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USCOEGA – Yobe State, Nigeria<sup>2</sup>Mathematics Department,  
MAUTECH, Yola, Adamawa State,  
Nigeria

\*Corresponding author's email:

[\\*stado@mautech.edu.ng](mailto:stado@mautech.edu.ng)Cite this: *CaJoST*, 2021, 1, 43-54

## Biological Control of Vector-Borne Viral Diseases in Solanaceous Vegetable Plants: Mathematical Modeling Approach

Timothy A. Shamaki<sup>1\*</sup>, Samuel Musa<sup>2</sup> and Olaide A. Adesanya<sup>2</sup>

A mathematical model for the biological control of Vector-Borne Virus Diseases (VBVDs) in solanaceous vegetable plants is examined. The study adopts linearization method. Disease-free equilibrium and endemic disease equilibrium states have been established. The basic reproduction number for the control of VBVDs is obtained. Analytically, the local and global stability of disease-free equilibrium and disease endemic equilibrium were established. The numerical simulation carried out on the model have proven that ladybug beetles can be used as biological control agents in tackling viruses being transmitted by aphids, thrips and whiteflies in solanaceous vegetable plants.

**Keywords:** Biological Control; Disease Free Equilibrium; Disease Endemic Equilibrium; Basic Reproduction Number; Local Stability and Global Stability.

**1. Introduction**

Plants are essential to life. They are a source of food, medicine, clothing, and are important to a healthy environment [1]. Unfortunately, plants can become infected with diseases and this has continued to ravage on one-third of food quantity produced worldwide [2]. According to Agrios [3], an estimated average of fourteen percent (14%) of world crops are lost to diseases due to pests. In fact, the issue of diseases transmitted by pests has for long being a major setback to agricultural practices [4]. Admittedly, pests and diseases are constant threats to global agriculture and so present-day farming requires new and adaptive strategies to lessen the economic impact of pests and diseases on cropping systems [5].

With a projected number of 9.7 billion people by the year 2050, the world population and its continuing growth is heavily dependent on a steady agricultural output in order to attain sustainable food supplies [6]. For that reason, it is desirable that applied mathematics works out a science based modalities for solution to the looming acute global food shortage since the conventional methods of chemical control as well as the Integrated Pest Management (IPM) are associated with a lot of detrimental challenges. Moreover, quality research is highly needed in the aspect of biological control most especially by considering the fact that there are many mathematical models that described the interaction between vectors and humans, but there are not as many that describe the

relationship between plants and vectors [7]. Accordingly, a number of studies have been carried out on strategies for the management of plant pathogens but only a few had adopted mathematical modeling approach [8].

Holt *et al.* [9] proposed a model, based on a small number of biologically important parameters, to assess the impact of management variables. Their model mainly focused on selection of healthy rather than diseased plants, and the roguing of diseased cassava. Some years later, [10] formulated a simple model describing disease dynamics, using the same notation as [9]. But Jeger *et al.* [10] examined the theoretical bases for their model and its use in the evaluation of control strategies with regard to interactions between the host plant, the virus and the vector. Hence, they established that vector activity and behaviour, especially in relation to virus transmission, are important determinants of the rate and extent of epidemic development.

Also [11] considered a plant disease model with impulsive replanting of healthy plants and removing of infected plants. They adopted Floquet's theorem and small amplitude perturbation. Thus, the locally stability was established for sufficient conditions under which the infected plant have free periodic solution. Moreover, permanence of the system was investigated by means of numerical simulations. Under certain parameter spaces, it was shown

that a nontrivial periodic solution emerged via a supercritical bifurcation.

Similarly, [12] considered classical epidemic deterministic epidemic model for the spread of the pine wilt disease which has vector mediated transmission. The analysis of the model showed that its dynamics were completely determined by

the basic reproduction number  $R_0$ . And by using a Lyapunov function and a LaSalle's invariant set theorem, they proved the global asymptotical stability of the disease-free equilibrium. Hence, if  $R_0 \leq 1$ , the disease free equilibrium is globally asymptotically stable, and the disease will be eliminated. If  $R_0 > 1$ , a unique endemic equilibrium exists and is shown, using the geometric approach method for global stability, to be globally asymptotically stable, under certain restrictions on the parameter values.

One limitation in [1] is that, it fails to acknowledge the reality associated with insect vectors, as there is hardly a case where only one class of vector patronizes a certain plant. Therefore, this study shall complement [1] by considering specific Vector-Borne Viral Diseases VBVDs and by making advances by means of extending the vector pests into three classes. We focused on how biological control can be achieved through theoretical research using mathematical modeling technique. We focused on common viral diseases that affect solanaceous vegetable plants, specifically the Yellow Leaf Curl Virus (YLCV), Spotted Wilt Virus (SWV) and Cucumber Mosaic Virus (CMV). Obviously, these three viruses operate together within solanaceous vegetable plants [13].

It is a common knowledge that these plant viruses also require some sort of carrier, known as vectors, to transmit the pathogen from plant to plant. Thus, as plants are sedentary hosts, insects are commonly associated with the transmission of these viruses. Consequently, the study is delineated to a class of aphids (*green peach aphids*), thrips (*T. tabasi*) and whiteflies (*Bemisia tabasi*) because they are reported to be the pathogenic carriers and being the common problem associated with solanaceae plants which can be controlled by natural predatory enemies - ladybugs (*hippodamia convergens*).

Essentially, [14] argued that due to seasonality, the insect vectors are mostly active in the warm months and almost dormant in the cooler ones, and their model was analyzed for unspecified plant with periodic coefficients. But in this study, we considered three solanaceae plants (i.e. tomato, pepper and eggplant). Furthermore, [7]

contended that, contrary to claim in [15] model, there is no empirical evidence for plant to plant viral transmission. Accordingly, they made extra assumptions and proposed new model which serves as the basis of this research.

We based our research on solanaceae plants (i.e. tomato, pepper and eggplant) because they are among the world's most cultivated crops and given proper conditions and regular maintenance, they are relatively easy to grow [13]. But we noticed that this type of considerations will necessitates alteration of Jackson's model since the targeted plants are cultivated within one season. Nevertheless, as for the control measure, we maintained the use of bio-control, as this will guarantee a safer, economical and better means of safeguarding the planet.

## 2. Materials and Methods

### 2.1 Description of the Mode

In this study, the total population under consideration consist of three distinct subclasses. The solanaceae vegetable plants  $N(t)$ , these are further compartmentalized into Susceptible plants  $S(t)$ , Infected plants  $I(t)$  and Recovered Plants  $R(t)$  respectively.

The vector pests  $V(t)$  are categorized based on their species and according to their epidemiological status as susceptible aphids  $A_s(t)$  as well as the infected aphids  $A_I(t)$ , susceptible thrips  $T_s(t)$  and infected thrips  $T_I(t)$  and lastly susceptible whiteflies  $W_s(t)$  and infected whiteflies  $W_I(t)$  in that respective order. Moreover, we maintain a class of predators  $P(t)$ .

We assume the prey (vectors) population finds ample food at all times and are not limited by their number or space. And the environment does not change in favour of some species of insect vectors. We assume the food supply of the predator population depends entirely on the prey population. And the predators will prefer aphids, thrips and whiteflies in that order. We consider that predators,  $P(t)$  feed on both infectious and susceptible pests and do not get sick by the pathogen, vectors are just source of natural food to the predator population. As a control measure, we assume a fixed number  $V$  at which predators are being added and they

reproduce (offspring production) at a rate  $\omega$  which is proportional to the number of predators. Furthermore, we suppose the infected insect vectors cannot reproduce but can be attacked by the predator equally. In our study, the Recovered plants  $R(t)$  are those who were once infected but are thought to be wiping out due to severe ill-health or death because they are not contributing in the transmission dynamic of the disease. Thus, it is reasonable to assume that plants are relatively fixed, because when a plant dies by a pathogen or natural death in farms, it is replaced with new healthy ones. The new replaced plant assumes to be susceptible. Primarily, we neglected other means of viral transmission such as air (wind) and water and assume that the infected insect vectors do not recover from the virus. And lastly, we assume the rate at which the insect vectors enter into the system is through immigration at constant rate  $n_i$  or comes in through birth  $b_i$  which is proportional to the number of susceptible vectors.

The forces of infection are  $\left(\frac{a_1\beta_1A_I}{K} + \frac{a_2\beta_2T_I}{K} + \frac{a_3\beta_3W_I}{K}\right)$ ,  $\left(\frac{a_1B_1A_S I}{K}\right)$ ,  $\left(\frac{a_2B_2T_S I}{K}\right)$  and  $\left(\frac{a_3B_3W_S I}{K}\right)$ ,

which are the viral transmission rates from infected vectors to susceptible plants, also transmission rates from infected plants to susceptible aphids, thrips and whiteflies respectively due to bite by vector pests. The plants can natural recovery from the viruses at the rate  $\gamma$  which is proportional to the number of infected plants at time  $t$ . The recruitment rate

into susceptible plants  $\mu(K - N)$  is by replanting and it depends upon the available size of land occasioned by death of infected plants

$I(t)$  and recovered plants  $R(t)$ . The recruitment into susceptible vector is at constant rate of

immigration  $n_i$  and by reproduction rate  $b_i$  being proportional to the number of susceptible vector pests into their respective classes. The rate at which the predators enter into the system is through immigration at constant rate  $\nu$  or increase through birth rate  $\omega$  which is proportional to the number of predators.

The natural death rate of subclasses  $S(t), I(t), R(t)$  is given by  $\mu$ , and the natural

death rate of subclasses  $V_s(t)$  and  $V_I(t)$  is given by  $m$  and natural death rate of predators  $P(t)$  is  $\delta$  while the disease induced death rate

in subclass  $I(t)$  is  $d$ . The predators compete with one another for prey at a constant rate  $\varepsilon$ .

We maintained  $\alpha_4$  as a conversion efficiency of predators due to preying on insect vectors. We supposed that  $c_i, i = 1, 2, 3$ , are proportion of contacts that is with susceptible vectors while  $(1 - c_i)$  are proportion of contacts that are with infected insects respectively. For all parameters with subscript numbered 1, 2, 3; the first goes to aphids, thrips takes the second, and whiteflies the third respectively.

**Table 1: The Model Parameters and their Meaning**

Notation	Description of the Parameters
$\beta_i, i = 1, 2, 3$	Transmission rate of viral infection from insect vectors to plants.
$B_i, i = 1, 2, 3$	Transmission rate of viral infection from plants to insect vectors.
$a_i, i = 1, 2, 3$	Biting rate of plants due to aphids, thrips and whiteflies respectively.
$b_i, i = 1, 2, 3$	Reproductive rate of insect vectors into their respective populations.
$c_i, i = 1, 2, 3$	Proportion of contacts between predators and insect vectors.
$n_i, i = 1, 2, 3$	Immigration rate into aphids, thrips and whiteflies sub populations.
$m$	Natural death rate of vectors.
$K$	Total plant host population (Carrying Capacity).
$\gamma$	Natural recovery rate of plants.
$\mu$	Plants replanting rate and constant natural death.
$\alpha_3$	Saturation of predators due to insects.
$\alpha_4$	Conversion efficiency of predators due to preying on insect vectors.
$\theta$	Constant of proportionality between predators and preys.
$d$	Death rate of infected plants due to the disease.
$\delta$	Natural death rate of predators.

$\omega$  Reproductive (offspring production) rate of predators.  
 $\nu$  Rate at which predators are added.

$t$  Time.

The flow diagram of the model can be seen in Figure 1 below:

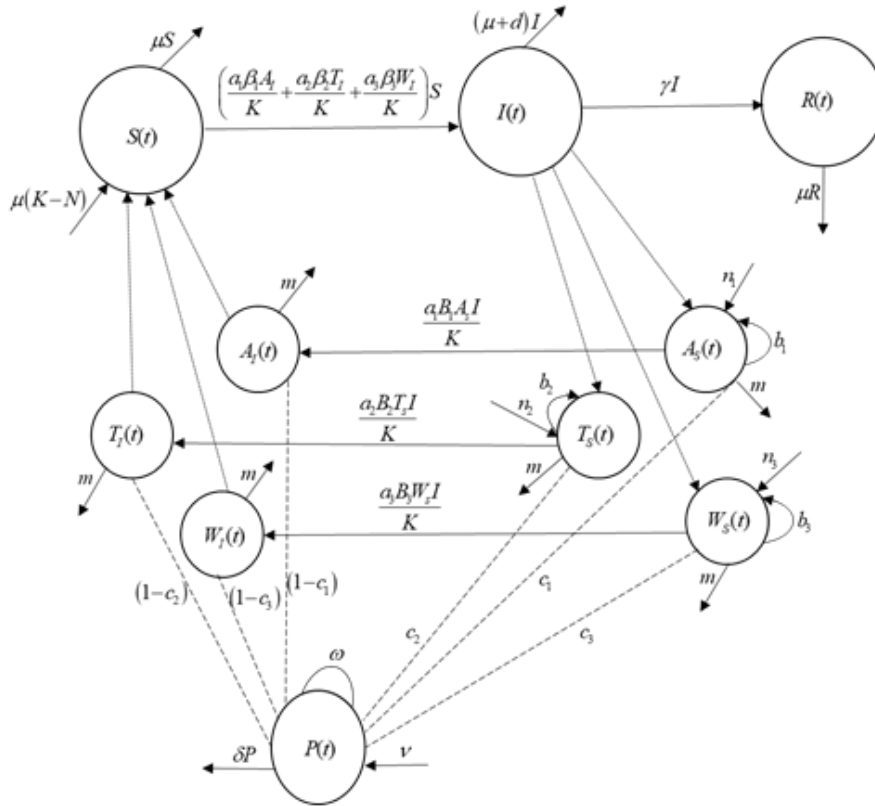


Figure 1: Flow diagram of Model

**2.2 Formulation of the Model**

$$\left. \begin{aligned} \frac{dS}{dt} &= \mu(K-N) - \left(\frac{a_1\beta_1 A_1}{K} + \frac{a_2\beta_2 T_1}{K} + \frac{a_3\beta_3 W_1}{K}\right)S - \mu S \\ \frac{dI}{dt} &= \left(\frac{a_1\beta_1 A_1}{K} + \frac{a_2\beta_2 T_1}{K} + \frac{a_3\beta_3 W_1}{K}\right)S - (\gamma + d + \mu)I \\ \frac{dR}{dt} &= \gamma I - \mu R \\ \frac{dA_1}{dt} &= n_1 + b_1 A_1 - \frac{a_1 B_1 A_1 I}{K} - (c_1 \theta P + m)A_1 \\ \frac{dT_1}{dt} &= n_2 + b_2 T_1 - \frac{a_2 B_2 T_1 I}{K} - (c_2 \theta P + m)T_1 \\ \frac{dW_1}{dt} &= n_3 + b_3 W_1 - \frac{a_3 B_3 W_1 I}{K} - (m + P\theta c_3)W_1 \\ \frac{dA_2}{dt} &= \frac{a_1 B_1 A_1 I}{K} - (m + (1-c_1)\theta P)A_1 \\ \frac{dT_2}{dt} &= \frac{a_2 B_2 T_1 I}{K} - (m + (1-c_2)\theta P)T_1 \\ \frac{dW_2}{dt} &= \frac{a_3 B_3 W_1 I}{K} - (m + (1-c_3)\theta P)W_1 \\ \frac{dP}{dt} &= \nu + P(\omega - \delta) + \alpha_1 \theta P [c_1 A_1 + (1-c_1)A_1 + c_2 T_1 + (1-c_2)T_1 + c_3 W_1 + (1-c_3)W_1] \end{aligned} \right\}$$

$$\left. \begin{aligned} \mu(K-N) - f_1 - \mu S &= 0 \\ f_1 - (\gamma + d + \mu)I &= 0 \\ \gamma I - \mu R &= 0 \\ n_1 + b_1 A_1 - f_2 - (c_1 \theta P + m)A_1 &= 0 \\ n_2 + b_2 T_1 - f_3 - (c_2 \theta P + m)T_1 &= 0 \\ n_3 + b_3 W_1 - f_4 - (m + P\theta c_3)W_1 &= 0 \\ f_2 - (m + (1-c_1)\theta P)A_1 &= 0 \\ f_3 - (m + (1-c_2)\theta P)T_1 &= 0 \\ f_4 - (m + (1-c_3)\theta P)W_1 &= 0 \\ \nu + P(\omega - \delta) + \alpha_1 \theta P [c_1 A_1 + (1-c_1)A_1 + c_2 T_1 + (1-c_2)T_1 + c_3 W_1 + (1-c_3)W_1] &= 0 \end{aligned} \right\}$$

Where the forces of infection  $f_1 = \left(\frac{a_1\beta_1 A_1}{K} + \frac{a_2\beta_2 T_1}{K} + \frac{a_3\beta_3 W_1}{K}\right)S$ ,

**3. Results and Discussion**

**3.1 Equilibrium Points of the Model**

At the steady states, all the equations of the system (1.1) equate to zero. Now, the system of equation becomes

$$f_2 = \frac{a_1 B_1 A_s I}{K}, \quad f_3 = \frac{a_2 B_2 T_s I}{K} \quad \text{and}$$

$$f_4 = \frac{a_3 B_3 W_s I}{k} \quad \text{respectively}$$

**3.1.1 Disease-Free Equilibrium** ( $E_1^*$ )

In the absence of a disease, there will be no need of biological control mechanism, and then  $I = A_I = T_I = W_I = P = 0$ . Thus, the system of equation has disease-free equilibrium  $E_1^* = \{S^*, I^*, R^*, A_s^*, T_s^*, W_s^*, A_I^*, T_I^*, W_I^*, P^*\}$ .

$$E_1^* = \left\{ (K - N), 0, 0, \frac{n_1}{m - b_1}, \frac{n_2}{m - b_2}, \frac{n_3}{m - b_3}, 0, 0, 0, 0 \right\}$$

**3.1.2 Endemic Equilibrium** ( $E_2^{\#}$ )

Assume that the diseases occur. Then, the system (1.2) can be written as

$$\left. \begin{aligned} \mu(K - N) - f_1^{\#} - \mu S^{\#} &= 0 \\ f_1^{\#} - (\gamma + d + \mu) I^{\#} &= 0 \\ \gamma I^{\#} - \mu R^{\#} &= 0 \\ n_1 + b_1 A_s^{\#} - f_2^{\#} - (c_1 \theta P^{\#} + m) A_I^{\#} &= 0 \\ n_2 + b_2 T_s^{\#} - f_3^{\#} - (c_2 \theta P^{\#} + m) T_I^{\#} &= 0 \\ n_3 + b_3 W_s^{\#} - f_4^{\#} - (c_3 \theta P^{\#} + m) W_I^{\#} &= 0 \\ f_2^{\#} - (m + (1 - c_1) \theta P^{\#}) A_I^{\#} &= 0 \\ f_3^{\#} - (m + (1 - c_2) \theta P^{\#}) T_I^{\#} &= 0 \\ f_4^{\#} - (m + (1 - c_3) \theta P^{\#}) W_I^{\#} &= 0 \\ v + P^{\#}(\omega - \delta) + \alpha \theta P^{\#} [c_1 A_s^{\#} + (1 - c_1) A_I^{\#} + c_2 T_s^{\#} + (1 - c_2) T_I^{\#} + c_3 W_s^{\#} + (1 - c_3) W_I^{\#}] &= 0 \end{aligned} \right\}$$

On solving the equations, we have endemic equilibrium

$$E_2^{\#} = \{S^{\#}, I^{\#}, R^{\#}, A_s^{\#}, T_s^{\#}, W_s^{\#}, A_I^{\#}, T_I^{\#}, W_I^{\#}, P^{\#}\}$$

where

$$S^{\#} = \frac{\mu K (K - N)}{a_1 \beta_1 A_I^{\#} + a_2 \beta_2 T_I^{\#} + a_3 \beta_3 W_I^{\#} + \mu K},$$

$$I^{\#} = \frac{\mu (K - N) (a_1 \beta_1 A_I^{\#} + a_2 \beta_2 T_I^{\#} + a_3 \beta_3 W_I^{\#})}{(d + \gamma + \mu) (a_1 \beta_1 A_I^{\#} + a_2 \beta_2 T_I^{\#} + a_3 \beta_3 W_I^{\#} + \mu K)},$$

$$R^{\#} = \frac{\gamma (K - N) (a_1 \beta_1 A_I^{\#} + a_2 \beta_2 T_I^{\#} + a_3 \beta_3 W_I^{\#})}{(d + \gamma + \mu) (a_1 \beta_1 A_I^{\#} + a_2 \beta_2 T_I^{\#} + a_3 \beta_3 W_I^{\#} + \mu K)},$$

$$A_s^{\#} = \frac{n_1 K}{a_1 B_1 I^{\#} - b_1 K + c_1 \theta P^{\#} K + m K},$$

$$T_s^{\#} = \frac{n_2 K}{a_2 B_2 I^{\#} - b_2 K + c_2 \theta P^{\#} K + m K},$$

$$W_s^{\#} = \frac{n_3 K}{a_3 B_3 I^{\#} - b_3 K + c_3 \theta P^{\#} K + m K},$$

$$A_I^{\#} = \frac{a_1 B_1 n_1 I^{\#}}{(m + (1 - c_1) \theta P^{\#}) (a_1 B_1 I^{\#} - b_1 K + (c_1 \theta P^{\#} + m) K)},$$

$$T_I^{\#} = \frac{a_2 B_2 n_2 I^{\#}}{(m + (1 - c_2) \theta P^{\#}) (a_2 B_2 I^{\#} - b_2 K + (c_2 \theta P^{\#} + m) K)},$$

$$W_I^{\#} = \frac{a_3 B_3 n_3 I^{\#}}{(m + (1 - c_3) \theta P^{\#}) (a_3 B_3 I^{\#} - b_3 K + (c_3 \theta P^{\#} + m) K)},$$

$$P^{\#} = \frac{v}{\delta + \alpha \theta [(c_1 A_I^{\#} - A_I^{\#}) + (c_2 T_I^{\#} - T_I^{\#}) + (c_3 W_I^{\#} - W_I^{\#})] - \alpha \theta (c_1 A_s^{\#} + c_2 T_s^{\#} + c_3 W_s^{\#}) - \omega}$$

**3.2 The Basic Reproduction Number** ( $R_0$ )

Using the Next Generation Matrix (NGM) method, we have

$$f = \begin{pmatrix} 0 & 0 & \frac{a_1 \beta_1 S}{K} & \frac{a_2 \beta_2 S}{K} & \frac{a_3 \beta_3 S}{K} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{a_1 B_1 A_s}{K} & 0 & 0 & 0 & 0 \\ \frac{a_2 B_2 T_s}{K} & 0 & 0 & 0 & 0 \\ \frac{a_3 B_3 W_s}{K} & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$v = \begin{pmatrix} \gamma + d + \mu & 0 & 0 & 0 & 0 \\ -\gamma & \mu & 0 & 0 & 0 \\ 0 & 0 & (1 - c_1) \theta P + m & 0 & 0 \\ 0 & 0 & 0 & (1 - c_2) \theta P + m & 0 \\ 0 & 0 & 0 & 0 & (1 - c_3) \theta P + m \end{pmatrix}$$

$$\begin{pmatrix} 0 & 0 & \frac{a_1\beta_1(K-N)}{mK} & \frac{a_2\beta_2(K-N)}{mK} & \frac{a_3\beta_3(K-N)}{mK} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{a_1B_1n_1}{(m-b_1)(\gamma+d+\mu)K} & 0 & 0 & 0 & 0 \\ \frac{a_2B_2n_2}{(m-b_2)(\gamma+d+\mu)K} & 0 & 0 & 0 & 0 \\ \frac{a_3B_3n_3}{(m-b_3)(\gamma+d+\mu)K} & 0 & 0 & 0 & 0 \end{pmatrix} \quad \begin{aligned} L_5 &= \frac{a_2B_2n_2}{K(m-b_2)}, & L_6 &= \frac{a_3B_3n_3}{K(m-b_3)}, \\ L_7 &= \frac{c_1\theta n_1}{m-b_1}, & L_8 &= \frac{c_2\theta n_2}{m-b_2}, & L_9 &= \frac{c_3\theta n_3}{m-b_3} \\ L_{10} &= \alpha_4\theta \left( \frac{c_1n_1}{m-b_1} + \frac{c_2n_2}{m-b_2} + \frac{c_3n_3}{m-b_3} \right) + \omega - \delta. \end{aligned}$$

Using the Sarrus' rule, the characteristic equation of (1.8) is given by

$$(-m-\lambda)^3(-\mu-\lambda)^2(-(\gamma+d+\mu)-\lambda)((b_1-m)-\lambda)((b_2-m)-\lambda)((b_3-m)-\lambda)(L_{10}-\lambda)=0 \quad (0.1)$$

$R_0$  is defined as the  $\rho$  (spectral radius) of (1.7), which is the dominant eigenvalue. Thus,

$$R_0 = \sqrt{\frac{(K-N)}{m(\gamma+d+\mu)K^2} \left( \frac{n_1\beta_1B_1a_1^2}{(m-b_1)} + \frac{n_2\beta_2B_2a_2^2}{(m-b_2)} + \frac{n_3\beta_3B_3a_3^2}{(m-b_3)} \right)}$$

$i=1,2,3, \lambda_i = (b_i - m) < 0$  provided that  $m > b_i$ . In the same way, for

### 3.3 Stability Analysis

#### 3.3.1 Local Stability of Disease-Free Equilibrium

**Theorem** The disease-free equilibrium point is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

**Proof** The Jacobian matrix of the system (1.1) evaluated at  $E_1$  is given by:

$$J_{(E_1)} = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & -L_1 & L_2 & L_3 & 0 \\ 0 & -(\gamma+d+\mu) & 0 & 0 & 0 & 0 & L_1 & L_2 & L_3 & 0 \\ 0 & \gamma & -\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -L_4 & 0 & -(m-b_1) & 0 & 0 & 0 & 0 & 0 & -L_7 \\ 0 & -L_5 & 0 & 0 & -(m-b_2) & 0 & 0 & 0 & 0 & -L_8 \\ 0 & -L_6 & 0 & 0 & 0 & -(m-b_3) & 0 & 0 & 0 & -L_9 \\ 0 & L_4 & 0 & 0 & 0 & 0 & -m & 0 & 0 & 0 \\ 0 & L_5 & 0 & 0 & 0 & 0 & 0 & -m & 0 & 0 \\ 0 & L_6 & 0 & 0 & 0 & 0 & 0 & 0 & -m & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & L_{10} \end{pmatrix}$$

where

$$\begin{aligned} L_1 &= \frac{a_1\beta_1(K-N)}{K}, \\ L_2 &= \frac{a_2\beta_2(K-N)}{K}, & L_3 &= \frac{a_3\beta_3(K-N)}{K}, \\ L_4 &= \frac{a_1B_1n_1}{K(m-b_1)}, \end{aligned}$$

Since all the parameters are positive. It is obvious that, for some factors  $((b_i - m) - \lambda)$

$$(-m-\lambda)^3, i=4,5,6, \quad \lambda_i = -m < 0$$

Furthermore, considering the factor  $(-\mu-\lambda)^2, i=7,8, \quad \lambda_i = -\mu < 0$ . And

for  $\lambda_9 = -(\gamma+d+\mu) < 0$ , the result is clear.

Similarly, for  $\lambda_{10} < 0$  the result is obtainable if the inequality

$$\alpha_4\theta \left( \frac{c_1n_1}{m-b_1} + \frac{c_2n_2}{m-b_2} + \frac{c_3n_3}{m-b_3} \right) + \omega < \delta$$

holds.

Thus, the disease-free equilibrium state is locally asymptotically stable.

#### 3.3.2 Global Stability of Disease-Free Equilibrium

To establish the global stability of the disease-free equilibrium (DFE) state, we construct a suitable Lyapunov function.

**Theorem** The disease-free equilibrium state is globally asymptotically stable if  $R_0 \leq 1$  and unstable if  $R_0 > 1$ .

**Proof**

To prove this theorem, we consider the following Lyapunov function:

$$L(t) = \kappa_1 \left( S - S^* - S^* \ln \frac{S}{S^*} \right) + \frac{1}{(\gamma + d + \mu)} I + \frac{1}{\gamma} R + \kappa_2 \left( A_S - A_S^* - A_S^* \ln \frac{A_S}{A_S^*} \right) + \kappa_3 \left( T_S - T_S^* - T_S^* \ln \frac{T_S}{T_S^*} \right) + \kappa_4 \left( W_S - W_S^* - W_S^* \ln \frac{W_S}{W_S^*} \right) + \frac{1}{m} (A_I + T_I + W_I)$$

where some constant  $\kappa_i > 0, i = 1, 2, 3, 4$  are to be determined.

First, it is not hard to see that  $L(t) = 0$  at DFE state  $E_1^* = (S^*, I^*, R^*, A_S^*, T_S^*, W_S^*, A_I^*, T_I^*, W_I^*)$ .

To establish that  $L(t) > 0$  for all  $(S^*, I^*, R^*, A_S^*, T_S^*, W_S^*, A_I^*, T_I^*, W_I^*)$ , it is enough to notice that

$$\kappa_1 S^* \left( \frac{S}{S^*} - 1 - \ln \frac{S}{S^*} \right) > 0$$

, because the function  $f(x) = x - 1 - \ln x$  achieves a global minimum at  $x = 1$  and  $f(1) = 0$  for all  $x > 0$  and  $x \neq 1$ . So, the first term is positive. And the remaining three terms in logarithmic form are also clearly positive.

Secondly, the coefficients are chosen in such a way that the positive terms in the function  $L(t)$  cancels with negative terms in the system of equations (1.1). Additionally,

$L(S, I, R, A_S, T_S, W_S, A_I, T_I, W_I)$  is also clearly radially unbounded. Now, we take the derivative of  $L(t)$  with respect to  $t$ :

$$\frac{dL}{dt} = \kappa_1 \left( 1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \frac{1}{(\gamma + d + \mu)} \frac{dI}{dt} + \frac{1}{\gamma} \frac{dR}{dt} + \kappa_2 \left( 1 - \frac{A_S^*}{A_S} \right) \frac{dA_S}{dt} + \kappa_3 \left( 1 - \frac{T_S^*}{T_S} \right) \frac{dT_S}{dt} + \kappa_4 \left( 1 - \frac{W_S^*}{W_S} \right) \frac{dW_S}{dt} + \frac{1}{m} \left( \frac{dA_I}{dt} + \frac{dT_I}{dt} + \frac{dW_I}{dt} \right)$$

$$\begin{aligned} \frac{dL}{dt} = & \kappa_1 \left( 1 - \frac{S^*}{S} \right) \left( \mu(K - N) - \left( \frac{a_1 \beta_1 A_I}{K} + \frac{a_2 \beta_2 T_I}{K} + \frac{a_3 \beta_3 W_I}{K} \right) S - \mu S \right) \\ & + \frac{1}{(\gamma + d + \mu)} \left( \left( \frac{a_1 \beta_1 A_I}{K} + \frac{a_2 \beta_2 T_I}{K} + \frac{a_3 \beta_3 W_I}{K} \right) S - (\gamma + d + \mu) I \right) \\ & + \frac{1}{\gamma} (\gamma I - \mu R) + \kappa_2 \left( 1 - \frac{A_S^*}{A_S} \right) \left( n_1 + b_1 A_S - \frac{a_1 B_1 A_S I}{K} - m A_S \right) \\ & + \kappa_3 \left( 1 - \frac{T_S^*}{T_S} \right) \left( n_2 + b_2 T_S - \frac{a_2 B_2 T_S I}{K} - m T_S \right) + \kappa_4 \left( 1 - \frac{W_S^*}{W_S} \right) \left( n_3 + b_3 W_S - \frac{a_3 B_3 W_S I}{K} - m W_S \right) \\ & + \frac{1}{m} \left( \frac{a_1 B_1 A_S I}{K} + \frac{a_2 B_2 T_S I}{K} + \frac{a_3 B_3 W_S I}{K} - (m A_I + m T_I + m W_I) \right) \end{aligned}$$

$$\begin{aligned} \frac{dL}{dt} = & -\mu \kappa_1 \frac{(S - S^*)^2}{S} + \kappa_1 \frac{a_1 \beta_1 A_I (N - S)}{K} + \kappa_1 \frac{a_2 \beta_2 T_I (N - S)}{K} \\ & + \kappa_1 \frac{a_3 \beta_3 W_I (N - S)}{K} - \kappa_1 (a_1 \beta_1 A_I + a_2 \beta_2 T_I + a_3 \beta_3 W_I) \\ & + \frac{1}{(\gamma + d + \mu)} \left( \left( \frac{a_1 \beta_1 A_I}{K} + \frac{a_2 \beta_2 T_I}{K} + \frac{a_3 \beta_3 W_I}{K} \right) S - (\gamma + d + \mu) I \right) \\ & + \frac{1}{\gamma} (\gamma I - \mu R) - \kappa_2 \frac{\{A_S (m - b_1) - n_1\} \{A_S K (m - b_1) + a_1 B_1 A_S I - n_1 K\}}{A_S K (m - b_1)} \\ & - \kappa_3 \frac{\{T_S (m - b_2) - n_2\} \{T_S K (m - b_2) + a_2 B_2 T_S I - n_2 K\}}{T_S K (m - b_2)} \\ & - \kappa_4 \frac{\{W_S (m - b_3) - n_3\} \{W_S K (m - b_3) + a_3 B_3 W_S I - n_3 K\}}{W_S K (m - b_3)} \\ & + \frac{1}{m} \left( \frac{a_1 B_1 A_S I}{K} + \frac{a_2 B_2 T_S I}{K} + \frac{a_3 B_3 W_S I}{K} - m (A_I + T_I + W_I) \right) \end{aligned}$$

Thus, for some suitable values of  $\kappa_i > 0, i = 1, 2, 3, 4$  it implies  $L'(t)$  being negative whenever  $R_0 \leq 1$  and  $L'(t) = 0$  if and only if  $S = S^*, A_S = A_S^*, T_S = T_S^*, W_S = W_S^*$  and  $I = R = A_I = T_I = W_I = 0$ .

Consequently, the largest compact invariant set in

$$\left\{ (S^*, I^*, R^*, A_S^*, T_S^*, W_S^*, A_I^*, T_I^*, W_I^*) \in \Omega, L' = 0 \right\},$$

when  $R_0 \leq 1$ , is the singleton  $\{E_1^*\}$  which is the disease free equilibrium state. Hence, according to LaSalle's invariance principle [16], this guarantees that the DFE state is globally asymptotically stable (GAS) in  $\Omega$ .

### 3.3.3 Local Stability of Endemic Equilibrium Theorem 4

The disease endemic equilibrium point is locally asymptotically stable if  $R_0 > 1$  and unstable if  $R_0 < 1$ .

**Proof :**

The Jacobian of the system (1.1) at  $E_2^{\#}$  as presented below

$$J_{(E_2^\#)} = \begin{pmatrix} -M_1 & 0 & 0 & 0 & 0 & 0 & -M_{21} & -M_{22} & -M_{23} & 0 \\ M_{10} & -M_2 & 0 & 0 & 0 & 0 & M_{21} & M_{22} & M_{23} & 0 \\ 0 & \gamma & -\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -M_{11} & 0 & M_3 & 0 & 0 & 0 & 0 & 0 & M_{27} \\ 0 & -M_{12} & 0 & 0 & M_4 & 0 & 0 & 0 & 0 & M_{28} \\ 0 & -M_{13} & 0 & 0 & 0 & M_5 & 0 & 0 & 0 & M_{29} \\ 0 & M_{11} & 0 & M_{15} & 0 & 0 & M_6 & 0 & 0 & M_{30} \\ 0 & M_{12} & 0 & 0 & M_{17} & 0 & 0 & M_7 & 0 & M_{31} \\ 0 & M_{13} & 0 & 0 & 0 & M_{19} & 0 & 0 & M_8 & M_{32} \\ 0 & 0 & 0 & M_{16} & M_{18} & M_{20} & M_{24} & M_{25} & M_{26} & M_9 \end{pmatrix}$$

where

$$M_1 = \frac{a_1\beta_1 A_I^\# + a_2\beta_2 T_I^\# + a_3\beta_3 W_I^\# + \mu K}{K},$$

$$M_3 = b_1 - \frac{a_1 B_1 I^\# + c_1 \theta K P^\# + m K}{K},$$

$$M_5 = b_3 - \frac{a_3 B_3 I^\# + c_3 \theta K P^\# + m K}{K},$$

$$M_6 = (c_1 - 1)\theta P^\# - m, \quad M_7 = (c_2 - 1)\theta P^\# - m,$$

$$M_8 = (c_3 - 1)\theta P^\# - m,$$

$$M_9 = \alpha_4 \theta \left[ (1-c_1)A_I^\# + (1-c_2)T_I^\# + (1-c_3)W_I^\# + c_1 A_s^\# + c_2 T_s^\# + c_3 W_s^\# \right] - \delta + \omega$$

$$M_{10} = \frac{a_1\beta_1 A_I^\# + a_2\beta_2 T_I^\# + a_3\beta_3 W_I^\#}{K},$$

$$M_{11} = \frac{a_1 B_1 A_s^\#}{K}, \quad M_{12} = \frac{a_2 B_2 T_s^\#}{K},$$

$$M_{13} = \frac{a_3 B_3 W_s^\#}{K},$$

$$M_{15} = \frac{a_1 B_1 I^\#}{K}, \quad M_{16} = \alpha_4 c_1 \theta P^\#,$$

$$M_{17} = \frac{a_2 B_2 I^\#}{K}, \quad M_{18} = \alpha_4 c_2 \theta P^\#,$$

$$M_{19} = \frac{a_3 B_3 I^\#}{k},$$

$$M_{20} = \alpha_4 c_3 \theta P^\#, \quad M_{21} = \frac{a_1 \beta_1 S^\#}{K},$$

$$M_{22} = \frac{a_2 \beta_2 S^\#}{K}, \quad M_{23} = -\frac{a_3 \beta_3 S^\#}{K},$$

$$M_{24} = \alpha_4 (1-c_1)\theta P^\#, \quad M_{25} = \alpha_4 (1-c_2)\theta P^\#,$$

$$M_{26} = \alpha_4 (1-c_3)\theta P^\#, \quad M_{27} = -c_1 \theta A_s^\#,$$

$$M_{28} = -c_2 \theta T_s^\#, \quad M_{29} = -c_3 \theta W_s^\#,$$

$$M_{30} = (c_1 - 1)\theta A_I^\#, \quad M_{31} = (c_2 - 1)\theta T_I^\#$$
 and
$$M_{32} = (c_3 - 1)\theta W_I^\#.$$

By Sarrus' rule, the characteristic equation of  $J(\lambda)$  evaluated at  $(E_2^\#)$  is  $(-M_1 - \lambda)(-M_2 - \lambda)(-\mu - \lambda)(M_3 - \lambda)(M_4 - \lambda)(M_5 - \lambda)(M_6 - \lambda)(M_7 - \lambda)(M_8 - \lambda)(M_9 - \lambda) = 0$

It could be observed that  $-M_1, -M_2$  and  $-\mu$  have obviously satisfied the negativity requirement of stability. Additionally, the expressions  $M_3, M_4$  and  $M_5$  have equally satisfied the negativity condition provided that

$$b_1 < \frac{a_1 B_1 I^\# + c_1 \theta K P^\# + m K}{K},$$

$$b_2 < \frac{a_2 B_2 I^\# + c_2 \theta K P^\# + m K}{K}$$
 and
$$b_3 < \frac{a_3 B_3 I^\# + c_3 \theta K P^\# + m K}{K}$$
 respectively. So

also is  $M_6, M_7$  and  $M_8$  provided the inequalities  $m > (c_1 - 1)\theta P^\#, m > (c_2 - 1)\theta P^\#$  and  $m > (c_3 - 1)\theta P^\#$  hold respectively. And

lastly, for  $M_9$  we suppose that  $\alpha_4 \theta \left[ (1-c_1)A_I^\# + (1-c_2)T_I^\# + (1-c_3)W_I^\# + c_1 A_s^\# + c_2 T_s^\# + c_3 W_s^\# \right] > \delta + \omega$  is satisfied. Hence, the endemic equilibrium is locally asymptotically stable.

### 3.3.4 Global Stability of Endemic Equilibrium Theorem

If  $R_0 > 1$  the disease endemic equilibrium point of the model (1.1) is globally asymptotically stable.

**Proof:**

To establish the global stability of the disease endemic equilibrium state, we construct the following positive Lyapunov function  $V(t)$  by adopting the idea in [17] as follows;

$$V(t) = (S - S^\# \ln S) + (I - I^\# \ln I) + (R - R^\# \ln R) + (A_s - A_s^\# \ln A_s) + (T_s - T_s^\# \ln T_s) + (W_s - W_s^\# \ln W_s) + (A_I - A_I^\# \ln A_I) + (T_I - T_I^\# \ln T_I) + (W_I - W_I^\# \ln W_I) \tag{0.2}$$

By direct calculation of the derivative of  $V(t)$  along the solutions curves gives



$$\begin{aligned} \frac{dV}{dt}(S^\#, I^\#, R^\#, A_S^\#, T_S^\#, W_S^\#, A_I^\#, T_I^\#, W_I^\#, P^\#) &= \left(1 - \frac{S^\#}{S}\right) \frac{dS}{dt} + \left(1 - \frac{I^\#}{I}\right) \frac{dI}{dt} + \left(1 - \frac{R^\#}{R}\right) \frac{dR}{dt} \\ &+ \left(1 - \frac{A_S^\#}{A_S}\right) \frac{dA_S}{dt} + \left(1 - \frac{T_S^\#}{T_S}\right) \frac{dT_S}{dt} + \left(1 - \frac{W_S^\#}{W_S}\right) \frac{dW_S}{dt} + \left(1 - \frac{A_I^\#}{A_I}\right) \frac{dA_I}{dt} + \left(1 - \frac{T_I^\#}{T_I}\right) \frac{dT_I}{dt} + \left(1 - \frac{W_I^\#}{W_I}\right) \frac{dW_I}{dt} \end{aligned} \tag{0.3}$$

Consequently,

$$\begin{aligned} \frac{dV}{dt} &= \left(\frac{S-S^\#}{S}\right) \frac{dS}{dt} + \left(\frac{I-I^\#}{I}\right) \frac{dI}{dt} + \left(\frac{R-R^\#}{R}\right) \frac{dR}{dt} + \left(\frac{A_S-A_S^\#}{A_S}\right) \frac{dA_S}{dt} + \left(\frac{T_S-T_S^\#}{T_S}\right) \frac{dT_S}{dt} \\ &+ \left(\frac{W_S-W_S^\#}{W_S}\right) \frac{dW_S}{dt} + \left(\frac{A_I-A_I^\#}{A_I}\right) \frac{dA_I}{dt} + \left(\frac{T_I-T_I^\#}{T_I}\right) \frac{dT_I}{dt} + \left(\frac{W_I-W_I^\#}{W_I}\right) \frac{dW_I}{dt} \end{aligned} \tag{0.4}$$

Substituting  $S = (S - S^\#)$ ,  $I = (I - I^\#)$ ,  
 $R = (R - R^\#)$ ,  $A_S = (A_S - A_S^\#)$ ,  
 $T_S = (T_S - T_S^\#)$ ,  $W_S = (W_S - W_S^\#)$ ,  
 $A_I = (A_I - A_I^\#)$ ,  $T_I = (T_I - T_I^\#)$ , and  
 $W_I = (W_I - W_I^\#)$  into (1.1)

$$\begin{aligned} \frac{dS}{dt} &= \mu(K - N) - \left(\frac{a_1\beta_1(A_I - A_I^\#)}{K} + \frac{a_2\beta_2(T_I - T_I^\#)}{K} + \frac{a_3\beta_3(W_I - W_I^\#)}{K}\right)(S - S^\#) - \mu(S - S^\#) \\ \frac{dI}{dt} &= \left(\frac{a_1\beta_1(A_I - A_I^\#)}{K} + \frac{a_2\beta_2(T_I - T_I^\#)}{K} + \frac{a_3\beta_3(W_I - W_I^\#)}{K}\right)(S - S^\#) - (\gamma + d + \mu)(I - I^\#) \\ \frac{dR}{dt} &= \gamma(I - I^\#) - \mu(R - R^\#) \\ \frac{dA_S}{dt} &= n_1 + b_1(A_S - A_S^\#) - \frac{a_1B_1(A_S - A_S^\#)(I - I^\#)}{K} - (c_1\theta(P - P^\#) + m)(A_S - A_S^\#) \\ \frac{dT_S}{dt} &= n_2 + b_2(T_S - T_S^\#) - \frac{a_2B_2(T_S - T_S^\#)(I - I^\#)}{K} - (c_2\theta(P - P^\#) + m)(T_S - T_S^\#) \\ \frac{dW_S}{dt} &= n_3 + b_3(W_S - W_S^\#) - \frac{a_3B_3(W_S - W_S^\#)(I - I^\#)}{K} - (m + (P - P^\#)\theta c_3)(W_S - W_S^\#) \\ \frac{dA_I}{dt} &= \frac{a_1B_1(A_S - A_S^\#)(I - I^\#)}{K} - (m + (1 - c_1)\theta(P - P^\#))(A_I - A_I^\#) \\ \frac{dT_I}{dt} &= \frac{a_2B_2(T_S - T_S^\#)(I - I^\#)}{K} - (m + (1 - c_2)\theta(P - P^\#))(T_I - T_I^\#) \\ \frac{dW_I}{dt} &= \frac{a_3B_3(W_S - W_S^\#)(I - I^\#)}{K} - (m + (1 - c_3)\theta(P - P^\#))(W_I - W_I^\#) \end{aligned}$$

It follows that

$$\begin{aligned} \frac{dV}{dt} &= \left(\frac{S-S^\#}{S}\right) \left\{ -\left(\frac{a_1\beta_1(A_I - A_I^\#)}{K} + \frac{a_2\beta_2(T_I - T_I^\#)}{K} + \frac{a_3\beta_3(W_I - W_I^\#)}{K}\right)(S - S^\#) \right\} \\ &+ \left(\frac{I-I^\#}{I}\right) \left\{ \left(\frac{a_1\beta_1(A_I - A_I^\#)}{K} + \frac{a_2\beta_2(T_I - T_I^\#)}{K} + \frac{a_3\beta_3(W_I - W_I^\#)}{K}\right)(S - S^\#) - (\gamma + d + \mu)(I - I^\#) \right\} \\ &+ \left(\frac{R-R^\#}{R}\right) \left\{ \gamma(I - I^\#) - \mu(R - R^\#) \right\} \\ &+ \left(\frac{A_S-A_S^\#}{A_S}\right) \left\{ n_1 + b_1(A_S - A_S^\#) - \frac{a_1B_1(A_S - A_S^\#)(I - I^\#)}{K} - (c_1\theta(P - P^\#) + m)(A_S - A_S^\#) \right\} \\ &+ \left(\frac{T_S-T_S^\#}{T_S}\right) \left\{ n_2 + b_2(T_S - T_S^\#) - \frac{a_2B_2(T_S - T_S^\#)(I - I^\#)}{K} - (c_2\theta(P - P^\#) + m)(T_S - T_S^\#) \right\} \\ &+ \left(\frac{W_S-W_S^\#}{W_S}\right) \left\{ n_3 + b_3(W_S - W_S^\#) - \frac{a_3B_3(W_S - W_S^\#)(I - I^\#)}{K} - (m + (P - P^\#)\theta c_3)(W_S - W_S^\#) \right\} \\ &+ \left(\frac{A_I-A_I^\#}{A_I}\right) \left\{ \frac{a_1B_1(A_S - A_S^\#)(I - I^\#)}{K} - (m + (1 - c_1)\theta(P - P^\#))(A_I - A_I^\#) \right\} \\ &+ \left(\frac{T_I-T_I^\#}{T_I}\right) \left\{ \frac{a_2B_2(T_S - T_S^\#)(I - I^\#)}{K} - (m + (1 - c_2)\theta(P - P^\#))(T_I - T_I^\#) \right\} \\ &+ \left(\frac{W_I-W_I^\#}{W_I}\right) \left\{ \frac{a_3B_3(W_S - W_S^\#)(I - I^\#)}{K} - (m + (1 - c_3)\theta(P - P^\#))(W_I - W_I^\#) \right\} \end{aligned}$$

Collecting the positive and the negative terms together in (1.16) we have

$$\begin{aligned} \frac{dV}{dt} &= K^+ - K^- \text{ where } K^+, K^- \text{ refer to sum of all positive and negative terms in (1.16)} \\ &\text{respectively; hence, } \frac{dV}{dt} \text{ is negative definite,} \\ &\text{if } K^+ < K^- \text{ meaning that } \frac{dV}{dt} < 0. \text{ It follows} \\ &\frac{dV}{dt} = 0 \end{aligned}$$

therefore that  $\frac{dV}{dt} = 0$  if and only if  $S = S^\#, I = I^\#, R = R^\#, A_S = A_S^\#, T_S = T_S^\#, W_S = W_S^\#, A_I = A_I^\#, T_I = T_I^\#$  and  $W_I = W_I^\#$ .

Therefore the largest compact invariant set in  $\left\{ (S^\#, I^\#, R^\#, A_S^\#, T_S^\#, W_S^\#, A_I^\#, T_I^\#, W_I^\#) \in \Omega : \frac{dV}{dt} = 0 \right\}$

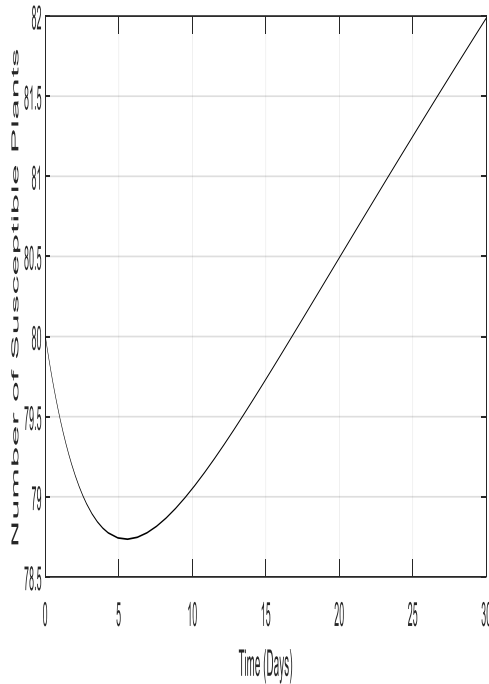
is the singleton  $\{E_2^*\}$ . By LaSalle's invariant principle, then it implies that  $E_2^{**}$  is globally asymptotically stable (GAS) in  $\Omega$  if  $K^+ < K^-$

**3.3.5 Numerical Results**

The model has ten compartments; thus, it will be jam-packed to present all the numerical experiment at once. Therefore, we hereby present the results based on interest about population categories as follows:

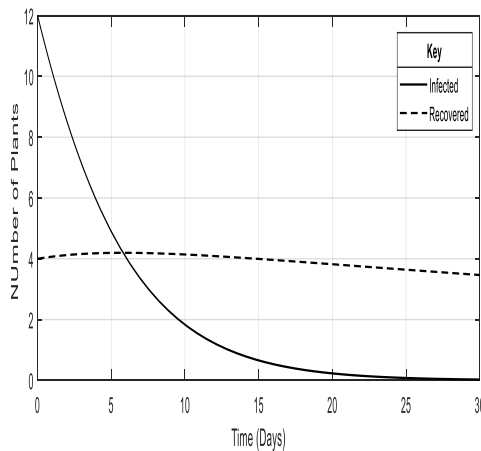
**Experiment One:**

An experiment to examine the long term effect of biological control on the population of susceptible solanaceous plants.



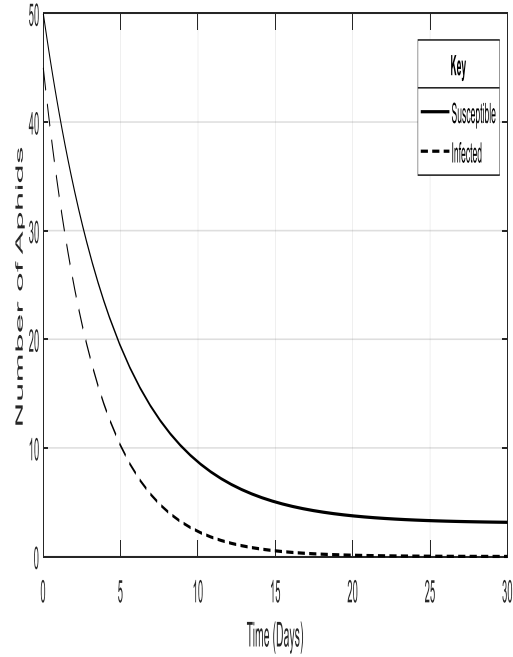
**Experiment Two**

An experiment to study the long term effect of biological control on the populations of infected and recovered solanaceous plants.



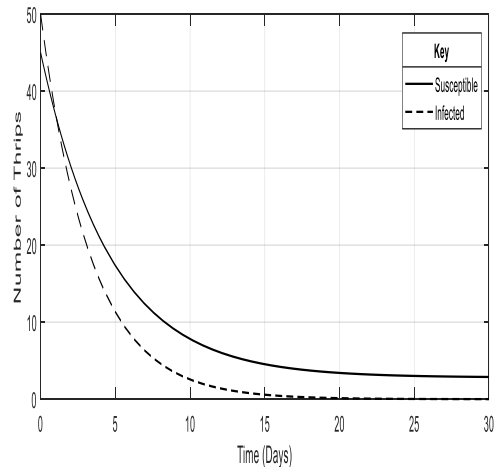
**Experiment Three:**

An experiment to explore the efficacy of using biological control measure on the population of aphids.



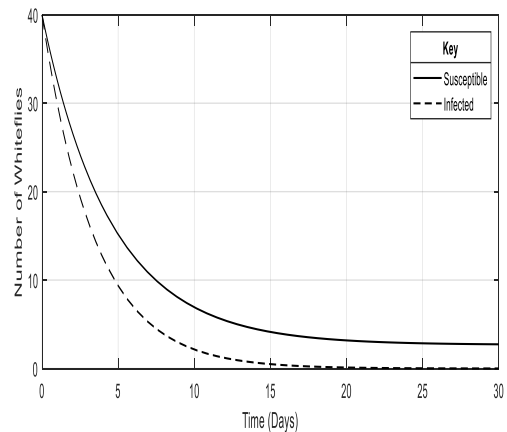
**Experiment Four:**

An experiment to explore the efficacy of using biological control measure on the population of thri



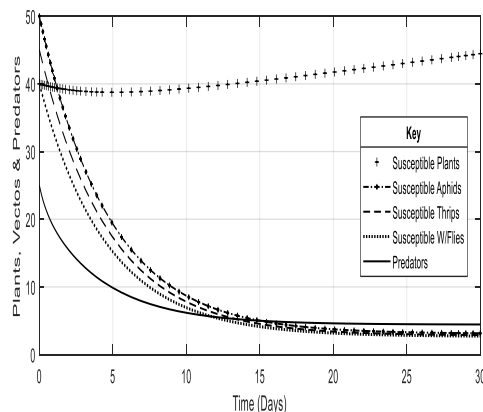
**Experiment Five:**

An experiment to explore the efficacy of using biological control measure on the population of whiteflies.



**Experiment Six:**

An experiment to investigate the efficiency of using ladybugs beetle as biological control agent for solanaceous vegetable plants.



The result of the first experiment indicates that, the number of susceptible solanaceous plants will drop initially and then re-bounce back and to grow continually until it reaches the farm carrying capacity. This is informed by constant replanting of plants and due to effect of reduction in forces of infection as a result of biological control strategy. Furthermore, the second experiment suggests that the biological control strategy is indeed very effective as it wipes away the infected plants within a month.

In the third, fourth and fifth experiments, the number of infected aphids, thrips and whiteflies respectively will tends to zero within one month signifying an important landmark of how effective the biological control mechanism works. In general, every good biological control strategy must preserve a number of susceptible aphids, thrips and whiteflies at least below certain acceptable economic threshold as vectors are needed for pollination purposes. This model achieves this requirement even when the number of infected thrips is more than susceptible ones and with equal number of susceptible and infected whiteflies. And lastly, experiment six guarantees that ladybugs will co-exist with aphids, thrips and whiteflies in solanaceous vegetable plants successfully having got rid of the infected solanaceous plants, eradicated infected aphids, thrips and whiteflies respectively.

**4. Conclusion**

In this study, we modified Jackson and Chen-Charpentier (2018) by considering specific Vector-Borne Viral Diseases VBVDs and by extending the vector pests into three classes. Our focus is about how biological control can be achieved through theoretically research using mathematical modeling technique. Method of

linearization was employed and the outcome of the analytical result shows that, both DFE and DEE are locally and globally asymptotically stable whenever  $R_0 < 1$  and  $R_0 \geq 1$  respectively. The results of the numerical experiments carried out on the model indicate that use ladybug beetles as biological control agents in tackling aphids, thrips and whiteflies within solanaceous vegetable plants will result into fruition.

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**Conflict of interest**

The authors declare no conflict of interest.

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