models. We consider the Lyapunov function of the form $L = \sum P_i(R_i - R_i^* \ln(R_i))$.

So we define the Lyapunov function as $L(S_T, I_T, S_V, I_V) = P_1(S_T - S_T^* \ln(S_T)) + P_2(E_T - E_T^* \ln(E_T)) + P_3(I_T - I_T^* \ln(I_T)) + P_4(S_V - S_V^* \ln(S_V)) + P_5(I_V - I_V^* \ln(I_V))$.

We can now verify the condition $\dfrac{dL}{dt} \leq 0$.

Proof. The time derivative of L is $\dfrac{dL}{dt} = P_1(1-\frac{S_T^*}{S_T})\dfrac{dS_T}{dt} + P_2(1-\frac{E_T^*}{E_T})\dfrac{dE_T}{dt} + P_3(1-\frac{I_T^*}{I_T})\dfrac{dI_T}{dt} + P_4(1-\frac{S_V^*}{S_V})\dfrac{dS_V}{dt} + P_5(1-\frac{I_V^*}{I_V})\dfrac{dI_V}{dt}$

$= P_1(1-\frac{S_T^*}{S_T})(\lambda(1-\frac{N_T}{K}) - \frac{\beta_1 I_V S_T}{N_T} - \sigma S_T) + P_2(1-\frac{E_T^*}{E_T})(\frac{\beta_1 I_V S_T}{N_T} - \beta_2 E_T - \sigma E_T) + P_3(1-\frac{I_T^*}{I_T})(\beta_2 E_T - \sigma I_T) + P_4(1-\frac{S_V^*}{S_V})(b\Lambda + p\tau - \frac{\eta I_T S_V}{N_V} - \varphi S_V - \varepsilon S_V) + P_5(1-\frac{I_V^*}{I_V})((1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} - \varphi I_V - \varepsilon I_V)$.

At an endemic equilibrium point D^* we have $\lambda(1-\frac{N_T}{K}) = (\frac{\beta_1 I_V}{N_T}+\sigma)S_T^*$, $\frac{\beta_1 I_V S_T}{N_T} = (\beta_2+\sigma)E_T^*$, $\beta_2 E_T. = \sigma I_T^*$, $b\Lambda + p\tau = (\frac{\eta I_T S_V}{N_V} + \varphi + \varepsilon)S_V^*$ and $((1-b)\Lambda + (1-p)\tau + \frac{\eta I_T S_V}{N_V} = (\varphi+\varepsilon)I_V^*$.

$= P_1(1-\frac{S_T^*}{S_T})((\frac{\beta_1 I_V}{N_T}+\sigma)S_T^* - (\frac{\beta_1 I_V}{N_T}+\sigma)S_T) + P_2(1-\frac{E_T^*}{E_T})((\beta_2+\sigma)E_T^* - (\beta_2+\sigma)E_T) + P_3(1-\frac{I_T^*}{I_T})(\sigma I_T^* - \sigma I_T) + P_4(1-\frac{S_V^*}{S_V})(\frac{\eta I_T}{N_V}+\varphi+\varepsilon)S_V^* - (\frac{\eta I_T}{N_V}+\varphi+\varepsilon)S_V + P_5(1-\frac{I_V^*}{I_V})((\varphi+\varepsilon)I_V^* - (\varphi+\varepsilon)I_V)$.

$= -P_1(1-\frac{S_T^*}{S_T})^2\sigma)S_T - P_1(\frac{\beta_1 I_V}{N_T}(1-\frac{S_T^*}{S_T})(1-\frac{S_T^* I_V^*}{S_T I_V}) - P_2(1-\frac{E_T^*}{E_T})^2(\beta_2+\sigma)E_T - P_3(1-\frac{I_T^*}{I_T})^2\sigma I_T - P_4(1-\frac{S_V^*}{S_V})^2(\varphi+\varepsilon)S_V - P_4\frac{\eta I_T S_V}{N_V}(1-\frac{S_V^*}{S_V})(1-\frac{I_T^* S_V^*}{I_T S_V}) - P_5(1-\frac{I_V^*}{I_V})^2 I_V(\varphi+\varepsilon)$.

$= -P_1(1-\frac{S_T^*}{S_T})^2\sigma)S_T - P_2(1-\frac{E_T^*}{E_T})^2(\beta_2+\sigma)E_T - P_3(1-\frac{I_T^*}{I_T})^2\sigma I_T - P_4(1-\frac{S_V^*}{S_V})^2(\varphi+\varepsilon)S_V - P_5(1-\frac{I_V^*}{I_V})^2 I_V(\varphi+\varepsilon) + P(S_T, E_T, I_T, S_V, IV)$,

where $P(S_T, E_T, I_T, S_V, I_V) = (-P_1(\frac{\beta_1 I_V}{N_T}(1-\frac{S_T^*}{S_T})(1-\frac{S_T^* I_V^*}{S_T I_V}) - P_4\frac{\eta I_T S_V}{N_V}(1-\frac{S_V^*}{S_V})(1-\frac{I_T^* S_V^*}{I_T S_V})) < 0$.

The function $P(S_T, E_T, I_T, S_V, I_V)$ is non-positive by considering the method used by [8]. Thus $P(S_T, E_T, I_T, S_V, I_V) < 0$ for all $S_T, E_T, I_T, S_V, I_V > 0$. Hence, $\dfrac{dL}{dt} \leq 0$ and $\dfrac{dL}{dt} = 0$ if and only if $S_T = S_T^*, E_T = E_T^*, I_T = I_T^*, S_V = S_V^*$ and $I_V = I_V^*$. Therefore the largest

invariant set such that $\frac{dL}{dt} = 0$ is the singleton $\{D^*\}$ which is our endemic equilibrium point. By LaSalle's invariant principle in [11] we conclude that D^* is globally asymptotically stable if $R_0 > 1$ and unstable if $R_0 < 1$. So, we establish the following lemma.

Lemma 4.3. *The endemic equilibrium point D^* of the model systems (1) and (2) is globally asymptotically stable if $R_0 > 1$ and unstable otherwise.*

5. Numerical Simulation and Discussion

The main intention of this paper is to assess the impact of number of bemisia tabaci interacting with tomato plants in transmission of TYLV. In order to support the analytical results, numerical simulations were performed using different values for the initial number of vectors but constant number of host population (tomato plants) and using numerical value of parameter taken from literature while some were estimated. Fixed variables used are S_T=2000, E_T=I_T=0, while other variable were varying (S_V=I_V=500, 2500 and 5000). The final time is t_f=90 days, and the rest parameters values are shown in the Table 5.1.

Table 3: Parameter values for the model

Parameter	**Value** day^{-1}	**Reference**
λ	0.01	[4]
Λ	0.0118	[18]
ε	0.3	[4]
b	0.5	[7]
p	0.8	[4]
τ	0.267	[4]
φ	0.01	[7]
σ	0.015	[10]
β_1	0.01	[4]
β_2	0.075	[4]
η	0.33	Estimated
K	2000	Estimated

Graphs of susceptible tomatoes, exposed tomatoes and infected tomatoes were plotted against time by varying number of vector in the field. Computations were done using Matlab with the ode45. This function implements a Runge-Kutta method with a variable time step.

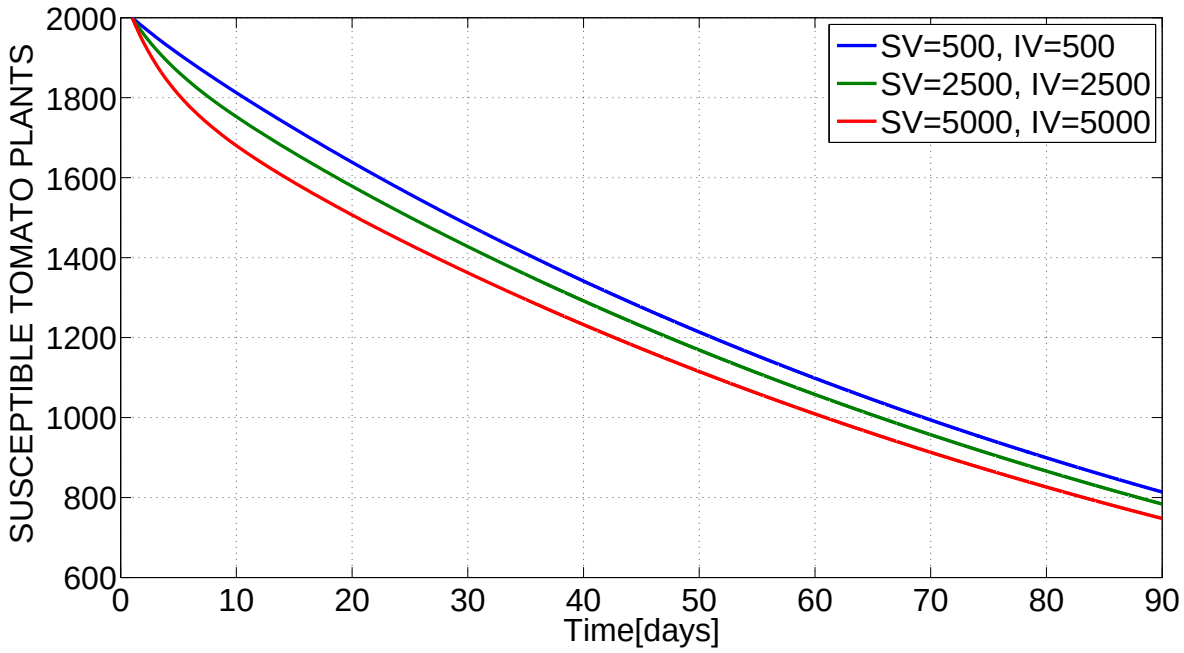

FIGURE 2. Graph of susceptible tomato plants vs. time for the first 90 days of growth

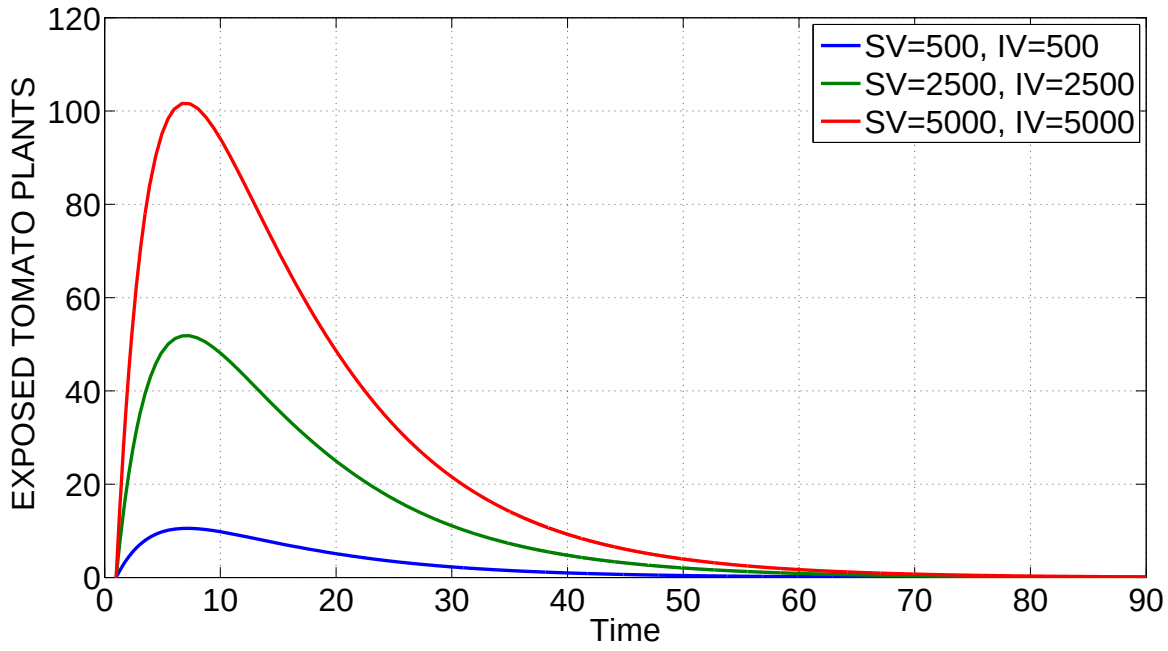

FIGURE 3. Graph of exposed tomato plants vs. time for the first 90 days of growth

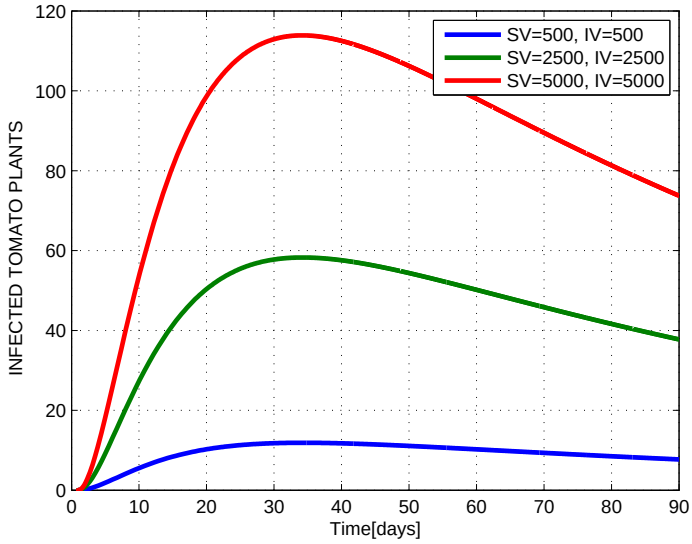

FIGURE 4. Graph of infected tomato plants vs. time for the first 90 days of growth

Numerical simulation results in Figure 5.1 shows significant decrease in the number of susceptible plants due to increase of number of vectors in the field. Figure 5.2 shows an increase in the number of exposed plants due to increase of number of vectors in the field and Figure 5.3 shows also the increase in the number of infected plants due to increase of number of vectors. This implies that the increase of number of vectors around the field lead to decrease of amount of tomato yield in that field due to TYLCV.

6. Conclusion

We computed for the basic reproductive number (R_0) that can be used to determine whether eradication of TYLCV will occur ($R_0 < 1$) or if disease will continue to increase ($R_0 > 1$). Numerical simulation of this model allows us to describe numerically dependence between the number of vectors and infection transmission to tomato plants. A better understanding of plant-virus-vector systems was obtained with the development of model incorporating crop fields and vector populations with migration of vectors between fields. We notice that in order to lessen the infections, intervention strategies need to be focused on killing vectors so that we reduce the contact between bemisia tabaci and plant host. Disease controlling in one tomato field may impact the population dynamics and proportions of susceptible and infectious vectors and affects disease dynamics in other fields due to vector migrations [6]. There is a need of effective insecticides application that would reduce the whitefly population and keep the tomato population stable. Therefore, this modelling approach can be modified and then used to evaluate control strategies of the disease.

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