International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

Mathematical Modelling And Bifurcation Analysis Of Vector-Borne Diseases Of Crop Biomass

Vijai Shanker Verma[1], Archana Singh Bhadauria[2], Manik[3*], Ramwant Gupta[4]
[1,2,3]Department of Mathematics and Statistics, Deen Dayal Upadhyaya Gorakhpur University, Gorakhpur, India
[4]Department of Botany, Deen Dayal Upadhyaya Gorakhpur University, Gorakhpur, India
Email: vsverma.mathstat@ddugu.ac.in[1], archana.mathstat@ddugu.ac.in[2],ramwant.bot@ddugu.ac.in[4]
*Corresponding Author: Manik, manik.student@ddugu.ac.in[3]

Abstract
This study presents mathematical modelling and bifurcation analysis of vector-borne crop diseases by incorporating non-linear saturation rate of Holling type II. A mathematical model is developed by taking into account the logistic crop growth and vector dynamics. Before showing the existence of two equilibrium points (the disease-free and the endemic), the basic properties of the model are discussed and then the expression for basic reproduction number is obtained by using the next generation matrix method. Furthermore, bifurcation analysis is done by using the centre manifold theory. Sensitivity analysis is performed for the basic reproduction number which is shown with the help of a bar chart. Model parameters with positive sensitivity indices significantly impact the spread of crop diseases, while those with negative indices have a lesser effect. Numerical simulations are done and the results are displayed graphically to justify the analytical findings. This model aids in developing strategies to control vector-borne diseases in crops, enhancing productivity and food security.

Keywords: *Holling type II; Logistic growth; Insect vectors; Sensitivity Analysis.*

1. INTRODUCTION

Since the beginning of agriculture, humans have acquired and developed their knowledge to produce agricultural crops (i.e. food) more efficiently and to enhance the crop yields, while promoting the development of the human society. With an increasing world population, humans need the increased amount of food along with other eatables and usable in which crop production plays an important role in providing sufficient food security. Diseases of agricultural crops cause substantial economic losses and reduce food security at household, national and global levels, if not managed [1–3]. Diseases of crops pose a worldwide challenge to optimal food production and food security. To reduce the impacts of diseases in crop production, chemical sprays are mostly use to eliminate disease-causing pathogens found in the soil and different parts of crops [4-7]. For a number of years, pesticides/insecticides were developed as a solution to combat the spread of pests and diseases. Unfortunately, we now know that most of these pesticides have caused a lot of damage to the crops. Consequently, there is need to develop sustainable approaches to maintain yields and reduce the use of chemical products as much as possible in order to protect the biodiversity, decrease the risk of cancer or other diseases and also to protect our Earth for the future generations [8]. Vector-borne diseases have co-evolved with crops for millennia, making them an essential component of agro-ecosystems. There is a complicated web of mutual relationships between the pests and diseases that affect cultivated crops. There are two primary categories of procedures that can be employed to deal with these systems; these categories correspond to the scientific fields where modelling has evolved in a wide range of ways. The first group deals with the dynamics of pathogen populations and includes Pests and Disease Models (PDM), which allow crops to grow both temporally and spatially. The second group deals with the agricultural losses and concentrates on how host-pathogen interactions affect crop biomass/yield productivity and physiological processes. Physical, biological, social, and economic factors all have a significant impact on these two major categories of processes, while growing the crops biomass/yield. The estimation of the effects of pests and diseases on agricultural production is a crucial aspect in the formulation and evaluation of scenarios that influence farmers income and food security. There has been a transition in the type of models required for making quantitative assessments of yield loss, necessitating models with wider applicability. This shift is driven by the need to consider the effects of climate change and the desire to enhance the capability of providing global estimates. To fulfil these dual requirements, investigators encounter challenges such as the scarcity of reference data and the necessity to enhance the robustness and applicability of simulation models under varying conditions. Historically, barriers such as the intricacy of PDM and

the absence of standardized protocols for data collection, model development, and evaluation have hindered the creation of comprehensive modelling tools and a unified community focused on pest and disease modelling. Despite the extensive knowledge available regarding pest and disease modelling, as well as crop modelling within scientific circles, the exchange of this knowledge remains relatively limited [1].

To understand the complexity of crop diseases, multidisciplinary approach integrating biological and mathematical expertise must be taken. The study of eco-epidemiological models allows us to evaluate the effectiveness of different methods of disease and vector control.

It is well known that mathematics is incredibly important for understanding the whole world. It helps us in assessing the effects of different events and make wise decisions for policies. Using mathematical models (more specifically, epidemiological models), scientists can learn about how diseases spread and suggest good ways to control them.

Epidemiological modelling is an important tool to study diseases in crops and exhaustive research has been designed in this area [9,10]. Epidemiological models can identify knowledge or data gap and hence prioritize further research efforts [9]. In this connection, Tang et al. [11] have proposed a mathematical model for crop population with aims to eradicate infected crops or maintain the number of infected crops below the economic threshold. Gao et al. [12] have developed a mathematical model for crop disease due to virus with periodic environment and pulse rouging and have reported that when the infection rate is high, it may be impossible to eradicate the disease by simply rouging the infectious crops. Jackson et al. [13] have presented a model, in which they have considered the interaction between crops, virus and the insect vector that transfers the virus from one crop to another crop. There are many ways that crop viruses interact with the insect vectors and make crops infected with viruses. These infections may be harmful not only to the crops themselves but also to the ecosystem that depends on them. Such infections can have a negative impact on crop production and can create a challenge for human survival and the complete ecosystem. Viruses need a method of transportation to spread from one crop to another crops. Typically, an insect vector is the mode of crop virus transmission. An insect during movement must come in contact with the infected crops, usually by feeding on it, obtain the virus and transmit the virus to another disease resistant crops.

In view of the above, we propose to study a compartmental model to investigate the interaction between crops, virus and insect vectors. We shall take non-linear saturation constant rate of the Holling type II and logistic growth of crop biomass [14]. Non-linear saturation constant function represents saturation in the interaction of vectors with disease resistant crops due to application of various control strategies like fungicides, implementing cultural practices to inhibits fungal growth, etc [15]. We create the compartmental model by considering four compartments which are put into two groups, crop biomass/yield and vectors. The description of the proposed mathematical model is summarised below:

DESCRIPTION OF MATHEMATICAL MODEL

Four compartments are taken into consideration in two disjoint classes of crop biomass/yield and vectors. The crop biomass $N_c(t)$ is divided into two sub-classes; first the disease resistant crop biomass/yield $R_c(t)$ and second the infected crop biomass/yield $I_c(t)$. Thus, the crop biomass $N_c(t)$ at any time t is given by $N_c(t) = R_c(t) + I_c(t)$. Again, the vector population $M_v(t)$ is divided into two sub-classes: namely susceptible vectors $S_v(t)$ and infected vectors $I_v(t)$. Thus, the vector population $M_v(t)$ at any time t is given by $M_v(t) = S_v(t) + I_v(t)$. The recovered vector class is not included in this model, since infected vectors do not get damaged or spoiled due to the infection. Disease resistant crops do not have the disease but could acquire the disease if infected with the virus. The infected crops have the virus but can not directly transmit to the disease resistant crops. Additionally, since the infected crops could damage from the viral infection, their damage rate is higher than that of crops that do not have the virus. We also assume that as soon as a crop damages either from the infection or from a natural death, it is immediately replaced with a new crop by beneficiaries. Thus, it is reasonable to assume that the crop biomass/yield remains fixed, say K_c which is the carrying capacity for the crops growing on specific land. The insects do not have the virus but can obtain the virus if they come in contact with an infected crops. Infected insect vector can transmit the virus to disease resistant crops through contact. We assume no vertical transmission of the virus with neither crops nor vectors. Moreover, we assume that the virus does not harm the vector and thus the vector does not defend against the virus and it retains the virus for the rest of its life [3,10,16].

International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

The compartmental diagram of the model is shown in **Figure 1**. In the flow diagram, the solid lines represent an individual moving from one class to the next, whereas the dashed lines indicate contact between the two classes. A list of model parameters with description is summarized in **Table 1**.

The proposed compartmental model is developed on the basis of following assumptions:

(i) The disease resistant crops are assumed to be generated by the logistic growth rate $r(R_c + I_c)\left(1 - \frac{R_c+I_c}{K_c}\right)$. It is further assumed that the disease resistant crops decrease due to infection transmission rate with infected vectors at a rate $\frac{\alpha I_v R_c}{1+\beta I_v}$. The growth of disease resistant crops is further reduced by dR_c. Due to death of infected crops due to disease, it is also assumed that the of disease resistant crops is increased by θI_c.

(ii) The infected crops are assumed to be generated by the infection of the disease resistant crops at a rate $\frac{\alpha I_v R_c}{1+\beta I_v}$. It is also assumed that the population of infected crops is further decreased by dI_c and upon infection and display of symptoms, the crops spoiled from infection by θI_c.

(iii) It is further assumed that the population of susceptible vectors is generated by the recruitment of vectors by a recruitment rate A and it is decreased after the effective contact with the infected crops at a rate $\frac{\alpha_1 I_c S_v}{1+\beta_1 I_c}$.

(iv) It also is assumed that the population of infected vectors is generated at a rate $\frac{\alpha_1 I_c S_v}{1+\beta_1 I_c}$, and this population is further decreased by μI_v, due to the natural death of vectors.

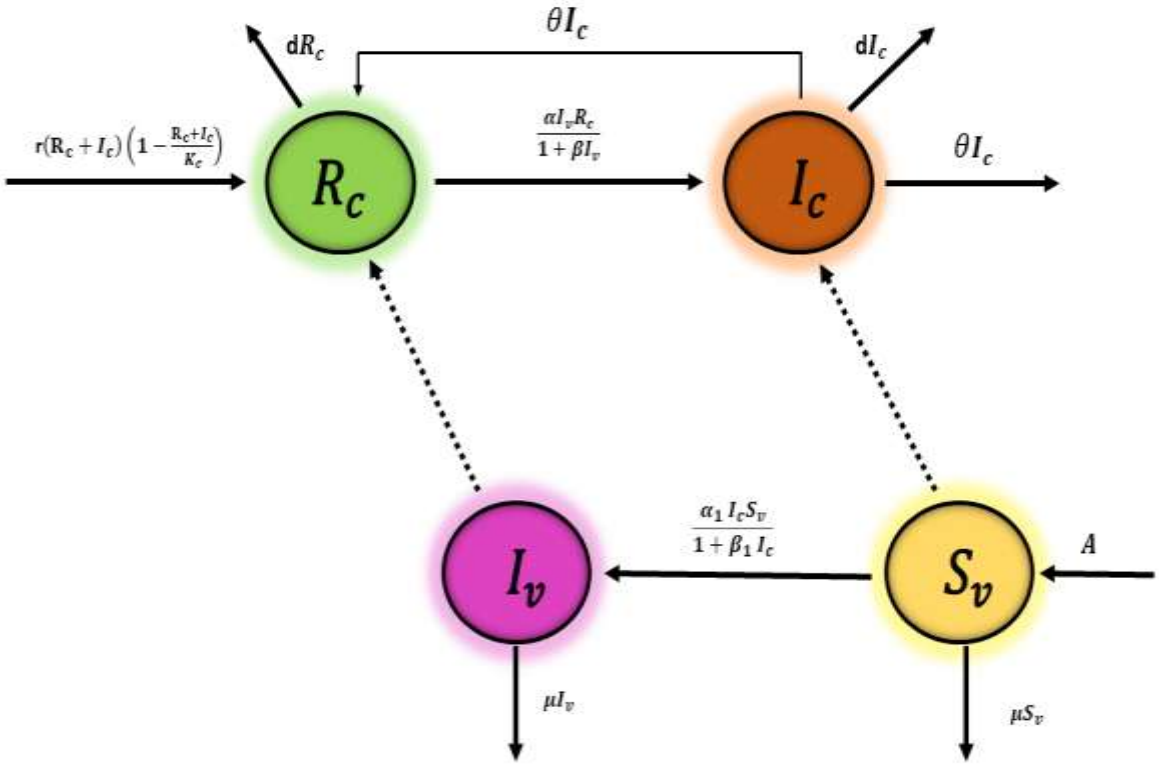

Figure 1: Schematic Diagram of the Proposed Model.

On the basis of above assumptions, the model is developed as follows:

$$\left.\begin{aligned}
\frac{dR_c}{dt} &= r(R_c + I_c)\left(1 - \frac{R_c+I_c}{K_c}\right) - dR_c - \frac{\alpha I_v R_c}{1+\beta I_v} + \theta I_c \\
\frac{dI_c}{dt} &= \frac{\alpha I_v R_c}{1+\beta I_v} - dI_c - \theta I_c \\
\frac{dS_v}{dt} &= A - \frac{\alpha_1 I_c S_v}{1+\beta_1 I_c} - \mu S_v \\
\frac{dI_v}{dt} &= \frac{\alpha_1 I_c S_v}{1+\beta_1 I_c} - \mu I_v
\end{aligned}\right\} \quad (1)$$

with initial conditions:

$$R_c(0) > 0,\ I_c(0) \geq 0,\ S_v(0) > 0,\ I_v(0) \geq 0.$$

Table 1: List of Model Parameters with Description with Corresponding Values

Parameter	Description	Value	Source
r	Intrinsic growth rate of crop biomass/yield	0.1	Assumed
K_c	Carrying capacity of crop biomass/yield	1000	[14]
α	Transmission rate of crop biomass/yield due to vectors	0.0021	Assumed
α_1	Transmission rate of vectors due to crop biomass/yield	0.0003	Assumed
β	Saturation rate of crop biomass/yield due to vectors	0.007	Assumed
β_1	Saturation rate of vectors due to crop biomass	0.01	[3]
d	Natural damage rate of crops	0.01	[18,19]
θ	Damage rate of infected crops	0.01	[19]
A	Recruitment rate of vectors	0.0002	Assumed
μ	Natural death rate of vectors	0.002	Assumed

2. BASIC PROPERTIES OF THE MODEL

It is important to note that the solutions of the model within the region defined by Ω are to be shown positively invariant for all $t \geq 0$ so that the system (1) be mathematically and epidemiologically meaningful and well-posed.

2.1 Non-negativity and Boundedness of Solution

To determine whether solutions are non-negative and constrained, we have the following theorem:

Theorem 2.1 *Every solution of the system (1) with initial conditions is non-negative for all* $t \geq 0$.

Proof: From the system of equations (1), we obtain

$$\frac{dR_c}{dt}|_{R_c=0} = \frac{rI_c}{K_c}(K_c - I_c) + \theta I_c \geq 0\ , \qquad \frac{dI_c}{dt}|_{I_c=0} = \frac{\alpha I_v R_c}{1+\beta I_v} \geq 0,$$

$$\frac{dS_v}{dt}|_{S_v=0} = A \geq 0, \qquad \frac{dI_v}{dt}|_{I_v=0} = \frac{\alpha_1 I_c S_v}{1+\beta_1 I_c} \geq 0.$$

This implies that every solution of the system (1) is non-negative for all $t \geq 0$.

2.2 Invariant Region

Theorem 2.2 *The feasible region of the solution of the system (1) is uniformly bounded. The region* Ω *of the system (1) is defined by* $\Omega = \Omega_c \times \Omega_v$, *where*

$$\Omega_c = \{(R_c, I_c) \in R_+^2 : 0 \leq N_c \leq K_c\}, \quad (2)$$

$$\Omega_v = \{(S_v, I_v) \in R_+^2 : 0 \leq M_v \leq \frac{A}{\mu}\} \quad (3)$$

Proof: Since $N_c(t) = R_c(t) + I_c(t)$, therefore, differentiating $N_c(t)$ w.r.t. 't' and using the first two equations of system (1), we get

$$\frac{dN_c}{dt} \leq rN_c - \frac{rN_c^2}{K} \tag{4}$$

Now, integrating (4) by the method of separation of variables, we get

$$N_c(t) \leq \frac{N_c(0)K_c e^{rt}}{K_c + N_c(0)(e^{rt}-1)} \tag{5}$$

Hence, if $0 \leq N_c(0) \leq K_c$, then we get $\lim sup_{t\to\infty} N_c(t) \leq K_c$.

Thus, the feasible region of the solution of the system (1) for the crop population is given by

$$\Omega_c = \{(R_c, I_c) \in R_+^2 : 0 \leq N_c \leq K_c\} \tag{6}$$

Also, since $M_v(t) = S_v(t) + I_v(t)$, therefore, differentiating $M_v(t)$ w.r.t. t and using the last two equations of the system (1), we get

$$\frac{dM_v}{dt} = A - \mu M_v \tag{7}$$

Solving equation (7), we get

$$M_v(t) = M_v(0)e^{-\mu t} + \frac{A}{\mu}(1 - e^{-\mu t}) \tag{8}$$

Hence, if $0 \leq M_v(0) \leq \frac{A}{\mu}$, then we get $\lim sup_{t\to\infty} M_v(t) \leq \frac{A}{\mu}$.

Thus, the feasible region of the solution of the system (1) for the vector population is given by

$$\Omega_v = \{(S_v, I_v) \in R_+^2 : 0 \leq M_v \leq \frac{A}{\mu}\} \tag{9}$$

Consequently, the feasible region of the system (1) is given by $\Omega = \Omega_c \times \Omega_v$ and is positively invariant. Hence, the solutions of the system (1) are bounded.

Therefore, the model is mathematically and epidemiologically suitable to conduct the study.

3. EQUILIBRIUM POINTS AND BASIC REPRODUCTION NUMBER (R_0)

The system (1) has two equilibrium points, namely a disease-free equilibrium point E_0 and an endemic equilibrium point E^*.

3.1 Existence of Disease-free Equilibrium (DFE)

Disease-free equilibrium E_0 exists, when there are no infections in the crops under consideration and this is possible only when $I_c = 0$ and $I_v = 0$. To find the disease-free equilibrium (E_0), we equate the right-hand side of the system (1) to zero and then solving for the non-infected state variables, we obtain

$$R_c = \frac{K_c(r-d)}{r} \quad \text{and} \quad S_v = \frac{A}{\mu} \tag{10}$$

Hence, there exists a disease-free equilibrium $E_0\left(\frac{K_c(r-d)}{r}, 0, \frac{A}{\mu}, 0\right)$. It is noteworthy that the disease-free equilibrium point is biologically feasible when $r > d$.

3.2 Existence of Endemic Equilibrium (EE)

Endemic equilibrium $E^*(R_c^*, I_c^*, S_v^*, I_v^*)$ with endemic state variables R_c^*, I_c^*, S_v^* and I_v^* exists, when disease persists in the crops. To obtain endemic equilibrium, we equate the right-hand side of system (1) to zero and then, we solve them for endemic state variables $E^*(R_c^*, I_c^*, S_v^*, I_v^*)$ as follows:

$$r(R_c^* + I_c^*)\left(1 - \frac{(R_c^* + I_c^*)}{K_c}\right) - dR_c^* - \frac{\alpha I_v^* R_c^*}{1+\beta I_v^*} + \theta I_c^* = 0 \tag{11}$$

$$\frac{\alpha I_v^* R_c^*}{1+\beta I_v^*} - dI_c^* - \theta I_c^* = 0 \tag{12}$$

$$A - \frac{\alpha_1 I_c^* S_v^*}{1+\beta_1 I_c^*} - \mu S_v^* = 0 \tag{13}$$

$$\frac{\alpha_1 I_c^* S_v^*}{1+\beta_1 I_c^*} - \mu I_v^* = 0 \tag{14}$$

Solving equation (13) in terms of I_c^*, we get

$$S_v^* = \frac{A}{\left(1+\frac{\alpha_1 I_c^* S_v^*}{1+\beta_1 I_c^*}\right)} \tag{15}$$

Using this value of S_v^* in (14) and then solving for I_v^*, we get

$$I_v^* = \frac{A\alpha_1 I_c^*}{\mu[\mu(1+\beta_1 I_c^*)+\alpha_1 I_c^*]} \tag{16}$$

Using this value of I_v^* in (12) and then solving for R_c^*, we get

$$R_c^* = \frac{[\mu\{\mu(1+\beta_1 I_c^*)+\alpha_1 I_c^*\}+A\alpha_1 I_c^*\beta](d+\theta)}{A\alpha_1\alpha} \tag{17}$$

Using the above endemic state variables in (11) and then solving for I_c^*, we get

$$I_c^* = \frac{A\alpha\,\alpha_1 K_c (r-d)-\mu^2 r(d+\theta)}{(\mu^2 r\beta_1+r\alpha_1\mu+rA\beta\alpha_1)(d+\theta)+A\alpha\alpha_1 r} \tag{18}$$

Thus, there exists an endemic equilibrium $E^*(R_c^*, I_c^*, S_v^*, I_v^*)$, where

$$R_c^* = \frac{[\mu\{\mu(1+\beta_1 I_c^*)+\alpha_1 I_c^*\}+A\alpha_1\beta I_c^*](d+\theta)}{A\alpha_1\alpha}, \qquad S_v^* = \frac{A}{\left(1+\frac{\alpha_1 I_c^* S_v^*}{1+\beta_1 I_c^*}\right)}$$

$$I_c^* = \frac{A\alpha\alpha_1 K_c(r-d)-\mu^2 r(d+\theta)}{(\mu^2 r\beta_1+r\alpha_1\mu+rA\beta\alpha_1)(d+\theta)+A\alpha\alpha_1 r}, \qquad I_v^* = \frac{A\alpha_1 I_c^*}{\mu[\mu(1+\beta_1 I_c^*)+\alpha_1 I_c^*]}$$

3.3 Basic Reproduction Number (R_0)

The expression for the basic reproduction number R_0 for the system (1) can be obtained by using the next generation matrix method as follows [17]:

To employ next generation matrix method, we take only the infected compartment from the system (1) and thus, we have the following infective class sub-systems:

$$\frac{dI_c}{dt} = \frac{\alpha I_v R_c}{1+\beta I_v} - dI_c - \theta I_c \tag{19}$$

$$\frac{dI_v}{dt} = \frac{\alpha_1 I_c S_v}{1+\beta_1 I_c} - \mu I_v \tag{20}$$

The RHS of the above infective class sub system can be written as matrix f and g, where

$$f = \begin{bmatrix} \frac{\alpha I_v R_c}{1+\beta I_v} \\ \frac{\alpha_1 I_c S_v}{1+\beta I_c} \end{bmatrix} \quad \text{and} \quad g = \begin{bmatrix} (d+\theta) I_c \\ \mu I_v \end{bmatrix} \tag{21}$$

The associated transmission matrix (F) of f and the transition matrix (G) of g at $E_0\left(\frac{K_c(r-d)}{r}, 0, \frac{A}{\mu}, 0\right)$ are respectively given by

$$F = \begin{bmatrix} 0 & \frac{\alpha K_c(r-d)}{r} \\ \frac{\alpha_1 A}{\mu} & 0 \end{bmatrix} \quad \text{and} \quad G = \begin{bmatrix} d+\theta & 0 \\ 0 & \mu \end{bmatrix} \tag{22}$$

Now, the next generation matrix of the model is given by FG^{-1} and the basic reproduction number R_0 is determined by the spectral radius ρ of FG^{-1} which is the largest eigenvalue of the matrix FG^{-1}. Now, we find

$$G^{-1} = \begin{bmatrix} \frac{1}{(d+\theta)} & 0 \\ 0 & \frac{1}{\mu} \end{bmatrix} \text{ and so } FG^{-1} = \begin{bmatrix} 0 & \frac{\alpha K_c(r-d)}{\mu r} \\ \frac{\alpha_1 A}{\mu(d+\theta)} & 0 \end{bmatrix} \tag{23}$$

The eigenvalues of the product matrix FG^{-1} are given by

$$\begin{vmatrix} -\lambda & \frac{\alpha K_c(r-d)}{\mu r} \\ \frac{\alpha_1 A}{\mu(d+\theta)} & -\lambda \end{vmatrix}=0$$

or $$\lambda^2 - \frac{\alpha\alpha_1 A K_c(r-d)}{\mu^2 r(d+\theta)} = 0 \quad (24)$$

Solving the above equation (24), we get

$$\lambda = \pm\sqrt{\frac{\alpha\alpha_1 A K_c(r-d)}{\mu^2 r(d+\theta)}}$$

Thus, we have $$\rho(FG^{-1}) = \sqrt{\frac{\alpha\alpha_1 A K_c(r-d)}{\mu^2 r(d+\theta)}}$$

Therefore, the expression for the basic reproduction number R_0 is given by

$$R_0 = \sqrt{\frac{\alpha\alpha_1 A K_c(r-d)}{\mu^2 r(d+\theta)}}$$

4. STABILITY ANALYSIS

Let f_1, f_2, f_3 and f_4 denote the left-hand side of the equations of the system (1) i.e.

$$f_1: \frac{dR_c}{dt} = r(R_c + I_c)\left(1 - \frac{R_c+I_c}{K_c}\right) - dR_c - \frac{\alpha I_v R_c}{1+\beta I_v} + \theta I_c$$
$$f_2: \frac{dI_c}{dt} = \frac{\alpha I_v R_c}{1+\beta I_v} - dI_c - \theta I_c$$
$$f_3: \frac{dS_v}{dt} = A - \frac{\alpha_1 I_c S_v}{1+\beta_1 I_c} - \mu S_v$$
$$f_4: \frac{dI_v}{dt} = \frac{\alpha_1 I_c S_v}{1+\beta_1 I_c} - \mu I_v$$

Then, the Jacobian matrix of the system (1) is given by

$$J(E) = \begin{bmatrix} \frac{\partial f_1}{\partial R_c} & \frac{\partial f_1}{\partial I_c} & \frac{\partial f_1}{\partial S_v} & \frac{\partial f_1}{\partial I_v} \\ \frac{\partial f_2}{\partial R_c} & \frac{\partial f_2}{\partial I_c} & \frac{\partial f_2}{\partial S_v} & \frac{\partial f_2}{\partial I_v} \\ \frac{\partial f_3}{\partial R_c} & \frac{\partial f_3}{\partial I_c} & \frac{\partial f_3}{\partial S_v} & \frac{\partial f_3}{\partial I_v} \\ \frac{\partial f_4}{\partial R_c} & \frac{\partial f_4}{\partial I_c} & \frac{\partial f_4}{\partial S_v} & \frac{\partial f_4}{\partial I_v} \end{bmatrix} = \begin{bmatrix} A_{11} & A_{12} & 0 & \frac{-\alpha R_c}{(1+\beta I_v)^2} \\ \frac{\alpha I_v}{1+\beta I_v} & -(d+\theta) & 0 & \frac{\alpha R_c}{(1+\beta I_v)^2} \\ 0 & \frac{-\alpha_1 S_v}{(1+\beta_1 I_c)^2} & \frac{-\alpha_1 I_c}{1+\beta_1 I_c} - \mu & 0 \\ 0 & \frac{\alpha_1 S_v}{(1+\beta_1 I_c)^2} & \frac{\alpha_1 I_c}{1+\beta_1 I_c} & -\mu \end{bmatrix} \quad (25)$$

where

$$A_{11} = r - \frac{2r(R_c+I_c)}{K_c} - d - \frac{\alpha I_v}{1+\beta I_v}, \qquad A_{12} = r - \frac{2r(R_c+I_c)}{K_c} + \theta$$

The eigenvalues of the Jacobian matrix J are given by $|J - \lambda I| = 0$.

4.1 Local Stability of Disease-free Equilibrium Point

To discuss local stability of disease-free equilibrium point $E_0\left(\frac{K_c}{r}(r-d), 0, \frac{A}{\mu}, 0\right)$ based on basic reproduction number R_0, we have the following theorem:

Theorem 4.1 *If $R_0 < 1$, then the disease-free equilibrium point E_0 is locally asymptotically stable.*

Proof The Jacobian matrix of the system (1) at the point $E_0\left(\frac{K_c}{r}(r-d), 0, \frac{A}{\mu}, 0\right)$ is obtained by using $\left(\frac{K_c}{r}(r-d), 0, \frac{A}{\mu}, 0\right)$ in (25) and is given by

$$J(E_0) = \begin{bmatrix} -r+d & -r+2d+\theta & 0 & -\frac{\alpha K_c(r-d)}{r} \\ 0 & -(d+\theta) & 0 & \frac{\alpha K_c(r-d)}{r} \\ 0 & \frac{-\alpha_1 A}{\mu} & -\mu & 0 \\ 0 & \frac{\alpha_1 A}{\mu} & 0 & -\mu \end{bmatrix} \quad (26)$$

The characteristic equation for the above Jacobian matrix is

$$[-(r-d)-\lambda][\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3] = 0 \quad (27)$$

where

$$\left.\begin{aligned} B_1 &= \{(d+\theta)+2\mu\} > 0 \\ B_2 &= \{\mu(d+\theta)(2-R_0^2)+\mu^2\} > 0,\ if\ R_0 < 1 \\ B_3 &= \{(d+\theta)\mu^2(1-R_0^2)\} > 0,\ if\ R_0 < 1 \end{aligned}\right\} \quad (28)$$

Clearly, one eigenvalue is $-(r-d)$. The rest three eigenvalues are computed from the cubic equation $\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3 = 0$ by using Routh-Hurwitz criterion. The roots of the above cubic equation will have negative real parts if $B_1 > 0, B_3 > 0$ and $B_1B_2 - B_3 > 0$.

Thus, the disease-free equilibrium point E_0 is locally asymptotically stable if $R_0 < 1$.

4.2 Sensitivity Analysis of Basic Reproduction Number R_0

Based on each of the parameters from the expression of the basic reproduction number R_0, sensitivity analysis is performed to check the sensitivity of the basic reproduction number [17]. In order to reduce the effect of virus, it is necessary to control the parameter values to make $R_0 < 1$. Therefore,

we compute the sensitivity indices (i.e. the rate of change) of R_0 with respect to the main parameters. For example, the sensitivity indices of R_0 with respect to the parameter α is given by

$$S_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha}.\frac{\alpha}{R_0}, \text{ where } R_0 = \sqrt{\frac{\alpha\alpha_1 AK_c(r-d)}{\mu^2 r(d+\theta)}} \quad (29)$$

The sensitivity indices of R_0 with respect to various parameters are computed and the corresponding sensitivity indices are displayed in **Table 2.**

Table 2: Sensitivity Indices of R_0 w. r. t. Various Model Parameters

Parameter	θ	α	d	α_1	μ	K_c	r	A
Sensitivity Index	-0.25	0.5	-0.31	0.5	-1.0	0.5	0.06	0.5

From the above table, it can be noted that the parameters α, α_1, K_c, r and A have positive sensitive indices showing that there is more impact on the spread of the disease in the crops. The parameters d, μ and θ have negative sensitive indices showing that there is less impact on spread of the crop disease.

The bar chart of the sensitive indices of R_0 is shown in **Figure 2**. It can also be noted from **Figure 2,** that the parameter α, α_1, K_c, r and A have the positive impact on the spread of the crop disease, whereas the parameters d, μ and θ have negative impact on the spread of the crop diseases.

International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

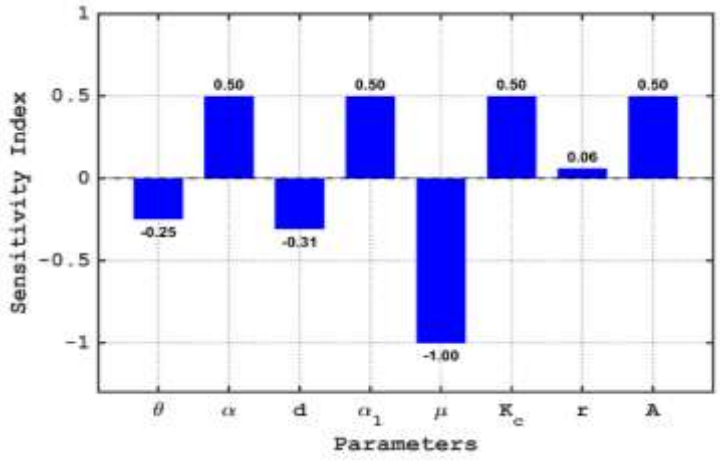

Figure 2: Bar Chart of Sensitivity Indices of R_0 w. r. t. Model Parameters

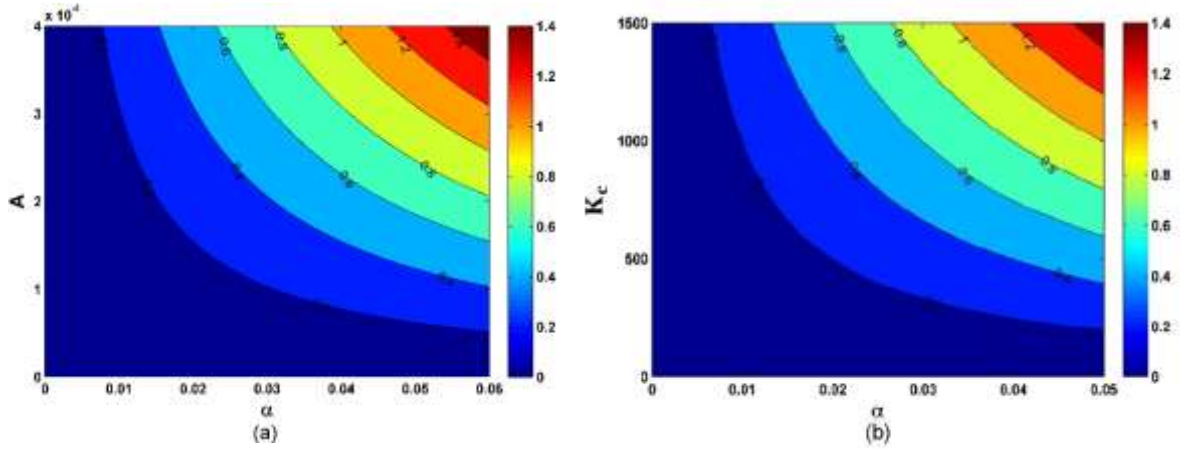

Figure 3: Contour Plot of R_0 with (a) (α, A)-plane and (b) (α, K_c)-plane

The contour plot of R_0 w. r. t. parameters (α, A) and (α, K_c) are shown in **Figure 3,** from which it is observed that only increasing the value of α can shift $R_0 < 1$ to $R_0 > 1$.

4.3 Local Stability of Endemic Equilibrium Point

We describe the local stability of endemic equilibrium point in the following theorem:

Theorem 4.2 *The endemic equilibrium point $E^*(R_c^*, I_c^*, S_v^*, I_v^*)$ is locally asymptotically stable if $R_0 > 1$.*

Proof. The Jacobian matrix of system (1) at endemic equilibrium point E^* is given by

$$J(E^*) = \begin{bmatrix} a_{11} & a_{12} & 0 & a_{14} \\ a_{21} & a_{22} & 0 & a_{24} \\ 0 & a_{32} & a_{33} & 0 \\ 0 & a_{42} & a_{43} & a_{44} \end{bmatrix} \tag{30}$$

where the non-zero entities of the above matrix are given as follows:

$$a_{11} = r - \frac{2r(R_c^*+I_c^*)}{K_c} - d - \frac{\alpha I_v^*}{1+\beta I_v^*},\ a_{12} = r - \frac{2r(R_c^*+I_c^*)}{K_c} + \theta,\ a_{14} = -\frac{\alpha R_c^*}{(1+\beta I_v^*)^2},\ a_{21} = \frac{\alpha I_v^*}{1+\beta I_v^*},$$
$$a_{22} = -(d+\theta),\ a_{24} = \frac{\alpha R_c^*}{(1+\beta I_v^*)^2},\ a_{32} = -\frac{\alpha_1 S_v^*}{(1+\beta_1 I_c^*)^2},\ a_{33} = -\frac{\alpha_1 I_c^*}{1+\beta_1 I_c^*} - \mu,\ a_{42} = \frac{\alpha_1 S_v^*}{(1+\beta_1 I_c^*)^2},$$
$$a_{43} = \frac{\alpha_1 I_c^*}{(1+\beta_1 I_c^*)},\ a_{44} = -\mu$$

The characteristic equation of the above matrix is given by

$$\lambda^4 + M_1\lambda^3 + M_2\lambda^2 + M_3\lambda + M_4 = 0 \tag{31}$$

where

$$\left.\begin{aligned} M_1 &= -\{a_{22} + a_{33} + a_{44} + a_{11}\} \\ M_2 &= \{a_{22}a_{11} + a_{33}a_{11} + a_{44}a_{11} + a_{33}a_{22} + a_{44}a_{22} + a_{33}a_{44} - a_{42}a_{24} - a_{12}a_{21}\} \\ M_3 &= \{a_{14}a_{42}a_{21} + a_{44}a_{12}a_{21} + a_{33}a_{12}a_{21} + a_{24}a_{42}a_{33} + a_{42}a_{24}a_{11} - a_{44}a_{22}a_{11} \\ &\quad -a_{33}a_{22}a_{11} - a_{33}a_{44}a_{11} - a_{22}a_{33}a_{44} - a_{24}a_{32}a_{43}\} \\ M_4 &= \{a_{11}a_{22}a_{33}a_{44} + a_{24}a_{32}a_{43}a_{11} + a_{14}a_{32}a_{42}a_{21} - a_{24}a_{42}a_{33}a_{11} \\ &\quad -a_{14}a_{42}a_{33}a_{21} - a_{21}a_{12}a_{33}a_{44}\} \end{aligned}\right\} \tag{32}$$

Using the Routh-Hurwitz criterion, the root of the equation (31) will have negative real parts if

$$M_1 > 0, M_2 > 0, M_3 > 0, M_4 > 0 \text{ and } \quad M_3(M_1M_2 - M_3) > M_1^2M_4 \tag{33}$$

Hence, all the eigenvalues of the equation (33) at E^* have negative real parts.

Thus, the endemic equilibrium point E^* is locally asymptotically stable, if $R_0 > 1$.

4.4 Bifurcation Analysis

To study the possibility of existence of the bifurcation of the system (1) at $R_0 = 1$, we apply the centre manifold theory [17] to analyze the dynamics of the system. By this theory, the stability of the equilibria of the model can be completely characterized by the bifurcation coefficients m and n given by

$$m = \sum_{i,j,k=1}^{4} z_k\, u_i u_j \frac{\partial^2 h_k}{\partial x_i \partial x_j}(E_0), \quad n = \sum_{i,k=1}^{4} z_k u_i \frac{\partial^2 h_k}{\partial x_i \partial \alpha^*}(E_0)$$

where $u = (u_1, u_2, u_3, u_4)^T$ and $z = (z_1, z_2, z_3, z_4)$ are the right and left eigenvectors respectively obtained from $J(E_0)u = 0$ and $zJ(E_0) = 0$ and h_k is the right hand side of the system (1) with the change of variables $R_c = x_1$, $I_c = x_2$, $S_v = x_3$, and $I_v = x_4$.

Using the vector notation $X = (x_1, x_2, x_3, x_4)^T$, the system (1) can be expressed in the form $\frac{dX}{dt} = (h_1, h_2, h_3, h_4)^T$ as:

$$\left.\begin{aligned} \frac{dx_1}{dt} &= r(x_1 + x_2)\left(1 - \frac{x_1+x_2}{K_c}\right) - dx_1 - \frac{\alpha x_1 x_4}{1+\beta x_4} + \theta x_2 \\ \frac{dx_2}{dt} &= \frac{\alpha x_1 x_4}{1+\beta x_4} - dx_2 - \theta x_2 \\ \frac{dx_3}{dt} &= A - \frac{\alpha_1 x_2 x_3}{1+\beta_1 x_2} - \mu x_3 \\ \frac{dx_4}{dt} &= \frac{\alpha_1 x_2 x_3}{1+\beta_1 x_2} - \mu x_4 \end{aligned}\right\} \tag{34}$$

Now, we have the following theorem:

Theorem 4.3 *The proposed system (1) shows the forward bifurcation at* $R_0 = 1$.

Proof: Let us assume $\alpha = \alpha^*$ as bifurcation parameter at $R_0 = 1$.

Putting $R_0 = 1$ in the expression $R_0 = \frac{\alpha\alpha_1 K_c A(r-d)}{\mu^2 r(d+\theta)}$ and simplifying, we have

$$\alpha = \alpha^* = \frac{\mu^2 r(d+\theta)}{\alpha_1 K_c A(r-d)} \tag{35}$$

Now, the Jacobian matrix of the system (35) at the disease-free equilibrium E_0 is given by

International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

$$J(E_0) = \begin{bmatrix} -r+d & -r+2d+\theta & 0 & -\alpha^* K_c\left(1-\frac{d}{r}\right) \\ a_{21} & -(d+\theta) & 0 & \alpha^* K_c\left(1-\frac{d}{r}\right) \\ 0 & -\frac{\alpha_1 A}{\mu} & -\mu & 0 \\ 0 & \frac{\alpha_1 A}{\mu} & 0 & -\mu \end{bmatrix} \tag{36}$$

The right eigenvector $u = (u_1, u_2, u_3, u_4)^T$ is determined from $J(E_0)u = 0$ so that, we have

$$(-r+d)u_1 + (-r+2d+\theta)u_2 + \left(-\alpha^* K_c\left(1-\frac{d}{r}\right)\right)u_4 = 0 \tag{37}$$

$$-(d+\theta)u_2 + \left(\alpha^* K_c\left(1-\frac{d}{r}\right)\right)u_4 = 0 \tag{38}$$

$$\left(-\frac{\alpha_1 A}{\mu}\right)u_2 - \mu u_3 = 0 \tag{39}$$

$$\left(\frac{A_1 A}{\mu}\right)u_2 - \mu u_4 = 0 \tag{40}$$

Solving equation (40) in terms of u_4, we get

$$u_2 = \frac{\mu^2}{\alpha_1 A}u_4 \tag{41}$$

Using this value of u_2 in (39) and then solving for u_3, we get

$$u_3 = -u_4 \tag{42}$$

Again, using the value of u_2 in (37) and then solving for u_1, we get

$$u_1 = -\frac{\mu^2}{\alpha_1 A}u_4, \text{ where } \quad u_4 > 0 \tag{43}$$

The left eigenvector $z = (z_1, z_2, z_3, z_4)$ is determined from $zJ(E_0) = 0$ so that, we have

$$(-r+d)z_1 = 0 \tag{44}$$

$$(-r+2d+\theta)z_1 - (d+\theta)z_2 - \frac{\alpha_1 A}{\mu}z_3 + \frac{\alpha_1 A}{\mu}z_4 = 0 \tag{45}$$

$$-\mu z_3 = 0 \tag{46}$$

$$\left(-\alpha^* K_c\left(1-\frac{d}{r}\right)\right)z_1 + \alpha^* K_c\left(1-\frac{d}{r}\right)z_2 - \mu z_4 = 0 \tag{47}$$

Solving equations (44) and (46), we get

$$z_1 = z_3 = 0 \tag{48}$$

Using the above values of z_1 and z_3 in (45) and then solving for z_2, we get

$$z_2 = \frac{\alpha_1 A}{(d+\theta)\mu}z_4, \text{ where } \ z_4 > 0 \tag{49}$$

The non-zero second order partial derivatives of $h_i,\ i = 1,2,3,4$ are given as follows:

$$\frac{\partial^2 h_2}{\partial x_4 \partial x_1} = \frac{\partial^2 h_2}{\partial x_1 \partial x_4} = \frac{\alpha}{(1+\beta x_4)^2}\alpha, \ \frac{\partial^2 h_2}{\partial x_4 \partial x_4} = \frac{2\alpha x_1 \beta}{(1+\beta x_4)^3}, \ \frac{\partial^2 h_4}{\partial x_2 \partial x_2} = \frac{2\alpha_1 x_3 \beta_1}{(1+\beta_1 x_2)^3},$$

$$\frac{\partial^2 h_4}{\partial x_3 \partial x_2} = \frac{\partial^2 h_4}{\partial x_2 \partial x_3} = \frac{\alpha_1}{(1+\beta_1 x_2)^2}, \ \frac{\partial^2 h_2}{\partial x_1 \partial \alpha^*} = \frac{x_4}{(1+\beta x_4)}, \ \frac{\partial^2 h_2}{\partial x_4 \partial \alpha^*} = \frac{x_1}{(1+\beta x_4)^2}$$

All the other second order partial derivatives of $h_i, i = 1,2,3,4$ are found to be zero.

Now, based on the centre manifold theory, the coefficients m and n are given by

$$m = \sum_{i,j,k=1}^{4} z_k u_i u_j \frac{\partial^2 h_k}{\partial x_i \partial x_j}(E_0) = -\frac{2\alpha\mu}{(d+\theta)} + \frac{2\alpha K_c (r-d)\beta\alpha_1 A}{r\mu(d+\theta)} + \frac{2\mu^3\beta_1}{A} + \frac{2\mu^2}{A}z_4\mu_4^2 < 0$$

International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

$$n = \sum_{i,k=1}^{4} z_k u_i \frac{\partial^2 h_k}{\partial x_i \partial \alpha^*}(E_0) = \frac{\mu}{\alpha} z_4 u_4 > 0$$

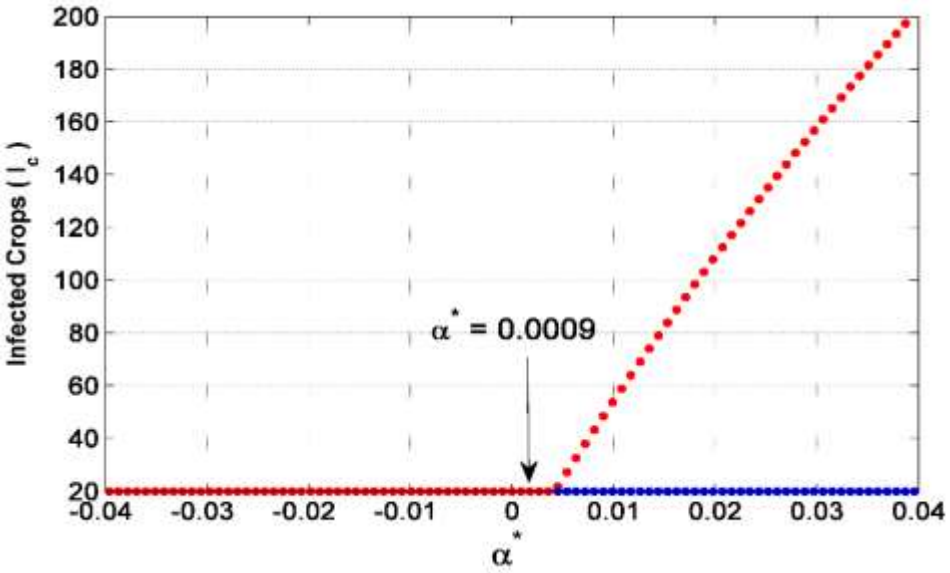

Figure 4: Diagram showing Forward Bifurcation

Here, it is seen that m is negative and n is positive, therefore, the system (1) shows the forward bifurcation at $R_0 = 1$ and if $R_0 > 1$, then at least one stable endemic equilibrium exists.
In the forward bifurcation diagram, the parameter values are taken from **Table 1** except the value $\alpha = 0.0009$.

5. NUMERICAL SIMULATIONS

In order to discuss the quantitative behaviour of the system (1), the numerical simulations were performed to support the analytical results. Some parameter values were assumed and some of them were taken from the various literature. Basic reproduction number R_0 is computed by using parameter values given in Table 1, and by assuming the initial data as $R_c(0) = 980,\ I_c(0) = 20, S_v(0) = 200, I_v(0) = 50$ and is found to be $R_0 = 1.35$. Now, for the value of $R_0 = 1.35 > 1$, we discuss the numerical simulation for the existence of endemic equilibrium point.
From Figure 5(a), we note that the disease resistant crop (biomass/ yield) decreases logistically while the infected crop (biomass/ yield) increases logistically and from Figure 5(b), it is noted that the susceptible vector population decreases logistically while infected vector population increases exponentially with $R_0 = 1.35 > 1$.
From Figure 6(a), we observe that due to implementation in the control strategies, if the infection transmission rate α of the crop biomass/ yield decreases, then the infected crop biomass/ yield decreases and from Figure 6(b), we observe that if the number of infected vectors decreases, then the value of transmission rate α decreases, leading to the decrease of the basic reproduction number R_0.
From Figure 7(a), we note that if the value of infection transmission rate α_1 of vector decreases, then the number of infected crops decreases and from Figure 7(b), we observe that the number of infected vectors decreases as the value of α_1 decreases.
From Figure 8(a) and Figure 8(b), we observe that if the saturation rate β of the crops increases, then the number of infected crop biomass/ yield and infected vectors decrease.
From Figure 9(a) and Figure 9(b), we observe that if the saturation rate β_1 of the vector increases, then the number of infected crop biomass/ yield and infected vectors decrease.

International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

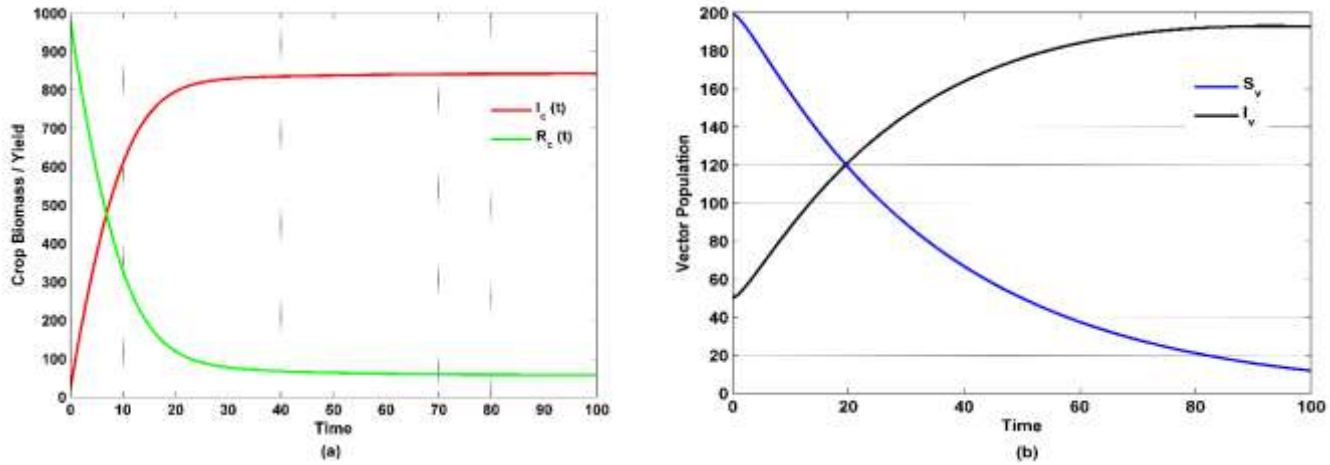

Figure 5: Variation of Crop biomass/ yield and Vector Population with time for $R_0 = 1.35$

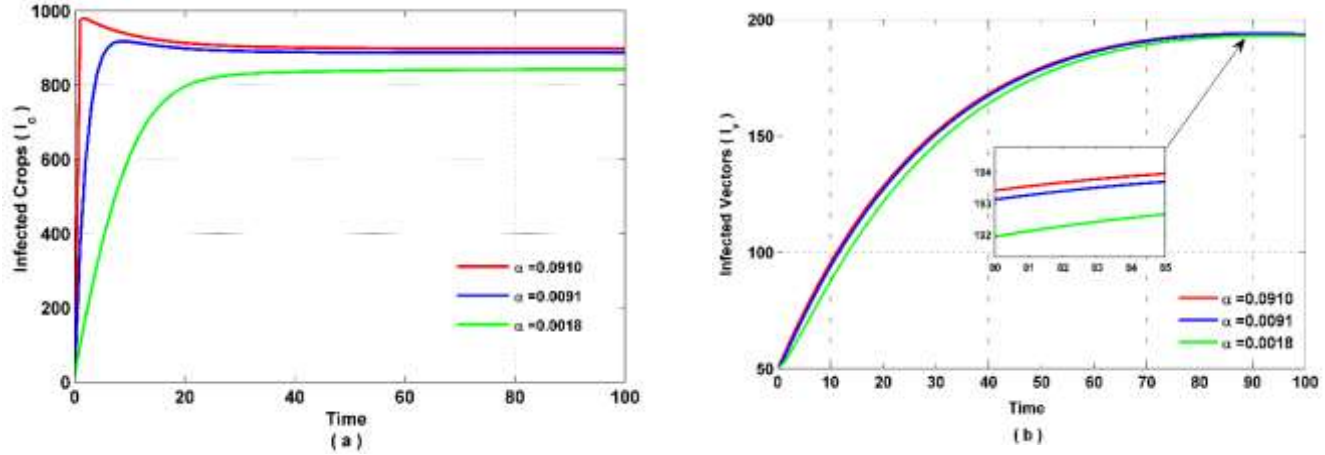

Figure 6: Variation of Infected Crop biomass/ yield and Infected Vector Population with Varying Effects of Parameters at Different Values of α.

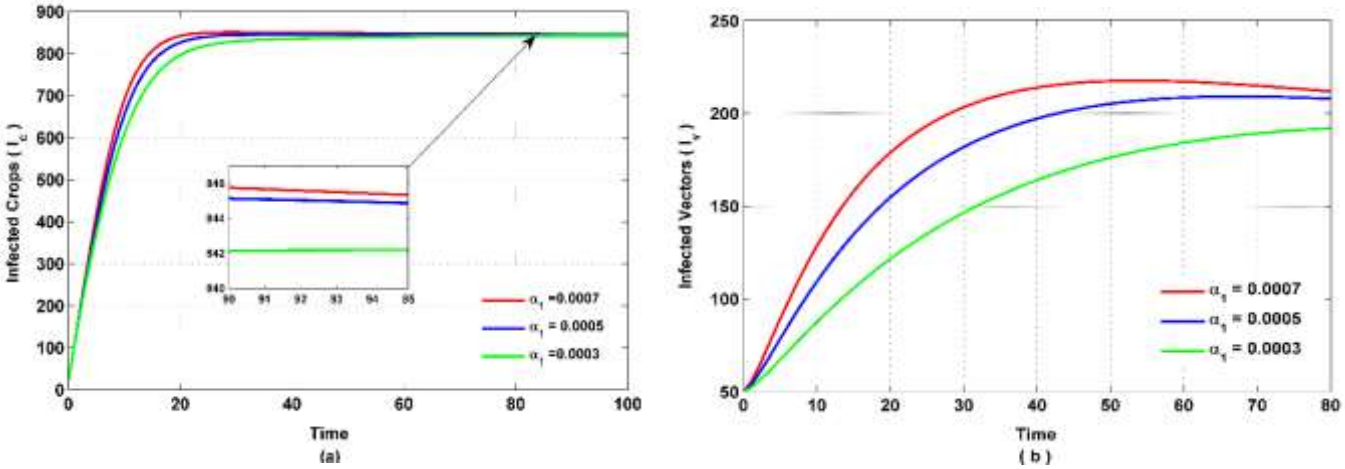

Figure 7: Variation of Infected Crop biomass/ yield and Infected Vector Population with Varying Effects of Parameters at Different Values of α_1.

International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 5, 2025
https://theaspd.com/index.php/ijes

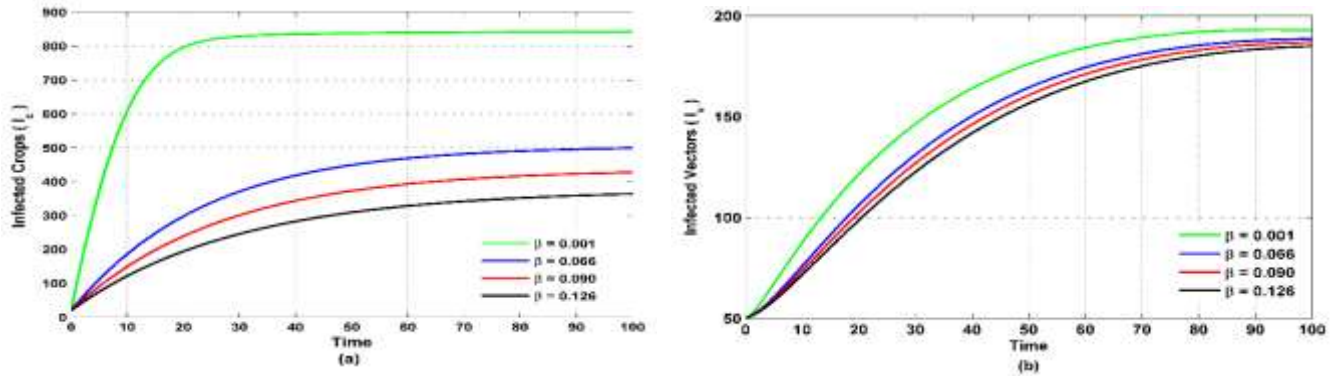

Figure 8: Variation of Infected Crop biomass/ yield and Infected Vector Population with Varying Effects of Parameters at Different Values of β.

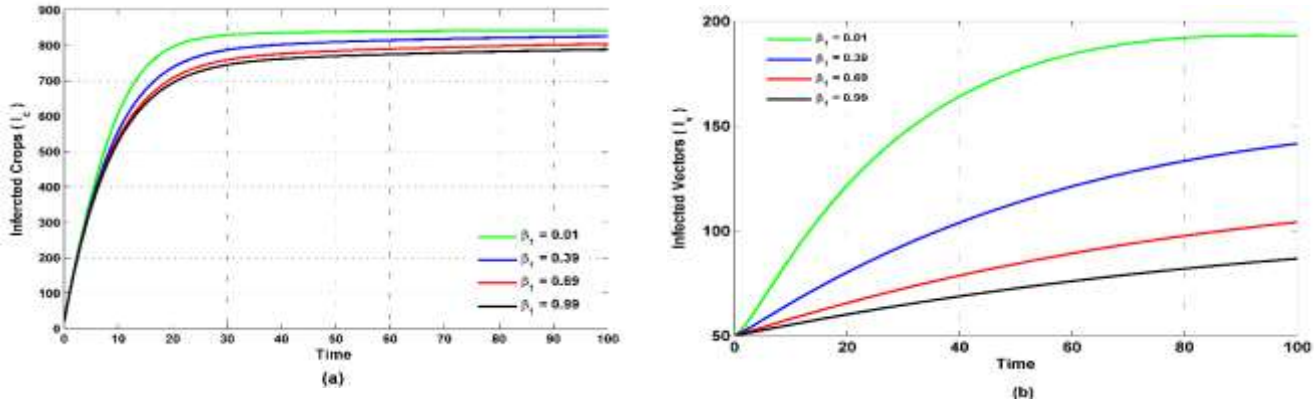

Figure 9: Variation of Infected Crop biomass/yield and Infected Vector Population with Varying Effect of Parameters at Different Values of β_1.

6. DISCUSSION AND CONCLUSIONS

Mathematical modelling and bifurcation analysis of vector-borne crop diseases are presented in this paper by incorporating non-linear saturation rate of Holling type II. First of all, a mathematical model is developed by taking into account the logistic crop growth and vector dynamics. In the present model, four distinct compartments have been considered and the model is described by using a system of ordinary differential equations. Then, the basic properties of the model such as the non-negativity, boundedness and existence of solution are discussed. Thereafter, two equilibrium points, namely disease-free equilibrium (E_0) and endemic equilibrium (E^*) are computed. The basic reproduction number is calculated by using the next generation matrix method. The disease-free equilibrium point is found to be locally asymptotically stable and the endemic equilibrium point of the system is studied by using the Routh-Hurwitz criterion. Sensitivity analysis of the basic reproduction number is performed to identify the influential parameters in the spread of the crop diseases. It has been shown that the transmission rate (α) of the crop biomass due to vectors, the transmission rate (α_1) of vectors due to crop biomass, the recruitment rate (A) of vectors, carrying capacity (K_c) of crop biomass and intrinsic growth rate (r) crop biomass show positive impact on the spread of crop diseases. By using centre manifold theory, it has been shown that the model system exhibits the forward bifurcation at $R_0 = 1$. The numerical simulations of the model indicate the need for implementing effective control interventions to manage the disease in the crops. It has been also shown that if the saturation rate (β) of the crop biomass and the saturation rate (β_1) of the vectors increase, then the number of infected crops and number of infected vectors both decrease. This model will be useful in developing strategies to

control vector-borne diseases at both the vegetative and the reproductive systems of the crop biomass/yield that leads to higher production and productivity for food security.

ACKNOWLEDGEMENT

The corresponding author extends gratitude to the CSIR-NET for providing financial support for the research work (Junior Research Fellow of CSIR Sanction Award Letter No. 09/0057(14059)/2022-EMR-I).

CONFLICTS OF INTEREST: The authors declare no conflict of interest.

REFERENCES

1. Fedele, G., Brischetto, C., Rossi, V., & Gonzalez-Dominguez, E. (2022). A systematic map of the research on disease modelling for agricultural crops worldwide. Plants, 11(6), 724.
2. Savary, S., Nelson, A., Pangga, I., Aunario, J., Hijmans, R., Wassmann, R., & Willocquet, L. (2010). Food security, crop health, and global change. Producing more with less: Exploring farm-based approaches to improve productivity and providing options to farmers in adapting to climate change.
3. Stella, I. R., & Ghosh, M. (2018). Modelling and analysis of plant disease with delay and logistic growth of insect vector. Communications in Mathematical Biology and Neuroscience. https://doi.org/10.28919/cmbn/3751
4. Ajilogba, C. F., & Babalola, O. O. (2013). Integrated management strategies for tomato Fusarium wilt. Biocontrol Science, 18(3), 117–127.
5. Pelczar, M. J., Shurtleff, M. C., Kelman, A., & Pelczar, R. M. (2020). Plant disease. Encyclopaedia Britannica.
6. Mao, L., Jiang, H., Zhang, L., Zhang, Y., Sial, M. U., Yu, H., et al. (2017). Replacing methyl bromide with a combination of 1,3-dichloropropene and metam sodium for cucumber production in China. PLoS One, 12(11), e0188137.
7. Leadbeater, A. (2015). Recent developments and challenges in chemical disease control - A review. Plant Protection Science, 51(4), 163–169.
8. Chapwanya, M., & Dumont, Y. (2021). Application of mathematical epidemiology to crop vector-borne diseases: The cassava mosaic virus disease case. Infectious Diseases on Our Planet, 57–95.
9. Bazarra, N., Colturato, M., Fernández, J. R., Naso, M. G., Simonetto, A., & Gilioli, G. (2022). Analysis of a mathematical model arising in plant disease epidemiology. Applied Mathematics and Optimization, 85(2), 19.
10. Seidu, B., Asamoah, J. K. K., Wiah, E. N., & Ackora-Prah, J. (2022). A comprehensive cost-effectiveness analysis of control of maize streak virus disease with Holling's Type II predation form and standard incidence. Results in Physics, 40, 105862.
11. Tang, S., Xiao, Y., & Cheke, R. A. (2010). Dynamical analysis of plant disease models with cultural control strategies and economic thresholds. Mathematics and Computers in Simulation, 80(5), 894–921.
12. Gao, S., Xia, L., Liu, Y., & Xie, D. (2016). A plant virus disease model with periodic environment and pulse rouging. Studies in Applied Mathematics, 136(4), 357–381. https://doi.org/10.1111/sapm.12109
13. Jackson, M., & Chen-Charpentier, B. M. (2017). Modeling plant virus propagation with delays. Journal of Computational and Applied Mathematics, 309, 611–621.
14. Anggriani, N., Ndii, M. Z., Arumi, D., Istifadah, N., & Supriatna, A. K. (2018). Mathematical model for plant disease dynamics with curative and preventive treatments. AIP Conference Proceedings, 2043(1).
15. Holcman, E., Sentelhas, P. C., Spósito, M. B., Conceição, M. A. F., & Couto, H. T. Z. (2019). Yield and quality of grapes cultivated under plastic coverings with different downy mildew control strategies. Journal of Phytopathology, 167(3), 185–195.
16. Meng, X., & Li, Z. (2010). The dynamics of plant disease models with continuous and impulsive cultural control strategies. Journal of Theoretical Biology, 266(1), 29–40.
17. Melese, A. S., Makinde, O. D., & Obsu, L. L. (2022). Mathematical modelling and analysis of coffee berry disease dynamics on a coffee farm. Mathematical Biosciences and Engineering, 19(7), 7349–7373.
18. Jackson, M., & Chen-Charpentier, B. M. (2018). A model of biological control of plant virus propagation with delays. Journal of Computational and Applied Mathematics, 330, 855–865. https://doi.org/10.1016/j.cam.2017.01.005
19. Stella, I. R., & Ghosh, M. (2019). Modeling plant disease with biological control of insect pests. Stochastic Analysis and Applications, 37(6), 1133–1154.