J. Math. Computer Sci., 39 (2025), 474–499

Online: ISSN 2008-949X

Journal of Mathematics and Computer Science

Journal Homepage: www.isr-publications.com/jmcs

Mathematical modeling of begomovirus dynamics in tomato fields using whitefly and fungal biocontrol

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Abstract

Tomato cultivation in Burkina Faso and elsewhere constitutes a source of income for farmers. Tomato is a very important fruit for a good complete diet, unfortunately low productivity is caused by various factors including climate change, fungal plant diseases linked to pathogens and spread by insect pests. Every year farmers suffer huge losses due to the tomato yellow virus (begomovirus), which is transmitted by insect vectors Bemisia Tabaci. However, we analyze these insect management problems using mathematical models. In this paper we developed a mathematical model of tomato yellow leaf curve virus (TYLCV) disease in the tomato plantation with the growth rate of insects vectors following the logistic function, taking into account the two latent stages of the vegetative phase and the generative phase of tomato plants. We determined the value of the basic reproduction number $\mathcal{R}_0$ of the model from the dominant eigenvalue of the next generation matrix. In practice the basic reproduction number represent the number of new cases generated by an infectious individual during its infection phase . But in theory we use the basic reproduction number to analyze the stability of the equilibrium states of the model. This parameter is used to evaluated the speed of spread of the virus in the population. The results illustrates that when $\mathcal{R}_0 < 1$, the disease-free equilibrium point is asymptotically globally stable and the model has an endemic equilibrium point which is asymptotically globally stable when $\mathcal{R}_0 > 1$. We have also provided numerical simulation examples describing the population of the model that was developed. The numerical simulation results illustrate that for a use of Verticillium lecanii at a dose greater than 10% the population of tomato plants latent, infected in the vegetative and in the generative phase will experience extinction, just like the susceptible and infected population of Bemisia Tabaci. In the last part of our work, we also calculated the sensitivity indices of certain parameters that are very sensitive to the variation in the value of the basic reproduction number. These findings provide a scientific basis for optimizing biocontrol strategies in tomato farming.

Keywords: Latent stage, logistic function, Verticillium lecanii, basic reproduction number, differential equations, numerical simulations, sensitivity index.

2020 MSC: 34B18, 34D23, 34E05.

1. Introduction

Solanum Lycopersicum L. (S. Lycopersicum L.), which belongs to the nightshade family is one of the most cultivated plants in the world (Barboza et al. and Gadji et al. [6, 16]). In Burkina Faso, tomatoes are the second most important market garden crop, after onions, with a production estimated at more than 10000 tonnes in 2014 (Son et al. [43]). However during these recent years have seen a decline in yields from 11.3 tonnes/ha in 2010 to 9.7 tonnes/ha in 2014 (Son et al. [43]). These reductions in yield are

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doi: 10.22436/jmcs.039.04.05

Received: 2024-11-27 *Revised:* 2025-02-09 *Accepted:* 2025-03-11

mainly due to a complex of pests, the main ones of which are Ralstonia solanacearum (Smith); Fusarium oxysporum (Scheltdl), Bemisia tabaci (Gen); Helicoverpa armiga (Hub) and Tutaa absoluta (Meyrick) (Son et al. [43]). In particular, a diversity of species of Bemisia tabaci on market garden crops is known in the Western part (Gnakine et al. [19]). The studies carried out have show the presence of the MED-Q 1, MED-Q 3, biotypes, the ASL biotype and the SSA biotypes thanks to the mitochondrial marker which codes for the cytochrome Oxidase 1. The MED-Q 1 biotype, recognized as predominant, is often encountered in sympathry with ASL on the same host plants (Rouamba et al. and Son et al. [40, 43]). Pest control programs and vectors of TYLCV consists of using insecticides including Verticillium lecanii (Son et al. [43]), hybrid seeds, crop rotations and crops of resistant varieties. Facing the threat of TYLCV against tomato cultivation in Burkina Faso.

Despite the advantages that tomatoes represents for the well-being of populations, their cultivation faces numerous major biotic constraints which severely threaten production with enormous drops in yield (AL-MUSA, Gadji et al., and Sabuka et al. [2, 16, 41]). It is a virus belonging to the Geminiviridae family, it was recognized and identified for the first time in Israel in 1930. Since 1935, this virus has been a main permanent pest of the Jordan valleys. This virus has affected more than 30 tomato-producing countries. It is commonly found in tropical and subtropical regions (Czosnek et al. [14]). Tomato yellow leaf curl virus (TYLCV) has been found in several regions of the world such as the Mediterranean, Japan, China and many other countries (Akad et al. [1]). TYLCV is a virus that affects tobacco, cassava, cotton, red chilli and other cash crops (Jones et al. [26]). Many developing countries are also experiencing damage to tomato crops linked to the virus due to climate change and the very high costs of pesticides to combat the vectors responsible for TYLCV disease (Glick et al. [17]). We can cite as examples of African countries most exposed to TYLCV disease: Egypt, Sudan, Tunisia, Burkina Faso, Nigeria, Senegal, Cabo-Verde, Mali, Ivory Coast, Gambia, Mauritania, and Tanzania (Kashima et al. [28]). In Tanzania a first investigation was carried out in 1993-1994 on a new distinct Gomini tomato, the virus was identified by Akad et al. [1]. TYLCV is transmitted by the insect vector Bemisia Tabaci commonly called silver whitefly (Mehta et al. [33]). The virus is transmitted to adult stages of the whitefly. In general, the female whitefly transmits more than her male. There white whitefly can contract the virus when it feeds, sucks plant juices through infected plant leaves for 14mn-30mn. After a duration of 21h-24h of incubation inside the insect it can transmit the virus to other tomato plants. A period feeding of at least 15mn on new tomato plants is sufficient to retransmit the virus. The whitefly retains the virus for up to 20 days after an acquisition period and loses subsequently the transmission capacity to other factories (Butter et al. [8]). The symptoms develop on young plants after 10 to 14 days (Cruz Vargas-De-Leon et al. [44]), however, disease caused by TYLCV is easily identified when tomato plants are infected at seedling level, specially when infection occurs before the following stage. During this stage the plants are very sensitive to the infection. Whitefly populations decline during heavy rainy seasons, resulting in a regression to infection of tomato plants. Also during the night the whitefly settle on the underside of the leaves of tomato plants. In the spring and summer seasons whitefly populations migrate, leading to a reduction in the spread of TYLCV.

Since the highlighting of epidemiological models, models of fungal diseases have received much attention from researchers. For example (Sabuka et al. [41]) have developed an epidemiological model on which the vectors only make a transient visit to the culture and do not reproduce. They also estimate the parameters of the virus disease case of Tomato Leaf Curl virus (TYLCV) in India. They results showed that the use of the protective net combined with the growth of resistant varieties constitutes a potential for both the reduction of the Bemisia tabaci population and immigration to cultivation which reduces inoculation of the virus by insects that affect the crop (Sabuka et al. [41]). Many researchers have conducted studies on mathematical models of plant disease in particular (Moore et al. [34]), which carried out research on host-vector epidemiological models with direct transmission and considered a delay punctual (Zhou et al. [47]). Then the plant epidemiological model with non-linear and bilinear cases was analyzed by Jeger et al. [22]. While mathematical models of the spread of transmitted diseases by insects linked to climate change was analyzed by Murwayi et al. [35] and BioM conference. A numerical simulation to

understand the behavior of the model was given by Gnakine et al. [19]. Also, mathematical modelling of plants, vector-borne diseases were discussed by Nakazawa et al. [37] and by Rida et al. [39], combining predator-prey models with host models. Vectors to determinate the indirect effects of predators on the dynamics of host vectors. The theory and analysis of plant pathology, as well as information on plant disease outbreaks can be found by Madden et al. [31], moreover the modeling of plant diseases caused by viruses was discussed by Jeger et al. and Jeger et al. [23, 25], as well as the mathematical models of interactions transmitted by soil and microbial antagonists such as controls were carried out by Cunniffe et al. [13]. Amelia et al. in [3] studied a mathematical model of the propagation of the tomato yellow leaf curl virus (TYLCV) of red chilli using Verticillium lecanii and the results were satisfactory, they showed at the end of their work that Verticillium lecanii could be a means of controlling the TYLCV. But they did not take into account the laten stages because they did not take into account the latency period of the transmission of TYLCV. In [5], Atifah et al. studied an SI mathematical model of the spread of tomato yellow leaf curl virus using Verticillium lecanii as a means to control the TYLCV disease. In this model the authors are not take into account the latency period of the spread of tomato yellow leaf curl virus. Speaking of whiteflies that invade many cultures, there are mathematical models Keller Segel models, which better describe the evolution of some species, the passage of single-celled organisms to more complex structures, these are paradigmatic models that observe the propagation of species in space according to the concentration of sites in food for their feeding. This attraction is based on chemotaxis. In [10], the authors studied a nonlinear attraction-repulsion chemotaxis model with a logistic function, applied to the diffusion of dictystelium according to the concentration of sites in food. Li et al. studied in [46] a model of the chemotaxis type of attraction-repulsion with zero flux with linear and superlinear production. It is a model of the Keller Segel type idealizing the phenomena of aggregation in situations where certain populations are attracted by a signal that they absorb themselves and a repulsive model where the populations are repelled.

This present paper is an extension of the work of Amelia et al. [3]. This study uniquely incorporates latent stages of vegetative and generative tomato plants into the mathematical model, offering new insights into disease control dynamics. Taking into account these latency stages is linked to the latency period before the appearance of the symptoms disease on the tomato leaves which is 10 to 14 days (Cruz Vargas-De-Leon et al. [44]). Verticillium lecanii as a control population of Bemisia tabaci. To know the spread of the yellow virus in the tomato plant, other knowledge is necessary to obtain an in-depth mathematical analysis. Indeed the growth function that we used in this paper is a logistic function, this function is considered the most illustrates model of the growth dynamics of organisms in restricted environments compared to linear functions. This function assumes that the growth rate of a given population is proportional to the population size (Busenberg et al. [7]). The epidemiological system in which we were interested is a tomato growing area in which tomato plants and insects pests Bemisia tabaci coexist, defined by a system of first degree ordinary differential equations.

The paper structured in these following points. In Section 2, we proposed a model of TYLCV. In Section 3, we use the theory of uniform persistence to analyze the global behavior of the model. In Section 4, we proceed to determine the disease free equilibrium point in order to compute the basic reproduction number which we use as instruments to predict the evolution of the disease and we study the global stability of the disease free equilibrium point using the Castillo-Chavez theory. In Section 5, we prove that the model has an endemic equilibrium and we also examine its overall stability through a Lyapunov function and the Invariance principle of Lasalle. In Section 6, numerical simulations are provided in order to illustrate our theoretical results. In Section 7, we proposed the study of the sensitivity of certain parameters to observe which have an impact on the basic reproduction number. We end this work by a conclusion.

2. Mathematical modeling of the spread of begomovirus

2.1. Model formulation

We formulate a model of tomato disease dynamics tomato yellow leaf curl virus (TYLCV), following

the ideas of the models presented by Amelia et al. and Atifah et al. [3, 5]. We have taken into account of latent stage to observe its impact on the dynamics of begomovirus transmission in tomato plants because the period of appearance of TYLCV symptoms on tomatoes which corresponds to the latency period of the disease is 10 to 14 days (Cruz Vargas-De-Leon et al. [44]) and populations of vectors in mathematical analysis. Recall that "latent stage refers to the period during which individuals are infected but asymptomatic, a critical factor in understanding disease dynamics". We consider a tomato field initially made up of young plants designated susceptible vegetative plants noted S_v and mature production plants designated susceptible generative plants noted S_g. We note, however, that the susceptible vegetative plant can therefore freely become a susceptible generative plant with a rate of becoming α. Following a submersion of the whiteflies noted S_{BT} in the field. Some wearing begomovirus which is a disease of tomato yellow leaf curl virus (TYLCV), can during their feeding, contaminate both types of plants. Thus the vegetative plants are infected at the β_1 rate by a whiteflies carrying the begomovirus noted I_{BT} and the generative plants are infected at the β_2 by a whiteflies carrying the begomovirus. Taking into account this first infection, vegetative plants can become exposed vegetative plants but not infectious noted L_v at the β_1 passage rate and generative plants can become exposed generative plants noted L_g at the β_2 passage rate. After the incubation period of the virus which is approximately 10 to 14 days (Cruz Vargas-De-Leon et al. [44]), the vegetative plants exposed L_v become an infectious plants noted I_v with a β transfer rate and the exposed generative plants also become infectious generative plants with a θ transition rate. This during the whiteflies susceptible S_{BT}, during the stings of the leaves of tomato plants to feed on the sap can contract the virus at the rates of acquisition γ_1 by the whiteflies S_{BT} to feed on infected vegetative plants I_v and γ_2 by S_{BT} during the sting of the infected generative plants. We manage to note that the tomato plant population consists of susceptible vegetative plants, latent vegetative plants, infected vegetative plants, susceptible generative plants, latent generative plants, infected generative plants which we represent respectively by S_v, L_v, I_v, S_g, L_g, I_g and the population of vectors is made up of susceptible insects and infected insects, which we represent respectively by S_{BT}, I_{BT}. Insect recruitment follows the function of logistics growth rate $r\left(1-\frac{S_{BT}}{K}\right)$, where K is the carrying capacity Bemisia tabaci in the tomato plantation and r being the intrinsic growth rate of Bemisia tabaci. The logistic growth rate assumes that the whiteflies population grows quickly when small but slows down as it approaches the carrying capacity K. We did not take the latent stage into account since 21h-24h is enough to the insect contaminated by infected plant leaves to retransmit the virus by nourishing, by sucking plant juices. A feeding period of at least 15mn on the new tomato host is necessary for virus transmission. Our main objective of this modeling is to introduce a control variable, which is the use of a Verticillium lecanii pesticide to protect or sustainably control plant crops, precisely the tomato yellow leaf curl virus (TYLCV). To this we take for δ_p the efficiency of use of Verticillium lecanii and $1-\delta_p$ as a representation of pesticide inefficiency. After introduction of the pesticide Verticillium lecanii at the δ_p rate to slow down the different infections we observe the transition changes in the two types of populations. $\beta_1(1-\delta_p)$ is the new infection rate to tomato plants during the vegetative phase S_v by an whitefly infected I_{BT} and is influenced by the effectiveness of the use of Verticillium lecanii, to become an exposed vegetative plant and $\beta_2(1-\delta_p)$ is the new infection rate to tomato plants during the vegetative phase S_v by an whitefly infected I_{BT} and is influenced by the effectiveness of the use of Verticillium lecanii, to become an exposed vegetative plant. Also the new infection rate controlled by the effectiveness of the use of Verticillium lecanii to susceptible whiteflies S_{BT} by infected vegetative plants is $\gamma_1(1-\delta_p)$ and the new infection rate controlled by the effectiveness of the use of Verticillium lecanii to susceptible whiteflies S_{BT} by infected generative plants is $\gamma_2(1-\delta_p)$. $\theta_{BT}\delta_p$ is the mortality rate controlled by the use efficiency of the pesticide Verticillium lecanii of the population of susceptible whiteflies and infected whiteflies during the initial instant of homogeneous spraying of all types of tomato plants. These two rates μ_p and μ_{BT} designate respectively the natural mortality rate of the plant for all the different compartments of the tomato plant and of susceptible and infected whiteflies. Thus the dynamics of the mathematical model formulated from the spread of yellow virus of tomato plants includes the following hypothesis.

(H1) Whiteflies exhibit no feeding preference and feed uniformly across all parts of the tomato plant.

(H2) Tomato plants are susceptible to infection (during the vegetative or generative period) in the event of direct contact with an infected insect vector.

(H3) When an individual is infected within both populations remains infected constantly throughout its lifespan, i.e., can not recover.

(H4) The infection rate of the tomato plant is by contact (occurs at time).

(H5) All populations of the tomato plant were sprayed with Verticillium lecanii.

(H6) The infection rate of Tabaci when is an interaction between an infected plant during the vegetative phase is influenced by the effectiveness of the use of Verticillium lecanii.

The flow chart of the spread of yellow virus in tomato plants combined with the using of the insecticide Verticillium lecanii is visible in the Diagram 1, with the definition of the variables and parameters used1 is also given by the Tables 1 and 2.

Table 1: Summary of biological variables.

Variables	Biological significance	Unity
$S_v(t)$	Population of susceptible vegetative tomato plants at time t	Individual plant
$L_v(t)$	Latent vegetative tomato plants population at time t	Individual plant
$I_v(t)$	Infected vegetative tomato plants population at time t	Individual plant
$S_g(t)$	Susceptible generative tomato plants population at time t	Individual plant
$L_g(t)$	Latent generative tomato plants population at time t	Individual plant
$I_g(t)$	Infected generative tomato plants population at time t	Individual plant
$S_{BT}(t)$	Susceptible Bemisia tabaci population at time t	Individual vector
$I_{BT}(t)$	Infected Bemisia tabaci population at time t	Individual vector

Table 2: Summary of biological parameters

Parameters	Biological significance	Unity
N_p	Total population of tomato plants at initial time	Individual plant
r	The insect Bemisia tabaci birth rate	day^{-1}
Λ	The number of tillers or replanting of tomato plants	day^{-1}
α	The growth rate of tomato plants from the vegetative phase to become generative plants	day^{-1}
θ	The growth rate of tomato plants from the latent vegetative phase to become infected vegetative plants	day^{-1}
β	The growth rate of tomato plants from the latent generative phase to become infected generative plants	day^{-1}
β_1	The infection rate of tomato plants during the vegetative phase	$(individual \times day)^{-1}$
β_2	The infection rate of tomato plants during the generative phase	$(individual \times day)^{-1}$
γ_1	The infection rate of Bemisia tabaci when is an interaction between an infected plant during the vegetative phase	
γ_2	The infection rate of Bemisia tabaci when is an interaction between an infected plant during the generative phase	$(individual \times day)^{-1}$
δ_p	The efficacy of use of Verticillium lecanii	
μ_p	The mortality rate of the tomato plant	day^{-1}
μ_{BT}	The natural mortality rate of Bemisia tabaci	day^{-1}
θ_{BT}	The mortality rate of Bemisia tabaci caused by the fungus Verticillium lecanii	day^{-1}

2.2. *Compartmental diagram of the dynamics of yellow virus propagation*

The compartmental diagram Figure 1 illustrates the transitions between susceptible, latent, and infected states for both tomato plants and whiteflies, as well as the effect of control strategies like pesticide application.

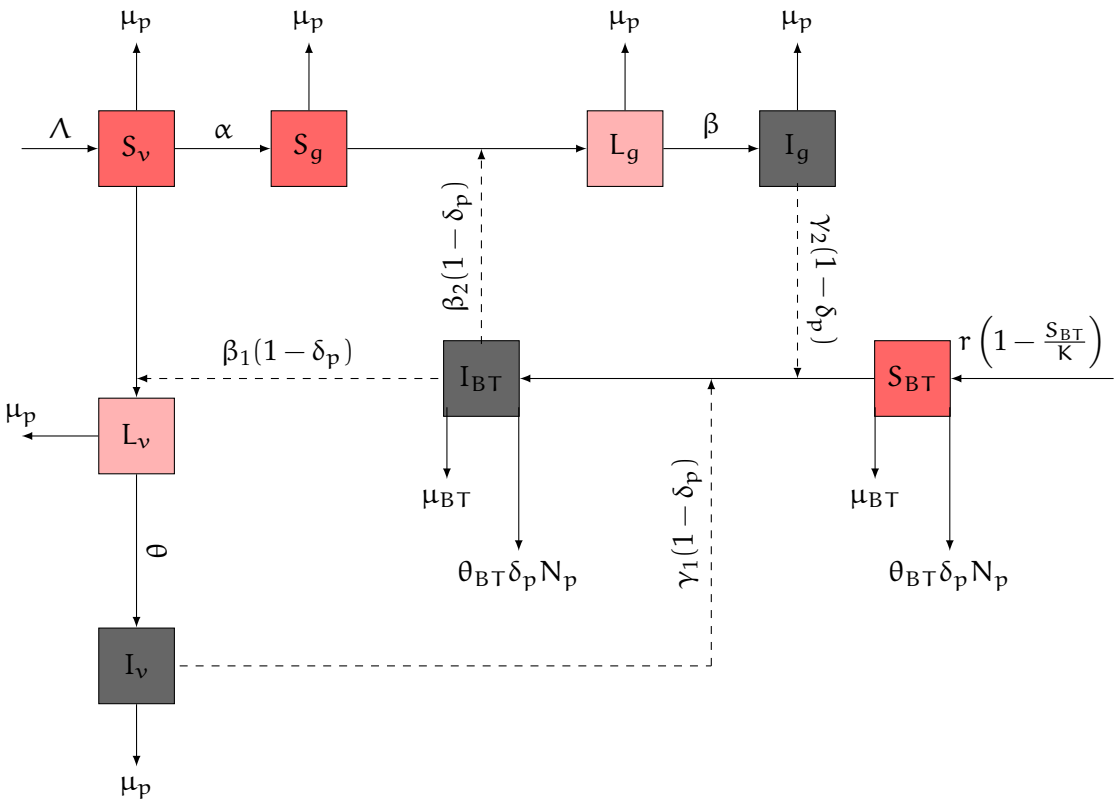

Figure 1: Compartmental diagram for transmission dynamics TYLCV.

2.3. *Mathematical model*

By taking stock of the input-output masses in the different classes suitably to the mathematical formulation, the model of the dynamics of the propagation of the virus host-insect interaction vectors is described by the following system (2.1)

$$\begin{cases} \begin{cases} \dot{S}_v = \Lambda - \alpha S_v - \beta_1(1-\delta_p)S_v I_{BT} - \mu_p S_v, \\ \dot{L}_v = \beta_1(1-\delta_p)S_v I_{BT} - \theta L_v - \mu_p L_v, \\ \dot{I}_v = \theta L_v - \mu_p I_v, \end{cases} \\ \begin{cases} \dot{S}_g = \alpha S_v - \beta_2(1-\delta_p)S_g I_{BT} - \mu_p S_g, \\ \dot{L}_g = \beta_2(1-\delta_p)S_g I_{BT} - \beta L_g - \mu_p L_g, \\ \dot{I}_g = \beta L_g - \mu_p I_g, \end{cases} \\ \begin{cases} \dot{S}_{BT} = r\left(1-\frac{S_{BT}}{K}\right) S_{BT} - \gamma_1(1-\delta_p)I_v S_{BT} - \gamma_2(1-\delta_p)I_g S_{BT} - (\theta_{BT}\delta_p N_p + \mu_{BT})\, S_{BT}, \\ \dot{I}_{BT} = \gamma_1(1-\delta_p)I_v S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT} - (\theta_{BT}\delta_p N_p + \mu_{BT})\, I_{BT}, \end{cases} \end{cases} \tag{2.1}$$

with the initial conditions

$$S_v(0) > 0,\ L_v(0) \geqslant 0,\ I_v(0) \geqslant 0,\ S_g(0) \geqslant 0,\ L_g(0) \geqslant 0,\ I_g(0) \geqslant 0,\ S_{BT}(0) \geqslant 0,\ I_{BT}(0) \geqslant 0. \tag{2.2}$$

We give explanatory details of the differential equations which govern our model (2.1).

The first equation of system (2.1), $\dot{S}_v = \Lambda - \alpha S_v - \beta_1(1-\delta_p)S_v I_{BT} - \mu_p S_v$, describes the dynamics of susceptible vegetative tomato plants. Recruitment into this class is represented by Λ, loss to generative phase by αS_v, and infection by $\beta_1(1-\delta_p)S_v I_{BT}$, which accounts for pesticide efficiency. The second equation of system (2.1), $\dot{L}_v = \beta_1(1-\delta_p)S_v I_{BT} - \theta L_v - \mu_p L_v$, describes the dynamics of latent vegetative tomato plants. $\beta_1(1-\delta_p)S_v I_{BT}$ is the mass of vegetative plants infected but asymptomatic, θL_v is the mass of asymptomatic infected plants towards infectious plants and deaths of asymptomatically infected plants as $\mu_p L_v$. The third equation of system (2.1), $\dot{I}_v = \theta L_v - \mu_p I_v$, describes the dynamics of infected vegetative tomato plants. θL_v correspond to the flux of latent vegetative tomato plants who have become infectious vegetative plants and $\mu_p I_v$ the deaths of the infected vegetative tomato plants. The fourth equation of system (2.1), $\dot{S}_g = \alpha S_v - \beta_2(1-\delta_p)S_g I_{BT} - \mu_p S_g$, describes the dynamics of susceptible generative tomato plants. αS_v is the mass of susceptible vegetative plants who have become generative plants while remaining susceptible, infection by $\beta_2(1-\delta_p)S_g I_{BT}$ and the deaths by the $\mu_p S_g$. The fifth equation of system (2.1), $\dot{L}_g = \beta_2(1-\delta_p)S_g I_{BT} - \beta L_g - \mu_p L_g$, describes the dynamics of latent generative tomato plants. $\beta_2(1-\delta_p)S_g I_{BT}$ is the mass of susceptible generative plants who have become infected generative plants but asymptomatic and the deaths by $\mu_p S_g$. The sixth equation of system (2.1), $\dot{I}_g = \beta L_g - \mu_p I_g$, describes the dynamics of infected generative tomato plants. The news infected generative plants by βL_g and the deaths by $\mu_p I_g$. The seventh equation of system (2.1), $\dot{S}_{BT} = r\left(1-\frac{S_{BT}}{K}\right)S_{BT} - \gamma_1(1-\delta_p)I_v S_{BT} - \gamma_2(1-\delta_p)I_g S_{BT} - \theta_{BT}\delta_p N_p S_{BT} - \mu_{BT} S_{BT}$, describes the dynamics of susceptible whiteflies population. $r\left(1-\frac{S_{BT}}{K}\right)S_{BT}$ is the logistic growth of the whitefly population. The losses of susceptible whiteflies are of two types: infected whitefly population by contact with an infected vegetative plant during their feeding by $\gamma_1(1-\delta_p)I_v S_{BT}$, infected whitefly population by contact with an infected generative plant during their feeding by $\gamma_2(1-\delta_p)I_g S_{BT}$ and deaths from susceptible whiteflies are of two types: deaths linked to pesticide use by $\theta_{BT}\delta_p N_p S_{BT}$ and the natural deaths by $\mu_{BT} S_{BT}$. The eighth equation of system (2.1), $\dot{I}_{BT} = \gamma_1(1-\delta_p)I_v S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT} - \theta_{BT}\delta_p N_p I_{BT} - \mu_{BT} I_{BT}$, describes the dynamics of infected whiteflies population. $\gamma_1(1-\delta_p)I_v S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT}$ is the news whiteflies becoming infected, deaths linked to pesticide use by $\theta_{BT}\delta_p N_p I_{BT}$ and the natural deaths by $\mu_{BT} I_{BT}$.

3. Mathematical analysis of the TYLCV model

In this section, we must prove that the solutions of system (2.1) exists, are positive and bounded for all time (t) that means the TYLCV model has eco-epidemiologically meaningful.

3.1. Existence and uniqueness of solutions

Model system (2.1) describes a system of autonomous first order differential equations, then we can further rewrite this system in the following form:

$$\dot{y}(t) = \varphi(y(t)), \tag{3.1}$$

where $y(t) = (S_v(t), L_v(t), I_v(t), S_g(t), L_g(t), I_g(t), S_{BT}(t), I_{BT}(t))^T$ and φ is a class $\mathscr{C}^\infty$ function of $\mathbb{R}^8$ in $\mathbb{R}^8$, defined by:

$$\varphi(y) = \begin{pmatrix} \Lambda - \alpha S_v - \beta_1(1-\delta_p)S_v I_{BT} - \mu_p S_v \\ \beta_1(1-\delta_p)S_v I_{BT} - \mu_p L_v \\ \theta L_v - \mu_p I_v \\ \alpha S_v - \beta_2(1-\delta_p)S_g I_{BT} - \mu_p S_g \\ \beta_2(1-\delta_p)S_g I_{BT} - \beta L_g - \mu_p L_g \\ \beta L_g - \mu_p I_g \\ r\left(1-\frac{S_{BT}}{K}\right)S_{BT} - \gamma_1(1-\delta_p)I_v S_{BT} - \gamma_2(1-\delta_p)I_g S_{BT} - \theta_{BT}\delta_p N_p S_{BT} - \mu_{BT} S_{BT} \\ \gamma_1(1-\delta_p)I_v S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT} - \theta_{BT}\delta_p N_p I_{BT} - \mu_{BT} I_{BT} \end{pmatrix},$$

with $y = (S_v, L_v, I_v, S_g, L_g, I_g, S_{BT}, I_{BT})^T \in \mathbb{R}^8$.

Theorem 3.1. *For any initial conditions* (2.2)*, the system* (3.1) *admits a unique maximal solution.*

Proof. The function φ associated with system (3.1) is of class $\mathscr{C}^\infty$, then it is also of class $\mathscr{C}^1$. So φ is locally Lipschitzian on $\mathbb{R}^8$ such that $S_v(0) > 0$. Therefore we have existence and uniqueness of the maximal solution of the Cauchy problem associated with the system defined by (3.1) on $[0, T_{max}[$, where $T_{max} > 0$, for any initial condition (2.2) . □

3.2. Non-negativity of solutions

We need to find solution of each equation of the model system (2.1) for testing positivity.
We thus have the following theorem.

Theorem 3.2. *For any initial condition* (2.2) *the maximal solution of the system* (2.1) *is non-negative.*

Proof. Let's proceed absurdly by setting: $t_1 = \sup_{\{\forall t \in [0;T_{max}[\}} \{S_v(u) > 0,\ H(u) \geqslant 0\}$ with

$$H(t) = \min\{ L_v(t),\ I_v(t),\ S_g(t),\ L_g(t),\ I_g(t),\ S_{BT}(t),\ I_{BT}(t)\}.$$

Since the maximal solution exists, t_1 is well posed or is well defined in $\mathbb{R}_+ \cup \{+\infty\}$. If $t_1 = +\infty$ there are done. Assume that $t_1 \in \mathbb{R}_+$, in this case $S_v(t_1) = 0$ or $H(t_1) < 0$.

First case. We assume that $S_v(t_1) = 0$. From the first equation of the system (2.1), we obtain

$$S_v(t) \geqslant S_v(0) \exp\left(\int_0^t [-\beta_1(1-\delta_p)I_{BT}(s) - \alpha - \mu_I]ds\right),$$

$\forall t \in [0, T_{max}[$ or $S_v(0) > 0$ we get $0 = S_v(t_1) \geqslant S_v(0) \exp\left(\int_0^{t_1} [-\beta_1(1-\delta_p)I_{BT}(s) - \alpha - \mu_I]ds\right) > 0$, which is contradiction.

Second case. We assume that $H(t_1) < 0$. If $H(t_1) = L_v(t_1)$, then from the second equation of the system (2.1), we obtain:

$$L_v(t) \geqslant L_v(0) \exp(-(\theta + \mu_p)t),\ \forall t \in [0, T_{max}[, \quad 0 > L_v(t_1) \geqslant 0,$$

which is contradiction. We can also make similar contradictions for the other variables. □

3.3. Boundedness of the solutions

Here, we must show that the maximal solution is bounded in the subset $\Gamma \subset \mathbb{R}^6_+ \times \mathbb{R}^2_+$.
Let us consider the domain Γ defined by $\Gamma = \Gamma_1 \times \Gamma_2 \subset \mathbb{R}^8_+$ with

$$\Gamma_1 = \left\{ (S_v, L_v, I_v, S_g, L_g, I_g)^T \in \mathbb{R}^6_+ \mid S_v + L_v + I_v + S_g + L_g + I_g \leqslant \frac{\Lambda}{\mu_p} \right\},$$
$$\Gamma_2 = \left\{ (S_{BT}, I_{BT})^T \in \mathbb{R}^2_+ \mid S_{BT} + I_{BT} \leqslant \frac{rK}{4\min(\mu_I, \theta_{BT}\delta_p N_p)} \right\}.$$

Theorem 3.3. *The set* Γ *is positively invariant for the system* (2.1)*. Furthermore model* (2.1) *is biologically feasible in the domain* Γ*.*

Proof. The evolution of the system (2.1) is governed by the following system

$$\begin{cases} \dot{S}(t) = \Lambda - \mu_p S(t), \\ \dot{V}(t) = r\left(1 - \frac{S_{BT}(t)}{K}\right) S_{BT}(t) - \theta_{BT}\delta_p N_p V(t) - \mu_{BT} V(t), \end{cases} \tag{3.2}$$

where $S = S_v + L_v + I_v + S_g + L_g + I_g$ and $V = S_{BT} + I_{BT}$. From the first equation of system (3.2) and by applying the formulation of the constant variation between 0 and t, we obtain the following solution

$$S(t) = \frac{\Lambda}{\mu_P} + \left(S(0) - \frac{\Lambda}{\mu_P}\right) e^{-\mu_p t},$$

since $S(0) = S_v(0) + I_v(0) + S_g(0) + L_g(0) + I_g(0) > 0$, then S is continuous, positive, and increasing on $\mathbb{R}^6_+$ and $\lim_{t\to+\infty} S(t) = \frac{\Lambda}{\mu_p}$. Then $0 < S(t) \leqslant \frac{\Lambda}{\mu_p}$, $\forall t \in [0, T_{max}[$. From the last equation of the system (3.2), we get

$$\dot{V}(t) = f(S_{BT}(t)) - \theta_{BT}\delta_p N_p V(t) - \mu_{BT} V(t) \leqslant \frac{rK}{4} - \min(\theta_{BT}\delta_p N_p, \mu_{BT})V(t),$$

where $f(S_{BT}(t)) = r\left(1 - \frac{S_{BT}(t)}{K}\right) S_{BT}(t)$ and with $\sup_{t\in[0,+\infty[} f(S_{BT}(t)) = \frac{rK}{4}$. By the comparison principle we obtain

$$V(t) \leqslant \frac{rK}{4\min(\theta_1\delta_p N_p, \mu_I)}\left(1 - e^{-\min(\theta_{BT}\delta_p N_p, \mu_{BT})t}\right) + V(0)e^{-\min(\theta_{BT}\delta_p N_p, \mu_{BT})t},$$
$$V(t) \leqslant \frac{rK}{4\min(\mu_I, \theta_{BT}\delta_p N_p)}.$$

So $0 \leqslant V(t) \leqslant \frac{rK}{4\min(\mu_I, \theta_{BT}\delta_p N_p)}$, $\forall t \in [0, T_{max}[$. Thus S and V are bounded and $T_{max} = +\infty$. Consequently, all solutions of the system (2.1) with initial conditions remain positively invariant in the compact domain Γ for all $t \geqslant 0$. □

4. Disease free-equilibrium point

The system (2.1) admits two disease-free equilibrium points given by

$$\mathcal{D}^0 = \left(\frac{\Lambda}{\alpha+\mu_p}, 0, 0, \frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}, 0, 0, 0, 0\right),$$
$$\mathcal{D}^1 = \left(\frac{\Lambda}{\alpha+\mu_p}, 0, 0, \frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}, 0, 0, \frac{K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r}, 0\right),$$

for $r > \mu_{BT} + N_p\delta_p\theta_{BT}$. Although we have two equilibrium points without disease but only one of the two is used for determine the basic reproduction number and analysis of the stability of the equilibrium point without disease, in particular the equilibrium point $\mathcal{D}^1$. This choice is justified by the fact that in $\mathcal{D}^0$ the population of Bemisia tabaci is zero, it is not relevant enough because the population of the insect as a vector for spreading the disease does not exist. However, it would be more ingenious to look in the numerical simulation section when there is persistence of endemic equilibrium in what percentage of dose of use of Verticillium lecanii we would be able to return to equilibrium without disease and without the vector TYLCV transmitter, i.e., at DFE $\mathcal{D}^0$.

4.1. Basic reproduction number and local stability of disease-free equilibrium

The basic reproduction number $\mathcal{R}_0$ is very important in epidemiology because it is used to determine the number of secondary infections caused by a primary infection in populations sensitive (Castillo-Chavez et al. [9, 11]). We use the standard next generation matrix method developed by Diekman et al., Heffernan et al., and Van den Driessche et al. [15, 22, 45], where F is the new infection matrix and V is the transfer. In system (2.1), we only consider variables in which the infection is progressing, i.e., L_v, I_v, L_g, Ig, and I_{BT}. The corresponding equations can be rewritten as follows:

$$\dot{Y}_I = F(Y_I) - V(Y_I).$$

Let $Y_I = (L_v, I_v, L_g, I_g, I_{BT})$ be an infection state describes new infections arising in state Y_I and $V(Y_I)$ the terms of transfers between compartments or outside compartments:

$$F(Y_I) = \begin{pmatrix} \beta_1(1-\delta_p)S_v I_{BT} \\ 0 \\ \beta_2(1-\delta_p)S_g I_{BT} \\ 0 \\ \gamma_1(1-\delta_p)I_v S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT} \end{pmatrix} \quad \text{and} \quad V(Y_I) = \begin{pmatrix} (\theta+\mu_p)L_v \\ -\theta L_v + \mu_p I_v \\ (\beta+\mu_p)L_g \\ -\beta L_g + \mu_p I_g \\ \theta_{BT}\delta_p N_p I_{BT} + \mu_{BT} I_{BT} \end{pmatrix}.$$

The Jacobian matrix R of F and H of V evaluated in $\mathcal{D}^1$ are, respectively,

$$R = \begin{pmatrix} 0 & 0 & 0 & 0 & \beta_1(1-\delta_p)\frac{\Lambda}{\alpha+\mu_p} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_2(1-\delta_p)\frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\gamma_1(1-\delta_p)K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r} & 0 & \frac{\gamma_2(1-\delta_p)K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r} & 0 \end{pmatrix}$$

and

$$H = \begin{pmatrix} \theta+\mu_p & 0 & 0 & 0 & 0 \\ -\theta & \mu_p & 0 & 0 & 0 \\ 0 & 0 & \beta+\mu_p & 0 & 0 \\ 0 & 0 & -\beta & \mu_p & 0 \\ 0 & 0 & 0 & 0 & \theta_{BT}\delta_p N_p + \mu_{BT} \end{pmatrix},$$

$$H^{-1} = \begin{pmatrix} \frac{1}{\theta+\mu_p} & 0 & 0 & 0 & 0 \\ \frac{\theta}{\mu_p(\theta+\mu_p)} & \frac{1}{\mu_p} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\beta+\mu_p} & 0 & 0 \\ 0 & 0 & \frac{\beta}{\mu_p(\beta+\mu_p)} & \frac{1}{\mu_p} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\theta_{BT}\delta_p N_p+\mu_{BT}} \end{pmatrix}.$$

The next generation matrix of system (2.1) is given by

$$M = RH^{-1} = \begin{pmatrix} 0 & 0 & 0 & 0 & M_{15} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & M_{35} \\ 0 & 0 & 0 & 0 & 0 \\ M_{51} & M_{52} & M_{53} & M_{54} & 0 \end{pmatrix}$$

with

$$M_{15} = \frac{\beta_1(1-\delta_p)\Lambda}{(\alpha+\mu_p)(\theta_{BT}\delta_p N_p+\mu_{BT})}, \qquad M_{35} = \frac{\beta_2(1-\delta_p)\alpha\Lambda}{\mu_p(\alpha+\mu_p)(\theta_{BT}\delta_p N_p+\mu_{BT})},$$

$$M_{51} = \frac{K\gamma_1\theta(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r\mu_p(\theta+\mu_p)}, \qquad M_{52} = \frac{K\gamma_1(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{\mu_p r},$$

$$M_{53} = \frac{K\gamma_2\beta(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r\mu_p(\beta+\mu_p)}, \qquad M_{54} = \frac{K\gamma_2(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r\mu_p}.$$

The eigenvalues for the matrix M are $0, \sqrt{M_{15} \times M_{51} + M_{35} \times M_{53}}, -\sqrt{M_{15} \times M_{51} + M_{35} \times M_{53}}$. The basic reproduction rate of the system (2.1) given by the dominant eigenvalue of RH^{-1}, is

$$\mathcal{R}_0 = (1-\delta_p)\sqrt{\frac{K\Lambda(r-\mu_{BT}-N_p\delta_p\theta_{BT})[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]}{r\mu_p^2(\alpha+\mu_p)(\theta+\mu_p)(\beta+\mu_p)(\theta_{BT}\delta_pN_p+\mu_{BT})}}.$$

Choosing H for host and V for vector. We let $\mathcal{R}_{0H^vV} = \frac{\beta_1(1-\delta_p)\Lambda}{(\alpha+\mu_p)(\theta_{BT}\delta_pN_p+\mu_{BT})}$ the number of tomato vegetative plants phase infected by one infectious Bemisia tabaci vectors over its infection period; $\mathcal{R}_{0H^gV} = \frac{\beta_2(1-\delta_p)\alpha\Lambda}{\mu_p(\alpha+\mu_p)(\theta_{BT}\delta_pN_p+\mu_{BT})}$ the number of tomato generative plants phase infected by one infectious Bemisia tabaci vectors over its infection period; $\mathcal{R}_{0VH^v} = \frac{K\gamma_1(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{\mu_pr}$ the number of Bemisia tabaci vectors infected by one infectious tomato vegetative plants phase over its infection period; and $\mathcal{R}_{0VH^g} = \frac{K\gamma_2\beta(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r\mu_p(\beta+\mu_p)}$ the number of Bemisia tabaci vectors infected by one infectious tomato generative plants phase over its infection period, $\mathcal{R}_0$ can be written as

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_{0H^vV} \times \mathcal{R}_{0VH^v} + \mathcal{R}_{0H^gV} \times \mathcal{R}_{0VH^g}}.$$

4.2. Globally stability of the disease-free equilibrium point $\mathcal{D}^1$

In this work we study the global asymptotic stability of the disease-free equilibrium using the theorem of Castillo-Chavez et al. [11]. Let's rewrite our model and we list two conditions, if they are met they will guarantee the global asymptotic stability of disease-free equilibrium.

$$\begin{cases} \dot{X} = F(X,Y), \\ \dot{Y} = G(X,Y),\ G(X,0) = 0, \end{cases} \tag{4.1}$$

where $X = (S_v,\ S_g,\ S_{BT}) \in \mathbb{R}^3_+$ is the vector representing the stage of the different compartments of sensitive plants and insects and the vector $Y = (L_v, I_v, L_g, I_g, I_{BT}) \in \mathbb{R}^5_+$ represents the stage of compartments of the different plants and insects carrying the diseases (plants and latent insects and infected). Let's $\mathcal{D}^1 = (X^*, 0)$ represents the disease-free equilibrium point. The two conditions below guarantee global asymptotic stability of $\mathcal{D}^1$.

(i) $\dot{X} = F(X,0)$, X^* is globally asymptotically stable;

$$X^* = \left(\frac{\Lambda}{\alpha+\mu_p}, \frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}, \frac{K(r-\mu_I-N_p\delta_p\theta_1)}{r}\right).$$

(ii) $G(X,Y) = D_YG(X^*,0)Y - \widehat{G}(X,Y)$, $\widehat{G}(X,Y) \geqslant 0$, for $(X,Y) \in \Gamma$,

where $D_YG(X^*,0)$ is a Metzler Matrix(the diagonal elements are non-negative) and is also the Jacobian of $G(X,Y)$ taken in $(L_v, I_v, L_g, I_g, I_{BT})$ and evaluated in

$$\mathcal{D}^1 = (X^*,0) = \left(\frac{\Lambda}{\alpha+\mu_p}, \frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}, \frac{K(r-\mu_I-N_p\delta_p\theta_1)}{r}, 0, 0, 0, 0, 0\right).$$

If system (4.1) satisfies the above conditions, then according to Castillo-Chavez [11].

Theorem 4.1. *The equilibrium point $\mathcal{D}^1$ of the system (2.1) is globally asymptotically stable in Γ when $\mathcal{R}_0 < 1$.*

Proof. We begin our proof by defining new variables and by dividing the system into sub-systems $X = (S_v, S_g, S_{BT})$ and $Y = (L_v, I_v, L_g, I_g, I_{BT})$ with two vector-valued functions $G(X,Y)$ and $F(X,Y)$ given by

$$F(X,Y) = \begin{pmatrix} \Lambda - \alpha S_v - \beta_1(1-\delta_p)S_vI_{BT} - \mu_pS_v \\ \alpha S_v - \beta_2(1-\delta_p)S_gI_{BT} - \mu_pS_g \\ r\left(1-\frac{S_{BT}}{K}\right)S_{BT} - \gamma_1(1-\delta_p)I_vS_{BT} - \gamma_2(1-\delta_p)I_gS_{BT} - \theta_{BT}\delta_pN_pS_{BT} - \mu_{BT}S_{BT} \end{pmatrix},$$

$$G(X,Y)=\begin{pmatrix}\beta_1(1-\delta_p)S_\nu I_{BT}-\mu_p L_\nu\\ \theta L_\nu-\mu_p I_\nu\\ \beta_2(1-\delta_p)S_g I_{BT}-\beta L_g-\mu_p L_g\\ \beta L_g-\mu_p I_g\\ \gamma_1(1-\delta_p)I_\nu S_{BT}+\gamma_2(1-\delta_p)I_g S_{BT}-\theta_{BT}\delta_p N_p I_{BT}-\mu_{BT}I_{BT}\end{pmatrix}.$$

Now we consider the reduced system $F(X,0)$ defined by

$$\begin{cases}\dot{S}_\nu=\Lambda-(\alpha+\mu_p)S_\nu,\\ \dot{S}_g=\alpha S_\nu-\mu_p S_g,\\ \dot{S}_{BT}=r\left(1-\frac{S_{BT}}{K}\right)S_{BT}-(\theta_{BT}\delta_p N_p+\mu_{BT})S_{BT}.\end{cases}\tag{4.2}$$

Now we can explicitly solve the three equations of system (4.2) and arrived at explicit solutions

$$\begin{aligned}S_\nu(t)&=\frac{\Lambda}{\alpha+\mu_p}+\left(S_\nu(0)-\frac{\Lambda}{\alpha+\mu_p}\right)e^{-(\alpha+\mu_p)t},\\ S_g(t)&=\frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}+\left(S_g(0)-\frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}\right)e^{-(\alpha+\mu_p)t},\\ S_{BT}(t)&=\frac{K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r-cste e^{-(r-\mu_{BT}-N_p\delta_p\theta_{BT})t}},\ cste>0,\end{aligned}$$

where $cste$ is an constant. When t tends to infinity we obtain the equilibrium point X^*. We note that this asymptotic dynamics is independent of the initial conditions in Γ, therefore the convergence of the solutions of the reduced system (4.2) is global in Γ. We now calculate $G(X,Y)=D_Y G(X^*,0)Y-\widehat{G}(X,Y)$ and show that $\widehat{G}(X,Y)\geqslant 0$.

$$D_Y G(X^*,0)=\begin{pmatrix}-\mu_p & 0 & 0 & 0 & \beta_1(1-\delta_p)\frac{\Lambda}{\alpha+\mu_p}\\ \theta & -\mu_p & 0 & 0 & 0\\ 0 & 0 & -(\beta+\mu_p) & 0 & \beta_2(1-\delta_p)\frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}\\ 0 & 0 & \beta & -\mu_p & 0\\ 0 & T_{52} & 0 & T_{54} & -(\theta_{BT}\delta_p N_p+\mu_{BT})\end{pmatrix},$$

$$T_{52}=\frac{\gamma_1(1-\delta_p)K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r},\ T_{54}=\frac{\gamma_2(1-\delta_p)K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r},$$

and

$$\widehat{G}(X,Y)=(1-\delta_p)\begin{pmatrix}\beta_1\left(\frac{\Lambda}{\alpha+\mu_p}-S_\nu\right)I_{BT}\\ 0\\ \beta_2\left(\frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}-S_g\right)I_{BT}\\ 0\\ \gamma_1\left(\frac{K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r}-S_{BT}\right)I_\nu+\gamma_2\left(\frac{K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r}-S_{BT}\right)I_g\end{pmatrix}.$$

Let's consider this following system:

$$\begin{cases}\dot{S}_\nu=\Lambda-\alpha S_\nu-\beta_1(1-\delta_p)S_\nu I_{BT}-\mu_p S_\nu,\\ \dot{S}_g=\alpha S_\nu-\beta_2(1-\delta_p)S_g I_{BT}-\mu_p S_g,\\ \dot{S}_{BT}=r\left(1-\frac{S_{BT}}{K}\right)S_{BT}-\gamma_1(1-\delta_p)I_\nu S_{BT}-\gamma_2(1-\delta_p)I_g S_{BT}-\theta_{BT}\delta_p N_p S_{BT}-\mu_{BT}S_{BT}.\end{cases}\tag{4.3}$$

Since the positivity of the solutions of the system (3.1), permits us to obtain these three following inequalities according of system (4.3),

$$\dot{S}_\nu\leqslant\Lambda-\alpha S_\nu-\mu_p S_\nu,\quad \dot{S}_g\leqslant\alpha S_\nu-\mu_p S_g,\quad \dot{S}_{BT}\leqslant r\left(1-\frac{S_{BT}}{K}\right)S_{BT}-\theta_{BT}\delta_p N_p S_{BT}-\mu_{BT}S_{BT}.$$

Solving the last three inequalities by separation of variable principle and using the standard comparison principle, we obtain

$$\limsup_{t\to+\infty} S_v \leqslant \frac{\Lambda}{\alpha+\mu_p}, \qquad \limsup_{t\to+\infty} S_g \leqslant \frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}, \qquad \limsup_{t\to+\infty} S_{BT} \leqslant \frac{K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r}.$$

Then we show that $0 < S_v \leqslant \frac{\Lambda}{\alpha+\mu_p}$, $0 \leqslant S_g \leqslant \frac{\alpha\Lambda}{\mu_p(\alpha+\mu_p)}$ and when $r > u_{BT} + N_p\delta_p\theta_{BT}$ implies $0 \leqslant S_{BT} \leqslant \frac{K(r-\mu_{BT}-N_p\delta_p\theta_{BT})}{r}$. Therefore $\widehat{G}(X,Y) \geqslant 0$, $\forall (X,Y) \in \Gamma$. The two conditions of Castillo-Chavez et al. are verified, then the DFE equilibrium point $\mathcal{D}^1$ of the system (2.1) is globally asymptotically stable in Γ when $\mathcal{R}_0 < 1$. □

5. Existence and global stability of the endemic equilibrium

5.1. Existence of endemic equilibrium point

Theorem 5.1. *The system (2.1) admits at least one positive endemic equilibrium point* $\mathcal{D} = (S_v^*, L_v^*, I_v^*, S_g^*, L_g^*, I_g^*, S_{BT}^*, I_{BT}^*)$ *whenever* $\mathcal{R}_0 > 1$.

Proof. By setting The second members of each equation of system (2.1) to zero, we obtain

$$S_v^* = \frac{\Lambda}{\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*}, \tag{5.1}$$

$$L_v^* = \frac{\beta_1(1-\delta_p)\Lambda I_{BT}^*}{(\theta+\mu_p)(\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*)}, \tag{5.2}$$

$$I_v^* = \frac{\theta\beta_1(1-\delta_p)\Lambda I_{BT}^*}{\mu_p(\theta+\mu_p)(\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*)}, \tag{5.3}$$

$$S_g^* = \frac{\alpha\Lambda}{(\mu_p+(1-\delta_p)\beta_2 I_{BT}^*)(\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*)}, \tag{5.4}$$

$$L_g^* = \frac{\beta_2(1-\delta_p)\alpha\Lambda I_{BT}^*}{(\mu_p+\beta)(\mu_p+(1-\delta_p)\beta_2 I_{BT}^*)(\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*)}, \tag{5.5}$$

$$I_g^* = \frac{\beta_2(1-\delta_p)\beta\alpha\Lambda I_{BT}^*}{\mu_p(\mu_p+\beta)(\mu_p+(1-\delta_p)\beta_2 I_{BT}^*)(\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*)}, \tag{5.6}$$

$$S_{BT}^* = -\frac{K(c_0+c_1 I_{BT}^*+c_2(I_{BT}^*)^2)}{r\mu_p(\mu_p+\theta)(\mu_p+\beta)(\mu_p+(1-\delta_p)\beta_2 I_{BT}^*)(\alpha+\mu_p+(1-\delta_p)\beta_1 I_{BT}^*)}, \tag{5.7}$$

where

$$\begin{aligned}
c_0 &= \mu_p^2(\theta+\mu_p)(\beta+\mu_p)(\alpha+\mu_p)(\theta_{BT}\delta_p N_p+\mu_{BT}-r),\\
c_1 &= \mu_p(\theta+\mu_p)(\beta+\mu_p)(1-\delta_p)(\mu_p\beta_1+\beta_2(\alpha+\mu_p))(\theta_{BT}\delta_p N_p+\mu_{BT}-r)\\
&\quad +(1-\delta_p)^2\Lambda[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)],\\
c_2 &= \mu_p(\theta+\mu_p)(\beta+\mu_p)(\mu_p+\alpha)(1-\delta_p)^2(\theta_{BT}\delta_p N_p+\mu_{BT}-r)\beta_1\beta_2+\Lambda(1-\delta)^3(\beta+\mu_p)\gamma_1\theta\beta_1\beta_2.
\end{aligned}$$

By substituting the three equations (5.3), (5.6), and (5.7) into eighth equation of the system (2.1) we obtain

$$\mathcal{H}(I_{BT}^*) = \lambda_4(I_{BT}^*)^4+\lambda_3(I_{BT}^*)^3+\lambda_2(I_{BT}^*)^2+\lambda_1(I_{BT}^*)+\lambda_0, \tag{5.8}$$

where

$$\begin{aligned}
\lambda_4 &= -r\mu_p^2(1-\delta_p)^4(\theta+\mu_p)^2(\beta+\mu_p)^2(\theta_{BT}\delta_p N_p+\mu_{BT})\beta_1^2\beta_2^2 < 0,\\
\lambda_3 &= -r2\mu_p^2(1-\delta_p)^3(\theta+\mu_p)^2(\beta+\mu_p)^2(\mu_p\beta_1+\beta_2(\alpha+\mu_p))(\theta_{BT}\delta_p N_p+\mu_{BT})\beta_1\beta_2
\end{aligned}$$

$$
\begin{aligned}
&- K\Lambda(1-\delta_p)^3\gamma_1\theta\beta_1\beta_2(\beta+\mu_p)c_2\\
\lambda_2 &= -r\mu_p^2(1-\delta_p)^2(\theta+\mu_p)^2(\beta+\mu_p)^2(\theta_{BT}\delta_p N_p+\mu_{BT})(\mu_p\beta_1\beta_2+(\mu_p\beta_1+\beta_2(\alpha+\mu_p)^2)\\
&- K\Lambda(1-\delta_p)^3\gamma_1\theta\beta_1\beta_2(\beta+\mu_p)c_1 - K(1-\delta_p)^2\Lambda[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]c_2,\\
\lambda_1 &= -r\mu_p^3(1-\delta_p)(\theta+\mu_p)^2(\beta+\mu_p)^2(\alpha+\mu_p)(\theta_{BT}\delta_p N_p+\mu_{BT})(\mu_p\beta_1+\beta_2(\alpha+\mu_p))\\
&- K\Lambda(1-\delta_p)^3\gamma_1\theta\beta_1\beta_2(\beta+\mu_p)c_0 - K(1-\delta_p)^2\Lambda[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]c_1,\\
\lambda_0 &= -K(1-\delta_p)^2\Lambda[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]\mu_p^2(\theta+\mu_p)(\beta+\mu_p)(\alpha+\mu_p)(\theta_{BT}\delta_p N_p\\
&+\mu_{BT}-r) - r\mu_p^4(\theta+\mu_p)^2(\beta+\mu_p)^2(\alpha+\mu_p)^2(\theta_{BT}\delta_p N_p+\mu_{BT}),\\
&= K(1-\delta_p)^2\Lambda(r-\theta_{BT}\delta_p N_p+\mu_{BT})[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]\mu_p^2(\theta+\mu_p)(\beta+\mu_p)\\
&\times(\alpha+\mu_p) - r\mu_p^4(\theta+\mu_p)^2(\beta+\mu_p)^2(\alpha+\mu_p)^2(\theta_{BT}\delta_p N_p+\mu_{BT})\\
&= (\mathcal{R}_0^2-1)r\mu_p^4(\theta+\mu_p)^2(\beta+\mu_p)^2(\alpha+\mu_p)^2(\theta_{BT}\delta_p N_p+\mu_{BT}).
\end{aligned}
$$

Now, using the Descartes sign rules (Bruce Anderson et al. [4]) for $\mathcal{H}(I^*_{BT})$-polynomial see (5.8). The number of positive solutions is given in Table 3. A positive value for $\mathcal{H}(I^*_{BT})$ is not sufficient to obtain an

Table 3: Descartes rule for $\mathcal{H}(I^*_{BT})$-polynomial.

λ_4	λ_3	λ_2	λ_1	λ_0	$\mathcal{R}_0$	number of positive solutions
−	−	−	−	−		0
−	+	−	−	−		0 or 2
−	−	+	−	−		0 or 2
−	−	−	+	−	$\mathcal{R}_0<1$	0 or 2
−	+	+	−	−		0 or 2
−	+	−	+	−		2 or 4
−	−	+	+	−		1 or 2
−	+	+	+	−		0 or 2
−	−	−	−	+		1
−	+	−	−	+		1 or 3
−	−	+	−	+		1 or 3
−	−	−	+	+	$\mathcal{R}_0>1$	1
−	+	+	−	+		1 or 3
−	+	−	+	+		1 or 3
−	−	+	+	+		1
−	+	+	+	+		1

endemic equilibrium of system (2.1). We also need to verify if the corresponding value of I^*_{BT}, determined from equation (5.8), is positive. If both $\mathcal{H}(I^*_{BT})$ and I^*_{BT} are positive, according to equation (5.1)-(5.7), all state variables are positive, so it corresponds to an endemic equilibrium. □

5.2. Global stability of the endemic equilibrium

Theorem 5.2. *Assume that the solution of the system (2.1) satisfies one of those hypotheses*

$$S^*_v \leqslant S_v,\ I^*_v \leqslant I_v,\ S_g \leqslant S^*_g,\ I^*_g \leqslant I_g,\ S^*_{BT} \leqslant S_{BT},\ I_{BT} \leqslant I^*_{BT}. \tag{H8}$$

In that case, all positive endemic equilibrium points $\mathcal{D} = (S^*_v, L^*_v, I^*_v, S^*_g, L^*_g, I^*_g, S^*_{BT}, I^*_{BT})$ of the system (2.1) are globally asymptotically stable whenever $\mathcal{R}_0 > 1$.

Proof. Theorem (5.2) can be proved by using a Lyapunov function. We adopt the Lyapunov function used in (Cruz Vargas-De-Leon et al., Guo et al., Lenhart et al., and Seal et al. [12, 18, 20, 30, 42]). Under

equations of the system (2.1), if the system (2.1) meets $\mathcal{D}$ (Phongchai Jittamai1 et al. [38]), then we obtain

$$\begin{cases} \Lambda = \beta_1(1-\delta_p)S_v^*I_{BT}^* + (\alpha+\mu_p)S_v^*, \\ (\theta+\mu_p) = \beta_1(1-\delta_p)\frac{S_v^*I_{BT}^*}{L_v^*}, \\ \mu_p = \theta\frac{L_v^*}{I_v^*}, \\ \mu_p = \alpha\frac{S_v^*}{S_g^*} - \beta_2(1-\delta_p)I_{BT}^*, \\ (\beta+\mu_p) = \beta_2(1-\delta_p)\frac{S_g^*I_{BT}^*}{L_g^*}, \\ \mu_p = \beta\frac{L_g^*}{I_g^*}, \\ (\theta_{BT}\delta_p N_p + \mu_{BT}) = \gamma_1(1-\delta_p)I_v^* + \gamma_2(1-\delta_p)I_g^* - r\left(1-\frac{S_{BT}^*}{K}\right), \\ (\theta_{BT}\delta_p N_p + \mu_{BT}) = \frac{\gamma_1(1-\delta_p)I_v^*S_{BT}^*}{I_{BT}^*} + \frac{\gamma_2(1-\delta_p)I_g^*S_{BT}^*}{I_{BT}^*}. \end{cases} \tag{5.9}$$

Either Φ the function defined on $\mathbb{R}_{++}$ by: $\Phi(\xi) = \xi - 1 - \ln\xi$. The function Φ is positive for all $\xi \in \mathbb{R}_{++}$. Consider the Lyapunov candidate function $\mathcal{G}_\xi$ defined by

$$\mathcal{G}_\xi = \sum_{\xi,\ \xi^*\in\mathbb{R}_{++}} \xi^*\Phi\left(\frac{\xi}{\xi^*}\right),$$

where $\xi = S_v, L_v, I_v, S_g, L_g, I_g, S_{BT}, I_{BT}$ and $\xi^* = S_v^*, L_v^*, I_v^*, S_g^*, L_g^*, I_g^*, S_{BT}^*, I_{BT}^*$. The time derivative of $\mathcal{G}_\xi$ computed along solutions of the system (2.1) is given by Let's calculate

$$\dot{\mathcal{G}}_{S_v} = \left(1-\frac{S_v^*}{S_v}\right)\dot{S}_v = \left(1-\frac{S_v^*}{S_v}\right)(\Lambda - \alpha S_v - \beta_1(1-\delta_p)S_vI_{BT} - \mu_pS_v).$$

Using the first equation of the system (5.9) we have

$$\begin{aligned} \dot{\mathcal{G}}_{S_v} &= \left(1-\frac{S_v^*}{S_v}\right)(\beta_1(1-\delta_p)S_v^*I_{BT}^* + (\alpha+\mu_p)S_v^* - \beta_1(1-\delta_p)S_vI_{BT} - (\alpha+\mu_p)S_v) \\ &= -(\alpha+\mu_p)\frac{(S_v-S_v^*)^2}{S_v} + \left(1-\frac{S_v^*}{S_v}\right)(\beta_1(1-\delta_p)S_v^*I_{BT}^* - \beta_1(1-\delta_p)S_vI_{BT}) \\ &= -(\alpha+\mu_p)\frac{(S_v-S_v^*)^2}{S_v} + \beta_1(1-\delta_p)\left(1-\frac{S_v^*}{S_v}\right)(S_v^*I_{BT}^* - S_vI_{BT}) \\ &= -(\alpha+\mu_p)\frac{(S_v-S_v^*)^2}{S_v} + \beta_1(1-\delta_p)\left(S_v^*I_{BT}^* - S_vI_{BT} - \frac{S_v^*}{S_v}S_v^*I_{BT}^* + S_vI_{BT}\frac{S_v^*}{S_v}\right) \\ &= -(\alpha+\mu_p)\frac{(S_v-S_v^*)^2}{S_v} + \beta_1(1-\delta_p)S_v^*I_{BT}^*\left(1-\frac{S_vI_{BT}}{S_v^*I_{BT}^*} - \frac{S_v^*}{S_v} + \frac{I_{BT}}{I_{BT}^*}\right). \end{aligned}$$

Let's calculate $\dot{\mathcal{G}}_{L_v}$

$$\dot{\mathcal{G}}_{L_v} = \left(1-\frac{L_v^*}{L_v}\right)\dot{L}_v = \left(1-\frac{L_v^*}{L_v}\right)(\beta_1(1-\delta_p)S_vI_{BT} - (\theta+\mu_p)L_v).$$

Using the second equation of the system (5.9) we have

$$\begin{aligned} \dot{\mathcal{G}}_{L_v} &= \left(1-\frac{L_v^*}{L_v}\right)\left(\beta_1(1-\delta_p)S_vI_{BT} - L_v\left(\beta_1(1-\delta_p)\frac{S_v^*I_{BT}^*}{L_v^*}\right)\right) \\ &= \beta_1(1-\delta_p)\left(1-\frac{L_v^*}{L_v}\right)\left(S_vI_{BT} - L_v\frac{S_v^*I_{BT}^*}{L_v^*}\right) \\ &= \beta_1(1-\delta_p)\left(S_vI_{BT} - L_v\frac{S_v^*I_{BT}^*}{L_v^*} - S_vI_{BT}\frac{L_v^*}{L_v} + \frac{L_v^*}{L_v}L_v\frac{S_v^*I_{BT}^*}{L_v^*}\right) \end{aligned}$$

$$= \beta_1(1-\delta_p)S_\nu^* I_{BT}^* \left(1 + \frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*} - \frac{L_\nu}{L_\nu^*} - \frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*}\right).$$

Let's calculate $\dot{\mathcal{G}}_{S_\nu} + \dot{\mathcal{G}}_{L_\nu}$:

$$\dot{\mathcal{G}}_{S_\nu} + \dot{\mathcal{G}}_{L_\nu} = -(\alpha+\mu_p)\frac{(S_\nu - S_\nu^*)^2}{S_\nu} + \beta_1(1-\delta_p)S_\nu^* I_{BT}^* \left(2 - \frac{S_\nu^*}{S_\nu} - \frac{L_\nu}{L_\nu^*} - \frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*} + \frac{I_{BT}}{I_{BT}^*}\right).$$

Let's now set: $\rho_1 = 2 - \frac{S_\nu^*}{S_\nu} - \frac{L_\nu}{L_\nu^*} - \frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*} + \frac{I_{BT}}{I_{BT}^*}$ and by addition by subtracting ρ_1 to $1 + \ln\frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*}$, we have

$$\begin{aligned}
\rho_1 &= -\frac{S_\nu^*}{S_\nu} + 1 + \ln\frac{S_\nu}{S_\nu^*} - \frac{L_\nu}{L_\nu^*} + 1 + \ln\frac{L_\nu}{L_\nu^*} + \frac{I_{BT}}{I_{BT}^*} - 1 - \ln\frac{I_{BT}}{I_{BT}^*} - \frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*} + 1 + \ln\frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*} \\
\rho_1 &= -\Phi\left(\frac{S_\nu^*}{S_\nu}\right) - \Phi\left(\frac{L_\nu}{L_\nu^*}\right) + \Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) - \Phi\left(\frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*}\right). \qquad (5.10)
\end{aligned}$$

Let's calculate $\dot{\mathcal{G}}_{I_\nu}$:

$$\dot{\mathcal{G}}_{I_\nu} = \left(1 - \frac{I_\nu^*}{I_\nu}\right)\dot{I}_\nu = \left(1 - \frac{I_\nu^*}{I_\nu}\right)(\theta L_\nu - \mu_p I_\nu).$$

Using the third equation of the system (5.9) we have

$$\begin{aligned}
\dot{\mathcal{G}}_{I_\nu} = \left(1 - \frac{I_\nu^*}{I_\nu}\right)(\theta L_\nu - \mu_p I_\nu) &= \theta\left(1 - \frac{I_\nu^*}{I_\nu}\right)\left(L_\nu - I_\nu\frac{L_\nu^*}{I_\nu^*}\right) \\
&= \theta\left(L_\nu - I_\nu\frac{L_\nu^*}{I_\nu^*} - L_\nu\frac{I_\nu^*}{I_\nu} + L_\nu^*\right) = \theta L_\nu^*\left(1 - \frac{I_\nu}{I_\nu^*} + \frac{L_\nu}{L_\nu^*} - \frac{L_\nu}{L_\nu^*}\frac{I_\nu^*}{I_\nu}\right).
\end{aligned}$$

Let's now set $\rho_2 = 1 - \frac{I_\nu}{I_\nu^*} + \frac{L_\nu}{L_\nu^*} - \frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*}$ and by addition by subtracting ρ_2 to $1 + \ln\frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*}$, we have:

$$\begin{aligned}
\rho_2 &= -\frac{I_\nu}{I_\nu^*} + 1 + \ln\frac{I_\nu}{I_\nu^*} - \frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*} + 1 + \ln\frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*} + \frac{L_\nu}{L_\nu^*} - 1 - \ln\frac{L_\nu}{L_\nu^*} \\
\rho_2 &= -\Phi\left(\frac{I_\nu}{I_\nu^*}\right) - \Phi\left(\frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*}\right) + \Phi\left(\frac{L_\nu}{L_\nu^*}\right). \qquad (5.11)
\end{aligned}$$

Let's calculate $\dot{\mathcal{G}}_{S_g}$:

$$\dot{\mathcal{G}}_{S_g} = \left(1 - \frac{S_g^*}{S_g}\right)\dot{S}_g = \left(1 - \frac{S_g^*}{S_g}\right)(\alpha S_\nu - \beta_2(1-\delta_p)S_g I_{BT} - \mu_p S_g).$$

Using the fourth equation of the system (5.9) we have

$$\begin{aligned}
\dot{\mathcal{G}}_{S_g} &= \left(1 - \frac{S_g^*}{S_g}\right)\left(\alpha S_\nu - \beta_2(1-\delta_p)S_g I_{BT} - S_g\left(\alpha\frac{S_\nu^*}{S_g^*} - \beta_2(1-\delta_p)\frac{S_g^* I_{BT}^*}{S_g^*}\right)\right) \\
&= \alpha\left(1 - \frac{S_g^*}{S_g}\right)\left(S_\nu - S_g\frac{S_\nu^*}{S_g^*}\right) + \beta_2(1-\delta_p)\left(1 - \frac{S_g^*}{S_g}\right)\left(S_g\frac{S_g^* I_{BT}^*}{S_g^*} - S_g I_{BT}\right) \\
&= \alpha\left(S_\nu - S_g\frac{S_\nu^*}{S_g^*} - \frac{S_g^*}{S_g}S_\nu + S_\nu^*\right) + \beta_2(1-\delta_p)\left(S_g I_{BT}^* - S_g I_{BT} - S_g^* I_{BT}^* + S_g^* I_{BT}\right) \\
&= \alpha S_\nu^*\left(1 + \frac{S_\nu}{S_\nu^*} - \frac{S_g}{S_g^*} - \frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}\right) + \beta_2(1-\delta_p)S_g^* I_{BT}^*\left(-1 + \frac{S_g}{S_g^*} - \frac{S_g}{S_g^*}\frac{I_{BT}}{I_{BT}^*} + \frac{I_{BT}}{I_{BT}^*}\right).
\end{aligned}$$

Let's calculate $\dot{\mathcal{G}}_{L_g}$:

$$\dot{\mathcal{G}}_{L_g} = \left(1 - \frac{L_g^*}{L_g}\right)\dot{L}_g = \left(1 - \frac{L_g^*}{L_g}\right)(\beta_2(1-\delta_p)S_g I_{BT} - (\beta + \mu_p)L_g).$$

Using the fifth equation of the system (5.9) we have:

$$\begin{aligned}
\dot{\mathcal{G}}_{L_g} &= \left(1 - \frac{L_g^*}{L_g}\right)\left(\beta_2(1-\delta_p)S_g I_{BT} - L_g\beta_2(1-\delta_p)\frac{S_g^* I_{BT}^*}{L_g^*}\right) \\
&= \alpha\left(1 - \frac{L_g^*}{L_g}\right)\left(L_\nu - L_g\frac{L_\nu^*}{L_g^*}\right) + \beta_2(1-\delta_p)\left(1 - \frac{L_g^*}{L_g}\right)\left(S_g I_{BT} - L_g\frac{S_g^* I_{BT}^*}{L_g^*}\right) \\
&= \beta_2(1-\delta_p)S_g^* I_{BT}^*\left(1 - \frac{L_g}{L_g^*} + \frac{S_g I_{BT}}{S_g^* I_{BT}^*} - \frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}\right).
\end{aligned}$$

Let's calculate $\dot{\mathcal{G}}_{S_g} + \dot{\mathcal{G}}_{L_g}$:

$$\dot{\mathcal{G}}_{S_g} + \dot{\mathcal{G}}_{L_g} = \alpha S_\nu^*\left(1 + \frac{S_\nu}{S_\nu^*} - \frac{S_g}{S_g^*} - \frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}\right) + \beta_2(1-\delta_p)S_g^* I_{BT}^*\left(-\frac{L_g}{L_g^*} + \frac{S_g}{S_g^*} + \frac{I_{BT}}{I_{BT}^*} - \frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}\right).$$

Let's now set $\rho_3 = 1 + \frac{S_\nu}{S_\nu^*} - \frac{S_g}{S_g^*} - \frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}$ and by addition by subtracting ρ_3 to $1 + \ln\frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}$, we have

$$\begin{aligned}
\rho_3 &= \frac{S_\nu}{S_\nu^*} - 1 - \ln\frac{S_\nu}{S_\nu^*} - \frac{S_g}{S_g^*} + 1 + \ln\frac{S_g}{S_g^*} - \frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*} + 1 + \ln\frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}, \\
\rho_3 &= \Phi\left(\frac{S_\nu}{S_\nu^*}\right) - \Phi\left(\frac{S_g}{S_g^*}\right) - \Phi\left(\frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}\right).
\end{aligned} \tag{5.12}$$

Let's now set $\rho_4 = -\frac{L_g}{L_g^*} + \frac{S_g}{S_g^*} + \frac{I_{BT}}{I_{BT}^*} - \frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}$ and by addition by subtracting ρ_4 to $1 + \ln\frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}$, we have

$$\begin{aligned}
\rho_4 &= -\frac{L_g}{L_g^*} + 1 + \ln\frac{L_g}{L_g^*} + \frac{S_g}{S_g^*} - 1 - \ln\frac{S_g}{S_g^*} + \frac{I_{BT}}{I_{BT}^*} - 1 - \ln\frac{I_{BT}}{I_{BT}^*} - \frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*} + 1 + \ln\frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}, \\
\rho_4 &= -\Phi\left(\frac{L_g}{L_g^*}\right) + \Phi\left(\frac{S_g}{S_g^*}\right) + \Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) - \Phi\left(\frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}\right).
\end{aligned} \tag{5.13}$$

Let's calculate $\dot{\mathcal{G}}_{I_g}$:

$$\dot{\mathcal{G}}_{I_g} = \left(1 - \frac{I_g^*}{I_g}\right)\dot{I}_g = \left(1 - \frac{I_g^*}{I_g}\right)\left(\beta L_g - I_g\beta\frac{L_g^*}{I_g^*}\right).$$

Using the sixth equation of the system (5.9) we have:

$$\dot{\mathcal{G}}_{I_g} = \beta\left(1 - \frac{I_g^*}{I_g}\right)\left(L_g - I_g\frac{L_g^*}{I_g}\right) = \beta\left(L_g - I_g\frac{L_g^*}{I_g^*} - L_g\frac{I_g^*}{I_g} + L_g^*\right) = \beta L_g^*\left(1 + \frac{L_g}{L_g^*} - \frac{I_g}{I_g^*} - \frac{I_g^*}{I_g}\frac{L_g}{L_g^*}\right).$$

Let's now set: $\rho_5 = 1 + \frac{L_g}{L_g^*} - \frac{I_g}{I_g^*} - \frac{I_g^*}{I_g}\frac{L_g}{L_g^*}$ and by addition by subtracting ρ_5 to $1 + \ln\frac{I_g^*}{I_g}\frac{L_g}{L_g^*}$, we have

$$\begin{aligned}
\rho_5 &= \frac{L_g}{L_g^*} - 1 - \ln\frac{L_g}{L_g^*} - \frac{I_g}{I_g^*} + 1 + \ln\frac{I_g}{I_g^*} - \frac{I_g^*}{I_g}\frac{L_g}{L_g^*} + 1 + \ln\frac{I_g^*}{I_g}\frac{L_g}{L_g^*}, \\
\rho_5 &= \Phi\left(\frac{L_g}{L_g^*}\right) - \Phi\left(\frac{I_g}{I_g^*}\right) - \Phi\left(\frac{I_g^*}{I_g}\frac{L_g}{L_g^*}\right).
\end{aligned} \tag{5.14}$$

Let's calculate $\dot{\mathcal{G}}_{S_{BT}}$:

$$\dot{\mathcal{G}}_{S_{BT}} = \left(1-\frac{S^*_{BT}}{S_{BT}}\right)\dot{S_{BT}} = \left(1-\frac{S^*_{BT}}{S_{BT}}\right)\left(r\left(1-\frac{S_{BT}}{K}\right)S_{BT} - \gamma_1(1-\delta_p)I_\nu S_{BT} - \gamma_2(1-\delta_p)I_g S_{BT} - (\theta_{BT}\delta_p N_p S_{BT} - \mu_{BT})S_{BT}\right).$$

Using the eighth equation of the system (5.9) we have

$$\begin{aligned}
\dot{\mathcal{G}}_{S_{BT}} &= \left(1-\frac{S^*_{BT}}{S_{BT}}\right)(\gamma_1(1-\delta_p)I^*_\nu S_{BT} + \gamma_2(1-\delta_p)I^*_g S_{BT} \\
&\quad - \gamma_1(1-\delta_p)I_\nu S_{BT} - \gamma_2(1-\delta_p)I_g S_{BT}) - \frac{r(S_{BT}-S^*_{BT})^2}{K} \\
&= \gamma_1(1-\delta_p)(-I^*_\nu S^*_{BT} + I^*_\nu S_{BT} + I_\nu S^*_{BT} - I_\nu S_{BT}) \\
&\quad + \gamma_2(1-\delta_p)(-I^*_g S^*_{BT} + I^*_g S_{BT} + I_g S^*_{BT} - I_g S_{BT}) - \frac{r(S_{BT}-S^*_{BT})^2}{K} \\
&= -\gamma_1(1-\delta_p)I^*_\nu S^*_{BT}\left(1-\frac{I_\nu}{I^*_\nu}-\frac{S_{BT}}{S^*_{BT}}+\frac{I_\nu S_{BT}}{I^*_\nu S^*_{BT}}\right) \\
&\quad - \gamma_2(1-\delta_p)I^*_g S^*_{BT}\left(1-\frac{I_g}{I^*_g}-\frac{S_{BT}}{S^*_{BT}}+\frac{I_g S_{BT}}{I^*_g S^*_{BT}}\right) - \frac{r(S_{BT}-S^*_{BT})^2}{K} \\
&= -\gamma_1(1-\delta_p)I^*_\nu S^*_{BT}\left(1-\frac{I_\nu}{I^*_\nu}-\frac{S_{BT}}{S^*_{BT}}+\frac{I_\nu S_{BT}}{I^*_\nu S^*_{BT}}+\frac{I_\nu S^*_{BT}}{I^*_\nu S_{BT}}-\frac{I_\nu S^*_{BT}}{I^*_\nu S_{BT}}\right) \\
&\quad - \gamma_2(1-\delta_p)I^*_g S^*_{BT}\left(1-\frac{I_g}{I^*_g}-\frac{S_{BT}}{S^*_{BT}}+\frac{I_g S_{BT}}{I^*_g S^*_{BT}}+\frac{I_g S^*_{BT}}{I^*_g S_{BT}}-\frac{I_g S^*_{BT}}{I^*_g S_{BT}}\right) - \frac{r(S_{BT}-S^*_{BT})^2}{K}.
\end{aligned}$$

Let's now set: $\rho_6 = 1-\frac{I_\nu}{I^*_\nu}-\frac{S_{BT}}{S^*_{BT}}+\frac{I_\nu S_{BT}}{I^*_\nu S^*_{BT}}+\frac{I_\nu S^*_{BT}}{I^*_\nu S_{BT}}-\frac{I_\nu S^*_{BT}}{I^*_\nu S_{BT}}$ and by addition by subtracting ρ_6 to $1+\ln\frac{I_\nu S^*_{BT}}{I^*_\nu S_{BT}}$, we have:

$$\begin{aligned}
\rho_6 &= -\frac{I_\nu}{I^*_\nu}+1+\ln\frac{I_\nu}{I^*_\nu}-\frac{S_{BT}}{S^*_{BT}}+1+\ln\frac{S_{BT}}{S^*_{BT}}+\frac{I_\nu S_{BT}}{I^*_\nu S^*_{BT}}-1-\ln\frac{I_\nu S_{BT}}{I^*_\nu S^*_{BT}}, \\
\rho_6 &= -\Phi\left(\frac{I_\nu}{I^*_\nu}\right)-\Phi\left(\frac{S_{BT}}{S^*_{BT}}\right)+\Phi\left(\frac{I_\nu S_{BT}}{I^*_\nu S^*_{BT}}\right). \qquad (5.15)
\end{aligned}$$

Let's now set: $\rho_7 = 1-\frac{I_g}{I^*_g}-\frac{S_{BT}}{S^*_{BT}}+\frac{I_g S_{BT}}{I^*_g S^*_{BT}}+\frac{I_g S^*_{BT}}{I^*_g S_{BT}}-\frac{I_g S^*_{BT}}{I^*_g S_{BT}}$ and by addition by subtracting ρ_7 to $1+\ln\frac{I_g S^*_{BT}}{I^*_g S_{BT}}$, we have:

$$\begin{aligned}
\rho_7 &= -\frac{I_g}{I^*_g}+1+\ln\frac{I_g}{I^*_g}-\frac{S_{BT}}{S^*_{BT}}+1+\ln\frac{S_{BT}}{S^*_{BT}}+\frac{I_g S_{BT}}{I^*_g S^*_{BT}}-1-\ln\frac{I_g S_{BT}}{I^*_g S^*_{BT}}, \\
\rho_7 &= -\Phi\left(\frac{I_g}{I^*_g}\right)-\Phi\left(\frac{S_{BT}}{S^*_{BT}}\right)+\Phi\left(\frac{I_g S_{BT}}{I^*_g S^*_{BT}}\right). \qquad (5.16)
\end{aligned}$$

Let's calculate $\dot{\mathcal{G}}_{I_{BT}}$:

$$\dot{\mathcal{G}}_{I_{BT}} = \left(1-\frac{I^*_{BT}}{I_{BT}}\right)\dot{I_{BT}} = (1-\frac{I^*_{BT}}{I_{BT}})(\gamma_1(1-\delta_p)I_\nu S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT} - (\theta_I\delta_p N_p S_{BT} - \mu_I)I_{BT}).$$

Using the ninth equation of the system (5.9) we have:

$$\dot{\mathcal{G}}_{I_{BT}} = \left(1-\frac{I^*_{BT}}{I_{BT}}\right)\left((\gamma_1(1-\delta_p)I_\nu S_{BT} + \gamma_2(1-\delta_p)I_g S_{BT} - I_{BT}\left(\frac{\gamma_1(1-\delta_p)I^*_\nu S^*_{BT}}{I^*_{BT}} + \frac{\gamma_2(1-\delta_p)I^*_g S^*_{BT}}{I^*_{BT}}\right)\right)$$

$$= (\gamma_1(1-\delta_p)I_\nu^* S_{BT}^* \left(1 - \frac{I_{BT}}{I_{BT}^*} + \frac{I_\nu S_{BT}}{I_\nu^* S_{BT}^*} - \frac{I_\nu I_{BT}^*}{I_\nu^* I_{BT}}\right) + \gamma_2(1-\delta_p)I_\nu^* S_{BT}^* \left(1 - \frac{I_{BT}}{I_{BT}^*} + \frac{I_g S_{BT}}{I_g^* S_{BT}^*} - \frac{I_g I_{BT}^*}{I_g^* I_{BT}}\right).$$

Let's now set: $\rho_8 = 1 - \frac{I_{BT}}{I_{BT}^*} + \frac{I_\nu S_{BT}}{I_\nu^* S_{BT}^*} - \frac{I_\nu I_{BT}^*}{I_\nu^* I_{BT}}$ and by addition by subtracting ρ_8 to $1 + \ln \frac{I_\nu S_{BT} I_{BT}^*}{I_\nu^* S_{BT}^* I_{BT}}$, we have:

$$\rho_8 = -\frac{I_{BT}}{I_{BT}^*} + 1 + \ln \frac{I_{BT}}{I_{BT}^*} + \frac{I_\nu S_{BT}}{I_\nu^* S_{BT}^*} - 1 - \ln \frac{I_\nu S_{BT}}{I_\nu^* S_{BT}^*} - \frac{I_\nu I_{BT}^*}{I_\nu^* I_{BT}} + 1 + \ln \frac{I_\nu S_{BT} I_{BT}^*}{I_\nu^* S_{BT}^* I_{BT}},$$
$$\rho_8 = -\Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) + \Phi\left(\frac{I_\nu S_{BT}}{I_\nu^* S_{BT}^*}\right) - \Phi\left(\frac{I_\nu S_{BT} I_{BT}^*}{I_\nu^* S_{BT}^* I_{BT}}\right). \tag{5.17}$$

Let's now set: $\rho_9 = 1 - \frac{I_{BT}}{I_{BT}^*} + \frac{I_g S_{BT}}{I_g^* S_{BT}^*} - \frac{I_g I_{BT}^*}{I_g^* I_{BT}}$ and by addition by subtracting ρ_9 to $1 + \ln \frac{I_g S_{BT} I_{BT}^*}{I_g^* S_{BT}^* I_{BT}}$, we have:

$$\rho_9 = -\frac{I_{BT}}{I_{BT}^*} + 1 + \ln \frac{I_{BT}}{I_{BT}^*} + \frac{I_g S_{BT}}{I_g^* S_{BT}^*} - 1 - \ln \frac{I_g S_{BT}}{I_g^* S_{BT}^*} - \frac{I_g I_{BT}^*}{I_g^* I_{BT}} + 1 + \ln \frac{I_g S_{BT} I_{BT}^*}{I_g^* S_{BT}^* I_{BT}},$$
$$\rho_9 = -\Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) + \Phi\left(\frac{I_g S_{BT}}{I_g^* S_{BT}^*}\right) - \Phi\left(\frac{I_g S_{BT} I_{BT}^*}{I_g^* S_{BT}^* I_{BT}}\right). \tag{5.18}$$

After summation taking into account the respective coefficients of the respective equations (5.10)-(5.18), we obtain

$$\begin{aligned}
\dot{\mathcal{G}}_\xi = &-(\alpha+\mu_p)\frac{(S_\nu - S_\nu^*)^2}{S_\nu} - \frac{r(S_{BT} - S_{BT}^*)^2}{K} + \alpha S_\nu^* \left[\Phi\left(\frac{S_\nu}{S_\nu^*}\right) - \Phi\left(\frac{S_g}{S_g^*}\right) - \Phi\left(\frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}\right)\right] \\
&+ \theta L_\nu^* \left[-\Phi\left(\frac{I_\nu}{I_\nu^*}\right) - \Phi\left(\frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*}\right) + \Phi\left(\frac{L_\nu}{L_\nu^*}\right)\right] + \beta L_g^* \left[\Phi\left(\frac{L_g}{L_g^*}\right) - \Phi\left(\frac{I_g}{I_g^*}\right) - \Phi\left(\frac{I_g^*}{I_g}\frac{L_g}{L_g^*}\right)\right] \\
&\times \beta_1(1-\delta_p)S_\nu^* I_{BT}^* \left[-\Phi\left(\frac{S_\nu^*}{S_\nu}\right) - \Phi\left(\frac{L_\nu}{L_\nu^*}\right) + \Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) - \Phi\left(\frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*}\right)\right] \\
&+ \beta_2(1-\delta_p)S_g^* I_{BT}^* \left[-\Phi\left(\frac{L_g}{L_g^*}\right) + \Phi\left(\frac{S_g}{S_g^*}\right) + \Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) - \Phi\left(\frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}\right)\right] \\
&+ \gamma_1(1-\delta)I_\nu^* S_{BT}^* \left[\Phi\left(\frac{I_\nu}{I_\nu^*}\right) + \Phi\left(\frac{S_{BT}}{S_{BT}^*}\right) - \Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) - \Phi\left(\frac{I_\nu S_{BT} I_{BT}^*}{I_\nu^* S_{BT}^* I_{BT}}\right)\right] \\
&+ \gamma_2(1-\delta)I_g^* S_{BT}^* \left[\Phi\left(\frac{I_g}{I_g^*}\right) + \Phi\left(\frac{S_{BT}}{S_{BT}^*}\right) - \Phi\left(\frac{I_{BT}}{I_{BT}^*}\right) - \Phi\left(\frac{I_g S_{BT} I_{BT}^*}{I_g^* S_{BT}^* I_{BT}}\right)\right].
\end{aligned}$$

Let's put $\chi = \max\left\{\beta_1(1-\delta_p)S_\nu^* I_{BT}^*,\ \theta L_\nu^*,\ \alpha S_\nu^*,\ \beta_2(1-\delta_p)S_g^* I_{BT}^*,\ \beta L_g^*,\ \gamma_1(1-\delta)I_\nu^* S_{BT}^*,\ \gamma_2(1-\delta)I_g^* S_{BT}^*\right\}$. Then we get again:

$$\begin{aligned}
\dot{\mathcal{G}}_\xi \leqslant &-(\alpha+\mu_p)\frac{(S_\nu - S_\nu^*)^2}{S_\nu} - \frac{r(S_{BT} - S_{BT}^*)^2}{K} + \chi\left[-\Phi\left(\frac{S_\nu^*}{S_\nu}\right) - \Phi\left(\frac{L_\nu^*}{L_\nu}\frac{S_\nu I_{BT}}{S_\nu^* I_{BT}^*}\right) - \Phi\left(\frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*}\right) + \Phi\left(\frac{S_\nu}{S_\nu^*}\right)\right. \\
&\left. -\Phi\left(\frac{S_g^*}{S_g}\frac{S_\nu}{S_\nu^*}\right) - \Phi\left(\frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}\right) - \Phi\left(\frac{I_g^*}{I_g}\frac{L_g}{L_g^*}\right) + 2\Phi\left(\frac{S_{BT}}{S_{BT}^*}\right) - \Phi\left(\frac{I_\nu S_{BT} I_{BT}^*}{I_\nu^* S_{BT}^* I_{BT}}\right) - \Phi\left(\frac{I_g S_{BT} I_{BT}^*}{I_g^* S_{BT}^* I_{BT}}\right)\right].
\end{aligned}$$

Φ being a positive definite function on the interval $[1;+\infty[$, then

$$\Phi(\xi_1) + \Phi(\xi_2) \leqslant \Phi(\xi_1\xi_2), \forall\ \xi_1,\ \xi_2 \in [1;+\infty[. \tag{5.19}$$

Of the inequality (5.19), we get

$$\dot{\mathcal{G}}_\xi \leqslant -(\alpha+\mu_p)\frac{(S_\nu - S_\nu^*)^2}{S_\nu} - \frac{r(S_{BT} - S_{BT}^*)^2}{K} - \chi\left[\Phi\left(\frac{I_\nu}{I_\nu^*}\right) + \Phi\left(\frac{I_g}{I_g^*}\right) + \Phi\left(\frac{S_g^*}{S_g}\right) + \Phi\left(\frac{I_\nu^*}{I_\nu}\frac{L_\nu}{L_\nu^*}\right)\right.$$

$$+\Phi\left(\frac{S_g^*}{S_g}\frac{S_v}{S_v^*}\right)+\Phi\left(\frac{I_g^*}{I_g}\frac{L_g}{L_g^*}\right)+\Phi\left(\frac{L_g^*}{L_g}\frac{S_g I_{BT}}{S_g^* I_{BT}^*}\right)+\Phi\left(\frac{L_v^*}{L_v}\frac{S_v I_{BT}}{S_v^* I_{BT}^*}\right)\Big].$$

Under the hypothesis (H1), we have $\dot{\mathcal{G}}_\xi < 0$. Moreover $\mathcal{G}_\xi > 0$, $\forall\ S_v, L_v, I_v, S_g, L_g, I_g, S_{BT}, I_{BT} \in \mathbb{R}_+$, such that $S_v > 0$ and $\dot{\mathcal{G}}_\xi = 0$ for $S_v = S_v^*$, $L_v = L_v^*$, $I_v = I_v^*$, $S_g = S_g^*$, $L_g = L_g^*$, $I_g = I_g^*$, $S_{BT} = S_{BT}^*$, $I_{BT} = I_{BT}^*$. Therefore, the largest compact invariant set in $\left\{(S_v, L_v, I_v, S_g, L_g, I_g, S_{BT}, I_{BT}) \in \Gamma \mid \dot{\mathcal{G}}_\xi = 0\right\}$ is the singleton $\mathcal{D} = (S_v = S_v^*, L_v = L_v^*, I_v = I_v^*, S_g = S_g^*, L_g = L_g^*, I_g = I_g^*, S_{BT} = S_{BT}^*, I_{BT} = I_{BT}^*)$. According to the asymptotic stability theorem to the invariance principle of Lasalle [29], the endemic equilibrium point $\mathcal{D}$ of the system (2.1) is globally asymptotically stable whenever $\mathcal{R}_0 > 1$. □

6. Numerical simulation

To determine the effectiveness of a control policy, it is necessary to assess the number of whitefly in the plantation. Bemisia tabaci insect pest control strategy or policy is to use the insecticide called Verticillium lecanii for the protection of tomato plants and eliminate this tomato yellow leaf curve virus (TYLCV) in the tomato plantation. The numerical simulations that we propose in this subsection aim to illustrate the asymptotic behavior of the number of infected plants as a function of the reproduction number basic $\mathcal{R}_0$ as predicted by the properties. The simulation parameters for the model (2.1) come from the scientific literature review which are given in the Table 4. The parameters Λ, r, μ_p were selected based on tomato plant growth and pest mortality data from agricultural studies. The infection rates β_1, β_2 were adapted from epidemiological models. To do this digital simulations, we carry them out in three scenarios. In the first scenario, we provide simulations to illustrate the study of the endemic equilibrium point of the theoretical part. In the second scenario we also provide simulations to illustrate the study of the disease-free equilibrium point to corroborate our theoretical results. In the last scenario seen that the presence of susceptible whiteflies poses a negative impact on the growth and productivity of tomato plants, we evaluated the effectiveness of pesticide use to allow the manager to have a tomato field without tomato yellow leaf curl virus (TYLCV) disease and without tomato whiteflies The simulations will be based mainly on the function Odeint of the Scipy.integrate of python.

Table 4: Parameter values of the TYLCV model.

Valeurs/Parameters	$\mathcal{R}_0 < 1$	$\mathcal{R}_0 > 1$	Sources
K	100	100	Assumed
Λ	10	10	Atifah et al. [5]
N_p	160	160	Amelia et al. [3]
r	0.5	0.5	Assumed
δ_p	0.035	0.001	Assumed
α	0.1	0.1	Butter et al. [8]
θ	0.01	0.01	Amelia et al. [3]
β	0.1	0.1	Butter et al. [8]
β_1	0.03	0.03	Amelia et al., Atifah et al. [3, 5]
β_2	0.03	0.03	Amelia et al., Atifah et al. [3, 5]
γ_1	0.025	0.025	Amelia et al., Butter et al. [3, 8]
γ_2	0.2	0.2	Amelia et al., Atifah et al. [3, 5]
μ_p	0.3	0.3	Amelia et al., Maruthi [3, 32]
μ_{BT}	0.07	0.07	Amelia et al., Atifah et al. [3, 5]
θ_{BT}	0.05	0.05	Amelia et al., Atifah et al. [3, 5]
T	150	150	Amelia et al. [3]

6.1. Scenario 1: endemic equilibrium simulation

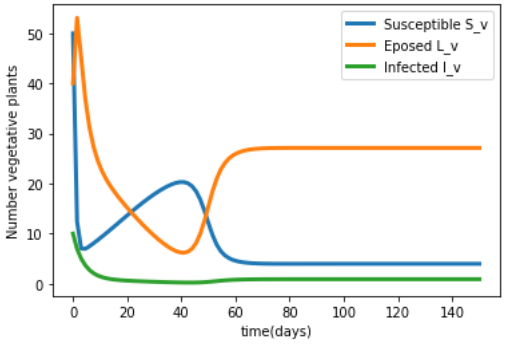

Figure 2: Population dynamics of susceptible (S_v), latent (L_v) and infected (I_v) vegetative tomato plants under varying doses of Verticillium lecanii. The x-axis represents the time passing in days and the y-axis represents by population size given in number of vegetative plants. $\mathcal{R}_0 = 3.6997 > 1$.

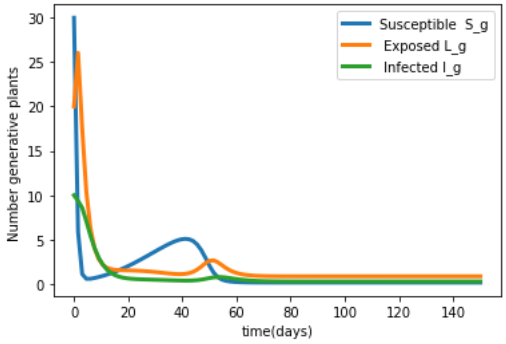

Figure 3: Population dynamics of susceptible (S_g), latent (L_g) and infected (I_g) vegetative tomato plants under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of generative plants. $\mathcal{R}_0 = 3.6997 > 1$.

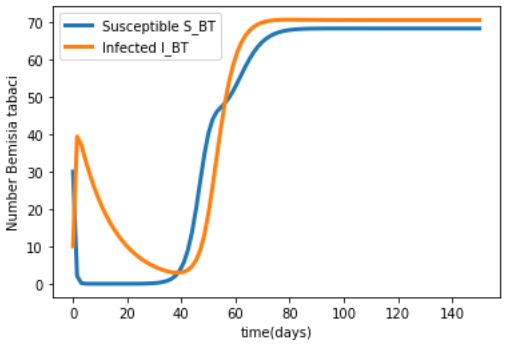

Figure 4: Population dynamics of susceptible (S_{BT}) and infected (I_{BT}) whiteflies population under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of whiteflies. $\mathcal{R}_0 = 3.6997 > 1$.

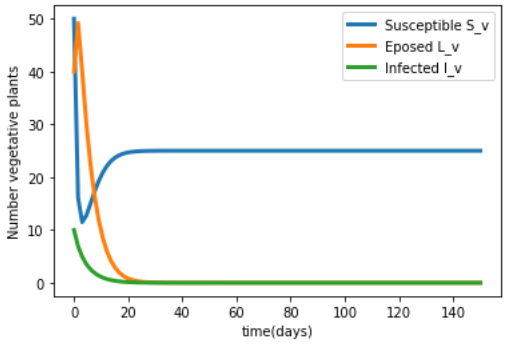

Figure 5: Population dynamics of susceptible (S_v), latent (L_v) and infected (I_v) vegetative tomato plants under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of vegetative plants. $\mathcal{R}_0 = 0.3981 < 1$.

First result: The results of this first part of the numerical simulation illustrate the persistence of the disease (TYLCV) in the tomato plantation. A simulation of the system (2.1) for $\mathcal{R}_0 = 3.6997 > 1$, using initial conditions $S_v(0) = 50$, $L_v(0) = 40$, $I_v(0) = 10$, $S_g(0) = 30$, $L_g(0) = 20$, $I_g(0) = 10$, $S_{BT} = 30$, $I_{BT} = 10$. Figure 2 represents the populations dynamics of tomato plants in vegetative phase: Susceptible S_v (blue curves), Exposed L_v (yellow curves), Infected I_v (green curves). Figure 3 represents the Populations dynamics of tomato plants in generative phase : Susceptible S_g (blue curves), Exposed L_g (yellow curves), Infected I_g (green curves). Figure 4 represents the populations dynamics of vectors Bemisia tabaci: Susceptible S_{BT} (blue curves), Infected I_{BT} (yellow curves). Simulation results illustrate that applying Verticillium lecanii at doses exceeding 0.1% effectively allows the virus to persist in the tomato plantation and this endemic equilibrium point (EE) is globally asymptomatically stable. This is consistent with the theoretical results obtained in the Theorem 5.2. We will illustrate through numerical simulation that the endemic equilibrium (EE) converges towards the disease free equilibrium (DFE) for slightly increased efficiency rate of Verticillium lecanii in the simulation part which follows.

6.2. Scenario 2: disease free-equilibrium simulation

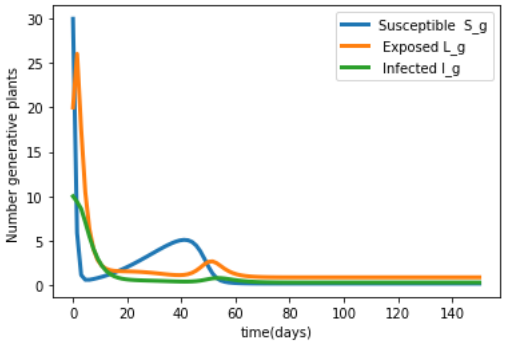

Figure 6: Population dynamics of susceptible (S_g), latent (L_g) and infected (I_g) generative tomato plants under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of generative plants. $\mathcal{R}_0 = 0.3981 < 1$.

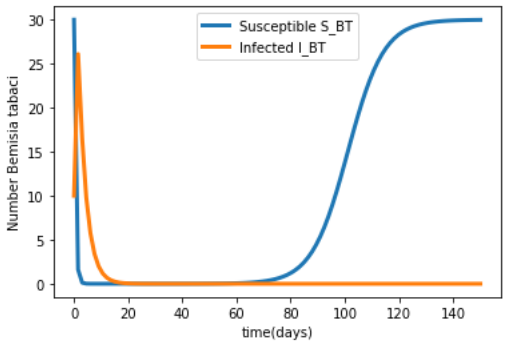

Figure 7: Population dynamics of susceptible (S_{BT}) and infected (I_{BT}) whiteflies population under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of whiteflies. $\mathcal{R}_0 = 0.3981 < 1$.

Second result: The results of this first part of the numerical simulation illustrate the extinction of the disease (TYLCV) in the tomato plantation. A simulation of the system (2.1) for $\mathcal{R}_0 = 0.3981 < 1$, using initial conditions $S_v(0) = 50$, $L_v(0) = 40$, $I_v(0) = 10$, $S_g(0) = 30$, $L_g(0) = 20$, $I_g(0) = 10$, $S_{BT} = 30$, $I_{BT} = 10$. Figure 5 represents the populations dynamics of tomato plants in vegetative phase: Susceptible S_v (blue curves), Exposed L_v (yellow curves), Infected I_v (green curves). Figure 6 represents the populations dynamics of tomato plants in generative phase : Susceptible S_g (blue curves), Exposed L_g (yellow curves), Infected I_g (green curves). Figure 7 represents the populations dynamics of vectors Bemisia tabaci: Susceptible S_{BT} (blue curves), Infected I_{BT} (yellow curves). Simulation results illustrate that applying Verticillium lecanii at doses exceeding 3.5% effectively reduces infected populations and drives the system to a disease-free equilibrium and this disease free equilibrium point (DFE) is globally asymptomatically stable. This is consistent with the theoretical results obtained in the Theorem 4.1. We will illustrate through numerical simulation that the disease free equilibrium point (DFE) converges towards the disease free equilibrium (DFE) without whiteflies bemisia tabaci in the tomato plantation for a efficiency rate of Verticillium lecanii that exceeds 10% in the simulation part which follows.

6.3. Scenario 3: return disease free-equilibrium simulation

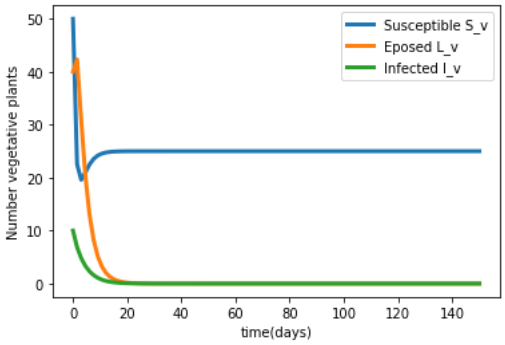

Figure 8: Population dynamics of susceptible (S_v), latent (L_v) and infected (I_v) vegetative tomato plants under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of vegetative plants. $\mathcal{R}_0 > 1$ and $\delta_p = 0.1$.

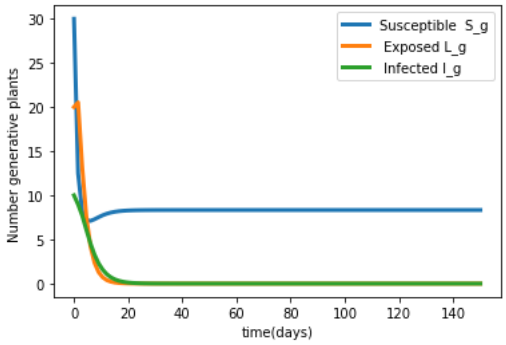

Figure 9: Population dynamics of susceptible (S_g), latent (L_g) and infected (I_g) vegetative tomato plants under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of generative plants. $\mathcal{R}_0 > 1$ and $\delta_p = 0.1$.

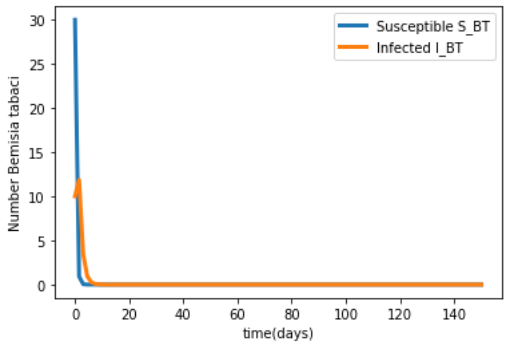

Figure 10: Population dynamics of susceptible (S_{BT}) and infected (I_{BT}) whiteflies population under varying doses of Verticillium lecanii. The x-axis represents the time passing in days, the y-axis represents by population size given in number of whiteflies. $\mathcal{R}_0 > 1$ and $\delta_p = 0.1$.

Third result: The results of this first part of the numerical simulation illustrate the extinction of the disease (TYLCV) and eradicate of the whiteflies in the tomato plantation. A simulation of system (2.1) for $\mathcal{R}_0 = 3.6997 > 1$, using initial conditions $S_v(0) = 50$, $L_v(0) = 40$, $I_v(0) = 10$, $S_g(0) = 30$, $L_g(0) = 20$, $I_g(0) = 10$, $S_{BT} = 30$, $I_{BT} = 10$. Figure 8 represents the Populations dynamics of tomato plants in vegetative phase: Susceptible S_v (blue curves), Exposed L_v (yellow curves), Infected I_v (green curves). Figure 9 represents the populations dynamics of tomato plants in generative phase: Susceptible S_g (blue curves), Exposed L_g (yellow curves), Infected I_g (green curves). Figure 10 represents the populations dynamics of vectors Bemisia tabaci: Susceptible S_{BT} (blue curves), Infected I_{BT} (yellow curves). Simulation results illustrate that applying Verticillium lecanii at doses exceeding 10% effectively reduces infected populations and drives the system to a disease-free equilibrium without the whiteflies.

7. Sensitivity analysis

To determine the best way to reduce tomato leaf curl virus and mortality due to TYLCV, it is necessary to know the relative importance of the factors responsible for its transmission and prevalence. The initial transmission is directly related to the basic reproduction number and the prevalence is directly related to the endemic equilibrium point, in particular the parameters β_1, β_2, γ_1, γ_2, μ_p, and δ_p are relevant for the tomato population; we calculate the sensitivity indices of the basic reproduction number $\mathcal{R}_0$. These sensitivity indices indicate how crucial each parameters is for disease transmission and prevalence. Sensitivity analysis is commonly used to determine the robustness of model (2.1) predictions to parameter values (since there are usually errors in the collection of parameters data and assumed parameters). It would be used to describe parameters that have a high impact on the basic reproduction number $\mathcal{R}_0$ and should be used for intervention strategies. The local sensitivity analysis is based on the normalized forward sensitivity index $\mathcal{R}_0$.

7.1. Description of sensitivity analysis

Sensitivity indices allow us to measure the relative change in a state variable when a parameter changes. The normalized forward sensitivity index of a variable to a parameter is ratio of relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives Rimbaud [21]. The normalized forward sensitivity index of a variable, $\mathcal{R}_0$ that depends differentiable on a parameter, p, is defined as:

$$\Gamma_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}. \tag{7.1}$$

We are specifically interested in studying the sensitivity of certain parameters of $\mathcal{R}_0$, such as the infection rates and the mortality rate of the tomato plant, here, $p = \beta_1$, β_2, γ_1, γ_2, μ_p and δ_p which are part of the

model parameter value summarized in the Table 4. We analyze the sensitivity of these parameters value from equation (7.1), when the TYLCV is persist in the tomato plantation, i.e., $\mathcal{R}_0 > 1$. When p taken the certain value of $\mathcal{R}_0$, the sensitivity index $\mathcal{R}_0$ are given by Table 5. Table 5 presents the expressions and

Table 5: Sensitivity indices of certain parameters of $\mathcal{R}_0$.

Parameter	Value	$\frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}$	Sensitivity index
β_1	0.03	$+\frac{\beta_1\gamma_1\mu_p(\theta+\mu_p)(\beta+\mu_p)}{2[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]}$	$+0.7$
β_2	0.03	$+\frac{\alpha\beta\beta_2\gamma_2(\theta+\mu_p)}{2[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]}$	$+0.5$
γ_1	0.025	$+\frac{\beta_1\gamma_1\mu_p(\theta+\mu_p)(\beta+\mu_p)}{2[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]}$	$+0.7$
γ_2	0.2	$+\frac{\alpha\beta\beta_2\gamma_2(\theta+\mu_p)}{2[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]}$	$+0.5$
μ_p	0.3	$-\frac{(\theta+\mu_p)[\gamma_1\beta_1\mu_p(\alpha+2\mu_p)(\beta+\mu_p)^2+\beta\alpha\beta_2\gamma_2(2\alpha\beta+(3\alpha+3\beta+4\mu_p)\mu_p)]}{2(\beta+\mu_p)(\alpha+\mu_p)[\gamma_1\mu_p\beta_1\theta(\beta+\mu_p)+\gamma_2\beta_2\beta\alpha(\theta+\mu_p)]}$	-3.7
δ_p	0.001	$-\frac{\delta_p[2r+(\mu_{BT}+N_p\delta_p\theta_{BT})(-2+r(1-\delta_p)^2N_p\theta_{BT})]}{2(1-\delta_p)(r-\mu_{BT}-N_p\delta_p\theta_{BT})}$	-0.0014

sensitivity index values of the β_1, β_2, γ_1, γ_2, and μ_p, which are essential for the dynamics of tomato plant populations and whiteflies and which impact the basic reproduction number $\mathcal{R}_0$. The results indicate that the interaction rates β_1 (rate of transmission of the virus to vegetative plants by the witheflies), β_2 (rate of the transmission of the virus to generative plants by the whiteflies), γ_1 (rate of the transmission of the virus to whiteflies by the infected vegetative plants), γ_2 (rate of the transmission of the virus to whiteflies by the infected generative plants), between populations of tomato plants and whiteflies are parameters more sensitive to $\mathcal{R}_0$. In transmission sites, a decrease or increase of 10% of these parameters leads respectively to a decrease or an increase in the basic reproduction number of 7%, 5%, 7%, 5%, and a 10% increase in the μ_p and δ_p parameters results in a decrease in the basic reproduction number of 37% and of 0.014%. We see that the expressions of these sensitivity indices of the parameters considered therefore depend on the plant parameters and those of the virus vectors. Change the population size will impact the sensitivity indices of tomato plant parameters. We clearly see that the sensitivity indices at the level of vegetative phase have a grater impact on the basic reproduction number $\mathcal{R}_0$ than the others, notably, the parameters β_1 and γ_1. This is explained by the fact that tomato plants in the vegetative phase are much more preferred by whiteflies for their food and the more whiteflies prefer vegetative plants, the higher the rate of acquisition of the virus by susceptible whiteflies, which also reflects the sensitivity index of the γ_1 parameter which is as crucial as the β_1 parameter. For a better control strategy, it would be necessary to spray the vegetative phases well to avoid increasing the numbers of infected plants and as well the number of infected whiteflies. Given that the prevalence of the disease is directly at the endemic equilibrium point, if at the level of the vegetative phase the spraying is perfectly controlled, we will also have a lower prevalence of TYLCV disease in the tomato plantation.

8. Discussion and conclusion

We have discussed a mathematical model of the spread of yellow virus in tomato plants with logistical functions and use Verticillium lecanii as a means of control of disease transmission. It can be seen that the disease spread model has two equilibrium points, namely the Disease-Free Equilibrium point and the Disease Endemic Equilibrium point. This during the disease-free equilibrium point (DFE) $\mathcal{D}^1$ is stable if $\mathcal{R}_0 < 1$, with pesticide use efficiency at the rate 3.5% and the model will have an Disease Endemic Equilibrium point (EE) $\mathcal{D}$ is stable when $\mathcal{R}_0 > 1$. The persistence of the Disease Endemic Equilibrium point (EE) is due to the effectiveness rat use to control Bemisia tabaci insects, which is of 0.1%. The numerical simulations illustrate show that a use of Verticillium lecanii at 10% for the population of latent plants, infected in the vegetative and generative phases will experience extinction, just like the susceptible and infected population of Bemisia tabaci. However, we note that taking into account the latent stage makes it possible to reduce the number of infected plants for both vegetative and generative plants.

Therefore the rate of use of the pesticide Verticillium lecanii will have less negative impact on the health and production of generative plants and as well on public health for consumption of tomato fruits. The calculation of the sensitivity indices in the last section illustrated that for a good disease control strategy, the manager must pay more attention to the health of the vegetative plants to avoid a wide spread of the disease because the vectors certainly have a preference for vegetative plants than generative plants for their food. In conclusion, to avoid having a negative impact on the development of tomato plants and on public health when the dose or the spray effectiveness level is high enough, we prefer to use other means such as uprooting of infected plants, burning and covering the growing area with greenhouses to control the spread of tomato yellow virus.

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